# A NEUROPSYCHOLOGICAL TYPOLOGY OF ADULT OFFENDERS:

# A DEVELOPMENTAL/PREVENTATIVE PERSPECTIVE

by

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A Neuropsychological Typology of Adult Offenders A Developmental/Preventative Perspective

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ABSTRACT

An expanded Halstead Reitan Neuropsychological Test Battery was administered to 584 adult offenders (OF), 132 normal controls (NC), and 494 acute psychiatric patients (PP). Subjects were between 18 and 44 years of age. Age effects are analyzed and normative data for all measures are provided. Factor analysis of a subset of measures yielded five factors interpreted as: 1) Sensory Motor Spatial Perceptual Organization; 2) Nonverbal Perceptual Reasoning and Abstraction; 3) Fine Temporal Perceptual Motor Speed; 4) Dynamic Verbal Processing; and 5) WCST Perseveration. Separate Modal Profile Analyses of factor scores (4 profiles) and Wechsler scale scores (4 profiles) produced an empirical typology of neuropsychological functioning. Impairment analyses indicate substantial neuropsychological impairment in the OF and PP groups, especially on measures of executive function and verbal capacities. The neuropsychology of the prefrontal cortex and of Attentional Deficit Hyperactivity Disorder and Conduct Disorder is reviewed. This review, in the context of the present findings, supports a working hypothesis that the executive deficits and behavioral disinhibition of offenders represent heterotypic continuities of brain dysfunction among some children with disruptive behavior disorders. Implications for research, the potential of neuropsychology for offenders, and the early identification and remediation of high risk children are discussed.

# DEDICATION

To Darlene, Deedre, & Eric

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# PREAMBLE

In a particularly candid report to the Canadian House of Commons, the Standing Committee on Justice and the Solicitor General, under the Chairpersonship of Bob Horner (1993) Member of Parliament, noted that the Canadian Justice System is inherently inadequate. Specifically, it noted that the conventional crime control model:

1. Fails to cope with the actual quantity of crime . . . .
2. Fails to identify many criminal offenders and bring them to justice . . . .
3. Fails to rehabilitate those offenders who are identified by the justice system . . . .
4. Fails to address the underlying factors associated with crime and criminality. (p. 1)

The Committee also examined the cost of crime in Canada. It concluded that in 1989-90 the services of the Canadian Criminal Justice System cost $7.7 billion. Further, it noted that incarcerating another 7,000 individuals would incur capital costs of $1 billion and operating yearly costs of $300 million thereafter. Not included in the costs of maintaining the Criminal Justice System are costs resulting from property loss and damage. For example, the Insurance Bureau of Canada estimated that residential, commercial, and automobile thefts alone cost insurance companies $2 billion a year.

The Committee also noted that many of the most severe losses from crime cannot be quantified in monetary terms as these impose physical,

emotional, and psychological damage on victims. As well, in innumerable ways, crime affects the quality of life of all citizens.

In view of their findings the Committee observed that "threats to the safety and security of Canadians will not be abated by hiring more police officers and building more prisons" (p. 2), and concluded that the collective response to crime must shift to prevention efforts. In this regard they proposed that the focus should increasingly be directed towards "at-risk young people and on the underlying social and economic factors associated with crime and criminality" (p. 2).

In their review of witnesses' accounts of factors associated with crime they heard many views and concluded:

> These accounts of the conditions that contribute to crime and criminality make clear that there is no single root cause of crime. Rather it is the outcome of the interaction of a constellation of factors that include: poverty, physical and sexual abuse, illiteracy, low self-esteem, inadequate housing, school failure, unemployment, inequality, and dysfunctional families. (pp. 11-12)

The Committee's observation that the pathways to criminality are diverse and multifactorial is clearly congruous with the consensus in the scientific literature. As well, the Committee noted that a minority of male offenders are responsible for the majority of all crimes committed. Moreover, they note that this minority of persistent offenders typically began their offending early in life. It is also reasonable, in a Bayesian probabilistic

sense, to believe that most persistent offenders have been affected by the adverse factors noted by the Committee as contributory to crime, yet the great majority of individuals do not become persistent offenders despite in many instances extremely deleterious environmental conditions. It would thus seem critical to identify critical variables that discriminate the vulnerables from the less vulnerable.

Towards this endeavor, a number of researchers have stressed that biological factors have been largely ignored, yet they may be key. More specifically, theorists have proposed that subtle brain dysfunctions and anomalies associated with the prefrontal cortex and the dominant hemisphere may prove to be especially critical risk factors in the development of persistent criminality, either directly or through an interactive or transactional association with familial, social, and environmental factors.

In neuropsychology, owing to the revolutionary advances in neuroscience, theories regarding brain-behavior relationships have undergone dramatic transformation and development. For instance, in the past decade, views as to the role and importance of the prefrontal cortex in development have traversed a course from essentially unclear and largely ignored, to a central focus of attention in neuropsychology and general agreement that its role is pervasive and central to psychological and social development.

In view of these advances, it seems reasonable to hypothesize that subtle, though increasingly measurable, brain dysfunctions could be critically involved in the development of persistent criminality. To the degree that

such variables were uncovered, it would permit the elaboration of preventative programs targeted specifically at those children whose relatively silent deficits, when young, place them at great risk in later years, particularly when combined with adverse environmental circumstances. Such an approach could be expected to have considerably greater yield than broad approaches which, for instance, must await the elimination of poverty. Accordingly, one purpose of this study is to offer a reasoned opinion concerning the potential of such an approach.

In view of the enormous personal, social, and economic costs associated with criminality, it is imperative that the proverbial neuropsychological "stone be turned" in the search to identify high risk children and that ways be found to reassign their developmental trajectory. As Boll (1985) put it: "All disorders will not prove to produce neuropsychological mischief. Some will. Which disorders, what mischief, and what remedy represents the frontier of neuropsychological health care at least for the rest of this century" (p. 484).

# OVERVIEW

## Introduction

Antisocial behavior is generally assumed to be a result of the interplay of many factors and influences. Consistent with this view, a biopsychosocial developmental perspective has been advanced as essential to understanding, preventing, predicting, and managing antisocial behavior. Within such a framework, theorists and researchers have proposed that neuropsychological functioning may play a key role in both the development and persistence of antisocial behavior patterns. Accordingly, the main objectives of this research have been to evaluate the relevance of neuropsychological theories for understanding persistent criminality and to develop an empirical neuropsychological typology of adult offenders, including normative data. The following material reviews the theoretical considerations, empirical findings, and general conclusions/implications of the present study.

## Neuropsychological Theory/Literature Review

Neuropsychological theories of antisocial behavior emphasize that neuropsychological deficits, particularly those associated with the prefrontal cortex compromising executive function, and dominant hemisphere deficits compromising verbal abilities, may present as significant risk factors for the development of persistent antisocial behavior patterns. More recently, such theories have been extended to account for some of the symptoms and behaviors of young offenders and children diagnosed as having Attention

Deficit Hyperactivity Disorder and Conduct Disorder. These groups have been shown to be at substantial risk for developing antisocial disorders as adults.

The evaluation of neuropsychological theories began by examining: a) theoretical perspectives on the role of the prefrontal cortex in adults; b) the effects of lesions of the prefrontal cortex in adults on both cognitive functioning and personality; and c) the role of the prefrontal cortex in early development by presenting theoretical analyses, associated research, and case studies of children who sustained early damage to the prefrontal cortex.

On the basis of literature reviewed in the above areas, it was concluded that theorists accorded the prefrontal cortex, a supraordinate controlling, monitoring, and integrating function with respect to cognitive, emotional, and behavioral processes. Further, they emphasized its specific importance for executive function and the elaboration of internal language as a basis for self-regulation.

Analysis of the prefrontal lesion literature, including experimental studies, defined a broad range of cognitive deficits primarily associated with dorsolateral lesions and disinhibitory affective phenomena associated with orbital-limbic lesions.

The main cognitive sequelae included deficits in working memory, abstract thinking, verbal and design fluency, cognitive flexibility, conditional associative learning, recency and frequency estimations, planning, strategy

application, hypothesis generation, memory for temporal order, problem solving, and passive avoidance.

Lesions of the orbital-limbic area of the prefrontal cortex produce a distinct syndrome characterized by some combination of the following symptoms: diminished emotional self-control, dramatic personality changes, reduced self-reflective awareness, indifference to emotional feelings or conflicts, affective disorder, increased sexual and aggressive drive disinhibition, increases in impulsive and antisocial behaviors, and reduced tolerance to alcohol.

Recent work has demonstrated that the prefrontal cortex plays a role throughout development beginning in infancy. Human infants have been shown to have a limited capacity as early as 6 to 12 months of age to develop internal representations and delay responding, thus permitting deliberate behavior choices. This early capacity has been described as the cornerstone of cognitive development and it is seen as the foundation of working memory. Several studies have noted that the development of prefrontal capacities occurs as a multistage process between infancy and middle adolescence, at which time adult-like performance is the norm.

Case studies demonstrate that early damage to the prefrontal cortex can result in a severe impact on the development of executive function, behavioral self-regulation, as well as personality and social development.

Noting that the behavioral deficits exhibited by persistent adult offenders imply cognitive and personality deficits commonly observed in

patients with prefrontal damage, several investigators have administered neuropsychological measures to a variety of criminal groups including psychopaths. Reviews of these studies have generally concluded that the findings are inconclusive with respect to psychopaths, and that studies of other criminal groups that produced positive findings, require replication.

Several researchers have focused on the behavioral disinhibition or impulsivity of offenders and have proposed that dysfunction of the prefrontal cortex may be common to offenders and prefrontal patients. Overall, it is noted that few, if any studies, can be considered to have investigated a broad range of neuropsychological abilities among a wide spectrum of offenders.

As well, Attention Deficit Hyperactivity Disorder has been reconceptualized over the past two decades from an aggregate of symptoms of inattention, impulsivity and over-activity to a spectrum disorder reflecting dysfunction of the prefrontal cortex. In particular, prefrontal dysfunction impacting executive function, working memory, and behavioral inhibition have been postulated. Recently a major theorist in this area has postulated that a primary deficit in delayed responding, mediated by the prefrontal cortex, can account for most of the diverse manifestations of the disorder. He has further suggested that much of the deficits in self-regulation noted in this disorder relates to a lack of development of internal language, also considered to be primarily a prefrontal function. Prefrontal dysfunction,

especially resulting in behavioral disinhibition and verbal deficits, have also been attributed to children with Conduct Disorder and to young offenders.

In sum, it has been proposed that some persistent offenders are characterized by neuropsychological deficits typically observed in patients with prefrontal and/or dominant hemisphere damage. Similarly, it has been noted that children with Attention Deficit Hyperactivity Disorder, Conduct Disorder, or both disorders, are at a disproportionate risk for later developing antisocial disorders, also often present with neuropsychological deficits. To determine to what extent neuropsychological deficits mediate a negative outcome within these diagnostic groupings will require further research. The present empirical study was designed to examine the presence of neuropsychological deficit within a large,diverse sample of adult offenders.

## An Empirical Typology of Adult Offenders

### Measures/Subjects

An expanded Halstead-Reitan Neuropsychological Test Battery supplemented by several other neuropsychological tests emphasizing verbal and memory abilities and the Wechsler Intelligence Scales was administered to 584 adult offenders (OF), 132 normal controls (NC), and 494 acute psychiatric patients (PP). All subjects were between 18 and 44 years of age. The offender group consisted of 584 North American male Caucasians (mean age 28.78 years) serving prison terms in excess of two years and admitted to the Regional Psychiatric Centre (Pacific) in Abbotsford, British Columbia between September 1978 and July 1986.

Analysis

a) Age Effects

Analyses were carried out to examine whether differences in neuropsychological test performance could be attributed to age across the three groups of subjects: OF, NC, and PP. Overall, in these analyses, it was found that age-group test performance correlations were not significant on most measures. Significant relationships were as follows: a) within the NC group, there was a tendency for psychomotor abilities and verbal working memory to decline between 18 and 44 years of age, although this trend was not as evident in the OF and PP groups; and b) within the OF group, the younger group was noted to be more proficient on tasks requiring perceptual motor speed and spatial perceptual organization, and this group had lower scores on some Wechsler verbal scales. Offender normative tables are provided by age-group for all measures.

b) Group Performance Contrasts

The relative performance of the NC, OF, and PP groups was compared across all measures. The most remarkable finding was that for almost all test performance measures, excepting some simple perceptual, sensory, and motor measures, the performance of the NC group was significantly higher than that of the OF group, and in turn, the OF group performed significantly better than the PP group. The overall level or degree of impairment was also noted to be substantial. Among the OF group, e.g., between 30% and 57% performed 1 *SD* below the NC mean and between 9% and 30% performed

2 *SD*'s below this mean depending on the neuropsychological measure considered. For Wechsler variables, relative levels of impairment were even greater, e.g., performance of the OF group was 1 *SD* below the NC group mean in 75%, 60%, and 78% of cases on Verbal IQ, Performance IQ, and Full Scale IQ, respectively. Corresponding percentages 2 *SD*'s below the NC mean were 46%, 26%, and 45%.

Among the PP group the degree of impairment was exceptional. Across all of the neuropsychological measures examined, between 44% and 80% scored $\leq 1$ *SD* below the control mean and between 21% and 55% scored $\leq 2$ *SD*'s below this mean.

On the Wechsler Full Scale IQ composite measure, 85% of the PP group scored $\leq 1$ *SD* below the control mean and 59% scored $\leq 2$ *SD*'s below this mean.

On the basis of group contrasts across, it is evident that offenders are characterized by substantial levels of impairment, though not as severe as the impairment levels observed among the psychiatric patients.

c) Factor Analyses

Factor analysis of a representative set of neuropsychological variables was conducted. A five factor Principal Components followed by Varimax solution was interpreted.

Factor 1 was interpreted to reflect Sensory Motor Spatial Perceptual Organization and this factor was especially marked by high loadings from the

Tactual Performance Test. These abilities have traditionally been inferred to relate primarily to non-dominant temporal and parietal systems.

Factor II, labelled Nonverbal Perceptual Reasoning and Abstraction, was mainly defined by performance on the Halstead Category Test. The implicit task requirements of this factor mainly include nonverbal perceptual abstraction, logical analysis, and the capacity to adapt flexibly to feedback of changing problem definition. Considered from the perspective of neuropsychological theory, the Category Test represents an exemplar of executive function in the nonverbal perceptual sphere.

Factor III was especially marked by loadings of Purdue Pegboard measures. Task analysis indicates that a capacity to flexibly resolve the timing of sensory motor relationships is required. The factor was labelled Fine Temporal Perceptual Motor Speed. Research has suggested that systems for performance on this task are likely widely distributed, but the prefrontal cortex may contribute to the development and coordination of these systems.

The fourth factor was labelled Dynamic Verbal Processing. Tests loading on this factor reflect the capacity to sustain attention (e.g., the Speech Sounds Perception Test), but also flexibly shift attention (e.g., the Trails B test and Wisconsin Card Sorting Test), although all tests that implicate verbal abilities load on this factor. Overall, verbal processing is involved and, neuropsychologically, frontal verbal-attentional and posterior verbal systems appear to be mainly involved.

The fifth factor labelled WCST Perseveration was fairly exclusively defined by overall success on the WCST and especially, perseverative errors on this test. The WCST is factorially complex and success on this measure requires a number of distinguishable abilities, e.g., the formation of verbal concepts, maintaining and shifting set. Thus, this factor appears to isolate the capacity to shift set and respond according to a different principle, once information is provided that the previous response is inconsistent with the 'now' operative set. Research reviewed in Chapter III suggests that the dorsolateral prefrontal cortex may be critically implicated, although damage elsewhere may also interfere with this capacity to shift set and change behavioral response.

In sum, five interpretable factors accounting for nearly 60% of the neuropsychological test variance represented were obtained.

d) Modal Profile Analysis

Modal Profile Analysis was applied to the five neuropsychological factors. The results of this analysis indicated that 100% of subjects could be classified in terms of four bipolar profiles. The first profile especially emphasized differential abilities along Factor II, Nonverbal Perceptual Reasoning and Abstraction. The second profile distinguished groups with relatively high/low WCST perseveration in contrast to relatively low/high verbal processing abilities. Profile 3 classified a group characterized by relatively high Sensory Motor Spatial Organization (Factor I) combined with poor dynamic verbal processing abilities (Factor IV) and a group with a

converse ability profile. The fourth profile reflected a group with remarkably high (in relative terms) Fine Temporal Perceptual Motor Speed (Factor III) and above average abilities across the other four factors. The negative pole of this profile reflected a converse ability profile.

A Modal Profile Analysis was also conducted on the Wechsler subtest scales. A four profile solution classifying 90% of subjects was selected as most informative and interpretable. The first profile identified two groups of subjects whose performance among the Wechsler performance subtests was highly variable. One group was characterized by relatively high performance on the Picture Completion and Picture Arrangement subtests compared to relatively low performance on the Block Design and Object Assembly subtests. The other group had a converse pattern. The ability to verbally mediate visual spatial relations was hypothesized to discriminate the two groups. The second profile identified a group characterized by relatively high verbal attention (Digit Span and Arithmetic) and relatively low motor dexterity (Object Assembly and Digit Symbol), and a second group with the opposite performance pattern. The third profile reflected divergent relative performance on measures of simple attention and psychomotor ability (Digit Span and Digit Symbol) with acquired verbal abilities (Information, Vocabulary, and Comprehension). The fourth Wechsler profile generally discriminated subjects with differential visuospatial organizational abilities (Picture Completion, Picture Arrangement, Block Design, and Object Assembly) and verbal abilities (Information, Vocabulary, Arithmetic,

Comprehension, and Similarities). This profile thus reflected groups with large Verbal IQ - Performance IQ splits.

Classification analysis indicated, that unlike the case with neuropsychological profiles, there were associations between Wechsler profile type and group (NC, OF, and PP) membership. The most notable association was relative to Profile 4- which reflects high performance on visuo-spatial abilities relative to verbal and psychomotor abilities. This profile was uncommon among the NC group, but 28.7% of the OF group and 24.6% of the PP group were classified into this profile.

The last portion of the analysis examined the intersection of neuropsychological and Wechsler profiles. It was observed that any given Wechsler profile combines in relatively uniform proportions with all the neuropsychological profiles. Thus, classification on Wechsler profiles does not predict neuropsychological profile subtypes and vice versa. In other words, the information provided by the two classification systems is relatively independent and additive or cumulative. By way of illustration, examples of profile intersects were discussed. Neuropsychologically, it was suggested that the neuropsychological profiles tend to reflect executive and adaptive functioning, emphasising prefrontal cortical function, while the Wechsler profiles are more related to temporal/parietal brain function.

In sum, an offender's overall neuropsychological profile can be characterized by the conjunction of his neuropsychological and Wechsler profiles.

Conclusions

1. The results of this large scale study indicate that, as a group, serious adult offenders are characterized by a high level of neuropsychological impairment. Three types of impairment are especially observed. These include: deficits in executive function, deficits in verbal capacities, and, a third type with significant impairment across a broad range of capacities.

2. Taking into account the differentiation by type and level of impairment, it is appropriate to invoke heuristic models of brain dysfunction to account for the observed pattern of deficits. In particular, prefrontal dysfunction, dominant hemisphere dysfunction, and diffuse dysfunction appear to be particularly relevant heuristic constructs.

3. Considering the nature of the neuropsychological impairment observed in this study, neuropsychological theories of disruptive behavior disorders and outcome studies of children with such disorders, it is proposed, as working theory, that it is reasonable to view the impairment of offenders as heterotypic continuities of brain dysfunction among some children with disruptive behavior disorders.

Treatment and Research Implications

1. The finding that offenders, as a group, are characterized by significant levels of neuropsychological impairment implies that neuropsychology may provide a scientific basis contributing to the understanding, treatment, and management of offenders. Similarly,

neuropsychological factors may be particularly relevant for the prevention and prediction of recidivism.

2. A related implication is that offenders handicapped by significant neuropsychological deficit may benefit from psychological, medical, and social assistance designed to ensure a positive re-adjustment to society. In the absence of such programming, poor adjustment and significant recidivism should be anticipated.

3. A further implication is that neuropsychological procedures may be particulary valuable in the identification and treatment of children at high risk to develop antisocial behavior disorders.

4. Further research is necessary to replicate the empirically recovered neuropsychological typology of this study and to provide normative comparisons at the subtype level. As well, research is required to establish both the internal and external validities of the identified subtypes, including the impact of neuropsychological factors on developmental trajectories. Finally, it is recommended that research designed to explore prefrontal orbital dysfunction in offenders should be attributed a high priority.

# CHAPTER I INTRODUCTION

## SECTION I ORGANIZATION OF THE INTRODUCTION

This research is primarily concerned with delineating the neuropsychological characteristics of offenders and associated neuropsychological mechanisms. Practical considerations relate to the relevance of neuropsychology for clinical intervention, correctional programming for offenders, early identification and remediation of children at high risk for later offending, and general maladaptive adjustment due to neuropsychological factors.

Section II of this introduction briefly describes the aims of the present research. In Section III, by way of background to this neuropsychological study of offenders, the Neuropsychiatric and Psychopathy traditions are discussed. Section IV is concerned with the rationale for applying clinical neuropsychological methods to the evaluation of offenders. In Section V, classification and definitional problems are noted to have hindered progress in neuropsychiatric research. Taxonomic issues are considered to be the focal problem addressed by the present research, in particular, the need to develop a neuropsychological typology of offenders is emphasized. A multivariate, multidimensional approach is contrasted with the bivariate approach which is characteristic of much of the research on psychopathy. Section VI identifies certain limitations and the scope of the present research, while Section VII discusses the purpose or motivation underlying

this research. Lastly, Section VIII provides a summary of the overall organization of the dissertation.

## SECTION II AIMS OF THE RESEARCH

This research has several objectives, some theoretical, and some practical, and which are of varying breadth. Overall, the purpose is to evaluate neuropsychological theories that have been proposed to be relevant to persistent criminality. A related objective will be to assess the potential benefit of conceptualizing neuropsychological involvement in terms of heuristic models of brain function. Another general aim will be to assess the implications of the empirical findings for offender assessment and treatment, and provide some further directions for research. In a broader context, the potential role of neuropsychological methods in preventative programming will be examined.

The empirical objectives relate to the analysis of neuropsychological test data collected on a large sample of serious adult offenders (OF) and contrasts of these results with samples of normal controls (NC) and acute psychiatric patients (PP). Specifically, the main empirical objective of this research is to specify the extent and type of brain dysfunction observed in the OF sample, especially by:

1. Providing normative data for the neuropsychological measures included in the study for the OF, PP, and NC samples.

2. Developing an empirical typology for each of the three samples, OF, PP, and NC, based on their respective test performance using Modal Profile Analysis (MPA).

3. Comparing and contrasting both the normative data and the empirically derived modal profiles across the OF, PP, and NC groups.

This research is directed towards a neuropsychological classification of a broad spectrum of serious adult offenders and is anchored in two traditions of research directed at subgroups of offenders primarily characterized by violent and/or persistent antisocial behavior. By way of providing some background to the present approach, these two traditions are briefly discussed below.

## SECTION III BACKGROUND

### The Neuropsychiatric Perspective

Elliott (1978), writing from a neuropsychiatric perspective concluded that, "Almost all of the clinical features of the psychopath can be produced by physical disorders of the brain" (p. 146). Within the neuropsychiatric tradition, the focus has especially been on relationships between EEG abnormalities and aggression, beginning with the EEG studies by Hill and Watterson (1942) and Hill and Pond (1952). Other studies have examined EEG variables and syndromes of episodic violence or dyscontrol (e.g., Bach-y-Rita, 1975; Bach-y-Rita, Lion, Climent, & Ervin, 1971; Bach-y-Rita & Veno, 1974; Blumer, Williams, & Mark, 1974; Elliott, 1978, 1982; Ervin, 1969; Goldstein, 1974; Lion & Penna, 1974; Monroe, 1970, 1978; Shah &

Roth, 1974; Sherwin, 1980; Sweet, Ervin, & Mark, 1969; Williams, 1969a, 1969b, 1975).

Capitalizing on improved brain imaging technology, investigators within this tradition have focused on neuroanatomical and metabolic anomalies associated with pathological aggression (e.g., Volkow & Tancredi, 1987). Weiger and Bear (1988) have proposed a neurological model of aggression delineating syndromes associated with dysfunction determined by the hierarchical position of the neural structure affected. Recent summaries of the neuropsychiatric perspective on pathological aggression have been provided by Elliot (1990) and Pincus and Lewis (1991). The neuropsychiatric perspective, despite its primary focus on aggression and its typical restriction to hospitalized patients, has provided an impetus for more systematic investigation of brain dysfunction in offender populations. Its technologies are seen as complementary to potential clinical neuropsychological approaches and no doubt the importance and relevance of this window on brain function would be clarified by a taxonomy of clinical neuropsychological deficits by permitting a more precise focus on particular types of neuropsychological dysfunctions among subgroups of offenders.

Research on Psychopathy

The second tradition relates to the concept of psychopathy. It has an extensive history and focuses on psychological factors stressing impairments in affective or emotional reactivity which lead to an inability to profit from experience, to view oneself as others do and to achieve "true and abiding

loyalty to any principle or any person" (Cleckley, 1976, p. 375). A related perspective on antisocial behavior considers defective socialization as fundamental. This conceptualization was formally set out by Gough (1960). The central tenet of his analysis was that antisocial individuals lack the ability to view themselves as a social object within a social context. As the concept of psychopathy will be examined in some detail in Section V, the objective here is simply to indicate that recently there has been considerable interest in identifying subtle autonomic differences which could, with reference to animal learning and social learning models, account for the observed psychological and socialization deficits manifested by some offenders. As witness to the increased interest in the biological substrate of psychopathy, several sophisticated reviews have recently been published (e.g., Hare & Schalling, 1978; Jeffrey, 1979; Mednick, Moffit, & Stack, 1987; Mednick & Volavka, 1980; Moyer, 1976).

Recently, a notable shift in psychopathy research has involved a reinterpretation of many of the earlier studies of autonomic deficit in psychopaths as due, not specifically to autonomic deficits per se, but attributable to prefrontal cortical dysfunctions that result in increased impulsivity and behavioral disinhibition. Examples include, for instance, Gorenstein's (1991) cognitive reinterpretation of this research and Hare (1979), Hare and McPherson's (1984), as well as Hare and Jutai's (1988) focus on differences in the linguistic processing of psychopaths.

The premise of the present research, while recognizing the contributions of the neuropsychiatric and psychopathy approaches to our understanding of antisocial behavior, proposes that a multivariate classification of the neuropsychological characteristics of a representative sample of offenders would provide an important qualifying dimension for focusing specific studies of psychopathy and/or aggression. The rationale for a neuropsychological approach is further discussed below.

## SECTION IV HISTORICAL CONSIDERATIONS

### Application of Neuropsychological Methods to Offenders

Several investigators have proposed that neuropsychological conceptualizations and heuristic models of brain function may in certain instances complement our understanding of an offender's behavioral patterns and personality characteristics, particularly in cases where the antisocial behavior pattern is persistent and appears pathological (Buikhuisen, 1989; Gorenstein, 1991; Hall & McNinch, 1988; Miller, 1987; Moffitt, 1993a, 1993b; Yeudall, 1977; Yeudall, Fedora, & Fromm, 1986).

The potential relevance of neuropsychology for populations such as offenders is due to advances in the understanding of brain systems/ behavior relationships which extend far beyond the original focus in clinical neuropsychology on identifying variables sensitive to brain lesions of particular location, type, and status (Benton, 1987, 1994; Reitan, 1994). Boll (1985), Chelune and Moehle (1986), Costa (1983, 1988), and Puente (1992) have documented remarkable progress in clinical neuropsychology

which they attribute to the clinical sensitivity of neuropsychological tools and the strategic interface of clinical neuropsychology with the neurosciences, behavioral sciences, clinical medicine, and applied psychology.

Rourke (1982) analysed the development of neuropsychology in terms of three interrelated and progressive phases. During the first phase, with its peak between 1945 and 1965, the main concern was with diagnosis. Relating performance on standardized tests to the presence of documented cerebral lesions was vital. Neuropsychological tests were judged during this period by their ability to localize cerebral lesions.

During the late 60's and early 70's, the focus, influenced by developments in cognitive psychology, shifted to the analysis of the cognitive structure of neuropsychological tests. The role of the clinician also evolved beyond diagnostics to include delineation of the cognitive/behavioral deficits associated with a particular lesion.

This second phase was also marked by the publication of major conceptual models of brain-behavior relationships, e.g., Luria's, " Higher Cortical Functions in Man" (1966); Pribram's, "Languages of the Brain" (1971); and Sperry's work with split-brain patients in (Sperry, Gazzaniga, & Bogen, 1969) provided new insights into the differential contribution of the left and right hemispheres in processing information. Thus, gradually, the empirical brain-behavior relationships established during the first phase were incorporated within broader conceptualizations of brain function.

Rourke (1982) has termed the third and present mode "dynamic neuropsychology" (p. 3). The emphasis is on the developing brain and its interaction with an "individual's approach to material-to-be-learned" (p. 3). While Rourke's (1982) analysis of the current phase in neuropsychology certainly remains applicable today, Rourke and Del Dotto (1994) stress that "we currently understand much more about developmental neuropsychological-performance interactions than we do about developmental neuropsychological-performance interactions" (p. 13). Still, the greatest developments in clinical neuropsychology in the past decade surely relate to discoveries and transformations in conceptualizing the role and functions of the prefrontal cortex in adults and its critical developmental importance.

Chelune and Moehle (1986) have also noted a transformation of the role of clinical neuropsychology to include the interaction between an individual's neuropsychological functioning, personal dynamics and the environmental context. According to Chelune and Moehle, the questions presently asked of neuropsychologists go beyond neurodiagnosis and delineation of cognitive/behavioral strengths and weaknesses to require prescriptive advice and decisions, e.g., Should John return to work? or Will psychotherapy be effective for Tom?.

In effect, questions concerning everyday potential for interpersonal, social, educational, vocational and behavioral adjustment are key issues to which the clinician must respond. Neuropsychological tests have been

successfully utilized to predict: quality of life in patients with closed head injury (Heaton & Pendleton, 1981; Klonoff, Costa, & Snow, 1986); the future adjustment of epileptics while still in high school (Dodrill & Clemmons, 1984); the future employment status of diverse patients referred for neuropsychological testing (Heaton, Chelune, & Lehman, 1978); the everyday life functioning in chronically ill persons (McSweeny, Grant, Heaton, Prigatano, & Adams, 1985); and the prognosis in treatment of alcoholics (Parsons, 1994). These examples attest to the sensitivity of neuropsychological tests to predict a variety of outcomes across several impaired populations. For instance, Townes et al. (1985) have developed a neuropsychological classification system for psychiatric patients to evaluate their general competence irrespective of their psychiatric diagnosis.

Boll (1985) has also identified a trend within neuropsychology toward a more "psychological" rather than neurodiagnostic emphasis. He sees "neuropsychologists providing with increasing sophistication, psychological descriptions designed to assist in understanding the whole person" (p. 474). He also states that "neuropsychological investigations are no longer solely oriented towards the diagnosis of brain damage nor can neuropsychological examinations be seen as appropriate only for those patients with clear and obvious neurological diseases and disorders" (p. 474). The suggestion is, that for many individuals who experience psychological or behavioral disorders, neuropsychological dysfunction may form part of the etiology or the symptom complex with therapeutic implications.

In effect, a wide diversity of clinical groups in addition to neurological patients have been investigated neuropsychologically. A common rationale for conducting such neuropsychological studies is to determine the neuropsychological consequences and the cognitive, behavioral and psychological sequelae of various known or suspected insults to the brain. Examples include studies evaluating the effects of mild head injury (e.g., Barth et al., 1983) and the effects of environmental toxins (e.g., Needleman et al., 1979; Reidy, Bowler, Rauch, Pedroza, 1993; Yeates & Mortensen, 1994).

Another class of studies which is primarily correlational in design examines the neuropsychological functioning of groups of individuals characterized by psychological or behavioral disorders. While the specific rationale may vary from study to study, the objective is typically to relate cerebral dysfunction to the etiology, symptoms, treatment or progression of the disorder. Thus, there is a burgeoning neuropsychological literature on psychiatric disorders (e.g., Franke et al., 1993; Heaton, Baade, & Johnson, 1978; Kolb & Whishaw, 1983), learning disabilities (e.g., Pennington, 1991; Rourke, 1985; Rourke & Del Dotto, 1994), Attention Deficit Hyperactivity Disorder (e.g., Hinshaw, 1994a; Moffitt, 1990), Conduct Disorder (Hinshaw, 1994b; Moffitt, 1993a; Moffitt & Lynam, 1994), as well as many other diagnostic groupings.

The potential relevance of neuropsychology for understanding the development of a host of disorders and conditions has been accelerated by

relatively recent advances in understanding the ubiquitous role of the prefrontal cortex and its critical role in psychological development. Developments in brain imaging, e.g., Regional Cortical Blood Flow (rCBF), Positron Emission Tomography (PET), and Magnetic Resonance Imaging (MRI) studies are increasingly permitting a neural systems approach to delineating relationships between cognitive and emotional functions and brain structured activation indices (cf. Parks, Crockett, & McGeer, 1989; Ruff et al., 1989). In particular, as temporal and spatial resolution increases and methodology becomes more sophisticated, the dynamic participation of different brain regions can be examined on cognitive tasks with greater precision. For instance, Bench et al. (1993) demonstrated that prefrontal cortical activation, was accompanied by reciprocal inhibition of bilateral parietal structures during performance of the Stroop task. Thus, a more dynamic evaluation of neuropsychological function is presaged.

Heuristic Models in Neuropsychology

Tucker and Derryberry (1992) have noted that the dominant metaphor for the mind in cognitive science is no longer the linear computer-like cognitive processor, but the neural net model involving distributive processing with the capacity to integrate cognitive and emotional information and act adaptively. Thus, heuristic models of neuropsychological functioning rather than dichotomous approaches designed to test for brain damage in different groups are seen to be potentially more productive. Support for using heuristic models comes from investigators of neurological populations,

such as Stuss (1992) who stated that "There is a growing awareness that frontal lobe mental processes may be described as psychological constructs rather than as anatomically localized functions" (p. 9). He further noted by way of example that "individuals who have suffered a head injury will exhibit significant dysfunction in 'frontal abilities', but attribution to the frontal lobes exclusively or even primarily is not possible or necessary . . . the dysfunctions appear to be real; the underlying pathophysiology, however, is uncertain (p. 9). There is considerable support for investigating clinical groups neuropsychologically, even if the presence or absence of brain damage per se may not be the main issue, although the measures employed have typically been validated in neurological samples with known lesions.

The research to be described in this dissertation involves the application of neuropsychological tests to a group of offenders. These measures have typically been found to discriminate or localize brain damage in neurological patients with an acceptable degree of sensitivity and specificity. To establish neuropsychological test validity, two methods have been proposed: double dissociation and reciprocal disability (cf. Jones, 1983). In the former method, subjects are grouped on the basis of known lesions (type and/or location) and group membership is found to predict reciprocal impairments in test performance. Further, it is concluded that particular functions are characteristically sustained at known neural locations. In the method of reciprocal disability, group membership is based on reciprocal impairments in test performance, a relationship is subsequently demonstrated between

impairments and lesion type and/or location, and it is concluded that particular functions are sustained at particular neural locations. In the present study, the design could be framed within the reciprocal disability method, however, the critical step of verifying that a lesion is in fact present is not possible and hypotheses regarding damage or dysfunction will thus only be made inferentially.

Another critical interpretive issue that will be addressed in this study is the significance that can be attached to a particular level of test dysfunction. The interpretive significance of similar neuropsychological test performance across different clinical groups cannot be assumed to be equivalent. However, the domain of neuropsychological performance tapped by existing neuropsychological measures would seem to be an efficient point of entry in examining the neuropsychological characteristics of a clinical group such as offenders. This rationale has been implicit or advanced in other studies and populations noted earlier which have imported the methods of neuropsychology in studying a variety of clinical groups and behavioral conditions.

## SECTION V STATEMENT OF THE PROBLEM

Increasingly, the need for a broad biopsychosocial perspective in understanding and remediating the development of severe aggression and antisocial behavior is being advocated, (e.g., Ellis and Hoffman, 1990; Farrington, 1987; Fishbein, 1990; Hinshaw, 1994b; Hinshaw & Anderson, in press; Loeber et al., 1993; Mednick, 1977a, 1977b; Nachshon, 1982;

Trasler, 1978, 1987; Weiner & Wolfgang, 1989; Yeudall, 1977; Yeudall, Fedora, & Fromm, 1986).

Fishbein (1990) succinctly emphasized that antisocial behavior is multiply determined and that traditional approaches ignoring biological factors have resulted in, at best, an incomplete approach:

> For several decades, mainstream criminology has been dominated by sociological and political perspectives. Although findings from these fields must not be discarded or underplayed, considered alone, they do not offer a complete assessment of the contributions to criminal behavior. Data currently being generated from numerous behavioral sciences, such as behavioral genetics, physiological psychology, psychopharmacology, and endocrinology, indicate that biological factors play an equally significant role in the development of antisocial behavior and should be considered accordingly. Incorporation of the theoretical parameters and findings of these behavioral sciences into a criminological framework would yield valuable information regarding processes underlying antisocial behavior. Such a multidisciplinary approach is likely to enhance capabilities to predict, prevent, and manage antisocial behavior. (p. 27)

The inherent complexity of the issue implies a need for multi-causal models. Models which must incorporate the developmental interplay of psychobiologic, psychological, familial, socioeconomic, and sociocultural

factors. Essentially, calling for a paradigm shift from bivariate to multivariate research, Hinshaw and Anderson (in press) predicted:

> Investigations across multiple areas of interest will derive increasing benefit from important work on subtyping; prospective studies of antisocial behavior from extremely early development (e.g., infancy) will come of age; designs explicitly incorporating integration of biologic, psychosocial, and wider systems context will become de rigeur; and large-scale clinical trials will be recognized not only for their importance clinically, but also for their ability to yield causal inferences about underlying psychopathologic mechanisms. Work in the field is maturing at a rapid pace, but in light of the havoc wreaked by persistent antisocial behavior patterns, progress is achingly slow. There can be cause for optimism only if ideologic rancor gives way to concentrated scientific efforts aimed at understanding and reducing aggression and antisocial activity. (p. 35)

It has thus become increasingly clear that further understanding of psychopathological disorders and conditions will only be efficiently achieved by relating their development and evolution to data, including developmental data from other domains, such as the neurosciences, e.g., neurochemistry, behavioral genetics, neuropsychology, and the psychosocial behavioral domains.

In the domain of psychopathology the need for multidimensional classification schemes has been noted for some time, and hotly debated as the categorical versus dimensional issue, (e.g., Blashfield & Draguns, 1976; Livesley, Schroeder, Jackson, & Jang, 1994; Widiger & Trull, 1991). Both Livesley et al. (1984) and Widiger and Trull (1991) have noted a long term trend increasing with each edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) toward a more dimensional approach. The lack of a dimensional classification system in defining major psychiatric disorders has been noted to be a major impediment to the development of psychiatric genetics (cf. Ciaranello & Ciaranello, 1991).

A similar debate has also occurred with respect to defining childhood psychopathology. For example, Quay, Routh, and Shapiro (1987) have suggested criteria for the elaboration of any future syndromes. They proposed that such constructs, which can subsume both the notions of categorical and dimensional data, should be discriminable from other syndromes and be reliably diagnosed or measured. Furthermore, they should be associated with different causes, outcomes or respond to different interventions. Other authors in the childhood and adolescent psychopathology area have also demonstrated that categorical versus dimensional approaches are not mutually exclusive, indeed, integration of the two approaches should improve both clinical communication and scientific analysis (e.g., Achenbach, 1993; Cantwell & Baker, 1988; Shekim et al., 1986).

The co-morbidity problem, which is seen to obfuscate the interpretation of research results in childhood psychopathology studies (e.g., Achenbach, 1993; Hinshaw, Lahey, & Hart, 1993; Shaywitz & Shaywitz, 1991) may be, at least partially resolved, by an integrated categorical/dimensional approach. In such a system, subtypes within broader spectrum disorders could be identified and provide for much finer discriminations for research and clinical usage. Considerable emphasis on this approach is evident in some learning disability research, e.g., Pennington (1991), Rourke (1991), and Rourke and Del Dotto (1994). A pertinent illustration of the benefits of such an approach is provided by a study conducted by Pennington, Groisser, and Welch (1993). These authors have demonstrated at least three distinct subgroups of children within their sample of hyperactive children. One group was characterized only by phonological processing deficits; another by executive function deficits; and a third group by both types of deficits. The implication of etiology and for treatment were suggested to be different for each of the three groups.

Another area where subtyping is critical and hampering progress is in tracing the trajectory of Attention Deficit Hyperactivity Disorder (ADHD), Conduct Disorder (CD), and learning disordered children who traverse very different 'antisocial' developmental courses; some relatively benign; some of an apparent persistent nature. A classification system including subtyping would likely greatly facilitate our understanding of the risks and interventions required by such children. (cf. Faraone et al., 1993; Loeber, 1991; Loeber et

al., 1993; Lynam, Moffitt, Stouthamer-Loeber, 1993; Moffitt, 1990; Moffitt, 1993b; White et al, 1994).

Lastly, with respect to the definition of Antisocial Personality Disorder (APD) and psychopathy, Sutker (1994) has pointed out that a basic consensus as to the unitary nature of these constructs is lacking. Conceptual argument regarding the unitary or homogeneous nature of psychopathy has provoked sometimes intense and at other times, acrimonious, debate. For example, Blackburn (1988) has reflected on the scientific basis for psychopathy as a construct and concluded that it represented a mythical entity encompassing a variety of deviant personalities. As such, he questioned its value as a focus for theory, research, or clinical intervention. There is also a danger pointed out by Gunn and Robertson (1976) that as an all encompassing term, there are clearly dangers that labelling someone as a psychopath essentially could lead to moralizing and be prejudicial to their opportunity to receive treatment.

This is not to diminish the advances in the operalization of the construct of psychopathy which have taken place over the past 15 years. Most notable, in this respect, is the work of Hare and his co-workers with the Psychopathy Checklist (e.g., Hare, 1980; Hare, 1985; Hare & Harpur, 1986; Hare et al., 1990; Harpur, Hakstian, & Hare, 1988). Within prison populations, this checklist does identify particularly serious antisocial individuals (Harris, Rice, & Quincey, 1994) and the instrument has been shown to have some capacity to contribute to the prediction of criminal

recidivism, e.g., Hart, Kropp, and Hare (1988). However, even those accepting the notion of psychopathy as a disease class have sought to qualify its various phenomenological variants or subtypes. Consider the following review by Thomas-Peter (1992):

> There is a long history associated with the attempt to be more precise and definitive about the sub-classification of 'psychopathy'. Cleckley (1976), Hare and Cox (1978) and Roth (1990) have reviewed some of the early attempts to classify 'psychopathic' patients. Henderson (1939) suggested that there is the Inadequate, Unusual and Creative; Karpman (1941) distinguished between Inadequate, Unusual, Eccentric, Creative and the Sociopathic; Karpman (1955) suggested the Aggressive-predatory and the Passive-parasitic; Arieti [1957; referred to by Widom (1973)] distinguished between the Pseudo-psychopathic and the True psychopathic types, with four sub-types of the latter. These were the Simple, Complex, Dysocial and Paranoic; Craft (1966) suggested Immature, Inadequate and Vicious. (p. 337)

Sutker (1994) commented on psychopathy as a problematic construct ". . . . because the disorder, if it is such, is of unknown, partially unknown, and multifactorial, multidimensional causation" (p. 109). She also suggests that theories regarding mental deficit in psychopathy, including neuropsychological theories, have been limited by the heterogeneity of the disorder as she states:

> Though some appealing hypotheses have been presented, implicating motivational and fear or anxiety systems, learning capabilities or systems, and cognitive processing systems, the findings from the laboratory cannot be integrated adequately into meaningful theoretical positions because of the failure of scientists to build consensus about the particular or specific behavioral constellation or syndrome under scrutiny. Examining the work of one investigator in relation to another takes on the complexion of comparing apples and oranges. (p. 107)

In sum, addressing a problem as complex as antisocial behavior requires a solid anchoring of variables within a broad operational matrix. In most instances this means that classification schemes within domains must be developed and related to one another. Furthermore, developmental dimensions must be incorporated across the span of domains represented. In a sense, the view is that a prior multivariate operationalization of constructs must precede bivariate experimentation. Thus, in the most general sense, state laws precede process law, that is, one must be able to assess someone's current status before determining how he/she became that way. A multivariate, multidimensional classification system is required. The objectives of this study pale in the face of such a mammoth task, yet even the longest voyage begins with a single step. In this instance, improving upon neuropsychological taxonomy is the goal.

Neuropsychological Taxonomy/Typology

To achieve many of the aims of the present research, e.g., specifying the nature and degree of neuropsychological dysfunction in the offender sample, evaluating the similarities and differences between the offender, normal control, and psychiatric samples or evaluating neuropsychological theories in relation to the empirical findings, it will first be necessary to establish a classification scheme delineating the neuropsychological characteristics of the groups represented.

In psychology, the use of formal taxonomic methods and strategies is relatively new. The pioneering work of Sneath and Sokol (1973) in the development of numerical taxonomies has been elaborated and a generalized psychological classification methodology has been proposed by Skinner (1977, 1979, 1981). The applicability of Skinner's model to the classification of human performance has been emphasized by Fleishman, Quaintance, and Broedling (1984) and emphasized for neuropsychology by several authors, e.g., Adams (1985), Morris and Fletcher (1988). Empirically, the model has been used extensively in the validation of neuropsychological subtypes of learning disabilities among children (e.g., Rourke, 1985, 1991; Pennington, 1991), but also in other domains, such as the personality domain (Skinner & Jackson, 1978; Jackson, 1989).

Skinner's (1981) classification model includes three dimensions; a theory formulation component, internal and external validity. The theory formulation component involves the selection of measures and the rationale

for their inclusion, as well as hypotheses as to the specific groups that exist. The internal validity component relates to the reliability, domain sampling, and replicability of the classification. The external validity facet primarily relates to the capacity of the classification scheme to predict external variation in other domains, such as etiology, treatment response, diagnostic variables and so on.

Skinner (1978, 1981) and Skinner and Shue (1982) have proposed Modal Profile Analysis (MPA) as a most suitable analytic model. The model locates relatively homogeneous subsets of individuals by identifying individuals with similar coordinates in multidimensional space. The coordinates of an individual in this space are the shape of the individual's profile. Severity or magnitude and deviation from the target homogeneous clusters of individuals are determined through profile elevation.

The homogeneous subsets so derived, or modal types, have been defined by Skinner (1981) as an ideal type representing "a hypothetical pattern of attributes" (p. 72) that is characteristic of an individual or subgroup of individuals in a population. In the study described here, it is hypothesized that a relatively small number of groups can be defined in terms of distinct patterns of neuropsychological performance, though differing in elevation.

The domain sampling in this study is captured by a large neuropsychological test battery and represents the wisdom of a half century of research in defining critical brain function variables chosen to reflect

cognitive, perceptual, motor, and sensory neuropsychological abilities. (For a detailed description of the measures included in this study see Chapter III, Section III).

In sum, from a classification perspective, it is hypothesized that among the individuals represented in this study, a small number of subgroups will be identified which share distinct patterns of neuropsychological performance.

## SECTION VI LIMITATIONS/SCOPE OF THE PRESENT STUDY

The present research is designed to examine and contrast the neuropsychological capacities of large groups of offenders, psychiatric patients, and normal participants. Despite generally large sample sizes, generalizability issues remain with respect to all three groups. Although the relative equivalence of the reference normative data utilized in this study on a subset of variables common to the broad normative system developed by Heaton, Grant, and Matthews (1991) inspires confidence, it seems clear that more large scale normative studies are still required. With regard to the offender sample included, it is not possible to ascertain the variety of selection biases that brought them for assessment and/or treatment to the institution in which they were assessed, and while no gross factors emerge to suggest systematic bias, there is no substitute for probabilistic sampling and thus, issues of generalizability remain, as does the need for replication. Similar considerations apply to the psychiatric sample, however, in this instance, although difficult to quantify, it is believed that they represent the more difficult and seriously mentally ill cases given the reputation of the

facility in which they were assessed for accepting cases which psychiatric wards in general hospitals would be most inclined to send to a more specialized facility. Questions of generalizability thus clearly arise with respect to this group and the degree of neuropsychological deficit is almost certainly greater than that observed among psychiatric patients generally.

The clinical neuropsychological measures administered to participants in this study represent a broad and fairly inclusive/representative array of instruments available to clinical neuropsychologists. However, a review of the measures and techniques presently being examined experimentally foretells of a new generation of clinical instruments, and by those standards, the current tests used may appear limited (cf. Heinrichs, 1990). A particular area of difficulty lies in the development of clinical instruments which will capture deficits associated with dysfunction of the orbital prefrontal regions of the brain. Unfortunately, clinical neuropsychology has not focused until very recently on the measurement of orbital prefrontal functions, although theoretical views as to its role would suggest that this area may be an area of exceptional relevance for understanding the development of offenders and their behavior. This is seen as a particularly regrettable limitation of the present study.

The potential significance of the findings in this study would be increased if empirically recovered neuropsychological subtypes could be related to other diagnostic methods of ascertaining and localizing brain damage or dysfunction. Unfortunately, the clinical functional findings of this

study cannot be related to more precise, though sometimes invasive methods of documenting brain anomalies at a physiologic or anatomical level. It is thus limited to explication at the level of functional constructs, but some encouragement is taken in the knowledge that progress in taxonomy should facilitate more crucial studies of the interrelationship of functional with structural/metabolic indices.

The focus of this research is on developing a taxonomy/typology and normative indices of neuropsychological functioning. However, developing a taxonomy/typology in one domain begs research to relate such a classification scheme to taxonomies or critical variables in other domains, including developmental parameters, psychiatric diagnosis, measure of outcome, and so on. Should the present study identify certain subsets of individuals characterized by relatively clear-cut functional patterns, it may provide an impetus for more specific retrospective or prospective studies. The limitations and restrictions discussed above will temper and require qualification of findings, still, the hope is that this research will engender enthusiasm for mapping taxonomy and developing typologies of individual differences in various domains thus forming a basis for more refined theoretical investigations and ultimately improved and more scientifically based therapeutic interventions.

As can be inferred from the above, the scope of the present study is, in the main, limited to the variables included and the groups contrasted. There is throughout, an emphasis on prefrontal function and its developmental

aspects. Also pertinent, of course, are social, familial, dynamic, and neurochemical influences, but these considerations have been generally taken to be beyond the scope of this study. As well, highly pertinent issues of therapeutic intervention have not been addressed in any detail, although all of the above areas are relevant to the understanding of antisocial behavior and its prevention. Still, the present research will hopefully contribute to an integrated and balanced understanding of antisocial behavior and its neuropsychological substratum.

## SECTION VII PURPOSE/MOTIVATION FOR THIS RESEARCH

This research has two primary objectives. The first objective is to develop a neuropsychological typology of offenders. In effect, the aim is to identify subtypes of offenders who are characterized by common patterns of neuropsychological function and dysfunction. The second objective is to assess the implications of the typology from a developmental/preventative perspective. Specifically, the question is: do particular adult neuropsychological profile types imply particular developmental neuropsychological difficulties which in retrospect can reasonably be seen to have contributed to a maladaptive life course? If so, a further question relates as to whether early identification and intervention could have ameliorated the situation.

Regarding the first objective, practical motivations for developing a neuropsychological classification system relate to the ability of neuropsychological assessment to improve clinical input into the assessment,

treatment, and correctional management of offenders. Clinically, if the population of offenders is characterized by a high level of neuropsychological impairment, consideration of neuropsychological functioning should improve clinical assessment, including a better understanding of offenders' developmental history and their current behavioral patterns. Moreover, it should assist in defining treatment needs and optimize intervention strategies. At a broader level, this should result in efficiencies related to the identification of groups of offenders with common treatment needs and the development of specifically tailored treatment programming. For instance, a specific benefit of including neuropsychological status in overall clinical assessment relates to the potential to examine its relationship with therapeutic response to psychiatric medications, thus contributing to the optimization of psychiatric interventions. Finally, a typology delineating neuropsychological strengths and dysfunctions provides a succinct scheme for communication among professionals, but it also can provide an effective basis from which to provide consultations to correctional and parole personnel.

While the more immediate motivation for this research is to improve clinical service delivery to offenders, a further aim is to provide a basis upon which to conduct more in depth and refined neuropsychological investigations. For instance, what is the significance of the empirically derived subtypes; how do they relate to a host of factors such as other indices of brain function, or social, behavioral, learning, and personality

variables? Other questions relate to general therapeutic implications and the potential for specific remediation of specific subtypes. Perhaps of greatest interest and relevance are questions related to the impact of the subtypes, viewed developmentally, on the evolution of persistent antisocial behavior patterns. Moreover, where the impact is thought to have been significant; what are the prospects for early identification of the problematic subtypes, as well as the implementation of compensatory and remedial programming?

Clearly, this research cannot offer definitive responses to questions related to the developmental impact or potential for early remediation/compensation of various childhood neuropsychological deficits. However, neuropsychological theory and studies which bear on these issues are reviewed, discussed, and an attempt is made to relate developmental neuropsychological theory and research to the findings of this study.

## SECTION VIII ORGANIZATION OF THE DISSERTATION

This section concludes Chapter I. Its purpose has been to present the general aims of this research and to discuss historical approaches to offenders that have examined brain function in offenders. Further, the rationale for employing the methods of clinical neuropsychology with offenders was examined and the development of a methodology to classify offenders in terms of their neuropsychological capacities was set out as the study's primary objective. Limitations and scope of the present research as well as the purposes for conducting this research were also discussed.

Chapter II, Literature Review, is concerned especially with three bodies of literature:

1. Literature regarding the role of the prefrontal cortex. This is considered from three perspectives, theoretical views, the effects of prefrontal lesions, and the role of the prefrontal cortex viewed developmentally.

2. Literature which relates either to the application of neuropsychological theory to account for the behavior of offenders or empirical studies of their neuropsychological characteristics.

3. Theoretical and empirical neuropsychological literature regarding children and adolescents with disruptive behavior disorders who present a disproportionate risk for becoming offenders. This chapter concludes with a summary.

Chapter III, Method:

1. Identifies the participants in this research and outlines testing and data collection procedures.

2. Provides a description and review of the psychometric properties of each of the neuropsychological tests employed in this research.

3. Describes the study's main hypotheses, methodology, analyses, and ethical considerations.

Chapter IV, Results, describes the results of analyses conducted. In particular:

1. Age effects on neuropsychological performance is analyzed.

2. Norms are presented for each neuropsychological measure included in the battery.

3. Impairment levels in the normal controls, offenders, and psychiatric patient samples are contrasted.

4. Selected neuropsychological tests are factor analysed.

5. Modal profiles were derived and samples are classified across neuropsychological factors and Wechsler scales.

6. A summary of findings is provided.

Chapter V, Discussion and Conclusion, reviews and discusses the main findings of this research, identifies priorities for further research. Clinical implications for adult offenders, young offenders, and children at high risk for maladaptive behavior are considered. Lastly, conclusions are summarized.

# CHAPTER II LITERATURE REVIEW

## Introduction

This research is concerned with the neuropsychological status of adult offenders viewed developmentally. The main empirical aim is to develop a neuropsychological typology of offenders, thus providing a method whereby offenders can be succinctly classified according to their neuropsychological strengths and weaknesses. The theoretical impetus stems largely from theorists and researchers who have proposed that neuropsychological deficits, particularly those associated with the prefrontal cortex compromising executive function and the left hemisphere compromising verbal abilities, may present significant risk factors for the development of persistent antisocial behavior. Several authors have also noted that childhood diagnoses of Attention Deficit Hyperactivity Disorder (ADHD) and Conduct Disorder (CD) present a significant risk factor for the development of later antisocial behavior. Other authors have hypothesized that neuropsychological deficits may be a common factor linking ADHD and CD to persistent forms of adolescent offending and ultimately to adult offending.

The hypotheses enumerated above define the required scope for the literature review supporting the present research. In particular, proposals implicating the prefrontal cortex in offending require as complete an understanding as possible of its role, both in adults and its function throughout childhood and adolescent development. Review of this literature comprises the first three sections of this review.

Section I focuses on theories regarding the role of the prefrontal cortex in adulthood. Section II examines the effects of lesions of the prefrontal cortex in adults from a theoretical perspective. This is followed by a discussion of research findings related to the impact of prefrontal lesions on cognitive and personality function. Section III addresses the early developmental role of the prefrontal cortex. Recent theoretical positions are discussed, experimental findings reviewed, and illustrative cases demonstrating the impact of early prefrontal lesions are presented.

The remainder of this literature review is concerned with the application of neuropsychological theories and mechanisms to adult offenders and groups of children and adolescents at risk for becoming serious adult offenders. As well, the developmental outcome of these groups is examined. Specifically, in Section IV, literature relating theories of prefrontal dysfunction to adult offenders and associated research findings are discussed. In Section V, neuropsychological theories of ADHD, CD, and adolescent offenders, as well as related research findings, are presented. This is followed by a review of studies examining the adolescent and adult outcome of ADHD and CD. Lastly, Section VI provides a summary of the topics reviewed in this chapter.

## SECTION I ROLE OF THE PREFRONTAL CORTEX IN ADULTHOOD

### Introduction

This section begins by sketching historical problems in localizing the functions of the prefrontal cortex. Next a brief overview of the anatomy of

the prefrontal cortex is presented and this is followed by a discussion of theoretical perspectives and a brief summary of these concludes this section.

Perspectives On The Prefrontal Cortex

Localization of brain function has been a major experimental focus over the past century (Philips, Zeki, & Barlow, 1984). In this context, establishing the role of the prefrontal cortex has been particularly challenging and remains an area of active speculation (Weinberger, 1993). Mesulum (1986) stated that "few subjects in neurology have presented with such enigma and paradox as the behavioral affiliates of the prefrontal cortex" (p. 320). Benton (1991a), in his historical review of the prefrontal cortex considered the description of the distinctive composition of the prefrontal cortex, e.g., Brodmann's maps and the anatomical connections of the prefrontal cortex with other regions, particularly with the dorsomedial nucleus of the thalamus to be major contributions. He noted that by 1950, animal experimentation indicated that prefrontally injured animals did indeed exhibit distinctive cognitive defects and alterations of personality, but neither the behavioral descriptions nor the explanations proposed received wide acceptance. At the clinical level he noted that a vast array of diverse behavioral deficits of a cognitive, affective, and interpersonal nature had been described in association with disease of the prefrontal region, but that the "mixture of deficits was far too variegated to permit the formulation of a satisfactory description in terms of one or two basic disabilities" (pp. 25-26).

Despite this, he observed that the term 'frontal lobe syndrome' had been adopted.

Benton (1991a) considered recent advances, particularly since 1970, to have been so radical and far reaching as to justify the designation of developments prior to 1950 as early history. Goldman-Rakic (1984) described recent developments in tracing neuronal connections, electrophysiological analysis of neuronal activity, neurochemical characterization (especially of its selective dopamine input), and advances in brain imaging as encouraging. Moreover, research elucidating the development of psychological processes associated with the early development of the prefrontal cortex suggests potential for a better understanding of psycho-developmental processes (cf. Goldman-Rakic, 1987a, 1987b).

Accordingly, the goal of this section, further to an introductory overview of the anatomy of the prefrontal cortex, is to present, though necessarily in a partial and selective, though, hopefully, representative way, the perspectives of several prominent investigators and theorists in this area. Next, literature relating to the cognitive and personality sequelae associated with human prefrontal lesions will be considered. This, in turn, will be followed by a discussion of recent experimental findings with human subjects and a brief overview of the effects of neurological diseases which affect the prefrontal cortex. Further to this, views and findings relating to the prefrontal cortex in early development and outcomes associated with early damage to the

prefrontal cortex will be discussed. Lastly, a summary and conclusions concerning the material reviewed in this section will be presented.

Overview Of The Anatomy Of The Prefrontal Cortex

Traditionally, the human frontal cortex has been divided into three principal regions: the precentral cortex, the prefrontal cortex and the limbic cortex. The prefrontal cortex makes up most of the frontal cortex and encompasses the pole of the lobe. It is anterior to both the precentral and premotor regions of the frontal lobes.

The prefrontal cortex has been defined as that portion of the cortex that receives projection fibres from the mediodorsal nucleus of the thalamus. The arrangement of projections to thalamus is quite discrete; the orbital aspect is linked with the pars magnocellularis and the dorsolateral aspect with pars parvocellularis. In terms of Brodmann's cytoarchitectural designations, the prefrontal cortex includes areas 9, 10, 11, 12, 46, 47, 13, 14, and 15 (Jouandet & Gazzaniga, 1979, p. 28).

Structures within the prefrontal cortex are highly interconnected, but these intrinsic connections are poorly understood in all species. As well, with the exception of a few primary sensory and motor areas, all neocortex is interconnected with one or another portion of prefrontal cortex (Barbas, 1992). The prefrontal cortex is also connected to the premotor region and thus, indirectly to the motor cortex.

Other salient connections include numerous structures of the diencephalon, mesencephalon and the limbic system. The prefrontal cortex

has been shown to have linkages with visual, auditory and somatosensory association areas. Except for efferents to basal ganglia which appear to be unreciprocated, all linkages are bi-directional.

Dorsolateral prefrontal cortex is primarily connected to the lateral thalamus, the anterodorsal caudate, the hippocampus, and the neocortex. The orbital region of prefrontal cortex, in addition to its connections with the medial thalamus, has direct links with the hypothalamus, the ventrolateral caudate, and the amygdala (Damasio, 1985; Fuster, 1989; Goldman-Rakic, 1987a, 1988; Nauta, 1971; Stuss & Benson, 1986).

Pandya and Barnes (1987) conclude that the dorsolateral and orbital regions reflect archi- and paleocortical origins respectively, and produce two distinct anatomical/functional systems. The dorsal system involving the medial surface of the frontal lobe and the superior part of the dorsolateral surface is strongly interconnected with the posterior parietal lobe and cingulate gyrus. In their view, this system mediates sequential processing of spatially related and motivational material. The second system involves the orbital surface and more ventrolateral regions, and appears to mediate emotional tone. It is linked to three independent, but functionally linked limbic systems - the limbic lobe, the septo-hypothalamo-mesencephalic continuum and a peripheral visceroendocrine system. These two systems (dorsal and orbital) interact, positioning the frontal lobe as a final point of informational integration from the external sensory and internal limbic worlds (Stuss, Gow, & Hetherington, 1992).

In sum, the prefrontal cortex is bi-directionally linked with limbic motivational systems, reticular activating arousal systems, sensory associational systems, and motor regions. Thus, anatomically, the prefrontal cortex is strategically situated to perform integrative and regulatory functions.

Theoretical Perspectives

Benton (1991a) characterized the legacy of work done on the prefrontal cortex prior to 1950 as an "embarrassment of riches" (p. 26) reflecting the extensive evidence that the prefrontal cortex comprised a number of distinct and critical anatomic-functional areas in the absence of theories regarding the neural mechanisms involved or more molar theoretical models. Stimulus-response theories from mainstream psychology, e.g., Hull (1943), did not provide mechanisms that could account for complex mental processes, such as those associated with complex goal attainment. Miller, Galanter, and Pribram (1960), and Pribram (1969) were among the first to propose a more complex monitoring system to provide for complex goal attainment.[1]

Pribram: T-O-T-E Executive Monitor

Miller, Galanter, and Pribram (1960) addressed the insufficiency of the reflect arc (e.g., Hull, 1943) to account for complex, cognitively based behavior. For example, they rather amusingly chastised Tolman, and cognitive theorists generally, for ignoring the specific mechanisms involved.

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[1]The writer is indebted to Stuss and Benson (1986) for their approach to the analysis of historical milestones in the theoretical understanding of the prefrontal cortex.

They characterized Tolman's rats as "left buried in thought; if he gets to the food-box at the end that is his concern not the concern of the theory" [(Guthrie, 1935) quoted in Miller et al. (1960 p. 9)].

Miller et al. (1960) recognized, as did Tolman (1948) and other cognitive theorists of the time, that stimulus response theories are inadequate to account for behavior that results from complex cognition. They presented the problem as follows: "The gap from knowledge to action looks smaller than the gap from stimulus to action -- yet the gap is still there, still indefinitely large" (p. 9).

To fill the gap, Miller et al. (1960) proposed that a complex feedback system is operative wherein incongruencies between an organism and the stimulus result in a testing operation that continues until the incongruity is resolved. The basic unit of organization they postulated was the T-O-T-E (test-operate-test-exit). T-O-T-E's can be hierarchically organized into complex contingencies which serve to define the nature and sequence of components of behaviors necessary to achieve a goal. Thus behavior is construed as a function of an external representation of a neuroprogram (Pribram, 1969).

According to Pribram (1969), the T-O-T-E system can be conceptualized as a four-fold division of brain function. There are both external and internal representations. The external representation includes the sensory areas and their association areas. Internal representation is a function of the limbic system and frontal association areas.

External representations permit discrimination and can be formed into plans by limbic and subcortical processes. In this system, frontal regions store plans while awaiting implementation, but these plans are originally formulated in posterior regions then transferred to frontal regions. Brody and Pribram (1978), and Pribram (1973) later emphasized the factor of context. Thus, behavior at any given time is determined by an analysis of the consequences of behavior and "the frontal cortex is especially concerned in structuring context-dependent behaviors" (Pribram, 1973, p. 308). This accords a higher order control function to the prefrontal cortex. Indeed, Pribram (1987, p. 33-34) postulates that a major function of dorsolateral convexity of the prefrontal cortex (DLPC) is to anticipate the impact of future behavior contingent on alternative courses of action.

While attributing an important role to the prefrontal cortex was not entirely original, the emphasis on an active monitoring (cybernetic) system, guiding behavioral decisions in response to changes in the environmental context, clearly represented a break with stimulus-response theories of behavior. Pribram (1987) concluded "that the function of the anterior frontal cortex is to relate the processes served by the limbic forebrain to those of the somatosensory motor systems" (p. 32).

Teuber: Corollary Discharge

Teuber's (1964, 1966) theory of frontal lobe function was considered radical, as he proposed that action began in frontal systems rather than in posterior sensory regions, whereas the opposite had been assumed by

previous neuroscientists. In fact, he proposed that not only did action begin in frontal motor systems, the frontal lobe influenced sensory perception per se. His classic illustration of this phenomenon relates to voluntary eye movement which he explained as follows:

> During a voluntary shift of gaze, the environment stands still - presumably because a corollary discharge from the oculomotor to the visual mechanism prepares the latter for the change in relative position which will result from the ocular motion. . . . Passive motion of the eyeball, as can be produced by pushing against the eye, makes the world move, because the counterbalancing corollary discharge is missing. (Teuber, 1964, p. 419)

Thus Teuber postulated that there is an anticipatory discharge from motor to sensory areas preparing or presetting the sensory structures for the predicted changes induced by voluntary movement. Previous conceptualizations in terms of stimulus-response were inadequate, therefore, to account for perceptions, but by hypothesizing an effector function for the frontal lobe, the perception of voluntary actions could be accommodated.

Teuber (1964) worked with frontal lobe patients and clearly demonstrated his theory with tasks which were dependent on the control of movement such as visual search and the rod and frame task of visual and postural verticality. Obviously, deficits in performing these tasks could be the result of failure to send signals downstream to sensory areas or communication from sensory to frontal lobe and basal ganglia. In any event,

corollary discharge theory implies reciprocal interaction and thus a system within which damage at any non-redundant or critical point could threaten the overall function of smooth motoric flow and the sensory experience of same.

Nauta: Structure-Function Inference

Nauta (1971, 1973), especially on the basis of the neuroanatomic relationships between the prefrontal cortex and other aspects of brain, inferred both an effector and sensor function for the prefrontal cortex. He noted that the prefrontal cortex is placed in reciprocal relationship with two major functional zones: a) the visual, auditory and somatosensory zones owing to its reciprocal connections with parietal and temporal cortices, and b) the telencephalic limbic system including the hypothalamus and both meso- and diencephalic structures connected to the hypothalamus.

The prefrontal cortex is thus the foremost structure in a unique position to integrate and synthesize the inner and outer sensory worlds. Nauta (1971) stated "the available anatomical evidence indicates the frontal cortex as one, and perhaps the only, realm of the neocortex where neural pathways representing the internal milieu converge with conduction systems re-representing the external environment as reported by all exteroceptive modalities" (p. 182).

Nauta (1971) speculated that in the normal course of behavioral planning, the individual decides upon a particular course of action as a result of thought processes which relate various alternative courses to their

affective and motivational implications. He thus suggests that the prefrontal cortex would be centrally involved in "behavioral anticipation" (p. 83) and lesions which interfere with critical frontal-limbic relationships could thus be understood to result in the characteristic "loss of foresight" (p. 83) associated with frontal lobe lesions.

It will be recalled that Teuber (1964) suggested an effector function for the frontal lobe with respect to exteroceptive processing mechanisms. According to this theory, the frontal lobe, by a mechanism of corollary discharge, presets sensory processing mechanisms for anticipated sensory charges that would result from impending motor output. Nauta (1971, 1973) extends this concept of corollary discharge to mechanisms dealing with interoceptive information, as well as exteroceptive mechanisms. In this way a mental template of plans could be established thus accounting for the capacity to develop and pursue future goals in normal function. Problems in establishing and maintaining long term goals are common sequelae of frontal lobe lesions.

> Such a pre-setting could be thought to establish a temporal sequence of affective reference points serving as 'navigational markers' and providing, by their sequential order, at once the general course and temporal stability of complex goal- directed forms of behavior. (p. 183)

Nauta also suggested that frontal lobe lesions, as they affect fronto-limbic associations, could result in what he termed "interoceptive agnosia" (p. 82)

which would relate to an impairment in a person's ability to integrate information from "his internal milieu with the environmental reports provided by his neocortical processing mechanisms" (p. 182). Such a disconnection between cognition and affects could of course account for the classical inconsistency between voiced intention and behavior observed with frontal lobe patients.

Damasio: Hierarchical Integration

Damasio's (1979, 1985) approach to inferring frontal lobe functions is similar to Nauta's (1971, 1973) in that he relies heavily on anatomical brain relationships and the clinical and behavioral effects of specific disruptions in those relationships determined through animal experimentation and human clinical studies. Intervening progress in anatomy, as well as increased specificity in animal and human studies, has allowed Damasio to extend Nauta's theories and to be more precise in defining frontal lobe functions.

Damasio (1979, 1985) proposes broad evaluative and regulatory functions for the frontal lobes guided by a supraordinate principle of preservation of an individual's equilibrium. Thus, for Damasio, the general functions of the frontal lobes are to judge and regulate ongoing external perception and, based on this perception, to plan and execute the most appropriate response, i.e., in accordance with its purposes. Thus, the frontal lobes analyze sensory data and organize responses according to a hierarchy of goals. This is accomplished through a series of gating mechanisms. First, a lower system gating is performed at the level of the hypothalamus where

the pleasure/pain and the motivational valence of a stimulus is evaluated. This process is effectively automatic, however, complex information that requires evaluation of both external and internal implications and the postulation of goals demands participation of the frontal lobes. Damasio (1985) suggests that the orbital prefrontal region is critical for this higher social evaluation. As he states "We believe the orbital sector of the frontal lobe contains a variety of primary hypothalamic mechanisms of response, substituting more elaborate forms of action suitable to complex social behaviors" (p. 369).

Damasio (1985) identifies the dorsolateral sector of the frontal lobe as critical for higher cognitive activity providing for organization of mental contents, planning of future actions and creative thinking. The mesial sector of the frontal lobe is important in the expression and experience of affect and drive. Damasio attributes a global function to the frontal lobe "to handle hyper-complex environmental contingencies in the framework of the individual's own history, and in the perspective of his desired future course" (Damasio, 1979, p. 371). However, his perspective is clearly toward the eventual analysis of frontal structural/functional systems, as he concludes that "the notion of a single frontal lobe syndrome is just as absurd as the notion of a single frontal lobe function" (Damasio, 1985, p. 369).

Luria: Integrated Functional Systems

Luria (1970, 1973a,b) broadly sketched the brain into three functional units: a) the subcortex, especially the reticular activating system which

regulates wakefulness and mental tone; b) the occipital, parietal and temporal cortex involved in the reception, analysis and storage of information; and c) the frontal lobes which he considered to be the unit for programming, regulation, and verification of activity.

Each of these units can be further differentiated into three, hierarchically-organized, cortical zones: a) the primary (projection) area which receives and transmits information to the periphery; b) the secondary (projection-association) area, which processes information and prepares programs; and c) the tertiary areas which are functionally complex and require the participation of many cortical areas (Luria, 1973a, p. 43). Luria (1980) postulated four primary frontal cortex functions. First, the frontal cortex is intimately involved in the analysis and synthesis of impulses associated with motor processes. Secondly, through its reciprocal connections with the reticular activating system, it has an important role in the regulation of activity states. Thirdly, through its association with limbic and associated structures concerned with interoception, it is highly involved in the regulation of body states; and, fourthly, Luria states:

> The frontal lobes synthesize the information about the outside world received through the exteroceptors and the information about the internal states of the body AND THAT they are the means whereby the behavior of the organism is regulated in conformity with the effect produced by its actions. (p. 263)

For Luria, the prefrontal lobes which represent the tertiary zone are a "superstructure above all other parts of the cerebral cortex, so that they perform a far more universal function of general regulation of behavior" (1973a, p. 89).

Luria and Homskaya (1964), and Luria (1980) suggested that speech and particularly inner speech is what provides for mediation of complex cognition and the ability to carry out complex plans. The frontal lobes are concerned with main plans and intentions and the use of inner speech as a component of a mechanism such as Miller, Pribram, and Galanter's (1960) T-O-T-E "makes it an important component of the 'system with the highest level of self-regulation' as human voluntary activity may be described" (Luria, 1980, p. 293).

Shallice: Information-Processing Approach

Shallice (1988) and Norman and Shallice (1980/1986) found that Luria's position on the functions of the frontal lobes was compatible and complementary to perspectives from cognitive psychology. Within their information-processing model, Luria's frontal functions of programming, regulation and verification is reflected by the construct of the Supervisory Attentional System (SAS).

The basic units within Shallice's system are cognitive units. These units are characterized as specific brain functions which are presently understood to be relatively localized anatomically, e.g., language and visuo-spatial abilities. Those functions can be organized and integrated to represent

routine behaviors which are termed schemata. As an example, a schema for driving a car would be dependent on visuo-spatial, manual and appropriate recognition systems. Schemata are often organized hierarchically such that you may consider a "source" schema for driving a car superior to its component schemata representing various sub-activities, e.g., braking and steering. Schemata represent routine operations, usually over-learned sequences of behavior, though through concatenation and hierarchical organization, they may be complex when regarded as a whole, for example, driving to and from work.

