#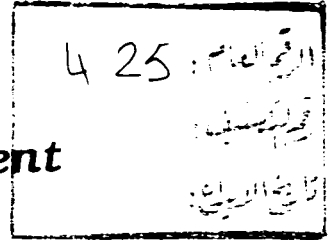 A COMPARATIVE STUDY BETWEEN CHILDREN WITH TEMPORAL LOBE EPILEPSY AND THOSE WITH GRAND MAL EPILEPSY REGARDING SOME COGNITIVE AND PERSONALITY FACTORS

Submitted in Fulfilment

For

Ph. D. Degree in Childhood studies
Medical Department

By
Ashraf Abdel Raouf Mohammed Osman

Supervised By

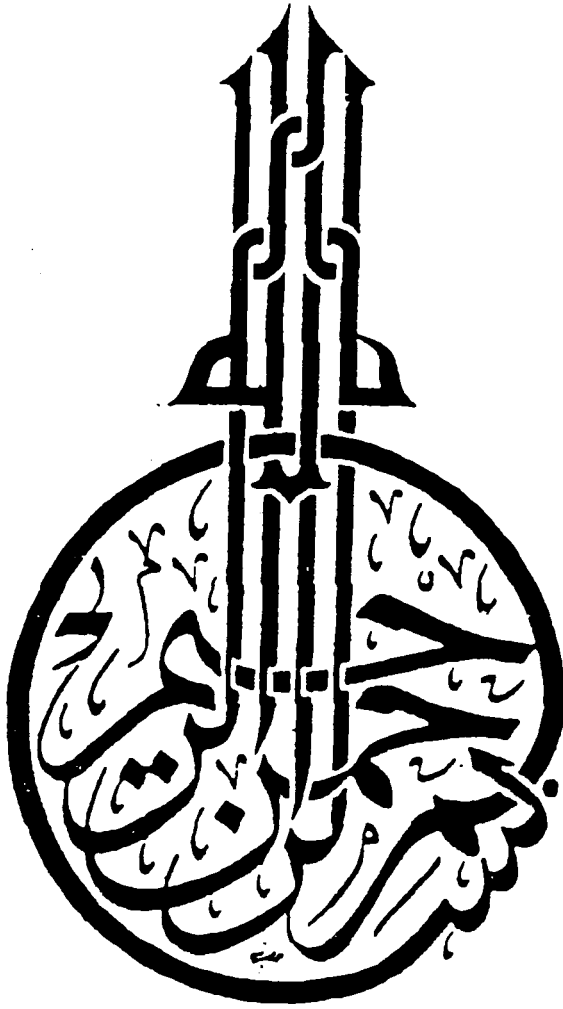| Prof. Dr Kadry Mahmoud Hefny	Prof. Dr Abdel Latif Moussa Osman	Prof. Dr Afaf Hamed Khalil
Prof. Head, of Department of psychology Inst. Post. Grad. Child Studies Ain Shams University	Prof. Head, of Department of Neurology Faculty of Mediine Al - Azhar University	Prof of Psychiatry. Faculty of Mediine Ain - Shams University

1997

« فتعالى الله الملك الحق ولا تعجل بالقرءان من
قبل أن يقضى إليك وحيه وقل رب زدنى علما »

صدق الله العظيم

(طه : ١٤٤)

Acknowledgment

Contents

LIST OF TABLES

I GENERALLY

1) PSYCHODEMOGRSAPHIC PROFILE

I-   COMPARISON BETWEEN GME, TLE & CONTROL
GROUPS CONCERNING:

2) ILLNESS PROFILE

II -   COMPARISON BETWEEN GME, TLE & CONTROL
GROUPS CONCERNING THE FOLLOWING :

3) THERAPEUTIC ISSUES

III-   COMPARISON BETWEEN GME, TLE & CONTROL
GROUPS CONCERNING THE FOLLOWING:

4) INVESTIGATIONS

A) PSYCHOMETRY

I) ASSESSMENT OF COGNITIVE

FUNCTIONS:

II) ASSESSMENT OF MOOD:

III) ASSESSMENT OF PERSONALITY:

B) EEG

II) IN DETAILS

1) PSYCHODEMOGRSAPHIC PROFILE

I - COMPARISON BETWEEN GME & TLE GROUPS CONCERNING THE FOLLOWING:

Socio-Economic Factors

2) ILLNESS PROFILE

II-   COMPARISON BETWEEN GME & TLE GROUPS
CONCERNING :

III-   COMPARISON BETWEEN GME & TLE GROUPS
CONCERNING:

        3) FREQUENCY

EXPLANATION OF ILLNESS PROFILE TABLES (156--85)

3) THERAPEUTIC ISSUES

IV - EEG

LIST OF FIGURES

Introduction

The central Agency for Mobilisation and statistics stated that the number of children ranging between 9-15 years of age are about 9,642,042 (16.26%) of the total population. (4,719,843 males and 4,421,900 females), according to the 1996 census.

The prevalence rate of epilepsy in a sample of Egyptian community was (12.9/1000), and it is higher in rural Areas (15.8/1000) than in urban anes (9.4/1000). (Tallawy et al. 1996).

So, the at-risk group to considered will constitute 124,382. It represents a great problem, as these children are the back bone of our community, and being epileptic is a danger which will affect their personality, cognitive functions and achievement.

Unfortunately there is no epidemiological study for epileptic children in age group between 9-15 years.

The prevalence was 4.2 in 1,000 in children aged up to 16 years in Sweeden country. **(Sidenvall, et al., 1995)**

According to Sweden estimation there is about 2-3/1000 of the Sweeden study concern the age of 9-15 years. This means that in Sweden there is about 12000-18000 child in this age group are susceptable for epilepsy

The importance of lightning the problem of cognitive dysfunction in epileptic children stemed from the fact that these group of children, who are considered to be the future productive & driving power of the society.

---

* Arabic References

Aim of the Study

This study is designed to identify differences between two groups of children affected with epilepsy.

Aim of the work was designed to answer a number of questions :

What are the profiles of cognitive dysfunction of Grand Mal Epilepsy compared to Temporal Lobe Epilepsy in a sample of Egyptian epileptic children ?

Is it possible to prevent cognitive deterioratioin in Egyptian epileptic children or to reduce its sequalae ?

What is the effects of antiepileptic drugs (AED'S) on cognitive and psychological functions ?

Does psychodemographic variables have contribution to cognitive and psychological day functions ?

It is possible to prevent personality deterioration in Egyptian epileptic children or to reduce its sequlae ?

(1)

- **Epileptic children; cognitive functions & Behaviour**
- **Cognitive functions**
- **Effect of Epilepsy on Memory**
- **Behaviour**

I- Epileptic Children: Cognitive function & Behaviour:

Overall. children with epilepsy have poorer concentration and mental processing and are less alert than age-matched controls. The relationship between cognitive functioning and epilepsy is complex. however. with widely differing degrees of intellectual impairment - ranging from minimal to severe and progressive - related to diverse types of epileptic seizures. syndromes. and etiological factors. Prolonged and frequently repeated seizures are typically associated with more severe effects on cognitive functioning. particularly if epilepsy is symptomatic i.e.. secondary to a demonstrable brain lesion. A combinationi of such factors may contribute to the mental deterioration seen in many children suffering from severe epilepsy. **(Dam 1990)..**

However. there are also reports of children with epilepsy having normal or higher than average mean scores on IQ tests which is not significantly different from control groups or their own siblings supporting the notion that a seizure disorder can be compatible with the normal range of intellctual abilities **(Cull 1988   ),**

Recently. attention has been drawn to the fact that IQ levels can fluctuate markedly in children with a seizure disorder. It is, therefore, important to bear in mind that. with respect to prognosis. a single assessment may be misleading. and a more reliable pattern of ability will be shown on repeated testing **(Bourgeois et al., 1983; Rodin et al., 1986).**

Cognitive impairment is regarded as the link between epileptic conditions and the inability to learn in school. The neuropsychological

approach to learning disabilities in epilepsy, therefore. first concentrates on analyzing the differential effects of epileptic factors on cognitive function. The impacy of seizure activity, localization of epileptogenic food and antiepileptic treatment on cognitive functioning can be evaluated based upon the results of continuous assessment with a computerized neuropsychological test system. Second learning disabilities may be evaluated on observations made during classroom performance. Three issues seem to predominate in learning studies among disabled children with epilepsy : test-retest variability, deterioration. and the supposed specificity of the learning disabilities. **(Aldenkimp et al 1990).**

Cognitive function :

Most children with epilepsy do not have learning disability but a small subgroup of children shows decreasing I.Q. because mental age does not increase at the same rate as chronological age. Our previous extensive studies have failed to show any relationship between I.Q. decrease and age of onset of epilepsy, total number of seizures. antieplieptic medicaion, sex, or initial I.Q.

These results suggest that if children with gross EEG abnormality are offered early treatment, cognitive slowing might be avoided in some cases. Effective treatment in all these conditions might avoid or limit permenant cognitive impairment. **(Fowler et al, 1995)**

Aetiologically :

The neuropsychological changes obserbed after right temporal lobe resections indicate a role in memory for nonverbal material by the

nondominant temporal lobe, but the relative contribution of the hippocampal subfields to this process has not been established.

These findings provide further evidence of a role of the right hippocampus (and possibly the left) in nonverbal memory and the presence of subfield specificity. **(Mat Kovic et al, 1995).**

Although patients with left hippocampal atrophy (HA) have predominantly verbal memory deficits, the relationship between degree of HA and severity of memory impairment has not been extensively investigated. The degree of right HA and severity of nonverbal memory impairment were not associated. There is a close association between left HA and verbal memory deficits. Intact nonverbal memory is not a reliable indicator of a normal right hippocampus. **(Kilpatrick et al, 1995).**

The effect of TLE on cognitive potentials apparently is not due to overall hippocampal atrophy and sclerosis affecting all effective Refractory periods (ERPs) equally, but instead may show material-specific differences with possible consequences for neuropsychological performance. **(Grunwald et al, 1995).**

The atieplieptic drug vigabatrin (VGB) increases brain levels of the major inhibitory neurotransmitter γ-aminobutyric acid (GABA) through inhibition of GABA transaminase, the enzyme which catalyzes the breakdown of GABA.The findings thus far show few cognitive side effects of VGB and suggest that the cognitive effects of barbiturates and

benzodiazepines are not the simple result of enhanced GABA nergic activity. **(Kimford 1995).**

Aetiologically, there are two groups of epilepsies. The 'idiopathic' or 'uncomplicated' epilepsies, where no cause can be established, and symptomatic epilepsies which are associated with organic pathology or complicated by other neurological problems.

In symptomatic epilepsy IQ scores are significantly lower than those of children with idiopathic epilepsy. Conversely, children with idiopathic epilepsies are more likely to conform to the expected normal distribution for intellectual ability *(Cull 1988).*

As a corollary to this, **Ellenberg et al. (1984)** have presented evidence that neurological status withing the first year of life, prior to the first seizure, may be an important prognostic indicator with respect to intellectual ability at seven years of age.

Thus, an organic aetiology for the seizures or a seizure disorder complicated by further neurological problems would appear to be a poor prognostic indicator for intellectual ability and schoolastic performance.

Seizure variable :

type :

In contrast to findings in adults, little is known about cognitive dysfunctions in children and adolescents with temporal lobe epilepsy. Therefore, we investigated whether the neuropsychological profiles of children were related to site of focus and determined which changes in cognitive performances resulted from partial temporal lobe resection.

(Lendt et al. 1995). found that LT patients showed a decrease in performance in verbal (50%) and visual memory (42%). RT patients showed mainly a loss in visual memory (38%). Preoperative profile and postoperative findings suggest global cognitive dysfunction in children and adolescents with LT impairment. which emphasizes the significance of the left hemisphere for children's cognitive development.

The relationship between seizure type and intellectual ability is unclear. However, some investigators have found no relationship between petitmal seizures and IQ. Children with grand mal seizures. psychomotor or partial seizures have also been found to be unimpaired on tests of intellectual ability.

In a direct comparision of different seizure types. found that children with partial seizures obtained better scores on some Wechsler subtests than did those with generalized (**Cull 1988**).

Some studies have found no difference in the degree of retardation with respect to seizure type **(Keith et al., 1955).**

Little in the way of a clear relationship between seizure type and poor attainments or educational underachievement has been reported although major seizures have been implicated but focal seizures have not **(Cull 1988**).

Age of onset :

By contrast, an early age of onset of the seizure disorder has more consistently been associated with lower IQ values.

*A long duration of seizure disorder has been negatively associated with IQ in three studies (**Cull 1988**   ).*

*An early age of onset and longstanding duration of epilepsy have also been implicated in learning difficulties (**Rodin, 1968**).*

Seizure frequency :

Partial seizures account for 40% of childhood seizures. Prognosis depends on association with definite cerebral lesions.

*Therapy with either CBZ or PB was equally effective in seizure control. (**Maria, 1995**).*

*With respect to seizure frequency, the evidence would support the thesis that frequent seizures may have a detrimental effect on IQ (**Cull 1988**   ).*

*The degree of seizure control would also seem to be important, good control being associated with higher IQ values. But again, there are conflicting reports (**Halstead, 1957**)*

*It would seem that attainments and learning patterns are not related to seizure frequency, although, again, seizure control may be important (**Cull 1988**   ).*

EEG :

*An abnormal EEG would seem to be associated with a low IQ. No clear relationship has been sestablished with respect to attainments and school performance (**Cull 1988**   ).*

Findings indicate that memory functions of the epileptic dominant hemisphere are weakly disrupted, whereas memory functions of the epileptic nondominant hemisphere are more strongly disrupted. Consequently, memory functions of the epileptic dominant hemisphere are relatively good, but memory functions of the epileptic nondominant hemisphere are very poor. **(Sang, 1995).**

Antiepileptic medication :

The differences in memory performance before and after valproate (VPA), pheytion (PHT), or carbamazepine (CBZ) discontinuation were not significant in any patient. We conclude that the memory effect of Phenobarbitone (PB) therapy was mostly transient. Effects of VPA. PHT. and CBZ require further study. **(Ming Xu et al, 1995).**

The results suggest that VGB exerts as slight. selective. and transient effect on cognitive functions. **(Czapinsk et al, 1995).**

Oxcarbazepine (OCBZ), the 10-keto analogue of carbamazepine (CBZ), has a different metabolic profile from the older agent, avoiding production of the pharmacologically active CBZ-10, 11-epoxide.

A preliminary report comparing the cognitive effects of OCBZ and phenytoin in newly diagnosed epilepsy showed no difference between the drug. OCBZ is a promising new antiepileptic drug that may have a bening effect of cognition at therapeutic dosage. **(Brodie, 1995).**

Sex :

The finding with respect to sex and intellectual ability are

conflicting. Early studies report a superiority for boys or no difference. whereas others have found girls to be superior.

There is however. some suggestion that boys are more likely to underachieve at school. and to be impaired with respect to reading skills (*Cull 1988*   ).

Family and environmental influence :

Environmental factors have received scant attention in the epilepsy literature. The few available studies suggest that parental attitudes and the reactions of a child's peer can influence the academic attainment and school performance of the child with epilepsy. ((*Cull 1988*   ).

Behaviour :

The major contributions of the behavioral approach to epilepsy has been (a) the recognition that external factors influence the probability of occurrence, the start, duration, intensity, and inhibition of the epileptic seizure; and (b) the teaching of the individual to identify these "risks" and learn to cope with these factors in a way that prevents or reduces risk for and even aborts seizure occurrence. **(Anne, 1995).**

Prevalence of behaviour disorder :

Twenty-four percent of patients were clinically anxious. 10% were clinically depressed. and approximately one fifth felt stigmatized by their epilepsy. Many side effects of antiepileptic drug treatment were reported.

with CNS side effects being the most common. Rates of employment and marriage were low as compared with the U.K. national average.

*Multiple regession analysis was conducted to establish the contribution of clinical and demographic variables (age, sex, duration, seizure type, frequency, and severity) to psychosocial functioning. **(Gus. et al, 1995).***

Factors affecting behaviour :

Aetiology :

*Organic pathology has been reported for the majority of children exhibiting behaviour disorders and large numbers of children with behaviour disorders have been found in brain-damaged populations **(Christine, 1988).***

*Single-gene partial epilepsies have recently been recognized in which affected individuals have homogeneous partial seizure semiology within syndromes such as autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE) and autosomal dominant rolandic epilepsy. We describe a single-gene partial epilepsy syndrome with different electroclinical partial seizure semiology in different individuals. **(Ingrid et al, 1995).***

Epilepsy variables :

Seizure type :

The relationship between behaviour disturbance and seizure type is

even less clear. An increased occurrence of behaviour disorder has been found to be associated with focal seizures, temporal lobe epilepsy; complex partial seizures; grand mal seizures; and petit mal seizures **(Christine, 1988).**

Behavioral disorders (apart from peri-ictal phase), especially aggression, have been observed in adults and children with temporal lobe epilepsy (TLE) in about one third of cases.

Fifty-eight percent of the children with behavioural distubances showed improvement of their psychic disorder after surgery. All these patients remained seizure-free. **(Kowalik et al, 1995).**

Age of onset :

There is increased rates of behaviour disturbance with early age of onset, there are conflicting reports concerning the role of duration of the seizure disorder **(Christine, 1988).**

Seizure frequencey :

Positive associations between seizure frequency and behaviour disturbance have been reported in some studies **(Christine, 1988).**

The possibility of psychological intervention for the management of seizures seems promising, especially for patients whose seizures are not controlled by medication.

Seizure reduction was maintanined at 4 and 8-week follow-up, suggesting that group-based psychological intervention can be beneficial for the managment of epileptic seizures. **(Spector et al, 1995).**

EEG :

Significant clinical benefit was manifested in good control of epileptic seizures, especially generalized and partial complex seizures and epileptic status with abnormal EEG in 60% of patients. Improvement in cognitive functions and psychological disturbances was achieved in 85% of treated children. The psychotropic effect of Tegretol CR was effective in periodical psychotic and behavior disturbances in epilepsy. There was no incidence of adverse reaction, and all children tolerated the drug well. **(Ignatowicz et al, 1995).**

Antiepileptic medication :

Drug-related Antiepileptics (AE) were observed consisting mainly of irritability, hyperactivity, behavioral disturbances, and drowsiness. **(Gherpelli et al, 1995).**

VGB was effective in achieving both excellent seizure control and behavioral improvement. vigabentine (VGB) was well tolerated; **(Dana et al, 1995).**

Phenobarbitone, phenytoin, and sodium valproate have all been implicated in behavioural disturbance. However, sodium valproate has also been suggested to exert no significant effect on behaviour, and beneficial effect have been reported for phenytoin. Carbamazepine has been associated with considerable improvements in behaviour; however, a small number of children may show idiosyncratic negative reactions (*Cull 1988*).

(Harry 1995) *found in the original data from my work with felbamate, noting its relation to symptoms of depression, psychosis, and apathy syndromes, and review of the literature on the early data available on behavioral manifestations related to the use of lamotrigine, vigabatrin, gabapentin, oxcarbazepine, zonisamide, flunarizine, stiripentol, tiagabine, and other AEDs will be reviewed. Clinical implications, theoretical mechanisms of action.*

Sex :

No significant difference between the number of boys and girls exhibiting behaviour disturbance has been reported in some studies.

Aggressive, antisocial behaviour disorders have been associated with boys, whereas neurotic disturbances are found more frequently in girls (*Cull 1988* *).*

Family and environmental influences :

We believe close and rich communication between many families and hospital staffs and sharing the diffculties with which their families are dealing is necessary, particularly when patients have behaviroal difficulty in addition to seizure disorders. ***(Teruyo et al, 1995).***

Limited physical activity and fitiness probably result from factors such as fear of inadequacy, overprotection or insufficient information and might seriously influence the general health and quality of life. ***(Bernhard et al, 1995).***

Music therapy :

Music therapy provides a social relationship contained within therapeutic boundaries. In this presentation, we assess at the interaction of music therapy with a child with intractable epilepsy who has developed a series of ritualized behaviors. We trace the music therapy sessions in a 12-month period and observe music as the link between the child and the therapist which gives him the security to alter slowly some of his behaviours. **(Kate et al. 1995).**

Behaviour and cognitive Function

There seems to be a clear link between poor cognitive function and behaviour disturbance. Thus low IQs are more likely to be associated with behaviour disorder. similarly for poor school performance.

Thus. it can be tentatively concluded that intellectual ability may be influenced by the presence of brain damage. factors related to the epilepsy itself and an abnormal EEG. and may be exacerbated by anti-epileptic medication.

Poor attainments and scholastic ability are accounted for in part by a low IQ, but there remains a significant number of children of good intellectual ability who are underachieving.

Aetiology, factors related to the epilepsy itself, EEG abnormality, gender and medication, may all have a role to play in the genesis of behaviour disorder. (***Cull 1988***).

Family activity in new-onset Epilepsy :

Children with epilepsy have more social problems and participate in fewer social activities than their peers. Increased parental supervision

and decreased family leisure activities have been hypothesized to contribute of these social problems.

Reduced child activity was related to seizure frequency (chi square = 10.5, p < 0.01) but to parent supervision of family leisure activity. **(Joan et al, 1995).**

Effect of Epilepsy on Memory

In general, the literature suggests that epilepsy is associated with an increased risk of cognitive problems, especially memory dysfunctions. We examined memory functions in patients with epilepsy who all complain of memory problems in daily life.

The relationship between memory tests and epileptogenic factors such as etiology, age at onset, number of years with seizures, seizure frequency, and antiepileptic medication was explored. **(Hendriks et. al, 1995).**

Factors Influencing Memory Disorders in Epilepsy :

1- brain damage :

In patients with left (dominant) temporal lobe epilepsy (TLE), a typical neuropsychological finding is impairment of verbal long-term memory. Epilepsy surgery, however, also carries the risk of inducing additional neuropsychological deficits, however, we noted that the postoperative neuropsychological outcome with regard to delayed free recall capacity can be predicted accurately by the corresponding negativity of the right medial temporal lobe ($r= 0.83$, $p < 0.0005$). **(Elger et al, 1995).**

These results argue that the hippocampal region plays a more important role than the entorhinal cortex in human memory. **(Miller et. al, 1995).**

2- *The frequency of seizures :*

The frequency of seizures does play a role, both directly and indirectly. Directly, because seizures with loss of consciousness disturb mental functioning, not only duing the seizure but also afterwards, sometimes for several days.

An ongoing process may occur in chronic epilepsy because of cerebral damage due to repeated seizures.

Decreased performances are correlated with increasing discharge rates, or length of the discharges, and generalized discharges are worse than focal discharges, (Pierre et al 1988).

Glowinski (1973) considered that subictal electrical discharges in temporal lobes disturb memory functioning by directly interrupting memory storage.

3- *Duration of fits :*

A long duration of epilepsy impairs memory functioning. Ten years appears to be a crucial ength. In fact, a long duration generally means seizures repeated during many years, and a prolonged therapy. So neither the duration nor the frequency is an independent variable. (Pierre et al 1988).

4- *Age at onset*

An early onset is a recognized deleterious factor. Besides childhood is a very important period for learning, a period during which one learns how to learn. (Pierre et al 1988).

5- EEG and clinical epilepsy

Considerable evidence has confirmed a complementary specialization of the temporal lobes for information processing and retention in man.

According to this hypothesis, early isolated cerebral lesions of the LH will strongly influence cognitive functions of the RH, whereas isolated lesions of the RH do not influence LH functions to the same extent due to a lesser reorganization capacity of the LH. **(Bölling et al, 1995).**

Anticonvulsant drugs

Cognitive effects are sometimes the effects of chronic treatment, but most of our knowledge is produced by normal volunteer studies that use periods of drug exposure varying from 1 day to 1 month. **(Aldenkamp, et al, 1995).**

Patients with toxic levels perform worse than patients with non-toxic levels (Matthews and Harley, 1975).

Phenobarbital, phenytoin and primidone intake is clearly an important factor responsible for memory impairment in epileptic patients.

Sodium valproate and carbamazepine are certainly less toxic **(Dodrill et al.1977).**

Results suggest that VGB may be considered a first-choice drug in focal epilepsies with CPS and a valid alternative when other monotherapies are ineffective or poorly tolerated. **(Paolo et al, 1995).**

Studies of the cognitive effects of tiagabine (TGB) have already been completed in three countries. The world-wide data collected to date

*suggests that TGB offers relief from seizures without significant cognitive side effects. (**Dodrill, 1995**).*

*However, anti-epileptic drugs cannot entirely account for the defect in memory noted in epileptic patients. (**Pierre et al 1988**).*

Emotional state and Memory :

*A patient's emotional state of motivations may alter memory performance (**Lishman, 1972; Hirtz et al. 1985**).*

(2)

Relation between childhood epilepsy and cognitive deterioration.

Relation between childhood epilepsy and cognitive deterioration :

The number of children with epilepsy who experience difficulties at school is significantly greater than in their non-epileptic peers.

On the other hand, their performance is no worse than that of children with learning problems or of behaviourally disturbed children

Impaired performance is seen particularly on tests of reading, spelling, arithmetic and word recognition **(Seidenberg et al., 1986)**, but not on design copying **(Ross et al.1978)** or tests of a creative rather than academic orientation **Ross, 1973.**

Poor performances have been reported on tests of psychomotor function, even in the presence of a normal IQ. By contrast no differences with respect to memory functions have been reported in comparision with matched control groups.(*Cull 1988* ·).

A useful framework for examining childhood epilepsy and cognitive deterioration is provided by confsidering the following questions. 1. Is epilepsy associated with deterioration ? 2. Does epilepsy cause deterioration ? 3. Does deterioration cause epilepsy ? 4. Do both epilepsy and deterioration reflect some underlying causative brain abnormality ? 5. When epilepsy and cognitive deterioration occur together, what factors might be important ? *(Besag, 1988)*

Most of these quesion can be answered through the follwoing conclusions .

1. Most children with epilepsy are of normal intelligence and their intellectual ability does not deteriorate.

2. Because of this. group epidemiological studies are unlikely to demonstate any effect of epilepsy on intellectual functioning.

3. A small sub-group of children who have epilepsy do deteriorate intellectually.

4. Some of the children with epilepsy who deteriorate have recognized preexisting brain damage but some do not.

5. A bsence seizures. unaccompanied by seizures of other types. probably have a good prognosis. Infantile spasms and the Lennox-Gastaut syndrome generally have a poor prognosis for future intellect. Mixed epilepsy, when several seizure types occur together also probably has a poor prognosis.

6. Status epilepticus can undoubtedly cause intellectual and neurological deterioration.

7. There is a possibility that phenytoin causes a chronic encephalopathy with intellectual deterioration.

8. Whenever a child deteriorates intellecually. with or without seizures, the possibility of cerebral pathology must always be considered. e.g. neurodegenerative disease brain tumour.

9. In those children with epilepsy who do deteriorate. although there is conflicting evidence, the literature suggests that early age of onset, frequent seizures, prolonged seizures. association with pre-existing brain damage and mixed seizure type may all contribute to the intellectual deterioration. (*Besag, 1988*)

(3)

Anxiety, Mood changes and Epilepsy.

Anxiety and Mood changes in Epilepsy :

Anxiety :

Anxiety and depression are the most common psychiatric conditions associated with epilepsy. Understanding the development & maintenance of these symptoms requires investigate of interrelations between biological and psycholocial factors. They have attemped. using a multietiolocial model, to investigate certain Cognitive mediators that may be movwed **(John et al., 1995)**

Depression :

Depression has been associated with epilepsy since some of the earliest medical recordings.

Depressive mood disorders are often associated with epilepsy, and the severity of depression may seriously influence the prognosis of the disease, thus increasing the risk of suicide. Therefore, anti-epileptic drugs (AEDs) combined with anti-depressants (AD) are often routinely administred clinically. Determination of plasma levels of some of these drugs has allowed evaluation of the kinetic modifications in the course of such combined therapies, with doses adjusted in case of subtherapeutic or toxic levels. **(Monace et al., 1995).**

Peri-Ictal Depression

Characteristically, the alteration of mood lasts longer than an aura or postictal automatism, and it can have dangerous consequences, as evidenced by **Betts (1982)** who reported a patient who cut his throat during an ictal depressive state.

Prolonged depressive mood swing have been recorded during status epilepticus *(Well, 1975; Lim et al., 1986)*.

Interictal Depression

No studies have directly addressed the question as to whether depression occurs more frequently in people with epilepsy compared to other conditions, but conclusions may be drawn indirectly from the results of investigations using standardizes rating scales. Thus, many studies employing the Minnesota Personality Inventory (MMPI) have indicated that depression scores are raised in people with epilepsy, with special reference to those with psychomotor/ temporal lobe epilepsy *(Mary, 1988)*.

Postictal depression is a complex phenomenon generated by more than one inhibitory system. *(Mares, 1997)*.

Depression is more common is people with epilepsy (PWE) as compared with control groups. Depression is associated with psychosocial complications of epilepsy (increased life events, poor adjustment to seizures, financial stress), but is not, by and large, correlated with epilepsy variables such as age of onset of epilepsy or seizure type or frequency. Antiepileptic drugs (AEDs) on the other hand have a marked effect on mood. Phenobarbital has been implicated in the emergence of both depressive symptomatology and suicide, and a similar association has been shown with vigabatrin.

*Of a series of psychiatric symptoms an reactions. only depression seems to predominate in idiopathic and cryptogenic epilepsy and can be attributed to epilepsy per se. **(Bogliun, et al., 1997).***

Mood disorders occur frequently in people with epilepsy, but this applies predominantly to anxiety, depressive symptomatology and depressive illness.

*With regards to depressive symptoms. several studies using standardized rating scales have found the depression scores to range from moderate to severe and to compare favourably to studies on depressed patients without epilepsy. **(Mary 1988).***

(4)

- The effect of anticonvulsant drugs on mood and cognitive functions.

- The effect of type of therapy on cognitive function.

- The effect of type of therapy on behaviour changes.

I- The effect of Anticonvulsant drugs on mood and cognitive function

In recent years that patients with epilepsy may suffer from cognitive disruption or behaviour distubances has become accepted.

The effect of type of therapy on Cognitive Function

Monotherapy

Patients were given psychological testing on a battery which included memory, attention and concentration. perceptual speed. decision making speed and motor speed tasks.

Experimental and clinical data suggest that lamotrigine (LTG) is relatively nonsedative and that it has positive effects on cognition and affect. **(Colin 1995).**

The potential cognitive effects associated with gabapentin (GBP, Neurontin) administration were studied in patients with partial seizures in a double-blind, dose-controlled, multicenter clinical trial evaluating conversion to GBP monotherapy and in an ongoing open-label extension study. **(John 1995).**

Sabril (bigabatrin, VGB) (Marion Merrell Dow) (500-mg tablets) is a promising new anticonvulsnat drug specifically designed to enhance γ-aminobutyric acid (GABA) function in the CNS.

Some patients who became seizure-free after the addition of Sabril

could subsequently taper or discontinue other antiepileptic drugs. There was evidence of adverse reactions during treatment. and tolerance was good in all children. *(Ignatowicz et al., 1995)*.

They evaluated safety and efficacy of Depakote (divalproex sodium. VPA) monotherapy in the treatment of patients with complex seizures (CPS) administred to achieve high (80-150 µg/ml) total trough plasma concentrations of VPA. with a low VPA concentration group (25-50 µg/ml) as active control. *(Chris 1995)*.

They used monotheraphy with valproate (VPA) for initial treatment of newly diagnosed epileptic patients with partial seizures. simple or complex, and primary or secondarily generalized seizures. They had received VPA (Convulex) 900 mg/day in divided doses.

VPA monotherapy is a good choice for initial treatment because it has a wide spectrum of activity. *(Johan et, al, 1995)*.

Andrewes et al. (1986) compared new referrals with epilepsy, well controlled on single drug therapy, with either phenytoin or carbamazepine and an untreated control group with respect to their performance on a number of cognitive tasks.

They noticed significant differences between the groups in favour of carbamazepine was reported to have the least effect on a number of tasks including finger tapping, colour naming, a Peg board test, a digit symbol substitution test, and discriminative reaction time. Phenytoin and phenobarbitone provided the worst scores, and correlations between deterioration and serum levels were noted *(Smith et al., 1985)*.

There are differences in the behavioural toxicity profile of the anticonvulasant drugs with regards to their impact on congnitive function. In all of the studies carbamazepine comes out as the drug with the least impact, while phenytoin, and where studied clonazepam, appear to have maximal effect. **(Michael et al., 1988)**.

Polytherapy :

Although individual drugs may thus have a different impact with regards to cognitive function, polytherapy itself may be an important variable.

Lowering the anticonvulsant prescription load of children improves cognitive performance, while increasing it has a deleterious effect on learning abilities.

Vigabatrin (VGB) has been tested in several clinical studies and to date appears to have no demonstrable effects on cognitive function despite its powerful anticonvulsant effect.

A difficulty of interpretation is the number of variables that may influence cognition, such as polytherapy and seizure frequency, for which these studies have not been able to control. In this study, healthy volunteers were examined for the effect of VGB on cognitive function and mood to assess the effect of the drug in the absence of such contaminating factors. **(Trimble et al., 1995)**.

The seurm valproate (VPA) concentration and the clinical effects of polytherapy with other antiepileptic drugs [phenobarbital (PB), clonazepam, diazepam, clobazam (CLB), and ethosuximide (ESM), were

estimated. VPA serum levels were reduced when it was combined with PB. CLB administered together with VPA led to an increase in the serum concentration of the CLB. VPA serum levels showed no significant changes when VPA was combined with either ESM or 1-4 benzodiazepines. The best therapeutic effects were noted after polytherapy with VPA, ESM, and CLB in primary generalized seizures. **(Sobaniec et al., 1995).**

II- The effect of type of therapy on Behaviour changes.

Monotherapy :

The behaviour of more patients deteriorated on phyenytoin, and improvements were seen with both phenobarbitone and carbamazepine.

With regards to the relationship of individual drugs to mood, it is interesting that there at least four studies in epileptic patients showing a significant negative relationship between serum levels of carbamazepine and scores on a mood or behaviour rating scale.

VGB appears to be an effective and safe antiepileptic drug as primary monotherapy, with fewer cognitive side effects than CBZ. **(Reetta et al., 1995).**

All patients reported that their epilepsy had improved during the study period; this opinion was shared by the physicians of 20 patients. Results support the concept that successful pharmaco therapy has both socioeconomic consequences and a positive impact on the patients life. **(Ylinen, et. al, 1995).**

Lamotrigine (LTG) is a new antiepileptic drug (AED) chemically unrelated to the currently used AEDs. It has proved efficacious in treating patients with partial seizures and generalized tonic-clonic seizures (GTC)s not satisfactorily controlled by other AEDs. **(Severi et al., 1995).**

Polytherapy :

Reports by **(Shorvon et al., 1979),** suggest that rationalization of polytherapy brings improvement of mood.

Rationalization of polytherapy showed :

Significant improvements on rating scales of mood in particular for anxiety and depression. Following the carbamaxepine substitution, patients rated themselves as less anxious and more lively during the follow-up period. With regards to depression.

When patients were divided into those with high or low initial (prechange) depression scores, those with the higher scores showed significant improvements following the change to carbamazepine. **(Mary, 1988).**

Discontinuation of Antlepleptic drugs :

As to EEG findings, remaining paroxysmal discharges did not necessarily mean the occurrence of relapse, but the changes in background acivity with age, which may indicate CNS maturation, were significantly different between the patients with or without relapses. Our results suggest that the factors related to age should be considered

according to the epileptic syndrome when one decides to discontinue AED treatment. *(Tohru et al., 1995).*

Immune Response and childhood convulsions :

Immune response did not correlate with clinical evolution: moreover, difference were not observed between idiopathic and symptomatic epileptic children. *(Rancci et al., 1995).*

Newly diagnosed patient :

VGB may be considered a first-choice drug in newly diagnosed partial epileptic patients, owing to its good efficacy and tolerability profile. *(De Feo, et al, 1995).*

Refractory epilepsy :

We noted no side effects of VGB. The children were continued on combination therapy without alteration in previous anticonvulsant drugs. *(Koul, et al, 1995).*

Efficacy of VGB analyzed by seizure type showed 66% of patients with complex partial seizures to have complete remission. Our results indicate that VGB has significant antiepileptic activity when added to the standard therapy of some children with intractable epilepsy. *(Matinez, 1995).*

TPM is a sate and valuable AED in treating some patients with refractory partial seizures. *(Kugoh, 1995).*

Status tpilepticus :

Status epilepticus (SE) demands a prompt and efficient treatment. Overall, with standard treatment, 50-80% of all patients with SE will respond to an appropriate therapeutic workup; the rest represent so-called refractory SE (RSE), with severe consequences either for the RSE itself or the pharmacological risk of the more aggressive therapy which is required. **(Gonzalez, 1995).**

Surgery :

"Cerebral plasticity" has been suggested to allow better compensation for epilepsy surgery excisions if performed early rather than later in childhood. The findings suggest that after temporal lobe surgery, with these pathologies and good seizure outcome, few children have cognitive deficit and the likelihood of this is not reduced by early surgery. **(Oxbury et al., 1995).**

The effects of anticonvulsants on cognitive function and behaviour can be summerized in the following :

1) Behaviour : Carbanazepine pheytion and sod ralpment have minimd effect and phembarbition. clon a zepam / impeir behaviour.

2) Gognitive functions : Carbamazepine and phenobarbition have a minimized effect but clonazepam and phenobarbiton impair functions to a greater extent. **(Michael, 1988).**

With regards to congnitive function. most studies contrast the more detrimental effects of phenytoin with the least detrimental effects of carbamazepine, drugs such as sodium valproate appearing to hold an

intermediate position. Phenobarbitone and clonazepam are more likely to be associated with the phenytoin spectrum of activity, although further investigations of these drugs are warranted.

In contrast, with the behavioural studies, carbamazepine can be more effectively contrasted with phenobarbitone. Several studies, and many reports, suggest that carbamazepine is psychotropic, while phenobarbitone may exacerbate or even precipitate affective disturbances in adults and conduct disturbances in children. The relationship to serum levels with regards to the behavioural profile of carbamazepine is striking **(Michael, 1988).**

Even stronger evidence for the psychotropic effects of this drug emerges from the growing literature of its effective use in non-epileptic patients with bipolar affective disorder who receive carbamazepine, either acutely in the management of mania, or for the prophylaxis of mainc depressive illness **(Post, 1985).**

(Jinggang Yin, et al, 1995). compared cognitive function in 56 new epileptic patients well controlled on single-drug therapy with either phenytoin (PHT,23) or carbamazepine (CBZ, 16) with that in an untreated control group (17).

There were no significant differences between the groups in terms of age, I.Q., seizure type, duration of epilepsy, duration of treatment, or duration of seizure freedom. Serum levels of each drug were measured immediately after testing by high-performance liquid chromatography. Signicant differences between the groups in favor of CBZ were noted for each of the memory tasks. Comparison of the two drug groups on word

recall test again showed a poorer performance of the PHT group. In the list-learning task, the CBZ group showed a trend to learn more rapidly than did the PHT group during the first four trials.

No significant difference, were noted between the groups in the decision-making task or the tracking task. The mood ratings were not significantly different between the groups. Higher CBZ levels were related to low rating for anxiety, depression, and fatigue. The results of the cognitive effects of PHT and CBZ in new-onset epilepsy patients receiving single-drug therapy are in keeping with the findings in chronic epileptic patients, and normal volunteers, these findings have implications for the choice of drug in the management of epilepsy and also for the reported claims of a psychotropic effect of CBZ. *(Jinggang et al., 1995).*

Although it is not suggested that all of the cognitive and behavioural problems that patients with epilepsy suffer from are related to anticonvulsant drugs, and number of other variables are urgently in need of further investigation in their own right, it is suggested that these data have importance for the management of epilepsy, particulary when patients or their relatives complain of such problems. *(Michael, 1988).*

(5)

Antieplieptic   drug   and
performance.

Antiepileptic Drugs and Performance

Patients receiving carbamazepine (Tegretol) had more marked changes in different EEG parameters as compared with patients receiving valproate. Among seizure types, patients with only generalized seizures tended to show fewer alteration than patients with partial seizures. **(Mihaly et al., 1995).**

There was higher incidence of significant side effects with carbamazepine versus vigabatrin (VGB). **(Piattella et al., 1995).**

Further studies of monotherapy in newly diagnosed patients with any types of seizures is recommended for better definition of the efficacy and safety profile of VGB. **(Herronz, et. al, 1995)**

These results prove the efficacy of VGB monotherapy in childhood partial epilepsy as initial treatment even in further randomized, double-blind, controlled clinical trials are necessary. **(Gobbi, et. al, 1995)**

Significant impairments in memory tasks and attention were poted, with worsening observed after anticonvulsant therapy. Factors such as type of epilepsy, frequency of and age at onset of seizures adversely affected the cognitive function of the patients. In addition, results of the Epilepsy were standardized with the common psychometric test, i.e., Wechsler Adult Intelligence Scale. **(Ogunrin et al., 1995).**

Continuation of antiepileptic drugs :

Cognitive function is frequently impaired in children with epilepsy, compared with age-matched controls. It can be hard to evaluate the significance of various contributory factors. The effects of antiepileptic drugs may be studied in children who have outgrown their epilepsy but are still being treated.

A multicenter study to assess various aspects of cognitive function in children with different forms of epilepsy, both during and after treatment with antiepileptic drugs, is currently under-way. Definitive results are not yet available; interim analysis of the findings suggests that short-term memory is decreased in all subgroups of children being treated for epilepsy, compared to controls. **(Blennow et al., 1990)**.

AED's with cognitive function :

The effects of antiepileptic drugs (AEDs) on cognitive function and behavior in children are reviewed on the basis of published studies. Individual AEDs have been shown to differ-the deleterious effects of phenytoin generally contrasting with the relatively minimal effects of valproate and carbamazepine. Some of the differences between results may be attributed to the psychological tests used and to age differences. However, there appears to be a dissociation between AEDs that affect higher cognitive function, e.g., phenytoin, and those mainly affecting motor functioin, e.g., carbamazepine, which appears to increase speed of performance. AEDs should be prescribed with care in children with epilepsy, taking account of their differing effects on cognitive fuction and behavior **(Trimble, 1990)**.

The ability to examine a large number of patients with well-documented and classified seizures who were relatively drug-free is a unique feature of this study.

When the raw data were examined, no dramatic differences in the performance of patients taking Carbamazepine (CBZ), Phenobarbitone (Pb), Primidone ()PRM) or Phenytoine (PHT) were found. As expected, the barbiturates fared somewhat less well overall than did CBZ or PHT, but even this difference rarely reached statistical significance. When the results of all of the subtests were combined in the total BTB score, significant differences between the four drugs emerged. CBZ consistently had less effect on cognitive function as measured by this test battery than Pb, PHT, or PRM at all rating periods, this finding confirms the results of other studies which have indicated that CBZ has fewer cognitive effects than PHT or the barbituates. Perhaps more importantly, this study emphasizes the need to control for age, education and IQ when assessing individual neuropsychological test results.

*Even though the total toxicity battery did reveal some differences in the cognitive effects of these four drugs, the most striking finding is that there were so few meaningful differences in the individual subtests. It is likely that when patients are managed by experienced 'epileptologists', adverse effects of AEDs are minized. This combined with a study design which encouraged physicians to lower the dose when 'unacceptable' toxicity intervened resulted in a very different subject population (**Dennis, 1988**).*

(6)

Childhood Epilepsy and
education.

Childhood Epilepsy and Education :

Learning difficulties in children with epilepsy may be caused by brain damage and should be investigated. In many cases, however, seizures and/or electroencephalographic (EEG) findings are the only signs of pathology. Frequency and type of seizures may be determining factors that should, if necessary, be evaluated by long-term EEG monitoring, preferably during school performance or in conjunction with neuropsychological assessment. This may prove that subclinical epileptiform discharges in the EEG can adversely affect the child's performance. Secondary psychological problems in epilepsy patients, combined with side effects of antiepileptic drugs, may cause or heighten learning problems. Prophylactic control of seizures with one appropriate drug may alleviate learning problems. **(Henriksen, 1990).**

School aged epileptic children :

* The over all prevelence of epilepsy 9at least two now febrle seizures after first week of life) in school-aged children in the UK is of the order of 4-5 children per 1000.

* A further 2 per 1000 children have been babelled as having epilepsy by at least one doctor but there is reasonable doubt concerning the reliability of the diagnosis.

* a bout half the children with epilepsy have not had a fit in the past two years. Their epilepsy is quiescent as active epilepsy. **(Euan et al., 1988).**

Computerized neuropsychological testig with simultaneous EEG recording may reveal the influence of epileptifrom discharges on cognitive fuctioin and also help to evaluate the effects of antiepileptic drugs **(Henriksen, 1990).**

School Role :

Some parents wish to keep their child's epilepsy a secret. This poses a potential danger to the child and needless problems for school staff if they have not been told and prepared for seizures to occur. Doctors must encourage the parents to share the problem with the school.

It is vital that all paediatricians have had on appointment a substantial element of paediatric neurology and child psychiatry n their training in order to bring a wide experience in the diagnosis and treatment of handicap to the school health service.

Optimal care is most likely when parents, teachers and older children have a clear understanding about the nature of the child's particular form of epilepsy, the rationale of treatment and prognosis. This can only be imparted by doctors.

Periodic re-examination and ressessment are needed whilst the seizure tendency is active. **(Euan et al., 1988).**

Information to the school and the parents concerning the patient's abilities and limitations may be as important as seizure control. Specialized teaching should be started early, when necessary, with the patient integrated into a normal school if possible. However, good

*functioning in a special school is preferable to marginal functioning in a normal school (**Henriksen 1990**).*

Temporal lobe seizures are typically manifest by a short outburst of abnormal

Uncoordinated behaviour, clinically typical cases can be found despite lack of characteristic EEG changes. Affected children usually function in the mentally normal range but may develop bizarre defence mechanisms that try the patience of their parents, teachers and friends; close cooperation between paediatrician and child psychiatrist is needed.

Currently four drugs are widely used to treat epilepsy in children :

*Primidone, a relatively old drug, avriant of phenobarbitone which appears to cause less irritability. Sodium valproate, as a general purpose anticonvulsant, particularly effective in petit mal and grand mal seizures. Carbamazepine was initially promoted as a treatment for temporal lobe epilesy though is now regarded as valuable in most types of childhood epilepsy, apart from classic petit mal. Clonazepam is a long-acting benzodiazepine; the fact that it has tended to be reserved for more obscure and difficult forms of seizure disorder has limited practical experience in its use as a first-line anticonvulsant in straightforward cases. (**Euan et al., 1988**).*

Objective assessment of subclinical epileptiform activity makes it easier to treat the pathology identified by the EEG with optimal dosage of the most appropriate drug. A balance is required because epileptiform

discharges and even occasional seizures may be less disabling than side effects from large doses of several drugs *(Henriksen 1990).*

Learning disabilities in Epilepsy :

Subclinical generalized spike-wave discharges are often accompanied by transitory cognitive impairment, demonstrable by psychological testing during EEG recording. Transitory cognitive impairment is demonstrated most readily by difficult tasks and during generalized regular spike-wave bursts lasting for more than three seconds, But can also be found during briefer and even focal discharges. That this is not simply a consequence of global inattention is shown by the fact that focal discharges exhibit some specificity: left-sided focal spiking is more likely to produce errors on verbal tasks, for instance, whereas right-sided discharges are more often accompained by impairment in handling nonverbal material. Both learning difficulties in general and specific abnormal patterns of cognitive fuctioning are well documented in children with epilepsy and are most pronounced in those with frequent interictal discharges. However, there is now evidence that intermittent cognitive impairment due to the discharges themselves contributes significantly to such neurophysiological abnormalities. The significance of transitory cognitive impairment accompanying subclinical EEG discharges for everyday functioning is uncertain, but there is experimental evidence that subclinical discharges may be accompanied by disruption of educational skills in children or by impairment of driving performance in motorists. In some individuals, suppression of discharges by antiepileptic drugs has demonstrably improved psychological function, but further work is required to determine the indicatioins for such treatment. *(Binnie, 1990).*

(7)

- **Assessment of cognitive function in epileptic patients (Methods and problems).**

- **Assessment of Behaviour disorders in epilepsy.**

I) Assessment of cognitive fuction in epileptic patients Methods and problems.

The methods used in assessing cognitive fnctions have undergone a considerable development from the early days, when the estimates were based solely on clinical impressions. The development of intelligence tests and neuropsychological test batteries, and the construction of memory tests and tests of specific cognitive fuctions, have given the clinicians and researchers a wide selection of tools. Furthermore, developments in the field of microcomputers have stimulated the construction of compurter-based tests which can be efficiently administered and scored. **(Jolanta, 1988).**

Some of the most frequently utilized tools in cognitive assessment of epileptic patients and to discuss the problems encountered in the interpretation of various studies will disscussed here. The assessment in three areas will be considered : intelligence, memory and specific cognitive functions.

i- Assessment of Intelligence :

Since the 1950s, the Wechsler scales, the WAIS and the WISC, have been the most commonly employed instruments for the assessment of intelligence in patients with epilepsy.

Intellectual level :

Since the early decades of this century the question has been asked whether the IQ of people with epilepsy was different from those of the normal population. **(Jolanta, 1988).**

*Children with epilepsy have been found to have a similar IQ to their non-epileptic siblings. although there was more variability in IQ and a higher rate of mental retardatioin among the patients. Both these differences disappeared when children who were impaired since infancy were excluded **(Ellenberg et al., 1986).***

*There is a wide range of abilities among epileptic patients, which extends between mental retardation and very superior level of fuctioning. Therefore, attempts to answer a general question about intelligence level in epileptic populations are not well directed **(Jolanta, 1988).****

Intellectual deterioratioin :

*Studies done by **(Lesser et al., 1986)** have indicated that. in general, no deterioration of intelligence occurs in a majority of patients with epilepsy except for certain groups. Rodin found a slight but significant fall in Performance IQ for patients with initially high IQ, and those with high seizure requency. Bourgeois found no deterioratioin of IQ in a large group of children with epilepsy except in a small subgroup of patients. These patients has a higher incidence of toxic serum drug levels, earlier onset of seizures and poorer control of seizures. Control of seizures was found to be associated with IQ in a study by **(Rodin et al. 1986),** who noted a stable or increases IQ for patients in remission, but a decrease in IQ or patients with poorly controlled epilepsy. These changes were small, however, and not statistically significant. There have been reports of a marked deterioration of IQ in over 15% of residential school patients and in patients with a history of status epilepticus or a cosiderable lifetime number of tonic-clonic seizures **(Jolanta, 1988).***

*They are two major problems with assessing changes of intelligence over time. One is associated with the relative dependence of IQ scores on attained educational level. **(Dikmen, 1980)** found that among his patients. those with early onset epilepsy showed significant decreases on the WAIS, but he pointed out that these results could also be related to differences in years of schooling. The educational differences. in turn. could have either a cognitive or psychological basis. for example. **(Long et al., 1979)** found that parents of epileptic children have diminished expectations for their child's academic performance. The IQ deterioratioin might not be linked causaly to epilepsy, but to the psychosocial consequences of the disease.*

*The other problem stems from the practice effects of repeated IQ test. Studies of recent outcome of the WAIS show a tendency for IQs to rise on the second testing, particularly for the Performance IQ, where the gains can be quite large **(Matarazzo et al., 1984).***

Lateralizing implication of Verbal-Performance IQ discrepancy:
*It is widely assumed that a differential perormance on verbal versus nonverbal (Performance) tests on the WAIS reflect the lateralization of the lesion or focus of seizure discharge. Patients with left hemisphere lesion or focus are expected to show a relative impairment on the Verbal IQ score while conversely, patients with right hemispheric lesion or focus will have impaired performance IQ relative to their Verbal IQ, for this reason the Wechsler scales have been used extensively in patients with temporal lobe epilepsy (TLE). The laterality effects of Wechsler scores in temporal lobe patients were found in some studies but not in others **(Jolanta, 1988).***

On the whole, however, the evidence about lateralizing implications of the Wechsler scales is weak and not consistent for the following reasons. First, many so called non-verbal tests require verbal-symbolic function for their solution, therefore impaired Performance IQ score may be due either to impaired left or right hemispheric function. Secondly most of the Perormance subtests are timed whilst Verbal tests are not. Any factor which affects the speed of mental processing, such as a lesion, irrespective of its location, EEG abnormality, or antiepilptic medication may result in a lower Perormance than Verbal IQ score. Thirdly, according to wechsler's own evidence, the relative balance between Verbal and Performance IQ depends on the overall intelligence level *(Jolanta, 1988).*

The importance of IQ test :

Besides using intelligence tests to assess a patient's general level of functioing, psychologists usually explore the pattern of subtests' scores for possible specific deficits. For example, poor performance on Digit Span suggests attentional disturbances, whilst the Digit Symbol test is sensitive to impairment with a lesion in any location **(McFie, 1969)**. The Verbal-Performance discrepancy is a useful guide to lateralization in a individual case, but should be corroborated with relevant medical and educational data and other tests results **(Bornstein, 1983).**

ii- *Assessment of Memory and Learning :*

Memory impairment associated with epilepsy was one o the earliest recognized deficits mentioned in the literature **(Gowers, 1881)**. It is also the most common problem reported by the patients themselves.

Methods of assessment of memory and learning :

*There are very few standardized memory tests. The most frequently used is the Wechsler Memory Scale, consisting of seven tests and yielding a Memory Quotient (MQ) In practice a selection from the WMS is made and these are used independently. Verbal tests most commonly used are the Logical Memory (short prose passage), Associate Learning (learning of easy-and difficult-to-associate pairs of words) and Digit Span (the longest string of digits recalled). The only non-verbal test in WMS is Visual Reproduction (drawing geometrical designs of progressive difficulty level). Among other standardized memory tests are the Rey Auditory Verbal Learning Test **(RAVLT)** consisting of words recall on several successive trials, and the Benton Bisual Retention Test, requiring reprodction of geometrical figures **(Jolanta, 1988).***

Memory function in patients with generalized versus focal epilepsy :

***(Quadfasel et al., 1955)** compared patients with psychomotor and generalized seizures, who were matched for IQ, educatioin, age of onset and duration of seizures. They used the WMS and found that patients with generalized seizures had normal memory function, but the patients with psychomotor seizures showed a verbal memory impairment. This deficit was associated with a general impairment in verbal functioning as measured by the verbal-conceptual tasks of the Wechsler-Bellevue. However, there were no differences between the groups on the Visual Reproduction test.*

Memory function in right TLE versus left TLE patients :

A number of studies have suggested that the side of lesion determines the kind of material more susceptible to deficit. Left-sided lesions result in memory impairment for verbal material, whilst right-sided or non-verbal material. The evidence for this pattern of deficits was obtained from surgical paients who has undergone a unilateral temporal lobectomy for treatment of epilepsy. But in patients without gross cerebral damage the evidence of interhemispheric differences for verbal and non-verbal material is less clear.

*Several studies distinguished between the STM and LTM impairment, or interpreted their results in terms of differential sensitivity on short-or longterm memory testing to deficits **(Jolanta, 1988).***

***(Ladavas et al. 1979)** investigated left versus right hemisphere memory function as assessed by the STM and LTM performance on a variety of verbal and non-verbal test. Although the STM tasks did not differentiate between the left ad right temporal lobe foci patients, the LTM tasks did not differentiate between the left and right temporal lobe foci patients, the LTM tasks did so, in the expected verbal/non-verbal direction. Similar results were obtained by **(Delaney et al. 1980)** who found that differences between the right ad left TLE patients were not apparent on immediate recall of either the verbal or non-berbal material, but only on delayed recall.*

Subictal EEG discharges and memory function :

*Several investigators have pointed to the disruptive effects of subictal (subclinical) discharges on memory. **Gloinski (1973)** first proposed this as a tentative hypothesis, which was supported later by studies comparing memory performance with EEG recordings for*

example, **Loiseau et al. (1983)** found that the patients with interictal bilateral spike and wave complexes had the poorest results on memory test.

Memory function and anticonvulsant drugs :

In the last decade or so there has been an icreasing awareness of the toxic effect of anticonvulsant drugs on cognitive function, and particularly of the harmful effects of polytherapy. This may occur even when drugs are kept within the therapeutic range, but the studies which have dealt specifically with memory impairment and anticonvulsants are few **(Jolanta, 1988)**.

(MacLeod et al. 1978) investigated memory function in epileptic patients treated with medium and high therapeutic doses of phenobarbiton. They found impaired STM performance as measured by the Sterberg scanning task when the drug level was high. Access to LTM. as measured by a test of identifying letters as the same either by their appearance (e.g. AA) or their name (e.g. Aa), was not affected.

The Sternberg task was also used by **(Andrewes et al. 1984, 1986)**, and revealed a relative memory impairment in patients treated with phenytioin, compared to those treated with carbamazepine the Sternberg task and latter-identifying task **(MacLeod et al., 1978)** utilize time of response as a memory measure, have fast speeds of presentatioin and are carried out over a longer passage of time than most memory test. it is arguable whether they are tests of memory, reaction time, or vigilance.

Thompson and Trimble have investigated memory among other cognitive functions in a number of studies with epileptic patients, and volunteers given anti-epileptic drugs. They have consistently employed the same battery of tests. Their memory test consists of immediate and delayed recall of twenty words and twenty pictures, followed by a recognition test of the items embedded in distractor items. **(Thompson et al., 1982)** found improved memory function in patients undergoing reduction in polytherapy or substitution of their.

iii- Assessment of other specific congnitive functions :

While global intellectual ability may be unaffected in a majority of patients with epilepsy, specific cognitive function, such as mental speed, attention and vigilance may show subtle impairments, and therefore measures of these abilities are often included in assessment procedures.

Mental speed :

Mental speed, or psychomotor speed, is most commonly measured by Reaction Time (RT). Simple Reaction Time (SRT) requires a response to a simple, unambiguous stimulus delivered at irregular intervals over a period of time. In Choice Reaction Time (CRT) alternative responses need to be made to two or more stimuli. There are difference of opinion whether the SRT is more sensitive to impairment, but some authors believe that the CRT is a better measure **(Jolanta, 1988).**

(Schwab, 1939) was the first who explored the effects of

generalized spikewave discharge on patients perjormance using a Reaction Time test and simultaneous EEG recording.

Attention, concentration and vigilance :

Specific cognitive functions such as attention. concentration and vigilance are poorly defined in the studies, and the terms are frequently used interchangeably. The term vigilance neers to general reactivity of the organism to incoming stimuli, whilst attention implies more selective response to a given stimulus or stimuli. and presupposes at least a moderate level of vigilance **(Bruhn, 1970)**. Most investigators however. do not make a clear distinction between attention, concentration and vigilance.

The most commonly used task in the assessment of vigilance or sustained attention is the Continuous Performance Test **(CPT)** **(Rosvold et al. 1959)**.

Impairment of sustained attention. as measured by the CPT. is patients with centrencephalic epilepsy was already noted in early studies, have demonstrated that this deficit was associated with spikewave activity, as shown by simultaneous EEG monitoring and task performance. Symmetrical, regular and synchronous bursts produced more deficit in performance than other bursits **(Jolanta, 1988)**.

There have been reports of inattentiveness of children with epilepsy **(Stores, 1973)**, but the outcome of the studies is not clear for instance, **(Stores et al. 1978)** found impaired attention in epileptic boys but not girls. In this study the CPT was used and the differences emerged in the second 8 min of the tests. In most other studies the statistical analysis of

the data was not detailed enough to reflect changes of performance over time.

Both the Stroop test and the visual scanning task have been used in a series of drug studies of Thompson and Trimble. In the study of the effects of reduction of plytherapy or substituting one of the drugs with CBZ, the performance on the visual scanning task improved in patients after changes in therapy, but the Stroop test showed no significant changes **(Thompson et al., 1982).**

II- Assessment of Behaviour disorders in epilepsy.

Impaired cognitive functioning impedes the development of age-appropriate adaptive behaviour, thus adding to the burdens of many children with epilepsy. Detailed neuropsychological assessment can identify the uderlying ability-related impairments that contribute to the adaptive behaviour deficiencies evidenced, particularly in the home and school settings. This information can serve as the basis of a multidisciplinary treatment plan tailored to individual needs. Ideally, the need for such a treatment plan should be perceived early in childhood, so that it can be developed and applied preventively. The same remedial principles also apply in older children and are here set out in detial **(Strang, 1990).**

(Fenton, 1981), outlines the clinical features of a behaviour disorder as follows :

Irritability, moodiness and sometimes frank mood changes, social withdrawal, quarrelsomeness, and paranoid attitudes, temper tantrums

and occasionally explosive aggressiveness, impulsiveness, restlessness, poor attention span, distractability and lack of application, disturbed inter-personal relationships and sometimes delinquent behaviour.

Behavioral problems to be assessed :

Immature personality, Irresponsible behaviour, temper tantrums, aggressive, tormenting, rebellious, physically aggressive, violent outbursts, verbal aggression, suicidal attempts, attention-secking, mood swings and apathy. **(Thompson, 1988).**

Behaviour characteristics

attributed by some to patients with epilepsy such as hypergraphia and religiousity **(Fedio, 1986)** would not be conceived of as behaviour disorders unless they were sufficiently severe to have a disruptive influence on an individual's life. Indeed some of these characteristics may actually have beneficial consequences depending on an individual's lifestyle and occupation.

Methods of Assessment :

Interview :

This represents the most frequently used form of assessment of behaviour disorders. Interviews vary in the degree of structure imposed, but the unstructured interview is probably the most commonly employed form of assessment in the United Kingdom. A major aim of the interview is to obtain a clear account of the problem behaviour, in particular, what it is, when it happens and how frequently it happens **(Thompson, 1988).**

*In epilepsy, the temporal relationship of the problem behaviours to seizures should always be explored. Occasionally what is described as a problem behaviour in the interview by a family may be an actual siezure, or be occurring in the postictal phase of the attack (**Fenton, 1986**). A patient's knowledge and attitude to his epilepsy may also underlie behaviour problems.*

Total relaince on information obtained from an interview is generally unwise.

Questionnaires

Questionnaires and inventories are generally viewed as being more objective and standardized than an interview. Many measures available are quick and inexpensive to administer in terms of staff time and material. The content of such questionnaires can vary considerably. The most structured and widely-used techniques in the field of epilepsy are personality inventories such as the Minnesota Multiphasic Personality Inventory.

*Some questionnaires take considerable time to administer and score and this greatly reduces their practical value. patients with limited ability and literacy skills may not adequately understand all of the questions, and naturally this reduces the value of several measures currently available (**Thompson, 1988**).*

Self-monitoring :

A variety of procedures including diaries, mechanical counters, daily charts and portable monitoring devices, exists to obtain self-observation data.

These methods can be particularly useful in gaining baseline information about the frequency of certain behaviours which cannot be observed readily, or monitored easily by other individuals. (Bornstein et al., 1986).

Direct observation :

Direct observation procedures can be classified into simulated observation and observation in natural settings. The former are specifically constructed to evoke a sample of behaviour. The most popular mehtod is role play. In this situation the participants act through a particular situation

Direct observation in natural setting can be a valuable source of information. a variety of recording procedures exists. the selection of which often depends on the nature of the presenting problem behaviour (Thompson, 1988).

Neuropsychological assessment

Patients with epilepsy most likely to present with behaviour difficulties generally have difficult-to-control seizures and evidence of underlying brain damge. These patients are also at risk for deficits of cognitive functioning and for intellectual deterioration.

Neuropsychological information, when combined with data of a more observational nature, can be very informative regarding reasons for difficult behaviour (Thompson, 1988).

Additional methods :

Medical and social records often provide valuable information in the assessment of behaviour problems.

When behaviour disturbance is acute in onset or inconsistent with past behaviour, the measurement of antiepileptic drug serum levels may be valuable. Patients may be more likely to present with problems when drug levels are high, particularly in the toxic range **(Reynolds, 1986).**

Aggressive Behaviour :

The association between aggressive behaviour and epilepsy has a long history.

Unfortunately, there still exists a lack of understanding about epilepsy that leads some professionals and families to accept difficult behaviour as part of the epileptic condition. More investigations into the assessment and non-pharmacological treatment of difficult behaviour in patients with epilepsy seems worthwhile and to be of direct relevance to those individuals who care for patients with problematic epilepsy **(Thompson, 1988).**

(8)

- **Electroencepholozaphic parameters.**
- **Other Investigations.**

I- Electroencephalography parameters in assessing the cognitive function in children with epilepsy :

Many biological and psychological possibilities have to be considered when attempting to explain cognitive dysfunction in the individual child with epilepsy. Electroencephalographic (EEG) information. which may be particularly relevant in some children. has mainly been studied in relation to the possible direct effects of seizure discharges on learning and behavior. Such discharges can be divided into transient, brief or prolonged. Prolonged seizure discharges includes nonconvulsive status epilepticus during wakefulness and status epilepticus during slow-wave sleep. In addition to the influence of seizure discharges. preliminary findings suggest that some children with epilepsy might have a subtle disorder of arousal mechanisms in sleep, possibly associated with impaired daytime performance. **(Stores, 1990).**

Neuropsychological assessment :

The variety of cognitive dysfunctions related to learning disabilities in children with epilepsy have been studied by linking electroencephalogram (EEG) and computerized neuropsychological testing. This showed that "subclinical" discharges impaired performance in 61% of the patients on a simple and a choice reaction time test, although some discharges lasted one second only. Neuropsychological investigation of subclinical EEG discharges may help to determine their adverse effect on learing. **(Rugland, 1990).**

Transitory Cognitive Impairment :

Subclinical epileptiform discharges are a common phenomenon in patients with epilepsy but also occur in children without a history of clinical seizures. Formal testing during EEG recording demonstrated transistory cognitive impairment (TCI) in 30-50% of patients. Discharges <3 s are technically difficult to examine and, in general, are considered to be very unlikely to impair patients performance.

These preliminary results suggest that TCIs are more common than generally acknowledged and could interfere with learning abilities of the affected children. Appropriate testing in children with subclinical discharges therefore, appears necessary. **(Pressle et al, 1995).**

The occurrence of generalized spike-wave activity during absence seizures was one of the first discoveries of clinical electroencephalography, yet it was soon relaized that such discharges were not necessarily accompanied by any clinical manifestations **(Colin, 1988).**

There was transitory cognitive impairment (TCI) during generalized subclinical EEG discharges, both in patients who appeared to be seizure-free and even in people not thought to have epilepsy (Ishiharea and Yoshii, 1967), however, the demonstration of TCI depends both on the psychological test employed and the nature of the EEG discharges. **(Peter, 1988).**

TCI and Neuropsychological Testing

This last finding has interesting theoritical implications. It had

*hitherto been assumed that TCI involved a global impairment of vigilance or attention or a reduction in the brain's overall rate of transmission of information (**Hutt et al., 1977**).*

Practical Implications of TCI :

Few investigators who has studied TCI appear to have addressed its possible practical consequences for day-to-day cognitive functioning.

*Indvidual patients are on record as having shown a worthwhile improvement of cognitive functioning and social adaptation when subclinical discharges were suppressed by antiepileptic drugs. (Whether an indication for treatment exists in only a small minority, or in many people with subclinical discharges, is unknown nor are any criteria established for identifying such patients. The need for further research in this area is obvious and urgent (**Colin, 1988**).*

Cognition and EEG laterization :

*Seizure phase with diffuse asynchronous quasiperiodic activity occurred more frequently (in 70% of the seizures) than did "classic" periodic 3-7-Hz activity in unilateral temporal regions (49%). Periods with simultaneous occurrence of two or more rhythmical activities (44%) and bilateral onset (21%) were common. This method is useful for improved quantitative assessment of dynamically changing rhythms in ictal surface EEG and can provid information not available from conventional analysis. (**Hilfiker et al, 1995**).*

*Although **(Whitfield, et al. 1995)** did not reach statistical significance, the data strongly suggested that either bilateral or left EEG lateralization is associated with verbal or global rather than visual memory problems.*

II- EEG discharges and behaviour in Epilepsy :

This was based on the idea that paroxysmal disorders of behaviour arise from paroxysmal discharges of subcortical structures which were not necessarily recorded in the EEG from the surface of the cortex.

*It was the recognition that electrical discharges in the EEG correlated with observed seizure behaviour that lead to a taxonomy of epilepsy based on EEG waveforms. Indeed, the first international classification of epilepsy relied heavily on different EEG pictures for different diagnoses **(Coline, 1988)**.*

*In the 1960s, in the UK, it was standard teaching that an epileptic seizure had to be accompanied by an alteration in cortical rhythms. Alterations in behaviour not accompanied by such changes were thought not to be due to epilepsy. **(Peter, 1988)**.*

II- Other investigations MRI and SPECT :

HS frequently exists in the presence of other MRI-detectable abnormality in the adjacent anterior temporal lobe. The basis of this abnormality must be explored in surgical specimens. **(Mitchell, et al, 1995).**

The demonstration of structural lesions in epileptic patients has a great significance in the classification, prognosis, and treatment of the disease. **(Gatzonis et al, 1995).**

Their suggested from findings and in respect to positive flase results. interictal SPECT may prove useful in delineating an epileptic focus. thus leading the way to surgical treatment and drug strategy.

Both generalized and focal epileptic seizures are associated with an increase in cerebral blood flow in the ictal state. In the interictal state in flow is decreased in the focal area region to below the normal level. $[^{99m}Tc]$ HmPAO SPECT previously was demonstrated to add to the localization of epileptic foci. To assess the contribution of $[^{99m}Tc]$ HmPAO SPECT to localization of epileptiform foci in cryptogenic partial epilepsy we compared SPECT results with EEG epileptiform activity.

SPECT may be considered a valuable additional diagnostic procedure in cryptogenic partial epilepsy and in epilepsy surgery candidates. We believe that it may become a helpful adjunctive test when EEG cannot accurately localize the epileptiform focus or electrographic seizure. **(Lopez et al, 1995).**

Because of the relative localizing value of each noninvasive procedure in patients with partial epilepsy. combination of different investigation procedures is needed. usually including chlinical examination, video EEG monitoring, and magnetic resonance imaging. In addition to these investigations, we performed a standardized protocol including neuropsychological and noninvasive neurophysiological testing.
(Derambure, et al, 1995).

Localization of the anatomic region responsible for seizures is critical in presurgical evaluation for medically refractory partial epilepsy.

Application of image registration techniques combining multiple modalities of functional imaging provides further understanding of both the functional nature and anatomic localization of the epileptogenic zone. These early results suggest that noninvasive interictal investigations may be able to play an increasing role in presurgical epilepsy evaluation.
(Knowlton, et al, 1995).

