NEUROPSYCHOLOGICAL DEFICITS
IN BORDERLINE PERSONALITY DISORDER

BY

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DECLARATION

I, Christine Anne Human,
hereby declare that this dissertation is my own work
and has not been presented for any degree at another university.

The work reported in this dissertation was performed
in Wards 4 and 5 of the Tara Hospital, Gauteng,
under the guidance and supervision of
Dr. Mara Profis.
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ABSTRACT

The relatively rapid development of biological approaches to various psychological conditions, has prompted clinicians and researchers to investigate Borderline Personality Disorder more thoroughly.

Research has evidenced the uniqueness of Borderline Personality Disorder in terms of description, aetiology and treatment. Of the various aetiologies proposed, the neuropsychological deficit approach is one which is still in its infancy and which may have promise for new treatment strategies. Latest developments delineate neuropsychological deficits in the areas of memory, perception and visuospatial ability. These factors are important for psychotherapeutic purposes.

The purpose of this study was to further existing knowledge as regards the aetiology of Borderline Personality Disorder in order to initiate new treatment modalities and management strategies. The study examined whether a battery of neuropsychological tests could detect organic dysfunction in the areas of construction, orientation and attention, memory, perception and concept formation and reasoning in twenty inpatients diagnosed according to DSM-IV criteria, with Borderline Personality Disorder. Two control groups were used, one comprising twenty inpatients diagnosed according to DSM-IV criteria with Personality Disorders from Axis II, Clusters A or C; and the other comprising twenty normal volunteers. Neuropsychological functioning assessed, included measures of attention, construction, visual and auditory-verbal memory, perception, and concept-formation and reasoning.

Measurement instruments used in this study included the Digit Symbol subtest of the WAIS-R; Rey Complex Figure; Logical Memory subtest of the WMS-R, Gottschaldt Embedded Figures Test; and the Wisconsin Card Sorting Test.
Analysis of variance, multivariate analysis of variance and post hoc tests revealed significant deficits in neuropsychological performance among the borderline personality disorder group and the control group of other personality disorders but not the normal volunteer group. Dysfunction was particularly significant in the areas of attention, visuospatial ability, perceptual organization, and ability to maintain cognitive set. These deficits do not appear to have been attributable to attention deficit disorder, attention deficit hyperactivity disorder, temporal lobe epilepsy, head injury, a concurrent Axis I diagnosis such as major depressive disorder, or current drug and/or alcohol abuse. The observed deficits suggest new ways of understanding the development and maintenance of Borderline Personality Disorder, and provide indications for treatment.

In conclusion, it is recommended that full use be made of the measurement instruments used in this study as diagnostic aids to enhance the effectiveness of treatment modalities. It is further recommended that research in this topic be repeated and extended using a larger sample and matched controls.
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CHAPTER ONE

INTRODUCTION

1.1 INTRODUCTION

Borderline Personality Disorder (BPD) was first recognized as a legitimate nosologic entity in 1980 by DSM-III (American Psychiatric Association, 1980). The understanding of BPD has advanced rapidly since then and has recently been accepted into the International Classification of Diseases (World Health Organization, ICD-10; 1992), where it is classified as a sub-category of "emotionally unstable personality disorder".

Borderline personality disorder has been described as being both a problem and a challenge for mental health professionals in the twentieth century. Herein, it is a disorder with problems in terms of its prognosis as well as its very poor response to treatment. In addition, it is the most lethal of psychiatric disorders with suicide rates of between 3% to 9.5% (Kjelsberg, Eikeseth & Dahl, 1991; McGlashan, 1986; Silver & Cardish, 1991; Stone, 1989 cited in Tasmar, Hales & Frances, 1989).

In addition to the high suicide rate, the morbidity rate for Borderline Personality Disorder is equally high. Herein, the Borderline Personality Disorder is not only prone to major depression but is also a condition that is associated with intense, unstable interpersonal relationships, self-damaging impulsivity, inappropriate or uncontrolled anger, affective instability and physically self-damaging acts (Andrulonis, Glueck & Stroebel, 1981). This would seem to indicate that not only does it have a high degree of suicide potential but it is also personally and socially as well as in a familial sense, a very damaging disorder. Despite repeated attempts at improving the treatment of Borderline Personality Disorder, there is still a very high dropout rate as two thirds of these patients discontinue treatment within the
first three months (Kelly, Soloff, Cornelius, George, Lis & Ulrich, 1992; Skodol, Buckley & Charles, 1983). Additionally, Goldberg, Schulz, Schulz, Resnick, Hamer and Friedel (1986), and Soloff, George, Nathan, Schulz, Ulrich and Perel (1986), have not found any psychotropic medication to be effective, even with the supplementation of mood stabilizers such as Lithium Carbonate and/or anticonvulsants such as Carbamazepine (Cowdry & Gardner, 1988). The attempts at developing a comprehensive and effective treatment for Borderline Personality Disorder by Linehan and Wasson (1990) have met with slightly more success, but not sufficient to warrant it as a successful treatment.

