RETROACTIVE EFFECTS OF ECT ON MEMORY

by

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DECLARATION

I declare that the dissertation for the degree M.Sc.(Clin.Psych.) at the University of the North hereby submitted by me, has not previously been submitted by me for a degree at this or another university, and that it is my own work in design and in execution and that all material contained therein has been duly recognized.

DEDICATED TO

The troubled souls and those who have devoted their lives to help them.

"Of the many treatments of the severe mentally ill, none is so effective for specific clinical disorders, and so misunderstood as induced convulsions"

Katz, 1974, pxi.

"How is it that one branch of medicine - neurology strives to prevent convulsions, while another branch - psychiatry uses convulsions as therapy: is this not an odd ethical dilemma"

Friedberg, 1976, p.20.

OUTLINE OF CONTENTS

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Chapter 1

NATURE AND PURPOSE OF THE INVESTIGATION

1.1 Introduction

Electroconvulsive therapy (ECT) is a form of treatment commonly used in contemporary psychiatric practise. It is induced by administering a controlled amount of electrical current to the brain. The treatment produces cerebral phenomena corresponding to a grand mal seizure in epilepsy. Tonic and clonic convulsions of the body muscles occur unless they are blocked by muscle relaxants. The therapeutic effect rests upon the seizure event. Muscular contractions are of no significance (Heshe et al, 1978).

ECT has been introduced in 1938 by Italian physicians, Cerletti and Bini (Arnot, 1975). Its innovation has been considered a great humanistic triumph in convulsive treatment because of the simplicity, safety and reliability of the method of electrical induction. The treatment has replaced pharmacological convulsive therapies e.g. camphor which were difficult to induce, unsafe, shock-producing and distressful to patients (Ilaria and Prange, 1975).

Convulsive therapy was used for the first time by Oliver in 1785 (Ilaria and Prange, 1975). He produced the convulsions by administering camphor.

The rationale of convulsive treatment and impetus to its development was however provided by Von Meduna in Hungary in the early 1930's. Von Meduna observed in his clinical practise that mentally ill patients suddenly lose their symptoms when they get a spontaneous convulsion. He further observed that the coincidence of epilepsy and schizophrenia in the same patient was rare. These observations led him to speculate and hypothesise that convulsions and mental illness are antagonistic and that the elicitation of the former could treat the latter. On the basis of these hypotheses, he investigated the effectiveness of camphor and its product, Metrazol in treating schizophrenia. Metrazol convulsions produced striking improvement in some patients (Arnot, 1975; Freedman et al, 1976).

Schizophrenics were the target patient population for convulsive therapies until the indiscriminate use of ECT with various psychiatric conditions established that the treatments actually have a favourable effect on affective disorders (Ilaria and Prange, 1975; Freedman et al, 1976). ECT literature (e.g. d'Elia, 1970; Fink, 1974; Ottonson, 1974; Ilaria and Prange, 1975; Strömgren and Juul-Jensen, 1975) indicates that the prominent therapeutic effect lies in the antidepressive action of the treatment while disturbance in memory is its principal side-effect. The impairment persists beyond the disappearance of other side-effects, for instance confusion, disorientation and impaired concentration (Janis, 1950). Memory disturbance following ECT is the area that is being investigated in the present study.

1.2 Implications of the investigation

ECT is a treatment at stake. Clinicians and also patients and their relatives have ambivalent feelings towards it because of its possible adverse effect on memory and other brain functions. Uncertainties about ECT effects are shown by the doctors' avoidance of the treatment and reluctance to give it prior consideration in the prescriptions. This is evident in the following statement by Freedman, Kaplan and Sadock (1976, p.506), authors of a well-known psychiatry textbook:

> many psychiatrists believe that ECT should be used with caution and only after other therapeutic approaches have been given trial.

A similar warning is given by Swift and Asuni (1975, p.195):

> unnecessary ECT should be avoided as each convulsion is thought to cause a small degree of brain damage.

The treatment is abolished in some countries e.g. China and the Soviet Union (Swift and Asuni, 1975).

Close examination reveal that the disfavour of ECT is often not based on its realistic appraisal but in the mistaken beliefs and prejudice held about it by both the public and doctors (Heshe and Roeder, 1976). Another important factor militating against its universal acceptance is the lack of sufficient knowledge regarding its cost-benefit. While the clinical efficacy of ECT has been demonstrated beyond

doubt, it is the cost of the treatment to one's bodily and psychological integrity that still need to be assessed to some degree of certainty. The cost value of the treatment should then be weighed against the economy and side-effects of chemotherapy and the discomfort, anguish or even danger of no treatment. The present research undertaken is seen as an essential and useful approach towards the goal of comparative cost-benefit evaluation of ECT with other treatments.

An investigation of the possible amnesic effects of ECT is also important in the understanding of brain-behaviour relationships. Knowledge of the relationship between the physical state of the brain and mental functions is of practical significance to a clinician first, because the activity of the brain is critically involved with the control of human behaviour and because the impairment of normal brain functioning underlies many forms of neuropsychological disorders and abnormal behaviour. Concerning the theme of brain-behaviour relationships, an investigation of the effects of ECT on memory should:

a) further our understanding on the involvement of ECT with crucial memory and other mental functions of the patient;

b) provide data for speculations and deducttions as to the mechanism of action of ECT;

c) clarify us about how memory is stored and organized; and

d) yield information regarding the neurological basis of memory storage and memory disruption processes.

Lastly, the research should have implications for remarks that ECT is undesirable, for the minimization of ECT amnesia and for means and ways of improving memory functioning after the treatment.

1.3 Research problem

Some progress has been made in the evaluation of the effects of ECT on memory. However, as far as can be ascertained from the review of literature, there is a lack of clinical research which varied and widened the learning-ECT interval to determine the effects of ECT on memory consolidation. Consolidation is a process responsible for the formation of permanent storage of information we acquire from our experiences and learning (Miller and Springer, 1973). Dornbush and Williams (1974, p.203) expose the problem as follows:

> It is surprising that so few studies have examined a range of learning-ECT intervals, particularly longer intervals.... In each of these studies only one learning-ECT interval was employed, or in cases where more than one interval was studied it was at unrealistically short durations.

Learning-ECT interval manipulation is the most relevant procedure in evaluating hypotheses on the effects of ECT on consolidation. The intervals can be lengthened to represent memory for remote events and shortened to represent memory for recent events. In this manner, it can be

1) determined what the effect of ECT is on information which is at different stages of consolidation;

2) verified if ECT-RA is a function of consolidation time;

3) ascertained how long prior to treatment ECT affects learning.

4) The varying of learning-ECT interval is the unconventional test procedure one can employ to account for persisting memory complaints in patients receiving bilateral ECT.

Theorists (John, 1967; Cermak, 1972) hypothesize consolidation to occur in short-term memory and to cease as soon as information is transformed to long-term memory. Interference with the consolidation process has been offered as an explanation for retrograde amnesia effects of ECT and other amnesic agents in man (Grossman, 1967; Dornbush and Williams, 1974; McGaugh, 1974). The hypothesis is being challenged by evidence that information which can be assumed to be consolidated or represented in long-term memory is also affected by ECT (Miller and Springer, 1973; Miller, 1978). As a result, Miller and Springer (1973) and Patterson et al (1978) advocate an alternative hypothesis of a retrieval defect in the ECT produced retrograde amnesia. However, the consolidation hypothesis has not been evaluated in

the light of research findings by Squire and his colleagues (1976), which suggest that consolidation continues in long-term memory and that ECT produces a consolidation-like defect in retrograde amnesia of long-term memory.

1.4 Hypotheses

It is hypothesized that ECT will affect information registered in memory according to how remote it is from the time of treatment; and that the consolidation hypothesis will be plausible to explain the nature of defect produced by ECT on information retention the latter which can be assumed to be represented by long-term memory.

1.5 Purpose of the investigation

The purpose of the research is to investigate

1) if ECT affects differently recent and remote long-term memory represented by ranges of learning-ECT intervals; and

2) if the consolidation hypothesis explains the nature of defect produced by ECT on acquired information which can be assumed to be represented in long-term memory.

Chapter 2
PREVIOUS INVESTIGATIONS

Research interest on the effects of ECT on memory was stimulated by the memory disturbance complication of this treatment. Clinicians observed that after a course of ECT treatments, patients fail to remember events, names, some aspects of personal life history and sequence of events which they recalled with ease before the treatment. This memory loss is called retrograde amnesia. Although there was an indication that ECT could cause anterograde amnesia, that is, the impairment of learning and retention power of events experienced after the seizure, most patient complaints represented the former type of amnesia (Dornbush and Williams, 1974).

Despite their speculations, clinical observers could not give answers to crucial questions about the following aspects of memory impairment produced by ECT:

a) the nature and extent of amnesia;

b) the desirability of the effect;

c) its neurological basis;

d) its relation to the treatment process and

e) reversibility.

The lack of information about the above-mentioned aspects caused many physicians to refrain from ECT in favour of drug treatment when the latter was introduced. This resulted in a sharp decrease in the use of ECT during the mid-1950's (Fink, 1974). It was however not possible to discard ECT completely because a growing number of patients who were believed to respond favourably to ECT showed poor improvement on drug treatment. Doctors were confronted with a conflict situation. They either had to use chemotherapy which appeared to be less effective but safer than ECT in treating psychiatric conditions such as depression or choose ECT whose mechanism of action and amnesia basis were not known for better results. The problem highlighted the need for information about the decisive amnesic effects of ECT outlined above. The impetus behind the wide scale experimentation and clinical research on the effects of ECT on memory which started in the late 1940's and which gained momentum in the 60's is thought to have been generated by the crisis.

Initial investigations were largely confined to the study of the effects of electroconvulsive shock (ECS) on learning and memory in animals (McGaugh and Williams, 1974). It can however not be inferred that results from laboratory animal studies are applicable to man. First and foremost, animal memory cannot be reconciled with human memory. Man's memory is by far superior and more intricate than that of the animal. This is shown by the fact that he can learn and retain large quantities of material. He has the ability to reproduce meaningless or difficult material which he memorizes, e.g. nonsense syllables. Furthermore, his

possession of the faculty of language gives him an advantage to alternative methods of memory testing such as recall and subjective reporting. Many manifestations of memory disturbance e.g. displacement, distortions and partial recollections can be detected from the description of man's experiences. It is also difficult to know what animals experience intrapsychically following the treatment with ECS because of absence of speech. Only their overt behavioural manifestations can be studied. The notable differences in the effects of ECS and ECT namely, that a single ECS in animals can produce gross memory disturbance which is unobservable in patients who receive as many as five ECT treatments (McGaugh and Williams, 1974) is another limitation in drawing conclusions from animal studies.

Although findings from animal research cannot always be reliably applied to man, it should not be ruled out that certain valid generalizations can be made. Animal experimentation is in fact absolutely essential because societal restrictions, humanitarian reasons and psychological codes of ethics limit drastic research undertakings on man. In fact the scientist can determine the involved risks in his experimental procedures and obviate them in man if he starts his investigations on animals.

Retrograde amnesia (RA) following ECT

Since the present study deals with retrograde amnesic effects of ECT it will be relevant if the review of previous research is confined to the theme treated.

That a series of ECT's lead to RA is a well-established fact. The earliest clinical study exposed by ECT literature to have produced evidence of RA is the one conducted by Mayer-Gross around 1942 (McGaugh, 1974). Numerous subsequent experimental studies by for example, Duncan, Gerard, Hunt, McGaugh, etc. (cited by McGaugh, 1974 and Deutsch, 1973) and clinical studies (e.g. Janis, 1950; Strain et al, 1968; d'Elia, 1970; Miller, 1970; Squire, 1975; and Squire et al, 1976) confirm the early clinical impressions and findings by Mayer-Gross that repeated ECT treatments cause RA.

Nature and extent of RA

RA and anterograde amnesia result from the alteration of normal brain activity. They are not only produced by ECT. Other treatments affecting the central nervous system functioning such as flurothyl (also called Indoklon Convulsive Therapy, ICT) (Cherkin, 1974) and lithium salts (Kropf and Müller-Oerlinghausen, 1979) as well as antibiotics (e.g. puromycin and actinomycin D) (Chapouthier, 1973) impair memory. The antibiotics affect memory by inhibition of protein synthesis and by a possible action on synaptic transmission (Deutsch, 1973; and Chapouthier, 1973). RA is also not uncommon in patients who suffer brain injury in consequence of accidents or diseases.

What is striking about RA resulting from ECT is that it is fragmentary. Its fragmentary nature is seen by the partial and scattered memory loss for names, people, events and some areas of experiential memory

(Valentine et al, 1968). Squire et al (1976) confirmed that ECT-RA is selectively produced by demonstrating that amnesia for temporal sequence of events is more severe than other aspects of memory, for example, the description of the events (cf. names of radio programmes). Sequential memory, however seems to be easily destructible, for patients with amnesic syndromes generally perform relatively poor in it (Squire et al, 1976). In other words, they find it more difficult to report precisely the time during which a past event occurred than say, to describe the event.

It is not clear why memory aspects are differently affected in ECT-RA. One possible explanation is that the retention quality of the different perspectives of things we learn is not the same. This is actually what Mandler and Ritchey (1977) have discovered in memory for pictures: the inventory of objects in the pictures were significantly longer retained than the descriptive information of the objects or memory of the overall spatial composition of the scene. It is important to investigate the influence of retention quality in ECT-RA because there is a possibility that memory for the sequence of events established by Squire and his associates to be severely disrupted by ECT is having low retention quality.

The affective quality of memory could be another reason for the selective effect of ECT on it. It has been revealed by Janis (1948; 1950; 1951) and Arnot (1975) that there is a relationship between the emotional quality of experiences and retrograde amnesia following ECT. Unpleasant emotional experiences that

evoked anxiety, tension and fear were found to be significantly less remembered after ECT than pleasant memories. Stieper et al (1951) also reported that ECT distrupts personal memories more than impersonal memories. It is presumed further, that emotionally important and familiar events may be better remembered after ECT than unfamiliar ones (Dornbush and Williams, 1974). In view of the findings by Holmes (1970) that forgetting of unpleasant personal experiences is related to a decrease in affective intensity of such experiences, it needs to be investigated whether the mode of action of ECT on memory is not to diminish the emotional intensity of memory experiences. It could also be considered if ECT is not ameliorating affective intensity of these disturbances. Since ECT is particularly effective with these disorders, the reduction of emotional intensity is a hypothesis of the mechanism of ECT action that could explain the psychological effect of ECT on both memory and psychiatric symptoms. It further needs to be researched whether there is no relationship between affective memory changes in altered brain function produced by ECT and brain damage, in the light of a report by Wechser (1973) that brain-injured patients are unable to recall effectively charged stories. This could gauge the extent of harm done by the treatment to the brain.

