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**EXECUTIVE FUNCTION AND MEMORY
DEFICITS IN PATIENTS WITH COMPLEX
PARTIAL SEIZURES OF TEMPORAL
LOBE ORIGIN**

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DECLARATION

This study is entirely my own work and has not been submitted to any other organisation for any other degree. The confidentiality of the participant's scores is guaranteed.

Grant Schofield: _____

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ABSTRACT

A controlled study of the higher function constructs of patients with complex partial seizures of temporal lobe origin (Temporal Lobe Epilepsy) on neuropsychological measures of memory (short-term memory, verbal memory, visual memory and learning) and executive function (perseveration response, shift-of-set, planning abilities, abstract thought). Control Group matched to Experimental group on the variables of age, gender, race and educational level. Findings suggest that the experimental (TLE group) is impaired on all memory measures except learning. The experimental group also performs significantly poorly on all the measures of executive function. A correlation also exists between the performance by the experimental group on the measures of memory and executive function, suggesting that depressed executive function in these patients is related to depressed memory functions.

Introduction.

It is argued that our unique set of cognitive functions are the entities that separate us from all other animals. It is through these higher mental functions with which we experience our existence. It is extremely ironic that the most powerful force on the planet, if not the universe, lacks the ability to understand itself. However, with every passing year we know a little more about the brain, and maybe soon will truly understand brain function. Currently four disciplines, psychology, neurology, psychiatry and neurophysiology, are working both in isolation and together for the common goal of understanding the most complex phenomenon we probably will ever know (see, Andreasen, 1997).

Put very simply, this dissertation seeks to describe some of the cognitive deficits that individuals with temporal lobe epilepsy are prone to, such as memory deficits and executive function deficits.

The dissertation attempts to follow the American Psychological Association's Publication Manual guidelines; however it does not follow it religiously. There are six chapters.

Chapter 1 provides an over-view of the clinical syndrome of temporal lobe epilepsy (TLE), discusses the various underlying causes of epilepsy and its treatment.

Chapter 2 is the literature analysis that discusses previous research in temporal lobe epilepsy and the cognitive functions that this research has studied. Literature on anticonvulsant effects on cognition is also discussed.

Chapter 3 provides the rationale and aims. It was felt that these should only be fully discussed after the literature review.

Chapter 4 discusses the methodology adopted in the study, and describes the assessment tests that are used.

Chapter 5 contains the results of the tests, and a graphical approach to their presentation is used to facilitate easy understanding.

Chapter 6 contains the discussion and conclusion of the study.

The appendices house a copy of the subject information and consent form, the subject information sheet and a copy of the data collection sheet used during the assessment of the each sample member.

A note on the terminology used:

The term temporal lobe epilepsy (TLE) is slowly being replaced with the term complex partial seizures of temporal lobe origin. The names are currently used interchangeably in clinical practice and have been used interchangeable in this dissertation - as they denote the same clinical syndrome. Also, temporal lobe epilepsy with epileptogenic focus in the right hemisphere is termed R-TLE and similarly TLE in the left temporal lobe L-TLE, these are standard terms.

In section 2.2.3 neuropsychological tests are mentioned that are not part of the battery used in this research project. Rather than describing them in the text, it is less cumbersome to place the title of the main construct that they are believed to assess in the footnote form.

CHAPTER 1

1.1. Epilepsy.

Seizures are not uncommon events and a variety of seizure disorders affect about 0.5% of the general population (Hauser, Annegers & Rocca, 1996). A seizure occurs when there is an abnormal neuronal discharge in the brain that may spread to recruit neurones localised in many distance parts of the brain - this generally occurs during a tonic-clonic seizure (grand-mal seizure in old terminology). Not all seizures are this dramatic, many occur without anyone noticing, including the individual experiencing them - these, once referred to as petit-mal, are now termed absence seizures or complex partial seizures.

Seizures that occur in a specific region of the brain are termed focal, occasionally these focal seizures may spread, recruiting neurones throughout the brain and causing the commonly known motor convulsions that the general population terms an *epileptic attack*. The aetiologies of epilepsy vary from head trauma to chemical imbalances. However, more than often the aetiology is unknown - this is termed idiopathic epilepsy. The time interval between a seizure is known as the interictal period, and the period of seizure as the ictal period. The time interval of the interictal period is thus entirely dependent on seizure frequency. The interictal period varies from a few seconds if seizure frequency is very high to months and even longer with a very low seizure frequency. Other types of seizures exist, please refer to table 1 for a listing.

Electroencephalograph (EEG) studies of epilepsy show during the interictal period *spike-wave* activity. These are not seizures, but indicate that the individual has a higher probability than those people with out *spike-wave* activity of experiencing seizures. Furthermore, it is these spikes that some researchers attribute as the aetiology of cognitive deficits found in certain patients with temporal lobe epilepsy (REF NEEDED). One EEG recording session has only a 5% probability of observing *spike-wave* activity in epileptic patients. However, the more EEG sessions conducted the higher the probability of observing *spike-wave* activity (Murdoch, personal communication).

Table 1a

Types of various seizures.

(Modified from Walsh, 1994, p. 121)

MAIN GROUP	
Generalised	Tonic-Clonic, Absences, Akinetic, atonic, tonic, clonic, bilateral myoclonic, and others
Partial (Seizures with a focus)	Partial seizures with elementary symptomology, with motor symptoms, with sensory or somatosensory symptoms, with autonomic symptoms.
Partial seizures with complex symptomology	With impairment of consciousness only, With cognitive or affective or psycho-sensory or psycho-motor symptomology
Unilateral seizures	
Unclassified	

Table 1b.

International classification of the epilepsies

(Modified from Walsh, 1994, p. 121)

MAIN GROUP	CLINICAL NAME/SYNDROME
Generalised Epilepsy	Primary, Secondary, Undetermined
Partial (Focal or local)	Jacksonian epilepsy, Temporal Lobe epilepsy, Psychomotor seizures
Unclassified	

1.2. Underlying Neuronal Mechanism of Epilepsy.

The underlying neurophysiological mechanism behind epilepsy is poorly understood.

Two classes of cortical neurones exist; namely pyramidal and stellate neurones. Pyramidal a larger and far more numerous (Barr & Kiernan, 1993). Pyramidal cells are arranged vertically with the a dendrite extending towards the pia mater and the axon extending in a caudal direction (Wyllie, 1993). This axon may either extend to extracortical sites or synapses locally (REF NEEDED). Furthermore, pyramidal cells have axons that are excitatory, that is they depolarise cells on which they synapse. This arrangement assigns them a pivotal role in the production of both local and distant excitation, and thus pyramidal cells are deeply involved in epileptogenesis and its propagation (Wyllie, 1993, NEED REF).

It is believed that excitatory synaptic potentials play three roles in epileptogenesis. Firstly, they contribute to the generation of paroxysmal depolarisation shifts in individual neurones. Secondly,

they provide the mechanism by which synchronisation of the epileptiform discharges is achieved. Thirdly, they may be responsible for slow depolarisations and burst discharges in neurones (Wyllie, 1993).

The probability of a group of neurones exhibiting an epileptic discharge is dependent on three factors. Firstly, the properties of the cell membranes in the group. Secondly, the degree of excitatory coupling amongst the neurones. That is the number of excitatory synapses in the group. Thirdly, the integrity of inhibitory control mechanisms. When any of these are influenced, that is the cell membrane resting membrane potential is altered, there is an increase in the degree of excitatory coupling amongst the neurones and thirdly there is a loss of the inhibitory control amongst the neurones, the results may be an epileptogenic focus.

In summary, it is known that epilepsy is represented by an abnormal firing of neurones. This can be visualised on an EEGs during a seizure. Neurones transfer information within themselves with the use of action potentials (AP's), and to each other through synaptic chemical transfer, as well as gap junctions (electrical transfer) - the chemical synaptic method being the most common (Norton, 1996). Action potentials spread through the neurone and, through chemical synaptic transmission and gap junctions might augment or attenuate the formation of action potentials in neighbouring neurones. It is known that in epilepsy action potentials spread from the epileptogenic focus inducing 'chaotic' firing patterns throughout the brain. The production of an AP is entirely dependent on the neurone's cell membrane's resting membrane potential. Thus by affecting a change in the resting membrane potential an AP may be induced or inhibited. Many exogenous substances lower the resting membrane potential and others increase the resting membrane potential; these substances may induce seizure activity or inhibit it respectively. Many anticonvulsants owe their properties to their ability to increase membrane potential through a variety of different cellular mechanisms, thereby decreasing the probability that an action potential will develop.

Most physiologists propose that the mechanism underlying epilepsy can be demonstrated and studied by the experimental process termed *kindling* (Wasterlain, Farber & Fairchild, 1985; Wasterlain, Farber & Fairchild, 1986; McNamara, 1986; Adamec, 1990; Khurgel & Ivy, 1996).

1.2.1. Kindling.

The Kindling experimental model involves the application of an electrical sub-threshold stimulus to a neuronal membrane that does not produce a seizure; however, after repeated application of the stimulus a seizure may occur. It is through this process that the cellular changes that are believed to be the physiological mechanisms behind epilepsy may be studied (Trimble & Bolwig, 1992). It is believed that the repeated application of the same sub-threshold stimulus induces a number of cellular changes both to the pre-synaptic membrane, such as, augmented glutamate release (Glutamate is a neurotransmitter / modulator that generally functions on receptors that when stimulated lower membrane potential of the post-synaptic membrane - increasing the potential that an action potential will develop in the membrane), and the post-synaptic membrane.

Other cellular changes that are believed to occur are increased sensitivity of the NMDA receptor (these are the receptors that interact with glutamate) and enhanced voltage-gated Ca^{2+} conductance into the neurone. When Ca^{2+} enters the cell it moves the resting cell membrane potential in a positive direction, thereby increasing the probability of an action potential (Norton, 1996).

1. 3. Treatment of Epilepsy.

There are two current approaches for the treatment of the seizure disorders. Anticonvulsant medication and, if the epilepsy is focal, the epileptogenic focus known, and the epilepsy is intractable, surgical destruction of brain tissue where the focus is located.

Auxiliary treatment from the allied health disciplines may also be incorporated into treatment regimens. Psychological intervention in the form of support, behaviour modification and counselling may all minimise the effects epilepsy has upon the patient's functioning (Goldstein, 1990). Unfortunately the literature concerning the efficacy of these interventions does not favour favourable outcomes in these cases (Ibid.).

1.5. Temporal Lobe Epilepsy (TLE).

Temporal lobe epilepsy denotes a form of epilepsy that is focal with epileptogenesis in the temporal lobes. And as TLE has a determinable focus, temporal lobe epilepsy is also a focal epilepsy. Focal epileptogenesis is characterised by high-frequency synchronous discharges in a group of cortical neurones (Wyllie, 1993). The temporal lobe is more epileptogenic than any other lobe of the cerebrum, it is estimated that amongst the focal seizures about 50% originate in the temporal lobe (Niedermeyer, 1990). It also refers mainly to complex partial seizures and these may or may not generalise. About 50 % of patients with temporal lobe epilepsy have either unilateral or bilateral secondary generalised tonic-clonic seizures (Wyllie, 1993).

The term temporal lobe epilepsy is currently being replaced with complex partial seizures of temporal lobe origin and thus these two terms describe the same clinical pattern. As already mentioned, temporal lobe seizures do not always generalise and thus the patient is free of the typical motor symptomology of *grand-mal* seizures. It is estimated, however, that *grand-mal* seizures (generalised tonic-clonic) co-occur in a large number of temporal lobe epileptics. A typical temporal lobe epileptic may have between three and ten complex partial seizures a week but only 1 *grand-mal* seizures a month (Niedermeyer, 1990). Nevertheless, patients with complex partial seizures of temporal lobe origin have some very specific symptomology.

The inter-ictal behaviour of patients with TLE (as a group) tends towards emotional and attitudinal extremes, and as a group these individuals are prone to a variety of unpleasant personality characteristics (Lezak, 1995; See Bear, Levin, Brown & Steer, 1982; Waxman & Geschwind, 1975).

Apart from Axis II personality traits, which may often severely affect the patient's social functioning, the common ictal symptomology includes: Auras, unpleasant olfactory perceptions, *jamias vu, jamais extendu*, micropsia and macropsia (Walsh, 1994).

Depending on the where the focus is located in the temporal lobe the following ictal signs and symptoms generally occur. With a rhinecephalic focus the individual usually experiences strange, indescribable feelings, hallucinations and interpretative illusions. Seizures with their focus in the amygdala generally manifest rising epigastric discomfort, nausea, marked autonomic signs, borborygmi, belching, flushing of the face, followed then by staring and oral-alimentary

automatisms and confusion. Seizures that arise in the lateral posterior temporal lobe general manifest with auditory or visual hallucinations, language dysfunction followed by dysphasia, confusion and occasionally starting with automatisms. Insular seizures generally manifest during the ictal period with vestibular or auditory hallucinations, borborygmi, belching and autonomic signs, unilateral facial twitching, paresthesiae, olfactory and gustatory hallucinations (Niedermeyer, 1990). The inter-ictal period is usually devoid of any of these signs and symptoms; however, various aspects of their cognitive functioning during the inter-ictal period is affected. As this aspect of temporal lobe epilepsy forms the main focus of this dissertation, it will be further discussed in chapter 2.

Thus, this disorder that to the general population may seem not as serious as seizure disorders of the generalised tonic-clonic type can wield untold effects on the sufferer's social, psychological and cognitive functioning. This disorder can have negative effects on an individual's schooling, thereby relegating them to more menial types of employment and preventing them from fulfilling their academic or educational potential. The unpleasant personality characteristics and the isolation that may follow from their pathological behaviour may serve further to isolate the TLE patient, which may account for the increased suicide and divorce rates (Bladin, 1992; Barraclough, 1987).