It would appear from the above that the problem herein is that it has been virtually impossible for science to describe either the psychobiology or the psychology of the Borderline Personality Disorder. Whereas it has been clearly defined as a nosological entity, it is unclear what the underlying psychobiological mechanisms are. There has been mention of severe childhood trauma (Browne & Finkelhor, 1986; Finkelhor, Hotaling & Lewis, 1990; Herman & van der Kolk, 1987; Herman, 1992; Russell, 1986); and deleterious childhood experiences (Links, Steiner & Huxley, 1988; Paris, Zweig-Frank & Guzder, 1994; Parker, 1983) interacting with possible neurological predispositions in the form of possible electrophysiological disturbances (Andrulonis et al., 1981; Martin, de Meo & Frances, 1989; van Reekum, 1993).

No large-scale study has ever been undertaken to determine the nature of the underlying aetiology of Borderline Personality Disorder. Given the poor response to treatment based on pure biological mechanisms as well as based on pure psychological mechanisms, it would appear necessary to embark on the analysis of the neuropsychological substrate of the Borderline Personality Disorder. However, before this can be undertaken, it would be important to first of all describe Personality Disorder and Borderline Personality Disorder in that context.
CHAPTER TWO

PERSONALITY DISORDERS

2.1 INTRODUCTION

Reber (1985, p.533) states that 'personality' is one of the classic “chapter heading” words in psychology: meaning that it is a term which is so resistant to definition and so broad in usage that no coherent simple statement about it can be made. Without becoming embroiled in a particular personality theory definition, the author has chosen Kaplan et al.'s definition of personality as “the totality of emotional and behavioural traits that characterize the person in day-to-day living under ordinary conditions, and it is relatively stable and predictable over time” (Kaplan, Sadock & Grebb, 1994, p.731).

As with personality, personality disorders resist precise definition. They cannot be regarded as categorical entities like schizophrenia or bipolar disorder where one either does or does not have the disorder. Rather, they are dimensional constructs which vary in severity and thus there are difficulties in defining the border between normality and abnormality (Parker and Boyce in Beumont & Hampshire, 1989). The DSM-IV maintains that the essential feature of a personality disorder “is an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual’s culture and is manifested in at least two of the following areas: cognition, affectivity, interpersonal functioning, or impulse control” (DSM-IV, American Psychiatric Association, 1994, p.630).

2.2 NATURE AND DEFINITION

The personality disorders may be defined as “chronic, pervasive, and inflexible patterns of perceiving and responding to the environment that are sufficiently maladaptive to cause disruption in functioning and environmentally generated
subjective distress” (Meyer & Deitch, 1996). The features of a personality disorder usually become recognizable during adolescence or early adult life. It is only when personality traits are inflexible and maladaptive and cause either significant functional impairment or subjective distress that they constitute a class of personality disorder. However, most individuals in need of a personality disorder diagnosis do not originally think there is any compelling reason for changing themselves and any such realization comes only when they move into situations that require higher levels of intimacy or more flexible behavioural adaptations (Turkat, 1990). The fact that they cannot meet these requirements results in coercion from the environment, or at least feedback that they cannot ignore, resulting in referral to therapy.

According to Kaplan et al. (1994) the symptoms of a personality disorder tend to be alloplastic in that they are capable of adapting and altering the external environment; ego-syntonic in that they are acceptable to the ego; and resistant to a feeling of anxiety about the displayed maladaptive behaviour. Therefore, persons who have personality disorders are more likely to refuse psychological help and to deny their problems than are persons with anxiety disorders, depressive disorders, or obsessive compulsive disorder.

The next section gives details of the three clusters of personality disorders according to the DSM-IV (APA, 1994) classification and elucidates some of the core behavioural manifestations of the different types of personality disorders.