There is strong evidence that the learning-ECT interval determines the memory of experiences affected by ECT (d'Elia, 1970; Dornbush and Williams, 1974; Harper and Wiens, 1975). Events occurring shortly before the treatment suffer greater RA. Patients may

for example not recall what they were thinking or doing immediately prior to the treatment. Evidence for the memory loss for immediate events was also obtained in animal studies (cf. McGaugh, 1974). Behavioural performances acquired by laboratory animals just before ECS were severely disrupted. Arnot (1975) compares this memory loss to an erasure of a tape recording while Wingfield (1978) and Valentine et al (1968) relate it to RA seen after brain concussion. The idea expressed by these researchers is however the same, for in concussion the victim completely forgets immediate events leading up to the accident in which the head was injured. The explanation given to this phenomenon is that memory for events prior to the accident or ECT are interfered with before their permanence is established in the relatively permanent long-term memory.

It could not be established how far back in time memory is susceptible to the effects of ECT, until recently. By using objective tests that assess recent and remote memory, Squire and his associates (1975; 1976; 1977) found that RA can extend to events that occurred many years before the treatment. The results also revealed that conventional bilateral ECT (induced by placing electrodes bilaterally on the anterior temporal areas of the head) cause memory loss for remote events in addition to recent events. No measurable impairment in memory for remote events was observed in right unilateral ECT (that is, ECT applied from an electrode position on the non-dominant hemisphere of the brain). Squire et al studies as well as those of Heshe et al (1978) support previous

research findings (Cannicoct and Waggoner, 1967; Zinkin and Birtchnell, 1968; Sutherland et al, 1969; Castello et al, 1970; and d'Elia, 1970) that bilateral ECT produces greater RA than unilateral ECT. The EEG findings confirm that unilateral treatment has a gentler action on the brain than bilateral treatment (Strömgren and Juul-Jensen, 1975). It is assumed that bilateral ECT produces severe RA because it requires a higher threshold number of pulses (that is, a larger amount of electrical energy) to produce a tonic-clonic convulsion than unilateral ECT; and that there is a more widespread involvement of crucial brain structures in bilateral ECT (d'Elia, 1970; Weaver et al, 1978).

Since the unilateral treatment technique was introduced to experiment on the reduction of memory disturbance, its demonstrated minimal effects on memory led to its recommendation as the standard treatment. A further advantage of unilateral ECT is that one can avoid involvement of the treatment with crucial mental functions because the left and right hemispheres exclusively specialize in different mental processes. The left or dominant hemisphere in the right-handed man deals mainly with auditory and verbal functions and the right or nondominant hemisphere with visual and non-verbal functions (d'Elia, 1970; Robertson and Inglis, 1977). Thus, in a carpenter who earns a living by nonverbal proficiency, a unilateral dominant ECT may be preferable, despite the greater risk to his verbal functions. Possible damage to the speech area in a person who uses verbal skills in his occupation may be avoided by applying unilateral ECT on the non-dominant hemisphere. The claims that unilateral nondominant ECT

produce less amnesia than unilateral dominant ECT (e.g. Costello et al, 1970) are no longer acceptable for, mental functions in the nondominant hemisphere are impaired by the former unilateral mode of ECT as much as the dominant hemisphere localized functions by the latter (d'Elia, 1976; Robertson and Inglis, 1977, 1978). This is clearly expressed in the following quotation (Robertson and Inglis, 1977, p.302):

> it can no longer be taken for granted that unilateral nondominant ECT is altogether simply less damaging than dominant or bilateral ECT. It may well be the case that it selectively impairs, albeit temporarily, a less evident, but nevertheless important set of skills in a predominantly verbal organism.

Although unilateral ECT treatment is desirable from the viewpoint of less amnesic effects, its apparent inferior therapeutic results (Strömgren and Juul-Jensen, 1975; Strain et al, 1968) still makes the traditional bilateral ECT a treatment of choice among clinicians. According to Strain et al, unilateral ECT requires a large number of treatments and lengthened hospitalization to be as effective as bilateral ECT. Since there is a general agreement that the number and frequency of ECT treatments are positively related to the degree of RA (Fink, 1974), it should be assumed that the memory disturbance brought about by repeated unilateral ECT over a long period may be tantamount to the amnesic effects produced by bilateral ECT.

The mode of electrical conduction employed by some kinds of ECT machines which can be alternating, unidirectional or square-wave, etc., are also factors that influence the extent of RA. The

alternating current used by ECT apparatus like those designed by Cerletti and Bini deliver greater amounts of electrical energy to the brain. This is demonstrated by the rate and amplitude of EEG changes (Fink, 1974). By changing the wave form of the stimulating current, one is able to reduce the amount of energy. Unidirectional current (or modified sine wave) and pulsating currents that use very brief and frequent periods of peak voltage over the course of stimulation are believed to decrease the quantity of electrical force reaching the brain and RA (Weaver et al, 1978; Robertson and Inglis, 1977).

Permanence and reversibility of RA

The question of permanence or reversibility of ECT-RA has been and continues to be a matter causing concern because of persisting memory complaints by patients who are beyond assessment by conventional tests procedures.

In the studies of the deminishment of RA following ECT treatment, Janis (1949; 1950) detected residual memory impairments for names, events and aspects of the individual's life history. The memory loss continued to manifest itself after the transient organic reactions e.g. confusion, disorientation, etc., had disappeared. RA did not clear up completely during the follow-up assessment which was made three-and-half months after the completion of treatment.

Squire, Chace and Slater (1976) established

recently that the reversibility of ECT-RA varies according to type of memory. The patients recovered memory for names of past television programmes one to two weeks after a course of ECT. Their memory deficit for temporal order information remained unchanged three weeks after the last ECT. The researchers were still making a long-term follow-up study to determine how long after ECT the memory disturbance would remain. However, the findings of the research study by Squire and Chace published during the previous year, that is in 1975, suggested that memory substantially recovers six to nine months after ECT. There was no evidence of persisting memory impairments. Nevertheless, the subjects who had received bilateral ECT treatment frequently complained that their memory was not as good as before.

Because of continual complaints of memory loss by some patients, evidence for absence of residual memory impairment does not appear to be sufficient to contra-indicate perpetual memory disturbance after ECT. One concurs with Small (1974) and Squire and Chace (1975) that the standard test procedures might fail to detect the somewhat permanent memory impairment. It is therefore important to seek alternative or unconventional methods of memory assessment which may be sensitive for the undetected memory failure. Varying the learning-ECT interval appears to be one of the desirable procedures in the assessment of RA. Holmes' technique (1970) for measuring amnesia by asking subjects to keep a diary for everyday experiences is also a relevant testing procedure, particularly with the literate. Harper and Wiens (1975) recommends the technique for ECT research.

To conclude on the aspect of permanence of RA, one would accept the statement made by Barbizet (cited by Robertson and Inglis, 1977) on the conventional bilateral mode of ECT treatment that memory appears to be intact or to recover completely after a single ECT, but that its recovery becomes less and less satisfactorily as the series is prolonged.

The relationship of RA to the therapeutic effect of ECT

Two different views on the relation of RA with the treatment process of ECT can be distinguished. One view which is dualistic, considers RA as an undesirable side-effect of ECT which is independent or unrelated to the therapeutic action (e.g. Inglis, 1969; d'Elia, 1970; Ottonson, 1974). It supports the unrelatedness of the amnesic and therapeutic effects of ECT from evidence that:

1) patients who are effectively treated with ECT do not necessarily manifest RA, nor do patients who experience extreme memory deficits after ECT show significant recovery;

2) the effects occur via different mechanisms of action. The therapeutic effect is mainly determined by the seizure and memory impairment by the amount of electrical energy;

3) the durability of the therapeutic effect is unrelated to the duration of memory disturbance;

4) conditions producing memory disturbances such as anoxia and brain injury have no positive effect on the psychiatric disorder which responds most favourably to ECT, namely depression; and

5) that there is no memory disturbance from antidepressive drugs.

The other view holds that memory disturbance and the therapeutic effect are two inseparable aspects and that they are in some way positively correlated (e.g. Janis and Astrachan, 1951; Arnot, 1975; Zornetzer, 1974). This view which may be called unitary spells out that psychiatric symptoms, e.g. hallucinations, delusions and depression are eliminated by the amnesic effect of ECT and that patients who improve show forgetting of unpleasant past experiences which tend to evoke guilt, anxiety and lowered self-esteem. One of the advocates of this view, Arnot (1975), believes that ECT should be superior to psychotropic drugs which leave the unpleasant experiences related to the psychiatric disturbances intact. The shortcoming of this point of view is that it would not be unpleasant memory alone which is lost for neutral and desirable memories might be affected as well. It does not appear that ECT can be manipulated so as to circumscribe RA to unpleasant memories. In any case, failure to recall what one considers important could make one anxious, irrespective of the emotional quality of the material.

The relationship between memory disturbance and the curative effect of ECT is indeed debatable. Suffice

it to say that the production of RA is not the objective of the treatment but a secondary unpleasant effect. In actuality, RA qualifies to be called a side-effect because amnesia is generally not a desirable consequence of any treatment. The "forgetting of the past" encouraged in psychotherapy and remission of wrong-doings is in fact the reduction of anxiety associated with unpleasant memories but not their erasure. To treat people with painful memories by making them directly amnesic of their unhappy experiences sound to be impersonal and crude. The reality testing of those treated might be impaired by such a kind of therapeutic action and their problems may be aggravated.

It should be pointed out on the other hand, that available evidence is not sufficient yet to assert that RA and the therapeutic effect are coincidental and independent. As stated by Fink (1974, p.6) there may be an interaction between the mechanisms underlying the effects in a sense that "memory changes are derived from the same biochemical mechanisms which underlie the physiologic changes necessary for improvement in depressed patients". The role of the electrical current in RA and its therapeutic effect also makes their dichotomy invisible. RA is caused directly by the electrical current while the therapeutic effect is indirectly produced by the current via the convulsion. The effects are so bound up together by the therapeutic procedure that their mutual exclusion does not seem possible. One can therefore only minimize memory impairment in ECT but not avoid it. It might however be better for the patient to be able again to adapt to

life outside an institution with a little less memory of the past than to continue suffering with intact memory. In other words, ECT might be desirable and necessary if its cost can be minimal and its benefit maximal.

The neurophysiological basis of RA

Brain stimulation research using electrical current explored the neurophysiological basis of RA resulting from ECT. It investigated the changes in neural activity which are essential for producing ECT-RA, the brain sites in which the changes occur as well as the biochemical effects of ECT that determine RA (Zometzer, 1974).

That convulsions or seizures are related to the neural alterations underlying RA has been refuted by McGaugh and his research associates (1974). By means of ECS on laboratory animals they demonstrated that generalized brain seizure activity is not necessary or sufficient to yield RA. Subseizure stimulation, that is, the application of electrical current levels below tonic convulsion thresholds, could readily produce RA. Further, RA could be produced in animals that have been anesthetized with ether prior to the ECS treatment so that no seizures occur.

By investigating whether specific brain regions differ in thresholds for RA produced by electrical stimulation, it was discovered that some brain areas are more involved than others in the production of RA

(Zornetzer, 1974; McGaugh, 1974). The implicated structures are the amygdala caudate nucleus, hippocampus and medial thalamus. Low intensity electrical stimulation of these sites could result in RA. It was however, not established by the studies whether RA is due to alterations in the stimulated structures, or to effects produced elsewhere in the brain, for as pointed out by d'Elia (1970, p.57)

> memory deficit may not be directly attributable to the damaged structure itself but to secondary effects on some other structure.

Observations of memory impairment involving the temporal lobe where the implicated brain structures are affected by different pathological conditions that result from cerebral anoxia, monoxide poisoning, chronic alcoholism and surgical ablations, support the hypothesis that these structures are responsible for RA in ECT (d'Elia, 1970; Ellis et al, 1979). There is a qualitative similarity in RA following ECT and RA produced by the above-mentioned conditions. It should be pointed out however, that the findings do not suggest that the implicated brain regions in ECT-RA are the memory centres. Memory does not appear to be a product of any anatomically distinct structure or pathway, but a function of the integrity of the whole brain. What the research findings imply is that the brain structures and its systems may be critical for the maintenance of memory.

With regard to the biochemical basis for RA, recent studies (e.g. Essman, 1974; and Dunn et al, 1974) indicate that ECT reduces protein synthesis at the

synapses. It is believed that the inhibition of protein synthesis by ECT is achieved through its action on serotonin or some other biogenic amine. Structural changes in the synaptic membranes are effected which render the synapses temporarily nonfunctional. The inactivation of the synapses and the consequent depression in protein synthesis obstruct chemical events which operate in memory processes thereby bringing about amnesia.

Nature of the defects in ECT-RA

Although there is agreement that ECT might interfere with neurochemical and electrophysiological processes of the brain by disrupting the synthesis of macromolecules such as RNA or protein and by modifying the electrical activity of the brain (as shown by EEG changes; Small, 1974), there is no consensus yet on the nature of defect in ECT-RA. The ECT literature produces strong evidence for a defect in the consolidation of information or memory storage (McGaugh, 1974; Dornbush and Williams, 1974; Ellis et al, 1979). The hypothesis is however being challenged by Patterson et al (1978) and Miller and Springer (1973) who maintain that ECT causes a defect in retrieval of information from long-term memory.

Consolidation is a process responsible for the formation of permanent storage of information we acquire from our experiences and learning (Miller and Springer, 1973). It ensures that the memory trace endures until the permanent storage of information

occurs. A memory trace is a neural representation or a neurophysiological imprint in the brain of what we learn. The trace first appears as a short-term memory. During such a period, it can decay easily or be interfered with by other traces. Consolidation changes the memory trace into a state of permanence by converting it into long-term memory. Once consolidated, the information encoded in the trace is stored in the brain and can be available for retrieval. Retrieval is the "search process by which stored information is reactivated" (Fromholt et al, 1973, p.467). Information can be retrieved from storage by either direct recall or recognition.

