NEUROPSYCHOLOGICAL FUNCTIONS FOLLOWING CLOSED HEAD INJURY IN INFANTS AND PRESCHOOLERS

A Dissertation

Presented to

the Faculty of the Department of Psychology

University of Houston

In Partial Fulfillment

of the Requirements for the Degree

Doctor of Philosophy

By

Linda Ewing-Cobbs

May, 1985

NEUROPSYCHOLOGICAL FUNCTIONS FOLLOWING CLOSED HEAD INJURY IN INFANTS AND PRESCHOOLERS

An Abstract of a Dissertation

Presented to

the Faculty of the Department of Psychology

University of Houston

In Partial Fulfillment

of the Requirements for the Degree

Doctor of Philosophy

By

Linda Ewing-Cobbs

May, 1985

Abstract

The traditional plasticity hypothesis proposes that recovery from brain injury is enhanced in children as compared with adults. To evaluate the plasticity hypothesis, performance on intelligence, motor, expressive language, and receptive language tests was examined during the subacute stage of recovery as well as eight months post-injury in 21 infants and preschoolers who sustained a closed head injury (CHI). Children with severe injuries (n=13) exhibited impaired consciousness for at least one day while children sustaining mild-moderate injuries (n=8) were rendered comatose for less than 24 hours. The design was a 2 (severity) x 2 (time of testing) factorial. To evaluate the effect of severity of CHI and to compare neuropsychological performance on baseline and follow up examinations, a repeated measures multivariate analysis of variance (MANOVA) was performed on summary scores from each of the four neuropsychological areas. On the baseline evaluation, children sustaining severe CHI were impaired on intelligence ($p<.005$), motor ($p<.005$), expressive language ($p<.05$), and receptive language ($p<.001$) functions relative to children with mild-moderate injuries. To determine which skill areas were most disrupted by CHI, the baseline and follow up summary scores from the 1) intelligence and motor and

2) expressive and receptive language areas were compared using a within-subjects MANOVA. Motor scores were more impaired than the intelligence scores on the baseline ($p<.05$) and follow up ($p<.05$) evaluations. Expressive language skills were significantly impaired relative to the receptive language skills on the baseline evaluation ($p<.05$). However, no significant differences were present on the follow up evaluation. The level of expressive language skills improved considerably over time while changes in performance on receptive language tasks was modest. The duration of impaired consciousness was a better predictor of the level of neuropsychological performance during the subacute stage of recovery from CHI than Glasgow Coma Scale scores obtained either at admission or 24 hours post injury.

Significant disruption of ability was observed in all of the skill areas examined and persistent behavioral deficits were apparent eight months post injury in children sustaining severe injuries. These findings are incompatible with the traditional plasticity hypothesis. Moreover, the incidence of dysphasia/dyspraxia was higher in the severely injured children than previously reported in older children and adults sustaining CHI. Since language skills develop rapidly during the preschool years, these findings support the hypothesis that skills in a rapid stage of development are more vulnerable to the effects of cerebral injury than are well consolidated skills.

Table of Contents

Table of Contents (contd.)

List of Tables

List of Figures

Neuropsychological Functions Following Closed Head Injury in Infants and Preschoolers

The assertion that recovery of cognitive functions following brain injury is better in children than adults has been questioned (St. James-Roberts, 1979). Simplistic statements describing the relationship between age at the time of cerebral insult and the severity of cognitive sequelae are not tenable (Levin, Benton, & Grossman, 1982; Fletcher, Ewing-Cobbs, McLaughlin, & Levin, 1985). The greater plasticity of the developing brain has been credited with enhanced recovery in children. Rather, outcome is determined by a variety of interacting variables. Differences in outcome may be related to the type of cerebral insult; some age-related differences have been obtained for focal as opposed to diffuse brain injuries (Levin, Ewing-Cobbs, & Benton, 1984). Moreover, the types of insults producing brain injury in children and adults differ: while adults often sustain focal injury due to cerebrovascular infarcts or tumors, children are more likely to sustain diffuse encephalopathies. Closed head injury (CHI), which often produces diffuse or multifocal brain injury, occurs frequently in all age groups. Comparisons of cognitive

outcome following CHI in school aged children and adolescents or adults suggest that minimal, if any, sparing is observed in children (Brink, Garrett, Hale, et al., 1970; Levin et al., 1982). Few studies have examined cognition in head injured children under the age of six; at the present time, it is unclear how very young children are affected by CHI.

The purpose of the present study is to examine the characteristics and severity of subacute intellectual, linguistic, and motoric dysfunction in infants and preschoolers sustaining a closed head injury. In addition, the amount of recovery evidenced in these skills at least six months following the injury will be addressed. Issues pertaining to the plasticity of the developing brain will be reviewed. Studies of posttraumatic intellectual, linguistic, and motoric performance in children will be presented.

Age, Plasticity, and Equipotentiality

The traditional view that the immature brain is more "plastic" than the mature brain has been challenged (St. James-Roberts, 1979). The traditional view proposes that the developing CNS is less vulnerable to disruption and exhibits more functional recovery than the mature CNS (Bastian, 1898; Smith, 1981). This view is inconsistent with findings in the developmental pathology literature. In general, the effects of disturbance to a growing organ are likely to be more severe and

persistent than to a mature organ (Robinson, 1981). As noted by Fletcher, Levin, and Landry (1984), the preferential recovery of the immature brain is usually explained by reference to three variables: age, plasticity, and equipotentiality. Despite the wide reliance on these variables in the interpretation of behavioral studies, none of them have provided a coherent basis for explaining supposed age-based differences in recovery (Fletcher et al., 1984). Although age has often been used to explain different recovery characteristics, it does not have direct explanatory significance (Fletcher & Satz, 1983). Age is a temporal marker; it is neither a necessary nor sufficient explanation for development (Siegel, Bisanz, & Bisanz, 1983). Focusing on age at injury has de-emphasized a number of critically important interactive variables. The functional maturation of the involved substrate, age at assessment, testing procedures, and environmental factors converge to significantly influence outcome measures (St. James-Roberts, 1979; Teuber & Rudel, 1962).

The term "plasticity" continues to be widely employed without adequate definition. In this review, plasticity refers to the supposedly greater behavioral recovery in children following a central nervous sustem insult. The mechanism(s) underlying the greater recovery in children have not been adequately specified. Vicarious functioning, the ability of one brain area to assume the function of another damaged area, has been the mechanism most commonly

proposed to explain preferential recovery in children (St. James-Roberts, 1979). Since the immature brain is presumed to be less functionally committed, the probability of vicarious function is enhanced (St. James-Roberts, 1979). However, little evidence has been presented that supports any age related differences; both the mechanisms involved and the supporting behavioral data remain equivocal (St. James-Roberts, 1979).

Within the context of plasticity arguments, equipotentiality refers to the notion that both cerebral hemispheres may provide equally good substrates for language development (Dennis & Whitaker, 1976). Thus, early brain injury would be expected to produce less severe residual deficits. As predicted by vicarious function theory, either hemisphere could compensate for damage to the other. However, results of anatomical, behavioral, and electrophysiological studies support a model of brain development that is invariant rather than equipotential (Kinsbourne, 1975). Additionally, Dennis and associates have demonstrated a variety of lateralized information processing deficits in infantile hemiplegics following hemidecortiction (e.g., Dennis & Lovett, 1981; Dennis & Whitaker, 1976; Kohn & Dennis, 1974).

After reviewing studies of recovery from localized cerebral trauma in children, St. James-Roberts (1979) concluded that age at insult was not a causal factor in recovery. He was unable to support either

corollary of the traditional view, i.e., that brain injury was less severe or persistent in children. Consistent with these findings, examination of the effects of diffuse insult to the developing brain did not support traditional plasticity hypotheses (Levin et al., 1984). Evidence is accumulating indicating that children may exhibit more severe cognitive sequelae following diffuse brain insult than either adolescents or adults. The view that children exhibit less severe sequelae following brain injury has been overstated (St. James-Roberts, 1979). Much of the data that have been interpreted to support the traditional plasticity view are derived from studies of children with acquired aphasia (e.g., Basser, 1962; Lenneberg, 1967). Since much of the controversy surrounding plasticity constructs is related to recovery from early brain injury, studies of recovery from cerebral insult in animals and man are discussed in the following sections.

Early Brain Injury

At the present time, neither the "early plasticity" nor the "early vulnerability" viewpoints can account for the contradictory findings regarding the effect of early brain injury on subsequent cognitive development (Taylor, 1984). In addition to age at the time of cerebral insult, other equally important variables that affect the quality of recovery deserve consideration. The functional maturation of the involved tissue, age at the time of assessment, testing procedures,

and environmental factors interact with age at injury (Finger & Stein, 1982; St. James-Roberts, 1979). To examine this issue, selected studies of cerebral insult in animals and man are reviewed with an emphasis on the effect of lesions incurred early in life (for a more comprehensive review, refer to Almli & Finger, 1984; Finger & Almli, 1984; Finger & Stein, 1982). As will be seen, contradictory findings have emerged in studies assessing recovery from brain injury across species.

Animal Studies

Behavioral deficits. In the 1930's and 1940's, Kennard (1938, 1942) examined the effects of perinatal ablation of the motor cortex in monkeys. Initial observations indicated that the motor impairments exhibited by animals with early lesions were much less severe than those produced by comparable lesions in mature animals. These findings, in addition to observations of sparing of function in neonatal monkeys and kittens subjected to somatosensory (Benjamin & Thompson, 1959), visual (Tsang, 1937), or prefrontal (Tucker & Kling, 1967) cortical lesions, led to the generalization that brain injury sustained early in life was associated with significantly fewer behavioral deficits. This generalization was dubbed the "Kennard principle." Although pioneering animal lesion experiments were uniformly enthusiastic regarding the extent of recovery from focal lesions in immature animals, in recent years many investigators have

not replicated such extensive sparing of function. The increasing use of appropriate methodological controls, anatomical tracing techniques, and more sophisticated testing paradigms has resulted in substantial qualifications of the "early sparing" hypothesis.

The complexity of perinatal lesion effects is reflected by the contradictory findings in many studies examining behavioral capabilities following early and late lesions. Due to the complexity of the factors influencing outcome, sparing of function may be observed under one set of conditions and not another (Finger & Stein, 1982). Four general perspectives have emerged from recent studies of recovery from early brain injury: 1) early sparing, 2) no sparing, 3) early vulnerability, and 4) developmental change. Examples of each position are provided. Significant sparing of visual discrimination skills has been reported following perinatal ablation of the striate cortex in a variety of species (Spear, 1984). However, the sparing of visual functions is not complete. Although visual acuity and monocular functions appear to be spared after neonatal lesions in kittens, binocular vision is compromised irrespective of age at the time of lesioning (Spear, 1984). Villablanca, Burgess, and Sonnier (1984) concluded that neonatally hemispherectomized kittens exhibited greater sparing than adults on the vast majority of sensory, motor, and neurobehavioral tests. In addition, the sparing persisted into adulthood. The authors inferred that their findings unambiguously

supported the Kennard principle.

Other studies have not identified sparing of skills in animals lesioned in infancy as compared to adulthood. For example, Whishaw and Kolb (1984) administered a comprehensive battery of tests to rats hemidecorticated either in infancy or adulthood. They concluded that behavioral impairments observed during development of rats decorticated during infancy were also observed in adult preparations. Both infant and adult rats were impaired on measures of limb placement and complex locomotion. Simons and Finger (1983) observed that large lesions of the sensorimotor cortex severely impaired tactile learning in rats lesioned in infancy or adulthood. Similarly, the majority of studies of subcortical ablation do not indicate sparing of function in infant operates (Johnson & Almli, 1978). Few studies support the early vulnerability hypothesis. However, Gramsbergen and IJkema-Paasen (1984) identified more locomotor impairment in infant as compared to adult rats subjected to hemicerebellectomy.

The developmental change hypothesis seems to have garnered the most support. As suggested by Nonneman, Corwin, Sahley, and Vicedomini (1984), early brain injury can be either more or less debilitating than comparable injury sustained later in life. The effects may depend upon the maturational status of the region ablated and of

other anatomically and functionally related tissue. Longitudinal examination of animals receiving cortical ablations early in life suggests that the effects produced by lesions are modified as the animal matures. Some investigators have reported that initial sparing following early lesions may be significantly, if not totally, reduced following long postoperative recovery periods (Almli, Golden, & McMullan, 1976; Goldman, 1974; Hicks & D'Amato, 1975). In contrast, other lesion experiments suggest that some deficits observed in the early phases of recovery are diminished as the animal matures (Finger & Stein, 1982).

In an elegant series of frontal lobe ablation studies in rhesus monkeys, Goldman (1974) demonstrated that the type of deficits observed may vary depending on both age at the time of lesioning and age at the time of behavioral assessment. Goldman proposed that a variable rate of development exists among the prefrontal functional system that subserves performance on delayed alternation tasks. Subcortical areas (head of the caudate nucleus and the dorsomedial nucleus of the thalamus) mature the earliest, followed by orbitofrontal and then dorsolateral cortex. Damage to any component of this system in adult animals produces both immediate and long term impairment on spatial reversal and delayed response tasks. In contrast, the effect of perinatal lesions depends upon the specific component damaged. In monkeys, perinatal lesions of subcortical

components are associated with both immediate and long term behavioral deficits. Early lesions of the orbitofrontal cortex produce immediate behavioral deficits. However, by approximately 2 1/2 years, performance is normalized as the dorsolateral cortex appears to mediate delayed response tasks. The effects of early orbitofrontal lesions are consistent with recovery of function. In contrast, early dorsolateral lesions yield a picture of initial sparing of function. As the animals mature, delayed deficits are apparent since the dorsolateral tissue, which mediates performance in adults, is not available. Since this substrate was unavailable for the infant operates, delayed deficits in their behavior appeared when in the normal course of development the delayed alternation task is mediated by the dorsolateral cortex. Goldman stated:

> "functions will appear preserved when the neural substrate is removed in infancy and animals are evaluated at prefunctional stages for the development of the substrate. Indeed, functions will appear to have been compensated when, in fact, they had not been lost" (p. 168).

Alexander and Goldman (1978) used reversible cryogenic depression to temporarily inactivate dorsolateral tissue in normally developing animals. While the performance of juvenile monkeys was

not affected by this procedure, adult animals displayed a temporary disruption of delayed alternation performance. This suggests that the dorsolateral cortex was not contributing to the performance of the juvenile monkeys and is consistent with the developmental change position.

Nonneman et al. (1984) compared lesion effects in the prefrontal system in monkeys and rats. These species vary in the degree of cerebral maturation present at birth. The brain of the neonatal monkey is more mature than that of the rat. The long term effects of both subcortical and cortical lesions during infancy in the monkey are more debilitating than comparable lesions in the rat. In adult animals of both species, lesions produce serious immediate and long term deficits. Interestingly, Goldman and Galkin (1978) performed bilateral dorsolateral prefrontal cortical ablations in fetal rhesus monkeys. One monkey was spared to evaluate resultant behavioral capabilities on tasks regarded as indicants of frontal lobe dysfunction. The monkey displayed a dramatic sparing of function on delayed spatial response, visual pattern discrimination, spatial delayed alternation, and object discrimination reversal tasks when tested from one until 2 1/2 years of age. Nonneman et al. (1984) inferred that prenatal ablation in the monkey two months before birth produces the same degree of behavioral sparing as neonatal removal of the medial

prefrontal cortex in the rat.

Anomalous CNS growth. Damage to the CNS in infants and adults does not always result in comparable anatomical sequelae. While organisms are very young, the type and degree of effects observed in response to injury may be greater than those observed later in life. A significant reduction in brain weight indicative of local and remote cell loss typically accompanies perinatal brain injury; lesions in adults yield more localized effects (Kerr, 1975; Isaacson, 1975; Rosenzweig, Bennett, & Alberti, 1984). Neuronal remodeling occurs following central nervous system lesions in both young and adult animals. While collateral sprouting may occur in adults in restricted regions, remodeling may be much more extensive in younger animals (Cotman & Nieto-Sampedro, 1982; Gramsbergen and IJkema-Paasen, 1984). The most common anatomical alteration following perinatal ablation is the growth of anomalous fiber tracts as axons sprout into denervated regions (Gramsbergen & IJlema-Paasen, 1984; Hicks & D'Amato, 1970; Schneider & Jhavari, 1974; Spear, 1984; Villablanca et al., 1984). It is possible that the functional outcome of anomalous projections varies as a function of the maturity of the neural substrate at the time of injury. Even though sparing of function is sometimes achieved through these rerouted pathways, the resulting behaviors may be maladaptive.

In several studies, sparing of function was presumably related to

the growth of anomalous projections. Villablanca et al. (1984) identified greater sparing in neonatally hemispherectomized kittens than adults on a variety of tasks. Additionally, the sparing of function persisted into adulthood. The authors documented extensive neuronal remodeling that consisted of the presence of axon terminals in brainstem areas. These projections are not observed in animals hemispherectomized during adulthood. Hicks and D'Amato (1970) reported few behavioral differences in rats hemispherectomized early as compared to later in life. In addition, they observed that only the infant rats developed a small, uncrossed corticospinal tract after hemispherectomy.

Spear (1984) identified greater sparing or recovery following neonatal as compared to adult visual cortex ablations on visual discrimination tasks. Neonatal lesions produced anomalous projections from the retinal ganglion cells to the thalamus as well as enhanced projections from the thalamus to the posteromediolateral suprasylvian cortex. These connections may provide the cortical neurons with responsiveness to both the ipsilateral and the contralateral eye.

Behavioral abnormalities have been correlated with the presence of anomalous fiber tracts. So and Schneider (1976) removed the superficial layers of the right superior colliculus in neonatal

hamsters. Immature axons originating in the retinal ganglion cells of the opposite eye crossed the midline and terminated in the remaining deeper layers. However, the fibers also recrossed the midline and innervated the wrong (ipsilateral) superior colliculus. When food was presented in the left visual field, the hamsters turned away from the food rather than toward it. Similarly, Gramsbergen and IJlema-Paasen (1984) reported that hemicerebellectomy performed in infant rats produced more locomotor impairment than comparable ablation in mature animals. Early ablation was associated with extensive neuronal remodeling. Aberrantly projecting cerebellar cerebrorubral fibers projecting onto the ipsilateral side were identified through autoradiographic tracing of radioactively labeled isotopes. Moreover, neurophysiological examination of the rerouted fiber tracts revealed functional activity related to locomotor behavior.

Summary. In recent years, the generality of the "Kennard principle" has been questioned. Behavioral outcome following brain injury is dependent on a variety of factors that interact with age at injury. The effects of perinatal lesions are complex; recent investigations have yielded inconsistent findings regarding sparing of function following early lesions. While some studies are consistent with the "early plasticity" hypothesis, others support the "early vulnerability"

hypothesis. Subcortical lesions are usually associated with comparable deficits in neonates and adults during both acute and long term phases of recovery. Cortical lesions produce a wider variety of outcomes; sparing or recovery of function is more likely following cortical ablation. However, recovery is not invariant. This dissociation may be related to the functional maturation of the ablated tissue and related neuronal systems. Subcortical regions appear to mature prior to functionally related cortical tissue. The more diffusely organized cortex may be better able to partially compensate for the function of damaged tissue, particularly if tissue fragments are spared. Sparing of function appears to be most likely if the lesion is incurred prenatally or perinatally. Anomalous neuronal growth is more common following early ablation. However, depending on the accuracy of the termination sites of rerouted fiber tracts, either beneficial or maladaptive behaviors may be produced.

