NEUROPSYCHOLOGICAL IMPAIRMENT IN CHILDREN FOLLOWING HEAD INJURY

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Declaration by Candidate

I hereby declare that this thesis is my own work and that it has not been submitted to any other University

Signed by candidate

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ABSTRACT

There is a high incidence of head injury in children, yet few studies have systematically studied cognitive outcome. This study was designed as a survey to (a) establish the nature of intellectual and neuropsychological deficits that occur after head injuries of differing severity in children aged 6 to 14 years, (b) establish the nature of recovery curves in the first year after injury, and (c) determine which medical and psychosocial factors are associated with poor cognitive outcome and which functions show persisting impairment.

From 1134 children admitted with head injury to Red Cross and Groote Schuur Hospitals during a 2 year period, a consecutive sample of all those who had post-traumatic amnesia (PTA) over 1 hour, a compound depressed or basal skull fracture, a seizure, or any evidence of neurological involvement, was collected (n=388). Further requirements that they should be between 6 and 14 years, English or Afrikaans speaking, and have no history of significant cerebral pathology or mental retardation, reduced the sample to 123 children. Severity groups were formed according to the length of PTA: 56 moderates (PTA less than 1 day), 40 severes (PTA 1 to 7 days), and 28 very severes (PTA more than 7 days). They were matched for age, sex, socioeconomic status and ethnic group with 46 controls who had traumatic injury not involving the head. Detailed accident, medical and psychosocial data were collected. The children were assessed on a battery of tests covering intelligence, language, motor speed, visuographic and memory functions, as soon as they were out of PTA (T1), 3 months later (T2), and at 1 year post-injury (T3). The 4 groups are compared at each interval on Tukey's studentized range test and the extent of recovery within and between the groups is compared by repeated measures analysis of variance.

The very severes score significantly lower than the controls and the other head-injured groups on almost all measures at all test occasions. Recovery is spread over both parts of the year (T1 to T2, and T2 to T3), but they do not reach control group levels. Just over 80% have scores below at least 95% of the controls on five or more of the neuropsychological measures at 1 year, 60% have a FSIQ below 80, 60% have defective memory, and about 40% have impaired language and visuomotor functions. This severity of injury is associated with persisting impairment.

The severes have defective scores relative to the controls on many measures at the initial and 3-4 month assessment. Visuographic abilities recover by the 3-4 month assessment and motor speed by 1 year. At 1 year, scores are still significantly lower than those of the controls on IQ, naming and memory measures. At 1 year, 18% have scores below 95% of the controls on five or more of the neuropsychological measures, 23% have a FSIQ below 80, 20-30% have language deficits, and 25% memory deficits. There is thus definite transient impairment, and about a quarter have persisting impairment.

The moderates have initial deficits relative to the controls on memory, IQ and the Purdue pegboards. Recovery, particularly on measures tapping attention and speed, occurs within the first 3 months. At 1 year, mean scores are significantly below those of the controls on IQ, memory and picture naming. Only 9% have persisting impairment on five or more neuropsychological measures and only 8% have a PIQ below 80.

Recovery in the head injured does not usually exceed in magnitude the gains shown by the control group, but the measures on which the head injured show gains and the timing of gains, differs. The very severes and the severes show significant recovery over both parts of the year, whereas the moderates and controls tend to have gains in the first 3

months only. In the very severes, recovery of some functions (eg memory and visuographic) ceases at 3-4 months, while recovery on complex motor measures may not begin before then.

Intelligence, memory and picture naming are the functions most sensitive to head injury and deficits on these measures persist in all the head-injured groups.

The relationship of medical and psychosocial data collected at intake to two outcome measures, PIQ and a Neuropsychological Impairment Index (NII), at 1 year was determined by multiple regression analysis. NII outcome was affected by more of the medical and fewer of the psychosocial variables than PIQ and showed a closer dose-response relationship with severity of injury as measured by PTA length.

The probability of a BAD NII outcome was increased as a function of PTA length, by neurosurgery, other surgery, complications, clinical evidence of a basal skull fracture, male sex and degree of psychosocial adversity, and decreased by the presence of a skull fracture on x-ray. Using these variables, NII outcome was correctly predicted in 89% of cases. Adding in neuropsychological variables measured at intake, NII outcome was correctly predicted in 96% of cases.

A low PIQ was predicted by long PTA, absence of a basal skull fracture, psychosocial adversity, absence of pre-school attendance, female sex and Afrikaans language. Medical and psychosocial variables at intake accounted for 41% of the variance in PIQ at 1 year. PIQ measured once out of PTA accounted for 74% of the variance in PIQ at 1 year.

Poor premorbid school progress contributed to the probability of a BAD NII outcome (particularly in the moderate and severe groups) and to a lower PIQ.

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GLOSSARY

Coma: "Not obeying commands, not uttering words, and not opening the eyes" (Jennett & Teasdale, 1981, p.80). Operationally defined as GCS $\leq$8.

CR: Consistent retrieval.

CT: Computerized tomography.

DAI: Diffuse axonal injury.

FSIQ: Full scale IQ.

GCS: Glasgow Coma Scale (Jennett & Teasdale, 1981).

GOS: Glasgow Outcome Scale (Jennett & Bond, 1975).
Satisfactory outcome:
Good Recovery, Moderately Disabled
Poor outcome:
Severely Disabled, Vegetative, Dead.

ICP: Intracranial pressure.

LH: Left hemisphere.

LTM: Long-term memory.

LTS: Long-term storage.

Mild head injury: coma, if any, lasting less than 6 hours; PTA less than 8 days.

MRI: Magnetic Resonance Imaging.

NII: Neuropsychological Impairment Index.

PIQ: Performance IQ.

PTA: Post-traumatic amnesia
Moderate: less than 24 hours.
Severe: 1 to 7 days.
Very Severe: more than 7 days.

PTD: Post-traumatic disorientation.

PVS: Persistent vegetative state.

RH: Right hemisphere.

SAH: Subarachnoid haemorrhage.

SES: Socioeconomic status.

Severe head injury: coma lasting 6 or more hours; PTA over 7 days.

STM: Short-term memory.

T1, T2, T3: Test 1, Test 2, Test 3.

UCT: University of Cape Town.

VIQ: Verbal IQ.

WISC-R: Wechsler Intelligence Scale for Children-Revised (Wechsler, 1974).

1. INTRODUCTION

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1.1 Background

There is a high incidence of head injury in children yet few studies have systematically examined the nature of neuropsychological deficits and patterns of recovery. It has been estimated that 1 in 10 children in the United Kingdom suffer a serious accident every year and head injury is the major cause of admission to hospital after trauma (Craft, Shaw & Cartlidge, 1972). In studies from Israel, England and Canada, it has been estimated that from 50 to 75% of all paediatric hospital admissions for trauma are for head trauma (Attias, Tal, Winter & Jaffe, 1982; Jamison & Kaye, 1974; Klonoff, 1971). In the USA, head injury is the cause of approximately 100,000 paediatric hospital admissions per year (Kraus, Fife & Conroy, 1987) and the average annual incidence rate for children under 15 has been estimated as 220 per 100,000 (Annegers, 1983).

Accidents are the major cause of death in children after the first year of life and head injury is the major contributor to this mortality (Craft, et al, 1972; Divisional Council of the Cape Mortality Statistics, 1982). A recent 15 year retrospective survey of non-natural deaths in children under 15 in greater Cape Town found that, excluding those where multiple injuries were given as the cause of death, 25% of deaths were attributable to head injury alone (Knobel, de Villiers, Parry & Botha, 1984). Mortality rates for children receiving head injuries are lower than those in adults, ie of the order of 10 to 30% versus 30 to 50%, (Bruce, Schut, Bruno, Woods & Sutton, 1978; Jennett, et al, 1977), and this raises the question of increased morbidity among the survivors.

Varying rates of intellectual impairment, neuropsychological deficits and/or scholastic failure have been reported following head injury. Some investigators (eg Bruce, 1983), have claimed that most children will make normal school progress after even severe injury, whereas others (eg Klonoff, Low & Clark, 1977) have reported that almost a quarter of children with relatively minor head injury still have neuropsychological deficits 5 years after injury. Two weeks of unconsciousness has been regarded as the cut-off point beyond which normal school progress is unlikely (Heiskanen & Kaste, 1974; Vapalahti, cited Leary, 1982), yet "mental restitution" following even 4 weeks of coma has been reported (Carlsson, von Essen & Löfgren, 1968).

1.2 Methodological Differences

The difficulties in reaching consensus about the consequences of head injury have, at least in part, been attributable to variations in research methodology. Studies have differed in terms of the samples considered, the intervals at which follow-up was done, whether the design was prospective or retrospective, the use of a control group, the tests used, the methods of statistical analysis, and hence in their conclusions. The samples studied have varied in terms of the population served by the hospital; in whether admissions were consecutive or selected; the severity of injury included and the lesions excluded within severity categories; the age range included, and in whether those with previous head injury or other cerebral pathology, psychiatric conditions, and/or school failure were excluded (Dikmen, Reitan & Temkin, 1983; Gulbrandsen, 1984; Levin, Benton & Grossman, 1982; Williams, Gomes, Drudge & Kessler, 1984).

In the English literature, there are only two prospective studies of neuropsychological impairment in children following head injury which have defined their samples clearly, reassessed patients at regular intervals and employed a control group. Rutter, Chadwick and co-workers in Britain followed up a mild and a severely head-injured group with matched controls for two and a quarter years (Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Chadwick, Rutter, Shaffer, Shrout, 1981; Rutter, Chadwick & Shaffer, 1984; Rutter, Chadwick, Shaffer & Brown, 1980). (This study is referred to as the Chadwick-Rutter study, 1981, hereafter). They concluded that children with a post-traumatic amnesia (PTA) exceeding 3 weeks in duration tended to have persistent intellectual impairment, those with PTA between 2 and 3 weeks had mostly transient impairment, and only 10% of those with PTA less than 2 weeks had even transient impairment.

Klonoff and co-workers in Vancouver retested a large group of paediatric head injuries and matched controls at yearly intervals for 5 years (Klonoff & Low, 1974; Klonoff & Paris, 1974; Klonoff, Low & Clark, 1977). Their consecutive admission sample has to be regarded as predominantly a mildly head-injured group as the majority was not unconscious following head injury, only 3% were unconscious between 1 and 24 hours and only 4% were unconscious over 24 hours. Their findings suggest that there is both transient and persistent neuropsychological impairment in the mildly head-injured. Firm conclusions cannot be drawn from the study, however, as the results were not analysed according to severity and 50% dropped out over the follow-up period.

Studies with controls, but which followed subjects retrospectively at irregular intervals, may indicate the extent of deficits attributable to head injury (eg Colam, 1984; Dencker, 1960; Rune, 1970), but do not allow comment

on the time course of recovery. Studies with no controls, but groups with different severities of injury (eg Levin & Eisenberg, 1979a,b; Winogron, Knights & Bawden, 1984) allow some consideration of how severe the head injury must be in order to produce deficits, although estimating the status of the mildly head-injured from test norms may not be appropriate (Brooks et al, 1984; Dikmen, McLean & Temkin, 1986) and the chronicity of deficits has tended to range from days to years in these studies.

In studies with no controls but a fixed assessment interval, it is possible to consider the time span of recovery (eg Black, Blumer, Wellner & Walker, 1971) or the persistence of deficits (eg Flach & Malmros, 1972; Rowbotham, MacIver, Dickson & Bousfield, 1954), but it is not possible to determine to what extent improvement is attributable to recovery from head injury rather than other factors, or the extent to which deficits, learning and/or behaviour problems exceed those which would have occurred in such samples in the absence of head injury.

1.3 Aims

1. To establish the nature of the intellectual and neuropsychological deficits that occur after head injury in children aged 6 to 14 years.

    a. How severe must the head injury be to produce deficits?

    b. What functions are affected by head injury?

2. To develop recovery curves for the first year of injury showing the improvement of such deficits.

    a. Is the rate of improvement in the head injured greater than can be attributed to practice or maturation alone?

b. Do different severities of injury improve at different rates?

c. What is the rate and magnitude of improvement of specific abilities?

d. Is there a return to the pre-injury state?

3. To determine which factors predict the nature and extent of persistent residual neuropsychological deficits.

a. Which medical and psychosocial factors are associated with poor neuropsychological outcome?

b. Which functions show persistent impairment?

The intention of the present study is to analyse the performance of head-injured children on a battery of intelligence and neuropsychological tests and to attempt to answer the questions formulated above. The remainder of this chapter deals with general issues and specific methodological problems encountered in answering these questions, and leads to the formulation of hypotheses.

1.4 Factors Influencing Outcome

The outcome of head injury is influenced by three sets of factors: the degree of primary injury, the effect of secondary insults as systemic and intracranial mechanisms come into play, and the premorbid status of the individual receiving the head injury (Bruce, 1983; Miller, 1983). The next three chapters consider these factors in detail. There is an increasing tendency to classify brain damage as focal or diffuse. Adams, Doyle, Graham, Lawrence and McLellan (1986), cite surface contusions, intracranial haematomas and damage due to high intracranial pressure (ICP), with shift and herniation of the brain as the principal types of focal damage, while diffuse axonal injury (DAI), diffuse hypoxic brain damage and diffuse brain swelling constitute the diffuse forms of brain damage following trauma to the head.

It has been asserted that the severity of the diffuse brain injury rather than the presence or lateralization of a focal lesion is the primary determinant of recovery in children and adolescents (Levin, Eisenberg, Wigg & Kobayashi, 1982, p.672). Several authors have warned that estimates of laterality and attempts to relate neuropsychological deficits to the intrahemispheric site of a mass lesion may be inappropriate in severe head injury where focal damage is superimposed on diffuse or multifocal damage, the extent of which may not be neurologically or radiologically evident (Brooks, 1984, p.57; Chadwick, Rutter, Thompson & Shaffer, 1981, p.119; Levin, Benton & Grossman, 1982, p.7). The severity of the diffuse injury is measured by indices such as the depth and duration of coma and the length of PTA (see chapter 3). However, some researchers have been influenced by the presence of skull fractures or focal pathology rather than the length of the disturbance of consciousness, and have classified injury as severe or moderate rather than mild if there are intracranial lesions or focal neurological signs (eg Annegers, 1983; Ewing-Cobbs, Levin, Eisenberg & Fletcher, 1987; Winogron et al, 1984). Influenced by the International Data Bank studies, head injury is usually only classified as severe if there is coma persisting for at least 6 hours, which, in most cases, corresponds to a PTA in excess of 7 days (Jennett & Teasdale, 1981).

The Chadwick-Rutter study (1981) did not regard mild head injury as typically producing even transient cognitive impairment, but this does not correspond with the findings of other researchers in the child head-injuries field (eg Gulbrandsen, 1984; Levin & Eisenberg, 1979a,b; Winogron et al, 1984), or with findings in the adult head injury literature (eg Alves & Jane, 1985; Binder, 1986; Dikmen et al, 1986; Gronwall & Wrightson, 1974, 1981; Levin, Mattis, et al, 1987). One of the problems is that neither children

nor adults who receive head injuries are a random sample of the population. They tend to have a higher incidence of social disadvantage, scholastic backwardness and behaviour disturbance prior to their head injury (see chapter 4). Any direct relationship between brain and behaviour is moderated by a number of variables (Fletcher & Taylor, 1984). Without methodological control of psychosocial factors, it is difficult to determine whether problems found after the head injury are, in fact, attributable to the head injury. Evidence of a "dose-response" relationship, in which the magnitude of the deficit increases with the severity of injury, is needed (Brooks, 1987; Rutter et al, 1980). But, as Szatmari (1985) concluded:

> Any study then, in developmental neuropsychiatry, is incomplete unless measures of psychosocial disadvantage are also included in the equation. The effects of brain dysfunction should be compared with the effects of psychosocial disadvantage. The interaction of these two, whether additive, interactive, or synergistic, is an important question for future research (p.165).

1.5 The Patterns of Impairment

In terms of the functions affected by head injury, the literature on intellectual, language, visuomotor and memory impairment in children following head injury, is reviewed in chapters 5 to 8. Brain damage at any age has been regarded as having a greater effect on complex information processing, problem-solving, and new learning ("fluid intelligence") than on previously stored knowledge ("crystallized intelligence"), (Cattell, 1963; Hebb, 1942; Russell, 1986; Spreen, Tupper, Risser, Tuokko & Edgell, 1984). Children are still in the process of acquiring knowledge, vocabulary and problem-solving skills, so that brain damage may therefore impede intellectual development

(Chadwick & Rutter, 1984). As children have less stored knowledge and fewer learned strategies than adults, they have to rely more on fluid intelligence when taking intelligence tests, which may account for the greater sensitivity of intelligence tests to cognitive impairment in children than adults (Boll & Barth, 1981).

Rutter and colleagues (1984) have stated that, in the absence of some degree of global intellectual impairment, specific deficits other than in visuomotor speed are unusual after head injury. However, neither the Chadwick-Rutter study (1981) nor the study by Klonoff and colleagues (1977), included an adequate measure of memory functions. Studies that have done so, have found memory to be amongst the functions most seriously impaired both in the early months following head injury (Levin & Eisenberg, 1979a,b) and years later (Baracchini-Muratorio, Cantini & Pruneti, 1985; Gaidolfi & Vignolo, 1980). In adults with head injury, memory deficits have been shown to persist when intelligence has recovered (Conkey, 1938; Levin, Goldstein, High & Eisenberg, 1988; Stuss et al, 1985). Systematic study of memory functions in children following head injury is needed.

Too much stress may have been placed on intelligence as the measure of brain damage in children. When the effects of intelligence have been controlled in studies of head injured children, deficits on at least some of the other measures have remained (Chadwick, Rutter, Shaffer & Shrout, 1981; Klonoff & Low, 1974). Furthermore, studies of children with brain damage not due to head injury, have reported an excess of scholastic difficulties in those with intelligence in the average range, suggesting the presence of cognitive deficits not detected on IQ tests (Addy, 1987; Duffner, Cohen & Parker, 1988; Ellenberg, McComb, Siegel & Stowe, 1987; Prigatano, Zeiner, Pollay & Kaplan, 1983; Rutter, Graham & Yule, 1970).

Slowed visuomotor and visuospatial skills have been particularly noted after head injury, both in the early phases and as persisting deficits (Bawden, Knights & Winogron, 1985; Chadwick, Rutter, Shaffer & Shrout, 1981; Colam, 1984; Klonoff et al, 1977; Levin & Eisenberg, 1979a,b). Language functions have been less sensitive to head injury, but deficits, particularly in language production (eg naming and word fluency) and also in comprehension, but usually not in repetition, have been reported (Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Ewing-Cobbs et al, 1987; Levin & Eisenberg, 1979a,b).

1.6 Recovery Issues

Rutter and co-workers (1984) have stipulated that deficits detected after head injury may be attributed to brain trauma only if there is a pattern of recovery in which the deficit is greatest immediately after damage, and progressive improvement occurs during the following months. Studies of children and adults after head injury have shown this to be the general trend (eg Bond, 1975; Brooks & Aughton, 1979b; Chadwick-Rutter study, 1981; Groher, 1977; Klonoff et al, 1977; Levin, Benton & Grossman, 1982; Mandleberg & Brooks, 1975).

Although Rutter et al (1984) suggest that "conversely, the absence of any recovery phase would provide strong circumstantial evidence that the initial deficit was not due to brain injury" (p.88), several cautions should be held in mind:

1. There may be considerable variation in the amount of recovery shown by individuals with severe head injury, some

showing steep recovery over time, some showing none and others even showing a decline (Brooks & Aughton, 1979b; Brooks et al, 1984; Lezak, 1979).

2. Functions may improve at different rates, with the onset of improvement, particularly for more complex measures, sometimes occurring relatively late in the recovery process (at least in adults: eg Brooks et al, 1984; Conkey, 1938; Lezak, 1979; Najenson, Sazbon, Fiselzon, Becker & Schechter, 1978).

3. In children, cognitive deficits which are unexpected in terms of the degree of intellectual recovery attained, may emerge long after brain insult and hinder academic progress (Alajouanine & Lhermitte, 1965; Costeff, Groswasser, Landman & Brenner, 1985; Gronwall, unpublished paper; Levin, Benton & Grossman, 1982; Shaffer, Bijur, Chadwick & Rutter, 1980).

4. Learning and the development of new skills are also influenced by emotional and social variables (Ewing-Cobbs and Fletcher, 1987; St James-Roberts, 1979; Trevarthen, in press). These variables may limit recovery of old skills as well as the development of new age appropriate skills, independent of the severity of brain injury.

1.6.1 Practice Effects

Gains on tests do not necessarily indicate recovery from head injury, but may be attributable to factors such as practice on the tests or familiarity with the situation and what is required (Brooks et al, 1984; Dikmen & Temkin, 1987). A control group is therefore needed to determine how much improvement is likely on the grounds of repetition alone. However, there is a problem in assuming that "true recovery" in the head injured group is whatever gain is left over once control group gains have been subtracted. The severely head-injured may be too impaired at the initial

assessment to gain the same amount of information about the tests and strategies for doing them as the non-head-injured, and gains may therefore be attributable to genuine recovery rather than practice (Brooks, 1987).

Mandleberg and Brooks (1975) did not find a significant practice effect on Wechsler IQs when they grouped head-injured adults according to the number of times they had done the test.

Shatz (1981) and Seidenberg, O'Leary, Giordani, Berent and Boll (1981) have reviewed studies of adults with brain disorders and have stressed that there may be no gains on IQ or neuropsychological test scores unless there is an improvement in the underlying condition. Serial testing of children with brain disorders (eg chronic epilepsy, tumours) has also shown that, far from being a practice effect, there may be a decline at the first retesting following treatment (surgery, radiation, and/or medication) and that there may later be improvement, deterioration or no change, depending on the response of the underlying condition (Addy, 1987; Duffner et al, 1988; Ellenberg et al, 1987; Powell, Polkey & McMillan, 1985). Children with chronic, stable brain damage may have no significant IQ gains on retesting, while their controls show a significant practice effect (Klonoff & Low, 1974).

Patients with mild and moderate head injury are sufficiently intact to learn from experience (Dikmen & Temkin, 1987). However, it is these patients who have a high incidence of premorbid school backwardness and behaviour problems (Rutter et al, 1980) and, as has been suggested for adults, those with minimal resources may be least able to cope with the cognitive inefficiency associated with mild head injury, and may be critically

limited by such an injury (Barth et al, 1983). Expecting such patients to show improvement of a magnitude greater than that found in the controls before any gains are attributed to recovery from head injury (Chadwick-Rutter, 1981), seems a very stringent requirement (cf Brooks et al, 1984).

In serial testing of children, results must be adjusted for age as performance expectations differ according to age, and maturation and new learning may otherwise obscure deficits (Kinsbourne & Hiscock, 1983). The problems of using raw scores in groups matched on mean age have been demonstrated recently in the equipotentiality debate, and a plea has been made for the use of tests with normative data (Bishop, 1988).

1.6.2 Severity Effects

It has been suggested that recovery from head injury is proportional to the initial deficit, ie those with the greatest initial deficit show the most improvement, but also have greater residual deficits (Dikmen et al, 1983; Dikmen & Temkin, 1987; Uzzell, Obrist, Dolinkskas, Wiser & Langfitt, 1987). This position receives support from the Chadwick-Rutter study in which the severely injured had steeper recovery curves on performance IQ than the controls and the mildly injured, and showed persistent deficits at 2.25 years (1981). Other childhood studies with information on both severity of injury and the time course of recovery are lacking.

1.6.3 Time Course

It appears that recovery occurs within the first 6 weeks to 3 months in adults with minor head injury (see Binder, 1986 for a review; Levin, Mattis et al, 1987), but continues over a longer period where there is a severe head injury (Brooks et al, 1984). Substantial cognitive recovery

occurs in the first year after head injury in children (Black et al, 1971; Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Klonoff et al, 1974, 1977). Systematic study of recovery within the first year in children is lacking. On the basis of the few tests they applied at 4 months, Chadwick and co-workers suggest that much of the recovery has occurred by then, whereas inspection of the data of Black and colleagues suggests a drop from the initial to the 3 month assessment.

In adults with head injury, recovery curves appear to be steepest in the first six months and then to level out (Bond, 1975; Brooks, Aughton, Bond, Jones & Rizvi, 1980; Mandleberg & Brooks, 1975; Mandleberg, 1976; Parker & Serrats, 1976), although this may depend upon the form of statistical analysis used (Dikmen et al, 1983). Changes may, however, continue over years (Klonoff et al, 1977; Thomsen, 1984; van Zomeren & Deelman, 1978).

Different functions recover at different rates. In terms of the overall level of functional recovery, few patients change category on the Glasgow Outcome Scale (GOS) after 6 months (Jennett & Teasdale, 1981). Performance IQ tends to be more affected and to continue to improve for longer than Verbal IQ (Chadwick-Rutter study, 1981; Filley, Cranberg, Alexander & Hart, 1987; Mandleberg & Brooks, 1975). In adults, performance on simple measures (eg simple reaction time, RT; motor speed and strength) has been regarded as recovering fairly rapidly, while performance on more complex measures (eg choice RT, higher level cognitive functions and new learning) has been reported to continue to improve over a longer period (Ben-Yishay & Diller, 1983a; Conkey, 1938; Dikmen et al, 1983; Levin, Benton & Grossman, 1982; Lezak, 1979; Rimel, Giordani, Barth, Boll & Jane, 1981; van Zomeren & Deelman, 1978). Such a distinction may

not apply to children with various abilities at different stages of development. Costeff and co-workers (1985) reported that recovery of speech and motor abilities in head-injured children continued for longer and appeared more complete than recovery of learning and social abilities: no improvement of cognitive abilities was observed after the first 6 to 12 months. Pelco (1985) found IQ gains to have reached a plateau by 12 months, even in very severely injured children and adolescents who had been in prolonged coma.

Different rates of recovery have been postulated according to whether deficits are due to focal or diffuse lesions. Auerbach (1983) has claimed that specific disorders (eg of language, spatial organization, attention and memory) related to focal lesions may recover more rapidly (within a few months) than disorders of mental control related to the "core syndrome" associated with diffuse injury. Difficulties with mental control (ie initiating, sustaining and shifting attention appropriately) are likely to be shown up on tasks that are complex, have competing stimuli, or require speeded performance.

Very recently, attempts have been made to determine whether the specific neuropathology of the injury sustained (focal, diffuse, contusional, hyperaemic, hypoxic, etc) does not produce particular clinical subsyndromes within the severe head injury category (Alexander, 1987; Filley et al, 1987; Uzzell et al, 1987). Such factors may account for variations in recovery after head injury.

1.7 Persisting Impairment

Persisting impairment is best defined in terms of a matched control group, rather than in terms of attainment of "normal" levels. As a recent study of minor head injury in

adults in three different centres showed, what is normal in one area is not normal in another area, and socio-cultural factors can produce apparently defective scores in controls (Levin, Mattis et al, 1987). Whereas achievement of a level below average may constitute recovery for some, for others attainment of an average level may not represent a return to premorbid functioning. The possibility that those who are above average premorbidly may recover better from mild cerebral insult than those with fewer resources, has also been raised (Barth et al, 1983).

Furthermore, both the Klonoff and Chadwick studies show that matched controls who start at an average level, may achieve above average levels when they are tested several times, thereby raising the level at which multitested head injured may be regarded as recovered (Klonoff et al, 1977; Chadwick-Rutter, 1981).

While it is of importance to establish which groups of head-injured children have significantly reduced scores for functions at a particular time interval, it is also of clinical interest to know the proportion of such groups with defective scores. Studies providing this information have used cut-points 1 or 2 standard deviations below control or norm group means; or the 5th percentile (Chadwick-Rutter, 1981; Ewing-Cobbs et al, 1987; Klonoff et al, 1977; Winogron et al, 1984).

Medical and social variables related to overall outcome or to the presence of persisting impairment on neuropsychological tests have tended to be analysed on a univariate basis, as if the predictors were independent of each other (Berger, Pitts, Lovely, Edwards & Bartkowski, 1985; Wagstyl, Sutcliffe & Alpar, 1987; Winogron et al, 1984; Zuccarello, Facco, Zampieri, Zanardi & Andrioli, 1985). Multiple regression techniques may better identify

the factors predicting persisting impairment and eliminate variables which are redundant (Chadwick, Rutter, Thompson & Shaffer, 1981; Klonoff et al, 1977; Williams et al, 1984).

Concepts of better recovery in children than adults following brain injury and of greater plasticity in the immature central nervous system (CNS) have recently come under attack (Bishop, 1981; Chelune & Edwards, 1981; Fletcher & Satz, 1983; Robinson, 1981; St. James-Roberts, 1979). Brain injury appears to produce both subtle deficits related to the site of the lesion and a general overall loss in cognitive ability. Persisting general and specific impairments following head injury are reviewed in chapters 5 to 8.

1.8 The University of Cape Town (UCT) Childhood Head Injury Survey

A multidisciplinary team formed by the Neurosurgery Department at the University of Cape Town (UCT), carried out a retrospective survey of 1820 children admitted between 1966 and 1981 with head injuries to neurosurgery wards at Red Cross and Groote Schuur Hospitals (de Villiers, Jacobs, Parry & Botha, 1984). Long-term follow-up of the 191 severely head injured children in this group was attempted, but only 80 had full addresses and only 27 of these were traced. They were tested on the neuropsychological battery designed for the present study (Colam, 1984). A prospective study was initiated in mid-June 1983 and all children admitted with suspected head injury were screened for inclusion until mid-June 1985. The aims were to determine:

1. the incidence of paediatric head injury in greater Cape Town,
2. its causes and associations,
3. the psychosocial background of the head-injured,
4. their clinical presentation and medical management,
5. their physical and psychological outcome, and
6. rehabilitation needs and possible prevention.

This thesis focuses on aim 5 and considers only the cognitive aspects of psychological outcome in children aged 6 to 14 years. The psychosocial background information on these children has been analysed (Theron, 1987) and can be used in describing the sample and in determining the predictors of neuropsychological outcome, but obviously was not available when the neuropsychological study was designed. The clinical details are still being analysed and will be published in due course by Fowler and de Villiers. The psychosocial follow-up contains details on physical complaints, behaviour changes, family, school and peer group relationships after head injury. When it comes to the overall functioning of the child, cognitive factors cannot be viewed in isolation from emotional, motivational and social factors, but this data has not yet been analysed and an integration of all the information collected is beyond the scope of the present study.

1.9 Hypotheses

Too little has been firmly established about the consequences of head injury in children to allow for theory building. The natural course of recovery needs to be known before the effects of any rehabilitation programme can be determined. This study was undertaken as a survey to determine the nature of neuropsychological impairment and factors influencing recovery. Hypotheses are therefore stated in very general terms:

A. Nature of Deficits

1. Both the mild and severely injured will show initial deficits.
2. The more severe the injury, the greater the deficit.
3. In the very severely head injured, all functions, intellectual, visuomotor, language and memory, will show deficits.
   (The nature of the deficits expected is summarized at the end of the review chapters on intelligence, language, visuomotor and memory functions).

B. Recovery

1. The degree of improvement will be related to severity of injury sustained, with the most severely injured showing the greatest amount of recovery.
2. The very severely injured will not have recovered to control group levels by 1 year.
3. Most of the recovery in the mildly injured will occur in the first 3 months, by which time most functions will have reached control group levels.

C. Persistent Deficits

1. Memory and problem-solving skills, particularly, are likely to remain impaired in the more severely injured, but it is expected that deficits will be widespread.
2. The severity of diffuse injury will be of major importance in determing outcome.
3. Premorbid psychosocial factors are likely to influence outcome.

2. PRIMARY AND SECONDARY INJURY AND OUTCOME

2. PRIMARY AND SECONDARY INJURY AND OUTCOME

Primary brain damage occurs at the time of head injury and may be focal, (contusions plus lacerations) and/or diffuse, (widespread stretching and shearing of axons). Secondary brain damage may occur as a result either of the primary intracranial injury, (intracranial haematoma, brain swelling, oedema, infection and hydrocephalus), or as a result of various extracranial factors causing secondary cerebral insult (eg respiratory inadequacy, hypovolaemia, hypotension, anaemia or hyponatremia).

Primary and secondary events can interact to produce a variety of pathophysiological states with heterogeneous outcomes. In most studies, the effect of various events upon outcome has been determined in terms of the incidence of mortality or severe disability, rather than the incidence of neuropsychological deficits. Nonetheless, the assumption is that the factors producing severe disability after head injury are also those which are associated with the production of milder disabilities (Adams, Graham & Gennarelli, 1985).

2.1 Primary Injury

There is evidence that the severity of the diffuse injury occurring at the time of impact is more important than focal injuries in determining the overall quality of recovery, but focal lesions may be associated with specific neuropsychological deficits (Levin, Benton & Grossman, 1982). The flow diagram, Figure 1, from Ommaya (1982) illustrates the mechanical inputs by which focal and diffuse injuries are produced.

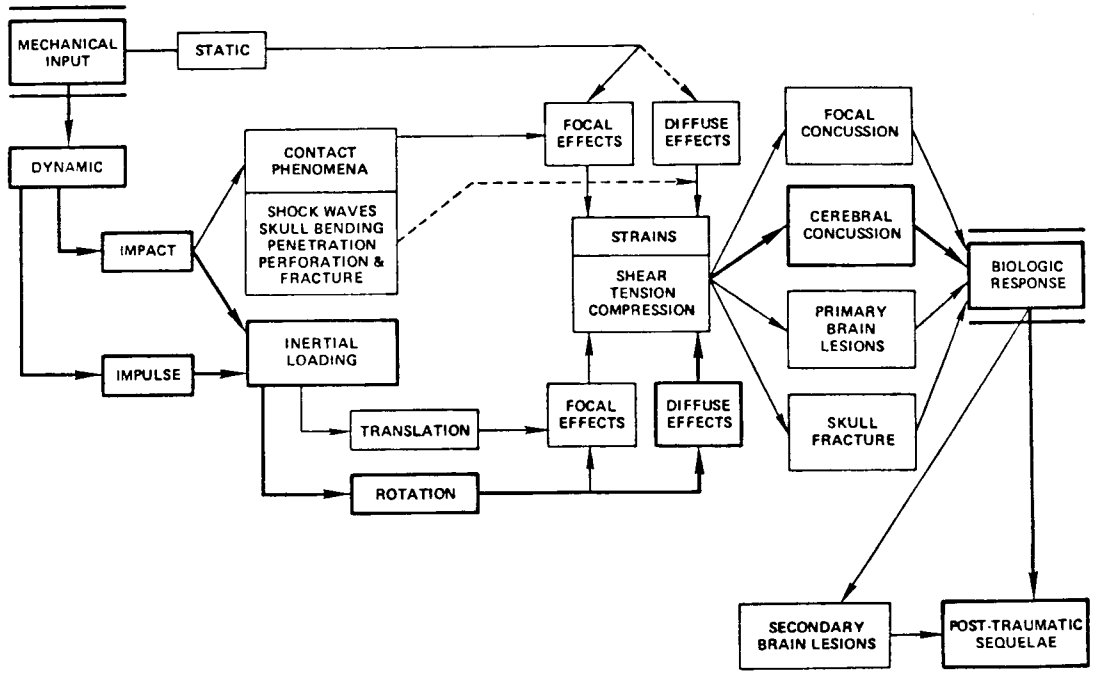

Figure 1. Critical Factors in the Mechanics of Head Injury (Ommaya, 1982, p.1880)

Contact phenomena produce primarily focal injuries when the skull is deformed or penetrated at the site of contact and injures the underlying brain (Figure 1).

2.1.1 Skull Fractures

The presence of a skull fracture is evidence that impact to the head has been of considerable force. Skull fractures occur in 4 to 8% of children visiting emergency rooms for minor head injury (Harwood-Hash, Hendrick & Hudson, 1971; Leonidas, et al, 1982) and in about 25% of all those admitted to hospital for head injury in the U.S.A., Canada and Glasgow (Harwood-Hash et al, 1971; Ivan, Choo & Ventureya, 1983; Jamison & Kaye, 1974; Kraus et al, 1987). Although skull fractures may be more common in children who develop intracranial complications and may be statistically correlated with mortality, most children with skull fractures do not develop complications and there seems to be agreement that fractures are poor predictors of outcome

(Kraus et al, 1987; Leonidas et al, 1982; Wagstyl, et al, 1987).

Closed depressed fractures occur in 1.5 to 5% of children admitted for head injury and compound depressed fractures in about 1.5% (Ivan et al, 1983; Jamison & Kaye, 1974; Kraus et al, 1987). Amongst admissions to neurosurgery units, however, there may be a higher incidence of children with depressed fractures and dural tears (eg 9% in Chodkiewicz, Redondo, Merienne, Cioloka & Terrazas, 1975; and 16% in Berger et al, 1985; 8% in Black, Jeffries, Blumer, Wellner & Walker, 1969). Open depressed fractures carry an increased risk of secondary damage from intracranial infection and post-traumatic epilepsy.

Basal skull fractures are evidence of severe impact and may also be complicated by intracranial infections (Jellinger, 1983). In terms of neuropsychological functioning, the effects of a force sufficient to produce a basal fracture, has not been investigated.

2.1.2 Contusions and Lacerations

The main cause of damage in nonmissile head injury is the extent and direction of inertial loading (acceleration-deceleration), (Gennarelli & Thibault, 1985). As is shown in Figure 1, translational or linear acceleration produces only focal injuries, the result of contact between the viscoelastic brain and the rigid, bony compartments of the skull, but loss of consciousness does not result unless there is rotational acceleration as well. Contusions characteristically occur at the anterior poles and the undersurfaces of the frontal and temporal lobes and at the edges of the tentorium and the falx where the skull is irregular. They are most severe at the crests of the gyri, but may extend through the cortex into the white matter.

Computerized tomography (CT) studies underestimate the incidence of haemorrhagic contusions and magnetic resonance imaging (MRI) has detected more lesions and shown them to be larger in volume and to have more extensive surrounding oedema than is shown on CT scan (Levin, Amparo et al, 1987; Snow, Zimmerman, Gandy & Deck, 1986). Such studies have confirmed the predominance of frontal and temporal lobe sites and have shown that, with minor and moderate head injury in adults, contusional lesions may resolve over 1 to 3 months with a parallel improvement on neuropsychological tests of executive and memory functions.

Incidence rates of contusions in children vary from 3 to 16%, as opposed to about 30% in adults (eg Bruce, 1983; Kraus et al, 1987; Zimmerman & Bilaniuk, 1983). Incidence has been reported as least in the infant and young child and as increasing with approach to adolescence and in association with a depressed skull fracture. Based on radiological and clinical findings, Shapiro and Marmarou (1982) found that 41% of 22 severely head-injured children, excluding those with surgical mass lesions, had contusions. The significance of contusions lies in the secondary processes of haemorrhage, oedema and swelling that they may initiate, although Zimmerman and Bilaniuk (1983) claim that less mass effect is seen in children than in adults.

Shaffer, Chadwick and Rutter (1975) found focal residual neurologic abnormality such as hemiplegia or aphasia at follow-up at least 2 years later in only 13% of children with compound depressed skull fractures (with dural penetration or focal brain contusion) . The laterality of the hemisphere damaged had little effect on the pattern of cognitive deficits, but the extent of cognitive impairment was associated with the severity of diffuse damage as reflected in duration of unconsciousness.

2.1.3 Diffuse Axonal Injury (DAI)

Prolonged coma from the time of injury is associated with diffuse axonal injury. Depending on the magnitude, duration, and direction of rotational acceleration, there may be widespread shearing of axons throughout the cerebral hemispheres and upper brain stem, and tissue tears may be present in the corpus callosum and dorsolateral quadrant of the rostral brainstem (Adams et al, 1985; Gennarelli & Thibault, 1985). In children, as in adults with diffuse shearing injuries, mortality is high and those surviving may be in a persistent vegetative state or have severe disability (Bruce et al, 1981; Shapiro, 1985; Zimmerman & Bilaniuk, 1983).

Diffuse axonal injury can only be seen microscopically and even significant axonal changes may escape the resolution powers of CT scanners. Normal initial CT has been reported in association with diffuse atrophy on later scan (Lobato et al, 1986; van Dongen & Braakman, 1980) and with DAI findings at autopsy (Snoek, Jennett, Adams, Graham & Doyle, 1979). Magnetic resonance imaging, however, can detect changes in the deep white matter not demonstrated by CT scan (Snow et al, 1986).

Axonal damage is a progressive event leading to atrophic enlargement of the ventricles and deepening of sulci on CT from 2 to 3 weeks after injury, indicating long tract degeneration. Late ventricular enlargement has been associated with longer coma or PTA and with poorer overall outcome and defective neuropsychological test performance (Cullum & Bigler, 1986; Lobato et al, 1986; Meyers, Levin, Eisenberg & Guinto, 1983; van Dongen & Braakman, 1980; Wilson et al, 1988).

There is thought to be a continuum of DAI, with the lesser forms of white matter damage corresponding to the entity of mild concussion (Adams et al, 1985; Langfitt & Gennarelli, 1982; Ommaya, 1982; Pilz, 1983). In experiments with subhuman primates, the extent of coma produced by angular acceleration has been correlated with the extent of DAI found (Gennarelli, Thibault et al, 1982).

Microscopic evidence of DAI has been found in humans sustaining minor head injuries and dying of other causes (Oppenheimer, 1968; Pilz, 1983). It is likely that, in any given injury, many more axons are damaged and rendered non-functional than are actually severed (Langfitt & Gennarelli, 1982). The work of Povlishock (1985) with cats has shown that even the most minor form of head injury with transient concussion leads to abnormalities of axonal functioning, but that stretching of nerve fibres does not always lead to irreversible structural damage. The Pennsylvania group has suggested that the improvement in the memory, learning and visual-motor scores of DAI survivors in the first post-traumatic year may be because many axons are stretched, rather than destroyed, increasing their potential for later recovery (Uzzell, et al, 1987).

2.1.4. The Spectrum of Cerebral Concussion

The awake state requires that the neural activity of the ascending reticular activating system of the brain stem should be projected to the cerebral cortex of both hemispheres, either directly or via hypothalamic-diencephalic centres, and that there should be feedback from the cortex to the brain stem and diencephalic centres (Gennarelli, 1982). Diffuse brain injury disrupts the functioning of the cerebral hemispheres and alters consciousness by disconnecting them from brain stem centres. Consciousness may also be lost by compression of, or

haemorrhaging into, the brain stem as a result of a supra-tentorial mass. Isolated primary brain stem damage as a mechanism of unconsciousness in head injury is disputed (Adams et al,1985; Jane, Steward & Gennarelli, 1985).

Ommaya (1982) and Gennarelli (1982) have proposed a graded spectrum of concussive injuries ranging from mild confusion to coma and death. In the diagram from Ommaya (1982) below, the first three grades of injury represent the mild concussive syndromes in which consciousness is preserved, but there are varying degrees of confusion, disorientation and amnesia. Ommaya has postulated that there is cortical-subcortical disconnection in these mild injuries, but that coma results only when the diencephalic-mesencephalic core is affected.

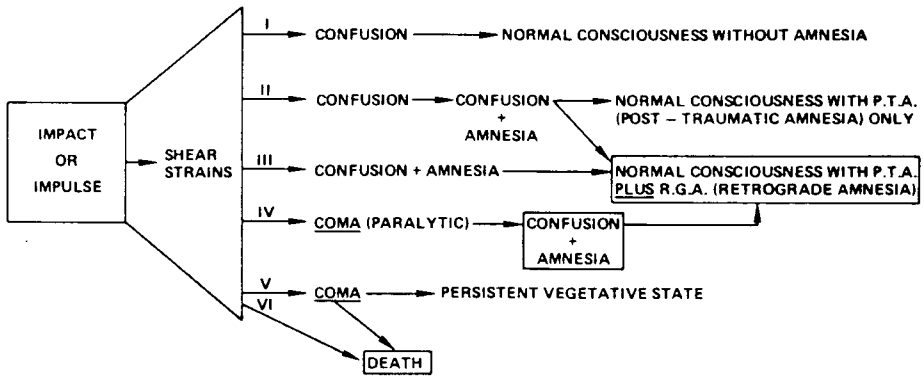

Figure 2. Syndromes of Cerebral Concussion

Grades I and II represent cortical-subcortical disconnection; II and III represent cortical-subcortical-diencephalic disconnection; IV and VI represent cortical-subcortical-diencephalic-mesencephalic disconnection (Ommaya, 1982, p.1882).

In the mildest grade of concussion, there is momentary confusion. With more severe injury, there is confusion and amnesia, but coordinated sensorimotor activities still continue as, for example, in some sporting injuries.

Amnesia for the impact and the events immediately preceding it may also develop after the event (Russell, 1971) although such retrograde amnesia may later shrink again. With classical cerebral concussion (Grade IV onwards), there is loss of consciousness and sensorimotor paralysis. The sequence of recovery is consistent and the patient awakes confused before regaining alertness and orientation. There is always retrograde and post-traumatic amnesia (Ommaya, 1982).

The pattern of recovery in head injury, with memory functions being the first to be affected and the last to recover, suggests that the cerebral hemispheres rather than the brain stem are the major recipients of trauma in acceleration-deceleration injuries (Gennarelli, 1982; Symonds, 1962). When axonal disruption extends to the diencephalon and brain stem, there is deep unconsciousness. Abnormal brain stem signs such as decorticate or decerebrate posturing may occur (Gennarelli, 1982). Ommaya (1982) has suggested that in a persistent vegetative state in which brain stem function is all that survives, the outer brain layers are disconnected from the recovering brain stem.

2.2 Secondary Injury

As in primary injury, the damage associated with secondary brain injury may be focal and/or diffuse. The principal types of focal damage include the intracranial haematomas and the damage secondary to raised intracranial pressure (shift, herniation of the brain and infarction), whereas diffuse secondary damage takes the form of hypoxia, ischaemia and diffuse brain swelling (Adams et al, 1986). Infections may be diffuse (meningitis) or localized (abscess), while epileptic seizures may be either focal or generalized. Intracranial and extracranial factors can act together to aggravate the primary insult.

2.2.1 Intracranial Haematomas

It has been stated that children tend to have small haematomas in association with generalized brain swelling, rather than large clots in association with marked midline shift as seen in adults (Lobato et al, 1983). A proportion of patients with intracranial haematomas may have a lucid interval before losing consciousness, indicating a less severe degree of primary injury. In the Children's Hospital of Philadelphia series, a mass lesion was not found to affect the outcome (Bruce et al, 1978), but Berger and co-workers (1985) found that nearly 50% of children with intracranial mass lesions, regardless of type, died or were severely disabled. Filley et al (1987) found that severely injured children with focal intracerebral lesions superimposed on diffuse brain injury had a worse outcome than those with diffuse injury alone. A multicentre study has shown that, given the same level of coma, different lesions may have vastly different outcomes, with acute subdural haematomas having the least favourable prognosis (Gennarelli, Spielman et al, 1982). Intracranial haematomas are classified according to the level of their location eg extradural, subdural, intracerebral, etc.

2.2.1.1 Extradural Haematomas

Classically, extradural haemorrhages are due to tearing of the middle meningeal vessels in the temporal regions, but in 20 to 30% of cases the haemorrhage is in the frontal or parietal region, or in the posterior fossa where the origin may be venous (Jellinger, 1983). CT studies give an incidence of 6 to 8% in children with severe head injury (Berger et al, 1985; Bruce et al, 1979; Zimmerman & Bilaniuk, 1983).

Mortality and disability rates for epidural haematomas have dropped dramatically in centres with CT scanners (Bruce, 1983). There should be no neuropsychological effects unless prolonged cerebral compression secondary to such a haematoma occurs. Dhellemmes, Lejeune, Christiaens & Combelles (1985) reported that 33% of 144 children with extradurals had residual disabilities including hemiparesis and seizures. Behaviour change or scholastic backwardness occurred in 19%, and mental retardation in an additional 5%. Outcome, however, was related to the post-operative length of coma.

2.2.1.2 Subdural Haematomas

In contrast to extradurals, subdural haematomas are associated with primary damage to the underlying brain and carry a significant morbidity and mortality. The most common cause of bleeding is the stretching and tearing of the bridging veins between the cortex and the venous sinuses. Other causes are the tearing of dural sinuses and vessels in contusion and lacerations (Jellinger, 1983). Haemorrhage into the subdural space tends to be extensive and may spread diffusely over the hemispheres. Acute subdurals occur in only about 5 to 8% of head-injured children (Bruce, 1983), but reported mortality rates range from 20 to 85% (Jellinger, 1983; Pagni et al, cited Shapiro, 1985).

Follow-up CT scans show reabsorbtion of the haematoma with time, whether treated surgically or not. If there is accompanying parenchymal damage, it is common for the ventricles to enlarge and for cortical sulci to expand as well as areas of lucency to occur at the grey/white matter junction secondary to encephalomalacia (Zimmerman & Bilaniuk, 1983).

2.2.1.3 Intracerebral Haematomas

Direct injury to a penetrating blood vessel within the brain parenchyma or extensive bleeding at the site of a contusion leads to the formation of an intracerebral haematoma. Most occur in the frontal and temporal lobes, often deep within the white matter. Intraventricular rupture may occur as the haematoma bursts through the white matter. Intracerebral haematomas are far less common in children than in adults, and the incidence is usually about 5% in children with severe head injuries (Berger et al, 1985; Bruce et al, 1979). Small superficial intracerebral haematomas may result from depressed skull fractures where bone fragments have been driven into the brain (Pang, 1985).

2.2.2 Subarachnoid Haemorrhage (SAH)

SAH is produced by injury to the leptomeningeal blood vessels and is usually associated with contusional injury. It is most marked over the parasagittal regions but may also occur in the basal cisterns. Bruce et al (1979) reported SAH as the most frequent CT finding, occurring in 79% of their severely head-injured children.

2.2.3 Brain Swelling

The most common response to head trauma in children is diffuse brain swelling which occurs in about one third (Bruce et al, 1981; Esparza et al, 1985; Snoek et al, 1979; Zimmerman & Bilaniuk, 1983). It can occur after relatively mild head injury with no loss of consciousness as well as after more severe injury, and Gennarelli (1982) has suggested that it may account for the lethargy seen following classical concussion.

Diffuse cerebral swelling is thought to be attributable to hyperaemia. It can be diagnosed on CT scan from absence or compression of the lateral and third ventricles and

perimesencephalic cisterns, but may be missed unless there is serial scanning as children normally have small ventricular systems (Bruce et al, 1981; Zimmerman & Bilaniuk, 1983).

In the study by Bruce and co-workers, the CT pattern of diffuse swelling was seen in only 15% of the children with a Glasgow Coma Scale score of greater than 8, whereas it occurred in 50% of those with a score of 8 or less. Diffuse brain swelling is usually associated with a satisfactory outcome, except where there are other lesions and oedema developing 1 to 5 days after injury further increases ICP (Berger et al, 1985; Cordobes et al, 1987; Esparza et al, 1985; Lobato et al, 1983).

In a study by the University of Pennsylvania School of Medicine, 62% of the patients with severe head injury whose CT scan only showed diffuse swelling had good recovery on the GOS in contrast to only 18% of those patients whose CT scans showed the haemorrhages of DAI (Uzzell et al, 1987). In the small subgroups that could be neuropsychologically tested, those with diffuse swelling performed at substandard levels on tests of learning and visuomotor speed, although their performance was superior to that of patients with DAI. They also showed little improvement over the next few months, suggesting that recovery had taken place in the 3 months prior to testing.

2.2.4 Oedema

Oedema is an increase in brain volume that results from an accumulation of water in the interstitial and/or intracellular spaces. Vasogenic oedema is associated with damage to the walls of the cerebral capillaries as a result of which fluid escapes into the extracellular space of the white matter. It occurs commonly in association with

contusions and haematomas and under the site of skull fractures. Resolution of oedema related to contusions may occur within 10 days in mild cases and within 4 weeks in severe cases (Snow et al, 1986). Oedema may compromise cerebrovascular perfusion, alter ICP and cause brain shift and herniation (Tornheim, 1985).

2.2.5 Intracranial Hypertension

An increase in the volume of any of the three components of the intracranial compartment, blood, brain or cerebrospinal fluid, will tend to increase intracranial pressure (ICP). Thus cerebrovascular engorgement, an intracranial haematoma, oedema or defective cerebrospinal fluid absorbtion or circulation may all contribute to intracranial hypertension. In children with severe head injury, about half have an initially raised ICP (Bruce et al, 1981; Esparza et al, 1985; Zimmerman & Bilaniuk, 1983), and others may have a delayed increase in ICP such that, overall, an elevation of ICP occurs in about 70 to 86% (Alberico, Ward, Choi, Marmarou & Young, 1987; Bruce, 1983; Shapiro & Marmarou,1982). Elevation of ICP in isolation from other clinical data is a poor predictor of outcome (Humphreys, 1983; Narayan et al, 1981).

Few studies have considered intracranial hypertension in relation to neuropsychological outcome. Winogron et al (1984) found no correlation between test scores and ICP values. At the hospitals involved in the present study, patient numbers, staff shortages and restrictions on intensive care facilities prevent the routine measurement of ICP in severely head-injured children, so no attempt has been made to verify these findings.

2.2.6. Brain Shift and Herniation

With a supratentorial mass lesion or diffuse swelling, the cerebral hemispheres and brain stem are displaced downwards through the tentorial hiatus into the posterior fossa. If the lesion is unilateral, the brain may be displaced across the midline with distortion of the cingulate gyrus under the falx, and lateral as well as downward displacement of the medial temporal lobe. Unchecked herniation affects the diencephalon and upper brain stem and consciousness is progressively impaired. Herniation of the cerebellum through the foramen magnum compresses the medulla and leads to vasomotor and respiratory disturbances which eventually cause respiratory arrest and death (Jennett & Teasdale, 1981; Teasdale & Mendelow, 1984).

Raised pressure and brain herniation cause necrotic lesions in the parahippocampal gyri and the medial occipital cortex and may be responsible for a considerable proportion of the ischaemic damage found at autopsy in the basal ganglia and cerebellum (Graham, Adams & Doyle, 1978). Memory disabilities in survivors of severe head injury may be related to the hippocampal necrosis (Teasdale & Mendelow, 1984).

2.2.7 Hypoxic Ischaemia

There is a very high incidence of hypoxic brain damage in patients dying of nonmissile head injuries (Graham et al, 1989). In the neuropathological study of 151 cases by Graham et al (1978), the areas most commonly affected included the hippocampus (81%) and the basal ganglia (79%). Almost half the cases had ischaemic damage in the cerebral cortex, most frequently in the arterial boundary zones, but also in the territories supplied by particular arteries. There were significant correlations between ischaemic brain damage and either raised ICP or an episode of hypoxia.

Severe and diffuse cortical necrosis occurred in a few patients who had experienced cardiac arrest or status epilepticus.

The causes of systemic hypoxia may be multiple and include airway obstruction, chest injuries, aspiration pneumonia, cardio-respiratory arrest, systemic hypotension and post-traumatic epilepsy. Arterial hypoxemia has been found in one third of patients admitted with severe head injuries (Miller, 1983). Head-injured children are more prone to vomit than adults and this may increase the risk of a blocked airway or aspiration pneumonia (Jennett, 1972). The contributions of additional injuries and of post-traumatic epilepsy to secondary brain damage in children are dealt with briefly below.

2.2.8 Multiple Injury

Multiple injury may be associated with hypoxaemia, hypovolaemia, hypotension, anaemia and sepsis, all of which may increase the risk of secondary brain injury. In the study of Mayer, Walker, Shasha, Matlak & Johnson (1981), severe multiple trauma more than doubled the chances of a poor outcome. Fewer children with multiple trauma than with head injury alone made a Good Recovery (58% versus 70%). In a later, larger, study these findings were confirmed, and mortality and morbidity were found to increase linearly with increases in scores on the Modified Injury Severity Scale (MISS) (Walker, Storrs & Mayer, 1984). Children with pulmonary injury were particularly at risk of a poor outcome. Similarly, Luerssen, Klauber & Marshall (1988) found the effect on mortality to be greatest for chest and abdominal injuries and least for injuries to the pelvis and extremities in children under 15.

The management of additional injuries may involve the use of drugs, particularly anaesthetic agents, which can affect cerebral blood flow either directly (by vasoconstriction or dilatation) or indirectly by influencing blood pressure or respiration, thereby increasing the risk of iatrogenic brain injury (Jennett & Teasdale, 1981).

Most studies of multiple injury have considered the associated increase in mortality rather than any more subtle rating of outcome. The study of Zuccarello et al (1985) found that, whereas the rates for Moderate and Severe Disability were the same in the multi-injured as in those with head injury alone, fewer had a Good Recovery (16% as opposed to 37%). Some studies have found little or no effect for additional injury on outcome rating (eg Berger et al, 1985; Wagstyl et al, 1987), but none appear to have looked at the effect on neuropsychological status.

2.2.9 Post-traumatic Seizures

Epileptic seizures following head injury pose a threat to the brain in that there may be associated respiratory and cardiovascular insufficiency which compromise brain oxygenation. The brain's need for oxygen increases dramatically during a seizure and the cerebral metabolic rate may be increased by up to three times the normal rate (Jennett & Teasdale, 1981). The risk of an early seizure (within 1 week of injury) is higher in children than in adults. Seizures have been reported in from 1.5 to 10% of all children admitted for head injury; 13 to 15% where the occurrence of a seizure has been amongst the admission criteria; and 20 to 35% of children with severe head injuries (Annegers et al, 1980; Black, Shepard & Walker, 1975; Chodkiewicz et al, 1975; Desai et al, 1983; de Villiers et al, 1984; Eiben et al, 1984; Hahn, Fuchs, Flannery, Barthel & McLone, 1988; Hendrick & Harris, 1968; Ivan et al, 1983; Jamison & Kaye, 1974; Jennett, 1973;

Snoek, Minderhoud & Wilmink, 1984). The incidence of early seizures increases with severity of injury in the studies cited, and Black et al (1975) reported that the incidence of seizures appeared to increase progressively with the duration of coma.

Brain contusions and haematomas also increase the risk of early epilepsy and seizures have been reported in 19 to 30% of children with such injuries (Annegers et al, 1980; Black et al, 1975; Desai et al, 1983; Hendrick & Harris, 1968). Hahn et al (1988) found that 43% of children with diffuse brain swelling had seizures, but that children with open depressed skull fractures had only a slightly higher incidence of seizures (14%) than children without skull fractures (10%).

Desai et al (1983) have suggested that low socio-economic status may be associated with additional insults that increase vulnerability to seizures. In their paediatric group, which consisted almost entirely of poor patients from an inner city community, 11% of those with mild head injury had seizures in contrast to 1% of patients in the series of Annegers et al (1980) which was drawn from a middle class community.

Seizures have tended to occur within the first 24 hours after injury (Desai et al, 1983; de Villiers et al, 1984; Jennett, 1973), often within the first 2 hours (Black et al, 1975; Hahn et al, 1988). When clinical seizure type has been reported, early seizures have been partial in well over half the children (Black et al, 1975; Jennett, 1973; Kollevold cited Hauser, 1983). Status epilepticus is more common in children, particularly those under 5 years, than in adults, and may produce severe hypoxic brain damage (Jennett & Teasdale, (1981).

2.3 Conclusions

It has been suggested that focal and diffuse brain damage resulting from head injury may produce different neuropsychological syndromes and deficits which recover at different rates (Alexander, 1982, 1987; Auerbach, 1983; Stern & Stern, 1985). It may not be possible, however, to know which deficits are attributable to which lesions, particularly when focal damage is superimposed upon diffuse damage which is not readily detectable. Diffuse axonal injury is only evident microscopically. Even in the more severe cases, the typical lesions in the corpus callosum and rostral brain stem may be missed if the CT cuts are at the wrong level (Snoek et al, 1979). Diffuse brain swelling may be missed if CT scanning occurs too early after injury, or if small ventricles are interpreted as normal in children. Diffuse hypoxic ischaemia is not demonstrable in life and the possibility of such damage can only be inferred from the presence of multiple injuries or complications, particularly those which involve the respiratory system or blood loss and hypotension. Hypoxic episodes may not be documented if resuscitation occurred prior to hospital admission. Surgery and the associated drugs and anaesthesia may also contribute to interference with the blood and oxygen supply to the brain, as may early seizures.

Focal brain lesions such as intracranial haematomas are well visualized on CT scan, but some contusions may be missed and their size and the extent of the surrounding oedema underestimated. Raised intracranial pressure and brain shift may be suspected clinically and confirmed by CT scan. Unless there is infarction, the associated focal hypoxic damage may not be radiologically visible, and even then the extent of the underlying diffuse damage is a matter of speculation. The severity of the diffuse injury is correlated with the length of coma and of PTA. The measurement of such severity indices is the subject of the next chapter.

3. SEVERITY INDICES AND THE PREDICTION OF OUTCOME

3. SEVERITY INDICES AND THE PREDICTION OF OUTCOME

Outcome from head injury is dependent primarily upon the severity of the diffuse injury sustained. Measures of severity dealt with in this chapter include the depth of coma, the duration of coma and the length of PTA. These measures, which recur regularly in the literature as powerful predictors of outcome, allow for a grading of severity and so are useful in rating consecutive admissions where the majority of patients will have only mild head injury. Signs of brain stem dysfunction such as unreactive pupils, disturbed eye movements, abnormal oculocephalic or oculovestibular responses, are associated with poor outcome, particularly death (Jennett & Teasdale, 1981), but are not likely to be present in many children in a sample of consecutive admissions. Further factors possibly contributing to outcome and not already dealt with in the previous chapter, include the mechanism of injury, age at injury, and the presence of an abnormal EEG.

3.1 The Depth of Coma

The Glasgow Coma Scale (GCS) was developed so that relatively inexperienced medical personnel could reliably describe degrees of coma without reference to supposed underlying anatomical sites of dysfunction (Jennett & Teasdale, 1981). It allocates points for the best level of responsiveness in each of three areas: eye opening, motor response and verbal performance, as shown in Table 1.

Table 1

Glasgow Coma Scale

Eye Opening		Best Motor Response		Best Verbal Response	
Spontaneous	4	Obeys	6	Oriented	5
To speech	3	Localizes	5	Confused conversation	4
To pain	2	Withdraws	4	Inappropriate words	3
No response	1	Abnormal flexion	3	Incomprehensible sounds	2
		Extensor response	2	No response	1
		No response	1		
Range of Scores 3-15					

Coma has been defined as "not obeying commands, not uttering words, and not opening the eyes" (Jennett & Teasdale, 1981, p.80). Summed scores on the GCS of 7 and under, all correspond to this definition of coma, as do 53% of scores totalling 8. Patients are usually regarded as having sustained a severe head injury if in coma (GCS$\leq$8) for at least 6 hours, either immediately after trauma or following a lucid interval or operation for haematoma. Comparable mortality and morbidity rates for patients with similar GCS scores have been reported at the different centres contributing to the International Data Bank (Braakman, Gelpke, Habbema, Maas & Minderhoud, 1980; Jennett et al, 1979). The 6 hour cut-off was chosen to exclude those in coma temporarily for reasons such as convulsions or extracranial factors such as hypotension or hypoxia. Early treatment, particularly operation for acute subdural haematomas, has been realized to affect outcome, and GCS is assessed immediately after noncranial resuscitation in the National Coma Data Bank series (Eisenberg, 1985).

The deeper the coma, the greater the likelihood of a poor outcome. Earlier studies were likely to have almost 100% death rate for those admitted flaccid (GCS=3) and rates between 50 and 70% for those admitted showing decerebrate posturing (GCS=4), (eg Chodkiewicz et al, 1975). In some recent studies, these low coma scale scores still have a high mortality rate (eg 85% in Humphreys, 1983; 78% in Wagstyl et al, 1987), and those who survive are likely to have severe disabilities or continue in a persistent vegetative state (PVS), with few (eg 11% in Wagstyl et al, 1987) or none (Humphreys 1983; Kraus et al, 1987) making a satisfactory recovery. However, other studies have reported a mortality rate of only about 60% in those admitted in a flaccid coma (Berger et al, 1985; Bruce, 1983) and far lower in those admitted decerebrate (eg 13%, Bruce, 1982). The survivors are reported to have a satisfactory outcome, although there is a high percentage of neurological and cognitive defects (Bruce, 1983; Raphaely, Swedlow & Downes, 1980). The better outcome in these series is attributed to better management of hypoxia, hypercarbia and intracranial hypertension which cause secondary brain injury.

Bruce (1982) has claimed that children admitted with GCS scores of 6 and above should not die from primary injury and should make a good recovery. In studies from Canada, Britain and Italy, however, a poor outcome (death or severe disability) has been reported for between 20 and 30% of children admitted with GCS scores between 5 and 8 (Humphreys 1983; Wagstyl et al, 1987; Zuccarello et al, 1985). Wagstyl and co-workers confirmed the observation made in other studies that outcome is difficult to predict for those admitted with GCS scores between 5 and 8, and reported that outcome is better if there is no deterioration within the first 24 hours. The majority of children admitted with GCS scores above 8 make a satisfactory recovery. GCS is related

to the type of brain injury and Kraus et al (1987) reported that, whereas 98% of those admitted with concussion only had GCS scores of 13 and above, only 38% of those with haemorrhage, contusion or laceration had a GCS of 13 or higher. Hennes, Lee, Smith, Sty and Losek (1988) have similarly reported altered mental states to be more common in those with CT scan abnormalities than in those without abnormalities.

Few paediatric studies have considered the relationship of GCS scores to neuropsychological outcome. Winogron et al (1984) found significant correlations between several neuropsychological test scores and GCS. Almost 90% of children and adolescents with defective scores on 30% or more of the neuropsychological tests had GCS scores less than 8, whereas 77% of those with most tests within the normal range had GCS scores above 8. Levin, Eisenberg and co-workers (1982) found a nonsignificant trend for lower IQs in those with GCS at or below 8 versus those with GCS over 8. A disproportionate number of children (40%) in the more severe group had IQs below 80. Children in the lower GCS group also had significantly lower memory scores at initial assessment and a higher proportion had memory deficits at follow-up.

3.2 Duration of Coma

That length of coma is an important predictor of severe disability can be seen in the reports from rehabilitation centres. Although many of the patients in prolonged coma may achieve physical independence in terms of ambulation and self-care, it is evident that few achieve scholastic or vocational goals, and almost all will have cognitive, emotional or adaptive problems (Brink, Garrett, Hale, Woo-Sam & Nickel, 1970, 1978, 1980; Filley et al, 1987; Pelco, 1985; Stover & Zeiger, 1976). The majority of

patients with coma lasting less than 3 months are physically independent, but only about a third or less of those with longer coma achieve such independence (Brink et al, 1978, 1980). In the study by Stover and Zeiger (1976) a third of those with coma lasting between 2 and 12 weeks needed supervision of their daily activities.

Eiben et al (1984) found that with coma under 21 days, 73% were independent but with coma over 21 days, 78% were dependent. Filley et al (1987) found no statistically compelling relationship between duration of coma and outcome (GOS; school/vocational functioning; social behaviour). However, this was because patients with coma less than a week could have severe impairment of functioning. Patients with long coma had poor outcomes: 62% of those with coma between 1 week and 1 month had severe impairment of school/vocational functioning and none with coma over a month had a good outcome on any criterion. Several studies have stressed 2 weeks coma as the cut-off point after which poor outcome is more likely (Facco et al, 1986) or, only exceptionally, will a child make normal progress at school (Heiskanen & Kaste, 1974; Vapalahti, cited Leary, 1982).

Intellectual deficits have been more common in children with prolonged coma. Brink et al (1970) found that only one third of children in coma over 1 week had normal intelligence. There was an almost linear relationship between coma duration and IQ. Johnston and Mellits (1980) with a sample of 40 children in coma over 24 hours found correlates of the order of .5 between coma duration and outcome in terms of motor function, self care skills, intellectual and language abilities. Pelco (1985) assessing a group of very severely injured children and young adults (mean coma duration 60 days) found coma length to be negatively correlated with post-injury IQ and with the

percentage of premorbid IQ recovered. Multiple regression equations which included coma duration were better able to predict outcome for those remaining in PVS and for those recovering to premorbid IQ level, than for those with persisting impairment of intelligence.

In samples of head-injured patients in which fewer have prolonged coma, the relationship between coma duration and cognitive deficit is less clear cut: Brooks et al, (1980), found no significant relationship between duration of coma and performance on intelligence, language and memory tests at 1 year post-injury in adults, while Parker and Serrats (1976) found no relationship between coma duration and learning skills. In both these studies, the period of post-traumatic amnesia or disorientation, rather than the length of unconsciousness, was related to cognitive deficit. Comparing children who had not been in coma with those in coma for under and over 24 hours, Levin and Eisenberg (1979b) found that early impairment of language, visuospatial, memory, somatosensory and motor functions was more frequent in those in coma over 24 hours. Unequivocal intellectual impairment (IQ below 70) at 6 months or more after injury was confined to those in coma over 24 hours. In a study of focal injury, Chadwick, Rutter, Thompson and Shaffer (1981), found that differences on intelligence and other tests at 2 years were attributable to longer periods of unconsciousness, rather than to the laterality of lesion: those with unconsciousness lasting more than 72 hours had significantly lower scores than those with unconsciousness of less than 72 hours' duration.

3.3 Post-traumatic Amnesia (PTA)

Post-traumatic amnesia refers to the period immediately following head trauma in which consciousness is disturbed such that current events are not stored (Russell & Smith, 1961). Most of the variables important for the prediction of outcome from severe head injury (eg duration of coma, presence of brain stem signs, haematoma, etc) cannot be applied to cases of less severe injury, but the end of PTA (defined as the point at which continuous memory returns) can be determined whether there is prolonged coma or merely minutes of confusion. Russell (1971) considered that the return of full orientation signalled the end of PTA, and Jennett and Teasdale (1981) made a similar observation in the International Data Bank series. In this series, 94% of the survivors of severe head injury (coma equal to or longer than 6 hours) had a PTA of more than 1 week. PTA duration tended to be from three to four times the length of the period of unconsciousness (Jennett & Teasdale, 1981, p.90; Miller, 1983, p.41). In less severe cases, however, the correlation between duration of coma and PTA length may be only modest as cases with relatively brief coma may have a disproportionately prolonged PTA (Levin, O'Donnell & Grossman, 1979). These authors also found PTA to be longer in patients with severe diffuse closed head injury or bilateral mass lesions than in patients with a unilateral focal mass lesion.

Disturbances of cognitive function in patients still in PTA are striking and intellectual functions may be affected (Mandleberg, 1975) as well as new learning and retention (Fodor, 1972; Levin, High & Eisenberg, 1988). Disturbances in evoked potentials in patients still in PTA have been reported as physiological correlates of the cognitive disorders found (Papanicolaou et al, 1984).

In the experience of Gronwall and Wrightson (1980) the degree of disorientation does not always correlate with the duration of PTA. In cases of minor head injury, they found that although most patients examined while in PTA were disoriented, several patients with a return of continuous memory were also disoriented, while others without continuous memory were oriented. Disorientation has several components which may recover at different rates: items to do with person tend to recover first, while some items to do with time recover last, and there is mixed intermediate recovery of person, place and time items (Daniel, Crovitz & Weiner, 1987).

When head injuries of varying severity have been included, studies have shown the length of PTA to be related to a number of outcome parameters such as neurological signs and symptoms (Russell, 1971); rating on the GOS (Brooks, Hosie, Bond, Jennett & Aughton, 1986; Levin, O'Donnell & Grossman, 1979); the presence of psychiatric sequelae (Bond, 1975; Brown, Chadwick, Shaffer, Rutter & Traub, 1981; Lishman, 1968) and the extent of, or rate of recovery from, cognitive impairment (Brooks et al, 1980; Mandleberg, 1976). Longer PTA has been shown to be related to greater impairment or slower recovery in the following neuropsychological areas: rate of information-processing (Gronwall & Wrightson, 1974, 1981); intellectual functioning (Bond, 1975; Brooks et al, 1980; Chadwick-Rutter, 1981; Winogron et al, 1984); and learning and memory (Brooks 1974, 1976; Brooks & Aughton, 1979a; Brooks et al, 1980; Gronwall & Wrightson, 1981; Parker & Serrats, 1976). In the Glasgow studies by Brooks and co-workers, PTA duration was found to be a better predictor of cognitive functioning after head injury than length of coma.

Studies not finding a relationship between PTA and cognitive outcome have tended to be based on small samples (eg Smith, 1974; Stuss et al, 1985) or have considered a narrow range of severity of injury, such as only mild and moderate head injury (eg Bornstein, Miller & Van Schoor, 1989). Nonetheless, there can be considerable individual variation in the amount of cognitive impairment occurring in different PTA severity categories (Brooks, 1984).

PTA is typically assessed by checking orientation or recall of day to day information, although there may not be absolute correspondence between these measures (Fortuny, Briggs, Newcombe, Ratcliffe & Thomas, 1980; Gronwall & Wrightson, 1980; Levin, O'Donnell & Grossman, 1979). Brief durations of PTA may be difficult to determine, but categorization into time spans such as less than 5 minutes or from 5 to 30 minutes is usually possible, although length of PTA may alter later in some mildly-injured patients (Gronwall & Wrightson, 1980). Islands of memory for specific events may occur before the patient regains continuous memory (Russell, 1971). Fortuny and co-workers (1980) found that estimates of PTA based on day to day recall of pictures and staff names corresponded closely to those of neurosurgeons based on interviews, suggesting that in a prospective study, independent observers can agree on PTA length.

3.4 Mechanism of Injury

The mechanisms of injury vary at different age levels and are associated with different pathophysiological states. In overseas studies, motor vehicle accidents are the major cause of head injury in patients between 5 and 65 years, whereas falls predominate at the younger and older age levels (Alberico et al, 1987; Luerssen et al, 1988). The

majority of severe injuries in children over 5 are traffic-related and there is a high incidence of diffuse injuries and a relatively low incidence of operable mass lesions (Alberico et al, 1987; Bruce et al, 1979; Zuccarello et al, 1985). Bruce and co-workers (1979) have suggested that the better recovery in children than in adults following head injury may be related to the fact that most are pedestrians hit at relatively low speeds, whereas the adults tend to be passengers in high-speed collisions and the degree of primary brain damage may be very different. Cumpsty (1986) has reported that in pedestrian injury, the majority of children are launched on impact and have acceleration-deceleration injuries. There may then be secondary impact to the head as the relative top-heaviness of children makes them likely to land head first.

In the retrospective study of head injury in the Cape Peninsula (de Villiers et al, 1984), transport-related accidents (predominantly pedestrian), falls, and assault/abuse accounted for 88% of all causes of head injury in children under 15.

Falls, in contrast to traffic-related injuries, are associated with a higher proportion of operable mass lesions, and coma may be associated with brain compression from an intracerebral haematoma (Alberico et al, 1987; Kalsbeek et al, 1980, cited Goldstein & Levin, 1987). Falls are the most frequent cause of subdural haematomas (Shapiro, 1985). Most falls occur on the level, but high falls can have a poor prognosis. Children decerebrate following a fall of three storeys or more are likely to die, whereas children decerebrate following low-speed pedestrian injury have a good chance of recovery (Bruce, 1982).

Assaults produce a higher proportion of surgically treatable mass lesions than of diffuse injuries (Alberico et al, 1987). Blows to the head with blunt objects are frequently associated with depressed skull fractures and underlying contusions or with penetration of the brain by bone fragments (Ommaya, 1982; Zimmerman & Bilaniuk, 1983). In missile injury, eg gunshot wounds, a shower of bone fragments may penetrate the brain at the point of impact; the missile may lacerate the brain tissue in its path, and the shock waves propagated may be intense (Gennarelli & Thibault, 1985).

In the study of Rivara (1984), which excluded motor vehicle accidents and intentional trauma, nearly all skull fractures beyond the toddler ages were caused by sports and recreational equipment. In a Scottish survey, 23% of new head injuries in the under 15 year old group coming to Emergency Departments were attributed to sport. Most of the head injuries were mild, but intracranial haematomas, persisting disabilities and deaths all occurred (Jennett & Teasdale, 1981).

In summary then, motor-vehicle related trauma tends to produce diffuse acceleration-deceleration brain injury, whereas non-motor-vehicle related trauma is associated with a higher incidence of focal mass lesions (Alberico et al, 1987). Mechanism of injury, however, has not proved a useful indicator of outcome and is not mentioned in many predictive studies.

3.5 Abnormal EEGs

Abnormal EEGs are far more frequent amongst head-injured children than among children generally and more frequent in children with greater severity of injury (Enomoto, Ono, Nose, Maki & Tsukada, 1986; Ivan et al, 1983). In children with relatively mild head injury, the EEG is abnormal in the early phase in from 56 to 65% (Enomoto et al, 1986; Klonoff et al, 1977). In both these studies the most common abnormality was slow wave activity, either diffuse, or predominantly in the occipital region. Asymmetry of background also occurred (Klonoff et al, 1977). In children with mild injuries, EEG abnormalities have tended to diminish in number and magnitude over time, and the majority have had normal tracings at 1 year (Enomoto et al, 1986; Ivan et al, 1983; Klonoff et al, 1977). In the study by Klonoff and co-workers, EEG recordings showed a more rapid rate of recovery than did neuropsychological test findings. At the 5 year follow-up, only 12% of the children had abnormal EEGs, whereas 24% had residual neuropsychological deficits. Abnormal EEG at intake did not contribute significantly to the prediction of residual neuropsychological deficits.

In the study by Winogron and colleagues (1984), the relationship between EEG abnormalities and deficits on neuropsychological tests approached statistical significance. An average of 88% of patients defined as "severely abnormal" in terms of percentage of neuropsychological tests on which they showed impairment, also had EEG abnormalities. However, the temporal relationship of EEG recording to testing is not clear in this study and there are no details on the nature or severity of the EEG abnormalities.

EEGs, although showing abnormalities in relationship to severity of injury, intracranial haematoma and focal signs, have not been regarded as providing additional data for determining prognosis in terms of gross outcome (Jennett & Teasdale, 1981, p.285). It is possible, however, that in a prospective study of head injuries of varying severity, EEGs may be found to contribute to the prediction of neuropsychological deficits.

3.6 Age Effects

Differences in the mechanism of injury, the frequency of haematomas, response of the brain to injury and the likelihood of systemic complications at different ages, contribute to relationship between increasing age and poor outcome (Alberico et al, 1987; Bruce et al, 1979). Children with head injuries have a higher percentage of good outcomes than adult patients and a significantly lower mortality rate (Alberico et al, 1987; Luerssen et al, 1988). Within the paediatric group there may also be age effects and a worse outcome in infants and preschool children than in older children has been reported in several studies (Alberico et al, 1987; Esparza et al, 1985; Hendrick, Harwood-Hash & Hudson, 1964; Humphreys, 1983; Luerssen et al, 1988; Mahoney et al, 1983).

Trends in their data towards a poorer cognitive outcome in younger children (defined as under 6, under 8, or under 10) have been noted by Filley et al (1987), Baracchini-Muratorio and co-workers (1985) and Brink and co-workers (1970) respectively. Other studies have not found significant effects for age of injury on cognitive recovery from head injury in children (Chadwick-Rutter study, 1981; Klonoff & Low, 1974; Klonoff et al, 1977; Levin & Eisenberg, 1979a,b), although Levin, Eisenberg and co-workers (1982)

did find more of the severely injured children than of the adolescents to have IQs under 80. Shaffer and associates (1980) found that the prevalence of reading backwardness was significantly higher in those with a longer duration of unconsciousness who had been injured before the age of 8.

The orderly development of cognitive skills in children is dependent upon normal brain growth and differentiation. Any loss of skills or interference with learning ability from even transient brain dysfunction may slow the order, rate and level of future development (Boll & Barth, 1981). Brain development appears to follow a reliable sequence with discrete regions progressing in "spurts" in terms of the generation of nerve cells and connections, with the frontal regions having a protracted process of development (Greenough, Black & Wallace, 1987; Stuss & Benson, 1986). Different brain structures may be responsible for behavioural functions at different ages, and later developing systems may compensate for earlier functional deficits or may reveal deficits not evident earlier (Bolter & Long, 1985; Goldman, 1974; Luria, 1963; Robinson, 1981). This means that brain damage may have no observed consequences until the skills subserved by the damaged region would emerge in the normal course of maturation (Kinsbourne & Hiscock, 1983). This theory may explain the high prevalence of reading backwardness and of other scholastic disabilities in children seemingly recovered from head injury.

4. THE PREMORBID DEVELOPMENT OF THE HEAD-INJURED CHILD

4. THE PREMORBID DEVELOPMENT OF THE HEAD-INJURED CHILD

4.1 Introduction

The cognitive consequences of head injury must be evaluated not only in terms of the extent of primary injury and secondary damage, but also in terms of the premorbid capacity of the individual. There is evidence that children who receive head injuries are not a random sample of the population (Jennett, 1972; Rutter et al, 1984). They tend to come from socioeconomically disadvantaged backgrounds or from stressed families (Brown et al, 1981; Fabian & Bender, 1947; Harrington & Letemendia, 1958; Hjern & Nylander, 1964; Klonoff 1971; Rune, 1970; Shaffer, Chadwick & Rutter, 1975) and to have a higher incidence of behaviour disturbances and learning disabilities prior to their head injuries (Black et al, 1969; Brown et al, 1981; Rutter et al, 1980; Costeff et al, 1985; Craft et al, 1972; Dressler, 1984; Fabian 1956; Mahoney et al, 1983). It therefore must be determined whether cognitive problems and behavioural disorders found after head injury were not, in fact, present premorbidly.

Another question, seemingly not addressed in the literature, is whether the environmental and personal factors that limited premorbid development may not also limit the potential for recovery? The following sections consider socioeconomic disadvantage, family stresses and premorbid behaviour disturbances in head-injured children and the relationship of such factors to premorbid cognitive development.

An analysis of the psychosocial backgrounds of the children aged 6 to 14 years in the UCT Head Injuries Survey has already been carried out (Theron, 1987) and findings are included in the sections below.

4.2 Socioeconomic Disadvantage

4.2.1 Socioeconomic Disadvantage in the Head Injured

Overseas studies of head injured children have indicated that children from the lower socioeconomic levels are over-represented, that there is a higher incidence of unskilled or unemployed fathers, that these children tend to come from overcrowded areas where housing is poor, and that there is a high incidence of single parents or broken or unhappy marriages (Brown et al, 1981; Klonoff, 1971; Woo-Sam, Zimmerman, Brink, Uyehara & Miller, 1970).

It has been suggested that these social problems reduce parental supervision and increase the child's risk of an accident (Gratz 1979; Husband & Hinton, 1972; Rivara, 1982; Wadsworth, Burnell, Taylor & Butler, 1983).

The UCT retrospective study of head-injured children in the Cape Peninsula (de Villiers et al, 1984) found higher percentages of coloured and black children in their sample than in the corresponding population. This suggested that socioeconomic factors were operative, in that these groups have lower levels of education, occupation and income than the under-represented white group (Central Statistical Services, 1982; Wilson & Ramphele, 1989). The UCT prospective head injury survey has confirmed the over-representation of the less privileged ethnic groups in that 75% of the head injured are coloured, 16% black and 9% white in comparison with population figures of 64% coloured, 10% black and 26% white, for 5 to 14 year olds in the Cape Peninsula (Theron, 1987).

4.2.2 Socioeconomic Disadvantage in the "Coloured" Population of the Western Cape

The "coloured" population of the Cape Peninsula is a Western orientated group of mixed descent. The report of the Theron Commission (1976) shows them to be a people in transition, a mosaic of heterogeneous subgroups, each with its own stratification pattern dependent upon its origin, skin colour, religion, social and professional status and life style. Twenty percent are employers, professional and clerical people with a middle class life style, and 40% are unemployed or underemployed and live in chronic poverty. Social class, as measured by the breadwinner's occupation, has been shown to correlate with a number of variables within coloured families, such as parental education, marital state, income, occupation density and the standard of care given to the child (Molteno, 1985).