The hypothesis of a consolidation defect in ECT-RA comes from observations of a greater RA for short training-ECS intervals and better retention when longer intervals are used in animal laboratory studies. Experimenters interpreted the longest training-ECS interval at which ECS was not producing significant amnesia to mean the end of the period necessary for completion of consolidation (McGaugh, 1974; Miller, 1979). By using this method, different consolidation times ranging from seconds to minutes and hours have been indicated (Dornbush and Williams, 1974). However, the findings of a study by Squire and his associates (1976) appear to dispute the limits of the consolidation period proposed by earlier studies on animals. The researchers state that the neural substrate of memory may change over the years to make the stored material difficult to disrupt. This sugests that the process of consolidation may continue for a longer time than initially expected.

It has been concluded by consolidation theorists that ECT and its counterpart ECS disrupts neural consolidation by destroying the memory trace before it is committed to long-term memory. However, clinical observations and research reports (cf. Squire, 1975; Janis, 1949; and Miller, 1978) reveal that information for remote events which can be assumed to be consolidated or represented in long-term memory is affected by ECT (or ECS) in addition to information which is not stored in long-term memory. This point is clearly expressed in the following excerpt from Miller (1978, p.119-120):

> ECS is capable of producing amnesia for information acquired 24 hours earlier, information that may reasonably be assumed to be well represented in passive storage by this time .... The results suggest that ECS is capable of acting upon information that is already in passive storage. Accepting this finding, we must look for a mechanism by which amnesic agents can act on information in passive storage in addition to whatever we believe ECS is doing to information that is uniquely in active storage.

A consolidation defect implies the destruction of the memory trace and the permanence of ECT-RA. The hypothesis is a valid interpretation for a memory disturbance which is not restored. What leads to the rejection of the consolidation hypothesis as a sole explanation of the defect in ECT-RA, is evidence that ECT or ECS also impairs memory traces which can be assumed to be represented in long-term memory; and that some memory lost during ECT can be spontaneously recovered or reinstated by means of reminder clues (Miller and Springer, 1973; Patterson, Lawler and Rochester, 1978). A defect in retrieval has been postulated as an alternative hypothesis to account for

supposes that the neural processes whose function is to reactivate adequately stored information, are impaired by ECT without interfering with memory storage.

It appears that a retrieval defect can be ascribed to what Reichert et al (1976) described as the prograde effects of ECT. The researchers state that the prograde effects are deficits in performance e.g. of memory tasks due to altered attentional and motivational processes following the treatment. Since there is reason to believe that lowered motivation, confusion and impaired concentration makes remembering difficult, the presence of these behavioural signs during retention testing should be assumed to impair retrieval. As soon as the prograde effects vanish, persisting memory loss can be regarded as impairment in storage or consolidation and natural decay of memory. The presence or absence of prograde effects (or organic reactions) is in other words a plausible criterion which might serve to reliably differentiate one defect from the other in ECT-RA.

Conclusion

To sum up, earlier investigations have made significant progress in the evaluation of the retrograde amnesic effects of ECT. However, not all facets of the problem have been explored and explained. There is paucity of research that sought to determine the effects of ECT on memory consolidation by employing the learning-ECT intervals. The nature of the defect in ECT-RA and the real relationship of RA to the therapeutic effects of ECT also remain to be demonstrated.

Chapter 3
EVALUATION OF ECT

Current status

Since the early 1940's, ECT has been the primary method for administering convulsive therapy. It outdated pharmaconvulsive therapies which were difficult to induce and regulate with regard to dosage. Although ECT was described as crude and shock-producing when it was still used without the general anaesthetic, its induction effects were nevertheless better tolerated by patients than the distress produced by convulsant drugs. The attainment of unconsciousness with drug convulsive therapy came after patients have suffered excruciating pain (Heshe et al, 1978).

ECT is widely used in Africa (Swift and Asuni, 1975). South Africa is no exception. It is also a common treatment modality in European countries such as Denmark (Heshe and Roeder, 1976) and Sweden (Frederiksen and d'Elia, 1979). Heshe and Roeder report that 22 210 ECT treatments were given in Denmark during the 1972-1973 fiscal year. In Sweden about 4 per cent admitted for psychiatric treatment receive ECT. An estimated 10 000 ECT treatments are given daily in the United States (Mulaik, 1979). The treatment is obsolete in the Soviet Union, China and some European countries (Swift and Asuni, 1975).

In a survey of ECT practise in countries where it is accepted, Asnis et al (1978) found that not all psychiatric hospitals prefer to use it. In some hospitals doctors declined to prescribe ECT because of inadequate skill in the technique of its application. It was found that in other hospitals, the treatment is disfavoured by hospital administrators particularly those with nonmedical background, for example clinical psychologists. The disuse of ECT was also attributed to the fewer number of admissions who deserved it.

Myths and criticisms and polemics on ECT arising both within and outside clinical circles explain why it is a centre of great controversy. It is alleged by critics that ECT is not very scientific because its mode of action is not well-known (Frederiksen and d'Elia, 1979). It is also claimed that it is noxious. Each time an ECT is given, some brain cells are believed to be destroyed. This notion has been stressed by Friedberg (1977) despite contrary evidence that organic side-effects are transient and reversible.

Another major criticism levelled against ECT is that it is antipsychotherapeutic. It is argued that it acts against psychotherapy by reinforcing repression of conflicts; by making the patient see his symptoms as an uncontrollable biological process; and by encouraging the patient to avoid considering the situational and experiential factors that contribute towards his mental disturbance. Amnesia of the content of psychotherapy sessions is believed to be produced and in some cases, patients are reported to have even failed to recognise the psychotherapist (Friedberg, 1977).

Mass media especially news-papers reveal that some members of the public construe ECT to be a behaviour therapy technique whose objective is to modify behaviour by producing shock. The inadvertent propagation of misnomers such as "electroshock", "shock" or "shock therapy" by mental health professionals is responsible for this conception (Frederiksen and d'Elia, 1979). It is also proposed by the Press that some segments of the society regard ECT as an infliction of torture on and perpetuation of ill-treatment of the mentally ill by acting out aggression on them.

Therapeutic efficacy and indications

Six controlled studies which conform to basic methodological requirements reviewed by Barton (1977) constitute overwhelming evidence that ECT is an effective treatment for most depressions. Although Costello et al (1970) has questioned its absolute efficacy, its comparative effectiveness with newer psychotropic drugs clearly demonstrates its superiority in relieving the depressive conditions. For example in a large study that consisted of 437 patients conducted by De Carolis (see Avery and Lubrano, 1979), ECT was significantly superior to imipramine (commonly known as Tofranil). The same applies to well-known American studies carried out by Greenblatt and his associates (1964; 1966) and the British study conducted under the auspices of the Medical Research Council (1965). Findings of these two major studies indicate that ECT produce faster amelioration of depression symptoms and

marked improvement than Tofranil, Nardil or phenelzine and placebo.

Best results from ECT occur in patients with endogeneous depression. The benefit for this condition approaches 100 per cent (Freedman et al, 1976; Ilaria and Prange, 1975; Greenblatt, 1977). Suicidal patients respond extremely well to ECT. The treatment is heralded as life-saving to such patients (Katz, 1974) and its immediate application to them is advised. ECT is also favourable to bipolar depression. Involutional melancholia which is not complicated by schizophrenic features also responds well to ECT. However in neurotic depressions, the results are less reliable. Neurotic patients who are likely to respond well are those who manifest true depressive episodes without colouring with schizoid behavioural patterns (Freedman et al, 1976).

A combination of ECT and drugs in the treatment of depression does not enhance the final outcome of treatment (Greenblatt, 1977). Antidepressant drugs are only advised after successful ECT to maintain the improved status and to reduce the incidence of relapse (Ilaria and Prange, 1975; Greenblatt, 1977).

In schizophrenia, ECT is effective when the illness has an acute onset and is of short duration. The presence of an identifiable precipitating event, the manifestation of major affective elements and a well-adjusted premorbid personality are regarded as favourable prognostic factors of ECT in schizophrenia. ECT is not very successful in chronic cases of schizophrenia such as the long-berm hospitalized

patients and those with repeated readmissions. Of the various subtypes of schizophrenia, catatonic excitement responds best followed by catatonic stupors. Acute paranoid schizophrenia in young patients is also reported to be responding favourably to ECT (Freedman et al, 1976; Greenblatt, 1977).

Regarding the effectiveness of ECT when compared with other treatments (e.g. chemotherapy and individual psychotherapy) in schizophrenia, May and his associates (1976) found that patients who received ECT stayed in the hospital for a shorter period than those with drug or psychotherapy alone.

There are too few studies to draw firm conclusions concerning the combinations of drugs with ECT in schizophrenia. There is suggestive evidence however that a combination of ECT and chlorpromazine (Largactil) may be better than chlorpromazine alone (Greenblatt, 1977).

Other conditions which respond to ECT are acute delirium, postpartum psychosis and toxic infectious psychosis. Patients who are epileptic and even mentally retarded sometimes respond to ECT. However, pharmacotherapy is recommended as a treatment of priority in these cases (Asnis et al, 1978; and Freedman et al, 1976).

Physical contra-indications to ECT are brain tumors, recent cerebrovascular accidents, advanced emphysema or other severe pulmonary pathological conditions, a recent fracture of one of the major bones and a major surgery

within the preceding month. In patients with cardiovascular complications, for example cardiac failure, cardiac arrthymias and severe hypertension, ECT is either used with caution or contra-indicated when the cardiovascular condition is not stabilized (Freedman et al, 1976; Asnis et al, 1978; Mulaik, 1979).

Physiological conditions such as pregnancy and age do not represent contra-indications to ECT. The treatment is not dangerous to the expectant mother or foetus, nor does it accelerate the termination of pregnancy. Both young and old persons can be treated with ECT (Freedman et al, 1976; Swift and Asuni, 1975).

Fatalities with ECT are very rare. The deaths that occur are reported to have a questionable relation to ECT (Heshe and Roeder, 1976) or to be chiefly caused by cardiac complications (Mulaik, 1979).

Technique and administration

General preparation of the patient for ECT include depriving him of food for at least 4 hours before the treatment to prevent regurgitation and aspiration of regurgitated material; emptying of bladder to prevent urination during the convulsion; loosening of any tight clothing; administration of atropine about one half hour before the treatment to decrease secretions and minimize the risk of aspiration of saliva into lungs; removal of dentures and use of mouth gags to protect teeth and tongue during the contraction of the jaw muscles.

In the standard bilateral ECT technique, the seizure is accomplished by placing electrodes bilaterally on the anterior temporal areas of the brain. A technique of anterior bifrontal ECT suggested by Inglis (1970) is also used. It has an advantage of reducing the severity of memory disturbance. Electrodes are placed over one cerebral hemisphere in unilateral ECT treatments. On areas where electrodes are placed skin resistance is reduced by applying a salt water solution. A modified procedure of ECT is then followed by first giving a short-acting intravenous anaesthetic to the patient. The anaesthetic alleviates apprehension and distress that accompany seizure induction. The preferred barbiturate anaesthetics are Methohexital (Brevital) and Thiopental. Propanidid, a short-acting nonbarbiturate is favoured by some clinicians because of the fewer complications it produces (Ilaria and Prange, 1975; Freedman et al, 1976). Premedication with barbiturates is avoided in patients with cardiac complications. Following the anaesthetic is the intravenous injection of a short-acting muscle relaxant, for example succinylcholine. Muscle-relaxant drugs decrease the strong muscular contractions during the convulsions. This modified ECT procedure has replaced straight or crude ECT, that is, the application of ECT without the benefit of premedication, in the form of the general anaesthetic and muscle relaxants. It obviates the shock and complication of fractures encountered in the latter method of ECT (Swift and Asuni, 1975).

The amount and duration of electrical current vary according to person, sex and his age. Female and old patients usually have a higher threshold than male

and young patients. The threshold may however be considerably raised by the barbiturate anaesthetic used in premedication. The electrical parameters are normally set for a voltage between 70 and 150V and for a duration that ranges from 0,1 to 1 second (Ilaria and Prange, 1975). Ilaria and Prange indicate that most patients convulse at a setting of 70 to 130V applied for 0,35 seconds. In the modified ECT, the seizure is induced after a lapse of about 45 seconds of premedication.

The original apparatus for supplying and delivering stimulus current built by Bini has undergone some modifications. Present apparatus use unidirectional current or square wave pulses. It is claimed that these currents reduce dosage to a considerable degree and that confusion and memory disturbance will be minimized as a result. Alternating current employed in the old apparatus imposed far more current on the brain tissue than was necessary for the production of a seizure (Heshe et al, 1978; Weaver et al, 1978).

Another modification found in the modern ECT apparatus is a device for allowing a gradual build-up of current intensity. This technique is called "glissando". With this method, a convulsion closely resembling that of epilepsy is elicited. The gradual peaking of the stimulus current favour muscular relaxation and lead to fewer skeletal complications. Glissando technique is valuable to the doctor if he wishes to keep the dosage of the muscle relaxant low (Ilaria and Prange, 1975).

The electrically induced seizure is clinically the same as the spontaneous grand mal seizure of epilepsy. The only difference is that in the induced seizure, the onset is more brisk and the tonic response more forceful. The tonic phase lasts for about 10sec. and is followed by the clonic phase which has a durability of 30 to 40sec. A nonanaesthetized patient may immediately start to convulse if a supramaximal stimulus is used.

Convulsive movements are only slight in modified ECT. After the cessation of movements, the patient remains in a state of apnea which is prolonged if muscle relaxants are used. The patient is bag-breathed with oxygen during this period until he resumes breathing. In nonanaesthetized patients, a deep noisy breathing starts at the termination of a convulsion. Profuse sweating and salivation also occur. Although consciousness is regained a few minutes after the treatment, the ECT patient remains in a clouded state for 15 to 30 minutes. As a result of this, he is kept in the treatment area for at least an hour. In addition to confusion and disorientation, after-effects in the form of headaches, nausea and pain in the jaw and neck muscles may be experienced.