Contention scheduling is the process whereby routine actions or thinking operations are selected. This scheduling is triggered by sensory perception or by the output of other schemata. Once selected, a schema remains active unless it reaches its goal or is inhibited by a higher level controlling schema. When contention scheduling fails or when there is no known solution, appeal is made to the highest level in this system, the SAS. The SAS is required to account for problem solving activities and the phenomenological distinction between "willed" and "non-willed" action. Shallice (1988) describes the role of the SAS which he attributes to the frontal lobe as follows:

> The Supervisory System - which has access to a representation of the environment and of the organism's intentions and cognitive capacities . . . is held to operate not by directly controlling behavior, but by modulating the lower level contention-scheduling system by activating or inhibiting particular schemata. It would be involved in the genesis of

willed actions and required in situations where the routine selection of actions was unsatisfactory - for instance, in coping with novelty, in decision making, in overcoming temptation, or in dealing with danger. (p. 335)

Shallice's model is compelling as it seems to account for many "executive" problems associated with frontal lobe lesions yet making understandable the preservation of many subordinate functions and behavior, for example, performance on IQ tests which would not implicate the SAS to a great extent.

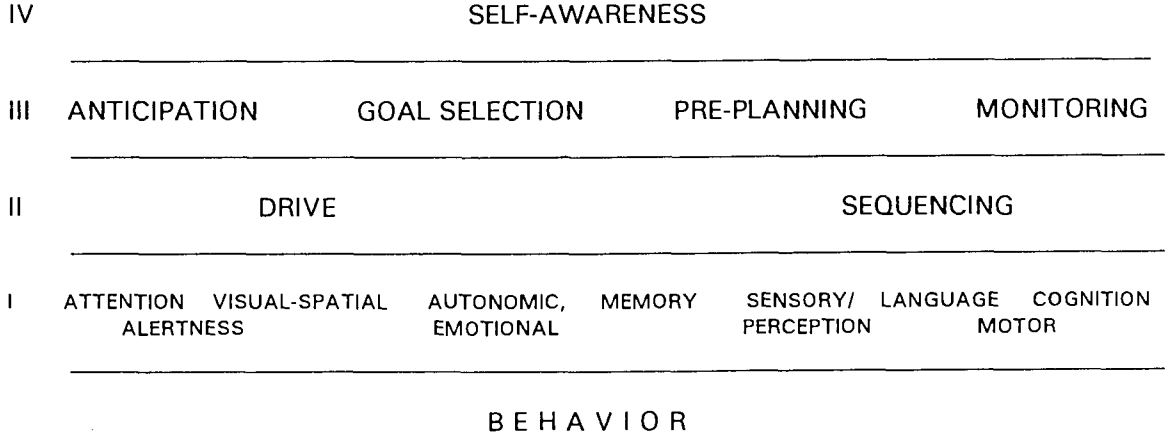
Stuss and Benson: A Behavioral Anatomical Theory

Stuss and Benson (1986), further to an exhaustive review of matters related to the frontal lobes, present a comprehensive theory of frontal lobe function which they term "A Behavioral Anatomical Theory".

Their theory is best described with reference to their diagram of the "Hierarchy of Brain Function" reproduced below.

Figure 2.1

Hierarchy of Brain Function



Note: Adapted from Stuss and Benson (1986, p. 284). Notation of Levels I - IV have been added here by the writer to facilitate reference.

LEVEL I: Posterior Basal Systems

Level I in this model represents "organized integrated fixed functional systems" (p. 240), such as sensory, motor, memory, language and visuo-spatial function. While these systems are interrelated, both anatomically and functionally, they are considered posterior/basal functions within Stuss and Benson's model. Each of these Level I functions is reciprocally connected to and influenced by the frontal cortex. The frontal structures exercise a supervisory or executive role vis-à-vis these posterior functions. While the basic activities of posterior functional systems may remain intact in the event of damage to frontal structures, control of the functions may be altered. This is in keeping with Stuss and Benson's demonstration throughout their text that frontal lobe pathology causes only indirect disturbances to many functional systems. As a corollary, they indicate that frontal lobe function typically cannot be adequately assessed by independent analysis of posterior functional systems.

Beyond the level of posterior functional systems, Stuss and Benson (1986) propose three conceptually distinct, hierarchical and successively more abstract divisions of frontal lobe systems identified as Level II, Level III, and Level IV in the figure above.

Level II: Functional Systems

Two frontal functional systems, (a) sequence, set and, integration, and (b) drive, motivation, and will, are construed as parallel to the Level I posterior/basal systems discussed above.

(a) Sequence, Set & Integration

Sequence, set, and integration are viewed by these authors to be functions localized to the dorsolateral convexity of the prefrontal cortex. By sequence, they refer to the ability to structure events and behavior in the temporal domain. Set refers to the ability to extract the key data from multiple bits of information and to form this material into sets of related information, thus, set formation allows the production of new, more complex information from available sequences of data. Integration in this perspective refers to the ability "to extract chosen bits from a number of related or unrelated sets of information and to integrate these data into novel knowledge (information) or into an understanding of a complex situation" (p. 242).

(b) Drive, Motivation & Will

Changes and disturbances in drive are a common observation further to frontal lobe lesions. While the most common alteration is apathy and a general decrease in activity, excessive drive, apparently based on decreased ability to inhibit action, is also observed. Stuss and Benson (1986) believe that medial sagittal frontal structures, particularly the cingulate gyrus and the supplementary motor area, are involved with the initiation of both motor and

mental activities while damage to the orbital region can result in a decreased ability to inhibit drive, reflecting inadequate mental control over behavior. The ubiquitous nature of abnormal drive in clinical populations with prefrontal lesions and its variable effects on behavior led these authors to consider drive as a separate prefrontal cortex function which impacts posterior functional systems.

Motivation and will are viewed as closely related to drive. Motivation is seen to reflect a degree of intellectual control over drive. While will has metaphysical connotations, it is argued that human will represents an important brain activity which can be altered by focal frontal damage and is accorded the status of a frontal function.

Thus, at Level II, two units of mental activity, sequence and drive, localized to the dorsolateral frontal convexity and the medial/orbital structures respectively, are proposed as frontal functional systems that interact, but as superordinates, with posterior functional systems.

LEVEL III: Executive Function

At an independent and conceptually higher level, Stuss and Benson (1986) postulate an "executive function". In their system, executive function is a prefrontal lobe function which is required in nonroutine, novel situations that require new solutions. Executive function is characterized by "anticipation, goal selection, pre-planning (means-end establishment), monitoring, and use of feedback (if-then statements)" (p. 244). Thus,

executive function is a superordinate control function which these authors further qualify as principle control, program control and sequence control.

To illustrate these forms of control, these authors consider the steps involved in preparing a report as follows. One of the basic guiding principles would be that the report should be readable. Thus, principle control specifies the goal and program control would then specify the contingencies which would apply, a set of if-then decisions would be established to evaluate if change is needed, to determine how it should be done and eventually to carry it out. Sequence control in this context would involve the establishment and monitoring of the order of activities required to produce the intended report.

An important concept associated with executive function in this system is that prefrontal structures are important when new solutions are being developed or new activities are being learned. Once such solutions or activities have become routine, they can be carried out by other (posterior) functional systems.

LEVEL IV: Self Awareness

Self-awareness and self-consciousness are hypothesized to be the highest attributes of the frontal lobes. Stuss and Benson (1986) point out that while self-awareness and self-consciousness have traditionally been viewed as the purview of philosophy, theology and psychology, recently neuroscientists, information-processing theorists and metatheorists have all examined consciousness from their respective perspectives. These authors

view this as a level of functioning where "the mind-brain dichotomy is more clearly expressed and loses sharp differentiation" (p. 249).

The self-awareness level is hypothesized on the basis of the characteristic effects of frontal lesions, especially the resultant shallowness of interest, loss of self-concern and impairment in self-monitoring. Self-awareness in this system functions integrally with executive functions though it is conceptually at a higher level and is responsible for coherence throughout the functional hierarchy.

Stuss and Benson (1986) conclude that "it is through understanding of the influence of prefrontal brain structures on mental activity that the true essence of humanness will be approached. The frontal lobes are the key to the highest human functions" (p. 249).

Ingvar: Memory of the Future

Ingvar (e.g., 1979, 1983a, 1983b, 1985) has, over the past 25 years, conducted many regional cerebral blood flow studies involving human subjects performing a variety of behavioral (motor) or cognitive tasks. As well, he has evaluated cortical blood flow in a number of clinical groups which demonstrate frontal pathology.

On the basis of these studies, as well as others which he has reviewed, he has concluded that cognitive and behavioral tasks involving a sequential form, e.g., problem solving, give rise to an activation of frontal/prefrontal cortical areas. Such action plans for movements as well as for cognition, i.e., in problem solving, contain programs in the form of temporally

structured neural events which can be remembered, and thus, can critically determine the future. Such plans, Ingvar (1985) has termed "memory of the future" (p. 128). Such a memory of the future Ingvar believes, has its own neuronal substrate which is widely represented in frontal cortical areas. This is in contrast to episodic memory which pertains to actual sensory and cognitive percepts in the past and in the present. Thus, for Ingvar, there is a separate neuronal future system in the brain which is selectively responsible for the temporal structuring of future behavior and cognition.

In addition to increases in frontal activation, where the future is concerned, Ingvar (1985) reviews evidence to suggest that patients whose disorder is frontal (usually resulting in a reduction of one's plans and interest in the future) show reduced frontal metabolic activity. This is particularly true of cases of organic dementia characterized by symptoms of progressive apathy and a striking lack of plans for the future.

Ingvar proposes that it is the recognition of the serial nature of events that allows us to recognize them as meaningful, as he states: "it is the temporal structure, the serial nature, of the sensory input which is a prerequisite for the experience of causality and hence, the production of serial neuronal actions underlying the anticipatory concepts of the future" (1985, p. 134). Ingvar goes a step further and suggests that serial concepts or "memories" of the future provide a general basis for the perception of the meaning of events as these are contrasted (consistent or divergent) with our inner templates of the future.

Fuster: Mediating Cross-Temporal Contingencies

Fuster (1980, 1985, 1989) postulates that the supraordinate function of the prefrontal cortex is to construct "temporal structures of behavior with a unifying purpose or goal. In other words, the structuring of goal-directed behavior" (1989, p. 158). Fuster conceptualizes all behavior in terms of a hierarchical order of temporally structured units or temporal gestalts. These temporal gestalts are defined in terms of a purpose and their significance is derived from their relationship to each other and to the ultimate goal. Thus successive units with short term goals make longer units and thus hierarchies are formed. Within this scheme purposes may vary widely, e.g., from satisfying a primary drive to the attainment of intellectual or aesthetic goals. However, Fuster notes that only the highest levels of a behavioral hierarchy would likely come under the control of the prefrontal cortex.

Fuster identifies three parameters that determine the critical involvement of the prefrontal cortex: novelty, complexity, and time. Novelty and complexity must exceed some still to be specified threshold, but Fuster states that the behavioral units under definition by the purpose at hand must be at least partly novel and formed to meet new and changing requirements. Complexity of the behavioral units is a function of the number and variety of sensory, motor, and cognitive elements. Unlike novelty which is a sufficient criterion for engaging the prefrontal cortex, complexity is not, as routine or instinctual patterns of behavior are not within the purview of the prefrontal cortex.

Where contingent behavioral units or gestalts are temporally spaced, e.g., such as in experimental delayed response tasks, time alone can require the participation of the prefrontal cortex to mediate such cross-temporal contingencies. Fuster proposes that this temporal mediation is accomplished by a neural mechanism localized to the prefrontal cortex which involves three distinct but complementary sub-functions. His proposal is elaborated below with reference to the delayed response paradigm.

The origins of Fuster's theory of prefrontal cortex function relate to the delayed response deficit, first reported by Jacobsen (1935, 1936), that monkeys with frontal ablations display. In the classic version of the delayed response test, the monkey is required, on every trial, to manually choose one of two identical objects under which, a few seconds before, food has been placed in full view of the animal. The concealed food is the reward for making the right choice between the two objects. In order to succeed, information must be retained over a period of delay (typically an opaque screen is placed between the monkey and the two wells during the delay period) and secondly, the alternate choice must be suppressed, even though it may have been the last position to have been reinforced. There is still some debate over the precise nature of the deficit, but it seems to have been established beyond dispute that animals with prefrontal lesions are incapable of performing delay-tasks. Some investigators have emphasized mnemonic process (e.g., Fuster, 1989; Jacobsen 1935, 1936), whereas some have highlighted spatial perception or spatial orientation as the source of the

deficit. Others have centered on attention, proprioception or developing a central representation of kinesthesis (for reviews, see Fuster, 1989, Goldman-Rakic, 1987a, Pribram, 1987, and Stamm, 1987). It has also been confirmed by a variety of techniques that monkeys require an intact prefrontal dorsolateral convexity to perform delayed response tasks. Investigatory techniques have included lesions (e.g., Goldman & Rosvold, 1970); localized electrical stimulation (e.g., Stamm, 1969); localized cooling (e.g., Fuster & Alexander, 1970); single unit recordings (e.g., Fuster, 1973, 1987); localized dopamine depletion (e.g., Brozoski, Brown, Rosvold, & Goldman 1979); and selective $D_1$ antagonists (Sawazuchi & Goldman-Rakic, 1991).

The delayed response task has three main components: a) the presentation of the visual cue, b) an enforced delay period, and c) a motor response which is contingent on the original cue and followed by reward if correct.

Utilizing single unit microelectrode methodology, several studies reviewed by Fuster (1980, 1987, 1989) have demonstrated clear-cut temporal correlations between patterns of prefrontal cell activity and delay task facets (e.g., Fuster & Alexander 1970; Fuster, 1973). More specifically, for some cells in the dorsolateral convexity, the elevation of activity begins and terminates with the delay, suggesting that these cells are involved in the retention of cue features during the delay. During the delay, a substantial number of prefrontal cells show a tendency to gradually

diminish firing as the delay progresses while others show a gradual increase in firing in apparent anticipation of the choice and motor responses (Fuster, Bauer, & Jervey, 1982). Based on several studies involving such correlations between task facets and patterns of cell firings, Fuster concludes that the functions of short-term memory (mediation during the delay) and anticipatory response set are primarily functions of the dorsolateral convexity, while control of interference and reward (reinforcement) appears related to the orbital aspects of the prefrontal cortex in monkeys.

For Fuster, the delayed response task epitomizes the principle of cross-temporal contingency and the three cognitive functions that are necessary to accomplish the task are seen by him to be generalizable functions of the prefrontal cortex. Fuster does not claim that the prefrontal cortex carries out these functions in isolation, rather the evidence is that many brain regions participate. Notwithstanding, the prefrontal cortex appears to be essential to their successful operation. Fuster concludes that whereas the criterion of double dissociation may not be formally met, it nevertheless appears that given an intact dorsolateral convexity, the delayed task can be performed independent of the inferotemporal cortex (Fuster, Bauer, & Jervey, 1985); the parietal cortex (Quintana, Fuster, & Yajeya, 1989), and an intact hippocampus is not necessary (e.g., Winocur, 1991). Selective damage to the dorsolateral convexity does not impair other cognitive functions, such as

discrimination learning or object reversal and recognition tasks (see Fuster, 1989 and Pribram, 1987 for reviews).

The first two functions are temporally symmetrical functions. The first is a retrospective function which Fuster terms provisional or short-term memory. He defines this as memory about the sensory and motor events in a temporal sequence toward a goal. Thus, it permits reference to any event in a behavior sequence to prior events and to the original plan. This requires that the global scheme and relevant events must be temporally represented and retained. Thus, this form of memory is distinguished by context, not content, such as memory for objects. "Its context is provided by the behavioral action and limited by its accomplishment" (Fuster 1989, p. 163).

The second symmetrical function Fuster terms anticipatory set which implies both foresight and preparatory action. It involves the anticipation of events in the behavioral structure and preparation for action. While it is prospective, Fuster explains it is rooted in past experience which permits the subject to anticipate a range of possible contingencies. He compares this function to Luria's presynthesis or Piaget's (1952) concept of "schema". He further suggests that the function of the frontal eye field, which he believes is the likely source of corollary discharge to the visual system, represents in microcosm the anticipatory function of the prefrontal cortex in the formation of behavioral structures. Both of the above symmetrical functions, Fuster believes, are localized to the dorsolateral convexity.

Referring to the delayed response task, the first function would allow the subject to retain in short-term memory the location, spatial configuration, and sensory properties of the baited object. The second function of set prepares the subject to respond on the basis of experience to the appropriate object, i.e., if stimulus configuration 'X', then response toward object 'Y'. There are, however, several potential sources of interference which can interfere with the successful completion of the task. Firstly, the subject could be distracted from the task by extraneous stimuli or secondly by internal stimuli such as previous memory traces, e.g., to the previously successful, but now irrelevant behavioral structures. Other sources would include impulses toward immediate gratification. Thus Fuster has proposed a third function of prefrontal cortex, "control of interference" which he believes is localized to the orbital/medial region. This function appears to be a general inhibitory function suppressing previous memories and not presently appropriate impulses. The role of the prefrontal cortex in mediating a temporally extended, contingency interdependent behavioral structure, has been illustrated with reference to the delayed response test. Further, a mechanism comprised of three integrated, cognitive subfunctions (i.e., short-term memory, anticipatory set, and interference control) has been proposed. For Fuster, this illustration represents, in microcosm, the broad, critical, and pervasive effector role of the prefrontal cortex. Fuster (1990) further explains this role in relation to the perceptual action cycle.

The perceptual action cycle relates to the pattern in all forms of behavior, from the simplest and most automatic to the deliberate, that motor action is not only triggered by sensory signals, but also regulated over time by the sensory feedback generated by changes that action itself induces in the environment. The prefrontal cortex in this process has a central and pivotal role in uniting perception and movement. Fuster (1990) notes that "each area in the succession of cortical areas making up a sensory pathway projects to frontal cortex and at each step, as the sensory hierarchy progresses through associative cortex - a progressively higher stage is reached by the motor hierarchy made up by frontal areas" (p. 177).

To illustrate these relationships, Fuster presents the analogy of the upward slope of a mountain representing sensory organization increasing in complexity toward the top where polymodal association areas of the prefrontal cortex are reached while on the opposite descending slope the motor hierarchy goes from the most complex level of organization in dorsolateral convexity through to premotor and on to the primary motor area. In this analogy the prefrontal cortex is thus at the apex of the mountain, performing its hypothesized, central effector function (cf. Fuster, 1990).

Fuster (1989), earlier noted that the perception/action dichotomy is probably marked by the central sulcus in non-human primates. He concludes "The evolutionary development of ever higher areas of association in posterior as in anterior cortex, may reflect the opening up of ever greater

possibilities for abstract, (i.e., symbolic) perception and for elaborate and deliberate goal-directed action" (p. 175).

In this perspective, human language represents the most highly differentiated aspect of the perception/action cycle. Accordingly, Wernicke's area in the left angular gyrus (perceptual and prerolandic), Fuster speculates, likely represents a phylogenetic differentiation of the polymodal association cortex about the posterior extremity of the superior temporal sulcus, while Broca's area (motor and postrolandic), likely arose out of ventrolateral, prefrontal or premotor cortex.

Recent studies (Quintana, Yajeya, & Fuster, 1988; Quintana & Fuster, 1992) provide further support for Fuster's theory concerning the perception action cycle. For instance, in the 1992 study, monkeys performed a visuo-motor task with temporal and spatial separation between the cue (color) and the direction of response (left or right). During the delay between them, sensory-coupled cells which had been activated by the cue, decelerate their firing as the response approaches. Simultaneously, motor-coupled cells in both prefrontal and parietal cortex accelerated their firing and this acceleration was proportional to the degree that response direction was predictable. These findings were interpreted as an indication of the cooperation of prefrontal and parietal neurons in cortical networks that represent visuospatial information and that bridge the temporal gaps between sensory information and consequent motor responses.

Fuster (1993) concluded that the prefrontal cortex is critically involved in the temporal organization of action, whether in terms of skeletal movements, ocular motility, or spoken language. In his view, neural representation within each domain is represented hierarchically with the more general or global aspects represented in prefrontal cortex. With regard to spoken language, for example, he has suggested that "the syntax, the ideas, logical statements, and perhaps even sentences would be represented in prefrontal cortex, whereas the words . . . would be represented in premotor and motor cortex" (p. 162).

Fuster's general view of the supraordinate role of the prefrontal cortex in developing and executing behavioral plans has been reviewed. As well, albeit in sketchy form, three cognitive subfunctions; short-term memory, anticipatory set, and interference control, have been described. The first two of these functions Fuster localized to the dorsolateral convexity in non-human primates, and the third, to the orbital/medial cortex.

Fuster's (1989, 1990) theoretical presentation goes considerably further in elucidating a model of 'how' the prefrontal cortex, in concert with other brain structures, likely develops and executes complex behavioral structures which are extended temporally and relate to goals of varying complexity and abstractness. For the present purposes, it is important to note that the neural mechanism proposed by Fuster with its components illustrated in the resolution of delayed response task is hypothesized to underpin executive function. In view of Fuster's treatment of the human lesion data (discussed

later in this review), it is also clear he believes that such a mechanism or set of processes could also accommodate processing complex symbolic cognition and other complex psychological functions.

Summary

Despite great differences in their theoretical orientation, and the databases from which they developed their views, the works reviewed here on the main functions of the prefrontal cortex are in remarkable agreement. All authors reviewed consider executive function, i.e., organizing behavior in terms of its consequences, to be a primary role of the prefrontal cortex. Executive function is generally agreed to involve capacities for formulating goals, planning, and carrying out plans effectively (cf. Lezack, 1982; Duncan, 1986). Pribram's main focus was to hypothesize a mechanism (T-O-T-E) to monitor progress toward a goal. Teuber emphasized the effector role of the prefrontal cortex and Nauta extended this view to accommodate complex goal-directed behavior. Damasio proposed that behavior was guided by the prefrontal cortex subject to the principle of preserving an individual's equilibrium. For Luria, the prefrontal cortex is the means that regulates the organism to achieve the effect produced by its actions. Shallice's construct of Supervisory Attentional System is dedicated to executive decision-making. In Stuss and Benson's model, Executive Function is considered an independent level of function above which there is only one further level of organization, the Self-Conscious level. In Ingvar's view, the future system of the prefrontal cortex is selectively responsible for

the temporal structuring of future behavior. Finally, the role of the prefrontal cortex in the temporal structuring of goal-directed behavior is at the core of Fuster's proposals, particularly where novel, complex, or temporally discontiguous behavioral structures are required. The notion of executive function implies a hierarchical organization of perception and behavior. This is explicit in most of the theories presented. Language, because of its potential to be logically structured, is seen to be vital for the programming and regulation of behavior. This is particularly the case in Luria's, and Stuss and Benson's approach.

It is language which allows the creation of a cognitive architecture which serves to facilitate programming of voluntary behavior and the mediation of long-term goals. Thus, the creative, analytic, and synthetic capacities of language are seen to be primarily vested in the prefrontal cortex. Specific psychological or cognitive processes include foresight, abstract reasoning, social sensitivity, empathy and self-awareness.

Another vital role attributed to the prefrontal cortex by these authors relates to the dynamic interplay of exteroceptive and interoceptive processes. The prefrontal cortex, in its executive role, evaluates progress toward a goal in terms of inner templates for action. It is at this level that affective and motivational considerations enter the decision-making calculus. In its monitoring and regulating role, the prefrontal cortex mediates the transactions of an individual with his perceptual world and these are

determined by the context of the situation and the impact of alternative courses of actions.

Lastly, most authors assume a need for energy, and while the source is generally seen to be of limbic origin, the prefrontal cortex is postulated to mediate this energy and damage to the prefrontal cortex typically results in disorders of drive and arousal. The control, elaboration, and modulation of emotional behavior are generally considered to be critical prefrontal functions. This concludes the theoretical overview of prefrontal functions, the next sub-section will examine the clinical syndromes which result from damage to the prefrontal cortex.

## SECTION II EFFECTS OF PREFRONTAL LESIONS

### Introduction

This section reviews the effects of prefrontal lesions from a theoretical point of view. The cognitive sequelae associated with the prefrontal syndrome are then considered. This overview is followed by a review of experimental work conducted on patients who have sustained damage to the prefrontal cortex. Associated changes in personality are also reviewed.

### Prefrontal Clinical Syndromes

Among the first descriptions of cognitive and personality sequelae associated with frontal lobe damage were those provided by Harlow (1868). He describes the case of Phineas Gage who was apparently an intelligent and capable foreman of a railroad construction crew who sustained a severe frontal lobe injury when an iron tamping bar was blown upwards into his left

maxilla of the midline of the frontal skull. Remarkably, he survived the injury with full physical recovery. In contrast, executive, emotional, social, and behavioral sequelae were striking as described by Harlow:

> His physical health is good, and I am inclined to say that he is recovered . . . . The equilibrium or balance, so to speak, between his intellectual faculty and animal propensities, seems to have been destroyed. He is fitful, irreverent, indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned in turn for others appearing more feasible. A child in his intellectual capacity and manifestations, he has the animal passions of a strong man. Previous to his injury, though untrained in the schools, he possessed a well- balanced mind, and was looked upon by those who knew him as a shrewd, smart businessman, very energetic and persistent in executing all his plans of operation. In this regard his mind was radically changed, so decidedly that his friends and acquaintances said he was "no longer Gage". (pp. 339-340)

Scientifically, it would not be possible to determine that the effects were attributable to the specific lesion, in this instance a large bilateral lesion with a medial focus (Stuss & Benson, 1986, p. 225), as the possible impact of an

irritative focus about the primary lesion and diaschisis cannot be ruled out. In actual fact (MacMillan, 1992), Gage's skull was exhumed 12 years after his death and his brain had not been autopsied at the time of his death. Notwithstanding, all of the features of Gage's symptomatology are expectable prefrontal sequelae.

Since Harlow's case report, a remarkable spectrum of behavioral change has been attributed to prefrontal lesions (Luria, 1980; Mesulam, 1986; Stuss & Benson, 1986) yet, systematic investigations of these brain-behavior relationships have only begun (Mattson & Levin, 1990; Trimble, 1990). The inherent complexity of the prefrontal cortex with its associational character, limitations in imaging technology, as well as serious methodological and measurement problems, have made it difficult to establish unambiguous brain-behavior relationships. For instance, Damasio (1985) has suggested six principle areas of difficulty. Firstly, lesions are highly variable, e.g.: a) the location, size, nature, depth, brain maturational stage and time since occurrence, vary from case to case; b) the characteristics of an individual prior to sustaining a lesion can strongly influence the manifest sequelae, e.g., age, education, premorbid personality, intellect and acculturation, are important variables which partially determine outcome (see Goldstein & Levin, 1985, for a review of the significance of these antecedents in closed head injuries); c) there have been many methodological and measurement difficulties. Obtaining adequate samples with comparable lesions, as well as appropriate contrasts has generally been problematic; d) the changes

secondary to prefrontal lesions are often qualitative, they may be subtle and of a type which is difficult to measure, e.g., in the affective or social domain; e) co-occurring damage both near and distal to the site of the primary lesion often presents as a confound; and f) also, in certain studies, pre-existing psychopathology, e.g., in the prefrontal leucotomy studies, can obfuscate interpretation (e.g., Freeman & Watts, 1950; Greenblatt, Arnot, & Soloman, 1950).

Despite these constraints, certain findings have proven to be robust and there is general agreement that lesions of the prefrontal cortex can result in a broad range of sequelae, including changes and problems with self-reflective awareness, self-monitoring, executive functions, creativity, foresight, abstract reasoning, concept formation, attention, empathy, social sensitivity, emotional and affective self-regulation, behavioral impulsivity, disinhibition, and perseveration (e.g., Luria, 1980; Prigatano, 1992; Stuss & Benson, 1986; Stuss & Gow, 1992; Stuss, Gow, & Hetherington, 1992).

Although the clinical literature often refers to the 'frontal lobe syndrome', this term should be reserved for the global debilitating effects of massive frontal lesions (cf. Luria, 1980). In practice, clinical outcome is highly variable, both in terms of severity and symptomatology, and partial or modular syndromes have been proposed, especially on the basis of clinical observation of the effects of focal and relatively small lesions in different regions of the prefrontal cortex. In particular, three relatively distinct syndromes are generally proposed or acknowledged (Brown, 1985; Damasio,

1985, Fuster, 1989; Luria, 1980; Malloy, Bihrle, Duffy, & Cimino, 1993; Stuss & Benson, 1986).

Three prefrontal regions, dorsolateral, superior-medial, and orbital/lower medial have been related to distinct sequelae configurations. The superior-medial syndrome is marked by akinesia and is often associated with mutism, gait disturbances, and incontinence. Lesions limited to the dorsolateral regions, particularly of the dominant hemisphere, typically compromise cognitive functioning and result in some admixture of difficulties with: planning and intentional behaviors; evaluation of the consequences of one's actions; higher cognitive functioning involving abstract reasoning and concept formation; sustaining attention, concentration, and motivation; the effective use of language to regulate future behavior; and distractibility, impulsivity, and disinhibition. Lesions limited to the orbital limbic area of the prefrontal cortex typically result in some mosaic of the following: diminished emotional self-control; dramatic personality changes; reduced self-reflective awareness; indifference to emotional feelings or conflicts; affective disorders; increased sexual and aggressive drive disinhibition; increases in impulsive and antisocial behaviors; and reduced tolerance to alcohol (Brown, 1985; Damasio, 1985; Fuster, 1989; Luria, 1980; Malloy et al., 1993; Stuss & Benson, 1986; Trimble, 1990).

Clinically, focal damage to the dorsolateral and orbital regions of the prefrontal cortex has been linked to cognitive deficits and personality change, respectively. This dichotomy is not exclusive since damage in one

area of the prefrontal cortex can impact other areas because of the highly interconnected nature of these areas. Psychologically, cognitive, and emotional functioning are highly interdependent. Traditionally, however, cognitive and personality changes have been treated separately even though, as Blumer and Benson (1975) noted, most clinical presentations reflect features of both syndromes. Consider, for instance, the case of Phineas Gage presented above.

Prefrontal Syndrome - Cognitive Aspects

Historically, there have been several attempts to account for diverse prefrontal sequelae in terms of a unitary function. Brickner (1936) suggested a loss of the power of synthesis of simpler thought processes into more complex structures. Rylander (1939) proposed a defect in reasoning. Goldstein (1944) emphasized the loss of "an abstract attitude" (p. 188). Robinson (1946) stressed attentional difficulties, while Halstead (1947) believed that an abstraction factor which he considered to be the fundamental growth principle of the ego, was responsible.

Other authors, e.g., Pribram (1987) and Damasio (1985) have emphasized the heterogeneity and modularity of prefrontal functions. Luria (1980), Stuss and Benson (1986), and Damasio (1985) have focused on the hierarchical nature of brain functions, stressing interactive brain systems and executive control functions. Fuster (1989) has suggested that the homogeneity-heterogeneity debate may be idle given that these perspectives may be reconcilable if differences in the level of analysis and technologies

are taken into account. Thus, what constitutes the critical features of the syndrome and the nature of various levels of integration-dissolution processes require further definition.

Bianchi (1922) suggested that one of the main results of prefrontal damage was a loss of behavior being controlled by its purpose. This notion is at the core of current conceptualizations of executive function. Referring to the dorsolateral convexity syndrome, Luria (1980) stated "The essential feature is the lack of continuous comparison between the plan of action and the results actually attained, and this is evidently responsible for the disturbance of critical values (p. 361). Fuster (1989), in clear agreement, stated "The most distinctive disorder arising from prefrontal damage is the inability to initiate and carry out new and goal-directed patterns of behavior" (p. 131). Luria (1980) concluded that its effect on formal measures of intelligence is not major: "The frontal syndrome is characterized by an inherent contradiction - the potentially preserved "formal intellect" and the profoundly disturbed intellectual activity" (p. 360). By formal intellect, Luria refers to many intellectual activities, such as those tapped by standardized intelligence (IQ) tests. Stuss and Benson (1986) thoroughly reviewed the issue of the impact of prefrontal lesions on standardized IQ tests and concluded that "findings of IQ deterioration . . . are rare, whereas the number of studies reporting negative results is overwhelming" (p. 197).

Luria (1969, 1980) suggested that the "fundamental deficit" was with logical relationships. Whether in planning or logical reasoning, the capacity

to sustain attention (in the sense of short term working memory) to logically related sequential elements and to suppress or inhibit irrelevant associations would appear to be critical to goal attainment. This capacity to selectively search and selectively focus on the sequential ordering of intellectual activity appears to be what is disrupted by frontal lesions according to Luria (1980, p. 554). Fuster's (1989) notion of the elements of a plan deriving meaning by their relationship (causal relevance) to a goal is also pertinent.

The nature of this deficit is epitomized by Luria's (1980) view that "in discursive intellectual activity and in the solution of problems . . . the person solving a problem must analyze its requirements, select the essential relationships and discover the intermediate aims and operations by which these aims may be secured" (p. 580).

Luria (1969) stresses that this deficit in selective logical operations is particularly evident where alternatives or choices which have an element of conflict are present or where new schema must be developed "when intellectual operations demand the creation of a program of action and a choice between several equally probable alternatives, the intellectual activity of patients with a marked 'frontal syndrome' is profoundly disturbed" (p. 749).

Stuss and Benson (1984, 1986) stress the hierarchical organization of brain functions with the highest levels corresponding to personality constructs, such as the self. Such high levels of integration, in their view, are maximally associated with the prefrontal cortex. Specifically, they

identify four discrete, cognitive deficits which, according to their analysis, are dependent on the dorsolateral convexity. These include: a) a deficit in the ordering or handling of sequential behaviors, b) impairment in establishing or changing a set, c) impairment in maintaining a cognitive set, and d) dissociation of knowledge from the direction of response.

Fuster (1989) considers executive functions from the perspective of the cognitive functions required for the temporal integration of behavior. He has proposed that many of the sequelae attributed to prefrontal lesions can be accounted for in terms of three discrete cognitive functions: a) defective short-term memory (alternatively labelled working or provision memory), b) defective planning, and c) defective control of interference.

Fuster postulates that the first two functions are primarily dependent on the dorsolateral convexity while the third, control of interference, is considered to relate to the orbital/medial prefrontal cortex. He describes short-term memory as closely associated with sustained attention and essentially context dependent, apparently concerned with retrospective events which are related to present goals. The planning function is closely related to foresight and Fuster compares it to Ingvar's (1985) notion of having a memory for the future. Fuster (1989) explains that "Whereas faulty memory deprives the frontal patient of the ability to use experience of the recent past, faulty foresight deprives him or her of the ability to plan for the future" (pp. 138-139). The third function, control of interference, Fuster

considers is closely related to attention and its role is to inhibit impulses that are not timely and contextually relevant.

Executive and specific cognitive deficits hypothesized to be differentially associated with prefrontal lesions were discussed above, especially with reference to the writings of Luria, Stuss and Benson, and Fuster. Next, experimental evidence for these formulations is reviewed.

Prefrontal Lesions - Experimental Findings

Theories regarding the role of the prefrontal cortex emphasize executive function, yet experimental work has generally emphasized cognitive functions which are thought to underpin executive function. Luria (1980) emphasized the role of language in behavioral planning and regulation, and several authors have since elaborated upon his ideas (e.g., Alexander, Benson, & Stuss, 1989; Goldberg & Bilder, 1987; Kaczmarek, 1984, 1987; Stuss & Benson, 1987).

Ingvar (1993) has reviewed a number of metabolic studies of language and cognitive functioning, and concluded that neuronal action programs for speech are localized in the prefrontal cortex and that these structures not only are involved in speech production, but also participate in speech perception.

One procedure which has been extensively utilized in lesion studies is fluency tasks (oral and written) where the requirement is to generate as many words as possible that begin with a particular letter over a specified time period (cf. Thurstone, 1938). This procedure was found to be

particularly sensitive to left anterior lesions in several studies (Benton, 1968; Crockett, Bilsker, Hurwitz, & Kozak, 1986; Milner, 1964; Pendleton, Heaton, Lehman, & Hulihan, 1982). Several verbal fluency studies using Positron Emission Tomography (PET) technology have identified maximal involvement of the mid-dorsolateral prefrontal cortex in normal controls (Boivin et al., 1992; McCarthy, Blamire, Rothman, Gruetter, & Shulman, 1993; Parks et al., 1988). In these studies many other brain structures are also activated, likely due to distributive processing, however, the main interpretation is that the site of word representations is in the superior temporal regions and that inhibitory modulation of these areas by the left prefrontal cortex is the basis of intrinsic word generation (Frith et. al, 1991b). Rocchetta and Milner (1993) contrasting word recall ability from categorized lists among normals, left and right frontal, and temporally-lesioned subjects, concluded that the left prefrontal region is indispensable for normal strategic retrieval from verbal memory and the inhibition of interference.

Petrides and Milner (1982) demonstrated that patients with left anterior lesions had difficulties with memory for temporal order, regardless of whether the stimuli were verbal or visual-spatial, while patients with right anterior lesions had difficulty only with spatial material. Patients with temporal lesions had no difficulty on these tasks. These findings were corroborated by studies by McAndres and Milner (1991), Smith and Milner (1988), and Wiegerama, VanDerScheer, and Hijman (1990). Butters, Kaszniak, Glisky, Eslinger, and Schacter (1994) recently confirmed

discrimination deficits across a variety of conditions. Petrides (1991a,b) demonstrated that in primates, lesions confined to the middle sector of the dorsolateral frontal cortex cause a striking impairment in the ability to monitor self-generated responses which appears to implicate memory for temporal order. Further, Petrides, Alivisatos, Meyer, and Evans (1993) demonstrated in a PET study with normal controls that the mid-dorsolateral cortex was also primarily activated during a task where subjects were required to monitor numbers they generated. Of further interest, there were no statistical differences in the degree of activation where the numbers were provided by the experimenter along with the instruction to monitor. The authors suggested that the critical feature may be the process of monitoring rather than whether the stimuli were internally or externally generated. In another study, Petrides, Alivisatos, Evans, and Meyer (1993) it was demonstrated that the monitoring of self-generated choices from a set of abstract designs involved greater activation of the right mid-dorsolateral frontal cortex than the left. Where the stimuli are numerical or verbal, the reverse is evident. Frith, Friston, Liddle, and Frackowiak (1991a) had normal controls in a PET study make a series of responses to a sequence of stimuli. For the routine acts, each response was completely specified by the stimulus. For the willed acts the response was open-ended and involved a deliberate choice. The acts involved speaking a word or lifting a finger. The willed conditions were associated with activation of the mid-dorsolateral frontal cortex. The experimenters interpreted this as evidence that the mid-

dorsolateral frontal cortex is responsible for volitional behavior. Petrides, Alivisatos, Meyer, and Evans (1993) commented that monitoring was involved in this study and that in itself could account for the activation, since in their study, activation was present where monitoring in working memory was required whether the task elements were self-generated or experimenter generated.

Petrides and Milner (1982), and Petrides (1985a,b, 1990) have shown that frontal lobe patients are deficient in conditional associative learning tasks. In this experimental paradigm subjects first learn associations between two sets of stimuli, e.g., abstract designs are paired with lights of differing colors. Once the associations are learned, subjects are required on the critical experimental trials to identify the particular colored lights that had been previously paired with particular designs. In non-human primates it has been demonstrated that cytoarchitectonic areas 46 and 9 play a critical role in performing non-spatial self-ordered tasks (Petrides, 1991a,b). Petrides has shown that lesions of areas 46 and 9 do not significantly affect performance on conditional tasks. In contrast, however, lesions located just posterior to the mid-dorsolateral frontal cortex have a devastating effect on conditional tasks (Halsband & Passingham, 1982; Petrides, 1982, 1985a), but do not affect performance on the self-ordered tasks. Petrides, Alivisatos, Evans, and Meyer (1993), in a PET study, demonstrated the same relationships to hold in humans. They concluded that these adjacent areas involve two distinct functional systems. The mid-dorsolateral frontal cortex

(areas 46 and 9) being responsible for certain aspects of working memory and the adjacent area posterior to this region being an essential component of a neural circuit that mediates an aspect of behavioral responses involving the selection of appropriate stimuli depending on particular environmental contingencies. These authors also construe this as evidence of the specific modularity of the prefrontal cortex.

In a working memory PET study, Paulesu, Frith, and Frackowiak (1993) found, consistent with the findings of Frith et al. (1991b), that phonological store appeared to be primarily related to temporal supramarginal gyrus (left) and that Broca's area (44) is critical to a subvocal rehearsal system. However, Grasby, Frith, Friston, Bench, Frackowiak, and Dolan (1993), contrasting subspan (5 words) and supraspan (15 words) memory tasks, noted that increases in activation in the supraspan condition were maximal in the dorsolateral prefrontal cortex bilaterally. These authors proposed that the bilateral increases in activation of prefrontal cortex relate to the organization and use of strategies for dealing with larger amounts of verbal information. Thus, the prefrontal cortex appears to be critically involved in a number of higher-order, cognitive functions, including working memory, language production and perception, divergent thinking, e.g. verbal fluency, memory for temporal order, and conditional associative learning.

Several studies have confirmed planning and mental programming deficits in patients with frontal lobe lesions (Alivisatos, 1992; Karnath, Wallesch, & Zimmermann, 1991; Owen, Downes, Sahakian, Polkey &

Robbins, 1990; Vilkki & Holst, 1991). Vilkki (1992) compared deficits in these areas in patients with frontal lobe injuries and those with traumatic head injury. He found them to be similar and in contrast to patients with posterior lesions.

Over the past decade there has been extensive work related to impaired executive function in Parkinson's disease (for reviews, see Brown & Marsden, 1990; Dubois, Pillon, Agid, & Boller, 1991; Rashin, Borod, & Tweedy, 1990). Of particular interest here is the performance on frontal lobe tests of such patients on and off L-Dopa. For example, Lange et al. (1992) found that patients' performance following L-dopa withdrawal selectively impaired performance on the Tower of London Test, a test of spatial working memory and a measure of attentional set-shifting. In contrast, no exacerbations were noted on memory and paired associate learning deficits. These findings permit hypotheses related to test performance and deficiencies in neurotransmitter systems.

As well, the substrate of cognitive flexibility has been examined in Parkinson's disease (Richards, Cote, & Stern, 1993). These authors were able to demonstrate that Parkinsons' patients have no difficulty on the Stroop Color-Word Test (Stroop, 1935) which requires the ability to inhibit distraction by stimuli that compete for attention. However, their performance on the Odd Man Out test (Flowers & Robertson, 1985) which requires set shifting was deficient, thus identifying a specific deficit as opposed to generalized cognitive or attentional deficit.

Eslinger and Grattan (1993) add another dimension to the cognitive flexibility issue. They draw a distinction between reactive flexibility which requires set shifting in relation to external demands to manage changing circumstances and attain long-term goals (such as required by the Wisconsin Card Sorting Test or the Odd Man Out test noted above) and spontaneous flexibility, such as required by divergent production tests, e.g., verbal fluency, design fluency or, in general, measures which require the generation of a diversity of responses. In this regard they contrasted patients who had basal ganglia lesions and patients with frontal lobe lesions. They found both groups had a similar degree of impairment in reactive flexibility, but the frontal lobe group was markedly poorer with spontaneous flexibility, providing some hypotheses as to the relative contribution of basal ganglia and frontal lobe structures to different aspects of cognitive flexibility.

The work of Cicerone, Lazar, and Shapiro (1983), Shallice (1982), Smith and Milner (1984), and Shallice and Evans (1978) indicated that generating hypotheses and estimates in the context of problem-solving poses particular difficulties for frontal lobe patients. Other deficits include difficulties making frequency (Smith & Milner, 1988) or recency judgements (Milner, Corsi, & Leonard, 1991). Similar monitoring deficits described as capture errors have recently been demonstrated in the control of sequencing tasks by Della Malva, Stuss, and Willmur (1993). Also, deficits associated with impulsivity have been noted in patients with frontal lesions (Miller, 1992; Wilkins,

Shallice, & McCarthy, 1987) and with traumatic brain injury (TBI) (Parasuraman, Mutter & Molloy, 1991).

Several investigations of an adaptation of the Delayed Response (DR) and Delayed Alternation (DA) tests with clinical groups, e.g., Huntington's Disease, Korsakoff Syndrome, Broca's Aphasia (Oscar-Berman, Zola-Morgan, Oberg, & Bonner, 1982), bilateral prefrontal lesions, anterior communicating artery disease (Freedman & Oscar-Berman, 1986a), as well as Parkinson's disease with and without dementia (Freedman & Oscar-Berman, 1986b) have generally found that, as is the case with non-human primates, diseases which affect the dorsolateral convexity of the prefrontal cortex affect DR performance, and those that affect the orbital/medial aspect of the prefrontal cortex affect DA, and where both sites are damaged by the disease, both DR and DA are affected. For example, Freedman et al. (1986a) contrasted patients with frontal lesions, amnesic patients, Korsakoff patients, and alcoholic controls. All subjects had also been administered the Wisconsin Card Sorting Test (WCST). Remarkable correlations were obtained between DA and the WCST ($r = .72$, $p < .05$), but especially with DR and the WCST ($r = .86$, $p < .01$). Frontal patients were dramatically poorest at DR. Oscar-Berman, McNamara, and Freedman (1991) suggest that dopamine may be critical for DR and that cholinergic innervation of orbital prefrontal cortex may be critical for DA.

Anticipatory behavior deficits have been examined in closed head injury (Bleiberg, Freedman, Schueneman, Merbitz, & Swartz, 1985) and their

deficits have been compared to patients with cerebral vascular accident (CVA) (Freedman, Bleiberg, & Freedland, 1987). These studies demonstrated that patients with TBI had severe difficulties performing a simple avoidance task, i.e., a light signalled that an 87 decibels white noise would follow 5 seconds later. To avoid the irritating noise, subjects had to push a lever from left to right within 5 seconds of the light being presented. However, subjects had no difficulty with escape behavior when the light and noise were paired, but had difficulty mediating the contingency over a 5 seconds delay. Freedman et al. (1987) found that the TBI group was much more impaired on this task than CVA patients, perhaps because of more frontal lobe involvement in the former group. Rothke, Bleiberg, and Freedland (1987) examined the correlations between performance on the avoidance task and neuropsychological measures with TBI patients from the two studies above. They found correlations of .72 ($p < .001$) with the Halstead Category Test, -.55 ($p < .001$) with Performance IQ, and -.47 ($p < .01$) with the Memory Quotient of the Wechsler Memory Scale.

Lezack (1982) has complained that neuropsychologists have focused on cognitive assessment often to the point of ignoring executive functions. Shallice and Burgess (1991) also have pointed out that the implicit approaches adopted in the design of neuropsychological tests perhaps provide too much structure. They note that in performing these tasks patients typically are presented with a single explicit problem to tackle at one time, the trials tend to be short, initiation is strongly prompted by the

examiner, and that trial success is usually clearly characterized. As such, planning over an extended period of time is rarely required nor is priority setting required in the face of competing task demands.

They demonstrated, using an open-ended testing procedure which required the execution of tasks of modest difficulty containing some interdependencies and stretched over an extended period of time, that even frontal lobe patients who did well on neuropsychological tests did poorly on this sort of ecologically representative type of task. They proposed that a key deficit in these patients could be called a strategy application deficit (see also Goldstein, Bernard, Fenwick, Burgess, & McNeil, 1993).

Above, a brief overview and examples, especially of cognitive deficits associated with frontal lobe lesions, have been presented. Research related to clinical measures of brain dysfunction, including putative measures of prefrontal dysfunction, will be further addressed in Chapter III where the measures employed in the present research are reviewed. In the next section, the effects of such lesions on personality functioning are considered.

Prefrontal Syndrome - Personality Effects

Parallel to the differential effects of dorsolateral and orbital/medial lesions on cognitive abilities, two general patterns of personality change have been noted in the literature (Stuss & Benson, 1986). Further to their review of such personality effects, Blumer and Benson (1975) noted that the dorsolateral convexity pattern was often characterized by apathy and

indifference which they referred to as a pseudodepressed syndrome and concluded that the most salient features of the orbital/medial pattern were tendencies toward puerility and euphoria which they termed pseudopsychopathic. Despite these prototypical characterizations, they noted that actual clinical presentations usually consisted of some admixture of traits and symptoms derived from both prototypes. In accord with this basic dichotomy, Fuster (1989) refers to an apathetic and an euphoric syndrome. The former syndrome, he states, typically involves low awareness, lack of initiative, hypokinesis, and a generalized blunting of affect and emotional responses.

It is difficult to disentangle the extent to which cognitive deficits impact on affectivity and emotionality. However, with respect to apathy consequent to dorsolateral lesions, it may be reasonable to speculate that such blunting could be the result of a loss of executive function and a corresponding lack of a sense of agency. Brown (1985) has put forth the view that actions elaborate intentions and "the feeling that one is an agent who acts on an environment" (p. 38). He concluded that a sense of the future with accompanying anticipation and forward growth was vital as it effected a sense of intentionality. In his view, this loss may be the most critical deficit produced by prefrontal lesions. He concluded "The loss of this active or volitional relation to the world is ultimately, the most profound effect of damage to the frontal lobes" (p. 38).

Duncan (1986) has noted that the result of frontal damage has been described as behavior that has lost its active character becoming a passive reaction to environmental inputs. In Fuster's (1989) model, the prefrontal cortex represents action in its broadest sense and drives perception in the action/perception cycle. Possibly, characteristic reductions in drive, experienced emotion, and a passive attitude can be partially understood in terms of a loss of the capacity to experience actions in a future context. To the extent that such a shift toward a passive mode results in reduced monitoring and active analysis of potentially critical contingencies, risks for maladaptive behavior may increase.

Stuss and Benson (1986) have reviewed several studies which have found an association between left prefrontal lesions and depression. In fact, the more anterior the lesion, the greater the level of depression. The causal path, however, remains unclear. Similarly, Grafman, Vance, Weingartner, Salazor, and Amin (1986) noted that Vietnam veterans with penetrating head injuries affecting the left dorsolateral region reported more persistent anger and hostility than others with lesions in other cortical regions.

Whether considered as a result secondary to cognitive-executive deficit resulting in reduced motivation, or more direct effects on personality function, including affective and emotional responsivity, the effects of prefrontal dorsolateral lesions can be exceedingly destructive and can have a pervasive impact on psychosocial adjustment (Stuss & Gow, 1992; Stuss, Gow, & Hetherington, 1992; Prigatano, 1992).

In contrast with dorsolateral lesions, Luria (1969) described the effects of orbital lesions as producing marked personality changes. "The impulsiveness, uninhibitedness, and inadequately critical attitude of these patients may lead to disturbances in their actions . . . and to significant disturbances in their intellectual processes which become impulsive and uncontrollable in character" (p. 752).

Fuster (1989), as noted above, highlighted the elevated mood that sometimes is associated with orbital lesions, but he has also focused on irritability, distractibility, impulsivity, hyperactivity, drive disinhibition and inappropriate social conduct as common sequelae. He also pointed out the improper social conduct of such patients, stating that "They may show . . . a blatant disregard for even the most elementary ethical principles" (p. 143).

Stuss and Gow (1992) reviewed and compared the effects of frontal lobe injury with those of TBI and concluded that descriptions were dramatically similar although the neuropathology is not typically confined to the prefrontal cortex in the case of TBI. It is not plausible to think in terms of focal frontal lobe brain damage after TBI (cf. Newcombe, 1982), and diffuse axonal injury particularly in brain stem regions has often been cited as primary, though this remains an area of controversy (Prigatano, 1992; Stuss & Gow, 1992). Prigatano (1992) has reviewed the personality disturbances associated with TBI and summarized these as: a) emotional and motivational disturbances, including irritability, agitation, anger, and episodic dyscontrol; b) emotional lability and rapid mood changes, including aspontaneity and loss of interest

in the environment; and c) depression, anxiety, sensitivity to stress, catastrophic reaction and unawareness of deficits. This review is strikingly similar to the conclusions of Stuss, Gow, and Hetherington (1992) who have analysed the effects of damage to frontal systems in terms of pathological changes in drive, mood, affect, self-reflectivity, and syndromes of confabulation. They suggest that confabulation, hypomania, and irritability are predominantly associated with right hemisphere lesions.

Inappropriate social conduct may imply an empathic deficit which could be produced by prefrontal damage affecting a variety of psychological processes. In certain instances, a failure to grasp the contingencies at a cognitive level (e.g., if hurtful to me . . . then hurtful to other) may be responsible. In other instances, a generalized disinhibition of primary drives could be understood to override normal (acquired) inhibitory controls and produce inappropriate conduct. An analysis of patient EVR (Eslinger & Damasio, 1985) and a subsequent study in which patient EVR was included, Damasio, Tranel, and Damasio (1990) provide hypotheses and experimental evidence concerning the possible mechanisms implicated.

Patient EVR was, at age 35, a successful professional, described by Damasio et al. (1990) as "a happy, married father of two, who led an impeccable social life and was a role model to younger siblings" (p. 81). He developed an orbitofrontal meningioma and in order to achieve its successful surgical resection, a bilateral excision of orbital and mesial cortices was performed.

Extensive neuropsychological testing further to surgery indicated intact neuropsychological abilities with Verbal IQ at the 97th percentile and Performance IQ at the 99th percentile. On Wechsler memory tasks, his performance was also at the 99th percentile.

Measures characteristically sensitive to prefrontal damage, including the Wisconsin Card Sorting Test, the Halstead Category Test and cognitive estimations, including recency and frequency, posed no problem for him. In an interview situation, Eslinger and Damasio (1985) noted that he could reason through subtle hypothetical social problems and displayed a sophisticated understanding of world affairs and of moral and ethical dilemmas.

By contrast, his social judgement and personal organization deteriorated. He became involved in a home building partnership with a man of questionable reputation, and despite warnings by family and friends, invested all his savings. The venture failed and ended in bankruptcy. He subsequently drifted through several jobs, being fired for tardiness and disorganization, although his skills, manners and temper were appropriate.

His personal life also deteriorated. His wife of 17 years divorced him, he subsequently remarried, but this relationship also ended in divorce. Eslinger and Damasio (1985, p. 1737) characterized his condition as "acquired sociopathy". Damasio et al. (1990) reasoned that removal of orbitofrontal cortex likely produced a disconnection between dorsolateral cortex and limbic structures that affected "two kinds of regulatory activity:

1. 'Modulation' of innate hypothalamic drives that are informed with regard to environmental rules and contingencies.

2. Activation of higher cortices by basic drives and tendencies (p. 1740)."

Damasio et al. (1990) contrasted the skin conductance responses (SCR's) of 5 normal controls, 5 patients with bilateral lesions in orbital and lower medial regions with severe deficits in social conduct, judgement and planning and 6 patients who had lesions outside the ventromedial frontal cortices and no acquired defects in social conduct. The paradigm involved recording SCR's to neutral (non-target) slides and socially charged (target) slides depicting social disaster, mutilation, and nudity under two conditions. In the first condition (PASSIVE) the subject simply viewed the target and non-target slides. This condition was followed by a second condition (ACTIVE) where the subject was required (following the presentation of each slide in the same order) to give a verbal response to each picture.

The basic outcome of the experiment is captured by patient EVR's response pattern. His orienting SCR's were similar to normal and brain damaged controls. His SCR's under the ACTIVE condition were also normal, however, there was virtually no response in the PASSIVE condition. Damasio et al. (1990) account for this non-response as a defect due to "an inability to activate somatic states linked to punishment and reward, that were previously experienced in association with specific social situations and that must be reactivated in connection with anticipated outcomes of

response options" (p. 81). They further speculate that failure to reactivate, in their terms, 'somatic markers' would deprive someone "of an automatic device to signal ultimately deleterious consequences relative to responses that might nevertheless bring immediate reward (or alternatively, signal ultimately advantageous outcomes relative to responses that might bring immediate pain)" (p. 81).

These results are compelling and are consistent with other case reports, e.g., Malloy et al. (1993) and Meyers, Berman, Scheibel, and Hayman (1993) that have noted a psychopathic behavioral outcome further to circumscribed orbital (particularly left) lesions. As well, these findings fit well with the theoretical analysis of neural mechanisms of emotion put forth by Tucker and Derryberry (1992) and Derryberry and Tucker (1992) which attributes to the orbital prefrontal cortex a central common pathway for emotional influences by integrating and representing the regulatory influences of lower limbic and paralimbic systems. In their view the prefrontal cortex, in conjunction with the cognitive-perceptual representations of prefrontal cortex, provides a basis for executive functioning. In their formulation the main contribution of the orbital cortex is to provide the directed attention, motivation, and self-control necessary for adaptive functioning.

Above, two distinct groupings of personality effects attributable to lesions of the prefrontal cortex were discussed. First, a syndrome reflecting apathy, blunted, and constricted affect, reduced motivation and personal

inadequacy which were maximally associated with dorsolateral lesions was identified. Secondly, a syndrome more typically associated with orbital/medial lesions, reflecting disinhibition of basic drives, impulsivity, hyperactivity, distractibility, periodic euphoria, but also irritability and social insensitivity, was discussed. This latter syndrome appears to involve neglect, indifference, or non-consideration of affective-emotional factors in behavioral planning that can result in social misconduct to the degree that it has been labelled acquired sociopathy by Damasio et al. (1990). These personality effects appear to be produced by the dissolution of previously acquired integrated intellectual and emotional processes possibly through disruption of pathways mediating these processes.

Given that damage to the prefrontal cortex in adults can severely impact the developed personality, questions arise regarding the effects of early damage to the prefrontal cortex and indeed as to its role in development. These are the concerns of the following section.

## SECTION III PREFRONTAL CORTEX IN PSYCHOLOGICAL DEVELOPMENT

### Introduction

The role of the prefrontal cortex in psychological development has recently captured considerable interest. This section begins by reviewing this development, and associated experimental and clinical test findings. Case studies are presented and some conclusions tendered.

Prefrontal Cortex - Developmental Considerations

Although certain authors, e.g., Russell (1948) attributed a critical role to the prefrontal cortex in development, the more common view, until recently (e.g., Golden, 1981; Bolter & Long, 1985) has been that the prefrontal cortex only begins to function at some point in early adolescence. Dennis (1991) observed that the role of the immature prefrontal cortex has been ignored or regarded as largely non-functional. She attributed this to a lack of clinical research, an incomplete reading of developmental neuroanatomical data, a focus within a hierarchical perspective on the most evolved aspects of the prefrontal cortex, and to a lack of a developmental perspective. As noted by Segalowitz and Rose-Krasnor (1992), the situation has recently changed dramatically and developmental neuroscience has progressed so far that ". . . . we are now beginning to map the role of frontal lobe maturation onto normal psychological development" (p. 2).

Experimental and Clinical Test Findings

This section begins with a review of the developmental work of Goldman-Rakic and that of Diamond which together appear to have kindled a keen and increasing interest in the role of the prefrontal cortex in human development. Further to this, recent experimental data corroborating the role of the prefrontal cortex early in development, as well as theoretical perspectives are discussed. Next, studies which have attempted to trace the acquisition of prefrontal capacities will be examined. Following this, general views as to the impact of early prefrontal lesions will be discussed and

illustrative case studies of children who sustained early prefrontal damage will be presented.

Noting the remarkable parallel between the delayed response task and the Piajetian $A\bar{B}$ task, a measure of stage IV Object Permanence, Goldman-Rakic and Diamond have conducted experiments which have established the functional equivalence of these two tasks for both human infants and infant rhesus monkeys (Diamond, 1985, 1988, 1990a,b; Diamond & Doar, 1989; Diamond & Goldman-Rakic, 1985, 1986, 1989; Goldman-Rakic, 1987a).

In the $A\bar{B}$ (pronounced A-not-B) paradigm, the child watches as a reward is hidden in one of two spatially separated locations. This is followed by a delay of several seconds and then the child is permitted to find the reward. Thus, within an individual trial the task appears to be formally equivalent to the delayed response task described earlier. There are however, certain procedural differences. For example, in the $A\bar{B}$ task, the sequence of events (especially where the toy will be hidden) is under the control of the experimenter, whereas in the delayed response task the sequence from trial to trial is typically predetermined according to a quasi-random schedule.

As well, in $A\bar{B}$ , the tradition has been to keep the toy at the same location until it is first discovered by the child while in delayed response the action of the reward is quasi-random from the outset. Other differences include the fact that the child is sitting in mother's lap in $A\bar{B}$ , while the subject in delayed response is caged. The reward, in $A\bar{B}$ has been a toy, while in delayed response, it has been food. In $A\bar{B}$, the experimenter has

typically distracted the infant during the delay, while in delayed response, an opaque screen is interposed between the subject and the reward.

Diamond and Goldman-Rakic (1989) compared both rhesus monkeys and human infants on the $A\bar{B}$ task, and Diamond and Doar (1989) compared both rhesus monkeys and infants on delayed response. The functional equivalence of the two tasks appears beyond doubt as both the type of errors and the age X delay curves are nearly identical. In the case of rhesus monkeys, ablation experiments demonstrate that an intact dorsolateral prefrontal cortex is required to accomplish either task.

By way of comment, it is recalled that the delayed response task illustrates Fuster's (1989) theory in microcosm. Thus, the essential equivalence of the delayed response task to the Piajetian $A\bar{B}$ task situates Goldman-Rakic's and Diamond's work squarely within his theory and therefore may serve to link an essential role of the prefrontal cortex in development with its role in adult functioning. In these studies it has been found that the developmental progression that occurs between 7.5 months and 12 months in human infants (from not being able to mediate a delay to being able to mediate a 10 second delay on $A\bar{B}$) is compressed in infant rhesus monkeys to between 2 and 4 months of age. Goldman-Rakic (1987b) has determined that the period in infant monkey between 2 and 4 months of age corresponds to a period where synaptic density in the principal sulcus is at its peak. Also Lidow, Goldman-Rakic, and Rakic (1991) have found that in the postnatal development of neurotransmitter systems in

the monkeys' cortex, monoaminergic, cholinergic, and GABAergic receptors reach maximum number and concentration between 2 and 4 months, then decline to mature levels. This rise and fall is synchronous for all receptor and cortical areas investigated, including the prefrontal cortex. The corresponding period of synaptic excess in humans begins around 8 months and reaches a maximum at 2 years of age. They note, however, that the improvement in the delay tolerated is not likely to be a simple function of synaptic density since density remains relatively constant in the monkeys over the 2 to 4 month period. These authors hypothesized that factors responsible for functional maturation, in addition to gradual synapse elimination, are likely continued myelination, further regulation of receptors, biosynthesis of neurotransmitters and peptides, as well as improved synaptic efficiency at the molecular level.

Between the age of 2 and 4 months, in addition to the ability to perform $A\bar{B}$ and delayed response, the infant monkey also develops the capacity to reach for an object in a direction away from the line of sight. Goldman-Rakic (1987a) believes that the common requirement in these two tasks is the ability to guide response choice by stored information or internal representation. She sees this capacity to be "a building block, if not a cornerstone of cognitive development" (p. 380) and the central role of the prefrontal cortex is to guide voluntary behavior on the basis of an internal representation. This represents the foundation of working memory. Goldman-Rakic (1990) elaborates on its importance:

> The significance of working memory for higher cortical function is not necessarily self-evident. Perhaps even the quality of its transient nature misleads us into thinking it is somehow less important than the more permanent archival nature of long-term memory. However, the brain's working memory function, i.e., the ability to bring to mind events in the absence of direct stimulation, may be its inherently most flexible mechanism and its evolutionarily most significant achievement. Thus, working memory confers the ability to guide behavior by representations of the outside world rather than by immediate stimulation, and thus to base behavior on ideas and thoughts. (p. 333)

Diamond (1990a, 1990b) has distinguished the inhibition component from the short memory component as these are joined in the $A\bar{B}$ task and has shown an increase in the ability to inhibit in human infants between 8 and 12 months which seems proportional to the increases in short-term memory over the same period. In Diamond's (1991a,b) view, the capacity to inhibit the predominant response permits choice and control over action and thus makes possible the emergence of intentionality and the capacity to construct relations.

Thus Piaget's classical insight into the development of intentionality appears to be explicated in terms of prefrontal function. In Piaget's (1952) scheme, Stage IV's "coordination of secondary schemes", the infants truly begin to combine schemes in complex ways. In particular, planning and

intentionality emerge. This is the beginning of means-end behavior. Old schemes can now be applied to new situations. The capacity for anticipation is nicely illustrated by Piaget's observation:

> She likes the grape juice in a glass, but not the soup in a bowl. She watches her mother's activity. When the spoon comes from the bowl, her mouth remains closed. Her mother tries to lead her to make a mistake by taking a spoon from the bowl and passing it by the glass before offering it to Jacqueline. But she is not fooled. (p. 249)

Goldman-Rakic and Diamond, through their demonstration of the functional equivalence of delayed response and Piaget's $A\bar{B}$ task, and the apparent mutual dependence of successful resolution of these tasks upon the developing prefrontal cortex provide a conduit, linking traditional developmental theory to current views related to the functions of the developing prefrontal cortex.

Further evidence that the prefrontal cortex is critically involved in the $A\bar{B}$ task comes from electrophysiological (EEG) measures of prefrontal activation (Bell & Fox, 1992; Fox & Bell, 1990). In these studies the pattern of EEG data, particularly in the frontal region, was strongly associated with performance on the $A\bar{B}$ task. Infants who exhibited an increase in their ability to tolerate delay displayed a corresponding increase in frontal EEG activity. Infants who were unable to tolerate increased delay on the $A\bar{B}$ task did not display the corresponding frontal effects.

Involvement of the prefrontal cortex in mediating emotional behavior in infants has also been demonstrated with EEG indices. For example, Dawson, Panagiotides, Grofer Klinger, and Hill (1992), in an EEG study of 21 month old infants, found that generalized increases in frontal lobe activation occurred during high emotional arousal with hemispheric asymmetries correlated with the infants' capacity to mobilize sequentially organized regulatory behavior during states of high negative emotional arousal. These authors attribute increased prefrontal activation to cortical influences on subcortical structures. Campos, Campos, and Barrett (1989) have characterized emotions as processes of establishing, maintaining, and/or disrupting the relations between the person and the internal or external environment. Thus, regulatory processes, action tendencies, and coping strategies are perceived as integral components of, rather than responses to, emotions. This focus on relatedness especially implicates the prefrontal cortex in emotional development given its critical role in mediating relationships.

Thatcher (1991, 1992a, 1992b) has charted EEG changes from early infancy through adolescence and noted a number of growth spurts in EEG coherence involving prefrontal cortex. He believes that the cyclical pattern recognized during development reflects a dialectical process which iteratively and sequentially reorganizes intracortical connection systems. He describes the process as a spiral staircase in which intracortical connection systems are reorganized each time the spiral sweeps around forming successively

higher levels of integration. Thatcher (1991) concluded that "the unfolding and elaborations of connections with the different zones of the frontal lobes seems to be a dominant feature of human cerebral development and this process appears to occur in stages or as a discontinuous function of age" (pp. 416-417). Thatcher (1991) has suggested that this process can be visualized by:

> . . . . the metaphor of a symphony orchestra in which the conductor of the symphony is the frontal lobes and the nonfrontal regions of the cortex represent the various musical sections of the symphony: the wind section, the string section, the percussion section, and so on. Human development is where a certain amount of maturation is required before a part or whole section of the orchestra comes on line connecting with the conductor resulting in the evolution of richer and deeper music at each step. In humans, the various sections of the nonfrontal cortex come on line by sequentially establishing connections with the frontal lobes at different ages until at adulthood the full cerebral ensemble is in orchestration. (p. 417)

Case (1992) has reviewed the developmental literature concerned with attention (working memory), executive, and self-reflexive processes. Within a neo-Piajetian framework she identified four major stages of cognitive development characterized by a four-phased, recursive process within each stage (see also Lewis, 1994). She then compared cognitive growth cycles with Thatcher's EEG growth changes and noted a close parallel, both in

terms of the development sequence, i.e., time line, and in the recursive dynamic proposed for producing movement through each stage. She argues that the two sets of changes, i.e., cognitive and EEG, are different manifestations of a common underlying process which is frontally mediated.

While Case has related data from developmental psychology to EEG parameters of physiological change, Welsh and Pennington (1988) have identified several areas where developmental research could be accommodated within a theoretical framework of prefrontal development, particularly in the areas of self-control behavior, problem solving, and metacognitive functioning. Notwithstanding, given the older view that prefrontal function does not come on stream until early adolescence, few studies, until recently, have specifically addressed the parameters of executive skill development. Becker, Isaac, and Hynd (1987) and Passler, Isaac and Hynd (1985) adapted some Lurian frontal tasks for use with school age children. They noted qualitatively different levels of performance at different ages and concluded that the development of prefrontal functions is a multistage process. For example, on certain tasks children at 6 years of age demonstrated some capacity for flexible strategic behavior whereas on other tasks perseveration was observed in 12 year old children. Chelune and Thompson (1987) demonstrated that the Wisconsin Card Sorting Test could be clinically sensitive in children as young as 7 and that adult-like performance levels were achieved by children aged 10 to 12. Casey, Bronson, Tivnan, Riley, and Spenciner (1991) demonstrated that abilities in

sequential planning ability were distinct from measures of intelligence in children as young as 4 and 5 years of age. This is consistent with the notion of the early expression of specific prefrontal functions.

Welsh, Pennington, and Groisser (1991) assembled a battery of putative prefrontal measures designed to tap executive processes and administered these to subjects ranging in age from 3 to 12, and to a 28 year old contrast group. Factor analysis yielded three factors which reflected speeded responding, set maintenance, and planning. They noted three age levels (6 years, 10 years, and adolescence) at which different aspects of executive tasks could be performed at adult levels.

Levin et al. (1991) also examined the development of executive abilities in a sample of children aged 7 to 15. Their battery had some test overlap, e.g., Wisconsin Card Sorting Test, with that of Welsh et al. (1991) and specifically targeted problem solving/concept formation, memory tasks, and verbal and design fluency. Factor analysis of this battery produced a three factor solution with factors related to semantic association/concept formation, freedom from perseveration, and planning/strategizing. They also noted major differences in ability between 7 and 8 year old children compared to 9 to 12 year old children and also noted further advances in the 13 to 15 year old group, particularly on the more complex tasks such as the Tower of London task. Thus, these two studies provide strong evidence that prefrontal skills develop in a stage-like manner throughout childhood. Further, as noted by Welsh et al. (1991), these skills appear to be relatively

uncorrelated with intelligence. Although recommended by Levin et al. (1991), measures of prefrontal skills have not as yet been systematically examined in children with prefrontal lesions and a taxonomy for classifying executive function largely remains to be developed and refined (cf. Welsh et al., 1991). A review of the clinical case literature describing the deficits and outcomes associated with early prefrontal lesions provides broad indications of the importance of an intact prefrontal cortex for normal development.

Case Studies

Benton (1991b) has noted the "extreme paucity of suitable case material" (p. 276) for understanding the consequences of early prefrontal damage and for developing theory regarding its role throughout development. Very recently, more cases have been presented emphasizing different effects, but on the whole a fairly consistent phenomenological picture emerges and conclusions can be tentatively drawn at least as to the range of impacts.

Perhaps the most famous prefrontal case is Ackerly's patient J.P. (Ackerly, 1964; Ackerly & Benton, 1947; Benton, 1991b). J.P. exhibited bilateral prefrontal lobe atrophy purportedly as a result of a congenital and idiopathic degenerative process, although Ackerly (1964) also refers to a head injury at age 4. The patient was born in 1912 and assessed by Halstead, among others, and followed until he was 50 years of age in 1962. Benton (1991b) summarized his notable characteristics as having average general intelligence, lacking anxiety and concern, exhibiting marked

impulsivity, displaying unexpected and inappropriate sexual conduct, and being boastful yet polite which Ackerly (1964) described as Chesterfieldian Manners. Ackerly (1964) considered his aloneness, despite superficial sociability, as a primary social defect and emphasized his incapacity for emotional relationship. It is also noteworthy that as a child he displayed quite serious behavioral difficulties, including bullying, stealing, running away, poor school work, although he was an excellent speller and fluent talker. His classroom behavior was clearly abnormal. Benton (1991) reported that he masturbated openly in class and on one occasion defecated in a classmate's glove and then carefully replaced the glove in the child's coat pocket. Ackerly (1964) emphasized his lack of developmental progression, as he put it, "experience is not the best teacher with him" and "'out of sight, out of mind' appears to be the core of his defect" (p. 203). Neuropsychological testing indicated an inability to perform the Halstead Category Test and the Wisconsin Card Sorting Test. He was described as locked in the present without reference to the past or future. Between 1932 and 1944 he was arrested 15 times. Most arrests were related to disorderly conduct charges, three drunk charges, and two automobile thefts. The tragic nature of this case seems expressed by Ackerly (1964) who noted J.P.'s ". . . . detachment . . . from . . . anything that gave meaning to life, love, friendship, comradeship. He is indeed a veritable stranger in this world with no other world to flee for comfort" (p. 204).

Mateer and Williams (1991) presented four cases; SS, MB, JB, and RD who had sustained traumatic frontal lobe injury between 3 and 9 years of age and were followed up for 3 to 7 years. Comparing the four cases, she noted that consistent patterns of cognitive and behavioral dysfunction emerged. Despite an unremarkable pre-injury history and no subsequent impairment in IQ, all four children exhibited impulsivity, distractibility, and were described as irritable, temperamental, and moody. As well, all four exhibited attentional impairment and academic production deficits, three were overactive and exhibited academic achievement deficits, and all exhibited significant social problems. Mateer and Williams (1991) description of deficits bear a striking resemblance to those exhibited by children with Attention Deficit Hyperactivity Disorder and this relationship will be reviewed in some detail later.

Price, Daffner, Stowe, and Mesulam (1990) reported on two adults with severe behavioral disorders further to early damage to prefrontal cortex. In the case of G.K., who was a 31 year old male, bilateral prefrontal damage had been incurred at 1 week of age. Adult Magnetic Resonance Imaging (MRI) revealed bilateral lesions extending from cortex to the caudate nuclei, more on the left than the right. There was a mild exvacuo enlargement of the right lateral ventricle. Neuropsychological assessment indicated normal IQ (Full Scale IQ 96) with severe deficits noted on attentional, organizational, and mental flexibility skills. Piajetian development was limited to concrete operational thinking and Flavel's (1968) perspective taking task suggested an

egocentric perspective. Moral development was assessed to be at Kohlberg's (1976) early conventional (Stage 3) level. G.K.'s history revealed that he was always considered immature and that serious behavioral difficulties were first identified at age 8. He did not respond to parental discipline, sought immediate gratification, never developed adequate friendships, and blamed his difficulties on others. Under firm guidance and after two school transfers, he graduated from high school. He joined the Marine Corps, but was dishonorably discharged after 6 weeks. Over the following next 10 years he was hospitalized 27 times in psychiatric facilities and imprisoned 8 times on charges of assault, forgery, grand larceny, drug involvement, and lewd behavior. He was also charged with arson of two public buildings. He was an active bisexual, often traded cigarettes for oral sex, and masturbated in public. In hospital, he was suspected of raping 2 female patients.

