A BEHAVIOURAL APPROACH TO THE REHABILITATION OF SEVERE BRAIN INJURY

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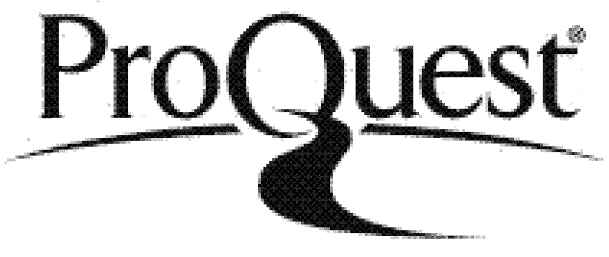

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ABSTRACT

The opportunity for the research leading to this thesis arose following the opening of a brain injury rehabilitation unit at St Andrew's Hospital in 1979. This Unit was (and probably is) quite unique in its character because it provides a comprehensive brain injury rehabilitation programme within the structure of a token economy system, utilising a variety of behaviour management techniques.

The idea for such a unit was conceived by Dr Peter Eames, MRCP,MRCPsych, Consultant Neuropsychiatrist at St Andrew's Hospital, Northampton. For some time, he had been aware of the lack of treatment facilities for patients who, after sustaining severe brain injury, usually as a result of head trauma, presented a disturbance of behaviour. When this occurred, the patient in question would often be denied clinical rehabilitation because the behaviour disturbance, frequently involving aggressive, sexual or some other provocative or threatening behaviour, would be regarded as unmanageable by staff working on conventional rehabilitation units. Dr Eames was disturbed by the fact that many of these patients were relegated to

psychiatric, geriatric or subnormality hospitals, often wihout any attempt at rehabilitation, simply because the appropriate clinical facility was not available.

The Kemsley Unit, in which most of this research took place, was designed to provide clinical rehabilitation for this patient population, utilising the skills of a multidisciplinary team and, in particular, a neuropsychiatric and neuropsychological perspective. In this respect, it answered the needs of brain injury rehabilitation discussed by Powell (1981), Goldstein and Ruthven (1981) and Brooks (1984).

The thesis is divided into five sections:-

Section 1 contains two chapters which introduce the problem of severe brain injury and its effect upon the individual in society. Chapter 1 deals with the problem of behaviour as it affects the family and rehabilitation services. It also takes a critical look at the way behaviour disorders have conventionally been described:- using psychiatric terms of reference, and argues that a more precise description can be made using a behavioural perspective. Chapter 2 examines in some detail the major types of behaviour disorder, suggesting a simple taxonomy for such disorders , with the aim of separating neurologically mediated behaviours from those for which some other mechanism is responsible.

Section 2 provides a description of the treatment environment and the patients (Chapter 3), and an argument

for single-case design methods in this type of research (Chapter 4). The reason for including Chapter 3 is the importance of understanding how this particular behaviour system works, prior to a presentation of the treatment programmes.

Section 3 contains four chapters which describe a behavioural approach to the management of different problems in brain injury rehabilitation. Chapter 5 deals with the management of aggression, using single case studies to illustrate the methods used and the outcome of treatment. Chapter 6 presents a group study of 30 patients which shows the effectiveness and generality of a time-out room procedure. Chapter 7 is directed at the management of sexual disorders and unacceptable habits while Chapter 8 demonstrates the application of these procedures in different rehabilitation activities.

Section 4 also has four chapters, each dealing with different aspects of attentional problems and their effect on behaviour and the re-learning of new responses. Chapter 9 provides an introduction while Chapter 10 presents a group study showing the involvement of attention in simple discrimination learning. Chapter 11 tries to identify which component of attention is most affected by brain injury while Chapter 12 presents various treatment methods which have been used to improve attentional aspects of behaviour.

Section 5 contains Chapter 13 which deals with an

important aspect of "personality change" following severe brain injury, in an attempt to determine its relationship to the injury.

Finally, Chapter 14 provides a discussion of the major findings and their implication for the development of brain injury rehabilitation.

When reading this thesis one must not lose sight of the fact that it descibes the problems presented by the most severe brain injuries, with the exception of those patients who remain in a persisting vegetating state and who, by implication, are not involved in rehabilitation. Although the relative number of patients with these problems is small (approximately 1000 each year), those affected are often young adults, who may have the misfortune of ending up in entirely unsuitable surroundings (geriatric or subnormality wards), simply because some form of suitable management has not been provided to control or eliminate their unacceptable behaviour.

SECTION 1

INTRODUCTION

CHAPTER 1

BEHAVIOUR DISORDERS FOLLOWING SEVERE BRAIN INJURY

Introduction

The effect of severe brain injury on behaviour and emotional adjustment is being increasingly recognised (London 1967; Bond 1975; Brooks and Aughton 1979; Lezak 1978; Wood and Eames 1981; McKinley et al 1981; Wood 1984a). Behaviour change as a result of head injury affects recovery in two major ways. In the first instance it affects the relationship between the head injured person and his family (Bond and Brooks 1976; Oddy et al 1978; Lezak 1978; Rosenbaum and Najenson 1976; Thomsen 1974; Panting and Merry 1972). Secondly, recovery may be prejudiced because such behaviour may reduce or even eliminate chances of rehabilitation (Miller and Cruzat 1981) and also prevent such individuals being accepted by the community, making some form of residential care necessary, often in chronic psychiatric wards.

Effects on Family

London (1967), commenting from clinical experience, stated that one of the most distressing effects of severe head injury was the alteration of the patient's personality, the burden of which usually falls on the family. This clinical observation was later confirmed by other studies (Panting and Merry 1972; Romano 1974;

Thomsen 1974,1984; Bond 1975; Lezak 1978; Oddy, Humphrey and Uttley 1978), making it apparent that the stress experienced by the family persists well after neurological recovery is complete and after the patient has returned to work.

A long term study of this problem by Weddell, Oddy and Jenkins (1980), using interview techniques and questionnaires to investigate emotional and behaviour changes in head injured patients, found that the behaviour disorder was not only extremely stressful to the family group but persisted for a length of time that was often quite disproportionate to the severity of the initial injury (established by length of post traumatic amnesia). They also found that the emotional changes described by the family did not always correspond to conventional psychiatric syndromes and would not fit into the formal taxonomy of psychiatry. This lack of a reliable psychiatric frame of reference suggests that psychological techniques, such as those measures of behaviour and personality described by Powell (1981a), may be of more value in providing a description of behaviour change after such injuries.

The persistence of behaviour disorders and their effect on the family was confirmed by McKinley et al (1981). They did a follow-up study, over 12 months, of 55 severely head injured patients and found an association between the level of stress felt by the family and the

mental and behavioural changes shown by the patient. There also appeared to be a difference in the time scale for the emergence of various behaviour characteristics. In the early recovery period slowness, tiredness, irritability and poor memory were mainly described; features of the well known post concussional syndrome. Other characteristics, such as bad temper, were not reported until later (12 months) in the recovery phase.

A study by Rosenbaum and Najeson (1976) concentrated on the effect that severe brain injury had on the **wives** of brain injured patients. They interviewed each subject one year after the injury had taken place, assuming this to be one of the most difficult periods for the wife because it is often the time for the "moment of truth", when the actual implications of the husband's disability have to be faced. Hopes for a complete recovery have diminished and the wife has to make major adjustments in her life and come to terms with the reality of living with a person who may be both physically disabled **and** changed in terms of temperament and emotionality. They compared the wives of brain injured patients with the wives of paraplegics. Both groups had sustained severe physical injuries but the paraplegics had not lost any of their intellectual ability and their basic temperament had not changed.

The results of this study showed that the wives of the brain injured patients were significantly more depressed

than the wives of paraplegics, also the interpersonal relationships with husbands and friends had been more affected in the brain injured group. The wives of the brain injured patients engaged in very few "pleasant" activities either at home or outside. Rosenbaum and Najeson offered a conclusion (made from a behavioural perspective) to explain the feelings of the wives. They stated that, "their (the wives) level of activities had increased, yet it was followed by a lower rate of positive reinforcement than before the accident". It was assumed that this lack of reinforcement was related to the depression reported by the wives.

Effect On Rehabilitation

The second effect of behaviour change concerns the patient's acceptability by rehabilitation specialists. Although little formal reference is made to this fact in academic journals, (although see Miller and Cruzat 1981), it is generally recognised by those professionals who work in rehabilitation medicine, that traumatically brain injured patients are not popular because of their often irritating, threatening and embarrassing behaviour, as well as their general lack of motivation. This is not normally a feature of orthopaedic patients for example, who are probably the other main recipients of rehabilitation. Miller and Cruzat (1981) suggested that one reason head injured patients were unpopular was

because of their poor concentration. Wood (1984b) supports this and cites a number of references which describe poor concentration in this group of patients, adding that poor **concentration** usually implies poor **cooperation.** Rehabilitation therapists feel a sense of frustration and poor job satisfaction in such circumstances.

DEFINING ORGANIC BEHAVIOUR DISORDERS

1. A Psychiatric Approach

Psychologists working with brain injured patients have tended to neglect behaviour disorders. This may be because such behaviour has tended to be defined in a rather nebulous or vague way as, for example, "personality change". This has been a favourite term of neurologists and psychiatrists describing patients involved in litigation after brain injury. In cases of less severe head injury a lack of a more precise description has led to the development of a jaundiced attitude on the part of many doctors, influenced by Miller's (1966) claim that such behaviour was part of a "compensational neurosis", if not frank malingering. This has been a very persistent view amongst members of the medical profession, even though evidence from **controlled** studies has been presented in opposition (Kelly 1981).

Even when behaviour problems have been more severe and obviously 'organic' in character, little has been

suggested or done to remedy the problem. One reason for this is the implicit assumption that such behaviour was an extension of some premorbid personality trait and therefore, essentially irreversible (Symonds 1937). Lishman (1977) however, describing the **neuro**-psychiatric tradition, appears to reduce the significance of pre-morbid characteristics. He suggests that this is one of the major ways in which "neuropsychiatry differs from ordinary psychiatry"..... "psychological disturbances resulting from brain pathology share a degree of common ground which allows them to cut across differences in background, personality and social situations". He felt that organic disorders have certain features in common which usually allow them to be distinguished from non-organic mental illness. Lishman did acknowledge however, the difficulties in assessing the origins of more complex "mental phenomena", especially changes in emotion, personality and other complex aspects of behaviour.

Bond (1984) when discussing the psychiatry of closed head injury takes a more cautious view. Citing the study by Jamieson and Kelly (1973) he points out that victims of head injury have premorbid personal and social characteristics which **"predispose"** them to injury. The most pertinent characteristics of later behaviour problems were, "a degree of immaturity of personality and behaviour". He concludes however, that there is no clear

evidence that any other specific characteristics are linked to a predisposition to injury.

A study by Oddy (1984) shows that evidence from follow-up studies on the social consequences of such injuries, which include the likelihood or speed of return to work, fails to support Symonds' idea. Another criticism is made by Powell (1981a) who feels that such clinical impressions are, "largely unsubstantiated by careful measures of behaviour or personality change". He feels that as a result of the medical "myth" (Kelly 1981) a clinical stereotype of the brain injured person appears to have been perpetuated, which Powell feels has obscured nearly all of the details of what must be a very complex relationship between brain and personality.

Powell refers to a study by Roberts (1976) to support this view. This study measured the sequelae of 359 head injuries but included only one "simple scale" as a measure of personality. From this Roberts derives what Powell calls "an orthodox stereotyped view", that the pattern of personality change is one of "frontal euphoria, disinhibition, or anergia associated with tense irritability". One can only agree with Powell in adopting a somewhat cynical attitude to such results which, given a large clinical group to study, fail to find evidence of several different clusters of personality types or behavioural abnormalities. This seems to indicate the need for a more detailed description of personality

characteristics following brain injury.

Psychiatric Disorders And Their Relation To Brain Injury

Bond and Brooks (1976) and Bond (1979) trace the development of physical, mental and social consequences following head injuries with different degrees of severity, in a way which Bond (1984) describes as their "natural history". He regards this time scale of events during recovery as being of some importance, both for determining an accurate prognosis and to anticipate likely changes that may affect rehabilitation.

Bond (1984) describes the different stages in recovery from brain injury when psychiatric disturbances emerge. The first stage may be observed as the patient comes out of coma. Disorientation for time, place and person are usually present as well as cognitive, perceptual, mood and behaviour changes. Psychiatric phenomena may include delusions, especially paranoid delusions, with hypomania and visual hallucinations also being briefly present.

The considerable **variation** in these symptoms is commented on by Lishman (1978) and also Bond (1984). The degree of such variation Lishman regards as depending upon, (1) the severity of injury, (2) the pre-traumatic personality (a contradiction on his earlier comment about neuropsychiatric diagnosis of other mental sequelae) and (3) the patient's present surroundings. Both Lishman and Bond accept that "acute post-traumatic psychoses" resolve

spontaneously during neurological recovery or respond quickly to phenothiazine medication.

It is not difficult, at this stage of recovery, to see how behaviour changes are largely due to cognitive abnormalities which affect clarity of thinking, stability of mood and consequently, behavioural adjustment. As such, these conditions emulate transient psychiatric disorders but, occurring as they do in the context of neurological disturbance, usually carry the label "organic confusional state".

There appear to be some problems however, defining psychiatric illnesses that appear later in recovery and continue over a long period of time. At this stage it is more difficult to describe **organic** behaviour change as something which can be directly attributed to the effects of brain injury. A lot depends on (1) the severity of the injury, and (2) the time of onset for the abnormal behaviour. The type of behaviour pattern which emerges seem to be less influential, although certain symptom patterns are obviously organic, e.g. the Capgras Syndrome.

Lishman (1968) attributes the majority of **chronic** organic behaviour disorders to diffuse and widespread damage of the brain. In a study of 670 cases with penetrating missile wounds, he found that 64% developed mild psychiatric problems and 21% more severe problems. Psychiatric disability was significantly related to two

separate but related factors, (1) the volume of brain tissue destroyed and (2) the depth of penetration.

Other studies have also reported that the **amount** of damage is a crucial variable in such cases. Flor-Henry (1969) found that the incidence of psychotic illness in a group of temporal lobe epileptics was as high as 79% if bilateral (structural) damage was in evidence. This is what Powell (1981b) refers to as the **"mass action principle"** or the **"damagedness hypothesis".** Powell also found that more severe psychiatric disturbances occurred when injuries to the left hemisphere were sustained. The study by Flor-Henry and another by Gregoriades, et al (1971) found that most patients with schizophrenia and paranoid disorders had focal brain damage in their dominant hemispheres. The opposite however, was the case for manic depressives; of the nine diagnosed as such in the Flor-Henry study, all had foci in the non-dominant hemisphere. Bond (1984), summarising evidence from head injury studies, points out that patients, whether schizophrenic, hypomanic or manic-depressive, all had premorbid personality characteristics which were the dominating features of their disturbed mental states. This does not support the findings of Achte et al (1967) and Achte et al (1969) who produced evidence that the incidence of schizophrenia after missile wounds of the brain is above the expected incidence of one percent for the general population. They quote 2.6% for psychoses

resembling schizophrenia but only 0.4% for frank schizophrenia. The subjects of these studies were soldiers, yet no attempt seems to have been made to control for the effects of pre-injury stress that might be implicated in the development of a psychotic illness.

Davidson and Bagley (1969) reviewed the evidence regarding the incidence of schizophrenic-like psychoses and brain injury and found it to be above the expectation for the population as a whole, concluding that **trauma** is a direct precipitating factor. They found that genetic and constitutional factors were less important than amongst patients who develop naturally occurring schizophrenia and that the psychoses were related to the severity of diffuse brain injury with evidence suggesting that damage to systems within the temporal lobes may well be the origin of the abnormal state.

Lishman (1978) reviewed the evidence for depressive psychoses after brain injury. He concluded that affective disorders occur after injuries of all severity and may be quite marked even after minor injuries. Achte et al (1967) reported that depression is more common than mania. Bond attributes this to a growing awareness of the physical, mental and social consequences of injury.

A study by Powell (1979) helped establish that the involvement between head injury and personality change was something more than purely a psychological reaction

to the effects of the injury. He compared a head injured group with an orthopaedic group and found a higher incidence of psychiatric abnormality in the former, even though both groups were comparable regarding emotional responses to injury and experienced similar post traumatic environmental problems.

Lishman (1973, 1978) appears less convinced however. From the results of a study on the chronic sequelae of head injury (1973) he suggests that although brain injury emerges as undeniably important, its contribution to psychiatric sequelae was little more than "one fifteenth part of the total causation of long term psychiatric disability". He felt that the greater part was due to a secondary reaction to the injury. He describes how the effects of intellectual impairment, environmental factors (such as domestic troubles, occupational difficulties, compensation problems) as well as the emotional impact and repercussions of the injury itself, together with premorbid personality and "mental constitution" are of profound importance when determining the nature of an actual psychiatric disability more than 12 months after injury.

Relatively speaking, there are few **psychiatric** problems that continue to affect the long term recovery or social adjustment in the majority of severely brain injured adults. Classical organic psychoses, such as the Capgras syndrome, are quite rare and usually respond to

medication. Mood disorders, such as manic depression or hypomania appear more frequently and are less reliably controlled by medication, probably because the psychiatric disorder, per se, inevitably interacts with neurological mechanisms which normally control mood and emotion. These mechanisms are invariably damaged after severe brain injury, often as a consequence of frontal lobe injury, and this results in a loss of neuro-modulatory mechanisms (Wood and Eames 1981).

Problems With The Psychiatric Approach

A major disadvantage of a psychiatric approach is its tendency to label many transient **behaviour** problems as more enduring **psychiatric** problems. This is most likely during the acute phase of recovery when many problems of disturbed behaviour result from cognitive changes which interfere with clear thinking creating suspiciousness, irritability, disorders of thinking or an aggressive response. These are frequently treated with major sedative drugs, such as the phenothiazines which create their own management problems, such as an increased risk of epilepsy.

Many problems of aggressive behaviour are actually the result of **episodic dyscontrol** (Mark and Ervin 1970), due to an electrical (epileptic) anomaly, often associated with the medial portion of the temporal lobes (Wood and Eames 1981, Wood 1984a, Bond 1984). Often in such cases,

one observes a paradoxical situation in the prescribing of doctors who, on the one hand, prescribe anticonvulsant medication prophylactically, to prevent grand mal seizures, (a practice which has been shown to be ineffective; McQueen et al 1983)), whilst on the other hand, almost cancelling out their effect by prescribing phenothiazines, to control aggression or other disturbed behaviour (which may be a product of such an electrical anomaly).

Another unwanted effect of prescribing phenothiazines is to possibly delay recovery in some way. Sedation maintains the patient in a state of post traumatic confusion longer than would otherwise be the case. This means that clarity of thinking is delayed and other cognitive problems prolonged. This form of medication can also delay physical recovery because whilst a patient is sedated physical functions, such as swallowing, are impaired, so the removal of a naso-gastric tube for example, might be delayed because the patient is unable to develop an adequate swallow reflex, with the result that they frequently choke when encouraged to try semi-solid food.

Recovery from brain injury is, by implication, a **dynamic** process. Alterations in behaviour, awareness, intellectual capability and emotional control may occur because of changes to the environment, medication or control of neurological sequelae such as epilepsy. On

the other hand, a psychiatric label tends to be an **enduring** construct which may misrepresent the emotional or adjustment process of an individual recovering from brain injury. This of course does not mean that psychiatric labels should not be used when there is clear evidence of a psychiatric abnormality. As already acknowledged, manic-depressive mood changes, hypomania, and schizophrenic like psychoses, are all seen following such injuries and require specialist psychiatric treatment. Unfortunately however, many disturbances of mood and behaviour, which are primarily neurological in origin, tend to get 'labelled' using a psychiatric frame of reference, often leading to an ineffective method of treatment.

Problems of assigning labels have been demonstrated in a different context by Mischel (1968). He presented evidence to show that behaviour patterns tend to be situation specific. Behaviour disorders resulting from a damaged brain are, in the months following injury, almost certainly undergoing a process of recovery. This means that their clinical presentation is likely to vary from day to day, but with a probable trend towards recovery over a period of weeks. Psychiatric diagnoses however, tend to reflect presumed personality **traits.** Mischel showed that such traits seldom account for more than 10% of behaviour variation in a normal population. In the case of a brain injured population this may be even less.

The variability in behaviour is largely explained by an interaction between the person and the situation (Powell 1981b). The nature of this interaction concerns the way in which the individual perceives and is able to interpret the situation and plan a response. This is inevitably impaired by severe brain injury, making judgement less reliable and behaviour less consistent than would be expected from a normal person, or even somebody with a psychiatric abnormality.

2. A Behavioural Description.

Behaviour problems which are attributed to some "emotional disorder" need not be considered in generalised or ill-defined terms. Through the aid of an objective **observational analysis**, they can be defined in terms of explicit environmental events which, once identified, can potentially be manipulated. Such close observation also helps to identify neurologically mediated behaviours because their characteristics (see section 2) distinguish them from environmentally elicited responses.

Analysis of Behaviour. Functional analysis is an analysis of the inter-relationships between environmental and behavioural events. It is assumed that knowledge of such relationships will allow human behaviour to be predicted and controlled (Davy 1981). An analysis of behaviour gives an opportunity to describe behaviour objectively. It is therefore, a **dynamic** process. As the observed behaviour alters, so does the programme designed to accommodate and control it. This further results in a change in the way that behaviour is described, allowing scope for further alterations in the way the community, family or the "caring professions" think of such behaviour.

Recording Behaviour. Accurate behaviour analysis requires an objective recording of behaviour, avoiding, where possible, the use of potentially misleading labels and the tendency to **interpret** behaviour. Instead, we record only:-

1) Stimuli which are present when behaviour is displayed.

2) Any consistent relationships which exist between different events in a person's life.

3) (For the purpose of identifying organically determined behaviour), the absence of such relationships during the presence of a consistent or frequent behaviour response.

Often, nursing or medical casenotes will describe a patient as having had "a fit". From a behavioural point of view this is a very inadequate description of behaviour because it does not specify the **kind** of behaviour that took place, thereby making it difficult to decide whether the fit was genuine, (grand mal or psychomotor), or some other kind of behaviour phenomenon. A similar problem occurs with the umbrella term "aggression". If the objective is to try and classify aggressive behaviour into that which is purposeful and goal directed, or alternatively, the consequence of some neurological event over which the patient has little or no control, then a more detailed description of the behaviour involved in the aggressive act must be given. For example:-

1) Is the aggression directed towards staff, other patients or items of furniture?

2) Was the aggression physical or verbal?

3) Was the aggression an isolated incident or does it occur frequently and if so, does it occur hourly, daily or weekly?

4) Do the episodes of aggression last a matter of seconds, minutes or continue until they are interrupted by another individual?

5) Is damage inflicted and if so is it minimal, moderate or severe?

6) Is the aggression consistently directed at staff, patients or objects?

7) If towards staff or patients is it directed towards any individual person in particular or does it appear not to be selective.

One can of course succumb to the reductionist argument that the term "aggression" itself is a label or interpretation of behaviour, which makes the rest of this analysis invalid. While acknowledging this problem it will not be discussed further because clinically, a more pragmatic view must be adopted.

Kanfer and Saslow (1969) argue that a precise specification of the problem, in terms of observable reference, allows agreement not only on its existence and the most likely instances when it will occur, but will also help to discriminate between purposeful and goal

directed behaviour and that behaviour which is mediated by some different process, (in this case a neurological abnormality). As Sandler and Davidson (1973) stated, "a better understanding of pathological conditioning can be accomplished by analysing the interactions between (a) the variables involved in an individual's behavioural history, and (b) those determinants currently impinging upon the organism. With the knowledge of the former we can better predict how the latter will influence behaviour."

London (1972), argued that behaviour modification is a commitment to the notion that all psychological problems require a functional analysis. Davy (1981) states that there is no doubt that behaviour therapists value the importance of functional analysis and gives three reasons for this:-

(i) Davy (1981) has summarised evidence to show that many principles of conditioning derived from animal studies could not be compared to a human learning situation.

(ii) Cognitive behaviour techniques have been developed with some success but such procedures do not fit easily into traditional animal behavioural models.

(iii) There have been recent changes of emphasis in animal learning studies which reduces the **inductive** approach to recearch, characterised by the cataloguing of controlling variables (Skinner 1950), to a more **deductive**

approach, emphasising the need to describe the mechanisms underlying learning.

This last reason is central to this thesis. It is not enough to show that the behaviour of the severely brain injured will respond to procedures developed from conditioning techniques, some attempt must also be made to describe the functional relationships underlying neuropsychological processes affected by brain injury. This may allow some insights into the mechanisms of learning in the brain injured.

This idea derives some support from Beaumont (1983a). He states that neuropsychology rests entirely upon inferences about brain organisation derived from the observation of behaviour. There are important limitations to this approach because, as Beaumont points out, in the normal individual there is no way in which behaviour can be directly related to brain events. Weiskrantz (1973) argues that this is because the brain is treated as a **dependent** experimental variable when, as Beaumont suggests, it should be treated as the **independent** variable, with behaviour as the dependent variable. In psychology experiments, the independent variable is nearly always a stimulus-variable; some **input** to the organism. The dependent variable is the converse of this; a response, which indicates an **output** from the organism.

In clinical neuropsychology our interest is on how damage to the brain has affected behaviour. It is

impossible in such cases, to control the location or nature of pathology, making observations of the independent variable difficult to control. This means that individual patients must be selected and studied so that different forms of behaviour (the dependent variable) will tell us something about the mechanism producing them - the brain, (the independent variable).

This is precisely the approach adopted by this analysis of behaviour. In experimental neuropsychology, techniques are used to study the (usually cognitive) behavior of normal subjects, which may vary as a function of their brain state. The experimenter seeks to vary this state by introducing drugs or inducing fatigue to see how presumed alterations in the state of the brain will affect the performance of the subjects.

The conclusions reached from such methods remain tenuous because they rely on inferences about the way "damage" to the brain might affect the "functions" which control behaviour. The clinical combination of behavioural and neuropsychology used in this study allows the brain to become the dependent variable because the environment, if not constant, can be controlled. This provides us with two kinds of opportunity: one is to observe the responses of **different** brain injured persons to the **same** set of stimuli or events; the second is to observe the response pattern of a **single** patient, with a particular kind of brain injury, to similar environmental manipulations **over**

a period of time.

To gain insight into brain-behaviour relationships it is not sufficient to adopt the "radical behavioural" approach, described by Thomas and Blackman (1976) as a method of explaining behaviour without reference to "inner causes". The **neuro-behavioural** approach applied here adopts an earlier behavioural model, the **S-O-R** model (Woodworth 1929, Lindsley 1964, Keehn 1969). Here, **O** represents the condition of the behaving organism (considered in mentalistic terms) involving intentions, purposes or wishes. After severe brain injury the disruption of neural mechanisms seems to demand acknowledgement of the **O** variable because the antecedents of behaviour are often organic, rather than environmental. As such it becomes important to know the workings of the "black box", to try and determine whether any regularly occurring behaviour is elicited environmentally and therefore able to be predicted. Alternatively, behaviour which occurs spasmodically or cyclically, with no warning and with no apparent relationship to environmental events may be identified as neurologically mediated, leading to medical methods of management as opposed to pychological.

The approach of this thesis conforms to the experimental process of "behavioural psychology", advocated by Powell (1981a) for use by rehabilitational and

neuropsychologists involved with the treatment of brain injured patients. The implication is that all treatment approaches, with respect to brain injury, should have a theoretical and scientific footing, an argument recently revived by Baddeley (1984). Powell proposes that behaviour therapy, by its very nature, is an experiment because of the controlled manner in which techniques are applied. He further states that because behaviour therapy involves learning, it implicitly modifies the function of almost every region of the brain. This makes it a "legitimate aim of behaviour therapy to directly attempt the modification of the brain itself".

Powell emphasizes the strong association which exists between behavioural psychology and neuropsychology. This presumably has strengthened since neuropsychologists have shown (albeit reluctantly), a willingness to engage in therapy, using their traditional skills of assessment to detect, describe and localise the functional deficit in ways that predict the impact of such dysfunctions on a person's life outside hospital.

Developing from this interaction is the role of the rehabilitation psychologist concerned with retraining and re-education of a variety of basic behavioural skills for life in the community. Powell becomes rather critical of the rehabilitation psychologist for preferring and "limiting" their use of behavioural methods to the operant paradigm (e.g. Fordyce 1971; Hollan 1973). He

fears that the main problem with this is that when a rehabilitation centre is run along operant lines all cases tend to go to the same programme, with the result that brain injured cases are not necessarily picked out or seen as a special group.

This is not necessarily true. For at least six years, rehabilitation specialists have been advocating the need to separate brain injured from general rehabilitational problems (Evans 1983). This thesis is concerned with the first project to properly achieve this separation, allowing a comprehensive brain injury rehabilitation programme to be run entirely on operant conditioning lines, yet flexible enough to allow other forms of social learning to be supplied where appropriate. Information obtained from this programme has been assembled for presentation with the following aims:-

(1) To provide a **description** of organically determined disorders of behaviour.

(2) To develop psychological methods of treatment appropriate for the management of these disorders in a rehabilitational setting. Treatment is aimed both at controlling problem behaviours as well as promoting the learning of those socially adaptive and functional behaviours that are necessary for daily living.

(3) To examine the role of attention in behavioural learning following brain injury.

CHAPTER 2

ORGANICALLY DETERMINED DISORDERS OF BEHAVIOUR

An important reason for observing and describing behaviours objectively is to allow categories of behaviour to be linked together in a "functional" way (Kanfer and Saslow 1969). This procedure allowed Wood (1984) to propose "a simple taxonomy" of organically determined behaviour. The classification used in this taxonomy does not necessarily depend upon some common organic, psychiatric or neurological abnormality, intead, it is determined by the **effect** the behaviour has on other individuals, the environment, or the selection of treatment methods for its control and modification.

Three basic behavioural categories seem to emerge which can be designated; **positive, negative and dissociative** behaviour disorders. These are not mutually exclusive; a patient may present one, or any combination of behaviours from each category either at the same or different times. Their significance as separate categories lies in the fact that the behaviours in each category appear to have a different meaning, either in terms of aetiology, the choice of treatment programme and, perhaps most importantly, the expectation of treatment outcome.

2.1 POSITIVE BEHAVIOUR DISORDERS

Positive disorders include behaviours which **actively** interfere with the rehabilitation process or the social acceptability of the patient. They are largely antisocial behaviours and can be divided into at least two major subcategories; **aggression** and **disinhibition.**

2.1(i) Aggression

Johnson (1972) argued that the term aggression has so many meanings and qualifications that, in effect, it has lost its meaning. Bandura (1973) however, suggested that this diversity is useful because the different meanings reflect different approaches to the study of aggression and different ideas as to what is involved in such behaviour. This approach is criticised by Tedeschi, Melburg and Rosenfeld (1981) who regard every text on the topic of aggression as going through the same litany of problems but without making any difference to the **treatment** of the topic.

In this thesis the approach towards aggressive behaviour (as to any other behaviour) is to comment on its objective characteristics. This involves adopting a fairly **broad defnition** of the term as:- (a) Behaviour which is damaging to individuals or property.

(b) Attitudes, moods or gestures which people find threatening or intimidating.

(c) Purposeful behaviour which disrupts activities.

From systematic observations of aggressive behaviour it

became possible to make inferences about the neuropsychological systems that mediate or promote such behaviours, particularly by observing the effects of medication in association with behaviour management, contrasted to behaviour management used alone.

Aggressive behaviours often follow severe brain injury, whether caused by trauma (Lishman 1978; Wood and Eames 1981; Wood 1984), subarachnoid haemorrhage (Walton 1952; Logue et al 1968; Story 1970), or infection (Greenwood et al 1983). Such behaviours occur in a range of individuals, from those who are impulsive or disinhibited in character, to those in which driveless, apathetic and lethargic behaviours predominate.

Certain kinds of aggressive behaviour disorders have been associated with different kinds of brain injury. Impulsive and unprovoked aggression occurs in connection with **temporal lobe** damage or EEG abnormalities (Mark and Ervin 1970; Elliot 1982; Bond 1984). Poor emotional control which causes individuals to over-react to frustration is another type of aggression, frequently seen in association with frontal lobe injury (Lishman 1978). A third kind of aggressive behaviour **follows** brain injury but does not appear to have any **causal relationship.** With this type of aggression the behaviour associated with it can nearly always be seen to result in some kind of reward, (however gratuitous), to the individual making the aggressive response.

This thesis does not seek to imply that an actual division can be made, whereby one can predict the kind of behaviour disorder from the knowledge of the type or locus of brain injury. One important reason for this stems from an earlier discussion; behaviour disorders vary, not only on the basis of predominant neuropathology but also on the basis of premorbid personality and the personal circumstances or environmental pressures which follow injury (Lishman 1978; Powell 1981b; Bond 1984). There is however, a definite pattern of behaviour within each category which can be observed, either in an individual patient or in groups of patients, over a period of time. This division does not imply that such behaviours are mutually exclusive. It is possible to see more than one type of behaviour in an individual patient who demonstrates aggressive responses.

A common characteristic of brain injury is reduced tolerance for frustration or pressure of any kind. This is a major reason why many patients become management problems after severe brain injury. Often, they are subjected to pressures they find difficult to bear. These can be indirect pressures, of the kind produced when patients find themselves discharged as "neurologically intact". This leads to an expectation that they are fit to return to work. Many patients however, experience problems of concentration, mental or physical fatigue and

other behavioural or psychological consequences of brain injury. As a result they find it difficult to maintain the level of efficiency they were accustomed to before brain injury. The problems they encounter may result in temper out-bursts, mainly within the home, and the development of an aggressive personality with poor tolerance of frustration and little control over emotional responses (McKinley et al 1981; Thomsen 1984). Other pressures may be more direct. An example of this occurs in patients receiving rehabilitation. Often, they find themselves having to cope with new and different kinds of information, or being made to perform exercises (e.g. in physiotherapy) which may be difficult and painful. Their lack of tolerance may cause some individuals to react to such pressures and behave in a threatening way towards therapists, being aggression and/or producing a tirade of abuse, which is just as unacceptable. Such behaviour may result in a patient being discharged before rehabilitation is complete.

The relationships between aggressive behaviours and different neurological abnormalities has already received comment (Bond 1984; Wood 1984). The division of frontal verses temporal lobe type aggression is rather artificial but conventional. The description of aggressive behaviours will be given according to this division, whilst acknowledging the over-lapping characteristics of many forms of aggression and the fact that individual

patients may have more than one type of aggressive problem. This anomaly will be discussed later in this thesis.

'Temporal Lobe' Aggression. This is a consequence of damage to the medial portions of the temporal lobes. Sometimes it is associated with structural damage seen on a brain scan, at other times it is associated with paroxysmal abnormalities of the EEG, but the response can occur in a stereotyped form without any obvious evidence of brain damage or neurological abnormality. The behaviour pattern involves a fairly sudden, often unprovoked, outburst of aggressive behaviour, quite primitive in nature and poorly organised. It is usually directed at the nearest object or person and has a very destructive quality, smashing furniture, spitting, scratching or other frenzied activity. It is usually short-lived and often followed by a feeling of remorse, especially when the individual is aware of what he/she has done.

Awareness of such behaviour is variable. Some patients have total amnesia for this aggressive period. Others have a vague idea of what they are doing while they are doing it, while other patients have full awareness, yet little or no control over their actions. It is these patients who are most likely to show the greatest remorse.

This type of aggressive behaviour has been described in

the literature since the original reference by Kaplan (1899) who described an "explosive diathesis". "Following the most trivial and impersonal causes, there is the effect of rage with its motor accompaniments. There may be the most gross gesticulation, excessive movements of the face and a quick, sharp explosiveness of speech; there may be cursing and outbreaks of violence which are often directed towards things; there may or may not be amnesia for these afterwards. These outbursts may terminate in an epileptic fit."

The "epileptic" component has been variously acknowledged since Kaplan's description. Hooper et al (1945), described such "diathesis" as a symptom that occurs after head injury, in epilepsy, in mania, in depression and among aggressive psychopaths. They regarded it as a "constitutional pre-disposition" and not as a direct consequence of head injury, but did acknowledge that it was commonly seen following head injury. Like more recent authorities (e.g. Lishman 1978) they sought to distinguish such aggression from the state of irritability which is common after head injury. In the 12 cases presented as part of their study they stress that 4 had no evidence of irritability as such and that the "explosive element" of the aggresive behaviour was quite different from the background behaviour, whether it be one of normal mood, irritability or depression.

It is not very difficult to distinguish between

"epileptic aggression" and other forms of aggression. Lishman describes epileptic aggressive attacks as usually beginning abruptly, having an explosive element, whereas other aggressive responses often occur after a gradual build up of anger before the peak of disturbance is reached. Unlike 'normal' aggression, epileptic aggression also usually ends abruptly, with spontaneous resumption of the patient's normal personality. Lishman also confirms that during the aggressive outburst the behaviour is particularly purposeless and unmotivated, whereas other acts of aggression are often directed at specific targets. He feels however, that loss of awareness or amnesia for the attack is a less critical factor because while a patient's expression may clearly reveal that consciousness is clouded, subsequent amnesia will not always occur. Memory can be blurred during phases of intense emotion due to dissociation, which has no epileptic associations whatever.

The circumstances in which such attacks occur must be taken into account. Ordinary aggressive behaviour is likely to be regularly precipitated by circumstances within the patient's environment. Careful investigation may reveal these as specific stresses. In the case of "aggressive psychopaths", aggression is used gratuitously, as a part of their wide ranging personality and social maladjustment. Systematic observation will usually reveal that such behaviour meets with some

reward. In the case of temporal lobe disorders however, there is frequently no pattern to this behaviour. It is not confined to certain situations, times or individuals and usually there is no (or only minimal) provocation, certainly no premeditation.

The question of a pre-disposing aggressive trait is regarded as important, especially where medico-legal aspects of brain/head injury are concerned. Lishman (1978) states that the distinction between premorbid or post-traumatic causation can be difficult, especially if the patient had an unstable personality before injury. The existence of a temporal lobe abnormality on EEG examination cannot be regarded as conclusive evidence in support of a post-traumatic effect because a high proportion of non-brain injured patients with aggressive personality disorders show temporal lobe abnormalities on the EEG (Hill 1952; Williams 1969).

There are however, a number of individuals who can be shown to have developed aggressive behaviours characteristic of "epileptic aggression", immediately or shortly after some injury to the brain. Specific illustrations of this are available in the literature: in patients who have sustained head injury (Wood and Eames 1981; Wood 1984); following herpes simplex encephalitis (Greenwood et al 1983); while Storey (1970) found the same effect in patients following subarachnoid haemorrhage.

'Frontal Lobe' Aggression. Lishman (1978) describes the "symptom of reduced control over aggression". This is discussed in a section on personality change after head injury deals primarily with the effects of frontal lobe injury. Unfortunately, Lishman does not make it clear whether he attributes this particular aggressive characteristic specifically to frontal lobe injury. He just states that difficulty inhibiting an emotional response "is frequently seen after head injury and often enough in relative isolation from other problems to suggest it may be founded in some focal cerebral pathology".

These patients typically over-react to minor provocation or frustration. The characteristic feature is that the emotional response escalates to a level quite disproportionate to the eliciting event and, once started, the patient seems to have little or no control over its course. In contrast to 'epileptic aggression' behaviour associated with the frontal lobes is almost always provoked (however minimally) and usually directed towards the source of such provokation. Again, Lishman has distinguished between this type of behaviour and that produced by post traumatic irritation.

'Learned' Aggression. Some aggressive behaviour is not directly an organically determined response being mainly controlled by environmental contingencies and some form

of reinforcement incentive. Its inclusion in this taxonomy is merited by the fact that usually, such behaviour did **not** have a premorbid history. Although not having any obvious organic characteristic or 'eliciting mechanisms', the behaviour does have something in common with the "frontal lobe" type of aggression, lacking as it does a degree of normal inhibition and described by the relatives of the person as being "out-of-character". In our experience, such purposeful aggression is not related to a paricular type of brain injury. It appears to be a characteristic that may occur in isolation or in parallel with the more obvious organically determined disorders.

This behaviour seems to exist as a result of associational learning, beginning during the early recovery period when a patient is emerging from coma. It is often the case on busy neurosurgical or accident and emergency units, that the principle, "quiet behaviour is good behaviour", is adopted by nursing staff. This means that patients who lie quietly in bed, who are undemanding and present no trouble, go un-noticed and therefore, in a behavioural sense are not rewarded, being left to their own devices in between meal times or medication. Other behaviours however, such as shouting, incontinence or walking aimlessly about the ward, are seen as "bad behaviours", in need of control by the nursing staff. Attention is given to this behaviour, usually by talking to the patient, telling him off, returning him to his

bed/cubicle, or any of a number of things that are likely to directly or indirectly reinforce such behaviour and increase its possible frequency.

From this, an unintentional shaping procedure can develop. Patients gradually associate (whilst still in a disoriented, confused or partially conscious state) disruptive behaviour with attention. Slightly later in recovery, with an increase of awareness, a form of cognitive learning takes over, the patient realising that there is an increased chance of getting his own way if he behaves in a disruptive, intimidating or directly aggressive manner. As a result, this kind of response is reinforced, frequently developing into attention seeking behaviour.

The above explanation does not allow for the fact that many patients with aggressive characteristics are quite unpleasant, menacing and distasteful individuals. It is probable therefore, that in many instances the behaviour was a premorbid disposition, or personality trait, that was either not reported or which remained latent and which was allowed to emerge following injury.

2.1(ii) Disinhibited Behaviours

Disinhibited behaviours of various kinds are commonly seen in association with frontal lobe lesions. The 'syndrome' can include coarse social behaviour, over-familiarity, often of a sexual kind, which in more severe cases can include very inappropriate sexual behaviours. Disinhibited patients often make gross errors of judgement, are tactless, over-talkative, and show a marked indifference to the effect their behaviour has on others and a similar lack of concern for the future consequences of their actions. The more frequent and socially important aspects of this behaviour disorder will be discussed in turn.

Sexual Behaviours. Inappropriate or uncontrolled sexual behaviour is perceived by many people as being just as distressing or threatening as aggression. Sexual disinhibition varies considerably in its nature and intensity including tactless attempts at intimacy, conversation loaded with sexual innuendo, inappropriate touching, lewd remarks and, in more severe cases, indecent exposure and public masturbation.

There appear to be three types of change in sexual behaviour following brain injury.

(1) The first is a more or less permanent state of promiscuity or coarse social behaviour with a lot of sexual innuendo and inappropiate touching. This is the sexual behaviour that is typically associated with

disinhibition. As such, the behaviour is not specifically sexual but is simply one feature of a behaviour pattern which includes many aspects of the 'disinhibition syndrome'.

(2) An alternative state occurs following injuries to the frontal lobe which has the effect of making the individual underaroused, lethargic, apathetic and disinterested in most things, including sex. Spouses, and the patients themselves, describe how sexual **ability** remains intact. They are able to achieve and maintain an erection but their **interest** in sex is diminished. This rarely occurs in isolation, being part of a broader spectrum of behaviours which are similarly affected.

(3) There is however, one kind of aberrant sexual behaviour that can be isolated in an otherwise normal behaviour pattern. This typically occurs after some temporal lobe injuries, when sudden and impulsive behaviour changes, specifically of a sexual type, are produced. These are associated with epileptic involvement and are characterised by the fact that inter-ictal behaviour appears normal in every way, particularly sexually.

This characteristic tends to distinguish temporal lobe sexual disorders from frontal lobe disorders where the sexual problem is more or less a continuous feature of the person's behaviour. Temporal lobe sexuality is, by definition, epileptic in nature (Powell 1981 a,b) and has

been described as producing:- hypersexual episodes (Blumer 1970), transvestism (Davis and Morgenstern 1960), exhibitionism (Hooshmand and Brawley 1970) and fetishism (Mitchell, Falconer and Hill 1954).

Powell (1981) discusses these conditions, concluding that they are probably due to the influence of an epileptic discharge upon the limbic structures linked to the temporal lobe. This is a generally accepted view because of the association between sexual activity and limbic structures, such as the amygdala and hypothalamus. Changes in sexual behaviour following head injury however, probably occur more frequently in association with damage to the frontal lobes and their limbic connections. There does not appear to be any real evidence that frontal lesions themselves specifically affect sexual drive (Powell 1981 a,b). In the absence of limbic lesions it seems that sexual behaviour can carry on as normal after brain injury (Post et al 1968; Miller 1954 and Pippard 1955). These studies, on cases of frontal lobectomy or modified leucotomy, failed to find evidence of sexual abnormality or a change in sexual arousal. More recently, Freeman (1973) gave the Kinsey sexual interview to 64 patients before and after frontal topectomy. He found that only ten took more interest in sex afterwards, while another ten were less interested.

One cannot assume that there is sufficient correspondence between brain injury inflicted surgically and brain

injury sustained traumatically to allow a straight forward generalisation from surgical studies to other brain injuries. However, Powell is probably correct because data to be presented later suggests that sex drive does not seem to be particularly affected by damage to the brain, except when it occurs as a part of a more widespread disinhibited behaviour pattern.

Insight. Some degree of lack of insight is an almost inevitable consequence of severe frontal lobe injury. It appears to underlie, and in part explain, the curious anomalies of reasoning and judgement which become particularly marked when matters bearing on the patient's own conception of his circumstances are discussed. In such cases the patient may utterly reject evidence which is at variance with his own beliefs and will attempt to reconcile any inconsistencies stemming from this in the most facile and implausible manner (Whitty and Zangwill 1977). Zangwill (1953) made an interesting proposal with regard to loss of insight. He maintained that difficult behaviour, such as poor reasoning ability and failure to accept what to others is an obvious statement of fact, is not actually a **feature** of lack of insight but a **consequence** of lack of insight. He regards it as a deliberate attempt by the patient to maintain a consistent attitude towards himself and his circumstances, as a defence against a "catastrophic reaction". It would appear that Zangwill's interpretation

is more appropriate to patients who demonstrate denial after brain injury. If patients with lack of insight could actively (consciously or unconsciously) construct such a defensive framework, then one might expect that they could also actively control other aspects of their behaviour. This does not seem to be the case in circumstances where lack of insight exists in association with a variety of inappropriate and socially unacceptable behaviours.

2.2 NEGATIVE BEHAVIOUR DISORDERS

Blunted affect, psychomotor retardation and loss of initiative, are some of the more commonly observed behavioural characteristics seen after severe brain injury. The patient is often unwilling (or unable) to initiate behaviour and can display great difficulty trying to perform the most routine activities of ordinary life. Behaviours that were once automatic can now only be carried out with a great deal of effort. The major component of such a behavioural syndrome is the development of a state of **apathy.** It is what the patient **won't** do that becomes the problem, rather than the inappropriate or threatening behaviour which characterise the positive behaviour disorders.

Lack of effort may be a direct consequence **of** the brain injury or an emotional reaction **to** the injury. The latter condition will be considered in the next section on dissociative disorders. This section deals with circumstances where the brain injury **imposes** a state of apathy and lethargy, over which the patient has little or no control and which poses very difficult problems for rehabilitation and independent living.

A lack of purposeful or goal-directed behaviour may occur following damage to the frontal lobes or high brain stem structures. The behaviour that can be expected from such injuries varies according to the severity of the damage

and the neuropsychological mechanism involved, for example, arousal, drive or motivation.

Arousal Disorders

Arousal is generally used to describe a state of an organism on some continuum, such as sleep - wakefulness. Frequently, this continuum is refered to as **consciousness**, but this can lead to confusion because it also implies a degree of awareness of the self and the environment, which leads on to the further implication of a **cognitive** involvement. The mechanisms of consciousness have been discussed by Plum and Posner (1980), while the definitions of the terminology used have been more recently commented upon by Eames and Wood (1984).

Basically, there are two major aspects of conscious behaviour: **content of awareness** and **arousal.** Content represents the sum of cognitive and affective mental functions. Obviously we can never gain direct access to this area but it is reasonable to infer that a brain injury, which interferes with cognition and therefore the interpretive functions of the brain, must also reduce, to some extent, the person's awareness of himself and his environment. (This idea will be discussed in the next section describing dissociative disorders).

Arousal on the other hand is a physiological construct, more closely related, behaviourally at least, to **alertness.** While it is evident that cognition cannot occur without some degree of arousal, evidence presented

by Plum and Posner shows that arousal in itself does not guarantee cognition.

Probably, the most severe form of an arousal disorder following head injury is a **Persisting Vegetative State** (Jennett and Plum 1972). After severe head injury some patients remain comatose for days or weeks. Many of these would subsequently die from respiratory infection or other complications. Modern nursing and medical care has prevented this happening in a large number of cases, ironically preserving a number of individuals in a persisting state of coma. Continuous sleep-like coma rarely lasts longer than 2-4 weeks however, whatever the brain injury (Plum and Posner 1980). After that time patients develop a **chronic unresponsive state** in which they show periods of apparent wakefulness, random eye movements and primitive postural reflex and motor activity but give little or no evidence of recognising or interpreting the environment, or having any other mental content. Most remain in this state for months or even years and a few ultimately regain limited speech and volitional motor activity. All such patients have severe intellectual and neurological deficits (Walton 1977) due to diffuse damage of the cortex, specific diencephalic structures or the brain stem. Usually however, it is due to damage in all three areas (Jennett and Plum 1972).

Although there are similarities in the general pattern of behaviour exhibited by patients with arousal disorders,

there has been an effort to distinguish arousal deficiencies, caused by damage to frontal lobe structures from those which are seen following brain stem lesions. Luria (1973) comments that arousal disorders are usually seen in frontal lobe lesions only when the damage is widespread and (almost always) bilateral. It would appear difficult to isolate such large lesions from possible brain stem involvement because they often occur in association with hydrocephalus, which causes **coning**, whilst traumatic lesions frequently involve brain stem structures because of **rotation** effects.

Behaviour disorders following such damage will vary depending upon the size of the lesion. When the lesion is small, patients may show a reduction in their level of inactivity, described by Lishman (1978) as aspontaneity, slowing and inertia. The speech of such patients is laboured and there may be periods during which mental and physical activity virtually come to a halt. Luria remarks that the problems arising from small lesions become particularly manifest when patients are expected to complete relatively difficult behavioural tasks that require the formation of a plan.

With large bilateral lesions of the frontal lobes **anergy** is usually the main behavioural characteristic. This results in a major breakdown of purposeful and directed behaviour. Luria describes an extreme form of arousal disorder which he attributes to "massive damage of the

frontal lobes", called the **apathico-akinetic-abulic syndrome.** In this state, patients lie completely passive, express no wishes or desires and make no requests, to the point that not even a state of hunger may rouse them to take necessary action- "even if hungry and thirsty they make no active attempt to take food or drink placed on their table" (Luria 1973).

Luria emphasised that although there is a breakdown of organised behaviour in such cases, patients are visibly aware of any change taking place in their environment, such as "someone coming in, a squeaking of a door, or coughing by a patient in the next bed". These events often arouse "a strong orientating reflex, most frequently manifested as movement of the eyes or sometimes turning the head toward the stimulus". Such patients still find it almost impossible to perform a definite action in response to a command. They will however, perform various automatic actions, such as pulling at a sheet, picking up and dropping, in a repetitive manner, articles with their fingers, or repeatedly scraping the wall near their bed. Often they appear involuntarily attracted by a small spot on the wall and will make attempts to clean it, not stopping even when asked. In contrast, they rarely take an examiners hand when instructed, but if the examiner puts his own hand into theirs they are likely to grasp it, often without being able to terminate this grasping

reflex. Luria describes such disorders of arousal caused by frontal lobe damage as **secondary inactivity.**

It would be surprising, however, given the complex anatomy of the frontal lobes and their connections with other cortical and limbic structures, if arousal disorders were the legacy of frontal lesions alone. Indeed, Lishman points out that "features closely akin to those seen with frontal lesions may occur with diencephalic and brain stem lesions." Damage to deep midline structures, post diencephalic areas and upper brain stem, often produce solemness, hypersomnia and states of profound stupor. A recent study by Trimble and Cummings (1981) found lesions of the brain stem resulted in a lowering of arousal which was represented by a degree of apathy and withdrawal. Eames and Wood (1984) have also associated arousal disorders with brain stem lesions.

Luria describes arousal disorders following brain stem lesions as **primary inactivity** because they produce a loss of cortical tone, due to lesions of the upper part of the brain stem, hypothalamus, mammilary bodies and hippocampus. Because the constant input to and output from these structures is interrupted (in traumatic head injury, usually by the rotation effects of the brain around the axis of the brain stem, as a result of deceleration forces) a whole series of psychological processes will be affected.

Probably the best known arousal disorder linked to brain stem damage is **akinetic mutism.** This was described by Cairns (1952) and Plum and Posner (1980) as a state in which the patient lies immobile and mute, but (in distinction to the state of PVS) appears fully alert, although exhibits virtually no spontaneous movement or speech. Such patients do however, follow moving objects and keep their eyes vigilant.

Lishman draws attention to the fact that although such patients are apparently in an akinetic state of muteness and immobility, they can, if forcibly roused, prove to be normally orientated. Walton (1977) on the other hand, stated that "although the patients eyes remain apparently alert to moving objects, strong afferent stimuli are incapable of arousing him". The general view however, seems to favour an orienting response in akinetic states. Luria describes patients lying passively, frequently in a drowsy or an "oneroid" state, making no effort to follow instructions which involve displaying a motor or verbal response, but still showing a tendency to respond spontaneously to "irrelevant stimuli". Cairns, in his (1941) original description of akinetic mutism, also supports the apparent existence of an orientating responses by stating that "when following the movement of objects, they may be diverted by sound". There is of course no way of knowing whether such individuals are **aware** of their environment whilst in this state.

Damasio (1979), also described a state of mutism with "bradykinesia" in association with **frontal** lesions. Essentially the same behaviour is observed and the patient is able to show an orienting response, providing dorsolateral or cingulate gyrus damage has been avoided.

Drive Disorders

Drive problems seem to occur following less severe frontal injuries or diffuse cortical damage of the kind seen following anoxia. Characteristically, a person loses interest in the environment, taking a less active part on social occasions. Behaviours that were once performed automatically and which were taken for granted, only seem to be completed at the expense of great effort. This increase in the amount of effort needed to perform simple day to day activities of daily living can lead to problems of psychological adjustment, possibly causing a depressive reaction. It may be that one consequence of this would be the development of an apathetic state. Some patients in this condition may indulge in excessive behaviours such as eating or smoking but appear unable or unwilling to work for these enjoyments or make any effort to overcome obstacles in the way of such rewards. In such circumstances not only will the individual go without, but will often appear unaffected by this self-imposed deprivation.

Lishman (1978) describes less severe forms of arousal disorders where frontal lesions lead to a loss of drive,

characterised by aspontaneity, slowing and inertia. Damasio also remarks on the frequent tendency of frontal lobe lesions to produce "a more or less profound disinclination to act". He directly attributes this to a loss of **drive** which "lessens the inclination to wash, dress or initiate any purposeful activity."

Unlike arousal disorders, patients with a disturbance of drive may spontaneously **describe** an interest in some activity but, if given the opportunity, fail to **show** any such interest. This is more of an inability to translate interest (a cognitive component) into the physical activity necessary to partake of such interest (the drive component). As such, the disturbance of behaviour may more correctly be regarded as a problem of **drive.**

Motivational Disorders.

Millikan (1970) in a symposium on behaviour changes following cerebro vascular accidents (Benton 1970), identified a decrease in motivation as being of critical importance when predicting recovery. He acknowledged that patients with such problems were difficult to rehabilitate and that any prognosis regarding a return to work must be guarded.

Clearly, motivation is central to rehabilitation following any form of disablement. In head injury it is a major problem because various aspects of motivation can be disturbed (Belmont et al 1969, Field 1976, Powell 1981). Powell draws support for his view from a review of

animal studies. He cites an experiment by Kolb, Whishaw and Schallert (1977) showing that frontal lesions in rats caused a disturbance of their ability to learn and maintain sequences of behaviour which were similar to behaviour problems seen in humans after such frontal damage. Powell comments on the interesting fact that the performance of these animals could be improved if the reinforcement was made more attractive (such as by giving crushed chocolate instead of pellets) or if a state of food deprivation was introduced. Human clinical studies have shown essentially the same effect with such patients (Wood and Eames 1981, Wood 1984a). Powell concludes from the animal studies that motivation is a significant factor in determining the degree of recovery from frontal lobe injury and, by implication, the kind of response expected from rehabilitation.

The effect of motivation in rehabilitation was described by Belmont et al (1969) who, during a controlled trial, found that brain injured patients (compared to orthopaedic patients) showed motivated and directed behaviour **only** whilst the therapist was actually present. In the context of negative behaviour disorders, motivational deficiencies appear to affect the amount of **effort** a patient is willing to make to achieve some kind of reward. The cost, in terms of effort, usually appears to the patient to be excessive, relative to the nature of the reward. The situation may be aggravated by a degree

of apathy, (drive disorder), which further reduces the **incentive** value of any reward.

We can describe the level of desirability attached to any goal, as creating a degree of **incentive.** In normal individuals incentive is assumed to depend on some balance between inherent properties of the goal (or reinforcer) and the particular appetites of the organism, ('smarties' may hold little incentive for someone who dislikes chocolate).

It would appear therefore, that another necessary ingredient for incentive is the ability to experience pleasure or pain. This has been referred to as hedonic responsiveness and used to explain aspects of schizophrenic psychopathology (Kraeplin 1913, Rado 1956, Cook and Simukonda 1981). Wood and Eames (1981) reported that following many cases of severe brain injury there appeared to be changes in the quality or degree of hedonic responsiveness which affected a patient's attitude to recovery, limiting (sometimes eliminating) their response or attempts at rehabilitation, making it very difficult to identify rewards for effort. A similar comment has been made by Goldstein and Ruthven (1981) with respect to patients with frontal or limbic lesions.

A reasonable degree of hedonic responsiveness appears to be a prerequisite for motivation. The amount of effort a person with a given level of drive, is prepared to exert in order to obtain a given goal, depends directly upon

the extent to which the patient sees that goal as rewarding. If the patient is anhedonic (having no or at least a diminished sense of reward) it may be that no degree of effort is likely to be made to achieve any goal.

2.3 DISSOCIATIVE BEHAVIOUR DISORDERS

Dissociation has been desribed by Davidson and Neale (1974) as a process whereby, in an attempt to control an unpleasant emotional response, a group of mental processes split off from the main stream of consciousness, causing behaviour to lose its relationship with the rest of personality. The concept of dissociation is much older than this however, being originally introduced by Janet (1907). Like Charcot before him, Janet emphasized the neurological - physiological aspects of thinking. He believed that thought (and therefore behaviour), was a result of a synthesis of the multiple and varied experiences to which we are daily exposed. He used the term "dissociation" to describe the pathological condition brought about by a breakdown in this synthesis. Factors which lead to dissociation are partly hereditary and partly the result of some kind of physical or psychological "shock". In this way, Janet associated psychopathological reactions with physiological states. The concept of dissociation was incorporated by Freud into his system of mental defence mechanisms and regarded by Breuer and Freud to be the basic phenomenon of the hysterical neuroses.