Whereas the majority of the rural coloured population is Afrikaans speaking, only 64% of the metropolitan population of the Cape Peninsula is Afrikaans speaking, although a third of the English speakers have Afrikaans backgrounds (Scheffer, 1983). English is more common amongst the better educated and amongst those aspiring to the middle classes. Afrikaans is perceived as the language of the less educated, of unsophisticated country people, and of the apartheid government (McCormick, 1986). In the city, many people speak both languages. There is considerable language mixing and nonstandard Afrikaans constitutes the norm in informal discourse (McCormick, 1986; Scheffer, 1983).

Racial legislation has to a large extent influenced standards of housing and education, availability of medical care and community resources in South Africa. Available housing is overcrowded and about 50% of coloured families share a house with at least one other family. Tuberculosis

and other infectious diseases associated with poor housing conditions are major health problems (Molteno, Kibel & Roberts, 1986). Infant mortality declined dramatically from 1968 to 1977, but still has a Third World rather than a First World pattern, and the incidence of low birth weight children is high (Molteno et al, 1986). The ratio of expenditure on the education of White, Indian, Coloured, and African children is of the order of 100:63:43:14 respectively (derived from Corke, 1984). The pupil:teacher ratio in coloured schools in 1983 was 27:1 and half the pupils dropped out with 4 years or less of education and are thus functionally illiterate (Nasson, 1986). In the epidemiological data collected on the families of 1,000 coloured children born consecutively in Cape Town, it was found that a third of the mothers were single parents, 20% were under 20 years of age and 65% had less than standard 6 (8th grade) education (Molteno, et al, 1980).

These findings suggest that, in general, coloured children receiving head injuries are likely to have a high level of psychosocial disadvantage.

4.2.3 Socioeconomic Disadvantage Within the Prospective Sample

Analysis of the psychosocial backgrounds of the 6 to 14 year old head-injured children in the University of Cape Town Head Injury Survey found a high level of adversity related to socioeconomic conditions (Theron, 1987). In 46% of families the breadwinner was unskilled, had unstable employment or was unemployed. A third of the children were being reared by single parents or were not with their own parents. Half lived in overcrowded conditions. In 20% of the families, one parent suffered from physical illness and 13% of the children had chronic illness (eg asthma, bronchitis). Nutrition was inadequate or unbalanced (eg insufficient protein) for 22% of the children. Seventy

percent had not attended preschool and 65% had mothers with less than Std 6 education.

There was a suggestion of greater socioeconomic adversity in the group with very severe head injuries (PTA over 7 days) than in the other groups as the monthly income per dependent (R41) was lower and more children (34%) had inadequate nutrition.

Differences in the cause of injury in the UCT survey compared with overseas studies also suggest that a lack of a safe environment and of protective parents contributes to head injury in the Cape Peninsula. Assaults accounted for 13% of head injuries in the 6 to 14 year old group versus about 4% in overseas studies; while pedestrian traffic accidents are the major cause of head injury in the 1 to 5 year-olds, compared with domestic falls in overseas studies (Theron, 1987).

4.2.4 The Relationship Between Socioeconomic Level and Intelligence

Numerous studies have demonstrated an association between socioeconomic status (SES) and IQ or academic achievement (Rutter & Madge, 1976; see White, 1982 for a review). Social disadvantage tends to have a cumulative effect in that poor nutritional state in the mother, inadequate perinatal care, undernutrition, poor housing, large family size, low IQ and occupational status and unemployment in the parents, as well as lack of stimulating books and toys and inadequate facilities for studying, and a home atmosphere which is stressed or not encouraging of academic achievement, tend to occur in association with each other (Molteno, 1985; Rutter & Madge, 1976; Theron, 1987; Wolff, 1971). Studies such as the Collaborative Perinatal Project in the USA and the National Survey in Britain have shown that these factors contribute to cognitive development, although no single factor predicts

cognitive development with any degree of confidence (Nichols & Chen, 1981; Rutter & Madge, 1976; Spreen et al, 1984).

Sociological variables appear to be more important predictors of learning difficulties in children than pre- and perinatal complications (Nichols & Chen, 1981). Nutrition, except in the period before conception and in the embryonic phase, is not of critical importance for the child's intellectual development and only limits development of intellectual functioning if coupled with social deprivation and lack of stimulation (Stein & Susser, 1985). Similar conclusions have been reached by Montagu (1971) and Rutter and Madge (1976), who regarded malnutrition as affecting development through changes in attention, responsiveness and motivation rather than through a more direct impairment of cognitive competence.

The effects of a disadvantaged background are most evident in academic achievement (least evident in measured intelligence) and there is some evidence that retardation of verbal and academic skills is greater in older than younger children (Rutter & Madge, 1976). Such children are at risk for failure on reading and writing tasks (Rutter & Madge, 1976; Snow, Dubber & de Blauw, 1982).

It is clear from the previous sections that conditions of socioeconomic disadvantage that have been associated with low IQ and learning difficulties are present in the local community. Molteno (1985) following a random sample of 187 children from 1,000 consecutive births, found infant milestones to be normal by international standards but language delay was evident at 30 months, and development at 5 years (fine motor, language and drawing skills) correlated with social variables such as the social class of the breadwinner, income, occupation density, family stability and the mother's education. Other local studies

standardizing intelligence tests on large population samples have found a linear relationship between SES and IQ scores (Van den Berg, 1985). Claassen (cited Van den Berg, 1985) found there was no effect for race when SES was partialled out, but the SES index was distributed very differently for the different race groups, with 17% of the white children in comparison with 85% of coloured and 79% of Indian children being rated as socioeconomically deprived. SES accounted for a third of the variance in IQ scores and this makes it very important that the effects of SES should be controlled in any study of the effects of head injury.

4.3 Family Circumstances

4.3.1 Family Circumstances in the Head Injured

Studies from several countries have reported a higher incidence of family stresses and of parental psychiatric disturbance in the backgrounds of head-injured children than in control groups or in the population generally (Brown et al, 1981; Harrington & Letemendia, 1958; Hjern & Nylander 1964; Klonoff, 1971; Rune, 1970; Shaffer et al, 1975). A high incidence of marital instability and/or unhappiness amongst the parents of head injured has been reported by Klonoff (1971) and by Shaffer and co-workers (1975). Psychiatric disturbances including malaise and depression in the mothers, and alcoholism and criminality in the fathers have also been reported (Brown et al, 1981; Hjern & Nylander, 1964; Shaffer et al, 1975). The tension, disruption and neglect associated with such family circumstances may mean that the child's physical and intellectual as well as emotional needs are not met (Brown & Davidson, 1978; Rutter & Madge, 1976).

Family problems are common in the head-injured children in the UCT prospective survey: half the children come from homes with 4 or more children; half have disharmony in the

home; and a third of the mothers and of the fathers have psychiatric problems, mostly alcohol abuse. Such problems were rated as leading to neglect of the child in half of the head-injured cases, versus one quarter of the controls (Theron, 1987). Severity of injury appeared to be of importance in that exposure in the home to alcoholism, drug abuse, violent behaviour and criminal offences was more common in the children with very severe head injuries (PTA over 7 days) than in the less severely injured.

4.3.2 Family Circumstances and Intelligence

Home, social and economic problems that militate against a stable and supportive environment for the child, parents who are unable to function as teachers, and home values that place little emphasis on book and/or school learning, are some of the factors that limit intellectual development (Thorndike & Hagen, cited Van den Berg, 1985). After re-analysing 101 studies dealing with the contribution of socioeconomic factors to academic achievement, White (1982) concluded that "home atmosphere" correlated more highly with academic achievement than did any single or combined group of traditional SES indicators (eg occupational status, educational level or income) and accounted for about 30% of the variance. In a study of local children, Molteno (1985) found that the mother's personality and family stability, as well as SES indicators, correlated with the motor and language development of the children at 30 months and at 5 years.

School backwardness is also more common in families where there are 4 or more children. Large family size tends to be associated with other adverse variables such as poverty, overcrowding, marital unhappiness and frequent moves. Even when overcrowding and lack of household amenities are taken into account statistically, the association between family size and achievement remains and

individuals from such families tend to have lower verbal intelligence and inferior reading attainment (Rutter & Madge, 1976).

In conclusion, it seems that factors in the home that limit the responsiveness of the parents affect the development of the child and may contribute to inattention and a lack of motivation at school. In a disadvantaged sample, it would be very difficult to control all possible adverse factors statistically, but there is a need to assess their contribution to the cognitive outcome of head-injured children.

4.4 Behaviour Disturbance

4.4.1 Behaviour Disturbance in the Head-Injured Child

Several studies have reported a higher incidence of premorbid behaviour problems in children who suffer head injuries than among children in general (Black et al, 1969; Brown et al, 1981; Craft et al, 1972; Fabian, 1956; Hjern & Nylander 1964; Rutter et al, 1980). Most of these authors specifically mention restlessness, impulsivity, hyperactivity and/or distractibility as premorbid characteristics. Researchers have also suggested that it is because boys are more impulsive and aggressive and exhibit more risk-taking behaviour that they are over-represented in studies of children having accidents (usually double the number of girls), (Jones, 1980; Manheimer & Mellinger, 1967; Rivara, 1982; Rutter et al, 1984).

In the UCT prospective survey, boys are over-represented amongst the mildly injured, but the sex distribution is more equal in the severely injured (Theron, 1987). Furthermore, as in the Chadwick-Rutter study (Rutter et al, 1980) it is the mildly injured and not the severely injured who show an increased level of premorbid behaviour

disturbance. The behaviours on which the head-injured children in the local survey differed significantly from the controls premorbidly were restlessness, fidgetiness, irritability, eating problems, lying and quarrelling (Theron, 1987).

4.4.2 Previous Head Injury

Early studies found that fewer than 2% of head-injured children had had previous head injuries or previous trauma leading to hospitalization (eg Jamison & Kaye, 1974; Partington, 1960). However, Hjern & Nylander (1964) found that 30% of their 162 head-injured children had had previous accidents requiring medical treatment and 10.5% had had previous head injuries. These figures more closely approximate those in the UCT Head Injuries Survey, as about 30% of the head-injured children have had previous accidents requiring medical treatment and 17% have had previous mild head injuries (severe head injury was grounds for exclusion). Previous head injuries were more common in the mildly head injured (22%) than in the severely head injured (12%) in the UCT survey (Theron, 1987).

Studies of accident repeaters have suggested that it is their behaviour that puts them at risk for accidents. Previous accidents are associated with male sex (Annegers et al, 1980; Klonoff, 1971), and with mischievous, daring and extroverted behaviour (Attias et al, 1982; Jones, 1980; Manheimer & Mellinger, 1967). As it has been shown that adults with previous head injury have a greater initial deficit and take longer to recover mentally (Gronwall & Wrightson, 1975), the effect of previous head injury on children's recovery will need to be determined in this study.

4.4.3 Behaviour Disturbance and Scholastic Achievement

Children with low IQ and who are poor scholastic achievers, show a higher incidence of behaviour disorders than is found in the population generally (Robertson & Juritz, 1988; Rutter, 1981). There is a considerable overlap between hyperactive-impulsive children and children with learning difficulties and below average school performance. Boys are over-represented in both groups (Nichols & Chen, 1981; Rutter, 1984). A recent review of the problems of hyperactive children (Douglas, 1984) has suggested that they have defective attentional, inhibitory, arousal and reinforcement processes. At a superficial level, the behavioural descriptions of hyperactive or attention deficit disordered (ADD) children (eg Douglas, 1984), are remarkably similar to those of head-injured adults (eg Walsh, 1985). Such children have problems in concentrating, in inhibiting responses, in making the effort to apply strategies, in attending to errors, and they need external reinforcement and structuring to perform adequately. Such problems may be premorbid characteristics of up to about a third of children who receive mild head injuries (Brown et al, 1981; Black et al, 1969) and it is important that they should not be regarded as the consequences of head injury.

4.4.4 School Backwardness in the Head Injured

Premorbid school backwardness and/or learning difficulties have been reported as occurring in about a third of the children sustaining head injuries, whether these were predominantly mild injuries (eg Rutter et al, 1980; Hjern & Nylander, 1964; Klonoff & Paris, 1974) or severe injuries (eg Costeff et al, 1985; Mahoney et al, 1983). The Chadwick-Rutter group (1981) found 35% of their mildly head-injured children to have below average school achievement premorbidly in contrast to 14% of the severely injured. The mild group continued to have lower IQs than

the controls and 44% had reading backwardness throughout the 2 year follow-up. These deficits were regarded as attributable to their disadvantaged backgrounds rather than to the head injury. There was, however, no investigation to indicate whether the premorbidly backward children or others within the mild group, had the lower IQ scores and/or the reading backwardness.

In their study of children with focal head injury, Shaffer and co-workers (1980) found 55% to have reading backwardness 2 years or more after injury. (The expected prevalence of reading backwardness in a predominantly lower-class British inner-urban area was about 20%). Severe reading backwardness was associated with a history of post-traumatic cerebral oedema, and this association remained when the effects of social disadvantage were controlled. In boys under 8, reading backwardness was also associated with the length of coma. Other studies have also reported that, particularly where there is prolonged coma, it is duration of coma rather than premorbid academic status or pre-injury IQ that determines cognitive outcome (Dressler, 1984; Pelco, 1985).

In the UCT prospective survey, 42% of the head injured had failed a standard or were struggling at school. A recent study of head-injured adults has reported that up to 50% had poor premorbid academic performance and suggested that this is a risk factor for head injury (Haas, Cope & Hall, 1987). In studying the effects of head injury, it is important to realise that performance below test norms is not necessarily attributable to the head injury. Head-injured children need to be compared with controls from the same socioeconomic level, and those with and without premorbid scholastic difficulties need to be differentiated.

4.5 Conclusions

Socioeconomic disadvantage, family stresses, behaviour disorders and low IQ or poor school achievement tend to be associated and it is children with these histories that are over-represented in head-injured samples. Composite measures of psychosocial adversity have been devised by the British group (Brown et al, 1981; Shaffer et al, 1975). They found that new behaviour disturbance after head injury was related not only to the severity of injury, but also to psychosocial adversity and premorbid behaviour status. However, they did not investigate the relationship between these latter variables and cognitive outcome. In the local head injury survey, a similar scale to assess psychosocial adversity was devised (Hemp, Cumpsty & Theron, 1985) and found to correlate with premorbid behaviour disturbance (Theron, 1987).

Psychosocial variables are very complex and it would be difficult to control all of their effects statistically. It would also not be desirable to eliminate variables that are the natural characteristics of the groups under study. In the local head injury survey, as in the Chadwick-Rutter (1981) study, it appeared that children who suffered severe head injuries differed from those who suffered mild head injuries (Theron, 1987). Those with very severe head injuries tended to have the higher levels of socioeconomic disadvantage (in terms of poverty, poor nutrition, etc) and the greater exposure to adverse home environments (alcoholism, violence, neglect, etc). These children, nonetheless, had no greater premorbid behaviour disturbance than was found in the control group, and boys and girls were almost evenly represented. They were slightly younger than the other head injured, and were predominantly the pedestrian victims of road traffic accidents. In contrast, the children in the moderately head-injured group were

predominantly boys and had a higher frequency of premorbid behaviour disturbance. They were older and had a far wider range of accidents in which their behaviour probably put them at risk of injury.

In the present study, it will be important to consider the effects of psychosocial adversity, premorbid behaviour disturbance and premorbid school backwardness in determining cognitive outcome.

5. INTELLECTUAL AND SCHOLASTIC OUTCOME

Addendum

"Clinical neuropsychology is an applied science concerned with the behavioural expression of brain dysfunction" (Lezak, p.7, 1983).

"Intelligence is the aggregate or global capacity of the individual to act purposefully, to think rationally and to deal effectively with his environment" (Wechsler, cited Sattler, p.34, 1982).

The term "intelligence" as used in this text refers to performance on specific tests of cognitive ability. It is recognised that such measures do not necessarily reflect innate intelligence and that composite IQ scores in the brain disordered may be misleading (cf Lezak Chapter 2, 1983; Sattler Chapter 3, 1982).

5. INTELLECTUAL AND SCHOLASTIC OUTCOME

Two questions were posed in the Introduction about the nature of intellectual deficits after head injury: what severity of head injury is needed to produce transient or persistent deficits, and, are some cognitive functions more impaired than others? In this discussion the effects of mild head injury on intelligence-test performance and scholastic functioning are considered before the effects of severe head injury. The nature of deficits and the pattern of recovery is then examined.

5.1 Severity of Injury and Transient/Persistent Deficits

5.1.1 Mild Head Injuries

The Chadwick-Rutter study (1981) was concerned with the question of a threshold for intellectual impairment. Their mildly head-injured group with a PTA of less than 1 week had IQs that were lower than those of the controls at intake and throughout follow-up. However, they had not been matched to the controls for SES level and the group contained more socially disadvantaged children and a higher proportion of children had premorbid school backwardness. In determining which children would be classified as showing impairment after head injury, Chadwick and co-workers stipulated that there should be a pattern of recovery. They defined recovery as a gain on Performance IQ (PIQ) from initial assessment to the 1 year assessment that exceeded that found in 95% of the control group (ie a gain of 24 points or more). Those with such a gain were classified as having had transient impairment if their PIQ at final assessment was within one standard deviation of the control group's mean, and as having persistent impairment if their PIQ was below this cut-point. On this basis, only 2 of the 29 mildly head injured (7%) were regarded as having shown transient impairment, none as having persistent impairment. Rutter and co-workers (1984) concluded that "most of the children

with PTAs of less than 2 weeks showed no impairment, transient or persistent" (p.89). The mildly head injured with a deficit on PIQ, but no pattern of recovery (24%), were discarded from the analysis and their deficit assumed to have antedated the injury.

There was, however, some evidence of at least transient impairment within the mild group in that those with PTA from 1 to 7 days had a mean Verbal IQ (VIQ) and PIQ significantly below those of children with PTA of less than 1 day at the initial assessment, and nonsignificantly lower at the subsequent assessments. (These subgroups did not differ in terms of social disadvantage). Evidence of longer term problems possibly related to the head injury may be found in the fact that 45% of the mildly head-injured children had reading backwardness persisting at 2.25 years after injury, whereas only 35% were reported as having had below average school achievement prior to injury.

Within the limitations imposed by their lack of sorting for severity of injury, both the other prospective follow-up studies of head-injured children have suggested transient as well as persistent cognitive impairment in the mildly head injured (Black et al, 1971; Klonoff & co-workers, 1974; 1977). In the Klonoff study, only 17 children were unconscious for more than 1 hour, yet at intake the IQ for the 231 head-injured children was 10 points below that of their matched controls (Klonoff & Low, 1974). The head injured had significantly lower scores on most measures in an extensive neuropsychological battery at initial assessment and were still significantly lower than the controls on more than half the measures at 1 year post-injury. The number of measures on which they were impaired declined each year. Nonetheless, at 5 years post-injury, 24% of the 117 remaining children were found to have persistent neuropsychological impairment, 21% had failed or quit school, 8% were in special or remedial classes, and 24%

had neurological sequelae on examination. EEG was abnormal in 12%, and 40% still had complaints (eg personality change in 21%, headaches in 16%, learning problems in 11% and mood changes in 9%). There was an overlap of residual effects in that half the children rated as neuropsychologically impaired were also rated as neurologically impaired and/or had abnormal EEGs.

Taking school progress after head injury as an independent criterion of outcome, of the 33 children who had school failure or remedial class placement, Klonoff and co-workers found 46% to have neurological sequelae, 40% to have residual neuropsychological impairment and 6% to have abnormal EEGs. Overall, 67% of these 33 children had residual effects in at least one area (neurological, neuropsychological and/or EEG), suggesting that head injury contributed to their school difficulties.

In the study by Black et al (1969, 1971), only about 9% of the sample had unconsciousness between 1 and 24 hours and only 4% were known to have been unconscious for more than 24 hours. At initial assessment, however, more than 50% had PIQs below 90 and at 5 years more than 45% had Full Scale IQs (FSIQ) below 90. With no controls, it cannot be concluded that the deficit was attributable to head injury.

Gulbrandsen (1984) concluded that neuropsychological sequelae could be demonstrated 4 to 8 months after concussion producing less than 15 minutes of unconsciousness in children who had no neurological symptoms, few subjective complaints and were functioning normally in school. In a carefully controlled study with matched controls, she found the control group to be superior on 29 out of 32 variables in a neuropsychological battery. The concussion factor accounted for differences on the Wechsler Comprehension and Picture Completion subtests, and differences on the Category Test, Tactile Performance Test and Grooved Pegboard Test.

The presence of at least transient deficits is supported by the studies of Levin & Eisenberg (1979a,b) who found that more than a quarter of 14 children admitted awake and responsive to commands and without neurologic deficit were clearly impaired on language, visuospatial, somatosensory and motor-speed tests performed at discharge or after a longer interval. No comment can be made on transient effects on intellectual abilities as intelligence was not tested until a year or more after injury, when impairment was found to be limited to those with coma lasting over 24 hours.

Although intelligence tends not to be impaired in adults following mild head injury (McMillan & Glucksman, 1987), deficits demonstrated in the early days following mild head injury have included slowing of the rate of information-processing, and disturbances of orientation, attention, memory and visual-motor speed (Gronwall & Wrightson, 1974; 1981; Levin, Mattis et al, 1987; McLean et al, 1983; McMillan & Glucksman, 1987). Within the mild head-injury range a severity gradient with those with longer PTA obtaining lower scores, has been found (Gronwall & Wrightson, 1981; McLean et al, 1983). These deficits have tended to clear at about 4 to 6 weeks, but even such temporary impairment in children could interfere with the acquisition of new skills and with school learning.

In the studies of consecutive admissions, the percentage of children reported as failing or showing more persistent educational backwardness after head injury is probably not much higher than the percentage of severe head injuries in the sample. Thus Rowbotham and associates (1954) found 10% had to be placed in a lower standard at follow-up at 1 year, while Harrington & Letemendia (1958) found temporary educational backwardness in 23% of their casualty group sample, and more persistent educational backwardness in a further 13%, compared with a pre-accident

backwardness rate of 8%. Hendrick and co-workers (1964) commented that school work tended to suffer for 1 to 3 months after injury but usually returned to pre-traumatic levels thereafter, except in severe cases. Burkinshaw (1960) found a severity effect on school work in 135 children followed up by questionnaire 1 to 6 years post-injury: worse school work was reported in 5.5% of those with no loss of consciousness, in 10% of those with unconsciousness of less than 1 hour, and in 38% with unconsciousness of over an hour.

In a more detailed follow-up study, Rune (1970) found no apparent effect on IQ, reading comprehension or spelling abilities after a head injury sufficient to render the child unconscious. Studying all the primary school children in Umea, he found no significant differences between those who had been admitted to hospital for such an injury, those cared for at home (presumably less severe) and 100 controls. No differences were found in terms of neurologic state, EEG or psychiatric state either. Lundar and Nestvold (1985) have also reported normal school progress in all but 1 of 126 children after traffic accidents resulting in predominantly minor head injury.

However, as already mentioned, the possibility of late effects on school progress was raised in the studies by Klonoff and co-workers (1977) and Chadwick and co-workers (Chadwick, Rutter, Brown, Shaffer & Traub, 1981). The study of children with focal head injury by Chadwick and co-workers also found a high prevalence of reading difficulties (55%) more than 2 years after injury, although only a quarter of the sample had been unconscious for more than 3 days (Chadwick, Rutter, Thompson & Shaffer, 1981; Shaffer et al, 1980). Gronwall and co-workers in Auckland found that a far higher percentage of reading problems were reported by the parents of children who as preschoolers had sustained minor head injuries (76%), than by the parents of children who had had minor accidents not involving the head (15%). A

difficulty was that only a very small portion of the total sample could be traced, but work in progress seems likely to confirm an unduly high prevalence of reading problems following minor head injuries (Gronwall, unpublished paper).

More persistent cognitive deficits in at least a proportion of those with mild head injury have been suggested by the follow-up studies of Winogron et al (1984) and Flach and Malmros (1972). Winogron and co-workers found that 12% of those with moderate head injury (defined as unconsciousness over 20 minutes and/or intracranial lesions, EEG abnormalities or neurological signs) as well as of those with mild head injury (defined as unconsciousness less than 20 minutes) had PIQs and FSIQs two or more standard deviations below the mean when they were tested approximately a year post-injury. A quarter also had scores two or more standard deviations below the normative group on 20% or more of the tests in the neuropsychological battery.

Increasing the length of follow-up to 10 years post-injury, Flach and Malmros (1972) reported that 43% of their 53 children with mild head injuries (simple skull fractures and/or concussion) were rated as "organic" on psychological tests (although perhaps 8% of these would have earned such a rating premorbidly). Twenty-five percent of this mild sample were rated as socially maladjusted and almost all of them were regarded as organic on their tests. Children with complicated fractures who had been unconscious less than 1 hour tended to be socially adjusted, to be rated as normal by their schools and not to have organic disturbances on psychological tests.

This study highlights the importance of social adaptation following head injury. Parents' attitudes to head injury can influence adaptation (Brown et al,1981; Hjern & Nylander, 1964). Post-concussional symptoms and behavioural changes, which may have a greater effect on a child's adaptation after head injury than the presence of

cognitive or physical disabilities, are common after mild head injury (Black et al, 1971; Boll, 1983; Klonoff & Paris, 1974; Klonoff et al, 1977; Lundar & Nestvold, 1985; Rowbotham et al, 1954).

5.1.2 Severe Head Injuries

Turning to the more severe spectrum of head injury (coma of more than 6 hours or PTA exceeding 7 days), most studies have reported significantly lower mean scores on a variety of measures for such children, but the proportion of children impaired is also of interest, as are the variables associated with good or poor recovery. Generalizations are difficult as the studies have included different ranges of coma and PTA duration. Most of those reporting on children in coma exceeding 6 hours have rated from 40% to 50% as having had persistent cognitive deficits (eg Flach & Malmros, 1972; Hjern & Nylander, 1962; Levin & Eisenberg et al, 1982; Winogron et al, 1984); whereas the studies from rehabilitation centres which have included children with coma lasting more than a week have found from 60% upwards to have persistent impairment (eg Brink et al, 1970; Filley et al, 1987; Stover & Zeiger, 1976).

The Chadwick-Rutter study (1981) found a clear pattern of increases in the extent of IQ test impairment with increases in severity of injury when they divided the severely head-injured into those with PTA of 1 to 2 weeks, 2 to 3 weeks and 3 weeks plus. Imposing a recovery criterion (large gains on PIQ), they regarded 32% as having had only transient intellectual impairment and 28.5% as having persisting impairment. Cognitive impairment was associated with the presence of neurological abnormalities, but could exist independent of such abnormality.

In the studies by Levin, Eisenberg and co-workers (1979a,b & 1982) almost all the children in coma exceeding 24 hours and more than half the adolescents were initially impaired in language, visuospatial, somatosensory and motor-

speed composite functions. At the longer term follow-up 40% of 15 children tested had a PIQ below 80 and 33% had a VIQ below 80.

In the study of Winogron and co-workers (1984) 47% of the 17 severely injured subjects had an IQ two or more standard deviations below the test mean at approximately 1 year. A very similar figure (46%) was given by Flach and Malmros (1972) when 125 children were tested 10 years after injury, only a third of whom had severe head injury. Thirty-seven percent had intellectual deterioration reflected by a reduced Performance IQ and a wide dispersion of subtest results, while disturbances of abstract thinking, concentration and/or slowness occurred in 22%. Half to three quarters of those with brain stem involvement, complicated fractures with laceration, or who had had cardiac or respiratory arrest were rated as organic on their tests or as socially maladjusted because of their slowness. Even when not rated as maladjusted, they were noted "to behave as if the top has been cut off their personality. They lack constructive imagination, power of abstraction and maturity" (p.14). This remark is echoed with regard to the school performance of children in regular classes following head injury by Kaiser and Pfenninger in Switzerland (1984). These authors further stated that altered personality (reduced endurance and control of emotions) was more frequent than loss of intelligence or psychomotor retardation.

The study of Colam (1984) was based on children admitted to the neurosurgery department of the hospitals in the UCT prospective study. The sample may have been biased as only 27 out of 191 children who had sustained head injuries of sufficient severity to cause loss of consciousness 3 to 15 years previously, responded to a letter requesting follow-up. Wechsler IQs and scores on the motor, language, visuographic and memory tests tended to decrease with increasing length of PTA, with the worst

scores consistently attained by those with PTA exceeding 21 days. A PIQ below 68 occurred in 64% of those with PTA of more than 1 week, but in none of the small subsample with PTA of less than 1 week.

School attendance may be seriously disrupted by severe head injury. Studies from countries including Britain, USA, Israel, Denmark and Switzerland have commented that children returning to regular class following severe head injury have needed special tutoring, have taken longer to learn, were no longer above average or were in some way not the same as they were before injury, in spite of still attaining relatively normal IQ scores (Costeff et al, 1985; Flach & Malmros, 1972; Fuld & Fisher, 1977; Gruskiewicz, Doron, & Peyser, 1973; Naughton, 1971; Raphaely et al, 1980). Head-injured children often perform well below the expectations raised by their intelligence quotients (Brink et al, 1970; Costeff, 1985; Fuld & Fisher, 1977; Mahoney et al, 1983; Rutter, 1982). The qualitative comments of authors may thus be of more clinical value than their quantitative findings. Neuropsychological difficulties may also not be immediately apparent, but only emerge as new developmental tasks (eg reading or abstract reasoning) make demands for new skills (Boll & Barth, 1981; Bishop, 1981; Haarbauer-Krupa, Henry, Szekeres & Ylvisaker, 1985). Costeff and co-workers (1985) have claimed that early prognoses tend to be over optimistic and that the full degree of learning difficulty and/or school failure may only be apparent after several years.

Naughton (1971) reported that only one third of 38 survivors of severe head injury were able to return to school within 6 months and that 21% were unable to return within 2 years. He typified the children as having diminished alertness and self control and stated that they had difficulty in dealing effectively with situations that required quick perception, normal retention, sustained attention and concentration, grasp of something new and emotional control appropriate to their age.

Heiskanen and Kaste (1974) in a follow-up study 4 to 10 years after injury of children who had been in coma for more than 24 hours, reported one quarter as in special school or unable to return to school and a further quarter as struggling. Almost all of those succeeding fairly normally had been unconscious less than 15 days, whereas only 1 of 9 in coma for more than 2 weeks was managing even moderately well. They suggested that 2 weeks is the upper limit for unconsciousness after which only exceptionally will a child be able to make normal progress at school. Vapalahti from Finland also stated this limit (Leary, 1982). The far more optimistic claims of Carlsson and co-workers (1968) of full mental restitution after even 4 weeks of coma, were based on definition of "coma" as a far lighter state than is reflected in current definitions (Jennett & Teasdale, 1981).

Modern management techniques have contributed to better outcome following severe head injury (Bruce, 1983; Gobiet, 1977), but there is, nonetheless, evidence that the outcome may not be as good as claimed. For example, Bruce (1983) reported that up to 70% of the most severely head injured (GCS 3 and 4) can function normally at school. A more detailed report of the same group, however, stated that, while 77% of the 120 children with severe head injury eventually returned to a regular school setting, psychological testing showed significant slowing of response, reduction in Performance IQ and difficulty with auditory and visual perception in the group (Raphaely et al, 1980).

The studies by Baracchini-Muratorio and co-workers in Italy (1985) and Mahoney and associates in Baltimore (1983) both claimed that just over 60% of children with severe head injuries were within the boundaries of normality (defined as IQs over 80) or had attained premorbid levels. However, reading of the details in both studies suggests significant

neuropsychological impairment in the children regarded as normal. In the Italian study, discrepancies between VIQ and PIQ, plus the performance on other tests, suggests that these totals are masking specific deficits. Furthermore, IQs in the low average range may represent reduced functioning in multitested children (cf Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Klonoff et al, 1977). In the Baltimore study, only 9% of 34 survivors of severe head injury were reported as having severe intellectual and motor handicap, and a further 53% as having learning or behavioural disorders, but half this latter group were claimed to have had such problems prior to injury and therefore to have returned to pre-traumatic status. The case examples given, however, show that above average intelligence may coexist with marked perceptual and learning deficits (case 1), and that post-traumatic learning difficulties may differ in nature and magnitude from those present pre-accident (case 2).

Costeff and co-workers (1985) separated the outcome of children with premorbid cognitive and behavioural difficulties (37%) from that of children without such difficulties. It was clear, nonetheless, that almost all the children had lost cognitive abilities: "frank dementia" occurred in 17%, 31% were failing or newly placed in special class, and a further 29% had new learning difficulties although they had normal IQ or were in the same class as before. Only 5% of the 36 cases (14%) were still rated as normal students, but were average performers whereas previously they had been superior.

The Costeff sample was from a rehabilitation centre and in half of the cases coma exceeded 3 weeks. It was estimated that they represented the 5% most severely damaged survivors of head injury (Groswasser, Costeff & Tamir, 1985). Other studies from rehabilitation centres where children have been in prolonged coma, have also reported a high percentage of cognitive, social and emotional problems

persisting once focal, motor and sensory deficits have resolved and daily activities have been resumed (Brink et al, 1970; 1978; 1980; Filley et al, 1987; Eiben et al, 1984; Pelco, 1985; Stover & Zeiger, 1976). Studying children 1 to 7 years after coma which had lasted more than 1 week, Brink et al (1970) reported one third to have normal intelligence, one third to be borderline retarded, and one third to be retarded (IQ under 70). Nearly 80% were attending classes for the physically or educationally handicapped and those in normal classes were not performing up to their intelligence scores.

In the study by Eiben and co-workers (1984), cognitive and communication problems contributed the most to dependency. Filley and associates (1987) found school or vocational functioning to be impaired in about 60% with coma lasting between 1 week and 1 month and in almost all those in coma more than 1 month. Behavioural problems were again very prominent (88%) and were characterized by deficits in arousal and emotional modulation. Similarly, Stover and Zeiger (1976) reported that none of their patients in coma exceeding 1 week had made a complete recovery to pre-injury personality, psychological and physical status, although 28% had only minimal physical or mental handicap. Pelco (1985) found IQ tested 0 to 6 months post-injury to correlate highly with IQ tested 13 to 18 months post-injury. Recovery of IQ had plateaued in all but 2 out of 109 patients by 12 months. Only 28% of the sample recovered to within one standard error of premorbid IQ on multiple retestings.

5.1.3 Conclusions on Severity

From the studies reviewed it would appear that mild head injury can produce at least transient cognitive impairment and that there may be persisting impairment in from 12% to 25% of cases. IQ may be relatively normal, but there are cognitive difficulties which may hinder school progress for some months, and later learning difficulties, particularly reading backwardness, are unexpectedly

frequent. Severe head injury (producing coma exceeding 6 hours, PTA over 7 days), definitely impairs cognitive functioning and persistent impairment may be found in from 40% upwards, with some deficits only emerging when new learning skills are required at school. With coma exceeding 1 week, over 60% are likely to have persisting impairment and this percentage rises where patients with longer coma are included. A return to the rate of cognitive growth and emotional mastery that might have occurred pre-injury is rare after 2 weeks of coma. With severe head injury, the rule of thirds seems to apply: one third are severely impaired, one third moderately impaired, and one third apparently coping, but not at pre-injury level. Cognitive and behavioural problems are likely to be more limiting than physical problems and school progress is likely to be below that expected from the IQ score.

5.2 Test Patterns

5.2.1 Factors on the Wechsler Scale

The Wechsler Intelligence Scale for Children-Revised (WISC-R, Wechsler 1974) has been found to measure three factors identified as (1) verbal comprehension or verbal-educational (all Verbal scale subtests except Arithmetic and Digit Span), (2) perceptual organization or spatial-mechanical-practical (all Performance scale subtests except Coding), and (3) freedom from distractibility (FFD, the Arithmetic, Digit Span and Coding subtests) (Blaha & Wallbrown, 1984; Sattler, 1982). Ownby and Matthews (1985) have suggested that the third factor may be more complex than FFD, and may measure complex cognitive processes related to strategy usage in an academic context. Blaha and Wallbrown (1984) have suggested that only Arithmetic and Digit Span, which rely on concentration and short term memory (STM), be used to define FFD.

5.2.2 Test Patterns after Brain Damage

Research has not supported early investigators' beliefs about a uniform set of behavioural consequences following brain injury in children (Fletcher & Taylor, 1984) and there is no predictable profile of test scores "specifically, necessarily or even dependably" associated with such damage (Boll & Barth, 1981, p.425). There are, however, three trends which are important for research studies based on differences between groups (but which are of little value for the clinician in dealing with the individual). Firstly, the main effect of bilateral or diffuse brain damage in children is on intelligence; secondly, visuospatial skills tend to be more affected than verbal abilities, and, thirdly, there is a variability in cognitive skills and an inconsistency of response (Boll & Barth, 1981; Chadwick & Rutter, 1984; Rutter, 1982; Szatmari, 1985).

These features are reflected on intelligence scores by a general lowering of scores, large verbal-performance discrepancies and a wide subtest scatter. Such patterns have in fact been found to characterise children with head injury (Baracchini-Muratorio et al, 1985; Bawden et al, 1985; Black et al, 1971; Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Chadwick, Rutter, Thompson & Shaffer, 1981; Filley et al, 1987; Flach & Malmros, 1972; Klonoff & Low, 1974; Levin & Eisenberg, 1979b; Raphaely et al, 1980; Richardson, 1963). Support for the claim that diffuse brain damage in children lowers general intelligence has come from studies of such diverse conditions as meningitis, encephalitis, kernicterus and rubella (Klonoff & Low, 1974), CNS treated cancer (Fletcher & Copeland, 1988; Moss, Nannis & Poplack, 1981), gross undernourishment (Stoch, Smythe, Moodie & Bradshaw, 1982), tumours (Eiser, 1981; Ellenberg, et al, 1987), and epilepsy in association with other neurological disorders (Addy, 1987; Rutter et al, 1970). Bruce has commented that after radiation therapy, tumour therapy and head injury, the neuropsychological deficits in

children look remarkably similar: the incidence of focal abnormalities is low, but diffuse abnormalities, particularly in respect of Performance IQ, are marked (see Discussion, Levin & Eisenberg et al, 1982).

The trend for Performance Scale or visual-motor tests to be more impaired than verbal tests has been reported in children with various conditions producing diffuse or even unilateral brain damage (Boll & Barth, 1981; Chadwick & Rutter, 1984; Dennis et al, 1981; Riva & Cazzaniga, 1986). This pattern has been variously attributed to the speeded nature of the tests, or the greater demands made on attentional skills, fluid intelligence, and/or intermodal processing of unfamiliar stimuli (Fletcher & Copeland, 1988; Telzrow, 1987).

Focal or lateralized lesions in children have been reported to lower both Verbal and Performance IQ (Bishop, 1981; Woods, 1980) and to produce less specific cognitive effects than those found in adults (Chadwick & Rutter, 1984; McFie, 1961). In adults, a trend for left hemisphere (LH) lesions to impair verbal tests and for right hemisphere (RH) lesions to impair visual-spatial tests has been reported in association with trauma (Newcombe, 1969; Uzzell, Zimmerman, Dolinskas & Obrist, 1979) and other brain pathology (Bornstein & Matarazzo, 1982; Herring & Reitan, 1986; McFie, 1975; Warrington, James & Maciejewski, 1986). This adult pattern of lateralized effects may be more apparent when lesions occur late rather than early in childhood (ie after 5 years) (Vargha-Khadem, O'Gorman, & Watters, 1985; Woods, 1980).

The study of focal head injury by Chadwick, Rutter, Thompson & Shaffer (1981) found no locus or laterality effects when children were tested at least 2 years after compound skull fractures with dural tearing. This may have been because the lesions were no longer acute, or had been too superficial to impair functioning. Cognitive deficits

were slightly more marked on visual-spatial and visual-motor tests than on verbal tests. PIQ was associated with severity of injury and VIQ with SES. There was a consistent tendency for tests of scholastic attainment to be more impaired with LH lesions.

Reservations about attributing functional deficits to localized lesions when head injury involves diffuse damage and nonspecific effects, were expressed in the Introduction. Most tests are multidetermined and there may be several explanations for failure or success on a particular test.

5.2.3 Test Patterns in Disadvantaged Children

Like brain damage, socioeconomic disadvantage tends to have a generally lowering effect on development, intelligence and academic achievement (Kaufman, 1979; Molteno, 1985; Rutter & Madge, 1976; Sattler, 1982; van den Berg, 1985). Lower socioeconomic groups may have a mean IQ half to one standard deviation below the mean of the standardization sample (Munday & Rosenberg, 1979). The mean of these low SES groups is in the low 90's, which was what a preliminary analysis suggested would be the case for the control group in the present study (Hemp et al, 1985).

In contrast to the trend for brain-damaged children, however, socioeconomically deprived children tend to have discrepancies favouring their Performance IQ over their Verbal IQ (Claassen & du Plessis, 1986; Hemp et al, 1985; Kaufman, 1979; Rutter & Madge, 1976). Children with PIQ greater than VIQ tend to score lower on measures of academic achievement than children with the reverse pattern (Moffitt & Silva, 1987).

5.3 Recovery

Patterns of recovery can only be determined from studies with serial testing. As these are few in the child head-injury literature, reference will be made to the adult head-injury literature.

5.3.1 The Rate and Magnitude of Recovery

The time course of recovery in children with mild head injury has not been as thoroughly investigated as in adults with mild head injury. Deficits in adults in information-processing, reaction time, attention and memory, have tended to clear at about 4 to 6 weeks (Dikmen et al, 1986; Gentilini et al, 1985; Gronwall & Wrightson, 1974; Levin, Mattis et al, 1987; McLean et al, 1983), although the head injured may have had scores which were nonsignificantly lower than those of the controls, and practice effects may have been mistaken for recovery in those studies where controls were not retested.

In the study by Klonoff and associates (1977), recovery appeared to continue over a longer period as the number of tests on which head injured and controls differed diminished each year, but it may have been the more severely injured who were responsible for this course. The group with residual neuropsychological impairment at 5 years were characterized by low initial Full Scale IQ (92) relative to the controls (116), which was still significantly lower at 5 years (99 vs 120). In contrast, the group without residual neuropsychological impairment had a higher initial IQ (106) and showed recovery such that at 5 years their IQ (116) was no longer significantly lower than that of the controls. IQ as measured during initial hospitalization was the variable most predictive of sequelae in the neurological, subjective, personality and intellectual areas. The only other variable approaching significance was minutes of unconsciousness.

In the study by Black et al (1969; 1971) there was a slight increase each year in the percentage obtaining an IQ above 90, with drops at 3 months and 5 years. However, as numbers varied vastly from occasion to occasion (n=12 to 62), no conclusions can be drawn about the time course of recovery, other than that a greater percentage started with PIQ impaired than with VIQ impaired, and this trend had reversed by 2 years.

Chadwick, Rutter, Brown, Shaffer & Traub (1981) administered Digit Span and Coding to the severely head injured at 4 months and found much of the recovery to have taken place by then. Gains on both VIQ and PIQ were significant in the severely injured in the first year, but not thereafter. Costeff and associates (1985) also reported that no further recovery of cognitive functions took place after 6 months to 1 year. In adult studies with testing in the course of the first year, VIQ has appeared to recover within the first 3 to 6 months and PIQ within 6 to 12 months, (except where PTA was either over 4 or over 8 weeks, when recovery continued for longer, but was more gradual and less complete) (Bond, 1975; Mandleberg, 1976).

In the Chadwick-Rutter study (1981), gains in the first year on PIQ were larger than those on VIQ, but whereas VIQ had recovered by the 1 year testing, PIQ was still significantly lower in the severely head injured than in the controls at 2.25 years. This has been the pattern in several studies of adult head injured (Becker, 1975; Brooks & Aughton, 1979b; Mandleberg, 1976; Mandleberg & Brooks, 1975; Roberts, 1979).

Klonoff and co-workers (1977) did not differentiate between VIQ and PIQ, but FSIQ remained significantly reduced in the head injured relative to the controls throughout the 5 year follow-up. In contrast, performance on tests in the neuropsychological battery that were based on speeded

performance appeared to be reduced only at intake and perhaps at the 1 year follow-up, and errors on some perceptual-spatial tasks continued only up to the 2 year follow-up. This again suggests that intelligence is amongst the functions most severely affected by head injury.

5.3.2 Severity of Injury and Recovery

The Chadwick-Rutter study (1981) found the magnitude of recovery on VIQ and PIQ within the severe group in the first year to exceed that shown by their controls. Gains on verbal fluency, manual dexterity, the Continuous Performance test and paired associate learning were also significantly greater than those shown by the controls. Gains in the mildly head-injured group did not exceed those shown by the controls and they were therefore regarded as showing practice effect rather than recovery. (The subgroup with PTA lasting 24 hours or more had greater gains on PIQ than the subgroup with PTA less than 24 hours).

In the Klonoff and Low (1974) study, a large number (n=196) of children with head injuries of mixed severity were retested at 1 year. They showed significant gains on almost all measures, but none of their gains on the IQ scales or subtests (and only isolated gains on the extensive battery of neuropsychological tests) were of a greater magnitude than those found in the matched controls. (A similar pattern emerged when gains over 5 years were considered Klonoff et al, 1977). In contrast, the children with chronic brain damage (from meningitis, encephalitis, kernicterus and rubella) did not show significant gains on the IQ subtests at retesting at 1 year, and the gains shown by their matched controls were significantly larger (Klonoff & Low, 1974). These findings suggest that, when age effects are controlled, there may be no significant gains in the absence of improvement in the underlying condition, and that what intact controls may learn as a result of practice on the tests may outstrip what can be learned by the brain damaged.

5.4 Conclusions

Mild head injury appears to produce transient cognitive impairment which recovers, at least in most adults, within about 6 weeks. It is suspected that there is a subgroup within the mildly injured that do not recover as well on IQ or neuropsychological tests. Whether these are the more severely injured or are duller to start with, is not known.

In the severely injured, the deficit is greatest initially and most of the significant recovery that occurs, occurs within the first year, although a more gradual recovery may continue thereafter. It is not clear that the head injured will show gains on IQ of a greater magnitude than are found in controls. It appears, referring also to the studies mentioned in the Introduction, that brain-damaged groups show no gains on IQ in the absence of improvement in the underlying condition, whereas controls show practice effects. Performance scale (visual-motor, visual-spatial, speeded) tests tend to show both greater initial impairment than the Verbal scale tests and steeper improvement over time, but nonetheless may still be significantly impaired years afterwards, when Verbal Intelligence has recovered. Reading and other scholastic difficulties may emerge late after head injury and be out of proportion to IQ recovery.

5.5 IQ Patterns Expected in the UCT Survey

1. The controls will have PIQ greater than VIQ.
2. The head injured will have VIQ greater than PIQ.
3. PIQ will show greater improvement over time than VIQ.
4. Those with poor premorbid school progress will have a lower IQ than those with adequate premorbid progress, but differences between the groups will be based on severity of head injury.

6. SPEECH AND LANGUAGE DEFICITS

6. SPEECH AND LANGUAGE DEFICITS

The pattern of language deficits which develops in children following cerebral insult is very similar, whether the insult is diffuse (as in closed head injury) or is the result of a focal left hemisphere lesion of traumatic or other origin. The most obvious deficits are in expressive language, and initial mutism is followed by a reduction of spontaneous speech, writing and gesture. Dysarthria, hesitations and impoverishment of the lexical stock are common, with particular difficulty in naming and word fluency. Disorders of reception are not as obvious and are less common, but are found if complex verbal functions are assessed. Logorrhea and paraphasias are rare. (Alajouanine & Lhermitte, 1965; Chadwick-Rutter study, 1981; Colam, 1984; Cooper & Flowers, 1987; Gaidolfi & Vignolo, 1980; Guttman, 1942; Hecaen, 1976, 1983; Johnston & Mellits, 1980; Levin & Eisenberg, 1979a,b; Vargha-Khadem et al, 1985; Winogron et al, 1984; Woods & Teuber, 1978).

In adults, a similar pattern following closed head injury, is found. Naming and word fluency are the most impaired, reception problems are found if looked for, and disorders of repetition are rare. (Brooks & Aughton, 1979a; Brooks et al, 1980; Groher, 1977; Heilman, Safran & Geschwind, 1971; Levin, Grossman & Kelly, 1976; Levin, Grossman, Sarwar & Meyers, 1981; Najenson et al, 1978; Sarno, 1980, 1984; Thomsen, 1975, 1984). Verbal circumlocution and paraphasias are more common in adults than in children following closed head injury (Levin, 1981), but neologisms and paraphasias have been reported in children with aphasia secondary to mild closed head injury (van Dongen & Visch-Brink, 1988).

6.1 Hemispheric Lateralization and Plasticity

Theories suggesting early equipotentiality of the cerebral hemispheres for language development, a more

bilateral representation of language in children than in adults with lateralization occurring as the child matures, and greater plasticity in ability to transfer functions from one area to another, have recently come under critical review (see Bishop, 1981; Carter, Hohenegger & Satz, 1982; Fletcher & Satz, 1983; Hecaen, 1983; Pirozzolo, Campanella, Christensen & Lawson-Kerr, 1981; Satz & Bullard-Bates, 1981; Witelson, 1987; Woods & Teuber, 1978). Early specialization of the left hemisphere for language has been suggested by anatomical, electrophysiological and perceptual asymmetries in very young infants (Kinsbourne & Hiscock, 1983). Studies of children suffering unilateral lesions after speech has been acquired, have found aphasia to occur in about 74% of children with left hemisphere lesions and in from 5% to 14% of those with right hemisphere lesions (Hecaen, 1983; Woods & Teuber, 1978).

Woods and Carey (1979) stated that there is an inverse relationship between age at lesion and the degree of language recovery, a position which is supported by the study of Vargha-Khadem et al (1985). However, this relationship is far from clear and, although recovery may be dramatic in many children, the symptoms may be unremitting in 25 to 50% and there is a high incidence of late cognitive and academic problems in those "recovered" from aphasia (Alajouanine & Lhermitte, 1965; Satz & Bullard-Bates, 1981).

6.2 Severity of Injury and the Prevalence of Deficits

In children or adults with severe head injury clinically evident aphasia occurs in only about 3% (Costeff et al, 1985; Filley et al, 1987; Heilman et al, 1971; Levin, Madison et al, 1983). However, speech and language disturbances occur in a far higher percentage, particularly in the early stages. The intrahemispheric location of any mass lesion is a major determinant of acute linguistic defects, but duration of coma is more closely related to linguistic functions in the later stages of

recovery (Levin, Grossman, Sarwar & Meyers, 1981). Johnston and Mellits (1980) found that longer coma duration in children (half of whom had a traumatic aetiology) was related to poorer language outcome and that all 16 of the children in coma over 21 days had very low expressive language quotients.

Levin and Eisenberg (1979a,b) found language deficits on testing in almost one third of their series of 64 children and adolescents assessed prior to discharge or within 6 months following closed head injury. Defects occurred in those with mild and moderate head injury ($\pm$22%) as well as in the more severe cases. A more detailed study of language functions after head injury by the same group (Ewing-Cobbs, Levin, Eisenberg & Fletcher, 1987) found at least 20% of children suffering mild and moderate/severe head injury to have defective scores (below the sixth percentile) on language tests administered at about one month after head injury. Winogron et al (1984) found errors on an aphasia screening test administered at approximately 1 year post-injury to be correlated with Glasgow Coma Scale score on admission and with the duration of altered consciousness.

6.3 The Nature of Speech and Language Deficits

6.3.1 Mutism

In the International Data Bank studies, only 16% of head-injured patients who could open their eyes following coma were unable to utter words, and only 4% of those who could obey commands did not speak (Jennett & Teasdale, 1981). However, studies of children, particularly of those with severe injury, report a far higher incidence of mutism in the early recovery period. For example, Filley and co-workers (1987) report initial mutism in 40%, the same rate occurring in those with diffuse lesions alone and in those with diffuse plus focal lesions; while Hecaen (1983) reported that mutism occurred in half his sample of 34

children with left focal lesions and was highest in those with a traumatic aetiology (85% of 12 cases) and, disregarding aetiology, was associated with fronto-rolandic lesions (63%) rather than temporal lesions (10%). Mutism following closed head injury has been found to occur in association with left hemisphere cortical lesions or basal ganglia lesions (Levin, Madison et al, 1983). In children with severe head injury and brain stem involvement, speech returns late in the recovery process and in the study of Gruskiewicz and co-workers (1973), 60% had speech disturbances in the absence of signs of dominant hemisphere involvement. Where these disturbances persisted, it was in association with residual brain stem signs.

Even where there is not actually mutism, spontaneous speech may be extremely limited and Alajouanine & Lhermitte (1965) report that it was almost nil in their 32 cases with left hemisphere lesions (13 of whom had had a traumatic insult). There may be many reasons other than brain damage why a young child remains silent after head injury, including for example, strangeness or a lack of formal speech. Todorow (1975) described a "Sleeping Beauty" syndrome in children following head injury and regarded akinetic mutistic states early on in recovery as psychological reactions. No doubt an Intensive Care Unit is a frightening experience for a child, but interpretation of the psychomotor inhibition as purely psychological, seems questionable. Reductions in spontaneous verbal expression and linguistic elaboration were still present 6 months after left hemisphere lesions in the studies of Alajouanine and Lhermitte (1965) and of Hecaen (1983). Gaidolfi and Vignolo (1980) report such reductions in 19% of a sample of 21 children with diffuse head injury followed up nearly 10 years later.

6.3.2 Echolalia

Echolalia may follow mutism in diffuse cerebral dysfunction, or may occur in association with large frontal lesions. Levin (1981) cites Stengel's differentiation of automatic and mitigated echolalia. The former is parrot-like, whereas in the latter there may be a questioning repetition of the words spoken by others, often with a change of personal pronoun. Mitigated echolalia may facilitate comprehension and a change from automatic to mitigated echolalia may be a sign of clinical improvement. Echolalia has been noted to occur in very severe head injury in association with other symptoms such as dementia, global aphasia or disinhibition (Thomsen & Skinhoj, 1976).

6.3.3 Dysarthria

Pathology in the motor speech system, manifested in articulation, respiration and voice difficulties, is common in severe head injury. Both Brink and co-workers in 1970 and Costeff and co-workers in 1985 reported 40% of their severely injured (rehabilitation centre) children to have dysarthria and unusually slow speech. In head-injured adults, these motor aspects of speech may show the most prolonged disturbance and still be present 10 to 15 years later (Najenson et al, 1978; Thomsen, 1984). Thomsen found dysarthria to occur in association with motor impairment and brain stem damage. Alajouanine and Lhermitte (1965) found dysarthria in 69% of their children with left hemisphere lesions. It occurred in association with severe hemiplegia. Hecaen (1983) found articulation difficulties in 52% of his sample with left hemisphere lesions, more common in those with a traumatic aetiology (66%), related to anterior (81%) rather than temporal (20%) lesions, and more frequent in those under 10 years. Coordination of articulation may also be affected by cerebellar pathology producing a characteristic disruption of the rhythm of speech called scanning (Rapin, 1982).

6.3.4 Anomia

Difficulty in word finding, particularly in confrontation naming, is the most commonly reported language disturbance in children and adults following closed head injury. It is related to the severity of injury sustained and may persist when other language disturbances have disappeared (Chadwick, Rutter, Shaffer & Shrout, 1981; Levin & Eisenberg, 1979a,b; Levin, Grossman & Kelly, 1976; Levin, Grossman, Sarwar & Meyers, 1981; Sarno, 1980, 1984).

Anomia occurs commonly in diffuse or bihemispheral conditions and as an end state of several aphasic syndromes, as well as specifically in association with lesions of the dominant angular gyrus (Benson & Geschwind, 1971; Black, Black & Droge, 1986; Kirshner, Casey, Kelly & Webb, 1987). Confrontation naming may be impaired by a failure in perception and recognition of an item, in elicitation of the name, or in activation of the motor articulatory sequence of the name (Goodglass, cited Kirshner et al, 1987).

Anomic errors after closed head injury have included semantic approximation (eg "snout" for tusk), circumlocution (eg "to make music" for the pedals of a piano) and concrete representation (eg "orange" for circle) (Levin, 1981).

Ewing-Cobbs and co-workers (1987) found confrontation naming to be significantly reduced in children and adolescents tested at about 1 month after moderate/severe head injury when they were compared with those sustaining mild head injury. However, the mean score of the moderate/severe group was within normal limits and only 12% had defective scores (ie below the sixth percentile on test norms). Van Dongen and Visch-Brink (1988) found neologisms to disappear rapidly in the first few days after mild head injury which produced aphasia, and all naming errors to have completely resolved by 6 months.

Considering children with severe head injury, the Chadwick-Rutter study (1981) found speed of picture naming to be significantly lower than that of matched controls at 1 year after injury, while Colam (1984) found reduced naming speed 3 to 15 years after injury. These findings may reflect a general reactive slowness rather than a language deficit, but Colam's subgroup whose PTA had lasted over 21 days was also defective in the number of pictures named.

Where there are focal left hemisphere lesions, naming may be strikingly impaired (Vargha-Khadem et al, 1985) and naming difficulties may persist for years, such that vocabulary limitations are mentioned in school reports (Hecaen, 1983).

6.3.5 Verbal Fluency

Word association fluency (generating words in categories or beginning with particular letters), has been found to be one of the most efficient language predictors of severity of injury in adults (Brooks et al, 1980; Levin et al, 1976). In the Chadwick-Rutter study (1981) verbal fluency (listing animal names) was impaired in the severely injured children at intake, but differences from the controls were no longer significant at 4 months. Ewing-Cobbs and co-workers (1987) found word fluency scores to be below the expected level at 1 month after mild as well as after moderate/severe head injury. Defective scores occurred in 8% of the mildly head injured and 30% of those with moderate/severe head injury. Winogron et al (1984) found significant differences in the expected direction between children with mild, moderate and severe head injury on a fluency test at approximately a year post-injury, while Colam (1984) found head-injured children's ability to generate words to be significantly lower at 3 to 15 years post-injury.

6.3.6 Perseveration

Thomsen (1975) reported that perseveration tended to persist along with word retrieval difficulties in patients who had been aphasic following severe diffuse closed head injury. Levin, Amparo and co-workers (1987) found that patients with frontal lesions visualized on MRI after minor or moderate head injury tended to make more frequent perseverative errors than the subgroup with temporal lobe lesions. Alajouanine and Lhermitte (1965) reported that perseveration was absent in 32 aphasic children with left hemisphere lesions.

Albert and Sandson (1986) differentiate three types of perseveration. Recurrent perseveration is the inappropriate occurrence of a previous response following an intervening production or subsequent stimulus. Continuous perseveration is the inappropriate prolongation or continuation of a behaviour. Stuck-in-set perseveration is the inappropriate maintenance of a category or framework. Albert and Sandson found perseveration on either confrontation naming or drawing to command to be more frequent in patients with left hemisphere lesions, than in patients with right hemisphere lesions or in the normal controls. Increased perseveration was found in those with greater word finding difficulty. Right hemisphere lesioned patients showed little perseveration, and then it was of the continuous rather than of the recurrent type.

6.3.7 Comprehension

Levin and Eisenberg (1979a,b) reported comprehension disturbances on the Token Test in 11% of children and adolescents in the early stages of recovery after closed head injury. Ewing-Cobbs et al (1987) found 8% of cases with mild head injury to have defective Token Test scores at about 1 month after injury, compared with 25% of cases with moderate/severe head injury. Mean scores for both groups were within the normal range and the groups did not differ

significantly from each other. Costeff and co-workers (1985) stated that many of their severely injured children showed speech defects in association with apparent deficits in semantic processing. These resolved in all but 1 child, but could still be found on testing. In Colam's local study (1984), head-injured children did not differ significantly from their matched controls on the Token Test at 3 to 15 years post-injury, although the differences on Part 5 approached significance, which may suggest some difficulty with more complex language comprehension or may reflect the greater demands made on short term auditory memory by this section of the test. Gaidolfi and Vignolo (1980) found from 5 to 10% of their sample to be impaired on the Token Test years after head injury.

Difficulties in sound and language processing have been suggested in studies of mild diffuse head injury. Gulbrandsen (1984) found that concussion contributed significantly to differences on the Category test and WISC Comprehension test, but not to errors on an aphasia test at 4 to 8 months post-injury. Klonoff et al, (1977) found their head-injured children to have significantly lower scores than the controls for 1 to 2 years post-injury on speech perception, sound recognition and the Category test. There was no measure of expressive language in the Klonoff study.

In adults with severe head injury, there is steady recovery of language functions within the first 3 to 9 months with visual and auditory comprehension preceding oral expression, reading and writing (Brooks & Aughton, 1979b; Groher, 1977; Najenson et al, 1978). In children with left hemisphere lesions, disturbances of comprehension occur in one third. Alajouanine and Lhermitte (1965) found such disturbances to be severe in only 12% of their sample, while Hecaen (1983) found the disturbances to occur exclusively in the early stages and the disturbances to be greater and more frequent with anterior than with temporal lesions. By 6

months, both studies report comprehension of spoken speech at near normal levels. Johnston and Mellits (1980) reported that, at about 10 months, recovery of language comprehension parallelled that of language expression in children recovering from coma, and the extent of recovery was associated with the duration of the coma.

6.3.8 Repetition

Repetition disorders receive very little mention in the literature on aphasia in children. Levin and Eisenberg (1979b) report disorders of repetition in 4% of their series of children and adolescents with closed head injury. Ewing-Cobbs and co-workers (1987) found about 12% of the mildly head injured and about 33% of the moderate/severe group to be impaired on sentence repetition at about 1 month after injury.

6.3.9 Reading, Writing and Scholastic Difficulties

Few of the paediatric head injury studies have looked at writing disorders, but in the left hemisphere lesion groups, these disorders are prominent and persistent, particularly in children under 10 (Alajouanine & Lhermitte, 1965; Hecaen, 1983). Ewing-Cobbs and colleagues (1987) found lower mean scores on writing to dictation and writing to copy in children with moderate/severe head injuries than in those with mild injuries. Scores of the mildly head injured were within the normal range, but about a third of the more severe group had defective scores at 1 month.

Chadwick and co-workers found two thirds of those with PTA over 3 weeks to have reading backwardness at the initial assessment and 44% to have such backwardness at 1 year (Chadwick, Rutter, Brown, Shaffer & Traub, 1981). A similar pattern was found for Arithmetic. Almost all of this subgroup were having difficulties at school or were placed in special school. The mildly head injured exhibited an extremely high rate of reading backwardness throughout (ie

about 44%), but as they showed no pattern of recovery, this poor performance was not attributed to the head injury.

The British study following up traumatic focal injury in children 2 or more years later, found a slight but consistent trend for scholastic difficulties (reading, arithmetic and spelling) to be associated with left hemisphere lesions (Chadwick, Rutter, Thompson & Shaffer, 1981). The prevalence of reading difficulties was high in this sample regardless of the side of injury and 55% of the children had a reading age at least 1 year behind their chronological age, while 33% were at least 2 years behind (Shaffer et al, 1980). Boys injured under the age of 8 who had been in coma over 3 days had a higher rate of reading backwardness than any other age-sex group.

Studies of children with left hemisphere lesions (eg Alajouanine & Lhermitte, 1965) or with aphasia following a history of brain injury (eg Cooper & Flowers, 1987) have reported scholastic difficulties persisting in almost all cases. None of Alajouanine and Lhermitte's cases made normal school progress in spite of relative recovery of language functions and average or low average intelligence (1965). Cooper and Flowers (1987) found arithmetic and spelling difficulties to be prominent and two thirds of their cases were receiving academic assistance.

6.4 Recovery

Studies of language recovery in children after head injury are lacking. In adults, most of the recovery after head injury appears to occur in the first 3 to 9 months, with recovery of visual and auditory comprehension preceeding recovery of oral expression, reading and writing (Brooks & Aughton, 1979b; Groher, 1977; Najenson et al, 1978). However, language deficits may persist beyond this period in one third of subjects with severe closed head injury or with focal missile injury (Levin, Grossman, Sarwar

& Meyers, 1981; Najenson et al, 1978; Newcombe, 1969). Thomsen (1975, 1984) and Sarno (1980, 1984) have described subclinical aphasic deficits persisting in adults years after severe closed head injury.

It is not known whether recovery of language functions after head injury occurs from intra- or interhemispheric reorganization. An evoked potential study (Papanicolaou, Levin & Eisenberg, 1984) has suggested that recovery in adults after left hemisphere damage which produced aphasia may be by means of a shift in the hemispheric mediation of language. The role of the right hemisphere in normal language development and in recovery of language after left hemispheric lesions is controversial (Benson, 1986). It appears that the right hemisphere can recognize (decode) many printed and spoken nouns, some verbs and some adjectives; that imageability is an important factor, that grammatical structure is not handled, and that the ability to produce (encode) language is extremely limited (Benson, 1986).

A recent examination of data from amobarbital, temporal lobectomy and dichotic listening studies has found that interhemispheric reorganization of speech is associated with left focal lesions which occurred before the age of 6, but that intrahemispheric speech reorganization is associated with lesions occurring at later ages (Satz, Strauss, Wada & Orsini, 1988).

6.5 Language in Socioeconomically Disadvantaged Children

The language of children from low-income backgrounds and educationally disadvantaged parents has been described as deficient because such children did not express abstract concepts, did not answer questions pointedly, assumed much information on the part of the listener and used more nonverbal communication than other children (studies cited Feagans, 1982). More recent studies have shown that

disadvantaged children learn all the uses of language, but differ from other children in the frequency with which they use complex structures and the purpose for which they use language (Farran, 1982; Tough, 1982). Tough (1982) found 3 year-olds from disadvantaged backgrounds to have shorter utterances and to use language less often to talk about past and future experiences, to give explanations or to reflect feelings than children from educationally advantaged backgrounds.

In a local study of a disadvantaged section of the Cape community, Molteno (1985) found that, whereas infant milestones were normal, language comprehension and expression were relatively delayed by 2.5 years. Verbal skills were correlated with parental education and family stability. Farran and Feagans have also found maternal education and family stability to act in combination to affect language development (Farran, 1982). The stresses of poverty leave little time or energy for playing interactive language games and reading books to children (Snow et al, 1982). Reading of books has been shown to stimulate the most complex speech (Tough, 1982). The work of Vygotsky (1962) has shown how using words helps stimulate the development of concepts that cannot be abstracted directly from concrete experience.

At kindergarten level, children from disadvantaged backgrounds are less able than other children to put what they know into words and are at a slight disadvantage in learning new material, particularly if it is presented verbally (Farran, 1982). A disadvantaged background affects verbal abilities more than spatial abilities, and older children are more affected than younger children (Rutter & Madge, 1976). Children from lower socioeconomic levels are less able to understand and use language to meet the demands of the classroom situation (Feagans, 1982) and are most at risk for failure on tasks of reading and counting (Rutter & Madge, 1976; Snow et al, 1982).

6.6 Speech and Language Deficits Expected in the UCT Survey

As the sample is disadvantaged, language scores below the test norms are expected in the control group. In the head-injured group, deficits relative to the controls in language production, eg naming and word fluency, are expected, and may be evident other than on specific measures. Difficulties in comprehension are expected to be found on testing, but not to be clinically evident. Measures of repetition are less likely to differentiate the head injured from the controls. Speech impairment, perseveration and echolalia are likely to be associated with very severe head injury.

7. VISUOMOTOR IMPAIRMENT FOLLOWING HEAD INJURY

7. VISUOMOTOR IMPAIRMENT FOLLOWING HEAD INJURY

Several studies have reported that impairment following head injury in children is likely to be most obvious on visual-motor and visual-spatial tasks, particularly if quick responses are required (Bawden et al, 1985; Chadwick-Rutter study, 1981; Dencker, 1960; Gulbrandsen, 1984; Levin & Eisenberg, 1979a).

Several abilities may be involved in performing these tasks, for example, motor skills, visual perception, spatial understanding, speed of response, ability to sustain attention, planning, integration and execution. Disruption of performance may be the result of focal lesions producing specific neuropsychological syndromes or may be attributable to nonspecific generalized disorders of attention and cognition, associated with diffuse injury (Auerbach, 1983; Stern & Stern, 1985).

7.1 Impairment on Motor Tests Following Head Injury

Impairment on tests requiring fine-motor coordination has commonly been found in the early stages following head injury on measures such as foot-tapping, finger-tapping, pegboard tests and mazes, and may be related to the severity of injury sustained (Chadwick-Rutter, 1981; Klonoff et al, 1977; Levin & Eisenberg, 1979a,b). Differences between head-injured and non-head-injured groups on motor tests may persist at 1 year and the Chadwick-Rutter study (1981) found manual dexterity on a pegboard task to be significantly poorer in their mild as well as in their severely injured group relative to the controls. The severely injured were still impaired on this task and on a copying task at the 2 year follow-up (Chadwick, Rutter Shaffer & Shrout, 1981). Klonoff and co-workers (1977), with a relatively mildly injured sample, found performance on finger-tapping to be impaired for 1 or 2 years post-injury, maze coordination for

up to 3 years post-injury, and Grooved Steadiness to be among the few tests still impaired at 5 years post-injury.

In a local head-injured group, performance on the Purdue pegboard was found to be impaired 3 to 15 years post-injury, particularly in those with PTA over 21 days (Colam, 1984). Differences between the head-injured and controls were nonsignificant on successive finger-tapping, although those with the longest PTA tended to have the lowest scores. In the studies from Ontario (Bawden et al, 1985; Winogron et al, 1984), it was also the more severely injured who had the lower scores and a higher frequency of impairment on motor tests at 1 year. On finger-tapping and the Purdue, only from 6 to 12% of the mild and moderately injured were two standard deviations below the mean, but almost half the severes were at this level. Of all the tests performed, the motor tests had the highest correlations with duration of altered consciousness (Winogron et al, 1984). No impairment on non-speeded motor tests such as grip strength, drawings and fine-motor steadiness, was found, however. (Bawden et al, 1985).

In terms of recovery rate, in the study by Klonoff and colleagues (1977), the number of visual-motor tests on which the head-injured differed significantly from the controls decreased each year. In the Chadwick-Rutter study, the severely injured showed greater improvement than the controls, on manual dexterity and the Continuous Performance Test, from the initial assessment to the 1 year follow-up (Chadwick, Rutter, Shaffer & Shrout, 1981).

7.2 Aetiology of Motor Disorders

Clumsiness and incoordination are very nonspecific complaints and may be the end result of many different motor and sensory disorders (Rapin, 1982). When deficits are mild, it is very difficult to know whether clumsiness of the hand or fingers denotes a corticospinal deficit, pathology

in the subcortical control circuits, or an apraxia or disorder of programming (Lezak, 1983; Rapin, 1982).

Brain damage, whether diffuse or unilateral, has been reported as impairing the performance of both hands on simple motor tasks such as pegboards and repetitive finger-tapping (Boll & Barth, 1981; Leonard, Milner & Jones, 1988; Lezak, 1983; Vaughan & Costa, 1962; Wyke, 1971). With unilateral lesions, the main deficit is in the finger movements of the contralateral hand, but ipsilateral deficits also occur and are more common after left hemisphere (LH) than right hemisphere (RH) lesions (Haaland, Harrington & Yeo, 1987; Trope, Fishman, Gur, Sassman & Gur, 1987).

Performance on complex motor tasks such as sequential movements, copying movements and bimanual coordination is disrupted in both hands by unilateral frontal lesions (eg the work of Luria, Kolb, Kimura, Milner & Jason, cited Jason, 1986; Roy, 1982). Again there is a tendency for impairment to be more marked after LH than RH damage (eg Leonard et al, 1988).

The supplementary motor areas, (thought to be concerned with automatic repetitive movements such as finger-tapping and overlearned speech sequences), have been considered as a possible source of ipsilateral control and to have, in conjunction with the ascending dopaminergic tracts, a crucial role in the activation and coordination of intentional movement (Meador, Watson, Bowers & Heilman, 1986).

The left hemisphere appears to be dominant for the learning and recall of skilled movements and to exert an influence over the ipsilateral as well as the contralateral hand. Apraxia, an inability to perform intentional movements that is not the result of sensory or motor deficits, has been far more frequently associated with LH

than RH lesions (see Geschwind & Galaburda, 1985, for a review of dominance, and Kirshner, 1986, and Roy, 1982, for reviews of apraxia). Performance can be disrupted by lesions along the pathway from the dominant parietal lobe to the premotor cortex and across the corpus callosum to the right premotor centre (Heilman, Rothi & Kertesz, 1983; Jason, 1986; Milner & Kolb, 1985; Wyke, 1971). Disruption of the basal-ganglia-thalamus-cortex loop as well as of cortico-cortical connections may produce apraxia (De Renzi, Faglioni, Scarpa & Crisi, 1986).

From the above findings, it is evident that either diffuse damage in head injury or specific focal lesions, (particularly of the dominant parietal lobe, either frontal region or the corpus callosum), may impair performance on tests of manual dexterity. Differentiation of groups with and without "brain damage" may be good on such tests, but individuals may be misclassified (Lezak, 1983). Poor performance on motor tests, while reflecting pathology in the nervous system, may have little relationship to the child's higher mental functions (Boll & Barth, 1981; Rapin, 1982).

There may be considerable intra-individual variability on fine-motor skills. Different types of activity (eg hand grip, finger-tapping, unimanual peg placement, bimanual coordination and the more perceptually based tactile skills) are relatively independent of each other (Spreen et al, 1984).

7.3 Developmental Issues

Motor dexterity increases with age throughout childhood. Depending on the particular skill assessed, there is a rapid increase between 4 and 8 years and a relative plateau from 8 or 10 years (eg on successive finger-taps; pegboard tasks). Initially, girls are superior to boys on these timed tasks and show earlier control of

overflow movements. In the majority of children, right-sided ability tends to be established first and initially to be markedly better than left-sided skill, but this marked asymmetry decreases by about age 8 (Connolly & Stratton, 1968; Denckla, 1973, 1974; Gardner & Broman, 1979; Wolff, Gunnoe & Cohen, 1983, 1985).

7.4 Correlates of Poor Motor Performance in Children

Motor performance, particularly in young children, is likely to be correlated with other cognitive functions. Wolff and co-workers (1985) have shown that developmental motor signs in kindergarten children (eg mirror movements, speed and rhythmicity of timed manoeuvres) may account for a significant proportion (15% to 20%) of the variance in reading in first grade. Bishop (1980) found that children clumsy on a motor tracing task, showed more cognitive impairment on IQ and reading scores. Boll (1971) found correlations between measures of motor speed and strength of grip and WISC verbal tests (not the performance tests) in both normal and brain-damaged children. Several studies by Boll and Reitan and co-workers have found performance on motor or sensorimotor tasks in normal and brain damaged children to be associated with level of performance on cognitive tasks (see Boll & Barth, 1981).

7.5 Impairment on Visuoperceptual and Visuoconstructional Tests Following Head Injury

Head-injured children have been reported as impaired on visual perceptual tasks (eg Picture Completion, figure and picture matching, colour naming, and the analysis of complex figures) which do not involve motor activities such as drawing or assembling. Klonoff et al (1977) found such difficulties to persist for 1 or 2 years in their sample with relatively mild head injury. The Chadwick-Rutter study (1981) found figure-matching accuracy was significantly lower in the mildly head-injured group as well as in the

severely head-injured group relative to the controls at 1 year post-injury.

Levin and Eisenberg (1979a,b) found about one third of children and adolescents to be impaired on a mixture of visuospatial and visuomotor tasks (Bender Gestalt drawings, construction of block designs and discrimination of photographs of faces) in the early stages of recovery after head injury. This impairment was related to the severity of injury sustained. Six months after mild concussion, Gulbrandsen (1984) did not find impairment on the Trail-Making Test, which requires scanning and tracking, as well as recognition of symbols, but Klonoff et al (1977) found lowered scores on this test to persist at 1 and 2 years post-injury in their mixed severity head-injured group.

On other drawing tests, Klonoff et al (1977) found reduced scores only at the initial assessment. Bawden et al (1985) found scores which were defective in the severely head injured approximately 1 year after head injury, but not significantly lower than those of the mildly head injured. However, defective performances on drawings tests persisting years after severe head injury have been reported: Colam (1984) found two thirds of his sample (and almost all of those with PTA over 3 weeks) to have scores below those of the controls on the Beery drawings and on drawing crosses in circles (Detroit Motor Speed and Precision) 3 to 15 years after head injury; while Baracchini-Muratorio et al (1985) found reduced scores on the Benton and the Rey figure copying 2 to 6 years after injury. It appears that impairment on visuospatial and visuomotor tasks persists, particularly in those with severe head injury.

7.6 Aetiology of Visuoperceptual and Visuoconstructional Disorders

Visual-perceptual tasks may be impaired by visual inattention or deficits in visual recognition, organization and scanning. Visuoconstructive tasks such as drawing and assembling require an appreciation of spatial relationships as well as visuoperceptual abilities and a certain level of motor development and organization (Benton, 1979; Lezak, 1983; Sattler, 1982).

Many of the functions involved in visuospatial tasks have been regarded as predominantly under RH control. Deficits in visual discrimination, figure-ground differentiation, visual synthesis, facial recognition, localization of points in space, judgement of direction and distance, and topographic orientation, have been predominantly associated with posterior lesions and have tended to be more frequent and severe following RH than LH lesions (Benton, 1979; Kertesz, 1983). Deficits on drawing and assembling tasks, have also been reported as more frequent and severe following RH than LH lesions, particularly of the parietal area (eg Heilman, Bowers, Valenstein & Watson, 1986; Mesulam, 1981; Villa, Gainotti & De Bonis, 1986).

Difficulties in spatial organization following RH lesions have been reported in young children as well as in adults, eg on drawings and block-play (Stiles-Davis, Janowsky, Engel & Nass, 1988), and on visual route-finding and geometric form recognition (Rudel, Teuber and Twitchell (1974). Selective impairment of the performance scale of the WISC has also been documented in children acquiring RH lesions after the age of 1 (McFie, 1961; Riva & Cazzaniga, 1986; Woods, 1980).

Evidence of the specialization of the RH for visuospatial processing and the direction of attention has come from studies of cerebral blood flow, oculomotor movements, reaction times, EEGs, and evoked potentials (eg Deutsch, Bourbon, Papanicolaou & Eisenberg, 1988; Kinsbourne & Hiscock, 1983; Mesulam, 1981; Sava, Liotti & Rizzolatti, 1988). Either there is a greater involvement of the RH than of the LH on visuospatial tasks, or the RH attends to visual and somatosensory stimuli from the ipsilateral as well as the contralateral hemispace, while the LH attends only to the contralateral hemispace (Davidoff, 1982; Heilman et al, 1986). The functional asymmetry of the RH for visuospatial processing, however, is not as marked as the LH advantage over the RH for language processing (Kertesz, 1983; Kimura, cited Mesulam, 1981).

Visuospatial and visuomotor tasks used in assessing brain dysfunction tend to be complex, to contain competing stimuli, and often to be speeded. Deficits on such tasks are frequent after brain damage, regardless of the site of lesion (Benton, 1979; Kertesz, 1983). Rapin (1982, p.29), has commented that tasks such as the Bender Gestalt and the Beery, widely used in screening for brain damage, owe their efficiency to the broad range of skills required and therefore to the wide range of cerebral systems they engage.

Studies have documented a high frequency of peripheral and central lesions in the visual system following closed head injury (Levin, 1985). Transient visual disturbances such as hemianopsia and cortical blindness have been reported in children following very minor head injury (eg Haas, Pineda & Lourie, 1975; Snoek et al, 1984). Hypersensitivity to light after minor head injury has been reported in young adults (Waddell & Gronwall, 1984). Spreen and co-workers (1984) have suggested that, developmentally, the visual system may be more susceptible to subtle brain dysfunction or a limitation of brain cell growth than other

parts of the nervous system, and that such susceptibility is reflected in visual-processing disturbances. Consistent with such an hypothesis are the findings by Gronwall and co-workers that pre-school children with minor head injuries have impairment of some pre-reading skills such as visual closure and visual memory, and have an abnormally high incidence of reading problems later (Gronwall, unpublished paper and letter July, 1987).

Performance in kindergarten on visual-perceptual processing and copying tasks has been shown to correlate with reading ability and with scores on other academic achievement tests (maths and spelling) in first and second grade (Fletcher & Satz, 1980; Melamed & Melamed, 1985). The frequency of visual-perceptual deficits after head injury may contribute to the high prevalence of scholastic difficulties found after head injury.

7.7 Speed and Complexity

It may be that visuomotor tasks are vulnerable to the effects of brain damage not because of deficits in visuospatial or motor skills, but because they present complex stimuli and may demand speeded responses.

7.7.1 Speed

Bawden et al (1985) pointed out that the severely head-injured children and adolescents in their study did not suffer a generalized deficit in motor or visual-spatial skills as there were no statistically significant differences between the groups on the non-speeded tests such as gripstrength, fine-motor steadiness, developmental drawings and the Target Test, or on some moderately-speeded tests such as Object Assembly and Picture Arrangement. The highly speeded tests, however, (such as Coding, pegboards and finger tapping) all showed significant differences. They considered the deficits might be attributable to

motoric slowing or slowed decision-making or altered response biases.

Chadwick and co-workers have also commented that it was performance on the nonverbal timed tests of hand-eye coordination that continued to show impairment, even after the effects of IQ were partialled out (Chadwick, Rutter, Shaffer & Shrout, 1981). However, many of the tests did not show any significant impairment on the timed component at 1 year; and there were no significant differences between the severely head-injured and their controls at 2.25 years on reaction time (RT) tests and the PASAT (Paced Auditory Serial Additions Test), both of which require rapid responses. In the Klonoff study (1977), inspection of the results suggests that significant differences between head-injured and control children on the visual-motor and visual-spatial tests were as likely to be on error scores as on the time scores. These findings do not suggest that speed alone was the cause of poor performance on these tasks. It should also be noted that in the Bawden et al study (1985) the scores of the severely head-injured group were nonsignificantly lower than those controls.

7.7.2 Complexity

Chadwick and co-workers specifically analysed their results to determine the effects of task complexity and duration upon performance (Chadwick, Rutter, Shaffer & Shrout, 1981). Findings were inconsistent with the view that head injury results in greater impairment on the more complicated tasks (eg Stern & Stern, 1985). Task duration also did not appear to have a significantly greater negative effect on the performance of the head injured than of the controls. (Studies of adults with head injuries have also been inconsistent with regard to finding increasing deficits with increasing task complexity; see MacFlynn, Montgomery, Fenton & Rutherford, 1984 for a summary of RT studies).

It would seem then, that it is probably the broad range of abilities engaged by these tasks plus their demands for attention to multiple aspects, planning and integration and sometimes speed, that make them sensitive to the effects of both focal and diffuse brain damage.

7.8 Developmental Issues

The process of postnatal visual development is viewed as a progressive encoding of increasingly complex aspects of the visual stimulus. Theories of perceptual development stress either an increasing ability to discriminate distinctive features in the environment or an increase in prototype matching. Gross (1985) suggests that these processes are probably complementary rather than antagonistic.

Visual attention may include several components (eg search, filtering and priming) which may mature at different rates. Search (the ability to scan a stimulus quickly and effectively) reaches near adult levels of efficiency by 7 to 8 years, whereas filtering (the ability to ignore irrelevant stimuli in the visual field) and priming (the ability to maintain or change a cognitive strategy) both show substantial improvement after 7 years and continue to improve throughout the elementary school years (eg Enns & Cameron, 1987).

Delayed maturation of attentional processess may characterize restless, hyperactive children who tend to be over-represented amongst children suffering mild head injuries (Brown et al, 1981; Theron, 1987). Studies have failed to show any basic visual or auditory perceptual defects in hyperactive children, or that there are filtering, discrimination, or concurrent processing problems, but the major disability has appeared to lie in sustaining attention and inhibiting impulsive responding

(Rutter, 1984; Spreen et al, 1984). These attention deficits may affect the development of cognitive abilities and lead to scholastic problems, consequences that would be difficult to disentangle from the effects of head injury.

There is a long developmental delay between the visual perception of figures and the ability to draw them. Drawing relies heavily on planning and motor programming (eg selection of a starting point, direction and speed of movement) and visual and kinaesthetic feedback are then necessary for the detection and correction of errors. It is possible on drawing tests to differentiate errors attributable to faulty perception from others due to faulty execution (Sattler, 1982), but scoring systems do not typically make such differentiation.