ECT is given in a short course of four to eight treatments. It is rarely extended beyond twelve treatments (SAMJ, 1979). However, an enormous number of ECT treatments ranging between 50 and 100 were given to patients more than a decade ago. For example, it is reported by Bromberg (1975) that a certain patient once received as many as 120 ECT treatments. Treatments are

usually given on alternate days although daily treatments may be given for acutely disturbed patients. It is normally required that the patient receiving ECT be hospitalized. The treatment is rarely given on outpatient basis. One reason for this arrangement is to make the clinical observation of the patient possible while he is treated.

Biochemical changes following ECT and their relation to therapeutic and side-effects

ECT activates wide areas of the brain thereby causing many demonstrable biochemical changes (Kety, 1974; Ottonson, 1974). With repeated applications of ECT at optimal rates, the biochemical changes accumulate to produce persistent changes in the physical state and mental condition or behaviour of the patient. The produced changes can be associated with either the therapeutic or side-effects of the treatment. It is however, difficult to differentiate biochemical changes which are causally related to the therapeutic effect from those which are consequent to it (Kety, 1974).

Biogenic amines

There is an increase in the levels of biogenic amine neurotransmitters norepinephrine (Schildkraut and Draskoczy, 1974), serotonin or 5-Hydroxytryptamine (Valzelli and Garattini, 1974; Kety, 1974) and dopamine (Kety, 1974) following a series of electroconvulsive seizures. In some cases the change in the biogenic amine turnover is partially caused by the stress and

apprehension that precede the application of ECT. This is inferred from the release of the amines observed in rats which were only exposed to the stress of the shock situation (Kety, 1974). Besides this observation, the discharge of biogenic amines is generally not an uncommon finding in stressful situations.

The changes in norepinephrine and serotonin appear to occur in association with clinical remission of affective disorders. This is suggested by evidence that serotonin is deficient in the brains of both maniac and depressed patients and that norepinephrine is abundant in the central synapses of manic and hypomanic psychopathological conditions but deficient in the depressive states (Kety, 1974). The efficacy of the production of these biogenic amines in affective disorders is further suggested by evidence that the two major classes of antidepressant drugs, the monoamine oxidase inhibitors and the tricyclic antidepressants facilitate the actions of norepinephrine and serotonin in the brain (Freedman et al, 1976).

Alterations in serotonin metabolism produced by ECT appear to be therapeutic in schizophrenia in view of the hypothesis of faulty metabolism of the neurotransmitter in this condition (Schwartz, 1978). A rise in the levels of dopamine after a series of ECT could underly the improvement of schizophrenia since effective antischizophrenic drugs e.g. phenothiazines increase the brain levels of this biogenic amine by accelerating its formation (Freedman et al, 1976). Dopamine disturbance is strongly implicated in the etiology of schizophrenia (Levitt and Lonowski, 1975; Schwartz, 1978).

Brain enzymes

Effects of repeated electroconvulsive treatments lead to an increase in brain weight which is accompanied by an increase in the activity of the brain enzyme monoamine oxidase (MAO). The increase in MAO activity persists long after the completion of ECS series (Pryor, 1974). There is no change in other brain enzymes e.g. cholinesterase, acetylcholinesterase, succinic dehydrogenase and cathechol-0-methyl transferase (COMT).

MAO's function is to destroy dopamine and norepinephrine to aldehydes by deaminating them. The aldehydes are further converted by other enzymes to acids and alcohol. That ECT induces an increased activity of MAO is confirmed by the finding of an increase in deaminated metabolites of norepinephrine in the brain following repeated ECS (Schildkraut and Draskoczy, 1974). It is not clear if increased MAO activity is therapeutically relevant in schizophrenia, but a low MAO activity has been suggested in this psychiatric condition (Schwartz, 1978). What is well-established however is that the inhibition of MAO activity is desirable in depression because it results in the accumulation of norepinephrine (and other catecholamines) which stimulate the CNS and thus elevating the mood (Freedman et al, 1976; Krikstone and Levitt, 1975).

Acetylcholine

There is an increase in the levels of brain

acetylcholine during the seizure and an increase in the turnover of the neurotransmitter following a series of ECT (Karczmar, 1974). The changes in acetylcholine concentration indicate that the ECT seizures activate the cholinergic nervous system (i.e. the acetylcholine system). Since the acetylcholine system is coupled with synapses and systems operated by other transmitters, its stimulation by ECT activates these systems thereby causing multiple neurochemical changes particularly with regard to catecholamines and serotonin (Karczmar, 1974). The possible therapeutic value of these neurotransmitters has been considered. Karczmar points out that the neurotransmitter GABA is also affected in the activation of the acetylcholine system.

Mineral and water metabolism

The change in mineral and water metabolism observed in affective disorders returns to normal following treatment with ECT (Ottonson, 1974). However, it is not very clear if the restoration of the distribution of water and electrolytes is caused by the emotional reaction of the patients to ECT or the treatment itself because the changes have been observed in patients who were only premedicated (with atropine and intravenous barbiturate) for ECT. What can be stated however is that the changes in mineral and water metabolism represent more or less direct consequences of basic neurochemical changes underlying the therapeutic effect of ECT (Ottonson, 1974).

Cerobrovascular permeability

Increased cerebrovascular permeability lasting for several days occurs after ECT (Ottonson, 1974). This change has been detected with trypan blue and cocaine measures. It is believed to be caused by the production of carbon dioxide and other acid substances in the brain during the seizure. Both Fink (1974) and Ottonson (1974) indicate that the increase in cerebrovascular permeability results in altered levels of electrolytes and the appearance of neurotransmitters such as norepinephrine and acetylcholine in the intercellular fluids. The degree of increased cerebrovascular permeability produced by ECT is the same as that caused by antidepressant drugs (e.g. imipramine). Ottonson believes that the permeability changes are relevant for both the antidepressive effect and the memory disturbance side-effect of ECT.

Diencephalic influences

The variables regulated by the diencephalic nuclei such as body weight, appetite, sleep, digestion, circulation, libido and menstruation often change early in the course of ECT (Ottonson, 1974). ECT is believed to stimulate the diencephalic centres which control these autonomous and visceral functions. Since these variables show disturbance in affective disorders, their change following ECT is believed to underly the antidepressive effect of the treatment.

Protein synthesis

Dunn et al (1974) have shown that the seizure of the electroconvulsive shock inhibits protein synthesis. The depression in protein synthesis was evidenced by significant reduction in the incorporation of the amino acids into brain proteins of mice after ECS.

The mechanism that brings about the inhibition of protein sythesis is the fall in adenosine triphosphate (ATP) levels on which protein synthesis is absolutely dependent. The decrease in ATP is caused by anoxia since the change in ATP is not observed during convulsions when animals are oxygenated. Another factor that accounts for the decrease in protein synthesis is the changes in ion concentration due to massive depolarization of brain tissue by ECT (Dunn et al, 1974).

There is a significant correlation between the effects of ECT on protein synthesis and retrograde amnesia (RA) which suggests a causal relationship between the two phenomena (Kety, 1974; Dunn et al, 1974). The production of amnesia suggest that the effects of the ECT seizure are concentrated at the synapse where the crucial role of protein synthesis in memory appears to be exerted (Dunn et al, 1974).

Essman (1973; 1974) has demonstrated an ability of serotonin injected into the brain to cause memory disturbance. This finding raises the possibility that ECT-amnesia may be produced through its action on serotonin or another biogenic amine rather than directly upon protein.

Cerebral hypoxia

Excessive oxygen demands generated by the seizure causes cerebral hypoxia. In modified ECT, hypoxia is only local in brain areas such as the hippocampus which have a higher oxygen consumption than other parts. What reduces hypoxia in modified ECT is the minimization of muscular convulsions and the supply of oxygen throughout the seizures. Cerebral hypoxia is a side-effect of ECT and it is related to the memory disturbance which result from the treatment (Ottonson, 1974).

The mechanism of ECT action

The behaviour of an organism is directly the result of events that occur in the nervous system. Thus, by directly or indirectly altering the nervous system processes, ECT influences behaviour. The means by which the ECT seizure produces physiological and biochemical changes which underly persistent behavioural changes is mainly by altering synaptic transmission. Synapses are the target of ECT action for as pointed out by Dunn et al (1974) they are more profoundly affected by the brain seizures than any other parts of the brain. This is shown by the sudden release of many different neurotransmitters, for example acetylcholine, serotonin and the catecholamines norepinephrine and dopamine. These synaptic transmitters are not only released; their synthesis, storage, reuptake and degradation are also modified by alterations in neural firing produced by ECT. Furthermore, ECT alters impulse conduction within

the neuron by affecting membrane ionic permeabilities and cellular metabolism. Since the etiological theories of affective disorders, psychoses and schizophrenia reveal biochemical disturbance in the amount and metabolism of neurotransmitters in these psychiatric disorders, it is obvious that the modification of some neurotransmitters can bring about therapeutic effects.

The activation of brain structures such as the diencephalic nuclei in the brain stem by the seizure is another mode of therapeutic action of ECT. It has been stated that the stimulation of the diencephalic centres produce a gain in weight, an increased appetite, increased duration and amount of Stage 4 sleep and increased libido which characterize clinical remission of depression symptoms and improvement in behaviour (Ottonson, 1974). Arnot (1975) suggests that the physiological changes (e.g. increased appetite) and mood changes that result from the stimulation of brain areas such as the hypothalamus or the diencephalic centres come about in two ways. The changes are produced by both the passage of electrical current through the brain (ESB) and the response of the brain area to the crisis produced in the body by the seizure. This means that the mechanism of therapeutic action of ECT might not only occur via the seizures alone.

Psychological theories on the mode of action of ECT have been postulated. However, they have not been corroborated by research. They include repression, amnesia of unpleasant experiences which evoke anxiety, disruption of the ongoing brain circuits and in this

way interrupting the abnormal mental processes that give rise to morbid thinking and deviant behaviour, and the psychological reorganization of experience which takes place in the newly formed cerebral circuits (Janis, 1950; Fink, 1974; Arnot, 1975). The flaw in these theories is that they exclude the significant neurochemical events which play a central role in the therapeutic effect of ECT.

One agrees with Fink (1974) who states that neither the psychological nor the somatic theories of the action of repeated electrical seizures are tenable when considered separately. We need therefore to have combined biological and psychological theories in order to explain the mechanism of ECT action adequately. The neurophysiologic-adaptive hypothesis put forward by Fink (1974) is a step in the right direction. With this hypothesis, Fink attempts to interrelate neurophysiologic events of the seizure with the psychological organization of the patients. He postulates that ECT produces clinical improvement by changing the pathophysiology of the psychiatric disorders. He believes that in altering the pathophysiological states, ECT stimulate behavioural adaptations to modify the psychological aspects of the illness and to facilitate the reappearance of premorbid personality. What is questionable however is whether physiological and neurochemical changes produced by ECT can successfully alter the processing of real life experiences and organization of psychological functioning without supportive psychotherapy or psychological intervention in the patient's problems.

Conclusion

To close this chapter, one can say that the ECT technique has undergone few changes as compared to its original application. Its use is now only modified by premedication (with anaesthetics and muscle-relaxants) and electrode placement to minimize its complications, side-effects and distress to the patient. The treatment has definite useful indications for psychiatric disorders on which it can be used with good therapeutic results. The wide scale research on it has provided ample data to speculate and to make hypotheses and conclusions on its mechanism of action. The problem is however that its survival is threatened by ethical dilemmas, prejudice and lack of clarity on some of its clinical effects which need to be resolved by clinical conferences and not by way of research alone. There is no doubt that it served a valuable clinical purpose over a long period of time and it can continue to do so if more scientific data can be made available.

Chapter 4
MEMORY

Interest in the knowledge of memory dates back to the time of well-known philosophers, Plato and Aristotle. However, experimental investigations in memory were only begun in the nineteenth century by Ebbinghaus. His work laid a foundation to the scientific study of human memory. Today, memory is a focus of multiple disciplines such as physiology, biochemistry, neurology and psychiatry in addition to psychology. Numerous journals in these fields give special attention to this subject. Many facets of memory have already been explored and explained by the research conducted so far.

Memory can be described as the mental faculty which enables us to call facts or past experiences back to mind, and which allows our behaviour to be influenced by our past experience. In other words, memory makes the relationship between past and present experiences possible. Every form of behaviour has involvement with memory in a sense that what we realize, do, feel, think and dream about is mostly influenced by our past experience. We know who we are and that we exist because of our past experience. We reproduce mental images, re-experience past emotional experiences and recognize what we perceive from information stored in our memory. Without memory all our experiencing would be new. In a nutshell, it can be pointed out that

memory is essential for the psychological integrity of the individual.

What makes up our memory is the knowledge or skill we acquire through study, practise and experiences. Memory is in this way not mutually exclusive of learning. The arbitrary distinction between the two mental functions is that while learning is involved in the acquisition and storage of information, memory is concerned with its retention and retrieval.

Distinction between types of memories

Man's abstract thinking ability appears to be the limit in the number of distinctions that can be made between types of memories. Some of the important kinds of memories described by memory students (Richter, 1966; Brown, 1977; Harris, 1978) are the following:

a) Episodic memory is memory for specific events, dates and names. For example, if one posts a letter, the time, place and manner of postage of the letter is recorded in one's episodic memory.

b) Semantic memory contains a system of rules of the skills we learn. One's knowledge of the meanings of words and the rules for combining them into sentences represent this type of memory. Mental skill such as the solution of a square root is semantic memory.

c) Personal memory records feelings and ideas.

d) Image memory enables us to recall images of past events.

e) Physical skill or motor memory is memory for physical performances such as walking, dancing and driving.

f) Depending on the sensory modality involved one can talk of either visual, auditory, tactile or olfactory memory.

g) Verbal memory is the ability to remember the exact words of a statement. Nonverbal memory involves the retention of figures such as geometrical designs and other abstract symbols.

h) Rote memory and logical memory are distinguished according to the quality of retrieved material. The former refers to capacity for reproducing material in the exact form in which it was presented. The latter is memory for reproducing the gist or meaning of learned material without the original form.

i) Metamemory is knowledge of what memory is and ability to use prescribed methods to memorize the given tasks.

Distinction between short-term and long-term memory

Short-term memory is retention of items over a period of seconds (Baddeley and Warrington, 1970; Watkins, 1978). It contains information which the subject has not been allowed to think about, rehearse or organize. The capacity for short-term memory to hold information is very limited. All information we

can retain in short-term memory, that is without rehearsal, is about seven items. These items of information could be numbers, letters, words or events. Physiologically, short-term memory is a condition in which a neural pattern is activated (Cermak, 1972).