Brain Injury And Hysterical Behaviour

It may appear very unusual to attribute hysterical features to individuals who, by any clinical measure, have sustained a very severe brain injury. The relationship is however, an established one. Whitlock (1967) found that of 56 patients admitted to psychiatric units with hysterical conversion symptoms, 63.5% had evidence of preceding or accompanying brain damage. This compared to only 5% of a control group, matched for age and sex, who were suffering from depressive or anxiety states. Slater (1965) also found in his sample that 60% of hysterical patients had a neurological disorder, while Merskey and Buhrich (1975) reported 48% in their study on hysteria and organic brain disease.

There is not a universal agreement about this association however. Roy (1982) criticises these studies because they came from hospitals with, what he calls, "a large neurological interest", (The National Hospital, The Maudsley and King's College Hospitals, London). Roy assessed the relationship by investigating a number of cases of hysteria at a psychiatric hospital where there was no specialised neuropsychiatric interest (Roy 1979). Of 31 patients who were discharged from the Clarke Institute of Psychiatry, Toronto, over a seven year period, with the diagnosis of hysterical neurosis ("meeting strict criteria"), only one patient had a neurological disorder. These patients were matched with

another group of inpatients discharged with a diagnosis of depressive neurosis. No significant difference was found between the hysterics and depressives for "organic brain disease". Roy argues that his data challenges the inevitably of a "relationship" between hysteria and brain disease. He stated that, "the further one gets from specialised neurological referral centres and influential teachers with a known interest in hysteria, the weaker the association between hysteria and brain disease".

Roy's argument appears to imply that the studies by Whitlock and others, suggests some kind of **causal** relationship between brain injury and hysterical characteristics. This is clearly not the case. It is simply an attempt to show that even in the **presence** of known brain injury, hysterical behaviours may emerge.

In this context, Slater (1982) argues that there is not much point calling such states "hysterical" because this carries implications and confusing pre-conceptions which can be avoided if we restrict ourselves to the term "dissociation". He emphasizes that, "dissociative symptoms do not mean hysteria". Dissociative symptoms he regards as "commonly occurring in a variety of functional and organic syndromes, especially conditions likely to cause changes in cerebral physiology".

It is clear therefore, that the use of the terms 'dissociation' and 'hysteria', in the context of brain injury, must be more clearly defined. The first thing to

emphasise in this respect is that such descriptions do **not** necessarily refer to long standing personality traits or individuals with a long history of immature or bizarre behaviour. In four years of head injury rehabilitation we have identified only ten patients (20%) who were clearly observed to have such characteristics. Of these only one was known to have presented problems during adolescence which, in retrospect, could predict later problems of emotional adjustment and behaviour control.

In a brief review of the syndrome of hysteria, Guze (1983) seeks to distinguish "hysteria" from "conversion states". The former he regards as a "polysymptomatic disorder that begins early in life, chiefly affects women and is characterised by recurrent, multiple somatic complaints, often described dramatically". This is refered to as "Briquets syndrome", after Briquet (1859), who made the first attempt to describe hysteria in terms of a syndrome. Slater (1982) argues however, that Briquet's syndrome, as a clinical entity, is not properly established. 'Conversion symptoms' on the other hand are more specific in their presentation and suggest a neurological disease, but without a satisfactory neurological explanation for the symptoms. They can also occur in the **absence** of a psychiatric illness.

The descriptions of 'dissociative' behaviour given here refer mainly to conversion symptoms. Sometimes, a more general disruption of behaviour is also present with the

conversion symptom, producing a clinical state which parallels that of Briquet syndrome. It is therefore difficult to fit such behaviour into either of these clinical categories so, wherever possible, the behaviour will be described as 'dissociative' without any further reference.

Examples of Dissociative Behaviour

The use of the term "dissociative behaviour" as a description of a post traumatic behaviour disorder, is based on a number of careful observations of the behaviour of individuals during the process of rehabilitation. These reveal that many patients present a degree of physical and mental handicap for which a satisfactory neurological explanation is hard to find. Inevitably, such behaviours prejudice both the outcome of rehabiliation and potential recovery from brain injury.

Goldstein and Ruthven (1981) give an example of such behaviour in association with brain damage. Their patient had sustained "massive traumatic brain damage to the frontal lobes". He was left with a severe speech deficit, a spastic paralysis of one arm and severe intellectual impairment. When he walked, he adopted a staggering gait and would often lose balance. The interesting aspect of this patient was that although he suffered massive brain damage and had genuine intellectual, speech and motor sequelae, there was no neurological basis for the gait disturbance. Physically, the patient should have been

able to walk reasonably well but, for some reason, refused to make the effort, or was unable to make the effort, to progress in this area.

A similar description was given by Wood (1984a) of a girl who suffered an intracranial haemorrhage which damaged large areas of the right fronto-parietal cortex. This resulted in a left hemiplegia with severe contraction of the left arm and (apparently) no movement of either the left or right leg. The neurological basis of such paralysis was in doubt however, especially when it was noted that she could spontaneously extend both legs out straight, usually at times when it was most inconvenient for staff who were trying to manouevre her wheelchair. Efforts to bend or lower the leg at such times were met with great resistance (hysterical rigidity, Pincus 1982), showing considerable strength in the muscles of that leg. Similarly, her left arm could be used at **her will.** She was observed to suddenly reach out with that arm and grab a sandwich (to which she was not entitled) off a tea trolley and place it in her mouth before staff could intervene. The arm then returned to its original contracted position.

Pressure was directed at this patient to demonstrate her capabilities for leg movement during walking and weight bearing exercises. Possibly, to avoid such pressure, she took the opportunity, during the night, to hold her foot against a hot water pipe, first the sole of her foot and

then the top of the toes and instep, until the skin had blistered so badly that, when it was discovered, she had to go to the General Hospital for burns surgery, thus assuring that she would be out of the rehabilitation programme for some time.

Pincus (1982), Lishman (1978) and many others, describe paralysis and disturbances of gait as some of the more common characteristics of conversion hysteria. Pincus states that hysterical paralysis is usually paralysis of **movement** rather than paralysis of individual muscles and usually involves more than one extremity. Hysterical gait (abasia) can also be observed in such patients. This has a bizarre character, with the affected leg possibly being dragged along the ground and not circumducted as in normal hemiplegia. This is sometimes accompanied by a "high stepping gait", the good leg being raised off the floor as though the patient were climbing stairs, dragging his affected leg behind him. When the paresis is genuine, the patient takes every care not to fall. In dissociative conditions however, the patient is seen to fall frequently, yet hardly ever in circumstances where injury might occur.

Speech disturbances are another common dissociative characteristic, usually following a genuine speech disorder (dysphasia or anarthria). In cases of continuous anarthria/aphonia, where there is a lack of neurological evidence to support an organic speech loss, there is

often reason to suspect that a degree of dysphasia may be present (possibly to protect themselves from emitting incoherent speech). Such patients sometimes present as being entirely mute, when noises, if not speech sounds, can be expected. Other patients indulge in ritual 'babbling' routines. From the speech therapy point of view these babbling sounds are rich in phonemic content and are capable of being turned into meaningful speech, if only the patient would co-operate. Usually however, such patients rarely produce these sound sequences to command or in a way that can be used as a basis for therapy.

Disorders of expressive speech are also quite common. Those experienced include continuous 'ingressive speech' (speech spoken on inhalation rather than exhalation), soft dysphonic speech, high pitched speech or an inability to produce propositional speech to command (for example, "describe what you see outside the window"), when the patient has produced normal propositional speech of a similar kind during ordinary conversation.

Another interesting dissociative feature can be described as 'hysterical blindness'. This is often seen in patients who are described as being cortically blind but observation of many of their manipulative behaviour characteristics, 'game playing' tendencies and lack of real effort in rehabilitation activities, makes it reasonable to include them in the dissociative category.

Examples of such states include a patient who, when out for a walk in the park with ward staff, was so badly behaved that she was 'left' to find her way home. Careful covert observation showed that this patient, after a period of deliberation, headed in the right direction, but managed to walk into **every** tree on her way back to the ward (which was situated at the edge of the park). The same patient, when asked to name the colours of the rainbow, quickly named all, except the colour red. No inducement on the part of the speech therapist could get her to name red, either in abstract, or in association with an object, such as a telephone kiosk. Another patient with similar 'blindness', spontaneously described the make and colour of a sports car whilst out for a walk, saying how she would like to own one, yet would make no effort at such perceptions during her therapy activities.

No attempt has been made to relate such phenomena to 'blind sight', an ability associated with cortical blindness and regarded as "extensive" following traumatic brain damage (Beaumont 1983b). To do this would require a degree of cooperation from the patient to be able to demonstrate the ability for stimulus identification and discrimination (Torjussen 1978). In the above examples such cooperation was clearly not available, also, one needs to consider the patient's perceptions in the context of other behaviour characteristics.

Motivation And Awareness

The interesting feature of all these cases, and that which distinguishes them as "dissociative", is the fact that not only do such individuals make no effort towards achieving progress but they use all their effort to maintain the status quo. To achieve this requires a considerable degree of manipulation, which implies motivation and a degree of intent. Naish (1982) refers to this and describes it as the essence of the Medical Research Council's definition of hysteria in 1941, (War Memorandum number 4) - "a condition in which mental and physical symptoms, not of organic origin, are produced and maintained by motives, **never fully conscious**, directed at some real or fancied gain to be derived from such symptoms".

Naish remarks that **motive** is accepted by Psychiatrists as an integral part of hysterical behaviour. This is not supported by the comments of Slater (1982) however. He described a range of dissociative states during the last war, all of which were occasioned by acute stress. Because such symptoms have the effect of taking the person out of the stress situation and into the safety of a hospital, they were regarded as being motivated. However, some patients, who were in a state of altered consciousness, wandered (in confusion) into far greater danger than they would have otherwise experienced if they had kept their wits. There were also situations when,

through dissociation, a paralysed limb prevented a person running for safety, keeping them in a far more precarious situation and one from which their survival was due to a great deal of luck. In these situations, Slater feels that the emotional trauma disrupted the integrated activity of the nervous system in a way which makes no sense in terms of motivation.

The importance of motivation in directing such behaviour remains unclear. In patients who display such behaviours following brain injury motivation is not simply absent (as might be expected from the comments of impaired cognition and reduced awareness, mentioned in the section on arousal), on the contrary, motivation does appear to be present, but in a negative sense because the patient is seen as making **effort**, either to maintain the status quo, or even in some cases to make themselves worse, or at the very least, **appear** worse. In such cases one can never be entirely certain to what extent patient's are aware of their behaviour.

Perhaps a better way of describing such behaviour is as a disturbance of **will** (Kraepelin 1905). He regarded hysterical behaviour as being governed by hypobulic mechanisms - conditions where will or volition become impaired. To some extent this is what distinguishes dissociative behaviour, which is motivated, conscious and described as 'malingering', from other states, where the will is directed towards some gain by processes not

clearly understood, which may be wholly or partly subconscious (Eames and Wood 1984). It is in this respect that the description of such patients made by Paget (1873) is so apt - "they say 'I cannot', it looks like 'I will not', but it is, 'I cannot will'".

SECTION 2

A DESCRIPTION OF THE TREATMENT ENVIRONMENT AND METHOD

CHAPTER 3

THE TREATMENT ENVIRONMENT

Introduction

This chapter is designed to provide a description of the setting in which this study was made, the procedures adopted and the patients who provided the data. It is divided into the following sections:-

(1) The Kemsley Unit, which provided the subjects for this study.

(2) The structure of the token economy.

(3) Behaviour recording procedures.

(4) The behaviour modification techniques used for individual treatment programmes.

(5) The patients and control subjects who were involved in different parts of the study.

3.1 The Unit

The Kemsley Unit opened in January 1979. It was designed as a specialized rehabilitation unit for patients with post traumatic behaviour disorders, severe enough to prevent rehabilitation taking place in conventional units or the person being accepted back into the community. The unit offers a full range of rehabilitative therapies, organised and applied according to the principles of behaviour modification in the setting of token economy.

The criteria for admission were that a patient both **needed** and would **benefit** from rehabilitation, but had behaviour disorders that were sufficiently severe to

prevent this in a standard setting. The corollary, to "need and benefit" from rehabilitation, means that individuals with deteriorating neurological conditions(dementia) or congenital brain damage (cerebral palsey) were effectively excluded from admission.

The building is a self contained unit within the grounds of a psychiatric hospital. It has 14 beds divided into nine single rooms and five individual cubicles. Patients have a lounge, with television, video and hi-fi units, a separate dining room and a large activities room. Physiotherapy and occupational therapy facilities are available plus a kitchen, used to train patients in domestic and cooking skills.

The unit also has access to hospital facilities such as a gymnasium, swimming pool and garden allotment. There are opportunities for horse riding, shopping expeditions or other town visits, providing opportunity for therapy to take place outside the confines of the unit. An industrial therapy unit, as well as a sheltered workshop are available when work assessment or work training is required.

The nursing staff consists of four nurses on each day shift and two on a night shift. Usually, only two of the day nurses on each shift are qualified, the others being nursing assistants or psychology graduates working as therapy assistants prior to receiving clinical training.

Therapy staff comprise, two physiotherapists, two

occupational therapists, an occupational therapy helper and a speech therapist. They are employed full time on the unit, working closely with each other and with the nursing staff to provide an organised and intensive treatment programme. A social worker is available to liaise with families and assist with discharge planning. The unit is supervised by a Consultant Neuropsychiatrist and Neuropsychologist who are continuously available to give advice and direction regarding treatment problems and who see individual patients for evaluation and assessment.

3.2 The Programme

All treatment is provided within the token economy system. This has undergone various changes and refinements in its evolution over five years but basically consists of **three token levels.** A patient would be admitted to the **first** and most basic level of the token economy very shortly after arrival on the unit. At this level, tokens are paid every quarter-hour for co-operative and effortful behaviour in therapy activities. Patients would fail to earn tokens if their behaviour was aggressive or frankly unco-operative or for any obvious attempt at manipulation, either of the system or the members of staff applying it.

Patients graduate on to the **second** token level when their behavioural state has stabilised and their co-operation in therapy more reliable. This would be determined by the

patient showing a record of consistently high token earnings over a period of two or three weeks. On level two, tokens are paid at half-hourly intervals. While there is essentially no difference in the criteria for earning or not earning tokens, staff's expectations are significantly different for patients on level two because they expect them to show a degree of spontaneity and initiative for directing and organising their behaviour and maintaining a standard of dress, conversation and social skills.

The final token level, **(three)** acts as a 'pre-discharge' level. Here, patients are paid tokens on an hourly basis, the expectation being that they should be able to maintain good, co-operative and effortful behaviour over that period. Failure to do so, at any time within that interval, would mean a loss of tokens for the whole period. At this level standards of social skills are particularly important because the patient is spending much of his/her time off the ward and in contact with members of the community.

The significance of three different **token** levels is that patients of different **ability** levels (physical, intellectual and social) can be reasonably well catered for. It avoids a ceiling or floor effect in the behavioural demands placed on different patients of varying abilities. Incentive for moving up the token levels is provided by offering better and a greater

variety of rewards on levels two and three. This can be an effective motivator when all 'privileges' are determined by the token exchange.

The most basic and probably most controversial form of this procedure is the potential loss of meals if a patient has not earned sufficient tokens to pay for them. In such situations either a part meal can be purchased, usually the main course, but not a dessert. In the event of the loss of a whole meal, a complan (milk based) substitute is offered. To avoid ethical problems a '15% rule' exists. This means that staff are not to deny patients, or, to put it another way, patients are not allowed to lose, more than 15% of their meals per month. Any variation in this rule has to be agreed by the hospital ethical committee.

Other rewards or privileges available include the regular issue of cigarettes for smokers and the use of sweets or other consumables, which in some instances are used instead of tokens to provide immediate and tangible reinforcement. Evening shop, watching television, telephone calls, visits to relatives, or time off the ward for trips to the local pub,theatre, cinema or other leisure pursuits are also contingent upon achieving levels two or three.

3.3 Recording Behaviour

The token economy system provides a structure within which a rehabilitation or behaviour modification programme can be organised. It acts as a background or platform from which individual treatment programmes can be applied. It also provides a way of **measuring** behaviour change or evaluating progress over a period of months, which would be difficult if one had to rely on the memories of individual members of staff. The way behaviour programmes are organised virtually demands that a careful and almost continuous record is kept of progress. When regular recordings are not possible, time samples of behaviour are collected at intervals throughout the treatment programme to evaluate progress.

Behavioural Baselines. To accurately define the existence of problem behaviours and measure their frequency, relative to acceptable or adaptive behaviours, it is often necessary to record a **'behavioural baseline'.** This is a very time consuming task. It involves observing and recording everything a patient does over a period of at least 12 hours; for example, watches clock (45 secs), scratches nose, drums fingers etc.. From the complete behaviour baseline a number of behaviours can be identified, on the basis that they, (1) occur with unusual frequency, (2) are inappropriate or (3) anti-social. This allows a number of behavioural categories to be assembled (Table 3.a) and graphed for

future reference (Fig 3.1). These act as the **behavioural baseline** because they show the relative frequency and/or duration of each behaviour. They also act as a basis for further recordings of specific items of behaviour, which are designated '**target behaviours**', i.e. those behaviours most in need of treatment.

TABLE 3.a

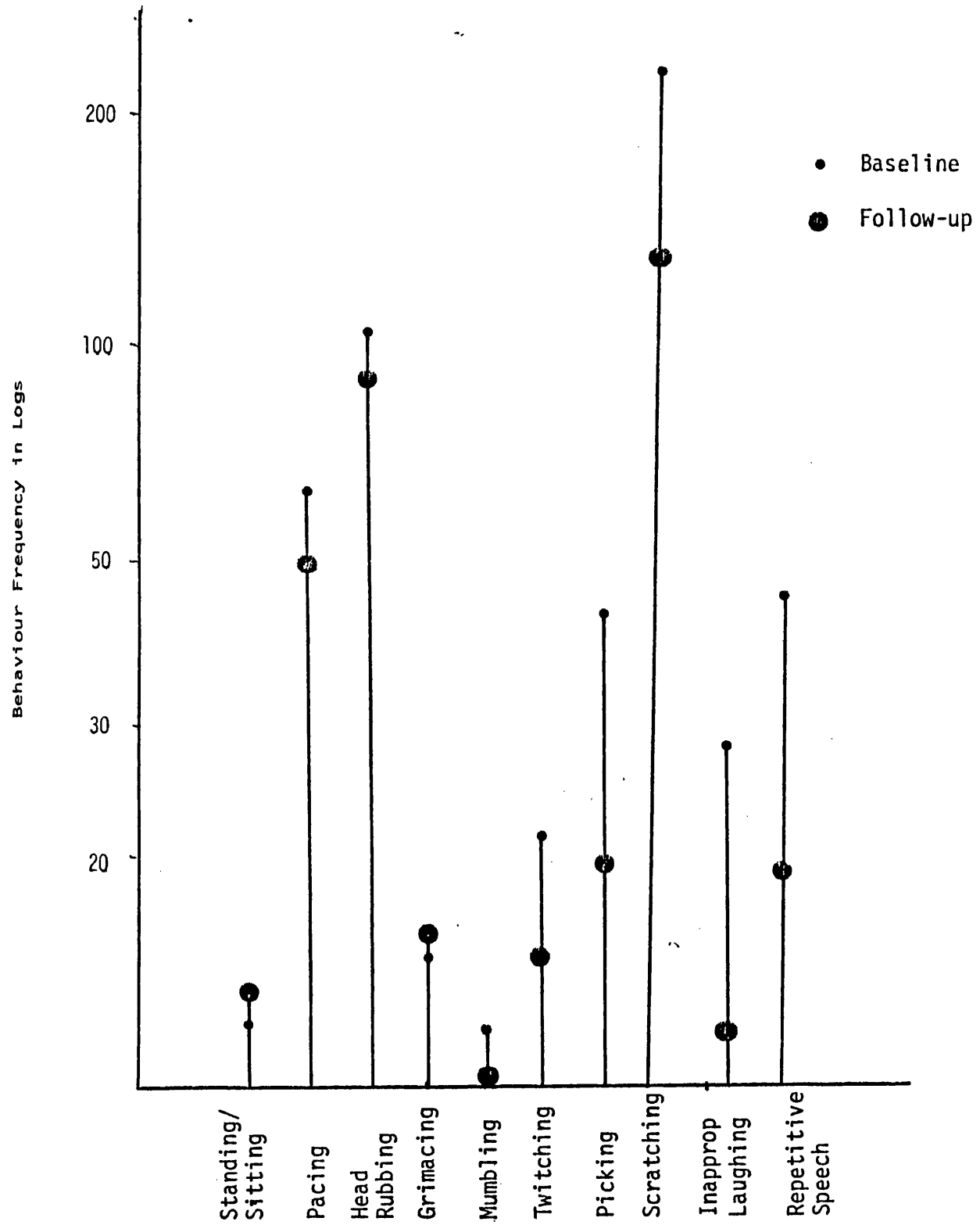
BEHAVIOURAL BASELINE

Six hour frequency baseline

BEHAVIOUR	**TIME OF DAY**					
	8.0	9.0	10.0	11.0	12.0	13.0
Standing/ Sitting	-	-	2	8	2	-
Pacing	13	10	5	11	19	6
Rubs head	24	15	11	10	17	8
Grimaces	5	1	5	-	4	-
Mumbling	1	2	2	4	2	1
Twitching	1	10	4	3	2	2
Picking	5	12	14	5	5	3
Scratching	36	22	42	35	57	44
Laughing	2	4	5	6	9	3
Repetitive Speech	5	1	12	22	2	2

FIG 3.1

Baseline and Follow-up Frequency Of Inappropriate Behaviour



The response of such target behaviours to a behaviour programme can be determined by two types of behaviour recording:-

(1) The first involves **continuous frequency recordings.** This is a very difficult recording procedure for a busy rehabilitation unit to implement and is normally restricted to behaviours which require a time out room or 'positive punishment' procedure (see later for explanation of these procedures). In such instances, every incident is immediately recorded in a daily log, together with the time of observation.

(2) It would be practically impossible to monitor all behaviours in this way so most other observational recordings employ a **time sample.** To collect a reasonable sample of behaviour it is not necessary to record behaviour over 12 **consecutive** hours. Recordings can be made on the basis of two, one hour periods each day; one hour in the morning and one in the afternoon, over a period of six days covering each hour between 08:00 and 20:00 hours. This means that after a period of treatment, one of the target behaviours is placed under close observation so that the frequency and/or the duration of its occurrence can be recorded. This allows a reasonably accurate measurement of the course of a behaviour and its response to a particular treatment programme.

Behaviour recordings using continuous or time sampling methods provide nearly all the data presented in the chapters describing the outcome of treament.

Behaviour Rating Scale. When a number of behavioural observations have to be made on the same patient or different patients over time, recording by means of individual baselines becomes time consuming and impractical (particularly in a clinical environment). Consequently, such labour intensive methods, which yield high reliability recordings, are reserved for particularly important or controversial programmes (time out room, positive punishment), where accuracy of recording is essential. Other behaviours which need to be measured are observed and recorded using a **behaviour rating scale.**

The rating scale assessment was devised to record those behaviours important to the patient group under observation. (When we started the rehabilitation programme a rating scale appropriate for our needs was not available. Consequently we developed our own, based on our knowledge of the kind of behaviours presented by our patient group, especially those behaviours likely to interfere with the rehabilitation process). The scale was used to identify, define and plot the course of behaviours that were unacceptable, antisocial, or likely to interfere with rehabilitation. It included behaviours

that were relatively easy to observe and identify and staff were instructed that no effort should be made to **interpret** any observations, thereby avoiding subjectivity creeping into the analysis.

The data collected by the rating scale process were never used to measure the outcome of specific treatment programmes neither were they used for within group comparisons. The information provided by such an assessment represents a **trend** of behaviour change, both for specific behavioural areas and at different times throughout the treatment programme. Consequently such data are not used in this study as a measure of individual treatment effects. It is still important to describe this scale in some detail however, because (1) it is important as part of the structured behaviour environment and in the clinical management of day to day behaviours; (2) it provided a framework for staff, who were inexperienced in behavioural methods, to observe and describe behaviours; (3) it aided recall of specific behaviours that might otherwise have been forgotten.

The scale used during the time of this study underwent two revisions but the version in table 3.b is the one that has been mainly used up to the present time. Independent ratings were made by eight members of staff, four therapy staff and four nursing staff, (always the charge nurse and staff nurse from each shift). This made it possible to cover a 12 hour time span each day. Each

staff member filled in the rating scale independently and without reference to the ratings of other members of staff. The eight independent ratings were then listed on a master scoring sheet and the average taken for each behaviour and for groups of behaviours under observation. Ratings of this kind were completed on each patient every three months. The staff were asked only to rate behaviour that was observed during the previous fourteen days. Although not based on any experimental evidence, a 14 day period was regarded (by the unit supervisors) as the limit of reliable memory span for observations of this kind.

The rating of behaviour was done using a ten point scale (1-10). All the items on the scale were negative ones, meaning that they represented bad or undesired behaviours. Consequently, a rating of 1 meant that the behaviour in question was either absent or hardly ever seen, while a rating of 10 meant that the behaviour represented a severe problem and was present virtually all of the time. Points within the scale were not designated as mild, moderate or severe, in order to avoid unnecessarily biasing observations.

TABLE 3.b

BEHAVIOUR RATING SCALE

Manipulation

1. Disobeys/ignores ward rules.
2. Poor time keeping.
3. Manipulates system.
4. Attempts to abscond.
5. Fakes illness.
6. Tells lies.
7. Attention seeking behaviour.
8. Argues unnecessarily.

Aggression

1. Impulsive temper outbursts.
2. Premeditated acts of aggression.
3. Physical aggression: severity
   frequency
4. Damages property during aggression.
5. Verbally threatening behaviour.
6. Non-verbal threats.
7. Tries to dominate/manipulate others.
8. Prvokes/annoys others.
9. Intolerant of others.
10. Argues unnecessarily.
11. Steals from others.

(Table 3.b Cont.)

Self Injury

1. Self abusive behaviour.
2. " " as act of bravado.

Non-cooperation

1. Disrupts group activities.
2. Refuses to participate in groups.
3. Refuses to take medicine.
4. Does not cooperate with staff.
5. Will not accept criticism.

Sexual Behaviour

1. Exposes body - unintentionally.
2. " " - deliberately.
3. Displays provocative behaviour.
4. Masturbates in public.
5. Touches self inappropriately.
6. Disinhibited sexual talk.

Social Presentation

1. Poor personal hygiene.
2. Wears clothes inapprpriately.
3. Untidy appearance.
4. Poor table manners.
5. Does not converse spontaneously.

(Table 3.b Cont.)

Drive and Motivation

1. Slow in activities.
2. Overactive.
3. Easily distracted.
4. Stares into space for long periods.
5. Erratic moods over short periods.

odd Behaviour

1. Talks to self.
2. Has obsessional tendencies.
3. Has phobic tendencies.
4. Repetitive, stereotyped behaviour.
5. Unacceptable vocal habits.
6. " " physical habits.

Orientation and Insight

1. Poorly oriented in time.
2. " " " space.
3. Talks unrealistically about behaviour.
4. Unrealistic attitude towards future.

A.D.L. Rating Scale. Table 3.c gives an example of another behaviour rating scale designed to record a patient's performance in **activities of daily living, (ADL).** This was developed in association with an occupational therapist to record, in a reasonably reliable manner over time, how well a patient could cope in different functional areas. This was also a ten point scale, but with a positive bias, so the higher the score the better the behaviour.

The important aspect of this scale is that two sets of ratings were made, based on **ability** and **performance.** This is because it became apparent from observations of behaviour in specific areas, such as occupational therapy, that patients did not always give of their best. Clearly, in a rehabilitation setting, it is very important to gain as much co-operation and effort from patients as is reasonably possible. As brain injured patients are rarely highly motivated to participate in rehabilitation activities, it was important to provide a measure of just how much effort they were putting into their activities programme because this was directly related to the kind of privileges they received.

Ability was determined not by what the therapist **thought** the patient was capable of doing, but on **observations** of the **best** behaviour demonstrated in a particular skill during the assessment period. The **ability** score therefore, represented a standard to which the more

general responses of that patient (in a specific skill) could be compared. (In rehabilitation terms, this score could only improve because it indicates the degree of recovery of a functional skill. Although we would not expect such a level of behaviour all the time, this score does represent a standard which can be used to measure general aspects of behaviour).

Contrasted to this, the performance scale represented a variable feast. Not only did it provide a measure of how much daily variation there was in a patient's ability, it also showed when a patient was not making an effort to respond up to his/her maximum level of capability.

The example given in table 3.c is not as detailed as the behaviour rating scale. This is because the A.D.L. scale represents those **general** areas of interest to the occupational therapist. She would include under those headings specific activities appropriate for the patient at different stages in treatment. Progress would be evaluated, not only on the basis of a comparison with previously recorded behaviours (to assess progress), but according to other activities available to the patient during the period for which they are being assessed. This allows a degree of flexibility to be maintained regarding a patient's assessment, which might not be available if the therapist's evaluation was restricted to a narrow set of behaviours.

These rating scale methods are not presented as being a

very accurate way of recording behaviour (although they were subject to inter-rater reliability measures such as those proposed by Ayllon and Milan (1979) for behaviour rating procedures).The assessments suggested that some behaviours under observation were situational-specific (Mischel 1968), having the kind of pattern which suggested that attention seeking or some other form of reinforcement incentive was operating. This helped separate behaviours which were purposeful and goal directed from organically mediated behaviours.

Table 3.c

A.D.L. RATING SCALE

Mobility

1. Independent- w,chair; frame; stcks.
2. Can negotiate- stairs; doorways.
3. Unassisted walking.

Orientation

1. For place.
2. For time.

Public Amenities

1. Public transport.
    (a) Bus; train; taxi.
    (b) Road sense.
    (c) Organisation of journey.
2. Telephone.
    (a) Private/public 'phone.
    (b) Uses directory.
    (c) Uses operator.
3. Lifts; escalators.
4. Restaurants.
    (a) Able to read/order from menu.

Domestic

1. Cooking.

    (a) Organisation in kitchen.

    (b) Ability to plan menu.

    (c) Use of appliances.

    (d) Preparation of food.

    (e) Washing up.

2. Table skills.

    (a) Use of cutlery.

    (b) Eating habits.

    (c) Appetite.

    (d) Posture.

3. Hygiene.

    (a) Washes/baths independently.

    (b) Cleans teeth.

    (c) Combs hair.

    (d) Shaves.

    (e) Uses sanitary towel.

    (f) Continence of urine/faeces.

Personal Tidyness

1. Changes clothes/underwear appropriately.

2. Keeps room tidy.

3. Makes bed in morning.

4. Uses laundary.

5. Irons clothes.

6. Repairs clothes.

Dressing/Undressing

1. Top half.

2. Bottom half.

Occupational Skills

1. Concentration span.

2. Hand/eye coordination.

3. Ability to work unsupervised.

4. Flexibility.

5. Motivation.

3.4 Behaviour Modification Procedures

In addition to the token economy programme a number of individual treatment programmes were organised, utilising various established techniques of behaviour modification. The aims of the total behavioural approach was to:-

1. To control or eliminate undesirable behaviours (e.g. temper outbursts, antisocial or disinhibited behaviours.
2. To shape existing behaviours into more appropriate or constructive forms e.g. social skills.
3. To evince behaviours where drive and motivational deficits predominate.

The techniques used to achieve these aims can be described as follows.

1. **Time Out Procedures.** (Negative Punishment). The concept of time-out has traditionally been based on the idea that many behaviours develop and are maintained by the attention they receive from peers or 'significant others' from which the individual receives some gratuitous reward. More recently it has been acknowledged that such time-out procedures have a rather punishing element (Kanfer and Phillips 1970) because the behaviours for which time-out is used and the ways in which it is applied do not conform to the original definition of time-out. On the Kemsley Unit, three methods of time-out were used.

(1) **Time-out-on-the-spot (TOOTS).** This is used in accordance with the original concept of time-out, denying attention to behaviours which themselves are deliberately attention-seeking or likely to be made worse by attention, for example screaming, complaining or demanding behaviours. Depending upon the behaviour and the context in which it occurred the **TOOTS** procedure would either involve a member, or more than one member of staff carrying on a conversation or an activity, apparently oblivious to the inappropriate behaviour in question. In other situations it might involve the member of staff walking away from the person or the behaviour but making sure that they maintained an indifferent attitude whilst doing so, careful not to give the impression that the behaviour was having any effect on the staff member.

TOOTS procedures are often difficult to apply, because human nature usually demands that we take notice of inappropriate behaviours around us. It is usually perceived by staff as being somewhat stressful because of the circumstances in which the procedure often occurs, as for example in the face of attention seeking responses, and the length of time the **TOOTS** procedure has to be maintained.

(2) **Situational Time-Out (STO).** For behaviours which exceed the reasonable expectations of a **TOOTS** programme an STO procedure is used. This simply involves the

removal of the patient, either from the activity itself to another part of the room or from the room to the passage-way outside. When this is done, no comments are made to the patient, such as saying that they have behaved badly. Again, an attitude of indifference and calm is maintained by the staff member. Often, although it is not necessary, situational time-out will lead to a loss of tokens for that period.

(3) **Time-Out Room.** Some behaviours are of course too extreme or too dangerous to allow this kind of casual, although structured, response. In cases which involve physical aggression the use of a **time-out room** is mandatory. This method of time-out involves placing the patient (sometimes against their will) into a locked room for periods of five minutes or multiples of five minutes if behaviour continues to be disturbed. It is this use of time-out that can be construed as punishing, especially when the patient resists and needs to be 'man handled' into the room. When this is done, staff are instructed to use "passive resistance" or "passive force" to restrain the patient, whilst refraining from talking, either to each other or the patient, in a way that would give attention, and thereby reward, to such behaviour. It is usual to lose tokens following a time-out **room** incident.

Although mandatory for aggression, the time-out room system has been used for verbally abusive behaviours and,

rather more controversially, to provide the kind of immediate and obvious feedback for inappropriate sexual behaviours or even inappropriate speech (see chapter 8, section 3).

All time-out room incidents are entered into a special book, giving the date and time of the incident, a short description of the behaviour and the period of time for which the patient was detained. The nurse or therapist then signs that entry and all entries are later counter-signed by the Consultant Neuropsychiatrist or Neuropsychologist at the weekly ward round. Examples of such procedures, used in a controlled situation, are given in chapter 5.

Positive Punishment. There are occasions when a positively punishing event is more effective than the negative punishment of time-out. In fact, experience has shown that sometimes, this may be the only technique adequate to eliminate unwanted behaviours.

Positive punishment has been applied in a variety of ways but traditionally involved the delivery of an electric shock or unpleasant noise. There are practical difficulties in administering contingent reinforcement in this form. Consequently we have used aromatic ammonia vapour, kept in small, easily carried wick bottles, which a staff member would administer, immediately after the observation of a specified undesirable behaviour, by holding for a second or so, under the patient's nose.

This particular punishing stimulus was chosen because it is **harmless** and easy to administer contingently. It has been used in other treatment settings, mainly mental handicap, proving to be effective in the elimination of undesirable behaviours (see Chapter 7), which have not responded to other methods of behaviour control.

Shaping Procedures. Shaping can be described as a series of successive approximations towards the desired behaviour pattern. Shaping procedures are described in chapter 8. These involve situations where a particular behaviour is broken down into small sub-units, each being individually practised and selectively reinforced so that one does not proceed from the first unit of behaviour to the next until the first is being executed properly and consistently.

Shaping behaviour in this way is particularly effective during social skills training where many different aspects of behaviour are involved. For many severely injured patients with perceptual, organisational or other cognitive problems, even the relatively simple behaviour of greeting somebody involves shaping a response pattern, which includes observing personal space, establishing eye contact, correct body movements, appropriate verbalisation and the importance of a graceful exit.

Positive Reinforcement. Shaping appropriate behaviour requires the systematic application of positive reinforcement. Reinforcement of this kind is not

restricted to tokens or back-up reinforcers, such as previously described, but should also include a liberal amount of social reinforcement, such as praise and encouragement, in order to feed-back to the patient, in a socially desirable way, the effect of his/her behaviour.

Experience has taught us that social reinforcement is probably the most difficult kind of positive reinforcement to provide on a **continuous** basis. Staff are aware that tokens or tangible reinforcers have to be administered for certain kinds of behaviour. Unfortunately they sometimes present such reinforcers in a mechanical and cheerless way which totally undermines the potential of such a reinforcement system. This means that senior staff must maintain constant vigilance to ensure that other members of staff project an atmosphere of encouragement, rather than allow a sterile system of reinforcement to develop, which compares more to the Skinner box than to any kind of human interaction.

Consolidation and Generalisation. One of the criticisms directed at behaviour management is that behaviour is only maintained while the patient is actually on a specialised unit. This is not necessarily the case, but a lot depends on the kind of treatment and the kind of patient being treated. There is more chance of behaviour change being maintained if the treatment programme, (1) is allowed to last long enough to **consolidate** such

behaviour change and (2) includes methods to **generalise** a new behaviour response to other situations. This helps the patient develop an adaptive and flexible behaviour pattern appropriate to a variety of situations or environmental needs.

Consolidation of behaviour is helped by the patient **over-learning** the required behavioural response. Over-learning is achieved by contingently reinforcing minimum or even accidental behaviour responses that are within the same category as the target behaviour under formal control. This means that a patient with disinhibited sexual behaviour who inappropriately touches female staff, might be subject to a time-out procedure not only for sexual touching but for all touching, even brushing one's shoulder while passing in a corridor. This apparently harsh procedure may seem unfair but it does impress on patients (who may have limited insight into the effects of their behaviour) the need to carefully monitor their behaviour, increasing the probability that this will lead to better behaviour control, more socially acceptable behaviour and improved opportunities for social reintegration of a more conventional kind.

Generalisation is necessary to ensure that the behaviour is maintained in a variety of situations outside the specialist unit. This means that, whenever possible, advantage is taken of opportunities to reinforce a target

behaviour in community situations or wherever real life opportunities present themselves. In the early stages of treatment it is common to see inappropriate behaviours rapidly extinguish, only to re-emerge if a 'new face' appears on the unit or when the patient's environment changes, such as during weekend leave when the cues which have come to be associated with reinforcement, are not available.

An important part of generalisation involves **weaning** the patient off the reinforcement system. Weaning can be done by gradually reducing the frequency of reinforcement, for example by altering the reinforcement schedule to either a fixed interval or variable interval basis, thus expecting the patient to demonstrate more behaviour for less frequent reward. At the same time it is best to replace the tangible back-up reinforcers with more social reinforcement. Examples of this procedure are given in chapter 8.

3.5 THE PATIENTS AND CONTROL SUBJECTS

In Patient Group

This was a group of 48 very severely brain injured patients who were receiving treatment at the Kemsley Unit after being found unsuitable for rehabilitation on other (conventional) units. Tables 3.d and 3.e provide the relevant data on this group.

Data on 29 of these patients are used in chapters 5,6,7,8, and 11, to illustrate single-case design methods, demonstrating the effect of specific behavioural techniques. Data from the other patients are included in other studies investigating (1) the effect of brain injury on discrimination learning, (2) aspects of attentional control, (3) speed and capacity for information processing, and (4) response to attention management procedures.

Their status as a **group** rests on the conventional measure for severity of brain injury (Russell 1971). In addition they all had behaviour problems which post dated such injury. There were however, considerable within-group differences. (1) The **nature** of brain injury varied producing different behaviour problems. This is not simply a case of traumatic v's other types of brain injury. Even within the traumatic group distinctions could be made according to the **type** of injury, whether it was **predominantly:**- frontal, temporal or brain stem. The type of injury could dictate many factors: (1) presence

or absence of physical sequelae,(hemiparesis, ataxia, or various other kinds of spasticity); (2) degree and nature of neuropsychological deficit (dysphasias, visuo-spatial deficits, or intellectual blunting). This information is contained in table 3.e..

In chapter 6 a crude division is made based on the **predominant** brain injury, defined by abnormality of the EEG or CAT scan. It must be emphasised however, that divisions of this kind must be regarded as somewhat artificial when they are applied to patients who have sustained a severe **traumatic** brain injury, where the **rotation** effects from sudden deceleration inevitably produce widespread, if often microscopic, brain damage (Oppenheimer 1968; Strich 1969). Such divisions are however, unavoidable for some studies. Where these exist it must be remembered that the groupings **cae**not be mutually exclusive, as might be desirable or possible in other kinds of experimental investigations.

TABLE 3.d

Characteristics of the in-patients who act as the "experimental" subject group in this study

MALES N=31

Closed head injury = 27

Haemorrage = 3

Encephalitis = 1

	Age	PTA	TSI	IQ
Mean	31.6	3.2	60.5	82.1
S.D.	14.4	1.6	34.4	6.5
Range	13-67	1-6	12-132	65-110

FEMALES N=17

Closed head injury = 15

Hypoxia = 2

	Age	PTA	TSI	IQ
Mean	30.4	3.23	64.7	89.1
S.D.	10.4	2.57	44.1	11.2
Range	19-55	.5-10	15-24	67 115

P.T.A.= Post traumatic amnesia.

T.S.I.= Time since injury.

Table 3.e

Number of Patients with Neurological and Neuropsychological sequelae in the in-patient group

Neurological		
Anosmia	=	9
Dysconjugate gaze	=	7
Visual field defects	=	8
Facial weakness	=	10
Truncal Ataxia	=	9
Hemiparesis	=	16
Tremor	=	7
Oral dyspraxia	=	13
Dysarthria	=	20

Neuropsychological		
Dysphasia	=	9
Dyspraxia	=	5
Dysgnosia (spatial)	=	17
(tactile)	=	6
*Severe memory Defect	=	15
*Blunted intelligence	=	21

* Severe enough to interfere with activities of daily living e.g., perseverative or confabulatory ideas.

Out Patient Group

The second group of subjects is composed of 32 **moderately** head injured patients (Russell 1971), who were attending as outpatients for neuropsychological assessment, either to monitor their recovery in preparation for a return to work, or in anticipation of psychological counselling or attentional retraining. This group of patients had all sustained a significant brain injury which had required hospitalisation for several days or weeks. They are distinguished from the inpatient group by having a much shorter length of PTA ($t=4.98$; $DF=78$; $P<0.01$). They were also different in the fact that their behaviour was generally much more appropriate although six patients were disinhibited, euphoric and in many respects sexually provocative. Fifteen patients also had a temporal lobe abnormality which gave rise to outbursts of argumentative or aggressive behaviour.

Twenty five of these patients were capable of employment on the open market and, in fact, 16 were actually employed at the time of attending outpatient clinic. Three patients had severe physical handicaps of the kind seen in a large proportion of the inpatient group. **The characteristics of this group are given in the method sections of the relevant studies.**

CONTROL SUBJECTS.

Three different groups of control subjects were used during this thesis.

The **first group** comprised 20 high grade **mentally handicapped** individuals, (mean I.Q= 62.25), who were inpatients at St Andrew's Hospital. The A.D.L. abilities of this group were, in many ways, similar to the in-patient brain injured group. These patients were therefore used to control the **intellectual** variable in the study of discrimination learning. Their data characteristics can be seen in chapter 10, table 10.1.

A **second group** of **orthopaedic** control subjects comprised 41 patients attending an orthopaedic outpatients clinic after sustaining various boney injuries, mainly in road traffic accidents. They act as a control group for the chapter 13 study on hedonic responsiveness.

The **final group** of control subjects consisted of 30 **normal** controls, selected from hospital staff, friends, relatives and members of the community, They were roughly matched to subjects in the patient groups, on the basis of occupation, age, sex and intellectual level. The characteristics of these groups will be given in the method sections of the relevant studies.

CHAPTER 4

SINGLE-CASE STUDY DESIGNS

Introduction

Clinical explanations of psychological problems and the effects of treatment usually begin with a careful case study of an individual patient (Shapiro 1970). A criticism of this method is that although often fascinating, it frequently fails to eliminate alternative explanations influencing the results of treatment and therefore "weakens" the conclusions that can be drawn (Kazdin 1980). In applied psychology, as in medicine, the therapist is required to provide evidence of the efficacy of his interventions and show that clinical changes are the result of a treatment effect rather than (1) spontaneous recovery, (2) a response to uncontrolled environmental variables, or (3) a charismatic therapist (Hersen and Barlow 1976).

Traditionally, attempts to measure or demonstrate treatment effects have utilised the classical group comparison experimental technique. This requires two or more groups to be compared on certain variables, one group being designated as the treatment group while the other(s) either receives no treatment or some comparison

treatment. Pre and post treatment measures are obtained and compared across groups. If the experimental group shows a significant improvement over the other group(s) the treatment is described as a success.

This traditional experimental/control group method may be well suited to classical forms of statistical analysis but does not lend itself to studies evaluating treatment outcome (Barlow and Hersen 1973). There are a number of reasons for this.

(1). Many clinicans object to witholding treatment from a no-treatment control group. This is based on the assumption that the treatment is going to have some beneficial effect (in which case one may question the need to evaluate its effectiveness in the first place). Although Hersen and Barlow (1976) regard this as an illogical objection, it is nevertheless very influential.

(2). Group Studies demand a collection of a large sample of patients who are homogenous with respect to:- symptom characteristics, age, length of illness and social background, criteria which may be very difficult to satisfy. Indeed, it has been argued that such groups are rarely truly homogenous (Kiesler 1966). This can mean that when specific questions about the effects of treatment in such groups are considered, many important aspects, pertaining to individuals in that group, are obscured because of the range of individual differences which patients present. As Marks (1972) stated, ten

patients who are homogenous for obsessive-compulsive neurosis may bring entirely different histories, personality variables and environmental situations to the treatment setting and will respond in various ways to treatment.

Shallice (1979) states that group studies in neuropsychology are particularly susceptible to the problem of heterogeneity. Not only are there problems of age differences, premorbid intellectual skills and extent of brain damage in patients forming a group but there is also the interaction between aetiology and the site of the main lesion. These factors compound the difficulty of making an inference about a particular neuropsychological function from group data.

Chassan (1967), pointed out that even if the experimental groups were assembled on the basis of homogenous characteristics, the outcome data frequently ignores such characteristics when describing the results. Consequently, the clinican often cannot determine which particular patient characteristics were correlated with improvement neither does he know to what extent a given patient under his care is similar to patients who have improved (or even deteriorated) within the context of an overall group improvement.

(3). The lack of true homogeneity points to another problem of group design - the **degree** of improvement that individuals in the group make to a particular treatment.

This can mean that the success of the experimental group may be due to a minority of patients making large changes in treatment measures, while the majority make only small changes or no changes at all. These individual variations are masked by the group average (Barlow and Hersen 1973).

Hersen and Barlow conclude that the results of group analyses are not readily translated into information that can be used by the practising clinician who is trying to answer a very fundamental question - "Is this treatment helping my patient?" Any evaluation of individual treatment effects must demonstrate not only a relationship between treatment and subsequent change in behaviour (Yule and Hemsley 1977) but also make an attempt to relate any differences in response trends to underlying functional mechanisms.

The Single Case Method

One way of evaluating behaviour change is the use of single case design (Barlow and Hersen 1973; Thomas and Blackman 1976; Hersen and Barlow 1976; Yule and Hemsley 1977; Kasdin 1980; Baddeley 1984). Single case methods employ an intra-subject replication design which has been extensively used in studies of behaviour modification (Baer et al 1968). This method examines treatment effects with individual patients using designs that do not require the patient to be subject to a 'guinea pig'

experimental approach and, to some extent, avoids the ethical problem of witholding treatment during a control phase. The effective application of single case methods involves the following stages.

1. Functional Analysis of Behaviour. As stated earlier, a functional analysis is an analysis in terms of the inter-relationship between environment and behavioural events. One way of establishing such a relationship is by means of a radical behavioural approach. Radical behaviourists, following the tradition of Skinner, have tended to emphasise the importance of identifying the variables (usually environmental) which control behaviour. Any functional relationships which might exist between the stimulus and behavioural response appear to be less important.

This does not appear to be a realistic method for examining the behaviour of a brain damaged group. Many of the variables controlling behaviour are organic rather than environmental. It is of some importance therefore, to know the workings of the 'black box' in order to be able to determine any organic factors which are likely to affect behaviour. If these are predictable and identifable by their relationship to certain types of behaviour, then not only will it help distinguish organic from non-organic behaviours, but may also improve treatment methods, such as the development of

neuro-chemical controls for behaviours which respond to organic as opposed to environmental variables.

2. Baselines. The end result of a functional analysis of behaviour is the identification of various target behaviours. These are considered to be in need of change because either they interfere with the process of rehabilitation or represent undesirable behaviours that would not be accepted by the community or long care residential facilities upon which a patient might depend for long term accommodation.

Once target behaviours have been selected there is an initial period of observation, involving the recording of the frequency with which the behaviour occurs. This period is defined as the **baseline** (Hersen and Barlow 1976). Traditionally, it has been designated as the **A** phase of the study and its primary purpose is to have a standard by which the effectiveness of a treatment intervention may be evaluated.

In clinical research it is generally recognised that before accepting a baseline measure as being reliable its stability (interpreted as minimum variation around an average level) and range of variability must be carefully examined (Krasner 1971; Risley and Wolf 1972; Leitenberg 1973). Sidman (1960) recommended that for stability, rates of behaviour should be within a 5% range of variability.

This poses significant practical and ethical problems to

many cllinicans who have to decide how long they need keep a patient under observation before starting treatment. As McNamara and MacDonough (1972) stated, "How long is long enough for a baseline?" Hersen and Barlow state that there is no simple response or formula to answer this question. Yule and Hemsley (1977) consider that the length of baseline depends mainly on the clinical condition itself. They criticise the idea that baselines must be collected until stability is attained. Even Sidman acknowledged that the applied clinical researcher, by virtue of his subject matter, with lack of control over both the behavioural history of the patient and over the optimum length of treatment, is at a distinct disadvantage and, as such, must take variability, "as he finds it and deal with it as an unavoidable fact of life". This consideration still ignores the fact that some conditions may follow a predictable pattern which is cyclical or otherwise non-linear (Yule and Hemsley 1977).

Even when clinical conditions are such that a long baseline is possible, the nature of the behaviour problem may not permit such a luxury. There is always the ethical problem of how long the clinical researcher can withold a treatment application. This is particularly so when the behaviour in question is considered dangerous, either to the patient himself (as in self injury) or to other patients or members of staff (when the treatment

involves aggressive or violent behaviour). In such conditions, an initial baseline period (the A phase) may be clinically difficult and experimentally unnecessary if one of three conditions applies.

(1) The first involves the possibility of carrying out a B-A-B design, which may still allow one to demonstrate the controlling characteristics that the treatment has over the behaviour in question.

(2) A baseline may be unnecessary if similar behaviours to the one in question have, on previous occasions, responded well when the same treatment proramme has been applied to other patients.

(3) Thirdly, 'no baseline observations will be necessary,' (Yule and Hemsley 1977) if reliable witnesses have observed a particular problem over a period of time and are able to report on its persistence, unacceptability, destructiveness, and even give a reasonable estimate of its frequency, on a day to day basis, or its duration over a period of time.

Hersen and Barlow describe an extremely variable baseline pattern as the one most frequently obtained during clinical research. This is true of the data in this study and few cases allow the luxury of long baseline measures to help establish stability. This particularly occurs when aggressive, unpleasant or inappropriate behaviours are being treated. Many of the patients entering the Kemsley programme were referred because of severe

aggressive tendencies involving actual physical assault, either on other patients or members of nursing or therapy staff. It must be appreciated therefore, that after some patients had been observed to respond successfully to certain behaviour modification procedures, it was considered clinically undesirable, unnecessary and dangerous to allow baseline measurements to continue.

The majority of behaviour programmes presented in this thesis do have adequate baseline data, according to the criteria of Barlow and Hersen (1973; - 3 data points), or Gelfand and Hartmann (1975 - 10-12 data points), to allow a reasonable amount of data for analysis. This always depended upon the clinical exigencies existing at the time.

Statistical Methods

In single case design the use of statistics is directed at establishing a significant treatment effect. In this thesis, significance was determined by a trend analysis, applying a variation of an analysis of variance (Meddis 1973). The response trends were basically of three types.

The first is described as a **type 1** trend, (fig 4.1). It shows a significant response trend (at least $P< 0.05$), compared to a relatively stable baseline. Figure 4.2) shows a **type 2** trend. This is where there is no evidence of a significant trend, because of a step-like drop in the frequency of the response. The significance of the trend is determined by a comparison of the baseline data

with that collected over the first 7 or 8 weeks of treatment (analysed using a students t). Analysis of the first 7 or 8 weeks of treatment was based on two reasons:

(1) many programmes run or are reliably recorded only for that length of time; (2) to show a treatment effect, without a significant (type 1) response trend, we would expect the change in behaviour to occur rapidly and be maintained for at least that period.

A **type** 3 trend (fig 4.3), is one which causes problems for statistical analysis because it has a variable baseline (either ascending or descending). Also, there may be little correspondence between statistical and clinical improvement. In such cases the relative merits of the case will be discussed.

Where data is organised on the basis of a group design conventional methods of inferential statistics will be used, the main emphasis being on the repeated measures ANOVA to analyse learning data.

Figure 4.1

TYPE 1 TREND

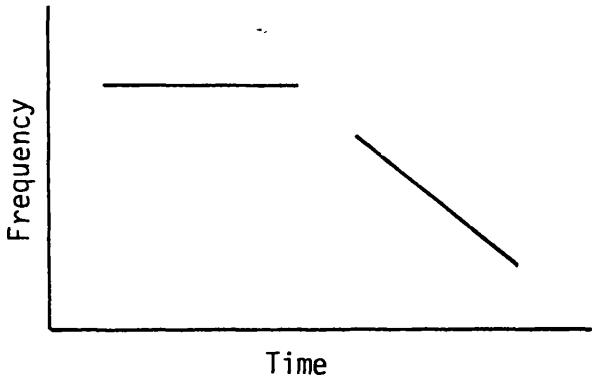


Figure 4.2

TYPE 2 TREND

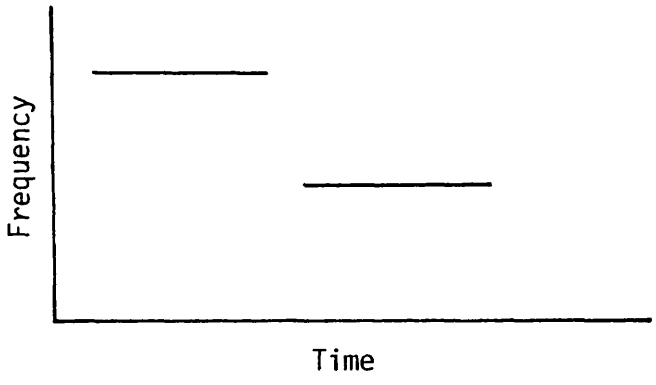
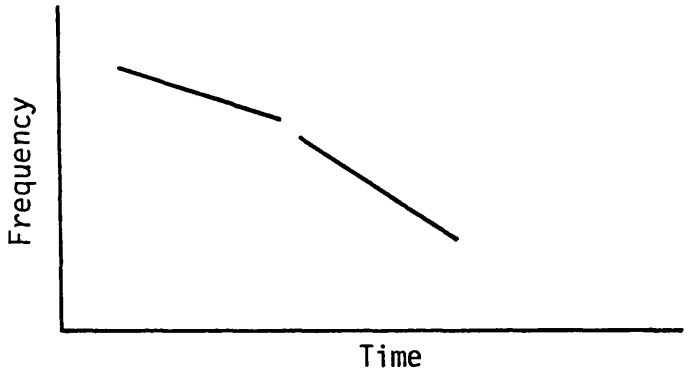


Figure 4.3

TYPE 3 TREND



Design of Behaviour Programmes

Reversal/Withdrawal Designs

There are essentially two types of experimental design that can be used to show the controlling effect of treatment variables during single case studies. They are called the "reversal" and "multiple baseline" design (Baer, Wolf and Risley 1968; Lazarus and Davison 1971; Campbell and Stanley 1966; Miller 1973).

Reversal Designs. Reversal usually involves the removal (withdrawal) of the treatment variable that is applied after baseline measurement has been concluded (Hersen and Barlow 1976). The A-B-A design is the prototype reversal design and one expects that a withdrawal of the B (treatment) phase after behaviour change has been successfully demonstrated, will effect a return in the frequency of the target behaviour back to baseline levels. If this is seen, it is taken to indicate a controlling effect of the treatment variable over the behaviour in question.

Leitenberg (1973) drew a distinction between the terms "reversal" and "withdrawal". He regarded the latter as a more accurate description of the procedure in an A-B-A or A-B-A-B design. Hersen and Barlow (1976) support this distinction because they point out that in behaviour modification studies, the usual procedure is to withdraw the treatment variable, rather than actually reverse it, which would mean exchanging one treatment variable for

another. In the series of studies to be discussed, both reversal and withdrawal procedures are used and will be described accordingly.

There are several problems which affect a clinician who is attempting to demonstrate the effectiveness of a specific treatment using an A-B-A design or one of its variants. If an A-B-A design is used, there is the immediate ethical objection that you are withdrawing a treatment effect that might have been beneficial to the patient and then effectively leaving the patient without any treatment at all. This problem is somewhat alleviated when an A-B-A-B design is used. Another problem is deciding when to actually withdraw the treatment variable. This may be decided by the limitations of time imposed by (1) the treatment setting, (2) the nature of the target behaviour (whether the behaviour is dangerous to others or producing self injury) and, related to this, (3) the attitudes of staff who are in day to day contact with a patient being treated (Johnson 1972).

The clinical researcher has never adequately answered such criticisms yet is still faced with the problem of demonstrating a treatment effect. This problem is compounded when, as is sometimes the case, a behavioural change is irreversible because the patient has acquired a new skill which alters his interaction with the environment or alters the behaviour of people within his

environment towards him, in such a way that they themselves cannot reverse. So great are the problems that Hersen and Barlow recommend that a clinical researcher using the withdrawal design must ensure that they have the following:-

(1) Full Staff Co-operation on an apriori basis.

(2) Some assurance that withdrawal of treatment will not lead to environmental disruptions (no injury to subject or others in the environment)

(3) The withdrawal period will be relatively brief.

(4) That outside environmental influences will be minimised throughout baseline treatment and withdrawal phases.

(5) Final reinstatement of treatment to its logical conclusion will be accomplished as soon as it is technically feasible.