Early Brain Injury in Man

Comparison of outcome following brain injury in children and adults is complicated by several factors. The etiologies producing brain damage vary with age. Focal lesions produced by cerebrovascular disease and cortical tumors occur infrequently in children. However, diffuse cerebral insult produced by infectious disease is more common in children. Even when children and adults are affected by the same etiology, the child's brain may respond differently to the insult (e.g., Bruce et al., 1979). In contrast to animal experiments that may control for the locus and extent of lesion, prelesion training, and the postlesion interval before retesting, clinical investigators can rarely control variables other than the injury-test interval. When the long-term effects of brain injury in children and adults are compared, the chronicity of the brain injury is often greater than when brain injury occurs in adulthood. Despite these methodological shortcomings, studies of recovery from brain injury in man converge with findings from the animal lesion literature in disputing a simplistic view of cerebral plasticity (Levin et al., 1984). Early studies of cognitive outcome following brain injury sustained in childhood supported the "early plasticity" hypothesis. The major source of data was provided by studies examining acquired aphasia in children that identified rapid and often complete recovery of language functions

(Alajouanine & L'hermitte, 1965; Basser, 1962; Lenneberg, 1967). In addition, Rudel, Teuber, and Twitchell (1974) noted the frequent absence of focal somatosensory deficts following early brain injury. Case studies describing above average intellectual performance in individuals sustaining severe early cerebral insults provided further support for the early plasticity hypothesis (e.g., Smith & Sugar, 1975). To further examine the early plasticity hypothesis, studies examining cognitive outcome in children subsequent to localized and diffuse brain injuries are reviewed.

Localized Cerebral Insult

Age at the time of injury may influence the speed of cognitive recovery, the pattern of cognitive deficits, and the eventual degree of intellectual impairment (Rutter, Chadwick, & Shaffer, 1983). While the effect of brain injury appears to be similar in school-aged children, greater age-based differences may be present in children sustaining injuries during infancy. As noted by Rutter and colleagues (1983), the effects of brain injury in children will likely be different than in adults. Age-based differences in intellectual functioning following focal cerebral insult have been documented. To illustrate, discrepancies in performance on the Weschler intelligence scales appear to differ according to age. Localized cerebral insult in adults is often associated with a discrepancy between verbal and performance

abilities. Injury to the dominant hemisphere commonly reduces performance on language based tasks while exerting a minimal effect upon nonverbal skills. Damage localized to the nondominant hemisphere is associated with deficits in nonverbal functions; linguistic skills are typically not affected. In contrast, the effects of locus and laterality of cerebral lesions in children appear to be less striking and less consistent than in adults (Rutter, 1981). This has been reported for children with unilateral penetrating head injuries with gross damage to the underlying cortex (Chadwick, Rutter, Thompson, & Shaffer, 1981), hemispherectomized infantile hemiplegics (Levin et al., 1984), children with temporal lobe tumors (Mulhern, Crisco, & Kun, 1983), and children with acquired aphasia (Hécaen, 1976). However, lateralized cognitive deficits in children with temporal lobe seizure disorders have been reported (Fedio & Mirsky, 1969).

Intellectual functions. The effects of lateralized brain injury on cognitive functions vary with the age at injury. Woods (1980) examined the effect of laterality of unilateral hemispheric insult on Weschler IQ scores in 50 patients with a unilateral hemiplegia. Early cases exhibited a hemiplegia prior to their first birthday; the onset of hemiplegia in the late lesion group ranged from one to 15 years. Long term intellectual evaluations were conducted from two to 25 years post

onset. Patients with early lesions of either hemisphere had significant reductions in both Verbal (VIQ) and Performance (PIQ) intelligence quotients. Late left hemisphere lesions were associated with lowered Verbal and Performance IQ scores while late right hemisphere lesions reduced only the PIQ. Annett (1973) reported that there was no evidence of selective impairment in either the VIQ or the PIQ according to the side of hemiplegia in 106 infantile hemiplegics evaluated at ages five to 18.

Significant intellectual deficits are common following hemidecortication for infantile hemiplegia. The mean intelligence quotients reported in major studies are 60 (McFie, 1961), 64.4 (Carlson, Netley, Hendrick, & Pritchard, 1968), and 69.5 (Strauss & Verity, 1983). Differences in performance on verbal versus nonverbal tasks are difficult to compare since the status of the remaining hemisphere may be compromised to varying degrees. In a review of published studies of intellectual functioning subsequent to hemispherectomy for intractible seizures, Levin and associates (1984) inferred that the VIQ and PIQ were comparably depressed in patients with the onset of pathology during infancy or childhood. Scores were depressed irrespective of the side of the hemispherectomy. The authors suggested that the pattern of scores was similar to the nonspecific intellectual deficit that persists after the onset of infantile

hemiplegia (Woods, 1980). In contrast to the above findings, hemispherectomy in adults is associated with lateralized performance deficits. Adult right hemispherectomy patients displayed marked visual spatial deficits with intact verbal abilities. The severity of aphasia following adult left hemispherectomy has precluded administration of the verbal scale; visual spatial skills were intact.

Acquired aphasia in children. Acquired aphasia refers to language impairment that occurs after language has developed normally. Much of the evidence for the early plasticity position has been derived from studies of childhood aphasia. According to Bullard, Satz, and Speedie (1980), two theoretical positions regarding the cortical organization for speech have been articulated: 1) the developmental maturation hypothesis (Lenneberg, 1976), and 2) the developmental invariance hypothesis (Kinsbourne, 1975).

Lenneberg (1967) inferred that equipotentiality exists until the onset of speech. At that time, lateralization of function progresses as one hemisphere assumes language functions. Injury during infancy does not impair language development since the left and right hemispheres provide equally good substrates for language development. Dominant hemispheric insult at successively later ages progressively results in symptoms that resemble adult aphasias. According to Lenneberg, by mid-adolescence the adult pattern of

symptoms and recovery characteristics is established.

In contrast, Kinsbourne (1975) inferred that the anatomical substrate for speech is asymmetric at birth, e.g., that the planum temporale is larger and has a more extensive vascular system in the left hemisphere language areas. Based on electrophysiological, neuroanatomical, and behavioral studies, Kinsbourne (1975) suggested that the anatomical asymmetry paralleled a functional asymmetry that was consistent with left hemisphere specialization for speech irrespective of age. In general, the concept of equipotentiality is inconsistent with evidence from studies of infantile hemidecorticates (e.g., Dennis, 1980) that indicate the left hemisphere is prepotent for language development (Fletcher et al., 1984).

Language after hemispherectomy. Recent reviewers (e.g., St. James-Roberts) of the literature on infantile hemiplegia and early hemidecortication have stressed the methodological shortcomings that detract from most of the early outcome studies. These problems include the frequent finding of mental retardation that may adversely affect language acquisition, a lack of quantitative assessment of language, and inadequate statistical analysis. However, there is agreement that infantile left hemispherectomy may be compatible with substantial language development. It appears that the intact right hemisphere is capable of supporting language development during

infancy and early childhood and may even assume language more adequately after removal of an epileptogenic left hemisphere. Linguistic deficts appear to be more severe and persistent as age at onset increases. Dennis and associates (Dennis, 1980; Dennis & Kohn, 1975) have identified significant linguistic competence in a selected group of hemidecorticates who were functioning above the borderline range of intelligence. Despite the impressive language acquisition observed in the left hemidecorticates, examination of later developing linguistic skills indicated that these children were clearly inferior to right hemidecorticates on a variety of syntactic and semantic tasks. Other studies examining linguistic skills in children suggest that significant deficits are present. Day and Ulatowska (1979) reported normal expressive language in an infantile hemiplegic hemispherectomized at four years of age. Despite normal speech functions, the linguistic content was impoverished and deficits were aparent in grammar and in the comprehension of verb tenses, comparatives, and irregular plurals. Left hemispherectomy in a 10 year old produced long term expressive deficits characterized by limited spontaneous speech and significantly impaired academic performance (Gott, 1973). Examination of left hemispherectomy in adults is limited due to the short postoperative recovery periods due to tumor regrowth. Burklund and Smith (1977) and Smith (1974)

described two adults; receptive language skills were least affected and showed the greatest improvement over time. Expressive skills were severely impaired although one case was eventually able to speak in short propositional sentences. Production and comprehension of written language remained severely compromised. It appears that left hemispherectomy in infants and young children is compatible with substantial language development. However, the degree of linguistic competence is extremely variable. Such extensive recovery of language skills has not been documented in adults. The short duration of postoperative survival prevents determination of the eventual degree to which the mature right hemisphere may mediate language.

Characteristics of acquired aphasia in children. Acquired aphasia in children is thought to differ from the adult aphasias along three dimensions: 1) frequency, 2) symptoms, and 3) prognosis (Satz & Bullard-Bates, 1981). Historically, acquired aphasia in children following dominant hemispheric insult is reportedly rare when compared with adults (Cotard, 1868; Denckla, 1979). However, in a critical review of this topic, Satz and Bullard-Bates (1981) concluded that acquired aphasia is not rare in children if the lesion is unilateral and involves the language areas. After infancy, the risk of aphasia is comparable in right handed children and adults sustaining a left hemisphere injury.

Acquired language disorders in children are associated with different symptoms and recovery characteristics than are seen in adult aphasics. As noted by Dennis (1980), the usual frame of reference for describing language performance in childhood aphasics is the adult aphasia classification system. She argued that a more appropriate frame of reference would be the child's language system prior to the trauma. Dennis (1980) suggested that the discrepancy between the commonly identified expressive aphasia in children as compared to the classical adult aphasic syndromes may be partly due to the mixed etiologies in the child studies. The major studies in this area examined children with vascular insult, trauma, infection, and neoplasms (e.g., Alajouanine & L'hermitte, 1965; Guttman, 1942; Hécaen, 1976; Woods & Teuber, 1978). The importance of etiology in childhood aphasia was stressed by Guttman (1942) and van Dongen and Loonen (1977). Children with traumatic injuries usually exhibit expressive deficits and have a generally good prognosis while vascular lesions produce more varied and persistent symptoms. Since trauma cases are overrepresented in many studies, it is difficult to dissociate the contribution of age from that of etiology in the pattern of symptoms (Ewing-Cobbs, Fletcher, Landry, & Levin, in press). The few studies of aphasia secondary to vascular disease in children show striking parallels with adult symptomatology (Aram, Rose, Rekate, & Whitaker, 1983; Dennis,

1980).

Acquired aphasia in children is associated with a reduction in expressive language. This nonfluent aphasia was often initially characterized by mutism or decreased initiation of speech (Hécaen, 1976). Syntax was simplified rather than erroneous; no child exhibited agrammatism similar to that described in adults (Alajouanine & L'hermitte, 1965). In contrast to adults, phonemic and semantic paraphasias, as well as receptive language impairment, occurred infrequently. However, articulatory disturbances, naming disorders, dyscalculia, and dysgraphia were frequent and persistent (Alajouanine & L'hermitte, 1965; Guttman, 1942; Hécaen, 1976). Some differences in symptoms attributable to age have emerged. Children under the age of 10 exhibited a reduction of spontaneous speech and articulation disturbances irrespective of the lesion site (Alajouanine & L'hermitte, 1965; Guttman, 1942). In older children, articulation disturbances were less common and paraphasias occurred more frequently. The incidence of reading comprehension and writing disorders was reduced (Alajouanine & L'hermitte, 1965; Guttman, 1942). Guttman (1942) stated that children older than 10 displayed either some reduction in expressive language or "the picture generally seen in the adult" (p. 209).

The prognosis for recovery from acquired aphasia is regarded as

more favorable in children than in adults (Hécaen, 1976). Estimates of recovery range from 50% (van Dongen & Loonen, 1977) to 75% (Alajouanine & L'hermitte, 1965) to nearly 100% (Basser, 1962). Despite the relatively favorable prognosis for recovery of speech, persistent deficits in writing, naming, reading comprehension, and arithmetic functions have been documented (Alajouanine & L'hermitte, 1965; Hécaen, 1976). Consistent with these findings, none of the children performed at age level scholastically (Alajouanine & L'hermitte, 1965).

The effect of age on the recovery of language skills remains unclear. Woods and Teuber (1978) concluded that recovery from aphasia is more likely following lesions sustained prior to age eight. However, the time course of recovery was variable in children aged 1 to 15 years at insult. Woods and Carey (1979) inferred that the extent of recovery from aphasia was inversely related to the age at onset. The influence of age on the rate of recovery is unclear. Alajouanine and L'hermitte (1965) reported no age effects in the speed of recovery. In contrast, Aram et al. (1983) inferred that linguistic recovery subsequent to basal ganglia lesions may be more rapid and complete in children than in adults.

Summary. Localized brain injury in children is often associated with more generalized cognitive deficits than are observed in adults. However, specific cognitive functions may be spared or may exhibit dramatic recovery. Brain damage incurred during the first year of life

appears to yield the greatest generalized reduction in cognitive capacity. This finding is consistent with the reports in the animal literature describing more generalized cerebral consequences following early ablation. Significant recovery of language functions has been reported in children with acquired aphasia. However, many studies included children with posttraumatic aphasia, which recovers to a greater extent than aphasia produced by other etiologies. Many studies report a high percentage (25-50%) of persistent deficits one year post onset. Although specific symptoms may resolve, cognitive impairment and scholastic difficulties remain. Aphasia in children tends to be nonfluent while symptoms in adolescents and adults are more variable. Since many studies examined samples composed of mixed etiologies, it is unclear to what extent the differential symptoms reflect age as opposed to etiology. The linguistic deficits in children are not invariant. Rather, expressive disorders are commonly associated with deficiencies in writing, naming, and arithmetic skills. The nature of linguistic impairment varies with age. Children under 10 years of age at insult exhibited more expressive disorders. Older children and adolescents were more likely to exhibit paraphasias as well as a mixture of receptive and expressive deficits. Although language functions appear to recover significantly following localized brain injury, similar recovery has not been reported for other skill areas, such as

visuospatial, memory, or motor skills.

Generalized Cerebral Insult

In comparison to the inconsistent relationship between localized cerebral involvement and behavioral recovery in children and adults, recent studies examining outcome in children following diffuse cerebral insult consistently report more severe cognitive deficits in children. O'Leary and Boll (1984) stated that the effects of generalized brain damage on intelligence test scores differed significantly in children and adults. Accoring to Boll (1978), serious brain damage in adults may have a minimal effect on IQ scores; vocabulary skills are relatively resistant to disruption. O'Leary and Boll (1984) suggested that intellectual impairment was very common following cerebral insult in children; moreover, vocabulary was particularly likely to be impaired. Similarly, Rutter (1981) inferred that bilateral widespread brain damage in children was often associated with significant intellectual deficits. He inferred that pediatric brain injury may primarily influence general cognitive abilities (Rutter, 1982). Boll and Barth (1981) reviewed studies of early generalized brain damage in children and concluded that there was a higher incidence of discrepancies between the VIQ and PIQ than in adults. The VIQ tended to be higher than the PIQ in the majority of children. Taylor (1984) suggested that vocabulary and other verbal

skills may be as impaired as nonverbal skills in children sustaining early CNS disorders. Although no unitary pattern of deficits has been identified, several studies suggest that the PIQ may be more affected than the VIQ by disorders producing diffuse cerebral involvement (e.g., Boll & Barth, 1981; Chadwick, Rutter, Shaffer, & Shrout, 1981; Dennis, Fitz, Netley, et al., 1981; Hammock, Milhorat, & Baron, 1976; Taylor et al., in press). These reports of relatively global consequences of early generalized brain injury are consistent with Hebb's (1942) hypothesis that early brain injury may preferentially disrupt new learning, abstraction, and problem solving abilities. Since young children have acquired less information and fewer skills, brain injury may result in a reduction of cognitive capacity that eventually constrains the level of cognitive development. In contrast, previously learned skills appear to be relatively insensitive to brain damage. For example, skills such as vocabulary knowledge and verbal comprehension are relatively insensitive to brain injury in adults but may be significantly affected in children (Taylor, 1984).

Generalized brain injury produced by infectious diseases, seizure disorders, and cranial irradiation appears to be associated with a worse prognosis in children than adults. Infectious diseases, such as encephalitis, meningitis, and Reye's syndrome, are associated with more severe residual cognitive impairment when contracted during

infancy as opposed to later childhood (Davidson, Willoughby, O'Tuama, Swisher, & Benjamins, 1978; Taylor, Michaels, Mazur, Bauer, & Liden, in press; Wright, 1978). Neurological and intellectual sequelae occur frequently when meningitis is contracted during the first year of life (Wright, 1978). Similarly, Davidson and colleagues (1978) identified age effects in recovery from Reye's syndrome. All of the children with IQ's in the average to above average range were at least two years old at the time of onset.

Cranial irradiation and chemotherapy administered to children with acute lymphocytic leukemia may produce long term structural changes in the brain characterized by cerebral atrophy and calcification (Mulhern, Crisco, & Kun, 1983). Prophylactic CNS irradiation in children with acute lymphocytic leukemia is associated with significant cognitive deficits as compared to children receiving delayed irradiation or alternate treatments (Eiser, 1978; Meadows, Massary, Fergusson, et al., 1981). Due to methodological limitations of many studies (e.g., small samples, inadequate control groups, limited neuropsychological evaluation), the pattern of deficits associated with combined modality CNS treatment varies. Although CNS irradiation is associated with a general lowering of scores across a broad spectrum of neuropsychological functions, motor, visual motor, spatial memory, and arithmetic functions appear to be the most vulnerable

(Copeland, Fletcher, Pfefferbaum-Levine, et al., in press). Deficits of comparable severity have been identified approximately one year (Rowland, Glidewell, Sibley, et al., 1982) and eight years (Copeland et al., in press) following treatment. These findings indicate that the level of cognitive deficit may be stable over time; CNS irradiation may be associated with a defict as opposed to a lag in development. However, longitudinal evaluation is necessary to confirm this possibility (Copeland et al., in press). Studies of CNS irradiation suggest that the degree of cognitive impairment is most pronounced in children who were two to five years of age when cranial irradiation was administered in comparison to older children receiving the same treatment regimen (Copeland et al., in press; Meadows et al., 1981).

Examination of treatment effects in children with brain tumors indicates that children receiving cranial irradiation in addition to surgery exhibit a lower level of intellectual functioning than children receiving only surgical intervention (Hirsch, Renier, Czernichow, et al., 1979; Kun, Mulhern, & Crisco, 1983; Raimondi & Tomita, 1979). Studies examining the effect of age at diagnosis in children with heterogeneous tumor locations and treatments suggest that children less than four (Danoff, Cowchock, Marquette, et al., 1982) or six (Eiser, 1979) years of age were more debilitated in comparison with older children. In contrast, Kun and associates (1983) did not idenitfy

age effects in their sample. Mulhern, Crisco, and Kun (1983) inferred that the literature on pediatric brain tumor survivors was consistent with the following preliminary conclusions: 1) children with brain tumors exhibit a high incidence of intellectual impairment; 2) CNS irradiation was associated with alteration in neuropsychological function; and 3) a greater prevalence of neuropsychological deficits is apparent in young children. The deficits identified in children receiving cranial irradiation for brain tumors or leukemia/lymphoma are consistent with those obtained from studies of diffuse infectious insult that identified more serious sequelae in infants and very young children.

