Speed of Decision-making Following Closed Head Injury: The Effects of Speed-Accuracy Tradeoff on P300 and Reaction Time

by

Diana L. Deacon-Elliott

Thesis submitted to the School of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Doctor of Philosophy

School of Psychology
University of Ottawa
Ottawa, Ontario
February 29, 1988

ABSTRACT

It is generally acknowledged that reaction time (RT) is prolonged in the head-injured. The locus of the delay in RT is poorly understood. Recent evidence suggests, however, that the major portion of the RT delay may occur subsequent to the stimulus evaluation period. In response to a task relevant stimulus, a late positive wave, P300, is often observed. The latency of P300 has recently been used to compliment the behavioural measure, RT. P300 has been found to be primarily sensitive to stimulus evaluation time whereas RT is thought to be determined by both stimulus-evaluation and response production times. In the head-injured, P300 is delayed by 50-70 ms suggesting a moderate slowing of stimulus evaluation time. By contrast RT is usually 100-150 ms longer in these patients. The major portion of the RT delay may therefore be due to factors associated with response production. It is known that response production time is affected by the subjects' relative bias toward speed or accuracy of responding. It has also been proposed that the head-injured may respond more cautiously (perhaps stressing accuracy at a cost of speed). This thesis attempted to reduce the RTs of head-injured subjects to within their P300 peak latencies. Various manipulations which increased the subjects emphasis on speed were presented in three different studies. P300 and RT were recorded in a group of severely (mean coma duration = 28 days) head-injured outpatients (mean time since injury = 4.8 years) and a group of matched controls. In the first experiment, instructions to subjects emphasized either speed or accuracy of responding. RT was

significantly reduced in both groups by speed instructions whereas P300 latency was not affected. Notwithstanding the reduction, patient RTs remained 134 ms longer than those of controls. Furthermore, patient RTs exceeded their P300 latencies by 76 ms. These findings suggested a bias towards accuracy which was resistant to manipulation by simple experimental instructions. A second study was therefore conducted in an attempt to further constrain RT. A criterion window was provided within which subjects were required to respond. Feedback (FB) was also provided which informed subjects in some conditions of their speed of responding and in others of their accuracy. RTs were reduced by approximately 100 ms relative to the accuracy condition of the first study. The reduction of RT was greatest in a condition in which responding was confined to within a very narrow time limit and feedback informed the subject whether the response had occured within this window. In this condition RTs of both groups preceeded their P300 latencies. RT in this condition may thus have been primarily determined by stimulus evaluation time. The third study was designed to assess possible carry-over of training with the FB and criterion window. The narrow criterion window and FB on response speed were presented over a series of trials. The cues to speed of responding were then gradually removed. A significant carry-over was observed, such that RTs continued to be reduced when the cues were removed. The three experiments therefore demonstrated that the RTs of the head injured are subject to manipulation. Furthermore, training with a criterion window and FB on response speed may have an enduring effect on RT.

Acknowledgements

The completion of this thesis would not have been possible without the assistance of my supervisor Dr. Kenneth Campbell. In this and in other projects his technical, methodological, and editorial skills have been invaluable. I am grateful to have benefitted from his competance and intelligence, and recognize the diplomacy with which he has dealt with my own occasional "information processing deficits".

Although many of my colleagues have assisted me in various ways, two in particular deserve mention. Dr. Ian Bell spent hours of his time instructing me on the use of BMDP and SPSS programmes. Dr. Braxton Suffield generously provided the demographic, neurological and audiometric data on the patients tested in these studies.

Several of our support staff also made substantial contributions to this project. The equipment which timed and delivered FB in the third study was designed by Herman van den Bergen and constructed by himself and Robert Spratt. Madan Makasare wrote the software which was used to deliver the auditory stimuli and average the behavioural and physiological data. Diane Brazeau typed the manuscript with efficiency and patience.

Needless to say, there would be no research without subjects. I would like to express my appreciation to those who volunteered their time in order that these experiments could be made possible.

Finally, thanks to Michel Girodo for his consideration and tolerance during the final stages of this project.

TABLE OF CONTENTS

Chapter

PREFACE

The thesis will be presented in five chapters. The relevant research literature is reviewed in Chapter I. Chapters II-IV will consist of a series of three different experiments. The tables and figures which will be referred to are included at the end of each Experiment. A general discussion of the experiments will be provided in Chapter V. References appear at the end of the thesis.

CHAPTER I

Introduction

Epidemiology of Closed Head Injury

The incidence of closed head injury (CHI) is difficult to establish due to the variety of sources from which statistics are derived. A number of official surveys have been recently conducted in the United States. In 1975, the U.S. National Institute of Neurological and Communication Disorders and Stroke (NINCDS) used a health interview questionnaire to sample a population of 116,000 non-institutionalized American residents. Their results (reported by Kraus, 1980a) indicate a rate of six head injuries per 1000 person or approximately 1,275,000 cases per year. Included in this estimate were cases of skull and facial bone fracture which may or may not have involved CNS trauma. The NINCDS has also published the findings of the National Head and Spinal Cord Injury survey (Kalsbeek, McLaurin, Harris & Miller, 1980). Data for the survey were gathered from U.S. hospital admission records for the years 1970 through 1974. These authors report a rate of 204 new head injuries per 100,000 persons in 1974 alone. The number of persons having sustained CHIs during the 1970-1974 period was estimated to be 439 per 100,000 persons.

Several other surveys have reported on statistics obtained from hospital records. The United States Hospital Discharge Survey compiled a nation wide inventory of CHI patients discharged from civilian hospitals in 1975. The estimated rate of CHIs discharged from hospital in this year was 170 per 100,000 persons. The survey did not include patients dis-

charged from veterans or military hospitals. Patients who died as a result of their injuries were included in the study. Those treated without being admitted were not included, nor were those still receiving outpatient care (Anderson, Kalsbeek, & Hartwell, 1980).

Field (1976) has published statistics on the occurrence of CHI in England and Wales for 1972. His report was based on a combination of hospital admission records and data from the National Study of General Practice for 1970-1971. Field estimated an occurrence rate of 4.3 cases per 1000 persons. Since both private practice and hospital records were used, patients who received both services would have been counted twice.

In general, the surveys based on hospital admission records tend to underestimate the incidence of CHI. Excluded from these figures are patients who are treated without being admitted and persons who died before reaching hospital. Hawthorne (1978) surveyed Scottish hospitals over a two week period. He found that only one of every six CHI victims who attended emergency rooms were admitted for treatment. Field (1976) estimates that persons who die before being admitted account for up to 60% of CHI fatalities.

In Canada a now dated report indicates there are approximately 4400 deaths due to CHI each year (Hay, 1967). Various figures are available for different regions of the United States. In 1978 these ranged from 20 to 25 fatalities per 100,000 (Jennett & Bond, 1975; Rosenthal, 1983). This rate is more than twice that reported in Great Britain (Jennett & Teasdale, 1981). In one American study (Rimel & Jane, 1983), fatalities accounted for only seven percent of those CHI patients admitted to a

Virginia hospital. Four percent of their sample remained in a vegetative state, 8% were severely disabled, 12% were moderately disabled and 69% showed good recovery.

American statistics indicate that 70% of coma inducing CHIs result from motor vehicle accidents (MVAs) (Miller, Sweet, Narayan, & Becker, 1978). This incidence has been found to be as high as 82% amongst 15-29 year olds (Marshall et al., 1983). One Canadian study (Parkinson, Stephenson, & Phillips, 1985), showed that at least one half of all CHIs occur as a result of MVA. World health statistics for 1975 indicate that in Canada the annual rate of road deaths was 30 per 100,000. Canada and West Germany had the highest rates of road deaths of any nations surveyed (Jennett & Teasdale, 1981). Since this time legislation has been introduced in many countries regulating lower speed limits and the compulsory use of seat belts. In Australia, the introduction of seatbelt legislation has decreased the number of serious MVA related head injuries by 8% (Trinka, 1986).

The most recent Canadian statistics (Dalkie & Mulligan, 1986) indicate a 3% decrease in the number of MVA related hospital admissions, despite a 7% rise in the number of MVAs reported to police. This same report indicates that the annual number of MVA fatalities has decreased by 12% between 1980 and 1985. Given that head injuries constitute a large proportion of MVA fatalities the effect of legislation on reducing the number of CHIs has been substantial.

Patient Characteristics

The range and average ages of CHI victims vary among sources. Most authors agree, however, that the incidence of CHI peaks in adolescence and young adulthood. The steepest rise occurs between the ages of 15 and 24 (Fields, 1976; Kraus, 1980a). In this age range, MVAs predominate as the cause of injury. Injury as a result of falls is more common among children and the elderly (Rimmel & Jane, 1982). In young adults, male CHI victims outnumber females by a ratio of 4:1 (Fields, 1976; Kerr, Kay, & Lassman, 1971; Kraus, 1980b).

The socioeconomic status of CHI patients has been investigated in a number of British studies. Steadman and Graham (1970) found no relationship between socioeconomic status and the incidence of CHI. Kerr (1971) however found CHIs to be more frequent among low socioeconomic classes. In their sample, and that studied by Rimel and Jane (1982), lower socioeconomic status was also associated with a higher incidence of CHI due to assault. In higher socioeconomic classes, injury due to sports was more prevalent than amongst the lower classes. In the sample studied by Rimel and Jane, traffic accidents were not related to socioeconomic status.

The marital status of CHI victims also appears to differ from what would be expected from the population at large. Rimel and Jane note that 59% of this CHI group were single at the time of injury, which is 20% higher than which would be expected from healthy adults in the same age range. In the remainder of their sample, 29% were married and the others were widowed or divorced. Kerr et al. (1971) found the divorce rate in CHI victims to be four times that of the rest of the American population.

In a study of emergency room attenders at Vancouver General Hospital, Klonoff (1971) found an unusually high incidence of CHI in children from lower socioeconomic backgrounds in which marital discord was present.

Other factors that may place persons at risk of incurring a head injury are psychiatric disturbance and drug or alcohol abuse. Most studies report having observed relatively small numbers of CHI patients with previous psychiatric disorders. As Levin, Banton, and Grossmand (1982) note, however, few studies have compared these figures with the general incidence of these disorders. In their own studies, Levin et al. found that at least 20% of CHI patients screened for research purposes had histories of psychiatric disorder and/or substance abuse. Field (1976) cites evidence that chronic alcoholics may account for between one-quarter to one-half of all severe head injuries. Consumption of alcohol prior to injury is also frequently reported (Field, 1976; Williams, Peat, Crouch, Wells, & Finkle, 1985). According to Field (1976), the ingestion of alcohol is reported in approximately 29% of male and in 10% of female CHI victims who are 15 years of age or older.

Pathophysiology of Closed Head Injury

Head injuries may be loosely classified into two diagnostic categories; missile and non-missile or closed head injury (Levin et al., 1982). Lesions sustained as a result of missile injury are largely determined by the size, velocity and trajectory of the foreign body as it passes through the brain (Levin et al., 1982). In the case of gunshot wounds, the

implosion of skull fragments often produces additional cortical lacerations (Russel, 1947).

Depressed skull fractures may also be present in "closed" head injuries (Levin et al., 1982). The hallmark of closed head injuries, however, is damage sustained as a result of the acceleration and deceleration of the brain relative to the skull (Teasdale & Mendelow, 1984). These forces cause mechanical deformation of the brain, producing focal, as well as diffuse microscopic lesions. Brain injury which occurs at the time of impact is referred to as "primary" damage. It is irreversible by treatment (Teasdale & Mendelow, 1984). During recovery most CHI victims risk developing complications which are secondary to the initial insult on the brain. Primary and secondary brain injury will be considered separately.

Primary damage

Focal lesions. Cerebral contusions have been radiologically determined in about 40% of CHI victims (Dublin, French, & Renwich, 1977). When skull fractures are present sub-coup lesions are found directly under the site of impact (Ommaya, Grubb, & Naumann, 1971). The size of lesion is usually related to the surface area of the object which collides with the head (Lindenberg, 1971). Gurdjian and Gurdjian (1976) note, however, that collision with a large, flat surface does not appear to cause focal injury at the site of impact, although contrecoup lesions may result. Contrecoup lesions are those which appear at distant sites, antipodal to the area of

impact (Gurdjian & Gurdjian, 1976). They are thought to be incurred as a result of inertial forces causing the brain to move within the skull.

Movement of the brain may be linear or rotational (Holbourn, 1943; Pudenz & Shelden, 1946). In either case, it is those areas of the brain which are immediately adjacent to the bony irregularities of the skull that are most often damaged (Adams, Graham, Murray, & Scott, 1982; Alexander, 1982; Ommaya et al., 1971). For this reason, the frontal-orbital and temporal-polar regions are particularly at risk. It has been suggested that these areas stand the greatest chance of injury, regardless of the site of impact (Ommaya et al., 1971). Conversely, contusions of the occipital lobe are rarely seen.

Diffuse axonal damage. Using elaborate wax and gelatin models, Holbourne (1943) has demonstrated that rotational movement of the brain is necessary to produce diffuse damage. He proposed that diffuse damage would proceed from the outermost extremities of the brain and move progressively inward. This model would therefore predict that microscopic damage would be confined to the cortex in the case of milder injuries. With more severe rotational movement of the brain, the diencephalon and brainstem would ultimately be affected. This contention has since received support from animal studies (Ommaya & Gennarelli, 1974; Pudenz & Sheldon, 1946), and postmortem autopsies of fatal CHI (Adams et al., 1982). Electrophysiological observations of CHI patients have also been consistent with this view (Greenberg et al., 1980).

Strich (1956) was the first to histologically verify diffuse microscopic lesions in CHI victims. Her earliest investigations were performed

on the brains of patients who had survived for several months after having sustained severe injuries. Cases were selected which were uncomplicated by hematoma, herniation, or contusions. Post-mortem examination revealed Wallerian degeneration, primarily of large, myelinated axons, concentrated in the periventricular region, the corpus callosum, and the cerebral peduncles.

In these same areas, "retraction balls" become apparent within a few hours after injury (Levin et al., 1982). These are extrusions of axoplasm which escape from the damaged cell. Retraction balls which form at the distal end of axons are reabsorbed within weeks, and the degenerated portion of the axon replaced by microglial scar tissue. Those which form proximally to the cell body may remain for several months (Teasdale & Mendelow, 1984).

The extent of diffuse damage sustained may be difficult to determine soon after injury (Filley, Cranberg, Alexander, & Hart, 1987). During recovery however, diffuse damage may be indicated radiologically by a proportional reduction in cerebral white matter. Within two weeks of injury thinning of the corpus collosum and ventricular enlargement may be apparent. In the latter condition, known as "hydrocephalus exvacuo", an increase in the size of the ventricular cavities is observed as the degeneration of surrounding white matter becomes complete (Adams et al., 1977; Strich, 1970).

Secondary damage. Most secondary damage occurring after CHI may be traced to either ischemia (the disruption of the brain's supply of glucose or oxygen) or factors which raise intra-cranial pressure (ICP). The

occlusion of, or damage to cerebral blood vessels, will produce ischemic lesions if the brain is deprived of glucose and oxygen for any length of time. The perfusion rate of blood may also be altered by raised ICP, however, despite adequate circulation.

Graham and Adams (1971) report that ischemic necrosis is present in approximately 50% of fatal CHIs. Ischemic lesions were most often observed in the hippocampus (81% of cases) and the basal ganglia (79% of cases). Forty-six percent of larger ischemic lesions were found in the cortex.

Ischemia is often closely associated with edema. Edema, or swelling of the brain, can produce ischemic lesions as a result of compression of the cerebral vasculature (Teasdale & Mendelow, 1984). The reverse is also true; ischemia may precipitate edema. Several types of edema have been identified in which fluid accumulates either (1) extracellularly, (2) intracellularly (in areas of the brain which have been damaged) or, (3) in the tissue surrounding the ventricles. Hypoxia, (the depletion of oxygen) as well as hypercapnia, (the presence of excessive carbon dioxide) lead to increases in cerebral blood volume. If the walls of blood vessels are damaged, fluid accumulates in the extracellular space where large molecules are allowed to pass through the blood-brain barrier. Fluid may accumulate intracellularly as a result of ischemic loss of energy supply. An accumulation of CSF around the ventricles (interstitial edema) is usually associated with obstructive hydrocephalus (Teasdale & Mandelow, 1984).

Brain edema, hydrocephalus, and intracranial hematoma contribute to the elevation of ICP. Elevated ICP develops in about 75% of CHIs (Miller, Becker, Ward, Sullivan, Adams, & Rosner, 1977), and is associated with poor prognosis. Raised ICP and elevated cerebral blood flow have recently been found to correlate negatively with neuropsychological outcome 96 hours after injury (Uzell, Obrist, Dolinskas, & Langfitt, 1986). Furthermore, in one study (Narayan, Greenberg, Miller, & Enas, 1981), a 51% mortality rate was observed amongst patients with elevated ICP, as compared to a 16% mortality rate in other cases. It has been suggested that diffuse ischemic lesions caused by raised ICP account for 50% of fatalities after CHI (Bakay & Glasauer, 1980). The expansion of mass lesions or vascular occlusion often cause localized elevations of IPC. Pressure differences then develop between cerebral compartments, causing their contents to shift and herniate (Levin et al., 1982). In these cases the parahippocampal gyrus and uncus are frequently displaced downward, through the tentorial hyatus. Localized elevations of ICP also commonly cause compression of the brainstem and midbrain resulting in herniation of the midbrain against the tentorium. In addition, portions of the cerebellum are often forced through the foramen magnum (Jennett & Teadale, 198 ; Levin et al., 1982).

Fortunately, the secondary effects of CHI may be arrested and often reversed by medical intervention (Teasdale, & Mendelow, 1984). It is perhaps due to recent advances in emergency and neurosurgical monitoring that a greater percentage of CHI victims now survive.

Summary

The primary mechanisms which produce CHI are inertial forces which cause the brain to move relative to the skull. These typically produce focal, as well as diffuse, microscopic lesions. Focal lesions are usually concentrated frontotemporally and orbitofrontally due to the irregularity of the skull in these areas. Microscopic lesions are found mostly in large myelinated axons, and therefore in areas with high concentrations of these.

Secondary brain injury most often develops a consequence of ischemia or raised ICP, due to edema, hematoma, or obstructive hydrocephalus. When the elevation of ICP is localized herniation of the brain may result. Most secondary effects of CHI may be arrested by medical intervention.

Reaction Time

Donders (1868) was the first to isolate individual "stages" of response processing. His work is best known for three prototypic reaction time (RT) experiments. In the most basic of these, a single stimulus was presented which required a single invariant response. This "simple RT" task was used as a measure of a "movement time" and was assumed not to involve "stimulus discrimination" or "evaluation" (distinguishing between stimuli within the context of the task) or "response choice" or "production" (choosing from amongst the response alternatives presented in a task and executing the appropriate response) processes or stages. Stimulus evaluation processes were invoked by a second task. In this task, subjects responded to only one of two stimuli that were presented. A

third task required that subjects respond differentially to two stimuli, thus presumably engaging response choice and production processes.

Donders proposed that independent estimates could be obtained by subtracting RTs recorded during the different tasks of the different stages of processing. He assumed a "serial" model of processing in which stages were linearly dependent upon each other (i.e. the completion of one stage necessarily preceded the activation of the next). The stages of response processing which were postulated by Donders are still recognized by most contemporary authors, albeit in some cases, by different names. In more recent approaches, some authors have recognized additional stages of stimulus "encoding" or "input" (Saunders, 1977; Sternberg, 1969; Theios, 1973), "response organization" or "motor adjustment", (Saunders, 1974; Sternberg, 1969; Theios, 1973), "response initiation" (Miller, 1983) and "response output" or "execution" (Theios, 1973). Serial models have formed the basis of many theories of information processing. It is now apparent, however that they have difficulty in accounting for all instances of decision-making.

Since Donders pioneering work, RT has been measured under a virtually exhaustive variety of conditions. Some proponents of serial models, (Sternberg for example), hold that the independence of stages may be demonstrated through factor analytic techniques. Sternberg (1969) suggested that the orthogonality of the factor structure of RTs indicate independent stages of processing. More recently, however, many of the variables affecting RT have been found to interact with each other. The results of recent physiological studies (Coles, Gratton, Bashore, Eriksen,

& Donchin, 1985) indicate furthermore that some stages may begin before the completion of the preceding stages. "Parallel" models, which allow for overlap between stages have been proposed, to better account for these findings (i.e. Eriksen & Schultz, 1979; Grice, Nullmeyer, & Spiker, 1982; McClelland, 1979; Miller, 1982; Ratcliff, 1978). The most salient criticism of the parallel models is perhaps that they have not been rigourously tested. This is, in a large part, owing to the fact that it is extremely difficult to design experiments to test the very complex assumptions raised by parallel models of information processing. It is also the case that behavioural measures such as RT infer individual stages of processing retrospectively after the completion of the response. This imposes a great disadvantage in the testing of parallel models where it may be necessary to measure each overlapping stage directly.

A further limitation of RT as a measure is imposed by the interpretation of mean or median RTs. Global measures such as these do not adequately represent the subject's full range of RTs. Some authors have thus examined how fast a subject is capable of responding while still responding accurately, or conversely, at what point in the RT distribution errors occur. Measures of central tendency obscure this information. A number of authors have attempted to isolate specific stages based on patterns or trends in the single RTs (Brewer & Smith, 1984; Link, 1980; Luce, 1986; Pachella, 1974; Rabbit, 1979). The elaborate mathematical analysis performed by these authors are often based on controversial assumptions making interpretation of such labourious analyses quite difficult. To meet the mathematical assumptions on which these sophisticated analyses

are based often require several hundred (if not thousands) of trial presentations. They are therefore hardly practical for use with certain clinical populations (such as CHI patients) who tire easily.

An alternative favored by some researchers is to combine the use of RT and physiological measures. Physiological measures allow the experimenter direct access to mental events occurring prior to, during and after the completed response (Donchin, 1979; Campbell, 1985; Miller, 1986). Components of the evoked potential (EP) are particularly useful for these purposes. Several EP components, which will be discussed in a later section, have been observed to be sensitive to different stages of information processing. Before discussing these, a brief review will be provided of some of the variables which have been observed to influence RT.

Stimulus variables. The sensory modality of stimuli has been found to affect RT (Brebner & Welford, 1980). Visual stimuli, for example have been found to require 10-30 ms longer to be decoded than auditory stimuli (Kemp, Coppée, & Robinson, 1937; Marshall, Talbot, & Ades, 1943) perhaps because of the extra processing time required for the stimulus to reach the sensory cortex. Within both the visual and auditory modalities stimulus input time has generally been found to be inversely related to its intensity (Hovland, 1937; Mansfield, 1973; Pieron, 1920). Hovland (1936) however has demonstrated that absolute intensity is less important in this regard than is the intensity of a stimulus relative to background "noise" or the intensity of stimuli to which the subject was previously exposed.

Stimulus duration has also been found to be inversely related to RT in both the auditory (Raab, 1962; Wells, 1913) and visual (Mansfield, 1973; Turvey, 1973) modalities. Stimulus duration may influence RT by controlling the amount of time over which sensory information may accumulate (Brebner & Welford, 1980). Similarly, increasing the area of stimuli has been found to decrease RT in the visual (Froeberg, 1907, Ferree & Rand, 1927) and gustatory modalities (Elsberg & Spotnitz, 1938). The complexity of visual stimuli has been found to affect RT. More complex stimuli have been suggested to prolong RTs as a result of the greater number of features which must be encoded. It has also been suggested, however, that stimulus identification time may be longer for these stimuli due to the variety of labels which may be attached to them (Theios, 1975).

Discriminability may be manipulated either by eroding the stimulus (Chase & Posner, 1965; Sternberg, 1964) or by decreasing the difference between stimuli (Crossmann, 1955; Hennon, 1906; Kellog, 1931; Johnson, 1939; Pike, 1968). In either case, RT is prolonged as discriminability decreases. Stimulus ensemble size does not appear to affect identifications time (Alluisi, Strain & Thurmond, 1964; Brainard, Irby, Fitts, & Alluisi, 1962; Morin & Forrin, 1962, 1965; Theios, 1973).

Stimulus probability has been repeatedly demonstrated to exert an influence on RT (Hick, 1952; Hyman, 1953; Kaufman, Lamb, & Walter, 1970; Krinchick, 1969; Lamb & Kaufman, 1965). In general, more frequent stimuli are associated with shorter RTs. The locus of the effect of stimulus probability on RT is not well understood. Some authors favour the theory that stimulus probabilities establish a perceptual bias or "expectancy"

such that subjects are more prepared to perceive frequently occurring stimuli (Bertelson, 1966; Bertelson & Tisseyre, 1966; Besner, 1977; Klatzky & Smith, 1972; Miller & Pachella, 1973). The subjects' preparedness is therefore thought by these authors to facilitate sensory processing of the stimulus. Other investigators have proposed that the effect of stimulus probability on RT may be due to the facilitation of response production rather than the perception of stimuli (Biederman & Stacy, 1974; Dillon, 1966; Hawkins, MacKay, Holley, Fiedin, & Cohen, 1973; Morin & Forrin, 1963; Rabbit, 1959; Sanders, 1970; Theios, 1975). According to this view, the degree to which a response is invoked will be influenced by the probability of the associated stimulus being presented.

Duncan-Johnson and Donchin (1981) recorded RT and evoked potentials during different conditions of stimulus (and response) probability. The latency of P300, a waveform used as a measure of stimulus evaluation time, was found to vary significantly as a function of stimulus probability. The degree of variability produced in P300 was much less than that observed in RT. They concluded therefore, that a small portion of the variation in RT was due to manipulation of stimulus evaluation time. A much larger portion of the variation in RT appeared to be associated with response production.

Expectancy is also dependent upon the interstimulus interval (ISI) or in some tasks the response-stimulus interval. Kornblum (1969) notes that RT is determined by both the duration of ISI on a given trial and the duration of ISIs on preceding trials. RT tends to increase as ISI increases and is shorter for fixed, rather than variable ISIs.

Response variables. In some tasks, stimuli and the responses corresponding to them are paired on the basis of some salient feature such as a spatial position, shape or colour which each have in common. In this case, stimulus and response are said to be "compatible". When responses are designated arbitrarily, or in such a way as to hinder the association between stimulus and response, the stimulus response (S-R) relationship is "incompatible". RT is longer when S-R compatibility is low and shorter when it is high (Alluisi, Strain, & Thurmond, 1964; Fitts & Sierger, 1953; Fitts & Deninger, 1954).

The number of response alternatives has been manipulated in many studies, with somewhat inconsistent results. RT has often been found to be prolonged as the number of alternatives increases (Brainard, Irby, Fitts, & Alluisi, 1962; Hick, 1952; Hyman, 1953; Theios, 1973). In other experiments, however, similar manipulations produced no significant variation in RT (Davis, Moray, & Treisman, 1961; Fitts & Switzer, 1962; Morin, Konick, Troxell, & McPherson, 1965). Kornblum (1965) notes that S-R compatibility may have interacted with the number of alternatives in the Davis et al. and Fitts and Switzer studies. Several authors have observed that the number of possible responses has a much greater effect on RT when S-R compatibility is low (Broadbent, 1971; Fitts & Posner, 1967; Kornblum, 1969; Leonard, 1961). Kornblum (1969) has also observed stimulus sequencing to interact with these factors. Stimulus repetitions were less influenced than non-repetitions by the combined effects of S-R compatibility and the number of response choices.

The effect of practice on RT may also interact with S-R compatibility (Smith, 1968). Practice on a task with high S-R compatibility usually decreases overall RT, and minimizes the effect of the number of response choices (Davis et al., 1961; Mowbray & Rhoades, 1959). Under conditions of low S-R compatibility, however, practice may have virtually no effect on RT (Nickerson, 1969).

Speed of responding will depend, in a large part, on the subjects criterion for responding. Subjects who are very cautious will respond more slowly than those who are not. These subjects may effectively trade speed for accuracy, in order to meet a certain criterion level of performance. Experimentally, speed-accuracy traded-off may be manipulated by varying instructions to subjects. When subjects are instructed to emphasize response speed, RT may decrease, but the number of errors will generally increase. Accuracy is thus "trade-off" for speed. By contrast, under accuracy instruction a decrease in the number of errors is traded-off by longer RTs.

It is apparent that choice RT can be altered by many different experimental manipulations. There is less agreement among authors, however, as to the stage or stages through which the effects of each of these variables are mediated. In studying clinical populations it may be of greater interest to determine why RT is delayed as opposed to simply the amount by which it is delayed. Evoked potentials are sensitive to mental events not easily accessed by RT alone and, therefore may provide insight into the locus of RT differences in clinical populations. A late

positive waveform, P300, has now been widely adopted to complement RT as a measure of mental chronometry. A review of this component will follow.

Summary. Most authors recognize stages of information processing which are the same as, or variations of those proposed by Donders (1868). There is considerable debate however, as to the chronological relationship among stages. Many variables have been identified which affect RT. Furthermore, there appears to be a good deal of interaction among the hypothetical stages.

The disadvantage of RT as a measure is that it cannot easily be used to observe covert processes. Several authors claim to have circumvented much of this difficulty through the use of single trial RT analyses. The methods employed in these studies are nevertheless, extremely labourious with highly controversial assumptions. Even if these limitations were removed, the large number of trials required for the elaborate mathematical computations are impractical for use with clinical populations. The combination of RT and evoked potentials has been suggested as an alternative measure.

The P300

A late positive wave, the P300 was initially reported by Sutton (1965). It is recorded maximally in parieto-central regions of the scalp, in response to rare, task relevant stimuli. Since its discovery, attempts have been made to relate the variability in P300 amplitude and latency to different psychological phenomena (see Johnson, 1986; Pritchard, 1981).

P300 Amplitude

Numerous studies have demonstrated that P300 amplitude is inversely related to the global probability of occurrence of the eliciting stimulus (Campbell, Courchesne, Picton, & Squires, 1979; Duncan-Johnson & Donchin, 1977; Friedman, Simsom, Ritter, & Rapin, 1975; Johnson & Donchin, 1980; Kutas, McCarthy, & Donchin, 1977; Tueting, Sutton, & Zubin, 1970). In addition to variability produced by changes in the global probability of stimuli, P300 amplitude is also sensitive to the sequential probabilities of stimuli. Stimuli which are repeated in a sequence elicit smaller P300s than non-repeated stimuli, even though the global probability of the two stimuli might be equal (Squires et al., 1976). These relationships have been consistently observed regardless of the type of task or stimuli employed. Even the unexpected omission of a stimulus presented in a regular train will also elicit a large P300 (Picton & Hillyard, 1974; Ruchkin & Sutton, 1978; Sutton, Tueting, Zubin, & John, 1967; Weinberg, Walter, & Crow, 1970).

Donchin and his colleagues have interpreted the P300 literature in terms of models of "working memory" (Donchin, 1975; Donchin et al., 1978; Duncan-Johnson & Donchin, 1982). Individuals form expectancies about their environments on the basis of past events. These events are stored in a working memory system which is continually updated as hypotheses about the environment are disconfirmed. They suggest that the P300 may reflect the "contextual updating" processes which are invoked by unexpected events. The nervous system

> "continuously generates hypotheses about the environment, which are then validated against the input information. The hypothe-

> sis ... might be 'strongly held' (that is, the organism has a clear expectation as to what should happen)... The disconfirmation of such hypotheses does yield a P300. Alternately, if the hypotheses ... are weak, in the sense that several alternate hypotheses might be entertained with realtively equal probability, then the confirmation of either of these hypotheses ... will yield a P300" (Donchin, 1975, p. 213).

It has been suggested (Johnson, 1986) that "stimulus meaningfulness" may be the most salient aspect of many variables influencing P300 amplitude.

Johnson and Donchin (1978) and Campbell et al. (1979), have observed that P300 amplitude varies proportionnally to the amount of information which is actually extracted by the subject. In turn, the amount of information extracted from a stimulus may be dependent upon the level of attention paid to the stimulus (Johnson, 1986). If however subjects are required to perform a secondary task and thus "actively" ignore irrelevant stimuli, the ignored stimuli may fail to elicit P300s (Duncan-Johnson, & Donchin, 1977). Under these conditions P300s may be evoked only when the stimulus is so intrusive that it demands attention (Ritter, Vaughn, & Costa, 1968; Squires, Donchin, Squires & Grossberg, 1977).