Considering the problematic nature of the reversal design one is extremely fortunate when reversals happen 'accidentally'. In any clinical study, a programme may be discontinued for a variety of reasons. The patient may become physically ill and therefore not amenable to behavioural treatment; the member of staff carrying out the programme may be ill and therefore not available; human factors within the clinical environment may affect the programme, for example, lack of co-operation by nursing staff. These sometimes unavoidable intrusions into a behaviour programme, can often 'accidentally'

demonstrate the effectiveness of the treatment programme. Reference to figure 8.3(i) is an example of this. It shows the effect of a positive reinforcement programme, using tokens which could be exchanged for food, to improve the enunciation of polysyllabic words in a dysarthric patient who was making little effort during a conventional speech therapy programme. There is a considerable improvement in the clarity of his speech during the B (reinforcement) phase of treatment. Withdrawal of treatment, when the patient went home for one week, is associated with a rapid return to the A (baseline) measure with an equally rapid improvement following the reinstigation of the treatment variables. Figure 7.5(i), shows a similar effect when a positive punishment programme (using aromatic ammonia vapour) to prevent an exaggerated nose-picking habit, was stopped before a stable behaviour rate had been established (similar examples have been presented by Johnson 1972 and Hersen and Barlow 1976). In this case, the programme was stopped because the therapy staff were not entirely happy with the procedure used and, at the time of the programme, found it an inconvenient method to apply. Not only did the behaviour return to the baseline measure of the A phase but far exceeded it. This is often the case when a negative or positively punishing behaviour programme is stopped before the target behaviour has been properly consolidated at a new (low) level. Again,

following the re-application of the treatment procedure, the desired response was obtained.

An example of a reversal (as opposed to withdrawal) design (Leitenberg 1973) is shown in figure 8.3(iv). This describes an attempt to improve the content of conversation in a patient with a predominantly frontal lobe injury. The first A phase shows a consistently high rate of inappropriate conversation over a period of three weeks. This was followed by one week of selective reinforcement for appropriate speech whilst ignoring inappropriate conversation (see Ayllon and Michael 1959). The second A phase shows that this procedure was unsuccessful. It was realised that the patient was unable to exert the cognitive control over this impulsive conversational behaviour to deploy alternative conversational strategies. The C phase therefore, represents a reversal procedure where selective reinforcement was replaced by a procedure we termed, "cognitive over learning". The effect of this procedure can be seen by comparing it to the third A phase.

Multiple baseline design. The three examples already described show the effectiveness of treatment using withdrawal and reversal procedures. There remain however, certain procedures that cannot be withdrawn once they have been applied, either because it would be unethical to do so or because the behaviour change produces its own environmental change, with the result

that the original conditions cannot be reproduced.

This has been used as an argument to preclude the use of such single case designs in research and is regarded by some as a major limitation in experimental methodology (Bandura 1969). Bandura argues that because some therapeutic procedures produce learning that will not reverse when the procedure is withdrawn, it becomes impossible to isolate the treatment variable. To overcome this difficulty, some studies have withdrawn the treatment variable early in the B phase to effect a reversal (Kazdin 1973; Leitenberg 1973).

A more suitable method of overcoming such criticisms with this group of patients is the use of a multiple baseline design. This, in many ways, is an ideal strategy for studying the effects of treatment variables because withdrawal of treatment variables is not required in order to show the controlling effects of those particular techniques.

The rationale for the multiple baseline design was demonstrated by Marks and Gelder (1967) using a within subject multiple baseline strategy to evaluate the effect of electrical aversion for sexual deviation. Baer, Wolf and Risley (1968) followed this by showing an inter-subject multiple baseline, which they describe as being useful for comparative purposes when treating similar behaviour disorders. Both procedures are employed in this study.

In the intra-subject design, different behaviours are identified and measured over time to provide baselines against which changes can be evaluated. The inter-subject designs focus on only one behaviour and record changes that occur when the treatment variable is introduced. The cardinal rule, that only one variable should be changed at a time when proceeding from one phase to the next (Barlow and Hersen 1973) was observed throughout all single case procedures but was particularly important in the multiple baselines because of the extended nature of the behaviour programmes, often lasting for several weeks and sometimes running into several months.

Summary

The single case method is not without its critics. Bandura (1969) stated that although useful for investigating the control processes of behaviour it cannot be employed in studying learning phenomena in which certain experiences produce a more or less irreversible change in the behaviour of an organism. He also points to problems evaluating findings when large successive changes in behaviour occur rapidly and consistently across different subjects. This is a problem of statistical analysis; how to determine whether the magnitude of change produced by a given treatment exceeds the variability resulting from uncontrolled factors operating while the treatment condition is not in effect?

Hersen and Barlow (1976) are not blind to the limitations of single case design. In summarizing criticisms made by Keisler (1971) they point out that single case design has difficulty demonstrating that it is equally effective when applied to a range of patients with similar behaviour disorders or, whether it would be as effective if employed by a wide range of therapists. Another criticism describes the problem of specificity and whether a particular treatment effect would work if applied in a different environment or clinical setting. This last issue is singled out as having retarded the development of single case methodology in applied research and limited the study of a single case for any other purpose than the generation of hypotheses. Shallice (1979) suggests however, that the developments in this method over the last 20 years, "Far outweigh the undoubted problems to which it is subject".

SECTION 3

TREATMENT OF ORGANICALLY DETERMINED DISORDERS OF BEHAVIOUR

CHAPTER 5

SINGLE CASE STUDIES IN THE MANAGEMENT OF AGGRESSIVE BEHAVIOUR

Introduction

From the discussion of aggressive behaviour in section 2 we can assume that behaviour management may be influenced by two broad classes of aggressive behaviour. The first is neurologically mediated aggression, the second follows some form of behavioural learning. During rehabilitation, the **control** of such behaviour, to allow recovery to continue with as few complications as possible, is clearly very important. This section attempts to illustrate how, following severe brain injury, behaviour problems perceived and described as difficult, aggressive or disruptive, respond to one method of patient management:- the use of a **time-out programme** in the setting of a **token economy system.**

5.1 Neurologically Mediated Aggression

A patient's response to behavioural methods of treatment seems to vary according to the predominant or recognisable pattern of brain damage or neurological abnormality. This means that individual patients may differ considerably, both in their pattern of response and the necessary duration of treatment, depending on the **type** of injury they receive.

Neurological conditions mediating behaviour disorders require, in the first instance, that the precipitating variables producing such behaviour be brought under clinical control. This has proved to be relatively straight-forward for aggressive behaviours precipitated directly as a result of a neurological event (e.g. temporal lobe disturbance) which appears to be largely beyond the individual's control. It is much more difficult however, to control behaviours when frontal lobe injury has damaged the modulatory controls that normally inhibit many of the more extreme or inappropriate emotional responses.

Examples of these behaviours are presented in order to demonstrate:-

(1) The limitations of a "conditioning" programme if neurological variables (the **O** factor referred to previously) are not taken into account.

(2) The variability of response when neurological factors predominate.

(3) The success of a behavioural procedure which acknowledges the above factors and is continued long enough for a new response pattern to be consolidated.

Neurologically 'Precipitated' Aggression.

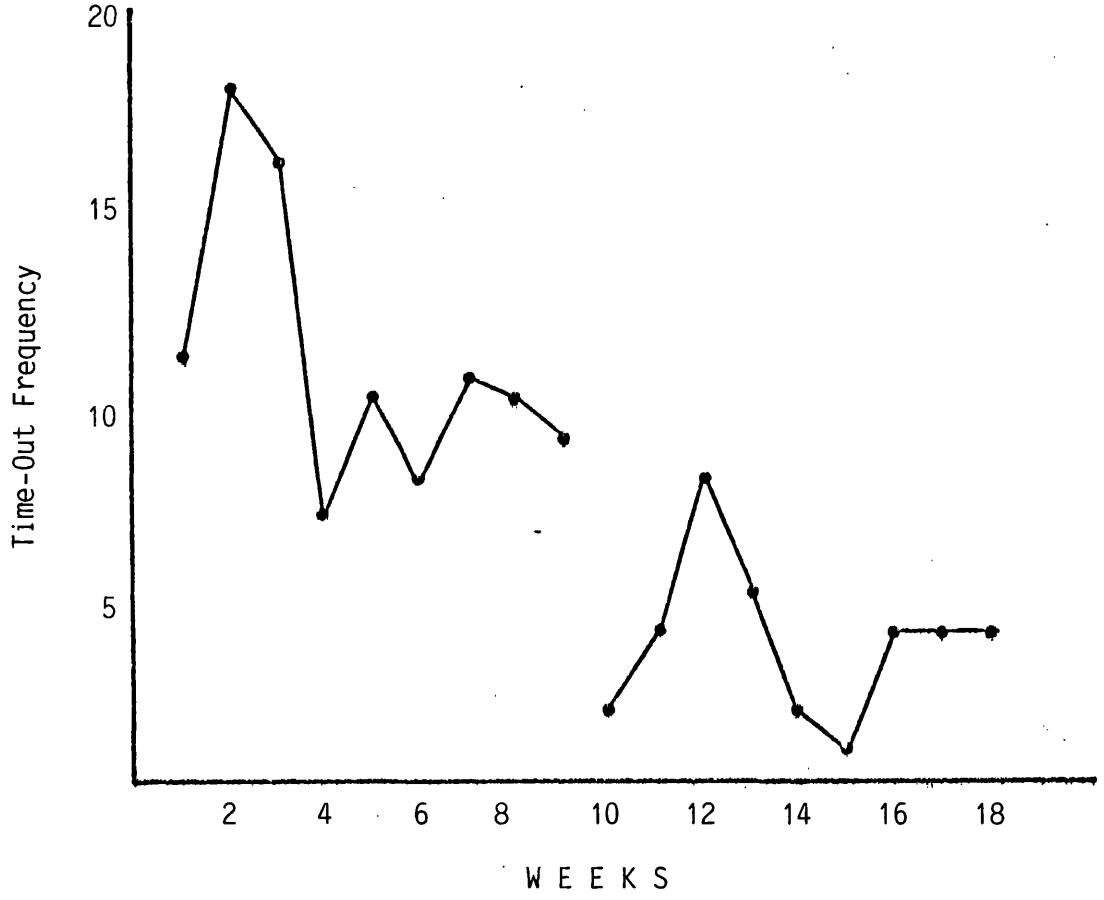
The result of a complex interaction between neurological and behavioural variables means that one cannot rely on **psychological** methods alone to control **neurologically** mediated behaviours. This is demonstrated in the following case studies of 3 patients with aggressive reactions pecipitated directly by a 'neurological event'.

Temporal lobe aggression. Figure 5.1 (i) illustrates the progress of a 29 year old patient who had sustained a severe head injury (PTA= 5 months) 5 years previously. Intellectual skills were moderately impaired but because he had sustained a brain stem injury in addition to cortical damage, many physical sequelae were prominent, including a right sided hemiparesis and a severe scanning dysarthria. In addition to this he had developed a very aggressive manner. From early in recovery his behaviour had been characterised by explosive, impulsive and unprovoked aggressive outbursts, which led to his early discharge from another rehabilitation unit.

From the time of his admission these behaviours were managed using only a **time-out room** programme. As the graph shows this did not produce any significant

FIGURE 5.1(i)

NEUROLOGICALLY MEDIATED AGGRESSION



(- Time-Out.No meds. -) (- Time-Out+ meds. -)

Comparison of 9 weeks of time-out for aggressive behaviour to 9 weeks using time-out + carbamazepine.

improvement. The frequency of aggression over the first nine weeks (pre-medication) showed some change but there was no evidence of a significant trend towards improvement ($F= 2.55$; $df= 1/7$). Shortly after admission, EEG studies showed that this patient had an abnormal left temporal lobe focus. Evidence from other sources (Mark and Ervin 1970), describing **non**-head injured patients, suggested that such abnormalities might be responsible for **initiating** aggressive behaviour. Consequently, an anticonvulsant drug (carbamazepine), used for the control of temporal lobe epilepsy, was introduced. The record of the second 9 weeks of treatment shows that the administration of carbamazepine was immediately followed by a significant (and permanent) clinical improvement in the patient's behaviour.

The shape of the graph suggests a "step down" in the frequency of aggression, typical of the **Type 2** trend referred to in the method section. The analysis for trend (during the second treatment phase) was not significant ($F= 0.15$; $df= 1/7$), but there was a significant difference between the means of the two samples ($t= 8.28$; $df= 16$; $P< 0.001$), which suggests that the introduction of carbemazepine to control the neurological variables produced the **treatment effect** rather than the behaviour modification procedure per se.

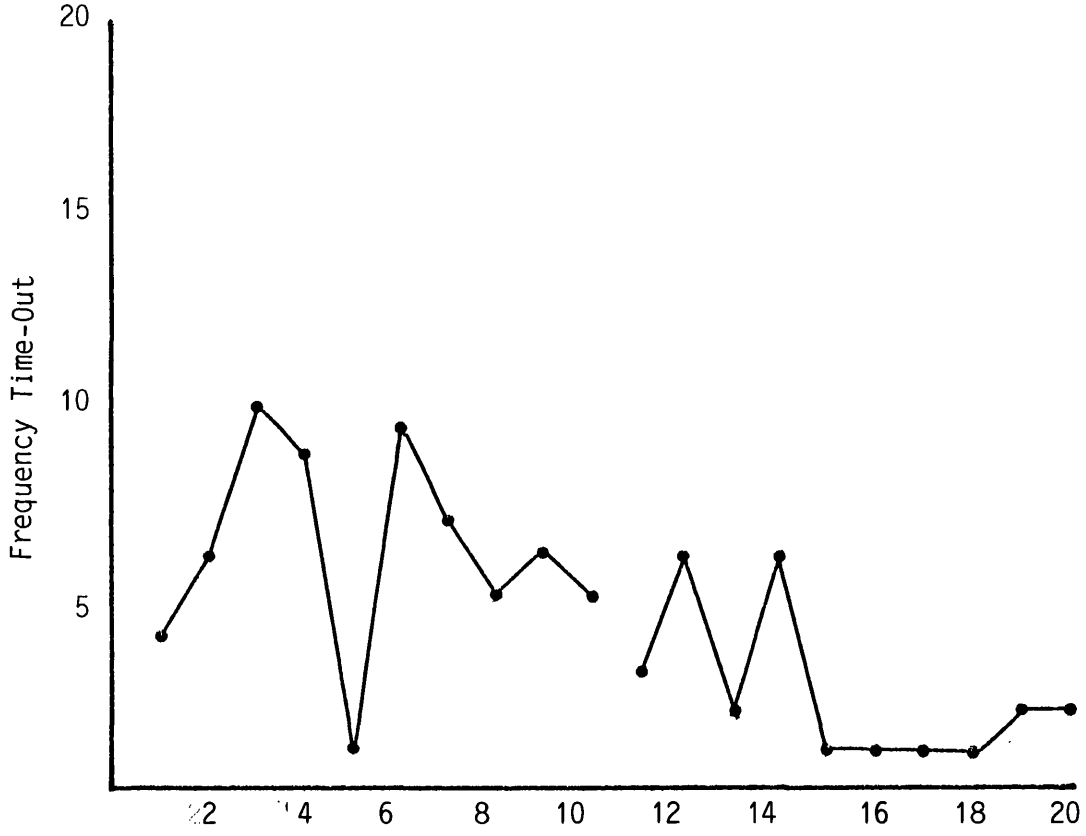
Temporal with frontal lobe damage. Figure 5.1(ii) illustrates a similar behaviour pattern. The aggressive behaviour occurred in a 21 year old patient who, following an intra-cranial haemorrhage 3 years previously, had both a temporal lobe EEG abnormality **and** a major frontal lobe injury. In addition to producing a severe intellectual defict, dysphasia and a left hemiparesis, his behaviour was marked by impulsive temper outbursts, made worse by a loss of control. This allowed his temper to escalate into behaviour that was so dangerous it caused the nursing staff at his referring hospital to initiate a threat of strike action.

For ten weeks after admission, treatment consisted only of a time-out room procedure. After this period he was placed on carbamazepine which, from clinical observation, reduced the frequency of his **unprovoked** aggressive outbursts.

The analysis of the frequency of time-out over the treatment period shows no significant trend, either for the behaviour management or the medication plus behaviour management phases of his treatment, ($F= 0.25$; $df= 1/8$, and $F= 2.25$; $df=1/8$ respectively). However, a significant difference was recorded between the means of the two treatment periods ($t= 2.83$; $df=18$; $p<0.02$), again indicating a **type 2** trend and suggesting successful intervention by carbamazepine.

FIGURE 5.1(ii)

NEUROLOGICALLY MEDIATED BEHAVIOUR



Comparison of 10 weeks time-out for aggressive behaviour to 10 weeks using time-out + carbamazepine.

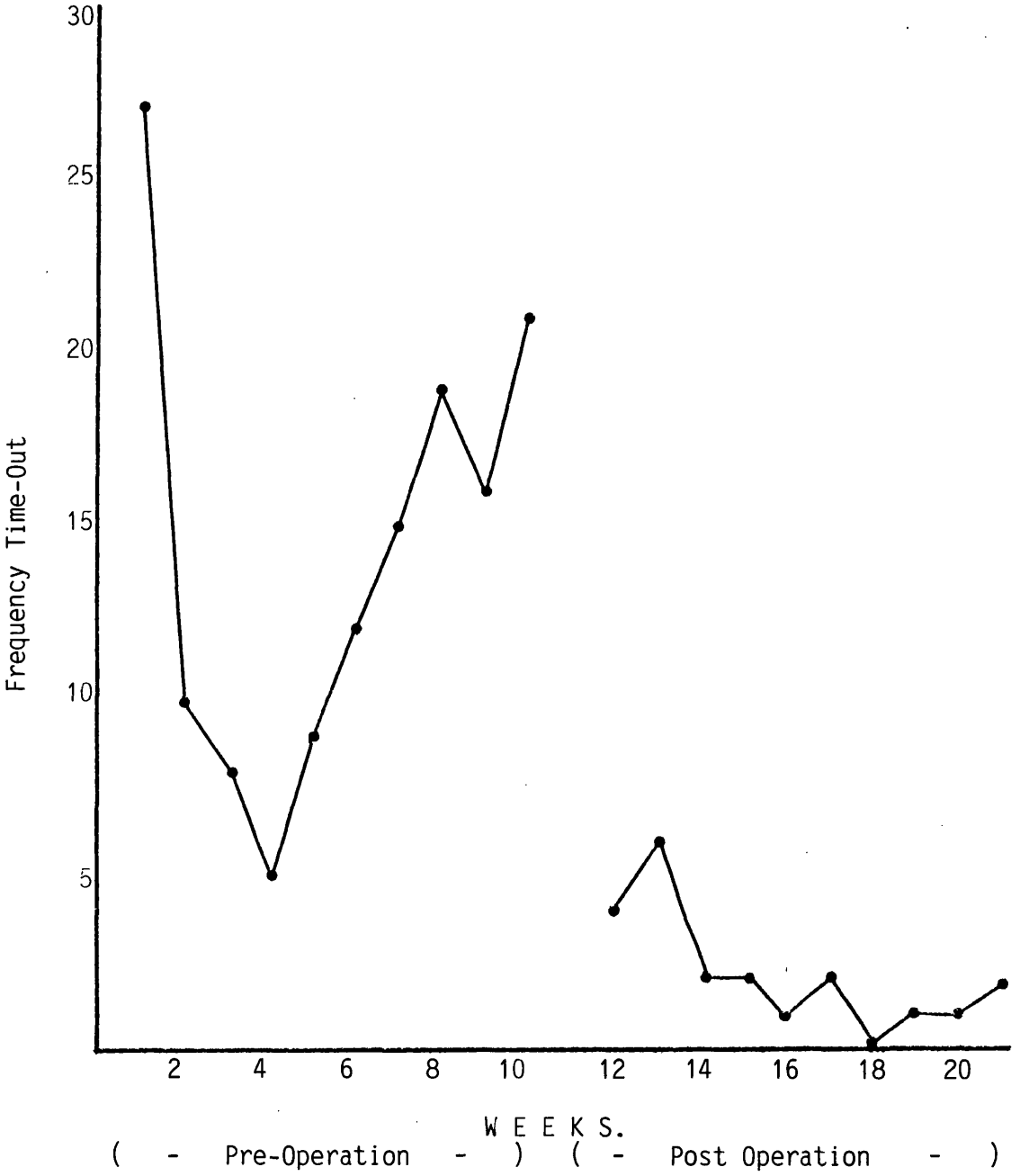
Temporal lobe aggression related to transient hydrocephalus. The aggressive behaviour pattern seen in figure 5.1(iii) occurred in a 19 year old patient who had sustained a severe head injury (PTA= 3 months) 28 months earlier. He had no serious physical sequelae but presented an almost complete anterograde memory defect and major intellectual impairment. From admission, the patient was known to have an abnormal EEG, suggesting that an electrical anomaly was responsible for his **unprovoked**, volatile outbursts of quite savage acts of aggression, nearly always followed by remorse.

This was treated from the onset by a combination of carbamazepine and behaviour management but with only a temporary reduction in aggressive behaviour and no evidence of a treatment effect ($F= 0.386$; $df= 1/8$).

It was then noticed that the patient had an intermittent discharge of cerebro-spinal fluid (CSF), which presented as an occasional runny nose. Neurological investigations showed that this intermittent CSF discharge was due to increases in intracranial pressure, forcing CSF through a tear in the dura. The build up of pressure within the skull, resulting in the CSF leak (which then reduced that pressure), probably precipitated a temporal lobe discharge, inevitably having a profound effect on behaviour. The patient was transferred back to a neurosurgical unit long enough for a ventriculo-peritoneal shunt to be inserted. In this case

FIGURE 5.1(iii)

NEUROLOGICALLY MEDIATED BEHAVIOUR



The effect of post-traumatic hydrocephalus on the incidence of aggressive outbursts during a time-out programme for aggression.

therefore, one must compare the frequency of aggression before and after the shunt operation to reduce intracranial pressure.

Although the were no trend effects in the data for the post-op phase ($F= 3.412$; $df= 1/8$), there is a highly significant difference between phases ($t= 7.23$; $df=18$; $p< 0.001$) indicating another type 2 trend.

Summary. The experience we have accumulated indicates that **established** behaviour disorders associated with EEG temporal lobe abnormalities, respond best to a combination of medication **plus** behaviour management, rather than either on its own. One frequently comes into contact with patients who have unquestionable 'epileptic' aggressive disorders and who are treated with carbamazepine on an outpatient basis and therefore, not subject to behaviour control. These patients frequently do far less well than patients who have had the opportunity of receiving medication in addition to behaviour management. Conversely, the first two examples in this section show the limited effectiveness when a purely psychological method of management (time-out) is used to try and control "epileptic" disorders of aggression.

Uncontrolled ('Frontal') Aggression.

The types of aggression described above illustrate what might be termed, **positive aggression,** precipitated **directly** as a result of an elecrical disturbance. When brain injury involves **frontal** areas however, inhibitory or modulatory systems may be damaged. Inappropriate behaviour may follow, not as the result of a 'positive' **precipitating** neurological event, but the lack of any control process which otherwise might **inhibit** it. In such circumstances the patient typically over-responds, reacting in a disproportionate way to quite trivial provocation.

Problems in the control of disinhibited out-bursts. When frontal brain injury is severe, outbursts of behaviour can occur which are difficult to control, even with a combination of medication and behaviour management. Figure 5.1(iv) shows the **clinical** failure of this combined approach which attempted to eliminate the disturbed behaviour of a 57 year old patient who, 20 months previously had sustained bilateral damage to the fronto-parietal cortex following a sub-dural haemorrhage. He was incontinent of urine and faeces and his walking was prevented by a dense left hemiplegia. He presented a major problem for nursing management because of his almost continuous shouting and attention seeking behaviour, constantly calling to the nursing staff for

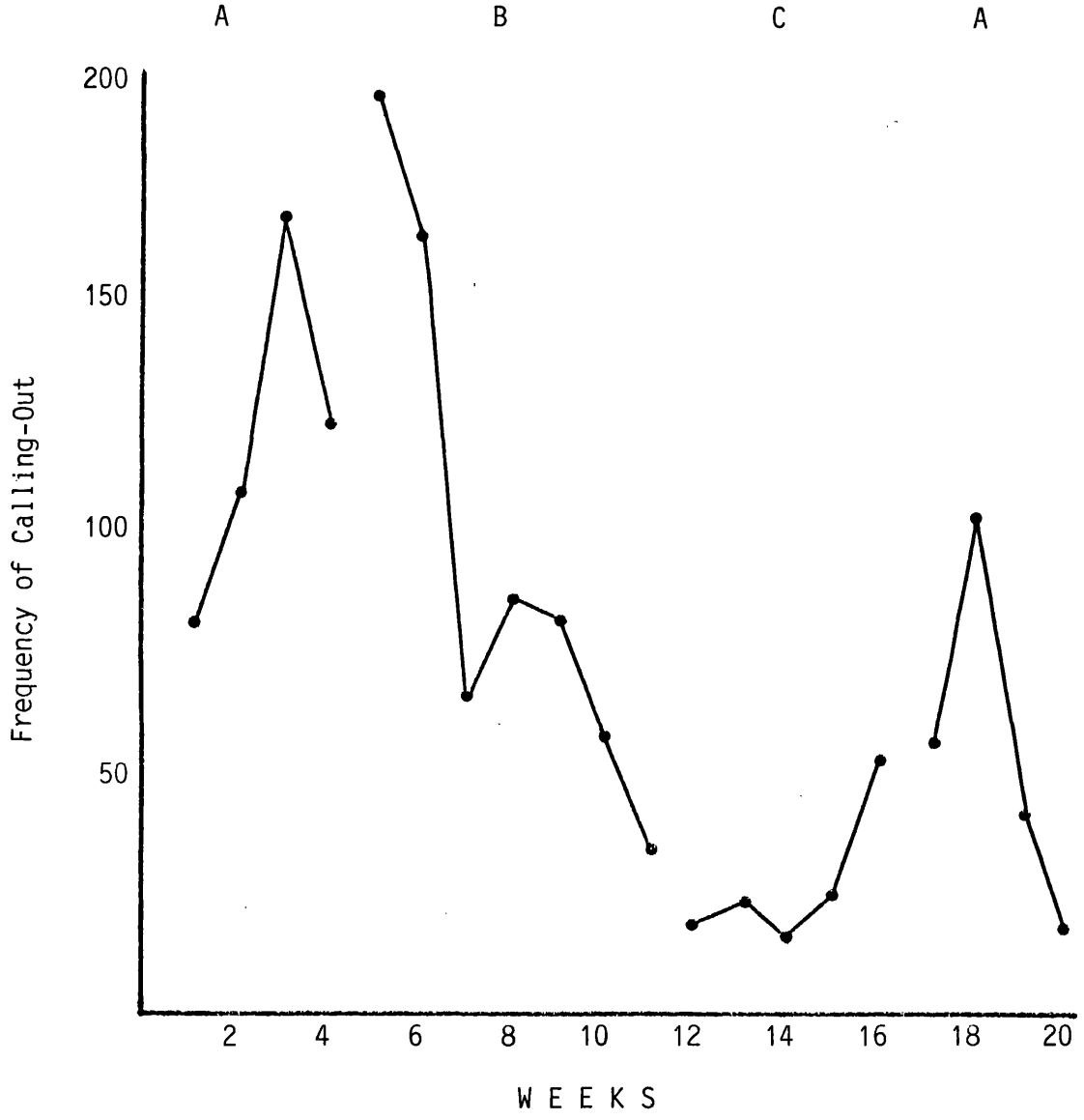
aid. This seriously prejudiced his future placement, making chronic psychiatric care the most likely alternative. The frequency of his shouting varied considerably throughout the treatment period, always remaining above a level considered satisfactory in clinical terms.

Treatment initially consisted of the time-out room for periods of five minutes, each time he shouted. This procedure made things worse because, on being taken to the time-out room he would scream abuse, continuing to do so whilst in the room, making it very difficult for us to allow him out without positively reinforcing such behaviour.

To avoid this problem a method of **positive punishment** was tried. This involved the contingent presentation of aromatic ammonia vapour to the nose each time he shouted. At first there was a worsening of his behaviour. In addition the staff were unhappy because the patient screamed that we were "torturing" him, which upset some of the nurses! The programme was maintained however, resulting in a sharp drop in the frequency of his shouting. This allowed us to return to the more expedient use of **TOOTS**. Unfortunately, this was probably introduced before the previous method of reinforcement had consolidated a reduced frequency of responding. It was also done without weaning the patient from one kind of reinforcement and onto another. The result was a sharp

FIGURE 5.1(iv)

NEUROLOGICALLY MEDIATED BEHAVIOUR



A combination of negative and positive punishment to control verbal outbursts in a patient with predominantly frontal-lobe damage.

rise in the shouting behaviour.

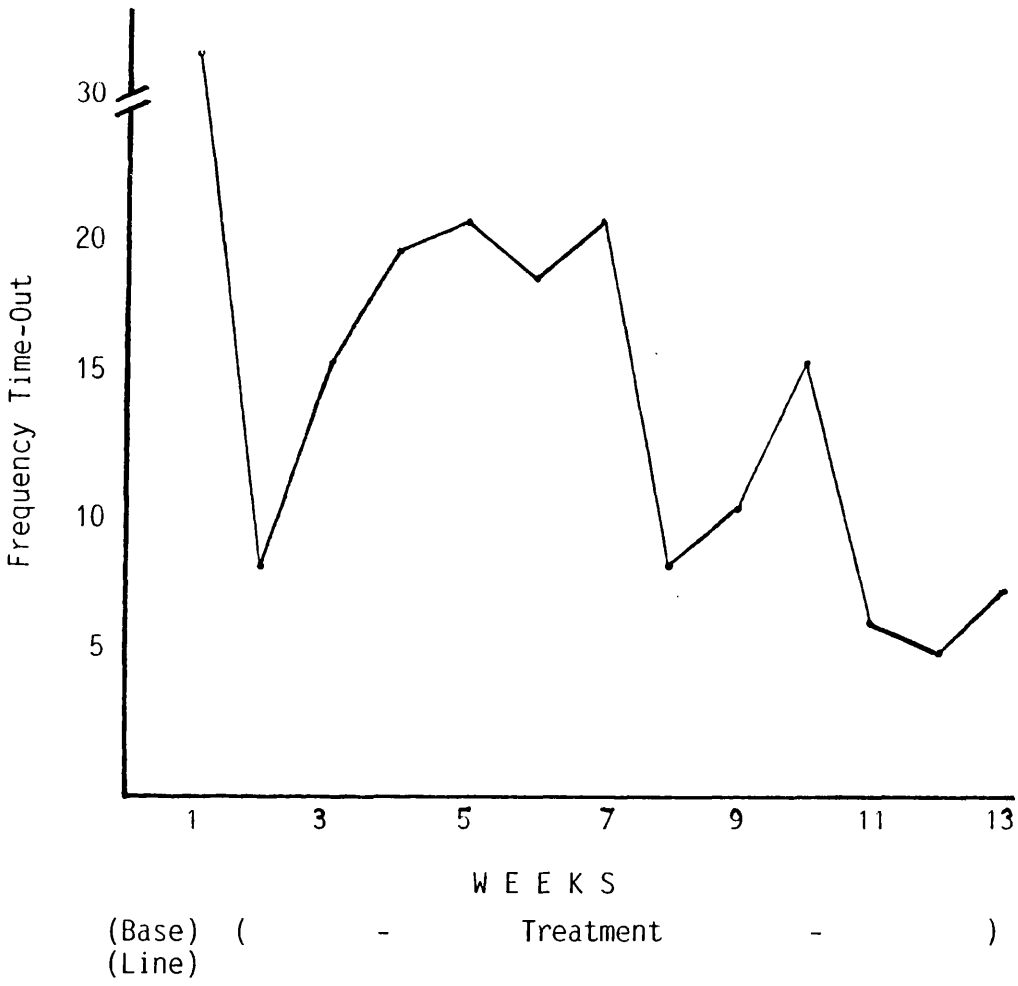
A significant response trend does emerge for the treatment period as a whole, ($F = 10.70$; $df = 1/18$; $P < 0.01$). This is characteristic of the type 3 trend referred to in the method section and not one which would be considered clinically successful.

The problem of maintaining control in a patient with frontal lobe injury. A similar problem of control in a 43 year old patient with frontal lobe injury is seen in figure 5.1(v). He had sustained a severe head injury (PTA unknown) 10 **years** before. Most of the intervening years, prior to this belated treatment attempt, had been spent on a chronic psychiatric ward. He was intellectually well preserved and the only physical legacy of the injury was a dysarthria and very mild left hemiparesis, somewhat exacerbated by an iatrogenic problem of tardive dyskinesia, produced by long term phenothiazine medication given to control his aggressive behaviour.

Although a time-out room programme had an immediate effect, significantly reducing the frequency of **abusive** behaviour, the control could not be maintained. A Student's t analysis, comparing the seven **day** baseline recording with the first seven **days** of the time-out room programme (table 5.a) confirms the **immediate** treatment effect ($t = 2.367$; $df = 12$; $P < 0.05$). This was not maintained however, and the abusive outbursts continued

FIGURE 5.1(v)

NEUROLOGICALLY MEDIATED AGGRESSION



Response to a time-out programme for verbal abuse by a patient with severe frontal lobe damage.

to increase in frequency for another 5 weeks before coming under the control of the treatment programme. Persistence by the nursing staff in applying this method led to a significant (type 1) trend towards improvement from the **third** week of treatment onwards (F= 10.92 df= 1/7; P< 0.05).

Table 5.a

	DAYS						
	1	2	3	4	5	6	7
Baseline	13	5	3	0	10	3	3
Treatment	2	3	1	2	0	0	0

Comparison of a 7 day baseline showing frequency of aggressive behaviour with first 7 days of treatment (t= 2.27; df= 12; p< 0.04).

Summary. The increase in frequency of aggressive behaviour after control is initially obtained is a common feature of frontal lobe disorders, illustrating how tenuous such control is with such patients, especially during the early stages of treatment. This is complicated by the fact that the **act** of timing-out such patients often, in itself, initiates a verbally abusive response which has to be ignored (TOOTS) by the nursing staff. The patients represented by figures 5.1(iv,v), were both receiving carbamazepine at the time of their treatment programme. The pattern of their response and the frequency of aggression suggests that such medication has little **direct** effect on what Lishman (1978) called the "loss of control syndrome" as opposed to the **positive** aggressive behaviours (produced **directly** as a result of a neurological event). This clinical assumption will be tested in a later section which deals with a group study of aggression management.

5.2 Learned Aggression

Learned aggression is distinguished by its purposeful nature, the fact that it nearly always has some reinforcement value and because it frequently occurs in ways that can be described as premeditated (such as in situations where there are few, if any, male staff available). The **learned**, as opposed to organic determinants of the behaviour, are primarily based on **clinical** observations, but it can be assumed that there is **no** evidence of an EEG abnormality, structural brain damage or any other clinical indication to suggest that the behaviour in question is **beyond the control** of the patient.

It must be emphasised however, that in the cases described, such behaviour was **not** a pre-injury characteristic. This fact must implicate the brain injury in some way, either by producing a tempory disinhibition of otherwise latent or controlled aggression, or through a process of 'conditioning' during emergence from coma, when disturbed, agitated or demanding behaviour gains attention from nursing staff, thereby serving to reinforce this as a style of behaviour.

A series of 6 case studies are presented to show the effects of different time-out procedures in the management of serious aggressive behaviour which, at the time of admission to the unit, had become an established part of the patient's post-traumatic behaviour pattern.

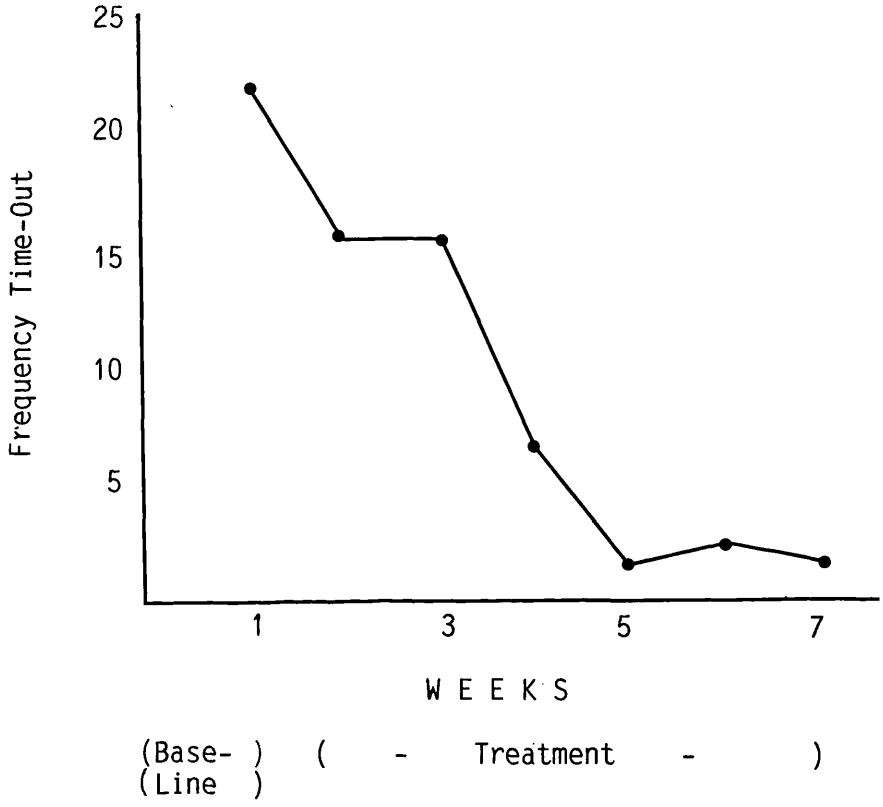
The absence of a pre-treatment (A) baseline in the figures must be considered in the context of the pre-admission history of all these patients (Yule & Hemsley 1977) and the consideration of safety requirements for other patients and staff.

Time-out room programmes for physically aggressive behaviour. Figures 5.2(i) and 5.2(ii) are typical examples of how aggression can prevent rehabilitation. The patient in figure 5.2(i) was a 27 year old male who had sustained a severe head injury (PTA= 2 months) 5 years earlier. He was dysphasic, had diminished insight, a right hemiparesis, reduced drive and had been receiving Phenytoin (anticonvulsant medication) prophylactically. This had progressively interfered with his intellectual abilities. During the previous 5 years he had remained in a wheelchair, incontinent of urine and faeces, receiving no structured rehabilitation. Earlier in his recovery, attempts by therapists to approach him resulted in him shouting abuse, adopting a threatening manner and attempting to punch them if they came within range. This behaviour was still very much in evidence at the time of his admission

The time-out room procedure produced a steady decline in the aggressive response. Clinical improvement was paralleled by a significant statistical (type 1) trend over the treatment period ($F= 83.03$; $df= 1/5$; $p<0.001$).

FIGURE 5.2(i)

LEARNED AGGRESSION



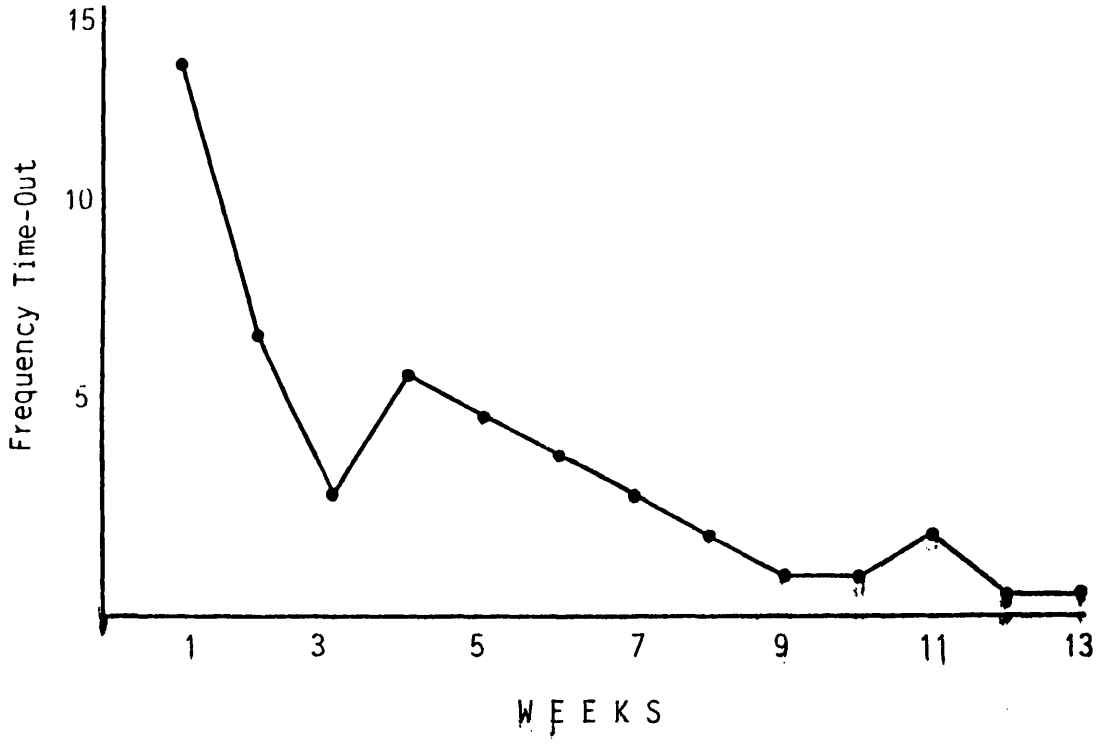
A time-out room programme to control learned aggressive behaviour.

His less aggressive manner allowed more therapy to be given and the patient made excellent progress towards greater mobility and independence.

Figure 5.2(ii) shows how another learned response - striking at staff with a blind persons walking stick, was eliminated using a time-out room programme. This 28 year old man had been blinded as a result of a head injury (PTA= 1 month) sustained 4 years earlier. In addition to his permanent visual deficit he had also suffered an injury to the hypothalamus, producing hyperphagia. Any attempt to deny him access to food resulted in a show of temper and an indiscriminate attempt to lash out with his white cane. He was obese and needed to have his eating habits and the aggressive response controlled before the Institute For The Blind would accept him for training.
The graph shows a rapid decrease in his aggressive behaviour ($F= 161.3$; $df= 1/6$; $p< 0.001$) which was maintained throughout the period of rehabilitation and after discharge. Probably one reason the reinforcement programme was so effective, was that it combined time-out with loss of tokens, meaning that he would probably lose part, or all of his next meal. In this way the programme capitalised on his main source of motivation and used that to help establish the control process.

FIGURE 5.2(ii)

LEARNED AGGRESSION



Time-out room programme to control an aggressive response to frustration.

Situational time-out and TOOTS procedures in the control of disruptive behaviour. Under the label "learned aggression" we can include various forms of disruptive behaviour. These require other forms of time-out procedure; **situational** and **time-out-on-the-spot.** Figures 5.2 (iii,iv) show how effective the simple procedure of situational time-out (removing the patient from the group, linked to a loss of tokens for that period) can be when applied to patients who are disrupting group activities.

Figure 5.2(iii) records the response of the patient originally described in figure 5.2(i). Once the time-out room procedure had controlled the physical aggression we were able to implement a second programme for disruptive behaviour. This involved moving him, in his wheelchair, from the room, with subsequent loss of tokens, each time he shouted or was otherwise a problem in group activities. Although the graph shows a good response during the treatment phase, a significant trend over the first 8 weeks was not recorded ($F= 4.17$; $df=1/6$). There was a difference however, between the twelve day baseline (table 5.b) and the first twelve days of treatment ($t= 2.69$; $df= 22$; $p< 0.02$) and this was maintained during the rest of the treatment period ($t= 8.98$; $df= 7$; $P< 0.001$).

FIGURE 5.2(iii)

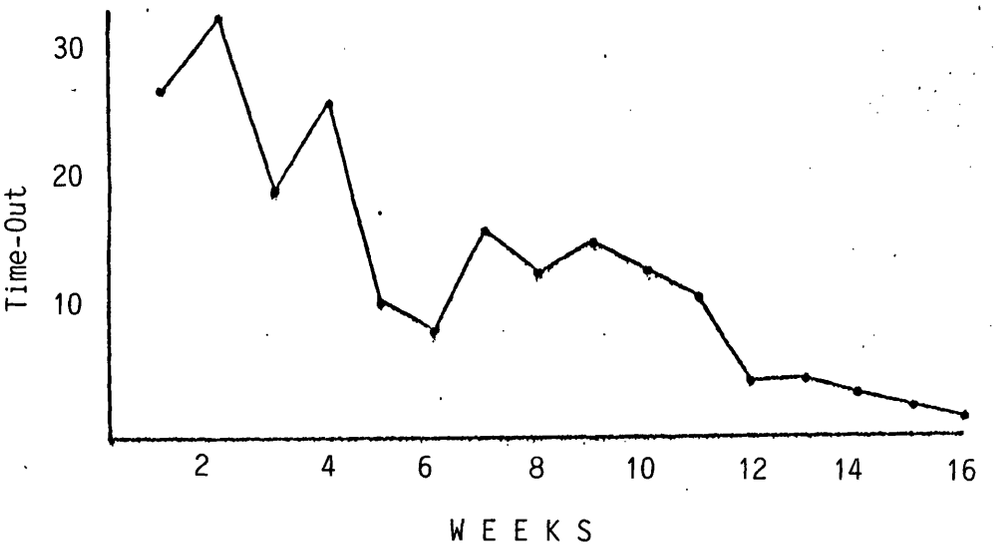
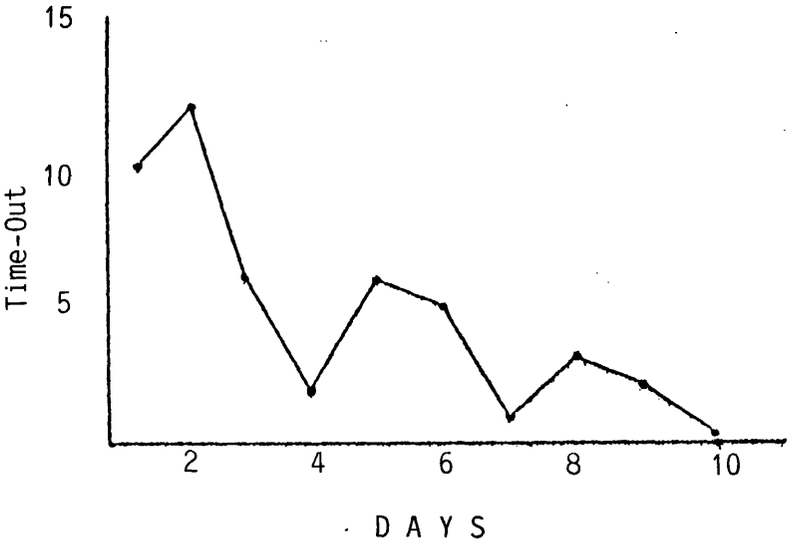


FIGURE 5.2(iv)



Two examples of the use of situational time-out to control abusive or attention-seeking behaviour that was disrupting group activities.

TABLE 5.b

	D A Y S											
	1	2	3	4	5	6	7	8	9	10	11	12
Baseline	4	5	5	3	3	4	4	4	9	6	4	9
Treatment	3	3	0	3	3	3	3	4	6	5	5	4

Frequency of disruptive behaviours during 12 day pretreatment baseline, compared to first 12 days of treatment (t= 2.2; df= 12; p< 0.03).

Figure 5.2(iv) shows a similar procedure in another patient who had sustained injury 5 years previously. Part of his behaviour involved repeated attention seeking responses, calling out to therapists in group activities, sometimes in a disinhibited and flirtatious way. He too was in a wheelchair, so the procedure applied with the previous patient was repeated.

Analysis of the first 8 weeks data shows a significant treatment effect and a type 1 trend, ($F=9.77$; $df=1/6$; $p< 0.05$). He was subsequently better behaved in group sessions and this allowed the therapy staff to involve him in more treatment activities, especially social skills training.

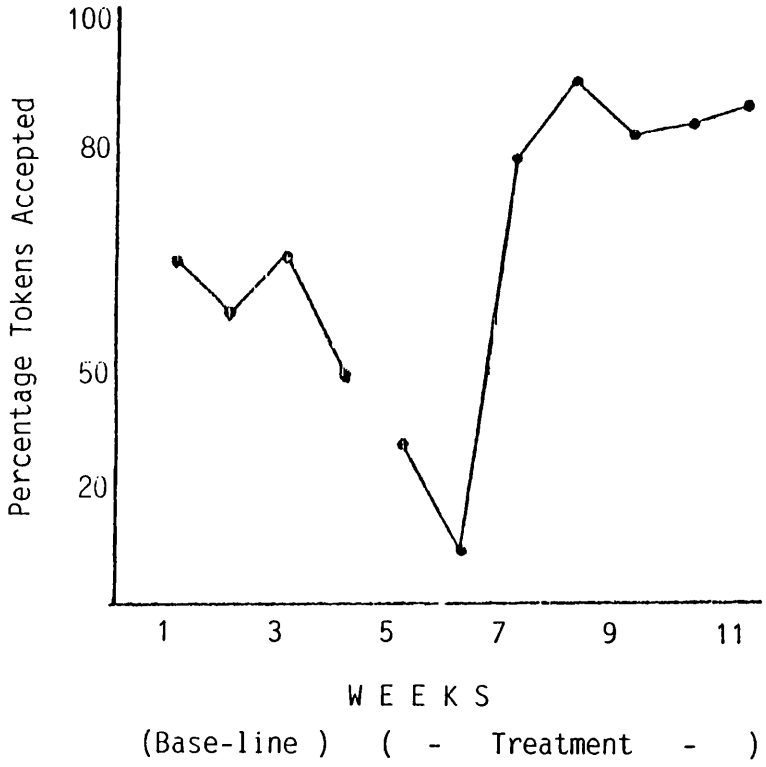
The use of a 'TOOTS' programme to eliminate attention seeking responses.

The next two examples are of Time-out-on-the-spot (TOOTS). Recording a TOOTS programme is not easy because of the way TOOTS is applied, i.e. not acknowledging the behaviour in any way. Usually TOOTS is applied by a number of staff on a continuous basis. To stop and record each instance would be very time consuming, clinically difficult and impractical. Some programmes have been recorded however, not by counting the frequency of TOOTS but by obtaining a measure of the behaviour which TOOTS is aimed at changing. Two of these are illustrated in figures 5.2(v,vi).

Figure 5.2(v) concerns a 22 year old patient who sustained a brain stem injury 4 years previously. The duration of PTA was not known but the physical sequelae of gross ataxia and oro-facial dyspraxia reflected the severity of the injury.

The behaviour problem was his refusal to accept payment of tokens earned. It was difficult to know whether this was due to a known (premorbid) trait of stubborness, or a feature of his organically determined psychiatric state (a Capgras syndrome). In any event, it inevitably led to a loss of privileges he would otherwise have received. It was noted that he appeared to derive pleasure from staff trying to persuade him to take these tokens. This

FIGURE 5.2(v)



A T.O.O.T.S programme:contingent withdrawal of social reinforcement & attention for refusing to accept earned tokens.

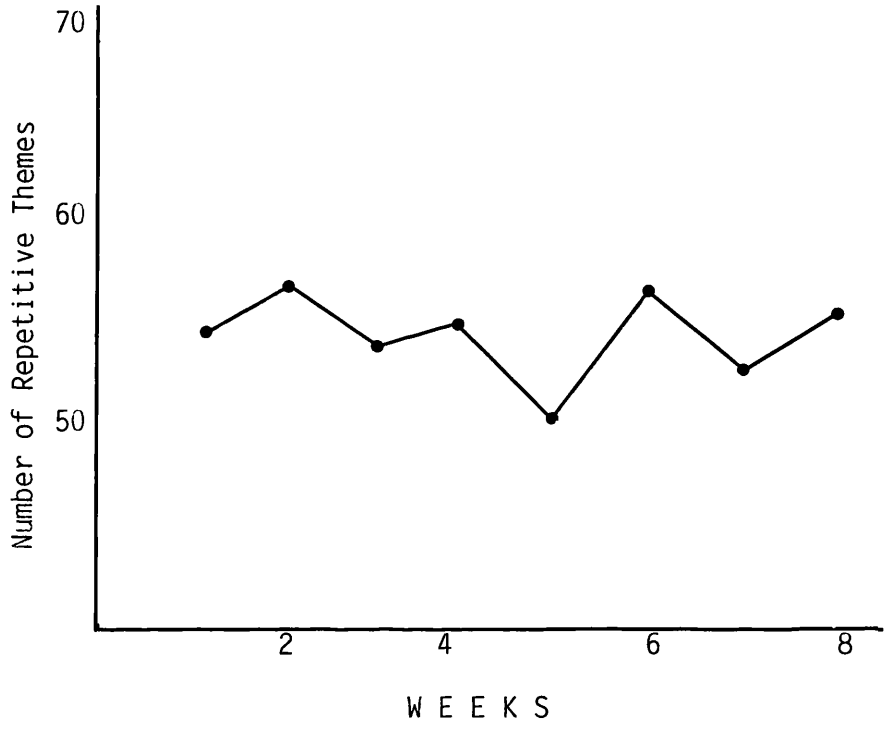
behaviour was therefore regarded as attention seeking, being maintained by the social reinforcement he was receiving at such times.

Withdrawal of such reinforcement was achieved by the TOOTS programme and the effect can be seen by the improvement in token earnings. Trend analysis for the treatment phase shows a type 1 trend ($F= 7.92$; $df=1/5$; $p< 0.05$).

A major limitation with TOOTS procedures is that very often, patients need to have a degree of social perceptiveness to realize that it is being applied. As such, patients with frontal lobe injuries, lacking not only self awareness but often in "other awareness", fail to respond to this relatively subtle form of behaviour modification. This is illustrated in figure 5.2(vi), which is included at this point to emphasise the limitations of a behaviour programme that fails to consider the **O** factors mediating behavioural learning.

Details of this patient are given in chapter 8 (fig 8.3(iv) when a major treatment programme is described. In brief however, the behaviour problem involved making a nuisance of herself by cornering people and engaging in conversation which was repetitive and inappropriate. Initially, treatment was based on selective reinforcement, as described by Ayllon and Michael (1959) to control the content of speech in a schizophrenic. This

FIGURE 5.2(vi)



The influence of severe frontal-lobe injury on a T.O.O.T.S programme for inappropriate conversation. The patient was unable to recognize the cues used in this type of reinforcement contingency.

lady however, had sustained a significant frontal lobe injury. Her ability to perceive the effect her behaviour was having on others was severely blunted, consequently she lacked many important social skills.
The graph shows that the TOOTS programme, designed to ignore inappropriate comments and socially reinforce others, did not effect a reduction in her inappropriate approach behaviour or modifying her type of conversation ($F = 0.052$; $df = 1/6$).

Summary. These case studies indicate that, in the absence of neurologically mediating factors, behavioural methods alone are sufficient to control and eliminate aggressive and disruptive behaviours that have, as their origin, a severe brain injury.

Conclusions

The above studies serve to illustrate that the **pattern**, as well as the frequency of behaviour, differs according to the presence or absence of neurological mediators and also the type of brain injury. There is however, some consistency in their response to behavioural methods of treatment. In order to establish the generality of this procedure as a method of patient management, the next chapter presents a group study analysing the treatment outcome of all patients admitted to the rehabilitation programme over a four year period, who have a history of aggressive behaviour **post-dating** their injury.

CHAPTER 6

A GROUP STUDY OF THE MANAGEMENT OF AGGRESSION

The single case studies presented in chapter 5 suggest that the outcome of treatment for post traumatic aggressive behaviours will depend upon the degree to which neurological factors are involved in such aggression. Behaviour associated with frontal lobe damage or temporal lobe (EEG) abnormality, will respond differently to treatment if compared to aggressive behaviours which follow brain injuries of **equal severity**, but without evidence of the residual focus of damage seen in the other two groups.

The following hypotheses can be made:-

Hypothesis 1. The frequency of aggressive or disruptive behaviour will vary according to the predominant neurological abnormality, evidenced by EEG or brain scan.

Hypothesis 2. The behaviour disorders included in this study will, in general, show a significant response to behavioural treatment, using a time-out room programme.*

Hypothesis 3. This treatment effect will occur, irrespective of the nature of neurological involvement, but may differ according to the neurological abnormality known to be present.

*** The presumed treatment effect cannot always be measured against a baseline or control group. It would appear reasonable however, that the length of time these behaviours had existed following injury (at least two years), and the fact that they were severe enough to warrant specialist treatment, would satisfy the criteria for baselines which Yule and Hemsley (1977) described as being appropriate for clinical research.**

METHOD

Subjects

The first thirty patients admitted to the Kemsley Unit because of aggressive or disruptive behaviour, were retrospectively divided into three groups according to their **objective** neurological and behaviour characteristics (figures 6.a, 6.b). This division was made on the basis of information contained in their medical notes on admission (C.T.Scan and/or EEG reports), or obtained as part of routine investigations during treatment.

In one sense the subjects in this study are an unselected group because they comprise all the patients who were referred to this unit over a period of four years with a history of aggressive behaviour. In another sense the patients were highly selected because, as a group, they

represent the most severe cases of their type and were all referred because their management had proved too difficult for conventional rehabilitational units. Such behaviour was not known to be a feature of their premorbid personality or behaviour pattern but had been in evidence since their brain injury, causing major management problems.

Group 1, (n=10), included patients with temporal lobe EEG abnormalities. Their progress in rehabilitation was being disrupted by occasional (sometimes frequent) outbursts of aggressive or disruptive behaviour that was unprovoked , poorly directed and usually short lived. This group were all receiving carbamazepine (400 mgs bd), on the advice of a consultant neuropsychiatrist.

Group 2, (n=10) consisted of patients who had brain scan reports describing evidence of frontal lobe injury. The presence or absence of such injury was the only criterion. Neither the time from injury when the scan was recorded nor the extent of such damage was taken into consideration.

The behaviour of these patients had also been observed by our rehabilitation staff. It was described as being disinhibited, with little or no control over emotional responses, resulting in a disproportionate reaction to frustration. They also presented other behaviour characteristics associated with frontal lobe injury (such

as described in chapter 2).

Group 3, (n=10) did not have evidence of a temporal lobe EEG abnormality or structural damage on a CAT scan, but displayed aggressive behaviour of a kind that was described by ward staff as attention seeking, threatening, purposeful and sometimes premeditated. Because there was no evidence to suggest that this was a prominent feature of their **premorbid** behaviour it seems reasonable to describe this as a learned behavioural response, developed since and as a consequence of brain injury.

Table 6.a suggests that the three groups cannot be distinguished on the basis of the severity of injury (measured by post traumatic amnesia). Four patients were not head injured (three intracranial haemorrhage and one anoxia) but it is not thought that this made any appreciable difference to the results.

Apart from carbamazepine the only other medication that might be considered capable of having an effect on behaviour was baclofen (20 mgs bd), given to three patients in group 1 and one patient in each of groups 2 and 3,and propanolol (640mgs bd), given to one patient in group 1 and two patients in group 3. Phenothiazines or other major sedative drugs were not used.