Males tend to outrank females in respect of the maturation and efficiency of visual-spatial processing, but their superiority may not be fully manifest until puberty (Geschwind & Galaburda, 1985; Rapin, 1982). On paper and pencil tests of spatial ability, Johnson and Meade (1987) found a reliable male advantage from age 10, with a smaller advantage evident earlier. However, differences in the drawing performance of boys and girls were nonsignificant on the Revised Beery and for most ages on the Bender Gestalt (Beery, 1982; Sattler, 1982).

Verbal encoding strategies may also come into play on visuomotor/visuospatial tasks (eg Coding has a loading on the verbal comprehension factor) and poor performance may be attributable to factors other than impaired spatial reasoning or deficient motor skills. Sattler (1982) has stated that, as well as neurological deficits, maturational delay, limited intelligence and unfamiliarity with the testing situation may impair performance. The next section considers the vulnerability of visuomotor tasks to disadvantage and deprivation.

7.9 Deprivation Issues

Amante, VanHouten, Grieve, Bader and Margules (1977) found that perceptual-motor deficits varied according to socioeconomic class level (SES) and ethnic group membership. There was a positive correlation between SES and the level of visual-motor development achieved on drawing tests (eg the Bender and Frostig). Considering IQ and other tests, they concluded that the prevalence of CNS damage or dysfunction in children, or its presumed correlates, was inversely correlated with SES and that there was a concentration of pathology in minority group populations. Marked differences in health status, in the quantity and quality of nutrition, prenatal and postnatal care, and perhaps of environmental stimuli, were cited as contributing to the gradient of neurological integrity.

Koppitz (cited Sattler, 1982) found social or cultural deprivation to affect Bender Gestalt scores and that ethnic minority children tended to score lower than white children. Beery (1982) has pointed out that such differences may be statistically significant in large samples, but that ethnic background has accounted for only about one percent of the variance. Brain-injured and low IQ children, however, do less well than normal children on the Beery drawings (Beery, 1982).

In a local study of 10 year-old coloured children, Freind (1973) found that middle and lower class children made significantly more errors on the Bender drawing test than did upper class children. Skolimowska (1978), however, found that when children with below normal school achievement were excluded, the Bender performance of local coloured children between 6 and 10.5 years was consistently similar to normative group data, at all ages and socioeconomic levels. Beery drawing scores were significantly lower, but were based on the pre-Revision

norms of the Beery in which ethnic background and income level factors were not as well controlled as in the Revised standardization (Breen, 1982).

7.10 Conclusions and Implications for Research

Disruption of many functions may contribute to poor performance on visuomotor and visuospatial tests and they are likely to be sensitive to brain dysfunction, whether focal or diffuse. Visuographic tests may be affected by socioeconomic disadvantage. Deficits on these tests may exist prior to head injury in a percentage of the sample and may be markers of scholastic difficulties.

8. MEMORY IMPAIRMENT FOLLOWING HEAD INJURY

8. MEMORY IMPAIRMENT FOLLOWING HEAD INJURY

Head injury may produce three types of memory impairment: post-traumatic amnesia (PTA), which includes the period of clouding and disorientation of consciousness and lasts until continuous day to day memory returns (Russell & Smith, 1961); retrograde amnesia (RA), which refers to loss of memory for events immediately preceding injury, is usually very brief, lasting only seconds or minutes, but which may, in very severe cases, cover months or years (Russell & Smith, 1961), and anterograde amnesia (AA), an ongoing memory deficit which effects new learning and recall. This chapter deals with this last type of memory deficit.

8.1 Memory Deficits in Children after Head Injury

8.1.1 The Nature of the Deficit

Systematic investigation of memory functions in children following head injury appears to be lacking in the published literature. However, studies that have included a memory test have consistently found impairment of memory functions in head-injured children (Baracchini-Muratorio et al, 1985; Chadwick, Rutter, Thompson & Shaffer, 1981; Colam, 1984; Fuld & Fisher, 1977; Gaidolfi & Vignolo, 1980; Levin & Eisenberg, 1979a,b; Levin, Eisenberg, Wigg & Kobayashi, 1982; Lewis, Satz, Light, Asarnow & Haist, 1987; Molodetskikh, 1987; Richardson, 1963).

Levin and Eisenberg and co-workers (1979a,b, 1982) found that memory was the neuropsychological function most frequently affected in the early stages of recovery from head injury. Nearly half of their total series of mild and severely injured children and adolescents had evidence of a deficit in verbal learning (selective reminding of word lists) and/or visual recognition (continuous recognition of designs). By comparison, only about one-quarter to one-third manifested a deficit on any of the other

neuropsychological functions assessed (ie language, visuospatial, somato-sensory and motor speed).

The deficit was related to the severity of injury sustained. Children and adolescents admitted in coma (GCS ≤8) had significantly lower scores than those with milder head injuries (GCS >8) (1982). The majority of those in the more severe groups (ie those who had experienced coma, whether less than or more than 24 hours) had clearly defective memory functions (ie scores that were two standard deviations or more below the norms for the test) (Levin & Eisenberg, 1979a,b). Analysis of the consistent retrieval (CR) scores on word lists suggested a deficit even in the mildly head-injured adolescents when they were compared with a normal group (1979a). Memory could be impaired in the absence of a generalized cognitive deficit, a finding supported by Gaidolfi and Vignolo (1980) and Baracchini-Muratorio and co-workers (1985), who reported half the children with memory deficits to have intelligence in the average range.

There may be material-specific deficits following head injury, related to the site of the lesion. Levin and Eisenberg (1979b) found a relationship between a LH mass and defective retrieval of verbal material in 6 out of 7 patients with such lesions. Chadwick and co-workers in their study of focal head injury found that children with LH lateralized lesions took more trials to criterion on a word-learning test and that those with RH lesions had fewer items correct on the first trial of a faces-learning test (Chadwick, Rutter, Thompson & Shaffer, 1981). Molodetskikh (1987) found more of the children with severe head injuries (58%) than of those with moderate head injuries (28%) to have disturbance on the Luria verbal memory tests in the acute phases after head injury, but also that children with left hemisphere lesions had lower scores.

8.1.2 Recovery and Persistent Impairment

In the study by Klonoff and colleagues (1977) 10% of the children have complaints of poor memory and/or concentration at the 5 year follow-up, but unfortunately their battery included no test of memory. Levin, Eisenberg and co-workers (1982) showed that a memory deficit on selective reminding and continuous recognition tests persisted at follow-up when severely head-injured children were compared with mildly head-injured children. However, little can be inferred about the process of recovery as intervals between injury and assessment vary vastly for the children included (0.2 to 52 months). It appears that about 6 out of 15 in the severely head-injured sample (40%) had scores on selective reminding that were below the second percentile of locally collected norms, both at the baseline and follow-up assessment. More than half of those to whom the continuous recognition test was readministered, had impaired scores at follow-up.

In the adolescent sample, the severely injured showed some improvement from baseline to follow-up, but their scores were still lower than those of the more mildly injured at follow-up. A major flaw in this study is that the more mildly head-injured children and adolescents were not retested if their baseline scores were within the normal range, but their baseline scores were re-entered in the follow-up analysis. There is no comment on the performance of the mildly head injured relative to the normative group.

Lewis and co-workers (1987) have reported deficits in free recall of a word list persisting at 1 year after mild head injury in children whose performance on intelligence, vocabulary, pictorial and spatial memory tests was similar to that of matched controls. In this study, children with severe head injury performed significantly worse than the control group on all measures of intelligence and memory.

Other studies have also reported memory deficits in children, often persisting years after head injury. In the study by Baracchini-Muratorio and co-workers (1985), 8 out of 11 severely injured children (73%) had memory defects on visual retention tests at follow-up from 2 to 5 years later. Gaidolfi and Vignolo (1980) found 5 out of 21 (24%) of patients sustaining head injury during school years had defective memory in terms of the number of trials needed to learn a word list when assessed at least 7 years after injury.

Colam (1984), using selective reminding of the shopping lists devised for the present study, found only nonsignificant differences between head-injured and control children tested some 3 to 15 years post-injury, but the lowest scores were found in the group with PTA 3 weeks and longer.

The Chadwick-Rutter group (1981) found differences to be no longer statistically significant between their severely head-injured group and the controls on a paired-associate learning task at both 4 months and 1 year post-injury, although the head injured had a trend to higher error scores on both learning and delayed recall.

8.1.3 Summary

In the literature on head injury in children, a memory deficit appears to be one of the most frequent consequences of head injury, occurring even in mild cases. The deficit with closed head injury appears to be generalized, affecting both verbal and visual retention, with some indication of a material-specific deficit at least in relation to left temporal injury. Patterns of recovery have not been established. The mild and moderately injured are evidently likely to be within normal limits, but it is not clear at what time interval after injury this may be expected, and, in the studies cited, they have not been contrasted with

retested controls. The more severely head injured show improvement in that scores are not necessarily significantly lower than those of controls, but a proportion, varying from one quarter to three quarters, evidently have memory deficits persisting years after injury.

A better understanding of the nature of the memory deficit following head injury, the patterns of recovery, and the predictors of persistent deficit, was sought in the adult head-injury literature. Issues are highlighted below by reference to well known studies and a comprehensive literature search has not been undertaken.

8.2 Memory Deficits in Adults after Head Injury

8.2.1 The Nature of the Deficit

Memory deficits occurring in adults after head injury appear to be generalized, covering both verbal and visual retention (eg Brooks, 1972, 1974, 1976; Brooks & Aughton, 1979a,b; Brooks et al, 1980; Groher, 1977; Gronwall & Wrightson, 1981; Levin, Mattis et al, 1987; Sunderland, Harris & Baddeley, 1983).

Short-term memory (STM), the span for immediate recall, tends to be intact following head injury in adults (see Levin, Benton & Grossman, 1982 for review of studies), except in the early stages when the patient is still in PTA (Mandleberg, 1975) or when there is severe head injury producing protracted coma or oculovestibular defects (Levin, Grossman & Kelly, 1976). This fits with the theory that STM is impaired where there is generalized cognitive impairment, but not where there is a specific memory deficit without such generalized impairment (Kopelman, 1987).

It is not clear whether the long-term memory deficit is in encoding, storage, retrieval or all three processes. Richardson and colleagues have attributed the memory deficit in closed head injury to a selective impairment in the use

of mental imagery as a form of elaborative encoding (Richardson, 1979; Richardson & Barry, 1985; Richardson & Snape, 1984). In studies based primarily on minor head injuries, they have shown a deficit in the learning of concrete but not abstract words. This deficit could be eliminated with the use of imagery instructions and did not occur in the recognition of faces or pictured objects. However, even in their own study (Richardson & Barry, 1985) there was a trend for the head injured to be worse than the controls in the recognition of faces, and a recent study (Light & Lewis, 1988) has not supported their contention that memory deficit after severe head injury is limited to the recall of concrete words. A problem in reaching conclusions about the relative impairment of verbal and visual memory is that the tests used have not been equated for difficulty (Strauss & Allred, 1987).

The failure of the head injured to use spontaneously strategies which would aid recall has been highlighted in studies by Crosson, Novack, Trenerry and Craig (1988) and by Levin and Goldstein (1986). It was found that the head injured differed from controls in their use of clustering and subjective organization. Levin and Goldstein comment on the head injured's "passive approach" to learning. These findings and those of Richardson and colleagues are reminiscent of what has been said of memory deficits in patients with frontal lobe lesions (see Aetiology below).

A deficit in the LTM storage of head-injured patients has been suggested by Brooks (1975) and by other studies which have found adequate immediate recall, but a deficit in delayed recall (eg Stuss et al, 1985; Dikmen et al, 1986). Using the selective reminding technique, Levin and co-workers (Levin, Benton & Grossman, 1982) and Gronwall and Wrightson (1981) have shown deficits in the amount of verbal material in long term storage and in the consistent retrieval of such material by head-injured patients.

In terms of intrusion errors, the head injured have been reported to have a higher frequency of extra-list intrusions (confabulations) than controls (eg Brooks, 1975; Levin & Goldstein, 1986), with a particular increase in the severely disabled (Levin, Grossman, Rose & Teasdale, 1979). The position with regard to intrusions from previously learned lists (proactive interference) is not clear as Brooks (1975) found the head injured had fewer such intrusions than the controls and regarded this as indicating poor storage, whereas Parker and Serrats (1976), found that the severely head injured tended to perseverate 2 or 3 words from previous lists.

Gronwall and Wrightson (1981, p.894), have suggested that closed head injury has at least three different effects on memory:

1. there may be a deficit in information-processing ability which could affect performance on memory tasks when complex processing is required or when time constraints are imposed;

2. there is a deficit in the ability to place material in long-term storage, which appears to be related to PTA duration;

3. there is a deficit in the ability to retrieve material from memory once it has been stored. An analysis of their own data, plus that of Levin, Grossman, Rose and Teasdale (1979), suggested that, when low IQ cases have been excluded, about one-quarter of head-injured cases still have a retrieval deficit, regardless of severity of injury.

Head injury may also impair memory for remote events in adults (Levin et al, 1985). Remote memory in children could be indicated by performance on general knowledge tests (eg Information on the WISC-R), but it would be difficult to ascertain whether the child had ever stored the information (Hemp, 1986).

The position of Richardson and colleagues that there is no specific impairment of memory following head injury and that deficits not attributable to a disruption in the use of mental imagery may be attributable to broader cognitive deficits (Richardson & Barry, 1985), seems untenable. Memory defects may be more frequent or severe in their occurrence than other neuropsychological deficits, may occur in the absence of other changes and where intellectual functions are within the normal range (eg Fodor, 1972; Levin, Amparo et al, 1987; Levin & Eisenberg, 1979b; Levin, Goldstein, High & Eisenberg, 1988; Roberts, 1979; Stuss et al, 1985; Uzzell et al, 1987), and, as is detailed in the section below, memory functions may lag behind other neuropsychological functions in their rate of recovery.

8.2.2 Focal Effects

A material-specific deficit has been demonstrated in adult closed head injury patients in that a left temporal mass has been associated with impairment of verbal learning on the selective reminding technique, whereas no such association was shown for a left nontemporal mass or a right hemisphere mass (Levin, Benton & Grossman, 1982). Presence or locus of a mass lesion was unrelated to long term recovery in this study. Brooks et al (1980) similarly reported that the side of the haematoma was irrelevant to cognitive outcome. Smith (1974) studied patients many years after head injury and found both verbal and visual memory to be more impaired when impact had been on the right side of the head. As site of impact may have little to do with the pattern of brain lesions sustained, interpretation of this finding as reflecting left-sided brain damage seems suspect and highlights the problem of relating deficits in closed head injury to focal lesions when there is no control for concomitant diffuse effects (Chadwick, Rutter, Thompson & Shaffer, 1981; Levin, Benton & Grossman, 1982).

Qualitative analysis of small samples of closed head injury patients with minor or moderate head injury (ie no coma) suggested that those with temporal lesions on MRI (n=4) had disproportionate impairment of both verbal and visual memory functions, in contrast to those with frontal lesions (n=4) who in turn had slightly greater impairment of executive functions (Levin, Amparo et al, 1987). However, confirmation of these trends must await the investigation of larger numbers of patients.

8.2.3 Prediction of Memory Deficits

Studies have consistently shown a relationship between PTA duration and the severity of memory deficit in the adult head injured. Those with longer PTA have either had lower memory test scores (eg Brooks, 1976; Brooks & Aughton, 1979a; Brooks et al, 1980; Gronwall & Wrightson, 1981; McLean et al, 1983; Richardson & Snape, 1984) (although these differences have not always reached statistical significance), or they have had a slower rate or lesser extent of recovery and a higher percentage with persisting impairment (eg Brooks & Aughton, 1979b; Parker & Serrats, 1976; Russell & Smith, 1961). The nature of errors or quality of learning may also be associated with the length of disorientation (Parker & Serrats, 1976).

Where PTA has not been reported as associated with the extent of memory deficit, there has been a tendency for the PTA subgroups to have been very small (eg Smith, 1974; Stuss et al, 1985) or only a limited range of PTA has been included (eg Bornstein et al, 1989). In contrast to PTA, subdivisions for duration of coma have not been found to be related to the extent of memory deficits in some studies (eg Brooks & Aughton, 1979b; Parker & Serrats, 1976). However, when groups have been simply dichotomized, either into those with or without coma (eg the studies of Levin and colleagues, see Levin, Benton & Grossman, 1982) or into those with coma lasting more or less than 6 hours (Vilkki,

Poropudas & Servo, 1988), or 2 weeks (Lezak, 1979), then those with the longer coma have had the worse performance. The severity of diffuse brain injury as reflected by oculovestibular deficit, low scores on GCS and residual cerebral atrophy on CT scan has been found to be related to persistent memory deficit (Cullum & Bigler, 1986; Levin, Benton & Grossman, 1982). As mentioned above, lateralized focal lesions have not been related to persistent memory deficits (eg Brooks et al, 1980; Levin, Benton & Grossman, 1982), although Thomsen (1977) found that in a severely head-injured group, aphasics had worse verbal learning than nonaphasics.

8.2.4 Recovery and Persistent Impairment

8.2.4.1 Mild Head Injury

Even mildly head-injured adults have been found to have a deficit in new learning and recall which appears to recover within the first few months (Dikmen et al, 1986; Levin, Amparo et al, 1987; Levin, Mattis et al, 1987; McLean et al, 1983; Parker & Serrats, 1976; Ruesch, 1944). They have been shown to be either "within normal limits" on test norms (eg Levin, Benton & Grossman, 1982, p.112) or to have scores that are nonsignificantly lower than those of controls (Levin, Amparo et al, 1987; Levin, Mattis et al, 1987). Full recovery has not been definitely established, however, as they have not been contrasted directly with retested controls.

A percentage may well not be within normal limits by the 3 month assessment, as was the case for 4 out of 9 patients with moderate head injury in the study by Levin, Amparo et al (1987). Parker and Serrats (1976) found only 71% of those with post-traumatic disorientation (PTD) lasting less than 24 hours to have a normal shaped learning curve by 3 months, 82% by 6 months, and 92% from the 1 year assessment onwards.

8.2.4.2 Severe Head Injury

For the more severely injured, the major improvement also appears to have occurred in the early months post-injury, but a larger percentage of these groups than of the more mildly injured have continued to show impairment of memory functions at one year and later (eg Brooks & Aughton, 1979b; Groher, 1977; Levin, Benton & Grossman, 1982; Lezak, 1979; Parker & Serrats, 1976; Uzzell et al, 1987).

Groher (1977) found the changes between the first and second assessments (at approximately 1 and 2 months post-injury) to be significant. Thereafter there was a general trend to continued improvement, but if memory functions were going to recover after 4 months, it appeared that they would have done so by 2 months. Lezak (1979) found that the greatest changes in the percentage within normal limits occurred between the first and second assessments which took place approximately within the first and second halves of the first year post-injury.

In the Parker and Serrats study (1976), which was a selected sample of those with early memory problems on story recall, both the rate and the extent of recovery were related to the length of PTD. In the groups with the longest PTD, fewer patients achieved normal learning curves and they took longer to do so. The nature of the learning failure also showed a change over time. The percentage of those able to recall only the last items on the list ("demented" learning) declined rapidly in the shorter PTD groups, but 50 to 60% of those with PTD over 1 month continued to show such learning curves from the initial to the 2 year follow-up.

It should be noted that patterns of recovery over time may be very variable and Brooks and Aughton (1979b) found that some patients showed continued improvement over all test occasions, others showed no change and the test scores

of some dropped. A similar variability for the Lezak (1979) and Parker and Serrats (1976) studies can be assumed as the percentage showing normal performance decreased in some groups at some points.

These studies appear to show that, with some exceptions, recovery has stopped or slowed by about 1 year after injury (Brooks et al, 1984). Memory may recover at a slower rate than other functions and Brooks and Aughton (1979b), with a mixed severity sample, found that tests of memory, but not those of language and intelligence, continued to show significant differences between the head injured and the controls at 6 months and 12 months. Studies detailing the persistence of memory deficits in the more severely injured years after head injury include those of Lezak, 1979; Smith, 1974; Sunderland et al, 1983 and Thomsen, 1984. The study by Sunderland and colleagues shows that there is a poor correspondence between subjective reports and objective testing of memory deficits.

8.2.5 Conclusions

It appears from the studies of adults, that the mildly head-injured show transient impairment of memory function with the majority improving to within normal limits by 3 months post-injury. In the more severely injured, recovery also tends to be most striking in the early months, but improvement continues over the first year with few significant changes thereafter. Depending on the severity of injury, from one to three quarters have persistent memory deficits. Memory functions recover more slowly and deficits may persist more frequently than is the case for other neuropsychological functions.

8.3 Aetiology of Memory Deficits

Memory defects occurring after head injury may be part of a general process of cognitive impairment, or they may be related to specific lesions disrupting memory functions.

Lesions which have been found to impair memory functions specifically, have involved the diencephalon, the medial temporal structures and the basal forebrain. The nature of the memory deficit produced may depend upon the site of the lesion and the particular connections disrupted (see Parkin, 1984; Mayes, 1986).

In closed head injury, Jennett (1969) has argued for the relationship of the memory deficit to temporal lobe damage or dysfunction. Lesions of the medial temporal structure predominantly affect the acquisition and retrieval of new learning. Walsh (1985) has suggested that memory deficits after head injury are part of a general failure of adaptive behaviour and are related to frontal lobe dysfunction. Frontal lobe dysfunction impairs memory, because there is a failure to organize material adequately to facilitate encoding and retrieval (Luria, 1973; Mayes, 1986). In addition, patients with frontal lobe lesions have been found to have a deficit for the temporal aspects of contextual memory, a sensitivity to proactive interference, a tendency to confabulate and a failure to censure errors.

The vulnerability of the frontal and temporal lobes in closed head injury has been established by radiological, neuropathological and experimental animal studies (Adams et al, 1985; Graham et al, 1978, 1989). Even in mild head injury, frontal and temporal lesions have been amongst the most common found on MRI and may resolve within 1 to 3 months, with a parallel improvement on tests of executive and memory functions (Levin, Amparo et al, 1987).

No selective deficit has been established as underlying the memory problems following closed head injury. It is possible that memory functions are poor after head injury as a consequence of temporal lobe dysfunction affecting primary learning and recall, or as a consequence of frontal lobe dysfunction affecting strategies for encoding and retrieval. It seems likely that narrow localization is inappropriate

and that there is widespread disruption of the neuronal networks involved in performance on memory tasks.

8.4 Developmental Issues

The improvement of memory functions in children with age is thought to come from increasing general knowledge about the semantic relationship between concepts and an increasing awareness of memory strategies and the need to use them (Ornstein & Corsale, 1979; Pressley, Heisel, McCormick & Nakamura, 1982). Rehearsal only develops at about 7 years and chunking of items and clustering into categories do not occur spontaneously until about 9 or 10 years (Kail, 1979). Spontaneous use of imagery strategies is not acquired until adolescence, when the use of elaborative strategies increases dramatically (Pressley et al, 1982). Primary school children may initially be guided by automatic associations rather than by deliberate grouping strategies (Schneider, 1986).

What is said about the use of memory strategies in the literature on children is very reminiscent of what has been said about frontal lobe patients (see previous section) and closed head injury patients (eg Richardson & Barry, 1985). Children can be trained to use strategies that will enhance recall to adult levels, but they do not spontaneously use such strategies and the training tends not to generalize from one task or situation to another (Gross, 1985; Pressley et al, 1982).

In considering "deficit populations", such as the retarded children and the learning disabled, Pressley et al have reported that they are less likely than normal children to use efficient strategies; the retarded children never thinking to do so and the learning disabled having a lag in strategy development (p.138, 1982). Head-injured children may well constitute another such deficit population, able to

execute strategies, but unlikely to do so spontaneously, or to generalize rehabilitation training.

8.5 Deprivation Issues

In studies of verbal memory in head-injured adults, it has been found that sociocultural factors may influence performance. Richardson (1979) found that SES accounted for a significant amount of the variance in the recall of abstract and concrete nouns following head injury. Levin, Mattis and co-workers (1987) found that controls in the Bronx performed at the same level on a verbal memory test as the patients with minor head injury at two other centres and would have been regarded as neuropsychologically impaired if test norms had been used.

Some researchers have argued that memory tests should be independent of intelligence, but Mayes (1986) has said that this may not be feasible as intelligence test performance may depend upon various types of memory, and greater intelligence should be associated with more effective coding and retrieval of semantic information. Children of working class parents tend to have IQs slightly below the mean for total population sample (Sattler, 1982). However, research in Groote Schuur Hospital found that memory test performance was not correlated with IQ in the multicultural, predominantly lower socioeconomic population served by the hospital (Hemp, 1977).

8.6 Memory Deficits Expected in the UCT Survey

The selective reminding technique of Buschke (1973, 1974) will be used to test learning of a shopping list. Deficits in Recall, Long Term Storage and Consistent Retrieval are expected in the head injured. Interference from confabulated items may be excessive, but it is not clear whether interference at the later test sessions from items on previously learned lists is to be expected or not.

9. METHOD

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9. METHOD

9.1 Head-Injured Subjects

The 123 children who are the subjects of the present study are a subgroup of the 1134 children under 14 years consecutively admitted to trauma units at Red Cross and Groote Schuur Hospitals between mid-June 1983 and mid-June 1985 for observation following head injury.

9.1.1 Selection Criteria

Inclusion criteria for the present study are summarized in Table 2.

Table 2
Inclusion Criteria

	Severity Criteria		Additional Criteria
	Any of the following:		
1.	Loss of consciousness lasting longer than 5 minutes.	1.	Age between 6 and 14 years.
2.	Post-traumatic amnesia (PTA) lasting 1 hour or longer.	2.	English or Afrikaans speaking.
3.	Glasgow Coma Scale (GCS) of 12 or less.	3.	No history of mental retardation.
4.	Persistent drowsiness.	4.	No history of head injury or cerebral
5.	Focal neurological signs.		disease of significance.
6.	Intracranial haematoma.	5.	Out of PTA by 6 months.
7.	Compound depressed fracture.	6.	Willing to be tested.
8.	Basal skull fracture.		
9.	Seizure.		

The severity criteria presented in Table 2 are those of the UCT Child Head Injuries Survey and were intended to ensure that children with more than a very brief disturbance of consciousness, with any neurological involvement or who had received a severe blow, were followed up. Only 388 of

the children admitted for observation met any of these criteria and 39 of these died.

The additional age and language criteria were imposed so that children could be assessed on a uniform neuropsychological test battery. The 186 children under 6 years and a further 30 Xhosa-speaking children over 6 years are the subjects of separate studies. Children with a definite history of mental retardation (n=3) or with cerebral pathology of significance (n=1) were excluded so that the effects of acute head injury would not be obscured by previously existing conditions. Three children meeting the other inclusion criteria were still in PTA at 6 months: 1 remained in coma (PVS), while the 2 who emerged from coma had not recovered to a testable level within the time course of the study. A further 3 children, all with mild head injuries, failed to keep their testing appointments. The remaining 123 children were divided into severity categories according to their length of PTA.

9.1.2 Determination of PTA length

PTA was categorised as none, less than 1 hour, 1 to 24 hours, or the number of days was specified. PTA length was determined by the nursing sisters of the head-injury team who visited the child as soon after the accident as possible and then daily until discharge. Where necessary, the notes of the psychologists who also visited the children to determine readiness for testing were consulted, or details were sought from parents. Children were questioned to determine orientation for person, place, and time, but more emphasis was placed on eliciting evidence of continuous memory.

9.1.2.1 Orientation Data

In this disadvantaged sample, many children clearly not in a confusional state could not give basic personal details such as age, birthdate, or address. They often did not know the name of their school, their teacher, or what standard they were in. Many knew their parents worked, but not where they went or what work they did. They could name brothers and sisters, but, the younger children particularly, could not give ages. In terms of place, some had no concept of a "hospital", but it could be established that they knew they were not at home. In terms of time, many of the children did not know the days of the week or the months of the year and few knew the time of day. Some of the younger children did not know concepts such as "yesterday" and "tomorrow", or "morning" and "afternoon".

Children who could give accurate orientation data plus some account of what had happened to them were rated as out of PTA. Children who could not give orientation data, but showed evidence of continuous memory, were also rated as out of PTA.

9.1.2.2 Evidence of New Memories

For children with PTA less than 1 day, the establishment of new memories could be determined by discussion of the journey to hospital, Trauma Unit, transfer to the wards, trips to x-rays, theatre and so forth. For the children with longer PTA, details of treatments, visitors, meals, ward activities, and what they had been told about the accident, could be elicited. Accuracy of information given was checked with parents and ward staff and with police and ambulance records. Where a child was vague after a clear memory (eg loading into the ambulance) this memory was regarded as an island and not the end of PTA.

The head-injury sisters were dressed in casual clothes and were special visitors to the head-injured children in the wards. They planted tokens such as sweets and crayons and engaged in activities about which questions could be asked the next day. Children discharged from hospital still in PTA were brought back at weekly or two weekly intervals and tested when the parents felt that the confusion had cleared and the child was able to give an account of recent events.

Discrepancies in PTA estimates between the nursing sisters and the psychologists were few and were resolved by study of the detailed notes recorded. In one case parents had given conflicting stories of the day of the accident in order to cover up an assault (cf Bruce, 1984), and in another memories had clearly been laid down inside the estimated PTA and the child's confusion was attributable to Diphenylhydantonin toxicity.

9.1.3 PTA Severity Groups

The children were grouped into 3 severity categories based solely on PTA length: the moderates had PTA less than 24 hours, the severes had PTA of 1 to 7 days, and the very severes had PTA over 7 days (Jennett & Teasdale, 1981, p.90). This classification resulted in a total of 55 moderates, 40 severes, and 28 very severes. The distribution of PTA length within the severity groups is summarized in Table 3.

Table 3

PTA Length Within the Severity Groups

Group	PTA Length	n
Moderates	None	12
	Less than 1 hour	3
	1 to 24 hours	40
Severes	1 to 2 days	17
	2 to 7 days	23
Very Severes	1 to 2 weeks	7
	2 to 3 weeks	8
	>3 weeks	13

The children in Table 3 with no PTA or PTA less than one hour are those admitted to the study on neurological and/or radiological grounds (severity criteria 5 to 9 in Table 2) and are grouped with those with a moderate PTA. In the very severe group, PTA ranged from 8 to 65 days.

9.1.4 Accident Details (see Appendix A1, p.430).

The relationship between severity of injury and cause of injury is illustrated in Figure 3, where it can be seen that, the more severe the injury in terms of PTA length, the more likely that the accident was traffic related. MVA in Figure 3 refers to a motor vehicle accident, pedestrian (PED.) or other (passenger, cyclist, fall from a moving vehicle).

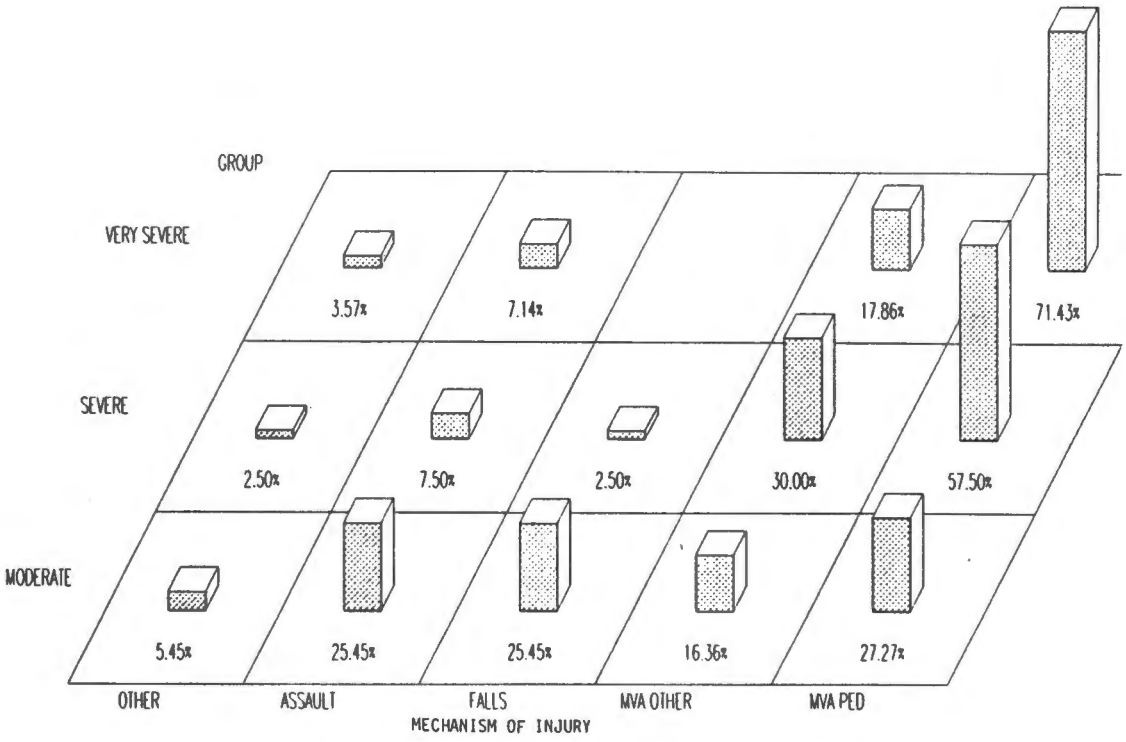


Figure 3. Mechanism of Injury

Very severes: As is shown in Figure 3, the majority of the children in this group had traffic-related accidents: 20 were pedestrians and 5 were passengers. The accidents tended to occur at high speed with acceleration-deceleration, with or without direct impact to the head. Of the 3 children not involved in traffic accidents, 1 was kicked by a horse and 2 had open head injuries (one was intentionally hit with a spade and the other was accidentally shot by a rifle). This gunshot wound was not excluded from the study as M.K. was unconscious for 2 weeks and her neuropsychological picture was very similar to that of the children with nonmissile head injury.

Severes: In this group, 45 out of the 55 children had traffic related accidents: 23 were pedestrians, 5 were passengers, 5 were cyclists, and 2 fell from moving vehicles. Three of the children were assaulted: one was struck by a brick, one by a fist, and one by a hammer.

The child who fell, dropped about 7 metres from a second-storey balcony and the remaining child was knocked on the head in a rugby game.

Moderates: This group sustained a far more varied range of accidents than the more severely injured children (see Figure 3). Less than half the accidents were traffic related: 15 were pedestrians, 5 fell from moving vehicles, 3 were passengers and 1 was a cyclist. The pedestrians were usually hit by slow moving vehicles. Falls and assaults were equally represented in this group with 14 of each. The falls were from beds, playground equipment, stairways and skateboards, but also occurred on the level with heads striking the ground or walls.

Of the children who were assaulted, 4 were hit by bricks and bottles aimed at someone else, while the remaining 10 were directly assaulted with a hammer, a spade, stones or bricks, and blows or kicks. In 6 cases these intentional assaults were perpetrated by other children.

There are 3 children in the other category: 1 was struck by the crank handle of a truck, 1 by a golf club and 1 by a knee in a rugby game.

9.1.5 Clinical Details (see Appendix A1, p.430).

9.1.5.1 Glasgow Coma Scale (GCS) Scores

Glasgow Coma Scale scores (see Table 1, p.41) were determined after admission to Trauma Unit. Wherever possible, the score assigned by the neurosurgeon or the head injuries nursing sister was recorded, as this assessment tended to be after stabilization by the admitting doctor. In the case of late admission from other centres, the description of the previous medical attendant was used to

determine the GCS score. The relationship of GCS scores on admission to PTA length is shown in Figure 4.

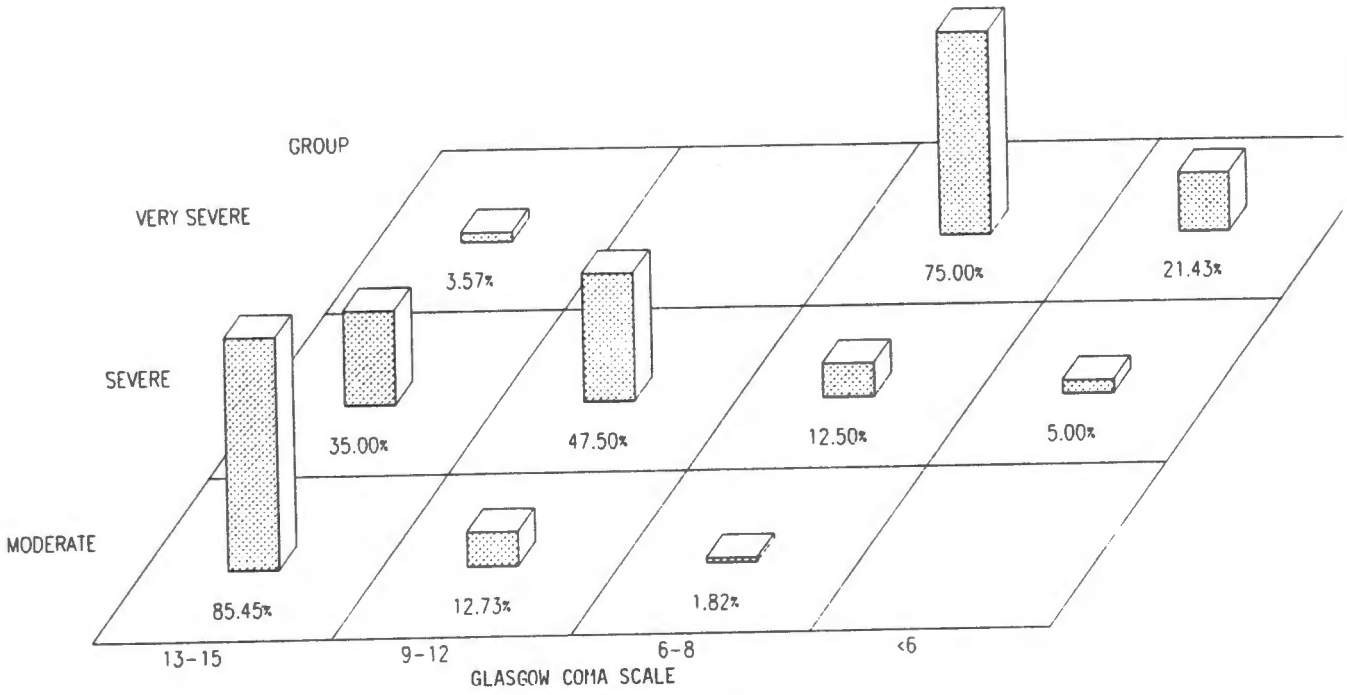


Figure 4. GCS Scores and PTA Length

As can be seen in Figure 4, there is a good correspondence between level of consciousness on admission and severity classification based on the length of PTA. All the very severes, except for C.P. who was a late admission whose earlier length and depth of unconsciousness was unknown, had GCS scores of 8 or below. All the moderates, except 1 child who had convulsions, had GCS scores above 8. In the severe group, the children admitted with scores of 8 or less tended to be hypovolaemic or to have had seizures.

9.1.5.2 Duration of Coma

For practical purposes, coma was taken as a Glasgow Coma Scale score of 8 or less as this corresponds to the International Data Bank practice (Jennett & Teasdale, 1981) and is also the cutoff point for severe head injuries in the studies of Levin and Eisenberg (1979a,b). Figure 5 illustrates the relationship in the present sample between the length of unconsciousness and the severity of injury classified according to PTA length.

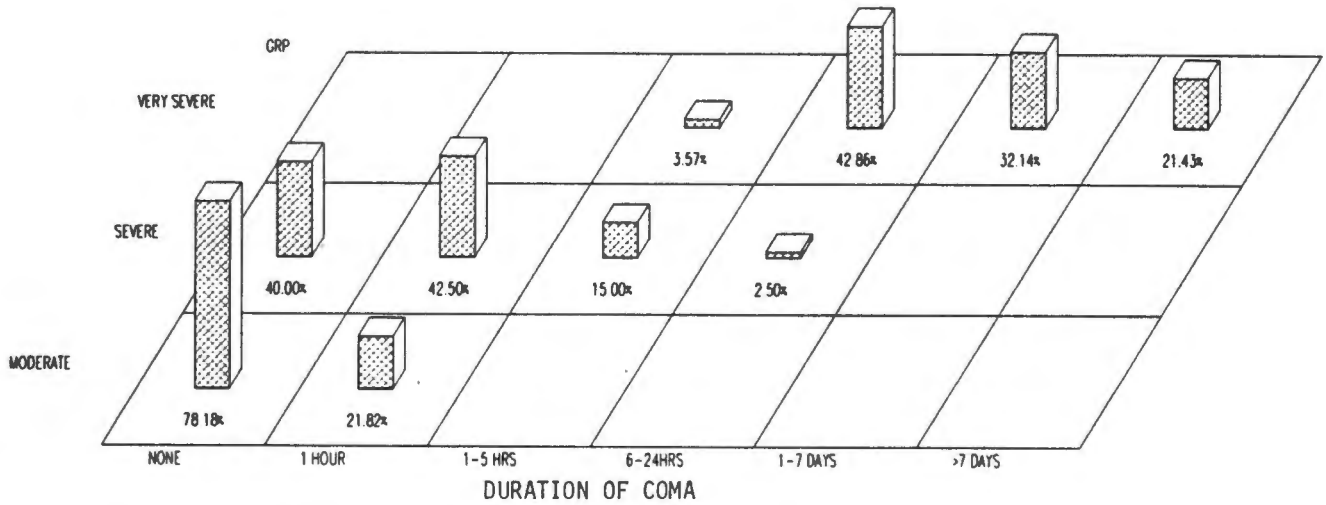


Figure 5: Coma Duration and PTA Length

With the exception of C.P., all the children rated very severe according to their PTA length were in coma for at least 6 hours. The maximum coma length was 16 days. Although children with long coma tended to have long PTA, several children with coma lasting less than 1 day had PTA lasting 3 to 4 weeks.

Only 1 child rated as severe was unconscious for more than 6 hours. He remained unresponsive for 9 hours, but this appeared to be the result of intravenous Valium given for seizure control. Of the 6 severes rated as unconscious between 1 and 5 hours, 3 had PTAs of 7 days, while the

remaining 3 had PTAs of 3 and 4 days and either definite or suspected associated focal hemispheral injury.

In summary, there may be several explanations for an initial low GCS score, but those rated very severe according to PTA are, in fact, those in coma over 6 hours.

9.1.5.3 Skull Fractures

Skull x-rays were carried out on 118 of the 123 children and the nature of the fractures found within each severity group are detailed in Table 4. It should be noted that a child could have more than one type of fracture.

Table 4

Skull Fractures in Children X-rayed

	Moderates (n=53)		Severes (n=39)		Very Severes (n=26)		Total (n=118)	
	%	(n)	%	(n)	%	(n)	%	(n)
Any skull fracture	**30.2**	(16)	**56.4**	(22)	**46.2**	(12)	**42.4**	(50)
Vault linear	**13.2**	(7)	**38.5**	(15)	**26.9**	(7)	**24.6**	(29)
Closed depressed	**3.8**	(2)	**2.6**	(1)	**3.9**	(1)	**3.4**	(4)
Compound "	**11.3**	(6)	**7.7**	(3)	**15.4**	(4)	**11.0**	(13)
Basal	**5.7**	(3)	**17.9**	(7)	**3.9**	(1)	**9.3**	(11)
Jaw/face/orbit	**1.9**	(1)	**5.1**	(2)	**3.9**	(1)	**3.4**	(4)

Children with skull fractures other than compound depressed or basal were not admitted to the multidisciplinary study unless they met other neurological criteria. Table 4 shows that about 40% of the total sample have skull fractures on x-ray. Compound depressed fractures constitute 11% of the total sample and dural tears were noted at operation in 9 out of 13 of these children. Although less than 10% of the head injured had x-ray evidence of a basal skull fracture, clinical evidence of such a fracture was present in 21% of the total sample, ie in 12 moderates (22%), 11 severes (28%) and 3 very severes (11%).

Skull fractures were related to the mechanism of injury in that children who were assaulted or fell, and pedestrians whose head was directly struck or who landed head first tended to have fractures. Twelve out of the 20 very severely head-injured pedestrians had no skull fracture.

9.1.5.4 Focal and Diffuse Lesions

At the time of the study, CT scanning facilities were available only at Groote Schuur Hospital and children were not transferred from Red Cross Hospital for this investigation unless the CT result was necessary for medical management and the child could be moved safely (ie was not on a ventilator, in traction or recovering from surgery). The scanner then available was an Elscint 905, not a particularly high resolution model. At the conclusion of the sampling period, all scans were checked by a neuroradiologist and a neurosurgeon. No reports were altered. The numbers in each severity group undergoing CT scan and the abnormalities found are presented in Table 5.

Table 5

CT Scan Results

Result	Moderates (n=12)	Severes (n=14)	Very Severes (n=21)	Total (n=47)	%
Normal	2	5	3	10	**(21.3)**
Diffuse brain swelling	2	5	7	14	**(29.8)**
Brain swelling plus focal lesion	1	0	8	9	**(19.1)**
Focal lesion only	7	4	3	14	**(29.8)**

Nearly 80% of those scanned had abnormal findings (Table 5). Twenty-three children had diffuse brain swelling, 9 in association with an additional lesion. In some of those with a normal CT scan, it was felt that the scan was done too soon after injury for brain swelling to be detected. There were also other children who were not scanned whose clinical picture (a plateau or deterioration in the level of consciousness) suggested the possibility of brain swelling.

Twenty-three children had focal lesions and in 14 there was no additional generalized brain swelling. As scanning was not carried out on all children and CT scan does not detect many of the focal lesions evident on MRI, it is possible that the frequency of focal lesions in the sample is far higher than is suggested in Table 5. Table 6 below details lesions found either on CT scan or during surgery. It should be noted that a child could have more than one type of lesion.

Table 6

Focal Lesions Detected on CT Scan or at Surgery

	Moderates (*n*=55)		Severes (*n*=40)		Very Severes (*n*=28)		Total (*n*=123)	
	%	(*n*)	%	(*n*)	%	(*n*)	%	(*n*)
Any focal lesion	16.4	(9)	17.5	(7)	46.4	(13)	23.6	(29)
Laceration	7.3	(4)	5.0	(2)	10.7	(3)	7.3	(9)
Contusion	5.5	(3)	7.5	(3)	21.4	(6)	9.8	(12)
Haemorrhage/Haematoma								
- Extradural	1.8	(1)	2.5	(1)	-	(0)	1.6	(2)
- Subdural	3.6	(2)	-	(0)	3.6	(1)	2.4	(3)
- Intracerebral	3.6	(2)	5.0	(2)	14.3	(4)	6.5	(8)

As may be seen in Table 6, a focal or mass lesion was detected in nearly a quarter of the total head-injury sample and in almost half of the very severe group. In both the

moderate and the severe groups, 18% were found to have such lesions.

Laceration plus contusion occurred in association with compound depressed skull fracture. The extent of the damage ranged from very small dural tears with little deeper involvement (2 moderates and 1 severe) to fairly extensive damage with deeper involvement. Three children (1 severe and 2 very severes) had extruded necrotic brain tissue.

Contusions alone were detected in 12 cases and underlay skull fractures in all but 2, both of whom were thought to have contrecoup injuries. Perifocal oedema produced shift across the midline in 1 severe and 3 very severe cases. The extradural haematomas occurring in 2 children were adjacent to linear skull fractures in both cases and both children had shift of the midline structures on CT scanning. Subdural haematomas were evacuated in 2 moderates (1 of whom was a haemophiliac) several days after falls, and a small subdural was detected on CT scan in a very severe and not evacuated. None of the 3 children with subdurals had skull fractures, but all had lateral brain shift.

Eight children had intracerebral haematomas. One of the moderates was the haemophiliac who also had a subdural haematoma and shift. The other moderate, 1 severe and 1 very severe had extruded intracerebral clots in association with compound depressed fractures. One severe had shift in association with an intracerebral haematoma and perifocal oedema. The 3 remaining very severes with intracerebral haematomas were all acceleration-deceleration injuries and 2 of them had had intraventricular haemorrhages.

Two children (1 moderate, 1 severe) not included in Table 6 had clinical features which suggested subarachnoid haemorrhage.

9.1.5.5 Focal Neurological Signs

Limb movements were rated as normal, weak or paralysed at admission. Localized muscular-skeletal injuries occasionally obscured neurological weakness, which could also be very transitory, or even change sides. Hemiparesis was rated by the psychologists at initial testing, and accepted only if supported by medical notes indicating weakness at admission and/or the finding of a hemiparesis by the paediatric neurologist at 6 months. On this basis, 2 children had a right hemiparesis; 5 had a left hemiparesis; 1 had a spastic quadriparesis, worse on the right; and 1, the child with the gunshot wound, had a spastic quadriplegia. Two of the children with a left hemiparesis had tremor that was worse on the right side. All except 1 of these children (J.G., a severe) were rated as very severe head injuries. J.G. was the only child who was clinically aphasic, although several had speech and language problems.

An attempt was made to determine subgroups with lateralized focal injury. This was possible on a clinical basis if EEG and psychological test findings could be used to make the decision as well as site of injury, neurological signs and radiological evidence, but not possible on any of these grounds alone. For example, several of the children with a hemiparesis had evidence of bilateral brain damage, and CT scan evidence of a unilateral lesion could also be contradicted by other clinical findings. It was decided that classification into lateralized groups was inappropriate, particularly in the more severe injuries (cf Chadwick, Rutter, Thompson & Shaffer, 1981; Levin, Benton & Grossman, 1982).

9.1.5.6 Seizure Activity and EEG Findings

Table 7

Seizure Activity, Anticonvulsants and EEG Findings at Admission

	Moderates (n=55) %	(n)	Severes (n=40) %	(n)	Very Severes (n=28) %	(n)	Total (n=123) %	(n)
Any seizure	**23.6**	(13)	**17.5**	(7)	**35.7**	(10)	**24.4**	(30)
Focal	**10.9**	(6)	**10.0**	(4)	**21.4**	(6)	**13.0**	(16)
Generalised	**2.7**	(7)	**7.5**	(3)	**14.3**	(4)	**11.4**	(14)
Anticonvulsants	**21.8**	(12)	**17.5**	(7)	**53.6**	(15)	**27.6**	(34)
	(n=43)		(n=33)		(n=22)		(n=98)	
EEG done								
Normal	**51.2**	(22)	**24.2**	(8)	**22.7**	(5)	**35.7**	(35)
Abnormal	**48.8**	(21)	**75.8**	(25)	**77.3**	(17)	**64.3**	(63)

Early seizures (50% in the first hour, 90% in the first 24 hours) occurred in a quarter of the total sample and were more frequent in the very severely injured than in the moderates and severes (Table 7). Focal and generalized seizures occurred with about equal frequency. Only 1 child out of 17 with depressed skull fracture, simple or compound, had a seizure. Those with intracranial haematomas and contusions not associated with depressed skull fractures had an increased frequency of seizures (44%).

EEGs, done during hospitalization or when the child returned for the initial neuropsychological assessment, were abnormal in 64% of the sample, with the severes and very severes having a higher frequency of abnormal EEGs than the moderates (Table 7). The most common finding was generalized slowing, often asymmetrical or tending to be more marked posteriorly. There was a focal abnormality in about 30% of the children, either nonspecific slow activity or spike-wave or sharp waves.

9.1.5.7 Additional Injuries

Table 8

Additional Injuries (not exclusive)

	Moderates (*n*=55)		Severes (*n*=40)		Very Severes (*n*=28)		Total (*n*=123)	
	%	(*n*)	%	(*n*)	%	(*n*)	%	(*n*)
Any additional injury	14.6	(8)	30	(12)	60.7	(17)	30.1	(37
Chest	1.8	(1)	10	(4)	10.7	(3)	6.5	(8
Abdomen	3.6	(2)	5	(2)	3.6	(1)	4.1	(5
Limb or pelvis	10.9	(6)	25	(10)	60.7	(17)	26.8	(33
Cervical spine	-	(0)	-	(0)	7.1	(2)	1.6	(2

As is shown in Table 8, the prevalence of additional injuries doubles for each severity group. Chest or abdominal injuries occurring in the moderates were relatively minor and did not interfere with respiration or result in blood loss. In contrast, the very severes with chest injuries needed ventilation and one had a respiratory arrest. Several of the children (21% of the very severes), had more than one additional injury. As part of the multidisciplinary study, the Injury Severity Score (ISS), which takes account of the number and severity of additional injuries, is being calculated for each child, but was not available for the present analysis.

9.1.5.8 Surgery

Table 9

Neurosurgery and Other Surgery

	Moderates (n=55) %	(n)	Severes (n=40) %	(n)	Very Severes (n=28) %	(n)	Total (n=123) %	(n)
Neurosurgery	**18.2**	(10)	**10.0**	(4)	**14.3**	(4)	**14.6**	(18)
Other surgery	**7.3**	(4)	**17.5**	(7)	**25.0**	(7)	**14.6**	(18)
Both	-	(0)	-	(0)	3.6	(1)	0.8	(1)

Craniectomies account for most of the neurosurgery in Table 9. Otherwise, 4 moderates and 1 severe had extradural and subdural haematomas evacuated, and the very severe with a gunshot wound had shattered bone and necrotic tissue removed. Surgery not involving the head mostly involved the setting of limb fractures. One severe had facial surgery under a general anaesthetic.

9.1.5.9 Complications

Table 10

Complications (not exclusive)

	Moderates (n=55) %	(n)	Severes (n=40) %	(n)	Very Severes (n=28) %	(n)	Total (n=123) %	(n)
Any complication	**9.1**	(5)	**37.5**	(15)	**46.4**	(13)	**26.8**	(33)
Respiratory	**1.8**	(1)	**5.0**	(2)	**25.0**	(7)	**8.1**	(10)
Blood supply	**1.8**	(1)	**17.5**	(7)	**14.3**	(4)	**9.8**	(12)
Infection	**3.6**	(2)	**17.5**	(7)	**25.0**	(7)	**13.0**	(16)
Other	**1.8**	(1)	**2.5**	(1)	**14.3**	(4)	**4.9**	(6)

Complications occurred in almost one third of the children (Table 10) and were more frequent in the more severely injured. Respiratory complications, which included aspiration leading to chest infection, the need for assisted ventilation, and respiratory arrest, were more frequent and more severe in the very severes. A child with moderate head injury had an episode of stridor from a neck haematoma. Most of the children with reduction in cerebral blood supply were hypotensive on admission from hypovolaemia related to extracranial injuries.

Infections, for the most part, were related to wound sepsis, although 2 children had urinary tract infections and 2 an unexplained pyrexia. One severe was pyrexial with a subarachnoid haemorrhage and another became pyrexial post-operatively. Another severe (A.D., an assaulted child with a closed head injury) required repeated surgery for a brain abscess. Two very severes, one of whom had a compound depressed fracture, developed meningitis.

Other complications occurred mostly in the very severe group, where 2 children had episodes of marked hypertension, 1 child had uncontrollable seizures and cardiac arrhythmias, and another developed a metabolic disturbance after a respiratory arrest. The moderate and severe children listed as having other complications both had persistent cerebrospinal fluid leaks.

9.1.6 Medical Variables for Outcome Prediction

The relationship between the following clinical variables and neuropsychological outcome at 1 year will be determined: mechanism of injury, GCS score, duration of coma, length of PTA, skull fracture on x-ray, evidence of basal fracture, EEG abnormality, seizure activity, additional injury, complications and surgery. The effect

of focal versus diffuse injury cannot be reliably determined across the total sample as too few children had CT scans, but will be considered within the very severe group.

9.2 Controls

Children with trauma not involving the head were collected from trauma units and convalescent homes in the Cape Peninsula. Whenever the psychologists were free, all children available were assessed. Very dull children were not excluded unless the social worker obtained an independent history of mental retardation or cerebral pathology of significance. A total of 50 children were collected, 4 had to be discarded: 2 as they had, in fact, received minor head trauma, 1 as he had a history of encephalitis and epilepsy, and 1 who had a further accident and joined the head-injured group part-way through the study.

9.2.1 Accident Details

Falls were the most common cause of injury in the control group and accounted for 44% of accidents. Most were at ground level, but others were from trees, playground equipment, horses and double-bunks. Traffic-related accidents accounted for 28% of injuries, with more than half of these being pedestrian traffic accidents. There were no assaults, and 28% had miscellaneous causes of injury (4 ran into objects, 3 had an object fall on them, 2 were kicked in play, 2 had penetrating injuries, 1 caught his arm in a spin-dryer and 1 hurt himself while running). Most had orthopaedic injuries with broken arms and legs, several had injuries requiring plastic surgery (burns were excluded); 4 had osteitis (in all cases the parents related the onset to trauma, but this may have been coincidental) and a few had little more than stitches for superficial wounds.

Matching for length of stay in hospital was not attempted, but the controls from convalescent homes would have had a hospital stay about equivalent to that of children with a very severe head injury, whereas those in hospital only overnight had an experience similar to that of children with moderate head injuries.

Parents were told that children who had had accidents were being followed up to see if there were any behaviour changes; they would be told the results of the testing and the child would be given a free neurological examination at 6 months post-injury.

9.3 Matching of Head-Injured and Control Subjects

It was intended that the controls be proportionately matched to the head-injured groups for age, sex, ethnic group and socioeconomic status (SES). Initially all children available were tested. At the later stages, care was taken not to exceed the age and sex ratios of the head-injured group.

Table 11

Matching of the Head-Injured and Control Groups

	Controls	Head Injured			
	(*n*=46)	Moderates (*n*=55)	Severes (*n*=40)	Very Severes (*n*=28)	Total (*n*=123)
			Age		
Mean (SD)	9.5 (2.1)	10.0 (2.4)	8.8 (2.1)	8.8 (2.4)	9.3 (2.4)
Range	5.8-12.9	5.9-13.8	5.8-13.8	6.2-12.3	5.8-13.8
			Sex		
%Boy:girl	70:30	73:27	65:35	54:46	66:34
			Ethnic Group		
% Coloured: White	87:13	89:11	90:10	96:4	91:9

Note. Children who would turn 6 before the 3 month assessment were included.

9.3.1 Age

The average age of the combined head-injured group is not significantly different from that of the controls (Table 11), [$t(167)=0.11$, $p=.913$]. However, in an overall comparison of the four groups, age differences are statistically significant, [$F(165,3)=2.98$, $p=.033$]. None of the pairwise comparisons are significant, although differences between the moderates and the severes and between the moderates and the very severes approach statistical significance: $t(94)=6.45$, $p=.013$ and $t(82)=4.86$, $p=.030$ respectively (Note: p must be <.01 to be significant with six comparisons). In view of this finding, all neuropsychological test scores will be age adjusted.

9.3.2 Sex

There are no significant differences in the sex ratios (Table 11) of all four groups, $X^2(3, n=169)=3.30$, $p=.348$. Where appropriate, different norms will be used in transforming the neuropsychological test scores of boys and girls.

9.3.3 Ethnic Group

The groups do not differ significantly in the proportions of coloured and white children included $X^2(3, n=169)=1.39$, $p=.708$. There are only 11 white children in the study, only 1 of whom is in the very severe group. (Note: Black children were excluded from the present analysis on the grounds of being Xhosa-speaking). No further account is taken of ethnic group in this study.

9.3.4 Socioeconomic Status (SES)

SES was determined on the basis of the father's occupation, or the mother's occupation if there was no father or he was unemployed. Table 12 summarizes the distribution of occupational levels within the groups.

Table 12

Socioeconomic Status (SES)

	Controls (n=46) % (n)	Head Injured: Moderates (n=55) % (n)	Severes (n=40) % (n)	Very Severes (n=28) % (n)
1. Professional/ Managerial	4 (2)	5 (3)	2.5 (1)	0 (0)
2. Administrative/ Clerical	13 (6)	7 (4)	15 (6)	11 (3)
3. Skilled manual/ Semiskilled	41 (19)	35 (19)	30 (12)	21 (6)
4. Unskilled labourers	28 (13)	35 (19)	42.5 (17)	46 (13)
5. Pension, disability grant	7 (3)	11 (6)	5 (2)	18 (5)
6. Unemployed	7 (3)	7 (4)	5 (2)	4 (1)
Median SES	3	4	4	4

As is shown in Table 12, the bulk of the sample comes from social classes 3 and 4. On the median test, there are no significant differences in social class between the groups, $X^2(3, n=169)=2.19$, $p=.534$. The effect of SES on tests will be investigated and adjustments made where necessary.

9.3.5 Language

Language was not considered in collecting controls as it was thought that this factor would occur in the sample as in the population, ie about 64% would be Afrikaans speaking and 36% English speaking (Scheffer, 1983). However, all of the very severe head injuries are Afrikaans speaking and 78% of both the severes and moderates. This compares with 63% of the control group.

To some extent language is linked with socioeconomic level in that English is the language of choice in upwardly mobile coloured families, even where the parents have an Afrikaans background (Scheffer, 1983). When language functions are assessed, the influence of being English or Afrikaans speaking will be determined and adjustments made to test scores if necessary.

9.4 Psychosocial Background (see Appendix A2, p.431).

The detailed collection and analysis of psychosocial background data for the head injured and controls is a separate part of the UCT Child Head Injuries Survey (see Theron, 1987). However, details on psychosocial adversity, premorbid school backwardness and premorbid behaviour disturbance which are considered in the prediction of cognitive outcome, are summarized here.

9.4.1 Psychosocial Adversity

The distribution of various psychosocial adversity factors within the groups is summarized in Table 13 below. Percentages are rounded. A psychosocial adversity score was calculated for each child, based on one point for the presence of each of the factors (Theron, 1987).

Table 13

Psychosocial Adversity

	Controls		Head Injured					
			Moderates		Severes		Very Severes	
	(*n*=44)		(*n*=55)		(*n*=40)		(*n*=28)	
	Psychosocial Adversity Factors							
	%	(*n*)	%	(*n*)	%	(*n*)	%	(*n*)
Large family size	52	(23)	46	(25)	48	(19)	46	(13)
Overcrowding	39	(17)	55	(30)	45	(18)	57	(16)
Unskilled/unemployed	36	(16)	38	(21)	53	(21)	61	(17)
Maternal education < Std.6	59	(26)	75	(41)	73	(29)	86	(24)
Mother psychiatric	5	(2)	33	(18)	48	(19)	21	(6)
Father psychiatric	21	(9)	29	(16)	25	(10)	43	(12)
Single parent	27	(12)	31	(17)	28	(11)	29	(8)
Disharmony in home	23	(10)	51	(28)	58	(28)	43	(12)
Ill health, either parent	16	(7)	20	(11)	13	(5)	29	(8)
Nutrition	9	(4)	20	(11)	20	(8)	3	(10)
Median Psychosocial Adversity Score	3		4		4		4.5	

It is evident in Table 13 that there is a high level of psychosocial adversity present in the controls as well as in the head injured. Large family size (4 or more children), overcrowding (more than 3 people per room or more than 2 per bed) and single parents (or broken homes), are common problems. Psychiatric disturbance in the mother (either alcoholism or nervous complaints for which she received medical treatment), is far more common for the head injured (35%) than for the controls (5%). Psychiatric disturbance in the father, which was predominantly alcoholism, is more frequent for the very severely injured than for the other groups.

Generally, the very severely injured appear to have slightly greater socioeconomic disadvantage than the others, in that more of their breadwinners are unemployed or unskilled; fewer of their mothers have attained Std 6 education; more of their parents have physical ill-health, and over a third of the children have inadequate or unbalanced nutrition. In contrast, psychiatric problems in the mothers and disharmony in the home are somewhat more frequent for the moderates and severes than for the very severes.

9.4.2 Premorbid Scholastic Achievement

In determining premorbid levels of cognitive functioning, the ideal would be to have premorbid IQs and school reports. Most children had not had previous IQ tests and only about 30% of schools responded to requests for reports. Table 14 summarizes the school history of the different groups.

Table 14

School History

	Controls		Head Injured					
			Moderates		Severes		Very Severes	
	%	(n)	%	(n)	%	(n)	%	(n)
Preschool Attenders								
	(n=44)		(n=55)		(n=40)		(n=28)	
	57	(25)	**71**	(39)	**65**	(26)	**79**	(22)
School Achievement								
	(n=39)		(n=50)		(n=33)		(n=23)	
Above average	**28**	(11)	**20**	(10)	**15**	(5)	**22**	(5)
Average	**43.5**	(17)	**40**	(20)	**39**	(13)	**35**	(8)
Struggling	**2.5**	(1)	**16**	(8)	**9**	(3)	**9**	(2)
Failed	**26**	(10)	**24**	(12)	**36**	(12)	**35**	(8)

More than half the controls and 70% of the head injured (Table 14) did not attend preschool. Rating of school achievement was based on details given by the parents. For the purpose of analysis, children rated as struggling or as having ever failed, are regarded as having poor school progress and the others as adequate school progress. Calculations based on Table 14, show 28% of the controls as opposed to 42% of the head injured to have poor school progress.

A few of the children had not started school (2 controls, 8 head injured) or had not attended for long enough for their progress to be rated (2 controls, 9 head injured). Of the schoolgoers, 29% of the controls and 37% of the head injured had changed school at least once.

9.4.3 Premorbid Behavioural Adjustment

At the initial psychosocial interview, usually carried out while the child was still in hospital, parents were asked to describe the child's premorbid development, health, school performance, family and peer relationships (see Appendix A2, p.431) and were finally asked the questions on the Rutter Parent Scale (Rutter, Graham & Yule, 1970). This is normally a self-administered questionnaire, but as several parents were illiterate or might not understand the language used, the questions were put to all parents by the interviewers. Children with Rutter scores of 13 and over were considered to have behaviour disturbance. Table 15 presents the median Rutter scores and the percentage rated as disturbed.

Table 15

Rutter Parents Scale: Median Scores and Frequency of Behavioural Disturbance

	Controls	Head Injured		
	(n=45)	Moderates (n=55)	Severes (n=40)	Very Severes (n=27)
Median	4	11	10	7
Range	0-22	0-40	0-33	0-31
% Disturbed (n)	13% (6)	42% (23)	35% (14)	15% (4)

It can be seen in Table 15 that it is the children who received moderate and severe head injuries who had the higher rate of premorbid behaviour disturbance. The rate of premorbid behaviour disturbance in those receiving very severe head injuries is no different from that of the control group.

9.4.4 Previous Head Injury

Cerebral pathology of significance was an exclusion criterion for the neuropsychological study. Nonetheless, analysis of the psychosocial background data showed 5 controls (11%), 12 moderates (22%), 5 severes (12.5%) and 4 very severes (14%) to have had previous head injuries. Most of these were very minor, with scalp bleeding and occasionally, vomiting and/or stitches. Only 1 child, a moderate, was reported as having been unconscious briefly, and 1 control and 1 moderate were reported as having had fractured skulls.

9.4.5 Psychosocial Variables for Outcome Prediction

In the analysis to determine which variables influence neuropsychological outcome at 1 year, the following psychosocial variables will be considered: age, sex, language, psychosocial adversity score, SES, preschool attendance, premorbid scholastic level, Rutter score, and previous head injury.

9.5 Procedure

All children admitted to Trauma Unit with suspected head injuries were screened by the head injury team's nursing sisters within 24 hours. If they met any of the severity inclusion criteria for the study (Table 2, p.138), accident, clinical and management details were filled in on a prepared format (summarized Appendix A1, p.430). Psychosocial background data was collected by the social worker (summarized Appendix A2, p.431).

Children were visited daily to determine when they were out of PTA and sufficiently comfortable to be tested. Children who were discharged still in PTA were brought back at weekly intervals and tested when they were judged to be clear (see pp.140-142 on PTA assessment).

9.5.1 Test Intervals

The median length of time from injury to the first assessment, T1, was 6 days for the moderates (range 1 to 38 days; 90% within 14 days), 10 days for the severes (range 3 to 46 days; 90% within 24 days), and 28 days for the very severes (range 9 to 73 days; 90% within 55 days). Head-injured and control children were retested, T2, as close as practically possible to 3 months after their first assessment. The median interval from T1 to T2 was 3.1 months and for 93% of the children the interval was less than 4 months. (Note: Because of differences in the timing of T1, this meant that in terms of time post-injury, the moderates were retested at a median of 3.3 months, the severes at a median of 3.5 months and the very severes at a median of 4.4 months).

The third assessment, T3, was done as close as possible to 1 year post-head-injury. This fixed time interval was chosen to make the present study comparable with other studies. The median time lapse from injury to T3 was 12.3 months for the moderates, 12.4 months for the severes and 12.5 months for the very severes, and 93% of the head injured were retested within 14 months of injury. The controls were rescheduled at 12 months after T1. The median time between T1 and T3 was 12.2 months and 90% were retested within 14 months. The few children who were late for reassessments were scattered over the groups and were considered to counterbalance each other.

9.5.2 Test Conditions

Testing was carried out by the clinical psychologists of the neurosurgery department, in a quiet office where possible, but on the ward if the child was in traction or no other room was available. All follow-up assessments were carried out in offices. The variation in test conditions at intake, T1, for the different groups, is shown in Table 16.

Table 16

Test Conditions at Initial Assessment (T1)

	Controls		Head Injured					
			Moderates		Severes		Very Severes	
	(n=46)		(n=55)		(n=40)		(n=28)	
	%	(n)	%	(n)	%	(n)	%	(n)
Test Setting								
Office	**50%**	(23)	**69%**	(38)	**82.5%**	(33)	**68%**	(19)
Quiet ward	**26%**	(12)	**20%**	(11)	**10%**	(4)	**14%**	(4)
Noisy ward	**24%**	(11)	**11%**	(6)	**7.5%**	(3)	**18%**	(5)
In Traction or Impeded								
	48%	(22)	**13%**	(7)	**17.5%**	(7)	**21%**	(6)

As can be seen in Table 16, more of the control group children than of the head-injured were tested in the wards and in traction. There was concern that children tested in noisy wards and in traction would show greater gains from T1 to T2 than those tested in easier conditions and that this would favour improvement in the controls rather than in the head injured. However, a comparison within the control group found no significant differences in the FSIQ gains from T1 to T2 between those tested in different settings, [median test $X^2(2, n=40)=2.32$, $p=.31$].

The most difficult tests for children in traction or otherwise impeded at T1 were the Detroit and the Beery. Within the control group, there were no significant differences between those in traction versus those unimpeded in the size of gains from T1 to T2 on the Detroit (Wilcoxon $Z=-0.12$, $p=.90$) or the Beery (Wilcoxon $Z=0.91$, $p=.36$). It can be concluded, therefore, that test conditions at T1 did not affect the size of gains on tests between T1 and T2.

9.5.3 Attrition

Of the 123 head-injured children, 1 moderate and 1 very severe were tested only at T1, and 2 moderates and 2 severes came only at T1 and either T2, or T3. The attrition rate was 2.4% at T2 and 4.1% at T3 and only 4.9% for either follow-up. This is so low that there is no need to compare these children statistically with those remaining. The very severe child lived in Johannesburg, other children moved and some parents were unable to take time off or simply failed to keep appointments.

In the control group, 2 of the 46 were present only at T1, and 5 came only at T1 and either T2 or T3. This is an attrition rate of 8.7% at T2 and 10.9% at T3, and 15.2% on either occasion. The 7 controls who were not present on all testing occasions (again for reasons to do with their parents' difficulties in bringing them) were not significantly different on median tests in terms of their age, social class and IQ from the 39 who were present.

The low rate of attrition is remarkable, particularly considering the difficulties in getting to hospital once unrest and riot conditions broke out from September 1985, in the follow-up period. The team made every effort to build relationships and overcome difficulties: eg meals were provided, employers requested not to deduct wages for days missed; transport costs refunded to the poor; letters written to schools, housing authorities and welfare agencies and referrals made to other hospital departments whenever necessary.

9.6 Test Battery (see Summary Sheet, Appendix A3, p.432).

9.6.1 Principles of Test Selection

Measures of general ability and of specific neuropsychological functions were required. Measures shown in other studies to be sensitive to brain injury were favoured. It was expected that, as a result of head injury and/or cultural deprivation, many children would score below their age levels, so tests with a wide range and particularly those with a low floor, were chosen. Where available, tests with age norms were used. Internationally used tests were preferred above South African tests to make the results of this study comparable with those of other studies. (Neither South African, nor international tests, have been standardized on the population predominating in this study). It was further required that the tests should be portable for ward usage, repeatable, and relatively culture-free. These principles led to the selection of the battery listed from 9.6.3 to 9.6.7.

9.6.2 Age Adjustment of Test Scores

To make the groups comparable and also to be able to determine which children have defective scores at one year post-injury, it is essential that scores are adjusted for age. Where possible, test scores are expressed as standard scores and scores other than the familiar Wechsler scores were converted to T scores (mean=50, standard deviation=10). Where only the mental age equivalents of scores were available, scores are expressed either as age ratios, (ie $\frac{MA}{CA} \times 100$) or, where there was a limited range of age equivalents such that these were not available for low or high scores, as a percentage of the expected score for age, (ie $\frac{\text{raw score}}{\text{expected score for age}} \times 100$). Where the age equivalents were based on inadequate normative samples, or where it was shown that language, sex or SES affected

control group scores as well as age (see Statistical Methods), adjustment was done by analysis of covariance. (Note: Analysis of covariance could not be used on all measures in the study to give uniformity as the effects of age in the various groups differed significantly on some measures).

9.6.3 Intelligence

Wechsler Intelligence Scale for Children - Revised (WISC-R), (Wechsler, 1974).

Verbal subtests: Information, Comprehension, Arithmetic and Digit Span.
Performance subtests: Picture Completion, Block Design, Coding and Mazes.
Subtotals prorated to give Verbal IQ, Performance IQ and Full Scale IQ.

American questions were rephrased in local terms and the Groote Schuur Hospital Afrikaans version was used where necessary. Object Assembly was omitted because of the repeat-testing design, Picture Arrangement because of cultural loading, and Similarities because local children tend to score low on this subtest.

9.6.4 Motor Speed and Coordination

1. Purdue Pegboard: (Purdue Research Foundation, 1948). Pegs placed in 30 seconds: Preferred hand, Nonpreferred hand, Both hands. Scores are transformed into T scores, using the Gardner and Broman norms for boys or girls (1979).

2. Successive Finger Taps: (Denckla, 1973, 1974). Time taken to do 20 successive taps, ie 5 sets of 4, with the thumb moving from the index finger to the little finger; Preferred hand, then the Nonpreferred hand. The Denckla norms are guidelines based upon small samples of children aged between 5 and 10 years. Analysis of covariance is therefore used to adjust scores for age and sex as necessary.

3. Incoordination on WISC-R Mazes: A qualitative rating of incoordination as none, some or marked, is given based upon observation of the child's performance. Children who hit the maze walls because of difficulty with pencil control are rated as incoordinate.

9.6.5 Visuographic

1. Detroit Motor Speed and Precision: (Baker & Leland, 1967). The score is the number of crosses placed within 2 minutes inside circles which diminish in size from row to row. The age equivalents of scores ranging from 7 to 164 were available and scores are transformed into age ratios.

2. Beery Developmental Test of Visual Motor Integration: (Beery, 1982). The score is the number of designs correct. Scores are transformed into T scores using the manual.

9.6.6 Language

9.6.6.1 Production

1. Word fluency: The score is the number of different words produced in one minute after the examiner has given the instruction: "I want to see how many different words you can say in one minute. You say the words as fast as you can and I will count them. Any words will do, like 'dog', 'chair', 'blue', 'jump'. Alright? Begin!"
Expected scores for ages 6 through 15 were available from a local standardization of this test at the Athlone School Clinic (Child Guidance Clinic, unpublished study). Age equivalents of low scores were not available and scores are therefore expressed as a percentage of the expected score for age (see Appendix B1, p.433).

2. Renfrew Picture Naming: (Renfrew, 1968). Two measures are derived: the number of pictures named correctly out of 59, and the speed of naming, based on the average length of time taken for the first 10 pictures. Renfrew norms extend only to age 8. Scores on both measures are adjusted by analysis of covariance for age, and also for sex, language and SES, where these factors affect control group scores significantly.

9.6.6.2 Repetition

1. Sentence Repetition: (Detroit Auditory Attention Span for Related Syllables, Baker & Leland, 1967). The test was translated into Afrikaans. Age equivalents of scores obtained by children between 3 and 19 years were available, and scores are transformed into age ratios.

2. Digit Repetition: The score is the number of digits forward repeated on the WISC-R (Wechsler, 1974). As the age equivalents of low and high scores are uncertain, scores are expressed as a percentage of the expected score for age. At age 6 the expected score is 4, at ages 6.5 through to 10 years the expected score is 5, and at age 10 years and upwards, 6 is the expected score (Junior SA Individual Scales, 1981; Terman-Merrill, 1973).

9.6.6.3 Comprehension

1. Stanford-Binet Pictorial Identification: (Terman-Merrill, 1973). The score is the number of pictures out of 6 correctly pointed out in response to simple questions (eg "show me the one we cook on", "show me the one we carry when it is raining"). This is a screening test and scores are analysed in terms of those achieving perfect scores, and those not.

2. Token Test for Children: (Di Simondi, 1978). Parts 1, 3 and 5 were administered. The Afrikaans translation of the Logopaedics Department was used. Parts 1 and 3 are reported in terms of the percentage of children achieving the expected score for their age. Part 5 scores are adjusted by analysis of covariance for age and language.

9.6.6.4 Speech Ratings

1. Articulation: is rated as normal, immature or impaired. Immature includes lisping (eg the distorted articulation of s, z, th and sh sounds) and the substitution of w for r sounds (Pirozzolo et al, 1981). Impaired includes defects in articulation, resonance, stress and intonation more severe than those expected on the basis of immaturity (Sarno, 1980).

2. Perseveration: is rated as none, some or marked and refers to the perseveration of nonverbal responses as well as perseveration of words, ideas and/or approaches to problem-solving.

3. Echolalia: is rated as none, some or marked and refers to the unintentional echoing of words, phrases and/or questions.

9.6.7 Memory

Selective reminding of a 12 item shopping list over 8 trials was used (Buschke, 1973,1974). Three lists were derived (Appendix B2, p.434) and presented in a random order over the three test sessions. Scores derived, (illustrated in the Results section), include the sum over 8 trials for Recall, for Long Term Storage (LTS) and for Consistent Retrieval (CR). Each item given that was not on the list at a test occasion, scored a point for Confabulation or for Interference if it had been on a previous list. Repetition of items was checked as none, some or marked.

The length of the test battery (1.5 to 2 hours) was such that a visual retention test could not be included.

9.7 Statistical Methods

All statistical analyses were carried out using SAS (SAS Institute, 1985) or BMDP (1985).

9.7.1 Data Cleaning

Before analysis, extensive data cleaning was done. Variables were examined for internal anomalies and, if they appeared on either the medical or psychosocial data sets as well as the neuropsychological data set, were cross-tabulated and inconsistencies investigated.

9.7.2 Data Transformations

Where necessary, variables were transformed for analysis to T scores, age ratios or percentage scores, or by analysis of covariance, as outlined in Section 9.6.2. To adjust variables by analysis of covariance, the following procedure was used. Within the control group, linear regression of the variables age, sex, language and social class was carried out on the test raw scores. (These results are not reported). If any of these four variables was found to be significant ($p<.05$) in the regression within the controls, analysis of covariance was used to estimate the coefficients which had to be used in the adjustment of the scores of individuals in all groups. The coefficients were estimated by the pooled estimate of the slope in the analysis of covariance. Cases with large standardised residual and large Cook's distance were excluded before the analysis of covariance because of their large influence on the estimate of the coefficient. Where scores were adjusted by analysis of covariance for age, all scores were adjusted to 114 months at T1, 117 months at T2, and 126 months at T3, the mean ages of all individuals at these time points. For the analysis of recovery over time, all scores were adjusted to 126 months.

9.7.3 Comparisons of Groups at a Specific Time, (ie T1, T2 and T3

All cases who have data available at a given assessment are included in the comparison of the 4 groups at that assessment. This is so as to use as much of the data as possible. On the motor tests, for example, many children have missing information at the first assessment because of arm injuries or traction, but they are included in the analysis of the later assessments where information is available.

In the case of continuous variables, if the assumptions of normal distribution apply, the groups are compared using Tukey's studentized range at an overall .05 level of significance. If the data is skewed or has some outlying values which would influence the mean, pairwise comparisons are carried out with the nonparametric Wilcoxon rank sum test, if the overall Kruskal-Wallis chi-squared test is significant. The median test is used when several observations have tied values.

In the case of categorical variables, the groups are compared on the Pearson chi-squared test, or, if the expected frequency in any cell is less than 5, Fisher's exact p-value is calculated. On all the nonparametric tests, a significance level of .01 is needed to compensate for the 6 pairwise comparisons.

At the 1 year assessment (T3), each head-injured group is compared with the control group with respect to the percentage of cases classified as persistently impaired on each of a number of variables. As three comparisons are made, p needs to be less than .017 to maintain an overall level of significance of .05. Persisting impairment is defined as a score worse than that obtained by 95% of the controls at T3. The control group 5th percentile is used as the cut-point unless this does not make clinical sense, when two standard deviations below the control group mean is used.

9.7.4 Comparisons of Groups Over Time

The analysis of recovery over time uses only individuals with information at all three assessments. For each continuous variable the data is summarised in a box plot by group and assessment. In the case of normally distributed data, repeated measures analysis of variance is used (PROC GLM in SAS). This analysis tests for significant

changes over two time periods (T1 to T2 and T2 to T3) and whether the groups differ as to the amount of change over these two periods. If the groups differ as to the amount of change over a time period, Tukey's studentized range test is performed on the changes to ascertain which groups differ from which. Within each group, paired t-tests are performed to determine which changes over time differ significantly from zero, and whether the change over the period T1 to T2 differs significantly from the change T2 to T3.

In the case of non-normally distributed continuous data, pairwise Wilcoxon or median tests are carried out to determine whether the groups differ as to the amount of change over a time period. The statistical significance of changes within each group is assessed on the signed rank test. In the case of categorical variables, the statistical significance of changes within each group is determined on McNemar's test.

9.7.5 Predicting Outcome

Two measures of outcome are defined: Performance IQ (PIQ) at T3 and a composite Neuropsychological Impairment Index (NII), which is dichotomized into GOOD/BAD outcome, depending on the number of variables on which an individual has persisting impairment at T3. The association between NII outcome and medical and psychosocial variables in the head injured is initially assessed univariately by means of median, chi-squared or Fisher's exact tests. Next, multiple logistic regression is used to predict NII outcome from the medical, psychosocial and neuropsychological variables, either separately or in combination. For children with missing neuropsychological data at T1, the mean value of their severity group on that variable is entered in the regression to minimize the number of cases excluded. The programme, BMDPLR, generated design variables for the

categorical medical and psychosocial variables and these will be given in the Outcome Section along with the coefficients obtained. A backwards stepping procedure is used, ie the stepping procedure starts with all the possible predictor variables in the model. The decision to remove or enter any variable from the model, is based on an estimate of the asymptotic covariance matrix of the coefficients, from which an approximate F-value is computed. The limits of the p-values for variables to be entered into or removed from the model, are 0.15 and 0.10 respectively. The constant term is retained in the model throughout. The percentage of children who are correctly predicted by the best models, using the 0.5 cut-point on the predicted probabilities, is provided.

For PIQ, multiple linear regression was performed (Draper & Smith, 1981). This was done by all subsets regression using BMDP9R. Cp, the mean squared error of prediction, was used as the criterion for selecting the best model. PIQ at T3 is also predicted from PIQ at T1 using Spearman rank correlation and calculating R^2 (the percentage of the variance accounted for).

9.7.6 Additional Analyses

Groups are compared on the appropriate parametric or nonparametric tests according to whether the data is continuous (normally distributed or not), or whether it is categorical.

Matching variables: Matching of the four groups is compared on an overall F test and pairwise t-tests for age; by the median test for SES, and by chi-squared tests for sex and ethnic group.

Effects of Test Conditions: In post-hoc analyses on gains within the control group from T1 to T2 under different test conditions, the median test or the Wilcoxon was used.

VIQ versus PIQ: Discrepancies between VIQ and PIQ within the four groups are compared on paired t-tests at each test occasion. Gains on VIQ versus gains on PIQ are compared within each group for each of the recovery intervals using paired t-tests.