Long-term memory is retention of items over longer intervals for example minutes, hours, days or even weeks (Baddeley and Warrington, 1970; Atkinson and Shiffrin, 1976; Watkins, 1978). This memory involves some form of permanent structural change in the brain. It is characterized as stable, long-lasting and to be constituted by organized and well-rehearsed information.

Distinction between immediate, recent and remote memory

A rough distinction is made between immediate, recent and remote memory on the basis of the period which intervenes between acquisition of information and its retrieval from storage. These memories overlap because the time distinctions between them are relative and arbitrary. For example, a researcher might designate memory for material learnt minutes ago to be in the immediate past, a day ago to be recent, and retention of material learnt a week ago to be remote. However, in general immediate memory refers to events experienced moments ago that is, during the past seconds to few minutes. In this way, immediate memory includes short-term memory and it is often used to characterize this memory. If used to measure retention of rehearsed information, immediate memory can represent long-term memory. Both recent and remote

memory are forms of long-term memory which can be at different stages of consolidation (defined in Chapter 2), because the process of consolidation is a function of time (Squire, 1976; Gregg, 1975; Richter, 1966). Recent memory is for things learnt in the past ten minutes to hours and several days. Richter (1966) includes events that occurred weeks and a few months ago in recent memory. The retention of events that occurred a long while ago up to those experienced during one's childhood has been used to characterize remote memory.

The structure of the memory system

A number of theorists beginning with Plato, have proposed models on the structure of the human memory system. The essence of knowing the structure of memory is to understand the processes involved in the encoding, retention and retrieval of information. The encoding stage represents the acquisition and registration of information or the formation of a memory trace. Retention is the maintenance of the stored information. The storage or storing of information involves both the encoding and retention stages of memory. Retrieval is a means of rendering stored or retained information available when it is required. It is carried out by means of recall and recognition (Morris, 1978).

Plato based his model on wax writing tablets. He likened the act of writing on wax tablets to memory storage. The impressions on the tablets represented memory. How vivid the writing appeared, showed what and how much was remembered. The wax tablet hypothesis

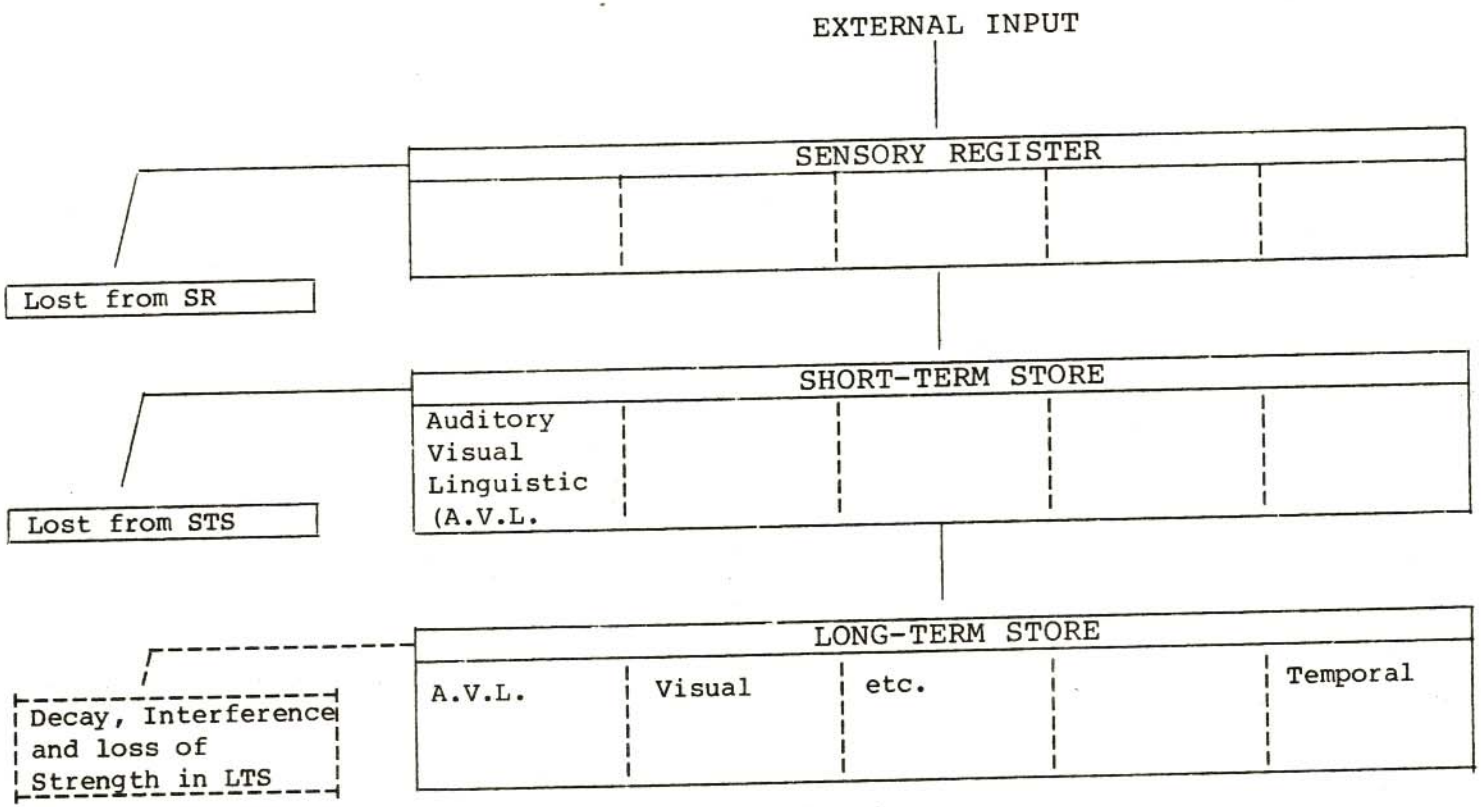
is simple in nature but it gives one an idea of the memory porcess.

The advent of machines such as the tape recorder and the computer made it possible to explain the memory structure clearly. Although these machines are far less complicated and dynamic than the human memory system, an analogy can nevertheless be made between their system of retention and that of man. The tape recorder has mechanisms for storage and retrieval processes. Storage is initiated by the microphone part which converts the sound waves in the air into patterns of magnetism. The patterns are recorded on the tape and stored over a period of time. Retrieval is enacted by a mechanism in the tape recorder which picks up the magnetic codes from the tape and reconvert them into corresponding sounds. Further, the recorder can demonstrate the decay and interference of information in the memory store. There may be a poor reproduction or none at all if there is a poor functioning of the recording or sound production components in the recorder. The shrinking of the tape and interference by some source of magnetism may also bring about a weak sound. Information fed into the computer is stored in the locations of the memory discs in the form of electric charges. Each location has an address. Suppose one location contains the balance of an account and a client wishes to know its contents, the address and instruction to retrieve the acquired information is given to the computer. When the contents are found, they are decoded from the memory codes and printed on a paper by the computer.

The generally accepted models of human memory

are those postulated by Norman, Broadbent and Atkinson and Shiffrin (Cermak, 1972). The models have similar structural properties. The only difference between them is in the terminology used for the components and certain hypotheses about processing of information in the memory stores. Atkinson and Shiffrin's model (Atkinson and Shiffrin, 1976; 1977) is chosen for description here because of its comprehensiveness and adoption in most textbooks. These authors see the memory system to be structurally divided into three stores called the sensory register (SR), the short-term store (STS) and the long-term store (LTS). The sensory register which is termed peripheral store by other theorists, e.g. Broadbent (Cermak, 1972) exists at the periphery of the perceptual system. There are two sensory registers. One, the iconic memory, registers visual images. The other sensory register, the echoic memory encodes auditory stimuli.

When physical stimuli in the form of light or sound impinge on the sense modalities of the individual, an impulse is immediately transmitted to the respective sensory register. Information decays in a matter of milliseconds in these stores unless it is passed into the short-term store. The STS is our "working memory". It has a limited capacity. Its contents are susceptible to disruption, but the time required for the information to be lost is relatively longer than for the sensory register. To keep information in STS and thereby build up registration in long-term storage, the individual is equipped with certain control processes. Atkinson and Shiffrin (1976, p.39) define control processes as "any scheme, coding technique or mnemonic used by the subject



Atkinson and Shiffrin's model of the memory system (1976).

in his effort to remember". Control processes work within and between each of the structural components of the memory system. They decide what information to attend to and to transmit to the next memory store. In the sensory register the control processes select particular portions of information for transfer to the short-term store and allow the rest to decay. Control processes in the STS determine what and how many items the subject should rehearse in order for their registration to take place in the long-term store.

The LTS has an infinite capacity to hold information. It is a relatively permanent store. It is subject more to interference than to decay. Interference is the loss of information in the memory trace due to incoming information or to confusion by competition among multiple traces. Control processes in the LTS are concerned with storage of information and the search for and retrieval of information from the stores. The search processes attempt to locate or find the sought for trace. If the trace is available, it is retrieved.

The concept of a memory trace

A memory trace is conceived by neuropsychologists (Malcolm, 1977; Bower, 1977) to be a neural imprint left by experience in the brain. It is in other words a neural representation of what we perceive or learn. The trace is continuously present in the person's brain as long as he remembers the experience or is influenced by it. It has a psycho-physical property because it

involves neural activity and the mental picture or image of the past experience. Further, it possesses a similar structure to what is remembered. It is however subject to disruption by natural decay, interferences by other traces and destruction by amnesic agents, for example ECT. When this happens amnesia may occur.

That a memory trace is still existing or available can be accurately determined by retrieval through recognition (Watkins, 1978). Recognition testing assesses whether the individual can identify what he has perceived before. If the subject fails to recognize an item which he registered, it indicates that the memory trace of that item is destroyed. Retrieval via recall assesses both the availability and the accessibility of registered information (Watkins, 1978). As a result when recall fails it is not clear whether the memory trace is available but inaccessible or unavailable altogether.

Phases of memory storage

a) Labile or consolidation phase

Experiments (John, 1967; Dornbush and Williams, 1974) on the formation of memory suggest that there is a labile period early in the registration of memory, during which information is susceptible to external interference. The phase extends over a period of time ranging from seconds to hours, days or even weeks. During this period, a labile representation of

experience gives way to a more stable long-term memory. The transformation of information from the labile phase into a stable one is known as consolidation. Physiologically, the process of consolidation characterizes the change of information encoded in the memory trace into a structural or chemical format. According to Hebb's theory (Cermak, 1972) what happens in the brain during the first part of this phase is neuronal reverberation. Cermak cites experimental evidence in favour of reverberatory physiological activity which lasts a few seconds to minutes during the consolidation phase. It is believed that the neuronal activity of reverberation causes an ionic shift in the neurons which produces a sustained change in the cytoplasmic concentration of a polypeptide, thereby bringing about long lasting and stable changes in the chemistry and reactivity of the neuron (John, 1967).

Evidence for the existence of a labile or consolidation phase come from observations of a temporal gradient of RA (that is, greater RA for recent experiences and less RA for remote events) following head injury, anaesthesia, ECS and ECT (Chapter 2). It was concluded that the occurrence of RA in these cases is due to interferences with ongoing brain activity of consolidation before memory traces are permanently fixated.

Mechanisms and locality of the consolidation process

A number of workers have tried to find the

anatomical locus of the consolidation process. Lesion studies carried out by Penfield and his associates (Cermak, 1972) originally located the mechanism of consolidation within the hippocampus. However, the bilateral removal of the hippocampus in animals failed to disrupt the process of consolidation. The observed behavioural deficits following such hippocampal ablations were described as impairments in discrimination, motivation, inability to alter behaviour, etc., rather than interference with consolidation (Cermak, 1972; Miller, 1978).

In some experiments, local electrical stimulation was used to localize brain regions involved in the consolidation phase. Glickman, Olds and Zuckerman (Cermak, 1972) demonstrated that electrical stimulation of any number of widespread cortical areas of the brain disrupts the consolidation process. The findings were like those found with lesions in that no one particular brain area was detected which is solely or principally necessary for consolidation to occur. ECS and ECT have not been useful in the localization of consolidation because of their generalized effects on the brain.

Another approach used in localizing consolidation has been to find brain areas which would facilitate or impede consolidation when stimulated or interfered with. Attempts to speed up the consolidation process have used low doses of CNS stimulants such as strychnine, picrotoxin and d-amphetamine. The drugs facilitate consolidation of memory when administered following learning (Dawson and McGaugh, 1972). These research workers point out that stimulant drugs might facilitate

consolidation by increasing the involvement of cells participating in memory storage and by changing the state of arousal of the organism. They state that the brain structures implicated in storage by CNS stimulants are intimately involved in arousal systems of the forebrain and brain stem. Strychnine and picrotoxin act mainly on the midbrain and medullary centres (Botha, 1978/1979). However, the different modes of action of a wide variety of CNS stimulants used in memory research makes it difficult to analyze brain sites and mechanisms involved in consolidation (Dawson and McGaugh, 1973). John's interpretation (1967) of the general effect of CNS stimulants as being an intensification of the reverberatory activity resulting in greater consolidation per unit of time and the accelerated rate of the chemical process mediating consolidation appears to be acceptable.

Deutsch (1973) and Ilyutchenok et al (1973) obtained evidence for the involvement of the cholinergic system in the formation of memory traces. According to Deutsch's observation, the postsynaptic membranes at cholinergic synapses increase their excitability and become more sensitive to the transmitter acetylcholine after learning. An increase in the amount of learning led to an increase in conductance at the cholinergic synapses. These results are evidence that synaptic conductance alters as a result of learning.

The cholinergic substrate which participates in mechanisms regulating the formation of memory traces has been found by Ilyutchenok et al (1973) to be located in the cerebral cortex, the amydaloid complex

of the limbic system and the ascending reticular activation system (ARAS). Ilyutchenok and his associates believe that the influence of these brain structures upon memory formation occur via increased reverberation in neuronal circuits and blockade of the retroactive interference of acquired information with subsequent sensory flow.