A similar number of patients in each group had the kind

of neurological and neuropsychological sequelae associated with severe brain injury (see table 6.b) e.g. hemiparesis, wheelchair mobility, speech disorders and intellectual defects. These were not thought to influence the treatment outcome in any way. Time since injury was such that spontaneous recovery could not be expected over the time these data was collected. It must be remembered that most of the patients had remained this way for at least two years and their behaviour was, if anything, deteriorating.

TABLE 6.a

Data of subjects in the three groups

	Grp 1	Grp 2	Grp 3
Males =	7	8	8
Females =	3	3	1
Age X =	25.8	35.1	25.2
(Range)	17-30	19-57	19-43
*P.T.A X =	3.0	3.2	3.1
(Range)	2-5	2-4	2-12
!T.S.I. X =	41.0	145.0	65.6
(Range)	28-72	24-108	29-132
Trauma	9	8	9
"I.C.H.	1	2	0
(Other)	0	0	1+

* Post traumatic amnesia; (months): Head Injuries Only.

! Time since injury (months)

+ Anoxia

" Intracranial Haemorrhage.

Table 6.b

The main neurological and neuropsychological sequelae presented by the three groups.

	Grp 1	Grp 2	Grp 3
Anosmia	3	2	3
Visual field defs	3	3	1
Dysconjugate eye m'mt	3	2	2
Facial weakness	4	1	2
Truncal ataxia	2	0	2
Hemiparesis	4	6	4
Incontinence	2	2	0
Dysphasia	2	2	3
Dysarthria	5	5	3
Severe memory impmt	7	4	3
Major cognitive deficit	6	8	5

Procedure

The patients were all taking part in the brain injury rehabilitation programme described in chapter 3. They had all been discharged from conventional rehabilitation units because of their unmanageable behaviour.

The method of behavioural control employed in this study was a time-out room procedure: following any aggressive incident, the patient was placed in an empty (locked) side room for intervals of 5 minutes or multiples of 5 minutes if behaviour continued to be disturbed. Each episode of time-out was recorded in a special time-out book, together with the reason for time-out and the actual times the patient was placed in the room and taken out. Each entry was later countersigned by the neuropsychiatrist or neuropsychologist attached to the unit.

For the purpose of this study the data for the first twenty five weeks following admission was used. This was not an entirely arbitary cut-off point; it was chosen to demonstrate the length of time such behaviours can take to respond to treatment and also the variability of response within that time. A longer period of behaviour recording was not possible because of the increasing drop-out rate that takes place after this time (due to patients being discharged from treatment).

Patients were not allocated blindly to different groups. They were selected according to their behavioural

characteristics and/or information contained in their case notes describing neurological abnormalities such as: (i) EEG's which were considered (by a neuropsychiatrist) on clinical grounds, to show epileptogenic activity, (ii) brain scan evidence of predominant frontal lobe injury with corresponding behaviour traits. In the circumstances it would have been difficult, although not impossible, for someone other than the experimenter to have allocated patients to different groups. While accepting this as a weakness in the design, it must be realised that (i) all the patients fulfilling the criteria for this treatment were included, (ii) the selection criteria were quite objective and familiar to all members of the treatment team, thus reducing the possibility of experimenter bias.

RESULTS

The frequency of aggressive behaviour in the three groups was determined by counting the number of time-out room incidents for each patient over a twenty-five week period. Table 6.c shows the mean and standard deviation for the three groups of patients, using observations made at every five weeks, to illustrate the pattern of change and relative frequency. (The same measures of central tendency were made for each week during treatment and the pattern was essentially the same. Data are presented at this interval to facilitate ease of comparison).

Table 6.c

Mean and standard deviation data sampled at 5 week intervals throughout treatment

		Grp 1		Grp 2		Grp 3	
		X	SD	X	SD	X	SD
	1	11.8	7.2	17.9	23.5	5.7	5.7
W	5	9.7	15.5	9.6	7.2	3.0	2.3
E	10	5.8	6.1	4.1	4.4	0.3	0.6
E	15	3.7	5.6	4.9	5.3	1.4	2.1
K	20	1.8	2.9	3.4	4.2	2.0	3.2
	25	1.8	1.7	3.7	3.6	0.9	0.6

To test the hypotheses stated in the introduction a 2 way ANOVA with repeated measures over one factor was used. As table 6.c suggests, there was considerable variation of the individual frequencies of time out between the three groups. To overcome the marked skewness of the data a logarithmic transformation, $Y=\ln(1+X)$, of the frequencies was made before the analysis. The ANOVA results are given in table 6.d.

Table 6.d

Repeated Measures ANOVAR Comparing Frequency Of Aggression And Response To Treatment Between The Three Groups.

	SS	DF	MS	F
Between S's	363.02	29		
Between Gps	97.94	2	48.97	4.988*
S's W'thn Gps	265.08	27	9.81	
R'pted M'sres	118.84	24	4.95	11.428 **
Interaction	18.39	48	0.38	0,88
Error-W	280.75	648	0.43	
Total	780.96	74		

** p 0.01

* p 0.05

HYPOTHESIS 1. A significant between groups difference, ($F=4.988$; $df=2/29$; $p< 0.05$), confirms the claim of the first hypothesis. Further analysis of this difference was made using a one way analysis of variance to compare the frequency of all time-out incidents over each five week period throughout treatment. The same logarithmic transformation was used to overcome skewness . Table 6.e shows the values of **F** for the five periods of analysis. A significant difference separates the groups throughout treatment ($p< 0.01$). This would not necessarily be predicted from the data in table 6.c or figure 6(i) which

uses **median** values to correct for skewed data. Consequently, the difference must reflect the individual differences in the patients' response to this kind of treatment.

TABLE 6.e

A comparison of between group ratios showing that the three groups record a different frequency of aggressive behaviours throughout treatment

Weeks	F	DF	P
1-5	11.91	2/74	0.01
6-10	24.38	"	0.01
11-15	12.88	"	0.01
16-20	7.74	"	0.01
21-25	11.03	"	0.01

HYPOTHESIS 2. If the argument for a clinical baseline, presented in the introduction is accepted, then the repeated measures analysis of the combined scores for the three groups shows a significant response to treatment ($F= 11.428$; $df= 24/48$; $p< 0.01$).

The length of time such treatment might need to continue was determined using a students 't', to assess the departure of each group mean from zero. Analysis was made for each five week period to indicate the point of

extinction of the behaviour problem. The results displayed in table 6.f suggest that although the graph in figure 6(i) gives evidence of a significant trend (treatment effect), it is only after 20 weeks that one of the groups (group 3) shows a non-significant result, indicating that the **group** had finally learned total control of their disruptive tendencies.

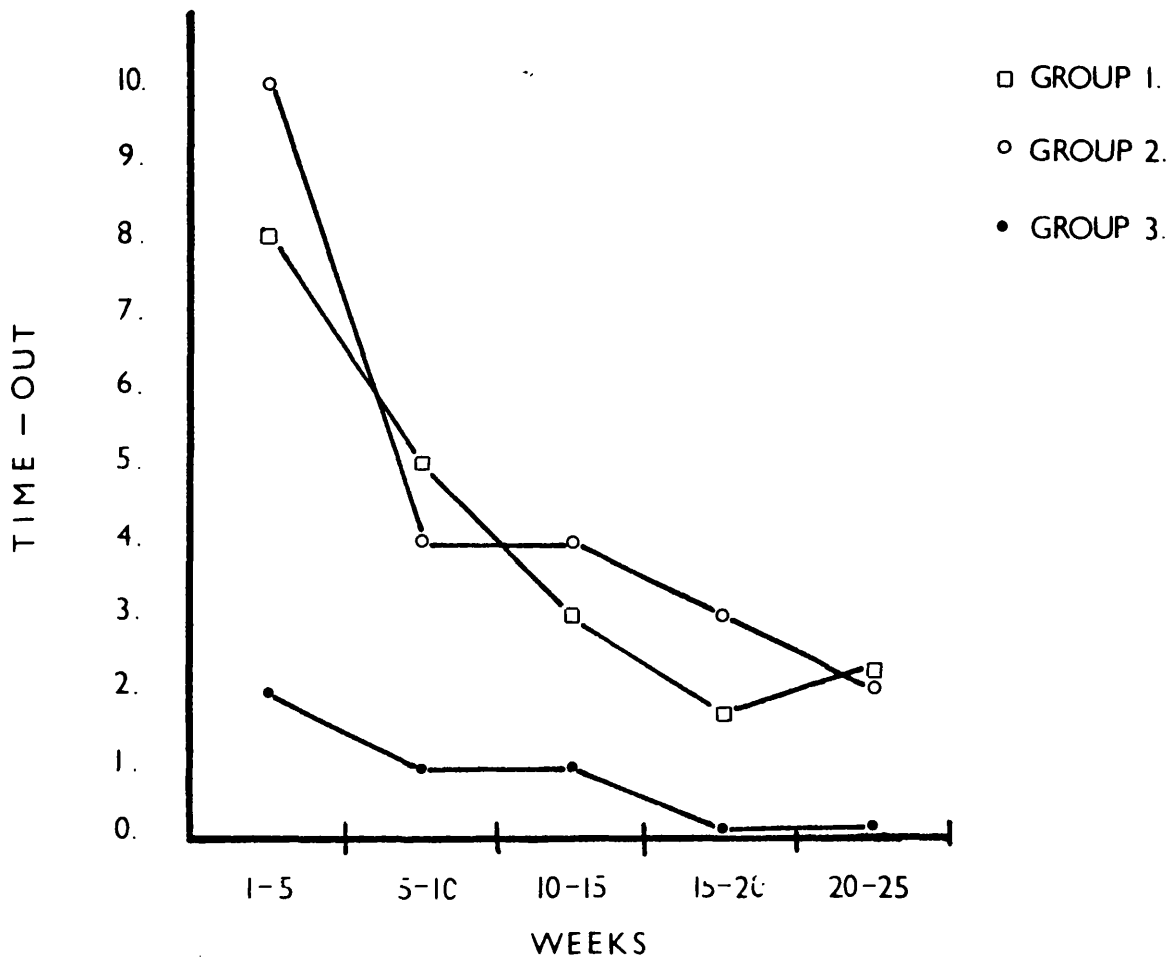
TABLE 6.f

Difference of the mean group score from zero to indicate the elimination of aggressive tendencies

	VALUES OF t			
WEEKS	**GRP1**	**GRP2**	**GRP3**	
15-20	**5.63****	**6.97****	**4.06****	
21-25	**4.81****	**4.97****	**3.85 ns**	****= p 0.01**

HYPOTHESIS 3. There was no support for the idea that the three different groups would show different response trends over the treatment period (F= 0,88). The idea that different trends may emerge was predicted from clinical observations of **individual** patients undergoing treatment. These individuals were known to have particular kinds of neurological involvement, assumed to be responsible for their variable and unpredictable behaviour during the time-out programme. This finds some support in the data

FIGURE 6.1



Median scores showing the frequency of "Time-Out" incidents over each five week period throughout treatment.

of groups 1 and 2 (table 6.c which show considerable variation, indicating extremes of behaviour for **individual** patients. However, this did not result in a between **groups** difference at any point in the treatment programme.

DISCUSSION

It cannot be said that these are mutually exclusive groups in terms of their neuropathology. A patient with a temporal lobe focus on an EEG could also have frontal lobe damage which would not be revealed on a CAT scan and vice-versa. Similarly, the group described as having "learned" behaviour has obviously sustained extensive, if microscopic lesions (Strich 1969) which could affect frontal or temporal areas or the connections between them. It does appear from the evidence however, that one can discriminate between these three groups of patients in terms of the overall frequency of their aggressive behaviours and that this frequency can be associated with what is known about such patients' predominant neurological abnormality.

It is clear that whether the behaviour problem be a consequence of an electrical (epileptic) abnormality, frontal lobe disinhibition or a learned characteristic, a successful outcome can be expected using behavioural methods, or a combination of behaviour management with medication, (especially when medication alone has been

unsuccessful).

Although the analysis failed to show an interaction effect, to indicate different response trends **between** the three groups, notice should be taken of the variability of behaviours **within** groups. Our clinical observations would suggest that many **individual** patients who have a **learned** aggressive response, respond faster and more reliably to a time-out procedure than patients with obvious neurological abnormality. This is not necessarily because they start out with less frequent aggression. As the group data might suggest, individual patients in group 3 often equal or exceed patients of the other groups for frequency or severity of aggression. It does appear however, that when there is no obvious neurological abnormality either producing the behaviour or interfering with associational learning (time-out), then response to treatment can often be better. In this respect, it is interesting to note that although the groups with abnormal EEG's or frontal lobe damage tended to have much higher frequencies of aggression than patients with learned aggressive disorders, their pattern of response to treatment was fundamentally the same, although they take longer to learn behaviour control than the patients of group 3.

The important feature of this treatment programme is **not** that difficult patients, so long after treatment, can learn control, but the **length of time** treatment of this

kind needed to continue to produce this control, irrespective of the underlying reason for the behaviour disorder. This was predicted by Wood and Eames (1981). Unfortunately many patients with behaviour disorders fail to obtain twenty five weeks of any kind of treatment, least of all rehabilitation. It is clear from this analysis however, that in general terms, this is the minimal requirement to ensure that a behaviour disorder has been reliably controlled, allowing conventional rehabilitation to proceed.

CHAPTER 7

PROBLEMS OF CONTROL

Inappropriate Sexual Behaviour

Aggression is not the only behaviour perceived as threatening and unacceptable by rehabilitation staff or members of the community. Unwanted sexual advances, especially when made in a coarse, loud, disinhibited way, usually in public and often by individuals who "look odd" and seem unresponsive to reason, can be difficult to deal with. This is a particular problem when the protaganist is viewed as a "handicapped" person, for whom allowances should be made.

Rehabilitation can be seriously prejudiced by such behaviour. Therapists who feel intimidated may refuse to treat, spend less time treating or not give the quality of treatment which would otherwise be available. Such reactions are unfortunate but understandable. They do however, emphasise the need for a method of management that will help control, even eliminate this kind of behaviour, facilitating the transfer of such individuals back into the community.

As in most disorders, treatment should try and direct itself at the **cause** of the problem (if such cause

exists). With sexual behaviours however, the causes remain somewhat vague. In chapter 2, the review of sexual disorders after brain injury suggested that they could originate either as part of a temporal lobe disturbance (when they appear as intermittent behaviours, totally out of character with the rest of behaviour) or as part of a wider disturbance of behaviour (such as in the context of a frontal lobe syndrome).

This information was based on brain injuries other than those caused by head trauma. Before proceeding to the methods and effects of treatment therefore, a review of severe head injured patients, attending the authors out-patient clinic, will be made to determine (1) the incidence of inappropriate sexual behaviour after severe traumatic brain injury and (2) whether an association exists between different types of injury and different forms of sexual behaviour.

7.1 Sexual Behaviour Following Severe Brain Injury

Subjects. A review was made of 132 severely brain injured patients (PTA > 1 week) who had attended for neuropsychological assessment after head injury. The information was gathered over a period of 3 years. The characteristics of this group are presented in table 7.1(a).

Table 7.1 (a)

Patient Characteristics

(N = 132)

	AGE	P.T.A*	T.S.I+	I.Q.
Mean	30.4	3.2	60.5	82.1
S.D.	10.4	2.5	34.4	15.9
Range	13-67	1-6	12-132	65-110

* PTA= Post traumatic amnesia, in months

+ TSI= Time since injury, in months

Procedure. Part of the clinical interview routinely deals with changes of behaviour, noticed either by the patient or the nearest relatives (the latter interviewed independently). Information regarding **sexual** behaviour is not always obtained from direct questioning, often it is spontaneously offered (as a complaint) by the spouse.

When however, the behaviour is more disinhibited in nature there are usually many references to the fact in the medical history.

Information is obtained on the following issues:-

(1) Has sexual **ability** been affected, i.e. is the patient able to obtain and maintain an erection.

(2) Has sexual **arousal** changed, i.e. is the patient more, or less interested in sex.

(3) Has the **quality** of sexual behaviour changed, i.e. has the patient adopted any bizarre or unusual interests with respect to the sexual relationship.

It must be appreciated that these questions would be very difficult to present in a standard questionnaire format. The fact that the data were collected from an unstructured interview does not necessarily affect the accuracy of such data however, or its ability to identify the presence of such problems.

For the purpose of this study only question 2, relating to **change in sexuality** will be examined, to determine whether any particular type of brain injury or pattern of behaviour is more associated with inappropriate sexuality than any other.

Results.

(1) Sexuality in relation to the cause of injury. Table 7.1(b) shows the incidence of the different **causes** of injury. A **deceleration** injury involves a road accident or a fall, (any injury where a moving head comes into contact with a stationary object, producing sudden inertia and a rotation of the brain within the skull). Conversely, injury from an '**assault**' means that a moving object has come into contact with a stationary head, e.g. being hit by a falling brick! (**Missile wounds** obviously produce a penetrating injury.)

These three different **causes** produce very different **effects** on brain tissue. The concussive injury from deceleration is much more diffuse than that from an assault injury, while a missile wound is often very localised with respect to the concussive damage it causes, especially when the missile is low velocity.

The disproportionate nature of this data accurately reflects the number of road traffic injuries relative to other injuries to the brain (Field 1976) but prevents any analysis being made of the association between cause of injury and this kind of behaviour.

Table 7.1(b)

Nature of Injury in Relation to the Frequency of Sexual Problems

Type Of Injury (N)	Sexual Problems(N)
Deceleration = 117	34 (29%)
'Assault' = 12	2 (16%)
Missile Wound = 3	0 (0%)

(2) Relationship between type of injury and sexuality. Table 7.1(c) shows that of 132 patients with severe head injury, 68 were identified, from their clinical presentation, medical history, and C.T. scan evidence (when available), to have sustained predominantly frontal lobe injury. From the medical histories it was known that 11 had orbito-frontal lesions, regarded by Lishman (1978) and Powell (1981 a,b) as the frontal injury most likely to result in sexual changes. All of these patients had some degree of behavioural disinhibition, varying from a rather euphoric disposition with bland indifference to circumstances, to a marked state of insightless and poorly controlled behaviour with inappropriate sexual advances being one of the main features.

Table 7.1(c)

Type of Injury in Relation to Sexuality

	Frontal	B.Stem	Temporal	None*
Sex Prob.	31	0	1	4
No Prob.	37	20	26	13

* None = No evidence of residual brain damage.

The 20 brain stem injuries were identified by independent neurological opinion on the basis of clinical examination, while the 27 temporal lobe patients were all reported to have abnormal foci on EEG recordings. The remainder had no residual neurological, radiological or electrophysiological abnormality. Severe intellectual impairment was the main legacy of this group.

Of these patients, 36 presented with altered sexuality that was noticed and reported by families or 'significant others'. Only 9 were reported to have **increased** sexuality (7 of these had orbito-frontal injuries), the others showing a lack of interest, ability or both.

Analysis of this data was made using Chi Square with the Yates correction for small numbers (table 7.1(d)). The numbers of patients with sexual changes, but without frontal injury, was so small that for the purpose of analysis the patients were grouped according to **frontal** or **non-frontal** injuries. The analysis showed that the

number of frontal lobe patients displaying changes in sexuality is unlikely to be due to chance (X2= 21.85; df=1; p,0.001).

7.1(d)

Relationship of Frontal Brain Injury to Changes in Sexuality

Number of brain injured pts= 132

" " frontal lobe " = 68

CHI SQU MATRIX=

	Frontal	Non-Frontal	
Sexual Problem	31	5	(36)
No Sex Problem	37	59	(96)
	(68)	(64)	(132)

CHI SQU= 21.85 (Yates Correction)

Probability due to chance p< 0.001

Commentary. The data suggests that a significant relationship does exist between frontal lobe injury, a disinhibited pattern of behaviour, and changes in sexuality. It must be remembered however, that the patients included in this small study are not necessarily representative of a severe brain injury group. These patients were all **clinical** referrals, made **post** injury, when a more representative sample would probably be obtained in a **prospective** study, following-up a number of consecutive cases of severe brain injury. Consequently, the proportion of frontal cases may be inflated (although I know of no study giving the relative numbers of different types of injury in a sample of brain injury).

7.2 Single Case Studies In The Management Of Sexual Disorders

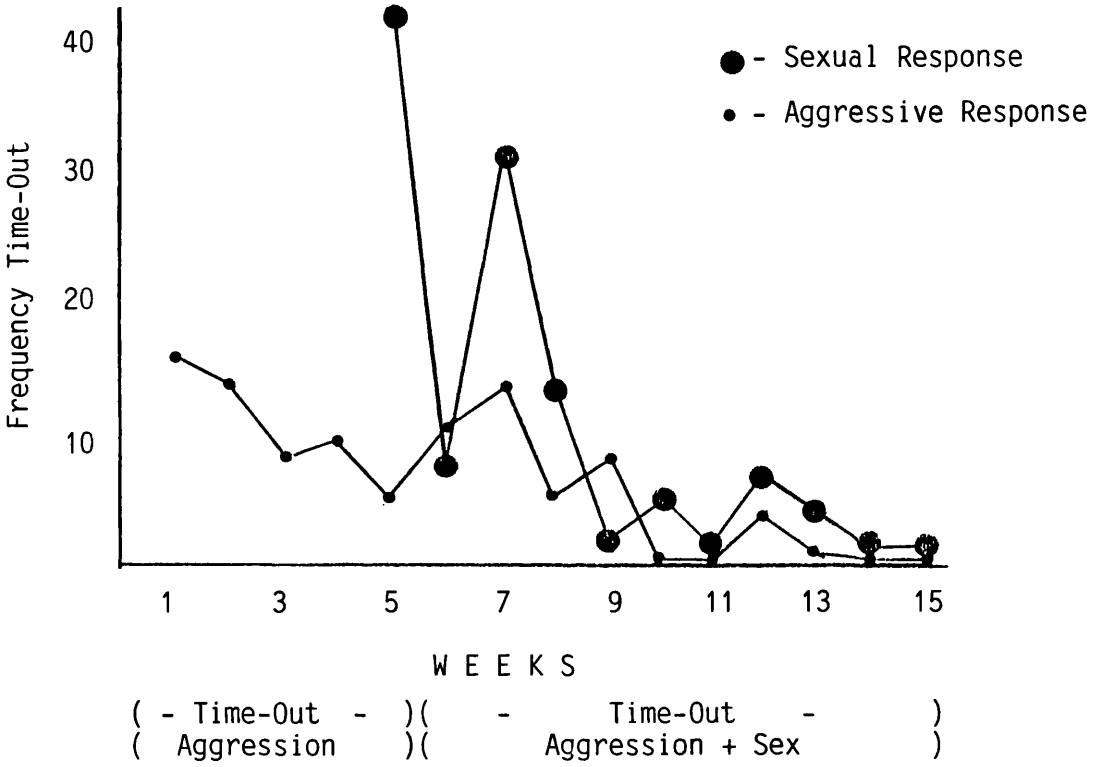
Inappropriate sexuality usually occurs in the context of a more general state of disinhibition, where embarrassing and provocative behaviours predominate and any attempt at imposing sanctions or control meets with an emotional or aggressive outburst. Such behaviour can however, respond quite well to behaviour management procedures (time-out) as the following case studies show.

Sexual behaviour and aggression. Figure 7.2(i) shows how this method succeeds in controlling the problem of inappropriate sexual behaviour in a patient who also exhibited aggressive outbursts. The patient was an 18 year old man who suffered frontal and temporal lobe damage as a result of a head injury (PTA= 4-6 months) 2 years earlier. In addition, he had a mild brain stem injury, which produced problems of balance, due to truncal ataxia. He also had visual field defects and a scanning dysarthria.

His behaviour was characterised by outbursts of aggression, associated with a temporal lobe EEG abnormality, treated with carbamazepine. He was also sexually disinhibited and was reported to have approached women and young girls in the vicinity of his home. After admission he made several advances to female staff, behaviour which had led to his discharge from another

FIGURE 7.2(i)

SEXUAL BEHAVIOUR



Control of inappropriate sexual behaviour.The significant trend for aggression is reversed when, at week 6, the programme is expanded to include time-out for sexual behaviour.

rehabilitation centre.

The first problem for patient management was aggression control. This was dealt with in the usual manner, employing a time-out room procedure. By week 5 of this programme the frequency and severity of his aggression had responded to treatment, showing a type 1 trend ($F= 27.42$; $df= 1$; $p< 0.05$).

Once aggression had been reduced to a manageable level the programme was able to incorporate sexual responses. At this time (week 5) a behavioural baseline, over one week, recorded 40 inappropriate sexual responses. A programme of contingent time-out rapidly reduced the sexual behaviour ($t= 3.96$; $df= 7$; $p< 0.01$) but appears to be associated with a corresponding increase in aggressive behaviour. This period of instability affects behaviour generally, in particular his sexual behaviour. Such variability may simply be a feature of the temporary increase in responding, often seen after commencement of a treatment programme. On the other hand it may indicate the effects of increased frustration on a young man who, as a result of brain injury, has poor control of behaviour and a low tolerance for tension of any kind.

From week 6 of the programme (week 1 for sexual behaviour), a type 2 trend emerges ($F= 2.74$; $df= 1/6$: $t= 9.74$; $df= 7$; $p< 0.001$), indicating a significant drop in sexual tendencies. His behaviour became more manageable and appropriate, giving him more time in therapy and

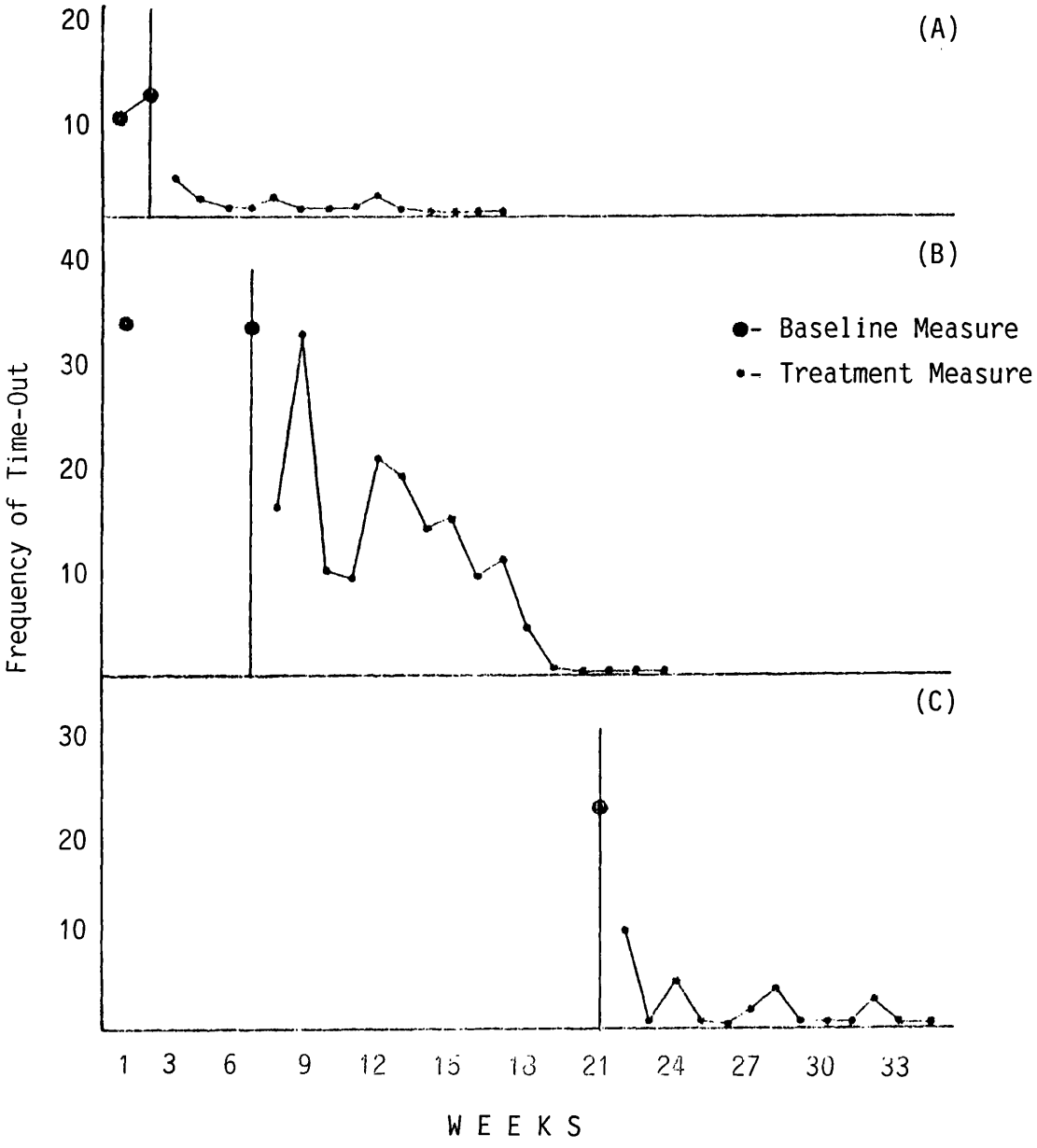
subsequent improvement in balance and mobility. The programme later generalised to other therapy areas and, through the use of week-end leave, to his home environment. This eventually led to his successful discharge home.

Multiple baseline study to demonstrate the use of time-out as an effective method of management for sexual behaviour.

Figure 7.2(ii) presents a multiple baseline study of three patients, showing the continuity of this procedure over 34 weeks. The clinical history of each patient is very similar. They are all males in their early twenties who had sustained severe head injury in road traffic accidents more than three years before entering this programme. Their injuries all involved a major frontal lobe component and their behaviour was generally disinhibited and sexually provocative. The only difference, in terms of possible treatment effects, is that the second patient in the series had sustained a more diffuse injury, which had a greater affect on behaviour generally. In each case, the sexual behaviour was a major reason for previous lack of rehabilitation.

In the **first patient** there is obviously no type 1 trend in the time-out curve. However, the difference between the mean of the treatment phase compared to baseline shows a significant (type 2) treatment effect (t= 25.0;

FIGURE 7.2(ii)
SEXUAL BEHAVIOUR



A 3-Subject multiple baseline study to show the effect of a time-out programme on the frequency of inappropriate sexual behaviour.

df= 7; p< 0.001). The response curve over the first 8 treatment weeks for the **second patient** also shows a type 2 trend (t= 6.29; df= 7; p< 0.01). The absence of a type 1 trend is probably due to the resurgence of sexual responses 4 weeks into treatment, emphasizing the tendency of frontal lobe injuries to lose control of the target response during early stages of treatment. The response of the **third patient** is more typical of the type 2 trend recorded in figure 7.2(i) and the first patient in this multiple baseline (t= 16.63; df=7; p< 0.001).

Summary. The 4 patients described above responded in different ways and over different times but **all** made a successful response which was maintained after they were discharged back into the community. It is important to note the tendency for treatment directed at sexual behaviours, to provoke aggressive behaviours, requiring that both be dealt with at the same time.

7.3 Habit Disorders

The behaviour disorders described so far have all been controlled using methods of negative punishment (time-out; loss of tokens). Occasionally however, behaviours will present themselves which respond better to methods of positive punishment, using aromatic ammonia as the noxious stimulus.

Following brain injury, behaviours may develop which take the form of "habits". These can range from manneristic behaviours, which have an autistic quality, (repetitive responses such as scratching, picking at clothing or tapping furniture), to habits which possess repulsive and nausea producing qualities, such as protracted nose picking, spitting or throat clearing. These behaviours are very unacceptable, devoid of social skills and most unlikely to endear the person, displaying the habit, to members of the community with whom they come into contact. Figures 7.3(i,ii,iii) illustrate how control was achieved over three "nauseating" behaviours.

An intra-subject multiple baseline to show the effect of different management procedures on habit behaviours.

The patient had received a severe head injury (PTA unknown) five years before whilst in her early teens. Subsequently, she developed certain autistic tendencies, some of which included manneristic behaviours (repeatedly picking fluff off the floor or her clothes). In addition

she had a tendency to throw anything she could get her hands on, (ashtrays, cups, telephones). In addition to throwing objects she would also pick them up and bang them down, either repetitively or as a prelude to throwing them . Finally, she displayed a frequent, protracted and particularly unpleasant nose picking habit which, besides nauseating onlookers, also interfered with therapy activities because she would pick her nose instead of getting on with her work.

Because of the problem they produced (having already precipitated her discharge from another unit), the throwing and banging behaviours were dealt with concurrently. This is somewhat unusual, but they were observed to be produced as part of the same response. The nose picking, a separate behaviour, would normally be attended to when the other behaviours came under the control of the reinforcement contingency. In this case however, the former was difficult to achieve whilst, in the mean time, the nose picking was becoming more and more repulsive and purposeful. As a result a positive punishment programme was introduced 10 weeks after admission, during which time the time-out programmes for throwing and banging had been in process.

Programme 1 (for banging) uses an **A-B-C** reversal design, replacing the time-out room (**B** phase) for TOOTS (**C** phase). Programme 2 employs an **A-B-C-B-C** reversal design,

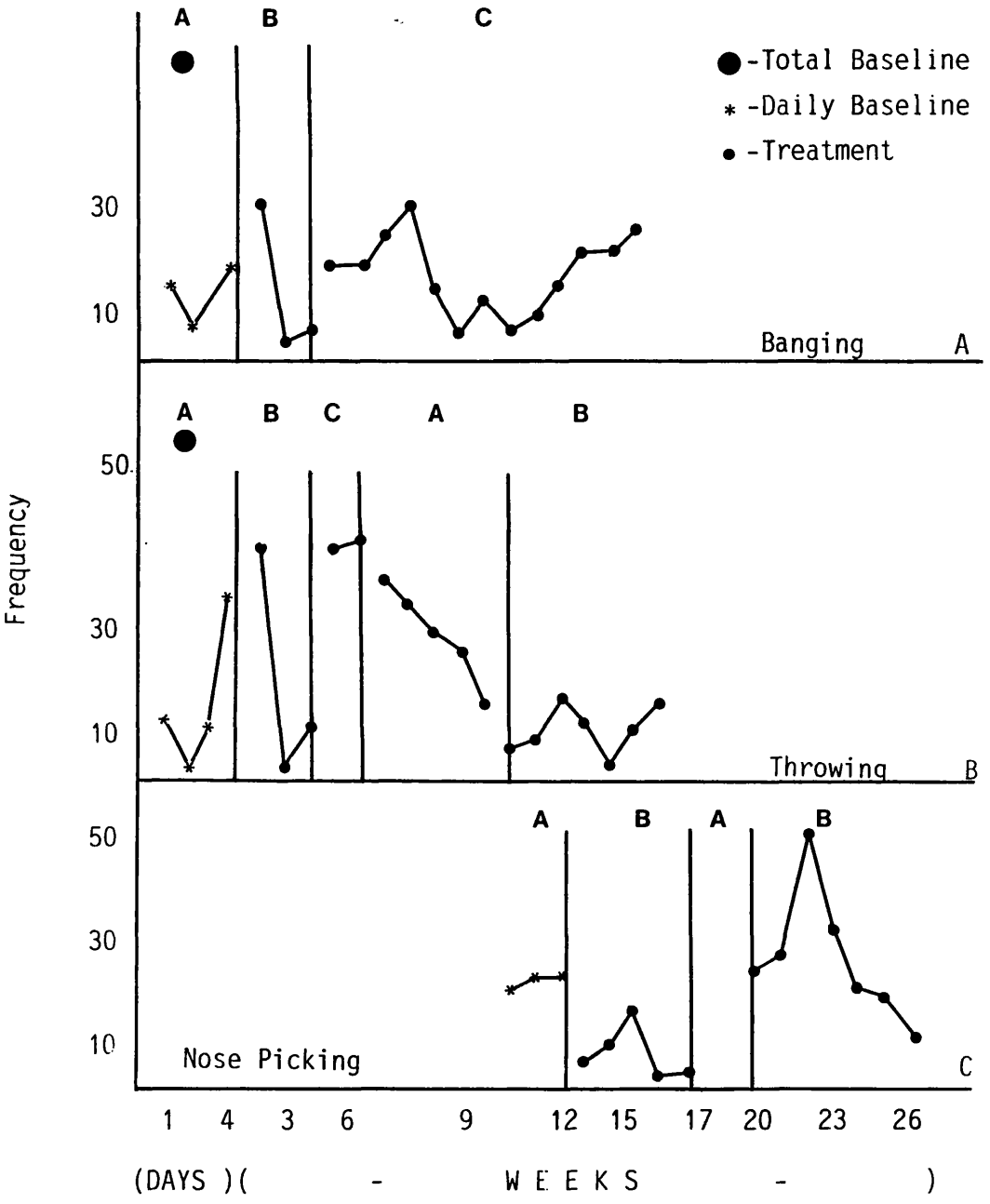
whilst the last procedure for nose picking uses an **A-B-A-B** withdrawal design. Response to treatment is illustrated in figure 7.3(i).

Although there was a good initial response to the time-out procedures in programmes A and B, it was not maintained. Both programmes were changed to T.O.O.T.S. procedures because of the ethical issues surrounding the use of a time-out room, for what was essentially non-aggressive behaviour. This change in reinforcement strategy proved to be premature because, as the graphs show, both behaviours increased in frequency (the throwing nearly to baseline levels).

The banging continued to be managed using a TOOTS procedure. The reduction in frequency between the 4 days of the A phase and the first 8 weeks of the C stage shows this to be statistically significant ($t = 13.91$; $df = 7$; $p < 0.001$) but without evidence for a significant trend during the C stage ($F = 3.61$; $df = 1/5$). This behaviour was still too frequent to be considered a clinical success. In programme B the re-introduction of a time-out room procedure managed to re-gain control of the throwing. This later returned to a TOOTS programme with reasonable success. Comparison of the A and second C stage shows a significant reduction ($t = 48.2$; $df = 5$; $p < 0.001$) but, because of the variable response rate, still too unreliable to be regarded as a successful treatment outcome (another type 3 trend).

FIGURE 7.3(i)

HABIT BEHAVIOURS

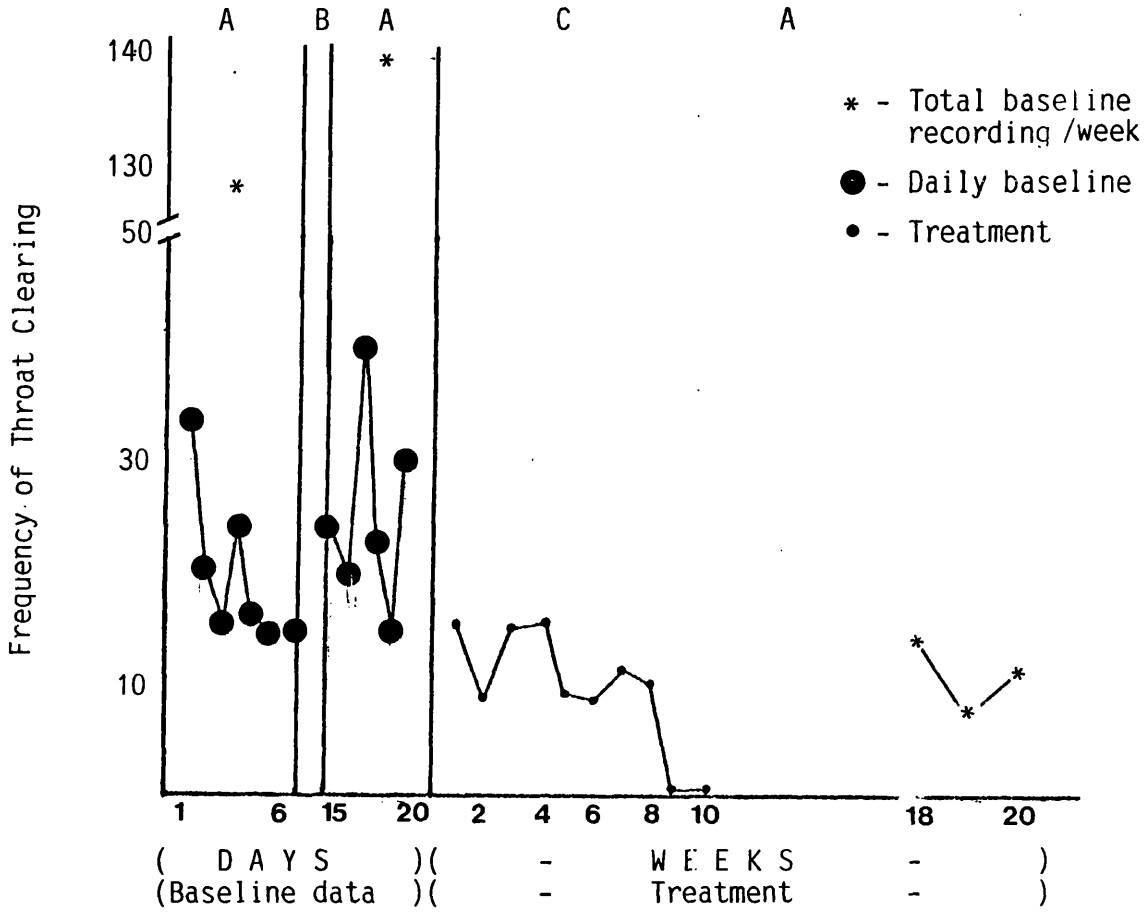


Intra-subject multiple baseline dealing with three problem behaviours, each receiving different reinforcement contingencies. Programmes 1&2 use negative punishment. Programme 3 uses positive punishment.

This was not the case with the nose picking however. Using a positive punishment (ammonia) programme the behaviour showed a significant drop in frequency. If the last 14 baseline days are compared to the first 14 days of treatment a type 2 trend emerges ($t = 3.80$; $df = 12$; $p < 0.01$). The programme was discontinued before the treatment effect had been consolidated (for reasons of ward management unrelated to this programme), re-creating the **A** phase and a withdrawal design. This resulted in the target behaviour increasing to baseline levels. Reintroduction of the **B** phase was not immediately effective and there was no evidence for a significant trend ($F = 2.68$; $df = 1/5$). A consistent improvement was recorded after the third week of this phase however, resulting in virtual elimination of the habit for the rest of her in-patient period. The other behaviours also responded over time allowing her to return home with successful results.

Positive punishment for a patient with persistant throat clearing. Details of this patient have already been given in chapter 5 (figure 5.2(ii)). In addition to his aggressive behaviour he had developed a very unpleasant throat clearing habit which, as the **daily** baseline measure shows, occurred with a high frequency. This was not a recent development but something which had been gaining strength for some time since his accident. He

FIGURE 7.3 (ii)



A positive punishment programme, using a reversal design, to reduce a nauseating throat-clearing habit. The B phase represents the first treatment method (massed practice), the C phase is the second treatment (aromatic ammonia).

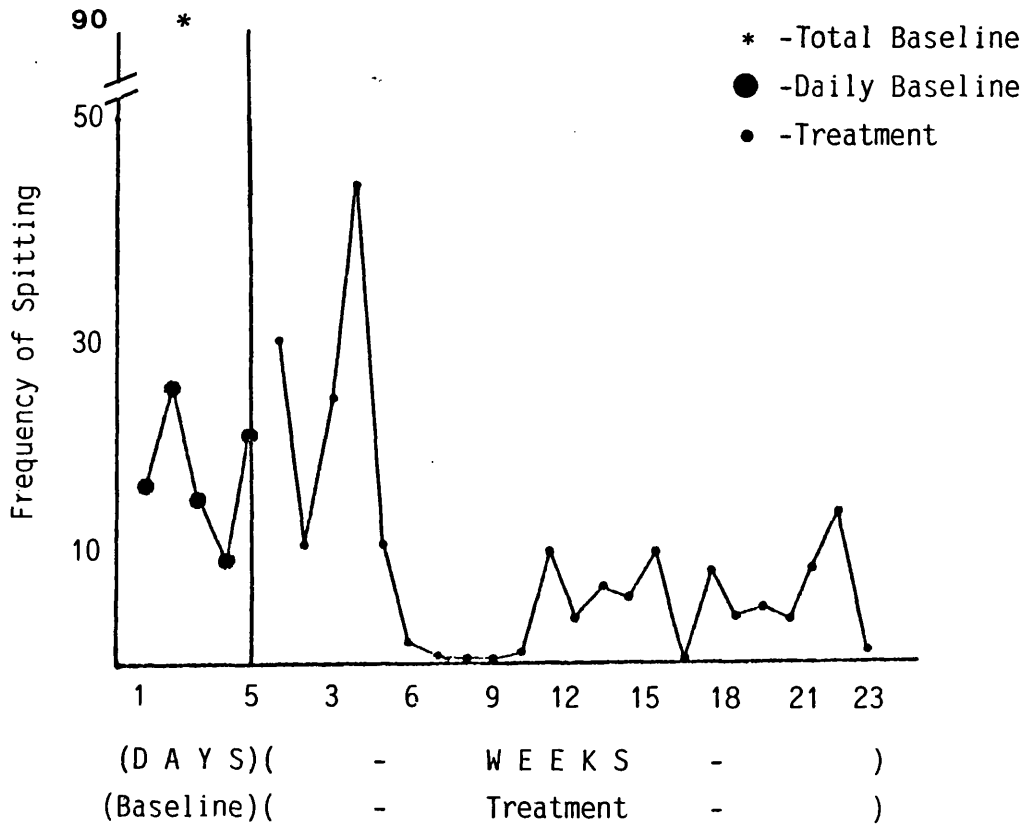
made no effort to spontaneously control this habit, indeed, once aware that it affected members of staff, the behaviour appeared to be used purposefully.

The positive punishment procedure was not the first treatment applied however. Figure 7.3(ii) shows that an **A-B-A-C-A-C** design 'evolved', as a result of the first programme failing to produce any clinical improvement. This allows a comparison of his response to alternative treatment approaches.

The first treatment (**B** phase) involved a **massed practice** procedure which required the patient to indulge his habit for periods of one hour, three times each day. This method proved singularly unsuccessful, as a comparison of the two baseline periods show ($t = 1.04$; $df = 9$;). In comparison, the transfer to a positive punishment programme was dramatically successful, reducing the frequency of this habit from an average of 19.7 a **day** to 15 a **week** ($t = 16.43$; $df = 6$; $p < 0.001$). This reduction in reponse, obtained after only seven treatment days, was maintained throughout the treatment period. No significant increase occurred after a period of no treatment ($t = 1.34$; $df = 9$).

A positive punishment procedure to prevent spitting. This patient had developed a habit of spitting on the floor in the patients lounge and therapy areas. A baseline recording showed this to occur, on average, 117 times

FIGURE 7.3 (iii)



Reduction of spitting within the ward area during a positive punishment programme using aromatic ammonia vapour.

each week. The patient had sustained a very severe head injury (PTA= 6 months) 4 years previously, aged 17. Damage to the frontal lobes was the primary injury but he had also sustained some brain stem damage. His behaviour was very disinhibited, he was extremely distractable and lacked almost any kind of control over emotional responses. Spitting was only one of many specific behaviours in need of control. Besides being a dirty (and the way he did it) very unpleasant habit, it was probably the main obstacle in the way of getting him accepted for long term residential care by a Cheshire Home.

Figure 7.3(iii) shows that in the first **week** his spitting was reduced to 30 incidents. However, rather typical of a frontal lobe patient, this control was not maintained. Comparison of the first 8 weeks of treatment with the baseline does indicate a treatment effect however($t = 16.43$; $df=7$; $p < 0.001$).

Summary. This increase in a behaviour response two to four weeks into treatment, emerges as a regular feature of frontal lobe patients. In most cases it is quickly followed by a reduction in frequency, which usually remains stable. The pattern shown in figure 7.3(iii) is typical, the patient showed a further escalation of spitting 15 weeks into the programme. This is probably because the nursing and therapy staff who administered the treatment became less vigilant of this behaviour with

the passage of time, allowing a variable reinforcement ratio (which was not sufficiently intensive to control this response) to replace a continuous ratio.

Commentary. The above case studies provide some important guides to behavioural treatment with severely brain injured patients:-

(i) Treatment needs to be maintained for quite long periods before a **treatment effect** is consolidated. This is particulaly the case with frontal lobe injuries.

(ii) A continuous reinforcement ratio needs to be maintained far longer than appears to be the case in other areas where treatment of this kind is applied.

These facts will need to be acknowledged when describing other treatment programmes

CHAPTER 8

APPLICATIONS OF BEHAVIOURAL TECHNIQUES IN BRAIN INJURY REHABILITATION

Introduction

A large repertoire of behaviours are needed to reliably carry out those activities of daily living which determine our level of social and domestic independence. Most people usually take these skills for granted because, through a long process of (behavioural) learning, many complex behaviours have become largely automatic. These range from the act of talking or walking, to the sequence of behaviours involved in organising one's shopping.

Following severe brain injury many, once automatic behaviours, become seriously disrupted. Van Zomeren et al (1984) provide an explanation for this in terms of information processing deficits. This will be discussed in some detail in later chapters. From the rehabilitation perspective however, it means that patients have to re-learn a range of quite basic behaviours; for example the coordination of balance, posture and gait, necessary for independent walking; the control of tongue and lip movements in relation to breathing, to allow clearly enunciated speech, while, at a more cognitive level, the patient must learn to order the sequence of behaviours

that unite to make a cup of tea or a simple meal.

The difficulties a patient may have in re-learning functional skills depends on (1) the extent of cognitive impairment and learning disability; (2) the degree to which the patient has developed **compensatory** behaviours (behaviours which have been learned as a way of avoiding a function or getting around a problem of dysfunction). These two conditions are not mutually exclusive. When they co-exit in the same patient they can create significant problems of new learning.

To re-establish these functional skills another of the behavioural techniques described earlier (**shaping**) can be used. This is a procedure developed from operant conditioning, used when the behaviours to be changed are complex or occur infrequently. The procedure is to reinforce **successive approximations** to the desired response, starting with a simple response and building up to a more exact and complex one. Many of the patients described in this study, not only have major cognitive problems, but are also several years post injury. This means that behaviours, be they inappropriate or compensatory, have been present for some time and are unlikely to show sudden or dramatic improvements when subjected to behavioural treatments. Consequently, many constructive or adaptive responses have to be developed gradually by the process of shaping.

8.1 Activities of Daily Living (ADL)

"ADL" skills are probably the most important and wide ranging skills to be considered in the rehabilitationists ambit. They include those skills which determine whether an individual can be considered capable of independent life outside an institution; such as ability to use public amenities/transport; a knowledge of road safety; shopping and budgeting skills; cooking skills, including safety in a kitchen; personal hygiene and care of laundry. To illustrate the effectiveness of behaviour techniques in this area 7 case studies, organised as one single-subject and two multiple-baseline studies, are presented to show the effect of behaviour management on a morning hygiene programme.

The seven patients in this study had all been provided with help in washing and dressing for several years. Paradoxically, the nursing care they received had made them more **dependent** than independent, which is often the case in centres dealing with **handicapped** people. Re-acquiring self help skills after so many years of having things done for them was obviously going to meet with some resistance.

A single subject design illustrating the use of a behaviour management approach to improve independence during a morning dressing and hygiene programme.

This was designed to show that patients can increase their level of independence during morning activities which include getting out of bed, washing, room tidying, dressing and arriving for breakfast on time.

The importance of establishing independence in this area will be appreciated by the knowledge that not only do such behaviours present a great burden to nursing staff, who have the responsibility of getting the patients ready for the "working day", but also that many of the more pleasant, long term residential units (e.g. Cheshire Homes), often refuse to accept people without such basic independence skills.

The programme required a patient to learn a sequence of behaviour which involved:- getting out of bed, washing and then dressing. To facilitate learning, the behaviour was broken down into small units. The patient was given a prompt to begin each unit. If she responded correctly after each prompt, reinforcement (a token or in some cases a point) would be given, plus praise and encouragement. The token or point could be exchanged for a reward (usually breakfast) immediately after the programme had been completed.

An example of the command-reward procedure is :-

Pull back the sheets --- 1 token

Get out of bed --- " "

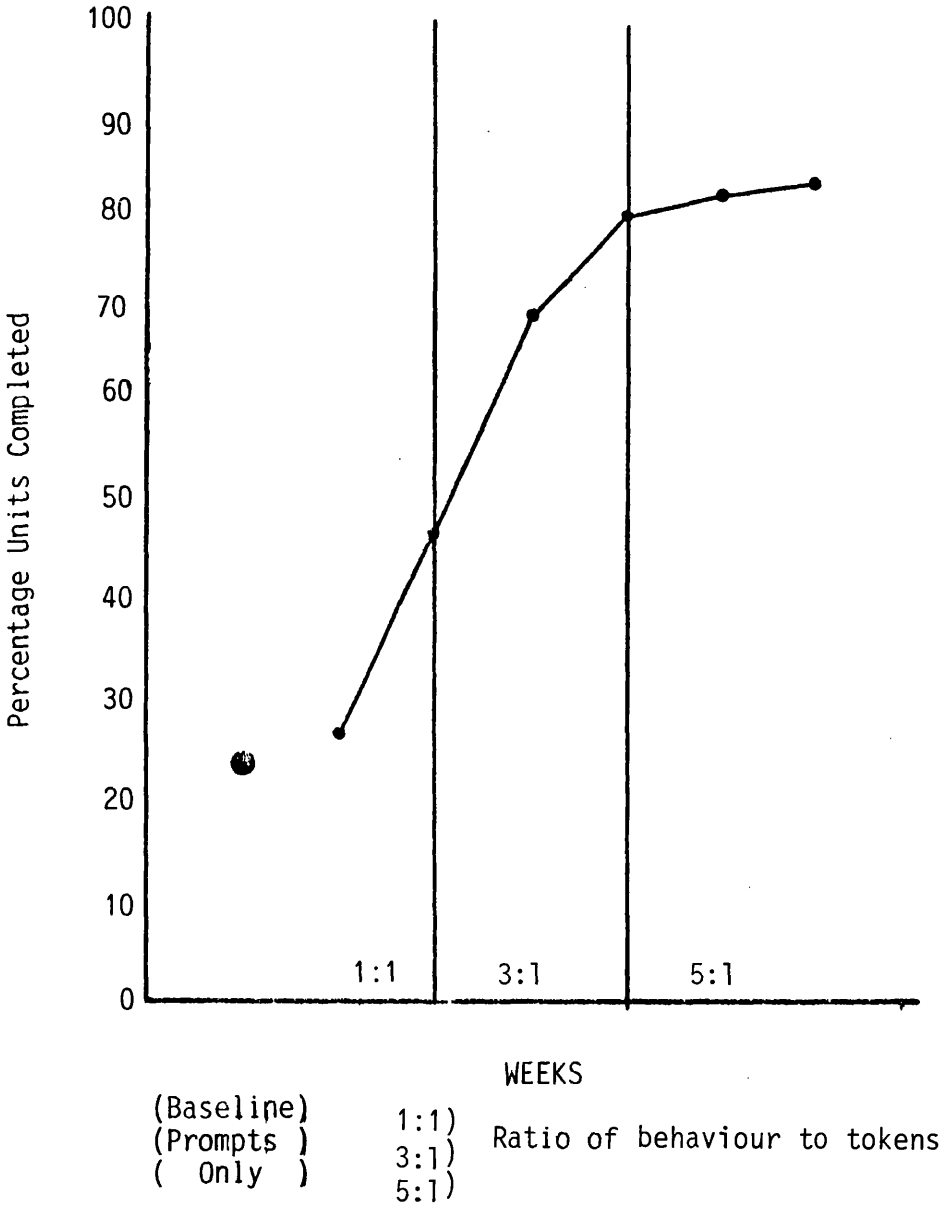
Walk to the wash basin - " "

Once the patient had become established on this programme, earning consistently above a certain minimum (of which they were unaware) the ratio of prompts to token earnings would change from a one to one ratio, to a three to one and then a five to one ratio. This meant that for a 3:1 ratio the prompt would be extended to include more behaviours :-

Pull back the sheets, get out of bed and walk to the wash basin --- 1 token

Figure 8.1(i) shows the effect of this procedure on the "autistic" type patient described in chapter 7 (figure 7.3(i)). After several years of being very dependent in this area the patient shows evidence of a steady improvement. This does slow down somewhat when the different reinforcement ratio's are introduced but not to the point that it interferes with the overall significance of the trend ($F= 21.89$; $df= 1/4$; $p<0.01$).

FIGURE 8.1(i)



Improvement in self help skills during a morning dressing and hygiene programme.

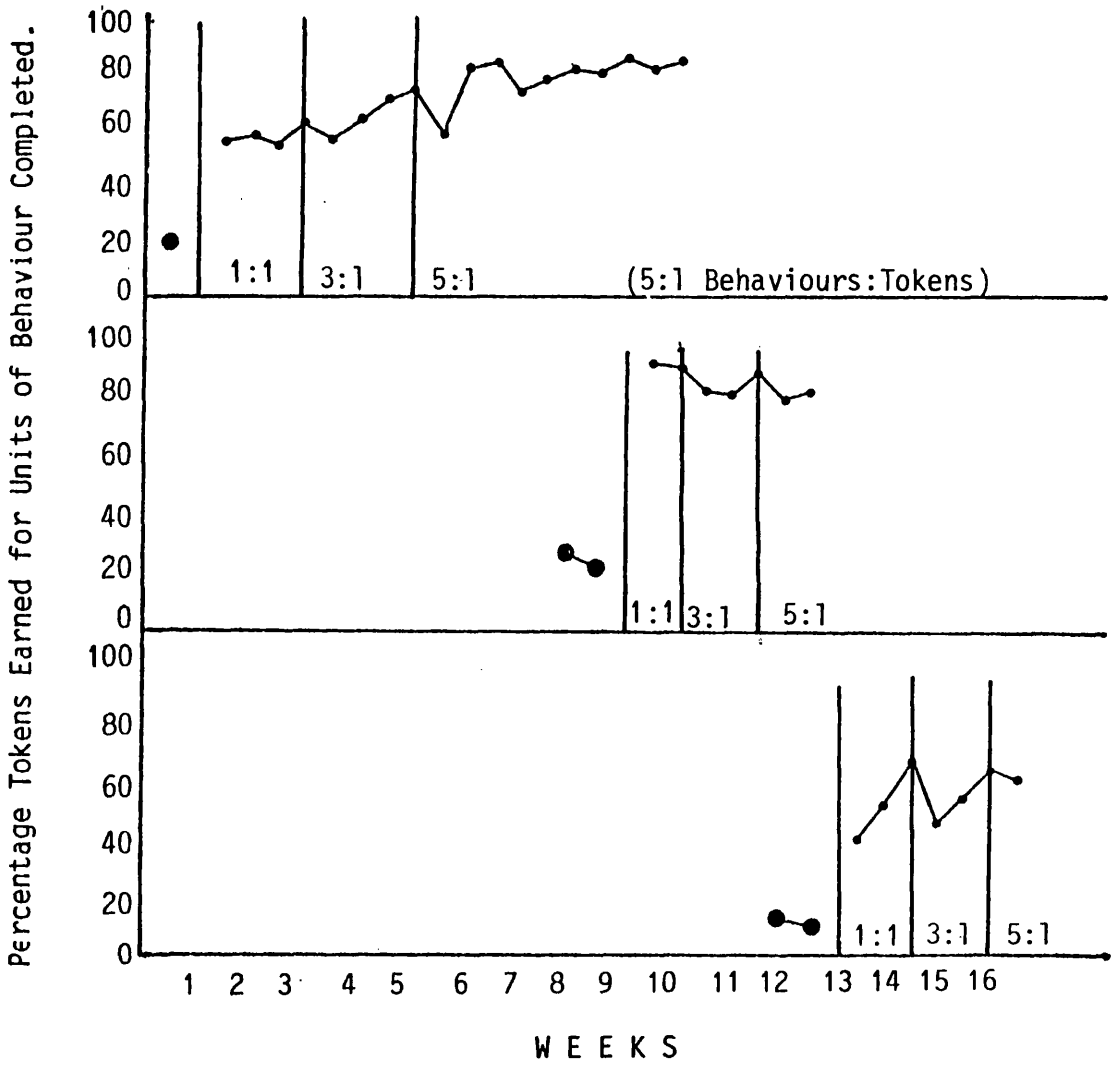
Two multiple baseline studies showing the general effect of the 'morning programme

These studies show that improvement in this area of independence can be obtained through different forms of reinforcement contingency. Figure 8.1(ii) shows the **number of tokens earned** for correctly **responding** to prompts while figure 8.1(iii) records the **reduction in the number of prompts** needed for these behaviours to occur.