The amount of information transmitted by stimulus may also be modulated by "equivocation". The term "equivocation" describes a loss in the amount of information transmitted due to the subjects uncertainty as to what they have perceived (Ruchkin & Sutton, 1978; Sutton, 1979). Signal detection experiments have found P300 amplitude to be larger when subjects' accuracy and certainty are high (Hillyard, Squires, Bauer, & Lindsay, 1971; Squires, Hillyard, & Lindsay, 1973). The opposite has been found when the degree of stimulus discrimination is manipulated. Stimuli

which are less discriminable generally elicit small P300s (Ford, Roth, & Kopell, 1976; Johnson & Donchin, 1978).

Posner (1978) proposed that P300 reflects the activation of a system responsible for "limited capacity conscious processing". This possibility has been tested in a number of dual task experiments (Isreal, Chesney, Wickens, & Donchin, 1980; Isreal, Wickens, Chesney, & Donchin, 1980; Towle, Heuer, & Donchin, 1980). The introduction of a second task was found to decrease P300 amplitude, suggesting that P300 may index the availability of processing resources. In the Isreal, Chesney et al. study P300 was smaller when tones were counted while subjects performed a visual tracking task. No further decrement in P300 was observed, however when the task was modified to include the manual tracking of the stimulus. This has led some authors to speculate that the limited capacity system described by Posner may be limited only with regard to perceptual processing resources (Isreal, Chesney et al, 1980a; Pritchard, 1981). Further experimentation is needed in order to verify this hypothesis.

P300 latency

Rohrbaugh, Donchin and Eriksen (1974) suggest that the diversity of factors affecting P300 amplitude may indicate that it is actually a complex of waves. Squires, Squires, and Hillyard (1975) have in fact recorded two apparently distinct varieties of P300s. An early (220-280 ms) frontocentral waveform "(P3a)" was observed to be evoked by very rare, novel stimuli, regardless of the subjects' level of attention. A later (310-380 ms), parietocentral waveform "(P3b)" was elicited by task

relevant, rare stimuli only when the subject actively detected them. Friedman, Vaughan, and Erlenmeyer-Kimling (1978) have provided evidence that the P3a may be further decomposed into two waveforms. The earliest of these (198 ms) had a more frontal distribution than the later (286 ms) frontocentral component. A fourth variety of P300 is virtually identical in latency to the centro-parietal oddball P300 (310-320 ms). It is elicited however by stimuli which provide feedback to subjects regarding their performance on a task. The feedback P300 is recorded more anteriorly than the oddball P300 (Campbell et al., 1979; Deacon-Elliott, Campbell, & Proulx, 1987; Johnson & Duncan, 1978). An early (240-320 ms) "P300e" has been identified which may be emitted when the absence of a stimulus provides feedback (Ruchkin, Sutton, Munson, Silver, & Macar, 1981).

In the past decade, the latency of the late task relevant P300 (P3b) has engendered considerable interest as an index of the timing of decision-making processes. Early investigations suggested that P300 reflected the termination of a preparatory set (Rohrbaugh et al., 1974; Desmedt, 1981; Desmedt, & Debecker, 1979) or possibly the termination of the stimulus classification process itself (Ritter, 1972).

A substantial body of literature now exists which suggests that P300 latency is primarily sensitive to stimulus evaluation time, and is minimally affected by response choice time. Kutas, McCarthy and Donchin (1979) manipulated stimulus evaluation time in a task which measured P300 and RT under three levels of semantic discriminability. The task was run under both speed and accuracy instructions. P300 latency and RT increased significantly as the difficulty of stimulus discrimination increased.

Speed-accuracy instructions, however had significant effects on RT only. RT was approximately 136 ms shorter under speed instructions than under accuracy instructions, whereas P300 latency remained relatively stable. Under speed instructions, RT and P300 were closely correlated, with RT occurring only slightly after, or in some instances, before P300. In the accuracy instructions conditions P300 latency and RT latency became dissociated, RT latency being much longer than P300. The authors concluded that P300 latency was mainly determined by stimulus evaluation time, whereas RT was sensitive to this and other processes.

In a follow-up study, (McCarthy, & Donchin, 1981) the physical discriminability of stimulus was manipulated. These authors manipulated stimulus discriminability by embedding the target words "left" and "right" in a background of irrelevant character. In a "noise" condition, other letters of the alphabet made up the background. In the high discriminability condition the symbol "#" was presented in the background. Response choice time was also manipulated by varying stimulus-response compatability. The cue words "same" or "opposite" were presented before each stimulus. Subjects were instructed to press the button on the right in response to the target word "right" and the button on the left in response to the target word "left" when these were preceded by the cue word "same". The cue word "opposite" indicated that subjects were to press with the left hand to the word "right" and with the right hand to the word "left". P3 latency was again found to be selectively influenced by complexity of the stimulus evaluation process whereas stimulus discriminability and stimulus-response compatibility had additive effects on RT.

The data analysis performed by McCarthy and Donchin has been discussed by Magliero, Bashore, Coles, and Donchin (1984). These authors note that dual peaks were observed for P300, in addition to a well-developed CNV. These factors may therefore have confounded their identification of P300. Magliero et al. have since performed a series of similar follow-up experiments. (Magliero et al., 1984) which essentially replicate the findings of McCarthy and Donchin. The first of their studies was designed to verify that the P300 had been correctly identified by McCarthy and Donchin. In a second study, the procedures employed by the McCarthy and Donchin were replicated in order to assess the effects of stimulus response compability on P300 latency. Both studies concurred with the conclusions of McCarthy and Donchin -- P300 is primarily sensitive to the duration of the stimulus evaluation process and is relatively unaffected by S-R incompatibility.

In a related experiment, Duncan-Johnson and Kopell (1981) varied stimulus-response compatibility using a modified version of the Stroop color-word interference test. Subjects were presented with the names of colors printed in either their corresponding color of ink (i.e. "RED" printed in red ink) or in an incongruent colour of ink (i.e. "RED" printed in blue ink). Non-colour words were also presented. The color of ink was varied, being primary colors in some conditions and less discriminable colors in others (i.e. reddish purple or bluish green).

Subjects were required to read the printed words in one condition, and in another condition to name the colour of the ink in which the words were printed. Color discriminability had a significant effect on P300

latency. The trials on which intermediate hues were presented elicited later P300s than trials requiring the naming of primary colors. Color-word incongruity was found to have no significant effect on P300 latency, although RT was significantly delayed. The authors state that the invariance of P300 latency under color-word incongruity condition marks the response execution stage as the locus of the Stroop effect on RT.

Other authors have manipulated stimulus discriminability by varying the similarity between two classes of stimuli. Fitzgerald and Picton (1981), for example have observed P300 latency to increase as the frequency difference between target and standard tone pips decreased. Similar findings have been reported for visual stimuli (Mulder, Gloerich, Brookhuis, van Dellen, & Mulder, 1984). In this study, subjects were asked to respond to a vertically oriented line of a certain length. In the "similar" (re: low discriminability) condition the target was surrounded by other shorter lines with the same orientation. In the "discrimination" condition distractor stimuli were replaced by data. Stimulus-response compatability was manipulated by varying the position of the target relative to the response key. It was found to have no significant effects on P300 latency. The P300 latencies in the compatable and incompatable conditions, were in fact, virtually identical.

Other studies have however presented evidence suggesting that P300 latency may not reflect stimulus evaluation time exclusively. Two studies (Brookhuis, Mulder, & Gloerich, 1983; Mulder, Gloerich, Brookhuis, van Dellen & Mulder, 1984) have reported that it is also sensitive to relative response frequency (i.e., the frequency with which a given response is

called for). In these studies, P300 was recorded during a Sternberg task. Memory set items were presented in a "study" condition which was followed by the presentation of probe items. Subjects indicated whether or not items had appeared in the memory set. The probability of memory set items being presented in the probes series varied among trials. In some subjects P300 latency decreased for both positive and negative responses, as the probability of each of these increased. Since response probability was manipulated independently of stimulus probability, P300 latency may be somewhat affected by response choice time.

Corroborating evidence for this point of view is provided by the studies of Ragot and Renault (1981) and Ragot (1984). In contrast to the findings of most other studies, these authors have observed P300 latency to vary significantly under conditions which manipulated stimulus-response spatial compatability. In a visual discrimination task, Ragot and Renault varied the spatial position of the stimulus display, with respect to the responding hand . RT had previously been found to be shortest when stimuli are presented to the visual field on the same side as the hand used for responding (Craft & Simon, 1970). This is also the case when subjects respond with crossed hands, so that the right hand presses the left button and vice versa (Butshel & Rizzolati, 1977). The effect is therefore probably not due to the organization of anatomical connections between the visual field receiving stimulation and the responding hand. It is more likely that RT is delayed due to S-R incompatability (Ragot & Renault, 1981).

Ragot and Renault found both P300 and RT to be delayed by S-R spatial incompatibility, whether or not subjects responded with crossed hands. Responding with crossed hands in the high S-R spatial compatibility condition had no effect on P300 latency, although RT was significantly prolonged. The effects of S-R spatial compatability on P300 latency suggest that it may be generated after the response choice stage. Since no further delay of P300 latency was observed in the crossed hands condition, P300 is probably not involved in motor programming.

Ragot (1984) has replicated these findings. He has also observed that the motor potentials associated with these tasks are maximally recorded contralateral to the responding hand. P300s recorded during the same tasks were recorded symmetrically bilaterally. This finding, therefore provides further evidence that P300 is not involved in motor programming.

In spite of the apparent contradictory differences, the findings of Ragot et al. can be reconciled with others. Manipulations of S-R compatibility or speed/accuracy tradeoff do produce small changes in P300 latency. The absolute amount of the increase in P300 latency ranged from 14 ms in the Magliero et al. study to 50 ms in one condition of the Kutas et al study. The variation in P300 latency reached statistical significance in some studies (Duncan-Johnson & Kopell, 1981; Kutas et al., 1977; Magliero et al., 1984; Ragot, 1984; Ragot & Renault, 1981) but not others (McCarthy & Donchin, 1981). The extent of P300 variation in Ragot and Renault (1981) and Ragot (1984) is therefore similar to that reported by other studies. From these studies it may be concluded that P300

latency is primarily sensitive to processes leading to stimulus evaluation, although response choice processes may account for a small proportion of its variation.

While P300 latency may serve as an approximate index of stimulus evaluation time, the actual decision probably occurs before its peak onset (Goodin & Aminoff, 1984). Donchin (1979) and Duncan-Johnson and Donchin (1981) note that in order to be classified as "rare" or "not rare" a stimulus must first be evaluated. They suggest that since P300 amplitude is sensitive to stimulus probabilities, it may be an index of a "post-decisional event". From this point of view, stimulus evaluation would appear to precede even the peak of P300.

Desmedt (1981), and Desmedt and Debecker (1979) have proposed that P300 represents the "transient (partial) inhibition of the MRF (midbrain reticular formation) neuromodulation pressure on the telencephalon that is invoked by the central processor in the prefrontal cortex upon completing identification of a target..." This view differs from Donchin's contextual updating model in at least one important aspect. Desmedt's model suggests that P300 indexes the termination of a process, as opposed to Donchin's, which implies that P300 reflects the activation of a process. Desmedt's model therefore places an even greater emphasis on the post-decisional nature of P300.

Despite the fact that P300 may occur sometime after stimulus discrimination, it is nevertheless a valuable index of this process. The "liberal" estimate provided by P300 may be used appropriately when an outside limit for stimulus evaluation time must be determined. In the

series of studies which follows, P300 will be recorded in order to determine what portion of the subject's RTs are determined by processes other than stimulus evaluation.

Summary. The amplitude of a late (250-500 ms) positive component, the P300 has been observed to be determined by many experimental variables including the subjective probability of stimuli, the amount of meaning associated with the stimulus, and the amount of information which is extracted by the subject.

The latency of P300 is primarily sensitive to stimulus evaluation time but may be somewhat affected by manipulations of response choice time. Since accurate responses can occur before the peak of P300 latency it is probably not a "real time" index of the stimulus evaluation process. Despite this fact it is useful as a liberal estimate of the time at which stimulus evaluation is complete.

Reaction Time After Closed Head Injury

Conkey (1938) was perhaps the first to document the slowing of cognitive processes as a result of CHI. These deficits were later quantified by Reusch (1944) who collected reaction time (RT) data from a group of 57 CHI victims. Twenty-five of the patients tested by Reusch had recently sustained trauma. The other 32 patients were described as being in the chronic stages of recovery. Exact descriptions of the cases (for example, length of time since injury, duration of coma or PTA) were not provided. Simple RTs for the two patient groups did not differ significantly. RTs for both patient groups were however significantly

delayed compared to controls. The control subjects in this study did not appear to have been matched on age, sex, IQ or level of education.

In a later study Dencker and Lofving (1958) studied monozygotic twins in which one of the pair had sustained CHI. Patients were studied, on average, 10 year after their initial injuries. Two-thirds of the patients had spent one hour or less in PTA. Using their uninjured probands as controls, Dencker and Lofving found no impairment in simple RT in the CHI group. These results may nevertheless be due to the mild degree of injury sustained by these patients or the fact that they had recovered for such a lengthly period.

Severely injured CHI patients were tested by Norrman and Svahn (1961). The 22 patients who participated in the study had been unconscious for one week or more and had recovered for two or more years since their injuries. Patient RTs were compared with those of neurotic controls and RT norms which had been established for healthy adults. On a simple RT task, the performance of CHI patients was not found to differ significantly from either of the control groups. The CHI patients were however significantly slower than the control groups on a choice RT task.

The tendency for CHI patients to be impaired on only choice RT tasks has also been noted by Miller (1970). Miller collected data from five CHI victims who had been in PTA for at least one week, having sustained their injuries from three to twelve months previously. Subjects performed a choice RT task in which visually presented digits served as stimuli. The task involved pressing the correspondingly numbered button. The number of alternatives varied between condition. Miller found a dramatic increase

in patient RTs as the number of choices were increased. When regression lines were fitted to the data, patient and control were found to differ significantly in their slopes but not in their intercepts. The regression lines of both groups intercepted the Y-axis at the "zero" information point. This suggested that the RT differences between patients and controls were due to differences in the actual decision-making process, rather than in sensory or motor processes.

Visual RTs were examined by Gronwall and Sampson (1974) under 2, 4, 6, 8 and 10 choice conditions. They tested twelve mildly concussed CHI patients within 24 hours of injury. Subjects were required to indicate which of a series of "pinball"-type alleys a ball had been dropped. Response buttons, numbered from one to ten were located at the bottom of each alley. In one condition button numbers corresponded to the number of the alleys above them. In a second condition button numbers were reversed. In this task, only the eight and ten choice conditions revealed a significant difference between groups. As in Miller's study, the slope of the regression lines differed but not the intercept. Gronwall and Sampson therefore also concluded that the patient RTs were most likely delayed due to increased "central processing time" rather than slower movement time.

The studies reviewed thus far would suggest that choice RT is more affected by CHI than simple RT. This being the case, RT would appear to be delayed due to cognitive rather than motor factors. Some studies have nevertheless, noted exceptions to this rule. Klensch tested 76 patient who had sustained mild head injury. In contrast to what others had reported, Klensch found both simple visual and simple auditory RT to be

prolonged in his sample of patients. Curiously, choice RT was not significantly different from that of controls. The absolute between group differences were however similar (41 ms for simple and 36 ms for choice RTs). In Klensch's choice RT task, subjects were instructed to respond to the combination of a light flash and tone, but not to the tone alone. It is possible that the task may not have taxed cognitive decision-making processes to the same degree as one requiring an overt response to all stimuli. The small RT delay observed on the choice task may therefore be explained by the relative ease of the task.

More substantial delays in visual simple RTs in head-injured children have been observed by Winogron (1986). The patients in this study were divided into two groups according to severity of injury. Patients whose Glasgow Coma Scale Ratings were less than seven at the time of injury were considered to be severely injured, whereas those with a score from 7 to 15 were considered mildly injured. The average length of time since injury was one year. Simple RT was found to be significantly prolonged in the severely head injured group (by approximately 125 ms) relative to controls. There were no significant differences, however, between the RTs of the two patients groups, or between the mildly injured patients and controls. The simple RTs in this study, it should be noted, were extremely long (500 - 600 ms) for both groups. It is unclear if this were due to the age of subjects, or the instructions that they received. Subjects were told that trials would be cancelled if they lifted their finger from a "home plate" button before the stimulus was presented.

In another recent study van Zomeren and Deelman (1981a) measured visual RT under both choice (R-2 and, 4) and simple (R-1) conditions (R-1, R-2, R-3, ... Rn indicate the number of response choices). The 20 patients who participated in this study had sustained mild CHI (coma duration was less than 1/2 hr). Although their recovery time was not specified, it was stated that eight of the patients had returned to work and were free of symptoms. In another three patients only "non-neurological" deficits were apparent. A previous study on brain-damaged subjects (Benton & Blackburn, 1955) had demonstrated that such patients were not disproportionally slower on bimanual tasks (where subjects responded using both hands). This being the case, van Zomeren and Deelman ran the R-2 and R-4 conditions both unimanually and bimanually. An auditory warning signal was presented 1 s. prior to the visual stimulus to be discriminated. Closed head-injured subjects were found to be significantly slower on all tasks, although much less so on the bimanual task. Patient RTs were delayed significantly more than those of controls when the number of response alternatives was increased. The data also suggested that task complexity had a greater effect on patients who had sustained more severe injuries.

A follow-up study by van Zomeren and Deelman (1981a) investigated choice RT in three groups of patients with CHIs or different severities. Eleven patients in each of the three groups were tested. Group classification was based on coma duration being either (a) less than one hour, (b) from one hour to one week, or (c) longer than one week. The three groups were matched on age and average recovery time (148 days).

Subjects were tested only on the simple and four choice unimanual tasks. The results again indicated that severely injured patients were more affected by task complexity than the mildly injured group. A comparison of individual patients in the "severe group" suggested that this disadvantage decreased somewhat as a function of time since injury.

In order to construct recovery curves for RT another study was run in which three patient groups were repeatedly tested on simple and choice RT (R-4) (van Zomeren & Deelman, 1981b). Patients were divided into groups using the same criteria as in the preceding study (coma duration of less than one hour (N = 27), one hour to one week (N = 18), or more than one week (N = 12)). Testing was to take place at five weeks, and five, eight, 14 and 20 months following injury. The moderately and severely injured groups, however could not be tested before ten weeks of recovery.

RT discriminated between the three patient groups and decreased as a function of recovery time. Three interactions were observed. Severely injured patient were significantly more affected by the number of choice alternatives (one vs four) than mildly injured patients. Furthermore, the RTs of severely injured patients decreased over successive testing sessions to a greater extent than those of midly injured patients. The choice RTs of both groups continued to improve over all testing sessions whereas simple RTs levelled off by eight months. At almost two years (22 months) after the initial injury, the choices RTs of patients in the "mild" and "moderate" groups were similar to those of controls. Their simple RTs remained significantly later than controls (20 and 25 ms respectively). The RTs of the severely injured group remained sig-

nificantly delayed for both choice (80 ms delay) and simple RT (24 ms delay) tasks.

In a more recent study (MacFlynn, Montgomery, Fenton, & Rutherford, 1984) serial testing was performed on 24 patients who had sustained mild CHI (PTA was in all cases less than 24 hrs). Testing took place at 24 hrs, six weeks and six months after their injuries. Patient choice RTs showed significant improvement at each testing session. By six months, patient RTs were actually faster than those of controls. These results are therefore similar to those obtained from the mildly impaired group studied by van Zomeren and Deelman. The choice RTs of MacFlynn et al's group of patients, however, seem to have recovered more quickly and more fully than those of van Zomeren and Deelman patients.

There is agreement between the studies reviewed thus far on a number of issues. The choice RTs of CHI patients are usually delayed relative to controls. The extent of this delay is affected by both severity of injury and the number of response alternatives. Furthermore, whatever deficit may account for the slowing of RT appears to recover somewhat over time, although residual delays in RT may persist indefinitely in severe CHIs. What has yet to be determined from these studies is the locus of the RT effect. In other words what process or stage of response production is affected by CHI?

van Zomeren and Deelman (1981) and van Zomeren, Brouwer and Deelman (1981) have partitioned RT into two subcomponents: "decision time" and "movement time". They attempted to determine what portion of the RT delay might be attributed to "central information processing" and/or "motor"

factors, respectively. Eight severely injured CHI patients (PTA = 7 to 124 days) were tested between six weeks and six months following their injury. Mounted on the response box were a "home" button and four response keys. Subjects were instructed to rest their index finger on the "home" button and release it only after deciding which response key to press. Simple and choice (R-4) visual RTs were recorded. The authors reasoned that "decision time" was the delay between stimulus onset and the time taken to release the "home" button whereas "movement time" was the time between releasing the home button and pressing the response button.

Decision time was found to be significantly longer in patients. Moreover decision time increased significantly in both groups as greater numbers of stimulus and response alternatives were presented (R-4). This effect was significantly greater for patients than for controls. Movement time was not significantly different between patients and controls. It is interesting to note that the movement time of both groups, however, were significantly longer in the R-4 compared to R-1 task. The observation of a complexity effect on movement time suggests that it is influenced by information load as well as factors influencing motor output.

Several criticisms of the methods employed in the van Zomeren et al. studies can be raised. Subjects were instructed to release the home button only after response choice and presumably stimulus evaluation were complete. This implies that a serial model of information processing was assumed (ie: response initiation would begin only after stimulus evaluation and response choice were completed). Given the interaction between cognitive factors and movement time in this and other studies (see Fleer,

1972; Wadem Newell, & Wallace, 1978) such an assumption may be tenuous. Secondly, even if response processing did occur serially, the authors had no means of determining if subjects had in fact complied with their instructions.

Decision time and movement times were also recorded in a later experiment by these authors (van Zomeren & Deelman, 1981c). This experiment employed only the R-4 task which was presented with and without a distracting stimulus. Twenty CHI patients were tested whose injuries ranged from mild to severe on the Russell Scale. At the time of testing, the patients had recovered for three to twelve months. Two conditions were presented. In the control condition, the stimuli were the illumination of the response buttons. Subjects were required to press whatever response button had lit up. In the "distraction" condition an irrelevant button was illuminated simultaneously with the response button. RT was again partitioned into decision time and movement time. The decision times of patients were found to be delayed significantly more than those of controls in the distraction condition. Movement time was significantly longer in patients but did not vary significantly between conditions.

The longer movement times of patients are what might be expected in light of the earlier findings of van Zomeren et al. In their previous study, the presentation of four response alternatives caused a much larger delay in patient movement times. The exaggeration of decision time in the distractor condition has been interpreted by the authors as evidence for an increase in response choice time. The response buttons in this study, were however also the stimuli. It is therefore possible that the il-

lumination of the irrelevant button increased stimulus discrimination time rather than response choice time.

Winogron (1986) similarly studied the consequences of the severity of CHI in children. In these experiments subjects were presented with two lines and were asked to indicate which was the longest. The mean RTs in the first experiment were 1097 ms for the severely injured group, 918 ms for the mildly injured group and 821 ms for the control group. The RTs of the severely injured group were significantly longer than both other groups. The RT difference between mildly-injured patients and controls however did not reach significance. In a second study, the RTs of the three groups were reduced using a criterion window within which subjects were told to respond. In addition, feedback (FB) was provided informing the subjects of either the speed or the accuracy of responding. Three criterion windows were used which were 1 s, 1.5 s or 2 s in duration. RTs were shortest when the 1 s criterion window was provided. In this condition, RTs were 825 ms for the severely injured group, 750 ms for the mildly injured group and 650 ms for the control group. The manipulations employed in this study therefore resulted in a 200-250 ms decrease in the RTs for all groups. In spite of this however, patients RTs remained 100 and 200 ms longer than controls for the severely and mildly injured groups respectively. Nevertheless, the fact that RTs could be shortened by as much as 250 ms in CHI patients suggests that a good portion of the overall decision time is due to response production time and not stimulus evaluation time.

One final study should be reviewed that measured the effect of fatigue on RT. Dencker and Lofving (1958) tested 28 CHI patients (previously described) on continuous RT tasks involving ten response alternatives. RT and accuracy were monitored over the duration of a 15 minute RT task. Although patients made more errors than controls there was no increase in errors over time. RT was likewise significantly longer for patients but did not change over time. This study therefore provides no evidence that fatigue or lapses of vigilance affect the performance of CHI patients on RT tasks.

Summary

The studies reviewed here are in agreement on a number of points. In the majority of studies, choice RT has been found to be more affected by CHI than simple RT. Relative to controls, CHI patient simple RTs are usually delayed by about 20-30 ms. This difference reached statistical significance in some cases but not in others. Choice RT however is usually delayed by 80 - 200 ms. In exception to this generality are the studies by Winogron (1986) which found much larger delays in both simple (125 ms) and choice (180-280 ms) RT.

Severity of CHI has been found to significantly affect RT. Patients with more severe injuries have generally slower RTs which are affected to a greater extent by increasing the number of stimulus and response alternatives. There appears to be an inverse relationship between recovery time and RT. Speed of responding in choice RT recovers more slowly than simple RT. In the severely injured patients, studied by van

Zomeran et al. (1981b), both simple and choice RT were found to be impaired after two years of recovery.

Attempts have been made to measure decision time independently of movement time. The results of these studies are difficult to interpret since "movement time" appears to interact with response choice time.

Electrophysiological Studies of Information Processing in the Head Injured

Choice RT studies have generally demonstrated a significant delay in information processing following CHI. They have been less instructive, however in determining the locus of this deficit. This is perhaps due to the difficulty in interpreting group differences in mean (or median) RT. As has already been pointed out, quantification of the time spent in various hypothetical stages of information processing is difficult when these events must be inferred retrospectively. Global psychological states such as attentiveness or arousal are equally difficult to measure for the same reason. Deficits in these states are nevertheless often suggested as causes for the slowing of decision making in the head-injured (e.g., Gronwall & Sampson, 1974).

Many cognitive events or states may be studied directly using electrophysiological measures (Hillyard & Kutas, 1983). A limited number of the electrophysiological studies with CHI patients have examined attention and vigilance. "Phasic" attention, or the ability to concentrate attention for limited periods of time, has been studied using the contingent negative variation (CNV). Some authors claim that the CNV may reflect preparation to respond (Walter, 1964), or orienting and arousal (Loveless & Sandford, 1974). It develops following the presentation a "warning stimulus" (WS) which informs the subject of the impending

delivery of an "imperative stimulus" (which must be responded to). In the classic "Go/No Go" paradigms one of two stimuli follow the warning signal. Subjects must respond to one stimulus (S2) and ignore the other.

McCallum and Cummins (1973), applied this paradigm to CHI patients. The results of this study are however difficult to interpret. The authors found a significant attenuation of the CNV in their sample of patients. The patients tested, however, included neurosurgical cases of mixed etiology, only one fifth of whom had suffered CHIs.

Rizzo, Amabile, Caporali, Spadora, Zanas, and Morocutti (1978) tested 27 CHI patients who had been in coma for between two and 90 days (mean = 15 days). They had been injured at least five months prior to testing. The CNVs of patients in this study were significantly different from those of controls only with respect to the area encompassed by the CNV. RTs were not recorded.

In a later study (Curry, 1980), 25 patients were tested immediately following the resolution of PTA. The duration of PTA was less than one day for 14 patients, less than seven days for five of the patients, and longer than seven days in the remaining six patients. Curry modified the standard Go/No Go paradigm such that the warning stimulus informed the subject of whether or not a response would be required to the subsequent, imperative stimulus. In two of the most severely injured patients, CNVs were absent or attenuated. In roughly one half of the other patients exaggerated CNVs were recorded to warning stimuli regardless of whether or not they signalled that a response would be required. Well-developed CNVs were observed in control subjects only when the warning stimulus signalled that they should prepare to respond.

Suffield (1985) studied moderate to severly head-injured outpatients. He sub-divided the CNV into early and later intervals. Rohrbaugh and Gaillard (1983) had suggested that the early portion of the CNV might reflect a process of orientation and has thus been termed the "O-wave". A later portion of the CNV may reflect an expectancy to respond to the imperative stimulus and thus has been termed, the "E-wave". Suffield used a paradigm in which WS was followed 1.5s later by S2. S2 could be either a high or a low frequency tone pip. The subjects were informed to respond to one frequency ("Go") and to withold responding to the other ("No Go"). The pitch of WS was varied, being also high or low. In WS-Stop trials, WS informed the subject that no response was required to the second stimulus regardless of its pitch. In WS-Go trials, a response might be required depending on the frequency of the second stimulus. Suffield reported that the head-injured had little difficulty with the task. As expected, their RTs were significantly longer than matched controls. Two distinctive CNV patterns were observed for the patients. In WS-Stop trials, a small O-wave initially developed in frontal sites for controls and then returned to baseline. No E-wave was apparent. For one group of the patients, an unusually large O-wave developed and only slowly returned to baseline. It was as if these patients were needlessly preparing to respond to the second stimulus. In WS-Go trials, these patients had normal E-waves. However, a second group of patients had unusually small E-waves in central areas. Their O-waves were normal in WS-Stop trials. It was as if these patients were not developing adequate preparation to respond. Remarkably, both groups had similarly long median RTs.

These studies may indicate that many CHI victims have impairments of "phasic" or directed attention. The poor performance of these patients on

many tasks has also been attributed to fatigability or low arousal (Conkey, 1938; Reusch, 1944; Gronwall & Sampson, 1974).

Tonic alertness has been studied in a number of vigilance tasks. Dencker and Lofving (1958) monitored choice RT during a 15 minute continuous task but did not record physiological data. Overall, patients gave fewer correct response. No interaction was observed however, between time and accuracy or time and RT.

Heart rate variability (HRV), which is thought to be associated with the amount of effort invested in a task (Mulder, 1980) was measured by Brouwer and Wolffebaar (1985). Eight moderately to severely head injured subjects and eight controls performed a discrimination task in which they were asked to detect the weaker of two auditory stimuli. Subjects were tested during a 30 minute and a 10 minute session. HRV was found to be significantly greater for both groups in the second half of the 30 minute task. There was no interaction however of groups and time on task.

Brouwer and van Zomeren (1985) performed an auditory vigilance task in which RT and EEG were recorded simultaneously. The 14 patients in this study had sustained severe CHIs within the six months preceding testing. During the second half of the 30 minute testing session, 50% of control subjects showed performance decrement and EEG signs of drowsiness. By contrast, vigilance became impaired in only 18% of patients. Rather surprisingly, the patients in this study were therefore more alert than controls.

Similar findings have been reported in head injured children by Martinius, Hoffmann, Mayer and Kliapera (1977). The children were divided into two groups according to severity of injury. The least severely injured group (coma duration = < 5 days) had normal RTs and accuracy

levels, but EEG indices of arousal were significantly higher (i.e. patients were more aroused) than that of controls. In the severely injured group (coma duration = > 5 days) RT and accuracy were impaired relative to controls but EEG arousal was normal.

Floria-Ciocoin (1979) tested two groups of CHI patients. Those described as having "post-traumatic neurosis" showed more EEG signs of arousal than control. In patients with "post traumatic dementia", the opposite was observed. It has been suggested (Van Zomeren, 1981) that mild to moderately injured patients may exert more effort in order to cope with the demands of a task, whereas such compensatory strategies are ineffective for severely injured patients. Although this has yet to be determined, neither the behavioural nor the physiological data provide any evidence of decreased arousal or vigilance in CHI patients.

A limited number of studies have also recorded P300 in CHI patients (reviewed by Papanicolaou, 1987). Papanicolaou, Levin, Eisenberg et al. (1984) recorded P300 in 18 CHI patients. Severity of injury was not quantified, although it was stated that it was comparable in all patients. Passively evoked P300s to rare tones were found to be later in those patients who were still in PTA but were normal in patients who were alert and oriented.

Passively elicited P300s are perhaps less sensitive to the effects of CHI than those recorded during the active performance of a target detection task. Curry, Cooper, Cummins, and Skidmore (1985) and later Campbell, Houle, Lorrain, Deacon-Elliott and Proulx (1986) recorded ERPs during auditory and visual oddball tasks. In both studies the amplitude of P300 was markedly attenuated in the head-injured. In the Campbell et al. study, P300 was also attenuated in the head injured when a stimulus

was omitted from a train of regularly occurring stimuli. A second experiment by Campbell et al. (1986) investigated the possible interacting effects of fatigue. Whether task duration was 1, 4 or 16 min, P300 was consistently attenuated in the head-injured. Across all conditions of this study, Campbell et al. found that P300 latency was delayed by 50-75 ms in the head-injured. Interestingly when RT was also measured, it was slowed by approximately 150 ms. The behavioural hit rate was essentially identical between controls and patients. Misses and false positives were, in fact, quite rare.