Pharmacological studies do not give convincing evidence for the involvement of RNA and protein synthesis in memory consolidation. Earlier findings by Agranoff, Barondes, Flexner and their associates that RNA and protein synthesis inhibitors such as the antibiotics actinomycin D, puromycin and cycloheximide disrupt memory consolidation, have been disputed by Nakajima (1973). He demonstrated the effects of actinomycin D and puromycin to be the development of debilitation and not the destruction of memory traces. Cyclohemixide produced conditioned aversion. These drugs did not appear to inhibit cerebral protein synthesis in the hippocampus and the limbic system. There has been no confirmation also of the claim that substances facilitating RNA and protein synthesis for example TCAP enhance consolidation (Nakajima and Essman, 1973; Rose and Haywood, 1977). Despite lack of evidence for the involvement of RNA and protein synthesis during the early phase of consolidation, it is believed that there is creation of additional proteins within some minutes of registration and that this new protein is a few minutes after registration critical to the retention of information (Booth, 1973). An alternative hypothesis put forward by Miller (1978) is that consolidation during the labile phase of memory might involve proteins or polypeptides manufactured prior to

learning. In this case, protein synthesis observed in the brain following learning (Rose and Haywood, 1977) might be a mechanism for stable or long-term storage.

b) The stable phase

During consolidation which may in fact last longer than the traditional time limits set by experimental studies (Squire et al, 1976; Morrel, 1969), information is transformed from the labile storage into a stable storage. In the stable storage information acquires a long-term quality. It becomes resistant to disruption and persists through sleep, unconsciousness and excitement for a good part of our lifetime. There are physiological and structural changes which are hypothesized to be the mechanisms for and basis of stable information storage.

Physiological and biochemical basis of stable memory storage

The hypothetical mechanisms for stable storage are the actual growth of the neuron and its synapses, the increase of synaptic transmission by change of the chemical state between synapses; and RNA and protein synthesis.

(i) Anatomical changes

Correlative studies of structural brain changes and memory reveal that there is increased neuronal

growth after training or experiencing of environmental complexity by animals (McGaugh, 1976; Brown, 1977). There is an increase in the number of dendrite growths and spines on the neuron, and in the size or thickness of the synapse. The increase in brain weight observed after learning or stimulation is thought to be the result of neuronal growth. Memory has however not been demonstrated to be a direct consequence of these anatomical changes (McGaugh, 1976).

(ii) Alterations in synaptic conductance

Changes in synaptic transmission during the stable phase are implicated by cholinergic, serotonergic and adrenergic activity. The involvement of cholinergic mechanisms in retention has been stressed by Ilyutchenok et al (1973). With regard to serotonergic activity, Essman (1973; 1974) found that the transmitter serotonin (5-hydroxytryptamine) interacts with brain protein sythesis in the processing of memory. Amnesia following ECS appeared to be produced by the inhibitory effect of the released 5-HT on cerebral protein sythesis. Results suggesting the operation of adrenergic transmitters in memory come from the studies by Cohen and Hamburg (Schwartz, 1978). Norepinephrine depletion or beta adrenergic blockade seemed to prevent the formation of long-term memory traces when administered within 3 hours after training.

(iii) DNA and RNA

That information can be stored chemically has been suggested by considerable evidence that hereditary information is stored in the DNA molecule (Brown, 1977). However, the claim that this molecule is also involved in the stable storage of information acquired through learning has been refuted by Vendreley and Vendreley (Cermak, 1972). New learning fails to change its composition. The molecule remains extremely stable throughout life. The only way in which it can be altered is through mutation. If learning or training could restructure or change the DNA molecule, it would be possible to influence the genetic make-up of offspring.

It has been possible to show by chemical methods that there are qualitative and quantitative changes in ribonucleic acid (RNA) contents of the brain in trained animals (Chapouthier, 1973; McGaugh, 1976). The changes were not observed in naive animals. The only difficulty in interpreting chemical changes associated with learning and memory is in distinguishing between the changes that occur after learning and the ones that occur after stimulation without learning.

The strongest evidence in favour of RNA as a molecular basis of memory to date is the results of McConnell and his colleagues (Nakajima and Essman, 1973; Chapouthier, 1973) who demonstrated retention of learning after regeneration and transfer of memory through cannibalism in planarians. Planarians are wonderful animals in that they are not only capable of

learning. They regenerate into two complete individuals of the same species when cut up into suitably sized pieces. Retention of learning in the regenerated planarians has been confirmed by other workers for example, Corning and John (Chapouthier, 1973; Nakajima and Essman, 1973). The experimental procedure followed by Corning and John has been to block the transfer of RNA from the tail section to the regenerated head. In this way the "head" planarian was allowed to retain RNA while the planarian that grew from the tail ("tail" planarian) was not. The former planarian was found to retain learning. No retention was evidenced in the latter.

Although it was not possible to replicate the other finding by McConnell that memory can be transferred cannibalistically, there is however well-established evidence for the transfer effects of RNA which are extracted from the brains of trained animals and injected intracerebrally into naive animals (Fjerdingstad, 1973). These findings suggest the possibility of memory transplantation.

(iv) Proteins

That changes in brain nucleoproteins might be the basis for memory has been postulated by Katz and Halstead as far back as 1950 (Richter, 1966). There is a high rate of protein synthesis in the brain. Qualitative and quantitative differences in the brain proteins of naive and trained animals have been shown (Booth, 1973; Chapouthier, 1973; Rose and Haywood, 1977). This finding indicates that learning or

formation of memory causes a change in brain proteins and that it produces amounts of newly synthesized proteins. Booth (1973) thinks that continued augmentation of protein synthesis could be necessary for the long-term stabilization of the memory traces. Protein metabolism is crucial for the synthesis of neural transmitters and it has been hypothesized that proteins could be involved in storage of learning by determining activation patterns in synapses and the formation of new synapses (Schwartz, 1978).

Ungar and his colleagues (1972) have made an important discovery of a "crude" protein involved in the stable information storage. It was found that during learning there is formation of peptides in the brains of trained animals when injected into them in sufficient doses. Fjerdingstad (1972) confirms this finding. The results strongly suggest that information provided by a learning experience is encoded into the molecular structure of the proteins or polypeptides. It is also indicated by the research finding that it is not the RNA substance itself which is responsible for the transfer effects of learning but the peptide molecules linked to it.

Anatomical localization of the stable memory storage

Memory seems to be diffusely represented throughout the brain for as pointed out by John (1967) and Dawson and McGaugh (1973), it appears that the memory trace spreads with time to encompass more of the brain and that more and more brain cells are recruited

into the storage process following a learning experience. Attempts to localize brain structures involved in memory storage are therefore not based on the assumption that memory is a localizable process but on the belief that there are "hot spots", that is, brain areas which are more critical than others in retention (Zornetzer, 1974).

The usual methods employed to localize the neurological circuitry closely involved in memory storage, have been to investigate brain-damaged patients, to make lesions in animal brains and to stimulate brain regions. Studies of brain lesions in man (Walker, 1956; Scoville and Milner, 1957; Milner, 1972; Penfield and Mathieson, 1974) strongly implicate the temporal lobe in memory functioning. The important structures identified in this lobe are the amygdala and the hippocampus. Electrical stimulation of the brain (ESB) research also point out the significance of these brain structures in memory (Zornetzer, 1974). Other specific brain sites implied by ESB are the caudate nucleus and the medial thalamus. The production of lesions in the brains of animals have largely yielded discrepant and contradictory results to that of man (Norman, 1973). For example, in a study of memory in monkeys it was found that memory deficits are more obvious in the lesions involving the frontal lobe cortex than the temporal lobe (Iverson, 1973).

Retrieval mechanism

The mechanism for the retrieval of memory traces

is of a cholinergic nature (Ilyutchenok et al, 1973). It was observed that changes in cholinergic activity affect the retrieval of memory traces. Retrieval failure resulted when animals were tested under the influence of scopolamine, an anticholinergic drug. Following an administration of anticholinesterase galanthamine, animals retrieved the memory trace of the learned response. There is also considerable empirical support that transmitter availability is a necessary condition for retrieval of memory (Dawson and McGaugh, 1973). These results indicate prospects for the facilitation of retrieval by means of pharmacological agents. Dawson and McGaugh raise the possibility of alleviating the amnesic effects of ECT by increasing the availability of CNS transmitter substances or their precursors

Concluding comment

Not everything is known about memory. Continued research by neuroscientists in the fields of biochemistry, physiology and psychology is very necessary to develop its scientific study to a point where we can clearly describe not only the mechanisms and material basis of learning and memory but the sequence of neural and chemical events which commence with functional changes for example neuronal reverberation, to structural modifications which characterize permanent memory storage.

Chapter 5
METHOD OF INVESTIGATION

5.1 Sample

The study was conducted on 40 hospitalized patients selected from the group diagnosed as schizophrenies by psychiatrists. One-half of these patients constituted the experimental group. They were prescribed ECT with the supplement of tranquilizers. The other 20 patients who represented the control group were treated with tranquilizers only.

Patients were selected by assessing them individually on the following criteria:

(a) Literacy They must have gone to school up to at least Standard II. With this minimum educational level, patients are expected to possess metamemory.

(b) Age Because of the age-relatedness of memory (Botwinick and Storandt, 1974), the age of patients included in the study was restricted to the minimum of 15 years and the maximum of 40 years. Old patients are avoided since they might have a decline in their memory potential. Children are excluded for they might only possess rudimentary metamemory capabilities.

(c) There should be no history of alcoholism, drug addiction, epilepsy or organic brain syndrome.

(d) Patients must be cooperative and manageable.

(e) They should be receptive that is, show interest or involvement in the memory tasks given and be alert.

(f) They should have ability to learn. They should be able to master the given tasks in the first three trials.

(g) They should score 100 per cent on the recall tests administered a few minutes after learning.

(h) They must not have received ECT during the past 6 months.

Table 1: DESCRIPTION OF SAMPLE

ECT Group		Control Group
Number of patients	20	20
Sex Male	11	14
Sex Female	9	6
Educational Std Distribution		
Range	Std2-Std8	Std2-Std8
Mean	5,30	5,65
Standard deviation	1,20	1,24
Age Distribution		
Range	15-39	17-36
Mean	26,45	25,30
Standard deviation	6,14	4,43

Schizophrenic patients were used as a study group because they were the diagnostic group in majority. Further, they comprised a large number of those receiving ECT. Also, the method of investigation suited these patients for they learn readily as compared to depressed patients to whom ECT is specifically indicated. By confining oneself to the psychodiagnostic group of schizophrenia, it could be possible to satisfy the essential condition of the homogeneity of the population.

5.2 Independent Variable: ECT

The patients in the experimental group received six doses of bilateral ECT from an Ectonustim model apparatus which is a combined ECT device and cerebro-stimulator. As in many hospitals, the conventional bilateral ECT is the only mode of ECT that was used. Forceps type electrodes were fitted on the anterior temporal areas of the scalp. An instant impulse of the duration of 3 seconds was induced at the voltage output of 220. The stimulus produced a grand mal seizure in each patient. At least half an hour before the application of ECT, patients were premedicated with atropine 0,6 mg. This was followed by Brietal Sodium anaesthetic and muscle relaxant Scoline which were administered intravenously a few moments before the elicitation of seizures. During anaesthesia, patients were artificially oxygenated until the resumption of spontaneous breathing. Patients received ECT treatment three times per week on alternate days that is, Mondays, Wednesdays and Fridays for two weeks.

5.3 Criterion Variable: Tests (see appendices)

In view of the unsuitability of the conventional memory tests for the objectives of the research, tests were developed. The test materials were mainly prepared to measure long-term memory in semi-literate subjects and which can be consolidated with ease. To facilitate consolidation the following measures were taken:

(a) the number of learnt items were limited to five. Research (Cermak, 1972) reveals that we can retain items of information, be it words, letters, numbers or events in memory with ease when they do not exceed seven at a time;

(b) familiar objects and events were used where applicable;

(c) a rehearsal encoding technique which facilitates both the acquisition and consolidation of information was employed. According to Norman (1973), this technique requires the subject to deliberately repeat the material contained in short-term memory so that information is transferred to a more permanent storage system. In the study, the rehearsal technique was applied by asking the patients to recite the items they learn.

(d) the sensory modalities of vision, audition and touch were used by letting the patients listen to the names of objects they see and by instructing them to feel the objects.

Wooden Models Test (WMT)

This test is intended to measure long-term recall and recognition. It consists of 10 sets of wooden models namely chairs, keys, guitars, stars, fish, hats, cars, bottles and the alphabet letters C and L. Each set for example, the set of chairs has three models of chairs having the same size but different shapes. After preliminary testing it was ad hoc accepted that the models can be easily learnt and remembered because of their concrete nature, familiarity and involvement with all sensory modalities. During learning, the subject is shown one model from each set. Shortly after learning he is supposed to recall the types of models he has seen to prove that he has memorized them. Following ECT, the subjects are again tested for recall of the models. To test recognition, the subject is shown the model he saw amongst two other models of the same set which they were never shown before. The subject is expected to recognize from the shape the design he has previously seen.

Ten Face Test (TFT)

The TFT has been used because of the general finding that humans have excellent memory for pictures and faces (Paivio, 1969; Sampson, 1970). For example, Bahrick et al (1975) have shown that people can recognize pictures of high school classmates whom they have not seen for many years. It has also been demonstrated by Nickerson (1968), Shepard (1967) and

Standing et al (1970) that even when the subjects are briefly exposed to a large number of pictures, recognition rates tend to remain very high for several days. Goldstein and Chace (1970) have also shown that pictorial stimuli have an unusually large storage and retrieval capacity.

The purpose of the TFT is to measure recognition and paired-association. The test is composed of front views of 10 unknown passport size human faces, half of which are males and the other half females. During learning, the subject is shown 10 faces and told the name of the person whose face is being seen. The subject is supposed to demonstrate that he is retaining the faces and their names by recalling the name of each face minutes after learning. Recognition is tested after the completion of ECT by asking the subjects to choose the faces they saw amongst 10 other new faces which have the same number of male-female distinctions. To measure paired-associate memory, the subject is reminded about the names of all the faces and then asked to match each name with a face or vice versa.

Personal Information Questionnaire (PIQ)

The PIQ has been developed by Janis (1950) to assess the effects of ECT on personal memories of their subjects. To suit the patients of this clinical study, the questionnaire was adapted by substituting, modifying and excluding some questions. The purpose of the questionnaire was to establish rapport with the patients and to determine the effects of ECT on

episodic memory of the patients' life history events. The PIQ was therefore not intended to evaluate the hypotheses of the study. The questionnaire was administered to each patient individually during the selection interview and after a course of ECT. The responses were recorded verbatim. During the post-treatment interview, it was assessed if the patient is able to recall personal memories elicited in the pre-ECT interview.