The deleterious effect of seizure disorders in young children has recently been identified. The age at onset of seizure disorders is a major determinant of the level of cognitive functioning (Levin et al., 1984). Unfortunately, in many studies it is difficult to dissociate the effects of age of onset from the duration of epilepsy, duration of anticonvulsant medication, and the number of seizures. Dikmen, Mathews, and Harley (1975) examined intellectual functioning in adult patients with motor seizures. Patients with later onset (10-25 years old) of convulsive disorder were functioning at a higher intellectual level than patients diagnosed prior to age six. Children with either generalized tonic-clonic or partial seizure disorders originating early

in life exhibited more severe neuropsychological deficits than older children (O'Leary, Lovel, Sackellares, et al., 1983; O'Leary, Seidenberg, Berent, & Boll, 1981). Chevrie and Aicardi (1978) demonstrated that afebrile seizures occurring during the first year of life were associated with a poor prognosis; over half of the patients exhibited severe mental retardation. The prognisis was poor in children with either status epilepticus, partial, or generalized seizures. The results of these studies indicate an inverse relationship between the age of onset of seizure disorders and the quality of long term cognitive functioning. However, the early onset of seizures may be indicative of more severe cerebral pathology.

Summary and Conclusions

Studies of focal and generalized brain insult in children do not clearly provide support for either the early plasticity or the early vulnerability hypotheses. Focal injury to either hemisphere during infancy appears to be associated with a generalized lowering of intelligence test scores on long term follow up. However, the immature brain appears to compensate considerably for linguistic deficits. The capacity of the immature CNS to compensate for other types of deficits, such as visual spatial or motor impairment, may be significantly more limited. Although resolution of specific linguistic deficits is striking in children, significant residual impairment is

present in academic achievement and general cognitive functions. Focal left hemisphere injury incurred after infancy also appears to produce more generalized impairment of verbal and nonverbal skills than is observed following left hemisphere injury in adulthood. Right hemisphere lesions sustained after infancy appear to primarily affect nonverbal functions. The deficits observed in children suggest that the effects of focal brain injury are different than in adults; the effects tend to be more generalized, particularly following damage during infancy or left hemisphere involvment during childhood. The age at onset of generalized brain injury appears to be inversely related to the quality of long term cognitive outcome. It appears that the immature brain is particularly susceptible to the effects of diffuse pathology.

Investigations of neuropsychological functions following early brain injury have not identified unitary patterns of dysfunction (Taylor, 1984). Different patterns of impairment may be present depending upon the location and extent of cerebral involvement, etiology, the degree of cerebral maturation at the time of insult, and the age at testing (St. James-Roberts, 1979). Unfortunately, most studies of cognitive outcome have been limited to cross-sectional assessment of intellectual functions. This type of study tends to yield "all or none" statements of recovery or deficit that are based on inadequate data (Taylor, 1984). To illustrate, Taylor and associates (in press)

demonstrated that children contracting meningitis during the first three years of life performed differently than sibling controls on a variety of measures. The meningitis group was impaired on measures of PIQ and Full Scale IQ. Deficits were also apparent on measures of fine motor coordination, gross motor and somatosensory skills, as well as verbal learning and memory. However, their performance did not differ relative to controls on measures of academic achievement. These findings are not consistent with all or none notions of sparing or deficit and emphasize the need to evaluate a broad range of skill areas.

Intelligence tests may not assess the same types of abilities in children and adults (Boll, 1978). Intellectual performance in adults appears to be more dependent upon the retrieval and appropriate application of previously acquired information. In contrast, assessment of intellectual ability in children may depend more heavily on abstraction and problem solving skills (O'Leary & Boll, 1984). Since brain injury commonly disrupts new learning, problem solving, and abstraction, children may be disadvantaged on measures of intellectual performance. Moreover, comparisons of the level of verbal and nonverbal skills based on discrepancies between the VIQ and the PIQ may be misleading. These scales evaluate different types of abilities. The VIQ is based largely on the retrieval of previously

learned information. The PIQ primarily assesses solving unfamiliar problems within a limited time frame and has a significant motor component (Fletcher, Ewing-Cobbs, McLaughlin, & Levin, in press). Interpretation of these scales based on comparisons of verbal versus nonverbal skills or left versus right hemispheric damage is simplistic. Furthermore, the abilities measured by individual intelligence scale subtests are dependent upon the combined functioning of many component skills. For example, performance on the block design subtest is dependent on visual perception, visual motor integration, speed of performance, analysis of the design into component parts, formation of a plan to reconstruct the design, carrying out the plan, and checking the results to determine if it matches the original. A deficit in any skill area may impair performance. Generalized deficits, such as difficulties focusing or sustaining attention, may also negatively impact performance. Clearly, analysis of neuropsychological performance on tasks assessing discrete behaviors is needed to isolate the sources of performance deficits.

In addition to examination of a broad range of neuropsychological skills, longitudinal evaluations are necessary to characterize the developmental course of recovery from brain injury. As indicated by Goldman (1974), the effects of early lesions may be altered as development progresses. While some skill areas may exhibit sparing

of function, others may be associated with a stable deficit or increasing impairment. Longitudinal evaluation would provide a means of evaluating developmental change in targeted skill areas. Skills in which persistent deficits were present could be contrasted with those showing sparing or normalization of function over time. In children, recovery of function needs to be dissociated from continued maturation. The use of longitudinal designs provides a means of characterizing outcome in different skill areas in terms of lag, delay, and deficit constructs that would indicate how a given cerebral insult impacts subsequent development (Satz, Fletcher, Clark, & Morris, 1981).

Closed Head Injury in Children

Closed head injury sustained by children differs from adult injuries along several dimensions. The epidemiology, mechanisms, and pathophysiology of closed head injury vary with age. These areas are reviewed in addition to studies of posttraumatic intellectual, academic, linguistic, and motoric performance following pediatric closed head injury.

Epidemiology

Epidemiologic studies of closed head injury in the United States yield only a rough approximaton of the actual incidence rate (Levin et al., 1982). The main sources of variation across studies are due to differences in reporting cases of mild head injury and mortalities (Levin et al., 1982). Incidence rates in the United States range from a low of 195/100,000 (Annegers, Grabow, Kurland, & Laws, 1980) when the criterion for inclusion was evidence of "pressured brain involvement" to a high of 600/100,000 (Caveness, 1977) when milder injuries were included. Studies of closed head injury in children conducted in England and Wales suggest that accidents comprise 16% of all hospital admissions for children under 15 years of age; more than one-third of all pediatric accident cases and 41% of pediatric mortalities were related to head injury (Craft, 1972; Field, 1976). In fact, the mortality associated with pediatric head injury is five times greater than leukemia, which is the second leading cause of death in

children (Annegers, 1983).

The cause of injury differs in children and adults. While approximately one-half of adult cases are involved in high speed motor vehicle accidents (Levin et al., 1982), falls are responsible for half of the pediatric injuries. Even though motor vehicle accidents account for nearly one third of pediatric truama cases, many of these are auto/pedestrian accidents occurring at low speeds (Levin et al., 1982). Jennett (1972) reported that the main categories of injury in children under the age of 15 were 1) domestic injuries (27%), 2) sport/leisure accidents (21%), 3) falls (16%), and 4) road accidents (9%).

Age and sex are related to the incidence of closed head injury. The ratio of injured mlaes to females ranged from 2:1 to 3:1 (Annegers et al., 1980; Craft, 1975; Field, 1976). The incidence of injuries in females was fairly constant from 0 through 20 years and then dropped markedly in the early 20's. Males displayed significant increases in incidence rates until the early 20's (Annegers et al., 1980).

Mechanisms and Pathophysiology

The primary mechanism producing brain injury following closed head injury is diffuse cerebral injury occurring at the time of impact (Adams, Mitchell, Graham, & Doyle, 1977; Strich, 1961). Holbourn (1943) developed an experimental model to examine mechanical forces associated with blunt cerebral trauma. He suggested that rotational

acceleration of the head produces shear strain, or the pulling apart of axons and disruption of cell bodies. After observing shear strains produced by subjecting gelatinous models of the brain to sudden rotation, Holbourn reported that shear strain was most pronounced in the region of the anterior temporal and orbital frontal lobes. Ommaya and Gennerelli (1974) inferred that rotational acceleraiton of the brain was necessary for loss of consciousness and production of diffuse and focal brain lesions. Based on animal experiments, they reported that the major damage was present on the surface of the brain and zones of changes in the density of cerebral tissue. In contrast, if the skull moves in a horizontal plane, more localized effects such as circumscribed contusions and intracerebral hematomas were visualized (Ommaya & Gennarelli, 1974). Autopsy studies of patients immediately rendered comatose without evidence of hypoxia or increased intracranial pressure indicated that diffuse brain damage can occur as a primary event. Moreover, persistent coma was attributed to diffuse white matter damage as opposed to specific focal lesions (Adams et al., 1977). Strich (1961) documented widespread degeneration of the white matter based on autopsy material. In addition, she indicated significant degeneration in fiber bundles (anterior commisure and the corpus callosum) as well as in ascending and descending brainstem tracts. In addition to diffuse shearing of neuronal fibers, secondary insults to the brain commonly occur

following severe CHI. Edema, diffuse cerebral swelling, and increased intracranial pressure may contribute to diffuse injury by producing hypoxic ischemia or contributing to an expanding mass effect (Levin et al., 1982).

It is unclear how the mechanics of injury differ in childhood as compared to adulthood. Strich (1961) suggested that the shearing strains produced by rotational acceleration were less pronounced in smaller brains. This could result in less microscopic neuronal injury in infants and young children in comparison to adults following comparable blunt trauma. In addition, it is unclear how the gelatinous consistency of the partially myelinated brain is affected by mechanical distortion (Mealey, 1968). Since children are more likely to be injured by falls or low speed accidents, many pediatric injuries may result in less severe rotational acceleration (Levin et al., 1982). Injuries sustained by adults in high speed motor vehicle accidents likely yield greater diffuse brain injury. For example, Jamison and Kaye (1974) observed that neurologic sequelae were present only in children injured in road traffic accidents.

Deformation or bending of the skull occurs to a greater degree in infants and young children (Mealey, 1968). The flexible, incompletely fused cranial plates and underdeveloped bony projections result in the skull absorbing more of the forces of impact (Mealey, 1968). Since less energy is directly transmitted to the brain, less focal injury may

be sustained (Craft, 1972). However, Gurdjian (1971) speculated that the comparatively shallow cerebral convolutions of the pediatric brain would promote greater deformation upon impact, enhance shearing effects, and increase the likelihood of brainstem injury.

Bruce and colleagues (1979) examined pathophysiology and outcome following severe closed head injury in 85 children and adolescents with Glasgow Coma Scale (GCS) scores of 8 or less. The most common CT scan findings were 1) subarachnoid hemorrhage and 2) general cerebral swelling. Based on regional cerebral blood flow studies, the general cerebral swelling was attributed to vascular congestion produced by increased blood volume and flow. In adults, general swelling is often associated with edema, which involves accumulation of excess water in the brain parenchyma. Cerebral blood flow may be decreased (Zimmerman, Bilaniuk, Bruce, et al., 1978).

In Bruce et al.'s (1979) sample, the outcome was very favorable in view of the severity of injury. Either a good recovery or only a moderate disability was reported in 87.5 percent; one-half of the sample exhibited pupillary abnormalities while nearly one third had bilaterally unreactive pupils. Given the high proportion of children with signs of brainstem dysfunction, these findings lend some support to Gurdjian's (1971) suggestion of increased shearing effects and brainstem involvement in children. According to Bruce et al. (1979), "it is possible that the threshold for coma is lower in children than in

adults and that the same input force may produce a worse neurological picture in the child than in the adult " (p. 188). Excluding the cases that were brain dead on admission, the overall mortality rate was 9 percent. Mortality rates in adults with comparable GCS scores have ranged from 30 to 50 percent (Becker, Miller, Ward, et al., 1977; Jennett et al., 1977).

Studies comparing outcome in children and adults must be interpreted cautiously. Many factors associated with CHI vary with age. The cause of injury, mechanisms of impact, and the pathophysiological response of the brain differ in children. The extent to which these variables influence outcome measures is unclear.

Intellectual and Academic Sequelae

Persistent intellectual and academic problems are encountered by children following severe head injury. Levin and Eisenberg (1979b) examined intellectual recovery at least six months post injury in 23 children and adolescents. Intelligence test scores below 85 (which fell below the preinjury level estimated from school records) were common in children rendered comatose for greater than 24 hours. Virtually all of the other children, who either had no loss of consciousness or were comatose for less than one day, recovered to an IQ of at least 85. Chadwick, Rutter, Shaffer, and Shrout (1981) evaluated intellectual recovery in 25 children following severe head injury that produced post traumatic amnesia for at least one week.

Compared to matched orthopedic controls, the head injured children were impaired on both the VIQ and the PIQ at four months post injury. By one year post injury, impairment was present only on the PIQ. These findings are consistent with patterns of intellectual recovery in adults that indicate a more rapid return of the VIQ than the PIQ to the normal range (Mandleberg & Brooks, 1975). As noted by Chadwick and associates, post traumatic deficits are most likely on tasks requiring rapid motor responses. Consistent with the above findings, Flach and Malmros (1972) reported an intellectual decline in 80 percent of their sample; the PIQ was most affected.

In general, most studies suggest that intellectual sequelae persist following severe closed head injury (Levin et al., 1982). Levin and Eisenberg (1979a) compared premorbid estimates of intellectual functioning obtained from academic records to WISC-R scores obtained at least six months post injury. Only a partial intellectual recovery was achieved by most children. Similarly, Richardson (1962) reported a 10 to 30 point decrement in IQ following severe injuries. Despite the significant intellectual sequelae commonly identified, prospective longitudinal evaluations assessing intellectual recovery at yearly intervals have shown progressive increments in IQ up to 5 years post injury following predominantly mild or moderately severe injuries (Black, Blumer, Wellner, & Walker, 1971; Klonoff, Low, & CLark, 1977).

Examination of the effect of age at injury on subsequent intellectual functioning has yielded inconsistent findings. Lange-Cosack, Wider, Schlesener, Grumme, and Kubicki (1979) evaluated long term outcome four to 14 years post injury in 50 children less than six years of age at injury. The late sequelae of head injury were most severe in infants and young children as compared to school aged children and adolescents. Brink, Garrett, Hale, Woo-Sam, and Nickel (1970) evaluated the quality of outcome in 52 children and adolescents one to 7 years following injuries producing coma persisting for at least one week. Even though coma duration was longer in children at least eight years of age at injury, the IQ scores were significantly higher than in children younger than eight at injury. In contrast to the above findings, other investigators have not identified interactions between age at injury and the severity of intellectual impairment (Chadwick et al., 1981; Gaidolfi & Vignolo, 1980; Klonoff et al., 1977; Levin & Eisenberg, 1979a).

Scholastic achievement is often significantly affected by moderate or severe head injury. Despite the importance of academic skills for the adaptive functioning of the child, few investigators have examined the type and severity of post traumatic academic difficulties. This is a serious omission since scholastic performance is a major developmental task facing children and adolescents.

Chadwick, Rutter, Thompson, and Shaffer (1980) examined

scholastic achievement in 97 children sustaining a unilateral compound depressed skull fractures with gross damage to the underlying cortex. Age at injury was not associated with substantial differences in IQ scores. By at least two years post injury, there was a tendency for cognitive scores to be more impaired if a left hemisphere injury was sustained before five years of age. However, the authors stressed that age at injury was not a major variable determining the extent of persistent cognitive impairment. Similarly, Shaffer, Bijur, Chadwick, and Rutter (1980) evaluated reading ability in 88 children with unilateral depressed skull fracture producing cortical lacerations. Fifty-five percent were reading one or more years below their chronological age; 33 percent performed at least two years below age level. Age at injury was not related to reading impairment. However, the prevalence of reading delays was significantly higher in children injured prior to 8 years of age who sustained longer periods of unconsciousness. While the duration of coma was significantly related to reading delays in the younger children, no association was observed between coma duration and reading difficulties in children at least eight years of age at injury.

Klonoff et al. (1977) indicated that 26 percent of children less than nine at injury had either failed a grade or been placed in resource classes. Twenty-one percent of the older children received special placements. These rates are striking in view of the generally mild

injuries in this sample. Other studies have confirmed that a high proportion of children require special classroom placement (Brink et al., 1970; Flach & Malmros, 1972; Fuld & Fisher, 1977; Heiskanen & Kaste, 1974; Richardson, 1963).

Language Functions

Levin and Eisenberg (1979a) examined post traumatic language dysfunction following closed head injury using the Neurosensory Center Comprehensive Examination for Aphasia (NCCEA) (Spreen & Benton, 1969). Sixty-four children and adolescents were evlauated within six months of injury. Linguistic deficits were observed in 31 percent. The most common deficits were dysnomia for objects presented visually (13%) or tactually to the left hand (12%). Auditory comprehension was impaired in 11 percent of the sample while verbal repetition was affected in only four percent. A higher incidence of linguistic disturbance was identified in head injured adults receiving similar evaluations. One-half of the adults exhibited dysnomia and/or decreased verbal fluency while one-third were impaired on measures of auditory comprehension (Levin & Eisenberg, 1979b; Levin, Kelly, & Grossman, 1976). Levin and Eisenberg (1979b) inferred that their findings were consistent with previous studies of acquired aphasia that suggest more rapid and complete recovery in children. These findings are compatible with studies indicating better linguistic recovery following head injury than other etiologies (Guttman, 1942; van Dongen

& Loonen, 1977).

Ewing-Cobbs, Fletcher, Landry, and Levin (in press) examined linguistic disturbance in 24 children (ages 5-10) and 33 adolescents (11-15) who sustained a nonmissile head injury. The severity of injury was grouped into mild and moderate/severe categories. The latter category included patients with positive computed axial tomographic (CT) scan findings, neurologic deficit, and/or coma persisting for at least 15 minutes. Mild injuries were unconscious for less than 15 minutes and had negative neurological and CT findings. Linguistic functions were evaluated during the subacute stage of recovery following resolution of post traumatic amnesia. The NCCEA was administered within five months of injury. To examine types of linguistic disturbance and reduce the number of dependent variables, the subtests were grouped into Naming, Expressive, Receptive, and Graphic categories based on the face validity of the subtest content. A composite score for each grouping was derived by averaging the age-corrected percentiles obtained on each of the component subtests. The scores of the children and adolescents were comparable on the Naming, Expressive, and Receptive groupings. The children were impaired relative to the adolescents on the Graphic composite score, which was composed of measures of written language. As compared to mild injuries, individuals sustaining moderate/severe injuries were impaired on the Naming and Graphic groupings. Linguistic deficit

occurred with the greatest frequency on measures of describing the function of objects (19%), repeating sentences (21%), word fluency (20%), writing to dictation (29%), copying sentences (19%), and comprehension of syntactically complex sentences (18%).