Price et al. (1990) emphasized severe deficits of comportment, judgement, and insight despite no evidence of limiting deficits of language, memory, or visuospatial skills in daily activities. They noted that severe early bifrontal lesions appear to lead to more chaotic and aberrant behavior than similar lesions acquired during adulthood. They suggest that early prefrontal lesions may selectively interfere with the acquisition of insight, foresight, social judgement, empathy, and abstract reasoning. Further, they speculated that a subset of criminals could have underlying bifrontal

dysfunction acquired early in life or prenatally, and that this dysfunction may not be detectable by conventional neurodiagnostic methods.

Slomka, Tarter, and Hegedus (1984) report on the disastrous developmental history of a child born with agenesis of the frontal lobes. The child presented extreme behavioral developmental issues and is mentioned here because of the extreme nature of his prefrontal structural deficit.

Marlowe (1992) reported on a 7 year old child, P.L., who sustained a penetrating injury to the prefrontal brain from a lawn dart at age 3 years 11 months. He had a depressed skull fracture in the right frontal region with lacerations to the underlying dura and cortical surface. Bony fragments, hair, and grass were imbedded 1 ½ cm deep in the brain parenchyma. Following surgery, all neurological and EEG findings have been unremarkable. Neuropsychological assessment at age 5 years, 1 month indicated a Wechsler Preschool and Primary Scale of Intelligence (WPPSI) Verbal IQ of 122, a Performance IQ of 123, and a Full Scale IQ of 119. At age 6 years, 1 month a Verbal IQ of 119, a Performance IQ of 132, and a Full Scale IQ of 128 was noted on the Wechsler Intelligence Scale for Children - Revised (WISC-R). Tactile sensibilities improved on the left hand and he was disproportionately slow with his left hand on the Tactual Performance Form Test. Also difficulties were noted with visuospatial organization and production on the Hooper Visual Organizational Test and the Osterreith Complex Figure Test. The author noted, however, that the most dramatic

changes were in self-regulatory functions qualitatively in the classroom and on neuropsychological tests.

> He had problems in maintaining a mental set: he was able to build an awareness of social and task requirements, but had extreme difficulty in maintaining it to completion. He became distracted. As a result of his impulsivity he had difficulty in inhibiting his actions long enough to execute strategic planning. . . . Although he verbally mediated a strategic plan, he was not able to utilize it to inhibit or otherwise purposefully direct his own behavior. (p. 210)

Behavioral problems were also noted:

> In the first grade he was suspended for refusing to attend class and assaulting the vice-principal when she attempted to physically confine him to the school grounds. He threw things at, kicked, hit, or cursed anyone who challenged or thwarted his impulses. He felt no remorse following these behaviors and, therefore, refused to apologize. (p. 210)

Marlow (1992) commented that the frequency, intensity, and consistency of P.L.'s emotional outbursts, while characteristic of persons with frontal lobe lesions, represented a striking change from previous personality, as well as normal development. She further noted that it is not so much the size of the lesion in this instance, but rather its frontal localization which interferes with development.

Grattan and Eslinger (1991) reported on a female, D.T., who sustained a subarachnoid hemorrhage in the left prefrontal region at age 7. When evaluated at age 33 it was revealed that her development was not particularly remarkable until early adolescence, i.e., symptoms were delayed. However, assessed as an adult she exhibited interpersonal difficulty, erratic work record, inappropriate social and sexual conduct, poor judgement, impaired management of practical matters, and little empathy. Other reports, e.g., C.L.Q. Thompson (1970), documented a history of unmanageable behavior, inattentiveness, indifference, deficient self-control, and violence, including murder by a patient who sustained a severe head injury at age 7 that had resulted in frontal lobe atrophy.

Perhaps at odds with the consistent presentation of serious consequences following early prefrontal lesions is Hebb's patient K.M. (Hebb & Penfield, 1940; Hebb, 1945) whom Hebb provocatively described (Hebb & Penfield et al., 1940) as a "strikingly easy going, carefree fellow" (p. 12), although even Hebb conceded that he demonstrated a lack of concern for the future. The term provocative is used because the case is sometimes taken to demonstrate that Hebb considered the frontal lobes to be unimportant after early development, while his concern related primarily to methodological proof of their importance. The facts of the case involved a youth (K.M.) who suffered traumatic damage to the frontal poles at age 16. Post-injury he developed a seizure disorder and a behavioral disturbance. Hebb and Penfield et al. (1940) described him as "childish, violent, stubborn,

and distractible with gross defects of memory and ordinary judgement" (p. 10). Eleven years post-injury he underwent bilateral pole resection for intractable epilepsy with dramatic improvement in behavior such that his relatives reported that "he has become normal in every way" (p. 10). However, Ackerly (1964) saw K.M. at age 49 and reported that his sister noted that "K.M. had never grown up since the accident, has always been a teenage boy in his interests and behavior except that he has never been interested in girls" (p. 211). As well, Ackerly (1964) reports that K.M.'s sister, brother, and employer noted that he needed to be taken care of "If he were alone for very long, he would not feed himself properly or even change his clothes. He certainly would not bathe" (p. 211). Ackerly (1964) also commented on the need to observe such cases longitudinally to see the full impact of frontal lesions and despite some contrary opinion from P.R. Milner, Ackerly concluded that K.M. demonstrated frontal lobe psychopathology, concluding that K.M.'s ". . . . behavior has been characteristically simple, bland, stereotyped, perseverative, and punctuated by outbursts of irritability when crossed or restricted and . . . less capable of planned productive work than he was pre-operatively, at age sixteen" (p. 218).

In their review, Grattan and Eslinger (1991) concluded that K.M. was among "the highest functioning and independent of the childhood cases" (p. 292). It is also noted that he was, developmentally speaking, quite far along given his age of 16.

Conclusions

What appears to be evident in reported cases of early prefrontal involvement is a pattern where the cognitive deficits are subtle and not tapped by traditional intellectual measures, yet their impact on behavior, personality, and social development is major. In this regard Dennis (1991) has proposed a heuristic based upon cognitive psychology to capture the developmental impact of prefrontal lesions. In addition to attention, regulation, and executive control, she emphasizes deficits in semantic representations (propositions concerned with meaning) and intentional representation (propositions concerned with the knowledge and beliefs that people entertain about themselves and each other). She noted that these knowledge-based systems may undergo further elaboration or meta-representation and are involved in monitoring the match between internal states and the external world. Her work (e.g., Dennis, 1988; Dennis & Barnes, 1990) and that of others (e.g., Chapman et al., 1992) indicate abnormal discourse patterns in head injured children which may provide some insights into the nature of the underlying deficits which contribute to the impacts on social and personality development.

The above review of case studies suggests a critical yet subtle role for the prefrontal cortex given the apparent impact of early damage on executive function, behavioral self-regulation, personality and social development. Perhaps in Hebb's (1949) terms we are now asking "the proper questions in such investigations" (pp. 287-288). Nevertheless, case studies have

limitations and a clear understanding of the qualitative parameters regarding the impacts of early prefrontal lesions on development will require further systematic research.

In the above three sections, literature related to: a) theories regarding the role of the prefrontal cortex; b) the effects of prefrontal lesions in adults on cognitive and personality function; and c) the developmental aspects of prefrontal function from the perspective of theory, research findings, and case studies has been discussed. Section IV considers literature regarding hypotheses and associated research that persistent adult criminals exhibit prefrontal and other neuropsychological deficits. In Section V, literature addressing similar hypotheses with respect to groups of children and adolescents at risk for adult offending is reviewed.

This organization of the literature review is consistent with the overarching hypothesis of the present research which considers whether neuropsychological deficit, in particular prefrontal dysfunctions, characterize adult offenders and groups at marked risk to become adult offenders, specifically, children and adolescents with Conduct Disorder (CD), Attention Deficit Hyperactivity Disorder (ADHD), or both disorders, and adolescent offenders.

## SECTION IV ADULT OFFENDERS

### Introduction

The first portion of this section reviews neuropsychological theories and empirical findings related to adult offenders. Particular reference will be

made to the work of Yeudall and his co-workers. Next, several studies which have specifically addressed the frontal lobe hypothesis of criminality are reviewed. This is followed by Gorenstein's re-interpretation of much psychophysiological research on psychopathy in terms of a prefrontal disinhibition hypothesis. Lastly, a psychological model of disinhibition is discussed.

## Neuropsychology Of Adult Offenders: Theory And Findings

### Yeudall And Co-Workers

Yeudall and his co-workers (Fromm-Auch & Yeudall, 1983; Yeudall, 1977, 1978a, 1978b, 1979; Yeudall, Fedora, Fedora, & Wardell, 1981; Yeudall & Fromm-Auch, 1979; Yeudall, Fedora, & Fromm, 1986) have extensively researched neuropsychological variables with criminal offenders. In broad terms, they have addressed the hypothesis that neuropsychological variables play an important role in persistent criminality. They argue, with reference to Tittle's (1983) analysis of social and criminality correlates, that psychosocial perspectives on criminality have difficulty accounting for criminality in normal psychosocioeconomic environments and are limited in accounting for the fact that only a small proportion of individuals become persistent criminals from any given socioeconomic or psychological milieu. As well, however, it is noted (Yeudall, Fedora, & Fromm, 1986) that the combination of lower socioeconomic status and brain dysfunction would be particularly disadvantageous, as compensatory resources, e.g., early diagnosis/early remediation, would be less likely to occur amongst such

groups. Accordingly, they hypothesized that the existence of a specific neuropsychological dysfunction interacts with the adverse environments, significantly increasing the probability of an individual developing a persistently criminal life style.

Yeudall, Fedora, and Fromm (1986) also emphasize the impact of early brain dysfunction in producing behavioral and learning deficits which increase the risk for poor adjustment. In particular, early dysfunction of neocortical limbic systems, would be expected to interfere with normal social and moral development.

In conceptualizing brain-behavior relationships of relevance to criminality, they propose a tripartite model of brain organization. The three dimensions are: a) dominant versus non-dominant hemispheric functions, b) anterior versus posterior brain functions, and c) subcortical versus cortical brain systems. The relevance of dominant hemispheric functions to criminal behavior, in these authors' view, relates to the putative role of the dominant hemisphere in regulating social behavior, particularly through the use of language and processing information in a sequential manner.

In general, in accord with Flor-Henry's (1983, 1985) hypothesis that reciprocal inhibitory/facilitory relationships exist between the hemispheres via the corpus callosum, they attribute an overall inhibitory function to the dominant hemisphere and an overall excitatory function to the right hemisphere regarding mood, erotic states, and aggressive behavior.

Yeudall, Fedora, and Fromm (1986) also propose a balanced reciprocal relationship between cortical and subcortical systems. They emphasize that damage to the brain stem and midbrain structures, particularly vulnerable in cases of TBI, can impact arousal, mood, drive, motivation, and cognitive processing, either intrinsically or through compromised neural conduction to higher cortical centers. Therefore, in their view, assessment of the integrity of these structures is important.

Anterior versus posterior cortical systems form the third dimension of the Yeudall, Fedora, and Fromm (1986) model. In this respect, they emphasize that clinical syndromes associated with dorsolateral and orbital prefrontal areas produce symptoms that ". . . . have, in many instances, a rather striking resemblance to behavioral traits of persistent criminals" (p. 122). These authors further propose that the prefrontal cortex, like the temporal cortex, is functionally divided into cognitive (dorsolateral convexity) and limbic functions (anterior and mesial portions). They consider that anatomically these structures are optimally situated for the integration of information from the external world with internal states in a way that is closely identified with the person's emotional life, his instinctive feeling and activities, as well as his visceral responses to environmental change (cf. Williams, 1969a).

To address and evaluate the various potential sources of brain dysfunction associated with persistent criminality Yeudall, Fedora, and Fromm (1986) report on the implementation of a multimodal assessment

approach. This approach involves administering neuropsychological measures to address cortical function and includes psychophysiological measures to assess limbic and basal ganglia perturbations. Averaged evoked potential measures are proposed to assess the integrity of the brainstem and midbrain thalamic region, further power spectral EEG measures under passive and active states of cognitive activity are identified as global integrative assessment measures.

While in general agreement with the need for a multimodal approach to assessment, the primary concern of this research relates to neuropsychological variables in criminal populations and this literature is reviewed below.

Despite major reviews of frontal lobe dysfunction and antisocial behavior (Kandel & Freed, 1989) and a review of the neuropsychology of the aggressive psychopath (Miller, 1987) very little research has actually been carried out in populations of incarcerated criminals. The most extensive research in this area has been done by Yeudall and his co-workers at the Alberta Hospital in Edmonton, Alberta, Canada. The subjects included a high incidence of referrals from the courts for neuropsychiatric assessment, a fact that led Raine and Scerbo (1991) to note the possibility of some pre-selection biasing towards a high incidence of neurological damage. Raine and Scerbo (1991) thus emphasized the need for independent replication of this work.

The neuropsychological test battery utilized at the Alberta Hospital consisted of an extension of the Halstead-Reitan Neuropsychological Test Battery by Dr. Yeudall to encompass measures of verbal concept formation (e.g., Wisconsin Card Sorting Test), verbal fluency tests, and verbal memory tasks (cf. Royce, Yeudall, & Bock, 1976; Yeudall, 1977).

Test profiles were evaluated clinically in each case and classified according to the following criteria:

1. Normal, borderline normal or abnormal: Normal and borderline normal profiles contained few test scores within the brain-damaged range of performance and these test scores did not form consistent patterns; in contrast, abnormal profiles contained many or all tests within the critical range or a selective number of tests which formed a consistent pattern.

2. Lateralized hemispheric dysfunction: bilateral asymmetrical hemisphere dysfunction within each abnormal profile was classified as either dominant greater than nondominant hemisphere dysfunction (D > ND), or nondominant greater than dominant hemisphere dysfunction (ND > D). Dysfunction that appeared exclusively dominant or nondominant was also classified as D > ND and ND > D, respectively.

3. Intrahemispheric dysfunction: dysfunction within the hemisphere was classified as anterior greater than posterior (A > P), or posterior greater than anterior (P > A) dysfunction. (Yeudall, Fedora, & Fromm, 1986, p. 143)

Study I: Yeudall (1978a)

Subjects in this study consisted of 25 aggressive criminals who were consecutively assessed and fulfilled Cleckley's (1976) criteria of psychopathy, and 25 aggressive criminals who were characterized as having a predominance of affective, as opposed to psychopathic symptomatology. The previous mean conviction rate for the two groups was 11.8 and 7.2 convictions respectively. They did not differ significantly with respect to age, WAIS Full Scale IQ. (97.0 vs 95.7), education or handedness.

A clinical diagnosis was made as to the existence of brain dysfunction and its asymmetrical nature based on each subjects neuropsychological profile according to the criteria listed above. This evaluation on the basis of 19 (76%) of the aggressive-psychopaths and 22 (88%) of the depressive-aggressive patients showed mild to severe impairment on test variables sensitive to brain dysfunction of the temporal and frontal regions of the brain. Of the 19 aggressive-psychopaths, 15 (79%) displayed greater dominant (D>ND) hemisphere dysfunction, whereas all 22 (100%) of the affective-aggressive patients displayed nondominant (ND>D) hemisphere dysfunction. Comparison of the two groups in regard to asymmetrical signs based on neuropsychological interpretation revealed significant results (Chi squared = 8.19, $p > .005$, $df = 1$, Phi = 44). A two-way discriminant function based on 27 neuropsychological variables (2:1 subject to variable ratio) classified 88.0% of the aggressive-psychopaths and 91.3% of the affective-aggressive criminals, whereas when based on the 11 subtests of

the WAIS, classification of the groups were 64% and 76% respectively. The combined WAIS and neuropsychological variables resulted in 100% classification of both groups. When the two aggressive groups were compared with a control group ($n = 25$) matched for age (but not for IQ and education) a three-way stepwise discriminant function classified 88.0% of the aggressive-psychopaths, 84.0% of the affective-aggressive criminals and 92.0% of the controls. These high classification rates may, however, be somewhat spurious as the subject to variable ratio was sub-optimal in these discriminant comparisons (cf. Fletcher, Rice, & Ray, 1978).

Study II: Yeudall, Fedora, and Fromm (1986)

A second study contrasted 25 criminal patients diagnosed as severe personality disorders with affective features with the aggressive-psychopathic group from Study I above. As was the case with the aggressive-depressed patients from Study I, the severe personality disorder with affective features patients in Study II had a greater proportion of lateralized deficits attributable to the non-dominant hemisphere. A comparison of the lateralized impairments displayed by the personality disorder-affective patients and the psychopaths from Study I yielded a $X^2 = 8.19 (p < .005)$. A two-way discriminant function analysis of these two forensic groups, based on combinations of the WAIS subtests and neuropsychological variables, yielded 100% correct classification for both groups. Lower percentages of correct classifications were obtained by only using the 11 WAIS subtests (64% and 79% for the psychopathic and

control group, respectively) or only the neuropsychological variables (88% and 91% respectively). The authors concluded that:

> . . . . the intrahemispheric dysfunction between the psychopaths and depressive groups was similar - dysfunction in the temporal and frontal regions of the brain. However, lateralized hemisphere dysfunction differentiated the groups: dominant or left hemisphere dysfunction was predominant in the psychopathic group, while the depressive and personality disorder-affective patients showed a greater incidence of nondominant hemispheric dysfunction. (p. 144)

Study III: Yeudall and Fromm-Auch (1979)

In this study 115 criminal psychopaths were categorized according to most serious crime into three groups: Homicide, Rape, and Violence Causing Bodily Harm. These authors reported that 91% of the profiles were abnormal while 72% of these reflected greater dominant than non-dominant dysfunction. These three groups were then contrasted with a control group ($n = 25$) in three separate two-way discriminant function analyses based on the neuropsychological and WAIS variables. Correct classifications of 100%, 96.4%, and 97.9% respectively for the three criminal groups were reported.

Yeudall and Fromm-Auch (1979) also report on an analysis of 24 persistent male sex offenders referred from forensic wards at a mental hospital. They noted that 96% of these profiles were considered to be

abnormal with a 70% vs 30% greater involvement of dominant hemisphere functions over non-dominant functions.

The above studies by Yeudall and his co-workers suggest a high incidence of neuropsychological deficit among their court referred patients. In all the above studies, clinical judgements determined in a dichotomous fashion the presence or absence of prefrontal involvement and/or the relative involvement of the dominant versus non-dominant hemisphere.

Other Neuropsychological/Intelligence Studies

Spellacy (1978) contrasted the performance of 40 violent and 40 nonviolent male prisoners from a Canadian medium security penitentiary on a 31 variable neuropsychological battery, including the WAIS. In this study the violent prisoners were significantly poorer than the nonviolent prisoners on 21 of the 31 variables. He concluded that differences in cognitive, language, perceptual, and psychomotor abilities suggested that impaired brain function contributed to the poor control seen in the violent group.

Bryant, Scott, Golden, and Tori (1984) also contrasted prisoners, convicted violent offenders and nonviolent property offenders, on the Luria-Nebraska Neuropsychological Battery (LNNB). They found that the violent offenders scored in the pathological range on measures of reading, writing, and arithmetic skills. They also noted the impaired performance of the violent group on tasks requiring complex integration of information from sensory processing systems and interpreted their results as indicating

compromised executive function, including deficits in sustained attention and concentration in the violent group.

Other investigators have examined intelligence in relation to adult offenders. Wiens, Matarazzo, and Gaver (1959) reported VIQ significantly lower than PIQ in a group of male offenders. Holland, Beckett, and Levi (1981) evaluated a group of male offenders on intelligence, personality, and offense severity measures (participants were required to possess a 6th grade reading level). They found a negative correlation between severity of offense and level of intelligence.

Heilbrun (1979, 1982, 1990) has sought to develop models relating intelligence, empathy, and impulsivity to type and severity of offense. Heilbrun (1982) found a higher risk for violence in low-IQ psychopaths. He believes that both cognitive control of impulses and empathy are reduced in low-IQ psychopaths as a function of ineffective information processing/low IQ. Heilbrun characterizes three types of psychopaths: a) impaired processing psychopath - combining poor cognitive control, insensitivity to others' feelings, and low intelligence. In his view, this type is prone to violent behavior; b) sadistic, effective processing psychopath - in this type, high IQ and well-developed empathy promote arousal and sadistic reinforcement by enhancing awareness of the pain and distress of the victim; and c) defensive, impaired processing psychopathy- characterized by high self-reinforcement, poor impulse control, low empathic ability, and low IQ.

This type is the least dangerous in terms of the prevalence of violence, but the most dangerous in terms of the severity of violence.

The Frontal Lobe Debate

Gorenstein (1982), arguing from the position put forth in "Disinhibitory Psychopathology" (Gorenstein & Newman, 1980), sought to test the hypothesis that psychopaths would show evidence of deficit on standard neuropsychological tests. He thus administered a battery of putative frontal measures, including the Wisconsin Card Sorting Test (WCST), the Stroop Color-Word Interference Test (STROOP), the Sequential Matching Memory Task (SMMT), Anagrams, and spontaneous reversals of the Necker Cube to three groups of male subjects. Two groups, the Psychopaths (n = 20) and Controls (n = 23), were drawn from 49 consecutive admissions (there were 6 exclusions) to two Veterans Administration hospitals. Most subjects were inpatients or participating in the hospital's residential substance abuse program. Twenty-three subjects were receiving treatment for substance abuse, 13 for primary psychological complaints and 7 for both. Once tested, the groups were formed on the basis of a self-report behavioral checklist and the Socialization scale (So) of the California Psychological Inventory. To qualify for assignment to the psychopathic group, subjects had to meet the criteria for a diagnosis of Antisocial Personality Disorder (APD) and score below the normative mean for prison inmates on the So scale. A third group (n = 18) of male college students were also tested and included in the study. Findings were that psychopathic subjects relative to controls were

significantly poorer on perseverative errors on the WCST, SMMT errors and Necker Cube reversals. A discriminant analysis, including four variables; WCST perseverative errors, SMMT errors, Necker Cube reversals, and time taken on Stroop interference were included in a discriminant function analysis yielding an overall hit rate of 85%. On the basis of these findings Gorenstein concluded that these patients suffered from a major cognitive deficit involving the failure to modulate dominant response sets.

Hare (1984) took exception to Gorenstein's (1982) conclusions and was critical, especially of his rationale for forming the psychopathic group. Hare (1984) addressed the issue of frontal lobe deficits in psychopaths by contrasting medium security inmates rated high, medium, and low on psychopathy as determined by the Psychopathy Checklist (PCL) (Hare, 1980). Subjects were administered the WCST, the SMMT, and the Necker Cube. No control group was included. In general, Hare found no significant differences on the measures between prisoners rated high on psychopathy and those rated medium or low.

In a subsequent study, Hart, Forth, and Hare (1990), using the same design, i.e., contrasting medium security inmates high, medium, and low on psychopathy (Psychopathy Checklist-Revised; Hare, 1990), generally failed to find significant differences across neuropsychological tests between subjects at differing levels of psychopathy and they concluded that the overall level of neuropsychological impairment was low.

Hoffman, Hall, and Bartsch (1987) report a failure to replicate Gorenstein's (1982) finding although it is difficult to appreciate this conclusion given that few statistics are provided, e.g., age of subjects or test results. Devonshire, Howard, and Sellars (1988) contrasted two samples of Special Hospital patients in the U.K. and a control group on the Nelson's Modified Wisconsin Card Sorting Test (MWCST) (Nelson, 1976). They failed to find significant differences between patients suffering from a 'Psychiatric Disorder' or patients classified as 'Mentally Ill'. Also, no differences on high and low psychopathy groups on the Psychopathy Checklist were obtained. These authors then divided the patient groups according to Blackburn's criteria of primary and secondary psychopaths. Secondary psychopaths (who are thought to be anxious) were significantly poorer on the MWCST. Controls performed significantly better than both patient groups on all MWCST test measures.

Smith, Arnett, and Neuman (1992) contrasted 69 male minimum security incarcerates, rated high and low on the Psychopathy Checklist, on a battery of tests which they assembled to tap left hemisphere (verbal) and frontal (executive) functioning. They further subdivided groups according to an anxiety measure. Their main finding was that low anxiety psychopaths performed more poorly on Block Design (WAIS-R) and Trail Making B, possibly replicating the anxiety effect noted by Devonshire et al. (1988). Smith et al. (1992) concluded that their study offered no support for the hypothesis that psychopaths are characterized by low verbal or deficient left

hemisphere functioning, but was not inconsistent with an hypothesis of deficiencies in frontal lobe functioning.

Findings reviewed above are conflicting. Probable factors include highly diverse samples, including primarily substance abusers in treatment, mentally ill patients, minimum and medium security inmates, as well as differences in measures employed across studies. In agreement with Miller (1987), the most reasonable conclusion is that the findings are inconclusive. Despite the limitations of the contrasting groups methodology utilized in these studies dogmatic conclusions about the neuropsychology of psychopathy are drawn on the basis of relatively unique samples and one or a few measures putatively associated with prefrontal function. At best, the relationship of prefrontal function and psychopathy remains unclear and inadequately evaluated.

In the next subsection, the work of Gorenstein and others which originates in experiments conducted with lesioned animals, reaches conclusions that implicate the prefrontal cortex and dominant hemisphere in antisocial individuals. The path to this conclusion results from a reinterpretation of previous psychopathy research.

Gorenstein's Interpretation

MacMillan (1992), in tracing the acceptance of the notion of inhibition, noted that methodological and factual complexities aside, the greatest barrier was conceptual, i.e., the ". . . . difficulty of granting that stimulation could produce inhibition" (p. 100). He noted that despite rather clear experimental

evidence, e.g., demonstrations that electrical stimulation of the vagus nerve affected heart beat, and clinical evidence of disinhibition after frontal lobe lesions, e.g., the case of Phineas Gage, the neurological community just could not recognize inhibitory processes for what they were and the debate continued well into the 20th Century. Today, well beyond simple motoric inhibition, the frontal lobe is recognized as a central inhibitory centre extensively involved in the inhibition or suppression of responses or associations that are not relevant to the task at hand. Dempster (1992) noted that inhibitory functions of the prefrontal cortex appear to be the last to develop fully and the first to undergo involution later in life.

Gorenstein and Newman (1980), in their seminal paper on disinhibitory psychopathology noted parallels and similarities in the behavior and disposition of animals and humans who sustained damage to the neural system comprising the prefrontal cortex, the septum and the hippocampus (SHF system). They proposed difficulties in five distinct areas of adaptive functioning: a) avoidance of incidental punishment, b) anticipation of aversive events, c) mediation of temporal intervals, d) responsiveness to rewards, and e) stimulation seeking. Their analysis suggested that a variety of clinical groups, e.g., psychopaths, hysterics, early onset alcoholics, hyperactive children, and non-pathological impulsive personalities, could be seen to suffer from a subtle weakness of the inhibitory functions of this system, particularly the prefrontal cortex (cf. Gorenstein, 1990).

Gorenstein and Neuman (1980), thus proposed that criminal psychopathy may be construed to be the result of deficient prefrontal functioning. Of particular interest here is Gorenstein's (1991) analysis and reinterpretation of autonomic findings with criminal psychopaths as a cognitive deficit implicating prefrontal cortical structures.

Gorenstein (1991) proposed that the criminal behavior of psychopaths (or sociopaths) is characterized by three main features which lead to an inference of mental abnormality or psychopathology. First, he noted the relatively early onset of antisocial behavior, secondly, the enduring pattern and resistance to change, and thirdly, its inadequate motivation. With regard to the latter feature he notes that often the individual will commit gross transgressions against persons or property for exceedingly small returns. He further notes that these individuals appear to lack the usual sense of self-preservation and their behavior appears to be aimed often at nothing greater than the immediate gratification of transient desires. As such, it is often considered impulsive.

Gorenstein began his analysis by presenting hypotheses originally advanced by Lykken (1957) that individuals with psychopathic personality features are defective in their ability to develop conditioned fear responses and because of this defect they are relatively incapable of learning to avoid the aversive consequences of their behavior. He notes with respect to Lykken's classic passive avoidance paradigm that both groups of inmates in his study, those meeting as well as those not meeting Cleckley's criteria,

were indistinguishable from each other, but clearly deficient from non-inmate controls in learning to avoid an incidental punishment contingency. With regard to the conditioned fear hypothesis Lykken (1957) exposed the three groups to a series of conditioning trials involving a buzzer paired with shock. Consistent with the group differences in passive avoidance, the skin conductance of both inmate groups was significantly lower (both in absolute and relative to baseline) than that of non-incarcerated controls. As groups I and II themselves did not differ, it appeared that antisocial individuals were in fact defective in fear-mediated avoidance.

Historically, Lykken's findings fit well within Mowrer's (1947) two-factor theory of learning which could account for the disinhibited behavior of some criminals, since, because of their inability to develop conditioned fear, they would not receive reinforcement for the inhibition of punished behavior because there is little or no fear arousal to provide the basis for that reinforcement through drive reduction. Accordingly, these individuals would likely commit violations that others would learn to avoid. In the above experiment both of Lykken's groups exhibited passive-avoidance deficits yet clearly the application of such a deficit to all criminals seemed unlikely. Schachter and Latané (1964), using the criteria of 'emotional flatness and/or incorrigibility' to define a sociopathic group and 'normal' to define a second group of inmates, found, in using Lykken's passive-avoidance maze that, as predicted, the sociopathic group showed no trend toward avoidance whereas the 'normal' group gradually reduced their proportion of shocked errors over

time. In a second experiment, Schachter and Latané (1964) using two similarly selected new groups, first injected the subjects with adrenaline. They found the effect of adrenaline on the sociopathic prisoners to be dramatic as these prisoners now reduced their proportion of shocked errors considerably over the course of the experiment. Further, no such effect was observed with the 'normal' group, indeed this group failed to show any avoidance learning whatsoever with adrenaline. This study was taken as firm evidence that the avoidance deficit exhibited by some prisoners might be produced by a defect in autonomic arousal.

Further to the above finding, general studies reviewed in Hare (1970) and Hare (1978) investigated the low arousal hypothesis. Hare (1965a), for example, investigated the autonomic responsivity of three groups: a group of prisoners meeting Cleckley's criteria for psychopathy, a non-psychopathic prisoner group, and a group of adult education students. The experimental procedure was simple; subjects observed the numbers 1 - 12 as these appeared in order on a memory drum. Beginning with trial two, as subjects had been informed, an electric shock was delivered coincident with number 8. Findings were that the Cleckley group relative to the other two groups had a lower overall level of skin conduction suggesting a lower level of tonic autonomic arousal. Secondly, the Cleckley psychopathic group exhibited little rise in skin conductance in anticipation of the shocked number 8 and whatever rise occurred came only as the shock was imminent. Hare (1965a) characterized this finding as "a steeper than normal gradient of fear arousal

and response inhibition" (p. 445). Thirdly, despite the above, the skin conductance to the shock itself among the psychopathic group was as great as that of the student group. Gorenstein (1991) reasoned that this indicated that a defect in the autonomic system's ability to respond was not the issue, but rather, that some deficiency in associative processes signalling the shock was involved. Hare (1965b) noted that Cleckley psychopaths were deficient in acquiring a conditioned GSR to a tone paired with shock and, where some conditioning did occur, it was less apt to generalize. A third study by Hare (1965c) indicated that college students who scored high on the Pd scale of the MMPI, like psychopathic groups, produced smaller skin conductance responses in anticipation of shock than low Pd subjects. On the basis of these studies Gorenstein noted that even in a non-pathological group a relationship between fear conditionability and socialization patterns was observed.

Schmauk (1970) applied Lykken's (1957) avoidance task to a group of prisoners (labelled primary sociopaths) which were characterized by high Pd on the MMPI and low on MMPI anxiety indices, a second group of prisoners (labelled neurotic sociopaths) characterized by high Pd and high on anxiety measures, and a third group of 'normal controls'. This study basically replicated Lykken's findings with primary sociopaths being poorest at avoidance learning, normal controls best, and neurotic sociopaths intermediate. However, in a second study using 3 new groups based on the same criteria as above, Schmauk found that in a version of Lykken's mental

maze in which loss of money replaced shock as the punishment for selected errors, both prisoner groups avoided punishment with the same proficiency as the normal controls. From the standpoint of fear arousal, when loss of money was the punishment, the anticipatory GSR's of all three groups were equally high.

A number of motivational arguments could be advanced to account for the responses of the primary and secondary sociopaths in the Schmauk experiment, but as Gorenstein (1991) has pointed out the finding of:

> . . . . normal avoidance learning coupled with normal autonomic anticipation of punishment in even his most extreme antisocial group provides additional, critical evidence that what we are dealing with is not simply a matter of a defective autonomic nervous system, one that simply is too weak to sustain avoidance learning. Instead, it seems that at least some sort of anomaly of central processing or "cognition" must be involved. (p. 107)

Gorenstein (1991), with reference to Rescorla's (1987) re-statement of the classical conditioning paradigm, (that perception of relationships are involved) posits that the deficit in classical aversive conditioning exhibited by antisocial individuals "is indicative of a general inability or disinclination to develop mental representations, not only of aversive contingencies, but of relations among many kinds of events" (p. 108).

In support of his conclusion, Gorenstein (1991) reports on a study by Gullick, Sutker, and Adams (1976) which examined paired associate learning

under conditions of 2, 4, and 8 second intervals between presentation of stimulus pairs. Three groups of prison inmates were included in the study. Group I labelled "sociopathic addicts" had a history of heroin addiction and T scores >70 on Pd and Ma scales of the MMPI, but not on other scales, Group II had similar MMPI elevations, but no history of heroin addiction or abuse, and a third (control) group exhibiting no T score elevation >70 on the MMPI except for some minor exceptions. All groups were screened to eliminate high scorers on the Welsh Anxiety Scale. Results were that no differences were noted when the interstimulus presentation time was 2 or 4 seconds, however, when it was 8 seconds, both "sociopathic" groups were impaired in their acquisition rates; this was particularly true of the addicts.

Gorenstein (1991) interpreted this study as reflecting evidence that antisocial individuals appear to have a general deficit in the mental representations of contingent relations. Another study (Painting, 1961) reviewed by Gorenstein also demonstrated that "primary psychopaths" have difficulties developing/maintaining mental representations of contingent relationships, particularly where these are temporally discontiguous. It seems notable that Gorenstein's deduction that the temporal bridging of contingencies is central to the notion of disinhibition is, in essence, fully congruent with Fuster's (e.g., 1989) proposals characterizing the role of the prefrontal cortex as mediator of cross temporal relationships.

Gorenstein (1991) concluded that antisocial individuals appear to have difficulty forming mental representations irrespective of whether punishment

or avoidance play a role. Still he reasoned that while this weakness should result in a diminished responsiveness to the prospect of future punishment, in itself, it does not provide a convincing rationale for producing criminal behavior. Accordingly, he examined other parallels between the septal hippocampal frontal (SHF) syndrome in animals and the behavior of antisocial individuals looking to factors that might impel toward antisocial behavior.

The SHF syndrome in animals results in a diminished capacity to sustain goal-directed activity and to modulate impulsive responding, although many other behavioral functions remain intact (e.g., McCleary, 1966; Newman, Gorenstein, & Kelsey, 1983). The usual explanation is that these animals (e.g., McCleary, 1966; Gorenstein, 1982; Newman et al., 1983) have an enhanced inclination to emit dominant or prepotent responses (cf. Fuster, 1989). In humans with prefrontal lesions, Luria (1980) has termed this stereotypical responding or the now more common notion of perseverative responding. In Fuster's theory, for example, such perseverative responding could be the result of prefrontal dorsolateral dysfunction, e.g., inability to change sets or orbital dysfunction which could interfere with set maintenance through distractibility.

Gorenstein (1991) thus proposes that antisocial individuals, with a syndrome similar to the SHF syndrome in animals, can be expected to exhibit a diminished responsiveness to the prospect of future punishment because of their impaired capacity to sustain a mental representation of a hypothetical punishing event. However, he argued that the case with

rewards is quite different in that they are usually based on some concrete inherent need. He thus suggests that antisocial individuals will actually be excessively responsive to needs and wants, not because they are undeterred by the prospect of punishment, but because these individuals do not govern themselves by higher, more abstract mental representations. Moreover, he states, once reward-seeking behavior has been initiated, it should be relatively inflexible since the absence of mental representations do not allow the individual to remove himself psychologically from a situation and evaluate performance by measuring it against some internal standard or code.

Newman, Patterson, and Kosson (1987) provided further evidence of this phenomenon. They devised a study in which subjects were to turn over computer-simulated "playing cards" one at a time. Each time a face card was turned up the subject received five cents, but every time a number card was revealed he lost five cents. The subject could choose to stop and collect any accumulated earnings. For the first 10 cards, the reward/punishment ratio was 9/1. The purpose was to instill a dominant response tendency. With every subsequent block of 10 cards however, the rate of punishment increased by 10% such that by the last block of 10 cards, every card would result in a loss of five cents. Subjects in this study were prisoners who rated high and low in psychopathy according to Hare's (1980) 22 item checklist. In this study prisoners rated high in psychopathy exhibited a pronounced perseverative tendency, continuing to play cards

long after the controls had elected to stop. Indeed, 75% of the high psychopathy subjects played the whole deck of 100 cards even though they lost on 19 of the last 20 trials. Cumulative earnings for the group rated high in psychopathy was less than half for the group rated low.

In a second part of this study, with 2 naive groups rated as above, the conditions were modified such that a visual monitor was provided of cumulative rewards and punishments, and also a 5-second interval was imposed between trials. Under these conditions the perseverative responding of the high psychopathy group was completely eliminated. Apparently with external cues provided, high psychopathy prisoners were able to regulate their behavior as well as low psychopathy subjects did through their own internal regulatory mechanisms.

On reflection, this study brings to mind Goldman-Rakic's (1987a) notion of the role of the prefrontal cortex in developing internal representations or Luria and Homskaya's (1964) notion of monitoring behavior through silent speech. Also the notable lack of skin conduction in Damasio et al. (1990) study of patients with lesions of orbital cortex under PASSIVE as opposed to ACTIVE conditions may be pertinent.

Recently, Bechara, Damasio, and Anderson (1994) have elaborated a novel task, with some similarity to the card playing task, which, in their view, simulates real-life decision-making in that it presents subjects with uncertainty of both premises and outcomes, and their linkages to reward and punishment. Their interpretation is that prefrontal patients with orbital

lesions are "myopic" with respect to future consequences and guided by immediate prospects only. In sum, they are impulsive in the extreme and fit both Gorenstein's model of psychopathy, as well as an interpretation of psychopathy as being related to prefrontal orbital dysfunction which is not readily evaluated by traditional neuropsychological tests.

Gorenstein's (1991) analysis suggests that the findings in the study described above may explain why antisocial individuals can perform adequately in avoiding the loss of small rewards in Lykken's mental maze (Schmauk, 1970), but they are nonetheless unable to avoid serious losses in real life. He noted that in real life, avoidance is not the sole incentive and typically an individual is simultaneously concerned with satisfying concrete needs. Thus, if an individual is inclined to be subject to dominant responding due to a diminished capacity for internal mental representation, then the seeking of material gratification will overwhelm other considerations.

A study by Newman and Kosson (1986) has demonstrated the effect of active responding for reward on passive avoidance. In this study a Go/No Go paradigm was utilized. In the first condition subjects were rewarded with ten cents whenever a button was pushed in response to a 'go stimulus' and lost ten cents whenever the button was pushed in response to a 'no go stimulus'. In this condition prisoners rated high in psychopathy (Hare, 1980) made significantly more errors of commission than prisoners rated low. In a second condition however, where only punishment was used, i.e., a subject

lost ten cents for either responding to the 'no go stimulus' or for not responding to the 'no go stimulus', there was no difference between prisoners rated high or low on psychopathy. In sum, high psychopathy prisoners achieved normal passive avoidance where no reward was made contingent on a response, however, when a contingency was introduced, the capacity for passive avoidance was disrupted.

Gorenstein (1991) suggested that antisocial individuals are not equally responsive to loss of rewards even in cases in which the two contingencies are equated for value because reward-seeking virtually always requires less verbal mediation. As he put it:

> This is because, in the case of reward-seeking activity, behavior can largely be sustained by a simple state of need. But in the avoidance of loss, the organism must respond on the basis of its ability to anticipate a potential state of need, . . . I would suggest this model is readily applied to the real-life failures of antisocial individuals. That is, the losses they sustain represent the dissipation of gains that they have failed to protect trying to satisfy some current state of need or desire. (p. 120)

Gorenstein noted that his reasoning concerning the relative responsivity to the presentation versus the loss of reward has considerable theoretical and empirical support originating in Gray's (1971, 1972, 1982, 1987a) theory of brain function and this is considered next.

Gray's Theory/Application To Disinhibition

Gray (1972) has proposed that differential responsivity to different forms of reinforcement can be dissociated neurologically. Specifically Gray (1972, 1982, 1987a) has proposed that two separate (though reciprocally interconnected) brain systems, the Behavioral Activation System (BAS) and the Behavioral Inhibition System (BIS), mediate different types of reinforcement. The BAS mediates contingencies involving the presentation of reward and the termination or omission of aversive stimuli. The BIS on the other hand mediates contingencies involving the presentation of aversive events and the termination or omission of rewards. According to Gray (1982), the septal hippocampal frontal (SHF) system can be conceptualized as an hypothetical behavioral inhibition system. It is activated by punishment cues and novel stimuli. Once it is activated, its functions include: a) interrupting ongoing behavior that may lead to aversive consequences; b) augmenting arousal to intensify behavior immediately following an interruption; and c) recruiting attention for analysis of the environment, particularly novel aspects. According to Gray (1972, 1982, 1987a, 1987b) the SHF system underpins anxiety. It governs orienting and its activation results in increased electrodermal activity and motoric quiescence that facilitates passive avoidance. Fowles (1980, 1988) has reviewed a large number of studies of heart rate and electrodermal responsivity and concluded that elevated heart rate is the primary psychophysiological indicator of arousal of the BAS whereas electrodermal

activity reflects arousal of the BIS. Thus, Gray's theory appears to anticipate the dissociation of approach and avoidance capabilities in antisocial individuals.

Gorenstein (1991) has noted however, that while this model appears to accommodate the greater responsiveness to the gaining of reward than to its loss (BAS arousal being greater than BIS arousal), it does not appear to account for the findings that antisocial individuals seem to be capable of avoiding punishment involving the loss of reward, at least when there is no conflicting approach contingency, yet incapable of avoiding punishment involving electric shock under the very same circumstances (e.g., Schmauk, 1970). As Gorenstein notes, in this case differentiation in terms of BIS and BAS activity is not possible since both the presentation of shock and the loss of reward are subserved by the same system and therefore theoretically equivalent. Gorenstein proposes to resolve this dilemma by appealing to his cognitive perspective which maintains that in the experimental paradigms utilized, it is easier to sustain a cognitive representation of the loss of reward because there are more external cues associated with it than, for instance, with shock. In general, Gorenstein proposes that the reduced electrodermal response of antisocial individuals in anticipation of shock (e.g., Hare, 1978) is an indication of a general tendency not to maintain the cognitive set necessary to suppress dominant responding. This perspective seems already to have considerable support in other theories and empirical findings related to dominant anterior dysfunction or verbal mediation deficits and these will

be reviewed later. Patterson and Newman (1993) have attempted to explicate disinhibitory processes in psychological terms. As such, a review of their presentation may serve to bridge Gorenstein's cognitive model of SHF function to broader dominant hemisphere or cognitive mediation explanations of antisocial behavior.

Psychological Mechanisms Of Disinhibition

Patterson and Newman (1993) elaborated a four stage model of response modulation associated with the passive avoidance paradigm, but generalizable to other situations, e.g., the Wisconsin Card Sorting Test or the Delayed Response Paradigm.

Their theory focuses on response modulation which they define as a complex process involving the temporary suspension of a dominant response set and a brief concurrent shift of attention from the organization and implementation of goal-directed responding to its evaluation. Adaptive responding requires making some adjustment to the response set once the corrective information is accommodated which includes response inhibition, selection of an alternative response strategy, or no adjustment where the response is evaluated to be appropriate.

Stage 1

This stage relates to the establishment of a dominant set for reward. As long as an appetitive motivational state exists, goal-directed behaviors are most likely to be emitted. Cognitively, an effortful allocation of attention to goal-relevant environmental stimuli and an expectation of reward is typical.

At this stage, over-focusing may result in discounting or neglecting cues for punishment or frustration (Newman et al., 1983; Newman, Patterson, & Kosson, 1987; Siegel, 1978).

Physiologically, this stage is marked by an increase in heart rate (Fowles, 1980, 1988) accompanying activation of the BAS. The control of motor activation and output has been attributed mainly to the mesolimbic dopamine system (Iversen, 1977) and both approach and active avoidance related to reward learning and learned relief (Beninger, 1989) also implicate this dopamine system. Individual differences in forming approach sets and the intensity of their maintenance are the main variables of interest here.

Stage 2

In stage 1 a dominant set is established. Stage 2 relates to an increment in arousal that follows when an obstacle or aversive event disrupts the reward associated with the dominant set. The first consequence of this disruption is an automatic call to process it (Öhman, 1979) and, secondly, an increase in arousal reflecting an effortful emotional reaction in view of the mismatch between reality and expectation. Patterson and Newman (1993) believe that the arousal at this stage is largely a function of individual reactivity to aversive events and view it as independent from the strength of one's bias to adopt motivational sets, i.e., stage 1.

Stage 3

Following the unexpected aversive event and the ensuing disruption of the dominant behavior, a coping response fuelled by the arousal increment of

stage 2, an effortful adaptive switch to a passive gathering set would be typical of non-disinhibited individuals. In contrast, disinhibited individuals respond such that the dominant response is facilitated rather than suppressed, particularly where reward cues remain in the immediate stimulus context. Passive avoidance is improbable in such situations. The non-disinhibited approach here is to inhibit ongoing behavior and initiate information processing and problem-solving so as to adjust their actions and expectations in keeping with the set derived through the accommodation of the unanticipated feedback. Gray (1987a) has noted that serotonergic regulation is critical to this process and Thiebot, Hamon, and Soubrié (1984) have isolated specific 5HT pathways that inhibit dopamine pathways underlying approach behavior and are necessary for behavioral inhibition in the presence of aversive cues. Patterson and Newman (1993) propose that at stage 3, non-disinhibited individuals answer the call to process feedback, or to use Teuber's (1964) terminology, they are not impervious to error information. There is a shift from automatic to controlled processing and such processing "involves conscious rehearsal, (re) evaluation of the situation . . . and language mediation" (p. 721). In contrast, disinhibited individuals "do not pause, process and then go on" (p. 721). Patterson and Newman (1993) note that according to their analysis there are two routes to impulsivity here; first, through perseveration of the dominant set, and secondly, through an associative deficit at stage 4.

Stage 4

Patterson and Newman (1993) note that the failure to reflect, to pause and process the cues for punishment or frustration interferes with retrospective reflection. This later term they define as "the process whereby causal associations are formed between behaviors and their consequences, as well as the stimuli that predicted those consequences" (p. 722). They suggest that this process involves passive information gathering about relations among stimuli to form a temporal cognitive map and their retrospective processing of that information before renewed action. It would seem to follow that prospective reflection could be similarly degraded and lead to poor judgement (cf. Fuster, 1993 re: his concept of the prefrontal representation of action plans).

The conclusion reached by Patterson and Newman (1993) regarding the preference for rewards over avoidance of losses is strikingly similar to Gorenstein's (1991) analysis described above as they state "Because disinhibited individuals form relatively fewer inhibitory associations involving cues that predict aversive events, their responding is likely to be swayed more by expectations of reward than by the associative products of retrospective reflection . . . ." (p. 722).

In accord with Gorenstein (1991), Patterson and Newman (1993) examined recent evidence (Newman, Patterson, Howland, & Nichols, 1990) that low-anxious psychopaths' uninhibited responding for reward, despite punishment, is linked to low reflectivity, which in turn likely underlies their

poor passive avoidance. In one of the experiments described by Patterson and Newman (1993), low anxious psychopaths defined using Hare's (1980) criteria of psychopathy, and anxiety ratings as measured by the Welsch Anxiety Scale, it was found that when only noncontingent reward incentives were used that psychopaths showed no evidence of greater activation (response speed) in the presence of rewards than non-psychopaths. This finding, unlike the speeded-up performance of extroverts (Nichols & Newman, 1986) suggests that while both groups can be considered disinhibited, the analysis in terms of the Patterson and Newman (1993) model is different. Whereas, in the case of extroverts, the interpretation of increased BAS activity may be explanatory, it does not hold for psychopaths, as they showed no speeded up responding. Newman et al. (1990) and Patterson and Newman (1993) conclude that because of the greater influence of the dominant set, psychopaths are less likely to process the adverse implications of their response. Thus, they conclude as did Gorenstein, that, in the final analysis, the main deficit does not appear to be autonomic, but rather cognitive.

As Patterson and Newman (1993) state:

> . . . psychopaths appear to process environmental stimuli, ones that are initially less salient, incompletely or not at all when the dominant response involved instrumental action. Also when aversive contingencies that were initially less salient become manifest, psychopaths appear to have difficulty switching their attention to

> them . . . with a learning history deficient in processing and forming inhibitory associations, psychopaths are less likely to develop a repertoire of automatic attentional responses to stimuli predictive of aversive events. (p. 729)

This section of the literature review has focused primarily on the neuropsychology of adult offenders in terms of theories and studies applying traditional clinical neuropsychological tests to adult offenders and the re-interpretation of the psychopathy literature, especially by Gorenstein and the exposition of a psychological model of impulsivity. The next task is to review the neuropsychological aspects of children with behavior disorders and their developmental outcome in relation to antisocial behavior.

## SECTION V DISRUPTIVE BEHAVIOR DISORDERS NEUROPSYCHOLOGY/DEVELOPMENTAL OUTCOME

### Introduction

The aims of this section are: a) to examine neuropsychological theories and findings of Attention Deficit Hyperactivity Disorder (ADHD) and Conduct Disorder (CD); b) review studies of developmental outcome and studies of the neuropsychological characteristics of young offenders/delinquents; and c) discuss linkages between behavior disorders, neuropsychological functioning and antisocial outcome. However, before beginning this task, comments on the prevalence and co-morbidity of ADHD and CD, and associated taxonomic problems are warranted.

Co-Morbidity Attention Deficit Hyperactivity Disorder and Conduct Disorder/ Taxonomic Issues

Both ADHD and CD are common disorders, the consensus of opinion seems to be that approximately 3 to 5% of the childhood population has ADHD (American Psychiatric Association, 1987), but estimates have varied between 1 and 20% (Ross & Ross, 1982; Sandoval, Lambert, & Sassone, 1980). Szatmari, Offord and Boyle (1989) estimate the prevalence of this disorder at between 3 and 5% of all school-aged children. Szatmari, Offord, and Boyle (1989) reported the results of a survey of the Province of Ontario and found the prevalence of ADHD to be 9% in boys and 3.3% in girls. For boys, there were age variations with a prevalence estimated at approximately 10% in the 4-11 age group and 7.3% in the 12 to 16 age group. Barkley (1990) estimates that 5 to 6% of children between 4 and 16 years of age are likely to be diagnosed as ADHD.

Estimates of the prevalence of Conduct Disorder in the general population range from approximately 3 to 7% (Shapiro & Hynd, 1993). Szatmari, Offord, and Boyle (1989), in the Ontario study, observed a rate of 6.5% for boys between 4 and 11 years of age (1.8% for girls) and 10.4% for boys 12 to 16 years of age (4.1% for girls) for a combined rate of 8.1% for boys aged 4 to 16 years (2.7% for girls). The co-occurrence of ADHD and CD has created interpretive difficulties. Despite specific diagnostic criteria for ADHD and CD, there has been much controversy regarding the syndromal independence of ADHD and CD (e.g., August & Stewart, 1982;

Biederman, Newcorn & Sprich, 1991; Prior & Sanson, 1986; Shapiro & Garfinkel, 1986; Stewart, Cumming, Singer & DeBlois, 1979; Trites & LaPrade, 1983; Werry, Reeves & Elkind, 1987). At the centre of the argument is the finding that in nearly all the large factor analytic studies of behavior ratings a hyperactivity factor and a conduct problem or aggression factor have emerged and once the orthogonality constraint has been removed, interfactor correlations have been high. Hinshaw (1987) reviewed 47 factor analytic studies and found interfactor correlations as high as .90 with a median correlation in the region of .55 to .60. Szatmari, Boyle & Offord (1989) reviewed 20 studies comparing children diagnosed with ADHD or CD and concluded that where differences occurred, they were small and of uncertain clinical relevance. In their own study they found that ADHD children were 14 times more likely than controls to warrant a co-diagnosis of CD. In large multisource, multiage studies, (Ferguson & Horwood, 1993; Ferguson, Horwood & Lloyd, 1991) correlations between trait measures of conduct disorder/oppositional behaviors and indicators of attention deficits ranging between .80 and .85 were reported. Barkley, DuPaul and McMurray (1990) estimate that 20 to 30 percent of ADHD children will warrant a diagnosis of CD and by adolescence this increases to 40 to 60 percent. Taylor (1988), in his review of the ADHD diagnosis, concluded that basic nosological questions are not resolved, and Quay, Routh and Shapiro (1987) suggest that the ADHD diagnosis does not qualify as a syndrome as distinct from CD. The diagnostic practices between the United Kingdom and North

America also add to the uncertainty since most children diagnosed as ADHD in North America would receive a diagnosis of CD in the United Kingdom (Taylor et al., 1987).

Some authors have suggested that the co-occurrence of ADHD/CD represent a distinct hybrid disorder typically of greater severity and with a poorer prognosis. Walker, Lahey, Hynd and Frame, (1987) noted the marked severity of their jointly diagnosed subgroup. As well, Szatmari, Boyle, and Offord (1989) concluded that a co-morbid ADHD and CD showed the impairing features of both conditions, the developmental delays of ADHD youngsters and the psychosocial disadvantages of their CD group, but in a different configuration than would be expected by a simple addition of the two disorders. Although studies are inconsistent (Hinshaw, 1994b), children having both ADHD and CD appear to display greater under-achievement (Hinshaw, 1992), greater rates of peer rejection (Milich & Landau, 1982) and qualitatively different and usually poorer response to medication (Barkley, McMurray, Edelbrock & Robbins, 1989; Klorman et al., 1988).

In addition to the high co-morbidity of ADHD with CD, ADHD has been shown to be highly correlated with school under-achievement (Barkley, 1990) and specific co-morbidity rates of 10% to 25% with formal learning disabilities have been reported (Hinshaw, 1992; Semrud-Clikeman et al., 1992). Furthermore, Biederman et al. (1991) have estimated the overlap of ADHD with anxiety disorders at 25%. The literature is inconsistent in reported rates of the co-occurrence of ADHD with mood disorders. Munir,

Biederman and Knee (1987) reported that 32% of children in their sample with ADHD had a co-occurring major affective disorder while Biederman et al. (1991), in their review of this issue, noted that reported rates of co-morbidity with affected disorders ranged from near chance levels to over 70%. Studies of CD co-morbidity report overlap with academic under-achievement, anxiety and depression. These studies are far less definitive, however, than those for ADHD (Hinshaw, Lahey & Hart, 1993).

In sum, high rates of co-morbidity between ADHD and CD, as well as significant co-morbidities of these disorders with academic under-achievement, learning disabilities and affective disorders have been reported.

The lack of nosological precision in distinguishing between ADHD and CD, as well as the uncertain influence of other co-occurring disorders, implies a need for caution in interpreting studies of neuropsychological function and outcome for ADHD and CD samples. In the absence of a more differentiated diagnostic classification scheme which would identify subgroups at phenomenological levels, relationships with specific neuropsychological deficits and particular outcomes, e.g., vulnerability for substance abuse or antisocial behavior with and without aggressive features, can be expected to be obfuscated. Indeed, given the heterogeneity and wide array of manifestations of ADHD/CD, any single or over-arching hypothesis of the underlying mechanism is likely to be inaccurate for a substantial proportion of children with disruptive behavior disorders.

However, fruitful hypotheses as to important factors in subgroupings may be derived from existing studies.

Neuropsychology of ADHD

In his historical review of ADHD, Barkley (1990) noted that between 1900 and 1960, children having significant problems with attention, impulse control and over-activity were considered to have minimal brain damage (MBD) given their phenotypic resemblance to children who had sustained brain trauma (cf. Mateer & Williams, 1991). In the 1960's the focus shifted to hyperactivity, and while the disorder was no longer attributed to brain damage, a focus on brain mechanisms prevailed. Prognosis was felt to be relatively benign as it was thought the disorder was outgrown by puberty. In the 1970's the focus shifted to deficits in attention and impulse control, and Barkley credits Douglas' (1972) address to the Canadian Psychological Association as the impetus for this shift. Douglas further elaborated and researched this theory of hyperactivity (Douglas, 1980a, 1980b, 1983; Douglas & Peters, 1979) and it formed the focus of much research for the following 15 years (Barkley, 1990). Douglas' position was that four major deficits could account for symptoms of ADHD: a) deficits in the investment, organization and maintenance of attention and effort; b) inability to inhibit impulsive responding; c) inability to modulate arousal levels to meet situational demands; and d) an unusually strong inclination to seek immediate reinforcement.

Douglas' perspective was largely reflected in the reconceptualization of the Hyperkinetic Reaction of Childhood (DSM-II, American Psychiatric Association, 1968) to Attention Deficit Disorder (DSM-III, American Psychiatric Association, 1980) which provided specific behavioral criteria relating to attention, impulsivity and hyperactivity. The 1980's witnessed considerable research on these definitional criteria with the result that a single list of symptoms and a single cut-off score replace the three separate lists (Inattention, Impulsivity and Hyperactivity). The item list was now more empirically derived from field studies and "Hyperactivity" was re-introduced in the diagnostic title, i.e, ADHD. DSM-IV (American Psychiatric Association, 1994) reinstituted the categories of Inattention, Hyperactivity and Impulsivity, but provides for three distinct types: Inattentive, Hyperactive/Impulsive and Combined. The net effect of this latter change is to elevate inattention without hyperactivity and impulsivity to a distinct disorder.

Barkley (1990) proposed that in 1980 that a consensus definition of ADHD would have focused on developmentally inappropriate degrees of inattention, overactivity and impulsivity. Over the past 20 years research on ADHD has shifted from a focus on hyperactivity per se to research on attentional processes and then to a focus on more complex cognitive processes that appear to underlie the surface symptoms of ADHD. Recently, theoretical approaches have broadened, in particular, more global self-regulatory deficits have been emphasized and the attentional deficits are

being addressed in a multidimensional framework. In these revised perspectives the symptoms of ADHD have been referenced, especially to theories of prefrontal cortical functions. Representative perspectives are discussed.

Parallels With Frontal Lobe Dysfunction

Mattes (1980) reviewed parallels between symptoms of the hyperkinetic syndrome and clinical manifestations of frontal lobe dysfunction. He concluded that frontal lobe dysfunction might be more explanatory and parsimonious than hyperkinesis in that it "might suggest a more primary deficit that can manifest itself in symptoms, such as impulsivity and poor response to reinforcement, in addition to hyperactivity and poor attention span" (p. 366).

Evans, Gualtieri and Hicks (1986) and Gualtieri and Hicks (1985) also noted parallels between the symptoms of hyperactivity and the effects of frontal lesions in animals and humans. Focusing on excessive intrasubject variability of autonomic, electrocortical and behavioral response, they characterized ADHD as reflecting a "*trait* of varying *states*" (Evans et al., 1986, p. 275). They postulated a neural basis in frontal-striatal dopamine systems and felt the net impact of the disorder to result in limbic-frontal dissociation, emphasizing that lack of persistence on an action plan could be the result of the failure of the frontal lobe to influence the limbic system to generate motivation, or alternatively, failure of the limbic system to communicate motivational influences to frontal areas. This disconnection,

they proposed, could account for experimental findings in passive-avoidance, conditioned skin resistance and conditioned emotional reaction. They reasoned that no conditioned response is formed because of the disconnection of cognition and affect.

Whereas Douglas (1972) was among the first to focus on attentional and impulsive aspects of ADHD, she has subsequently emphasized (Douglas, 1983, 1988) defective self-regulatory or "executive" processes and demonstrated (Douglas & Benezra, 1990) that supraspan memory tasks which require organized, deliberate rehearsal strategies, sustained strategic effort, and careful consideration of alternatives, were especially impaired in ADHD children. Shue and Douglas (1992) have proposed that frontal lobe dysfunction may be shown to be a viable neuropsychological model for understanding the self-regulatory deficits exhibited in attention inhibition, arousal and abnormal responses to reinforcement, and that examining functional differences in frontal lobe subsystems in ADHD children may be a fruitful endeavor.

Voeller (1991a, b) postulated that the syndrome of ADHD likely reflects dysfunction in several different neural systems. He proposed that a simple inattention subgroup, characterized by sensory-attentional deficits (stimulus-detection deficits, hypoarousal and impaired vigilance) may represent a "posterior group" with dysfunction primarily associated with right parietal dysfunction. This would be consistent with recent neural differentiation of attentional processes (Cohen, Sparling-Cohen & O'Donnell, 1990; Colby,

1991; Posner, 1988) and the typology obtained by Hart et al. (1993) which appears to have influenced categorization in DSM-IV, but somewhat inconsistent with the empirical findings of Swanson et al. (1991) who found that posterior-based covert shifts of attention were found to be normal in ADHD children whereas anterior overt shifts were not.

Voeller characterizes a second subgroup as "anterior" with dysfunction in frontal nigro-striatal circuits. He notes that these children would be expected to manifest motor-intentional deficits and response inhibition deficits. This identification is consistent with Heilman, Voeller and Nadeau's (1991) assignment of a gating function to the striatum such that volition is not correctly transcoded into action. In their view, disorder at this level would lead both to a form of inattention where stimuli that should lead to action do not and difficulties with response inhibition where stimuli that should not lead to action do elicit a response. Voeller characterizes his third group as "ventral" and suggests dysfunction of the limbic-nucleus accumbens systems, including the mesolimbic dopamine system. He would expect this group to manifest high levels of restlessness and overactivity, as well as cognitive deficits.

Another prefrontal perspective can be subsumed within the notion of disinhibitory psychopathology. Within this approach, poor inhibition of motoric responses rather than attentional deficits per se are seen as the critical feature of ADHD. This position has been articulated by Gorenstein

(1990) and Quay (1988a,b), and represents an extension of Gray's (1982) theory regarding inhibitory and excitatory behavior response systems.

Barkley (1994) has presented an integrated theory of ADHD in which the diverse manifestations and experimental findings of the disorder are reduced to a primary deficit in delayed responding or impaired response inhibition mediated by the orbital frontal cortex.

> I now believe that the manifold deficits witnessed in ADHD can be reduced to a single deficiency in the capacity to delay responding to a signal, event or stimulus. . . . . The capacity for delayed responding is apparently mediated by the orbital frontal cortex . . . . (p. 14)

Barkley (1994) maintains that a deficit in delayed responding has critical impacts on cognitive development. He analyses deficits in ADHD with reference to Bronowski's (1977) model of cognitive development. According to this model, the capacity to delay responding permits four cognitive processes to develop: a) it permits individuals to separate the message from its emotional significance. This allows for a separation of affect; b) it makes possible the prolongation of information and the evaluation of its significance relative to memories for the past and action proposals for the future. The function, according to Barkley, is similar to the notion of working memory as employed by neuropsychologists (cf. Pennington, in press). Conceptually, it seems particularly close to Fuster's (1989, p. 163) symmetrical functions of short-term memory and anticipatory set; c) internalization of language

becomes possible. This function appears to relate to the development of internal representations of external reality. It permits reflection, deliberation and the generation of internal rules governing behavior; and d) a process referred to as reconstitution, this function relates to the manipulation, analysis, and synthesis of internal representations. Hypothetical models can be elaborated providing for problem-solving, imagination, and creative capacities.

Barkley (1994) has thus proposed that Bronowski's (1977) cognitive model, which includes four incremental stages of development, are all dependent on the ability to delay responding and critically contingent on the prefrontal cortex. The consequence of a deficit in delayed responding is behavioral disinhibition, especially due to a lack of cognitive development. It is this relative underdevelopment of cognitive self-control structures which place ADHD children at risk for developing conduct disorders.

> Disinhibited individuals, who as a consequence, are more stimulus bound to the immediate context, are more affectively reactive to situations, are less adequately rule-governed and task-persistent, have a more delimited sense of past and future, are less able to engage in analysis and synthesis (reconstitution), and perform more poorly under conditions of delayed or partial consequences, should have a much greater probability for later oppositional defiant and conduct disorder. (p. 43)

Regional Cortical Blood Flow (rCBF) and Positron Emission Tomography (PET) studies of brain metabolism provide some support for an anterior localization in ADHD. Lou, Henrickson, and Bruhn (1984) found reduced rCBF to the frontal lobes in children with ADHD. Blood flow increased in those children who then received methylphenidate. In an expanded sample, Lou, Hendricksen, Bruhn, Borner and Nielsen (1989) reported maximal differences between ADHD children and controls in the basal ganglia. Further, in a PET study, Zametkin et al. (1990) reported that parents of ADHD children who themselves had residual ADHD symptomatology, but had not been treated with stimulant medication had an overall reduction in cerebral glucose utilization, but this was especially pronounced in frontal areas. In a further PET study with adolescents having ADHD, Zametkin et al. (1993) failed to replicate the finding of an overall decrease in glucose utilization found with adults, but specific differences between adolescent children and controls were found. Left anterior metabolic rate was found to correlate with measures of symptom severity ($p < .001$ to .009, $r = -56$ to -.67). These authors considered the small sample size, the inclusion of some ADHD children with co-existing learning disability, and differences in technical scanning parameters may possibly have contributed to the failure to replicate the overall reduced metabolism finding of their 1990 adult study.

In a further study, Ernst et al. (1994) also failed to find overall decreases in metabolism. In the Ernst et al. study, when data from the Zametkin et al. (1993) study were pooled, significant decreases in metabolism were noted in

the left anterior frontal region, the right posterior temporal regions, and the left posterior frontal region. They highlighted the left anterior frontal region as being of particular interest as it corresponded to the most notable area of discrimination in the adult study by Zametkin et al. (1990). Ernst et al. (1994) also emphasized that female adolescents in their study showed the greatest differences in metabolic activity.

No evidence of structural differences in Computerized Tomography (CT) scan studies of ADHD children has been found (Harcherik et al. (1985). However, Hynd, Semrud-Clikeman, Novey and Eliopulas (1990) found that their sample of ADHD children did not show the usual R>L frontal asymmetry using Magnetic Resonance Imaging (MRI) scans. However, Semrud-Clikeman et al. (1994) reported MRI findings indicating the splenial area of the corpus callosum to be smaller in children with ADHD than in a sample of normally developing children. They suggest that these differences may relate to sustained attention deficits which in turn ultimately impact capacities for self-regulation.

In sum, recent perspectives on ADHD increasingly seek to account for the diverse symptoms and manifestations of the syndrome in terms of a more parsimonious or primary underlying deficit. These characterizations include notions of behavioral disinhibition, executive dysfunction, impaired working memory, impaired rule-governed behavior, impaired delayed responding or response inhibition, and a failure to develop internal language. All of these deficits are readily related to dysfunction of the prefrontal cortex

and the syndrome is thus, increasingly seen, to relate to neuropsychological dysfunction. Recently, metabolic studies have provided some support for primary localization of the disorder to the prefrontal cortex. Next, support for this view from neuropsychological studies is reviewed.

Neuropsychological Findings

A number of studies reviewed below have examined the performance of ADHD children on purported measures of prefrontal function. It should first be noted, however, that the ecological validity of laboratory and analogue assessment instruments have generally been quite low, correlating only moderately with rating and clinical diagnostic criteria (Barkley, 1991). The reasons are not entirely clear, but in part, this may be due to the apparent ability of ADHD children to respond "normally" in situations of high structure (e.g., testing situations) or where motivation is high (e.g., individual attention, immediate feedback). With regard to neuropsychological assessments per se, additional issues arise. For example, many of the measures have a very low ceiling, further, they may not be sensitive to the developmental aspects of prefrontal function. Secondly, behavioral disinhibition, which is the most prominent feature of ADHD addressed in these studies may relate primarily to orbital/medial dysfunction, which is not seen to be as critical for cognitive functioning as dysfunction associated with dorsolateral prefrontal structures, and thirdly, in several studies ADHD and control groups are first equated on intelligence variables. Clearly, frontal lobe hypotheses argue for the partial independence of intelligence variables

and measures of executive function, yet early executive dysfunction surely would impact intelligence variables, thus, in these studies it seems possible that alignment on intelligence variables would underestimate the relative executive dysfunction of the ADHD groups. Random sampling may yield greater differences. Fourthly, the putative tests used have been criticized for their lack of ecological validity and may not even identify relatively severe executive dysfunction, even where it is demonstrable with more appropriate techniques (cf. Shallice & Burgess, 1991). Finally, given the heterogeneity of ADHD, its diverse manifestations, its comorbidity with other disorders and the likely heterogeneity of neural systems implicated, large important relationships in subgroups would likely be muted by a contrasting groups design. Yet despite all these factors biasing against significant findings, the majority of studies have found differences between ADHD groups and contrast groups. These studies are reviewed below.

Several studies (Barkley, Grodzinsky & DuPaul, 1992; Boucagnani & Jones, 1989; Chelune & Baer, 1986; Fischer, Barkley, Edelbrock & Smallish, 1990; Grodzinsky & Diamond, 1992; Loge, Staton & Beatty, 1990; Pennington, Groisser & Welsh, 1993) have examined differences between ADHD groups and normal controls on the WCST. Of these 7 studies, 5 studies found significant deficits in ADHD children relative to controls on WCST measures traditionally thought to be most sensitive to frontal lobe dysfunction. These being perseverative responses, perseverative errors, and number of categories achieved. In contrast, Barkley et al. (1992) did not

find significant differences between groups, but this was a small study, including only 12 subjects per group, and there was prior equating on intelligence variables. Loge et al. (1990) also failed to demonstrate significant differences on the WCST, but differences were found on other putative measures of frontal lobe function, e.g., the California Verbal Learning Test (Delis, Kramer, Kaplan, Ober & Fridlund, 1983), the Brown Peterson Short-Term Memory Test (Ryan & Butters, 1980) and measures of vigilance. Fisher et al. (1990) compared ADHD and control adolescents on the WCST and found no significant differences. However, the possibility that ceiling effects may account for this cannot be ruled out. Consider that in the Boucagnani and Jones (1989) study, 7 year old ADHD children had 51 perseverative errors versus 26 for controls, but for children aged 10+, nearly identical error scores were obtained. Consider also the findings of Pennington et al. (1993) who demonstrated that children with ADHD differed from controls on measures of executive function, including the WCST, but children with ADHD and Reading Disability (RD) did not, whereas children with ADHD only, like controls, were distinct from the ADHD+RD group and the RD group on phonological processing measures. These authors argued that the ADHD symptomatology of the ADHD+RD group was likely attributable to a different etiology and a different dysfunctional neural substrate.

Five of the above studies utilized the Stroop Test (Stroop, 1935) and in each of these studies, significant differences between ADHD groups and

normal controls were reported. Results for other putative measures of frontal lobe function, e.g., the Trail Making Test, measures of verbal fluency, and the Continuous Performance Test, have been mixed (cf. Barkley et al., 1992). However, these authors point to the many methodological problems, e.g., failure to control for comorbidity and inherent limitations of the measures selected, such as their sensitivity to age, as obfuscating interpretation.

Additionally, it seems reasonable to expect that even with a tightly defined group of ADHD subjects, subgroups could be further defined in terms of subtypes of executive deficits (Kemp & Kirk, 1993), but this will require further taxonomic work. On balance, frontal lobe theories of ADHD are compelling and the weight of experimental evidence supports such conceptualizations. Further clarification of the relationships between theory and measurements of prefrontal functioning with diagnostic parameters is necessary, however. Moreover, much more subtle paradigms assessing prefrontal deficits may be required. For instance, Ross, Hommer, Breiger, Varley, and Radant (1994) were able to identify subtle, but highly significant differences in eye movement inhibitory behavior in ADHD children in a sophisticated occulomotor delayed response task.

Neuropsychology of Conduct Disorder

The most salient neuropsychological theory of CD is Quay's (1986, 1988a, 1993) theory of disinhibition. Note that he also postulated this theory for ADHD (Quay, 1988b). The theory represents a downward

extension to children with CD of Gray's theory that in adult antisocial disorders there is an imbalance of the Behavioral Activation System (BAS) and the Behavioral Inhibition System (BIS). Consider, however, Gorenstein's (1991) critique of this approach and his cognitive mediation hypothesis. Briefly, Quays' (1988b) theory proposes that in children with CD, there is an imbalance in favor of the BAS over the BIS. The BIS is activated by signals of impending punishment, signals recently associated with the failure of a formerly expected reward to appear (frustrative non-reward), or novel stimuli. The BIS produces outputs that inhibit ongoing behavior, but also increases arousal, attention and information processing of the stimuli. The BAS mediates contingencies involving the presentation of reward and the termination or omission of aversive stimuli.

Quay proposes that CD involves a persistently overactive reward system that predominates over the BIS. The theory has received some empirical support. Shapiro, Quay, Hogan and Schwartz (1988) tested the oversensitivity to reward hypotheses. The reward task was Newman et al.'s (1987) computerized card game that is rigged so that there is a constantly decreasing probability that a play will be rewarded. On this measure, children who warranted a diagnosis of CD played significantly more cards, i.e., were more perseverative or disinhibited than controls. All subjects were attending a school for seriously emotionally disturbed students in South Florida and had a mean IQ of 87. CD children were also significantly less effective on a delay task which rewarded inhibiting responses for a short

time. This study was partially replicated by Daugherty and Quay (1991). In this study children in grades 3 to 6 were grouped on the basis of screening scales and grouped into four categories labelled CD, Attention Deficit Disorder (ADD), ADHD/CD or anxious withdrawn (AW). Results were that groups CD and ADHD/CD were significantly more perseverative on the card task. No group differences were obtained on a delay task. Quay's theorization is intended to apply to what is generally referred to in the literature as Unsocialized Aggressive Conduct Disorder (UACD) and not all conduct disorders.