EEG :

The sleep EEG in children with epilepsy or febrile convulsions often shows spikes in midline regions (Cz, Pz) on the scalp, but the origin in the brain of these spikes is not clear. I observed midline spikes which were associated with spikes in the temporal regions. The patiens with these EEGs were 2 girls aged 7 and 10 years with febrile convulsions. Because the midline spikes and the temporal spike was consdidered to be in the same region of the brain. Because the origin of the temporal spikes was

believed to be the hippocampus, the origin of the midline spikes was also consideredthe hippocampus. The midline spikes were also believed to be recorded on the scalp through the ventricles and the longitudinal fissure, which were filled with cerebrospinal fluid of very low electrical resistance. For other electrical brain activities such as the K-complex in sleep EEG and P300, the potentials of which are also maximal in the midline region and the origin of which is not yet known, the distribution of these potentials on the scalp may be interpreted by the explanation presented. **(Ichijoh, 1995)**.

We noted a high correlation between the sum of spike detections in a given patient and the concordance between the human raters with respect to between raters and the corrected automated analysis.

Our data suggest, especially in patients with numerous spikes, that automated analysis (after elimination of artifacts) yields reliable and clinical useful measures of interictal EEG alterations. **(Pelzl et al, 1995)**.

Explored dipole source localization using brain electrical source analysis (BESA). Patients included those with epilepsy referred for surgery and those under medication control.

However, many epileptic subjects demonstrate two or more sources, some in close proximity and some at a distance. The appearance of a topographically shifting surface maximum during a single discharge signals the presence of at least two temporally distinct sources. **(Frank, et al, 1995)**.

Therefore, we conclude that in some cases of CCL subcortical neurophysiological mechanisms can explain EEG features, as well as in holoprosencephaly. **(Elia et al, 1995)**

Ambulatory EEG monitoring provides information about the clinical course of epilepsy with regard to the recording of nocturnal events (electric seizures) and probably could be useful in diagnosing the type of epilepsy. **(Pacifici et al, 1995).**

TGB does not cause deterioration in cognitive performance 'or produce any slow-wave activity or other new constant abnormalities on EEG during longer follow-up with higher doses of TGB (30-80 mg/day). **(Reetta et. al, 1995).**

Subject and Methods

Subjects & Methods

Our sample consisted of epileptic patients, whose diagnoses were based on the criteria and electroencephalographic (EEG) classification of epileptic seizures according to the International League against Epilepsy 1993.

Pilot study :

Before starting to collect the data, a pilot study was carried out in :

1) The Psychiatry and Neurology Centre for school pupils in El Dokki District

2) The General polyclinic for school pupils in Al-Haram District

The aim of this pilot study was :

a) To determine the sample size.

b) To determine the organizational and administrative procedure needed for the research work.

c) To detect the best methods of applicatioin of the questionnaire.

d) To reveal any modifications needed in the questionnaire.

e) To detect the difficulties that might arise and how to deal with them.

Feedback of the Pilot Study :

Following the pilot study, we found out that the ratio between TLE and GME is 2:3.

We found out that the tools were suitable, comprehensible and easy, however some modification of the questionaire concerning epilepsy was essential

---

TLE = Temporal Lobe Epilepsy
GME = Grand Mal Epilepsy

Site and sample of the study :

The study was conducted in two sites :

1) The Psychiatry and Neurology Centre for school pupils in El Dokki District.

The centre, recruits patients from Giza and northern upper Egypt. it is a governmental one. affilated to the Central Health Insurance Authority and offers medicine free of charge to its patients.

Around 40 to 50 patients are attend the centre daily, namely about 1000 monthly, 60.2% of whom suffered from epilepsy. 51.2% of epileptic children could be diagnosed as GME, 32.9% as TLE and 15.9% is patients suffered from other types of epilepsy.

From the epileptic attendats 10.6% were new cases and 90% were recurrent.

2) The General Polyclinic for School Pupils in Al-Haram District. The Neuropsychiatry clinic recruits patients from Giza. and northern Upper Egypt.

It is a governmental institution affiliated to the General Health Insurance Authority, and offers medicine free of charge. About 25 to 30 patients are attended the clinic daily, namely about 700 patients a month, 69.7% of which are suffered from epilepsy. 50% of epileptic children could be diagnosed as GME, 32% as TLE and 18% as patients suffered from other types of epilepsy.

From the epileptic attendats 11.5% were new cases and 88.5% were recurrent.

The members of this sample were selected during the period from February 1995 throught August 1996.

Inclusion Criteriae :

Epileptic pupils attended in the outpatient clinics of Epilepsy

Age : *9 - 15 years*

Sex *: Males & Females*

Educational level *: all members of the samples were school children. This was a necessity so as to be able to complete all components in methodological procedure.*

Exclusion Criteriae :

1) *Patients with IQ below 70*

2) *Patients with any medical disease*

3) *Patients with history suggesting any neurological disorder other than epilepsy.*

4) *Patients with history suggesting any psychiatric disorder.*

Mean age of GME and TLE samples

Gender	GME	TLE	t	P
Male (mean.)	12.08 yrs.	‾ 11.6 yrs.	1.95	0.076
Female (mean.)	11.88 yrs.	11.88 yrs.	1.84	0.091
Total	23.96	22.94	-	-

Epileptic sample attending the two sites of study (6-18 years)

	GME	TLE	Others
Site I	308	198	96
Site II	244	156	88
Total	552	354	184
Patients fulfilling inclusion criteria	338	236	127
Random sample	169 every second	118 every second	-
Drop out	23	12	-
Rest sample	146	106	-

From 308 GME patients from site (1) and 244 from site (2) , 338 patients fulfilled the inclusion criteria. Cases chosed through random sample of every second case giving 169 case with drop out of 23 , the rest were 146.

From 198 TLE patients from site (1) and 156 from site (2) , 236 patients fulfilled the inclusion criteria. Cases chosed through random sample of every second case giving 118 case with drop out of 12, the rest were 106.

Cases were selected through random sample fullfilling inclusion criteria and rueling out exclusion criteria.

Control group :

The control group comprised 93 apparently healthy matched indivduals were chosen from patients' sibs and schoolmats with no history suggesting Epilepsy or other medical, neurological or psychriatric diseases

Fig. (1): Epileptic sample of patients' attending in the first site of study (6-18 years)
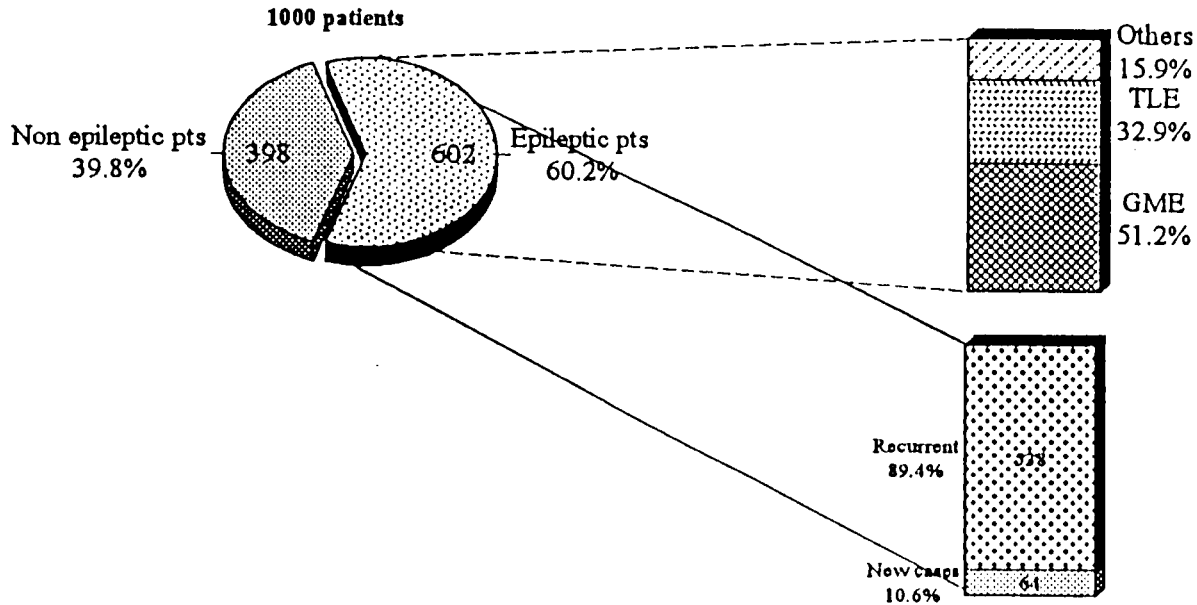
Fig. (1): Epileptic sample of patients' attending in the first site of study (6-18 years)
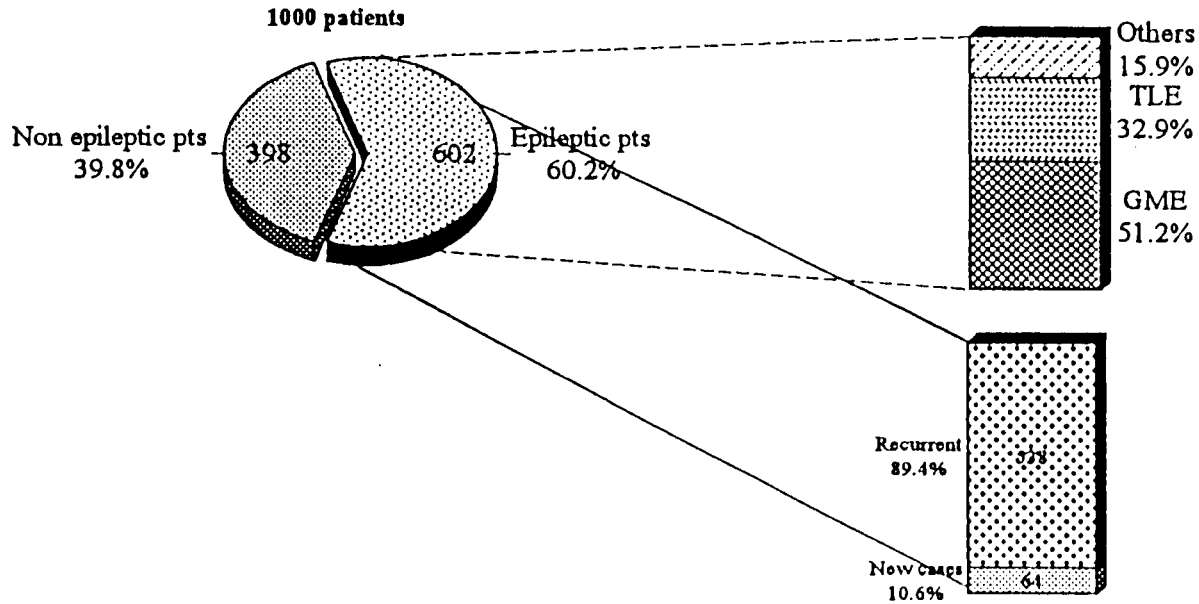
Tools Applied in this work

1) **Semistructured psychiatric interview.**

2) **Questionaire of Epilepsy.**

3) **Psychometric battery :**

    I- For Cognitive functions :

        a) Coding (SubWechseler)

        b) Digit Span (SubWechseler)

        c) Similarities (SubWechseler)

        d) Cancellation letters

        e) Matching Familiar Figure Test (MFFT).

    II- For personality assessement and measurement of mood :

        a) Children Depression Inentory (CDI)

        b) The children Anxiety scale (CAS)

        c) Junior Eysenk Personality Questionaire (JEPQ)

4) **Questionaire for Socio-economic state.**

5) **Electroencephalogram (EEG)**

6) **Others investigations :**

    BEAM, MRI, CAT and serum level of antiepileptics (only in some cases).

Psychometric battery :

    I- For Cognitive functions :

        a) Coding (SubWechseler)   [The Digit Symbol Modalities test (D.S.M.T.)]

It is the substitution Format of Wechsler's Digit Symbol subtests. Digits (Numbers) are printed while symbols are written. It consists of 4 rows containing a total of 100 small blank squares (1st 6 for pretest training) each paired with a randomly number. from (1-9).

Above these rows is a printed key that pairs each number with a different nonsense symbol. The subject is asked to fill the squares with the symbol which according to the key, is paired to the number abone the square. Speed is icquired and after 120 seconds the test is to terminated.

The Subject is scored according to the numbers of squares he fills in correctly. (El.Kholi, 1985).

This test measures :

Persistency. Sustained attention. Learning ability. Psycho motor speed and Visu- motor coordination.

Coding is affected to a great extent by organic lesions. Speed and accuracy in performance of this test reflects the level of mental ability.

*b) * **Digit Span (SubWechseler) :***

For immediate and short term memory .

The test has three scores, one for digit forward, one for digit backward and a total digit span score which is the sum of the former two. The digits are presented in series increasing in lenght starting from 3 and extending to 9 digits for the forward direction and 8 digits for the backward dircetion.

a) Digit Forward : The subject is presented with pre recorded digit sequences at a rate of 1 digit per second, and asked to repeat them exactly as he heard them. The subject score is the number of digits in the longest correctly repeated sequence.

b)   *Digit Backward : The subject is asked to say them in an exactly reversed order. The subject score is the number of digits in the longest correctly repeated sequence.*

Note : (Each section is discontinued when the subject fails to repeat both number sequence of a pair).

c)   *Total Digit-Span : Is the sum of the scores of the two sections.* **(El.Kholi, 1985).**

Digil Span : *measures.*

Forward : *Immediate memory and Auditory attention*

Backward : *Short memory and Internal visual scanning (Weinbery) (Melika. 1996).*

The total degree may be balanced (10). but on looking at the two parts of the test we find differences between forward and backword.

The difference in degree between the sound people can never reach 5 degrees.

Education has a considerable positive impact on the forward form.

For the forward form the upper level of normal is 6 rough numbers and 3 rough numbers indicates below normal degree.

For the backward form the upper level of normal is 5 rough number and 2 rough numbers indicat below normal degree.

In term of rough numbers the normal difference between forward and backword numbers is 2 with SD± (.59-2) backward.

Backward test is related to memory rather than attention.

c) * **The Similarities test (Sub-Wechseler) :**

Twelve couples of words requiring the subject to say in what way are two things alike (Anastasi, 1972).

It is subtest from Wechsler test for children intellegence.

The smilarities scale was applied to the subjects so as to save the subject's time and effort for the main battery of tests.

The choice of the similarities test was based on the following facts :

1) This scale is the best at representing intellegence with its verbal and performance components (Wechsler 1944).

2) Similarities scale showed the highest correlation with the total score on the W/B thus reflecting a high degree of validity. The similarities correlates with the total scale 0.73 in different age group (IBID PP 94.).

3) Similarities scale have a high degree of reliabitly ranging between 0.79 - 0.87 **(El. Kholi,1985).**

Similarities scale measures, concept formation abstract and concrete thinking **(Meleka, 1996).**

d) Cancellation letters :

This consists of a printed sheet with 323 random Arabic single letters. The subject is asked to mark each (ب) and (ن).

The sheet included 38 (ب) and 32 (ن) letters.

The subject is scored according to the time of performance and number of errors.

Cancellation letters measure attention and responce rate (El. Kholi, 1985).

e) Matching Familiar Figure Test (MFFT) :

This test measure reflectivity versus impulsivity as bipolar dimension measured jointly by error and latency **(Kagen 1964).**

The examiner showed the child twelve groups of pictures. each of which in two pages one over the other.

Before doing this, the examiner presents them with two groups for practice.

There was a picture on the one page and six pictures on the oposing page.

The examiner asked the child the point to the picture in the lower side which is like that in the upper side.

The examiner would record latency by seconds until the first response comes. The total number of errors of each group is calculated.

If the subject is correct, the examiner will praise him.

If wrong, the examiner will tell the child "No that is not the right one. Find the one that is just like this one (point)". Continue to code responses without time recording until the child makes a maximum of six errors or gets the correct item. If incorrect, the examiner will show the right answer **(Kagen 1964).**

II- For personality assessement and measurement of mood :

a) Children Depression Inentory (CDI)

This test was done by Marria Kovacs **(Kovacs, 1983).** It covers a wide range of depression symptons, including mood disorders and the ability to enjoy the development of growth functions self assertion and

personal behavior with others as well as circumstances surranding school activities **(Gharieb, 1985).**

The scale has been established in its full form at 1982 including 27 Questions, each has 3 answers ranging from 0, 1, 2 degrees accoring the patient responce, then the results were matched with the norms that aranged according to sex and age.

The test was traslafed and acclamatized according to Egyptian norms by **(Gharieb, 1985.)**

This survey inventory covers 27 symptoms:

Sadness, pessimism, feeling of failure, general feeling of loss of joy, ill temper pessemistic anxiety, self hatred felling of guilt, suicidal thoughtr, inbility to tolerate frustration, loss of social awareness, hesitation, body disfigureement, loss of volition for schoolastic achievement, insomnia, Fatigue, loss of appetite, loneliness, loss of schoolastic enjoyment, social isolation, loss of friends, school perofmence deterioration, low self esteam, feeling of being outcast, disobedience and social troubles, **(Kovacs, 1983).**

Items of this test concentrate on the effect left by depression on children's circumstances, as at school for instance.

This test is suitable for children from 7 to 18 in age.

It consists of 27 group of sentenses each of which of 3 parts, with the examined having to chose one of them.

Each sentense take from zero to two in terms of degrees in accordance with the severity of the sympotom.

So, the total score ranges from zero to 54 interms of degrees.

b) Assessment of Anxiety. (The children Anxiety scale- CAS).

This scale was designed by **Abd-Hamied and El-Nial (1991)**. It is an arabic version derived from Children's Manifest Anxiety Scale (CMAS) by Mc Candless, Castaneda and Palermo. This scale consists of 36 statements and measures all the aspects of the anxiety which include the following :

1) Somatic features.   4) Emotional features.

2) Physiological features.   5) Mental features, and

3) Motor features.   6) Social features.

Each category could be assesed by 6-statments. The child after reading each statment by himself or by the interviewer answers either yes or no, if the answer is yes he scores 1 and if it is no he scores 0. The total degree ranged between 0-36, the cut off point of the scale is 18 and above 18 the child is considered to have high anxiety as a state.

The authors prepeared the (CAS) to be applied on the children at the primary and preperatory schools and both the reliability and the validity of the scale were assessed by the authors.

c) Junior Eysenk Personality Questionnaire. (JEPQ)

Measure the dimensions of extraversion versus introversion, neuroticism versus psychotocism, lie scale & criminility according to Eysenk (1964). This J.E.P.Q. consists of 96 questions inorder to measure these four aspects of the personality. Instructions for literate subjects are printed on each copy of the J.E.P.Q. these should be read aloud to groups of subjects or be read silently by subject tested individually. When the questionnaires are collected after completion, care should be taken to check that all questions have been answered.

The questionnaires are scored by using the appropriate scoring sheet there are four keys for the junior version of the E.P.Q. *

Socioeconomic assessment

As affect completely personality changes in epileptic patient. This is through (Questionnaire for Socioeconomic State).

The questionnaire is in Arabic and prepared to include most of the risk factors that might be associated with psychiatric disorders in the children. It consists of 32 items. This questionnaire was applied to all the sample, the parents answered it at home and the questionnaire was recollected on the second day. The questionnaire was designed by the worker and was accepted by the supervisors to be used in this work. A social class classification was devised in Britain 50 years ago and is based on a grading of occupation according to their level of skill and financial rewards and on the general status they confer in the community. This classification was derived from **Barker, 1987.**

Investigations :

Electroencephalogram (EEG) :

It was done in the unit of Neurophysiology in El Haram Polyclinic, using a Japanese-made apparatus. (NIHON-KOHDEN) with 8 channels.

Routine EEG, were performed for all patients and control groups using a vega-10-10 channel machine. Electrodes were placed according to the 10/20 international system of electrode placement.

EEG was obtained using the bipolar and refrential montages standard for our laboratory.

Provocation techniques included hyperventilation were done to patients and control groups.

---

* *See Appendix*

Procedures

Cases and control groups were subjected to :

1) Physical and Neurological examinations :

Patients and control groups had undergone examination to rule out any physical, mental or neurological diseases.

2) Psychiatric Interview :

(Semistructured interview) adopted in the Institute of Psychiatry and Neruology, Ain Shams University

3) Questionnaire on childhood Epilepsy :

Prepared by the worker modified after pilot study and accepted by supervisors.

The Quessionaire is conducted to cover the following :

1) Aetiology and precepitating factors

2) Seizure onset, past seizure frequency, current seizure frequency, longest seizure free period, date of last seizure & history of status epilepticus (if present).

3) - Current seizure pattern, prodromal symptoms, aural, Ictal and post-ictal states.

   - Factors affecting seizure control and general principles which were used for management.

4) Type of drug intake, monotherapy or polytherapy controlled seizure or not. Dose, duration of intake, side effects, serum level if possible and if the patient is compliant or not (see appendix).

All data obtained are enlisted in a sheet and computerized to be accessible for statistics.

Data were obtained from both patients in and members of their family.

Statistical analysis

The data was collected and proccessed to a personal completer (PC) IB compatible and analysis was done with the aid o the programe (SPSS) statistical package for social science version 6. 0.

The statistical test used are :

1) Prevalence :

Definition : The prevalence (or, more property, the point prevalence) or a disease in population is the proportioin of that population having the disorder at a given point in time.

a- Prevalence is defined in terms of a single point in time even though the actual process of data collection may take place over days, weeks or years.

b- Thus, prevalence provides a static measure of disease frequency, analogous to a singe frame of a motion picture.

The calculation

a- Prevalence is calculated using the formula

$$Prevalence = \frac{\text{total number of diseased individuals at given time}}{\text{total population}}$$

b- The numerator encompasses both new and on going cases o the disease. **(Knapp and Miller, 1992)**

2) the mean = the arithmatic average

$$\text{The mean "M"} = \frac{\Sigma X}{N}$$

where Σ = the sum of,

X = individual values,

N = the number of cases

3) Standard deviation (SD)

It is the square root of the variance. It gives an estimate of the average deviation around the mean

$$\text{S.D.} = \sqrt{\frac{\Sigma X2 - \frac{(\Sigma X)^2}{n}}{n - 1}}$$

where $\Sigma X2$ = the sum of squares of the individual values, and

$(\Sigma X)2$ = the square of the same of the individual values

4) T. test for independent samples :

$$\text{T.} \frac{\overline{X1} - \overline{X1}}{\sqrt{\frac{SD_1^2}{N1} + \frac{SD_2^2}{N2}}}$$

Where X1 = the mean of the first group

Where X2 the mean of the second group

SD_1 = standard deviation o the first group

SD_2 = standard deviation o the first group

N_1 = number of cases in group one

N_2 = number of cases in group two

5) Chisquare test X^2.

$$X^2 \overset{\text{sum of}}{\sum} \frac{(O - E)^2}{E}$$

Where O = is the observed value

E = Expected value

6) Fisher excate test

For expected numbers less than five.

7) One way analysis of variance (ANOVA)

The total sum of squares about the mean

$$\sum X_T^2 \frac{(\sum x T)^2}{E}$$

The "between groups" sum of squares

$$\frac{(\sum x\ 1)^2}{N_1} + \frac{(\sum x\ 2)^2}{N_2} + \qquad + \frac{(\sum x\ m)^2}{N_m} + \frac{(\sum x\ T)^2}{N_T}$$

Where

$N\ T$ = Total no. of observation

$N_1, N_2, \dots\dots, N_m$ = number in individual group

$\sum x\ T$ = total of al values

$\sum x\ 1, \sum x\ 2, \dots\dots, \sum x_m$ = total of the values in each group.

Results

RESULTS:
I) General .
II) Details .

RESULTS:

I) General .

1) Psychodemographic profile .

2) Illness profile .

3) Therapeutic issues .

4) Investigations .

   a) Psychometry .

   b) EEG .

1) Psychodemographic profile.

(Table 1) Comparison between GME and TLE groups through :

	GME		TLE		P<0.05 (SIG.)
	NO=146		NO= 106	%	
I- SEX					
MALE	83	56 85	56	48 29	NS
FEMALE	63	43 15	50	51 72	
II- FAMILY SIZE					
<4	52	35 62	22	20 75	0 0108
>4	94	64 38	84	79 25	
III- SIB ORDER					
FIRST TWO	81	55 48	56	52 83	NS
MORE	65	44 52	50	47 17	
IV- ROOMS					
1-2	78	53 42	34	32 08	0.00078
MORE	68	46 58	72	67 92	
V- WANTED					
YES	124	84 93	69	65 09	0.0002414
NO	22	15 07	37	34 91	
VI- CLASS					
RURAL	89	60 96	63	59 43	NS
URBAN	57	39 04	43	40 57	
VII- PARENTAL SEPARATION					
YES	32	23 53	33	31 13	NS
NO	104	76 47	73	68 87	
XII- FAMILY INCOME					
HIGH	91	62 33	43	40 57	0.0006312
LOW	55	37 67	63	59 43	
XIII- EDUCATED PARENTS					
YES	68	46 58	61	57 55	NS
NO	78	53 42	45	42 45	

The family size of GME patients are significantly higher in number than that of TLE patients .

Also , family size in cases of GME patients is higher than TLE patients (P< 0.05)

GME patients are wanted children more than TLE patients (P < 0.001)

Test used :

Chi-square test .

P (< 0 .05) = Significant .

P (> 0.05) = Non- significant .

2) Illness profile.

(Table 2) COMPARISON BETWEEN GME GROUP,
& TLE GROUP CONCERNING
<u>ONSET OF SEIZURES</u>

	GME NO=146		TLE NO= 106		P
I- AGE OF ONSET					
ONSET < 2YEARS	4	2.7%	5	4.7%	
ONSET (2- 4) YEARS	14	9.6 %	16	15.1%	NS
ONSET > 4 YEARS	128	87.7%	85	80.2%	
II- TYPE OF ONSET					
ABRUPT	38	26.1%	37	34.9%	NS
GRADUAL	108	73.9%	69	65.1%	
(WITH PRECEPITATING FACTORS)					

This table shows, no significant difference between GME and TLE groups as regards age of onset (whether below 2 years, between 2 to 4 years or above 4 years) or regards type of seizure (whether abrupt or gradual).

ONSET OF SEIZURES

Test used :
   Chi-square test .
   P (< 0 .05) = Significant .
   P (> 0.05) = Non- significant .

81

2) Illness profile.

(Table 3) COMPARISON BETWEEN GME GROUP, .
& TLE GROUP CONCERNING
FREQUENCY OF SEIZURES

	GME NO=146 %		TLE NO= 106 %		P
I- CURRENT FREQUENCY					
1 DAY	9	6.2	24	22.6	< 0.001.
1 WEEK	21	14.4	24	22.6	
1 MONTH	44	30.1	24	22.6	
NONE	72	49.3	34	32.2	
II- PAST SEIZURE FREQUENCY					
1 DAY	45	30.8	62	58.5	< 0.001
1 WEEK	34	23.3	10	9.4	
1 MONTH	67	45.9	34	32.1	

FREQUENCY OF SEIZURES :

* Current seizure frequency :-

This table shows a significant difference between GME and TLE groups concerning current seizure frequency. (< 0.001)

GME > TLE :

frequent seizure/month

none frequent seizures

TLE > GME

frequent seizure/day

frequent seizure/week

* Past seizure frequency :-

This table shows a significant difference between GME and TLE groups concerning past seizure frequency (< 0.001)

GME > TLE :

Post seizuire frequency/week

Post seizuire frequency/month

Test used :

Chi-square test .

P (< 0.05) = Significant .

P (> 0.05) = Non- significant .

82

2) Illness profile.

(Table 4) DURATION

COMPARISON BETWEEN GME GROUP, & TLE GROUP CONCERNING DURATION

	GME NO=146		TLE NO= 106		P
I- DURATION OF ILLNESS					
< 6 MONTHS	27	18.5%	9	8.5%	< 0.0 5
(6 MONTHS - 2 YEARS)	42	28.8%	23	21.7%	
(2 YEARS- 5 YEARS)	42	28.8%	47	44.3%	
>5 YEARS	35	23.9%	27	25.5%	
II- LONGEST SEIZURE Free PERIOD					
< 6 MONTHS	80	54.8 %	81	76.4%	< 0.05
(6 MONTHS - 2 YEARS)	66	45.2 %	25	23.6%	

Duration of illness :

This table shows, a significant difference between GME & TLE concerning duration of illness.

GME > TLE :

Duration (< 6 months)

Duration (6 months - 2 years)

TLE > GME

Duration (> 5 years)

Duration (2 years - 5 years)

Longest seizure Free period :

This table shows. a significant difference between GME & TLE groups concerning longest seizure free period.

GME > TLE :

Longest seizure free period (6 months - 2 years)

TLE > GME

Longest seizure free period (<6 months)

Test used :
   Chi-square test.
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant .

2) Illness profile.

(Table 5) COMPARISON BETWEEN GME GROUP,
& TLE GROUP CONCERNING
STATUS EPILEPTICUS, FAMILY & PAST HISTORY

	GME NO=146		TLE NO= 106		P
I- STATUS EPILEPTICUS HISTORY					
WITH	13	8.9%	18	16.98 %	NS
WITHOUT	133	91.1%	88	83.01%	

This table shows, no significant difference between GME and TLE groups as regards status epilepticus history.

II- POSITIVE FAMILY HISTORY					'
WITH	27	18.5%	21	19.8%	NS
WITHOUT	119	81.5%	85	80.9%	...

This table shows, no significant difference between GME and TLE groups as regards positive family history.

III-POSITIVE PAST HISTORY					
WITH	3	2.1%	8	7.55%	NS
WITHOUT	143	97.9%	98	92.45%	

This table shows, no significant difference between GME and TLE groups as regards positive past history.

STATUS EPILEPTICUS‹ FAMILY AND PAST HISTORY.

Test used :
Chi-square test.
$P (< 0 .05) = Significant$.
$P (> 0.05) = Non- significant$.

84

2) Illness profile.

(Table 6) COMPARISON BETWEEN GME GROUP.
& TLE GROUP CONCERNING
PERIICTAL CHANGES

	GME NO=146		TLE NO= 106		P
I- PRODROMA					
WITH	54	36.99%	64	60.38 %	0.0002392
WITHOUT	92	63.01	42	39.62%	
(Figure, 6 & 7)					
II- AURA					
WITH	86	58.90%	101	95.28	0.0000000001477
WITHOUT	60	41.10	5	4.72%	
(Figure, 8 & 9)					
III-ICTAL CHANGES					
WITH	113	77.4%	101	95.28	0.0008971
WITHOUT	33	22.6%	5	4.72%	
IV- POST ICTAL CHANGS					
WITH	117	80.14	101	95.28	0.0005123
WITHOUT	29	19.86	5	4.72%	
(Figure, 10)					
V- FACTORS AFFECTING ICTUS					
WITH	113	77.4%	83	78.3%	NS
WITHOUT	33	22.6%	23	21.70%	
(Figure, 11)					

PERIICTAL CHANGES

This table shows, no significant difference between GME and TLE groups as regards factors affecting ictus .

In the other hand , it shows a significant difference between GME and TLE groups as regards the following :

1 - Patients with prodroma .
2 - Patients with aura .
3 - Patients with ictal changes .
4 - Patients with post-ictal changes .

Test used :
   Chi-square test .
$P (< 0.05) = Significant$.
$P (> 0.05) = Non- significant$.

85

2) Illness profile.

(Table 7) COMPARISON BETWEEN GME GROUP.
& TLE GROUP CONCERNING
CAUSES

	GME NO=146 %		TLE NO= 106 %		P
I- CEREBRAL:(Figure, 12)					
WITH	84	57.53%	38	35.85 %	0.0006727
WITHOUT	62	42.47%	68	64.15%	
II-SYSTAMIC:(Figure, 13)					
WITH	89	60.96%	90	84.91%	0.00003518
WITHOUT	57	39.04%	16	15.09%	
III-IATROGENIC :					
WITH (Figure, 14)	16	10.96%	3	2.83%	0.0158
WITHOUT	130	89.04	103	97.17%	

CAUSES:

This table shows, a significant difference between GME and TLE
groups as regards cerebral , systemic and iatrogenic aetiology

GME patients shows tendency to cerebral and iatrogenic aetiology
TLE patients shows tendency to systemic aetiology .

Test used :
    Chi-square test .
$P (< 0 .05) = Significant$.
$P (> 0.05) = Non\text{-} significant$.

86

2) Illness profile.

(Table 8)

Comparison between GME & TLE Concerning their Psychiatic Morbidity.

	GME NO=146	%	TEL NO=106	%	P
I Patients with Psychiatric Morbidity :	46	31.5	42	39.6	
Conversion Disorder .	10	6.9	3	2.83	
Conduct Disorder .	16	10.95	17	16.03	NS
Anxiety Disorder .	17	11.6	19	17.91	
Sex Disorder .	3	2.05	3	2.83	
II Patients without Psychiatric Morbidity:	100	68.5	64	60.4	NS

This table shows, no significant difference between GME and TLE groups as regards psychiatric morbidity .

TLE patients shows tendency to conduct disorder, sex disorder and anxiety disorder . GME patients shows tendency to conversion disorder

Test used :
   Chi-square test .
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant .

2) Illness profile.

(Table 9) Comparison between GME and TLE groups through :

Organic disease, Pregnacy troubles, Labour troubles and Neurotic tiaits History.

	G. ME		TLE		P
	No = 146	%	No = 106	%	
I - WITH ORGANIC DISEASE					
YES	104	71 23	58	54 72	0 006909
NO	42	29 77	48	45 28	.
II - PREGNANCY TROUBLES					
YES	21	14 38	25	23 58	NS
NO	125	85 62	81	76.42	
III - LABOUR TROUBLES					
YES	28	19 18	30	28 3	NS
NO	118	80 82	76	71.7	
IV - NEUROTIC TRAITS					
YES	119	81 51	80	75.47	NS
NO	27	18.49	26	24.53	

This table shows, no significant difference between GME and TLE groups as regards pregnancy troubles, Labour show tendency to organic disease.

Test used :
  Chi-square test .
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant .

88

3) Therapeutic issues.

Correlation Between GME, TLE & Control Groups Concernir

(Table 10) DIFFERENT TYPES OF THERAPY

TYPE OF SEIZURES	GME		TLE		P
	NO=146	%	NO=106	%	
TYPE OF THERAPY					
A- MONOTHERAPY	97	66.44	55	51.89	
B- POLYTHERAPY	48	32.88	50	47.17	0.0218
C- NO THERAPY	1	0.07	1	0.9	

This table shows, a significant difference between GME and TLE groups as regards type of therapy whether mono or polytheray.

It shows that there is a tendency of GME patients to take monotherapy, in the other hand TLE used more frequently polytherapy.

Test used :

Chi-square test .

P (< 0 .05) = Significant .

P (> 0.05) = Non- significant .

3) Therapeutic issues.

Table II) COMPARISON <u>BETWEEN SIDE EFFECTS / EACH Drug</u>
<u>IN BOTH GROUPS GME & TLE PATIENTS</u>

	GME		TLE		P
	NO OF DRUG	NO OF SE	NO OF DRUG	NO OF SE	
<u>DEPAKINE</u>.	44	6	51	11	NS
<u>TEGRETOL</u>.	127	36	46	21	0.0324
<u>EPANUTIN</u>.	18	2	8	0	NS
<u>RIVOTRIL</u>.	4	2	9	5	NS

<u>*SIDE EFFECTS / EACH DRUG*</u>

GME . Suffered Significantly From Tegretol side effects than did
TLE .

<u>*Test used :*</u>
<u>Chi-square test</u> .
$P (< 0.05) = Significant$.
$P (> 0.05) = Non\text{-} significant$.

90

<u>*AED'S*</u> <u>*Trade Name*</u>	<u>*AED'S*</u> <u>*Scientific Name*</u>
- **Depakine** - **Tegretol** - **Epanutin** - **Rivotril**	- Sodium Valproate - Carbamazepine - Hydantoin - Clonazepam

CORRELATION BETWEEN AMOUNT OF DRUG INTAKE /EACH DRUG

1) MONOTHERAPY	GME (SE)		TLE (SE)		P
	NO OF DRUG	%	NO OF DRUG	%	
DEPAKINE .	44	30.1	51	49.04	0.002207
TEGRETOL .	127	86.99	46	43.4	0.00000000007606
EPANUTIN .	18	12.33	8	7.55	NS
RIVOTRIL .	4	1.37	9	8.49	0.0156

This table shows, no significant difference between GME and TLE groups as regards Epanutin intake.

However, there is significant difference between GME patients to take Tegretol, in the other hand TLE uses more Depakine and Rivotril.

2) *POLYTHERAPY*	GME		TLE		P
	NO=146	%	NO=106	%	
DEPAKINE TEGRETOL.	26	17.8	35	33.01	0.004390
TEGRETOL. EPANUTIN	10	6.85	2	1.89	NS
DEPAKINE EPANUTIN	6	4.11	2	1.89	NS
EPANUTIN RIVOTRIL	2	1.37	0	0	NS
TEGRETOL. RIVOTRIL. DEPAKINE	4	2.74	7	6.6	NS
TEGRETOL. DEPAKINE EPANUTIN	2	1.37	0	0	NS
TEGRETOL. RIVOTRIL. EPANUTIN	0	0	2	1.89	NS

(Table 12) AMOUNT OF DRUG INTAKE / EACH DRUG :

This table shows, no significant difference between GME and TLE groups as regards combined therapy of Tegretol/Epanutin, Depakine/Epanutin, Epanutin/Rivotril, Tegretol/Rivotril/Depakine, Tegretol/Depakine/Epanutin or Tegretol/Rivotril/Epanutin.

In the other hand, there is significant difference between GME patients as regards combined therapy of Depakine and Tegretol. There tendancy of TLE to Depakine/Tegretol combination more than GME.

Test used :
  Chi-square test .
$P (< 0.05) = Significant$.
$P (> 0.05) = Non\text{-} significant$.

AED'S *Trade Name*	*AED'S* *Scientific Name*
- Depakine	- Sodium Valproate
- Tegretol	- Carbamazepine
- Epanutin	- Hydantoin

3) Therapeutic issues.

(Table 13) DURATION OF DRUG INTAKE

DURATION OF DRUG INTAKE IN BOTH GROUPS GME & TLE

DURATION OF DRUG IN TAKE	GME		TLE		P
	NO=146	%	NO=106	%	
<2YEARS	97	66.44	75	70.75	
(2-5) YEARS	30	20.54	28	26.4	
> 5 YEARS	19	13.01	3	2.83	0.004696

Thes table shows a significant difference beween GME and TLE concerning duration drug intake :

GME > TLE :

In duration of drug intake (> 5 years)

Test used :

Chi-square test .

$P (< 0.05) = Significant$.

$P (> 0.05) = Non- significant$.

92

(Table 14) COMPLIENCE, SEIZURE CONTROL, SIDE EFFECTS & HIGH SERUM LEVEL.

	GME		TLE		P
	NO=146	%	NO=106	%	
I- COMPLIENCE					
A- COMPLIENT PATIENTS	100	68.49	70	66.04	**NS**
ON MONOTHERAPY	77	77	45	42.45	0.0000049
ON POLYTHERAPY	23	23	25	35.71	
B- NON COMPLIENT PATIENTS	46	31.51	36	33.96	
ON MONOTHERAPY	23	50	9	25	0.0213
ON POLYTHERAPY	23	50	27	75	

This table shows, no significant difference between GME and TLE groups as regards complience .

There is a significant difference between GME and TLE groups as regards type of complient patient (on monotherapy or polytherapy GME patients shows tendency to complient on monotherapy more than TLE patients .

This table shows, no significant difference between GME and TLE groups as regards non- complience .

There is a significant difference between GME and TLE groups as regards type of non- complient patient (on monotherapy or polytherapy GME patients shows a tendency to non- complient on monotherapy more than TLE patients .

II-SEIZURE CONTROL					
A- CONTROLLED SEIZURE	71	48.63	35	33.02	**0.0132**
ON MONOTHERAPY	59	83.1	23	66.71	0.0443
ON POLYTHERAPY	12	16.9	12	34.29	
B- NON CONTROLLED SEIZURE	75	51.37	34	32.08	
ON MONOTHERAPY	44	58.67	30	42.25	0.0474
ON POLYTHERAPY	12	16.9	12	34.29	

This table shows, no significant difference between GME and TLE groups as regards controlled seizures .

There is a significant difference between GME and TLE groups as regards type of controlled seizure patients (on monotherapy or polytherapy GME patients shows a tendency of controlled seizure patients on monotherapy more than TLE patients .

This table shows, no significant difference between GME and TLE groups as regards non- controlled seizures .

There is a significant difference between GME and TLE groups as regards type of non- controlled seizure patients (on monotherapy or polytherapy GME patients shows a tendency of non- controlled seizure patients on monotherapy more than TLE patients .

Test used :

Chi-square test .

$P (< 0.05) = Significant$.

$P (> 0.05) = Non- significant$.

<u>COMPLIENCE, SEIZURE CONTROL,SIDE EFFECTS &</u>
<u>HIGH SERUM LEVEL</u>

III- SIDE EFFECTS					
WITH	43	29.45	29	27.36	**NS**
WITHOUT	103	70.56	77	72.64	

This table shows, no significant difference between GME and TLE groups as regards side effects .

IV-HIGH SERUM LEVEL					
WITH	35	23.97	22	20.75	**NS**
WITHOUT	111	76.03	84	79.25	

This table shows, no significant difference between GME and TLE groups as regards high serum level .

(Table 15) <u>COMPLIENCE, SEIZURE CONTROL, SIDE EFFECTS & HIGH SERUM LEVEL</u>

<u>Test used :</u>
   <u>Chi-square test .</u>
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant .

94

4) Investigations.

a) Psychometry.

i - Cognitive functions.

COMPARISON BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN SUBWECHSELER'S

CODING TEST

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	7.1	7.0	7.9
S.D	2.4	2.4	2.5

Tests used one way ANOVA and t test

f for all groups   = 3.6   p< 0.05 sign.
t test between 1 & 3 =2.28   p< 0.05 sign.
t test between 2 & 3 =2.4   p< 0.05 sign.
t test between 1 & 2 =2.28   p> 0.05 not significant

(Table 16) CODING TEST

This table   Shows, a Significant difference between the 3
groups in one way ANOVA test and a significant increase in control
group more than GME & TLE group on T. test.

Group 1   = GME GROUP

Group 2   = TLE GROUP

Group 3   = CONTROL GROUP

95

4) Investigations.

a) Psychometry.

i - Cognitive functions.

COMPARISON BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN SUBWECHSELER'S

DIGIT SPAN TEST

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	8.6	8.3	8.6
S.D	2.5	2.7	2.4

Tests used one way ANOVA and t test

f for all groups = 0.48
t test between 1 & 3 = 0.04
t test between 2 & 3 = 0.82
t test between 1 & 2 = 0.86

$p > 0.05$ not significant
$p > 0.05$ not significant
$p > 0.05$ not significant
$p > 0.05$ not significant

(Table 17) DIGIT SPAN TEST

This table shows, no significant difference between the three groups regarding their scores in Digit span test.

Group 1 = GME GROUP

Group 2 = TLE GROUP

Group 3 = CONTROL GROUP

4) Investigations.

a) Psychometry.

i - Cognitive functions.

CORRELATION BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN SUBWECHSELER'S
SIMILARITIES

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	11.8	12.6	13.9
S.D	2.5	2.2	2.3

Tests used one way ANOVA and t test

f for all groups = 21.6   p< 0.0001 highly sign.
t test between 1 & 3 =6.3   p< 0.0001 highly sign.
t test between 2 & 3=4.1   p< 0.0001 highly sign.
t test between 1 & 2=2.4   p< 0.05 sign.

(Table 18) SIMILARITIES

This table shows, a singnificant difference between the three groups using one way ANOVA test . Both groups scored significantly less than control group. The performance of TLE group is signinficantly better than GME group in Similarities test.

Group 1   = GME GROUP
Group 2   = TLE GROUP
Group 3   = CONTROL GROUP

4) Investigations.

a) Psychometry.

i - Cognitive functions.

CORRELATION BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN
CANCELLATION LETTERS TEST (ERRORS)

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	1.7	1.6	0.16
S.D	3.2	2.3	0.39

Tests used one way ANOVA and t test

f for all groups =17.3
t test between 1 & 3 =5.7
t test between 2 & 3 =6.1
t test between 1 & 2 =0.33

$p < 0.001$ highly sign.
$p < 0.001$ highly sign.
$p < 0.001$ highly sign.
$p > 0.05$ not significant

Group 1   = GME GROUP
Group 2   = TLE GROUP
Group 3   = CONTROL GROUP

(Table 19) CANCELLATION LETTERS TEST (ERRORS)

This table shows, a significant difference between the three groups way ANOVA test .Performance of control group is significantly better than other two groups. On compared GME and TLE groups we don't find any signinficance in Cancellation Letters Test (Errors).

4) Investigations.

a) Psychometry.

i - Cognitive functions.

<u>CORRELATION BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN</u>

<u>CANCELLATION LETTERS TEST (TIME)</u>

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	2.2	2.2	2.0
S.D	0.85	0.71	0.81

<u>*Tests used one way ANOVA and t test*</u>

f for all groups =1.7   p> 0.05 not significant
t test between 1 & 3 =1.6   p> 0.05 not significant
t test between 2 & 3 =1.8   p> 0.05 not significant
t test between 1 & 2 =0.21   p> 0.05 not significant

(Table 20) <u>*CANCELLATION LETTERS TEST (TIME)*</u>

This table shows, no significant difference between the three groups inone way ANOVA test .On compared GME and TLE groups and each group with control group we don't find any signinficance in Cancellation Letters Test (time).

<u>*Group 1*</u>   = *GME GROUP*

<u>*Group 2*</u>   = *TLE GROUP*

<u>*Group 3*</u>   = *CONTROL GROUP*

4) Investigations.

a) Psychometry.

i - Cognitive functions.

<u>CORRELATION BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN</u>

MACHING FAMILIAR FIGURE TEST -TOTAL
(MFFT)

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	10.3	11.6	11.4
S.D	4.0	6.3	4.2

<u>Tests used one way ANOVA and t test</u>

f for all groups = 2.6 $p > 0.05$ not significant

t test between 1 & 3=2.07 $p < 0.05$ sign.

t test between 2 & 3=0.2 $p > 0.05$ not significant

t test between 1 & 2=1.8 $p > 0.05$ not significant

(Table 21) MACHING FAMILIAR FIGURE TEST-TOTAL ERRORS (MFFT)

This table shows, no significant difference between the three groups in one way ANOVA test .Performance of GME group is significantly better than control group. On compared TLE , GME and control groups we don't find any signinficance in Matching F miliar Figure Test-Total

---

<u>Group 1</u> = GME GROUP

<u>Group 2</u> = TLE GROUP

<u>Group 3</u> = CONTROL GROUP

4) Investigations.

a) Psychometry.

i - Cognitive functions.

CORRELATION BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN

MACHING FAMILIAR FIGURE TEST -MEAN TIME (MFFT)

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	20.8	17.9	15.6
S.D	11.0	8.8	8.3

Tests used one way ANOVA and t test

f for all groups =8.2 $p< 0.001$ highly sign.
t test between 1 & 3 =1.1 $p< 0.001$ highly sign.
t test between 2 & 3 =1.9 $p> 0.05$ not significant
t test between 1 & 2 =2.2 $p< 0.05$ significant

(Table 22) MACHING FAMILIAR FIGURE TEST-MEAN TIME (MFFT)

This table shows, a significant difference between the three groups in one way ANOVA test .Performance of control group is significantly better than TLE group andperformance of TLE group is significantly better than GME group On compared GME, TLE amd control groups we don't find any signinficance in Matching F miliar Figure Test-   Time.

Group 1   = GME GROUP
Group 2   = TLE GROUP
Group 3   = CONTROL GROUP

4) Investigations.

a) Psychometry.

i - Cognitive functions.

**CORRELATION BETWEEN GME GROUP, TLE GROUP &
CONTROL GROUP IN**

MACHING FAMILIAR FIGURE TEST -TOTAL TIME
(MFFT)

	GME GROUP	TLE GROUP	CONTROL GROUP
MEAN	252.4	225.3	188.0
S.D	135.6	114.8	100.2

Tests used one way ANOVA and t test

f for all groups =8.08 $p < 0.001$ highly sign.
t test between 1 & 3 =4.2 $p < 0.001$ highly sign.
t test between 2 & 3 =2.4 $p < 0.05$ significant
t test between 1 & 2 =1.7 $p > 0.05$ not significant

(Table 23) MACHING FAMILIAR FIGURE TEST-TOTAL TIME (MFFT)

This table shows, a significant difference between the three groups way ANOVA test Performance of control group is significantly better than other two groups. On compared GME and TLE groups we don't find any signinficance in Maching Familiar Figure Test-Total Time (MFFT).

Group 1   = GME GROUP
Group 2   = TLE GROUP
Group 3   = CONTROL GROUP

THE CHILDREN DEPRESSION INVENTORY (CDI)

	GME GROUP	TEL GROUP	CONTROL GROUP
MEAN	12.7	12.	12.6
S.D	5.0	5.8	5.6

f test = .60   P > 0.05 NS

This table shows,non siginificant difference between the scores of the three groups using one way ANOVA test. in the children Depression inventory (CDI)

(Table 24) F, MEAN AND SD OF DIFFERENT GROUPS OF (CDI)

THE CHILDREN ANXIETY SCALE (CAS)

	GME GROUP	TEL GROUP	CONTROL GROUP
MEAN	17.0	16.8	16.6
S.D	6.7	6.8	6.7

f test = 2.1   P > 0.05 NS

(Table 25) F, MEAN AND SD OF DIFFERENT GROUPS OF (CAS)

This table shows,non siginificant difference between the scores of the three groups using one way ANOVA test. in the children Anxiety   scale (CAS)

Group 1   = GME GROUP
Group 2   = TLE GROUP
Group 3   = CONTROL GROUP

Fig. (3):Comparison between GME group TLE group & control group in the children anxiety scale

Anxiety +ve
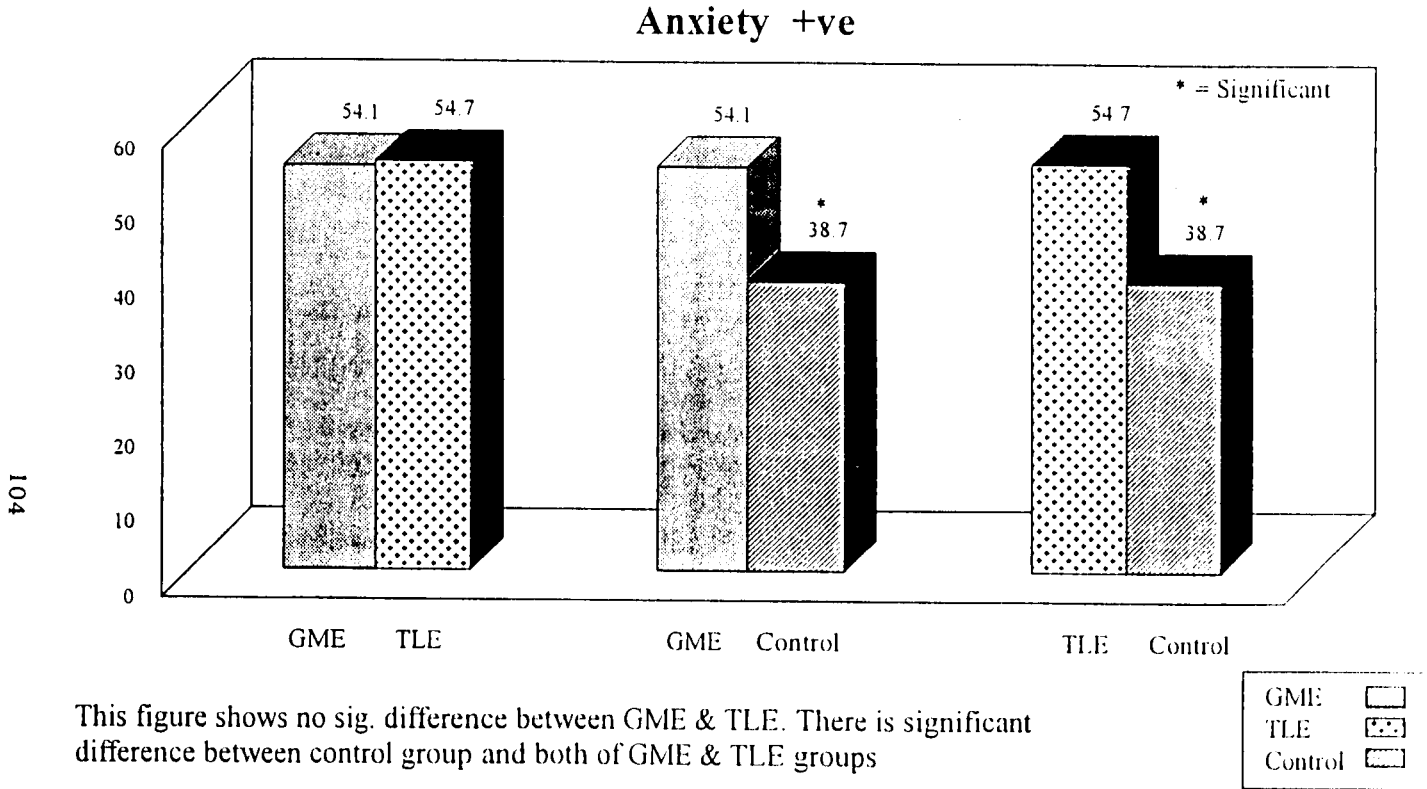
This figure shows no sig. difference between GME & TLE. There is significant difference between control group and both of GME & TLE groups

Test used :

Chi-square test .

$P (< 0.05) = Significant$.

$P (> 0.05) = Non- significant$.

Fig. (4):Comparison between GME group TLE group & control group in the children depression inventory (CDI)

Depression +ve

This figure shows no sig. difference between GME, TLE & control groups
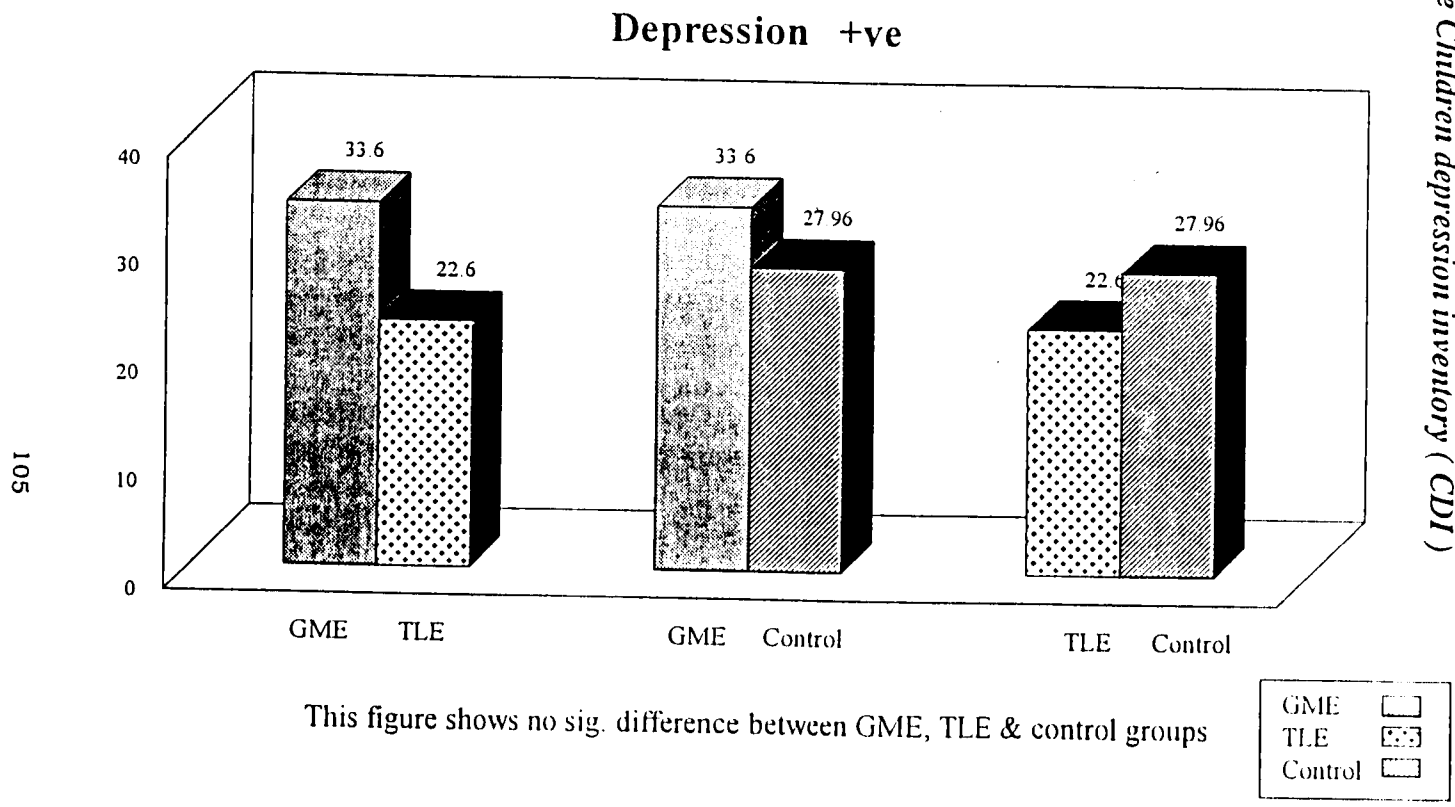
Test used :
   Chi-square test ,
P (< 0.05) = Significant .

iii - Assessement of personality.

THE VARIABLE	GME		TLE		CONTROL		P VALUE
	NO=146	%	NO=106	%	NO=93	%	·
NEUROTICISM	28	19.2	17	16	6	6.5	NS
PSYCHOTOCISM	38	26	34	32.1	22	23.7	NS
INTEROVERSION	67	45.9	57	53.8	49	52.7	NS
LIESCALE	86	58.9	53	50	50	53.8	NS

(Table 26) JUNIOR EYSENK PERSONALITY QUESTIONAIRE (JEPQ)

This table shows,non siginificant difference between the scores of the three groups using *Chi-square test* .

Test used :

Chi-square test .

P (< 0 .05) = Significant .

P (> 0.05) = Non- significant .

4) Investigations.

b) EEG.

CORRELATION BETWEEN GME GROUP, & TLE GROUP CONCERNING EEG

EEG Pattern	GME		TLE	
	No = 146	%	No = 106	%
1. GENERALIZED ACTIVITY	144	78.18	0	0
2. RIGHT TEMPORAL DISCHARGES	0	0	35	33.02
3. LEFT TEMPORAL DISCHARGES	0	0	57	53.77
4. BI - TEMPORAL DISCHARGES	0	0	14	13.21
5. TEMPORAL & SECONDRY GENERALIZATION	28	19.2	0	0
6. NONE (FREE EEG)	4	2.7	0	0

(Table 27)

<u>This table shows :</u>

As regardes the EEG of the GME patients , there are 78.18 % with generalized activity , 19.2 % with Temporal discharges with secondry generalization and 2.7 without EEG changes .

As regardes the EEG of the TLE patients , there are 33.02 % with right temporal discharges , 53.77% with left temporal discharges and 13.21% bi-temporal discharges ,

1) Psychodemograhic profile.

(The relation between Psychodemographic variables and performance on different psychometric tests).

a)Psychometry.

i- Cogni tive funchtions

- Coding test

- Digit span test

- Similarities

- Cancellation letters test (Time)

- Cancellation letters test (Errors)

- Maching familiar figure test-Total and Mean time (MFFT)

- Maching familiar figure test-Total errors (MFFT)

ii- Assessement of mood.

- The Children anxiety scale (CAS).

- The children depression inventory (CDI).

iii- Assessement of personality.

- Psychoticism

- Neuroticlsm

- Extraversion

- Introversion

- Lie scale.

RESULTS:

II) Details .

1) Psychodemographic profile .

2) Illness profile .

3) Therapeutic issues .

4) Investigations .

    a) Psychometry .

    b) EEG .

3) SIB ORDER :

COMPARISON BETWEEN FIRST TWO SIB ORDER PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

Table 32) - FIRST TWO

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	ERROR
GME	81	29	16	4	14	19	42	13	12	3	35	49	26	2	4
TLE	56	27	11	0	8	9	31	18	9	1	28	29	18	2	10
P<0.05		NS	NS	NS	NS	NS	NS	0.0269	NS	NS	NS	NS	NS	NS	0.0141

This Table Shows,

Significant (P<0.05): Psychoticism & MFFT (ERROR) TEST. TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN SIB MORE > FIRST TWO ORDER PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS TIME	ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	ERROR
GME	65	29	16	0	14	13	35	18	14	4	27	35	23	0	4
TLE	50	24	12	0	5	13	26	12	8	4	27	23	6	2	6
<0.05		NS	NS	—	NS	NS	NS	NS	NS	NS	NS	NS	0.004201	NS	NS

This Table Shows,

Significant (P<0.05): Lie scale TEST. GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 33) - MORE

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

4) ROOMS:

COMPARISON BETWEEN MORE THAN TWO ROOMS PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 34)   - 1-2

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	68	21	11	1	12	13	40	14	15	1	32	38	29	2	3
TLE	72	36	16	0	8	17	40	21	11	2	43	36	15	3	10
P<0.05		0.0214	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.005457	NS	NS

This Table Shows,

Significant (P<0.05): CODING & DEPRESSION TEST. GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN (1-2) ROOMS PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 35)   - MORE

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	78	39	21	3	16	14	38	21	13	4	32	43	26	0	5
TLE	34	15	7	0	7	5	19	12	4	2	16	16	8	1	8
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

I) SEX :

COMPARISON BETWEEN MALE PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

Table 28) - MALE

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	83	39	12	1	14	15	48	19	20	2	42	62	35	3	5
TLE	56	25	12	0	7	11	35	16	12	3	27	29	10	1	8
P<0.05		N S	0.0216	N S	N S	N S	N S	0.0286	N S	N S	N S	N S	0.0206	N S	N S

This Table Shows,

Significant (P<0.05): DIGIT SPAN, Psychoticism TLE > GME & DEPRESSION TEST.   GME > TLE

(NS) Non-Significant (P>0.05) :ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TEST.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN FEMALE PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

Table 29) - FEMALE :

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	63	23	19	3	12	14	31	25	8	3	24	24	15	0	4
TLE	50	26	10	0	5	11	24	16	6	2	28	23	15	3	9
P<0.05		N S	N S	N S	N S	N S	N S	N S	0.0127	N S	N S	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05): Neuroticism TEST. GME > TLE

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESIS.
** CHI-SQUARE TESTS.

2) FAMILY SIZE :

COMPARISON BETWEEN FAMILY SIZE < 4 MEMBERS PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

Table 30) - <4

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism *	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	52	24	10	3	7	11	25	9	6	1	21	28	18	0	4
TLE	22	13	6	0	6	3	16	7	2	2	10	12	7	1	4
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE.
NS) Non-Significant (P>0.05) :ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN FAMILY SIZE > 4 MEMBERS PATIENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

Table 31) - >4

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	94	37	22	0	21	18	53	28	24	4	42	54	33	2	5
TLE	84	37	17	0	7	17	44	22	14	3	43	42	17	4	12
P<0.05		NS	NS	—	0.0104	NS	NS	NS	NS	NS	0.0242	NS	0.0393	NS	0.0344

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS, DEPRESSION GME > TLE & MFFT (ERROR) TEST, INTEROVERSION TLE > GME
NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

5) WANTED:

COMPARISON BETWEEN PATIENTS WHO ARE WANTED BY PARENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 36) - YES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	124	54	24	3	23	25	60	33	19	4	57	70	45	1	7
TLE	69	31	11	0	7	16	38	15	10	4	34	34	13	1	8
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0113	NS	NS

This Table Shows,

Significant (P<0.05): Depression TEST. GME > TLE
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN PATIENTS WHO ARE NOT WANTED BY PARENTS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 37) - NO

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism *	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	22	8	7	0	4	5	16	4	4	1	8	9	7	1	0
TLE	37	21	13	0	5	6	23	16	7	2	22	18	11	2	7
P<0.05		NS	NS	—	NS	NS	NS	0.0492	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): Psychoticism TEST. TLE > GME
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

6) CLASS

COMPARISON BETWEEN RURAL PATIENTS & DIFFERENT
PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 38)   – RURAL

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	89	38	19	2	15	12	48	19	15	3	37	53	30	0	5
TLE	63	32	16	0	7	10	32	20	9	2	36	31	16	3	14
P<0.05		N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	0.038	0.002294

This Table Shows,

Significant (P<0.05):MFFT (TIME) & (ERROR) TESTS. TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
*   FISHER EXACT TESTS.
**   CHI-SQUARE TESTS.

COMPARISON BETWEEN URBAN PATIENTS & DIFFERENT
PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 39)   – URBAN

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	57	23	13	2	13	19	30	17	12	3	27	30	20	2	2
TLE	43	19	6	0	4	11	24	11	8	2	19	20	10	1	3
P<0.05		N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
*   FISHER EXACT TESTS.
**   CHI-SQUARE TESTS.

7) PARENTAL SEPARATION

COMPARISON BETWEEN PATIENTS SUFFERING FROM PARENTAL SEPARATION & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 40) - YES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Tie scale **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	32	11	6	1	2	5	18	4	8	1	17	19	8	0	1
TLE	33	16	7	0	3	7	18	9	7	1	16	12	0	0	4
P<0.05	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.008151	-	NS

This Table Shows,

Significant (P<0.05): DEPRESSION TEST. GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN PATIENTS NOT SUFFERING FROM PARENTAL SEPARATION & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 41) - NO

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Tie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	101	46	25	3	25	23	55	27	19	4	47	56	42	2	8
TLE	73	36	15	0	10	11	40	23	11	3	40	41	25	4	12
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

8) FAMILY INCOME :

COMPARISON BETWEEN PATIENTS WITH SUFFICIENT FAMILIAL INCOME & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 42) – HIGH

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism *	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
GME	91	36	18	3	15	18	44	24	16	2	41	51	35	2	7
TLE	43	19	10	0	3	7	24	15	5	2	26	22	9	3	8
P<0.05		0.006249	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0437	NS	NS

This Table Shows,

Significant (P<0.05):DEPRESSION TEST. GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN PATIENTS WITHOUT SUFFICIENT FAMILIAL INCOME & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 43) – LOW

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
GME	55	25	14	1	13	10	32	12	12	4	22	29	17	0	2
TLE	63	33	13	0	10	12	35	19	12	1	30	31	16	1	7
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05):NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

9) EDUCATED PARENTS:

COMPARISON BETWEEN PATIENTS WITH EDUCATIONAL PARENTS
& DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

Table 44)   - YES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	68	28	18	1	16	14	35	16	15	3	28	33	21	2	4
TLE	61	30	15	0	6	8	36	23	11	4	29	29	16	3	10
P<0.05		N S	N S	N S	0.0390	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05):CANCELLATION LETTERS (TIME) TEST. GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER EXACT TESTS.
**   CHI-SQUARE   TESTS.

COMPARISON BETWEEN PATIENTS WITHOUT EDUCATIONAL PARENTS
& DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

Table 45)   - NO

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	78	34	14	3	13	15	46	21	15	2	37	47	32	0	4
TLE	45	21	8	0	7	11	23	10	6	1	26	23	8	1	6
P<0.05		0.006248	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	0.008024	N S	N S

This Table Shows,

Significant (P<0.05):DEPRESSION & CODING TESTS . GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER EXACT TESTS.
**   CHI-SQUARE   TESTS.

PATIENTS WITH ONSET OF SEIZURES <2Y

(Table 46) < 2 YEARS

	NO	CODING *	DIGIT SPAN . .	SIMILARITIES	CANCELLATION LETTER		ANXIET	sychoticis *	Neuroticism *	xteroversio	nteroversion * *	Lie scale	Depression	MFFT	
					TIME	ERROR								TIME	ERROR
					2	2	0	4	0	0	4	2	0	0	2
GME	4	2	2	0	2	2	0	4	0	0	4	2	0	0	0
TLE	5	0	0	0	0	0	0	2	2	0	5	2	0	0	NS
P<0.05	NS	NS	NS	—	NS	NS	—	NS	NS	—	NS	NS	—	—	

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

PATIENTS WITH ONSET OF SEIZURES (2-4Y)

(Table 47) (2-4) YEARS

	NO	CODING * *	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET * *	sychoticis *	Neuroticism *	xteroversio *	nteroversion * *	Lie scale * *	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
					4	0	6	4	6	0	4	10	2	0	0
GME	14	6	2	0	4	0	6	4	6	0	4	10	2	0	0
TLE	16	7	0	0	2	5	7	5	2	2	9	5	2	0	0
P<0.05	NS	NS	NS	—	NS	NS	NS	NS	NS	NS	NS	0.0281	NS	—	—

This Table Shows,
Significant (P<0.05): LIE SCALE TEST.   GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 48) ﹥ 4 YEARS

	NO	CODING ••	DIGIT SPAN ••	SIMILARITIES •	CANCELLATION LETTER		ANXIET ••	sychoticis ••	Neuroticism ••	xteroversio •	nteroversion ••	t le scale ••	Depression ••	MFFT	
					TIME	ERROR								TIME	ERROR
GME	128	44	24	4	14	28	76	32	34	2	62	74	46	4	6
TLE	85	51	18	0	7	14	55	28	14	2	55	57	35	0	7
P<0.05		0.0002293	N S	N S	N S	N S	N S	N S	N S	N S	0.0194	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05): CODING & INTEROVERSION TESTS.   TLE ﹥ GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
•   FISHER EXACT TESTS.
••   CHI-SQUARE TESTS.

2) TYPES OF ONSET :

(Table 49) ABRUPT

PATIENTS WITH ABRUPT ONSET

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio *	interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	38	7	7	0	7	7	19	11	11	0	11	7	11	0	4
TLE	37	21	0	0	5	5	16	11	11	5	27	21	11	0	0
P<0.05		0.0008	0.019	—	N S	N S	N S	N S	N S	N S	0.0001374	0.0006	N S	—	N S

This Table Shows,

Significant (P<0.05): CODING , INTEROVERSION & Lie scale TEST. TLE > GME   DIGIT SPAN, GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 50) GRADUAL

PATIENTS WITH GRADUAL ONSET WITH PRECEPITATING FACTORS

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio *	interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	108	44	15	4	22	19	74	37	22	0	59	56	30	0	11
TLE	69	42	5	0	5	16	32	16	5	5	58	37	11	0	0
P<0.05		8.97E-03	N S	N S	1.79E-02	N S	0.003376	N S	0.0179	0.0177	0.00006187	N S	N S	---	0.0156

This Table Shows,

Significant (P<0.05): CODING , TLE > GME CANCELLATION LETTERS (TIME), ANXIETY, Neuroticism,
                Exteroversion, INTEROVERSION & MFFT (ERROR) TEST.   GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

1) Psychodemograhic profile (detailed)

(The relation between Psychodemographic variables and performance on different psychometric tests).

a) Psychometry.