Ewing-Cobbs et al. (in press) found no evidence for sparing of linguistic function in children as compared to adolescents. Written language skills were significantly reduced in children regardless of the severity of injury. Examination of the component subtests indicated that children exhibited difficulty writing to dictation, but not copying sentences, when compared to adolescents. Similarly, Hécaen, Perenin, and Jeannerod (1984) reported that dysgraphia was more prevalent in children less than 10 years of age than in older children. Given that written language skills are more firmly established in adolescents than in children, these findings are consistent with the hypothesis that traumatic brain injury may disproportionately affect rapidly developing as opposed to automatized skills. The pattern of deficits obtained is consistent with previous studies of acquired langauge disturbances in children that identified primary difficulties with naming, verbal expression, and written language (Hécaen, 1976). In accord with previous studies of CHI in adults (Levin et al., 1976), the severity of diffuse brain injury, as opposed to the presence of a focal lesion, appears to be the overriding determinant of linguistic sequelae.

Recent findings converge in identifying subtle language processing deficiencies that persist following pediatric head injury. Chadwick et al. (1981) reported that object naming latency was impaired one year post injury. By two years post injury, performance was in the normal range. Similarly, Gaidolfi and Vignolo (1980) identified residual impairment of oral expression, which was characterized by a reduction in spontaneous speech, in four of 21 children evaluated approximately 10 years following severe head injury.

Motor Functions

Few studies have examined recovery of motor skills following pediatric CHI. Brink and colleagues (1970) examined restitution of motor functions in 46 children and adolescents who sustained very severe injuries. The median duration of coma was four weeks; all of the patients received intensive rehabilitation at a residential center. Motor functions were evaluated from one to seven years following the injury. Either unilateral or bilateral spasticity was present in 93 percent of the sample. Ataxia was evident in 62 percent. Nearly half of the patients had articulation deficits. The degree of physical recovery was not related to age. Brink, Imbus, and Woo-Sam (1980) expanded these findings in a prospective one year follow up study of 344 patients rendered comatose for at least 24 hours. They inferred that the majority of pediatric cases may achieve significant functional motor recovery after prolonged coma. Despite the marked resumption

of daily self care skills, only 10 percent of the sample had a normal neurological examination one year post injury. Patients with injuries due to nonaccidental trauma (e.g., child abuse) had a poorer outcome compared to those with accidental injuries. If such cases were excluded, children less than five years of age had a slightly better outcome than the total series.

Black, Blumer, Wellner, and Walker (1970) examined the recovery of neurological functions (e.g., motor or cranial nerve involvement) in 105 children with injuries of varying severity. They concluded that 34 percent of the patients presented with some neurological deficit. The rate of deficit decreased to 15 percent within three months and remained constant after that time. A large majority of the residual abnormalities were relatively minor.

Neuropsychological assessment of motor functions following pediatric CHI has revealed significant deficits in motor coordination. Based on a sample of children composed mostly of mild and moderate injuries, Klonoff et al. (1977) identified significant reductions in finger and foot tapping speed and slowed visuomotor performance one year post injury. Similarly, Chadwick et al. (1981) reported deficits in finger tapping and manual dexterity on baseline and one year follow up examinations. Levin and Eisenberg (1979a,b) observed that reaction time was frequently prolonged following CHI in adolescents whereas thumb-finger opposition was slow in head injured children.

Age at Injury

Brain injury may preferentially disrupt new learning (Hebb, 1942). Rutter (1981) inferred that young children may be disproportionately affected by cerebral insult since learning is necessary for the acquisition of new skills. Head injured children may be significantly at risk for cognitive delays since learning and memory are often impaired. In fact, difficulties learning and retaining new information are the most common cognitive sequelae following pediatric CHI (Levin & Eisenberg, 1979a).

The influence of age at injury on the cognitive skills of children and adolescents is unclear. No consistent pattern has emerged regarding the effect of age at injury upon cognitive outcome. This may reflect the restricted age ranges examined and the relatively few studies that have compared the neurobehavioral outcome of CHI in children of different ages (Ewing-Cobbs, Fletcher, & Levin, 1985). Several studies have reported that the age at injury was unrelated to either the rate of recovery or the severity of cognitive sequelae (Chadwick et al., 1981; Klonoff et al., 1977; Levin & Eisenberg, 1979a; Levin, Eisenberg, Wigg, & Kobayashi, 1983). With the exception of Klonoff and associates, these studies were restricted to children who were at least five years old at injury. In contrast, studies including infants and/or preschoolers have documented more severe long term cognitive deficits in younger children (Brink, et al., 1970; Chadwick, et al.,

1981; Lange-Cosack, Wider, Schlesner, Grumme, & Kubicki, 1979; Shaffer et al., 1980). However, the samples evaluated by Brink et al. (1970) and Lange-Cosack et al. (1979) appear to include young children with nonaccidental injuries. Cognitive outcome may be poorer in such children due to premorbid social factors and the cumulative neurologic effect of repeated blows to the head.

Rutter, Chadwick, and Shaffer (1983) tentatively concluded that the most striking age effects likely occur between lesions sustained during infancy and those incurred later in childhood. Age effects in school-aged children are likely to be minimal. The available data on pediatric CHI do not permit evaluation of the effect of age at injury on cognition. Based on clinical observation, Ewing-Cobbs and associates (1985) suggested that the types of deficits associated with severe CHI may vary somewhat with development. Preschoolers, in whom cognitive skills are rapidly developing, typically exhibit generalized cognitive impairment. Significant attentional, fine and gross motor, intellectual, linguistic, and visuospatial disturbances are common. These children may be at risk for the development of significant academic delays. The neuropsychological profiles of school aged children and adolescents are fairly similar; memory, visual spatial, visual motor, and attentional difficulties predominate. In addition, adolescents often exhibit marked difficulties with later-developing functions such as social judgment, planning, and strategy usage.

Additional research on age-related factors is clearly needed.

The effects of brain injury may be influenced by the stage of development of the cognitive function being evaluated (Rutter, 1982). Since children are in the process of developing skills, brain injury may produce deficits that reflect the degree to which a skill has been acquired. Skills that are in a stage of rapid development may be more vulnerable to disruption by brain injury than more fully developed skills (Ewing-Cobbs et al., in press). For example, Ewing-Cobbs and associates (in press) identified greater impairment in written language skills, such as writing to dictation, in children as compared to adolescents. As a skill becomes overlearned and automatized, it may be more resistant to disruption. Many skills develop rapidly prior to age six. If skills that are developing rapidly are susceptible to disruption, then brain injury in young children may produce more significant cognitive deficits than comparable damage in older children. Systematic evaluation of cognitive and motor functions in young children following CHI has not been undertaken. The purpose of this study is to evaluate the nature and severity of cognitive deficits in infants and preschoolers during the subacute stage of recovery from CHI. The recovery of function in intellectual, language, and motor skills at least six months post injury will be evaluated. The following hypotheses were examined:

Hypothesis 1. Children with severe injuries will be more impaired on

measures of intelligence, expressive and receptive language, and gross motor functions than children with mild/moderate injuries on the baseline evaluation.

Hypothesis 2. On the initial assessment, gross motor and expressive language functions will be more impaired than receptive language and intellectual functions.

Hypothesis 3. Both expressive and receptive language functions will be acutely affected by severe CHI. Expressive language is expected to be more impaired than receptive language.

Hypothesis 4. Significant recovery will be apparent in all skill areas by the follow up evaluation. Less recovery will be apparent in gross motor than in intellectual and language skills.

Hypothesis 5. It is expected that the duration of impaired consciousness will be a better predictor of subacute neuropsychological deficit than either the GCS scores obtained at admission or 24 hours post injury.

Method

Subjects

Neuropsychologic functions were examined in 21 children four months through five years of age who sustained a closed head injury. All of the children were hospitalized at Hermann Hospital. The children were participants in a longitudinal study examining neurobehavioral outcome following CHI. Criteria for exclusion from the study were 1) a history of prior central nervous system insult or neuropsychological disorder; 2) positive indications of developmental delay, such as delayed achievement of major developmental milestones; and 3) suspected neglect or child abuse.

Demographic information regarding age, sex, ethnicity, socioecomonic status, and the injury-test interval is provided in Table 1. The mean age of the children was 32.7 months; age at the time of injury ranged from four to 64 months. The ratio of males to females was 4 to 3. The distribution of sex corresponds with epidemiological studies indicating that males are slightly more likely than females to sustain a CHI in the preschool years (Annegers et al., 1980). The socioeconomic background of the families was estimated using the Hollingshead Two Factor Index of Social Position (Hollingshead & Redlich, 1958). As indicated by Table 1, the majority of the sample is composed of the middle to lower social classes. The distribution of socioeconomic status in children with mild-moderate and severe CHI

Table 1
Demographic Information by Severity of Injury

Variable	Severity of injury	
	Mild-moderate (n=8)	Severe (n=13)
Age (months)		
M	26.0	36.8
SD	16.3	18.7
Range	4-53	8-64
Sex		
F	4	5
M	4	8
Ethnicity		
Black	1	2
White	6	9
Hispanic	1	2
Socioeconomic status†		
M	3.37	3.92
Injury-test interval (days)		
Baseline		
M	21.5	34.7
SD	10.6	22.8
Follow up		
M	260.0	242.5
SD	118.5	74.0

† Based on the Hollingshead Two Factor Index

was comparable ($t > -1$). Eleven of the children were injured in motor vehicle accidents. Five children, all sustaining severe CHI, were involved in motor vehicle-pedestrian accidents. The remaining five children sustained injuries secondary to falls or sports injuries.

Procedure

Baseline and follow up evaluations were conducted to examine neuropsychological sequelae subsequent to CHI in infants and preschoolers. Baseline evaluations were conducted during the subacute phase of recovery from CHI. Since it is not possible to determine the duration of posttraumatic amnesia in very young children, the children were evaluated when they began to resume normal childhood activities. Although the majority of children were evaluated while hospitalized, the remainder were tested as outpatients. To evaluate the extent of recovery in intellectual, language, and motor skill areas, a follow up evaluation was conducted at least six months after the injury. Each participant was individually evaluated by a trained psychometrician. The mean injury-test interval was 29.7 days for the baseline assessment and 8.3 months for the follow up evaluation (see Table 1).

Neurologic Indices of Injury Severity

Several measures of injury severity were examined to determine their influence on measures of cognitive functioning. The duration of impaired consciousness, Glasgow Coma Scale score, and the results of

Table 2

Neurologic Indices of Injury Severity

Variable	Mild-Moderate (n=8)	Severe (n=13)
Duration of impaired consciousness (days)		
M	.09	7.91
SD	.27	9.05
Range	0-.75	1.5-35
Acute Glasgow Coma Scale score		
GCS ≤ 8	0	11
GCS > 8	8	2
Pupillary abnormality		
None	8	11
Unilaterally unreactive	0	2
Bilaterally unreactive	0	0
Motor abnormalities		
None	8	3
Right hemiparesis	0	8
Left hemiparesis	0	2
Facial paralysis	0	2
Seizure		
None	7	7
Day of injury only	1	2
Multiple occurrences	0	4

Table 3

Duration of Impaired Consciousness in Severely Injured Children

Duration (days)	Number
1 ≤ 7	9
8 ≤ 14	2
15 ≤ 21	1
22 ≤ 28	0
29 ≤ 35	1

computed tomography represent the major injury variables. The major neurologic variables are presented in Table 2.

Duration of impaired consciousness. The duration of impaired consciousness is regarded as an indicant of the overall severity of injury in adults. It is unclear whether the duration of impaired consciousness is a comparable measure of injury severity in infants and very young children. In infants, this measure was defined as the number of days elapsing between the injury and the time at which the child indicates recognition of his/her surroundings by purposeful or goal directed movement (e.g., reaching for an object, crying to indicate a need). In children ages 3 through 5, the duration of impaired consciousness was defined as the number of days elapsing before the child obeyed a simple command (e.g., open your eyes; squeeze my hand). The distribution of impaired consciousness in children with severe CHI is presented in Table 3.

Glasgow Coma Scale. The severity of injury was also assessed using the Glasgow Coma Scale (GCS) (Teasdale & Jennett, 1974). The GCS evaluates three components of consciousness: eye opening, motor response, and verbal response. This scale yields a composite score ranging from 3 (no eye opening, motor response, or verbal response) to 15 (spontaneous eye opening, responds to simple commands, and is oriented). According to the GCS, coma is defined as the absence of eye opening, failure to respond to commands, and failure to utter

Table 5

The Glasgow Coma Scale

Eye Opening

1 None
2 To Pain
3 To Speech
4 Spontaneous

Motor Response

1 None
2 Extension
3 Abnormal Flexion
4 Withdrawal
5 Localizes Pain
6 Obeys Command (Goal directed movement)

Verbal Response

1 None
2 Incomprehensible
3 Inappropriate (Cries)
4 Confused (Cries to indicate needs)
5 Appropriate (Babbles or attempts to communicate through either gestural or verbal means)

Table 5

Glasgow Coma Scale Scores by Severity of Injury

	Severity of injury	
Time of observation	Severe (n=8)	Mild-Moderate (n=13)
Admission		
3-8	0	11
9-12	1	2
13-15	7	0
24 hours post injury		
3-8	0	8
9-12	1	5
13-15	7	0

recognizable words. A GCS score of 8 or less corresponds to this definition. GCS scores were obtained on admission to the hosptial as well as 24 hours following the injury. Since the GCS was devised for adults, it was modified slightly to accomodate the behavioral capabilities of infants. Table 4 contains the point values and criteria for the GCS. Modifications for children ages 4 through 35 months of age are indicated by parentheses. In addition, the GCS scores obtained at admission and 24 hours following the injury are presented in Table 5.

Computed Tomography. Computed tomography provides a means of visualizing the nature of brain injury. CT scans obtained within two weeks of admission were reviewed by either a neurosurgeon or a radiologist to determine the type of cerebral insult sustained. Four of the children in the mild-moderate group did not receive CT. Previous studies suggested that pediatric head injury was associated with a decreased incidence of mass lesions and an increased incidence of generalized cerebral swelling as compared to adults (Bruce et al., 1979; Jennett, 1972). However, as indicated in Table 6, a significant proportion of the children sustained focal cerebral injuries. This suggests either that the sample is not representative of the total population or that the type of cerebral damage is different in younger children. Additionally, the use of improved imaging techniques during the past decade may have revealed focal lesions not detected by

Table 6

Computed Tomographic Findings in Head Injured Children

Severity of Injury	Initial CT	Second CT
Mild-moderate		
A.B.	SAH, depressed left frontal fx, right frontal fx.	---
L.M.	Right temporal-parietal epidural hematoma	---
J.R.	WNL	---
J.T.	Posterior corpus callosum hematoma, left fronto-parietal hygroma	same, left parietal depressed fx.
Severe		
S.A.	Basilar fx.	---
J.A.	Depressed right frontal fx.	---
P.A.	Right frontoparietal subdural hematoma	Right hemisphere edema, right frontal subdural
P.B.	Right temporal basilar fx., pneumocephalus	Right temporal basilar fx.
C.B.	WNL	Bifrontal and left temporal hygromas (14)
J.C.	WNL	---
C.C.	Left parietal contusion, left frontal edema, bifrontal and left parietal fxs	Bifrontal edema, left parietal contusion (4)

Table 6 (contd.)

Severity of Injury	Initial CT	Second CT
J.C.	Midbrain contusion, left frontal epidural hematoma, left frontal fx., facial fx's	Unchanged (5)
A.C.	Bifrontal contusions, right fronto-parietal fx	Unchanged, plus right parietal contusion
J.H.	WNL	Right occipital contusion (14)
W.P.	Left cerebellar contusion, SAH, lateral ventricular enlargement	Unchanged, plus intra-ventricular hemorrhage (2)
K.P.	Midbrain and right temporal contusion, right temporal subdural hematoma, left temporal fx	---
B.P.	WNL	Cerebral atrophy (7)

Note. SAH=subarachnoid hemorrhage, fx=fracture, WNL=within normal limits. The initial CT scan was performed on the day of admission. Values in parentheses indicate the number of days post injury the second CT scans were performed.

previous technologies.

Neuropsychological Assessment

Several tests were selected to provide standardized measures of general intellectual ability, expressive and receptive language skills, and motor functions. The best available measure of each skill area was selected. The tests administered to children with mental ages in the four through 42 and 43 through 72 month ranges are listed in Table 7.

Group 1. Intellectual skills in children ages 4 to 30 months were evaluated with the Bayley Scales of Infant Development (BSID) (Bayley, 1969). The BSID Mental Scale yields a Mental Development Index (MDI) that reflects the child's performance on a variety of language, visual motor, visual spatial, and problem solving tasks. The Stanford-Binet Intelligence Scale Form L-M (SBIS) (Terman & Merrill, 1972) was administered to children ages 30 to 42 months. The SBIS is composed predominantly of language, visual discrimination, and visual motor items. Language functions were assessed using the Sequenced Inventory of Communication Development (SICD) (Hedrick, Prather, & Tobin, 1975). The SICD evaluates both receptive and expressive language in children from 4 to 48 months of age. This inventory yields a Receptive Communication Age (RCA) and an Expressive Communication Age (ECA) that is expressed in months. Motor skills were evaluated with the Bayley Motor Scale which primarily assesses

Table 7

Neuropsychological Assessment According to Mental Age

Skill area	Mental age	
	4-42 months	42-72 months
Intellectual		
	Bayley Scales of Infant Development Mental Scale (MDI) Stanford-Binet Intelligence Scale	McCarthy Scales of Children's Abilities (GCI)
Language		
Expressive	Sequenced Inventory of Communication Development (ECA)	McCarthy Verbal Scale
Receptive	Sequenced Inventory of Communication Development (RCA)	Peabody Picture Vocabulary Test-Revised (PPVT-R)
Motor		
	Bayley Scales of Infant Development Motor Scale (PDI)	McCarthy Motor Scale

gross motor coordination. This scale yields a Physical Development Index (PDI).

Group 2. To examine intellectual functioning, the McCarthy Scales of Children's Abilities (McCarthy, 1972) was administered to children ages 42 to 72 months of age. This scale yields a General Cognitive Index (GCI) reflecting performance on language, numerical, and visual spatial tasks. Expressive language skills were assessed using the McCarthy Verbal Scale. Receptive language was assessed with the Peabody Picture Vocabulary Test-Revised (PPVT-R) (Dunn & Dunn, 1981), which measures vocabulary knowledge. Gross motor and visual motor performance were assessed by the McCarthy Motor Scale.

Composite neuropsychological measures. Scores obtained from each skill area were combined to yield summary variables for use in statistical analyses. All of the measures of intellectual functioning, as well as the PPVT-R, were standardized with a mean of 100 and a standard deviation of 15. The McCarthy Verbal and Motor scales had a mean of 50 and a standard deviation of 10. To ensure comparability across measures, the McCarthy Verbal and Motor scores were converted to the same scale as the intelligence scores. The SICD Receptive and Expressive Communication Ages are expressed in months. To make them comparable to the other scores, they were converted into IQ equivalents using the following formula: (Communication Age/Chronological Age)*100. Although this procedure

yields a mean of 100, the standard deviation is not necessarily comparable to the other measures.

The composite intellectual variable is composed of the standard scores from the Bayley, Binet, or McCarthy tests. The composite motor score is based on combining the standard scores obtained from the Bayley or McCarthy motor scales. Expressive language was examined by combining the SICD expressive scale and the McCarthy verbal scale. The receptive language composite score reflects performance on the SICD receptive scale or the PPVT-R.