While there have been many biochemical and psychophysiological investigations of children with CD, there have been almost no neuropsychological studies (Shapiro & Hynd, 1993; Moffitt, 1993a). A recent study, however, of 20 boys aged 10 to 13½ years in residential treatment (Warr-Leeper, Wright, & Mack, 1994) disclosed a high degree of language impairments. Coincidentally, 16 of these behaviorally disordered children had co-diagnoses of ADHD. In contrast, a significant number of studies with young offenders/juvenile delinquents have been reported. These are reviewed below.

Neuropsychology of Delinquency

Neuropsychological studies of young offenders/delinquents have focused primarily on verbal deficits and deficits in executive function/behavioral disinhibition (Moffit & Henry, 1991, 1993; Moffitt & Lynam, 1994; Pennington & Bennetto, 1993).

Verbal Deficit

It is generally accepted in the literature that measures of IQ typically obtained on juvenile delinquents (JD) are approximately one half a standard deviation below their non-delinquent peers (Hirschi & Hindelang, 1977; Wilson & Hernstein, 1985). Moffitt (1993a) contends that this likely represents a significant underestimate for persistent juvenile offenders who have a long history of delinquency. Indeed, Moffitt (1993b) has proposed that neuropsychological variables may be potent discriminators of life course versus temporary juvenile offenders. The relationship between lower IQ and delinquency also holds prospectively (Denno, 1989; Moffitt, 1990) and Moffitt and Silva (1988a,b) have shown this not to be simply a matter of detection as self-identified delinquents also have low IQ's.

Another robust finding is the P>V sign in Corrections psychological studies which relates to a difference of one standard deviation on the Wechsler scales in favor of Performance IQ over Verbal IQ as a discriminator or predictor of delinquency. Three major reviews of numerous studies in this area (Prentice & Kelly, 1963; West & Farrington, 1973; Walsh, 1992) suggest that the high incidence of P>V in delinquent populations has been consistently demonstrated. Walsh (1992) also concluded on the basis of his study that the P>V finding is not an artifact of socioeconomic status and not merely a function of low Verbal IQ. Broader neuropsychological studies have also identified significant verbal deficits among JD's.

Berman and Siegal (1978), utilizing the Halstead-Reitan Neuropsychological Test Battery reported a high incidence of impaired functioning in juvenile delinquents, particularly on verbal measures, although the delinquent sample performed significantly worse on most measures. Also using the Halstead Reitan Battery, Fitzhugh (1973) found JD's to be significantly poorer than emotionally disturbed adolescents on many measures.

Sobotowicz, Evans, and Laughlin (1987) contrasted 50 incarcerated delinquents with 50 high school subjects on a broad neuropsychological test battery. Subjects were matched for age, race, and social class. Their sample consisted of four groups; normal, juvenile delinquent, learning disabled, and juvenile delinquent plus learning disabled. The normal group scored significantly higher than the other three groups on many of the more complex, abstract, and/or language-related measures. In general, the delinquent and learning disabled group was most impaired, a result replicated by Henry, Moffitt, and Silva (1992). Moffitt (1993b) reports on a large factor analytic neuropsychological study of New Zealand youth. She found that delinquents versus non-delinquents were substantially lower for verbal and auditory-verbal memory factors than for factors representing visual-motor integration, visuospatial, and mental flexibility functions. She also found that a subgroup of delinquents with ADHD histories were particularly low on verbal measures. Two other neuropsychological studies demonstrating deficits particularly on verbal measures in delinquents are

those of Wolff, Waber, Bauermeister, Cohen, and Ferber (1982) and Karniski, Levine, Clarke, Palfrey, and Meltzer (1982). Studies with seriously delinquent or violent/assaultive adolescents suggest that this subgroup of delinquents may be particularly impaired (Brickman, McManus, Grapentine, & Alessi, 1984; Krynicki, 1978; Lewis et al, 1988; Spellacy, 1977).

Overall, the evidence for lower intellectual functioning in general, and relatively weaker verbal than visuospatial-perceptual organizational neuropsychological function appears to be a robust finding across most studies. The verbal difficulties are broad, affecting listening and reading measures, verbal expressive, and verbal memory abilities.

Executive Deficit/Behavioral Disinhibition

Theoretical proposals postulating frontal lobe dysfunction as a risk factor for delinquency (Buikhuisen, 1987; Gorenstein, 1990; Pontius, 1972; Quay, 1988a; Yeudall, Fromm-Auch, & Davies, 1982) are similar to hypotheses advanced to account for adult antisocial behavior and typically focus on symptoms associated with frontal lobe dysfunction, such as planning deficits, behavioral disinhibition, self-regulatory deficits, impulsivity, or attentional deficits.

Pontius (1972), based on Luria and Teuber's conceptions of frontal lobe function, argued that frontal lobe dysfunctions due to a developmental lag or neuropathological deficit may be the basis for some forms of delinquency. She suggested that such impairment would result in delinquents not being able to shift the principle of actions of an ongoing activity, i.e., a

dissociation between knowing and doing. Pontius and Ruttiger (1976) found found that 70% of normals, but only 47% of delinquents (as rated by teachers) told stories that demonstrated the ability to switch the course of narrative action properly in response to new circumstances.

There have since been a number of studies addressing more specific aspects of prefrontal function in JD's. Skoff and Libon (1987) administered the Wisconsin Card Sorting Test, Porteus Mazes, Trails B, Verbal Fluency, and four additional executive tasks to 22 incarcerated delinquents. Comparing the results of the JD's to published norms they noted that one third of their subjects scored in the impaired range on the battery taken as a whole. Appellof and Augustine (1985) reported (in abstract form) not finding differences between 30 male delinquents and 30 controls on several measures, including the WCST, Verbal Fluency, and Trails B. In contrast, Moffitt and Henry (1989) reported that differences on the WCST, Verbal Fluency, Trails B, Mazes, and a rating of planning in drawing the Rey Osterreith Complex Figure Test significantly discriminated self-reported early delinquents from non-delinquents in the study after the effects of IQ were statistically controlled.

Yeudall et al. (1982) compared institutionalized delinquents to adolescents from regular classrooms in a study that used a modified Halstead-Reitan Neuropsychological Test Battery. They reported an exceptionally high incidence of abnormal profiles in the delinquent group (84%; $\underline{n}=99$) as compared with their non-delinquent group (11%; $\underline{n}=47$).

Most of the abnormal profiles were interpreted as reflecting anterior brain dysfunction.

Lueger and Gill (1990) designed a study to specifically address the issue of frontal lobe deficits in JD's who also warranted a diagnosis of CD. The study involved contrasting scores on the WCST, the Sequential Matching Memory Test (SMMT), The Hand Movements Test from the Kaufman Assessment Battery for Children, Trails A and B, and the Auditory Verbal Learning Test. In this study, 21 court-referred male adolescent subjects who met the diagnosis for CD were contrasted with 20 male normal controls. CD adolescents were mean age 15.0 while controls were 16.2. The groups were virtually identical in ethnic composition and not significantly different on the Peabody Picture Vocabulary Test - Revised (PPVC-R). Significant differences favoring the Normal controls were found for all tests except Trails A and B. Perseverative errors on the WCST, SMMT errors, and the Hand Movements tests yielded a canonical correlation of .66 with group membership. A discriminant function with these three measures correctly classified 18 of 20 (90%) control subjects and 17 of 21 (81%) of the conduct disorder subjects for an overall hit rate of 85%. This study is particularly interesting in suggesting that JD's with CD can be discriminated from Normal controls on frontal lobe measures despite similar verbal IQ as measured by the PPVC-R.

Linz, Hooper, Hynd, Isaac, and Gibson (1990) failed to find differences between conduct disordered juveniles and normal comparisons on Lurian

measures of frontal lobe function, but the measures selected appeared to have very low difficulty levels and differences may have been masked by ceiling effects. Hurt and Naglieri (1992), also contrasting delinquents and normals on Lurian tasks failed, as well, to find significant differences on measures of planning, simultaneous and successive cognitive processing, but did find large significant differences on measures of receptive and expressive language.

Other studies not specifically addressing frontal lobe hypotheses, have found differences on putative frontal measures. Berman and Siegal (1976) found that delinquents scored poorly on the Category Test and Trails B. Wolff et al. (1982) reported significantly poorer performance by delinquents on the Stroop Color Word Test and measures of selective attention. Krynicki (1978) noted the poor performance of delinquents on verbal fluency and motor perseveration. Other studies, e.g., Brickman et al. (1984), Karniski et al. (1982), noted deficits in motor sequencing tasks.

Consistent with Gorenstein's and Quay's hypotheses, Scerbo et al. (1990) found deficits in passive avoidance and reward dominance effects in incarcerated JD's with a high level of psychopathic features compared to those with fewer features. Finally, consistent with verbal mediation theories, Raine, O'Brien, Smiley, Scerbo, and Chan (1990) found reduced lateralization in Verbal Dichotic Listening in adolescents rated high on psychopathy features compared to those rated low.

In sum, neuropsychological theories implicating executive dysfunction, behavioral disinhibition, related to dysfunction of the prefrontal cortex and dysfunction of cortical structures mediating language abilities which have been advanced as risk factors for adult offending, have also been proposed to be relevant to children with ADHD, CD, and adolescent offenders. Research findings with respect to these three groups, typically, though not universally, have found some support and, in most cases, significantly poorer performance has been observed by subjects belonging to these groups as compared to normal control contrast groups. Many methodological factors, such as diagnostic heterogeneity, inadequate measures, inadequate representation of the relevant performance domain, may have led to the under-estimation of differences between groups. A clearer understanding of the relationship between neuropsychological variables and these three groupings will result from a better classification of neuropsychological abilities on the one hand and refinement of diagnostic groups through subtyping on behavioral and personality dimensions. Nonetheless, the evidence reviewed above appears to indicate that a significant proportion of children with ADHD, CD, and adolescent offenders exhibit significant neuropsychological deficits. How, specifically, neuropsychological deficit relates to poor outcome for these groupings remains to be empirically determined. However, for the ADHD and CD groups as a whole, there does appear to be an elevated risk for poor outcome, often poor adjustment, and

in a disproportionate number of cases, clear antisocial behavioral progressions are noted.

Developmental Outcome Studies ADHD and CD

ADHD

Over the past two decades there has been a proliferation of prospective, longitudinal, cross-sectional, and follow-back outcome studies of children diagnosed with ADHD, CD or learning disabilities. For reviews see Barkley (1990), Barkley, Fischer, Edelbrock, and Smallish (1990), Farrington, Loeber, and Van Kammen (1990), Farrington and West, 1992, Hinshaw (1994a, 1994b), Hinshaw and Anderson (in press), Lie (1992), Lilienfeld and Walsman, 1990, O'Shaughnessy (1993), Pennington (1991), Robins and Price (1991), Satterfield, Hoppe, and Schell, 1982, Storm-Mathisen and Vaglum (1994), and Zoccolillo, Pickles, Quinton, and Rutter (1992). While most studies have focused on the relationship of diagnosis to outcomes related to delinquency and antisocial behavior, psychiatric status, substance abuse, and personality features have also been examined.

Bearing in mind the confusing definitional issues related to diagnosis and the transitory nature of some forms of juvenile delinquency, it is critical to qualify statistical relationships between diagnosis of ADHD, CD, learning disabilities, and future negative outcomes, including antisocial behavior, antisocial personality disorder, and notions of psychopathy, yet these relationships are so robust that authors have provided estimates of risks. For instance, Barkley (1990) conservatively estimates that as many as 25 to

40 percent of previously diagnosed ADHD children will develop antisocial personality disorders. Hinshaw's (1994a) analysis of the follow-up literature led him to conclude that one-fourth to one-half of ADHD subjects followed into their teenage years developed antisocial behavior and substance abuse with delinquency or incarceration a common outcome (p. 85). Robins (1978) has concluded that adult antisocial personality disorder never occurs in the absence of marked antisocial behavior prior to age 18. As distressing as these figures are, there is also evidence and hypotheses that specific subtypes within these disorders may be identifiable with remedial implications. For instance, in his comprehensive analysis, Lie (1992) concluded that a diagnosis of ADHD without co-existing CD does not produce an increased frequency of delinquency. However, "In subjects with ADHD as well as conduct problems in childhood, conduct problems and not ADHD predict the prognosis which is worse than for those with CD and without ADHD. ADHD combined with delinquency indicates a high rate of subsequent law breaking" (Lie, 1992, p. 5).

Other studies have sought to identify early precursors of later disruptive behavior disorders and antisocial behavior on the basis of temperament, perinatal complications, early language ability, and early impulse control variables, e.g., Kandel and Mednick (1991), Kelso and Stewart, 1986, McGee, Partridge, Williams, and Silva (1991), Sanson, Smart, Prior, and Oberklaid (1993), Satterfield, Swanson, Schell, and Lee (1994), Shoda,

Mischel, and Peake, (1990), Stattin, Klackenberg-Larsson (1993), White, Moffitt, Earls, Robins, and Silva (1990),

Examining relationships between diagnostic categories of CD and ADHD and associated negative outcomes has been a major focus of research over the past two decades. More recently (e.g., Loeber et al., 1993), there has been a focus on identifying specific pathways to adolescent and adult antisocial behavior within a multidimensional framework. As well, research has begun to examine (e.g., Moffitt, 1990; Moffitt, 1993a,b; Moffitt & Lynam, 1994) the developmental impact of neuropsychological variables for negative developmental trajectories within groups of children diagnosed with ADHD/CD and those with both disorders. Generally, it appears that children with behavior disorders are at significant risk for later offending. Understanding the specific role of neuropsychological variables in modulating this risk will require further research.

## SECTION VI SUMMARY OF THE LITERATURE REVIEW

### Introduction

This literature review has had three main goals. The first goal was to develop an understanding of the role of the prefrontal cortex in adults and its function throughout childhood development. This was achieved by examining: a) theoretical perspectives on the role of the prefrontal cortex in adults; b) the effects of lesions of the prefrontal cortex in adults on both cognitive functioning and personality; and c) the role of the prefrontal cortex in early development by presenting theoretical analyses, associated research,

and case studies of children who sustained early damage to the prefrontal cortex.

The second goal was to review neuropsychological theories and associated research which examined whether neuropsychological deficits, particularly those associated with prefrontal cortex, but also language deficits associated with dominant hemisphere function, are related to persistent adult offending. The third goal was to review the neuropsychology of Attention Deficit Hyperactivity Disorder (ADHD), Conduct Disorder (CD), and adolescent offenders, since these groups have been found to be at considerable risk for developing antisocial behavior patterns, including offending as adults. Of primary interest was whether neuropsychological deficits, e.g., executive dysfunction and behavioral disinhibition observed in adult offenders, could be considered heterotypic continuities of neuropsychological characterizations of children with ADHD, CD, or both disorders.

Role of the Prefrontal Cortex

There have been remarkable advances in understanding the role of the prefrontal cortex over the past 50 years. The review of theoretical perspectives on the functions of the prefrontal cortex noted considerable agreement among theorists as to its overall regulatory function, including organizing behavior in terms of its consequences, monitoring and integrating exteroceptive and interoceptive pocesses, as well as controlling, elaborating,

and modulating emotional influences. Specific contributions by theorists are highlighted below.

Pribram (Miller et al., 1960; Pribram, 1973) proposed that a complex feedback system was necessary to permit complex goal attainment and suggested that a mechanism such as test-operate-test-exit (TOTE) could serve to incrementally propel an organism toward a goal. Tueber (1964, 1966) emphasized the effector role of the frontal lobe. Contrary to previous hypotheses, he proposed that action began in frontal systems and through a mechanism of corollary discharge in which an anticipatory discharge from motor to sensory areas prepared sensory structures for the predicted changes induced by voluntary movement.

Nauta (1971, 1973), based on the unique strategic anatomic position of the prefrontal cortex, proposed that it was ideally situated to integrate exteroceptive and interoceptive modalities. Thus, goals could be achieved through an integration of thought, behavior, and affect.

Damasio (1979, 1985) extended Nauta's neuro-anatomic approach and proposed that preservation of an individual's equilibrium was related to maintaining a balance between exteroceptive and interoceptive processes. He also emphasized the modular and hierarchical organization of prefrontal structures.

Luria (1970, 1973a,b, 1980) referred to the frontal lobes as the neural unit for programming, regulation, and verification of activity. He characterized the frontal lobes as the superstructure responsible for the

regulation of behavior. He also emphasized the role of language, particularly inner speech, in the mediation of complex cognition and complex goal attainment.

Shallice (1989) and Norman and Shallice (1980/1986) incorporated much of Luria's theory within an information processing model. They emphasised executive and decision-making functions of the prefrontal cortex with their construct of "Supervisory Attentional System".

Stuss and Benson (1986), also influenced by Luria, elaborated a four level hierarchy of brain functions. Three of these levels are considered to be primarily frontal functions:

1. Functional systems of a) sequence, set, and integration and, b) drive, motivation, and will.

2. Executive function involving a) anticipation, b) goal selection, c) pre-planning, monitoring, d) the use of feedback.

3. Self-awareness.

Ingvar (1979, 1983a, 1983b, 1985) emphasized the future orientation of the prefrontal cortex. He emphasized the relationship between temporal structures and the perception of causality.

Fuster (1989, 1993) characterizes the prefrontal cortex as the mediator of cross temporal contingencies. He accords a supraordinate role to the prefrontal cortex in developing and executing behavioral plans. Especially on the basis of experimentation with non-human primates, he has proposed a mechanism or model which subserves this function. The subcomponents of

the mechanism involve short-term memory, anticipatory set, and interference control. The dorsolateral and the orbital/medial prefrontal cortex collaborate in its execution.

In sum, theoretical perspectives on the prefrontal cortex have emphasized its supraordinate, controlling and integrating function with respect to cognition, emotion, and behavior. Executive function, the importance of internal language, specific operational mechanisms, and hierarchical models of organization have been emphasized. The effects of lesions, summarized next, provide further understanding of its role and organization.

Lesion Sequelae

Three prefrontal regions, dorsolateral, superior-medial, and orbital/lower medial have been related to distinct sequelae configurations. The superior-medial syndrome is marked by akinesia and is often associated with mutism, gait disturbances, and incontinence.

Lesions limited to the dorsolateral regions, particularly of the dominant hemisphere, usually compromise cognitive function with an admixture of deficits associated with: a) planning and intentional behaviors; b) evaluation of the consequences of one's actions; c) higher cognitive functioning involving abstract reasoning and concept formation; d) sustaining attention and concentration; e) the effective use of language to regulate future behavior; and f) distractibility, impulsivity, and disinhibition.

There have been attempts to account for this broad array of deficits in more parsimonious terms. Luria (1969, 1980) emphasized a defect in selective logical operations. Stuss and Benson (1984, 1986) focused on the disruption of four functions: a) handling sequential behaviors, b) establishing or changing problem sets, c) maintaining problem sets, and d) integrating knowledge with response direction. Fuster proposes that defective short-term memory and defective planning related to defective prospective memory are primarily responsible.

Lesions of the orbital-limbic area of the prefrontal cortex also produce a distinct syndrome typically characterized by some combination of the following: diminished emotional self-control; dramatic personality changes; reduced self-reflective awareness; indifference to emotional feelings or conflicts; affective disorders; increased sexual and aggressive drive disinhibition; increases in impulsive and antisocial behaviors; and reduced tolerance to alcohol. Damasio et al. (1990) propose that lesions of the orbital limbic area produce a disconnection between dorsolateral prefrontal cortex and limbic structures resulting in a lack of cognitive modulation of basic drives and a failure of basic drives to activate higher cortices.

Experimentally, it has been difficult to demonstrate deficits associated with orbital/lower medial cortex, however, Damasio et al. (1990) have demonstrated a lack of automatic emotional responsivity (GSR response) in such patients to socially charged stimuli.

With respect to cognitive sequelae associated primarily with dorsolateral prefrontal lesions, several experimental techniques, in addition to clinical neuropsychological measures, have demonstrated deficits in working memory, verbal and design fluency, cognitive flexibility, conditional associative learning, recency and frequency estimations, planning, strategy application, hypothesis generation, memory for temporal order, problem solving, and passive avoidance.

Thus, clinical and experimental research have demonstrated sequelae defining a broad range of cognitive deficits primarily associated with prefrontal dorsolateral lesions and disinhibitory affective phenomena associated with orbital-limbic lesions. These sequelae have been noted in adult patients and are thought to result from the dissolution of structures consolidating cognitive and personality organization. Next, the role of the prefrontal cortex in the development of those structures is summarized.

Developmental Role of the Prefrontal Cortex

Until quite recently, the common view was that the prefrontal cortex did not play a major role until adolescence. Recently, work conducted by Goldman-Rakic (e.g., Diamond & Goldman-Rakic, 1986, 1989; Goldman-Rakic, 1987a) with infant non-human primates and human infants has demonstrated that as early as 6 to 12 months of age, human infants show the capacity to develop internal representations and delay responding, thus, permitting deliberate behavior choices. This early capacity has been described by Goldman-Rakic as the cornerstone of cognitive development, it

is seen as the foundation of working memory. These findings have since been corroborated by electrophysiological research (e.g., Bell & Fox, 1992). Electrophysiological activity of the prefrontal cortex has also demonstrated its involvement in mediating the emotional behavior of infants.

Several studies reviewed (e.g., Welsh et al., 1991; Becker et al., 1987; Levine et al., 1991) have noted that the development of prefrontal capacities occurs as a multistage process between early childhood and middle adolescence, at which time adult-like performance is the norm. To date, very little work has actually focused on developing a taxonomy of childhood executive or prefrontal function (cf. Welsh et al., 1991), however, some indication of the importance of the prefrontal cortex for normal development comes from case studies.

Case studies reviewed here (e.g., Grattan & Eslinger, 1991; Marlow, 1992; Mateer & Williams, 1991) demonstrate that early damage to the prefrontal cortex can result in a severe impact on early manifestations of executive function, behavioral self-regulation, as well as personality and social development. Phenomenologically, serious ADHD symptomatology is manifest and behavioral disorders are common.

In sum, the view of the relative importance of the prefrontal cortex has been dramatically transformed such that early damage or dysfunction is seen to pose serious developmental risks in several areas. Next, the neuropsychology of adult offenders is summarized.

Neuropsychology of Adult Offenders

Yeudall and his co-workers (e.g., Yeudall, Fedora, & Fromm, 1986) postulated that many of the behavioral deficits exhibited by persistent adult offenders implied cognitive and personality deficits commonly observed in patients with prefrontal damage. To test this hypothesis, they conducted several studies in which they administered a broad clinical neuropsychological test battery to large groups of adult offenders (e.g., Yeudall, 1977; Yeudall & Fromm-Auch, 1979). Using clinical criteria, they evaluated offenders' test profiles and categorized them according to their degree of impairment. Their main conclusions were that offenders were characterized by a high level of impairment on measures traditionally associated with prefrontal (executive function) and left hemisphere damage (verbal functions). Depressive-aggressive offenders were inclined to show a higher degree of non-dominant hemisphere deficits, while psychopathic offenders were more inclined to manifest verbal deficits. Other neuropsychological studies also demonstrated neuropsychological impairment in offender samples (e.g., Spellacy, 1978) and low intellectual abilities (e.g., Heilbrun, 1982, 1990).

The issue of prefrontal deficit among psychopaths was also raised by Gorenstein (1982) and several studies examining this issue produced conflicting findings. In general, reviews of the neuropsychology of offenders by Kandel and Freed (1989) and Miller (1987) found that the empirical research on this issue was inconclusive.

Gorenstein (1982), based on the animal model of the septal hippocampal frontal syndrome (SHF), reasoned that offenders may be characterized by a disinhibitory syndrome attributable to prefrontal dysfunction. With reference to Gray's theory (e.g., 1972), he reinterpreted the literature on psychopathy and autonomic reactivity. Gorenstein concluded that evidence for an autonomic deficit was lacking, and that Gray's theory proposing an over-active Behavioral Activation System (BAS), producing a heightened sensitivity to rewards, did not fully account for the behavioral disinhibition of offenders. He proposed that the central deficit related to a compromized capacity to develop mental representations of relationships among events.

Next, a four stage psychological model to explain behavior disinhibition advocated by Patterson and Neuman (1993) was presented. As a closing comment on this section, it is noted that few studies have attempted to investigate a broad range of neuropsychological abilities among the full spectrum of adult offenders. Yeudall and his co-workers conducted major studies in this respect, but given the hospital setting, questions of representativity have arisen and reporting of findings was done on the basis of clinical judgements rather than specific test results.

Neuropsychology of Childhood & Adolescent Behavior Disorders

Attention Deficit Hyperactivity Disorder (ADHD)

The conceptualization of Attention Deficit Hyperactivity Disorder (ADHD) has been transformed over the past two decades from a disorder defined by inattention, impulsivity, and overactivity (cf. Douglas, 1972) to a spectrum

disorder reflecting dysfunction of the prefrontal cortex (e.g., Evans, Gualtieri & Hicks, 1986; Mattes, 1980). In particular frontal dysfunctions, impacting executive function, working memory (Shue & Douglas, 1992), and behavioral inhibition (Gorenstein, 1990; Quay, 1988b) have been proposed. Most recently, Barkley (1994) has postulated that a primary deficit in delayed responding, mediated by the prefrontal orbital cortex, can account for most of the diverse manifestations of the disorder. Barkley further suggests that much of the associated deficits in self-regulation relate to the lack of development of internal language, also considered to be primarily a prefrontal function.

Some support for localizing the disorder to the prefrontal cortex comes from brain metabolic studies (e.g., Lou et al, 1984) and neuropsychological studies. The latter have generally found that children with ADHD perform poorly on clinical neuropsychological measures relative to controls (cf. Barkley et al., 1992).

Conduct Disorder (CD)

The primary neuropsychological theory of Conduct Disorder (CD) is that of Quay (1986, 1988a, 1993). This theory relates to Gray's (e.g., 1972) theory of disinhibition. In the case of children, it is posited that they have an overactive Behavioral Activation System (BAS) which leads to a hypersensitivity to reward. There have virtually been no comprehensive neuropsychological investigations of CD, although lower IQ, especially Verbal IQ, has been a consistent finding (Moffitt & Lynam, 1994).

Experimental paradigms addressing the hypersensitivity to rewards hypothesis have produced significant results, however, Gorenstein's (1991) view that a cognitive difficulty in maintaining abstract representation of punishment would provide an alternative explanation of findings. Clear interpretation of many of the studies of ADHD and CD are obfuscated by the high rates of comorbidity between these, as well as with other disorders.

Delinquency

Neuropsychological studies of young offenders have generally identified deficits in verbal abilities (cf. Moffitt, 1993; Moffitt & Lynam, 1994). As well, studies which have assessed executive dysfunction have typically confirmed such deficits (e.g., Lueger & Gill, 1990), although there have been non-significant findings (e.g., Linz et al., 1990). Similarly, studies addressing the disinhibition hypotheses have yielded positive results (e.g., Scerbo et al., 1990).

Taken as a whole, despite methodological limitations, there appears to be considerable evidence that a significant proportion of children and adolescents with ADHD and CD, or both disorders, are characterized by impairment on measures designed to assess prefrontal functions, especially executive function and behavioral disinhibition. Adolescent offenders also present a similar picture.

Finally, development outcome studies of children with ADHD, CD, or both disorders, suggest that a disproportionate number of these children will become serious adolescent and adult offenders. To what extent

neuropsychological variables mediate a negative outcome within these groups remains an important goal for further research.

# CHAPTER III METHODOLOGY

## SECTION I INTRODUCTION

The primary empirical focus of this research is to compare and contrast the performance of three groups of participants, normal controls (NC), offenders (OF), and acute psychiatric patients (PP) on an extensive battery of neuropsychological measures. The purpose is to provide a neuropsychological classification scheme including a typology and normative data for the OF and PP groups.

This chapter is divided into four sections. This introduction comprises Section I. Section II identifies the three groups of participants in the study, i.e., the NC, OF, and PP groups, and describes testing and data collection procedures. Section III provides a review of the neuropsychological measures employed in this research. Section IV briefly describes the methodology, hypotheses to be evaluated and an overview of statistical analyses which are planned. The section concludes with a discussion of ethical considerations.

## SECTION II PARTICIPANTS

Three groups of participants are included in this study. As a result of a longstanding collaboration agreement between the Alberta Hospital, Edmonton, Alberta and the Regional Psychiatric Centre (Pacific) operated by the Correctional Service of Canada, data on normal controls and acute psychiatric patients were provided by the Alberta Hospital, and data on the offender group was obtained from the Regional Psychiatric Centre (Pacific).

Offenders

The offender group consists of 584 North American male, Caucasian adult offenders (mean age 28.78 years) serving prison terms in excess of two years and admitted to the Regional Psychiatric Centre (Pacific) in Abbotsford, British Columbia between September 1978 and July 1986. These participants were consecutive admissions to the Assessment Program except that native Indian or part native Indian, psychotic or organic brain syndrome patients, and patients for whom communication in English was problematic due to cultural background factors were excluded from the study.

The Regional Psychiatric Centre, during the period of data collection, was a 135 bed psychiatric facility operated by the Correctional Services of Canada and accredited by the Canadian Council of Hospital Accreditation. The Centre principally provided psychiatric/psychological programming for the approximately 1,500 federal incarcerates in the Pacific Region of Canada. At the time, the Assessment Program, consisting of 15 beds, provided assessments of voluntary candidates for three programs at the Centre: a) a 30 bed program for clients convicted of sexual offenses; b) a 30 bed program for clients with personality disorders, but who were not adjudicated for sex offenses; and c) a 30 bed program for clients who were assessed to be of limited intellectual ability or to be particularly vulnerable emotionally.

The general criteria for admission to these programs were that admission was voluntary and the clients were advised that they could terminate their treatment program at any time. Further, clients who were still awaiting adjudication on charges or appealing a conviction were not admissible.

Selection criteria for the 30 bed Sex Offender Program and the 30 bed Personality Disorder Program were that intellectual ability be generally within the average range. These two programs were insight oriented and recruitment was biased towards clients with good verbal communication skills, while the third program typically included a majority of clients with more limited intellectual ability.

A survey of the characteristics of offenders serving 6 months or longer in treatment at the Centre conducted by Mandelzys (1979) reported overall, (including clients who were excluded from the present study, e.g., psychotic patients) that approximately 25% of the patients were convicted of sex offenses, of which approximately 80% were of a violent nature. Almost 19% of the population were charged with either murder or manslaughter, and for the group as a whole, approximately 75% of all of the offenses were violent in nature.

Overall, while demographic variables other than age, such as criminal offense history is unavailable for the present sample, there is no reason to expect that the composition of the present sample varies greatly from that described by Mandelzys (1979) and indeed, there should be no question that the offender group in this study consisted of very serious offenders.

It is difficult to establish the representativeness of the present sample to the general population of inmates because of the complexity of human characteristics. Generalization will therefore have to be made with some caution until the results of this investigation are replicated in other correctional settings.

Contrast Groups

The normal controls consisted of 132 male community volunteers (mean age 28.94 years) and forms part of the Alberta Hospital's Research Centre's neuropsychological database. The participants included in the normative database, except for a small number of exclusions and additions (to accommodate the parameters of this study) are essentially those whose normative data have been previously reported in Yeudall, Reddon, Gill, and Stefanyk (1987) and Yeudall, Fromm, Reddon, and Stefanyk (1986).

The psychiatric patients group consists of 494 acute male patients (mean age 27.92 years) admitted to the Admission Unit at the Alberta Hospital in Edmonton, Alberta.

In sum, a large offender sample and two contrast groups are provided for comparison in this study, the total number of participants is 1,210.

Testing Procedures

Hospital policy throughout the period where the data of the present study was collected was to administer the battery of measures listed below in Table 3.1 to all admissions on the assessment ward prior to placement on

a treatment program. The only exception was for clients who were unable to comply with the assessment procedures, e.g., some psychotic patients.

Approximately 90% of the neuropsychological testing was administered by two experienced psychometricians who were further trained in the standardized administration of the neuropsychological battery at the Alberta Hospital in Edmonton, Alberta. The remaining approximately 10% of cases were tested by two registered clinical psychologists employed at the Centre.

Testing typically varied between five and eight hours depending upon the level at which the client was functioning. Testing breaks were provided as required and testing sessions did not exceed 2 hours in the morning session or afternoon session.

Data for each variable was recorded on a customized data sheet and subsequently transcribed onto PC floppy disc for statistical analysis. The policy and procedures for the collection of data for both the NC and PP groups was identical, although conducted at the Alberta Hospital.

SECTION III NEUROPSYCHOLOGICAL MEASURES

The neuropsychological measures included in this study were based on the neuropsychological battery assembled by Yeudall and his co-workers at the Alberta Hospital, Edmonton, Alberta in 1978. It consists essentially of the Halstead-Reitan Battery supplemented by several other measures emphasizing verbal and memory abilities incorporated from several other neuropsychological labs and researchers, e.g., Benton, Milner, and Williams.

Normative data on most measures have been published by Yeudall, Fromm, Reddon, and Stefanyk (1986) and Yeudall et al. (1987).

TABLE 3.1

List of Neuropsychological Measures In Order of Review

1. Wisconsin Card Sorting Test (Grant & Berg, 1948).
2. Halstead Category Test (Halstead, 1947).
3. Controlled Word Association Test (Benton & Hamsher, 1978).
4. Written Word Fluency (Lezack, 1983; Yeudall, 1983).
5. Trail-Making Test (Adjutant General's Office, War Department, 1944).
6. Tactual Performance Test (Halstead, 1947).
7. Seashore Rhythm (Seashore, 1919; Seashore, Lewis, & Saetveit, 1960).
8. Seashore Speech Sounds Perception Test (Seashore, Lewis, & Saetveit, 1960).
9. Dynamometer Test (Reitan & Wolfson, 1985).
10. Finger-Tip Number Writing Perception (Reitan, 1969).
11. Tactile Form Recognition (Reitan, 1969).
12. Finger Localization (Reitan, 1969; Benton, 1955).
13. Face Hand Test (Reitan & Wolfson, 1985).
14. Purdue Pegboard (Purdue Research Foundation, 1948).
15. Symbol Digit Modalities (Smith, 1968, 1973, 1982).
16. Symbol-Gestalt (Stein, 1962, 1970).

17. Coloured Progressive Matrices (Raven, 1949).
18. Williams' Clinical Memory - Subtests: Verbal Learning and Non-Verbal Learning (Williams, 1968).
19. Memory For Designs (Graham & Kendall, 1960).
20. Language Modalities Test for Aphasia (Wepman & Jones, 1961).
21. Minute Estimation Test (Benten, Van Allen, & Fogel, 1964).
22. L.J. Tactile Recognition (Yeudall, 1983).
23. Annett Measures (Annett, 1970).
24. The Wechsler Adult Intelligence Scale (WAIS) or Wechsler Adult Intelligence Scale - Revised (WAIS-R), Wechsler (1981).

Below, each of the measures listed in Table I above is described and associated research briefly discussed. Where available, psychometric indices are provided. Tests reviewed are grouped into four sets: A. Putative measures of prefrontal cortex function; B. Halstead-Reitan Battery Tests utilized in the current study except for the Halstead Category Test and the Trail-Making Test which are viewed under A. above; C. Selected neuropsychological tests employed in the current study; and D. The Wechsler Intelligence Scales.

Putative Measures of Prefrontal Function

Tests reviewed here include:

1. Wisconsin Card Sorting Test (WCST) (Grant & Berg, 1948)
2. Halstead Category Test (Halstead, 1947)
3. Controlled Word Association Test (Benton & Hamsher, 1978)

4. Written Word Fluency Test (Lezack, 1982; Yeudall, 1983)

5. Trail-Making Test (Adjutant General's Office, War Department, 1944)

Wisconsin Card Sorting Test (WCST)

The WCST was originally developed as a quantifiable measure of "human abstraction and shift of set" (Berg, 1948; Grant & Berg, 1948). In this study, the WCST manual and cards, as distributed by Wells Printing Company, were used (Grant & Berg, undated). A slightly modified version has been developed by Heaton (1981) and is distributed by Psychological Assessment Resources.

The WCST consists of 2 decks of 64 cards on which one to four symbols are printed. These symbols are of varying forms (crosses, circles, squares, or triangles), colors (red, green, blue or yellow), or number (one, two, three, or four). An example would be a card with three (number) blue (color) circles (form). No two cards are identical. Subjects are provided with a deck of 64 cards. In front of them are placed four stimulus cards arranged horizontally. The cards are: one red triangle, two green stars, three yellow crosses, and four blue circles. The subject is instructed to place each consecutive card from the deck of cards in front of one of the four stimulus cards, wherever he or she thinks it should be placed. The subject is informed only whether each response is "right" or "wrong" depending on which "correct" principle is operative at the time. Once a subject has made 10 consecutive "correct" responses, originally to color, the criterion is changed without notice to form, until the subject has made 10 sorts to this

criterion, then the criterion is changed to number without notice, then back to color, and so on.

Measures derived in this study include:

1. Subtests Completed
2. Perseverative Errors
3. Non-Perseverative Errors
4. Unique Errors
5. Total Errors
6. Total Correct

Traditionally the WCST has been viewed as a measure primarily of the ability to form cognitive sets or concepts, to maintain them in the face of distraction and to shift cognitive sets. Bond and Buchtel (1984) noted 11 distinct "cognitive capacities" (p. 253) required to achieve an efficient performance on the test. More recently, computer models have been elaborated to accommodate various theories of neurocognitive function, e.g., Parks, Levine, Long, Crockett et al. (1992) have abstracted the principles of the WCST and demonstrated how it could be performed by a parallel distributed process. Dehaene and Changeux (1991) have developed algorithms to solve the test and abstracted three critical requirements: a) the ability to change the current rule when negative reward occurs, b) the capacity to memorize previously tested rules to avoid testing them twice, and c) the ability to reject some rule choices apriori by reasoning. Sullivan, Mathalon, Zipursky, Kersteen-Tucker et al. (1993) factor analyzed the WCST

results of a sample which included schizophrenics, alcoholics and controls yielding three factors: Perseveration, Inefficient Sorting and Non-Perseverative Errors.

On logical grounds, the WCST appears to be factorially complex as well. Wang (1987) has noted four distinct processes involved in successful resolution of the WCST. These include:

1. Education of hypotheses.
2. Hypotheses testing in relation to feedback.
3. Maintaining set.
4. Recognizing changes in conditions and flexibility in thinking in order to shift response approach.

Wang (1987) believes that processes 2) and 4) are particularly dependent upon the frontal lobe whereas 1) and 3) may be only indirectly so. It seems possible that both the orbital/medial prefrontal cortex and dorsolateral prefrontal cortex may jointly contribute to the resolution of the WCST. Consider Fuster's (1989) perspective on prefrontal function. In terms of Wang's analysis, both short term retrospective memory and prospective memory are required to accomplish processes 2) and 4). However, orbital prefrontal integrity would be required according to Fuster in order to avoid distraction. Clearly the WCST also represents in microform Fuster's critical prefrontal function of mediating cross temporal contingencies. This aspect would perhaps be more easily appreciated if the WCST stimuli were removed from a subject's view for short periods between feedback and response

facets. Finally, the WCST taps fundamental aspects of logical cause/effect relationships, i.e., at any given point of negative feedback reasoning is required that e.g., if principle A (color) is not operative, then principle B (shape) or C (numeric) must be applied.

In practice, clients are seen to do poorly on the WCST for four main reasons: a) an inability to deduce the principle, e.g., form; b) perseverative responding; c) difficulties maintaining set; and d) impulsive responding. It must be concluded that the WCST is factorially complex and interpretation of results with both individual and group data must consider this. Although norms are currently available, test-retest, split-half, or other forms of reliability are not currently available (Spreen & Strauss, 1991).

Research Findings

Milner's (1963a,b; 1964) studies of patients with frontal and nonfrontal excisions for relief of epilepsy consistently found that the performance of the former, especially with regard to perseverative errors on the WCST, was generally much poorer than that of patients with nonfrontal excisions. Subsequent studies have only partially supported these findings. Milner (1971) had suggested that left frontal structures were especially crucial for success on the WCST, however, while generally confirming that frontal patients were worse than nonfrontals on the WCST, laterality effects were not significant in studies by Drewe (1974), Malmo (1974), Heaton (1981) and Taylor (1969). Robinson, Heaton, Lehman and Stilson (1980) also found that both left and right frontally damaged patients were significantly

more impaired than nonfrontal groups on perseverative errors. However, as in the Bornstein (1986a) study, no laterality effects were noted. As well, Robinson et al. (1980) and Pendleton and Heaton (1982) failed to discriminate a diffuse brain damage group from the frontally damaged group.

Recent studies have cast further doubt on the specificity of the WCST as a frontal measure. In particular, Grafman, Jonas and Salazar (1990) found in a large study involving 421 brain damaged patients and 84 controls, that while all brain damaged groups committed significantly more perseverative errors on the WCST and achieved fewer categories than controls, differences between prefrontally damaged groups and groups with damage at all other locations were not statistically discriminable. Similarly, Anderson, Damasio, Jones and Tranel (1991) were unable to discriminate frontal ($\underline{n}=49$) from nonfrontally ($\underline{n}=24$) damaged patients. Noting the factorial complexity of the WCST and the functional diversity of the frontal cortices together with their interactions with nonfrontal cortices, these authors stressed their nonsurprise with their findings while noting that a better understanding of brain systems may yield consistent relationships between these and WCST performance. Evidence for a systems perspective (cf. Parks, Crockett & McGeer, 1989) is suggested by the findings of Hermann, Wyler and Richey (1988). These authors investigated the WCST performance in pre-surgical patients with complex partial seizures (CPS) of dominant ($\underline{n}=16$) or nondominant ($\underline{n}=19$) temporal lobe origin and a control group ($\underline{n}=6$) of epilepsy controls. Utilizing Heaton's (1981) cut-off score of 20

perseverative errors on the WCST, 74% (14 of 19) of the nondominant, 39% (6 of 16) of the dominant temporal lobe patients, and 16% (1 of 6) of the epileptic controls had scores of 20 or more perseverative errors, placing them in the "frontal" brain damage range.

The first 17 patients (7 dominant hemisphere, 10 nondominant hemisphere) who underwent temporal lobectomy were retested 6 months post-operatively. Pre-operatively, this group had a mean perseverative error rate of 27.8 whereas post-operatively, their error rate was only 14 which is well below Heaton's brain damage cut-off score of 20. Hermann et al. (1988) argued that this reduction in error scores was not due to practice effects, but due to a reduction in neural noise propagated from the epileptic focus to the prefrontal cortex. In support of their argument they noted that they were able to demonstrate orbitofrontal spiking during intra-operative electrocorticography during pre-resection and that this spiking is markedly attenuated or disappears upon post-resection. Further, these authors note the direct link of the hippocampus which is highly epileptogenic with the dorsolateral prefrontal cortex.

The findings above, taken as a whole, lead to the conclusion arrived at by Anderson et al. (1991) and supported by Mountain and Snow (1993) that performance on the WCST cannot be interpreted in isolation as a measure of frontal lobe damage.

This conclusion should not be interpreted to mean that the prefrontal cortex is not crucial, or the main structure involved, in coordinating and

integrating the diverse facets of the WCST tasks. Milner's (1963a,b; 1964) findings that excisions of the dorsolateral aspect (dominant and nondominant) interfere with performance on the WCST has been replicated in several studies. What has also been observed is that damage in other brain regions, particularly diffuse damage often also results in poor WCST performance. This may be partially due to disruption of structures and functions recruited by the prefrontal cortex in performing its role vis-à-vis the WCST. These observations are consistent with a systems perspective, e.g., Parks, Levine, Long, Crockett et al. (1993). For instance, Owen et al. (1993) reported that on the WCST, patients with frontal lobe damage had particular difficulty shifting attention from a previously irrelevant dimension in contrast with medicated patients with Parkinson's disease who were worse at shifting to a previously irrelevant dimension. Possibly this finding reflects a contribution from the basal ganglia required in the performance of the WCST. At a broader level, Kimberg and Farah (1993) have created a computer model wherein they have demonstrated that a weakening of associations among elements in working memory could impair performance on several frontal tasks including the WCST. Thus a perspective that recognizes that the dorsolateral prefrontal cortex is dependent on other prefrontal structures and beyond the prefrontal cortex can accommodate these experimental findings. Also problematic is the report by, e.g., Bigler (1988) and Anderson et al. (1991) that certain patients with large prefrontal lesions can successfully perform the WCST. It appears that no study to date

has closely examined such cases anatomically to specifically determine if structures thought to be critically involved in the performance of the WCST, e.g., Broadmann Area 9 were affected.

Compelling evidence that the WCST is especially dependent on prefrontal cortex has been obtained from Regional Cerebral Blood Flow (rCBF) and Positron Emission Tomography (PET) studies of patients with schizophrenia. Early studies (e.g., Ingvar & Franzen, 1974) demonstrated that patients with schizophrenia had reduced rCBF in dorsolateral prefrontal regions during both resting and various conditions of cortical activation, i.e., following the presentation of cognitive and sensory stimuli. They observed that the degree of prefrontal hypometabolism was correlated with clinical ratings of negative symptoms and they postulated that hypoactivity of prefrontal cortex was the mechanism responsible. Later studies (e.g., Berman, Torrey, Daniel, & Weinberger, 1992; Weinberger, Berman & Illowsky, 1988; Weinberger, Berman & Zec, 1986) utilizing the WCST as an activation challenge demonstrated that unlike normal controls, medication free, as well as treated patients with schizophrenia, show an inability to increase rCBF in the dorsolateral prefrontal cortex above a sensory motor task control baseline while performing the WCST.

Berman et al. (1992) examined monozygotic twins discordant for schizophrenia and found that the affected twin consistently had less prefrontal rCBF during the WCST than the unaffected twin. Weinberger, Berman, Suddath, and Torrey (1992) replicated this finding with another

group of twins discordant for schizophrenia and concluded that the WCST performance difficulties correlated with hypofrontality are due to disease-related causative factors and not genetic factors per se.

Weinberger, Berman, Suddath and Torrey (1992) found a strong relationship between prefrontal rCBF, perseverative errors on the WCST and both right and left hippocampal volumes in schizophrenia affected discordant twins. Such relationships were not observed among the unaffected twins. Stabenau and Pollin (1993) replicated these findings and also related them to neuroanatomic indices. Specifically, they found that affected monozygotic twin pairs showed reduced prefrontal rCBF activation during the WCST and this correlated with decreased hippocampal volume as determined by Magnetic Resonance Imaging (MRI). Raine, Lencz, Reynolds, Harrison et al. (1993) found with a sample of 17 patients with schizophrenia, 18 psychiatric controls and 19 normal controls that the schizophrenics had the most perseverative errors of the three groups on the WCST and the smallest prefrontal brain structures as assessed by MRI.

In sum, a strong relationship between prefrontal metabolic activation using rCBF technology and success on the WCST has been observed in studies which have contrasted schizophrenic patients and controls. These consistent findings strongly suggest that the prefrontal cortex is intimately involved in the performance of the WCST (cf. Weinberger & Berman, 1988).

PET studies have been less consistent due to methodologic factors (Hyde & Weinberger, 1990), although these authors concluded that in "every PET

or rCBF study that has examined patients during a cognitive task that emphasized prefrontal function, patients with schizophrenia have been 'hypofrontal'" (p. 278). Rubin, Holm, Friberg, Videbech (1991), using Single Photon Emission Computed Tomography (SPECT) have demonstrated the typical result reported above in first episode (pre-medication) schizophrenic and schizophreniform disorder. The authors noted these patients had impaired ability to activate mainly the left prefrontal cortice and had poor WCST performance. Of interest, they noted that the control subjects suppressed striatal activity during the WCST, whereas the patients did not.

Adams et al. (1993) administered the WCST to older alcoholics (mean age 51.1 years) and noted hypometabolism in the mesial frontal cortex, but not the left prefrontal area. Perhaps, these subjects could not get beyond the attentional requirements of the test to engage in its logical requirements. Berman, Doran, Pickar, and Weinberger (1993) contrasted schizophrenic patients, depressed patients, and normal controls in a rCBF study using the WCST as a challenge. They noted the commonly observed hypofrontal focus among the schizophrenic patients, but noted no differences between controls and depressed patients, although the latter had difficulties on the WCST. They concluded that the pathophysiological mechanisms underlying hypofunction in depression and schizophrenia may be different.

In normals, Cantor-Graae, Warkentin, Franzén, and Risberg (1993), using the WCST and a word fluency test observed prefrontal augmentations in rCBF with the latter challenge, but not with the WCST. They concluded that

the word fluency test posed a greater challenge. In a SPECT study, Rezai et al. (1993) contrasted activation produced by the WCST, the Continuous Performance Test, the Tower of London, and Porteus Mazes in a large sample (15 subjects per measure) of normals. They found significant increases in frontal regions in all tests except for the Porteus Mazes. Of particular interest, the WCST produced a left prefrontal activation pattern, whereas the Continuous Performance Test and the Tower of London produced mesial and bilateral activation which they suggest may reflect stimulation of midline attentional circuits. Lastly, Arnett et al. (1994) contrasted three groups of patients with multiple sclerosis characterized by frontal white matter lesions, minimal frontal lesion, and non-frontal lesions of comparable size. They noted that the group with maximal frontal lesions were most impaired on the WCST.

On the basis of the above review, despite certain anomalous findings, it would seem difficult to argue that the left dorsolateral prefrontal cortex is not critically involved in the performance of the WCST. There is also evidence that mesial prefrontal areas are also necessary, particularly with respect to maintaining attentional set, (e.g., Adams, et al., 1993). Other studies (e.g., Cantor-Graae et al., 1990) which did not demonstrate a prefrontal focus may be accounted for by differential sensitivities or to ceiling effects among normals. Also, in many studies, highly significant results were obtained despite small group samples. Thus, the WCST

appears to be a prefrontal measure of exceptional sensitivity, if not of equivalent specificity.

Halstead Category Test (HCT)

The HCT, developed by Halstead (1947) is also part of the Halstead-Reitan test battery (Reitan & Davison, 1974). Throughout this test the client is seated in front of an opaque glass screen on which a total of 208 slides are serially projected. Below the screen are response keys which consist of four numbered lights with a spring loaded switch below each light. Initially the client is told that he/she will see a series of slides on the screen and that each slide will remind her/him of a number between 1 and 4. The client is then instructed to push the switch corresponding to the number that he/she is reminded of and that a correct response will be followed by the sound of a bell. If, on the other hand, the client presses a switch corresponding to an incorrect answer, a noxious-sounding buzzer will be heard.

The test consists of 7 subtests and prior to each test the client is told that there is a common principle or idea that governs the determination of the correct response in each subtest. Each set of stimuli is organized on the basis of a different principle, such as number of objects or ordinal position of an odd stimulus. As the test progresses the principles become more complex. Stimuli differ along several dimensions, such as size, shape, color, position and solidness of the figure. Clients must use feedback they receive from their correct and incorrect guesses on the series of items in each subtest to infer the principle underlying each subtest. The seventh test is

different and introduces a memory component as this subtest repeats items from the first six subtests (Spreen & Strauss, 1991; Jarvis & Barth, 1994).

Psychometric Properties

The odd-even split-half method and coefficient alpha have been used to calculate internal consistency estimates for the HCT. Consistently high reliability coefficients in excess of .95, have been reported in both samples of normal and brain damaged adults (Charter, Adkins, Alekoumbides & Seacat, 1987; Moses, 1985; Shaw, 1966).

Retest reliability in excess of .90 has been reported (Goldstein & Watson, 1989) with chronic brain damaged groups. With short test re-test periods and diagnostic groups with unstable pathology, the reliabilities are somewhat lower. Eckardt and Matarazzo (1981) obtained a reliability coefficient of .74 in a sample of hospitalized alcoholics and .87 in a sample of hospitalized nonalcoholics. Bornstein (1985) reported a coefficient of .70 in a sample of volunteers from a university community.

Studies examining the relationship between intelligence measures and the HCT have yielded differing results. Reitan (1956) found correlations of -.58 and -.64 between the HCT and the Wechsler-Bellevue Verbal IQ and the Performance IQ respectively. Lin and Rennick (1974) found correlations of -.51 and -.68 between Wechsler Adult Intelligence Scale (WAIS) Verbal IQ and the HCT for two age samples of epileptic patients. Shore, Shore and Pihl (1971) reported a correlation of .84 between selected WAIS performance subtests and HCT errors for a sample of subjects without brain

damage. Goldstein and Shelly (1972), Lansdell and Donnelly (1977) also noted their highest correlations with WAIS performance tests. Cullum, Steinman, Bigler, and Erin (1984) found PIQ correlated -.52 and VIQ -.31 for their sample of head injured patients. Wiens and Matarazzo (1977) reported a modest correlation of -.29 between the HCT and the WAIS in a sample of young police applicants. In a mixed sample of 619 neuropsychiatric brain and non- brain damaged patients, Goldstein and Shelley (1972) reported correlations between the HCT and WAIS subtests ranging from .39 (Comprehension) to .63 (Block Design). In a normative sample of 127 males, Yeudall et al. (1987) reported correlations of -.39 and -.36 between the HCT, Verbal IQ, and Performance IQ respectively. Corrigan, Agresti and Hinkeldey (1987), in a study specifically designed to assess the HCT-WAIS Verbal IQ and Performance IQ relationships in a sample of 102 subjects with either closed head injury or cerebrovascular accident found a HCT-Performance IQ correlation of -.64, however, HCT-Verbal IQ was not significantly correlated (r-.11) in this study. The lack of consistency in HCT-Verbal IQ and HCT-Performance IQ correlations does not readily lend itself to interpretation, although it does suggest that homogeneity of covariance assumptions across diagnostic samples should be made with caution.

Although correlations between HCT and Performance IQ are often substantial and the HCT typically has aligned itself with Performance IQ subtests in factor analyses (Lansdell & Donnelly, 1977; Moehle, Rasmusen & Fitzhugh-Bell, 1990; Reitan & Wolfson, 1993; Swiercinsky, 1979), there

have been exceptions, e.g., Thomas and Trexler (1982) found only a modest loading on a factor interpreted as right hemisphere cognition and motor performance. Further, despite its relationship with Perfromance IQ, the HCT has not shown the potential to predict cerebral lateralization in clinical lesion studies (Bigler, Steinman & Newton, 1981a,b; Bornstein, 1986a; Chapman, Boring & Wolff, 1959; Cullum et al., 1984; Doehring & Reitan, 1962; Reitan & Wolfson, 1993; Shure & Halstead, 1958), yet it has been found to be the most sensitive single test in the Halstead-Reitan battery for distinguishing brain damaged patients from normals (Klove, 1974; Lezack, 1983; Reitan & Wolfson, 1993; Wheeler, Burke & Reitan, 1963).

HCT-WCST Contrasts

Wang (1987) has pointed out that the originators of the WCST and the HCT (Grant & Berg, 1948; Goldstein & Scheerer, 1941; Halstead, 1947, 1950; Weigl, 1941) shared the same zeitgeist and in both instances the objective was to develop a measure of abstraction ability. Several studies have since examined the empirical and conceptual similarities between these two tests (Bond & Buchtel, 1984; Bornstein, 1986a; King & Snow, 1981; Pendleton & Heaton, 1982; Perrine, 1993; Rothke, 1986). Empirically, King and Snow (1981) reported correlations of -.53 and -.43 for their brain damaged and normal control groups respectively between the number of categories achieved on the WCST and the total number of errors on the HCT. Similarly, Pendleton and Heaton (1982) found correlations of .5 and .55 respectively for their brain damaged and control groups between

perseverative errors on the WCST and total errors on the HCT. In view of these modest correlations (but which may be spuriously low, cf. Bond and Buchtel (1984) mainly because of restricted variances) most authors have assumed at least partial independence of these measures and have recommended the use of both measures. There is some debate, however, as to the nature of the different abilities tapped by these two measures. Rothke (1986), in a study specifically designed to examine these relationships found that order of administration had no significant effect and concluded that this was evidence of their nonsimilarity. They reasoned that otherwise practice effects would be manifest. Rothke (1986) also addressed whether cuing with respect to intertest shifts in sorting demands was critical. He found that eliminating set shifting cues with the HCT has little effect, likely due to implicit information within the test of the need to shift set. Rothke (1986) concluded that mental set shifting was of paramount importance in the WCST. They attributed this capacity to the frontal lobes. Similarly Pendleton and Heaton (1982) found that while both measures were often in agreement in predicting the presence of lesions, the WCST had an advantage in identifying focal frontal lesions while the HCT was superior in identifying nonfrontal focal or diffuse lesions. Conceptually, most authors suggest that the overall cognitive demands of the HCT are far greater than those of the WCST. More specifically, Bond and Buchtel (1984) state that the perceptual-abstraction and hypothesis generation and testing requirements of the HCT are greater than those of the WCST. Perrine (1993) concluded that

the WCST was associated with attribute identification (discrimination of relevant features) while the HCT was more related to rule learning which involves the deduction of classification rules. Spreen and Strauss (1991) stress the capacity of the WCST to identify perseverative tendencies while they suggest that the HCT is a more sensitive and difficult measure of abstraction ability.

In sum, the two tests are not identical. Both appear to be factorially complex, certainly not pure measures of prefrontal function, and further delineation of the ability components involved is required (cf. Wang, 1987).

Controlled Word Association Test (CWAT) & Written Word Fluency Test (WWFT)

The CWAT (Benton & Hamsher, 1978) is part of the Benton battery and is included in the Multilingual Aphasia Examination. The test requires the subject to say as many words as he can (given certain constraints, e.g., no proper nouns) in a 1-minute period for each of the letters, F, A, and S. The average of the total responses for each of the three letter trials and their total average is scored.

Psychometric Properties

Snow, Tierney, Zorzitto, Fisher, and Read (1988) reported one-year retest reliability in older adults to be .7 for F, .6 for A, and .71 for S. desRosiers and Kavanagh (1987) reported a retest (with inter-test intervals between 19 and 42 days) reliability of .88.

Research Findings

Several studies have found the CWAT to be particularly sensitive to patients with frontal, particularly left frontal lesions (Benton, 1968; Crockett et al., 1986; Pendleton & Heaton, 1982; Perret, 1974; Ramier & Hécaen, 1970). Welsh et al. (1988) found, in a normative developmental study, that controlled word association abilities were among the latest of prefrontal abilities to mature. Spellacy and Brown (1984) noted that poor word fluency combined with poor school achievement test scores were a significant predictor of recidivism in adolescent offenders.

SPECT CWA studies have identified maximal activation of the mid-dorsolateral prefrontal cortex in normal subjects (Boivin et al., 1992; Parks et al., 1988) and similar findings were reported in an Echoplanar Magnetic Resonance study (McCarthy et al., 1993). Cantor-Graae et al. (1993) have reported significant rCBF in the left dorsolateral prefrontal cortex during CWA testing in normal subjects.

In many of the above studies, many other brain structures were noted to be activated, however, Frith et al. (1991) have postulated that words are represented in the superior temporal regions and that inhibitory modulation of these areas by the left prefrontal cortex is the basis of intrinsic word generation.

The Written Word Fluency Test (WWFT) (Lezack, 1982, p. 333; Yeudall, Fromm, Reddon, & Stefanyk, 1986) is identical to the CWAT except that the subjects are required to write rather than orally state words beginning with

"F", "A", and "S". Although norms have been published (Yeudall, Fromm, Reddon, & Stefanyk, 1986), practically no research findings have been published.

Trail Making Test (TMT)

The TMT comprises two subtests, A and B. In Part A the numbers from 1 to 25 are randomly presented in a standardized format and the subject is required to sequentially connect the numbers. In Part B, thirteen numbers (1-13) and twelve letters (A through L) are randomly presented in a standardized format and the subject is required to sequentially connect the first number (1) with the first letter (A), then the second number with the second letter, and so on (i.e., 1 → A → 2 → B → 3 → C . . .). Time to completion for both Parts A and B are recorded.

Psychometric Properties

Goldstein and Watson (1989) reported reliability coefficients ranging between .69 and .94 for Part A and between .66 and .86 for Part B for an alcoholic/trauma and a vascular disorder group respectively. Charter et al. (1987) found reliabilities of .89 and .92 for Part A and Part B respectively on alternate forms. In a similar study, desRosiers and Kavanagh (1987) reported reliabilities of .80 and .81 respectively. Dye (1979) and Stuss, Stethem, and Poirier (1987) reported practice effects after a short interval and after one week for both Part A and Part B. Lezack (1983), in the course of three administrations at six month intervals, found practice effects only for Part A and reliabilities of .98 for Part A and .67 for Part B (coefficient of

concordance). Matarazzo et al. (1974) administered the test, 12 weeks apart, and reported reliabilities of .46 and .44 (Part A and Part B respectively) for young normal controls and .78 and .67 respectively for older patients with cerebrovascular disease.

The TMT is derived from the Army Individual Test Battery (Adjutant General's Office, 1944). Analysis of its performance requirements suggests that it is a multifactorial measure requiring visuospatial scanning, sequential processing, motor speed, mental flexibility and sustained concentration. In view of this complexity a general sensitivity to brain damage can be expected, however, specificity can be expected to be quite low. Golden, Osmon, Moses and Berg (1981) consider Part A as primarily a nondominant hemisphere measure and Part B as primarily a dominant hemisphere measure. Consistent with this, Lezack (1983 p. 558) cites evidence suggesting poor lateralizing power. Reitan and Wolfson (1985) have suggested that different aspects of the task reflect separate contributions of each hemisphere, i.e., manipulation of alphanumeric symbols is viewed as a left hemisphere task, while the scanning and visual search of the test stimuli represent a nondominant hemisphere task. It would appear that Part B requires more information processing than Part A and large differences between Part A and Part B have been interpreted as indicative of left lateralized lesions (Lewinshon, 1973; Wheeler & Reitan, 1963), however, more recent studies have not confirmed this (Schreiber, Goldman, Kleinman, Goldfader & Snow, 1976; Wedding, 1979).

Though apparently of limited lateralizing potential, the TMT has been found to be a particularly sensitive measure of brain damage (Dodrill, 1978b; Mutchnick, Ross & Long, 1991; O'Donnell, 1983). This has been noted in several studies, e.g., including closed head injury patients (desRosiers & Kavanagh, 1987) and alcoholism (Grant, Adams & Reed, 1984; Grant, Reed & Adams, 1987). It has been shown to differentiate adjustment disorders from more serious psychiatric illnesses in a psychiatric emergency room setting (Galynker & Harvey, 1992) and to distinguish depressive disorder subtypes (Austin, Ross, Murray, O'Carrol et al., 1992). Finally, Jarvis and Barth (1994, p. 46) consider the TMT to provide evidence of prefrontal deficits where nonfrontal deficit is not evident and other prefrontal measures suggest deficit.

In sum, the TMT has been shown to be a reliable and sensitive measure of brain dysfunction and though multifactorial in nature, it is likely differentially sensitive to prefrontal function where other brain regions are not compromised.

Tactual Performance Test (TPT)

The TPT uses Halstead's (1947) modification of the Sequín-Goddard Board. It is a formboard with 10 geometrically cut-out spaces and wooden blocks designed to fit into the spaces. During administration the subject is blindfolded prior to seeing the board and places the blocks onto the formboard 3 times, first with the preferred hand, then with the non-preferred hand and lastly, with both hands. Time for each trial is recorded and after

the third trial the subject is requested to draw, from memory, as many blocks as they can remember in their correct spatial location. Six measures are used from this procedure: a) preferred hand time, b) non-preferred hand time, c) both hands time, d) total time to place the blocks on the board (Time), e) number of shapes correctly recalled (Memory), and f) number of correctly recalled shapes that also are correctly located on the subject's reproduction of the formboard (Location).

The TPT appears to be multifactorial in that it incorporates motor functioning, tactile form discrimination, problem-solving, spatial reasoning and memory skills.

Psychometric Properties

Charter et al. (1987) reported odd-even reliabilities for age- and education-corrected scores in 123 young adults as: a) .60 - .78 for blocks per minute, b) .77 - .93 for Times, c) .64 for Memory, and d) .69 for Location. Similar values were also reported for a combined sample of normal and brain damaged subjects. Goldstein and Watson (1989) reported retest reliabilities after intervals of 4 - 469 weeks for 150 neuropsychiatric patients ranging from .66 to .74 for time, .46 to .73 for memory and .32 to .69 for location.

Research

Dodrill (1978a) and Reitan (1959, 1964) have reported that right-hand times were significantly lower for left-hemisphere-damaged patients than for right-hemisphere-damaged patients and left-hand time was significantly

slower for right-hemisphere-damaged patients. In the Dodrill study, right-left differences were found to correctly classify 76% of the lateralized cases. Reitan (1964) also noted that subjects with right-hemisphere lesions performed significantly slower with the left hand than did subjects with left-cerebral lesions. These studies and Schreiber, Goldman, Kleinman, Goldfader and Snow (1976) also found that patients with right-hemisphere damage perform slower than patients with left-hemisphere damage.

Wheeler, Burke, and Reitan (1963) found that TPT total time was highly ranked in a discriminant analysis comparing right-vs-left-hemisphere patients, however, Goldstein and Shelly (1972) reported no significant differences in Total Time for their groups of right- and left-hemisphere-damaged patients.

Simple discrimination of brain damage groups with the TPT, e.g., Mutchnick, Murray, Ross and Long (1991) has typically been successful with the Total Time measure, however, results with the Location and Memory scores have often been equivocal (Thompson & Parsons, 1985). The TPT has also been shown to be sensitive to the effects of cerebral dysfunction in a number of disorders: multiple sclerosis, e.g., Ivnik (1978); chronic obstructive pulmonary disease, e.g., Prigatano, Parsons, Wright, Levin and Hawryluk (1983); Parkinson's Disease, e.g., Reitan and Boll (1971); Huntington's Chorea, e.g., Boll, Heaton and Reitan (1974), and alcoholics, but not drug abusers (Parsons & Farr, 1981).

Seashore Rhythm Test (SRT)

The SRT is a subtest of the Seashore Tests of Musical Talent (Seashore, 1919; Seashore, Lewis & Saetveit 1960) and was incorporated in Halstead's (1947) original battery. The task requires that subjects distinguish between 30 pairs of rhythmic beats as being the same or different. The presentation of items is standardized on an audio tape and the test is scored for number of errors. Task analysis suggests that this measure of nonverbal auditory perception involves immediate auditory memory, rhythm discrimination and sustained attention (Yeudall et al., 1987).

Psychometric Properties

Goldstein and Watson (1989) report a reliability of between .50 and .68 for 2 groups of neurological patients and a group of schizophrenic patients. Bornstein (1983) reported a Cronbach's alpha of .78 for 376 patients referred for neuropsychological examination.

Research

Some authors have suggested that the SRT may be differentially sensitive to right temporal lesions (Golden et al., 1981; Lezack, 1983; Long & Hunter, 1981). However, the evidence is that while the test is sensitive to brain damage, it has not been shown to be discriminate right-lesioned patients from left-lesioned patients (Boone & Rausch, 1989; Karzmark, Heaton, Lehman, & Crouch, 1985; Mutchnick et al., 1991; Reitan & Wolfson, 1989; Sherer, Parsons, Nixon, Adams, & Russell, 1991). Young and Delay (1993) found high correlations between Signal Detection

procedures and scores on the SRT providing a measure of concordant validity. Spreen and Strauss (1991) suggest that the SRT, while sensitive to brain damage, fails to provide much by way of unique variance (p. 276).

Seashore Speech Sounds Perception Test (SSPT)

The SSPT was included in Halstead's (1947) original battery. The test is composed of 60 nonsense syllables that involve the digraph ee in the middle portion of each syllable with different beginning and/or ending consonants. The items are presented in six series of 10 and are standardized on audio tape.

Yeudall et al. (1987) analysed the task as requiring the ability to discriminate phonems and match them with graphems. Some reading ability is implicitly required, as well as the ability to sustain attention.

Psychometric Properties

Goldstein and Watson (1989) report reliabilities of .80 and .88 for two neurological groups. Bornstein (1982) reported splithalf reliabilities of .74 and .87 in two samples of neuropsychological patients.

Research

The SSPT, though apparently more reliable and more sensitive to the presence of brain damage (Alekoumbides et al. 1987; Mutchnick et al., 1991; Sherer et al., 1991) than the SRT, it does not reliably discriminate neurological patients with left-vs-right-hemisphere lesions (Reitan, 1990). Further to an analysis by Bolter, Hutcherson and Long (1984), Reddon, Schopflocher, Gill and Stefanyk (1989) identified some bias due to the

format of the recording form and shortforms have been demonstrated to be highly correlated with the original version (Ryan & Larsen, 1983).

Strength Of Grip Test (SOGT)

A measure of strength of grip (Lezack, 1983) obtained with the Smedley hand dynamometer has been added to the Halstead-Reitan Neuropsychological Test Battery by Reitan and others (Reitan & Davison, 1974). The instrument is adjusted to accommodate the size of the client's hand and the subject is instructed to stand, holding the instrument at his/her side, pointed toward the floor with the arm held straight at the elbow. The client is then instructed to squeeze the dynamometer as hard as he/she can. The score is an average of two consecutive trials within 5 kg. for each hand. The test, providing there are no significant peripheral factors, is thought to reflect the integrity of the motor strip (Swiercinsky, 1978).

Psychometric Properties

The SOGT has been shown to be quite stable for each hand even with intervals of up to 30 months between trials. Reliability coefficients ranging from .52 to .96 have been reported for both normal and neurological samples (Dodrill, 1978a; Matarazzo et al., 1974; Provins & Cunliffe, 1972; Reddon, Stefanyk, Gill & Renney, 1985). Intermanual differences have not been shown to be reliable (Provins & Cunliffe, 1972; Sappington, 1980).

The SOGT has been successfully used to determine speech laterality (Strauss & Wada, 1988) in differentiating brain damaged from normal sample, and in detecting laterality of brain lesion (Bornstein, 1986c; Dodrill,

1978a; Finlayson & Reitan, 1980; Hom & Reitan, 1982; York Haaland & Delayney, 1981). It has been assumed that right-handed people should perform better on the dynamometer with their right hand (Reitan & Wolfson, 1985), but there is considerable variability in the normal population (Koffler & Zehler, 1985; Lewandowski, Kobus, Church & Van Orden, 1982).

Finger-Tip Number Writing Perception Test (FTNWPT)

The FTNWPT is a test of graphesthesia, forming part of the Reitan-Klove Sensory-Perceptual Examination (Reitan, 1969). The task requires that the subjects recognize numbers written on their finger-tips in the absence of visual cues. Neuropsychologically, it is primarily a parietal lobe measure (Swiercinsky, 1978).

Normative data are available for this measure, however, no reliability studies are available. Havey (1990) found that the FTNWPT discriminated children (9-14) who were reading disabled from other learning disabled children. Similarly, Boll (1978) reported a strong relationship between this measure and academic performance in both brain-impaired and normal children aged 9-14 years.

Tactile Form Recognition Test (TFRT)

The TFRT was developed by Reitan and Klove (Reitan, 1969) as a measure of astereognosis. The task requires subjects to identify simple geometric shapes in the absence of visual cues (Yeudall et al., 1987). Test administration has been modified by Yeudall (1983) so that the preferred hand is tested first and only one trial per hand is given unless an error is

committed. Unlike the L. J. Tactile Form Recognition Test (LJTFRT) described below, the shapes in the TFRT cannot be verbally coded thus reducing the potential for verbal compensation.

No reliability indices are available on this measure although norms have been published (Yeudall, Fromm, Reddon, & Stefanyk, 1986). Larrabee, McKeever, Ferguson and Rayport (1980) demonstrated tactile anomia in a patient who had undergone callosotomy. Boll, Richards and Berent (1978) found a strong relationship between academic performance and tactile perceptual skills in both a normal and a brain damaged group of children aged 9-14. Similarly, Yamamoto (1982) found significant differences between normal and learning disabled children aged 9-15 years of age.

Finger Localization Test (FLT)

Yeudall (1983) modified the administration of the FLT from the procedures of Reitan's finger Agnosia Test (Reitan, 1969) and Benton's Finger Localization tests (Benton, 1955). In the current administration this sensory-perceptual measure requires subjects to identify, without visual cues, fingers touched by the examiner. Each of the 11 subtests begins with the preferred hand, both verbal and nonverbal responses are elicited on trials of both single and double sequential stimulation.

Reliability data is not available for this measure, although Yeudall, Fromm, Reddon, and Stefanyk (1986) have published norms. Both diffuse brain injury and dominant parietal lesions have been associated with difficulty on this task (Strub & Black, 1985). Finger localization in

kindergarten children has consistently been shown to be predictive of later reading abilities (e.g., Lindgren, 1978; Badian, McAnulty, Duffy & Als, 1990; Zung, 1986)

Face Hand Test (FHT)

The FHT is a measure of tactile inattention. Utilizing the method of double stimulation, subjects are required to identify, with eyes closed, which hand and which cheek have been lightly touched by the examiner (Green & Fink, 1954). When errors are made on this test, further testing for consistency of suppression is carried out. The test is viewed primarily as a measure of parietal lobe integrity (Reitan & Wolfson, 1985; Swiercinsky, 1978).

Norms are available (Yeudall et al., 1987), but reliabilities have not been reported. The task has been reported to be highly discriminating of organic versus psychiatric disorders (Patten & Lamarre, 1989). Performance on the test has been related to diffuse brain damage (Lishman, 1978) and has been found to be impaired in patients with right hemisphere lesion with neglect (Feinberg, Haber, Lawrence & Stacy, 1990) and to discriminate patients with organic disorders from patients with affective disorders (Bulbena & Berrios, 1993).

Purdue Pegboard (PPB)

The Purdue Pegboard was developed as a measure of manipulative dexterity in the 1940's by the Purdue Research Foundation. Administration of the test requires subjects to place metal pins in a wooden board within

which holes have been bored. Each 30 second trial session with the dominant, nondominant, and both hands is preceded by a short practice session. A fourth trial requires subjects to construct assemblies consisting of a pin, washer, collar, and a second washer. This portion lasts 60 seconds and speed is emphasized throughout the test.

The PPB task is seen to require fine motor skills and responsiveness to visual and tactile feedback (Bourassa & Guion, 1959; Fleishman & Ellison, 1962; Fleishman & Hempel, 1954).

Psychometric Properties

Tiffin and Asher (1948) have reported single trial test-retest reliability coefficients of .63 to .68 in a sample of college students. Tiffin's (1968) manual reports a range of single trial test-retest correlation between .60 and .76. In a recent reliability study, Reddon, Gill, Gauk, and Maerz (1988) reported on the results of five repeated administrations (once per week over a five week period). They reported mean test-retest reliabilities between .63 and .67 on the left and right hand among males. For both hands and the assemblies measure, reliabilities of .81 were reported for males.