2.3 CLASSIFICATION AND DIAGNOSTIC CRITERIA

The personality disorders have traditionally been grouped into three clusters which DSM-IV (APA, 1994) bases on descriptive statistics:

Cluster A includes the Paranoid, Schizoid and Schizotypal personality disorders, as these are denoted by peculiar or eccentric behaviour. The prominent clinical features of Paranoid personality disorder are lifelong suspiciousness, mistrust,
hypervigilance, hypersensitivity to praise and criticism, and a pervasive tendency to ascribe malicious intent to the actions of others and events. The defining clinical feature of Schizoid personality disorder is a pervasive indifference to social and family relations and a constricted range of affect whereas Schizotypal personality disorder is marked by pervasive deficits in interpersonal relatedness and peculiarities of ideation, appearance, and behaviour.

**Cluster B** focuses on dramatic, erratic and emotionally labile behaviour and includes the Histrionic, Narcissistic, Antisocial, and Borderline personality disorders. The core features of Histrionic personality disorder include self-dramatization, self-centeredness, seductiveness, excessive demands for approval, and shallow and labile expressions of affect. Narcissistic personality disorder is characterized by an exaggerated sense of self-importance, often combined with an underlying feeling of inferiority, and these persons tend to be demanding and exploitive. Antisocial personality disorder has as it core symptoms impulsivity, lack of remorse and empathy, and a sense of moral, vocational, and social irresponsibility which often leads to criminal behaviour. Borderline personality disorder (BPD), which is the subject of this study is discussed in detail in Chapter three.

**Cluster C**, which emphasizes chronic fearfulness and/or avoidance behaviours, includes the Avoidant, Dependent, and Obsessive-compulsive personality disorders. Avoidant personality disorder is composed of several key traits such as hypersensitivity to the possibility of rejection, humiliation, or shame, the result of which is a withdrawal from social contact. The core features of Dependent personality disorder include submissiveness, passivity, timidity, and clinging. Persons diagnosed with Dependent personality disorder tend to be excessively dependent on others, turning over to another person the responsibility for deciding the course of one's life. Fear of abandonment seems to underlie this passivity. Obsessive-compulsive personality disorder is defined by excessive preoccupation with trivial details at the cost of spontaneity and effectiveness. The traits of Obsessive-compulsive personality disorder reflect abnormalities in all three
aspects of mental life: behaviour, thought and mood. The behavioural aspect includes preoccupation with lists, rules, and schedules, etcetera, whereas the cognitive component usually takes the form of overconscientiousness, moral rigidity, or repetitive worries and self-reproaches that one cannot obliterate from one's mind.

There is also a catch-all category termed Personality Disorder NOS (301.90), used for individuals who do not fit any of the criteria for a specific category, yet clearly fall within the overall patterns of the personality disorders (DSM-IV, APA, 1994).

It should be noted that this clustering system, although useful, has serious limitations and has not been consistently validated. In addition, individuals frequently present with co-occurring personality disorders from different clusters (Pfohl in Dunner, 1993).

General diagnostic criteria for a personality disorder according to DSM-IV (APA, 1994) are listed in Table 2.3A.

2.4 AETIOLOGY

The idea of a personality disorder as being traits or trait clusters that are unified, long-standing, deeply ingrained and which handicap the individual in dealing with the world, is essentially a psychodynamic concept. However, various aetiological pathways embracing biological, psychological, and social factors are purported to be instrumental in the development of personality disorders. The personality disorder categories in DSM-IV (APA, 1994) derive from four separate theoretical frameworks: the dynamic, trait, biological, and sociological models. What follows is a discussion of research findings and pertinent theories.
### TABLE 2.3A.

**DSM-IV General diagnostic criteria for a Personality Disorder**

(A) An enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture. This pattern is manifested in two (or more) or the following areas:

1. cognition (i.e. ways of perceiving and interpreting self, other people, and events)
2. affectivity (i.e., the range, intensity, liability, and appropriateness of emotional response)
3. interpersonal functioning
4. impulse control.

(B) The enduring pattern is inflexible and pervasive across a broad range of personal and social situations.

(C) The enduring pattern leads to clinically significant distress or impairment in social, occupational, or other important areas of functioning.

(D) The pattern is stable and of long duration and its onset can be traced back at least to adolescence or early adulthood.

(E) The enduring pattern is not accounted for as a manifestation or consequence of another mental disorder.

(F) The enduring pattern is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., head trauma).

2.4.1 Biological factors

The biological model ascribes personality to genetic or biological predispositions, and there is growing evidence of the heritability of personality traits in healthy, separately reared monozygotic twins. Current models are based on basic biological disposition anchored in neurotransmitter activities.

Evidence that genetic factors may have a bearing on the aetiology of personality disorders was demonstrated in the United States in an investigation of psychiatric disorders in 15,000 pairs of twins. Among monozygotic twins the concurrence of personality disorders was several times higher than among dizygotic twins. More recently, in a study on multiple measures of personality and temperament, occupational and leisure-time interest and social attitudes, it was found that monozygotic twins reared apart are about as similar as those reared together (Kaplan et al., 1994).