CHAPTER 2

2.1. Cognitive Functioning in T.L.E..

There still exists doubt that individuals diagnosed with complex partial seizures of temporal lobe origin report deficits in cognitive functioning. Although many studies have demonstrated memory deficits, personality abnormalities, difficulties with certain aspects of executive function and other aspects of higher function, a significant number have also failed to demonstrate the existence of memory and other cognitive deficits in patients with temporal lobe epilepsy.

A significant limitation regarding the research on TLE is that a large amount of the data has been generalised from research conducted on patients who have undergone a temporal lobe resection for the treatment of intractable complex partial seizures. Furthermore, the commonly held notion of a differential performance between L-TLE and R-TLE samples on specific cognitive functions is not universally agreed upon in the literature. Nevertheless, there is a strong belief, amongst both clinicians and researchers, that L-TLE patients report more deficits on measures of verbal memory than R-TLE patients.

Memory has been the main focus of most studies concerned with temporal lobe epilepsy, obviously because the temporal lobe are believed to mediate memory (Squire, 1986; Zola-Morgan & Squire, 1990).

Views on anticonvulsant mediation of the cognitive deficits reported has changed from the universal belief that they severely interfere with cognition to recently reported research that claims that they have minimal, if any, effects.

2.2. Literature Analysis.

2.2.1. Memory and Related Constructs.

The domain of memory represents an important aspect of cognitive function (Grant & Adams, 1986; Hermann, Seidenberg, Haltiner & Wyler, 1992; Lezak, 1995; Walsh, 1994). The cognitive function of memory involves a number of complex systems through which an individual registers, stores, retains and then retrieves previous experiences (Lezak, 1995).

The cognitive concept of memory may further be divided into a number of commonly accepted sub-groups - although there is not universal agreement concerning all of these sub-groups. The groups are:

- (1) Short Term Memory (Working Memory)
- (2) Long-Term Memory
- (3) Visual Memory
- (4) Verbal Memory
- (5) Explicit Memory (Requires conscious effort to retrieve these memory)
- (6) Implicit Memory (Memories are retrieved without conscious effort - Usually motor memories)

The neurophysiology of memory is poorly understood - the most current prevalent theory is Long Term Potentiation (Bergado, Krug, Ruthrich & Matthies, 1988; Buzsaki, 1989; Goddard & Douglas, 1975; Teyler & Discenna, 1984). It is currently assumed that the more medial structures of the temporal lobe play a vital role in the consolidation of information into long-term store (Squire, 1986; Zola-Morgan & Squire, 1990). Thus, from a physiological point of view, damage to the medial structures of the temporal lobes should only result in long-term memory deficits.

The past and current literature reports that a variety of memory deficits exist in patients with temporal lobe epilepsy - especially with regards to long term memory (Lavadas, Umilta & Provincial, 1979). The majority of studies have sampled from a pool of patients who have undergone a temporal lobe resection. However, the memory deficits that are associated with

complex partial seizures of a temporal lobe origin in non-surgical cases is far less clear (Mungas, Ehlers, Walton & McCutchen, 1985).

Memory deficits have been reported for over a hundred years in individuals with seizure disorder (Grant & Adams, 1986). But, a number of studies have failed to find any significant differential performance between patients with temporal lobe epilepsy and other seizure disorders as well as normal controls (Mayeux, Brandt, Rosen & Benson, 1980; Mignone, Donnelly & Sadowksy, 1970; Mirksy, Primac, Maran, Rosvold & Stevens, 1960; Rodin, Katz & Lennox, 1976; Scott, Moffett, Matthews & Ettlinger, 1967; Stevens, Milstein & Goldstein 1972). Other studies report memory deficits that do not differ according to laterality of seizure focus (Dennerll, 1964; Glowinski, 1973; Hermann, Wyler, Richey & Rea, 1987; Schwartz & Dennerll, 1969). Whilst a few studies report lateralised memory deficits - usually with poor performance on verbal memory measures in left-temporal lobe patients and non-verbal memory deficits associated with R-TLE patients (Grant & Adams, 1986).

A study conducted by Glowinski as far back as 1973 represents one of the first systematic studies concerning memory functions in TLE patients not undergoing a temporal lobectomy. The study, conducted on patients with unilateral temporal lobe epilepsy, reported that these patients suffered from a marked short term memory deficit, that was displayed on the Weschler Memory Scale, when compared with a comparable group of centrencephalic epileptics. This study further reports that the TLE group had particular difficulty in integrating and memorising meaningful verbal information. Furthermore, these group differences are independent of I.Q., age, recall of digits, education, age of onset, and medication. Interestingly, there are no significant findings with respect to the laterality of the temporal lobe lesion.

Some researchers argue that the reported long term memory deficits in patients with seizure disorder of a temporal lobe origin may not exist and the data that report these cognitive deficits has arisen due to a communication disorder in TLE patients. Hermann, *et al.* (1987) argue that almost no studies control for other higher function disorders that may cloud the results obtained. This, especially in research, may be undetected when research batteries are limited to only the construct that is to be investigated. Furthermore, long-term memory deficits may appear to exist due to a deficit in other memory constructs. For example, the inability to encode information (learning deficits). Also, cognitive dysfunctions or disorders, such as the communication disorder may appear, especially during a neuropsychological assessment, as a memory deficit, while the

patient may in reality experience normal memory abilities during daily functioning. According to Hermann, *et al.* (1987), amongst researchers and clinicians it is implicitly understood that critical supportive cognitive systems must be intact if one, *especially in research*, wishes to draw inferences concerning the integrity of an individual's memory (Italics my own). The authors list deficits such as stimulus reception and response output systems that could distort neuropsychological test data concerning memory integrity.

Similarly, a recent study by Grippo, Pelosi, Mehta & Blumhardt (1996), found significant differences between controls and temporal lobe epileptic subjects on both the digit span forwards and backwards. The authors concluded that although memory deficit in TLE is generally considered a long-term memory deficit, short-term memory processing problems contribute to the overall deficit. Unfortunately, there was no control for medication and lateralisation of epileptogenic focus was only known in 48% of the experimental group. Controls were matched on age, gender and years of education.

The research regarding the neuropsychological effects of unilateral temporal lobe resection due to intractable seizures has demonstrated a clear relationship between right temporal lobe resection and impairment of non-verbal memory and a one between a left temporal resection and verbal memory (Meyer & Yates, 1955; Milner, 1970, 1975; Wiengartner, 1968).

Studies concerning verbal learning and verbal memory abilities conducted on TLE patients report that these patients perform poorly compared with controls on measures of these cognitive constructs. In a study conducted by Hermann, *et al.* (1987) the sample with left-temporal lobe seizures scored significantly worse than both right-temporal lobe seizure subjects and matched controls. Interestingly, no significant differences are reported between the right hemisphere group and the controls on the measure of verbal learning. It is noteworthy that this study reports no significant differences amongst all three groups on the measure of recognition memory. Significant differences with regards to retrieval efficiency are reported between all groups. The left-hemisphere performing the most poorly.

It appears from the literature discussed in the previous paragraph that patients with left-temporal lobe epilepsy perform poorly on measures of verbal memory, whilst right-temporal lobe epileptic patients perform poorly on measures of non-verbal, perceptual memory (Delaney, Rosen, Mattson & Novelly, 1980; Hermann, *et al.*, 1987; Meyer & Yates, 1955). The literature is equivocal

with regards to the effects of laterality of the seizure disorder with regards to memory deficits. Whilst most of the literature reviewed thus far in this section suggests that left hemisphere epileptics perform poorly on measures of verbal and semantic memory, recent studies have refuted this notion. For example, Saykin, Gur, Sussman, O'Connor & Gur, (1989) report that laterality has no significant effect on deficit performance in semantic and figural memory, with age of onset and the length of time with active seizures a more powerful predictor of memory deficit severity.

The literature regarding complex partial seizures in foci other than in the temporal lobes too is contradictory. For example, there is lack of agreement about the performance of patients with complex partial seizures of frontal origin. Some authors hold that these individuals present with no memory deficits (Milner & Teuber, 1968). Whilst other authors report that these patients experience difficulty with tasks of delayed paired comparisons and tactile continuous-recognition memory (Prisko, 1963).

In conclusion, although there is a mountain of research concerning memory and temporal lobe epilepsy, there is no uniformity with regards to results. Although, the majority of studies have found memory disturbance in patients with complex partial seizures of temporal lobe origin, causative and affecting variables are still debated. Furthermore, there is no clear indication in the literature as to which aspects of memory are impaired in these individuals. Therefore, studies that investigate memory performance in patients with complex partial seizures of temporal lobe origin are still warranted.

2.2.2. Executive Function.

There is a paucity of research on the construct of executive function, with majority of this limited research focusing on perseveration and shift-of-set only.

Executive function consists of those higher mental functions that allow an individual to engage successfully in behaviour that is independent, purposive and self-serving (Lezak, 1995).

Executive function has traditionally believed to be mediated by the frontal lobes (Walsh, 1994). One may regard the construct as consisting of the following *sub-constructs*: Abstract thought, concept formation, shift-of-set and reasoning (Schofield & Nielsen, 1998).

Two current neurophysiology theories serve to explain why an abnormal discharge in the temporal lobes would cause a functional abnormality in the frontal cortex. There are two possibilities why temporal lobe abnormality affects executive function, if it does affect it all. The first possibility is that there is an abnormal neuronal discharge during the interictal period that reaches the frontal cortex. The second, certain executive functions are mediated by the temporal cortex. This precisely what the two hypotheses propose (Hermann & Seidenberg, 1995).

The *Noctiferous Cortex Hypothesis* postulates that the epileptogenic cortex (temporal lobes) adversely affects the frontal lobes or those extra-temporal regions that are believed to mediate executive function. In other words, an abnormal discharge from the temporal lobes reaches the frontal lobes thereby affecting the cognitive functions that are mediated by the frontal cortex.

The *Hippocampal Hypothesis* postulates that the human hippocampi are directly responsible for mediating those executive functions that are depressed in individuals with complex partial seizures of temporal lobe origin. In other words, those executive functions affected in individuals with temporal lobe epilepsy are mediated by temporal structures.

Reports first surfaced in the 1940's noting improved executive function after the resection of the epileptogenic lesion (Hebb & Penfield, 1940). Worked conducted mainly by Hermann at the Department of Neurology, University of the Wisconsin in the 1980's identified that patients diagnosed with temporal lobe epilepsy displayed increased perseveration responses, a decrease in the number of categories satisfied on the Wisconsin Card Sorting Test and poor performance on

measures of tracking (Hermann, Wyler & Richey, 1988). This study has been replicated (see, Corcoran & Upton, 1993; Hermann, Seidenberg, Haltiner & Wyler, 1991; Strauss, Hunter & Wada, 1993; Trenerry & Jack, 1994).

Most studies have utilised the Wisconsin Card Sorting Test when investigating temporal lobe seizures and executive function. Thus there is a paucity of literature concerning the other constructs of executive function.

From an anatomical view point, one would expect that if higher functions such as shift-of-set are affected in patients with temporal lobe epilepsy, then one would expect other higher functions that are believed to be mediated by the frontal cortex to be affected. Therefore, one should expect that patients with complex partial seizures of temporal lobe origin to have depressed executive function.

If the *Nociferous Cortex Hypothesis*, which postulates that an abnormal discharge in the temporal lobes has frontal lobe ramifications, is correct and as the evidence lends itself to this hypothesis rather than the *Hippocampal Hypothesis* (see, Hermann & Seidenberg, 1995), then there is a significant amount of research that needs to be undertaken in this area. Also, as executive function may arguably be one of the most important higher function constructs for adequate daily functioning of the patient (Lezak, 1995), then should this be impaired then in patients with complex partial seizures of temporal lobe origin, then an extremely important area of clinical neuropsychological research has been neglected.

2.2.3. Anticonvulsants and Cognitive Function.

Drug trials conducted on normal volunteers have reported that these individuals experience measurable cognitive deterioration on phenytoin, carbamazepine and other anticonvulsants (Thompson & Trimble, 1981). However, recent studies have refuted this finding. (See, Prevey, Delaney, Cramer, Cattanach, Collins & Mattson, 1996).

The literature is contradictory regarding the effects of the various anticonvulsants on cognitive performance. The following anticonvulsants will be reviewed: Phenytoin, Carbamazepine and Sodium Valproate, because these three are the main first line treatments for patients with epilepsy, the majority of the literature concerning epilepsy, cognition and medication discusses these anticonvulsants, and most of the sample in this dissertation is comprised mainly of subjects on monotherapy, with an almost equal split between Carbamazepine and Sodium Valproate, the remainder on a variety modern anticonvulsant and polytherapy (See Chapter 5, Section 1). When studying cognition and anticonvulsants the research generally focuses on the variables of anticonvulsant type and serum plasma levels.

Most clinicians seem to view that carbamazepine has the most significant cognitive side-effects (M.Lucas, personal communication).

Unfortunately, there very few studies that have researched anticonvulsant effects on cognition in a systematic manner (Thompson & Trimble, 1982; Trimble & Reynolds, 1976). Thompson & Trimble (1982) report that patients on carbamazepine perform better on cognitive measures of memory, concentration, mental speed and motor speed.

In 1986, Andrews, Bullen, Tomlinson, Elwes and Reynolds compared the cognitive performances on tasks of short-term memory scanning, word list learning, memory for prose, decision making and tracking tasks. Three groups were researched: Untreated Group of Temporal Lobe Epileptics, Monotherapy Carbamazepine and Monotherapy Phenytoin. Significant differences between the groups on the tests of memory are reported in favour of carbamazepine. However, no significant differences are reported on the measure of reaction time. Importantly, no significant differences are reported between the groups on decision making tasks and tracking tasks - *executive function*.