The first patient in figure 8.1(ii) suffered an anaesthetic accident which resulted in diffuse cortical damage. She was cortically blind but, more importantly, presented a dissociative behaviour pattern, with many characteristics that were regarded by psychiatrists in the hospital as histrionic or hysterical. Her lack of independence in this area was more a matter of disinclination than disability. The structured behaviour programme, with its reinforcement contingency, produced an immediate improvement ($F= 15.0$; $df=1/6$; $p<0.01$). This increase in her level of personal independence was maintained (and even continued to improve) throughout the next eight weeks

The second patient was referred to in chapter 6 (figure 6.2(vi)). Her problem was poor hygiene, she also took an inordinate amount of time to get ready each morning. In this case, points were earned for responding to prompts within a certain time limit. A significant type 2 trend

FIGURE 8.1(ii)



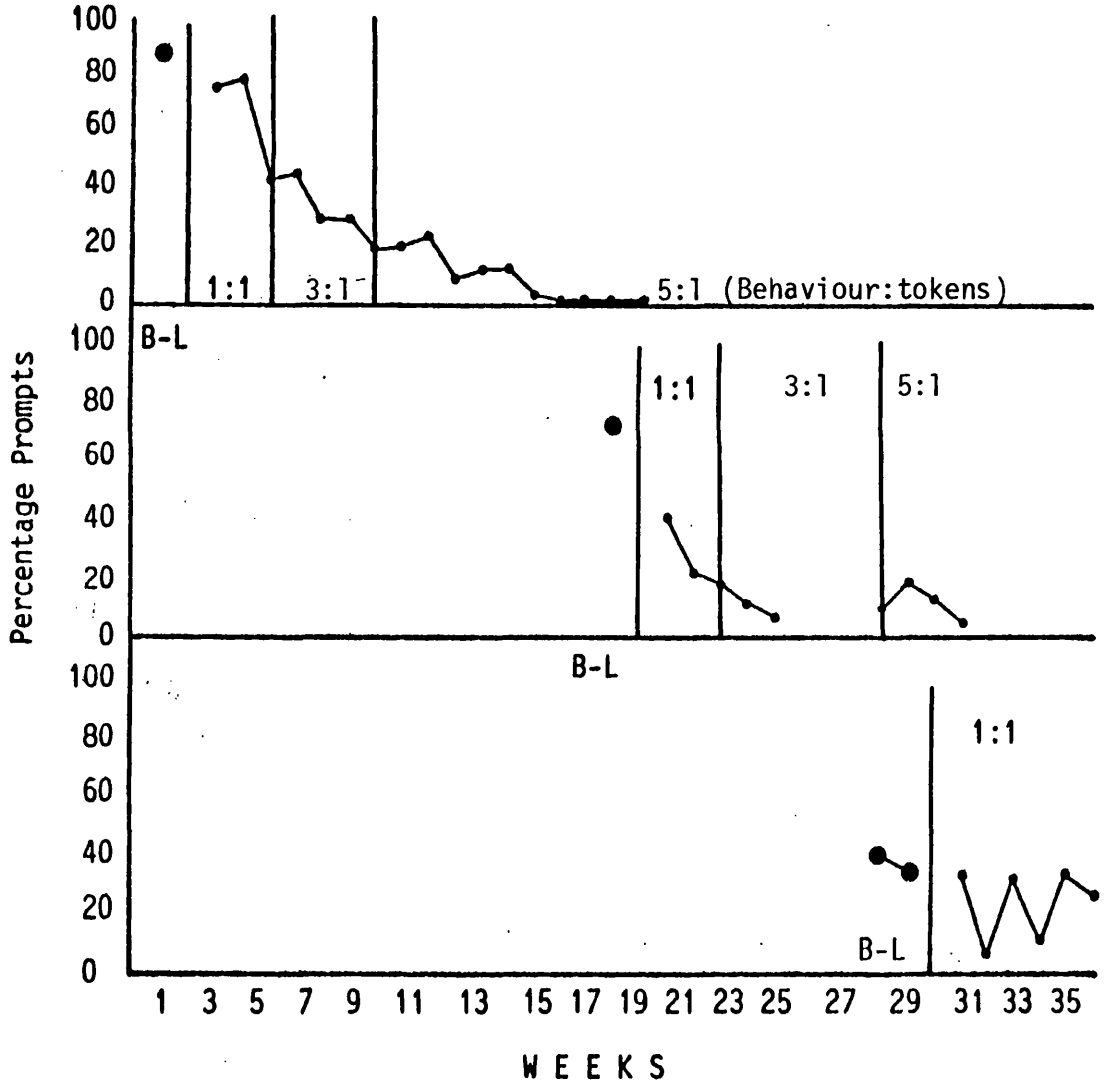
Multiple-baseline design to show improvement in self help skills during positive reinforcement of individual units of behaviour.

(t= 28.4; df= 6; p< 0.001) was recorded. The initial improvement dropped slightly but overall was maintained at a high level. During the first seven treatment weeks however, the trend actually showed a significant return towards baseline (F= 8.39; df=1/5; p<0.05). This halted after the seventh week (although the recording had stopped by that time) and a more stable (improved) response pattern was maintained.

The final patient in this series had also sustained a severe frontal injury. He did not record a significant trend (F=2.54; df=1/5), probably because of the marked 'dips' in his progress each time the reinforcement schedule was changed. A students t analysis, comparing the seven treatment weeks to baseline, confirms that a type 2 treatment effect was obtained (t= 4.74; df= 6; p< 0.001).

The second baseline study, figure 8.1(iii) shows a similar improvement in personal independence, this time making token payments contingent upon the **number of prompts.** This procedure was chosen because the other programmes had shown that while some patients could make an adequate response after several prompts, they were less able to respond to a **single** prompt. This would result in them consistently failing to earn, thus invalidating the **shaping** procedure. Consequently, tokens were earned on a "sliding-ratio" which increased both on

FIGURE 8.1(iii)



Multiple-baseline showing the reduction of prompts needed during a morning dressing and hygiene programme. after positive reinforcement had been introduced for individual units of behaviour. The token:prompts ratio changed as the behaviour improved.

the basis of behaviours:tokens and also number of prompts:tokens. Because there are two variables affecting the delivery of reinforcement the learning paradigm is more difficult than the one described in the previous figures. Rather ironically, the patients were also ones with more severe cognitive deficits than those described above. These circumstances probably account for the improvement in self-help skills not being as immediate or dramatic in these patients as the others.

The first two patients in the series displayed frontal lobe characteristics which affected the organization of many aspects of their behaviour; even so, they showed a significant trend ($F= 38.3$; $df=1/6$; $p< 0.01$: $F= 8.96$; $df=1/3$; $p< 0.05$). This can be contrasted to the third patient who had sustained a severe brain stem as well as frontal lobe injury and failed to show a consistent response ($F= 0.005$; $df=1/4$). The programme was discontinued after the sixth treatment week.

Summary. The multiple baseline designs confirm the effectiveness of behaviour programmes directed at improving organizational and independence skills. The method is sufficiently flexible to achieve the same results through different reinforcement contingencies and with patients of different levels of ability.

A small group study to show the effect of a behaviour management approach on activities of daily living

The development of many other ADL skills are aided by the pressure and structure offered by the token economy. This, together with a time-out facility, maintains a degree of control, so that cooperation and effort can be reliably obtained, allowing therapy to continue. The all-embracing nature of this approach means that there need be only a few programmes (such as the ones described above) which require specially organised, contingent reinforcement programmes.

The successful influence of behaviour management can however, be inferred from an analysis of the improvement in ADL behaviours measured by the rating scale described in chapter 3.

Subjects. A study has been made of eleven patients, admitted to the treatment programme over a two year period. They do not include all the patients admitted during that time (of which there were 15). The others were not included because they did not stay for a full treatment period (at least twelve months).

There is no control group against which to compare the progress of these patients. This is acknowledged as a weakness in the design but inevitable in the (clinical) circumstances. However, in many respects these patients act as their own control because, prior to admission,

they had shown no sign of improvement in ADL skills and were considered to be in need of full time nursing care. Any change in this status must surely be attributed to the behavioural environment because neither spontaneous recovery (a period they were now out of) nor previous 'conventional' rehabilitation procedures had succeeded in achieving any improvement.

Procedure. During the first 4 weeks of admission an assessment is carried out, by each therapy specialty, of the patients' ability. This baseline is then compared to later assessments, carried out at three monthly intervals. If we accept the admission assessment as a baseline, reflecting the patients level of ability for months (sometimes years) before admission, then any improvement can be interpretted as an effect of the behaviour management system.

Results. The data in table 8.1(a) gives the mean scores and standard deviations for all the patients in each assessment category, recorded at the time of their admission assessment and immediately before discharge. Analysis of the difference in scores between admission and discharge shows significant effects. The abilities scale gives a t statistic of $t = 8.75$; $df = 12$; $p < 0.001$) and the perfomance scale a statistic of $t = 9.30$; $df = 12$ $p < 0.001$.

Table 8.1(a)

	Admission				Discharge			
Category	Ability	!	Perfomance		Ability	!	Performance	
Mobility	68.0	28.6!	61.2	32.6	78.2	24.1!	74.1	26.6
Table Habits	84.4	16.9!	71.8	23.9	93.7	7.1!	84.5	12.7
Hygiene	83.4	18.3!	75.8	22.5	91.7	11.8!	84.3	15.6
Activity	63.4	24.8!	53.0	23.5	80.8	17.8!	70.8	18.3
Social	67.3	21.4!	55.3	22.1	84.2	22.6!	75.0	21.7
Personal care	64.4	26.6!	54.3	21.0	79.2	19.5!	66.4	20.5
Occupation	66.8	20.5!	53.0	23.3	84.3	10.5!	72.4	13.5

A subsequent follow-up study (Eames and Wood in preparation) allowed us to determine whether this improvement was maintained. No significant changes between the pre-discharge assessment and follow-up assessment (made 6 months to 24 months later) were recorded.

Analysis of the individual subject scores for each category shows that although individual **performance** scores were always lower than the **ability** scores, the difference only reached significance on two occasions, at both times during the discharge assessment. The categories were table habits ($t = 2.09$; $df = 20$; $p < 0.05$) and occupational ability ($t = 2.29$; $df = 20$; $p < 0.05$).

Summary. The evidence from single case studies is far more impressive in demonstrating the effect of behavioural techniques in this area than is the analysis of ADL scores. When assessing the significance of these latter results however, consideration should always be given to the fact that, prior to admission, all these patients had a history of making little, if any, response to ADL therapy administered by conventional rehabilitational therapy procedures.

Traditionally ADL therapy has been an area dominated by occupational therapists. There are however, two good reasons why this role should be shared by the nursing staff. First, many patients remain potentially aggressive or belligerently uncooperative. The presence of a nurse working alongside the O.T. means that difficult behaviours are more easily dealt with (e.g. by means of time-out). Secondly, many of the A.D.L. activities are basic to nursing management (personal hygiene, dressing, continence). It is therefore necessary to include nurses in the training of these behaviour skills so that they can carry on the treatment at times when the O.T is off duty and formal therapy not available.

The importance of adapting traditional therapy approaches is that some principles of learning will help individuals with cognitive problems to organise and sequence their behaviour more efficiently. This will be illustrated in other behavioural approaches to rehabilitation.

8.2 PHYSIOTHERAPY

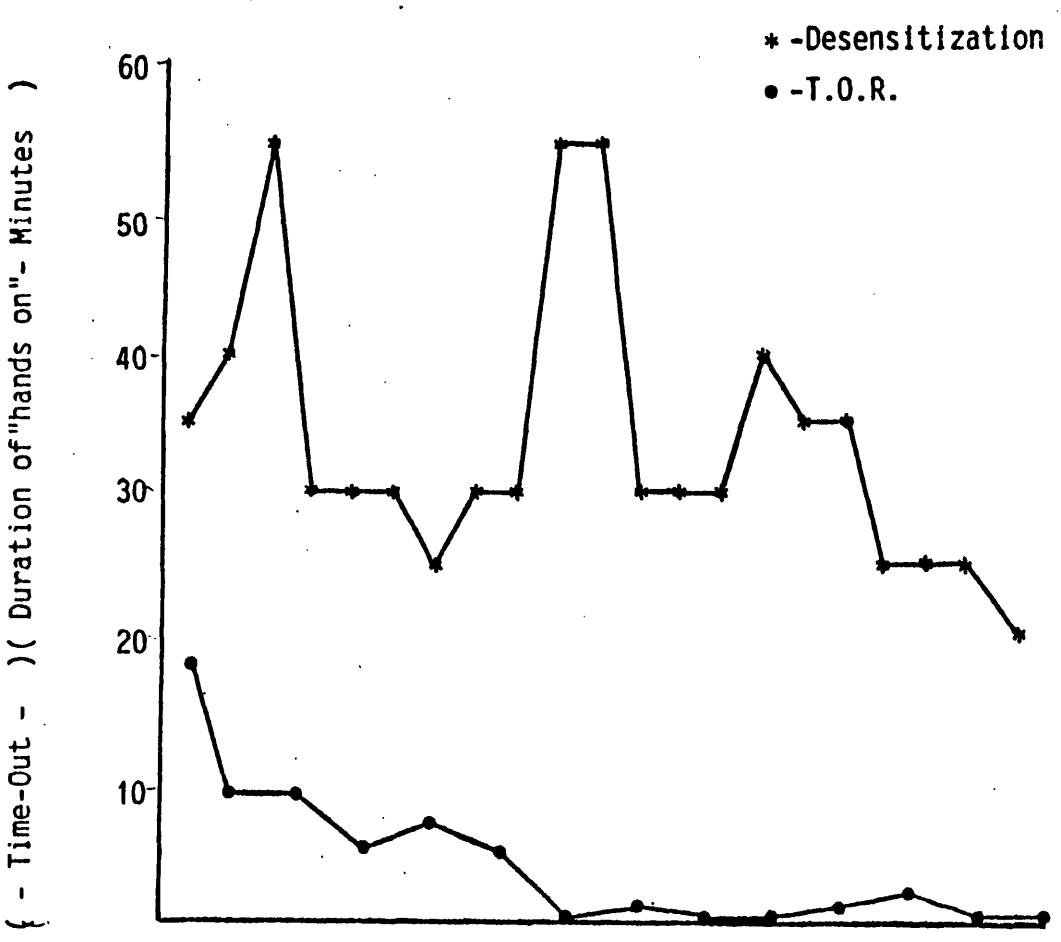
Most ADL activities require a degree of mobility on the part of the patient to move around a home or kitchen or negotiate stairs, escalators or other obstacles found in public places. The re-establishment of independent walking ability several years after injury has been treated with scepticism by many neurologists or rehabilitation specialists. Our experiences however, have shown that such improvements are eminently possible.

Often, the reasons why patients remain wheelchair dependent for so long are due to secondary factors, such as aggressive behaviour, which prevent therapy being applied early on in recovery (as in the case illustrated in 5.2(i)). In many respects this is understandable because physical therapy can be painful to patients with spastic rigidity of the muscles. This, combined with their limited control over emotional responses can easily create a situation where aggressive behaviour predominates.

The combination of desensitisation and time-out used to control 'learned' aggression which interfered with the application of therapy

Sometimes, as a result of such experiences early on in recovery, patients become sensitised to being touched. Figure 8.2(i) illustrates such a case which was treated by a combination of **desensitisation** and time-out. The

FIGURE 8 2(i)



Two methods used to eliminate aggressive response during physiotherapy. The top curve illustrates a "hands on". desensitization programme to reduce anxiety of being touched. The bottom curve shows a Time-Out Room programme used contingently, each time aggression was shown outside the desensitization procedure.

patient, previously described in 7.1(i), resented being touched. This made physiotherapy very difficult because it provoked aggressive behaviour. The sensitivity to being touched was dealt with by passively restraining her, while members of staff "touched" her (placed their hands on her arms, legs, shoulders etc.). A time-out room procedure was implemented for any aggressive behaviour that took place **outside** the desensitisation periods. Two measures are therefore available; (1) during desensitisation; the length of time she struggled or shouted before settling down and accepted the kind of physical contact which is inevitable in physiotherapy, (2) from the time-out programme; the number of aggressive incidents each week.

Figure 8.2(i) shows that there was no improvement in the first measure ($F= 5.43$; $df= 1/6$). She continued to respond in a difficult and uncooperative manner throughout the desensitisation sessions. The other procedure was far more successful, producing a type 1 trend ($F= 26.2$; $df= 1/6$; $p< 0.01$).

Inevitably there was some cross-over effect from the two programmes which, even if not making her more amenable to being touched, did improve her level of control which altered her response to physiotherapy.

Shaping a 'transfer' response in a patient who had failed to learn from a conventional therapy approach

One of the basic exercises in physiotherapy is teaching a

patient to transfer from wheelchair to chair. This is important for independence because it allows the patient to use the toilet or get in and out of bed without assistance.

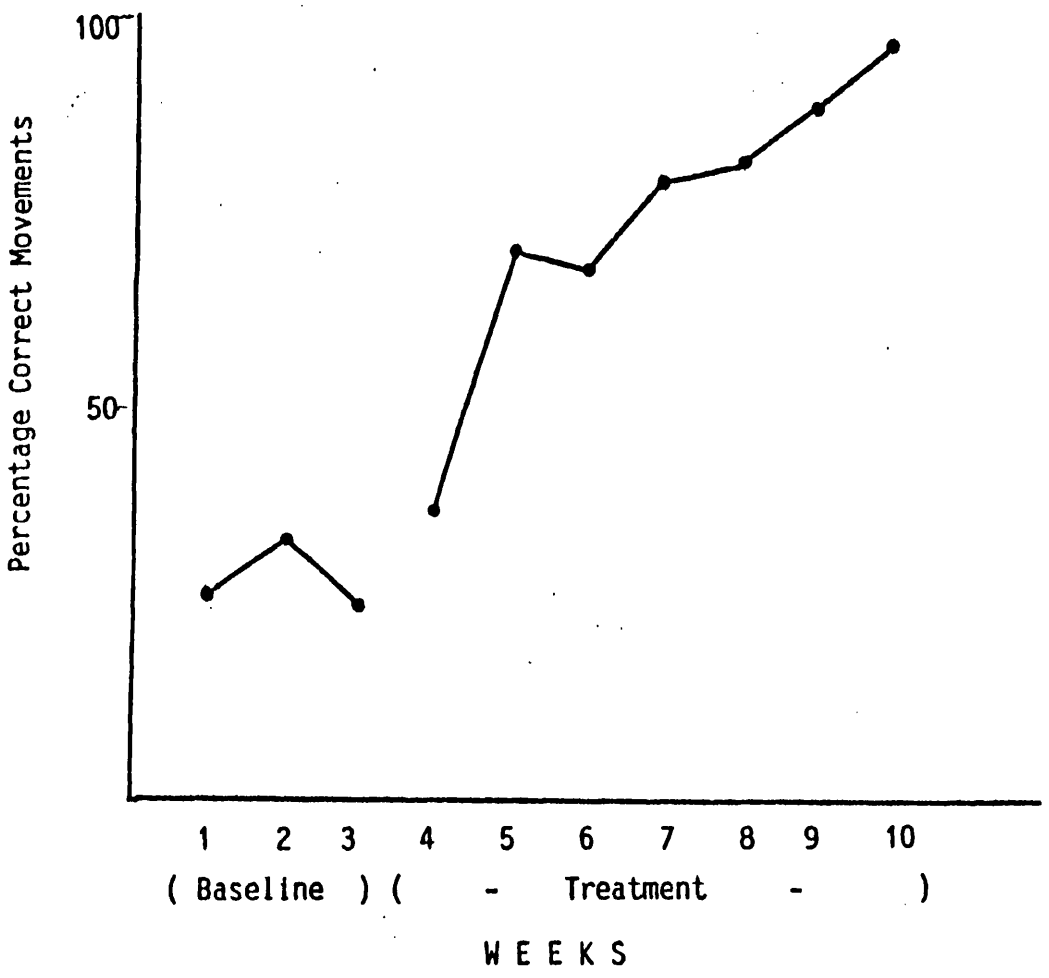
It is sometimes the case that severe brain injury produces cognitive problems that make it difficult for individuals to learn this simple procedure. Possibly, this is because they cannot **attend** to all the units of behaviour which combine to make up this response, also it is possible that the requirements of the procedure are presented in a way that makes it difficult to learn continuity of movement.

This was the case with the patient in figure 8.2(ii). He sustained his injury, five years previously (age 22). The severe brain injury (PTA= 2 months) had left him with a dense right hemiparesis, an expressive dysphasia and diffuse intellectual impairment. Physiotherapists had tried for several weeks, without success, to improve his transfer skills. Until these were established he could not move onto other aspects of the rehabilitational programme which demanded a greater degree of physical independence.

A programme was introduced which made allowance for his intellectual state. Functional analysis of the movements involved in transfer showed that they could be reduced to six units of behaviour:-

(1) Positioning his bottom on the chair.

FIGURE 8.2(ii)



Improvement in transferring from wheelchair to chair using a shaping procedure and contingent reinforcement.

(2) The angle of his trunk in relation to his legs.

(3) Shifting the point of balance forward onto his legs.

(4) Controlled extension of his knees.

(5) Rotation of body from his knees.

(6) Lowering bottom onto chair by shifting his balance and controlling knee flexion.

The training programme was organised to practice each of these discrete movements independently, tokens being awarded contingently for each correct movement. Only when a particular movement was reliably established did the programme move on to practice the next movement, initially in isolation but gradually linking it to the previous movement. The graph shows the success of this procedure, measured against a three week baseline, where shaping had not been employed. A type 1 trend was recorded ($F= 26.68$; $df= 1/5$; $p<0.01$).

Using positive reinforcement to encourage walking

The process of **teaching** a patient to walk does not necessarily mean that they will **spontaneously** walk. This is particularly true of patients who have been in a wheelchair for many years and who have got into the **habit** of wheeling themselves around rather than walking. Figure 8.2(iii) illustrates this problem.

The 29 year old patient arrived for admission in a wheelchair. Like the previous patient, he had a severe head injury (PTA = 5 months) five years previously, having sustained brain stem as well as cortical damage.

Originally, his independent walking ability was affected by a left hemiparesis. His present lack of independent mobility was due to aggressive non-cooperation with therapists, combined with his parent's tendency to treat him as a "cripple" rather than encouraging attempts at independence. He reached the stage in therapy where he **could** walk but **woudn't** walk.

To encourage ambulance the corridor was divided into ten yard lengths. To begin with he was rewarded with tokens, simply for distance walked. As he progressed his gait was **shaped** into a more normal walking pattern. This was achieved by only giving him tokens if he followed the therapists instructions regarding the development of an acceptable gait, e.g.-

(1) Proper paces; his steps had to cross each other instead of stopping parallel. This ensured that each step moved him forward.

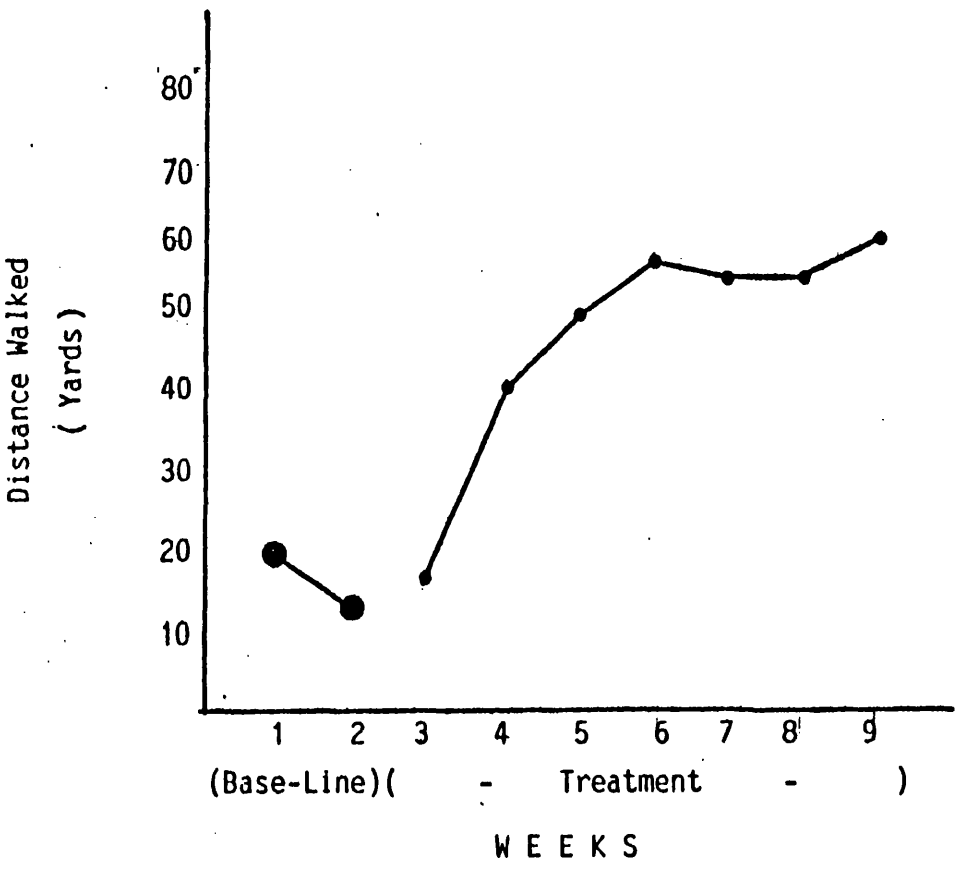
(2) Not adopting a wide - base for his gait.

(3) Maintaining correct body posture.

(4) Maintaining momentum.

Reinforcement was contingent upon him arriving at a ten yard marker having executed a walking pattern acceptable to his current stage of therapy. As therapy progressed the expectations regarding a more normal walking pattern increased, meaning that it became more difficult to earn reward. This reinforcement contingency, rewarding successive approximations to the desired response, proved

FIGURE 8.2(iii)



Increasing the distance of independent walking using a fixed-interval positive reinforcement schedule.

to be successful in increasing distanced walked ($F = 10.69$; $df = 1/5$; $p < 0.05$).

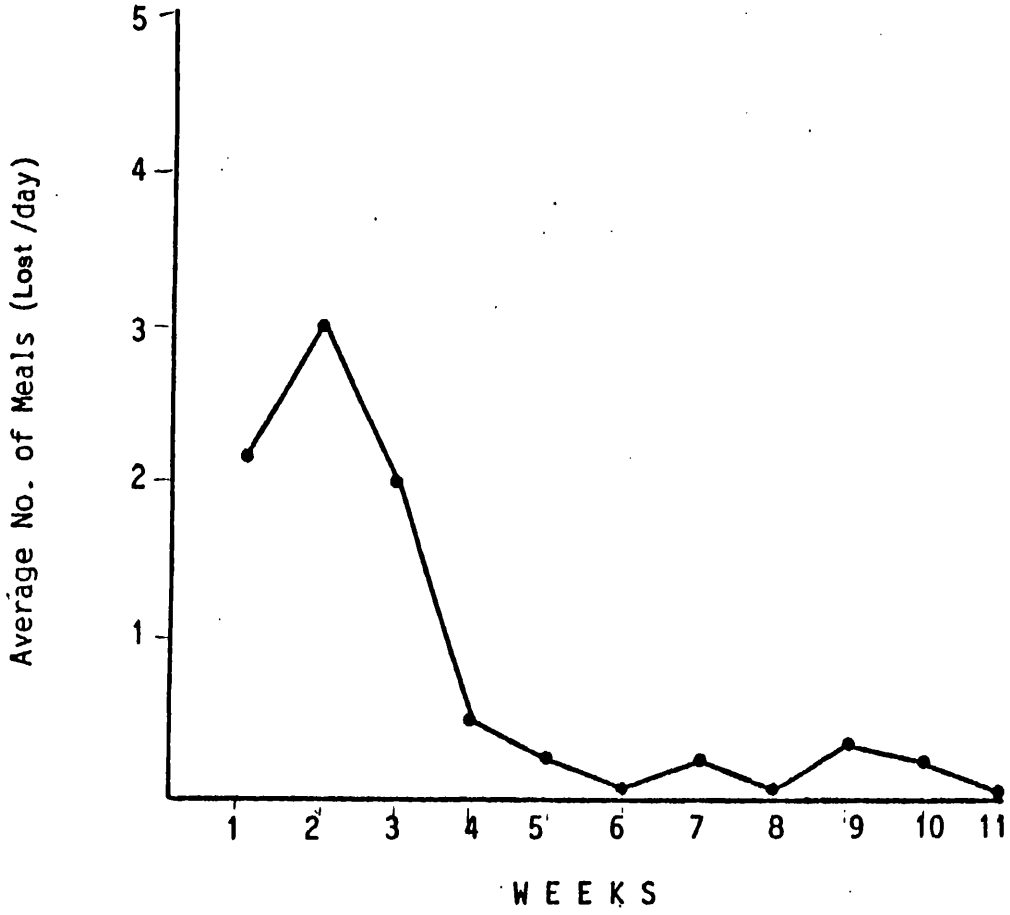
Encouraging walking through the use of negative punishment

Another programme to encourage independent walking in a patient who had the **ability** to walk but refused to **exercise** such ability is shown in figure 8.2.(iv). This 53 year old patient lost most of his left anterior temporal lobe as the result of an assault with a blunt weapon, ten years previously. He had been on a chronic psychiatric ward since that time, although having no history of psychiatric disorder, other than lacking cooperation, enthusiasm and drive.

An effort was being made to find him a place in more pleasant surroundings. This required a greater degree of mobility than had been exhibited over the previous ten years. The mild residual hemiparesis, originally responsible for his wheelchair state, could not now be regarded as neurologically responsible for that requirement. As a result of his early uncooperative behaviour, no further physiotherapy had been offered and his lack of effort and reliance on a wheelchair had been reinforced by hospital staff. Once we were sure that he possessed the physical ability to walk, the only impediment being his lack of effort, a behaviour programme was implemented to correct the problem.

This patient was, unfortunately, somewhat indifferent to

FIGURE 8.2(iv)



Improvement of independent walking using meals as contingent positive reinforcement.

the usual rewards available as positive reinforcers. He did not drink or smoke, had no family to visit or telephone and was not motivated by the 'consumables' available for contingent reinforcement. He did enjoy his food however. Once general agreement had been obtained from ward staff that meals were a very potent reinforcer it was decided to make the receipt of a meal contingent upon him **walking** to the dining room (unaided) to get it. Under the ambit of **meals** we included morning coffee and biscuits as well as afternoon tea and cakes, making it possible to receive five meals a day. If he refused to walk to the dining room for his meal a milk based substitute was offered instead.

The graph shows that although there was some initial resistance, the patient did make the required response ($F= 62.87$; $df= 1/6$; $p< 0.001$) which was maintained for the rest of his stay. Unfortunately, on return to the psychiatric hospital (prior to further placement) the nursing staff failed to reinforce walking and the earlier behaviour was resumed.

A programme of selective reinforcement to encourage swallowing

Some physical problems cross the boundary of theraputic specialties. An example would be swallowing difficulties, which requires a speech therapist to work on lip closure, tongue movement and position, and a physiotherapist to work on posture, to facilitate the swallow reflex. Figure

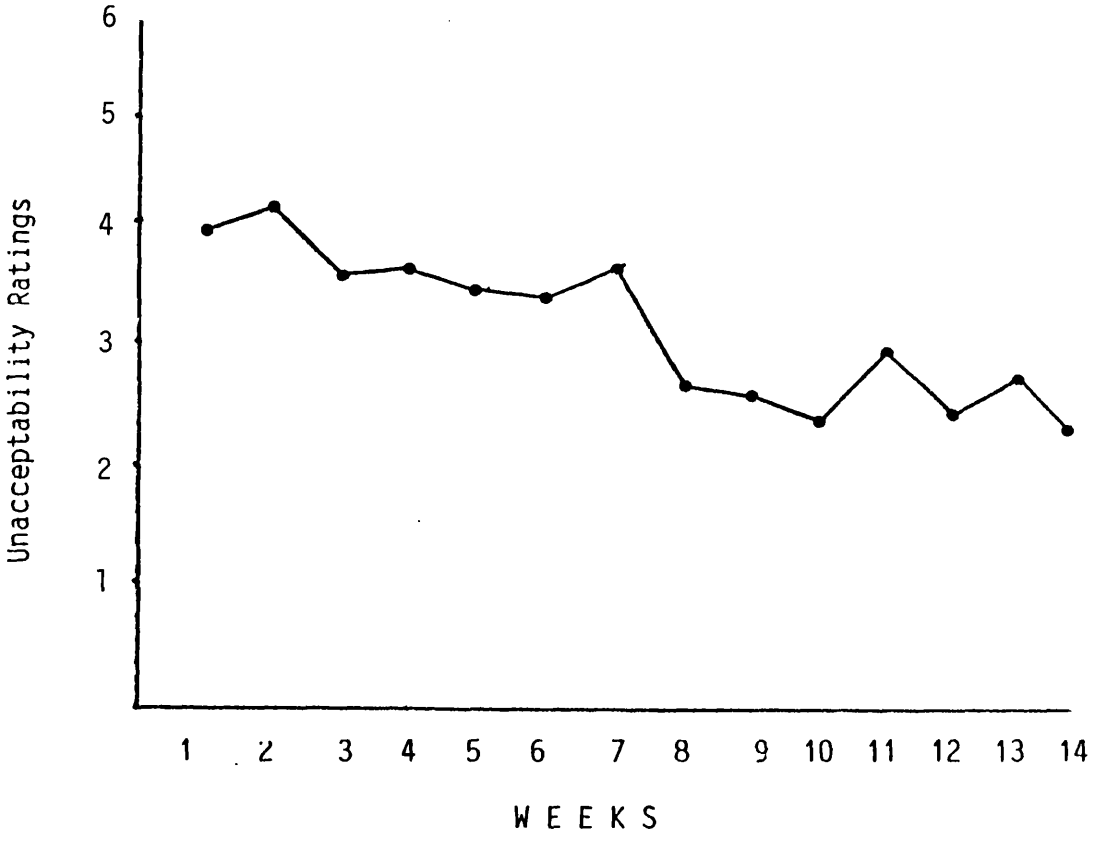
8.2(v) shows how the use of contingent positive reinforcement can be used by therapists working in pairs to improve a behavioural response.

The example is of a patient with predominently brain stem injuries. She had sustained a head injury (PTA = 3 months) 4 years previously and had been fed by a naso-gastric tube since then because she "refused" to make any effort to swallow. The reason for this is almost certainly because during the early stages of recovery, when removal of the naso-gastric tube was first attempted, a tracheostomy scar made swallowing difficult and painful. As a consequence the response had developed aversive consequences.

It was noted that this patient was attention-seeking. Consequently, attention was used to reinforce efforts at swallowing. Advice from the dietician was obtained regarding which foods are easiest to swallow. The programme used a selective reinforcement procedure, delivering contingent positive reinforcement (lots of attention and praise) each time a swallow response followed food being placed in the patient's mouth. If she refused the food or ejected it from her mouth with her tongue the response was ignored.

Again, a shaping procedure was used. Initially, any swallow was reinforced, even a reflex swallow, obtained by placing food at the back of her mouth. During the next phase reinforcement was only given following **attempts** at

FIGURE 8.2(v)



Improved swallowing in a patient who had been maintained on a naso-gastric tube for two years and who refused to attempt to swallow. Progress rated on a 1-6 scale where 6=no effort and 1= purposeful swallow.

a **purposeful** swallow. Eventually, when more solid food was provided, reinforcement was made contingent upon chewing, moving the food to the back of the mouth and a purposeful swallow. Any response which involved screaming, dribbling the food out of the sides of her mouth or purposely spitting it out were ignored.

The graph shows the progress made using this method. It is based on a score giving six possible responses.

(1) Chewing and purposeful swallowing: score =1.

(2) Purposeful swallow only: = score 2.

(3) Reflex swallow with minimal food loss: score =3.

(4) Reflex swallow with substantial food loss: score =4.

(5) No swallow, food dribbled out of sides of mouth: score =5.

(6) Purposefully spitting food out: score 6.

A baseline is not recorded but can be assumed to be a total absence of any kind of attempt at swallowing during the previous 4 years.

A successful treatment effect was recorded, showing a type 1 trend ($F= 10.94$; $df= 1/6$; $p< 0.05$). This patient progressed to eat a full diet, even managing to win the 'eat a doughnut competition' during the unit sports day!

Summary. Shaping procedures appear to be effective in helping patients acquire independent physical ability. Not only do they provide a motivational incentive for patients, (who otherwise would make little effort to re-develop mobility so late after injury) but circumvents some of the 'cognitive' demands of learning by breaking down complex responses into smaller, more manageable units which can be individually practiced whilst providing feedback of results.

8.3 SPEECH THERAPY

Dysphasia and dysarthria are common legacies of severe brain injury. In the case of **traumatic** brain injury, global dysphasia is rare, compared to its incidence in stroke yet in both conditions the impairment shows a substantial degree of spontaneous recovery (Kertesz 1979). Far more common, protracted and socially handicapping are the problems of communication produced by dysarthria. The high incidence of this speech problem is attributable to the mechanical forces which operate during many traumatic brain injuries (deceleration and rotation). These result in movement of the brain around the axis of the brain stem , damaging fibres between the brain stem and higher neuro-anatomical centers. The two nerves important to speech production arise from the **medulla** (IX,X nerves). These control the actions of the palate and pharynx while, in addition, the IX nerve carries sensations from the back of the tongue and throat. Some damage to these nerves is almost inevitable following severe decelerarion injuries or in circumstances where the "second" injury (Jennett 1976) is a result of hydrocephalus, forcing the brain down onto the brain stem. Dysarthria is a frequent result.

Academically and clinically however, dysarthria has been overshadowed by the neuropsychologically more interesting deficit, dysphasia, even though there is some persuasive

evidence that speech.therapy for dysphasia, given in the early recovery period after brain injury (at least in stroke), does not produce any effect that could not normally be expected from spontaneous recovery (Sarno 1981). Whatever the outcome of that controversy, which deals specifically with the effects of brain haemorrhage, there does not appear to be any data on speech therapy delivered to traumatic or stroke patients **years** after injury, at a time when spontaneous recovery can no longer be expected.

Because most of the patients admitted to our unit suffered brain damage through head injury their primary problem is dysarthria, dysphasia being present in a less severe form and then more often as a higher level language disorder. The examples to be described are therefore divided into 2 major areas, (1) the **production** and **enunciation** of speech, (2) the **content** of conversation. This is in place of nominal, automatic or propositional speech patterns that are affected by dysphasia.

Positive reinforcement to improve production of polysyllabic speech

Figure 8.3(i) demonstrates a treatment procedure to improve enunciation in a severely dysarthric 22 year old patient, five years post injury. He had suffered, like most severely dysarthric patients, a brain stem injury,

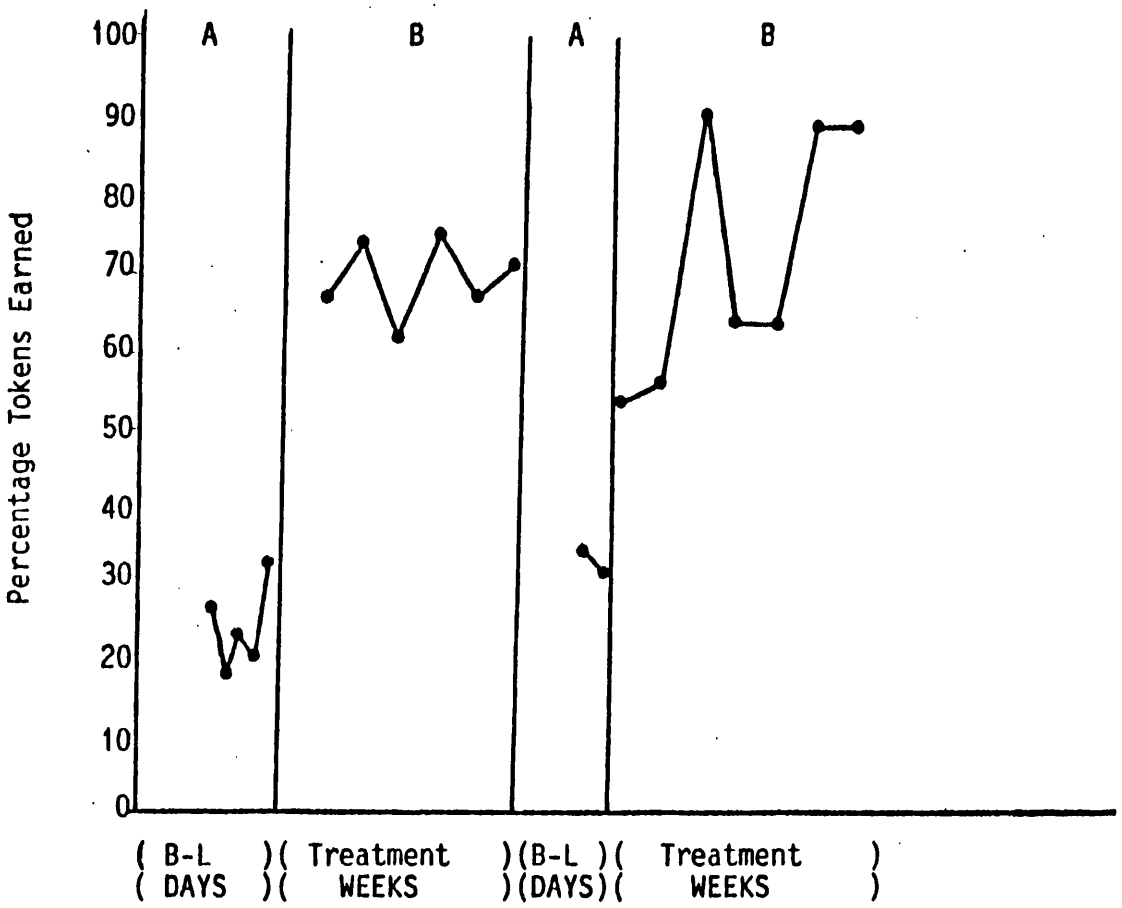
so the dysarthria was a feature of quite severe oral dyspraxia.

Initially, the speech therapist used a conventional (non-behavioural) approach but was unsuccessful in producing the kind of sustained effort and cooperation needed to make progress. The patient had reached the stage where he could produce speech sounds but he would not make the effort at linking these sounds together in order to produce polysyllabic speech.

The behaviour programme made positive reinforcement (initially a smartie and later tokens to exchange for smarties), contingent upon the enunciation of polysyllabic speech sounds. A five day baseline measured his usual rate of responding before tokens were introduced. The sudden improvement in response can be directly measured by the frequency of contingent reinforcement.

There were no type 1 trend effects in the data ($F= 0.07$; $df=1/4$) but a comparison of the treatment mean, during the first A phase, with the average for the baseline period, shows that a type 2 treatment effect was obtained ($t= 4.51$; $df=5$; $p<0.01$). This patient remained difficult and unmotivated, with the result that a temporary pause in the programme, when he went home on leave, produced a marked deterioration in the clarity of his speech. The subsequent improvement ($t= 6.48$; $df= 6$; $p< 0.01$) when sessions restarted confirms the effect of the

FIGURE 8.3(i)



Positive reinforcement programme to improve cooperation and production of clear speech during individual therapy sessions.

reinforcement contingency in achieving cooperation and effort to produce improvement in speech. Unfortunately, the above programme could not be described as a clinical success because the response to reinforcement never generalised to produce conversational speech, neither could it be controlled by non-contingent reinforcement.

A multiple baseline study showing the effect of a time-out room programme on the generalisation of clear speech

Generalisation of non-dysarthric speech has always proved difficult. Attempts to encourage the production of clear speech through contingent positive reinforcement failed, probably because there was so little of it to reinforce! This, plus the fact that the predominently frontal lobe patients involved in this study appeared relatively impervious to the reasons for such reinforcement led to the assumption that a more successful outcome would be likely if contingent negative punishment were used.

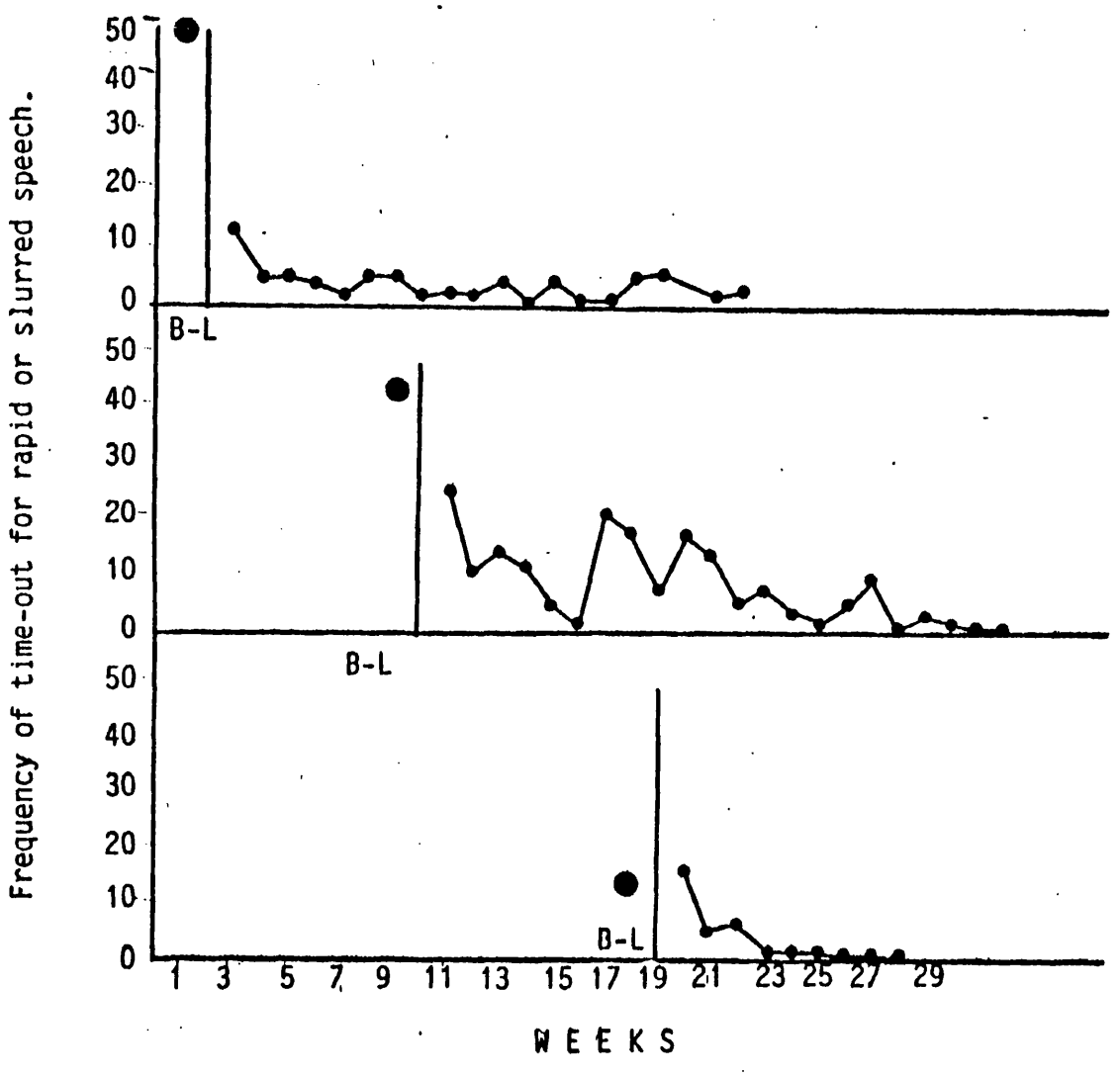
Figure 8.3(ii) provides an example of this procedure using a three-subject multiple baseline design. The three patients all had communication problems subsequent to sustaining severe head injuries. Problems involved either rapid or slurred speech and the patients had remained in this state for between two and four years. All had shown the ability to speak clearly in speech therapy sessions,

using a treatment which slowed the production of speech to not more than five words per breath. This required the patient to develop a pattern of inhalation and swallowing, slowing down the production of speech to a rate that allows the patient to maximise the techniques learned in therapy.

This improvement was showing no sign of generalising outside of therapy sessions however, even though contingent, positive reinforcement was given for clearly enunciated speech. The reason for this was unclear. It probably has something to do with the fact that all the patients had major frontal lobe injuries which affected their ability to divide attention (Van Zomeran et al 1984) consistently between their breathing pattern, word rate and any other activity engaged in at the time. If this was the case then the positive reinforcement was not helping the patients direct attention to the relevant aspects of speech. In an attempt to help focus their attention, the more 'noticeable' form of negative punishment reinforcement was provided.

The programme involved the patient receiving two minutes in the time-out room (preceded by the discriminative simulus - **"speech"**) every time the incorrect speech pattern was noted. To ensure a uniform approach by staff, for this somewhat irregular use of time-out, the speech therapist produced a tape recording of each patient's clear speech during therapy. This was used as a general

FIGURE 8.3(ii)



Multiple-baseline design showing the effect of a time-out procedure on the production of slurred or rapid speech. This represents an attempt to generalize skills learned in speech therapy to the less "controlled" ward environment.

model for the patient's speech. When satisfied that the treatment team understood the speech requirements of five words per breath, the treatment programme began.

In each case it produced a successful treatment effect. The first patient showed an immediate type 2 improvement in controlled speech ($t= 70.15$; $df=6$; $p< 0.001$), which was maintained throughout the treatment period. The response of the second patient was also immediate. A comparison of the baseline with the first seven **days** of treatment shows a significant effect ($t= 9.57$; $df=6$ $p< 0.001$). A type 1 trend is also recorded for the first six weeks ($F= 25.74$; $df= 1/4$; $p< 0.01$) but was interrupted by the characteristic loss of control seen in frontal patients. After that the behavioural control remains unreliable but there is a significant trend over the remaining 8 weeks of treatment ($F= 9.96$; $df=1/6$; $p< 0.05$). Patient 3 also shows a significant type 1 trend ($F= 16.31$; $df=1/6$ $p< 0.05$), the inappropriate speech pattern being eliminated after only six weeks.

Examples of behavioural techniques to improve the content of speech

In addition to improving the **production** of speech, behavioural methods can be used to control its **content.** Two examples are given. The first (figure 8.3(iii)) involves trying to reduce the frequency of swear words in conversational speech as part of a more general social

skills programme.

The patient was a 46 year old man who had sustained a head injury (PTA unknown) nine years before and, like one other of the subjects in this study, spent most of the subsequent years on a chronic psychiatric ward where his behaviour had become more and more institutionalised. In addition to adopting a very aggressive manner he frquently used a wide range of swear words, making his behaviour most unacceptable. Damage to the frontal lobes was a major legacy of his injury. This meant that much of his behaviour had a disinhibited flavour, with little control over the **initiation** of a response. Careful observation confirmed his ability to control the **development** of that response however. Consequently, the sudden impulsive swearword in response to frustration could be accepted as being beyond his control but his tendency to continue a tirade of obscenities, many of which were of his own construction and used to villify specific individuals, was totally unacceptable.

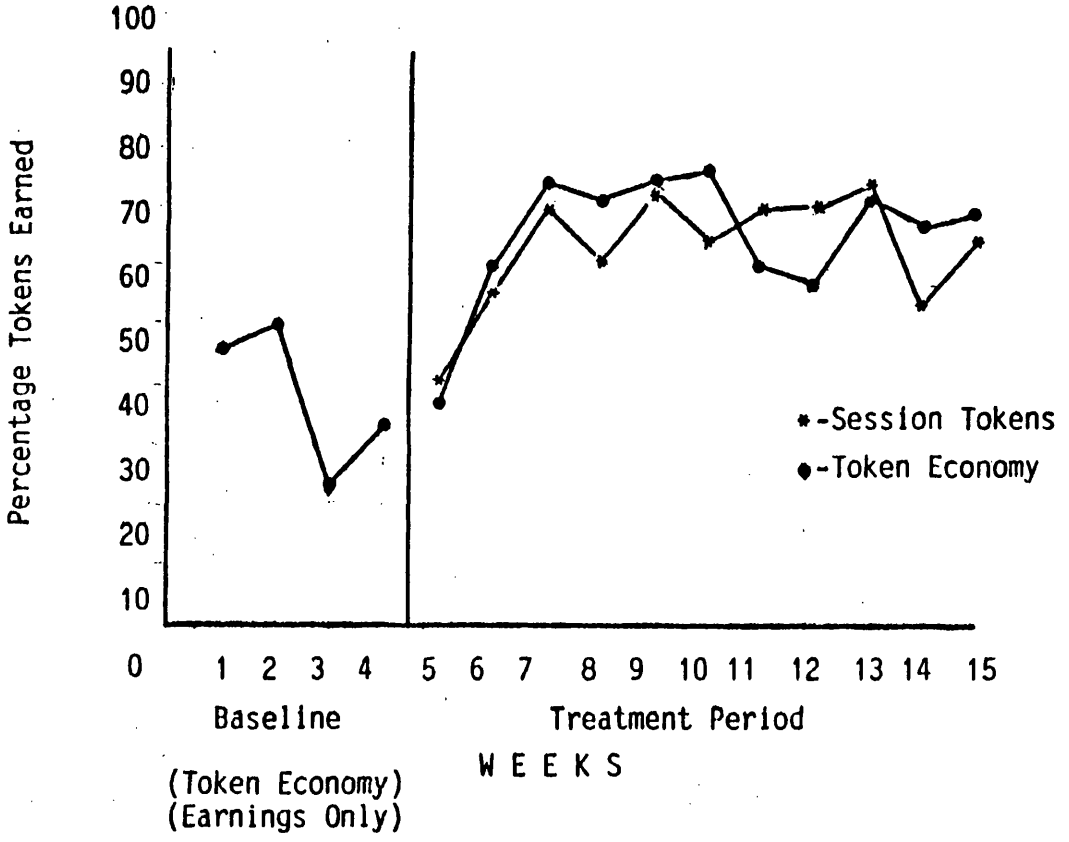
Initially an attempt to modify the content of speech was made using non-contingent negative punishment (loss of tokens which were paid on a fixed interval basis). This meant that if he directed a string of invectives at anybody he would lose his tokens for that period, but may not learn of his loss until 15 or more minutes later. The procedure failed, so a sessional programme, using contingent positive reinforcement, was introduced. This

was designed to increase the frequency of reinforcement by allowing more opportunity for conversation to take place in a controlled setting. The idea was to reward periods of appropriate speech, with the hope that this might generalise from within the structured interview to the more general ward environment.

The programme was based on a number of half hour sessions, during which different members of staff would keep the patient engaged in conversation. The idea was to get the patient to do most of the talking, providing reward (a token) for every two minutes of non-swearing speech. These tokens were accumulated and could be exchanged immediately after each session for a drink or chocolate.

Figure 8.3(iii) shows the steady growth in his sessional-token and token-economy earnings. The significance of this comparison is that he was still capable of losing tokens on the general ward programme for swearing. The parallel improvement in general token earnings after the start of this programme ($r = .700$; $df = 9$; $p < 0.02$) suggests that the improvement seen in sessions generalised to other treatment activities. A type 1 trend towards improvement is not found ($F = 0.03$; $df = 1/5$), but comparison of the last 14 days of the baseline with the first 14 of treatment does confirm a type 2 trend ($t = 4.33$; $df = 12$; $p < 0.01$).

FIGURE 8.3(iii)



During a twenty minute daily session, swearing behaviour was reduced by reinforcing two-minute periods of conversation without swearing. This figure shows both the improvement in non-swearing conversation, measured by the increase in session token earnings, and the concomitant improvement in general behaviour, measured by the increase in earnings on the token economy.

A different programme for **control of speech content** is shown in figure 8.3(iv). This patient had developed the tendency to speak on stereotyped conversational themes which included the history of her accident, her desire to write a book, her admiration for psychologists, doctors or any other health profession. On first meeting this lady she could appear a reasonable conversationalist, but she could not control her tendency to "button hole" any likely listener and launch into what became known as her "repetitive themes". Even more inappropriate was her disinhibited manner, which produced other repetitive phrases such as -" I think you're wonderful, you really are".

The negative effect these themes had on people was so great that they significantly prejudiced her chance for long term care. This lady was capable of living in the community with a companion. Her conversation was so insufferable however, that unless it could be controlled the alternative - institutional care would be more likely.

In many respects, this behaviour was similar to the inappropriate speech of a schizophrenic girl described by Ayllon and Michael (1959). This responded to selective reinforcement; attention given to appropriate speech but not inappropriate. Such a method was initially tried here and continued for a number of weeks. It failed to produce any difference in this conversational tendency (see fig

5.2(vi)). When this was discussed earlier, the probable reason for this programme failure was considered to be due to her predominantly frontal lobe injury preventing her perceiving either the effects of her behaviour on others or the application of TOOTS by the ward staff.