Genetic studies conducted on personality disorders by clusters, demonstrate differing correlational factors. For example, Cluster A (Paranoid, Schizoid, and Schizotypal) is more common in the biological relatives of schizophrenics than among control groups. Among the Cluster B group of personality disorders (Antisocial, Borderline, Histrionic, and Narcissistic), a genetic predisposition associated with alcoholism has been demonstrated in antisocial personality disorder; a strong association was found between histrionic personality disorder and somatization disorder (Briquet's syndrome); and patients with borderline personality disorder have more relatives with mood disorders than control groups. The Cluster C group of personality disorders (Avoidant, Dependent, and Obsessive-compulsive) may also have a genetic base. Obsessive-compulsive traits are more common in monozygotic twins; and patients with Obsessive-compulsive personality disorder have shown some signs associated with depression (Kaplan et al., 1994).

Research conducted on specific personality disorders within the clusters has yielded differing findings. With regard to Cluster A personality disorders, the
aetiology of Paranoid personality disorder is unclear. Familial and adoption studies of the biological relatives of schizophrenics have suggested that genetic factors may play an aetiological role in this disorder, and it has been shown that the risk for Paranoid personality disorder was highest in relatives of patients with Paranoid Delusional Disorder (Kendler, Masterson & Davis, 1985).

In Schizoid personality disorder there has been an association made between social isolation and eye movement dysfunction (EMD). A neurointegrative deficit reflected in EMD is thought to hamper the development of the relationship between parent and child, creating a dysynchrony in attachment with subsequent psychological sequelae (Siever & Davis, 1991).

Schizotypal personality disorder also appears to have psychobiological markers. Baron, Asnis and Gruen (1983) found an increased prevalence of Schizotypal relatives in the biological relatives of schizophrenics as compared with relatives of controls and that these Schizotypal relatives had decreased activity of plasma amine oxidise. In addition, Siever (1985) found that eye movement dysfunction (EMD) was associated with Schizotypal personality disorder in a group of college students identified to be at high risk for the development of schizophrenia. Evidence that there are strong psychophysiological similarities between Schizotypal personality disorder and schizophrenia is accumulating. In a recently published study, eye tracking accuracy in patients with Schizotypal personality disorder was compared with controls with non-schizophrenia-related personality disorders and with normal controls. Both schizotypal and schizophrenic subjects demonstrated significantly more impaired tracking than did the other personality disordered control group and the normal control group (Siever, Keefe, Bernstein & Coccaro, 1990).

As regards cluster B personality disorders, the degree of empirical support available is either scant or differs substantially. Although psychological factors have been researched in Histrionic and Narcissistic personality disorders respectively, there appears to be no information on the neurobiology of these
disorders. On the other hand, Antisocial personality disorder (ASPD) has been extensively researched in this area. Twin and adoption studies suggest a strong genetic component for both ASPD and criminal behaviour. Concordance for antisocial behaviour among monozygotic and dizygotic twins is as high as 51 percent and 22 percent, respectively. Offspring of adult criminals have rates of ASPD ranging from 6 to 36 percent (Rutter, MacDonald & Couteur, 1990). In addition, electroencephalographic (EEG) abnormalities, especially in the frontal and temporal lobes, have been correlated with antisocial and criminal behaviour (Kandal & Freed, 1989). Biological and psychophysiological studies have focused on the sensation-seeking, impulsive, and criminal behaviours exhibited by ASPD patients. Diminished functioning of both the serotonergic and nonadrenergic systems has been associated with impulsivity and violence (Marin, de Meo & Francis, 1989). The aetiology of Borderline personality disorder is discussed extensively in the next chapter (Chapter 3).

The inhibited personality types in the Cluster C group tend to have been the ones that Freud and his pioneers first encountered and treated with psychoanalysis. Aetiology was thought to lie in the domain of inhibition in the assertion of socially acceptable impulses, and little or no attention was given to possible biological factors. To date there does not seem to be any research conducted in this sphere on Dependent personality disorder, However, there is some data suggesting a measure of heritability in Obsessive-compulsive personality disorder but the evidence is not strong and is partly indirect. The concordance rate for compulsive personality in monozygotic twins is greater than that in dizygotic twins, yet in a family study of Obsessive-compulsive personality disorder, despite the higher incidence of mental illness in the close relatives of the patients (as compared with those of the controls), those illnesses were more apt to be either depressive or other types of Cluster C disorders than Obsessive-compulsive personality disorder itself. OCPD may thus get its impetus from a genetic factor, but is given final shape by rearing patterns that overemphasize conformity, neatness, and punitiveness (McKeon & Murray, 1987).
2.4.2 Psychological Factors

The dynamic model is based on internal organizing psychology where results from conflict-resolving early life experience are highlighted and developmental factors are emphasized. Data is inferred from patients undergoing intensive psychoanalytic therapies. As mentioned before, most DSM-IV categories originate from the dynamic model, and therefore it is appropriate to discuss the historical underpinnings and philosophy of this approach.