A large P300 can also be recorded to feedback stimuli informing subjects of the correctness of their decisions (Johnson & Donchin, 1978; Campbell, Courchesne, Picton, & Squires, 1979). This P300 tends to take on a more anterior distribution than the parieto-central P300 observed in oddball tasks. Deacon-Elliott and Campbell (1987) have observed that the feedback P300 is also attenuated and is prolonged by approximately 50 ms. in the head-injured.

The fact that P300 is delayed in CHI patients may indicate that they experience some degree of difficulty in stimulus evaluation. As mentioned RT, however, is usually delayed much in excess of this. A slowing of the stimulus evaluation stage could therefore not account for all of the delay in patient RT. Since a good portion of the RT delay occurs between P300 and the completion of the response, processes leading to response choice and/or execution appear also to be susceptible to the effect of CHI. The RT studies which have been reviewed indicate that motoric deficits have a minimal effect on RT, as compared to factors which determine central processing time. RT therefore appears to be delayed in part due to a deficit in stimulus classification processes, but also to a deficit

in carrying out response choice and production. A major objective for this thesis will be to manipulate the subjects' emphasis on either speed or accuracy of responding.

Van Zomeran (1981) suggests that following CHI "... there might be a change in the criterion ß or an increased cautiousness ...". This has also been suggested by Brooks (1974). If patients were in fact adopting a more stringent criterion for responding, this might cause them to stress accuracy at a cost of speed. This could explain the delay in response choice and production times. Lowering the subjects' level of cautiousness less emphasis on accuracy should be accompanied by a reduction in RT. By contrast, increasing their level of cautiousness (more emphasis on accuracy) should increase RT. It is expected that patient RTs may be decreased at least to their P300 latencies. Since stimulus evaluation is complete by this time, RT will probably be reduced without any great difference in accuracy. Three different experiments are described in which classical paradigms were adopted from cognitive psychology to manipulate the speed-accuracy tradeoff function. In the first study RT will be manipulated using experimental instructions to stress either speed or accuracy of responding. The second study provided more stringent controls over speed of responding in order to further reduce RT. In this study, subjects were required to respond within a defined interval. Furthermore, feedback was provided informing the subject that the response had occurred within the window or that the response choice was correct. In a third study, cues for speed of responding were gradually removed in order to determine whether speeded performance would be maintained after their removal.

CHAPTER II

Experiment 1

Perhaps the most persistent disability arising from closed head injury (CHI) is a slowing of decision-making. In the past 40 years this deficit has been examined in a number of reaction time (RT) studies (Dencker & Lofving, 1958; Gronwall & Sampson, 1974; Klensch, 1973; McFlynn, Montgomery, Fenton, & Rutherford, 1984; Miller, 1970; Norrman & Svahn, 1961; Ruesch, 1944; van Zomeren & Deelman, 1981a, 1981b). In most studies, RT has been found to be delayed when two or more stimulus and/or response alternatives were presented. Delays in "simple" RT, involving a single, invariant stimulus and response have been also reported although with less consistency (Klensch, 1973; Ruesch, 1944; van Zomeren & Deelman, 1981a; Winogron, 1986). The extent of delay in simple RT is normally much less than in choice RT.

Since simple RT is comparatively less affected by CHI than choice RT, a purely motor disorder would not appear to account for the entire delay seen in RT. Miller (1970), has postulated that a cognitive, rather than a motor deficit might be primarily responsible for the slower RTs of the head injured.

In an effort to clarify this issue van Zomeren and Deelman (1981) partitioned RT into two subcomponents, that they labelled "decision time" and movement time. Subjects performed a visual discrimination task. They were instructed to move their hand from a "home plate" button only after deciding which response key to press. The time preceding the release of

the home plate key was recorded as "decision time". The time between releasing the home plate key and pressing the response button was considered "movement time". Decision time was found to be significantly prolonged in the head injured. As the number of stimulus and response alternatives increased (from one to four), patient RTs increased significantly more than those of controls.

Patient movement times were not significantly longer than those of controls. The number of response alternatives however had significant effects on the movement times of both groups. "Movement time" thus appeared to be affected by central processing time.

In a follow-up study (van Zomeren & Deelman, 1981c) only the four-choice condition was employed. Patient movement times were found to be significantly longer in this task. Nevertheless, cognitive processes most likely contributed to the duration of what the authors called "movement time". It is therefore difficult to determine the extent to which motor deficits influenced patient RTs.

As noted in the first chapter of this thesis, an inherent disadvantage of RT as a measure is that the stages of information processing leading to response production cannot easily be observed independently of one another. Some researchers have recently turned to event-related potentials (ERPs) as chronometric markers of the decision making process. These allow the experimenter more direct access to the covert mental events leading up to response production obviating the need to infer their timing through the elaborate and laborious analysis of single trial RTs.

A late (250-450 ms) positive component, "P300", has come to play a particularly significant role in the study of decision-making. P300 is maximally recorded in parieto-central areas, in response to rare, task-relevant stimuli. There is evidence which suggests that its latency may be primarily determined by stimulus classification time and only minimally affected by response selection time (Brookhuis, Mulder, & Gloerich, 1983; Duncan-Johnson, & Kopell, 1981; Fitzgerald, & Picton, 1981; Kutas, McCarthy, & Donchin, 1979; Magleiro, Bashore, Cole, & Donchin, 1984; McCarthy, & Donchin, 1979; Mulder, Gloerich, Brookhuis, van Dellen, & Mulder, 1984; Ragot, 1984; Ragot, & Renault, 1981). Campbell, Houle, Lorrain, Deacon-Elliott and Proulx (1986), reported that the P300s of CHI patients were delayed by only 50-70 ms in comparison to matched controls, whereas RT was prolonged by approximately 150 ms. This would suggest that stimulus evaluation processes are indeed prolonged in the head-injured. A good portion of the delay in patient RTs however would appear to be associated with processes other than stimulus classification.

van Zomeren (1981), and Brooks (1974) have suggested that CHI patients may respond more cautiously than controls. A number of studies have demonstrated that RT increases as the subjects emphasis on accuracy becomes more stringent (Brewer & Smith, 1984; Lappin & Disch, 1977, Pachella, 1974; Wood & Jennings, 1976). Several recent studies have observed, furthermore that the P300 - RT time interval varies as a function of the subjects trading speed for accuracy (Kutas, McCarthy & Donchin, 1977; Ford, Pfefferbaum, Tinklenberg & Kopell, 1982; Ford, Johnson, Wenegrat, & Kopell, 1983). When subjects are instructed to

respond as quickly as possible, without regard for the number of errors committed, RT occurs only slightly after, or often before P300 peak latency. When accuracy is stressed, however, the two become dissociated. RT occurs much later, while P300 latency remains relatively unchanged. It is possible that CHI patients adopt a response strategy through which they emphasize accuracy at a cost of speed. This might account for the patient RT - P300 latency dissociation. If so, it should then be possible to shorten RT using classical manipulations employed in cognitive psychology. It is also possible that the slowing of patient RTs could be due to a residual motor deficit. In this case, manipulations designed to shorten RT should be ineffective.

In the present study, instructions to subjects will be altered to emphasize speed of responding in some conditions and accuracy on others. It is expected that under speed instructions, patient RTs will be shortened to within the range of their P300 latencies (stimulus evaluation time). Past research has indicated that such instructions should have minimal effect on P300 latency. P300 latency should therefore remain relatively stable.

Methods

Subjects

Eight closed head injury victims were matched with controls on the basis of gender (6 males, 2 females), age (mean = 27 range = 19 to 46 for patients, mean = 26 range = 19 to 36 for controls) and years of education

(mean = 14, range = 10 to 19 for patients, mean = 13 range = 11 to 16 for controls). The demographic and neurological data for individual patients is presented in Table 1.1. The hospital records of patients did not clearly specify the duration of coma or PTA. It was therefore necessary to estimate these, as well as Glasgow Coma Scale ratings, on the basis of the information that was supplied. The mean duration of coma at the time of injury was estimated to be 40 days (range = 13 to 75 days). Estimates of PTA duration could only be made for six of the eight patients. The mean PTA duration for the group was 71 days (range = 30 to 150 days). According to the Russell Severity Scale (Russell, 1932) which classifies CHIs according to PTA duration, the group of patients in the present study would be considered to have sustained very severe injuries. By the time of testing, an average of 4.6 years had elapsed since their injuries (range = 3.7 to 6.9 years) (see Table 1.1).

The mean Glasgow Coma Scale score (Teasdale & Jennett, 1974) rating was estimated to be 4.7 (range = 4 to 5). A rating of 8 or less on this scale is associated with deep coma, and poor prognosis for recovery. Patients were assessed six months before agreeing to participate in the study on the Glasgow Outcome Scale (Jennett & Bond, 1975). The mean score for the group was 1.6 with a range of 1 to 3 (a level 0 or 1 rating corresponds to "good recovery" whereas a rating of 2 or 3 corresponds to "moderately" disabled). CT scans were not performed on all patients. In some of the patients for whom this information was available, CT scans indicated the presence of focal lesions. The lesions were heterogeneous

with respect to size and location (see Table 1.2). In addition to focal lesions many patients showed evidence of diffuse damage.

Prior to testing, patients underwent audiometric assessment using the method of limits and brainstem evoked potentials. The frequencies of the tones used were 500, 1000, 2000 and 4000 Hz. All patients had normal hearing (within 30 dB ISO of threshold) for these frequencies in at least one ear. Control subjects reported having no known auditory or neurological impairments.

Subjects were solicited on a volunteer basis. They received an honorarium of $20.00 for their participation in the study.

Procedure

Following the placement of electrodes, subjects were seated in a sound-attenuated testing chamber. The ambient noise level, as measured by a Bruel and Kjaer model 2209 sound level meter was approximately 45 dB SPL. Stimuli were 80 dB SPL, 2000 Hz "target" and 1000 Hz "standard" tone pips. Total stimulus duration was 55 ms, including a rise and fall time of 5 ms. Stimuli were presented monaurally to the subject's better ear through Telephonics TDH-39 headphones. A locally designed auditory stimulus generator delivered the accoustic signals. The rate of presentation was 1/1.1 sec. Stimuli were triggered and timed by a Cromenco Z-2 microcomputer (see Makasare, Campbell & Bell, 1985). Two hundred stimuli were presented in each trial. Standard/target probability was in .50/.50 or .70/.30 in different conditions. The order of presentation of the standard and target stimuli was determined by a random Bernoulli sequence.

The subjects task was to press one button upon detection of the standard and another upon detection of the target. The two response buttons were separated by a distance of approximately 2.5 cm. Subjects responded with the index and middle fingers of their dominant hand.

Two conditions were presented in which instructions to subjects emphasized either speed or accuracy of responding. In the accuracy condition, subjects were asked to respond as quickly as possible following presentation of the stimulus, but emphasis was placed on avoiding errors. In the speed condition, subjects were instructed to respond as quickly as possible, regardless of the number of errors committed.

Within each instructional set (speed vs. accuracy) the probability of target presentation was also manipulated being, as mentioned, either .50 or .30. The resulting four condition (two instructional sets x two target probabilities) were run randomly. The four conditions were run a second time in reverse order to verify replicability of results. The total testing time was approximately 90 min.

Reaction times which fell outside the sweep were automatically rejected. In cases where subjects pressed two buttons, or pressed the same button twice, only the first response was recorded.

EEG Recording. The electroencephalograph (EEG) and electrooculogram (EOG) were recorded using Beckman Ag/AgCl electrodes. Scalp electrodes were affixed using collodion impregnated gauze and adhesive collars. Prior to the application of electrodes, the skin was abraded with a sterile needle in order to reduce skin potential artifact (Hillyard & Picton, 1972). Inter-electrode impedence was below 1 KOhm. The EEG was

recorded from midline, frontal and central scalp locations (international 10-20 system; Jasper, 1958) and referred to the mastoid ipsilateral to the ear of presentation. Vertical and horizontal EOG activity were simultaneously monitored by electrodes placed on the supraorbital ridge of the left eye and the infraorbital ridge of the right eye. EEG and EOG signals were amplified on a Nihon Kohden ME-95D electroencephalograph. The high filter was set at 35 Hz. The time constant was 1.5 s.

EEG signals were averaged on-line by the Cromemco microcomputer. Averaging of the physiological activity began 200 ms prior to stimulus onset and continued for another 1000 ms. following it. A total of 300 data points was sampled (i.e., one every 4 ms.). Sweeps in which EOG or Fz activity exceeded $\pm$100 μv were rejected from the averaging process. Trials in which behavioural responses occurred outside of the sweep time (i.e., 1000 ms) were also rejected. The averaged waveforms and RTs were stored on diskette for subsequent off-line plotting and scoring.

Data Scoring

Behavioural data. The subjects behavioural response was sorted according to stimulus-response contingencies (i.e. correct and incorrect responses for each of the two stimuli presented). The proportion of standards and targets correctly and incorrectly detected were used to calculate the signal detection measure d'. RT was measured with a resolution of 4 ms by the Cromemco microcomputer. The subject's RT was also fed into the A/D converter for display purposes. RT histograms were plotted on the basis of 10 ms bins.

Physiological data. The physiological data were also sorted according to stimulus-response contingencies. The plotted waveforms were scored by visual inspection. P300 was scored at the Pz scalp location. It was identified as the largest positive component in the 250 to 500 ms post-stimulus epoch. The identification of P300 was based in part, on the replicability of positive waves in this time interval. In cases in which two equally large dual positive peaks were apparent in both replications, the earliest of the two was identified as P300. When no P300 was apparent a "missing data" value was assigned for that condition.

Statistical analyses. RT, P300 amplitude, and P300 latency (at Pz) were submitted to four-way analysis of variance (ANOVA) having one between factor, group (patients vs controls) and three within factors; Instruction (speed vs accuracy), standard:target Probability (70:30 vs 50:50) and Replication (first vs second). Separate ANOVAs were calculated on standard and target stimuli for each of the dependent measures.

Tables of means of the various dependent measures will be presented at the end of each chapter. The corresponding ANOVA tables are included as Appendices.

Results

Behavioural Data

Accuracy was quite high for both groups in all conditions. The overall percentage of correct responses was 95% for both patients and controls (see Table 1.3). The small number of errors was therefore

insufficient to permit meaningful comparisons of the signal detection measure d'. Similarly, the low error rate precluded the averaging of physiological activity during error trials. Only data based on correct detections will therefore be presented.

Table 1.4 presents mean RT data for the standard stimuli while those for targets are presented in Table 1.5. A main effect of probability was found for RTs to standard stimuli ($F(1,10) = 27.93$, $p < .01$). RTs were significantly shorter when the probability of standard occurrence was high (mean = 359 ms, SD = 62 ms) than when it was low (mean = 393 ms, SD = 60 ms). Instruction also had significant effects on RTs to standard tones ($F(1,10) = 18.60$, $p < .01$). Under speed instructions RTs were significantly shorter (mean = 346 ms, SD = 48 ms) than under accuracy instructions (mean = 407 ms, SD = 74 ms). Significant group differences were also noted ($F(1,10) = 28.69$, $p < .01$). The mean RTs were 451 ms (SD = 56 ms) for patients and 302 ms, (SD = 56 ms) for controls. There were no significant Instruction x Group or Probability x Group interactions.

Stimulus probability had no significant effect on RTs to targets. A main effect of instruction was observed ($F(1,10) = 24.10$, $p < .01$). RTs were decreased from 427 ms (SD = 72) under accuracy instructions, to 368 ms (SD = 57) under speed instructions. Significant group differences were also observed for RT to target stimuli ($F(1,10) = 15.07$, $p < .01$). The means were 457 ms (SD = 60) for patients and 338 ms (SD = 69) for controls. Again there were no significant interactions.

Physiological Data

Individual patient and control waveforms following standard and target stimuli are superimposed in Figure 1.1. The speed condition in which standard:target probability was 70:30 was arbitrarily chosen for illustration. While there is some within group variability, this variability is considerably less than that observed between groups. The grand average of these subjects is illustrated in Figure 1.2. The grand average appears to be a faithful representation of the general within group pattern of responding and will therefore be employed for illustrative purposes in the remainder of this chapter. The amplitude of P300 to standard stimuli (see Table 1.6) was significantly larger when its probability of occurrence was .50 (mean = 5.11 μv, SD = 3.5 μv) than when it was .70 (mean = 4.00 μv, SD = 2.3 μv) ($F(1,11) = 8.38$, $p < .01$). Instructional set also had significant effects on the amplitudes of P300s to standard stimuli ($F(1,11) = 7.95$, $p < .01$). Under speed instructions P300 amplitudes (mean = 5.1 μv, SD = 5.1 μv) were significantly greater than under accuracy instructions (mean = 4 μv, SD = 2.50 μv). P300 amplitude to standards were significantly larger for controls (mean = 6.40 μv, SD = 3.19 μv) than for patients (mean = 2.71 μv, SD = 1.78 μv) ($F(1,11) = 14.90$, $p < .01$).

In the case of target stimuli (see Table 1.7) P300 amplitude was significantly larger when its probability of occurrence was set at .30 (mean = 9.30 μv, SD = 4.26 μv) than when it was set at .50 (mean = 6.36 μv, SD = 2.95 μv) ($F(1,11) = 20.94$, $p < .01$). Mean P300 amplitudes for patients (mean = 5.04 μv, SD = 2.06 μv) and controls (mean = 10.62 μv, SD

= 5.17 μv) also differed significantly ($F(1,11) = 10.86$, $p < .01$). There were no significant interactions.

Mean P300 latency following the standard stimuli are presented in Table 1.8. The effects of replication, probability, and instruction were not significant. Significant differences were observed however between groups ($F(1,11) = 10.04$, $p < .01$). Patient P300 latencies to standard tones (mean = 368 ms, SD = 44 ms) were significantly longer than those of controls (mean = 310 ms, SD = 33 ms).

Similarly, there were no significant effects of replications, probability, or instruction on P300 latencies for target stimuli (see Table 1.9). Group differences were however significant, ($F(1,11) = 12.33$, $p < .01$). Patient P300 latencies (X = 366 ms, SD = 39 ms) were again longer than those of controls (X = 308, SD = 34 ms).

Discussion

The effects of the various experimental manipulations on ERP waveforms are presented in Figures 1.3 and 1.4. Although the topography of the late positive wave was not quantified, it was apparent in the individual waveforms and in the grand averages, that it was maximum in parieto-central areas of the scalp. This was consistent between groups and across experimental manipulations. Its scalp topography and its relatively long latency (300-380 ms), therefore provide convincing evidence that this late positive wave is essentially identical to the "P3b" or "P300" often reported in the literature (Donchin, 1981).

The effect of stimulus probability on RT was significant for standard stimuli but not for targets. Subjects may therefore have developed a response bias for the more frequently occurring standard stimuli since their global probability of occurrence was greater than that of targets. Duncan-Johnson and Johnson (1982) have suggested that response preparation may be affected by an adjustment of the subjects' criterion on the basis of stimulus probability. In their study, RT was reduced by 48 ms when stimulus probability changed from .20 to .50. This is similar to the 34 ms RT difference between the p = .50 and p = .70 conditions in the present study. In addition to the effects on RT, Duncan-Johnson and Donchin also observed a significant decrease in P300 latency as stimulus probability increased. The absence of stimilar findings in the present study was perhaps due to differences in the nature of the task employed. Duncan-Johnson and Johnson provided a warning stimulus which signalled the probability of the physically identical imperative stimulus which was to be presented. The probability of the imperative stimulus "matching" the warning stimulus varied between conditions. The decrease in P300 latency which was observed in their study may therefore have been due to the facilitation of stimulus evaluation processes.

P300 amplitude was, as would be predicted, significantly larger for both standards and targets as the probability of each was decreased (Duncan-Johnson & Donchin, 1982). P300 amplitude was significantly larger under speed instruction than under accuracy instructions for standard stimuli, but not targets. Kutas, McCarthy and Donchin (1977), and Pfefferbaum, Ford, Wenegrat, and Kopell, (1983) also report larger P300

amplitudes under speed instructions. In the Kutas et al. study the P300 amplitude differences were no longer apparent following latency adjustment of the single trial data. It is possible that the P300s recorded in the accuracy condition of their study were therefore diminished by latency jitter (small variation in the peak latency of a waveform). In the Pfefferbaum et al. study, however, P300 amplitude differences withstood latency adjustment. They suggest that P300 may have been larger under speed instructions due to the greater levels of effort, attention or arousal associated with performance in this condition. It is possible that the P300s in the present study were increased under speed instructions due to either a reduction of latency of jitter, or greater effort, attention and arousal, as suggested by Pfefferbaum et al.

Patient P300 amplitudes were significantly smaller than those of controls. The overall mean difference (4.64 μV) which was observed was similar to that reported in earlier studies (Campbell et al, 1986; Curry et al., 1985; Papanicolaou, 1986).

As in the Kutas et al. (1977), and Pfefferbaum et al. (1983) studies, P300 latency remained relatively stable in spite of instructional manipulations. Speed-accuracy tradeoff therefore appears to have little effect on stimulus classification time. In the present study, patient P300 latencies were found to be approximately 60 ms longer than those of controls. This is also consistent with the literature (Campbell et al, 1986; Curry et al., 1985; Papanicolaou, 1986). The 58 ms delay found in P300 patient latencies was much smaller than the 134 ms delay observed in their RTs (for speed and accuracy conditions combined). A substantial

P300-RT interval was thus observed, regardless of the manipulations. The overall 60 ms decrease in (patient and control) RT which occurred under the influence speed instructions was furthermore much less than that observed in other speed-accuracy tradeoff studies. Pfefferbaum et al. for example, report a differences of 235 ms between of speed and accuracy conditions. The purpose of the manipulations employed in this study was to shorten patient RTs to within their P300 peak latencies. There are at least two reasons why this may not have been accomplished. It is possible that patient RTs were delayed due to a motor deficit, in which case they could not easily be shortened to P300 latency. It seems more likely, however, that subjects stressed accuracy in responding, despite instructions to the contrary. Accuracy levels in this study were extremely high. Yellott (1971) notes that when subjects emphasize speed of responding, error rates increase as a result of "fast-guessing". In the present study error rates were extremely low, possibly suggesting more cautious responding. Furthermore, an examination of the strategy employed in individual error trials revealed that errors did not appear to be the result of fast-guessing. In many subjects, particularly patients, RTs on error trials were actually longer than those on the three preceding correct trials. Vickers (1980) has observed that subjects tend to commit "slow errors" (errors associated with slower RTs) when they are stressing accuracy over speed.

It is possible that patients were unable to accurately monitor their speed of responding. The second study in this thesis was therefore designed to provide more stringent control over speed of responding.

Subjects were provided with a time window within which responses were to occur. Furthermore, feedback on speed of responding was also provided.

Table 1.1

Demographic and Neurological Data

ID	age at injury	years educ.	GCS	coma dur.	PTA	GOS	Expl	Exp2	Exp3
JC	31.3	19	4	1	10	1	*	*	4.3
KD	18.9	14	5	14	30	1	3.7	5.7	5.0
LD	19.6	12	5	13	30	1	5.2	5.2	6.5
PD	23.1	10	4	7	60	2	*	3.7	3.7
OE	20.1	17	5	75	—	2	6.9	6.9	*
BG	20.1	15	7	7	25	1	*	*	4.2
BL	20.9	13	6	30	60	1	*	6.5	6.5
JM	18.0	13	5	73	150	2	4.0	4.0	5.3
GM	21.6	15	4	50	121	1	4.8	4.8	6.1
SM	22.5	13	5	30	60	1	3.8	3.8	5.1
HM	45.1	19	5	30	60	2	3.9	3.9	5.2
GP	18.4	12	4	11	30	1	*	5.3	*
LS	19.2	13	5	21	30	1	*	3.1	3.1
MM	22.7	10	4	35	—	3	4.6	4.6	5.9

* = did not participate

Table 1.2

Locations of verified lesions

Patient	Hematomas	Contusions
BL	right frontal	
JM	bilateral subdural	left diffuse
GM		bilateral diffuse
GP	right fronto-temporal	right fronto-temporal
LS		right diffuse
PD	bilateral temporal	
KD	right temporal	
OE		
BG		
HM		

Table 1.3

Percentage Number of Correct Responses and Errors (SDs are in brackets)

Condition		Mean Proportion of Correct Responses			
		Patients		Controls	
		Hits	Correct rejections	Hits	Correct rejections
Speed inst. P=50.50	1	.95	.93	.99	.93
	2	.94	.95	.95	.91
Speed inst. P=70:30	1	.97	.94	.98	.89
	2	.96	.89	.99	.85
Accur. inst. P=50:50	1	.95	.94	.99	.96
	2	.96	.95	.99	.98
Accur. inst. P=70:30	1	.99	.95	.99	.91
	2	.97	.94	.99	.94
		.96	.93	.98	.92

Condition		Mean Proportion of Errors			
		False Alarms	Misses	False Alarms	Misses
Speed inst. P=50:50	1	.07	.05	.07	.01
	2	.05	.06	.09	.05
Speed inst. P=70:30	1	.06	.03	.11	.02
	2	.11	.05	.15	.01
Accur. inst. P=50:50	1	.06	.04	.04	.01
	2	.05	.04	.02	.01
Accur. inst. P=70:30	1	.05	.01	.09	.01
	2	.06	.03	.06	.01
		.07	.03	.08	.02

Table 1.4

Mean reaction times (ms) to standard stimuli (SDs are in parentheses)

	Standard:Target Probability		Patients	Controls
Speed Instructions	P= .50	1	454 (67)	283 (21)
		2	446 (61)	263 (36)
	P= .70	1	395 (55)	241 (34)
		2	444 (92)	236 (17)
Accuracy Instructions	P= .50	1	465 (64)	340 (92)
		2	482 (60)	408 (75)
	P= .70	1	452 (68)	331 (107)
		2	465 (56)	310 (66)

Table 1.5

Mean reaction times (ms) to target stimuli (SDs are in parentheses)

	Standard:Target Probability		Patients	Controls
Speed Instructions	P= .50	1	430 (55)	297 (41)
		2	420 (67)	296 (66)
	P= .30	1	447 (52)	305 (63)
		2	454 (60)	294 (50)
Accuracy Instructions	P= .50	1	457 (37)	356 (99)
		2	490 (56)	424 (72)
	P= .30	1	462 (66)	371 (104)
		2	490 (86)	361 (55)

Table 1.6

Mean P300 amplitudes at Pz (μV) for standard stimuli (SDs are in parentheses)

	Standard:Target Probability		Patients	Controls
Speed Instructions	P= .50	1	4.47 (1.70)	6.69 (4.53)
		2	2.88 (1.58)	7.92 (3.03)
	P= .70	1	1.55 (1.23)	7.00 (3.32)
		2	2.80 (1.71)	7.46 (2.65)
Accuracy Instructions	P= .50	1	4.00 (3.34)	6.39 (2.58)
		2	2.62 (2.45)	5.88 (2.39)
	P= .70	1	2.01 (1.02)	4.77 (2.89)
		2	1.31 (1.18)	5.10 (4.12)

Table 1.7

Mean P300 amplitudes at Pz (μV) for target stimuli (SDs are in parentheses)

	Standard:Target Probability		Patients	Controls
Speed Instructions	P= .50	1	5.11 (2.60)	7.84 (4.90)
		2	4.44 (0.90)	10.84 (5.16)
	P= .30	1	5.00 (2.80)	12.88 (5.84)
		2	5.37 (1.94)	13.21 (6.84)
Accuracy Instructions	P= .50	1	3.43 (.77)	10.21 (4.01)
		2	3.68 (1.60)	5.29 (3.66)
	P= .30	1	7.40 (3.09)	12.09 (5.20)
		2	5.87 (2.74)	12.63 (5.71)

Table 1.8

Mean P300 latency at Pz (ms) for standard stimuli (SDs are in parentheses)

	Standard:Target Probability		Patients	Controls
Speed Instructions	P= .50	1	360 (42)	313 (30)
		2	372 (62)	303 (29)
	P= .70	1	364 (32)	303 (27)
		2	384 (36)	293 (35)
Accuracy Instructions	P= .50	1	366 (45)	327 (52)
		2	363 (42)	307 (30)
	P= .70	1	360 (33)	313 (30)
		2	374 (56)	317 (34)

Table 1.9

Mean P300 latencies at Pz (ms) for target stimuli (SDs are in parentheses)

	Standard:Target Probability		Patients	Controls
Speed Instructions	P= .50	1	351 (36)	307 (35)
		2	349 (36)	310 (24)
	P= .30	1	364 (47)	300 (36)
		2	380 (43)	310 (33)
Accuracy Instructions	P= .50	1	379 (34)	307 (27)
		2	369 (49)	303 (34)
	P= .30	1	369 (34)	317 (56)
		2	366 (30)	307 (27)

Table 1.10

Summary of ANOVAS

	Standards			Targets		
Main Effects	RT	P300 amp	P300 lat	RT	P300 amp	P300 lat
Group	P>C	C>P	P>C	P>C	C>P	P>C
Replication	NS	NS	NS	NS	NS	NS
Probability	.70<.50	.50>.70	NS	NS	.30>.50	NS
Instruction	SP<ACC	SP>AC	NS	SP<ACC	NS	NS
Interaction						
GxR	NS	NS	NS	NS	NS	NS
GxP	NS	NS	NS	NS	NS	NS
GxI	NS	NS	NS	NS	NS	NS
RxP	NS	NS	NS	NS	NS	NS
RxI	NS	NS	NS	NS	NS	NS
PxI	NS	NS	NS	NS	NS	NS

Figure 1.1

ERPs and RT histograms of patients (N = 6) and controls (N = 8) during the speed instruction condition when the standard:target probability was 70:30. Individual subjects have been superimposed.

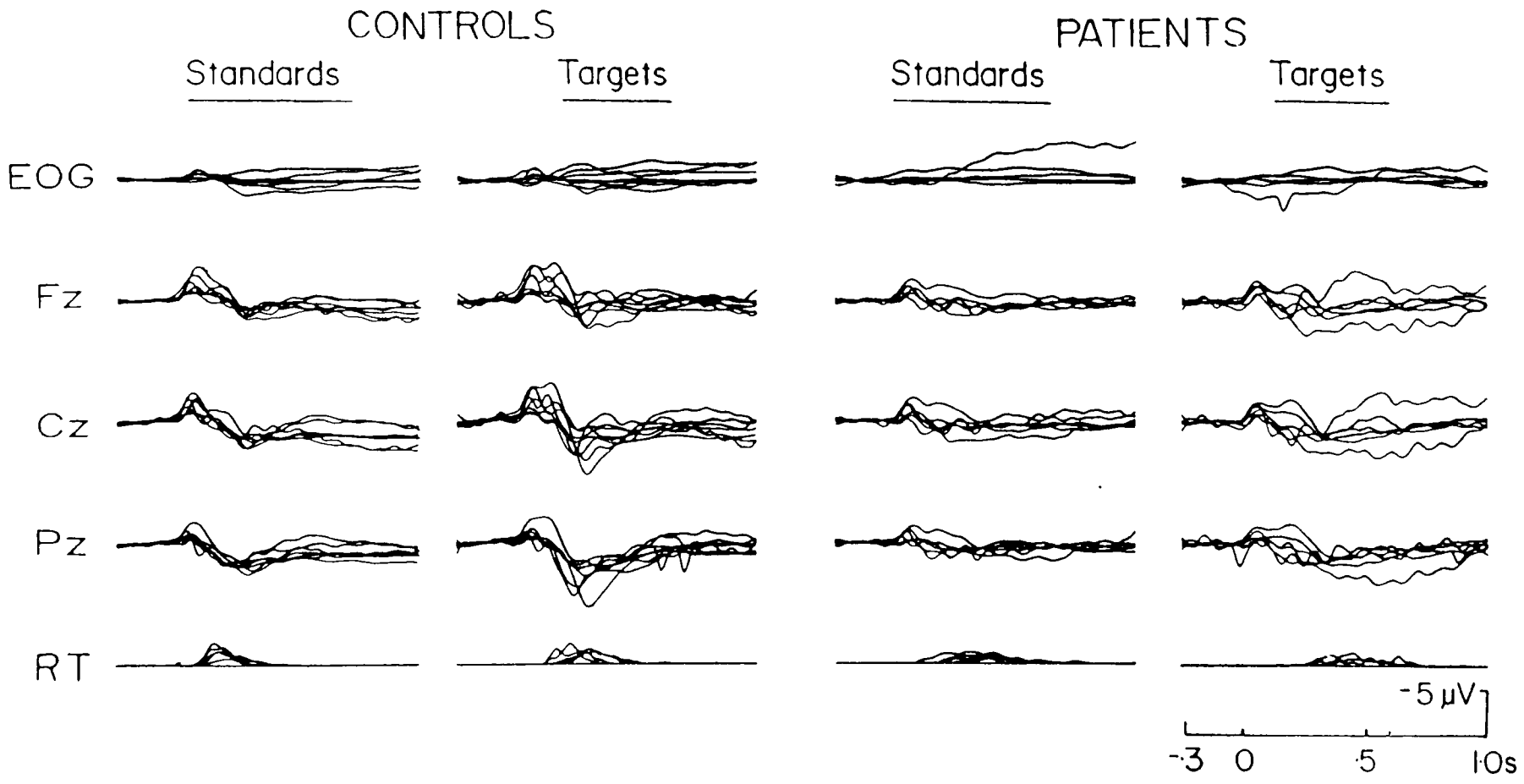

CONTROLS
Standards
Targets
PATIENTS
Standards
Targets
EOG
Fz
Cz
Pz
RT
-5 μV
-.3
0
.5
1.0s

Figure 1.2

Grand average ERPs and RT histograms for patients (N = 6) and controls (N = 6) during the speed instruction condition when standard:target probability was 70:30. Patients are represented by the broken line and controls by the solid line.