Research

Costa (1969) and Costa, Vaughan, Levita, and Farber (1963) reported high discrimination between brain damaged and non-brain damaged subjects with this instrument. Significant lateralization rates were also reported. Similar general discriminating (80% correct classification) potential was also

reported by Vega (1969) on a small sample of brain-damaged patients and controls.

Symbol Digit Modalities Test (SDMT)

The SDMT (Smith, 1968, 1973) is a written and oral coding task where subjects are provided with a symbol/number key including 9 symbols with the numbers 1 to 9 present and immediately below. Subjects (in the written portion) code the number corresponding to the symbol in a square box below each symbol. In the oral portion, subjects are required to voice the corresponding number. The score on this measure is the number of correct codings produced in 90 seconds on both the written and oral portions. The written portion is similar to the Symbol Digit subtest of the WAIS-R (Wechsler, 1981) except that in the former the number corresponding to the symbol is recorded while in the latter the symbol is recorded.

Research

A correlation between the SDMT and Digit Symbol subtest of the WAIS of .91 was reported by Morgan and Wheelock (1992) among subjects referred for neuropsychological evaluation.

Symbol-Gestalt Test

This test (Stein, 1962, 1970) involves the reproduction of unfamiliar asymmetrical symbols, each of which is associated with the numbers 1 to 9. Task facets involve visual scanning and visuo-motor coordination, as well as visual associative memory. Royce et al. (1976) included this measure in their study, otherwise, little specific research has been reported, although

norms have been provided by Yeudall, Fromm, Reddon, and Stefanyk (1986).

Colored Progressive Matrices (CPM)

This test was developed as a "culture fair" test of general intellectual ability for children aged 5-11 (Raven, 1949; Raven, 1965; Raven, Court, & Raven, 1977). The test consists of 3 subtests of 12 items each. Each item consists of a geometric design with a portion absent. Apart from the design, choices are presented, one of which would correctly complete the design. The task is to select the "puzzle piece" necessary to complete the design. There is an increase in complexity across the three subtests. The task requires visuospatial analysis and sustained attention (Costa, 1976; Lezack, 1983).

Psychometric Properties

Carlson and Jensen (1981) report test-retest reliability coefficients ranging from .81 to .87 in a sample of children ranging in age from 5½ to 8½ years of age. A split-half reliability coefficient of .90 was reported by Freyberg (1966) on a large sample of 6 and 7 year old children.

Research

In neuropsychological research, the CPM has been found to be an effective discriminator of brain damaged from normal controls (Dils, 1960; Evans & Marmorston, 1964). The test is believed to be particularly sensitive to post-Rolandic lesions (Costa, 1976; Denes, Semenza, Stoppa, & Gradenigo, 1978; Gainotti, Caltagirone, & Miceli, 1977). Villa, Gainotti, de-

Bonis, and Marra (1990) found the CPM to be particularly sensitive to parietal lesions, while Drebing et al. (1990) found that in contrasting left and right hemisphere brain damaged patients, the two groups could not be significantly differentiated after partialling out the effects of hemineglect.

Williams' Clinical Memory

Two parts of this battery are employed in this study: Verbal Learning and Non-Verbal Learning (Williams, 1968).

The Verbal Learning subtest is essentially a paired associate learning task. The test requires a subject to learn short definitions (1-2 words) of eight unfamiliar words. First, the subject is shown the unfamiliar words to ensure that the meanings are actually not known, then the subject is given the meaning by the examiner. On test trials, after each word is presented orally, the subject is asked to provide the meaning. Errors are corrected by providing the correct response on each trial. The test continues until the subject has completed a trial (8 words) without errors or five trials have been administered.

The Non-Verbal subtest involves a display of 4 wooden blocks, 3 inches square, which are located in a larger board one foot square. Each of the 4 individual wooden blocks contains 9 plastic pegs, for each block 1 of the 9 pegs is fixed while the remainder can be pulled out. The first part of the task is for the subject to identify the fixed peg in each of the four blocks, then on the test trial, the subject is asked to identify the position of the fixed peg in each of the four blocks. Where a mistake is made, the fixed peg is

located by the subject and then the task proceeds to the next block. The test is discontinued either after all four fixed pegs are identified on a trial or five trials are completed. A second part of the test, either after a correct trial or 5 attempts, the four blocks are rotated 90° clockwise in view of the subject. As in the first part of this test, the subject must again identify the fixed peg in each of the four blocks. This part of the test continues until all fixed pegs are located in a single trial or 5 trials are administered.

Williams (1968) reports on many studies utilizing his memory scales that show clear discriminations between normal control and various pathological populations, however, psychometric properties, e.g., reliabilities, are generally not reported.

Memory For Designs (MFD)

The MFD involves the presentation of simple geometric designs and the reproduction of these designs from immediate memory (Graham & Kendall, 1960). These authors report that the MFD significantly differentiates brain-disordered patients from those without brain disorder, but that a good performance on this test does not indicate an intact brain, i.e., it is a highly specific measure of short-term visual memory. Graham and Kendall (1960) report test-immediate re-test reliability coefficients of .85 in normal adults and .88 in brain-disordered patients.

Language Modalities Test For Aphasia

The Language Modalities Test For Aphasia (Wepman & Jones, 1961) measures elementary language skills and has been found to be primarily

useful for localizing focal dysfunction associated with dominant hemisphere deficits. It has many items in common with the Reitan Aphasia screening test (Reitan & Wolfson, 1993). Normative data are available (Yeudall et al., 1987), but reliability studies do not appear to have been conducted given the pathognomonic nature of the items.

Minute Estimation

This task requires the subject to indicate when a minute has elapsed. The subject, where necessary, is requested not to count, to visualize a clock face or count heartbeats. The score is the average of 3 valid trials (Benton, Van Allen, & Fogel, 1964). Reliability data do not appear to have been published.

L.J. Tactile Form Recognition Test (LJTFRT)

The LJTFRT (Yeudall, 1983) was designed as a nonverbal form of the Tactile Form Recognition Test which forms part of the Halstead-Reitan Neuropsychological Test Battery. The test consists in subjects being given four (one at a time) asymmetrically shaped plastic wafers to feel and then visually identify an identical shape from an array of four shapes. The times taken to point to the appropriate shapes in the array and the number of errors for the preferred, nonpreferred, and both hands are recorded. Few errors are made by neurologically intact subjects on this test. It was suggested (Yeudall, Fromm, Reddon, & Stafanyk, 1986) that performance on this task would relate to right parietal functions. No reliabilities have been

reported for this measure, but Yeudall, Fromm, Reddon, and Stefanyk (1986) have provided normative data.

Annett Measures

The Annett Handedness Questionnaire (Annett, 1970) includes the following twelve questions. Responses options for each item include left hand, right hand, or both hands. The questions are -

Which hand do you use to:

1. To write a letter legibly?
2. To throw a ball to hit a target?
3. To hold a racket in tennis, squash or badminton?
4. To hold a match whilst striking it?
5. To cut with scissors?
6. To guide a thread through the eye of a needle (or guide needle on to a thread)?
7. At the top of a broom while sweeping?
8. At the top of a shovel when moving sand?
9. To deal playing cards?
10. To hammer a nail into wood?
11. To hold a toothbrush while cleaning your teeth?
12. To unscrew the lid of a jar?

The hand preferred for writing is taken as a measure of 'preferred hand' where required for the administration of other neuropsychological tasks. The sum of items for which the right hand or both hands is used is calculated

(ANNETT), as well as the sum for the right, left, and both hands, ANN-R, ANN-L, ANN-B respectively.

The Wechsler Scales

Among the array of available intelligence tests, the family of Wechsler scales is considered among the most eminent (Hill, Reddon, & Jackson, 1985) and has been singularly preferred by neuropsychologists (Lezack, 1983). The history of this family of tests began with the Wechsler-Bellevue Forms I and II (Wechsler, 1939, 1941, 1944) and for the adult forms to the Wechsler Adult Intelligence Scale and its revision (WAIS - Wechsler, 1955; WAIS-R - Wechsler, 1981).

The Wechsler scales have been extensively reviewed by Anastasi (1968, 1988), Matarazzo (1972), Lezack (1983), and Kaufman (1990) has provided a comprehensive review of intelligence testing, including the Wechsler measures.

The data sets in this study include both WAIS and WAIS-R measures, however, WAIS scores were converted by linear equating to WAIS-R scores prior to further statistical analyses.

The WAIS-R includes 11 subtests divided into two sections: Verbal subtests and Performance subtests. The Verbal subtests are composed of the following tests: Information, Comprehension, Arithmetic, Similarities, Digit Span, and Vocabulary. The Performance subtests include Picture Completion, Picture Arrangement, Block Design, Object Assembly, and Digit

Symbol. For a detailed description of the subtests the reader is referred to the references cited above or the test manual (Wechsler, 1981).

Psychometric Properties

Wechsler (1981), based on the WAIS-R normal standardization sample of 1,880 cases representing all age groups included (16-74), reports split half coefficients for Verbal IQ, Performance IQ, and Full Scale IQ of .97, .93, and .97 respectively. Among the Verbal subtests excepting Digit Span average split half coefficients range from .84 to .96, and among the Performance subtests excepting Digit Symbol coefficients range from a low of .68 for Object Assembly to a high of .87 for Block Design. Reliability coefficients on two age groups (25-34 and 45-54), with first and second testing between 2 and 5 weeks for the first group and 2 and 7 weeks for the second group ranged between .89 and .97. Among the subtests retest coefficients ranged from a low of .67 for Object Assembly to a high of .93 for Vocabulary. The majority of coefficients were between .82 and .93 suggesting overall that the WAIS-R is a highly reliable instrument.

Independent test-retest replication in a clinical sample of brain-damaged and psychiatric patients (Ryan, Georgemiller, Geisser, & Randall, 1985) retested at intervals between 2 and 144 weeks confirmed high reliabilities of .79, .88, and .86 for Verbal IQ, Performance IQ, and Full Scale IQ respectively. Thompson and Molly (1993) also confirmed high test-retest reliabilities among 16 year old students tested at either 3 or 8 month intervals. They also noted, by way of caution, that in the 8 month retest

group, IQ's increased significantly above expectations due simply to practice effects, thus implicating educational and maturational effects between 16 and 17 years of age. The effect was particularly notable among adolescent boys. In sum, however, the WAIS-R appears to be a highly reliable measure.

Research

The Wechsler scales have been extensively researched across numerous clinical groups, including psychiatric and neurological patients. In particular, a large number of studies have examined the factor structure of the WAIS-R and this continues to be a matter of some debate. Hill et al. (1985) reviewed over 65 factor analytic studies of the Wechsler scales conducted between 1941 and 1984. They concluded, upon a conjoint review of a number of factor extraction "rules" that a one or two factor solution to be preferable. They noted that a three factor solution, Verbal, Performance, and Freedom from Distractibility, has been typically reported and seems to be the preferred solution in clinical applications. Subsequent studies Gutkin, Reynolds, & Galvin (1984), Fowler, Zillmer, and Macciocchi (1990), and Canavan and Beckmann (1993) confirm a range of interpretation of between one and three factors, but in all instances, a large 1st principal component "g" is invariably extracted.

The Wechsler scales have been researched extensively in the neuropsychological literature, particularly as a lateralization instrument (e.g., Fitzhugh & Fitzhugh, 1964; Russell, 1979; Warrington, James, Maciejewski, 1986), but with only moderate success (e.g., Reitan & Wolfson, 1993).

Reitan and Wolfson (1993) attribute this result in part to conceptual differences in the development of intelligence scales versus neuropsychological batteries. They note the former are more concerned with the measurement of general intelligence in normal populations and concerned historically with questions primarily of academic potential and success. In neuropsychology, however, the focus has been on adaptive abilities and a basic orientation toward the biological adequacy of brain functions. In their review of the specific neuropsychological relevance of the Wechsler scales, they conclude that they are relatively "limited in reflecting the range of intellectual and cognitive functions subserved by the brain, even though the subtests are useful in the assessment of brain-behavior relationships when complemented by other neuropsychological tests" (p. 106).

Reitan and Wolfson (1993) concluded that the Wechsler Verbal subscales lack neuropsychological discriminating power because of their low demand on verbal problem-solving abilities and have demonstrated that a verbal task requiring verbal problem-solving (Word Finding) is superior to the Wechsler scales in lateralized dominant hemisphere damage (Reitan, Hom, & Wolfson, 1988). With respect to the Performance subtests they discuss research demonstrating specific relationships between Picture Arrangement and right anterior temporal lesions, Block Design and right parietal lesions, and the consistent sensitivity of the Digit Symbol subtest to cerebral impairment.

As for the general sensitivity of the Wechsler measures to cerebral impairment, they note the superiority of the Category test and the Impairment Index in such discriminations (cf. Reitan, 1959; Reitan & Wolfson, 1993). Lezack (1988) has also analysed the neuropsychological limitations of the Wechsler scales and called for a new generation of "neuropsychological sound" (p. 360) test instruments.

Wechsler scale variability has been a subject of considerable interest and has been researched from a number of perspectives, including its normal/abnormal and neurodiagnostic implications (e.g., Matarazzo, Daniel, Prifitera, & Herman, 1988; Matarazzo & Herman, 1984; McLean, Kaufman, & Reynolds, 1989; Ryan, Paolo, & Van Fleet, 1994). In forensic applications distinctions between indices of scatter as reflecting "statistical significance" versus "abnormality" have been controversial (Reed, 1988). In the area of learning disabilities Wechsler pattern profile analysis has been extensively examined (e.g., Rourke, 1991).

In this research several additional WAIS-R measures and indices of scatter were computed. These include:

1. Verbal IQ - Performance IQ (VPDIF)
2. Minimum of all scale scores (MINF)
3. Maximum of all scale scores (MAXF)
4. Average range of all scale scores (RANF)
5. Minimum of all Verbal scale scores (MINV)
6. Maximum of all Verbal scale scores (MAXV)

7. Average range of all Verbal scale scores (RANV)
8. Minimum of all Performance scale scores (MINP)
9. Maximum of all Performance scale scores (MAXP)
10. Average range of all Performance scale scores (RANP)

## SECTION IV METHODOLOGY/HYPOTHESES/ANALYSES

The rationale for this study is primarily exploratory and descriptive. The main purposes are: a) to examine the factorial structure of neuropsychological variables in three groups of subjects (NC, OF, and PP); b) to examine whether subsets of subjects share common patterns of organization of neuropsychological abilities and whether particular patterns are differentially associated with clinical group status; and c) to contrast the level of performance of the three groups on neuropsychological variables and pattern of organization subgroupings across the NC, OF, and PP groups.

Realistically, previous research does not provide a framework for developing and evaluating specific hypothesis apriori with respect to these broad objectives, nevertheless, each analysis rests upon implicit assumptions and hypotheses, e.g., factor analysis assumes that the rank of a data matrix can be reduced and the number of factors problem can be formulated in hypothesis testing terms.

The literature review on the neuropsychological abilities of offenders, however, does suggest two primary questions:

1. Are offenders characterized by deficits in verbal abilities thought to be primarily mediated by dominant hemisphere structures and do they exhibit executive function deficits?

2. What is the degree of impairment in these two general areas?

The present study addresses these questions, but goes beyond to examine a broader span of neuropsychological functioning.

Analyses undertaken to evaluate these hypotheses, to identify both qualitative and quantitative differences between the NC, OF, and PP groups are delimited and described in detail in Chapter IV. Thus, at this juncture, only the general logic of the approach taken toward the analysis of the data is summarized as follows

1. The validity of the control norms employed in this as an appropriate contrast group will be evaluated,

2. Analyses will be conducted to evaluate age effects that may contaminate interpretation and the need to stratify groups by age will be determined.

3. Descriptive normative statistics on all measures for the OF group stratified by age will be provided.

4. Employing analysis of variance methods, contrasts between the three data groups will be compared.

5. A subset of neuropsychological variables for which data is available or can be estimated on each subject and each group will be factor analysed. These analyses will be conducted separately for each group and the

feasibility of employing a combined factor solution for all three groups will be evaluated.

6. Modal Profile Analyses based on the neuropsychological factor analytic solution will be conducted separately and a combined modal profile analysis will be carried out for both the Wechsler intelligence and neuropsychological variables separately.

7. With reference to the combined modal profiles, impairment levels for each of the subjects (NC, OF, and PP) will be evaluated with reference to the original test variable performance of subjects classified in each modal profile to evaluate relative levels of impairment by modal profile for each of the groups with particular reference to the OF group. This series of analyses further described, presented, and discussed in Chapter IV will form the basis for evaluating the empirical objectives of this research.

Ethical Considerations

The methodology employed in this proposed research involves a retrospective analysis of the neuropsychological test results of incarcerated offenders who participated in a multidisciplinary assessment. Clearly, it would have been most appropriate to request permission of these clients to make use of their results for group research purposes. Regrettably, this was not done and it is thus important to establish that this study does not constitute a significant invasion of their privacy or violation of their right to informed consent.

The issue of informed consent is seemingly never fully satisfied with incarcerated clients, however, anonymity and confidentiality of results has been fully respected in this study. Kazdin (1980) has stated that invasion of privacy pertains to how information is obtained and how it is used. He specifically noted that "there are many kinds of research where consent of the individual is neither possible nor especially crucial, as in the case of studying archival records for groups of subjects" (p. 394).

Acknowledging that it would have been ideal to have requested permission of each participant in this study, anonymity and confidentiality have been safeguarded throughout this study. The writer was the chief clinician under which the research data was collected. The potential of this research to provide a large amount of information regarding the neuropsychological status of a large group of offender assumes importance in view of the equivocal nature of previous research in this area. Accordingingly, the writer feels obligated to conduct this study, given that anonymity and confidentiality have been safeguarded throughout.

Next, in Chapter IV we consider the results and discussion of the methodology proposed in this section.

# CHAPTER IV RESULTS

## SECTION I INTRODUCTION

The primary purpose of this chapter is to report the results of statistical analyses conducted on the neuropsychological characteristics of the three groups represented in this research; normal controls (NC), offenders (OF), and psychiatric patients (PP), and group contrasts. A further objective is to develop a neuropsychological classification scheme common to all three groups. The chapter is divided into nine sections, each section is designed to contribute to the empirical objectives of this research.

In Section II, data preparation procedures are described and a listing of all variables and associated acronyms are provided. Section III examines the validity and general adequacy of the normal control norms employed in this study. This is a critical step since the normative performance of the NC group forms the basis for comparisons with the OF and PP groups. In Section IV, age effects on neuropsychological performance are evaluated within the full age range (18-44) represented in this study across all three samples.

Section V presents separate norms for two age groupings of the OF group; 18 to 29 and 30 to 44 years of age, and the combined sample. Age differences in performance between the two age groupings are also discussed.

Section VI is concerned with contrasting the performance of the NC, OF, and PP groups at the level of performance on individual measures. The

performance of the OF group on selected tests is discussed with reference to that of other clinical groups reported in the literature.

Section VII reports on a factor analysis of a representative subset of neuropsychological measures. Separate analyses were conducted for each sample, congruence coefficients between solutions were computed and found to justify a combined factor analysis. This combined solution is reported, including extension loadings of neuropsychological and Wechsler variables, not included in the factor analysis. The resulting five factor solution is then interpreted and discussed.

Section VIII reports on the separate Modal Profile Analysis of the neuropsychological factors and Wechsler subtests. Profile interpretation, classification rates by group sample, and impairment analysis by profile is provided. Lastly, the intersection of neuropsychological and Wechsler profiles is discussed and illustrated.

Section IX provides an overall summary of major findings of the analyses conducted in this chapter.

## SECTION II DATA PREPARATION

Testing procedures, manual test data recording, and subsequent transcription of data onto PC floppy disk for the OF group was reported in Chapter III, Section II. Data was recorded only for Caucasian males who were not psychotic or diagnosed to have organic brain damage, and represented consecutive admissions to the Regional Psychiatric Centre between September, 1978 and July, 1986. This initial data base of 615

cases was further reduced to 584 cases by excluding offenders who were less than 18 years of age or more than 44 years of age.

Table 4.1 lists the variable labels and acronyms comprising the full set of variables for which data was collected in the OF group. There was some missing data on some variables mainly because some tests were eliminated from the battery over the course of the data collection period. The main rationale for eliminating certain tests related to their perceived lack of clinical usefulness, e.g., simple sensory measures, although work volume constraints also contributed. The number of observations for each variable listed in Table 4.1 ranges between 365 and 584 for the OF group. The number of observations on each variable for the NC, OF, and PP groups is listed in Tables 4.6 and 4.7.

The data was subjected to visual and computer programmed validity checks. For instance, each observation was verified to be within its allowable range. If a variable score was found to be out of range, it was reclassified as a missing data point. Overall, the data collection and transcription process appears to have been very reliable since less than 2% of subjects had any observations that were out of range.

Similar data collection procedures and validity checks were conducted on the data for the NC and PP group samples provided by J. R. Reddon, Ph.D., of the Alberta Hospital. Overall, a high degree of confidence in the integrity of the data appears justified. There are of course, missing data for a substantial number of subjects in each of the groups and the management of

this issue will be addressed as required as part of the description of the analyses and results reported below. Overall, no systematic factors of clinical import are believed to have affected the extent of missing data across samples, i.e., decisions not to test an individual on a particular test was not based on apriori clinical grounds, rather the test battery approach was adhered to across all samples, though the battery itself was not completely uniform over time or samples.

TABLE 4.1

Reference Variable List and Acronym

| | VARIABLE | ACRONYM |
|---|---|---|
| 1 | Age of participant | AGE |
| 1a | Age grouping variable | YAGE |
| 2 | Annett scale score | ANNETT |
| 3 | Annett right preferences index | ANN-R |
| 4 | Annett left preferences index | ANN-L |
| 5 | Annett left & right preference index | ANN-B |
| 6 | WAIS-R Verbal intelligence quotient | VIQ |
| 7 | WAIS-R Performance intelligence quotient | PIQ |
| 8 | WAIS-R VIA-PIQ score | VPDIF |
| 9 | WAIS-R Full Scale intellectual quotient | FIQ |
| 10 | WAIS-R minimum of all scale scores | MINF |
| 11 | WAIS-R maximum of all scale scores | MAXF |
| 12 | WAIS-R average range of all scale scores | RANF |

# A NEUROPSYCHOLOGICAL TYPOLOGY OF ADULT OFFENDERS: A DEVELOPMENTAL/PREVENTATIVE PERSPECTIVE

by

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[ v. 2 ]

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| | VARIABLE | ACRONYM |
|---|---|---|
| 13 | WAIS-R Information scale score | IN |
| 14 | WAIS-R Digit Span scale score | DSP |
| 15 | WAIS-R Vocabulary scale score | VO |
| 16 | WAIS-R Arithmetic scale score | AR |
| 17 | WAIS-R Comprehension scale score | CO |
| 18 | WAIS-R Similarities scale score | SI |
| 19 | WAIS-R minimum of all Verbal scale scores | MINV |
| 20 | WAIS-R maximum of all Verbal scale scores | MAXV |
| 21 | WAIS-R average range of all Verbal scale scores | RANV |
| 22 | WAIS-R Picture Completion scale score | PC |
| 23 | WAIS-R Picture Arrangement scale score | PA |
| 24 | WAIS-R Block Design scale score | BD |
| 25 | WAIS-R Object Assembly scale score | OA |
| 26 | WAIS-R Digit Symbol scale score | DSY |
| 27 | WAIS-R minimum of all Performance scale scores | MINP |
| 28 | WAIS-R maximum of all Performance scale scores | MAXP |
| 29 | WAIS-R average range of all Performance scale scores | RANP |
| 30 | Coloured Progressive Matrices | CPM |
| 31 | Finger Tapping (preferred hand) | FTAPP |
| 32 | Finger Tapping (non-preferred hand) | FTAPNP |
| 33 | Dynamometer (preferred hand) | DYNP |
| 34 | Dynamometer (non-preferred hand) | DYNNP |

| | VARIABLE | ACRONYM |
|---|---|---|
| 35 | Trail Making A | TRAILA |
| 36 | Trail Making B | TRAILB |
| 37 | Purdue Pegboard (preferred hand) | PURDP |
| 38 | Purdue Pegboard (non-preferred hand) | PURDNP |
| 39 | Purdue Pegboard (both hands) | PURDB |
| 40 | Purdue Pegboard (assemblies) | PURDA |
| 41 | Tactual Performance Form Board (preferred hand) | TACPERP |
| 42 | Tactual Performance Form Board (non-preferred hand) | TACPERNP |
| 43 | Tactual Performance Form Board (both hands) | TACPERB |
| 44 | Tactual Performance Form Board (memory) | TACPERFM |
| 45 | Tactual Performance Form Board (location) | TACPERFL |
| 46 | Tactile Form Recognition (right hand time) | TACFRP |
| 47 | Tactile Form Recognition (left hand time) | TACFRNP |
| 48 | L.J. Tactile Recognition (preferred hand errors) | LJPERR |
| 49 | L.J. Tactile Recognition (preferred hand time) | LJPTIM |
| 50 | L.J. Tactile Recognition (non-preferred hand errors) | LJNPERR |
| 51 | L.J. Tactile Recognition (non-preferred hand time) | LJNPTIM |
| 52 | L.J. Tactile Recognition (both hands time) | LJBTIM |
| 53 | L.J. Tactile Recognition (both hands errors) | LJBERR |
| 54 | Finger Tip Number Writing (preferred hand) | FTIPNP |
| 55 | Finger Tip Number Writing (non-preferred hand) | FTIPNNP |
| 56 | Face Hand (right side) | FHANDR |

| | VARIABLE | ACRONYM |
|---|---|---|
| 57 | Face Hand (left side) | FHANDL |
| 58 | Finger Localization (preferred hand single stimulation) | FLOCPS |
| 59 | Finger Localization (preferred hand double stimulation) | FLOCPD |
| 60 | Finger Localization (non-preferred hand single stimulation) | FLOCNPS |
| 61 | Finger Localization (non-preferred hand double stimulation) | FLOCNPD |
| 62 | Symbol Digit Modalities (oral) | SDIGO |
| 63 | Symbol Digit Modalities (written) | SDIGW |
| 64 | Aphasia (errors) | APHAS |
| 65 | Seashore Speech Sounds (errors) | SSERR |
| 66 | Seashore Rythm (errors) | SEASHR |
| 67 | Memory For Designs (number correct) | MFD |
| 68 | Symbol Gestalt (3 minute total) | SGEST1 |
| 69 | Minute Estimation (60 second period) | MINEST |
| 70 | Oral Word Fluency | OWTOT |
| 71 | Written Word Fluency | WWTOT |
| 72 | Williams Verbal Paired Association (trials) | VLNTRI |
| 73 | Williams Verbal Paired Association (total errors) | VLTOTE |
| 74 | Williams Non-Verbal Learning (trials) | NVLNTRI |
| 75 | Williams Non-Verbal Learning (errors) | NVLLE |
| 76 | Williams Non-Verbal Learning (trials rotated) | NVLRNTRI |

| | VARIABLE | ACRONYM |
|---|---|---|
| 77 | Williams Non-Verbal Learning (errors rotated) | NVLRE |
| 78 | Wisconsin Card Sorting Test (subtests) | WCSSUB |
| 79 | Wisconsin Card Sorting Test (perseverative errors) | WCSPER |
| 80 | Wisconsin Card Sorting Test (non-perseverative errors) | WCSNPER |
| 81 | Wisconsin Card Sorting Test (correct) | WCSCOR |
| 82 | Wisconsin Card Sorting Test (unique errors) | WCSUNI |
| 83 | Wisconsin Card Sorting Test (total errors) | WCSTOT |
| 84 | Halstead Category Subtest I (errors) | HCAT1 |
| 85 | Halstead Category Subtest II (errors) | HCAT2 |
| 86 | Halstead Category Subtest III (errors) | HCAT3 |
| 87 | Halstead Category Subtest IV (errors) | HCAT4 |
| 88 | Halstead Category Subtest V (errors) | HCAT5 |
| 89 | Halstead Category Subtest VI (errors) | HCAT6 |
| 90 | Halstead Category Subtest VII (errors) | HCAT7 |
| 91 | Halstead Category Subtest (total errors) | HCATERR |
| 92 | Halstead Category Subtest (correct) | HCATCOR |
| 93 | Halstead Category Subtest (incorrect) | HCATINC |

SECTION III CONTROL NORMS

Adequate normative data derived from normal populations are crucial for both clinical neuropsychological interpretation and research, yet this has been a particularly problematic area in neuropsychology. Previous meta-analytic approaches, though, (e.g., Gaskin, 1989; Steinmeyer, 1986) have not fully succeeded in systematically relating neuropsychological performance in controls to demographic characteristics, especially due to an inability to control for age effects. As well, in many of the studies reviewed by these authors, the "normal" controls were contaminated by conditions which are now known to affect neuropsychological performance, e.g., alcoholism, psychiatric, and other medical conditions. Moreover, normative data for young adults was especially poor, but this situation has improved considerably with publications by Yeudall, Fromm, Reddon, and Stefanyk (1986) and Yeudall et al. (1987) providing normative data on normal controls aged 40 and under, and the inclusion of controls between 20 and 80 years of age in the normative system provided by Heaton et al. (1991).

In the present study, the control norms provided by Yeudall, Fromm, Reddon, and Stefanyk (1986) and Yeudall et al. (1987) form the normative base for comparison with the two clinical groups included in this study, i.e., offenders and psychiatric patients, and cover all variables employed in this study. Yeudall et al. (1987) report on norms for neuropsychological measures included in the Halstead-Reitan Battery. Norms for other

neuropsychological measures included in this study reported in Yeudall, Fromm, Reddon, and Stefanyk (1986) were based on the same group of participants. Thus, to the extent that confidence can be established in the norms provided on Halstead-Reitan tests, then this confidence reasonably generalizes to the measures reported in Yeudall, Fromm, Reddon, and Stefanyk (1986) for the non-Halstead-Reitan measures included in this study. The Heaton et al. (1991) normative system provides an opportunity to compare these two normative derivations and the results of this comparison on Halstead-Reitan measures common to both normative systems and these are reported in Table 4.2 below.

TABLE 4.2

* T Scores of Yeudall Control Norms

| Halstead-Reitan Test Measure | MEAN | Raw Score** | Scale Score | T Score |
|---|---|---|---|---|
| Halstead Category | 32.93 | 33 | 10 | 45 |
| Trails A | 24.03 | 24 | 11 | 49 |
| Trails B | 53.58 | 54 | 11 | 50 |
| Tactual Performance (Memory) | 8.31 | 8 | 11 | 47 |
| Tactual Performance (Localization) | 5.38 | 5 | 11 | 48 |
| Seashore Rhythm | 27.33 | 27 | 11 | 48 |
| Speech Sounds | 3.86 | 4 | 11 | 49 |
| Finger Tapping DH | 51.99 | n/a | 11 | 47 |

| Halstead-Reitan Test Measure | MEAN | Raw Score** | Scale Score | T Score |
|---|---|---|---|---|
| Finger Tapping NDH | 47.64 | n/a | 11 | 46 |
| Hand Dynamometer DH | 52.92 | n/a | 12 | 49 |
| Hand Dynamometer NDH | 49.48 | n/a | 12 | 49 |
| | | | | Mean = 47.9 |

* As determined by tables provided by Heaton et al. (1991) for males ages 20-34 years of age with 12 years of education.

** Rounded to the nearest whole number where applicable.

Heaton et al.'s (1991) normative system provides age, gender, and education corrected T scores for normals ranging between 20 to 80 years of age. To obtain an individual's T score on a given test, a raw score is compared to a table of scale scores. As a second step the scale score is then referenced to the appropriate age, gender, and education table which provides a corresponding T score reflecting deviation from normal performance on the particular test examined. Table 4.2 above was derived through this procedure for the eleven Halstead-Reitan measures included in the table employing group mean scores as initial test data scores. Examination of the table indicates that the means on each measure for the two normative systems within the age group represented here are remarkably consistent. Indeed, the means of the normative data included in this study place between a minimum T score of 45 (Halstead Category Test) and a maximum T score of 50 (Trails B). The average T score across all tests compared is 47.9 suggesting that the Yeudall, Fromm, Reddon, and

Stefanyk (1986) norms are consistently comparable to Heaton et al.'s (1991) norms.

Other published studies reporting on the test performance of normals appear to be generally consistent with the Heaton and Yeudall results. Using the Halstead Category Test (HCT) as a general reference, it is noted that Gaskin (1989) identified 38 studies of normals comprising 1,824 subjects in total. These normal subjects representing subjects from all ages had a global mean of 36.73 errors on the HCT (*SD* 19.48). Gaskin (1989) identified 7 studies, excluding studies reported upon by Heaton or Yeudall where the mean age was comparable ($\leq$33 years) among the normal subjects. In only one of these studies (Mack & Carlson, 1978) did the mean error rate among normals exceed the means for normals reported by Heaton and Yeudall above. In this case the subjects had a mean age of 25 years (31 females, 9 males) and were recruited from hospital staff and the university body. There was no screening for medical condition, and while the error rate was considerably higher than for all other reported studies of younger normals (49 errors), the exceptionally large *SD* of nearly 28 suggests that some of these subjects must have had highly impaired scores. Consider that in the Matarazzo et al. (1974) study which included patrolman applicants (mean age 24) the mean HCT error rate was 22.83 (*SD* 19.15), further, these authors reported that only one of their 29 subjects had an error score exceeding 45 errors. Thus, the results of Mack and Carlson (1978) do not

appear to be consistent with most studies which screened their subjects for contaminating conditions. Indeed, several studies, e.g., O'Donnell, Kurtz, and Ramanaiah (1983), and Skenazy and Bigler (1984) report mean error rates of $<25$ among their control samples. Overall, the means provided by Yeudall and Heaton appear reasonable in contrast to other studies which have reported on the HCT performance of screened normal controls.

The normative data utilized in this study is nearly identical to the data reported by Yeudall, Fromm, Reddon, and Stefanyk (1986) and Yeudall et al. (1987) except for slight modification by J.R. Reddon, Ph.D. to exclude subjects less than 18 years of age and add subjects in Alberta Hospital data base between the ages of 41-44.

Since the main focus of the present research is on offenders aged 18-44, the availability of normal control data for this age range is critical since age effects have been consistently reported to be associated with neuropsychological performance, especially after age 45. It is nevertheless important to examine the possible effects of age within the present samples.

SECTION IV AGE AND EDUCATION EFFECTS ACROSS SAMPLES

It has been clearly established that advancing age generally affects neuropsychological performance among normals. For instance, Reitan (1955) demonstrated that after age 45 there is a progressive decline on the measures of the Halstead-Reitan Battery and these findings utilizing the Halstead Impairment Index were replicated by Prigatano and Parsons (1976).

Bak and Greene (1980) contrasted two groups of normals aged 50 to 62 years of age with a group of 67 to 86 year olds and observed that age accounted for a mean of 14% of the variance among the younger group, and 33% among the older group on the Halstead-Reitan measures included in their study.

Heaton et al. (1991), in their normative study, administered the Halstead-Reitan Test Battery and other neuropsychological tests to a sample of 553 normal subjects which included 319 subjects between 20 and 40 years of age, 134 between 41 and 59, and over 100 older than 60. In this study, they reported large declines in test performance associated with advancing age. Examples include 48% of variance attributable to age on the Halstead Impairment Index, 38% on the Category Test, and 34% on Trails B, while relatively small effects were noted on motor measures, e.g., 9% on Finger Tapping and 2 to 4% on the Dynamometer. Tests most affected by age appear to be those reflecting complex problem-solving, conceptual ability, cognitive flexibility, incidental memory, and psychomotor speed. In general, decreases in neuropsychological performance associated with advancing age are thought to be due to morphological brain changes. For instance, Coffey et al. (1992) examined 76 healthy adults 46 of whom were 60 or older and documented Magnetic Resonance Imaging (MRI) morphological changes, including decreased volume in the frontal lobes, the temporal lobes, and amygdala-hippocampal complex. Other changes included increasing volumes

of the third and lateral ventricals, and hyperintensity in the deep white matter and the pons. Notably, however, several elderly subjects did not manifest these changes and this is consistent with exceptional performance of some elderly "normals" on neuropsychological measures, suggesting that in some older normals, accelerated decline is not inexorable.

Little research into age effects in younger subjects, e.g., 18 to 45 years has been reported, although Yeudall et al. (1987) reported relatively small age effects. Their sample included subjects between 15 and 40 years old. Among males the highest age-variable correlation among the Halstead-Reitan measures were .30 for Trails B, .27 and .28 for Finger Tapping, .32 and .34 for Dynamometer, and .24 for Localization on the Tactual Performance Test. These results suggest that strong age effects are not observed until age 40 or later consistent with observations made by Reitan and Wolfson (1993).

All statistical analyses were conducted on an IBM PC 486 DX33. Unless otherwise stated, programs from SPSS/PC and V.5.0 (Norusis 1992) were utilized.

Table 4.3 below reports age-group correlations for males aged 18 to 44 on all measures employed in this study across the NC, OF, and PP groups. Age-group measure correlations noted to be significant ($p \leq .01$) or an absolute value $\geq .20$ for any group are discussed. Also, consistency in associative trends across groups is discussed.

The first and readily ascertained observation is that there are very few substantial age related shifts in performance. These shifts appear to be greatest among the NC group and are reflected in two areas, mainly: a) decreasing psychomotor abilities with age exemplified by age group - measure correlations of -.29 (Trails B), -.32 (Symbol Digit Modalities-Oral), and -.33 (Symbol Digit Modalities - Written); and b) a modest decline in verbal working memory with increasing age, as reflected by increased errors on the Williams' Verbal Paired Association Test (age group - VLTOTE correlation of .37).

The direction of these correlations is consistent with aging effects reviewed above, although relatively modest when compared to older normal samples. On other measures, the NC age group correlations are generally in the direction of reduced or maintained performance, but these correlations are typically very small and non-significant, e.g., VIQ ($r = -.01$) and FIQ ($r = .04$).

Within the OF group three observations are noted. Relative to NC their verbal abilities actually increase significantly, as measured by Verbal Wechsler intelligence measures, e.g., among the OF group (VIQ, $r = .18$; IN, $r = .21$; CO, $r = .21$). As well, the decline on psychomotor tasks which typically include a verbal component, e.g., Trails B and Symbol Digit Modalities, was considerably lower than for the NC group. This was also true for VLTOTE (a verbal paired-associate learning task) which places heavy

demands on verbal working memory. No single clear hypothesis is proposed for this apparent relative increase in verbal abilities among the OF group, but possible contributing factors may include an emphasis in prisons on educational programming, and reduced impulsivity associated with aging. In terms of the differences observed in the PP group, the above noted trends are generally more consistent with those of the OF group than with those of the NC, but also typically less pronounced.

In sum, differential patterns of aging effects were noted in the OF and PP groups compared to the NC, specifically, relative gains by the former 2 groups in verbal abilities and slower decline in psychomotor abilities was observed. However, in general, age effects were very small across the neuropsychological battery, including the Wechsler scales. Accordingly, except for the next section which deals exclusively with OF group age norms, the age grouping factor will be dropped in subsequent analyses.

Education has also been noted in several studies to be associated with IQ (e.g., Matarazzo, 1972, pp. 228-229 & p. 296) and substantial correlations have been noted in normal controls, but the relationship is less clear among neuropsychological variables (Leckliter & Matarazzo, 1989). Heaton et al.'s (1991) normative system does provide for education attenuated T scores on neuropsychological variables and Heaton's (1992) IQ norms do so as well. In general, educational effects have been shown to be much less influential than age effects and Reitan and Wolfson (1993) have

noted that in neurological populations they are all but non-existent. Yeudall et al. (1987) noted only minimal educational effects among their normal sample. Similarly Prigatano and Parsons (1976) reported a maximum correlation of .28 between education and various neuropsychological tests within their psychiatric sample. In considering both offenders and psychiatric patients, it is often difficult, if not impossible, to assess a common educational rating scheme, e.g., should special education classes be considered equivalent for these purposes; how to factor in social promotions, partial failures, equivalency statuses, and so on? In sum, decades ago, when an individual's education was often truncated for reasons other than ability, such as economic and other pragmatic reasons, educational achievement may have been less correlated with ability. Due to the above noted difficulties, no attempt has been made in this study to systematically evaluate educational effects.

TABLE 4.3

AGE GROUP CORRELATIONS *

(ages 18-29; 30-44)

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| ANNETT | r | -.04 | .01 | .07 |
| | n | ( 128) | ( 569) | ( 480) |
| | Pvalue | P= .636 | P= .857 | P= .120 |
| ANN-R | r | -.03 | .01 | .07 |
| | n | ( 128) | ( 569) | ( 480) |
| | Pvalue | P= .749 | P= .880 | P= .105 |
| ANN-L | r | .04 | -.01 | -.07 |
| | n | ( 128) | ( 569) | ( 480) |
| | Pvalue | P= .636 | P= .857 | P= .120 |
| ANN-B | r | -.04 | .00 | -.02 |
| | n | ( 128) | ( 569) | ( 480) |
| | Pvalue | P= .668 | P= .996 | P= .667 |
| VIQ | r | -.01 | .18 | .15 |
| | n | ( 109) | ( 584) | ( 448) |
| | Pvalue | P= .895 | **P= .000** | **P= .002** |
| PIQ | r | .10 | .08 | .02 |
| | n | ( 109) | ( 584) | ( 447) |
| | Pvalue | P= .302 | P= .056 | P= .668 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| VPDIF | r | -.10 | .11 | .14 |
| | n | ( 109) | ( 584) | ( 446) |
| | Pvalue | P= .292 | **P= .009** | **P= .003** |
| FIQ | r | .04 | .15 | .10 |
| | n | ( 109) | ( 584) | ( 446) |
| | Pvalue | P= .709 | **P= .000** | P= .034 |
| MINF | r | .11 | .08 | .04 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .252 | P= .041 | P= .463 |
| MAXF | r | -.13 | .01 | .05 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .168 | P= .786 | P= .270 |
| RANF | r | -.20 | -.06 | .03 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .037 | P= .118 | P= .483 |
| IN | r | -.08 | .21 | .21 |
| | n | ( 109) | ( 584) | ( 448) |
| | Pvalue | P= .417 | **P= .000** | **P= .000** |
| DSP | r | -.06 | .05 | .02 |
| | n | ( 109) | ( 584) | ( 447) |
| | Pvalue | P= .562 | P= .211 | P= .664 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| VO | r | .19 | .22 | .19 |
| | n | ( 109) | ( 582) | ( 448) |
| | Pvalue | P= .045 | **P= .000** | **P= .000** |
| AR | r | .07 | .11 | .15 |
| | n | ( 109) | ( 584) | ( 448) |
| | Pvalue | P= .483 | **P= .007** | **P= .001** |
| CO | r | .10 | .21 | .16 |
| | n | ( 109) | ( 584) | ( 448) |
| | Pvalue | P= .298 | **P= .000** | **P= .001** |
| SI | r | -.01 | .05 | .07 |
| | n | ( 109) | ( 584) | ( 447) |
| | Pvalue | P= .911 | P= .219 | P= .128 |
| MINV | r | .04 | .14 | .11 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .650 | **P= .001** | P= .016 |
| MAXV | r | -.02 | .16 | .17 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .814 | **P= .000** | **P= .000** |
| RANV | r | -.06 | .05 | .10 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .508 | P= .253 | P= .036 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| PC | r | .12 | .01 | .00 |
| | n | ( 109) | ( 584) | ( 447) |
| | Pvalue | P= .202 | P= .748 | P= .954 |
| PA | r | .06 | -.04 | -.09 |
| | n | ( 109) | ( 584) | ( 447) |
| | Pvalue | P= .512 | P= .280 | P= .066 |
| BD | r | -.10 | -.11 | -.15 |
| | n | ( 109) | ( 584) | ( 446) |
| | Pvalue | P= .302 | **P= .009** | **P= .001** |
| OA | r | -.03 | -.05 | -.10 |
| | n | ( 109) | ( 583) | ( 446) |
| | Pvalue | P= .735 | P= .205 | P= .039 |
| DSY | r | -.20 | -.03 | -.06 |
| | n | ( 109) | ( 584) | ( 445) |
| | Pvalue | P= .035 | P= .507 | P= .192 |
| MINP | r | .05 | -.02 | -.08 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .571 | P= .579 | P= .104 |
| MAXP | r | -.14 | -.08 | -.09 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .152 | P= .048 | P= .053 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| RANP | r | -.16 | -.08 | -.05 |
| | n | ( 109) | ( 581) | ( 441) |
| | Pvalue | P= .096 | P= .059 | P= .315 |
| CPM | r | -.09 | .07 | .11 |
| | n | ( 132) | ( 497) | ( 474) |
| | Pvalue | P= .318 | P= .115 | **P= .015** |
| FTAPP | r | .10 | .03 | -.01 |
| | n | ( 129) | ( 455) | ( 485) |
| | Pvalue | P= .284 | P= .559 | P= .862 |
| FTAPNP | r | .21 | .07 | -.04 |
| | n | ( 129) | ( 457) | ( 485) |
| | Pvalue | P= .019 | P= .159 | P= .344 |
| DYNP | r | .20 | .02 | .01 |
| | n | ( 130) | ( 570) | ( 476) |
| | Pvalue | P= .021 | P= .621 | P= .866 |
| DYNNP | r | .18 | -.00 | .04 |
| | n | ( 130) | ( 569) | ( 477) |
| | Pvalue | P= .045 | P= .940 | P= .423 |
| TRAILA | r | .10 | .14 | .12 |
| | n | ( 132) | ( 582) | ( 491) |
| | Pvalue | P= .253 | **P= .001** | **P= .008** |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| TRAILB | r | .29 | .17 | .12 |
| | n | ( 132) | ( 579) | ( 491) |
| | Pvalue | **P= .001** | **P= .000** | **P= .008** |
| PURDP | r | -.04 | -.13 | -.10 |
| | n | ( 131) | ( 492) | ( 490) |
| | Pvalue | P= .617 | **P= .005** | P= .024 |
| PURDNP | r | -.06 | -.05 | -.07 |
| | n | ( 131) | ( 493) | ( 491) |
| | Pvalue | P= .498 | P= .224 | P= .101 |
| PURDB | r | -.16 | -.09 | -.08 |
| | n | ( 131) | ( 492) | ( 490) |
| | Pvalue | P= .074 | P= .053 | P= .066 |
| PURDA | r | -.16 | -.08 | -.11 |
| | n | ( 131) | ( 480) | ( 488) |
| | Pvalue | P= .067 | P= .081 | P= .016 |
| TACPERP | r | -.00 | .16 | .09 |
| | n | ( 131) | ( 517) | ( 445) |
| | Pvalue | P= .967 | **P= .000** | P= .056 |
| TACPERNP | r | .03 | .12 | .15 |
| | n | ( 131) | ( 515) | ( 445) |
| | Pvalue | P= .749 | **P= .005** | **P= .001** |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| TACPERB | r | .03 | .17 | .11 |
| | n | ( 131) | ( 512) | ( 444) |
| | Pvalue | P= .777 | **P= .000** | P= .020 |
| TACPERFM | r | .10 | -.17 | -.14 |
| | n | ( 131) | ( 517) | ( 445) |
| | Pvalue | P= .250 | **P= .000** | **P= .003** |
| TACPERFL | r | -.10 | -.20 | -.14 |
| | n | ( 131) | ( 516) | ( 445) |
| | Pvalue | P= .256 | **P= .000** | **P= .004** |
| TACFRP | r | -.00 | .08 | -.16 |
| | n | ( 119) | ( 365) | ( 148) |
| | Pvalue | P= .997 | P= .107 | P= .048 |
| TACFRNP | r | -.04 | .10 | -.12 |
| | n | ( 119) | ( 370) | ( 149) |
| | Pvalue | P= .677 | P= .047 | P= .154 |
| LJPERR | r | .00 | .10 | .05 |
| | n | ( 123) | ( 373) | ( 456) |
| | Pvalue | P= .991 | P= .066 | P= .264 |
| LJPTIM | r | -.08 | .16 | .04 |
| | n | ( 123) | ( 372) | ( 455) |
| | Pvalue | P= .409 | **P= .002** | P= .409 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| LJNPERR | r | .16 | .07 | .07 |
| | n | ( 123) | ( 374) | ( 456) |
| | Pvalue | P= .078 | P= .153 | P= .131 |
| LJNPTIM | r | .01 | .17 | .04 |
| | n | ( 123) | ( 374) | ( 455) |
| | Pvalue | P= .915 | **P= .001** | P= .410 |
| LJBERR | r | .17 | .08 | .08 |
| | n | ( 123) | ( 373) | ( 456) |
| | Pvalue | P= .056 | P= .139 | P= .082 |
| LJBTIM | r | -.03 | .13 | .06 |
| | n | ( 123) | ( 374) | ( 455) |
| | Pvalue | P= .703 | **P= .010** | P= .182 |
| FTIPNP | r | -.03 | .08 | -.09 |
| | n | ( 130) | ( 368) | ( 401) |
| | Pvalue | P= .702 | P= .137 | P= .076 |
| FTIPNNP | r | -.13 | -.01 | -.13 |
| | n | ( 130) | ( 369) | ( 401) |
| | Pvalue | P= .152 | P= .852 | **P= .007** |
| FHANDR | r | .03 | -.03 | .08 |
| | n | ( 128) | ( 372) | ( 236) |
| | Pvalue | P= .766 | P= .612 | P= .246 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| FHANDL | r | .14 | -.02 | .10 |
| | n | ( 128) | ( 372) | ( 236) |
| | Pvalue | P= .104 | P= .658 | P= .141 |
| FLOCPS | r | -.02 | .11 | -.02 |
| | n | ( 130) | ( 367) | ( 487) |
| | Pvalue | P= .811 | P= .043 | P= .615 |
| FLOCPD | r | .06 | .03 | .08 |
| | n | ( 130) | ( 367) | ( 487) |
| | Pvalue | P= .503 | P= .527 | P= .067 |
| FLOCNPS | r | .10 | .01 | .02 |
| | n | ( 130) | ( 367) | ( 487) |
| | Pvalue | P= .267 | P= .887 | P= .620 |
| FLOCNPD | r | .13 | .09 | .07 |
| | n | ( 130) | ( 367) | ( 487) |
| | Pvalue | P= .133 | P= .096 | P= .115 |
| SDIGO | r | -.32 | -.11 | -.15 |
| | n | ( 107) | ( 375) | ( 153) |
| | Pvalue | **P= .001** | P= .027 | P= .073 |
| SDIGW | r | -.33 | -.06 | -.16 |
| | n | ( 107) | ( 376) | (153) |
| | Pvalue | **P= .001** | P= .277 | P= .043 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| APHAS | r | -.14 | .06 | -.05 |
| | n | ( 127) | ( 368) | ( 398) |
| | Pvalue | P= .123 | P= .243 | P= .337 |
| SSERR | r | -.11 | .11 | .07 |
| | n | ( 132) | ( 569) | ( 485) |
| | Pvalue | P= .217 | P= .011 | P= .129 |
| SEASHR | r | .09 | .01 | .02 |
| | n | ( 132) | ( 559) | ( 452) |
| | Pvalue | P= .327 | P= .862 | P= .739 |
| MFD | r | .09 | -.08 | -.12 |
| | n | ( 129) | ( 572) | ( 241) |
| | Pvalue | P= .295 | P= .060 | P= .065 |
| SGEST1 | r | -.04 | -.08 | -.01 |
| | n | ( 130) | ( 386) | ( 146) |
| | Pvalue | P= .631 | P= .099 | P= .882 |
| MINEST | r | .04 | -.05 | -.04 |
| | n | ( 130) | ( 385) | ( 145) |
| | Pvalue | P= .664 | P= .307 | P= .629 |
| OWTOT | r | -.04 | -.07 | -.03 |
| | n | ( 132) | ( 568) | ( 487) |
| | Pvalue | P= .621 | P= .103 | P= .449 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| WWTOT | r | .03 | -.02 | -.09 |
| | n | ( 104) | ( 559) | ( 204) |
| | Pvalue | P= .744 | P= .632 | P= .206 |
| VLNTRI | r | .25 | .08 | .14 |
| | n | ( 131) | ( 533) | ( 474) |
| | Pvalue | **P= .004** | P= .073 | **P= .003** |
| VLTOTE | r | .37 | .09 | .12 |
| | n | ( 131) | ( 532) | ( 474) |
| | Pvalue | **P= .000** | P= .035 | **P= .007** |
| NVLNTRI | r | .14 | .11 | .11 |
| | n | ( 132) | ( 386) | ( 470) |
| | Pvalue | P= .099 | P= .030 | P= .022 |
| NVLE | r | .11 | .14 | .11 |
| | n | ( 132) | ( 384) | ( 470) |
| | Pvalue | P= .208 | **P= .005** | P= .016 |
| NVLRNTRI | r | .09 | .12 | -.01 |
| | n | ( 132) | ( 386) | ( 470) |
| | Pvalue | P= .310 | P= .021 | P= .805 |
| NVLRE | r | .08 | .12 | .03 |
| | n | ( 132) | ( 385) | ( 470) |
| | Pvalue | P= .393 | P= .024 | P= .497 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| WCSSUB | r | .06 | -.11 | -.13 |
| | n | ( 108) | ( 574) | ( 448) |
| | Pvalue | P= .513 | **P= .010** | **P= .007** |
| WCSPER | r | .01 | .02 | .02 |
| | n | ( 108) | ( 570) | ( 448) |
| | Pvalue | P= .935 | P= .624 | P= .639 |
| WCSNPER | r | .06 | .05 | .15 |
| | n | ( 108) | ( 571) | ( 448) |
| | Pvalue | P= .533 | P= .265 | **P= .001** |
| WCSCOR | r | .02 | .05 | .10 |
| | n | ( 108) | ( 571) | ( 448) |
| | Pvalue | P= .836 | P= .233 | P= .034 |
| WCSUNI | r | -.12 | .06 | -.00 |
| | n | ( 108) | ( 571) | ( 448) |
| | Pvalue | P= .204 | P= .123 | P= .999 |
| WCSTOT | r | .01 | .05 | .10 |
| | n | ( 108) | ( 571) | ( 448) |
| | Pvalue | P= .953 | P= .225 | P= .028 |
| HCAT1 | r | .09 | -.06 | -.01 |
| | n | ( 129) | ( 575) | ( 467) |
| | Pvalue | P= .286 | P= .126 | P= .781 |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| HCAT2 | r | .10 | .03 | .01 |
| | n | ( 129) | ( 574) | ( 467) |
| | Pvalue | P= .266 | P= .547 | P= .761 |
| HCAT3 | r | .23 | .21 | .20 |
| | n | ( 129) | ( 572) | ( 467) |
| | Pvalue | **P= .008** | **P= .000** | **P= .000** |
| HCAT4 | r | .02 | .13 | .08 |
| | n | ( 129) | ( 564) | ( 467) |
| | Pvalue | P= .852 | **P= .002** | P= .093 |
| HCAT5 | r | -.03 | .09 | .09 |
| | n | ( 129) | ( 552) | ( 467) |
| | Pvalue | P= .747 | P= .046 | P= .045 |
| HCAT6 | r | -.12 | .11 | .08 |
| | n | ( 129) | ( 549) | ( 467) |
| | Pvalue | P= .192 | **P= .009** | P= .074 |
| HCAT7 | r | .11 | .22 | .13 |
| | n | ( 129) | ( 545) | ( 467) |
| | Pvalue | P= .196 | **P= .000** | **P= .006** |
| HCATCOR | r | .10 | .19 | .22 |
| | n | ( 129) | ( 570) | ( 467) |
| | Pvalue | P= .282 | **P= .000** | **P= .000** |

| Variable | | NC | OF | PP |
|---|---|---|---|---|
| HCATINC | r | .10 | .16 | .19 |
| | n | ( 129) | ( 567) | ( 467) |
| | Pvalue | P= .257 | **P= .000** | **P= .000** |
| HCATERR | r | .10 | .24 | .16 |
| | n | ( 129) | ( 582) | ( 467) |
| | Pvalue | P= .244 | **P= .000** | **P= .000** |

* correlation coefficients - 2 tailed significance ($P < .01$)

SECTION V OFFENDER GROUP NORMS STRATIFIED BY AGE

Tables 4.4 and 4.5 provide norms for the OF group for neuropsychological and Wechsler measures respectively. These norms are presented for the combined sample of offenders aged 18 to 44, and for two subgroupings aged 18 to 29 and 30 to 44. Means, standard deviations, ranges, and the number of subjects for each measure is provided by the tables. Also, 2-tailed t-tests for equality of means and Levene's Test for Equality of Variance for the two age subgroups were computed. Where P values are significant ($P \leq .01$), these are presented in bold type in the tables.

The normative tables for the OF group, stratified by age, are self-explanatory and, thus, require little comment, except for noting age effects. The average age of the 18 to 29 age grouping is 24.20 years and this, of course, is significantly different from the older group, aged 30 to 44, whose mean age is 34.80.

A review of Table 4.4 (Neuropsychological Variables) reveals, in general, few substantial differences in test performance due to age. However, although differences are small, the younger group showed a statistically significant ($P \leq .01$) superior performance relative to the older group on tasks which involved: a) perceptual motor speed. Tests with a high requirement of this type include Trails A and B, and the Purdue Pegboard; and b) spatial-perceptual organizational and/or non-verbal memory abilities. Tests tapping these abilities include the Tactual Performance Formboard, Williams' Non-Verbal Learning, and the Halstead Category.

In general, statistically significant age group differences among verbal measures were uncommon. Exceptions include the Seashore Speech Sounds Test and the number of categories achieved on the Wisconsin Card Sorting Test. In both instances, the younger group had the better performance.

Inspection of Table 4.5 (Wechsler Variables) indicates that on all Wechsler verbal subtests, except for Digit Span and Similarities, the older group out-performed the younger group. The Wechsler verbal scales are inclined to reflect gradual increases in verbal knowledge. Perhaps, then, this result reflects the influence of prison education programs.

Among Wechsler performance subtests, only differences on the Block Design subtest were significant, ($P \leq .01$). On this measure, which implicates, especially, visual spatial/perceptual organizational abilities, results are consistent with findings among the neuropsychological measures

requiring these abilities, a modest decline in the older group of offenders was observed.

The results of the correlational analysis presented in Section IV contrasting the 18 to 29 and 30 to 44 age groupings across the NC, OF, and PP samples, and the analysis of multiple t-tests comparing the test performance of these two age groupings of the OF group, can be summarized as follows: a) within the NC group, there is a tendency for psychomotor abilities and verbal working memory to decline with age. This trend was not as evident for the OF and PP groups as for the NC group; b) within the OF group, the younger group was noted to be more proficient on tasks requiring perceptual motor speed and spatial perceptual organization, but had lower scores on some Wechsler verbal scales, especially those susceptible to improvement through education. This latter difference may thus represent an educational, rather than an age effect.

Overall, the most significant finding is the absence of age effects noted across all three samples for most measures. Where significant differences were observed, they did not reveal large performance differences. On balance, it does not appear that the age effects noted justify separate consideration of the two age groupings in the subsequent analyses conducted in this study.

The norms presented for the two age groups of offenders are based on large samples, and, therefore, for clinical purposes, it is recommended that

the age factor be retained, as it may add precision, particularly, in the case of some Wechsler verbal measures.

TABLE 4.4

Neuropsychological Measures

Offenders Normative Statistics by AGE GROUP

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| AGE | (1)18-44 | 28.78 | 2vs.3 | 6.32 | 2vs.3 | 18.0-44.0 | 584 |
| | (2)18-29 | 24.20 | **P=.000** | 3.16 | **P=.000** | 18.0-29.9 | 332 |
| | (3)30-44 | 34.80 | | 3.93 | | 30.0-44.0 | 252 |
| CPM | (1)18-44 | 3.47 | 2vs.3 | 3.43 | 2vs.3 | .00-27.0 | 497 |
| | (2)18-29 | 3.27 | P=.115 | 3.27 | P=0.86 | .00-21.0 | 296 |
| | (3)30-44 | 3.76 | | 3.65 | | .00-27.0 | 201 |
| FTAPP | (1)18-44 | 51.24 | 2vs.3 | 6.90 | 2vs.3 | 12.2-70.8 | 455 |
| | (2)18-29 | 51.07 | P=.559 | 6.63 | P=.868 | 32.2-70.8 | 252 |
| | (3)30-44 | 51.46 | | 7.23 | | 12.2-67.2 | 203 |
| FTAPNP | (1)18-44 | 46.78 | 2vs.3 | 6.44 | 2vs.3 | 17.1-69.8 | 457 |
| | (2)18-29 | 46.40 | P=.159 | 6.50 | P=.859 | 17.1-69.8 | 252 |
| | (3)30-44 | 47.25 | | 6.34 | | 29.0-62.6 | 205 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| DYNP | (1)18-44 | 49.56 | 2vs.3 | 7.99 | 2vs.3 | 20.5-76.0 | 570 |
| | (2)18-29 | 49.41 | P=.621 | 8.06 | P=.977 | 22.6-76.0 | 324 |
| | (3)30-44 | 49.75 | | 7.90 | | 20.5-73.5 | 246 |
| DYNNP | (1)18-44 | 47.43 | 2vs.3 | 7.90 | 2vs.3 | 16.5-74.0 | 569 |
| | (2)18-29 | 47.45 | P=.940 | 7.87 | P=.889 | 17.5-73.3 | 324 |
| | (3)30-44 | 47.40 | | 7.95 | | 16.5-74.0 | 245 |
| TRAILA | (1)18-44 | 34.95 | 2vs.3 | 16.74 | 2vs.3 | 10.0-163.0 | 582 |
| | (2)18-29 | 32.95 | **P=.001** | 14.43 | P=.081 | 10.0-100.5 | 331 |
| | (3)30-44 | 37.58 | | 19.08 | | 13.9-163.0 | 251 |
| TRAILB | (1)18-44 | 80.87 | 2vs.3 | 38.62 | 2vs.3 | 28.4-312.0 | 579 |
| | (2)18-29 | 75.15 | **P=.000** | 32.16 | **P=.000** | 28.4-278.2 | 329 |
| | (3)30-44 | 88.40 | | 44.71 | | 30.9-312.0 | 250 |
| PURDP | (1)18-44 | 15.15 | 2vs.3 | 2.16 | 2vs.3 | 5.0-21.0 | 492 |
| | (2)18-29 | 15.37 | **P=.005** | 2.06 | P=.139 | 5.0-21.0 | 293 |
| | (3)30-44 | 14.81 | | 2.26 | | 7.0-20.0 | 199 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| PURDNP | (1)18-44 | 14.67 | 2vs.3 | 2.07 | 2vs.3 | 3.0-20.0 | 493 |
| | (2)18-29 | 14.76 | P=.224 | 2.15 | P=.756 | 3.0-20.0 | 293 |
| | (3)30-44 | 14.53 | | 1.95 | | 9.0-20.0 | 200 |
| PURDB | (1)18-44 | 11.95 | 2vs.3 | 1.92 | 2vs.3 | 4.0-24.0 | 492 |
| | (2)18-29 | 12.09 | P=.053 | 1.91 | P=.548 | 4.0-18.0 | 293 |
| | (3)30-44 | 11.74 | | 1.93 | | 5.0-24.0 | 199 |
| PURDA | (1)18-44 | 36.09 | 2vs.3 | 7.21 | 2vs.3 | 14.0-56.0 | 480 |
| | (2)18-29 | 36.57 | P=.081 | 7.39 | P=.588 | 14.0-56.0 | 284 |
| | (3)30-44 | 35.40 | | 6.90 | | 17.0-55.0 | 196 |
| TACPERP | (1)18-44 | 396.90 | 2vs.3 | 180.02 | 2vs.3 | 85.0-900.0 | 517 |
| | (2)18-29 | 371.84 | **P=.000** | 161.83 | **P=.004** | 102.0-900.0 | 298 |
| | (3)30-44 | 431.0 | | 197.49 | | 85.0-900.0 | 219 |
| TACPERNP | (1)18-44 | 290.52 | 2vs.3 | 162.71 | 2vs.3 | 69.0-900.0 | 515 |
| | (2)18-29 | 273.50 | **P=.005** | 152.92 | P=.034 | 69.0-900.0 | 298 |
| | (3)30-44 | 313.90 | | 172.92 | | 83.9-900.0 | 217 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| TACPERB | (1)18-44 | 163.14 | 2vs.3 | 106.33 | 2vs.3 | 38.6-900.0 | 512 |
| | (2)18-29 | 147.94 | **P=.000** | 86.90 | **P=.000** | 38.6-900.0 | 295 |
| | (3)30-44 | 183.80 | | 125.39 | | 52.0-900.0 | 217 |
| TACPERFM | (1)18-44 | 7.87 | 2vs.3 | 1.57 | 2vs.3 | 0.00-10.0 | 517 |
| | (2)18-29 | 8.11 | **P=.000** | 1.46 | P=.033 | 2.0-10.0 | 297 |
| | (3)30-44 | 7.55 | | 1.66 | | 0.0-10.0 | 220 |
| TACPERFL | (1)18-44 | 4.76 | 2vs.3 | 2.63 | 2vs.3 | 0.00-10.0 | 516 |
| | (2)18-29 | 5.20 | **P=.000** | 2.57 | P=.677 | 0.0-10.0 | 297 |
| | (3)30-44 | 4.16 | | 2.61 | | 0.0-10.0 | 219 |
| TACFRP | (1)18-44 | 6.41 | 2vs.3 | 3.39 | 2vs.3 | 2.0-28.1 | 365 |
| | (2)18-29 | 6.17 | P=.107 | 3.15 | P=.197 | 2.0-20.5 | 210 |
| | (3)30-44 | 6.74 | | 3.68 | | 2.5-28.1 | 155 |
| TACFRNP | (1)18-44 | 5.35 | 2vs.3 | 3.99 | 2vs.3 | 1.0-46.8 | 370 |
| | (2)18-29 | 4.99 | P=.047 | 3.67 | P=.241 | 1.0-46.8 | 214 |
| | (3)30-44 | 5.83 | | 4.34 | | 1.1-45.0 | 156 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| LJPERR | (1)18-44 | .35 | 2vs.3 | .65 | 2vs.3 | 0.00-4.0 | 373 |
| | (2)18-29 | .29 | P=.066 | .60 | P=.055 | 0.00-3.0 | 215 |
| | (3)30-44 | .42 | | .71 | | 0.0-4.0 | 158 |
| LJPTIM | (1)18-44 | 30.77 | 2vs.3 | 18.06 | 2vs.3 | 7.5-146.0 | 372 |
| | (2)18-29 | 28.35 | **P=.002** | 16.58 | **P=.007** | 7.5-146.0 | 215 |
| | (3)30-44 | 34.10 | | 19.47 | | 8.6-109.7 | 157 |
| LJNPERR | (1)18-44 | .20 | 2vs.3 | .48 | 2vs.3 | 0.0-3.0 | 374 |
| | (2)18-29 | .17 | P=.153 | .43 | **P=.006** | 0.0-2.0 | 215 |
| | (3)30-44 | .24 | | .53 | | 0.0-3.0 | 159 |
| LJNPTIM | (1)18-44 | 27.79 | 2vs.3 | 20.61 | 2vs.3 | 4.0-134.0 | 374 |
| | (2)18-29 | 24.73 | **P=.001** | 17.53 | **P=.001** | 4.0-127.4 | 215 |
| | (3)30-44 | 31.93 | | 23.59 | | 5.3-134.0 | 159 |
| LJBERR | (1)18-44 | .11 | 2vs.3 | .36 | 2vs.3 | 0.0-2.0 | 373 |
| | (2)18-29 | .08 | P=.139 | .31 | **P=.003** | 0.0-2.0 | 215 |
| | (3)30-44 | .14 | | .41 | | 0.0-2.0 | 158 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| LJBTIM | (1)18-44 | 22.80 | 2vs.3 | 15.23 | 2vs.3 | 3.9-137.2 | 374 |
| | (2)18-29 | 21.06 | **P=.010** | 12.58 | **P=.008** | 4.8-77.0 | 216 |
| | (3)30-44 | 25.18 | | 18.03 | | 3.9-137.2 | 158 |
| FTIPNP | (1)18-44 | 1.66 | 2vs.3 | 2.11 | 2vs.3 | 0.0-15.0 | 368 |
| | (2)18-29 | 1.52 | P=.137 | 1.99 | P=.156 | 0.0-12.0 | 214 |
| | (3)30-44 | 1.85 | | 2.26 | | 0.0-15.0 | 154 |
| FTIPNNP | (1)18-44 | 1.08 | 2vs.3 | 1.75 | 2vs.3 | 0.0-9.0 | 369 |
| | (2)18-29 | 1.10 | P=.852 | 1.68 | P=.708 | 0.0-9.0 | 213 |
| | (3)30-44 | 1.06 | | 1.84 | | 0.0-9.0 | 156 |
| FHANDR | (1)18-44 | .38 | 2vs.3 | 1.22 | 2vs.3 | 0.0-14.0 | 372 |
| | (2)18-29 | .41 | P=.612 | 1.33 | P=.383 | 0.0-14.0 | 215 |
| | (3)30-44 | .34 | | 1.07 | | 0.0- 7.0 | 157 |
| FHANDL | (1)18-44 | .45 | 2vs.3 | 1.16 | 2vs.3 | 0.0-13.0 | 372 |
| | (2)18-29 | .47 | P=.658 | 1.27 | P=.459 | 0.0-13.0 | 215 |
| | (3)30-44 | .42 | | 1.00 | | 0.0- 8.0 | 157 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| FLOCPS | (1)18-44 | .77 | 2vs.3 | 1.25 | 2vs.3 | 0.0-8.0 | 367 |
| | (2)18-29 | .66 | P=.043 | 1.22 | P=.452 | 0.0-8.0 | 212 |
| | (3)30-44 | .92 | | 1.27 | | 0.0-7.0 | 155 |
| FLOCPD | (1)18-44 | 3.37 | 2vs.3 | 3.07 | 2vs.3 | 0.0-20.0 | 367 |
| | (2)18-29 | 3.28 | P=.527 | 2.98 | P=.683 | 0.0-15.0 | 212 |
| | (3)30-44 | 3.48 | | 3.19 | | 0.0-20.0 | 155 |
| FLOCNPS | (1)18-44 | 1.10 | 2vs.3 | 1.65 | 2vs.3 | 0.0-10.0 | 367 |
| | (2)18-29 | 1.08 | P=.887 | 1.57 | P=.318 | 0.0-10.0 | 212 |
| | (3)30-44 | 1.11 | | 1.77 | | 0.0-10.0 | 155 |
| FLOCNPD | (1)18-44 | 3.83 | 2vs.3 | 3.25 | 2vs.3 | 0.0-18.0 | 367 |
| | (2)18-29 | 3.59 | P=.096 | 2.96 | **P=.011** | 0.0-13.0 | 212 |
| | (3)30-44 | 4.16 | | 3.60 | | 0.0-18.0 | 155 |
| SDIGO | (1)18-44 | 56.21 | 2vs.3 | 13.82 | 2vs.3 | 24.0-104.0 | 375 |
| | (2)18-29 | 57.57 | P=.027 | 13.75 | P=.869 | 27.0-104.0 | 215 |
| | (3)30-44 | 54.38 | | 13.75 | | 24.0-98.0 | 160 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| SDIGW | (1)18-44 | 47.26 | 2vs.3 | 10.58 | 2vs.3 | 17.0-85.0 | 376 |
| | (2)18-29 | 47.78 | P=.277 | 10.50 | P=.841 | 23.0-85.0 | 215 |
| | (3)30-44 | 46.58 | | 10.68 | | 17.0-74.0 | 161 |
| APHAS | (1)18-44 | 1.11 | 2vs.3 | 2.09 | 2vs.3 | 0.0-15.0 | 368 |
| | (2)18-29 | 1.00 | P=.043 | 1.81 | **P=.006** | 0.0-12.0 | 212 |
| | (3)30-44 | 1.26 | | 2.43 | | 0.0-15.0 | 156 |
| SSERR | (1)18-44 | 5.82 | 2vs.3 | 4.07 | 2vs.3 | 0.0-34.0 | 569 |
| | (2)18-29 | 5.45 | **P=.011** | 3.95 | P=.033 | 0.0-34.0 | 327 |
| | (3)30-44 | 6.33 | | 4.19 | | 0.0-32.0 | 242 |
| SEASHR | (1)18-44 | 4.30 | 2vs.3 | 3.33 | 2vs.3 | 0.0-24.0 | 559 |
| | (2)18-29 | 4.27 | P=.862 | 3.36 | P=.282 | 0.0-18.0 | 321 |
| | (3)30-44 | 4.32 | | 3.29 | | 0.0-24.0 | 238 |
| MFD | (1)18-44 | 42.96 | 2vs.3 | 3.90 | 2vs.3 | 16.0-45.0 | 572 |
| | (2)18-29 | 43.22 | P=.060 | 3.91 | P=.080 | 16.0-45.0 | 327 |
| | (3)30-44 | 42.60 | | 3.86 | | 19.0-45.0 | 245 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| SGEST1 | (1)18-44 | 52.04 | 2vs.3 | 16.92 | 2vs.3 | 14.0-117.0 | 386 |
| | (2)18-29 | 53.26 | P=.099 | 16.88 | P=.044 | 18.0-117.0 | 222 |
| | (3)30-44 | 50.38 | | 16.88 | | 14.0- 99.0 | 164 |
| MINEST | (1)18-44 | 54.22 | 2vs.3 | 21.22 | 2vs.3 | 7.5-156.0 | 385 |
| | (2)18-29 | 55.16 | P=.307 | 20.32 | P=.452 | 7.5-134.6 | 225 |
| | (3)30-44 | 52.91 | | 22.42 | | 12.3-156.1 | 160 |
| OWTOT | (1)18-44 | 12.14 | 2vs.3 | 3.75 | 2vs.3 | 2.1-30.7 | 568 |
| | (2)18-29 | 12.36 | P=.103 | 3.68 | P=.491 | 3.0-30.7 | 324 |
| | (3)30-44 | 11.84 | | 3.82 | | 2.1-25.6 | 244 |
| WWTOT | (1)18-44 | 10.83 | 2vs.3 | 3.29 | 2vs.3 | 0.0-26.6 | 559 |
| | (2)18-29 | 10.89 | P=.632 | 3.15 | P=.666 | 2.7-22.7 | 321 |
| | (3)30-44 | 10.75 | | 3.46 | | 0.0-26.6 | 238 |
| VLNTRI | (1)18-44 | 3.54 | 2vs.3 | 1.24 | 2vs.3 | 1.0-5.0 | 533 |
| | (2)18-29 | 3.46 | P=.073 | 1.28 | P=.044 | 1.0-5.0 | 304 |
| | (3)30-44 | 3.66 | | 1.18 | | 1.0-5.0 | 229 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| VLTOTE | (1)18-44 | 9.10 | 2vs.3 | 7.53 | 2vs.3 | 0.0-38.0 | 532 |
| | (2)18-29 | 8.50 | P=.035 | 7.23 | P=.098 | 0.0-36.0 | 303 |
| | (3)30-44 | 9.89 | | 7.84 | | 0.0-38.0 | 229 |
| NVLNTRI | (1)18-44 | 1.80 | 2vs.3 | 1.08 | 2vs.3 | 1.0-5.0 | 386 |
| | (2)18-29 | 1.70 | P=.030 | .96 | P=.026 | 1.0-5.0 | 222 |
| | (3)30-44 | 1.94 | | 1.20 | | 1.0-5.0 | 164 |
| NVLE | (1)18-44 | 1.26 | 2vs.3 | 2.24 | 2vs.3 | 0.0-16.0 | 384 |
| | (2)18-29 | .99 | **P=.005** | 1.74 | **P=.000** | 0.0-13.0 | 221 |
| | (3)30-44 | 1.63 | | 2.74 | | 0.0-16.0 | 163 |
| NVLRNTRI | (1)18-44 | 1.99 | 2vs.3 | 1.38 | 2vs.3 | 1.0-5.0 | 386 |
| | (2)18-29 | 1.85 | P=.021 | 1.31 | P=.032 | 1.0-5.0 | 223 |
| | (3)30-44 | 2.18 | | 1.45 | | 1.0-5.0 | 163 |
| NVLRE | (1)18-44 | 1.64 | 2vs.3 | 2.83 | 2vs.3 | 0.0-15.0 | 385 |
| | (2)18-29 | 1.36 | P=.024 | 2.54 | **P=.011** | 0.0-15.0 | 223 |
| | (3)30-44 | 2.02 | | 3.16 | | 0.0-15.0 | 162 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| WCSSUB | (1)18-44 | 5.20 | 2vs.3 | 1.77 | 2vs.3 | 0.0-6.0 | 574 |
| | (2)18-29 | 5.37 | **P=.010** | 1.57 | **P=.000** | 0.0-6.0 | 329 |
| | (3)30-44 | 4.98 | | 1.98 | | 0.0-6.0 | 245 |
| WCSPER | (1)18-44 | 9.92 | 2vs.3 | 10.63 | 2vs.3 | 0.0-66.0 | 570 |
| | (2)18-29 | 9.73 | P=.624 | 9.76 | P=.057 | 0.0-55.0 | 327 |
| | (3)30-44 | 10.17 | | 11.72 | | 0.0-66.0 | 243 |
| WCSNPER | (1)18-44 | 9.82 | 2vs.3 | 8.93 | 2vs.3 | 0.0-52.0 | 571 |
| | (2)18-29 | 9.46 | P=.265 | 8.75 | P=.238 | 0.0-50.0 | 328 |
| | (3)30-44 | 10.30 | | 9.16 | | 0.0-52.0 | 243 |
| WCSCOR | (1)18-44 | 18.38 | 2vs.3 | 15.87 | 2vs.3 | 0.0-80.0 | 571 |
| | (2)18-29 | 17.70 | P=.233 | 14.74 | P=.022 | 0.0-80.0 | 328 |
| | (3)30-44 | 19.30 | | 17.26 | | 0.0-80.0 | 243 |
| WCSUN1 | (1)18-44 | 2.11 | 2vs.3 | 4.41 | 2vs.3 | 0.0-28.0 | 571 |
| | (2)18-29 | 1.87 | P=.123 | 4.18 | P=.023 | 0.0-28.0 | 328 |
| | (3)30-44 | 2.44 | | 4.69 | | 0.0-28.0 | 243 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| HCAT5 | (1)18-44 | 12.40 | 2vs.3 | 6.53 | 2vs.3 | 0.0-40.0 | 552 |
| | (2)18-29 | 11.93 | P=.046 | 6.14 | **P=.003** | 0.0-30.0 | 321 |
| | (3)30-44 | 13.05 | | 7.00 | | 1.0-40.0 | 231 |
| HCAT6 | (1)18-44 | 7.26 | 2vs.3 | 5.81 | 2vs.3 | 0.0-38.0 | 549 |
| | (2)18-29 | 6.71 | **P=.009** | 5.32 | **P=.001** | 0.0-38.0 | 320 |
| | (3)30-44 | 8.02 | | 6.37 | | 0.0-35.0 | 229 |
| HCAT7 | (1)18-44 | 3.86 | 2vs.3 | 2.42 | 2vs.3 | 0.0-13.0 | 545 |
| | (2)18-29 | 3.42 | **P=.000** | 2.20 | P=.026 | 0.0-12.0 | 319 |
| | (3)30-44 | 4.48 | | 2.58 | | 0.0-13.0 | 226 |
| HCATCOR | (1)18-44 | 8.09 | 2vs.3 | 4.36 | 2vs.3 | 0.0-24.0 | 570 |
| | (2)18-29 | 7.39 | **P=.000** | 3.91 | **P=.002** | 0.0-23.0 | 326 |
| | (3)30-44 | 9.02 | | 4.75 | | 0.0-24.0 | 244 |
| HCATINC | (1)18-44 | 11.66 | 2vs.3 | 7.80 | 2vs.3 | 0.0-57.0 | 567 |
| | (2)18-29 | 10.58 | **P=.000** | 6.50 | **P=.001** | 0.0-45.0 | 324 |
| | (3)30-44 | 13.09 | | 9.07 | | 1.0-57.0 | 243 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| WCSTOT | (1)18-44 | 21.94 | 2vs.3 | 18.45 | 2vs.3 | 0.0-90.0 | 571 |
| | (2)18-29 | 21.13 | P=.225 | 17.70 | P=.115 | 0.0-90.0 | 328 |
| | (3)30-44 | 23.03 | | 19.39 | | 2.0-90.0 | 243 |
| HCAT1 | (1)18-44 | .06 | 2vs.3 | .26 | 2vs.3 | 0.0-2.0 | 575 |
| | (2)18-29 | .07 | P=.126 | .30 | **P=.002** | 0.0-2.0 | 329 |
| | (3)30-44 | .04 | | .19 | | 0.0-1.0 | 246 |
| HCAT2 | (1)18-44 | .40 | 2vs.3 | .57 | 2vs.3 | 0.0-4.0 | 574 |
| | (2)18-29 | .38 | P=.547 | .54 | P=.087 | 0.0-2.0 | 329 |
| | (3)30-44 | .41 | | .62 | | 0.0-4.0 | 245 |
| HCAT3 | (1)18-44 | 14.56 | 2vs.3 | 10.93 | 2vs.3 | 0.0-36.0 | 572 |
| | (2)18-29 | 12.55 | **P=.000** | 10.16 | **P=.000** | 0.0-36.0 | 328 |
| | (3)30-44 | 17.26 | | 11.36 | | 0.0-36.0 | 244 |
| HCAT4 | (1)18-44 | 10.0 | 2vs.3 | 9.93 | 2vs.3 | 0.0-40.0 | 564 |
| | (2)18-29 | 8.88 | **P=.002** | 9.25 | **P=.001** | 0.0-39.0 | 325 |
| | (3)30-44 | 11.52 | | 10.63 | | 0.0-40.0 | 239 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| HCATERR | (1)18-44 | 51.09 | 2vs.3 | 27.09 | 2vs.3 | 7.0-130.0 | 582 |
| | (2)18-29 | 45.38 | **P=.001** | 24.71 | **P=.002** | 7.0-121.0 | 332 |
| | (3)30-44 | 58.67 | | 28.27 | | 8.0-130.0 | 250 |