(Tables from number 28 till 45)

i- Cognitive funchtions

Coding test

Non-Significant :

No Signifcant difference was found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female. (Tables 28, 29)

Family Size : more than 4 members or less (Tables 30, 31)

Sib-order : First two or more (Tables 32, 33)

Rooms : more than two (Table 35)

Wanted : Whether child was wanted or not (Tables 36, 37)

Residence : Rural or Urban (Tables 38, 39)

Parentral Separation : Whether present or not (Tables 40, 41)

Family income : low (Table 43)

Educated parents : educated (Table 44).

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME	*in high family income (Table 42)*
GME > TLE	*in patients live in up to two rooms (Table. 34) and patients with non educated parents (Table 45).*

Digit span test

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : *Female patients (Table 29)*

Family Size : *more than 4 members or less (Tables 30, 31)*

Sib-order : *First two or more (Tables 32, 33)*

Rooms : *Up to two rooms or more (Tables 34, 35)*

Wanted : *Whether child was wanted or not (Tables 36, 37)*

Residence : *Rural or Urban (Tables 38, 39)*

Parentral Separation : *Whether present or not (Tables 40, 41)*

Family income : *whether high or low (Tables 42, 43)*

Educated parents : *Whether educated or not (Tables 44, 45)*

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME   *Male patients (Table 28)*

Similarities

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female (Tables 28, 29)

Family Size : more than 4 members or less (Tables 30, 31)

Sib-order : First two or more (Tables 32, 33)

Rooms : Up to two rooms or more (Tables 34, 35)   '

Wanted : Whether child was wanted or not (Tables 36, 37)

Residence : Rural or Urban (Tables 38, 39)

Parentral Separation : Whether present or not (Tables 40, 41)

Family income : whether high or low (Tables 42, 43)

Educated parents : Whether educated or not (Tables 44, 45)

Cancellation letters test (Time)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female (Tables 28, 29)

Family Size : less than four members (Table 30)

Sib-order : First two or more (Tables 32, 33)

Rooms : Up to two rooms or more (Tables 34, 35)

Wanted : Whether child was wanted or not (Tables 36, 37)

Residence : Rural or Urban (Tables 38, 39)

Parentral Separation : Whether present or not (Tables 40, 41)

Family income : whether high or low (Tables 42, 43)

Educated parents : not (Table 45)

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE   in patients with big family size (more than 4 members) (Table 31), and with educated parents (Table 44).

Cancellation letters test (Errors)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female (Tables 28. 29)

Family Size : more than 4 members or less (Tables 30. 31)

Sib-order : First two or more (Tables 32. 33)

Rooms : Up to two rooms or more (Tables 34. 35)   '

Wanted : Whether child was wanted or not (Tables 36. 37)

Residence : Rural or Urban (Tables 38. 39)

Parentral Separation : Whether present or not (Tables 40. 41)

Family income : whether high or low (Tables 42. 43)

Educated parents : Whether educated or not (Tables 44. 45)

Matching familiar figure test-Total and Mean time (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female (Tables 28, 29)

Family Size : more than 4 members or less (Tables 30, 31)

Sib-order : First two or more (Tables 32, 33)

Rooms : Up to two rooms or more (Tables 34, 35)

Wanted : Whether child was wanted or not (Tables 36, 37)

Residence : Urban (Table 39)

Parentral Separation : Whether present or not (Tables 40, 41)

Family income : whether high or low (Tables 42, 43)

Educated parents : Whether educated or not (Tables 44, 45)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME   In rural residence (Table 38)

Matching familiar figure test-
Total errors (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female (Tables 28. 29)

Family Size : less than 4 members (Table 30)

Sib-order : More than two (Table 33)

Rooms : Up to two rooms or more (Tables 34. 35)

Wanted : Whether child was wanted or not (Tables 36. 37)

Residence : Urban (Table 39)

Parentral Separation : Whether present or not (Tables 40. 41)

Family income : whether high or low (Tables 42. 43)

Educated parents : Whether educated or not (Tables 44, 45)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME In big family size (Table 31). in rural residence (Table 38) and first two sib-order (Table 32).

ii- Assessement of mood.

The Children anxiety scale (CAS).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female (Tables 28, 29)

Family Size : more than 4 members or less (Tables 30, 31)

Sib-order : First two or more (Tables 32, 33)

Rooms : Up to two rooms or more (Tables 34, 35)

Wanted : Whether child was wanted or not (Tables 36, 37)

Residence : Rural or Urban (Tables 38, 39)

Parentral Separation : Whether present or not (Tables 40, 41)

Family income : whether high or low (Tables 42, 43)

Educated parents : Whether educated or not (Tables 44, 45)

The children depression inventory (CDI).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Female patients (Table 29)

Family Size : Less than 4 members (Table 30)

Sib-order : First two (Table 32)

Rooms : More than two rooms (Table 35)

Wanted Child : Not (Table 37)

Residence : Rural or Urban (Tables 38. 39)

Parentral Separation : Not (Table 41)

Family income : Low (Table 43)

Educated parents : Educated (Table 44)

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE Male patients (Table 28), big family size (more than four members) (Table 31), sib-order (more than two) (Table 33), low rooms numbers (up to two rooms) (Table 34), wanted child (Table 36), patients with parentral separation (Table 40), patients with high family income (Table 42), and patients with non educated parents (Table 45).

129

iii- Assessement of personality.
Psychoticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Female patients (Table 29)

Family Size : more than 4 members or less (Tables 30, 31)

Sib-order : First two or more (Table 33)

Rooms : Up to two rooms or more (Tables 34, 35)

Wanted : Wanted child (Table 36)

Residence : Rural or Urban (Tables 38, 39)

Parentral Separation : Whether present or not (Tables 40, 41)

Family income : whether high or low (Tables 42, 43)

Educated parents : Whether educated or not (Tables 44, 45)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME In male patients (Table 28), first two sib-order patients (Table 32), and non wanted child during pregnancy (Table 37).

Neuroticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

> **Gender** : Male patients (Table 29)
>
> **Family Size** : more than 4 members or less (Tables 30. 31)
>
> **Sib-order** : First two or more (Tables 32. 33)
>
> **Rooms** : Up to two rooms or more (Tables 34. 35)
>
> **Wanted** : Whether child was wanted or not (Tables 36. 37)
>
> **Residence** : Rural or Urban (Tables 38. 39)
>
> **Parentral Separation** : Whether present or not (Tables 40. 41)
>
> **Family income** : whether high or low (Tables 42. 43)
>
> **Educated parents** : Whether educated or not (Tables 44, 45)

Significant :

There is significant difference between GME and TLE in the following :

> **GME > TLE**   Female patients.   (Table 29)

Introversion

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the psychodemographic profile in the following :

Gender : Male or Female patients (Tables 28, 29)

Family Size : Less than 4 members (Table 30)

Sib-order : First two or more (Tables 32. 33)

Rooms : Up to two rooms or more (Tables 34. 35)

Wanted : Whether child was wanted or not (Tables 36, 37)

Residence : Rural or Urban (Tables 38. 39)

Parentral Separation : Whether present or not (Tables 40. 41)

Family income : whether high or low (Tables 42, 43)

Educated parents : Whether educated or not (Tables 44, 45)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME In big family size (more than four members) (Table 31).

Lie scale

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between **GME** and **TLE** concerning the psychodemographic profile in the following :

Gender : Male or Female patients (Tables 28. 29)

Family Size : more than 4 members or less (Tables 30. 31)

Sib-order : First two or more (Tables 32. 33)

Rooms : Up to two rooms or more (Tables 34. 35)

Wanted : Whether child was wanted or not (Tables 36. 37)

Residence : Rural or Urban (Tables 38. 39)

Parentral Separation : Whether present or not (Tables 40. 41)

Family income : whether high or low (Tables 42. 43)

Educated parents : Whether educated or not (Tables 44. 45)

2) Illness profile.

(The relation between Illness variables and performance on different psychometric tests).

a) Psychometry.

i- Cognitive funchtions

- Coding test

- Digit span test

- Similarities

- Cancellation letters test (Time)

- Cancellation letters test (Errors)

- Maching familiar figure test-Total and Mean time (MFFT)

- Maching familiar figure test-Total errors (MFFT)

ii- Assessement of mood.

- The Children anxiety scale (CAS).

- The children depression inventory (CDI).

iii- Assessement of personality.

- Psychoticism

- Neturoticlsm

- Extraversion

- Introversion

- Lie scale

1) PAST SEIZURE FREQUENCY :

PAST. SEIZURE EVERY DAY (AT ONSET OF DISEASE)

Table 51) FREQUENT / DAY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	PSYCHOTIS **	NEUROTIC.	Ieroversl *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	45	19	13	2	7	4	22	11	4	0	26	22	11	0	4
TLE	62	34	10	0	5	16	39	21	8	5	42	36	29	0	8
P<0.05		NS	NS	NS	NS	0.0267	NS	NS	NS	NS	NS	NS	0.0184	---	NS

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS (ERROR) & DEPRESSION TEST.   TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

PAST. SEIZURE EVERY WEEK AT ONSET OF DISEASE

Table 52) FREQUENT / WEEK

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	PSYCHOTIS *	NEUROTIC. *	Ieroversl	Interoversion **	Lie scale **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	34	9	7	2	7	9	19	9	11	0	13	24	13	0	0
TLE	10	5	0	0	3	3	5	3	0	0	8	8	3	0	0
P<0.05		NS	NS	NS	0.0354	NS	NS	NS	NS	---	0.0495	NS	NS	--	--

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS (TIME) & Interoversion TEST.   TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 53) FREQUENT / MONTH

PAST. SEIZURE FREQ EVERY MONTH AT ONSET OF DISEASE

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion	Introversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	67	24	4	0	9	13	44	18	18	0	29	35	22	4	4
TLE	34	18	5	0	5	3	10	10	5	0	23	23	8	0	0
P<0.05		NS	NS	—	NS	NS	0.0005557	NS	NS	—	NS	NS	NS	NS	NS

This Table Shows,
Significant (P<0.05): ANXIETY TEST.   TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

2) CURRENT SEIZURE FREQUENCY :

Table 54) FREQUENT / DAY

CURRENT SEIZURE FREQUENCY / DAY

	NO	CODING *	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticis *	Neuroticism *.	xteroversio	Interoversion * .	Lie scale **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	9	0	2	0	0	2	7	0	2	0	0	7	0	0	2
TLE	24	21	3	0	3	5	21	5	0	0	13	13	8	0	0
P<0.05		NS	NS	—	NS	NS	NS	NS	NS	—	0.0148	NS	NS	—	NS

This Table Shows,

Significant (P<0.05): INTEROVERSION TEST.   TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER EXACT TESTS.
**   CHI-SQUARE TESTS.

CURRENT SEIZURE FREQUENCY / MONTH

(Table 55) FREQUENT / MONTH

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism *	xteroversio	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	44	18	7	2	9	14	28	9	5	0	25	30	12	2	2
TLE	24	8	3	0	3	5	13	5	3	0	19	19	8	0	0
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	—	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER EXACT TESTS.
**   CHI-SQUARE TESTS.

CURRENT SEIZURE FREQUENCY / WEEK

(Table 56) FREQUENT / WEEK

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio *	nteroversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	21	12	5	2	2	5	9	7	5	0	7	9	5	0	0
TLE	24	16	3	0	3	5	5	11	8	3	8	13	13	0	0
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0381	---	---

This Table Shows,   *   **

Significant (P<0.05): DEPRESSION TEST.   TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER   EXACT   TESTS.
**   CHI-SQUARE   TESTS.

NO CURRENT SEIZURE FREQUENCY ·

(Table 57) NONE

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism *	xteroversio *	nteroversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	72	23	9	0	12	7	41	18	18	2	30	37	28	0	5
TLE	34	13	8	0	3	8	13	11	3	3	21	19	8	0	0
P<0.05		NS	NS	---	NS	NS	NS	NS	NS	NS	NS	NS	NS	---	NS

This Table Shows,   *   **

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER   EXACT   TESTS.
**   CHI-SQUARE   TESTS.

PATIENTS WITH DURATION OF ILLNESS <6 MONTHS

Table 58) <6 MONTHS

	NO	CODING *	DIGIT SPAN .*	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicls **	Neuroticlsn **	xteroverslo *	nteroverslon *	Lle scale **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	27	8	2	2	0	4	23	4	13	0	15	15	8	2	2
TLE	9	0	2	0	2	0	9	5	5	2	2	5	0	0	0
P<0.05		N S	N S	N S	N S	N S	N S	0.0455	N S	N S	N S	N S	N S	II S	N S

This Table Shows,

Significant (P<0.05): Psychoticism Test.   TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER   EXACT   TESTS.
**   CHI-SQUARE   TESTS.

PATIENTS WITH DURATION OF ILLNESS (6 MONTHS - 2YEARS)

ble 59) (6 MONTHS - 2 YEARS)

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicls **	Neuroticlsn *	xteroverslo *	nteroverslon **	Lle scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	42	19	8	2	8	4	21	15	8	2	19	21	21	2	0
TLE	23	12	2	0	2	5	14	7	2	0	12	16	12	0	5
P<0.05	-	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S	II S	0.00/85

This Table Shows,

Significant (P<0.05): MFFT (ERROR) TEST.   TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER   EXACT   TESTS.
**   CHI-SQUARE   TESTS.

Table 60) (2 YEARS - 5 YEARS)

PATIENTS WITH DURATION OF ILLNESS (2 YEARS - 5YEARS)

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism	xteroverslo	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	42	10	6	0	6	15	25	10	6	0	23	25	13	0	4
TLE	47	35	14	0	5	9	28	14	5	0	35	30	14	0	2
P<0.05		1.825E-08	N S	—	N S	N S	N S	N S	N S	—	N S	N S	N S	—	N S

This Table Shows,

Significant (P<0.05): CODING TEST. TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 61) > 5 YEARS

PATIENTS WITH DURATION OF ILLNESS > 5YEARS

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroverslo *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	35	19	10	0	6	2	19	10	6	0	13	23	8	0	2
TLE	27	12	0	0	2	7	12	9	5	2	18	14	7	0	0
P<0.05		0.007262	N S	—	N S	N S	N S	N S	N S	N S	0.0212	N S	N S	—	N S

This Table Shows,

Significant (P<0.05): CODING GME > TLE & INTEROVERSION TEST. TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

2) LONGEST SEIZURE FOR PERIOD :

LONGEST SEIZURE FREE PERIOD < 6 Months

(Table 62) < 6 MONTHS

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psycholicism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	80	31	18	5	8	16	49	16	18	3	34	52	18	5	3
TLE	81	45	3	0	6	18	42	21	9	9	48	51	27	0	6
P<0.05		0.0327	0.0003989	0.0284	N S	N S	N S	N S	N S	N S	0.0334	N S	N S	0.0284	N S

This Table Shows,

Significant (P<0.05): CODING, Interoversion TLE > GME   DIGIT SPAN, SIMILARITIES, & MFFT (TIME) TEST. GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESIS.
** CHI-SQUARE TESIS.

LONGEST SEIZURE FREE PERIOD (6 Months -2 Years)

(Table 63) (6 MONTHS - 2 YEARS)

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	66	21	10	0	16	13	34	21	13	0	29	34	16	0	3
TLE	25	15	6	0	3	3	12	12	6	0	18	12	9	0	0
P<0.05		0.0141	N S	—	N S	N S	N S	N S	N S	—	0.0168	N S	N S		N S

This Table Shows,

Significant (P<0.05): CODING & Interoversion TEST. TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESIS.
** CHI-SQUARE TESIS.

1) STATUS EPILEPTICUS HISTORY

PATIENTS WITH HISTORY OF STATUS EPILEPTICOUS

(Table 64) WITH

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET *	sychoticis *	Neuroticisn *	xteroversio *	Interoversion	Lie scale	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	13	8	2	0	4	2	2	4	2	0	6	6	2	0	0
TLE	18	9	2	0	2	5	14	2	2	2	9	12	9	0	0
P<0.05		N S	N S	—	N S	N S	N S	N S	N S	N S	N S	N S	0 0468	- -	—

This Table Shows,

Significant (P<0.05): DEPRESSION TEST.   TLE > GME
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

PATIENTS WITH NO HISTORY OF STATUS EPILEPTICOUS

(Table 65) WITHOUT

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticisn **	xteroversio *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	133	44	25	4	36	25	84	31	34	2	51	76	44	4	10
TLE	88	44	12	0	9	16	51	37	14	2	62	46	25	0	7
P<0.05		0 0119	N S	N S	0 00234	N S	N S	0 003133	N S	N S	0 0000029047	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS (TIME) GME > TLE , Psychoticism & INTEROVERSION TESTS. CODING, TLE > GME
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

2) POSITIVE FAMILY HISTORY :

(Table 66) WITH

PATIENTS WITH POSITIVE FAMILY HISTORY

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticls **	Neuroticism *	xteroverslo *	nteroverslon **	Lie scale **	Depresslon	MFFT	
					TIME	ERROR								TIME	ERROR
GME	27	12	5	3	5	5	16	5	5	0	21	14	12	0	3
TLE	21	8	0	0	3	3	11	8	3	3	16	8	5	0	3
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS		NS

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

143

(Table 67) WITHOUT

PATIENTS WITH NEGATIVE FAMILY HISTORY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticls **	Neuroticism **	xteroverslo *	nteroverslon **	Lie scale **	Depresslon **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	119	38	19	5	19	24	72	27	24	3	43	60	36	3	7
TLE	85	46	11	0	8	13	46	19	8	0	51	46	24	0	3
P<0.05		0.001500	NS	NS	NS	NS	NS	NS	0.0344	NS	0.0007470	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): CODING & INTEROVERSION TESTS. TLE > GME , Neuroticism, GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

3) POSITIVE PAST HISTORY :

WITHOUT POSITIVE PAST HISTORY

(Table 68) WITH

| | NO | CODING | DIGIT SPAN | SIMILARITIES | CANCELLATION LETTERS | | ANXIETY | Psychoticism | Neuroticism | Exteroversion | Interoversion | Lie scale | Depression | MFFT | |
		**	**	*	TIME	ERROR	**	**	**	*	**	**	**	TIME	ERROR
GME	143	48	25	8	23	25	85	33	28	3	60	83	38	3	10
TLE	98	48	13	0	13	19	59	27	13	3	63	54	38	0	8
P<0.05		0.0164	NS	0.0439	NS	NS	NS	NS	NS	NS	0.0006593	NS	0.0482	NS	NS

This Table Shows,

Significant (P<0.05): CODING, SIMILARITIES, Interoversion & Depression TEST.
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

WITH POSITIVE PAST HISTORY

(Table 69) WITHOUT

| | NO | CODING | DIGIT SPAN | SIMILARITIES | CANCELLATION LETTERS | | ANXIETY | Psychoticism | Neuroticism | Exteroversion | Interoversion | Lie scale | Depression | MFFT | |
		*			TIME	ERROR	*				*	*	*	TIME	ERROR
GME	3	0	0	0	0	3	0	0	0	0	0	0	0	0	0
TLE	8	3	0	0	0	5	5	0	0	0	5	5	5	0	0
P<0.05		NS	—	—	—	0.0009111	NS	—	—	—	NS	NS	NS		

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS (ERROR) TEST.
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

8) WITH ORGANIC DISEASE :

COMPARISON BETWEEN PATIENTS WITH HISTORY ORGANIC DISEASE
& DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 70) - YES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism *	Exteroversion *	Interoversion **	Lie scale	Depression **	MFFT TIME	MFFT ERROR
GME	104	47	26	4	23	25	55	26	18	5	44	56	35	1	8
TLE	58	28	7	0	5	8	33	16	9	1	29	34	16	1	6
P<0.05		NS	NS	NS	0.0294	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS (TIME) TEST. GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN PATIENTS WITHOUT HISTORY ORGANIC DISEASE
& DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 71) - NO

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
GME	42	12	6	0	4	4	19	12	11	0	20	24	11	1	1
TLE	48	24	16	1	7	12	23	17	8	2	28	19	9	3	9
P<0.05		0.0384	0.0359	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0333

This Table Shows,
Significant (P<0.05): CODING, DIGIT SPAN, & MFFT (ERROR) TEST. TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

9) PREGNANCY TROUBLES :

COMPARISON BETWEEN PATIENTS WITH HISTORY OF PREGNANCY & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 72) - YES

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psycholicism *	Neuroticism *	Exteroversion *	Interoversion **	Lie scale **	Depression *	MFFT TIME	MFFT ERROR
GME	21	8	4	0	2	4	12	4	5	0	12	13	5	0	0
TLE	25	14	9	0	5	6	14	9	5	1	13	6	1	0	4
P<0.05		N S	N S	—	N S	N S	N S	N S	N S	N S	N S	0.00930	N S	---	N S

This Table Shows,

Significant (P<0.05): Lie scale TEST.   GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN PATIENTS WITHOUT HISTORY OF PREGNANCY & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 73) - NO

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psycholicism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
GME	125	54	27	4	28	26	65	35	22	5	53	70	43	2	6
TLE	81	38	14	1	8	15	44	23	12	3	45	46	22	3	10
P<0.05		N S	N S	N S	0.0391	N S	N S	N S	N S	N S	N S	N S	N S	N S	0.0481

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS (TIME) GME > TLE   & MFFT (ERROR) TEST.   TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

10) LABOUR TROUBLES :

COMPARISON BETWEEN PATIENTS WITH LABOUR TROUBLES & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 74) - YES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
GME	28	11	5	0	5	7	14	8	5	2	12	10	9	0	2
TLE	30	16	9	0	4	9	18	13	6	1	17	7	4	0	3
P<0.05		NS	NS	—	NS	NS	NS	NS	NS	NS	NS	NS	NS	—	NS

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN PATIENTS WITHOUT LABOUR TROUBLES & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 75) - NO

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
GME	118	51	27	4	28	23	65	29	22	3	51	63	44	2	5
TLE	76	36	14	0	9	16	38	19	10	5	42	42	21	4	10
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0232

This Table Shows,

Significant (P<0.05): MFFT (TIME) TEST. TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II) NEUROTIC TRAITS :

COMPARISON BETWEEN PATIENTS WITH NEUROTIC TRAITS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 76) - YES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	119	58	27	3	26	26	66	28	24	5	53	68	44	1	7
TLE	80	40	14	0	9	16	46	25	11	4	39	40	19	3	10
P<0.05		0.006249	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0448	NS	NS

This Table Shows,

Significant (P<0.05):DEPRESSION TEST. GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

COMPARISON BETWEEN PATIENTS WITHOUT NEUROTIC TRAITS & DIFFERENT PSYCHOLOGICAL & COGNITIVE SCALES CONCERNING GME & TLE GROUPS

(Table 77) - No

	NO	CODING *	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism *	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	27	3	4	0	2	4	12	8	4	0	12	13	8	1	1
TLE	27	12	10	0	5	4	16	8	6	2	18	12	5	1	8
P<0.05		0.006249	NS	—	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0285

This Table Shows,

Significant (P<0.05):CODING GME > TLE & MFFT (ERROR) TEST. TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

1) PRODROMA:

- WITH

- WITHOUT

(Table 78)   PATIENTS WITH PRODROMA

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	nteroversion **	Lle scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	54	18	8	0	10	12	32	6	6	0	24	30	8	0	6
TLE	64	32	5	0	5	11	35	13	8	3	46	40	16	0	5
P<0.05		N S	N S	—	N S	N S	N S	2 92E-04	N S	N S	2.51E-03	N S	N S	—	N S

This Table Shows,

Significant (P<0.05): INTEROVERSION & Psychoticism TEST.   TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER   EXACT   TESTS.
**   CHI-SQUARE   TESTS.

(Table 79) PATIENTS WITH AURA

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	nteroversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	86	28	18	2	18	16	50	18	14	0	32	44	20	0	6
TLE	101	57	13	0	11	16	54	30	11	5	72	57	27	0	8
P<0.05		3.25E-04	N S	N S	N S	N S	N S	N S	N S	N S	0.0000029+6	N S	N S	--	N S

This Table Shows,

Significant (P<0.05): CODING & Interoversion TEST. TLE > GME
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER EXACT TESTS.
**   CHI-SQUARE   TESTS.

(Table 80) PATIENTS WITH ICTAL CHANGES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER TIME	CANCELLATION LETTER ERROR	ANXIET **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
GME	113	44	23	6	19	19	67	27	21	0	46	59	29	0	8
TLE	101	58	13	0	13	13	60	30	11	3	67	60	32	0	8
P<0.05		0.00001908	N S	N S	N S	N S	N S	0.0237	N S	N S	0.002919	N S	N S	---	N S

This Table Shows,

Significant (P<0.05); CODING, Psychoticism & Interoversion TESTS TLE > GME

(NS) Non-Significant (P>0.05) ; ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 81) PATIENTS WITH POST-ICTAL CHANGES

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	Interoversion	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	117	44	23	6	19	19	71	23	21	0	50	62	31	4	8
TLE	101	57	16	0	11	16	57	32	11	3	73	59	30	0	8
P<0.05		5.44E-03	N S	N S	N S	N S	N S	1.19E-06	N S	N S	1.152E-05	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05): CODING, Neuroticism, Psychoticism & INTEROVERSION TESTS. TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

– *WITH*

– *WITHOUT*

(Table 82) . <u>PATIENTS WITH FACTORS AFFECTING ICTUS</u>

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	nteroversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	113	40	21	6	19	19	63	27	23	2	52	50	31	0	8
TLE	83	37	8	0	5	13	51	21	8	3	57	46	27	0	5
P<0.05	N S	N S	N S	N S	0.0228	N S	N S	N S	0.0422	N S	0.001608	N S	N S	--	N S

This Table Shows,   **   **

Significant (P<0.05): CANCELLATION LETTERS (TIME), Neuroticism, Psychoticism GME > TLE & INTEROVERSION TESTS.   TLE > GME
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
*   FISHER EXACT TESTS.
**   CHI-SQUARE   TESTS.

(Table 83) PATIENTS WITH CEREBRAL AETIOLOGY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	84	41	19	3	14	16	46	16	14	0	41	39	32	0	3
TLE	38	16	6	0	3	3	9	13	6	0	22	22	16	0	3
P<0.05	NS	NS	NS	NS	NS	NS	0.001399	NS	NS	—	NS	NS	NS	----	NS

This Table Shows,

Significant (P<0.05): ANXIETY TEST. GME > TLE
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
FISHER EXACT TESTS.
CHI-SQUARE TESTS.

(Table 84) PATIENTS WITH SYSTEMIC AETIOLOGY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	89	43	22	3	22	16	54	27	19	0	38	54	22	3	3
TLE	90	58	13	0	13	16	51	26	13	3	61	54	29	0	6
P<0.05		2.96E-02	NS	NS	NS	NS	NS	NS	NS	NS	0.0007391	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): CODING & INTEROVERSION TESTS. TLE > GME
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
FISHER EXACT TESTS.
CHI-SQUARE TESTS.

3) IATROGENIC:

- WITH

- WITHOUT

(Table 85)   PATIENTS WITH IATROGENIC AETIOLOGY

	NO	CODING *	DIGIT SPAN	SIMILARITIES *	CANCELLATION LETTER		ANXIET *	sychoticis *	Neuroticism	xteroversio	nteroversion *	Lie scale *	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	16	8	0	3	5	3	11	5	0	0	5	8	8	0	0
TLE	3	3	0	0	0	0	0	0	0	0	3	3	0	0	0
P<0.05		N S	—	N S	N S	N S	N S	N S	—	—	N S	N S	N S		—

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
*   FISHER EXACT TESTS.
**   CHI-SQUARE TESTS.

2) Illness profile. (Detailed)

(The relation between Illness variables and performance on different psychometric tests).

a) Psychometry.

i- Cognitive functions

Coding test

Non Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures

Age of onset : whether below two years (Table 46), between two and four years (Table 47)

Frequencey of Seizures

Past seizure frequency : whether frequent per day (Table 51), frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58), between six months up to two years (Table 59), and above 5 years (Table 61)

History

Status epilepticus history : Patients with. (Table 64)

Positive famiy history : Patients with. (Table 66)

Positive past history : Patients without. (Table 69)

Organic disease history : Patients with (Table 70)

Pregnancy troubles history : whether with (Table 72) or without it. (Table 73)

Labour troubles history : whether with (Table 74) or without it (Table 75)

Peri-ictal Changes

Patients With presence of Prodroma. (Table 78)

Patients With presence of Factors affecting ictus (Table 82)

Possible Causes

Patients With presence of Cerebral Causes (Table 83)

Patients With presence of Systemic Causes (Table 84)

Patients With presence of Iatrogenic Causes (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Age of onset : Above four years. (Table 48)

Type of onset : whether Abrupt (Table 49) or gradual. (Table 50)

Duration of Illness : Between two years up to five years (Table 60)

Longest seizure free period : *whether below six months (Table 62) or between six months up to two years (Table 63)*

Status epilepticus history : *Patients without (Table 65)*

Positive famiy history : *Patients without (Table 67)*

Positive past history : *Patients with (Table 68)*

Organic disease history : *Patients without (Table 71)*

Neurotic Taits : *Patients with. (Table 76)*

Patients With presence *of Aura (Table 79)*

Patients With presence *of Ictal changes (Table 80)*

Patients With presence *of Post. ictal changes (Table 81)*

GME > TLE

Duration of Illness : *Above five years.*

Neurotic Taits : *Patients without. (Table 77)*

Digit span test

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46). between two and four years (Table 47) or above four years. Table 48)

Type of onset : Gradual. (Table 50)

Frequencey of Seizures :

Past seizure frequency : whether frequent per day (Table 51). frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54). frequent per week (Table 56). frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58). between six months up to two years (Table 59), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : Between six months up to two years. (Table 63)

History

Status epilepticus history : whether with (Table 64) or without it (Table 65)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : whether with (Table 68) or without it (Table 69)

Organic disease history : Patients with (Table 70)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : *whether with (Table 74) or without it (Table 75)*

Neurotic Taits : *whether with (Table 76) or without it (Table 77)*

Peri-ictal Changes

With presence *of Prodroma (Table 78)*

With presence *of Aura (Table 79)*

With presence *of Ictal changes (Table 80)*

With presence *of Post. ictal changes (Table 81)*

With presence *of Factors affecting ictus (Table 82)*

Possible Causes

With presence *of Cerebral Causes (Table 83)*

With presence *of Systemic Causes (Table 84)*

With presence *of Iatrogenic Causes (Table 85)*

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Organic disease history : *Patients without (Table 71)*

GME > TLE

Type of onset : *Abrupt (Table 49)*

Longest seizure free period : *below six months (Table 62)*

Similarities

Non-Significant :

No Significant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46), between two and four years (Table 47) or above four years (Table 48)

Type of onset : whether Abrupt (Table 49) or gradual (Table 50)

Frequency of Seizures :

Past seizure frequency : whether frequent per day (Table 51), frequent per week (Table 52) or frequent per month (Table 63)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58), between six months up to two years (Table 59), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : Between six months up to two years (Table 63)

History

Status epilepticus history : whether with (Table 64) or without it (Table 65)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : without (Table 69)

Organic disease history : whether with (Table 70) or without it (Table 71)

Pregnancy troubles history : With (Table 72)

Labour troubles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : whether with (Table 76) or without it (Table 77)

Peri-ictal Changes

Patients With presence of Prodroma. (Table 78)

Patients With presence of Aura (Table 79)

Patients With presence of Ictal changes (Table 80)

Patients With presence of Post. ictal changes (Table 81)

Patients With presence of Factors affecting ictus (Table 82)

Possible Causes

Patients With presence of Cerebral Causes (Table 83)

Patients With presence of Systemic Causes (Table 84)

Patients With presence of Iatrogenic Causes (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE

Longest seizure free period : below six months (Table 62)

Positive past history : with (Table 68)

Cancellation letters test (Time)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46). between two and four years (Table 47) or above four years (Table 48)

Type of onset : Abrupt (Table 49)

Frequencey of Seizures :

Past seizure frequency : whether frequent per day (Table 51) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56). frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58). between six months up to two years (Table 59), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : whether below six months (Table 62) or between six months up to two years (Table 63)

History

Status epilepticus history : With (Table 64)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : With (Table 68)

Organic disease history : Without (Table 71)

163

Pregnancy troubles   history : *whether with (Table 72) or without it (Table 73)*

Labour troublles   history : *whether with (Table 74) or without it 'Table 75)*

Neurotic Taits   : *whether with (Table 76) or without it (Table 77)*

Peri-ictal Changes

Patients With presence   of Prodroma. *(Table 78)*

Patients With presence   of Aura *(Table 79)*

Patients With presence   of Ictal changes   *(Table 80)*

Patients With presence   of Post. ictal changes *(Table 81)*

Possible Causes

Patients With presence   of Cerebral Causes *(Table 83)*

Patients With presence   of Systemic Causes *(Table 84)*

Patients With presence   of Iatrogenic Causes *(Table 85)*

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Past seizure frequency : *Frequent per week* *(Table 52)*

GME > TLE

Type of onset : *Gradual. (Table 50)*

Status epilepticus history : *Without (Table 65)*

Organic disease history : *With (Table 70)*

Pregnancy troubles history : *Without (Table 73)*

164

Patients With presence of Factors affecting ictus (Table 52)

Cancellation letters test (Errors)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46), between two and four years (Table 47) or above four years (Table 48)

Type of onset : whether Abrupt (Table 49) or gradual (Table 50)

Frequencey of Seizures :

Past seizure frequency : whether ,frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 55), frequent per month or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58), between six months up to two years (Table 59), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : whether below six months (Table 62) or between six months up to two years (Table 63)

History

Status epilepticus history : whether with (Table 64) or without it (Table 65)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : whether with (Table 68) or without it (Table 69)

Organic disease history : whether with (Table 70) or without it (Table 71)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : whether with or without it.

Peri-ictal Changes

Patients With presence of Prodroma. (Table 78)

Patients With presence of Aura (Table 79)

Patients With presence of Ictal changes (Table 80)

Patients With presence of Post. ictal changes (Table 81)

Patients With presence of Factors affecting ictus (Table 82)

Possible Causes

Patients With presence of Cerebral Causes (Table 83)

Patients With presence of Systemic Causes (Table 84)

Patients With presence of Iatrogenic Causes (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Past seizure frequency : Frequent per day (Table 51)

GME > TLE

Positive past history : Without (Table 69)

Matching familiar figure test-Total and Mean time (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether above two years (Table 46), between two and four years (Table 47) or above four years.

Type of onset : whether Abrupt (Table 49) or gradual (Table 50)

Frequencey of Seizures :

Past seizure frequency : whether frequent per day (Table 51), frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : *whether below six months (Table 58), between six months up to two years (Table 59), between two years up to five years (Table 60) or above five years (Table 61)*

Longest seizure free period : *Between six months up to two years (Table 63)*

History

Status epilepticus history : *whether with (Table 64) or without it. (Table 65)*

Positive famiy history : *whether with (Table 66) or without it (Table 67)*

Positive past history : *whether with (Table 68) or without it (Table 69)*

Organic disease history : *whether with (Table 70) or without it (Table 71)*

Pregnancy troubles history : *whether with (Table 72) or without it (Table 73)*

Labour troublles history : *With (Table 74)*

Neurotic Taits : *whether with (Table 76) or without it (Table 77)*

Peri-ictal Changes

Patients With presence of Prodroma. *(Table 78)*

Patients With presence of Aura *(Table 79)*

Patients With presence of Ictal changes *(Table 80)*

Patients With presence of Post. ictal changes *(Table 81)*

Patients With presence of Factors affecting ictus *(Table 82)*

Patients With presence of Cerebral Causes (Table 83)

Patients With presence of Systemic Causes (Table 84)

Patients With presence of Iatrogenic Causes (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Labour troublles history : Without (Table 75)

GME > TLE

Longest seizure free period : below six months (Table 62)

Matching familiar figure test-
Total errors (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46), between two and four years (Table 47) or above four years (Table 48)

Type of onset : Abrupt (Table 49)

Frequency of Seizures :

Past seizure frequency : whether frequent per day (Table 51), frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : whether below six months (Table 62) or between six months up to two years (Table 63)

History

Status epilepticus history : whether with (Table 64) or without it (Table 65)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : whether with (Table 68) or without it (Table 69)

Organic disease history : With (Table 70)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : With (Table 76)

Peri-ictal Changes

Patients With presence of Prodroma. (Table 78)

Patients With presence of Aura (Table 79)

Patients With presence of Ictal changes (Table 80)

Patients With presence of Post. ictal changes (Table 81)

Patients With presence of Factors affecting ictus (Table 82)

Possible Causes

Patients With presence of Cerebral Causes (Table 83)

Patients With presence of Systemic Causes (Table 84)

Patients With presence of Iatrogenic Causes (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Duration of Illness : Between six months up to two years (Table 59)

Organic disease history : Without (Table 70)

Pregnancy troubles history : Without (Table 73)

Neurotic Taits   : Without (Table 77)

GME > TLE

Type of onset : Gradual (Table 50)

ii- Assessement of mood.

The Children anxiety scale (CAS).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 40), between two and four years (Table 47) or above four years (Table 48)

Type of onset : Abrupt (Table 49)

Frequency of Seizures :

Past seizure frequency : whether frequent per day (Table 51) or frequent per week (Table 52)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months(Table 58), between six months up to two years(Table 59), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : whether below six months (Table 62) or between six months up to two years (Table 63)

History

Status epilepticus history : whether with (Table 64) or without it (Table 65)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : whether with (Table 68) or without it (Table 69)

Organic disease history : whether with (Table 70) or without it Table 71)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : whether with (Table 76) or without it (Table 77)

Peri-ictal Changes

Patients With presence of Prodroma. (Table 78)

Patients With presence of Aura (Table 79)

Patients With presence of Ictal changes (Table 80)

Patients With presence of Post. ictal changes (Table 81)

Patients With presence of Factors affecting ictus (Table 82)

Possible Causes

Patients With presence of Systemic Causes (Table 84)

Patients With presence of Iatrogenic Causes (Table 85)

Significant :

There is significant difference between **GME** and **TLE** in the following :

TLE > GME

Past seizure frequency : Frequent per month (Table 53)

Patients With presence of Cerebral Causes (Table 83)

GME > TLE

Type of onset : Gradual. (Table 50)

173

The children depression inventory (CDI).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46). between two and four years (Table 47) or above four years (Table 48)

Type of onset : whether Abrupt (Table 49) or gradual (Table 50)

Frequency of Seizures :

Past seizure frequency : whether frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54). frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58), between six months up to two years (Table 59), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : whether below six months (Table 62) or between six months up to two years (Table 63)

History

Status epilepticus history : Without (Table 65)

Positive famiy history : Whether with (Table 66) or without it (Table 67)

Positive past history : Without (Table 69)

Organic disease history : whether with (Table 70) or without it (Table 71)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : Without (Table 77)

Peri-ictal Changes

Patients With presence *of Prodroma.* (Table 78)

Patients With presence *of Aura* (Table 79)

Patients With presence *of Ictal changes* (Table 80)

Patients With presence *of Post. ictal changes* (Table 81)

Patients With presence *of Factors affecting ictus* (Table 82)

Possible Causes

Patients With presence *of Cerebral Causes* (Table 83)

Patients With presence *of Systemic Causes* (Table 84)

Patients With presence *of Iatrogenic Causes* (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Past seizure frequency : Frequent per day (Table 51)

Current seizure frequency : Frequent per week (Table 56)

Status epilepticus history : With (Table 64)

Positive past history : With (Table 68)

GME > TLE

Neurotic Taits : With (Table 76)

iii- Assessment of personality.
Psychoticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46), between two and four years (Table 47) or above four years (Table 48)

Type of onset : whether Abrupt (Table 49) or gradual (Table 50)

Frequency of Seizures :

Past seizure frequency : whether frequent per day (Table 51), frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56), frequent per month(Table 55) or none (Table 57)

Duration

Duration of Illness : whether between six months up to two years (Table 59). between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : whether below six months (Table 62) or between six months up to two years (Table 63)

History

Status epilepticus history : With (Table 64)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : whether with (Table 68) or without it (Table 69)

Organic disease history : whether with (Table 70) or without it (Table 71)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : whether with (Table 76) or without it (Table 77)

Peri-ictal Changes

Patients With presence of Aura (Table 79)

Patients With presence of Factors affecting ictus (Table 82)

Possible Causes

Patients With presence of Cerebral Causes (Table 83)

Patients With presence of Systemic Causes (Table 84)

Patients With presence of Iatrogenic Causes (Table 84)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Duration of Illness : Below six months (Table 58)

Status epilepticus history : Without (Table 65)

Patients With presence of Prodroma (Table 78)

Patients With presence of Ictal changes (Table 80)

Patients With presence of Post. ictal changes (Table 81)

Ne uroticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46), between two and four years (Table 47) or above four years (Table 48)

Type of onset : Abrupt (Table 49)

Frequency of Seizures :

Past seizure frequency : whether frequent per day (Table 51), frequent per week (Table 52) or frequent per month (Table 53)

Current seizer frequency : whether frequent per day (Table 54), frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)

Duration

Duration of Illness : whether below six months (Table 58), between six months up to two years(Table 59), between two years up to five years (Table 60) or above five years (Table 61)

Longest seizure free period : whether below six months (Table 62) or between six months up to two years (Table 63)

History

Status epilepticus history : whether with (Table 64) or without it (Table 65)

Positive famiy history : With (Table 66)

Positive past history : whether with (Table 68) or without it (Table 69)

Organic disease history : whether with (Table 70) or without it (Table 71)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : whether with (Table 76) or without it (Table 77)

Peri-ictal Changes

Patients With presence of Prodroma (Table 78)

Patients With presence of Aura (Table 79)

Patients With presence of Ictal changes (Table 80)

Patients With presence of Post. ictal changes (Table 81)

179

Possible Causes

Patients With presence *of Cerebral Causes* (Table 83)

Patients With presence *of Systemic Causes* (Table 84)

Patients With presence *of Iatrogenic Causes* (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE

Type of onset : *Gradual* (Table 50)

Positive famiy history : *Without* (Table 67)

Patients With presence *of Factors affecting ictus* (Table 82)

Introversion

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : whether below two years (Table 46) or. between two and four years (Table 47)

Type of onset : whether Abrupt (Table 49) or gradual (Table 50)

Frequency of Seizures :

Past seizure frequency : whether frequent per day (Table 51). or frequent per month (Table 53)

Current seizer frequency : whether frequent per week (Table 56). frequent per month (Table 55) or non (Table 57)

Duration

Duration of Illness : whether below six months (Table 58). between six months up to two years (Table 50) or between two years up to five years (Table 60)

History

Status epilepticus history : With (Table 64)

Positive famiy history : whether with (Table 66) or without it (Table 67)

Positive past history : With (Table 68)

Organic disease history : whether with (Table 70) or without it (Table 71)

Pregnancy troubles history : whether with (Table 72) or without it (Table 73)

Labour troublles history : whether with (Table 74) or without it (Table 75)

Neurotic Taits : whether with (Table 76) or without it (Table 77)

Possible Causes

Patients With presence of Cerebral Causes (Table 83)

Patients With presence of Iatrogenic Causes (Table 85)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Age of onset : *Above four years (Table 48)*

Past seizure frequency : *Frequent per week (Table 52)*

Current seizer frequency : *Frequent per day (Table 54)*

Duration of Illness : *above five years (Table 61)*

Longest seizure free period : *whether below six months (Table 58) or*

between six months up to two years (Table 59)

Status epilepticus history : *without (Table 65)*

Positive past history : *Without (Table 69)*

Periictal Changes

Patients With presence of Prodroma *(Table 78)*

Patients With presence of Aura *(Table 79)*

Patients With presence of Ictal changes *(Table 80)*

Patients With presence of Post. ictal changes *(Table 81)*

Patients With presence of Factors affecting ictus *(Table 82)*

Patients With presence of Systemic Causes *(Table 83)*

Lie scale

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the Illness profile in the following :

Onset of Seizures :

Age of onset : *whether below two years (Table 46) or above four years (Table 56)*

Type of onset : *Gradual (Table 50)* .

Frequency of Seizures :

Past seizure frequency : *whether frequent per day (Table 51), frequent per week (Table 52) or frequent per month (Table 53)*

Current seizer frequency : *whether frequent per day (Table 54), frequent per week (Table 56), frequent per month (Table 55) or none (Table 57)*

Duration

Duration of Illness : *whether below six months (Table 58), between six months up to two years (Table 59), between two years up to five years (Table 60) or above five years (Table 61)*

Longest seizure free period : *whether below six months or between six months up to two years.*

History

Status epilepticus history : *whether with (Table 64) or without it (Table 65)*

Positive famiy history : *whether with (Table 66) or without it (Table 67)*

Positive past history : *whether with (Table 68) or without it (Table 69)*

Organic disease history : *whether with (Table 70) or without it (Table 71)*

Pregnancy troubles history : *without (Table 73)*

Labour troublles history : *whether with (Table 74)or without it (Table 75)*

Neurotic Taits : *whether with (Table 76) or without it(Table 77)*

Peri-ictal Changes

Patients With presence *of Prodroma (Table 78)*

Patients With presence *of Aura (Table 79)*

Patients With presence *of Ictal changes (Table 80)*

Patients With presence *of Post. ictal changes (Table 81)*

Patients With presence *of Factors affecting ictus (Table 82)*

Possible Causes

Patients With presence *of Cerebral Causes (Table 83)*

Patients With presence *of Systemic Causes (Table 84)*

Patients With presence *of Iatrogenic Causes (Table 85)*

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Type of onset : Abrupt (Table 49)

GME > TLE

Age of onset : Between two and four years (Table 47)

Pregnancy troubles history : With (Table 72)

3) Therapeutic issues.

(The relation between Therapeutic variables and performance on different psychometric tests).

a)Psychometry.

i- Cogniitive funchtions

- Coding test

- Digit span test

- Similarities

- Cancellation letters test (Time)

- Cancellation letters test (Errors)

- Maching familiar figure test-Total and Mean time (MFFT)

- Maching familiar figure test-Total errors (MFFT)

ii- Assessement of mood.

- The Children anxiety scale (CAS).

- The children depression inventory (CDI).

iii- Assessement of personality.

- Psychoticism

- Neuroticlsm

- Extraversion

- Introversion

- Lie scale

PATIENTS ON MONOTHERAPY MEDICATION

(Table 86) PATIENTS ON MONOTHERAPY MEDICATIOIN

| | NO | CODING ** | DIGIT SPAN ** | SIMILARITIES * | CANCELLATION LETTERS | | ANXIETY ** | Psychoticism ** | Neuroticism ** | Exteroversion * | Interoversion ** | Lie scale ** | Depression ** | MFFT | |
					TIME	ERROR								TIME	ERROR
GME	97	30	12	4	16	18	48	26	16	2	24	56	36	2	8
TLE	55	25	14	0	5	12	36	18	14	5	33	25	14	0	4
P<0.05		NS	0.0481	NS	NS	NS	NS	NS	NS	NS	0.00002619	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): DIGIT SPAN & Interoversion TEST. TLE > GME
(NS) Non-Significant (P>0.05) :ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

PATIENTS ON POLYTHERAPY MEDICATION

(Table 87) PATIENTS ON POLYTHERAPY MEDICATION

| | NO | CODING ** | DIGIT SPAN ** | SIMILARITIES * | CANCELLATION LETTERS | | ANXIETY ** | Psychoticism * | Neuroticism ** | Exteroversion * | Interoversion ** | Lie scale ** | Depression ** | MFFT | |
					TIME	ERROR								TIME	ERROR
GME	48	20	8	2	2	12	30	6	8	2	26	30	14	2	2
TLE	50	27	4	0	4	7	23	4	4	0	30	28	20	0	2
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 88) PATIENTS ON MONOTHERAPY WITH DEPAKINE

PATIENTS ON MONOTHERAPY WITH DEPAKINE

	NO	CODING	ING DIGIT S	SIMILARITIES	CANCELLATION LETTER		ANXIET	sychoticis	Neuroticism	xteroversio	GIT SPAN Int	PAN Intero	Depression	MFFT	
					TIME	ERROR								TIME	ERROR
GME	3	2	0	2	2	0	4	2	2	0	4	6	2	0	2
TLE	7	0	0	0	0	0	5	2	2	0	5	2	5	0	0
P<0.05		NS	—	NS	NS	—	NS	NS	NS	—	NS	NS	NS	—	NS

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS

I- LOW DOSE:

(Table 89) 1) WIHT LOW DOSE

	NO	CODING	DIGIT SPAN	SIMILARITES	CANCELLATION LETTER		ANXIET	sychoticis	Neuroticism	xteroversio	Interoversion	Lie scale	Depression	MFFT	
					TIME	ERROR								TIME	ERROR
GME	6	2	0	2	2	0	2	2	2	0	2	6	2	0	0
TLE	5	0	0	0	0	0	3	2	0	0	3	0	3	0	0
P<0.05		NS	—	NS	NS	—	NS	NS	NS	—	NS	6.78E-03	NS	—	—

This Table Shows,

Significant (P<0.05): Lie scale TEST.   GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS

II- HIGH DOSE:

(Table 90) 2) WITH HIGH DOSE

	NO	CODING	DIGIT SPAN	SIMILARITES	CANCELLATION LETTER		ANXIET	sychoticis	Neuroticism	xteroversio	Interoversion	Lie scale	Depression	MFFT	
					TIME	ERROR								TIME	ERROR
GME	2	0	0	0	0	0	2	0	0	0	2	0	0	0	2
TLE	2	0	0	0	0	0	2	0	0	0	2	0	0	0	2
P<0.05		—	—	—	—	—	NS	—	—	—	NS	—	—	—	NS

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

AED'S Trade Name	AED'S Scientific Name
- Depakine	- Sodium Valproate
- Tegretol	- Carbamazepine
- Epanutin	- Hydantoin

(Table 31) PATIENTS ON MONOTHERAPY WITH TEGRETOL:

PATIENTS ON MONOTHERAPY WITH *TEGRETOL*

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio *	GIT SPAN int **	PAN intero **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	37	28	12	2	16	18	44	24	14	2	36	50	34	2	0
TLE	48	25	14	0	5	12	31	16	12	5	28	23	9	0	0
P<0.05		0.0000	0.0356	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0119	NS	NS

Table (51) Shows.

Significant (P<0.05): CODING & Depression TEST. ** GME > TLE ** DIGIT SPAN TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

I- LOW DOSE: (Table 92)   1) WITH LOW DOSE

	NO	CODING	DIGIT SPAN	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	67	24	12	2	12	14	40	18	10	2	26	36	30	2	0
TLE	34	21	12	0	3	9	20	12	7	5	21	18	5	0	2
P<0.05		0.0184	NS	NS	NS	NS	NS	NS	NS	NS	0.0395	NS		NS	NS

This Table Shows,

Significant (P<0.05): CODING & INTEROVERSION TEST.   * TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- HIGH DOSE: (Table 93)   2) WITH HIGH DOSE

	NO	CODING *	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sycholicis **	Neuroticism **	xteroversio	Interoversion **	Lie scale **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	20	4	0	0	4	4	4	6	4	0	10	14	4	0	0
TLE	12	4	2	0	2	3	11	4	5	0	7	5	4	0	0
P<0.05		NS	NS	—	NS	NS	9.30E-05	NS	NS	—	NS	NS	NS	—	—

This Table Shows,

Significant (P<0.05): ANXIETY TEST.   * TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

AED'S Trade Name	AED'S Scientific Name
- Depakine	- Sodium Valproate
- Tegretol	- Carbamazepine
- Epanutin	- Hydantoin
- Rivotril	- Clonazepam

(Table 94) PATIENTS ON POLYTHERAPY WITH DEPAKINE&TEGRETOL :

PATIENTS ON POLYTHERAPY WITH DEPAKINE& TEGRETOL

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET	sychoticis	Neuroticism *	xteroversio *	GIT SPAN Int **	PAN Intero **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	26	10	6	2	2	6	18	0	4	2	14	18	3	0	0
	35	21	2	0	2	7	16	2	2	0	23	21	18	0	0
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0	1.73

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

I- LOW DOSE:

(Table 95)   1) WIHT LOW DOSE

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticis *	Neuroticism *	xteroversio	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	14	4	2	0	0	2	12	0	2	2	6	8	6	0	0
TLE	17	9	2	0	0	2	7	2	2	0	9	5	9	0	0
P<0.05		NS	NS	—	—	NS	1.13E-02	NS	NS	NS	NS	NS	NS	—	—

This Table Shows,

Significant (P<0.05):ANXIETY TEST. GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- HIGH DOSE:

(Table 96)   2) WITH HIGH DOSE

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis	Neuroticism *	xteroversio	Interoversion **	Lie scale **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	12	6	4	2	2	4	6	0	2	0	8	10	2	0	0
TLE	18	9	0	0	2	5	7	0	0	0	12	14	7	0	0
P<0.05		NS	3.73E-02	NS	NS	NS	NS	—	NS	—	NS	NS	NS	—	—

This Table Shows,

Significant (P<0.05): DIGIT SPAN TEST. GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS

AED'S Trade Name	AED'S Scientific Name
- Depakine	- Sodium Valproate
- Tegretol	- Carbamazepine
- Epanutin	- Hydantoin

(Table 97) PATIENTS ON POLYTHERAPY WITH EPAUNTIN & TEGRETOL

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis *	Neuroticism *	xteroversio *	GIT SPAN Int **	PAN intero **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	26	10	6	2	2	6	18	0	4	2	14	18	8	0	0
TLE	35	21	2	0	2	7	16	2	2	0	23	21	18	0	2
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	—	NS

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 98) PATIENTS ON POLYTHERAPY WITH EPAUNTIN & DEPAKINE.

PATIENTS ON POLYTHERAPY WITH EPANUTIN & DEPAKINE.

I- HIGH DOSE

	NO	CODING *	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET *	sychoticis *	Neuroticism *	xteroversio	GIT SPAN Int *	PAN intero *	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	6	2	0	0	0	0	6	4	0	0	4	2	0	0	0
TLE	2	2	2	0	0	0	2	2	2	0	0	0	2	0	0
P<0.05		NS	NS	—	—	—	NS	NS	NS	—	NS	NS	NS	—	—

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- LOW DOSE ; NONE

AED'S Trade Name	AED'S Scientific Name
- Depakine	- Sodium Valproate
- Tegretol	- Carbamazepine
- Epanutin	- Hydantoin
- Rivotril	- Clonazepam

(Table 99) PATIENTS ON POLYTHERAPY WITH DEPAKINE, RIVOTRIL & TEGRETOL

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sycholicis *	Neuroticism *	xteroversio *	GIT SPAN Int **	PAN Intero **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	26	10	6	2	2	6	18	0	4	2	14	18	3	0	0
TLE	35	21	2	0	2	7	16	2	2	0	23	21	18	0	2
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	—	NS

This Table Shows,

Significant (P≤0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 100) PATIENTS ON POLYTHERAPY WITH EPAUNTIN & DEPAKINE & Rivotril

I- HIGH DOSE

	NO	CODING *	DIGIT SPAN	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sycholicis	Neuroticism *	xteroversio	GIT SPAN Int **	PAN Intero **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	4	2	0	0	0	2	4	0	2	0	4	4	4	2	0
TLE	7	5	0	0	2	0	5	0	0	0	7	7	2	0	2
P<0.05		NS	—	—	NS	NS	NS	—	NS	—	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- LOW DOSE ; NONE

AED'S *Trade Name*	*AED'S* *Scientific Name*
- Depakine	- Sodium Valproate
- Tegretol	- Carbamazepine

(Table 101) POLYTHERAPY (HIGH DOSE)

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET	sychoticis *	Neuroticism *	xteroversio	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	30	18	6	2	2	10	16	6	6	0	18	20	8	2	2
TLE	27	14	0	0	4	5	12	0	0	0	21	21	9	0	2
P<0.05		N S	0.0429	N S	N S	N S	N S	0.0429	0.0429	—	N S	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05): Psychoticism & Neuroticism TEST & DIGIT SPAN GME > TLE
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 102) POLYTHERAPY (AVERAGE DOSE)

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticis *	Neuroticism *	xteroversio	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	18	4	2	0	0	2	14	0	2	0	8	10	6	0	0
TLE	21	13	4	0	0	2	11	4	4	0	9	7	11	0	0
P<0.05		0.0127	N S	—	—	N S	N S	N S	N S	—	N S	N S	N S	—	—

This Table Shows,

Significant (P<0.05): CODING TEST. TLE > GME
(NS) Non-Significant (P>0.05): OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED:
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 103) COMPLIANT PATIENTS:

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychotlcls **	Neuroticism **	xteroverslo *	Interoversion **	Lle scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	100	37	18	2	18	18	57	28	18	2	46	57	39	2	2
TLE	70	32	11	0	5	14	46	20	14	5	39	37	25	0	7
P<0.05		NS	NS	NS	0.0417	NS	NS	NS	NS	NS	0.0148	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS TEST. GME > TLE INTEROVERSION TEST. TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS
** CHI-SQUARE TESTS

I- COMPLIANT PATIENTS ON MONOTHERAPY

(Table 104)   1) COMPLIANT PATIENTS ON MONOTHERAPY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychotlcls **	Neuroticism **	xteroverslo *	Interoversion **	Lle scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	77	28	11	0	14	11	39	20	14	0	37	46	28	2	2
TLE	45	20	11	0	5	9	30	16	11	5	28	18	14	0	2
P<0.05		NS	NS	—	NS	NS	NS	NS	NS	0.0126	NS	0.0352	NS	NS	NS

This Table Shows,

Significant (P<0.05): Exteroversion TLE > GME & Lle scale TEST. GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- COMPLIANT PATIENTS ON POLYTHERAPY

(Table 105)   2) COMPLIANT PATIENTS ON POLYTHERAPY

	NO	CODING *	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychotlcls **	Neuroticism *	xteroverslo *	Interoversion **	Lle scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	23	9	7	2	5	7	18	7	5	2	9	11	11	0	0
TLE	25	11	0	0	0	5	16	5	2	0	11	18	11	0	5
P<0.05		NS	0.01	NS	0.0466	NS	NS	NS	NS	NS	NS	NS	NS	—	NS

This Table Shows,

Significant (P<0.05): DIGIT SPAN & CANCELLATION LETTERS(TIME) TEST . GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 106) NON-COMPLIANT PATIENTS:

	NO	CODING **	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	46	16	2	5	2	14	23	11	14	2	20	28	16	2	2
TLE	36	20	7	0	5	5	18	7	7	0	25	16	14	0	2
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	0.019	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): INTEROVERSION TEST.   TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

I- NON-COMPLIANT PATIENTS ON MONOTHERAPY

(Table 107)   1) NON-COMPLIANT PATIENTS ON MONOTHERAPY

	NO	CODING	DIGIT SPAN *	SIMILARITIES *	CANCELLATION LETTER		ANXIET	sychoticis	Neuroticism	xteroversio	Interoversion *	Lie scale	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
GME	23	7	0	5	2	7	9	9	5	2	7	11	7	0	0
TLE	9	5	2	0	0	2	7	2	2	0	7	5	0	0	0
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0423	NS	NS	---	---

This Table Shows,

Significant (P<0.05): INTEROVERSION TEST.   TLE > GME

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- NON-COMPLIANT PATIENTS ON POLYTHERAPY

(Table 108)   2) NON-COMPLIANT PATIENTS ON POLYTHERAPY

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticis *	Neuroticism **	xteroversio	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	23	9	2	0	0	7	14	2	9	0	14	16	9	2	2
TLE	27	16	5	0	5	2	11	5	5	0	18	11	14	2	2
P<0.05		NS	NS	—	NS	NS	NS	NS	NS	—	NS	0.045	NS	NS	NS

This Table Shows,

Significant (P<0.05): Lie scale TEST.   GME > TLE

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 109) PATIENTS WITH CONTROLLED SEIZURE :

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticism **	Neuroticism **	xteroverslo *	interoverslon **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	71	29	17	0	17	13	36	23	13	0	34	40	31	2	8
TLE	35	16	9	0	0	5	25	11	9	2	14	28	14	0	5
P<0.05		N S	N S	—	0.001582	N S	0.0423	N S	N S	N S	N S	0.0169	N S	N S	N S

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS, GME > TLE ANXIETY & Lie scale TEST.   TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

I- PATIENTS ON MONOTHERAPY   (Table 110)   1) PATIENTS ON MONOTHERAPY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticium **	xteroverslo *	interoverslon **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	59	25	13	0	17	6	27	17	6	0	25	34	25	2	8
TLE	23	9	7	0	0	2	14	7	7	2	9	18	9	0	2
P<0.05		N S	N S	—	0.009847	N S	N S	N S	N S	N S	N S	N S	N S	N S	N S

This Table Shows,

Significant (P<0.05): CANCELLATION LETTERS (TIME) TEST.   GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- PATIENTS ON POLYTHERAPY   (Table 111)   2) PATIENTS ON POLYTHERAPY

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticium *	xteroverslo	interoverslon **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	12	4	4	0	0	6	8	6	6	0	8	6	6	0	0
TLE	12	7	2	0	0	2	11	5	2	0	5	9	5	0	2
P<0.05		N S	N S	—	—	N S	N S	N S	N S	—	N S	N S	N S	—	N S

This Table Shows,

Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 112) PATIENTS WITHOUT CONTROLLED SEIZURE

	NO	CODING **	ING DIGIT S **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	GIT SPAN Int **	PAN Intero **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	75	19	13	6	8	17	40	17	17	4	29	46	15	2	2
TLE	71	34	9	0	9	14	39	18	11	2	48	30	23	0	5
P<0.05		0.007805	N S	0.0437	N S	N S	N S	N S	N S	N S	0.0004641	0.0211	N S	N S	N S

This Table Shows,
Significant (P<0.05): CODING , Interoversion TLE > GME & Lie scale TEST, SIMILARITIES GME > TLE
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

I- PATIENTS ON MONOTHERAPY   (Table 113)   1) PATIENTS ON MONOTHERAPY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	44	6	6	6	6	15	21	13	10	2	17	23	6	0	2
TLE	30	11	5	0	5	9	20	14	7	2	20	9	5	0	0
P<0.05		0.0208	N S	N S	N S	N S	N S	N S	N S	N S	0.0179	N S	N S		N S

This Table Shows,
Significant (P<0.05): CODING & INTEROVERSION TEST. TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

II- PATIENTS ON POLYTHERAPY   (Table 114)   2) PATIENTS ON POLYTHERAPY

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism *	xteroversio *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	31	13	8	2	4	2	19	4	6	2	13	23	6	0	2
TLE	41	23	5	0	5	5	18	5	5	0	28	20	18	0	5
P<0.05		N S	N S	N S	N S	N S	N S	N S	N S	N S	0.0253	0.0295	N S		N S

This Table Shows,
Significant (P<0.05): Lie scale GME > TLE & INTEROVEF   TLE > GME
(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 115) WITH PRESENCE OF DRUG SIDE EFFECTS

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTER		ANXIET **	sychoticis **	Neuroticism **	xteroversio *	nteroversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GME	43	20	4	2	4	14	20	12	10	2	26	24	14	2	2
TLE	29	20	5	0	5	5	18	9	7	2	14	16	11	0	2
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

3) *Therapeutic issues (detailed)*

(The relation between Therapeutic variables and performance on different psychometric tests).

a)Psychometry.

i- Cognitive funchtions

Coding test

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monotherapy medication *(Table 86)*

Patients on polytherapy medication *(Table 87)*

Patients on monotherapy with Sodium Valproate (Depakine) *: Whether with low (Table 89), high dose (Table 90) or both (Table 88)*

Patients on monotherapy with Carbamazepine (Tegretol) *; with high dose (Table 93)*

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) *; Whether with low (Table 95), high dose (Table 96) or both (Table 94)*

Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) *(Table 97)*

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) *(Table 98)*

Patients on polytherapy with Sodium Valproate (Depakine),

Clonazepam (Rivotril) and Carbamazepine (Tegretol) (Table 99)

Patients on polytherapy with Phenytoin (Epanutin). Clonazepam **(Rivotril) and Sodium Valproate (Depakine)** (Table 100)

Patients on polytherapy with high dose (Table 101)

Compliance

Compliant Patients (Table 103)

Compliant Patients on Monotherapy (Table 104)

Compliant Patients on Polytherapy (Table 105)

Non-Compliance

Non-Compliant Patients (Table 106)

Non-Compliant Patients on Monotherapy (Table 107)

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures (Table 109)

Patients with controlled seizures on monotherapy (Table 110)

Patients with controlled seizures on polytherapy (Table 111)

Whithout controlled seizure

Patients without controlled seizures (Table 112)

Patients without controlled seizures on polytherapy (Table 114)

Patients with side effects (Table 115)

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Patients on monotherapy with Carbamazepine (Tegretol) : *(Low dose) (Table 92)*

Patients on polytherapy with average dose *(Table 102)*

Patients without controlled seizures on monotherapy *(Table 113)*

GME > TLE

Patients on monotherapy with Carbamazepine (Tegretol) *: Both (low and high dose) (Table 91)*

Digit span test

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on polytherapy medication *(Table 86)*

Patients on monotherapy with Sodium Valproate (Depakine) ; *Whether with low (Table 89), high dose (Table 90) or both (Table 88)*

Patients on monotherapy with Carbamazepine (Tegretol) *(Low.dose*

(Table 92)

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) : Whether with low (Table 95), high dose (Table 96) or both (Table 94)

Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) (Table 97)

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) (Table 98)

Patients on polytherapy with Sodium Valproate (Depakine), Clonazepam (Rivotril) and Carbamazepine (Tegretol) (Table 99)

Patients on polytherapy with Phenytoin (Epanutin), Clonazepam (Rivotril) and Sodium Valproate (Depakine) (Table 100)

Patients on polytherapy with average dose (Table 101)

Compliance

Compliant Patients (Table 103)

Compliant Patients on Monotherapy (Table 104)

Non-Compliance

Non-Compliant Patients (Table 106)

Non-Compliant Patients on Monotherapy (Table 107)

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures (Table 109)

Patients with controlled seizures on monotherapy *(Table 110)*

Patients with controlled seizures on polytherapy *(Table 111)*

Whithout controlled seizure

Patients without controlled seizures *(Table 112)*

Patients without controlled seizures on monotherapy *(Table 113)*

Patients without controlled seizures on polytherapy *(Table 114)*

Side Effects

Patients with side effects *(Table 115)*

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Patients on monother apy medication *(Table 86)*

Patients on monotherapy with Carbamazepine (Tegretol) (Both) *(Table 91)*

GME > TLE

Patients on monotherapy with Carbamazepine (Tegretol) ; high dose *(Table 93)*

Patients on polytherapy with high dose *(Table 101)*

Compliant Patients on Polytherapy *(Table 105)*

Similarities

Non-Significant :

No Significant difference found in different psychological & cognitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication (Table 86)

Patients on polytherapy medication (Table 87)

Patients on monotherapy with Sodium Valproate (Depakine) : Whether with low (Table 89), high dose (Table 90) or both (Table 88).

Patients on monotherapy with Carbamazepine (Tegretol) : Whether with low (Table 92), high dose (Table 93) or both (Table 91)

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) ; Whether with low (Table 95), high dose (Table 96) or both (Table 94)

Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) (Table 97)

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) (Table 98)

Patients on polytherapy with Sodium Valproate (Depakine), Clonazepam (Rivotril) and Carbamazepine (Tegretol) (Table 99)

Patients on polytherapy with Phenytoin (Epanutin), Clonazepam (Rivotril) and Sodium Valproate (Depakine) (Table 100)

Patients on polytherapy with high dose (Table 101)

Patients on polytherapy with average dose (Table 102)

Compliance

Compliant Patients *(Table 103)*

Compliant Patients on Monotherapy *(Table 104)*

Compliant Patients on Polytherapy *(Table 105)*

Non-Compliance

Non-Compliant Patients *(Table 106)*

Non-Compliant Patients on Monotherapy *(Table 107)*

Non-Compliant Patients on Polytherapy *(Table 108)*

Controlled Seizure

Patients with controlled seizures *(Table 109)*

Patients with controlled seizures on monotherapy *(Table 110)*

Patients with controlled seizures on polytherapy *(Table 111)*

Whithout controlled seizure

Patients without controlled seizures on monotherapy *(Table 113)*

Patients without controlled seizures on polytherapy *(Table 114)*

Side Effects

Patients with side effects *(Table 115)*

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE

Patients without controlled seizures. (Total) Table 112

Cancellation letters test (Time)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication (Table 86)

Patients on polytherapy medication (Table 87)

Patients on monotherapy with Sodium Valproate (Depakine) : Whether with low (Table 89), high dose (Table 90) or both (Table 88)

Patients on monotherapy with Carbamazepine (Tegretol) : Whether with low (Table 92), high dose (Table 93)or both (Table 91)

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) ; Whether with low (Table 95), high dose 96 or both 94

Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) (Table 97)

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) (Table 98)

Patients on polytherapy with Sodium Valproate (Depakine).

Compliance

Non-Compliance

Controlled Seizure

Whithout controlled seizure

Side Effects

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE

Compliant Patients. (Total) *(Table 103)*

Compliant Patients on Monotherapy *(Table 104)*

Patients with controlled seizures (Total) *(Table 109)*

Patients with controlled seizures on monotherapy *(Table 110)*

Cancellation letters test (Errors)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication *(Table 86)*

Patients on polytherapy medication *(Table 87)*

Patients on monotherapy with Sodium Valproate (Depakine) ;
Whether with low *(Table 89)*, high dose *(Table 90)* or both *(Table 88)*

Patients on monotherapy with Carbamazepine (Tegretol) ; Whether with low *(Table 92)*, high dose *(Table 93)* or both *(Table 91)*

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) ; Whether with low *(Table 95)*, high dose *(Table 96)* or both *(Table 94)*

Compliance

Non-Compliance

Controlled Seizure

Whithout controlled seizure

Patients without controlled seizures (Table 112)

Patients without controlled seizures on monotherapy (Table 113)

Patients without controlled seizures on polytherapy (Table 114)

Side Effects

Patients with side effects (Table 115)

Matching familiar figure test-Total and Mean time (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monotherapy medication (Table 89)

Patients on polytherapy medication (Table 87)

Patients on monotherapy with Sodium Valproate (Depakine) ;
Whether with low (Table 89), high dose (Table 90) or both (Table 88)

Patients on monotherapy with Carbamazepine (Tegretol) ; Whether with low (Table 92, high dose (Table 93) or both (Table 91).

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) ; Whether with low (Table

95), high dose (Table 96) or both (Table 94).

Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) (Table 97).

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) (Table 98).

Patients on polytherapy with Sodium Valproate (Depakine), Clonazepam (Rivotril) and Carbamazepine (Tegretol) (Table 99).

Patients on polytherapy with Phenytoin (Epanutin), Clonazepam (Rivotril) and Sodium Valproate (Depakine) (Table 100).

Patients on polytherapy with high dose (Table 101).

Patients on polytherapy with average dose (Table 102).

Compliance

Compliant Patients (Table 103).

Compliant Patients on Monotherapy (Table 104).