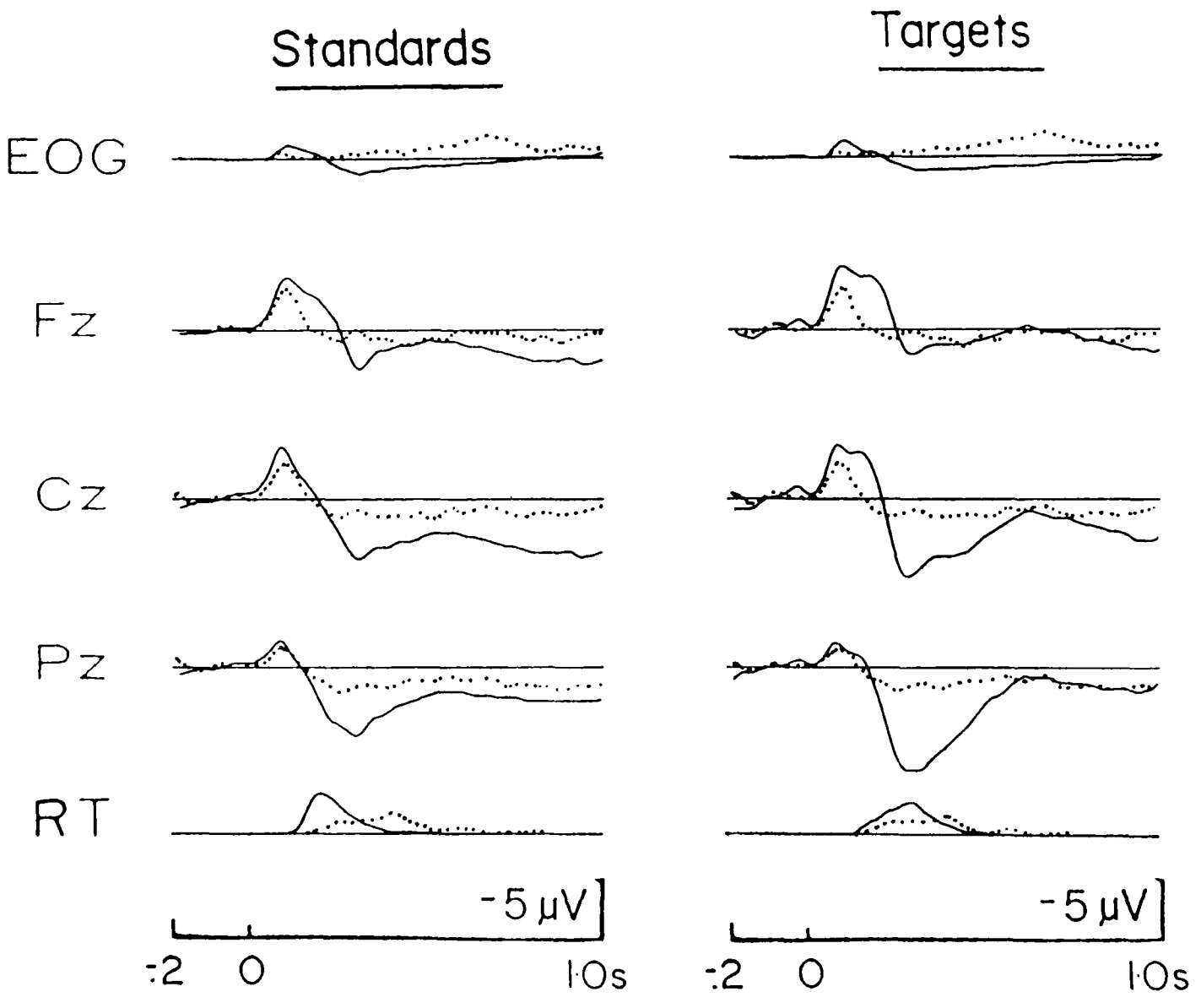

Standards
Targets
EOG
Fz
Cz
Pz
RT
-5 μV
-5 μV
-2
0
1·0s
-2
0
1·0s

Figure 1.3

Grand average ERPs and RT histograms of patients (N = 6) and controls (N = 6) over conditions of speed and accuracy instructions when the standard:target probabilities were 50:50. Patients are represented by the broken line, and controls by the solid line.

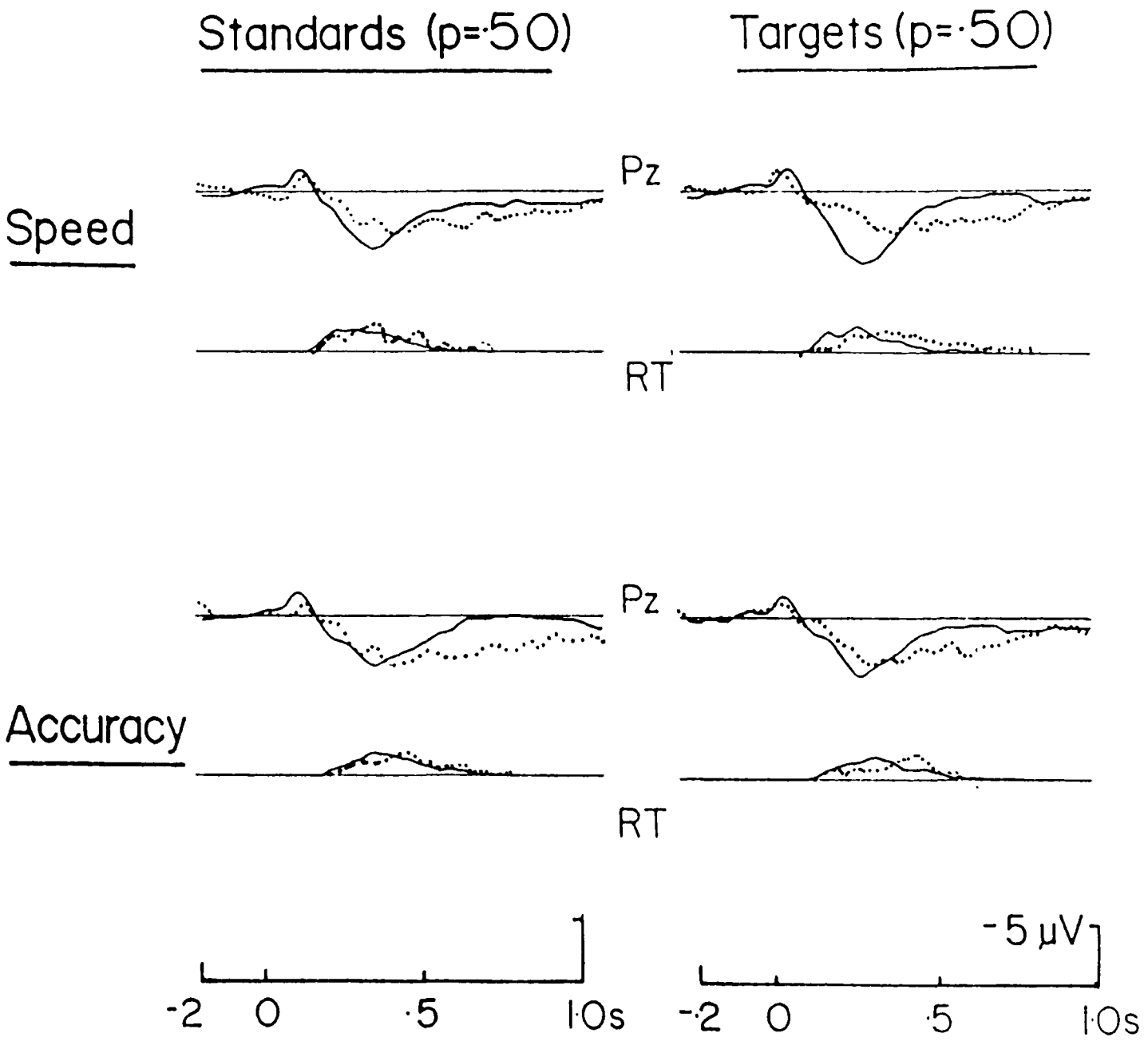
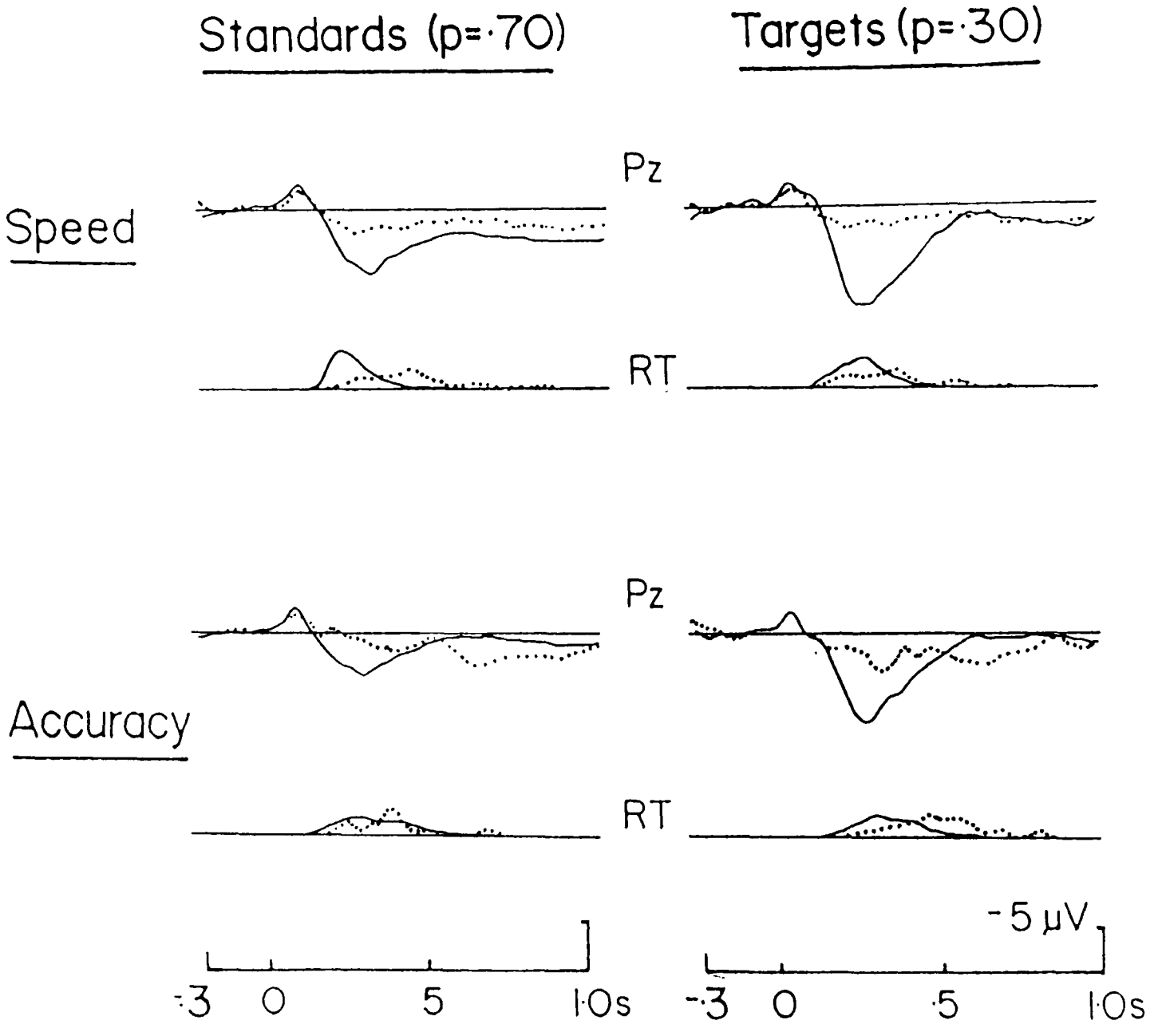

Standards (p=·50)
Targets (p=·50)
Speed
Pz
RT
Accuracy
Pz
RT
-5 μV
-2
0
·5
1·0s
-2
0
·5
1·0s

Figure 1.4

Grand average ERPs and RT histograms of patients (N = 6) and controls (N = 6) over conditions of speed and accuracy instructions when the standard:target probabilities were 70:30. Patients are traced in the broken line and controls in the solid line.


Standards (p=·70)
Targets (p=·30)
Speed
Pz
RT
Accuracy
Pz
RT
-5 μV
-3
0
5
1·0s
-3
0
·5
1·0s

CHAPTER III

Experiment 2

In the first experiment, patient reaction times remained 71 ms later than P300 latency, despite instructions to emphasize speed of responding. In previous physiological studies, speed instructions have been shown to reduce RT to within P300 latency (Kutas McCarthy, & Donchin, 1977; Pfefferbalm, Ford, Wenegrat, Roth, & Kopell, 1983). The absolute reduction in RT was also much less than that observed in these.

Several authors have demonstrated that RT may also be reduced when subjects are provided with time windows within which responses must occur, and/or feedback (FB) on response speed (Fitts, 1966; Pachella, 1974; Pfefferbaum et al., 1983; Shouten & Bekker, 1967; Wood & Jennings, 1976). Winogron (1986) has recently found significant decreases in the RTs of head-injured children when FB and time windows were provided. The RTs of both patients and controls in Winogron's study were nevertheless very long (mean = 738 ms for patients and 650 ms for controls). The residual delay in RT was perhaps due to both the complexity of the task, and the fact that the time windows were all "wide" (1.0, 1.5 and 2.0 s). Physiological measures were not recorded in this study. It is difficult therefore to determine whether patient RTs could have been further decreased. The purpose of the present study was to attempt to shorten patient RTs to their P300 peak latency using FB and more stringent time constraints than those employed by Winogron.

The time windows provided in this study were predetermined time limits within which subjects were required to respond. In "speed" conditions, FB informed subjects of whether their responses had been within the time window or not. In accuracy conditions, FB informed subjects whether their responses corresponded to the stimulus that had been presented. It was expected that RT could be constrained to within the P300 peak latency using these methods.

Methods

Subjects

Subjects were 12 closed head-injured outpatients and 12 matched controls. Eight subjects in each group had participated in the first study. Controls were matched with patients on the basis of gender (8 males and 4 females) age (mean = 23 range = 19 to 46 for patients, mean = 25, range = 20 to 38 for controls), and years of education (mean = 13.4, range = 10 to 19 for patients, mean = 14.4, range = 11 to 21 for controls). The demographic and neurological data for individual patients are presented in Table 2.1. As in the first study, the duration of coma and PTA, and Glasgow Coma Scale ratings were estimated from the information provided by hospital records. The mean duration of coma was estimated to be 32.4 days (range = 7 to 75 days). PTA duration could be estimated in only ten of the twelve patients. The mean PTA duration for these patients was 57.4 days (range = 30 to 150 days). According to the Russell Severity Scale, the patients in the present study would therefore be considered to

have sustained "very severe" injuries. The average Glasgow Coma Scale rating was 4.75 (range = 4 to 6). By the time of testing the average length of time since injury was 4.6 years (range = 3.1 to 6.9 years). The mean Glasgow Outcome Scale rating was 1.5 (range = 1 to 3) (see Table 2.1).

The results of C.T. scans were not available for all patients. As in the first study however, focal lesions were identified in many patients. The location of these and radiologically verified diffuse damage are presented in Table 2.2.

Prior to testing, patients underwent audiometric screening as described in the first study. Control subjects reported having no known audiometric or neurological disturbances.

Subjects were solicited on a volunteer basis and received a $20.00 honorarium for their participation in the study.

Procedure

Subjects performed an auditory discrimination task. The procedure and equipment were essentially identical to that described in Experiment 1. The task again required that one of two buttons be pressed, according to whether a "standard" or "target" tone pip had been detected. In this study, however, subjects were provided with a "window" that defined the time limit within which a response should occur. The window was "narrow" in some conditions and "wide" in others. The duration of the standard and target tone pip defined the width of the window. Windows were 50 ms longer for patients in both the "wide" and "narrow" conditions. Since

this approximated the patient-control P300 latency difference (and theoretically, the difference in stimulus evaluation time), it was expected that the level of accuracy would be the same for the two groups. Subjects also received feedback (FB) concerning their performance. FB was delivered 800 ms after the onset of the auditory stimulus. The illumination of a green light-emitting diode (LED) informed the subject that their response was "correct". A red LED informed the subject that the response was "incorrect". The LED remained on for 580 ms. The LED was mounted in a black case at eye level 1.5 m in front of the subjects. In some conditions FB was based on the speed of the subject's response (i.e., whether the response was within the window or not). In other conditions, FB was based on the accuracy of the subject's response (whether it was in concordance with the stimulus that had been presented). If the subject failed to respond before the onset of FB, incorrect FB was delivered. In actual trials, such occurrences were exceedingly rare. Because of the increase in the inter-trial interval (compared to Experiment 1), the total number of trials per conditions was decreased to 150. To maintain a satisfactory signal:noise ratio for the standard and target waveforms, the probability of target occurrence was held constant at .40 in all conditions. The inter-trial interval (time between onset of standard or target stimuli) was 3 sec.

The following conditions were run: Speed Feedback - Narrow Time Window (SFB-NW): In this condition, the duration of the stimulus (the "window") was 300 ms for controls and 350 ms for patients. Instructions to subjects stressed that correctness was based on the response being

within the window, and not on accuracy (whether the correct button had been pressed). FB was not provided concerning accuracy. Correct FB thus informed the subject that the response was within the window. Incorrect FB informed subjects that their response was too slow (i.e., outside of the window).

Accuracy Feedback, Narrow Time Window (AFB-NW): Subjects were instructed to attempt to respond within the duration of the stimulus (i.e., within the time window). Stimulus duration was 300 ms for controls and 350 ms for patients. FB was provided on the basis of accuracy. "Correct" FB indicated that the appropriate button had been pressed, "incorrect" FB that the wrong button had been pressed. No FB was provided concerning the actual speed of responding (i.e., whether it was within the window or not).

Speed Feedback, Wide Time Window (SFB-WW): A wider time interval for responding was used in this condition. It was set at 450 ms for controls and 500 ms for patients. In every other respect however, this condition was identical to that of condition 1. The three conditions were run randomly and replicated once in reverse order.

Recording Procedure

Behavioural and physiological data were recorded using the equipment described in Experiment 1. In all conditions trials were sorted according to stimulus type and accuracy of responding (whether the appropriate response had been made). Trials in which a response was not made within the sweep were automatically rejected. Signal averaging was modified

however to permit the recording of the response to FB (for a discussion of this issue, see Deacon-Elliott & Campbell, 1987). EEG signals were therefore recorded over a 1800 ms sweep beginning at 300 ms prior to stimulus onset and continuing for another 1500 ms following it. Sampling occurred every 6 ms (i.e., a total of 300 points were sampled). The physiological data were scored according to the procedures described in experiment 1.

Statistical Analysis

Reaction time, d', P300 amplitude and P300 latency were submitted to a three-way analysis of variance (ANOVA) with a single "between" factor, Group (patients vs controls), and two "within" factors, Replications (first vs second) and Instruction (SFB-NW vs AFB-NW vs SFB-WW). A level of significance $p < .05$ was adopted. When appropriate, Greenhouse-Geisser (Greenhouse & Geisser, 1959) correction procedures were employed. When a significant conditions effect was observed, a Tukey post-hoc procedure was applied to determine differences among the means.

Results

Behavioural Data

Accuracy was somewhat lower than in the first experiment. Collapsing across conditions and stimuli, patients detected correctly 87% and controls 86% of the stimuli. The mean proportion of errors and correct responses during each condition are presented in Table 2.3.

Sensitivity, as indexed by d', was significantly influenced by the experimental manipulations ($F(2,38) = 16.19$, $p < .01$). The mean d's (see Table 2.4) were 1.69 (SD = 1.18) in the SFB-NW condition, 2.77 (SD = 1.33) in the AFB-NW condition, and 2.59 (SD = 4.74) in the SFB-WW condition. Post-hoc testing indicated that d' was significantly lower in SFB-NW than in AFB-NW or SFB-WW. There were no significant differences in d' between patients and controls ($F(1,19) = < 1$).

Despite the fact that the error rate increased there were again an insufficient number of errors to permit meaningful analysis of either the RT or evoked potential data on these trials. Results will therefore be presented for trials on which stimuli were correctly detected.

Mean RTs for the standard stimulus are presented in Table 2.5. For these stimuli, there was a tendency for RTs to be shorter on the second (replication) trial of each condition (mean = 293 ms, SD = 33 ms) than on the first (mean = 315 ms, SD = 47 ms). The effects of replication, however failed to attain significance ($F(1,18) = 4.09$, $p < .06$). Significant main effects of condition were observed ($F(2,36) = 5.07$, $p < .01$). Post-hoc testing indicated that RT was significantly shorter in SFB-NW (mean = 277 ms, SD = 72 ms) and SFB-WW (mean = 305 ms, SD = 53 ms) than in the accuracy condition (mean = 332, SD = 57 ms). Significant group differences were also observed ($F(1,18) = 72.24$, $p < .01$). Patient RTs (mean = 355, SD = 81 ms) were significantly longer than those of controls (mean = 253, SD = 60 ms). The condition by group interaction was not significant.

Reaction times to target stimuli are presented in Table 2.6. They did not vary significantly over replications ($F(1,18) = 1.38$). Significant differences were observed, however, among experimental conditions ($F(1,18) = 24.02$, $p < .01$). Post-hoc testing identified significant differences between the SFB-NW condition (mean = 280 ms, SD = 61 ms) and the AFB-NW condition (mean = 336 ms, SD = 75 ms). Significant group differences were also observed ($F(2,36) = 6.17$, $p < .01$). The RTs of patients (359 ms, SD = 93 ms) were significantly longer than those of controls (264 ms, SD = 43 ms). The condition group interaction was not significant.

Physiological Data

Figure 2.1 illustrates the superimposition of all patient and all control ERPs and RT histograms for the narrow window, accuracy condition. Figure 2.2 presents the grand averages of these waveforms. As may be observed the grand average, in general, is typical of the group response. P300 is largest at Pz and decreases in amplitude in frontal regions. Figure 2.3 illustrates the grand average of the "full" sweep period (from the pre-stimulus period to the end of FB) for the same condition. The frontal, and to a lesser extent, central P300 appear to be affected by a slow CNV-like waveform that develops in preparation for FB. Figure 2.4 presents the ERPs and RT histograms across all conditions.

Table 2.7 presents the P300 amplitude data for the standard stimuli. P300 amplitude did not vary significantly across replications or conditions ($F < 1$). Control P300s (mean = 7.71 μV, SD = 3.94 μV) were however

significantly larger than those of patients (mean = 4.43 μV, SD = 4.42 μV) ($F(1,17) = 5.02$, $p < .05$). No significant interactions were apparent.

For target stimuli (Table 2.8) significant differences were observed among conditions ($F(2,26) = 3.46$, $p < .05$). P300 was significantly larger in the SFB-WW (mean = 6.48 μV, SD = 3.66 μV) condition than in the others (mean = 5.95 μV, SD = 4.88 μV and 5.78 μV, SD = 4.07 μV) for SFB-NW and AFB-NW respectively. P300 following targets was significantly larger for controls (mean = 11.37 μV, SD = 3.79) than for patients (mean = 6.53 μV, SD = 4.08) ($F(1,13) = 10.92$, $p < .01$).

The latencies of P300s evoked by standard tones (see Table 2.9) did not vary significantly between replications ($F(1,17) = 3.95$) or among conditions ($F(2,34) = 1.93$). Significant group differences were observed ($F(1,17) = 9.63$, $p < .01$). Patient P300 latencies (mean = 344 ms, SD = 48 ms) were found to be significantly longer than those of controls (mean = 293 ms, SD = 32 ms).

The latency of P300 following target (see Table 2.10) stimuli did not significantly vary between replications ($F(1,16) = 3.77$, $p < .07$) or among conditions ($F(2,36) = 2.81$, $p < .07$). Between group differences were again significant ($F(1,16) = 9.61$, $p < .01$). The mean P300 latencies were 336 ms (SD = 52) for patients and 284 ms (SD = 30) for controls.

Discussion

Patient RTs were reduced by an average of approximately 100 ms relative to the accuracy condition of the first study. Neither the

percentage of correct responses, nor d' varied significantly between groups. The reduction of patient RTs thus appears to have been accomplished with no greater cost in accuracy than for controls.

Despite the reduction of patient RTs, they remained approximately 100 ms longer than those of controls. On the other hand, patient P300 latencies were delayed by an average of only 52 ms compared to controls. Overall it would appear that patient RTs continued to be somewhat longer than their P300 latencies. Notable differences were observed, however within the experimental conditions. In some conditions, RT was later than P300, whereas in others it preceded it. The exaggeration of RT was most pronounced in the accuracy FB condition. In this condition P300 latency was an average of 56 ms earlier than RT. The provision of accuracy feedback may therefore have counterbalanced the effects of the narrow time window, thereby maintaining an emphasis on accuracy.

The shortest RTs were recorded in the SFB-NW condition. Patient RTs in this condition occurred at about the same time as the peak of P300. This was observed despite a 20-30 ms decrease in P300 latency, relative to the first study. The P300 latency shift is comparable to that described by Kutas et al. and Pfefferbaulm et al. under similar speed instruction influences. The decreases in RT and P300 latency were not likely due to the spontaneous recovery of patients. In the accuracy condition, patient RTs still remained longer than P300 latency and were 126 ms longer than the RTs of controls.

P300 amplitude was largest when speed FB and a wide criterion window were provided. In other studies P300 amplitude was greater when speed of

responding, as opposed to accuracy was stressed (Kutas, McCarthy, & Donchin, 1977; Pfefferbaum, Ford, Wenegrat, & Kopell, 1983). The mechanism by which speed instructions enhanced P300 in these earlier studies was not clear. In the Kutas et al. study, the P300 amplitude differences under speed and accuracy conditions were removed by latency adjustment. This would indicate that speed instructions decreased "latency jitter" in their study, thereby decreasing, on average the peak amplitude of P300. In the Pfefferbaum et al. study, P300 amplitude differences withstood latency adjustment. As discussed in the first study, these authors suggested that factors such as the subjects level of attention, arousal, or the effort expended under the two condition might better account for the observed P300 amplitude differences.

It is therefore curious that in the present study, P300 was not largest in the SFB-NW condition where RT was shortest. Not only was the emphasis on speed greatest in this condition, but attention, arousal and effort could be assumed to be greater as well. One explanation may be that the extreme emphasis on speed in this condition resulted in subjects being less certain of their decisions. As mentioned in the general discussion, Ruchkin and Sutton (1981) have observed that P300 may be attenuated by equivocation. The smaller d's observed in this condition would support this suggestion.[1]

[1] The decrease in d' was probably due to an increase in the background noise. In speed conditions, subjects were forced to respond before sufficient evidence (derived from the signal) had accumulated (see Vickers, 1980). It should be noted that the alteration of the variation noise distribution is a violation of the assumptions of signal detection theory (Green & Swets, 1966). As such, the use of d' as a measure of discriminability can be questioned.

The results of this study suggests that if patients are provided with a means to monitor their speed of responding and/or feedback is provided, RT can be reduced. Furthermore the fact that in speed conditions, RT can equal P3 latency suggests that the delay in RT is not a consequence of residual motor deficits. The slowness of responding observed in so many studies of the head-injured is not "permanent". It is very much dependent on the experimental methods employed and is subject to manipulation. The third experiment will attempt to examine possible carry over effects. Will RT continue to be constrained to the time when stimulus evaluation is complete after time windows and FB are removed?

Table 2.1

Demographic and Neurological Data

ID	age at injury	years educ.	GCS	coma dur.	PTA	GOS	Expl	Exp2	Exp3
JC	31.3	19	4	1	10	1	*	*	4.3
KD	18.9	14	5	14	30	1	3.7	5.7	5.0
LD	19.6	12	5	13	30	1	5.2	5.2	6.5
PD	23.1	10	4	7	60	2	*	3.7	3.7
CE	20.1	17	5	75	—	2	6.9	6.9	*
BG	20.1	15	7	7	25	1	*	*	4.2
BL	20.9	13	6	30	60	1	*	6.5	6.5
JM	18.0	13	5	73	150	2	4.0	4.0	5.3
GM	21.6	15	4	50	121	1	4.8	4.8	6.1
SM	22.5	13	5	30	60	1	3.8	3.8	5.1
HM	45.1	19	5	30	60	2	3.9	3.9	5.2
GP	18.4	12	4	11	30	1	*	5.3	*
LS	19.2	13	5	21	30	1	*	3.1	3.1
MM	22.7	10	4	35	—	3	4.6	4.6	5.9

* = did not participate

Table 2.2

Locations of verified lesions

Patient	Hematomas	Contusions
BL	right frontal	
JM	bilateral subdural	left diffuse
GM		bilateral diffuse
GP	right fronto-temporal	right fronto-temporal
LS		right diffuse
PD	bilateral temporal	
KD	right temporal	
OE		
BG		
HM		

Table 2.3

Number of correct responses (SDs are in parentheses)

		Mean Proportion of Correct Responses			
Condition		Patients		Controls	
		Hits	Correct rejections	Hits	Correct rejections
Speed FB	1	.84	.77	.79	.67
Narrow win	2	.86	.73	.87	.73
Accuracy FB	1	.93	.87	.94	.81
Narrow win	2	.95	.88	.94	.86
Speed FB	1	.85	.84	.90	.89
Wide win	2	.89	.79	.93	.81
		.89	.85	.91	.80

		Mean Proportion of Errors			
Condition		False Alarms	Misses	False Alarms	Misses
Speed FB	1	.23	.16	.33	.21
Narrow win	2	.27	.14	.27	.13
Accuracy FB	1	.13	.07	.19	.06
Narrow win	2	.12	.05	.14	.06
Speed FB	1	.16	.15	.11	.10
Wide win	2	.21	.11	.19	.07
		.15	.11	.20	.09

Table 2.4

d'

Condition	replication	patients	controls
Speed FB	1	1.79 (1.45)	1.76 (1.21)
Narrow window	2	1.64 (1.17)	1.55 (0.87)
Accuracy FB	1	2.58 (1.49)	2.55 (1.28)
Narrow window	2	3.31 (1.20)	2.64 (1.36)
Speed FB	1	2.71 (0.94)	2.37 (1.10)
Wide window	2	2.91 (1.28)	2.38 (1.43)

Table 2.5

Mean reaction times (ms) to standard stimuli (SDs are in parentheses)

Condition	Replication	Patients	Controls
Speed Feedback	1	360 (83)	234 (55)
narrow window	2	278 (114)	233 (34)
Accuracy Feedback	1	399 (76)	272 (56)
narrow window	2	392 (69)	261 (27)
Speed Feedback	1	359 (94)	265 (31)
wide window	2	341 (49)	252 (36)

Table 2.6

Mean reaction times (ms) to target stimuli (SDs are in parentheses)

Condition	Replication	Patients	Controls
Speed Feedback	1	350 (73)	242 (27)
300 ms window	2	296 (121)	231 (21)
Accuracy Feedback	1	370 (139)	278 (56)
300 ms window	2	406 (71)	289 (32)
Speed Feedback	1	381 (86)	280 (36)
300 ms window	2	352 (66)	268 (41)

Table 2.7

Mean P300 amplitudes (μV) for standard stimuli (SDs are in parentheses)

Condition	Replication	Patients	Controls
Speed Feedback narrow window	1	4.95 (4.64)	6.01 (6.61)
	2	4.76 (4.57)	8.06 (3.68)
Accuracy Feedback narrow window	1	3.01 (4.48)	8.53 (3.91)
	2	3.85 (4.96)	7.74 (2.68)
Speed Feedback wide window	1	5.33 (4.38)	7.17 (3.36)
	2	4.66 (3.48)	8.77 (3.40)

Table 2.8

Mean P300 amplitude (uV) for target stimuli (SDs are in parentheses)

Condition	Replication	Patients	Controls
Speed Feedback	1	6.89 (4.70)	10.85 (6.00)
narrow window	2	4.94 (4.46)	10.32 (3.72)
Accuracy Feedback	1	5.79 (4.09)	11.39 (1.84)
narrow window	2	5.89 (3.77)	10.66 (4.09)
Speed Feedback	1	8.70 (5.08)	12.85 (4.34)
wide window	2	7.00 (2.36)	12.14 (2.73)

Table 2.9

Mean P300 latencies (ms) for standard stimuli (SDs are in parentheses)

Condition	Replication	Patients	Controls
Speed Feedback narrow window	1	338 (37)	280 (22)
	2	333 (41)	297 (30)
Accuracy Feedback narrow window	1	353 (59)	294 (29)
	2	344 (54)	299 (43)
Speed Feedback wide window	1	350 (48)	293 (33)
	2	345 (48)	293 (35)

Table 2.10

Mean P300 latencies (ms) for target stimuli (SDs are in parentheses)

Condition	Replication	Patients	Controls
Speed Feedback narrow window	1	325 (26)	282 (29)
	2	342 (54)	308 (31)
Accuracy Feedback narrow window	1	314 (55)	281 (31)
	2	332 (50)	256 (26)
Speed Feedback wide window	1	362 (89)	282 (27)
	2	338 (33)	294 (37)

Table 2.11

Summary of ANOVAs

		Standards			Target	
	d'	RT	P300 amp	P300 lat	RT	P300 amp	P300 lat
Main Effects							
Group	NS	P>C	C>P	P>C	P>C	C>P	P>C
Replication	NS	NS	NS	NS	NS	NS	NS
Condition	SFB-NW< AFB-NW +SFB-WW	SFB-NW +SFB-WW <AFB-NW	NS	NS	SFB-NW< AFB-NW	SFB-WW> AFB-NW +SFB-NW	NS
Interactions							
GxR	NS	NS	NS	NS	NS	NS	NS
GxC	NS	NS	NS	NS	NS	NS	NS
CxR	NS	NS	NS	NS	NS	NS	NS

Figure 2.1

ERPs and RT histograms for the accuracy feedback, narrow window condition. Individual patient (N = 10) and control (N = 11) data have been superimposed.