Design

Index variable. The major index variable was severity of CHI. The severity of injury was grouped into mild-moderate and severe categories. The mild-moderate injury group was composed of children exhibiting impaired consciousness, defined as an inability to follow simple commands or to display goal-directed movements, for less than one day. CT scan abnormalities and neurologic deficit may have been present. Severe injury was defined as producing a state of impaired consciousness for at least one day; in addition, CT scan abnormalities and neurologic deficit may have been present.

Dependent variables. The dependent variables are the composite intelligence, motor, expressive language, and receptive language scores obtained during the baseline and follow up examinations.

Design. The design is a 2 (severity) x 2 (time) factorial. The

severity factor is composed of mild-moderate and severe injury. The time factor consists of neuropsychological testing at conducted at baseline and six month follow up intervals.

Results

Profiles of neuropsychologic impairment in the subacute and long term phases of recovery from head injury were examined. The influence of severity of injury on intellectual, motor, expressive language, and receptive language performance was evaluated. In addition, the contribution of indices of neurologic impairment to the prediction of neuropsychologic performance was investigated. Since language functions have not been described in preschool-aged children sustaining CHI, the nature and severity of linguistic deficits were discussed.

Severity of Injury and Time of Testing Effects

To evaluate the impact of severity of injury on neuropsychologic performance, a repeated measures MANOVA was performed on each of the intellectual, motor, expressive language, and receptive language composite scores. Severity of injury was the index variable; dependent variables were the composite scores obtained on the baseline and follow up evaluations. Descriptive statistics for each of the composite measures obtained at baseline and follow up are detailed in Tables 8 and 9, respectively. In addition, the degrees of freedom and F ratios obtained in the MANOVA analyses are presented in Table 10 .

Intellectual functions. Children with severe CHI consistently

performed at a lower level than children sustaining mild-moderate injuries on baseline and follow up measures of intellectual functioning ($F(1,19)=12.09$, $p < .005$). The mild-moderate group scored an average of 104.3 points at baseline while the severely injured children averaged 75.3 points. Analysis of the difference in performance at baseline and follow up suggested that significant recovery was achieved by the head injured children ($F(1,19)=16.35$, $p < .001$). A significant interaction effect was obtained, suggesting that the amount of recovery differed across the severity groups ($F(1,19)=4.04$, $p < .06$). Examination of the group means revealed that children sustaining severe injuries exhibited a greater degree of recovery than children sustaining mild-moderate injuries. By the follow up examination, children in the mild-moderate group gained an average of three IQ points while scores for the severe group increased 11.3 points. This difference is illustrated in Figure 1 by the steeper slope in the line depicting recovery over time in severely injured children. Repeated measures analysis indicated that the severe group began to "catch up" to the performance of less severely injured children. However, a ceiling effect may be present in the scores of the children with mild-moderate injuries. Given the high baseline IQ scores and the small increment in performance over time in this group, intellectual functions may have been minimally disrupted. Compared to the

Table 8

Performance on Baseline Neuropsychological Tests by Severity of Injury

	Severity of injury	
Function	Mild-moderate	Severe
Intellectual		
n	8	13
M	104.3	75.3
SD	16.1	14.9
Range	85-135	50-93
Motor		
n	8	10
M	96.4	66.6
SD	25.5	19.1
Range	50-131	50-103
Expressive language		
n	8	12
M	87.7	70.7
SD	16.6	17.2
Range	60-111	50-94
Receptive language		
n	7	12
M	98.1	74.9
SD	13.9	16.9
Range	50-124	50-103

Table 9

Performance on Follow Up Neuropsychological Tests by Severity of Injury

Function	Severity of injury	
	Mild-moderate	Severe
Intellectual		
n	8	13
M	107.3	86.7
SD	16.7	18.1
Range	89-137	50-112
Motor		
n	8	10
M	105.3	76.2
SD	13.7	18.2
Range	90-130	50-102
Expressive language		
n	8	12
M	96.6	84.1
SD	15.9	17.4
Range	76-121	50-108
Receptive language		
n	7	12
M	101.7	81.6
SD	10.7	19.6
Range	89-114	50-132

Table 10

Severity and Time of Testing Effects for Composite Neuropsychological Scores (MANOVA)

Composite measure	df	F ratios Severity	Time	Severity x Time
Intelligence	(1,19)	12.09***	16.35****	4.04†
Motor	(1,15)	10.90***	5.77*	0.05
Expressive	(1,18)	4.82*	9.70**	0.34
Receptive	(1,17)	10.36***	1.85	0.15

† $p < .06$
* $p < .05$
** $p < .01$
*** $p < .005$
**** $p < .001$

Figure 1
Performance on Intelligence and Motor Composite Scores on Baseline and Follow Up Evaluations by Severity of Injury

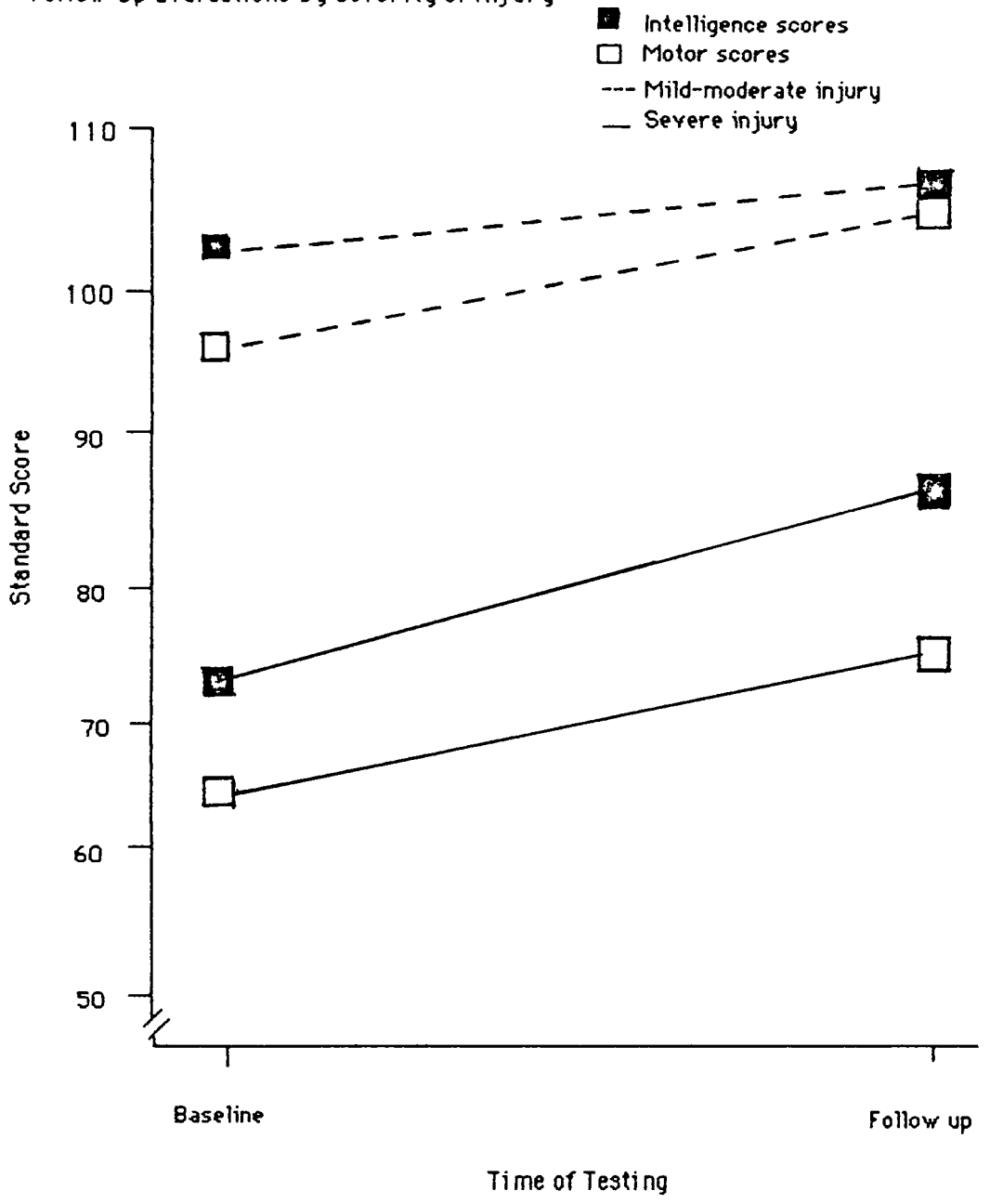
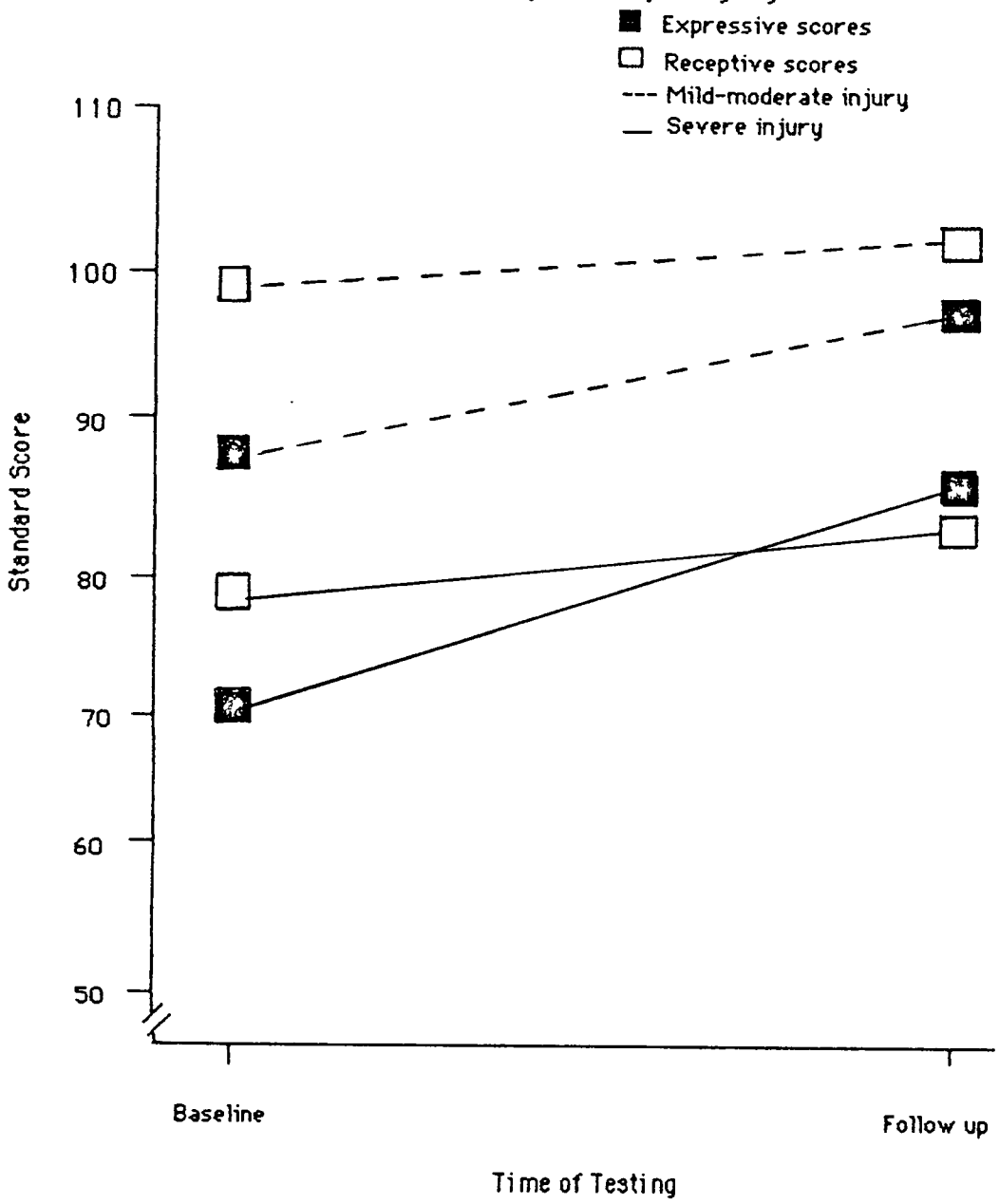


Figure 2
Performance on Expressive and Receptive Language Composite Scores on Baseline and Follow up Evaluations by Severity of Injury



restricted range of scores in the mild-moderate group, even modest changes in intellectual performance in the severely injured children would approach statistical significance. Despite the intellectual recovery by the follow up assessment in children with severe CHI, a 21 point difference was present between the groups. Significant impairment in intellectual functions persisted in severely injured children by nine months post injury.

Motor functions. The performance of severely injured children on measures of motor functioning was impaired relative to children with mild-moderate injuries ($F(1,15)=12.90$, $p < .005$). On the baseline evaluation, the group means were 66.6 and 96.4, respectively. Performance improved significantly in both groups by the follow up assessment ($F(1,15)=5.77$, $p < .05$). Children in each severity group gained approximately nine points by the follow up testing. However, performance of the severely injured children remained significantly impaired. The mean composite score was 76.2, which was 29 points below that obtained by the mild-moderate group. The rate of recovery across time was similar in both severity groups; no interaction between severity of injury and time of testing was obtained. As indicated in Figure 1, the recovery slopes for the severity groups are parallel. Even though the mean baseline performance of the mild-moderate group was in the normal range, the significant

increase in performance over time indicates that motor functions were initially reduced by the CHI. As expected, the motor performance of children sustaining severe CHI did not show signs of catching up to the performance of less severely injured children over time.

Expressive language. Children with severe injuries exhibited greater difficulty on measures of expressive language skills than children with mild-moderate CHI ($F(1,18)=4.82$, $p < .05$). The mean score for severely injured children at baseline was 70.7 while the mild-moderate group averaged 87.7 points. The low mean score in the mild-moderate group may reflect impairment in expressive functions. Performance on expressive language tasks was significantly improved by the follow up assessment ($F(1,18)=9.70$, $p < .01$). Severely injured children gained 13.4 points while performance of the mild-moderate group increased 9 points. Examination of the interaction between severity of injury and time of testing indicated that the amount of recovery was comparable in both severity groups. Figure 2 presents the nearly parallel slopes depicting recovery of expressive language function by severity of injury. By approximately nine months after the injury, performance of the severely injured children was 12.5 points below the mild-moderate group. Despite the significant gain of expressive language skills in severely injured children, their performance did not catch up to that of children with

less significant injuries. Moreover, given the improvement over time in expressive language skills in the mild-moderate group, the failure to obtain significant differences between the severity groups suggests that performance was depressed in both groups.

Receptive language. Receptive language skills were significantly reduced in children sustaining severe injuries as compared to children with mild-moderate injuries ($F(1,17)=10.36$, $p < .005$). At baseline, the group means were 74.9 and 98.1, respectively. However, performance on the follow up assessment was not significantly different from performance on the initial evaluation. On the follow up examination, the mild-moderate group gained 3.6 points while the severely injured children gained 6.7 points. The amount of recovery of receptive language skills over time was similar in both groups as indicated by a nonsignificant interaction effect. As depicted in Figure 2, the slopes of the recovery curves are parallel. By nine months post injury, the severe group performed 20.1 points below the comparison group. Given the lack of "catch up", severely injured children exhibited a receptive language deficit relative to children with mild-moderate injuries.

Comparison of performance in different domains. To determine which skill areas were more likely to be impaired, a 2 (severity) X 2 (task) within-subjects MANOVA was employed to compare composite

Table 11

Comparison of Baseline and Follow Up Performance in Neuropsychological Functions (MANOVA)

Neuropsychological functions	df	F ratios		
		Severity	Task	Severity x Task
Baseline assessment				
Intelligence versus Motor	(1,16)	13.68**	5.44*	0.01
Expressive versus Receptive	(1,16)	7.60*	6.26*	0.14
Follow up assessment				
Intelligence versus Motor	(1,17)	11.73***	5.72*	1.86
Expressive versus Receptive	(1,18)	6.23*	0.04	0.27

* $p < .05$

** $p < .01$

*** $p < .005$

scores. The first analysis compared performance on the composite intellectual and motor scores. Since the language scores were based in part on transformation of mental ages into IQ equivalents as opposed to deviation scores, the language scores were analyzed separately. Due to the small number of participants and the restricted degrees of freedom, it was not possible to examine recovery over time using an omnibus MANOVA. Therefore, a 2 x 2 MANOVA was performed on the follow up 1) intelligence and motor and 2) receptive and expressive language composite scores. The degrees of freedom and F ratios for the baseline and follow up analyses are provided in Table 11 .

Comparison of performance on the baseline intelligence and motor composite scores revealed that motor scores were significantly more reduced than intelligence scores ($F(1,16)=5.44$, $p<.05$). Due to missing data on motor functioning in three children, the analysis was based on 18 cases. Collapsing across severity, the means on the intelligence and motor variables were 87.2 and 78.9, respectively. No interaction between severity of injury and performance on the composite scores was obtained. Children with severe injuries were more impaired in both skill areas than children sustaining mild-moderate CHI. The disparity between the level of intelligence and motor performance is illustrated in Figure 1.

At the follow up examination, performance on motor tasks was

significantly below performance on measures of intellectual functioning ($F(1,17)=11.73$, $p < .05$). Collapsing across severity, the mean intelligence score was 94.5 and the mean motor score was 88.5. A persistent deficit was present by approximately eight months post injury in motor skills as compared to intellectual skills. The amount of recovery was comparable in both severity groups as suggested by a nonsignificant severity by task interaction effect.

Performance on the baseline expressive and receptive language composite scores was examined in 18 children. One child was unable to perform expressive language tasks due to a tracheostomy; receptive skills were not evaluated in two children. Collapsing across severity, the mean expressive score was 76.6 and the mean receptive score was 85.3. A main effect for task was obtained ($F(1,16)=6.26$, $p<.05$). Expressive skills were significantly more impaired than receptive skills during the subacute stage of recovery from CHI. No interaction between the severity of injury and performance on measures of expressive and receptive tasks was obtained. Figure 2 depicts the relationship between language scores and injury severity. As can be seen, the relationship between injury severity and performance on language tasks is similar in the severity groups. Moreover, the level of performance on expressive tasks is clearly lower than performance on receptive language tasks.

By the follow up examination, no main effect for task was obtained, suggesting that expressive language scores improved relative to the receptive language scores. Independent of severity of injury, a mean of 89.1 was obtained for the expressive scores while the receptive scores averaged 90 points. The level of expressive language functions caught up to the level of receptive skills. Recovery of language functions occurred in both severity groups as indicated by a nonsignificant severity x task interaction.

Neurologic Indices of Injury Severity

Hierarchical regression was used to examine the ability of several indices of neurologic impairment to predict the level of performance on the baseline intelligence, motor, expressive language, and receptive language composite scores. Each composite score was analyzed separately. The index variables were the duration of impaired consciousness, the GCS score obtained 24 hours post injury, and the initial GCS score reported on hospital admission. Each of the three index variables was separately entered into the regression equation to determine if a significant increment in predictive ability was achieved. The index variable entered on the first step was the duration of impaired consciousness. The GCS scores obtained at 24 hours post injury and the initial GCS score were entered on the second and third steps, respectively. The index variables were

intercorrelated. The duration of impaired consciousness was significantly correlated with both the 24 hour GCS score ($r=-.59$, $p<.005$) and the acute GCS score ($r=-.49$, $p<.05$). In addition, the two GCS scores were highly intercorrelated ($r=.91$, $p<.001$). Table 12 contains the multiple R, R^2, R^2 adjusted for shrinkage, and change in the F ratio obtained at each step in each regression analysis.