When psychology gained its independence from philosophy and became a science in the second half of the nineteenth century, its goal was to use a laboratory-based introspection to discover the basic elements of mental life in the human adult. This approach led by Wilhelm Wundt was known as the structural approach and emphasized the analysis of conscious processes into their fundamental elements and the laws that govern these elements. Sigmund Freud, one of the chief proponents of the psychoanalytical model, digressed from Wundt with his radical approach to the study of human beings. Rather than treating consciousness as the centre of mental life, Freud “likened the mind to an iceberg, only a small segment of which protruded above the surface of the water” (Hjelle & Ziegler, 1981, p.29).

He theorised that individuals are in a perpetual state of conflict motivated by a comprehensive realm of mental functioning - unconscious sexual and aggressive urges. His account of psychosexual development is based on the premise that sexuality begins at birth and progresses through a biologically defined set of erogenous zones until adulthood is reached. He believed that personality developed by proceeding through five psychosexual stages: oral, anal, phallic, and genital. Personality traits were thought to be related to a fixation at one of the psychosexual stages of development. For example, passive and dependent personality traits were conceived as being due to fixation at the oral stage when dependence on others for the intake of food is prominent. Subsequently, Wilhelm Reich coined the term “character armour” to describe characteristic defence mechanisms which individuals use to protect themselves from internal impulses
and interpersonal anxiety. Currently, on Axis II of the DSM-IV, each personality disorder has a cluster of defences that help a psychodynamic clinician recognize the type of character pathology present. For example, individuals with Schizoid personality disorder tend to use withdrawal as a defence whereas Borderline personality disorder is associated with splitting (Kaplan et al., 1994).

From a psychodynamic point of view, another central feature of personality disorders is a person's internal object relations. Melanie Klein evolved a theory which is linked to drives and postulates that the ego undergoes a splitting process to deal with the fear of annihilation. She viewed projection and introjection as the primary defence mechanisms operative in the first months of life. The infant projects its fear of annihilation into the mother and then fears attack from the "bad mother". The resulting anxiety causes the infant to organize experience ambivalently by splitting love and aggression into good and bad objects. As these disparate views are integrated, the infant fears that it may have harmed or destroyed its mother through its hostile and sadistic fantasies directed toward her (Goldstein, 1989).

Similarly, Winnicott conceptualised a "true self" which develops in response to the "holding environment" provided by a "good-enough mother". If the infant experiences a traumatic disruption of his developing sense of self, a false self emerges providing a protected exterior behind which the true self hides and maintains its integrity by monitoring and adapting to the conscious and unconscious needs of the mother. Winnicott also developed the concept of the transitional object (for example, a dummy, a blanket, or a teddy bear) which serves as a substitute for the mother during the infant's attempt to separate and become independent (Berzirganian, Cohen & Brook, 1993; Winnicott, 1991).

Although the abovementioned psychodynamic theories differ in their aetiological focus, they share one common assumption, that is, that personality disorders or "character disorders" are rooted in early childhood experiences.
The more serious personality disorders (for example, Antisocial personality disorder and Borderline personality disorder) are thought to stem from disturbances at certain early stages of psychosexual development originating in the early pre-oedipal one-to-one relationship with the infant's mother or other primary caretaker. A disturbed relationship interferes with the main developmental tasks at this stage: psychological separation from the mother figure in conjunction with a comfortable sense of relatedness to her and a growing ability to perceive oneself and others as unique, complete individuals. Failure to achieve this development leads to a flawed sense of self and difficulty in relating to others as an adult (Bootzin & Acocella, 1988).

Less severe personality disorders (for example, Obsessive-compulsive personality disorder) are grounded in disturbed parent-child relations in the later oedipal phase. Strong ambivalent reactions to mother and father at this stage of childhood can lead to a rigid and distorted view of the self - one based on needs and fantasies rather than reality (Bootzin & Acocella, 1988; Kaplan et al., 1994).