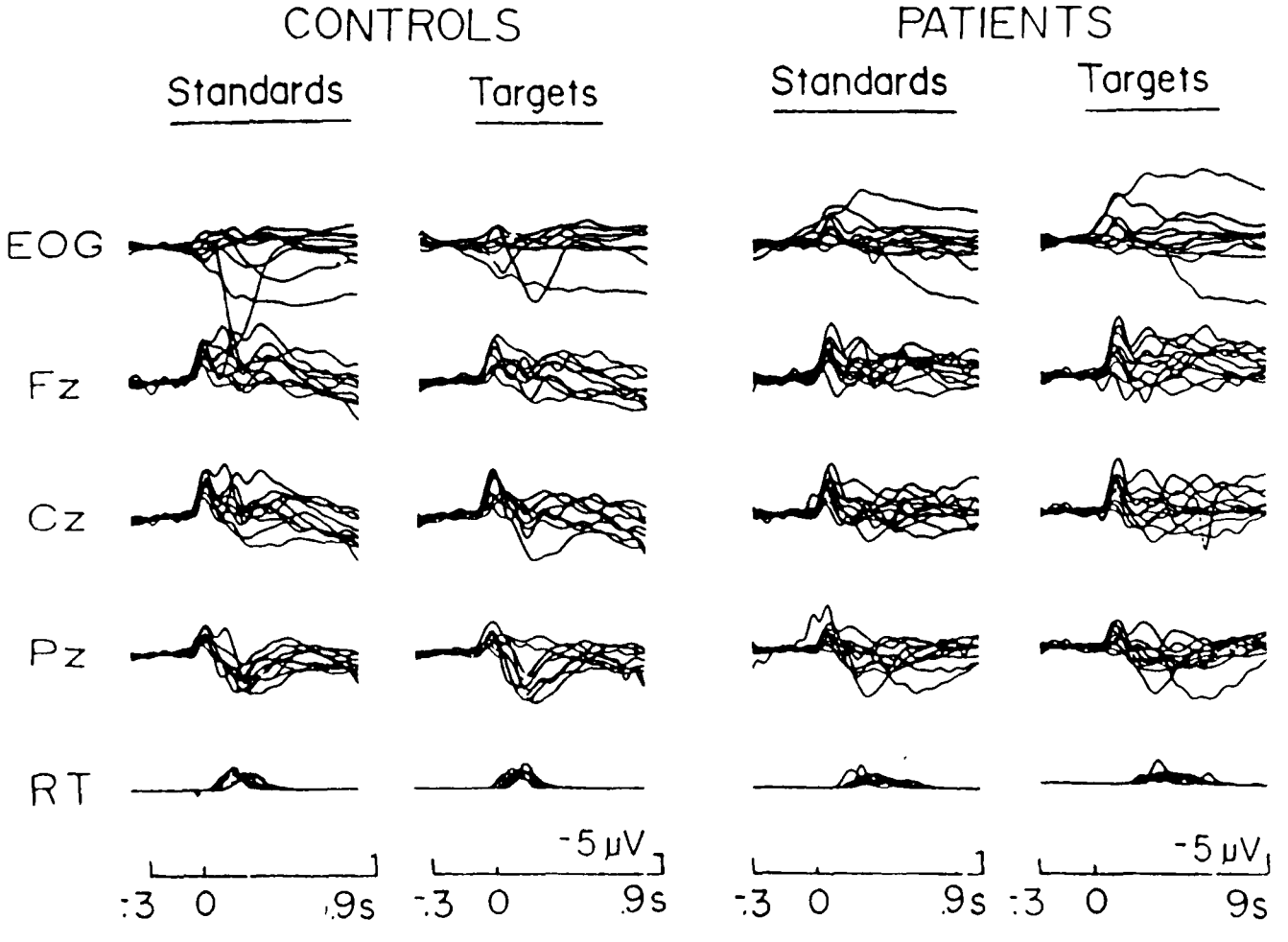

CONTROLS
PATIENTS
Standards
Targets
Standards
Targets
EOG
Fz
Cz
Pz
RT
-5 μV
-5 μV
-.3 0 .9s
-.3 0 .9s
-.3 0 .9s
-.3 0 .9s

Figure 2.2

Grand average of patients (N = 11) and controls (N = 10) in the accuracy feedback narrow window condition. Patients are represented by the thick line and controls by the thin line.

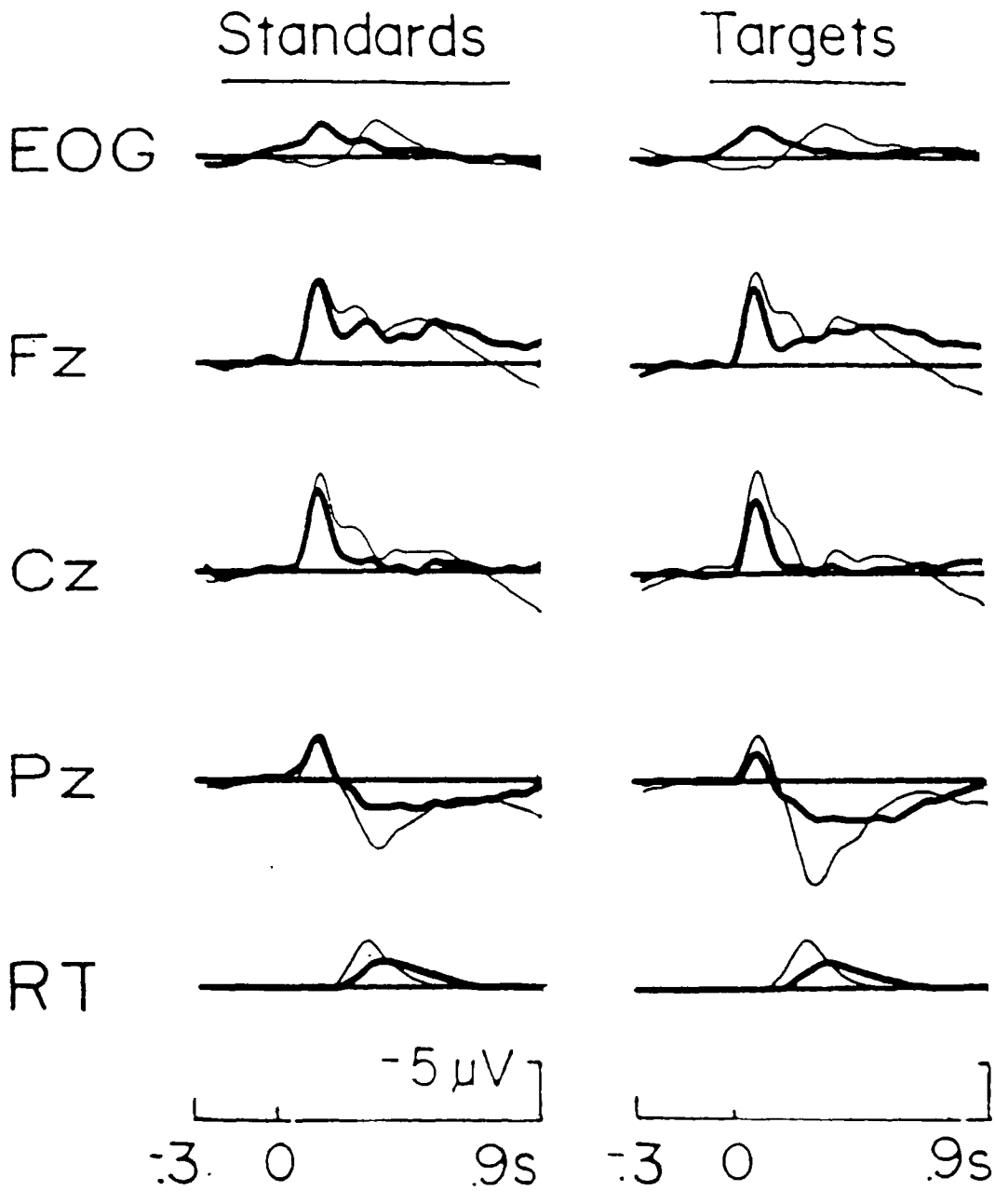

Standards
Targets
EOG
Fz
Cz
Pz
RT
-5 μV
-3
0
.9s
-3
0
.9s

Figure 2.3

Grand average of the complete sweep, including the delivery of FB for the accuracy condition. Patients are represented by the broken line and controls by the solid line.

FB: Accuracy (narrow window)

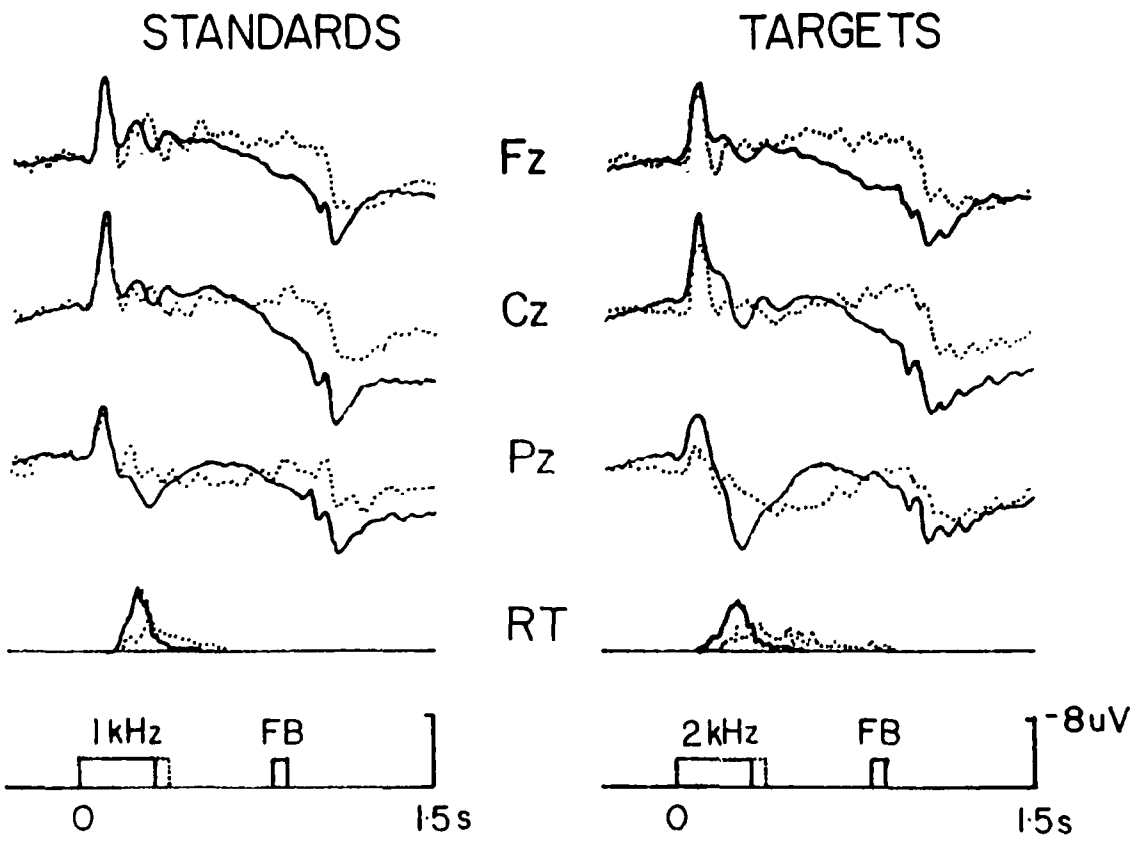


Figure 2.4

Grand average ERPs and RT histograms for patients (N = 11) and controls (N = 10) across conditions. Patients ERPs are represented by the thick line, and those of controls by the thin line.

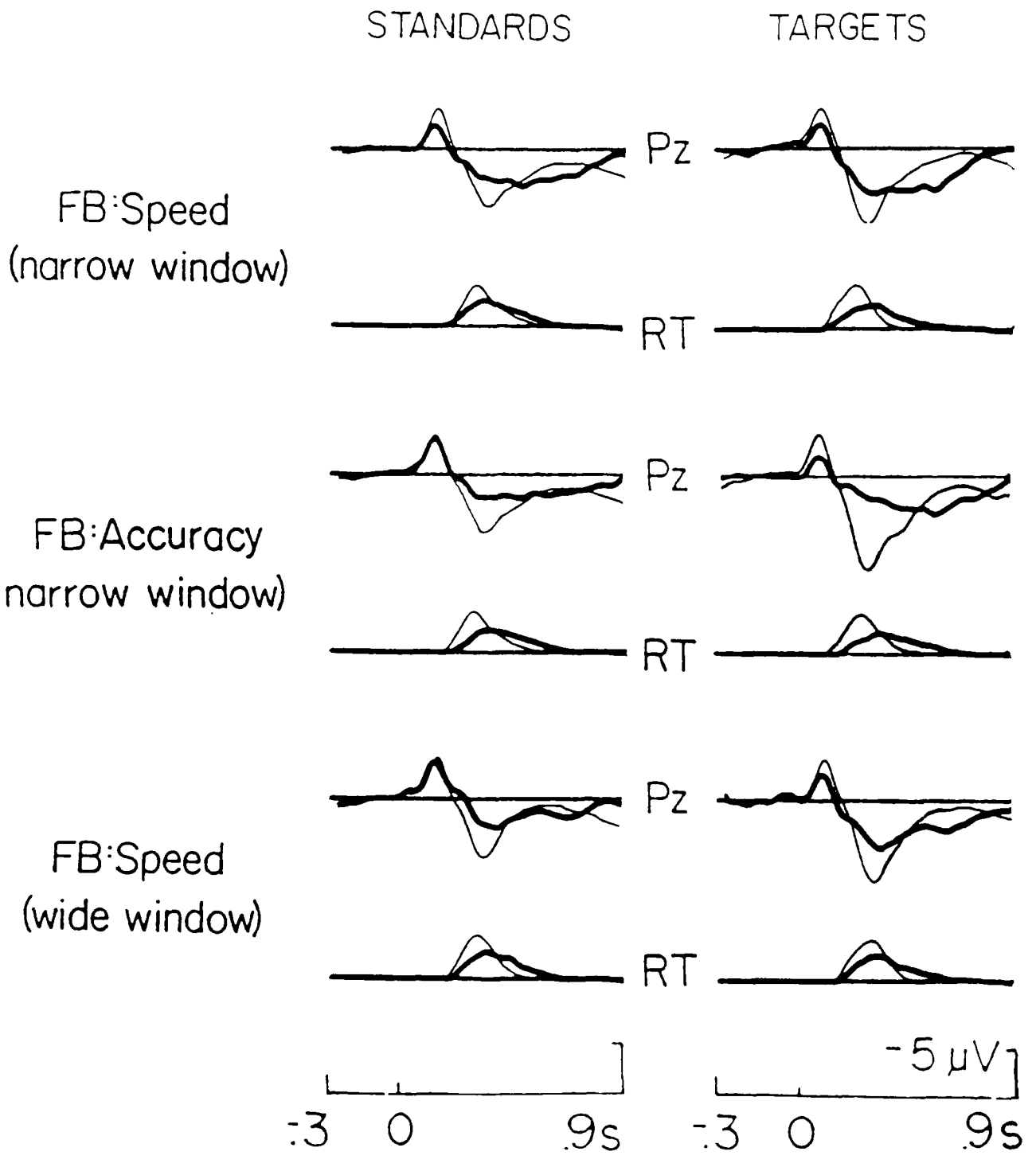
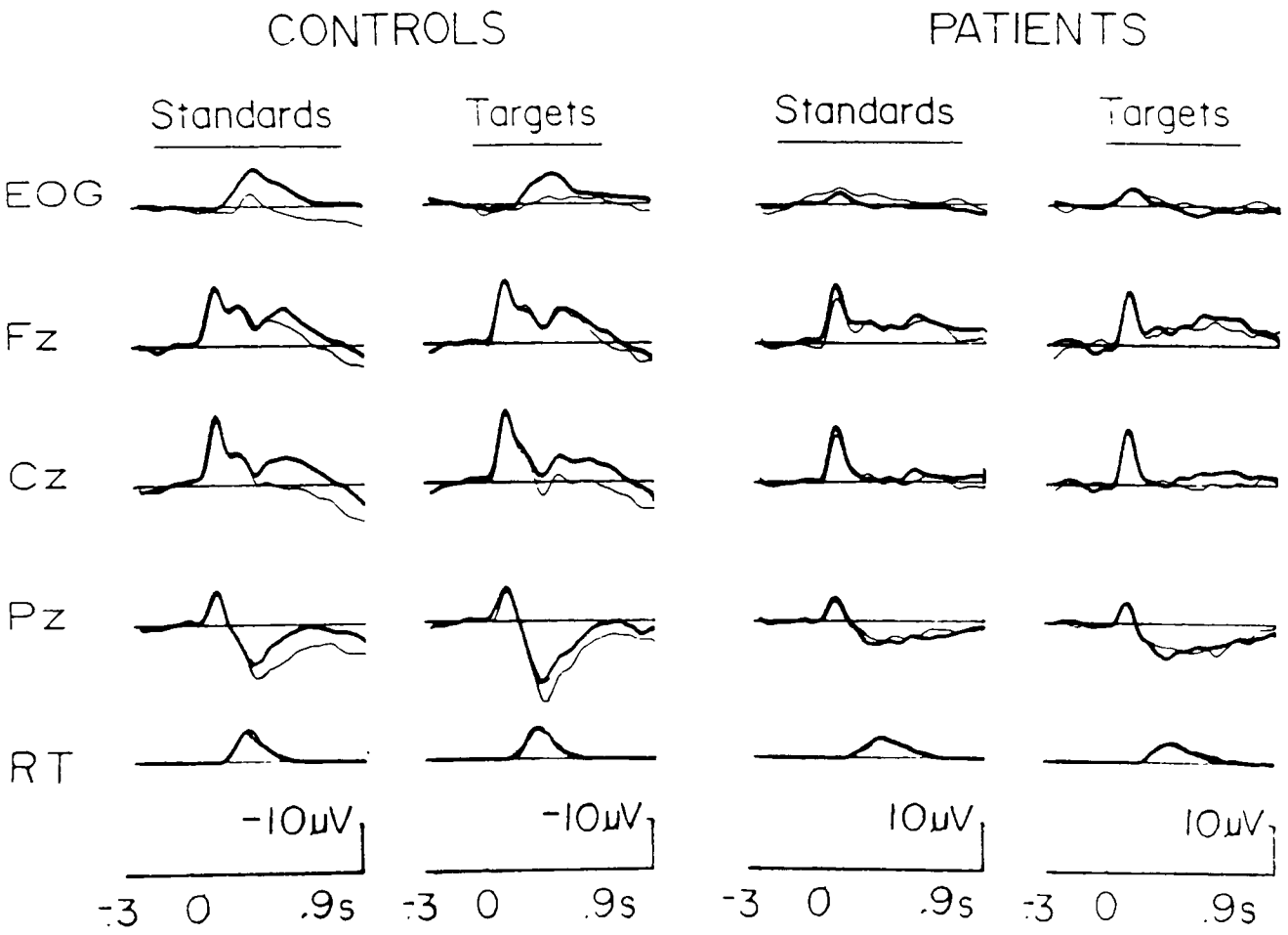

STANDARDS
TARGETS
FB:Speed
(narrow window)
Pz
RT
FB:Accuracy
narrow window)
Pz
RT
FB:Speed
(wide window)
Pz
RT
-5 μV
-.3
0
.9s
-.3
0
.9s

Figure 2.5

Grand averages of patients (N = 11) and controls (N = 10) during the first and second presentation of the accuracy condition. ERPs recorded during the first replication are traced in the thick line whereas those recorded during the second are traced in the thin line.


CONTROLS
PATIENTS
Standards
Targets
Standards
Targets
EOG
Fz
Cz
Pz
RT
-10μV
-10μV
10μV
10μV
-3 0 .9s
-3 0 .9s
-3 0 .9s
-3 0 .9s

CHAPTER IV

Experiment 3

In the second experiment, the reaction times of CHI patients were reduced through the use of time windows and feedback (FB) informing subjects of their speed of responding. The greatest reduction of patient RT was observed during trials in which both a "narrow" time window and speed FB were provided.

The third experiment will examine "carry-over" effects. If RT can be reduced through the use of a time window and FB, will subjects maintain speeded performance after removal of these cues?

Methods

Subjects

Subjects were 12 closed head-injured outpatients and 12 matched controls. Eleven of the patients had participated in at least one of the preceding studies. Controls were again matched with patients on the basis of gender (8 males and 4 females, in both groups) age (mean = 29, range = 22 to 50 for patients, mean = 26, range = 20 to 38 for controls) and years of education (mean = 14, range = 10 to 19 for patients, mean = 15, range = 11 to 21 for controls). The demographic and neurological data for individual patients are presented in Table 3.1. As in the preceding studies, the duration of coma and PTA, and Glasgow Coma Scale ratings were estimated from the information provided by hospital records. The mean coma duration was 26 days (range = 1 to 50 days). PTA duration could be

estimated in only 11 of the 12 patients. The mean duration of PTA for these patients was 53 days (range = 10 to 150 days). According to the Russell Severity Scale, all of the patients who participated in this study would be considered to have sustained severe to very severe injuries. The mean Glosgow Coma Scale rating was 4.9 (range = 4 to 7 days). As mentioned in the first study, an overall rating of eight or less on this scale is associated with poor prognosis. By the time of testing, the mean length of time since injury was 5.1 years (range = 3.1 to 6.5 years). The mean score on the Glasgow Outcome Scale, which was completed approximately 18 months prior to testing was 1.4 (range = 1 to 3). This rating corresponds to a level of recovery in which the patient is capable of performing activities of daily living but is not self-sufficient (see Table 3.1).

The results of CT scans were available for eight of the 12 patients. Focal lesions were identified in some of these patients. The location of these, as well as radiologically identified diffuse damage is presented in Table 3.2.

Prior to testing, patients underwent audiometric screening as described in the first study. Control subjects reported having no known audiometric or neurological disorders. Subjects were solicited on a volunteer basis and received a $20.00 honorarium for their participation in the study.

Procedure

Stimuli were 80 dB SPL 1000 "standard" and 2000 Hz "target" tone pips. The duration of stimuli varied between conditions. The rise-and-fall time was 5 ms. The stimuli were delivered through Telephonics TDH39 headphones at a rate of 1/2.5 sec. With the exception of one subject with unilateral hearing loss, tones were presented monaurally to the right ear. Standard and targets were delivered randomly with equal probability of occurrence. The Cromemco Z-2 microcomputer again controlled the timing and random sequencing of the stimuli. Each condition consisted of a train of 200 stimuli.

The testing situation was identical to that described in the second experiment. The subject's task was to press one of two buttons, depending upon whether the 1000 or 2000 Hz tone pip had been detected. Subjects were instructed to respond as quickly as possible, even if this involved making errors.

In some conditions, a criterion window provided subjects with a "time limit" within which responses were to occur. In other conditions, no window was provided. Furthermore, in some conditions subjects received FB which informed them of whether the response was within the time limit. In other conditions, FB was absent.

In conditions in which stimulus duration was used as a criterion window, it was set at 300 ms for controls and 350 ms for patients (in the previous experiments, patient P300s were at least 50 ms longer than those of controls, suggesting a possible difference in stimulus evaluation time of this magnitude.). In conditions in which no criterion window was

provided, stimulus duration was 50 ms. Since accurate detection could not possibly occur within this time period, stimulus duration could not serve as a time window.

Feedback was provided by one of two light emitting diodes (LEDs), which were illuminated 780 ms after the onset of the auditory stimulus. The duration of the LED was 580 ms. A green LED indicated that the response had occurred within the time limit (300 ms for controls, 350 ms for patients) even when a criterion window had not been provided. A red LED indicated that the response had occurred outside of the time limit. In conditions in which FB was not provided, the red LED was illuminated, regardless of the speed of responding. The principle aim of this study was to examine possible carry-over effects of the provision of a time window and FB. For this reason, conditions were presented in a specific invariant order.

Conditions were run in the following order:

1) Win - FB-. Neither a criterion window nor FB were provided. The absence of either a time window or FB thus formed a baseline for comparison to subsequent conditions.
2) Win + FB-. The time limit for responding was established by the duration of the stimulus but no FB was provided.
3) Win - FB+. A criterion window was not provided but FB was delivered which informed subjects of whether the response had occurred within the time limit.
4) Win + FB+. Both a criterion window and FB were provided.

Prior to each trial, subjects were told whether FB or criterion windows would be provided.

It was expected that the beneficial effects of training with FB and a time window might continue once these cues were removed. The four conditions were therefore run again but in reverse order. Thus, on the last presentation of conditions, subjects received neither feedback nor a criterion window. This allowed carry-over to be assessed by comparing the two replications of the Win - FB- conditions (before and after training).

Prior to testing, a practice trial of 100 stimuli was given in order to familiarize subjects with the task. The recording procedures and scoring of the physiological data were identical to those described in the second study.

Statistical analysis. RT, d', and P300 amplitude and latency were submitted to a three-way analysis of variance (ANOVA) with a single "between" factor, Group (patients vs controls) and two "within" factors, Replications (first vs second) and Condition (FB - Win-, FB - Win+, FB + Win-, FB + Win+). Results were considered significant at $p < .05$. Greenhouse Geisser (Greenhouse & Geisser, 1959) correction procedures were employed where applicable. When a significant effect of Condition was observed, a Tukey post-hoc test was applied to determine differences amongst the means.

Results

Behavioural data

The number of correct responses and errors committed for each stimulus is presented in Table 3.3. Collapsing across conditions and stimuli, the percentage of correct responses was 84% for patients and 88% for controls. Significant main effects for the Condition factor were found for d' ($F(3,36) = 12.02$, $p < .01$). It was highest when neither a time window nor FB was presented (mean = 3.19, SD = 1.13). The mean d's were 2.27 (SD = 1.40) for patients and 2.16 (SD = 1.28) for controls ($F<1$). (See Table 3.4).

The mean RTs to standard stimuli are presented in Table 3.5. A replication x condition interaction tended towards significance ($F(3,51) = 2.75$, $p < .07$). RT decreased by 44 ms on the replication of FB - Win- and by 38 ms on replication of FB - Win+. Significant main effects were found for Condition ($F(3,51) = 8.77$, $p < .01$). Simple main effects testing indicated that RT was significantly shorter when FB was provided alone or with a time window, than when no cues to speed were provided. Collapsing across conditions and replications, RT to standards was significantly longer for patients (mean = 373, SD = 97 ms) than for controls (mean = 287, SD = 50 ms) ($F(1,17) = 8.74$, $p < .01$).

The mean RTs for targets tones are presented in Table 3.6. RT decreased significantly on replications being, on average 26 ms shorter on these trials ($F(1,17) = 6.10$, $p = .02$). Significant differences were also observed between conditions ($F(3,51) = 9.28$, $p < .01$). Simple main

effects testing indicated that RT to targets was significantly shorter in the FB+WIN+ and FB+WIN- conditions (by 55 and 43 ms respectively) than in FB-WIN- (baseline) condition. Patient RTs to target tones (F = 328 ms, SD = 64 ms) were significantly longer than those of controls (mean = 269 ms, SD = 47 ms) ($F(1,17) = 9.68$, $p < .01$).

Physiological data. All patients and control ERP waveforms and RT histograms are superimposed for the first presentation of the FB+WIN+ condition in Figure 3.1. The grand averages of the two groups are presented in Figure 3.2. As may be observed, there is some intra-group variability. P300 is maximum in parieto-central areas of the scalp. It is generally prolonged and attenuated in patients. P300 amplitude following the standards did not vary significantly between replications ($F < 1$) or conditions ($F < 1$) (see Table 3.7). Control P300 amplitudes (mean = 10 μv, SD = 6.41) tended to be larger than those of patients (mean = 6 μv, SD = 3,98 μv) ($F(1,15) = 3.25$, $p < .09$). Mean P300 amplitudes to target stimuli (Table 3.8 and Figure 3.2) also did not vary significantly across replications ($F(1,15) = p < 1.0$) or conditions ($F(3,45) = 1.40$). Control P300 amplitudes (mean = 11 μv, SD = 4.58 μv) however were significantly larger than those of patients (mean = 6 μv, SD = 4.03 μv), $F(1,15) = 3.25$, $p < .09$).

The latency of P300s to standard stimuli (see Table 3.9 and Figure 3.3) did not vary significantly across replications ($F(1,16) = 2.14$) or conditions ($F(3,48) = < 1.0$). It was significantly prolonged in patients (mean = 345 ms, SD = 54 ms) as compared to controls (mean = 307 ms, SD = 33 ms) ($F(1,16) = 5.03$, $p < .05$).

P300 following targets was about 12 ms later on replication trials (see Table 3.10 and Figure 3.3). Although slight, this difference was nevertheless statistically significant ($F(1,15) = 5.43$, $p < .05$). No significant effects of condition were noted ($F(3,45) = < 1.0$). Patient P300 latencies (mean = 341 ms, SD = 61 ms) tended to be longer than those of controls (mean = 304 ms, SD = 33 ms) ($F(1,15) = 3.47$, $p < .08$).

Discussion

The P300 amplitude data for each group were comparable to those of the second study. As in the second study, P300 amplitudes were somewhat larger than those recorded in Experiment 1. It was noted in the second study that Kutas, McCarthy and Donchin (1977) and Pfefferbaum, Ford, Wenegrat, Roth, and Kopell (1983) have reported higher amplitude P300s in speed compared to accuracy conditions. In this experiment, emphasis was placed consistently on speed, thus perhaps explaining the large P300 waveforms.