Performance on measures of intellectual functioning was predicted by the duration of impaired consciousness ($F(1,18)=15.89$, $p < .001$). The multiple R was .68 . The R^2 adjusted for shrinkage was .44, indicating that the duration of impaired consciousness accounted for 44 percent of the variance in intellectual performance. Inclusion of the 24 hour GCS score in the regression equation produced a .13 increment in R^2 ($F_{inc}(2,17)=5.65$, $p < .05$). With both variables in the equation, the R^2 adjusted for shrinkage was .55, suggesting that these variables were very sensitive predictors of intellectual deficit. After step 3, with GCS 1 added to the equation, no significant increase in predictive ability was achieved.

The results of hierarchical regression analysis on the composite motor score indicated that the duration of impaired consciousness was a significant predictor of the level of performance $R^2=.24$, ($F(1,15)=4.71$, $p < .05$). The duration of impaired consciousness, when adjusted for shrinkage, explained 19 percent of the variability in

Table 12

Hierarchical Regression of Neurologic Indices on Baseline Composite Neuropsychologic Scores

Variable	df	Multiple R	R^2	Adjusted R^2	R^2 change	F change
Intelligence						
DIC	1,18	.68	.47	.44	.47	15.89**
GCS 24	2,17	.77	.60	.55	.13	5.65*
GCS 1	3,16	.78	.61	.54	.01	.57
Motor						
DIC	1,15	.49	.24	.19	.24	4.71*
GCS 24	2,14	.65	.42	.34	.18	4.33'
GCS 1	3,13	.65	.43	.29	.01	.18
Expressive language						
DIC	1,17	.43	.18	.14	.18	3.86'
GCS 24	2,16	.49	.25	.15	.06	1.24
GCS 1	3,15	.56	.32	.18	.07	1.64
Receptive language						
DIC	1,16	.55	.30	.26	.30	7.00*
GCS 24	2,15	.60	.36	.27	.05	1.22
GCS 1	3,14	.61	.37	.24	.02	0.41

Note. DIC=duration of impaired consciousness, GCS 24=Glasgow Coma Scale Score obtained 24 hours post injury, GCS 1 = Glasgow Coma Scale score obtained on admission.

Note. ' $p < .07$

* $p < .05$

** $p < .001$

motor scores. Inclusion of the 24 hour GCS score resulted in a significant increment in R^2 ($F_{inc}(2,14)=4.33$, $p < .06$). After step 2, with two variables in the equation, the adjusted R^2 was .42, indicating that 42 percent of the variance in motor scores was explained. Addition of the acute GCS score in the equation did not produce a significant increment in R^2.

A trend was obtained suggesting that the level of expressive language scores was related to the duration of impaired consciousness ($F(1,17)=3.86$, $p<.07$). The adjusted R^2 was .14, indicating that 14 percent of the variance was explained. Inclusion of the 24 hour GCS score on the second step did not yield a significant increment in R^2. Addition of the acute GCS score on the third step did not improve prediction of expressive scores. When adjusted for shrinkage, 18 percent of the variability in expressive language performance was accounted for by inclusion of all of the index variables.

The duration of impaired consciousness was significantly related to performance on receptive language tasks. After step 1, with the duration of impaired consciousness in the equation, $R^2=.30$ ($F(1,16)=7.00$, $p<.05$). Addition of the 24 hour and acute GCS scores on steps 2 and 3 did not reliably improve R^2. With all three variables in the equation, the adjusted R^2 =.24, indicating that 24

percent of the variability in receptive language scores was explained.

The duration of impaired consciousness appears to be the best predictor of performance on intelligence, motor, expressive, and receptive language tasks. Addition of the 24 hour GCS score significantly improved prediction on measures of intellectual and motoric functioning. The 24 hour GCS score did not produce a significant increment in R^2 when included in the regression equations for either of the language measures. The acute GCS score did not reliably contribute to predictive ability in any of the analyses. As indicated by the R^2 values, knowledge of the duration of impaired consciousness and the 24 hour GCS score explained a large proportion of the variance in neuropsychologic performance.

Linguistic Disturbances following CHI

Subacute language functions. The type of linguistic disturbance observed in severely injured children during the subacute stage of recovery from CHI was examined. Of the 13 children sustaining severe CHI, 12 received a baseline assessment of expressive language. The remaining child had a tracheostomy performed for subglottal stenosis and consequently was not able to perform expressive language tasks. Receptive language was assessed in 12 children. Table 13 describes the major expressive and receptive language findings in severely injured children. In addition, the duration of impaired consciousness, duration of mutism, baseline IQ scores, age at assessment, and the injury-test interval are provided for each child.

The expressive langauge deficits observed in four children shared features with the symptoms associated with apraxia of speech. Apraxia of speech is characterized by difficulty voluntarily initiating speech. In the acute phase of recovery, mutism is typically observed. As speech functions improve, difficulties selecting and sequencing phonemes are apparent. The phonemic errors often consist of omission and/or substitution. Transition from phoneme to phoneme is disrupted, resulting in a loss of automaticity. Speech improves to slow, effortful speech and explosive articulation. In adults, apraxia of speech is not associated with agrammatism, anomia, or dysphasia.

Table 13

Type of Linguistic Disturbance Observed in Severely Injured Children

Case	DIC† (days)	Mutism (days)	Expressive Functions	Receptive Functions	IQ	Age (mos.)	ITI††
S.A.	2.0	0	Expressive functions age-appropriate except for oral vocabulary. McCarthy Verbal=90	Mild receptive deficit. Difficulty following complex commands and understanding grammatical structures. PPVT-R=80	93	64	15
J.A.	1.5	0	Expressive functions intact except for mild repetition deficit. No aphasic symptoms. McCarthy Verbal=85	Receptive vocabulary slightly below age level (child bilingual). PPVT-R=86	90	56	27
P.A.	35.0	35	Occasional consonant-vowel combinations. No imitation of sounds or words. No gestural communication. ECA < 50	Localized sounds, no response to name. RCA < 50	<50	18	75
P.B.	1.5	7	Occasional jabbering. Attempted to imitate words. Did not name objects or gesturally respond to questions. ECA= 66	Did not respond to specific words or follow directions. RCA=66	78	17	12

Table 13 (contd.)

Case	DIC† (days)	Mutism (days)	Expressive Functions	Receptive Functions	IQ	Age	ITI
C.B.	7.0	9	Occasional spontaneous 1 word utterances. Did not name objects. Imitated speech sounds but not words. ECA=65	Receptive vocabulary age-appropriate. Followed complex commands, identified body parts. RCA=90	61	31	13
J.C.	5.0	10	Speech moderately dyspraxic. Difficulty initiating and organizing speech. Many phonemic omissions and substitutions. Occasionally identified objects. Spontaneous 1 word utterances characterized by decreased intelligibility. ECA=83	Correctly identified body parts and objects. Matched pictures to objects. Followed 1 stage commands. RCA=71	70	45	26
C.C.	3.0	>26	Severely dyspraxic speech; buccofacial apraxia. Unable to initiate vocalization or imitate oral movements. Relied on gestural communication. ECA < 50	Inconsistently followed 2 stage commands. Identified body parts and objects, good comprehension of single words. RCA=84	78	33	28

Table 13 (contd.)

Case	DIC† (days)	Mutism (days)	Expressive Functions	Receptive Functions	IQ	Age	ITI
J.C.	7.0	16	Speech severely dyspraxic. Initially unable to initiate vocalization or imitate oral movements. Dysarthric. McCarthy Verbal=70	Identified body parts and objects, comprehension of single words appropriate. Decreased comprehension of grammatical structures. PPVT-R=84	73	59	71
A.C.	3.0	6	Occasional consonant-vowel combinations. Localized sounds and imitated speech sounds. ECA=78.	Attends to speech, responded to gestures. RCA=78	93	10	42
J.H.	9.0	18	Speech severely dyspraxic; buccofacial apraxia; dysphagia. Right central facial weakness. Occasional single word utterances. Imitated motor acts using the upper extremities but not oral movements or nonspeech sounds. Communicated largely using gestures. ECA < 50	Moderate receptive deficit. Completed two stage commands, identified common objects and body parts. RCA=76	75	30	26

Table 13 (contd.)

Case	DIC† (days)	Mutism (days)	Expressive Functions	Receptive Functions	IQ	Age	ITI
W.P.	4.0	3	Left facial weakness. Jaws wired. Speech characterized by reduced articulatory precision, labored output, mildly decreased vocal intensity, and reduced voluntary initiation. McCarthy Verbal=83	Followed complex commands, identified objects by function. Receptive skills close to age level.	76	58	58
K.P.	10.0	2	Significantly decreased vocal intensity, articulatory precision, and voluntary initiation of speech. Right sided central facial paralysis. ECA=94	Age-appropriate identification of colors, pictures. Receptive vocabulary and comprehension of spoken sentences within normal limits. RCA=103	92	45	21
B.P.	15.0	--	Tracheostomy performed for subglottal stenosis.	Responded to name, carried out requests accompanied by gestures. Did not appear to comprehend words. RCA < 50	<50	24	54

†DIC=duration of impaired consciousness

††ITI=injury-test interval

It is the isolated loss of the ability to articulate words. Dysphagia, buccofacial dyspraxia, and upper extremity weakness may be present. In adults, apraxia of speech has been identified in patients with vascular lesions involving Broca's area, the left facial sensorimotor region, or both areas. Additionally, the subcortical connections between the two areas may be affected.

The children with dyspraxic speech difficulties ranged in age from 30 to 59 months at the time of the initial language evaluation. Interestingly, these children were rendered mute for longer periods of time than children with other language disorders. The duration of mutism was defined as the number of days a child was unable to spontaneously produce or imitate speech sounds after recovering from coma. The children with dyspraxic deficits were mute from at least 10 to greater than 26 days. CT scan findings in these children are as follows: C.C.--left parietal contusion and bifrontal edema, J.C.--WNL, J.C.--midbrain contusion and left frontal epidural hematoma, and J.H.--right parietal-occipital contusion. Three of the four children had a right hemiparesis while the remaining child (C.C.) did not exhibit gross motor abnormalities. In addition to dyspraxic speech deficits, two of the children also clearly exhibited buccofacial apraxia. They were unable to perform voluntary movements of the oral apparatus such as protruding the tongue, blowing, or sucking although

they were able to imitate motor acts involving the hands. Phonetic production was characterized by omission and substitution of phonemes (e.g., horsie=or-hee; tree=ree).

A primary dysarthria was observed in two children (W.P. and K.P.). Their speech was characterized by reduced articulatory precision, effortful output, decreased vocal intensity, and reduced voluntary initiation of speech. Spontaneous speech was not produced for 2 to 3 days following improvement in their level of consciousness. Scores on the McCarthy Verbal Scale were in the normal range and specific receptive language disorders were not observed. Both of the children had facial weakness.

The remaining children exhibited relatively nonspecific linguistic disturbances. Two children exhibited gross mental deficiency (IQ < 50). Language functions were severely depressed. Two children (S.A. and J.A.) were not mute and did not exhibit specific expressive or receptive deficits. Speech was normal with no evidence of an acquired language disorder. The remainder of the sample (P.B., C. B., and A. C.) were rendered mute for approximately a week; expressive and receptive language functions were adversely affected. While verbal output was clearly reduced and simplified, linguistic deficits did not conform to a specific pattern.

Recovery of linguistic functions. Children exhibiting oral/verbal

dyspraxic difficulties during the subacute stage of recovery from CHI displayed significant recovery of language functions by the follow up examination. Two of the four dyspraxic children had persistent articulation disturbances; one of the children with articulation deficits continued to exhibit dyspraxic speech characterized by omission of phonemes located in varying positions within words. The articulation of the other two children was within normal limits. Based on samples of spontaneous speech, the content of speech was simplified in two children and the mean length utterance was slightly below expected levels. Receptive language skills were age-appropriate in 3 of the children. The remaining child exhibited significant attentional deficits that adversely affected his performance on receptive tasks. In fact, three of the four children had significant difficulty focusing and sustaining attention.

Language functions in the dysarthric children were age-appropriate at follow up. One child continued to exhibit dysarthria while the spontaneous speech of the other child was within normal limits. Significant recovery of expressive and receptive skills was observed in the remainder of the sample without gross mental defect.

Discussion

The effect of mild-moderate and severe CHI on intellectual, gross motor, expressive language, and receptive language performance was examined. Linguistic disturbances identified in the present study were compared with profiles of acquired language disorders in children and head injured adults. The contribution of indices of neurologic impairment to the prediction of subacute neuropsychologic impairment was discussed.

Severity of Injury

As predicted by hypothesis 1, children sustaining severe CHI were significantly more impaired on measures of intellectual, motor, expressive language, and receptive language functions than children with mild-moderate injuries. This finding is consistent with the observation that brain injury may produce generalized cognitive impairment in children (Rutter, 1982). The performance of children with mild-moderate injuries was clearly reduced on the baseline assessment of motor and expressive language skills as indicated by the significant degree of recovery by the follow up evaluation. The impairment observed in children with mild-moderate CHI supports the idea that there is a continuum of neurologic impairment associated with different degrees of severity of CHI. Oppenheimer (1968) performed autopsies on adults sustainig comparatively mild head injuries who died

from non-CNS complications. Similar to findings in severely injured patients, shear strains and neuronal degeneration were present. Even though children in the mild-moderate group were rendered unconscious for less than one day (mean duration=2 1/4 hours), significant cognitive sequelae were identified. These results parallel the findings reported by other investigators that indicate clear dose-response relationships between the severity of pediatric CHI and the degree of neuropsychological sequelae (e.g., Bawden, Knights, & Winogron, 1985; Chadwick et al., 1981; Levin & Eisenberg, 1979a).

Neuropsychologic Functions

Baseline assessment. To determine which areas of functioning were most affected in the subacute stage of recovery from CHI, performance in 1) intelligence and motor and 2) expressive and receptive language skill areas was compared. As expected, motor and expressive language functions were significantly more impaired than intelligence and receptive language skills on the baseline evaluation. Motor skills were severely disrupted in children sustaining significant injuries. Children aged two and three appeared to be particularly vulnerable to the effects of CHI. Children with hemipareses had a marked disturbance of gross motor coordination that resulted in postural control deficits. They were often unable to sit without support or crawl although a fine pincer grasp of the unaffected hand

was preserved. The postural control in older children with a hemiparesis was less severely affected.

The intelligence scores of children sustaining severe CHI were clearly depressed at baseline. Two of the severely injured children were mute during the initial assessment. Although this reduced their scores, it was felt that the IQ score was an accurate assessment of the childrens' level of functioning if not their intellectual ability. IQ scores in children with mild-moderate CHI were relatively stable over time, suggesting that IQ may not be a particularly sensitive measure of dysfunction in such children.

Comparison of performance in expressive and receptive language areas revealed that expressive skills were significantly more affected than receptive skills. As predicted by hypothesis 3, both expressive and receptive deficits were present in children sustaining severe injuries. Receptive language skills appeared to be fairly robust in children with mild-moderate injuries; the scores at baseline were high and little improvement was observed over time. In children sustaining more significant injuries, the contribution of attentional deficits to impaired performance on receptive language tasks is unknown.

Recovery of skills. By the follow up evaluation, significant recovery was identified in intelligence, motor, and expressive language skills. Despite the recovery observed over time, children

with severe injuries continued to perform below the mild-moderate group on all of the neuropsychological measures. As predicted by hypothesis 4, less recovery was observed in motor skill areas than in intellectual functions. Since the level of expressive skills caught up to the level of the receptive skills by the follow up evaluation, motor skills appear to be the most vulnerable to the effects of CHI.

The rate of recovery in children with severe injuries was greater than in children sustaining mild-moderate injuries on the intelligence measures. Since the change over time in children with lesser injuries was modest, the performance of children with severe injuries began to catch up to the performance of the mild-moderate group. However, children with severe injuries continued to perform below children with mild-moderate injuries by eight months post injury. The rate of recovery was comparable in both severity groups on measures of motor, expressive language, and receptive language functioning.

Comparison of the follow up intelligence and motor scores revealed that motor functions were significantly lower. This represents a deficit in motor skills and suggests that motor skills are more vulnerable to the effects of CHI than are intellectual skills. This finding is consistent with follow up data on children with cerebral involvement from other etiologies that indicate persistent motor involvement (e.g., infantile hemiplegia, cerebral palsy). Gross

motor functions have not been systematically evaluated following pediatric CHI. However, persistent fine motor and visual motor deficits have been identified in head injured children at least one year post injury despite impressive recovery of intellectual functioning (e.g., Bawden et al., 1985; Chadwick et al., 1981; Klonoff et al., 1977).

Comparison of recovery on expressive and receptive language functions revealed that no significant differences were present on the composite scores obtained on the follow up evaluation. Children with severe injuries continued to perform below the level of children with less severe injuries on both measures of language skills. While significant recovery over time was apparent on the expressive measures, changes in receptive language functioning were modest. By eight months post injury, performance on expressive language measures caught up to the level of the receptive language skills.

The disparity in recovery between motor as compared to intellectual and language skills may reflect the type of neural tissue mediating these behaviors. As observed by Goldberger (1974), recovery may be more likely following ablation of "loosely coupled" versus "tightly coupled" tissue. Motor functions are largely subserved by somatotopically organized tissue; there may be little flexibility in the ability of motor functions to be structurally and/or functionally

reorganized. In contrast, intellectual and language functions are subserved by more "loosely coupled" tissue. At the neural level, there may be more capacity for reorganization of function, in addition to increased use of behavioral compensatory strategies. Moreover, many motor systems are composed of subcortical structures. Since CHI appears to disproportionately affect the subcortical white matter, it is possible that the neural substrate for motor functions is subjected to greater diffuse injury. Results from the animal literature suggest that damage to subcortical structures is associated with relatively persistent behavioral deficits (Almli & Johnson, 1976). Higher cognitive functions are primarily subserved by cortical mechanisms; outcome following cortical injury is more variable.

The results of the present study are inconsistent with the traditional plasticity hypothesis (e.g., Smith, 1981). The traditional view proposes that children are 1) less affected by cerebral insult and 2) show more functional recovery than observed in adults. The first tenet was not supported in the present study. Children sustaining severe CHI exhibited significant disruption of all of the functions examined. Moreover, even children with less significant injuries were initially impaired on several measures of cognitive and motor functioning. In the present study, severely injured children displayed persistent deficits on the follow up evaluation in all of the skill areas

relative to children with mild-moderate injuries. Within the time frame of this study, the neuropsychological profiles of the severely injured children do not support the second tenet of the traditional plasticity view. However, it is likely that the severe injury group will continue to make significant cognitive gains over time. The eventual level of performance of children under six years of age who sustain severe injuries is unknown at the present time. Even if many skills recover to age-appropriate levels, the acquisition of more complex skills, such as reading and writing, may be affected. To illustrate, Shaffer et al. (1980) identified a higher prevalence of delayed acquisition of reading skills in children injured prior to eight years of age than in older children sustaining unilateral depressed skull fractures with cortical lacerations. These findings are reminiscent of the results of Goldman's (1974) studies of dorsolateral frontal cortex ablation in infant monkeys. As the monkeys matured, the delayed development of deficits became apparent when the substrate required for performance on more complex tasks was not available.