Adequate versus Poor Scholars: The IQs of adequate versus poor scholars within each group are compared on t tests. Group mean IQs are adjusted by analysis of covariance for school progress, and each group is compared with the control group using t tests.
In the Outcome section, adequate and poor scholars are compared (a) on PIQ dichotomized as ≥ 80 versus <80 and (b) NII outcome dichotomized as GOOD/BAD, using chi-squared tests for overall comparisons and Fisher's exact p within the groups.

The Effects of PTA Length (Post-hoc analyses)
The head injured are further subdivided into 7 or 8 groups with increasing PTA length and mean or median scores calculated on the memory measures, and on PIQ and NII at T3. Spearman rank correlations for PTA in days with (a) the memory measures at T1, T2 and T3, (b) with PIQ at T3, and (c) with NII at T3 are calculated.

The Independence of Tests and the Influence of IQ
Within the language, visuomotor and memory results sections, the overlap between the different tests used and the influence of IQ on them are determined by Spearman rank correlations within the control group at T1.

Note. All statistical symbols were italicized but could not be reproduced on the laser printer.

10. INTELLIGENCE RESULTS

10.1 Format

WISC-R IQ results are first tabulated for all subjects present at each testing occasion and the significance of differences between the groups is determined. Confidence limits for the multiple group comparisons are given in Appendix C. Sub-analyses of Verbal-Performance discrepancies within the groups, and of the effects of premorbid school progress on IQ at T1, follow. The repeated measures analysis of changes (a) over time and (b) between the groups, are then presented, based on the reduced sample present on all three test occasions. The distribution of scores in these subjects is depicted in box plots, and the size of changes from T1 to T2 and from T2 to T3 is tabulated. Significant changes within each group are described and changes in the first recovery period (T1 to T2) are compared with those in the second recovery period (T2 to T3). (Paired t-tests and p values are tabulated in Appendix C). Changes in VIQ are compared with changes in PIQ. The percentage of subjects with persisting impairment of IQ at one year post-injury is determined.

The details, first of the Verbal and then of the Performance subtests, are then presented in the same format: differences between the groups, changes over time, and the percentage with persisting impairment on each subtest.

10.2 Differences Between the Groups

The WISC-R Verbal, Performance and Full-Scale IQs obtained by the controls and head-injured groups at the three test occasions are summarized in Table 17. Groups not sharing a single letter subscript (a,b or c) in Table 17 at a particular test occasion differ significantly at $p<.05$ on Tukey's studentized range test. Confidence limits for the multiple group comparisons are given in Table C1, p.435.

Table 17

WISC-R: Verbal, Performance and Full-Scale IQs at T1, T2 and T3

Test Occasion	Controls M	Controls SD	Moderates M	Moderates SD	Severes M	Severes SD	Very severes M	Very severes SD
Verbal IQ								
T1	(n=46)		(n=55)		(n=39)		(n=28)	
	91.2$_a$	12.5	**83.7**$_b$	13.0	**83.5**$_b$	14.3	**72.2**$_c$	13.3
T2	(n=42)		(n=53)		(n=40)		(n=27)	
	93.5$_a$	14.4	**87.9**$_{ab}$	13.5	**85.5**$_b$	14.3	**73.6**$_c$	12.4
T3	(n=41)		(n=53)		(n=38)		(n=27)	
	96.1$_a$	14.9	**89.7**$_{ab}$	14.5	**87.7**$_b$	14.7	**77.7**$_c$	12.1
Performance IQ								
T1	(n=46)		(n=55)		(n=40)		(n=27)	
	95.4$_a$	13.5	**84.7**$_b$	17.3	**86.4**$_b$	16.8	**63.9**$_c$	14.1
T2	(n=42)		(n=53)		(n=40)		(n=26)	
	103.5$_a$	14.1	**93.6**$_b$	17.0	**93.6**$_b$	17.7	**73.0**$_c$	11.5
T3	(n=41)		(n=53)		(n=38)		(n=26)	
	105.6$_a$	15.8	**96.4**$_b$	16.3	**99.8**$_{ab}$	19.2	**79.2**$_c$	12.8
Full Scale IQ								
T1	(n=46)		(n=55)		(n=39)		(n=27)	
	92.5$_a$	12.7	**82.9**$_b$	14.9	**83.9**$_b$	15.0	**65.7**$_c$	13.7
T2	(n=42)		(n=53)		(n=40)		(n=26)	
	98.0$_a$	13.8	**89.5**$_b$	15.2	**88.4**$_b$	15.6	**70.9**$_c$	11.6
T3	(n=41)		(n=53)		(n=38)		(n=26)	
	100.4$_a$	14.9	**92.2**$_a$	15.1	**92.9**$_a$	17.1	**76.8**$_b$	12.2

Note. Means not sharing a subscript at a test occasion differ significantly on Tukey's studentized range at $p<.05$.

Table 17 shows that the very severes have significantly lower Verbal (VIQ), Performance (PIQ) and Full Scale (FSIQ) IQs than the controls and than the other head-injured groups on all three test occasions. The severes are significantly lower than the controls on all three intelligence scales at T1 and T2. There is a trend for them to have lower mean IQs at T3, but this reaches statistical significance only for VIQ.

The moderates have significantly lower mean IQs than the controls on all three scales at T1 and a trend to lower IQs at T2 and T3. These differences are statistically significant for PIQ and FSIQ at T2 and for PIQ at T3, while the FSIQ difference (8.2 points) approaches statistical significance (confidence limits -0.017 to 16.34). There are no statistically significant differences between the moderates and the severes on any of the mean IQs.

If the moderates and severes are combined at T3 to make a single group (n=91) which has PTA less than 8 days, (ie a group comparable to the "mild" head-injury group in the Chadwick-Rutter, 1981, study), then their mean VIQ becomes 88.9 (SD 14.6), PIQ 97.8 (17.5), and FSIQ 92.5 (15.9). These means are all significantly lower than the corresponding IQs of the controls [$t(130)=2.62$, $p<.01$; $t(130)=2.42$, $p<.05$ and $t(130)=2.69$, $p<.01$ respectively].

10.3 Verbal versus Performance IQ

The pattern of verbal-performance discrepancies within the groups was analysed using paired t tests. Table 18 below summarizes the size of VIQ minus PIQ discrepancies within each group on each test occasion and indicates whether the discrepancy is statistically significant.

Table 18

WISC-R: Verbal-Performance Discrepancies at T1, T2 and T3

	Controls		Head Injured					
			Moderates		Severes		Very severes	
	M	SD	M	SD	M	SD	M	SD
T1	(n=46)		(n=55)		(n=39)		(n=27)	
	-4.22*	11.3	**-1.00**	12.2	**-3.41**	12.7	**8.26*****	11.2
T2	(n=42)		(n=53)		(n=40)		(n=26)	
	-9.90***	13.0	**-5.70*****	11.2	**-8.15*****	12.8	**0.42**	11.6
T3	(n=41)		(n=53)		(n=38)		(n=26)	
	-9.41***	14.9	**-6.64*****	12.8	**-12.1*****	10.4	**-1.46**	10.3

* V-P discrepancy significant within that group at p<.05.
*** p<.001.

Within the control group (Table 18) the PIQ is consistently significantly higher than VIQ [t(45)=-2.53, p<.05; t(41)=-4.95, p<.001; t(40)=-4.05, p<.001; at T1, T2 and T3 respectively]. Within the moderate head-injured group and also within the severe head-injured group, there are no significant V-P differences at T1 [t(54)=-0.61, p>.05; t(38)=-1.68 p>.05, respectively], but PIQ is significantly higher than VIQ by the second test occasion, T2, [t(52)=-3.69, p<.001 for the moderates and t(39)=-4.01, p<.001 for the severes] and remains higher at T3 [t(52)=-3.73, p<.001 and t(37)=-6.14, p<.001, for the moderates and severes respectively].

Within the very severe group, the opposite pattern prevails and PIQ is significantly lower than VIQ at T1 [t(26)=3.85, p<.001], but there are no significant differences between VIQ and PIQ at T2 and T3 [t(25)=0.19, p>.05 and t(25)=-0.71, p>.05, respectively].

10.4 The Effect of Premorbid School Progress

Ratings of scholastic progress pre-injury were obtained from the parents at the intake interview as explained in Method, p.163. Poor scholastic progress was less common in the control group (28%) than in the head-injured groups (40%, 45% and 43% of the moderates, severes and very severes respectively). Table 19 presents IQs at initial assessment for those with adequate (average or above average) scholastic progress versus those with poor progress (struggling, having failed, in special class, or dropped out). Numbers are fewer than in Table 17 as progress was unknown for several children. It should be noted that parental judgments were not necessarily valid. Differences between the IQ means of adequate and poor scholars were analysed using t tests.

Table 19

WISC-R IQs: Adequate versus Poor Scholars at T1

	Controls		Head Injured					
School Progress			Moderates		Severes		Very Severes	
	M	SD	**M**	SD	**M**	SD	**M**	SD
			Verbal IQ					
	(n=28)		(n=30)		(n=17)		(n=13)	
Adequate	**95.1**	12.7	**88.4**	12.5	**90.8**	11.6	**77.9**	14.0
	(n=11)		(n=20)		(n=15)		(n=10)	
Poor	**84.2***	9.8	**76.6****	7.5	**73.7*****	12.9	**64.2***	11.0
			Performance IQ					
	(n=28)		(n=30)		(n=18)		(n=12)	
Adequate	**98.9**	13.7	**89.1**	15.5	**94.1**	17.5	**68.8**	13.1
	(n=11)		(n=20)		(n=15)		(n=10)	
Poor	**89.8**	13.3	**76.9****	11.9	**79.1***	14.9	**58.7**	13.9

* Significant differences between adequate and poor scholars within that group at p<.05; **p<.01, ***p<.001.

Table 19 shows that the poor scholars within each group have significantly lower verbal IQs than the adequate scholars, [$t(37)=2.57$, $p<.05$; $t(48)=3.8$, $p<.001$; $t(30)=3.94$, $p<.001$; and $t(21)=2.53$, $p<.05$ for the controls, moderates, severes and very severes respectively]. The poor scholars also tended to have lower PIQs than the adequate scholars and these differences are significant within the moderate and severe groups [$t(48)=2.97$, $p<.01$, and $t(31)=2.63$, $p<.05$ respectively] and approach significance within the control and very severe groups [$t(37)=1.88$, $p=.067$ and $t(20)=1.75$, $p=.096$ respectively].

An analysis of covariance was carried out to adjust the IQ means to remove the effects of premorbid school progress. For VIQ, the regression coefficient subtracted 13.17 points from the IQ of adequate scholars. The adjusted VIQ means for the controls, moderates, severes and very severes respectively (with the standard deviation in brackets) are 90.6 (11.8), 83.8 (11.7), 83.8 (11.7) and 72.5 (11.7). These adjusted means differ significantly, $F(3,140)=11.48$, $p<.001$. Pairwise comparisons of each head-injured group with the controls find the moderates, severes and very severes all to still have significantly lower VIQs than the controls when the effects of premorbid school progress are removed, [$t(87)=2.72$, $p=.0074$; $t(69)=2.43$, $p=.0163$ and $t(60)=5.87$, $p=.000$, respectively. Note, on three comparisons, p must be less than .0167 to be significant at the overall 5% level].

For PIQ, the regression coefficient subtracted 11.82 points from the adequate scholars, and the groups still differ significantly once the means are adjusted, $F(3,140)=21.08$, $p<.001$. The adjusted means (with the standard deviation in brackets) for the controls, moderates, severes and very severes are 95.1 (14.4), 84.3 (14.4), 88.1 (14.4) and 65.0 (14.4) respectively. Pairwise comparisons

of the controls with the moderates and of the controls with the very severes still find the differences to be significant $t(87)=3.50$, $p<.001$ and $t(59)=7.83$, $p<.001$ respectively; however, the comparison between the controls and the severes just fails to reach statistical significance $t(70)=2.06$, $p=.0413$.

Inspection of the plot of the residuals in the severe group shows one case to differ markedly from the others. This is an extremely bright child with a PIQ 26 points above the next highest PIQ in the group. If this case is removed, the adjusted means become 86.5 (13.8) for the severes compared with 95.1 (13.9) for the controls and this difference is statistically significant, $t(69)=2.62$, $p=.0098$.

The head-injured groups thus remain significantly different from the controls in terms of their Verbal and Performance IQs when differences between the groups in school progress are removed by covariance. This means that the observed differences cannot be attributed to different rates of premorbid scholastic backwardness in the groups. School progress as a factor influencing test results is not investigated again until the Outcome Section (Chapter 14).

10.5 Changes Over Time

The distribution of IQs on the WISC-R for those able to complete the test on all three test occasions is presented in Figure 6 for VIQ and Figure 7 for PIQ.

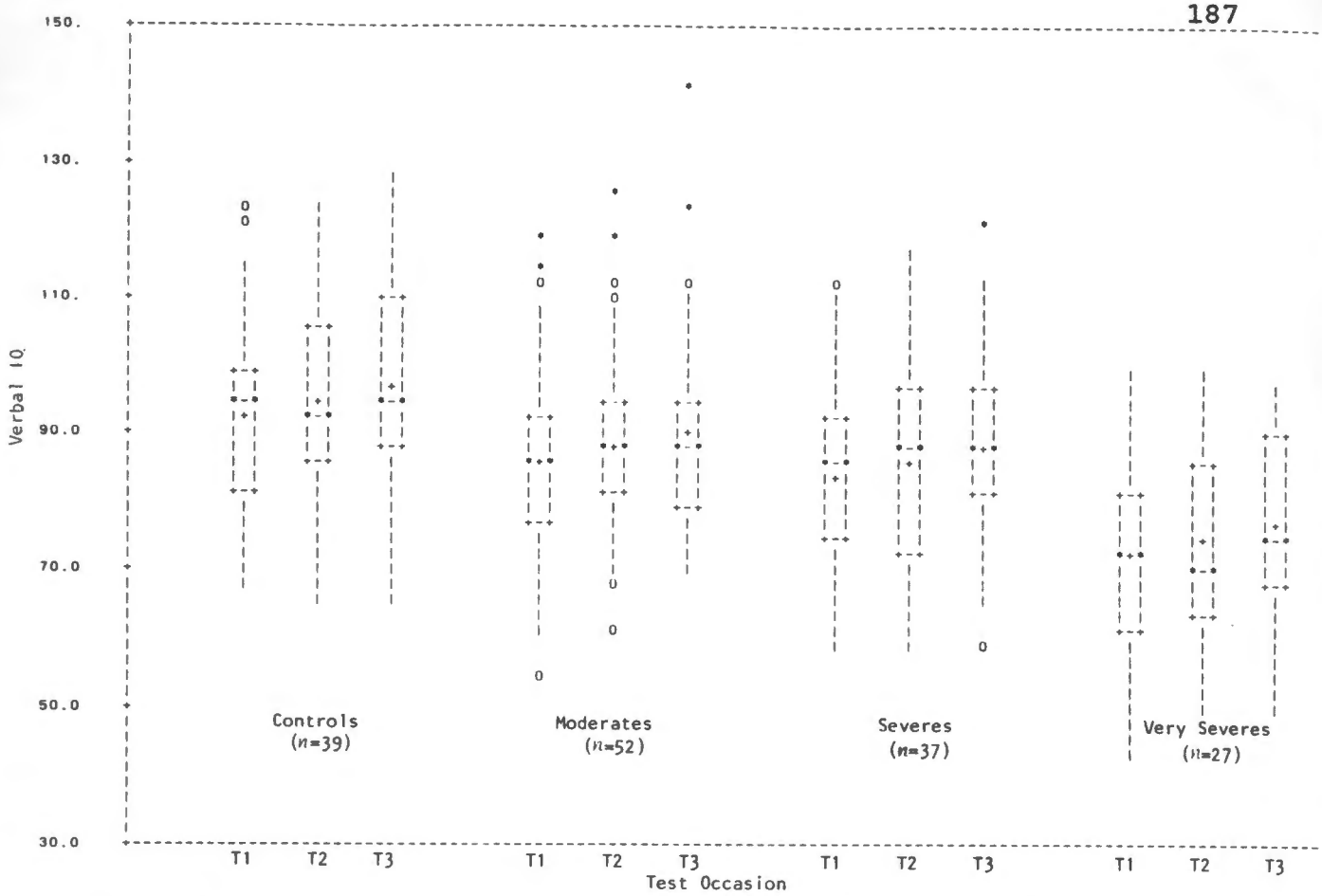


Figure 6. WISC-R Verbal IQs at Successive Test Occasions.

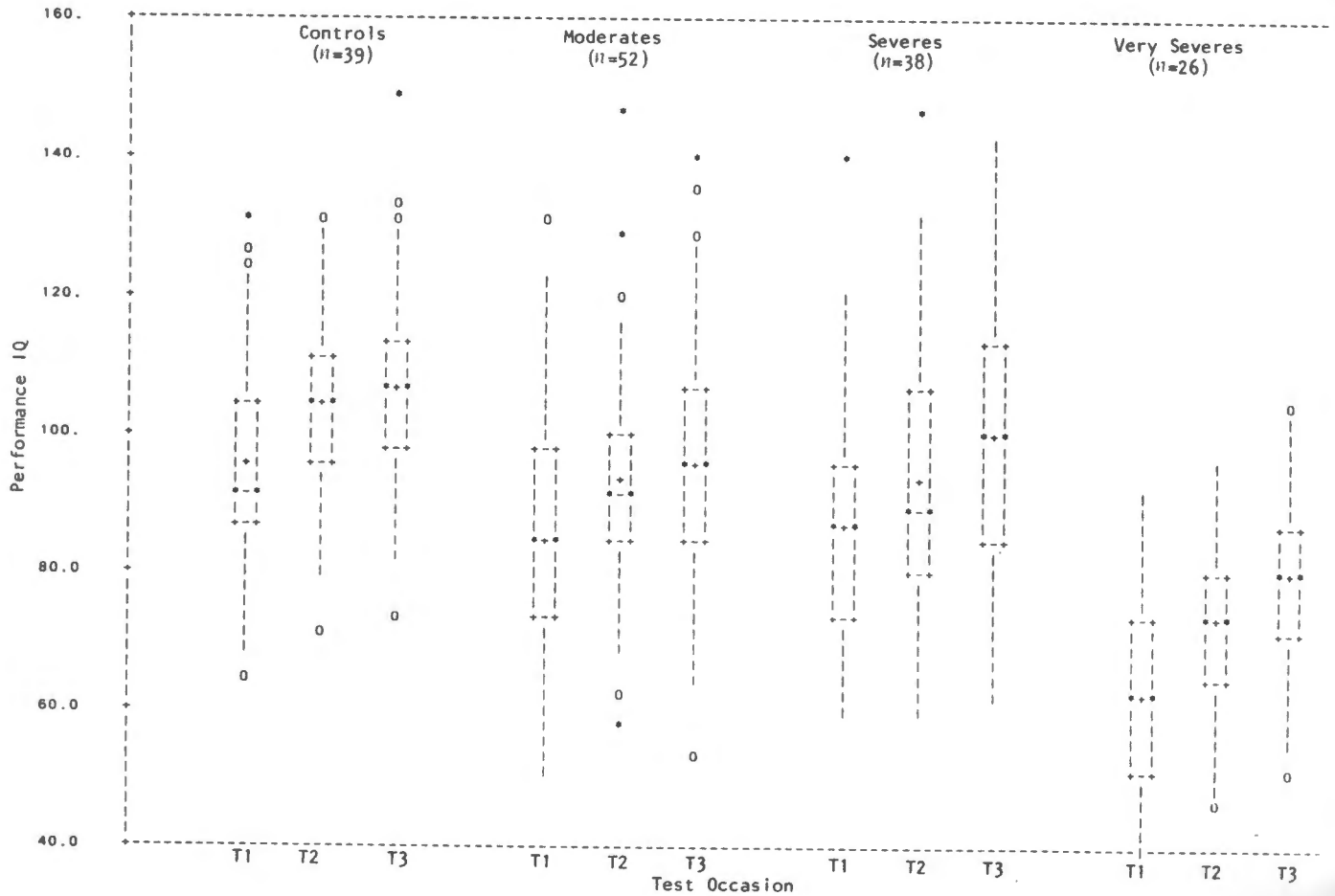


Figure 7. WISC-R Performance IQs at Successive Test Occasions.

Table 20 summarizes the mean size of IQ changes within each group over the two recovery intervals, T1 to T2 and T2 to T3, and the overall significance of these changes over time and between groups on the repeated measures analysis of variance.

Table 20

WISC-R IQs: Mean Changes Over Time

	Groups								Repeated Measures Analysis	
	Controls		Moderates		Severes		Very Severes			
Recovery	(n=39)		(n=52)		(n=37)[a]		(n=27)[b]		Over Time	Between Groups
Interval	M	SD	M	SD	M	SD	M	SD	F(1,151)[c]	F(3,151)
Verbal IQ										
T1 to T2	2.0	6.2	3.8	6.2	2.4	7.5	2.2	7.2	22.21***	0.63
T2 to T3	2.2	6.2	1.6	6.2	1.8	6.6	4.0	7.6	20.83***	0.97
Performance IQ										
T1 to T2	8.3	8.1	8.2	9.2	6.5	7.4	9.7	9.0	134.51***	0.77
T2 to T3	2.9	9.4	2.6	6.8	6.5	9.8	6.2	7.8	42.60	2.32
Full Scale IQ										
T1 to T2	5.4	5.2	6.1	6.7	4.6	6.1	6.0	7.4	111.14***	0.43
T2 to T3	2.6	5.9	2.5	6.1	4.4	6.6	5.8	6.3	54.85***	2.22

[a]n=38 for PIQ, [b]n=26 for PIQ, FSIQ, [c]df=150 for FSIQ.
*** p<.001.

The F values in the Over Time column in Table 20 show that there are significant overall gains on VIQ, PIQ and FSIQ over both recovery intervals. Within each group, the significance of IQ changes in each recovery interval was determined, and the changes in the two intervals compared (see Table C2, p.436 for the paired t-test and p values).

10.5.1 Within Groups

Within the very severe group, there are significant gains on PIQ and FSIQ in the first recovery period and on all three IQs in the second recovery period (Table 20, Table C2). There are no significant differences in the size of gains in the first versus the second recovery period. Figure 8 details the individual changes in PIQ in the very severes over the successive test occasions. It can be seen that there is considerable individual variation. The children starting with PIQ below 50 tend to remain low, although 3 have gains of more than 20 points.

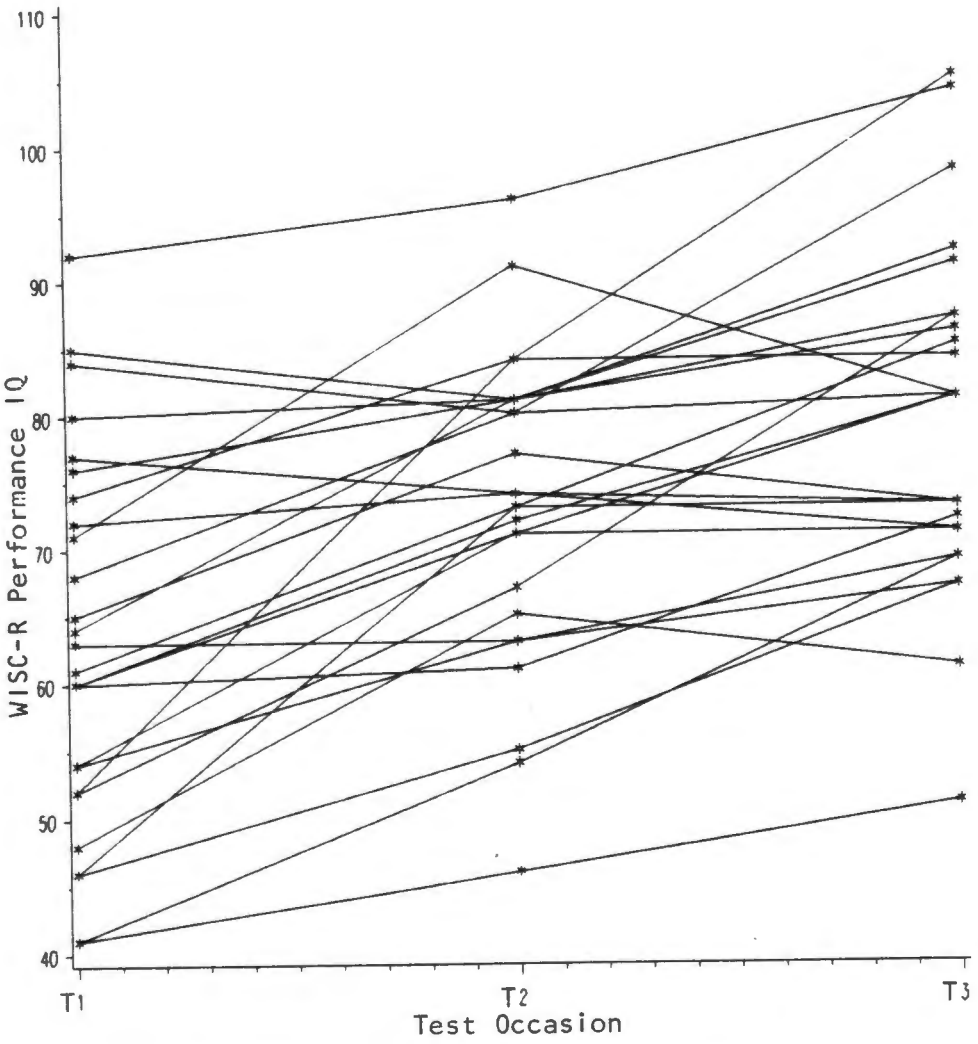


Figure 8. WISC-R PIQs: The Very Severes at Successive Test Occasions.

Within the severe group (Table 20, Table C2), there are significant gains on PIQ and FSIQ in both recovery periods and no significant differences in the size of the gains between the two recovery periods. Gains on VIQ approach significance in both the first and the second recovery periods [$t(36)=1.96$, $p=.058$; and $t(36)=1.85$, $p=.073$, respectively].

Within the moderate group (Table 20, Table C2), there are significant increases on VIQ, PIQ and FSIQ in the first recovery period (T1 to T2) and further significant gains on PIQ and FSIQ in the second recovery period (T2 to T3), while the gain on VIQ approaches significance [$t(51)=1.82$, $p=.074$]. The gains in PIQ and FSIQ are significantly greater in the first than in the second recovery period.

A similar pattern prevails within the control group (Table 20, Table C2). All three IQs increase significantly from T1 to T2 and there are further significant gains in VIQ and FSIQ from T2 to T3, while the gain in PIQ approaches significance, $t(38)=1.95$, $p=.058$. Gains on PIQ and FSIQ are significantly greater in the first than in the second recovery period.

10.5.2 Verbal versus Performance IQ

Paired t-tests were carried out within each group to determine whether there were significant differences in the gains in PIQ versus those in VIQ. In all groups, the gain in PIQ is significantly greater than that in VIQ in the first recovery period, [$t(38)=-3.55$, $p<.001$; $t(51)=-3.63$, $p<.001$; $t(36)=-2.63$, $p=.012$ and $t(25)=-4.53$, $p<.001$, for the controls, moderates, severes and very severes respectively]. There is a trend for PIQ gains to be larger than VIQ gains in the second recovery period, but this is statistically significant only in the severe group, [$t(36)=-2.74$, $p<.01$].

10.5.3 Between the Groups

As is to be seen from the F values in the last column of Table 20, the groups do not differ significantly from each other in the size of gains made in VIQ, PIQ or FSIQ over either recovery interval. However, the F values for the differences between the groups in PIQ and FSIQ gains in the second recovery period approach statistical significance (p=.077 and p=.089, respectively).

The proportion of each group making losses, modest or large gains on PIQ from T1 to T3 is summarized in Table 21 below.

Table 21

WISC-R PIQ: Group Gains and Losses T1 to T3

PIQ Change	Controls (n=41) %	(n)	Moderates (n=53) %	(n)	Severes (n=38) %	(n)	Very Severes (n=26) %	(n)
Loss	**19.5**	(8)	**7.6**	(4)	**2.6**	(1)	**7.7**	(2)
Gain 1-10	**34.2**	(14)	**41.5**	(22)	**34.2**	(13)	**19.2**	(5)
Gain 11-20	**24.4**	(10)	**32.1**	(17)	**42.1**	(16)	**34.6**	(9)
Gain >20	**22.0**	(9)	**18.9**	(10)	**21.1**	(8)	**38.5**	(10)

Table 21 shows that losses in PIQ over the 1 year period were less common in the head injured than in the controls and that the very severes tended to make large gains.

10.6 Persisting Impairment on the WISC-R

The percentage of each group still showing intellectual impairment at 1 year is of interest, as well as the interrelationship between the mean IQs in Table 17 already discussed. Cut-points which are clinically meaningful, but which define no more than 5% of the

controls as impaired, were determined. Table 22 summarizes the cut-points, the percentage of head injured in each group with IQs below these points, and indicates where these percentages significantly exceed the 5% impairment in the control group on either chi-squared or Fisher's exact tests, whichever was appropriate.

Table 22

WISC-R IQs: Percentage With Impairment at 1 Year Post-Injury

IQ	Cut-Point	Controls (n=41) %	(n)	Moderates (n=53) %	(n)	Severe (n=38) %	(n)	Very severes (n=27)[a] %	(n)
VIQ	<75	**4.9**	(2)	**11.3**	(6)	**21.1**	(8)	**40.7*****	(11)
PIQ	<80	**4.9**	(2)	**7.6**	(4)	**10.5**	(4)	**46.2*****	(12)
FSIQ	<80	**4.9**	(2)	**17.0**	(9)	**23.7***	(9)	**61.5*****	(16)

[a] n=26 PIQ, FSIQ.
* Differs significantly from controls at p<.017, *** p<.0003

As is shown in Table 22, a significantly greater proportion of the very severes than of the controls is rated as impaired on all three IQs, [VIQ, $X^2(1, n=68)=13.54$; PIQ, $X^2(1, n=67)=16.4$; and FSIQ, $X^2(1, n=67)=26.0$] and a significantly greater proportion of the severes is impaired on FSIQ, [$X^2(1, n=79)=5.82$; p values as indicated in Table 22]. Other differences are not statistically significant. [Note. For three comparisons, p must be <.017 to be significant at an overall .05 level, <.003 for the .01 level, and <.0003 for the .001 level].

10.7 WISC-R Verbal Subtests

10.7.1 Differences Between the Groups

The means of the Verbal subtests, based on all subjects present at a testing occasion, are presented in Table 23. Multiple group comparisons were carried out using Tukey's

studentized range test and the confidence limits of the differences between the means are given in Table C3, p.437.

Table 23

WISC-R: Verbal Subtests at T1, T2 and T3

	Controls		Head Injured					
			Moderates		Severes		Very severes	
Variable	M	SD	M	SD	M	SD	M	SD
Initial Assessment (T1)								
	(n=46)		(n=55)		(n=39)		(n=28)	
Information	8.3_a	2.7	7.2_a	2.6	7.2_a	3.0	5.3_b	2.8
Comprehension	8.0_a	2.2	7.2_{ab}	2.6	7.3_{ab}	3.3	5.6_b	2.5
Arithmetic	9.3_a	2.9	8.1_a	2.6	8.1_a	3.2	6.2_b	2.5
Digit Span	8.7_a	3.4	7.0_b	3.0	6.9_{bc}	2.7	5.0_c	2.6
Three Months Later (T2)								
	(n=42)		(n=53)		(n=40)		(n=27)	
Information	8.9_a	3.1	7.6_{ab}	2.5	7.1_{bc}	2.8	5.6_c	2.7
Comprehension	8.6_a	2.6	8.0_a	2.7	7.7_a	3.4	5.6_b	1.6
Arithmetic	9.6_a	2.8	8.7_a	2.5	8.3_a	2.7	5.9_b	2.4
Digit Span	8.8_a	3.6	8.0_a	3.1	7.3_{ab}	2.6	5.7_b	3.0
One Year Post-Injury (T3)								
	(n=41)		(n=53)		(n=38)		(n=27)	
Information	9.4_a	3.4	8.3_{ab}	2.4	7.3_{bc}	3.0	5.5_c	2.5
Comprehension	9.2_a	2.4	8.2_a	3.0	8.2_a	3.1	6.4_b	1.8
Arithmetic	9.6_a	2.8	8.9_a	2.8	9.1_a	2.8	6.9_b	2.2
Digit Span	9.5_a	3.6	8.0_{ab}	3.4	7.6_{ab}	2.7	6.8_b	3.3

Note. For each subtest, means not sharing a subscript at a test occasion, differ significantly on Tukey's studentized range test at $p<.05$.

As can be seen from the subscripts in Table 23, the very severes score significantly lower than the controls on all Verbal subtests at all test occasions. There is a consistent trend for their scores to be lower than those of the moderates as well, and differences reach statistical

significance for Information and Arithmetic on all test occasions, for Digit Span at T1 and T2, and for Comprehension at T2 and T3 (close at T1). Their scores also tend to be lower than those of the severes, and differences reach significance for Arithmetic at all three test occasions, Information at T1 (close at T3) and Comprehension at T2 and T3 (close at T1) and approach significance on Digit Span at T1 (see Table C3).

There is a trend for the severes to have lower verbal subtest scores than the controls, but differences reach statistical significance only for Information at T2 and T3, and Digit Span at T1 and approach significance for Digit Span at T3. There are no significant differences between the severes and the moderates at any test occasion.

The moderates also tend to have slightly lower scores than the controls, but differences reach statistical significance only for Digit Span at T1.

10.7.2 Changes Over Time

The repeated measures analysis based only on subjects present on all three testing occasions shows that there are no significant differences between the groups with respect to the amount of change shown on any of the verbal subtests over either recovery interval (F values in the last column of Table 24).

Table 24

WISC-R Verbal Subtests: Mean Changes Over Time

Recovery Interval	Controls (n=39) M	SD	Moderates (n=52) M	SD	Severes (n=37) M	SD	Very Severes (n=27) M	SD	Repeated Measures Analysis: Over Time F(1,151)	Between Groups F(3,151)
					Information					
T1 to T2	0.4	1.7	0.4	2.0	-0.1	1.5	0.3	1.6	3.34	0.68
T2 to T3	0.4	1.5	0.5	1.7	0.1	1.9	-0.1	1.4	3.09	1.19
					Comprehension					
T1 to T2	0.6	1.9	0.8	1.6	0.7	1.9	0.2	2.0	13.41***	0.67
T2 to T3	0.5	1.6	0.2	1.4	0.2	1.7	0.8	1.4	11.95***	1.34
					Arithmetic					
T1 to T2	0.2	1.9	0.7	2.1	0.4	2.7	-0.1	1.9	2.90	0.82
T2 to T3	0.1	1.9	0.2	1.9	0.6	2.2	0.9	2.0	7.00**	1.42
					Digit Span					
T1 to T2	0.1	1.8	0.9	1.9	0.5	2.1	0.8	2.0	12.43***	1.25
T2 to T3	0.5	2.3	0.0	2.2	0.4	1.9	1.1	2.3	7.99**	1.54

**p<.01, ***p<.001

Not all verbal subtests show significant increases over time when the groups are taken together (F values in the second last column of Table 24). There are significant overall gains in both recovery periods (T1 to T2, and T2 to T3) on Comprehension and Digit Span; significant gains in the second recovery period only for Arithmetic, and no significant gains in either recovery interval for Information.

Within each group, paired t-tests were carried out to determine which of the verbal subtest changes shown in Table 24 reach statistical significance. Table C4, p.438 , gives the paired-t values and their probability levels. The very severes have significant gains on Digit Span from T1 to T2, and on Comprehension, Arithmetic and Digit Span from T2 to

T3. The only significant increase in the severe group is on Comprehension in the first recovery interval, while the moderates have significant increases on Comprehension, Arithmetic and Digit Span in the first recovery interval and on Information in the second recovery interval. The controls have a significant increase on the Comprehension subtest in the second recovery period. For none of the groups do the subtest changes in the first recovery interval differ significantly from those in the second recovery interval, although there is a trend within the moderate group for the gains on Comprehension and Digit Span in the first interval to exceed those in the second interval ($p=.071$ and $p=.053$ respectively).

10.7.3 Persisting Impairment

Cut-points for defining persisting impairment on the Verbal subtests were determined according to the criterion that such scores should be exceeded by at least 95% of the controls at T3. Table 25 summarizes the scaled score cut-points, and the number of controls as well as the number of head injured falling below them. Subtests on which the percentage of head injured significantly exceeds the percentage of controls on 2-tailed Fisher's exact tests are indicated.

Table 25

WISC-R Verbal Subtests: Percentage With Impairment at 1 Year Post-Injury

Sub-Test	Scaled Score Cut-point	Controls (n=41) %	(n)	Moderates (n=53) %	(n)	Severes (n=38) %	(n)	V.Severes (n=27) %	(n)
Inform	<4	**2.4**	(1)	**0.0**	(0)	**13.2**	(5)	**22.2***	(6)
Arith	<6	**4.9**	(2)	**9.4**	(5)	**7.9**	(3)	**25.9**	(7)
Comp	<6	**2.4**	(1)	**15.1**	(8)	**18.4**	(7)	**33.3****	(9)
Digit	<5	**4.9**	(2)	**9.4**	(5)	**10.5**	(4)	**18.5**	(5)

* Differs from controls at p<.017, ** p<.003.

A significantly higher proportion of very severes than of controls were rated as having persistent impairment on Information and Comprehension. The percentage of very severes impaired on Arithmetic approaches significance (Fisher's exact p=.024) as does the percentage of severes impaired on Comprehension (Fisher's exact p=.025).

10.8 WISC-R Performance Subtests

10.8.1 Differences Between the Groups

The means of the performance subtests, based on all subjects present at a testing occasion, are summarized in Table 26. Groups not sharing a common letter subscript in Table 26 differ significantly at that test occasion on Tukey's studentized range at p<.05. The confidence limits of the differences between the group means are given in Table C5, p.439.

Table 26

WISC-R: Performance Subtests at T1, T2 and T3

Variable	Controls M	Controls SD	Head Injured: Moderates M	Moderates SD	Severes M	Severes SD	Very severes M	Very severes SD
			Initial Assessment (T1)					
	(n=46)[d]		(n=55)		(n=40)[d]		(n=28)[e]	
Picture Completion	**10.4**$_a$	2.9	**7.9**$_b$	2.9	**8.5**$_b$	3.2	**5.1**$_c$	2.6
Block Design	**9.3**$_a$	3.1	**7.3**$_{bc}$	3.3	**8.0**$_{ab}$	3.1	**5.5**$_c$	3.4
Coding	**8.1**$_a$	2.6	**6.4**$_b$	3.3	**6.4**$_{ab}$	3.2	**2.9**$_c$	2.0
Mazes	**9.4**$_a$	3.0	**9.2**$_a$	3.8	**8.8**$_a$	3.6	**4.3**$_b$	2.4
			Three Months Later (T2)					
	(n=42)		(n=53)		(n=40)[f]		(n=26)[g]	
Picture Completion	**11.3**$_a$	3.3	**8.9**$_b$	3.0	**9.8**$_{ab}$	3.1	**6.4**$_c$	2.5
Block Design	**10.1**$_a$	3.0	**8.5**$_a$	3.2	**8.5**$_a$	3.4	**5.9**$_b$	2.7
Coding	**8.8**$_a$	2.9	**7.8**$_a$	3.1	**7.5**$_a$	3.7	**4.6**$_b$	2.8
Mazes	**11.7**$_a$	3.3	**10.9**$_a$	4.0	**10.4**$_a$	3.9	**6.8**$_b$	3.0
			One Year Post-Injury (T3)					
	(n=41)		(n=53)		(n=38)		(n=26)[g]	
Picture Completion	**11.5**$_a$	3.1	**9.9**$_b$	2.6	**10.6**$_{ab}$	2.9	**7.1**$_c$	2.4
Block Design	**9.9**$_a$	3.7	**8.8**$_{ab}$	3.1	**8.9**$_{ab}$	3.8	**6.9**$_b$	3.1
Coding	**9.9**$_a$	3.0	**8.1**$_b$	3.0	**8.5**$_{ab}$	4.4	**5.4**$_c$	3.3
Mazes	**11.9**$_a$	3.2	**11.2**$_a$	3.6	**11.5**$_a$	3.6	**8.1**$_b$	3.3

Note. For each subtest, means not sharing a subscript at a test occasion differ significantly Tukey's studentized range test at $p<.05$.
[d]n=37 for Coding, [e]n=27 for Block Design and Mazes, n=25 for Coding, [f]n=39 for Coding, [g]n=27 on Picture Completion.

Table 26 shows that there is a consistent trend for the very severes to have lower scores than the other groups on all the performance subtests at all test occasions. These differences reach statistical significance on all comparisons with the controls; on all comparisons with the moderates, except on Block Design at T1 and T3; and on all comparisons with the severes, except on Block Design at T3.

The severes show a trend to lower subtest scores than the controls on all test occasions, but the only difference to reach statistical significance is that for Picture Completion at T1, while that for Coding approaches significance at T1 (Table C5).

The moderates are significantly lower than the controls on Picture Completion on all test occasions; on Coding at T1 and T3; and on Block Design at T1, while difference at T2 approaches significance (Table C5).

10.8.2 Changes Over Time

Table 27

WISC-R Performance Subtests: Mean Changes Over Time

Recovery Interval	Groups: Controls (n=39)[a] M	SD	Moderates (n=52) M	SD	Severes (n=38)[b] M	SD	Very Severes (n=27)[c] M	SD	Repeated Measures Analysis: Over Time F(1,152)[d]	Between Groups F(3,152)[d]
Picture Completion										
T1 to T2	1.1	2.2	0.8	2.0	1.2	1.8	1.5	3.0	40.87***	0.53
T2 to T3	0.3	2.2	1.0	1.9	0.9	2.2	0.7	2.0	17.49***	0.97
Block Design										
T1 to T2	0.7	1.9	1.1	2.1	0.5	2.7	0.5	2.4	14.64***	0.71
T2 to T3	-0.1	1.9	0.3	1.9	0.3	2.4	1.0	2.1	4.89*	1.26
Coding										
T1 to T2	0.5	2.2	1.3	2.1	0.9	1.8	1.7	2.1	39.98***	1.98
T2 to T3	1.2	2.3	0.1	2.2	1.2	2.0	0.9	2.6	18.14***	2.07
Mazes										
T1 to T2	2.4	3.3	1.6	3.3	1.3	3.1	2.6	2.4	58.30***	1.49
T2 to T2	0.4	3.6	0.3	2.6	1.3	3.3	1.3	2.6	11.16**	1.22

* $p<.05$, ** $p<.01$, *** $p<.001$.

[a] n=31 on Coding; [b] n=36 on Coding; [c] n=26 on Block Design and Mazes, n=24 on Coding.

[d] df=151 for Block Design and Mazes, df=139 for Coding.

The repeated measures analysis of changes in Performance subtest scores (Table 21) shows that all the subtests have significant overall gains over time for both recovery intervals. Differences between the groups with respect to the amount of change are not significant, however, for either recovery interval.

Within each group, paired t-tests were carried out to determine which changes in Table 27 are significant. Table C6, p.440 summarizes the paired-t values and their probability levels. In the first recovery interval (T1 to T2), the very severes have significant gains on Picture Completion, Coding and Mazes, while in the second recovery interval (T2 to T3), gains on Block Design and Mazes are significant and those on Picture Completion approach significance (p=.098). There are no significant differences in the size of gains on any subtest in the first versus the second recovery interval.

Within the severe group, there are significant increases on Picture Completion, Coding and Mazes in both recovery intervals, but no significant differences in the size of gains over the two recovery intervals.

Within the moderate group, the increases on all four performance subtests are significant in the first recovery interval, but only Picture Completion shows a significant gain on the second recovery interval. The increase in Coding is significantly greater in the first than in the second recovery interval, and there is a trend for the gains on Block Design and Mazes in the first interval also to exceed those in the second interval (p=.094 and p=.081, respectively).

Within the control group, significant increases occur on Picture Completion, Block Design and Mazes in the first recovery period, and on Coding in the second recovery period. The gain on Mazes is significantly greater from T1 to T2 than from T2 to T3 and the gain on Block Design from T1 to T2 almost reaches significance in exceeding the slight loss from T2 to T3 (p=.066).

10.8.3 Persisting Impairment

Table 28 presents the scaled score cut-points on the Performance subtests exceeded by at least 95% of the controls at T3. (No controls scored below 6 on Coding, but 5 [12%] scored at 6). Table 28 indicates on which subtests the percentage of head injured falling below the cut-points significantly exceeds the percentage of controls falling below. The significance of differences between each of the head-injured groups and the controls was determined on chi-squared or 2-tailed Fisher's exact tests, as appropriate.

Table 28

WISC-R Performance Subtests: Percentage Impairment at 1 Year Post-Injury

Sub-Test	Scaled Score Cut-point	Controls (n=41) %	(n)	Moderates (n=53) %	(n)	Severe (n=38) %	(n)	V.Severes (n=26)[a] %	(n)
P.Comp	<7	**4.9**	(2)	**7.6**	(4)	**7.9**	(3)	**33.3****	(9)
B.Des	<4	**4.9**	(2)	**3.8**	(2)	**2.6**	(1)	**15.4**	(4)
Coding	<6	**0.0**	(0)	**17.0***	(9)	**21.1****	(8)	**50.0*****	(13)
Mazes	<7	**4.9**	(2)	**11.3**	(6)	**10.5**	(4)	**34.6****	(9)

[a]n=27 on Picture Completion.
* Differs from controls at p<.017, ** p<.003, *** p<.0003.

A significantly higher percentage of very severes than of controls is rated as impaired on the Picture Completion and Mazes subtests (Fisher's exact p as indicated in Table 28) and on Coding [$X^2(1, n=67)=25.4$]. The percentages rated as impaired on Coding are also significantly higher in the severes and moderates than in the controls (Fisher's exact).

10.9 Summary

1. The very severes score significantly lower on both the Verbal and Performance scales of the WISC-R than the controls and the other head-injured groups at all test occasions. At the 1 year follow-up, 61% have a FSIQ below 80, in contrast to 5% of the controls.

2. The moderates and severes do not differ significantly from each other on VIQ, PIQ or any subtest at any test occasion. At the initial assessment, both groups have significantly lower VIQ, PIQ and FSIQ than the controls. At T2, PIQ and FSIQ remain lower in both groups, while VIQ (particularly Information) is also lower in the severes. At 1 year, PIQ (Coding and Picture Completion) is significantly lower in the moderates, while VIQ (Information) is still significantly lower in the severes than in the controls. If the moderates and severes are combined to form a single group with PTA less than 8 days (comparable to the mildly head-injured in the Chadwick-Rutter study, 1981), then VIQ, PIQ and FSIQ are all significantly lower in this combined group than in the control group at 1 year. FSIQ is below 80 in 17% of moderates and 24% of severes at 1 year.

3. The controls, as expected in a predominantly lower socioeconomic sample, have intake IQs slightly below the WISC-R mean of 100, and PIQ is significantly greater than VIQ.

4. Premorbid scholastic backwardness does lower VIQ and PIQ significantly, but differences between the controls and head-injured groups at T1 remain significant when the IQs are adjusted for scholastic level by analysis of covariance.

5. PIQ versus VIQ. PIQ appears more affected by head injury than VIQ initially. The pattern of PIQ greater than VIQ seen in the control group is absent in the moderates and severes at T1 and is reversed in the very severes. Gains in PIQ tend to be greater than those in VIQ, and are significantly greater within all groups in the first recovery interval. By T2, PIQ is greater than VIQ in the moderates and severes, and equal to VIQ in the very severes.

6. The improvement in intellectual functioning is of the same magnitude in all groups and recovery is not significantly greater in those with the most severe initial deficit. PIQ (and FSIQ) improves significantly in all groups from T1 to T2, whereas VIQ improves significantly in the controls and moderates, (approaches significance in the severes) but does not improve significantly in the very severes until the second recovery period. In the second recovery period, all groups again have IQ gains which either are significant or approach statistical significance. For the controls and the moderates, gains in the first 3 months tend to exceed those in the remainder of the year, while for the severes and the very severes, recovery is more even over the two intervals.

7. Persisting impairment was defined as a score below that of 95% of controls at 1 year. In the very severe group, VIQ and PIQ are impaired in just over 40%. Subtests most sensitive to persisting impairment in the very severes are Information, Comprehension, Picture Completion, Mazes and Coding. Only on the Coding subtest do a significant percentage of the moderates (17%) and of the severes (21%) have persisting impairment.

11. LANGUAGE RESULTS

11.1 Language Production Measures

11.1.1 Removal of Age, English/Afrikaans, SES and Sex Effects

1. Word fluency.

Analysis of covariance could not be used to adjust raw scores for age as the effects of age differed in the groups. Local norms based on both language groups (Appendix B1, p.433), were therefore used to transform scores into a percentage of the expected score for age. Multiple regression found no significant effects for sex or SES in the scores of the control group at T1.

2. Renfrew Picture Naming.

At T1, scores in the control group were significantly associated with age, language usage (English=1, Afrikaans=0) and SES (range, 1=professional to 6=unemployed, see p.159), but not with sex. Using age, language and SES as covariates, pooled estimates of the coefficients were determined and all cases were adjusted to Afrikaans language (0), SES 4 (unskilled labourer) and age 114, 117 or 126 months (ie 9.5, 9.75 or 10.5 years) as follows:

At T1, Renfrew Adjusted =
Renfrew + 0.261(114-age) + 5.853(0 -language) -1.847(4-SES).

At T2, Renfrew Adjusted =
Renfrew + 0.248(117-age) + 6.108(0 -language) -1.426(4-SES).

At T3, Renfrew Adjusted =
Renfrew + 0.225(126-age) + 3.938(0 -language) -2.671-SES).

(Four outlying cases were excluded in determining the coefficients at T3). For the repeated measures analysis of variance, results on all occasions were adjusted to age 126 months, using these equations.

3. Speed of Naming

At T1, scores in the control group were significantly associated with age and language, but not with sex or SES. Using age and language as covariates (and excluding 4 outlying cases at T1 and 2 cases at T2), pooled estimates of the coefficients were obtained and used to adjust all cases to Afrikaans language (0) and age 114, 117 or 126 months as follows:

At T1, Speed Adjusted =
Time in seconds - 0.0063(114 - age) - 0.2676(0 - language).

At T2, Speed Adjusted =
Time in seconds - 0.0052(117 - age) - 0.2598(0 - language).

At T3, Speed Adjusted =
Time in seconds - 0.0045(126 - age) - 0.1952(0 - language).

For the analysis of changes over time, all results were adjusted to age 126 months using these equations.

11.1.2 Differences Between the Groups

Measures of central tendency and dispersion for the groups on the three production measures are summarized in Table 29. For Word Fluency and Renfrew Naming, multiple pairwise comparisons were carried out using Tukey's Studentized range test. Confidence limits of the mean differences between the groups are given in Table C7, p.441. On Speed of Naming, a few extreme cases in the very severe group at T1 caused the mean of that group to lie above the 75th percentile (M=2.27, SD=1.51) and the nonparametric Wilcoxon rank sum test was therefore used to compare the groups (values in Table C7, p.441).

Table 29

Language Production Measures at T1, T2 and T3

Test Occasion	Controls		Head Injured					
			Moderates		Severes		Very Severes	
	Word Fluency: Percentage of expected score							
	M	SD	M	SD	M	SD	M	SD
	(n=46)		(n=55)		(n=38)		(n=28)	
T1	**91.5$_a$**	25.1	**82.4$_{ab}$**	35.5	**73.7$_b$**	32.7	**49.8$_c$**	24.0
	(n=42)		(n=53)		(n=40)		(n=27)	
T2	**92.9$_a$**	27.1	**84.9$_a$**	28.4	**80.8$_a$**	33.6	**62.3$_b$**	20.6
	(n=40)		(n=53)		(n=38)		(n=27)	
T3	**91.7$_a$**	22.1	**86.1$_a$**	26.0	**86.0$_a$**	30.9	**66.2$_b$**	22.6
	Renfrew Naming (Number correct)							
	M	SD	M	SD	M	SD	M	SD
	(n=46)		(n=54)		(n=39)		(n=28)	
T1	**34.4$_a$**	4.5	**31.3$_{ab}$**	6.0	**30.8$_b$**	6.6	**25.1$_c$**	8.3
	(n=42)		(n=53)		(n=40)		(n=27)	
T2	**37.2$_a$**	4.7	**34.3$_{ab}$**	6.2	**33.5$_b$**	6.7	**29.0$_c$**	6.7
	(n=41)		(n=53)		(n=38)		(n=27)	
T3	**40.6$_a$**	4.7	**37.2$_b$**	6.0	**36.5$_{bc}$**	6.8	**32.7$_c$**	6.8
	Speed of naming (seconds)							
	Median	25th 75th	**Median**	25th 75th	**Median**	25th 75th	**Median**	25th 75th
	(n=46)		(n=54)		(n=39)		(n=28)	
T1	**1.35$_a$**	1.16 1.51	**1.38$_a$**	1.24 1.62	**1.42$_a$**	1.22 1.59	**1.76$_b$**	1.42 2.20
	(n=42)		(n=52)		(n=40)		(n=27)	
T2	**1.19$_a$**	1.10 1.48	**1.27$_a$**	1.13 1.38	**1.26$_a$**	1.0 1.57	**1.48$_b$**	1.29 1.87
	(n=41)		(n=53)		(n=38)		(n=27)	
T3	**1.15$_a$**	1.06 1.26	**1.17$_a$**	1.0 1.37	**1.16$_a$**	1.04 1.24	**1.24$_a$**	1.09 1.54

Note. Groups not sharing a subscript at a test occasion differ significantly on Tukey's studentized range or, for Speed of Naming, the Wilcoxon at $p<.05$.

As is shown in Table 29, the general trend of scores on the production measures is in the expected direction, although the differences between the moderates and the severes are often very small and are not significant. The very severes differ significantly from all other groups on Word Fluency at all test occasions and on Speed of Naming at T1 and T2, but not at T3, where none of the groups differ

significantly [Kruskal Wallis $X^2(3, n=159)=2.93, p>.10$]. On Renfrew Naming the scores of the very severes are significantly below those of the controls and the moderates at all test occasions, and below those of the severes at T1 and T2, while the difference approaches statistical significance at T3 [3.76, confidence limits -0.19 to 7.71].

The severes name significantly fewer Renfrew pictures than the controls throughout and have a trend towards taking longer to find the names, but this is not significant at any test occasion. Their Word Fluency is significantly poorer than that of the controls at T1, but this difference is no longer significant at T2 and T3.

Although the moderates tend to have lower production scores than the controls throughout, differences reach statistical significance only on the Renfrew Picture Naming at T3, but are very close to significance at T1 [3.2, confidence limits -0.06 to 6.43] and close at T2 [2.9, confidence limits -0.40 to 6.11].

In terms of the level of language production achieved by the control group, the mean number of words produced in 1 minute is 92% of what is expected for age at T1, but the number of Renfrew Pictures named (M=34.5) is well below that achieved by British 8 year-olds (M=50, Renfrew, 1968).

11.1.3 Changes Over Time

Table 30 summarizes the size of changes on the language production measures over the successive test occasions. The significance of changes in the mean percentage scores on Word Fluency and in the mean number of pictures named on Renfrew Naming, was determined by repeated measures analysis of variance. The significance of changes between the groups in the median number of seconds taken to name the first 10 Renfrew pictures was determined on the Kruskal Wallis X^2.

Table 30

Language Production Measures: Changes Over Time

Recovery Interval	Group: Controls (n=39)[a]		Moderates (n=52)[b]		Severes (n=37)[c]		Very Severes (n=27)		Repeated Measures Analysis: Over Time	Repeated Measures Analysis: Between Groups
	Word Fluency: Changes in Percentage Scores									
	M	SD	M	SD	M	SD	M	SD	F(1,149)	F(3,149)
T1 to T2	1.53	17.3	2.07	27.8	8.25	27.2	14.09	21.3	10.30**	1.96
T2 to T3	-2.30	22.7	-0.15	27.8	4.30	34.5	3.96	24.1	0.40	0.48
	Renfrew Picture Naming: Changes in Number Named								F(1,150)	F(3,150)
T1 to T2	1.89	2.9	2.05	3.0	1.58	3.4	2.84	4.0	60.01***	0.81
T2 to T3	0.74	3.6	0.22	3.4	0.99	3.2	1.51	3.7	9.37**	0.90
	Speed of Naming: Changes in Time Taken (seconds)									Kruskal Wallis $X^2(3,n=153)$
	Median		Median		Median		Median			
T1 to T2	-0.04		-0.11		-0.09		-0.24			4.52
T2 to T3	0.02		-0.09		-0.11		-0.24			7.10

[a]n=38 on Word Fluency, [b]n=51 on Renfrew Naming, n=50 on Speed, [c]n=36 on Word Fluency.
** $p<.01$, *** $p<.001$.

The head-injured and control groups do not differ significantly from each other in the amount of change that occurs in any of the three language production measures over either recovery interval (Between Groups column, Table 30), although differences between the groups in the amount of change from T2 to T3 in Speed of Naming approach significance (p=.069) on the Kruskal Wallis). Overall, there are significant gains over both recovery intervals on the repeated measures analysis of variance of Word Fluency and Renfrew Naming (Over Time column, Table 30). The distribution of scores and the within group changes are presented below for each language production measure.

11.1.3.1 Word Fluency

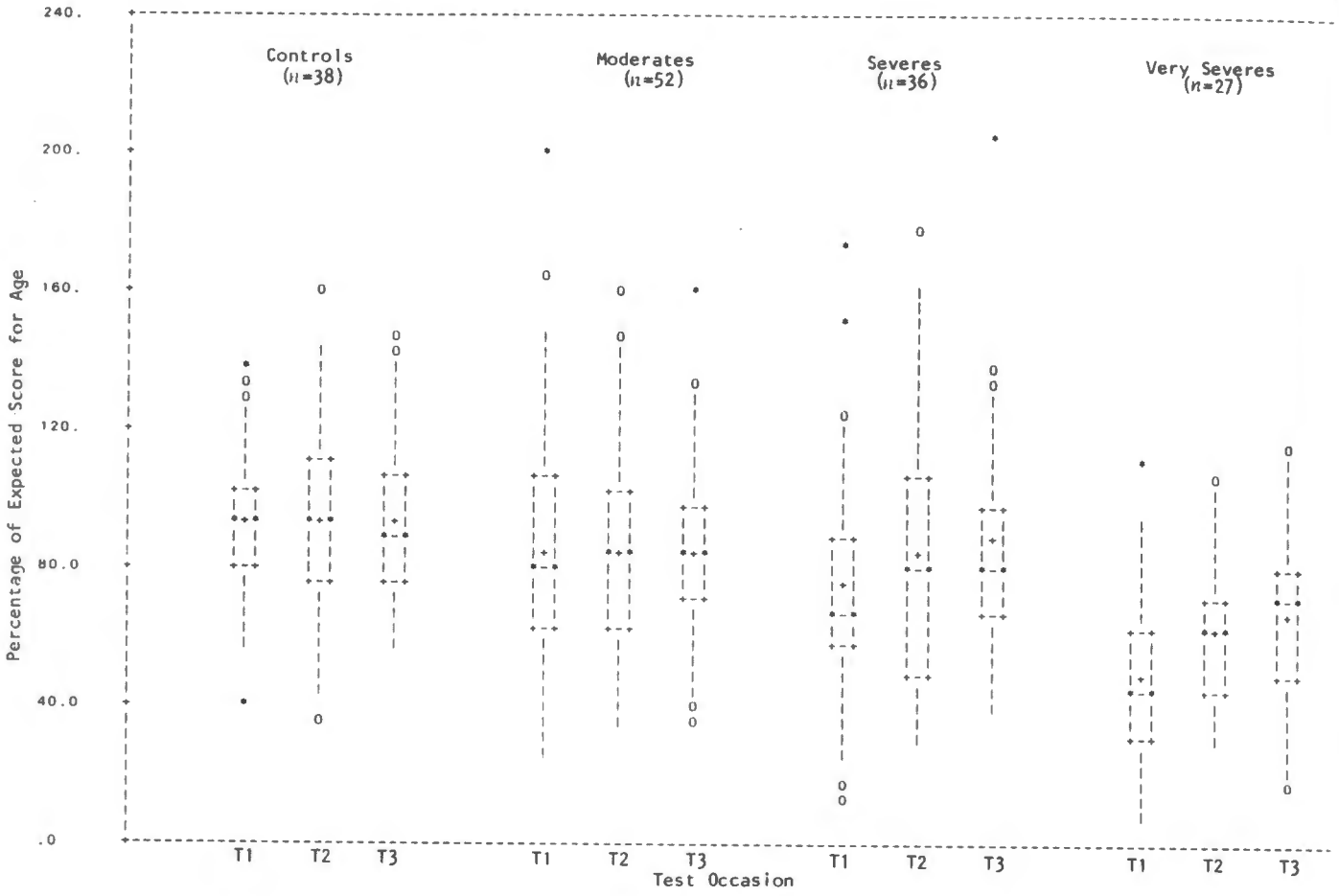


Figure 9. Word Fluency: Percentage Scores at Successive Test Occasions.

The distribution of the scores of those present on all three occasions is depicted in Figure 9 above. The significance of changes within the groups over the two recovery intervals was determined on paired t-tests (values in Table C8, p.442). In the first recovery period, changes are significant for the very severes only, and approach significance for the severes [t(35)=1.82, p=.078]. Changes in the second recovery period are not significant within any of the groups. The gains made in the first (T1 to T2) as opposed to the second (T2 to T3) recovery interval do not differ significantly within any of the groups.

11.1.3.2 Renfrew Naming

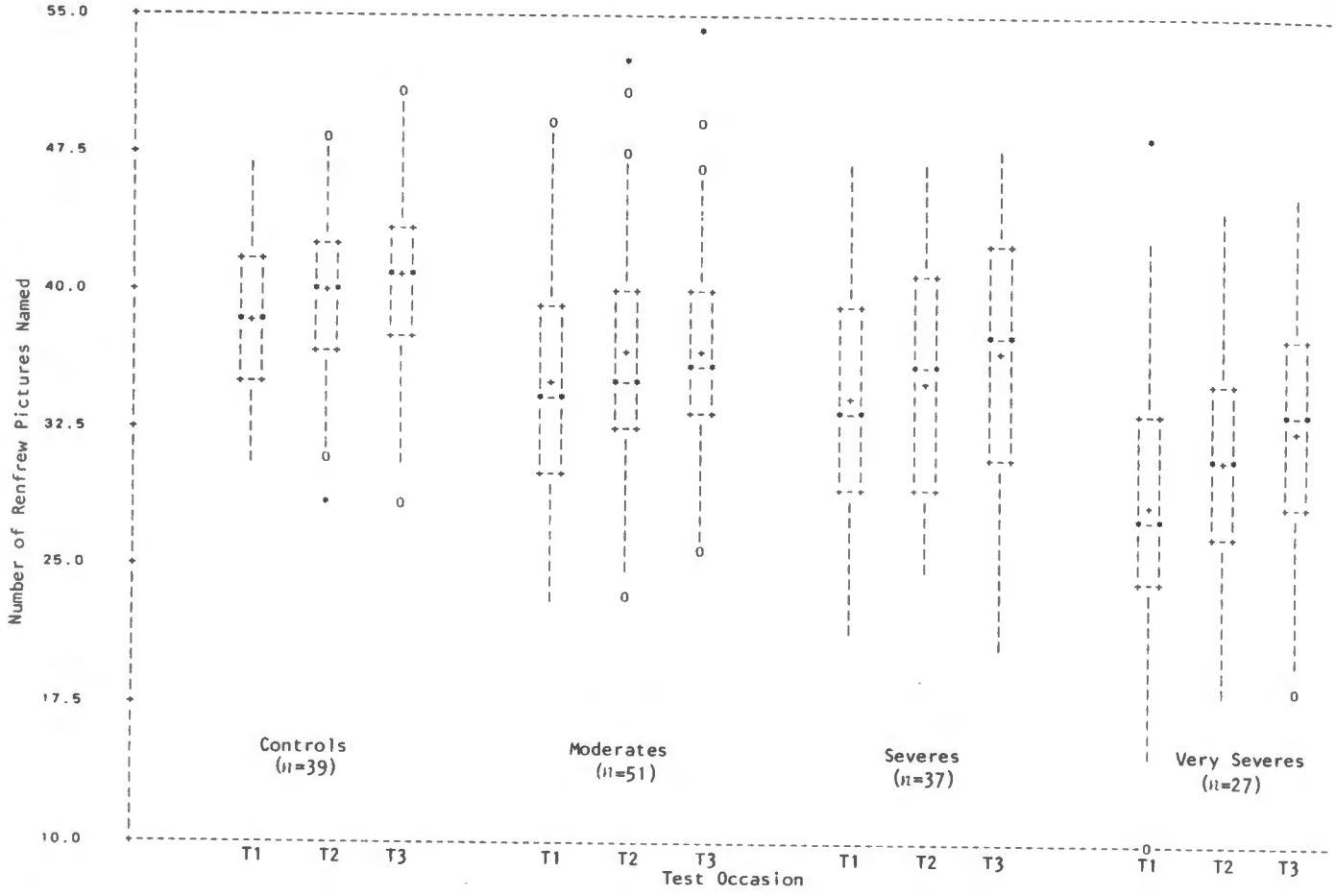


Figure 10. Renfrew Picture Naming: Adjusted Scores at Successive Test Occasions

The distribution of scores for the groups on the successive test occasions is depicted in Figure 10, with all scores adjusted to age 10.5 years. All groups show significant gains from T1 to T2 in the number of pictures named (see Table 30 for mean gains and Table C8, p.442 for paired t-values). The very severes show a significant gain in the second recovery interval as well, while the gain for the severes approaches statistical significance [t(36)=1.91, p=.064]. Although all groups appear to show greater gains in the first than in the second test interval (Table 30), this difference reaches significance only in the moderate group.

11.1.3.3 Speed of Naming

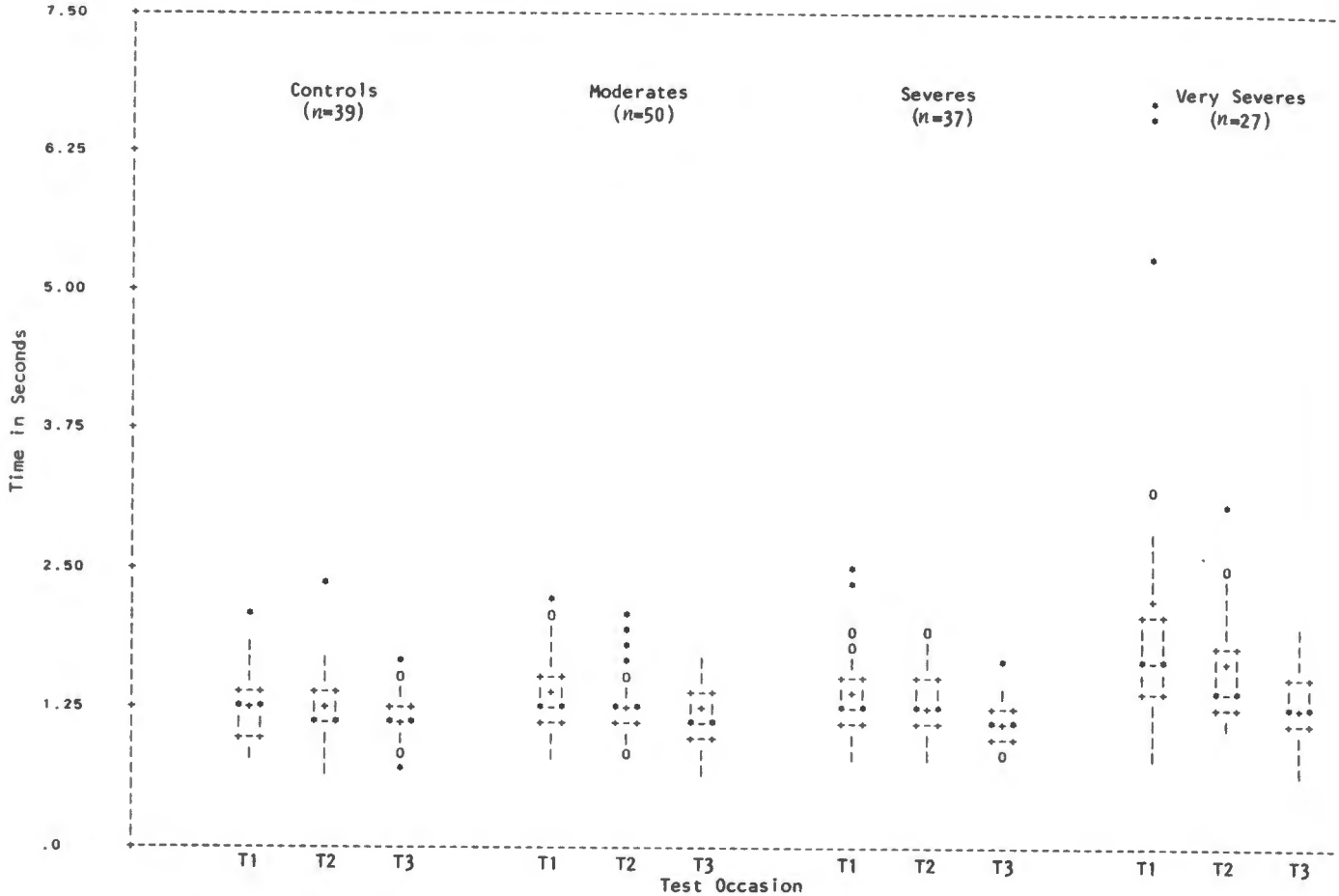


Figure 11. Speed of Naming: Time in Seconds at Successive Test Occasions

Figure 11 shows the distribution of the average length of time (in seconds) taken to name the first 10 Renfrew pictures over the successive test occasions, when all scores are adjusted to an age of 10.5 years. The significance of changes within the groups in the median length of time taken from T1 to T2 and from T2 to T3 was determined by the sign rank test (values in Table C8, p.442). The decrease in time taken at T2 in comparison with T1 is significant for the very severes and the moderates, and approaches significance in the severes (p=.070). There is a further significant decrease from T2 to T3 in the very severes and the severes. The decreases in time taken are very similar for the two recovery intervals and none of the comparisons between the intervals are significant within the groups.

11.1.3.4 Summary

In summary then, the very severes show significant gains on all three language production measures in the first recovery period and in the number of Renfrew Pictures named and Speed of Naming in the second recovery period as well. The severes have gains in the first recovery period that are significant on Renfrew Naming and just short of significance on Word Fluency and Speed of Naming, and gains in the second interval that are significant on Speed of Naming and approach significance on the Renfrew Naming. The moderates have significant gains on Renfrew Naming and Speed of Naming in the first recovery interval, but no further significant gains, and the controls have significant gains only in the number of Renfrew pictures named from T1 to T2. Yet, between the groups, none of these changes differ sufficiently to reach statistical significance.

11.1.4 Persisting Impairment on Language Production Measures

Impairment on language production measures at 1 year was defined as a score lower (or slower) than that obtained by 95% of the controls at T3. Table 31 summarizes the 5th percentile cut-points and the percentage of each group falling below them. The significance of differences between the controls and the head-injured groups was determined on chi-squared or Fisher's exact tests, as appropriate.

Table 31

Language Production Measures: Percentage with Impairment at 1 Year Post-Injury

Variable	Cut-point 5th percentile	Controls (*n*=41)^ %	(*n*)	Moderates (*n*=53) %	(*n*)	Severes (*n*=38) %	(*n*)	Very Severes (*n*=27) %	(*n*)
Word Fluency	<63.9%	5.0	(2)	18.9	(10)	21.1	(8)	37.0**	(10)
Renfrew Naming	<32	4.9	(2)	18.9	(10)	29.0*	(11)	48.2***	(13)
Speed of Naming	>1.63"	4.9	(2)	3.8	(2)	2.6	(1)	22.2	(6)

* Differ from controls at $p<.017$, ** $p<.003$, *** $p<.0003$.
^n=40 on Word Fluency.

As is shown in Table 31, a significantly greater percentage of very severes than of controls is rated impaired on Word Fluency and Renfrew Naming [Fisher's exact p=.002, and $X^2(1, n=68)=17.73$, p<.0001 respectively], and a significantly greater percentage of severes than of controls is impaired on Renfrew Naming [$X^2(1, n=79)=8.31$, p<.003]. It should be noted that it is not necessarily the same children who are impaired on the different language production measures in Table 31. Only 5% or less of the control, moderate and severe groups are impaired on more than one of these measures, whereas 33% of the very severes are impaired on more than one measure.

11.2 Language Repetition

11.2.1 Digit Repetition

Control group scores at T1 were significantly affected by age, but not by language, sex or SES. Adjustment by analysis of covariance could not be done as the effects of age were different in the various groups. Raw scores were

therefore analysed as a percentage of the expected score for age (see Method, p.169 and p.172). Table 32 summarizes the median number of digits forward repeated and the median percentage score for each group.

Table 32

Digit Repetition: Median Raw and Percentage Scores at T1, T2 and T3

	Controls	Head Injured		
Score		Moderates	Severes	Very Severes
	Initial Assessment (T1)			
	(n=46)	(n=55)	(n=39)	(n=28)
Digits forward	5	5	4	4
Percentage score	**91.7**	**83.3**	**80**	**80**
	3 Months Later (T2)			
	(n=42)	(n=53)	(n=40)	(n=27)
Digits forward	5	5	5	4
Percentage score	**100**	**83.3**	**83.3**	**80**
	1 Year Post-Injury (T3)			
	(n=41)	(n=53)	(n=38)	(n=27)
Digits forward	5	5	4.5	4
Percentage score	**100**	**83.3**	**81.7**	**80**

Although scores in Table 32 are in the direction expected according to severity of injury, none of the differences are statistically significant (median test X^2 values at T1, T2 and T3, df=3 and n=168, 162 and 159, are 5.70, 3.23 and 6.96 respectively, $p>.05$). When impairment on digit repetition at 1 year is defined as a score below that obtained by 95% of controls, there is poor differentiation of the groups, with scores below 60% of that expected for age occurring in 2 controls, 2 moderates (4%), 1 severe (3%) and 4 very severes (15%).

11.2.2 Sentence Repetition

11.2.2.1 Age, Language, SES and Sex Effects

Raw scores were transformed into age ratios using the Detroit norms (Baker & Leland, 1967). (Adjustment for age only was needed as the control group raw scores at T1 were not significantly affected by language, sex or SES).

11.2.2.2 Differences Between the Groups

Table 33

Sentence Repetition: Age Ratios at T1, T2 and T3

	Controls		Head Injured					
			Moderates		Severes		Very Severes	
Test Occasion	M	SD	M	SD	M	SD	M	SD
	(n=44)		(n=55)		(n=38)		(n=27)	
T1	**80.3$_a$**	27.8	**75.4$_a$**	29.6	**71.9$_a$**	20.3	**55.2$_b$**	13.2
	(n=42)		(n=53)		(n=40)		(n=27)	
T2	**79.2$_a$**	28.9	**78.0$_a$**	28.9	**74.0$_{ab}$**	22.5	**59.8$_b$**	11.5
	(n=41)		(n=53)		(n=38)		(n=27)	
T3	**79.2$_a$**	26.8	**77.2$_a$**	26.6	**75.0$_{ab}$**	25.8	**59.5$_b$**	12.5

Note. Groups not sharing a subscript at a test occasion differ significantly on Tukey's studentized range at $p<.05$.

As is shown in Table 33, only the very severes differ significantly from the controls on Tukey's studentized range test (confidence limits in Table C9, p. 442). At T1 the scores of the very severes are also lower than those of the moderates and the severes. At T2 and T3, their scores are still significantly lower than those of the controls and the moderates, but no longer significantly lower than those of the severes (although at T3 the difference, 15.5, confidence limits -0.67 to 31.6, approaches significance).

Scores for the severes and moderates are in the expected direction, but none of the differences reach significance. The low age ratio scores of the controls may reflect the deprivation in the sample, or suggest that the content of the sentences was culturally inappropriate.

11.2.2.3 Changes Over Time

The distribution of scores on Sentence Repetition for children who completed the test on all three occasions is depicted in Figure 12. The size and significance of changes over the two recovery intervals is summarized in Table 34.

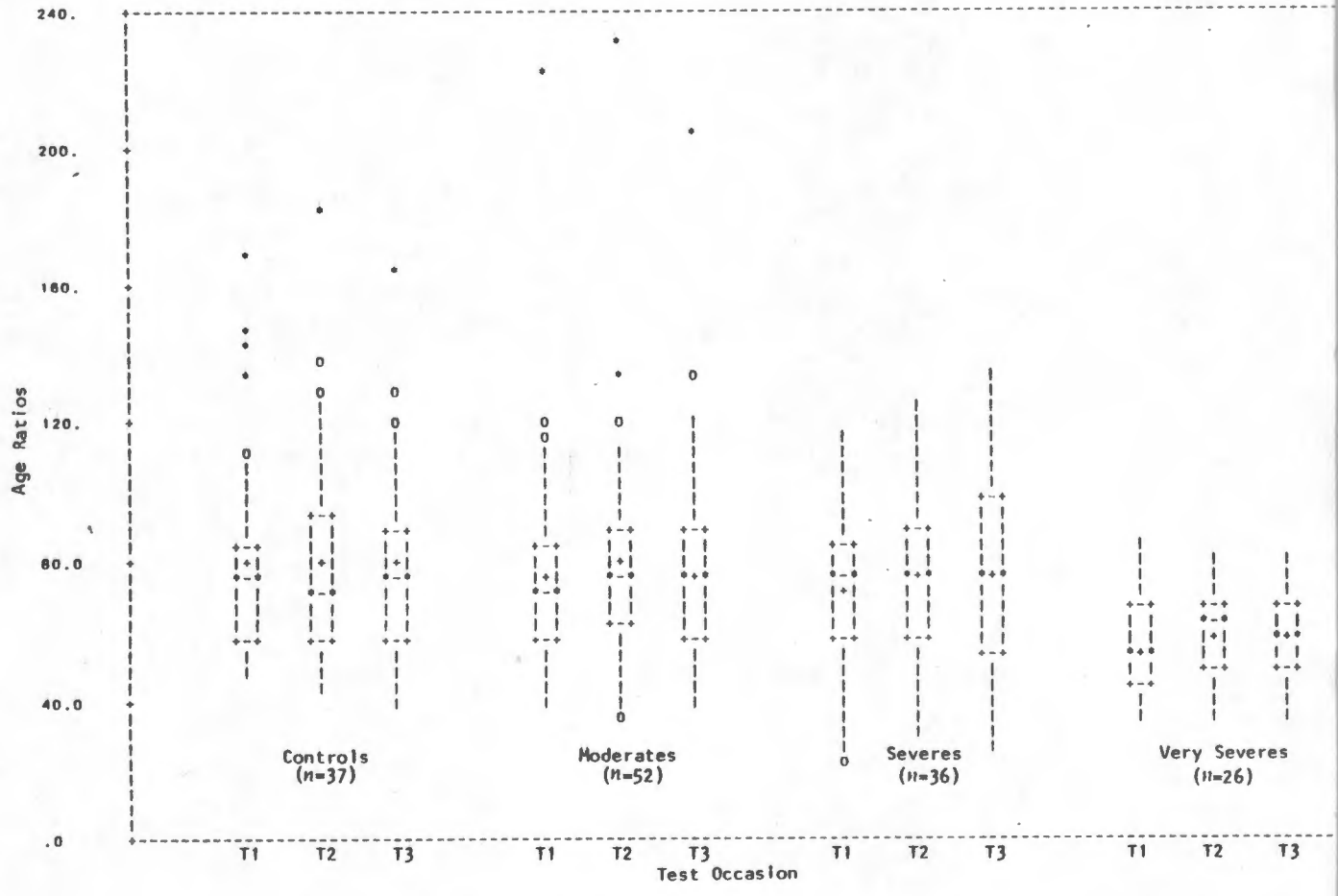


Figure 12. Sentence Repetition: Age Ratios at Successive Test Occasions

Table 34

Sentence Repetition Age Ratios: Mean Changes Over Time

Recovery Interval	Group: Controls (n=37) M	SD	Moderates (n=52) M	SD	Severes (n=36) M	SD	Very Severes (n=26) M	SD	Repeated Measures Analysis: Over Time F(1,147)	Between Groups F(3,147)
T1 to T2	-2.30	15.6	1.61	11.4	2.80	9.2	4.29	8.1	2.68	1.96
T2 to T3	-0.73	11.9	-0.99	9.9	0.84	8.2	-0.19	7.2	0.11	0.28

Inspection of Table 34 and Figure 12 suggests that the only change of note is in the very severe group from T1 to T2. Repeated measures analysis shows there to be no significant overall change over time in either recovery interval and no significant differences between the groups in the size of changes. Within the groups, the only gain to reach statistical significance on paired t-tests (Table C10, p.443) is that for the very severes from T1 to T2, while the gain for the severes approaches significance [t(35)=1.83, p=.076). The gain for the very severes appears greater in the first than in the second recovery period (when there is a loss), but the difference does not reach statistical significance [t(25)=-2.02, p=.055].

11.2.2.4 Persisting Impairment on Sentence Repetition

Children with scores lower than those of 95% of the controls at 1 year, were defined as having persisting impairment. The 5th percentile of the control group at T3 is 47.3 and 2 controls (4.9%), 3 moderates (5.7%), 2 severes (5.3%) and 6 very severes (22.2%) are rated as impaired. Differences between the control and head-injured groups are not statistically significant.

11.3 Language Comprehension

11.3.1 Stanford Binet Pictorial Identification

11.3.1.1 Differences Between the Groups

Pictorial Identification was used as a screening test. Table 35 presents the percentage of each group able to identify all 6 pictures at the three successive test occasions.

Table 35

Pictorial Identification: Number and Percentage of Children with Perfect Scores

	Controls	Head Injured			Chi-square
Test Occasion	(n=46)[a] % (n)	Moderates (n=55)[b] % (n)	Severes (n=40)[c] % (n)	Very Severes (n=28)[d] % (n)	(df=3)
T1	**78.3** (36)	**67.3** (37)	**42.5** (17)	**28.6** (8)	23.59***
T2	**90.5** (38)	**75.5** (40)	**57.5** (23)	**48.2** (13)	18.38***
T3	**95.1** (39)	**88.7** (47)	**76.3** (29)	**48.2** (13)	26.27***

[a]n=42 at T2, 41 at T3; [b]n=53 at T2 and T3; [c]n=38 at T3; [d]n=27 at T2 and T3.
*** p<.001.

The chi-squared analysis shows that there are significant differences between the groups in the percentage of children obtaining perfect scores at each test occasion. Inspection of Table 35 shows the percentages to be in descending order according to severity of injury. On pairwise comparisons of the groups (see Table C11 , p.443 for chi-squared values; p must be <.01 to be significant at an overall 5% level), significantly fewer very severes than controls achieve perfect scores at each test occasion. The percentage of very severes achieving perfect scores is also

significantly lower than that of the moderates at T1 and T3 and approaches statistical significance at T2 [X^2(1, n=80)=5.97, p=.015]. At T3, the difference between the severe and the very severe group also approaches statistical significance [X^2(1, n=65)=5.48, p=.019].

Significantly fewer severes than controls achieve perfect scores at T1 and T2 and this difference approaches significance at T3, [X^2(1, n=79)=5.82, p=.016]. Although fewer moderates than controls achieve perfect scores at each test occasion, these differences are not statistically significant. None of the differences between the moderates and the severes reach statistical significance, although they approach it at T1 [X^2(1, n=95)=5.79, p=.016].