For controls, RTs and P300 latencies were also comparable to those in the speed condition of the first study. In patients however, RT and P300 latency were shortened by 86 and 24 ms respectively relative to the speed condition of the first experiment. P300 latency differences as large as 30-40 ms have been observed in other studies manipulating speed-accuracy tradeoff (Kutas et al., 1977; Pfefferbaum et al., 1982). The 24 ms decrease in patient P300 latencies may therefore reflect the greater emphasis on speed in this study. Patient RT and P300 latency may have

been shorter in the present study due to the greater effectiveness of FB and time windows as compared to the use of simple speed instruction in the first experiment. Other studies in which FB and time windows were provided have reported similar reductions in the RTs of both normal and retarded subjects (Baumeister & Ward, 1967; Wade, Hoover & Newell, 1984; Wade & Newell, 1981; Pachella, 1974; Schouten & Bekker, 1967; Snodgross, Luce, & Galanter, 1967; Wood, & Jennings, 1976). Although Winogron (1986) reported much larger reductions in RT (200 - 250 ms) in terms of the overall proportion of reduction, the RT decrease was actually less.

Other authors (i.e., Fitts, 1966) have also reported small reductions of RT (a 38 ms difference between speed and accuracy conditions) in normal subjects. The discrepancy of findings in these studies may perhaps be due to the use of a wider (545 ms) criterion window in the study by Fitts, and the simultaneous provision of accuracy FB.

On the first condition of the present study, when neither FB nor windows were provided, the RTs of control subjects exceeded their P300 latencies by only 19 ms. In patients, however, RT exceeded these P300 latency by 87 ms. Patient RTs on this condition were therefore more greatly influenced by processes occurring subsequent to the completion of stimulus evaluation than were those of controls. As suggested in the previous studies, the exaggeration of the P300 - RT interval in patients may have been due to their adopting a response bias that emphasizes accuracy at a cost of speed. In the present study d' was lowest in conditions where RT was shortest. Pachella (1974) has noted that very small changes in the subject's accuracy of responding may produce large

differences in RT. The small change in the patient criterion for responding might thus have produced a marked decrease in their RT.

Following training with FB and a time window RT continued to be reduced when either FB, the time window or both were later removed on replication trials. It is unlikely that the improvement of RT over trials was due to simple practice effects. In an earlier study (Campbell, Houle, Lorrain, Deacon-Elliott & Proulx, 1986) practice and fatigue effects were specifically examined during a one hour auditory discrimination task. The testing session was divided into 16 one minute interval, 4 four minute intervals and 1 sixteen minute interval. Subjects were asked to press one of two buttons depending upon whether the standard or target tone was detected. FB and time windows were not provided, nor was their particular emphasis placed on speed or accuracy of responding. The results of this study indicated that "time on task" had little influence on either RT or P300. The reduction of RT in the last condition of the present study therefore probably reflects a carry-over of the effects of FB and the time window. Due to the carry-over design of this study it is difficult to determine the relative effectiveness of the use of FB vs the time window in reducing RT.

In the last presentation of the FB-WIN- conditions (where cues to speed were removed), patient RTs were on average of 59 ms shorter than they had been on the first presentation of this condition. Control RTs were also decreased over the same condition, however, and thus remained faster than (59 ms) those of patients. Given that patient P300s were delayed on this trial by an average of 35 ms relative to controls, there

remained a 25 ms delay which could not be accounted by a slowing of stimulus evaluation.

From the perspective of rehabilitation, however, it may be of more interest to compare patient RTs following training (the last replication of the FB-WIN- condition) to the "normal" RTs of controls before training (the first presentation of the FB-WIN- condition). The post- treatment RTs of patients were, in fact only 25 ms longer than what the control RTs had on the first replication. The residual 25 ms delay may be due to slower stimulus evaluation in patients, as their P300s were delayed by 35 ms on this trial. The signal detection measure of sensitivity, d', did not vary between groups. The RTs of patients were therefore reduced to within normal limits with no greater cost in sensitivity than in controls.

It is impossible to speculate over what period of time carry-over might be maintained after the removal of the cues for speeds of responding. The enduring reduction of RT in the present experiment, however, suggests that these manipulations might be useful in a therapeutic setting.

Table 3.1

Demographic and Neurological Data

ID	age at injury	years educ.	GCS	coma dur.	PTA	GOS	Expl	Exp2	Exp3
JC	31.3	19	4	1	10	1	*	*	4.3
KD	18.9	14	5	14	30	1	3.7	5.7	5.0
LD	19.6	12	5	13	30	1	5.2	5.2	6.5
PD	23.1	10	4	7	60	2	*	3.7	3.7
OE	20.1	17	5	75	—	2	6.9	6.9	*
BG	20.1	15	7	7	25	1	*	*	4.2
BL	20.9	13	6	30	60	1	*	6.5	6.5
JM	18.0	13	5	73	150	2	4.0	4.0	5.3
GM	21.6	15	4	50	121	1	4.8	4.8	6.1
SM	22.5	13	5	30	60	1	3.8	3.8	5.1
HM	45.1	19	5	30	60	2	3.9	3.9	5.2
GP	18.4	12	4	11	30	1	*	5.3	*
LS	19.2	13	5	21	30	1	*	3.1	3.1
MM	22.7	10	4	35	—	3	4.6	4.6	5.9

* = did not participate

Table 3.2

Locations of verified lesions

Patient	Hematomas	Contusions
BL	right frontal	
JM	bilateral subdural	left diffuse
GM		bilateral diffuse
GP	right fronto-temporal	right fronto-temporal
LS		right diffuse
PD	bilateral temporal	
KD	right temporal	
OE		
BG		
HM		

Table 3.3

Proportion of correct responses (SDs are in brackets)

		Mean Proportion of Correct Responses			
Condition		Patients		Controls	
		Hits	Correct rejections	Hits	Correct rejections
FB-WIN-	1	.89	.94	.99	.97
FB-WIN+	1	.93	.94	.91	.92
FB+WIN-	1	.84	.84	.86	.84
FB+WIN+	1	.84	.85	.84	.83
FB+WIN+	2	.77	.80	.87	.84
FB+WIN-	2	.74	.74	.87	.73
FB-WIN+	2	.78	.80	.88	.79
FB-WIN-	2	.82	.85	.89	.87
		.83	.85	.89	.86

		Mean Proportion of Errors			
Condition		False Alarms	Misses	False Alarms	Misses
FB-WIN-	1	.06	.11	.03	.01
FB-WIN+	1	.06	.07	.08	.09
FB+WIN-	1	.16	.16	.16	.14
FB+WIN+	1	.15	.16	.17	.16
FB+WIN+	2	.20	.23	.16	.13
FB+WIN-	2	.26	.26	.27	.13
FB-WIN+	2	.20	.22	.21	.12
FB-WIN-	2	.15	.18	.13	.11
		.15	.17	.14	.11

Table 3.4

Mean d's (SDs in parentheses)

Condition	replication	Patients	Controls
FB-WIN-	1	0.93 (0.42)	0.67 (0.52)
FB-WIN+	1	0.85 (0.35)	0.66 (0.39)
FB+WIN-	1	1.07 (0.59)	1.05 (0.61)
FB+WIN+	1	1.07 (0.29)	0.72 (0.41)
FB+WIN+	2	1.95 (1.63)	1.42 (1.29)
FB+WIN-	2	2.01 (1.61)	2.06 (1.24)
FB-WIN+	2	2.21 (1.45)	1.97 (1.17)
FB-WIN-	2	3.01 (1.43)	3.01 (0.95)

Table 3.5

Mean reaction times (ms) to standard stimuli (SDs in parentheses)

Condition	replication	Patients	Controls
FB-WIN-	1	433 (125)	338 (58)
FB-WIN+	1	416 (116)	289 (36)
FB+WIN-	1	376 (94)	272 (32)
FB+WIN+	1	342 (108)	263 (28)
FB+WIN+	2	349 (100)	260 (37)
FB+WIN-	2	346 (93)	279 (63)
FB-WIN+	2	347 (70)	287 (66)
FB-WIN-	2	379 (70)	306 (78)

Table 3.6

Mean reaction times (ms) to target stimuli (SDs in parentheses)

Condition	replication	Patients	Controls
FB-WIN-	1	391 (97)	318 (68)
FB-WIN+	1	356 (77)	274 (43)
FB+WIN-	1	332 (69)	254 (25)
FB+WIN+	1	311 (64)	251 (32)
FB+WIN+	2	293 (70)	242 (32)
FB+WIN-	2	297 (70)	265 (72)
FB-WIN+	2	316 (36)	260 (44)
FB-WIN-	2	326 (30)	285 (57)

Table 3.7

Mean P300 amplitudes (μV) for standard stimuli (SDs are in parenthesis)

Condition	replication	Patients	Controls
FB-WIN-	1	5.87 (2.51)	10.49 (5.04)
FB-WIN+	1	5.92 (3.85)	10.31 (6.16)
FB+WIN-	1	5.32 (4.05)	10.83 (6.69)
FB+WIN+	1	6.06 (4.48)	8.20 (3.87)
FB+WIN+	2	5.69 (4.48)	8.60 (6.48)
FB+WIN-	2	6.25 (4.36)	9.69 (7.61)
FB-WIN+	2	6.34 (3.96)	10.06 (5.16)
FB-WIN-	2	5.74 (4.15)	9.42 (5.32)

Table 3.8

Mean P300 amplitudes (uV) for target stimuli (SDs are in parenthesis)

Condition	replication	Patients	Controls
FB-WIN-	1	6.71 (2.8)	8.8 (3.64)
FB-WIN+	1	6.62 (3.4)	11.25 (4.81)
FB+WIN-	1	6.75 (4.4)	12.29 (4.08)
FB+WIN+	1	6.71 (4.2)	11.90 (4.10)
FB+WIN+	2	5.45 (4.34)	12.63 (5.24)
FB+WIN-	2	8.29 (4.30)	11.39 (7.08)
FB-WIN+	2	5.14 (4.66)	10.65 (3.28)
FB-WIN-	2	7.53 (4.16)	11.80 (4.46)

Table 3.9

Mean P300 latencies (ms) for standard stimuli (SDs in parentheses)

Condition	replication	Patients	Controls
FB-WIN-	1	327 (45)	309 (35)
FB-WIN+	1	356 (61)	303 (28)
FB+WIN-	1	345 (55)	300 (26)
FB+WIN+	1	334 (41)	308 (39)
FB+WIN+	2	344 (55)	307 (20)
FB+WIN-	2	343 (63)	302 (34)
FB-WIN+	2	350 (70)	319 (38)
FB-WIN-	2	363 (44)	309 (46)
	X		

Table 3.10

Mean P300 latencies (ms) for target stimuli (SDs in parentheses)

Condition	replication	Patients	Controls
FB-WIN-	1	323 (42)	308 (28)
FB-WIN+	1	320 (40)	310 (37)
FB+WIN-	1	341 (61)	322 (28)
FB+WIN+	1	352 (74)	328 (23)
FB+WIN+	2	352 (78)	331 (26)
FB+WIN-	2	338 (69)	330 (37)
FB-WIN+	2	352 (66)	326 (36)
FB-WIN-	2	347 (60)	331 (50)
	X		

Table 3.11

Summary of ANOVAs

		Standards			Target		
	d'	RT	P300 amp	P300 lat	RT	P300 amp	P300 lat
Main Effects							
Group	NS	P>C	NS	P>C	P>C	C>P	NS
Replication	NS	NS	NS	NS	1>2	NS	1<2
Condition	1,8>4,5	FB-WIN> FB+WIN- &FB-WIN+	NS	NS	FB-WIN> FB+WIN- &FB+WIN+	NS	NS
Interactions							
GxR	NS	NS	NS	NS	NS	NS	NS
GxC	NS	NS	NS	NS	NS	NS	NS
CxR	NS	NS	NS	NS	NS	NS	NS

Figure 3.1

ERPs and RT histograms for patients (N = 10) and controls (N = 10) during the first presentation of the FB+WIN+ condition. Tracings of individual subjects have been superimposed.

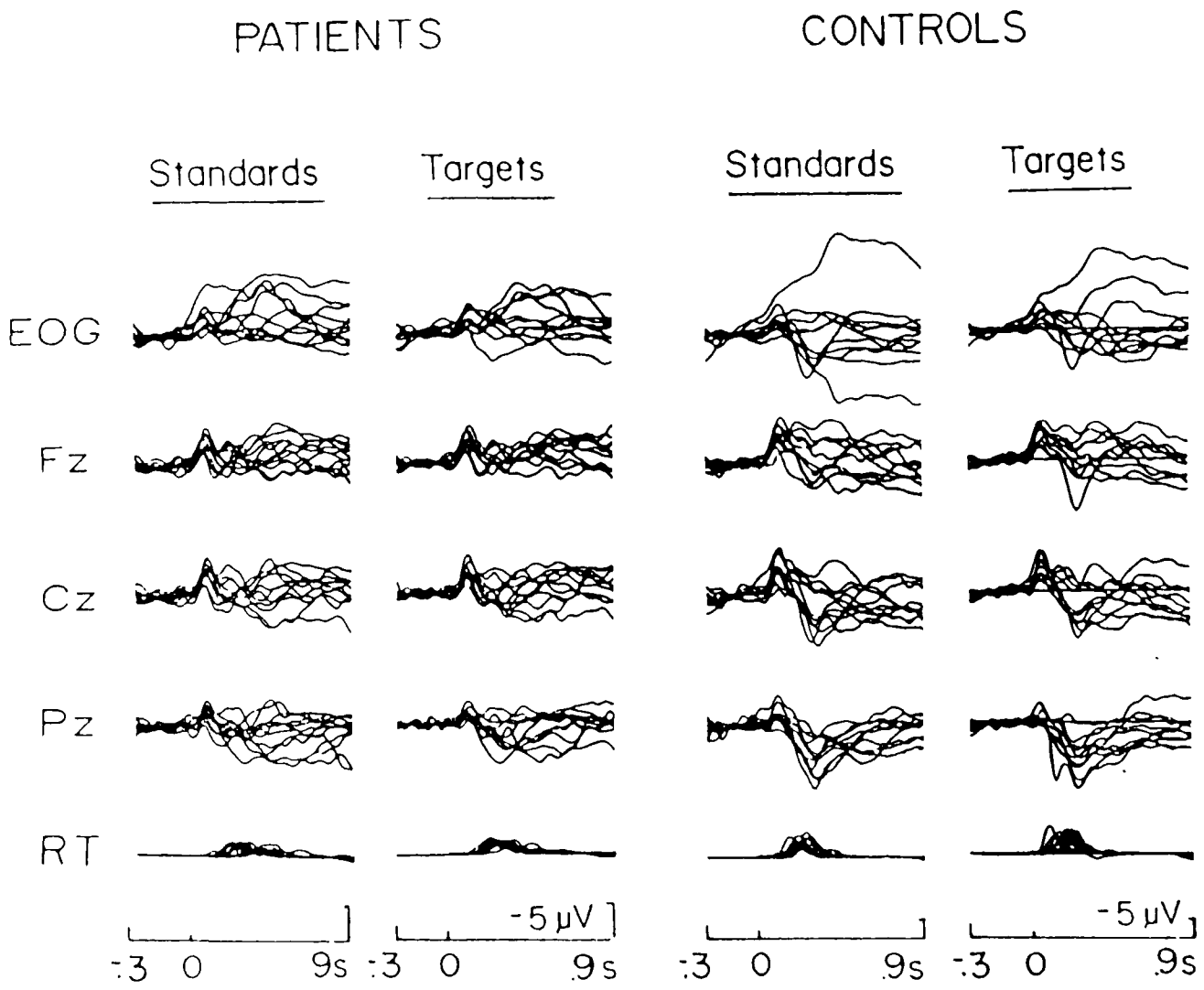

PATIENTS
CONTROLS
Standards
Targets
Standards
Targets
EOG
Fz
Cz
Pz
RT
-5 μV
-5 μV
-.3 0 .9s
-.3 0 .9s
-.3 0 .9s
-.3 0 .9s

Figure 3.2

Grand average ERPs and RT histograms for patients (N = 10) and controls (N = 10) during the FB+WIN+ condition. Patients are traced in the thick line and controls in the thin line.

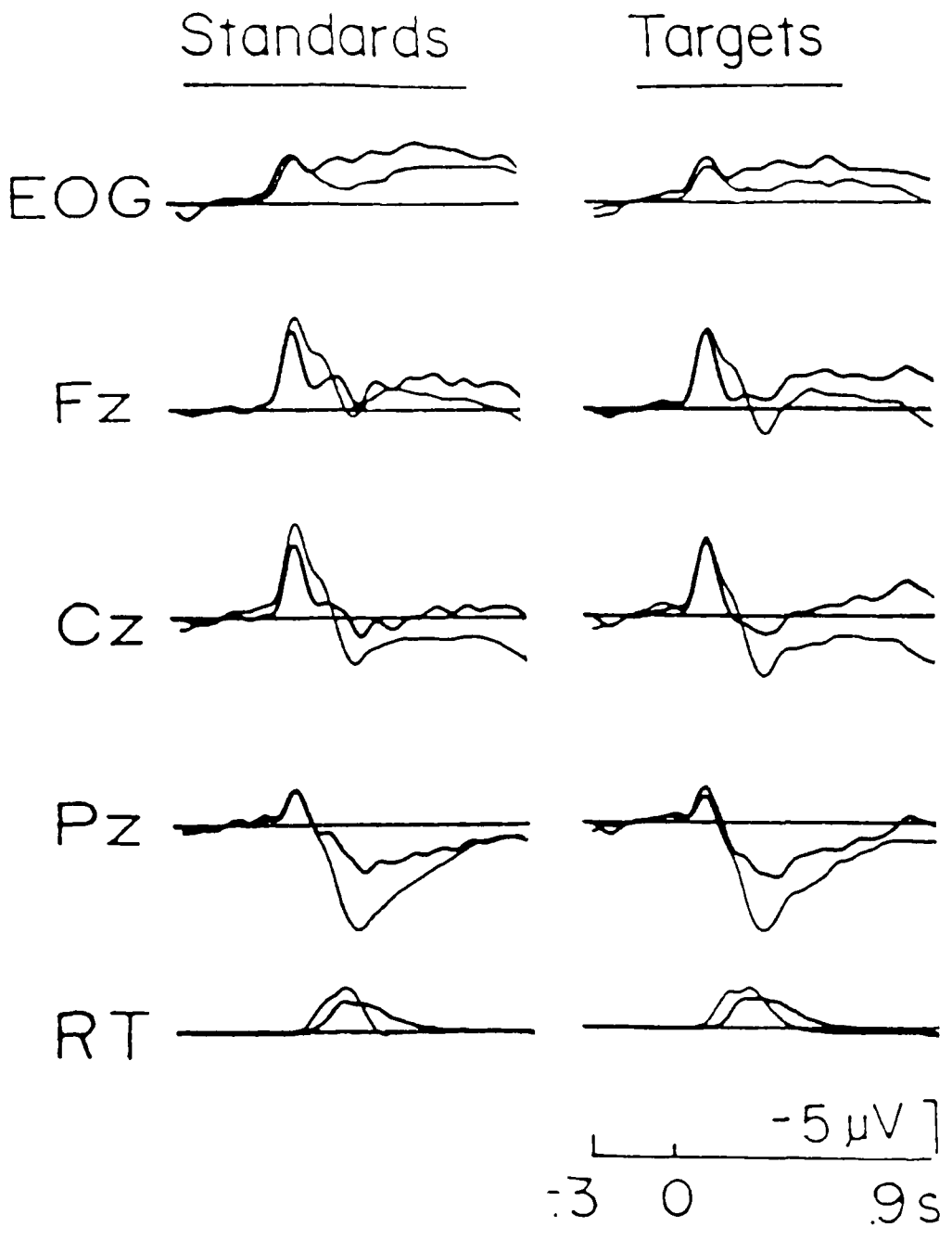
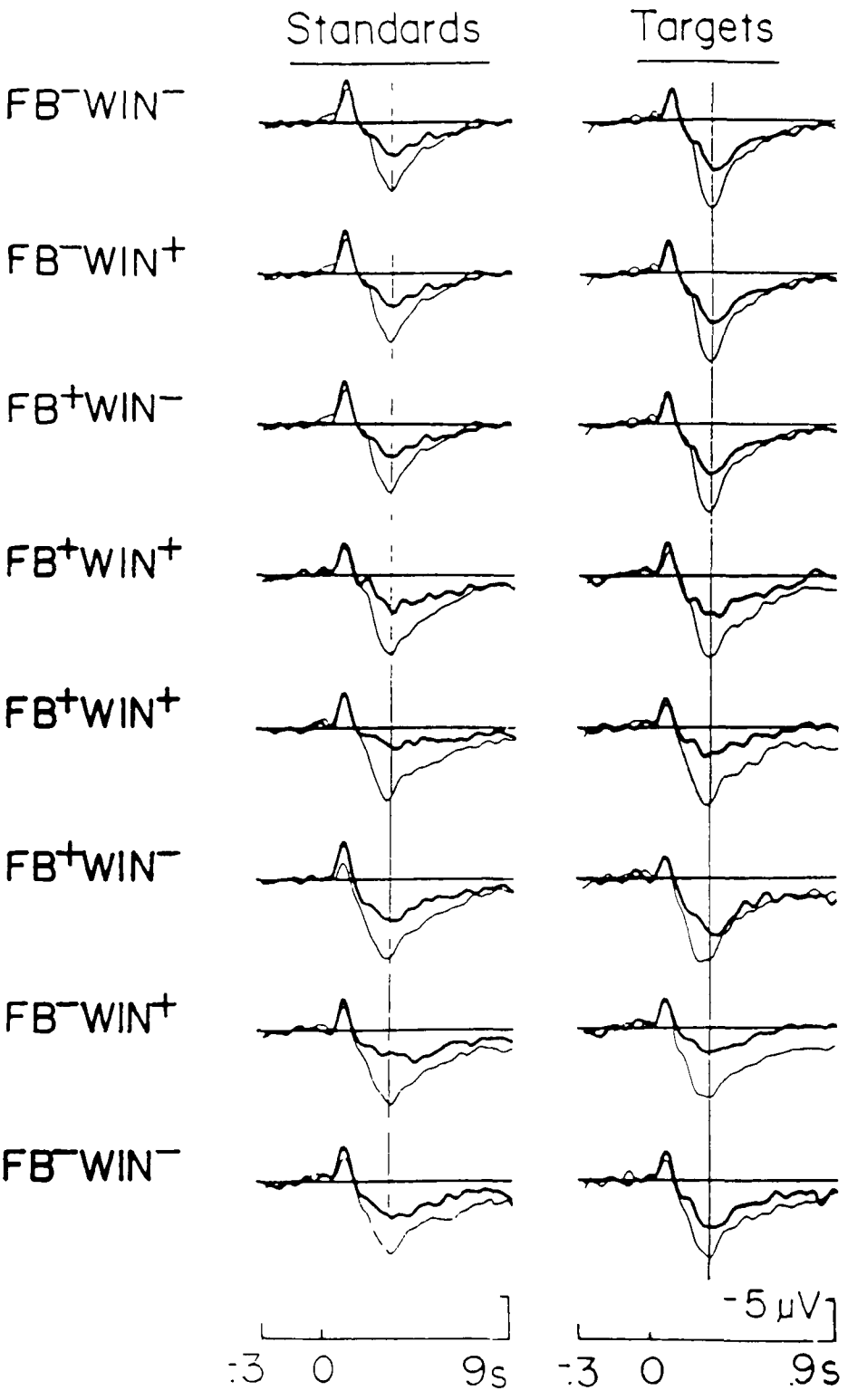

Standards
Targets
EOG
Fz
Cz
Pz
RT
-5 μV
-3
0
.9s

Figure 3.3

Grand average ERPs recorded from patients (N = 10) and controls (N = 10) at Pz. Patients are traced in the thick line and controls in the thin line. The order of conditions corresponds to the order in which they were presented.


Standards
Targets
FB⁻WIN⁻
FB⁻WIN⁺
FB⁺WIN⁻
FB⁺WIN⁺
FB⁺WIN⁺
FB⁺WIN⁻
FB⁻WIN⁺
FB⁻WIN⁻
-5 μV
-.3
0
.9s
-.3
0
.9s

CHAPTER V

General Discussion

A slowing of reaction time is one of the most commonly described deficits resulting from CHI (Dencker, & Lofving, 1958; Gronwall, & Sampson, 1974; Klensch, 1973; Miller, 1970; Norrman, & Svahn, 1961; Reusch, 1944; van Zomeren, Brouwer, & Deelman, 1981; van Zomeren, & Deelman, 1981). The locus of the delay in RT is nevertheless poorly understood. In general, choice RT has been found to be more affected than simple RT. Furthermore, the delay in patient RTs increases commensurately with the number of stimulus and alternatives. This would suggest that the RTs of the head-injured might be prolonged due to a slowing of cognitive processes rather than from a residual motor deficit. Since some other studies, (Klench, 1973; van Zomeren, & Deelman, 1981) have also reported significant delays in simple RT, however, the issue remains unresolved.

In this series of studies, RT was recorded in conjunction with P300, a waveform which has been proposed to be primarily sensitive to the duration of stimulus evaluation, and only minimally affected by response choice and production time (Brookhuis, Mulder, & Gloerich, 1983; Duncan-Johnson, & Kopell, 1981; Kutak, McCarthy, & Donchin, 1979; Magliero, Bashore, Coles, & Donchin, 1984; McCarthy, & Donchin, 1981; Mulder, Brookhuis, Gloerich, van Dellen, & Mulder, 1984; Ragot, 1984; Ragot, & Renault, 1981). Earlier studies have observed delays of approximately 50 ms in the P300s of the head-injured (Campbell, Houle, Lorrain, Deacon-Elliott, & Proulx, 1986; Curry, & Cooper, 1985). RT was delayed, however

by as much as 150 ms in these studies. This would suggest that at least part of the slowing of RT in the head injured is due to post-decisional processes. van Zomeren and his colleagues (1981) and Brooks (1974) have suggested that the head-injured may adopt a more stringent response criterion. A bias towards speed or accuracy has been noted to affect response production, but not stimulus encoding or evaluation (Pachella, 1974; Swanson, & Briggs, 1969). The disparity between patient RTs and P300 latencies might therefore indicate that they maintain a bias towards accuracy of responding, at a cost of speed.

The purpose of this thesis was to attempt to shorten patient RTs by manipulating their emphasis on either speed or accuracy of responding. It was hypothesized that by emphasizing speed of responding, patient RTs could be reduced to within their P300 latencies. Three experiments have been presented. In the first, RT was reduced significantly in both patients and controls using simple instructions that emphasized speed of responding. Despite the reduction in patient RTs, they remained 180 ms later than those of controls. Furthermore, the reduction in RT for both group was much less than that observed in other studies in which similar types of instructions emphasized speed (i.e., Pfefferbaum et al., 1982).

Pachella (1974) has pointed out that increases in speed of responding are generally accomplished at a cost of accuracy. Although RTs did decrease in the first study, there was no concomittant increase in the number of errors. This therefore suggests that patients may have continued to emphasize accuracy, in spite of an increase in their speed of responding. This point of view is reinforced through a comparison of RT-

P300 latency differences. In controls the mean RT preceded P300 peak latency during speed instruction conditions, whereas in patients the opposite was observed -- the P300 peak latency preceded mean RT.

It is possible that the head-injured are unable to "internally" monitor their speed of responding. Rabbitt (1979) suggests that RT is monitored on a trial-to-trial basis in order to maximize both the speed and the accuracy of responses. The monitoring of RT is thought by some authors to occur through the use of internal FB (Lundy, 1985; Welford, 1980). According to Rabbitt (1979), subjects gradually increase their speed of responding over successive trials, until an error is committed. On the trials following the commission of errors, RT increases until a pattern of correct responding is re-established. RT then decreases once more until it assymptotes at its former "safe" level. Ideally, single trial RTs should have been analyzed in this thesis. Unfortunately, a proper test of the Rabbitt model (or of any of the other models of trial-to-trial RT variation) requires many more trials than were presented in each condition. Rabbitt (1979) has suggested that the variability of single-trial RTs decreases when subjects learn, through monitoring their RTs to respond consistently within the same time period (the RT band which maximizes both speed and accuracy). It is therefore possible that the unusually long mean RT observed in experiment 1 may have been due to an inconsistency of responding. The manipulations employed in experiment 2 may have reduced this variability. In particular, extremely long RTs may have been removed. An examination of the control and patient RT histograms in the experiment 1 (Figure 1.3) revealed a tendency for positive

skewing on the part of the patients. The mean RT was therefore probably inflated by unusually long RTs. Nevertheless, a comparison of the peak of the RT histograms in experiments 1 and 2 indicates a negative shift in the latter. Hence the reduction in mean RT is probably not simply due to a correction of extreme scores. Rather, there appears to be a negative shift of the overall RT distribution curve. In experiment 1 the inability of patients to modify their responding in accordance with the (instructional) demands of the task, suggests that the system which monitors speed of responding in normal subjects may be deficient in the head-injured. Dysfunction of this system might explain their apparent bias towards accuracy, and the "uncertainty" which has been suggested by Brooks (1974) and van Zomeren (1981). It would also explain the overall attenuation of patient P300s. P300 has been proposed to reflect the resolution of uncertainty. Ruchkin and Sutton (1978) have related changes in P300 amplitude to "equivocation". Equivocation refers to a loss of information due to the subjects uncertainty as to what has been perceived. It is thought to have an attenuating affect on P300 amplitude. In the head injured, a loss of information may arise from their inability to monitor responses, resulting in later and smaller P300s.

In order to further constrain patient RTs, a second experiment was run which was designed to assist patients in monitoring their responses. Several studies have employed criterion time windows within which responses must occur and/or FB on the subjects speed of responding to constrain RT (Fitts, 1966; Pachella, 1974; Pfefferbaum et al., 1983; Shouten, & Bekker, 1967; Wood & Jennings, 1976). Winogron (1986) has recently

attempted to decrease the RTs of head injured children using these manipulations. The RTs in this study were very long for both groups, possibly owing to the difficulty of the discrimination tasks which was used and the young age of the patients. Although patient RTs were decreased (by 150 ms), they still remained 150 ms longer than those of controls. Winogron employed time windows, which were either 1, 1.5 or 2 s in duration. These may not have constrained RTs to the full extent possible. If the longer patient RT were due entirely to a delay in response production it should have been possible to decrease RT to within P300 peak latency. P300 was however, not recorded in Winogron's study. In the second study of this thesis, time windows were provided which were much smaller than those used by Winogron. The windows were either "narrow" (300 ms for controls, 350 ms for patients) in some conditions or "wide" (500 ms for patients, 450 ms for controls) in others. In different conditions FB was presented on either the subject's speed or their accuracy of responding. The manipulations were found to reduce RT by approximately 40 and 100 ms relative to the speed and accuracy conditions of the first study respectively. The proportion of RT reduction was therefore similar to that observed in other studies (Link, & Tindall, 1971; Pfefferbaum et al., 1983). The reduction of RTs was greatest for both groups in a condition in which the narrow time window and speed FB were provided. In this condition, on correct trials patient RT occurred at the same time as (or slightly earlier) than their P300 peak latency. It is therefore unlikely that in this condition patient RTs could have been reduced further. As in the first study, P300 latency did not differ

significantly among conditions although it was significantly prolonged in patients. The P300 amplitudes of both groups were somewhat larger than they had been in the first study, perhaps due to the greater cognitive effort as a consequence of the overall emphasis on speed (Kutas et al., 1977; Pfefferbaum et al., 1983).

It is quite possible that the experimental manipulations increased the amount of attention subjects paid to performing the task. van Zomeren has reviewed attentional deficits in the head-injured under the categories of (a) selected attention (the ability to focus on some events and disregard others), (b) divided attention (the ability to attend to several events simultaneously), and (c) the speed with which information can be processed. van Zomeren closely links speed of responding to level of attention. Thus, fast RTs are associated with a high level of attention whereas slow RTs are associated with a low level of attention. From this perspective, the decrease in RT observed in the present task may have been accomplished as a result of an increase in subjects' level of attention.

Gronwall and Sampson (1974) have suggested that arousal may be impaired by CHI. Although arousal has been examined in a number of studies there is thus far, virtually no evidence of lowered arousal in CHI patients (Brouwer, 1985; Brouwer, & Woffelaar, 1985; Dencker, & Lofving, 1958; Martinius et al., 1977; van Zomeren, 1981). Arousal, however, is admittedly difficult to manipulate and measure. The extent to which it may have been altered by the experimental manipulations is therefore difficult to determine.

The time window and FB may have provided subjects with information which allowed them to focus their attention on the salient aspect of the task (i.e.: speed or accuracy) and modify their responses accordingly. In control subjects, the FB and time constraints may have supplemented the information supplied by their internal monitoring system.