It is difficult to compare the recovery of cognitive function in children and adults. No direct statistical comparison has been performed across these age groups. The tests used to evaluate cognition in different age groups often vary considerably and make cross-age comparisons difficult. In addition, since children continue

to develop following a cerebral insult, neuropsychological functions need to be evaluated within a longitudinal framework. Behavioral recovery needs to be conceptualized in terms of rate, delay, deficit, and lag constructs to determine how a given cerebral insult affects subsequent development (Satz et al., 1975). Moreover, cerebral insults may differentially affect cognition depending on the stage of development of the skill being evaluated (Rutter, 1982). Ewing-Cobbs et al. (in press,a) examined the effect of age at CHI on composite linguistic measures in school-aged children and adolescents. Age at injury did not influence performance on summary measures of naming, expressive, and receptive language skills. However, written language skills were more impaired in children ages 5-10 than in adolescents. This finding was interpreted as indicating that emerging written language skills were prone to disruption by CHI. Since written language skills are more automatized and overlearned in adolescents, they may be less prone to disruption by cerebral insult. In view of these findings, simplistic statements describing the relationship between age at cerebral insult and cognitive status are not tenable (Levin et al., 1984; St. James-Roberts, 1979).

Linguistic Functions

It is difficult to classify the types of linguistic deficits observed in children with CHI. The classical aphasia classification system is

largely based on syndromes observed secondary to vascular lesions in adults. Since age and etiology variables significantly influence linguistic deficits, the aphasia classification system is not appropriate for use with children. Heilman, Safran, and Geschwind (1971) reported that only two percent of consecutive adult CHI admissions were rendered aphasic. Anomia and Wernicke's aphasia were identified. As observed by Sarno (1980), linguistic deficits following head trauma in adults differ considerably from the classical syndromes identified in stroke patients. If detailed examination of language functions is conducted with quantifiable instruments, the incidence of "subclinical aphasia" in patients with normal conversational speech is rather high in patients rendered comatose (Sarno, 1980). Similarly, Levin, Grossman, and Kelly (1976) reported that linguistic disturbances tended to be generalized and nonspecific, particularly when associated with prolonged coma. Although several instances of specific dysnomia or word finding difficulties were observed by Levin et al. (1976), language profiles consistent with other classical aphasic syndromes were not identified.

Language disturbances in head injured school-aged children and adolescents were examined by Ewing-Cobbs et al. (in press,a). Although speech samples were not available, it was estimated that less than 10 percent of the sample exhibited disruption in

conversational speech. The incidence of aphasic disorder in children sustaining CHI in the preschool years appears to be substantially higher than in older individuals. Excluding infants as well as children rendered mentally defective, six children out of 10 sustaining severe CHI had significant linguistic deficits. In these six children, spontaneous speech was substantially disrupted. Objective measures of expressive and receptive language functioning revealed significantly impaired performance. Although the children in the present study do not represent consecutive admissions since children with premorbid difficulties and those not speaking English were excluded, the sample is likely representative of children in this age range in terms of type and severity of CHI. Despite the fact that the sample appears to be biased in terms of a disproportionate number of children with right as opposed to left hemipareses, the number of children with aphasic disturbances is marked. It appears that young children are particularly susceptible to the disruption of language functions following CHI. Expressive and receptive language functions develop rapidly during the preschool years. By the time children are approximately five years of age, they are able to produce and comprehend the vast majority of basic syntactic constructions used by adults; vocabulary and phonological skills are well developed (Gardner, 1983; Gibson & Levin, 1975). The finding that expressive

and receptive language skills are vulnerable to disruption by CHI is consistent with the hypothesis that skills in a rapid phase of development are susceptible to the effects of brain injury.

and receptive language skills are vulnerable to disruption by CHI is consistent with the hypothesis that skills in a rapid phase of development are susceptible to the effects of brain injury.

Mutism was frequently observed in the severely injured children. Following severe CHI, recovery of eye opening and auditory comprehension for simple commands occurs prior to the production of speech (Bricolo, Turazzi, & Feriotti, 1980). Therefore, it is not surprising that mutism may occur for several days following restoration of the ability to follow commands. The mutism was not associated with a vegetative state, locked-in syndrome, or akinetic mutism. All of the children rendered mute were able to communicate using gestures and cooperated with testing procedures.

Mutism occurs much more frequently when the onset of cerebral pathology is rapid as opposed to slow (Hécaen, Perenin, & Jeannerod, 1984). Hécaen et al. (1984) reported that mutism was more common following trauma or cerebral vascular accidents than in children rendered aphasic by a tumor or abscess. In their sample, mutism was primarily associated with anterior lesions. However, documentation fo lesion sites by CT was not reported. Mutism has also been reported following basal ganglia lesions. Aram, Rose, Rekate, and Whitaker (1983) identified mutism and oral apraxia without dysarthria in a child secondary to an acquired vascular lesion in the left basal ganglia.

Levin, Madison, Bailey, et al. (1983) described mutism following severe CHI in nine adolescents and adults. Four of the patients had focal subcortical lesions while the remaining cases predominantly exhibited severe diffuse brain injury in conjunction with focal left hemisphere pathology. In the present sample, none of the children presented with basal ganglia pathology. Of the 11 children with mutism who were not rendered mentally deficient, only three had either anterior left hemispheric or bilateral anterior lesions. Since the duration of impaired consciousness ranged from 1.5 to 35 days in the mute children, the focal lesions visualized by CT were superimposed on severe diffuse brain injury. As noted by Levin et al. (1983), the contribution of subcortical injury to the presence of mutism may have been underemphasized.

The language deficits observed in the present sample are consistent with previous descriptions of acquired aphasia in children. All of the aphasic children exhibited a reduction in expressive language that was typically preceeded by a period of mutism. Significant articulation deficits were common. Neither agrammatic speech nor semantic paraphasias were identified. The incidence of receptive language impairment was rather high, likely reflecting the diffuse nature of CHI as well as attentional deficits.

Guttman (1942) described a prototypical "frontal aphasia" (or

apraxia of speech) in a six year old traumatically injured child. The aphasia was characterized by a lack of spontaneous speech, although the child attempted to produce speech sounds. The child was not able to follow commands to blow or whistle even though he was able to carry out simple requests. One week following the injury, he was able to produce stereotypic and automatized sequences when urged despite the lack of spontaneous speech. Dysarthria was present. Two days later, spontaneous speech gradually returned and was not characterized by agrammatic errors.

The types of deficits observed by Guttman (1942) were frequently identified in the present sample. He noted that recovery from this type of aphasia was rapid and that the prognosis was good. However, due to confounding variables, factors predictive of recovery from aphasia in children are unclear at the present time (Satz & Bullard-Bates, 1981). Both Guttman (1942) and van Dongen and Loonen (1979) reported that spontaneous recovery was related to both nonfluent aphasia and trauma. This is supported by the high rate of linguistic recovery identified in the present sample.

The rapid resolution of nonfluent aphasia needs to be investigated. Due to the limited verbal output present during the subacute stage of recovery, it is difficult to determine the nature of the linguistic impairment. The relative impairment in semantic/symbolic as opposed

to motor planning and output functions is unclear. Dissociation of these components is difficult since the level of output functions constrains the ability of the child to demonstrate competency on a variety of expressive language tasks. For example, school-aged children with severe CHI may perform significantly below age level on measures of written spelling while demonstrating age-appropriate recognition of correctly spelled words. Unfortunately, in young children, such alternate modalities are not sufficiently developed to permit expression of knowledge.

Neurological Indices of Injury Severity

Hierarchical regression analysis revealed that the duration of impaired consciousness was a good predictor of subacute performance on intelligence, motor, expressive language, and receptive language tasks. With only the duration of impaired consciousness in the regression equation, from 14 to 44 percent of the variance in the dependent variables was explained. Inclusion of either the acute or the 24 hour GCS scores did not consistently yield a significant increment in R^2. The duration of impaired consciousness appears to be a sensitive indicator of the severity of injury in young children.

There did not appear to be a consistent relationship between the presence of focal cerebral lesions and the pattern of neuropsychological scores. Due to the small sample size, statistical

analysis was not feasible. Since the primary mechanism involved in CHI is diffuse neuronal injury (Strich, 1956), individuals with focal cerebral injuries typically have focal lesions superimposed upon widespread neuronal damage. As observed by Levin, Eisenberg, Wigg, and Kobayashi (1982), the severity of diffuse brain injury, as opposed to the presence or lateralization of a focal lesion, appears to be the overriding determinant of the level of cognitive functioning. The duration of impaired consciousness, which is an indicant of the severity of diffuse brain injury, was the best predictor of cognitive functioning. In accord with previous studies, the duration of impaired consciousness was more strongly related to cognitive performance than was the GCS score (Ewing-Cobbs et al., in press; Levin et al., 1982; Levin et al., 1976).

Directions for Future Research

Neuropsychological deficits in head injured infants and preschoolers have been inadequately evaluated. Despite the potential differences in the brain's response to CHI at different ages, head injury provides a means of comparing acute deficits and long term recovery from brain injury across the lifespan. Using head injury as a model, issues related to cerebral plasticity may be addressed. Most studies of the effects of CHI on neuropsychological functioning have examined school-aged children, adolescents, and adults. It is likely that more

striking developmental differences will be apparent if younger children are included. For example, in the present study, the incidence of linguistic disturbance was much higher in children less than six years of age than that reported in older children or adults. In contrast to cerebrovascular accidents which occur infrequently in children, head injury occurs frequently in all age groups. Most cerebral injuries incurred during childhood are diffuse in nature; detailed evaluation of post-traumatic deficits may provide a model for studying recovery from diffuse brain injury.

Given the severity of neuropsychological deficits identified in this study, it is clear that young children need to be followed longitudinally. Presently, the degree to which the deficits resolve over time is unknown. Even if specific deficits resolve, residual impairment may adversely affect the development of more complex abilities. To illustrate, the vast majority of children who recover from acquired aphasia are unable to perform at age-appropriate levels scholastically. The effect of early CHI on later scholastic performance is unclear. Since scholastic achievement is a major developmental task awaiting each child, it is important to maximize each child's potential through periodic monitoring of cognitive status and remediation of neuropsychological deficits.

At the present time, there is no established means of evaluating the

severity of CHI in very young children. Given the high incidence of mutism in this age range, procedures such as the GCS, which evaluate verbal responses, may be inaccurate. Moreover, means of evaluating the behavioral responses of children too young to follow commands are not readily available.

To permit comparison across studies, a classification system for childhood aphasia needs to be developed. In addition to basic information on naming, repetition, written language, and the traditional indices of aphasic disturbance, the way in which cerebral insult affects the subsequent development of phonologic, syntactic, and lexical skills should be addressed.

References

Adams, J. H., Mitchell, D. E., Graham, D. I., & Doyle, D. (1977). Diffuse brain damage of the immediate impact type. Brain, 100, 489-502.

Alajouanine, T., & L'hermitte, F. (1965). Acquired aphasia in children. Brain, 88, 653-662.

Alexander, G. E., & Goldman, P. S. (1978). Functional development of the dorsolateral prefrontal cortex: An analysis utilizing reversible cryogenic depression. Brain Research, 143, 233-249.

Almli, C. R., & Finger, S. (Eds.). (1984). Early brain damage, volume 1: Neurobiology and behavior. New York: Academic Press.

Almli, C. R., Golden, G. T., & McMullan, N. T. (1976). Ontogeny of drinking behavior of preweanling rats with lateral preoptic damage. Brain Research Bulletin, 1, 437-442.

Annegers, J. F. (1983). The epidemiology of head trauma in children. In K. Shapiro (Ed)., Pediatric head trauma (pp. 1-10). Mount Kisco, NY: Futura.

Annegers, J. F., Grabow, J. D., Kurland, L. T., & Laws, E. R. (1980). The incidence, causes, and secular trends of head trauma in Olmstead County, Minnesota. Neurology, 30, 912-919.

Annett, M. (1973). Laterality of childhood hemiplegia and growth of speech and intelligence. Cortex, 9, 4-33.

Aram, D. M., Rose, D. M., Rekate, H. L., & Whitaker, H. A. (1983). Acquired capsular/striatal aphasia in childhood. Archives of Neurology, 40, 614-617.

Basser, L. S. (1962). Hemiplegia of early onset and the growth of speech with special reference to the effects of hemispherectomy. Brain, 85, 427-460.

Bastian, H. C. (1898). A treatise on aphasia and other speech defects. New York: Appleton.

Bawden, H.N., Knights, R. M., & Winogron, H. W. (1985). Speeded performance following head injury in children. Journal of Clinical and Experimental Neuropsychology, 7, 39-54.

Bayley, N. (1969). Manual for the Bayley Scales of Infant Development. New York: Psychological Corporation.

Becker, D. P., Miller, J. D., Greenberg, R. P., Young, H. F., & Skalas, R. (1977). The outcome from severe head injury with early diagnosis and intensive management. Journal of Neurosurgery, 47, 491-502.

Benjamin, R. M., & Thompson, R. F. (1959). Differential effects of cortical lesions in infant and adult cats on roughness discrimination. Experimental Neurology, 1, 305-321.

Black, P., Blumer, D., Wellner, A. M., & Walker, A. E. (1971). The head injured child: Time course of recovery, with implications for rehabilitation. Proceedings of an International Symposium on Head Injuries (pp. 131-137). Edinburgh: Churchill Livingstone.

Boll, T. J. (1978). Diagnosing brain impairment. In B. Wolman (Ed.), Diagnosis of mental disorders: A handbook. New York: Plenum.

Boll, T. J., & Barth, J. T. (1981). Neuropsychology of brain damage in children. In S. B. Filskov & T. J. Boll (Eds.), Handbook of clinical neuropsychology (pp. 418-452). New York: Wiley.

Bricolo, A., Turazzi, S., & Feriotti, G. (1980). Prolonged posttraumatic unconsciousness. Therapeutic assets and liabilities. Journal of Neurosurgery, 52, 625-634.

Brink, J. D., Garrett, A. L., Hale, W. R., Woo-Sam, J., & Nickel, V. L. (1970). Recovery of motor and intellectual function in children sustaining severe head injuries. Developmental Medicine and Child Neurology, 12, 565-571.

Brink, J. D., Imbus, C., & Woo-Sam, J. (1980). Physical recovery after severe closed head trauma in children and adolescents. Journal of Pediatrics, 97, 721-727.

Bruce, D. A., Raphaely, R. C., Goldberg, A. I., Zimmerman, R. A., Bilaniuk, L. T., Schut, L., & Kuhl, D. E. (1979). Pathophysiology, treatment, and outcome following severe head

injury in children. Child's Brain, 5, 174-191.

Burklund, C. W., & Smith, A. (1977). Language and the cerebral hemispheres. Neurology, 27, 627-633.

Carlsson, C. A., Von Essen, C., & Lofgren, J. (1968). Factors affecting the clinical course of patients with severe head injuries. Part 1: Influence of biological factors. Part 2: Significance of posttraumatic coma. Journal of Neurosurgery, 29, 242-251.

Caveness, W. F. (1977). Incidence of craniocerebral trauma in the United States, 1970-1975. Annals of Neurology, 1, 507.

Chadwick, O., Rutter, M., Shaffer, D., & Shrout, P. E. (1981). A prospective study of children with head injuries: IV Specific cognitive deficits. Journal of Clinical Neuropsychology, 3, 101-120.

Chadwick, O., Rutter, M., Thompson, J., & Shaffer, D. (1981). Intellectual performance and reading skills after localized head injury in childhood. Journal of Child Psychology and Psychiatry, 22, 117-139.

Chevrie, J. J., & Aicardi, J. (1978). Convulsive disorders in the first year of life: Neurological and mental outcome and mortality. Epilepsia, 19, 67-74.

Copeland, D. R., Fletcher, J. M., Pfefferbaum-Levine, B., Jaffe, N., Ried, H., & Maor, M. (in press). Long-term survivors of childhood cancer: Effects of CNS treatment. New England Journal of Medicine.

Cotard, J. (1868). Etude sur l'atrophie cerebrale. Paris: These.

Cotman, C. W., & Nieto-Sampedro, M. (1982). Brain function, synapse renewal, and plasticity. Annual Review of Psychology, 33, 371-401.

Craft, A. W. (1972). Head injury in children. In P. J. Vinken & G. W. Bruyn (Eds.)., Handbook of Clinical Neurology: Vol. 23 (pp. 445-458). New York: Elsevier.

Danoff, B., Cowchock, F., Marquette, C., Mulgrew, L., & Kramer, S. (1982). Assessment of long-term effects of primary radiation therapy for brain tumors in children. Cancer, 49, 1580-1586.

Davidson, P. W., Willoughby, R. H., O'Tuama, L. A., Swisher, C. N., & Benjamins, D. (1978). Neurological and intellectual sequelae of Reye's syndrome. American Journal of Mental Deficiency, 82, 535-541.

Day, P. S., & Ulatowska, H. K. (1979). Perceptual, cognitive, and linguistic development after early hemispherectomy: Two case studies. Brain and Language, 7, 17-33.

Denckla, M. B. (1979). Childhood learning disabilities. In K. M. Heilman & E. Valenstein (Eds.), Clinical neuropsychology (pp. 535-573). New York: Oxford University Press.

Dennis, M. (1980). Language acquisition in a single hemisphere: Semantic organization. In D. Caplan (Ed.), Biological studies of mental processes (pp. 159-185). Cambridge: MIT Press.

Dennis, M., Fitz, C. R., Netley, C. T., Sugar, J., Derek, C. F., Harwood-Nash, M. B., Hendrick, E. B., Hoffman, H. J., & Humphreys, R. P. (1981). The intelligence of hydrocephalic children. Archives of Neurology, 38, 607-615.

Dennis, M., & Lovett, M. (1981). Written language acquisition after left or right hemidecortication in infancy. Brain and Language, 12, 54-91.

Dennis, M., & Whitaker, H. A. (1976). Language acquisition following hemidecortication: Linguistic superiority of the left over the right hemisphere. Brain and Language, 3, 472-482.

Dikmen, S., Matthews, C. G., & Harley, J. P. (1975). The effect of early versus late onset of major motor epilepsy upon cognitive-intellectual performance. Epilepsia, 16, 73-81.

Dunn, L. M., & Dunn, L. M. (1981). Peabody Picture Vocabulary Test-Revised: Manual for forms L and M. Circle Pines, Minnesota: American Guidance Service.

Eiser, C. (1978). Intellectual abilities among survivors of childhood leukemia as a function of CNS irradiation. Archives of Diseases of Childhood, 53, 391-395.

Eiser, C. (1979). Irradiation and intellectual ability among children. In D. Osbourne, M. Gruneberg, & J. Eiser (Eds.), Research in

psychology and medicine (vol. 1). New York: Academic Press.

Ewing-Cobbs, L., Fletcher, J. M., Landry, S. H., & Levin, H. S. (in press). Language disorders after pediatric head injury. In J. Darby (Ed.), Neurological theory and speech and language disorders, vol. 2. San Diego: Grunne & Stratton.