Compliant Patients on Polytherapy (Table 105)

Non-Compliance

Non-Compliant Patients. (Table 106)

Non-Compliant Patients on Monotherapy (Table 107)

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures. (Table 109)

Patients with controlled seizures on monotherapy. (Table 110)

Patients with controlled seizures on polytherapy. (Table 111)

Whithout controlled seizure

Patients without controlled seizures. (Table 112)

Patients without controlled seizures on monotherapy. (Table 113)

Patients without controlled seizures on polytherapy. (Table 114)

Side Effects

Patients with side effects. (Table 115)

Matching familiar figure test-
Total errors (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication (Table 86)

Patients on polytherapy medication (Table 87)

Patients on monotherapy with Sodium Valproate (Depakine) ; Whether with low (Table 89), high dose (Table 90) or both (Table 88).

Patients on monotherapy with Carbamazepine (Tegretol) ; Whether with low (Table 92), high dose (Table 93) or both (Table 91.)

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) : Whether with low (Table 95), high dose (Table 96) or both (Table 94).

(Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) (Table 97).

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) (Table 98).

Patients on polytherapy with Sodium Valproate (Depakine), Clonazepam (Rivotril) and Carbamazepine (Tegretol) (Table 99).

Patients on polytherapy with Phenytoin (Epanutin), Clonazepam (Rivotril) and Sodium Valproate (Depakine) (Table 100).

Patients on polytherapy with high dose (Table 101).

Patients on polytherapy with average dose (Table 102).

Compliance

Compliant Patients *(Table 103).*

Compliant Patients on Monotherapy *(Table 104.)*

Compliant Patients on Polytherapy *(Table 105)*

Non-Compliance

Non-Compliant Patients *(Table 106).*

Non-Compliant Patients on Monotherapy *(Table 107)*

Non-Compliant Patients on Polytherapy *(Table 108)*

Controlled Seizure

Patients with controlled seizures (Table 109).

Patients with controlled seizures on monotherapy (Table 110).

Patients with controlled seizures on polytherapy Table 111).

Whithout controlled seizure

Patients without controlled seizures (Table 112).

Patients without controlled seizures on monotherapy (Table 113).

Patients without controlled seizures on polytherapy (Table 114).

Side Effects

Patients with side effects (Table 115).

ii- Assessement of mood.

The Children anxiety scale (CAS).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication (Table 86)

Patients on polytherapy medication (Table 87)

Patients on monotherapy with Sodium Valproate (Depakine) : Whether with low (Table 89) or both (Table 88)..

Patients on monotherapy with Carbamazepine (Tegretol) : Whether with low (Table 92). high dose (Table 93) or both (Table 91).

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) : Whether with high dose (Table 96) or both (Table 94).

Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) (Table 97).

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) (Table 98).

Patients on polytherapy with Sodium Valproate (Depakine), Clonazepam (Rivotril) and Carbamazepine (Tegretol) (Table 99).

Patients on polytherapy with Phenytoin (Epanutin), Clonazepam (Rivotril) and Sodium Valproate (Depakine) (Table 100).

Patients on polytherapy with high dose (Table 101).

Patients on polytherapy with average dose (Table 102).

Compliance

Compliant Patients (Table 103).

Compliant Patients on Monotherapy (Table 104)

Compliant Patients on Polytherapy (Table 105)

215

Non-Compliance

Non-Compliant Patients (Table 106).

Non-Compliant Patients on Monotherapy (Table 107)

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures on monotherapy (Table 110).

Patients with controlled seizures on polytherapy (Table 111).

Whithout controlled seizure

Patients without controlled seizures (Table 112).

Patients without controlled seizures on monotherapy (Table 113).

Patients without controlled seizures on polytherapy (Table 114).

Side Effects

Patients with side effects (Table 115).

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Patients on monotherapy with Carbamazepine (Tegretol) ; (High dose) (Table 93)

Patients with controlled seizures (Total) (Table 109)

GME > TLE

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) : (low dose) (Table 95)

The children depression inventory (CDI).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication (Table 86)

Patients on polytherapy medication (Table 87)

Patients on monotherapy with Sodium Valproate (Depakine) : Whether with low (Table 89), high dose (Table 90) or both (Table 88).

Patients on monotherapy with Carbamazepine (Tegretol) : Whether with low (Table 92) or high dose (Table 93).

Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol) ; Whether with low (Table 95), high dose (Table 96) or both (Table 94).

Patients on polytherapy with Phenytoin (Epanutin) and Carbamazepine (Tegretol) (Table 97).

Patients on polytherapy with Phenytoin (Epanutin) and Sodium Valproate (Depakine) (Table 98).

Patients on polytherapy with Sodium Valproate (Depakine), Clonazepam (Rivotril) and Carbamazepine (Tegretol)

(Table 99).

Patients on polytherapy with Phenytoin (Epanutin), Clonazepam (Rivotril) and Sodium Valproate (Depakine) (Table 100).

Patients on polytherapy with high dose (Table 101).

Patients on polytherapy with average dose (Table 102).

Compliance

Compliant Patients (Table 103).

Compliant Patients on Monotherapy (Table 104).

Compliant Patients on Polytherapy (Table 105)

Non-Compliance

Non-Compliant Patients (Table 106).

Non-Compliant Patients on Monotherapy (Table 107).

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures (Table 109).

Patients with controlled seizures on monotherapy (Table 110).

Patients with controlled seizures on polytherapy (Table 111).

Whithout controlled seizure

Patients without controlled seizures (Table 112).

Patients without controlled seizures on monotherapy (Table 113).

Patients without controlled seizures on polytherapy (Table 114).

Side Effects

Patients with side effects (Table 115).

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE

Patients on monotherapy with Carbamazepine (Tegretol) (Total)

(Table 91.)   ,

iii- Assessement of personality.

Psychoticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monotherapy medication *(Table 86).*

Patients on polytherapy medication *(Table 87).*

Patients on monotherapy with Sodium Valproate (Depakine) : *Whether with low (Table 89), high dose (Table 90) or both (Table 88).*

Patients on monotherapy with Tegretol ; *Whether with low (Table 92), high dose (Table 93) or both (Table 91).*

Patients on polytherapy with Sodium Valproate (Depakine) and Tegretol ; *Whether with low (Table 95), high dose (Table 96) or both (Table 94).*

Patients on polytherapy with Epanutin and Tegretol *(Table 97).*

Patients on polytherapy with Epanutin and Sodium Valproate (Depakine) *(Table 98).*

Patients on polytherapy with Sodium Valproate (Depakine) Rivotril and Tegretol *(Table 99).*

Patients on polytherapy with Epanutin, Rivotril and Sodium Valproate (Depakine) *(Table 100).*

Patients on polytherapy with average dose *(Table 102).*

Compliance

Compliant Patients (Table 103).

Compliant Patients on Monotherapy (Table 104).

Compliant Patients on Polytherapy (Table 105).

Non-Compliance

Non-Compliant Patients (Table 106).

Non-Compliant Patients on Monotherapy (Table 107)

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures (Table 109).

Patients with controlled seizures on monotherapy (Table 110).

Patients with controlled seizures on polytherapy (Table 111).

Whithout controlled seizure

Patients without controlled seizures (Table 112).

Patients without controlled seizures on monotherapy (Table 113).

Patients without controlled seizures on polytherapy (Table 114).

Side Effects

Patients with side effects (Table 115).

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE

Patients on polytherapy with high dose (Table 101).

Neuroticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication (Table 86)

Patients on polytherapy medication (Table 87)

Patients on monotherapy with Sodium Valproate (Depakine) :
Whether with low (Table 89), high dose (Table 90) or both (Table 88).

Patients on monotherapy with Tegretol ; Whether with low (Table 92), high dose (Table 93) or both (Table 91).

Patients on polytherapy with Sodium Valproate (Depakine) and Tegretol ; Whether with low (Table 95), high dose (Table 96) or both (Table 94).

Patients on polytherapy with Epanutin and Tegretol (Table 97).

Patients on polytherapy with Epanutin and Sodium Valproate (Depakine) (Table 98).

Patients on polytherapy with Sodium Valproate (Depakine), Rivotril and Tegretol (Table 99).

Patients on polytherapy with Epanutin, Rivotril and Sodium Valproate (Depakine) (Table 100).

Patients on polytherapy with average dose (Table 102).

Compliance

Compliant Patients (Table 103).

Compliant Patients on Monotherapy (Table 104)

Compliant Patients on Polytherapy (Table 105)

Non-Compliance

Non-Compliant Patients (Table 106).

Non-Compliant Patients on Monotherapy (Table 107)

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures (Table 109).

Patients with controlled seizures on monotherapy (Table 110).

Patients with controlled seizures on polytherapy (Table 111).

Whithout controlled seizure

Patients without controlled seizures (Table 112).

Patients without controlled seizures on monotherapy (Table 113).

Patients without controlled seizures on polytherapy (Table 114).

Side Effects

Patients with side effects (Table 115).

Significant :

There is significant difference between GME and TLE in the following :

GME > TLE

Patients on polytherapy with high dose *(Table 101).*

Introversion

$\mathcal{N}$on-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication *(Table 86)*

Patients on polytherapy medication *(Table 87)*

Patients on monotherapy with Sodium Valproate (Depakine) *: Whether with low (Table 89), high dose (Table 90) or both (Table 88).*

Patients on monotherapy with Tegretol *; Whether with low (Table 92), high dose (Table 93) or both (Table 91).*

Patients on polytherapy with Sodium Valproate (Depakine) and Tegretol *; Whether with low (Table 95), high dose (Table 96) or both (Table 94).*

Patients on polytherapy with Epanutin and Tegretol *(Table 97).*

Patients on polytherapy with Epanutin and Sodium Valproate

(Depakine) (Table 98).

Patients on polytherapy with Sodium Valproate (Depakine). Rivotril and Tegretol (Table 99).

Patients on polytherapy with Epanutin. Rivotril and Sodium Valproate (Depakine) (Table 100).

Patients on polytherapy with high dose (Table 101).

Patients on polytherapy with average dose (Table 102).

Compliance

Compliant Patients on Monotherapy (Table 104.)   ,

Compliant Patients on Polytherapy (Table 105)

Non-Compliance

Non-Compliant Patients on Polytherapy (Table 108)

Controlled Seizure

Patients with controlled seizures (Table 109).

Patients with controlled seizures on monotherapy (Table 110).

Patients with controlled seizures on polytherapy (Table 111).

Side Effects

Patients with side effects (Table 115).

Non-Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Compliant Patients (Total) (Table 103).

Non-Compliant Patients (Total) (Table 106).

Non-Compliant Patients on Monotherapy (Table 107).

Whithout controlled seizure :

Patients without controlled seizures (Total) (Table 112).

Patients without controlled seizures on monotherapy (Table 113).

Patients without controlled seizures on polytherapy. (Table 114).

Lie scale

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the therapeutic issues in the following :

Patients on monother apy medication *(Table 86)*

Patients on polytherapy medication *(Table 87)*

Patients on monotherapy with Sodium Valproate (Depakine) ; *High dose (Table 90).*

Patients on monotherapy with Tegretol ; *Whether with low (Table 92), high dose (Table 93) or both (Table 91).*

Patients on polytherapy with Sodium Valproate (Depakine) and Tegretol : *Whether with low (Table 95), high dose (Table 96) or both (Table 94).*

226

Patients on polytherapy with Epanutin and Tegretol (Table 97).

Patients on polytherapy with Epanutin and Sodium Valproate (Depakine) (Table 98).

Patients on polytherapy with Sodium Valproate (Depakine), Rivotril and Tegretol (Table 99).

Patients on polytherapy with Epanutin, Rivotril and Sodium Valproate (Depakine) (Table 100).

Patients on polytherapy with high dose (Table 101).

Patients on polytherapy with average dose (Table 102).

Compliance

Compliant Patients (Total) (Table 103).

Compliant Patients on Polytherapy (Table 105)

Non-Compliance

Non-Compliant Patients (Total) (Table 106)

Non-Compliant Patients on Monotherapy (Table 107)

Controlled Seizure

Patients with controlled seizures on monotherapy (Table 110).

Patients with controlled seizures on polytherapy (Table 111).

Whithout controlled seizure

Patients without controlled seizures on monotherapy (Table 113).

Side Effects

Patients with side effects (Table 115).

Significant :

There is significant difference between GME and TLE in the following :

TLE > GME

Patients with controlled seizures (Total) (Table 109).

GME > TLE

Patients on monotherapy with Sodium Valproate (Depakine) :

Whether with low (Table 89) or both (low and high dose).
(Table 88)

Compliant Patients on Monotherapy (Table 104)

Non-Compliant Patients on Polytherapy (Table 108)

Patients without controlled seizures (Total) (Table 112)

Patients without controlled seizures on polytherapy (Table 114)

4) Electroencephalogram (EEG)

(The relation between Electroencephalogram variables and performance on different psychometric tests).

a)Psychometry.

i- Cogniitive funchtions

- Coding test

- Digit span test

- Similarities

- Cancellation letters test (Time)

- Cancellation letters test (Errors)

- Maching familiar figure test-Total and Mean time (MFFT)

- Maching familiar figure test-Total errors (MFFT)

ii- Assessment of mood.

- The Children anxiety scale (CAS).

- The children depression inventory (CDI).

iii- Assessment of personality.

- Psychoticism

- Neuroticlsm

- Extraversion

- Introversion

- Lie scale

LEFT TEMPORAL & LEFT TEMPORAL & SECONDARY GENERALISED

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism *	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
LEFT TEMP.	35	10	13	0	4	6	20	11	6	1	22	16	7	0	9
Tem. 2nry GENER.	8	4	2	0	1	1	5	2	1	0	6	5	5	0	0
P<0.05		NS	NS	—	NS	NS	NS	NS	NS	NS	NS	NS	0.0476	NS	NS

This Table Shows,

Significant (P<0.05): Depression TEST. 2ry. GENERALISED > LEFT TEMP.

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

(Table 116) LEFT TEMPORAL VERSUS LEFT TEMPORAL WITH SECONDARY GENERALISED

BI-TEMPORAL & BI-TEMPORAL & SECONDARY GENERALISED

	NO	CODING **	DIGIT SPAN *	SIMILARITIES	CANCELLATION LETTERS		ANXIETY **	Psychoticism *	Neuroticism *	Exteroversion *	Interoversion **	Lie scale **	Depression *	MFFT	
					TIME	ERROR								TIME	ERROR
BI- TEMP.	14	8	4	0	0	2	9	5	4	1	6	6	3	1	1
I & 2nry GENER.	8	4	2	0	1	1	5	2	1	0	6	5	5	0	0
P<0.05		NS	NS	—	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

Is Table Shows,

Significant (P<0.05): NONE.

Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 117) BI-TEMPORAL VERSUS BI-TEMPORAL WITH SECONDARY GENERALISED

GENERALISED & NONE

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism *	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GENERALISED	114	48	22	4	22	25	58	27	19	3	49	60	37	1	9
NONE	4	2	2	0	0	1	1	0	1	0	3	2	3	0	1
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS.	NS	NS	NS	NS	0.00969	NS

s Table Shows,

Significant (P<0.05):MFFT (TIME) TESTS. GENERALISED > NONE.

Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 118) GENERALISED VERSUS NONE

GENERALISED & RIGHT TEMPORAL

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GENERALISED	114	48	22	4	22	25	58	27	19	3	49	60	37	1	9
RIGHT TEMP.	57	32	6	0	10	14	31	14	8	2	29	30	14	3	5
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,
Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) :ALL PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 94) GENERALISED VERSUS RIGHT TEMPORAL

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

GENERALISED & LEFT TEMPORAL

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GENERALISED	114	48	22	4	22	25	58	27	19	3	49	60	37	1	9
LEFT TEMP.	35	10	13	0	4	6	20	11	6	1	22	16	7	0	9
P<0.05		NS	0.0294	NS	NS	NS	NS	NS	NS	NS	0.0395	NS	NS	NS	0.0113

This Table Shows,
Significant (P<0.05): DIGIT SPAN, Interoversion & MFFT (ERRROR) TEST. TLE > GME
(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 120) GENERALISED VERSUS LEFT TEMPORAL

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

GENERALISED & BI-TEMPORAL

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS		ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT	
					TIME	ERROR								TIME	ERROR
GENERALISED	114	48	22	4	22	25	58	27	19	3	49	60	37	1	9
BI- TEMP.	14	8	4	0	0	2	9	5	4	1	6	6	3	1	1
P<0.05		NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,
Significant (P<0.05): NONE.
(NS) Non-Significant (P>0.05) :ALL PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 121) GENERALISED VERSUS BI-TEMPORAL

TESTS USED :
* FISHER EXACT TESTS.
** CHI-SQUARE TESTS.

GENERALISED & SECONDARY GENERALISED

	NO	CODING **	DIGIT SPAN **	SIMILARITIES *	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale	Depression **	MFFT TIME	MFFT ERROR
GENERALISED	114	48	22	4	22	25	58	27	19	3	49	60	37	1	9
2nry GENER.	28	12	8	0	5	4	17	8	8	1	12	16	12	0	1
P<0.05		NS	NS	NS	0.0104	NS	NS	NS	NS	NS	0.0242	NS	0.0393	NS	0.0344

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) :ALL PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 122) GENERALISED VERSUS SECONDAY GENERALISED

TESTS USED :
FISHER EXACT TESTS.
CHI-SQUARE TESTS.

RIGHT TEMPORAL & LEFT TEMPORAL

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression **	MFFT TIME	MFFT ERROR
RIGHT TEMP.	57	32	6	0	10	14	31	14	8	2	29	30	14	3	5
LEFT TEMP.	35	10	13	0	4	6	20	11	6	1	22	16	7	0	9
P<0.05		0.009956	0.0022	—	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.0281

This Table Shows,

Significant (P<0.05): CODING, *Rt*.TLE > *lt*.TLE DIGIT SPAN & MFFT (ERROR) TEST. *lt*. TLE > *Rt*.

(NS) Non-Significant (P>0.05) : OTHER PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 123) RIGHT TEMPORAL VERSUS LEFT TEMPORAL

RIGHT TEMPORAL & BI-TEMPORAL

	NO	CODING **	DIGIT SPAN **	SIMILARITIES	CANCELLATION LETTERS TIME	CANCELLATION LETTERS ERROR	ANXIETY **	Psychoticism **	Neuroticism **	Exteroversion *	Interoversion **	Lie scale **	Depression *	MFFT TIME	MFFT ERROR
RIGHT TEMP.	57	32	6	0	10	14	31	14	8	2	29	30	14	3	5
BI- TEMP.	14	8	4	0	0	2	9	5	4	1	6	6	3	1	1
P<0.05		NS	NS	—	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

This Table Shows,

Significant (P<0.05): NONE.

(NS) Non-Significant (P>0.05) : ALL PSYCHOLOGICAL & COGNITIVE SCALES.

(Table 99) RIGHT TEMPORAL VERSUS BI-TEMPORAL

TESTS USED :
FISHER EXACT TESTS.
CHI-SQUARE TESTS.

4) Electroencephalogram (EEG) (detailed)

(The relation between Electroencephalogram variables and performance on different psychometric tests).

**a)Psychometry.**

i- Cognitive funchtions

Coding test

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* **Generalised versus right temporal** *(Table 119)*

* **Generalised versus left temporal***(Table 120)*

* **Generalised versus bi-temporal** *(Table 121)*

* **Generalised versus secondary generalised** *(Table 122)*

* **Right temporal versus bi-temporal** *(Table 124)*

* **Left temporal versus bi-temporal** *(Table 125)*

* **Right temporal versus Right temporal with secondary generalisation** *(Table 126)*

* **Left Temporal versus left temporal with secondary generalisation** *(Table 116).*

* **Bi-Temporal versus bi-temporal with secondary generalisation** *(Table 117).*

* **Generalised versus none** *(Table 118).*

Significant :

There is significant difference between * *Right temporal versus left temporal (Rt> Lt. temporal) (Table 123).*

Digit span test

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the EEG in the following :

* *Generalised versus right temporal (Table 119)*

* *Generalised versus bi-temporal (Table 121)*

* *Generalised versus secondary generalised (Table 122)*

* *Right temporal versus bi-temporal (Table 124)*

* *Left temporal versus bi-temporal (Table 125)*

* *Right temporal versus Right temporal with secondary generalisation (Table 126)*

* *Left Temporal versus left temporal with secondary generalisation (Table 116).*

* *Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).*

* *Generalised versus none (Table 118)*

Significant :

There is significant difference between Generalised versus left temporal (Lt. temporal> Generalized) *(Table 120)* and right temporal versus left temporal (Lt. < Rt. temporal) *(Table 123).*

Similarities

Non-Significant :

No Signifcant difference found in different psychological &

congnitive scales between GME and TLE concerning the EEG in the following :

* *Generalised versus right temporal* *(Table 119)*

* *Generalised versus left temporal (Table 120)*

* *Generalised versus bi-temporal (Table 121)*

* *Right temporal versus left temporal (Table 123)*

* *Right temporal versus bi-temporal (Table 124)*

* *Left temporal versus bi-temporal (Table 125)*

* *Right temporal versus Right temporal with secondary generalisation (Table 126)*

* *Left Temporal versus left temporal with secondary generalisation (Table 116).*

* *Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).*

* *Generalised versus none (Table 118)*

Significant :

There is significant difference between Generalised versus Secondary generalised (Gen. > 2ry. Gen.) *(Table 122)*

Cancellation letters test (Time)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the EEG in the following :

* *Generalised versus right temporal (Table 119)*

* *Generalised versus left temporal* (Table 120)

* *Generalised versus bi-temporal* (Table 121)

* *Generalised versus secondary generalised* (Table 122)

* **Right temporal versus left temporal** (Table 123)

* **Right temporal versus bi-temporal** (Table 124)

* **Left temporal versus bi-temporal** (Table 125)

* **Left Temporal versus left temporal with secondary** generalisation (Table 116).

* **Bi-Temporal versus bi-temporal with secondary generalisation** (Table 117).

* *Generalised versus none* (Table 118)

Significant :

There is significant difference between right temporal versus Right temporal with secondary   generalisation (Rt. temporal > 2ry. Gen.) (Table 126)

Cancellation letters test (Errors)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* **Generalised versus right temporal** (Table 119)

* **Generalised versus left temporal** (Table 120)

* **Generalised versus bi-temporal** (Table 121)

* **Generalised versus secondary generalised** (Table 122)

* **Right temporal versus left temporal** (Table 123)

* *Right temporal versus bi-temporal* (Table 124)

* *Left temporal versus bi-temporal* (Table 125)

* *Left Temporal versus left temporal with secondary generalisation* (Table 116).

* *Bi-Temporal versus bi-temporal with secondary generalisation* (Table 117).

* *Generalised versus none* (Table 118)

Significant :

There is significant difference between right temporal versus Right temporal with secondary generalisation (Rt. temporal > 2ry. Gen.) (Table 126)

Matching familiar figure test-Total and Mean time (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* *Generalised versus right temporal* (Table 119)

* *Generalised versus left temporal* (Table 120)

* *Generalised versus bi-temporal* (Table 121)

* *Generalised versus secondary generalised* *Table 122*

* *Right temporal versus left temporal (Table 123)*

* *Right temporal versus bi-temporal (Table 124)*

* *Left temporal versus bi-temporal (Table 125)*

* *Right temporal versus Right temporal with secondary generalisation (Table 126)*

* *Left Temporal versus left temporal with secondary generalisation (Table 116).*

* *Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).*   ,

Significant :

There is significant difference between Generalised versus non (Gen. > None) *(Table 118)*

Matching familiar figure test-Total Errors (MFFT)

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* *Generalised versus right temporal (Table 119)*

* *Generalised versus bi-temporal (Table 121)*

* *Right temporal versus bi-temporal (Table 124)*

* *Left temporal versus bi-temporal* (Table 125)

* *Right temporal versus Right temporal with secondary generalisation* (Table 126)

* *Left Temporal versus left temporal with secondary generalisation* (Table 116).

* *Bi-Temporal versus bi-temporal with secondary generalisation* (Table 117).

* *Generalised versus none* (Table 118)

Non-Significant :

There is significant difference between Generalised versus left temporal (Lt. temporal > Gen.) *(Table 120)*,. Right temporal versus left temporal (Lt. > Rt. temporal) *(Table 123)* and Generalised versus secondary generalised (Gen. > 2ry. Gen.) *(Table 122)*.

ii- *Assessement of mood.*

The Children anxiety scale (CAS).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* *Generalised versus right temporal (Table 119)*

* Generalised versus left temporal (Table 120)

* Generalised versus bi-temporal (Table 121)

* Generalised versus secondary generalised (Table 122)

* Right temporal versus left temporal (Table 123)

* Right temporal versus bi-temporal (Table 124)

* Left temporal versus bi-temporal (Table 125)

* Right temporal versus Right temporal with secondary generalisation (Table 126)

* Left Temporal versus left temporal with secondary generalisation (Table 116).   ,

* Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).

* Generalised versus none (Table 118)

The children depression inventory (CDI).

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* Generalised versus right temporal (Table 119)

* Generalised versus left temporal (Table 120)

* Generalised versus bi-temporal (Table 121)

* Generalised versus secondary generalised (Table 122)

* Right temporal versus left temporal (Table 123)

* Right temporal versus bi-temporal (Table 124)

* Left temporal versus bi-temporal (Table 125)

* Right temporal versus Right temporal with secondary generalisation (Table 126)

* Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).

* Generalised versus none (Table 118)

Significant :

There is significant difference between left Temporal versus left temporal with secondary generalisation (Lt. temporal with 2ry. Gen > Lt. temporal) (Table 116).

iii- Assessement of personality.

Psychoticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the EEG in the following :

* Generalised versus right temporal (Table 119)

* Generalised versus left temporal (Table 120)

* Generalised versus bi-temporal (Table 121)

* Generalised versus secondary generalised (Table 122)

* Right temporal versus left temporal (Table 123)

* Right temporal versus bi-temporal (Table 124)

* Left temporal versus bi-temporal (Table 125)

* Right temporal versus Right temporal with secondary generalisation (Table 126)

* Left Temporal versus left temporal with secondary generalisation (Table 116).

* Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).

* Generalised versus none (Table 118)

Neuroticism

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* Generalised versus right temporal (Table 119)

* Generalised versus left temporal (Table 120)

* Generalised versus bi-temporal (Table 121)

* Generalised versus secondary generalised (Table 122)

* Right temporal versus left temporal (Table 123)

* Right temporal versus bi-temporal (Table 124)

* Left temporal versus bi-temporal (Table 125)

* Right temporal versus Right temporal with secondary generalisation (Table 126)

* Left Temporal versus left temporal with secondary generalisation (Table 116).

* Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).

* Generalised versus none (Table 118)

Introversion

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the EEG in the following :

* Generalised versus right temporal (Table 119)

* Generalised versus bi-temporal (Table 121)

* Right temporal versus left temporal (Table 123)

* Right temporal versus bi-temporal (Table 124)

* Left temporal versus bi-temporal (Table 125)

* Left Temporal versus left temporal with secondary generalisation (Table 116)

* Bi-Temporal versus bi-temporal with secondary generalisation (Table 117).

* Generalised versus none (Table 118)

Significant :

There is significant difference between Generalised versus left

temporal (Lt. temporal > Gen.) *(Table 120)*, right temporal versus Right temporal with secondary generalisation (Rt. temporal > Rt. with 2ry. Gen.) *(Table 126)* and generalised versus secondary generalised (Gen. > 2ry. Gen.) *(Table 122)*.

Lie scale

Non-Significant :

No Signifcant difference found in different psychological & congnitive scales between GME and TLE concerning the *EEG* in the following :

* **Generalised versus right temporal** *(Table 119)*

* **Generalised versus left temporal** *(Table 120)*

* **Generalised versus bi-temporal** *(Table 121)*

* **Generalised versus secondary generalised** *(Table 122)*

* **Right temporal versus left temporal** *(Table 123)*

* **Right temporal versus bi-temporal** *(Table 124)*

* **Left temporal versus bi-temporal** *(Table 125)*

* **Right temporal versus Right temporal with secondary generalisation** *(Table 126)*

* **Left Temporal versus left temporal with secondary generalisation** *(Table 116)*.

* **Bi-Temporal versus bi-temporal with secondary generalisation** *(Table 117)*.

* **Generalised versus none** *(Table 118)*

Fig. (6):Comparison between patients with podroma in both GME & TLE groups
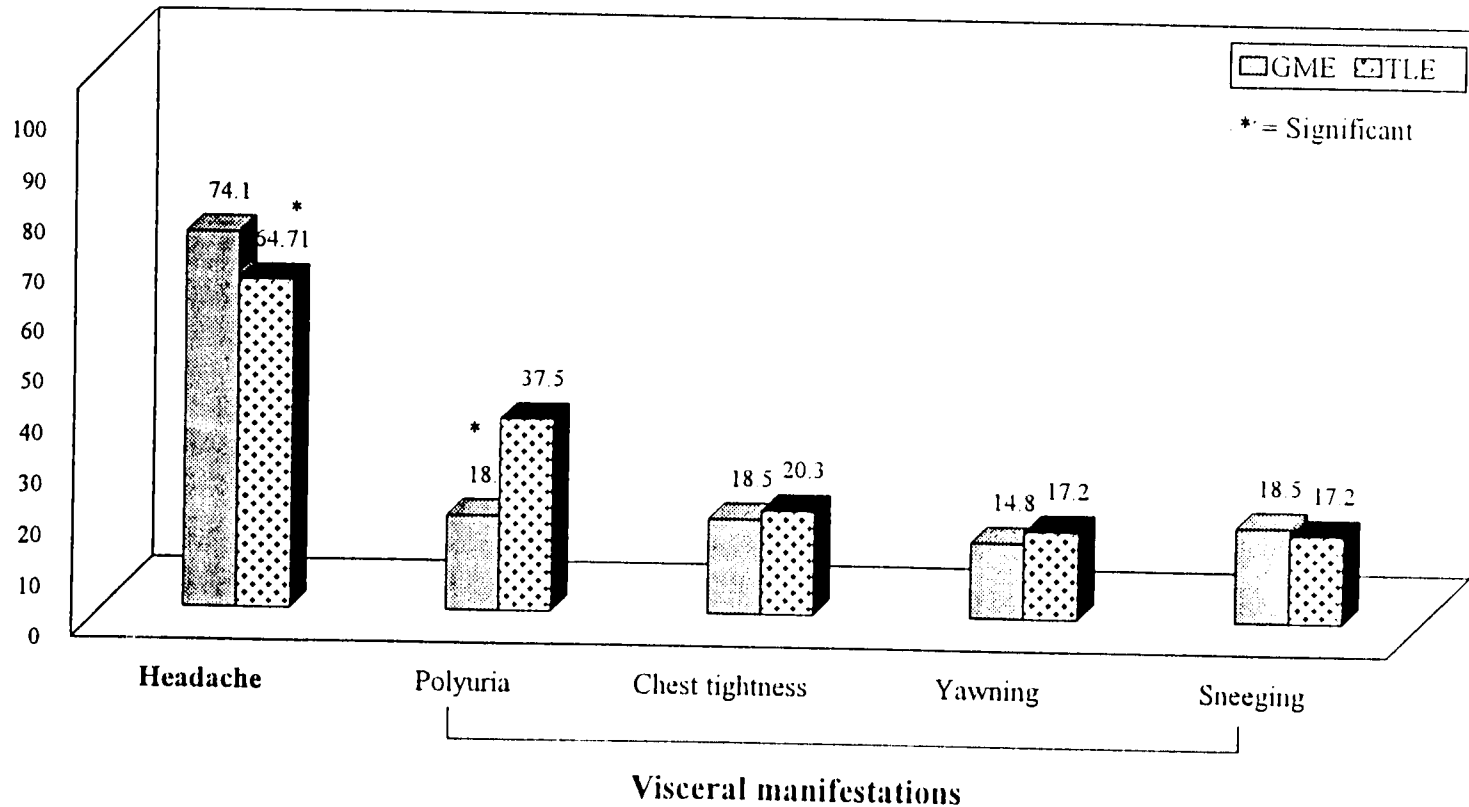
Test used :
Chi-square test .
P (< 0.05) = Significant

Fig. (7):Comparison between patients with podroma in both GME & TLE groups

Changes of mood
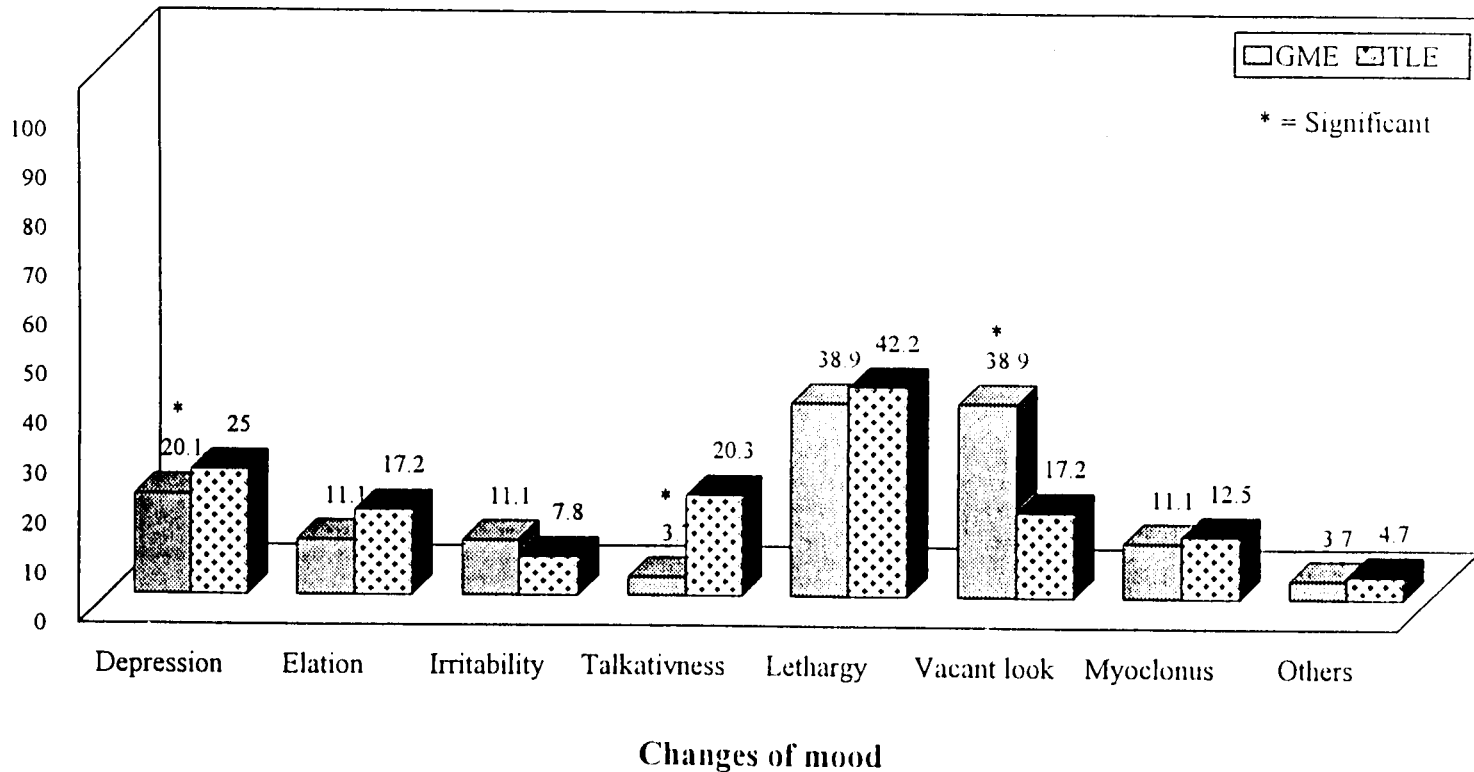
Test used :
   Chi-square test .

Fig. (8):Comparison between GME & TLE patients with AURA
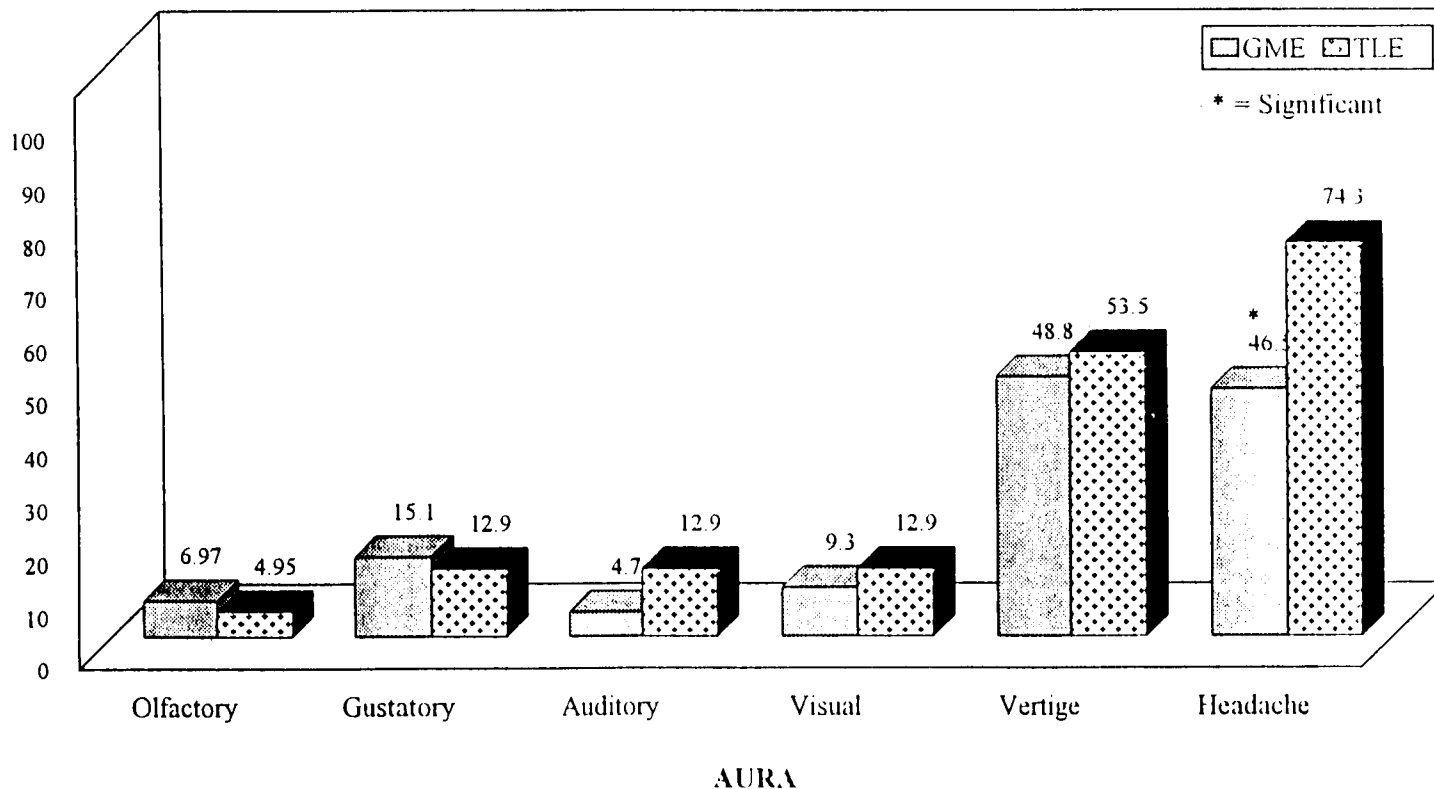
Test used :

Chi-square test .

P (< 0.05) = Significant .

Fig. (9):Comparison between patients with autonomic manfestation of AURA to patients without AURA
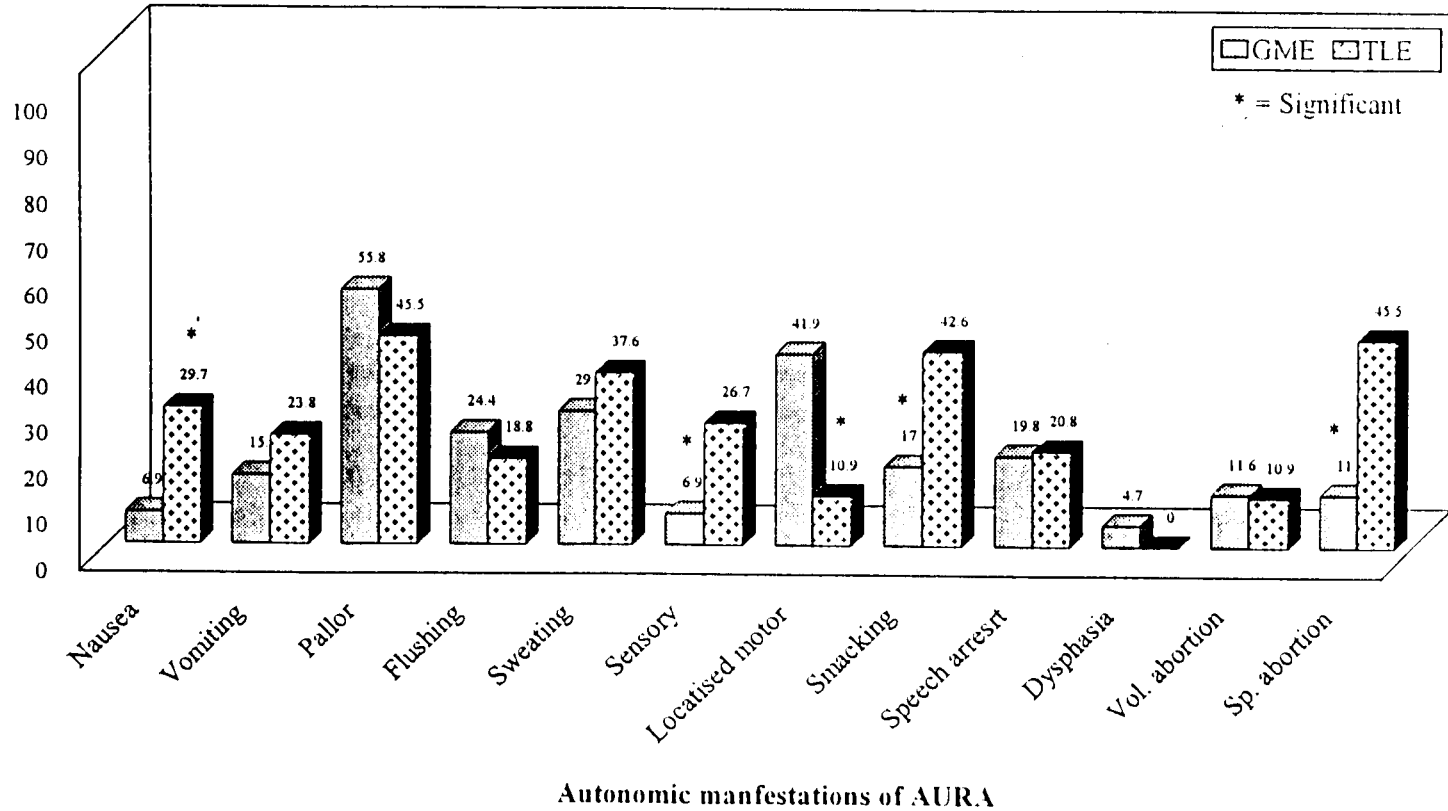
Autonomic manfestations of AURA

Test used :
   Chi-square test .

Fig. (10):Comparison between GME & TLE patients with post ictal changes

Fig. (10):Comparison between GME & TLE patients with post ictal changes

Legend: □ GME □ TLE
* = Significant

Values (GME, TLE):
- Aphasia: 21.4, 7.9
- Dysphasia: 30.8, 23.8
- Paresis: 11.1, 4.95
- Restlessness: 34.18, 39.6
- Automatiom: 12.8, 12.9
- Psychotic state: 35.9, 20.8 (*)

Test used :
Chi-square test .
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant

251
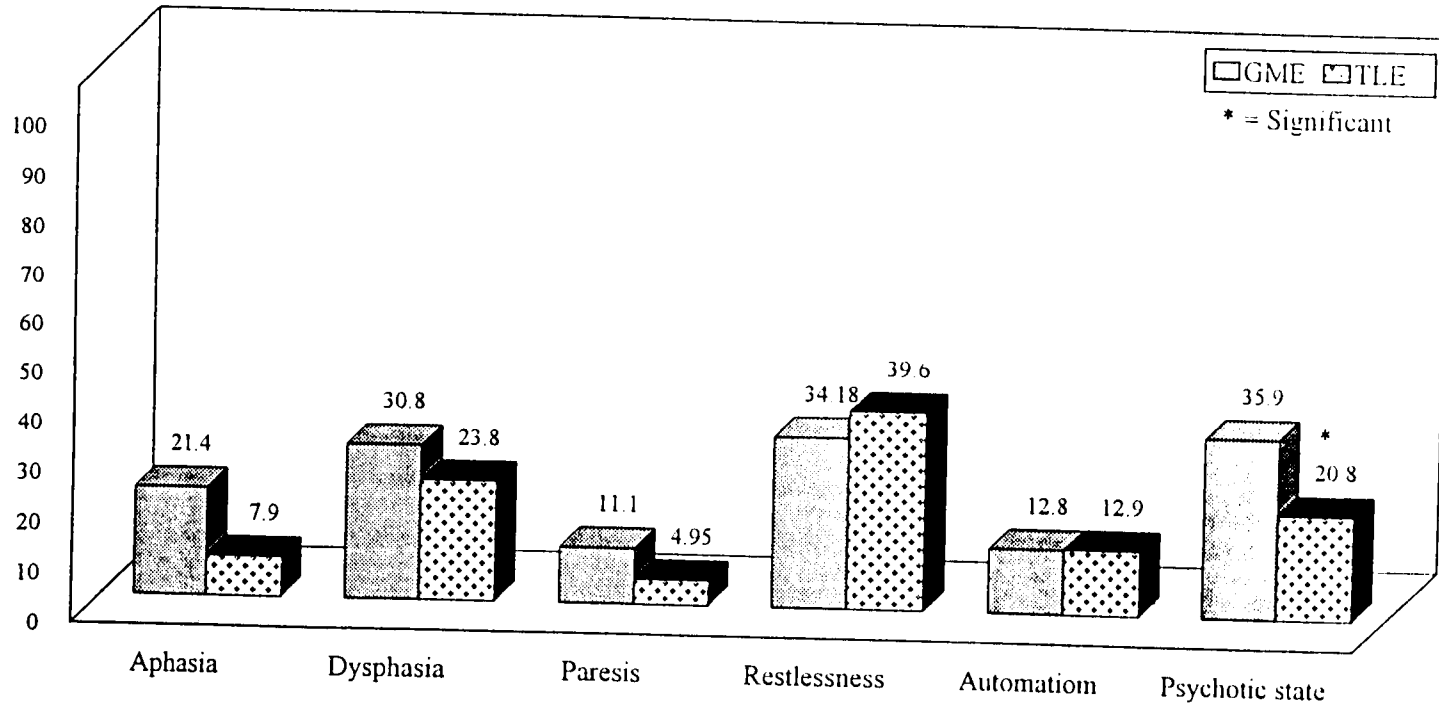
Fig. (11):Distribution of factors affecting ictus among GME & TLE patients
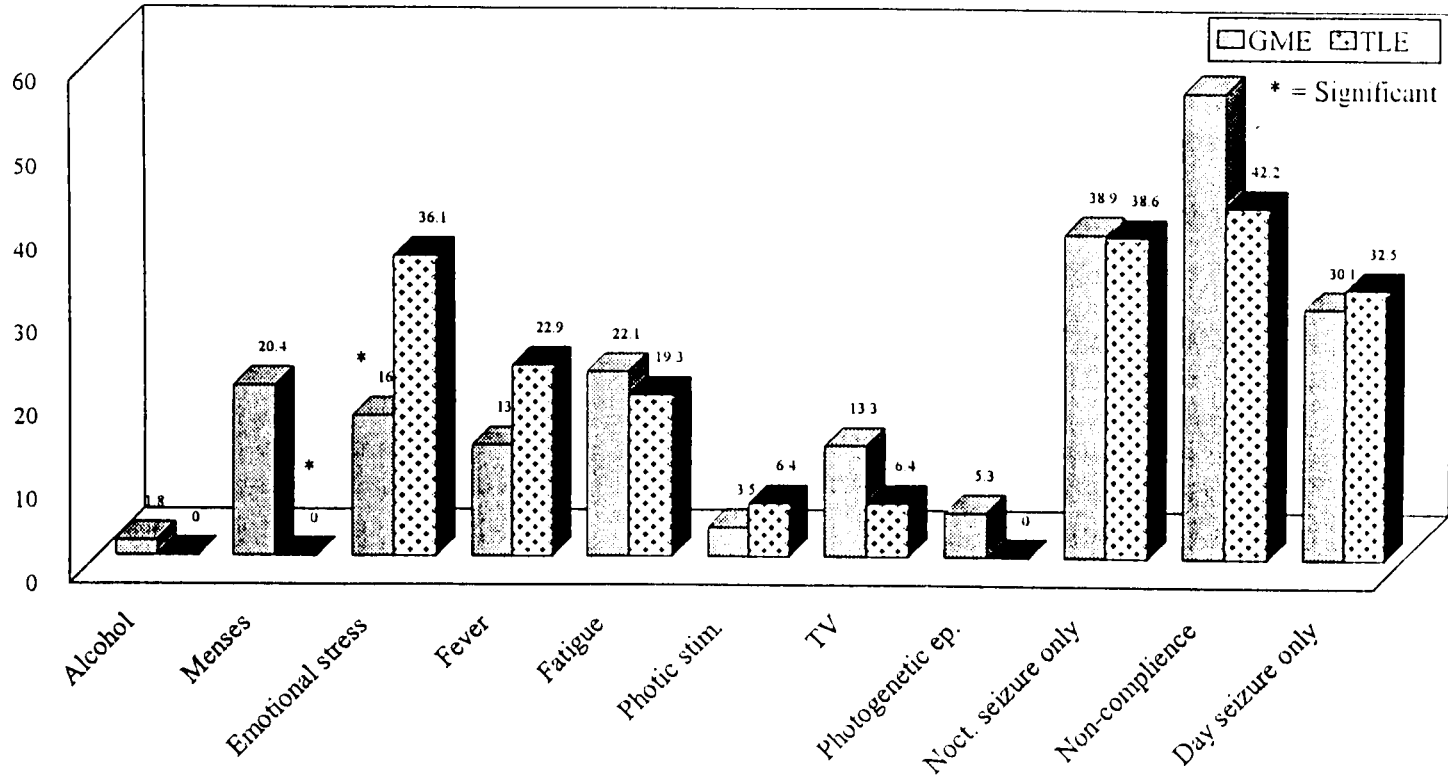
Test used :
  Chi-square test .
P (< 0 .05) = Significant .

Fig. (12):Comparison between GME & TLE concerning cerebral causes
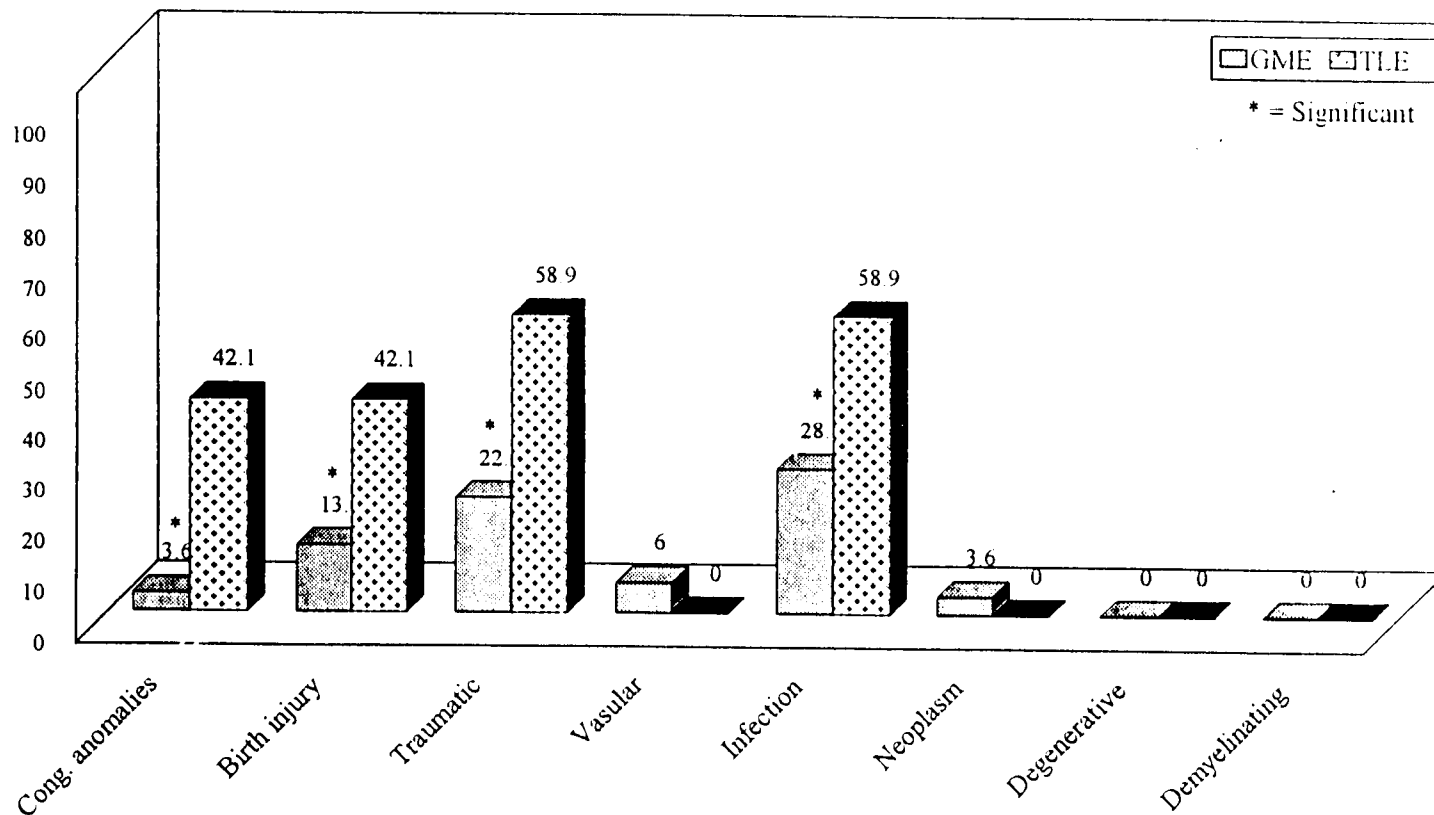
Test used :
   Chi-square test .
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant .

Fig. (13):Comparison between GME & TLE concerning systemic causes

254
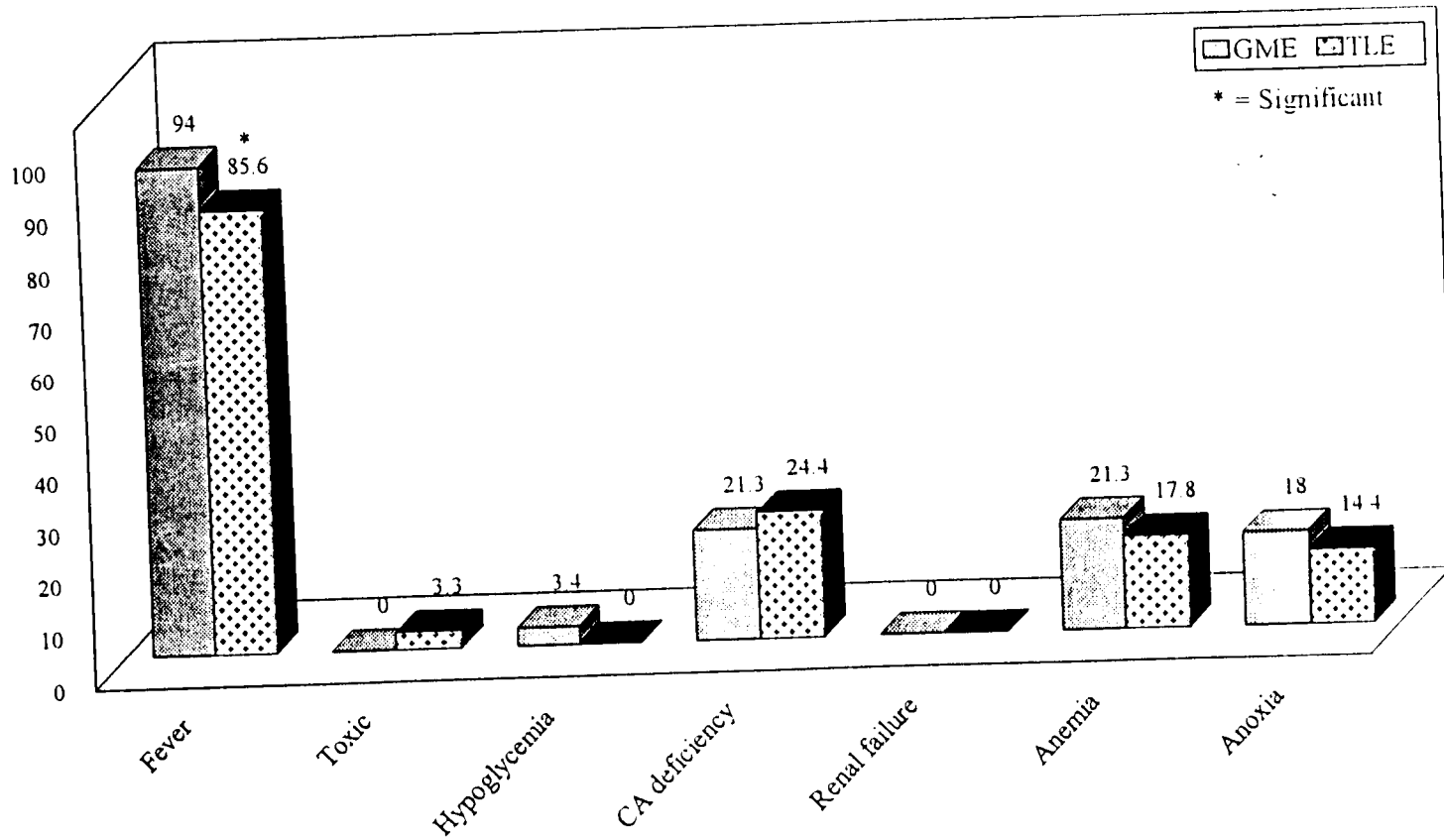
Fig. (14):Comprison between GME & TLE concerning Iatrogenic
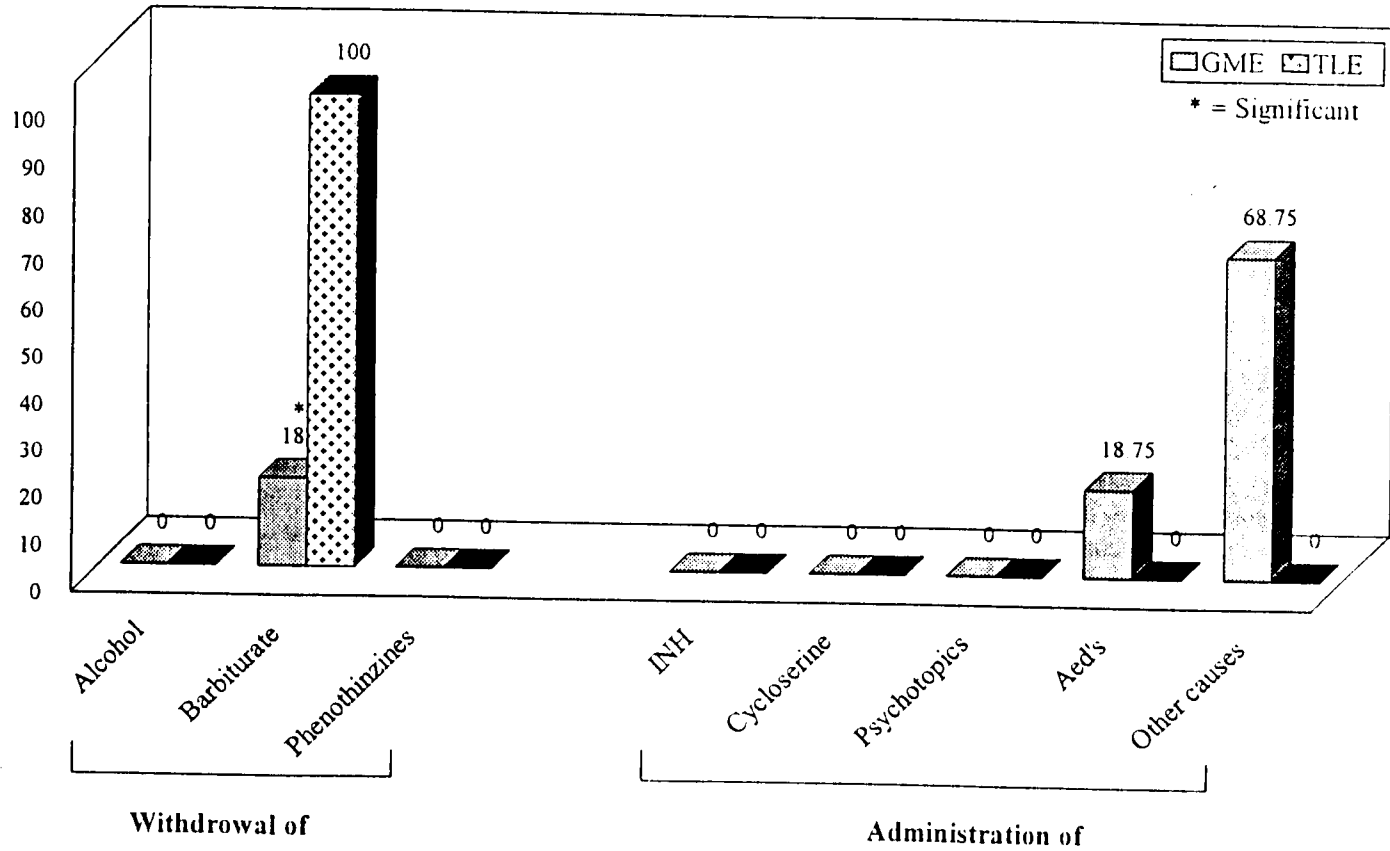
Test used :
Chi-square test .
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant .

Fig. (15):Comprison between GME, TLE patients & control groups among history of organic disorders
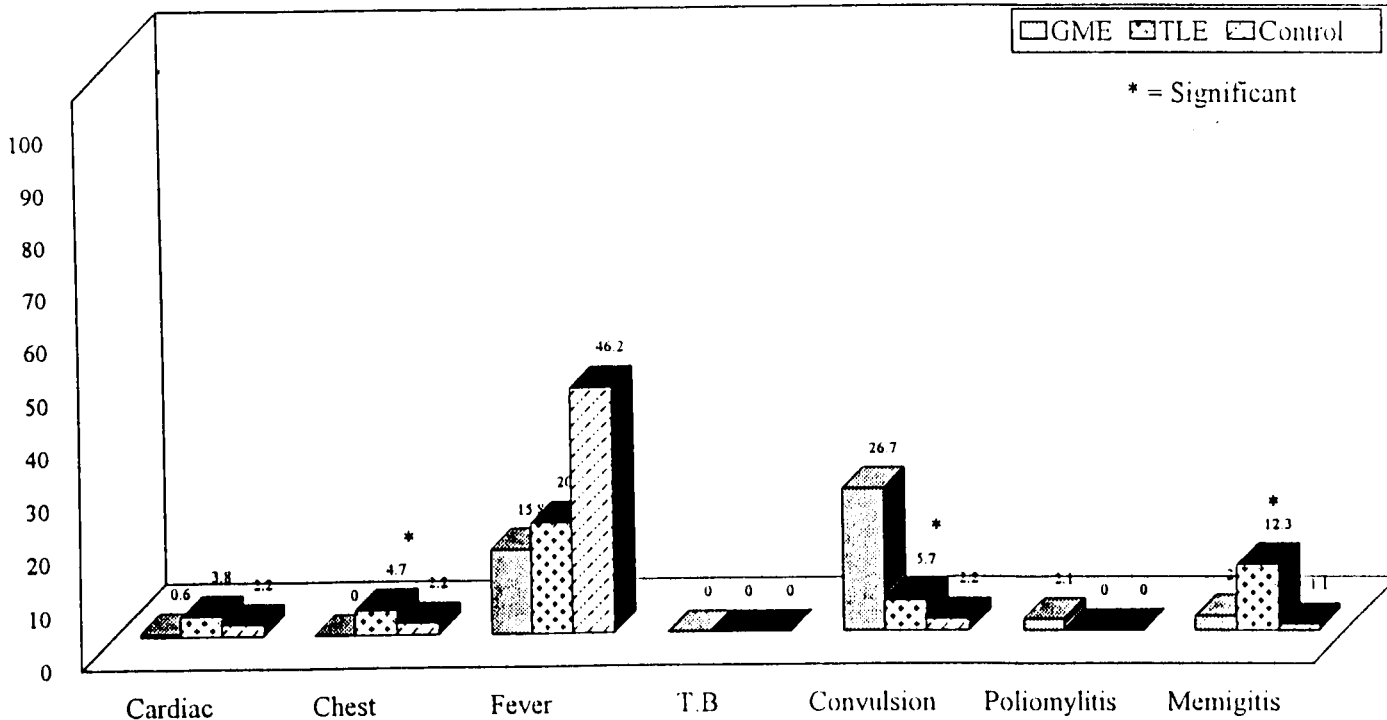
Test used :
   Chi-square test .
P (< 0.05) = Significant .

Fig. (16):Comparison between GME, TLE & control groups concerning the neuretic traits
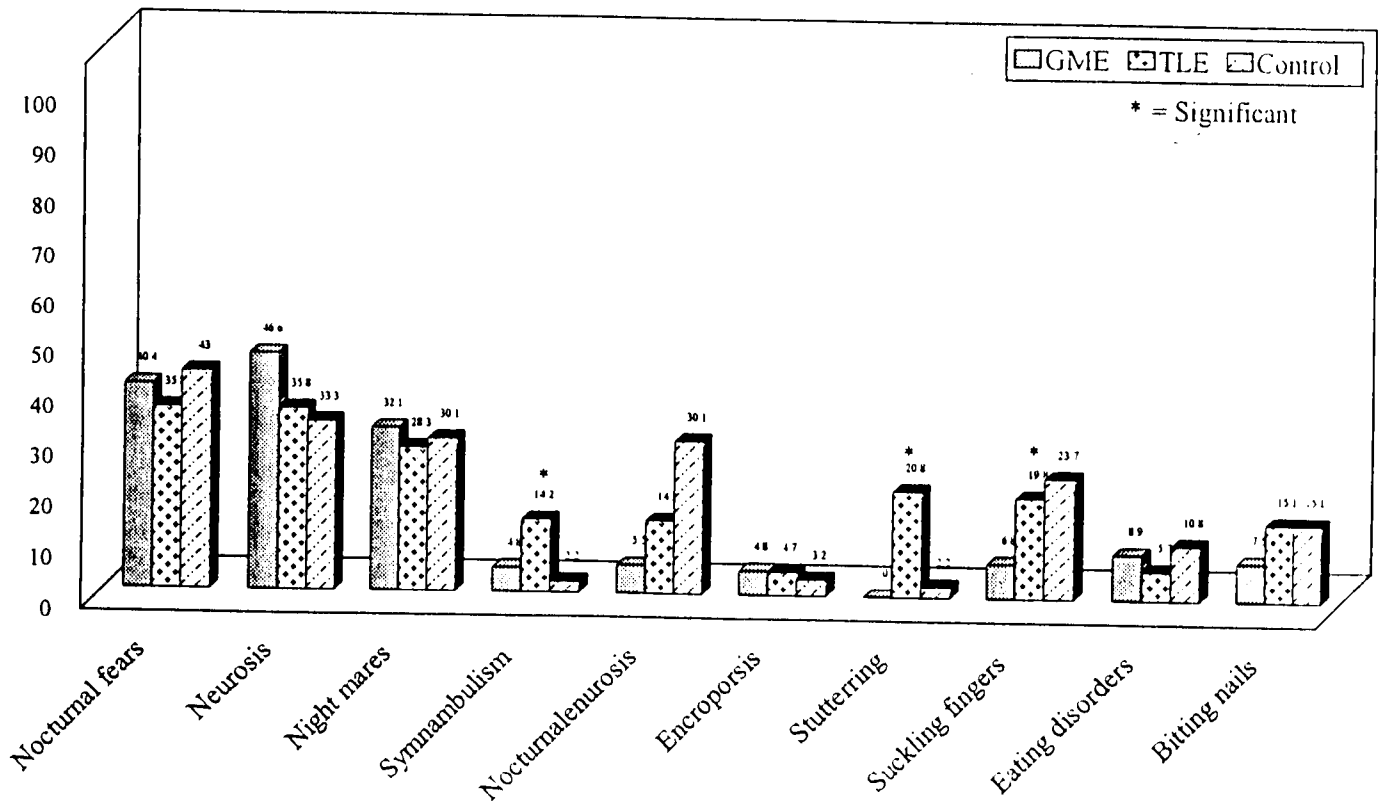
Test used :
   Chi-square test .
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant .