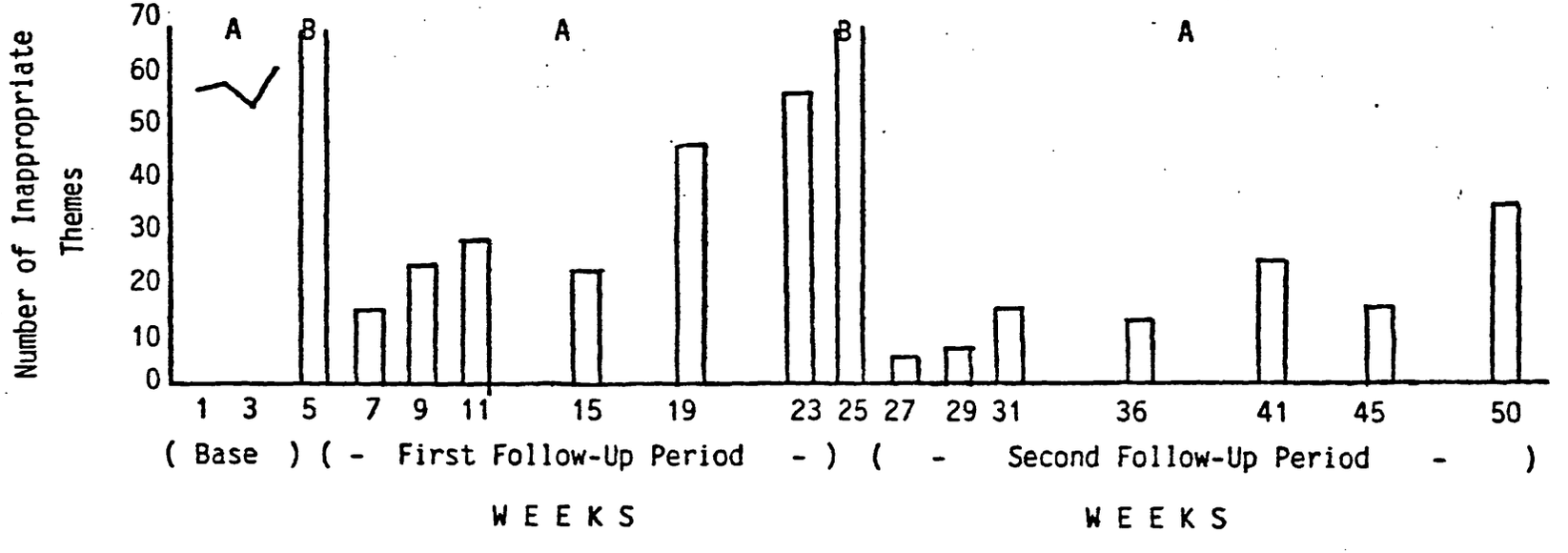
Consequently an effort was made to penetrate what appeared to be a 'cognitive barrier'. Like the patients described above this lady appeared unable to attend to a more appropriate response pattern. To overcome this we combined a behavioural and cognitive approach, based on a sessional, massed practice technique we described as **cognitive-over-learning.** For two hours every day the patient was made to repeat her repetitive themes, prefixed by the words -" I must not say", for example:

" I must not say you're wonderful you really are"

After every two minutes of such repetition she was allowed a break of one minute before continuing. One theme was pursued for fifteen minutes and then the staff member and the theme changed. Two such one hour sessions took place every day for five days.

Figure 8.3(iv) shows that this was successful in immediately reducing the frequency of these conversational themes. Comparison of the last 14 days of baseline data with the 14 baseline days after implementing the cognitive-over-learning programme shows a significant reduction in the frequency of inappropriate themes ($t = 5.73$; $df = 12$; $p < 0.001$). It was five months

FIGURE 8.3(iv)



Effect of massed practice procedure using a "cognitive over-learning" (C-O-L) strategy to modify the content of speech in a patient with severe frontal lobe injury.

before the themes returned to baseline levels. At that time the programme was repeated with similar effect but without a return to baseline. Comparison of the means for the seven weekly time samples with the original baseline data shows a significant difference ($t = 10.81$; $df = 6$; $p < 0.001$). This patient is now living in the community with a companion and maintaining a reasonable content of conversation.

Summary Speech therapy has proved a difficult area in which to produce clinical improvement of a kind which can be generalised to allow successful communication in community settings. The frontal lobe involvement in the patients studied may militate against a form of associational learning that relies on an operant approach and the ability of an individual to recognise social cues.

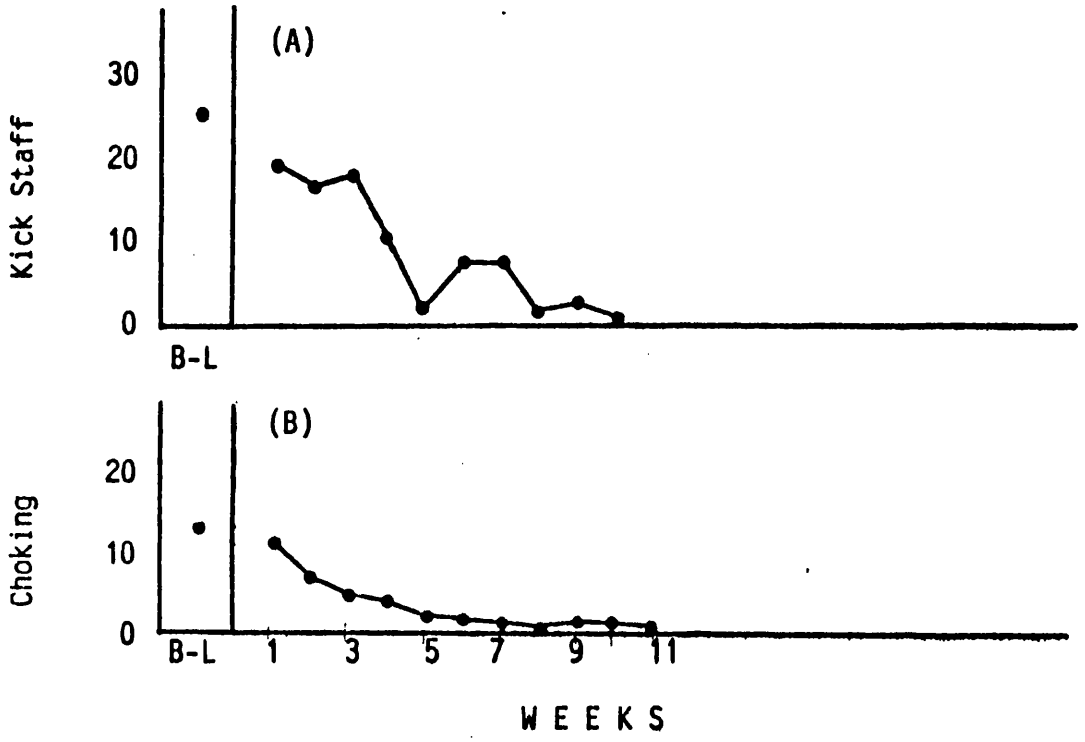
8.4 Behaviour Problems With "Inaccessible" Patients

Severe brain injury can produce cognitive deficits of such magnitude that the patients contact with the world may be limited to only one or two modalities. This problem becomes particularly acute when those "intact" modalities are not ones that make communication easy. Often cognitive impairment will produce or coincide with behaviour problems, either because the patient becomes frustrated due to his inability to communicate or the damaged cognitive mechanisms are part of a more widespread confusional state with agitated, restless and often aggressive behaviour. Figures 8.4(i,ii) illustrate the value of behavioural methods in circumstances where the patient is unable to communicate or translate perceptions of the environment in "cognitive" terms.

A time-out room aproach to reduce aggression in a man with no language ability

The example in figure 8.4 (i) is of a patient who lost nearly the whole of his left temporal cortex in a road traffic accident and, in addition, sustained a more global cortical injury, severely limiting any non-verbal understanding that might be present. He was unable to communicate, or give any sign (during the early stages of treatment) of being able to understand gestures. In addition he could not walk because of general weakness and a loss of balance, mainly as a result of being

FIGURE 8.4(i)



The effect of contingent negative punishment on aggressive behaviour,which prevented therapists help re-establish a swallow reflex in a patient who suffered frequent choking. This patient had suffered severe cognitive impairment and had no communication skills. The reduction in aggressive behaviour (graph A), allowed therapists to intervene and improve chewing and swallowing with a concomitant reduction in the frequency of choking.(graph B).

restricted to a chair for several months whilst a patient on a neurosurgical unit. He was incontinent of both urine and faeces and frequently choked, even on semi-solid food.

Because of the danger this latter problem presented, attempts to improve swallowing and chewing were considered a high priority. The administration of iceing and proprioceptive neuro-muscular facilitation therapy (PNF) was made very difficult by his tendency to kick out at anybody who came within range. To protect themselves, the staff at his previous hospital erected a wooden barricade around his legs to avoid being kicked on the shins while administering basic nursing care.

Various methods of controlling this problem had been tried, some based on drug therapy, others using admonishment or some other form of intervention which relied on verbal or visual communication to make him understand the inappropriate nature of his behaviour. Such methods met whith little success but he did show a good response to a behaviour programme that was able to give non-verbal feedback and "condition" a response without having to rely on cognitive interpretation. Each time he attemted to kick a patient or member of staff, he was placed (in his wheelchair) in the time-out room. The frequency of his kicking quickly reduced ($F = 20.06$; $df = 1/6$; $p < 0.01$) allowing speech and physiotherapists to provide the necessary therapy. It is interesting that a

corresponding improvement in swallowing occurred, the reduction in the number of choking incidents being highly correlated with the reduction in aggressive responses ($r = .849$; $df = 8$; $p < 0.01$).

A classical conditioning procedure to improve feeding behaviour

Figure 8.4(ii) shows the flexibility of a behaviour procedure with another patient who had sustained serious cognitive impairment following head injury. From a communication or social point of view he was "inaccessible", due to a severe dysphasia which incorporated jargon and echolalic qualities. He was thought to be cortically blind but later discovered to have severe astygmatism which, for practical purposes, restricted his vision so much that it made him virtually blind. Because he could not 'see' the presence of others or understand, through verbal cues, their intentions, he was difficult to nurse. Changing clothes, bathing or feeding resulted in an outpouring of jargon type speech, with attempts to strike out in all directions, making him a major management problem.

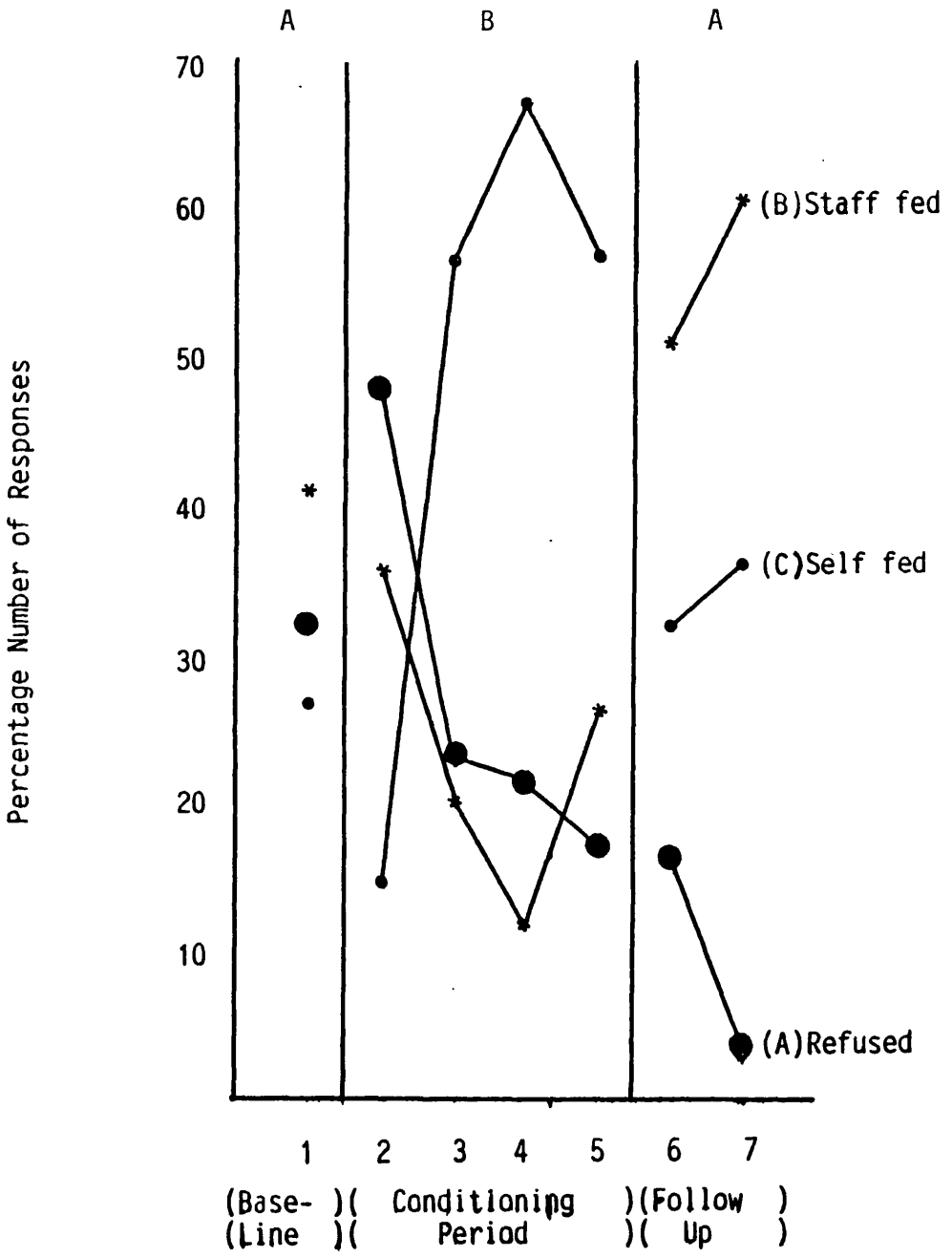
Operant conditioning, using a time-out room procdure for aggression, produced no improvement in his cooperation so an attempt at classical conditioning was made. Efforts were directed at his eating behaviour. The procedure involved providing an auditory cue, (a tone of

approximately 40 db). Awareness of this stimulus was confirmed by observing his ability to orient to the sound source (by head movements). This was accepted as reasonable perception. The cue was paired with the pressure of a hand on his shoulder (as an additional tactile cue to associate with if the sound pattern failed).

Records were kept of the patient's response once a spoon was placed at his lips. There was not a fixed number of presentations for each meal; the number varied according to the foods consistency, his appetite and behaviour. His response was recorded as (A) aggressive refusal; (B) passive acceptance of the food (assisted eating) and (C) holding up his hand to take the food to feed himself. An A-B-A design was made possible because the programme was, in the first instance, administered only for a few weeks to see if it worked.

The graph shows the response of each behaviour category, expressed as a percentage of the total number of responses for each week. Statistical analysis, using a students t, compared the responses in each category. These were expressed as a decimal fraction of all the responses. Comparison was made between the baseline and treatment phases (**A-B**) and baseline and follow-up phases (**A-A**). For the **refused food** and **self fed** conditions the variance in the data was too great to "legitimately" perform a standard t test, therefore a Bailey's Test,

FIGURE 8.4(ii)



Effect of a classical conditioning procedure, reducing the number of rejections of food and increasing self feeding behaviour.

which allows for inequality of variances, was performed.

The analysis did not show any significant change in the number of **refused food** scores between the **A-B** phase ($t = 0.436$; $df = 25.97$) but there was a significant reduction when the baseline and follow-up phases were compared ($t = 4.09$; $df = 19$; $p < 0.001$). Assisted **feeding by staff** also failed to show any significant change between the **A** and **B** phase ($t = 1.59$; $df = 33$) but does between the baseline and follow-up ($t = 2.39$; $df = 19$; $p < 0.02$). **Self feeding** shows significant changes between the **A-B** phase ($t = 2.22$; $df = 16.8$) but the baseline - follow-up comparison failed to reach significance ($t = 0.498$; $df = 19$). In the three conditions there was a significant difference between the treatment period and follow-up (A) $t = 2.89$; B) $t = 3.94$; C) $t = 2.08$; $df = 40$; $p < 0.05$).

The data provide a mixed treatment outcome. Although they point to an improvement in patient management, (in the sense that he refused less food and became easier to feed) they also suggests that the conditioning procedure itself - the establishment of self-feeding in response to a cue, had not consolidated. The programme did however provide a framework for the staff who continued to use the tone stimulus. This was associated with an improvement in his behaviour, an increase in weight and getting the patient accepted for long term care.

Summary and Conclusions

Behavioural approaches, employing different conditioning procedures and principles of learning, have been used successfully in many aspects of brain injury rehabilitation, either achieving progress after conventional approaches have failed or in facilitating the therapy procedure itself. The value of this approach for patients with major cognitive impairment and important perceptual deficits is particularly encouraging although one might have predicted such success from the animal work showing conditioning in decorticate mammals (see Oakley 1983 for a review). The constraints on this form of learning mainly involve the apparent insensitivity or lack of awareness of social reinforcement or other cues that normally influence behaviour. This was particularly a feature of those patients with primary frontal lobe involvement. The nature of this problem needs clarification if behavioural approaches to brain injury rehabilitation are to expand. This will therefore form the subject matter of the next section.

SECTION 4

THE ROLE OF ATTENTION IN BEHAVIOURAL LEARNING AND REHABILITATION

CHAPTER 9

THE EFFECT OF AN IMPAIRMENT OF ATTENTION ON BEHAVIOURAL LEARNING

Introduction

Problems of attention and concentration are frequent legacies of severe brain injury, often interfering with a patient's ability to participate in, or co-operate with, rehabilitative therapy. The significance of attention disorders has, until recently, largely been obscured by the emphasis given to the effects of memory impairment following brain injury (Newcombe 1979). This emphasis is beginning to shift to include the role of attention, possibly because of the central part that attention is now assumed to play in information processing models of memory (Atkinson and Shiffrin 1968; Craik and Lockhart 1972; Underwood 1978).

The importance of considering attention is illustrated by the fact that patients with even mild or moderate injuries may experience problems of concentration or speed of information processing which may continue for months (Gronwall and Sampson 1974) and sometimes years after injury, (Miller 1970; Van Zomeren and Deelman 1978; Van Zomeren, Deelman and Brouwer 1984).

Attention problems and behaviour

Attention disorders following brain injury have been described since the beginning of this century. Meyer

(1904) made reference to the difficulty that many traumatically brain injured patients have in concentrating, even during simple and interesting activities. The problem was also mentioned by Conkey (1938) who described the poor performance of head injured patients on tasks measuring concentration and focused attention. Goldstein (1939) also suggested that one of the most characteristic features of the "brain damage syndrome" was a "forced responsiveness" to stimuli. He thought that the patient's difficulty in concentrating on a task was due to involuntary attentiveness to other, non-essential information which was available at the same time. Similar comments were made by Ruesch (1944) and Denker and Lofving (1958) during observation of patients performing psychological tests.

McGhie (1969) describes the most common form of attention deficit associated with brain damage as, "the failure of the normal inhibitory process resulting in distractability". Patients are unable to focus attention on a particular stimulus because they are distracted by, and forced to respond to, external noises which the normal individual would be able to ignore. More recently, this problem has been recognised by Ben-Yishay, Rattock and Diller (1979), who refer to disturbances of concentration which they describe as "impersistence"; the inability to sustain focused attention long enough to complete even simple perceptual or motor tasks.

Attention Deficits and Information Processing

Shiffrin and Schneider (1977) describe two modes of information processing which help us understand the effect of an attention deficit on behaviour. They describe an attention-dependent form of **controlled** processing and a relatively attention-independent procedure called **automatic** processing. Controlled processing is required when a person has to cope with new information. It is a **conscious** mode of processing which is limited in its capacity, mainly because of the attentional requirements of the situation.

Learning a new task requires an individual to focus all his attention on the training procedure; this ensures efficient information processing. This effort will need to continue until the response is learned. The more the response is repeated the more "automatic" it becomes and the less (conscious) attention is needed until, in the end, even quite a complex response (for example driving a motor car) can be made in an almost automatic way.

Automatic processing, therefore, occurs without conscious control and proceeds without stretching the capacity of the information processing system. Because little attention is given in the automatic mode it allows an individual to expand his processing capacity. When for example, one has learned to drive, it is possible to both drive and listen to the radio at the same time,

whereas before driving proficiency had been obtained, the radio would have been an unwelcome distraction in the learning process.

Automatic processing following brain injury. Observations of behaviour following severe brain injury suggest that many automatic skills are affected, to the extent that many patients are forced to revert back to a more conscious (controlled) form of information processing. An example would be the disruption of walking ability, when brain injury damages the sensory-motor cortex. When this happens, a previously over-learned and automatic skill is turned into a complex motor response; the patient must attend to many basic functions involved in walking (for example, joint position, limb placement, posture and balance). The once fluent and effortless procedure becomes slow and hesitant, requiring the person to monitor several actions to achieve control and co-ordination of movement.

In this shift from automatic to controlled processing, a brain injured patient is ironically forced into **dividing** attention, at a time when information processing capacity is already reduced. Consequently, thinking becomes slow, often lacking the acuity and flexibility needed to rapidly adjust ideas (and behaviour) to changing environmental situations. Concentration is affected and individuals find that they are unable to do many things which, before their injury, were carried out in an almost

effortless manner.

Types of attention disorder. Two basic forms of attention disorder are revealed in this situation. One has been described as a **focussed attentional deficit" (FAD)** which occurs when an automatic response is replaced by a controlled response (the above example of learning to walk illustrates a FAD). The other disorder of attention, recognised by Shiffrin and Schneider (1977), is described as a **"divided attention deficit" (DAD)** which occurs when controlled processing is in use and where the limitations of the system fail to accommodate all the information necessary for optimal task performance.

Deficits of this kind are common in every day life (see Reason 1979). Van Zomeren (1981) gives an example of a DAD experienced when one has to cope with a variety of new information, such as might occur if one asked a stranger the way to an address in an unfamiliar part of town. The complicated list of instructions is more than most people can process within a specified time and, inevitably, one is left having to repeat the request for directions further down the road. This particular deficit probably explains why, for example, brain injured patients are unable to cope with more than one or two items of information on a shopping list. Many patients who complain of a memory impairment affecting day to day behaviour describe the frustration of going to the corner shop and only being able to remember one or two, out of

several items. Probably this can be explained as a limitation on their memory span, which can be explained as a divided attentional deficit.

In support of this divided attentional deficit, Miller (1970) showed how the more alternatives a patient has to respond to, the slower and more unreliable his response will be. Miller felt that the effect of a head injury must be to slow down decision-making and therefore information processing, a conclusion later reached by Gronwall and Sampson (1974) and Van Zomeren and Deelman (1976). This latter study, using a reaction time task, found that the amount by which the rate of information processing was slowed down was proportional to the length of coma after injury.

Reason (1979) also provides an explanation of the kind of errors we make in our day to day behaviour. This parallels many of the ideas proposed by Shiffrin and Schneider and appears to explain well the difficulties presented by the head injured patient. Reason describes different forms of conscious control over behaviour. For example, during the learning phase of an activity, the unskilled person relies heavily on the feedback available from a task and therefore consciously attends to the activity (controlled processing). Once the action is learned, the individual's performance is controlled by a series of **"pre-arranged instruction sequences"** that act independently of feedback information (automatic

processing), thus leaving the individual free to concentrate on other aspects of the same, or different, tasks. Reason suggests that **"critical decision points"** occur, even in familiar and well practised activities. If such a point occurs when actions or situations are common to two or more behaviours, then failure to attend correctly to the behavioural alternatives may result in a completely inappropriate behaviour occurring. The level of complexity of a particular behaviour and the predictability of the environment will influence the frequency with which these **"attentional switches"** need to occur.

CHAPTER 10

THE EFFECT OF AN ATTENTION DEFICIT ON DISCRIMINATION LEARNING AND ITS IMPLICATIONS FOR REHABILITATION OF THE SEVERELY BRAIN INJURED

Introduction

Miller (1980) argues that success in rehabilitation is prejudiced by the fact that conventional therapies do little to restore lost functional capacity after brain damage. He suggests that the emphasis in rehabilitation must be to develop more "relevant" methods for retraining the brain injured in the re-acquisition of skills necessary for activities of daily living.

Miller also points to the lack of data describing the learning **ability** of these patients, to help those who are concerned with the retraining of functional skills after brain injury. He suggests that until such data is available an alternative source of information can be obtained from an analysis of the learning characteristics of another group of brain damaged individuals - the mentally-handicapped. There is a substantial literature on the retraining of such patients, and superficially at least, Miller feels that the two groups may have some characteristics in common. If the parallel has any validity, then the special education literature may have

important implications for rehabilitation after head injury.

One important characteristic which both groups of patients share and which will inevitably affect rehabilitation is their slow rate of learning. Wood and Eames (1981) described this as a prominent feature affecting behavioural learning and rehabilitation in severely brain injured patients. The slow rate of learning in mentally-handicapped patients has undergone considerable investigation and Zeaman and House (1963) suggest that the reasons for such learning difficulties cannot be explained as a failure of instrumental learning (operant conditioning), but on the basis of attentional capabilities. They found that in a group of mentally handicapped patients the difference between fast and slow learning was not determined by the rate of improvement from trial to trial **once** learning had started, but rather in the number of trials needed **before** learning started.

In their experiments they presented subjects with two stimuli, differing on a number of dimensions (colour, size and form). The subjects were rewarded if they pointed to the stimulus defined by the experimenter as correct (e.g. blue, on one of the dimensions). They found that the normal and mentally handicapped groups did not differ in the **rate** of moving from the level of chance performance to 100% correct performance. Where they differed was in the number of chance responses which

occurred **prior** to learning. Whereas normal subjects needed few trials to start moving from chance to 100% correct responses, the mentally defective subjects remained at the chance level of performance for an inordinate length of time before showing evidence of learning. Once learning occurred, progress from the chance level of performance to the criterion was at a similar rate to that made by the normals.

From these results, Zeaman and House concluded that an inability to attend selectively to the relevant stimulus cues was the main reason for poorer overall performance on discrimination learning in the mentally handicapped subjects. The defect of learning was therefore a secondary effect of an attentional deficit.

Fisher and Zeaman (1973) reviewed the importance of attentional components in learning and suggest that in the Zeaman and House paradigm, learning to respond to the correct stimulus can be broken down into two processes, (1) learning to attend to the correct dimension and, (2) learning to associate the correct value of that dimension with a reward. If the subject fails to attend to the correct dimension, performance over a series of trials would be expected to remain at chance levels. However, once a degree of selective attention had been established, learning could occur, determined by the subjects ability to associate a particular response with a specific stimulus cue.

The learning that takes place in rehabilitation often includes **behavioural skills** appropriate to independent daily living such as, use of public amenities, food shopping, driving skills etc,. These involve being aware of those aspects of ones environment which act as cues for behaviour and allow the individual to exercise appropriate social skills. These cues may elicit or inhibit certain responses or guide our choice of response in some way.

Bandura (1969) and Kanfer and Phillips (1970) have emphasised that a major factor in social learning is the ability to **discriminate** relevant cues within the environment. This means that stimulus-response contiguity must be accompanied by discriminative observation to produce learning. For this to occur, it is necessary that the stimulus variables required for learning are **identifiable** to the learners. There have of course been studies on normal individuals indicating learning without "awareness" of a stimulus cue (Hefferline et al 1959; Keehn 1967). Bandura, on the other hand, argues that "simply exposing an individual to repeated stimuli does not guarantee that he will select from the total stimulus complex those cues necessary for learning to occur". This appears to be true for subjects with congenital brain damage as Cullen (1978) showed. These findings with the mentally handicapped and their apparent relationship to attention, may have major

implications for understanding the influence of attentional deficits after head injury, especially if we consider the ample clinical and experimental evidence of defects of attention after head injury, (Meyer 1904; Conkey 1938; Goldstein 1939; Denker and Lofving 1958; Miller 1970; Gronwall and Sampson 1974; Miller and Cruzat 1981). Just as attention deficits in mentally handicapped individuals can cause difficulties in learning, the same type of deficit may cause problems of learning after head injury. This can have important implications for the way rehabilitation is applied and the duration of treatment necessary to provide maximum benefit.

To determine the influence of attention on behavioural learning in the severely brain injured the present study compares the discrimination learning ability of three groups of subjects; (1) severely brain injured, (2) high grade mental handicap, and (3) normal controls. Comparisons of discrimination learning **ability** as well as differences in the **speed** of learning are obtained. The relationship of intelligence and attentional performance to discrimination learning ability is also examined.

It is expected that:-

(1) There will be significant differences in learning **ability** among the three groups. If intelligence is a relevant variable then the performance of the brain injured group should fall intermediate between the mentally handicapped group and the normal controls.

(2) When the **speed** of learning is compared, the brain injured and mentally handicapped groups are expected to show a similar (slow) speed of learning compared to the control group, with the brain injured group still being intermediate in terms of ability.

(3) In keeping with the Zeaman and House hypothesis, the performance of the brain injured on the discrimination learning task will vary more as a function of attention, measured by choice reaction time and auditory vigilance, than intelligence, measured by the Weschler Adult Intelligence Scale.

METHOD

Subjects

Group 1. This comprised thirty eight patients (male 25; female 13) receiving rehabilitation following severe head injury. Details of this group are contained in table 10.a. Clinical observations of these patients suggested that a number had difficulty making associations or discriminating social or environmental cues that determine many aspects of behaviour. These same patients also tended to be very distractible. They had problems sustaining or directing attention in the way McGhie (1969) described as "forced responsiveness".

Patients with dysphasia or any kind of residual motor or visual field defect, likely to interfere with task performance, were not included in the study.

Group 2. A group of twenty high grade hospitalised mentally handicapped patients (male 15; female 5) were included as a comparison (control) group to the brain injured because of similarities between their functional skills, personal independence and other behavioral learning characteristics. Many subjects in this group were extremely distractible, which affected their cooperation in occupational therapy activities and their poor performance in social skills training.

Group 3. A control group of 20 normal individuals,(male 10; female 10) of similar age and premorbid occupational level to group 1 was included. This control group comprised 4 hospital staff members - one domestic and 3 nursing assistants, and 16 individuals (hospital visitors, relatives of patients, personal friends), from the community. It is appreciated that a better control group could have been obtained by using orthopaedic, non-head injured patients, but these were not easily available at the time the study was completed.

N.B. Subjects in the two patient groups were not randomly selected. They represent all the patients who were able to enter the study over the period during which data was collected.

Table 10.a

Characteristics of the three groups in the discrimination learning study

	GRP 1 (S.B.I.) (N= 38)		GRP 2 (M.H.) (N=20)		GRP 3 (Normals) (N= 20)	
	MEAN	S.D.	MEAN	S.D.	MEAN	S.D.
AGE	28.6	10.4	19.0	2.3	28.5	9.9
I.Q.	87.7	11.3	62,2	3.2	99.7	8.8
* P.T.A.	3.3	1.8	--		--	
**T.S.I.	62.6	31.4	--		--	

* P.T.A.= Post Traumatic Amnesia (Months)

** T.S.I.= Time Since Injury (months)

PROCEDURE

All subjects were initially tested on a simple discrimination learning task. This required them to select one of two stimuli, differing both in colour (yellow and blue) and shape (square, triangle and circle). Each stimulus shape was approximately one and a half inches in diameter. They were placed three inches apart in front of the subject. The correct stimulus was a square, meaning that the subject had to ignore colour, (the irrelevant dimension) and the position of the square in relation to shape.

Colour and position was randomised over one hundred presentations (this being the limit of endurance for the two patient groups). Subjects were told whether their response was correct or not, the two patient groups receiving praise (social reinforcement) for accuracy. Learning was regarded as reaching criterion when twenty successive correct discriminations were made; (see Oakley (1983) for a similar procedure).

Following discrimination learning, all subjects were given two tests which require the ability to sustain attention, or to select relevant information from a stimulus set.

(1) Auditory Attention

A series of random digits was presented at a rate of one per second, through a set of "Dynamic" earphones. The volume was adjusted for each patient to a comfortable listening level. The experimenter monitored the patient's performance by following the digit series on a typed sheet. At random intervals a target (a series of three odd digits) was presented. The patient had to identify each target by tapping the table. The task lasted fifteen minutes and fifty target sets were included. The score was the total number of misses and false positive responses.

It might be argued that fifteen minutes is very short for a test of this kind. The limits were set according to the information obtained from pilot studies which confirmed the poor tolerance of the two patient groups. To make the test any longer would try the patience of subjects who were known to have behaviour disorders, which included aggressive, disruptive and non-cooperative tendencies.

(2) Choice Reaction Time

The "Leeds Psychomotor Tester" (Hindmarch 1979) was used to collect the choice reaction time data. This was arranged so that the stimulus and response panel were placed directly in front of the patient. The panel contained an arc of stimulus lights, six in all, below which was arranged a parallel arc of response buttons.

When a light was illuminated the subject was required to press, using the **preferred** hand, the button immediately below that light. Between responses the subject was required to keep their index finger on a centre button which was equidistant from the six response buttons. There was a constant inter-stimulus interval of six seconds, no other warning was given.

Other Procedural Points

All patients and controls were tested individually. A 'blind' testing procedure was used, whereby the experimenter who tested the subjects on discrimination learning was always different from the experimenter who tested them on measures of attention.

The measures of attention used in this study were previously employed by Wood and Cook (1979), investigating attentional ability in the siblings of schizophrenics. They have been employed in other studies investigating attention and concentration(e.g. Posner 1975; Van Zomeren and Deelman 1976,1978).

In addition to the attention measures, a measure of intelligence was obtained by pro-rating a 'short version' of the Wechsler Adult Intelligence Scale. This was composed of verbal measures of Vocabulary and Similarity, and Performance measures of Block Design and Picture Arrangement.

It is appreciated that many authorities argue against the use of I.Q. as a measure of brain injury (Meyer 1961;

Smith 1966; Miller 1978; Brooks 1984) because it is not sensitive to many of the more subtle deficits caused by brain lesions; a view shared by the author (Wood 1979). DeRenzi and Faglioni (1965) even advocate the superiority of attention measures, such as reaction time and vigilance, when trying to discriminate between normal and "pathological" subjects. The measure of intelligence in this study was included because its absence, during a pilot study, was questioned. Although on clinical grounds intelligence does not appear to influence discrimination ability its inclusion as a variable influencing learning must be considered.

RESULTS

Hypothesis 1

Discrimination learning **ability** was measured on the basis of whether subjects learned to criterion within 100 trials or not. Reference to table 10.b shows the difference in learning ability between the normal control group and the two patient groups. The control group reached criterion within thirty trials (allowing for the twenty consecutive correct responses needed to reach criterion), whereas neither patient group reached criterion in the time allowed.

Table 10.b

Means of the number of correct responses for each block of ten trials

	1	2	3	4	5	6	7	8	9	10
Grp 1	6.1	6.9	8.0	7.6	7.5	8.0	8.1	8.5	8.5	8.5
Grp 2	6.0	7.6	8.3	8.2	8.3	8.4	8.6	8.8	8.7	8.7
Grp 3	6.8	9.4	10	10	10	10	10	10	10	10

An analysis of variance was used to calculate the difference between the three groups. The 78 subjects were distributed in a 3 x 10 repeated measures design using unweighted means because of the different numbers in each group (Kirk 1968). This showed (table 10.c) that although the three groups made a significant improvement (responses above chance level) over the 100 trials ($F=23.024$; df,9/675; $p< 0.01$), the learning ability of the three groups was significantly different, ($F= 6.707$; df,2/75; $p< 0.01$).

Table 10.c

Summary table for 2X2 repeated measures ANOVA, comparing the discrimination learning performance of all subjects in each of the three groups

	SS	DF	MS	F	P
Between Grps	7.130	2	3.565	6.707	**
S's Wthn Grps	39.865	75	0.531		
Rep Measurs	7.725	9	0.855	23.024	**
Interaction	0.456	18	0.025	1.697	
Error-W	25.166	675	0.037		

A post hoc analysis of the difference between means using the Tukey test shows that the difference between groups 1 and 2 is not significant (mean diff= 0.35, C.diff= 0.389), whereas both groups 1 and 2 are significantly different from group 3 (mean diff= 1.02 & 1.37; $p < 0.05$ respectively).

This latter result was predicted but the lack of a between groups effect for the two patient groups means that the first hypothesis receives only partial support. It was predicted that if intelligence influenced learning then the brain injured group should perform at an intermediate level in terms of learning ability. This data however, shows that although there is a 25 I.Q. point difference between the two groups, the brain injury group are statistically no different to the mentally

handicapped on simple discrimination learning.

Hypothesis 2

If we exclude from the analysis the data of those subjects in groups 1 and 2 who **failed** to reach criterion, (n= 12 & 6 respectively), we find that only 5 subjects in group 1 and 1 in group 2 failed to learn by the 50th trial. A second repeated measures ANOVAR on these 50 trials (table 10.d) shows that the between group difference is eliminated, (F= 2.628, df,2/55).

Table 10.d

Summary table for 2X2 repeated measures ANOVA, comparing only those subjects from the three groups who learned to criterion

	SS	DF	MS	F	P
Between Grps	0.743	2	0.371	2.628	
S's Wthn Grps	7.782	55	0.141		
Rep measure	4.679	4	1.169	36.459**	
Interaction	0.092	8	0.011	0.361	
Error-W	7.059	220	0.032		

However, when the number of correct responses made by each group before reaching criterion (table 10.e) was analysed by a one way ANOVA, a significant between groups

difference was revealed (F= 4.972, df,2/56, p< 0.01). This suggests that although all the remaining subjects are **capable** of learning, their **speed** of learning remains quite different.

Table 10.e

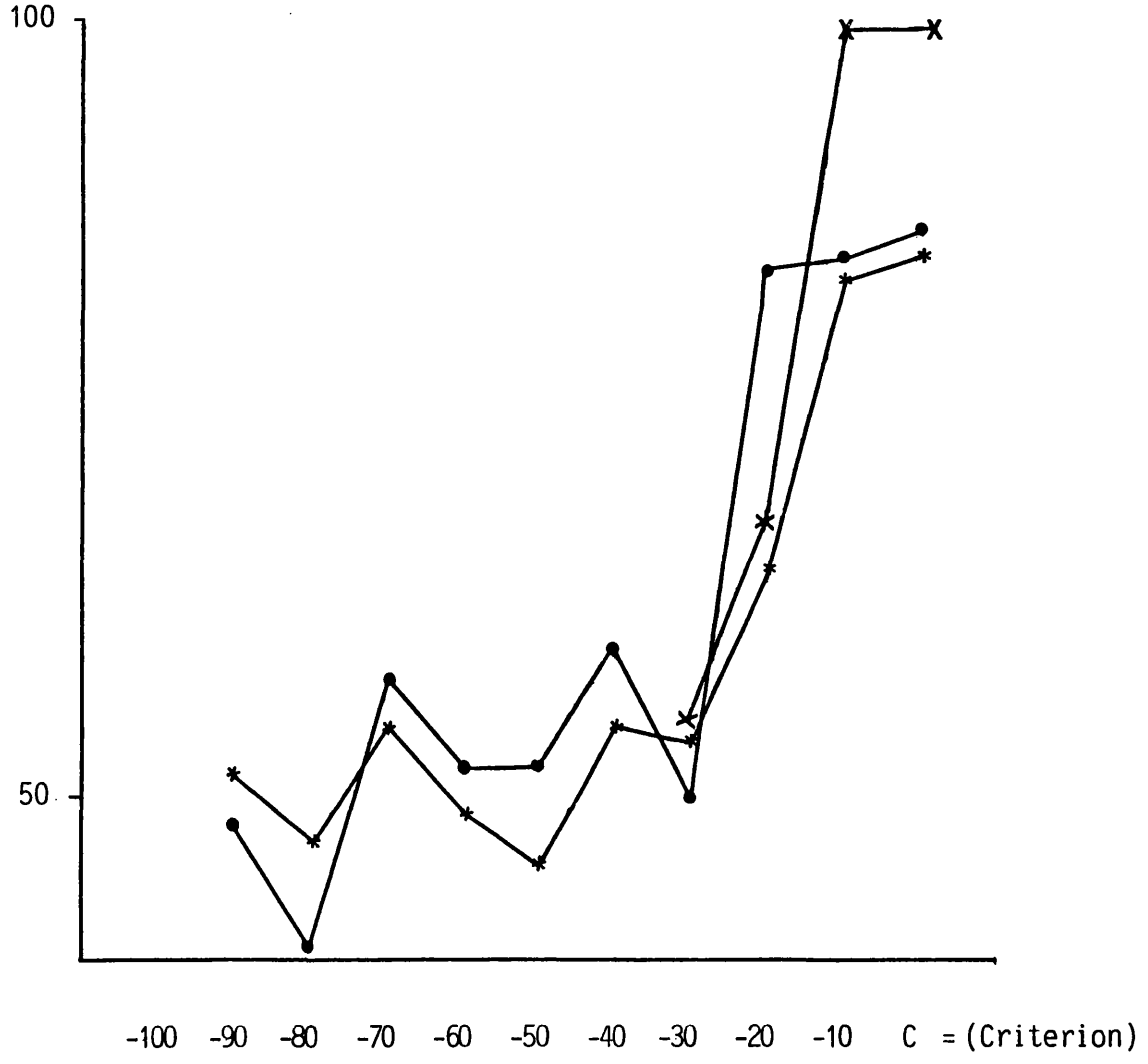
Mean number of correct responses for each ten trial block

	1	2	3	4	5	6	7	8	9	10
Grp 1	6.1	7.9	8.9	9.0	9.0	9.3	9.4	9.6	10	10
Grp 2	6.7	9.0	9.4	9.7	9.8	9.6	10	10	10	10
Grp 3	6.8	9.5	10	10	10	10	10	10	10	10

A post hoc comparison of means showed the significant difference to be between the brain injured and normal controls (mean diff.= 9.21; C diff= 7.89; p< 0.05). No difference was found between the patient groups (mean diff= 5.07, C diff= 7.89). On this occasion however, the mentally handicapped group,(again intermediate in terms of ability), did not significantly differ from the normal controls (mean diff= 4.14, C diff= 7.89). This suggests that slow response acquisition is more characteristic of the brain injured than mildly retarded individuals.

Differences in the speed of learning can be seen more clearly by converting the data into backward learning

Figure 10 (i)



Backward learning curve showing the performance of all subjects on a colour/form discrimination learning task.

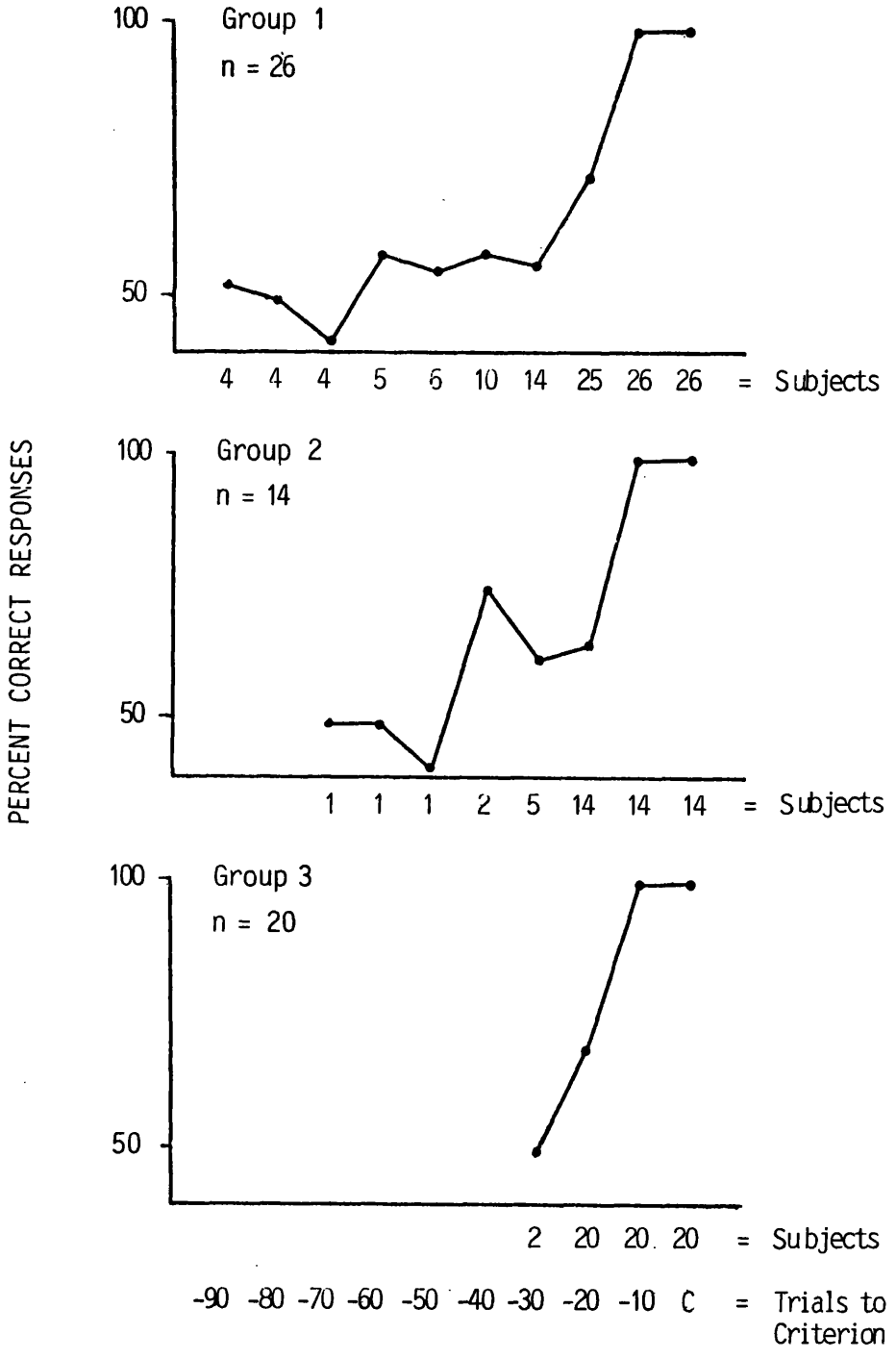


Figure 10 (ii) Discrimination learning in brain injured retarded and normal subjects. (a) Group 1. Backward learning curve showing percent correct responses in a group of twenty six severely brain injured patients on a colour/form discrimination task. (b) Group 2. Backward learning curve for fourteen mentally handicapped. (c) Group 3. Backward learning curve for twenty normal adult humans.

curves (Hayes 1953). This technique has been used by Zeaman and House (1963), and more recently Oakley (1983), to demonstrate group curves in the vicinity of criterion. Figure 10.1 shows the data for all the subjects (learners and non-learners). The number of responses at chance level for the two patient groups can be contrasted against the rapid progress towards criterion made by the controls. This can be seen more clearly in figure 10.2 which presents only the data of the learners from the three groups as separate graphs (grp 1 N= 24, grp 2 N= 14, grp 3 N= 20).

The learning curves for each of the three groups show the difference in the number of trials at chance level. They also show the number of subjects responding at chance level before the sudden and rapid increase in accurate discriminations, from chance level to the 100% correct. It is interesting to note that proportionally, there are more brain injured patients making up the tail of their learning curve than is the case with the mental handicap group. This is seen as providing further support for the inferior learning ability of the brain injured, relative to the mentally handicapped subjects.

HYPOTHESIS 3

Further between group measures have been made comparing the influence of intelligence and attentional variables on discimination learning. Within group measures have concentrated on examining such influences specifically in

the brain injured group which, besides being the largest group, also contained the largest proportion of non-learners (30%), allowing a reasonable within group comparison.

(i) Intelligence: Table 10.f shows how the three groups differed on measures of I.Q., C.R.T. and Vigilance. Analysis of the I.Q. data by a one way ANOVAR using the log transformation Y= ln(1+X) to avoid skewness shows (as expected) this difference to be highly significant (F= 106.273 df= 2/73, p< 0.0001)

Table 10.f

Comparison of group means and standard deviations for measures of I.Q., choice reaction time and auditory vigilance

	Grp 1		Grp 2		Grp 3	
	MEAN	S.D.	MEAN	S.D.	MEAN	S.D.
I.Q.	87.79	11.36	62.25	3.25	99.75	8.84
C.R.T.	910.48	292.12	859.50	349.42	648.00	124.86
VIG.	22.86	12.94	28.05	7.97	2.25	2.9

The interesting fact however, is that the direction of difference in I.Q. between the three groups is **not** reflected in their discrimination learning performance.

Group 1 is far superior to group 2 in terms of intelligence (mean diff= 24.4; C diff= 7.66; p< 0.01), but performed no differently to them on the discrimination learning task. Similarly, group 2 is significantly below group 3 on I.Q. (mean diff= 35.7; C diff= 7.66; p< 0.01) but equal to them in terms of learning ability. This suggests that I.Q. **alone** cannot be used to explain differences in performance across the three groups.

A within groups analysis on the influence of intelligence on discrimination learning was made by comparing the pro-rated full scale I.Q. scores of the learners (n=26) and non-learners (n=12) in the brain injured group. This failed to provide evidence of a significant within group difference, (t= 1.655, df 32).

(ii) CHOICE REACTION TIME. As above, a between groups analysis was made using a one way ANOVAR. A significant between group difference was found (F= 6.059, df= 2/65, p< 0.05), which a post hoc comparison shows is mainly due to differences between groups 1 and 3 (mean diff= 258.9; C diff= 214.2; p< 0.01). Comparison of groups 1 and 2 , or 2 and 3, failed to produce any significant differences. Group 2 continued to be intermediate with respect to their performance.

The within group analysis on the brain injured learners and non-learners for choice reaction time, shows a significant difference (t= 3.257 df= 25, p< 0.01),

suggesting that this measure is more sensitive to requirements of a discrimination learning task than I.Q..

(iii) AUDITORY ATTENTION. The between group difference continues to be significant (F= 32.968, df= 2/66 p< 0.001), and separates the groups in a way that is similar to their discrimination learning performance. Table 10.f shows that a clear distinction exists between the error scores of group 3 and either patient group, which post hoc comparisons show to be highly significant (Groups 1 and 3 mean diff= 20.6; C diff= 4.213; p<0.01; Groups 2 and 3 mean diff= 25.4; C diff= 4.213; p< 0.01).

The within group comparison supports the results of the other attention measure by recording a significant difference between the learners and non-learners on their performance on this task,(t= 3.652, df=34, p< 0.01).

Discussion

The results of this study suggest that problems of attention may interfere with aspects of behavioural learning and subsequently affect brain injury rehabilitation and the retraining of functional skills. The differences found between the performance of normal compared to brain injured or mentally handicapped subjects on the discrimination learning task were inevitably expected. The magnitude of this difference was not anticipated however, neither was the similarity in performance of the brain injured and mentally handicapped groups. Even when the data analysis was restricted to the learners of the three groups, thereby eliminating a between-groups effect, the brain injured patients still demonstrated a slow rate of learning, taking significantly longer to move from a chance level of responding than either the normal or mentally handicapped subjects.

The data would appear to support the Zeaman and House (1963) hypothesis that, discrimination learning, of the kind measured here, relies more on attentional factors than intellectual ones. The measures of intelligence suggest that the mentally handicapped group would be far inferior, in terms of discrimination learning ability, to the other two groups, in particular the normal controls. In fact, they fall intermediate between the normal and brain injured groups and not significantly different, in

their rate of learning, from either. When the performance of the brain injured learners and non-learners is compared, no difference is found between them on the basis of intelligence, whereas both attention measures emerge as being significantly different. This strongly suggests that attention factors have a far greater influence in associational (or behavioural) learning in the severely brain injured than might previously have been expected.

The importance of attention in behavioural, as opposed to cognitive, learning has been commented on in recent animal literature (Oakley 1979, 1981). Oakley (1983) summarised his experience of experiments on operant conditioning with decorticate rats. He found that such animals were still able to learn, in some cases as well as normal animals, but that attention was an important factor in such learning. The speed at which decorticate animals learned was improved if, first of all, they were trained in procedures which helped them focus attention on relevant cues.

This is not a recent observation. As early as 1935, Lashley described neocortically damaged animals as deficient in attention but recognised that, at the time, this was an unpopular and unacceptable explanation for observed responses. Oakley (1983) argues that associational mechanisms operate independently of cognitive functions following severe brain injury. He

bases this statement on the results of studies showing success of behaviour modification programmes irrespective of the level of residual cognitive ability in the patients involved, (Wood and Eames 1981; Wood 1984.)

Another observation that might reinforce this argument is that of the 26 brain injured patients who learned the discrimination task in the time allowed, five were unable to verbalise the solution, even though they had no defect of language or any other intellectual impairment that might prevent such an explanation. Of these patients, three had severe frontal lobe damage and two had diffuse cortical damage due to anoxia.

The indication that attention is of critical importance in discrimination and behavioural learning would appear to find some clinical support. Rehabilitationists and others working with the severely brain injured will be well aware that social behaviour, co-operation and general learning ability vary, not simply as a function of a person's intellectual ability, but according to how well they are able to concentrate or focus attention on a task. Meyer (1904) commented on the concentration difficulties exhibited by many brain injured patients, even during simple and interesting activities. Poor performance of head injured patients on tasks measuring concentration and focussed attention have also been described by Conkey (1938), Goldstein (1939), Ruesch (1944) and more recently by McGhie (1969) and Ben-Yishay

et al (1979).

Given the apparent importance and pervasiveness of this problem, there is little in the literature on brain injury or rehabilitation that offers help for those faced with the problem of re-training social or other behavioural skills in patients with this kind of difficulty. There would appear therefore, to be some merit in Miller's (1983) claim of parallels between methods of training functional skills in the severely brain injured and the mentally handicapped. It is hoped that this study will at least serve to illustrate the significance of attentional difficulties in simple discrimination learning and the effect it may have on social learning or other kinds of behavioural learning that take place during brain injury rehabilitation.

CHAPTER 11

OPERATIONAL COMPONENTS OF ATTENTION

Introduction

There has been some confusion regarding the concept of attention. Until recently there was not even an acceptable model of attention and when one did emerge it aroused much controversy and theoretical debate. Moray (1969) stated that the terminology relating to attention was "at best confusing and at worst a mess". There is however, some consensus about three 'operational' components of attention: alertness, selectivity and effort (Posner 1975; McGuinness and Pribram 1980; Eames and Wood 1984).

Alertness In one sense, attention refers to a state of alertness or arousal which allows us to recognise if information is present and direct our attention towards it. Alertness is a fluctuating condition of the central nervous system which may vary from a low level during sleep to a high level during wakefulness. Changes in alertness affect our performance on a variety of tasks. It is best measured by changes in the electrophysiology of the brain and thus is an area of study dominated by psychophysiologists (for a recent review see Gale and Edwards 1984).

Selectivity. Selective attention refers to the process of choosing some item of information from the environment in preference to others. Selected items are more likely to

affect our awareness, memory or behaviour than non-selected items which may be present at the same time. This aspect of attention was originally described by William James (1890) as "The taking possession by the mind, in a clear and simple form, one out of what seems to be several simultaneously possible objects or trains of thought". In saying this, James opened the door for studies on selective attention which have pre-occupied academic research on human attention ever since (McGuinness and Pribram 1980; Stevens 1981). Although James explained a number of attention characteristics, including the behavioural manifestations of attention, he never quite solved the problem concerning the mechanics of attention, especially its controlling processes. This probably led to the study of attention being rejected by the more dominant behavioural psychology of the late 1930's which regarded it as too mentalistic a concept to be worthy of scientific research.

Research into psychological aspects of selective attention was ironically revived by the behaviourists themselves. Observers of animal learning experiments, especially those investigating cue sampling and discrimination learning, were forced to acknowledge the existence of attention when animals were found to notice some stimuli but ignore others (Trabasso and Bower 1968; Honig 1969). Data suggesting the presence of attention variables were explained in terms of cue salience, assuming that particular stimulus parameters

were more noticeable or meaningful to the animal than others. This led to other studies on attention and learning, one of which was referred to in the previous section, dealing with attention and learning in the mentally handicapped (Zeeman and House 1963).

Effort The final aspect of attention involves the conscious effort that one gives to monitoring a stimulus. This implies a control process (McGuinness and Pribram 1980; Stevens 1981) that co-ordinates alertness, arousal and selectivity to **maintain** attention while directing it towards significant features of the environment. Van Zomeren and Deelman (1978) describe this aspect of attention as **"tonic alertness"**- "a continuing receptivity to stimulation extending over minutes or hours". It is this aspect of attention that is most frequently described as "concentration". As such it is clearly a very important component of attention and vital to the learning process. Unless patients are able to continuously direct attention during a rehabilitation task they are unlikely to learn to the best of their ability. If attention cannot be sustained, it matters little how quickly the individual orients to stimuli or how well he selects, from the environment, those stimuli which are relevant to a particular task.

The effective use of information processing depends therefore, on maintaining a **continuity** of information, to provide what Dimond (1980) refered to as "the running span

of experience". Driving a motor car, for example, relies on just this kind of continuity. We need to adjust present actions on the basis of what has preceded them, otherwise life becomes a series of discrete events, each event based on stimuli being processed at the time without any necessary relationship to what has gone before.

Evidence For An Attention Disorder

Three attention processes have been described which have some operational validity that may assist in the development of treatment procedures. Before designing a treatment programme which has some general application however, we must decide which of the three processes is most damaged by brain injury, because it is probable that different kinds of attention disorder will require different kinds of treatment (Wood 1984).

Table 11.1(a) provides data on a reference group of patients who acted as subjects for the collection of data throughout this series of studies. Some studies do not include data on all these patients. Where this is the case it can be assumed that the characteristics of the smaller group are not significantly different, in terms of the major criteria (PTA, TSI), from the group as a whole. Reasons will be given, as appropriate, when individuals from this reference group have not been used for specific measures.

Table 11.1(a)

Characteristics of the patients who participated in the series of attention studies

Group 1 (N = 20)

Type Of Brain Injury	N
Closed head (Deceleration)	12
Open head (Injuries)	6
Haemorrhage	2

	Age	PTA (mths)	TSI (mths)
Mean	31.1	3.43	58.4
S.D.	11.53	2.74	48.6
Range	19-62	0-10	11-120

11.1 A Critical Flicker Fusion Measure Of Behavioural Alertness.

Introduction: The problem in assessing alertness in the severely brain injured is that behavioural criteria (the patient being aware of and deliberately responsive to the environment) do not seem to parallel cognitive criteria, based on measures of central arousal (critical flicker fusion - **CFF**) or the **speed** at which patients demonstrate **awareness** of a stimulus (**decision CRT**).

One might assume that the behavioural differences between **hypo**-arousal and **hyper**-arousal described in chapter 2 (the former associated with lethargy and low drive, the latter with distractability) would be reflected in measures of CFF. The rational for this measure is based on the long association between CFF and changes in central nervous system activity (Irvine 1954). It has been described as a central measure of arousal (Claridge 1967) and used in various studies to examine the effects of drugs on levels of human arousal and information processing ability (Hindmarsh 1979).

Differences in CFF between a brain damaged and normal group would presumably indicate differences of arousal. It is of some interest therefore, to see if the same differences in arousal could be elicited between patients with very similar brain injuries but different **behavioural** sequelae of arousal, alertness and awareness.

Subjects: Group 1 comprised 20 severely head injured patients. The characteristics of this group are given in table 11.1(a). Of these in-patients from the Kemsley Unit, 6 showed behaviour associated with low arousal while another 6 displayed behaviour which suggested the opposite (e.g. distractability or over-awareness of novel stimuli in their environment). The other 8 patients were (behaviourally) 'normally aroused'.