* t-test for Equality of Means (2-tailed) P≤.01 in bold type

** Levene's Test For Equality Of Variances (2-tailed) P≤.01 in bold type

*** Rounded to one decimal point

TABLE 4.5

WAIS-R & Related Variables

Offenders Normative Statistics by AGE GROUP

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| AGE | (1)18-44 | 28.78 | 2vs.3 | 6.32 | 2vs.3 | 18.0-44.0 | 584 |
| | (2)18-29 | 24.20 | **P=.000** | 3.16 | **P=.000** | 18.0-29.9 | 332 |
| | (3)30-44 | 34.80 | | 3.93 | | 30.0-44.0 | 252 |
| ANNETT | (1)18-44 | 10.36 | 2vs.3 | 3.02 | 2vs.3 | 0.0-12.0 | 569 |
| | (2)18-29 | 10.34 | P=.857 | 2.97 | P=.796 | 0.0-12.0 | 322 |
| | (3)30-44 | 10.38 | | 3.10 | | 0.0-12.0 | 247 |
| ANN-R | (1)18-44 | 9.17 | 2vs.3 | 3.54 | 2vs.3 | 0.0-12.0 | 569 |
| | (2)18-29 | 9.15 | P=.880 | 3.47 | P=.425 | 0.0-12.0 | 322 |
| | (3)30-44 | 9.19 | | 3.63 | | 0.0-12.0 | 247 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| ANN-L | (1)18-44 | 1.64 | 2vs.3 | 3.02 | 2vs.3 | 0.0-12.0 | 569 |
| | (2)18-29 | 1.66 | P=.857 | 2.97 | P=.796 | 0.0-12.0 | 322 |
| | (3)30-44 | 1.62 | | 3.10 | | 0.0-12.0 | 247 |
| ANN-B | (1)18-44 | 1.19 | 2vs.3 | 2.06 | 2vs.3 | 0.0-11.0 | 569 |
| | (2)18-29 | 1.19 | P=.996 | 2.07 | P=.699 | 0.0-11.0 | 322 |
| | (3)30-44 | 1.19 | | 2.05 | | 0.0-10.0 | 247 |
| VIQ | (1)18-44 | 94.13 | 2vs.3 | 13.15 | 2vs.3 | 59.0-147.0 | 584 |
| | (2)18-29 | 92.08 | **P=.000** | 12.66 | P=.361 | 59.0-147.0 | 332 |
| | (3)30-44 | 96.83 | | 13.31 | | 62.0-130.0 | 252 |
| PIQ | (1)18-44 | 95.64 | 2vs.3 | 13.48 | 2vs.3 | 57.0-140.0 | 584 |
| | (2)18-29 | 94.71 | P=.056 | 12.99 | P=.417 | 57.0-134.0 | 332 |
| | (3)30-44 | 96.87 | | 14.02 | | 60.0- 40.0 | 252 |
| VPDIF | (1)18-44 | -1.51 | 2vs.3 | 11.86 | 2vs.3 | -43.0-40.0 | 584 |
| | (2)18-29 | -2.63 | **P=.009** | 12.22 | P=.373 | -43.0-40.0 | 332 |
| | (3)30-44 | - .03 | | 11.24 | | -33.0-30.0 | 252 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| FIQ | (1)18-44 | 94.11 | 2vs.3 | 12.92 | 2vs.3 | 62.0-142.0 | 584 |
| | (2)18-29 | 92.38 | **P=.000** | 12.12 | P=.145 | 69.0-142.0 | 332 |
| | (3)30-44 | 96.40 | | 13.59 | | 62.0-130.0 | 252 |
| MINF | (1)18-44 | 5.76 | 2vs.3 | 1.94 | 2vs.3 | 1.0-11.0 | 581 |
| | (2)18-29 | 5.62 | P=.041 | 1.91 | P=.867 | 1.0-11.0 | 330 |
| | (3)30-44 | 5.95 | | 1.96 | | 1.0-11.0 | 251 |
| MAXF | (1)18-44 | 12.85 | 2vs.3 | 2.47 | 2vs.3 | 5.0-19.0 | 581 |
| | (2)18-29 | 12.82 | P=.786 | 2.39 | P=.338 | 7.0-18.0 | 330 |
| | (3)30-44 | 12.88 | | 2.57 | | 5.0-19.0 | 251 |
| RANF | (1)18-44 | 7.08 | 2vs.3 | 2.10 | 2vs.3 | 2.0-14.0 | 581 |
| | (2)18-29 | 7.20 | P=.118 | 2.05 | P=.619 | 3.0-14.0 | 330 |
| | (3)30-44 | 6.93 | | 2.16 | | 2.0-13.0 | 251 |
| IN | (1)18-44 | 8.71 | 2vs.3 | 3.04 | 2vs.3 | 1.0-18.0 | 584 |
| | (2)18-29 | 8.14 | **P=.000** | 2.90 | P=.357 | 1.0-16.0 | 332 |
| | (3)30-44 | 9.45 | | 3.06 | | 1.0-18.0 | 252 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| DSP | (1)18-44 | 9.71 | 2vs.3 | 2.77 | 2vs.3 | 2.0-18.0 | 584 |
| | (2)18-29 | 9.59 | P=.211 | 2.74 | P=.879 | 3.0-18.0 | 332 |
| | (3)30-44 | 9.88 | | 2.80 | | 2.0-18.0 | 252 |
| VO | (1)18-44 | 8.54 | 2vs.3 | 2.70 | 2vs.3 | 1.0-17.0 | 582 |
| | (2)18-29 | 8.03 | **P=.000** | 2.59 | P=.442 | 1.0-17.0 | 331 |
| | (3)30-44 | 9.23 | | 2.69 | | 1.0-17.0 | 251 |
| AR | (1)18-44 | 9.22 | 2vs.3 | 2.80 | 2vs.3 | 2.0-17.0 | 584 |
| | (2)18-29 | 8.95 | **P=.007** | 2.70 | P=.323 | 2.0-16.0 | 332 |
| | (3)30-44 | 9.57 | | 2.89 | | 2.0-17.0 | 252 |
| CO | (1)18-44 | 9.27 | 2vs.3 | 3.09 | 2vs.3 | 3.0-16.0 | 584 |
| | (2)18-29 | 8.69 | **P=.000** | 3.02 | P=.882 | 3.0-16.0 | 332 |
| | (3)30-44 | 10.03 | | 3.02 | | 3.0-16.0 | 252 |
| SI | (1)18-44 | 8.94 | 2vs.3 | 3.01 | 2vs.3 | 1.0-18.0 | 584 |
| | (2)18-29 | 8.81 | P=.219 | 2.99 | P=.867 | 1.0-18.0 | 332 |
| | (3)30-44 | 9.12 | | 3.03 | | 1.0-17.0 | 252 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| MINV | (1)18-44 | 6.63 | 2vs.3 | 2.35 | 2vs.3 | 1.0-14.0 | 581 |
| | (2)18-29 | 6.35 | **P=.001** | 2.28 | P=.917 | 1.0-14.0 | 330 |
| | (3)30-44 | 7.01 | | 2.39 | | 1.0-13.0 | 251 |
| MAXV | (1)18-44 | 11.60 | 2vs.3 | 2.73 | 2vs.3 | 4.0-18.0 | 581 |
| | (2)18-29 | 11.22 | **P=.000** | 2.68 | P=.882 | 4.0-18.0 | 330 |
| | (3)30-44 | 12.08 | | 2.73 | | 4.0-18.0 | 251 |
| RANV | (1)18-44 | 4.96 | 2vs.3 | 2.06 | 2vs.3 | 1.0-12.0 | 581 |
| | (2)18-29 | 4.88 | P=.253 | 2.07 | P=.242 | 1.0-11.0 | 330 |
| | (3)30-44 | 5.08 | | 2.04 | | 1.0-12.0 | 251 |
| PC | (1)18-44 | 9.55 | 2vs.3 | 2.91 | 2vs.3 | 1.0-18.0 | 584 |
| | (2)18-29 | 9.52 | P=.748 | 2.78 | P=.076 | 2.0-18.0 | 332 |
| | (3)30-44 | 9.60 | | 3.08 | | 1.0-18.0 | 252 |
| PA | (1)18-44 | 9.43 | 2vs.3 | 2.85 | 2vs.3 | 1.0-19.0 | 584 |
| | (2)18-29 | 9.54 | P=.280 | 2.76 | P=.160 | 1.0-17.0 | 332 |
| | (3)30-44 | 9.28 | | 2.96 | | 2.0-19.0 | 252 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| BD | (1)18-44 | 10.03 | 2vs.3 | 2.65 | 2vs.3 | 3.0-17.0 | 584 |
| | (2)18-29 | 10.28 | **P=.009** | 2.63 | P=.708 | 4.0-17.0 | 332 |
| | (3)30-44 | 9.70 | | 2.64 | | 3.0-16.0 | 252 |
| OA | (1)18-44 | 9.45 | 2vs.3 | 2.84 | 2vs.3 | 1.0-17.0 | 583 |
| | (2)18-29 | 9.58 | P=.205 | 2.80 | P=.624 | 2.0-17.0 | 331 |
| | (3)30-44 | 9.28 | | 2.89 | | 1.0-17.0 | 252 |
| DSY | (1)18-44 | 7.77 | 2vs.3 | 2.28 | 2vs.3 | 2.0-17.0 | 584 |
| | (2)18-29 | 7.83 | P=.507 | 2.27 | P=.433 | 3.0-17.0 | 332 |
| | (3)30-44 | 7.70 | | 2.30 | | 2.0-14.0 | 252 |
| MINP | (1)18-44 | 6.63 | 2vs.3 | 1.91 | 2vs.3 | 1.0-13.0 | 581 |
| | (2)18-29 | 6.67 | P=.579 | 1.87 | P=.746 | 1.0-13.0 | 330 |
| | (3)30-44 | 6.58 | | 1.97 | | 1.0-13.0 | 251 |
| MAXP | (1)18-44 | 11.83 | 2vs.3 | 2.57 | 2vs.3 | 5.0-19.0 | 581 |
| | (2)18-29 | 12.02 | P=.048 | 2.48 | P=.386 | 6.0-18.0 | 330 |
| | (3)30-44 | 11.59 | | 2.68 | | 5.0-19.0 | 251 |

| Variable | AGE Group | MEAN | Sig.* | SD | Sig.** | Range*** | N |
|---|---|---|---|---|---|---|---|
| RANP | (1)18-44 | 5.20 | 2vs.3 | 2.13 | 2vs.3 | 1.0-13.0 | 581 |
| | (2)18-29 | 5.35 | P=.059 | 2.08 | P=.964 | 1.0-11.0 | 330 |
| | (3)30-44 | 5.01 | | 2.18 | | 1.0-13.0 | 251 |

* t-test for Equality of Means (2-tailed) P≤.01 in bold type

** Levene's Test for Equality of Variances (2-tailed) P≤.01 in bold type

*** Rounded to one decimal point

## SECTION VI GROUP PERFORMANCE CONTRASTS FOR NEUROPSYCHOLOGICAL VARIABLES

### Neuropsychological Variables

Table 4.6 provides descriptive statistics, including means, standard deviations, ranges, and number of subjects reported on for the NC, OF, and PP groups. As well, overall analysis of variance p values and subsequent Scheffé contrasts for the three groups are tabulated. Table 4.7 provides the same statistics for Wechsler variables.

Inspection of Table 4.6 indicates that for all variables, except age, differences were significant (ANOVA p values $\leq .0000$). Further, for a large majority of measures, Scheffé contrasts indicate that performance of the OF group was significantly poorer than that of the NC group, and, in turn, the performance of the PP group was significantly lower than that of the OF group (Performance of PP < OF < NC).

The only areas where the performance of the OF group was not significantly lower than that of the NC group was on a measure of aphasia (Aphasia Test), measures of motor speed (Finger Tapping), motor strength (Dynamometer - preferred hand), and simple perceptual and sensory tests (e.g., HCAT 1 & HCAT 2, Finger-Tip Name Writing, Tactual Form Recognition), but it seems that even a simple addition of complexity, e.g., going from single to double stimulation in the Finger Localization Test produces significant results. On no measure was the performance of the OF group superior to that of the NC group.

The performance of the PP group was significantly poorer than the NC group for all variables, including simple motor-sensory measures. Relative to the OF group, they were also significantly poorer on all measures except for Trails A and Face Hand Recognition.

Table 4.6a was developed to provide an index of the magnitude of the significant differences which are generally characterized by the performance relationship: PP < OF < NC, particularly on tests with a high cognitive processing component. Thus, Table 4.6a relates the relative performance of each group on representative cognitive measures to the mean performance of the NC group. For instance, inspection of Table 4.6 indicates that on the Halstead Category Test 13% of the NC group, 47% of the OF group, and 69% of the PP group were 1 *SD* below the normal control mean. If 2 *SDs* were taken to be an index of severe impairment, then 4%, 27%, and 51%of the groups would qualify respectively.

On other putative measures of prefrontal function, the OF and PP groups also show high levels of impairment, e.g., on the Wisconsin Card Sorting Test, Oral Word Fluency, and Trails B, 30%, 55%, and 46% of the OF group are 1 *SD* below the NC mean, while 17%, 9%, and 30% are 2 *SDs* below, respectively. Overall, between 30% and 57% of the OF group is 1 *SD* below the NC mean, and between 9% and 30% are 2 *SDs* below, depending on the measure. The PP group, by any account, is severely impaired with between 44% and 80% of test measure scores 1 *SD* and between 18% and 55% of these subjects score 2 *SDs* below the NC mean across the various

measures. Of course, to the extent that different brain systems are responsible for performance on different measures, these percentages do not suggest, on average, that members scoring well on one set of measures will do well on other measures. Rather, it suggests that different kinds of deficits may produce global impairment. Thus, average levels of impairment across tests really reflect minimum possible boundaries to overall impairment levels. The factor analyses and Modal Profile Analyses conducted in sections following should help to clarify these relationships.

Further appreciation of the level of impairment of the OF group relative to brain damaged groups can be gleaned from Gaskin's (1989) analysis. He tabulated the results of 120 studies representing 4,418 brain damaged patients who were administered the Halstead Category Test. The overall mean error score for all patients representing all ages was 72.30 (*SD* 30.74). It is well known, however, that performance on the HCT declines substantially with advancing age, e.g., Leckliter and Matarazzo (1989) found that across 3 studies that examined this relationship, the average correlation between age and HCT was .54. In older subjects, age effects can be quite dramatic, e.g., Mack and Carlson found the mean error score on the HCT to 91.73 for a group of 41 older normals (mean age 69.76).

Thus, examination of specific studies involving younger brain damaged groups may be more informative. Drudge, Williams, Kessler, and Gomes (1984) administered the HCT as part of a recovery study to 15 patients (13 male, 2 female, mean age 24.8) who sustained severe closed head injuries

shortly after their trauma and 1 year post trauma. Nine of the patients had received coma ratings of Grade III and 6 of Grade IV; both grades generally accepted as indicative of severe trauma and associated with poor global outcome and impaired intellectual recovery. These patients obtained mean HCT error scores of 89.3 post injury and 55.1 at 1 year follow-up. Dikmen, Reitan, and Temkin (1983) examined recovery of functions in a group of 27 patients (23 male, 4 female, mean age 24.62) who had sustained "mild to severe" (p. 333) head injuries with loss of consciousness of at least 1 hour and hospitalization. They were neuropsychologically evaluated subject to capacity post trauma, 12 months post trauma, and 18 months post trauma. Respective average HCT mean error scores were 43.51, 33.78, and 27.22 respectively.

Heaton et al. (1979) examined a chronic brain disorder group. The group consisted of 14 females and 11 males (mean age 28.3). Ten patients had traumatic head injuries, 6 with cerebral tumors, 2 with cerebrovascular accidents, 2 with cerebral anoxia, 2 with epilepsy (1 of whom had had a temporal lobectomy) and 1 each with multiple sclerosis, hydrocephalus, and an arteriovenous malformation. These patients had their neurological condition for over 1 year and their course was considered to be static or only very slowly progressive. Their mean HCT error score was 60.8 (*SD* 28.8). Dodrill and Dikmen (1978) assessed a group of 57 patients with seizure disorders and 45 patients with head injuries. For the combined group (mean age 26.45 years) they reported a mean HCT error score of 47.25 (*SD*

27.06). Dodrill and Clemmons (1984) contrasted the neuropsychological performance of 39 young adults with seizure disorders who had been evaluated, including the HCT, 3 to 11 years previously. They rated these subjects on measures of vocational adjustment, independence in living, and overall adjustment, then classified them as fully functioning or deficient functioning. The former group had previously obtained a mean HCT error score of 39.33 (*SD* 26.07) while the latter group's score was 70.26 (*SD* 30.02). A final example, Heaton, Nelson, Thompson, Burks, and Franklin (1985) contrasted the performance of 57 patients with relapsing-remitting multiple sclerosis (MS) with that of 43 who had chronic-progressive MS. Overall mean age was 37.38 years (the chronic-progressive group was significantly older). The relapsing-remitting group had a mean HCT error score of 34.98 (*SD* 23.49) while the chronic-progressive group had an error score of 56.84 (*SD* 30.57).

The above studies of younger neurological patients suggest that an error score of 51.09 (*SD* 27.09), which characterized the OF group in this study, represents a level of functional impairment that is often associated with significant brain damage in neurological samples. The mean HCT error score of 69.19 (*SD* 31.03) obtained by the PP group is greater than that reported for any psychiatric group of similar age reviewed by Gaskin (1989). Indeed, their level of impairment would appear to considerably exceed that of brain damaged groups of similar age (cf. Gaskin, 1989).

TABLE 4.6

Neuropsychological Variables

Descriptive Statistics For Normal Controls, Offenders, & Psychiatric Patients

ANOVA

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| AGE | (1)NC | 27.92 | 7.03 | 18.0-44.0 | 132 | .2997 | NC = OF | |
| | (2)OF | 28.78 | 6.32 | 18.0-44.0 | 584 | | NC = PP | |
| | (3)PP | 28.94 | 7.09 | 18.0-44.0 | 494 | | OF = PP | |
| CPM | (1)NC | 1.09 | 1.34 | 00.0- 6.0 | 132 | **.0000** | NC ≠ OF | ** |
| | (2)OF | 3.47 | 3.43 | 00.0-27.0 | 497 | | NC ≠ PP | ** |
| | (3)PP | 4.69 | 4.91 | 00.0-33.0 | 474 | | OF ≠ PP | ** |
| FTAPP | (1)NC | 51.99 | 7.78 | 26.6-71.0 | 129 | **.0000** | NC = OF | |
| | (2)OF | 51.24 | 6.90 | 12.2-70.8 | 455 | | NC ≠ PP | ** |
| | (3)PP | 48.01 | 8.56 | 10.2-73.6 | 485 | | OF ≠ PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| FTAPNP | (1)NC | 47.64 | 6.69 | 26.8-63.4 | 129 | **.0000** | NC = OF | | |
| | (2)OF | 46.78 | 6.44 | 17.1-69.8 | 457 | | NC ≠ PP | | ** |
| | (3)PP | 43.38 | 7.48 | 11.2-65.6 | 485 | | OF ≠ PP | | ** |
| DYNP | (1)NC | 52.16 | 10.06 | 30.0-83.0 | 130 | **.0000** | NC ≠ OF | * | |
| | (2)OF | 49.56 | 7.99 | 20.5-76.0 | 570 | | NC ≠ PP | | ** |
| | (3)PP | 44.97 | 10.38 | 8.0-76.0 | 476 | | OF ≠ PP | | ** |
| DYNNP | (1)NC | 49.48 | 9.64 | 26.8-73.0 | 130 | **.0000** | NC = OF | | |
| | (2)OF | 47.43 | 7.90 | 16.5-74.0 | 569 | | NC ≠ PP | | ** |
| | (3)PP | 41.66 | 9.76 | 12.5-67.5 | 477 | | OF ≠ PP | | ** |
| TRAILA | (1)NC | 24.03 | 7.12 | 12.0- 52.7 | 132 | **.0000** | NC ≠ OF | | ** |
| | (2)OF | 34.95 | 16.74 | 10.0-163.0 | 582 | | NC ≠ PP | | ** |
| | (3)PP | 38.03 | 37.31 | 11.4-627.0 | 491 | | OF = PP | | |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| TRAILB | (1)NC | 53.58 | 17.81 | 21.0-111.0 | 132 | .0000 | NC≠OF | ** |
| | (2)OF | 80.87 | 38.62 | 28.4-312.0 | 579 | | NC≠PP | ** |
| | (3)PP | 111.21 | 91.48 | 25.7-900.0 | 491 | | OF≠PP | ** |
| PURDP | (1)NC | 15.73 | 1.63 | 12.0-21.0 | 131 | .0000 | NC≠OF | * |
| | (2)OF | 15.15 | 2.16 | 5.0-21.0 | 492 | | NC≠PP | ** |
| | (3)PP | 13.7 | 2.42 | 4.0-20.0 | 490 | | OF≠PP | ** |
| PURDNP | (1)NC | 15.26 | 1.78 | 11.0-20.0 | 131 | .0000 | NC≠OF | * |
| | (2)OF | 14.67 | 2.07 | 3.0-20.0 | 493 | | NC≠PP | ** |
| | (3)PP | 12.68 | 2.26 | 5.0-19.0 | 491 | | OF≠PP | ** |
| PURDB | (1)NC | 12.79 | 1.41 | 9.0-16.0 | 131 | .0000 | NC≠OF | ** |
| | (2)OF | 11.95 | 1.92 | 4.0-24.0 | 492 | | NC≠PP | ** |
| | (3)PP | 10.24 | 2.14 | 4.0-17.0 | 490 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| PURDA | (1)NC | 39.21 | 5.37 | 22.0-56.0 | 131 | **.0000** | NC≠OF | ** |
| | (2)OF | 36.09 | 7.21 | 14.0-56.0 | 480 | | NC≠PP | ** |
| | (3)PP | 30.43 | 7.79 | 8.0-51.0 | 488 | | OF≠PP | ** |
| TACPERP | (1)NC | 295.61 | 124.72 | 99.0-808.0 | 131 | **.0000** | NC≠OF | ** |
| | (2)OF | 396.90 | 180.02 | 85.0-900.0 | 517 | | NC≠PP | ** |
| | (3)PP | 486.99 | 223.87 | 123.6-900.0 | 445 | | OF≠PP | ** |
| TACPERNP | (1)NC | 205.25 | 86.85 | 67.0-565.7 | 131 | **.0000** | NC≠OF | ** |
| | (2)OF | 290.52 | 162.71 | 69.0-900.0 | 515 | | NC≠PP | ** |
| | (3)PP | 396.70 | 224.47 | 37.7-900.0 | 445 | | OF≠PP | ** |
| TACPERB | (1)NC | 121.88 | 67.83 | 26.2-561.5 | 131 | **.0000** | NC≠OF | * |
| | (2)OF | 163.14 | 106.33 | 38.6-900.0 | 512 | | NC≠PP | ** |
| | (3)PP | 263.33 | 201.11 | 46.0-900.0 | 444 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| TACPERFM | (1)NC | 8.31 | 1.10 | 4.0-10.0 | 131 | **.0000** | NC≠OF | * |
| | (2)OF | 7.87 | 1.57 | 00.0-10.0 | 517 | | NC≠PP | ** |
| | (3)PP | 6.82 | 1.87 | 00.0-10.0 | 445 | | OF≠PP | ** |
| TACPERFL | (1)NC | 5.38 | 2.17 | 1.0- 9.0 | 131 | **.0000** | NC≠OF | * |
| | (2)OF | 4.76 | 2.63 | 00.0-10.0 | 516 | | NC≠PP | ** |
| | (3)PP | 3.28 | 2.34 | 00.0-10.0 | 445 | | OF≠PP | ** |
| TACFRP | (1)NC | 6.95 | 3.37 | 2.0-22.3 | 119 | **.0000** | NC=OF | |
| | (2)OF | 6.41 | 3.39 | 2.0-28.1 | 365 | | NC≠PP | ** |
| | (3)PP | 8.99 | 4.87 | 2.8-28.5 | 148 | | OF≠PP | ** |
| TACFRNP | (1)NC | 5.45 | 2.23 | 2.0- 14.2 | 119 | **.0000** | NC=OF | |
| | (2)OF | 5.35 | 3.99 | 1.0- 46.8 | 370 | | NC≠PP | ** |
| | (3)PP | 8.08 | 12.54 | 2.3-150.4 | 149 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| LJPERR | (1)NC | .26 | .56 | 00.0-3.0 | 123 | **.0000** | NC = OF | |
| | (2)OF | .35 | .65 | 00.0-4.0 | 373 | | NC ≠ PP | ** |
| | (3)PP | .56 | .89 | 00.0-4.0 | 456 | | OF ≠ PP | ** |
| LJPTIM | (1)NC | 35.19 | 21.12 | 6.5-126.0 | 123 | **.0000** | NC = OF | |
| | (2)OF | 30.77 | 18.06 | 7.5-146.0 | 372 | | NC ≠ PP | ** |
| | (3)PP | 47.32 | 31.74 | 8.1-238.2 | 455 | | OF ≠ PP | ** |
| LJNPERR | (1)NC | .10 | .37 | 00.0-2.0 | 123 | **.0000** | NC = OF | |
| | (2)OF | .20 | .48 | 00.0-3.0 | 374 | | NC ≠ PP | ** |
| | (3)PP | .45 | .76 | 00.0-3.0 | 456 | | OF ≠ PP | ** |
| LJNPTIM | (1)NC | 32.46 | 27.04 | 7.0-211.0 | 123 | **.0000** | NC = OF | |
| | (2)OF | 27.79 | 20.61 | 4.0-134.0 | 374 | | NC ≠ PP | ** |
| | (3)PP | 44.08 | 39.23 | 6.8-471.0 | 455 | | OF ≠ PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| LJBERR | (1)NC | .06 | .23 | 00.0-1.0 | 123 | **.0000** | NC = OF | |
| | (2)OF | .11 | .36 | 00.0-2.0 | 373 | | NC ≠ PP | ** |
| | (3)PP | .25 | .59 | 00.0-4.0 | 456 | | OF ≠ PP | ** |
| LJBTIM | (1)NC | 22.84 | 14.72 | 4.3- 85.0 | 123 | **.0000** | NC = OF | |
| | (2)OF | 22.80 | 15.23 | 3.9-137.2 | 374 | | NC ≠ PP | ** |
| | (3)PP | 36.87 | 28.41 | 5.3-274.8 | 455 | | OF ≠ PP | ** |
| FTIPNP | (1)NC | 1.85 | 2.10 | 00.0-10.0 | 130 | **.0000** | NC = OF | |
| | (2)OF | 1.66 | 2.11 | 00.0-15.0 | 368 | | NC ≠ PP | ** |
| | (3)PP | 2.90 | 3.19 | 00.0-16.0 | 401 | | OF ≠ PP | ** |
| FTIPNNP | (1)NC | 1.25 | 1.65 | 00.0- 9.0 | 130 | **.0000** | NC = OF | |
| | (2)OF | 1.08 | 1.75 | 00.0- 9.0 | 369 | | NC ≠ PP | ** |
| | (3)PP | 2.62 | 3.06 | 00.0-16.0 | 401 | | OF ≠ PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| FHANDR | (1)NC | .05 | .23 | 00.0- 1.0 | 128 | **.0013** | NC≠OF | * |
| | (2)OF | .38 | 1.22 | 00.0-14.0 | 372 | | NC≠PP | ** |
| | (3)PP | .49 | 1.18 | 00.0- 7.0 | 236 | | OF=PP | |
| FHANDL | (1)NC | .11 | .42 | 00.0- 3.0 | 128 | **.0004** | NC≠OF | * |
| | (2)OF | .45 | 1.16 | 00.0-13.0 | 372 | | NC≠PP | ** |
| | (3)PP | .62 | 1.44 | 00.0- 8.0 | 236 | | OF=PP | |
| FLOCPS | (1)NC | .49 | .86 | 00.0-4.0 | 130 | **.0000** | NC=OF | |
| | (2)OF | .77 | 1.25 | 00.0-8.0 | 367 | | NC≠PP | ** |
| | (3)PP | 1.04 | 1.63 | 00.0-9.0 | 487 | | OF≠PP | * |
| FLOCPD | (1)NC | 2.08 | 2.18 | 00.0-12.0 | 130 | **.0000** | NC≠OF | ** |
| | (2)OF | 3.37 | 3.07 | 00.0-20.0 | 367 | | NC≠PP | ** |
| | (3)PP | 4.28 | 3.69 | 00.0-21.0 | 487 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| FLOCNPS | (1)NC | .54 | .93 | 00.0- 4.0 | 130 | **.0000** | NC≠OF | ** |
| | (2)OF | 1.10 | 1.65 | 00.0-10.0 | 367 | | NC≠PP | ** |
| | (3)PP | 1.57 | 2.01 | 00.0-10.0 | 487 | | OF≠PP | ** |
| FLOCNPD | (1)NC | 2.65 | 2.63 | 00.0-16.0 | 130 | **.0000** | NC≠OF | ** |
| | (2)OF | 3.83 | 3.25 | 00.0-18.0 | 367 | | NC≠PP | ** |
| | (3)PP | 5.24 | 4.27 | 00.0-21.0 | 487 | | OF≠PP | ** |
| SDIGO | (1)NC | 68.05 | 11.56 | 47.0-104.0 | 107 | **.0000** | NC≠OF | ** |
| | (2)OF | 56.21 | 13.82 | 24.0-104.0 | 375 | | NC≠PP | ** |
| | (3)PP | 46.17 | 14.08 | 11.0- 97.0 | 153 | | OF≠PP | ** |
| SDIGW | (1)NC | 57.37 | 8.76 | 35.0-82.0 | 107 | **.0000** | NC≠OF | ** |
| | (2)OF | 47.26 | 10.58 | 17.0-85.0 | 376 | | NC≠PP | ** |
| | (3)PP | 39.44 | 12.15 | 9.0-69.0 | 153 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| APHAS | (1)NC | 1.01 | 1.11 | 00.0- 5.0 | 127 | **.0000** | NC = OF | |
| | (2)OF | 1.11 | 2.09 | 00.0-15.0 | 368 | | NC ≠ PP | ** |
| | (3)PP | 2.67 | 2.77 | 00.0-22.0 | 398 | | OF ≠ PP | ** |
| SSERR | (1)NC | 3.86 | 2.09 | 00.0-10.0 | 132 | **.0000** | NC ≠ OF | ** |
| | (2)OF | 5.82 | 4.07 | 00.0-34.0 | 569 | | NC ≠ PP | ** |
| | (3)PP | 6.87 | 5.21 | 00.0-37.0 | 485 | | OF ≠ PP | ** |
| SEASHR | (1)NC | 2.67 | 2.07 | 00.0- 9.0 | 132 | **.0000** | NC ≠ OF | ** |
| | (2)OF | 4.30 | 3.33 | 00.0-24.0 | 559 | | NC ≠ PP | ** |
| | (3)PP | 5.48 | 4.00 | 00.0-20.0 | 452 | | OF ≠ PP | ** |
| MFD | (1)NC | 44.44 | 1.10 | 40.0-45.0 | 129 | **.0000** | NC ≠ OF | ** |
| | (2)OF | 42.96 | 3.90 | 16.0-45.0 | 572 | | NC ≠ PP | ** |
| | (3)PP | 41.52 | 5.02 | 16.0-45.0 | 241 | | OF ≠ PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| SGEST1 | (1)NC | 74.25 | 20.79 | 29.4-120.0 | 130 | **.0000** | NC≠OF | ** |
| | (2)OF | 52.04 | 16.92 | 14.0-117.0 | 386 | | NC≠PP | ** |
| | (3)PP | 47.65 | 18.19 | 00.0- 98.0 | 146 | | OF≠PP | * |
| MINEST | (1)NC | 65.39 | 18.76 | 14.6-139.2 | 130 | **.0000** | NC≠OF | ** |
| | (2)OF | 54.22 | 21.22 | 7.5-156.1 | 385 | | NC≠PP | ** |
| | (3)PP | 52.10 | 23.71 | 6.0-176.7 | 145 | | OF=PP | |
| OWTOT | (1)NC | 15.15 | 4.07 | 6.7-29.3 | 132 | **.0000** | NC≠OF | ** |
| | (2)OF | 12.14 | 3.75 | 2.1-30.7 | 568 | | NC≠PP | ** |
| | (3)PP | 10.79 | 3.78 | 2.0-26.7 | 487 | | OF≠PP | ** |
| WWTOT | (1)NC | 13.78 | 3.09 | 7.3-21.7 | 104 | **.0000** | NC≠OF | ** |
| | (2)OF | 10.83 | 3.29 | 00.0-26.6 | 559 | | NC≠PP | ** |
| | (3)PP | 9.45 | 3.07 | 3.0-20.0 | 204 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| VLNTRI | (1)NC | 2.68 | 1.05 | 1.0-5.0 | 131 | **.0000** | NC≠OF | | ** |
| | (2)OF | 3.54 | 1.24 | 1.0-5.0 | 533 | | NC≠PP | | ** |
| | (3)PP | 4.27 | 1.10 | 1.0-5.0 | 474 | | OF≠PP | | ** |
| VLTOTE | (1)NC | 4.66 | 4.40 | 00.0-22.0 | 131 | **.0000** | NC≠OF | | ** |
| | (2)OF | 9.10 | 7.53 | 00.0-38.0 | 532 | | NC≠PP | | ** |
| | (3)PP | 16.63 | 10.94 | 00.0-40.0 | 474 | | OF≠PP | | ** |
| NVLNTRI | (1)NC | 1.89 | 1.05 | 1.0-5.0 | 132 | **.0000** | NC=OF | | |
| | (2)OF | 1.80 | 1.08 | 1.0-5.0 | 386 | | NC≠PP | | ** |
| | (3)PP | 2.34 | 1.40 | 1.0-5.0 | 470 | | OF≠PP | | ** |
| NVLE | (1)NC | 1.48 | 2.03 | 00.0- 9.0 | 132 | **.0000** | NC=OF | | |
| | (2)OF | 1.26 | 2.24 | 00.0-16.0 | 384 | | NC≠PP | | ** |
| | (3)PP | 2.54 | 3.65 | 00.0-18.0 | 470 | | OF≠PP | | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| NVLRNTRI | (1)NC | 1.52 | .95 | 1.0-5.0 | 132 | **.0000** | NC≠OF | | ** |
| | (2)OF | 1.99 | 1.38 | 1.0-5.0 | 386 | | NC≠PP | | ** |
| | (3)PP | 2.62 | 1.68 | 1.0-5.0 | 470 | | OF≠PP | | ** |
| NVLRE | (1)NC | .67 | 1.33 | 00.0- 6.0 | 132 | **.0000** | NC≠OF | * | |
| | (2)OF | 1.64 | 2.83 | 00.0-15.0 | 385 | | NC≠PP | | ** |
| | (3)PP | 3.50 | 4.66 | 00.0-20.0 | 470 | | OF≠PP | | ** |
| WCSSUB | (1)NC | 5.78 | .89 | 1.0-6.0 | 108 | **.0000** | NC≠OF | * | |
| | (2)OF | 5.20 | 1.77 | 00.0-6.0 | 574 | | NC≠PP | | ** |
| | (3)PP | 4.10 | 2.16 | 00.0-6.0 | 448 | | OF≠PP | | ** |
| WCSPER | (1)NC | 5.90 | 8.50 | 00.0-36.0 | 108 | **.0000** | NC≠OF | | ** |
| | (2)OF | 9.92 | 10.63 | 00.0-66.0 | 570 | | NC≠PP | | ** |
| | (3)PP | 15.15 | 13.33 | 00.0-67.0 | 448 | | OF≠PP | | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| WCSNPER | (1)NC | 6.01 | 5.79 | 00.0-37.0 | 108 | **.0000** | NC≠OF | | ** |
| | (2)OF | 9.82 | 8.93 | 00.0-52.0 | 571 | | NC≠PP | | ** |
| | (3)PP | 14.84 | 12.62 | 1.0-77.0 | 448 | | OF≠PP | | ** |
| WCSCOR | (1)NC | 9.70 | 14.34 | 00.0- 65.0 | 108 | **.0000** | NC≠OF | | ** |
| | (2)OF | 18.38 | 15.87 | 00.0- 80.0 | 571 | | NC≠PP | | ** |
| | (3)PP | 23.24 | 15.53 | 00.0-100.0 | 448 | | OF≠PP | | ** |
| WCSUNI | (1)NC | .97 | 2.72 | 00.0-18.0 | 108 | **.0000** | NC=OF | | |
| | (2)OF | 2.11 | 4.41 | 00.0-28.0 | 571 | | NC≠PP | | ** |
| | (3)PP | 3.21 | 5.57 | 00.0-43.0 | 448 | | OF≠PP | | ** |
| WCSTOT | (1)NC | 12.88 | 14.41 | 00.0- 55.0 | 108 | **.0000** | NC≠OF | | ** |
| | (2)OF | 21.94 | 18.45 | 00.0- 90.0 | 571 | | NC≠PP | | ** |
| | (3)PP | 33.21 | 20.91 | 2.0-106.0 | 448 | | OF≠PP | | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| HCAT1 | (1)NC | .04 | .19 | 00.0-1.0 | 129 | **.0000** | NC = OF | |
| | (2)OF | .06 | .26 | 00.0-2.0 | 575 | | NC ≠ PP | ** |
| | (3)PP | .18 | .54 | 00.0-5.0 | 467 | | OF ≠ PP | ** |
| HCAT2 | (1)NC | .47 | .60 | 00.0- 2.0 | 129 | **.0000** | NC = OF | |
| | (2)OF | .40 | .57 | 00.0- 4.0 | 574 | | NC ≠ PP | * |
| | (3)PP | .68 | 1.10 | 00.0-14.0 | 467 | | OF ≠ PP | ** |
| HCAT3 | (1)NC | 10.40 | 9.53 | 00.0-36.0 | 129 | **.0000** | NC ≠ OF | ** |
| | (2)OF | 14.56 | 10.93 | 00.0-36.0 | 572 | | NC ≠ PP | ** |
| | (3)PP | 18.10 | 11.73 | 00.0-36.0 | 467 | | OF ≠ PP | ** |
| HCAT4 | (1)NC | 4.77 | 5.02 | 00.0-30.0 | 129 | **.0000** | NC ≠ OF | ** |
| | (2)OF | 10.0 | 9.93 | 00.0-40.0 | 564 | | NC ≠ PP | ** |
| | (3)PP | 15.87 | 11.22 | 00.0-38.0 | 467 | | OF ≠ PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| HCAT5 | (1)NC | 9.90 | 6.26 | 1.0-27.0 | 129 | **.0000** | NC≠OF | ** |
| | (2)OF | 12.40 | 6.53 | 00.0-40.0 | 552 | | NC≠PP | ** |
| | (3)PP | 16.94 | 7.43 | 1.0-36.0 | 467 | | OF≠PP | ** |
| HCAT6 | (1)NC | 4.67 | 5.01 | 00.0-21.0 | 129 | **.0000** | NC≠OF | ** |
| | (2)OF | 7.26 | 5.81 | 00.0-38.0 | 549 | | NC≠PP | ** |
| | (3)PP | 11.58 | 8.06 | 00.0-36.0 | 467 | | OF≠PP | ** |
| HCAT7 | (1)NC | 2.70 | 2.04 | 00.0-10.0 | 129 | **.0000** | NC≠OF | ** |
| | (2)OF | 3.86 | 2.42 | 00.0-13.0 | 545 | | NC≠PP | ** |
| | (3)PP | 5.84 | 3.27 | 00.0-17.0 | 467 | | OF≠PP | ** |
| HCATCOR | (1)NC | 6.39 | 3.78 | 00.0-19.0 | 129 | **.0000** | NC≠OF | ** |
| | (2)OF | 8.09 | 4.36 | 00.0-24.0 | 570 | | NC≠PP | ** |
| | (3)PP | 11.58 | 5.56 | 1.0-28.0 | 467 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| HCATINC | (1)NC | 8.47 | 5.17 | 1.0-28.0 | 129 | **.0000** | NC≠OF | | ** |
| | (2)OF | 11.66 | 7.80 | 00.0-57.0 | 567 | | NC≠PP | | ** |
| | (3)PP | 17.02 | 10.38 | 2.0-75.0 | 467 | | OF≠PP | | ** |
| HCATERR | (1)NC | 32.93 | 17.84 | 9.0-106.0 | 129 | **.0000** | NC≠OF | | ** |
| | (2)OF | 51.09 | 27.09 | 7.0-130.0 | 582 | | NC≠PP | | ** |
| | (3)PP | 69.19 | 31.03 | 8.0-145.0 | 467 | | OF≠PP | | ** |

[1] rounded to 1 decimal point

* $p \leq 0.05$

** $p \leq 0.01$

TABLE 4.6a

*Level of Neuropsychological Test Impairment by Sample

| | NC GROUP | | | OF GROUP | | | PP GROUP | | |
|---|---|---|---|---|---|---|---|---|---|
| | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) |
| VARIABLE | <1SD** | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs |
| HCATERR | 13 | 6 | 4 | 47 | 32 | 27 | 69 | 57 | 51 |
| WCSTOT | 13 | 11 | 9 | 30 | 21 | 17 | 54 | 51 | 37 |
| WCSSUB | 7 | 6 | 6 | 19 | 18 | 18 | 50 | 48 | 46 |
| TRAILS B | 16 | 9 | 6 | 46 | 38 | 30 | 65 | 54 | 46 |
| TRAILS A | 17 | 7 | 2 | 47 | 39 | 31 | 49 | 40 | 32 |
| OWTOT | 11 | 4 | 1 | 55 | 22 | 9 | 54 | 35 | 18 |
| VLTOTE | 8 | 6 | 4 | 37 | 30 | 24 | 67 | 60 | 53 |
| SSERR | 9 | 6 | 2 | 32 | 23 | 17 | 40 | 32 | 24 |

| | NC GROUP | | | OF GROUP | | | PP GROUP | | |
|---|---|---|---|---|---|---|---|---|---|
| | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) |
| VARIABLE | <1SD** | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs |
| HCATERR | 13 | 6 | 4 | 47 | 32 | 27 | 69 | 57 | 51 |
| TACPERNP | 11 | 9 | 4 | 36 | 27 | 20 | 60 | 50 | 40 |
| CPM | 11 | 6 | 3 | 45 | 38 | 30 | 50 | 43 | 40 |
| MFD | 18 | 12 | 11 | 38 | 30 | 20 | 53 | 46 | 43 |
| PURDB | 20 | 16 | 8 | 45 | 32 | 22 | 80 | 65 | 55 |
| PURDA | 17 | 11 | 3 | 41 | 31 | 19 | 68 | 61 | 45 |
| SEASHR | 15 | 6 | 2 | 31 | 26 | 13 | 44 | 36 | 25 |
| SDIGW | 17 | 6 | 2 | 56 | 37 | 26 | 76 | 62 | 54 |
| SDIGO | 13 | 6 | 0 | 45 | 37 | 22 | 74 | 64 | 50 |
| SGEST1 | 18 | 9 | 2 | 57 | 40 | 11 | 67 | 47 | 21 |

*NC mean and SD forms basis for contrast

**Where necessary, interpolations were computed

Wechsler Variables

Tables 4.7 and 4.7a present the same statistics for Wechsler variables as was presented for neuropsychological variables above in Tables 4.6 and 4.6a, respectively. Inspection of Table 4.7 indicates that handedness, as measured by the ANNETT, did not differ across the NC, OF, and PP groups, although the OF group endorsed fewer right hand utilizations than the PP group, and also endorsed a greater use of both hands to perform a task than the NC group. However, differences were not large in absolute terms.

On the Wechsler scales, the performance relationship of PP<OF<NC was observed in all comparisons. Reference to Table 4.7a suggests that differences on Wechsler variables is, in general, greater than for neuropsychological variables. Consider, for example, that performance of the OF group was 1 *SD* below the NC group mean in 75%, 60%, and 78% of cases on Verbal IQ, Performance IQ, and Full Scale IQ, respectively. Indeed, 45% scored 2 *SDs* below the NC group Full Scale IQ mean reflecting exceptionally poor relative performance on this measure of intelligence. Individual scale scores were consistent with composite indices in reflecting poor performance among the OF and PP groups, although on the Picture Completion subtest, which would appear to involve relatively automatic perceptual processing, at least for the easier items, performance in these two groups was relatively better.

Overall, Verbal IQ was higher than Performance IQ by 3.85 and 2.05 points in the NC and PP groups, respectively, and these two groups were

not significantly ($P \geq .05$) different (VPDIF). The OF group had a higher Performance IQ than Verbal IQ by 1.51 points and this was significantly ($P \leq .01$) different from the NC and PP groups.

In sum, very large and statistically significant differences were observed on the Wechsler scales. The best performance was by the NC group, the poorest by the PP group, and the performance of the OF group was intermediate. In Section VIII, an attempt to further differentiate Wechsler scale performance will be made using Modal Profile Analysis. Next, in Section VII, the factor structure of the neuropsychological variables is the focus.

TABLE 4.7

WAIS-R & Related Variables

Descriptive Statistics For Normal Controls, Offenders, & Psychiatric Patients

ANOVA

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| AGE | (1)NC | 27.92 | 7.03 | 18.0-44.0 | 132 | .2997 | NC = OF | | |
| | (2)OF | 28.78 | 6.32 | 18.0-44.0 | 584 | | NC = PP | | |
| | (3)PP | 28.94 | 7.09 | 18.0-44.0 | 494 | | OF = PP | | |
| ANNETT | (1)NC | 10.13 | 3.21 | 00.0-12.0 | 128 | .7010 | NC = OF | | |
| | (2)OF | 10.36 | 3.02 | 00.0-12.0 | 569 | | NC = PP | | |
| | (3)PP | 10.39 | 3.06 | 00.0-12.0 | 480 | | OF = PP | | |
| ANN-R | (1)NC | 9.75 | 3.40 | 00.0-12.0 | 128 | **.0007** | NC = OF | | |
| | (2)OF | 9.17 | 3.54 | 00.0-12.0 | 569 | | NC = PP | | |
| | (3)PP | 9.96 | 3.23 | 00.0-12.0 | 480 | | OF ≠ PP | | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| ANN-L | (1)NC | 1.87 | 3.21 | 00.0-12.0 | 128 | .7010 | NC = OF | | |
| | (2)OF | 1.64 | 3.02 | 00.0-12.0 | 569 | | NC = PP | | |
| | (3)PP | 1.61 | 3.06 | 00.0-12.0 | 480 | | OF = PP | | |
| ANN-B | (1)NC | .38 | 1.00 | 00.0- 6.0 | 128 | **.0000** | NC ≠ OF | | * * |
| | (2)OF | 1.19 | 2.06 | 00.0-11.0 | 569 | | NC = PP | | |
| | (3)PP | .42 | 1.11 | 00.0- 9.0 | 480 | | OF ≠ PP | | * * |
| VIQ | (1)NC | 114.25 | 11.62 | 87.0-146.0 | 109 | **.0000** | NC ≠ OF | | * * |
| | (2)OF | 94.13 | 13.15 | 59.0-147.0 | 584 | | NC ≠ PP | | * * |
| | (3)PP | 91.04 | 13.66 | 54.0-131.0 | 448 | | OF ≠ PP | | * * |
| PIQ | (1)NC | 110.29 | 12.06 | 84.0-137.0 | 109 | **.0000** | NC ≠ OF | | * * |
| | (2)OF | 95.64 | 13.48 | 57.0-140.0 | 584 | | NC ≠ PP | | * * |
| | (3)PP | 89.01 | 14.34 | 47.0-138.0 | 447 | | OF ≠ PP | | * * |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| VPDIF | (1)NC | 3.85 | 13.27 | -30.0-41.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | -1.51 | 11.86 | -43.0-40.0 | 584 | | NC=PP | |
| | (3)PP | 2.05 | 12.40 | -35.0-54.0 | 446 | | OF≠PP | ** |
| FIQ | (1)NC | 113.99 | 11.34 | 86.0-141.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 94.11 | 12.92 | 62.0-142.0 | 584 | | NC≠PP | ** |
| | (3)PP | 89.21 | 13.27 | 53.0-132.0 | 446 | | OF≠PP | ** |
| MINF | (1)NC | 7.94 | 1.83 | 1.0-12.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 5.76 | 1.94 | 1.0-11.0 | 581 | | NC≠PP | ** |
| | (3)PP | 5.01 | 2.0 | 1.0-11.0 | 441 | | OF≠PP | ** |
| MAXF | (1)NC | 15.50 | 1.69 | 11.0-19.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 12.85 | 2.47 | 5.0-19.0 | 581 | | NC≠PP | ** |
| | (3)PP | 12.04 | 2.70 | 4.0-19.0 | 441 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * | Sig ** |
|---|---|---|---|---|---|---|---|---|---|
| RANF | (1)NC | 7.57 | 2.13 | 3.0-17.0 | 109 | .0536 | NC = OF | | |
| | (2)OF | 7.08 | 2.10 | 2.0-14.0 | 581 | | NC = PP | | |
| | (3)PP | 7.03 | 2.14 | 2.0-14.0 | 441 | | OF = PP | | |
| IN | (1)NC | 12.0 | 1.96 | 7.0-16.0 | 109 | **.0000** | NC ≠ OF | | ** |
| | (2)OF | 8.71 | 3.04 | 1.0-18.0 | 584 | | NC ≠ PP | | ** |
| | (3)PP | 8.56 | 3.01 | 1.0-16.0 | 448 | | OF = PP | | |
| DSP | (1)NC | 11.83 | 2.81 | 6.0-19.0 | 109 | **.0000** | NC ≠ OF | | ** |
| | (2)OF | 9.71 | 2.77 | 2.0-18.0 | 584 | | NC ≠ PP | | ** |
| | (3)PP | 8.49 | 2.65 | 1.0-18.0 | 447 | | OF ≠ PP | | ** |
| VO | (1)NC | 12.17 | 2.52 | 6.0-18.0 | 109 | **.0000** | NC ≠ OF | | ** |
| | (2)OF | 8.54 | 2.70 | 1.0-17.0 | 582 | | NC ≠ PP | | ** |
| | (3)PP | 8.58 | 2.86 | 1.0-18.0 | 448 | | OF = PP | | |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| AR | (1)NC | 12.54 | 2.52 | 6.0-17.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 9.22 | 2.80 | 2.0-17.0 | 584 | | NC≠PP | ** |
| | (3)PP | 8.65 | 3.03 | 1.0-17.0 | 448 | | OF≠PP | ** |
| CO | (1)NC | 11.89 | 2.73 | 5.0-18.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 9.27 | 3.09 | 3.0-16.0 | 584 | | NC≠PP | ** |
| | (3)PP | 7.95 | 2.94 | 1.0-16.0 | 448 | | OF≠PP | ** |
| SI | (1)NC | 11.89 | 3.03 | 3.0-18.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 8.94 | 3.01 | 1.0-18.0 | 584 | | NC≠PP | ** |
| | (3)PP | 8.44 | 3.48 | 1.0-19.0 | 447 | | OF≠PP | * |
| MINV | (1)NC | 9.24 | 2.05 | 3.0-15.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 6.63 | 2.35 | 1.0-14.0 | 581 | | NC≠PP | ** |
| | (3)PP | 6.05 | 2.39 | 1.0-13.0 | 441 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| MAXV | (1)NC | 14.88 | 1.98 | 9.0-19.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 11.60 | 2.73 | 4.0-18.0 | 581 | | NC≠PP | ** |
| | (3)PP | 11.03 | 2.85 | 3.0-19.0 | 441 | | OF≠PP | |
| RANV | (1)NC | 5.64 | 2.11 | 1.0-13.0 | 109 | .0054 | NC≠OF | ** |
| | (2)OF | 4.96 | 2.06 | 1.0-12.0 | 581 | | NC≠PP | * |
| | (3)PP | 4.99 | 2.02 | 1.0-12.0 | 441 | | OF=PP | |
| PC | (1)NC | 10.82 | 2.68 | 2.0-18.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 9.55 | 2.91 | 1.0-18.0 | 584 | | NC≠PP | ** |
| | (3)PP | 8.17 | 2.91 | 1.0-17.0 | 447 | | OF≠PP | ** |
| PA | (1)NC | 11.32 | 2.57 | 5.0-18.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 9.43 | 2.85 | 1.0-19.0 | 584 | | NC≠PP | ** |
| | (3)PP | 8.25 | 3.14 | 1.0-17.0 | 447 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| BD | (1)NC | 12.10 | 2.49 | 6.0-17.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 10.03 | 2.65 | 3.0-17.0 | 584 | | NC≠PP | ** |
| | (3)PP | 9.24 | 2.94 | 1.0-16.0 | 446 | | OF≠PP | ** |
| OA | (1)NC | 11.30 | 2.82 | 1.0-18.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 9.45 | 2.84 | 1.0-17.0 | 583 | | NC≠PP | ** |
| | (3)PP | 8.57 | 2.90 | 1.0-18.0 | 446 | | OF≠PP | ** |
| DSY | (1)NC | 10.88 | 2.70 | 5.0-17.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 7.77 | 2.28 | 2.0-17.0 | 584 | | NC≠PP | ** |
| | (3)PP | 6.71 | 2.52 | 1.0-19.0 | 445 | | OF≠PP | ** |
| MINP | (1)NC | 8.58 | 1.91 | 1.0-13.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 6.63 | 1.91 | 1.0-13.0 | 581 | | NC≠PP | ** |
| | (3)PP | 5.62 | 2.02 | 1.0-13.0 | 441 | | OF≠PP | ** |

| Variable | Group | Mean | SD | Range[1] | N | ANOVA p-value | Scheffé contrast | Sig * ** |
|---|---|---|---|---|---|---|---|---|
| MAXP | (1)NC | 14.22 | 1.89 | 10.0-18.0 | 109 | **.0000** | NC≠OF | ** |
| | (2)OF | 11.83 | 2.57 | 5.0-19.0 | 581 | | NC≠PP | ** |
| | (3)PP | 10.75 | 2.82 | 2.0-19.0 | 441 | | OF≠PP | ** |
| RANP | (1)NC | 5.64 | 2.28 | 1.0-14.0 | 109 | .0843 | NC=OF | |
| | (2)OF | 5.20 | 2.13 | 1.0-13.0 | 581 | | NC=PP | |
| | (3)PP | 5.13 | 2.16 | 1.0-13.0 | 441 | | OF=PP | |

[1] rounded to 1 decimal point

* $p \leq .05$

** $p \leq 0.01$

TABLE 4.7a

*Level of Impairment on Wechsler Scales by Sample

| | NC GROUP | | | OF GROUP | | | PP GROUP | | |
|---|---|---|---|---|---|---|---|---|---|
| | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) |
| VARIABLE | <1SD** | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs |
| VIQ | 16 | 8 | 4 | 75 | 64 | 46 | 79 | 69 | 54 |
| PIQ | 19 | 7 | 3 | 60 | 42 | 26 | 77 | 60 | 42 |
| FIQ | 19 | 5 | 2 | 78 | 63 | 45 | 85 | 71 | 59 |
| VPDIF | 13 | 8 | 4 | 29 | 12 | 4 | 16 | 8 | 3 |
| IN | 23 | 9 | 6 | 71 | 58 | 50 | 74 | 58 | 41 |
| DSP | 20 | 9 | 2 | 49 | 29 | 11 | 67 | 45 | 24 |
| VO | 21 | 8 | 1 | 76 | 58 | 24 | 71 | 55 | 38 |
| AR | 20 | 10 | 6 | 67 | 51 | 28 | 73 | 61 | 47 |

| | NC GROUP | | | OF GROUP | | | PP GROUP | | |
|---|---|---|---|---|---|---|---|---|---|
| | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) | (%) |
| VARIABLE | <1SD** | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs | <1SD | <1.5SDs | <2SDs |
| CO | 22 | 11 | 2 | 58 | 43 | 23 | 72 | 57 | 38 |
| SI | 18.3 | 9 | 4 | 51 | 34 | 17 | 59 | 44 | 30 |
| PC | 13 | 7 | 5 | 33 | 19 | 13 | 53 | 35 | 27 |
| PA | 22 | 9 | 1 | 49 | 30 | 14 | 65 | 51 | 32 |
| BD | 23 | 10 | 3 | 54 | 35 | 16 | 61 | 47 | 31 |
| OA | 16 | 9 | 3 | 44 | 24 | 13 | 53 | 35 | 20 |
| DSY | 23 | 10 | 1 | 63 | 36 | 25 | 86 | 76 | 45 |

*NC mean and SD forms basis for contrast

**Where necessary, interpolations were computed

## SECTION VII FACTOR ANALYSIS

Factor analysis has traditionally (e.g., Thurstone, 1947) been advocated as a method of explicating the basic or major underlying constructs within a domain in terms of their empirical referents. According to this view, a sample of tests representative of a domain, e.g., neuropsychological function, can be reduced to a much smaller number of empirically interpretable tests factors. Overall and Klett (1972, p. 90) noted that if indeed a few common traits operate to produce the manifest variance among an array of tests, factor analysis should reveal: (a) relative parsimony in the description of the elemental variance, (b) relative independence of the components of variance, and (c) psychological interpretability of the variance.

In the area of clinical neuropsychology most traditional approaches have operated within a clinical model supported by bivariate research, e.g., attaching particular significance to test-lesion location established through clinical correlation and bivariate contrasts. For instance, the Reitan and Wolfson (1993) model of clinical interpretation (cf. Jarvis & Barth, 1994) examines: (a) level of performance on tests within a set battery; (b) patterns of performance, but from a clinical integrative, not statistical perspective; (c) right-left differences, and; (d) pathognomonic signs of brain damage. Other approaches, e.g., Milner (1963a,b, 1971) have extensively examined particular brain-behavior relationships correlating test performance with lesion parameters. The Lurian tradition has been less empirically driven and

focuses primarily on the integration of clinical theory and clinical observation assisted by custom or individualized measurements.

Recent theoretical perspectives on brain-behavior relations have focused more on interactive brain systems and developmental considerations, and accorded a primary role to the prefrontal cortex (cf. Diamond & Goldman-Rakic, 1989; Fuster, 1993; Tucker & Derryberry, 1992). Within this evolving clinical and experimental neuropsychology, one of the main purposes of conducting factor analyses is to determine if these recent conceptualizations of brain function will be reflected by empirical test factors. More specifically, will a factor analysis of selected neuropsychological tests, across the three samples (NC, OF, and PP) examined in this study, yield an interpretable invariant factor structure? Among the first factor analytic studies of broad scope seeking to establish relationships between neuropsychological factors and brain damage is the classic study of Royce et al. (1976). Since then several studies, but with more specific hypotheses than the current one, have been published, e.g.: a) Leonberger, Nicks, Goldfader, and Munz (1991) have examined relationships between the Wechsler Memory Scale-Revised and the Halstead-Reitan Neuropsychological Battery in a sample of neuropsychiatric patients; b) Fowler, Zillmer, and Neuman (1988) examined the Halstead-Reitan Battery in a sample of neuropsychological psychiatric patients; c) Moehle, Rasmussen, and Fitzhugh-Bell (1990) examined the factor structure among a large battery of neuropsychological and intelligence variables in a sample of 1,376

neurological patients; d) Ernst, Warner, Hochberg, and Townes (1988) contrasted the WAIS and WAIS-R with the Halstead-Reitan Battery; e) Corrigan and Hinkelday (1988) addressed similar issues among patients in rehabilitation; and f) Swiercinsky and Howard (1982) conducted a series of factor analytic studies concerning a broad range of variables in neurological populations.

The present study is distinct from most previous neuropsychological factor analytic studies in that it focuses on cognitive and perceptual variables and de-emphasizes simple motor, sensory, and Wechsler intelligence variables. Further, it addresses the issues of factorial invariance across three sample groups; normal controls, serious offenders, and psychiatric patients.

Determining Variable X Subject Matrix

A prior condition for conducting a factor analyses followed by Modal Profile Analyses is that observations (data points) for all variables be available for all subjects included in the analyses. Accordingly, the first step in these analyses included developing a variables by subjects matrix with no missing data, or estimating missing data where only minimal data was absent.

Criteria for determining the composition of the variables to be included in the factor analysis were facilitated by the plan to also compute extension loadings for variables not included in the factor space (Dwyer, 1937). This approach permits additional guidance for factor interpretation while maximizing the overall number of subjects included in the analysis. An

example includes Oral and Written Word Fluency where 487 PP were administered the Oral Word Fluency test, but only 204 were administered the written form of this test. Thus, to maximize N and minimize the need to estimate missing data, the Written Word Fluency measure was excluded from the factor analysis. However, extension loadings for the Written Word Fluency test were computed. Secondly, strictly motor measures, such as the Dynamometer test, were excluded, given the focus here on perceptual/cognitive and complex motor/sensory tasks. Other variables excluded from the analysis included variables that together formed linear composites, e.g., sub-measures of the WCST were included, but not the Total Error score.

This procedure resulted in 28 variables being retained for the factor analyses (see Table 4.8).

The next procedure was to exclude any subject from any group who did not have complete WAIS-R data or otherwise had <95% complete data on the 28 variables selected for inclusion in the factor analysis. As a result, 94 subjects from the NC group, 519 subjects from the OF group and 397 subjects from the PP group remained in the composite sample for a combined total of 1,010 cases. Percentages with <5% missing data were 2.1, 31.4, and 22.7 percent respectively for the NC, OF, and PP groups. Across all groups, percentage of data which had to be estimated is reported in Table 4.8 below.

TABLE 4.8

Sample Size & Overall Percent Missing Data For

Variables Included in the Factor Analyses

| | Sample Sizes | | | Percent Missing Data |
|---|---|---|---|---|
| | NC | OF | PP | Overall |
| CPM | 94 | 449 | 378 | 8.8 |
| TRAILA | 94 | 519 | 396 | 0.1 |
| TRAILB | 94 | 516 | 396 | 0.4 |
| PURDP | 94 | 449 | 394 | 7.2 |
| PURDNP | 94 | 450 | 395 | 7.0 |
| PURDB | 94 | 450 | 394 | 7.1 |
| PURDA | 94 | 439 | 393 | 8.3 |
| TACPERP | 93 | 479 | 357 | 8.0 |
| TACPERNP | 93 | 480 | 357 | 7.9 |
| TACPERB | 93 | 478 | 356 | 8.2 |
| TACPERFM | 93 | 481 | 357 | 7.8 |
| TACPERFL | 93 | 480 | 357 | 7.9 |
| SSERR | 94 | 516 | 392 | 0.8 |
| SEASHR | 94 | 508 | 357 | 5.0 |
| OWTOT | 94 | 516 | 390 | 1.0 |
| VLTOTE | 93 | 485 | 386 | 4.6 |
| WCSSUB | 94 | 519 | 379 | 0.0 |

| | Sample Sizes | | | Percent Missing Data |
|---|---|---|---|---|
| | NC | OF | PP | Overall |
| WCSPER | 94 | 519 | 379 | 0.0 |
| WCSNPER | 94 | 519 | 379 | 0.0 |
| WCSCOR | 94 | 519 | 379 | 0.0 |
| WCSUNI | 94 | 519 | 379 | 0.0 |
| HCAT3 | 94 | 519 | 379 | 0.0 |
| HCAT4 | 94 | 519 | 379 | 0.0 |
| HCAT5 | 94 | 519 | 379 | 0.0 |
| HCAT6 | 94 | 519 | 379 | 0.0 |
| HCAT7 | 94 | 519 | 379 | 0.0 |

Estimating Missing Data

As noted in Table 4.8, overall missing data ranged from 8.8% for CPM to 0.0% for WSCT and HCT sub-indices. Missing data was estimated using BMDP statistical software program BMDPAM - Description and Estimation of Missing Data (1990). Using this program, a new variable by subject matrix was computed, including estimated data and the variable list was redesignated by adding the prefix N to the original variable acronym, e.g., CPM was renamed NCPM, which included the estimated data for all previously missing observations. In sum, data sets of 94 (NC), 519 (OF), and 379 (PP) subjects with complete or estimated data on 28 variables were prepared for the factor analytic studies presented below. Visual inspection of means, *SDs,* correlations, and factor/extension loading differences

between original variables and newly created variables which included estimated data, indicated only minimal changes resulted from the estimation procedure. For instance, extension loadings of the CPM variable and factor loadings of NCPM (which includes estimated data) were identical when rounded to 2 decimal points in the combined factor analysis.

Factor Analysis

The 28 variables listed in Table 4.8 were retained for factor analysis. Principal Components factoring followed by Varimax rotation (Kaiser, 1958) were completed for each group (NC, OP, and PP) and with the combined group containing all 1,010 subjects. Velicer's (1976) criterion favored a 4 factor solution, while Cattell's (1966) and Cattell and Vogelman's (1967) Scree Tests suggested a 4 or 5 factor solution for all samples including the combined sample. Reddon (1984) has noted Velicer's (1976) criterion to be somewhat conservative. An interpretability criterion suggested that both 4 and 5 varimax rotated factors solutions to be both highly interpretable. Eigenvalues for the combined 5 factor solution, selected for interpretation, are listed in Table 4.9 below.

TABLE 4.9

Eigenvalues for Combined 5 Factor Solution

(SPSS1PC + V5.0, 1991)

| Factor | Eigenvalue | Percent of Variance | Cummulative Percent |
|---|---|---|---|
| 1 | 9.15 | 35.2 | 35.2 |
| 2 | 1.99 | 7.7 | 42.9 |
| 3 | 1.56 | 6.0 | 48.9 |
| 4 | 1.35 | 5.2 | 54.1 |
| 5 | 1.27 | 4.9 | 58.9 |

Factor Solution Congruencies

Employing Reddon's (1994) Procrustes program, the factor structure of the combined varimax solution was compared with that of the separate NC, OP, and PP solutions. Each individual (NC, OP, and PP) varimax orthogonal solution was rotated to the combined varimax orthogonal solution. This resulted in high levels of congruence across samples. Congruence coefficients are reported in Table 4.10 below.

TABLE 4.10

Congruence Coefficients

| Factor | Samples | | | | | |
|---|---|---|---|---|---|---|
| | NC | Pvalue | OF | Pvalue | PP | Pvalue |
| I | .848 | .0001 | .981 | .0001 | .996 | .0001 |
| II | .884 | .0001 | .986 | .0001 | .992 | .0001 |
| III | .869 | .0001 | .965 | .0001 | .991 | .0001 |
| IV | .607 | .0127 | .879 | .0001 | .984 | .0001 |
| V | .789 | .0001 | .883 | .0001 | .965 | .0001 |

Note: P values were obtained from 10,000 randon permutations of rows of the hypothesis matrix (i.e., the varimax solution of the combined sample).

The factor structure of the 3 sub-samples (NC, OF, and PP) were judged to be equivalent to the combined sample. The NC group appears to fit marginally less well, attributable likely to proportionately smaller sample size and also relatively restricted variance on some measures. For the purposes here and further analyses, the combined solution will be considered the definitive solution.

Factor Identification

Five Varimax factors were selected for the combined sample for further analysis and interpretation. They were labelled as follows:

I Sensory Motor Spatial Perceptual Organization;

II Nonverbal Perceptual Reasoning and Abstraction;

III Fine Temporal Perceptual Motor Speed;

IV Dynamic Verbal Processing; and

V WCST Perseveration.

Factor loadings and extension loadings on these five factors are presented in Table 4.11 below. Loadings on Factor III were reflected in this and subsequent analyses to ensure that higher positive factor scores uniformly reflect poorer performance.