Fig. (17):Comprison between GME, TLE & control groups concerning Soduim Valproate (Depakine) side effects
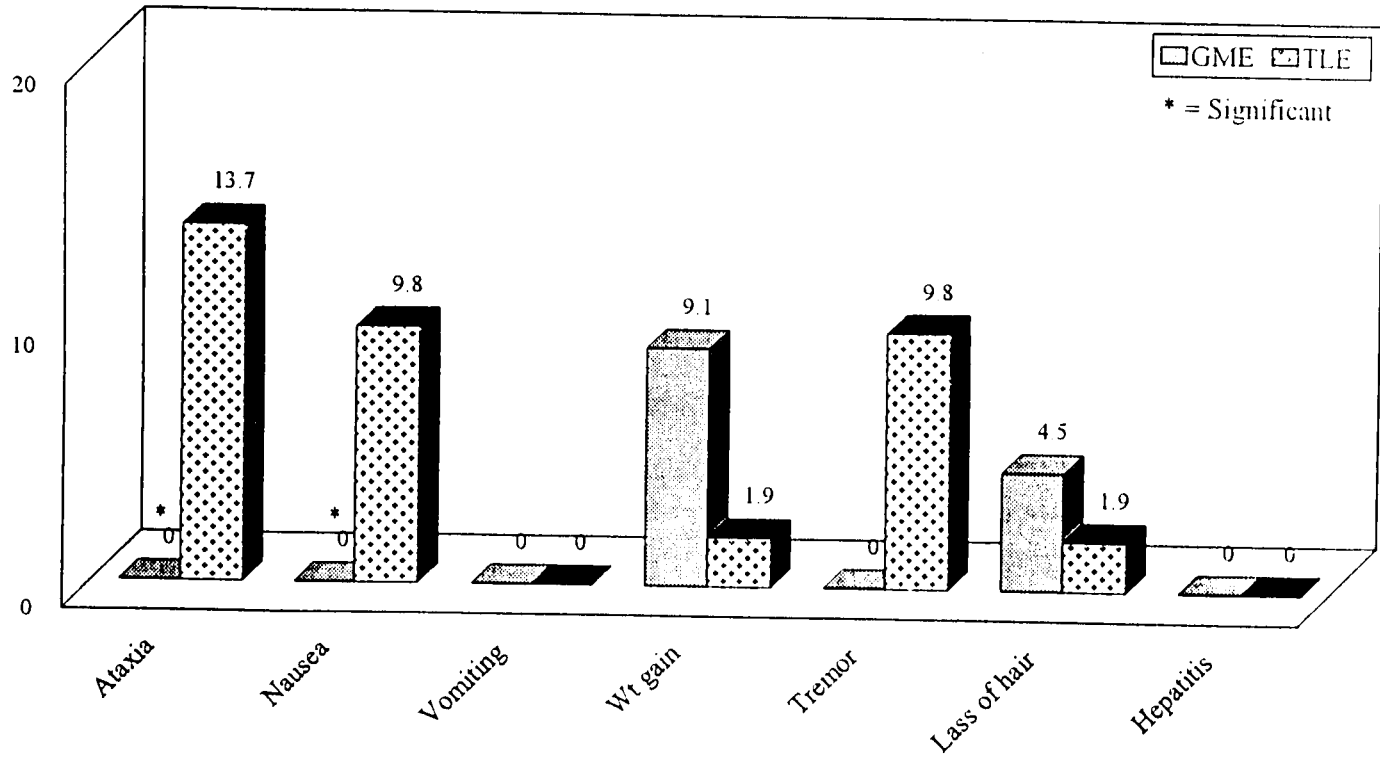
Test used :
    Chi-square test ,
P (< 0.05) = Significant .

Fig. (18):Comprison between GME, TLE & control groups concerning (Carbamazepine) Tegretol side effects
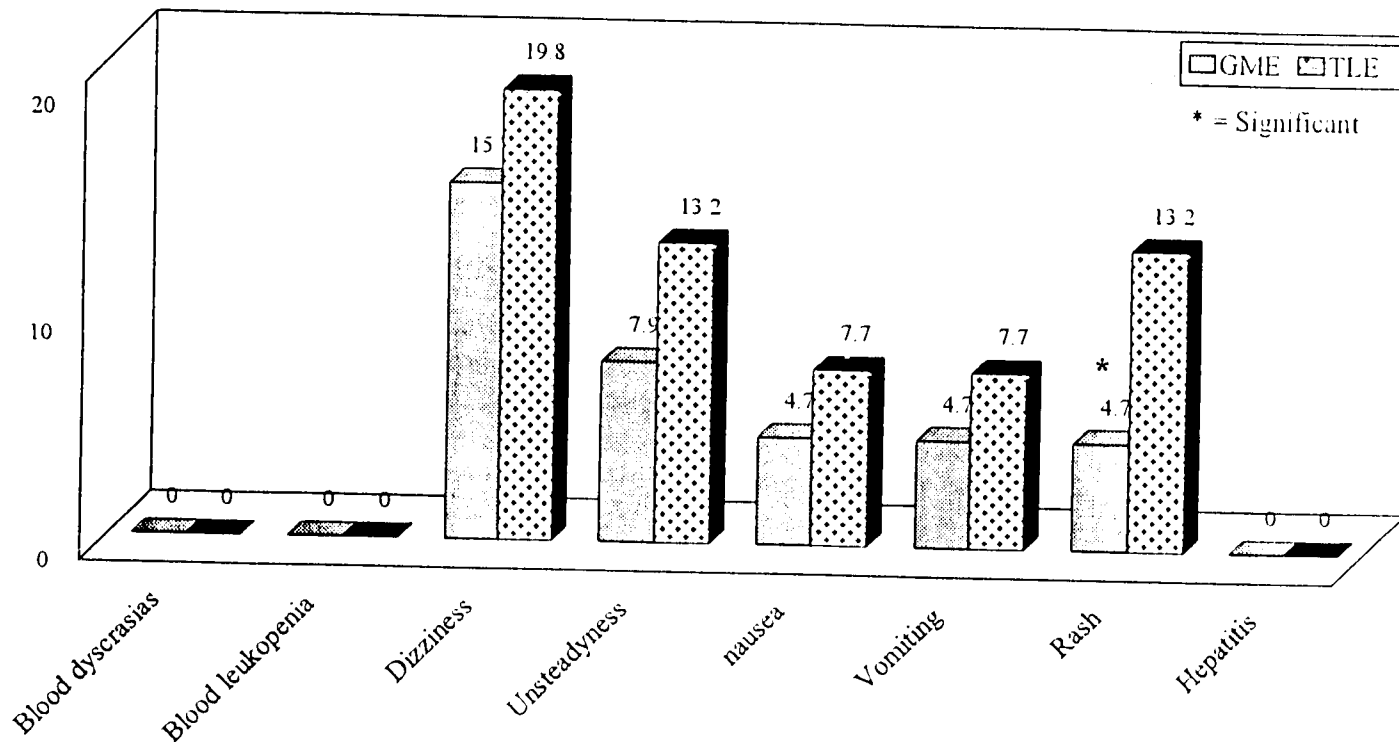
Test used :
  Chi-square test .
P (< 0.05) = Significant .
P (> 0.05) = Non-signif.

Fig. (19):Comprison between GME, TLE & control groups concerning Hydantoin (Epanutin) side effects
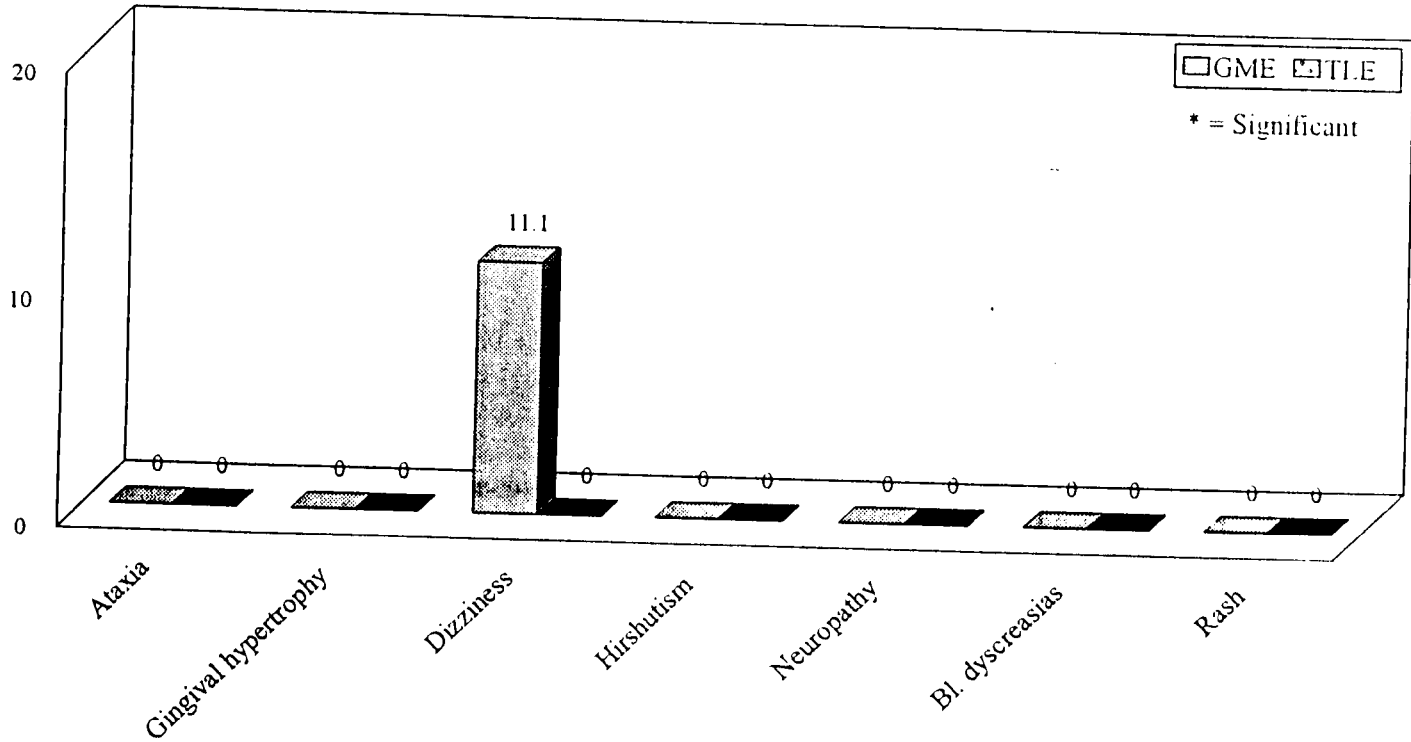
Test used :
Chi-square test .
P (< 0 .05) = Significant .
P (> 0.05) = Non- significant

Fig. (20):Comprison between GME, TLE & control groups concerning Colonazepame (Rivotril) side effects
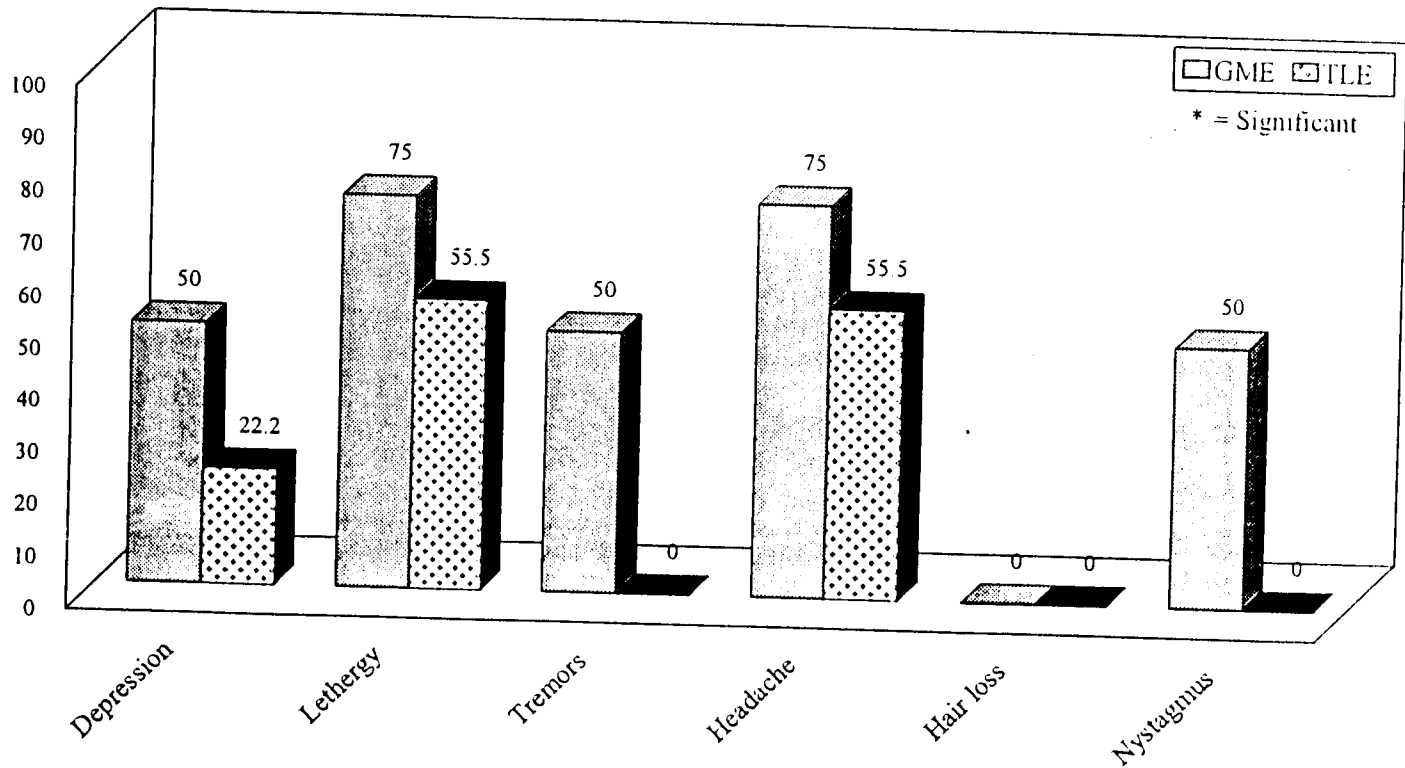
Test used :

Chi-square test .

P (< 0 .05) = Significant .

P (> 0.05) = Non- significant .

Fig. (21):Comparison between GME, TLE patients among the whole study

Through: Sub-wechseler

- Coding test
- Digit span test
- Similarities test
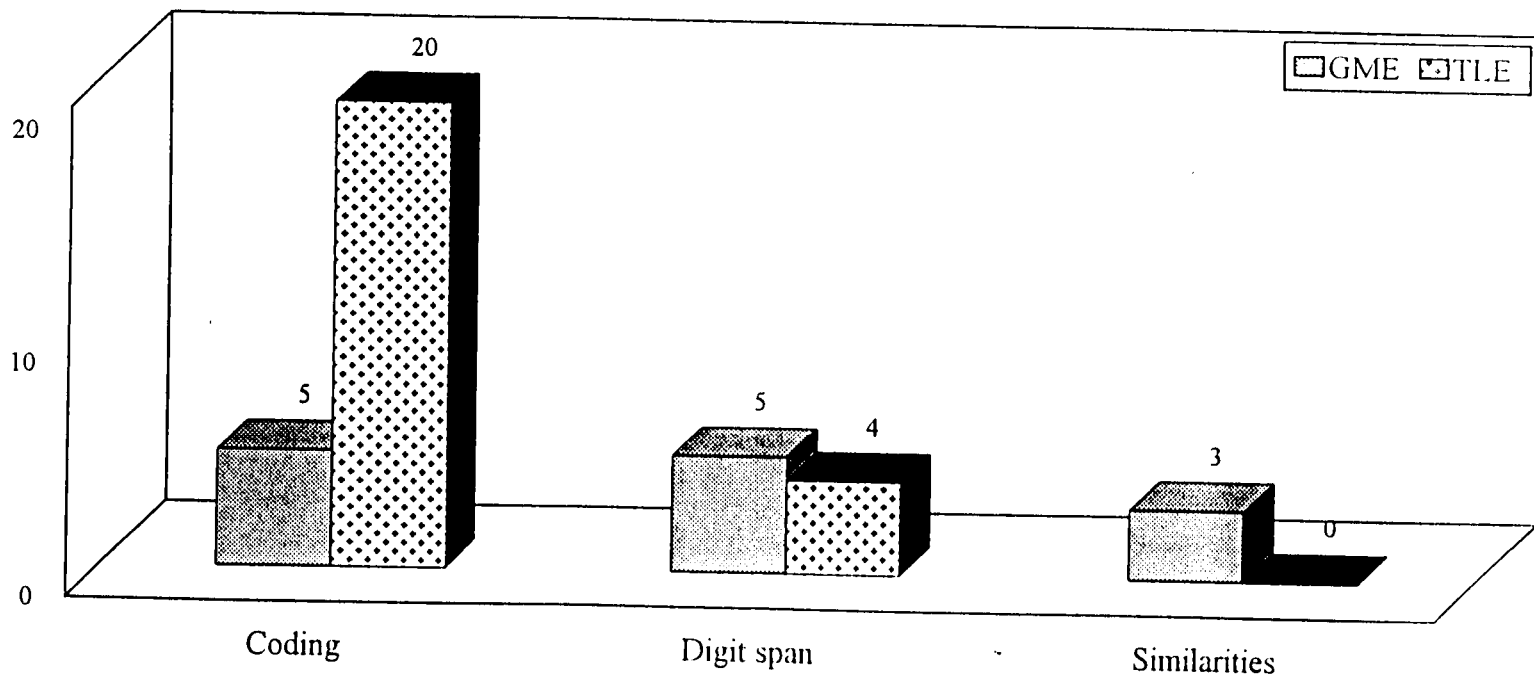
This figure shows an increased affection of TLE more than GME in three above tests

Fig. (22):Comparison between GME, TLE patients among the whole study Through:

- **Cancellation letters (Time & Errors**
- **MFFT (Time & Errors)**
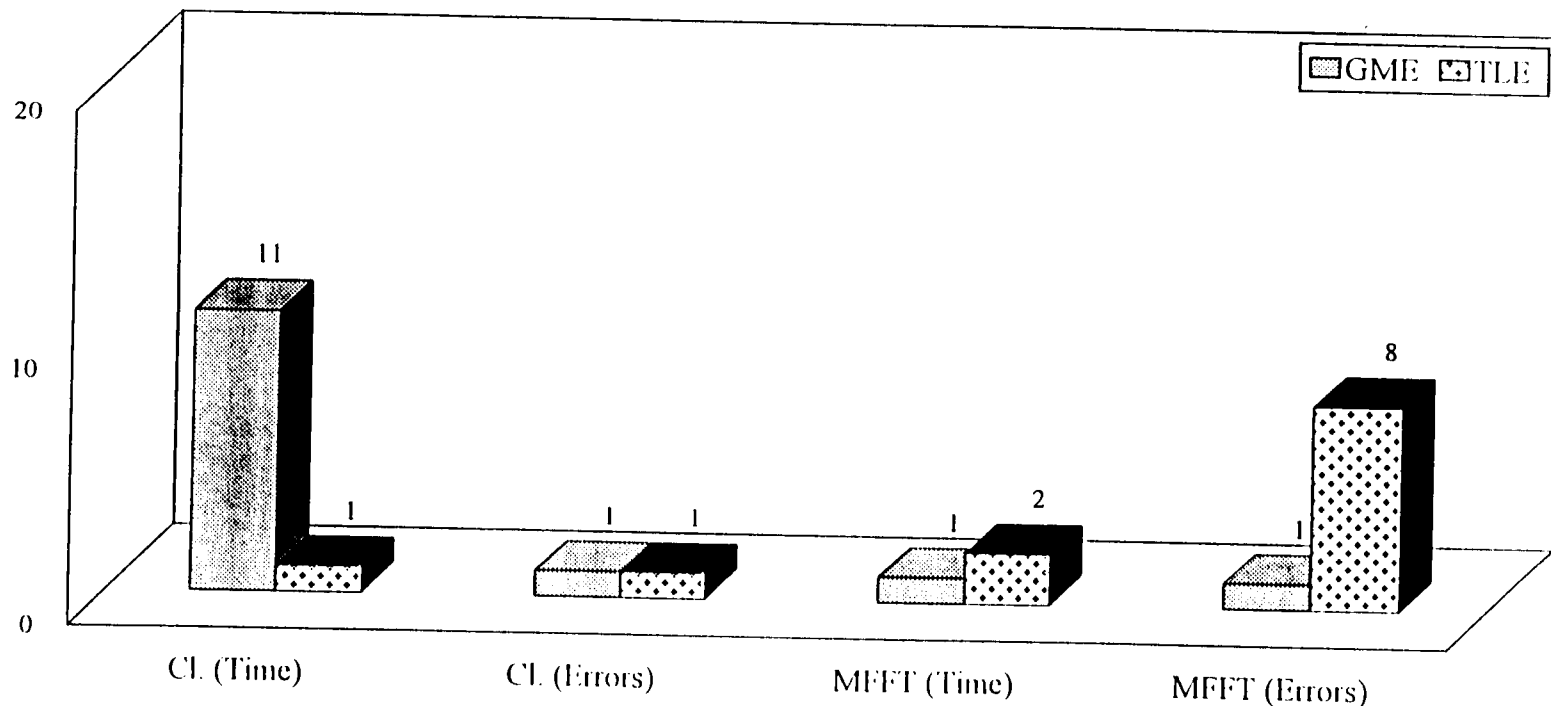
This figure shows an increased affection of GME more than TLE in Cancellation letters Time &
This figure shows an increased affection of TLE more than GME in other tests

Fig. (23):Comparison between GME, TLE patients among the whole study Through:

- **Anxiety**
- **Depression**
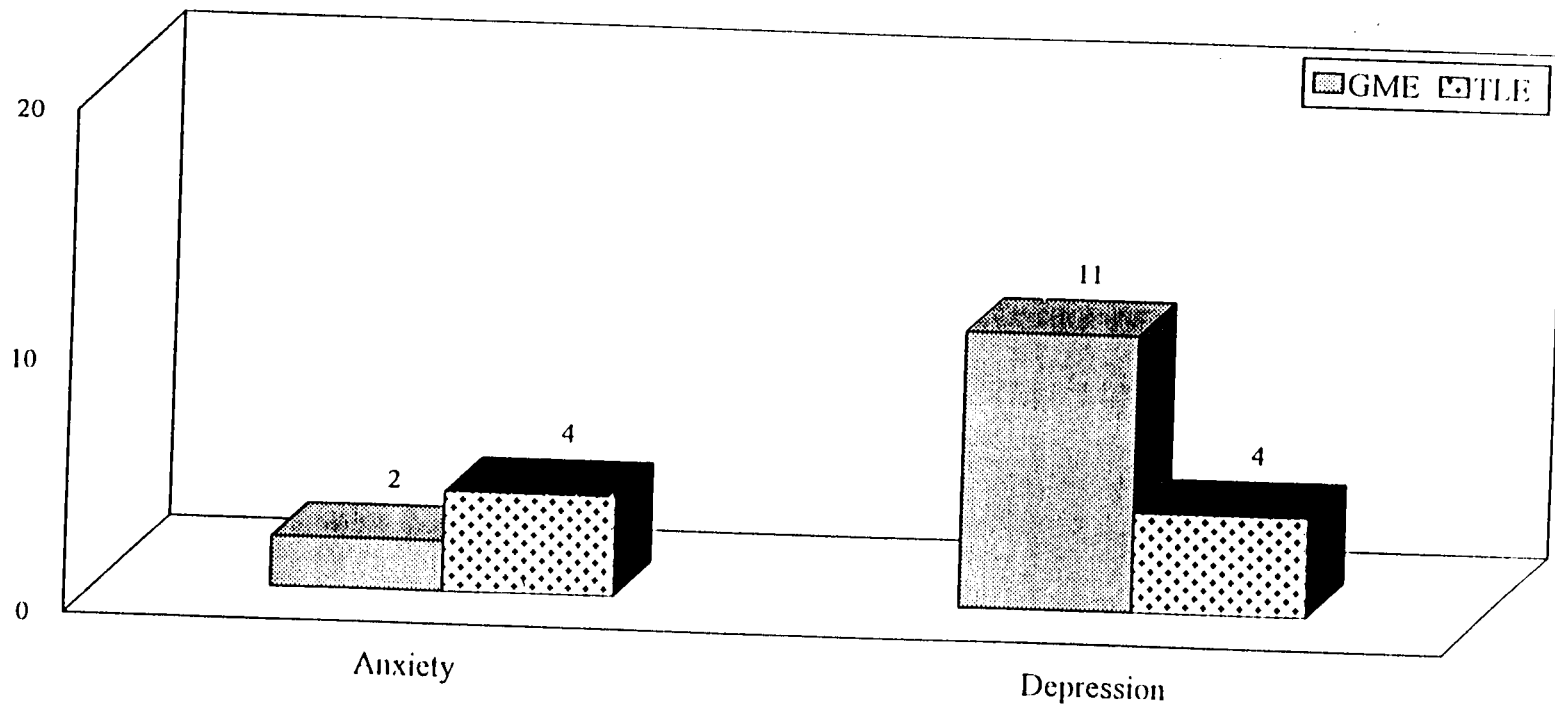
This figure shows an increased affection of GME more than TLE in Depression test (CDI)
This figure shows an increased affection of TLE more than GME in Anexity test (CAS)

Fig. (24):Comparison between GME, TLE patients among the whole study Through:

• JEPG
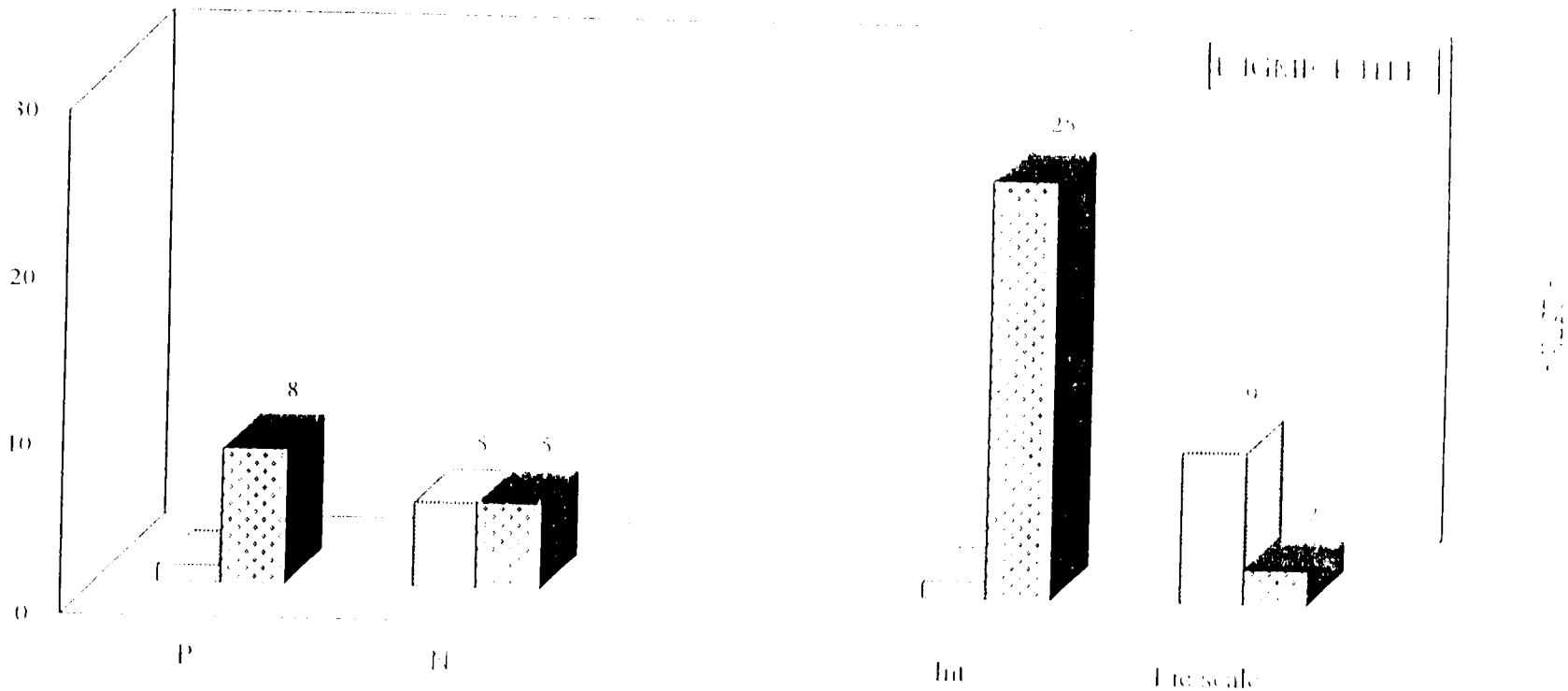
This figure shows an increased affection of GME more than TLE in neuroticism & lie scale
This figure shows an increased affection of TLE more than GME in psychoticism, introversion & extraversion

Discussion

Discussion

It is apparent to the clinician that children with epilepsy do not form a homogeneous group. But differ along a number of dimension , namely aetiology ; seizure variables (type, frequency, age of onset and duration of the seizure disorder): EEG activity ; antiepileptic medication ; sex and environment. Thus , it is important to raise the question as to the nature of scholastic performance . this may also help in understanding first, the discrepant findings with respect to intellectual ability; and secondly , the poor scholastic performance of children who are of at least average intellectual ability.(christine.1988).

PSYCHOLOGICAL TESTS: -

i- Assessement of cognition:

1-Coding:

In our study there were significant difference between GME, TLE patients & control group in sustained attention, persistency, pyschomotor speed, visuomotor coordination and Learning ability.

There was significant increase in control group more than GME and TLE patients.

This means that control group is better than both GME & TLE in sustained attention persistency, psychomotor speed, visuomotor coordination, & Learning ability.

Which means that epilepsy with its two varieties (GME & TLE) has a great role in decreasing sustained attention persistency psychomotor speed visuomotor coordination and learning ability.

The finding of this study contradict with results represented by christine, 1988 as regards contradictory results this part needs further study. Children with grand mal seizures, psychomotor or partial seizures have also been found to be unimpaired on tests of intellectual ability.

In a direct comparision of different siezure types, found that children with partial seizures obtained between scores on some Wechsler subtests than did those with generalized.

2-Digit span

In our study there were no significant difference between GME, TLE patients and control group in either or all of immediate memory, auditory attention, short memory and internal visual scanning.

This means that control group is not better than both epileptic groups (GME & TLE) in either or all of immediate .memory auditory attention, short memory & internal visual scanning.

Which means that epilepsy with its two varieties (GME & TLE) have no role in immediate memory, auditory attention, short memory & internal visual scanning.

This contradict with results reported by Ogunrin and his colleagues, 1995 as regards the significant impairments in memory tasks and attention were poted, with worsening observed after anticonuulsant therapy. Factors such as type of epilepsy, frequency of and age at onset of seizures adversely affected the cognitive function of the patients. In addition, results of the Epilepsy were standardized with the common psychometric test, i.e. Wechsler Adult Intelligence Scale.

3) similarities:

In our study there were significant difference between GME, TLE patients and control group in conceptualization, concrete thinking, and general mental ability.

There was an increase in control group more than GME and TLE patients.

There was also an increase in TLE patients more than GME patients.

This means that control group is better than both GME & TLE in conceptualization, concrete thinking and general mental ability TLE patients is better than GME patients.

Which means that Epilepsy specially GME has a great role in conceptualization, concrete thinking and general mental ability.

This goes with results reported by Blennow and his colleagues, 1990 as regards the cognitive function is frequently impaired in children with epilepsy, compared with age-matched controls. It can be hard to evaluate the significance of various contributory factors. The effects of antiepileptic drugs may be studied in children who have outgrown their epilepsy but are still being treated

4) Cancellation letters test (Time & Errors)

In our study there were significant difference between GME, TLE patients and control group in attention and response rate.

There was significant increase in control group more than GME & TLE patients.

There was no significant difference between GME & TLE patients but (GME>TLE) patients.

This means that control group is better than both GME & TLE in attention & response rate.

Which means that epilepsy with its two varieties (GME & TLE) has a great role in attention & response rate.

This goes with results reported by Aldenkamp and his colleagues, 1997 as regards the slowing is the most dominant

cognitive impairment for the children with epilepsy. This may dislose the specific influence of epilepsy on learning.

5) Matching familiar figure test-Total errors and total and Mean time (MFFT):

In our study there was significant difference between GME, TLE patients and control group (normal population) in impulsivity, attention & response rate.

There was significant increase in GME patients more than control group (normal population).

There was significant increase in TLE patients more than control group (normal population).

There was no significant difference between GME and TLE patients .

This goes with results reported by Harold and his colleagues, 1995 as regards the term "epileptiod personality" has been used to convey the seizure like quality of the characteristic outbursts. Which are not typical of the patient, and to convey the suspicion of an organic disease process. A number of associated features suggest the possibility of an epileptoid state: the patient may experience an aura post-ictal like changes in the sensorium, including partial or spotty amnesia; or hypersensitivity to photic, aural, or auditory stimuli. Persons with the disorder have a high incidence of hyperactivity, soft neurological signs, non specific electroencephalogram (EEG) findings, and accident-proneness.

Intermittent explosive disorder is found in persons who have discrete episodes of losing control of aggressive impulses, resulting in serious assault or the destruction of property.

The degree of aggressiveness expressed is grossly out of proportion to any stressors that may have helped elicit the episodes. The symptoms, with the patient may describe as spells or attacks, appear within minutes of hours and, regardless of duration, remit spontaneously and quickly. Each episode is usually followed by genuine regret of self-reproach. Signs of generalized impulsivity or aggressiveness are absent between episodes. The diagnosis of intermittent explosive disorder should not be made if the loss of control cam be accounted for by schizophrenia, antisocial or border-line personality disorder, attention - deficit / hyperactivity disorder, or substance intoxication.

Six categories of impulse - control disorders not elsewhere classified are listed in the fourth edition of Diagnostic and statistical Manual of Mental Disorders (DSM-IV): intermittent explosive disorder, ... etc.

Patient with disorders of impulse control show the following features: (1) The fail to resist an impulse, drive, or temptation to perform some action that is harmful to themselves or others. They may or may not consciously resist the impulse and may not plan the act. (2) Before committing the act, they feel an increasing sense of tension or arousal (3) while committing the act, they feel pleasure, gratification, or

release. The act is ego, syntonic in that it is constant after the act, the patients may or may not feel genuine regret, self-reproach, or guilt .

ii- Assessement of mood :

1- The children anxiety scale (CAS).

In our study there was no significant difference between GME, TLE patients and control group (normal population) in increasing anxiety.

GME, TLE group of patients increase anxiety significantly more than control group (normal population).

This goes with results reported by Mary 1988, as regards the Mode disorders occur frequently in people with epilepsy, but this applies predominantly to anxiety, depressive symptomatology and depressive illness .

This goes also with results reported by John and his colleagues, 1997 as regards the Anxiety and depression are the most common psychiatric conditions associated with epilepsy. Understanding the development & maintenance of these symptoms requires investigate of interrelations between biological and psychological factors.

And also with results reported by Collinge, and his colleagues, 1997 as regards the Fits increasingly experienced episodes diagnosed as panic attacks.

This goes also with results reported by Uhlmann and his colleagues, 1997 as regards the most epileptic patients are able to control physiological parameters by intensive training and, as a result, improve clinically. Nevertheless, behavior therapy seems to have some influence on the amount of seizure reduction.

2- The children depression inventory (CDI) :

In our study there was a significant difference between GME, TLE patients and control group (normal population) in increasing depression.

There was no significant difference between each group and other but there is increase in GME > TLE , GME > control group and TLE > control group .

This goes with results reported by Mary 1988, as regards the question as to whether those patients with temporal lobe epilepsy complex partial seizures are more prone to depression is largely unanswered. Brown and his team found that those with temporal lobe epilepsy complained of more irritability and impaired concentration and were rated as more depressed and "showed up", disagreeing with Trimble and Perez who found no relationship between type of epilepsy and depression.

Personality Inventory and left sided foci patients scored significantly higher than right-sided subjects on the trait depression and emotionality combined.

Camfield and his team assessed 27 children with lateralized temporal lobe epilepsy (14 = right, 13 = left), with parents completing a personality inventory for children: no right - left differences were found no depression or anxiety scores, Brandt and his team tested the laterality hypothesis, and failed to support the nation that sadness mood were associated with right temporal lobe disturbance.

This goes also with results reported by Katharine and his colleagues, 1995 as regards the This includes the ability of the physician to develop confidence in the patient's ability and for the patient to become partly responsible for supporting the physician's anxiety and utilizing the physician's knowledge as medication is withdrawn. It is a potentially refreshing, if occasionally difficult, experience!

Moreover, the phenomenology of the depression does not, on the whole, seem linked with epilepsy variables. Anticonvulsants can however affect the mental state, and the longer the duration of epilepsy the more severe the depression. The majority of studies implicated the left hemisphere with hints that temporal lobe/complex partial seizure patients are more vulnerable.

And also with results reported by Muscas and his colleagues, 1997 as regards the At this moment patients treated with GBP improved better than control in 2 neuropsychologicl tasks. No adverse effect on mood was observed in both the groups.

This goes also with results reported by Siffels and his colleagues, 1997 as regards the in psychological research of depression much attention has been given to cognition as aetiologic and mediating factors.

And also with results reported by Mares 1997, as regards the postictal depression is a complex phenomenon generated by more than one inhibitory system.

And also with results reported by Boglium, and his colleagues, 1997 as regards that of a series of psychiatric symptoms and reactions, only depression seems to predominate in idiopathic and cryptogenic epilepsy and can be attributed to epilepsy per se.

This goes with results reported by Lim and his colleagues, 1986 as regards the prolonged depressive mood swing have been recorded during status epilepticus.

And also with results reported by Mary 1988 as regards the Phenobarbital has been implicated in the emergence of both depressive symptomatology and suicide, and a similar associated has been shown with Vigabatrin.

And also with results reported by John and his colleagues, 1995 as regards the Anxiety and depression are the most common psychiatric conditions associated with epilepsy. Understanding the development & maintenance of these symptoms requires investigate of interrelations between biological and psychological factors

And also with results reported by Monace and his colleagues, 1995 as regards the Depressive mood disorders are often associated with epilepsy, and the severity of depression may seriously influence the prognosis of the disease, thus increasing the risk of suicide. Therefore, anti-epileptic drugs (AEDs) combined with anti-depressants (AD) are often routinely administered clinically. Determination of plasma levels of some of these drugs has allowed evaluation of the kinetic modifications in the course of such combined therapies, with doses adjusted in case of sub-therapeutic or toxic levels.

And also with results reported by Evan and his colleagues, 1988 as regards that no studies have directly addressed the question as to whether depression occurs more frequently in people with epilepsy compared to other conditions, but conclusions may be drawn indirectly from the results of investigations using standardizes rating scales. Uncoordinated behavior typical cases can be found despite lack of characteristic EEG changes.

iii- Assessment of personality:

(Psychoticim, Neuroticism, Introversion & Lie scale.)

1- Neuroticism:

In our study there was significant difference between GME, TLE patients and control group (normal population) in neuroticism where GME > TLE and TLE > control groups.

This goes with results reported by Fitzpatrick and his colleagues, 1997 as regards that there is a considerable literature examining the rates of personality disorder and abnormal personality traits in patients with epilepsy. Most of this research has relied on clinical interviews or the use of self-rating questionnaires to generate personality disorder diagnosis. These instruments have low levels of reliability and have been superceded by structured and semi-structured interviews.

2- Psychoticim:

In our study there was no significant difference between GME, TLE patients and control group (normal population) in psychoticism. But there are increase in psychoticism in TLE > GME and GME > control groups.

This goes with results reported by Traynor and his colleagues, 1997 as regards the Verbalization and vocalization are common in complex partial epilepsy and that vocalization has significant lateralising value to the non-dominant temporal lobe.

3- Introversion:

In our study there was no significant difference between GME, TLE patients and control group (normal population) in introversion. But there are increase in introversion in TLE > control and control > GME groups.

This goes with results reported by Adbulghani and his colleagues, 1997 as regards the Children with abnormal EEG had significant deterioration of personality and behavior adjustment. This was more apparent in patients with focal EEG dysrythmia. Intellectual deterioration was not related to EEG abnormalities.

4- Lie scale:

In our study there was no significant difference between GME, TLE patients and control group (normal population) in lie scale. But there are increase in lie scale in GME > control and control > TLE groups.

This goes with results reported by Fitzpatrick, and his colleagues, 1997 as regards that there is a considerable literature examining the rates of personality disorder and abnormal personality traits in patients with epilepsy.

This means that, there is significant difference between GME, TLE and control group in increasing neuroticism showing that, GME>TLE > control groups.

This goes with results reported by Acharya and his colleagues, 1997 as regards the WMS-R is useful in predicting

postoperative verbal memory changes following left temporal lobectomy.

This goes with results reported by Michael, 1988 as regards the effects of anticonvulsants on cognitive function and behaviour can be summarized in that Carbamazepine phenytion and sod. valproat have minimal effect and phenobarbiton and Clonazepam improve behaviour.

I) Psycho demographic profile:

N.B :

i- <u>Generally means</u> the comparison between GME, TLE and control groups concerning the Psycho demographic profile

ii- <u>In details means</u> the relation between different aspects of Psycho demographic profile and performance on the different psychometric tests

i- <u>Generally</u>

Our study showed that the family size of GME patients are significantly higher in number than that of TLE patients.

It shows also that the family income in GME patients was significantly higher than that of TLE patients.

And showed also that GME patients whom wanted during peri-natal period were significantly more than that of TLE patients whom wanted during the same period.

And showed that GME patients whom lived in small number of rooms were more significantly than TLE patients that live in small number of rooms.

In our study there were, no significant difference between GME and TLE groups as regards gender, sib-order, class, parental separation and patients with educated parents.

ii- In details

1) Gender:

In our study there was significant decreases in either or all of immediate memory, auditory attention, short memory and internal visual scanning in male TLE patients.

This means that there is significant decrease in either or all of immediate memory, auditory attention, short memory and internal visual scanning in male TLE patients. This is beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Henriksen and his colleagues, 1990 as regards the secondary psychological problems in epilepsy patients, combined with side effects of antiepileptic drugs, may cause or heighten learning problems.

There was also a significant increase in depression in male GME patients.

This means that there is significant increase in depression in male GME patients. This is beside the role of GME itself in increase depression .

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the many studies employing the Minnesota Personality Inventory (MMPI) have indicated that depression scores are raised in people with epilepsy, with special reference to those with psychomotor/ temporal lobe epilepsy.

There was also a significant increase in psychoticism in male TLE patients.

This means that there is significant increase in psychoticism in male TLE patients. This is beside the role of TLE itself in increasing psychoticism.

The finding of this study goes with results reported by Christine and his colleagues, 1988 as regards the aggressive, antisocial behaviour disorders have been associated with boys, whereas neurotic disturbances are found more frequently in girls.

In our study there was a significant increase in Neuroticism in female GME patients.

This means that there is significant increase in neuroticism in female GME patients. This is beside the role of GME itself in increasing neuroticism.

Our finding goes also with the results reported by Christine and his colleagues, 1988 as regards the aggressive, antisocial behaviour disorders have been associated with boys, whereas neurotic disturbances are found more frequently in girls.

2) Family Size:

In our study there were significant decreases in attention and response rate in GME patients with big family size (more than 4 members).

This means that big family size have a great role in decrease attention and response rate in GME patients.

This is beside the role of GME itself in decreasing attention and response rate.

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the children with epilepsy have more social problems and participate in fewer social activities than their peers. Increased parental supervision and decreased family leisure activities have been hypothesized to contribute of these social problems.

There was significant increase in impulsivity in TLE patients with big family size.

This means that big family size have a great role in increase impulsivity in TLE patients. This is beside role the of TLE itself in increasing impulsivity.

This finding goes with the results reported by Henriksen and his colleagues, 1990 as regards the secondary psychological problems in epilepsy patients, combined with side effects of antiepileptic drugs, may cause or heighten learning problems.

There was also significant increase in depression in GME patients with big family size.

This means that big family size have a great role in decrease depression in TLE patients. This is beside role the of TLE itself in decreasing depression.

The finding of this study goes with results reported by Runge and his colleagues, 1997 as regards the benefit of monitoring anticonvulsant levels in epilepsy is still controversial. When therapeutic drug monitoring (TDM) of anticonvulsants was introduced, it appeared to optimise anticonvulsant therapy.

There was also significant increase in introversion trait in TLE patients with big family size.

This means that big family size have a great role in increase introversion in TLE patients. This is beside role the of TLE itself in increasing introversion.

This goes with results reported by Mary 1988, as regards the A number of variables have been suggested with regard to the pathogenesis of the depression, and several predisposing or provoking factors include genetic endowment, patients fears, social stigmatization, adverse life events and past history of depressive illness.

This means that all changes occur only in big family size whether in GME or TLE patients. Which means that the big family size have a great role in decrease attention and response rate, increase impulsivity, increase depression and increase introversion.

3) Sib-order:

In our study there was significant increase in impulsivity in TLE patients with 1st two sib-order.

This means that the ordering of the 1st two suborder have a great role in increase impulsivity in TLE patients. This beside the role of TLE itself in increasing impulsivity.

The finding of this study goes with results reported by Graaf and his colleagues, 1997 as regards the besides determining the general prevalence of psycholpathology in a population of patients with seizures we will present outcome regarding the relationship between pseudoseizures and DSM-Axis-Idisorders, and between epilepsy and personlity disorders (DSM-Axis-II).

There was also a significant increase in depression in sib-order (more than two) GME patients with.

This means that the ordering of suborder more than two have a great role in increase depression in GME patients. This is beside the role of GME itself in increasing depression.

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the many studies employing the Minnesota Personality Inventory (MMPI) have indicated that depression scores are raised in people with epilepsy, with special reference to those with psychomotor/ temporal lobe epilepsy.

There was also a significant increase in psychoticism in first sib-order GME patients.

This means that the ordering of the 1ˢᵗ two suborder have a great role in increase psychoticism in TLE patients. This beside the role of TLE itself in increasing psychoticism.

These goes with results reported by Schmitz and his colleagues, 1995 as regards the both major depression and schizophrenia like psychosis were related to temporal lobe epilepsy. In addition, schizophrenic patients were characterized by numerous biological and social variables, which might be of etiological relevance. Depressive patients

were characterized by older age, which might reflect the higher incidence of major depression in the elderly.

There was also a significant increase in depression in GME patients without high family income.

This means that high family income do not contribute in increasing depression in GME patients and that depression in GME patients is concerned only to the disease.

This goes with results reported by Magaudda, and his colleagues, 1997 as regards the There is a high frequency of depression amongst epileptic patients (29% of our cases) and the type of depression was dysthymic in most cases, appearing as a state of "demoralization" secondary to a chronic invalidating illness.

This goes also with results reported by Betts 1982, as regards that characteristically, the alteration of mood lasts longer than an aura or postictal automatism, and it can have dangerous consequences, as evidenced by Betts (1982) who reported a patient who cut his throat during an ictal depressive state.

4) Rooms:

In our study there were significant decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in GME patients who live in up to two rooms.

This means that housing less than 3 rooms has a great role in decrease sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in GME patients. This beside the role of GME itself in decreasing that.

The finding of this study goes with results reported by Long and his colleagues, 1997 as regards the IQ deterioration might not be linked causaly to epilepsy, but to the psychosocial consequences of the disease .

There were also significant increases in attention and response rate in GME patients who live in low rooms number (up to two rooms).

This means that GME patients who live in low rooms number (up to two rooms) has a great role in decreasing attention and response rate. This is beside the role of GME itself in decrease that.

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the many studies employing the Minnesota Personality Inventory (MMPI) have

indicated that depression scores are raised in people with epilepsy, with special reference to those with psychomotor/ temporal lobe epilepsy.

5) Wanted child:

In our study there was significant increase in depression in GME patients whom were wanted child during pregnancy.

This means that wanted child during pregnancy has a great role in increase depression in GME patients. This is beside the role of GME itself in increasing depression.

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the depression is more common is people with epilepsy (PWE) as compared with control groups. Depression is associated with psychosocial complications of epilepsy (increased life events, poor adjustment to seizures, financial stress), but is not, by and large, correlated with epilepsy variables such as age of onset of epilepsy or seizure type or frequency. Antiepileptic drugs (AEDs) on the other hand have a marked effect on mood.

There was also significant increase in psychoticism in TLE patients whom were not wanted child during pregnancy.

This means that wanted child during pregnancy has a great role in increase psycholicism in TLE patients. This is beside the role of TLE itself in `increasing psychoticism.

This goes with results reported by Levav and his colleagues, 1997 as regards that greater complexity of familial aggregation of traits associated with seizure disorders than was evident from our preliminary results.

6) Residence : (Whether Rural or Urban)

In our study there was significant increase in impulsivity in TLE patients in rural residence.

These finding means that rural residence has a great role in increasing impulsivity in TLE patients. This is beside the role of TLE itself in increasing impulsivity.

This goes with the results reported by Henriksen and his colleagues, 1990 as regards the secondary psychological problems in epilepsy patients, combined with side effects of antiepileptic drugs, may cause or heighten learning problems.

4) Parental Separation: *(Whether present or not)*

In our study there was significant increase in depression in GME patients with parental separation.

This mean that parental separation has a great role in increase depression. This is beside the role of GME itself in increasing depression

The finding of this study goes with results reported by Jolanta and his colleagues, 1988 as regards the reports of a marked deterioration of IQ in over 15% of residential school patients.

8) Family Income: *(Whether high or low)*

a) High family income:

In our study there was significant increase in depression in GME patients with high family income.

This means that patients with high family income has a great role in increase depression in GME patients. This beside the role of GME itself in increasing depression.

This goes with results reported by Mary 1988 as regards that with regards to depressive symptoms, several studies using standardized rating scales have found the depression

scores to range from moderate to severe and to compare favorably to studies on depressed patients without epilepsy.

9) Educated Parents:

In our study there were significant decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients with non educated parents.

This finding means that non educated parents have a role in decreasing sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients. This is beside the role of TLE has itself in that.

The finding of this study goes with results reported by Fowler and his colleagues, 1955 as regards the most children with epilepsy do not have learning disability but a small subgroup of children shows decreasing I.Q. because mental age does not increase at the same rate as chronological age. Our previous extensive studies have failed to show any relationship between I.Q. decrease and age of onset of epilepsy, total number of seizures, antieplieptic medicaion, sex, or initial I.Q.

There were also decreases in attention and response rate in GME patients with educated parents.

This means that educated parents has no role in decrease attention and response rate, and only GME has a great role in decreasing that.

The finding of this study goes with results reported by Euan and his colleagues, 1988 as regards the some parents wish to keep their child's epilepsy a secret. This poses a potential longer to the child and needless problems for school staff if they have not been told and prepared for seizures to occur. Doctors must encourage the parents to share the problem with the school.

There was also significant increase depression in GME patients in patients with non educated parents.

This finding mean that non educated parents have a great role in increase depression. This is beside the role of GME itself in increasing depression.

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the many studies employing the Minnesota Personality Inventory (MMPI) have indicated that depression scores are raised in people with epilepsy, with special reference to those with psychomotor/ temporal lobe epilepsy.

II) Illness Profile:

N.B :

i- **_Generally means_** _the comparison between GME, TLE and control groups concerning the illness profile_

ii- **_In details means_** _the relation between different aspects of illness profile and performance on different psychometric tests_

1) Onset of Seizures :

i- **_Generally_**

In our study there was no significant difference between GME and TLE groups as regards age of onset (whether below 2 years, between 2 to 4 years or as above 4 years) or as regards type of seizure (whether abrupt or gradual).

ii- *In details*

In our study there were significant decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and Learning ability in TLE patients with age of onset above 4 years and in TLE patients whether the type of onset (abrupt or gradual).

This means that when onset of seizure is at age above 4 years, there is no role of that onset in decreasing. sustained attention, persistency, psychomotor speed, visuomotor coordination and Learning ability. And that TLE only has a great role in decreasing (that).

The finding of this study goes with results reported by Chan and his colleagues, 1997 as regards the epilepsy onset is most frequent between age 1-5 years. Patients without secondarily generalised seizures have better long-term prognoses.

There were also decreases in either or all of immediate memory, auditory attention, short memory and internal visual scanning in GME patients with abrupt onset of disease.

This finding means that abrupt onset has a great role in decrease either or all of immediate memory,

auditory attention, short memory and internal visual scanning in GME patients.

The finding of this study goes with results reported by Kim and his colleagues, 1997 as regards the prognosis of patients with Mesial Temporal Sclerosis (MTS) is not poor. The most important factor related with the prognosis was the age of seizure onset.

There was also significant decreases in attention and response rate in GME patients with gradual onset of disease.

This finding means that gradual onset of disease has a great role in decrease attention & response rate in GME patients.

The finding of this study goes with results reported by Mercade and his colleagues, 1997 as regards the early onset of disease and long time of duration of epilepsy were the more important factors involved in persistence of seizures.

Our finding goes also with results reported by Pang and his colleagues, 1997 as regards the international League Against Epilepsy (ILAE) recognizes several Idiopathic Generalised Epilepsies (IGE) with age-related onset.

There was also significant increase in impulsivity in GME patients with gradual onset of the disease.

This finding means that gradual onset of disease has a great role in increase impulsivity in GME

patients. This is beside the role of GME itself in increasing impulsivity that.

The finding of this study goes with results reported by Weber and his colleagues as regards 1997 the number of children who present for evaluation of new onset seizures and may or may not go on to develop epilepsy demonstrate mild cognitive impairment. In addition, a large number of these children have significant behavioural problems. Earlier attention to such difficulties may be warranted.

There was also significant increase in anxiety in TLE patients with gradual onset of disease.

This finding means that gradual onset of disease has a great role in increase anxiety in TLE patients

This is beside the role of TLE itself in increasing anxiety .

The finding of this study goes with results reported by Milewska and his colleagues, 1997 as regards the more frequency incidence of epileptic seizures and epileptic form discharges in children under 15 can be associated with immaturity of developing brain which may account for the increased seizure susceptibility.

There was also significant increase in neuroticism trait in GME patients with gradual onset of disease.

This finding means that gradual onset of the disease has a great role in increase neuroticism in GME patients. This is beside the role of GME itself in increase that.

The finding of this study goes with results reported by Quattrini and his colleagues, 1997 as regards the drug resistant patients showed positive correlation with mental retardation, complex partial seizures, pathological CT and/or MRI findings, very early age of epilepsy onset, higher frequency of status epilepticus.

There was also a significant increase in Introversion trait in TLE patients with onset of disease above 4 years.

This finding means that onset of epilepsy of more than 4 years of age has a great role in increase introversion in TLE patients .This is beside the role of TLE itself in increasing introversion .

Our finding goes with results reported by Joan and his colleagues, 1995 as regards the Children with epilepsy have more social problems and participate in fewer social activities than their peers. Increased parental supervision and decreased family leisure activities have been hypothesized to contribute of these social problems.

Reduced child activity was related to seizure frequency (chi square = 10.5, P < 0.01) but to parent supervision of family leisure activity.

There was also a significant increase in lie scale in TLE patients with abrupt onset of disease.

This finding means that abrupt onset of disease has a great role in increase lie scale in TLE patients . This is beside the role of TLE itself in increasing lie scale.

The finding of this study goes with results reported by Fitzpatrick and his colleagues, 1997 as regards the differences between both epileptic groups and between the childhood onset. The effects of developing epilepsy in childhood on the subsequent development of personality is discussed.

There was also a significant increase in lie scale in GME patients with age of onset between two and four years.

This finding means that when fits begins at the age of onset between 2 to 4 years has a great role in increase lie scale in TLE patients .This is beside the role of TLE itself in increasing lie scale.

Our finding goes with results reported by Christine and his colleagues, 1988 as regards the positive associations

between seizure onset and frequency and behaviour disturbance have been reported in some studies.

2) Frequency of seizures :

i-   Generally

a - Current seizure frequency:

In   our study there was a significant difference between GME and TLE groups concerning current seizure frequency (< 0.001).

Showing that GME is more than TLE :

- When the frequent seizure is every month

- And when there are non frequent seizures.

But it shows that TLE is more than GME:

- When the frequent seizure are every day

- And when the frequent seizure are every week

The finding of this study goes with results reported by Pierre and his colleagues, 1988 as regards the A long duration of epilepsy impairs memory functioning. Ten years appears to be a cruciallength. In fact, a long duration generally means seizures repeated during many years, and a prolonged therapy. So neither the duration nor the frequency is an independent variable.

b - Past seizure frequency:

In our study there was a significant difference between GME and TLE groups concerning past seizure frequency (< 0.001).

Showing that GME is more than TLE:

- *When the past seizure frequency was every week*
- *And when the past seizure frequency was every month*

The finding of this study goes with results reported by Cakmak and his colleagues, 1997 as regards the frequency of secondarily generalised seizures (2nd GS), the age of onset and duration of illness have been known to be related with prognosis of temporal lobe epilepsy (TLE). The clinical characteristics and responses to AEDs therapy were evaluated in patients with TLE and rare 2nd GS, by using age matched TLE patients with frequent 2nd GS. Seizures occurring in late phase have high risk of recurrence. This may be due to different mechanisms of seizures in acute and late phases, and this result may help to determine a therapeutic strategy.

There was also a significant increase in depression in TLE patients with history of current seizure frequency of frequent seizures every week.

This means that onset of current seizure frequency of frequent seizures every week has a great role in

increase depression in TLE patients .This beside the role of TLE itself in increasing depression.

The finding of this study goes with results reported by Boglium and his colleagues, 1997 as regards the depression is more common is people with epilepsy (PWE) as compared with control groups. Depression is associated with psychosocial complications of epilepsy (increased life events, poor adjustment to seizures, financial stress), but is not, by and large correlated with epilepsy variables such as age of onset of epilepsy or seizure type or frequency.

There was also significant an increase in introversion in TLE patients with history of current seizure frequency of frequent seizures every day.

This means that onset of seizures every day has a great role in increase introversion in TLE patients .This beside the effect of TLE itself in increasing introversion.

The finding of this study goes with results reported by Uhlmann and his colleagues, 1997 as regards the most epielptic patients are able to control physiological parameters by intensive training and, as a result, improve clinically. Nevertheless, behavior therapy seem to have some influence on the amount of seizure reduction.

ii- In details

a) Current seizure frequency: (whether frequent per day frequent per week, frequent per month on none).

There was no significant difference between GME and TLE concerning the relation between different aspects of current seizure frequency and performance on different psychometric tests

b) Past seizure frequency: (whether frequent per day frequent per week or frequent per month).

There were significant decreases in attention and response rate in TLE patients with history of past seizure frequency of frequent seizures every week.

This means that past seizure frequency of frequent seizures every

week has a great role in decrease attention and response rate in TLE patients .This is beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Milewska and his colleagues, 1997 as regards the more frequency incidence of epileptic seizures and eppileptiform discharges in children under 15 can be associated with immaturity of developing brain which may account for the increased seizure susceptibility.

Our finding goes also with results reported by Henriksen and his colleagues, 1990 as regards the frequency and type of seizures may be

determining factors that should, if necessary, be evaluated by long-term EEG monitoring, perferably during school performance or in conjunction with neuropsychological assessment

There were also significant decreases in attention and response rate in TLE patients with past seizure frequency of frequent seizures every day.

This means that past seizure frequency of frequent seizures every day has a great role in increasing attention and response rate in TLE patients .This is beside the role of TLE itself in increasing that.

The finding of this study goes with results reported by Uhlmann and his colleagues, 1997 as regards the most epileptic patients are able to control physiological parameters by intensive training and, as a result, improve clinically. Nevertheless, behavior therapy seem to have some influence on the amount of seizure reduction.

Our finding goes also with results reported by Pierre and his colleagues, 1988 as regards the decreased performances are correlated with increasing discharge rates, or length of the discharges, and generalized discharges are worse than focal discharges.

There was also a significant increase in anxiety in TLE patients with history of past seizure frequency of frequent seizures every month.

This means that past seizure frequency of frequent seizures every month has a role in increase anxiety in TLE patients .This is beside the role of TLE itself in increasing anxiety.

The finding of this study goes with results reported by Vermeulen and his colleagues, 1997 as regards the in children with recurrent seizure, subjective seizure severity measured with the Hague Seizure Severity was not different after 1 year of treatment.

Our finding goes also with results reported by Euan and his colleagues, 1988 as regards the about half the children with epilepsy have not had a fit in the past two years. Their epilepsy is quiescent as active epilepsy.

There was also a significant increase in depression in TLE patients with history of past seizure frequency of frequent seizures every day.

This means that past seizure frequency of frequent seizures every day has a great role in increase depression in TLE patients .This is beside the role of TLE itself in increasing depression.

The finding of this study goes with results reported by Boglium and his colleagues, 1997 as regards the series of psychiatric symptoms and reactions, only depression seems to predominate in idiopathic and cryptogenic epiulepsy and can be attributed to epilepsy per se.

Our finding goes also with results reported by Mary and his colleagues, 1988 as regards the depression is more common is people with epilepsy (PWE) as compared with control groups. Depression is associated with psychosocial complications of epilepsy (increased life events, poor adjustment to seizures, financial stress), but is not, by and large, correlated with epilepsy variables such as age of onset of epilepsy or seizure type or frequency. Antiepileptic drugs (AEDs) on the other hand have a marked effect on mood.

3) Duration of Illness:

i- Generally

In our study there was a significant difference between GME & TLE concerning duration of illness.,showing that GME is more than TLE:
- When the duration of illness is below (6 months)
- And when the duration of illness is between (6 months – 2 years)

But it shows also that TLE was more than GME:
- When the duration of illness was below (5 years)
- And when the duration of illness was between (2 years – 5 years)

The finding of this study goes with results reported by Badalian and his colleagues, 1997 as regards the clinical probation registered the fact that effectiveness of the given

combination in patients depends on types of seizures and the duration of the disease.

ii- *In details*

There were also decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients with duration of illness between two and four years.

This means that duration of illness between two and four years has a great role in decrease sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients This was beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Mercade and his colleagues, 1997 as regards the early onset of disease and long time of duration of epilepsy were the more important factors involved in persistence of seizures.

There was also a significant increase in introversion trait in TLE patients with duration of illness above five years.

This means that duration of illness of 5 years has a great role in increase introversion in TLE patients .This is beside the role of TLE itself in increasing introversion.

The finding of this study goes with results reported by Wolanczyk and his colleagues, 1997 as regards the patients

treated by many physicians in the history of illness were characterised by significantly lower optimism, lower self-esteem, lower value of achievement and lower perceived support (ANOVA). Those rated by physicians as non-compliant were less optimistic, had less certainty of school success and perceived less support than compliant subgroup.

There was also an increase in impulsivity in TLE patients with duration of illness between six months and two years.

This means that the duration of illness between of six months to two years have a great role in increase impulsivity in TLE patients. This is beside the role of TLE itself in increasing impulsivity.

The finding of this study goes with results reported by Salke and his colleagues, 1997 as regards the children and adolescents with severe epilepsy, the long-term hospitalisation may be inevitable. We asked if such a hospitalisation is justified in relation to the outcome.

There was also an increase in psychoticism trait in TLE patients with duration of illness below six months.

This means that duration of illness of less than 6 months has a great role in increase psychoticism in TLE patients .This is beside the role of TLE itself in increasing psychoticism.

The finding of this study goes with results reported by Czapinski and his colleagues, 1997 as regards the initially observed difference in drug effectiveness disappears in long-term therapy.

4) Longest seizure free period

i- Generally:

In our study there was a significant difference between GME & TLE groups concerning longest seizure free period showing that GME was more than TLE:

- When the longest seizure free period was between (six months and two years)

But it shows also that TLE was more than GME:

- When the longest seizure free period was less than 6 months.

ii- In details

In our study there were significant decrease in sustained attention, persistency, psychomotor speed, visumotor coordination and learning ability in TLE patients with longest seizure free period below six months up to two years.

This means that longest seizure free period up to 2 years has a great role in decreasing sustained attention, persistency, psychomotor speed, visumotor coordination and learning ability in TLE epileptic patients. This was beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Christine and his colleagues, 1988 as regards the long duration of seizure disorder has been negatively associated with IQ in three studies.

.

There were also significant decreases in either or all of immediate memory, auditory attention, short memory and internal visual scanning in GME patients with longest seizure free period below six months.

This means that shorter seizure free period up to 6 months has a great role in decrease either or all of immediate memory, auditory attention, short memory and internal visual scanning in GME patients.

This is beside the role of GME itself in decreasing that.

The finding of this study goes with results reported by Christine and his colleagues, 1988 as regards the increased rates of behaviour distrubance with early age of onset, there are conflicting reports concerning the role of duration of the seizure disorder.

There were also decreases in conceptualization, concrete thinking, and mental ability in GME patients with longest seizure free period below six months.

This means that shorter seizure free period up to 6 months has a great role in decreasing conceptualization, concrete thinking, and mental ability in GME patients. This was beside the role of GME itself in decreasing that.

Our finding goes with the results reported by Ignatowicz and his colleagues, 1995 as regards the significant clincal benefit was manifested in good control of epileptic seizures, especially generalized and partial complex seizures and epileptic status with abnormal EEG in 60% of patients.

There was also significant increase in impulsively in GME patients with longest seizure free period below six months.

This means that shorter seizure free period up to six months have a great role in increase impulsivity in GME. This was beside the role of GME itself in increasing impulsivity.

Our finding goes also with results reported by Christine and his colleagues, 1988 as regards the long duration of seizure disorder has been negatively associated with IQ in three studies.

There was also significant increase in introversion trait in TLE patients with longest seizure free period whether below six months or between six months up to two years.

This means that longest seizure free period up to two years have a great role in increase introversion in TLE patients . This is beside the role of TLE itself in increasing impulsivity.

Our finding goes also with the results reported by Coline and his colleagues, 1988 as regards the based on the idea that paroxysmal disorders of behaviour arise form paroxysmal discharges of subcortical structures which were not necessarily recorded in the EEG from the surface of the cortex.

5) Status Epilepticus history:

i-   Generally

In our study there was no significant difference between GME and TLE groups as regards Status Epilepticus history.

Our finding goes with the results reported by Ignatowicz and his colleagues, 1995 as regards the significant clinical benefit was manifested in good control of epileptic seizures, especially generalized and partial complex seizures and epileptic status with abnormal EEG in 60% of patients.

ii- *In details*

In our study there were significant decreases in sustained attention persistencey, psychomotor speed, visumotor coordination and learning ability in TLE patients without status epilepticus history.

This means that status epilepticus has no role in decreasing sustained attention, persistencey, psychomotor speed, visumotor coordination and learning ability in TLE patients and that TLE only had a great role in decreasing that.

The finding of this study goes with results reported by Misirli and his colleagues, 1997 as regards the status epilepticus (SE) is defined as prolonged or recurrent seizure which affect the consciousness or not, and persist for more that 30 minutes with no recovery period between seizures. It is important to recognise the aetiological factor to pervent the recurrence.

And goes also with results reported by Jolanta and his colleagues, 1988 as regards the reports of a marked deterioration of IQ in over 15% of residential school patients and in patients with a history of status epilepticus or a considerable lifetime number of tonic-clonic seizures.

There was also significant decreases in attention and response rate in GME patients without history of status epilepticus .

This means that status epilepticus has no role in decreasing attention and response rate in GME patients and that GME only was responsible for decreasing attention and response rate in epileptic patients.

The finding of this study goes with results reported by Weissenrieder and his colleagues, 1997 as regards the non-convulsive status epilepticus (NCSE) manifests in the majority of patients as confusional state with disturbed consciousness and postictal amnesia. There have however been reports of patients with NCSE without any alteration of consciousness. The ictal process remained confined to the right frontal are without propagation.

There was also significant increase in depression in TLE patients with history of status epilepticus history.

This means that status epilepticus has a great role in increase depression in epileptic patients. This was beside the role of TLE itself in increasing depression .

The finding of this study goes with results reported by Well, 1975; and Lim and his colleagues 1986, as regards the non-convulsive status epilepticus (NCSE) manifests in the majority of patients as confusional state with disturbed consciousness and postictal amnesia. There have however

been reports of patients with NCSE without any alteration of consciousness. The ictal process remained confined to the right frontal are without propagation. Prolonged depressive mood swing have been recorded during status epilepticus.

The finding of this study goes with results reported by Well, 1975; and Lim and his colleagues, 1986 as regards the prolonged depressive mood swing have been recorded during status epilepticus.

There was also significant increase in psychotrcism trait in TLE patients without status epilepticus history.

This means that status Epilepticus has no role in increase psychoticism in TLE patients and only TLE have a great role in increase psychoticism in epileptic patients.

The finding of this study goes with results reported by Dericioglu and his colleagues, 1997 as regards the psychogenic seizures are diagnosed in 13-28% of patients referred to Epilepsy Centers. The attacks are recorded during long term video EEG time consuming.

There was also significant increase in introversion trait in TLE pat0ients with history of status epilepticus.

This means that status epilepticus has no role in increase introversion in TLE patients and only TLE has

a great role in increase introversion in epileptic children.   .

The finding of this study goes with results reported by Evan and his colleagues, 1988 as regards the absence of studies that have directly addressed the question as to whether depression occurs more frequently in people with epilepsy compared to other conditions, but conclusions may be drawn indirectly from the results of investigations using standardizes rating scales.

Uncoordinated behaviour typical cases can be found despite lack of characteristic EEG changes.

6) Positive family history *(whether with or without it.):*

i- Generally

In our study there was, no significant difference between GME and TLE groups as regards positive family history.

Our finding goes also with the results reported by Pal and his colleagues, 1997 as regards the strong independent association of proxy infection markers with epilepsy suggests a role for infective agents through a mechanism other than febrile convulsions. A more specific study is required to clarify the important component (s) of the proxy index.

ii- *In details*

In our study there were also significant decreases in sustained attention, persistency, psychomotor speed, visvomotor coordination and learning ability in TLE patients without positive family history.

This means that positive family history has no role in decreasing sustained attention, persistency, psychomotor speed, visvomotor coordination and learning ability and only TLE have a great role in decreasing that .

The finding of this study goes with results reported by Murakami and his colleagues, 1997 as regards the cases with mesial temporal lobe epilepsy showed a high incidence of prolonged convulsions or febrile convulsion. However, the existence of prolonged convulsion or the severity of febril convulsions were not related to the later clinical course of mesial temporal lobe epilepsy.

There was also significant increase in neasotrcism trait in GME patients without positive family history.

This means that positive the family history have no role in increase neuroticism in epileptic patients and only GME have a great role in increase neuroticism in that.

The finding of this study goes with results reported by Scheffer and his colleagues, 1997 as regards the epilepsy are

genetically related and inherited in a polygenic fashion. There are other common, but less well recognised, form of generalised epilepsy that also have an inherited basis.

7) Positive past history : *(whether with or without it).*

i- Generally

In our study there was, no significant difference between GME and TLE groups as regards positive past history.

The finding of this study goes with results reported by Awaya and his colleagues, 1997 as regards the other characteristics were first seizure episode, normal past history and mental deterioration without motor deficits. These patients were similar to those reported by Brett (1967), but seizure frequency and sequelae were much more severe in our group. The aetiology is uncertain but might be viral.

ii- *In details*

In our study there were significant decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients with positive past history.

This means that the positive past history has a great role in decreasing sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE epileptic patients. This beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Zupanc and his colleagues, 1997 as regards the morbidity in our Epilepsy Monitoring Unit was low. There was no mortality. The riskd of status epilepticus are small but significant.

There were also decreases in conceptualization, concrete thinking, and general mental ability in GME patients with positive past history.

This means that the positive past history has a great role in decreasing conceptualization, concrete thinking, and general mental ability epileptic in GME patients. This beside the role of GME itself in decreasing that.

The finding of this study goes with results reported by Tourniaire and his colleagues, 1997 as regards the most deaths were linked to epilepsy as traumatic, sudden

unexplained deaths or status epilepticus. There was no influence of six, aetiology, age on immediate cause of death.

There was also significant decreases in a Hention and response rate in GME patients without positive past history.

This means that the positive past history has no role in decrease attention and response rate in epileptic patients GME only has a great role in decrease attention and response rate in epileptic patients.

The finding of this study goes with results reported by Callenbach and his colleagues, 1997 as regards the genetic factors played a role in about 10% children with epilepsy. The genetic influence is stronger in generalised epilepsy than in partial epilepsy.

There was also significant increase in depression in TLE patients with positive past history.