11.3.1.2 Changes Over Time

Mc Nemar's test was used to assess changes from T1 to T2 and T2 to T3 within the groups, based on the 39 controls, 52 moderates, 38 severes and 27 very severes who completed Pictorial Identification on all three occasions (see Table C12, p. 443 for values). Within the control group the percentage of children with perfect scores increases from 80% to 90% to 95% on the successive test occasions, but these changes are not significant. Within the moderate group, the increases from 67% to 75% and 89% of children with perfect scores approach significance between T2 and T3 (p=.07). Within the severe group, the change from 42% at T1 to 58% at T2 approaches significance (p=.077), while the increase from 58% at T2 to 76% at T3 is significant. Within the very severe group, the percentage with perfect scores increases from 30% at T1 to 48% at T2 and T3, but the increase is not significant.

11.3.1.3 Persisting Impairment on Pictorial Identification

Since 95% of the control group achieve perfect scores at the 1 year follow-up, impairment is defined as achieving less than a perfect score. Thus 2 controls (4.9%), 6 moderates (11.3%), 9 severes (23.7%) and 14 very severes (51.9%) are impaired on this screening test at 1 year. Differences between the controls and the very severes and between the controls and the severes are significant [$X^2(1, n=68)=19.96$, $p<.0001$; and $X^2(1, n=79)=5.82$, $p=.016$ respectively].

11.3.1.4 Summary

In summary, this screening test achieves surprisingly good differentiation of the groups at all three test occasions. There is a trend for more children to achieve perfect scores at each test occasion. However, it was not clear what the test was measuring. A child could fail an item for several reasons, eg inattention, failure to comprehend the question, failure to recognise the picture, or not knowing the answer.

11.3.2 Token Test

The Token Test was introduced as a more pure measure of comprehension than the Pictorial Identification. It was not administered to the first 10 head-injured children in the sample at T1, but was completed by them at T2 and T3. A further 10 children, 9 of them 6 year-olds, were unable to do the test as they did not know colour names.

11.3.2.1 Adjustment of Scores

Many children reached the test ceiling (10 out of 10) on Parts 1 and 3, with the result that scores were not normally distributed and could not be adjusted by analysis of covariance. Parts 1 and 3 were analysed in terms of the percentage of children achieving the expected score for age

(ie 10 for all subjects on Part 1, but 9 for those under 9.5 years on Part 3). Scores on Part 5 were normally distributed and analysis of control group scores at T1 showed there to be significant effects for language and age, but not for sex or SES. Using age and language (Afrikaans=0, English=1) as covariates, the pooled estimates of coefficients were obtained and all children adjusted to Afrikaans language (0) and an age of 114, 117 and 126 months:

At T1, Part 5 Adjusted = Part 5 score + 0.1143(114 - age) + 3.5497(0 - language).

At T2, Part 5 Adjusted = Part 5 score + 0.0871(117 - age) + 2.3635(0 - language).

At T3, Part 5 Adjusted = Part 5 score + 0.0829(126 - age) + 2.0362(0 - language).

11.3.2.2 Differences Between Groups

Table 36 summarizes the percentage of children with perfect scores for age on Parts 1 and 3, and the adjusted mean scores for Part 5.

Table 36

Language Comprehension: Token Test at T1, T2 and T3

Test Occasion	Controls	Head Injured			Statistic
		Moderates	Severes	Very Severes	
Part 1: Distribution of Perfect Scores					
	% (*n*)	% (*n*)	% (*n*)	% (*n*)	$X^2(df=3)$
	(*n*=45)	(*n*=44)	(*n*=36)	(*n*=23)	
T1	91.1 (41)	77.3 (34)	72.2 (26)	52.2 (12)	13.24**
	(*n*=42)	(*n*=48)	(*n*=35)	(*n*=26)	
T2	97.6 (41)	89.6 (43)	88.6 (31)	53.9 (14)	26.79***
	(*n*=41)	(*n*=52)	(*n*=35)	(*n*=26)	
T3	92.7 (38)	90.4 (47)	94.3 (33)	69.2 (18)	
Part 3: Distribution of Perfect Scores					
	% (*n*)	% (*n*)	% (*n*)	% (*n*)	$X^2(df=3)$
	(*n*=45)	(*n*=44)	(*n*=35)	(*n*=23)	
T1	68.9 (31)	52.3 (23)	48.6 (17)	8.7 (2)	22.23***
	(*n*=42)	(*n*=48)	(*n*=35)	(*n*=26)	
T2	69.1 (29)	70.8 (34)	62.9 (22)	34.6 (9)	10.79*
	(*n*=41)	(*n*=52)	(*n*=35)	(*n*=26)	
T3	85.4 (35)	67.3 (35)	57.1 (20)	46.2 (12)	12.72**
Part 5: Adjusted Scores					
	M *SD*	*M* *SD*	*M* *SD*	*M* *SD*	
	(*n*=45)	(*n*=44)	(*n*=35)	(*n*=22)	
T1	12.9_a 3.6	10.8_{ab} 4.0	11.0_a 4.5	8.0_b 4.7	
	(*n*=42)	(*n*=48)	(*n*=35)	(*n*=25)	
T2	13.9_a 2.9	12.9_a 3.9	13.1_a 3.8	10.6_b 3.6	
	(*n*=41)	(*n*=52)	(*n*=35)	(*n*=25)	
T3	15.5_a 3.1	14.1_{ab} 3.9	14.4_{ab} 3.5	12.8_b 4.3	

Note. Means not sharing a subscript at a test occasion differ significantly on Tukey's studentized range test at $p<.05$.

Table 36 shows that there are significant differences between the groups in the percentage of children achieving perfect scores on Parts 1 and 3 of the Token Test. (The

significance of overall differences on Part 1 at T3 could not be assessed as there were so few cases with scores below 10). Pairwise comparisons between the groups on Parts 1 and 3 were carried out using either chi-squared or Fisher's exact tests (values in Table C13, p.444). For Part 5, the significance of differences in group means is indicated by the subscripts in Table 36 and the confidence limits for Tukey's studentized range test are in Table C13.

Differences between the very severes and the controls are significant on all parts of the Token Test at all test occasions, except on Part 1 at T3, where differences approach significance (p=.018). There is also a trend for fewer very severes than moderates to have perfect scores on Parts 1 and 3 (significant for Part 1 at T2, and for Part 3 at T1 and T2), and for the very severes to have lower scores on Part 5 (significant at T2). There is a similar trend for fewer very severes than severes to have perfect scores on Parts 1 and 3 (significant for Part 1 at T2 and Part 3 at T1) and for scores to be lower on Part 5 (significant at T1 and T2).

There is a trend for fewer severes than controls to have perfect scores on Parts 1 and 3, particularly at T1 (see Table 36 and Table C12), but the only difference to reach statistical significance is that on Part 3 at T3. Differences between the severes and controls on Part 5 are in the expected direction, but are not statistically significant. None of the differences between the severes and the moderates are significant.

Differences between the moderates and the controls are in the expected direction, but are not significant.

11.3.2.3 Changes Over Time

The analysis of changes over time is based on children completing the Token Test on all three test occasions. For Parts 1 and 3, Table 37 summarizes the percentage of children achieving perfect scores on each test occasion. Differences in changes between the groups are not compared. For Part 5, Table 37 summarizes the mean size of changes in the adjusted scores over the two recovery intervals. The significance of these changes over time and between groups is determined by repeated measures analysis of variance. The distribution of the adjusted scores on Part 5 for those present on all three test occasions is shown in Figure 13.

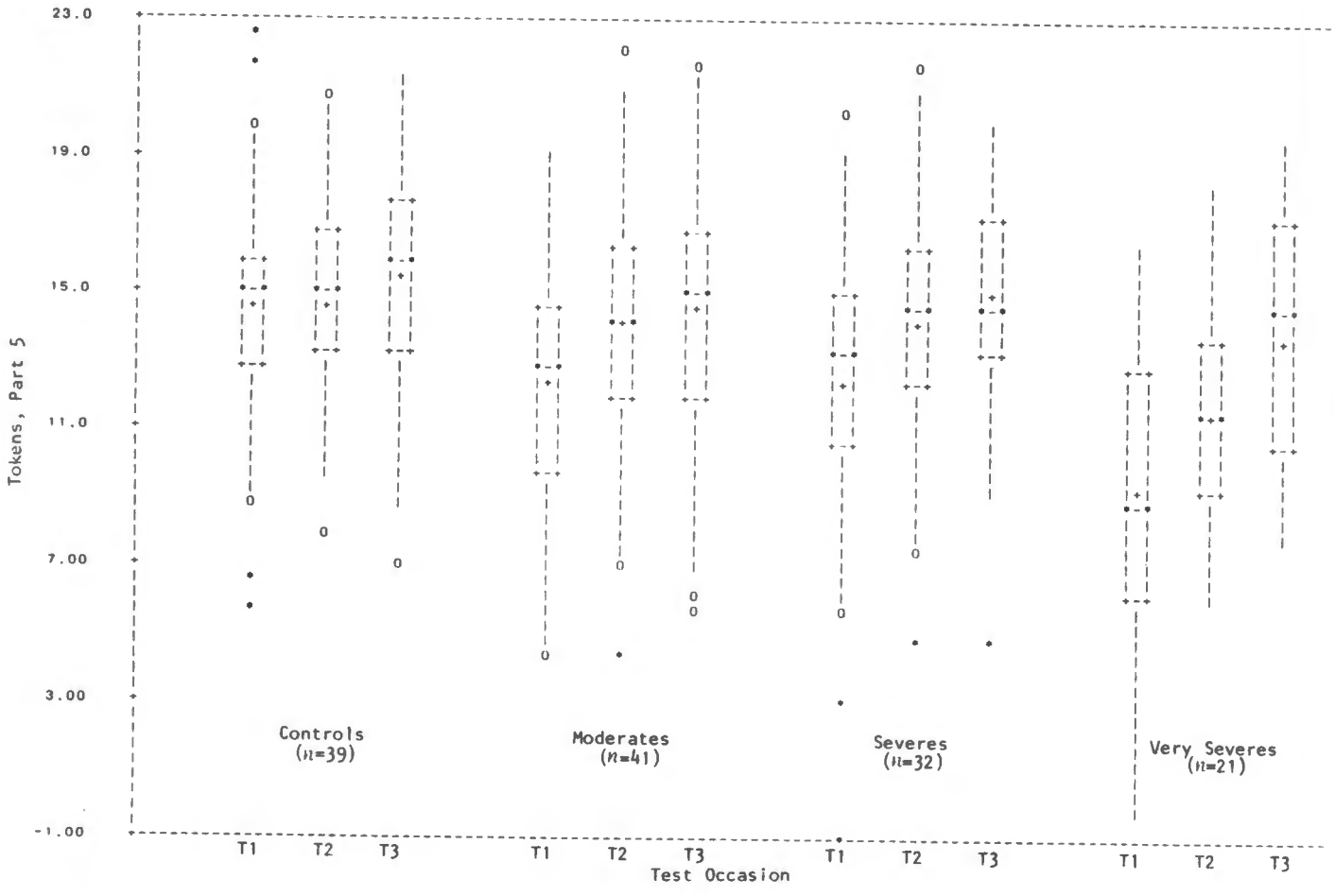


Figure 13. Token Test Part 5: Adjusted Scores at Successive Test Occasions

Table 37

Token Test: Changes Over Time

	Controls		Head Injured							
Time		(n=39)	Moderates (n=41)		Severes (n=32)		Very Severes (n=22)*			
Test Occasion	%	(n)	%	(n)	%	(n)	%	(n)		
			Part 1: Perfect Scores							
T1	92.3	(36)	80.5	(33)	81.3	(26)	54.6	(12)		
T2	97.4	(38)	90.2	(37)	93.8	(30)	59.1	(13)		
T3	92.3	(36)	87.8	(36)	100.0	(32)	68.2	(15)		
Test Occasion	%	(n)	%	(n)	%	(n)	%	(n)		
			Part 3: Perfect Scores							
T1	66.7	(26)	56.1	(23)	46.9	(15)	9.1	(2)		
T2	69.2	(27)	70.7	(29)	62.5	(20)	36.4	(8)		
T3	84.6	(33)	68.3	(28)	62.5	(20)	50.0	(11)		
Recovery Interval	*M*	*SD*	Part 5: Changes in Adjusted Scores *M*	*SD*	*M*	*SD*	*M*	*SD*	Over Time *F*(1,129)	Between Groups *F*(3,129)
T1 to T2	0.35	2.45	1.69	2.65	1.54	3.75	2.46	2.96	32.60***	2.71*
T2 to T3	0.81	2.34	0.40	2.74	0.90	2.34	1.97	2.55	20.68***	1.84

*n=21 on Part 5.
* $p<.05$, *** $p<.001$.

There is a significant overall increase in scores on Part 5 over both recovery intervals (see Table 37, Figure 13). The size of changes in the first, but not the second, recovery interval differs significantly between the groups (Table 37). Mean changes from T1 to T2 were therefore compared using Tukey's studentized range test (confidence limits in Table C14, p.445). The mean increase in the

scores of the very severes (2.46) is significantly larger than the mean increase of the controls (0.35). No other changes differed significantly between the groups.

Within Groups. The significance of changes within the groups was determined on McNemar tests for Parts 1 and 3, and on paired t-tests for Part 5 (values in Table C15, p.445). Within the control group, the only significant increase is in the score on Part 5 from T2 to T3, while the increase in the number obtaining perfect scores on Part 3 in the same period approaches significance (p=.058). Within the very severe group, scores on Part 5 increase significantly over both recovery periods. The number of children obtaining perfect scores on Part 3 also increases significantly between T1 and T2, but other increases are not statistically significant. Within the severe group, increases in the number of children with perfect scores are significant for Part 1 in the first recovery period, and scores on Part 5 increase significantly over both recovery periods. Within the moderate group, increases in the number of children with perfect scores on Part 1 and in the scores obtained on Part 5, are significant for the first recovery period. Changes on Part 5 in the first recovery period do not differ significantly from those in the second recovery period for any of the groups.

11.3.2.4 Persisting Impairment on the Token Test

At the 1 year follow-up there tended to be 3 controls (7%) clustered at the lower end of scores on Parts 1 and 3, one of whom was Q.L., a child with definite language impairment. It was therefore decided to set as cut-points scores which were exceeded by at least 93% of the controls. Cut-points are less than the maximum score of 10 on Part 1; less than 60% of what is expected for age on Part 3, and

less than the 5th percentile of the control group at T3 on Part 5. Table 38 summarizes the percentage of each group falling below these cut-points. The significance of differences between the control and head-injured groups was determined by Fisher's exact tests. Only the difference between the controls and the very severes on Part 5, is significant, while the difference on Part 1 is very close to being statistically significant (p=.018, but must be <.017 to be significant at an overall 5% level).

Table 38

Token Test: Percentage with Impairment at 1 Year Post-Injury

Token Test	Cut-Point	Controls (n=41) %	(n)	Moderates (n=52) %	(n)	Severes (n=35) %	(n)	Very Severes (n=26)[a] %	(n)
Part 1	<10	**7.3**	(3)	**9.6**	(5)	**5.7**	(2)	**30.8**	(8)
Part 3	<60%	**7.3**	(3)	**3.9**	(2)	**14.3**	(5)	**19.2**	(5)
Part 5	<10.74	**4.9**	(2)	**15.4**	(8)	**11.4**	(4)	**36.0****	(9)

[a]n=25 on Part 5.
** Differ from controls at p<.003.

11.4 Correlations Between the Tests

In order to determine whether the language tests were measuring separate functions, the interrelationship of control group scores was examined at T1. As is shown in Table 39, the correlations of the language tests with each other are either not significant or are low to moderate (.37 for Word Fluency and Speed; .46 for Renfrew and Speed; and .50 for Sentence Repetition and Token Test 5). Correlations of the language tests with Verbal IQ are not significant for the Renfrew and Speed of Naming, low for Word Fluency (.34), and moderate for Tokens Part 5 (.52) and Sentence Repetition (.59).

Table 39

Correlations Between Language Measures and VIQ

	VIQ	WORD	SENTREPR	Renfrew	Speed	TOKEN5A
VIQ		0.34881 0.0175 46	0.58633 0.0001 44	0.25934 0.0818 46	-0.06684 0.6590 46	0.51813 0.0003 45
WORD Flu			0.22138 0.1487 44	0.25221 0.0909 46	-0.36986 0.0114 46	0.01100 0.9428 45
SENTREPR				0.19056 0.2153 44	-0.20142 0.1898 44	0.49532 0.0007 43
Renfrew					-0.45985 0.0013 46	0.14263 0.3500 45
Speed						-0.18966 0.2121 45

Note. The first line gives the Spearman correlation coefficients, the second line the p value, and the third line n. Underlined coefficients, $p<.05$.

11.5 Summary of Language Test Results

1. The very severes have significantly lower scores than the controls on almost all language measures (production, comprehension and repetition), at the initial assessment and remain lower on all except Speed of Naming throughout the 1 year follow-up. Their scores are often significantly lower than those of the moderates and the severes as well. At 1 year, from one third to half have persisting impairment on Word Fluency, Renfrew Picture Naming, Pictorial Identification, and Part 5 of the Token Test.

2. The severes are significantly weaker than the controls on Word Fluency and Renfrew Naming measures at intake. They remain weaker on Renfrew Naming throughout and 29% have persistent impairment at 1 year. On the

comprehension tests, fewer are able to achieve perfect scores on Pictorial Identification throughout (24% have persistent impairment at 1 year), and there is a trend for fewer to succeed on the Token Test measures, particularly at intake. Repetition scores are not significantly lower at any point.

3. The moderates are not significantly different from the controls at any test occasion, except on Renfrew Picture Naming at T3.

4. The controls tend to score below the published norms of what is expected for age. This is most striking at the initial assessment on Renfrew Picture Naming and Sentence Repetition and less striking for Word Fluency, Digit Repetition and Token Test comprehension where they score at about 90% of what is expected for age.

5. Improvement on most language measures is either significant or close to significant over both recovery intervals for the very severes and the severes. However, for the moderates, significant improvement occurs only on a few measures (Renfrew, Speed and Tokens), and is limited to the first 3 month period. The control group has significant improvement only on a single measure in each recovery period.

6. Generally, production and comprehension measures appear more sensitive to the effects of head injury than repetition measures: persisting impairment at 1 year occurs in 24% to 29% of the total head-injured group on production, in from 11% to 25% on comprehension, and in only 6% to 9% on repetition.

7. Correlations between the separate language measures in the control group at T1 are mostly not significant or are low, except for that between Renfrew Naming and Speed of Naming, which are based on the same test, and that between Token Test Part 5, and Sentence Repetition, which are both correlated with Verbal IQ.

11.6 Speech Defects

11.6.1 Articulation Difficulties

Table 40 summarizes the prevalence of mild articulation difficulties and impaired speech within the groups at each test occasion. Mild articulation difficulty refers to immature speech or to problems reported as present premorbidly (eg stammering). Impaired speech refers to more severe difficulties in articulation, resonance, stress and intonation. The statistical significance of differences between control and head-injured groups was determined on Fisher's exact tests.

Table 40

Immature Articulation and Impaired Speech

	Controls		Head Injured					
			Moderates		Severes		Very Severes	
Test	(n=46)[a]		(n=53)[b]		(n=40)[c]		(n=27)[d]	
Occasion	%	(n)	%	(n)	%	(n)	%	(n)
Mild articulation difficulty								
T1	**4.3**	(2)	**1.8**	(1)	**5.0**	(2)	**3.6**	(1)
T2	**4.8**	(2)	**5.7**	(3)	**2.5**	(1)	**7.4**	(2)
T3	**2.4**	(1)	**-**	(0)	**2.6**	(1)	**3.7**	(1)
Impaired Speech								
T1	-	(0)	-	(0)	**12.5**	(5)	**32.1****	(9)
T2	-	(0)	-	(0)	**5.0**	(2)	**25.9****	(7)
T3	-	(0)	-	(0)	-	(0)	**14.8**	(4)

[a]n=42 at T2, 41 at T3; [b]n=55 at T1; [c]n=38 at T3; [d]n=28 at T1
** Differs from controls at p<.003 on Fisher's exact tests.

As is shown in Table 40, mild articulation difficulties occurred with about equal frequency in all groups. A total of 12 children, (9 of them 6 or 7 year-olds) were rated as having mild difficulties on at least one test occasion. The children still rated as having mild difficulties at 1 year are a 12 year-old control and an 8 year-old severe with mild stammers, documented in both as present prior to the study, and a 13 year-old very severe with a lisp on 's' sounds.

Impaired speech occurred exclusively in the severes and very severes (Table 40). The problem was evident at T1 in all 14 of the children who incurred such a rating at any test session. Problems tended to diminish in frequency and severity over time, although these changes were not statistically significant on the McNemar test. At 1 year, only 4 children, all very severes, are still rated as having speech impairment.

11.6.2 Perseveration

Perseveration could be indicated as none, some, or marked. Perseveration was not found in any of the controls and only in 1 moderate on one occasion. Table 41 presents the distribution of those showing any perseveration (some or marked). The statistical significance of differences between the control and head-injured groups was determined on Fisher's exact tests.

Table 41

Perseveration at T1, T2 and T3

Test Occasion	Controls (n=46)[a] %	(n)	Moderates (n=53) %	(n)	Severes (n=40) %	(n)	Very severes (n=27)[b] %	(n)
T1	-	(0)	-	(0)	22.5	(9)	46.4**	(13)
T2	-	(0)	-	(0)	2.5	(1)	29.6**	(8)
T3	-	(0)	1.9	(1)	-	(0)	14.8	(4)

[a]n=42 at T1, 41 at T3; [b]n=28 at T1.
** Differs from controls at p<.003.

As is shown in Table 41, perseveration was statistically significant in the severes and the very severes at the first test occasion and in the very severes at T2. At T3, the percentage of very severes approaches significance (Fisher's exact p=.022). Perseveration diminished in severity as well as frequency over the successive test occasions: 1 severe and 4 very severes had marked perseveration at T1, whereas only 1 very severe at T2 and none of the children at T3, had marked perseveration. At 1 year, 1 moderate gave an isolated example of perseveration, whereas the 4 very severes in Table 41 all had more than one example.

11.6.3 Echolalia

Six children, 1 severe and 5 very severes, were rated as showing echolalia at initial testing. Mitigated echolalia (repetition of questions) occurred in 4 cases and automatic echolalia in 2, both very severes. All except 1 of the children with echolalia also showed perseveration. No child showed echolalia at either T2 or T3.

11.6.4 Summary

Impaired speech, perseveration and echolalia are predominantly characteristic of only the most severely injured and diminish in frequency and severity over time.

12. VISUOMOTOR TEST RESULTS

12. VISUOMOTOR TEST RESULTS

Several children were unable to complete the visuomotor tests at intake (T1): 12 controls and 10 head injured had broken arms and a further 4 controls and 7 head injured were impeded by orthopaedic devices. Where weakness, increased tone or tremor impaired hand functioning, results were excluded from the analysis, except in determining the percentage of children with persisting impairment at T3 (cf Levin & Eisenberg, 1979a,b). Such problems affected the motor tests (Purdue pegboard and successive finger tapping) more often than the visuographic tests, (Detroit and Beery) tests. Four children had unilateral involvement and 4 children bilateral involvement.

12.1 Purdue Pegboard

12.1.1 Differences between the Groups

Raw scores were transformed into T scores, using the norms of Gardner & Broman (1979). (Analysis of control group raw scores at T1 showed a significant effect for age, but not for sex or SES). Findings are summarized in Table 42 and subscripts show that the very severe group differs significantly from the controls and the other head-injured groups on all three Purdue measures at all test occasions when Tukey's studentized range test is used to compare group means (see Table C16, p.446 for confidence limits).

Table 42

Purdue Pegboard: T scores at T1, T2 and T3

Test Occasion	Controls M	Controls SD	Head Injured: Moderates M	Moderates SD	Severes M	Severes SD	Very Severes M	Very Severes SD
Preferred Hand								
	(n=34)		(n=55)		(n=32)		(n=19)	
T1	**54.0$_a$**	13.1	**44.5$_b$**	11.4	**43.2$_b$**	9.9	**23.3$_c$**	15.4
	(n=42)		(n=53)		(n=38)		(n=22)	
T2	**55.8$_a$**	10.8	**53.9$_a$**	10.4	**50.7$_a$**	10.9	**37.0$_b$**	10.6
	(n=41)		(n=53)		(n=37)		(n=22)	
T3	**56.4$_a$**	10.0	**56.2$_a$**	9.6	**53.6$_a$**	9.3	**38.6$_b$**	13.0
Nonpreferred Hand								
	(n=41)		(n=54)		(n=36)		(n=18)	
T1	**54.0$_a$**	10.3	**47.5$_b$**	12.8	**43.5$_b$**	12.0	**22.2$_c$**	10.9
	(n=42)		(n=53)		(n=40)		(n=19)	
T2	**56.5$_a$**	8.6	**52.6$_{ab}$**	10.3	**49.0$_b$**	11.5	**39.1$_c$**	8.4
	(n=41)		(n=53)		(n=38)		(n=19)	
T3	**54.8$_a$**	9.7	**54.3$_a$**	10.9	**52.3$_a$**	10.3	**40.6$_b$**	11.6
Both Hands								
	(n=31)		(n=53)		(n=31)		(n=16)	
T1	**53.4$_a$**	9.1	**46.9$_{ab}$**	11.3	**41.5$_b$**	11.9	**19.5$_c$**	16.3
	(n=42)		(n=53)		(n=38)		(n=18)	
T2	**55.5$_a$**	8.4	**54.1$_a$**	10.4	**48.2$_b$**	10.5	**36.6$_c$**	7.0
	(n=41)		(n=53)		(n=37)		(n=18)	
T3	**55.9$_a$**	9.9	**53.0$_a$**	11.3	**49.6$_a$**	11.0	**40.2$_b$**	10.4

Note. Groups not sharing a subscript at a test occasion differ significantly on Tukey's studentized range test at $p<.05$.

The severe group also scores significantly lower than the control group on all three Purdue measures at T1 and is still significantly lower on the Nonpreferred hand and Both hands at T2. By the 1 year point (T3), none of the T scores are significantly lower than those of the controls, but the difference for Both hands approaches significance (6.3, confidence limits -0.08 to 12.6). The severes tend to perform at a slightly lower level than the moderates, but the only significant difference is on Both hands at T2.

The moderates score significantly lower than the controls on either hand used alone at T1, and the difference on Both hands approaches significance (6.5, confidence limits -0.39 to 13.4). The moderates no longer differ from the controls on any Purdue measures at T2.

12.1.2 Changes Over Time

The distribution of T scores on the Purdue measures for those able to complete the test on all three occasions is presented in Figure 14 for the Preferred hand, Figure 15 for the Nonpreferred hand and Figure 16 for Both hands. The mean size of changes in T scores from one test occasion to another and the F ratios for the overall significance of changes over time and between groups are summarized in Table 43.

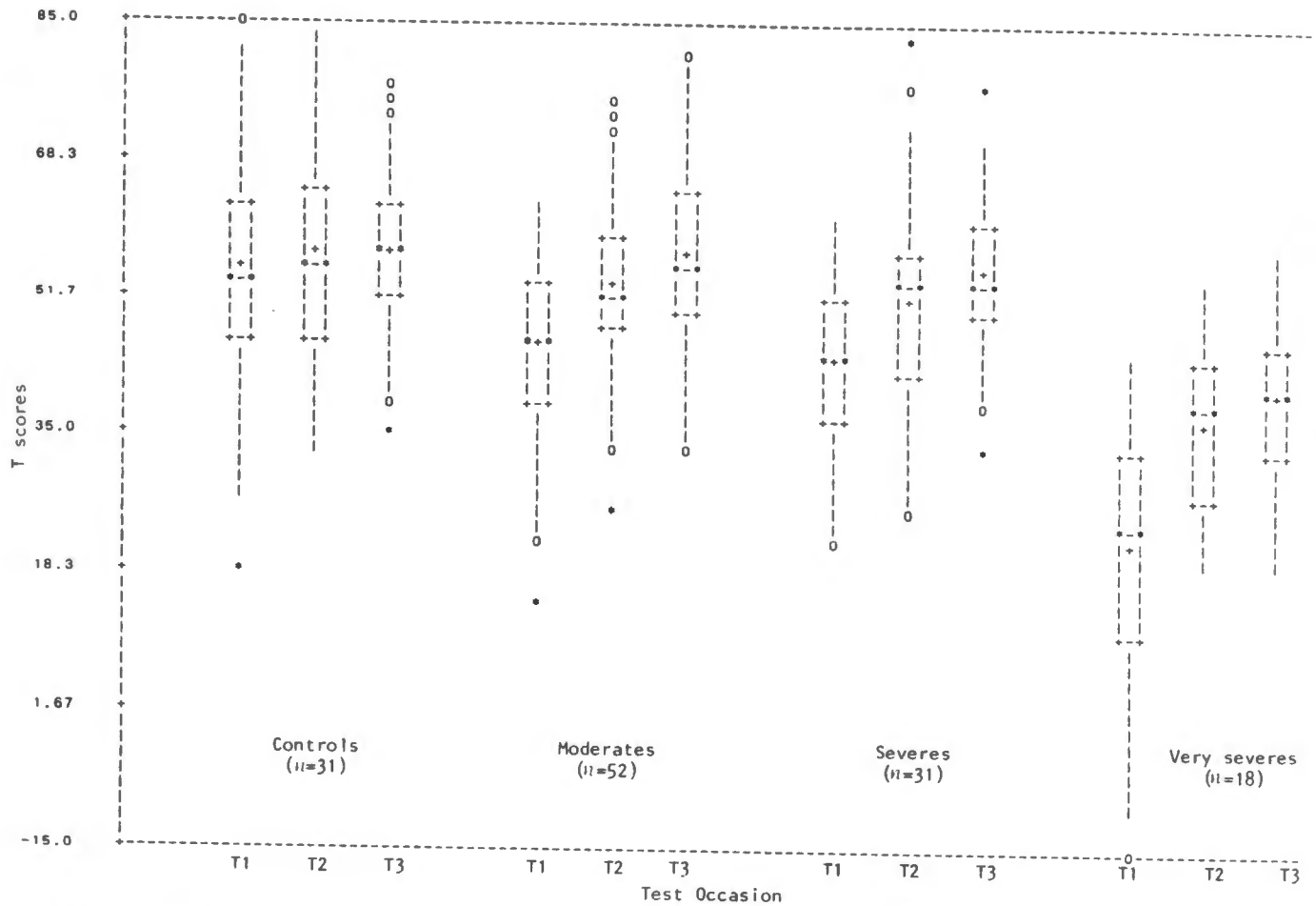


Figure 14. Purdue Pegboard: Preferred Hand T Scores at Successive Test Occasions.

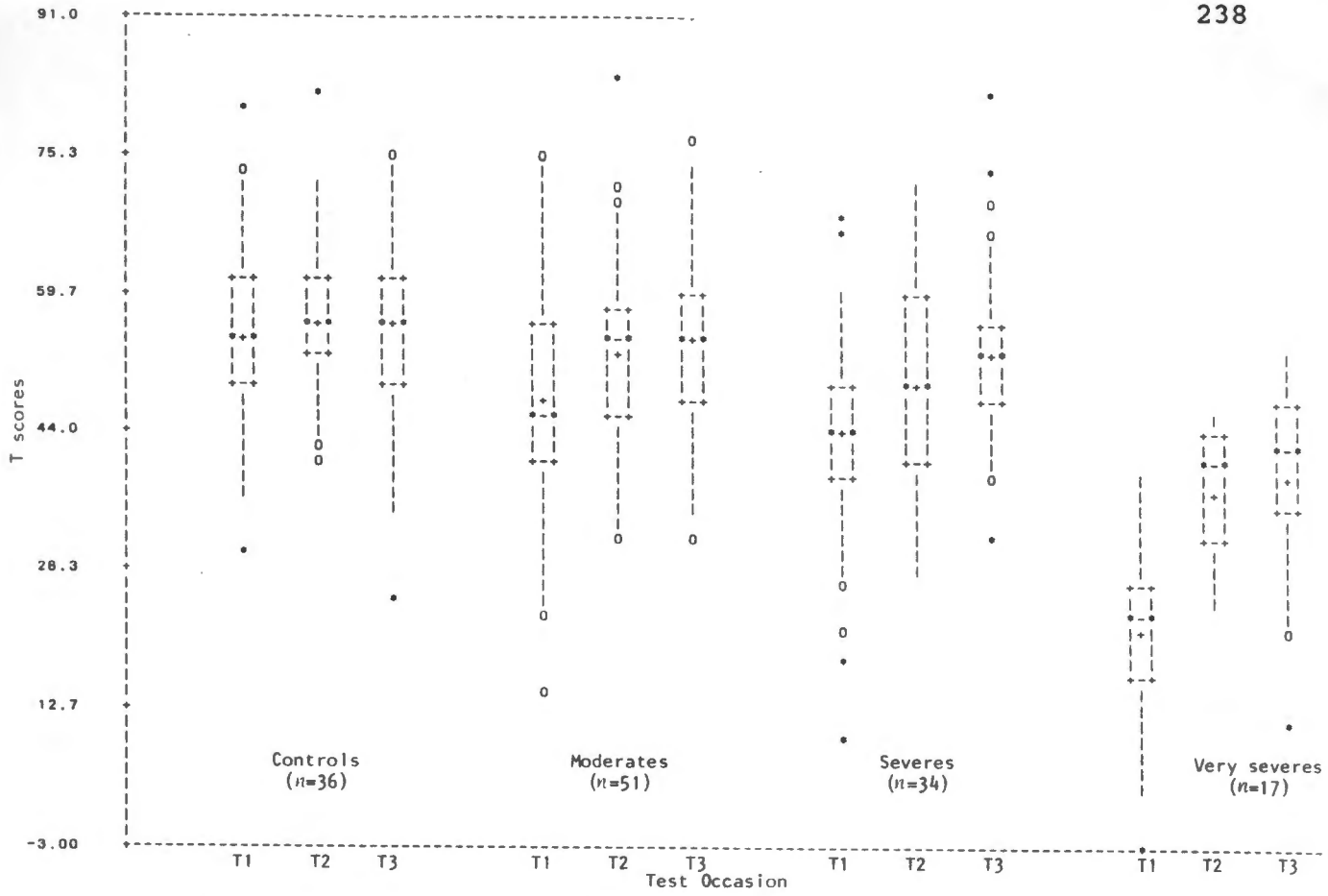


Figure 15. Purdue Pegboard: Nonpreferred Hand T Scores at Successive Test Occasions.

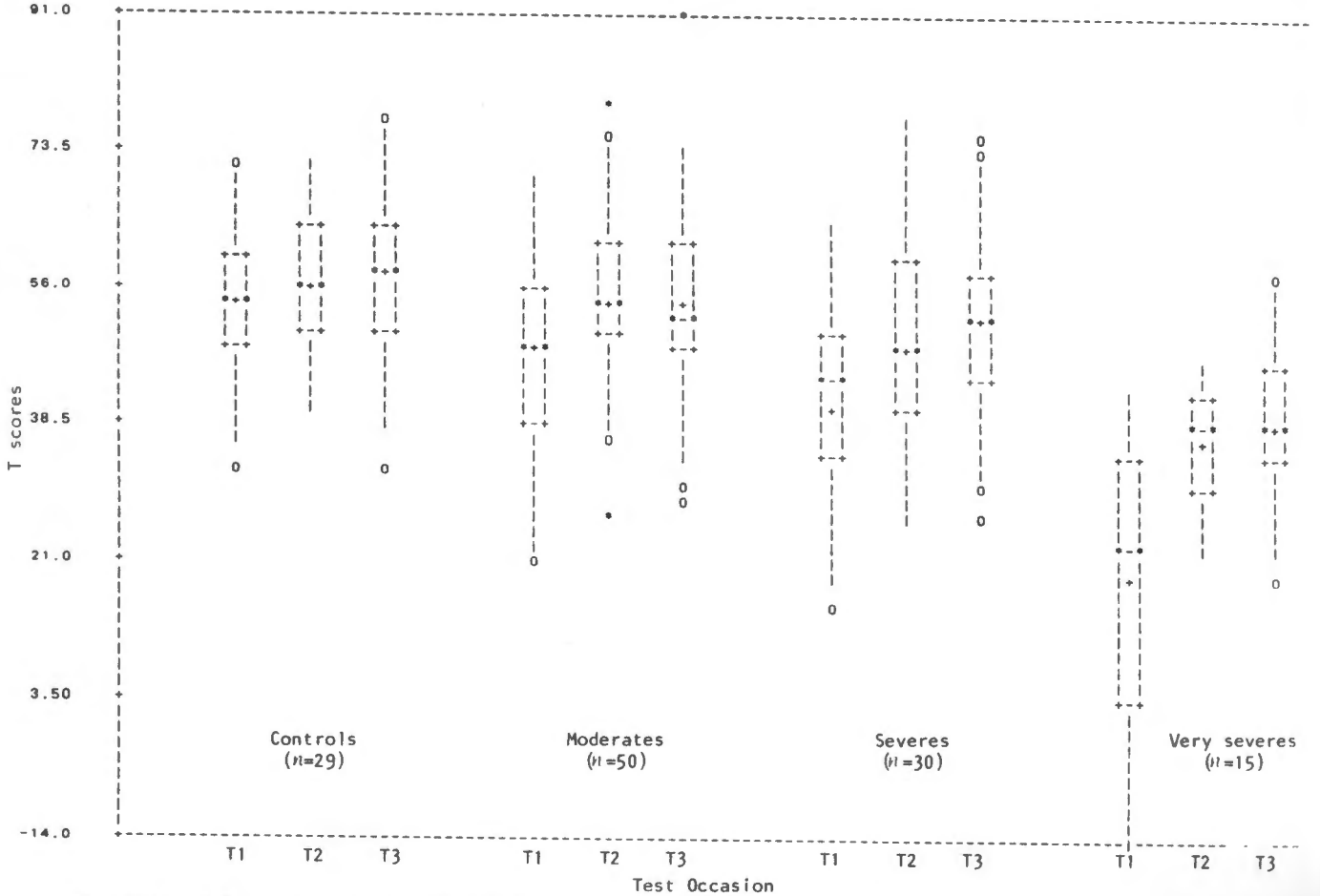


Figure 16. Purdue Pegboard: Both Hands T Scores at Successive Test Occasions.

Table 43

Purdue Pegboard T Scores: Mean Changes Over Time

Recovery Interval	Controls M	Controls S	Moderates M	Moderates SD	Severes M	Severes SD	Very Severes M	Very Severes SD	Repeated Measures Analysis: Over Time F value	Repeated Measures Analysis: Between Groups F value
Preferred Hand										
	(n=31)		(n=52)		(n=31)		(n=18)		(df=1,128)	(df=3,128)
T1 to T2	1.85	11.97	8.62	10.28	8.32	10.00	15.49	13.13	69.15***	6.02***
T2 to T3	1.00	8.31	2.53	9.33	2.84	11.30	2.24	8.38	5.91*	0.23
Nonpreferred Hand										
	(n=36)		(n=51)		(n=34)		(n=17)		(df=1,134)	(df=3,134)
T1 to T2	2.36	9.86	4.74	12.06	6.34	9.78	16.41	11.91	54.72***	6.67***
T2 to T3	-1.19	7.11	1.62	9.11	3.40	8.81	1.61	10.07	2.89 (p=.0914)	1.68
Both Hands										
	(n=29)		(n=50)		(n=30)		(n=15)		(df=1,120)	(df=3,120)
T1 to T2	2.79	8.69	7.14	9.18	7.51	9.91	17.26	14.04	79.13***	7.03***
T2 to T3	0.74	8.40	-1.04	8.98	2.37	8.29	3.53	8.40	2.73	1.59

* $p<.05$, *** $p<.001$.

12.1.2.1 Within Groups

As is shown in Table 43, in the first recovery period (T1 to T2), there is a significant overall increase in scores on all three Purdue measures. In the second recovery period (T2 to T3), there is a significant overall increase in the Preferred hand scores, while the over time F values for the Nonpreferred and Both hands approach significance (p=.091 and p=.101 respectively).

The significance of changes within each group were determined on paired t-tests (see Table C17, p.447). In each of the head-injured groups there are significant gains on all three Purdue measures from T1 to T2 (Table 43, Figures 14 to 16). In the second recovery period, there are further small gains, but only the gain of the severes on the

Nonpreferred hand reaches significance, while that of the moderates on the Preferred hand approaches significance (p=.056). The gains of the head injured tend to be larger in the first than in the second recovery interval, and are significantly larger for the very severes on all three Purdue measures, and for the moderates on the Preferred and Both hand measures (Table C17).

The changes in control group scores are small and are not significant for either recovery period.

12.1.2.2 Between Groups

There is a significant difference between the groups in the size of changes on the Purdue measures between T1 and T2, but changes from T2 to T3 do not differ significantly (F values in Table 43). Tukey's studentized range test was carried out to compare the size of changes in the groups in the first recovery period (confidence limits in Table C18,p. 447).The very severes have significantly greater increases on all Purdue measures than the controls, and a significantly greater increase than the other head-injured groups on the Nonpreferred hand and on Both hands. The moderates have a significantly greater increase than the controls on the Preferred hand.

12.1.3 Persisting Impairment on the Purdue Pegboard

Table 44 summarizes the cut-points (5th percentile of the control group at T3) used for determining impairment on the pegboards, and the percentage of each group falling below them. Only in the very severe group does the percentage rated as impaired significantly exceed the 5% of controls so rated. [Preferred hand, $X^2(1, n=68)=15.59$; Nonpreferred hand, $X^2(1, n=67)=14.25$; Both hands, Fisher's exact; p levels as indicated in Table 44].

Table 44

Purdue Pegboard: Percentage with Impairment at 1 Year Post-Injury

Hand	Cut-Point T Score	Controls (n=41) % (n)	Moderates (n=53) % (n)	Severes (n=38) % (n)	V.Severes (n=26)[a] % (n)
Pref.	<36.0	**4.9** (2)	**1.9** (1)	**5.3** (2)	**44.4***** (12)
Nonpref.	<37.3	**4.9** (2)	**7.6** (4)	**5.3** (2)	**42.3***** (11)
Both	<34.4	**4.9** (2)	**5.7** (3)	**13.2** (5)	**38.5**** (10)

[a]n=27 on Preferred hand.
** Differs from controls at p<.003, *** p<.0003.

12.2 Successive Finger Taps (Denckla, 1973, 1974).

Adequate norms are not available for this test and analysis of covariance was therefore used to adjust scores. The control group scores at T1 showed significant effects for age for either hand; an effect for sex on the Nonpreferred, but not the Preferred hand; and no effect for either SES or language. In calculating the coefficients for age and sex, pooled estimates from analysis of covariance were used and 1 outlying case excluded at all test occasions for the Preferred hand, another at T1 only, and 1 case excluded at T1 and T2 for the Nonpreferred hand. All cases were then adjusted by analysis of covariance to an age of 114, 117 or 126 months at T1, T2 and T3 respectively, and to male sex (boy=1, girl=2) for the Nonpreferred hand. For the analysis of changes over time, all scores were adjusted to 126 months, using these equations:

Preferred Hand

At T1,
Successive Adjusted = Time in seconds - 0.0796(114-age)
At T2,
Successive Adjusted = Time in seconds - 0.0708(117-age)
At T3,
Successive Adjusted = Time in seconds - 0.0636(126-age)

Nonpreferred Hand

At T1,
Successive Adjusted = Time in seconds - 0.08617(114-age)-1.7490(1-sex)
At T2,
Successive Adjusted = Time in seconds - 0.0668(117-age)-0.4474(1-sex)
At T3,
Successive Adjusted = Time in seconds - 0.0674(126-age)-1.1744(1-sex)

12.2.1 Differences Between the Groups

The means and standard deviations of the adjusted Successive tapping scores are summarized in Table 45. The confidence limits for Tukey's studentized range test are given in Table C19,p.448).

Table 45

Successive Finger Taps: Time to Complete 20 Taps (seconds)

	Controls		Head Injured					
			Moderates		Severes		Very severes	
	M	SD	M	SD	M	SD	M	SD
			Preferred Hand					
	(n=36)		(n=54)		(n=36)		(n=20)	
T1	**10.1_a**	2.0	**11.0_a**	2.2	**11.6_a**	4.1	**15.2_b**	4.7
	(n=42)		(n=53)		(n=38)		(n=21)	
T2	**9.0_a**	1.3	**9.6_{ab}**	2.2	**10.5_b**	2.4	**14.0_c**	3.2
	(n=41)		(n=53)		(n=37)		(n=22)	
T3	**8.2_a**	1.6	**8.4_a**	1.7	**8.5_a**	1.9	**11.2_b**	2.8
			Nonpreferred Hand					
	(n=45)		(n=52)		(n=39)		(n=18)	
T1	**11.0_a**	1.8	**11.8_a**	2.4	**12.4_a**	4.0	**16.0_b**	4.1
	(n=42)		(n=53)		(n=39)		(n=18)	
T2	**9.8_a**	1.8	**9.9_a**	1.8	**10.9_a**	2.6	**13.9_b**	3.2
	(n=41)		(n=53)		(n=38)		(n=19)	
T3	**9.3_a**	1.7	**9.5_a**	1.9	**9.3_a**	2.0	**11.8_b**	2.1

Note. Groups not sharing a subscript at a test occasion differ significantly on Tukey's studentized range test at $p<.05$.

As is shown in Table 45, the very severes are significantly slower than the controls and than the other head-injured groups in performing with either the Preferred or the Nonpreferred hand on all three test occasions.

There is a trend for the severes to be slower than the controls and the differences approach significance for each hand at T1 and are significant for the Preferred hand at T2. The severes tend to be slower than the moderates, but the differences are not significant.

There is a trend for the moderates to perform more slowly than the controls, particularly at T1, but these differences fall short of statistical significance.

12.2.2 Changes Over Time

The distribution of scores in seconds for those completing the successive taps on all three test occasions is depicted in Figure 17 for the Preferred hand and Figure 18 for the Nonpreferred hand, with all scores adjusted to age of 126 months. The mean changes in the number of seconds taken to complete the 20 successive finger taps are summarized in Table 46. The overall significance of changes over time and between the groups over the two recovery periods, is indicated by the F values in Table 46.

Table 46

Successive Finger Taps: Changes Over Time (seconds)

Recovery Interval	Controls M	Controls SD	Moderates M	Moderates SD	Severes M	Severes SD	Very Severes M	Very Severes SD	Repeated Measures Analysis: Over Time F value	Repeated Measures Analysis: Between Groups F value
					Preferred Hand					
	(n=32)		(n=51)		(n=35)		(n=19)		(df=1,133)	(df=3,133)
T1 to T2	-0.84	1.60	-1.01	2.15	-0.81	3.05	-0.91	2.71	17.01***	0.06
T2 to T3	-0.05	1.31	-0.65	1.78	-1.44	1.82	-2.26	2.30	46.57***	7.56***
					Nonpreferred Hand					
	(n=39)		(n=49)		(n=36)		(n=17)		(df=1,137)	(df=3,137)
T1 to T2	-0.67	1.74	-1.39	1.78	-1.15	2.79	-1.87	3.71	34.91***	1.23
T2 to T3	-0.04	1.96	0.13	1.79	-1.17	2.45	-1.55	2.41	11.83***	4.73**

** $p<.01$, *** $p<.001$.

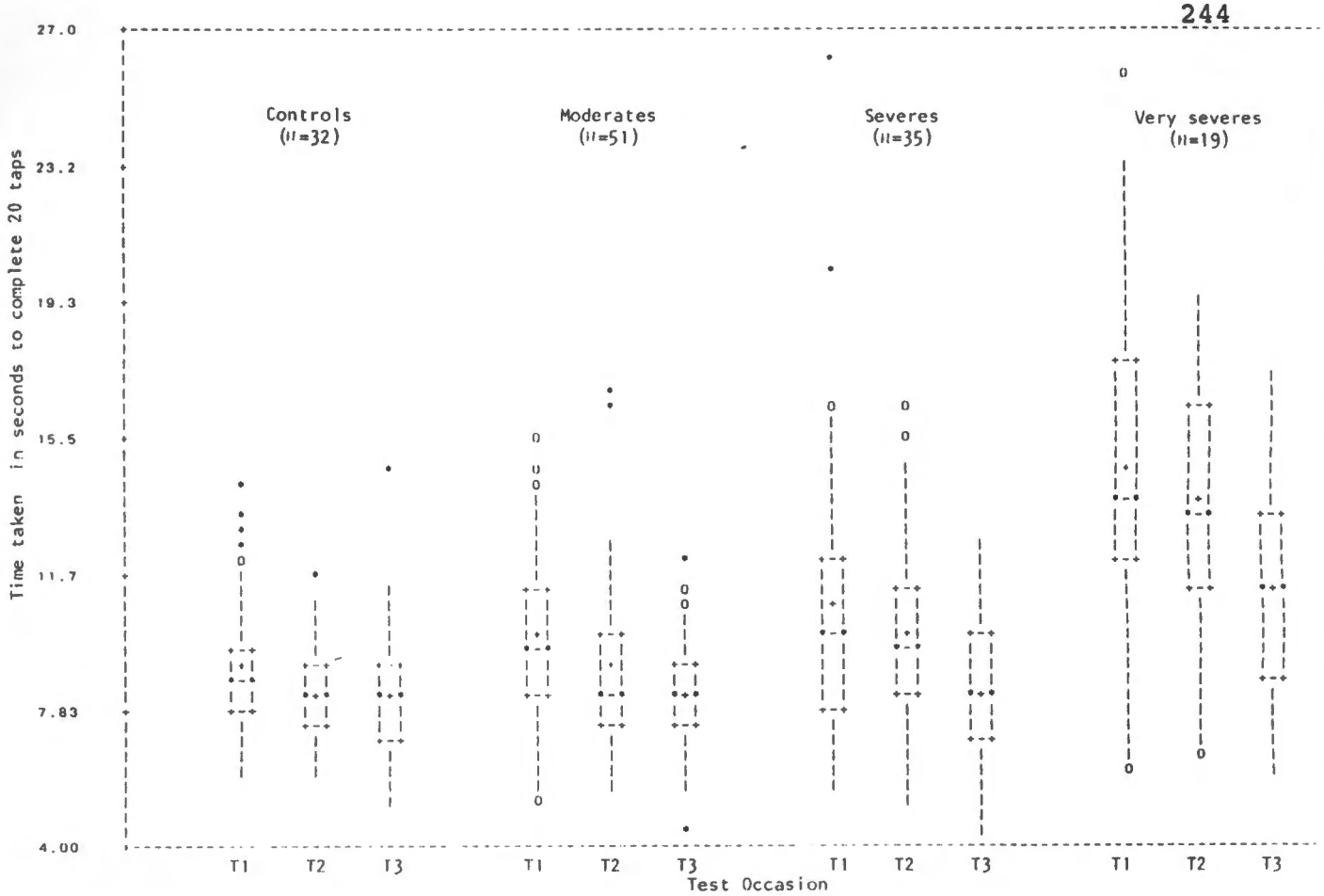


Figure 17. Successive Finger Taps Preferred Hand: Time Taken in Seconds.

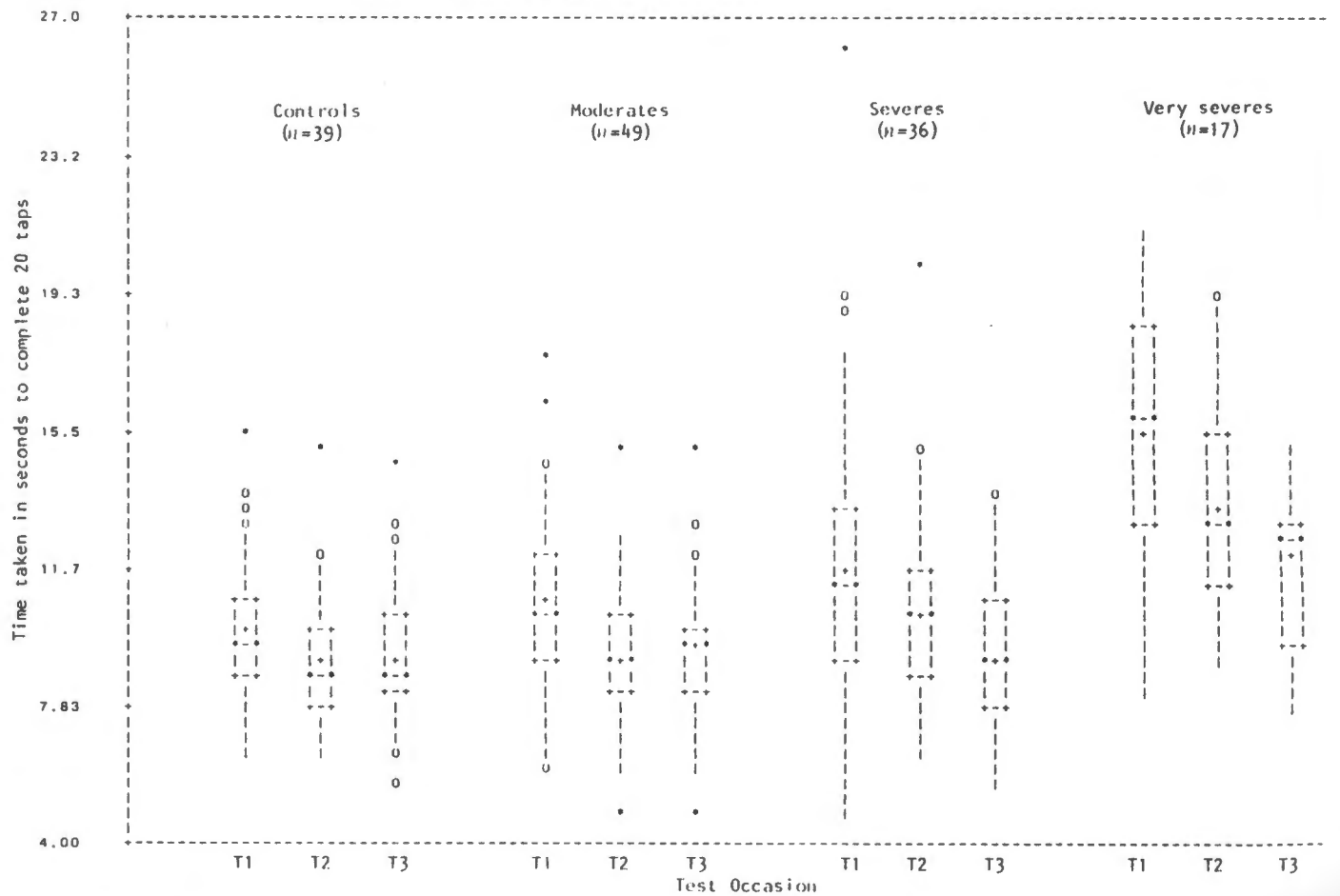


Figure 18. Successive Finger Taps Nonpreferred Hand: Time Taken in Seconds.

12.2.2.1 Within Groups

As is shown in Table 46, there is a significant overall decrease in the time taken to complete the task with either hand in both recovery periods. The significance of the changes within each group was determined on paired t-tests (see Table C20, p. 448). The controls show small but significant improvements in both hands from T1 to T2, but improvements from T2 to T3 are nonsignificant. The moderates similarly show significant improvements in both hands on the first recovery period, and a further small but significant improvement for the Preferred hand in the second recovery period. The improvement of the Nonpreferred hand in the first recovery period is significantly greater than the change in the second recovery period as this group were slightly slower at T3 than at T2.

Within the severe and very severe groups, there is significant improvement in the speed of both hands in the second recovery period. Improvements in the first recovery period are significant only for the severes' Nonpreferred hand, and approach significance for the Nonpreferred hand of the very severes ($p=0.54$). Differences between the two recovery intervals are not significant in either group.

12.2.2.2 Between Groups

In the second recovery period, there are significant overall differences between the groups in the size of the changes that occur in the time scores for both the Preferred and the Nonpreferred hand (see last column, Table 46). Tukey's studentized range test was carried out to determine which of the differences between the groups are significant (see Table C21, p. 449 for confidence limits). For the Preferred hand, the very severes and severes both show a significantly greater reduction in scores than is the case

for the controls, and the very severes also show a greater reduction than the moderates. For the Nonpreferred hand, the very severes and the severes show significantly greater reductions than the moderates, while the difference between the very severes and the controls approaches statistical significance (1.52, confidence limits -0.07 to 3.10).

12.2.3 Persisting Impairment on Successive Finger Taps

In defining impairment at 1 year, the 5th percentile of the control group at T3 was used as the cut-point for the Nonpreferred hand, and 2 standard deviations from the control group mean as cut-point for the Preferred hand. (The 5th percentile for the Preferred hand was 10.4 seconds which is close to what Denckla, 1985, found to be the mean for 10 year-old boys, viz 10.2 seconds). Table 47 summarizes the percentage of children in each group defined as impaired.

Table 47

Successive Finger Taps: Percentage with Impairment at 1 Year Post-Injury

Hand	Cut-Point (seconds)	Controls (n=41) %	(n)	Moderates (n=53) %	(n)	Severes (n=38) %	(n)	V.Severes (n=27)[a] %	(n)
Pref.	>11.83	2.4	(1)	3.8	(2)	5.3	(2)	44.4***	(12)
Nonpref.	>13.0	4.9	(2)	5.7	(3)	5.3	(2)	42.3***	(11)

[a]n=26 for Nonpreferred hand.
*** Differs from controls at p<.0003.

As is evident in Table 47, only in the very severe group does the percentage of children with persisting impairment exceed the percentage impaired in the control group [$X^2(1, n=68)=18.58$ for the Preferred hand, and $X^2(1, n=67)=14.25$ for the Nonpreferred hand).

12.3 Motor Incoordination on the WISC-R Mazes

As it appeared that some children were able to place pegs adequately yet showed poor pencil control, performance on the WISC-R Mazes was rated for incoordination. Table 48 summarizes ratings of none, some or marked incoordination in the groups. The chi-squared analysis is based upon a comparison of those with any incoordination, (ie some or marked), versus those with no incoordination.

Table 48

Incoordination on the WISC-R Mazes

Incoordination	Controls		Head Injured						Chi-square
			Moderates		Severes		Very Severes		
	%	(*n*)	%	(*n*)	%	(*n*)	%	(*n*)	(*df*=3)
Intake (T1)									
	(*n*=43)		(*n*=55)		(*n*=39)		(*n*=24)		
None	86.1	(37)	74.6	(41)	59.0	(23)	45.8	(11)	
Some	14.0	(6)	20.0	(11)	38.5	(15)	33.3	(8)	14.61**
Marked	-	(0)	5.5	(3)	2.5	(1)	20.8	(5)	
3 Months Later (T2)									
	(*n*=42)		(*n*=53)		(*n*=40)		(*n*=25)		
None	88.1	(37)	83.0	(44)	72.5	(29)	48.0	(12)	
Some	11.9	(5)	15.1	(8)	25.0	(10)	40.0	(10)	15.92**
Marked	-	(0)	1.9	(1)	2.5	(1)	12.0	(3)	
1 Year Later (T3)									
	(*n*=41)		(*n*=53)		(*n*=38)		(*n*=26)		
None	82.9	(34)	81.1	(43)	79.0	(30)	61.5	(16)	
Some	17.1	(7)	17.0	(9)	21.1	(8)	30.8	(8)	4.98
Marked	-	(0)	1.9	(1)	-	(0)	7.7	(2)	

Note. Chi-square is based on a two-way comparison of those with an without incoordination.
** $p<.01$, *** $p<.001$.

Table 48 shows that there is a significant difference between the groups in the percentage of children with and without incoordination at T1 and at T2. By 1 year (T3), group differences are no longer statistically significant. Chi-squared comparisons between the individual groups at T1 and T2 are summarized in Table C22, p. 449. At both these test occasions, a significantly higher percentage of very severes than of controls have incoordination. Differences between the very severes and the moderates approach significance at T1, (p=.013), and are significant at T2. At T1, the severes also have a significantly higher rate of incoordination than the controls, but this difference is no longer significant at T2, (p=.075). Differences between the moderates and the controls are not significant. Those rated as having marked incoordination at 1 year post-injury (ie 1 moderate and 2 very severes), were regarded as having persisting impairment in terms of this measure.

12.4 Detroit Motor Test of Speed and Precision (Baker & Leland, 1967)

12.4.1 Differences Between the Groups

The Detroit norms were used to transform the scores into age ratios. (Multiple regression analysis of the control group raw scores at T1 showed a significant effect for age, but not for sex or SES). The age ratio scores are summarized in Table 49.

Table 49

Detroit Motor Speed and Precision: Age Ratio Scores at T1, T2 and T3

	Controls		Head Injured					
			Moderates		Severes		Very severes	
	(n=42)[c]		(n=53)[d]		(n=39)[e]		(n=26)	
Variable	M	SD	M	SD	M	SD	M	SD
T1	**92.6$_a$**	12.1	**83.8$_a$**	20.7	**83.8$_a$**	15.6	**65.5$_b$**	17.0
T2	**94.9$_a$**	13.9	**89.8$_a$**	16.7	**88.5$_a$**	14.9	**68.3$_b$**	13.1
T3	**97.4$_a$**	12.9	**91.0$_a$**	15.9	**91.9$_a$**	15.5	**67.5$_b$**	12.9

Note. Groups not sharing a subscript differ significantly on Tukey's studentized range test at $p<.05$.
[c]n=36 at T1, 41 at T3, [d]n=54 at T1, [e]n=36 at T1, 38 at T3.

Analysis with Tukey's studentized range test shows that the very severe group differs significantly from the controls and from the other two head-injured groups at all three test occasions. No other group differences are significant, although the moderates and the severes tend to have slightly lower scores than the controls (confidence limits in Table C23, p.450).

12.4.2 Changes Over Time

Figure 19 depicts the distribution of Detroit age ratios in those children who completed the test on all three occasions. The mean changes in Detroit age ratios from T1 to T2 and from T2 to T3 are summarized in Table 50.

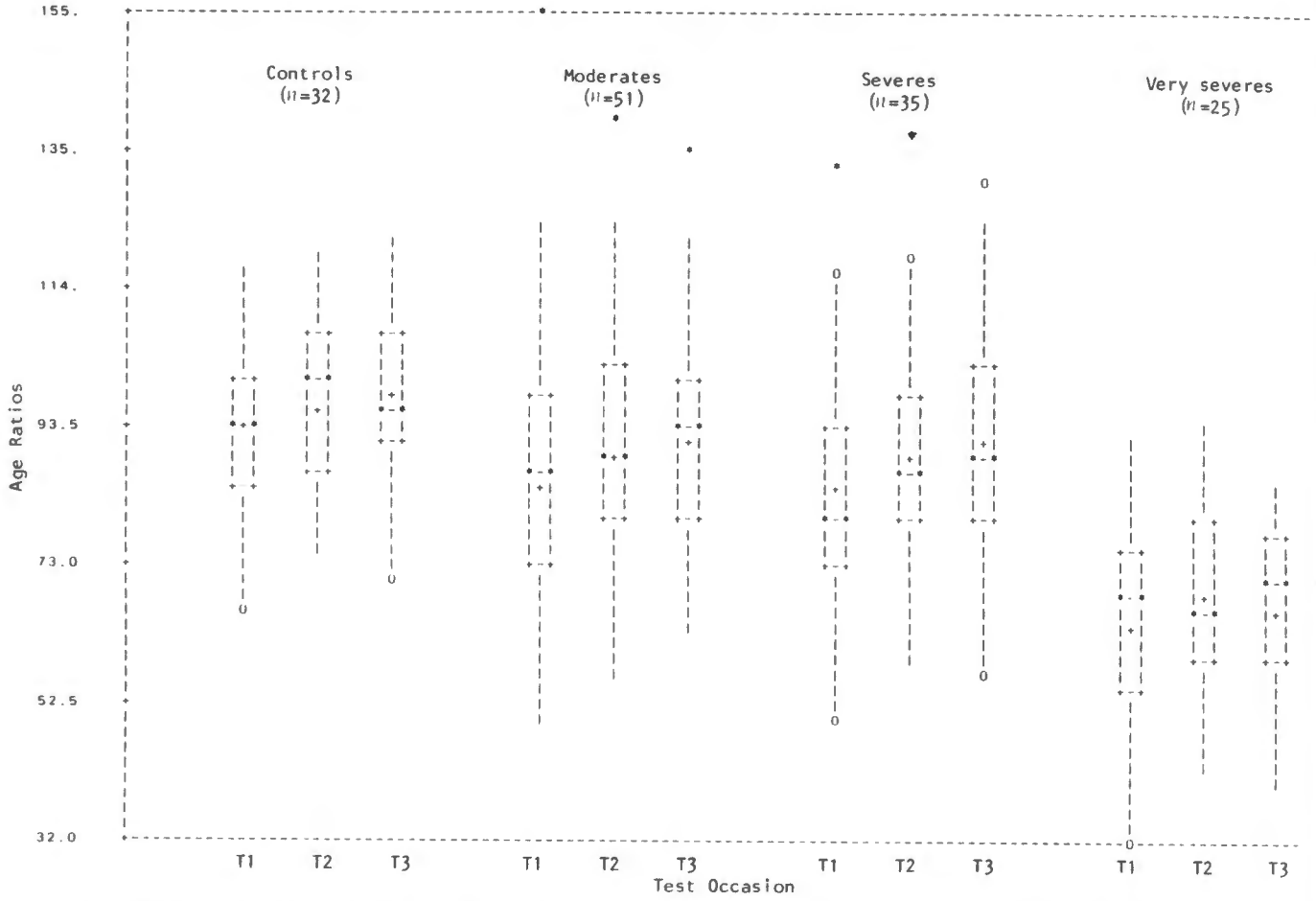


Figure 19. Detroit Motor Speed and Precision: Age Ratios at Successive Test Occasions.

Table 50

Detroit Age Ratios: Changes Over Time

Recovery Interval	Group: Controls (n=32) M	SD	Moderates (n=51) M	SD	Severes (n=35) M	SD	Very Severes (n=25) M	SD	Repeated Measures Analysis: Over Time F(1,139)	Between Groups F(3,139)
T1 to T2	4.13	10.62	4.69	12.71	4.28	9.15	4.16	9.76	20.75***	0.02
T2 to T3	1.16	8.56	1.18	10.56	2.82	7.81	-1.01	9.05	1.68	0.84

*** p<.001.

Repeated measures analysis of variance, found a significant overall increase in scores only from T1 to T2 (Table 50). Paired t-tests were used to determine the significance of changes within each group (see Table C24, p.450 for values). From T1 to T2 changes are significant within the control and all of the head-injured groups. From T2 to T3, only the gain of the severes is significant. Within each group, gains tend to be slightly larger in the first than the second recovery interval, but none of the differences between the two recovery intervals are significant. The groups do not differ significantly from each other in the size of changes that occur in either recovery period (Between groups column in Table 50).

12.4.3 Persisting Impairment on the Detroit

When persisting impairment at 1 year post-injury on the Detroit Motor Speed and Precision test is defined as an age ratio below that achieved by 95% of the controls at T3 (5th percentile=74.2), then 2 controls (4.9%), 12 moderates (22.6%), 2 severes (5.3%) and 20 very severes (74.1%) are rated as impaired. This percentage is significantly higher in both the very severes and the moderates than in the controls, [$X^2(1, n=68)=35.62$, $p<.0003$ and $X^2(1, n=94)=5.76$, $p=.016$, respectively].

12.5 Beery Developmental Test of Visual Motor Integration (VMI) (Beery, 1982)

Scores were transformed into T scores using the Beery norms. (Multiple regression analysis of the control group raw scores at T1, showed a significant effect for age, but not for sex or SES). Table 51 summarizes the T score means obtained by the groups on the successive testing occasions.

12.5.1 Differences Between the Groups

Table 51

Beery VMI: T scores at T1, T2 and T3

	Controls		Head Injured					
	(n=46)[d]		Moderates (n=53)[e]		Severes (n=39)[f]		Very Severes (n=26)	
Test Occasion	M	SD	M	SD	M	SD	M	SD
T1	**46.6_a**	7.5	**41.9_{ab}**	11.9	**40.4_b**	12.3	**33.0_c**	9.4
T2	**47.4_a**	10.2	**42.9_a**	11.8	**43.0_a**	10.9	**35.7_b**	6.8
T3	**45.3_a**	11.3	**42.8_a**	10.2	**45.7_a**	11.4	35.1_b	10.6

Note: Groups not sharing a subscript at a test occasion differ significantly on Tukey's studentized range test at $p<.05$.

[d] n=42 at T2, 41 at T3, [e] n=54 at T1, [f] n=38 at T3.

Multiple group comparisons were carried out using Tukey's studentized range test (confidence limits in Table C25, p. 451). As can be seen from the subscripts in Table 51, the very severes differ significantly from the controls and the other head-injured groups on all three test occasions, while the severes differ significantly from the controls at T1 only. The moderates score slightly lower than the controls, but none of the differences are statistically significant.

12.5.2 Changes Over Time

The distribution of T scores for those completing the Beery on all three test occasions is depicted in Figure 20. Table 52 summarizes the mean size of changes in T scores occurring over the two recovery periods and gives the F values from the repeated measures analysis of variance.

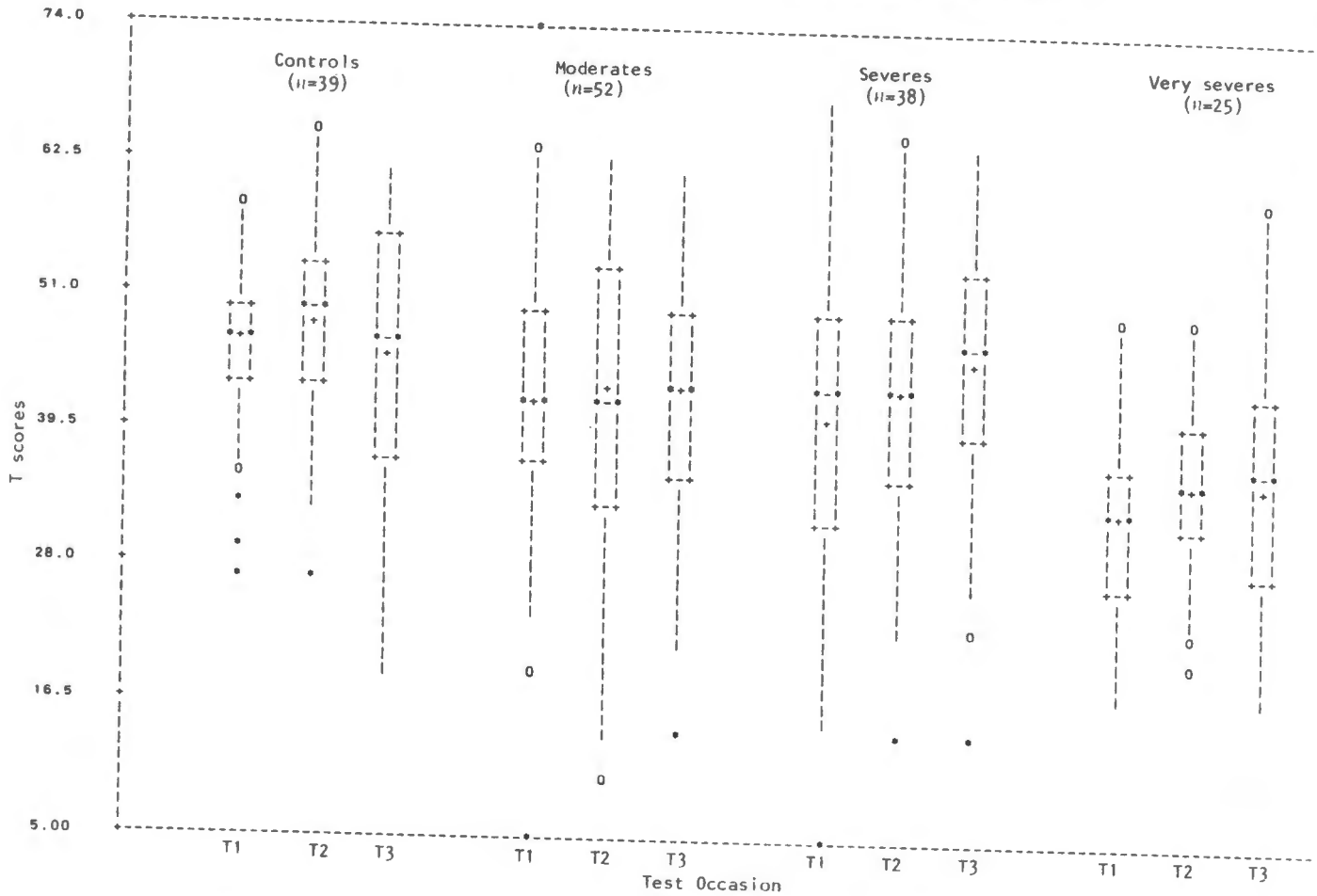


Figure 20. Beery VMI: T Scores at Successive Test Occasions.

Table 52

Beery VMI T Scores: Changes Over Time

Recovery Interval	Group: Controls (n=39) M	SD	Moderates (n=52) M	SD	Severes (n=38) M	SD	Very Severes (n=25) M	SD	Repeated Measures Analysis: Over Time F(1,150)	Between Groups F(3,150)
T1 to T2	1.38	6.03	0.33	8.86	2.76	6.65	3.34	7.53	9.82**	1.26
T2 to T3	-2.53	9.73	0.27	7.10	2.35	7.30	-0.24	9.29	0.00	2.28

** p<.01.

As is shown in Table 52 and Figure 20, changes are small in either recovery interval but there is nonetheless a significant overall increase in the Beery T scores from T1 to T2. Within the groups, paired t-tests (values in Table C26, p. 451) show the very severes and the severes to have significant increases from T1 to T2, while the gain of the severes from T2 to T3 approaches significance (p=.054). There are no significant differences within any of the groups in the size of the changes occurring in the first versus the second recovery period. Differences between the groups in the size of changes are not significant for either recovery period, but approach significance from T2 to T3 (Table 52, F=2.28, p=.082).

12.5.3 Persisting Impairment on the Beery VMI

The 5th percentile of the control group at T3 is 23.9 and 2 controls (4.9%), 2 moderates (3.8%), 2 severes (5.3%) and 5 very severes (19.2%), fall below this cut-point and are thus classified as impaired on the Beery. Differences between the control and head-injured groups are not significant on 2-tailed Fisher's exact tests.

12.6 Correlations Between the Motor and Visuographic Tests and PIQ

In order to determine the extent of overlap between the different motor and visuographic tests, the inter-relationships of the control group's scores at T1 were examined. Correlations with PIQ were also determined in order to assess the influence of intelligence on these tests. Table 53 summarizes the Spearman correlation coefficients and their significance.

Table 53

Correlations Between Motor Tests, Visuographic Tests and PIQ

	PIQ	TPEGPREF	TPEGNONP	TPEG2	SUCCPADJ	SUCNPADJ	DETROITR	TBEERY
PIQ		0.43290	0.33864	0.30832	-0.23413	-0.14983	0.46564	0.31356
		0.0106	0.0303	0.0915	0.1693	0.3259	0.0042	0.0336
		34	41	31	36	45	36	46
TPEGPREF			0.74387	0.53174	-0.21415	-0.36950	0.42893	0.03055
			0.0001	0.0025	0.2239	0.0343	0.0127	0.8638
			31	30	34	33	33	34
TPEGNONP				0.56117	-0.14618	0.00296	0.28295	-0.13401
				0.0010	0.4326	0.9853	0.1230	0.4035
				31	31	41	31	41
TPEG2					0.00134	-0.01008	0.40839	0.16984
					0.9944	0.9571	0.0251	0.3610
					30	31	30	31
SUCCPADJ						0.69160	-0.26642	-0.22634
						0.0001	0.1218	0.1844
						35	35	36
SUCNPADJ							-0.42247	-0.15687
							0.0115	0.3035
							35	45
DETROITR								0.40960
								0.0131
								36

Note. The first line gives the Spearman correlation coefficients, the second line the p value, and the third line n. Underlined coefficients, $p<.05$.

As is shown in Table 53, the various motor and visuographic tests do not correlate particularly with one another in the control group at T1: correlations between the Purdue Pegboard and the Successive finger taps are not significant, except for a low (.37) correlation on the Preferred hand. The motor measures do not correlate with the Beery and have either moderate (.41 to .43) or non-significant correlations with the Detroit. The Beery and Detroit have a moderate (.41) correlation. Correlations with PIQ are either very low or are not significant, except for moderate correlations for the Purdue Preferred Hand and the Detroit (.43 and .47 respectively). In the head-injured groups, correlations of the Detroit and the Beery with PIQ tend to be higher (eg .68 and .66 respectively for the total head-injured sample, $p<.001$).

12.7 Summary of Findings on the Motor and Visuographic Tests

1. The very severes perform at a significantly lower level than the controls and the other head-injured groups at all three test occasions on both the motor (Purdue pegboards and Successive finger tapping) and the visuographic (Detroit and Beery) measures. They also show more incoordination at T1 and T2. At 1 year about 40% show residual impairment on the motor tests, and 20% on the Beery and 76% on the Detroit.

2. There is a clear trend for the severes to have lower scores than the controls at intake (significant on the Purdue measures, incoordination and the Beery). Scores tend also to be lower at T2 (particularly on the motor tests where some Purdue and Successive finger tapping measures are significantly lower. No visuomotor scores are significantly lower than those of the controls at 1 year, and only about 5% of the group have persisting impairment on these measures. The only significant difference between the severes and the moderates is the lower score for the severes on Purdue Both hands at T2.

3. The moderates show a trend towards lower scores than the controls on the motor and visuographic tests at intake (T1), but the only significant differences are on the Purdue. No more than 5% show persisting impairment at 1 year post-injury, the same incidence as occurs by definition in the control group. An exception is the Detroit Motor Speed and Precision where 23% show persisting impairment.

4. The controls score at the expected levels for age on the motor tests, but slightly below expectations on the visuographic tests (eg a mean T score of 46.6 on the Beery and an age ratio of 92.6 on the Detroit).

5. Improvement on the motor measures tends to be greater in the head-injured, particularly the very severes, than in the control group. Recovery in the very severes and the severes is spread over the year. In the very severes, gains occur on all visuomotor measures in the first half of the year, except on the Successive finger taps which shows significant improvement only in the second part of the year. The very severes do not reach control group levels by 1 year post-injury.

   The severes show improvement on most measures in both parts of the year. They are at control group levels on the visuographic measures by 3 to 4 months and on the motor measures by 1 year post-injury.

   The moderates recover to control group levels on all visuomotor measures by 3 months. Recovery is significantly greater on most motor measures in the first than in the second recovery interval, and gains on the Purdue Preferred hand exceed those of the controls.

   The controls show improvement on the Successive finger taps and the Detroit in the first recovery interval and no further gains of significance thereafter. Their performance on the Purdue pegboards and the Beery drawings showed no practice effect at all.

6. The motor measures have low or nonsignificant correlations with each other, with the Beery drawings, and with PIQ in the control group. The Detroit has moderate correlations with the motor measures, the Beery and PIQ in the control group. In the head-injured groups as a whole, correlations of the visuographic measures, the Detroit and the Beery, with PIQ tend to be higher than in the control group.

13. MEMORY TEST RESULTS

13.1 Test Scoring

On each test occasion, a different shopping list was presented using the technique of selective reminding (Buschke 1973, 1974). Three measures were calculated to describe the results and are illustrated in Figure 21 below in a very severe and in a control. The item numbering on each trial reflects the order in which items were recalled. Recall, refers to the sum of items recalled on each of the 8 trials. Long term storage (LTS) refers to the sum of items that have once been retrieved without a reminder, those underlined in Figure 21. Consistent retrieval (CR) refers to the sum of items consistently retrieved without further reminders, those marked with a ▸ in Figure 21.

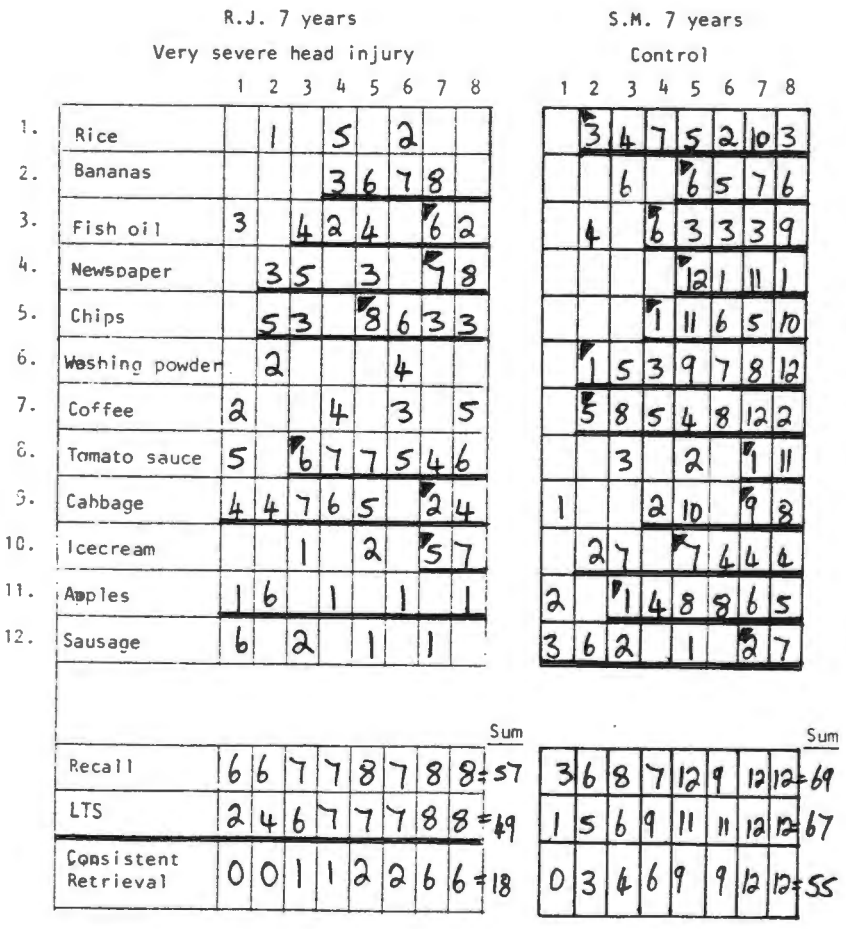


Figure 21. The Scoring of Recall, LTS and CR.

13.2 Adjustment of scores

Multiple regression analysis of the control group scores at T1 showed significant effects for age, but not for language, sex or SES. Pooled estimates of the coefficients of age were obtained by analysis of covariance using all groups and all subjects were adjusted to an age of 114, 117 and 126 months at T1, T2 and T3 respectively.

At T1

Recall adjusted = Recall total + 0.1840 (114 - age).
LTS adjusted = LTS total + 0.1760 (114 - age).
CR adjusted = CR total + 0.2746 (114 - age).

One extreme case was excluded before the LTS estimate was obtained and another before the CR estimate was obtained.

At T2

Recall adjusted = Recall total + 0.2168 (117 - age).
LTS adjusted = LTS total + 0.2447 (117 - age).
CR adjusted = CR total + 0.3278 (117 - age).

At T3

Recall adjusted = Recall total + 0.1400 (126 - age).
LTS adjusted = LTS total + 0.1722 (126 - age).
CR adjusted = CR total + 0.2432 (126 - age).

Three outlying cases were excluded before the Recall estimate was obtained and two of these were also excluded before the LTS estimate was obtained.

13.3 Differences Between the Groups

Figure 22 illustates the learning curves obtained by the different groups at T1 on Recall, LTS and CR.