5.4 Experimental design and procedure

Two ranges of learning-ECT intervals are used. The long learning-ECT interval is about 55 hours while the short learning-ECT interval is about 3½ hours. The intervals are employed to determine the effects of ECT on consolidation of remotely and recently learnt material respectively. For the purpose of this study, it was assumed that the material learnt during the two intervals is represented in long-term memory when ECT was commenced because the learnt items were rehearsed by recitation and registered at least 3 hours prior to treatment. However, the long-term memory in the short learning-ECT interval was recent and having a shorter period of consolidation (since consolidation is a function of time), as compared to long-term memory in the long learning-ECT interval. The ten wooden models of the WMT and the ten faces of the TFT were split into halves to form parallel tests. Patients were therefore shown five items of each test (that is, half the number of items in the original tests) during each learning-ECT interval.

Table 2: OUTLINE OF THE EXPERIMENTAL DESIGN

	Long Learning-ECT interval	Short Learning-ECT interval
Experimental Group	$X_1, X_2, X_3, \ldots, X_{20}$	$X_1, X_2, X_3, \ldots, X_{20}$
Control Group	$Y_1, Y_2, Y_3, \ldots, Y_{20}$	$Y_1, Y_2, Y_3, \ldots, Y_{20}$

Soon after learning, recall of the items seen was called for. This was to make certain that patients have registered the items of information in their memory. Recognition tests were only administered following ECT. Evaluations of the retroactive effects of ECT on memory were made a week after the conclusion of ECT series. According to Heshe et al (1978) this ECT-testing interval is optimal because most of the acute effects of ECT and the anaesthetic can be presumed to have disappeared. Since remembering depends partly on the extent to which the environment prevailing during the acquisition also prevails when retrieval is demanded (Metcalfe, 1966), the same environment was used for learning and retention testing.

Scoring of the tests

(a) WMT: Recall Patients were asked to recall the 10 types of wooden models they saw. A score was given for each type of model which the patient recalled. The score was assigned to the appropriate learning-ECT interval during which the model was shown to the patient.

WMT: Recognition After recall of the wooden models, the patients were asked to choose the model which they selected from amongst two new models. Each recognized model was given a score in the respective learning-ECT interval.

(b) TFT: Recognition A score was entered in the appropriate learning-ECT interval for each face which was correctly recognized from a total of 20 faces.

TFT: Paired-association A score was given for the face correctly matched with the name.

(c) PIQ Instead of remembering, forgetting was scored in the questionnaire. Patients obtained zero if they remembered all the details elicited before ECT. A minus score was given if a patient failed to recall a certain detail which he remembered before, for example, the name of a grandparent.

5.5 Statistical Analysis

The effect of ECT on consolidation of recently and remotely learnt material was determined by testing the significance of difference between the means of retention scores of items learnt during the two ranges of learning-ECT intervals with the t-test for two related samples (Roscoe, 1969):

$$t = \frac{\bar{D}}{S_{\bar{D}}} \text{ where } \bar{D} = M_2 - M_1 \text{ and } S_{\bar{D}} = \sqrt{\frac{\Sigma (D-\bar{D})^2}{N(N-1)}}$$

The t-test for two independent samples (Roscoe, 1969) was used to test the significance of difference between the means of retention scores in the experimental group and the control group:

$$t = \frac{M_x - M_y}{Sm_x - Sm_y} \text{ , where } Sm_x - m_y = \sqrt{\frac{SS_x + SS_y}{n_1 + n_2 - 2}\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}$$

The control group served as the baseline. It represented forgetting or natural decay of memory. In addition to normal forgetting, memory loss in the experimental group was attributed to the amnesic effects of ECT.

Chapter 6

RESULTS, DISCUSSION, IMPLICATIONS AND RECOMMENDATIONS OF THE RESEARCH FINDINGS

1 Results

Table 3 shows that patients receiving ECT recalled items of the WMT presented during the long learning-ECT interval significantly better than items presented for the short learning-ECT interval ($t = 4,4, df = 19, p<0,001$), in this way producing a temporal gradient of RA that is, less RA for remote events and severe RA for recent events as seen in Fig.1. There was no significant difference between the means of WMT items recalled from the two learning-ECT intervals in the control group. Recognition of the WMT items (Table 3, Fig.2) presented during the long learning-ECT interval was also significantly greater than the WMT items learnt during the short learning-ECT interval ($t = 2,87, df = 19, p<0,01$). No significant difference between the means of recognized WMT items was found in the two learning-ECT intervals of the control group.

There is no evidence of a temporal gradient of ECT produced RA from the TFT items. Means of the recognition of faces and name-face pairing from the two ranges of learning-ECT intervals were not significantly different (Table 3).

As seen on Table 4 the means of recall, recognition and paired-association remained significantly above those of the experimental group for all the tests. The significance of mean differences between the two groups indicate that ECT had a disruptive effect on memory.

Changes in the number of details remembered following ECT were significant ($t = 3,92$, $df = 18, p<0,01$) for five life-history questions of the PIQ (Table 5 and Table 6). Other questions of the PIQ required descriptive answers and were as a result not quantifiable.

Observations

Some ECT patients revealed post-treatment confusion, agitation and restlessness a week after ECT when retention testing was supposed to be done. Patients who were agitated were not cooperative. As a result, one had to delay testing with a number of days to let them calm down first. The side-effects were slight two weeks after the last ECT except in one female patient who was evaluated to be confused^{+++} following the treatment. All untestable patients were cooperative during this time although some predicted that they will not remember what they saw.

Uncertainty was evident in the responses given by ECT patients. Despite their doubts or uncertainly, their choice of items in the recognition were often correct. Patients completely denied having seen certain items of the tests. This was not observed in the control group and can be regarded as evidence for the destruction of memory traces in the ECT group.

The ECT treated patients were slow in reacting to stimuli and retarded in their speech. They were also less productive in their responses to the PIQ after ECT than before. However, their responses to questions were more relevant. ECT patients were further observed to rationalize their memory loss. For example one patient said she can't remember the name of her sister because she has passed away.

2 Discussion

The results indicate that ECT produces a more severe disruptive effect on recent experiences than remote experiences. Findings support those of other clinical and experimental studies (McGaugh, 1974; Zornetzer, 1974; Miller, 1970; and Squire et al, 1976) which demonstrated that ECT produces a temporal gradient of RA. Since it has been found that immediate events experienced just prior to ECT are almost totally erased (Valentine et al, 1968; Arnot, 1975), it can be maintained that ECT affects memory progressively less and less from immediate and recent experiences to remote experiences. Alternatively stated, ECT produces a time-dependent retroactive effect on memory. On the basis of these observations, it can be concluded that there is process which is turning older memories into a state which is less vulnerable to disruption by ECT, or which increases the resistance of memory to disruption with time. This process is consistent with the hypothesized consolidation action which transforms memory traces into a state of permanence and stability. However, the process of consolidation which is

established in the study appears to continue even in memory which can be assumed to be represented in long-term memory, in this way confirming the following postulation made by Morrel (1969, p.285): "it is not only the electrical or short-term aspects of memory which consolidate; some form of consolidation must also occur in the structural or 'permanent' stage of information storage". The finding by Squire et al (1976) that the neural substrate of memory may change over the years so that material in memory becomes more resistant to disruption is strongly suggested by the present data to mean the continuation of the consolidation process in long-term memory.

From the foregoing discussion, it can be deduced that memory traces for recent experiences were severely disrupted because the consolidation process did not strengthen the experiences enough to withstand the amnesic effects of ECT. A consolidation defect in ECT-RA is therefore implied by the findings. The validity of this hypothesis is confirmed by recognition failure which shows that memory traces were destroyed or rendered unavailable by ECT. A defect in consolidation in the produced RA is also suggested by anterior bitemporal electrode placement in the ECT treated patients. There is well-established evidence that the temporal lobe on which electrodes were placed, is the critical site for memory functioning in man (Walker, 1956; Scoville and Milner, 1957; Milner, 1972; Zornetzer, 1974). By applying ECT on this area, the critical structures and systems within the brain which must function properly following the acquisition of information by the individuals can be interfered with, in this way producing a defect in consolidation.

Retrieval defect in the RA is not ruled out by the findings because of the presence of other side-effects, such as confusion during retention testing when retrieval was demanded. Recall failures after ECT could also imply a difficulty to get access to some intact memory traces (that is a defect in retrieval) rather than their unavailability.

The uncertainty with which ECT patients recognized learnt items and their failure to recognize items which they recall with ease confirms the finding by Williams (1973) that bilateral ECT impairs the sense of familiarity. This is borne out by the fact that although patients show uncertainty in their responses or appear as if they are taking a guess, their choice of items is often correct. The disturbance appears to be related to jamais vu that is, "a feeling of unfamiliarity with a real situation which one has experienced" (Freedman et al, 1976, p.311). This indicates that one's sense of reality of things and the world could become temporarily impaired by ECT. Whether this effect can be therapeutic is not known, but it is decidedly a side-effect which should be taken note of. Retardation in thought processes and decreased productivity observed following ECT are like amnesia and confusion; also the signs of organic brain impairment produced by the treatment. The side-effects have been reported in a study by Janis and Astrachan (1951), but the conditions are believed to be reversible.

The magnitude of the produced RA and the long duration of side-effects such as confusion, agitation and restlessness observed in some patients are

consistent with the findings of the severe disturbing effects of the conventional bilateral ECT (d'Elia, 1970; Squire, 1977; Heshe et al, 1978). The pronunciation of the produced side-effects could also imply that quantities of ECT current (intensity and duration) were higher.

Conclusion

The purpose of the study was to determine if the consolidation theory explain the nature of defect produced by ECT on acquired information which was rehearsed in order to be represented in long-term memory. The theory was evaluated by varying the learning-ECT intervals to represent recent and remote long-term memory. It was hypothesized that retained information will be affected by ECT according to how remote it is from the treatment; and that the consolidation hypothesis will be plausible to explain the nature of defect produced in ECT-RA of long-term memory. The results support the hypotheses. Recent information was found to be more severely affected than remote acquired information. The consolidation hypothesis appears to be a valid interpretation for ECT-RA. However, a retrieval defect in RA was also implied by the results.

3 Implications and recommendations of the research findings

1 It is suggested that procedures already recommended by other research workers to

minimize the side-effects of amnesia, confusion, etc. be followed by:

(a) applying the technique of anterior bifrontal ECT (ABF/ECT) suggested by Inglis (1970). It has been found by Abrams and Taylor (Ilaria and Prange, 1975) that this technique reduce the disruptive effect of ECT on learning and memory without changing the clinical effectiveness.

(b) using only an electrical dosage sufficient to produce a seizure. Supraliminal stimulation that is, the use of greater electrical dosages than necessary for the production of seizures, produce more severe memory disturbances but no improvement in the treatment effect (Heshe et al, 1978). Ilaria and Prange (1975) recommend that stimulus current be delivered at a voltage between 70 and 150V for a varying duration between 0,1 and 1 second. They state however, that on average most patients will convulse at a setting of 70 to 130V applied for 0,35 seconds.

(c) considering the technique of unilateral ECT which was introduced by Lancaster, Steinert and Frost (Robertson and Inglis, 1977) to mild psychiatrically disturbed patients. Research (d'Elia, 1970; Zinkin and Birtchnell, 1968) reveals that retrograde amnesia is less pronounced after a generalized seizure induced by unilateral stimulation than when the stimulation is bilateral. The quantity of electrical energy reaching the brain tissue is

believed to be lower in unilateral ECT than bilateral ECT (d'Elia, 1970; Strömgrem and Juul-Jensen, 1975). Ilaria and Prange think that unilateral ECT has less amnesic effects because it produces only a unilateral dysfunction of the medial brain structures involved in memory consolidation. Unilateral ECT has almost replaced bilateral ECT in Sweden and Denmark (Asnis et al, 1978). For the severe mentally disturbed, bilateral ECT is still considered as a favourable treatment because of the apparent superior clinical outcome of this mode of ECT to unilateral ECT (Strain et al, 1968).

2 A psychodynamic study of the patients who improve or fail to improve on ECT treatment is necessary to establish more precise exclusive and inclusive criteria for the indications of the treatment and to maximize its benefit.

3 In addition to psychotropic drugs, ECT needs to be supplemented by rehabilitative and supportive psychotherapy measures after completion of the course. It is believed that patients can be fit to begin psychotherapy two weeks after the completion of the treatment.

4 The use of modified ECT that is, ECT under general anaesthetic (G.A.) should be encouraged as it was observed to obviate the distress commonly seen when straight or crude ECT is applied.

5 Patients referred to ECT as "shock" or "electricity". These misnomers should be discouraged because they are not consistent with the objective of the treatment. The use of these archaic terms also scare patients and their relatives.

6 The extent of the side-effects of ECT needs to be assessed to some degree of certainty to evaluate the cost-benefit of the treatment objectively.

7 It is recommended that research be done on the use of pharmacological agents to reduce the effects of ECT on memory. An attempt has already been made in this direction. Small et al (1977) investigaged whether the polypeptide ACTH 4-10 exerted anti-amnesic effects. Observations after a single ECT were suggestive of some positive effects, but studies between seizures after 5 or 6 ECTs showed no significant anti-amnesic effects. The polypeptide ACTH 4-10 is a nontoxic agent which facilitates memory, attention and cognitive functioning.

SUMMARY

The study investigated the retroactive effects of the traditional bilateral ECT on learning and memory aspects, particularly the consolidation of information. The subjects who participated in the study were selected from the hospitalized patient population diagnosed as schizophrenies. An experimental procedure of ranging the learning-ECT interval to represent memory for remote and recent events was followed. Remote memory was represented by a learning-ECT interval of just more than two days. A few hours learning-ECT interval represented memory for learning during the two intervals. The results show an adverse destructive effect for material learnt in the short learning-ECT interval. Findings indicate that defects occur in both consolidation and retrieval of information in the ECT produced retrograde amnesia. A defect in consolidation is suggested by severe disruptive effect of recent experiences and recognition difficulties. The latter is evidence for the destruction, or alternatively the unavailability of memory traces of what has been learnt. Retrieval defect could be implied by recall failures and the presence of side effects such as confusion during retention testing. The data is consistent with the findings of a temporal gradient in the ECT produced retrograde amnesia and the effects of bilateral ECT. Recommended procedures to minimize the side-effects of amnesia and confusion such as the use of anterior bifrontal electrode placement, a small electrical dosage

sufficient to produce a seizure, and unilateral ECT are suggested. The use of a modified ECT has been found to be desirable because it obviated the distressing effects of the treatment on the patients. There is necessity for a psychodynamic study of the patients who receive ECT to establish more precise exclusive and inclusive criteria of the treatment in order to maximize its benefit. Further investigations of the extent of its costs are required to evaluate its cost-benefit objectively.