This means that the positive past history has a great role in increase depression in TLE patients. This beside the role of TLE in increase depression.

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the with regards to depressive symptoms, several studies using standardized rating scales have found the depression scores to range from

moderate to severe and to compare favourably to studies on depressed patients without epilepsy.

There was also significant increase in introversion trait in TLE patients without positive past history.

This means that the positive past family history has no role in increase introversion in TLE patients and TLE itself has a great role in increase introversion in epileptic patients.

The finding of this study goes with results reported by Gary Haflied and his colleagues, 1997 as regards the curiously, patients generalised SE. the reasons for this are not clear. Patients with partial seizures had fewer lesions which is out of line with their high mortality rate. Some of the difference noted between the present study and the reports of others can be accounted for by the lack of previous seizures in our patients population.

8) *Organic disease:*

i- *Generally*

In our study we found that, GME patients showed a tendency to organic diseases.

This goes with results reported by Trimble and his colleagues, 1995 as regards the difficulty of interpretation is the number of variables that may influence cognition, such as

polytherapy and seizure frequency, for which these studies have not been able to control. In this study, healthy volunteers were examined for the effect of VGB on cognitive function and mood to assess the effect of the drug in the absence of such contaminating factors.

ii- *In details*

In our study there were significant decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients without organic disease history.

This means that history of organic diseaseing have no role in decrease sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients and that only. TLE have a great role in decreasing that in epileptic patients.

The finding of this study goes with results reported by Ellenbag and his colleagues, 1984 as regards the organic actiology for the seizures or a seizure disorder complicated by further neurological problems would appear to be a poor prognostic indicator for intellectual.

There were also significant decreases in either or all of immediate memory, auditory attention, short memory and internal visual scanning in TLE patients without organic disease history.

This means that history of organic disease have no role in decrease either or all of immediate memory, auditory attention, short memory and internal visual scanning in TLE patients and that only TLE have a role in decreasing that in epileptic patients.

The finding of this study goes with results reported by Trimble and his colleagues, 1995 as regards the prolonged and frequently repeated seizures are typically associated with more severe effects on cognitive functioning, particularly if epilepsy is symptomatic i.e., secondary to a demonstrable brain lesion.

There were also decreases in attention and response rate in GME patients with organic disease history.

This means that history of organic disease have a great role in decrease attention and response rate in GME patients. This is beside the role of GME in decreasing attention and response rate.

The finding of this study goes with results reported by Quattrini and his colleagues, 1997 as regards the try to understand the significance and mechanism of drug-resistance further parameter have been investigated, the most relevant of them are the duration of illness, the risk factor for epilepsy and the therapy.

There were also significant increase in impulsivity in TLE patients without organic disease history.

This means that history of organic disease have no role in increase impulsivity and only TLE have a great role in increasing impulsivity.

Our finding goes with the results reported by Christine and his colleagues, 1988 as regards the organic pathology has been reported for the majority of children exhibiting behaviour disorders and large numbers of children with behaviour disorders have been found in brain-damaged populations.

9) Pregnancy, Labour troubles and Neurotic traits history:

i- Generally

In our study there was no significant difference between GME and TLE groups as regards pregnancy, Labour troubles and Neurotic traits history.

ii- In details

a) Pregnancy trouble history:

In our study there were significant decreases in attention and response rate in TLE patients without pregnancy trouble history.

This means that history of pregnancy troubles have no role in decreasing attention or response rate in TLE patients and TLE only have a great role in decrease attention and response rate.

The finding of this study goes with results reported by Milewska and his colleagues, 1997 as regards the more frequency incidence of epileptic seizures and epileptiform discharges in children under 15 can be associated with immaturity of developing brain which may account for the increased seizure susceptibility.

There were also significant increase in impulsivity in TLE patients without pregnancy trouble history.

This means that history of pregnancy troubles have no role in increase impulsivity in epileptic patients and TLE only has a great role in increasing impulsivity.

The finding of this study goes with results reported by Abou – Khalil and his colleagues, 1997 as regards the history of Febril Convulsions (FC) preceding epilepsy is common, particularly in TLE. A genetic influence is demonstrated by the higher incidence of FHFC in patients with antecedent FC.

There were . also significant increase in lie scale in GME patients with pregnancy trouble history.

This means that pregnancy troubles have a great role in increase lie scale in GME patients. This is beside the role of GME in increasing lie scale.

The finding of this study goes with results reported by El-Radhi and his colleagues, 1997 as regards the known association between MC, recurrent FC and epilepsy may be due to a common facto of low body temperature at the onset of FC.

b) Labour Trouble History:

In our study there was significant increase in impulsively in TLE patients without labour troubles history.

This means that labour have no role in impulsivity in TLE patients and TLE only have a great role in increase impulsivity in epileptic patients.

Our finding goes also with the results reported by Ingrid and his colleagues, 1995 as regards the single-gene partial epilepsies have recently been recognized in which affected

individuals have homogeneous partial seizure semiology within syndromes such as autosomal dominant nocturnal rolandic epilepsy. We describe a signle-gene partial epilepsy syndrome with different electroclinical partial seizure semiology in different individuals.

c) Neurotic Traits:

In our study there were significant decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients with neurotic traits.

This means that neurotic traits has a great role in decrease sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients. This beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Selukov and his colleagues, 1997 as regards the course of the disease in that group of patient was characterised by steady increase of sympotoms and psychic deterioration with inertness, persistence, specific affective and mental disorders.

And goes also with the results reported by Schmitt and his colleagues, 1997 as regards the optimal therapy regimen for one of the commonest epilepsies of childhood a waits prospective evaluation.

There were also significant increase in impulsivity in TLE patients without neurotic traits.

This means that neurotic traits has no role in increase impulsivity in TLE patients and that only TLE have a role in increase impulsivity in epileptic patients.

Our finding goes with the results reported by Coline and his colleagues, 1988 as regards the based on the idea that paroxysmal disorders of behaviour arise form paroxysmal discharges of subcortical structures which were not necessarily recorded in the EEG from the surface of the cortex.

It was recognition that electrical discharges in the EEG correlated with observed seizure behaviour that lead to a taxonomy of epilepsy based on EEG waveforms. Indeed, the first international classification of epilepsy relied heavily on different EEG pictures for different diagnoses.

There were significant increase in depression in GME patients with neurotic traits.

This means that neurotic traits have a great role in increase depression in GME patients. This beside the role of GME in increasing depression in epileptic patients.

The finding of this study goes with results reported by Katharine and his colleagues, 1995 as regards the phenomenology of the depression does not, on the whole, seem linked with epilepsy variables. Anticonvulsants can however

affect the mental state, and the longer the duration of epilepsy the more severe the depression. The majority of studies implicated the left hemisphere with hints that temporal lobe/complex partial seizure patients are more vulnerable.

10) Peri-ictal Changes:

i- Generally

In our study there was no significant difference between GME and TLE groups as regards factors affecting ictus.

In the other hand, there was a significant difference between GME and TLE groups as regards the following :

1- patients with prodroma.

2- Patients with aura.

3- Patients with ictal changes.

4- Patients with post-ical changes.

The finding of this study goes with results reported by Betts and his colleagues, 1982 as regards the characteristically, the alteration of mood lasts longer than an aura or postictal automatism, and it can have dangerous consequences, as evidenced by him who reported a patient who cut his throat during an ictal depressive state.

ii- In details

a- Patients with presence of Prodroma:

In our study there was significant increase in psychoticism in TLE patients with presence of prodroma.

This means that the presence of prodoma in epileptic patients increases psychoticism in TLE patients. This beside the role of TLE in increase psychoticism.

This goes with results reported by Fitzpatrick and his colleagues, 1997 as regards that Much has been written about personality disorder and abnormal personality traits in patients with non epileptic seizures (NES). To date the majority of the research has identified personality disorders by means of clinical interview with the subject, or through the use of self-rating, forced choice questionnaires or inventories. It is well recognized that both these methods produce low levels of reliability.

Differences in personality traits between the groups were identified, as were rates of personality disorder.

There was also significant increase in Introversion in TLE patients with presence of prodroma.

This means that presence of prodroma has a great role in increase introversion in TLE patients. This beside the role of TLE itself in increase introversion.

The finding of this study goes with results reported by Betts and his colleagues, 1982 as regards the characteristically, the alteration of mood lasts longer than an aura or postictal automatism, and it can have dangerous consequences, as evidenced by him who reported a patient who cut his throat during an ictal depressive state.

b- Patients with presence of aura:

In our study there were significant decrease in sustained attention, persistency, psychomotor speed, visumotor coordination and learning ability in TLE patients with presence of aura.

This means that the presence of aura has a great role in decrease sustained attention, persistency, psychomotor speed, visumotor coordination and learning ability in TLE patients. This beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Manchanda and his colleagues, 1997 as regards the localising value of experiential auras and the lateralising value of viscerosensory auras are in keeping with the literature. The findings confirm a clinical impression that patients were experiential auras are more likely to have a psychiatric disorder.

There was also significant increase in introversion trait in TLE patients with presence of Aura.

This means that presence of aura has a great role in increase introversion in TLE patients. This beside the role of TLE itself in increasing introversion.

The finding of this study goes with results reported by Manchanda and his colleagues, 1997 as regards the localising value of experiential auras and the lateralising value of viscerosensory auras are in keeping with the literature. The findings confirm a clinical impression that patients were experiential auras are more likely to have a psychiatric disorder.

c- Patients with presence of Ictal changes:

In our study there were significant decrease in sustained attention, persistency, psychomotor speed, visumotor coordination and learning ability in TLE patients with presence of ictal changes.

This means that ictal change has a great role in decrease sustained attention, persistency, psychomotor speed, visumotor coordination and learning ability in

TLE patients. This beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Kim and his colleagues, 1997 as regards the gestural automatism contralateral dystonic CLD and tonic CLT were seen mainly in Mesial TLE (MTLE) and head version in Neocortical Temporal Lobe NTLE. These ictal features can be useful in differentiating MTLE from NTLE.

There was also significant increase in psychoticism in TLE patients with presence of ictal change.

This means that ictal changes has a great role in increase psychoticism in TLE patients. This beside the role of TLE itself in increasing psychoticism.

The finding of this study goes with results reported by Perucca and his colleagues, 1997 as regards the although most clinicians attempt to treat epilepsy on the basis of monotherapy, the need for polytherapy in fraction of the patient population makes drug interactions a significant issue. The problem and drug interactions also must be considered in the design and interpretation of clinical trials in which a new drug is added to pre existing treatment.

There was also significant increase in introversion in TLE patients with presence of ictal changes.

This means that ictal changes has a great role in increase introversion in TLE patients. This beside the role of TLE itself in increasing introversion.

The finding of this study goes with results reported by Abdulghani and his colleagues, 1997 as regards the children with abnorma EEG had significant deterioration of personality and behaviour adjustment. This was more apparent in patients with focal EEG dysrhythmia. Intellectual deterioration was not related to EEG abnormalities.

d) Patients with presence of Post ictal changes

(aphasia, dysphasia, paresis, restlessness, automotism and psychotic state) :

There were also significant decrease in sustained attention, persistency, psychomotor speed, visumotor coordination learning ability in TLE patients with presence of post ictal changes.

This means that post ictal changes has a great role in decrease sustained attention, persistency, psychomotor speed, visumotor coordination learning ability in TLE epileptic patients. This beside the role of TLE itself in decreasing that.

This finding goes with the results reported by Binnie and his colleagues, 1990 as regards the sub-clinical generalized

spike-wave discharges are often accompanied by transitory cognitive impairment, demonstrable by psychological testing during EEG recording.

There was significant increase in psycholicism in TLE patients with presence of post ictal changes.

This means that post ictal changes has a great role in increase psychoticism in TLE epileptic patients. This beside the role of TLE in increasing psychoticism in epileptic patients.

The finding of this study goes with results reported by Mares and his colleagues, 1997 as regards the postictal depression is a complex phenomenon generated by more than one inhibitory system.

There was also significant increase in Introversion in TLE patients with presence with presence of post ictal changes.

This means that post ictal changes has a great role in increase introversion in TLE patients. This beside the role of TLE in increasing introversion in epileptic patients.

The finding of this study goes with results reported by Mares and his colleagues, 1997 as regards the postictal depression is a complex phenomenon generated by more than one inhibitory system.0

e- ***Patients*** ***with*** ***presence*** ***of*** ***factors*** ***affecting*** ***ictus*** *(Alcohol, menses, emotional stress, fever, fatigue, photic stimulation, TV, photogenic epilepsy, nocturnal, seizure only, non-compliance and day seizure only):*

In our study there were decrease in attention and response rate in GME patients with presence one or more of factors affecting ictus.

This means that presence of factors affecting ictus has a great role in decrease attention and response rate in GME patients. This beside the role of GME itself in decreasing attention and response rate.

The finding of this study goes with results reported by Shewmon and his colleagues, 1997 as regards the cognitive improvement from epilepsy surgery may have more to do with elimination of interical disturbances than with cessation of seizures. It remains to be determined which children with frequent epileptiform discharges might benefit from surgical therapy.

There was also significant increase in neuroticism in GME patients with presence of factors affecting ictus.

This means that presence of factors affecting ictus has a great role in increase neuroticism in GME

336

patients. This beside the role of GME itself in increasing neuroticism. `

The finding of this study goes with results reported by Boglium and his colleagues, 1997 as regards the series of psychiatric symptoms an reactions, only depression seems to predominate in idiopathic and cryptogenic epiulepsy and can be attributed to epilepsy per se.

There was also significant increase in introversion in TLE patients with presence of factors affecting ictus.

This means that presence of factors affecting ictus has a great role in increase introversion in TLE patients. This beside the role of TLE itself in increasing introversion.

The finding of this study goes with results reported by Barclay and his colleagues, 1997 as regards the temporal lobe seizures with sympotoms including disorientation, déjà vu, unpleasant odours, balance disturbances, dizziness, and balkouts.

11) Causes:

i- Generally

In our study there was a significant difference between GME and TLE groups as regards cerebral, systemic and iatrogenic aetiologies.

The finding of this study goes with results reported by Hoare and his colleagues, 1997 as regards the cross-sectional design of the study did not make it possible to draw any definite conclusions about the causal or temporal relationship between low self-esteem and behavioural distrurbance. Once again, the potential value of prospective studies into the psychosocial adjustment of children with chronic illness is highlighted

Our finding goes also with the results reported by Graaf and his colleagues, 1997 as regards the Besides determining the general prevalence of psycholpathology in a population of patients with seizures we will present outcome regarding the relationship between pseudoseizures and DSM-Axis-Idisorders, and between epilepsy and personlity disorders (DSM-Axis-II).

GME patients shows tendency to cerebral and iatrogenic a etiology TLE patients shows tendency to systemic a etiology.

The finding of this study goes with results reported by Jolanta and his colleagues, 1988 as regards the patients without gross cerebral damage have the evidence of interhemispheric differrences for verbal and non-verbal material is less clear.

ii- In details

a) Cerebral causes *(congenital anomalies, birth injuries , traumatic, vascular, infection, neoplasm and demyelinating diseases):*

In our study there was a significant increase in anxiety in TLE patients with presence of cerebral causes.

This means that the presence of cerebral causes has a great role in increase anxiety in TLE patients. This beside the role of TLE itself in increasing anxiety.

The finding of this study goes with results reported by Besana and his colleagues, 1997 as regards their patients perinatal factors seam, still now, the main aetiological factor in drug resistan epilepsies; genetic causes as cerebral malformations and phakomatoses are also significant ethiopathogenetical factors.

Our finding goes also with the results reported by Bölling and his colleagues, 1995 as regards the according to this hypothesis, early isolated cerebral lesions of the LH with strongly influence cognitive functions of the RH, whereas isolated lesions of the RH do not influence LH functions to the same extent due to a lesser reorganization capacity of the LH.

And also goes with the results reported by Maria and his colleagues, 1995 as regards the partial seizures account for

339

40% of childhood seizures. Prognosis depends on association with definite cerebral lesions.

b) Systemic Causes *(Fever, toxic, hypoglycemia, CA deficiency, renal factors, anemia and anoxia):*

In our study there was a significant increase in introversion in TLE patients with presence of systemic causes.

This means that the presence of systemic causes has a great role causes in increase introversion in TLE patients. This beside the role of TLE itself in increasing introversion.

The finding of this study goes with results reported by Meador and his colleagues, 1997 as regards the alterations in cognitive function in patients with epilepsy may be due to the underlying disease process and to seizures themselves.

Our finding goes also with the results reported by Van Bogaert and his colleagues, 1997 as regards the patients with BCECS and unilateral focus do not show an asymmetrical distribution of cerebral glucose metabolism on PET. This suggests that PeT with FDG may help to differentiate the idiopathic cases from the symptomatic cases in children with partial epilepsy.

12) Serum Level:

In our study there was no significant difference between GME and TLE groups as regards high serum level.

The finding of this study goes with results reported by Mary and his colleagues, 1988 as regards the many studies employing the Minnesota Personality Inventory (MMPI) have indicated that depression scores are raised in people with epilepsy, with special reference to those with psychomotor/ temporal lobe epilepsy.

13) Psychiatric Morbidity:

In our study there was no significant difference between GME and TLE groups as regards psychiatric morbidity.

The finding of this study goes with results reported by Jinggang and his colleagues, 1995 as regards the implications

for the choice of drug in the management of epilepsy and also for the reported claims of a psychotropic effect of CBZ.

Our finding goes also with the results reported by Miller and his colleagues, 1995 as regards the results argue that the hippocampal region plays a more important role that the entorhinal cortex in human memory.

In our study TLE patients shows tendency to conduct disorder, sex disorder and anxiety disorder. GME patients shows tendency to conversion disorder.

The finding of this study goes with results reported by John and his colleagues, 1995 as regards the anxiety and depression are the most common psychiatric conditions associated with epilepsy. Understanding the development & maintenance of these symptoms requires investigate of interrelations between biologicial and psycholocial factors.

Our finding goes also with the results reported by Hoare and his colleagues, 1997 as regards the cross-sectional design of the study did not make it possible to draw any definite conclusions about the causal or temporal relationship between low self-esteem and behavioural distrurbance. Once again, the potential value of prospective studies into the psychosocial adjustment of children with chronic illness is highlighted.

And also goes with the results reported by Toone and his colleagues, 1997 as regards the number of previous studies

have suggested on association between left-sided temporal lobe epilepsy and schizophrenia.

III) Therapeutic issues.

N.B :

i- <u>Generally means</u> the comparison between GME, TLE and control groups concerning the Therapeutic issues

ii- <u>In details means</u> the relation between different aspects of Therapeutic issues and performance on the different psychometric tests

i- <u>Generally</u>

1- According to different types of therapy

In our study there was significant difference between GME and TLE groups as regards the type of therapy whether mono or polytherapy

It shows that there was a tendency of GME patients to take monotherapy. In the other hand TLE was used more frequently polytherapy

Our finding goes with the results reported by Thompson and his colleagues, 1982 as regards the study of the effects reduction of plytherapy or substituting one of the drugs with CBZ, the performance on the visual scanning task improved in patients after changes in therapy, but the Stroop test showed no significant changes.

And goes also with the results reported by Jinggang and his colleagues, 1995 as regards the comparison of the two drug groups on word recall test again showed poorer performcance of the PHT group. In the list-learning task, the CBZ group showed a trend to learn more rapidly than did the PHT group during the first four trials.No significant difference, were noted between the groups in the decision-making task or the tracking task.

2) According to side effects of AED's in general:

In our study there was no significant difference between GME and TLE groups as regards the side effects of the used AED's.

The finding of this study goes with results reported by Decker and his colleagues, 1997 as regards the using the "wait and see" approach may lead to underreporting. 2) Routine laboratory monitoring of epilepsy is not efficient, 3) conclusions (1) and (2) are supported by the literature. In particular sedation and cognitive impariments are said to be underreported. These finding have repercussions for patients

3) According to side effects of each AED Drug :

In our study GME patients were suffered significantly from Tegretol (Carbamazepine) side effects more than TLE patients.

When there were no significant differences between GME and TLE groups concerning the side effects of other antiepileptic drugs (Depakine, Rivotril and Epanutine)

The finding of this study goes with results reported by Kaufman and his colleagues, 1997 as regards the hepatotoxicity from anticonvulsants, especially cabamazepine and valproic acid, is a well known complication monitored by epileptologists. With the advent of these agents in the

345

treatment of bipolar affective disorders, it is cirtical that psychiatrists be aware of the need for liver function monitoring.

The finding of this study goes with results reported by Decker and his colleagues, 1997 as regards the particular sedation and cognitive impariments are said to be underreported. These finding have repercussions for patients management.

And goes also with the results reported by Haskem and his colleagues, 1997 as regards the side effects of sodium valproate could be attributed to the changes of the neurotransmitters.

And goes also with the results reported by Geladze and his colleagues, 1997 as regards the carbamazepine (CBZ) medication needs detailed longitudinal EEG control for right management of treatment.

And goes also with the results reported by Grace and his colleagues, 1997 as regards the clobazam does not appear to have adverse cognitive effects compared to standard monotherapy.

And goes also with the results reported by Schmitt and his colleagues, 1997 as regards the optimal therapy regimen for one of the commonest epilepsies of childhood a waits prospective evaluation.

And goes also with the results reported by Reetta and his colleagues, 1995 as regards the VGB appears to be an effective to be an effective and safe antiepileptic drug as primary monotherapy, with fewer cognitive side effects than CBZ.

And goes also with the results reported by Matthews and his colleagues, 1975 as regards the patients with toxic levels perform worse than patients with non-toxic levels.

And goes also with the results reported by Dodrill and his colleagues, 1977 as regards the sodium valproate and carbamazepine are certaily less toxic.

And goes also with the results reported by Piattella and his colleagues, 1995 as regards the higher incidence of significant side effects with carbamazepine versus vigabatin (VGB).

And goes also with the results reported by Ossetw and his colleagues, 1968 as regards the patients has a higher incidence of toxic serum drug levels, earlier onset of seizures and poorer control of seizures.

And goes also with the results reported by Gus and his colleagues, 1995 as regards the twentry-four percent of patients were clinically anxious. 10% were clinically depressed, and approximately one fifth left stigmatized by their epilepsy. Many side effects of antiepileptic drug treatment

were reported, with CNS side effects being the most common. Rates of employment and marriage were low as compared with the U.K. national average.

The finding of this study goes with results reported by Rancci and his colleagues, 1995 as regards the immune response did not correlate with clincial evolution; moreover, difference were not observed between idiopathic and symptomatic epileptic children.

4) Amount of drug intake (Monotherapy):

In our study there was no significant difference between GME and TLE groups as regards amount Of Epanutin intake.

However, there was significant difference between GME and TLE shows that GME patients was used Tegretol more than TLE and that TLE patients was used Depakine and Rivotril more than GME.

The finding of this study goes with results reported by Brodie and his colleagues, 1995 as regards the preliminary report comparing the cognitive effects of OCBZ and phenytoin in mewly diagnosed epilepsy showed no difference between the drug. OCBZ is a promising new antiepileptic drug that may have a bening effect of cognition at therapeutic dosage.

And goes also with the results reported by Trimble and his colleagues, 1990 as regards the however, there appears to be a dissociation between AEDs that affect higher cognitive function, e.g. phenytoin, and those mainly affecting motor function, e.g. carbamazepine, which appears to increase speed of performance.

And goes also with the results reported by Dennis and his colleagues, 1988 as regards the combined with a study design which encouraged physicians to lower the dose when unacceptable toxicity intervened resulted in a very different subject population

5) According amount of drug intake (polytherapy):

In our study there were no significant difference between GME and TLE patients as regards the combined therapy of Tegretol / Rivotril / Epanutine, Depakine / Epanutine, Rivotril / Epanutine, Tegretol / Rivotril / Depakine, Tegretol / Rivotril / Epanutine.

In the other hand, there was significant difference between GME patients as regards combined therapy of Depakine / Tegretol.

There was tendency of TLE to Depakine / Tegretol combination more than GME.

Our finding goes with the results reported by Kaufman and his colleagues, 1997 as regards the hepatotoxicity from anticonvulsants, especially cabamazepine and valproic acid, is a well known complication monitored by epileptologists. With the advent of these agents in the treatment of bipolar affective disorders, it is cirtical that psychiatrists be aware of the need for liver function monitoring.

And goes also with the results reported by Perucca and his colleagues, 1997 as regards the although most clinicians attempt to treat epilepsy on the basis of monotherapy, the need for polytherapy in fraction of the patient population makes drug interactions a significant issue. The problem od drug interactions also must be considered in the design and interpretation of clinical trials in which a new drug is added to pre existing treatment.

And goes also with the results reported by Czapinska and his colleagues, 1997 as regards the in cognitive functions, only patients on PHT showed transient and mil impairment of memory function and subjects on PB – memory deficits. The above cognitive finding combined with the frequency of adverse effects suggest the following order of drug selection: valproic acid, carbamazepine, phenobarbital and phenytoin.

6) According to the duration drug intake:

In our study there was a significant difference between GME and TLE patients concerning the duration of drug intake showing that:

GME was more than TLE patients in be Longer in duration of drug intake (speacially in patients with drug intake duration of more than 5 years).

The finding of this study goes with results reported by Vermeulen and his colleagues, 1997 as regards the in children with recurrent seizure, subjective seizure severity measured with the Hague Seizure Severity was not different after 1 year of treatment.

Our finding goes also with the results reported by Czapinski and his colleagues, 1997 as regards the studies directly evaluating the efficacy of such monotherapy when two drug are compared. The initially observed difference in drug effectiveness disappears in long-term therapy.

And goes also with the results reported by Hooge and his colleagues, 1997 as regards the however, the unfavourable ratio of therapyeutic dose versus dose inducing memory or motor impairment supports the prevailing notion that such adverse effects or the presently available compounds have precluded the use of NMDA antagonists as long-term therapies.

ii- *In details*

a) *Patients on monotherapy medication*

In our study there were significant decrease in either or all of immediate memory, auditory attention, short memory and internal visual scanning in TLE patients on monotherapy medication.

This means that monotherapy has a role in decreasing either or all of immediate memory, auditory attention, short memory and internal visual scanning in TLE patients. This beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Czapinski and his colleagues, 1997 as regards Studies directly evaluating the efficacy of such monotherapy when two drug are compared.The initially observed difference in drug effectiveness disappears in long-term therapy.

The finding of this study goes also with results reported by Conry and his colleagues, 1997 as regards the in view of the low risk of epilepsy and the known cognitive and behavioural effects of AED, AED should be used more cautiously in ECMO survivors.

b)Patients on Depakine (Na valproate) monotherapy:

In our study there was a significant increase in patients on Depakine (Na valproate) monotherapy lie scale in GME patients on monotherarpy with whether low dose or both low & high dose of depakine monotherapy.

This means that depakine monotherapy in high or low dose has a role in increase lie scale in GME patients. This beside the role of GME itself in increasing lie scale.

The finding of this study goes with results reported by Chris and his colleagues, 1995 as regards the they evaluated safety and efficacy of Depakote (divalproex sodium, (VPA) monotherapy in the treatment of patients with complex seizures (CPS) administered to achieve high $(80 - 15 \mu g/ml)$ total trough piasma concentrations of VPA, with a low VPA concentration group $(25-50 \mu g/ml)$ as active control.

c) Patients on Carbamazepine (Tegretol): monotherapy: (whether with low, high dose or both)

In our study there were significant decreases in sustained attention, persistencey, psychomotor speed, visuomotor coordination and learning ability in TLE

patients on low dose of monotherapy with Carbamazepine (Tegretol).

This means that Tegretol in monotherpay has a great role in decrease sustained attention, persistencey, psychomotor speed, visuomotor coordination and learning ability in TLE patients. This beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Trimble and his colleagues, 1990 as regards the individual AEDs have been shown to differ-the deleterious effects of phenytoin generally contrasting with the relatively minimal effects of valproate and carbamazepine.

There were also significant decreases in (either or all of immediate memory, auditory attention, short memory and internal visual scannings) in TLE patients on monotherapy with carbamazepire (Tegretol).

This means that monotherapy with tegretol has a role in decrease in TLE patients. This beside the role of TLE in decrease that.

The finding of this study goes with results reported by Reetta and his colleagues, 1995 as regards the VGB appears to be an effective an dsafe antiepileptic drug as primary monotherapy, with fewer cognitive side effects than CBZ.

There were also significant decreases in either or all of immediate memory, auditory attention, short memory and internal visual scannings in GME patients on monotherapy with high dose of carbamazepire (Tegretol).

This means that monotherapy with Tegretol in high dose has a role in decreasing either or all of immediate memory, auditory attention, short memory and internal visual scannings in GME patients. This beside the role of GME in decreasing that.

The finding of this study goes with results reported by Michael and his colleagues, 1988 as regards the differences in the behavioural toxicity profile of the anticonvulasant drugs with regards to their impact on cognitive function. In all of the studies carbamazepine comes out as the drug with the least impact, while phenytoin, and where studied clonazepam, appear to have maximal effect.

There were also a significant increase in anxiety in TLE patients on high dose of monotherapy with carbamazepire (Tegretol).

This means that monotherapy with Tegretol in high dose has a great role in increase anxiety in TLE patients. This beside the role of TLE in increase anxiety.

The finding of this study goes with results reported by Henriksen and his colleagues, 1990 as regards the balance is required because epileptiform discharges and even occasional seizures may be less disabling than side effects from large doses of several drugs.

There were also a significant increase in depression in GME patients on either high or low dose of monotherapy with carbamazepire (Tegretol).

This means that monotherapy with Tegretol whether low or high dose has a role in increase depression in GME patients. This is beside the role of GME in increase depression.

The finding of this study goes with results reported by Jinggang and his colleagues, 1995 as regards the findings have implications for the choice of drug in the management of epilepsy and also for the reported claims of a psychotropic effect of CBZ.

There were also significant decreases in sustained attention, persistencey, psychomotor speed, visuomotor coordination and learning ability in GME patients on either low or high dose of mono therapy with carbamazepine (Tegretol).

This means that monotherapy with tegretol either low or high dose has a role in decreasing sustained attention, persistencey, psychomotor speed, visuomotor coordination and learning ability in GME patients. This beside the role of GME in decreasing that in epileptic patients.

The finding of this study goes with results reported by Colin and his colleagues, 1995 as regards the patients were given psychological testing on a battery which included memory, attention and concentration, perceptual speed, decision making speed and motor speed tasks.Experimental and clinical data suggest that lamotrigine (LTG) is relatively nonsedative and that is has positive effects on cognition and affect

b) Patients on polytherapy medication:

1- With average dose:

In our study there were significant decrease in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in TLE patients on poly therapy with average dose.

This means that psytherotisy with average dose has a role in decreasing sustained attention, persistency, psychomotor speed, visuomotor coordination and

learning ability in TLE patients. This is beside the role of TLE itself in decreasing that.

The finding of this study goes with results reported by Trimble and his colleagues, 1995 as regards that although individual drugs may thus have a different impact with regards to cognitive function, polytherapy itself may be an important variable.Lowering the anticonvulsant prescription load of children improves cognitive performance, while increasing it has a deleterious effect on learning abilities.

The finding of this study goes with results reported by Trimble and his colleagues, 1995 as regards Prolonged and frequently repeated seizures are typically associated with more severe effects on cognitive functioning, particularly if epilepsy is symptomatic i.e., secondary to a demonstrable brain lesion.

2- With high dose:

In our study there was a significant increase in psychotocism in GME patients on high dose of polytherapy mediation.

This means that polytherapy with high dose has a great role in psychoticism in TLE patients. This is beside the role of GME itself in decrease psychoticism.

The finding of this study goes with results reported by Spector and his colleagues, 1995 as regards the possibility of psychological intervention for the management of seizures seems promising, especially for patients whose seizures are not controlled by medication.

There was also a significant increase in neuroticism in GME patients on high dose of polytherapy mediation.

This means that polytherapy in high dose has a great role in increase neuroticism in GME patients. This is beside the role of GME itself in increase neuroticism.

The finding of this study goes with results reported by Ignatowicz and his colleagues, 1995 as regards the psychotropic effect of Tegretol CR was effective in periodical psychotic and behavior disturbances in epilepsy.

3- Patients on polytherapy with Sodium Valproate (Depakine) and Carbamazepine (Tegretol):(Whether with low, high dose or both):

In our study there was a significant increase in anxiety in GME patients on low dose of polytherapy with sodium valproate (Depakine) and Carbamazepine (Tegretol).

This means that polytherapy with Depakine and Tegretol in low dose has a great role in increase anxiety in GME patients. This is beside the role of GME itself in increase anxiety.

The finding of this study goes with results reported by Bozdemir and his colleagues, 1997 as regards the although carbamazepine (CMZ) and sodium valproate (NaV) are used for the treatment of different type of seizures, it is well known that their effect is on the ionic channels, recent studies have suggested that the two drugs are equally efficacious in the treatment of both generalised treatment of partial seizures. We investigated the efficacy of these drugs in the treatment of partial sizures.

Our finding goes also with the results reported by Kaufman and his colleagues, 1997 as regards the Hepatotoxicity from anticonvulsants, especially cabamazepine and valproic acid, is a well known complication monitored by epileptologists. With the advent of these agents in the treatment of bipolar affective disorders, it is cirtical that psychiatrists be aware of the need for liver function monitoring.

7) Seizures control:

i- Generally:

a) Controlled seizures

In our study there was no significant difference between GME and TLE groups as regards patients with controlled seizures.

The finding of this study goes with results reported by Aldenkamp and his colleagues, 1995 as regards the cognitive effects are sometimes the effects of chronic treatment, but most of our knowledge is produced by normal volunteer studies that use periods of drug exposure varying form 1 day to 1 month.

Our finding goes also with the results reported by Wang and his colleagues, 1997 as regards the tolerance/dependence to antiseizure of CZP were not metabolic and mainly related to $GMBA_A - R$ down-regulation/up-regulation.

The finding of this study goes with results reported by Tohru and his colleagues, 1995 as regards the EEG findings, remaining paroxysmal discharges did not necessarily mean the occurrence of relapse, but the changes in background activity with age, which may indicate CNS maturation, were significant different between the patients with or without relapses. Our results suggest that the factors related to age

should be considered according to the epileptic syndrome when one decides to discontriue AED treatment.

There is also a significant difference between GME and TLE groups as regards type of controlled seizures patients (on monotherapy or polytherapy). GME patients shows a tendency to controlled seizure pateints on monotherapy more than TLE patients.

The finding of this study goes with results reported by Quattrini and his colleagues, 1997 as regards the drug resistant patients showed positive correlation with mental findings, very early age of epilepsy onset, higher frequency of status epilepicus. To try to understand the significance and mechanism of drug-resistance further parameter have been investigated, the most relevant of them are the duration of illness, the risk factor for epilepsy and the therapy.

The finding of this study goes with results reported by Rodin and his colleagues, 1986 as regards the control of seizures was found to be associated with IQ in a study (who noted a stable or increases IQ for patients in remission, but a decrease in IQ or patients with poorly controlled epilepsy. These changes were small, however, and not statistically significant.

The finding of this study goes with results reported by Halstead and his colleagues, 1957 as regards the degree of seizure control would also seem to be important, good control being associated with higher IQ values. But again, there are conflicting reports.

b)None controlled seizures

In our study there also was no significant difference between GME and TLE groups as regards patients non-controlled seizures.

The finding of this study goes with results reported by Nikolaishvili and his colleagues, 1997 as regards the general sign was dis-co-ordination between seizure control and normalisation of EEG patterns during the treatment. Even without seizures the daytiem EEG and 24h EEG monitoring show the focal dysrrhthmia appearance of slowing of sharp waves after stimulation (hyperventilation, sleep deprivation)

The finding of this study goes with results reported by Pauli and his colleagues, 1997 as regards the chemical Shift Imaging (CS) seems to be able to predict memory functions of patients in TLE.

. There is also a significant difference between GME and TLE groups as regards type of non-controlled seizure patients (on monotherapy or polytherapy). GME

patients shows a tendency to non-controlled seizure patients on monotherapy more than TLE patients.

The finding of this study goes with results reported by Czapinski and his colleagues, 1997 as regards the recent studies indicate a possibility of successful treatment of drug-resistant epileptic seizures employing new genration antiepileptic drugs used in monotherapy. However, the date are scant, similarly as studies directly evaluating the efficacy of such monotherapy when two drug are compared. The initially observed difference in drug effectiveness disappears in long-term therapy.

ii- In details

a) Patients without Controlled seizures:

In our study there were significant decreases in coceptualization, concrete thinking, and general mental ability in GME patients without controlled seizures on either monotherapy or polytherapy.

This means that uncontrolled seizures with mono or polytherapy has a great role in decreasing coceptualization, concrete thinking, and general mental ability in GME patients. This is beside the role of GME itself in decrease that.

The finding of this study goes with results reported by Trimble and his colleagues, 1995 as regards the difficult of interpretation is the number of variables that may influence cognition. Such as polytherapy and seizure frequency, for which these studies have not been able to control.

There was also significant increase in introversion in TLE patients without controlled seizures on either monotherapy or polytherapy.

This means that controlled seizures on mono or polytherapy has a great role in increase introversion in TLE patients. This is beside the role of TLE itself in increase introversion.

The finding of this study goes with results reported by Matinez and his colleagues, 1995 as regards the efficacy of VGB analyzed by seizure type showed 66% of patients with complex partial seizures to have complete remission. Our results indicate that VGB has significant antiepileptic activity when added to the standard therapy of some children with intractable epilepsy.

There was also significant increase in lie scale in GME patients without controlled seizures on either monotherapy or polytherapy.

This means that controlled seizures on mono or polytherapy has a great role in increase lie scale in GME patients. This is beside the role of GME itself in increase lie scale.

The finding of this study goes with results reported by Muscas and his colleagues, 1997 as regards the clinical trials do not provide all the information on practical use of antiepileptic drugs (AEDs) in patients with epilepsy of variable severity, especially regarding optimal daily dosage, and long-term efficacy and folerability. Through a multicentre, open-lable study, in condition as close as possible to clinical practice, the long-term efficacy, tolerability and safety of Gabapentin (GBP) as add-on therapy were assessed in a large population of patients with uncontrolled partial epilepsy in whom previous AEDs treatment was poorly tolerated.

The finding of this study goes also with results reported by Shorvon and his colleagues, 1979 as regards the their suggest that rationalization of polytherapy brings improvement of mood.

b)Patients with Controlled seizures:

In our study there were significant decreases in attention and response rate in GME patients with controlled seizures on either monotherapy or polytherapy.

This means that controlled seizures on monotherapy or polytherapy has a great role in decreasing attention and response rate in GME patients. This beside the role of GME itself in decrease that.

The finding of this study goes with results reported by Quattrini and his colleagues, 1997 as regards the drug resistant patients showed positive correlation with mental retardation, complex partial seizures, pathological CT and/or MRI findings, very early age of epilepsy onset, higher frequency of status epielpticus.

There were significant increase in anxiety in TLE patients with controlled seizures on either monotherapy on polytherapy.

This means that controlled seizures on mono or polytherapy has a role in increase anxiety in TLE patients. This beside the role of TLE in increase anxiety.

The finding of this study goes with results reported by Maria and his colleagues, 1995 as regards the therapy with either CBZ or PB was equally effective in seizure control.

There was also significant increase in lie scale in TLE patients with controlled seizures on either monotherapy or polytherapy.

This means that controlled seizures on mono or polytherapy has a great role in increase lie scale in TLE patients. This is beside the role of TLE itself in increase lie scale.

The finding of this study goes with results reported by Moran and his colleagues, 1997 as regards the patients referred for surgery of intractable TLE benefit from 0a structured psychiatric evaluation of their extensive and significant coexisting psychiatric illness and psychosocial disability.

8) Compliance:

I) Generally

a) Compliant patients

In our study there was no significant difference bettween GME and TLE groups as regards compliance.

The finding of this study goes with results reported by Ossetw and his colleagues, 1968 as regards the even though the total toxicity battery did reveal some differences in the

cognitive effects of these four drugs, the most striking finding is that there were so few meaningful differences in the individual subtests. It is likely that when patients are managed by experienced epileptologists adverse effects of AEDs are minized.

There is a significant difference between GME and TLE groups as regards type of compliant patients (on monotherapy or polytherapy). GME patients shows tendency to compliant on on monotherapy more than TLE patients.

The finding of this study goes with results reported by Henriksen and his colleagues, 1990 as regards the thompson and Trimble have investigated memory among other cognitive functions in a number of studies with epileptic patients, and volunteers given anti-epileptic drugs. They have consistently employed the same battery of tests. Their memory test consists of immediate and delayed recall of twenty words and twenty pictures, followed by a recognition test of the items embedded in distractor items. (Thompson et al., 1982). Found improved memory function in patients undergoing reduction in polytherapy or substitution of their discharges and even occasional seizures may be less disabling than side effects from large doses of several drugs.

b) Non compliant patients

In our study there also was no significant difference between GME and TLE groups as regards non-compliance.

The finding of this study goes with results reported by Trimble and his colleagues, 1995 as regards the although individual drugs may thus have a different impact with regards to cognitive function, polytherapy itself may be an important variable. Lowering the anticonvulsant prescription load of children improves cognitive performance, while increasing it has a deleterious effect on learning abilities.

There is a significant difference between GME and TLE groups as regards type of non-compliant patient (on montherapy or polytherapy).GME patients shows a tendency to non-compliant on monotherapy more than TlE patients

The finding of this study goes with results reported by Muscas and his colleagues, 1997 as regards the clinical trials do not provide all the information on practical use of antiepileptic drugs (AEDs) in patients with epilepsy of variable severity, especially regarding optimal daily dosage, and long-term efficacy and folerability. Through a multicentre, open-lable study, in condition as close as possible to clinical practice, the long-term efficacy, tolerability and safety of Gabapentin (GBP) as add-on therapy were assessed in a large population of

patients with uncontrolled partial epilepsy in whom previous AEDs treatment was poorly tolerated.

ii- In details

a) Compliant patients

i-Compliance on polytherapy:

In our study there were also significant decreases in either or all of immediate memory, auditory attention short memory and metnal visual scanning in GME complient patients or polytherapy.

This means that complient patients on polytherapy has a great role in decreasing either or all of immediate memory, auditory attention short memory and metnal visual scanning in GME patients. This beside the role of GME itself in decrease that.

The finding of this study goes with results reported by Kowalik and his colleagues, 1995 as regards the behavioral disorders (apart from peri-ictal phase), especially aggression, have been observed in adults and children with temporal lobe epilepsy (TLE) in about one third of cases.

Fifty-eight percent of the children with behavioural distubances showed improvement of their psychic disorder after surgery. All these patients remained seizure-free.

The finding of this study goes with results reported by Cerullo and his colleagues, 1997 as regards the intractable epilepsies are associated with more seizure types during illness evolution a probable expression of multiple independent EEG foci; epileptic drop attacks and mental deterioration.

There were also significant decreases in attention and response rate in GME compliant patients in either mono or polytherapy.

This means that compliant on mono or polytherapy has a great role in decrease attention and response rate in GME patients. This beside the role of GME itself in decrease attention and response rate.

The finding of this study goes with results reported by Murthy and his colleagues, 1997 as regards the severe moto and intellectual disabilities (SMID) is term used to describe a heterogeneous group of disorders, including cerebral palsy, profound mental retardation and intractable epilepsy. A few patients achieved control of epilepsy in adolescence, but the majority suffered from epilepsy throughout their lives.

Seizure seminology referable to temporal lobe and frontal lobe had lower remission rates and this was much more so in

patients with cryptogentic epilepsy. Life table analysis of the data will be presented.

ii- Compliance on monothrapy;

In our study there were significant decreases in attention and response rate in GME compliant patients on monotherapy.

This means that compliance of patients on monotherapy has a great role in decreasing attention and response rate in GME patients. This is beside the role of GME itself in decrease attention and response rate.

The finding of this study goes with results reported by Cerullo and his colleagues, 1997 as regards the intractable epilepsis are associated with more seizure types during illness evolution a probable expression of multiple independent EEG faci; epileptic drop attacks and metnal deterioration.

There was also significant increases in lie scale in GME compliant patients on monotherapy.

This means that compliant patients on mono or polytherapy has a great role in increase lie scale in

GME patients. This beside the role of GME role in increase lie scale.

The finding of this study goes with results reported by Andrewes and his colleagues, 1986 who compared new referrals with epilepsy, well controlled on single drug therapy, with either phenytoin or carbamazepine and an untreated control group with respect to their performance on a number of cognitive tasks.

The finding of this study goes also with results reported by Smith and his colleagues, 1985 as regards the noticed significant between the groups in favour of carbamazepine was reported to have the least effect on a number of tasks including finger tapping, colour naming, a Peg board test, a digit symbol substitution test, and discriminative reaction time. Phenytoin and phenobarbitone provided the worse scores, and correlations between deterioration and serum levels were noted.

iii- Compliance on either mono or polytharapy:

In our study there was significant increases in introversion in TLE compliant patients in either mono or polytherapy.

This means that compliant patients on mono or polytherapy has a great role in increase introversion.

This beside the role of TLE itself in increase introversion.

The finding of this study goes with results reported by Geladze and his colleagues, 1997 as regards the Carbamazepine (CBZ) medication needs detailed longitudinal EEG control for right management of treatment.

II)Non-compliant patients:

i- On monotherapy

In our study there was a significant increase in introversion in TLE non-compliant patients on monotherapy.

This means that non- compliance of patients on monotherapy has a great role in increase introversion in TLE patients. This beside the role of TLE itself in increase introversion.

The finding of this study goes with results reported by Murthy and his colleagues, 1997 as regards the seizure seminology referable to temporal lobe and frontal lobe had lower remission rates and this was much more so in patients with lower remission rates and this was much more so in patients with cryptogenic epilepsy. Life table analysis of the data will be presented.

ii- On polytherapy:

In   our study there was a significant increase in lie scale in GME non-compliant patients on polytherapy

This means that non - compliance of patients on polytherapy has a great role in increase lie scale in GME patients. This beside the role of GME itself in increase lie scale

The finding of this study goes with results reported by Salke and his colleagues, 1997 as regards the children and adolescents with severe epilepsy, along-term hospitalisation may be inevitable. We asked if such a hospitalisation is justified in relation to the outcome.

iii-Mono or polytherapy:

In our study there was a significant increase in introversion in TLE non-compliant patients in either mono or polytherapy.

This means that compliance of TLE patients on either mono or polytherapy has a great role in increasing introversion in TLE patients. This is beside the role of TLE itself in increase introversion.

The finding of this study goes with results reported by Wai – Keong and his colleagues, 1997 as regards the magnetic resonance imagining evaluation of patients with intractable partial epilepsy is useful. 43% have changes consistent with mesial temporal sclerosis and are possible candidates for epilepsy surgery.

IV) EEG:

N.B :

i- **_Generally means_** *the comparison between GME, TLE and control groups concerning the EEG.*

ii- **_In details means_** *the relation between different aspects of EEG and performance on the different psychometric tests*

i- Generaly:

In our study ,as regards the EEG of the GME patients neralized activity , 19.2% with temporal discharges with secondary generalization and 2.7 % without EEG changes .

And as regards the EEG of the TLE patients, were 33.02 % with right temporal discharges there , 53.77% with left temporal discharges and 13.21% with bi-temporal discharge

ii- *In Details*

In our study , Patients with EEG temporal focci showed significant decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability ability more than EEG patients with temporal focci.

The finding of this study goes with results reported by Jolanta and his colleagues, 1988 as regards the impairment of sustained attention, as measured by the CPT, is patients with centrencephalic epilepsy was already noted in early studies, have demonstrated that this deficit was associated with spikewave activity, as shown by simultaneous EEG monitoring and task performance. Symmetrical, regular and synchronous bursts produced more deficit in performance than other bursits.

Patients with EEG of ELT temporal focci decreases in either or all of immediate memory, auditory attention, short memory and internal visual scanning more than patients with EEG of Generalized subcortical activity and of Rt. temporal focci respectively.

Our finding goes with the results reported by Trimble and his colleagues, 1995 as regards the difficult of interpretation is the number of variables that may influence cognition. Such as polytherapy and seizure frequency, for which these studies have not been able to control.

Patients with EEG of similarities Generalized activity showed significant decreases in conceptualization concrete thinking, and general mental ability more than patients with EEGs of secondary generalization.

The finding of this study goes with results reported by Herronz and his colleagues, 1995 as regards the rufther studies of monotherapy in newly diagnosed patients with any types of seizures is recommended for better definition of the efficacy and safety profile of VGB.

Patients with EEG of Rt temporal focci showed significant decreases in attention and response rate more than EEG of secondary generalization.

The finding of this study goes with results reported by Henriksen and his colleagues, 1990 as regards the computerized neuropsychological testing with simultaneous EEG recording may reveal the influence of epileptifrom discharges on cognitive fuction and also help to evaluate the effects of antiepileptic drugs.

Patients with EEG's of the temporal focci showed significant increase in impulsively, response rate, more than Rt. temporal focci with EEG's of generalized subortral activity.

This goes with the results reported by Christine and his colleagues, 1988 as regards the in a direct comparison of different seizure types, found that children with partial seizures obtained better scores on some Wechsler subtests than did those with generalized.

Patients with EEG temporal focci showed significant decreases in sustained attention, ersistency, psychomotor speed, visuomotor coordination and learning ability ability more than EEG patients with temporal focci.

The finding of this study goes with results reported by Jolanta and his colleagues, 1988 as regards the impairment of sustained attention, as measured by the CPT, is patients with centrencephalic epilepsy was already noted in early studies, have demonstrated that this deficit was associated with spikewave activity, as shown by simultaneous EEG monitoring and task performance. Symmetrical, regular and synchronous bursts produced more deficit in performance than other bursits.

Our finding goes also with the results reported by Mar Kovic and his colleagues, 1995 as regards the neuropsychological changes obserbed after right temporal lobe resections indicate a role in memory for nonverbal material by the nondominant temporal lobe, but the relative

contribution of the hippocampal subfields to this process has not been established.

The relationship between seizure type and intellectual ability is unclear. However, some investigators have found no relationship between petitmal seizures and IQ. Children with grand and seizures, psychomotor or partial seizures have also been found to be unimpaired on tests of intellectual ability.

And goes also with the results reported by Ladavas and his colleagues, 1979 as regards the investigated left versus right hemisphere memory function as assessed by the STM and LTM performance on a variety of verbal and non-verbal test. Although the STM tasks did not differentiate between the left and right temporal lobe foci patients, the LTM tasks did not differentiate between the left and right temporal lobe foci patients, the LTM tasks did so, in the expected verbal/non-verbal direction. Similar results were obtained by (Delaney et al., 1980) who found that differences between the right and left TLE patients were not apparent on immediate recall of either the verbal or non-verbal material, but only on delayed recall.

Patients with EEG of TLE temporal focci decreases in either or all of immediate memory, auditory attention, short memory and internal visual scanning more than patients with EEG of Generalized subcortical activity and of Rt. temporal focci respectively.

The finding of this study goes with results reported by Mc Mackon and his colleagues, 1997 as regards the patients with right sided seizures showed a greater difference score on the VR than on the LM subtest (p<0.05), while those with left sided seizures foci showed greater difference scores of the LM compared to the VR subtests.

Our finding goes also with the results reported by Hendriks and his colleagues, 1997 as regards the patients with a left temporal focus were significant more impaired in learning verbal episodic knowledge, a left temporal focus, the performances on episodic memory tasks correlated with the scores on semantic memory tasks with regard to knowledge of categorical information. There is no relation with other aspects of semantic memory.

And goes also with the results reported by Sang and his colleagues, 1995 as regards their findings indicate that memory functions of the epileptic dominant hemisphere are weakly disrupted, whereas memory functions of the epileptic nondominant hemisphere are more strongly disrupted. Consequently, memory functions of the epileptic dominant hemisphere are relatively good, but memory functions of the epileptic nondominant hemisphere are very poor.

And goes also with the results reported by Bolling and his colleagues, 1995 as regards the considerable evidence has confirmed a complementary specialization of the temporal lobes for information processing and retention in man.

According to this hypothesis, early isolated cerebral lesions of the LH will strongly influence cognitive functions of the RH, whereas isolated lesions of the RH do not influence LH functions to the same extent due to a lesser reorganization capacity of the LH.

Patients with EEG of similarities Generalized activity showed significant decreases in conceptualization concrete thinking, and general mental ability more than patients with EEGs of secondary generalization.

The finding of this study goes with results reported by Chan and his colleagues, 1997 as regards the attempt to correlate these histopathologies with patients gender, seizure types, age of onset, MRI findings and surgical outcome. Epilepsy onset is most frequent between age 1-5 years. Patients without secondarily generalized seizures have better long-term prognoses.

Our finding goes also with the results reported by Lee and his colleagues, 1997 as regards the frequency of 2nd GS in patients with TLE was found to be an important factor in the prognosis, even in age matched comparisons.

Patients with EEG of Rt temporal focci showed significant decreases in attention and response rate more than EEG of secondary generalization.

The finding of this study goes with results reported by Henriksen and his colleagues, 1990 as regards the computerized neuropsychological testing with simultaneous EEG recording may reveal the influence of epileptifrom discharges on cognitive fuction and also help to evaluate the effects of antiepileptic drugs.

Patients with EEG's of Rt. temporal focci showed significant decreases in attention and response rate more than EEG's of secondary generalization.

Patients with EEG's of Rt. temporal focci showed significant decreases in attention and response rate more than EEG's of secondary generalization.

Patients with EEG's of generalized sub-cortical activity showed significant increase in impulsivety, more than patients with free EEG's with clinical convulsions.

The finding of this study goes with results reported by Aleksic and his colleagues, 1997 as regards the EEG follow-up has shown relationship between quantity of discharges and clinical findings.

Patients with EEG's of Lt. temporal focci showed significant increase in impulsivety, and decrease in

response rate and attention, more than patients with EEG's of Rt. temporal focci with EEG's of generalized sub-cortical activity.

Patients with EEG's of generalized sub-cortical activity showed significant increase in impulsivety and decrease attention and response rate more than patients with EEG's of secondary generalized activity.

No significant difference in anxiety between different groups

The finding of this study goes with results reported by Maryek and his colleagues, 1997 as regards the sometimes severeattacks, with prologed tonic conulsions occurred without loss of consciousness. Paroxysmal conditions were very frequent and sometimes accompanied with fear, anxiety. Differentiation between epileptic and pseudo-epileptic seizure was often difficult because of a typical EEG too. Cognitive defects, emotional instability increased rapidly. The psychosocial consequences were especially significant for the patients of efficiency age. In spite of intensive complex treatment the curable effect in most patients was poor.

Our finding goes also with the results reported by Binnie and his colleagues, 1990 as regards the in some individuals, suppression of discharges by antiepileptic drugs has

demonstrable improved psychological function, but further work is required to determine the indications for such treatment.

Patients with EEG's of Lt. temporal focci with secondary generalization showed significant increase in depression more than patients with EEG's of Lt. temporal focci only.

The finding of this study goes with results reported by Savic and his colleagues, 1997 as regards the none positive correlation between the number of seizure and the amount interictal epileptiform activity.

Our finding goes also with the results reported by Euan and his colleagues, 1988 as regards the uncoordinated behaviour clinically typical cases can be found despite lack of characteristic EEG changes.

Patients with EEG's of Lt. temporal focci showed a significant increase in introversion more than patients with EEG's of generalized sub-cortical activity.

Patients with EEG's of Rt. Temporal focci showed a significant increase in introversion more than patients

with EEG's of Rt. Temporal focci with secondary generalization.

Patients with EEG's of generalized sub-cortical activity showed increase in introversion more than patients with EEG's of secondary generalization.

The finding of this study goes with results reported by Abdulghani and his colleagues, 1997 as regards the children with abnorma EEG had significant deterioration of personality and behaviour adjustment. This was more apparent in patients with focal EEG dysrhythmia. Intellectual deterioration was not related to EEG abnormalities.

Our finding goes also with the results reported by Coline and his colleagues, 1988 as regards the based on the idea that paroxysmal disorders of behaviour arise form paroxysmal discharges of subcortical structures which were not necessarily recorded in the EEG from the surface of the cortex.

It was recognition that electrical discharges in the EEG correlated with observed seizure behaviour that lead to a taxonomy of epilepsy based on EEG waveforms. Indeed, the first international classification of epilepsy relied heavily on different EEG pictures for different diagnoses.

And goes also with the results reported by Binnie and his colleagues, 1990 as regards the sub-clinical generalized spike-wave discharges are often accompanied by transitory cognitive

impairment, demonstrable by psychological testing during EEG recording.

Summary
and
Conclusions

Summary

Our sample consisted of epileptic patients, whose diagnoses was based on the criteria and encephalographic classfication of epileptic seizures according to the International League against Epilepsy 1993.

Beore starting to collect the data, a pilot study were carried out in :

1) The Psychiatry and Neurology Centre for school pupils in El Dokki District

2) The General polyclinic for school pupils in Al-Haram Distric

Following the pilot study, we found out that the ratio between TLE and GME is 2:3.

We found out that the tools were suitable, comprehensible and easy, however some modification of the questionaire concerning epilepsy was essential.

First, centre, recruits patients from Giza and northern upper Egypt, it is a governmental one, affilated to the Central Health Insurance Authority and offers medicine free of charge to its patients.

Around 40 to 50 patients are attend the centre daily, namely about 1000 monthly, 60% of whom suffered from epilepsy. 50% of epileptic children could be diagnosed as GME, 33% as TLE and 17% as patients suffered from other types of epilepsy.

From the epileptic attendats 10% were new cases and 90% were recurrent.

Second centre is a governmental institution affiliated to the General Health Insurance Authority, and offers medicine free of charge. About 25 to 30 patients are attended the clinic daily, namely about 700 patients a month, 60% of which are suffered from epilepsy. 50% of epileptic children could be diagnosed as GME, 33% as TLE and 17% as patients suffered from other types of epilepsy.

From the epileptic attendats 10% were new cases and 90% were recurrent.

From 308 GME patients from site (1) and 244 from site (2) . 338 patients fulfilled the inclusion criteria. Cases chosed through random sample of every second case giving 169 case with drop out of 23 . the rest were 146.

From 198 TLE patients from site (1) and 156 from site (2) . 236 patients fulfilled the inclusion criteria. Cases chosed through random sample of every second case giving 118 case with drop out of 12. the rest were 106.

The members of this sample were selected during the period from February 1995 throught August 1996.

Tools Applied in this work

1) Semistructured psychiatric interview.

2) Questionaire of Epilepsy.

3) Psychometric battery :

   I- For Cognitive functions :

   Coding (SubWechseler), Digit Span (SubWechseler), Similarities (SubWechseler), Cancellation letters and Matching Familiar Figure Test (MFFT)..

   II- For personality assessement and measurement of mood :

   Children Depression Inentory (CDI), The children Anxiety scale (CAS), Junior Eysenk Personality Questionaire (JEPQ)

4) Questionaire for Socio-economic state.

5) Electro Encephalogram

6) Others investigations :

   BEAM, MRI, CAT and or in some cases Serum level

Conclusions.

In our study, there was no significant difference found in different psychological, cognitive and personality scales between GME and TLE concerning the psycho-demographic profile. Illness profile. therapeutic issues and electroencephalogram (EEG) apart from the following:

i- Assessment of cognition

1- Coding test:

a - Generally

- **There were significant difference between GME, TLE patients & control group in sustained attention, persistency, psychomotor speed, visuomotor coordination and Learning ability.**

- **There was significant increase in control group more than GME and TLE patients.**

b - In details

- **There were decreases in sustained attention, persistency, psychomotor speed, visuomotor coordination and learning ability in the following:**

  - TLE patients with onset of age above 4 years.
  - TLE patients with duration of illness between (2 - 4) years.
  - TLE patients with longest seizure fee period from below 6 months up to 2 years.
  - TLE patients without status epilepticus.
  - TLE patients without positive family history.
  - TLE patients with positive past history.
  - TLE patients with aura.

- TLE patients with ictal changes.
- TLE patients with past ictal changes.
- TLE patients without one or more of the organic disease.
- TLE patients with neurotic traits.
- GME patients without neurotic traits.
- GME patients bring in up to two rooms.
- TLE patients of non-educated parents.
- TLE patients on Tegretol monotherapy in low dose.
- GME patients on Tegretol monotherapy either on low or high dose.
- TLE patients on polytherapy with average dose.
- TLE patients without contributed seizures on monotherapy.
- Patents with Rt. Temporal focci more than Rt. Temporal focci in EEG.

2-Digit span test

a - Generally

- **There were no significant difference between GME, TLE patients and control group in either or all of immediate memory, auditory attention, short memory and internal visual scanning.**
- **This means that control group is not better than both epileptic groups (GME & TLE) in either or all of**

immediate. memory auditory attention, short memory & internal visual scanning.

b- In details:

- **There were decreases in either or all of immediate memory, auditory attention short memory and internal visual scanning in the following:**
    - GME patients with abrupt onset of disease.
    - GME patients with longest seizure free period is even less than 6 months
    - TLE patients without on or more of organic disease
    - TLE male patients
    - TLE patients on monotherapy medication
    - TLE patients on Tegretol monostherapy
    - GME patients on Tegretol monotherapy with high dose.
    - GME compliant patients on polytherapy
    - Patients with Rt. Temperal focci more than Rt. Temporal focci and generalized subcortical activity in EEG.

3) similarities test:

a - Generally

- **There were significant difference between GME, TLE patients and control group in conceptualization, concrete thinking, and general mental ability.**
- **There was an increase in control group more than GME and TLE patients.**

- **There was also an increase in TLE patients more than GME patients.**

b- In details.

- **There were decreases in conceptualization, concrete thinking, and general mental ability in the following:**
    - GME patients with longest seizure free period below six months.
    - GME patients with longest seizure free period is even less than six months.
    - GME patients with positive past history
    - GME patients in parents on monotherapy without controlled seizures
    - Patients with generalized subcortical activity more than patients with secondary generalization activity in EEG.

4) Cancellation letters test (Time & Errors)

a - Generally:

- **There were significant difference between GME, TLE patients and control group in attention and response rate.**
- **There was significant increase in control group more than GME & TLE patients.**

- **There was no significant difference between GME & TLE patients but (GME>TLE) patients.**

b- In details

- **There was significant decreases in attention and response rate in the following:**
  - GME patients with gradual onset of disease
  - TLE patients with past seizure frequency of every week.
  - TLE patients with past seizure frequency of every day.
  - GME patients without status epilepticus.
  - GME patients without positive past history .
  - GME patients with one or more of the factors affecting ictus.
  - GME patients with one or more of the organic disease.
  - TLE patients without pregnancy troubles.
  - GME patients in big family size.
  - GME patients with educated parents.
  - GME patients with controlled seizures on monotherapy.
  - GME patients on both either mono or polytherapy with controlled seizures.
  - GME compliant patients on both either mono or polytherapy.
  - GME compliant patients on monotherapy.
  - Patients with Rt. Temporal focci more than secondary generalized. activity in EEG.

5) Matching familiar figure test-Total errors and total and Mean time (MFFT):

a- Generally:

- There was significant difference between GME, TLE patients and control group (normal population) in impulsivity, attention & response rate.
- There was significant increase in GME patients more than control group (normal population).
- There was significant increase in TLE patients more than control group (normal population).
- There was no significant difference between GME and TLE patients .

b- In details

- There is significant increase impulsivity and decrease in attention and response rate in the following:
    - GME patients with gradual onset of disease
    - TLE patients with duration of illness between 6 months - 2 years.

- *GME patients with longest seizure free period is even less than 6 months*
- *TLE patients without organic disease*
- *TLE patients without pregnancy troubles*
- *TLE patients without labour troubles*
- *TLE patients without neurotic traits*
- *TLE patients with family sib-order up to 1st two sib-order*
- *TLE patients in big family size.*
- *TLE rural patients*
- *TLE patients in rural residence*
- *Patients with generalized sub cortical activity more than patients with free EEG*
- *Patients with left temporal focci more than patients with right temporal focci and patients with generalized sub-cortical activity.*
- *Patients with generalized sub-cortical activity more than patients with secondary generalization.*

ii- Assessement of mood :

1) The children anxiety scale (CAS).

a- Generally :

- **There was no significant difference between GME, TLE patients and control group (normal population) in increasing anxiety.**
- **GME, TLE group of patients increase anxiety significantly more than control group (normal population).**

b- In details

- **There is significant increase in anxiety in the following:**
  - TLE patients with gradual onset of disease.
  - TLE patients with past seizure frequency of every month seizure.
  - TLE patients with one or more of the cerebral causes
  - TLE patients on tegretol monotherapy with high dose
  - GME patients on polytherapy with depakine and tegretol
  - TLE patients on both either mono or polytherapy with controlled seizures.

2) The children depression inventory (CDI) :

a- Generally :

- **There was a significant difference between GME, TLE patients and control group (normal population) in increasing depression.**
- **There was no significant difference between each group and other but there is increase in GME > TLE , GME > control group and TLE > control group .**

b- In details

- **There is significant increase in depression in the following:**
  - TLE patients with current seizure frequency of every week.
  - TLE patients with history of past seizure frequency of frequent seizures every day.
  - TLE patients with status epilepticus
  - TLE patients with positive past history.
  - GME patients with neurotic traits
  - GME patients with history to be wanted during pregnancy
  - GME patients living in up to two rooms
  - GME patients with family order more than two sib-order
  - GME patients in big-family size
  - GME male patients
  - GME patients with non educated parents.
  - GME patients with high family income
  - GME patient with parental separation

- GME patients on tegrotol monotherapy with either low or high dose
- Patients with left temporal focci with second generalization more than left temporal focci only.

iii - Assessment of personality (Psychoticim, Neuroticism, Introversion & Lie scale.)

a- Generally

- There was significant difference between GME, TLE patients and control group (normal population) in neuroticism where GME > TLE and TLE > control groups.

- There was no significant difference between GME, TLE patients and control group (normal population) in psychoticism. But there are increase in psychoticism in TLE > GME and GME > control groups.

- There was no significant difference between GME, TLE patients and control group (normal population) in introversion. But there are increase in introversion in TLE > control and control > GME groups.

- There was no significant difference between GME, TLE patients and control group (normal population) in

*lie scale. But there are increase in lie scale in GME >
control and control > TLE groups.*

b- In details

1- There is significant increase in neuroticism in the following:

- *GME patients with gradual onset of disease*
- *GME patients without positive family history*
- *TLE patients with one or more of the factors affecting ictus.*
- *GME male patients.*
- *GME patients on polytherapy with high dose.*

2- There is significant increase introversion in the following:

- *TLE patients with onset of disease above 4 years*
- *TLE patients with current seizures frequency of every week*
- *TLE patients with duration of illness above 5 years*
- *TLE patients with longest seizures free period up to two years.*
- *TLE patients without status epilepticus.*
- *TLE patients without positive past history*
- *TLE patients with prodroma*
- *TLE patients with aura*
- *TLE patients with ictal changes*

- *TLE patients with post-ictal changes*
- *TLE patient with one or more of the factors affecting ictus.*
- *TLE patients with one or more of systemic causes.*
- *TLE patients in big family size.*
- *TLE patients on monotherapy without controlled seizures*
- *TLE patients on both either mono or polytherapy without controlled seizures.*
- *TLE compliant patients on both either mono or polytherapy.*
- *Patients with left temporal focci more than patients with generalized sub-cortical activity in EEG*
- *Patient with right temporal focci more than patients with right temporal focci with secondary generalization in EEG.*

3- There is significant increase lie scale in the following:

- *TLE patients with abrupt onset of disease*
- *GME patients with age of onset between 2 - 4 years.*
- *GME patients with pregnancy troubles*
- *GME patients with dapakine monotherapy whatever in high or low dose.*

4- There is significant increase in psychoticism in the following:

- *TLE patients with duration of illness below 6 months*
- *TLE patients without status epilepticus.*
- *TLE patients with prodroma*
- *TLE patients with ictal changes*
- *TLE patients with past-ictal changes*
- *TLE patients with history to be not wanted during pregnancy*
- *TLE patients with family order up to first two sib-order*
- *TLE male patients*
- *GME patients on polytherapy with high dose.*

Recommendations

Recommendations

1. Traditionally, the ketogenic diet has been used in children < 2 years. This has been due to the belief that it is harder to initiate and mintain ketosis in very young children. Efficacy of the diet in young children was unknown. These data suggest that the diet can be successfuly initiated and maintained in children under 2, and that it is excedingly effective. **(Swink et al., 1997).**

2. In spite of adequate knowledge on epidemiology and diagnosis of epilepsy, school teachers have limited awareness of management and social issues. The need exists for adequate educationi on the subject. **(Kondylis, et al., 1997).**

3. Teachers communicate their lack of confidence with this issue and desire more information on it. Personal attitudes towards epileptic children appear good. However, epilepsy is still a mysterious illness for many teachers and the inclusion of this topic in their training is recommended. **(De Marco, et al., 1997).**

4. Systematic education of headmasters was not considered a useful way of promoting better KAP. **(Vrba, 1997).**

5. It is necessary to provide further educaitoin for teachers in order to enable them to treat epileptic children properly. They should treat them individually but without segregation. **(Gebauer, et al., 1997).**

6. it is possible to train staff in the mainstream educaitonal environment in the special health needs of children with unstable epilepsy, thereby supporting continued attendance at the school of choice. **(Lanfear, et al., 1997).**

7. The indications are that schools will be enabled to respond more effectively to support children with epilepsy. **(Settle, 1997).**

8. Support groups improved behaviour relationship between parents and children. **(Souza, et al., 1997).**

9. The role of language dysfunction in the school performance of epileptic children should be evaluated further. **(Papavasitiou, et al., 1997).**

10. Children with severe or recurrent febrile seizures showed intellectual and motor deficits compared to children with one seizure or to healthy controls. Our results emphasise the necessity of preventing further FC. **(Koelfen, et al., 1997).**

11. The majority of physicians used some form of written material to assist parents gain knowledge about the medications prescribed. The information varies, though there are no major discrpancies. This gives an opportunity to create a standard that meets the needs of the parents and physicians rather than adhere to an industry-based standard. **(Buckley, et al., 1997).**

12. As well known, insurance compaines tend to refuse to draw up health, life or accidents policies to people who, during their life, have had epileptic seizures.
This is possibly because risks of everyday life in people with epilepsy are not well known; therefore, the person with epilepsy lacks an adequate evaluation of the risk connected with his illness, and particularly, with this type of seizure, considering the different evaluation of the risk among insurance companies and clinical epileptologists: companies tend to overestimate the risk, epileptologists to understimate it. **(Cornaggia, 1997).**

13. Religions such as Islam provide instructions concerning every aspects of one's life which the believers are supposed to follow.

Muslims who live in non-Islamic countries are more likely to come into contact with medical questions which might be in contrast to their moral code. Therefore, in the Western countries where there are large communities of Muslims, neurologists should know some basic principle of Islamic medical ethics. (Vanzan, 1997).