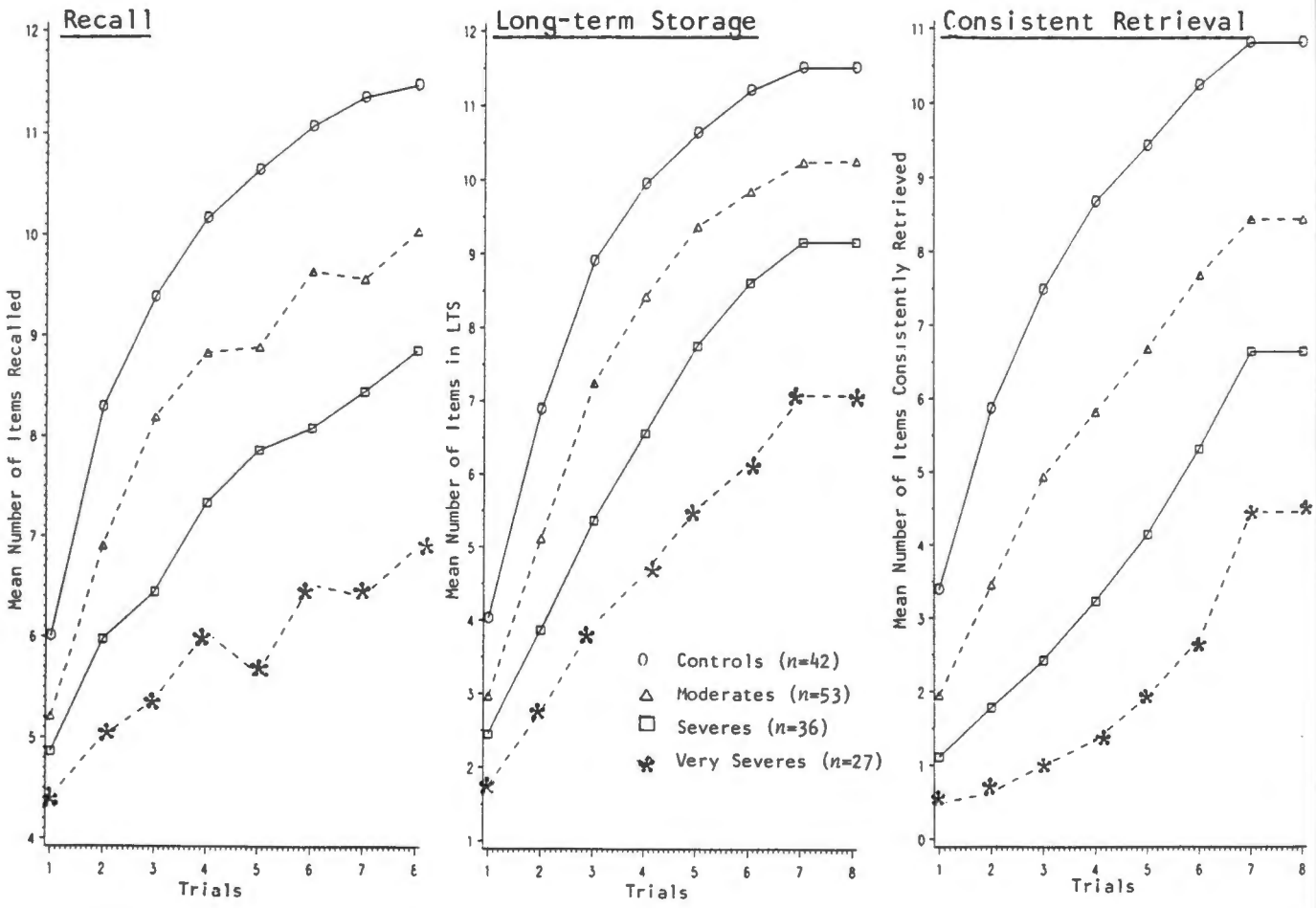


Figure 22. Learning Curves at T1 on Recall, LTS and CR

Figure 22 suggests that learning over trials differs according to the severity of injury. Analysis of differences was based on the summed scores over the 8 trials. Table 54 summarizes the means and standard deviations of the groups for each memory measure at T1, T2 and T3.

Table 54

Selective Reminding: Adjusted scores at T1, T2 and T3

Test Occasion	Controls (n=42[d]) M	SD	Head Injured: Moderates (n=53[e]) M	SD	Severes (n=36[f]) M	SD	Very severes (n=27[g]) M	SD
Recall								
T1	**78.0$_a$**	10.4	**65.9$_b$**	15.9	**59.4$_b$**	14.4	**47.7$_c$**	14.6
T2	**79.6$_a$**	12.0	**74.5$_{ab}$**	12.3	**71.7$_b$**	12.1	**63.1$_c$**	13.8
T3	**84.2$_a$**	7.0	**79 3$_b$**	7.9	**78.5$_b$**	8.3	**69.1$_c$**	12.0
Long-term Storage (LTS)								
T1	**74.3$_a$**	14.3	**62.1$_b$**	19.2	**54.6$_b$**	17.7	**40.2$_c$**	17.7
T2	**77.8$_a$**	13.5	**71.3$_{ab}$**	15.5	**69.1$_b$**	14.4	**58.4$_c$**	17.3
T3	**82.4$_a$**	7.7	**77.2$_a$**	9.7	**76.6$_a$**	10.6	**64.8$_b$**	13.7
Consistent Retrieval (CR)								
T1	**66.1$_a$**	19.6	**45.3$_b$**	25.9	**33.9$_b$**	20.8	**19.0$_c$**	17.2
T2	**69.7$_a$**	22.1	**60.8$_{ab}$**	23.6	**53.8$_{bc}$**	22.1	**39.6$_c$**	24.8
T3	**76.9$_a$**	16.0	**68.4$_{ab}$**	16.0	**63.5$_b$**	18.4	**50.5$_c$**	22.4

Note: Groups not sharing a subscript at a test occasion, differ significantly on Tukey's studentized range at $p<.05$. [a]n=41 at T2, 40 at T3; [e]n=52 at T2 and T3; [f]n=39 at T2, 37 at T3; [g]n=24 at T2.

As is shown in Table 54, when the group means are compared on Tukey's studentized range test, the very severes have significantly lower Recall, LTS and CR scores than the controls on all three test occasions. Their scores are also significantly lower than those of the moderates and the severes on all memory measures on all test occasions, with the single exception that the difference between their mean and that of the severes on CR at T2 (14.1 points) does not reach statistical significance (confidence limits -1.4 to 29.7). (Confidence limits for all between group comparisons are given in Table C27, p.452).

The severes also score significantly lower than the controls on all three measures on all three occasions, with the exception of LTS at T3, where the difference between the means (5.9) falls just short of statistical significance (confidence limits -0.25 to 12.0).

The moderates have significantly lower scores than the controls at T1. None of the differences with the control group are significant at T2. At T3, however, Recall is significantly lower and the differences between the controls and the moderates on LTS (5.2 points) approaches statistical significance (confidence limits -0.4 to 10.8).

None of the differences between the moderates and the severes on the memory measures are statistically significant. However, differences are consistently in the expected direction and are largest at T1, where the difference between the means on CR (11.4 points) falls just short of statistical significance (confidence limits -0.88 to 23.6), and least at T3.

13.4 Changes Over Time

Changes in the learning curves over the three successive test occasions are illustrated in Figure 23 on Consistent Retrieval. In the left-hand figure, the CR curves of the moderates at T1, T2 and T3 are contrasted with those of the controls, in the middle figure, the severes and controls are contrasted, and in the right-hand figure, the very severes and the controls.

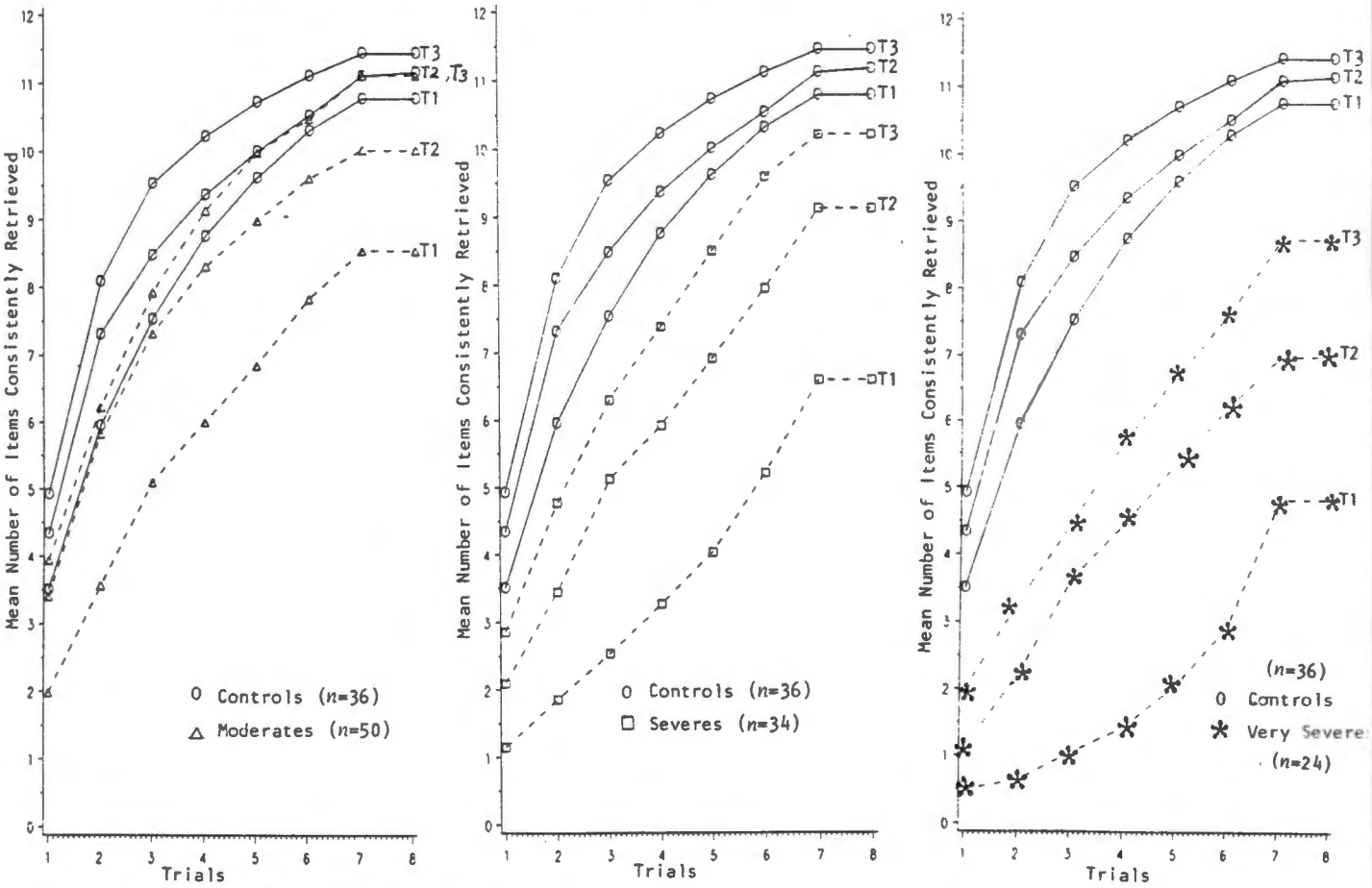


Figure 23. CR Learning Curves at Successive Test Occasions: Each Group in Comparison with the Controls

Figure 23 suggests that there is only a small improvement in CR in the control group over the successive test occasions, but larger gains in the head-injured groups, particularly from T1 to T2.

The repeated measures analysis is based on the scores summed across the 8 trials for Recall, LTS and CR, with all individuals adjusted by analysis of covariance to an age of 126 months (10.5 years). The distribution of the summed scores for those able to complete the selective reminding test on all three occasions is presented in Figure 24 for Recall, Figure 25 for LTS, and Figure 26 for CR. The mean size of changes from T1 to T2 and from T2 to T3 in summed scores, is tabulated for the groups. The significance of changes over time and between the groups is indicated by the F values in the last two columns of Table 55.

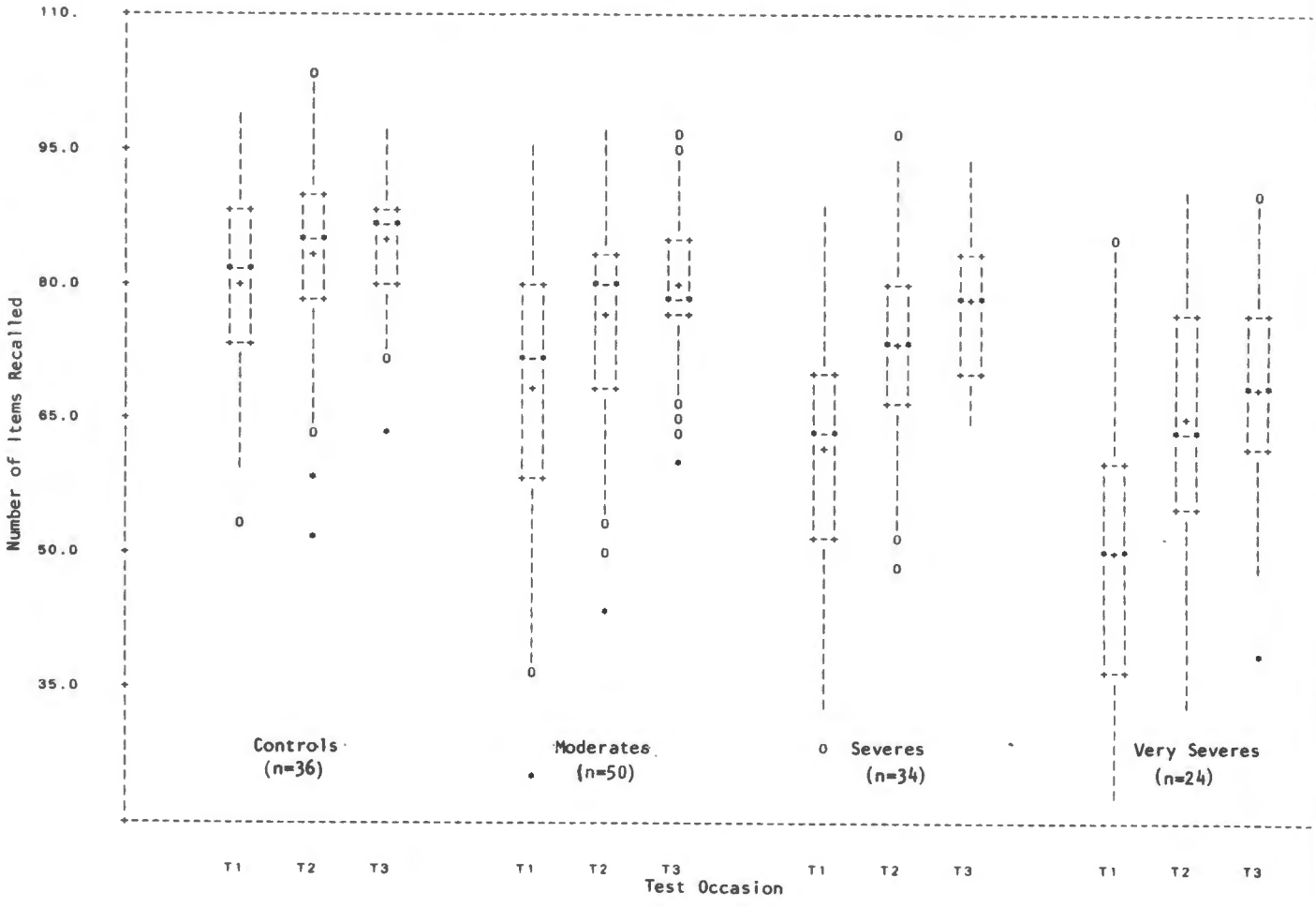


Figure 24. Recall: Adjusted Scores at Successive Test Occasions

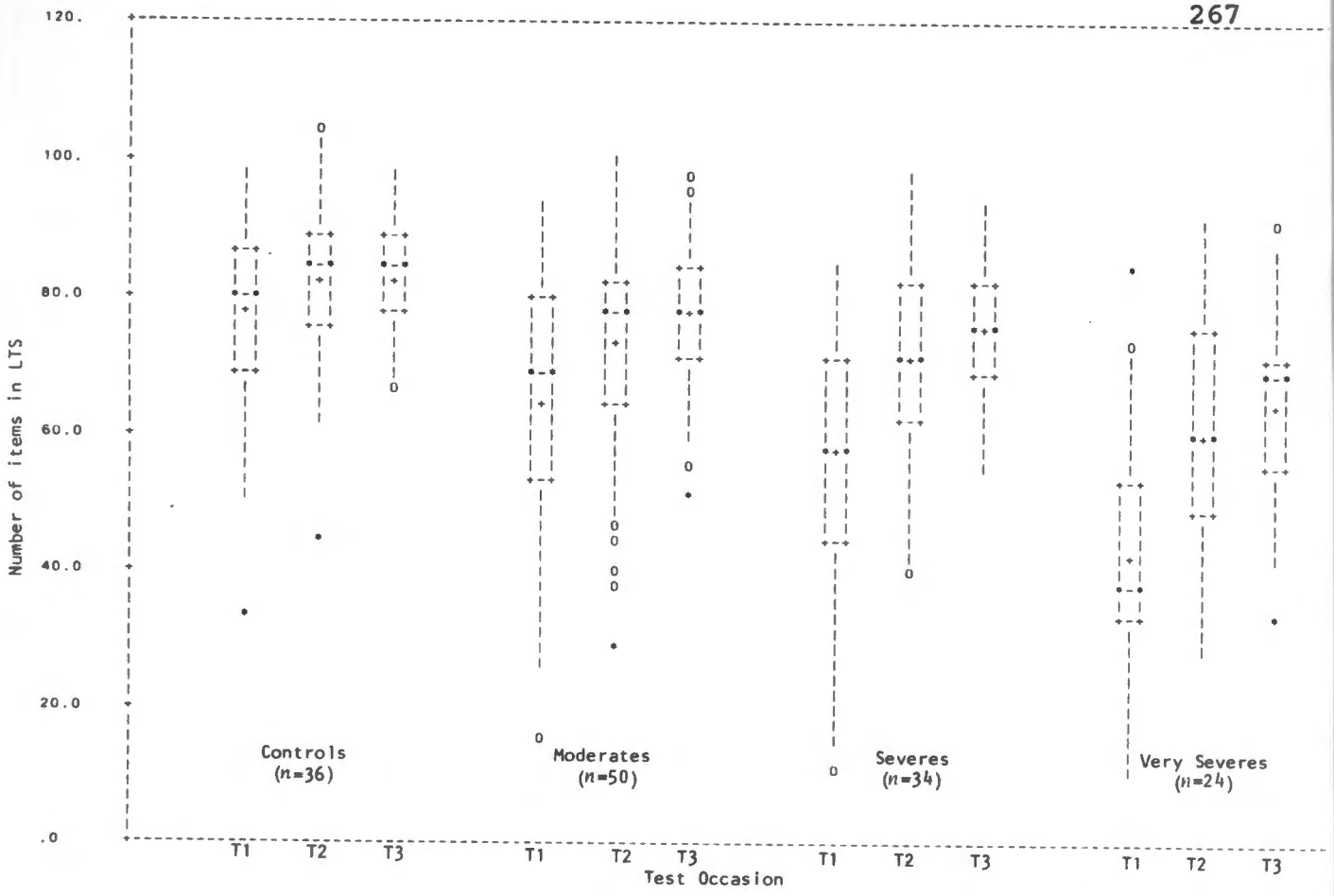


Figure 25. LTS: Adjusted Scores at Successive Test Occasions.

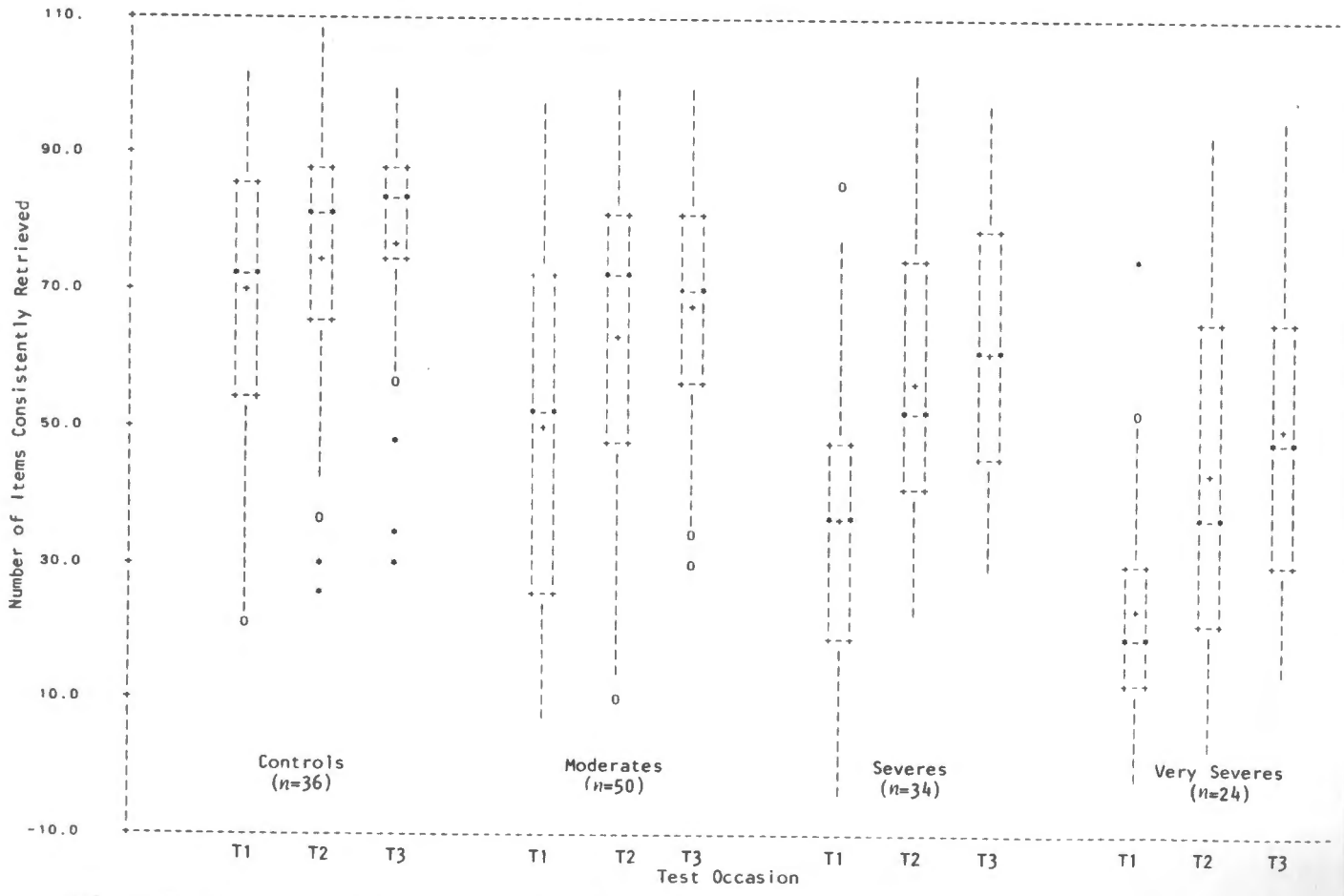


Figure 26. CR: Adjusted Scores at Successive Test Occasions.

Table 55

Selective Reminding: Mean Changes in Recall, LTS and CR Over Time

Recovery Interval	Controls (n=36) M	SD	Moderates (n=50) M	SD	Severes (n=34) M	SD	Very Severes (n=24) M	SD	Repeated Measures Analysis: Over Time F(1,140)	Between Groups F(3,140)
Recall										
T1 to T2	2.3	10.1	7.3	11.6	11.8	12.6	15.0	16.7	71.5***	6.07***
T2 to T3	1.4	6.9	2.9	9.0	4.4	10.3	3.5	12.9	13.7***	0.62
Long Term Storage (LTS)										
T1 to T2	4.7	15.6	8.2	16.9	13.5	16.1	18.5	21.6	56.33***	3.70
T2 to T3	1.1	9.1	3.8	13.2	5.2	11.8	3.7	17.5	9.64**	0.63
Consistent Retrieval (CR)										
T1 to T2	4.7	18.8	14.3	19.7	18.6	19.6	18.8	20.1	62.55***	3.44*
T2 to T3	2.3	12.6	4.1	15.4	6.4	18.3	6.7	22.2	11.30***	0.51

* $p<.05$, ** $p<.01$, *** $p<.001$.

9.4.1 Within Groups

As is shown in Table 55, there is a significant overall increase in scores on all three memory measures over both recovery periods. Paired t-tests (Table C28, p.453) show the increases for the very severes to be significant on all three memory measures within the first recovery period (T1 to T2), but not in the second recovery period (T2 to T3). The increases on Recall and LTS are significantly greater in the first than in the second recovery period for this group, and a similar trend exists on CR, although it is not statistically significant (p=.188).

For the severes, the increases on all three memory measures are statistically significant in both recovery

periods. All increases in the first recovery period are significantly greater than those occurring in the second recovery period (Table 55, Table C26).

For the moderates, increases within the first recovery period are significant on all three measures, and significant for Recall and LTS in the second recovery period, while the increase in CR approaches statistical significance ($t(49)=1.88$, $p=.066$). Increases in Recall and CR are significantly greater within the first than in the second recovery period (Table 55, Table C27).

None of the increases in the control group's memory scores are statistically significant and there are no significant differences in the gains made in the first as compared with the second recovery period (Table 55, Table C27).

13.4.2 Between Groups

On the repeated measures analysis there are significant differences between the groups in the size of the changes on Recall, LTS and CR in the first (T1 to T2) but not the second (T2 to T3) recovery period (last column, Table 55). The head-injured groups tend to have larger gains than the controls. Tukey's studentized range test was carried out to compare the size of gains in the different groups in the first recovery period. Confidence limits are given in Table C29, p. 453. The differences between the controls and the very severes are significant for all three measures, differences between the controls and the severes are significant for Recall and CR, while differences between the controls and the moderates are not significant. Differences between the very severes and the moderates approach significance on Recall (7.6, confidence limits -0.45 to 15.7), and on LTS (10.3, confidence limits -0.86 to 21.5).

13.5 Persisting Impairment on Selective Reminding

Impairment on Recall, LTS and CR at 1 year post-injury was defined as a score below that obtained by 95% of the controls at T3. The control group 5th percentile was used as the cut-point for Recall and LTS. The control group mean minus two standard deviations was used as the cut-point for CR as this cut-point made more clinical sense and still rated only 5% of the controls as impaired. The percentage of each group rated as impaired is summarized in Table 56. The significance of differences between the head injured and controls was determined on chi-square or Fisher's exact tests, as appropriate.

Table 56

Selective Reminding: Percentage with Impairment at 1 Year Post-Injury

Variable	Cut-Point	Controls (n=40) %	(n)	Moderates (n=52) %	(n)	Severes (n=37) %	(n)	Very Severes (n=27) %	(n)
Recall	<71	**5.0**	(2)	**15.4**	(8)	**24.3***	(9)	**59.3*****	(16)
LTS	<68	**5.0**	(2)	**15.4**	(8)	**18.9**	(7)	**44.4*****	(12)
CR	<45	**5.0**	(2)	**9.6**	(5)	**21.6**	(8)	**44.4*****	(12)

* Differs from controls at p<.017, *** p<.0003.

In Table 56 the percentage of children rated as having persistent impairment at 1 year post-injury increases with the severity of injury. Differences between the very severes and the controls are significant on Recall, LTS and CR [$X^2(1, n=67)=24.2$, 15.2 and 15.2 respectively, p values as indicated in Table 56] and the difference between the severes and the controls is significant on Recall [$X^2(1, n=77)=5.86$].

13.6 Confabulation

Each item "recalled" that was not on the shopping list and not an intrusion from a list presented at an earlier test, was counted as a single confabulation for that test occasion, no matter how many times it was repeated over the different trials. Table 57 summarizes the range of confabulations and the number of children confabulating in the different groups for those present on all three test occasions.

Table 57

Confabulation: Median, Range and Frequency at T1, T2 and T3

Test Occasion	Controls (n=36)		Head Injured: Moderates (n=50)		Head Injured: Severes (n=34)		Head Injured: Very Severes (n=24)	
			Number of Confabulations					
	Median	Range	**Median**	Range	**Median**	Range	**Median**	Range
T1	**1**	(0-5)	**2**	(0-7)	**2.5**	(0-15)	**2**	(0-10)
T2	**1**	(0-6)	**1**	(0-9)	**2**	(0-9)	**3**	(0-9)
T3	**0.5**	(0-5)	**1**	(0-4)	**1**	(0-5)	**2**	(0-7)
			Percentage of Children Confabulating					
	%	(n)	%	(n)	%	(n)	%	(n)
T1	**52.8**	(19)	**78.0***	(39)	**88.2***	(30)	**79.2**	(19)
T2	**66.7**	(24)	**64.0**	(32)	**67.7**	(23)	**79.2**	(19)
T3	**50.0**	(18)	**62.0**	(31)	**52.9**	(18)	**83.3***	(20)

* Differ from the controls at $p<.017$, ** $p<.003$.

Table 57 shows that the production of a few confabulated items was common in all groups. There is nonetheless a significant difference between the groups at T1 in the percentage of children producing confabulations [$X^2(3, n=144)=12.85$, $p=.005$). Pairwise comparisons of the head-injured groups with the control group, found significant differences between the moderates and the

controls and between the severes and the controls, while the difference between the very severes and the controls approaches statistical significance [$X^2(1, n=86)=6.06$, $p=.014$; $X^2(1, n=70)=10.47$, $p=.001$, and $X^2(1, n=60)=4.32$, $p=.038$, respectively].

At T2, differences between the groups in the percentages of children producing confabulations are not significant, [$X^2(3, n=144)=1.78$, $p>.10$]. At T3 the difference between the groups just reaches significance [$X^2(3, n=144)=7.75$, $p=.05$] and significantly more very severes than controls produce confabulations [$X^2(1, n=60)=6.89$, $p=.009$].

Because the production of a few (1 to 3) confabulations was common in all groups, it was decided to look at the percentage of children in the head-injured groups producing more confabulations than was the case for at least 95% of the control group. The cut-point was set at 5 confabulations and the frequency of <u>high confabulators</u> in the various groups is summarized in Table 58. The significance of differences between the head-injured and control groups was determined on chi-squared or Fisher's exact tests, as appropriate.

Table 58

<u>Confabulation: Percentage of High Confabulators at T1, T2 and T3</u>

	Controls		Head Injured					
			Moderates		Severes		Very severes	
Test Occasion	(n=36) %	(n)	(n=50) %	(n)	(n=34) %	(n)	(n=24) %	(n)
T1	5.6	(2)	10.0	(5)	32.4	(11)	16.7	(4)
T2	2.8	(1)	2.0	(1)	17.7	(6)	29.2*	(7)
T3	2.8	(1)	0	(0)	2.9	(1)	16.7	(4)

* Differ from controls at $p<.017$.

At T1, significantly more severes than controls have a high level of confabulation ($X^2(1, n=70)=8.30$, $p=.004$), but the differences between the moderates and the controls (Fisher's exact $p=.46$) and the very severes and the controls (Fisher's exact $p=.21$) are not statistically significant. At T2, significantly more very severes than controls are "high" confabulators (Fisher's exact $p=.005$), but the difference between the severes and the controls is not statistically significant (Fisher's exact $p=.052$). At T3, only the very severes tend to have a more "high" confabulators than the controls, but this difference is not statistically significant (Fisher's exact $p=.147$).

13.7 Repetition

Repetition of items was rated as none, some or marked. Children were encouraged to repeat the list in order to discover missing items, so it was expected that some repetition would be the most frequent rating. Marked was reserved for children who repeated items over and over again or very shortly after having first produced them, eg those who appeared to have forgotten what they had already said. Table 59 summarizes the frequency of repetition in the different groups, based on those completing the memory test on all three test occasions.

Table 59

Repetition: Percentage with "none", "some" and "marked" at T1, T2 and T3

Repetition	Controls (n=36) %	(n)	Moderates (n=50) %	(n)	Severes (n=34) %	(n)	Very severes (n=24) %	(n)
Intake (T1)								
None	**30.6**	(11)	**24.0**	(12)	**23.5**	(8)	**16.7**	(4)
Some	**69.4**	(25)	**74.0**	(37)	**70.6**	(24)	**58.3**	(14)
Marked	**0**	(0)	**2.0**	(1)	**5.9**	(2)	**25.0**	(6)
3 Months Later (T2)								
None	**27.8**	(10)	**18.0**	(9)	**8.8**	(3)	**4.2**	(1)
Some	**72.2**	(26)	**80.0**	(40)	**88.8**	(30)	**75.0**	(18)
Marked	**0**	(0)	**2.0**	(1)	**2.9**	(1)	20.8	(5)
1 Year Post-Injury (T3)								
None	**2.8**	(1)	**8.0**	(4)	**5.9**	(2)	**4.2**	(1)
Some	**94.4**	(34)	**90.0**	(45)	**79.4**	(27)	**70.8**	(17)
Marked	**2.8**	(1)	**2.0**	(1)	**14.7**	(5)	**25.0**	(6)

As is shown in Table 59, most children repeated items. The percentage with marked repetition in each head-injured group was compared with the percentage in the control group. Significantly more very severes than controls have marked repetition at T1, T2 and T3 [Fisher's exact p=.003, .008, and .013 respectively]. Differences between the severes and the controls and between the moderates and the controls are not significant.

13.8 Interference

Each item intruded at T2 and T3 that was from a previously learned shopping list scored 1 point on Interference. As can be seen in Table 60, the intrusion of a few items was common in all groups and there were no significant differences between the groups at either T2 or T3 in the percentage of children who showed such interference, [$X^2(3, n=143)=3.00$, $p>.10$ at T2, and $X^2(3, n=144)=0.78$, $p>.10$ at T3].

Table 60

Interference: Median, Range and Frequency at T1, T2 and T3

	Controls		Head Injured					
Test Occasion	(*n*=36)		Moderates (*n*=50)		Severes (*n*=33)		Very Severes (*n*=24)	
	Median Number of Items Intruded (range)							
	Median	Range	Median	Range	Median	Range	Median	Range
T2	0.5	(0-3)	1	(0-4)	1	(0-3)	0	(0-4)
T3	1	(0-5)	1	(0-6)	1	(0-8)	2	(0-5
	Percentage of Children with Intrusions							
	%	(*n*)	%	(*n*)	%	(*n*)	%	(*n*)
T2	50.0	(18)	54.0	(27)	66.7	(22)	45.8	(11)
T3	61.1	(22)	68.0	(34)	70.6	(24)	66.7	(16)

13.9 Memory Test Scores and IQ

There is no significant correlation between VIQ and the memory scores in the control group at T1 [Spearman rank correlations of VIQ with Recall, LTS and CR are .29, .26 and .14 respectively with $p>.05$ for each correlation]. Correlations between VIQ and memory scores in the total head-injured sample are low, but significant [.38, .35 and .37 for Recall, LTS and CR respectively, $p<.001$]. These correlations may be attributable to the association of both IQ and memory functions with severity of injury.

13.10 Memory Test Scores and PTA

In order to investigate the relationship between PTA length and memory scores more fully, the groups were further subdivided. The moderate group was divided into those with no PTA (n=12) plus 3 children with confusion lasting minutes, and those with PTA lasting from 1 to 24 hours; the severe group into those with PTA lasting 1 to 2 days and those with PTA lasting from 2 to 7 days; and the very severes into those with PTA lasting from 1 to 2 weeks and those with PTA over 2 weeks. The range of scores obtained on Recall by these PTA subgroups at T1 is depicted in Figure 27.

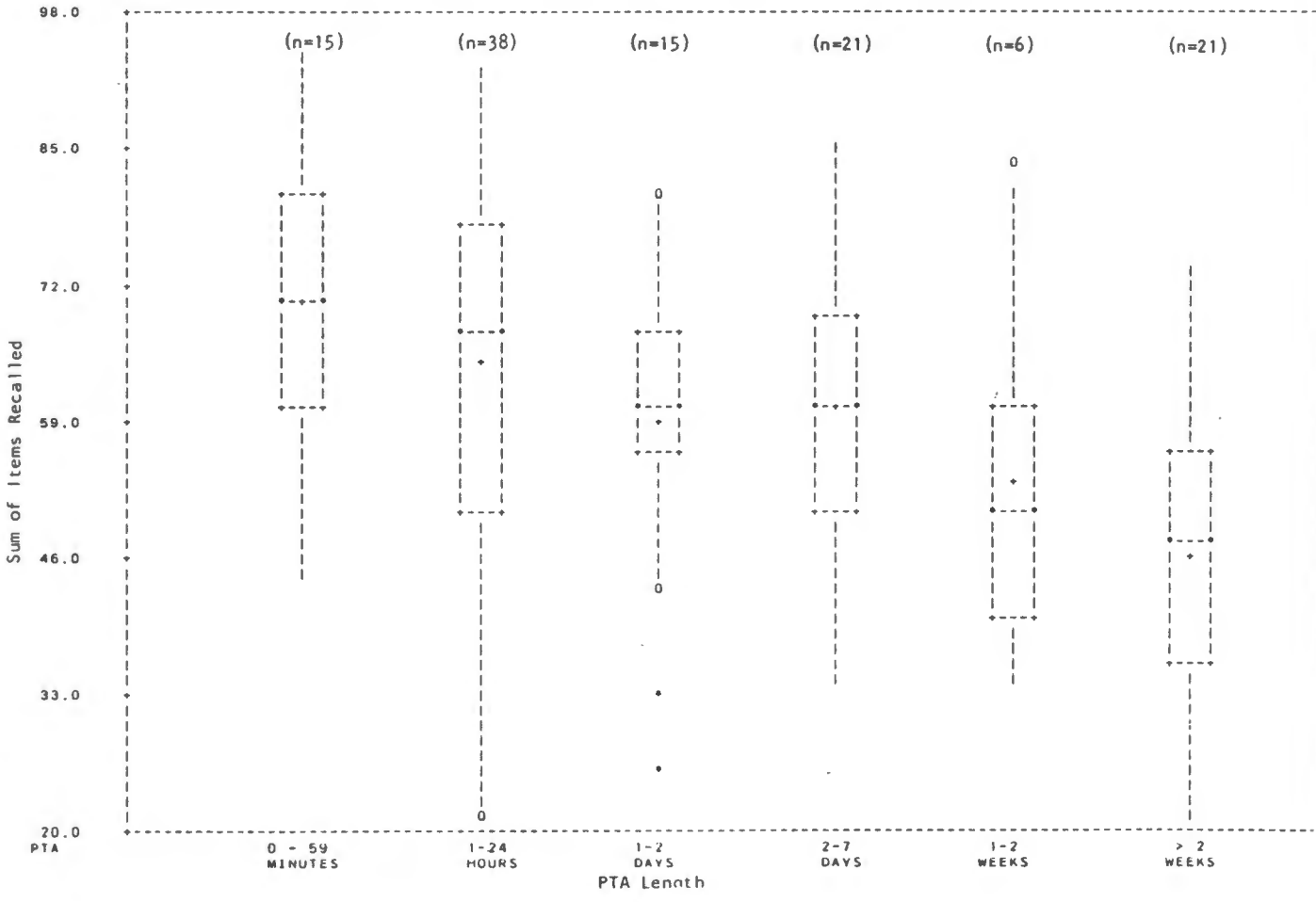


Figure 27. Recall and PTA Length

Table 61 summarizes the means and standard deviations obtained by the PTA subgroups on Recall, LTS and CR at T1, T2 and T3 and the Spearman rank correlations between scores and PTA ranked in days. Moderates with minutes of PTA were ranked as having a 0.5 day PTA, and those with between 1 and 24 hours as having a 1 day PTA.

Table 61

Recall, LTS and CR Scores and PTA Length

		Moderates		Severes		Very Severes		Spearman Rank Correlation with PTA in days
Test	Stat.	None (n=15)[a]	1-24 hrs (n=38)	1-2 days (n=15)[b]	2-7 days (n=21)[c]	1-2 wks (n=6)[d]	>2 wks (n=21)[e]	
				Recall				
T1	Mean	70.2	64.2	58.8	59.9	53.4	46.0	-0.44***
	SD	13.9	16.5	15.1	14.2	17.6	13.6	
T2	Mean	78.5	73.0	71.1	72.1	65.7	62.4	-0.32***
	SD	8.9	13.2	12.7	11.9	13.7	14.1	
T3	Mean	80.3	79.0	79.0	78.1	75.5	67.3	-0.32***
	SD	7.4	8.2	8.7	8.2	12.2	11.6	
				Long Term Storage (LTS)				
T1	Mean	65.4	60.9	54.6	54.7	46.9	38.3	-0.43***
	SD	17.3	20.0	19.1	17.0	24.1	15.7	
T2	Mean	75.0	70.0	68.8	69.2	57.0	58.8	-0.28**
	SD	11.5	16.6	15.0	14.4	19.4	17.3	
T3	Mean	77.8	77.0	77.2	76.2	70.3	63.2	-0.33***
	SD	10.1	9.7	11.3	10.3	15.0	13.3	
				Consistent Retrieval (CR)				
T1	Mean	52.4	42.5	34.5	33.5	27.8	16.5	-0.44***
	SD	24.2	26.3	20.7	21.4	24.7	14.2	
T2	Mean	67.2	58.4	51.8	55.2	40.6	39.4	-0.31***
	SD	19.4	24.8	22.2	22.4	26.3	25.2	
T3	Mean	70.5	67.6	63.9	63.2	59.5	48.0	-0.32***
	SD	12.5	17.2	20.0	17.8	27.3	20.9	

[a]n=14 at T2 and T3; [b]n=16 at T3; [c]n=23 at T2, 22 at T3; [d]n=5 at T2; [e]n=19 at T2.
** $p<.01$, *** $p<.001$.

As is shown in Table 61, there is a significant negative correlation between PTA length in days and scores on the memory measures. The association appears strongest immediately after injury and weaker thereafter.

13.11 Summary of Results

1. The very severes have significantly lower scores on Recall, LTS and CR than the controls at all test occasions. With a single exception, their scores are also significantly lower at all occasions than those of the other head-injured groups. At 1 year post-injury, about half show persistent impairment of learning ability, 25% have marked repetition and 17% have a high level of confabulation.

2. The severes similarly have lower scores than the controls on all three memory measures at all test occasions with the exception of LTS at T3. At 1 year post-injury, about 25% show persisting impairment of learning, and marked repetition occurs in 15%.

3. The moderates are impaired on all memory measures at T1, but are no longer significantly lower than the controls at T2. At 1 year, only Recall is significantly lower than in the controls and only 10% to 15% show persistent impairment of learning ability. Confabulation and repetition are no higher than in the controls.

4. The controls have only nonsignificant improvement on the three forms of the memory test on the successive test occasions. It may be that possibilities for improvement were limited by a ceiling effect on the later trials.

5. Recovery. All the head-injured groups show significant gains on the memory measures in the first recovery period and the moderates and the severes have significant gains in

the second recovery period as well. Within all head-injured groups, the gains from T1 to T2 exceed those from T2 to T3. In the first recovery period, the gains of the very severes and the severes are of a greater magnitude than those observed in the controls.

6. The memory scores are not correlated with VIQ in the control group, but are correlated with length of PTA in the head injured, with lower scores being associated with longer PTA.

14. OUTCOME CLASSIFICATION AND PREDICTION

14. OUTCOME CLASSIFICATION AND PREDICTION

Outcome at 1 year (T3) is defined in terms of the WISC-R PIQ or in terms of points on the Neuropsychological Impairment Index (NII), based on tests other than the WISC-R. In this section, problems with PIQ as a measure of adaptation are indicated, followed by the derivation of the NII. Medical and psychosocial factors contributing to BAD as opposed to GOOD NII outcome are tabulated on a univariate basis in order to compare findings with those of other studies.

As severity of injury, for example, is not taken into account in the univariate analyses, multivariate analyses (stepwise logistic regression for the NII and multiple linear regression for PIQ), are presented and the best-predicting model determined. These are exploratory analyses and all medical and psychosocial variables for which information is available, are entered. Variables on which information was complete for most cases were entered first and the best-predicting combination determined. Variables on which information was missing for several cases (eg premorbid school progress and EEG), were then added to this combination to determine whether they contributed to the prediction of outcome. Prediction of NII at T3 from test scores at T1 is also explored.

14.1 PIQ Outcome

14.1.1 PIQ and PTA Length

If a PIQ below the 5th percentile of the control group at 1 year (ie <80) is taken as indicating impairment, then 4 moderates (7.6%), 4 severes (10.5%) and 12 very severes (46%), (17% of the total head-injured sample), show impairment at 1 year. Although the percentage impaired is highest in the very severe group, there is no clear dose-response relationship between severity of injury as measured

by PTA and impairment of PIQ. When the PTA groups are subdivided to look at finer gradings of PTA against PIQ, the lack of a consistent dose-response relationship becomes even more apparent. Table 62 summarizes the median PIQ in each PTA severity subgroup and the percentage in each group with a PIQ below 80. Children with only minutes of PTA (n=3) are grouped with those with no PTA.

Table 62

PTA Length and PIQ at T3

No. PTA (n=14)	1 to 24 hours (n=39)	25 to 48 hours (n=16)	2 to 7 days (n=22)	1 to 2 weeks (n=6)	2 to 3 weeks (n=8)	>3 weeks (n=12)
PIQ Median						
94	96	102.5	92	77	83	72
PIQ Below 80						
% (n)	% (n)	% (n)	% (n)	% (n)	% (n)	% (n)
7.1 (1)	**7.7** (3)	- (0)	**18.2** (4)	**50.0** (3)	**12.5** (1)	**66.7** (8)

The inconsistent relationship between PTA length and PIQ at 1 year reflected in Table 62 may be partly a function of the small numbers in the subgroups, but this table should be compared with Table 65, p.288 which shows a consistent dose-response relationship between the categorical Neuropsychological Impairment Index and PTA subgroup. When PTA is ranked in days, the Spearman rank correlation with PIQ as a continuous variable is -.28 (p=.002), whereas it is .50 (p=.001) with NII. A low PIQ may be related to low premorbid ability, independent of the effects of head injury, and an adequate PIQ may nonetheless be associated with marked deficits on neuropsychological tests, as the later sections show.

14.1.2 PIQ and Premorbid School Progress

Premorbid IQs were not available so premorbid school progress was used as an indicator of premorbid ability. Table 63 summarizes the percentage of children with adequate or poor school progress who have a PIQ below 80 at T3.

Table 63

Percentage of Children with Adequate or Poor Premorbid School Progress having PIQ below 80

Controls		Head Injured					
		Moderates		Severes		Very Severes	
%	(n)	%	(n)	%	(n)	%	(n)
Adequate School Progress							
(n=25)		(n=29)		(n=16)		(n=11)	
4.0%	(1)	**3.5%**	(1)	**6.3%**	(1)	**27.3%**	(3)
Poor School Progress							
(n=11)		(n=19)		(n=15)		(n=10)	
9.1%	(1)	**10.5%**	(2)	**13.3%**	(2)	**70.0%**	(7)

Within each of the groups, the percentage of children with a PIQ below 80 at 1 year is more than twice as high for those whose premorbid school progress was poor as for those whose premorbid school progress was adequate (Table 63). A significantly larger proportion of the total head-injured group with poor school progress (25%) than of those with adequate school progress (8.9%) have a PIQ below 80 [$X^2(1, n=100)=4.74$, $p=.030$]. The difference in percentages approaches significance within the very severe group [Fisher's exact $p=.086$], but is not significant within the moderate and severe groups [Fisher's exact $p=.554$ and .600 respectively].

14.1.3 Adequate PIQ and Neuropsychological Impairment

The combination of a PIQ that is not below 80 with evidence of neuropsychological impairment on other tests, would make one question the adequacy of using a cut-point on

PIQ to define cognitive impairment. In the very severe group, 14 of the 26 children tested at 1 year have IQs above 80, (range 81 to 105), yet 10 of these have deficits on 5 or more of the other neuropsychological measures.

Case illustration: T.J., aged 8 years, has a PIQ of 104, but scores below 95% of the controls on the memory measures, on Renfrew naming, and on the Detroit Motor Speed and Precision test. He has borderline scores on the Purdue Pegboard; marked repetition on the memory test, and has a speech impairment and perseveration.

14.1.4 Recovery on PIQ and Transient and Persistent Impairment

The Chadwick-Rutter (1981) study stipulated that before any intellectual deficit could be attributed to head injury, there must be evidence of recovery, defined as a PIQ gain from initial to 1 year assessment of a magnitude greater than that found in 95% of the control group (ie 24 points or more). Children with such recovery and a PIQ one standard deviation or more below the control group mean at 1 year post-injury, were regarded as having persistent intellectual impairment, while those whose IQ was above this cut-point were regarded as having had transient intellectual impairment.

In the present study, 3 moderates, 2 severes and 5 very severes show a PIQ gain from T1 to T3 that exceeds that found in 95% of the controls (ie 27 or more points). Only 4 of the children with such gains, all very severes, have IQs one standard deviation or more below the control group mean at T3 (ie below 90). This means that, using the Chadwick-Rutter criterion, only 14.8% of the very severes, (3.4% of the total head-injured group), would be regarded as having persistent cognitive impairment and only 5.7% of the moderates, 5.3% of the severes, and 3.7% of the very severes (5% of the total head injured group) would be regarded as having had transient cognitive impairment. These figures appear to underestimate the amount of transient and persistent cognitive impairment indicated by the significant

lowering of test scores and the percentage found to be below 95% of controls at 1 year in the preceding Results chapters.

As neither a cut-point nor a recovery criterion on PIQ appears to detect all those found to have persisting impairment at 1 year on the neuropsychological tests, an index based on the neuropsychological battery was derived.

14.2 Neuropsychological Impairment Index (NII) Outcome

14.2.1 Derivation of NII

It was decided to create a NII by assigning 1 point for each measure on which the score at 1 year post-injury was below that obtained by 95% of the controls at the 1 year follow-up. Measures such as Digit Repetition and Token Test Part I on which a single mistake could lead to a rating of impairment and which did not differentiate the head injured from the controls, were discarded. In addition, each of the following was also awarded 1 point as they characterized less than 5% of the controls at 1 year: marked incoordination on the WISC-R mazes, more than 4 confabulations on the memory test, marked repetition on the memory test, a speech difficulty that was not immature articulation, and perseveration. Table 64 summarizes the cut-points for each measure (from the previous chapters), and the percentage of head injured falling below these points (ie those who scored 1 point on the NII in respect of that measure).

Table 64

Percentage with Impairment on Measures Contributing to the NII

		Moderates (*n*=53)		Severes (*n*=38)		Very Severes (*n*=27)	
	Cut-point	%	(*n*)	%	(*n*)	%	(*n*)
Memory							
Recall	<71	15.4	(8)	24.3	(9)	59.3	(16)
LTS	<68	15.4	(8)	18.9	(7)	44.4	(12)
CR	<45	9.6	(5)	21.6	(8)	44.4	(12)
Confabulation	>4	-	(0)	2.7	(1)	18.5	(5)
Repetition	Marked	1.9	(1)	13.5	(5)	25.9	(7)
Language							
Word Fluency	<64%	18.9	(10)	21.1	(8)	37.0	(10)
Renfrew Naming	<32	18.9	(10)	29.0	(11)	48.2	(13)
Speed (seconds)	<1.63"	3.8	(2)	2.6	(1)	22.2	(6)
Sentence Repetition	<47	5.7	(3)	5.3	(2)	22.2	(6)
Comprehension	<6	11.3	(6)	23.7	(9)	51.9	(14)
Tokens 3	<60%	3.9	(2)	14.3	(5)	19.2	(5)
Tokens 5	<10.7	15.4	(8)	11.4	(4)	36.0	(9)
Motor							
Purdue P (T)	<36	1.9	(1)	5.3	(2)	44.4	(12
Purdue NP (T)	<37	7.6	(4)	5.3	(2)	42.3	(11
Purdue Both (T)	<34	5.7	(3)	13.2	(5)	38.5	(10
Success. P (seconds)	>11.8	3.8	(2)	5.3	(2)	44.4	(12
Success. NP (seconds)	>13.0	5.7	(3)	5.3	(2)	42.3	(11
Visual Motor							
Detroit	<74	22.6	(12)	5.3	(2)	74.1	(20
Beery T	<24	3.8	(2)	5.3	(2)	19.2	(5
Other							
Speech	Impaired	-	(0)	2.6	(1)	14.8	(4
Perseveration	Any	1.9	(1)	-	(0)	18.5	(5)
Incoordination	Marked	1.9	(1)	-	(0)	11.1	(3)

The percentages impaired on the measures in Table 64, are based on the numbers attempting each test and may vary slightly from the total number in each group. In principle, children unable to do a test for reasons other than local injury, were given a NII point. This meant the child, M.K., with quadriparesis was scored as impaired on the motor tests, but taken as missing on the Beery and Tokens 5, which are tests of more than motor skills. Children who were unable to do the Token Test because they did not know colours (n=6), were given a single additional NII point, as was the child who refused the memory test.

The median number of NII points attained by each group out of a possible score of 22, is 0 for the controls, 1 for the moderates, 2 for the severes and 6 for the very severes. Figure 28 below shows the distribution of NII points within each group.

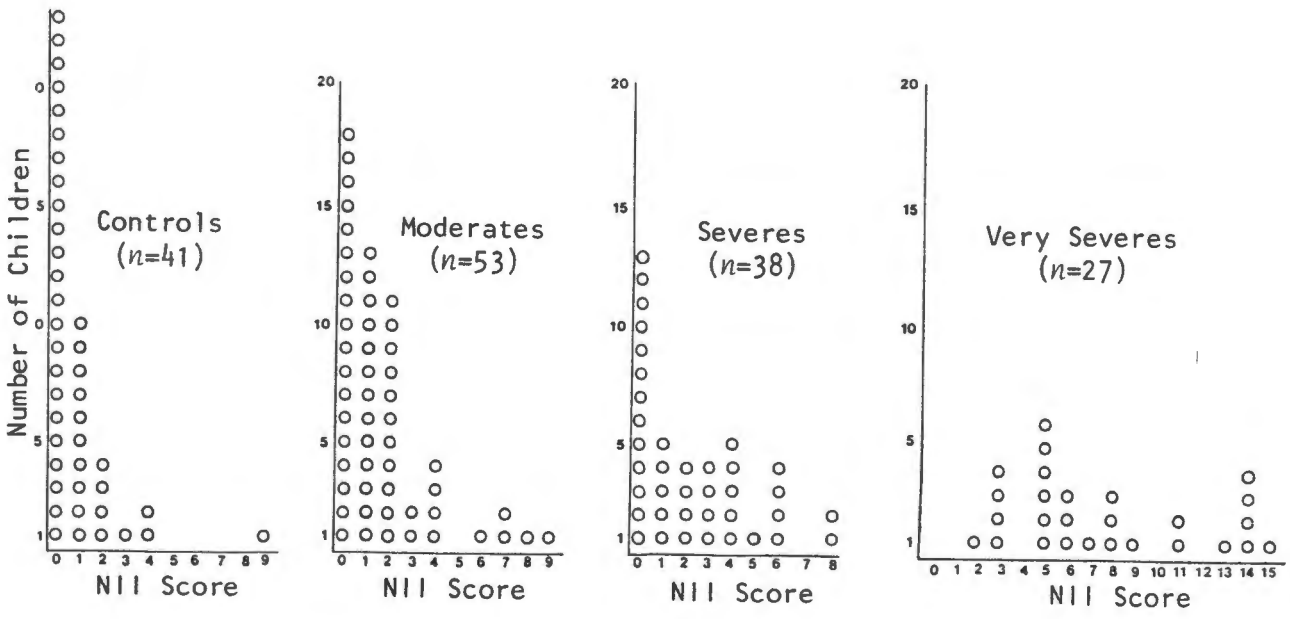


Figure 28. Distribution of NII Scores within the Groups.

As can be seen in Figure 28, NII points are not normally distributed. It was decided to define as impaired those having more NII points than at least 95% of the controls. A cut-point of 5 or more NII points, which included only 1 control (2.4%), was taken.

14.2.2 NII and PTA Length

A NII of 5 or more points (BAD NII outcome) occurred in 5 moderates (9.4%), 7 severes (18.4%), and 22 very severes (81.5%). When PTA length is further subdivided, the dose-response relationship between PTA and NII remains, as is shown in Table 65. The Spearman rank correlation between NII scores for the total head-injured group and PTA in days is .50, p=.001.

Table 65

PTA Length and NII at T3

No. PTA (n=14)	1 to 24 hours (n=39)	25 to 48 hours (n=16)	2 to 7 days (n=22)	1 to 2 weeks (n=6)	2 to 3 weeks (n=8)	>3 weeks (n=13)
NII Median						
1	1	2.5	1	5	5	9
NII ≥25						
% (n)	% (n)	% (n)	% (n)	% (n)	% (n)	% (n)
7.1 (1)	**10.3** (4)	**12.5** (2)	**22.7** (5)	**66.7** (4)	**75.0** (6)	**92.3** (12)

14.2.3 Borderline Cases

A concern was that some cases that might have been borderline on several tests, but not below 95% of the controls, might have been missed. A re-examination of all the contributing measures, giving a point to those below 90% of the controls, mostly raised the NII scores of those already rated as having a BAD outcome, but otherwise only clearly elevated the scores of 2 of the severes (previously at 4 points), to 7 points (J.L.) and 9 points (S.K.). None of the 5 very severes previously rated as GOOD NII outcome

reached 5 points with the addition of borderline points. It seemed, therefore, that with 2 exceptions, borderline cases were not being missed by using 5th percentile cut-points.

14.3 Agreement Between PIQ and NII Outcome

With both PIQ and NII dichotomized into GOOD versus BAD outcomes (<80 for PIQ BAD; ≥5 for NII BAD), for the 117 head injured rated on both indices at T3, there is agreement on 16 cases (80%) classified as BAD, and on 80 cases (82.5%) classified GOOD, ie on 82% of all head injured (kappa=0.50). In the moderate group (n=53), there is agreement on 2 cases (50%) classified BAD, and 46 cases (94%) classified GOOD, or 90.5% of all cases (kappa=0.39). In the severe group (n=38), there is agreement on 3 cases (75%) classified BAD, and 30 cases (88%) classified GOOD, or 89% of all cases (kappa=0.48). In the very severe group (n=26), there is agreement on 11 cases (92%) classified BAD, but on only 4 cases (28.5%) classified GOOD, or 58% of all cases (kappa=0.19). (Note: M.K. was unable to complete the Performance Scale and was classified on NII only).

Detailed study of the nature of the injuries, the psychosocial backgrounds, and the test patterns of the individuals on which the two outcome measures disagreed (summarized in the Discussion, p.359) suggested that the NII was more sensitive in detecting impairment attributable to the head injury than PIQ and was less influenced by psychosocial and premorbid factors. Therefore, a more detailed analysis of NII prediction than of PIQ prediction is given in the next sections.

14.4 Prediction of BAD Outcome on NII

14.4.1 Prediction from Medical Variables

14.4.1.1 Univariate Analysis

A univariate analysis of the medical variables available to a clinician from a patient's folder was first carried out. These variables are described in detail in the Method section. Table 66 summarizes the frequency of categorical (yes/no) conditions in the total head-injured sample and the significance of their association with BAD NII outcome, and Table 67 summarizes the association between the continuous variables (GCS, coma and PTA) and GOOD and BAD NII outcome.

Table 66

Medical Variables and BAD NII Outcome (Univariate Analysis)

Variable	n	% BAD	X^2	p
No complications	86	**19.8**	12.65	**.000**
Complications	32	**53.1**		
No additional injury	82	**19.5**	11.34	**.001**
Additional injury	36	**50.0**		
No surgery	83	**21.7**	11.23	**.001**
Other-surgery	18	**61.0**		
No early seizure	88	**23.9**	4.14	**.042**
Early seizure	30	**43.3**		
Not MVA	37	**18.9**	2.57	.109
MVA	81	**33.3**		
Normal EEG	33	**21.2**	1.06	.304
Abnormal EEG	61	**31.2**		
No surgery	83	**21.7**	Fisher's exact	.360
Neurosurgery	18	**33.3**		
No skull fracture	65	**32.3**	0.45	.504
Skull fracture on x-ray	49	**26.5**		
No clinical basal fracture	91	**29.7**	0.03	.871
Basal fracture, clinical	25	**28.0**		

Categorical variables with a significant association with BAD NII outcome on univariate analysis, (Table 66), are complications, additional injuries, surgery-other-than neurosurgery, and early seizures.

Table 67

GCS Score, Coma and PTA Length and NII Outcome

Variable	GOOD Outcome (n=84)	BAD Outcome (n=34)	Statistic
GCS (median score)	13	7.5	z=5.26***
Coma (median days)	0	0.63	X^2=34.82***
PTA (median days)	1	16	X^2=29.44***

*** p<.001

In those with a BAD NII outcome, GCS score was significantly lower (on the Wilcoxon test) and Coma and PTA duration significantly longer (on the median test) than in those with a GOOD NII outcome (Table 67).

Within the very severe group, an attempt was made to compare the NII outcome in those with diffuse, focal, or diffuse plus focal brain damage as based on CT scan and/or neurosurgical findings. All 6 children with CT scans showing only diffuse brain swelling, had a BAD NII outcome, as did all 6 children with only focal lesions (contusions, lacerations and haematomas with or without associated oedema), but only 6 out of the 8 children with both a focal lesion and generalized brain swelling had a BAD outcome (see Discussion, p.369). Since most children were not scanned and it was felt that both focal and diffuse lesions were missed on scanning, this variable was not included in further analyses.

14.4.1.2 Multivariate Analysis

A backwards stepwise logistic regression was carried out to determine which combination of the medical variables would best predict a BAD NII outcome. All medical variables in Tables 66 and 67 (except EEG which was available on only 91 cases) were initially entered, plus age and sex (see

Tables 68 and 69) and backward elimination was used to remove variables not contributing significantly to the outcome. Complete medical data was available on 114 cases. The variables retained in the best medical prediction model and their coefficients are:

PTA(0.274 per day); skull fracture(-0.951); other surgery(0.867); neurosurgery(0.867); male sex(0.827); complications(0.785) and basal clinical fracture(0.692). The constant =-1.407. [Coefficients of the categorical variables are multiplied by (1) if present and by (-1) if absent].

Variables with positive coefficients increase the probability of a BAD NII outcome, those with negative coefficients decrease the probability of a BAD outcome. Using the model to predict the probability of a BAD NII outcome, and taking a predicted probability >.5 as a predicted BAD outcome and a predicted probability $\leq$.5 as a predicted GOOD outcome, 25 (73.5%) of the BAD cases and 77 (96.3%) of the GOOD cases are correctly predicted, ie a total of 89.5% of cases are correctly predicted. In this model, the median probability of a predicted BAD outcome is .80 in those with a BAD outcome and .07 in those with a GOOD outcome.

14.4.2 Prediction from Psychosocial Variables

14.4.2.1 Univariate Analysis

Table 68 summarizes the contribution of the categorical psychosocial variables to a BAD outcome on the NII for the total head-injured sample and Table 69 the contribution of the continuous psychosocial variables on univariate analysis.

Table 68

Psychosocial Variables and BAD NII Outcome (Univariate analysis)

Variable	n	% BAD	X^2	p
English	21	**0.0**	10.34	**.001**
Afrikaans	97	**35.1**		
Rutter ≥13	40	**15.0**	5.23	**.022**
Rutter <13	77	**35.1**		
Adequate school	57	**17.5**	4.60	**.032**
Poor school	44	**36.4**		
No previous head injury	96	**32.3**	2.71	.100
Previous head injury	21	**14.3**		
SES 1 to 3	52	**21.2**	2.66	.103
SES 4 to 6	66	**34.9**		
Preschool	35	**22.9**	0.86	.354
No preschool	83	**31.3**		
Girl	42	**26.2**	0.22	.640
Boy	76	**30.3**		

Table 69

Psychosocial Adversity and Age and NII Outcome

Variable	GOOD Outcome (n=84)	BAD Outcome (n=34)	Wilcoxon Z	p
Adversity (median)	4	5	1.80	.072
Age (median years)	9.6	8.0	-1.43	.154

Amongst the categorical variables (Table 68), Afrikaans language, an absence of behavioural disturbance on the Rutter (score <13), and poor premorbid school progress are significantly associated with a BAD NII Outcome. A higher proportion of those without than with a previous head injury appear to have a BAD outcome, but this difference is not significant. Differences in age and psychosocial adversity

(Table 69) between those with a GOOD as opposed to a BAD outcome are not statistically significant. Premorbid school progress was the only psychosocial variable which produced significant effects on NII outcome within some of the severity subgroups as well as in the total head-injured sample. These effects are examined in the next section.

14.4.2.2 The NII and Premorbid School Progress

The distribution of BAD NII outcome (5 or more points) in the head injured with adequate versus poor premorbid school progress is summarized in Table 70. (School progress was unknown in the only control with BAD NII outcome).

Table 70

Percentage of Children with Adequate or Poor Premorbid School Progress having BAD NII Outcome

Moderates		Severes		Very Severes	
%	(n)	%	(n)	%	(n)
Adequate School Progress					
(n=29)		(n=16)		(n=12)	
0%	(0)	**0%**	(0)	**83.3%**	(10)
Poor School Progress					
(n=19)		(n=15)		(n=10)	
15.8%	(3)	**26.7%**	(4)	**90.0%**	(9)

Differences in the percentage with BAD NII outcome between those with adequate and poor school progress (Table 70) were significant in the severes, (Fisher's exact p=.043) and approached significance in the moderates (Fisher's exact p=.056), but were not significant in the very severes (Fisher's exact p=1.0). As premorbid school progress is not known for 17 of the head injured, its role in prediction will be considered only after the best predicting model based on the other variables has been determined.

14.4.2.3 Multivariate Analysis

When all the psychosocial variables in Tables 68 and 69 (except premorbid school progress) were entered in a stepwise logistic regression and those which did not contribute significantly to the outcome were eliminated, the terms remaining for the 116 cases with complete data were:

psychosocial adversity (0.241 per point); Rutter <13(0.710 if present, -0.710 if absent. The constant =-2.260.

Using this psychosocial model to predict BAD outcome on the NII, 82 (97.6%) of those with a GOOD outcome are correctly predicted, but only 7 (21.2%) of those with a BAD outcome are correctly predicted, ie only 76% of the total cases are predicted correctly. The median probability of a predicted BAD outcome is 0.36 in those with a BAD outcome and 0.24 in those with a GOOD outcome The sensitivity of prediction in the social model and the dichotomizing of predicted GOOD and BAD outcomes is not nearly as good as the medical model.

14.4.3 Prediction from Combined Medical and Psychosocial Variables

When all the medical variables (except EEG) and all the psychosocial variables (except premorbid school progress) were entered in a backwards stepwise logistic regression, the following terms remained in the equation as the most effective predictors of a BAD NII outcome:

male sex(0.927); psychosocial adversity(0.437 per point); PTA(0.280 per day); skull fracture(-1.223); basal clinical fracture(0.878); neurosurgery(0.952); other surgery(1.050), and complications(1.044). The constant =-3.141. [Categorical variables multiplied by (1) if present, (-1) if absent].

Data was complete on 112 cases. Prediction of NII outcome was correct in 25 (73.5%) of the BAD cases, and 76 (95%) of the GOOD cases (ie 88.6% of all cases). With this model, the median probability of a predicted BAD outcome was .90 in those with a BAD outcome, and .04 in those with a GOOD outcome, a better dichotomy than was achieved in the models using either the medical variables or the psychosocial variables alone.

EEG results were available for 91 of the 112 cases. Addition of EEG data to the variables already in the model for these 91 cases did not contribute significantly to prediction and the variable was eliminated. However, premorbid school progress was known for 97 of the cases and when added in, contributed significantly to the predictive power of the model. The best model for predicting BAD NII outcome in these 97 cases then became:

PTA(0.59 per day); skull fracture(-1.71); basal clinical fracture(1.67); neurosurgery(2.07); complications(2.19); poor school progress(3.17). The constant =-3.68. [The categorical variables were multiplied by (1) if present, by (-1) if absent].

Using this model, 23 (88.5%) of the BAD outcome cases and 68 (95.8%) of the GOOD outcome cases, ie 93.8% of the total, were correctly predicted. This is the best model for predicting a BAD NII outcome achieved so far. The median probability of a predicted BAD outcome is .995 in those with a BAD outcome, and .0095 in those with GOOD outcome.

14.4.4 Prediction from Neuropsychological Variables

In carrying out multiple regression analyses to predict cognitive outcome, there is an implicit assumption that the medical and psychosocial variables associated with a BAD neuropsychological outcome on this particular test battery,

will also be those associated with impairment on other neuropsychological batteries which measure similar functions. The best predictors of outcome on this particular battery at 1 year (T3), however, are likely to be the same neuropsychological variables measured at an earlier stage after injury (T1).

For exploratory purposes, scores at T1 on a range of the tests included in the battery (taking those most sensitive to head injury where there was a choice), were entered into a logistic regression as predictors of BAD NII outcome. The following variables at T1 were entered: VIQ, PIQ and FSIQ; Word Fluency, Renfrew Naming and Speed of Naming; Sentence Repetition; Token Test Part 5; Purdue Both hands, Successive finger tapping Nonpreferred hand; the Detroit and the Beery; and Consistent Retrieval. The scores were in their adjusted form as presented in the Results chapters. Using backwards elimination on the 118 cases, the following terms remained in the model:

VIQ(-0.095); Word Fluency(-0.018); Token Test Part 5 Adjusted(-0.133); Purdue Both hands(-0.132); Detroit(-0.087); and Consistent Retrieval Adjusted(-0.068). The constant term was 21.781. (Note: coefficients cannot be compared with each other as variables are on different unit scales).

This exploratory combination of neuropsychological variables at T1 correctly predicts 28 (82.4%) of the BAD cases and 80 (95.2%) of the GOOD cases (ie 91.5% of all cases) at T3. With this model, the median probability of a predicted BAD outcome is .94 in those with a BAD outcome, and .006 in those with a GOOD outcome. More cases are correctly classified by this model than by the combined medical-psychosocial model based on 112 cases (88.6%).

When the medical and psychosocial variables retained as best predictors in the earlier model are added to the six neuropsychological variables retained above, data is available on 114 cases, and the variables which best predict NII BAD outcome and their coefficients are as follows:

VIQ(-0.275); Word Fluency(-0.047); Purdue both hands(-0.125); Consistent Retrieval(-0.099); PTA days(0.417); psychosocial adversity(0.512); male sex(2.208); skull fracture(-1.367) and complications(1.646). The constant =25.366. [The categorical variables in the equation are multiplied by(1) if present, by (-1) if absent.

Prediction of outcome with this Neuropsychological-Medical-Psychosocial model is correct for 31 (91.2%) of the BAD cases and for 78 (97.5%) of the GOOD cases, ie 95.6% of all cases, the highest level of correct classifications obtained so far. The median probability of a predicted BAD outcome in this model is .999 in those with a BAD outcome and .0003 in those with a GOOD outcome, the best dichotomy achieved yet.

14.5 Multivariate Prediction of PIQ

As PIQ is a continuous variables, multiple linear regression could be used to predict PIQ at 1 year (T3). All medical and psychosocial variables (except EEG and school progress) were entered into the regression equation. Complete data was available on 111 cases. The model with the lowest Cp, ie with the smallest mean square error of prediction, included the following terms and regression coefficients:

psychosocial adversity(-1.72); PTA(-0.44); basal clinical fracture(6.05); sex (male=1, or female=2)(-7.82); English language(11.28); preschool(6.86); and the intercept, 109.268. The Cp value was 1.13, and the square of the regression coefficient was 0.41 (ie 41% of the variance was predicted).

When EEG normal/abnormal was added to these six variables for the 93 cases for which EEG was known, EEG was not included in the best-predicting regression equation. However, when premorbid school progress was added to the above six significant terms for the 98 cases for which it was known, school progress was retained in the best predicting model. For these 98 cases, the Cp-value of the model containing only the variables found to be significant in the larger data set, was 19.10 and R^2 equalled 47%. However, when school progress was included as a possible predictor in this subset of cases, the best model still predicted 47% of the variance, but the Cp value was reduced to 5.81. The coefficients of the significant variables in the model including school progress are:

PTA(-0.45); sex (male=1, female=2)(-12.46); English language(12.00); adequate school progress(12.55); and the intercept, 104.861.

PIQ at 1 year, however, is best predicted by PIQ as measured at intake, T1, [rho=.8629, p=.0001], which accounts for 74.5% of the variance.

14.6 Summary

1. A PIQ below 80 at 1 year post-injury occurred in 4.9% of controls, 7.6% of moderates, 10.5% of severes and 46% of very severes.

2. Children with PIQ above 80 could have persistent impairment at 1 year on several measures in the neuropsychological battery, suggesting that such a cut-point was missing children with cognitive impairment.

3. The Chadwick-Rutter recovery criterion of a PIQ gain from the initial to the 1 year assessment that exceeded gains shown by 95% of controls, classified only 14.8% of the very severes as having persistent impairment, and only 5.7% of moderates, 5.3% of severes and 3.7% of very severes as having had transient impairment. In terms of the preceding Results chapters, these figures appear a gross underestimation of the extent of both transient and persistent impairment after head injury.

4. A Neuropsychological Impairment Index (NII) was derived based on test scores and features at 1 year post-injury which were found in 5% or less of the controls.

5. A BAD NII outcome ($\geq$5 points) occurred in 2.4% of controls, 9.4% of moderates, 18.4% of severes, and 81.5% of very severes.

6. The severity of injury as indicated by PTA length shows a better dose-response relationship with BAD NII outcome than with PIQ below 80.

7. A greater proportion of those who had had poor, than of those who had had adequate, premorbid school progress had a PIQ below 80 and/or a BAD NII outcome at 1 year post-injury. Within the groups, poor premorbid school progress was associated with BAD NII outcome in the moderates and severes, but premorbid school status had no effect on outcome within the very severe group. For

PIQ outcome category, the opposite pattern prevailed and premorbid status was more closely associated with outcome in the very severely than in the moderately and severely head injured.

8. The two outcome measures (PIQ below 80, NII ≥5) agreed on the classification of 82% of cases as having GOOD or BAD outcome. Analysis by group of the cases on which the two measures disagreed (Discussion p.359 to p.362), suggested that the NII was more sensitive to brain injury, while PIQ was more influenced by premorbid factors.

9. In the multiple regression analyses, some of the medical variables usually associated with poor outcome from head injury (eg complications and neurosurgery) were associated with a BAD NII, but not with a PIQ below 80. PIQ was influenced by premorbid factors (eg preschool attendance and language usage) which did not influence NII.

10. Medical variables significantly associated with a BAD NII outcome on a univariate basis included GCS score, coma duration, PTA length, complications, additional injury, surgery other than neurosurgery, and occurrence of an early seizure.

11. Psychosocial variables significantly associated with a BAD NII outcome on a univariate basis included Afrikaans language, absence of premorbid behaviour disturbance, and poor premorbid school progress.

12. The probability of a BAD NII outcome at 1 year could be correctly predicted for from 88 to 96% of cases using various combinations of medical, psychosocial and early neuropsychological variables in logistic regression analyses.

13. Linear regression analysis, using a combination of medical and psychosocial variables available at intake, accounted for 41% of the variance in PIQ at 1 year. Measurement of PIQ once out of PTA accounted for 74.5% of the variance in PIQ at 1 year post-injury.

14. Knowledge of premorbid school progress could increase the accuracy of prediction of both PIQ and NII at 1 year post-injury.

15. DISCUSSION

15. DISCUSSION

15.1 Representativeness of the Sample

The head-injured children in the UCT study are comparable with those admitted to Neurosurgery or Trauma Units, rather than Emergency Rooms, in overseas studies. The severity criteria imposed (disturbance of consciousness lasting beyond 1 hour, neurological signs, a seizure or a basal skull fracture), plus the additional age and language criteria, produced a sample of 126 children between the ages of 6 and 14 years. Three children remained either unconscious or in PTA beyond 6 months. Prolonged coma is known to be associated with persistent cognitive impairment (Pelco, 1985; Woo-Sam et al, 1970) and it is the remaining, less severely injured children whose cognitive recovery is the focus of the present study.

Twenty-three percent of the sample have very severe head injury with PTA lasting over 7 days (coma lasting more than 6 hours); 32% have severe head injury (PTA 1 to 7 days) and 45% have moderate head injury (PTA less than 24 hours). In terms of the other prospective follow-up studies of children, this study contains more severely head-injured children than the studies by Black and co-workers (1969) and Klonoff and Low (1974) in which only 13% and 7% respectively had unconsciousness lasting over 1 hour. The group of very severes is comparable with the "severes" in the Chadwick-Rutter study (1981) in that no child has a PTA over 3 months, but the UCT sample contains a slightly greater proportion of children with PTA over 2 weeks. The inclusion of more severely injured children may be attributable to the fact that, whereas the milder head injuries remain in local hospitals, the more severe head injuries occurring over a wide country area are transferred to the neurosurgery units of the hospitals in the study. The moderates and severes in this study may be compared with the mildly head injured in the Chadwick-Rutter study (1981) and with the population studied by Klonoff et al (1977).

As in other studies of childhood head injuries, traffic accidents accounted for the majority of severe head injuries (Alberico et al, 1987; Bruce et al, 1979; Esparza et al, 1985), whereas the more mildly injured (moderates) have a broader range of accidents (Rutter et al, 1980). The percentage with skull fractures (42%), including compound depressed fractures (11%), is similar to that found in studies of admissions to neurosurgery units elsewhere (eg Berger et al, 1985; Black et al, 1969; Chodkiewicz et al, 1975; Rutter et al, 1980). Cerebral lacerations and/or contusions were detected in about 17% of the sample, and intracranial haematomas, as in other studies, occurred in less than 10% (Berger et al, 1985; Bruce, 1983; Zimmerman & Bilaniuk, 1983). Diffuse brain swelling alone was found in 30% of those scanned and occurred in addition to a mass lesion in another 20% of those scanned. A selective group was scanned, but these figures are similar to the 30 to 50% reported as having diffuse brain swelling by Bruce and co-workers (1981). At least 8% of the moderates and severes had diffuse brain swelling, probably more as the majority were not scanned. Bruce et al, 1981, reported 15% of those with GCS >8 as having diffuse brain swelling.

Psychosocial disadvantage is high in the families of the sample. In 40% of cases the breadwinner was unskilled, in 6% of cases neither parent had employment and in about 10%, the income came from old age pensions, maintenance or disability grants. Other studies where background conditions have been investigated, have reported that the disadvantaged are over-represented amongst the head injured (eg Klonoff 1971; Rutter et al, 1980; Desai et al, 1983; Dressler, 1984; Woo-Sam et al, 1970). In the Chadwick-Rutter study, 48% of the mildly head injured had breadwinners who were unskilled, semiskilled and unemployed (Rutter et al, 1980).

The rate of premorbid scholastic backwardness (42%) is also fairly representative. Studies have reported from one third to half of the subjects to have such backwardness (Costeff et al, 1985; Dressler, 1984; Haas et al, 1987;

Mahoney et al, 1983; Rutter et al, 1980). The percentage with behaviour disturbance prior to head injury is almost identical to that found in the Chadwick-Rutter study (Brown et al, 1981), with only about 15% of the very severely head injured having behaviour disturbance, while about 34% of the more mildly head injured (moderates and severes) show this feature. The rate of psychiatric disturbance, including alcoholism, in parents (about one third) may be somewhat higher than in other studies (eg Hjern & Nylander, 1964; Shaffer et al, 1975) but few investigators have elicited these details.

The head injured were matched for age, sex, ethnic group and socioeconomic status to a control group hospitalized for orthopaedic injuries. Psychosocial and medical data were collected at intake and all children were tested once out of PTA, 3 months later and at 1 year post-injury. Attrition is low (5%), which testifies to the good relationships maintained between the research team and the children and their parents (cf Brooks, 1987).

15.2 The Nature of Intellectual and Neuropsychological Deficits after Head Injury

Two questions were posed as to the nature of the cognitive deficit after head injury. Firstly, how severe must the head injury be to produce deficits? Secondly, what functions are affected? Table 71 summarizes the measures on which the head injured performed at a significantly lower level than the matched control group at the three test occasions.

Table 71

Summary of Measures Differentiating Head Injured from Controls at T1, T2 and T3

Function	T1	T2	T3
		Very Severes	
Intelligence	VIQ, PIQ, FSIQ (all subtests)	VIQ, PIQ, FSIQ (all subtests)	VIQ, PIQ, FSIQ (all subtests)
Motor	Purdue P, NP, Both Successive, P. NP Incoordination	Purdue P, NP, Both Successive P, NP Incoordination	Purdue P, NP, Both Successive P, NP
Visuographic	Beery; Detroit	Beery; Detroit	Beery; Detroit
Language	Words; Renfrew, Speed Sentence Repetition Pict.Ident.; Tokens 1,3,5 Speech; Perseveration	Words; Renfrew, Speed Sentence Repetition Pict.Ident.; Tokens 1,3,5 Speech; Perseveration	Words; Renfrew Sentence Repetition Pict.Ident.; Tokens 3,5
Memory	Recall, LTS, CR Marked Repetition	Recall, LTS, CR Repetition. Confabulation*	Recall, LTS, CR Repetition
		Severes	
Intelligence	VIQ, PIQ, FSIQ (D), (P.C.)	VIQ,PIQ, FSIQ (I)	VIQ (I)
Motor	Purdue P, NP, Both Incoordination	Purdue NP, Both Successive P	----------
Visuographic	Beery	----------	----------
Language	Words; Renfrew Pictorial Identification Perseveration	Renfrew Pictorial Identification	Renfrew Tokens 3
Memory	Recall, LTS, CR Confabulation*	Recall, LTS, CR	Recall, CR
		Moderates	
Intelligence	VIQ, PIQ, FSIQ (D), (PC, BD, Co)	PIQ, FSIQ (PC)	PIQ (PC, Co)
Motor	Purdue P, NP	----------	----------
Visuographic	----------	----------	----------
Language	----------	----------	Renfrew
Memory	Recall, LTS, CR	----------	Recall

I=Information, D=Digit Span, PC=Picture Completion, BD=Block Design, Co=Coding, P=Preferred, NP=Nonpreferred. Renfrew=Renfrew Naming, Speed=Speed of Naming, LTS=Long-term Storage, CR=Consistent Retrieval.

15.2.1 Severity Issues

Table 71 shows a dose-response relationship between the severity of head injury sustained and the presence of statistically significant cognitive deficits. The very severes have defective scores relative to the controls on intelligence, language, visuomotor and memory functions at intake and throughout the 1 year follow-up. Their performance tended to be at a lower level than that of the moderate and severely head-injured children as well on most measures at all test occasions. These findings support the hypothesis that the most severely injured would show the greatest impairment and would have deficits on all functions.

Generally, these findings of cognitive deficits after severe head injury are in keeping with those other studies which have found such deficits either in the early stages (Chadwick, Rutter Brown, Shaffer & Traub, 1981; Levin & Eisenberg, 1979a,b) or in the longer term (Baracchini-Muratorio et al, 1985; Black et al, 1969; Brink et al, 1970; Chadwick Rutter, Thompson & Shaffer, 1981; Colam, 1984; Costeff et al, 1985; Filley et al, 1987; Flach & Malmros, 1972; Levin, Eisenberg et al, 1982; Pelco, 1985; Raphaely et al, 1980; Winogron et al, 1984).

At the 1 year follow-up, Chadwick and co-workers found their severely head-injured children not to be defective relative to the controls on measures other than those of intelligence and visuomotor speed (Chadwick, Rutter, Shaffer & Shrout, 1981). The more extensive deficits found in the very severes in the present study may be due to (a) the inclusion of a higher proportion of children with PTA over 2 weeks (75%, versus 60% in the Chadwick-Rutter study), and/or (b) the use of measures more sensitive to head injury than those used in the Chadwick-Rutter study.

Table 71 shows the results of the severely head-injured group (PTA 1 to 7 days) to be intermediate to those of the very severes and moderates. Tested at a median of 10 days post-head injury, they showed significant deficits relative to the controls on intelligence, memory measures, language production measures, comprehension of pictures, and on some motor (Purdue Pegboards) and visuographic (Beery) measures. Three months later, they were still defective on most of these measures. At one year after injury, their scores are lower than those of the controls on memory, Verbal IQ and isolated language measures. PIQ and FSIQ and, to some extent, scores on the language measures tend to be slightly lower than those of the controls, even though the differences are not statistically significant, but scores on the motor and visuographic measures are very similar to those of the controls.