The ability to monitor behaviour is thought to be dependent on the integrity of the frontal lobes and their connections with other brain structures (Luria, 1973; Stuss, 1986; Stuss, & Benson, 1984, 1986). In CHI the frontal and temporal areas have been found to be most vulnerable to damage, regardless of the point of impact (Ommaya, Grubb, & Nauman, 1971). Clinical observations also underscore the possible involvement of the frontal lobes in CHI. Prigatano (1987) has reported that awareness of one's self and one's environment ("metacognition") is disturbed by CHI. Stuss (1987) has reported that tests traditionally thought to reflect frontal lobe functioning are most able to discriminate CHI patients from controls. He warns however that "even severe focal frontal lobe damage may not disturb the basic activities of various functional systems ... particularly ... if the examiner is a good tester, providing a controlled, standardized, structured environment and, in a sense, becoming the frontal lobes of the patient" (p. 174). For patients, the rigid time windows and feedback concerning whether the response was actually within the window may thus have provided this controlled, standardized and structured environmental means to monitor their speed of responding. As a note of caution, it should be stressed that there was little evidence of consistent frontal lobe damage in the present sample of CHI patients.

Unfortunately, CT scans were available for only a limited number of the patients. The small sample size did not permit statistical analysis of the radiological findings. Nevertheless, an examination of single cases did not reveal any striking differences between patients with known frontal pathology and those with other focal lesions or evidence of diffuse damage. It is possible to argue that the basis for evidence of damage -- the CT scan -- may not be a particularly good indicator of frontal lobe injury. It has been noted, for instance that the diffuse damage which characterizes CHI is not always apparent in CT scans (Filley, Cranberg, Alexander, & Hart, 1987). It is therefore difficult to make meaningful comparisons between these individuals on the basis of identifiable lesions. A consistent battery of neuropsychological tests was not administered to all patients. Even had scores on frontal lobe tests been abnormal, however this would not necessarily imply focal frontal lobe damage. Diffuse injury to the brain can provide neuropsychological profiles which are quite similar to those seen in focal frontal lobe pathology (Kinsbourne, 1977; Stuss, 1987).

The final study of this thesis was designed to examine carry-over of the beneficial effects of the manipulations. FB and time windows were presented separately on some trials and in combination on others. The FB and time window were then gradually phased out. A significant carry-over was observed. RT was shorter on replication trials than it had been on the first presentation of each condition. The reduction of RT was maintained even when cues to speed were removed. In an earlier study (Campbell et al., 1986) no change in RT was observed over a testing

session of similar duration. The effects of practice alone would therefore not appear to account for the greater reduction of RT on replication trials. It is more probable that the reduction of RT on these trials reflected a carry-over of the specific training provided by the manipulations. The monitoring of responses is thought to occur automatically, once a criterion level of performance is reached (Rabbit, 1979, 1981) at this point, FB provided by the subjects internal monitoring system becomes redundant (Annett, 1966). The enduring effect of FB and the time window is consistent with this theory. The patients may have required the information provided by these cues in order to maximize response speed, but were able to maintain faster responding once the cues to speed were removed.

As has been tentatively suggested, the manipulation may have subsumed certain monitoring functions which are normally performed by the frontal lobes. The frontal lobes are thought to be most active during the acquisition of behaviours. Once a task becomes overlearned, the frontal lobes are no longer necessary to perform it correctly (Damasio, 1979; Shallice, 1982; Mazzioti, & Phelps, 1986). The continued reduction of RTs after the removal of the FB and time window was therefore consistent with this view.

In this thesis it was demonstrated that the delay in RT imposed by the pathology of CHI could be reversed if cues to speed were provided. This suggested that the delay in patient RTs was due to a cognitive deficit, perhaps in the monitoring of performance. It also discredited the view that the RTs of the head injured may be delayed due to motor

slowing (van Zomeren, 1981). It is impossible to determine how enduring the effects of providing cues to speed might be. In the third study, both manipulations were removed in only one trial. Had subsequent trials been presented without cues to speed, patient RTs may have gradually increased to their former levels. The observation of carry-over on this, and other replication trials, however was at least encouraging.

Conclusions

1. The reaction times of the head-injured were delayed due to a bias towards accuracy of responding.
2. The RTs of CHI patients were altered by about 30% but only when FB and a time window were provided.
3. Emphasizing speed of responding had very little effect on P33 latency.
4. RT remains shorter for some time after the FB and time window are removed.
5. Providing cues to speed assists subjects in monitoring their RTs. Once a criterion level of performance is reached subjects can respond with the same speed in the absence of external information.

REFERENCES

Adams, J. H., Graham, D. I., Murray, L. S., & Scott, G. (1982). Diffuse axonal injury due to non-missile injury in humans: an analysis of 45 cases. Annals of Neurology, 12, 557-563.

Adams, J., Mitchell, D., Graham, D., & Doyle, D. (1977). Diffuse brain damage of immediate impact types. Brain, 100, 489-502.

Alluisi, E. A., Strain, G. S., & Thurmond, J. B. (1964). Stimulus-response compatibility and the rate of gain of information. Psychonomic Science, 1, 111-112.

Annett, J. (1966). A note on Davis's refutation of the expectancy hypothesis. Quarterly Journal of Experimental Psychology, 18, 179-180.

Bakay, L, & Glasauer, F. E. (1980). Head Injury. Boston: Little, Brown and Company.

Bakalska, B., & Angelov, A. (1963). Cited by Krinchick, E. P. (1969) in W. G. Koster (Ed.): Attention and Performance II. Amsterdam, North-Holland, pp. 27-36.

Begleifer, H., Porjesz, B., Chou, C., & Aunon, J. (1983). P3 and stimulus incentive value. Psychophysiology, 20, 95-101.

Benton, A., & Blackburn, H. (1957). Practice effects in reaction time tasks with brain injured patients. Journal of Abnormal Social Psychology, 54, 109-113.

Bertelson, P. (1961). Sequential redundancy and speed in a serial two-choice responding task. Quarterly Journal of Experimental Psychology, 13, 90-102.

Bertelson, P. (1966). Central intermittency twenty years later. Quarterly Journal of Experimental Psychology, 18, 153-163.

Bertelson, P., & Tisseyre, F. (1966). Choice reaction time as a function of stimulus versus response relative frequency of occurrence. Nature, 212, 1069-1070.

Besner, D. (1977). Character classification: Levels of processing and the effects of stimulus probability. Bulletin of the Psychonomic Society, 9, 337-339.

Biederman, I., & Stacy, E. W. (1974). Stimulus probability and stimulus set size in memory scanning. Journal of Experimental Psychology, 102, 1100-1107.

Botwinick, J., Brinley, J. F., & Robbin, J. S. (1958). The effect of motivation by electrical shocks on reaction-time in relation to age. American Journal of Psychology, 71, 408-411.

Brainard, R. W., Irby, T. S., Fitts, P. M., & Alluisi, E. A. (1962). Some variables influencing the rate of gain of information. Journal of Experimental Psychology, 63, 105-110.

Brebner, J. M., & Welford, A. T. (1980). An historical background sketch. In A. T. Welford (Ed.), Reaction Times, (pp.1-24). New York: Academic Press.

Brewer, N., & Smith, G. A. (1984). How normal and retarded individuals monitor and regulate speed and accuracy of responding in serial

choice tasks. Journal of Experimental Psychology: General, 43 (1), 71-93.

Broadbent, D. E. (1971). Decision and Stress. Academic Press, London.

Brooks, D. N. (1974). Recognition memory after closed head injury. Journal of Neurology, Neurosurgery and Psychiatry, 37, 794-801.

Brooks, N. (Ed.) (1984). Closed head injury, psychological, social, and family consequences. Oxford: Oxford University Press.

Brookhuis, K., Mulder, G., Mulder, L. J., & Gloerich (1983). The P3 complex as an index of information processing: The effects of response probability. Biological Psychology, 17, 277-296.

Brower, W. H. (1985). Limitation of Attention After Closed Head-Injury. Kripps repro moppel; geboren te T, imahi.

Campbell, K. B. (1985). Mental chrometry. I. Behavioural and physiological techniques. In B. D. Kircaldy (Ed.): Individual Differences in Movement, (pp. 117-145). Lancaster, Eng.: MTP Press Ltd.

Campbell, K. B., Courchesne, E., Picton, T. W., & Squires, K. C. (1979). Evoked potential correlates of human information processing. Biological Psychology, 8, 45-68.

Campbell, K., Houle, S., Lorrain, D., Deacon-Elliott, D., & Proulx, G. (1986). In W. C. McCallum, R. Zappeli and F. Denoth (Eds.): Cerebral Psychophysiology: Studies in Event-related Pottentials (EEG Suppl. 38), (pp. 486-488). Holland: Elsevier Science Publishers.

Chase, W. G., & Posner, M. I. (1965). The effect of auditory and visual confusability on visual and memory search tasks. Paper presented at

the meeting of the Midwestern Psychological Association, Chicago, May 1965.

Coles, M., Gratton, G., Bashore, T., Eriksen, C., & Donchin, E. (1985). A psychophysiological investigation of the continuous flow model of human information processing. Journal of Experimental Psychology: Human Perception and Performance, 11, 529-553.

Conkey, R. (1938). Psychological changes associated with head injuries. Archives of Psychology, 33 (232).

Courchesne, E., Courchesne, R., & Hillyard, S. (1978). The effects of stimulus deviation on P3 waves to easily recognized stimuli. Neuropsychologia, 16, 189-199.

Courchesne, E., Hillyard, S., & Galambos, R. (1975). Stimulus novelty, task relevance and the visual evoked potential in man. Electroencephalography and Clinical Neurophysiology, 39, 131-143.

Craft, J., & Simon, R. (1970). Processing symbolic information from a visual display. Interference from an irrelevant directional cue. Journal of Experimental Psychology, 83, 415-420.

Crossman, E. R. F. W. (1955). The measurement of discriminability. Quarterly Journal of Experimental Psychology, 7, 176-195.

Curry, S. H. (1980). Event related potentials as indicants of structural and functional damage in closed head injury. Progress in Brain Research, 54, 507-515.

Curry, S. H., Cooper, R., Cummins, B., & Skidmore, S (1985). Conference presentation. International congress of the electroencephalography and clinical neurophysiology. London, U.K.

Davis, R., Moray, N., & Treisman, A. (1961). Imitative responses and the rate of gain of information. Quarterly Journal of Experimental Psychology, 13, 78-89.

Deacon-Elliott, D., & Campbell, K. B. (1987). P3 evoked by visual feedback in normal and closed head-injured subjects. In R. Johnson, J. W. Rohrbaugh, and R. Parasuraman (Eds.): Current trends in event-related potential research. EEG Supl. 40, pp. 664-669.

Dencker, S., & Lofving, B. (1958). A psychometric study of identical twins discordant for closed head injury. Acta Psychiatrica Neurologica Scandinavia, 33, (122).

Desmedt, J. (1980). P300 in serial tasks: an essential post-decision closure mechanism. In Kornhuber, H. H. and Decke, L. (Eds.). Motivation, motor and sensory processes of the brain: Electrical potentials, behaviour and clinical use. Progress in brain research, Vol. 54 (pp. 682-686). Elsevier/North Holland, Amsterdam.

Desmedt, J. E. (1981). Scalp-recorded cerebral event-related potentials in man as a point of entry into the analysis of cognitive processing. In F. O. Schmitt, F. G. Worden, G. Adelman, and S. D. Dennis (Eds.): The Organization of the Cerebral Cortex. Cambridge, M.A.: MIT Press, pp. 441-473.

Desmedt, J. E., & Debecker, J. (1979). Wave form and neural mechanism of the decision P350 elicited without pre-stimulus CNV or readiness potential in random sequences of near-threshold auditory clicks and finger stimuli. Electroencephalography and Clinical Neurophysiology, 47, 648-670.

Dillon, P. J. (1966). Stimulus versus response decisions as determinants of the relative frequency effect in disjunctive reaction-time performance. Journal of Experimental Psychology, 71, 3221-330.

Donchin, E. (1975). Brain Electrical Correlates of Pattern Recognition in Biomedical Engineering. New York: John Wiley.

Donchin, E. (1979). Event-related potentials: A tool in the study of human information processing. In H. Begleiter (Ed.): Evoked Brain Potentials and Behavior. New York: Plenum Press.

Donchin, E. (1981). Surprise! ... Surprise? Psychophysiology, 18, 493-515.

Donchin, E., Kubovy, M., Kutas, M., Johnson, R., & Herning, R. (1973. Graded changes in evoked response (P300) amplitude as a function of cognitive activity. Perception and Psychophysics, 14 (2), 319-324.

Donders, F. C. (1868). On the speed of mental processes. Translated by Koster, W. G. (1969). Acta Psychological, 30, 412-431.

Dublin, A. B., French, B. N., & Rennick, J. M. (1977). Computed tomography in head injury. Radiology, 122, 365-369.

Duncan-Johnson, C. (1981). P300 latency: A new metric of information processing. Psychophysiology, 18, 207-215.

Duncan-Johnson, C. C., & Donchin, E. (1971). On quantifying surprise: The variation of event related potentials with subjective probability. Psychophysiology, 14, 456-467.

Duncan-Johnson, C., & Donchin, E. (1982). The P300 component of the event-related brain potential as an index of information processing. Biological Psychology, 14, 1-52.

Duncan-Johnson, C., & Kopell, B. (1981). The Stroop effect: brain potentials localize the source of interference. Science, 214, 938-940.

Elsberg, C. A., & Spotnitz, H. (1938). The sense of taste. Bulletin of the Neural Institute of New York, 7, 174-177.

Eriksen, C. W., & Schultz, D. W. (1979). Information processing in visual search: A continuous flow conception and experimental results. Perception and Psychophysics, 25, 249-263.

Ferrée, C. E., & Rand, G. (1927). Intensity of light and speed of vision studied with special references to industrial situations. Trans. Illum. Eng. Soc., 22, 79-110.

Fessard, A., & Kuchorski, P. (1934). Recherches sur les temps de réaction aux sens de hauteurs de l'intensités différentes. I. Année Psychologique, 35, 103-117.

Field, J. (1976). Epidemiology of head injury in England and Wales: With particular application to rehabilitation. Leicester: Printed for H. M. Stronery Office by Willsons.

Filley, C. M., Cranberg, L. D., Alexander, M. P., & Hart, E. J. (1987). Neurobehavioral outcome after closed head injury in childhood and adolescence. Archives of Neurology, 44, 194-198.

Fitts, P. M. (1966). Cognitive aspects of information processing: III. Set for speed versus accuracy. Journal of Experimental Psychology, 71, 849-857.

Fitts, P. M., & Deininger, R. (1954). S-R compatability: correspon-

dence among paired elements within stimulus and response codes. Journal of Experimental Psychology, 48, 483-492.

Fitts, P. M., Peterson, J. R., & Wolpe, J. (1963). Cognitive aspects of information processing: II. Adjustments to stimulus redundancy. Journal of Experimental Psychology, 65, 423-432.

Fitts, P. M., & Posner, M. I. (1967). Human Performance. Monteray: Brooks-Cole.

Fitts, P. M., & Sieger, C. M. (1953). S-R compatability: Spatial characteristics of stimulus and response codes. Journal of Experimental Psychology, 46, 199-210.

Fitts, P., & Switzer, G. (1962). Cognitive aspects of information processing: I. The familiarity of S-R sets and subsets. Journal of Experimental Psychology, 63, 321-329.

Fitzgerald, P. G., & Picton, T. W. (1981). Temporal and sequential probability in evoked potential studies. Canadian Journal of Psychology, 35, 188-200.

Fleer, R. E. (1972). Speed of movement under two conditions of response initiation and retardates. Perceptual and Motor Skills, 35, 140-142.

Ford, J., Mobs, R., Pfefferbaum, A., & Kopell, B. (1980). On the utility of P3 latency for studying cognitive processes. In H. H. Kornhuber and L. Deeke (Eds.). Progress in Brain Research, 54, 661-667. Elsevier, Amsterdam.

Ford, J. M., Pfefferbaum, A., Tinklenberg, J., & Kopell, B. (1982). Effects of perceptual and cognitive difficulty on P3 and RT in young

and old adults. Electroencephalography and Clinical Neurophysiology, 54, 311-321.

Ford, J. M., Roth, W. T., & Kopell, B. S. (1976). Auditory evoked potentials to unpredictable shifts in pitch. Psychophysiology, 13, 32-29.

Friedman, D., Simson, R., Ritter, W., & Rapin, I. (1975). The late positive component (P300) and information processing in sentences. Electroencephalography and Clinical Neurophysiology, 38, 255-262.

Friedman, D., Vaughan, H., & Erlenmeyer-Kimling, L. (1978). Stimulus and response related components of the late positive complex in visual discrimination tasks. Electroencephalography and Clinical Neurophysiology, 45, 319-330.

Froeberg, S. (1907). The relation between the magnitude of stimulus and the time of reaction. Archives of Psychology, 8.

Goodin, D., & Aminoff, M. J. (1984). The relationship between the evoked potential and brain events in sensory discrimination and motor response. Brain, 107, 241-251.

Goodin, D., Squires, K., Henderson, B., & Starr, A. (1978). An early event-related cortical potential. Psychophysiology, 15 (4), 360-365.

Graham, D., & Adams, J. (1971). Ischaemic brain damage in fatal head injuries. Lancet, 1, 265-266.

Green, D. M., & Swets, J. A. (1966). Signal detection theory and psychophysics. New York: Wiley.

Greenberg, R. P., Becker, D. P., Miller, J. D., & Mayer, D. J. (1977). Evaluation of brain function in severe head trauma with multi-

modality evoked potentials, Part II. Localisation of brain dysfunction and correlation with post traumatic neurological condition. Journal of Neurosurgery, 47, 163-177.

Greenhouse, S. W., & Geisser, S. (1959). On method in the analysis of profile data. Psychometrica, 24, 95-112.

Grice, G. R. (1968). Stimulus intensity and response evocation. Psychological Review, 75, 359-373.

Grice, G. R., Nullmeyer, R., & Spiker, V. A. (1982). Human reaction time: toward a general theory. Journal of Experimental Psychology: General, 111, 135-153.

Gronwall, D., & Sampson, H. (1974). The psychological effects of concussion. Auckland, Auckland University Press.

Gurdjian, E. S., & Gurdjian, E. S. (1976). Head injuries: mechanisms, diagnosis and management. Boston: Little, Brown, and Company.

Hawthorne, V. (1978). Epidemiology of head injuries. Scottish Medical Journal, 23, 92.

Hawkins, H. L., MacKay, S. L., Holley, S. L., Friedin, B. D., & Cohen, S. L. (1973). Locus of the relative frequency effect in choice reaction time. Journal of Experimental Psychology, 101, 90-99.

Hay, R. K. (1967). Neurosurgical aspects of traffic accidents. Canadian Medical Association Journal, 97, 1365-1368.

Herman, V. A. (1906). The time of perception as a measure of differences in sensation. Archives of Philosophy, Psychology and Scientific Method, 8, 1-75.

Hick, W. E. (1952). On the rate of gain of information. Quarterly Journal of Experimental Psychology, 4, 11-26.

Hicks, L. H., & Birren, J. E. (1970). Aging, brain damage, and psychomotor slowing. Psychological Bulletin, 74, 377-396.

Hillyard, S. A., & Kutas, M. (1983). Electrophysiology of cognitive processing. Annual Review of Psychology, 34, 33-61.

Hillyard, S., Squires, K. C., Bauer, J., & Lindsay, P. (1971). Evoked potential correlates of auditory signal detection. Science, 172, 1357-1360.

Hinrichs, J. V., & Krainz, P. L. (1970). Expectancy in choice reaction time: anticipation of stimulus or response. Journal of Experimental Psychology, 25, 330-334.

Hoffman, J., Houck, M., MacMillan, F. W. III, Simons, R., & Oatman, L. (1985). Event-related potentials elicited by automatic targets: A dual-task analysis. Journal or Experimental Psychology: Human Perception and Performance, 11, (1), 50-61.

Hoffman, J., Simons, R., & Houck, M. (1983). Event-related potentials during controlled and automatic target detection. Psychophysiology, 20 (6), 625-632.

Holbourn, A. (1943). Mechanics of head injuries. Lancet, 2, 438-441.

Holmes, J. L. (1926). RT to photometrically equal chromatic stimuli. American Journal of Psychology, 37, 414-417.

Hovland, C. I. (1936). The influence of adaptation illumination upon visual reaction time. Journal of General Psychology, 14, 346-359.

Hovland, C. I. (1937). The generalization of conditioned responses. II. The sensory generalization of conditioned responses with varying intensities of tone. Journal of Genetic Psychology, 51, 279-290.

Hyman, R. (1953). Stimulus information as a determinant of reaction time. Journal of Experimental Psychology, 45, 188-196.

Isreal, J., Chesney, G., Wicken, C., & Donchin, E. (1980). P300 and tracking difficulty: Evidence for multiple resources in dual-task performance. Psychophysiology, 17, 259-273.

Isreal, J. B., Wickens, C. D., Chesney, G. L., & Donchin, E. (1980). The event-related potential as an index of display monitoring workload. Human Factors, 22, 211-224.

Jasper, H. H. (1958). The ten twenty electrode system of the International Federation. Electroencephalography and Clinical Neurophysiology, 10, 371-375.

Jennett, B., & Bond, M. (1975). Assessment of outcome after severe brain damage. Lancet, 1, 480-487.

Johanson, A. (1922). The influence of incentive and punishment upon reaction time. Archives of Psychology, 54.

Johnson, D. M. (1939). Confidence and speed in the two-category judgement. Archives of Psychology, 34, 1-53.

Johnson, R. V. (1986). A triarchic model of P300 amplitude. Psychophysiology, 23, 367-384.

Johnson, R., & Donchin, E. (1978). On how P300 amplitude varies with the utility of the eliciting stimuli. Electroencephalography and Clinical Neurophysiology, 44, 424-437.

Johnson, R., & Donchin, E. (1980). P300 and stimulus categorization: Two plus one is not so different from one plus one. Psychophysiology, 17, 167-178.

Johnson, R., & Donchin, E. (1982). Sequential expectancies and decision making in a changing environment: An electrophysiological approach. Psychophysiology, 19 (2), 183-200.

Johnston, V. S., Miller, D., & Burleson, M. (1986). Multiple P3s to emotional stimuli and their theoretical significance. Psychophysiology, 23 (6), 684-694.

Jokobsen, J., Baadsgaard, S. E., Thomsen, S., & Henriksen, P. B. (1987). Prediction of post-concussional sequelae by reaction time test. Acta Neurologica Scandinavia, 75, 341-345.

Kalsbeak, W., McLaurin, R., Harris, B., & Miller, J. (1980). The national head and spinal cord injury survey: Major findings. Journal of Neurosurgery, 53, 19-31.

Karis, D., Chesney, G., & Donchin, E. (1983). "... 'twas ten to one; and yet we ventured ...": P300 and decision making. Psychophysiology, 20 (3), 260-268.

Kaufman, H., Lamb, J. C., & Walber, J. R. (1970). Prediction of choice reaction time from information of individual stimuli. Perception and psychophysics, 7, 263-266.

Kellog, W. N. (1931). Time of judgement in psychometric measures. American Journal of Psychology, 43, 65-86.

Kempt, E. H., Coppée, G. E., & Robinson, E. H. (1937). Electric

responses of the brainstem to unilateral auditory stimulation. American Journal of Physiology, 120, 304-322.

Kerkhof, G., & Uhelenbroek, J. (1981). P300 latency in threshold signal detection. Biological Psychology, 13, 89-105.

Kerr, T., Kay, D., & Lossman, L. (1971). Characteristics of patients, type of accident, and mortality in a consecutive series of head injuries admitted to a neurosurgical unit. British Journal of Preventative Social Medicine, 25, 179-185.

King, H. E. (1962). Psychomotor indications of behaviour disorder arising from neurologic trauma and disease. Psychiatric Communications, 5, 31- 35.

King, H. E. (1975). Psychomotor correlates of behaviour disorder. In M. Kietzman, S. Sutton, and J. Zubin (Eds.): Experimental approaches to psychopathology. New York: Academic Press.

Kinsbourne, M. (1977). Cognitive decline with advancing age: An interpretation. In W. Lynn Smith and W. Kinsbourne (Eds.): Aging and Dementia. New York: Spectrum Publications, pp. 217-235.

Klatzky, R. L., & Smith, E. E. (1972). Stimulus expectancy and retrieval from short-term memory. Journal of Experimental Psychology, 94, 101-107.

Klensch, H. (1973). Die diagnostiche Valenz der Reaktionzeitmessung bei verschiedenen zerebralen erkrankungen. Fortsch, Nerol. Psychiat., 41, 575.

Klonoff, H. (1971). Head injuries in children: predisposing factors,

accident conditions, accident proneness and sequelae. American Journal of Public Health, 61, 2405-2417.

Kornblum, S. (1969). Sequential dependencies as a determinant of choice reaction time: A summary. In W. G. Koster (Ed.): Attention and Performance II (pp. 54-57). North-Holland, Amsterdam.

Kornblum, S. (1973). Sequential effects in choice reaction time: a tutorial review. In S. Kornblum (Ed.): Attention and Performance II, (pp. 54-57). New York: Academic Press.

Kornblum, S. (1975). An invariance of choice reaction time with varying numbers of alternative and constant probability. In P.M.A. Rabbit and S. Dornic (Eds.): Attention and Performance, V, (pp. 366-382). New York: Academic Press.

Kraus, J. F. (1980a). A comparison of recent studies on the extent of the head and spinal cord injury problems in the United States. Journal of Neurosurgery (Suppl.), 53, 3-10.

Kraus, J. F. (1980b). Injury to the head and spinal cord: The epidemiological relevance of the medical literature published from 1960 to 1978. Journal of Neurosurgery (Suppl.), 53, 3-10.

Krinchick, E. P. (1969). The probability of a signal as a determinant of reaction time. In W. G. Koster (Ed.): Attention and Performance II (pp. 27-36). North-Holland, Amsterdam.

Krinchick, E. P., & Aleksandrova, L. N. (1966). O Soatnoshenii vremonnoj: alternatiunej neopredelennosti v uslovijakh peredachi informatsii chelovekom. Vasprasy psykhologii, 2, 25-34.

Kutas, M., McCarthy, G., & Donchin, E. (1977). Augmenting mental chronometry: The P300 as a measure of stimulus evaluation time. Science, 197, 792-795.

Lamb, J. C., & Kaufman, H. (1965). Information transmission with unequally likely alternative. Perceptual and Motor Skills, 21, 255-259.

Lappin, J., & Disch, K. (1972). The latency operating characteristic: I. Effects of stimulus probability on choice reaction time. Journal of Experimental Psychology, 92 (3), 419-427.

Lemmon, V. W. (1927). The relation of reaction time to measures of intelligence, memory and learning. Archives of Psychology, 94.

Leonard, J. A. (1961). Choice reaction time experiments and information theory. In C. Cherry (Ed.): Information Theory (pp. 129-146). Butterworths, London.

Levin, H. Benton, A., & Grossman, R. (1982). Neurobehavioural Consequences of Closed Head Injury. New York: Oxford University Press.

Lindenberg, R. (1971). Trauma of meninges and brain. In J. Minckler (Ed.): Pathology of the Nervous System, Vol. 2. New York: McGraw-Hill, pp. 1705-1765.

Link, S. W., & Tindall, A. D. (1971). Speed and accuracy in comparative judgements of line length. Perception and Psychophysics, 9, 284-288.

Loveless, N. E., & Sanford, L. J. (1974). Effects of age on the contingent negative variation and preparatory set in a reaction time task. Journal of Gerontology, 29, 52-63.

Luce, R. D., & Green, D. (1972). A neural timing theory for response times and the psychophysics of intensity. Psychological Review, 79, 14-57.

Luria, A. R. Higher Cortical Functions in Man, 2nd ed. 1980. New York: Basic Books, Inc.

MacFlynn, G., Montgomery, E., Fenton, G., & Rutherford, W. (1984). Measurement of reaction time following minor head injury. Journal of Neurology, Neurosurgery and Psychiatry, 47, 1326-1331.

Magliero, A., Bashore, T., Coles, M., & Donchin, E. (1984). On the dependence of P300 latency on stimulus evaluation processes. Psychophysiology, 21, 169-186.

Mansfield, R. J. W. (1973). Latency functions in human vision. Vision Research, 13, 2219-2234.

Marshall, L. F., Becker, D. P., Bowers, S. A., Cayard, C., Eisenberg, H., Gross, C. R., Grossman, R. G., Jane, J. A., Kunitz, S. C., Rimel, R., Tabaddor, K., & Warren, J. (1983). The national coma data bank. Part 1: Design purpose, goals and results. Journal of Neurosurgery, 59, 276-284.

Marshall, W. H., Talbot, S. A., & Ades, H. W. (1943). Cortical response of the anaesthesized cat to gross photic and electrical afferent stimulation. Journal of Neurophysiology, 6, 1-15.

Martinius, J., Hoffman, R., Mayer, F. X., & Kiepera, K. (1977). Hirnleistung-sswache mach scha delhirntrauma in kindesalter. Mschr: Kinderheilk, 125, 401-403.

McCallum, W. C., & Cummins, B. (1973). The effects of brain lessions on the contingent negative variation in neurosurgical patients. Electroencephalography and Clinical Neurophysiology, 35, 449-456.

McCarthy, G., & Donchin, E. (1981). A metric for thought: A comparison of P300 latency and reaction time. Science, 211, 77-80.

McGill, W. J. (1961). Loudness and RT. Acta Psychologica, 15, 193-199.

McGill, W. J. (1963). Stochastic latency mechanism. In R. D. Luce, R. R. Bush, & E. Galanter (Eds.): Handbook of Mathematical Psychology, Vol. I. New York: Wiley.

McLelland, J. R. (1979). On time relations and mental processes: a framework for analyzing processes in cascade. Psychological Reviews, 86, 287-330.

Merkel, J. (1885). Die zeitlichen Verhaltnisse der Willensthatigkeit. Philosophische Studien, 2, 73-127.

Meyer, D., Yantis, S., Osman, A., & Smith, J. (1985). Temporal properties of human information processing: Tests of discrete versus continuous models. Cognitive Psychology, 17, 445-518.

Michalewski, H., Prasher, D., & Starr, A. (1986). Latency variability and temporal interrelationships of the auditory event-related potentials (N1, P2, N2, and P3) in normal subjects. Electroencephalography and Clinical Neurophysiology, 65, 59-71.

Miller, E. (1970). Simple and choice reaction time following severe head injury. Cortex, 6, 121-127.

Miller, J. (1982). Discrete versus continuous stage models of human information processing. In search of partial output. Journal of

Experimental Psychology: Human Perception and Performance, 8, 273-296.

Miller, J. (1983). Can response preparation begin before stimulus recognition finishes? Journal of Experimental Psychology: Human Perception and Performance, 9 (2), 161-182.

Miller, J., Becker, D., Ward, J., Sullivan, H., Adams, W., & Rosner, M. (1977). Significance of intracranial hypertention in severe head injury. Journal of Neurosurgery, 47, 503-516.

Miller, J. D., Sweet, R. C., Navayan, R., & Becker, D. P. (1978). Early insults to the injured brain. Journal of the American Medical Association, 240, 439-442.

Miller, J. O., & Pachella, R. G. (1973). Locus of stimulus probability effect. Journal of Experimental Psychology, 101, 227-231.

Milner, D. (1986). Chronometric analysis in neuropsychology. Neuropsychologia, 24 (1), 115-128.

Morin, R. E., & Forin, B. (1962). Mixing of two types of S-R associations in a choice reaction time task. Journal of Experimental Psychology, 64, 137-141.

Morin, R. E., & Forin, B. (1963). Response equivocation and reaction time. Journal of Experimental Psychology, 66, 30-36.

Morin, R. E., & Forin, B. (1965). Information-processing: Choice reaction times of first- and third-grade students for two types of associations. Child Development, 56, 713-720.

Morin, R. E., Konick, A., Troxell, N., & McPherson, S. (1965).

Information and reaction time for "naming" responses. Journal of Experimental Psychology, 70, 309-314.

Mowbray, G. H., & Rhoades, M. V. (1959). On the reduction of choice reaction times with practice. Quarterly Journal of Experimental Psychology, 11, 16-23.

Mulder, G., Gloerich, A., Brookhuis, K., van Dellen, H., & Mulder, L. J. M. (1984). Stage analysis of the reaction process using brain-evoked potentials and reaction time. Psychological Research, 46, 15-32.