Temperamental factors identified in childhood may be associated with personality disorder in adulthood (for example, a temperamentally fearful child may develop Avoidant personality disorder.) “Goodness of fit” (that is, the match between the temperament of parent and child and child-rearing practices) if poor, may also give rise to the development of a personality disorder (Kaplan et al., 1994; Millon, 1981).

What defines the personality disorders from a dynamic viewpoint is a pattern of distorted or weakened ego functions which affect such capacities as perception, memory, language, learning, and motor behaviour. Each personality disorder shows a pattern of maladaptive behaviour in one or more of these areas. However, the origin of personality disorders is the subject of divergent viewpoints (Bootzin & Acocella, 1988).
2.4.3 **Social Factors**

The sociological model purports that personality is shaped by social circumstances, environmental factors, family and community influences, or the larger social fabric comprising social and political factors.

The behavioural perspective is relevant here, in that advocates of this perspective generally object to the concept of personality disorders because it implies the existence of stable, fixed personality traits. They view personality disorders as specific maladaptive responses that occur in response to specific stimuli and as a result of specific reinforcing consequences. To identify the sources of this behaviour, they look to the family. They regard the family as a learning laboratory. Two learning mechanisms which proponents of the behavioural perspective view as crucial in the creation of antisocial and aggressive behaviour are modelling and reinforcement. Research has shown that modelling can both teach aggressive behaviour and trigger specific aggressive acts (Bootzin & Acocella, 1988). With regard to the mechanism of reinforcement, Snyder (1977) found in his studies of children who seldom engage in antisocial behaviour that they have parents who consistently reinforce prosocial behaviour (for example, helpfulness, co-operation, affection) and ignore or punish antisocial behaviour.

On the other hand, advocates of the sociocultural perspective blame social injustices for most antisocial behaviour (including Antisocial personality disorder) and argue for changing the society, not individuals. For example, societies in which material luxuries are highly valued and widely displayed but in which only certain groups have access to such luxuries can engender a state of "anomie". The theory of anomie or normlessness among certain disadvantaged groups may be applicable to Antisocial personality disorder where criminal behaviour is the result of the injustices built into our society. Sociocultural theorists advocate that psychological processes are merely the products of large-scale social processes - processes that ensure the prosperity of certain social groups and the deprivation of others (McGarvey, Gabrielli, Beutler & Mednick, 1981).
Environmental influences are also thought to influence the course of personality development. Children taught to fear and avoid many kinds of persons and situations which most people would consider harmless, may for example, develop Avoidant personality disorder. Negative family environments where parental brutalization, incest and sexual molestation take place, may also be the breeding ground for the development of a personality disorder.

Social factors embrace the whole fabric of society and may converge and interact to produce a particular type of personality disorder in a predisposed individual.

2.5 SUMMARY

Personality disorders may be regarded as dimensional constructs which vary in severity and resist precise definition. It is only when personality traits are inflexible and maladaptive, causing significant functional impairment or subjective distress that they constitute a particular class of personality disorder. These traits are deeply ingrained, inflexible, and maladaptive. The foregoing discussion follows the DSM-IV classification of personality disorders into three broad clusters of maladaptive traits: Cluster A - odd or eccentric; Cluster B - dramatic, emotional or erratic; and Cluster C - anxious or fearful.

The nature, definition, classification, diagnostic criteria, and aetiology of personality disorders has been addressed in this chapter. The aetiology is approached from a multidimensional perspective as it seems that many factors interact and overlap in the pathogenesis of a particular personality disorder. Biological, psychological and social factors were delineated as being contributory to the development of a particular personality disorder.
2.6 CONCLUSION

Personality Disorders are probably the most common pattern of psychiatric disorder, and yet these disorders have seldom been given the empirical research which they deserve. Most of the research to date is based on clinical observation and is steeped in psychoanalytic nomenclature. However, despite the divergent views and theories on personality disorders and the fact that biological, psychological and social factors are not constant, one factor is constant - they all affect cognition. It is this factor which has inspired the researcher of this study to investigate neuropsychological functioning in Borderline Personality Disorder.

Chapter One described the problem and challenge which Borderline Personality Disorder represents, particularly from an aetiological point of view and thus also from a treatment perspective. Chapter Two presents an overview of personality disorders. The nature, definition, classification, diagnostic criteria and aetiology of personality disorders is discussed in this chapter before moving on to Chapter Three where Borderline Personality Disorder (BPD) in particular, is discussed in detail.
CHAPTER THREE

BORDERLINE PERSONALITY DISORDER

3.1 INTRODUCTION

Despite the voluminous literature on Borderline Personality Disorder, little of what has been written is firmly grounded in scientific research, and the numerous theories that abound are based on unsystematic clinical observations. As Aronson (1989, p.524) notes, "From a methodological perspective, almost the entire treatment literature overgeneralises from small patient samples, uses inconsistent diagnostic criteria, and relies heavily on anecdotal, uncontrolled, unsystematic, personal experience."