In 1987 O'Dougherty, Wright, Cox and Walson reported that carbamazepine plasma levels, at low concentration levels, produced a mild beneficial effect on speeded eye-hand co-ordination and more rapid processing of items in memory. Efficiency of learning new information displayed an inverse concentration-dependent relationship with carbamazepine serum plasma level, i.e. the higher the concentration level the poorer the performance on sustained attention, short-term memory scanning, the ¹Paired Associates Test and the ²Purdue Pegboard Test.

Duncan, Shorvon and Trimble (1990) report that on measures of mental speed, attention, performance of a learned skill, short-term memory, concentration and simple co-ordinated hand movements there is a significant improvement only on simple motor skills with the discontinuation of phenytoin, carbamazepine and sodium valproate. No significant differences are reported between the three anticonvulsants on the test of motor skill. No significant differences on the other cognitive measures are reported between the results obtained whilst the participants are on valproate acid and carbamazepine and the results of the tests after the discontinuation of these anticonvulsants. However, those on phenytoin score significantly better after discontinuation. These authors conclude that although all three anticonvulsants have deleterious effects on simple motor function, only phenytoin has negative effects on higher cognitive function. However, other studies have refuted the findings regarding phenytoin. Meador, Loring, Huh, Gallagher and King (1990) report no differential performance between patients diagnosed with temporal lobe complex partial seizures on carbamazepine, phenobarbital and phenytoin on the following tests: Digit span, Selective Reminding Test, ³Digit Symbol, ⁴Finger Tapping, ⁴Grooved Pegboard and a test of choice reaction time.

Gallassi, Morreale, Sarro, Marra, Lugaesi and Baruzzi (1992) report that epileptic patients administered carbamazepine do not perform significantly differently on measures of vigilance, attention, memory and visuo-motor control, whilst on carbamazepine and after discontinuation of the drug. However, these authors report that patients on sodium valproate score significantly poorer on tasks of attention, visuo-motor performance, verbal span and sensory discrimination, whilst on sodium valproate compared with the scores obtained after anticonvulsant discontinuance.

¹ VERBAL MEMORY
² MANUAL DEXTERITY
³ ORIENTATION AND ATTENTION
⁴ MOTOR FUNCTION

Pullianen and Jakelainen (1995) report that the long term cognitive side-effects of carbamazepine seem restricted to mainly some visually guided motor functions. Prevey, Delaney, Cramer, Cattanach, Collins and Mattson (1996) report that the impact of carbamazepine and sodium valproate monotherapy on cognitive functioning is similar and both drugs appear to have minimal (if any) negative side effects. Both drug groups in their study did not score significantly differently from the baseline performance.

Thus, although it is still strongly debated, the more recent literature seems to indicate that the anticonvulsant medications carbamazepine and valproate acid do not negatively affect cognition to any significant extent.

2.3. Gap in the Literature.

The majority of the neuropsychological research concerning cognitive functions in patients with complex partial seizures of temporal lobe origin has focused on memory, probably because the temporal lobes are believed to mediate many memory functions (REFS). However, many of the other higher functions are neglected. Although there is research detailing executive dysfunction in TLE patients, it has focused mainly on shift-of-set and perseveration, and appears to be more based in the historical localisational framework of neuropsychology, rather than the more modern clinical approach that seeks to describe and understand the functional abilities of the patient. Also, other aspects of executive function have been neglected.

CHAPTER 3

3.1. Rationale and Aims.

Cognitive deficits, no matter how moderate, can directly interfere with an individual's daily activities. Neuropsychological research on complex partial seizures of temporal lobe origin has neglected to fully investigate executive function. This is alarming as people with executive function deficits are often severely impaired in daily functioning, whilst often those with severe cognitive deficits other than executive impairment are able to function acceptably (Lezak, 1995).

Executive function is arguably the most important cognitive construct that humans poses. With it we are able to plan, reason and act in manner that ensures our well-being. One may hypothesise that as the temporal lobes are believed to mediate memory function research into executive function that is believed to be mediated by the frontal lobes has been neglected. However, the research has shown that patients with complex partial seizures of a temporal lobe origin display deficits in shift-of-set and perseveration. Thus if these two constructs are affected in patients with TLE, it is logical to assume that other believed frontal mediated constructs would be affected to too. Thus research into the various different aspects of executive function is warranted.

Although there is a mountain of research concerning memory and TLE, there is little consensus within the literature. Further research designed to clarify the confusing literature regarding memory deficits associated with unilateral TLE might lead to a better understanding of the psychological functions that the temporal lobes mediate, and provide fresh insights into the neuropsychological organisation of memory. In clinical psychology, it might serve to clarify the clinical picture and lead to the development of more sensitive neuropsychological instruments, and therefore also have broader applicability to the assessment of memory in general. If TLE proves to be an objectively describable syndrome that results in subtle memory deficits, it provides the opportunity for the development of even more sensitive memory tests, which may be used to assess subtle memory deficits resulting from a variety of aetiologies (Mungas, *et al.*, 1985).

Furthermore, memory has been investigated in this study as no research has been conducted that details any relationship between memory impairment and executive function in patients with complex partial seizures of temporal lobe origin.

The study is aimed at constructing a limited cognitive pattern for individuals with temporal lobe epilepsy, and asks the following research questions:

- 1) Do patients with complex partial seizures exhibit memory impairment?
- 2) Is there a correlation between performance on memory measures and measures of executive function?
- 3) The literature reports deficits in shift-of-set and perseveration, are other aspects of executive function affected in these patients?

This study hypothesises that although the research regarding memory function in patients with complex partial seizures is contradictory it does point to a memory disturbance in these individuals, thus we would expect that they will perform more poorly on the measures of memory. Secondly, as research has demonstrated that these individuals perform more poorly on measures of shift-of-set and display an increased perseveration response that they will perform more poorly on other measures aspects of executive functions. Thirdly, as previous research indicates that there is an abnormal discharge from the temporal to frontal lobes (see, Hermann *et al.*, 1995), one would expect that cognitive sequel of this to be a correlation between performance on memory measures and executive functions.

Thus the hypotheses are: The individuals with temporal lobe epilepsy will score significantly worse on the measures of memory than the controls. The T.L.E. group will also score more poorly on the measures frontal function, and there will be a correlation between the scores on the memory measures and the scores on the executive/frontal measures, suggesting that the temporal lobes influence the frontal lobes.

CHAPTER 4

METHODOLOGY, INSTRUMENTS AND PROCEDURES

4.1. Methodology.

4.1.1. Research Design.

The study consists of two groups. Firstly, an experimental group, consisting of participants who have received a diagnosis of Complex Partial Seizures of Temporal Lobe Origin or, in old terminology, temporal lobe epilepsy (TLE). Secondly, a control group of matched subjects. Each participant is administered a battery of neuropsychological and psychological tests. The test scores of each group are compared. The design is quasi-experimental as randomisation is not possible.

4.1.2. Inclusion Criteria for the Experimental Group.

- 1) Received a diagnosis of temporal lobe epilepsy.
- 2) No severe Axis I disorders.
- 3) No severe Axis III disorders (besides temporal lobe epilepsy)
- 4) No history of Axis I disorders.
- 5) No history of Axis III disorders.
- 6) Fluent in English.
- 7) Age between 18 and 55.
- 8) Not Alcoholic.
- 9) Seizures are generally controlled.
- 10) Not abusing narcotics.

4.1.3. Inclusion Criteria for the Control Group.

- 1) Never received a diagnosis of epilepsy
- 2) Never had a seizure.
- 3) Not an alcoholic.
- 4) No a substance abuser.
- 5) Fluent in English
- 6) Age between 18 and 55
- 7) Never received a significant Axis I disorder diagnosis.
- 8) No Axis III diagnosis.
- 9) Are matched to one of the participants in the Experimental Group with regards to age, gender, number of years of education and race.
- 10) Never had a head injury.

4.2. Subjects.

The total sample size is 50. N=25 in the experimental group and N=25 for the control Group. The TLE sample consists of 18 female and 7 male participants. The participants were sampled from a neurology outpatient's clinic at Johannesburg hospital and the majority of the sample was obtained from patients attending the Department of Psychiatry's Community Psychiatry Clinics. 22 participants in the experimental group were medicated with anticonvulsant medication, the remaining three were on anti-depressants, although they had the diagnosis of TLE. 1 participant in the experimental group was left handed and none of the control group. None of the participants were depressed, according to the Beck Depression Inventory, at the time of the assessment. The mean period of medication in this group was 4.7 years. This figure is independent of the medication type. All participants in the experimental group (except one participant) had been on the same medication for of or longer than three months. Furthermore, this group's individual social differences were minimal. They came from the same ethnic group, same social background and a similar economic division of society.

The control group (obtained from normal volunteers) was matched to the experimental group on age, gender, years of education and race. The control group was obtained from the general

caucasian population and none had any history of any severe Axis III disorders or any history of psychiatric illness. None of the participants in this group were on any form of medication. (See section 4.1.3 for exclusion criteria).

4.3. Instruments.

The following assessment measures are used:

- 1) Rey Complex Figure - Copy.
- 2) Rey Complex Figure - Immediate Recall.
- 3) Rey Complex Figure - Delayed Recall.
- 4) Rey-Auditory-Verbal-Learning Test (R.A.V.L.T).
- 5) Wisconsin Card Sorting Test - 64 Modified.
- 6) Austin Maze.
- 7) Digit Span Forwards.
- 8) Digit Span Backwards.
- 9) Similarities (SA-WAIS).
- 10) Tower of London.
- 11) Beck Depression Inventory (B.D.I).

* Interestingly, this individuals scores were the highest of the experimental group. Additionally, the individual's TLE was uncontrolled.

4.3.1. Wisconsin Card Sorting Test (Grant & Berg, 1948)

The Wisconsin Card Sorting Test (WCST) primarily assesses patient concept formation and reasoning. Lezak (1995) defines it as a test of “*abstract behaviour*” and “*shift of set*”. The WCST was first used as a measure of frontal lobe dysfunction (Milner, 1963). However, recent studies have debated the usefulness of the WCST as a predictor and indicator of frontal lobe dysfunction (Mountain & Snow, 1993). Furthermore, there, according to the previous authors, appears to insubstantial evidence that the WCST is useful for both clinical and research purposes (Ibid.). Nevertheless, the WCST is still a commonly accepted measure of shift-of set and perseveration, albeit unable to occasionally illustrate damage to the dorsolateral-anterior part of the frontal lobes. The construct validity of the WCST has been confirmed (O’Donnel, MacGregor, Dabrowski, Oestreicher & Romero, 1994). The WCST also appears to have good interrater reliability amongst both novice and experienced testers (Axelrod, Goldman & Woodard, 1992).

Two types of errors occur. Normal errors and perseverative errors. Perseverative errors occur when a patient continues to sort according to a previous successful strategy or in the case when no categories are satisfied, an initial erroneous guess. The other error occurs when the individual loses track of the sorting principle or matches by simple guessing (Lezak, 1995). The modified version of the test is used in this study. This approach uses only one of the pair of the 64 pack cards. The participant is required to place each card in the pack beneath one of the four stimulus cards. The examiner starts with colour as the first category, after 10 cards have been correctly place, the category is shifted to form then to number, and then back to colour.

Theoretically, a maximum of 6 categories can be satisfied. There is a concern that using the modified (1 pack) version of the WCST may yield a less accurate result. However, the literature reports that the single pack version yields similar results to the full version of the WCST (Axelrod, Henry & Woodard, 1992). Furthermore, there are three different versions of the modified-WCST. The one utilised in this study (WCST-64) is reported to yield comparable results to the full WCST than the other modified versions, such as the WCST-3.

4.3.2. Rey Complex Figure (Osterrieth, 1944)

The Rey Complex Figure (RCF) is arguably the most widely used assessment measure in neuropsychology. The RCF was designed to investigate perceptual organisation, visual memory

and constructional abilities in brain damaged individuals (Boone, Lesser, Hill-Guiterres, Berman & D'Elia, 1993). The test consists (in its full version) of three trials. The RCF-Copy, which requires the patient to copy the drawing, The RCF-immediate recall, which requires that the patient redraw the figure from memory shortly after having copied the figure. The RCF-delayed recall, which requires that the patient redraw the figure after between 30 to 60 minutes have elapsed since RCF-Copy trial. Research has shown that the RCF has good inter-rater reliability and construct validity (Carr & Lincoln, 1988). There are various scoring systems in use. The scoring system used in this study is illustrated in figure 2.

The participant is given a blank piece of paper and four coloured pens. The RCF is then placed in front of them. They are told to draw the figure as accurately as possible using the different coloured pens. The examiner tells them when to change the pen. The different colours are used to assess the manner in which they approached the drawing at the time of marking the RCF. In the second a third trial the participant must redraw the figure from memory. (See the following page for a description of the scoring system used)

4.3.3. Digit Span (Weschler, 1982)

The digit span (part of the Weschler) is used to assess attention (Lezak, 1995), and is a useful indicator of working memory. The digit span's construct validity as an attentional measure is high (Bornstein, 1983; Larrabee & Curtiss, 1995). The digit span can be separated into digit forwards and digits backwards and then scored separately, as is done in this study, or the two scores can be added together yielding one score. The reason for this separation on the scores is that the digit backwards tests yields information pertaining to mental tracking, which involves more than just attentional abilities and working memory, but also incorporates more advanced mental operations (Lezak, 1995).

4.3.4. Austin Maze (Walsh, 1994)

The Austin Maze is designed to test error utilisation (Walsh, 1994) and spatial or topographical memory and an over-all indicator of frontal lobe function (Bowden, Demedzic, Clifford & Hopper, 1992; Morrison & Gates, 1988). According to Walsh (1994), whenever there is frontal lobe involvement one would expect the termination of a lower error performance in subsequent trials. There is also a motor component to the Austin Maze. Thus, one would expect those with retarded motor ability to score significantly worse on the test. The higher the test score the worse the performance.