Ewing-Cobbs, L., Fletcher, J. M., & Levin, H. S. (1985). Neuropsychological sequelae following pediatric head injury. In M. Ylvisaker (Ed.), Closed head injury rehabilitation: Children and adolescents (pp.71-89). San Diego: College Hill.

Fedio, P., & Mirsky, A. F. (1969). Selective intellectual deficits in children with temporal lobe or centrencephalic epilepsy. Neuropsychologia, 7, 287-300.

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

Finger, S., & Almli, C. R. (Eds.). (1984). Early brain damage, volume 2: Neurobiology and behavior. New York: Academic Press.

Finger, S., & Stein, D. G. (1982). Brain damage and recovery: Research and clinical perspectives. New York: Academic Press.

Flach, J., & Malmros, R. (1972). A long-term follow-up study of children with severe head injury. Scandanavian Journal of Rehabilitation Medicine, 4, 9-15.

Fletcher, J. M., Ewing-Cobbs, L., McLaughlin, E. J., & Levin, H. S. (1985). Cognitive and psychosocial sequelae of head injury in children: Implications for assessment and management. In B. Brooks (Ed.), The injured child (pp. 30-39). Austin: University of Texas Press.

Fletcher, J. M., Levin, H. S., & Landry, S. H. Behavioral consequences of cerebral insult in infancy. In C. R. Almli & S. Finger (Eds.), Early brain damage, volume 1: Research orientations and clinical observations (pp. 189-213). New York: Academic Press.

Fletcher, J. M., & Satz, P. (1983). Age, plasticity, and equipotentiality: A reply to Smith. Journal of Consulting and Clinical Psychology, 51, 763-767.

Fuld, P. A., & Fisher, P. (1977). Recovery of intellectual ability after closed head injury. Developmental Medicine and Child Neurology, 19, 495-502.

Gaidolfi, E., & Vignolo, L. A. (1980). Closed head injuries of school aged children: Neuropsychological sequelae in early adulthood. Italian Journal of Neurological Sciences, 1, 65-73.

Gardner, H. (1983). Frames of mind: The theory of multiple intelligences. New York: Basic Books.

Gibson, E. J., & Levin, H. (1975). The psychology of reading. Cambridge, Mass: MIT Press.

Goldberger, M.E. (1974). Recovery of movement after CNS lesions in monkeys. In D. G. Stein, J. R. Rosen, & N. Butters (Eds.), Plasticity and recovery of function in the central nervous system (pp. 149-174). New York: Academic Press.

Goldman, P. S. (1974). An alternative to developmental plasticity: Heterology of CNS structures in infants and adults. In D. G. Stein, J. R. Rosen, & N. Butters (Eds.), Plasticity and recovery of function in the central nervous system (pp. 149-174). New York: Academic Press.

Goldman, P. S., & Galkin, T. W. (1978). Prenatal removal of frontal association cortex in the fetal rhesus monkey: Anatomical and functional consequences in postnatal life. Brain Research, 152, 451-485.

Gott, P. S. (1973). Cognitive abilities following right and left hemispherectomy. Cortex, 9, 266-274.

Gramsberben, A., & IJkema-Paasen, J. (1984). The effects of early cerebellar hemispherectomy in the rat: Behavioral, neuroanatomical, and electrophysiological sequelae. In S. Finger & C. R. Almli (Eds.), Early brain damage, volume 2: Neurobiology and behavior (pp. 155-178). New York: Academic Press.

Gurdjian, E. S. (1971). Mechanisms of impact injury of the head. In Head injuries: Proceedings of an international symposium held in Edinburgh and Madrid (pp. 17-21). Edinburgh: Churchill Livingstone.

Guttman, E. (1942). Aphasia in children. Brain, 65, 205-219.

Hammock, M. K., Milhorat, T. H., & Baron, I. S. (1976). Normal pressure hydrocephalus in patients with myelomeningocele. Developmental Medicine and Child Neurology, 18, Supp. 37, 55-68.

Hebb, D. O. (1942). The effect of early and late brain injury upon test scores, and the nature of normal adult intelligence. Proceedings of the American Philosophical Society, 85, 265-292.

Hécaen, H. (1976). Acquired aphasia in children and the ontogenesis of hemispheric specialization. Brain and Language, 3, 114-134.

Hécaen, H., Perenin, M. T., & Jeannerod, M. (1984). The effects of cortical leisons in children: Language and visual functions. In C. R. Almli & S. Finger (Eds.), Early brain damage, volume 1: Research orientations and clinical observations (pp. 277-298). New York: Academic Press.

Hedrick, D. L., Prather, E. M., & Tobin, A. R. (1974). Sequenced Inventory of Communication Development examiner's manual. Seattle: University of Washington Press.

Heilman, K. M., Saffran, E., & Geschwind, N. (1971). Closed head trauma and aphasia. Journal of Neurology, Neurosurgery, and Psychiatry, 34, 265-269.

Heiskanen, O., & Kaste, M. (1974). Late prognosis of severe brain injury in children. Developmental Medicine and Child Neurology, 16, 11-14.

Hicks, S. P., & D'Amato, C. J. (1970). Motor-sensory and visual behavior after hemispherectomy in newborn and mature rats. Experimental Neurology, 29, 416-438.

Hirsch, J. F., Renier, D., Czernichow, R., Benveniste, L., & Pierre-Kahn, A. (1979). Medulloblastoma in childhood. Survival and functional results. Acta Neurochirurgica, 48, 1-15.

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

Hollingshead, A. B., & Redlich, F.C. (1958). Social class and mental illness. New York: Wiley.

Isaacson, R. L. (1975). The myth of recovery from early brain damage. In N. E. Ellis (Ed.), Aberrant development in infancy (pp. 1-26). Hillsdale, NJ: Erlbaum.

Jamison, D. L., & Kaye, H. H. (1974). Accidental head injury in children. Archives of Diseases of Childhood, 49, 376-381.

Jennett, B. (1972). Head injuries in children. Developmental Medicine and Child Neurology, 14, 137-147.

Jennett, B., Teasdale, G., Galbraith, S., Pickard, J., Grant, H., Braakman, R., Avezaat, C., Maas, A., Minderhoud, J., Vecht, C. J., Heiden, J., Small, R., Caton, W., & Kurze, T. (1977). Severe head injury in three countries. Journal of Neurology, Neurosurgery, and Psychiatry, 40, 291-298.

Johnson, D., & Almli, C. R. (1978). Age, brain damage, and performance. In S. Finger (Ed.), Recovery from brain damage (pp. 115-134). New York: Plenum.

Kennard, M. A. (1938). Reorganization of motor functions in the cerebral cortex of monkeys deprived of motor and premotor areas in infancy. Journal of Neurophysiology, 1, 477-497.

Kennard, M. A. (1942). Cortical reorganization of motor function: Studies on a series of monkeys of various ages from infancy to maturity. Archives of Neurology and Psychiatry, 48, 227-240.

Kerr, F. W. (1975). Structural and functional evidence of plasticity in the central nervous system. Experimental Neurology, 48, 16-31.

Kinsbourne, M. (1975). The ontogeny of cerebral dominance. Annals of the New York Academy of Sciences, 263, 244-250.

Klonoff, H., Low, M. D., & Clark, C. (1977). Head injuries in children: A prospective five year follow-up. Journal of Neurology, Neurosurgery, and Psychiatry, 40, 1211-1219.

Kohn, B., & Dennis, M. (1974). Selective impairments of visuospatial abilities in infantile hemiplegics after right cerebral hemidecortication. Neuropsychologia, 12, 505-512.

Kun, L. E., Mulhern, R. K., & Crisco, J. J. (1983). Quality of life in children treated for brain tumors: Intellectual, emotional, and academic function. Journal of Neurosurgery, 58, 1-6.

Lange-Cosack, H., Wider, B., Schlesner, H. J., & Kubicki, St. (1979). Prognosis of brain injuries in young children (one until five years of age). Neuropaediatrie, 10, 105-127.

Lenneberg, E. (1967). Biological foundations of language. New York: Wiley.

Levin, H. S., Benton, A. L., & Grossman, R. G. (1982). Neurobehavioral consequences of closed head injury. New York: Oxford University Press.

Levin, H. S., & Eisenberg, H. M. (1979a). Neuropsychological impairment after closed head injury in children and adolescents. Journal of Pediatric Psychology, 4, 389-402.

Levin, H. S., & Eisenberg, H. M. (1979b). Neuropsychological outcome of closed head injury in children and adoelscents. Child's Brain, 5, 281-292.

Levin, H. S., Eisenberg, H.M., Wigg, N. R., & Kobayashi, K. (1982). Memory and intellectual ability after head injury in children and adolescents. Neurosurgery, 11, 668-673.

Levin, H. S., Ewing-Cobbs, L., & Benton, A. L. (1984). Age and recovery from brain damage: A review of clinical studies. In S. W. Scheff (Ed.), Aging and recovery of function in the central nervous system (pp. 169-205). New York: Plenum.

Levin, H. S., Grossman, R. G., & Kelly, P. J. (1976). Aphasic disorder in patients with closed head injury. Journal of Neurology, Neurosurgery, and Psychiatry, 39, 1062-1070.

Levin, H. S., Madison, C. F., Bailey, C. A., Meyers, C. A., Eisenberg, H.M., & Guinto, F. C. (1983). Mutism after closed head injury. Archives of Neurology, 40, 601-606.

Mandleberg, I. A., & Brooks, D. N. (1975). Cognitive recovery after severe head injury 1. Serial testing on the Weschler Adult Intelligence Scale. Journal of Neurology, Neurosurgery, and Psychiatry, 38, 1121-1126.

McCarthy, D. (1972). McCarthy Scales of Children's Abilities. New York: Psychological Corporaton.

McFie, J. (1961). The effects of hemispherectomy on intellectual functioning in cases of infantile hemiplegia. Journal of Neurology, Neurosurgery, and Psychiatry, 24, 240-249.

Meadows, A. T., Massary, D. J., Fergusson, J., Gordon, J., Littman, P., & Moss, K. (1981). Declines in IQ scores and cognitive dysfunctions in children with acute lymphosytic leukaemia treated with cranial irradiation. Lancet, 2, 1015-1018.

Mealey, J. Jr. (1968). Pediatric head injuries. Springfield: Charles C. Thomas.

Mulhern, R. K., Crisco, J. J., & Kun, L. E. (1983). Neuropsychological sequelae of childhood brain tumors: A review. Journal of Clinical Child Psychology, 12, 66-73.

Nonneman, A. J., Corwin, J. V., Sahley, C. L., Vicedomini, J. P. (1984). Functional development of the prefrontal system. In S. Finger & C. R. Almli (Eds.), Early brain damage, volume 2: Neurobiology and behavior (pp.138-154). New York: Academic

Press.

O'Leary, D. S., & Boll, T. J. (1984). Neuropsychological correlates of early generalized brain dysfunction in children. In C. R. Almli & S. Finger (Eds.), Early brain damage, volume 1: Research orientations and clinical observations (pp. 215-232). New York: Academic Press.

O'Leary, D. S., Lovell, M. R., Sackellares, C., Berent, S., Giordani, B., Siedenberg, M., & Boll, T. J. (1983). The effects of age of onset of partial and generalized siezures on neuropsychological test performance in children. Journal of Nervous and Mental Disease, 171, 624-629.

O'Leary, D. S., Seidenberg, M., Berent, S., & Boll, T. J. (1981). Effects of age of onset of tonic- clonic seizures on neuropsychological test performance in children. Epilepsia, 22, 197-204.

Ommaya, A. K., & Gennarelli, T. A. (1974). Cerebral concussion and traumatic unconsciousness: Correlation of experimental and clinical observations on blunt head injuries. Brain, 97, 633-654.

Raimondi, A. J., & Tomita, T. (1979). Advantages of total resection of medulloblastoma and disadvantages of full head postoperative radiation therapy. Child's Brain, 5, 550-551.

Richardson, F. (1963). Some effects of severe head injury. A follow-up study of children and adolescents after protracted coma. Developmental Medicine and Child Neurology, 5, 471-482.

Robinson, R. O. (1981). Equal recovery in child and adult brain? Developmental Medicine and Child Neurology, 23, 379-382.

Rosenzweig, M. R., Bennett, E. L., & Alberti, M. (1984). Multiple effects of lesions on brain structures in young rats. In S. Finger & C. R. Almli (Eds.), Early brain damage, volume 2: Neurobiology and behavior (pp. 49-72). New York: Academic Press.

Rowland, J., Glidewill, O., Sibley, R., Holland, J. C., Brecker, M., Tull, B., Berman, A., Glicksman, A., Forman, E., Harris, M., McSweeny, J., Jones, B., Black, M., Cohen, M., & Freeman, A. (1982). Effect of cranial radiation on neuropsychological function in children with acute lymphocytic leukemia. American Society of Clinical Oncology Abstracts, 23, 123.

Rudel, R. G., Teuber, H. L., & Twitchell, T. E. (1974). Levels of impairment of sensorimotor functions in children with early brain damage. Neuropsychologia, 12, 95-108.

Rutter, M. (1981). Psychological sequelae of brain damage in children. American Journal of Psychiatry, 138, 1533-1544.

Rutter, M. (1982). Developmental neuropsychiatry: Concepts, issues and problems. Journal of Clinical Neuropsychology, 4, 91-115.

Rutter, M., Chadwick, O., & Shaffer, D. (1983). Head injury. In M. Rutter (Ed.), Developmental neuropsychiatry (pp. 83-111). New York: Guilford.

St. James-Roberts, I. (1979). Neurological plasticity, recovery from brain insult, and child development. In H. W. Reese & L. P. Lipsett (Eds.), Advances in child development and behavior (vol. 14) (pp. 253-319). New York: Academic Press.

Sarno, M. T. (1980). The nature of verbal impairment after closed head injury. Journal of Nervous and Mental Disease, 168, 685-692.

Satz, P., & Bullard-Bates, C. (1981). Acquired aphasia in children. In M. T. Sarno (Ed.), Acquired aphasia (pp. 399-426). New York: Academic Press.

Satz, P., Fletcher, J. M., Clark, W., & Morris, R. (1975). Lag, deficit, rate, and delay constructs in specific learning disabilities. In A. Ansara, N. Geschwind, A. Galaburda, M. Albert, & N. Gartrell (Eds.), Sex differences in dyslexia. New York: Orton Dyslexia Society.

Schneider, G. E., & Jhavari, S. R. (1974). Neuroanatomical correlates of spared or altered function after brain lesions in the newborn hamster. In D. G. Stein, J. J. Rosen, & N. Butters (Eds.), Plasticity and recovery of function in the central nervous system (pp. 65-110). New York: Academic Press.

Shaffer, D., Bijur, P., Chadwick, O., & Rutter, M. (1980). Head injury and later reading disability. Journal of the American Academy of Child Psychiatry, 19, 592-610.

Siegel, A. W., Bisanz, J., & Bisanz, J. L. (1983). Developmental analysis: A strategy for the study of psychological change.

Contributions to Human Development, 8, 53-80.

Simons, D., & Finger, S. (1983). Neonatal vs. later sensorimotor cortex damage: The ability to use spared fragments of target tissue to guide tactile learning. Physiological Psychology, 11, 29-34.

Smith, A. (1974). Dominant and nondominant hemispherectomy. In M. Kinsbourne & W. L. Smith (Eds.), Hemisphere disconnection and cerebral function (vol. 2) (pp. 5-33). Springfield: Charles C. Thomas.

Smith, A. (1981). On the organization, disorganization and resolution of language and other brain functions. In Y. Lebrun & O. L. Zangwill (Eds.), Lateralization of language in the child. Lisse: Swets.

Smith, A., & Sugar, O. (1975). Development of above normal language and intelligence 21 years after left hemispherectomy. Neurology, 25, 813-818.

So, K. F., & Schneider, G. E. (1976). Abnormal recrossing retinotectal projections after early lesions in Syrian hamsters: A critical age effect. Anatomical Record, 184, 535-536.

Spear, P. D. (1984). Consequences of early visual cortex damage in cats. In S. Finger & C. R. Almli (Eds.), Early brain damage, volume 2: Neurobiology and behavior (pp. 229-252). New York: Academic Press.

Spreen, O., & Benton, A. L. (1969). Neurosensory Center Comprehensive Examination for Aphasia: Manual of directions. Victoria, B. C.: Neuropsychology Laboratory, University of Victoria.

Strauss, E., & Verity, C. (1983). Effects of hemispherectomy in infantile hemiplegics. Brain and Language, 20, 1-11.

Strich, S. J. (1961). Shearing of nerve fibers as a cause of brain damage due to head injury: A pathological study of twenty cases. Lancet, 2, 443-448.

Taylor, H. G. (1984). Early brain injury and cognitive development. In C. R. Almli & S. Finger (Eds.), Early brain damage, volume 1: Research orientations and clinical observations (pp. 325-346). New York: Academic Press.

Taylor, H. G., Michaels, R. H., Mazur, P. M., Bauer, R. E., & Liden, C. B. (in press). Cognitive, achievement, and behavioral outcomes six to eight years post-H influenzae meningitis.

Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. Lancet, 2, 81-84.

Terman, L. M., & Merrill, M. A. (1972). Stanford-Binet Intelligence Scale manual for the third revision. Boston: Houghton Mifflin.

Teuber, H. L., & Rudel, R. (1962). Behavior after cerebral lesions in children and adults. Developmental Medicine and Child Neurology, 4, 3-20.

Tsang, Y. C. (1937). Maze learning in rats hemidecorticated in infancy. Journal of Comparative Psychology, 24, 221-254.

Tucker, T. J., & Kling, A. (1967). Differential effects of early and late lesions of frontal granular cortex in the monkey. Brain Research, 5, 377-389.

van Dongen, H. R., & Loonen, M. C. B. (1977). Factors related to prognosis of acquired aphasia in children. Cortex, 13, 131-136.

Villablanca, J. R., Burgess, J. W., & Sonnier, B. J. (1984). Neonatal cerebral hemispherectomy: A model for postlesion reorganization of the brain. In S. Finger & C. R. Almli (Eds.), Early brain damage, volume 2: Neurobiology and behavior (pp. 179-210). New York: Academic Press.

Whishaw, I. Q., & Kolb, B. (1984). Behavioral and anatomical studies of rats with complete or partial decortication in infancy. In S. Finger & C. R. Almli (Eds.), Early brain damage, volume 2: Neurobiology and behavior (pp. 117-137). New York: Academic Press.

Woods, B. T. (1980). The restricted effects of right- hemisphere lesions after age one: Wechsler test data. Neuropsychologia, 18, 65-70.

Woods, B. T., & Carey, S. (1979). Language deficits after apparent clinical recovery from childhood aphasia. Annals of Neurology, 6, 405-409.

Woods, B. T., & Teuber, H. L. (1978). Changing patterns of childhood aphasia. Annals of Neurology, 3, 273-280.

Wright, L. (1978). A method for predicting sequelae to meningitis. American Psychologist, 33, 1037-1039.

Zimmerman, R. A., Bilaniuk, L. T., Bruce, D., Dolinskas, C., Obrist, W., & Kuhl, D. (1978). Computed tomography of pediatric head trauma: Acute general cerebral swelling. Radiology, 126, 403-408.