OPSOMMING

Die studie ondersoek die retroaktiewe uitwerking van die tradisioneel-bilaterale ESB op aspekte van leer en geheue, veral met verwysing tot die konsolidasie van inligting. Die proefgroep het bestaan uit geselekteerde pasiënte wat as skisofrene gediagnoseer is. Die eksperimentele prosedure wat gevolg is, het betrekking op die manipulasie van die leer-ESB-pouse om geheue vir afgeleë en resente gebeurtenisse te ondersoek. Afgeleë geheue is verteenwoordig deur 'n leer-ESB interval van meer as twee dae, terwyl geheue van resente gebeurtenisse deur 'n paar ure leer-ESB-pouse verteenwoordig is. Gelyksoortige leertake is gedurende die twee pouses aangebied. Die resultate dui op 'n ernstige steurende uitwerking op die stof wat gedurende die kort leer-ESB-pouse geleer is. Beskadiging van beide die konsolidasie en herwinning van informasie in die geproduseerde retivaktiewe amnesie word gesuggereer. 'n Gebrek aan konsolidasie is verder aangedui deur herkenningstekortkominge wat 'n aanduiding gee van die vernietiging of alternatiewelik die onbeskikbaarheid van geheuespore van wat geleer is. Herwinningsgebrek is geïmpliseer deur herroepingsgebreke en die teenwoordigheid van toevallige uitwerkings soos verwarring en onrus gedurende geheue toetsing. Die data dui op 'n temporale gradiënt in die ESB-geproduseerde retroaktiewe amnesie, en ook dat bilaterale ESB ernstige beskadiging meebring. Aanbevole prosedures om toevallige uitwerkings van amnesie en verwarring te minimaliseer, soos byvoorbeeld die gebruik

van anterieur bifrontaal elektrodeplasing, unilaterale ESB-behandeling en klein elektriese dosisse genoegsaam om konvulsies te presipiteer, word aanbeveel. Die gebruik van gewysigde ESB-tegnieke is wenslik om die ernstig beskadigende uitwerking van die behandeling te voorkom. Dit is ook noodsaaklik dat 'n psigo-dinamiese studie van pasiënte wat ESB ontvang gedoen word om sodoende meer presiese insluitende en uitsluitende kriteria vir aanduidings van die behandeling te ontwikkel. Meer navorsings oor die omvang van toevallige uitwerkings is nodig. Ten slotte is dit ook nodig dat 'n intensiewe studie onderneem word om die werklike koste-voordeel van die behandeling te bepaal.

APPENDICES

Table 3: DIFFERENCE IN MEAN RETENTION SCORES BETWEEN THE TWO RANGES OF LEARNING-ECT INTERVALS

TESTS	VARIABLE	LONG LEARNING-ECT INTERVAL	SHORT LEARNING-ECT INTERVAL	s	t 19 df	p
Recalled WMT items	Control Group	3,15	3,00	0,30	0,49	
	ECT Group	1,20	0,40	0,18	4,4	0,001
Recognized WMT items	Control Group	3,70	3,85	0,23	0,22	
	ECT Group	2,30	1,35	0,33	2,88	0,01
Recognized Faces	Control Group	3,40	3,30	0,29	0,67	
	ECT Group	1,70	1,50	0,31	0,64	
Name-Face Matching	Control Group	1,70	1,80	0,26	0,38	
	ECT Group	0,40	0,35	0,17	0,29	

Table 4: DIFFERENCE IN MEAN RETENTION SCORES BETWEEN THE CONTROL GROUP AND THE EXPERIMENTAL GROUP

TESTS	LEARNING-ECT INTERVAL RANGE	CONTROL GROUP $\bar{X}$	ECT GROUP $\bar{Y}$	s	t 18 df	p
Recalled WMT items	Long	3,15	1,20	0,37	5,28	0,001
	Short	3,00	0,40	0,41	6,34	0,001
Recognized WMT items	Long	3,70	2,30	0,43	3,25	0,01
	Short	3,85	1,35	0,49	5,10	0,001
Recognized Faces	Long	3,40	1,70	0,61	2,79	0,02
	Short	3,30	1,50	0,59	3,05	0,01
Name-Face Matching	Long	1,70	0,40	0,44	2,95	0,01
	Short	1,80	0,35	0,47	3,09	0,01

TEMPORAL GRADIENTS OF RETROGRADE AMNESIA

Fig 1

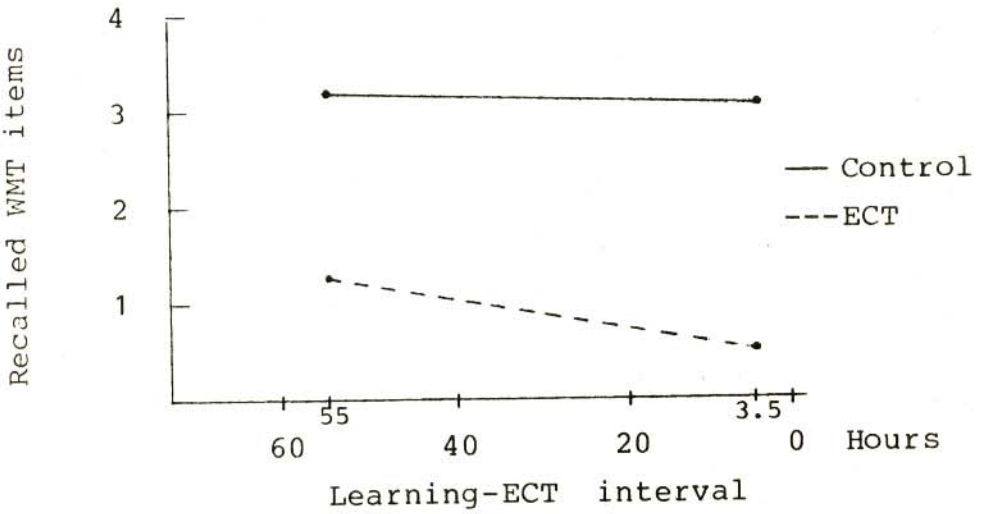


Fig 2

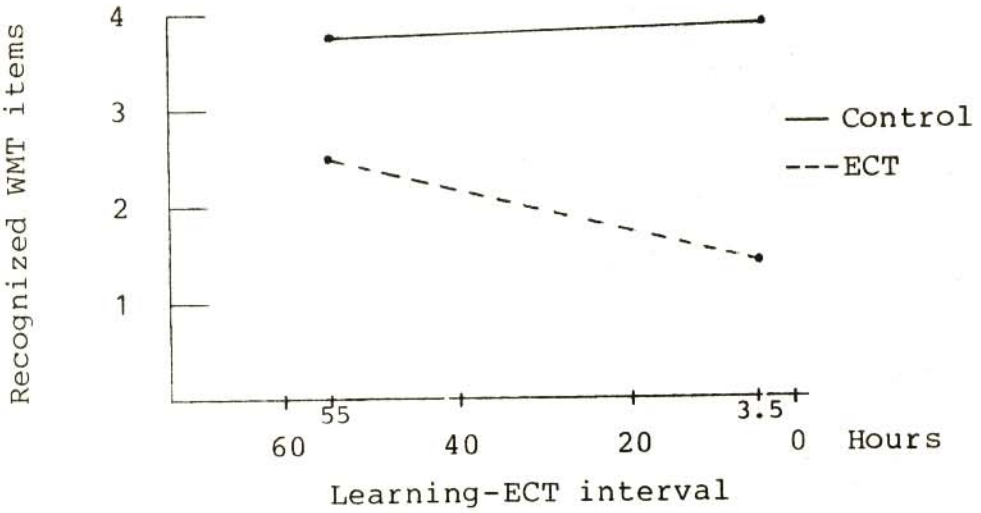


Table 5: CHANGES IN THE NUMBER OF DETAILS REMEMBERED FOLLOWING ECT FOR FIVE LIFE-HISTORY QUESTIONS OF THE PIQ

X ECT Group		Y Control Group	
Patient	Change	Patient	Change
1	-6	1	0
2	-4	2	-2
3	-6	3	0
4	-1	4	-1
5	-5	5	0
6	-8	6	0
7	0	7	-3
8	-3	8	-1
9	-2	9	0
10	-6	10	0
11	-7	11	0
12	-2	12	-1
13	-4	13	0
14	-9	14	-4
15	-1	15	-2
16	-4	16	0
17	-6	17	-1
18	-8	18	0
19	-3	19	0
20	-5	20	-2
N=20	$\bar{X}$=4,5	N=20	$\bar{Y}$=0,85

t=3,92 significant at 0,01 level

Table 6: FIVE LIFE-HISTORY QUESTIONS OF THE PIQ

11. Tell me the names of your brothers and sisters and their respective ages.

12. Tell me the names of your parents and grandparents.

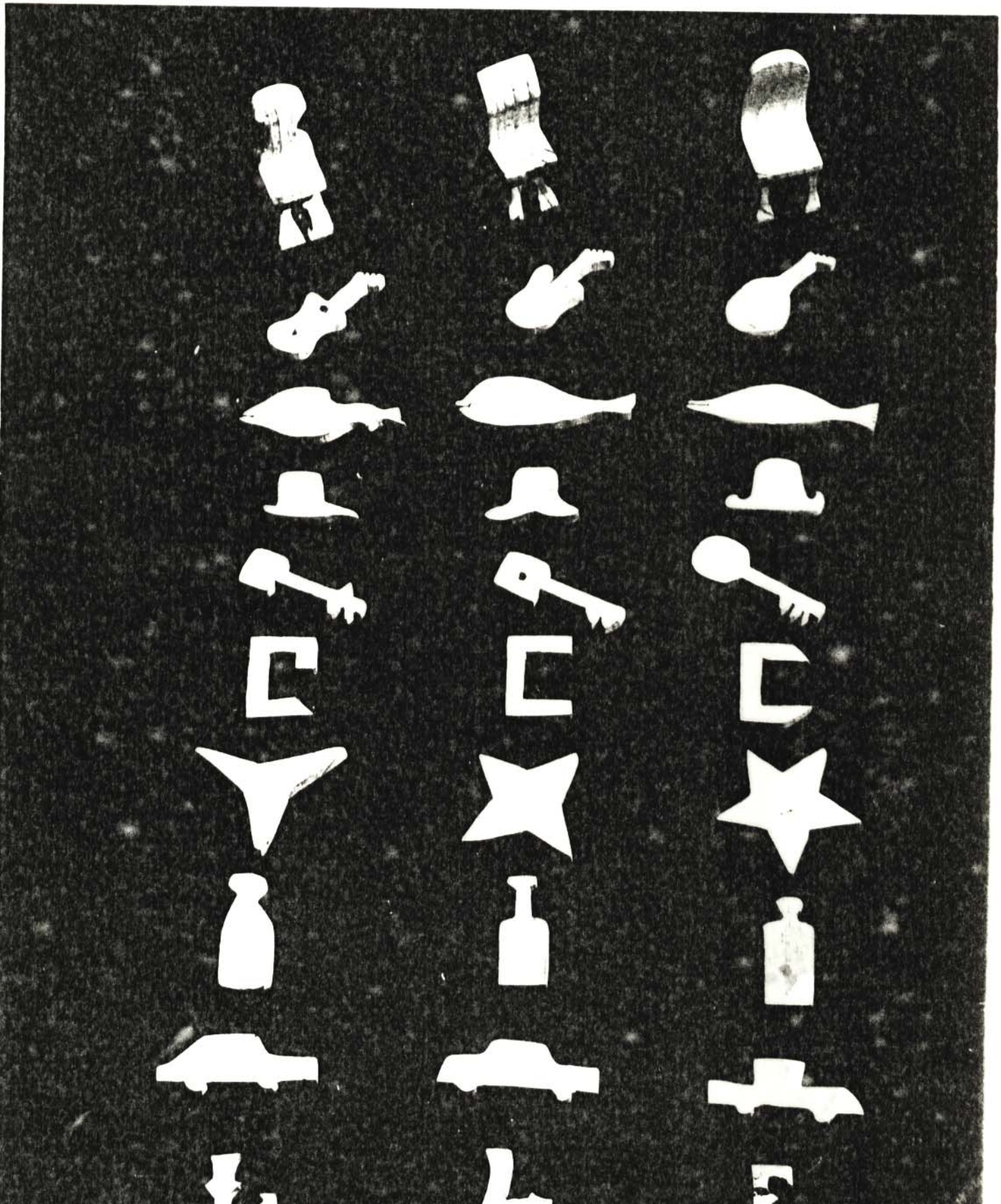
13. Tell me the names of dogs your family once owned including the ones they still keep.

14. Tell me the names of church denominations with erected church buildings in your village.

15. Tell me the name of the school you last attended including the names of the teachers and the headmaster.

PERSONAL INFORMATION QUESTIONNAIRE (PIQ)

1. What circumstances led to your coming to hospital?
2. How did the problem/illness start?
3. What does your family feel about it?
4. Where did you go for help before you came here?
5. What help did they give you?
6. Tell me of the worst experience you won't forget in your lifetime.
7. Everybody has his happiest moment in life. Tell me about the memorable happy occasion in your lifetime.
8. Tell me what you often do on Sundays.
9. Recall the last Christmas day and tell me how you experienced the occasion.
10. Describe how you picture yourself - what sort of person would you say you are?
11. Tell me the names of your brothers and sisters and their respective ages.
12. Tell me the names of your parents and grand-parents.
13. Tell me the names of dogs your family once owned including the ones they still keep.
14. Tell me the names of church denominations with erected church buildings in your village.
15. Tell me the name of the school you last attended including the names of teachers and the headmaster.



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