Group 2 included 10 normal control subjects matched for age with the patient group. Three were hospital employees, the others were from the community.

Procedure: A measure of CFF was obtained using the Leeds Psychomotor Tester apparatus (Hindmarsh 1979). A square of 4 small red lights, 1 inch apart, set against a black background was used to present the stimulus pattern. Initially, the 4 lights were illuminated at 60 Hz, which appeared as a **continuous** light. Then the frequency was reduced at a rate of 2 Hz per second until the subject perceived a discontinuous light pattern. At this point, the subject pressed a button to record the frequency at which this **flicker** occurred. Ten measures were obtained using a decreasing Hz frequency while another ten were obtained using an increasing frequency. In this latter condition the 4 lights commenced at a simultaneous flicker. The subject had to respond at the point where 'fusion' of the stimulus was observed.

A comparison was made of the ascending and descending series between groups 1 and 2. A **within** group analysis was also made to see if this task discriminated between the 6 patients at different ends of the behavioural continuum of alertness and the "normally" aroused patients. Table 11.1(b) shows how their head injury characteristics compare with the main group.

Table 11.1(b)

A division of the severe brain injured patients into groups which have different characteristics of behavioural arousal

	GROUPS		
	1	2	3
Arousal	(Low)	(High)	(Normal)
	(N=6)	(N=6)	(N=8)
AGE			
Mean	28.5	30.9	32.0
S.D.	11.3	9.7	7.8
PTA			
Mean	3.3	2.9	3.8
S.D.	2.0	1.7	2.9
TSI			
Mean	64.6	45.7	55.9
S.D.	46.7	53.3	50.0

RESULTS :Measures of the ascending and descending series were compared separately. The difference between patients and controls was measured, using an independent Student's t test. Differences between the means were not significant, either for the ascending (t= 1.22; df= 28) or the descending (t= 1.91; df= 28) CFF measure.

The within group analysis used a one way ANOVA to compare the means of the different patient sub- groups. Again, no difference was found between the ascending (F= 0.488; df= 19) or descending series (F= 0.975).

Commentary :The lack of a significant difference between these groups on a measure presumed to identify a level of central arousal may appear most unusual, considering the obvious behavioural differences. However, as a clinical measure the CFF has not provided reliable results. No reliable differences were found when the measure was applied to schizophrenics (Irvine 1954; McDonough 1960) although, more recently, apart from its successful use in drug studies, it has proved to be good at discriminating changes of visual perception in patients with multiple sclerosis (Daley et al 1979).

The results of this study however, suggest that **behavioural** indices of arousal can not be used to indicate changes in perceptual thresholds; that is, the individual

is **aware** of the presence of information, it is the ability to **act** upon that information that is impaired. This helps to support the earlier comments on arousal problems in which **drive**, as a behavioural component or **'executive'** response, was identified as the focus of impairment, rather than any changes in interest or motivation, which are regarded as involving a **cognitive** aspect.

11.2 A Dichotic Listening Study To Measure Selectivity.

Selective attention following head injury has been studied using the rational of the **filter** models, proposed by Broadbent (1958) and Treisman (1965). These explained selective attention on the basis that irrelevant information had to be actively filtered out of the information processing system either at the sensory input phase (Broadbent 1958) or the response selection phase (Treisman 1965).

Originally, selective attention was assumed to be a perceptual phenomenon occurring in an early stage of information processing. It gradually became clear however, that irrelevant information may produce intrusions on higher levels of thinking, forcing subsequent theories to locate the structural bottle neck in the **response selection** stage instead of the **stimulus selection** stage (Treisman 1965; Broadbent 1971).

Van Zomeran (1981) describes a number of studies which tested focused attention on the basis of a filter model (Denker and Lofving 1958; Gronwall and Sampson 1974) using a dichotic listening paradigm. Both these studies tested focused attention in patients who had suffered only minor brain injuries. In the former study 28 pairs of twins were studied, one of whom had sustained a head injury. This was not a realistic study however, because the injuries were very minor ones and, in addition, had been sustained 10 years previously. In the latter study only five patients

were tested and they had durations of PTA of less than one hour. Although tested within days of injury the sample is not large enough to allow generalisations to be made.

Both studies used a standard dichotic listening task where the subjects were intructed to 'shadow' the message in their left ear while ignoring information arriving in their right ear. In neither study were there any intrusions from the irrelevant messages so the authors concluded that their patients showed no deficit in selectivity.

Van Zomeren argues that these studies made use of very obvious cues to decide on the relevance of information (left ear or right ear). Moray (1969) has pointed out that ear of presentation is a very potent cue to selective attention. Thus, selection was possible on the basis of structural cues, helping to direct the subject's attention to information on the basis of some physical dimension. As such, the task did not demand much of a spontaneous selective strategy on the part of the patients, as would be the case in terms of the 'cocktail party phenomenon'.

There does not appear to be any evidence on the effects of dichotic listening in a group of very severely brain injured adults. In order to find out how such patients would cope on an information processing task and whether they could make use of the same cues as the non-severely injured patients in the above studies, their performance on a dichotic listening task was compred to a group of normal individuals.

Subjects

Group 1. 15 of the patients described in table 11.1(a) were used. The patient group (9 male, 6 female) had a mean PTA of 2. 9 months (SD= 1.2 months). They were all more than 2 years post injury. The 5 subjects excluded from the study all had language problems that could interfere with task performance. In all other respects they were the same as the original reference group.

Group 2. This consisted of 3 male and 3 female normal controls matched for mean age with group 1.

Procedure: Subjects were given a task which involved shadowing a series of digits presented to the left ear while ignoring a series of letters presented to the right ear. The task lasted approximately 14 minutes. Early attempts at administering this task showed that patients of this kind could not be relied on to maintain cooperation for much longer, any errors produced after this time would likely be an artefact of their short attention span rather than their selective attention ability.

The stimuli were presented serially, at one per second. For the first seven minutes, the patient was asked to repeat the digits being presented to the left ear. At the end of that interval they were told to change and shadow the letters being presented to the right ear. The errors recorded were (1) omission of a digit or letter, one error score for each omission (a sequence of five consecutive

digits omitted would equal an error score of five rather than one) and (2) an intrusion error or (3) an incorrect response where a number or letter, other than the one designated, was named.

Results: For the purpose of analysis, all the error scores were combined allowing an overall analysis of the two groups by an independent Student's t. Separate analysis was then made of the category errors to see if one type predominated over the others.

The total number of errors made by the patient group (mean= 11.8; sd=4.15) was significantly greater than that of the control group (mean= 5.1; sd= 2.03), t= 4.02; df= 20 $p< 0.001$.

Analysis of individual categories was made using a one way repeated measures ANOVA. This failed to reveal any differences in the control group (F= 0.296; df= 2/12) but the patient group showed a significant between categories effect (F= 3.498; df=2/28; $p<0.05$). A post hoc comparison of the data (Tukey test) showed that the larger number of omission errors relative to intrusion errors was significant (mean diff= 2.0; C diff= 1.29; $p< 0.05$), as was the case between omission errors and incorrect responses (mean diff= 1.80; C diff= 1.29; $p< 0.05$). No difference was found between intrusion errors and incorrect responses (mean diff= 0.2; C diff= 1.29).

Commentary: Using Van Zomeren's argument, the difference between patients and controls suggests that the patients in this study were unable to use even the fairly obvious cues for selection of information offered by the dichotic listening procedure. However, the definition of a selective attention deficit adopted by Van Zomeren, would suggest that these results are more indicative of a DAD than a FAD because the greater number of omissions, relative to other possible errors, would suggest that these patients had problems with the **speed** of processing rather than in the utilisation of the various structural cues offered by the experimental design.

11.3 Problems of Selection or Response Slowing?

The previous study shows that the selective attentional requirements of a dichotic listening task may not show impairment when **small** groups of patients with **minor** head injury are used but do when the performance of a very severely injured group is contrasted to normal controls. What is not fully established however, is the **nature** of this deficit, whether it is a problem of selectivity or of response slowing?

Head injury slows down the rate of information processing and makes it less efficient, especially when any kind of selective processing is involved. Important in this respect is the finding of Miller (1970; 1980), and later van Zomeren and Deelman (1978), that the more **bits** of information to be processed, the more the performance of the brain injury group differed from that of the normal control. Reaction time studies with brain injured subjects, using differing numbers of response alternatives (e.g. Miller 1970; Gronwall and Samson 1974; Van Zomeren 1981), show a linear relation between the number of stimulus variables and reaction time, both in control subjects as well as patients. Van Zomeren thought that this made it unlikely that the patients used a different "strategy" of processing from their normal controls.

His studies compared simple and four choice visual

reaction time in 20 subjects with varying severity of injury and compared their performance to control subjects matched for sex and age (Van Zomeren and Deelman 1976; 1978). Miller's study was restricted to only five subjects, with a PTA of "over a week". Although this puts them into the "very severe" category of concussion, using Russell's (1971) definition, the duration of PTA is still far less than the patients who are the subject of this thesis.

This is also true of the Gronwall and Samson study which used only mildly concussed subjects, and is probably true of the Van Zomeren and Deelman study because, although their patients were "in coma for at least half an hour" it does not appear that their post traumatic amnesia was of a duration similar to the present patient group. This is because the Van Zomeren and Deelman data was initially collected after a mean interval of 108 days, at which time many of the patients in the present group were either still in coma or P.T.A.. It will be interesting therefore, to see whether this same "strategy" of processing appears to apply in patients who are very severely brain injured

Two studies are presented. The first is a measure of choice reaction time (CRT), to determine differences in the **speed** of response between normals, non-severe and severely brain injured patients. The second study looks at the effects of increasing the number of

stimulus-response contingencies, contrasting predictions made from filter and 'two-process' models of attention.

11.3 (a) Choice Reaction Time.

Subjects

Group 1 (N=17), were drawn from the patient group represented in table 11.1(a). The 3 subjects not included all had CRT scores in excess of 6 seconds. Two patients were obviously not cooperating while the third was found to have visual field problems which, although not appearing to affect perceptual thresholds of the kind presented by the CFF stimulus, did interfere with the selection of one from six possible stimuli used in this task.

Group 2 (N=12), contained patients who were attending hospital as out-patients after sustaining a non-severe brain injury. Their mean PTA was only 1.75 weeks, which was significantly shorter than group 1 ($t= 4.23$; $df= 32$; $p< 0.001$). Also, the sequelae of their injuries were confined to fairly minor intellectual blunting, memory and concentration problems, rather than having the quite severe neurological problems, behaviour disorders and intellectual deficits of group one.

Group 3 (N=12), were normal controls, comprising members of the hospital staff and friends of hospital staff. The three groups were matched for age.

Procedure

The 6-choice reaction time apparatus was the same as that described in section five (Leeds psychomotor tester) where the total reaction time could be split up into decision time (DT) and motor time (MT). Six stimulus lights were arrayed in an arc on a response panel. This was placed immediately in front of the patient who was told to use the preferred hand (in all cases their dominant hand) to press the response buttons, also arrayed in a (parallel) arc immediately below the stimulus lights. Fifty stimulus presentations were used to determine the mean reaction time.

Because the purpose of this study was to determine changes in **selective** attention, the **decision** time was used to measure differences between the three groups. The subject was told to place their index finger on a button in the centre of the board, equidistant from the lights in the stimulus array. The decision time was the interval from stimulus onset until the subject moved his finger **off** the centre button (starting the motor response). A study involving motor response time will be described below.

Results

The data was analysed by a one way ANOVA. The results, as expected, show a significant difference between the three groups ($F= 26.18$; $df= 2/38$; $p< 0.001$). A post hoc analysis of the group means using a Tukey test shows the greatest difference to be between groups 1 and 3 (mean diff= 647.8; C diff= 235.9; $p< 0.01$). The two patient groups were also significantly different (mean diff= 463.1; C diff= 235.9; $p< 0.01$) but no significant difference was recorded between the moderately head injured group and the controls (mean diff= 184.75; C diff= 235.9).

It is interesting that a comparison of the means and standard deviation for the groups shows that those patients in group one with scores more than 2 SD's above the mean of group 3 (n= 6) all had major frontal lobe injury and recorded a decision time of more than 1 second.

Commentary: This finding is consistent with Van Zomeren and Deelman (1978) and other studies which have found slow reaction times to persist for an inordinate length of time after head injury. The significant difference between groups 1 and 2 also suggests that CRT acts as a good indicator of the severity of the injury in terms of its effect on cognitive function. The lack of significance between the means of groups 2 and 3 is unusual considering the previous reports of the sensitivity of CRT as a measure of brain injury (Miller 1970; Van Zomeren and Deelman 1978).

It is difficult to explain such results because group 2 had sustained a significant concussional injury. One answer might be the length of time between injury and this test being done (see TSI, table 11.1(a). However, this group still complained about **concentration** problems affecting behaviour. This may be a different attentional process to the one being measured in this reaction time study. Different results can be found when the same CRT procedure was administered as a continuous performance task (see study on focused attention).

11.3(b) Divided Attention and Reaction Time

The next study attempts to determine the influence of **FAD's** and **DAD's** in this group of patients by analysing the effects of increasing stimulus alternatives, thus comparing changes in reaction time to increasing amounts of distraction.

Subjects.

Group 1; 15 subjects from the group described in table 11.1(a) acted as the experimental group. Five subjects were excluded, 3 on the basis of visual field problems (see condition 1 in procedure for explanation) and 2 because they did not cooperate properly.

Group 2 comprised 10 subjects, matched for age, (5 male, 5 female), who were included as a normal control group.

Procedure

A task was designed and programmed on a Commodore 64 computer. Its primary use was **clinical**, to investigate subtle visual field deficits and to help train visual scanning and reaction time. The task presented visual and auditory stimuli, at random intervals and the former in random positions on a video monitor. The patient sat facing the screen at a distance of approximately 18 inches. Six reaction time (RT) conditions were possible in three stimulus conditions, each providing a separate reaction time (see table 11.3(a)).

Table 11.3(a)

Matrix of stimulus contingencies for each condition of the task

	1	2	3
RTs			SOUND STIMULUS
RTc		COLOUR CHANGE	COLOUR CHANGE
SRT	PINK DOT	PINK DOT	PINK DOT

CONDITIONS

Condition 1. In the first condition, the subject is required to focus attention on a square in the centre of the video screen. At random intervals a pink spot would appear in one or other of the screen quadrants, sometimes near the peripheral field of vision. The subject's task was to press the space bar of the computer keyboard each time a pink spot appeared. Sixty stimuli were presented in this way (randomised presentation, with 15 in each quadrant). The response times (designated **SRT1**) were automatically analysed by the computer, giving the mean

and standard deviation of the response times to stimuli from each quadrant of the screen. (This helped determine whether there were any subtle visual field deficits (blind spots) that were not apparent to ordinary clinical examination. If a large discrepancy was noted between the means of the quadrants then a visual field defect might be present, excluding the subject from the study. This explains the 3 patients excluded on the basis of "visual field" problems).

Condition 2. In the second condition, the screen presented stimuli in the same way as in the first condition but, at random time intervals, the **centre square** would change colour from black to green. This introduced a **colour distractor** to which the subject was expected to respond (in addition to the original stimulus of a pink spot). Throughout the second condition, 12 colour square changes and 48 dots were presented. A separate reaction time (designated **RTc2**) was recorded for the colour distractor, which could be compared to the S.R.T. in that condition **(SRT2)** as well as the S.R.T. in condition one **(SRT1).**

Condition 3. The third condition incorporates the second but, in addition to a colour distractor, introduces a **sound distractor.** At random intervals throughout the test a tone is presented, to which the subject must also

respond by pressing the space bar. Ten sound stimuli, 10 colour changes and 40 dots are presented. In this condition three scores were obtained, the simple reaction time to the randomly located dots **(SRT3)**, a separate reaction time for the colour distractor **(RTc3)** and a third reaction time for the response to sound **(RTs3)**.

Aims

This task is quite different to the selective attention tasks presented by Broadbent and others. It is designed to measure **controlled** processing, using the Shiffrin and Schneider paradigm of attention. A measure of **divided** attention is obtained by systematically increasing both the perceptual processing and response requirements of a task, using different sensory modalities (vision and sound) and visual scanning (to locate the dots). This might help establish the integrity of the selective mechanisms under conditions of increasing stimulus variability.

The studies of Van Zomeren, Miller and others have shown an increase in response time as a function of increasing the number of stimuli. They do not **explain** the nature of this response slowing however. If the Shiffrin and Schneider model is correct, all the responses involved in this study conform to the **controlled** mode of processing. This is because the patients have not had the opportunity to familiarise themselves with the changing response

requirements (allowing automatic responses) and, in any event, it is a task that, by virtue of its design, requires continuous conscious processing. This means that if a **DAD** occurs it will be for one of two reasons. The Shiffrin and Schneider model would predict that because the variety of stimulation and alternative response demands increase as the task progresses, attention would have to be divided over more stimulus factors and response contingencies. The effect of this should be to increase all reaction time scores **equally** across all stimulus-response conditions (i.e. in condition 3, all stimuli should be responded to in roughly the same time, with no difference occurring between SRT3-RTc3, SRT3-RTs3, RTc3-RTs3 yet a difference occurring between SRT1 and the stimulus-response contingencies of condition 3.

On the other hand, the **filter** models of attention would predict that **meaning** (wherever it occurs) would play a part in determining the response times because subjects would process faster, those stimuli that have more "meaning" for them. Clinical observations of distractibility suggest that **novelty** is the major factor likely to produce this response. Broadly speaking, novelty might be described as having "meaning" for these patients and where this exists, attention will be concentrated. If this is so then the response times will take on a different pattern. Instead of becoming

proportionally slower with incresing numbers of stimuli, the introduction of novel stimuli (even late on in a study) as one of several stimulus conditions requiring a response, should elicit a response which is significantly faster than non-novel stimuli in the same condition. In condition 3 therefore, the response to RTs3 should be faster than either SRT1 or RTc3.

RESULTS

Between-group analysis. There were highly significant differences between the performance of the two groups in each stimulus condition. These differences were measured using a t-test for independent means and the results can be seen in Table 11.3(b).

Table 11.3(b)

Comparison of group means and standard deviations. The response times (in msec) in each stimulus contingency have been analysed by independent Student's t with 23 degrees of freedom

Stimulus Contingency	Grp	Mean	s.d.	t	p<
SRT1	1	686	326	3.66	0.01
	2	303	45		
SRT2	1	926	301	3.02	0.01
	2	301	98		
RTc2	1	895	341	4.44	0.001
	2	385	86		
SRT3	1	1031	721	3.10	0.01
	2	318	52		
RTc3	1	880	338	5.35	0.001
	2	296	60		
RTs3	1	848	533	2.80	0.02
	2	327	151		

* Group 1 = Patients. Group 2 = Control

Within-Group Analysis. Inspection of the group data suggests that the patient group (grp 1) showed considerable variation in individual responses, both within and between stimulus conditions. This variation is mainly between subjects but, as table 11.3(b) shows, large differences also exist between conditions, for example, the increase in the SRT responses from condition 1 to condition 2 and from condition 2 to condition 3. Such an increase was not observed in the reaction times to other stimulus contingencies (colour-change or sound). To eliminate the subject effect on data analysis, the individual subject scores between each stimulus contingency were subtracted one from another. The difference scores were then tested for being significantly non-zero, using the Student's t test. This showed a significant effect between the SRT scores of conditions 1 and 2 ($t = 3.45$; $df = 14$; $p < 0.01$) and also between those of conditions 2 and 3 ($t = 2.67$; $df = 14$; $p < 0.02$) and a fortiori between those of conditions 1 and 3. No significant differences were observed between either the colour-colour or colour-sound conditions.

Inspection of the control group data (grp 2) reveals much less variability in responses between subjects and between conditions. The same analysis was used as for the patient group and no significant differences from zero were recorded.

Commentary Taken at their face value, the data appear to support a filter model of information processing. Response times to the original stimulus contingency increased significantly as extra stimulus contingencies were involved. The new stimulus contingencies however, did not, in themselves, record longer reaction times; their effect was simply to increase the reaction time to the original stimulus variable. If this can be interpreted as novelty having greater meaning, helping to focus attention on the novel stimulus, then the filter model would appear to explain this DAD better than the Shiffrin and Schneider model. The latter would predict an equal distribution for increased response times in conditions two and three.

Other factors must be considered before accepting the results of this study. The first is the effect of the sound stimulus on reaction time. To detect the other two stimulus conditions, the subject had to maintain an **active** attentional strategy (i.e. having actually to look for the stimulus). For the sound stimulus however, the subject could maintain **passive** attention because the sound stimulus **announced** itself to the subject, without him having to actually search for it. It was noted by the test examiner that on several occasions, patients were actually looking away from the monitor, failing to see the visual stimulus, but responding quickly to the far more obvious sound stimulus.

Another factor that must be considered is the difference between the visual stimulus affecting SRT1, SRT2 and SRT3, and the visual stimulus involving a colour change. The colour change was presented on each occasion in the same location, at the central field of vision. The presentation of the pink dot stimulus in the SRT contingencies however, was located in different parts of the screen. This means that for the SRT stimulus an **active search** of the screen had to be made, while in the colour change condition the stimulus presentation was more obvious.

11.4 Focused Components of Attention

An inability to maintain a focus of attention during therapy activities can present major problems when learning new skills or when giving co-operation to therapists. Distractability and short attention span are the major problems affecting focused attention. Although these two terms seem to be used in a synonymous way they do have different characteristics. A patient who has a short attention span is unable to **maintain** a focus of attention and therefore **passively** shifts attention to extraneous stimuli when maintenance lapses. The distractable patient, on the other hand, appears unable to **inhibit** extraneous stimuli, with the result that he cannot prevent the orienting response from occurring. Put more simply, the former problem involves being unable **to** attend, the latter problem involves being unable **not** to attend.

It is not easy to explain this difference in information processing terms, although an attempt has been made by Eames and Wood (1984). They suggest that distractability appears to involve a disinhibition of the orienting response; the patient being unable to avoid directing his attention to every novel stimulus which occurs in the environment. This could be due to over-arousal, which increases receptiveness to stimulation. Using the Broadbent (1971) paradigm, this would be explained as an interference with efficient **stimulus selection.**

Broadbent suggested that if we hold a particular attentional set (such as a position in space or a sensory modality) it is theoretically possible for a peripheral gate to be set up, which might allow in stimuli from one source but not from another. This kind of stimulus selection might become impaired in an over-aroused state, because an over-active phasic component would increase the reflexive response tendency, while the increased tonic aspect of alerting would reduce even further any voluntary control the person may have over a discriminative response to external signals.

The next study investigates the problems of **maintaining** a focus of attention over time. The first measure is of choice reaction time, used as a continuous performance task over 20 minutes. The second measure is the maintainance of a visual attentional set in preparation to respond to a visual stimulus presented at random intervals and locations on a video monitor.

Subjects

Both measures were administered to 15 patients from the original reference group (table 11.1(a)). The same group of normal controls mentioned in the above studies, were used for comparison.

Procedure

In the **reaction time** measure, the conditions were exactly the same as those previously described, except that motor time as well as decision time was recorded. On this occasion, the task was administered as a continuous performance task over 20 minutes. This means that as soon as a subject had completed his response to one stimulus, another stimulus was presented. The position of the stimulus in the six-light array was randomly determined. Several hundred response times were recorded for each subject over the 20 minute period. The mean reaction times were recorded for each 5 minute interval throughout the task.

The second measure presented a visual stimulus (pink dot) in random locations and at random intervals on a video screen. The time taken by the subject to respond to each stimulus (pressing the space bar of the computer keyboard) was the relevant measure. Stimuli were presented over a 30 minute interval, using a high rate of presentation to prevent unnecessary drowsiness occurring * (Posner 1975). Separate recordings of the mean reaction time for each five minute interval were made and analysed to determine any time on task decrement.

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* It is debatable whether this is the best measure. Studies on normals, Posner (1975) and on the brain

damaged (McDonald and Burns 1965) found that more errors are made to a low stimulus rate (12 signals per hour) than high rate (60 signals per hour). The choice was based on two factors. Firstly, with the present group of patients **cooperation** was always a significant variable. None of the subjects would have lasted an hour, either walking off or behaving in a difficult, possibly aggressive manner. Secondly, the important period in rehabilitation is a half hour, the duration of a therapy session. During this period bits of information are presented in a rapid and fairly lively manner (a high event signal presentation) suggesting that the high event ratio would prove to be a more suitable sampling procedure. Both high **and** low event procedures could not be used unless the measures were based days (in some cases weeks) apart to ensure cooperation. This seemed an impractical procedure.

Results

Both measures were analysed using a two way repeated measures ANOVA, with 28 subjects distributed in a 2 x 4 (choice reaction time) and 2 x 6 (visual attention) repeated measures design. Since block sizes were not equal, an unweighted means analysis was performed.

Choice Reaction Time. A significant between groups effect was recorded both for decision time (DT) ($F = 15.78$; DF 1/20; $p<0.01$) and motor time (MT) ($F = 27.7$; DF = 1/20; $p< 0.01$). The absence of a repeated measures effect (DT, $F= 0.915$; df=3: MT, $F= 1.00$; df=3) suggests that there was no time-on-task performance decrement over the 20 minute period, while the lack of any interaction effect (DT, $F= 1.07$; df=3: MT, $F= 1.7$; df=3) would rule out any difference in the pattern of responding between the groups.

Analysis of independent group means, comparing groups at 5 minute intervals, shows a significant difference between reaction times throughout the task (table 11.4(a).

Visual Attention. There is no significant between groups effect ($F= 3.568$; df=20), neither is there any evidence from the repeated measures factor to indicate an increase in response times over the 30 minute duration of the task ($F= 0.483$; df= 5).

The between-group means and standard deviations, showing the differences at 5 minute intervals, are given in table

11.4(b). Independent analysis at these intervals only revealed one significant difference, which occurred at the 10 minute interval ($t=2.21; df=20; p< 0.01$).

Tables 11.4(a) and 11.4(b) giving the mean, standard deviation and values of Student's t (with 20 degrees of freedom) for decision (DT) and motor (MT) choice reaction times and also for the visual attention (Vigilance) data. Response times recorded in milleseconds.

Table 11.4(a)

Decision Time

Intervals*	0 - 5		5 - 10		10 - 15		15 - 20	
Grps	1	2	1	2	1	2	1	2
Mean+	1.1	0.4	1.2	0.4	1.2	0.4	1.3	0.3
S.D.+	0.5	0.1	0.6	0.1	0.6	0.1	0.6	0.1
t	3.37		4.29		4.42		4.07	
p<	0.01		0.001		0.001		0.001	

Motor Time

Intervals*	0	5	5	10	10	15	15	20
Grps	1	2	1	2	1	2	1	2
Mean+	1.6	0.5	1.7	0.8	1.6	0.6	2.1	0.5
S.D.+	0.6	0.1	0.6	0.1	0.6	0.1	0.7	0.1
t	3.37		4.29		4.42		4.07	
p<	0.01		0.001		0.001		0.001	

* Time = Minutes. + Time = msecs.

Table 11.4(b)

Visual Attention

Intervals	Grp	Mean	s.d.	t	p<
0-5 mins	1	1753	3374	1.05	ns
	2	412	114		
5-10 "	1	916	572	2.21	0.05
	2	427	127		
10-15 "	1	982	747	1.99	ns
	2	409	91		
15-20 "	1	947	643	2.10	ns
	2	499	112		
20-25 "	1	906	664	1.80	ns
	2	445	121		
25-30 "	1	955	596	1.53	ns
	2	565	175		

* Time in minutes. + Time in seconds.

Commentary. The only result that could have been expected in this study was the between-group difference in reaction times. The lack of a difference in visual vigilance is hard to explain, other than on the basis of similar arousal levels, such as occurred in the CFF study. This would not account for the lack of a time on task decrement. The high ratio of stimulus presentation is probably responsible for this lack of effect, as would be predicted by McDonald and Burns (1965), even though their subjects were **not** a severely injured group.

Summary

The series of studies described above illustrates several interesting, and sometimes unexpected aspects of attention in this group of patients. The data also suggest ways in which disorders of attention, following severe brain injury, can influence behaviour.

The results of the first study suggest that the appearance of lethargic, driveless and 'under-aroused' behaviour does not necessarily imply any disorder of alertness or arousal per se (at least in the cognitive sense) if any value is placed on the CFF as a sensitive measure of arousal. From their awareness of, or sensitivity to changes in stimulus conditions these patients should at least be able to perceive and orient towards a stimulus.

The data from the next study provide some support for this orienting potential. Although, as a group, the patients were inferior to the controls on a dichotic listening task, the problem was not so much a failure to select the correct information, this can be inferred from the lack of any significant intrusion effects. The difference was due to a large number of omissions, which suggests that the patients knew what to attend to; their trouble was continuing to attend, implying a focusing rather than a selective failure.

The study on choice reaction time does not really add

anything to our understanding of the mechanisms of attention. The data do underline how much slower brain injured patients are from normals in responding when they have to monitor several possible stimulus locations at the same time. The data also point to the sensitivity of this measure, discriminating between different degrees of severity of brain injury.

The attention given to the **nature** of a deficit in selectivity may appear unusual in the context of a clinical thesis. It seems important however, to know what stimulus conditions most likely lead to a DAD and whether this can be avoided by systematically controlling environmental variables to preserve a degree of novelty and subsequently, better attentiveness on the part of the patient. In this respect the study of the effect of different stimulus conditions gave some interesting data, the analysis of which remains equivocal because of the variability between subjects. The indications however, point to novelty as a significant variable influencing both awareness **of** a stimulus and response **to** it. Consideration of the filter and process models of attention would implicate the effect of novelty as having a greater influence on the former than the latter.

Finally, the two measures designed to measure the maintainence of an attentional set produced some unexpected reults. The measure of CRT as continuous performance has been used by Benton and Joynt (1959) and

Costa (1962) to indicate performance decrements in the brain damaged. The lack of such evidence here may be due to the relative brevity of the task - 20 minutes (at least 30 minutes is designated as a "vigilance" period, Broadbent 1971; Posner 1975). Pilot studies showed that a number of patients gave up after even 15 minutes making the period of 20 minutes rather excessive for some with the possibility that cooperation would be lost. The lack of a performance decrement suggests however, that the active involvement of making a regular response off-set the development of any habituation effects.

The same cannot be said for the visual attention task which presented 60 signals over a 30 minute period. The lack of a between groups effect tends to reinforce the point made during the arousal study, that alertness is not a significant factor influencing behaviour. What this study showed is that the **attention span** of severely brain injured patients, under the right conditions, is as good as normal controls. The results might have been very different however, if a condition of distractability had been included.

What is apparent throughout this section is the variability between subjects on task performance. Not only is there little correspondence between a **behavioural** index of attention and attention measured on the above tasks but there is even considerable variability in task performance itself. This suggests that the criteria for

inclusion of patients into groups should be re-examined. This study used the conventional measure, duration of P.T.A.. It appears however, that for patients with injuries of this severity other factors should be considered. Probably the main variable affecting these data is the predominent type of injury sustained - frontal, diffuse cortical or brain stem. The size of this group was unfortunately too small to investigate these effects. Future studies which seek to examine the nature of attention disorder in very severe brain injury should take this factor into account.

CHAPTER 12

ATTENTION TRAINING

Introduction

Diller (1976) proposed a model for cognitive retraining in which the first step was the identification of the defective skill which needed to be remedied; (the next step being to select a task which adequately reflects this skill). Such a premise may appear so obvious that there is little merit including it in a "model" of cognitive retraining. It remains an embarrassing fact however, that the clinical practice of cognitive retraining largely ignores this basic rule.

Diller's brief paper was sufficient to influence a growing number of American psychologists with interests in rehabilitation and neuropsychology, generating a number of procedures for the remediation of cognitive deficit following brain injury. Diller and his colleagues (Diller et al 1972; Diller et al 1974) concentrated mainly on the problem of visual scanning and visuo-motor deficits. Success has been claimed by the above authors, suggesting that their training procedures can improve behaviour which relies on visual perception. The clinical value of such procedures and the degree to which they generalise from the training situation (using

laboratory tasks) remains in some doubt however. This is almost certainly because an appropriate way of measuring treatment does not appears to have been attempted. Where reports of success are presented, the evidence appears to be anecdotal or based on evidence of training task performance without evidence of more general clinical change.

Another cognitive retraining approach that has flourished over the last ten years has been directed at improving memory deficits following brain injury. Procedures used in this area have been investigated more systematically, often using single-case subject design (Gianutsos and Gianutsos 1979; Gianutsos 1980; Wilson 1982 and Wilson 1984).

The success of such training procedures remains equivocal. Properly controlled research designs do show that as a result of intensive and often long term training, even very severely amnesic patients will show a significant improvement on some measure of memory, compared to baseline. What has not been established however, is the generality of such memory training procedures to support their clinical utility. A general criticism of memory training is that training a patient to use a particular strategy: visual imagery (Wilson 1982), mnemonics (Crovitz 1979) or rehearsal strategies such as the PQRST method (Glasgow et al 1977), proves that the strategy can be **learned** but not successfully

applied during day to day behaviour. This seriously questions the practicality of such procedures.

One reason for patients being unable to **apply** such strategies **spontaneously** is the nature of the memory deficit. The clinical impression is not so much of **memory loss** but of a **failure to remember.** Typically, one finds that patients complain of problems of failing to remember something at an appropriate time. If they are reminded or cued in some way they will remember the information or the situation. It is the **spontaneity** of such recall that is affected. In the amnesic states of dementia or the Korsokoff syndrome the memory problem is quite different. There, patients actually **forget** the substance of memory and often, no amount of cuing will bring it back.

The implications are (Wood 1984b) that the memory retraining strategies do not work because the patients do not **spontaneously** remember to use them. It is argued that if information was processed efficiently, in an organised way, memory would improve, as well as many of the other cognitive and behaviour problems that rely on efficient information processing. This is the simple rationale for attempts at attention training, which is offered either as an alternative or a preliminary to other forms of cognitive training. Clearly, unless information is taken into the system, in a systematic and organised way, the system's ability to use that information or reproduce it appropriately will be

defective.

Different kinds of attention training procedures may be required depending on the nature of the attention disorder. This chapter describes three different ways of dealing with attention difficulties.

(1) The first is basic to therapy activities. It involves the problems produced by distractability or short attention span, which affect the patient's co-operation **in** therapy and consequently the potential benefits **of** therapy. The treatment of **sustained** attention will be illustrated using single-case subject designs.

(2) The second procedure is in many ways an extension of the first. It involves an attempt to increase attention span whilst, at the same time, improving the **efficiency** of information processing. This means that the patient not only maintains an attention set but has to make an active evaluation of the information presented and respond appropriately.

(3) The third procedure is a further extension of training. Not only does it require the maintenance of an attentional set with the appropriate processing of information, but it introduces **speed** factors which varies the **amount** of information the subject has to process at any given time. This is based on a computer task which is evaluated for its generality as a training task within experimentally controlled conditions.

12.1 Attention to Task Training

Increasing attention span through contingent token reinforcement is not a new idea. It has been successfully applied to increase attention and decrease overactivity in hyperkinetic children, (Staats 1968). There have been no attempts to apply this technique to the severely brain injured however. Consequently, a positive reinforcement procedure was used to see if it could successfully increase the duration of directed attention during therapy sessions. Tokens were given to the patient for directing attention (maintaining head posture and directing gaze toward the therapist) during therapy sessions. Examples of such attempts are given in two single case studies (Figures 12.1(i,ii)).

Subjects

The two patients studied were similar in many respects. Both had sustained a very severe brain injury in road traffic accidents 4 years earlier. Evidence of brain stem injury was present in both, together with areas of cortical damage. They had a PTA duration of more than 2 months and were distractable with short spans of attention. Their cooperation in therapy was minimal, not because they were beligerent or deliberately uncooperative but because they were continually attending to things which were not part of the therapy activity. This made any kind of treatment programme difficult to

pursue and was a source of continuous irritation to the therapy staff.

Procedure

Staff monitored a patient's behaviour during therapy sessions. A time sampling procedure was used to record behaviour. At two minute intervals the patient was recorded as either attending to task, or not attending. On the basis of this simple binary record, token reinforcement was delivered. A maximum of 15 tokens could be earned during a 30 minute session. The patient had to earn more than a designated number of tokens in order to exchange them for a reward (sweets, cigarette, etc). The number was fixed by the staff (without the patients knowledge) and organised according to a sliding scale, which increased as the patients behaviour improved.

Results.

Programme 1. Figure 12.1(i) shows the response to programme 1. This incorporated two therapy activities (speech and O.T.). Although there was an improvement in behaviour the response trends were not significant, either for the speech ($F= 1.096$; $df= 1/6$) or occupational therapy activities ($F= 1.434$; $df=1/6$). There was however, a significant improvement when the treatment phases were compared to baseline, using a Student's t analysis. Occupational therapy ($t= 6.54$; $df=7$; $p< 0.001$); speech therapy ($t= 6.12$; $df= 7$; $P< 0.001$). These scores indicate a type 2 curve (see page 120) which confirms a

treatment effect.

Programme 2. Here, a treatment effect was recorded by an A-B-A-B design (fig 12.1(ii)). The baseline **A** phases were recorded over two five day intervals. The recordings took place in different therapy activities during that time. It was not possible to restrict these measures to one particular therapy activity because the treatment time table varied from day to day and week to week. This might account for some of the variance in the data. Length of treatment in each phase was determined by other rehabilitation needs and in particular the need to avoid carrying out such training procedures at times when medication, which might alter arousal (and probably attention), was being used.

A non-significant trend was recorded for the first treatment phase ($F= 1.98$; $df= 1/7$) although there was a significant improvement compared to baseline ($t= 8.11$; $df= 7$; $p< 0.001$). On completion of the first treatment phase the behaviour recording shows a rapid return to pre-treatment levels (the second **A** phase). The reintroduction of the reinforcement procedure produced another non-significant trend towards improvement ($F= 1.22$; $df= 1/7$) but again the treatment phase was significantly better than the first baseline ($t= 3.57$; $df= 7$; $p< 0.01$), indicating that the changes in attention are due to a treatment effect, rather than an artifact of the general ward programme.

12.2 Special Task Training

One expects more from a treatment programme of this kind than simply compliance or cooperation; one hopes for cognitive improvement. Consequently a method was tried in which patients were trained on tasks which required not only the maintainance of an attentional-set but efficient information processing. To determine whether any improvement in performance would generalise to behaviour in other treatment areas two outcome measures were included, (1) a behaviour recording of attention during therapy activities and (2) measures of auditory recall memory.

Two tasks were chosen for this training programme; an **auditory vigilance task** of the kind described in chapter 10, to improve auditory/verbal aspects of information processing. The other concentrated on visual aspects of attention using the **Possum Basic Skills Teaching Machine.**

Subjects

Four patients from the Kemsley Unit were included in this training programme. Two were used in the above study and the other two have been described in the text relating to figures 7.3(iii) and 8.3(iv). In addition to all the severe head injury characteristics described earlier they also had greater difficulty maintaining attention than most other patients. This was creating particular problems in therapy activities and therefore interfering

with progress in rehabilitation.

Procedure

Training Tasks

Auditory attention. The auditory task was a version of the auditory attention task described in chapter 10 , but in this study, lasted 30 minutes instead of 15. It presented a sequence of digits to the patient at the rate of six digits per five seconds. At random intervals in the series a 'target' sequence of three odd digits was presented. The patient had to respond to this target by tapping the top of the table. Misses and false positives were recorded by the experimenter who followed the digit series from a printed sheet, on which he recorded the errors. Seventy targets were presented in each series.

The task was given once a day for twenty eight days. Four alternative tapes were available with a different digit series on each. The tape was changed every four days to prevent any possibility of a learning/practice effect occurring.

To ensure a reasonable degree of effort, performance on this task was linked to a system of token earnings. Each time the patient made a correct response he was rewarded by placing a token on the table. Every time an error occured a token was taken away. This negative punishment contingency not only balanced the reinforcement schedule

but also avoided a high proportion of false positive responses.

Visual Attention. The visual attention task used a Possum Basic Skills Teaching Machine (Possum). This piece of apparatus was originally designed as a teaching aid for mentally handicapped patients with communication problems. It was adapted to train visual attention by using a symbol matching task linked to a programme of contingent negative punishment (loss of tokens). It was carried out in parallel with the auditory attention task. The patient was required to sit in front of a panel (56X40 cm) on which was placed a transparency containing 32 symbols, divided into two 4X4 matrix panels, each cell measuring 7X15 cm. The matrix on the left hand panel contains 16 different symbols, one in each cell. The right hand matrix has the same 16 symbols but distributed in a different order throughout the matrix.

The task begins when one of the symbols of the left hand matrix is illuminated. The patient is required to move a scanning light, by pressing a button. This light travels from left to right across the top of the matrix; when the lights position coincides with the column containing the target symbol the patient stops the light, again by button press, to allow an alteration in movement, from the horizontal to the vertical. The movement down the column is started, again by button press, until the light is positioned over the target symbol. A final button

press stops the movement.

Task difficulty could be varied either by increasing or decreasing the speed of the scanning light. By increasing speed, the information processing requirements of the task are increased. Reducing speed increases the sustained attentional component because although the scanning light is easier to monitor the patient has to direct his attention to the task for a longer period.

Audio feedback is given on the patients response. A tune (positive feedback) if it is correct and a "raspberry" (negative feedback) if not. The stimulus illumination light on the left hand side of the screen will only advance after a correct match has been made. If an incorrect match is made then the same stimulus remains illuminated and the patient has to repeat the procedure.

In addition to a mis-match two other kind of errors were recorded: (1) **a column miss**, when the scanning light was allowed to move past the column which contained the target symbol: (2) **a symbol miss**, when the scanning light, although in the correct column, was allowed to move past the target symbol. These errors were included to increase the attentional requirements of the task.

To locate the 16 symbols in a matrix took on average 30 minutes. (This, added to the auditory vigilance task, gave each patient one hours training each day for a period of 28 days). In each case there is a five day baseline recording performance **without** token

reinforcement. This was particularly useful for the Possum task because it allowed us to select the optimum speed for the scanning light at the start of training.

Each time an error occurred the patient had a token removed from the "bank" of tokens placed by the side of the task (negative punishment). In order to exchange these for a reward after the session, the patient had to **retain** a certain number of tokens (determined on a sliding scale as the training progressed). This was preferred to the administration of tokens (positive reinforcement) because a pilot study showed it to be more effective and easier to administer. The tokens for both types of training task were added together to determine the type of back-up reinforcer available.

Outcome Measures

(A) Attention to task behaviour. This was measured by recording the attention to task of such patients both before training took place and at the end of the training period. This was a binary record, the patient was either observed to be attending (directing head and eyes towards therapist or therapy activity), or not attending. The recording was made as unobtrusively as possible, either by the therapists themselves or by a member of the nursing staff who would normally have been present at the time. It was not a continuous record of attending but sampled such behaviour at two minute intervals (as

described in the single case studies above).

(B) Auditory-Verbal Memory. It is reasonable to assume that any improvement in auditory information processing might improve performance on verbal memory tests that are presented in the auditory modality. To investigate this possibility three verbal-recall tests of memory were given before and after attention training. These were:-

(a) Digit Span from the Wechsler Memory Scale (WMS), Wechsler (1945) as a measure of immediate memory.

(b) Logical Recall Memory from the WMS, to see if training could improve the processing of information that had meaning and structure. Two measures were obtained; immediate story recall and recall after a delay of 30 minutes.

(c) Rey New Word Learning Test Rey (1959), to see if improved processing led to any improvement in new learning capability.

It is appreciated that four subjects is not enough to do a statistical comparison that will allow a proper evaluation of treatment outcome. This could not be avoided however. These were the only patients at the time who satisfied the criteria for admission into the study. This involved taking them out of other therapy activities that were regarded by the staff as equally, if not more, important than an untried procedure for improving attention.

Other Procedural Points

The general rehabilitation programme allowed only one patient to be trained at a time. An overlap of 5 days was organised however, so that baseline measures could be collected for each patient.

The baseline measures differed in two respects from the training programme. Firstly they were shorter, each task lasting 15 minutes as opposed to the 30 minutes during the formal training procedure. Secondly, performance was not associated with any reward, as was the case during training. These measures were obtained immediately prior to commencing the training programme.

Results

1. Training Tasks

The data for each week of the two training tasks are presented in table 12.2(a). A measure of improvement on both tasks was obtained using a two way repeated measures ANOVA distributing the 224 measures equally in a 2x28 design, analysing the daily error scores for each task.

All patients showed a progressive improvement as training continued,($F = 5.074$; $df = 27$; $P < 0.01$). This indicates not only an improvement in their ability to sustain attention for longer periods but an ability to process information more efficiently, allowing less errors to occur. There

was no difference in patient performance between the two training tasks (F= 0.596; df=1), suggesting that neither task proved too easy or difficult and that both were useful training procedures.

Table 12.2(a)

The mean error scores for both training tasks recorded at seven day intervals

	D A Y S				
	1	7	14	21	28
Auditory					
Mean	36.5	21.7	20.7	13.5	9.7
S.D.	17.9	14.7	9.9	8.6	6.7
Visual					
Mean	19.7	20.3	13.0	8.7	2.5
S.D.	8.3	7.9	6.4	5.3	1.2

2. Outcome Measures

2.A Attention To Task Training. The purpose of the training task was to see if any improvement **generalised** to other aspects of attention. To analyse the behavioural data the **'attending'** scores for the four patients, recorded during each two minute period of a thirty minute treatment session, were compared before and after training (Table 12.2(b)). A student's t for correlated samples showed a significant improvement after training ($t = 5.03$; $df = 14$; $p < 0.001$).

TABLE 12.2(b)

Total number of times the patients were observed to attend during a 30 minute treatment session

	TWO MINUTE INTERVALS														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Pre	12	14	6	6	8	10	10	8	10	9	10	6	7	3	4
Post	17	15	14	10	8	15	12	14	12	10	14	15	6	9	8

2.B Memory. The effect of improved auditory processing on verbal memory was measured by comparing the performance of the four patients on the three measures of memory before and after training. Table 12.2(c) shows that no improvement was obtained in any of the memory tests. Statistical analysis was not carried out because of the small number of patients and the apparent lack of effect.

Table 12.2(c)

Each subjects raw data from the memory tasks, used as an outcome measure of improved auditory information processing

		Digit Span		Logical Recall		Rey NWLT	
		Pre	Post	Pre	Post	Pre	Post
P	1	11	7	2	5	23	26
T	2	6	4	0	0	21	22
S	3	8	10	3	4	20	43
	4	9	9	6	7	23	26

Commentary. The results of this study show that attentional **behaviour** can be changed. Improvement in performance on both training tasks suggests that not only did patients manage to sustain attention for a longer period but, whilst doing so, they became more efficient at processing the information presented by such tasks. This is encouraging because it suggests that improvement in attentional aspects of behaviour will allow the patient to be more receptive to information presented during therapy activities, thereby improving the patients potential for further recovery. It is disappointing in this respect, that behavioural improvement did not translate into cognitive improvement of a kind measured by tests of auditory recall memory.

12.3 Controlled group study to evaluate a computer-based attention training programme.

Introduction

Computers have been used in an assessment or diagnostic capacity since the mid-seventies and studies by Lucas (1977) and Carr et al (1983) show that the majority of patients respond very well to computers, in some respects preferring them to a doctor or psychologist.

Gianutsos (1980; 1981), Lynch (1982) Skilbeck (1984) and Wood (1984b) have drawn attention to the use of micro-computers in rehabilitation. Lynch concentrates on the use of standard computerised games but as Wood points out, these have certain disadvantages because they are designed for entertainment, not therapy. It is not always easy to score a patient's improvement on a computer game in a way that is meaningful for therapy. Also there is a problem in knowing which particular cognitive function one is trying to train. Most games involve a multiplicity of skills, making it impossible to state, with any confidence, that a particular task is having a specific effect on any individual cognitive process. There is also the problem of establishing task difficulty when isolating and training a particular skill.

Gianutsos has pointed out that tasks can often be programmed and presented in a game form making them more

attractive to the patient. In a recent review of this area Miller (1984) agrees that computer procedures are attractive for the patient, allowing them to work independently, thus freeing the therapist's time for other things. Computer based training procedures can be adapted to the individual's needs and levels of ability. Accurate and objective record keeping of progress and performance is another advantage.

Miller argues that the effectiveness of computers in this area has not really been evaluated. Claims by Lynch (1982) that brain injured subjects improve their performance on computer based games do not attempt to show that this improvement was associated with change in any other aspect of behaviour. They also used subjects who varied considerably in the severity of their injury and usually provided training during the early recovery period. This could mean that cognitive abilities were recovering spontaneously and that any change which occurred at the time of computer training simply reflected such spontaneous recovery. Control groups were not used to substantiate their claims.

A further criticism of existing computer procedures in rehabilitation is that they often bear no relationship to an established **theory** of cognitive function. Baddeley (1984) points out how important it is to base treatment on a theory of cognitive processing. This way, one should be able to predict that an improvement on a training task

will produce an improvement in behaviours associated with the cognitive process upon which it is based.

Aims

The present study evaluates a computer based procedure, taking into account the pitfalls and criticisms referred to above. The subjects are all severely brain injured patients who are well out of the period when spontaneous recovery can be expected. The training procedure is based on an information processing model and independent outcome measures are included to determine its generality. A no-treatment patient control group and a normal control group are included to test for practice effects.

Subjects

Group 1. Ten severely brain injured subjects receiving in-patient rehabilitation comprised the experimental **(training)** group. Their characteristics can be seen by referring to table 12.3(a). They were the first ten patients who, on clinical criteria, were designated as having attention problems, mainly involving the speed or amount of information they could handle, evidenced by slow responses and divided attentional deficits. They were clearly well out of the period where spontaneous recovery could be expected and the clinical records of many of these patients indicated that they had achieved a plateau in their recovery or were considered to be at the limits of their potential recovery.

Table 12.3(a)

Subject data for the three groups

Group 1 = Patient Training Group

Group 2 = Patient Control Group (No training)

Group 3 = Normal Controls

	Groups					
	1		2		3	
	(N= 10)		(N= 10)		(N= 7)	
	Mean	SD	Mean	SD	Mean	SD
Age	28.4	8.7	27.2	9.9	29.4	10.3
PTA*	2.4	0.5	2.7	0.4	-----	
TSI**	27.5	5.8	36.5	15.6	-----	

* Post Traumatic Amnesia (Months)

** Time Since Injury (Months)

Group 2. This was a second patient group matched for severity of injury and time since injury with group one. They also were in-patients on the Kemsley Unit, receiving exactly the same general rehabilitation programme as the previous group. They were included to control any effects that the general rehabilitation programme might have on the aspects of attention measured in this study. This group completed the same baseline and follow-up outcome measures as group one but did not receive any training. Ethical problems were avoided by including these patients in the training procedure after this study had been completed (if their clinical condition still warranted such a move).

Group 3. This contained seven normal controls matched for age and sex with the two patient groups. The control subjects were all members of the community and had no experience of psychology or psychological testing methods.

Procedure

1. **Design of Study.** Table 12.3(b) illustrates the design of this study. Initially, the subjects all participated in several independent baseline measures. Because of the day to day variability in performance on cognitive tasks, the patients completed each baseline measure on two occasions. The **difference** between the scores is used for comparison purposes.

Group 1 received training on the computer task for one hour each day over a period of 20 days. At the same time they participated in the same rehabilitation proramme as group 2. The latter received no attention training.

The training period stretched over a period of 28 days. Training could not be carried out at weekends so the data were collected over 4 x 5 day intervals. At the end of the training period, the three groups repeated the baseline measures in the follow-up(1) condition. Again, each measure was repeated on two occasions and the difference score used.

After a second interval of 20 days, the outcome measures were repeated in the same way as previously described (follow-up(2)). This was to see if any improvement following attention training, was observed to continue or simply be a short term response, contingent upon the training procedure itself.

Table 12.3(b)

Design of computer training programme

Days	GROUPS		
	1	2	3
0	BASELINE		
1	TREATMENT	NO TREATMENT	NO TREATMENT
28	FOLLOW-UP (1)		
35	NO TREATMENT		
63	FOLLOW-UP (2)		

The Training Task. A training procedure was developed using a Commodore 64 computer with a 1541 disc drive, linked to an Epson RX80 printerand a Sony Trinitron monitor.

The task was directed at training information processing in the visual modality. The patient was required to monitor a line of symbols moving from left to right across a video screen. Figure 12.3(i) illustrates the screen presentation of the stimulus conditions existing during this task. The movement of these symbols was not continuous but occured as a series of discrete steps, movement not being perceived unless the subject continually scanned the line from left to right. Fixation at any given point does not produce an impression of movement.

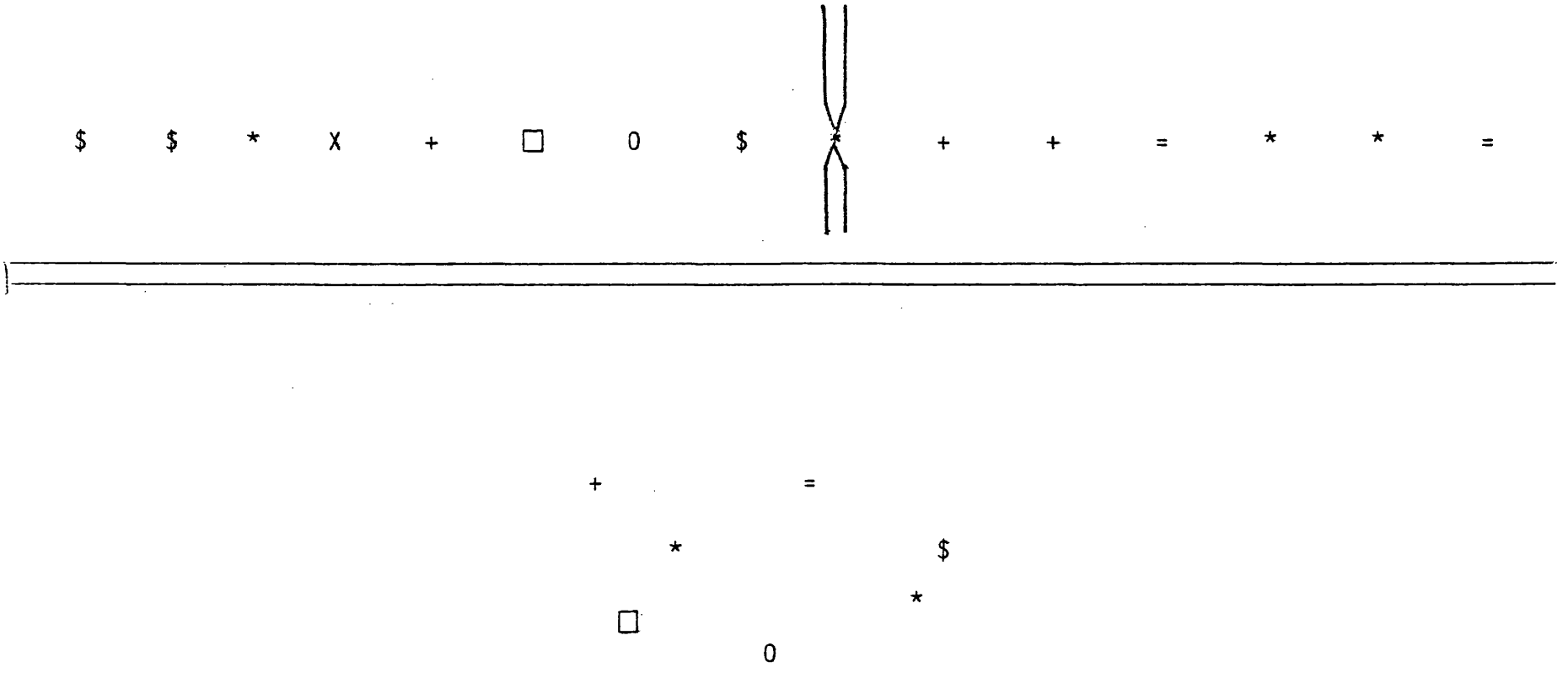
At randomly spaced intervals in the symbol series a target stimulus is located. This is represented in figure 12.3(i) by an asterisk. The symbols pass through a **'gate'**. When this target is located in the points of the gate the subject is required to make a response by pressing the space bar on the computer keyboard. The timing of this response is critical. Pressing the space bar either too soon, or after the target has passed through the points of the gate, will record a false positive error. Not pressing the space bar at all would indicate a 'symbol miss', subsequently scored as a different category of error.

Feedback of results is given according to the correctness of the patient's response. A correct response (a hit) is indicated by the symbol exploding, with the appropriate auditory feedback. If a miss or false positive occurs then a different kind of auditory feedback, a 'raspberry', is given.

The task did not require the subject to respond to every target; sometimes a response had to be inhibited. The decision to press or not to press the space bar on target presentation was determined by the presence or absence of the target stimulus in an **array** below a horizontal dividing line (fig 12.3(i)). This **array** would change in two ways at random intervals throughout the task. (1) The **position** of the symbols could change, forcing the patient to re-locate the presence of the target symbol in the array. (2) The second type of change involved not only a position change but the **omission** of the target symbol from the array. When this happened, the patient was required to with-hold a response when the target symbol passed through the gate. These changes occurred randomly throughout the task.

The level of difficulty was determined by the speed of the target line. Initially this was set according to the patient's anticipated level of ability, thereafter, the starting speed could be determined according to the patient's prior performance. During training the speed of the target line was adjusted to the patient's

FIGURE 12.3(i)



The screen layout of the attention re-training programme. The row of symbols in the top half of the screen contains a target symbol (*). The subject responds only when the symbol is both in the "gate" and the bottom symbol array.

performance. At the end of each game the computer automatically adjusted the speed of the target line according to the hit-rate recorded during the previous exercise. A hit ratio of more than 80% would result in an increase in speed by 5%. A hit ratio of below 40% on the other hand, will reduce the speed by the same amount. This ensures that the patient is always performing at an optimum level of performance. The computer recorded the hit and error rate automatically, printing the results out at the completion of each 'game'.

This task can be regarded as having the following requirements.

(a) **Visual Scanning**; the target line had to be scanned left to right to give the impression of movement and to judge the approach of the target into the gate. Also, the patient had to scan down the screen, in an organised way, to monitor any changes in the symbol array.

(b) **Perceptual Discrimination**; the size of the symbols was deliberately small (approximately 5 mm on the Trinitron screen). This meant that the patient needed to scrutinise each symbol to discriminate one from another in order to ensure that they responded correctly. Some symbols were similar in character e.g. $ and &.

(c) **Judgement and Anticipation**; the patient had to anticipate when to press the bar, based on the speed of

movement of the target line. Usually, several false positive errors occurred early in each training session until the patient judged the timing of a response.

(d) **Motor response**; even if the decision about **when** to respond is accurate, the motor response (in a choice reaction time paradigm) has to be made. One can regard (c) above as a decision time while (d) can be described as a motor response time.