TABLE 4.11

Five Factor Combined Varimax Solution & Extension Loadings

| VARIABLE | FACTOR I | FACTOR II | FACTOR III | FACTOR IV | FACTOR V |
|---|---|---|---|---|---|
| NTACPERB | **.77*** | .19 | .27 | .15 | .11 |
| NTACPERNP | **.76** | .20 | .27 | .12 | .09 |
| NTACPERP | **.70** | .16 | .24 | .12 | .09 |
| NTACPERFM | **-.64** | -.20 | -.17 | -.20 | -.13 |
| NTACPERFL | **-.57** | -.28 | -.16 | -.04 | -.19 |
| NTRAILB | **.44** | .25 | .21 | **-.40** | .03 |
| NTRAILA | **.33** | .16 | .12 | .28 | -.05 |
| NHCAT7 | .23 | **.80** | .18 | .16 | .10 |
| NHCAT6 | .26 | **.75** | .17 | .11 | .11 |
| NHCAT5 | .26 | **.73** | .11 | .06 | .12 |
| NHCAT4 | .21 | **.64** | .10 | .23 | .10 |
| NHCAT3 | .05 | **.57** | .13 | .12 | -.01 |
| NPURDB | -.23 | -.17 | **-.84** | -.13 | -.06 |
| NPURDNP | -.23 | -.16 | **-.84** | -.07 | -.07 |
| NPURDP | -.24 | -.17 | **-.79** | -.10 | -.07 |
| NPURDA | **-.37** | -.24 | **-.68** | -.20 | -.09 |
| NSEASHR | .28 | .15 | .02 | **.62** | .04 |
| NSSERR | **.32** | .07 | -.03 | **.60** | -.09 |
| NWCSNPER | .01 | .23 | .18 | **-.57** | **.48** |

| VARIABLE | FACTOR I | FACTOR II | FACTOR III | FACTOR IV | FACTOR V |
|---|---|---|---|---|---|
| NWCSSUB | .04 | -.29 | -.24 | **-.55** | **-.41** |
| NWCSUNI | -.21 | .10 | .14 | **.54** | **.33** |
| NOWTOT | -.28 | -.02 | **-.36** | **-.48** | .01 |
| NCPM | **.37** | **.39** | .08 | **.46** | .13 |
| NVLTOTE | **.40** | .29 | .27 | **.39** | .14 |
| NWCSCOR | .15 | .08 | .02 | .14 | **.85** |
| NWCSPER | .26 | .10 | .08 | -.00 | **.78** |
| EXTENSION LOADINGS Neuropsychological Measures | | | | | |
| FTAPP | -.19 | -.16 | -.26 | -.20 | -.07 |
| FTAPNP | -.17 | -.11 | -.25 | -.21 | -.09 |
| DYNP | -.25 | -.15 | -.17 | -.17 | -.04 |
| DYNNP | -.27 | -.16 | -.21 | -.15 | -.04 |
| LJPTIM | **.33** | .08 | .18 | .04 | .05 |
| LJNPERR | **.32** | .18 | .16 | .19 | .05 |
| LJNPTIM | **.33** | .12 | .19 | .09 | .05 |
| LJBERR | .22 | .22 | .14 | .21 | .01 |
| LJBTIM | **.42** | .20 | .24 | .11 | .08 |
| FTIPNP | **.30** | .21 | .10 | .26 | -.02 |
| FTIPNNP | **.33** | .22 | .16 | .29 | -.00 |
| FHANDR | .20 | .15 | .13 | .10 | .02 |
| FHANDL | .22 | .13 | .15 | .14 | .02 |

| VARIABLE | FACTOR I | FACTOR II | FACTOR III | FACTOR IV | FACTOR V |
|---|---|---|---|---|---|
| FLOCPS | .26 | .20 | .05 | .28 | -.00 |
| FLOCPD | **.35** | .22 | .13 | **.35** | .04 |
| FLOCNPS | .28 | .20 | .10 | .27 | .03 |
| FLOCNPD | **.35** | .25 | .14 | **.31** | .08 |
| SDIGO | **-.39** | -.22 | **-.34** | -.28 | -.17 |
| SDIGW | **-.37** | -.25 | **-.37** | **-.31** | -.17 |
| APHAS | .28 | .22 | .18 | **.36** | .05 |
| WWTOT | -.29 | -.09 | **-.36** | -.41 | .00 |
| VLNTRI | .25 | .24 | .19 | .28 | .13 |
| NVLNTRI | .27 | .24 | .16 | .14 | .03 |
| NVLE | **.31** | .24 | .19 | .20 | .03 |
| NVLRNTRI | **.40** | .27 | .18 | .15 | .08 |
| NVLRE | **.42** | .29 | .22 | .23 | .06 |
| WCSTOT | .10 | .22 | .18 | **.42** | **.79** |
| HCATCOR | .24 | **.70** | .15 | .08 | .08 |
| HCATINC | .15 | **.71** | .11 | .13 | .06 |
| HCATERR | .25 | **.89** | .16 | .19 | .11 |
| MFD | **-.42** | -.24 | -.15 | -.20 | -.08 |
| SGEST1 | -.24 | -.11 | **-.32** | -.23 | -.16 |
| MINEST | -.13 | -.12 | -.05 | -.16 | -.07 |
| AGE | .11 | .12 | .08 | .03 | .02 |

| VARIABLE | FACTOR I | FACTOR II | FACTOR III | FACTOR IV | FACTOR V |
|---|---|---|---|---|---|
| ANNETT | .02 | .01 | -.03 | -.01 | .02 |
| ANN-R | .06 | .04 | .04 | -.01 | .02 |
| ANN-L | -.02 | -.01 | .03 | .01 | -.02 |
| ANN-B | -.08 | -.07 | -.14 | .01 | .01 |
| EXTENSION LOADINGS Wechsler Variables | | | | | |
| VIQ | -.25 | **-.31** | -.15 | **-.40** | -.14 |
| PIQ | **-.38** | **-.35** | -.27 | **-.31** | -.16 |
| VPDIF | .17 | .06 | .15 | -.09 | .03 |
| FIQ | **-.33** | **-.36** | -.22 | **-.38** | -.17 |
| MINF | **-.32** | **-.31** | -.22 | **-.35** | -.14 |
| MAXF | **-.34** | **-.34** | -.19 | **-.39** | -.12 |
| RANF | -.11 | -.13 | -.03 | -.14 | -.01 |
| IN | -.15 | -.23 | -.09 | **-.30** | -.07 |
| DSP | -.24 | -.20 | -.14 | **-.40** | -.09 |
| VO | -.17 | -.22 | -.07 | **-.38** | -.11 |
| AR | -.27 | **-.31** | -.15 | -.30 | -.12 |
| CO | -.19 | -.28 | -.18 | -.29 | -.14 |
| SI | -.17 | -.26 | -.10 | **-.34** | -.13 |
| MINV | -.24 | -.28 | -.13 | **-.39** | -.13 |
| MAXV | -.23 | -.28 | -.13 | **-.40** | -.11 |
| RANV | -.04 | -.07 | -.02 | -.11 | -.00 |

| VARIABLE | FACTOR I | FACTOR II | FACTOR III | FACTOR IV | FACTOR V |
|---|---|---|---|---|---|
| PC | -.25 | -.28 | -.20 | **-.30** | -.12 |
| PA | -.29 | **-.30** | -.17 | -.26 | -.14 |
| BD | **-.42** | **-.37** | -.18 | -.21 | -.12 |
| OA | **-.36** | -.25 | -.19 | -.21 | -.12 |
| DSY | -.26 | -.23 | **-.37** | -.26 | -.15 |
| MINP | **-.34** | **-.31** | -.29 | **-.30** | -.15 |
| MAXP | **-.40** | **-.35** | -.24 | -.29 | -.14 |
| RANP | -.18 | -.15 | -.02 | -.09 | -.03 |

* Loadings ≥.30 reported in bold type

Factor Interpretation

The five factor solution is interpreted below. For each factor, interpretation is followed by a table which presents loadings and extension loadings ≥.30.

Factor 1 Sensory Motor Spatial Perceptual Organization

This first factor appears to be characterized by the ability to organize the relevant aspects of a perceptual field in relation to both sensory and motor behavior. Originally, this factor was the first factor to appear in the Aftanas and Royce (1969) analysis in a normal population and the Royce, Yeudall, and Bock (1976) study of brain damaged individuals. These authors labelled this factor perceptual organization and the specific loadings in that analysis are highly similar to the present factor loading structure, including major

contributions from the Tactual Performance Test, Trails tests, WAIS-R Block Design and Object Assembly subtests, as well as the Assembly measure from the Purdue Peg Board Test. In the Royce et al. (1976) study, sensory perceptual measures also loaded on their FACTOR VII. Specific sensory resolution measures are highly represented here in terms of the neuro-extension loadings and may have been identified as a separate factor had they been selected for inclusion in the present analysis. Of interest, FACTOR I and FACTOR VII of the Royce et al. (1976) study formed the highest salients on their 1st second order factor.

More recent factor analyses have also identified a large perceptual organization factor, e.g., in the Moehle et al. (1990) study, the Tactual Performance Test had the highest loading on their second factor and it was also the test with the highest loadings on the second factor in the Swiercinsky and Howard (1982) study. Further, the Tactual Performance Test also loaded highest on Factor I in Leonberger et al. (1991) factor analytic study. In the Corrigan and Hinkeldey (1988) study, the Tactual Performance Test basically defined the second factor in their first sample and the first factor in their second sample. In the present analysis, Wechsler extension loadings from PIQ, FIQ, BD, and OA, while modest, are clearly consistent with this major factor, given the common emphasis on sensory perceptual motor organization. In his review of studies of brain damaged

populations, Royce et al. (1976) found this factor to correlate with bilateral damage which was greater for regions of the right cerebral hemisphere. The relation of this factor to right hemisphere damage is also consistent with the evidence that on several visuo-spatial and tactual-spatial measures loading on this factor, the performance of neurological patients with damage localized to the right hemisphere is particularly impaired (McFie, Piercy, & Zangwill, 1950; Piercy & Smyth, 1962; Reitan, 1959, 1964; Warrington, 1969). The asymmetrical right greater than left hemisphere damage found to be correlated with this factor is consistent with the view that the right hemisphere is considered to primarily subserve a perceptual function and the left primarily subserving an executive output function (cf. Warrington, 1969).

Spatial-perceptual organizational abilities have traditionally been interpreted as being primarily subserved by the right (non-dominant) hemisphere, especially the temporal and parietal lobes.

TABLE 4.12

Factor I Sensory Motor Perceptual Organization

| Varimax* | | Neuro-Extension* | | Wechsler Extension* |
|---|---|---|---|---|
| NTACPERB | .77 | LJPTIM | .33 | PIQ -.38 |
| NTACPERNP | .76 | LJNPERR | .32 | FIQ -.33 |
| NTACPERP | .70 | LJNPTIM | .33 | BD -.42 |

| Varimax* | | Neuro-Extension* | | Wechsler Extension* |
|---|---|---|---|---|
| NTACPERFM | -.64 | LJBTIM | .42 | OA -.36 |
| NTACPERFL | -.57 | FTIPNP | .30 | |
| NTRAILB | .44 | FTIPNNP | .33 | |
| NTRAILA | .33 | FLOCPSD | .35 | |
| NPURDA | -.37 | FLOCNPD | .35 | |
| NSSERR | .32 | SDIGO | -.39 | |
| NCPM | .37 | SDIGW | -.37 | |
| NVLTOTE | .40 | NVLE | .31 | |
| | | NVLRNTRI | .40 | |
| | | NVLRE | .42 | |
| | | MFD | -.42 | |

*Loadings $\geq$.30 reported

Factor II Nonverbal Perceptual Reasoning and Abstraction

The second factor labelled Nonverbal Perceptual Reasoning and Abstraction is characterized by measures requiring nonverbal perceptual reasoning and abstraction. Loadings on this factor are dominated by subtests of the Halstead Category Test as are neuro-extension loadings. Wechsler Scale scores, particularly those involving spatial perceptual organization abilities, are modestly correlated, likely as underlying perceptual organization requirements for abstraction tasks. Royce et al. (1976) extracted two Halstead Category factors, VI and X. These authors argued

that solutions to subtests III, IV, V, and VI all involve abstract thinking, they note, however, that subtests III and IV consist of a series of oddity problems in which the spatial position is the key to the test solution. In subtests V and VI, the spatial position or orientation of the stimulus is irrelevant, but the configuration of the stimulus has to be represented numerically. In the present solution the extension loading of the WAIS-R Arithmetic subtest on this factor is consistent with this interpretation. Royce et al. (1976) argued that subtests III and IV do not involve any transformation, but rather the solution is dependent upon the mere cognition of classes, whereas subtest V and VI involve convergent thinking or require symbolic transformation.

In a factor analysis of Halstead-Reitan and Wechsler Memory scales, Leonberger et al. (1991) found that the Category Test and the Visual Paired Associate measures of the Wechsler Memory Scale-Revised defined a factor, perhaps due to their common requirement to retain nonverbal associations. In both the Ernst, Warner, Hochberg, and Townes (1988) and the Moehle et al. (1990) studies, the Category Test was absorbed within large visuo-spatial factors.

Royce et al. (1976) have noted that the factorial complexity of the Category Test should result in less localization and lateralization potential, although it has been shown to be highly sensitive to brain dysfunction generally, and Reitan and Wolfson (1993) insist it remains the most sensitive

measure in their battery to cerebral impairment. The identification of a clear specific Halstead Category factor in this study is remarkable, given that it has usually been absorbed into broad perceptual organizational factors in most studies.

Halstead (1947) proposed that the Halstead Category Test was a measure of biological intelligence. He considered that the frontal lobes were intimately involved in its successful resolution. An analysis of the cognitive requirements of the Halstead Category Test also suggest a primary role for the prefrontal cortex in executing its tasks. The Halstead Category Test's emphasis on perceptual organization, abstraction, logical analysis, nonverbal working memory, and contingent responding would seem to qualify it as an exemplar of executive function. Despite this, experimental research has not demonstrated a high level of specificity in discriminating pre-frontal lesions, although it appears to have the highest sensitivity of any neuropsychological measure to cerebral impairment generally, including prefrontal lesions (Reitan & Wolfson, 1993). These experimental findings are not inconsistent with the hypothesis that the prefrontal cortex has a primary role in success on the test, although it does suggest that impairment of many other brain regions may affect test performance. Thus, an intact prefrontal cortex appears to be a necessary, but not a sufficient condition, for success. In this factor analysis, many of non-executive perceptual organizational abilities required to

perform the Halstead Category Test are captured by Factor I, thus, Factor II may mainly reflect the contribution of the prefrontal cortex to success on the Halstead Category Test.

TABLE 4.13

Factor II Nonverbal Perceptual Reasoning and Abstraction

| Varimax* | | Neuro-Extension* | | Wechsler Extension* | |
|---|---|---|---|---|---|
| NHCAT7 | .80 | HCATCOR | .70 | VIQ | -.31 |
| NHCAT6 | .75 | HCATINC | .71 | PIQ | -.35 |
| NHCAT5 | .73 | HCATERR | .89 | FIQ | -.36 |
| NHCAT4 | .64 | | | AR | -.31 |
| NHCAT3 | .57 | | | PA | -.30 |
| NCPM | .39 | | | BD | -.37 |

*Loading $\geq$.30 reported

Factor III Fine Temporal Perceptual Motor Speed

Factor III appears to be defined by the Purdue Pegboard Test and reflecting the requirements of this test it has been labelled Fine Temporal Perceptual Motor Speed. A similar factor was identified by Royce et al. (1976) as Factor III in their analysis which they labelled Temporal Motor Speed. These authors concluded that perceptual aspects were more important than motor aspects. In this analysis, the fine perceptual motor requirements of the Purdue Pegboard Test appear to dominate. This is

supported by the fact that simple motor speed as reflected by the Finger Tapping Test has a loading of <.26. Further, other perceptual motor tasks with a cognitive component have only moderate loadings on this factor, e.g., WAIS-R Digit Symbol (-.37), Oral Word Fluency (-.36).

Several non-factorial studies suggest that performance on the Purdue Pegboard Test is particularly affected by right hemisphere dysfunction (e.g., Costa, Vaughan, Horowitz, & Ritter, 1969; Gazzaniga, Bogen & Sperry, 1964; Sperry, Gazzaniga, & Bogen, 1969; Vaughan & Costa, 1962). Vaughan and Costa (1962) emphasized that Purdue Pegboard performance implicates a relatively diffuse sensory motor system, while Sperry et al. (1969) emphasized the fine motor aspects.

Factor III seems to reflect the complexities of the Purdue Pegboard Test. Performance on this test would appear to be mediated by complex and diffuse neural systems responsible for the temporal resolution and coordination of fine motor sensory processing. As such, performance on the test can be expected to be highly sensitive to cerebral impairment generally, although it may be that the prefrontal cortex represents the highest neural articulation of the systems involved. Finally, this factor may support efficient psychomotor activities, such as reading, and contribute to efficient information processing.

TABLE 4.14

Factor III Fine Temporal Perceptual Motor Speed

| Varimax* | Neuro-Extension* | Wechsler Extension* |
|---|---|---|
| NPURDB -.84 | SDIGO -.34 | DSY -.37 |
| NPURDNP -.84 | SDIGW -.37 | |
| NPURDP -.79 | WWTOT -.36 | |
| NPURDA -.68 | SGEST1 -.32 | |
| NOWTOT -.36 | | |

* Loadings $\geq .30$

Factor IV Dynamic Verbal Processing

The fourth factor labelled Dynamic Verbal Processing is dominated by tasks which demand verbal short-term processing. The WCST loadings on this factor likely relate to concept formation, attentional set maintenance, and verbal short-term memory. The loading of the Childrens Colored Progressive Matrices Test may reflect its verbal logical reasoning and mental flexibility requirements.

The Seashore Rhythm Test loading likely relates to a general attentional component, while this may also be the main component factor involved in the Seashore Speech Sounds Perception Test. The Williams' Verbal Paired Associates Test also requires short-term verbal memory and concentration. Among the neuro-extension variables, the WCST loading of total errors is a logical association, and the Aphasia screening of verbal abilities fits well.

The double stimulation finger localization loadings are of particular interest given the many pre-school studies of finger localization have been shown to be predictive of later linguistic abilities.

Among the Wechsler subtests, both Verbal IQ and Digit Span have loadings of .-40. The other verbal subtests load between -.30 (Information) and -.38 (Vocabulary). In most factor analyses in which the Wechsler scales have been included, subtests such as Vocabulary and Information have defined a verbal factor and loadings here typically have been in the range of .75 to .90. This suggests that the present factor, while reflecting aspects of verbal ability, may be emphasizing verbal attentional or verbal processing aspects rather than acquired verbal abilities. The broad verbal/attentional factor recovered here would be expected to reflect the function of left (dominant) fronto-temporal structures involved in verbal processing. The emphasis may be on frontal attentional systems.

TABLE 4.15

Factor IV Dynamic Verbal Processing

| Varimax* | | Neuro-Extensions* | | Wechsler Extensions* | |
|---|---|---|---|---|---|
| NSEASHR | .62 | WCSTOT | .42 | VIQ | -.40 |
| NSSERR | .60 | APHAS | .36 | PIQ | -.31 |
| NWCSNPER | -.57 | FLOCPD | .35 | FIQ | -.38 |
| NWCSSUB | -.55 | FLOCNPD | .31 | IN | -.30 |
| NWCSUNI | -.54 | SDIGW | -.31 | DSP | -.40 |

| Varimax* | | Neuro-Extensions* | | Wechsler Extensions* | |
|---|---|---|---|---|---|
| NOWTOT | -.48 | | | | |
| NCPM | .46 | | | VO | -.38 |
| TRAILSB | -.40 | | | SI | -.34 |
| NVLTOTE | .39 | | | PC | -.30 |

* Loadings $\geq .30$

Factor V Wisconsin Card Sorting Test Perseveration

The fifth factor, labelled WCST Perseveration, does not appear to have been specifically identified previously. No other large factor analytic studies which have included the WCST have identified WCST perseveration as a separate factor, rather the WCST has typically loaded on a broad verbal factor. As discussed in Chapter III, the WCST is no doubt multifactorially complex. In this analysis difficulties with maintaining set and general verbal conceptual abilities appear to have been captured under Factor IV. Factor V emphasizes WCST perseveration as possibly related to left dorsolateral prefrontal dysfunction. Further indications of particular significance include virtual independence from Wechsler variables, e.g., the highest extension loading among this group being -.17 (FIQ). Secondly, previous studies (see Chapter III) have typically found correlations of between .40 and .50 between the Halstead Category Test and the WCST. In this study the highest loading of any Category Test variable was NHCAT5 (.12). It seems reasonable to infer that observed correlations between the HCAT and the

WCST were likely not due to the perseverative aspect of these measures. Two further observations are that the Wechsler scales correlate only modestly with this factor and age is not significantly related. This form of cognitive perseveration, although apparently specific, may nevertheless be of exceptional clinical significance, especially for offender and schizophrenic populations. As noted in Chapter III, research has especially implicated prefrontal dorsolateral structures in perseverative responding.

TABLE 4.16

Factor V WCST Perseveration

| Varimax* | | Neuro-Extension* | | Wechsler Extension* |
|---|---|---|---|---|
| NWCSCOR | .85 | WCSTOT | .79 | --- |
| NWCSPER | .78 | | | |
| NWCSNPER | .48 | | | |
| NWCSSUB | -.41 | | | |
| NWCSUNI | .33 | | | |

* Loadings ≥.30

Simple Structure Considerations

The simple structure obtained in this factor solution appears to be remarkable. Very few variables were found to be salient (loading ≥.30) for more than one factor. However, where variables were factorially complex, interpretation was usually not difficult. Trails B was found to load on Factor

I and Factor IV, .40 and .44 respectively. The loading on Factor I, is understandable in terms of the perceptual organizational requirements of the test while the loading on Factor IV likely relates both to the verbal flexibility and attentional requirements tapped by this factor. Similarly, Speech Sounds Perception had a loading of .32 on Factor I and this is likely due to its attentional component. Its loading of .60 on Factor IV is likely due to the specific verbal attentional aspect of this factor. The Childrens Progressive Matrices Test has both perceptual organizational and verbal logical requirements, and thus a shared variance with both Factor I and Factor IV is interpreted on those accounts. The Williams' Paired-Associates Test would be predicted to load on Factor IV because of the verbal memory demands and, while not considered to have significant perceptual organizational demands, its loading of .40 on Factor I is perhaps due to a general memory requirement spanning both verbal and non-verbal tasks. Oral Word Fluency has loadings of .36 on Factor III and -.48 on Factor IV. The interpretation is that the fine motor requirements of Factor III and the verbal capacities associated with Factor IV account for these loadings. Finally, the measures associated with the WCST load on both Factor IV and Factor V. The proposed interpretation is that the general verbal conceptual and set maintenance requirements account for loadings on Factor IV while conceptual rigidity and perseveration account for loadings on Factor V. In sum, it is suggested that the simple structure of this solution is remarkable and notable exceptions are readily interpretable.

Further Relationships

Perhaps the most notable relationship among the extension loadings and the Varimax solution is that the highest correlation of age with any factor is .11, supporting the earlier observation of quite limited age effects. In Section IV above, it was noted that, in general, verbal abilities as assessed by some Wechsler scales, were positively correlated with age, especially in the OF group. The failure to observe a high correlation of age with Factor IV, which has been interpreted to reflect dynamic verbal abilities, reinforces the notion that verbal knowledge, rather than dynamic verbal capacities, account for Wechsler verbal scale-age group correlations. That is, dynamic verbal abilities do not increase substantially in offenders compared to normal controls, but their verbal knowledge, as measured by IQ tests do, and likely this is attributable to prison education programs.

As a general comment, the argument has been made by Reitan and Wolfson (1993) that intelligence tests are biased toward predicting scholastic achievement while neuropsychological measures are more focused on dynamic functioning and adaptive abilities. This argument has been difficult to fully sustain because in most major factor analyses, the Wechsler measures have been included along with neuropsychological variables and likely because of their density in hyperspace, they have dominated factor solutions. The current finding of only modest $\leq .40$ loadings of Wechsler variables on neuropsychological factors in this study appears to support,

Reitan and Wolfson's (1993) assertion that many Wechsler variables are not highly related to dynamic/adaptive functioning.

Summary

Separate factor analyses of 28 neuropsychological variables were conducted on the NC, OF, and PP groups. In each case a five factor Varimax solution was selected as the most interpretable. A further analysis of congruence between these factor solutions and a factor analysis combining the NC, OF, and PP groups indicated a high level of concordance of the separate analyses with the combined solution. Accordingly, the combined 5 factor solution was selected. This solution exhibited a high degree of simple structure, and the factors were labelled and interpreted as reflecting five relatively independent dimensions of neuropsychological function. These 5 factors were labelled as follows: I Sensory Motor Spatial Perceptual Organization; II Nonverbal Perceptual Reasoning and Abstraction; III Fine Temporal Perceptual Motor Speed; IV Dynamic Verbal Processing, and; V WCST Perseveration. Measures previously associated with prefrontal function and dynamic verbal abilities were prominent in defining the combined solution. Relationships between neuropsychological theory and the empirically recovered factors will be further discussed in Chapter V. In the next section, the focus is on patterns of organization of neuropsychological abilities within individuals.

## SECTION VIII MODAL PROFILE ANALYSIS (MPA)

### Modal Profile Analysis - Neuropsychological Factors

Current conceptualizations of brain function postulate that the brain is intricately organized in terms of hierarchical neural systems. Further, these neural systems interact to produce behavior at a molar level. Within this perspective, the five relatively distinct empirically recovered test factors discussed in Section VII are hypothesized to reflect a level of functional organization of brain structures and processes.

The question addressed in this section relates to the organization of these five identified neuropsychological abilities within individuals. More specifically, is it possible to characterize individuals in terms of patterns of relative ability? For instance, can a group of subjects be identified which is characterized by relatively poor spatial perceptual ability, but relatively high verbal processing ability, irrespective of their absolute levels of ability? These questions relate to the relative organization of neuropsychological abilities within individuals, irrespective of their absolute capacity. A second question pertains to whether certain profiles are more associated with one group of subjects than another and a third question relates to the degree of impairment of individuals within profiles and this will be considered following the Modal Profile Analysis. Once these questions are addressed for the neuropsychological factors, a similar analysis is conducted on the Wechsler subtests. Lastly, the convergence or intersection of neuropsychological and Wechsler modal profiles is examined.

MPA Technical Description

Modal Profile Analysis is based on Sneath's (1975) vector model of disease and is consistent with the modern scientific interpretation of diseases according to the logic of classes (Taylor, 1980, 1981). The model locates relatively homogeneous subsets of individuals by identifying individuals with similar coordinates in multidimensional space. The coordinates of an individual in this space are the shape of individual's profile. Severity or magnitude and deviation from the target homogeneous clusters of individuals are determined through profile elevation.

Computationally, the model is based upon the singular value decomposition algorithm (Chambers, 1977; Kennedy & Gentle, 1980) and is termed Modal Profile Analysis (Skinner, 1977, 1981; Skinner & Sheu, 1982). Various studies that have used Modal Profile Analysis have demonstrated its value as a classification model (e.g., Jackson, 1978, Skinner, 1977; Skinner & Jackson, 1978; Smiley, 1977). Prior to the singular value decomposition, the matrix is standardized by variables (0 MEAN, 1 *SD*, i.e., z scores) to produce equal weighting of the variables. This is particularly important in numerical taxonomy so that artifactual differences in the scale of the variables do not influence the solution (Sneath & Sokal, 1973; Sokal, 1974). Next, the overall profile elevation and scatter are removed from each individual's profile and saved for the assessment of severity, magnitude, or size of type. In this way, profile shape, elevation, and scatter are not confounded (Cronbach & Gleser, 1953; Skinner, 1978). In addition,

removing the mean from each subject's profile has the effect of adjusting for base rates of responding and this facilitates the comparison of different types of subjects (Cunningham, Cunningham, & Green, 1977).

The next step is to decompose this double standardized matrix by the singular value decomposition algorithm and to rescale the (left hand) eigenvectors by their associated singular values. A lower order subspace or reduced rank solution is then obtained by selecting dimensions according to Horn's (1965) random data criterion. This lower order subspace has the property of being less prone to measurement error and is therefore more reliable than the full rank solution (Gleason & Staelin, 1973; Maxwell, 1975).

Computationally, the procedure thus far is equivalent to a Q type factor analysis. These dimensions or ideal types of people are then rotated to a simple structure criterion, such as Varimax (Kaiser, 1958). These rotated dimensions, ideal types, or homogeneous clusters of individuals are then projected into the attribute factor space by computing factor scores. Each individual is classified as belonging to one of these dimensions on the basis of their highest correlation with one of these factor scores. In the case where an individual has a high correlation with more than one type then the individual is classified as a mixed type. In actual practice, mixed types are infrequent and rarely consist of more than two types. Once these types have been derived, subtypes are derivable on the basis of the frequency distribution of the elevation and scatter parameters for individuals classified according to a particular subtype.

MPA Results: Neuropsychological Factors

The 5 varimax factors interpreted in Section VII were retained as variables for the Modal Profile Analysis (MPA).

MPA (Skinner & Sheu, 1982)[1] is set into three stages. In stage I, Modal Profiles are extracted for each subsample in the analysis. Stage II conducts a principal components factor analysis followed by varimax rotation to yield a common set of modal profiles for contrasting individual samples. Stage III provides the analysis of each sample by profile.

In stage I of the analysis, four profiles were computed for each of the three samples NC, OF, and PP. Employing 0.50 as the minimum correlation of a profile with a modal subtype, 100% of subjects in each sample were classified explaining 100% of the variance.

Stage II of the analysis yielded four profiles common to all three groups. These profiles based on the five factors derived discussed in Section VII above, are presented in Table 4.17 below.

[1]Permission to utilize this program is appreciated from H. Skinner, Ph.D. The extensive modifications made by D. Pritchard, Ph.D. and J.R. Reddon, Ph.D. to modify this program to function on a PC are also deeply appreciated.

TABLE 4.17

Neuropsychological Modal Profiles

| Profile* | | Factor I | II | III | IV | V |
|---|---|---|---|---|---|---|
| 1 | T scores | 55 | 31 | 51 | 59 | 56 |
| 2 | T scores | 44 | 48 | 47 | 38 | 67 |
| 3 | T scores | 32 | 53 | 51 | 63 | 53 |
| 4 | T scores | 46 | 44 | 30 | 48 | 43 |

* Profiles are reported such that the greater the T score, the greater the level of impairment for all factors.

Modal Profiles Interpretation

The purpose here is to examine each profile and describe its main characteristics with respect to the five factors derived from the factor analysis reported in Section VII.

The five factors were interpreted as:

I Sensory Motor Spatial Perceptual Organization;

II Nonverbal Perceptual Reasoning and Abstraction;

III Fine Temporal Perceptual Motor Speed;

IV Dynamic Verbal Processing; and

V WCST Perseveration.

Accordingly, each profile will be discussed with respect to its bipolar shape. To illustrate the significance of each type, reference will be made to one or more of the major neuropsychological test variables to illustrate the

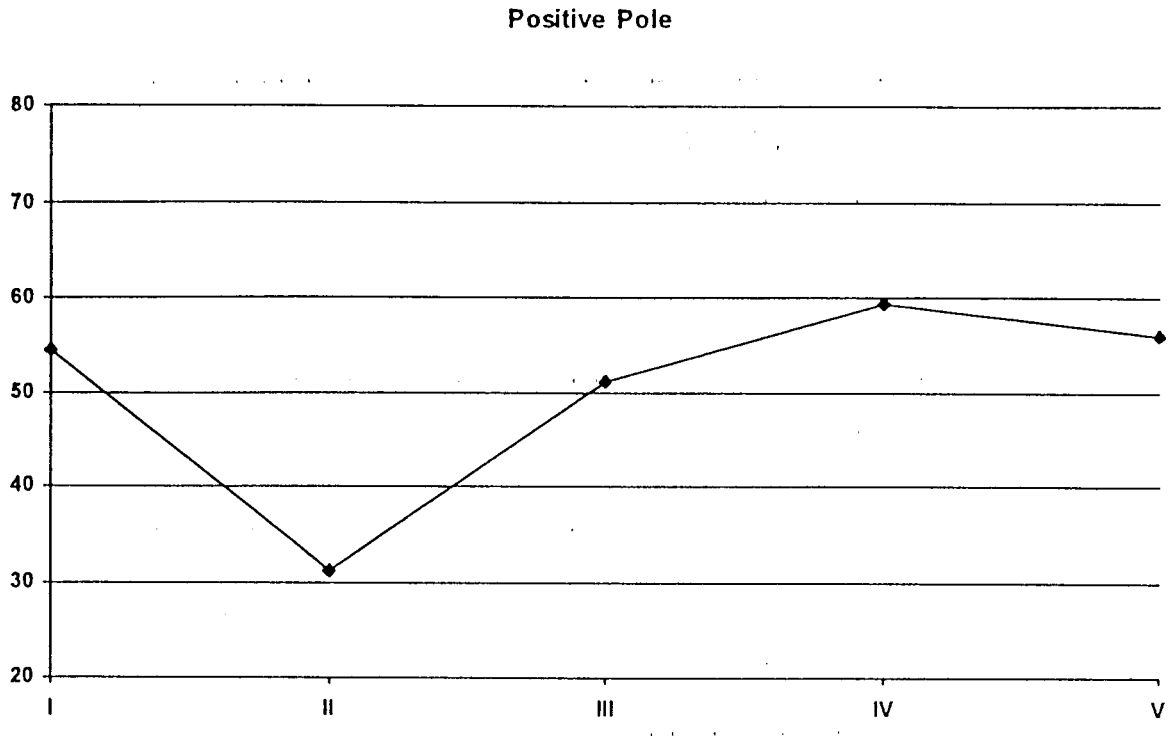
general contrast in neuropsychological abilities across the positive and negative poles of each profile.

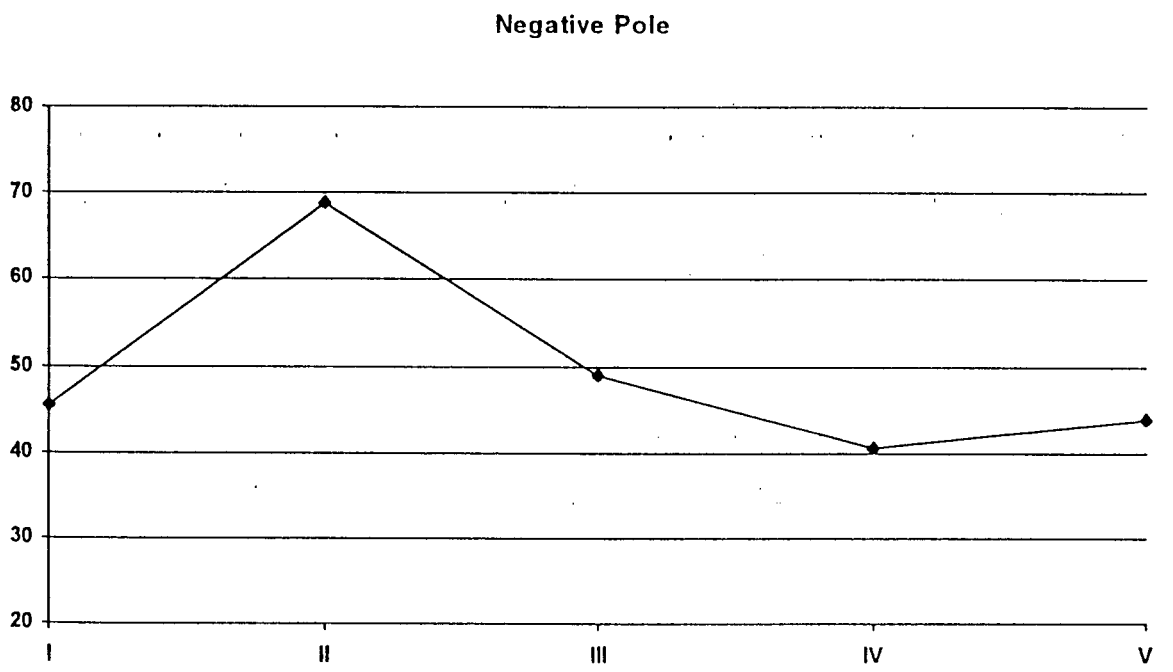
Profile 1 (See Figure 4.1)

The positive pole of Profile 1 is characterized by T scores of 55, 31, 51, 59, and 56 on Factors I through V respectively. It will be recalled that in this scheme a high T score reflects a high level of impairment, hence subjects classified according to the positive pole of this profile would be expected to have exceptional (T score, 31) nonverbal reasoning and abstraction abilities, typically marked by high performance on factor marker **Halstead Category Test** and relatively poor dynamic verbal processing abilities (T score, 59) as marked, e.g., by relatively poor performance on the **Speech Sounds Perception Test**. Performance on other factors range between T scores of 51 and 56, and are not particularly remarkable. Subjects classified at the negative pole of this profile have the reverse profile and a converse commentary would apply throughout, e.g., they would be expected to have very limited nonverbal reasoning skills, poor **Halstead Category Test** performance, and so on.

Figure 4.1

# Neuropsychological Modal Profile 1



Profile 2 (see Figure 4.2)

The positive pole of Profile 2 is defined by T scores of 44, 48, 47, 38, and 67 on Factors I through V. This profile is somewhat unusual in that subjects classified at the positive pole demonstrate a generally high (T score 38) level of verbal processing ability, e.g., **Speech Sounds Perception Test** or the **Seashore Rhythm Perception Test**, but are remarkably poor (T score 67) on verbal conceptual perseveration on the **WCST**. Subjects at the positive pole also have slightly above average abilities (relatively) on Factors I, II, and III, but not exceptionally so. As in the case of Profile 1, comments on subjects classified at the negative pole all apply conversely.

Profile 3 (see Figure 4.3)

The positive pole of Profile 3 reflects T scores of 32, 53, 51, 63, and 53 on Factors I through V. It is remarkable in distinguishing a group of subjects with exceptional sensory motor spatial perceptual organizational abilities (T score 32) marked by high performance on the **Tactual Performance Formboard Test** in contrast with relatively low dynamic verbal processing abilities (T score, 63) as marked here by the **Speech Sounds Perception Test**. The other T scores vary between 51 and 53 and are not notable. Of course, converse comments apply to subjects classified at the negative pole of this profile.

Profile 4 (see Figure 4.4)

Profile 4, with T scores of 46, 44, 30, 48, and 43 on Factors I through V is characterized by exceptional abilities on Factor III Fine Temporal Perceptual Motor Speed (the main marker for this factor is the **Purdue Pegboard Test**) and relatively higher scores across all four other factors. Subjects classified at the negative pole would have relatively poor fine motor coordination and also have modestly below average relative abilities across all other factors.

Classification

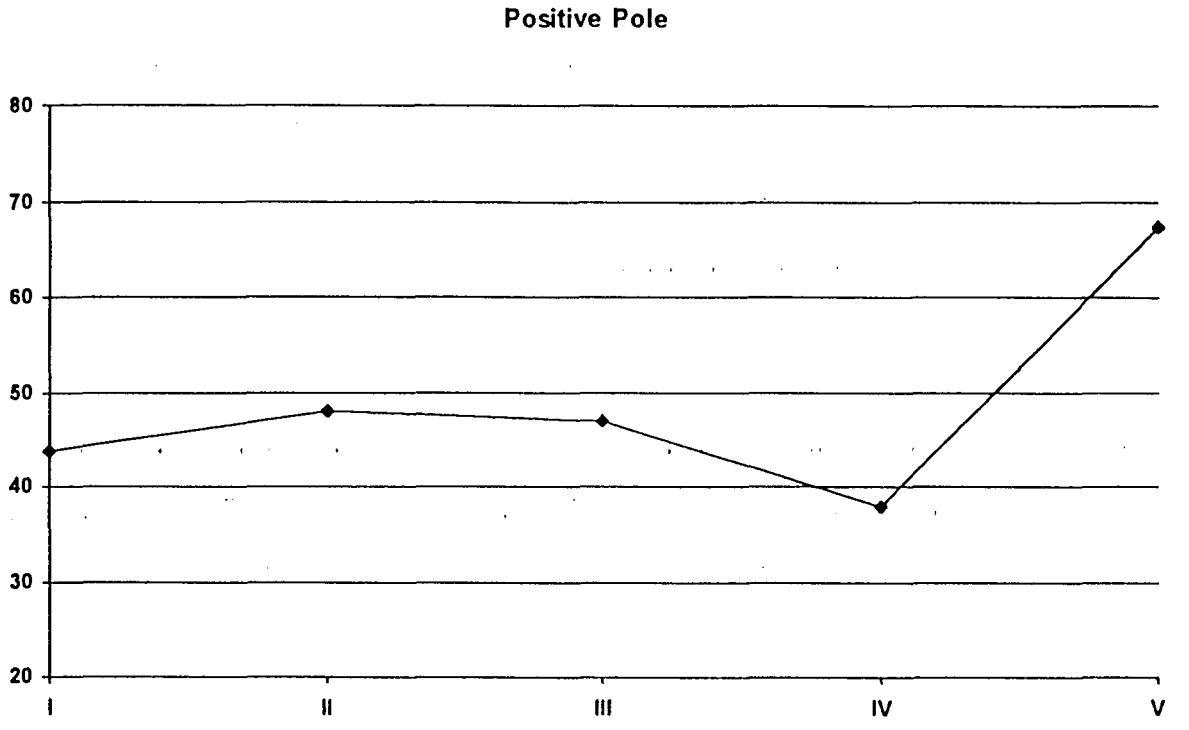
Stage III classifies each of the subjects by group according to membership in one of the 4 bipolar profiles. The results of this analysis were cross-tabulated and are reported in Table 4.18 below. It is noted that as in the case of the stage I analysis, all subjects were classified and 100% of the variance accounted for with a minimum profile - subtype correlation criterion for classification set at .50.

A chi-square coefficient examining the proportion of samples by profile type was significant ($P < .000$). The clearest trends was for a disproportionate number of subjects to be assigned to profile 4+ and 4-. These subjects, were characterized by relatively average cognitive neuropsychological abilities, however, they vary in terms of their fine perceptual motor function. The highest proportion of NC subjects (23.4%) are assigned to it. Profile 1-, reflecting high nonverbal perceptual reasoning

and abstraction abilities with relatively low verbal dynamic abilities, was also quite a common subtype among all three groups. Across samples, while there were differences, for example, the NC group appears under-represented on profiles 2-, 3+, and 3-, no major correlation of any sample

Figure 4.2

# Neuropsychological Modal Profile 2



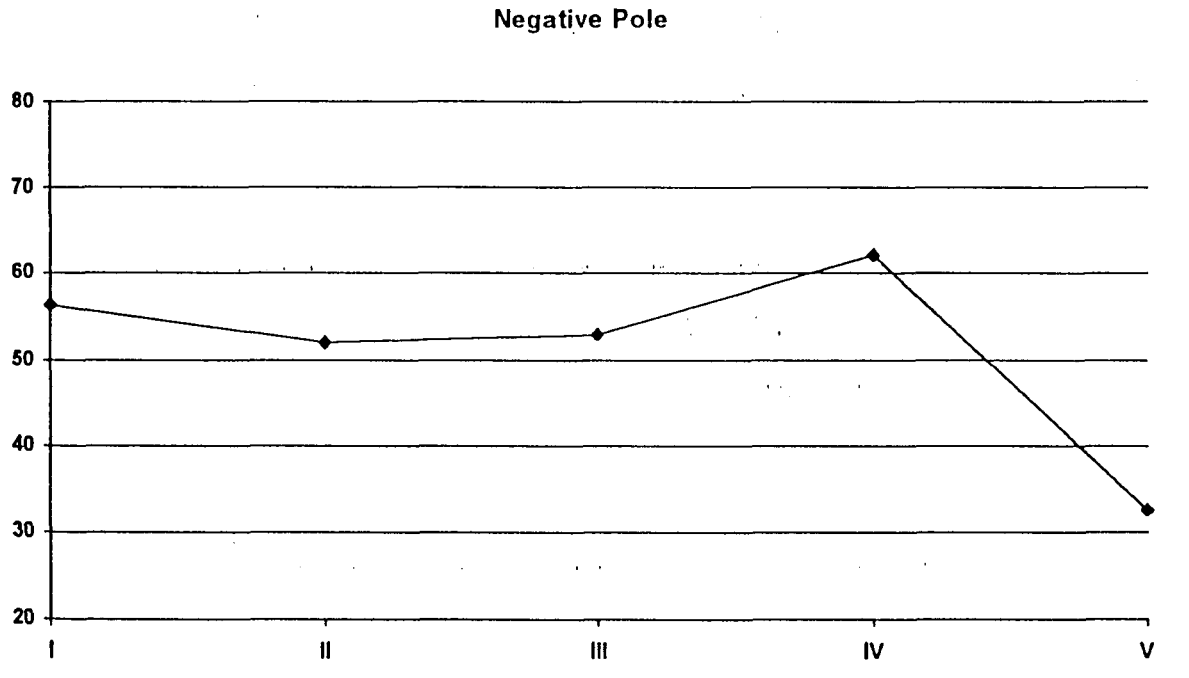
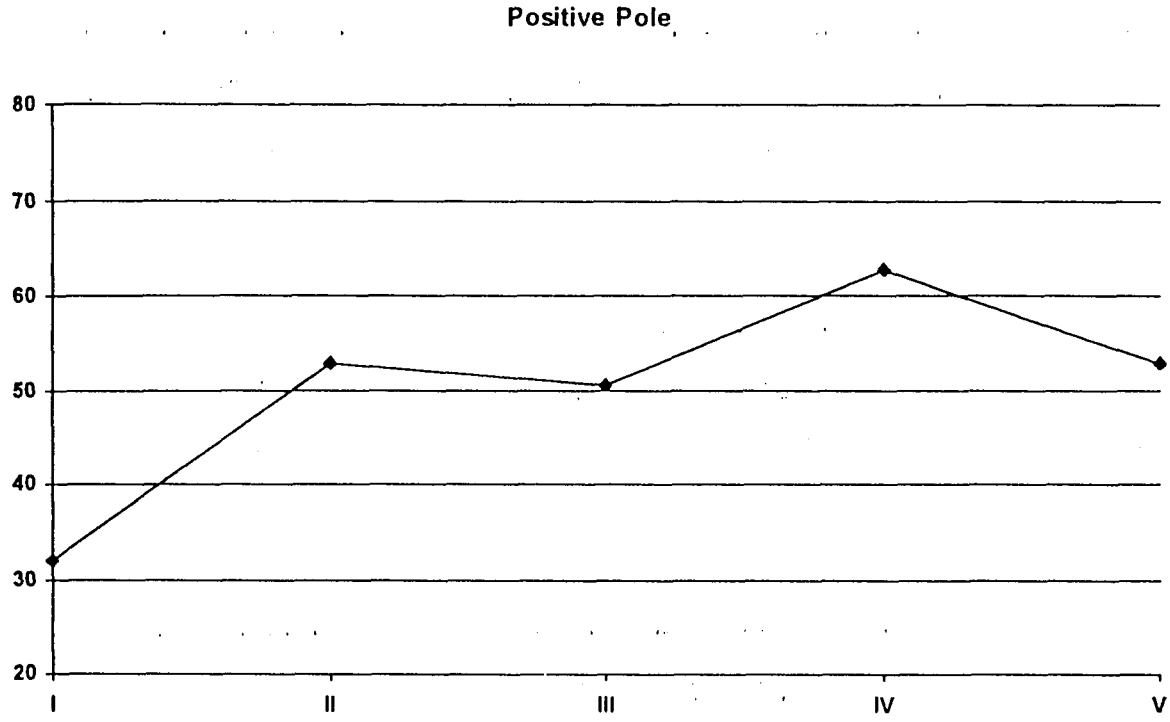



Figure 4.3

# Neuropsychological Modal Profile 3



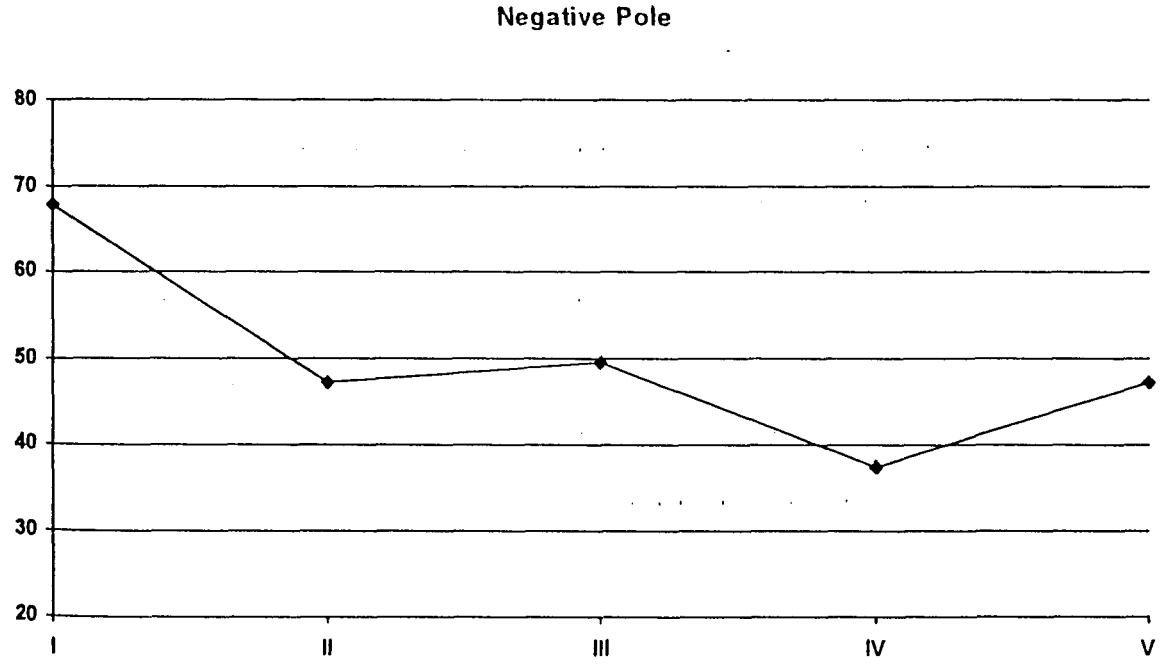
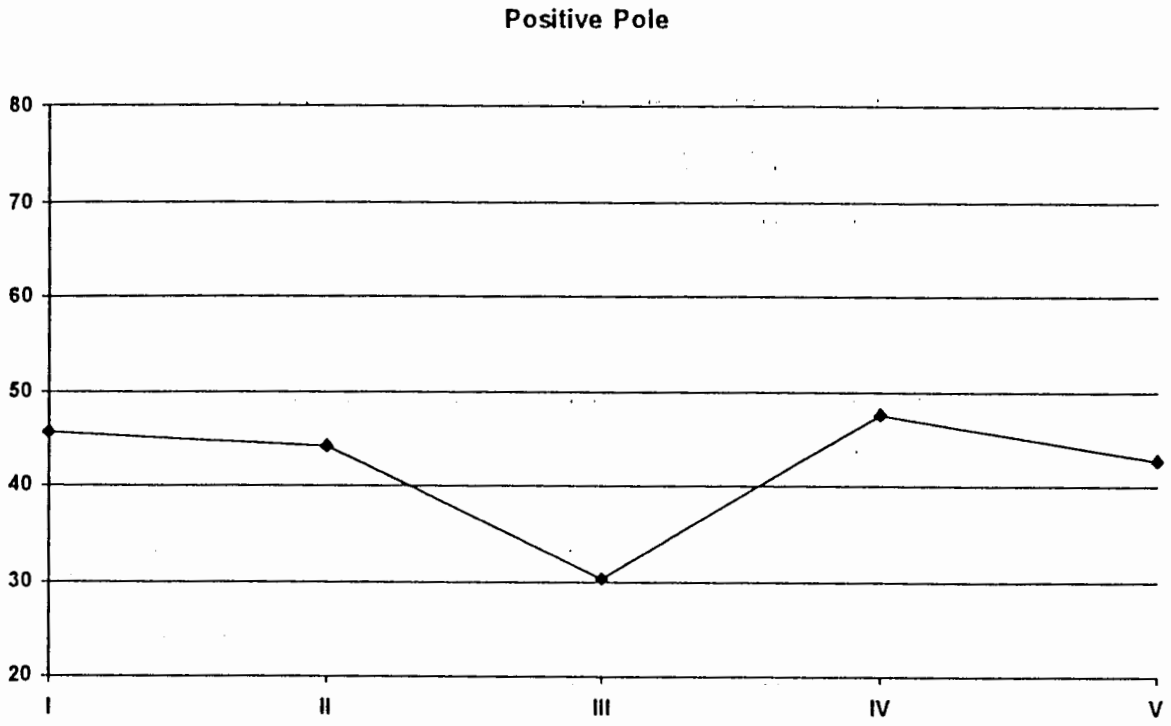
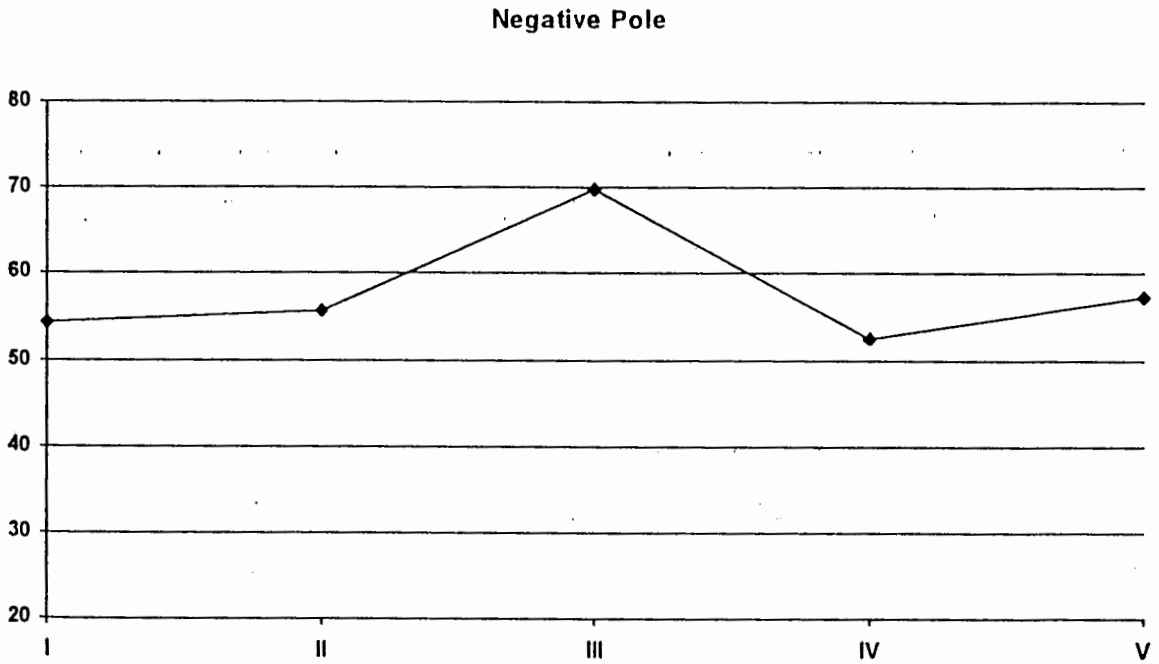



Figure 4.4

# Neuropsychological Modal Profile 4



with any profile type is observed. Discrimination among the three groups will, thus, depend on profile elevation. This issue is further addressed below.

TABLE 4.18

Modal Profile Classification by Sample

| Profile Type | SAMPLE NC | | OF | | PP | | Total | |
|---|---|---|---|---|---|---|---|---|
| | N | (%) | N | (%) | N | (%) | N | (%) |
| 1+ | 11 | (11.7) | 71 | (13.7) | 27 | (6.8) | 109 | (10.8) |
| 1- | 14 | (14.9) | 75 | (14.5) | 68 | (17.1) | 157 | (15.5) |
| 2+ | 13 | (13.8) | 48 | (9.2) | 49 | (14.9) | 120 | (10.8) |
| 2- | 7 | (7.4) | 67 | (12.2) | 44 | (11.1) | 118 | (11.7) |
| 3+ | 6 | (6.4) | 54 | (10.4) | 45 | (11.3) | 105 | (10.4) |
| 3- | 6 | (6.4) | 49 | (9.4) | 45 | (11.3) | 100 | (9.9) |
| 4+ | 22 | (23.4) | 68 | (13.1) | 71 | (17.9) | 161 | (15.9) |
| 4- | 15 | (16.0) | 87 | (16.8) | 38 | (9.6) | 140 | (13.9) |
| | Σ94 | | Σ519 | | Σ397 | | Σ1010 | |
| | | % 9.3 | | % 51.4 | | % 39.3 | | % 100 |

Impairment Analysis

In Table 4.18, the neuropsychological profiles of all subjects from the NC, OF, an PP groups were classified and tabulated according to their primary association with one of the 8 modal profile subtypes. These 8

subtypes represent the positive and negative poles of the 4 modal profiles that were derived from the Modal Profile Analysis based on the 5 neuropsychological factors derived in Section VII. According to this tabulation, it is observed that, overall, there were statistically significant differences (chi-square, $P<.01$) in the proportion of subjects from each group that was assigned to the 8 profiles. However, there was not a strong tendency for group membership to predict profile classification. Each group had profiles classified among all 8 profile types and the pattern of assignment to profile types was not strikingly different for any group. Thus, if the NC, OF, and PP groups are to be discriminated, this must be done, not on the basis of their pattern of neuropsychological abilities or profile types, but, rather, on the basis of level of abilities within each profile type.

One way to obtain an appreciation of differences in ability level for the NC, OF, and PP groups by profile type would be to examine their relative performance on the main neuropsychological factors that define each profile in terms of factor scores. It is suggested, however, that a comparison in terms of test markers is closer to the original data and will provide a good illustration of the significance of the modal profiles.

In Table 4.19, the relative performance of the NC, OF, and PP groups is tabulated on tests which have been selected as markers for factors which especially define profile types. For instance, consider the tabulations relative to profile 1+ and profile 1-. Profile 1+ was characterized by high level of performance on Factor II, Nonverbal Perceptual Reasoning and Abstraction

(T score, 31), and relatively poor performance on Factor IV, Dynamic Verbal Processing (T score, 59). Other Factor T scores are intermediate: Factor I, Sensory Motor Perceptual Reasoning (T score, 55); Factor III, Fine Temporal Perceptual Motor Speed (T score, 55); and Factor V, WCST Perseveration (T score, 56). Factor T scores for Profile 1- would reflect the opposite pole of the distribution, e.g., for Factor II, it would be 69, for Factor IV, 41, and so on.

With reference to Profile 1+, Table 4.19, it is noted that the Halstead Category Test and Speech Sounds Perception Test have been selected as markers for Factor II and Factor IV, respectively. The average *SD*, based on the NC sample, is reported for members from each sample classified to Profile 1+. It is noted that the members of the NC group assigned to Profile 1+ have an average Halstead Category Test score of .85 *SDs* above their own mean, and members of the OF and PP groups have *SDs* of .29 and -.41 relative to the overall NC mean. Similarly, for the Seashore Speech Sounds Test, the members of the NC, OF, and PP groups classified to this profile have average scores of .14, -1.48, and -1.81 *SDs* from the NC mean, respectively.

Across the 8 profile types, it will be noted that, in general, where profiles highlight negative neuropsychological ability, the most negative performance is found among the PP group followed by the OF group, and the NC group is the least impaired. Consider, e.g., the performance of the members assigned to Profile 2- on Speech Sounds. The PP, OF, and NC groups members had

average test scores of -4.35, -2.31, and -.28 *SDs* below the NC mean, respectively. There are some exceptions to this general trend, however. Consider that performance differences of NC and OF members assigned to Profile 3- is not large on the Tactual Performance Test measure, although all three groups demonstrate impairment.

TABLE 4.19

Performance of Groups Classified by Profile

| | | GROUP | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Profile | Marker(s) | NC | (N) | OF | (N) | PP | (N) | Total (N) |
| Profile 1+ | Halstead Category | +.85* | (11) | +.29 | (71) | -.41 | (27) | 109 |
| | Speech Sounds Perception | +.14 | (11) | -1.48 | (71) | -1.81 | (27) | 109 |
| Profile 1- | Halstead Category | -1.30 | (14) | -2.23 | (75) | -3.31 | (68) | 157 |
| | Speech Sounds Perception | +.11 | (14) | -.42 | (75) | -.29 | (68) | 157 |
| Profile 2+ | Speech Sounds Perception | +.24 | (13) | +.20 | (48) | +.23 | (49) | 120 |
| | WCST | -1.93 | (13) | -1.87 | (48) | -2.22 | (49) | 120 |
| Profile 2- | Speech Sounds Perception | -.28 | (7) | -2.31 | (67) | -4.35 | (44) | 118 |
| | WCST | +.68 | (7) | +.06 | (67) | -1.03 | (44) | 118 |

| | | GROUP | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Profile | Marker(s) | NC | (N) | OF | (N) | PP | (N) | Total (N) |
| Profile 3+ | Tactual Performance Non-Preferred Hand | +.60 | (6) | +.27 | (54) | +.54 | (45) | 105 |
| | Speech Sounds Perception | -.39 | (6) | -1.00 | (49) | -1.49 | (45) | 100 |
| Profile 3- | Tactual Performance Non-Preferred Hand | -1.89 | (6) | -2.00 | (54) | -4.68 | (45) | 105 |
| | Speech Sounds Perception | -.01 | (6) | -.34 | (49) | -.80 | (45) | 100 |
| Profile 4+ | Purdue Pegboard Assemblies | -.57 | (22) | -1.04 | (68) | -2.53 | (71) | 161 |
| Profile 4- | Purdue Pegboard Assemblies | +.43 | (15) | .29 | (87) | -.44 | (38) | 140 |
| | | | Σ94 | | Σ519 | | Σ1010 | |

* *SD* re: Control Norms.

Summary of MPA: Neuropsychological Factors

In Section VI above, it was concluded that the OF group, and especially the PP group, showed marked impairment on many neuropsychological measures. The factor analysis reported in Section VII identified 5 orthogonal factors accounting for approximately 59% of the variance among 28 neuropsychological measures. Modal Profile Analysis was applied to these 5 factors and this resulted in 4 bipolar modal profiles. All members of the NC, OF, and PP groups were classified as belonging to one of these modal types. Examination of the average scores (in terms of *SDs* from the NC mean) indicated a high degree of neuropsychological impairment among offenders and psychiatric patients. Although a high level of impairment was noted in the impairment analysis of Section VI, the foregoing modal profile impairment analysis illustrated how impairment along one or two dimensions of neuropsychological ability, nevertheless, can result in a global clinical level of impairment, despite areas of normal neuropsychological function. Thus, the overall degree of impairment in the OF and PP groups is most certainly greater than the average level of impairment across all measures, as different patterns, or types of impairment, are manifest.

Modal Profile Analysis Results: Wechsler Scales

The Wechsler scales have featured prominently in clinical neuropsychology. For instance, they are typically administered along with the Halstead-Reitan Neuropsychological Battery. As well, as noted earlier, where they have been factor analysed along with other neuropsychological

batteries, the verbal and performance scales have dominated the factorial solutions, perhaps because of their high reliabilities. Reitan and Wolfson (1993) have noted that Wechsler measures are useful in their own right, but the focus during their development was not primarily on adaptive abilities and functioning which has been the tradition in neuropsychology, but more on the prediction of scholastic achievement.

Accordingly, in this study, the goal was to examine test performance on Wechsler and neuropsychological variables separately. Thus, similar to the analysis of neuropsychological factors above, the purpose here is to conduct a modal profile analysis of the Wechsler subtests. Next, the profiles obtained will be interpreted and an impairment analysis across the NC, OF, and PP groups will be reported upon.

In the factor analysis of neuropsychological variables reported in Section VII, the Wechsler scales were included as extension variables. In that analysis, modest correlations of Wechsler variables were reported with the neuropsychological factors. Most notable: a) Performance IQ loaded -.38 on Factor I, Sensory Motor Perceptual Organization; b) Full Scale IQ and Performance IQ loaded -.36 and -.35 respectively on Factor II, Nonverbal Perceptual Reasoning and Abstraction; and c) Verbal IQ loaded -.40 on Factor IV, Dynamic Verbal Processing. These relationships, while non-trivial, suggest that the factor space of the neuropsychological variables only partially overlaps with that of Wechsler variables, although the nature of that overlap is not easily ascertained. In this analysis, it will be possible to

further examine those relationships by observing the intersection of neuropsychological modal subtypes with Wechsler modal subtypes. This analysis begins with a modal profile analysis of the Wechsler subtests.

Wechsler Modal Profiles

Modal Profile Analysis was conducted on the 11 Wechsler subtests: Information (IN), Digit Span (DSP), Vocabulary (VO), Arithmetic (AR), Comprehension (CO), Similarities (SI), Picture Completion (PC), Picture Arrangement (PA), Block Design (BD), Object Assembly (OA), and Digit Symbol (DSY). The NC (94) sample had complete data and missing data for the OF and PP groups was not re-estimated, resulting in a loss of 3, OF (516) subjects and 5, PP (392) subjects for a total of 1,002 subjects.

Modal Profile Analyses were conducted extracting 2, 3, 4, and 5 profiles. Upon review, on the basis of interpretability, the 4 profile solution was selected for interpretation and further analysis.

Stage II of the Modal Profile Analysis reports T scores on the 11 Wechsler subtests for each profile. These are presented in Table 4.20. As was the case for the neuropsychological profiles, high T scores reflect poor performance.

TABLE 4.20
Weschler Modal Profiles

| PRO-FILE | SUBTEST IN | DSP | VO | AR | CO | SI | PC | PA | BD | OA | DSY |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 53 | 52 | 47 | 59 | 49 | 50 | 34 | 31 | 65 | 63 | 48 |
| 2 | 49 | 72 | 50 | 63 | 45 | 45 | 48 | 54 | 51 | 38 | 35 |

| PRO-FILE | SUBTEST IN | DSP | VO | AR | CO | SI | PC | PA | BD | OA | DSY |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 3 | 57 | 41 | 58 | 47 | 62 | 58 | 55 | 46 | 51 | 50 | 25 |
| 4 | 39 | 50 | 41 | 45 | 45 | 40 | 63 | 59 | 64 | 65 | 41 |

Wechsler Profile Interpretation

Profile 1 (See Figure 4.5)

The positive pole of Profile 1 (see Figure 4.5) is defined especially by low levels of performance on the BD and OA scales in contrast to high performance on PC and PA scales. Mainly, this profile discriminates within the performance subtests, except for DSY, which is neutral. Why these scale performances are highly discrepant within a subset of individuals presents an interpretive challenge. Two hypotheses are considered: a) Reitan and Wolfson (1993) have suggested that an inability to perform the PA task is pathognomonic sign of right temporal lobe lesions. Thus, the negative pole of this profile may reflect relative dysfunction of temporal lobe structures relative to parietal lobe function, and the opposite configuration may reflect the converse; or b) a related functional hypothesis would be that the PC and PA subtests are facilitated by verbal mediation, and the positive pole reflects an inability to verbally mediate visuo-spatial material. Of course, both hypotheses may be tenable.

Profile 2 (See Figure 4.6)

The positive pole of Profile 2 reflects a high level of performance on the OA and DSY scales, combined with relatively low performance on the DSP and AR scales. The converse relationships apply to the negative pole. This profile seems to suggest that verbal attention and psychomotor abilities can be disassociated.

Profile 3 (see Figure 4.7)

Profile 3 seems to involve a contrast between verbal attention and psychomotor abilities, as reflected by the DSP and DSY subtests, with acquired verbal abilities (subtests IN, VO, and CO). Neuropsychologically, an orbital (DSP and DSY) versus a left temporal/parietal focus (IN, VO, and CO) is suggested.

Profile 4 (see Figure 4.8)

This profile clearly differentiates verbal and psychomotor from perceptual organizational abilities among the Wechsler scales. Neuropsychologically, a left hemisphere/orbital prefrontal versus right temporal and parietal structures appear to be differentiated. This profile may underlie the classical P>V finding among young offenders and adults reported in many studies.

Profile 4- was the most popular Wechsler subtype in this study; 28.7% of the OF group and 24.2% of the PP group, but only 3.2% of the NC group, are assigned to it. It appears to maximally represent divergent

Wechsler verbal/performance abilities. For instance, among the OF group having this profile, the mean verbal IQ was 87.45 and the mean performance IQ was 100.70.

In sum, four Wechsler modal profiles appear to provide a neuropsychologically interpretable differentiation of performance on the Wechsler scales. Next, classification of the NC, OF, and PP groups is discussed.

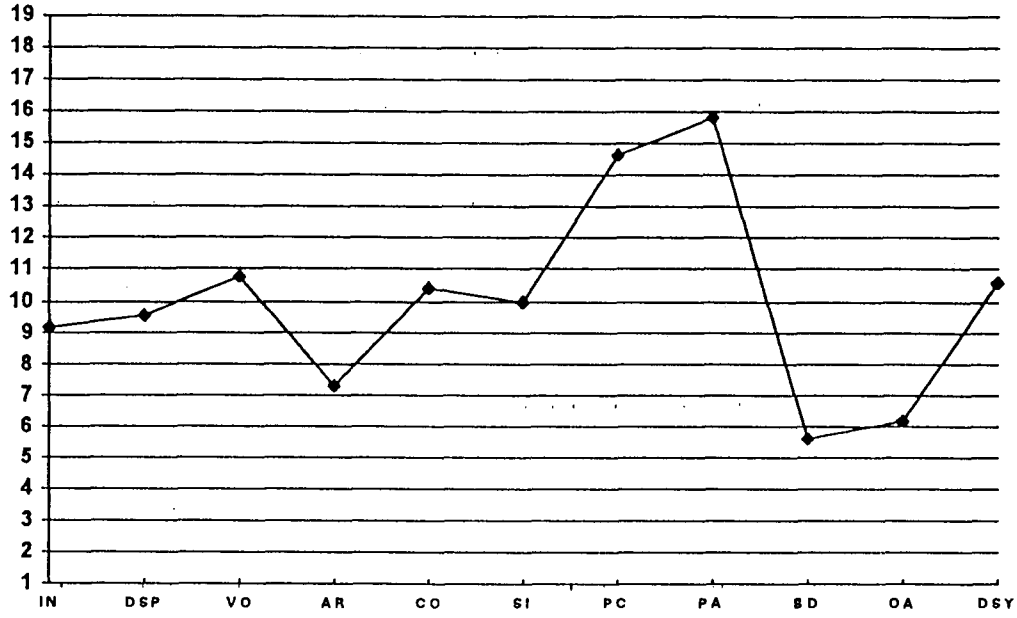
Wechsler Profile Classification

Stage III of the MPA program classified 90% of subjects with a profile - subtype correlation criterion of .40. A chi-square coefficient relating proportion of sample by profile type was significant ($P<.000$). Profile classification is reported in Table 4.21. Visual analysis of this table indicates, as mentioned above, that over 60% of all subjects are classified in profiles 2-, 3-, and 4-. Profile 2- classified 16.7% of all subjects and it represents 23.4% of the NC group, 16.1% of the OF group, and 16.7% of the PP group. This profile is characterized especially by relatively high performance on DSP compared to performance on OA and DSY. Profile 3- includes 21.3% of the NC group, 18.4% of the OF group, and 22.4% of the PP group. It is mainly defined by high scores on DSP and DSY, relative to scores reflecting verbal knowledge i.e., by the IN, VO, and CO scales. It may be influenced disproportionately by educational and cultural factors.