4.3.5. Similarities Test. (Weschler, 1982)

The Similarities test (part of the Weschler Adult Intelligence Batter) is primarily a test of verbal concept formation and reasoning (Bornstein, 1982). The test consists of twelve pairs of words that are conceptually related. The item difficulty ranges from the simplest concrete conceptual relationship to extremely difficult abstract relationships. Studies have found that individuals with left frontal and bilateral lesions score poorly on the test (See, Bogen, *et al.*, 1972; Mcfie, 1975).

4.3.6. The Rey-Auditory-Verbal-Learning Test (R.A.V.L.T). (Rey, 1964)

The R.A.V.L.T measures immediate memory span, provides a learning curve and reveals the presence or absence of learning strategies and provides insights into the patient's rate of learning, forgetting and encoding strategies (Lezak, 1995; Mitrushina, Satz, Chervinsky & D'Elia, 1991). The test variation used in this study consists of 8 trials. The first 5 trials entail reading a list of 15 words to the patient and asking them to repeat the words in any order directly after the examiner is finished. The sixth trial entails reading a new list of 15 words to the patient and asking them to repeat the new list, in any order. The seventh trial entails reading the first list of words to the patient and then again having them repeat as many as they can remember. The trials after the seventh trial may be modified in response to what the examiner wishes to assess. In this study the eighth trial entailed placing a page that had 30 words printed on it - all the words from the first list (in a random order), some of the word from the second list and a few words not contained in either list. The patient is then asked to circle the words that they remember from the first list only. The final trial entails asking the patient after a period of approximately thirty minutes has elapsed to repeat the words they can remember from the first list.

4.3.7. The Tower of London Test. (Shallice, 1982)

The Tower of London assesses primarily planning abilities, it also has a spatial working memory component (Elliott, Baker, Rogers, O'Leary, Paykel, Frith, Dolan & Sahakian, 1997; Lezak, 1995). A number of studies have showed that the Tower of London is a measure of frontal lobe function (Owen, Doyon, Petrides & Evans, 1996; Owen, Downes, Sahakian, Polkey & Robb, 1990). Unfortunately, the validity of the measure as a test of planning still needs to be shown (Kafer & Hunter, 1997).

The patient's task is to arrange three coloured rings on a board with three sticks of differing height to a predetermined position. The patient is asked to perform the operation in as few moves as possible. The levels of difficulty of each predetermined position depends on the number and complexity required to achieve the desired arrangement (Lezak, 1995).

The number of moves made by the patient is noted on each item (predetermined position) and then tallied to calculate the overall test score. Therefore, the higher the score the poorer the performance on the test.

4.3.8. The Beck Depression Inventory (B.D.I). (Beck, Ward & Mendelson, 1961)

As research has shown that mood disorders might negatively influence a wide range of higher functions (Lachner & Engel, 1994; Tancer, Brown, Evans & Ekstrom, 1990; Bulbena & Berris, 1993; Sternberg & Jorvik, 1976; Coello, Ardila & Rosselli, 1990), the B.D.I was employed to ensure that the sample was free from an affective disorder.

The instrument consists of 21 multiple choice. It is a widely accepted clinical instrument (Piotrowski & Lubin, 1990). The B.D.I's reliability is good on younger individuals and has high internal reliability (co-efficient alpha estimate of internal reliability = .79). The B.D.I scores correlate well with other instruments that assess mood states, such as the *Global Clinical Rating Scale* (Strober, Green & Carlson, 1981; Schaefer, Brown, Watson, Plemel, DeMotts, Howard, Petrik & Balleweg, 1985) Constructed validity has been found and replicated (Beck, Epstein, Brown & Steer, 1988).

4.4. Procedure.

Permission to conduct the study was obtained from the Medical Ethics Committee of the University of the Witwatersrand.

The subjects were asked to read and sign the subject information and consent form. Please refer to appendix 1 for a copy of the information and consent form. The assessment only commenced once the subject had read and signed the consent form.

The assessment session commenced with the assessor explaining to the participant what was going to happen during the assessment session and why the study was being conducted. The subject was given the consent form and asked to read through and sign it at the bottom if they agreed to participate. The participants were reminded that they were free to withdraw consent at any stage of the assessment - only one participant requested that the assessment end.

A blank piece of white paper was placed in front of the participant and then four coloured pens placed next to the sheet of paper. The RCF was then placed above the blank sheet. The following instructions were given:

“ I would like you to copy the figure inside this large rectangle. *The researcher indicated the rectangle with his finger.* Here are four coloured pens, begin drawing with the pen closest to you. I will tell you when to change pens. You can take as long as you want, and try and be as accurate as possible. You may not move the papers.”

When the participant indicated that they were finished, the RCF and the participant's copy was removed from sight. The participant was then asked to complete the subject questionnaire (See appendix 2).

A blank piece of paper was then placed in front of the participant once again. The following instructions were given:

“ Remember the strange drawing I asked you to copy earlier? I would like you to redraw it now from memory. You may take as long as you like.”

The WCST-64 was then administered. The instructions were as follows:

“ This test is unusual, because I cannot tell you exactly how to do it. What I can tell you is that these are four stimulus cards. *Pointing at them.* In this pack are cards with different shapes on them. Your task is to match them to one these four cards. I cannot tell you how to match them, but will tell you if your match is correct or incorrect. Are you ready?”

The Austin Maze was then administered. The instructions were as follows:

“This is the Austin Maze. Your task is to get from the start to the finish. *The start and finish were touched by the examiner.* When you press a button and get a green light it means you have gone the correct way. You can move back and forth and left and right, but not diagonally. *This was all indicated by the examiner with his finger.* You will do this test five times. The first time you must try and find the correct rout. The second, third, fourth and fifth time try and get as few red lights as you can. The route does not change.”
The number of errors was noted after each trial.

The Digit Span Forward was then administered, with the following instructions:

“Now I am going to say some numbers. You must repeat them after me. For example, if I say 7-1-6. You say... That’s correct. Are you ready?”

The Digit Span Backwards was then administered.

The instructions were as follows:

“Now I am going to say some more numbers. This time you must say them backwards. So, if I say 7-1-6 you say... That’s correct. Are you ready?”

The Similarities (SA-WAIS) was then administered.

The instructions were as follows:

“ Now I am going to read two words. I would like you to tell me how they are similar (alike).”

The Tower of London was then administered.

The instructions were as follows:

“ I would like you to make the same pattern on each of the cards, in as few moves as possible.”

The final assessment measure was the RCF-Delayed Recall.

“Remember the odd drawing I have asked you to draw twice. I would like you to draw it again from remember. Once you start you will start to remember it.”

After the participant had indicated that they had completed the RCF- Delayed Recall they were then asked to complete the Beck Depression Inventory.

4.5. Statistical Analysis.

Chapter 5 contains the summary of the statistical data. Each test is graded and the mean performance is calculated. As the study consists of two groups two common statistical techniques for comparing the significance of the two means are available: Student's t Distribution Test and Analysis of Variance (ANOVA). As ANOVA is argued to be the more powerful statistical test of the two, it is used for the analysis of the data.

ANOVA assumes the following (McCall, 1990):

- 1) The groups involved in the analysis are composed of randomly and independently sampled subjects.
- 2) The groups' scores being analysed are independent.
- 3) There is homogeneity of within-group variances.
- 4) The sample is drawn from a population of scores that is normal in form.

Correlations between performance on the different test measures in the experimental group are conducted using Pearson's Correlations.

CHAPTER 5

RESULTS

5.1. Group Equivalence Variables.

Table 1

Demographic Data for the Experimental and Control Groups.

VARIABLE	EXPERIMENTAL GROUP	CONTROL GROUP	P-LEVEL
Age	37.4 (17.3)	39.2 (15.2)	0.7312
# of School Years	10.2 (2.1)	11.3 (1.2)	0.5221
# of tertiary years	1.1 (1.2)	1.2 (2.3)	0.7655

Note: Values are mean (SD).

From table 1 one can note that the groups are equivalent on the matching criteria age and education. No statistical analysis is conducted on the two matching criteria as they are matched exactly.

Anticonvulsants effects were a concern when this study was conceived. Serum levels were obtained from as many participants as possible. However, these were often outdated or blatantly incorrect. For example, in once case Tegretol (carbamazepine) serum levels were ordered, but the laboratory conducted the a test for phenytoin. Thus, this study relies on the current research that suggests that anticonvulsants have little influence on cognitive performance.

5.2. THE REY COMPLEX FIGURE

5.2.1. The Rey Complex Figure-Copy.

FIGURE 2

Comparison of the RCF-COPY score means for the Experimental and Control Group

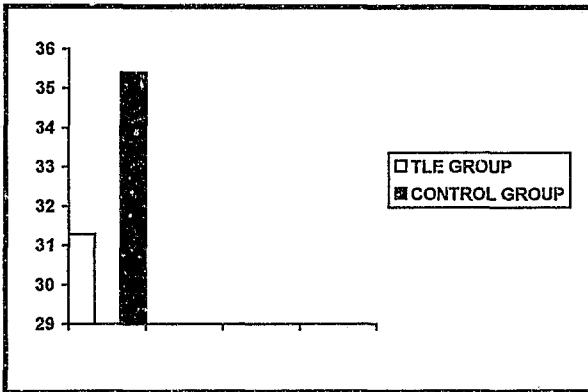


Table 2a

Analysis of Variance for the RCF-COPY score between the Experimental and Control Groups.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
RCF-COPY	411.220	49	212.180	51.17	**0.000
ERROR	4.14667				

**Note: This result is significant at $p < 0.01$.

From figure 2 one can note that the mean for the experimental group (mean=31.28; SD=2.70) is lower than the mean for the control group (mean=35.40; SD=1.00). ANOVA shows that the difference is significant, with the experimental group performing worse on the RCF-COPY. This indicates depressed constructional abilities in these individuals. Also, as the main copy approach adopted by this group as a whole is type IV onwards suggests impaired planning abilities, suggesting problems with planning in the group.

5.2.2. The Rey Complex figure- Immediate Recall.

FIGURE 3

The means of the RCF-immediate recall for the Control and Experimental Group.

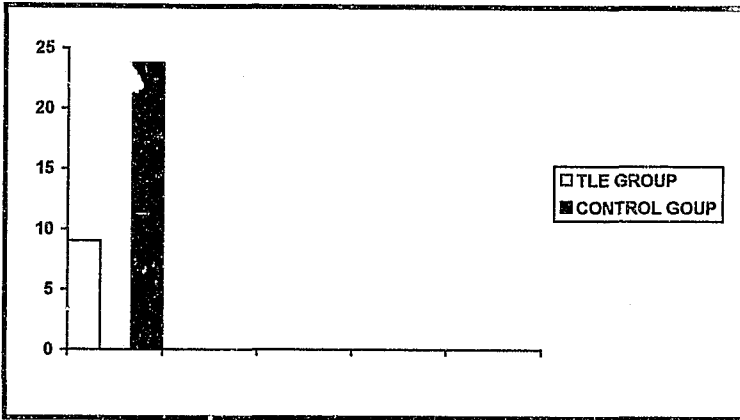


Table 2b

Analysis of Variance for the RCF-IMMEDIATE RECALL between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
RCF-IMM	3685.78	49	2723.22	135.80	**0.0000
ERROR	20.0533				

**Note: This result is significant at $p < 0.01$.

From figure 3 one can note that on the RCF-Immediate Recall the mean performance of the experimental group is lower than the control group's performance (mean=23.76; SD=2.48, for the experimental group; mean=9.00; SD=5.82, for the control group). Also the mean of mean=9.00 for the experimental group falls into the lowest percentile group for the norms of this measure (See Lezak,1983)

The differential performance between the two groups is significant as ANOVA shows a p-value below 0.01 (See table 2b).

5.2.3. The Rey Complex Figure -Delayed Recall.

FIGURE 4

The means of the RCF-Delayed for the Experimental and Control Group.

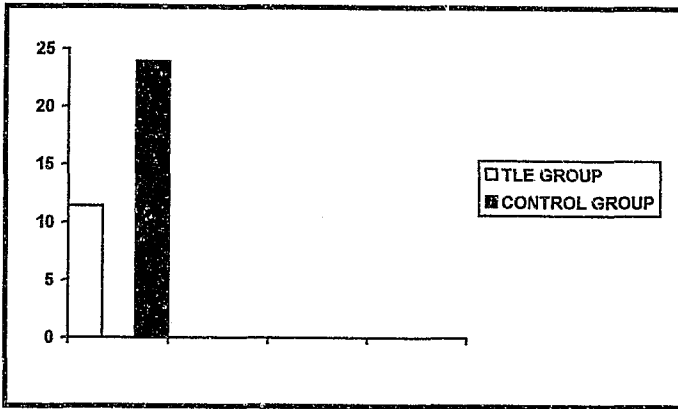


Table 2c

Analysis of Variance for the RCF-DELAYED between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
RCF-DEL	2842.50	49	2086.58	132.50	**0.0000
ERROR	15.7483				

****Note:** This result is significant at $p < 0.01$

Referring to Figure 4, as expected the control group's mean is higher than the experimental group's mean (mean=23.96; SD=2.49 and mean=11.04; SD=5.028, respectively). This result too is highly significant (See Table 2c) The results indicate that the more long-term visual memory store is affected in individuals with complex partial seizures of temporal lobe origin. Interestingly, both the experimental and control groups' mean are higher on the RCF-Delayed Recall compared with the RCF-Immediate Recall. However, ANOVA reports no significant difference between the two trials.