The purpose of this chapter is to give a fully comprehensive outline of the history, aetiology and current conceptualisation of BPD. As will be seen, BPD is a complex construct and therefore its aetiology is likely to be multifactorial. Although the author is specifically investigating neuropsychological deficits as an aetiological pathway in BPD, it is nevertheless acknowledged that unidimensional investigation is reductionistic, and may only be a partial explanation of the aetiology of BPD.

3.2 NATURE AND DEFINITION

"I come to explore the wreck,
The words are purposes.
The words are maps."

From Diving into the wreck: Poems (1991-72)
Adrienne Rich

Since the first clinical descriptions of the borderline patient almost sixty years ago (Stone, 1980), the borderline concept has traversed tumultuous waves of
vagueness, mystique, diversity, confusion, and uncertainty. Indeed, this lack of clarity has left theorists, researchers and clinicians in doubt as to what a "borderline" really is (Akiskal, Chen, Davis, Puzantian, Kasgarian & Bolinger, 1985; Gunderson, 1984; Johnson, 1988; Millon, 1992; Stone, 1986). As Hartocollis (1977) states: "The borderline case is less of a frontier than a no-man's-land, an entire field whose borders are vague."

Thus, Borderline Personality Disorder has been clumsily labelled due to an historical "hangover" steeped in immense confusion and evoking much curiosity. Survey of the literature on the borderline construct reveals both a plethora of terms and considerable disagreement amongst theorists concerning prime attributes of the syndrome (Millon in Clarkin, Marziali and Munroe-Blum, 1992, p.3). Borderline Personality Disorder has probably been around for centuries albeit cloaked in different apparel. With this in mind, what follows is a chronicle of the development of the concept "borderline" since its first inception in the early 1900's.

### 3.2.1 Historical Development of BPD

The concept of "borderline" has a long and complicated history, dating as far back as 1930 when Obendorf suggested that there was no hard and fast line between neuroses and psychoses (Egan, 1986).

In 1938 Adolf Stern introduced the term "Borderline" when he outlined the characteristics of a group of office patients "too ill for classical psychoanalysis" (Stone, 1980, p.6) and developed his theory that there is a realm of psychopathology that lies on the border between neurosis and psychosis (Paris, 1994). His clinical description included some characteristics which are recognisable in the current diagnostic criteria of BPD in the DSM-IV (APA, 1994): narcissism, "psychic bleeding", inordinate hypersensitivity, psychic rigidity, negative therapeutic reactions, feelings of inferiority, masochism, "wound

In 1941 Zilboorg introduced the term “Ambulatory Schizophrenia” for a milder group of individuals whose social facade was fairly well preserved and for whom hospitalisation was not necessary. Symptoms commonly displayed amongst this group included outward normality, dereistic thinking (thinking “away” from things), shallow human relationships (acquaintances but no friends), and incapacity to settle down to one job or life pursuit (Stone, 1980).

A year later in 1942, Helene Deutsch coined the term “as if” to mean poverty of object relations with the tendency to adopt the qualities of the other person as a means of retaining love. While maintaining a grasp on reality, this group displayed symptoms of depersonalisation which was not ego-alien: narcissism, masking of aggressive tendencies by passivity; and inner emptiness. Deutsch studied five patients, four of whom had a family history of psychosis, and this led her to conclude that the “as if” personality might represent the early phase of the schizophrenic process “before it built up to the delusional form” (Stone, 1980, p.13). Deutsch’s contribution lies in her emphasis on the pathology of object relations. This thought trend was pursued by Fairburn in 1944 and by Melanie Klein in 1952 who both reached similar conclusions shortly afterwards (Stone, 1980).

Between 1945-1946 Rapaport, Gill and Schafer used psychological tests (including the Rorschach) to differentiate types of schizophrenics. From a sample of seventy-five patients, they classified thirty-three of these as “preschizophrenic”. In 1947 Melitta Schmideberg favoured the term “borderline” to mean “in between neurosis and psychosis. These patients she found to be true to their type over long periods of time, and referred to them as “stable in their instability” (Stone, 1980, p.16). At about the same time Paul Federn used the term first coined by Bleuler “Latent Schizophrenia” to describe this group of patients who were “neither here or there” - that is, on the border between neurosis and psychosis.
In 1949 Hoch and Polatin followed with their term “Pseudoneurotic Schizophrenia” characterised by a symptomatic pattern of “panneurosis”, “pananxiety” and “pansexuality” (Gabbard, 1990). In the early 1950’s a number of psychoanalytic writers used the term “borderline schizophrenia” (Stone, 1980), and in 1953 Gustav Bychowski expanded on the term “latent psychosis”, giving the term more precise borders.