Children with this particular severity of injury have not been grouped separately in most other studies of head injury. The UCT severes may overlap with the Grade I and Grade II head injuries of Levin and Eisenberg (1979a,b) and with the mild and moderate head injuries of Winogron et al, (1984). A proportion of each of these groups were found to have neuropsychological deficits relative to test norms when tested at very varying intervals, but predominantly in the early months or first year after head injury. In the Chadwick-Rutter study, the subgroup of mildly head-injured children with PTA lasting 1 to 7 days had VIQ and PIQ significantly lower than those with PTA lasting less than 1 day, when tested at intake, but were not compared directly with the control group.

The moderates have significantly lower scores than the controls on IQ, Purdue Pegboard and memory measures at intake (Table 71). Scores on the language, visuographic and successive finger tapping measures also tended to be lower than those of the controls at intake, although differences did not reach statistical significance.

The moderates tended to do slightly better than the severes on most measures, particularly at T1, but differences were almost never statistically significant. Considering the results of the moderates and severes together, the hypothesis that those with a mild head injury would have an initial deficit relative to the controls, is supported.

The main study of comparison is that of Chadwick, Rutter, Brown, Shaffer and Traub (1981). Their mildly head-injured group (which is composed of children with moderate and severe PTA) did, in fact, have a significantly lower mean VIQ than the controls at intake, and a mean PIQ which was close to significantly lower than the control group mean. PIQ may well have been significantly lower if they had tested their subjects earlier. The mean injury-to-test interval for the mildly head injured in the Chadwick-Rutter study was 18 days, whereas the median injury-to-test interval in the present study was 6 days for the moderates and 10 days for the severes. In the children whose test sessions were split over a few days, we noted marked day to day improvement in concentration, motor control and speed. Recovery in the acute stages after head injury is expected as physiological abnormalities resolve.

The deficits that Chadwick and colleagues found in their mildly head-injured group were not regarded as attributable to head injury, but as existing prior to the injury. This was because their mildly head-injured group was not matched to the control group for SES and contained more disadvantaged children and a higher percentage with premorbid school backwardness. However, they did not determine whether it was the premorbidly backward children or others who had the low IQs. In the present study, the moderate and severe groups do not differ from the controls with respect to SES. They do contain a higher proportion of children who were backward at school premorbidly (42% versus 28% of controls), but when the influence of premorbid school backwardness on IQ is partialled out by analysis of

covariance, differences between these mildly injured groups and the controls remain significant.

Chadwick and colleagues (1981) did not assess functions other than intelligence in their mildly head injured at intake. In the present study, the reduced initial scores of the moderates and severes at intake on the memory and motor tests are unlikely to be due to differences in premorbid ability as the control group scores on these measures showed no significant effects for either intelligence or SES. Head injury, therefore, remains the most likely explanation of the deficits observed.

Klonoff and co-workers (Klonoff & Low, 1974); Klonoff et al, 1977) found impairment of intelligence and of many neuropsychological functions in a mildly head-injured sample. There was a clear pattern of many deficits present immediately after head injury, diminishing over the years, which suggests that the deficits were attributable to the head injury, and that there was recovery.

Studies of adults with mild head injuries have also found a pattern of at least transient cognitive deficits (see Binder's review, 1986; Levin, Amparo et al, 1987; Levin, Mattis et al, 1987; McMillan & Glucksman, 1987). Deficits in adults following mild head injury have tended not to be on intelligence tests, but it must be remembered that in children, intelligence tests tap more of fluid ability and less of crystallized ability than is the case for adults (Boll & Barth, 1981; Hebb, 1949).

In summary, Table 71 shows that there is persisting impairment of cognitive functions in the very severely head injured and at least transient impairment of cognitive functions in the less severely head injured. Deficits occurred even in those with PTA less than 24 hours.

15.2.2 The Pattern of Deficits

Considering the measures affected in the moderate and severely head injured (Table 71), intelligence appears to be amongst the functions most sensitive to the effects of head injury, as was suggested by Rutter and co-workers (1984), Boll and Barth (1981), and Klonoff and co-workers (Klonoff & Low, 1974; Klonoff et al, 1977). Motor dexterity as measured on the Purdue Pegboards appears sensitive, but the visuographic measures (Beery and Detroit) are relatively insensitive. Language measures, particularly of production, are sensitive to severe, but not moderate head injury, while memory tests appear very sensitive, even to moderate head injury. The specific nature of deficits on these measures is discussed in more detail in the next sections.

15.2.2.1 Intelligence

The prediction that PIQ would be more affected than VIQ by head injury, was confirmed. The control group had a mean PIQ greater than their mean VIQ (95.4 to 91.2 at T1), as was expected in a predominantly lower socioeconomic group (Kaufman, 1979; Rutter & Madge, 1976; Van den Berg, 1985). In contrast, this pattern was absent in the moderate and severely head-injured groups initially, while the very severely injured showed the reverse pattern, with VIQ significantly greater than PIQ (72.2 versus 65.7). In the very severe group, the deficit for PIQ relative to the controls at initial assessment was 30 points, compared with 21 points for VIQ. The Chadwick-Rutter study (1981) also found an initial PIQ deficit of 30 points in their severely injured, but the VIQ deficit was only 10 points. As in the present study, VIQ and PIQ deficits for the less severely injured were almost equal.

Most of the studies in which Performance tests have been reported as more affected than Verbal tests, have been based on the severely head injured (Costeff et al, 1988; Filley et al, 1987; Flach & Malmros, 1972; Levin & Eisenberg, 1979b; Raphaely et al, 1980) or on mixed severity groups (Black et al, 1969, Klonoff & Low, 1974). This

pattern has also been found in children with brain damage other than head injury (Boll & Barth, 1981; Bruce in Levin, Eisenberg, Wigg & Kobayashi, 1982; Chadwick & Rutter,1984; Dennis et al, 1981; Fletcher & Copeland, 1988; Prigatano et al, 1983; Riva & Cazzaniga, 1986; Rutter et al, 1970). Mandleberg and Brooks (1975) suggested that the Performance Scale is more affected than the Verbal Scale in head injury as these tests demand a complex integration of problem solving, perception, speed and new learning. In other words, they tap functions which are known to be particularly vulnerable to brain damage (Auerbach, 1983; Cattell, 1963; Hebb, 1942; Russell, 1986; Telzrow, 1987).

The pattern of subtest deficits in the moderates and severes suggests impairment of attention and mental control. The only verbal subtest significantly impaired in these groups at T1 was Digit Span, which is regarded as measuring distractibility (Blaha & Wallbrown, 1984; Sattler, 1982). Scanning on Picture Completion also appeared to be inadequate, and speed was reduced on the Coding subtest, although this latter finding fell short of statistical significance.

Deprivation in the sample as a whole (controls included) was evident on the Information and Comprehension subtests. Many children revealed a lack of knowledge of the world about them and of social rules. On the other hand, the Comprehension subtest also uncovered a greater exposure to adverse social conditions than is the case for more sheltered children. For example, answers about the need for police revealed a knowledge of drunkenness, dagga-smoking, violence, rape and murder. Generally, the level of expression was often poor and the meaning of answers was often implicit rather than explicit.

15.2.2.2 Visuomotor Functions

The independence of various visual-motor activities (pegboards, finger-tapping, maze drawing, putting crosses in circles, and copying figures), as suggested by Spreen and co-workers (1984), was demonstrated by the finding of no or

low correlations between these tasks in the control group. The highest correlation (.41) was between the Beery and the Detroit, both of which tasks involve paper and pencil skills. It was originally thought that the Detroit would be a test of motor speed and coordination rather than of visuographic skills, but analysis of the results collected on all groups within the first year of the study (Hemp et al, 1985), showed the Detroit scores to be as depressed as the Beery drawings in the scholastically deprived Xhosa-speaking children with moderate head injuries, while the pegboards and successive finger taps were at near normal levels. The correlation of the Detroit with PIQ (.47) in the control group also suggests that it is measuring more than motor speed.

The successive finger-tapping was a more difficult task for the children than the Purdue pegboard. The main difficulties on the Purdue related to clumsiness and slowness. On the successive finger taps, the children had to copy the examiner's sequenced movements. Practice was allowed until the child had grasped the task. In some children, it was evident that programming and not motor coordination was the problem. The finding of reduced scores in the very severes on these motor tasks is consistent with the findings of other studies which have reported deficits in manual dexterity persisting at 1 year and later in severely head-injured children (Bawden et al, 1985; Chadwick, Rutter, Shaffer & Shrout, 1981; Colam, 1984).

In the more mildly head-injured groups, the Purdue scores of the moderates and severes are significantly lower than those of the controls at T1 and there is a trend for successive finger taps to be inferior in both groups as well. At T2, the severes, (but not the moderates), have significantly lower scores than the controls on some Purdue and successive tapping measures. (The scores of the severes on Purdue Both hands at T2 are also significantly lower than those of the moderate group - the only difference between these groups that is <u>statistically</u> significant). None of the differences between the mildly head injured and the

controls are significant at 1 year. This means that manual dexterity has recovered in the moderately head-injured group by 3 months and in the severely injured group by 1 year. These findings contradict those of Chadwick and co-workers, who found the mildly head injured to have reduced manual dexterity at 1 year (Chadwick, Rutter Brown, Shaffer & Traub, 1981), and those of Klonoff and co-workers (1977) who found reduced scores on finger-tapping and maze coordination to persist for 1 or 2 years. The findings in the Klonoff study may have been influenced by the inclusion of severe as well as mildly head-injured cases. The severe group in the present study has a trend to lower scores at 1 year on the Purdue pegboards when both hands are used together.

Incoordination, as assessed from observation of pencil control on the Mazes subtest of the WISC-R, was more severe and more frequent in the very severes than in the other groups, particularly at T1 and T2. Twenty-one percent of the very severes have marked incoordination at T1 (12% at T2, 8% at T3), whereas none of the controls, and usually only 3% or less of the moderates and severes, have marked incoordination at any test occasion. Fifty-four percent of the very severes, 41% of the severes and 25% of the moderates, compared with 14% of the controls, had any incoordination on the Mazes at T1. By 1 year, any incoordination characterized 40% of the very severes, but occurred in 20% or less of the other groups. Klonoff et al (1977), report incoordination on Mazes persisting for 3 years in their mixed severity sample.

On the Detroit Motor Speed and Precision Test (Baker & Leland, 1967), children had problems in keeping their crosses within the circles, in making adequately formed crosses, in keeping their place and with directionality. Several children worked the first row from left to right and then came back from right to left. Children who worked downwards in columns were checked. Some children had to be prompted to go on from row to row on this task, and a few, all very severes, had to be prompted to continue from circle to circle. (If this test had been administered as a group

test and errors not checked as they appeared, scores would have been far lower).

The very severes were significantly poorer than all other groups throughout, and there was a trend for the moderates and severes to have lower scores than the controls, particularly at T1, although differences were not statistically significant. Similar measures of tracking and visuomotor coordination have been reported as impaired after head injury by other authors (eg Colam, 1984; Klonoff et al, 1977).

In copying the Beery drawings, the very severes and the severes had significantly lower scores than the controls at T1, and the scores of the very severes remained significantly lower throughout the one year follow-up. There was a trend for the moderates to have lower scores than the controls throughout, but these differences were not statistically significant. These findings are consistent with those of other workers who found visual-spatial difficulties on figure matching and figure-copying at one and more years after severe head injury (Baracchini-Muratorio et al, 1985; Chadwick-Rutter, 1981; Colam, 1984), but not consistent with those of Bawden et al, (1985) who found scores on developmental drawings not to be significantly reduced by head injury in the severely head injured at 1 year. Klonoff et al (1977) reported initial impairment on figure matching and copying tests, with reduced scores persisting only on some tests at 1 year post-injury in their mixed severity sample. Chadwick and colleagues found the mildly head injured also to be impaired on figure matching at 1 year. Figure matching in the Chadwick-Rutter study was regarded as a measure of attention and impulsivity rather than of visual perception (Chadwick, Rutter, Brown, Shaffer & Traub, 1981).

Errors in the present study appeared to be mostly in perception rather than execution. Designs were simplified and fragmented. Children had difficulty with orientation in space and with the integration of parts (in terms of

separation, overlap and relative size). There were also problems in motor planning, particularly with the size of angles and the orientation of lines. One child showed neglect of the right visual hemispace (drawings were reproduced in the left-hand side of the block and were incomplete on the right-hand side). Children with right hemisphere lesions showed particularly poor spatial integration of designs (parts grossly fragmented, directional confusion, and a loss of gestalt). These findings suggest visual-perceptual and motor programming problems in the head injured that have nothing to do with speed.

15.2.2.3 Language Functions

As in other studies of head injury, clinically evident aphasia was rare, but language deficits detectable on testing were fairly common (Costeff et al, 1985; Ewing-Cobbs et al, 1987; Filley et al, 1987; Heilman et al, 1971; Levin, Madison et al, 1983). Only one child, (J.G., with a PTA of 4 days and a comminuted fracture over the left hemisphere), was aphasic. She was unable to speak for several weeks and then had telegrammatic speech before normal speech returned. More subtle receptive aphasia occurred in 2 other children with closed head injuries and evidence of left hemisphere focal pathology (A.D., a severe, and M.G., a very severe).

As was expected, measures of language production and comprehension were more sensitive to the effects of head injury than measures of repetition. Digit Repetition was not significantly affected by head injury, even in the very severes. Table 71 shows the severes to have significant deficits on measures of both production and comprehension, but not of repetition , while the only language deficit detected in the moderates is on a production measure (Renfrew Naming).

It should be noted that the language measures were grouped on the basis of face validity (as in the study by Ewing-Cobbs et al, 1987). Significant correlations occurred between the language production measures in the control

group (eg Renfrew Naming and Speed of Naming; Word Fluency and Speed of Naming). A significant correlation also occurred between a repetition measure (Sentence Repetition) and a comprehension measure (Token Test Part 5), but was possibly attributable to the overlap of both measures with Verbal IQ. Details of deficits on the different language measures are discussed below.

Production Measures

Renfrew Picture Naming. Scores of the very severely and severely head injured were below those of the controls at all test occasions, while differences between the moderately injured and the controls approached significance at T1 and T2 and were significant at T3. Word retrieval, particularly on confrontation naming, has consistently been reported as impaired after closed head injury (Colam, 1984; Ewing-Cobbs et al, 1987; Levin & Eisenberg, 1979a,b; Levin, Grossman & Kelly, 1976; Sarno, 1980, 1984). Anomia has also been reported as a prominent or persistent feature of head injury which produces aphasia (Levin, Grossman, Sarwar & Meyers, 1981; Heilman et al, 1971; Newcombe, 1982; van Dongen & Visch-Brink, 1988), and of left hemisphere lesions of traumatic or other aetiology in children (Hecaen, 1983).

As anticipated, socioeconomic status affected scores on this test. Home language also affected control group scores and English-speaking children were better able to name pictures than Afrikaans-speaking children. This may reflect a tendency for upward-striving families to speak English to their children and to place a higher value on books and education (Scheffer, 1983). Scores were adjusted by analysis of covariance so that all children were regarded as Afrikaans speaking and in social class 4 (unskilled labourer). With these adjustments, the mean score of the control group at T1, aged an average of 9.5, years was below that of British 8 year-olds (34 versus 50).

Errors were common in all groups. Children who did not know the objects depicted gave the names of things they did know (eg the bear, squirrel and kangaroo, were called dogs,

cats and mice). Common misperceptions included the moon as a banana, the hinge as a book, and the thermometer as a ruler. Approximations also occurred on unknown objects (eg "man" for scarecrow; "house" for lighthouse). However, the misperceptions of the head injured were more striking, particularly at the first assessment (eg "torch" for key; "umbrella" for arrow; "tree" for lighthouse; "blankets" for mountains). Misperceptions could also be perseverated for several unknown objects (eg "pear" for waterfall and steeple; "window" for buckle, steeple and hinge). Approximations were common (eg "chicken" for duck; "blom" for blaar ["flower" for leaf]; "bird" for owl), as were part-whole substitutions (eg "carrots" for vegetables; "house" for chimney; "branch" for leaf; "hand" for finger). Circumlocutions also occurred (eg "smoke comes out" for chimney; "vissies vang" [catch fish] for net; "hemp, hande insteek" [shirt, hands in] for sleeve). Responses such as "dingus" (thing) with hand movements to show function (eg of a saw) also occurred.

Speed of Naming was significantly impaired only in the very severely injured and only at the first two test occasions. At the first test occasion, some of the very severes were so slow that the mean of the group lay above the 75th percentile. The finding of no significant deficit in speed of naming at 1 year is not consistent with that of the Chadwick-Rutter group (1981) or that of Colam (1984), but is consistent with other findings in the present study in which speeded measures generally tended to show significant recovery in the head injured.

Word Fluency. The very severes throughout and the severes at intake, found it difficult to produce the expected number of words for their age in one minute. Children succeeding on this task tended to look around the room and name what they saw, or list groups of items (eg colours, animals, furniture). This was a timed task, but children with low scores tended to run out of ideas before they ran out of time. Difficulties in verbal fluency have consistently been reported in other studies of head injury, particularly in the early stages (Chadwick-Rutter, 1981; Colam, 1984; Brooks et al, 1980; Levin et al, 1976) and have characterized the mild as well as the severely injured (Ewing-Cobbs et al, 1987; Winogron et al, 1984).

Repetition

Digit Repetition failed to differentiate the groups statistically, while Sentence Repetition was defective only in the very severes (Table 71). Other studies of children with head injury have also not found striking impairment of repetition (Colam, 1984; Ewing-Cobbs et al, 1987; Levin & Eisenberg, 1979b). Nonetheless, the qualitative nature of errors was useful clinically in alerting the examiner to possible problems in auditory processing or retention. Such children, (1 control, 4 moderates, 5 severes) had substitutions and omissions on both the repetition measures. On Sentence Repetition, they retained the sense of what was said, but not the actual words (eg "My dog chase the whitest cat"; "Die man het ons nuwe huis wit gepaint met groen

vensters" for "Die mans het ons nuwe huis wit geverf met donker groen rame"). On the memory test they confused sounds (eg "Jim" and "Tim" for "Vim", "ekskies" for "ertjies") or substituted a word that showed they retained the sense of what had been said (eg "Stuyvesant" for "cigarettes"). On the Token Test, they could not retain the first part of longer commands unless they used their fingers as markers while the instruction was completed. It is of interest that Klonoff et al (1977) also reported impairment in the mildly head injured in the processing of sounds.

The influence of Afrikaans home backgrounds on English-speaking children was evident in their verb usage on Sentence Repetition (eg "our new car have four red wheels", "Henry like to read his new book"). Home language was not found to have a significant effect on the scores of the control group children, however.

Comprehension

Comprehension, as assessed through picture identification in response to questions, and through responses to Token Test commands, tended to be significantly poorer in the severe and the very severely head injured than in the controls (Table 71). Differences between the controls and the moderates were in the expected direction although not statistically significant. This latter finding is similar to that of Ewing-Cobbs et al (1987), in which a proportion of the children with moderate/severe head injury had difficulties on the Token Test in the early stages after head injury, but scores were not significantly reduced. There appeared to be several possibilities other than a failure of comprehension that could account for deficits on both these tests, however. On Pictorial Identification, several children did not process the question accurately (eg pointed to the cat in response to "which one gives us milk?", or to the fish in response to "which one do we cook on?"), but others failed to scan the pictures adequately (eg pointed to the cow as having the longest ears without noticing the rabbit), or did not recognise the pictures (eg some thought the moon was a banana; one had never seen a

stove). Others understood the question, but did not know the answer (eg to "which one shines in the sky at night?" or "which one gives us milk?"). It was probably its multifactorial determination that made this test so sensitive to impairment following head injury.

Several children, mostly 6 and 7 years-olds, were unable to do the Token Test as they had not learned colour names. One 9 year-old, M.G., with a right occipito-parietal fracture, but diffuse brain swelling and a shift of 3mm to the right on CT scan, also could not recognise or name colours. Her background was extremely deprived and it was impossible to determine whether she had known colours prior to the injury.

Differences between the controls and the moderates and severes were not statistically significant, but it is evident that these groups had more difficulty initially than the controls in processing the simple commands on Parts 1 and 3 accurately (Table 36, p.223). At T1, only about 64% of the moderates and severes (versus 91% of controls) achieved perfect scores on Part 1, and only about 50% (versus 69% of controls) achieved perfect scores on Part 3. It seemed that lapses in concentration rather than defective comprehension led to the less than perfect scores, but an underlying deficit in the processing of sounds cannot be ruled out as Klonoff et al (1977) found significantly reduced scores, in their mixed severity group, on tests of both speech perception and sound recognition. In the present study, however, the mean scores of the moderates and severes on the more linguistically complex Part 5 are fairly similar to those of the control group, suggesting that there is no primary deficit in comprehension.

The very severe group differed significantly from the controls on the Token Test throughout follow-up. Impaired attention and retention and even spatial difficulties, as well as poor comprehension, appeared to interfere with performance. In other studies, comprehension deficits have tended to be found mostly in the early stages after head injury (Brooks & Aughton, 1979; Ewing-Cobbs et al, 1987; Groher, 1977; Najenson et al, 1978), and after LH insults (Alajouanine & Lhermitte, 1965; Hecaen, 1983).

Speech Difficulties

Mild articulation difficulties occurred in about 5% of all groups and related predominantly to immaturity. Most of the children with such lisps, stammers and mild mispronounciations were 6 and 7 year olds. In contrast, the rating of impaired speech occurred only in the severes (12.5% at T1) and very severes (32% at T1). In the early stages of recovery prior to testing, a few of the very severes were noted to be mouthing words without sounds or whispering and one had dysprosody. At the initial testing, one severe (J.G.) was aphasic and the very severe (M.K.) with quadriplegia following a gunshot wound, was only able to whisper. The majority of children rated as having impaired speech had slow or indistinct speech, sometimes with uneven voice production and difficulty in breath or pitch control. Brink and associates (1970) and Costeff and co-workers (1985) described dysarthria and slow speech in children following severe head injury, and Najenson et al, (1978) and Sarno (1980) detailed problems with articulation, respiration, resonance, stress and intonation in adults. None of the children in the present study had gross dysarthria and slurring was only evident in some as they tired.

Perseveration

Perseveration was related to severity of injury. At initial assessment it occurred in half of the very severes and 23% of the severes. It did not occur in the controls and only an isolated example was noted in a moderate. Perseveration was marked in 4 very severes and 1 severe at intake.

Recurrent perseveration, in which ideas and responses previously given recurred (Albert & Sandson, 1986), was the most common form of perseveration. Number responses on the Information subtest were sometimes perseverated (2 ears, 2 legs on a dog, 2 days in a week). Responses crediting either Jan van Riebeeck or P.W. Botha with achievements ranging from first settling in South Africa to inventing the electric light bulb, may have been examples of perseveration, but were not rated as such in isolation. On the WISC-R Comprehension subtest, themes of smoke and police commonly recurred; while one child responded "ek sal my Ma gaan sê" ("I'll tell my mother") to most of the questions. On Picture Completion, noses, eyes, legs or hair were perseverated inappropriately as missing parts. On the language tests, the Renfrew Picture Naming and, to a lesser extent, Sentence Repetition, also produced examples of recurrent perseveration. On Word Fluency and the memory test, there was occasional repetition of the same few items. Examples also occurred in conversation (eg P.W. listed all his brothers and sisters as aged 9 and in Std 2).

Continuous Perseveration, the inappropriate prolonging of behaviour, also occurred, but far less frequently and only in those showing recurrent perseveration as well. Examples included the continuation of finger tapping; 30 plus dots being drawn for the 8 dot Beery design and digits forward turning into counting. Phrases could also be perseverated (eg "en toe, en toe, en toe..." or "dit is, dit is..." ["and then...", "it is..."]).

Stuck-in set perseveration was also noted in a few of the children who had recurrent and continuous perseveration as well (eg continuing to tap their fingers when asked to do pegboards). More striking in a few, however, was an inability to grasp set and a tendency to persist in their own approach (eg naming on Picture Completion, copying the Coding symbols in sequence, or pointing to each Token in turn), regardless of repeated instructions. Two children (S.S. and R.Ja.) were unable to continue any task (pegboards, crosses in circles, Beery drawings, Coding, etc) without repeated prompting.

Thomsen (1975) and Albert and Sandson (1986) reported perseveration in association with word finding difficulties. Children in the very severe group with perseveration at T1, have fairly widespread residual neuropsychological deficits at 1 year. In the severe group, however, 8 out of the 9 children with perseveration at T1 have residual impairment at 1 year either exclusively or predominantly on language tests, particularly Renfrew Naming. Three have left focal lesions, none have right focal lesions. Interestingly, the 3 very severes with continuous perseveration on the Beery dots, all had right hemisphere focal lesions superimposed on diffuse pathology (R.B., S.S., and P.W). This is consistent with the finding of Albert and Sandson (1986) that where patients with right hemisphere lesions had perseveration, it was of the continuous type.

Echolalia

Echolalia was noted in some children while still in PTA. By the time of testing, it was no longer evident in conversation and it was not rated as severe in any of the children. Echolalia was associated exclusively with severe head injury and occurred in 5 of the very severes (18%) and in 1 severe (2.5%) at the initial assessment. All except 1 of the 6 children with echolalia also showed perseveration. Three of 6 were generally talkative and disinhibited. Mitigated echolalia (repetition of questions) which Stengel (cited Levin, 1981) regarded as less severe than automatic

echolalia (parroting) occurred in 4 children, all of whom had a GOOD outcome on the NII at T3. Automatic echolalia occurred in 2 children: P.W. echoed the last word of questions, while R.Ja. echoed each memory item and digit as they were presented and was unable to inhibit this response. Both these children had a BAD NII outcome with high scores (14 and 15) at T3. None of the children exhibited echolalia at the second or third assessments.

15.2.2.4 Memory

The selective reminding memory test appears to be one of the most sensitive measures within the battery for detecting impairment after head injury (Table 71). In the control group, scores on memory measures were not correlated with IQ, nor were they affected by SES. Such deficits as were found therefore, are likely to be due to the head injury. Across the groups, there was a clear dose-response relationship and lower scores were associated with increasing length of PTA. Even the mildly head injured showed significant deficits at initial assessment and residual deficits at 1 year.

The studies of Molodetskikh (1987) and of Levin and Eisenberg (1979b) found early phase memory deficits in at least a quarter of mildly head-injured children and adolescents, as well as in those with severe head injuries. Lewis and colleagues (1987) have reported deficits in verbal recall in children 1 year after mild head injury. Systematic follow-up studies of memory functions in mildly head-injured children are lacking, but studies of the severely head injured have found long-term deficits (Baracchini-Muratorio et al, 1985; Fuld & Fisher, 1977; Gaidolfi & Vignolo, 1980; Levin, Eisenberg, Wigg & Kobayashi, 1982; Richardson, 1963).

Chadwick and colleagues did not assess memory functions in the mildly head injured. In the severely head injured they found paired-associate learning to be defective at initial assessment, but not at 4 months or 1 year (Chadwick, Rutter, Shaffer & Shrout, 1981). There were only six paired-associates and it is possible that this task was not sufficiently taxing as a test of memory. Reduced scores at intake might have been due to generalized cognitive impairment which would have affected concentration and STM (Brooks, 1975; Gronwall & Wrightson, 1981; Kopelman, 1987).

The relationship between the extent of the memory deficit and the length of PTA confirms what has been found in studies of head injury in adults (Brooks, 1976; Brooks & Aughton, 1979a,b); Brooks et al, 1980; Gronwall & Wrightson, 1981; McLean et al, 1983; Parker & Serrats, 1976; Richardson & Snape, 1984; Russell & Smith, 1961).

In the present study, the memory deficit was evident in recall, storage and retrieval. In all the head-injured groups, fewer items were recalled on each trial; fewer were recalled even once without a reminder (evidence of long-term storage), and consistent retrieval of items in storage was very poor. These measures are obviously dependent upon one another (Spearman rank correlations ranged from .87 to .92) and were not directly contrasted. However, examination of the size of the deficits on each measure in the head-injured groups in relation to the control group (Table C27, p.452), or calculation from Table 54 (p. 263) of the head-injured groups' mean scores as a percentage of the control group's mean scores, shows the deficit to be greatest in consistent retrieval: the very severes at T1 have deficits relative to the controls of 30, 34 and 47 points on Recall, LTS and CR respectively, and their mean scores on Recall, LTS and CR are 61%, 54% and 29% respectively, of the control group's mean score. This suggests that the defect is one of memory storage and retrieval and not just one of generalized cognitive impairment affecting attention and therefore recall.

Successful approaches to the task included listing the items in the same order on each trial, with new items added at the end; grouping items by their initial sounds (eg Vim, vis and vuurhoutjies, or eiers and ertjies); or by association (eg fruit and vegetables; bread and cheese; rice, sausages and tomato sauce; cooldrink and chocolate), or visualizing the sections of a supermarket. Some of the head injured were too distracted by reminders of what they had missed to maintain any serial order list, and demonstrated only short-term memory, (ie recall of the items presented last). Generally, they made little use of grouping strategies. A passive approach to learning characterized by limited use of strategies, was noted in head-injured adults by Levin and Goldstein (1986) and by Crosson and associates (1988). To some extent the absence of strategies may be considered an age-related phenomenon (Kail, 1979), but scores were adjusted by analysis of covariance to allow for age, and at least some of the younger children were observed to be using strategies.

A few confabulations, (from 1 to 3), were common in all groups, but there was a trend for more of the head injured than of the controls (81% versus 53% at T1) to produce some confabulations and a trend for a greater proportion of the severes and very severes to be high level confabulators (ie produce 5 or more confabulations). There were also qualitative differences between the groups. Whereas the controls tended to eliminate confabulated items over trials, (the examiner said "no" to incorrect items), the very severes, particularly, were noted to perseverate incorrect items. This is a failure of error correction, as noted by Walsh (1985) and attributed to frontal lobe damage after head injury. Some of the children could perceive their errors, but still could not eliminate them (for example, they might continue to say "milk, no, not milk" on each trial).

Most confabulations reflected a lack of temporal context in that the items were from the children's own typical shopping lists rather than the list given by the examiner (eg tobacco, candles and yeast), but a few seemed quite out of context (eg pen, flowers, doll). A higher frequency of extra-list intrusions in adult head injured than in controls was reported by Brooks (1975), Levin and Goldstein (1986) and Levin, Grossman and colleagues (1979). In the present study, there were no differences between the head injured and the controls in the number of intrusions that were from previous lists. Brooks (1975) found the head injured to have fewer such intrusions than controls, while Parker and Serrats (1976) found the head injured to perseverate 2 or 3 words from previous lists.

Marked repetition in the present study could be of either correct or incorrect items and was scored when the child either reported the same items over and over, or produced them again in fairly short succession. Marked repetition characterized a quarter of the very severes throughout (and only 1 control on any occasion). Some of the head injured appeared to have forgotten what they had just said.

15.3 Patterns of Recovery

15.3.1 General Issues

The questions posed about recovery in the first year after head injury in children concerned whether the different severities of injury improve at different rates, whether there is a return to the pre-injury state, and the rate and magnitude of improvement of specific abilities. It can be seen in Table 71 that a return to the pre-injury state (defined as control group level) varies according to the severity of head injury sustained, and that, in the milder head injuries, different functions recover at different rates.

The very severes, as was hypothesized, have not reached control group levels at 1 year, except on a few isolated measures. The severes reached control group levels on visuographic measures by 3 months and motor measures by 1 year, but are still below the controls on some intelligence, language and memory measures at 1 year. The moderates, as was hypothesized, reached control group levels on almost all measures by 3 months. Functions not at the control group level in the moderates at 1 year include intelligence, memory and picture naming.

Table 72 summarizes the measures on which the different groups, including the controls, show significant improvement in either of the two recovery periods: from the initial assessment to that 3 months later (T1 to T2), and from then to 1 year after injury (T2 to T3). Measures on which there was significantly greater improvement in one recovery interval than in the other within a group, have an asterisk, while measures on which the head injured show a significantly greater amount of improvement than the controls, are underlined.

Table 72

Summary of Measures Showing Significant Improvement T1 to T2 and T2 to T3

2/12. 1yr.

Function	Recovery 1 (T1 to T2)	Recovery 2 (T2 to T3)
	Very Severes	
Intelligence	PIQ, FSIQ (D; PC, Co, Ma)	VIQ, PIQ, FSIQ (C, A, D; BD, Ma)
Motor	Purdue P*, NP*, Both	Successive P, NP
Visuographic	Detroit, Beery	---
Language	Words; Renfrew; Speed; Sent.; Tokens 3,5	Renfrew; Speed; Token
Memory	Recall*, LTS*, CR	---
	Severes	
Intelligence	PIQ, FSIQ (C; PC, Co, Ma)	PIQ, FSIQ (PC, Co, Ma)
Motor	Purdue P, NP Both; Successive NP	Purdue NP; Successive P, NP
Visuographic	Detroit; Beery	Detroit
Language	Renfrew; Tokens 1,5, Perseveration	Speed; Picture-Identification; Tokens 5
Memory	Recall*, LTS*, CR*	Recall, LTS, CT
	Moderates	
Intelligence	VIQ, PIQ*, FSIQ* (C, A, D; PC, BD, Co*, Ma)	PIQ, FSIQ (I; PC)
Motor	Purdue P*, NP, Both*; Successive P, NP*	Successive P
Visuographic	Detroit	---
Language	Renfrew*, Speed; Tokens 1,5	---
Memory	Recall, LTS, CR*	Recall, LTS
	Controls	
Intelligence	VIQ, PIQ*, FSIQ* (PC, BD, Ma*)	VIQ, FSIQ (C; Co)
Motor	Successive P, NP	---
Visuographic	Detroit	---
Language	Renfrew	Tokens 5
Memory	---	---

* Improvement significantly greater in Recovery 1 than in Recovery 2.
Underlined: Improvement significantly greater than in Control group.
I=Information, C=Comprehension, A=Arithmetic, D=Digit Span, PC=Picture Completion, BD=Block Design, Co=Coding, Ma=Mazes, P=Preferred, NP=Nonpreferred, Renfrew=Renfrew Naming, Speed=Speed of Naming, LTS=Long-Term Storage, CR=Consistent Retrieval.

The hypothesis that the most severely head injured would show the greatest amount of improvement is not supported as, with a few exceptions (Table 72), their gains are not of a larger magnitude than gains in the control group. Does this mean that the gains they show have to be attributed to practice effects rather than recovery? The general pattern of findings in Table 72 suggests that recovery, rather than practice, accounts for the gains in the head injured. As the deficit was greatest initially, so the greatest amount of recovery appears to occur in the first months after head injury. Recovery in the acute stages occurs as physiological abnormalities resolve (ie as cerebral blood flow and oxygenation return to normal, oedema subsides, cellular infiltrates resolve, pressure associated with haemorrhage or dynamic changes in cerebrospinal fluid dissipates, and neuronal input is restored (Kertesz, 1979; Teasdale & Mendelow, 1984).

The two physiological processes postulated as the major mechanisms of recovery after head injury are restitution of function, which refers to spontaneous physiological processes occurring in the acute phase after head injury, and substitution of function, which is not time-constrained and which continues into the chronic phase of recovery. Restitution of function includes such processes as axonal regeneration, collateral sprouting, denervation supersensitivity and release of neurons from the inhibitory effects of transneuronal and transynaptic degeneration and diaschisis. Substitution of function implies the dynamic reorganization of neural substrates (intra- or interhemispherically) to maintain or restore functions and the alteration of behavioural strategies (Finger & Stein, 1982; Luria, 1980, 1973; Rothi & Horner, 1983; St.James-Roberts, 1979). In the more severely head injured, recovery over a longer period would be expected on the basis of substitution of function, which may continue as long as there is learning potential (Rothi & Horner, 1983).

In Table 72 it is evident that the very severes show recovery which continues over the second half of the year. They also show improvement on more measures than do the moderates and the controls. All of the head-injured groups have some gains which are of a greater magnitude than those found in the control group. These are few, however. The requirement put forward by the Chadwick-Rutter group (1981) that in order for a cognitive deficit to be attributed to head injury, there should be recovery of a greater magnitude than that found in the controls, should be reconsidered and possibly discarded. Those with the greatest impairment may have the least potential for recovery (cf Pelco, 1985).

If the very severely head injured who had the clearest dose-response relationship between severity of head injury and the size of the initial deficit do not show greater gains over time than the controls, why should the less severely head injured who had lesser deficits? The mildly head-injured group includes children who tend to have had premorbid behaviour problems and to have come from homes where the parents have psychiatric disturbances and there is disharmony. Premorbid school backwardness is high. It is unlikely that such children would have the cognitive resources to make gains of a greater magnitude than the controls whose concentration and information-processing are not impaired and who are able to learn at the first test session strategies which they can retain and apply at the second test session.

In both the severely and mildly head injured, it is likely that it is improvement in the underlying condition (ie recovery from head injury), that accounts for the gains on tests and not practice effects. Various workers have shown that there are no gains on tests after brain damage unless there is improvement in the underlying condition (Klonoff and Low, 1974; Seidenberg et al, 1981; Shatz, 1981). It may be more important to consider the nature and timing of gains than their magnitude. The measures on which

the various groups improve differ and patterns within the groups are considered below.

15.3.2 Practice Effects in the Controls

Control group gains have to be regarded as practice effects or as recovery from the nonspecific, anxiety-provoking effects of having an accident, suffering injury and undergoing the strange experience of hospitalization (cf Richardson & Snape, 1984). Improvement due to maturation was eliminated by the use of age group norms or by adjusting scores for age by analysis of covariance. It should be noted that in children, unlike adults, a failure to improve a test performance may result in a lower score at retesting as, on most measures, improvement is expected on the grounds of maturation alone. Practice effects in the controls occur at the early reassessment (ie when they would be most expected) and, (except on the WISC-R and an isolated gain on Tokens Part 5), there is no further significant improvement in test performance from 3 months to 1 year.

Gains on IQ at 3 months (2 points on VIQ and 8 points on PIQ) are of the magnitude expected on retesting at such an interval (Sattler, 1982). Over the whole year, the control group gains on VIQ (4 points) and PIQ (11 points) are slightly larger than those found in the Chadwick-Rutter study (3 points and 7 points respectively, 1981). This is to be expected as the UCT controls were tested three times, the Chadwick-Rutter controls twice. Significant gains occur on the subtests where familiarity with the materials is likely to assist performance (eg Picture Completion, Block Design and Mazes).

The only other measures in the entire test battery on which the controls show significant gains, even at the first reassessment, are successive finger tapping, the Detroit Motor Speed and Precision, and the Renfrew Picture Naming. Scores on the two motor tests probably improved as performance was more automatic the second time around.

15.3.3 The Moderates

In the moderate group, Table 72 shows that gains tend to occur on more measures and to be significantly larger in the first than in the second recovery period (ie most of the recovery occurs in the first 3 months, as was hypothesized). Although gains on most measures are not of a significantly greater magnitude than control group gains, the measures on which the moderates show significant gains and the controls do not, are those which tap attention, information-processing, memory, and motor speed (eg Arithmetic, Digits, Coding and Comprehension on the WISC-R, the Token Test, Selective Reminding, Speed of Naming and the Purdue Pegboards). These are the functions reported to recover within the first 4 to 6 weeks after mild head injury in adults (Binder, 1986; Dikmen et al, 1986; Gentilini et al, 1985; Gronwall & Wrightson, 1981, 1974; Levin, Mattis et al, 1987; McLean et al, 1983).

In the second recovery period, the moderates have a further few significant gains which follow the pattern of recovery found in the severely and very severely head-injured groups rather than in the control group (Table 72): PIQ rather than VIQ shows a further significant gain, as does successive finger tapping and performance on the memory measures. The Information subtest (which did not improve significantly in the first few months) now shows a significant increase, possibly reflecting the improvement in memory functions.

15.3.4 The Severes

The severes show a pattern of recovery that is similar to that shown by the very severes (Table 72). There are significant gains on most measures and gains are spread over the two recovery periods. On the WISC-R, gains on PIQ exceed those on VIQ in both recovery periods. The subtests on which they have gains (Picture Completion, Coding and Mazes), suggest an improvement in attention and visuomotor speed. On the visuomotor tests, they show gains in both recovery periods. Gains on the more complex successive

finger taps are greater in the second than in the first recovery period, a pattern also observed in the very severes. In fact, the increase in speed of the severes on successive finger taps in the second recovery period, is of a significantly greater magnitude than that found in the controls (Preferred hand) and in the moderates (Nonpreferred hand).

Significant gains occur on several language measures in the first recovery period (Renfrew Naming, Tokens Parts 1 and 5) and gains approach statistical significance on several other language measures (Word Fluency, Speed of Naming, Sentence Repetition and Pictorial Identification). Further gains occur on language measures (Speed of Naming, Pictorial Identification and Tokens Part 5) in the second recovery period, as in the very severes. Speech impairment, which characterized 5 children (12.5%) at T1, characterizes only 2 (5%) at 3 months and none at 1 year. Echolalia has disappeared by 3 months in the only severe who exhibited it at intake. Perseveration, which characterized 9 severes (22.5%) at intake, occurs in only 1 severe (2.5%) at 3 months and in none at 1 year.

As in the other head-injured groups, gains on memory functions are significantly greater in the first than in the second recovery period. Gains in the first recovery interval exceed those found in the control group. Further gains occur in the second recovery period, as in the moderates. The percentage of severes with five or more confabulations drops from 32% at T1, to 18% at T2 and to 3% (the control group level) at T3. Marked repetition characterized only 6% of the group at T1 and 3% at T2, but occurs in 5 children (14%) at T3.

15.3.5 The Very Severes

The very severes show significant improvement on most measures in the first few months after head injury and recovery continues on several measures in the second part of the year (Table 72). Gains on motor and memory tests are greater in the first than in the second recovery period, and gains on these measures and on Token Test Part 5 are also of a greater magnitude than those found in the control group. These findings correspond to those in studies of adults with severe head injury: recovery tends to be greatest in the first 6 months following injury, but improvement continues over the first year, and halts before normal or control group levels are reached (Becker, 1975; Bond, 1975; Brooks & Aughton, 1979b; Groher, 1977; Levin, Benton & Grossman, 1982; Lezak, 1979; Parker & Serrats, 1976; Uzzell et al, 1987).

Given their striking deficits at T1, the improvement in the very severes is more likely to be attributable to resolving pathophysiology than to strategies learned in the first test session. As in the other head-injured groups, the pattern of their gains is also different in some respects from that found in the control group.

15.3.5.1 Intelligence

Unlike the controls and the moderates, the very severes do not have significant gains on VIQ in the first recovery period. The only verbal subtest to show significant improvement is digit span, which suggests an improvement in concentration. The very severes showed a nonsignificant loss on Arithmetic and several had clearly forgotten bonds and strategies once known by rote and still present at the initial assessment. In the second recovery period, significant improvement occurred on Arithmetic, Comprehension and Digit Span, which may reflect better control of attention and information-processing. The Information subtest, in contrast, showed a nonsignificant loss, which may reflect an inability to acquire new knowledge (or the effects of missed schooling).

Performance subtests increased significantly in both the first and the second recovery periods. Gains on PIQ exceeded gains on VIQ in the first recovery period, which resulted in a PIQ equal to VIQ (rather than a PIQ greater than VIQ as occurred in the controls and the less severely injured). The very severes, like the severes, improved both in terms of what they were able to do in the time limit, and in their understanding of tasks.

The findings in the very severe group differ from those in other studies in which recovery on VIQ occurs earlier and is more complete than recovery on PIQ (Becker, 1975; Bond, 1975; Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Mandleberg & Brooks, 1975). The differences seem to lie in a greater initial deficit in VIQ in the present study, no gains on VIQ until the second part of the year, and a VIQ which has not recovered to control group levels by 1 year. As in these other studies, gains on PIQ exceeded those on VIQ, and the deficit for the very severes on PIQ relative to the controls (26 points) is larger than the deficit on VIQ (18 points) at 1 year.

Mean gains on VIQ and PIQ from T1 to T3 (4 points and 16 points respectively) are not of a greater magnitude than those occurring in the control group, and are lower than those reported by Chadwick and co-workers for their severely head-injured group over the same time period (viz, 11 points on VIQ and 25 points on PIQ) (Chadwick, Rutter, Brown, Shaffer & Traub, 1981). The studies are not strictly comparable in that the starting levels of IQ are different and, in the present study, the head injured and controls were retested at 3 months as well as at 1 year. One would therefore expect greater practice effects in the UCT study than in the Chadwick-Rutter study. These occurred in the control group, but not in the very severes. As mentioned, the very severe group in the UCT study includes a higher proportion of cases with PTA exceeding 2 weeks than the severe group of the Chadwick-Rutter study. It may be that with more severe head injury, there is less potential for

intellectual recovery. In other words, recovery may not be proportional to the initial deficit, and very severe impairment may limit the potential for recovery. Dikmen and co-workers (1983) made this qualification when they suggested that recovery would be proportional to the initial deficit: "those surviving severe head injuries may have little potential for improvement over and beyond some very basic functional levels." (p.337).

The plot of PIQ recovery curves (Figure 8, p.189) shows considerable variation among individuals, as did similar plots published by Brooks and co-workers (1984). Whereas some of the children with the lowest PIQs (below 50) at initial assessment remain low, others have steep recovery curves (eg B.G. gained 27 points, P.Jo. 32 points). Overall, the trend was for the majority of the head injured to show gains on PIQ from T1 to T3, (only 6% show a loss, compared with 20% of the controls), and, particularly in the very severe group, gains tended to be large (11 or more points). The general pattern for PIQ is one of improvement, but not to control group levels.

15.3.5.2 Visuomotor Functions

The pattern of gains in the very severes on the visuomotor tests over the two recovery periods appears to reflect differences in the complexity of the tasks, with the simpler tasks showing earlier recovery. Gains on the Purdue pegboards (which involve more automatic movements than those required on the successive finger taps), were significant within the first recovery period (Table 72), and exceeded those of the controls who had performed almost at their maximum level at the first test occasion. Gains on the successive finger taps, which involved copying the examiner's sequenced movements, were not significant until the second part of the year. These gains then exceeded those of the controls whose performance improved only from the first to the second assessment. The Chadwick-Rutter (1981) study also reported gains on manual dexterity tasks in the first year after severe head injury which exceeded those of the controls. Both Conkey (1938) and Lezak (1979)

reported that improvement on simple tasks preceded improvement on more complex tasks in head-injured adults. Marked incoordination on the Mazes dropped from characterizing 21% of the very severes at T1 to characterizing 12% at T2 and 8% at T3. The frequency of any incoordination remained at just over 50% at T1 and T2, and dropped to 38% at T3.

Gains on the Beery and Detroit visuographic tests were significant for the very severes only in the first recovery period. Both measures have small, nonsignificant losses at T3. This means that the raw scores stayed the same. Klonoff and associates found that the gains of the head injured on various visual-spatial tasks did not exceed those of the control group over the first year (Klonoff & Low, 1974).

15.3.5.3 Language Functions

Recovery on language production, repetition and comprehension occurs in the first recovery period, and the very severes show further recovery on language production and comprehension measures in the second recovery period. On the Renfrew Picture Naming, both the accuracy with which pictures were named and the speed of naming, show improvement. Errors due to misperceptions and perseverations diminished. By 3 to 4 months children were more likely to offer different guesses for each picture or say "I don't know" than perseverate incorrect responses. Names of objects for which there had been circumlocution at T1, were produced, making it clear that word retrieval and not ignorance had been the problem. Improved word retrieval by T2 is also indicated by the significant gain in Word Fluency.

The improvement in Sentence Repetition from T1 to T2 may reflect improved attention and concentration. No further gains occurred, possibly because the more difficult sentences tapped memory functions, and these, as mentioned, showed no further improvement of significance after T2.

Comprehension as measured on the Token Test showed gains in both recovery intervals, but approximately half the very severes were unable to achieve perfect scores on Picture Identification and Parts 1 and 3 of the Token Test.

Difficulties in speech articulation tended to decrease in severity and frequency over time, although such changes could not be shown to be statistically significant. Impaired speech dropped from characterizing 32% of the very severes at intake, to 26% at 3 months, and 15% at 1 year. Three months after the first assessment, the aphasic child (J.G.) was speaking slowly, mostly in single words with each syllable stressed. By 1 year her speech was rated normal, although she was slightly slow in producing verbal responses. The paraplegic child (M.K.) had slightly soft speech, rated as normal from 3 months.

Perseveration dropped from characterizing 46% of very severes at intake to 30% and 15% at the successive test occasions. Marked perseveration (affecting several measures and which was of the continuous or stuck-in-set type, as well as recurrent) characterized only 1 very severe at 3 months, and no cases at 1 year, whereas it was noted in 4 very severes and 1 severe at the initial assessment. Echolalia had also resolved in all cases by T2.

It is evident that, in the present study, improvement occurred in language production and comprehension as well as in speech articulation over both parts of the year in the very severes. Chadwick and co-workers found verbal fluency to be recovered 4 months after severe head injury in children, and the magnitude of improvement to exceed that found in the control group (Chadwick, Rutter, Shaffer & Shrout, 1981). In adults sustaining head injury, Groher (1977) found improvement of receptive and expressive skills to occur predominantly within the first 4 months. A similar finding was made by Brooks and Aughton (1979b), who found recovery on the Token Test by 3 months and Word Fluency to be largely recovered by 6 months. Scores on these measures

were nonsignificantly lower than the control group scores at 1 year, however.

Najenson and co-workers (1978) found recovery of visual and auditory comprehension to precede recovery of oral expression, reading and writing. This recovery was said to be complete within the first 4 to 9 months, while speech articulation problems persisted. It should be noted, however, that recovery of comprehension and expression referred to simple measures (eg understanding of verbal orders and conversational speech). The patients in the Najenson study were unable to cope with standardized language tests. Groher (1977) and Sarno (1980, 1984), found deficits persisting on standardized tests once communication returned to normal. Johnston and Mellits (1980) found recovery of comprehension to parallel recovery of expressive language in children emerging from coma.

15.3.5.4 Memory

Marked gains in memory ability (of a greater magnitude than in the control group) occur in the very severes in the first recovery period. There are no further gains of significance in the second recovery period. This suggests that deficits are likely to be persistent. Marked repetition of items stays at the same level (25%) throughout, and the same proportion (17%) have 5 or more confabulations at T3 and T1 (although these are not necessarily the same children).

The present findings are similar to those reported in studies of adults with severe head injury, where it is clear that most of the improvement in memory functions occurs in the early months (Groher, 1977; Parker & Serrats, 1976). Parker and Serrats (1976) found that in the group with disorientation persisting over 1 month, there was no further increase in the percentage obtaining normal learning curves after 6 months, although some of the less severely injured continued to improve beyond this time. Very variable recovery was noted by Brooks and Aughton (1979b), Lezak (1979), and Parker and Serrats (1976).

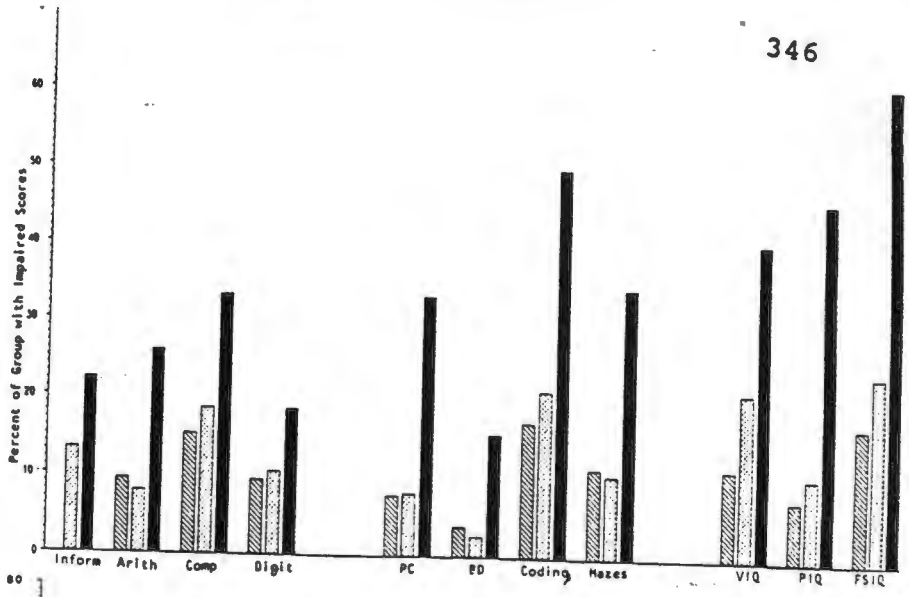
15.4 Persisting Impairment

Measures on which the head-injured children still have significantly lower mean scores than the controls at 1 year are summarized in the third column of Table 71, (p.310). The percentage of each group with persisting impairment on the different measures (defined as a score below 95% of the controls at 1 year) as was given in the separate Results chapters, is summarized graphically in Figure 29.

Figure 29. Persisting Impairment at 1 Year

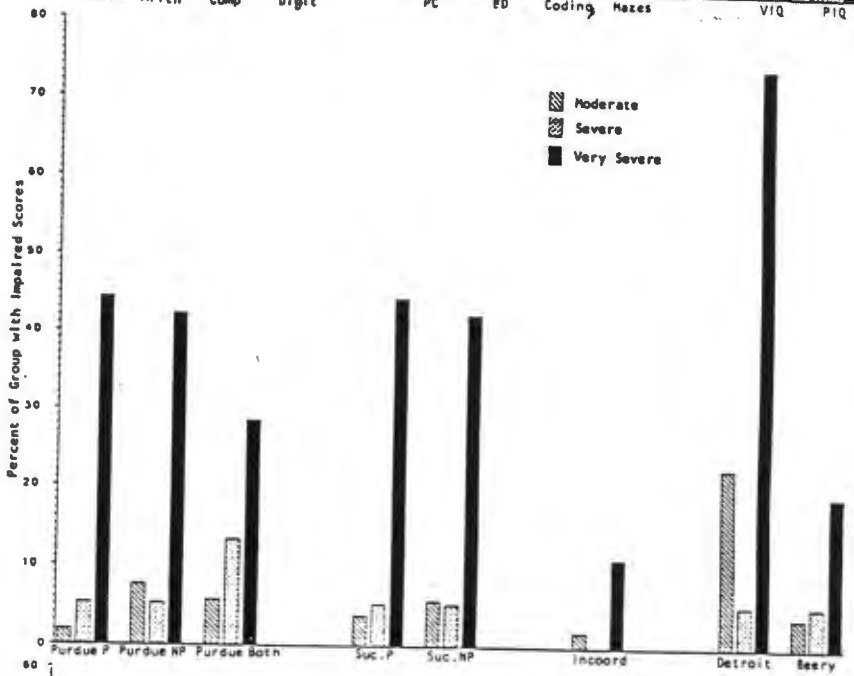
Intelligence

Inform=Information, Arith=Arithmetic
Comp=Comprehension, Digit=Digit Span
PC=Picture Completion, BD=Block Design



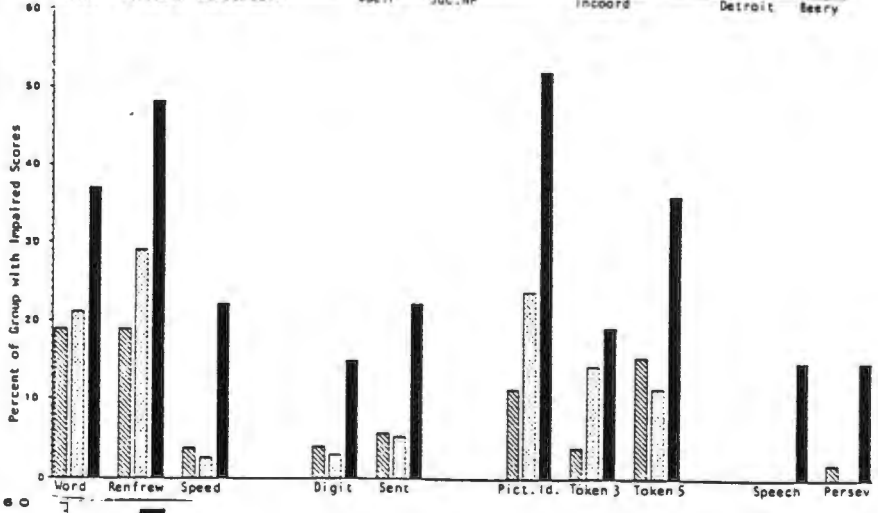
Visuomotor

P=Preferred, NP=Nonpreferred
Suc.=Successive
Incoord.=Incoordination on WISC-R Mazes



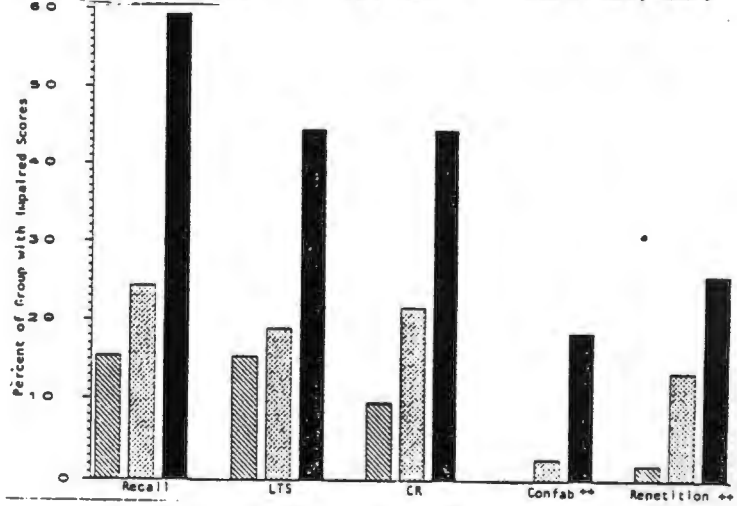
Language

Digit=Digit Repetition,
Sent.=Sentence Repetition
Pict.Id.=Pictorial Identification
Speech=Speech Impairment
Persev.=Perseveration



Memory

LTS=Long-term Storage
CR=Consistent Retrieval
Confab**=High level confabulators
Repetition**=Marked Repetition



15.4.1 Severity Issues

Most striking is the effect of severity of injury on test results at 1 year. In Table 71, it is evident that the very severes have significantly lower mean scores than the controls on almost all measures at 1 year. Figure 29 shows the very severely head-injured group to have the highest percentage of children with persistent deficits at 1 year. The measures most sensitive to impairment after head injury in this group are IQ, motor speed, language production and comprehension, and memory. Not sensitive (in terms of the percentage with persisting impairment) are the Beery, incoordination on the Mazes, Speed of Naming, language repetition and the easier parts of the Token Test.

In the severely and moderately head injured, scores are significantly lower than those of the controls only on IQ, memory, and Renfrew Naming (Table 71). The measures on which the percentage of severes impaired is significantly greater than the 5% of controls defined as impaired, are FSIQ, Coding, Recall, (CR approaches significance), Renfrew Naming, and Pictorial Identification. The percentage of moderates impaired is significantly above the control group level only on Coding subtest of the WISC-R and on the Detroit Motor Speed and Precision.

The nature and extent of the persisting impairment on the various functions is discussed briefly before turning to the classification of outcome and the factors associated with persisting impairment.

15.4.2 Intelligence

Intelligence is amongst the functions with the highest rate of persisting impairment (Figure 29). FSIQ is below 80 at 1 year post-injury in 17% of the moderates, 24% of the severes and 62% of the very severes, in comparision with 5% of the controls. None of the controls or moderates would be classified as mentally deficient (IQ below 70), but 3 severes (8%) and 6 very severes (22%) would be so classified. In the study of Winogron et al (1984), 12% of the mild and moderately head injured and 47% of the severely

head injured have a FSIQ two standard deviations or more below the test mean (presumably below 70) at approximately 1 year post-injury. The Winogron study, however, seems to include acute cases which may account for the higher percentage found to be mentally deficient. Other studies with long-term follow-up at 1 or more years, have found about 40% of their severely head injured to have IQs below 80 (Baracchini-Muratorio et al, 1985; Mahoney et al, 1983). Brink and co-workers (1970) reported 66% of severely injured children at a rehabilitation centre to have borderline or retarded intelligence.

At the other end of the spectrum, none of the very severes in the present study have above average intelligence at 1 year after head injury, in comparison with 17% of the controls and severes, and 9% of the moderates. The findings of the present study, in conjunction with the findings of the other studies mentioned above, provide support for the contention that in children, intelligence is amongst the functions most likely to be impaired by brain damage (Boll & Barth, 1981; Chadwick & Rutter, 1984).

In the very severe group, the percentage of children found to have persisting impairment on PIQ (46%) is similar to that found in other studies of children with severe head injury (eg Flach & Malmros, 1972; Levin, Eisenberg, Wigg & Kobayashi, 1982; Winogron et al, 1984), but higher than the percentage of those with PIQ below 90 at 1 year calculated from the data of the Chadwick-Rutter study (viz 32%, 1981). Coding appeared to be the subtest most sensitive to the effects of head injury and was impaired in a significant percentage of moderates (17%) and severes (21%) as well as in 50% of the very severes. The sensitivity of the Coding subtest to head injury has been reported in other studies (eg Bawden et al, 1985; Colam, 1984; Costeff et al, 1985). However, scrutiny of the names of those with persisting impairment on the Coding subtest revealed a disproportionate number with poor premorbid school achievement. Coding is known to correlate with academic achievement and it is suggested that it is this factor, rather than head injury,

that produced the increase in numbers impaired on Coding (see p.350).

In the very severe group, a third of the cases also had persisting impairment on Picture Completion and Mazes, compared with 15% on Block Designs. In the other head-injured groups, the percentages with persisting impairment on Performance subtests other than Coding were not significantly greater than the 5% defined as impaired in the control group.

Persisting impairment of VIQ occurred in 41% of the very severes, 21% of severes and 11% of moderates. In the moderate and severe groups, a slightly larger percentage had impairment on VIQ than on PIQ. Inspection of the names in these groups reveals a number of farm children amongst those with impaired VIQ. (Farm children tended to be the most disadvantaged children in the sample, with parents of no or limited education). In their study of focal head injury, Chadwick and co-workers found PIQ to correlate with length of unconsciousness and VIQ to correlate with SES (Chadwick, Rutter, Thompson & Shaffer, 1981).

In the very severe group, 33% had persisting impairment on the Comprehension subtest and 22% on Information. These findings may reflect a failure to acquire new knowledge. Colam (1984) also found Information to be significantly reduced in head-injured children compared with matched controls, several years after injury.

15.4.3 Visuomotor Functions

Persisting impairment on the tasks involving manual dexterity occurs in about 40% of the very severes and in no more of the moderates and severes than of the controls. On the visuographic measures, only 20% of the very severes are impaired on the Beery drawings, but 77% are rated as impaired on the Detroit at 1 year. Also showing persistent impairment on the Detroit are 23% of the moderately head injured. As the mean scores of the moderate group are not significantly reduced on any of the visuomotor tests at 1

year, this could be regarded as a chance finding. However, the percentage of moderates showing persistent impairment on the Detroit (23%) is very similar to the percentage showing persistent impairment on the Coding subtest of the WISC-R (21%), which also involves tracking and motor speed.

These measures are known to correlate with school achievement (Baker & Leland, 1967; Sattler, 1982). Inspection of the names of the moderates with persisting impairment on the Detroit shows 10 out of the 12 (83%) to have had poor premorbid school achievement, compared with a base rate of 40% in this group. All 6 of the moderates with persisting impairment on Coding who had poor premorbid school progress are also impaired on the Detroit. It is suggested that, in the moderate group, the Detroit is detecting a deficit that existed prior to the head injury in some of the children. This is not the case in the very severe group. Twenty very severes have persisting impairment on the Detroit and school progress is known in 17 of these cases. In only 8 of these cases (47%) was premorbid school progress poor, a rate very similar to the base rate in the entire very severe group (43%).

These figures for persisting impairment on visuomotor measures at 1 year correspond with the 40 to 47% of severely head injured and 6 to 12% of mild and moderately head injured found to have persisting impairment on visuomotor measures at 1 year by Winogron and associates (1984).

This same Canadian group (Bawden, Winogron & Knight, 1985) have claimed that unspeeded or moderately speeded tests are less sensitive to the effects of head injury than highly speeded tests. In the present study the Beery, which is the only untimed visuomotor test in the battery, detected fewer of the very severes (20%) as having persistent impairment at 1 year than were detected on the other visuomotor tests ($\pm$40%). However, the mean Beery score of the very severes at 1 year <u>is</u> nonetheless significantly lower than that of all the other groups.

Impairment on visuomotor tasks in the preschool phase has been found to correlate with later performance on reading and other scholastic tasks (eg Bishop, 1980; Boll & Barth, 1981; Wolff et al, 1985). As many of the children in the present study are still acquiring basic reading, writing and arithmetic skills, it is possible that present deficits in visual scanning and processing, understanding of spatial relationships, motor planning and visual-motor integration, may impair the acquisition of such skills. Where there was a premorbid deficit in the acquisition of these skills, it is possible that even a mild head injury may be critically limiting.

15.4.4 Language Functions

The controls rated as impaired on the language measures at 1 year tended to be those from deprived backgrounds and/or to have a history of school failure. Only 1 control, Q.L., clearly had language problems. On the Renfrew Naming and Word Fluency measures in Figure 29, from 37 to 47% of the very severes, from 21 to 29% of the severes, and 19% of the moderates show persisting impairment at 1 year. (Persisting impairment on at least one of the language production measures including Speed of Naming, occurred in 12% of controls, 36% of moderates, 47% of severes and 67% of very severes; while impairment on more than one measure occurred in 33% of the very severes, but in only 5% or less of the other groups). The Renfrew Picture Naming shows a better differentiation of the groups than Word Fluency. It was noted that the moderates impaired on Word Fluency tended to come from deprived backgrounds, whereas the severes impaired on the Renfrew included children with long PTAs (ie between 3 and 7 days).

Follow-up studies of language functions in children after head injury, are lacking. Persisting reduction in Word Fluency was reported by Colam (1984) and Winogron et al (1984), but the Chadwick-Rutter group (1981) found full recovery. Reductions in Speed of Naming were found by Colam (1984) and the Chadwick-Rutter group (1981), but differences

in the present study were nonsignificant. Gaidolfi and Vignolo (1980) found reduced verbal expression in 19% of children followed up 10 years after head injury. Word fluency and naming problems are commonly reported as persisting for years in adults with head injury (eg Sarno, 1980, 1984) and in children with left hemisphere lesions (eg Alajouanine & Lhermitte, 1965; Hecaen, 1983; Vargha-Khadem et al, 1985).

Impairment of comprehension (Pictorial Identification and Token Test in Table 71, Figure 29) persists in 36 to 52% of the very severes, 11 to 24% of the severes, and 11% to 15% of the moderates at 1 year. It was felt that these comprehension measures were multifactorial and that scores were affected by perceptual, attentional and memory deficits as well as difficulties in the processing of language. Other studies have found scores on the Token Test either to be nonsignificantly lower (Colam , 1984) or impaired in only a small percentage of children (Gaidolfi & Vignolo, 1980) tested years after head injury. Recovery of comprehension has also been reported in children after left hemisphere lesions (Alajouanine & Lhermitte, 1965; Cooper & Flowers, 1987; Hecaen, 1983). It seems likely, therefore, that factors other than comprehension account for some reduced scores in the present study.

The language measures on which the head injured are impaired correspond to those identified by Dennis (1987) as involving semantic competence. This factor involves the broad basis on which words are understood and used and Dennis found it to be impaired by left temporal damage. Deficits cannot confidently be attributed to focal lesions where there is the possibility of diffuse injury. It is of interest, however, that in the moderate and severe groups, 6 of the 11 children regarded as having possible left focal lesions had persistent impairment on some of the language measures, whereas none of the 7 children identified as having possible right focal lesions had persistent impairment on any of the language measures. No focal patterns could be identified in the very severe group, where

children with both LH and RH focal lesions had language impairment.

Language repetition, as in other studies, was not particularly sensitive to the effects of head injury, and 22% of the very severes and no more than 5% of the moderates and severes were defined as having persistent impairment. However, 10 children, (1 control, 4 moderates and 5 severes) with problems in auditory retention (detected as previously described from qualitative assessment of performance on Digit and Sentence Repetition, Selective Reminding, and Token Test Part 5), were referred to the Athlone School Clinic. Their parents reported difficulties at school with language, particularly spelling, phonics, and recitation.

It is important to try to determine whether these problems are attributable to the head injury. In 5 of the children, including the control, learning problems had preceded head injury. In 2 children (W.C. and R.H.), the learning problems emerged in the year following head injury such that both looked likely to fail Sub B, although they had done well in Sub A premorbidly and had average intelligence at T3 (FSIQs of 106 and 102). Both had PTAs of less than 2 days and no focal signs. Both had definite cognitive impairment at T1 (W.C. had echolalia and perseveration) and showed steep recovery over the year, although W.C. had persistent impairment at 1 year on several language measures.

The remaining 3 children had not gone far enough in school prior to injury for progress to be known. PTA lasted 24 to 48 hours and none had focal signs, although there was some evidence of left-sided injury: A.A. had a left-sided clinical basal fracture, M.Ko. and T.W. had left-sided focal slowing on EEG. A.A. and M.Ko. had a pattern of striking impairment at T1 (IQs of 69 and language and memory functions particularly affected) and marked gains over the year, implicating the head injury. Both had persisting impairment on language measures at 1 year. T.W., in

contrast, showed less cognitive impairment at T1 and only small gains over the year.

In conclusion, it appears that head injury could have been a contributory factor in at least 4 out of 9 children found to have auditory retention problems after mild head injury (ie PTA less than 7 days). Even where there are learning problems prior to the head injury, it is possible that there may be additional or more severe problems after head injury. This possibility is acknowledged for severe head injury by Costeff and colleagues (1985), but overlooked by Mahoney et al (1985). The analysis by Klonoff and co-workers of the neurological, EEG and neuropsychological deficts in children making poor progress at school after mild head injury, certainly suggested that the injury was contributary.

Impairment of speech persisted at 1 year in 4 very severes (14%), all boys. Three still spoke slowly and one of these had uneven voice production as well. The fourth boy had indistinct speech with swallowed words, but no slowing. Associated conditions in all 4 include hemi- or quadriparesis (as reported by Alajouanine & Lhermitte, 1965). Dysarthria as a persistent problem after severe head injury has been reported in from 12% (Filley et al, 1987) to just over 40% (Brink et al, 1970; Costeff et al, 1985) of children admitted to rehabilitation centres. Long-term dysarthria has been noted in adults after severe head injury (Sarno, 1980, 1984; Thomsen, 1984).

Perseveration at 1 year persisted in 4 of the very severes (15%). These children had been noted to have disinhibited chatter, echolalia or an inability to persist at tasks without prompting at T1. Perseveration was associated with persisting speech deficits in 2 cases and with poor memory (including marked repetition) in 3 cases, all of whom had a BAD NII outcome. In the case of R.B., who had a GOOD NII outcome, the perseveration seemed an isolated feature left over from a previously disinhibited state.

15.4.5 Memory

It is clear in Figure 29 that the percentage of children with persisting impairment of memory functions is related to the severity of injury sustained. Although the measures are highly intercorrelated (.86 to .93), it was not necessarily the same children who showed persisting impairment on Recall, LTS and CR. Ten percent of the controls in comparison with 20% of the moderates, 30% of the severes and 60% of the very severes, have persisting impairment on at least one of the three measures, but no controls, 6% of moderates and 11% of the severes, in contrast to 37% of the very severes, have persisting impairment on all three measures.

At 1 year, no moderates and 3% or less of the controls and severes are high level confabulators, in comparison with 17% of the very severes. Only isolated cases in the control and moderate head injury groups have marked repetition, in comparison with 15% of severes and 25% of very severes. Three out of the 5 severes with marked repetition have a BAD NII outcome, and a fourth is borderline, while the very severes with marked repetition have BAD outcome with high NII scores.

Memory deficits persisting after severe head injury have been reported in children and adolescents (Colam, 1984; Levin, Eisenberg, Wigg & Kobayashi, 1982) and are commonly reported in adults (eg Brooks, 1976; Brooks & Aughton, 1979b; Levin, Benton & Grossman, 1982; Lezak, 1979; Parker & Serrats, 1976).

Lewis and co-workers (1987) have found memory deficits persisting at 1 year in children after mild injury and, in the studies of adults with mild head injury, at least a percentage have had persisting memory deficits (Jansen, 1989; Levin, Amparo et al, 1987; Levin, Goldstein, High & Eisenberg, 1988; Stuss et al, 1985).

15.5 The Classification of Outcome

Two measures of cognitive outcome after head injury were investigated: the WISC-R Performance IQ (PIQ) and a Neuropsychological Impairment Index (NII) derived from a battery of tests measuring functions other than intelligence. Eight percent of the moderates, 10% of the severes and 46% of the very severes have a PIQ below 80, in comparison with 5% of the controls. In contrast, 9% of the moderates, 18% of the severes and 82% of the very severes, but only 2% of the controls, are rated as having persistent impairment on the NII. In general, defective PIQ does not show as good a dose-response relationship with PTA length as is shown by BAD NII outcome. When both are treated as continuous variables, the correlation of PIQ with PTA length ranked in days is .28, while that for NII with PIQ is -.50.

The 46% of very severes with PIQ impaired corresponds well with 40 to 50% found impaired in other studies of severe head injury (eg Baracchini-Muratorio et al, 1986; Flach & Malmros, 1972; Winogron et al, 1984). (Studies which have found a higher proportion of children with intellectual impairment, have come from the rehabilitation centres and have included a higher proportion of children who have been in prolonged coma (eg Brink et al, 1970; Costeff et al, 1985; Filley et al, 1987). However, there are two problems with accepting this figure of 40 to 50% as having cognitive impairment following severe head injury. One is that children who were of low intelligence premorbidly may be regarded incorrectly as showing cognitive impairment attributable to head injury. The other is the implication that the remaining 50 to 60% with IQs above 80 are <u>not</u> cognitively impaired as a result of head injury.

It was to avoid the first problem (falsely attributing low IQ to head injury) that the Chadwick-Rutter group (1981) imposed the criterion that there should be marked <u>recovery</u> (defined as a PIQ gain from the initial to the 1 year assessment greater than that found in 95% of controls),

before the deficit could be attributed to head injury. If this recovery criterion is applied to the present group, only 15% of the very severes and none of the other groups (ie 3% of the total head-injured group) would be regarded as having persistent intellectual impairment (ie recovery as defined, and a PIQ that was nonetheless one standard deviation or more below the control group mean at 1 year). A further 6% of moderates, 5% of severes and 4% of the very severes (ie 5% of the total head injury sample) would be regarded as having had transient intellectual impairment (ie recovery as defined, and a PIQ that was not one standard deviation or more below the control group mean at 1 year). These figures do not reflect the transient impairment that was found in the scores of the head injured relative to the controls at the initial assessment, nor do they reflect the percentage of very severes found to have persisting impairment relative to the controls on intelligence and neuropsychological measures at 1 year post-injury. Imposing such a steep recovery criterion before attributing the deficits found to the effects of head injury, appears unwarranted. When the head injured and controls have been matched for SES, age and sex, have had a similar experience of trauma and hospitalization, and a statistical check has been kept on possible confounding variables, as in the present study, it seems likely that deficits detected are attributable to head injury, regardless of the steepness of recovery curves.

It is accepted that evidence of recovery from a deficit present after a head injury is a strong indication that the deficit was a consequence of brain damage, but it is disputed that the absence of any recovery phase necesarily provides strong circumstantial evidence that the initial deficit was not due to head injury, as is claimed by Rutter and co-workers (1984, pp.87 to 88). The present study found very variable individual patterns of gains and losses after head injury, a finding consistent with other studies of head injury (eg Brooks & Aughton, 1979b; Brooks et al, 1984). After very severe head injury resulting in prolonged coma,

children may recover very little of their premorbid ability (eg Brink et al, 1970; Pelco, 1985).

Cognitive impairment which is not detected on the IQ tests has been found in studies of head-injured children which have included tests of other functions (eg Klonoff et al, 1977; Winogron et al, 1984), and suggested in the studies which have reported school learning difficulties out of proportion either to the severity of head injury sustained (Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Chadwick, Rutter, Thompson & Shaffer, 1981; Gronwall, unpublished), or to the level of IQ measured (Costeff et al, 1985; Mahoney et al, 1983). Reports on poor school functioning despite average intelligence in children with brain disorders other than head injury, have also suggested cognitive impairment which is not detected by intelligence tests (Addy, 1987; Duffner et al, 1988; Ellenberg et al, 1987; Prigatano et al, 1983).

An index of impairment at 1 year post-injury, based on the neuropsychological measures in the battery rather than on IQ, was developed as it was felt that PIQ was determined to a large extent by premorbid factors, and that several children with IQs above 80 did, in fact, have cognitive impairment. The Neuropsychological Impairment Index (NII) was based on the number of measures other than IQ on which the child had scores worse than those of 95% of the controls. In this respect it is similar to indices derived from their neuropsychological measures by Klonoff and co-workers (1977) and by Winogron and associates (1984). The battery covered visual-motor, language and memory functions and there were 22 variables on which a child could score 1 point (see p.286, Outcome results). Cut-points on the test scores were the control group 5th percentile or two standard deviations from the control group mean at 1 year. Also included were features that, in terms of their prevalence at intake or persistence at 1 year, were associated predominantly with severe head injury, namely perseveration, impaired speech, 5 or more confabulations and, less pathogonomic, marked repetition and marked incoordination.

Classification of a case as a BAD NII outcome was based on an NII score exceeding that obtained by 97.5% of the controls. Only 1 control, Q.L., was rated as having neuropsychological impairment, (NII ≥5 points), and this was confirmed independently by the Speech Therapy Department. Theoretically a child could obtain an NII score of 5 or more by scoring points in only one area, (visuomotor, language, or memory), but in practice this happened only in 1 of the 34 head injured classified as having BAD NII outcome (M.V., discussed later), whereas 12 (35%) had impairment in two areas and 21 (62%) had impairment in all three areas.

The use of cut-points on the tests to determine impairment could mean that children with borderline scores on several measures were missed. Re-analysis of the tests using the control group 10th percentile as the cut-point on each, suggested that this was the case for 2 children in the severe group, who each had 4 NII points using the 5th percentile, but had little effect on the scores of any of the very severes classified as GOOD. The 5th percentile was retained as the cut-point as it constituted the stricter criterion.

More head-injured children (n=34) are rated as cognitively impaired on the NII than have PIQ below 80 at T3 (n=20). There is agreement between the two outcome measures on 80% of the head-injured cases classified as BAD and on 82.5% of those classified as GOOD. However, this leaves 21 out of 117 cases (18%) on which there is disagreement as to whether the child is cognitively impaired or not. A detailed review by group of all cases for which the two outcome measures disagreed, suggested that children with definite brain injury, recovery of function over time, but persisting deficits in several areas (often including borderline PIQ), tend to be rated impaired on the NII, whereas children from deprived backgrounds with premorbid dullness, less evidence of diffuse damage, fewer complications, and either recovery by T2 or no pattern of recovery, tend to have PIQs below 80. The trends in each

group for those rated impaired on one measure but not the other are discussed below.

Agreement on the two outcome measures is lowest in the very severe group (58% of cases). Ten of the children with GOOD PIQ are classified as BAD outcome on the NII. NII scores range from 5 to 11. Seven of the children have scores of 5 or 6, which could suggest that their impairment is marginal, except that several have borderline scores (below the control group 10th percentile) on other tests contributing to the NII. Nine of the children have definite impairment in more than one neuropsychological area (language, visuomotor or memory), some in all three areas. The exception, M.V., has visuomotor and visuoperceptual impairment that could be related to focal (right parietal contusion) rather than diffuse injury. All 10 show a pattern of recovery on the neuropsychological tests from T1 to T3, particularly on tests of motor and memory functions. In spite of IQs above 80, all 10 also have evidence of intellectual impairment from head injury. They either have subtest scatter at T1 which suggests higher premorbid ability (n=8), and/or they have marked gains on PIQ from T1 to T3 (n=9).

There is evidence of brain dysfunction in all 10 of them. Nine have PTA lasting from 14 to 37 days (M.V. has 8 days of PTA). In only 2 of the 9 scanned was nothing abnormal detected: 3 had focal contusions underlying skull fractures, and 2 of these and 4 others had diffuse brain swelling. Nine of the 10, including M.V., had complications ranging from breathing difficulties to hypertension, repeated seizures, aspiration pneumonia and meningitis.

In contrast to this pattern of clear pathology, neuropsychological deficits, and recovery over time, the child C.P, with a PIQ below 80 but not impaired on the NII (2 points only), has a vague history, no specific deficits but a generalized dullness, and no pattern of recovery. She is the only child for whom length of unconsciousness is unknown (see p.145), but which could not have exceeded a few

hours, and head injury was suggested by a 6th nerve palsy and confusion. PTA was estimated at 9 days, but her confusion could have been attributable to a persistent temperature from wound infection, allergy to Penicillin, and/or a basic dull mental state, rather than amnesia. Both PIQ and VIQ remained at 74 throughout follow-up and the other tests showed little in the way of gains. The child of parents of limited education, she was repeating Sub A in a farm school when the accident occurred.

Among the 4 severes with BAD NII but GOOD PIQ outcome, scores on the NII range from 5 to 8. PIQ is borderline in T.W. (80), but average in the other 3 (92, 92 and 100). All 4 come from backgrounds with parents of limited education and, in the 2 for whom school progress is known (A.G. and A.D.), there is a history of repeated school failure. However, there is a clear evidence of brain injury in them all: PTAs of 4 to 5 days; complications such as rhinorrhoea, subarachnoid haemorrhage and temporal lobe abscess; and abnormal EEGs. Three were pedestrians, while A.D. is an assault with a left focal injury and specific deficits on language tests. All, except T.W., show a pattern of recovery over follow-up with gains on PIQ of 19, 20 and 29 points and marked improvement on the other tests. All are impaired in more than one area of the NII (language, visuomotor and memory) and 3 in all three areas.

The severe child (J.G.) with a PIQ below 80 at T3 but GOOD NII outcome, had a focal injury from an assault which shattered her skull, contused the underlying brain and rendered her aphasic with a right sided weakness. She was unconscious from 1 to 5 hours and had a PTA of about 4 days. Initially impaired on such tests as she was able to do at T1, she showed considerable recovery at T2, and by T3 had no clinically evident dysphasia, excellent memory functions, slight slowness in her right hand (accounting for her 2 NII points), while language tests were borderline but not defective. PIQ gained 10 points from T1 to T2. It is suggested that a PIQ of 78 and VIQ of 74 at T3 reflect only slight cognitive loss in an otherwise well recovered girl,

who, as the neglected child of parents of limited education, probably had low premorbid intelligence.

In the 3 moderates with impairment on NII but not on PIQ, there is evidence of deprivation and of premorbid dullness, but also of brain injury and complications (mild cerebellar signs, hypovolaemia from additional injury, seizures, cerebrospinal fluid leak, and abnormal EEGs). Two show marked recovery on the tests, particularly between T1 and T2, yet all 3 are impaired in each of the neuropsychological areas at T3 and are borderline on other neuropsychological tests (and on PIQ).

The 2 moderates with impairment on PIQ but not on NII, were both falls. One (J.N.) shows steady improvement on the tests over the year and is essentially recovered at T3 except for low PIQ (72) and 2 points on the NII for slight motor incoordination. The other child (P.H.) developed focal signs (seizures and hemiparesis on opposite sides) which resolved rapidly. CT showed a left occipital haemorrhagic contusion. There was dramatic recovery from T1 to T2, particularly on motor and memory tests. There is persisting impairment on none of the neuropsychological tests at T3, but the borderline Beery and PIQ of 54 versus a VIQ of 94 must be reflecting visual-spatial difficulties, although she was coping well at school.