The components of this training task indicate that it is very much related to improving psychomotor aspects of performance. Also, it provides a measure of the speed at which a patient is able to process and accurately respond to information which has to be scanned in an organised manner. As performance improves the speed of stimulus movement and change is increased, further increasing the information processing demands of the task because more information has to be processed in a shorter (or at least the same) time. The patient is therefore required to **divide** attention over a larger stimulus input.

Each patient was trained for 60 minutes a day for 20 days over a 28 day period. Unfortunately, training was not carried on during weekends.

Outcome Measures. In addition to the training programme a series of outcome measures were obtained to determine any generalisation effects from the training programme. These were as follows.

1. **Psychomotor Measures.**

(i) **Pursuit Rotor:** A standard pursuit rotor task using a circular track for the target light. Auditory feedback was provided to indicate time on target, recorded in seconds over five minutes.

(ii) **Digit Symbol:** This checking task from the Wechsler Adult Intelligence Scale,(Wechsler 1958) required the subject to write into a box a symbol, corresponding to a given number selected froma table above the display.

(iii) **Choice Reaction Time:** A six choice reaction time apparatus was used. This was the Leeds Psychomotor Tester described in chapter 11. One hundred decision and motor reaction times were recorded.

(iv) **Simple Rection Time:** The simple reaction time task (SRT) described in chapter 11 was used.

2. **Vigilance**

(i) **Visual Vigilance:** A specially designed computer task presenting stimuli (pink dots) on a video screen at random intervals over 30 minutes. The score was the mean reaction time (in milliseconds) showing the time each subject took to respond to a stimulus for each five minute period throughout the task. To maintain a high level of responding (Posner 1975) the high event ratio task was

chosen. This presented approximately 60 stimuli within that period.

(ii) **Choice Reaction Time (20 minutes).** This used the CRT as a continuous performance task, in the same way as that described in chapter 11.

3. Behaviour Recordings

(i) **Attention to Task Recording:** This was the kind of fixed interval behaviour recording described in chapter 11. Five, one hourly recordings were obtained over a period of five days.

(ii) **Attention Rating Scale.** Nursing and therapy staff were asked to bisect a line, one end of which rated "poor concentration in therapy", the other side, "good cooperation in therapy". No scale was marked on the line, to avoid unnecessary bias and the procedure approximated a "blind" rating because staff were not told whether the patient being rated was in the training group or not.

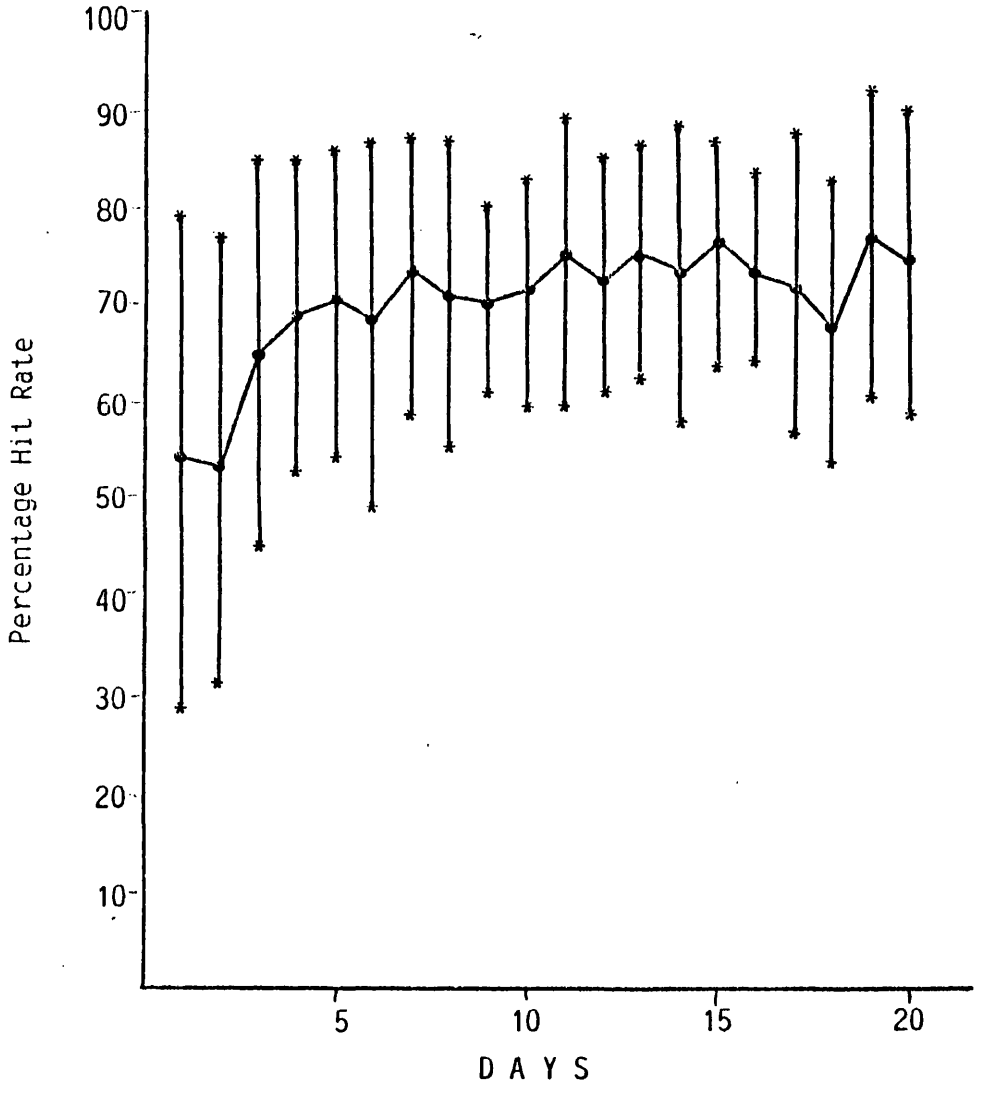
RESULTS

The Training Task

Figure 12.3(ii) shows the performance (hit rate) of the training group over 20 days. At the start of training there is considerable variability in performance. Over the training period this variability in individual performance becomes less, levelling off at around 25% of the mean hit rate for the group. The false positive and non-targets-hit scores are not represented because they were never higher than 5% of the hit-rate scores throughout the training period.

Figure 12.3(ii) shows a gradual trend toward improvement for the group over 7 days ($F = 19.6$; $df = 1/6$; $p < 0.01$), thereafter levelling off at around 70%. It must be remembered that the speed of stimulus change was continually adjusted by the computer according to the subjects performance. This means that the scores represent each subjects performance at the optimum speed for the individuals level of ability.

FIGURE 12.3(ii)



Means and Standard Deviations of the daily hit-rate for the training group (n=10) during an attention training programme.

Outcome Measures

Comparison of the three groups was made by calculating the **difference** scores for each subject between conditions, e.g. baseline and follow-up(1), baseline and follow-up(2). A between group comparison was made using a one way ANOVA, except for the behaviour measures when only the two patient groups were being compared; here the data was analysed by an independent Student's t test. The results are displayed in table 12.3(c).

The only measures to produce significant differences between baseline and follow-up(1) conditions were those which recorded **behaviour** changes (attention to task recording, $t=2.339$; $df=18$; $p< 0.05$: attention rating scale, $t= 2.176$; $df=18$; $p< 0.05$). The lack of significant difference scores for these measures, between follow-up(1) and follow-up(2), show that the changes were maintained for the **8** patients of group 1 who remained on the unit for the second follow-up assessment.

The only other measures to record a significant between-groups effect were the choice reaction time measures. These unfortunately cannot be interpreted as a treatment effect because they occur between the two follow-up conditions or between baseline and follow-up(2). This improvement must therefore be interpreted either as a practice effect or a general effect of the rehabilitation programme.

Table 12.3(c)

A one way ANOVA and independent Student's t analysis between baseline and follow-up conditions for each outcome measure. The analysis is performed on the subjects difference scores between each condition

F values = 2,27 df

t-values = 18 df

Measure	Conditions		
	BL-FU1	BL-FU2	FU1-FU2
1. PSYCHOMOTOR (F values)			
Pursuit Rotar	1.657	1.407	1.285
C.R.T.(x 100)	1.743	2.63	8.133**
Digit Symbol	1.768	0.512	2.361
S.R.T.	1.856	0.724	0.262
2. VIGILANCE (F values)			
Visual	1.951	2.012	1.243
C.R.T.(20 min)	0.141	8.79**	7.21**
3. BEHAVIOUR (t values)			
Attention To Task	2.339*	1.561	-
Attention Rating	2.176*	1.322	-

* = p< 0.05. ** = p< 0.01.

Commentary. There is no evidence to show that attention training can improve performance on tasks designed to measure psychomotor or vigilance performance. One obvious explanation for this is that they were the wrong measures, either because they were not sensitive enough or simply inappropriate to the kind of process being trained. This **may** be the case, but if it is, it is difficult to imagine what other (better) outcome measures could be used?

Another reason for lack of such improvement is that the training sessions (1 hour) were not long enough or did not occur with sufficient frequency (once per day) to effect any psychometric changes. It must be remembered however, that this was designed as a **clinical** procedure and, to a large extent, subject to the laws of parsimony with regard to the amount of time therapists can afford to spend on such activities. Had the training period lasted any longer any effect from training would run the risk of being masked by the more general effect of the total rehabilitation programme which, from the reaction time results, might already have been happening. One way to avoid this would be to have a shorter but more intensive period of treatment, providing 2 or more hours of training each day. The problem with such a proposal is that (a) it might be impractical, considering the other rehabilitation needs of a patient and (b) might be rejected by the patient out of boredom.

A final reason for any lack of effect is the severity of the brain injuries sustained by the treatment group. These patients represent a small proportion of survivors of severe head injury and, in many ways, are not typical of the majority of patients who are likely to need or benefit from such training. It is encouraging therefore, that even this severity of brain injury will respond **behaviourally** to a training programme of this kind. This is an important change because it helps improve the learning opportunities of the patient, with respect to the amount of cooperation he gives therapists, the total time spent in therapy and, potentially, improving the amount of information the patient is able to process at any time.

Summary

Throughout the different attention training procedures there has been a consistent improvement in behavioural aspects of attention. Apart from the rating scale measure used in the last study, which involves a subjective element, these recordings have maintained a **rigorous objectivity**, being carried out by individuals other than the experimenter. Although in such cases it was often known which of the patients had received training, as opposed to the control condition, it is unlikely that any observer bias influenced the recording.

It is disappointing that throughout this series of

studies, behaviour change was never directly associated with cognitive change. Inevitably, improvements in concentration meant improvement in cooperation, increasing learning potential and the benefits of therapy. It is important to realise however, that in the studies of special task training (study 2) and computer based training (study 3), the cognitive demands were considerable. The trends toward improvement in both conditions provides an important measure, in itself, of the patients improved ability, what was lacking was evidence of any generalisation effect that could be measured psychometrically.

Anecdotally there were a number of observations which indicate generalisation of attentional change. Rehabilitation staff made a number of comments about improved attention, cooperation, clarity of speech and better balance, all relating to patients who were in the process of attention training. The critisism of observer bias or "halo effect" cannot always be justified in such situations. Some rehabilitation staff did not know that the patient they were describing was involved in training. As for observer bias, it was obvious that the inclusion of "cognitive rehabilitation" into an already packed treatment programme, was not a popular idea with a group of therapists predisposed to the physical and behavioural sequelae of such injuries. Supportive comments for this procedure were not going to be easily

obtained, even in the face of evidence of its effectiveness; the contribution of such spontaneous comments was therefore a welcome surprise.

CHAPTER 13

ANHEDONIA, BRAIN INJURY AND REHABILITATION

Introduction

Anhedonia (Meehl 1962) is the inability of some individuals to experience pleasure. Hedonic responsiveness can therefore be assumed to be an important characteristic of normal behaviour and one that may be of central importance in determining the degree of effort an individual is willing to put into a rehabilitation programme (Wood and Eames 1981; Wood 1984). Unless an individual is able to identify a particular experience as pleasant and therefore desirable or, at the very least, able to acknowledge other experiences as unpleasant or painful and seek to avoid or escape them, any kind of behavioral learning, relying as it does on positive or negative reinforcement contingencies, will be prejudiced.

Hedonic responsiveness appears to play an important part in the concept of motivation. (It has been argued earlier in this thesis that the radical behaviouralist approach is not acceptable when considering behavioural learning in humans, especially brain damaged humans). Belmont et al (1969) point to the importance of motivation in rehabilitation, describing how brain injured patients are

particularly vulnerable to motivational deficits which impede the progress of rehabilitation.

The presence of anhedonia (to any degree) may complicate or limit an individual's potential for recovery because their 'neutral' attitude to their situation reduces motivation to overcome the obstacles which face individuals recovering from severe head injury. Attempts to change behaviour by the application of selective rewards are undermined because such patients fail to perceive any value or sense of pleasure in the reward.

Kraepelin (1913) and later Bleuler (1911; 1950) both viewed the loss of the experience of pleasure as one aspect leading to deterioration during a schizophrenic illness. Rado (1956, 1962) gave anhedonia a more central role in the development of schizophrenia, suggesting that it was genetically transmitted and may be present both in "overt schizophrenics" and also in "schizo-types" who do not actually undergo a psychotic breakdown. Anhedonia is presumed to prevent the development of a normal and healthy sexual interest, reduces zest for life, impairs the ability to relate with other people and weakens the feelings for joy, affection, love, pride and self-respect.

Anhedonia has not only been considered important in schizophrenia. Klein 1974 and Klein et al (1980) considered it to have a central role in endogenous depression. Klein hypothesized that pervasively

anhedonic depression was associated with a more favourable response to tricyclic antidepressant medication than was likely from other depressions.

A neurological and biological basis for anhedonia has been postulated more recently. Meehl (1962, 1973) integrated anhedonia into a theory of neurological dysfunction and schizophrenia. He proposed that a defect in the organising action of pleasure or reward means that the integrating mechanisms of behaviour, and hence the organisation and control of goal directed thinking and behaviour, become impaired. Wise and Stein (1973) take up this argument, suggesting that anhedonia is the result of a genetic defect, produced by excessive 6-hydroxydopamine, which damages the brain's reward mechanism. Wise (1980) in a review of the evidence, concludes that there is a motivational role for dopamine in behaviour. This involves the signalling of **reward**, "which is translated into the **hedonic** messages we receive as pleasure or euphoria". He described the dopamine synapse as the site in the brain where, "the hedonic impact of the sensory message is first associated with the sense impressions of the external events which constitute **natural rewards**".

Klein also described anhedonia as a specific functional disorder of the central nervous system. Fawcett et al (1983b) investigated this suggestion in a group of depressed patients, using a questionnaire (Fawcett et al

1983a) to assess anhedonia. They found that anhedonic depressed patients differed from hedonic depressed patients; the former reporting extremely low levels of pleasurable experiences and being more severely and hopelessly depressed. This group were however, less neurotic than those reporting a normal pleasure capacity (the hedonic group) who were more emotionally labile.

This line of research led Wise and Stein (1973) to suggest that anhedonia may be a progressive disorder that becomes more severe with time, due to a combination of a biochemical fault and the fragmentation of the person's psychosocial experiences. The existence of anhedonia means that a person's thoughts, feelings and actions are not directed by a unifying concept, purpose or goal. This in turn affects motivation and a breakdown of progress towards recovery through rehabilitation.

Wood and Eames (1981) discuss hedonic responsiveness in the context of motivational problems following severe brain injury. Clinically, they associate this concept with hysterical or dissociative states, which radically interfere with motivation. They assumed that the problem was likely to result from damage to the frontal or septal regions, or their connections. This receives support from the comment by Wise (1980), describing the importance of dopamine in this type of response..."the anatomically critical dopamine synapse (is) that of the frontal cortex". Damage to this area can, "leave patients in

contact with the **sensory** dimensions but not the **hedonic** impact of their environment". Further reference was made to this problem in Wood (1984). Here it is associated with an intrinsic lack of drive which limits the incentive value of many reinforcers used to increase effort and motivation in a behaviour management approach to head injury rehabilitation. It has also been discussed by Eames and Wood (1984), where the problem is referred to as dyshedonia, suggesting different degrees of the disposition between anhedonia and euphoria.

The existence of brain mechanisms that control the experience of pleasure and pain suggests that some brain lesions will produce clinical states in which the ability to feel such emotions will be disturbed. The diffuse pattern of brain lesions following severe deceleration head injury, which often affect the deep structures (especially the diencephalon), seem to produce a reduction in hedonic responsiveness. This is likely to reduce a patient's incentive to work towards potential goals in rehabilitation, meaning that they make little or no effort to overcome their disability

Measures of Anhedonia

Anhedonia has usually been measured by rating scale or questionnaire (Cautela and Kastenbaum 1967; MacPhillamy and Lewinsohn 1974; Chapman, Chapman and Raulin 1976 and Cook and Simukonda 1981). In reviewing such measures, Chapman et al conclude that rating scale procedures, for example, the Phillips Prognostic Rating Scale (Phillips 1953) are not well suited as a measure of individual differences in anhedonia because some of the experiences contained in those scales are not available to everyone. Furthermore, Chapman also found that when schizophrenics use a rating scale to report their experiences of pleasure, they tend to use the extreme and centre positions of scales and neglect the intermediate positions. Chapman constructed new scale for physical and social anhedonia using a true/false format. Physical anhedonia covers a wide variety of pleasures: admiring the beauty of sunsets, eating, drinking, singing; social anhedonia covers all pleasures derived from having friends and being with other people. Using these scales, Chapman et al showed that schizophrenics had significantly higher social and physical anhedonia scores, but that the variation in responses between schizophrenics and normals meant that the anhedonia scale would have little diagnostic value used alone.

Cook and Simukonda applied the anhedonia scales to a sample of British schizophrenic patients to try and

relate anhedonia scores to symptom patterns. They predicted that anhedonic patients would be most likely to exhibit symptoms of apathy and withdrawal. They found however, that the physical anhedonia scores were only slightly higher in schizophrenics and that there was a sex difference, the female scores being no different from the normal group. For social anhedonia a larger difference was found but, as in Chapman et als study, the overlap between schizophrenics and normals was so great that the scales were not helpful for diagnostic purposes. No link between anhedonia scores and the type of schizophrenic symptom was discovered.

The aim of the present study was to determine whether the Chapman anhedonia scales could distinguish between a group of head injured patients and a control group, consisting of orthopaedic patients who had sustained various traumatic (bony) injuries, but no head injury.

METHOD

Subjects

Fifty five head injured patients, 34 male and 21 female, were tested. They were divided into two groups according to the severity of their injury (assessed by PTA (see table 13.1).

Group 1. This comprised 32 patients who had sustained very severe head injury; they were, or had been, inpatients of the Kemsley Unit.

Group 2 There were 23 members of this group. All were outpatient referrals and could be described (according to Russell's (1971) criteria) as **moderate** head injuries.

Group 2 differed from the severely head injured group (grp 1) not only in the length of post traumatic amnesia but also with respect to the physical and cognitive sequelae from their injuries. Group 1, generally speaking, exhibited more neurological sequelae than did the out-patient group. There was however, no difference between the groups on 'time since injury' - the severely injured group being tested on average 5.1 years after injury while the moderate group were 4.2 years post injury ($t = 1.79$; $df = 53$).

None of the severely injured group had been employed since their injury and most had spent their time either in hospital or some form of medical institution. Of the moderately injured group, ten were at work at the time of testing and seven others had work experience between the

time of their injury and the time of testing.

Group 3. In addition to the head injured patients, a control group of 41 patients, who had sustained orthopaedic injuries, not including head injury, was examined. The severity of their injuries ranged from a single fracture to multiple fractures of the kind experienced in road traffic accidents.

Table 13.1(a)

Characteristics of the three groups

		Group 1 (Severe)	Group 2 (Moderate)	Group 3 (Orthopaedic)
Age	Mean	31.65	28.70	33.70
	S.D.	14.45	10.50	20.50
PTA	Mean	3.20*	5.50+	----
	S.D.	1.60*	2.40+	----
TSI	Mean	60.55*	48.20*	----
	S.D.	34.30*	10.44*	----

* Months + Days

Measures

An anglicised version of Chapman et al's 66 item version of the physical anhedonia scale and 47 item social anhedonia scale was used. In addition a 17 item 'infrequency scale', adapted by Chapman et al from Jacksons (1974) Personality Research Form was included as a control scale, to exclude subjects who did not take the questionnaire seriously, were deliberately lying, or were not attending properly to the content of the statements. The infrequency scale consisted of statements such as "I have never combed my hair" or "I grow all my own food". Subjects scoring 5 or more on this scale were excluded from the study.

Twenty five of the severe head injury group (Grp 1) completed the questionnaire while still inpatients. A further 17 patients were sent questionnaires through the post with a letter explaining the purpose of the questions. Of these questionnaires, 12 were returned. Only seven were included for analysis because the other five contained infrequency scores greater than five which made them invalid.

The 23 moderately injured patients (Grp 2) all completed their questionnaires at the time they attended hospital for follow-up assessment.

For the control group (Grp 3), questionnaires were posted to the homes of selected individuals who were due to attend the orthopaedic outpatient clinic, with a letter

of explanation and a request to complete the questionnaire and hand it to a research worker on their next visit to the outpatient clinic. A total of 163 questionnaires were posted, of which only 56 were returned over a period of three weeks. Sixteen of these were excluded from the analysis because they scored higher than five on the infrequency scale.

Other Procedural Points

The statements of the questionnaire (see appendix A) were re-phrased to remove obvious 'Americanisms' (e.g. sidewalk = pavement; gotten = have received). This was to make it easier to understand and avoid potential confusion in those individuals not used to such phraseology. It is very unlikely that any meaning was lost in this translation. The statements of the questionnaire were relatively short, easy to read and understand and phrased for true/ false answers.

Every effort was made to ensure that the severely head injured patients fully understood each statement. Patients with dysphasic language disorders were excluded from the study.

RESULTS

The data from the physical **p** and social **s** scales were analysed independently using a one-way analysis of variance with planned (post hoc) comparisons.

Analysis of the **p** scale data shows a significant between groups difference ($F= 8.44$; $df= 2/93$; $p< 0.01$). Post hoc comparisons using the Tukey test shows the differences to be between the severely and moderately head injured groups (mean diff = 5.29; C. diff = 4.27; $p< 0.05$) and the severely head injured and orthopaedic control groups (mean diff = 6.62; C diff = 4.27; $p< 0.05$). The moderate head injury group did not differ from the orthopaedic controls (mean diff= 1.33; C. diff= 4.27).

The **s** scale also produced a significant between groups difference ($F= 6.91$; $df= 2/93$; $p< 0.01$). The post hoc comparison again showed a significant difference between the severely head injured and the moderately head injured groups (mean diff = 6.67; C.diff = 5.07; $p< 0.05$) and another difference between the severe and orthopaedic groups (mean diff= 6.87; C. diff= 5.07; $p< 0.05$). The difference between the moderately head injured and orthopaedic controls failed to reach significance (mean diff = 0.2; C. diff= 5.07).

Group 1 recorded 12 (37.5%) **s** scale scores which were more than 1 s.d. above the mean for the control group. This compared with 6 (26%) for group 2 and 7 (17%) in group 3. On the **p** scale, 15 (46.8%) in group 1, 3 (13%)

in group 2 and 7 (17%) in group 3 scored 1 s.d. above the mean.

There was a high correlation between the **p** and **s** scales for groups 2 and 3 (Group 2, r= .748; df=21; p< 0.001: Group 3, r= .738; df=39; p< 0.001). Group 1 also recorded a significant correlation, but not at such a high level(r= .437; df=30; p< 0.02). These findings are consistent with the results of Chapman's studies on normal individuals which showed a correlation of 0.6 for males and 0.51 for females. Analysis for sex differences in this study showed the only difference to be on the **p** scale for group 1 (t= 2.470: df= 30; p<0.05).

Comparison of Statistical Data with Clinical Findings

The absence of a significant difference between the moderately injured patients and the controls suggests that change in hedonic responsiveness is not a general legacy of head injury. Consequently, a within-group analysis of the severely injured group was made to determine what characteristics (if any) of this group could be associated with dyshedonia.

1 Reinforcement Incentive. All the patients in group 1 were either being treated in the token economy programme or had been before discharge. The number of tokens earned by a patient is directly related to the amount of effort or cooperation they give during rehabilitation activities. It is generally assumed by the staff working with such patients that amount of effort is determined by

the patients level of motivation. This in turn is determined by the degree to which the patient perceives **reward** in being 'well'. The tokens therefore, constitute intermediate reinforcers which reflect the effectiveness of his attempts to recover from injury.

By calculating the mean token earnings for each patient, over a ten week period, it was possible to estimate an individual's sense of reward. This is because the tokens not only earned privileges but reflected improvement of independence skills, showing the patient that recovery was continuing and (presumably) fostering enthusiasm to make even greater efforts toward further recovery.

Token earnings of patients who scored more than 1 s.d. above the control group mean (Grp 3) on either the **p** and **s** scales, were compared with token earnings of other patients from Group 1 who scored around the control group mean. Data were available for 10 of the 12 high scorers on the **p** scale and 11 of the 15 high scorers on the **s** scale. The two scales were compared independently. Neither showed a significant difference between token earnings (**p**, $t=1.24$; $df=25$: **s**, $t=0.199$; $df=26$).

The relationship between token earnings and anhedonia was also analysed by a product moment correlation. The correlation between the **p** scale score and token earnings was significant ($r= .424$; $df= 30$; $p< 0.02$). The correlation between the **s** scale scores and token earnings failed to reach significance ($r= .311$ $df=30$).

Table 13.2

Division of 32 patients into different types of brain injury

TYPE OF INJURY	(N=)	> 1 S.D. ABOVE MEAN (N=)	
		p-scales-s	
Brain Stem	6	2	1
Frontal	13	5	3
B.Stem + Frontal	10	2	5
Other*	3	1	2

* Other = 2 diffuse anoxic injuries
1 temporo-parietal injury

Table 13.3

Comparison of physical and social anhedonia scores with different types of head injury

Groups	Frontal	B/Stem	F+B.S.	Other
p scale				
Mean	19.09	22.00	15.77	17.33
S.D.	7.11	8.64	3.56	8.01
s scale				
Mean	24.00	24.00	20.80	21.50
S.D.	9.62	9.25	6.91	3.51

(2) Type of Injury. The 32 patients in the severely injured group were divided according to their 'recognizable' brain damage (table 12.2). This included, (1) major brain stem involvement, (2) predominantly frontal lobe damage (3) a combination of brain stem and frontal injury or (4) some other kind of recognisable pattern of damage (one patient had evidence of temporal-parietal damage; 2 others suffered diffuse damage from hypoxia).

This division is inevitably a crude one. **All** patients had sustained severe injuries, most in car accidents, with almost inevitable deceleration effects, (diffuse, often microscopic damage, e.g. Stritch 1969). As such, injuries were **typed** on the basis of the most obvious or prominent damage, using (1) **independent** neurological examination, (2) C.T. scan or EEG reports, and (3) the medical notes from referring hospitals.

To determine any relationship between type of injury and anhedonia, patients from group 1, with scores more than 1 s.d. above the control group mean, were examined to see if they included significantly more frontal, frontal-brain stem or brain stem injuries than could be expected by chance. This data is included in table 13.2. The number of high **p** and **s** scores were tabulated separately.

Because of the small numbers in each category the analysis was made by grouping patients with frontal or

frontal-plus-brain stem damage and comparing them with those patients without prominent frontal signs. No significance was found between the frequencies in each cell for either the **p** or **s** scales, both yielding the same Chi-square value of 0.078 (using the Yates correction for small numbers).

Group 1 patients with different types of injury were also compared to see if they scored differently on the **p** and **s** scales (Table 12.3). No differences were found for either scale (**p**, F= 1.08; df=3/29 :**s**, F= 0.295; df= 3/29).

Patients with frontal and brain stem injuries were compared to the others on the basis of their token earnings (table 12.4). A one way ANOVA found no significant differences between the groups (F= 0.144; df=3/29).

Table 13.4

The performance of different types of brain injury during rehabilitation. A comparison of the tokens earned for cooperation and effort.

Groups	Frontal	B/Stem	F+B.S.	Other
Mean	89.07	89.93	90.88	88.66
S.D.	4.85	9.23	4.91	11.48

DISCUSSION

The results of this study are equivocal. They support the observed relationship between severe head injury and changes in hedonic responsiveness. They do not however, provide convincing evidence for the organic nature of anhedonia. They show that a lack of hedonic responsiveness is not a general feature of brain injury, there being no significant difference between moderate head injuries and orthopaedic controls. Its presence in a group of severely head injured patients may be a direct consequence, either of the severity of the injury itself or the **type** of injury, produced by the mechanical processes involved.

Within the 'severe' group there are a relatively large number of patients with predominantly frontal and/or brain stem injuries. There is however, no evidence of a significant **relationship** between these injuries and anhedonia. There is no evidence that these patients experience any loss of reinforcement incentive per se, either when their token earnings are compared to equally severe injuries **without** a frontal or brain-stem focus, or when the anhedonia scores are compared between the groups. There is however, a low correlation between token earnings and the physical anhedonia scores for the severe brain injury group as a whole. This might imply a tendency for such individuals to perceive some aspects of their situation in a pessimistic way, although it cannot

be assumed to reflect an organic change of affect.

There appears little doubt from clinical observations that motivational problems in rehabilitation are more frequently encountered with the head injured than with other traumatically injured groups. Conclusions drawn from more detailed behavioural observations of patients with motivational disorders in a rehabilitation setting (Wood and Eames 1981; Wood 1984; Eames and Wood 1984) which suggest that many motivational deficiencies can be attributed directly to the effects of brain injury, in particular, lesions involving frontal and high brain stem (limbic system) structures, are not supported by the evidence from this study.

Although the data must, in part, reflect some influence of an organic behaviour change, they are inevitably complicated by the fact that the questionnaire responses also include the individuals **perceptions** of the community and their place in it. The orthopaedic control group are likely to perceive their injuries as a temporary unpleasantness which, in some instances may produce a permanent minor handicap (e.g. a limp), but which do not dramatically affect their social or physical adjustment or occupational potential. Similarly, the moderately brain injured group, although having experienced a distressing time, having to cope with various cognitive changes, occupational changes and re-adjustments, in some cases domestic crises, nevertheless see themselves as

relatively normal individuals who do not stand out in a crowd. The severely brain injured group on the other hand, with their long history of institutional care and marked cognitive and physical change are more likely to have become demoralised and to have given up hope of living anything like a normal existence. It is this **reaction to injury** rather than any fundamental organic change caused by the injury, that could have produced the high scores of that group.

There is a complicating factor influncing patient responses. Some injuries produce a euphoric disposition, even inappropriate or disinhibited behaviour. Many of these patients are **not** aware that their relationship to society has been permanently changed. Some loss of insight was known to affect some members of **both** head injured groups. This is likely to affect the accuracy of their social perceptions and hence their response to certain questionaire items. When insight is retained, on the other hand, it produces an awareness that physical handicaps, when they exist, are often permanent and likely to restrict physical mobility and opportunity for social relationships. Cognitive changes, which include memory deficits, problems of organisation and planning ability, make such patients dependent upon others for many of their daily needs and, to a large extent, force them to see their potential for gainful employment (often the most important measure for establishing personal

independence) as being completely beyond their capability.

The questionnaire responses must therefore include a major psychological component, and a reaction to, rather than consequence of, the brain injury. This should be considered when preparing the brain injured for discharge from rehabilitation centres. A greater emphasis on psychological counselling, or cognitive restructuring of the kind described by Prigatano et al (1984) would be advisable, both to help the patient adjust to changed personal and social circumstances and to prevent negative reactions to their injuries,in the form of a sense of hopelessness and developing anhedonia. Such reactions will reduce the patient's motivation, effort and personal incentive to recover from head injury.

CHAPTEEN 14

DISCUSSION

The focus of neuropsychology has traditionally been on the cognitive sequelae of brain injury (Brooks 1984) but is now widening to include many emotional and behavioural sequelae that comprise 'psychosocial' functioning. The introductory chapters reviewed several follow-up studies and found that those individuals who, after many years, were still unable to work suffered not from motor deficits but from psychiatric and cognitive disabilities which included changes in temperament and poor memory. These 'psychosocial' changes are most distressing to relatives and cause major problems to rehabilitation professionals. All of these studies found a marked tendency for physical deficits to recover over this time but emotional disturbance to stay the same.

The study by Fahy et al (1967) reported that the "psychiatric aspects" of head injury sequelae were related to **severity** of injury. Patients who had PTA's of more than 22 days showed severe psychiatric disability. In their study, the correlation between PTA and psychiatric disability was 0.68. More recently Bond (1975) repeated this study and also found significant correlation between the duration of PTA and the severity

of mental and social disability.

The behaviour studies presented in this thesis provide evidence, from a variety of situations, to show that many of the problems presented by the brain injured can be controlled and need not interfere with rehabilitation. It seems however, that the important issue is not that behavioural methods **can** work, but **how to make them work**? This thesis has pointed to several ways in which severe brain injury can affect neurological modulatory mechanisms, cognition and/or 'personality', to interfere either with behavioural learning, behaviour control or both. To obtain a successful outcome from behavioural methods, these **'constraints'** (Wood and Eames 1981) must be considered.

13.1. The effect of brain injury on the modulatory mechanisms of behaviour

The influence of neurological factors mediating many inappropriate behaviours was described in Chapter 5. A broad division was made between **temporal lobe** abnormalities, which produce sudden, often unprovoked responses, either of an aggressive or sexual type, while on the other hand, **frontal lobe** damage results in an **escalating** behaviour response, usually elicited by some minor frustration over which the individual seems to have little control.

Responses which are **elicited** as a result of a temporal lobe disturbance cannot effectively be controlled by the

application of behaviour modification techniques alone. Experience has taught us that a **combination** of behaviour management and an appropriate anticonvulsant (Carbamazepine) produces very satisfactory results in a relatively short period of time. Only two case studies are available to illustrate this. The lack of further evidence is due to ethical problems; once an effective treatment is recognised it cannot be with-held from patients who require it.

Other evidence is presented in Chapter 6. This examines the treatment outcome of 30 patients, 10 with temporal lobe abnormalities. Baseline data was not available for comparison with the treatment phase, however, if one accepts the frequency of aggressive outbursts recorded at the time of admission as representative of their behavioural history over at least the previous two years then one can appreciate the effectiveness of this combined treatment procedure.

There is no evidence that the same combination of treatment (Carbamazepine plus behaviour management) has the same (immediate) effect upon frontal lobe behaviour disorders. This is probably because frontal brain damage interferes with the **inhibitory** controls that are normally exercised in emotional situations. In this condition aggression often forms part of a more global "frontal lobe syndrome", which includes a blunting of various social skills and a tendency towards coarse behaviour.

An explanation for some aspects of behaviour in patients with frontal lobe damage is provided in studies by Teuber (1964) and Luria (1966). These show that such injuries disturb the **modulating** function which the frontal lobes exert on complex sets of signals regulating our interaction with the environment. Teuber's studies suggest that patients with frontal lobe injury are able to anticipate the future course of events but lack the ability to picture themselves as a potential agent in relation to those events. Luria's studies suggest that frontal lobe damage has a disruptive effect on the programming of complicated activities, with the result that many aspects of behaviour are not properly regulated. His experiments revealed not only a disturbed regulation of activity but disordered feedback and the failure to correct errors. He extrapolates from such neuropsychological studies to state that, in every day life, such deficits could impair many aspects of behaviour. The disordered regulation of activity may underlie the apathy and aspontaneity of many patients with frontal lobe lesions while impaired feedback may account for their lack of critical attitude towards the results of their own behaviour and their failure to modify it.

Often, these behaviours occur in patients who show a lack of awareness of the effect their behaviour has on others. Grosswasser et al (1977) commented on such disorders,

reporting that they posed **serious rehabilitation problems**, and even after 30 months into recovery from head injury showed no evidence of improvement. Brooks (1984) reporting on the Grosswasser et al study, stated that, "these patients have access to intensive and widely based rehabilitation facilities, designed to deal with a full range of problems suffered by head injured patients". This rehabilitation programme was not structured using a behaviour management approach although, in addition to psychotherapy, behavioural techniques were 'offered'.

In the present study, the introduction of behavioural contingencies for the disturbed or disruptive behaviour of such patients, appears (in almost every case) to have an immediate effect, but one which cannot be controlled. Continuation of such programmes is likely to produce a more stable pattern only after a number of weeks (sometimes months) have elapsed. Such results still tend to repudiate the conclusion of Grosswasser et al.

The advantage of the present study is that it was able to evaluate a particular method of patient management using patients who, by most clinically accepted definitions, were well out of a period when significant spontaneous recovery could be expected in short periods of time. Grosswasser et al and Brooks (1984) indicate that 30 months is the period when most recovery will occur, although a slower rate of progress may continue for three

or four years after injury. Thomsen (1984) has even suggested that a gradual behavioural improvement may continue for five to six years after injury. This kind of improvement is very unlikely to have influenced the behaviour change recorded during these treatment programmes. It must be remembered, that the patients in this study had exhibited their particular pattern of behaviour since the early stages of recovery, almost without interruption, until being admitted into this specialist unit.

Another advantage of this study was that, in the cases presented, there was no use of any psychotropic medication, Before admission, many of these patients were receiving large doses of Chlorpromazine and Haloperidol, without any improvement being recorded in their clinical state.

The observations and measures of behaviour change as a result of behaviour management methods make it possible to confirm a treatment effect with many brain injured patients providing that the following conditions were observed.

1. The treatment programmes is **intensive** and provided in a structured environment, utilising continuous reinforcement for a longer period than appears to be recommended in most behaviour modification conditions (Davey 1981).

2. The overall behaviour programme must be allowed to

continue for a period of time that will allow (a) **consolidation** of a learned response, (b) **weaning** off the token or other reinforcement contingencies and (c) **generalisation** from the structured behavioural setting into other 'real life' situations.

3. Sedative or other psychotropic drugs should be avoided. The use of a 'chemical straight jacket' appears not to produce successful results when prescribed for aggressive behaviours. The clinical history of many of the patients admitted to this programme was proof of that and, in the author's experience of a similar behaviour management approach with disturbed aggressive psychotic patients, the same rules apply. The reasons for this are not clear. Following head injury Chlorpromazine appears to have an epileptogenic effect, which, if only minor, appears to excite an aggressive response. Drugs probably affect the already impaired cognitive system, blunting perceptions and interfering with associational learning, thereby undermining the value of behavioural approaches which rely on a person forming associations between behaviour and the reinforcement contingency that follows.

13.2 Drive related disorders

In many respects there is a thin dividing line between the purely neurological and purely neuropsychological effects on behaviour. This is exemplified by the continuum of arousal, drive and motivational.

Disturbances of this 'mechanism' can have a profound affect on behaviour, especially behavioural learning.

Sokolov (1963) regarded arousal as being important for maintaining in the individual the readiness to respond. It is linked by Posner (1975) to a state of 'consciousness' which, "affects general receptivity to input of information". If the level of arousal can be regarded as an important pre-condition for a drive state, then we must regard an individual's given level of drive as being dependent upon the variable state of arousal. This relationship would appear to re-state the 'Yerkes-Dodson' law which postulates an inverted **U** shaped function of efficiency depending on the degree of **arousal** of the organism. Thus, not only alertness but also the appropriateness of behaviour will depend upon the individual being optimally aroused.

When considering clinical management and potential for recovery, we should try and distinguish between arousal, drive and motivational disorders. Arousal disorders interfere with both the reception of information and the capacity of the system to generate effort or drive. In conditions we describe as **amotivational** the information is processed and analysed correctly but without the 'evaluation criteria' which determines the pleasurability of that information and therefore, the purposeful effort or drive necessary for a response to occur. In the normal individual, drive strength has been related to need on

the one hand and the attractiveness of the stimulus (reward) on the other (Hull 1943). The behavioural distinction between motivation and drive is that drive does not contribute to the **direction** of behaviour. If we interpret drive as a physiological component (arousal) any increase in this state will lead to agitated and non-directed behaviour. If however, the individual is able to attribute an increase in drive to a state of need, the behaviour can become goal directed.

In negative behaviour disorders this appears to be precisely where the breakdown between drive and motivation occurs. A frontal lobe patient for example, is often incapable of consistently directing behaviour towards a goal. This was first **al**luded to by Lashley (1938). He separated motivation from drive by stating that motivation could not involve "general drive". He also appears to emphasise the cognitive aspect of motivation by linking it to emotion, which "evaluates" the content of information being processed by the brain before behaviour is emitted.

Gray (1972) also appears to make this distinction. He distinguishes drive from emotion and motivation. Drive is described as an "internal process, affected by changes internal to the organism". Emotions were also regarded as internal states but these are mainly related to changes taking place external to the organism. Gray associates emotion with the process of learning and, by

implication, motivation. How strong this relationship is, is difficult to say. He does not appear to be suggesting that they are part of the same process.

13.3 Problems of Attention

There are many cognitive deficits influencing the degree and pattern of recovery and the eventual outcome in terms of return to work or acceptance by the community or ability for independent living.

The argument presented in this thesis implicates a disorder of the **information processing system** (referred to under the general term of attention) as the primary cognitive deficit responsible for many of the intellectual and memory impairments described and measured by conventional psychological tests.

The experiments performed in Chapters 10 and 11 appear to support this premise. Certainly, attention variables appear to influence behavioural learning more than a conventional measure of intelligence. This argument was later developed in Chapter 11 examining the difficulties such patients have in dividing their attention, causing a slowing of the information processing system and an increased tendency to make errors, mainly because of the **conscious** effort that is required for many, (once automatic) activities.

Studies that have been made by Van Zomeren and others fail to find evidence for a deficit of selective or focused attention following closed head injury. Van

Zomeren et al (1984) acknowledges that this matter is not yet settled because not all grades of severity have been tested. The results of these studies on more severely brain injured patients did find evidence of selective attentional deficits, even in the "most obvious" test situations, such as dichotic listening. Attention disorders of this severity are almost certainly responsible for the difficulties such patients have with regard to behavioural learning and are possibly related to apparent disorders of insight.

13.4 Personality and Mechanisms of Dissociation.

A major problem discussed and examined in this thesis was the reduction in motivation, not as a consequence of a drive disorder but as a disorder of normal hedonic responsiveness. This was presumed to occur directly or indirectly as a result of brain injury. Although there is a substantial literature describing hysterical reactions following brain injury, one must not lose sight of the possibility that the process of behavioural management, which confronts the individual with their behaviour, is likely to **precipitate** an avoidance reaction which may be a conscious decision not to cooperate, producing behaviour similar to that traditionally described as a hysterical personality disorder.

The question, to what extent the patient is **aware** of his behaviour in dissociative states, involves concepts such as **alteration of consciousness.** Slater and Sarjent

(1941) state that **concussion** is a frequent starting point for alterations of consciousness. In the case of dissociation, there appears to be a blocking of consciousness leading to a lack of awareness of the behaviour itself. Dissociative mechanisms are able, quite selectively, to block awareness of restricted sets of information. At one level for example, the instance of paralysis and loss of sensation from an entire limb makes it possible to interpret the clinical state in terms of a disturbance of selective attention (Eames and Wood 1984). Attempts are being made to account for this on the basis of central sensory gating mechanisms (Ludwig 1972).

Silverman (1968) offered an alternative paradigm for the study of altered states based on observations of different attentional styles in normal and psychiatric populations. Silverman identifies three major attentional styles describes as, intensive, extensive, and selectiveness.

Intensive refers primarily to responses which individuals make on a stimulus continuum which range from very weak to very strong. **Extensiveness** refers to the degree to which elements in a stimulus field are sampled. **Selectivity** of attention or 'field articulation' refers to responses which determine which elements in a stimulus field exert a dominant influence on the perceiver.

Silverman describes a paradox in sensory functioning,

most evident in individuals with altered states of consciousness, which may explain certain dissociative characteristics. On the one hand, they have lowered sensory thresholds and over-react to subliminal or marginal stimulation; on the other hand they are under-responsive to high intensity stimulation and therefore have higher pain tolerance. Pavlov (1957) explained such a paradox using the concept of 'protective inhibition'. This means that individuals who are hypersensitive to low intensity sensory stimulation, respond more strongly to it and therefore require a compensatory adjustment (under responsiveness) to protect themselves from strong stimulation.

The idea that **scanning** of information is different in ordinary states from altered states of consciousness is not proven. Individuals with altered consciousness may **appear** pre-occupied with a very narrow circle of ideas and therefore limited in the range of environmental stimuli to which they respond. Evidence from studies on normals and schizophrenics using measures of size constancy, (Gardner and Long 1962; Silverman 1964) showing the latter to consistently underestimate, are not directly transferable to a brain injured group. The differences in size or other 'constancies' (when they occurred) depended on the number of cues available when making the judgement. It might be reasonable to expect the same under-estimation from the brain injured because

of the evidence suggesting their poor use of environmental cues. This may reduce motivation because of the patients inability to perceive the true value of the reward.

14.4 Learned helplessness and dissociative behaviour

Silverman regards conditions of extensiveness and selectiveness to result in people thinking in very subjective and egocentric ways. They are concerned with ways in which the individual is prepared to **see** the world. This suggests that there may be certain parallels between the perceptions and behaviour of hysterics, following a major emotional crisis (such as would be experienced in severe head injury) and the theory of learned helplessness which results in depression and passivity (Seligman 1975).

Seligman presents a theory of helplessness which claims that exposure to uncontrollable events (in this case brain injury with major physical and mental sequelae) leads to a state of passive acceptance or withdrawal. In such conditions, animals learn that making a response to change their circumstances is unrewarding. He uses laboratory data from animal studies to show that **uncontrollability reduces motivation**, decreasing responses during instrumental learning, undermining the ability to **perceive** success, which finally **heightens emotionality.**

The effect of non-contingent electric shock or

unavoidable aversive conditioning and its relationship to what was described as 'experimental neurosis' has been known for some time (see Stroebel 1969 for a review). It does not offer a complete explanation of dissociative behaviour because it does not account for the fact that 'hysterics', such as those observed in our rehabilitation programme, are not entirely passive in their response. Rather ,they appear to work quite hard at retaining the status quo.

When faced with a distressing situation over which they **perceive** they have no control, some individuals develop a resigned, passive acceptance of their situation. This 'blocks' their ability to recognise response alternatives, focusing their efforts on maintaining their curr**ent** state, probably as a compensation strategy because they perceive more reward in being a total invalid than being a functioning 'cripple'.

In conclusion therefore, the incentive to initiate voluntary responses (make an effort!) in rehabilitation may be impaired by alterations in hedonic responsiveness on one hand and on the other, the experience, during early recovery, that effort of any kind is not going to result in significant or rapid progress towards a 'cure'. This results in a feeling of **loss of control** over the situation. The patient feels that outcome is independent of effort, diminishing the probability for any kind of

constructive response.

Maier and Testa (1974) showed that helplessness in itself, was not sufficient to produce a state of helplessness in rats. More important was whether a rat could **see** the relationship between response and outcome. If such perceptions were possible then the animals learned the response without difficulty. If however, they were unable to see response-outcome contingencies, learning ability was reduced, making **any** response less likely. Their conclusion was that a **cognitive deficit** of some kind increases the potential for learned helplessness in the rat (and probably for other organisms).

Experiencing a lack of control results in increased emotionality. Experimental studies show that there is a time factor involved in producing helplessness after uncontrollable shock. If animals are returned to the shuttle box more than 72 hours after first experiencing uncontrollable shock and then given a normal shock-avoidance test, they will learn normally; before 72 hours they are still likely to show helplessness. If however, the animals are subjected to **many** uncontrollable experiences over time, the duration of helplessness is significantly increased and interference with response initiation may become permanent (Seligman et al 1974).

The significance of examining this theory in some detail is that it has led to a 'cognitive' interpretation of a conditioning phenomenon (Davy 1981). This in itself is

important because there are prima facie reasons for thinking that many behaviours, developed and displayed in this group of patients, involve a cognitive element. Probably more important is the idea that the behaviours observed in learned helplessness bear a striking resemblance to some types of depression seen in man.

It would not take a great conceptual leap to apply such conditions to individuals who have experienced a major emotional crisis, of the kind that must occur upon first realising the effects of a brain injury. Loss of control over events would be one of the main realities. The feeling that whatever one attempts will produce no immediate effect must have a very discouraging influence. This can result, (for some individuals) in an increase in emotionality, causing them to withdraw from the reality of the situation. Attempts by rehabilitation therapists to make them participate in therapy only serves to focus attention on their disability, resulting in further withdrawal and increasing attempts to avoid reality.

The concept of avoidance has also been used in an explanation of hysterical behaviour by Eysenck (1981). He postulates a theory of hysteria based on his dimensional approach to personality (hysterics have a high E, high N). He explains this theory in terms of an approach-avoidance conflict.

Eysenck suggests that in such individuals, events that produce strong emotions (rehabilitation activities) also

produce high degrees of neuroticism and therefore, have aversive properties which would raise the avoidance gradient. The interesting aspect of this theory is the way it might explain much of the hysteric's bizarre behaviour. If all that was produced was avoidance, as would be the case with dysthymics (high N, high I) then the pattern of behaviour would be one of complete withdrawal (both factors working in the same direction to increase avoidance and lower approach behaviour).

This is not the observed pattern of hysterical behaviour in rehabilitation however. They are more manipulative and actively disruptive (the high E quality). The psychopath (according to Eysenck) on the other hand (high E, low N) would get as close to the goal box as possible (to use the animal paradigm) but the hysteric would find this approach behaviour inhibited by their high N factor which raises the avoidance gradient and lowers approach, leaving them in the worst possible position, caught in the **conflict** between approach and avoidance. This conflict would further increase the N factor (Stroebel 1969) and produce many of the maladaptive and bizarre behaviours seen in this group of patients.

Conclusion

It is not enough to show that a particular method of treatment can produce results. Ideally, one should be aware of the mechanisms which underlie the treatment. This allows (1) a more adaptive and innovative use of the treatment methods, (2) an opportunity to identify situations where it is unlikely to work and (3) an estimate of the probable duration (and cost) of such treatment. This thesis has provided information that (hopefully) will make it easier for the clinician or rehabilitationist to apply such treatment successfully.

A behavioural approach does not offer a treatment panacea for rehabilitation. It does however, offer a framework in which treatment can be given and its effectiveness evaluated. For the behaviourally disturbed brain injured patient it is surely better than the alternative which has been (and often still is) no treatment.

(APPENDIX A)

ANHEDONIA QUESTIONNAIRE

		TRUE	FALSE
1.	The beauty of sunsets is greatly over-rated.	T	F
2.	When watching a favourite television show I have usually found the presence of other people distracting.	T	F
3.	I have sometimes danced by myself just to feel my body move with the music.	T	F
4.	I seldom care to sing in the shower.	T	F
5.	I sometimes eat sweets.	T	F
6.	I've gone out of my way to watch children play even when I don't know them.	T	F
7.	Most jokes aren't as funny as many people pretend.	T	F
8.	Having close friends is not as important as many people say.	T	F
9.	I have usually really enjoyed talking with other people, even if I don't know them well.	T	F
10.	I could easily tell you the first 3 letters of the alphabet.	T	F
11.	I have had very little desire to try new kinds of food.	T	F
12.	Parties usually bore me.	T	F
13.	When eating a favourite food, I often try to eat slowly to make it last longer.	T	F
14.	I have never watched television.	T	F
15.	I've usually tried to meet new people whenever I get a chance.	T	F
16.	One food tastes as good as another to me.	T	F
17.	I often don't really enjoy jokes that other people seem to think are funny.	T	F
18.	The greatest satisfaction I have ever known is that of loving someone.	T	F
19.	Dancing, or the idea of it, has always seemed dull to me.	T	F
20.	I have really enjoyed laughing at jokes with groups of other people.	T	F
21.	I attach very little importance to having close friends.	T	F

		TRUE	FALSE
22.	I grow all the food that I eat.	T	F
23.	On seeing a soft, thick carpet, I have sometimes had the impulse to take off my shoes and walk barefoot on it.	T	F
24.	Writing letters to friends is more trouble than it's worth.	T	F
25.	When I have walked by a bakery, the smell of fresh bread has often made me hungry.	T	F
26.	I have had very little fun from physical activities like walking, swimming or sports.	T	F
27.	I can easily become fond of an attractive woman (man).	T	F
28.	A car ride is much more enjoyable if someone is with me.	T	F
29.	I think that flying a kite is silly.	T	F
30.	I usually find soft music boring rather than relaxing.	T	F
31.	I have never had a cut or scratch of any kind.	T	F
32.	I like to make long distance 'phone calls to friends and relatives.	T	F
33.	I have never gone into a store.	T	F
34.	The smell of dinner cooking has hardly ever aroused my appetite.	T	F
35.	When I pass by flowers, I often stop to smell them.	T	F
36.	I would just as soon play solitaire as play cards with other people.	T	F
37.	When taking a bath or shower, I have usually done it as quickly as possible just to get it over with.	T	F
38.	Sex is okay but not as much fun as most people claim it is.	T	F
39.	I have always had a number of favourite foods.	T	F
40.	When I'm extremely happy, I sometimes feel like hugging someone.	T	F
41.	Standing on a high place and looking out over the view is very exciting.	T	F
42.	I have not wanted to go out in the evening as much as other people.	T	F

		TRUE	FALSE
43.	Trying new foods is something I have always enjoyed.	T	F
44.	Going to parties isn't as much fun as many people claim.	T	F
45.	Getting together with old friends has been one of my greatest pleasures.	T	F
46.	I have never seen a live animal.	T	F
47.	I don't know why some people are so interested in music.	T	F
48.	Sex is the most intensely enjoyable thing in life.	T	F
49.	I usually want old friends to tell me everything they have been doing.	T	F
50.	Playing with children is a real chore.	T	F
51.	When my friends touch me I usually feel uncomfortable	T	F
52.	I have never combed my hair.	T	F
53.	I have seldom found any kind of sexual experience very enjoyable.	T	F
54.	I have usually tried to avoid romantic involvements.	T	F
55.	I have always enjoyed looking at photographs of friends.	T	F
56.	I have often enjoyed having long discussions with other people.	T	F
57.	It has always been very important for me to have at least one good friend.	T	F
58.	Meeting and getting to know new people has always been a chore for me.	T	F
59.	It has always made me feel good when someone I care about reaches out to touch me.	T	F
60.	I'd just as soon go to the movies alone as with a friend.	T	F
61.	I can speak eight foreign languages fluently.	T	F
62.	I have always loved having my back massaged.	T	F
63.	It has always made me uncomfortable to have someone confide in me about something personal.	T	F
64.	On a long drive, it makes no difference to me whether I'm alone or with someone else.	T	F

		TRUE	FALSE
65.	It is very unusual for me to feel lonely for someone.	T	F
66.	I greatly enjoy being physically close to members of the opposite sex.	T	F
67.	Although there are things that I enjoy doing by myself, I usually seem to have more fun when I've done things with other people.	T	F
68.	I have studied opera singing in Italy.	T	F
69.	It has often made me feel good to have people seek me out because they like being with me.	T	F
70.	There just are not many things that I have ever really enjoyed doing.	T	F
71.	I visited Easter Island last year.	T	F
72.	I have often enjoyed discussing women (men) with a good friend.	T	F
73.	I don't know why some people seem to get so much fun from telling jokes and funny stories.	T	F
74.	I have often enjoyed flirting with a woman (man).	T	F
75.	Making new friends is so difficult that it hardly seems worth making the effort.	T	F
76.	I have often felt like staring at an attractive woman (man).	T	F
77.	I have never ridden in a bus or taxi.	T	F
78.	I have usually had a really great time at parties.	T	F
79.	Being around children has usually bored me.	T	F
80.	I have never especially cared for women (men) to flirt with me.	T	F
81.	I enjoy holding hands with a woman (man).	T	F
82.	It's fun to sing with other people.	T	F
83.	I have yet to meet a woman (man) that I really feel attracted to.	T	F
84.	Many people attribute too much importance to "love" and affection.	T	F
85.	When I'm feeling a little sad, singing has often made me feel happier.	T	F
86.	I like playing with, and petting soft little kittens or puppies.	T	F

		TRUE	FALSE
87.	Going to the zoo is a lot of fun.	T	F
88.	I have never felt tired.	T	F
89.	I don't understand why people enjoy looking at stars at night.	T	F
90.	The taste of food has always been important to me.	T	F
91.	People often expect me to spend more time talking with them than I would like.	T	F
92.	When anticipating a visit with a friend, I have often felt happy and excited.	T	F
93.	Sunbathing isn't really more fun than lying down indoors.	T	F
94.	I have sometimes seen children playing.	T	F
95.	I have often found it difficult to resist talking to a good friend, even when I have other things to do.	T	F
96.	Flowers aren't as beautiful as many people claim.	T	F
97.	I have walked on a sidewalk.	T	F
98.	I enjoy the feel of silk, velvet or fur.	T	F
99.	The usual reason I have had long conversations with people is that they expect me to.	T	F
100.	The idea of going out and mixing with people at parties has always pleased me.	T	F
101.	I have usually found love-making to be intensely pleasurable.	T	F
102.	Sometimes I feel sleepy or tired.	T	F
103.	I have had a cold at least once in my life.	T	F

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A BEHAVIOURAL APPROACH TO THE REHABILITATION OF SEVERE BRAIN INJURY

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ABSTRACT

A number of studies have attempted to evaluate the outcome from severe brain injury. Originally, these concentrated on the physical sequelae of injury but more recent studies have investigated the 'psychosocial outcome' to determine the reaction of the family and the community to the effects of such injuries. Successive studies have shown that changes of behaviour, emotionality and intellect are far more important in determining the outcome of brain injury than is the presence or absence of physical handicap.

The more severe the injury, the more likely it will be that psychiatric or behaviour disorders will follow. The most immediate problem as a result of this behaviour change is the difficulties it introduces in the management of patients during rehabilitation. There are many instances when such behaviour problems will actually cause the patient to be discharged from a rehabilitation programme or be placed in a chemical 'strait jacket' in an attempt to control behaviour. Often, the end result is that the patient is transferred to a psychiatric or geriatric unit with only a remote prospect of further treatment or rehabilitation.

This study shows the effectiveness of a behaviour management programme, emphasizing methods of behavioural learning and its application to various aspects of rehabilitation following severe brain injury. Although basically a successful procedure, there are important constraints which prevent the acquisition of a behavioural response. Probably the most important and obvious constraint is that im posed by neurologically mediated behaviours (a consequence of temporal or frontal lobe injury). There in addition, important neuropsychological constraints, the main one being an impairment of information processing (attention). Finally, there is the constraint imposed by the nature of the behaviour change, sometimes resulting in a dissociative behaviour disorder with characteristics similar to those traditionally attributed to a hysterical personality. The thesis identifies, describes and discusses these constraints, offering, where possible, alternative methods of treatment.