Naatanen, R., Simpson, M., & Loveless, N. (1982). Stimulus deviance and evoked potentials. Biological Psychology, 14, 53-98.

Nettelbeck, T. (1980). Factors affecting reaction time: mental retardation, brain damage, and other psychopathologies. In A. T. Welford (Ed.): Reaction Times (pp. 355-401). Academic Press. London.

Nickerson, R. S. (1969). Same different response times a model and a preliminary test. In W. G. Koster (Ed.): Attention and Performance II (pp. 257-275). North-Holland, Amsterdam.

Norrman, B., & Svahn, K. (1961). A follow-up study of severe brain injuries. Acta Psychiatrica Scandinavia, 37, 236-264.

Okita, T., Wijers, A., Mulder, G., & Mulder, L. J. M. (1985). Memory search and visual spatial attention: An event-related brain potential analysis. Acta Psychologica, 60, 263-292.

Ommaya, A. K., & Gennarelli, T. A. (1974). Cerebral concussion and

transmatic unconsciousness: Correlation of experimental and clinical observations on blunt head injuries. Brain, 97, 633-654.

Ommaya, A., Grubb, R., & Naumann, R. (1977). Coup and contre-coup injury: observations on the mechanics of visible brain injuries in the rhesus monkey. Journal of Neurosurgery, 35, 503-516.

Pachella, R. G. (1974). The interpretation of reaction time in information processing research. In Kantowita, B. (Ed.): Human information processing: Tutorials in performance and cognition. Erlbaulm, New York, 41-82.

Pachella, R. C., & Pew, R. W. (1968). Speed-accuracy tradeoff in reaction time: effect of discrete criterion times. Journal of Experimental Psychology, 76, 19-24.

Papanicolaou, A. C. (1987). Electrophysiological methods for the study of attentional deficits in head injury. In H. S. Levin, J. Gratman and H. M. Eisenberg (Eds.): Neurobehavioural Recovery From Head Injury. New York: Oxford University Press.

Papanicolaou, A. C., Loring, D. W., & Eisenberg, H. M. (1985). Stimulus offset P300 and temporal resolution of uncertainty. International Journal of Psychophysiology, 3, 29-31.

Pfefferbaum, A., Ford, J. M., Wenegrat, B. G., Roth, W. T., & Kopell, P. S. (1983). Manipulation of P3 latency: speed vs. accuracy instructions. Electroencephalography and Clinical Neurophysiology, 55, 188-197.

Picton, T., & Hillyard, S. (1974). Human auditory evoked potentials.

II: Effects of attention. Electroencephalography and Clinical Neurophysiology, 36, 191-199.

Pieron, H. (1920). Nouvelles recherches sur l'analyse du temps de latence sensorielle et sur la loi qui relie ce temps à l'intensité de l'excitation. Année Psychologique, 22, 58-142.

Pieron, H. (1936). Recherches expérimentales sur la sensation vibratoire cutanée. Année Psycho., 36, 88-102.

Pike, A. R. (1968). Latency and relative frequency of response in psychophysical discrimination. British Journal of Mathematical and Statistical Psychology, 21, 161-182.

Polich, J. (1986). Normal variation of P300 from auditory stimuli. Electroencephalography and Clinical Neurophysiology, 65, 236-240.

Polich, J. (1986b). Attention, probability, and task demands as determinants of P300 latency from auditory stimuli. Electroencephalography and Clinical Neurophysiology, 63, 251-259.

Porjesz, B., Begleiter, H., Bihari, B., & Kissin, B. (1987). The N2 component of the event-related brain potential in abstinent alcoholics. Electroencephalography and Clinical Neurophysiology, 66, 121-131.

Posner, M. I. (1978). Chronometric explorations of mind. Hillsdale, N.J.: Erlbaum.

Prigatano, G. P. (1987). Psychiatric and Psychosocial Disturbance in Head Injury. In H. S. Levin, J. Gratman and H. M. Eisenberg (Eds.): Neurobehavioural Recovery From Head Injury. New York: Oxford University Press, pp. 215-231.

Pritchard, W. S. (1981). Psychophysiology of P300. Psychological Bulletin, 90, 506-540.

Pudenz, R., & Shelden, C. (1946). The lucite calvarium - A method for direct observation of the brain; II. Cranial trauma and brain movement. Journal of Neurosurgery, 3, 487-505.

Raab, D. H. (1962). Effects of stimulus-duration on auditory reaction-time. American Journal of Psychology, 75, 298-301

Rabbitt, P. M. A. (1959). Effects of independent variations in stimulus and response probability. Nature, 183, 1212.

Rabbitt, P. M. A. (1979). How young and old subjects monitor and control responses for accuracy and speed. British Journal of Psychology, 70, 305-311.

Rabbitt, P. M. A. (1981). Sequential reactions. In D. H. Holding (Ed.): Human Skills. Wiley, London, 153-175.

Ragot, R. (1984). Perceptual and motor space representation: An event-related potential study. Psychophysiology, 21 (2), 159-170.

Ragot, R., & Renault, B. (1981). P300 as a function of S-R compatability and motor programming. Biological Psychology, 13, 289-294.

Ragot, R., & Renault, B. (1985). P300 and S-R compatability: A reply to Magliero et al. Psychophysiology, 22, 349-352.

Ratcliff, R. A. (1978). A theory of order relations in perceptual matching. Psychological Review, 88, 552-572.

Renault, B., Ragot, R., Leseure, N., & Remond, A. (1982). Onset and

offset of brain events as indices of mental chronometry. Science, 215, 1413-1415.

Restle, F. (1961). Psychology of judgment and choice. New York: Wiley.

Rimel, R., & Jane, J. (1983). Characteristics of the head-injured patient. In Rosenthal, M., Griffith, E., Bond, M., & Miller, D. (Eds.): Rehabilitation of the head injured adult (pp 9-21). Philadelphia, F.A. Davis.

Ritter, W., Ford, J., Gaillard, A., Harter, M., Kutas, M., Naatanen, R., Polich, J., Renault, B., & Rohrbaugh, J. (in press). Cognition and event-related potentials I. The relation of negative potentials and cognitive processes. In J. Cohen, R. Karres and P. Tueting (Eds.): Annals of the New York Academy of Sciences, 24-38.

Ritter, W., Simson, R., & Vaughan, H. (1972). Association cortex potentials and reaction time in auditory discrimination. Electroencephalography and Clinical Neurophysiology, 33, 547-555.

Ritter, W., Simson, R., Vaughan, H., Friedman, D. (1979). A brain event related to the making of a sensory discrimination. Science, 203, 1358-1361.

Ritter, W., Simson, R., Vaughan, H. (1983). Event-related potential correlates of two stages of information processing in physical and semantic discrimination tasks. Psychophysiology, 20 (2), 168-179.

Ritter, W., Vaughan, H. G., & Costa, L. D. (1968). Orienting and habituation to auditory stimuli: A study of short term changes in averaged evoked responses. Electroencephalography and Clinical Neurophysiology, 25, 550-556.

Rizzo, P. A., Amabile, G., Caporali, M., Spadora, M., Zanac, M., & Morocatti, C. (1978). A CNV study in a group of patients with traumatic head injuries. Electroencephalography and Clinical Neurophysiology, 45, 281, 285.

Rohrbaugh, J., Donchin, E., & Eriksen, C. (1974). Decision making and the P300 component of the cortical evoked response. Perception and Psychophysics, 15 (2), 368-374.

Roth, W. T., Blowers, G. H., Doyle, C. M., & Kopell, B. S. (1982). Auditory stimulus intensity effects on components of the late positive complex. Electroencephalography and Clinical Neurophysiology, 54, 132- 146.

Roth, W., Ford, J., & Kopell, B. (1978). Long-latency evoked potentials and reaction time. Psychophysiology, 15, 17-23.

Rosenthal, M., Griffith, E., Bond, M., & Miller, D. (1983) (Eds.) Rehabilitation of the head injured adult. Philadelphia, F. A. Davis.

Ruchkin, D. S., & Sutton, S. (1978). Equivocation and P300 amplitude. In D. Otto (Ed.): Multidisciplinary perspectives in event-related brain potential research. Washington, 175-178.

Ruchkin, D. S., Sutton, S., Munson, R., Silver, K., & Macar, F. (1981). P300 and feedback provided by the absence of the stimulus. Psychophysiology, 18, 271-282.

Ruesch, J. (1944). Intellectual impairment in head injuries. American Journal of Psychiatry, 100, 480-496.

Ruesch, J. (1944b). Dark adaptation, negative after images,

tachistocopic examinations and reaction time in head injuries. Journal of Neurosurgery, 1, 243-251.

Russell, W. (1947). The neurology of brain wounds. British Journal of Surgery and War Surgery (Suppl.), 1, 250-252.

Saunders, A. F. (1977). Structural and functional aspects of the reaction process. In S. Dornic (Ed.): Attention and Performance, VI, pp. 3-25. Hillsdale, N. J., Erlbaum.

Schneider, W., Shiffrin, R. (1977). Controlled and automatic human information processing: I. Detection, search, and attention. Psychological Reviews, 84 (1), 1-66.

Shallice, T. (1982). Specific impairments of planning. In D. E. Broadbent and L. Weiskrantz (Eds.): The Neuropsychology of Cognitive Function. London: The Royal Society, pp. 199-209.

Simson, R., Vaughan, H., & Ritter, W., (1976). The scalp topography of potentials associated with missing visual or auditory stimuli. Electroencephalography and Clinical Neurophysiology, 42, 528-535.

Smith, E. E. (1968). Choice reaction time: an analysis of the major theoretical postions. Psychological Bulletin, 70, 77-110.

Snyder, E., Hillyard, S., & Galambos, R. (1980). Similarities and differences among the P3 waves to detected signals in three modalities. Psychophysiology, 17 (2), 112-122.

Soetens, E., Boer, L., & Hueting, J. (1985). Expectancy or automatic facilitation? Separating sequential effects in two choice reaction time. Journal of Experimental Psychology: Human perception and Performance, 11 (5), 598-616.

Squires, K. C., Donchin, E., Herning, R., & McCarthy, G. (1977). On the influence of task relevance and stimulus probability on event-related potential components. Electroencephalography and Clinical Neurophysiology, 42, 1-14.

Squires, K. C., Hillyard, S. A., & Lindsay, P. H. (1973). Vertex potentials evoked during auditory signal detection: relation to decision criteria. Perception and Psychophysics, 14, 265-272.

Squires, N. K., Donchin, E., Squires, K. C., & Grossberg, S. (1977). Bisensory stimulation: Inferring decision-related processes from the P300 component. Journal of Experimental Psychology: Human Perception and Performance, 3, 199-315.

Squires, N. K., Duncan-Johnson, C. C., Squires, K. C., & Donchin, E. (1976). Cross-model sequential effects on the P300 component of the event-related potential. Psychophysiology, 14, 96 (Abstract).

Squires, N. K., Squires, K. C., & Hillyard, A. (1975). Two varieties of long-latency positive waves evoked by unpredictable auditory stimuli in man. Electroencephalography and Clinical Neurophysiology, 38, 387-401

Steadman, J. H., & Graham, J. G. (1970). Rehabilitation of the brain-injured. Proceedings of the Royal Society of Medicine, 63, 23-28.

Sternberg, S. (1964).. Estimating the distributions of additive reaction-time components. Paper presented at the meeting of the Psychonomic Society, Niagara Falls, Ontario, October.

Sternberg, S. (1969). The discovery of processing stages: Extensions

of Donders' method. In W. G. Kaster (Eds.): Attention and Performance II (pp. 276-315). Amsterdam: North Holland.

Strich, S. (1956). Diffuse degeneration of cerebral white matter in severe dementia following head injury. Journal of Neurology, Neurosurgery, and Psychiatry, 19, 163-185.

Strich, S. (1969). The pathology of brain damage due to blunt head injuries. In A. E. Walker and M. Critchley (Eds.): The late effects of head injury (pp. 501-526). Springfield: Charles C. Thomas.

Strich, S. (1970). Lesions in the cerebral hemispheres after blunt head injury. In S. Sevitt and H. Stener (Eds.): The pathology or trauma (pp. 166-171). London: BMA House.

Stroop, J. (1935). Studies of interference in serial verbal reactions. Journal of Experimental Psychology, 18, 643-662.

Stuss, D. T. (1987). Contribution of frontal lobe injury to cognitive impairment after closed head injury: Methods of assessment and recent findings. In H. S. Levin, J. Grafman and H. M. Eisenberg (Eds.): Neurobehavioural Recovery from Head Injury. New York: Oxford University Press, pp. 166-177.

Stuss, D. T., & Benson, D. F. (1984). Neuropsychology of the frontal lobes. Psychological Bulletin, 95 (1), 3-28.

Stuss, D. T., & Benson, D. F. (1986). The Frontal Lobes. New York: Raven Press.

Suffield, B. J. (1985). The Cognitive Psychophysiology of Diffuse Damage: Contingent Negative Variation (CNV) Evidence of Over- and

Underprocessing After Severe Closed Head Injury. Unpublished Doctoral Dissertation, University of Ottawa, Ottawa.

Sutton, S. (1979). P300 - thirteen years later. In H. Begleiter (Ed.): Evoked brain potentials and behavior. New York: Plenum Press.

Sutton, S., Braren, M., & Zubin, J. (1965). Evoked-potential correlates of stimulus uncertainty. Science, 150, 1187-1188.

Swanson, J. M., & Briggs, G. E. (1969). Information processing as a function of speed versus accuracy. Journal of Experimental Psychology, 81, 223-239.

Taylor, D. (1976). Stage analysis of reaction time. Psychological Bulletin, 83, 161-191.

Teasdale, G., & Mendelow, D. (1984). Pathophysiology of closed head injury. In N. Brooks (Ed.): Closed head injury psychological, social, and family consequences, (pp. 4-36). Oxford: Oxford University Press.

Theios, J. (1973). Reaction time measurements in the study of memory processes: Theory and data. In G. H. Bower (Ed.): The psychology of learning and motivation: Advances in research and theory, (Vol. 7, pp. 43-85). Academic Press, New York and London.

Theios, J. (1973). The components of response latency in simple human information processing tasks. In Rabbit P. M. A. and Dornic, S. (Eds.): Attention, and Performance V. Academic Press: London.

Theios, J. (1975). The components of response latency in simple human information processing tasks. In P. M. A. Rabbit and S. Dornic

(Eds.): Attention and performance V. (pp. 418-440). Academic Press, London.

Towey, J., Rist, F., Hakerem, G., Ruchkin, D., & Sutton, S. (1980). N250 latency and decision time. Bulletin of the Psychonomic Society, 15, 365-368.

Towle, V., Heuer, D., & Donchin, E. (1980). On indexing attention and learning with event-related potentials. Psychophysiology, 17, 291 (abstract).

Trinca, G. W. (1986). The influence of seat-belt use on road accident injury patterns. Australia and New Zealand Journal of Surgery, 56, 13-17.

Trunkey, D. (1983). Trauma. Scientific American, 249, 28-36.

Tueting, P., Sutton, S., & Zubin, J. (1970). Quantitative evoked potential correlates of the probability of events. Psychophysiology, 7, 385-394.

Turvey, M. T. (1973). On peripheral and central processes in vision: Inferences from an information processing analysis of masking with patterned stimuli. Psychological Review, 80, 1-52.

Uzzell, B. P., Obrist, W. D., Dolinskas, C. A., & Langfitt, T. W. (1986). Relationship of acute CBF and ICP findings to neuropsychological outcome in severe head injury. Journal of Neurosurgery, 65, 630-635.

van Dellen, H., Brookhuis, K. A., Mulder, G., Okita, T., & Mulder, L. S. (1984). Evoked potential correlates of practice in a visual search

task. In D. Papakostopoulos, (Eds.): Clinical and Experimental Neuropsychophysiology. Groom, Helm, Beckenham, pp. 132-155.

van Zomeren, A. H., Brower, W. H., & Deelman, B. G. (1981). Attentional defecits: The riddles of selectivity, speed and alertness. In A. H. van Zomeran (Ed.): Reaction time and attention after closed head-injury. Lisse: Swets and Zeitlinger B.V., pp. 69-118.

van Zomeran, A. H., & Deelman, B. G. (1981a). Differential effects of simple and choice reaction time after closed head-injury. In A. H. van Zomeren (Ed.): Reaction time and attention after closed head injury, (pp. 23-27). Lisse: Sets and Zeitlinger B.V.

van Zomeren, A. H., & Deelman, B. G. (1981b). Long term recovery of visual reaction time after closed head injury. In A. H. van Zomeren (Ed.): Reaction time and attention after closed head injury, (pp. 50-64). Lisse: Swets and Zeitlinger B.V.

Verbaten, M. N. (1983). The influence of information on habituation of cortical, autonomic and behavioral components of the orienting response. In A.W.K. Gailard and W. Ritter (Eds.): Tutorials in ERP research: Endogenous components.

Vickers, D. (1980). Discrimination. In A. T. Welford (Ed.): Reaction Times. New York: Academic Press, pp. 25-72.

Wade, M. G., Newell, K. M., & Wallace, S. A. (1978). Decision time and movement time as a function of response complexity in retarded persons. American Journal of Mental Deficiency, 83, 135-144.

Walter, W. G. (1964). Slow potentials in the human brain associated

with expectancy, attention and decision. Archives of Psychiatry, 206, 309- 322.

Weinberg, H., Walter, W. G., & Crow, H. J. (1970). Intracerebral events in human related to real and imaginery stimuli. Electroencephalography and Clinical Neurophysiology, 29, 1-9.

Weiss, A. (1965). The locus of reaction time change with set, motivation and age. Journal of Gerontology, 20, 60-64.

Welford, A. T. (Ed.) (1980). Reaction Times. New York: Academic Press.

Wells, G. R. (1913). The influence of stimulus duration on RT. Psychological Monographs, 15, 1066.

Williams, A. F., Peat, M. A., Crouch, D. A., Wells, J. K., & Finkle, B. S. (1985). Drugs in fatally injured young male drivers. U.S. Public Health Report, 100 (1), 19-25.

Winogron, H. S. W. (1986). Reaction times and speed-accuracy tradeoff strategies in head-injured and normal children. Unpublished doctoral dissertation. Carleton University, Ottawa.

Wood, C. C., & Jennings, J. R. (1976). Speed-accuracy tradeoff function in choice reaction time: experimental designs and computational procedures. Perception and Psychophysics, 9, 93-102.

Yellott, J. I., Jr., (1971). Correction for guessing and the speed-accuracy tradeoff in choice reaction time. Journal of Mathematical Psychology, 8, 159-199.

Appendix A

ANOVA Tables for Experiment 1

ANOVA 1.1

Reaction time to standard stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Group (G)	519426.27	1	519426.27	28.69	0.00
Error	181056.61	10	18105.66		
Instruction (I)	87960.88	1	87960.88	18.60	0.00
IG	21743.29	1	21743.29	4.60	0.06
Error	47297.13	10	4729.71		
Probability (P)	25820.88	1	25820.88	27.93	0.00
PG	2531.85	1	2531.85	2.74	0.12
Error	9243.81	10	924.38		
Replication (R)	3327.66	1	3327.66	0.90	0.36
RG	855.16	1	855.16	0.23	0.64
Error	32106.13	10	3716.41		
IP	37.04	1	37.04	0.03	0.86
Error	11885.31	10	1188.53		
IR	1285.92	1	1285.92	0.42	0.54
Error	32106.37	10	3210.63		
PR	163.81	1	163.81	10.72	0.00
Error	11363.73	10	1136.37		

ANOVA 1.2

Reaction time to target stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Group (G)	326626.02	1	326626.02	15.07	0.00
Error	216784.79	10	21678.47		
Instruction (I)	80696.64	1	80696.64	24.10	0.00
IG	10539.80	1	10539.80	3.15	0.10
Error	33482.73	10	3348.27		
Probability (P)	96.34	1	96.34	0.04	0.85
PG	3520.37	1	3520.37	1.29	0.28
Error	27296.97	10	2729.69		
IP	3735.26	1	3735.26	3.04	0.11
Error	12300.84	10	1230.08		
Replication (R)	3958.65	1	3958.65	1.22	0.29
RG	48.90	1	48.90	0.02	0.90
Error	32407.54	10	3240.75		
IR	6485.75	1	6485.75	2.24	0.16
Error	28933.55	10	2893.35		
PR	2031.37	1	2031.37	2.39	0.15
Error	8483.35	10	848.33		

ANOVA 1.3

P300 amplitude to standard stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Group (G)	1411.80	1	1411.80	14.90	0.00
Error	1042.45	11	94.76		
Probability (P)	125.87	1	125.87	8.38	0.01
PG	22.64	1	22.64	2.82	0.12
Error	165.26	11	15.02		
Instructions (I)	121.76	1	121.76	7.95	0.01
IG	43.14	1	43.14	2.82	0.12
Error	168.46	11	15.31		
IP	10.57	1	10.57	0.87	0.36
Error	132.96	11	12.08		
Replication (R)	1.28	1	1.28	0.04	0.83
Error	325.62	11	29.60		
PR	20.81	1	20.81	1.02	0.33
Error	224.88	11	20.44		
IR	20.97	1	20.97	0.84	0.37
Error	275.12	11	25.01		

ANOVA 1.4

P300 amplitude to target stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Group (G)	3223.73	1	3223.73	10.86	0.00
Error	3266.38	11	296.94		
Probability (P)	900.52	1	900.52	20.94	0.00
PG	150.09	1	150.09	3.49	0.08
Error	472.98	11	42.99		
Instruction (I)	26.95	1	26.95	1.39	0.26
IG	40.36	1	40.36	2.08	0.17
Error	213.54	11	19.41		
PI	81.89	1	81.89	4.15	0.06
PIG	20.20	1	20.20	1.02	0.36
Error	217.16	11	19.74		
Replication (R)	11.03	1	11.03	0.63	0.44
Error	194.02	11	17.63		
PR	6.87	1	6.87	0.40	0.53
Error	186.96	11	16.99		
IR	121.70	1	121.70	2.75	0.12
Error	486.99	1	43.27		

ANOVA 1.5

P300 latency to standard stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Group (G)	88308.05	1	88308.05	10.04	0.00
Error	96763.09	11	8796.64		
Probability (P)	1.46	1	1.46	0.00	0.96
PG	809.15	1	809.15	0.83	0.38
Error	10677.38	11	970.67		
Instruction (I)	398.90	1	398.90	0.88	0.36
IG	1898.90	1	1898.90	4.17	0.06
Error	5010.71	11	455.51		
IG	17.94	1	17.94	0.05	0.81
Error	3591.66	11	326.51		
Repliction (R)	23.44	1	23.44	0.04	0.84
Error	6263.09	11	569.37		
PR	915.75	1	915.75	2.07	0.17
Error	4863.09	11	442.09		
IR	132.23	1	132.23	0.29	0.60
Error	5077.38	11	461.58		

ANOVA 1.6

P300 latency to target stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability
Group (G)	87590.10	1	87590.10	12.33	0.00
Error	78171.42	11	7106.49		
Probability (P)	586.08	1	586.08	1.06	0.32
PG	247.61	1	247.61	0.45	0.51
Error	6102.38	11	554.76		
Instruction (I)	775.09	1	775.09	0.89	0.36
IG	375.09	1	375.09	0.43	0.52
Error	9559.52	11	869.04		
PI	557.14	1	557.14	0.66	0.43
Error	9342.85	11	849.35		
Replication (R)	0.00	1	0.00	0.00	0.00
Error	2600.00	11	236.36		
PR	267.03	1	267.03	0.55	0.47
Error	5321.42	11	483.76		
IR	1108.05	1	1108.05	1.65	0.22
Error	7388.09	11	671.64		

Appendix 2

ANOVA Tables for Experiment 2

ANOVA 2.1

d'

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser Probability
Group (G)	2.53	1	2.53	0.52	0.48	
Error	92.85	19	4.88			
Condition (C)	28.23	2	14.11	16.19	0.00	0.00
CG	0.81	2	0.40	0.47	0.63	0.60
Error	33.13	38	0.87			
Repliction (R)	0.42	1	0.42	0.75	0.39	
RG	0.69	1	0.69	1.24	0.27	
Error	10.60	19	0.55			
RS	1.85	2	0.92	0.86	0.43	0.42
Error	40.96	38	1.07			

ANOVA 2.2

Reaction time to standard stimuli

	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser Probability
Group (G)	312730.93	1	312730.93	72.24	0.00	
Error	77920.47	18	4328.91			
Condition (C)	60390.97	2	30195.48	5.07	0.01	0.01
CG	11164.69	2	5582.34	0.94	0.40	0.39
Error	214516.68	36	5958.79			
Repliction (R)	14277.03	1	14277.03	4.09	0.06	
RG	5560.91	1	5560.91	1.59	0.22	
Error	62760.92	18	3486.71			
RS	5813.36	2	2906.68	0.93	0.40	0.36
Error	111969.96	36	3110.27			

ANOVA 2.3

Reaction time to target stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser Probability
Group (G)	268448.14	1	268448.14	24.02	0.00	
Error	201141.71	18	11174.53			
Condition (C)	66817.06	2	33408.53	6.17	0.00	0.00
CG	1779.64	2	889.82	0.16	0.84	0.79
Error	194967.29	36	5415.75			
Repliction (R)	2945.64	1	2945.64	1.38	0.25	
RG	1008.84	1	1008.84	0.47	0.50	
Error	38360.84	18	2131.15			
RS	17409.10	2	8704.55	2.18	0.12	0.15
Error	143813.03	36	3994.80			

ANOVA 2.4

P300 amplitudes to standard stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser Probability
Group (G)	300.16	1	300.16	5.02	0.03	
Error	1016.45	17	59.79			
Condition (C)	9.97	2	4.98	0.51	0.50	0.58
CG	30.73	2	15.36	1.56	0.22	0.22
Error	335.02	34	9.85			
Repliction (R)	6.27	1	6.27	0.44	0.51	
RG	6.38	1	6.38	0.45	0.51	
Error	214.90	17	14.28			
RS	3.83	2	1.91	0.25	0.77	0.68
Error	260.25	34	7.65			

ANOVA 2.5

P300 amplitude to target stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser Probability
Group (G)	524.64	1	524.64	10.92	0.00	
Error	624.31	13	46.02			
Condition (C)	68.12	2	34.06	3.46	0.04	0.05
CG	1.37	2	0.68	0.07	0.93	0.91
Error	225.77	26	9.83			
Repliction (R)	19.22	1	19.22	2.31	0.15	
RG	1.56	1	1.56	0.19	0.67	
Error	108.36	13	8.33			
RS	4.22	2	2.11	0.18	0.83	0.80
Error	311.31	26	11.97			

ANOVA 2.6

P300 latency to standard stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser Probability
Group (G)	72237.48	1	72237.46	9.63	0.00	
Error	127556.81	17	7503.34			
Condition (C)	2213.25	2	1106.62	1.93	0.16	0.17
CG	250.09	2	125.04	0.22	0.80	0.74
Error	19458.23	34	572.30			
Repliction (R)	0.63	1	0.63	0.00	0.96	
RG	1291.86	1	1291.86	3.95	0.06	
Error	5556.81	17	326.87			
RS	378.32	2	189.16	0.51	0.60	0.58
Error	12503.69	34	367.75			

ANOVA 2.7

P300 latency to target stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser Probability
Group (G)	56572.40	1	56572.40	9.61	0.00	
Error	94160.93	16	5885.05			
Condition (C)	4657.74	2	2328.87	2.81	0.07	0.08
CG	2942.92	2	1471.46	1.78	0.18	0.19
Error	26484.84	32	827.65			
Repliction (R)	2091.01	1	2091.01	3.70	0.07	
RG	698.42	1	698.42	1.26	0.27	
Error	8875.64	16	554.72			
RS	3357.21	2	1678.60	1.37	0.26	0.26
Error	39103.89	32	1221.99			

Appendix 3

ANOVA Tables for Experiment 3

ANOVA 3.1

d'

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geiser
Mean	539.20	1	539.20	53.52	0.00	
Group (G)	0.36	1	0.36	0.04	0.85	
Error	120.90	12	10.07			
Replications (R)	0.01	1	0.01	0.03	0.87	
RG	0.11	1	0.11	0.18	0.68	
Error	7.83	12	0.65			
Conditions (C)	40.58	3	13.52	12.02	0.00	0.00
CG	1.02	3	0.34	0.30	0.82	0.78
Error	40.51	36	1.12			
RC	1.80	3	0.60	2.05	0.12	0.13
Error	10.53	36	0.29			

ANOVA 3.2

Reaction time to standards

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser
Mean	16520361.83	1	16520361.83	509.36	0.00	
Group (G)	283375.31	1	283375.31	8.74	0.00	
Error	551365.72	17	32433.27			
Replications (R)	18818.79	1	18818.79	3.05	0.09	
RG	8048.71	1	8048.71	1.30	0.26	
Error	104956.49	17	6173.91			
Conditions (C)	75880.41	3	25293.47	8.77	0.00	0.00
CG	615.50	3	205.16	0.07	0.97	0.89
Error	147060.02	51	2883.52			
RC	12842.37	3	4280.79	2.75	0.05	0.07
Error	79372.62	51	1556.32			

ANOVA 3.3

Reaction times to target stimuli

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser
Mean	13514703.23	1	13514703.23	973.30	0.00	
Group (G)	134477.48	1	134477.48	9.68	0.00	
Error	236051.63	17	13885.39			
Replications (R)	24634.80	1	24634.80	6.10	0.02	
RG	7267.71	1	7267.71	1.80	0.19	
Error	68690.52	17	4040.61			
Conditions (C)	64799.19	3	21599.73	9.28	0.00	0.00
C	1229.19	3	409.73	0.18	0.91	0.84
Error	118697.23	51	2327.39			
RC	8265.46	3	2755.15	2.19	0.10	0.11
Error	64122.31	51	1257.30			

ANOVA 3.4

P300 amplitude to standard stimili

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser
Mean	8243.46	1	8243.46	54.84	0.00	
Group (G)	488.73	1	488.73	3.25	0.09	
Error	2254.74	15	150.31			
Replications (R)	0.79	1	0.79	0.11	0.74	
RG	4.46	1	4.46	0.63	0.43	
Error	106.20	15	7.08			
Conditions (C)	21.12	3	7.04	0.71	0.55	0.52
CG	19.36	3	6.45	0.65	0.58	0.56
Error	448.24	45	9.96			
RC	2.36	3	0.78	0.20	0.89	0.85
Error	173.73	45	3.86			

ANOVA 3.5

P300 amplitude to targets

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser
Mean	10967.10	1	10967.10	103.72	0.00	
Group (G)	745.20	1	745.20	7.05	0.01	
Error	1586.06	15	105.73			
Replications (R)	1.85	1	1.85	0.17	0.68	
RG	3.64	1	3.64	0.33	0.57	
Error	164.67	15	10.97			
Conditions (C)	30.89	3	10.29	1.40	0.25	0.25
CG	40.21	3	13.40	1.82	0.15	0.16
Error	331.82	45	7.37			
RC	39.89	3	13.29	2.63	0.06	0.07
Error	227.60	45	5.05			

ANOVA 3.6

P300 latency to standard stimili

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser
Mean	15322657.84	1	15322857.84	1476.16	0.00	
Group (G)	52250.34	1	52250.34	5.03	0.03	
Error	166084.94	16	10380.30			
Replications (R)	1841.84	1	1841.84	2.14	0.16	
RG	351.56	1	351.56	0.41	0.53	
Error	13800.72	16	882.54			
Conditions (C)	2010.74	3	670.24	0.61	0.61	0.58
CG	742.68	3	247.56	0.22	0.87	0.84
Error	53003.94	48	1104.24			
RC	1620.64	3	540.24	0.74	0.53	0.50
Error	35164.61	48	732.59			

ANOVA 3.7

P300 latency to target stimili

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Probability	Greenhouse Geisser
Mean	14058369.14	1	14058369.14	1058.80	0.00	
Group (G)	46033.02	1	46033.02	3.47	0.08	
Error	199164.35	15	13277.62			
Replications (R)	5636.23	1	5636.23	5.48	0.03	
RG	0.00	1	0.00	0.00	0.99	
Error	15430.26	15	1028.68			
Conditions (C)	2014.94	3	671.64	0.50	0.68	0.60
CG	1427.77	3	475.92	0.36	0.78	0.69
Error	59973.55	45	1332.74			
RC	2451.64	3	817.21	0.90	0.44	0.42
Error	40752.09	45	905.60			