5.3. The R.A.V.L.T.

5.3.1. Trial 1

FIGURE 5

The means for the first trial on the RAVLT between the Experimental and Control Group

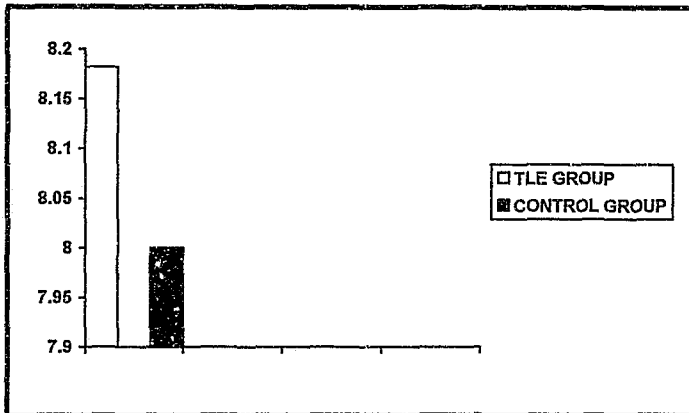


Table 3a

Analysis of Variance for the Trial 1 on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 1	281.660	49	0.38685	0.06	0.8047
ERROR	6.25051				

Note: This result is not significant at $p < 0.05$.

From figure 5 we see that the control group's mean is slightly lower than the experimental group's mean (mean=8.00; SD=2.54 and mean=8.18; SD=2.44, respectively). However, the difference is not significant (See Table 3a). This indicates that immediate verbal retention abilities of patients with TLE fall within normal functional parameters.

5.3.2. Trial 2

FIGURE 6

The means for the second trial on the RAVLT between the Experimental and Control Group

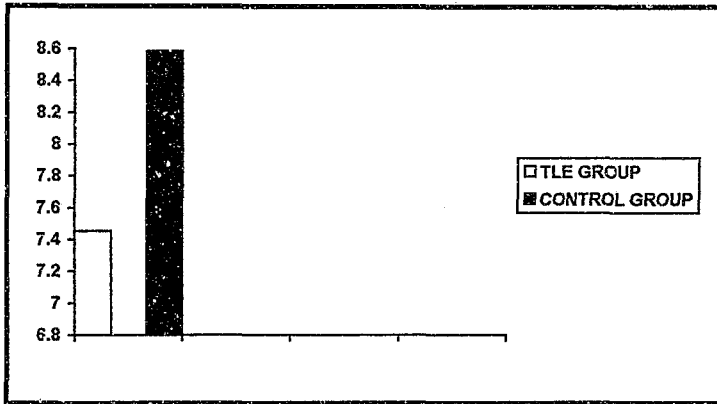


Table 3b

Analysis of Variance for Trial 2 on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 2	349.913	49	14.6252	1.92	0.1729
ERROR	7.62018				

Note: This result is not significant at $p < 0.05$.

Although the control's group's mean performance is better than the experimental group's performance the difference shown by ANOVA (See Table 3b), is not significant - indicating unaffected learning abilities in these individuals.

5.3.3. Trial 3

FIGURE 7

The means for the third trial on the RAVLT between the Experimental and Control Group

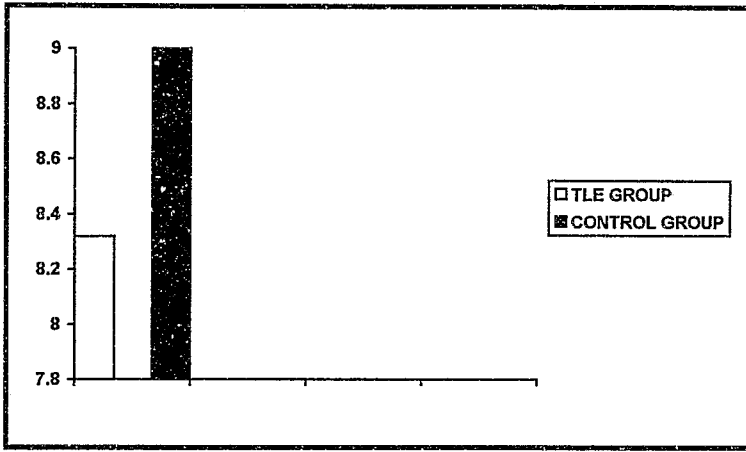


Table 3c

Analysis of Variance for Trial 3 on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 3	268.109	49	5.33597	0.89	0.3497
ERROR	5.97211				

Note: This result is not significant for $p < 0.05$.

As in trial 2, there is a higher mean performance by the control group (See Table 9). However, the difference is not significant.

5.3.4. Trial 4

FIGURE 8

The means for the fourth trial on the RAVLT between the Experimental and Control Group

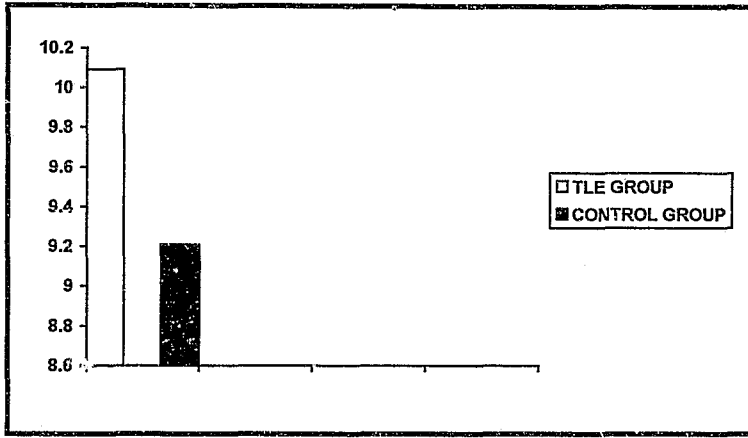


Table 3d

Analysis of Variance for Trial 4 on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 4	224.717	49	8.94088	1.74	0.1937
ERROR	5.13128				

Note: This result is not significant at $p < 0.05$.

As in trial 2 and 4, there is a higher mean performance by the control group (See Figure 8). However, the difference is not significant.

5.3.5. Trial 5

FIGURE 9

The means for the fifth trial on the RAVLT between the Experimental and Control Group

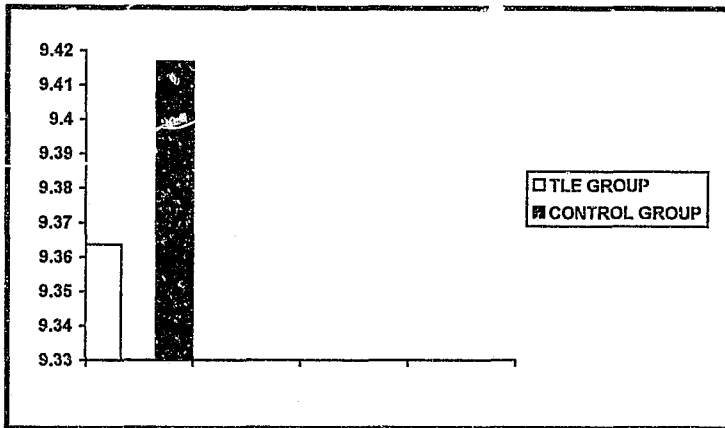


Table 3e

Analysis of Variance for Trial 5 on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 5	214.957	49	0.03228	0.01	0.9356
ERROR	4.88464				

Note: The result is not significant at $p < 0.05$.

As in trials 2,3 and 4 , there is a higher mean performance by the control group (See Figure 9). However, the difference is not significant.

5.3.6. Interference Trial

FIGURE 10

The means for the interference trial on the RAVLT between the Experimental and Control Group

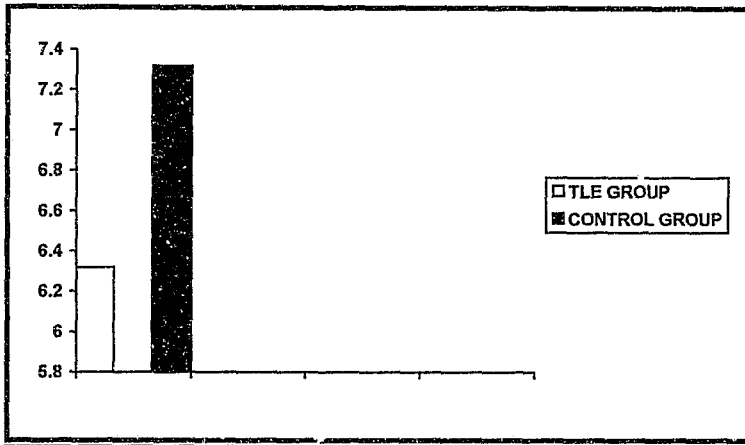


Table 3d

Analysis of Variance for the interference trial on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
INTERFERENCE TRIAL	181.957	49	11.7447	3.11	0.0848
ERROR	53.78251				

Note: The result is not significant at $p < 0.05$.

Although the control group's mean score is better on the interference trial (mean=7.32; SD=1.84, for the control group and mean=6.31; SD=2.08, for the experimental group), ANOVA shows the difference is not significant (See Table 3d).

5.3.7. Trial 6

FIGURE 11

The means for the sixth trial on the RAVLT between the Experimental and Control Group

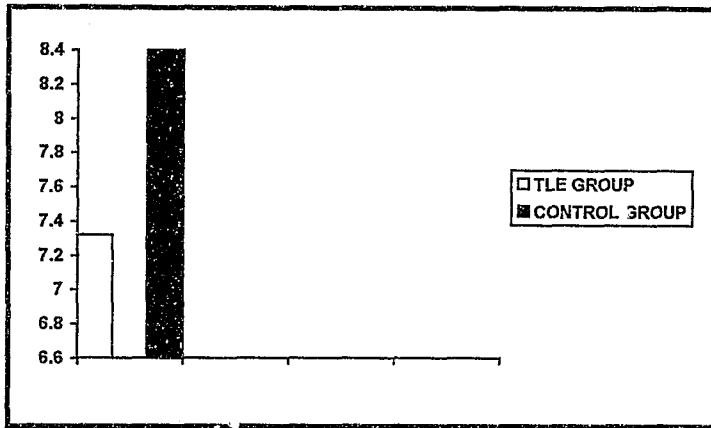


Table 3f

Analysis of Variance for Trial 6 on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 6	254.468	49	13.6954	2.56	0.1166
ERROR	5.35051				

Note: The result is not significant at $p < 0.05$.

A similar result to the first five and interference trials.

5.3.8. The Visual Trial.

FIGURE 12

The means for the visual trial on the RAVLT between the Experimental and Control Group

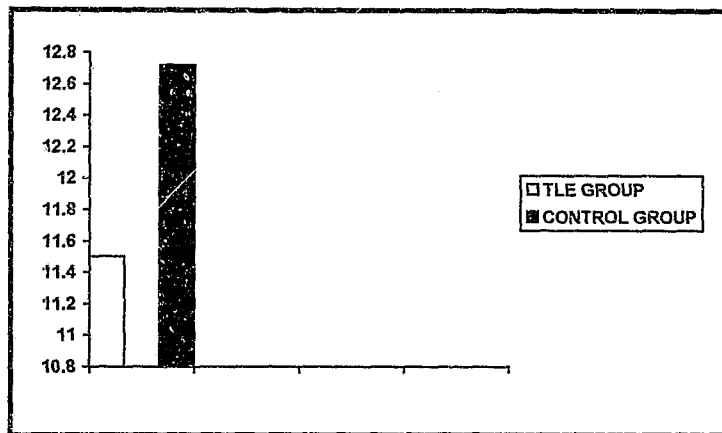


Table 3g

Analysis of Variance for the visual trial on the RAVLT between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
VISUAL TRIAL	267.957	49	17.4174	3.13	0.0837
ERROR	5.56759				

Note: Note the result is not significant at $p < 0.05$.

No significant group differences are reported - suggesting that visual verbal memories are unaffected in the experimental group.

5.3.9. The Delayed Recall Trial.

FIGURE 13

The means for the delayed recall trial on the RAVLT between the Experimental and Control Group

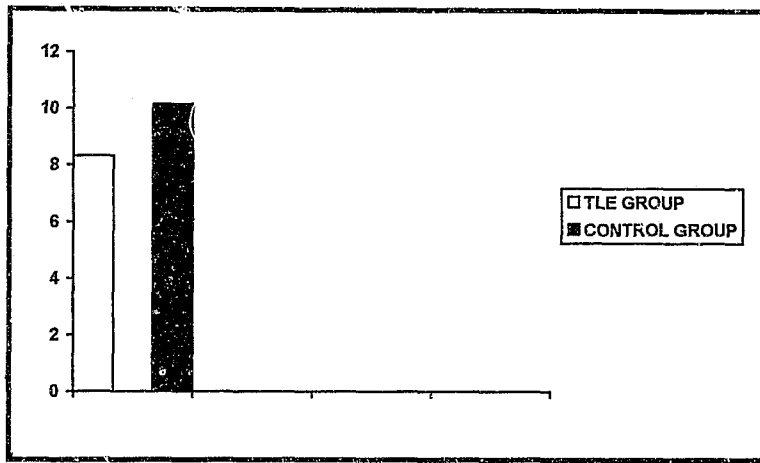


Table 3h

Analysis of Variance for the delayed recall trial on the RAVLT between the Experimental and Control Group.

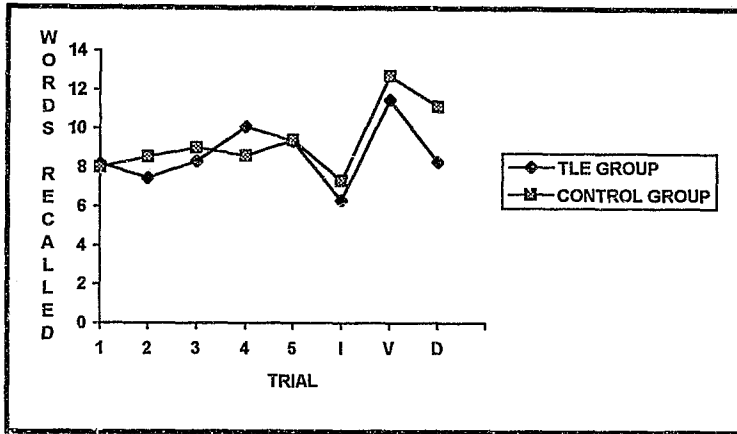
SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
DELAYED TRIAL	439.6971	49	39.6971	4.46	*0.0402
ERROR	8.89184				

*Note: This result is significant at $p < 0.05$.