14. *Most epileptic patients are able to control physiological parameters by intensive training and, as a result, improve clinically. Nevertheless, behavior therapy seem to have some influence on the amount of seizure reduction. (Uhlmann, et al., 1997).*

15. *The differentiation between epileptic and non-epileptic attack disorders (NAEDs) often requires the use of sophisticated diagnostic techniques such as long-term EEG-video monitoring. Further essential differentiation between psychological/psychiatric NAEDs and physiological/neurological NAEDs is however also required, but can often not be made with ictal EEG-video recording alone.*

16. *MRI, with optimising protocols, should be part of routine work-up in epileptic patients since it is an essential tool for the diagnosis of symptomatic epilepsy. (Consalvo et al., 1997).*

17. *There is convergence of severalmethods (ictal video-EEG, psychological and psychiatric MRI, SPECT, eventually SEEG) can minimise the probability of erroneous diagnosis. (Rektor et al., 1997).*

18. *A correct diagnosis of subtle epileptic seizure is needed for an adequate and sucessful treatment. (Derouaux et al., 1997).*

19. *Gestural automatism contralateral dystonic CLD and tonic CLT were seen mainly in Mesial TLE (MTLE) and head version in Neocortical Temporal Lobe NTLE. These ictal features can be useful in differentiating MTLE from NTLE. (Kim et al., 1997).*

References

References

Aarts J.H.P., Binnie C.D., Smit A.M. and Ilkins A.J. (1984);
> Selective cognitive impairment during focal and generalized epileptiform EEG activity. Brain, 107, p. 293-308.

Abdulghany M.O., Taman K.H., Bishry Z., Henfy K. (Ain Shams Univ., Cairo, Egypt). (1997);
> The Value of Electroencephalogrpahy in Prediction of Behavioural Changes in Epileptic children. Epilepsia, Vol. 38, Suppl. 3 p. 138.

Abou-Khalil B. (Vanderbilt Univ. Medical Center, Nashville, US).(1997):;
> Febrile Convulions Antecedent to Epilepsy and Family History of Febrile Convulsions and Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 122.

Acharya J., Dinner D.S., Chelune G.J., Marsilio D. (The Cleveland Clinic Foundation, US), (1997);
> Correlation of Verbal memory Change following Left Temporal Lobectomy with neuropsychology and the Intracarotid Amobarbital Procedure. Epilepsia, Vol. 38, Suppl. 3, P. 30

Adachi, Onuma, T., Muramatsu R., kato M., Ishida S. (National Leprasorium. Tama Zenshoen, Tokyo, Japan). (1997);
> Significance of the Factors Related to Epilepsy on Intelligence. Epilepsia, Vol. 38, Suppl. 3, P. 238.

Aikiä M., Kälviäinen R., Salmenperä T. Rikkinen P. (Dept. of Neurology, Juopio Univ. Hosp. and A.I. Virtanen Inst.,

Univ. of Kuopio, Finland). (1997);
Verbal memory in Newly Diagnosed and Chronic Epilepsy Epilepsia, Vol. 38, Suppl. 3, P. 126.

Aldenkamp A.P. and Vermeulen J., (1995):
Research on cognitive Effecs of Antiepileptic drugs Epilepsia. Vol. 36, Suppl. 3. P. 531.

Aldenkamp A.P., Alpherts W.C.J., Dekker M.J.A. and Overweg J., (1990):
Neuropsychological spects of learing Disabilities in Epilepsy.

Aldenkamp A.P., Diepman L.A.M. (Epilepsy Centre Kempenhaeghe, Heeze. The Netherlands.) (1997):
Factors Involved in Educational Retardation in Children with Epilepsy . Epilepsia, Vol. 38, Suppl. 3, P. 134.

Aleksic O., Radivojevic V., Krstic N., Ispanovic V. (Inst. for Mental Health, Belgrade, Yugoslavia.)(1997):
Subclinical Epileptiform Discharges in Cognitive and Behavioural Development. Epilepsia, Vol. 38, Suppl. 3, P. 50.

Andrewes, D.G., Tomilinson, L., Elwes, R.D.C. and Reynolds, E.H. (1984):
The influence of carbamazepine and phenytoin on memory and other aspects of cognitive function in new referrals with epilepsy, Acta Neurol Xcand., 69, 23-30.

Andrewes, D.G., Bullen, J.G., Tomlinson, L., Elwes, R.D.C. and Reynolds, E.H. (1986):
A comparative study of the cognitive effects of phenytoin and carbamazepine in new referrals with epilepsy, Epilepsia, 27,

18-134.

Arteaga Rodriguez C., Rodriguez Rivera L., Rojas Liranza Y. and
Vera Cuesta H. (Hospital Dr. Carlos J. Finlay, Havana,
Cubal) (1993):
Psychosocial Factors in Epilepsy Epilepsia, Vol. 36, Suppl 3,
P. S189.

Arts W.F., Geerts A., Johnson T., Brouwer O., Peters A., Stroink H.,
Van Donselaar C (Dept of Paediatric Neurology. Univ.
Hosp. Rotterdam, The Netherlands) (1997):
Prognosis of Childhood Epilepsy: Development of a Predictive
Model. Epilepsia, Vol. 38, Suppl. 3, P. 55.

Awaya Y., Hayashi K., Osawa M., Fukuyama Y. (Dept of Paediatrics,
Seibo International Catholic Hosp. Tokyo Women's
Medical College, Tokyo, Japan.)(1997);
Peculiar Types of Post-Encephalitic Epilepsy: A New
Syndrome. Epilepsia, Vol. 38, Suppl. 3, P. 5.

Badalian O.L., Avakian G.N., Nerobkova L.N., Markina N.V.,
Voronina T.A. Burd G.S. (Inst. of Pharmacology RAMS,
Russia.)(1997):
The Potentiation by Mexidol Anticonvulsive Effects of
Carbamazepine. Epilepsia, Vol. 38, Suppl. 3, P. 226.

Banks G.K., McCaughey L. R.G. Beran (Disability Resources Inc.,
Australia) (1997);
(The Sydney Inventory of Medication and Cognitive Impact
(SIMCI):An Outcome Measure of Clinical Drug Trials.
Epilepsia, Vol. 38, Suppl. 3, P. 61.

Barclay L. (Neurological Consulting Services, Tampa, US.)(1997);
Temporal Lobe Seizures and Cognitive Impaiment in Silicone
Toxocity. Epilepsia, Vol. 38, Suppl. 3, P. 6.

Beghi E., (Instituto Mario Negri, Milano, Italy) (1997);
Epilepsy and Every day Life-risks. Epilepsia, Vol. 38, Suppl.
3, P. 166.

Bell W.L., O'Donovan C.A., Sam M.C., Santos C.C., keyes J.W.,
Mathews V., Watson N. (Bowman Gray School of
Medicine, US.)(1997);
Comparison of MRI, Interictal SPECT, Ictal SPECT and PET
in Surgically-Proven Temporal Lobe Epilepsy. Epilepsia, Vol.
38, Suppl. 3, P. 8.

Berger A., Diener W., Schächtele M., Stephani U., Rating D. (Depts of
Neuropaediatrics, Univ. of Heidleberg. Freiburg, Kiel,
Stadt. Kinderklinik Karlsruhe, Germany.) (1997);
Complex Partial Seizures in Early Infancy with Benign
Outcome. Epilepsia, Vol. 38, Suppl. 3, P. 48.

Berg M.J., Erba G., Oestreich L., Burchfiel J.L. (Univ. of Rochester
Medical Centre. NY, US.)(1997);
New Onset Status Epilepticus with Bialteral Hippocampal and
Claustral Edema. Epilepsia, Vol. 38, Suppl. 3, P. 221.

Berkovic S.F. (Austin Repatriation Medical Centre, Australia.)(1997);
Familial Partial Epilepsia, Vol. 38, Suppl. 3, P. 168.

Bernhard J., Steinhoff, Karineususs, Cart D. Reimers, and Harald
Thegeder (Department of Clinical Neurophysiology and
Institute of Spors Science. Georg-August University of

Gottingen, Gottingen Germany) (1995):
(Physical Activity and Fitness of Epileptic Patients: A
Controlled Stdy). Epilepsia, Vol. 36, Suppl. 3, P. S183.

Besag F.M.C., Fowler M.F., Strang M., Pool F., Civers F. and Mills M.
(St Piers Lingfield, Lingfield, Surrey, U.K.) (1995):
Cognitive Slowing in Childre with Epilepsy. Epilepsia, Vol 36,
Supply. 3, p. S2.

Besana D., Cremonte M., Grillo E., Isocrono A., Rasmini P.
(Neuropsychiatric Univ, Italy.) (1997);
Ethiological Factors in Drug Resistant Epilepsies. Epilepsia,
Vol. 38, Suppl. 3, P. 4.

Betts, T.A. (1982):
Psychiatry and epilepsy: part one. In A Textbook of Epilepsy.
2nd editiaon pp. 227-270 (eds J. Laidlaw and A. Richens).
Churchill-Livingstone, Edinburgh.

Bhaucha N.E., Weiss M.G., Bharucha E.P., Jadhay S.S., Raven R.H.
Rajole B.K. (Bombay Hosp. Inst. of Medical Science,
India), (1997);
Socioculturl Aspects of Epilepsy in Developing Countries of
Asia. Epilepsia, Vol. 38, Suppl. 3, P. 166.

Bibileishvili S.H. (Inst. of Clinical Neurology, Georgia.) (1997):
Treatment of Epilepsy: Monotherapy or Polytherapy? Are We
Going Back? Epilepsia, Vol. 38, Suppl. 3, P. 82.

Binnie C.D., Channon S. and Marston D., (1990):
Learning Disabilities in Epilepsy: Neurophysiological Aspects
Epilepsia. 31 (Suppl. 4): S2.

Birnbaum A.K., Cloyd J.C., Nelson M.H., Remmel R.P. Rarick J.O., Leppik J.E. (1997);
Metabolism in Patients with Low Valproic Acid (VPA) Clearances.Epilepsia, Vol. 38, Suppl. 3, P. 45.

Blennow G., Heijbel J., Sandstedt P. and Tonnby B., (1990):
Discontinuation of Antiepileptic Drugs in Children who have Outgrown epilepsy: Effects on Cognitive Function. Epilepsia 31 (Suppl.4): S50.

Boglium G., Beghi E., Appolonio I., Spagnoli P. Fiordelli E. (Centro Epilepsia, Osp "S Gerardo", Monza, Italy). (1997):
Emotional Response to Stressful Events and Selected Psychopathology in Patients with Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 227.

Bolling T., Kramer A. and Haverkamp F. Universitatskinderklinik, Bonn, Germany, (1995):
Specific Cognitive Abilities in Epileptic Children in Department on Lateralized EEG Abnormalities: Evidence for a Higher Mental Vulnerability of the Left Hemisphere. Epilepsia, Vol. 36 Suppl. 3, P. S126.

Bornstein P.B. Hamilton S.B. and Bornstein M.T. (1986):
Self-monitoring procedures. In Handbook of Behavioural Assessment 2nd edition (eds. A.R. Ciminero, K.S. Calhoun and H.E. Adams), p. 176-222, John Wiley & Sons, New York.

Bourgeois, B.F.D., Prensky, A.L., Palkes, H.S., Talent, B.K. and Busch, S.G. (1983);
Intellegence in Epilepsy: a prospective stud inchildren. Ann. Neurol. 14, 438-444.

Bozdemir H., Sarica Y. (Cukurova Univ., School of Medicine, Dept. of Neurology, Adana, Turkey.) (1997):
The Efficacy of Carbamazepine and Sodium Valproate Monotherapies in partial Seizures. Epilepsia, Vol. 38. Suppl. 3, P. 67.

Bruhn, P. (1970):
Disturbance of vigilance in subcortical epilepsy. Acta Neurol. Scand., 46, 442-454.

Buckley D.J., Penney S. (Memorial Univ., Newfoundland, Canada.) (1997):
Written Information Regarding Antiepileptic Medication Given to Parents. A Canadian Perspective. Epilepsia, Vol. 38. Suppl. 3, P. 129.

Cakmak Ö., Baybas S., Kücükoglu H., Sözmen V., Aysal F., Atakli D. (Bakirkoy State Hosp. for Neurologic and Psychiatric Diseases, Istanbul Turkey.)(1997):
Prognostic Value of the Temporal Relationship between the First Seizure and Strok. Epilepsia, Vol. 38, Suppl. 3, P. 16.

Callenbach PMC, Geerts A.T., Arts W.F.M., Van Donselaar CA, Peters ACB, Stroink H., Brouwer O.F. (Dept of Neurology, Leiden Univ. Hosp., The Netherlands)(1997):
Familial Occurrence of Epilepsy in Children with Newly Diagnosed Seizures.Epilepsia, Vol. 38, Suppl. 3, P. 202.

Canadian Study Group for Childhood Epilepsy (IWK-Grace Health Centre, Alberta Children Hosp. Chedoke-McMaster Hosp. Canada) (1997):

Bozdemir H., Sarica Y. (Cukurova Univ., School of Medicine, Dept. of
Neurology, Adana, Turkey.) (1997):
The Efficacy of Carbamazepine and Sodium Valproate
Monotherapies in partial Seizures. Epilepsia, Vol. 38. Suppl. 3,
P. 67.

Bruhn, P. (1970):
Disturbance of vigilance in subcortical epilepsy. Acta Neurol.
Scand., 46, 442-454.

Buckley D.J., Penney S. (Memorial Univ., Newfoundland, Canada.)
(1997):
Written Information Regarding Antiepileptic Medication Given
to Parents. A Canadian Perspective. Epilepsia, Vol. 38. Suppl.
3, P. 129.

Cakmak Ö., Baybas S., Kücükoglu H., Sözmen V., Aysal F., Atakli D.
(Bakirkoy State Hosp. for Neurologic and Psychiatric
Diseases, Istanbul Turkey.)(1997):
Prognostic Value of the Temporal Relationship between the
First Seizure and Strok. Epilepsia, Vol. 38, Suppl. 3, P. 16.

Callenbach PMC, Geerts A.T., Arts W.F.M., Van Donselaar CA,
Peters ACB, Stroink H., Brouwer O.F. (Dept of Neurology,
Leiden Univ. Hosp., The Netherlands)(1997):
Familial Occurrence of Epilepsy in Children with Newly
Diagnosed Seizures.Epilepsia, Vol. 38, Suppl. 3, P. 202.

Canadian Study Group for Childhood Epilepsy (IWK-Grace Health
Centre, Alberta Children Hosp. Chedoke-McMaster Hosp.
Canada) (1997):

Comparison of the Cognitive Effects of Clobazam and Standard Monotherapy. Epilepsia, Vol. 38, Suppl. 3, P. 129.

Carton S., Coles A.J., Thompson P.J., Duncan J.S., (National Hosp. Neurology and Neurosurgery, London, England.)(1997);
Factors Related to Prognosis in Non-Epileptic Seizures. Epilepsia, Vol. 38, Suppl. 3, P. 242.

Cerullo A., Tinuper P., Riva R., Marini C., Rosati A., Avoni P., Baruzzi A. (Inst. of Clinical Neurology, Bologna, Italy) (1997);
Factors Influencing Prognosis in Partial Epilepsies, A Retrospective Study on 176 Patients. Epilepsia, Vol. 38, Suppl. 3, P. 49.

Chan B.S., lee M.A., Le Blanc F.E., Rewcastle N.B. (Univ. of Calgary, Canada.) (1997);
A Histopathology/Outcome Study from 103 Temporal Lobectomies for intractable Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 2.

Cheroiu I., Buraga I., Panea C., Buraga M., Iancu A. (Univ. Hosp. Bucharest, Romania) (1997);
Epileptic Seizures Associated with CNS Determination of Systemic Vasculitides.Epilepsia, Vol. 38, Suppl. 3, P. 142.

Chez M.G., Buchanon K. (Rush Medical School. Chicago, US.) (1997);
Age of Regression of language and Behaviour in Children Referred for Pervasive Developmental Delay Correlate to severity of Epileptiform Activity on 24 hours. EEG. Vol. 38, Suppl. 3, p. 50.

Cimiero A.R., Calhoun D.S. and Adams H.E. (1986):
> Handbook of Behavioural Assessment, 2nd edition. John Wiley & Sons. New York.

Clarke E.J., Jawad S.S.M. (Rhondda Neuropsych & Epilepsy Centre, Wales, UK.) (1997):
> Epilepsy and Psychosis: New Dimensions in Classification. Epilepsia, Vol. 38, Suppl. 3, P. 238.

Collinge H. (David Lewis Centre, Cheshire, U.K.) (1997);
> Anxiety or Epilepsy? A Case Study. Epilepsia, Vol., 38, Suppl., 3, P. 45.

Colin, D. Binnie (The Maudsley Aosprlae, London) (1988);
> Seizures, EEG, discharges and Cognition. Epilepsy, Behaviour and Cognitive Functiaon. P 48-66.

Conry J., Coffman C., Glass P. (Dept. of Neurology, Children"s National Medical Centre, Washington DC, Centre for Child Development and Neonatology, Washington, DC, US.) (1997):
> Incidence of epilepsy at 5 years of Age in Neonates Treated with ECMO. Vol., 38, Suppl., 3, P. 232.

Consalvo D., Silva W., Rugilo C., Salgado P., Centurion E., Saidon P., Schuster G., Komen S. (Epilepsy Centre. Neurology Division. Ramos Mesa Hosp., Femien Foundation, Conicet, Argentina.) (1997);
> MRI Findings in Output Epileptic Patients. Vol., 38, Suppl., 3, P. 9.

Cornaggia C.M. (Inst. of Psychiatry Clinic. Ospedale Policlinico,

Milano, Italy) (1997):
Attitudes of Insurance Companies towards People with Epilepy.Vol., 38, Suppl., 3, P. 167.

Czapinska E., Czapinski P., Terczynski A. (Polish-American Children's Hospital, Neurology Clinic, Collegium Medicum, Jagiellonian Univ., Krakow, Poland.) (1997);
Evaluation of Cognitive Functions and Adverse Effects in the Course of 36 month Monotherapy Employing Carbamazepine, Valproic Acid, Phenytoin or Pheno-barbital in Newly Diagosed Patients with Partial Complex Epileptic Siezures. Vol., 38, Suppl., 3, P. 58.

Czapinski P., Terczynski A., Czapinska E. (Neurology Clinic, Polish-American Children's Hosp., Collegium Medicum, Jagiellonian Univ., Poland) (1997);
Randomised 36-month Comparative Study of Valproic Acid, Phenytoin, Phenobarbital and Carbamazepine Efficacy in Patients with Newly Dignosed Epilepsy with Partial Complex Seizures. Vol., 38, Suppl., 3, P. 42.

Czapinski P., Terczynski A., Czapinska E. (Neurology Clinic. Polish-American Children's Hosp., Collegium medicum. Jagiellonian Univ., Pland) (1997);
Open Randomised Comparaive Study of Vigabatrin (VGB) and Lamotrigine (LTG) Efficacy in Monotherapy of patients with Drug Resistant Epilepsy with Partial Complex Seizures Resistant to Carbamazepine. Vol., 38, Suppl., 3, P. 35.

Dana Siskova, Jih Dolansky and jan Hadac (Thomayer's Hospital, Prague, Czech Republic), (1995);
Efficacy of Sabrii in Treatment of Epilepsy in Children.

Epilepsia, Vol. 36, Suppl. 3, P. 104. Epilepsia, 21; 123-135.

Dam M., (1990);
Children with Epilepsy: The effect of Seizures, Syndromes, and Etiological Factors on Cognitive Functioning. Epilepsia, 31 (Suppl. 4): S26.

De Boer H.M. (Heemstede, The Netherlands) (1997):
Culture, Gender and Eilepsy, Epilepsia. Vol. Suppl. 3, P. 52.

Deckers CLP, Hekster Y.A., Keyser A., Meinardi H. Renier W.D. (Inst. of Neurology, Univ. Hosp. Nijmegen, The Netherlands), (1997);
Monitoring Adverse Effects in Antiepileptic Drug Therapy: Wait and see or go for it?, Epilepsia, Vol. 38, Suppl. 3, P. 82.

De Feo M.R., Mecarelli O., Marciani M.G., Striano S., Ortenzi A., Aloisi P., D'Alessandro P. and M. Onofri (Department of Neurophysiopathology, Univelrsity "LA Sapienza," Roma, Italy), (1995);
Vigabatrin Monotherapy in Newly Diagnosed partial Epilepsy: An Open Multicenter Study. Epilepsia. Vol. 36, Suppl. 3, P. 105.

М
Delaney R.C., Rosen, R.J., mattson, R.H. and Novelly, R.A. (1980):
Memory function in focal epilepsy. A comparison of non-surgical unilateral temporal lobe and frontal lobe samples. Cortex. 16, 103-117.

Dennis B. Smith (Comprehensive Epilepsy Program of Oregon, Good Samaritan Hospital and Medical Center, Oregon, USA), (1988):

Anticonvulsants, seizures and performance: The Veteran's Adminstration experience. Via, Epilepsy, Behaviour and Cognitive Function. P 67-78.

De Marco, P., Calzolari S., Rozza L., Rauzi P.G., Bertamini C. (Hosp. 'S. Chiara'. Trento, Italy) (1997);
Epilepsy and School Teachers. Epilepsia, Vol. 38, Suppl. 3, P. 112.

Derambure Ph., Josien E., Dujardin K., Sauvage M.C., Sediri H., Jacquesson J.M. and Guieu J.D. (1995);
Characterization of Partial Epilepsy by Neurophysiological and Neuropsychological Evaluation. Epilepsia. Vol. 36, Suppl. 3, P. S76.

Dericioglu N., Saygi S., Ciger A. (1997);
The Value of Provocation Method in Patients with the Diagnosis of Probably Psychogenic Seizures. Epilepsia, Vol. 38, Suppl. 3, P. 16.

Diener W., Herold T., Mayer H., Bengel G., Berger A., Bruhl J., Dittrich C., Hoffmann-Riem M., Kretzer S., Kriebel U., Leineweber B., Löffler H., GroB-Selbeck G., Korinthenberg R., Schneble H., Stephani U., Rating D. (Dept. of Neuropaediatrics. Freiburg. Germany) (1997);
Paediatric Epilepsy Syndromes and Seizure Relapse during and after Drug Withdrawal. A Retrospective Multicentre Study. Epilepsia, Vol. 38, Suppl. 3, P. 55.

Dikmen S. (1980):
Neuropsychological aspects of epilepsy. In A Multidisciplinary Handbook of Epilepsy (ed. B.P. Herman).

Charles C. Thomas, Springfield. Illinois.

Dodd P., Redmond J. (St., James's Hosp. Dublin, Ireland) (1997):
Epilepsy and Changes in Behaviour. Epilepsia, Vol. 38, Suppl. 3, P. 90.

Dodrill C.B. and Troupin, A.S. (1977):
Psychotropic effects of carbamazepine in epilepsy. A double-blind comparison with phenytoin. Neurology, 27, 1023-1028.

Dodrill C.B., Arnett J.L., Sommerville K. and Mengel H. (1995);
Triagabine. Epilepsia, Vol. 36, Suppl. 3, P. S31.

Dodrill C.B. and Batzel L.W. (1986):
Interictal behavioural features of patients with epilepsy. Epilepsia, 27 (Suppl. 2), P. 564-576.

Dodrill C.B., Batzel L.W., Queisser H.R. and Temkin N.R. (1980):
An objective method for assessment of psychological and social problems among epileptics.

Dulac O. (1995):
Choice of Antiepileptic Drugs in Childhood. Epilepsia, Vol. 36, Suppl. 3, S90.

Dunne J.W., Heye N., Hart M.B., Field C.M., Lawson N.E. (Royal Perth Hosp., Australia)(1997);
Non-convulsive Status Epilepticus. A prosɼectie Study of 102 Patients. Epilepsia, Vol. 38, Suppl. 3, P. 49.

Echenne B., Rivier F., Roubertic A., Touzery A., Humbertclaude V.

(Service de Neuropediatric. Hôpital Saint Eloi, Montpellier. France.) (1997):
Simultaneous Occurrence of Beign Partial Epilepsy and Childhood Absence Epilepsy: Report of 2 Cases in 2 Families. Epilepsy, Vol. 38, Suppl. 3, P. 120.

Elger C.E., Grunwald Th., Lehnertz K., Helmstadter C. and Heinze H.J. (Department of Epileptology, University of Bonn Medical Center, Bonn, Germany). (1995);
Assessment of Pre- and Postoperative Verbal memory by Intracranial Cognitive Potentials in Temporal Lobe Epilepsy). Epilepsia. Vol. 36, Suppl. 3, P. 92.

Elia M., Musumeci S.A., Ferri R., Castano A., Azan G. and Stefanini MC. (1995);
Usual EEG Features in a Child with Cerebrocerebellar Lissencephaly). Epilepsia, Vol. 36, Suppl. 3, P. S78.

El-Kholi Susan (1985):
Attention in psychiatric patients, a psychometric study, MA. Thesis Cairo University.

Ellenberg, J.H., Hirtz, D.G. and Nelson, K.B. (1984);
Do seizures in children cause intellectual deteruration. Engl. J. med., 314, 1085-1088.

El-Radhi A.S.(Paediatric Dept. Queen Mary's Sideup NHS Trust. Kent. UK.) (1997);
Influence of Body Temperature on Subsequent Convulsions in Children Following the Initial Febrile Convulsion. Epilepsia, Vol. 38, Suppl. 3, P. 122.

Euan, M. Ross and Pat Tookey. Charing Cross Hospital and Institute
of Child Health, London, (1988);
Educational needs and Epilepsy in childhood via Epilepsy,
Behaviour and Cognitive Function, P. 87-95.

Fedio P. (1986);
Behavioural characteristics of temporal lobe epilepsy.
Psychiatric. Am. North Am., 283-292.

Fenton G.W., (1981);
Psychiatric disorders of Epilepsy: Classification and
Phenomenology. In Epilepsy and Psychiatry 9eds E.H.
Reynolds and M.R. Trimble). P. 12-26. Churchill-Livingstone.
New York.

Fenwick P., (1986):
Aggression and epilepsy. In Aspects of Epilepsy and
Psychiatry (eds M.R. Trimble and T.G. Bolwig). P. 31-57.
John Wiley & Sons. Chichester.

Fisher J.H., Andrews C.O., Fischer P.A., Luer M.S., Charbel F.T.
(Univ. of Illinois at Chicago, US.) (1997);
Diurnal Variations in the Pharmacokinetics of Gabapentin.
Epilepsia, Vol. 38, Suppl. 3, P. 33.

Fitzpatrick D., Kelly F., Liber C. (Mandsley Hosp., London, UK.)
(1997):
Personality and Non Epileptic Seizures. Epilepsia, Vol. 38,
Suppl. 3, P. 237.

Fitzpatrick D., Kelly F. (Maudsley Hosp., London UK.) (1997):
Epilepsy and personality Disorders. Epilepsy, Vol. 38, Suppl.

3, P. 24 & 141.

Frank Duffy H. and Cesare Lombroso T., (1995);
Dipole Source Localization in Epilepsy. Epilepsia, Vol. 36, Suppl. 3, P. S83.

Froescher W. (Dept. of Neurology and Epileptology, Weissenau, Germany.) (1997);
Choreiform Hyperkinesias Induced by Phenobarbital Withdrawal. Epilepsia, Vol. 38, Suppl. 3, P. 39

Gary Hadfield M., Fortner-Burton C.A., DeLorenzo R.J. (Medical College of Virginia/Virginia Commonwealth Univ., US.) (1997);
Status Epilepticus: Neuropathology by Seizure Type. Epilepsia, Vol. 38, Suppl. 3, P.2

Gatzonis S., Spanaki M., Kolindou A., Mandouvalos V., Kostamis P. and Papageorgiou C. (Neurological Department of Athens National University, Athens, Greece), (1995);
Comparative Study of SPECT, CT. MRI, and Long-Term Video EEG in Epileptic patients. P. S133.

Gebauer K., Kelehen A., Bozie K. Sekulic S., Vlajkovic K. (Inst. of Neurology,Psychiatry and Mental Health, Novi Sad. Yugoslavia) (1997):
Teachers' Attitudes to Epileptic Children. Epilepsia, Vol. 38, Suppl. 3, P. 112.

Geladze T. (Epilepsy Centre, Inst. of Neurology, Rep. of Georgia) (1997):
Management of Clinical-EEG; Side Effects of Carbamazepine

(CBZ). Epilepsy, Vol. 38, Suppl. 3, P. 70.

Ghepelli J.L.D., Guerrerio M.M., Rotta N.T., Costa J.C., de Manreza M.L.G., Reed U.C., Silva E.A., Nunes M.L. and Diament A., (University of Sao Paulo, Brazil), (1995):
The Brazilian Multicenter Study. (Vigabatrin in Refractory Childhood Epilepsy). Epilepsia, Vol. 36, Suppl. 3, p. S102.

Gibbon F.M., Wallace S.J. (Univ. Hosp. of Wales, UK), (1997):
Lamotrigine in Severe Infantile Epilepsies). Epilepsia, Vol. 38, Suppl. 3, P. 33.

Gibbs F.A., Lennox W.G. and Gibs E.I., (1936):
The electroencephalogram in diagnosis and in location of epileptic seizures. Archiv. Neurol. Psychiatry, 36, 1225-1235.

Glowinski, H. (1973):
Cognitive deficits in temporal lobe epilepsy. An investigation of memory functioning. J. Nerv. Ment. Dis., 157. 129-137.

Gobbi G., Bertani G., Pini A. Guerrini R., Tiberti A., Valseriati D., Martinelli O., Menegati E., Besama D., Rasmini P., Veggiotti P., Resi S., Lanzi G., Galeone D., Capovilla G. and Milani S. (Department of Neuropediatrics of Reggii Emilia, Pisa, Brescia, Alessandria, Pavia, Bari, Mantova and Department of Health and Biostatistics, Pisa, Italy). (1995):
(First-Line Vigabatrin Monotherapy in Childhood Partial Epilepsy). Epilepsia, Vol. 36, Suppl. 3, P. 102.

Gonzalez-Astiazaran A., Ruiz-Garcia M., Herrera Mora P., Ramfrez-Campos J., Irigoyen maldonado C., and Ceja

Meoreno H., (1995);
(Continuous infsion of Diazepam in Refractory Status Epilepticus in Childhood). Epilepsia, Vol. 36, Suppl. 3, P. S70-S1.

Gowers, W.R. (1881);
Epilepsy and Other Chronic Convulsive Diseases. Churchill, London.

Guo X., Yin J., Zhou S., (Dept of Neurology, Psychiatric Hosp. of Henan, P.R. China), (1997);
Cognitive Effects of Phenytoin and Carbamazepine in New Epilestic Children. Epilepsia, Vol. 38, Suppl. 3, P. 135.

Gus A. Baker, Ann Jacoby, and David W. Chadwick (Department of Neuroscience; and Centre for Health Services Research, Newcastle Upon Tyne, U.K.), (1995):
Psychosocial Findings of Cross-Sectional Commnity Study of People with Epilepsy. Epilepsia, Vol. 36, Suppl., 3, S185, P. 185.

De Graaf E.H., Kuyk J., Siffels M.C., Muller J.V., Zitman F.G., Spinhoven Ph., Swinkels M. (Epilepsy Centre, Heemstede, The netherlands) (1997):
Epilepsy and Psychopathology, Epilepsia, Vol. 38, Suppl. 3, P. 238.

Grunwald Th., Eler C.E., Lehnertz K., Beck H., and Heinze H.J., (1995):
(Material-Specific Alernations in Hippocampal Cognitive potentials in Temporal Lobe Epilepsy). Epilepsia, Vol. 36, Suppl. 3, P. S84.

Harry McConnell (Behavioral Epilepsy, medical College of Pennsylvania, Allegheny General Hospital, Pittsburgh, PA, U.S.A.). (1995):
Behavioral Effects of Newer Anticonvulsants) Epilepsia, Vol. 36, Suppl. 3, P. 128.

Halstead, H. (1957):
Abilities and behaviour of epileptic children. J. Ment.f Sci., 103. 28-47.

Hashem S., Hamdy S.M., Fahmy E. (Cairo Univ., Egypt), (1997);
The Effects of Sodium Vlproate on Man and White Albino Rats, Epilepsia, Vol. 38, Suppl. 3, P. 42.

Hauser W.A., (Columbia Univ., New York, US), (1997);
Mortality in Patients with Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 168.

Hendriks M., Duits P., Aldenkamp A.P. and Van der Vlugt H. (Dr. Hans Berger Clinc, Breda, The Netherlands), (1995):
(Memory Functions in Patients with Primary Generalized Sizures and Partial Seizures Originating from the left to Right Temporal Lobes). Epilepsia. Vol. 36, Suppl. 3, P. S125.

Hendriks M., Van Bronsyk K.L., Van Luytelaar ELJM, Aldenkamp A. (Dr Hans Berger Clinic, Netherlands) (1997):
Verbal Episodic and Semantic memory of Patients with left or Right Temporal Lobe Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 136.

Henriksen O., (1990):
Education and Epilepsy: Assessment and Remediation.

Epilepsia, 31 (Suppl. 4): S21.

Hermann B.P and Whitman S., (1986):
Psychopathology in Epilepsy: a multiaetiological model. In psychopathology in Epilepsy Social Dimensions (eds S. Whitman and B.P. Hermann). PP. 5-37. Oxford University Press, New York.

Hirtz, D.G. and Nelson, K.B. (1985):
Cognitive effects of antiepileptic drugs. In Recent Advances in Epileptology (ed. B.S. Meldrum), pp. 161-181,. Churchill, Edinburgh.

Herraz J.L., Camos J., Casas C., Nieto M., Arteaga R., Careaga J., Rodriguez-costa T. and Rufo M (Neuropediatrics Units, Hospital Valdecilla, University Santander; and Madrid, Murica and Sevilla, Spain). (1995):
Spanish Experience with Efficacy and Tolerability of Vigabatrin in 1997 Children with Refractory Epilepsy. Epilepsia, Vol. 36, Suppl. 3, P. S 103.

Hilfiker P. Mothersill I., and Kramer G., (1995):
Properties of Rhythmical activity in Partial Seizures Determined by a Computerized Recognition, Epilepsia, Vol. 36, Suppl. 3, P. S84.

Hirtz, D.G. and Nelson, K.B. 91985):
Cognitive effects of antiepileptic drugs. In Recent Advances in Epileptology (ed. B.S. Meldrum), pp. 161-181. Churchill, Edinburgh.

Ho A., Connolly M., Thornton N., Farrell K., (Neurosciences

Programme, British Columbia's Children's Hosp., Vancouver, Canada), (1997):
Assessment and Treatment of Non-Epileptic seizures in Children. Epilepsia, Vol. 38, Suppl. 3, P. 124.

Hoare P. (Royal Hosp. for Sick Cildren, Edinburgh, Scotland), (1997):
Self-esteem and Behaviour Adjustment in children with Epilepsy and Children with Diabetes. Epilepsia, Vol. 38, Suppl. 3, P. 170.

Hofmann W., Spreng T. (Neurological Clinic. Halle Saale, Germany), (1997):
Changes of EEG Activity due to Antiepileptic Treatment. Epilepsia, Vol. 38, Suppl. 3, P. 62.

Hooge R.D., Raes A., Van de Vijver G., Franck F., Van Boga and De Deyn P.P. (Laboratory of Neurochemistry and Behaviour, Born-Bunge Foundation, Dept. Neurology-Memory Clinic, Middelheim Hosp., Univ. of Antwerp (UIA). Laboratory of Electrobiology, born-Bunge Foundation, Univ. of Antwerp (RUCA), Belgium), (1997):
Anticonvulsant and Adverse Effects of the Orally Active NMDA Antagonist 4-mehyl-APPA and its carboxyethylester. Epilepsia, Vol. 38, Suppl. 3, P. 36.

Hutt. S.J., Newton J., and Fairweather, (1977):
Choice reaction time and EEG acivity in children with Epilepsy. Neutropsychologia. 15, 257-267.

Ischijoh S., (1995):
Midline Spikes Associated with Temporal Spikes in Childhood EEGs. Epilepsia, Vol. 36, Suppl. 3, P. S78.

Ignatowicz R., Chmielik J., Michalowicz R., Ignatowicz L., Jozwiak S. and karkowska B. (Department of Neurology, Child's Health Centre, Warsaw, Poland), (1995):
Sabril (Vigabatrin) Efficacy in Therapy of Epilepsy in children. Epilepsia, Vol. 36, Suppl. 3, P. S103.

Ignatowicz L., Ignatowicz R., Jozwaik S., Michalowicz R., Chmielik J. and Kmiee T. (Department of Neurology, Child's Health Centre, Warsaw, Poland), (1995):
Psychiatric Aspects of Epilepsy in Childhood Treated with Tegretol CR (Ciba-Geigy). Epilepsia, Vol. 36, Suppl. 3, P. 128-129.

Inami M., Oguchi T., Takahashi A., Hayama T., Shinkai Y., Murasaki M., (Dept. of Psychiatry, School of Med., Kitasato Univ., Japan), (1997):
Evaluation of the Psychological Symptoms on the Epileptic patients with MMPL-2. Epilepsia, Vol. 38, Suppl. 3, P. 240.

Ingrid Scheffer Hilary Phillips E., John Mully, Grant Sutherland, Simon Harvey A., Ian Hopkins J. and Samuel F. Berkovic, (1995):
Autosomal Dominant partial Epilepsy with Variable Foci is Not Allelic with Autosomal Dominant Nocturnal Frontal Lobe Epilepsy. Epilepsia, Vol. 36, Sppl. 3, P. S28, Faculty of Art. Supervised by M Soueif & A. Okasha, P. 51, 53.

Ioan Buraga and Ioana Cheroiu, (1995):
Clinical Study of Valproate Monotherapy in Newly Diagnosed Epileptic Patients. Epilepsia, Vol. 36. Suppl. 3, P. S66.

Izmeth A (Hesketh-Centre, Southport, UK) (1997):

A Study of Topirarmate in patients with learning Disability and Refractory partial Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 37.

Jinggang Yin, Susun Zhou Lixia Gao and Jinchuan Wang, (1995) :
(Comparative Study of the Cognitive Effects of Phenytoin and Carbmazepine in New Epileptic patients). Epilepsia. Vol. 36, Suppl. 3, P. S69.

Joanne Dahl, (1995):
(Physchological Factor Influencing Epileptic Seizures). Epilepsia, Vol. 36, Suppl. 3, P. S25.

Job A., Collings, Gus Bader A., Susan Tedman, Gary Banks and Michael Hills (Leeds Metropolitan University and Department of Neuroscience. The Walton Centre, Liverpool, England. (1995):
Cognitive Mediators of Psychiatric Morbidity in Epilepsy. P. 149.

Joels M., Karst H., Wadman W.J., and De Kloet E.R. (Inst. Neurobiology, Univ. of Amsterdam, and LACR, Leiden Univ., The Netherlands), (1997):
Steroid, Calcium and Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 52.

John Austin K. and David Austin R. (Indiana University School of Nursing, Bloomington, IN, U.S.A.) (1995);
Family Activity, Supervision and Child Activity in New-Onset Epilepsy. Epilepsia. Vol. 36, Suppl. 3, P. S171.

John L., Arnett and Carl B. Dodrill, (1995);

Effects of Gabapentin (GBP, Neurontin) on Cognitive Functioning and Mood. Epilepsia, Vol. 36, Suppl. 3, P. S32.

Johnson M.C., Thomas E.M., (Manchester Metropolitan Univ., UK), (1997):
Parents Experiences with Medical and Educational Professionals during the Management of Epilepsy in Children. Epilepsia, Vol. 38, Suppl. 3, P. 170.

Jolanta Ossein (Institute of Psychiatry, London), (1988):
Metods and problems in the assessment of cognitive function in epileptic patients. Via, Epilepsy, Behaviour and Cognitive Function. P 9-28.

Jongsma M.J., Mommers R. (Inst. Voor Epilepsiebestrijding, Heemstede. Academic Hosp. Nijmegen, The Netherlands), (1997):
Outcome of Psychogenic, Non-epileptic Seizures (a follow-up of an inpatient population). Epilepsia, Vol. 38, Suppl. 3, P. 237.

Jorge C.L., Yacubian E.M.T. Valerio R.M., Buchpiguel C., Dantas F. (Hosp. das Clinics da Facsldade de Medicina da Univ. de Sao Paulo, Brazil), (1997):
Ictal Single Photon Emission Computed Tomography in Extratemporal Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 9.

Kasteleun-Nolst Trenite, D.G.A. Riemersma, J.B.J., Binnie, C.D., Smit, A.M. and Meinardi, H. (1987):
The influence of subclinical epileptiform EEG discharges on driving behaviour. Electroencephal. Clin. Neurophysiol., 67, 167-170.

Kate Fuggle, Angela Fixter, and S.W. Brown, (1995):
>   Music Therapy in the Treatment of a Child with Epilepsy and
>   Autism. Epilepsia. Vol. 36; Suppl. 3, P. S74.

Katharine Rooth and Tim Betts (Brimingham University, Brimingham, U.K.) (1995):
>   (Who is in charge of My Epilepsy? Dialogue Between Patient
>   and Physician During Behavioural Treatment of Epileptic
>   Seizures. Issues of Patient Empowerment, Control and
>   Physician Anxiety. Epilepsia, Vol. 36, Suppl. 3, P. S129.

Kaufmann K.R., (UMDNJ-Robert Wood Johnson Med. School, New Branswick, US), (1997);
>   Carbamazepine, Hepatotoxicity, Organic Solvents and Paints.
>   Epilepsia, Vol. 38, Suppl. 3, P. 29.

Kerdar M.S., Ebner A. (Epilepsy Centre. Bethel, Germany) (1997):
>   Prevalence of Olfactory / Gustatory Aura in Patients with
>   Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 227.

Keith H.M., Ewert, J.C. Green, M.W. and Gage, R.P. (1955):
>   Mental status of children with convulsive disorders. neurology,
>   5, 419-425.

Kieslich M., Ehlers S., Zippel S., Schwabe D., Jacobi G., (Dept Paediatic Neurology, Univ. Frankfurt/Main, Germany) (1997):
>   Intractable Seizures and progressive Cerebraltrophy in
>   Erythrophagocytic Lymphohistiocytosis. Epilepsia, Vol. 38,
>   Suppl. 3, P. 1.

Kilpatrick C. Murri, V., Cook M., Andrewes D., Desmond P. and

Hopper J. (Royal Melbourne Hospital, Parkville, Victoria, Australia), (1995):
Severity of Verbal Memory Deficits Correlates with Degree of Left Hippocampal Atrophy. Epilepsia, Vol. 36, Suppl. 3, S2.

Kimford J. Meador (Medical College of Georgia, Augesta, GA, U.S.A.)(1995):
(Vigabatrin). Epilepsia, Vol. 36, Suppl. 3, P. S31.

Kim W.J., Lee J.H., Kim J.Y., Park S.C., Lee B.I. Kim D.I. (Depts of Neurology and Neuroradiology, College of medicine, Yonsei Univ., South Korea), (1997):
The Clinical Prognosis of Patients with mesial Temporal Sclerosis in RI. Epilepsia, Vol. 38, Suppl. 3, P. 10.

Kim Y., Bong Hong S. (Samsung medical Centre, Korea) (1997):
(Ictal Features Differentiating mesial from Neocortical Temporal Lobe Epilepsies). Epilepsia, Vol. 36, Suppl. 3, P. 18.

Knowlton, R. Rowley H., Wong S., Roberts T. and Laxer K., (1995):
Registration of Magnetoencephalography and High-Resolution Position Emission Temography with Magetic Resonance Imaging in the Evaluation of Nonlesional Partial Epilepsy. Epilepsia, Vol. 36, Suppl. 3, P. 83.

Kochen S., Giagante B., Oddo S., Saidon P., Centurion E., Consalvo D., Silva N. (Epilepsy Center, Neurology Division, Ranos Media Hosp., Argentina), (1997):
Spike and Wave complexes and Exacerbation o eizures by Carbamazepine. Epilepsia, Vol. 38, Suppl. 3, P. 75.

Koelfen W., Pehle K., Runde J., Koning S., Schultze C (Pediatric Hosp.

Univ. Manheim, Germany), (1997):
Is the Long-Term Follow-Up of Children with Febrile Convulsions Favourable" Epilepsia, Vol. 38, Suppl. 3, P. 121.

Koul R.L., Aithala G.R., Joshi R.M. and Elbualy M.S. (Sultan Qaboos University. Al Khod, Sultanate of Oman), (1995):
Vigabatrin as Add-on Therapy in Severaly Intractable Seizures in Children in Oman. Epilepsia Vol. 36, Sppl. 3, P. S103.

Kowalik A., Elger C.E., Zentner J. and Schram J., (Department of Epileptology and Neurosurgery. University of Bonn, Bonn, Germany), (1995):
(Behavioural Disturbances in Children with Temporal Lobe Epilepsy). Epilepsia, Vol. 36, Suppl. 3, P. 177.

Koyanagi M. (Tokyo Univ. School of Medicine, Japan), (1997):
(Ictal or Non-ictal Psychiatric Symptoms in Epilepsy) Epilepsia, Vol. 38, Suppl. 3, P. 127.

Krawetz P., Fleisher W., Pillay N., Arnett J., Staley D., Maher J. (Univ of Manitoba, Canada), (1997):
Family Functioning in Patients with pseudoseizures and Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 242.

Kucukali I., Atakli D., Hanogl L., Dogu O., Arpaci B., Kiirbas D., Baybas S., (KBakirkoy State Hosp. for Psychiatric and Neurological Diseases, Dept. of Neurology, Istanbul, Turkey), (1997):
Influence of Cognitive Testing on he Epileptic Focus. Epilepsia, Vol. 38, Suppl. 3, P. 239.

Kudo T., Hunakoshi A., Kageyama M., Homada K., Nomari Y., Tagi

K., (National Epilepsy Centre, Shizuolo Sp., japan), (1997):
Posictal Dysfunction of Verbal Memory in patients with
Temporal Lobe Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 126.

Kugoh T., Hosokawa K. (Department of Neuropsychiatry, Kagawa
Medical School, Kagawa, Japan), (1995):
Topiramate as Add-On Open Therapy in Patient with Partial
Seizures. Epilepsia, Vol. 36, Suppl. 3, P. S151.

Kurihara M., Kumagai K., Noda Y. (The Kanagawa Rehabilitation
Center, Japan), (1997):
Status Epilepticus in Patients with Severe Motor and
Intellectual Disabilities. Epilepsia, Vol. 38, Suppl. 3, P. 6.

Lanfear J.H., Rashid C., (De Montfort Univ. and Fosse Health
Community Trust, UK), (1997):
Who Cares for the Student with Unstable Epilepsy in
Mainstream Education? Epilepsia, Vol. 38, Suppl. 3, P. 113.

Ladavas, E., Umilta, C. and Provinciali, L. (1979):
Hemisphere-dependent cognitive performances in epileptic
patients. Epilepsia, 20, 493-502.

Lee B.J., Lee S.J., Park S.C. (Dept of Neurology, Yonsei Univ., College
of Medicine, Korea), (1997):
Clinical Characteristics and Prognosis of Temporal Lobe
Epilepsy According to the Frequency of Secondarily
Generalised Seizures. Epilepsia, Vol. 38, Suppl. 3, P. 62.

Lehnertz K., Elger C.E. (Clinic of Epileptology, Univ., Bonn,
Germany), (1997):
Inverse Reltionship between CBZ blood Level and Neuronal

Complexity Loss in the Epileptogenic Focus of Patients with Temporal Lobe Epilepsy. Epilepsia, Vol. 36. Suppl. 3, P. 30.

Lendt M., Helmstaedter C., and Elger C.E. (University Clinic of Epileptology, Bonn, Germany), (1995):
Pre- and Postoperative Neuropsychological Profiles in Children and Adolescents with Temporal Lobe Epilepsy. Epilepsia, Vol. 36, Suppl. 3, P. 93.

Leonard G., Andermann E., Ptito A., Schopflocher C. (Montreal Neurological Inst., Canada), (1997):
Cognitive Effects of Antiepileptic Drug Therapy During Pregnancy on School-age Offspring. Epilepsia, Vol. 38. Suppl. 3, P. 170.

Lespinet V., Lurton D., N'Kaoua B., Flori L., Rougier A., Claverie B. Laboratoire de Neuropsychologie Experimentale et Laboratoire de Neurobiologie et Neuroimagerie Experimentales, Univ. Victor Segalen Bordeaux, Clinical Neurological Sciences Southampton General hosp., Southampton, UK), (1997):
Verbal Memory Impairment in Right Temporal Lobe Epilepsy: Role of a Contralateral Dysfunction Evaluated by Xenon-CT Blood Flow Study. Epilepsia, Vol. 38, Suppl. 3, P.237.

Lespinet, V., N'Kaoua B., Rougier A., Claverie B. (Laboratoire de Neuropsychologie Experimentale et Laboratoire de Neurobiologie et Neuroimagerie Experimentales, Univ. Victor Segalen, Bordeaux, France), (1997):
Effect of Early Versus late Onset of Temporal Lobe Epilepsy on the Nature of Memory Deficits. Epilepsia, Vol. 38, Suppl. 3, P. 237.

Lesser, R.P., Luders, H., Wyllie, E., Dinner, D.S. and Morris, H.H. (1986):
  mental deterioration in epilepsy. Epilepsia, 27 (Suppl. 2), S105-S123.

Levav M., Mirsky A.F., Herault J., Xiong Andermann L., (National Inst. of Mental Health (NIMH), National Insts of Health and McGill Univ. Montreal Neuro Inst., US and Canada), (1997):
  (Familial Characteristics of Cognitive Impairment in Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 134.

Levy R.H. (Univ. of Washington, U.S., (1997):
  Rational Approach to Prediction of Drug Interactions Among Antiepileptic Drugs. Epilepsia, Vol. 38, Suppl. 3, P. 278.

Lishman, W.A. (1972):
  Selective factors in memory: Part 2: Affective disorder. Psychol. Med., 2, 248-253.

Lim, J., Yagnik, P., Schraeder, P. and Wheeler, S. (1986):
  Ictal catatonia as a manifestatiaon of nonconvulsive status epilepticus. J. Neurol. neurosurg. Psychiatry, 49, 833-836.

Loftus A., Saling M.M., Jackson G. Berkovic (Austin and Repatriation Medical Centre & The Univ. of melbourne, Austrialia), (1997):
  (Hippocampal T., Mapping and Verbal memory). Epilepsia, Vol. 38, Suppl. 3, P. 7.

Loiseau J., Loisea P. (Univ. Hosp., Bordeaux, France), (1997):

Mortality in Patients with Epileptic Seizures. Epilepsia, Vol. 38, Suppl. 3, P. 244.

Loiseau, P., Strube, E., Broustet, D., Battellochi, S., Gomeni, C. and Morselli, P.L. (1983):
Learning impairment in epileptic patients. Epilepsia, 24, 183-192.

Long, C.G. and Moore, J.R. (1979):
Parental expectations for their epileptic children. J. Child. Psychol. Psychiat., 20, 299-312.

Lopez-Martin V., Arcas J., Martinze-Bermejo A., Coya A., Rocha C., Castroviejo I. and Tendro A., (Neuropediatrics, Hospital la Paz, Madrid, Spain), (1995):
Single Photon (Emision computed Tomograph in Children with Partial Epilepsy). P. S133.

MacLeod, C.M., Dekaban, A.S. and Hunt, E. (1978):
Memory impairment in epileptic patients: selective effects of phenobarbital concentration. Science, 202, 1102-1104.

Magaudda A., Meduri M., Galitto G., Burzomati D., Ero C., DiPerri R. (Neurologic and Psyhiatric Clinics, Univ. of Messina, Italy), (1997):
The Psychopathology of Depression in Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 228.

Maestu P., Gomez-Utrero E., Martin G., Sola R., Ortiz T. (Univ Hosp. San Carlos, Madrid, Spain), (1997):
Are Memory Tasks More Sensible to Temporal Lobe Damage than Oddball Paradigm? Epilepsia, Vol. 38, Suppl. 3, P. 46.

Manchanda R., Freeland A., Schaefer B., McLachlan R., Blume W. (Univ. of Western Ontario, London, Canada), (1997):
Auras, Seizure Focus and Psychiatric Disorders. Epilepsia, Vol. 38, Suppl. 3, P. 228.

Manni R., Murelli R., Politini L., Ratti M.T., Sartori I., Galimberti C.A., Teratara A. (Inst. of Neurology, Mendino C. Univ. of Pavia, Italy), (1997):
Drowsiness in Epilepsy Patients: Assessment by Means of the Epworth Sleepiness Scale in167 Patients. Epilepsia, Vol. 38, Suppl. 3, P. 17.

Mares P. (Prague, Czech Republic), (1997):
Is Failure of Post-ictal Refractoriness One of the Mechanisms of Increased Epileptogenesis in Immature Brain? Epilepsia. Vol. 38, Suppl. 3, P. 106.

Maria Isabel Vergani C., (Hospital Infantil Menino Jesus, Sao Paulo, Brazil), (1995):
Partial Seizures in Children Study of 80 Cases. Epilepsia. Vol. 36 Suppl. 3, P. S202.

Marson A.G., Williamson P.R., Hutton J.L., Chadwick D.W., On behalf of the Monotherapy Trialists Collaboration (The Walton Centre, Liverpool, UK), (1997):
Valproate vs Carbamazepine: A Meta-analysis using inividual Patient Data of Randomised Controlled Comparative Monotherapy Trials). Epilepsia, Vol. 38, Suppl. 3, P. 42.

Martinez-Bermejo A., Lopez-Martin V., Arcas J., Roche C., Tenderoand A., Castroviejo I. (Neuropediatrics, Hospital La Paz, Madrid, Spain), (1995):

(Vigabatrin in Resistant Childhood Epilepsy). Epilepsia, Vol. 36. Suppl. 3, P. 103.

Maryek G., Golovach T., Mouzitchouk L., Kharchuk S. (Epilepsy Centre, Ukraine), (1997);
Chernobyl as the Factor of Some Intractable Eilepsy, Epilepsia, Vol. 38, Suppl. 3, P. 2.

Mary Robertson (Department of Psychiatry, University College London Medical School, London, England). (1995);
(Department in People with Epilepsy). Epilepsia Vol. 36. Suppl. 3, P. S211.

Mary Robertson M., The Middlesex Hospital, London. (1988):
Epilepsy and Mood. Epilepsy, Behaviour and cognitive Function. P. (145-152).

Mastrangelo M., Faldini P., Mariani E., Sergi P., Pastorillo G.C. (Inst. Clinici Perfezionamento, Milan, Italy), (1997):
(Neonatal Status Epilepticus and Epilepsy). Epilepsia, Vol. 38, Suppl. 3, P. 47.

Matarazzo, J.D. and Herman, D.O. 91984):
Base rate data for the WAIS-R: Test-retest, stabiality and VIQ-PIQ differences. J. Clin. neuropyschol., 6, 351-366.

McFie, J. (1969):
The diagnostic significance of disorders of higher nervous activity. In Handbook of Clinical neurology (eds P.J. Vinken and G.W. Bruyn), Vol. 4. ch. 1. North-Holland, Amsterdam.

Matkovic Z., Oxbury S.M., Hiorns R.W. Morris J.H., Carpenter K., Adams C.B.T., and Oxbury J.M. (Radcliffe Infirmary,

Oxford,U.K.) (1997):
Hippocampal Neuronal Density Correlates with preoperative Nonverbal Memory in Patients with Temporal Lobe Epilepsy. Epilepsia. Vol. 38, Suppl. 3, P. 93.

Maxoutova E., Sokolova L., Zheleznova E., Tokareva N. (State Russian Center for Psychiatry and Narcology, Moscow, Russia), (1997):
Psychopharmacotherapy of Epilepsy and Social Functioning of the Patients. Epilepsia, Vol. 38, Suppl. 3, P. 85.

Mayer H., Diener W., (Epilepsy Centre Kehl-Kork. Dept of Neuropaediatrics, Freiburg, Germany), (1997):
Cognitive Functions in Children with Different Epileptic Syndromes, whose Seizures Remitted: A Long-Term Follow-up. Epilepsia, Vol. 38, Suppl. 3, P. 134.

Mayer H., Diener W., (Epilepsy Centre, Khel-Kork, Dept of Neuropaediatrics, Freiburg, Germany), (1997):
The Cognitive and Psychosocial Development of Children with Benign Focal Syndromes. Epilepsia, Vol. 38, Suppl. 3, P. 134.

McMackin D., Kirker J., Phillips J., Staunton H. (Richmond Inst. for Neurology & Neurosurgery, Beaumont Hosp., Dublin, Ireland), (1997):
Improving the Utility of the Wechsler memory Scale-Revised in Determining Seizure Laterality). Epilepsia, Vol. 38, Suppl. 3, P. 47.

Meador K.J. (Medical College of Georgia, Augusta, Georgia, US), (1997):

Assessing Cognitive Effects of New AED Without the Bias of Practice Effects. Epilepsia. Vol. 38. Suppl. 3. P. 60.

Mercade J., Torras M., Ojea T. (HR Carlos Hoya Malaga, Spain), (1997);
(Intractable Partial Epilepsies. Clinical Review). Epilepsia. Vol. 38, Suppl. 3, P. 14.

Michael Privitera (Neurology Department, University of Cincinnati, Cincinnati, OH, U.S.A.), (1995):
Long-Term Cognitive Effects of Topiramate Epilepsia Vol. 36. Supl. 3, P. 153.

Michael Saling M., Marie F. O'Shea, and Samuel F. Berkovic (Austin Hospital, Victoria, Australia), (1995): Verbal Memory in Temporal Lobe Epilepsy: Cognitive Influences on Task-Specific Effects). Epilepsia, Vol. 36. sppl. 3. S3. 94.

Michael R. Trimable (National Hospitals for Nervous Diseases and Institute of Neurology, London) (1988):
Anticonvulsant drugs: mood and cognitive function, P. 135-142.

Mihaly Szabo, Andras Kondacs ad Miklos Szolics, (1995):
Analysis of EEG background activity in Epileptic Patients. Epilepsia, Vol. 36 Suppl. 3 P. S82.

Milewska D., Niedzielska K., Iwinska-Buksowicz B., (Inst. of Pschiatry and Neurology, Warsaw, Poland), (1997) :
Epileptic Seizures and Epileptiform Discharges in SSPE patients. Epilepsia, Vol. 38, Suppl. 3, P. 16.

Miller L.A. Lai R., and Munoz D.G., (University Hospital/U.W.O., Ontario, Canada), (1995):
Mesial Temporal Pathology and Human Memory. Epilepsia, Vol. 36, Suppl. 3, P. 93.

Milne E., Brien G.O. (Northgate Hosp., England), (1997):
Epilepsy in Learning Disabled offenders - Prevalence, Diagnosis and Impact of Treatment. Epilepsia, Vol. 38, Suppl. 3, P. 111.

Misirli H., Ozalp K., Tunali F., Erdogan N., Yasar Erenoglu (Haydarpasa Numune Hosp., Turkey), (1997):
Aetiology and Prognosis in Status epilepticus. Epilepsia, Vol. 38, Suppl. 3, P. 256,7.

Mitchell L.A., Jackson G.D., Fitt G.J., and Berkovic S.F. (Austin Hospital, Victoria, Australia), (1995):
Hippocampal Sclerosis is Frequently Associated with Anterior Temporal Lobe Abnormalities. Epilepsia. Vol. 36, Sppl. 3, P. S94.

Ming Xu Ke, Guei Fan Lou, Dan Zhang and Jain, Yan (Department of Pediatric Neurology, Children's Hospital of Capital Institute of Pediatrics, Beijing, P.R. China), (1995):
(Memory Study of Antiepileptic Drug discontinuation in Children with Epilepsy. Epilepsia, Vol. 36, Suppl. 3, P. S130.

Mojs E., Galas-Zgorzalewicz B. (Dept of Developmental Neurology, Univ. of Medical Sciences in Poznan, Poland), (1997):
Evaluation of Cognitive Abilities During Add-on Lamotrigine Therapy of Children and Adolescents with Intractable Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 115.

Monaco F., Torta R., Borio R., Cicolin A. and Ravizza L., (Neurological Clinic, Sassari and Department of Psychiatry, Torino University, Torino, Italy), (1985):
Epilepsy, Department, and Selective Serotonin Reuptake Inhibitor Drugs). Epilepsia Vol. 36, Suppl. 3, P. S175.

Montalenti E., Gaia S., Fiori L., Benna P. (Dipartimento di neuroscienze, Univ. di Torino. Ospedale Psichiatrico Fatebenefratelli, San Maurizio Canavese, Italy), (1997):
Clozapine-induced Seizures and EEG Alterations: Follow-up of 60 Patients. Epilepsia, Vol. 38, Suppl. 3, P. 3.

Moran P., McMacken D., Taylor D., Staunton H., Waddington J., O'Callaghan E., Phillips J. (Beaumont Hosp., Royal College of Surgeons in Ireland, Cluain Mhuire Family Centre, Dublin, Ireland), (1997):
Psychopahology and Psychosocial Disability in Treatment Resistant Temporal Lobe Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 166.

Mothersill IW (Swiss Epilepsy Centre, Zurich, Switzerland), (1997):
Non-epileptic is not Automatically Psychogenic. What has to be Excluded before the Diagnosis Can Be Made. Epilepsia, Vol. 38, Suppl. 3, P. 283.

Mrabet A., Fredj M., Gargouri A., Gouider R. (Neurological Dept Hopital Chalres Nicolle, Tunisia), (1997):
Familial Photosensitive Asence Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 120.

Murakami N., Ohno S., Asano T., Oka E. (Dept of Child Neurology, Okayama Univ. Medical School, Japan), (1997):

Prolonged Convulsions during Infancy and Early Childhood in Mesial Temporal Lobe Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 15.

Murthy JMK, Sitajayajaya L.S., Mantha S. (Nizam's Inst. of Medical Sciences, Hyderabad, India), (1997):
Remission in Partial Epilepsy - A Study from South India. Epilepsia, Vol. 38, Suppl. 3, P. 17.

Muscas G.C., Chiaramonti R., Borgheresi A., Campostrini R., Galli R., Gneri C., Murri L., Palumbo P., Rocchi R., Severi S., Ulivelli M., Vatti G.P., Zaccara G., (Dept of Neurology and Epilepsy Centre, Careggi Hosp., S. Salvi, Florence: "Prato 'Pisa: 'Siena: Arezzo, Italy), (1997):
Long-term Cognitive and Mood Effects of Gabapentin. Epilepsia, Vol. 38, Suppl. 3, P. 35.

Muscas G.C., Chiaramonti R., Poggiolini D., Paganini M. (Univ. of Florence and Careggi Hosp. Dept. of Neurology, and Epilepsy Centre, Florence, Italy) (1997);
A Survey of Gabapentin Treatment in Partial Epilepsy, Epilepsia, Vol. 38, Suppl. 3, P. 35.

Nam H., Lee S-K (Dept of Neurology, Seoul Nat'l Univ., Korea), (1997):
Material-specific Memory in TLE Depending on Language Dominance). Epilepsia, Vol. 38, Suppl. 3, P. 44.

Naz Yeni S., Karaagac N., Altinel A. (Univ. Istanbul, Cerrahpa SA Medical Fauclty, Turkey), (1997):
Post-ictal EEG after Generalised Tonic-Clonic Seizures Epilepsia, Vol. 38, Suppl. 3, P. 125.

Neufeld M.Y., Vishna T., Chistik V., Korczyn A.D., (Tel-Aviv Medical
        Centre, Israel), (1997):
        Seizure-related Injuries; Characteristics and Risk Factors.
        Epilepsia, Vol. 38, Suppl. 3, P. 241.

Nikolaishvili R., Tatishvili N., Geladze T. (Epilepsy Center, Inst. Of
        Neurology, Republic of Georgia), (1997):
        Relapse of Complex Partial Seizures after Wihdrawal of
        Medication. Epilepsia, Vol. 38, Suppl. 3, P. 238.

Ogunrin A.O., Adamolekun B., Ogunniyi A. and Aldekamp A.P.
        (University of Benin Teaching Hospital,
        Benin City, Nigeria(1995):
        (Determination of pattern and Prevalence of Cognitive
        Impairment in Nigerian Epileptic Patients Using the "Fepsy"
        Program. Epilepsia Vol. 36, Suppl. 3, P. 126.

Oosterhuis A., (Dr Hans Berger Kliniek, The Netherlands), (1997):
        The Psycho-Educational Approach to Epilepsy. Epilepsia, Vol.
        38, Suppl. 3, P. 88.

O'Shea M.E., Saling M.M., Bladin P.F., Berkovic S.F., (Univ. of
        Melbourne, Victoria/A & RML (Austin Campus).
        Australia), (1997) :
        An Investigation of Memory Self-report in Temporal Lobe
        Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 240.

Owczarek K., Jcdrzcjczak J., Majkowski J. (Dept. Neurology and
        Epileptology, Medical Centre for Post Graduate
        Education, Warsaw, Poland), (1997):
        Interpersonal Relations between Patients: Non-Epileptic to
        Epileptic and Epileptic to Epileptic patients during

Hospitalisation. Epilepsia, Vol. 38. Suppl. 3, P. 127,128.

Oxyury S.M., Adams C.B.T., Middleton J.A. and Oxybury J.M., (Radcliffe infirmary, Oxford, England). (1995):
Temporal Lobe Epilepsy Surgery in Young Children: Neuropsychological outcome. Epilepsia. Vol. 36, Suppl. 3, P. 242, 243.

Pacifici L. Faedda M.T., Ferrigno P., Vernieri F., & Rossini P.M. (1995):
Ambulatory EG Monitoring of ictal versus Inerictal Abnormalities in patients with GME. Epilepsia. Vol. 36, Suppl. 3, P. 77, 78.

Pal D.K. (Neurosciences Unit, Inst. of Child Health, London, UK), (1997):
A Case-Control Study of Risk Factors for Childhood Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 122.

Pang K.A., Chadwick D.W., (The Walton Centre for Neurology & Neurosurgery, Liverpool. UK), (1997):
Clinical Characteristics of Idiopathic Generalised Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 14.

Paolo Tanganelli and Giovanni Regesta (Department of Neurology, Epilepsy Center, San martino Hospital, Genova, Italy), (1995):
(Vigabatrin Versus Carbmazepine in Newly Diagnosed Epileptic Patients : A Randomized Response Conditional Cross-over Study). Epilepsia, Vol. 36, Suppl. 3, P. S104.

Papavasiliou A., Papadimitriou M., Manta C., Nikolaou A., (Neurology

Dept., Pendeli Children's Hosp. Athens, Greece), (1997):
School Performance in Epileptics and Some Factors
predisposing to Low Achievement. Epilepsia, Vol. 38, Suppl.
3, P. 118.

Papavasiliou A., Kondylis A., Kolaou A.N. (Neurology Dept. and
Developmental Clinic, Pendeli Children's Hospt., Greece),
(1997):
What do School Teachers Know about Childhood Epielpsy?.
Epilepsia, Vol. 38, Suppl. 3, P. 112.

Pauli E., Eberhard K., Schaafer I., Pickel S., Stefan H. (Dept. of
Neurology Dev. of Neuroradiology, Dept of Neurosurgery,
Univ. of Erlangen-Nuremberg, Germany), (1997):
Memory Function in patients with Intractable TLE:
Comparison with CSI. Epilepsia, Vol. 38, Suppl. 3, P. 225.

Pelzi, Mamoli B. and Spatt J., (1995):
Spike Detection in Partial Epilepsies; Reliability of an
Automted Analysis System in the Setting of Long-Term
video-EEG Monitoring. Epilepia, Vol. 36, Suppl. 3, P. S82.

Penniello M.J. Lecoutour X., Pottier M., Pignord, V. Jallon P., (1995):
Children Epilepsy in Basse-Normandie, France: Clinical
Epidemiology, Handicaps & Schoolina). Epilepsia, Vol. 36,
Suppl. 3, P. S17.

Perucca E., (Clin Pharm Unit, Univ of Pavia, Italy), (1997):
Clinical Significance of Drug Interactions in Epilepsy: A
Changing Scenery. Epilepsia, Vol. 38, Suppl. 3, P. 278.

Peter B.C. Fenwick (The Maudsley Hospital, London) (1988);

Seizaure, EEG discharges and Behaviour Viaf. Epilepsy, Behaviour and cognitive functions. P 51-66.

Phillips N., Lockard J., (Univ. of Washington, US), (1997):
Visual Recognition Memory in Monkeys Prenatally Exposed to Antiepilepic Compounds. Epilepsia, Vol. 38, Suppl. 3, P. 170.

Piatella L., Zamponi N. and Cardinali C. (Children's Neuropschiatric Ward, Ancona, Italy), (1995):
Vigabatrin Versus Carbamazepine in Newly Diagnosed Partial Epilepsy in Childhood. Epilepsia, Vol. 36, Suppl. 3, P. S103.

Pierre Loiseau, Evelyn Strube and Jean-Louis Signoret, (Hospital Pellegrin, University de Bordeaux de Brodeaux II. France and Hopital de la Salpetriere, Universite de Paris VI. France), (1988):
(Memory and Epilepsy) via Epilepsy, Behaviour and Cognitive Function Editted by M.R. Trimble and E.H. Reynolds. John Wiley & Sons Ltd. P. 165-176.

Post, R.M., Uhde, T.W., Joffe, R.T., Rov-Byrne, P.P. and Kellher, C. (1985):
Anti-convulsant drugs in psychiatric illness. In Trimble, M.R. (ed). The psychopharmacology of Epilepsy. Chichester, John wiley & Sons, pp. 141-171.

Pradella-Hallinan M. De Volder A., Djabraian A., Silva D.F. (Louvain Univ., Belgium & Federal Univ. of S Paulo, Brazil), (1997):
(Brain Glucose Metabolism in Epileptic Patients). Epilepsia, Vol. 38, Suppl. 3, P. 7.

Pressler R.M., Binnie C.D., Coleshil S.G., Crouchman M., Hughes E. (Inst. of Epilepsy, London, UK), (1997):
Benign Partial Epilepsy with Centro-Temporal and with Occipital Spikes and Learning Difficulties in Two Brothers. Epilepsia, Vol. 38, Suppl. 3, P. 138.

Pressler R.M. and Brandle U. (Department of Neuropediatrics. Free University Berlin, Germany), (1995); Transistory Cognitive Impairment During Very Brief Subclinical EEG Discharges in Children. Epilepsia, Vol. 36, Suppl., P. S93.

Quadfasel, A.F. and Pruyser, P.W. (1955):
Cognitive deficits in patients with psychomotor epilepsy. Epilepsia, 4, 80-90.

Quattrini A., Ortenzi A., Paggi A., Foschi N., Quattrini C. (Centro Epilepsia Ospedale Ancona, Italy), (1997):
Drug-Resistance in a Large Population of Epilepic Patients. Epilepsia, Vol. 38, Suppl. 3, P. 17.