Overall, this review suggests that BAD NII outcome is more closely associated with diffuse brain injury than is low PIQ, and is a more accurate indicator at 1 year of cognitive impairment which is attributable to head injury. These trends for NII outcome to be more influenced by the severity of injury and for PIQ to be more influenced by premorbid status and psychosocial factors, also emerged from the multiple regression analysis of the medical and psychosocial variables contributing to the prediction of outcome. Both NII outcome and PIQ are influenced by PTA length, but medical factors associated with poor outcome in other studies, eg complications, surgery and neurosurgery, did not contribute significantly to the prediction of PIQ,

but were retained in the best-predicting model for NII. Clinical evidence of a basal skull fracture, increased the probability of a BAD NII outcome, but contributed to an increase in PIQ. Both outcome measures are influenced by psychosocial adversity, but whereas PIQ is influenced by pre-school attendance and by English language usage (which is to some extent an indicator of upward social striving within the family, Scheffer, 1983), neither variable contributes significantly to the prediction of NII outcome.

15.6 The Influence of Premorbid School Progress on Outcome

Premorbid school progress, where known, also contributed significantly to the prediction of both outcome measures, as could be expected. The head-injured groups contained a higher proportion of children with poor school progress than the control group (42 versus 28%), but no adjustment was made to scores for this factor as (a) parents' judgement of school progress was not necessarily valid, and (b) reasons other than lack of ability could account for poor progress at school. Although school progress may not correlate absolutely with premorbid intelligence, it is likely that children who do well at school will be those with higher IQ and with the coping strategies necessary to do well on neuropsychological tests. Within the different severity groups, the effects of premorbid school progress on the two outcome measures vary. One would expect the effects of premorbid factors to be least where the effects of head injury are greatest, ie in the very severe group. Here again, the NII shows itself to be superior to PIQ in detecting the effects of head injury. For PIQ, premorbid school progress has no significant effect on the proportion of moderates and severes scoring below 80, but differences in the very severe group, where 27% with adequate school progress as opposed to 70% with poor school progress score below 80, approach statistical significance. For PIQ, there is no significant difference in the moderate and severe groups in the proportion scoring below 80 who had adequate as opposed to poor premorbid school progress. The difference comes nearest to significance in the very severe

group, where 27% with adequate school progress as opposed to 70% with poor school progress, have PIQs below 80 at 1 year.

In contrast, premorbid school progress has no effect on NII outcome in the very severes, where almost all have BAD outcome regardless of premorbid achievements, but has significant effects in the moderate and severe head injury groups. In the cases where school progress was known, all moderates and severes with BAD NII outcome had poor premorbid school progress (ie no moderates or severes with adequate school progress have a BAD NII outcome, whereas 16% of moderates and 27% of severes with poor premorbid school progress have a BAD NII outcome). School progress was not known in 2 moderates and 3 severes with BAD NII outcome. Inspection of the names shows all 5 to come from multiproblem families with parents of limited education; 4 have PIQs between 74 and 81 at T3; 1 of these (T.W.) and the child with PIQ of 100 (A.A.) were referred to the School Clinic for remediation of learning problems upon completion of the study. The expectation, admittedly speculative, is that they would not have done well at school prior to head injury. It seems then that children with resources sufficient to make adequate progress at school, are able to cope with the cognitive disruption produced by mild head injury (moderate and severe PTA), but that children who are already struggling at school, have an increased risk of not being able to cope cognitively if their resources are further reduced by mild head injury.

Other studies have identified about a third or more of head-injured children as having had poor premorbid school progress (Costeff et al, 1985; Dressler, 1984; Hjern & Nylander, 1964; Klonoff & Paris, 1974). Some investigators have regarded a lack of cognitive recovery or learning disabilities present after head injury as attributable entirely to the premorbid state and not the head injury (eg Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Mahoney et al, 1983). Where there is very severe head injury, the level of premorbid school progress may make little difference to outcome and almost all children may have new

cognitive difficulties (Costeff et al, 1985; Pelco, 1985). Where there is mild head injury, premorbid learning ability may interact with the effects of head injury to result in a definite cognitive deficit. New learning deficits in auditory processing and/or retention were suggested in at least some of the children in the present study after mild head injury (moderate or severe PTA). These findings are consistent with those of other studies which have reported an increased prevalence of school learning problems after mild head injury (Chadwick, Rutter, Brown, Shaffer & Traub, 1981; Gronwall, unpublished; Haarbauer-Krupa et al, 1985; Shaffer et al, 1980).

15.7 The Prediction of Outcome

Most studies have considered functional recovery as measured on the Glasgow Outcome Scale rather than cognitive outcome when looking at the predictive power of medical and psychosocial variables. Variables which predict physical independence are not necessarily the same as those which predict cognitive recovery (Brooks, 1987). As NII was regarded as a better indicator than PIQ of the cognitive effects of head injury at 1 year, the association between possible predictor variables and NII was analysed on a univariate as well as on a multivariate basis.Although some variables may be associated with outcome on a univariate basis, either by correlation or by significant differences between categorized groups, they may be eliminated in multivariate analysis by variables which better predict outcome in concert with each other. Univariate as well as multivariate associations are discussed below as most other studies have considered only univariate associations. Variables predicting PIQ at 1 year were determined on a multivariate basis only and are also discussed below as PIQ is such a widely used outcome measure.

15.7.1 PTA

On a univariate basis, PTA is significantly longer in those with BAD NII outcome (median 16 days) than in those with a GOOD NII outcome (median 1 day). Length of PTA ranked

in days is significantly correlated with the memory measures (-0.44 at T1 and -0.32 at T3), with PIQ (-0.28), and with NII (0.50). On the NII, only 17% of children with PTA less than 1 week have a BAD outcome, whereas the figures (admittedly based on small numbers) climb dramatically thereafter, 67% of those with PTA 1 to 2 weeks, 75% of those with PTA 2 to 3 weeks, and 92% of those with PTA over 3 weeks. On a multivariate basis, PTA contributes significantly both to prediction of a BAD NII outcome and to PIQ. In the prediction of both outcome measures, PTA is retained when GCS and length of coma are eliminated.

In the child head injury literature, these findings correspond to those of the Chadwick-Rutter group (1981) who found intellectual deficits increased in those with longer PTA, and to those of Winogron and associates (1984) who found duration of altered consciousness correlated with several measures in the neuropsychological battery used. In terms of adult head injury, Brooks (1974, 1976), Brooks and Aughton (1979a), Brooks et al (1980), Gronwall and Wrightson (1981), and Parker and Serrats (1976) found a significant association between PTA and learning and memory. Brooks and co-workers in the studies cited found PTA to be a better predictor of cognitive functioning than length of coma. A recent report by Shores (1989) using linear regression also found PTA to be a better predictor of cognitive outcome than GCS score or duration of coma.

Brooks (1984) pointed out that there could be considerable individual variation in the amount of cognitive impairment occurring in the different PTA severity categories. In the present study, in the very severe category, individuals with up to 25 days PTA escaped with little persisting neuropsychological impairment (GOOD NII outcome), while one of the highest NII scores (14) was attained by a child with 8 days PTA (P.J.). As we have seen, children in the moderate category with relatively brief PTA could have BAD NII outcome, presumably, because of the association of other factors (eg complications, poor premorbid school progress and so forth). PTA effects may be

more apparent early than late (Mandleberg, 1976) and PTA had a lower correlation with memory scores at 1 year than at initial assessment.

15.7.2 GCS

On a univariate basis, median GCS scores were significantly lower in those with a BAD NII outcome (7.5) than in those with a GOOD NII outcome (13). However, when all medical variables are entered, GCS does not contribute significantly to the prediction of a BAD NII outcome or to PIQ. GCS was possibly estimated too early in some cases, before there was stabilization of noncranial factors (see sample description, p.145).

Other studies which have found GCS score to contribute significantly to intellectual or neuropsychological outcome, have used univariate analysis with groups divided into GCS above and below 8 (Levin, Eisenberg, Wigg & Kobayashi, 1982; Winogron et al, 1984) or have used multivariate analysis (eg Williams et al, 1984, with adults), but have not included PTA as a possible predictor. Winogron and co-workers (1984) found a correlation of .44 between GCS scores and WISC-R PIQ, whereas the present study found a Spearman rank correlation of 0.25 (p=.007), possibly because all PIQs were those at 1 year, whereas Winogron and co-workers included children tested earlier. Correlation of GCS scores with NII scores is higher (Spearman rank correlation =-.45, p=.0001).

15.7.3 Coma Duration

Coma duration is significant on a univariate basis as those with BAD NII outcome have a longer coma duration (median 0.63 days) than those with a GOOD NII outcome (median 0.0 days). Spearman rank correlations of coma in days with both PIQ and NII (which are not given in the results) are also significant (-0.35 for PIQ, 0.48 for NII at T3). Nonetheless, coma length does not contribute significantly in multivariate analysis to the prediction of either NII BAD outcome or PIQ.

Associations between intelligence and coma have been found in studies in which there have been children with prolonged coma (eg Brink et al, 1970; Johnston & Mellits, 1980; Pelco, 1985; Woo-Sam et al, 1970). The longest coma duration in the present study was 16 days (J.E.). Analysis of subjects grouped into broad coma categories, (eg none, under 24 hours, and over 24 hours), has also found significant differences between the groups in terms of early deficits in neuropsychological functions and persistent intellectual deficit (Levin & Eisenberg, 1979b). In the present study, all except 1 of the children with coma lasting 6 hours or more are in the very severe group, and it is this group that has both early and persistent deficits in neuropsychological and intellectual functions. However, as has been found in adult head injury studies, coma length is not as good a predictor of intelligence, memory or language test performance at 1 year as is PTA (Brooks et al, 1980; Parker & Serrats, 1976).

15.7.4 Mechanisms of Injury

Most of the very severes (89%) sustained traffic related accidents, whereas other causes of injury were more frequent in the less severely injured, particularly in the moderates (see p.143). On a univariate basis, there is a trend for a greater proportion of those having traffic-related accidents to have a BAD NII outcome (33%) than is the case for those with other causes of injury (19%), but the difference is not statistically significant and mechanism of injury, dichotomized as MVA/not MVA, also does not contribute significantly in the multivariate analyses to the prediction of either NII outcome or PIQ. It appeared that pedestrians who were thrown into the air after being struck by a car had an increased risk of a BAD NII outcome (eg 10 out of 11 very severes and 4 out of 12 severes), presumably as this increased the likelihood of acceleration-deceleration and related DAI.

In other studies, mechanism of injury has also tended not to contribute significantly to the prediction of outcome

and has been eliminated by other variables (Jennett & Teasdale, 1981).

15.7.5 Diffuse versus Focal Injury

Analysis of NII outcome in those of the very severe group who had been scanned suggested that whereas all the children with either diffuse brain swelling alone or focal mass lesions alone had a BAD NII outcome, only 6 out of the 8 with both conditions had a BAD NII outcome. The 2 with both conditions who had a GOOD NII outcome (R.B. and S.S.) both had contusions with perifocal oedema underlying skull fractures. S.S. had a GCS of 8 lasting just on 6 hours, whereas R.B. had secondary deterioration with brain swelling and was unconsciousness less than 24 hours. The suggestion is that they had less primary injury than some of the other very severes.

In the moderate and severe head-injury groups, 18 children were noted to have focal lesions on CT scan or at surgery. None of those with haematomas and contusions detected on CT scan had a BAD NII outcome, but 1 severe with a depressed fracture and dural laceration and 1 severe with a left temporal abscess had a BAD NII outcome. Levin and Eisenberg (1979b) also found no association between the presence of an abnormality on CT scan and neuropsychological deficits.

As not all children in the study were scanned, (and as it seemed that resolution of the scanner available was such that both diffuse and focal pathology were not always detected), this variable could not be included in further analyses. It has been suggested that there may be different patterns of cognitive impairment and recovery following focal lesions, DAI or diffuse brain swelling, but this work has been based on very small numbers and awaits confirmation (Levin, Benton & Grossman, 1982); Uzzell et al, 1987). Uzzell, Obrist, Dolinskas and Langfitt (1986) and associates (1986), in a study of young adults, found increased blood flow (which is associated with diffuse brain swelling) to result in greater impairment of intelligence, particularly

of PIQ, than reduced blood flow. Filley et al (1987) found GOS category, vocational and social outcome to be worse in those with diffuse plus focal lesions, than in those with diffuse injury alone.

15.7.6 Skull Fractures

A child was entered as having a skull fracture if there was evidence of such a fracture on x-ray (linear, depressed, compound, basal or other). Children with clinical evidence of basal skull fractures were not entered unless x-ray showed a fracture. On a univariate basis, an almost equal proportion of children with and without skull fractures on x-ray had a BAD NII outcome (26% and 32% respectively). Only 1 of the 9 moderates and severes, but all 4 of the very severes, with compound depressed skull fractures had a BAD NII outcome. In the multivariate analysis, the presence of a skull fracture on x-ray decreased the probability of a BAD NII outcome.

Other studies have not found skull fractures to contribute significantly to the prediction of functional outcome (Kraus et al, 1987; Leonidas et al, 1982; Wagstyl et al, 1982). Williams and associates (1984) found the presence of a skull fracture to explain only 1% of the variance on a neuropsychological impairment index in adults.

15.7.7 Basal Clinical Fracture

Clinical evidence of a basal skull fracture occurred in 21% of the total head-injured sample. On a univariate basis, an equal proportion of children with and without basal clinical fractures had a BAD NII outcome (28% to 30%). Nonetheless, on a multivariate basis, clinical evidence of a basal skull fracture increased the probability of a BAD NII outcome, and was retained in the best-predicting model based on all the medical variables, and in the model based on both medical and psychosocial variables. In contrast, the presence of a basal skull fracture contributed significantly to increasing PIQ in multivariate prediction. Basal skull fractures are evidence of severe impact. Their contribution to neuropsychological outcome has not been investigated in

other studies. In the light of the contradictory contribution of such a fracture to the two outcome measures, further investigation is needed.

15.7.8 Seizures and Abnormal EEGs

On a univariate basis, a significantly higher proportion of children who have early seizures than of those who do not, have a BAD NII outcome (43 versus 24%). A quarter of the total sample had early seizures (mostly within the first 24 hours), but this variable does not contribute significantly to the probability of a BAD NII outcome or to prediction of PIQ when other clinical variables are included in the prediction equations.

The univariate finding contradicts that of Winogron et al (1984) who found no statistically significant association between the presence of seizures and the percentage of deficits on neuropsychological tests. The study by Winogron and colleagues was retrospective (which may mean that seizures, particularly focal seizures, were not as carefully observed and recorded as in the present study) and no details on the prevalence of seizures were given.

The presence of an abnormal EEG as opposed to a normal EEG did not differentiate significantly between the percentage of cases with a BAD NII outcome (31% to 21%). Abnormal EEGs occurred in a higher percentage of severes and very severes (76%) than of moderates (49%). EEG status also did not contribute significantly to the multivariate prediction of NII or PIQ outcome. Other studies have also found the proportion of abnormal EEGs to increase with severity of injury (Enomoto et al, 1986; Ivan et al, 1983), but the relationship between EEG abnormalities and neuropsychological deficits has tended to fall short of statistical significance (Winogron et al, 1984).

15.7.9 Neurosurgery

On a univariate basis, there was no difference between those who underwent neurosurgery versus those who had no surgery of any kind in terms of the proportion having a BAD

NII outcome (33% versus 22%). Nonetheless, in the multivariate analysis, having undergone neurosurgery contributed to the prediction of a BAD NII outcome. Craniectomies accounted for most of the neurosurgery. Only 5 children (4 moderates and 1 severe) had haematomas evacuated and none of these children were among those with a BAD NII outcome. None of the moderates, but 1 of the 2 severes and all 4 of the very severes who underwent neurosurgery for compound depressed skull fractures have a BAD NII outcome. It is not clear why undergoing neurosurgery should contribute to the prediction of a BAD NII outcome. It does not appear to be the nature of the lesion as most very severes have a BAD outcome regardless of the nature of the lesion, and only 1 of the 9 moderates and severes with compound depressed skull fractures has a BAD NII outcome. It is possible that undergoing an anaesthetic is a factor, as this would also explain why surgery other than neurosurgery also contributes to the probability of a BAD NII outcome.

15.7.10 Other Surgery

On a univariate basis, a significantly greater proportion of children undergoing surgery other than neurosurgery have a BAD NII outcome (61% versus 22%). On a multivariate basis, having other-surgery also contributed significantly to the best-predicting medical model, and to the combined medical-psychosocial model before school progress was added. Only 18 children amongst the 118 present at T3 underwent other-surgery, with a greater proportion of very severes (25%) than of the other groups undergoing such surgery (see Table 9, p.154). To some extent this variable reflects additional injuries of some severity, and it may be for this reason that it is associated with an increased risk of poor outcome (Mayer et al, 1984). However, it may be because such surgery is associated with the use of drugs which can affect cerebral blood flow and contribute to secondary insult (Jennett & Teasdale, 1981).

15.7.11 Additional Injury

A significantly greater proportion of children with multiple injuries than with head injury only, have a BAD NII outcome on univariate analysis (50% versus 19.5%). The proportion of children sustaining multiple injuries doubles in each severity group (15% of moderates, 30% of severes, 61% of very severes). Most of the additional injuries were limb fractures. In multivariate analysis, the presence of an additional injury does not contribute significantly to the prediction of either BAD NII outcome or to PIQ. Some studies have found multiple injury associated with poor functional recovery and particularly with increased mortality (Mayer et al, 1981; Walker et al, 1984; Zuccarello et al, 1985); whereas other studies have not found a significant association with GOS rating (eg Berger et al, 1985; Wagstyl et al, 1987). In the present study, it may be that the contribution of the more severe additional injuries to the prediction equation is subsumed under other variables such as complications, other surgery, and severity of injury (PTA), all of which do contribute significantly to the probability of a BAD NII outcome.

15.7.12 Complications

A greater proportion of children with complications than without complications have a BAD NII outcome (53% versus 20%). Respiratory and hypotensive complications were more common in the very severe group. Complications in the less severely injured groups were more likely to involve wound sepsis, unexplained pyrexia, or persistent cerebrospinal fluid leaks. In multivariate analysis, the presence of complications contributes significantly to the best-predicting models for BAD NII outcome. What this probably reflects is the likelihood that complications, particularly those which compromise the blood or oxygen supply to the brain, contribute to secondary brain injury (Teasdale & Mendelow, 1984).

15.7.13 Age

Although there was a trend for children with a BAD NII outcome to have a younger median age (8.0 years) than those with a GOOD NII outcome (9.6 years) this difference was not statistically significant, and age also did not contribute significantly in the multivariate analyses to the prediction of NII or PIQ outcome.

When age has been associated with functional outcome in children, it has been children under 5 who have had the poorer outcomes (Alberico et al, ,1987; Esparza et al, 1985; Hendrick et al, 1964; Humphreys, 1983; Luerssen et al, 1988; Mahoney et al, 1983). Studies which have found a trend towards a relationship between age and cognitive outcome have been those which have dichotomized their groups at 6, 8, or 10 years (eg Baracchini-Muratorio et al, 1985; Brink et al, 1970; Filley et al, 1987) and it may be the presence of children under 5 which accounts for the poorer outcome in the younger group. Chadwick, Rutter, Brown, Shaffer and Traub (1981), with a range of ages similar to those in the present study, found no significant effects for age (dichotomized at 10 years) on IQ, and the study by Klonoff et al (1977), found age not to be retained in the multivariate prediction of residual neuropsychological impairment.

15.7.14 Sex

The two outcome measures are affected in opposite ways by the sex of a child. On a univariate basis, the percentage of boys and girls with a BAD NII outcome is very similar (30% versus 26%). Within the various severity groups, there is a nonsignificant trend for more boys than girls to have a BAD NII outcome (13.5% to 0% in the moderate group, 21% to 14% in the severe group, and 87% to 75% in the very severe group). In terms of PIQ outcome, more girls than boys have PIQs below 80 (27% versus 12%), a difference which approaches significance. The median PIQ of the girls at T3 is 85, whereas the median for boys is 96. Within the severity groups, the median PIQ of the girls is consistently

slightly lower than that of the boys, and a slightly greater percentage of girls than of boys have PIQs below 80. On the multivariate analyses, male sex significantly increases the probability of a BAD NII outcome, whereas in the prediction of PIQ, female sex has a higher negative coefficient than male sex (ie reduces PIQ more).

It is difficult to make sense of these findings and they may represent statistical artefact. In general terms, when it comes to neuropsychiatric disorders (eg infantile autism, cerebral palsy, institutionalized retardation, epilepsy, delinquency, and learning disabilities) more boys are affected than girls (Taylor & Ounsted, 1972; Szatmari, 1985). Boys are generally over-represented in head-injured populations, particularly amongst the mildly head injured (Rutter et al, 1980). In the present study, 73% of the moderates, 65% of severes and 54% of the very severes are boys. On the grounds that scholastic backwardness increases the risk of head injury (Rutter et al, 1980; Dressler, 1984; Haas et al, 1987) and because boys are over-represented both amongst the scholastically backward (Nichols & Chen, 1981; Rutter, 1984) and amongst the head injured, one might expect to find more boys than girls with impairment on the neuropsychological battery. For the neuropsychiatric disorders, Taylor and Ounsted (1972) put forward the theory that, whereas fewer girls are affected, when they are affected, the consequences seem to be more severe than in boys, which may explain the lower PIQs.

15.7.15 Socioeconomic Status (SES)

For prediction purposes, SES was dichotomized as social classes 1, 2 and 3 versus 4, 5 and 6, ie professional, clerical and semi-skilled (44%), against unskilled, pensioners and the unemployed (56%). There was a nonsignificant trend for a higher proportion of those of lower as opposed to higher SES to have a BAD NII outcome (35% to 21%), but SES did not contribute significantly to the prediction of either NII outcome or PIQ in the multivariate analyses.

SES, along with age, sex and language, was entered into the preliminary multiple regression analysis of control group scores at T1 and not found to have a significant effect on any of the neuropsychological variables except Renfrew Naming, where its effects were partialled out by analysis of covariance. Although studies have found children from the lower SES groups to be over-represented in head-injured populations (eg Klonoff, 1971) they have not usually found a significant effect for SES on cognitive outcome (eg Woo-Sam et al, 1970). Chadwick Rutter, Thompson and Shaffer (1981) found VIQ, but not PIQ, to be affected by SES.

15.7.16 Language

None of the English-speaking children in the present study had a BAD NII outcome. To some extent this finding reflects the uneven distribution of language usage amongst the different severity groups: all of the very severes were Afrikaans speaking, compared with 78% of the moderates and severes. More Afrikaans is spoken in the rural areas than in the city (Scheffer, 1983) and rural children who suffered severe head injuries tend to be transferred to the city hospitals where the sample was collected.

In the multivariate analyses, language usage did not contribute significantly to the probability of a BAD NII outcome, but English language predicted a higher PIQ. In the prediction of PIQ, language usage remained in the regression equation even when school progress was added. To some extent this finding may also contain a socioeconomic factor in that English language usage may be evidence of upward social striving (McCormick, 1986; Scheffer, 1983).

15.7.17 Psychosocial Adversity

Psychosocial adversity was a composite measure with 1 point being scored for each of the following: the child was one of four or more children, lived in overcrowded conditions, lived with a single parent, the breadwinner was unskilled or unemployed, maternal education was less than

standard 6, nutrition was inadequate, either parent had physical illness, mother or father had psychiatric illness (including alcoholism), or if there was disharmony in the home. There was a trend for the median adversity scores to be higher for those with a BAD NII outcome (5) than for those with a GOOD NII outcome (4), and, when all medical and social variables were entered into the regression analyses, psychosocial adversity contributed significantly to the probability of a BAD NII outcome and to a decrease in PIQ. When the psychosocial variables were analysed alone, only psychosocial adversity and a low Rutter Parent scale score contributed significantly to the prediction of a BAD NII outcome. When premorbid school progress was added to the prediction equations, psychosocial adversity was eliminated. This suggests that the effects of psychosocial adversity on recovery from head injury are likely to be similar to their effects on progress at school premorbidly. This is understandable as stressed parents of limited resources may have as little to contribute to their child's care and recovery after head injury as they were able to contribute to his or her school career previously. Adversity is a very complex variable and its contribution is difficult to quantify. There were many examples of poverty and caring parents coexisting, and, even where there were drunken, abusive or neglectful parents, the child could still be receiving loving care from a grandparent, an older sibling or someone sharing the home.

In the Chadwick-Rutter study it was found that the development of new psychiatric symptoms after head injury was a function not only of the severity of injury, but also of the level of psychosocial adversity (Brown et al, 1981). However, this group did not investigate the effects of psychosocial adversity on cognitive recovery. The present study suggests that psychosocial factors play a role in influencing cognitive as well as behavioural recovery.

15.7.18 Pre-school Attendance

The majority of the sample (70%) had not attended pre-school even though children from poor and/or multiproblem families are given preference for places in state pre-schools and creches (Molteno, 1985). There were no significant differences in the percentage with a BAD NII outcome between those who had and who had not attended pre-school (23% versus 31%), and pre-school attendance did not contribute significantly in the multivariate analyses to the prediction of NII outcome. However, when all medical and psychosocial variables were taken together, attendance at pre-school did contribute significantly to increase PIQ at 1 year. (The median PIQ of those who had attended pre-school was 104 versus 87 in those who had not, and only 3% of those attending pre-school had a PIQ below 80, versus 23% of those who had not attended pre-school).

15.7.19 Behaviour Disturbance

Behaviour disturbance was defined as a score of 13 or more on the Rutter Parent Scale. Such behaviour disturbances characterized 42% of the moderates and 35% of the severes, but only 15% of the very severes. On a univariate basis, a BAD NII outcome occurred in significantly more of those who did not have disturbed behaviour than in those who did (35% versus 15%), and in the multivariate analysis of the psychosocial variables alone, the presence of behaviour disturbance decreased the probability of a BAD NII outcome. When the medical and psychosocial variables were combined, the presence or absence of behaviour disturbance did not contribute significantly to the prediction of either NII or PIQ outcome.

It would seem that behaviour disturbance may put the child at an increased risk of sustaining a mild head injury (Black et al, 1969; Craft et al, 1972; Rutter et al, 1984), but does not contribute significantly to the determination of cognitive outcome.

15.7.20 Previous Head Injury

Children with severe previous head injuries were not included for cognitive follow-up. About 17% of the total head-injured group had had minor head injuries at some time in the past. There was a trend for fewer of those who had suffered such injuries to have a BAD NII outcome than of those who had not (14% to 32%). In the multivariate analyses, previous head injury did not contribute significantly to the prediction of outcome on either NII or PIQ.

In adults it has been shown that those who have had previous head injuries have a greater initial deficit and take longer to recover (Gronwall & Wrightson, 1975). It is possible that this effect was not found in the present study because more of those with a moderate head injury (22%) than of those with a severe or very severe head injury (13%) had sustained a minor head injury previously. However, within the moderate group, none of those with a previous head injury have a BAD NII outcome or a PIQ below 80.

15.8 Very Severes with GOOD NII Outcome

Group trends are analysed in the next section which considers the statistical prediction of cognitive outcome from medical, psychosocial and neuropsychological variables. However, it is also useful to look on an individual basis at the 5 very severes (18%) who were not rated as having a BAD NII outcome. None were admitted in a deep coma, but with GCS scores of 8 or higher. They either had secondary deterioration within hours of injury or had a plateau which lasted for days. Only 2 were scanned and both had focal lesions with associated oedema and diffuse brain swelling. The suggestion, then, is of less primary damage. Four had additional injuries which could have contributed to the deterioration or plateau in their GCS scores.

PTA ranged from 9 to 25 days. The child with PTA of 9 days, C.P., was possibly misclassified, in that confusion attributable to other causes was mistaken for post-traumatic amnesia (see p.360). She was the only one of the 5 without behaviour changes at 1 year after head injury. Common complaints in the other 4 are dizziness, headaches and easy fatigueability. Overeating, listlessness and apathy, and moody, changeable behaviour, are each reported in at least 3 of the 4. Three are struggling at school, 2 of whom were above average previously. Disinhibition is reported in 2 of the 4. Analysis of behavioural changes after trauma is another part of the UCT Head Injury Survey and is not yet complete. The suggestion is, however, that these children have behavioural change which is likely to impede their school progress and they are not the same as they were prior to head injury, even though they do not have detectable cognitive impairment on the NII or PIQs below 80.

15.9 The Models Predicting Cognitive Outcome

In order to determine to what extent the probability of a BAD NII outcome could be predicted on the basis of such information as was to be found in the patient's medical folder without asking further questions of the parents, the medical variables discussed above, plus age and sex, were entered into a backwards stepping regression analysis. Long PTA, neurosurgery, other surgery, complications, clinical evidence of a basal fracture and male sex increased the probability of a BAD NII outcome, while the presence of a skull fracture on x-ray reduced the probability. Using the coefficients obtained in the analysis, 89.5% of cases were correctly predicted.

The model using medical plus psychosocial variables, correctly predicted about the same percentage of cases (88.6%) but the median probability of a GOOD or BAD NII outcome was better dichotomized. The variables contributing to the medical model were retained (with different coefficients) and the psychosocial adversity score also contributed to the probability of a BAD NII outcome. When

premorbid school progress was added for a reduced number of cases to the medical and psychosocial variables found significant for the total number of cases, PTA length, poor school progress, neurosurgery, complications and a clinical basal fracture contributed to the probability of a BAD NII outcome, while the presence of a skull fracture on x-ray reduced the probability of a BAD outcome. The model, with the coefficients given, correctly predicted 88.5% of the BAD cases and 96% of the GOOD cases (94% of all cases). This model may be suitable for use with other head-injured samples as, besides basic medical data, it requires knowledge only of whether the child is making adequate progress at school, or is struggling or has failed previously. It would be necessary to cross-validate the usefulness of these variables in predicting neuropsychological impairment in another sample of head-injured children. It would also have to be determined whether the model predicts impairment only on the particular battery of tests used, or whether these variables are associated with impairment on other neuropsychological batteries which measure similar functions.

In the model using psychosocial variables alone to predict the probability of a BAD NII outcome, the only variables retained were the psychosocial adversity score and the absence of disturbed premorbid behaviour (Rutter Parent Scale <13), and prediction of BAD cases was poor (only 21% correct) although prediction of GOOD outcome cases was good (97.6%) correct. The use of neuropsychological variables collected at intake to predict outcome at 1 year fared far better (91.5% of all cases correct), but the model which best predicted the probability of a BAD NII outcome was one which added the significant neuropsychological measures to the significant medical and psychosocial variables. This model, with the coefficients derived, predicted 91% of BAD cases and 97.5% of GOOD cases correctly (95.6% of the total cases). Again, the usefulness of these variables would need to be cross-validated on other head-injured children, and it is to be expected that this particular combination of neuropsychological with medical and psychosocial variables

would achieve its best level of prediction if the same neuropsychological battery were used.

16. CONCLUSIONS

16. CONCLUSIONS

The conclusions below were derived from a neuropsychological follow-up study of children between ages 6 and 14 admitted after head injury to UCT hospitals in the Cape Peninsula between mid-1983 and mid-1985. These children are typical of such admissions in that the disadvantaged sections of the population are over-represented. In terms of severity and nature of injury sustained and psychosocial background, they seem typical of children admitted to Trauma or Neurosurgery units as reported in overseas studies.

16.1 Severity Issues

There is clear evidence of a dose-response relationship between the severity of head injury sustained and the extent of cognitive deficit exhibited. This relationship is evident in the size of initial deficits, in the patterns of recovery shown, and in the persistence of cognitive deficits at 1 year post-injury.

The very severely head injured (those with PTA persisting over 7 days or coma, [GCS $\leq$8], lasting 6 or more hours) have significantly lower scores than the controls on almost all measures of intelligence, language, visuomotor and memory functions, both initially and throughout the one year follow-up. Their scores also tend to be below those of children sustaining moderate and severe head injury. Recovery of function occurs both in the first 4 months and in the second part of the year, but there is no return to control group levels. Just over 80% of this group have scores below at least 95% of the control group on five or more of the neuropsychological measures at 1 year after head injury. Sixty percent have a Full-Scale IQ below 80. About 60% have defective verbal memory, and about 40% have persistent impairment on language measures and on visuomotor measures. It can be concluded therefore that this severity

of head injury is associated with persistent cognitive impairment.

The severely head injured (PTA from 1 to 7 days, and either no unconsciousness or unconsciousness lasting 5 hours or less) were defective relative to the controls on many measures, particularly at the initial assessment and at the assessment 3 months later. Recovery of function occurred across both the first and second intervals, and this group has significantly lower scores than the controls only on IQ, memory and naming measures at 1 year after injury.

Generally, the scores of the severely head injured tended to be intermediate to those of the moderately and very severely head injured, particularly at the first assessment. Differences between the severely and very severely injured were often statistically significant, but almost none of the differences between the severely and the moderately head injured were statistically significant. At 1 year after head injury, 18% of the severely head injured have scores below 95% of the controls on five or more of the neuropsychological measures and 23% have a Full Scale IQ below 80. On the language measures, 20 to 30% show persisting impairment, and memory deficits occur in about 25%. On the visuomotor measures, however, there is recovery to the control level by 1 year and the percentage with persisting impairment does not exceed the 5% defined as impaired in the control group.

The moderately head injured, (PTA less than 24 hours, and who mostly had either no or brief unconsciousness), showed initial deficits relative to the controls on intelligence, memory and the Purdue pegboards when tested at a median of 6 days after injury. Recovery, particularly on measures tapping attention, information-processing and visuomotor speed, occurred in the first 3 months after injury, such that most measures were at control group level by 3 months. At 1 year, however, the mean scores of the moderates are still significantly below those of the controls on measures of intelligence, memory and picture

naming. Only 9% have persisting impairment on five or more measures in the neuropsychological battery at 1 year and only 8% have a PIQ below 80.

It can be concluded therefore, that mild head injury (PTA less than 8 days) produces transient impairment of cognitive functions. Deficits are greatest immediately after head injury and show progressive recovery over the following months. Less than 10% of those with PTA under 1 day, but up to 25% of those with PTA between 1 and 7 days, may show persisting impairment of cognitive functions.

16.2 Recovery Issues

Severity of injury determines patterns of recovery. The very severely head injured show recovery of functions continuing into the second half of the year. On some motor and memory measures, no further recovery occurs after the first 4 months. On more complex motor measures, recovery may not begin until after the first 4 months. The severely head injured show the recovery pattern characteristic of the very severely head injured. The moderately head injured have significantly greater recovery in the first 3 months than in the remainder of the year.

Recovery in the head injured does not usually exceed in magnitude the improvement shown by the control group, which is attributable to practice effects or to recovery from the nonspecific effects of trauma. The functions which show gains and the time span over which gains occur, however, differ in the head injured and the controls.

16.3 Functions Affected by Head Injury

Intelligence, memory and picture naming are the functions most sensitive to impairment after head injury in the present study. Deficits on these measures persist at 1 year in all the head-injured groups. Visuomotor deficits occur in the initial stages after mild head injury, but have

recovered by 3 months in those with moderate head injury and by 1 year in those with severe head injury.

Performance on the battery of neuropsychological tests, from which a Neuropsychological Impairment Index (NII) was derived, reflected more accurately than PIQ level the effects of head injury at 1 year.

A low PIQ after head injury is associated with long PTA, no clinical evidence of a basal skull fracture, premorbid psychosocial adversity, absence of preschool attendance, female sex and Afrikaans language usage. Adequate premorbid school progress predicts a higher PIQ than poor premorbid school progress.

The probability of a BAD NII outcome (NII $\geq$5) after head injury, is increased by the length of PTA, presence of neurosurgery, other surgery, complications, clinical evidence of a basal skull fracture, psychosocial adversity and male sex, and is decreased by the presence of skull fracture on x-ray. Poor premorbid school progress increases the probability of a BAD NII outcome.

16.4 Predicting Outcome: Medical Variables

While severity indices such as depth of coma (measured on the GCS) and duration of coma are associated with BAD NII outcome on a univariate basis, they do not contribute to the prediction of NII outcome (or to PIQ) when PTA is included in the equation. The presence of multiple injuries is significant in the univariate, but not the multivariate prediction of outcome. In the multivariate analysis, the presence of severe additional injury is probably reflected in the retention of complications and of surgery-other-than neurosurgery as predictors of outcome. The occurrence of an early seizure contributes to the univariate, but not the multivariate prediction of cognitive outcome. Clinical evidence of a basal skull fracture contributed to the probability of a BAD NII outcome on a multivariate basis, but, in contrast, predicted an increase in PIQ.

16.5 Predicting Outcome: Psychosocial variables

The level of premorbid psychosocial adversity is inversely related to PIQ and increases the probability of a BAD NII outcome. Preschool attendance increases PIQ. Knowledge of premorbid school progress, however, replaces the contribution of both psychosocial adversity and preschool attendance in predicting PIQ. Poor premorbid school progress contributes significantly to the probability of a BAD NII outcome, particularly in the moderate and severely head-injured groups. Premorbid behaviour disturbance increases the risk of suffering a mild head injury, but decreases the probability of a BAD NII outcome. Afrikaans language usage contributed to the probability of a BAD NII outcome only on a univariate basis, but contributed to the lowering of PIQ. Female sex was associated with a low PIQ, but male sex increased the probability of a BAD NII outcome. Previous minor head injury was more common in the mildly than the severely head injured, but did not contribute to predicting cognitive outcome.

16.6 Predicting Outcome: Neuropsychological Variables

Performance IQ on the WISC-R measured when the child is first out of PTA, is the best predictor of PIQ at 1 year, accounting for 74.5% of the variance.

The probability of a BAD NII outcome at 1 year is best predicted by a model using length of PTA, presence of complications, absence of a skull fracture on x-ray, psychosocial adversity and the scores on Word Fluency, Purdue Both hands, Consistent Retrieval and VIQ measured once the child is out of PTA. This model correctly predicts NII outcome in 96% of all cases.

16.7 Recommendations for Future Research

There is a need for further prospective longitudinal studies of cognitive functions in children suffering head injuries. The only two such previous studies which have matched controls were those of the Chadwick-Rutter group in Britain (1981) and of the Klonoff group in Canada (1977). In this latter study, half the sample was lost to follow-up over the 5 year period, and cases were not grouped according to severity of injury sustained, making it difficult to generalize findings. The very severely head injured in the present study were retested 2 years after head injury. A 5 year follow-up was planned, but not carried out for lack of resources.

The present findings should be extended by investigating various areas in more detail:

1. Do those with PTA less than 1 hour have transient cognitive deficits after head injury? The suggestion from the present data, where memory functions were analysed in this subgroup, is that scores are lower than those of the controls at the early assessment only.

2. What is the percentage of children likely to exhibit persisting impairment as a function of PTA length? Larger numbers of children in the subgroup with PTA between 1 and 2 weeks need to be followed up. The suggestion from the present study is that two thirds will have persisting impairment, but this was based on only 6 children.

3. What is the role of premorbid school backwardness or of pre-existing learning disabilities in predicting outcome after head injury? The University of Texas group (Levin & Eisenberg, 1979b; Ewing-Cobbs et al, 1987) exclude children without adequate premorbid school achievement from follow-up after head injury.

This may not be a realistic approach as studies, including the present one, have shown that such children constitute from one third to a half of head-injured populations (Costeff et al, 1985; Haas et al, 1987; Mahoney et al, 1983; Rutter et al, 1980). The present study rated reports of struggling or failure at school as poor premorbid school progress, but either may have been attributable to factors other than lack of ability. Several children who had failed previously, did well at school thereafter. A number of children had just started or were just about to start school when the head injury occurred (peak age for head injury in the UCT sample is 6 to 7 years) and it was not known whether any learning disabilities were present. A more in-depth study of the effects of low premorbid intelligence and of learning disabilities on outcome after mild head injury, is needed. This is unlikely to be achieved in the South African context where the children most likely to suffer head injuries, the socioeconomically disadvantaged, are least likely to attend schools where there is routine testing of intelligence and academic achievement.

4. Does the specific neuropathology sustained in head injury produce clinical subsyndromes? Much better techniques for imaging focal and diffuse lesions are now available than was the case when the present study was initiated. More thorough investigation of the relationships between focal lesions and specific deficits, and between the resolution of lesions, oedema, diffuse brain swelling and the course of cognitive recovery, are needed. However, these relationships may be difficult to establish statistically. Given the site of injury, the early neurological signs, secondary events, radiological investigations and neuropsychological test findings, we could discover clinical syndromes, but taking one or even several of these variables alone resulted in unacceptable classification of the localization of injury.

5. Are those who suffered a very severe head injury (PTA over 7 days) but who have no evidence of cognitive impairment at 1 year, behaviourally normal? The present study suggests that they are not and that this affects their school work. However, confirmation of this must await analysis of behaviour problems in the control group and of family circumstances which could have contributed to behavioural change. The Chadwick-Rutter study found that factors other than severity of head injury influenced behaviour after head injury (Brown et al, 1981).

6. What is the nature of deficits in specific cognitive areas? The present study provided some answers, but a more in-depth analysis of language, memory and visual-motor functions, perhaps on a theoretical basis, is needed. The role of attentional deficits as a possible core syndrome also warrants investigation. Difficulties in concentration and in fatigueability were observed in the present study, but no attempt was made to correlate these features with the cognitive deficits found.

7. What are the effects of rehabilitation on cognitive recovery? Some of the children in the present study had physiotherapy and/or occupational therapy in the acute stages after head injury. Once they were discharged, this would have been on no more than a weekly basis. In the long-term, excellent rehabilitation facilities are available at the Eros and Vista Nova schools for children with cerebral palsy. Children from this study were on the waiting lists for these schools, but were not likely to be admitted within the first year after head injury. In the subacute phase, then, no rehabilitation therapy was offered. Articles on rehabilitation training in children appear to be at the level of identifying problems, needs and objectives (eg Molnar & Perrin, 1983; Telzrow, 1985), but more structured models are

available (eg Haarbauer-Krupa et al, 1985) and need evaluation. The need for a multifaceted approach has been stressed by Ben-Yishay and Diller (1983b). This study has shown the advantages of using a multidisciplinary team to collect data to predict outcome and supports a multifaceted approach to head injury, both in research and management.

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APPENDIX

APPENDIX A - Data Summary Sheets

A1. Summary of Accident and Medical Data Collected
A2. Summary of Psychosocial Data Collected
A3. Neuropsychological Test Summary Sheet

APPENDIX B - Test Materials

B1. Word Fluency: Raw score age Equivalents
B2. Shopping Lists: Frequency count on items

APPENDIX C - Tables of Statistical Details

APPENDIX A - DATA SUMMARY SHEETS

Appendix A1 - Summary of Accident and Medical Data Collected

Identifying Data

Folder number, name, address, birthdate, sex, ethnic group, hospital, ward, date of injury, time of admission, referral source, first neurosurgical contact.

Accident

History: cause, time, place, custodian
Cause details:
Transport: type, weather, traffic density, visibility, speed and force of impact, driver, associated factors.
Fall: height of fall, prior activity.
Assault/Abuse: instrument, agent, intentionality.
Sport, Miscellaneous.

Management

Pre-hospital: help summoned, time till arrival, clinical state, lay management, transfer to hospital.
Hospital: surgery, medication, referrals, other.
Investigations: x-rays, CT, ultrasound, EEG, other.

Clinical Details

General: temperature, blood pressure, pulse, HB, respiration, infection.
Glasgow Coma Scale: admission, 24 hours.
Pupil size, reactivity; limb movements, cranial nerves, speech, vomiting, drowsy, irritable, vision.
Cranium: external injuries, clinical evidence of base of skull fracture.
Other injuries: location, details.
Seizures: activity, type, onset, duration.
Complications: aspiration, wound sepsis, meningitis, cerebrospinal fluid leak, metabolic disturbance, other.

Daily Progress Notes

GCS, orientation, activity, talking, feeding, emotional state, continence, aches and pains.

Discharge Data

Date, days in hospital, destination, medication, skull defect, wound healed.

Severity Classification

Period of coma, length of amnesia, death.

Follow-up

Appointments with neurosurgery and other departments; readmission details.

Appendix A2 - Summary of Psychosocial Data Collected

Identifying Data

Folder number, name, address, birthdate, sex, ethnic group, home language, religion, handedness.

Development and Health

Pregnancy, labour, delivery, post-natal state, milestones, crying frequency, serious illnesses.

Behaviour

Sleeping, eating, bedwetting, activity level, irritability, aggression, impulsiveness, talkativeness, carelessness, anxiety, moodiness, discipline problems, Rutter Parent Scale.

Education

Preschool attendance, school standard, school details, changes, regularity of attendance, progress, best subject, weakest subject, remedial teaching, study habits, relationship with teachers.

Social

Friends, extramural activities.

Income/Housing Details

Income source, total income, number of dependents, finance problems, housing type, number of rooms, facilities, rent, neighbourhood, number sharing room and bed with patient, residence changes.

Community Resources

Community centre, creche, clinic, public transport playground, shops, church, other.

Current Child Care/Social Problems

After school, holiday care; health of family members; psychiatric problem in family members; alcoholism, drug abuse, crime, anti-social behaviour in household members; child abuse/neglect; welfare organization involved.

Family Composition

Father and mother: age, relationship to patient, state of marriage, education and occupation details.
Siblings: age, sex, school standard, school failure, occupation.
Other occupants of home: number, age, sex, relationship details.
Family relationships, stress, leisure activities.

Appendix A3 - Neuropsychological Test Summary Sheet

NAME: ..... FOLDER NO: ..... ADDRESS: .....
AGE: ..... SEX: ....
BORN: ..... RACE: ..... PHONE: .....
DATE OF INJURY: ..... DATE: ..... ASSESSMENT: 1, 3/12, 6/12, 1 yr
DETAILS OF INJURY: Other Injuries:
GCS: Unconscious: Skull #: Neurological:
24 hr: PTA: Lesion: EEG:

A. INTELLIGENCE TEST S.Binet/WPPSI/WISC-R/S.A.WAIS Full Scale IQ: .....

Verbal IQ: ..... Performance IQ: .....
Information: ..... Picture Completion: .....
Comprehension: ..... Block Design: .....
Arithmetic: ..... Coding: ..... (Animal House)
Digit Span: ..... Mazes: ..... Incoordination: .....
(sentences)

B. MOTOR/VISUOGRAPHIC/SPEED HAND: .....

1. Purdue - Preferred R/L: ..... Non-preferred R/L: ..... Both: .....
2. Finger to thumb - R/L: ..... " R/L: .....
3. Detroit 2' Score: ..... Age equivalent: .....
4. Beery Score: ..... Age equivalent: .....

C. LANGUAGE - English/Afrikaans/Xhosa/Other

5. Word Fluency: ..... Age equivalent: ....
6. Comprehension: ..... Repeat: ....
9a. Sentence Repetition: ..... Age equivalent: ....
9b. Digits forward: .....
10. Renfrew Naming: ..... Age equivalent: ....
Time per word: .....

8. Token Test

Part	Raw	Age	Grade	Equi
I				
III				
V				

D. MEMORY (7) Trials: 1 2 3 4 5 6 7 8 Total

Recall: ..... Confabulation: ....
Longterm storage: ..... Repetition: ....
List learning: ..... Interference: ....

E. PERSONALITY

Rutter Parent Inventory: ..... Rutter Teacher Inventory: .....
Social disinhibition: ..... Psychosocial Adversity: .....

F. BEHAVIOUR DURING TESTING

Concentration: persists well; slightly distractable; very distractable; gives up easily
Activity: appropriate; listless; restless; very restless
Cooperation/Motivation: good; fair; poor
Affect: appropriate; shy; very shy; tearful; over cheerful; labile

Echolalia: some; marked Speech: OK; immature articulation; impaired
Perseveration: some; marked Tired: no; some; very Free; impeded; traction

Quiet office; disturbed office - Quiet ward; noisy ward

APPENDIX B - TEST MATERIALS

Appendix B1 - Word Fluency: Raw Score Age Equivalents

Age Equivalent	6				7					8			9				10				
Raw Score	15	16	17	18	19	20	21	22	23	23	24	25	26	27	27	28	29	30	31	31	
Age Equivalent	11				12					13			14				15				
Raw Score	32	33	34	35	35	36	37	38	38	39	39	40	40	41	41	41	41	42	42	42	

Appendix B2 - Shopping Lists: Frequency Counts on Items

Shopping Lists

The Child Guidance Clinic of the University of the Western Cape asked 79 children in standards 2 and 3 in the Manenberg area (lower socioeconomic) to write down lists of things they would buy when shopping at a supermarket. The frequency with which items appeared was counted. Three shopping lists were derived such that each contained staple foods, fruit, vegetables, treats, cleaning materials, etc, and an approximately even mixture of high and low frequency items. As can be seen, the total frequency count of the items on the three lists is very similar.

List A		List B		List C	
Item	Frequency	Item	Frequency	Item	Frequency
Sugar	69	Bread	60	Rice	55
Flour	41	Eggs	41	Fish oil	44
Pears	6	Peaches	0	Bananas	10
Matches	1	Cigarettes	4	Newspapers	2
Sweets	26	Oros	27	Chips	15
Tea	52	Cheese	50	Coffee	50
Vim	2	Toothpaste	8	Soap powder	27
Peanutbutter	16	Salt	18	Tomato sauce	12
Onions	16	Peas	46	Cabbage	21
Jelly	4	Chocolate	8	Icecream	10
Carrots	22	Beetroot	10	Apples	17
Fish	48	Soup	3	Sausages	3
Total	303	Total	275	Total	293

APPENDIX C - Tables of Statistical Details

Table C1

WISC-R IQs: Significance of Differences Between Groups, Confidence Limits

Group	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
				Verbal					
C-Mod	0.6	7.5	14.3	-1.7	5.7	13.0	-1.3	6.4	14.1
C-Sev	0.3	7.8	15.2	0.2	8.1	15.9	0.04	8.4	16.8
C-V.Sev	10.8	19.0	27.2	11.1	19.9	28.7	9.3	18.5	27.7
M-Sev	-6.9	0.3	7.5	-5.1	2.4	9.9	-5.9	2.0	9.9
M-V.Sev	3.6	11.5	19.5	5.8	14.3	22.7	3.3	12.1	20.9
S-V.Sev	2.8	11.3	19.7	3.0	11.9	20.8	0.7	10.1	19.4
				Performance IQ					
C-Mod	2.5	10.7	18.8	1.5	9.9	18.3	0.3	9.2	18.1
C-Sev	0.2	9.0	17.9	0.8	9.8	18.8	-3.8	5.8	15.4
C-V.Sev	21.6	31.5	41.4	20.3	30.5	40.6	15.6	26.3	37.0
M-Sev	-10.1	-1.7	6.8	-8.6	-0.1	8.5	-12.5	-3.4	5.7
M-V.Sev	11.2	20.8	30.4	10.8	20.6	30.3	6.9	17.1	27.4
S-V.Sev	12.3	22.5	32.6	10.4	20.7	30.9	9.7	20.6	31.4
				Full Scale IQ					
C-Mod	2.3	9.6	17.0	0.7	8.5	16.2	-0.02	8.2	16.3
C-Sev	0.6	8.6	16.6	1.3	9.6	17.8	-1.3	7.5	16.4
C-V.Sev	17.8	26.8	35.7	17.7	27.1	36.4	13.8	23.6	33.5
M-Sev	8.8	-1.0	6.7	-6.8	1.1	8.9	-9.0	-0.6	7.7
M-V.Sev	8.5	17.2	25.8	9.6	18.6	27.6	6.0	15.5	24.9
S-V.Sev	9.0	18.2	27.4	8.1	17.5	27.0	6.1	16.1	26.1

C=Controls, M or Mod=Moderates, S or Sev=Severes, V.Sev=Very Severes.

Table C2

WISC-R IQs: Significance of Changes Within Groups, Paired t-tests

Recovery Interval	Controls t	Controls p	Moderates t	Moderates p	Severes t	Severes p	V.Severes t	V.Severes p
				Verbal IQ				
T1 to T2	2.04	.049	4.40	.0001	1.96	.058	1.60	.123
T2 to T3	2.24	.031	1.82	.074	1.85	.073	2.77	.010
Diff.	0.11	.910	-1.64	.108	-0.36	.724	0.75	.458
				Performance IQ				
T1 to T2	6.35	.0001	6.42	.0001	5.38	.0001	5.47	.0001
T2 to T3	1.95	.058	2.79	.007	4.08	.0002	4.05	.0004
Diff.	-2.43	.020	-3.20	.002	0.01	.991	-1.29	.209
				Full Scale IQ				
T1 to T2	6.59	.0001	6.58	.0001	4.64	.0001	4.10	.0004
T2 to T3	2.76	.009	2.92	.005	4.01	.0003	4.73	.0001
Diff.	-2.07	.046	-2.48	.017	-0.17	.866	-0.05	.961

Diff=Difference (T1 to T2) - (T2 to T3).

Table C3

WISC-R Verbal Subtests: Significance of Differences Between Groups, Confidence Limits

Groups	Initial Assessment (T1)			3 Months Later (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
				Information					
C-Mod	-0.3	1.1	2.6	-0.2	1.2	2.7	-0.4	1.1	2.7
C-Sev	-0.5	1.1	2.6	0.2	1.8	3.4	0.4	2.1	3.8
C-V.Sev	1.3	3.0	4.7	1.6	3.3	5.1	2.0	3.9	5.7
M-Sev	-1.5	-0.04	1.4	-1.0	0.5	2.0	-0.6	1.0	2.6
M-V.Sev	0.2	1.9	3.5	0.4	2.1	3.8	1.01	2.8	4.5
S-V.Sev	0.2	1.9	3.7	-0.2	1.5	3.3	-0.08	1.8	3.6
				Comprehension					
C-Mod	-0.6	0.8	2.2	-0.9	0.5	2.0	-0.4	1.0	2.5
C-Sev	-0.8	0.7	2.3	-0.8	0.8	2.4	-0.6	1.0	2.6
C-V.Sev	0.8	2.4	4.1	1.2	2.9	4.7	1.02	2.8	4.5
M-Sev	-1.5	-0.07	1.4	-1.2	0.3	1.7	-1.5	-0.02	1.5
M-V.Sev	-0.01	1.6	3.2	0.7	2.4	4.0	0.1	1.7	3.4
S-V.Sev	-0.04	1.7	3.4	0.4	2.1	3.9	0.01	1.8	3.5
				Arithmetic					
C-Mod	-0.2	1.3	2.7	-0.5	0.9	2.3	-0.8	0.7	2.1
C-Sev	-0.3	1.3	2.9	-0.2	1.3	2.8	-1.1	0.5	2.1
C-V.Sev	1.4	3.1	4.9	2.0	3.7	5.4	1.0	2.7	4.5
M-Sev	-1.5	0.0	1.5	-1.05	0.4	1.8	-1.6	-0.1	1.4
M-V.Sev	0.2	1.9	3.6	1.2	2.8	4.4	0.4	2.1	3.7
S-V.Sev	0.05	1.9	3.7	0.7	2.4	4.1	0.4	2.2	4.0
				Digit Span					
C-Mod	0.2	1.7	3.3	-0.8	0.9	2.5	-0.3	1.5	3.2
C-Sev	0.2	1.9	3.6	-0.3	1.5	3.3	-0.05	1.9	3.8
C-V.Sev	1.9	3.7	5.6	1.2	3.2	5.2	0.6	2.7	4.8
M-Sev	-1.5	0.2	1.8	-1.04	0.7	2.4	-1.4	0.4	2.2
M-V.Sev	0.2	2.0	3.8	0.4	2.3	4.2	-0.8	1.2	3.2
S-V.Sev	-0.1	1.8	3.8	-0.4	1.7	3.7	-1.3	0.9	3.0

Table C4

WISC-R Verbal Subtests: Significance of Changes Within Groups, Paired t-tests

Recovery Interval	Controls t	Controls p	Moderates t	Moderates p	Severes t	Severes p	V.Severes t	V.Severes p
				Information				
T1 to T2	1.54	.132	1.39	.170	-0.33	.744	1.09	.287
T2 to T3	1.81	.078	2.30	.025	0.18	.860	-0.27	.791
Diff.	-0.06	.950	-0.37	.712	-0.35	.727	0.89	.384
				Comprehension				
T1 to T2	1.88	.068	3.45	.001	2.15	.039	0.49	.631
T2 to T3	2.06	.047	0.80	.429	0.86	.396	3.11	.005
Diff.	0.06	.956	1.84	.071	0.93	.358	-1.15	.261
				Arithmetic				
T1 to T2	0.78	.442	2.29	.026	0.99	.330	-0.31	.762
T2 to T3	0.17	.866	0.59	.558	1.66	.106	2.43	.022
Diff.	0.37	.714	1.05	.300	-0.24	.816	-1.87	.072
				Digit Span				
T1 to T2	0.45	.653	3.25	.002	1.30	.200	2.14	.042
T2 to T3	1.38	.175	0.00	1.000	1.36	.183	2.46	.021
Diff.	-0.72	.477	1.98	.053	0.05	.958	-0.40	.691

Table C5

WISC-R Performance Subtests: Significance of Differences Between Groups, Confidence Limits

Groups	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
				Picture Completion					
C-Mod	0.9	2.5	4.0	0.8	2.5	4.08	0.2	1.7	3.2
C-Sev	0.3	1.9	3.6	-0.2	1.6	3.3	-0.7	0.9	2.6
C-V.Sev	3.5	5.3	7.1	2.9	4.9	6.8	2.6	4.4	6.2
M-Sev	-2.1	-0.5	1.1	-2.5	-0.9	0.7	-2.3	-0.7	0.8
M-V.Sev	1.1	2.9	4.6	0.6	2.4	4.3	1.0	2.7	4.5
S-V.Sev	1.5	3.4	5.2	1.4	3.3	5.3	1.6	3.5	5.3
				Block Design					
C-Mod	0.3	2.0	3.7	-0.05	1.6	3.3	-0.8	1.0	2.9
C-Sev	-0.5	1.3	3.1	-0.1	1.7	3.5	-1.04	1.0	3.0
C-V.Sev	1.8	3.8	5.8	2.2	4.2	6.2	0.7	3.0	5.2
M-Sev	-2.4	-0.7	1.1	-1.6	0.05	1.7	-2.0	-0.1	1.8
M-V.Sev	-0.1	1.8	3.8	0.7	2.6	4.5	-0.2	1.9	4.1
S.V.Sev	0.4	2.5	4.6	0.5	2.6	4.6	-0.3	2.0	4.3
				Coding					
C-Mod	0.2	1.8	3.4	-0.7	1.0	2.7	0.01	1.9	3.7
C-Sev	-0.1	1.7	3.5	-0.5	1.3	3.2	-0.6	1.4	3.4
C-V.Sev	3.2	5.2	7.2	2.1	4.2	6.3	2.3	4.5	6.7
M-Sev	-1.7	-0.1	1.5	-1.4	0.3	2.1	-2.3	-0.5	1.4
M-V.Sev	1.6	3.4	5.3	1.2	3.2	5.2	0.5	2.7	4.8
S-V.Sev	1.5	3.5	5.5	0.8	2.9	5.0	0.8	3.1	5.4
				Mazes					
C-Mod	-1.5	0.3	2.0	-1.2	0.8	2.7	-1.1	0.7	2.6
C-Sev	-1.3	0.6	2.5	-0.8	1.3	3.4	-1.6	0.4	2.4
C-V.Sev	3.0	5.1	7.2	2.5	4.9	7.3	1.6	3.8	6.1
M-Sev	-1.5	0.3	2.1	-1.4	0.5	2.5	-2.2	-0.3	1.6
M-V.Sev	2.8	4.9	6.9	1.9	4.1	6.4	0.9	3.1	5.2
S-V.Sev	2.4	4.5	6.7	1.2	3.6	6.0	1.1	3.4	5.7

Table C6

WISC-R Performance Subtests: Significance of Changes Within Groups, Paired t-tests

Recovery Interval	Controls t	Controls p	Moderates t	Moderates p	Severes t	Severes p	V.Severes t	V.Severes p
			Picture Completion					
T1 to T2	1.48	.148	-0.31	.755	0.63	.534	0.93	.361
T2 to T3	3.13	.003	3.06	.004	4.22	.0002	2.53	.018
Diff.	0.80	.431	3.80	.0004	2.56	.015	1.72	.098
			Block Design					
T1 to T2	1.90	.066	1.71	.094	0.26	.799	-0.65	.522
T2 to T3	2.44	.019	3.84	.0003	1.16	.254	1.05	.302
Diff.	-0.16	.870	1.11	.274	0.82	.419	2.33	.028
			Coding					
T1 to T2	-1.06	.299	2.51	.015	-0.49	.629	1.01	.324
T2 to T3	1.21	.237	4.57	.0001	3.12	.004	4.08	.001
Diff.	2.87	.007	0.44	.662	3.44	.002	1.62	.118
			Mazes					
T1 to T2	2.22	.033	1.78	.081	-0.03	.978	1.54	.135
T2 to T3	4.67	.0001	3.49	.001	2.49	.017	5.47	.0001
Diff.	0.67	.506	0.95	.346	2.43	.020	2.66	.013

Table C7

Language Production Measures: Significance of Differences Between Groups

Word Fluency: Confidence Limits on Tukey's studentized range

Group	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-6.8	9.1	24.9	-7.2	8.0	23.2	-8.5	5.6	19.7
C-Sev	0.4	17.8	35.1	-4.2	12.0	28.3	-9.5	5.7	20.9
C-V.Sev	22.7	41.6	60.6	12.4	30.6	48.8	8.7	25.5	42.2
M-Sev	-8.0	8.7	25.4	-11.4	4.0	19.5	-14.1	0.1	14.4
M-V.Sev	14.2	32.6	50.9	5.2	22.6	40.0	4.0	19.9	35.7
S-V.Sev	4.1	23.8	43.5	0.2	18.5	36.9	2.8	19.7	36.6

Renfrew Picture Naming: Confidence Limits on Tukey's studentized range

Group	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-0.1	3.2	6.4	-0.4	2.9	6.1	0.1	3.4	6.7
C-Sev	0.1	3.6	7.2	0.2	3.7	7.1	0.6	4.1	7.7
C-V.Sev	5.4	9.3	13.2	4.3	8.2	12.1	4.0	7.9	11.8
M-Sev	-2.9	0.5	3.9	-2.5	0.8	4.1	-2.6	0.7	4.1
M-V.Sev	2.4	6.1	9.9	1.6	5.3	9.1	0.8	4.5	8.2
S-V.Sev	1.7	5.7	9.7	0.6	4.5	8.4	-0.2	3.8	7.7

Speed of Naming: Mann-Whitney Values

Groups	Initial Assessment (T1)		3 Months (T2)	
	z	*p*	*z*	*p*
C-Mod	-1.57	.1156	-0.86	.3922
C-Sev	0.97	.3298	1.24	.2155
C-V.Sev	4.26	.0000	3.84	.0001
M-Sev	-0.33	.7378	0.38	.7054
M-V.Sev	3.60	.0003	3.88	.0001
S-V.Sev	3.44	.0006	2.93	.0033

Table C8

Language Production Measures: Significance of Changes Within Groups

Recovery Interval	Controls		Moderates		Severes		Very Severes	
				Word Fluency				
	t	p	t	p	t	p	t	p
T1 to T2	0.55	.588	0.54	.594	1.82	.078	3.44	.002
T2 to T3	-0.62	.536	-0.04	.970	0.75	.460	0.86	.396
Diff.	-0.79	.433	-0.33	.741	-0.42	.674	-1.31	.202
				Renfrew Picture Naming				
	t	p	t	p	t	p	t	p
T1 to T2	4.05	.0002	4.86	.0001	2.87	.007	3.68	.001
T2 to T3	1.29	.203	0.47	.637	1.91	.064	2.14	.042
Diff.	-1.32	.194	-2.45	.018	-0.64	.525	-1.25	.222
				Speed of Naming				
	Sign Rank	p	Sign Rank	p	Sign Rank	p	Sign Rank	p
T1 to T2	-89	.217	-363.5	.0005	-120.5	.070	-99	.018
T2 to T3	-36	.620	-148.5	.153	-166.5	.012	-127	.002
Diff.	26	.722	145.5	.162	15.5	.821	38	.368

Table C9

Sentence Repetition: Significance of Differences Between Groups, Confidence Limits

Groups	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-8.2	4.9	18.0	-12.3	1.2	14.8	-11.4	1.9	15.3
C-Sev	-6.1	8.3	22.7	-9.3	5.2	19.7	-10.2	4.2	18.6
C-V.Sev	9.2	25.1	41.0	3.2	19.4	35.6	3.8	19.7	35.6
M-Sev	-10.3	3.4	17.1	-9.8	4.0	17.7	-11.3	2.3	15.9
M-V.Sev	4.9	20.2	35.5	2.7	18.2	33.7	2.6	17.7	32.9
S-V.Sev	0.4	16.8	33.1	-2.2	14.2	30.5	-0.7	15.5	31.6

Table C10

Sentence Repetition: Significance of Changes Within Groups, Paired *t*-tests

Recovery Interval	Controls		Moderates		Severes		Very Severes	
	t	*p*	*t*	*p*	*t*	*p*	*t*	*p*
T1 to T2	-0.90	.376	1.02	.312	1.83	.076	2.69	.013
T2 to T3	-0.37	.712	-0.72	.476	0.62	.542	-0.14	.892
Diff.	0.40	.688	-1.07	.288	-1.06	.298	-2.02	.055

Table C11

Pictorial Identification: Significance of Differences Between Groups

Groups	Initial Assessment (T1)		3 Months (T2)		1 Year (T3)	
	X^2	*p*	X^2	*p*	X^2	*p*
C-Mod	1.51	.219	3.59	.058	1.23	.267
C-Sev	11.57	.001	11.69	.001	5.82	.016
C-V.Sev	17.83	.000	15.27	.000	19.96	.000
M-Sev	5.79	.016	3.37	.066	2.46	.117
M-V.Sev	11.20	.001	5.97	.015	15.67	.000
S-V.Sev	1.37	.241	0.57	.451	5.48	.019

Table C12

Pictorial Identification: Significance of Changes Within Groups

Recovery Interval	Controls		Moderates		Severes		Very Severes	
	X^2	p	X^2	p	X^2	p	X^2	p
T1 to T2	1.5	.221	1.13	.289	3.13	.077	1.45	.228
T2 to T3	0.5	.480	3.27	.070	5.14	.023	0.25	.617

Table C13

Token Test: Significance of Differences Between Groups

Groups	Initial Assessment (T1)		3 Months (T2)		1 Year (T3)	
			Part 1: X^2 or Fisher's exact p			
	X^2	p	X^2	p	X^2	p
C-Mod	3.21	0.073	Fisher's	0.209	Fisher's	1.000
C-Sev	4.99	0.025	Fisher's	0.171	Fisher's	1.000
C-V.Sev	13.42	0.001	Fisher's	0.001	Fisher's	0.018
M-Sev	0.27	0.604	Fisher's	1.000	Fisher's	0.697
M-V.Sev	4.42	0.035	12.17	0.001	Fisher's	0.026
S-V.Sev	2.46	0.117	9.30	0.002	Fisher's	0.014
			Part 3: X^2 or Fisher's exact			
	X^2	p	X^2	p	X^2	p
C-Mod	2.57	0.109	0.03	0.854	4.02	0.045
C-Sev	3.39	0.066	0.33	0.567	7.52	0.006
C-V.Sev	22.08	0.001	7.72	0.005	11.68	0.001
M-Sev	0.11	0.744	0.59	0.483	0.93	0.335
M-V.Sev	12.26	0.001	9.09	0.003	3.24	0.072
S-V.Sev	10.02	0.002	4.76	0.029	0.72	0.395
			Part 5: Confidence Limits			
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-0.2	2.1	4.4	-1.0	1.0	2.9	-0.5	1.5	3.5
C-Sev	-0.5	1.9	4.3	-1.3	0.8	2.9	-1.1	1.1	3.3
C-V.Sev	2.1	4.8	7.6	1.0	3.3	5.7	0.4	2.8	5.2
M-Sev	-2.6	-0.2	2.2	-2.2	-0.2	1.9	-2.5	-0.4	1.7
M-V.Sev	-0.1	2.7	5.5	0.1	2.4	4.6	-1.0	1.3	3.6
S-V.Sev	0.01	2.9	5.8	0.1	2.5	4.9	-0.8	1.7	4.1

Table C14

Token Test: Differences Between the Groups in Changes from T1 to T2, Confidence Limits

Groups	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-3.1	-1.3	0.4
C-Sev	-3.0	-1.2	0.6
C-V.Sev	-4.2	-2.1	-0.03
M-Sev	-1.7	0.2	2.0
M-V.Sev	-2.8	-0.8	1.3
S-V.Sev	-3.1	-0.9	1.2

Table C15

Token Test: Significance of Changes Within Groups

Recovery Interval	Controls		Moderates		Severes		Very Severes	
	Part 1: McNemar's Test							
	X^2	p	X^2	p	X^2	p	X^2	p
T1 to T2	1.00	.317	5.00	.03	5.00	.025	0.20	.655
T2 to T3	1.00	.317	0.00	1.00	1.33	.248	2.67	.103
	Part 3: McNemar's Test							
	X^2	p	X^2	p	X^2	p	X^2	p
T1 to T2	0.00	1.00	3.77	.05	1.92	.17	6.00	.014
T2 to T3	3.60	.06	0.00	1.00	0.00	1.00	1.00	.317
	Part 5: Paired t-tests							
	t	p	t	p	t	p	t	p
T1 to T2	0.89	.380	4.09	.001	2.33	.027	3.80	.001
T2 to T3	2.16	.037	0.94	.352	2.18	.037	3.54	.002
Diff.	0.80	.431	-1.89	.066	-0.69	.496	-0.56	.581

Table C16

Purdue Pegboard: Significance of Differences Between Groups, Confidence Limits

Groups	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
				Preferred Hand					
C-Mod	2.6	9.5	16.4	-3.8	2.0	7.7	-5.3	0.3	5.8
C-Sev	3.0	10.8	18.5	-1.1	5.1	11.3	-3.2	2.8	8.8
C-V.sev	21.6	30.6	39.7	11.6	18.9	26.2	10.8	17.8	24.8
M-Sev	-5.8	1.3	8.3	-2.7	3.2	9.1	-3.1	2.6	8.3
M-V.Sev	12.7	21.1	29.5	9.9	16.9	23.9	10.9	17.6	24.3
S-V.Sev	10.8	19.9	29.0	6.3	13.7	21.2	7.9	15.0	22.1
				Nonpreferred Hand					
C-Mod	0.2	6.5	12.8	-1.4	3.9	9.3	-5.1	0.6	6.2
C-Sev	3.6	10.6	17.5	1.8	7.5	13.3	-3.6	2.6	8.7
C-V.Sev	23.2	31.8	40.5	10.2	17.4	24.6	6.6	14.2	21.8
M-Sev	-2.5	4.1	10.6	-1.9	3.6	9.0	-3.8	2.0	7.8
M-V.Sev	17.0	25.3	33.6	6.5	13.5	20.4	6.3	13.6	20.9
S-V.Sev	12.5	21.3	30.1	2.7	9.9	17.1	4.0	11.6	19.3
				Both Hands					
C-Mod	-0.4	6.5	13.4	-3.7	1.4	6.5	-3.0	2.8	8.7
C-Sev	4.2	11.9	19.6	1.7	7.3	12.8	-0.1	6.3	12.6
C-V.Sev	24.5	33.9	43.2	11.9	18.9	25.9	7.8	15.7	23.6
M-Sev	-1.5	5.4	12.3	0.6	5.9	11.1	-2.6	3.4	9.4
M-V.Sev	18.7	27.4	36.0	10.7	17.5	24.3	5.2	12.9	20.5
S-V.Sev	12.6	22.0	31.3	4.6	11.7	18.8	1.4	9.4	17.5

Table C17

Purdue Pegboard: Significance of Changes Within Groups, Paired t-tests

Recovery Interval	Controls t	Controls p	Moderates t	Moderates p	Severes t	Severes p	Very Severes t	Very Severes p
			Preferred Hand					
T1 to T2	0.86	.396	6.04	.001	4.64	.001	5.01	.001
T2 to T3	0.67	.508	1.96	.056	1.40	.172	1.13	.273
Diff.	0.28	.781	2.63	.011	1.68	.104	3.18	.006
			Nonpreferred Hand					
T1 to T2	1.44	.159	2.81	.007	3.78	.001	5.68	.001
T2 to T3	-1.00	.323	1.27	.211	2.25	.031	0.66	.519
Diff.	1.57	.126	1.21	.231	1.12	.273	4.14	.001
			Both Hands					
T1 to T2	1.74	.093	5.50	.001	4.15	.001	4.76	.001
T2 to T3	0.48	.637	-0.82	.415	1.56	.129	1.63	.126
Diff.	0.86	.396	3.83	.001	1.84	.077	2.68	.018

Table C18

Purdue Pegboard: Differences Between the Groups in Changes from T1 to T2, Confidence Limits

Groups	Preferred Hand Lower Limit	Preferred Hand Difference Between Means	Preferred Hand Upper Limit	Nonpreferred Hand Lower Limit	Nonpreferred Hand Difference Between Means	Nonpreferred Hand Upper Limit	Both Hands Lower Limit	Both Hands Difference Between Means	Both Hands Upper Limit
C-Mod	-13.3	-6.8	-0.2	-8.6	-2.4	3.8	-10.4	-4.4	1.7
C-Sev	-13.8	-6.5	0.8	-10.8	-4.0	2.8	-11.5	-4.7	2.0
C-V.Sev	-22.2	-13.6	-5.1	-22.4	-14.1	-5.7	-22.7	-14.5	-6.3
M-Sev	-6.2	0.3	6.8	-7.9	-1.6	4.7	-6.3	-0.4	5.6
M-V.Sev	-14.7	-6.9	1.0	-19.7	-11.7	-3.7	-17.7	-10.1	-2.5
S-V.Sev	-15.7	-7.2	1.4	-18.5	-10.1	-1.6	-17.9	-9.8	-1.6

Table C19

Successive Finger Taps: Significance of Differences Between Groups, Confidence Limits

Groups	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
				Preferred Hand					
C-Mod	-2.6	-0.9	0.9	-1.7	-0.5	0.7	-1.3	-0.2	0.8
C-Sev	-3.4	-1.5	0.4	-2.8	-1.5	-0.2	-1.5	-0.3	0.8
C-V.Sev	-7.4	-5.1	-2.8	-6.5	-5.0	-3.5	-4.4	-3.1	-1.7
M-Sev	-2.4	-0.6	1.1	-2.2	-0.9	0.3	-1.2	-0.1	1.0
M-V.Sev	-6.3	-4.2	-2.1	-5.9	-4.5	-3.0	-4.1	-2.8	-1.6
S-V.Sev	-5.8	-3.6	-1.3	-5.1	-3.5	-2.0	-4.1	-2.7	-1.4
				Nonpreferred Hand					
C-Mod	-2.4	-0.8	0.8	-1.2	-0.1	1.1	-1.3	-0.3	0.8
C-Sev	-3.1	-1.4	0.3	-2.4	-1.1	0.2	-1.2	-0.1	1.1
C-V.Sev	-7.2	-5.0	-2.9	-5.7	-4.1	-2.5	-3.9	-2.5	-1.1
M-Sev	-2.3	-0.6	1.0	-2.3	-1.0	0.2	-0.9	0.2	1.2
M-V.Sev	-6.3	-4.2	-2.1	-5.6	-4.0	-2.5	-3.6	-2.3	-0.9
S-V.Sev	-5.8	-3.6	-1.4	-4.6	-3.0	-1.3	-3.8	-2.4	-1.0

Table C20

Successive Finger Taps: Significance of Changes Within Groups, Paired *t*-tests

Recovery Interval	Controls		Moderates		Severes		Very Severes	
	t	*p*	*t*	*p*	*t*	*p*	*t*	*p*
				Preferred Hand				
T1 to T2	-2.99	.005	-3.36	.002	-1.58	.124	-1.46	.161
T2 to T3	-0.23	.822	-2.61	.012	-4.68	.001	-4.28	.001
Diff.	-1.70	.098	-0.75	.456	0.93	.358	1.39	.182
				Nonpreferred Hand				
T1 to T2	-2.38	.022	-5.45	.001	-2.47	.018	-2.08	.054
T2 to T3	-0.12	.906	0.51	.613	-2.86	.007	-2.66	.017
Diff.	-1.13	.264	-3.58	.001	0.02	.980	-0.24	.813

Table C21

Successive Finger Taps: Differences Between the Groups in Changes from T2 to T3, Confidence Limits

Groups	Preferred Hand			Nonpreferred Hand		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-0.5	0.6	1.6	-1.3	-0.2	1.0
C-Sev	0.3	1.4	2.5	-0.1	1.1	2.4
C.V.Sev	0.9	2.2	3.6	-0.1	1.5	3.1
M-Sev	-0.2	0.8	1.8	0.1	1.3	2.5
M-V.Sev	0.4	1.6	2.9	0.2	1.7	3.2
S-V.Sev	-0.5	0.8	2.1	-1.2	0.4	2.0

Table C22

Incoordination on WISC-R Mazes: Significance of Differences Between Groups

Groups	Initial Assessment (T1)		3 Months (T2)		1 Year (T3)	
	X^2	p	X^2	p	X^2	p
C-Mod	1.97	.161	0.48	.488	0.05	.823
C-Sev	7.64	.006	3.17	.075	0.20	.652
C-V.Sev	12.26	.001	12.82	.001	3.84	.050
M-Sev	2.55	.111	1.49	.222	0.07	.796
M-V.Sev	6.12	.013	10.29	.001	3.54	.060
S-V.Sev	1.03	.310	3.97	.046	2.31	.128

Table C23

Detroit Motor Speed and Precision: Significance of Differences Between Groups, Confidence Limits

Groups	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-0.8	8.8	18.3	-3.0	5.1	13.1	-1.5	6.4	14.3
C-Sev	-1.7	8.8	19.3	-2.3	6.4	15.1	-3.0	5.5	14.0
C-V.Sev	15.6	27.1	38.6	16.9	26.6	36.3	20.4	29.9	39.4
M-Sev	-9.6	0.0	9.6	-6.9	1.4	9.6	-9.0	-0.9	7.2
M-V.Sev	7.7	18.3	29.0	12.2	21.5	30.8	14.4	23.5	32.6
S-V.Sev	6.9	18.3	29.8	10.3	20.2	30.0	14.8	24.4	34.1

Table C24

Detroit Motor Speed and Precision: Significance of Changes Within Groups, Paired t-tests

Recovery Interval	Controls		Moderates		Severes		Very Severes	
	t	p	t	p	t	p	t	p
T1 to T2	2.20	.036	2.64	.011	2.77	.009	2.13	.043
T2 to T3	0.77	.449	0.80	.430	2.13	.040	-0.56	.580
Diff.	1.10	.280	1.25	.217	0.58	.563	1.70	.102

Table C25

Beery VMI: Significance of Differences Between Groups, Confidence Limits

Groups	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
C-Mod	-0.8	4.7	10.2	-1.1	4.5	10.2	-3.4	2.5	8.4
C-Sev	0.3	6.3	12.2	-1.6	4.5	10.5	-6.7	-0.3	6.0
C-V.Sev	6.9	13.6	20.3	4.9	11.7	18.5	3.1	10.2	17.2
M-Sev	-4.2	1.6	7.3	-5.8	-0.1	5.7	-8.8	-2.8	3.2
M-V.Sev	2.4	8.9	15.4	0.6	7.2	13.7	0.9	7.7	14.4
S-V.Sev	0.4	7.3	14.3	0.3	7.2	14.1	3.4	10.5	17.7

Table C26

Beery VMI: Significance of Changes Within Groups, Paired *t*-tests

Recovery Interval	Controls		Moderates		Severes		Very Severes	
	t	*p*	*t*	*p*	*t*	*p*	t	*p*
T1 to T2	1.43	.162	0.27	.788	2.56	.015	2.22	.036
T2 to T3	-1.63	.112	0.28	.784	1.99	.054	-0.13	.899
Diff.	1.75	.088	0.03	.974	0.22	.829	1.30	.208

Table C27

Selective Reminding: Significance of Differences Between Groups, Confidence Limits

Group	Initial Assessment (T1)			3 Months (T2)			1 Year (T3)		
	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit	Lower Limit	Difference Between Means	Upper Limit
				Recall					
C-Mod	4.6	12.1	19.7	-1.6	5.1	11.9	0.1	4.9	9.6
C-Sev	10.3	18.6	26.9	0.7	7.9	15.1	0.6	5.7	10.8
C-V.Sev	21.4	30.4	39.3	8.2	16.5	24.8	9.5	15.1	20.7
M-Sev	-1.4	6.5	14.3	-4.1	2.8	9.6	-4.0	0.9	5.7
M-V.Sev	9.6	18.2	26.8	3.4	11.3	19.3	4.9	10.2	15.5
S-V.Sev	2.5	11.8	21.0	0.2	8.6	17.0	3.7	9.4	15.0
				Long Term Storage (LTS)					
C-Mod	2.8	12.2	21.5	-1.6	6.5	14.6	-0.4	5.2	10.8
C-Sev	9.4	19.7	30.0	0.1	8.8	17.5	-0.2	5.9	12.0
C-V.Sev	23.0	34.1	45.3	9.4	19.5	29.5	11.0	17.7	24.3
M-Sev	-2.3	7.5	17.3	-6.0	2.3	10.5	-5.1	0.6	6.4
M-V.Sev	11.3	22.0	32.7	3.3	13.0	22.6	6.1	12.5	18.8
S-V.Sev	2.9	14.5	26.0	0.6	10.7	20.8	5.0	11.8	18.6
				Consistent Retrieval (CR)					
C-Mod	9.1	20.8	32.5	-3.6	8.9	21.4	-1.2	8.5	18.3
C-Sev	19.3	32.2	45.1	2.5	15.9	29.3	2.9	13.4	24.0
C-V.Sev	33.1	47.1	61.1	14.7	30.0	45.4	14.9	26.4	38.0
M-Sev	-0.9	11.4	23.6	-5.7	7.0	19.7	-5.0	4.9	14.9
M-V.Sev	12.8	26.2	39.7	6.3	21.1	39.9	6.9	17.9	28.9
S-V.Sev	0.4	14.9	29.3	-1.4	14.1	29.7	1.3	13.0	24.7

Table C28

Selective Reminding: Significance of Changes Within Groups, Paired t-tests

Recovery Interval	Controls t	Controls p	Moderates t	Moderates p	Severes t	Severes p	Very Severes t	Very Severes p
			Recall					
T1 to T2	1.35	.187	4.48	.001	5.46	.001	4.39	.002
T2 to T3	1.18	.248	2.25	.029	2.50	.018	1.33	.196
Diff.	0.36	.720	1.96	.055	2.37	.024	2.26	.033
			Long Term Storage (LTS)					
T1 to T2	1.78	.083	3.41	.001	4.89	.001	4.19	.004
T2 to T3	0.72	.476	2.00	.051	2.57	.015	1.04	.310
Diff.	0.96	.344	1.26	.214	2.19	.035	2.19	.039
			Consistent Retrieval (CR)					
T1 to T2	1.49	.146	5.11	.001	5.54	.001	3.53	.002
T2 to T3	1.07	.291	1.88	.066	2.04	.049	1.49	.151
Diff.	0.53	.601	2.50	.016	2.31	.027	1.36	.188

Table C29

Selective Reminding: Differences Between the Groups in Changes from T1 to T2, Confidence Limits

Groups	Recall Lower Limit	Recall Difference Between Means	Recall Upper Limit	Long Term Storage Lower Limit	Long Term Storage Difference Between Means	Long Term Storage Upper Limit	Consistent Retrieval Lower Limit	Consistent Retrieval Difference Between Means	Consistent Retrieval Upper Limit
C-Mod	-12.2	-5.1	2.0	-13.3	-3.5	6.3	-21.3	-9.6	2.1
C-Sev	-17.3	-9.6	-1.8	-19.6	-8.8	1.9	-26.8	-13.9	-1.1
C-V.Sev	-21.2	-12.7	-4.1	-25.7	-13.8	-2.0	-28.3	-14.2	-0.02
M-Sev	-11.7	-4.5	2.7	-15.3	-5.3	4.7	-16.3	-4.3	7.6
M-V.Sev	-15.7	-7.6	0.5	-21.5	-10.3	0.9	-17.9	-4.6	8.8
S-V.Sev	-11.8	-3.1	5.5	-17.0	-5.0	7.0	-14.5	-0.2	14.1