From Figure 13 we see that the control group's mean (mean=10.16; SD=2.83) is higher in this trial compared with the experimental group's mean performance (mean=8.31; SD=3.13). ANOVA shows (See Table 3h) that the result is significant at $p < 0.05$ - indicating impaired long-term verbal memory store abilities in TLE patients.

FIGURE 14

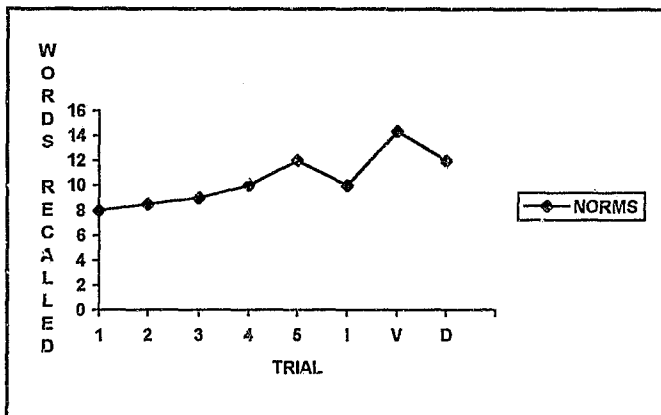
Average number of words recalled for both groups across all trials.



Note: The numbers 1-6 indicate the trial number on the RAVLT. I indicates the Interference Trial and V indicates Visual Trial and D indicates the delayed recall trial.

FIGURE 14B

Normal Performance derived from the Norms for the R.A.V.L.T



Note: The numbers 1-6 indicate the trial number on the RAVLT. I indicates the Interference Trial and V indicates Visual Trial and D indicates the delayed recall trial.

Although both groups display a similar curve in figure 14, neither group performs similar to the norms (figure 14b). A number of reasons may account for this result. One being that the R.A.V.L.T was conducted towards the end of the battery (See Appendix 2), this might have affected the result. Another being an administrative error.

Notwithstanding the previous statements, the experimental group does perform more poorly than the control group, albeit it not statistically, except in the delayed - where the differential performance is significant.

5.4. The Wisconsin Card Sorting Test.

5.4.1. Categories Satisfied.

FIGURE 15

Comparison of the means of the number of categories satisfied on the Wisconsin Card Sorting Test of the experimental and control groups.

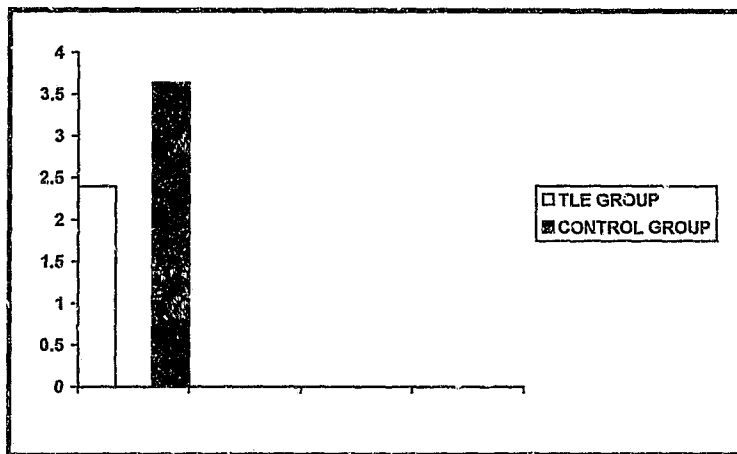


Table 4a

Analysis of Variance for the number of categories satisfied on the WCST between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
CATEGORIES SATISFIED	74.9800	49	19.2200	16.55	**0.0002
ERROR	1.16167				

**Note: The result is significant at $p < 0.01$.

From figure 15 the mean of the number of categories satisfied by the experimental group (mean=2.40; SD=1.00) is lower than the control group's mean (mean=3.64; SD=1.15). ANOVA, (See Table 4a), shows that the difference between the two groups is highly significant, $p < 0.01$ -

indicating a possible frontal dysfunction in the sample of TLE patients, or rather impaired shift of set abilities.

5.4.2. Perseverate Errors on the WCST.

FIGURE 16

The means for the number of perseverate errors on the WCST between the Experimental and Control Group

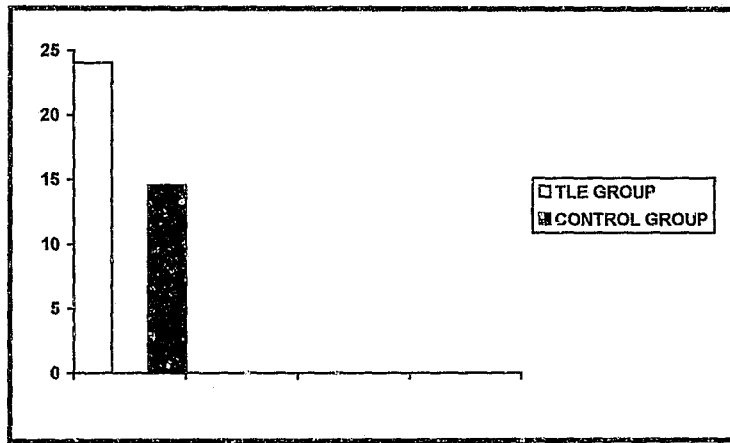


Table 4b

Analysis of Variance for perseverate errors on the WCST between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN-SQUARE	F-RATIO	P-LEVEL
PERSEVER- ATION	3200.50	49	1123.38	25.96	**0.0000
ERROR	43.2733				

****Note:** This result is significant at $p < 0.01$.

The mean of perseverate errors in the experimental group is higher than the control group (mean=24.04; SD=7.53 and mean=14.56; SD=5.46, respectively). Noting the information contained in table 4b we see that ANOVA shows that the differential mean amount of perseverate errors is significant at $p < 0.01$.

5.5. THE AUSTIN MAZE

5.5.1. TRIAL 1

FIGURE 17

Comparison of the means for the first trial on the Austin Maze between the Experimental and Control Group

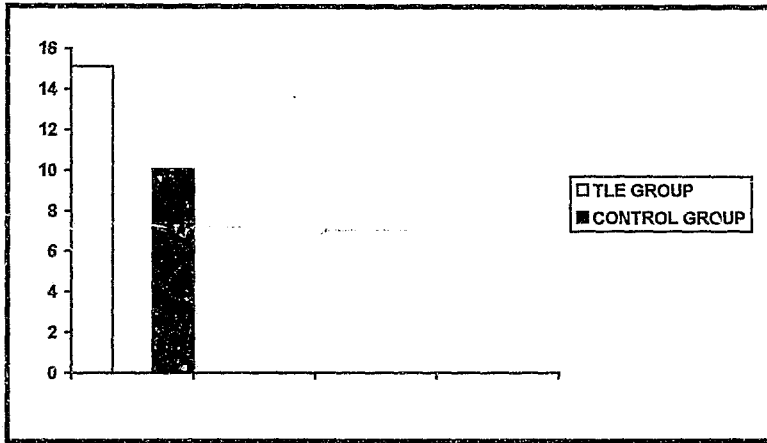


Table 5a

Analysis of Variance for Trial 1 on the Austin Maze between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
Trial 1	1953.16	42	59.6980	1.32	0.2563
ERROR	1953.16				

*Note: This result is not significant at $p < 0.05$.

MISSING CASES FROM EXPERIMENTAL GROUP = 6

The experimental group's mean performance is worse (but not significantly) in the first trial of the Austin Maze as indicated by the higher mean number of errors than the control, mean=15.63; SD=8.40, for the experimental group compared to mean=13.28, SD=5.09 for the control group. ANOVA shows that the difference not significant at $p < 0.05$ (See Table 5a).

A number participants from the experimental group on this task were unable to complete the task. This is indicated by the note "MISSING CASES". Unfortunately, there is insufficient literature to explain this phenomena. The same pattern is seen through all trials.

5.5.2. Trial 2

FIGURE 18

The means for the second trial on the Austin Maze between the Experimental and Control Group

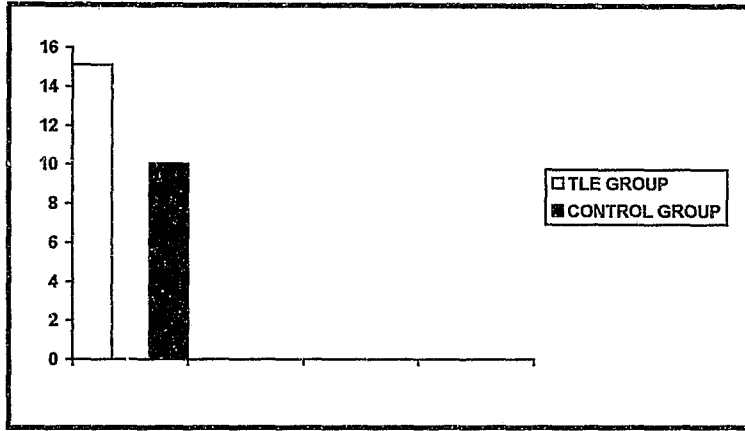


Table 5b

Analysis of Variance for Trial 2 on the Austin Maze between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 2	255.73	43	276.978	5.11	*0.0291
ERROR	54.2559				

*Note: This result is significant at $p < 0.05$.

MISSING CASES IN THE EXPERIMENTAL GROUP = 6

A similar performance to trial 1. ANOVA shows that the control's performance is significantly better, $p < 0.05$ (See Table 5b).

5.5.2 TRIAL 3

FIGURE 19

The means for the third trial on the Austin Maze between the Experimental and Control Group

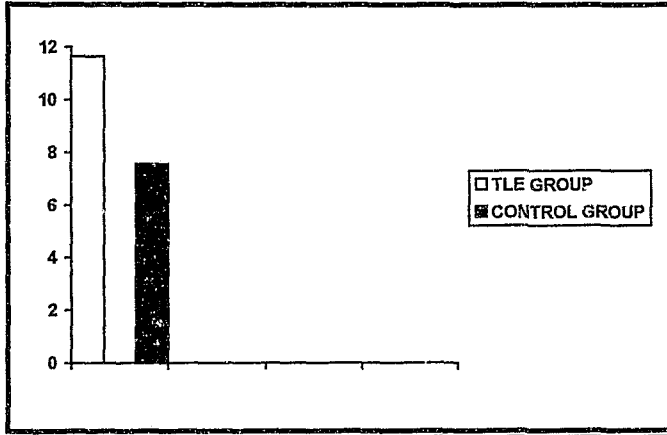


Table 5c

Analysis of Variance for Trial 3 on the Austin Maze between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 3	1203.22	43	178.964	7.34	*0.0097
ERROR	24.3948				

*Note: This result is significant at $p < 0.05$.

MISSING CASES IN THE EXPERIMENTAL GROUP = 6

The control group performs significantly better than the experimental group in this trial, with the result showing more significance [$p < 0.01$] than the first two trials.

5.5.4. TRIAL 4

FIGURE 20

The means for the fourth trial on the Austin Maze between the Experimental and Control Group

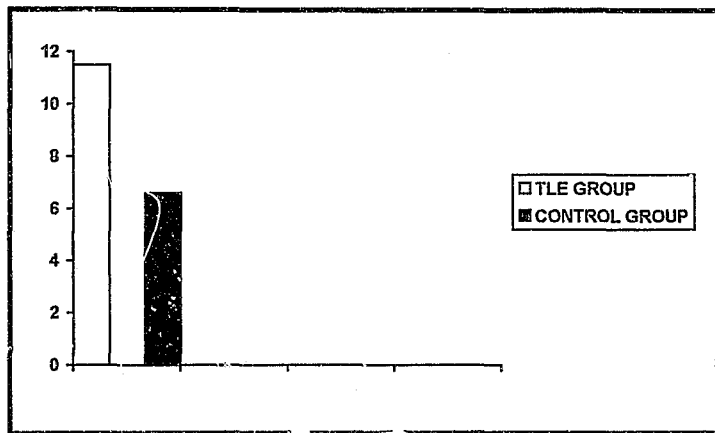


Table 5d

Analysis of Variance for Trial 4 on the Austin Maze between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
TRIAL 4	1803.77	41	251.267	6.64	*0.0137
ERROR	37,8659				

*Note: This result is significant at $p < 0.01$.

MISSING CASES IN THE EXPERIMENTAL GROUP = 7

A similar result to the third trial.

5.6. Digit Span.

5.6.1. Digit Span Forward.

FIGURE 21

Comparison of the means for the Digit Span Forward between the Experimental and Control Group.

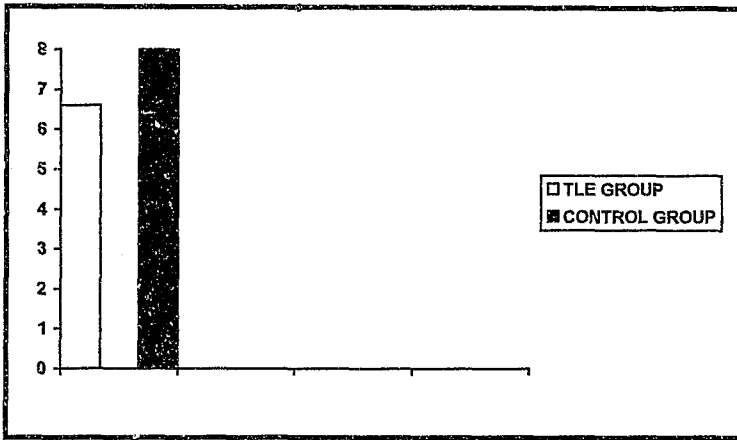


Table 6a

Analysis of Variance for the Digit Span Forward between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
DIGIT SPAN FORWARD	78.5000	49	24.5000	21.78	**0.0000
ERROR	1.12500				

**Note: This result is significant at $p < 0.01$.

The experimental group's mean on the Digit Span is significantly lower than the control group's performance. mean=6.6; SD=1.08, for the experimental group and mean=8.0; SD=1.04, for the control group (See Figure 21 and Table 6a).