Ravnik I.M., Vrba L., Burian M. (Centre for Epilepsy (Chidren and Adolescents) Clinical Centre, Faculty of Sport, Ljubljana, Slovenia), (1997):
Physical Education of Epileptic Children and Adolescents. Epilepsia, Vol. 38, Suppl. 3, P. 114.

Recktor I., Bradzdil M., Tyrlikova I., Svejdova M. (1st Clinic of Neurology Masaryk Univ. Brno, Czech Rep), (1997);
Epileptic Seizures, Pseudo-Epileptic Seizures and Psychically Evoked Epileptic Seizures. Epilepsia, Vol. 38, Suppl. 3, P. 14.

Reynolds, J.R. (1861):

Epilepsy: its symptoms, treatment and relatiaon to other chronic convulsive diseases. Churchill. London.

Reynolds, E.H. (1986):

Antiepileptic drugs and personality. In Aspects of Epilepsy and Psychiatry (eds M.R. Trimble and T.G. Bolwig), pp. 89-99. John Wiley & Sons, Chichester.

Rodin, E.A., Schmaltz, S. and Twitty, G. (1986):

Intellectual function of patients with childhood Epilepsy. Dev. Med. & Child Neurol, 28, 25-33.

Rodin, E.A. (1968):

The Prognosis of Patiens with Epilepsy. Charles C. Thomas. Springfield. USA.

Ronen G., Richrads J., Cunningham C., Rosenbloom D.. (McMaster Univ., Canada), (1997):

Valproic Acid-Placebo Double Blind Crossover Trial in Children with Behavioural and learning Problems. EEG. Epileptiform Bursts But No. 131. Clinical Seizures. Epilepsia, Vol. 38, Suppl. 3, P. 169.

Rosvold, H.E., Mirsky, A.F., Sarason, I., Bransome, E.D. and Beck, L.H. (1956):

A continuous performance test for brain damage. J. Consult. Psychol., 20, 343-350.

Ross, E.M. (1973):

Convulsive disorders in British Children. Proceedings of the Royal Society of Medicine, 66, 703-704.

Ross, E.M. and West, P.B. (1978):

>Achievements and problems of British eleven year olds with epilepsy. In Advances in Epileptology - 1977 (eds H. Meinardt and A.J. Rowan), pp. 34-37, Swets & Zeitlinger, B.V., Amsterdam.

Runge U., Pasquet J., Siegmund W., Franke G., Kessler Ch., (Dept of Neurology and Pharmacology Inst., Univ. of Griefswald. Germany), (1997):

>The Importance of Therapeutic Drug Monitoring in Optimising Anticonvulsant Therapy in Partial Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 30.

Sabhanvey J.W., Nadkarni V.V. and Deepak Jain (Choithram Hospital and Research Centre, Indore, India), (1995):

>Psychosocial Aspects of Epilepsy. Epilepsia, Vol. 36, Sppl. 3, P. S183.

Sakurai S., Motohka H., Ishida S., Takayama K., Ishii H., Maeda H. (Dep of Neuropsychiatry, Kurume Univ School of Med. Japan), (1997):

>Nonconvulsive Status Epilepticus and its 1231-IMP SPECT. Epilepsia, Vol. 38, Suppl. 3, P. 226.

Sarah Gehlert (The University Chicago, Chicago IL, U.S.A.), (1995):

>Cognitive Restructing for Psychosocial Problems in Epilepsy. Epilepsia. Vol. 36, Suppl. 3, P. S10.

Sälke-kellermann, R., Kopps Ch., Amaker Y., Brnner Th., Wehrli A., (Swiss Epilepsy Centre, Zurich, Switzerland), (1997):

>Long-term Hospitalisation of School Children Medical Effects. Epilepsia, Vol. 38, Suppl. 3, P. 45.

Sang-Doe Yi and Hong-keun Kim (Department of Neurology, keimung University; and Department of Psychotherapy, Taegu University, Taegu, Korea), (1995):
Effects of Temporal Lobe Epilepsy on Hemispheric Memory Functions: An Inracarotid Amobarbital Procedure Study). Epilepsia, Vol. 36, Suppl. 3, P. 125.

Sarac E., Misirli H., Ozge A., Erenoglu N.Y. (Haydarpasa Numune Hosp., Istanbul. Turkey), (1997):
Memory Dysfunctions in Primary Generalised Epilepsy. Epilepsia, Vol. 38, Suppl. 3, P. 239.

Sato M., Sato S., Chepurnva NE., Rodionova V.I., Chepurnov S.A., Ashmarin P., (Moscow State Univ., Moscow Russia), (1997);
Spatial Orientaion and Lateralisation in Cognitive Function of Children with Epilepsy and Mental Impairment. Epilepsia. Vol. 38, Suppl. 3, P. 133.

Savic C., Rajsic N., Sinnreich V., Propovic M., Buder N. (Inst. for Mental Health. Beograd, Yugoslavia), (1997):
The Effect of Carbamazepine (Tegretol) on Interictal Epileptiform Activity in EEG of Psychomotor Epilepsy Patients. Epilepsia, Vol. 38, Suppl. 3, P. 72.

Schacter S.C., Vasquez B., Fisher R, Bennett D. (Harvard Medical School, US), (1997);
Placebo-controlled Inpatient Monotherapy Trial of Oxcarbazepine. Epilepsia, Vol. 38, Suppl. 3, P. 30.

Schffer I.E., (Austin & epatriation Medical entre, Victoria, Australia), (1997):

(Genetics of Generalised Epilepsies). Epilepsia. Vol. 38. Suppl. 3. P. 169.

Schmitt-Mechelke (Children's Hosp., Lucerne, Switzerland), (1997):
Efficacy of CBZ in Benign Partial Epilepsy of Childhood (BPECT). Epilepsia, Vol. 38, Suppl. 3, P. 133.

Schmitz B., Robertson M. and Trimble M.R. (Department of Neuropsychiatry, Institute of Neurology, London England), (1995):
(Biological and Social Risk Factors for Depression and Schizophrenia-like Psychosis in Epilepsy). P. 175-179.

Schuler M., Donati F., Vella S., Ramelli G.P., Vassella F. (Division of Child Neurology, Dept of Paeditrics, Division of Clinical Neurophysiology, Dept. of Neurology, Univ. Hosp. of Berne, Switzerland), (1997):
School Performance and Social Integration of Epileptic Children. Epilpesia, Vol. 38, Suppl. 3, P. 113.

Schwab, R.S. (1939):
Method of measuring consciousness in attacks of petit mal epilepsy. Archf. neurol. Psychiat., 41, 215-217.

Seidenberg, M., beck, N., Geisser, M., Giordani, B., Sackellares, J.C., Berents, S. Deifuss, F.E. & Boll, T.J. (1986):
Academic achievement of cyhildren with epilepsy. Epilepsia, 27, 753-759.

Selukov G. (National Medical Univ., Dept of Psychiatry, Ukraine), (1997):
Psychophysiological Markers of Chronic Epilepsy. Epilpesia.

Vol. 38, Suppl. 3, P. 234.

Seppälä U., (Finnish Epilepsy Association, Finalnd), (1997):
    The Life of a Child with Epilepsy. Epilpesia, Vol. 38, Suppl. 3, P. 112.

Settle D. (David Lewis Organisation, UK), (1997):
    Epilepsy in Education: A National Training Programme. Epilpesia, Vol. 38, Suppl. 3, P. 114.

Severi S., Bianchi A., Zolo P. and Muscas G.C. (Neurology Department Arezzo Hospital; Careggi Firenze Hspital, Arezzo, Italy), (1995):
    Lamotrignine Monotherapy in patients with partial Epilepsy. Epilepsia Vol. 36, Suppl. 3, P. S113.

Sheng Liu Li, Guixiang Zhang and Shih-Jun Chen, (1995):
    Epidemiological Survey of Childhood Epilepsy. Epilepsia Vol. 36, Suppl. 3, P. S18.

Shewmon D.A., Warwick J., Pearcock J., (UCLA Medical School), (1997):
    Epilepsy Surgery in Children with Medically Controlled Seizures and Continuous Focal Interictal Discharges: cognitive and Developmental Oucomes US. Epilpesia, Vol. 38, Suppl. 3, P. 169.

Shroff D.N., Kumar S.P., Radhakrishnan K. (Sree Chitra Tirunal Inst. for Med. Sci. Technology (SCTIMST, India), (1997):
    Profile of Antiepileptic Pharmacotherapy: An Experience from a Tertiary Referral Centre in South India. Epilpesia, Vol. 38, Suppl. 3, P. 39.

Sidenvall R., Forsgren L., and Heijbel J., (1995):

>Prevalence and Other Characteristics of Children with Epilepsy in a Swedish Count. Epilepsia. Vol. 36, Suppl. 3, P. S16.

Shorvon, S. and Reynolds, E.H. (1979):

>Reductiaon in polypharmacy for epilepsy. Br. Med. J., 2. 1023-1025.

Siffels M.C., De Graaf E.H. (Inst. voor Epilepsiebestrijding, The Netherlands), (1997):

>Cognitions Concerning Seizures. Depression and Cognitive Behavioural Therapy. Epilpesia. Vol. 38. Suppl. 3, P. 103.

Smith, D.B., Craft, R.B., VA Cooperative Study Group (1985):

>Differential neurotoxicity of four major anticonvulsants. Ann. Neurol., 18, 119.

Sobaniec W., Sobaniec H., Sobaniec-Lotowska M., nd Kutak W., (1995):

>Interaction of Valporate with Other Antiepileptic Drugs in Children Treated for Epilepsy. Epilepsia. Vol. 36. Suppl. 3. S68.

Somoz M.J., Forlenza R.H., Brussino M. and Foglia V.L., (1995):

>Neuropidemilogical Study of Epilepsy in Primary schools in Buenos Aires City, Aregentina. Epilepsia, Vol. 36, Suppl. 3, P. S16.

Sorokina N.D., Karlov V.A. and Seltitsky G.V. (Institute of Higher Nervous Activity, Moscow, Russia), (1995):

>Cognitive Functions of patients with Epileptic Focus in

Parietal Regions. Epilepsia, Vol. 36, Suppl. 3, P. 125.

Souza EAP, Nista C. Scotoni A.E., Guerreiro M.M. (Neurology Dept. Unicamp, Brazil), (1997);
Support Groups for Parents: Identification of Family Factors toward Epilepsy. Epilpesia, Vol. 38, Suppl. 3, P. 114.

Spector S., Foots A. and Goldstein L.H. (Institute of Psychiatry and Epilepsy Unit, Maudsley Hospital, London, U.K. (1993);
Reduction in Seizure Frequency as a Result of Group Intervetion for Adults with Epilepsy.

Stores G., (1990):
Electroencephalographic Parameters in Assessing the Cognitive Function of Children with Epilepsy. Epilepsia. 31, (Suppl. 4) S45.

Stores, G. (1973):
Studies of attentiaon and seizure disorders. Dev. Med. Child Neurol., 15, 376-382.

Stores, G., Hart, J. and Piran, N. (1978):
Inattentiveness in schooolchildren with epilepsy. Epilepsia, 19, 169-175.

Strang J.D., (1990):
Cognitive deficits in Children : Adaptive Behaviour and Treatment Techniques. Epilepsia. 31, (Suppl. 4): S54.

Sugiyama O., Matsunaga M., Makazawa M., Shigematsu H., Morikawa T., Inoue Y., Fujiwara T., Yagi K. (National Epilepsy Centre. Shizuoka Higashi Hosp., Japan), (1997):

Social-developmental Delays of Pre-school Children with Epilepsy. Epilpesia, Vol. 38. Suppl. 3. P. 114.

Swink T., Vining E. Casey, Kelly, Pillas, Hsieh, Pyzik, Freeman (John Hopkins Medical Ints., US), (1997):
Efficacy of the Ketogenic Diet in Children Under 2 Years of age. Epilpesia, Vol. 38, Suppl. 3, P. 26.

Sykes C., McConnell H. (Inst. of Epileptology, Halifax, UK), (1997):
Reltionships of Depression and Epilepsy. Epilpesia. Vol. 38. Suppl. 3, P. 83.

Takei K., Miura H., Takanshi S., Shirai H., Sunaoshi W.. Hosoda N. (Dept of Paediatrics, Kitasato Univ. School of Medicine. Japan), (1997):
Long-term Outcome of Epilepsy in Children with Grand Mal Seizures on Awakening Treated with Sodium Valproate Monotherapy. Epilpesia, Vol. 38. Suppl. 3, P. 55

Teruyo Miyoshi, Tetuo Homma, Setsuko Sato, Shirou Ishida and Teiichi Onuma (Department of Nursing, Section of Epilepsy in Department of Psychiatry, National Center Hospital for Mental Nervous and Muscular Disease NCNP, Kodaira, Tokyo, Japan), (1995):
Family Care for Intractable Epilepsy : History and Experience of Parents-Hospital Staff Assocition in Epilepsy Unit in Japan. Epilepsia, Vol. 36, Suppl. 3, P. S182.

Tinuper P., Cerullo A., Cortelli P., Pierangeli G., Provini F., Marini C., Rosati A., Lugaresi E. (Inst. of Clinical Neurology, Univ of Bologna, Italy), (1997):
Ictal Autonomic Changes in Partial Epileptic Seizures

Epilpesia. Vol. 38. Suppl. 3. P. 113.

Thompson, P.J. and Trimble, M.R. (1982):
Anticonvulsant drugs and cognitive functions. Epilepsia. 23, 531-544.

Thompson, P.J., Sander, J.W.A.S. and Oxley, J. (1987):
Intellectual determination in severe Epilepsy. In Advances in Epileptology. Vol. 16(eds P. Wolf, M. Dan, D. Janzand. and F.G. Dreifuss), PP. 661-614, Raven Press, New York.

Thompson P.J., The Chalfont Centre for Epilepsy. Chalfont St Peter, Buckinghamshire, (1988):
Methods and problems in the assessment of behaviour disorders in epileptic patients via Epilepsy, Behaviour and Cognitive Function. P. (27-39).

Tohru Konishi, Yoshihiro Naganuma, Kazuhisa Hongou, Miyako Murakami, Miwa Yamatani and Shinichi Yagi (Department of Pediatrics, Toyama, Japan), (1995):
Discontinuation ofAntiepileptic Drug Therapy in Childhod Epilepsy: Factors Related to Age. Epilepsia. Vol. 36. Suppl. 3. P. 164.

Toone B.K., Mellers J.D.C. Lishmann W.A. (Inst. of Psychiatry, London, UK), (1997):
Epilepsy and Schizophrenia: an MRI Study., Epilpesia, Vol. 38, Suppl. 3, P. 228.

Tourniaire D., Remy C., Favel P. (Centre Medical La Teppe, France), (1997):
Death in an Epilepsy Centre. A 25 Year Survey. Epilpesia,

Vol. 38, Suppl. 3. P. 15.

Traynor B.J., Staunton H. (Beaumont Hosp., Richmond Inst., Dublin, Ireland), (1997):
Lateralising Value of Ictal Speech Manifestations in Complex Partial Epilepsy. Epilpesia, Vol. 38, Suppl. 3, P. 49.

Trimble M.R., (1990):
Antiepileptic Drugs, Cognitive Function and Behavior in Children: Evidence from Recent Studies. Epilepsia. 31 (Suppl. 4): S30.

Trimble M.R. and Thompson P.J., (1986):
Neuropschology of Epilepsy and its treatment. In Neuropsychological Assessment of Neuropsychiatric Disorders (eds I. Grant and K.M. Adams). P. 321-346. Oxford University Press, New York.

Trimble M.R. and Thomas L. (Institute of Neurology, Queen Square, London, England), (1995):
Effect of Vigabatrin on Cognitive Function in Volunteers. Epilepsia. Vol. 36, Suppl. 3, P. 109.

Trimble M.R., (1995):
Cognitive Effects of phenytoin, Phenobarbital. Carbamazepine, and Valproate. Epilepsia, Vol. 36, Suppl. 3. P. S31.

Tuxhorn I. (Bethel, Germany), (1997);
Clinicl Characteristics of Somatosensory Auras. P. 221.

Uhlmann C., Kotchoubey, B.,, Strehl U., Holzapfel S., Fröscher W.

(Die Weissenau, Dept, Neurology and Epileptology, Ravensburg, Germany), (1997):
Behavioural methods in Pharmco-Resistant Partial Epilepsies. Epilpesia, Vol. 38, Suppl. 3, P. 45.

Van Bogaert P., Wikler D., Damhaut P., Szeiwowski H., Goldman S. (ULB-Hopital Erasme, Brussels, Belgium), (1997):
Cerebral Glucose Metabolism in Benign Childhood Epilepsy with Centrotemporal spikes. Epilpesia, Vol. 38, Suppl. 3, P. 209.

Vanzan Paladin A. (ISNV, International Scool of Neurological Sciences. Venice, Italy), (1997):
Ethics and Epilepsy in the Islamic Wold. Epilpesia. Vol. 38. Suppl. 3, P. 282.

Veggietti P., De Agostini G., Muzio C., Fannetti L., Beocaria F., Lanzi G., (Fondazione C. Mondino IRCCS, Pavia, Italy, (1997):
Treatment with Vigabatrin in Paediatric Psychotic patients. Epilpesia, Vol. 38, Suppl. 3, P. 99.

Vella S., Ramelli G.P., Donati F., Vassella F., (Div. of Neuropaediatrics, Dept of Paediatrics, Div. of Clinical Neuropsychology, Dept of Neurology, Univ. of Berne, Berne, Switerland), (1997):
Childhood Seizures and Gender; Variations in Types of Seizures, Age of Onset. Duration of Treatment Until Remission and Familiarity of Epilepsy. Epilpesia, Vol. 38. Suppl. 3, P. 271.

Vrba L., Ravnik I.M., Burian M., (Centre for Epilepsy (Children and Adolescents), Clinical Centre, Ljubljana, Faculty of Sport, Ljubljana, Slovenia), (1997):

School Headmasters' Knowledge. Attitudes. practice (KAP) in Slovenia: Planning Global Action on Epilepsy in School-Field. Epilpesia, Vol. 38, Suppl. 3, P. 112.

Vermeulen J., Carpay H.A., (Meer & Bosch Epilepsy Centre. The Netherlands) (1997)::
Seizure Severity: a 1-year Follow-up of Children with Intractable Epilepsy. Epilpesia, Vol. 38, Suppl. 3, P. 15.

Wai-Keong N.G., Abdullah B.j., Tan C.T. (Univ. Malaya, Kuala Lumpur, Malaysia), (1997):
Etiology of Intractable focal Epilepsy in Malaysia by Clinical. Electroencephalograph and Magnetic Resonance (MR) Imaging. Epilpesia. Vol. 38, Suppl. 3, P. 1.

Wang L., Wang X (Beijing Medical Univ., PR China) (1997);
Antiepileptic Tolerance/Dependence of Clonzepam and Receptor Mechanisms. Epilpesia, Vol. 38. Suppl. 3. P. 225.

Weber A.M., Philbrook B., Mckellop J.M., De Grauw T., (Children's Hosp. Medical Center, Cincinnati, US), (1997):
Cognitive and Behavioural Characterisics of Children with New Onet Seizures. Epilpesia, Vol. 38, Suppl. 3, P. 164.

Wei-Ling L., Rajan U. (Dept of Neurology, Tan Tock Seng Hosp., Singapore, School Health Services, Singapore), (1997):
Psychosocial problems in School Children with Epilepsy in Singapore. Epilpesia, Vol. 38, Suppl. 3, P. 111.

Weissenrieder H., Mothersill I.W., Ried S., Grosin M., Kramer G. (Swiss Epilepsy Centre, Zurich, Switzerland), (1997);
Partial Epilepsy Manifesting as Nonconvulsive Status

Epilepticus. Epilpesia, Vol. 38, Suppl. 3, P. 157.

Weis M.E., Stefan H., Feistel H. (Neurological Dept Erlangen/ Darmstdt, Germany), (1997):
Focus Localisation by Interictal and ictal SPECT in Frontal Lobe Epilepsy. Epilpesia, Vol. 38, Suppl. 3, P. 8.

Wells, C.E. (1975):
Transient ictal psychosis. Arch. Gen. Psych., 32, 1201-1203.

Whitfield S.P., Newton E.R. and Crampton S. (David Lewis Centre, Cheshire, U.K. (, (1995):
Memory Difficulties and EEG Laterality in Children with Serve Epilepsy. Epilepsia, Vol. 36, Suppl. 3, P. S125.

Wilson S., Saling M., Bladin P., Withell P. (Austin & Repatriation Medical Centre, Australia), (1997):
Psychosocial Outcome After Anterior Temporal Lobectomy. Epilpesia, Vol. 38, Suppl. 3, P. 109.

Woermann F.G., Sisodiya S.M., Koepp M.,J. Free S.L., Shorvon S.D. Duncan J.S. (Inst. of Neurology, London, UK), (1997):
Widespread Cerebral Structural Changes in Patients with Idiopathic Generalised Epilepsy (IGE) - a Quantitative MRI Study. Epilpesia, Vol. 38, Suppl. 3, P. 229.

Wolanczyk T. (Dept. of Child psychiatry, Warsaw Medical Academy, Poland), (1997):
Biological and Medical Factors and Psychosocial Functioning in Adolescents with Epilepsy. Epilpesia, Vol. 38, Suppl. 3, P. 116.

Wolanczyk T., (Dept. of Child Psychiatry, Warsaw Medical Academy, Poland), (1997):

Psychosocial Functioning of Adolscents with Epilepsy. Epilpesia, Vol. 38, Suppl. 3, P. 116.

Yakup Sana Hacer Bozdemir, Nafiz Bozdemir, and Rasim Efe (C.U. School of Medicine and Family Medicine, Adana, Turkey), (1995):

Effect of Education on the Prognosis of Epilepsia, OL. 36. Suppl. 1, P. 182.

Yoshida M., Soga T., Haga M. Ohoti M. (Epilepsy Hosp. Bethel, Japan), (1997):

Long-term Follow up Study of Epileptic Spasms in Series. Etiological Cosniderations. Epilpesia, Vol. 38, Suppl. 3, P. 3.

Zentner, S. Steidele, A. Kowalik A. Hufnagel B. Ostertun, H.K. Wolf, M. Lendt, and C.E. Elger (1995):

(Surgical Treatment of Temporal Lobe Epilepsy in Children. Epilepsia, Vol. 36, Supl. 3, P. S23.

Zgorzalewicz M., Harasimczyk A. (Dept of Developmental Neurology Univ. of Medical Sciencies in Poznan, Poland), (1997):

Assessment of Quality of life in Epileptic School Children. Outcomes of Add-on Treatment with lamotrigine or Vigabatrin. Epilpesia, Vol. 38, Suppl. 3, P. 92.

Zupanc. M.I., Cascino G.D., Cicora K. (Mayo Clinic, Rocnester, US) (1997):

Morbidity and Mortality in Epilepsy Monitoring Unit-Mayo Experience 1994-96. Epilepsia, Vol. 38, Suppl. 3, P. 15.

Arabic
Summary

الملخص العربى

يعتبر مرض الصرع مشكلة كبيرة تهدد الأطفال المصابين به فى قدراتهم المعرفية والنفسية وتعوق أداءهم المدرسى فى الحياة العامة وهو لا شك مشكلة كبرى إذ إنها تمثل فى موضوع رسالتنا هذه شريحة كبيرة من المجتمع تقدر بـ ٩٦٤٢٠٤٢ تسعة ملايين وستمائة أثنين وأربعون الف وأثنين وأربعون نسمه أى ما يعادل (١٦,٢٦٪) ٤٧١٩٨٤٣ من الذكور ، ٤٤٢١٩٠٠ من الإناث حسب تعداد ١٩٩٦ ويعد مدى انتشارها (١٢,٩ /١٠٠٠) بشكل عام ، أما فى الأماكن الفقيرة (١٥,٨/١٠٠٠) وفى الأماكن الغنية (٩,٤ / ١٠٠٠) طبقاً لما جاء بالبحث المقدم من الدكتور التلاوى فى عام ١٩٩٦ وهو ما يعنى أن الشريحة المعرضة للمرض من الأطفال تقدر بعدد (١٢٤٣٨٢) .

وهو يمثل مشكلة كبيرة إذ أن الشريحة التى تصاب به فى دراستنا هذه (تتراوح بين ٩ : ١٥ سنة من العمر من كل من الذكور والإناث على حد سواء) وهى تعبر عن مستقبلنا المنتج وطاقتنا المحركة فى المجتمع كما أن الدراسات السابقه عن مرض صرع النوبات الأولية الكبرى وعلاقته بمرض وصرع الفص الصدغى على الأطفال وخاصة فى النواحى المعرفيه والنفسية كانت قليله ومن هنا جاء التفكير فى عمل هذا البحث .

خطوات البحث :

وعينتنا فى هذا البحث اختيرت على أساس التصنيف العالمى لمرضى الصرع لعام ١٩٩٣ وقد اختير ١٤٦ مريضاً من مرضى صرع النوبات الأولية الكبرى و ١٠٦ من مرضى صرع الفص الصدغى و ٩٣ من العينة الضابطة روعى فيها ضوابط السن والظروف الاجتماعية والنفسية والمرضية المختلفة وقد طبقت فى هذا البحث اختبارات نفسية وأخرى معرفية وتم الكشف الطبى والعصبى والنفسى على كل أفراد العينة مع مراعاة استخدام التقنيات الحديثة المختلفة عند الضرورة .

والأدوات المستخدمة هى مقابلة نفسية معدة خصيصاً لهذا الغرض وأخرى لمرضى الصرع هذا مع الاستعانة ببطارية معرفية من الاختبارات تتضمن ثلاثة اختبارات من

★ **وبالتفصيل :**

١ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار الشفرة فى مجموعتى المرض .

وجـد أن هناك فـروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود انخفاض فى الانتباه المتصل والمثابرة والسرعة النفس حركية والتوافق البصرى الحركى والقدرة على التعلم فى :

ـ العائلات ذات الدخل المرتفع (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

ـ المرضى الذين يعيشون فى غرفتين أو أقل وكذلك المرضى الذين لا يتمتع أهاليهم بقسط من التعليم (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٢ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار إعادة الأرقام فى مجموعتى المرض .

وجـد أن هناك فـروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود انخفاض فى أى أو كل من الذاكرة المباشرة والانتباه السمعى والذاكرة القريبة والاستبصار الداخلى فى :

ـ المرضى الذكور (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

٣ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار شطب الحروف (الزمن) فى مجموعتى المرض .

وجـد أن هناك فـروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود انخفاض فى الانتباه ومعدل الاستجابة فى :

ـ المرضى ذوى حجم العائلة الكبير وكذلك المرضى الذين يتمتع أهاليهم بقسط

‫٭ وبالتفصيل :‬

‫١ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار الشفرة فى‬
‫مجموعتى المرض ،‬

‫وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع‬
‫الفص الصدغى وهذا يعنى وجود انخفاض فى الانتباه المتصل والمثابرة والسرعة النفس‬
‫حركية والتوافق البصرى الحركى والقدرة على التعلم فى :‬

‫ـ العائلات ذات الدخل المرتفع (مرضى صرع الفص الصدغى أكثر من مرضى‬
‫صرع النوبات الكبرى الأولية) .‬

‫ـ المرضى الذين يعيشون فى غرفتين أو أقل وكذلك المرضى الذين لا يتمتع أهاليهم‬
‫بقسط من التعليم (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص‬
‫الصدغى) .‬

‫٢ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار إعادة الأرقام فى‬
‫مجموعتى المرض ،‬

‫وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع‬
‫الفص الصدغى وهذا يعنى وجود انخفاض فى أى أو كل من الذاكرة المباشرة والانتباه‬
‫السمعى والذاكرة القريبة والاستبصار الداخلى فى :‬

‫ـ المرضى الذكور (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات‬
‫الكبرى الأولية) .‬

‫٣ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار شطب الحروف (‬
‫الزمن) فى مجموعتى المرض ،‬

‫وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع‬
‫الفص الصدغى وهذا يعنى وجود انخفاض فى الانتباه ومعدل الاستجابة فى :‬

‫ـ المرضى ذوى حجم العائلة الكبير وكذلك المرضى الذين يتمتع أهاليهم بقسط‬

مناسب من التعليم (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٤ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار شطب الحروف (الأخطاء) فى مجموعتى المرض ،

ـ لم توجد أية فروق دالة بين مجموعتى المرضى .

٥ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار الاختيارات المألوفة (الزمن العام ومتوسط الزمن) فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى الاندفاعية فى :

ـ مرضى الأماكن الفقيرة (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

٦ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار الاختيارات المألوفة (الأخطاء) فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى الاندفاعية فى :

ـ العائلات ذات الحجم الكبير وفى مرضى الأماكن الفقيرة وفى الأطفال المتقدمين فى الترتيب بين إخوتهم (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

٧ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار الاكتئاب فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى الاكتئاب فى :

ـ المرضى الذكور والمرضى من العائلات نوى الحجم الكبير والمرضى المتأخرين فى

الترتيب العائلى والمرضى القاطنين بغرفتين أو أقل والأطفال المرغوبين أثناء الحمل والمرضى نوى الأهالى المنفصلة وكذلك المرضى نوى العائلات مرتفعة الدخل والمرضى الذين لم ينال أهاليهم قسط من التعليم (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٨ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار آيزنك للشخصية (سمة السيكوباتية) فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة السيكوباتية فى :

ـ المرضى الذكور والمرضى نوى الترتيب المتقدم بين إخوتهم والمرضى غير المرغوبين أثناء الحمل (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

٩ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار آيزنك (سمة العصاب) فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة العصاب فى :

ـ المرضى الإناث (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

١٠ ـ بالنسبة لأثر المتغيرات الاجتماعية المختلفة على اختبار آيزنك للشخصية (سمة الانطواء) فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة الانطواء فى :

ـ مرضى العائلات ذات الحجم الكبير (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

ثانيا : بالنسبة للمتغيرات المتعلقة بالمرض :

★ بوجه عام :

وجدنا فى دراستنا هذه ما يلى :

من ناحية بداية المرض :

لم نجد فى دراستنا أية فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بسن البداية ونوع النوبات سواء كانت مفاجئة أو كانت بالتدريج .

ومن ناحية تردد النوبات :

(١) تردد النوبات الحالى :

وجد فى هذه الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فى تردد النوبات الحالى ، وقد زاد مرضى صرع النوبات الكبرى الأولية عن مرضى صرع الفص الصدغى الذين تنتابهم النوبات بصفة دورية كل شهر وفى المرضى الذين توقفت عنهم النوبات فى الوقت الحالى ، بينما زاد مرضى صرع الفص الصدغى عن مرضى صرع النوبات الكبرى الأولية الذين تنتابهم النوبات بصفة دورية كل يوم وكذلك الذين تنتابهم النوبات بصفة دورية كل أسبوع فى تكرارها الحالى .

(٢) تردد النوبات عند بداية الحالة :

أظهرت الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بتكرار النوبات عند بداية المرض بصفة دورية كل أسبوع وكذلك تكرارها بصفة دورية كل شهر .

المــــــدة :

(١) مدة المرض :

أظهرت الدراسة فروقاً دالة بين الزيادة فى مجموعة مرضى صرع النوبات الكبرى الأولية عنها فى مرضى صرع الفص الصدغى فيما يختص بالمرضى الذين قلت عندهم مدة المرض عن ستة أشهر وأولئك الذين تراوحت مدة المرض لديهم بين ستة أشهر وعامين .

وعلى الوجه الآخر تبينت الزيادة الدالة فى مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية فيما يختص بمدة المرض التى تزيد عن خمسة سنوات وكذلك مدة المرض التى تتراوح بين سنتين إلى خمس سنوات .

(٢) المدة التى اختفت فيها النوبات عن المرضى :

أوضحت دراستنا هذه وجود فروق دالة بين مجموعتى المرضى فيما يختص بطول المدة التى خلت منها النوبات ، وقد ظهر منها وجود زيادة فى مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى فى أولئك الذين تراوحت المدة الخالية من النوبات بين ستة أشهر وسنتين ، بينما زادت مجموعة مرضى صرع الفص الصدغى عن مرضى صرع النوبات الكبرى الأولية فى أولئك الذين كانت المدة الخالية من النوبات لديهم أقل من ستة أشهر .

وجود النوبات الصرعية المستمرة :

لم تكن هناك فروقاً دالة فى هذه الدراسة بين مجموعتى مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بالنوبات الصرعية المستمرة .

وجود تاريخ سابق للمرض فى العائلة :

لم توضح الدراسة فروقاً دالة بين مجموعتى المرضى فيما يختص بالتاريخ السابق للعائلة .

وجود تاريخ سابق للمرض عند الشخص ذاته :

لم توضح الدراسة فروقاً دالة بين مجموعتي المرضى فيما يختص بالتاريخ السابق للمرض .

التغيرات المحيطة بالنوبات ذاتها :

أظهرت الدراسة فروقاً دالة أوضحت الزيادة لدى مرضى صرع الفص الصدغي عن مرضى صرع النوبات الكبرى الأولية فيما يختص بكل التغيرات المحيطة بالنوبات سواء قبلها أو أثناءها أو بعدها .

أسباب المرض :

أظهرت الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغي فيما يختص بالأسباب المخية والأسباب العامة والأسباب الناتجة عن خطأ الطبيب .

وقد أظهر مرضى صرع النوبات الكبرى الأولية ميلاً أكبر لوجود أسباب مخية وأخرى ناتجة عن خطأ الطبيب ، بينما أظهر مرضى صرع الفص الصدغي ميلاً أكثر لوجود أسباب عامة .

الاضطرابات النفسية المصاحبة للمرض :

لم تظهر هذه الدراسة فروقاً دالة بين مرض صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغي فيما يختص بالاضطرابات النفسية المصاحبة للمرض .

على أن مرضى صرع الفص الصدغي أبدوا قابلية لاضطرابات الاتصال بالآخرين واضطرابات الجنس والقلق ، بينما أبدى مرضى صرع النوبات الكبرى الأولية قابلية لاضطرابات التحول .

الأمراض العضوية :

أظهرت دراستنا وجود ميلاً دالاً من مرضى صرع النوبات الكبرى الأولية لوجود

أمراض عضوية أكثر من مرضى صرع الفص الصدغى .

وجود مشاكل فى الحمل والولادة ووجود سمات عصابية :

لم نجد فى هذه الدراسة أية فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بوجود مشاكل فى الحمل أو الولادة أو بوجود سمات عصابية .

٭ وبالتفصيل :

عند مقارنة نتائج أداء مجموعتى المرضى على مختلف الاختبارات المعرفية والنفسية من خلال شتى المتغيرات المتعلقة بطبيعة المرض وتاريخه وأسبابه ، وجد ما يلى :

١ ـ بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار الشفرة فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى الانتباه المستمر والمثابرة والسرعة النفس حركية والتوافق البصرى الحركى والقدرة على التعلم فى :

ـ المرضى الذين كانت بداية المرض لديهم فى سن أكبر من أربعة سنوات ، والمرضى الذين أصابهم المرض سواء بشكل مفاجىء أو بالتدريج فى بدايته وكذلك المرضى الذين كانت فترة امتنعت فيها النوبات عنهم من ستة أشهر إلى سنتين أو أقل من ستة أشهر على السواء والمرضى الذين لم يكن فى تاريخهم المرضى ذكر لنوبات مستمرة والمرضى الذين ليس فى تاريخهم إصابة عائلية بالمرض وكذلك المرضى الذين لهم تاريخ سابق بالمرض والمرضى الذين لم يصابوا بمرض عضوى وكذلك المرضى المصابين بسمات عصابية والآخرين الذين لديهم تغيرات مصاحبة للتشنجات سواء كانت قبلها أو أثناها أو بعدها (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

ـ المرضى الذين كانت مدة المرض لديهم أكثر من خمسة سنوات وألئك غير المصابين بسمات عصابية (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٢ - بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار إعادة الأرقام فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى أى أو كل من الذاكرة المباشرة والانتباه السمعى والذاكرة القريبة والاستبصار الداخلى فى :

- المرضى المصابين بمرض عضوى (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

- المرضى ذوى بداية المرض المفاجئة وأولئك الذين كانت مدة غيبة النوبات عنهم لا تتعدى ستة أشهر (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٣ - بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار المتشابهات فى مجموعتى المرض .

وجد أن هناك فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى تكوين المفهوم والتفكير التجريبى والقدرات العقلية العامة فى :

- المرضى الذين لم تتعد الفترة التى خلت من النوبات مدة ستة أشهر والمرضى المصابين بتاريخ مرض إيجابى (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٤ - بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار شطب الحروف (الزمن) فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى الانتباه وقدرة رد الفعل فى :

- المرضى الذين كان تردد النوبات عليهم فى بداية المرض سريعاً كل أسبوع

(مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

– المرضى الذين كانت بداية مرضهم تدريجياً والآخرين الذين لم يصابوا بنوبات صرعية مستمرة وأولئك المصابين بمرض عضوى فى تاريخهم المرضى وأيضا الذين أصيبوا باضطرابات أثناء الحمل والذين تأّلبت عليهم العناصر المؤثرة فى النوبات (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٥ – بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار شطب الحروف (الأخطاء) فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى الانتباه ومعدل الاستجابة فى :

– المرضى الذين كان تردد النوبات لديهم فى بداية الحالة دورياً فى كل يوم (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

– المرضى الذين لم يكن لهم تاريخ مرضى سابق (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٦ – بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار الاختيارات المألوفة (الزمن العام ومتوسط الزمن) فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى الاندفاعية فى :

– المرضى الذين لم تكن لديهم مشاكل مرضية عند الولادة (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

– المرضى الذين كانت فترة خلو النوبات لديهم أقل من ستة أشهر (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٧ ـ بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار الاختيارات المألوفة (الأخطاء) فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى الاندفاعية فى :

ـ المرضى الذين تتراوح مدة مرضهم بين ستة أشهر وسنتين وأولئك الذين كان لديهم مرض عضوى والمرضى الذين لم تكن لديهم مشاكل أثناء الحمل وكذلك الذين لم تكن لديهم سمات عصابية (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

ـ المرضى الذين كانت بداية مرضهم تدريجية (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٨ ـ بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار القلق فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى القلق فى :

ـ المرضى الذين كان تردد الحالة لديهم عند بداية المرض بصفة دورية كل شهر وأولئك الذين ظهر أن أسباب مرضهم مخية (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

ـ المرضى الذين كانت بداية مرضهم تدريجية (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٩ ـ بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار الاكتئاب فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى الاكتئاب فى :

- مرضى الذين كان تردد النوبات عندهم فى بداية المرض بصفة دورية كل يوم والمرضى الذين كان تردد النوبات الحالى لديهم دورياً كل أسبوع وأولئك الذين أصيبوا بنوبات مستمرة والمرضى نوى التاريخ السابق للمرض (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

- المرضى المصابين بسمات عصابية (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

١٠ ـ بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار آيزنك للشخصية (سمة السيكوباتية) فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة السيكوباتية فى :

- المرضى الذين كانت مدة المرض لديهم أقل من ستة شهور وأولئك الذين لم تكن لديهم نوبات صرعية مستمرة والمرضى الذين كانت لديهم تغيرات حول حدوث النوبات (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

١١ ـ بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار آيزنك (سمة العصاب) فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة العصاب فى :

- المرضى الذين كانت بداية المرض لديهم تدريجياً والمرضى الذين لم يكن لهم تاريخ مسبق بالمرض وأولئك الذين تضافرت عليهم العناصر المؤثرة فى المرض (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

١٢ ـ بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار آيزنك للشخصية (سمة الانطواء) فى مجموعتى المرض .

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع

الفص الصدغى وهذا يعنى وجود زيادة فى سمة الانطواء فى :

– المرضى الذين كانت بداية مرضهم فى سن أكبر من أربع سنوات والمرضى الذين كان تكرار النوبات فى بداية المرض بصفة دورية كل أسبوع وأولئك الذين كان تكرار نوباتهم الحالى بصفة دورية كل يوم والمرضى الذين زادت مدة المرض لديهم عن خمس سنوات وأولئك الذين كانت أكبر فترة خلت من النوبات أقل من ستة أشهر أو من ستة أشهر إلى عامين والمرضى الذين لم يكن لديهم نوبات صرعية مستمرة والمرضى الذين لم يكن لهم تاريخ مسبق للمرض وأولئك الذين أصابتهم التغيرات المصاحبة للنوبة سواء قبلها أو أثناءها أو بعدها والمرضى الذين تضافرت عليهم العناصر المؤثرة فى النوبة وأولئك الذين كان سبب مرضهم من الأسباب العامة (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

١٣ – بالنسبة لأثر المتغيرات المتعلقة بالمرض على اختبار آيزنك للشخصية (سمة الكذب) فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة الكذب فى :

– المرضى الذين كانت بداية مرضهم مفاجئة (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

– المرضى الذين تراوحت أعمارهم عند بداية مرضهم من سن سنتين إلى أربعة سنوات وأولئك الذين كانت لديهم متاعب أثناء الحمل فيهم (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

ثالثا : الجوانب الدوائية :

* بوجه عام :

(١) بالنسبة لنوع العلاج :

فى دراستنا هذه توجد فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى

صرع الفص الصدغى بالنسبة لنوع العلاج سواء كان المريض يتناول دواءً واحداً من مضادات الصرع أو أنه يتناول أكثر من دواء منها وهى تبين ميل مرضى صرع النوبات الكبرى الأولية لأخذ دواء واحد من مضادات الصرع بينما يميل مرضى صرع الفص الصدغى لأخذ أكثر من دواء منها .

(٢) بالنسبة للأعراض الجانبية لكل دواء على حدة :

أظهرت الدراسة أن مرضى صرع النوبات الكبرى الأولية يعانون بدرجة دالة من الأعراض الجانبية لعقار الكاربامازبين (التيجرتول) أكثر من مرضى صرع الفص الصدغى .

(٣) كمية الدواء المأخوذة :

أ – العلاج بدواء واحد من مضادات الصرع :

لم تظهر الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى بالنسبة للعلاج بعقار الفينيتوين (الإيبانوتين) .

وعلى الجانب الآخر ، أظهرت الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فى تناول عقار الكاربامازبين (التيجرتول) ، [مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى] .

كما أظهرت فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يتعلق بتناول عقارى الصوديوم فالبروات (الديباكين) والكلونازيبام (الريفوتريل) كل على حدة [ومرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية] .

ب – العلاج بأكثر من دواء واحد من مضادات الصرع :

لم تظهر الدراسة أية فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يتعلق بتناول أكثر من دواء واحد من مضادات الصرع الآتية :

− عقارى الكاربامازبين (التيجرتول) / والفينيتوين (الإيبانوتين) .

− الصوديوم ڤالبروات (الديباكين) / والفينيتوين (الإيبانوتين) .

− الصوديوم ڤالبروات (الديباكين) / والكلونازيبام (الريڤوتريل) .

− الصوديوم ڤالبروات (الديباكين) / والكلونازيبام (الريڤوتريل) / والكاربامازبين (التيجرتول) .

− الصوديوم ڤالبروات (الديباكين) / والفينيتوين (الإيبانوتين) / والكاربامازبين (التيجرتول) .

− الكلونازيبــام (الريڤـوتريل) / والفـينيتـوين (الإيبـانوتين) / والكاربامــازبين (التيجرتول) .

على أن الدراسة قد أظهرت فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فى تناول أكثر من عقار مضاد للصرع بينت أن هناك ميلاً لمرضى صرع الفص الصدغى لتناول عقارى الصوديوم ڤالبروات (الديباكين) / والكاربامازبين (التيجرتول) معاً .

(٤) مدة تناول الدواء :

أظهرت الدراسة وجود فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بمدة تناول الدواء ، وذلك فى المرضى الذين زادت مدة مرضهم عن خمس سنوات (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

(٥) التوائم مع العلاج :

أ − بالنسبة للمرضى المتوائمين :

لم تظهر الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بالتوائم مع العلاج بشكل عام .

بينمـا أظهرت فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيمـا يختص بالتوازم مع العلاج بالنسبة لنوع المريض المتوائم على دواء واحد (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

ب – بالنسبة للموضى غير المتوائمين :

لم تظهر الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بعدم التوازم مع العلاج بشكل عام .

بينمـا أظهرت فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بعدم التوازم مع العلاج بالنسبة لنوع المريض غير المتوائم على دواء واحد (مـرضى صـرع النوبات الكبـرى الأوليـة أكـثر من مـرضى صـــرع الفـص الصدغى) .

(٦) السيطرة على النوبات :

أ – بالنسبة للمرضى نوى النوبات تحت السيطرة :

لم تظهر الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بالمرضى نوى النوبات تحت السيطرة بشكل عام .

بينمـا أظهرت فروقـاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بالتوازم مع العلاج بالنسبة لنوع المريض نو النوبات تحت السيطرة على دواء واحد (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

ب – بالنسبة للمرضى نوى النوبات التى ليست تحت السيطرة :

لم تظهر الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بالمرضى نوى النوبات التى ليست تحت السيطرة بشكل عام .

بينمـا أظهرت فروقـاً دالة بين مرضى صـرع النوبات الكبرى الأولية ومرضى صرع

الفص الصدغى فيما يختص بالمرضى ذوى النوبات التى ليست تحت السيطرة بالنسبة لنوع المريض نو النوبات التى ليست تحت السيطرة على دواء واحد من مـضادات الصـرع (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صـرع الفـص الصدغى) .

(۷) بالنسبة للأعراض الجانبية للمرض :

لم تظهر الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بالأعراض الجانبية للمرض .

(۸) نسبة الدواء فى الدم :

لم تظهر الدراسة فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بنسبة الدواء فى الدم .

★ وبالتفصيل :

عند مقارنة نتائج أداء مجموعتى المرضى على مختلف الاختبارات المعرفية والنفسية من خلال شتى المتغيرات المتعلقة بالجوانب الدوائية المختلفة ، وجد ما يلى :

۱ - بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار النفرة فى مجموعتى المـــرض ،

وجـد أن هناك فروق دالة بين مـرضى صـرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى الانتباه المستمر والمثابرة والسرعة النفس حركية والتوافق البصرى الحركى والقدرة على التعلم فى :

- المرضى الذين يتناولون كميات قليلة من عقار الكاربامازبين (التيجرتول) والمرضى المتواثمين على أكثر من دواء من مضادات الصرع وبكميات مناسبة والمرضى الذين لم تنتظم لديهم النوبات رغم استمرارهم على عقار واحد من مضادات الصـرع (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأوليـة) .

- المرضى الذين يتناولون عقار الكاربامازبين (التيجرتول) سواء كان بكميات قليلة

أو كثيرة (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٢ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار إعادة الأرقام فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى أى أو كل من الذاكرة المباشرة والانتباه السمعى والذاكرة القريبة والاستبصار الداخلى فى :

– المرضى الذين يتناولون عقار واحد بوجه عام والمرضى الذين يتناولون عقار الكاربامازين (التيجرتول) – (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

– المرضى الذين يتناولون عقار الكاربامازين (التيجرتول) وحده وبكميات كبيرة وكذلك المرضى الذين يتناولون أكثر من عقار من مضادات الصرع وبجرعات كبيرة والمرضى المتوائمين على العلاج بأكثر من عقار واحد من مضادات الصرع (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٣ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار المتشابهات فى مجموعتى المرض ،

وجد أن هناك فروقاً دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى تكوين المفهوم والتفكير التجريبى والقدرات العقلية العامة فى :

– المرضى الذين ذوى النوبات التى ليست تحت السيطرة بصفة عامة (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

٤ – بالنسبة لأثر المتغيرات المتعلقة على اختبار شطب الحروف (الزمن) فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود نقص فى الانتباه وقدرة رد الفعل فى :

- المرضى المتوائمين مع العلاج بصفة عامة والمرضى المتوائمين على عقار واحد والمرضى نوى النوبات تحت السيطرة بشكل عام والمرضى نوى النوبات تحت السيطرة على عقار واحد (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغـــى) .

٥ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار شطب الحروف (الأخطاء) فى مجموعتى المرض ،

لم تظهر الدراسة أية فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار شطب الحروف (الأخطاء) فى مجموعتى المرض .

٦ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار الاختيارات المألوفة (الزمن العام ومتوسط الزمن) فى مجموعتى المرض ،

لم تظهر الدراسة أية فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار الاختيارات المألوفة (الزمن العام ومتوسط الزمن) فى مجموعتى المرض .

٧ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار الاختيارات المألوفة (الأخطاء) فى مجموعتى المرض ،

لم تظهر الدراسة أية فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار الاختيارات المألوفة (الأخطاء) فى مجموعتى المرض .

٨ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار القلق فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى القلق فى :

- المرضى الذين يتناولون دواء واحد من عقار الكاربامازبين (التيجرتول) بجرعات كبيـرة والمرضى نوى النوبات التى تحت السـيطرة بشكل عـام (مـرضى صـرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

- المرضى الذين يتناولون أكثر من عقار واحد ن مضادات الصرع متمثلاً فى عقارى الكاربامازبين (التيجرتول) / والصـوديوم ڤـالبروات (الديباكين) - (مـرضى صـرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

٩ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار الاكتئاب فى مجموعتى المرض ،

وجد أن هناك فـروق دالة بين مـرضى صـرع النوبات الكبرى الأولية ومرضى صـرع الفص الصدغى وهذا يعنى وجود زيادة فى الاكتئاب فى :

- المرضى الذين يتناولون دواء واحد من عقار الكاربامازبين (التيجرتول) بشكل عام (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

١٠ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار آيزنك للشخصية (سمة السيكوباتية) فى مجموعتى المرض ،

وجد أن هناك فـروق دالة بين مـرضى صـرع النوبات الكبرى الأولية ومرضى صـرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة السيكوباتية فى :

- المرضى الذين يتناولون أكثر من دواء من مضادات الصـرع بجرعات كبيرة (مرضى صرع النوبات الكبرى الأوليــة أكثر من مرضى صرع الفص الصدغى) .

١١– بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار آيزنك (سمة العصاب) فى مجموعتى المرض ،

لم تظهر الدراسـة أية فـروق دالة بين مـرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى فيما يختص بأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار آيزنك (سمة العصاب) فى مجموعتى المرض .

١٢ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار آيزنك للشخصية (سمة الانطواء) فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة الانطواء فى :

- المرضى المتوائمين مع العلاج بشكل عام والمرضى غير المتوائمين مع العلاج بشكل عام أيضاً والمرضى غير المتوائمين مع العلاج على دواء واحد والمرضى نوى النوبات التى ليست تحت السيطرة بشكل عام والذين يتناولون دواء واحد مضاد للصرع أو عدة أدوية (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

١٣ – بالنسبة لأثر المتغيرات المتعلقة بالجوانب الدوائية المختلفة على اختبار آيزنك للشخصية (سمة الكذب) فى مجموعتى المرض :

وجد أن هناك فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغى وهذا يعنى وجود زيادة فى سمة الكذب فى :

- المرضى ذوى النوبات التى تحت السيطرة بشكل عام (مرضى صرع الفص الصدغى أكثر من مرضى صرع النوبات الكبرى الأولية) .

- المرضى الذين يتناولون عقاراً واحداً هو عقار الصوديوم فالبروات (الديباكين) سواء بكميات قليلة أو بشكل عام والمرضى الذين يتناولون دواء واحد من مضادات الصرع والمرضى غير المتوائمين على أكثر من دواء واحد من ألوية الصرع والمرضى نوى النوبات التى ليست تحت السيطرة بشكل عام والمرضى نوى النوبات التى ليست تحت السيطرة مع تناول أكثر من عقار من مضادات الصرع (مرضى صرع النوبات الكبرى الأولية أكثر من مرضى صرع الفص الصدغى) .

رابعا : الفحوص الطبية :

١- رسم المـــخ :

* بوجه عام :

بالنسبة لرسم المخ لمرضى صرع النويات الكبرى الأولية أظهرت الدراسة وجود ٧٨ر١٨٪ من المرضى مصابين بنويات صرعية تحت القشرة المخية ، ١٩ر٢٪ منهم مصابين ببؤرة صرعية بالفص الصدغى مع تعميم ثانوى ، ٧ر٢٪ منهم بدون تغيرات فى رسم المخ .

وعلى الجانب الآخر ، بالنسبة لرسم المخ لمرضى صرع الفص الصدغى يوجد ٣٣ر٢٪ مصابين ببؤرة صرعية صدغية يمنى ، ٧٧ر٥٣٪ منهم مصابين ببؤرة صرعية صدغية يسرى ، ٢١ر١٣٪ مصابين ببؤرتين صرعيتين صدغية يمنى ويسرى .

* وبالتفصيل :

عند مقارنة نتائج أداء مجموعتى المرضى على مختلف الاختبارات المعرفية والنفسية من خلال شتى المتغيرات المتعلقة باختلافات رسم المخ الكهربائى ، وجد ما يلى :

١ – بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار التفرة فى مجموعتى المـــرض .

وجد أن هناك فروق دالة بين المرضى ذوى البؤرة الصدغية اليمنى فى رسم المخ الكهربائى والمرضى ذوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى وهذا يعنى وجود نقص فى الانتباه المستمر والمثابرة والسرعة النفس حركية والتوافق البصرى الحركى والقدرة على التعلم فى المرضى ذوى البؤرة الصدغية اليمنى فى رسم المخ الكهربائى أكثر من المرضى ذوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى.

٢ – بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار إعادة الأرقام فى مجموعتى المرض .

وجد أن هناك فروق دالة بين المرضى نوى البؤرة تحت القشرة المخية فى رسم المخ الكهربائى والمرضى ذوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى ، وتوجد فروق

دالة أخرى بين المرضى نوى البؤرة الصدغية اليمنى والمرضى ذوى البؤرة الصدغية اليسرى وهذا يعنى وجود نقص فى أى أو كل من الذاكرة المباشرة والانتباه السمعى والذاكرة القريبة والاستبصار الداخلى فى المرضى نوى البؤرة تحت القشرة المخية فى رسم المخ الكهربائى أكثر من المرضى نوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى وفى المرضى نوى البؤرة الصدغية اليمنى أكثر من المرضى نوى البؤرة الصدغية اليسرى

٣ - بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار المتشابهات فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين المرضى نوى البؤرة تحت القشرة المخية فى رسم المخ الكهربائى والمرضى ذوى البؤرة الصدغية مع تعميم ثانوى فى رسم المخ الكهربائى وهذا يعنى وجود نقص فى تكوين المفهوم والتفكير التجريبى والقدرات العقلية العامة فى المرضى ذوى البؤرة تحت القشرة المخية فى رسم المخ الكهربائى أكثر من المرضى نوى البؤرة الصدغية مع تعميم ثانوى فى رسم المخ الكهربائى .

٤ - بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار شطب الحروف (الزمن) فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين المرضى ذوى البؤرة الصدغية اليمنى فى رسم المخ الكهربائى والمرضى نوى البؤرة الصدغية اليمنى مع تعميم ثانوى فى رسم المخ الكهربائى وهذا يعنى وجود نقص فى الانتباه وسرعة الاستجابة فى المرضى نوى البؤرة الصدغية اليمنى فى رسم المخ الكهربائى أكثر من المرضى ذوى البؤرة الصدغية اليمنى مع تعميم ثانوى فى رسم المخ الكهربائى

٥ - بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار شطب الحروف (الأخطاء) فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين المرضى ذوى البؤرة الصدغية اليمنى فى رسم المخ الكهربائى والمرضى نوى البؤرة الصدغية اليمنى مع تعميم ثانوى فى رسم المخ الكهربائى وهذا يعنى وجود نقص فى الانتباه وسرعة الاستجابة فى المرضى نوى البؤرة الصدغية

اليمنى فى رسم المخ الكهربائى أكثر من المرضى نوى البؤرة الصدغية اليمنى مع تعميم
ثانوى فى رسم المخ الكهربائى

٦ ـ بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار الاختيارات المألوفة (الزمن العام ومتوسط الزمن) فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين المرضى نوى البؤرة تحت القشرة المخية فى رسم المخ
الكهربائى والمرضى نوى رسم المخ الكهربائى الطبيعى وهذا يعنى زيادة الاندفاعية فى
المرضى نوى البؤرة تحت القشرة المخية فى رسم المخ الكهربائى أكثر من المرضى نوى رسم
المخ الكهربائى الطبيعى

٧ ـ بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار الاختيارات المألوفة (الأخطاء) فى مجموعتى المرض ،

وجد أن هناك فروق دالة بين المرضى نوى البؤرة تحت القشرة المخية فى رسم المخ
الكهربائى والمرضى نوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى وكذلك المرضى
نوى البؤرة الصدغية اليمنى والمرضى نوى البؤرة الصدغية اليسرى وأيضاً بين المرضى
نوى البؤرة تحت القشرة المخية والمرضى نوى التعميم الثانوى بشكل عام وهذا يعنى أن
المرضى نوى البؤرة تحت القشرة المخية فى رسم المخ الكهربائى أكثر من المرضى نوى
البؤرة الصدغية اليسرى فى رسم المخ الكهربائى وكذلك المرضى نوى البؤرة الصدغية
اليمنى أكثر من المرضى نوى البؤرة الصدغية اليسرى وأيضاً بين المرضى نوى البؤرة تحت
القشرة المخية أكثر من المرضى نوى التعميم الثانوى بشكل عام .

٨ ـ بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار القلق فى مجموعتى المرض ،

لم تظهر فى هذه الدراسة أية فروق دالة بين مجموعتى المرض بخصوص التغيرات فى
رسم المخ الكهربائى .

٩ - بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار الاكتئاب فى مجموعتى المرض :

وجد أن هناك فروق دالة بين المرضى نوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى والمرضى نوى البؤرة الصدغية مع تعميم ثانوى فى رسم المخ الكهربائى وهذا يعنى زيادة الاكتئاب فى المرضى نوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى أكثر من المرضى نوى البؤرة الصدغية مع تعميم ثانوى فى رسم المخ الكهربائى .

١٠ - بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار آيزنك للشخصية (سمة السيكوباتية) فى مجموعتى المرض :

لم تظهر فى هذه الدراسة أية فروق دالة بين مجموعتى المرض بخصوص التغيرات فى رسم المخ الكهربائى .

١١- بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار آيزنك (سمة العصاب) فى مجموعتى المرض :

لم تظهر فى هذه الدراسة أية فروق دالة بين مجموعتى المرض بخصوص التغيرات فى رسم المخ الكهربائى .

١٢ - بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائى على اختبار آيزنك للشخصية (سمة الانطواء) فى مجموعتى المرض :

وجد أن هناك فروق دالة بين المرضى نوى البؤرة الصرعية تحت القشرة المخية فى رسم المخ الكهربائى والمرضى نوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى وكذلك بين المرضى نوى البؤرة الصدغية اليمنى والمرضى ذوى البؤرة الصدغية اليسرى مع تعميم ثانوى وهذا يعنى زيادة الانطواء فى المرضى نوى البؤرة الصرعية تحت القشرة المخية فى رسم المخ الكهربائى أقل من المرضى نوى البؤرة الصدغية اليسرى فى رسم المخ الكهربائى وكذلك بين المرضى نوى البؤرة الصدغية اليمنى أكثر من المرضى ذوى البؤرة الصدغية اليسرى مع تعميم ثانوى .

۱۲ – بالنسبة لأثر المتغيرات المتعلقة باختلافات رسم المخ الكهربائي على اختبار آيزنك للشخصية (سمة الكذب) في مجموعتي المرض ،

لم تظهر في هذه الدراسة أية فروق دالة بين مجموعتي المرض بخصوص التغيرات في رسم المخ الكهربائي .

ثانيا : القياس النفسي :

۱ – اختبار الشفرة ،

أظهرت الدراسة وجود فروق دالة بين مرضى صرع النوبات الكبرى الأولية ومرضى صرع الفص الصدغي والمجموعة الضابطة على الأداء في اختبار الأنوفا ذات الاتجاه الواحد ، كما أظهرت وجود زيادة دالة بالمجموعة الضابطة أكثر من مجموعتي المرضى على الأداء في اختبار (ت) وذلك في الأداء على اختبار الشفرة .

۲ – اختبار إعادة الأرقام ،

أظهرت الدراسة عدم وجود فروق دالة بين مجموعتي المرضى والمجموعة الضابطة على الأداء في كل من الاختبارين الأنوفا ذات الاتجاه الواحد واختبار (ت) .

۳ – اختبار المتشابهات ،

أظهرت الدراسة وجود فروق دالة بين الثلاث مجموعات (مجموعتي المرضى والمجموعة الضابطة) على الأداء في اختبار الأنوفا ذات الاتجاه الواحد ، وأظهرت زيادة دالة في المجموعة الضابطة أكثر من مجموعتي المرض على اختبار (ت) ، كما أظهرت وجود زيادة دالة في مرضى صرع الفص الصدغي أكثر من مرضى صرع النوبات الكبرى الأولية على اختبار (ت) .

٤ – اختبار تعلب الحروف (الأخطاء) ،

أظهرت الدراسة وجود فروق دالة بين الثلاث مجموعات (مجموعتي المرضى والمجموعة الضابطة) على اختبار الأنوفا ذات الاتجاه الواحد ، كما أظهرت وجود زيادة دالة في المجموعة الضابطة أكثر من مجموعتي المرضى على اختبار (ت) ، ولم تظهر الدراسة أية

فروق دالة بين مجموعتى المرضى على اختبار (ت) .

٥ – اختبار شطب الحروف (الزمن) ،

أظهرت الدراسة عدم وجود أية فروق دالة بين الثلاث مجموعات (مجموعتى المرضى والمجموعة الضابطة) على اختبار الأنوفا ذات الاتجاه الواحد أو على اختبار (ت) .

٦ – اختبار الانتقاء الاختيارى للصور المألوفة (الأخطاء) ،

لم تظهر الدراسة فروقاً دالة بين المجموعات الثلاثة (مجموعتى المرضى والمجموعة الضابطة) فى اختبار الأنوفا ذات الاتجاه الواحد بينما أظهرت وجود زيادة دالة فى المجموعة الضابطة أكثر من مجموعة صرع الفص الصدغى على الأداء فى اختبار (ت) ، ولا توجد فروق دالة بين مجموعتى المرضى على الأداء فى اختبار (ت) .

٧ – اختبار الانتقاء الاختيارى للصور المألوفة (الزمن الكلى) ،

لم تظهر الدراسة أية فروق دالة بين الثلاث مجموعات (مجموعتى المرضى والمجموعة الضابطة) على الأداء فى اختبار الأنوفا ذات الاتجاه الواحد بينما أظهرت وجود زيادة دالة فى مجموعة مرضى صرع النويات الكبرى الأولية أكثر من المجموعة الضابطة وأيضاً فى مجموعة مرضى صرع النويات الكبرى الأولية أكثر من مجموعة صرع الفص الصدغى ، ولم تظهر الدراسة فروقاً دالة بين مجموعة مرضى صرع الفص الصدغى والمجموعة الضابطة على الأداء فى اختبار (ت) .

٨ – اختبار الانتقاء الاختيارى للصور المألوفة (متوسط الزمن) ،

لم تظهر الدراسة أية فروق دالة بين الثلاثة مجموعات (مجموعتى المرضى والمجموعة الضابطة) على الأداء فى اختبار الأنوفا ذات الاتجاه الواحد بينما أظهرت وجود زيادة دالة فى مجموعة مرضى صرع النويات الكبرى الأولية أكثر من المجموعة الضابطة وأيضاً فى مجموعة مرضى صرع الفص الصدغى أكثر من مجموعة مرضى صرع النويات الكبرى الأولية ، ولم تظهر الدراسة فروقاً دالة بين مجموعة مرضى صرع الفص الصدغى والمجموعة الضابطة على الأداء فى اختبار (ت) .

٩ - اختبار القلق للأطفال ،

أظهرت الدراسة وجود فروق دالة بين الثلاثة مجموعات (مجموعتى المرضى والمجموعة الضابطة) على الأداء فى اختبار الأنوفا ذات الاتجاه الواحد كما أظهرت الدراسة زيادة مجموعتى المرضى كل على حدة بدرجة دالة أكثر من المجموعة الضابطة وذلك فى الأداء على اختبار الكاى٢ .

١٠- اختبار الاكتئاب للأطفال ،

أظهرت الدراسة وجود فروق دالة بين الثلاثة مجموعات (مجموعتى المرضى والمجموعة الضابطة) على الأداء فى اختبار الأنوفا ذات الاتجاه الواحد ولم تظهر الدراسة أية فروق دالة بين المجموعات الثلاثة فى الأداء على اختبار الكاى٢ .

١١- اختبار أيزنك للأطفال ،

لم تظهر الدراسة أية فروق دالة بين الثلاثة مجموعات (مجموعتى المرضى والمجوعة الضابطة) فى الأداء على اختبار أيزنك للأطفال على الأداء فى كل من اختبار الأنوفا ذات الاتجاه الواحد واختبار الكاى٢ .

المستخلص باللغة العربية

أخذت عينة هذه الرسالة من مرضى الصرع، والذين تم تشخيصهم من خلال الخصائص الاكلينيكية وتخطيط المخى الكهربائى لمرضى الصرع طبقا للتقسيم العالمى ضد الصرع. وقبل البداية فى تجميع البيانات، تم عمل دراسة مسبقة أجريت فى كل من:.

١) مركز الطب النفسى لطلاب المدارس بالدقى.

٢) عيادة الهرم الشاملة، التابع للهيئة العامة للتأمين الصحى.

وبعد الدراسة المبدئية، وجد أن النسبة بين مرضى صرع الفص الصدغى وصرع النوبات الصرعية الكبرى هى ٢ إلى ٣.

وتم استخدام عدة اختبارات منها مقابلة نفسية معدة خصيصا لهذا الغرض واخرى لمرضى الصرع هذا مع الاستعانة ببطارية معرفة من الاختبارات تتضمن ثلاثة اختبارات من اختبار وكسلر للأطفال هى الشفرة وإعادة الأرقام والمتشابهان، هذا بالإضافة إلى اختبار شطب الحروف واختبار الانتفاء الاختيارى للصور المألوفة، أما عن البطارية النفسية والمزاجية فهى تتضمن اختبار الاكتئاب للدكتــور/ غريــب عبــد الفتـاح واختبار القلق للأطفال واختبار ايزنك للأطفال. هذا بالإضافة إلى استخدام استبيان علـــى مختلـف النواحى الاجتماعية التى يتعرض لها المرضى والمجموعة الضابطة على السواء.

وقد وجد من الدراسة وجود فروق دالة بين مختلف القياسات النفسية والمعرفة بين مرضى صــرع الفص الصدغى ومرضى صرع النوبات الصرعية الكبرى، فيما يختص بما يلى:-

١)   المتغيرات الاجتماعية والاقتصادية.

٢)   المتغيرات المتعلقة بالمرض.

٣)   الجوانب الدوائية.

٤)   الفحوص الطبية.

أ) رسم المخ.

ب)   القياس النفسى.

الكلمات الكاشفة

- صرع النوبات الكبرى.

- صرع نوبات الفص الصدغى.

- الصرع.

- المتغيرات الاجتماعية والاقتصادية.

- المتغيرات المتعلقة بالمرض.

- الجوانب الدوائية.

- الفحوص الطبية.

جامعة عين شمس

الكلية :

رســـالـــــة ماجســتير / دكتـــوراه

اسم الطالب   : أشرف عبد الرؤوف محمد عثمان.

عنوان الرسالة : دراسة مقارنة بين الأطفال المصابين بصرع الفص الصدغى والنوبات الصرعيــة الكـبرى الأولية فى بعض المتغيرات المعرفة والشخصية.

اسم الدرجـــة   : (ماجســتير / دكتـــوراه)

لجنة الإشراف   :

١- الاسم/ أ.د/ قدرى محمود حنفى   ٢- الوظيفة/ أستاذ ورئيس قسم الدراسات النفسية/ جامعة عين شمس.

١- الاسم/ أ.د/ عبد اللطيف محمد عثمان ٢- الوظيفة/ أستاذ ورئيس قسم الدراسات العصبية/ جامعة عين شمس.

١- الاسم/ أ.د/ عفاف حامد خليل   ٢- الوظيفة/ أستاذ الأمراض النفسية/ جامعة عين شمس.

تاريخ البحث :   ٣١ / ٢ / ١٩٩٦

الدراسات العليا

ختم الإجازة :   أجيزت الرسالة بتاريخ (√) / √ / ١٩٩ √

موافقة مجلس الجامعة   موافقة مجلس الكلية

١٩٩ / /   ١٩٩٨/ ٤٠ / ١٦

جامعة عين شمس

الكلية :

<u>صفحة العنوان</u>

اسم الطالب   : أشرف عبد الرؤوف محمد عثمان.

الدرجة العلمية : دكتوراه الفلسفة.

القسم التابع له : الدراسات الطبية.

اسم الكلية   : معهد الدراسات العليا للطفولة.

الجامعة   : عين شمس.

سنة التخرج   :

سنة المنح   :

<u>شروط عامة</u>

يوضع شعار الجامعة على الغلاف الخارجى

بسم الله الرحمن الرحيم

« فتعالى الله الملك الحق ولا تعجل بالقرآن من

قبل أن يقضى إليك وحيه وقل ربى زدنى علما »

صدق الله العظيم

شكـــــر

اشكر السادة الأساتذة الذين قاموا بالإشراف وهم :-

١)   أ.د/ قدرى محمود حنفى.

٢)   أ.د/ عبد اللطيف موسى عثمان.

٣)   أ.د/ عفاف حامد خليل.

ثم الاشخاص الذين تعاونوا معى فى البحث وهم :-

(١

(٢

(٣

وكذلك الهيئات الآتية :-:

(١

(٢

(٣

دراسة مقارنة بين الأطفال المصابين بصرع الفص الصدغى والنوبات الصرعية الكبرى الأولية فى بعض المتغيرات المعرفية والشخصية

رسالة مقدمة
للحصول على درجة دكتوراه الفلسفة

فى
دراسات الطفولة - قسم الدراسات الطبية

مقدمة من
طبيب / أشرف عبد الرءوف عثمان

تحت إشراف

<table>
<tr><td>الأستاذة الدكتورة</td><td>الأستاذ الدكتور</td><td>الأستاذ الدكتور</td></tr>
<tr><td>عفاف حامد خليل</td><td>عبد اللطيف موسى عثمان</td><td>قدرى محمود حنفى</td></tr>
<tr><td>أستاذ الأمراض النفسية</td><td>أستاذ ورئيس قسم الأمراض العصبية</td><td>أستاذ ورئيس قسم الدراسات النفسية</td></tr>
<tr><td>كلية الطب</td><td>كلية الطب</td><td>معهد الدراسات العليا للطفولة</td></tr>
<tr><td>جامعة عين شمس</td><td>جامعة الأزهــر</td><td>جامعة عين شمس</td></tr>
</table>

١٩٩٧