Figure 4.5

## WAIS-R Modal Profile 1

**Positive Pole**




**Negative Pole**

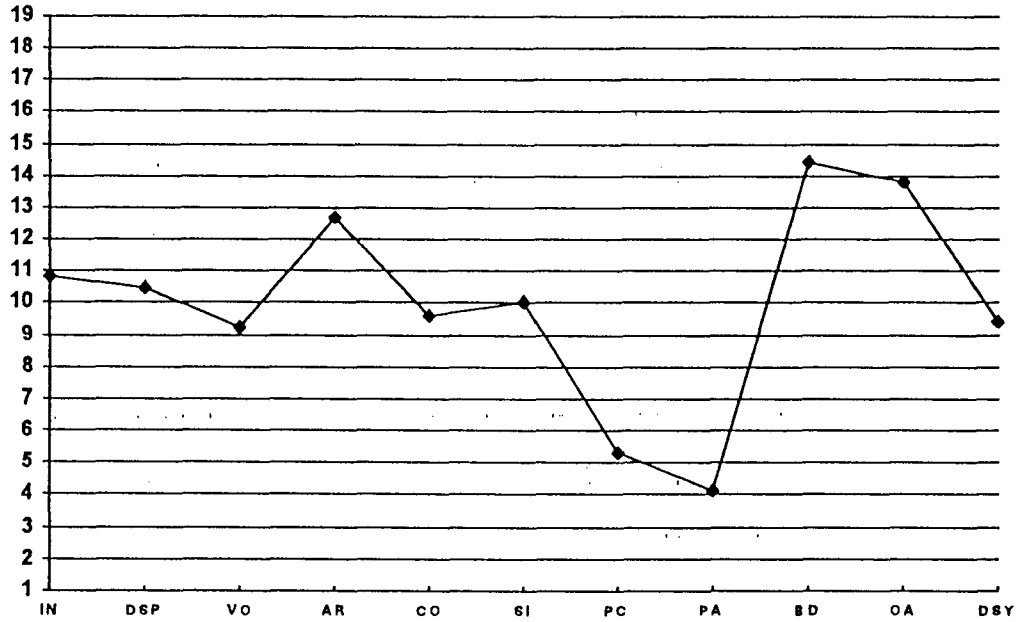
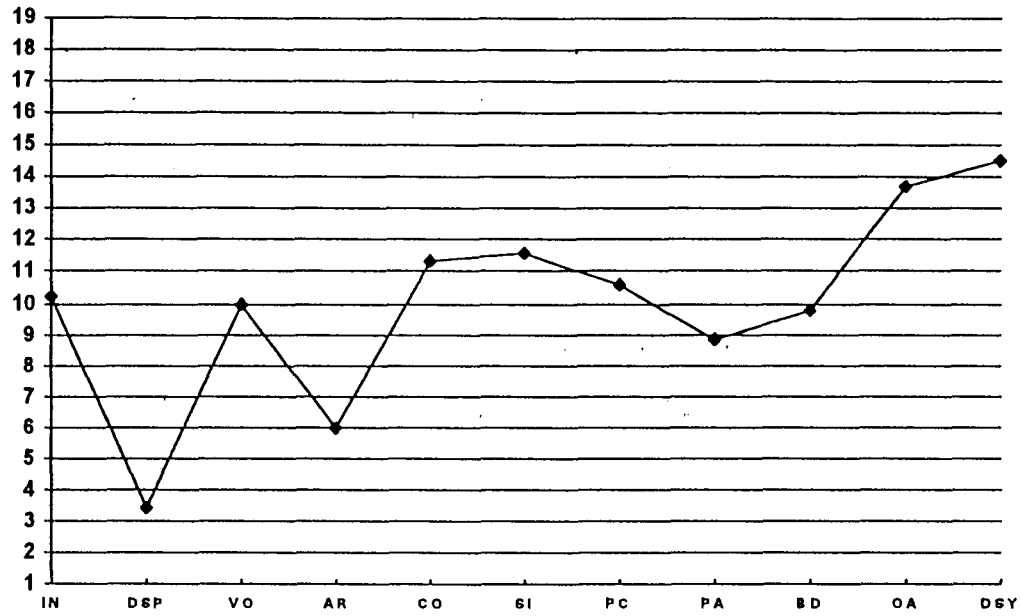


Figure 4.6

**WAIS-R Modal Profile 2**

**Positive Pole**




**Negative Pole**

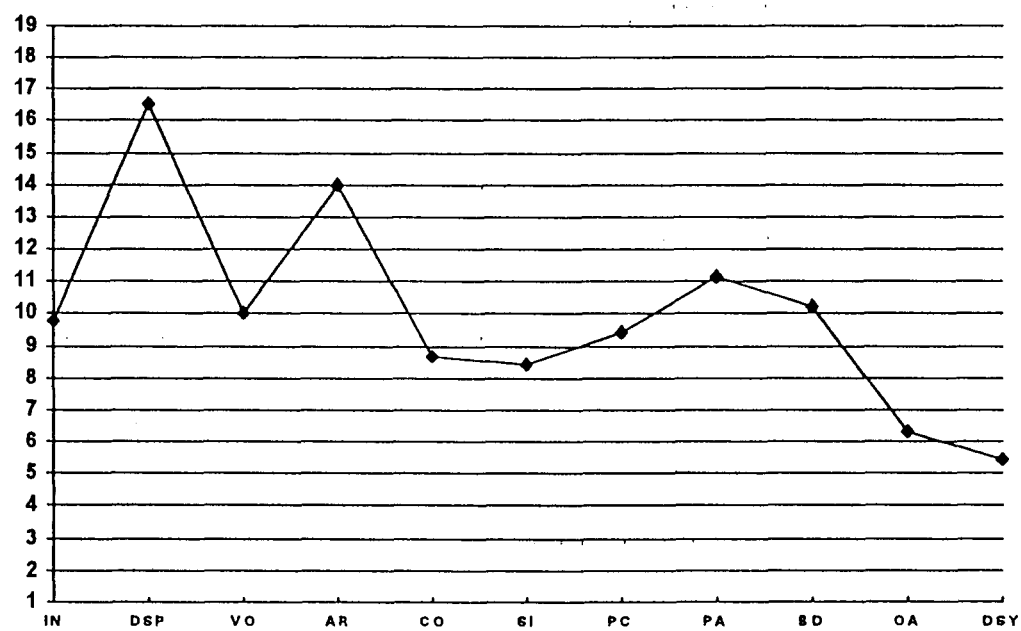
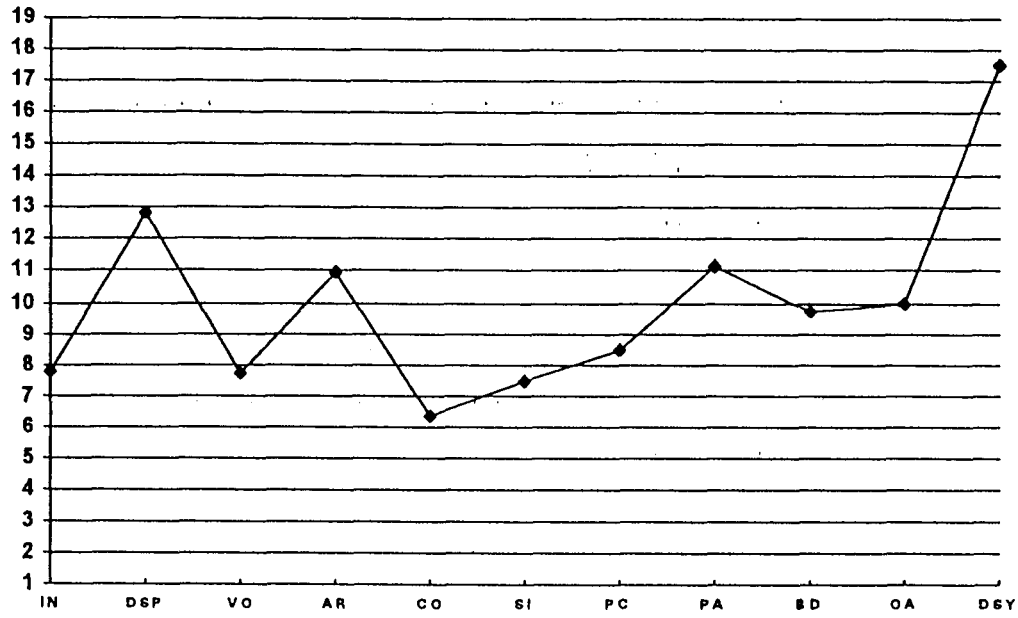


Figure 4.7

## WAIS-R Modal Profile 3

### Positive Pole




### Negative Pole

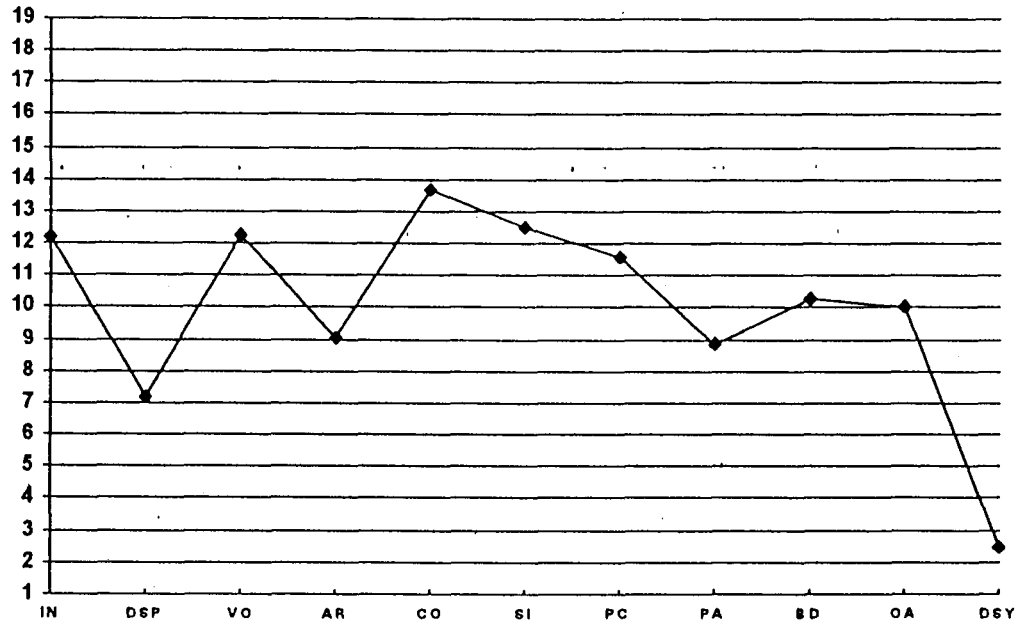
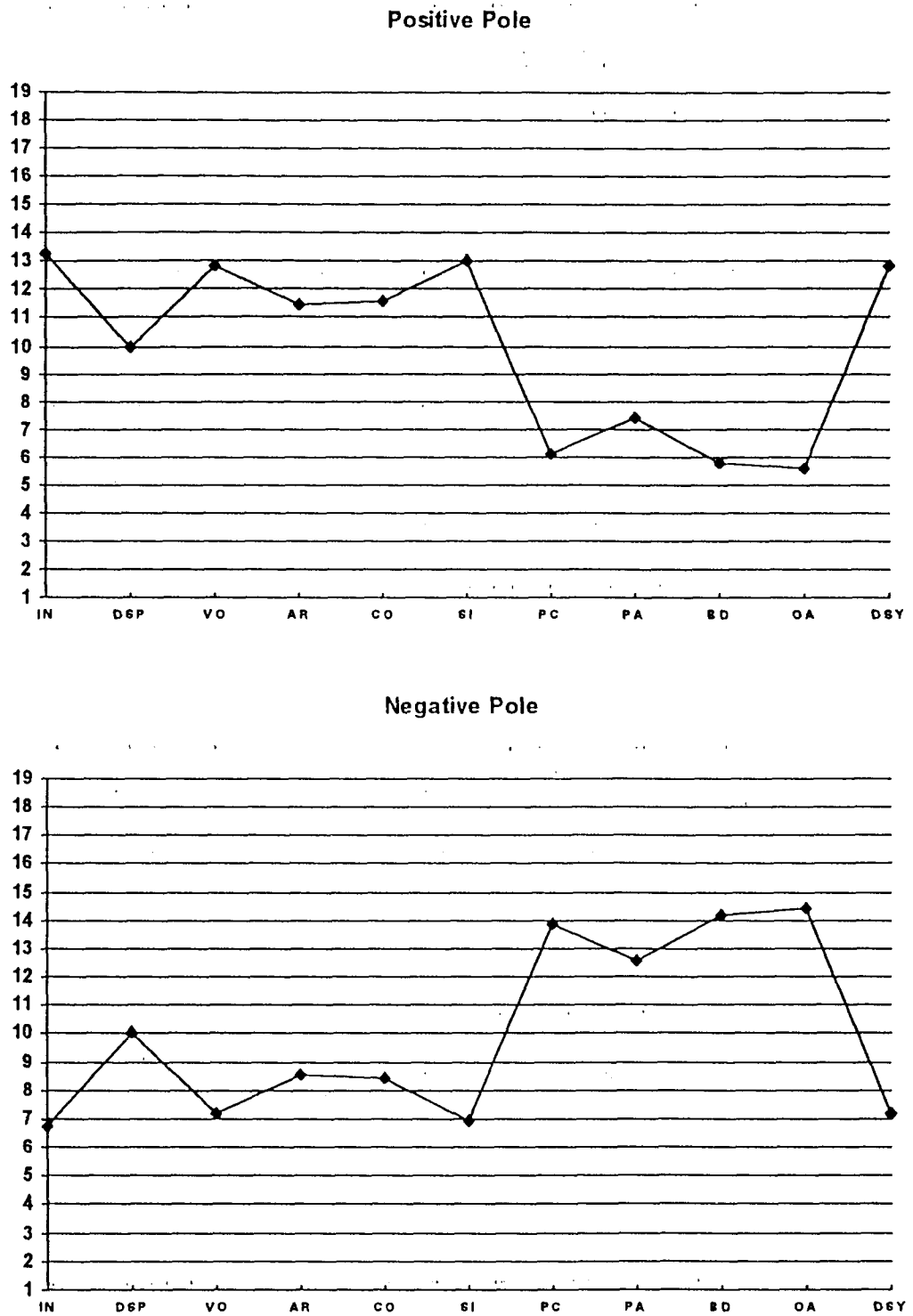


Figure 4.8

## WAIS-R Modal Profile 4



Lastly, Profile 4- was relatively infrequent in the NC group (3.2%), but common among the OF group (28.7%) and the PP group (24.6%). Thisprofile reflects poor verbal abilities and poor psychomotor abilities relative to visual spatial perceptual organizational ability, as represented among the Wechsler scales.

TABLE 4.21

Wechsler Profile Classification by Sample

| Profile Type | SAMPLE NC | | OF | | PP | | Total | |
|---|---|---|---|---|---|---|---|---|
| | N | (%) | N | (%) | N | (%) | N | (%) |
| 1+ | 4 | (4.3) | 25 | (4.8) | 10 | (2.6) | 39 | (3.9) |
| 1- | 16 | (17) | 39 | (7.6) | 40 | (10.2) | 95 | (9.5) |
| 2+ | 5 | (5.3) | 14 | (2.7) | 12 | (3.1) | 31 | (3.1) |
| 2- | 22 | (23.4) | 83 | (16.1) | 62 | (15.8) | 167 | (16.7) |
| 3+ | 7 | (7.4) | 34 | (6.6) | 17 | (4.3) | 58 | (5.8) |
| 3- | 20 | (21.3) | 95 | (18.4) | 88 | (22.4) | 203 | (20.3) |
| 4+ | 7 | (7.4) | 25 | (4.8) | 32 | (8.2) | 64 | (6.4) |
| 4- | 3 | (3.2) | 148 | (28.7) | 95 | (24.2) | 246 | (24.6) |
| Not Classified | 10 | (10.6) | 53 | (10.3) | 36 | (9.2) | 99 | (9.9) |
| | Σ94 | | Σ516 | | Σ392 | | Σ1002 | |
| | %9.4 | | %51.5 | | %39.1 | | %100 | |

Wechsler Impairment Analysis

Table 4.22 provides Wechsler scale scores for each group (NC, OF, and PP) on each of the eight Wechsler profiles. The table is self-explanatory and intended for normative reference. A persistent pattern indicating consistently poorer performance of the OF group, but particularly the PP group relative to the NC group, is readily ascertained. The differences are generally robust, e.g., consider the differential performance of the three groups classified to Profile 2- on DSY. The NC group had a scale score of 9.45, the OF group, 7.17, and the PP group, 5.81. These scores are .84, 1.65, and 2.14 *SDs* below the NC mean. Next, Wechsler/neuropsychological intersections are discussed.

TABLE 4.22

Wechsler Impairment Analysis

MEAN SCALE SCORES

| Profiles | Grp | IN | DSP | VO | AR | CO | SI | PC | PA | BD | OA | DSY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| -1 | NC | 11.00 | 8.00 | 13.25 | 11.00 | 11.25 | 10.50 | 13.00 | 14.25 | 9.50 | 9.25 | 12.00 |
| -1 | OF | 8.82 | 9.18 | 8.23 | 10.36 | 9.77 | 9.26 | 7.90 | 6.77 | 11.08 | 10.69 | 8.00 |
| -1 | PP | 8.70 | 8.40 | 8.70 | 7.60 | 8.00 | 7.40 | 10.40 | 10.90 | 7.00 | 7.70 | 7.50 |
| +1 | NC | 12.25 | 12.37 | 11.25 | 13.44 | 10.94 | 12.31 | 10.06 | 9.31 | 14.50 | 13.75 | 11.56 |
| +1 | OF | 8.84 | 9.32 | 8.88 | 8.12 | 9.44 | 9.32 | 11.16 | 12.12 | 8.40 | 7.12 | 8.64 |
| +1 | PP | 9.07 | 9.10 | 8.72 | 9.65 | 7.80 | 7.75 | 6.63 | 6.43 | 11.23 | 10.02 | 6.95 |
| -2 | NC | 11.14 | 14.27 | 12.09 | 13.45 | 11.27 | 10.05 | 10.45 | 12.91 | 12.00 | 9.73 | 9.45 |
| -2 | OF | 9.06 | 12.64 | 9.00 | 10.66 | 8.89 | 8.67 | 8.81 | 9.31 | 9.43 | 7.70 | 7.17 |
| -2 | PP | 8.02 | 10.45 | 8.42 | 9.98 | 7.61 | 7.61 | 7.06 | 7.94 | 8.03 | 6.47 | 5.81 |
| +2 | NC | 11.40 | 8.20 | 11.00 | 9.60 | 11.60 | 13.00 | 11.60 | 11.80 | 11.20 | 13.60 | 13.60 |
| +2 | OF | 9.71 | 8.43 | 9.93 | 8.21 | 10.43 | 11.07 | 10.07 | 10.50 | 11.36 | 12.14 | 10.93 |
| +2 | PP | 7.33 | 5.92 | 8.08 | 6.50 | 8.17 | 9.72 | 7.33 | 7.92 | 9.17 | 10.00 | 9.00 |

MEAN SCALE SCORES

| Profiles | Grp | IN | DSP | VO | AR | CO | SI | PC | PA | BD | OA | DSY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| -3 | NC | 13.00 | 10.55 | 13.90 | 11.80 | 13.95 | 13.30 | 12.10 | 10.90 | 12.35 | 12.05 | 8.85 |
| -3 | OF | 11.14 | 8.99 | 10.73 | 9.89 | 12.11 | 10.95 | 10.97 | 9.69 | 10.35 | 10.29 | 6.74 |
| -3 | PP | 10.32 | 7.89 | 10.49 | 8.93 | 9.84 | 10.58 | 9.75 | 8.52 | 9.08 | 9.10 | 5.69 |
| +3 | NC | 10.57 | 11.86 | 11.43 | 12.86 | 10.71 | 11.00 | 11.57 | 12.14 | 12.86 | 11.71 | 14.86 |
| +3 | OF | 7.41 | 10.29 | 7.15 | 8.97 | 7.00 | 7.53 | 8.18 | 9.12 | 9.32 | 9.35 | 10.65 |
| +3 | PP | 7.00 | 9.06 | 7.00 | 8.18 | 6.53 | 6.71 | 7.06 | 8.18 | 9.53 | 7.65 | 10.71 |
| -4 | NC | 10.33 | 11.00 | 10.67 | 12.00 | 10.00 | 11.00 | 13.00 | 12.00 | 15.33 | 13.67 | 10.67 |
| -4 | OF | 7.05 | 9.23 | 7.40 | 8.20 | 7.84 | 7.72 | 10.78 | 10.22 | 11.12 | 10.98 | 7.47 |
| -4 | PP | 6.25 | 7.86 | 6.65 | 7.05 | 6.61 | 6.96 | 9.19 | 9.51 | 10.27 | 10.11 | 6.52 |
| +4 | NC | 12.43 | 10.71 | 12.29 | 11.71 | 11.00 | 13.71 | 9.00 | 9.14 | 9.71 | 9.00 | 11.71 |
| +4 | OF | 12.00 | 10.80 | 11.24 | 10.88 | 12.28 | 11.60 | 8.56 | 8.92 | 8.04 | 6.92 | 9.52 |
| +4 | PP | 10.72 | 9.06 | 10.63 | 9.28 | 8.97 | 10.28 | 6.69 | 7.06 | 7.53 | 5.87 | 7.91 |

Neuropsychological/Wechsler Modal Profile Intersection

In Section VII, it was noted that the Wechsler scales extension loadings on neuropsychological factors were generally quite modest ($\leq .40$). This finding indicates that the neuropsychological and Wechsler variables are relatively independent, or occupy only a partially overlapping factor space. By analyzing the intersection of neuropsychological and Wechsler profiles, it can be expected that some conjoint profiles will reflect this common space to a greater degree than others. Examination of these conjoint profiles indicates that in most instances, each subprofile yields independent information for interpretation.

Table 4.23 provides information regarding the intersection of neuropsychological modal profiles with Wechsler modal profiles. Consider cell one, located in the upper left-hand corner of the table. This cell represents the number of individuals across all groups (NC, OF, and PP) who were classified in profile 4- among neuropsychological profiles and profile 4+ among the Wechsler profiles. Examination of cell one indicates that 12 subjects were jointly classified in this fashion. Further, line 2 indicates that 18.8% of subjects classified on Wechsler Profile 4+ were also classified on neuropsychological Profile 4-, 18.8% neuropsychological Profile 3-, and so on. Line 3 indicates that 8.7% of subjects classified on neuropsychological Profile 4- were also classified on Wechsler Profile 4+. Line 4 represents the percentage among all subjects who share a particular neuropsychological profile and a particular Wechsler profile.

It is noted with respect to Table 4.23 that Wechsler profiles 2-, 3-, and 4- are the most populous. Profile 2- reflects high performance on the DSP subtest with poor performance on the OA and DSY subtests. Profile 3- reflects poor performance on DSY and DSP, and Profile 4- reflects low verbal abilities. Thus, these profiles emphasize the deficits noted, especially in the OF and PP groups, and are the most common Wechsler profiles.

Another observation is that, generally, any given Wechsler profile combines in relatively uniform proportions with all the other neuropsychological profiles. This pattern reflects the general independence of the two data sets. Any particular conjunction may be accounted for in terms of a shared portion of variance and otherwise provides additional information concerning a group with a common profile intersect. Consider, e.g., Wechsler Profile-4, which is defined by relatively high spatial organizational abilities in conjunction with relatively low verbal and psychomotor abilities. It is the most common profile among both the OF group (28.2%) and the PP group (24.6%), but uncommon among the NC group (3.4%). This profile is almost evenly distributed, with the exception of Profile 3-, among both the positive and negative poles of the neuropsychological profiles. If these two data sets were highly correlated, then such a distribution would appear counterintuitive. Consider the following intersects:

1. In the case of the intersect of Wechsler Profile 4- with neuropsychological Profile 2- (33 subjects), the joint profile is characterized

by relatively low verbal and psychomotor abilities with relatively high spatial perceptual organizational abilities on the Wechsler scales in combination with very good performance on neuropsychological Factor V, WCST perseveration, and somewhat poorer verbal dynamic abilities (Factor IV). The combination of Wechsler Profile 4- with the positive pole of the neuropsychological Profile 2+ consisting of 32 subjects reflects Wechsler Profile 4-, but now this profile is associated with somewhat better verbal processing abilities, and a high level of perseveration on the WCST. Perseveration on the WCST would appear to add a great deal of interpretative information concerning the potential for positive adaptation in these two groups. Subjects in the intersect (4-, 2+), for instance, may be much more impulsive and less susceptible to psychotherapy than those in group (4-, 2-).

2. The intersect of Wechsler Profile 4- with neuropsychological profiles 1+ and 1- presents another interesting contrast. The first group (4-, 1+), comprised of 32 subjects, is characterized by high perceptual reasoning and abstraction abilities, and concrete spatial perceptual organizational abilities despite relatively low verbal and psychomotor abilities. The second group (4-, 1-) also has good spatial perceptual organizational abilities, but poor spatial perceptual abstract abilities combined with relatively low verbal and psychomotor abilities as measured by the Wechsler scales. From an interpretive perspective, these two groups appear very different and group 2,

depending on the absolute level of function, could be expected to have much greater difficulty with adaptive functioning.

Above, with reference to four out of 64 possible group intersects, it has been illustrated that consideration of neuropsychological profiles, in addition to Wechsler profiles, significantly alters the overall neuropsychological interpretation, particularly by including variables more closely associated with higher intellectual processes and adaptive functioning. The potential relevance of employing the Wechsler characterizations of ability and neuropsychological measures of executive and adaptive functioning, empirically recovered as modal profiles here, ultimately depends on the external validation of these profiles (cf. Skinner, 1981). Neuropsychological theory suggests that the neuropsychological profiles may be more relevant to executive and adaptive functioning, while Wechsler scales may be particularly valuable in assessing some aspects of verbal/educational achievement and concrete spatial perceptual organizational ability.

TABLE 4.23

Neuropsychological/Wechsler Modal Profile Intersection

| Wechsler Modal Profiles | | NEUROPSYCHOLOGICAL MODAL PROFILES 4- | 3- | 2- | 1- | 1+ | 2+ | 3+ | 4+ | Row Total | Row % |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 4+ | N | 12 | 12 | 9 | 14 | 3 | 7 | | 7 | 64 | 6.4 |
| 4+ | Row % | 18.8 | 18.8 | 14.1 | 21.9 | 4.7 | 10.9 | | 10.9 | | |
| 4+ | Column % | 8.7 | 12.0 | 7.7 | 9.0 | 2.8 | 5.9 | | 4.4 | | |
| 4+ | Total % | 1.2 | 1.2 | .9 | 1.4 | .3 | .7 | | .7 | | |
| 3+ | N | 16 | 8 | 7 | 12 | 3 | 3 | 6 | 3 | 58 | 5.8 |
| 3+ | Row % | 27.6 | 13.8 | 12.1 | 20.7 | 5.2 | 5.2 | 10.3 | 5.2 | | |
| 3+ | Column % | 11.6 | 8.0 | 6.0 | 7.7 | 2.8 | 2.5 | 5.8 | 1.9 | | |
| 3+ | Total % | 1.6 | .8 | .7 | 1.2 | .3 | .3 | .6 | .3 | | |
| 2+ | N | 9 | 1 | 7 | 2 | 2 | 1 | 5 | 4 | 31 | 3.1 |
| 2+ | Row % | 29.0 | 3.2 | 22.6 | 6.5 | 6.5 | 3.2 | 16.1 | 12.9 | | |
| 2+ | Column % | 6.5 | 1.0 | 6.0 | 1.3 | 1.9 | .8 | 4.8 | 2.5 | | |
| 2+ | Total % | .9 | .1 | .7 | .2 | .2 | .1 | .5 | .4 | | |

NEUROPSYCHOLOGICAL MODAL PROFILES

| Wechsler Modal Profiles | | 4- | 3- | 2- | 1- | 1+ | 2+ | 3+ | 4+ | Row Total | Row % |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1+ | N | 2 | 9 | 5 | 9 | 4 | 3 | 2 | 5 | 39 | 3.9 |
| 1+ | Row % | 5.1 | 23.1 | 12.8 | 23.1 | 10.3 | 7.7 | 5.1 | 12.8 | | |
| 1+ | Column % | 1.4 | 9.0 | 4.3 | 5.8 | 3.7 | 2.5 | 1.9 | 3.1 | | |
| 1+ | Total % | .2 | .9 | .5 | .9 | .4 | .3 | .2 | .5 | | |
| Not Classified | N | 19 | 3 | 15 | 7 | 13 | 10 | 11 | 21 | 99 | 9.9 |
| Not Classified | Row % | 19.2 | 3.0 | 15.2 | 7.1 | 13.1 | 10.1 | 11.1 | 21.2 | | |
| Not Classified | Column % | 13.8 | 3.0 | 12.8 | 4.5 | 12.0 | 8.4 | 10.6 | 13.1 | | |
| Not Classified | Total % | 1.9 | .3 | 1.5 | .7 | 1.3 | 1.0 | 1.1 | 2.1 | | |
| 1- | N | 6 | 7 | 12 | 14 | 14 | 10 | 11 | 21 | 95 | 9.5 |
| 1- | Row % | 6.3 | 7.4 | 12.6 | 14.7 | 14.7 | 10.5 | 11.6 | 22.1 | | |
| 1- | Column % | 4.3 | 7.0 | 10.3 | 9.0 | 13.0 | 8.4 | 10.6 | 13.1 | | |
| 1- | Total % | .6 | .7 | 1.2 | 1.4 | 1.4 | 1.0 | 1.1 | 2.1 | | |
| 2- | N | 15 | 24 | 12 | 32 | 12 | 27 | 18 | 27 | 167 | 16.7 |
| 2- | Row % | 9.0 | 14.4 | 7.2 | 19.2 | 7.2 | 16.2 | 10.8 | 16.2 | | |
| 2- | Column % | 10.9 | 24.0 | 10.3 | 20.5 | 11.1 | 22.7 | 17.3 | 16.9 | | |

| Wechsler Modal Profiles | | NEUROPSYCHOLOGICAL MODAL PROFILES 4- | 3- | 2- | 1- | 1+ | 2+ | 3+ | 4+ | Row Total | Row % |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 2- | Total % | 1.5 | 2.4 | 1.2 | 3.2 | 1.2 | 2.7 | 1.8 | 2.7 | | |
| 3- | N | 26 | 23 | 17 | 32 | 25 | 26 | 18 | 36 | 203 | 20.3 |
| 3- | Row % | 12.8 | 11.3 | 8.4 | 15.8 | 12.3 | 12.8 | 8.9 | 17.7 | | |
| 3- | Column % | 18.8 | 23.0 | 14.5 | 20.5 | 23.1 | 21.8 | 17.3 | 22.5 | | |
| 3- | Total % | 2.6 | 2.3 | 1.7 | 3.2 | 2.5 | 2.6 | 1.8 | 3.6 | | |
| 4- | N | 33 | 13 | 33 | 34 | 32 | 32 | 33 | 36 | 246 | 24.6 |
| 4- | Row % | 13.4 | 5.3 | 13.4 | 13.8 | 13.0 | 13.0 | 13.4 | 14.6 | | |
| 4- | Column % | 23.9 | 13.0 | 28.2 | 21.8 | 29.6 | 26.9 | 31.7 | 22.5 | | |
| 4- | Total % | 3.3 | 1.3 | 3.3 | 3.4 | 3.2 | 3.2 | 3.3 | 3.6 | | |
| | Column Total | 138 | 100 | 117 | 156 | 108 | 119 | 104 | 160 | 1002 | |
| | Total % | 13.8 | 10.0 | 11.7 | 15.6 | 10.8 | 11.9 | 10.4 | 16.0 | | 100.0 |

## SECTION IX SUMMARY

Chapter IV reports on the results of statistical analyses conducted to develop a method of classifying the neuropsychological capacities of serious adult offenders and to assess the nature and extent of impairment observed in this population. The offender group consisted of 584 adult offenders incarcerated in the Canadian federal correctional system who underwent assessment at the Regional Psychiatric centre in Abbotsford, British Columbia. The study's methodology required a comparison group of normal controls and a group of acute psychiatric patients was included to further contrast the neuropsychological performance of the offenders. Data for 132 normal control and 494 acute psychiatric patients was provided by J. R. Reddon of the Alberta Hospital, Edmonton, Alberta. Measures included the Halstead-Reitan Neuropsychological Test Battery supplemented by several other neuropsychological tests (emphasizing verbal and memory abilities) and the Wechsler Intelligence Scales.

Two major Halstead-Reitan normative studies have been published in the past decade (Heaton et al., 1991 and Yeudall et al., 1987). Using the same group of normal controls as in Yeudall et al. (1987), Yeudall, Fromm, Reddon, and Stefanyk (1986) also published norms on the other measures included in this study. Accordingly, it seemed preferable to use both sets of Yeudall norms, since they were based on a common subject pool. Comparison of the Yeudall norms with the Heaton norms on 11 Halstead-

Reitan measures were made, and it was concluded that the respective norms were consistently comparable (Section III).

The next step (Section IV) was to examine whether differences in neuropsychological test performance could be attributable to age across the NC, OF, and PP groups. Age group-test performance correlations were examined for age groups 18-29 and 30-44 in each sample. In Section V, age group-test performance relationships were further examined in the OF group. Overall, in these analyses, it was found that age-group test performance correlations were not significant on most measures. Significant relationships were as follows: a) within the NC group, there was a tendency for psychomotor abilities and verbal working memory to decline between 18 and 44 years of age, although this trend was not as evident in the OF and PP groups; and b) within the OF group, the younger group was noted to be more proficient on tasks requiring perceptual motor speed and spatial perceptual organization, and this group had lower scores on some Wechsler verbal scales. In view of the few notable age effects, it was decided to collapse the age grouping factor in subsequent analyses.

Tables 4.4 and 4.5, presented in Section V, provided norms on all measures included in this study for offenders aged 18-29, 30-44, and 18-44.

In Section V, the relative performance of the NC, OF, and PP groups was compared across all measures. The most remarkable finding was that for almost all test performance measures, excepting some simple perceptual, sensory, and motor measures, the performance of the NC group was

significantly higher than that of the OF group, and, in turn, the OF group performed significantly better than the PP group. The overall level or degree of impairment was also noted to be substantial. Among the OF group, e.g., between 30% and 57% performed 1 *SD* below the NC mean and between 9 and 30% performed 2 *SDs* below this mean, depending on the neuropsychological measure considered. For Wechsler variables, relative levels of impairment were even greater, e.g., performance of the OF group was 1 *SD* below the NC group mean in 75%, 60%, and 78% of cases on Verbal IQ, Performance IQ, and Full Scale IQ, respectively. Corresponding percentages 2 *SDs* below the NC mean were 46%, 26%, and 45%.

Among the PP group the degree of impairment was exceptional. Across all of the neuropsychological measures examined, between 44% and 80% scored $\leq 1$ *SD* below the control mean and between 21% and 55% scored $\leq 2$ *SDs* below this mean.

On the Wechsler Full Scale IQ composite measure, 85% of the PP group scored $\leq 1$ *SD* below the control mean and the performance of 59% of the PP group was $\leq 2$ *SDs* below this mean.

In Section VII, a representative subset (28 measures) of the neuropsychological battery that discriminated between the performance of the NC, OF, and PP groups (NC > OF > PP) were factor analysed. A five factor Principal Components, followed by Varimax solution, was conducted for the three groups. A factor congruence analysis across these groups indicated a high degree of concordance of factor structure across groups and

a combined 5 factor solution accounting for 58.9% of the test variance was interpreted. Simple structure was impressive and the common neuropsychological capacity underlying each factor was abstracted. Factor I was interpreted to reflect Sensory Motor Spatial Perceptual Organization and this factor was especially marked by high loadings from the Tactual Performance Test. These abilities have traditionally been inferred to relate primarily to non-dominant temporal and parietal systems. Factor II, labelled Nonverbal Perceptual Reasoning and Abstraction, was mainly defined by performance on the Halstead Category Test. The implicit task requirements of this factor mainly include nonverbal perceptual abstraction, logical analysis, and the capacity to adapt flexibly to feedback of changing problem definition. Considered from the perspective of neuropsychological theory, the Category Test represents an exemplar of executive function in the nonverbal perceptual sphere. The complexity of the task places demands on lower brain systems, e.g., nonverbal attention and spatial perceptual organization, thus, dysfunction of lower processing systems could interfere with performance on this factor, nonetheless, theory of the role of the prefrontal cortex suggests that it is critically implicated in this task.

Factor III was especially marked by loadings of Purdue Pegboard measures. Task analysis indicates that a capacity to flexibly resolve the timing of sensory motor relationships is required. The factor was labelled Fine Temporal Perceptual Motor Speed. Research has suggested that systems for performance on this task are likely widely distributed, but the

prefrontal cortex may contribute to the development and coordination of these systems. The fourth factor was labelled Dynamic Verbal Processing. Tests loading on this factor reflect the capacity to sustain attention (e.g., the Speech Sounds Perception Test), but also flexibly shift attention (e.g., the Trails B Test and Wisconsin Card Sorting Test), although all tests that implicate verbal abilities load on this factor. Overall, verbal processing is involved and, neuropsychologically, frontal verbal-attentional and posterior verbal systems appear to be mainly involved. The fifth factor labelled WCST Perseveration was fairly exclusively defined by overall success on the WCST and especially, perseverative errors on this test. The WCST is factorially complex and success on this measure requires a number of distinguishable abilities, e.g., the formation of verbal concepts and maintaining and shifting set. Thus, this factor appears to isolate the capacity to shift set and respond according to a different principle, once information is provided that the previous response is inconsistent with the 'now' operative set. Research reviewed in Chapter III suggests that the dorsolateral prefrontal cortex may be critically implicated, although damage elsewhere may also interfere with this capacity to shift set and change behavioral response.

The neuropsychological variables not selected for inclusion in the above factor analysis and Wechsler variables were projected onto the factor space by way of extension loadings. Three observations are noted with respect to these extension loadings: a) sensory perceptual measures were inclined to load on Factor I, Sensory Motor Spatial Perceptual Organization, likely

reflecting the sensory motor and perceptual requirements of this factor; b) notable Wechsler scale loadings were primarily on Factors I, II, and IV, reflecting the spatial perceptual requirements of the first two factors and the verbal aspects of Factor IV. Loadings, however, were $\leq .40$ indicating relative independence of these two data sets. Further, the highest loading of any Wechsler scale with Factor V, WCST perseveration, was Performance IQ (-.16), indicating the independence of Wechsler intelligence measures and WCST perseveration; and c) age was not highly associated with any factor confirming the general finding of few and limited age effects noted above. Lastly, handedness, as measured by the Annett measures, was not remarkably associated with any neuropsychological factor.

In Section VII, a Modal Profile Analysis was applied to the five neuropsychological factors derived in Section VI. The results of this analysis indicated that 100% of subjects could be classified in terms of four bipolar profiles. The first profile especially emphasized differential abilities along Factor II, Nonverbal Perceptual Reasoning and Abstraction. The second profile distinguished groups with relatively high/low WCST perseveration in contrast to relatively low/high verbal processing abilities. Profile 3 classified a group characterized by relatively high Sensory Motor Spatial Organization (Factor I) combined with poor dynamic verbal processing abilities (Factor IV) and a group with a converse ability profile. The fourth profile reflected a group with remarkably high (in relative terms) Fine Temporal Perceptual

Motor Speed (Factor III) and above average abilities across the other four factors. The negative pole of this profile reflected a converse ability profile.

An inspection of classification patterns across the NC, OF, and PP groups indicated that no profile type was highly associated with any group, however, an impairment analysis indicated a high level of group differentiation when profile elevation by profile type was considered. It was suggested that a severe impairment on any profile aspect could produce global impairment. It was concluded that both the OF and PP group are characterized by high levels of impairment, although the highest level of impairment was noted in the PP group.

A Modal Profile Analysis was also conducted on the Wechsler subtest scales. A four profile solution classifying 90% of subjects was selected as most informative and interpretable. The first profile identified two groups of subjects whose performance among the Wechsler performance subtests was highly variable. One group was characterized by relatively high performance on the PC and PA subtests compared to relatively low performance on the BD and OA subtests. The other group had a converse pattern. The ability to verbally mediate visual spatial relations was hypothesized to discriminate the two groups. The second profile identified a group characterized by relatively high verbal attention (DSP and AR) and relatively low motor dexterity (OA and DSY), and a second group with the opposite performance pattern. The third profile reflected divergent relative performance on measures of simple attention and psychomotor ability (DSP and DSY) with acquired verbal

abilities (IN, VO, and CO). The fourth Wechsler profile generally discriminated subjects with differential visuospatial organizational abilities (PC, PA, BD, and OA) and verbal abilities (IN, VO, AR, CO, and SI). This profile thus reflected groups with large Verbal IQ - Performance IQ splits.

Classification analysis indicated, that unlike the case with neuropsychological profiles, there were associations between Wechsler profile type and group (NC, OF, and PP) membership. The most notable association was relative to Profile 4- which reflects high performance on visuo-spatial abilities relative to verbal and psychomotor abilities. This profile was uncommon among the NC group, but 28.7% of the OF group and 24.6% of the PP group were classified into this profile.

The modal profiles accentuate discrepant performance across subtests. A table (4.22) reflecting the scale scores for each group for each profile subtype was provided for reference. As indicated in Section VI, the relative contrast in performance of the OF and PP groups is greater for Wechsler scales than for the more dynamic neuropsychological variables.

The last portion of the analysis examined the intersection of neuropsychological and Wechsler profiles. A general observation was that any given Wechsler profile combines in relatively uniform proportions with all the neuropsychological profiles. Thus, classification on Wechsler profiles does not predict neuropsychological profile subtypes and vice versa. In other words, the information provided by the two classification systems is relatively independent and additive or cumulative. By way of illustration,

examples of profile intersects were discussed. Neuropsychologically, it was suggested that the neuropsychological profiles tend to reflect executive and adaptive functioning, emphasising prefrontal cortical function, while the Wechsler profiles are more related to temporal/parietal brain function.

This concludes the summary of empirical findings. The purpose of the next chapter is to integrate these findings and consider their significance for research and clinical applications.

# CHAPTER V DISCUSSION AND CONCLUSION

## SECTION I INTRODUCTION

This chapter is divided into four sections. Further to this introductory section, Section II discusses the main findings of this research and Section III addresses proposals and priorities for research. In Section IV, conclusions regarding the potential role of neuropsychology for improving services to offenders, understanding their development, and for the early identification and remediation of high risk children are presented.

## SECTION II DIMENSIONS OF NEUROPSYCHOLOGICAL CAPACITY

Increasingly, neuropsychological capacities are viewed as the product of brain systems organized both laterally and hierarchically (cf. Damasio, 1985; Derryberry & Tucker, 1992; Fuster, 1993; Luria, 1980; Stuss & Benson, 1984b). This conceptualization suggests that success on particular neuropsychological measures can be construed as a function of relatively discrete processes at differing hierarchical levels, but also dependent on the lateral and hierarchical integration of neural systems. Compromised success on a given test could be due to relatively localized specific dysfunctions or discontinuities in more broadly integrated systems. This general perspective on neural organization, e.g., is illustrated by Stuss and Benson's (1986) representation of the functional organizational abilities (see figure 2.1). This is the context for discussing the factor structure of neuropsychological abilities empirically recovered in this study.

The domain representation of neuropsychological functions in this study was defined by selecting measures commonly used in clinical neuropsychological assessments. Most prominent were the measures of the Halstead-Reitan Neuropsychological Battery, supplemented mainly by tests of verbal and analytic ability, e.g., Williams' Paired Associates Test and the WCST, as well as the Wechsler scales. In the factor analysis of neuropsychological variables, measures of simple sensory and motor function were excluded because they were not found to highly discriminate between the offender group (OF) and the normal control group (NC). This was consistent with the hypothesis that differences between these groups should be mainly reflected on measures of verbal and executive function capacities.

Nonverbal Spatial Perceptual Processing

Nonverbal spatial perceptual processing abilities are highly represented in the Halstead-Reitan Battery and among the Wechsler scales. Three primary dimensions related to these abilities were identified. In the factor analysis of neuropsychological variables (Chapter IV, Section VII) Factor I, Sensory Motor Perceptual Organization and Factor II, Nonverbal Perceptual Reasoning and Abstraction appear relevant. Factor I was particularly marked by performance on the Tactual Performance Test (TPT). This measure requires spatial perceptual analysis derived entirely through haptic (tactual) sensory input. Thus, there is no visual contamination. In this respect, it is different from the visual spatial perceptual tasks of the Wechsler scales which appear

to rely mainly on visual sensory input, e.g., Block Design. In the factor analysis, the extension loadings of Block Design and Object Assembly on this factor were only -.42 and -.36 respectively, suggesting that the sensory registration modality (haptic vs. visual) is an important element to consider in evaluating perceptual organizational ability. Also, among the Wechsler Performance scales, a subset of individuals who performed well on Block Design and Object Assembly did poorly on the Picture Completion and Picture Arrangement subtests. The differential element here is likely the verbal mediation requirement of these latter subtests. Thus, while Factor I and the main Wechsler Performance subtests requires spatial perceptual organizational ability, the differences in sensory input modalities, haptic and visual, and the requirement of verbal mediation, also affect spatial perceptual organizational ability.

A major common dimension tapped by the measures included here appears to be the ability to organize the relevant aspects of a perceptual field in relation to both sensory and motor behavior. A spatial perceptual organizational factor has typically been recovered in neuropsychological factor analyses (e.g., Corrigan & Hinkeldey, 1988; Leonberger et al., 1990; Moehle et al., 1990; Royce et al., 1976; Swiercinsky & Howard, 1982) and both the TPT and Wechsler performance subtests have been associated with such a factor when they were both included. Traditionally, this ability has been interpreted to be dependent primarily on non-dominant temporal and parietal lobe structures. Deficits in spatial perceptual organizational ability

among brain damaged populations has been related to damage to these structures (cf. Royce et al., 1976).

Although the Halstead Category Test (HCT) was identified with a separate factor in this study, it also has typically loaded on a broad spatial perceptual organizational factor. The HCT requires the representation and abstract re-representation, logical analysis, and manipulation of organized visual spatial perceptions. Thus, success on it can be expected to depend on the temporal/parietal structures noted, but its appearance as a separate factor here suggests that 'concrete' spatial perceptual processing is a necessary, but insufficient basis to perform the HCT. Theory regarding the role of the prefrontal cortex would suggest that its integrity is critical to succeed on the HCT, as the task is an exemplar of nonverbal executive function. This interpretation could account for the significant, but modest loading (.37) of a concrete visual-spatial perceptual task, such as Block Design with Factor II. It seems reasonable to assume that the capacities required to perform Block Design are subsumed by the more complex requirements of the HCT. This interpretation is consistent with the notion of hierarchical organization of neural systems hypothesized earlier. Such an interpretation could also account for the observation (cf. Reitan & Wolfson, 1993) that the HCT is highly sensitive to damage in many brain areas and not specific to lesions in the prefrontal cortex. Research has noted the high sensitivity of the HCT to brain damage, but not a high specificity to anterior lesions. However, the critical question concerning the conditional probability

of success on the HCT, given prefrontal lesions, has not been specifically evaluated. A hierarchical model that views Factor I and Wechsler Performance subtests as reflecting primarily right temporal/parietal function and performance on the HCT as requiring prefrontal integrity, in addition, is proposed. In this context, factors which have included measures of spatial perceptual organization from the Halstead-Reitan Battery, the Wechsler scales, and the HCT are viewed as second order factors confounding the sensory modality for spatial perceptual organization and the level of analysis - concrete vs. abstract.

Verbal Abilities

Verbal abilities are represented in this analysis by Factor IV labelled Dynamic Verbal Processing and by the Wechsler verbal subtests. Factor IV appears to reflect abilities to: a) attend to auditory stimuli, e.g., as required by the Seashore Speech Sounds Test and Seashore Rhythm Test; b) concentrate and retain material in short-term memory, e.g., as required by the Williams' measures of verbal and nonverbal learning; and c) to maintain attentional set, but also flexibly shift sets, e.g., as required by the WCST and Trails B. However, the perseveration measure of the WCST does not load on this factor, thus shifting set as a function of logical analysis may not be an essential feature of this factor. Among the Wechsler Verbal subtests, Digit Span and Vocabulary had the highest loadings. Neuropsychologically, verbal-auditory attentional abilities appear to be especially represented by this factor and a dominant superior temporal-prefrontal processing system is

hypothesized to be responsible (cf. Reitan, Hom, & Wolfson, 1988; Stuss & Benson, 1986; Swiercinsky, 1979) in conjunction with prefrontal attentional selection and control mechanisms (cf. Cohen et al., 1993).

Modal Profile Analysis of the Wechsler scales found (Profile 3) that performance on a group of subtests involving attention and flexible processing (Digit Span and Digit Symbol) was independent of the ability to achieve on verbal subtests reflecting acquired verbal abilities in a substantial proportion of subjects. The subtests reflecting acquired verbal abilities include Information, Vocabulary, Comprehension, and Similarities.

In sum, among verbal abilities, Factor IV, Dynamic Verbal Processing, and two Wechsler subtests, Digit Span and Digit Symbol, seem to reflect verbal attention and flexible processing of verbal material, while the Wechsler Information, Vocabulary, Comprehension, and Similarities subtests reflect acquired verbal abilities. The relatively low loadings of most of these scales on Factor IV reflect relative independence of these two aspects of verbal ability.

Perseveration

Factor V, Wisconsin Card Sorting Test Perseveration, has not previously emerged as a separate factor. Rather, this measure of the WCST has typically been absorbed within a broad verbal ability factor. The identification of perseveration as a separate dimension, relatively independent of set maintenance and verbal ability is, however, consistent with theoretical positions, e.g., Fuster (1989) who attributes perseveration

to the dorsolateral prefrontal cortex and set maintenance to the orbitofrontal cortex. As well, Richards, Cote, and Stern (1993) have demonstrated a set shifting difficulty in Parkinsons' patients which they attribute to striatal dysfunction. Eslinger and Grattan (1993) proposed that set shifting on the WCST relates to external demands and is to be distinguished from spontaneous set shifting. This particular factor may isolate a specific shifting problem in relation to external demands. Such difficulties could be expected to be highly relevant to adaptive functioning.

Perceptual Motor Speed

Factor III, Fine Temporal Perceptual Motor Speed, implicates neural systems responsible for the temporal resolution and coordination of fine motor-sensory processing. Likely, these systems are diffusely distributed, however, coordination at the highest levels can reasonably be hypothesized to involve prefrontal structures. The relationship of these systems to psychomotor activities such as reading, information processing, or verbal fluency has not been clearly established, but the loadings of the Digit Symbol and Oral Word Fluency measures on this factor suggest a relationship.

Wechsler Scales

The factorial structure of the Wechsler scales can be inferred, to some extent, from results of the Modal Profile Analysis of these scales. Profile 1 indicated that performance on the Picture Completion and Picture Arrangement subtests can be relatively independent of the ability to succeed

on the Block Design and Object Assembly subtests. Thus, it was suggested that visual-spatial organizational ability can be further differentiated in terms of whether verbal mediation is required. Profile 2 indicated, especially, that relatively high performance on the Object Assembly and Digit Symbol can occur despite relatively low performance on the Digit Span and Arithmetic subtests. Perhaps where sensory motor perceptual skills are high, verbal attentional abilities are not as critical for success on the Object Assembly and Digit Symbol subtests. Profile 3 indicated that attentional and psychomotor abilities can, in certain cases, be relatively independent of acquired verbal abilities. Profile 4 indicated the relative independence of acquired verbal abilities and spatial perceptual organizational abilities.

Summary

Several relatively independent dimensions of neuropsychological ability were identified in this study. The major areas reflected appear to include:

1. Spatial perceptual organizational abilities as measured by Factor I (Nonverbal Spatial Perceptual Processing) which is primarily based on haptic sensory input, the Wechsler Block Design and Object Assembly subtests which rely on visual-sensory input, and the Picture Completion and Picture Arrangement subtests which appear to require verbal mediation of spatial relations.

2. Acquired verbal abilities. These abilities seem to be especially represented by the Wechsler Information, Comprehension, Vocabulary, and Similarities subtests. Both spatial perceptual organizational and acquired

verbal abilities have traditionally been associated with non-dominant and dominant posterior brain regions respectively and are not usually seriously compromised by prefrontal damage (cf. Stuss & Benson, 1986, pp. 196-198).

3. Verbal attention and processing. This capacity appears to be especially represented by Factor IV (Dynamic Verbal Processing) and appears to involve the ability to attend to verbal material, to maintain problem set, and to shift attention spontaneously. The Wechsler Digit Span and Digit Symbol subtests also appear to be related to this ability complex. Traditionally, auditory attention has been related to superior anterior temporal structures, while set maintenance and the capacity to shift spontaneously have been characterized as primarily associated with orbital prefrontal brain regions. It seems reasonable to hypothesize that systems involving these two regions collaborate in dynamic verbal processing.

4. Temporal perceptual motor speed. This dimension, characterized primarily by Factor III (Fine Temporal Perceptual Motor Speed) has been hypothesized to depend on widely distributed sensory motor perceptual systems, perhaps with their highest level of integration in the prefrontal cortex.

5. Executive function. Executive function has been defined as the capacity to formulate goals, plan, and systematically carry out and monitor progress toward a goal. It is dependent, in the case of complex goals, on the integration of several cognitive operations, including logical analysis,

abstraction, behavioral inhibition, planning, and translating thought into action. The review of the role of the prefrontal cortex presented in Chapter II concluded that it is critically involved in executive function. While many cognitive subfunctions, e.g., recency discrimination and oral word fluency, have been associated with prefrontal function, the capacity to anticipate, to select goals, to plan and monitor, and to use feedback are seen as crucial to goal attainment. In clinical neuropsychology, the Wisconsin Card Sorting Test and the Halstead Category Test reviewed in Chapter III have been traditionally viewed as complex measures of executive function. In the present study, Factor II (Nonverbal Perceptual Reasoning and Abstraction) was essentially defined by the Halstead Category Test, and Factor V (Wisconsin Card Sorting Test Perseveration) primarily reflects perseveration on this test. Accordingly, these two factors are seen to reflect aspects of executive function and to be dependent on prefrontal executive systems.

The major dimensions of neuropsychological ability discussed above are hypothesized to be reflections of relatively independent neural systems. If this is in fact the case, then individuals can be expected to vary in terms of their relative abilities across these dimensions.

Intra-Individual Organization of Abilities

Above, a dimensional array of neuropsychological capacities was discussed. A further question in this study was whether individuals could be grouped according to different patterns of ability. This was addressed

through Modal Profile Analysis (MPA). MPA of the neuropsychological factors produced four bipolar profiles classifying 100% of the participants in this study.

The first profile identified a group with divergent nonverbal perceptual reasoning and abstraction abilities relative to their performance on the other four neuropsychological factors. The second profile distinguished groups on the basis of relatively high or low WCST perseveration in contrast to low or high verbal processing abilities respectively. The third profile defined a group characterized by relatively high sensory motor spatial organizational abilities in contrast to their verbal processing abilities and a second group with a converse ability profile. The fourth profile classified groups with relatively high scores on Factor III (Fine Temporal Perceptual Motor Speed) and also relatively high abilities across the other dimensions. The negative pole of this profile characterized a group with a converse ability profile.

MPA of the Wechsler scales also resulted in four bipolar modal profiles. The first profile identified two groups who mainly had divergent performance among the Performance subtests of the WAIS-R. One group had relatively high performance on the PC and PA subtests combined with relatively poor performance on the BD and OA subtests. The group classified to the opposite pole had a converse ability profile. The second profile contrasted two groups with divergent verbal attentional (DSP and AR) abilities as compared with their motor dexterity (OA and DSY). The third profile discriminated a group with relatively low simple attention and psychomotor

ability (DSP and DSY) combined with relatively high acquired verbal abilities (IN, VO, and CO). The fourth Wechsler profile classified a group with relatively high visual spatial perceptual organizational abilities (PC, PA, BD, and OA), but relatively low verbal abilities (IN, VO, AR, CO, and SI) and psychomotor ability (DSY).

An individual's overall neuropsychological profile can be characterized by the conjunction of his neuropsychological and Wechsler profile. The evidence in this study and others (e.g., Kupke & Lewis, 1985; Moses, 1984a, 1984b) is that the abilities captured by the Wechsler scales and neuropsychological batteries are relatively independent. Moses (1984a), for example, found that IQ measures predicted 18% of the Halstead Reitan Neuropsychological Battery measures on average. In this study, the highest loading of any Wechsler measure with any neuropsychological factor was $\leq .40$. This strongly suggests that assessments and predictions based on Wechsler scales alone could be very misleading and the authors cited above recommended the use of both Wechsler and neuropsychological batteries when conducting assessments.

Impairment Levels

Specifying an individual's relative performance across neuropsychological and Wechsler variables, which modal profiles summarize, remains an incomplete characterization of ability. To complete this, three additional components must be added: 1) the degree of deviation (scatter) of the individual's profile from the modal profile, 2) the elevation of the profile

which specifies the level of absolute performance across the profile, and 3) normative characterization of the performance level. With respect to a given neuropsychological profile, the specific correlation of an individual's profile can be computed and the level of performance across the profile can be specified in normative terms. In the present study, the general normative level of performance for an individual on each of the factors or factor markers or in the case of the Wechsler scales, scale scores could be determined. Perhaps, if further research of groups within profiles were to be conducted, the performance of groups could be subdivided in terms of arbitrary divisions, e.g., lowest third, middle third, and highest third, or other performance criteria.

Among the neuropsychological profiles, profile type was not strongly associated with group membership. Discrimination of groups, therefore, depends mainly on level of performance within profile subtypes. As noted earlier, the size of the NC group does not justify such a breakdown. Nevertheless, the analyses conducted in Chapter IV, Section V indicated that the OF group is characterized by a high level of deficit across nearly all neuropsychological measures examined and the Wechsler scales. The performance, on average, of the PP group was considerably worse than that of the OF group. To reiterate, depending on the neuropsychological measure selected, between 30% and 57% of the OF group performed 1 *SD* below the NC mean and between 9% and 30% performed 2 *SDs* below this mean. On Wechsler measures, relative impairment was even greater. The performance

of the OF group was 1 *SD* below the NC group mean in 75%, 60%, and 78% of cases on Verbal IQ, Performance IQ, and Full Scale IQ respectively. As well, corresponding percentages 2 *SDs* below the NC mean were 46%, 26%, and 45%. Among the PP group, between 21% and 55% scored $\leq$2 *SDs* below the NC mean over the range of neuropsychological measures and 59% had a Full Scale IQ $\leq$2 *SDs* below the NC mean.

A comparison of the OF and PP groups on the Category Test discussed in Chapter IV, Section VI concluded that, on average, the performance of the OF group was comparable to younger neurological patients with a moderate degree of impairment across a variety of etiological groups. In contrast, the performance of the PP group on the Category Test was clearly higher than that typically reported for psychiatric patients. Indeed, their mean performance on this measure was nearly as impaired as the average for all brain damaged patients, including older patients reported on by Gaskin (1989).

The Category Test appears to represent the most sensitive single measure of brain impairment (Reitan & Wolfson, 1993). The fact that the OF group appears to score as poorly as many groups of neurological patients of similar age with demonstrated brain damage does not indicate that they are brain damaged. It does, however, suggest that their functional disabilities are highly significant and may be directly linked to adaptive and self-control deficits. These deficits, in turn, can reasonably be construed to represent risk factors for offending, directly or indirectly. In the case of brain

trauma, the adaptive deficits associated with dysfunction on neuropsychological tests, such as the Category Test, have been demonstrated to be highly destructive (cf. Stuss & Benson, 1986). Similar levels of test performance among offenders, it is argued, may indeed represent an even greater overall impact, since these deficits in all likelihood have been operative throughout development.

Only recently (cf. Barkley, 1990; Hinshaw, 1994a,b; Moffitt & Lynam, 1994), has it become clear that childhood diagnoses of Conduct Disorder and Attention Deficit Hyperactivity Disorder carry with them a high risk for general maladjustment and a highly disproportionate risk for antisocial behavior. These authors have proposed that neuropsychological deficit, particularly related to executive function and verbal abilities, may be critical in mediating these risks. This study has clearly indicated the presence of a high level of deficit in both executive function and verbal abilities among serious adult offenders. It thus appears that these deficits present some risk for antisocial behavior. It would seem important to conduct studies further characterizing the nature of the risk, protective factors, and the effects of interventions on negative developmental trajectories. As well, the findings of this study have implications for research in other areas and this is the focus of the next section.

## SECTION III RESEARCH IMPLICATIONS

### Internal/External Validation of Subtypes

The research findings reported in Chapter IV are based on a large sample of serious offenders. Nevertheless, it does represent a single study and the representativeness of the sample has not been fully established. As such, replication of the present findings is highly desirable. According to Skinner's (1981) classification model such a study, which ideally would include a larger normal control sample, would assess the internal validity of this study's empirical typology, i.e., its reliability, coverage, homogeneity, and robustness.

Skinner (1981) also identified a need for external validation of typologies through studies addressing predictive, descriptive, and clinical validities. Suggestions for such studies are briefly discussed below.

An important predictive study would examine the relationship between neuropsychological types and risk for reoffending. The hypothesis is that neuropsychological status will contribute to the determination of risk analysis. In general, risk for reoffending is expected to be significantly related to deficits in executive function and verbal abilities.

Clinical validation would examine the clinical and behavioral correlates of the profiles. Examples include the relationship between profile type and psychiatric diagnosis, especially the components of Antisocial Personality Disorder (APD). Further, specific relationships between profile type and

specific clinical traits such as aggression, impulsivity, depression, capacity for attachment, hostility, emotional lability, and anger should be explored.

Relationships between neuropsychological status and treatment variables are of particular interest. Certain profiles provide logical implications for therapeutic and correctional management approaches. For instance, individuals with good executive and verbal abilities can be expected to require different intervention strategies from those with poor abilities. The typology can provide a rationale for treatment program development and evaluation. Individuals characterized especially by poor executive function may be particularly responsive to tricyclic antidepressants due to their mood stabilizing and attention/cognitive enhancement characteristics.

Work on identifying developmental trajectories (e.g., Loeber et al., 1993; Moffitt, 1993b) has suggested that neuropsychological deficits are associated with risk for later offending. Retrospective studies linking adult neuropsychological profiles with developmental trajectories could provide important information for early identification of high risk children, as well as further characterize the developmental antecedents of neuropsychological subtypes.

Lastly, some subgroups are characterized by exceptional levels of deficit. As such, their functional deficits and test performance is similar to patients with significant brain damage. The neuropsychological status of these subgroups appear to warrant more intensive examination with the goal of

further characterizing the dysfunctional brain mechanisms and devising appropriate treatment and management regimes.

Assessment of Prefrontal Dysfunction

Theories of the prefrontal cortical systems, reviewed in Chapter II, propose that they exercise an overall regulatory function including: 1) organizing behavior in terms of its consequences (executive function); 2) monitoring, integrating, and maintaining homeostasis among exteroceptive and interoceptive processes; and 3) controlling, elaborating, and modulating emotional influences. The prefrontal cortex is seen as "key to the highest human functions" (Stuss & Benson, 1986, p. 249). Accordingly, the prefrontal cortex is viewed to have a critical role in the development of personality.

Although there exist general agreement that the prefrontal cortex exercises a pervasive role of critical importance, the scientific analysis and instruments to assess its functions lag far behind theoretical understanding (cf. Costa, 1988). The principal measures of prefrontal function employed in this study, e.g., the Wisconsin Card Sorting Test and the Halstead Category Test, can, at best, only be considered an incomplete basis for evaluation of dysfunction (cf. Bigler, 1988). Despite this, large statistically significant differences between offenders and normal controls were observed on these measures. Clearly, more ecologically valid measures of executive function (cf. Heinrichs, 1990; Lezack, 1982; Shallice & Burgess, 1991) and more factorially pure measures of associated cognitive processes (Lezack, 1988)

may refine and elucidate the critical processes involved. In general, these objectives are already priorities within clinical neuropsychology and the recommendation here is simply that advances in this area be incorporated within the application of neuropsychology to offenders. Another, more recent area of experimentation, involves the prefrontal orbital cortex and the assessment of the functions of this structure may be critical to executive function and highly pertinent to the assessment of offenders.

Traditionally, damage to the orbitofrontal prefrontal cortex has been associated (see Chapter II, Section II, subsection Prefrontal Syndrome - Personality Effects) with dramatic negative changes in personality often characterized by major increases in impulsivity, gross losses of social propriety, and disruptions in emotional self-control. It is important to recognize that changes may occur and may markedly affect adaptive functioning while cognitive and intellectual abilities are preserved. When such personality changes are due to lesions specific to the orbitofrontal region, Damasio et al. (1990) have referred to these sequelae as acquired psychopathy or sociopathy. Damasio et al. (1990) have proposed that a defect in the activation of somatic markers that must accompany the internal and automatic processing of possible response options may be responsible. Tranel (1994) proposed that damage to "orbital cortices would disable the automatic guiding system and call for nonautomatic, cost-benefit analysis of response options and outcomes" (p. 303).

Dysfunction of this system would reduce the likelihood that inappropriate appetitive or aversive behavior would be consciously perceived or modified through subcortical processes.

Damasio and Anderson (1993) have suggested that patients with orbitofrontal damage have: a) an inability to organize future activity and hold gainful employment; b) a diminished response to punishment; c) are inclined to present an unrealistically favorable view of themselves; d) correct, but stereotyped manners; e) a tendency to display inappropriate emotional reactions and these characteristics may be present in individuals of normal intelligence.

Tranel (1994) has contrasted the notion of acquired sociopathy with developmental psychopathy and concluded that while the former is typically more benign, the theoretical account of dysfunction in neurological mechanisms implicated may be similar. Tranel (1994) proposed that a neuroscience approach may shed "important light on the neural mechanisms underlying the standard developmental form of antisocial personality disorder" (p. 305). These comments are of particular interest given that the orbital cortex has been proposed as the primary site of dysfunction in Attention Deficit Hyperactivity Disorder which has a high association with Conduct Disorder and results in a disproportionate number of cases in later adult offending (cf. Barkley, 1994; Evans et al., 1986).

The application of recently developed experimental methodologies to patients with orbitofrontal damage has been very successful in discriminating

their deficits from other neurological patients (e.g., Bechara et al., 1994; Damasio et al., 1990). The application of these paradigms to offenders seems to offer considerable promise in conjunction with standard neuropsychological assessments in identifying high risk offenders, but also, as Tranel (1994) has noted, further investigation in this area "might facilitate development of more effective treatments for this highly refractory condition" (p. 305).

Exploration of orbital dysfunction in offenders, it is suggested, should be among the first research priorities of those responsible for adult offenders.

## SECTION IV CONCLUSIONS

The overall objective of this research has been to evaluate the potential relevance of neuropsychology for offenders within a developmental/preventative perspective. Accordingly, the purpose of this section is to offer conclusions regarding the potential of neuropsychology to provide a scientific basis for contributing to:

1. The clinical understanding of offenders and their behavioral patterns.

2. The clinical assessment, the prediction of offender behavior, and the development of intervention strategies, as well as programming for offenders.

3. An understanding of the role of neuropsychological factors regarding negative developmental trajectories and for providing a rationale for early identification and remediation of high risk children.

Clinical Understanding of Offenders

The antisocial behavior patterns of persistent offenders are typically characterized as having arisen early in life, to have been enduring and resistant to change. Often the antisocial behavior seems aimed at nothing more than the immediate gratification of transient desires. It appears undeterred by any sense of shame or remorse, despite flagrant transgressions on the integrity and rights of others. Indeed, even a sense of self-preservation is frequently lacking. As such, the behavior is considered to be impulsive to the degree that an abnormal mental condition can be inferred (cf. Gorenstein, 1991).

Given this phenomenological presentation comprising impulsivity, defective empathy development, and defective socialization, neuropsychological theory suggests the possibility of compromised brain function. In particular, the prefrontal cortex is seen to be critical to the development of adaptive behavioral and emotional self-regulation, reflecting empathy and social sensitivity. Moreover, these processes are seen to rely heavily on verbal mediation and the elaboration of internal language. Executive function and dynamic verbal processing capacities are the key constructs proposed to be related to the role of the prefrontal cortex in determining whether behaviors will be a reflection of deliberate choices or merely impulsive actions.

The constructs of executive function and dynamic verbal processing have been represented in this study in terms of traditional

neuropsychological measures. Consistently and unequivocally, offenders represented in this study were found to exhibit a high level of impairment in terms of their executive function and dynamic verbal processing capacities, relative to the normal controls with whom they were compared.

The interpretation of this high level of impairment does not necessarily reflect brain damage, but it does indicate a high level of functional deficit and at a level typically associated with brain damage in neurological populations. The significance of this level of impairment among offenders is difficult to evaluate given that it likely reflects the outcome of developmental deficiencies rather than the onset of dysfunction associated with brain trauma or disease which is characteristic of brain damaged populations. The implications of this high degree of chronic functional deficit are of major social significance. When combined with deleterious environmental circumstances, such deficit could be understood to account for the major phenomenological aspects of persistent offenders, specifically their characteristic impulsivity and their social/empathic insensitivity.

Neuropsychological theory and the empirical findings of this research suggest that heuristic formulations of prefrontal cortical dysfunction may provide theoretically appealing constructs for understanding the behavior of persistent offenders. Further, this heuristic conceptualization of impulsivity provides a framework for the development of assessment and treatment modalities, perhaps of considerable practical value and significance.

Moreover, to the extent that the prefrontal cortex is critical to the understanding of persistent offenders, a neuropsychological approach carries the potential for benefiting from future advances in the neuroscience of the prefrontal cortex.

Clinical Assessment/Prediction/Intervention

Theories of the prefrontal cortex propose that it is intimately related to the development and expression of personality. As a corollary, significant prefrontal dysfunction can be expected to significantly disrupt normal personality development. In particular, social and personality development have been noted to be severely affected by early damage to the prefrontal cortex. An understanding of an individual's neuropsychological capacities is thus seen as critical to the clinical assessment of personality function. With respect to the assessment offenders, their capacities for emotional self-control and emotional self-regulation are often key issues. Overall, neuropsychological assessment can contribute a great deal to clinical understanding and provide a sound basis upon which to address issues of risk and its management, treatment modality, and behavioral management strategy. A key objective of this research has been to develop an empirical typology or classification system to effectively and efficiently characterize an individual's neuropsychological characteristics to enhance clinical judgement.

Neuropsychological test data analysed in this study indicated that five neuropsychological factors accounted for the majority of variance in test performance. Modal Profile Analysis of these factors and Wechsler

intelligence scales resulted in four bipolar neuropsychological modal profiles and four Wechsler profiles. Typically, an individual's overall neuropsychological capacities could be described by specifying the modal neuropsychological profile and the modal Wechsler profile to which he belonged in conjunction with consideration of his level of performance across each of these profiles. This classification process succinctly describes an individual's capacities across five neuropsychological factors and the Wechsler scales. This scheme thus provides the clinician with a comprehensive summary of neuropsychological functions relevant to a broad range of clinical functions, including the prediction of criminal recidivism.

Further research examining the external correlates, e.g., risk for reoffending, of individuals classified to particular profiles at given levels of performance may add significantly to the clinical meaning that can be attached to an individual's classification.

An individual's neuropsychological profile provides the clinician with important information for developing an effective intervention strategy. In general, these capacities are highly relevant to selecting the most effective intervention strategy, and in particular, the level of directedness required in therapy. In many instances, a neuropsychological profile can be related to specific problems and may assist in defining the therapeutic agenda, e.g., increasing verbal mediation processing with the goal of increasing not only individual and social awareness, but also empathic processing.

In other situations, specific neuropsychological deficits, e.g., in attention and concentration, can be addressed through remedial programming or medical treatments. A common goal of therapy is to increase a client's self-knowledge and self-understanding. A neuropsychological assessment provides an objective basis to assist in this regard. Further, a neuropsychological profile highlights both strengths and weaknesses. These strengths can often be used to compensate for weakness or reveal adaptive resources which might otherwise be overlooked, e.g., vocational potential.

Lastly, the empirical typology developed in this study provides a basis for defining treatment and programming needs for groups of offenders accordingly to their common patterns of neuropsychological performance. Such an approach should result in improved treatment assignments and also provide a rational basis for increasingly specific treatments. In sum, neuropsychological capacities critically interact with personality and adaptive functioning. Integration of neuropsychological assessment within a broader clinical framework can be expected to result in remarkably improved service delivery.

Developmental/Preventative Considerations

Views regarding the developmental role of the prefrontal cortex have undergone dramatic transformation over the past decade. Its involvement in critical early cognitive functions has been demonstrated in infants less than one year old. Studies of children who sustained damage to the prefrontal cortex at an early age have demonstrated that the impact on personality,

social, and cognitive development can be severe. Disordered behavior and problems with emotional self-regulation appear to be common sequelae. Phenomenologically, these children often resemble children with Attention Deficit Hyperactivity Disorder and Conduct Disorder.

Neuropsychological studies of children with Attention Deficit Hyperactivity Disorder or with Conduct Disorder, or with both diagnoses, have been restricted by a lack of suitable measurement instruments for the assessment of prefrontal functions in children. Nevertheless, these studies typically have concluded that deficits in prefrontal functioning and verbal abilities are common among these children. Neuropsychological theories of Attention Deficit Hyperactive Disorder and of Conduct Disorder hypothesize that dysfunction of the prefrontal cortex, especially the orbital region, is critically implicated in both disorders.

Outcome studies of children with Attention Deficit Hyperactivity Disorder and Conduct Disorder note that these disorders carry a disproportionate risk for poor adult adjustment, including antisocial behavior patterns. Some authors, e.g., Moffitt and Lynam (1994) have proposed that the degree of neuropsychological deficit in executive and verbal capacities may mediate this risk. This view is certainly consistent with the high level of impairment in these capacities among the offenders assessed in the present study. Indeed, it seems more than plausible that the executive dysfunction and behavioral disinhibition observed in adult offenders could be considered

heterotypic continuities of neuropsychological deficits in children with Attention Deficit Hyperactivity Disorder, Conduct Disorder, or both disorders.

Overall, children with both disorders, combined with significant levels of executive and verbal processing abilities, appear to represent a high risk group. If the child also presents with a history of aggression, family dysfunction, physical or sexual abuse, or social disadvantage, the risk for serious problems seems uncomfortably large. Such an ostensible risk clearly warrants a preventative approach and implies a need for aggressive early intervention programming. While the group characterized above appears to be defined by a confluence of negative factors, retrospective analyses of the offenders classified in various prototypical neuropsychological profiles may shed further understanding of risk factors and prospective studies are clearly needed to identify high risk groups. Both theoretical formulations and empirical studies of children imply that neuropsychological variables may be highly relevant to the identification of children at high risk for severe maladjustment.

Early Remedial Programming

The Standing Committee on Justice and the Solicitor General (Horner, 1993) concluded that "hiring more police and building more prisons" would not contribute to the safety and security of Canadians and proposed that the most reasonable response to the problem of crime should shift forward identifying "at risk" children and that remedial efforts should be directed towards them.

The evidence reviewed in this study indicates that children who are diagnosed with ADHD with aggressive features and other impulsive and learning disabled children are at a high risk for poor developmental outcome. On the other hand, there is considerable evidence that children with ADHD and other developmental learning problems need not have a deleterious social outcome if their disorders and other limitations are managed sensitively within an integrated framework which is responsive to their unique social, educational, and treatment needs. This likely can best be designed through the development of comprehensive assessment strategies, appropriate remedial programming, and the sensitization and education of the parents, teachers, and other caretakers involved in their care.

The field of neuropsychology, it is suggested, has a great deal to offer in this respect, and recent advances in understanding the role of neuropsychological factors in early development have been remarkable. In the future, these developments should make a major positive contribution to the overall understanding and early remediation of children with ADHD and other children with learning and impulse disorders.

To properly implement successful remedial programs, schools will need to modify their management of such children, and develop special programming for them. In doing so, they should be able to count on the support of integrated multidisciplinary teams providing assessment, technical development, and treatment for the children involved. This will require a great deal of flexibility on the part of both school and mental health

professionals, and no doubt reorganization and priorization of resources will be needed. Realistically, it will also require greater support from society, including additional financial resources. In this regard, it is encouraging that the Standing Committee on Justice and the Solicitor General has recommended diversion, on an increasing basis, of a proportionately small, but significant amount of the approximately 7 billion dollars which is spent on the Canadian Justice System annually.

The Standing Committee proposed that such monies should be directed toward the care and remedial treatment of high risk children. The present study concludes that neuropsychological factors have a primary role in identifying such children and that neuropsychological approaches also can provide a scientific basis for developing remedial programming.

The linkage between neuropsychological factors and risk seems to be sufficiently established to justify the development of pilot remedial programs at this time.

A Concluding Comment

In the Preamble, attention was drawn to Boll's (1985) statement that "All disorders will not prove to produce neuropsychological mischief. Some will" (p. 484). The results of the present study indicate unequivocally that serious antisocial behavior disorders create a great deal of "neuropsychological mischief". Also, it was noted in the Preamble that the Standing Committee on Justice and the Solicitor General (Horner, 1993) proposed that substantive solutions to the problems of crime lie in prevention

and that the focus should be on children "at risk" (p. 2). The present study suggests that neuropsychological function is intimately associated with risk and that neuropsychology has much to contribute to both the identification of high risk children and to the development of intervention strategies.

Moreover, neuropsychology appears to have considerable potential to contribute to the development of a scientific basis in support of clinical services delivery. Correctional organizations that incorporate a neuropsychology program can expect improved services, but will also be poised to benefit from the current explosion in neuroscience and all that portends for the understanding, assessment, and treatment of offenders.

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