5.6.2. Digit Span Backwards.

FIGURE 22

Comparison of the means for the Digit Span Backwards between the Experimental and Control Group

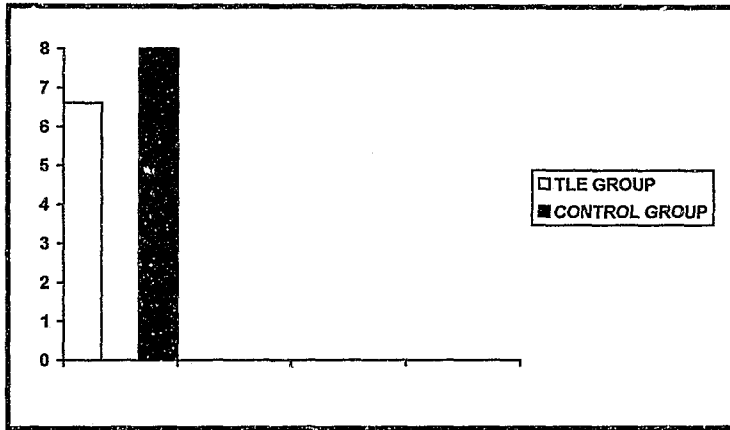


Table 6b

Analysis of Variance for the Digit Span Backwards between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
DIGIT SPAN BACKWARD	161.620	49	100.820	79.59	**0.0000
ERROR	1.26667				

****Note:** This result is significant at $p < 0.01$.

The experimental group's mean on the Digit Span is significantly lower than the control group's performance (See Figure 22 and Table 6a).

5.7. Similarities.

FIGURE 23

Comparison of the means on the Weschler Test of Similarities between the Experimental and Control Group

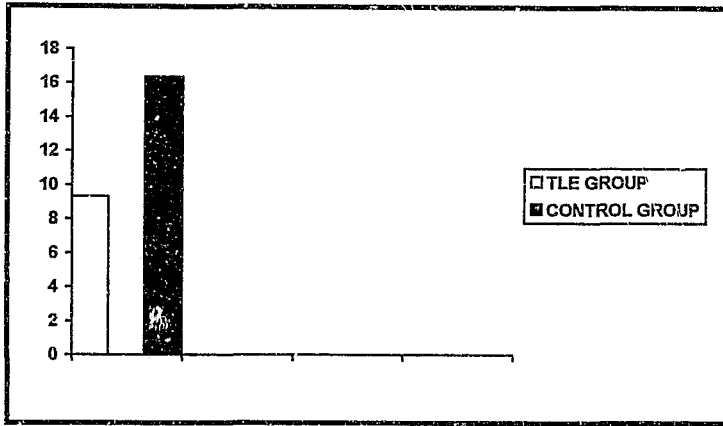


Table 7

Analysis of Variance for performance on the Weschler Test of Similarities between the Experimental and Control Group.

SOURCE	SUM-OF-SQUARES	df	MEAN SQUARES	F-RATIO	P-LEVEL
SIMILARITIES	1232.98	47	596.350	43.09	**0.0000
ERROR	13.8398				

**Note: This result is significant at $p < 0.01$.

MISSING CASES = 2

The experimental group's mean (mean=9.30; SD=4.51) compared with the control group's mean (mean=16.38; SD=2.79) indicates a poor mean performance by the TLE sample subjects. As one can note from Table 7, ANOVA shows this difference as highly significant, $p < 0.01$.

5.8. The Tower of London Test.

FIGURE 24

Comparison of the means of the number of moves on the Tower of London Test between the Experimental and Control Group

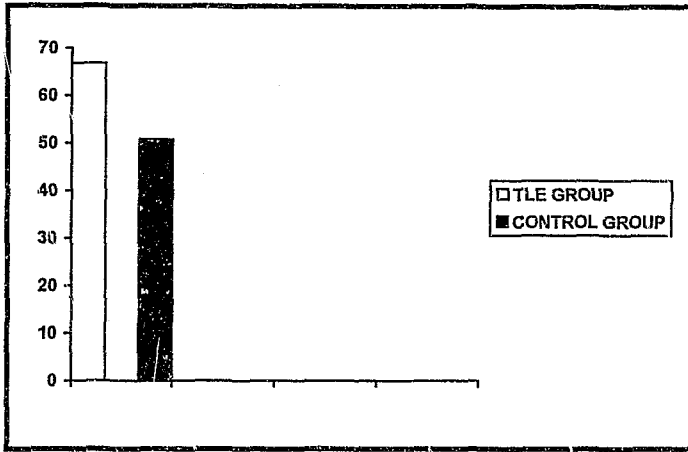


Table 8

Analysis of Variance for the number of moves on the Tower of London Test between the Experimental and Control Group.

SOURCE	SUM OF SQUARES	df	MEAN SQUARE	F-RATIO	P-LEVEL
# OF MOVES	6955.40	46	3017.57	34.48	**0.0000
ERROR	87.5074				

**Note: This result is significant at $p < 0.01$.

MISSING CASES = 3

The experimental group displays a higher mean number of moves mean=66.81, SD=12.195, indicating a poorer performance compared to the control's mean mean=50.76, SD=5.82. From table 8 we see that ANOVA shows this difference to be highly significant, $p < 0.01$.

5.9. Correlated Performance of the Experimental Group between the Measures.

Table 9.

Results of Pearson Correlations conducted on the Experimental Group's performance.

	RCF-COPY	RCF-IMM	RCF-DEL	RAVLT-IV	WISC-CAT	WISC-PER	AUSTIN-4	DS-F	DS-B	SIMILARITIES	T.OF LON
RCF-COPY	1.00										
RCF-IMM	0.76	1.00									
RCF-DEL	0.72	0.77	1.00								
RAVLT-IV	0.33	0.49	0.45	1.00							
WISC-CAT	0.65	0.71	0.75	0.38	1.00						
WISC-PER	-0.72	-0.68	-0.66	0.29	-0.82	1.00					
AUSTIN-4	-0.63	-0.59	-0.55	0.50	-0.65	-0.67	1.00				
DS-F	0.59	0.66	0.75	0.57	0.62	-0.66	-0.53	1.00			
DS-B	0.80	0.75	0.73	0.52	0.73	-0.72	-0.62	0.75	1.00		
SIMILARITIES	0.74	0.76	0.68	0.29	-0.63	-0.59	-0.69	0.58	0.63	1.00	
LONDON	-0.75	-0.71	-0.66	-0.24	-0.71	0.82	0.77	0.57	0.60	-0.70	1.00

*Note: negative correlations are an artefact of different scoring systems. With a negative correlation a better performance on one test is a high score whilst on the other test a better performance is a low score and visa versa.

From the above table one can see that on most memory measures a performance by a participant in the experimental group is met with a correlated performance on the measures of executive function. Thus, it appears that when there is fall out on measures of memory, there is also fall out on the measures of executive function. However, there is a poor correlation with most other measures and the RAVLT*, indicating that functions described by this measure are preserved in the experimental group.

Also there is a high correlation amongst the measures of executive function, indicating that in this group there is a uniformity of an individual's performance on all measures of executive function. Further evidence that the majority of these patients experience fall-out in executive function in general.

* Trial IV to conduct correlat.

CHAPTER 6

DISCUSSION

6.1. Summary of Experimental Group Performance Compared with the Normals.

Table 1

Summary of Cognitive Performance of the Experimental Group.

TEST	MAIN CONSTRUCT ASSESSED	PERFORMANCE OF EXPERIMENTAL GROUP COMPARED WITH CONTROL GROUP	P-VALUE
RCF-COPY	CONSTRUCTIONAL ABILITIES	POOR	0.000
RCF-IMMEDIATE RECALL	SHORT TERM VISUAL MEMORY	POOR	0.000
RCF-DELAYED RECALL	LONGER TERM VISUAL MEMORY	POOR	0.000
R.A.V.L.T (TRIALS 1-6)	LEARNING	NO DIFFERENTIAL PERFORMANCE	ALL P-VALUES IN TRIALS 1 -6 ABOVE 0.05
R.A.V.L.T DELAYED RECALL	LONG TERM VERBAL MEMORY	POOR	0.0402
AUSTIN MAZE (THE FIRST TRIAL IS NOT INCLUDED)	TOPOGRAPHICAL MEMORY AND ERROR UTILISATION	POOR	P-VALUES FOR TRIALS 1 AND 2 ABOVE 0.05. P-VALUES FOR TRIALS 3 AND 4 BELOW 0.05.
DIGIT SPAN FORWARD	ATTENTION AND WORKING MEMORY	POOR	0.000
DIGIT SPAN BACKWARDS	WORKING MEMORY MENTAL TRACKING	POOR	0.000
SIMILARITIES	CONCEPT FORMATION	POOR	0.000
WISCONSIN CARD SORTING TEST-CATEGORIES SATISFIED	SHIFT OF SET AND ABSTRACT BEHAVIOUR	POOR	0.000
WISCONSIN CARD SORTING TEST-PERSEVERATIVE ERRORS	MENTAL PESEVERATION	POOR	0.000
TOWER OF LONDON	PLANNING ABILITIES	POOR	0.000

From table 1 it appears (on the previous page) that the experimental group performs worse in the areas of attention, tracking, short-term-memory, long-term-memory and visual memory. Also, the experimental group exhibits significantly increased perseveration, depressed abstract thinking abilities and poorer planning. In addition they cannot shift-set as efficiently as the control group and show poor self-monitoring abilities.

Although the experimental group's performance is inferior to that of the controls, their performance on tests of verbal immediate memory is contradicted by their performance on the R.A.V.L.T.. The experimental group exhibits a normal learning curve and display normal short-term verbal memory, with regards to the controls. However, the experimental group performs statistically poorer than the controls on the delayed recall, indicating a problem with long-term memory store or encoding. This will be further discussed in section 6.3.

Although the experimental group did not perform significantly worse on Trial 1 of the Austin Maze ($p = 0.2563$), they performed significantly differentially on all other trials, suggesting that their performance on Trial 1 is coincidental. Interestingly, only the experimental group contained participants who were unable to complete the Austin Maze and the test had to be stopped. Unfortunately, there is no literature that the author is aware of that explains this phenomenon.

On the whole the patients with complex partial seizures of temporal lobe origin score significantly worse on the measures of executive function and memory, the only exception being the normal performance on the R.A.V.L.T. (excluding the delayed recall trial). The experimental group also displayed a normal learning curve. The results from the R.A.V.L.T., indicate that these patients' verbal short term memory and learning abilities are unaffected. However, their poor performance in the delayed recall indicated that these individuals do as a group have a deficit in the area of long term memory. Whether this is due to encoding problems or retrieval difficulties is difficult to determine.

The strong correlations (see table 9, chapter 5) of the performance by the experimental group between the measures of memory and executive function suggests that patients with complex partial seizures presenting with memory deficits probably will also be experiencing an executive function deficit.

Table 2

Mean Scores and Standard Deviations for the Two Groups Across All Tests

TEST	EXPERIMENTAL GROUP		P-VALUE	CONTROL GROUP	
	MEAN SCORE	SD		MEAN SCORE	SD
RCF-COPY	31.28	2.70	0.00**	35.40	1.00
RCF-IMM	9.00	5.82	0.00**	23.76	2.48
RCF-DEL	11.04	5.02	0.00**	23.96	2.49
RAVLT TRIAL 1	8	2.54	0.80	8.18	2.44
RAVLT TRIAL INTERFERENCE	6.31	2.08	0.08	7.32	1.89
RAVLT TRIAL DELAYED	8.31	3.13	0.04*	10.16	2.83
WCST SAT. CATEGORIES	2.40	1.00	0.00**	3.64	1.15
WCST ERRORS PERSEVERATE	24.04	7.53	0.00**	14.56	5.46
AUSTIN MAZE TRIAL 1	15.63	8.40	0.25	13.28	5.09
AUSTIN MAZE TRIAL 2	15.22	5.32	0.02*	10.40	5.32
AUSTIN MAZE TRIAL 3	11.80	6.04	0.00*	7.3	4.72
AUSTIN MAZE TRIAL 4	11.04	6.22	0.01*	6.2	4.95
DIGIT-SPAN FORWARDS	6.60	1.08	0.00**	8.00	1.04
DIGIT-SPAN BACKWARDS	6.50	1.01	0.00**	7.70	0.79
SIMILARITIES	9.30	4.51	0.00**	16.38	3.79
TOWER OF LONDON	66.81	12.19	0.00**	50.76	5.82

Note: The p-values are represented in table 1. * indicates significance <0.05. ** indicates significance <0.01.

6.2. Concentration.

The experimental group's performance on the Digit Span (both forwards and Backwards) is significantly below the normal group's performance - this indicates that these patients as a group may have low attention spans and depressed short-term memory functions, although there are contradicting results to this notion. (See section 6.5).

6.3. Memory.

The experimental group's performance on the R.A.V.L.T suggests that their verbal short-term-memory is unaffected. They do not perform statistically lower than the control group, but their results on the R.A.V.L.T are, on average, lower. This result somewhat contradicts their performance on the Digit Span. However, as the Digit Span is more sensitive to attentional deficits; this taken together with their performance on the R.A.V.L.T suggests that the experimental group's short-term memory functions fall within normal parameters when attentional deficits are taken into account.

Interestingly, the experimental group does not perform differentially on any of the RAVLT learning trials (trials 1-5). This suggests that short-term-memory and learning is unaffected and only remote-memory is affected in the experimental group. Furthermore, it is arguable that as the experimental group displays a normal learning curve on the RAVLT, this is more evidence that the results obtained on the Digit Span are due to attentional deficits in this group. Thus, this suggests that the subjects in the experimental group do not possess retroactive or proactive interference tendencies, nor tendencies to confusion or confabulation on memory tasks.

The experimental group's poor performance on the R.A.V.L.T delayed recall could be attributed to encoding problems, one may concluded that there are problems with encoding rather than retrieval because these scores on the are not significantly poorer on the task which gives them cues. Furthermore, the experimental group's performance on the R.A.V.L.T is similar to what is seen from patients with frontal damage.

6.3.2. Visual memory.

The poor performance by the experimental group on the RCF-immediate recall and RCF-delayed recall indicates that patients with TLE may perform poorly on tasks requiring visual memory. However, there are a number of possibilities that may cause the observed performance.

Firstly, concentration difficulties, as exhibited by the experimental group in their performance on the Digit Span, may affect the individual's ability to adequately perceive the relatively complex details found on the RCF.

Secondly, poor constitutional abilities may abate their performance on the memory trials of the RCF. As mentioned in Chapter 5, the experimental group favoured a type IV (onwards) approach when completing the RCF-copy. As some researchers and clinicians hold the belief that the copy approach to the RCF affects the scores obtained in the subsequent memory trials of the test.

However, owing to the experimental group's performance on the RAVLT-delayed (where they only performed poorly on the delayed trial) a long-term memory deficit may indeed be a factor in the results obtained. Most likely, a combination of all these factors is responsible for the poor performance.

6.5. Executive Function.

The experimental group displays an almost "frontal" damage cognitive pattern, with increased perseveration, inefficiency in shift-set, poor abstract thought, planning difficulties, poor self-monitoring and error-utilisation.

The entire construct of executive function (with all its sub-groups) appear depressed in the experimental group. Their poor performance on the Wisconsin Card Sorting Test (both perseverative errors and categories satisfied), the poor performance on the Tower of London, the poor performance on the Weschler Similarities Test, and the performance on the Austin Maze all indicate a definite executive function problem. Interestingly, the manner on which they approached the RCF-copy also points towards an executive function deficit.

This finding is in keeping with Hermann, *et al.*, (1995) who believe that the poor inter-ictal performance on measures of executive function is due to an abnormal discharge in the temporal lobes reaching the frontal areas. The finding that a poor performance on the memory measures (temporal lobes) is correlated with a poor performance on the executive function measures (frontal lobes) suggest that a temporal lobe dysfunction has frontal affects. In other words, the good correlations achieved between the measures of memory and executive function are in keeping with Hermann, *et al.* (1995), where they suggest that a poor performance on the WCST, both with regards to categories satisfied and perseveration response is due to a discharge from the temporal lobes to the frontal lobes. The results achieved support Hermann, *et al.* (1995) *nociferous* hypothesis because if there was no correlation between an experimental group participant's scores on memory and executive function measures it would that indicate that although as group these patients perform more poorly on measures of executive function, the poor performance on

executive function measures is not related to memory deficits, thereby contradicting the *nociferous hypotheses*.

Although the poor performance on the certain measures of executive function produced by patients with complex partial seizures of temporal lobe origin have been known about for over three decades (see. Milner, 1963), little research has been directed towards the entire construct of executive function. With the majority of the research directed at only a small part of the overall construct; namely shift-of-set and perseveration. Future studies need to be directed at other believed frontal mediate higher functions.

In conclusion this group displayed deficits on all measures of executive function and executive function is arguably one of the most important higher functions for adequate functioning the affects that these deficits have on individuals with complex partial seizures of temporal lobe origin need to be further investigate.

6.6. Conclusion.

It is important to reiterate that the issues concerning medication remain unresolved. However, from a practical perspective, these individuals need to function in society medicated and these cognitive deficits need to be taken into account when planning management. The results suggest that these individuals show a frontal-like-syndrome, and two theories have been developed to attempt to explain this performance, namely the Nociferous Hypothesis and Hippocampal Hypothesis.

The results support the findings by Hermann, Wyler and Ritchey (1988) as the experimental group significantly satisfied fewer categories on the WCST. The experimental group also displayed increased perseveration in the WCST. Thus, this research project's findings corroborate those of Hermann, Wyler and Ritchey (1988) and the replication research studies of Corcoran and Upton (1992), Hermann, *et al.* (1991), Strauss *et al.* (1993) and Trenerry and Jack (1994).

The test battery used in this study employs measures that assess more aspects of the construct of executive function. Tracking, planning, abstract thought and error utilisation have been investigated and it appears from the results that the experimental group is impaired on all utilised measures of executive functioning. One must naturally ask what the consequences of this are in the individual's life, and this is an important area for future psychological research.

The effects of age-of-onset, seizure frequency and laterality on executive function cannot be answered by this study, and these are also important areas for future neuropsychological research.

Although the results on the Digit Span from this study have produced similar results to those found by Grippo, *et al.* (1996) the conclusions reached by these authors is not supported by this study. These authors concluded that depressed short-term-memory is present in individuals diagnosed with temporal lobe epilepsy; however, all their measures of short-term memory are very sensitive to attentional deficits. Therefore, the hypothesis discussed in section 6.3 is not contradicted by the Grippo, *et al.* (1996) study.

The findings with regard to memory contradict the reported findings of Mirsky, Primac, Marsan, Rosvold and Stevens, (1960), Scott, *et al.* (1967), Migone, *et al.* (1970), Milstein and Goldstein (1972), Rodin, *et al.* (1976) and Mayeux, *et al.* (1980) who do not report any significant memory impairment in TLE patients. The results of the this research project are equivocal with the findings of Dennerll, (1964), Schwartz and Dennerll, (1969) and Hermann, *et al.* (1987), although the results of this research project cannot provide insights into the effects that laterality of seizure focus has on memory.

The first aim of this study was to investigate the performance of patients with complex partial seizures of a temporal lobe origin on a wide range of executive function tasks, than has previously been undertaken. From the results we may conclude that patients with temporal lobe epilepsy have a higher probability of executive function deficits.

The second aim was to investigate memory function in the TLE population group and then correlate it with executive function performance. Although the majority of the neuropsychological research concerning temporal lobe epilepsy has focused on memory, there is no consensus amongst researchers and in the literature as to which aspects of the complex process of memory are affected in this disorder. Therefore, these research findings may be added to the large amount of contradicting findings that presently exist. The present study found that remote visual and verbal memory and executive function to be impaired. The findings regarding immediate memory were, unfortunately, contradicting.

Finally, the fact that good correlations exist between the performance by the experimental group on the measures of memory and the measures of executive function indicate that a patients with complex partial seizures of temporal lobe origin with affected memory abilities are likely to display deficits in constructs of executive function.

6.7. Limitations and Flaws in the Research.

There are two main flaws to this research project that may limit the validity of the results. Firstly, this study does not control for medication or laterality of focus. Thus, no firm conclusions regarding these two variable can be drawn. Fortunately, the literature does accept the validity of studies that have not controlled for these two variables.

The study of higher function in patients with both Axis I and Axis III disorders is plagued with the problem of medication variable affects. This has complicated research and often produces research with debatable findings. Medication has not been adequately controlled for in this study for two reasons. Firstly, this study is not anatomically based, that is it does not make any claims as to which brain structures mediate which higher functions, but rather it seeks to describe what clinicians would expect to observe in their TLE patients that are on anticonvulsant medication.

Secondly, as the current research on anticonvulsant cognitive effects suggests that anticonvulsant medication has few effects on cognition, we need not be as concerned as we were when conducting research in this area. Especially since it is often extremely difficult, if not impossible, both logistically and ethically in neuropsychological research to control for medication.

Although both the experimental and control group are comprised from members of the same racial group, there is no matching on a socio-economic variable, furthermore, this variable was not quantitatively assessed, even though there are tests that can measure this construct. Unfortunately, they were, by error, not included in the original protocol and is a flaw in the research.

6.8. Suggestions for Further Research.

Further research is needed in the following areas.

- 1) Which variables affect the patients performance on executive functions assessment measures. Variables such as age-of-onset, seizure laterality, medication type.
- 2) What consequences depressed executive function abilities have on the patients psychosocial functioning.
- 3) Which areas of aspects of memory are affected. For example, do patients exhibit more an encoding deficit or retrieval deficit, etc.
- 4) Affects of anticonvulsants on cognition, especially the more modern pharmaceuticals and polytherapy.

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8. APPENDICIES

APPENDIX 1.

Demographic Data Collection Sheet

Subject #: _____
Subject Information
Place Assessed _____
Time: _____
Date: _____
Name: _____
Hospital #: _____
Date of Birth: _____.
Age: _____
Gender: _____
Home Language: _____
HLOE: _____
Occupation: _____
Handedness: _____
Other Disorders (Including Axis 1 and 2) _____
Referring Doctor: Dr. _____
Medication: _____
Period of time on this medication: _____
Serum Level: _____
Age that TLE started: _____
Head Trauma (if so when and how severe): _____
R-TLE or L-TLE: _____
Do the seizures generalise? _____
Are the seizures controlled? _____
If yes then # of times grand mal occurred: _____
Is consent formed signed: _____
Any other relevant information: _____

APPENDIX 2.

Results Sheet

1) RAVENS STANDARD PROGRESSIVE MATRICIES

A1	B1	C1	D1	E1
A2	B2	C2	D2	E2
A3	B3	C3	D3	E3
A4	B4	C4	D4	E4
A5	B5	C5	D5	E5
A6	B6	C6	D6	E6
A7	B7	C7	D7	E7
A8	B8	C8	D8	E8
A9	B9	C9	D9	E9
A10	B10	C10	D10	E10
A11	B11	C11	D11	E11
A12	B12	C12	D12	E12

2) REY COMPLEX FIGURE - COPY AND IMMEDIATE RECALL

COPY SCORE _____ TYPE _____ (DO NOT SCORE)
IMMEDIATE SCORE _____

3) RAVLT

SCORES 1 _____ 2 _____ 3 _____ 4 _____ 5 _____
INTERFER _____ 6 _____ VISUAL _____ DEL _____

4) ARITHMETIC (SA-WAIS)

SCORE: _____

5) WISCONSIN CARD SORTING

SCORES: (DO NOT SCORE)
CAT SAT: _____ . PERS.: _____ ERRORS: _____

6) AUSTIN MAZE

LEARNING TRIAL # ERROR: _____

TRIAL 1: _____

TRIAL 2: _____

TRIAL 3: _____

TRIAL 4: _____

REMARKS:

7) DIGIT SPAN (SA-WAIS)

FORWARD SCORE: _____

BACKWARDS SCORE: _____

8) SIMILARITIES

1) _____

2) _____

3) _____

4) _____

5) _____

6) _____

7) _____

8) _____

9) _____

10) _____

11) _____

12) _____

SCORE: _____ (DO NOT SCORE)

9) CONTROLLED ORAL WORD ASSOCIATION + FRUITS AND ANIMALS

F # OF WORDS. _____ . REPEATS: _____ .

A # OF WORDS. _____ . REPEATS: _____ .

S # OF WORDS. _____ . REPEATS: _____ .

FRUITS # OF WORDS: _____ . REPEATS: _____ .

ANIMALS # OF WORDS: _____ . REPEATS: _____ >

10) TOWER OF LONDON

MARK WITH A FORWARD SLASH THE NUMBER OF MOVES

ASTERICS IF PATIENT GIVES UP

2M: _____

2M: _____

3M: _____

3M: _____

4M: _____

4M: _____

4M: _____

5M: _____

5M: _____

5M: _____

TOTAL # OF MOVES: _____

10) TEST OF REMOTE MEMORY

SCORE: _____

11) RCF-DELAYED RECALL (DO NOT SCORE)

SCORE: _____

12) RAVLT- DELAYED RECALL

CORRECT: _____ INTRUSIONS: _____ ERRORS: _____

13. B.D.I

SCORE: _____.

TIME OF FINISH: _____

APPENDIX 3

Subject Information and Consent Form

Subject # 018

SUBJECT INFORMATION AND CONSENT FORM

My name is *Grant Schofield and I am conducting research on a number of areas of cognition (patterns of thinking) with people diagnosed with temporal lobe epilepsy.

We would appreciate it if you would take part in a 90 minute session where you would be asked to complete a number of neuropsychological tasks, which consist of written work and answering questions. Also we may need to take some of your blood in order to see at what level your medication currently is. This research aims to provide us with the effects that your epilepsy has upon your cognition.

The information obtained will be kept confidential. The results of the tests will be confidential, and at no time will your name be released to anyone besides your doctor.

Participation in this study is completely voluntary, and you are free to participate or withdraw your consent and to discontinue participation at any time. A signed copy of this form will be made available to you should require one.

I have fully explained the procedures, identified those which are investigational, and have explained their purpose.

DATE: _____ Grant Schofield: _____

I have been fully informed as to the procedures to be followed. In signing this consent form, I agree to this method of testing and I understand that I am free to refuse to participate or withdraw my consent and discontinue my participation in this study at any time. I understand also that if I have any questions before or after the session, they will be answered.

DATE: 25/5/97 Subject/Patient: Grant

* I may not be the individual conducting the tests with you. If Felicity is unable to answer any questions you have, or if you would like to ask me any questions please feel free to telephone me on 647-2349 (W)

Author Schofield G M

Name of thesis Executive Function And Memory Deficits In Patients With Complex Partial Seizures Of Temporal Lobe Origin
Schofield G M 1998

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