After Stern, Robert Knight (1954) was the next to recommend the term “borderline” and define it in a methodical fashion as being a band of the psychopathological spectrum between neurosis and psychosis, where diagnosis is “often difficult, and equally often obscured behind a show of dramatic symptoms.” He adopted a more egopsychological approach than some of his predecessors and emphasized severe weakening of many ego functions such as secondary process thinking, realistic planning, and defences against primitive impulses (Gabbard, 1990). Although Knight did not use the term “borderline schizophrenia”, his borderline cases are nearer the schizophrenic end of the psychotic spectrum than are later theorists such as Grinker and Kernberg (Stone, 1980).

By the mid-fifties John Frosch introduced the term “psychotic character” and made further contributions to psychoanalytic theory with his work on disorders of impulse control. Following a psychodynamic model, Easser and Lesser (1965) drew attention to a group of patients who were hysterical in their outward manifestations yet somehow more deeply disturbed than their classically psychoneurotic counterparts. The researchers reclassified these patients, some of whom had psychotic features and were elsewhere called borderline, as “hysteroid” (Stone, 1980).

Up to this point most authors were referring to seriously ill patients who were frequently institutionalized but who did not meet the usually accepted criteria nor
show signs of serious deterioration of the more severe disease entities like the schizophrenic or affective psychoses (Stone, 1980).

In the early 1960's Grinker, Werble and Drye attempted to objectify the diagnosis of borderline in some methodical fashion with their statistical analysis of sixty borderline patients hospitalised in Chicago. A cluster analysis of data on these patients suggested that there were four subgroups of borderline patients occupying a continuum from the *psychotic border* (type I) all the way to the *neurotic border* (type IV). In between the two extremes a group with predominantly negative affects and difficulty maintaining stable interpersonal relationships (type II) and another group (type III), were characterised by a generalised lack of identity, needing to borrow identity from others. Their criteria for the Borderline Syndrome were anger, defect in affectional (interpersonal) relations, absence of consistent self-identity, and depression. Grinker's four subtypes of borderline patients included: (1) The Psychotic Border; (2) The Core Borderline Syndrome; (3) The Adaptive, Affectless, Defended, "As if" Persons; and (4) The Border with the Neuroses (Stone, 1980, p.29). Grinker, Werble & Drye (1968) also attempted to identify common denominators in the borderline syndrome that were present regardless of the subtype. They identified four key features: (1) anger as the main or only affect; (2) defects in interpersonal relationships; (3) absence of consistent self-identity, and (4) pervasive depression. This empirical study made a significant contribution in finding that the borderline syndrome was clearly distinct from schizophrenia - that is, these patients did not deteriorate into frank schizophrenia over time - rather they were stable in their instability throughout the course of their illness. Grinker et al's (1968) concept of "borderline" starts to approach the way we understand BPD today.

Until the 1960's there were no formal diagnostic criteria for BPD - however, there were this group of patients characterised by a recognizable syndrome of impulsivity, affective instability, and unstable relationships. A breakthrough occurred in the mid-seventies when Gunderson and Singer developed a truly operational definition of BPD in the form of a semi-structured interview to
systematise the approach to diagnosis of Borderline personality disorders. Research conducted using this instrument suggested that a definition of BPD could be based on the criteria of intense, unstable interpersonal relationships, manipulative suicide attempts, an unstable sense of self, negative affects, egodystonic psychotic experiences, impulsivity, and low achievement (Gunderson, 1984). This work continued and strengthened the validity of BPD as a clinical entity, and by 1987 Gunderson and Zanarini were able to rank order discriminating features based on research focussed on descriptive characteristics: (1) intense and unstable interpersonal relationships; (2) chronic self-destructive behaviour; (3) chronic abandonment fears; (4) chronic dysphoric affects; (5) cognitive distortions; (6) impulsivity; and (7) poor social adaptation (Gunderson & Zanarini, 1987).

While Gunderson (1984) and Grinker et al. (1968) focussed primarily on descriptive diagnostic criteria, Otto Kernberg (1967, 1975) attempted to characterize borderline patients from a psychoanalytic perspective. Using a combined ego psychological object relations approach, he coined the term "borderline personality organization" to encompass a group of patients who showed characteristic patterns of ego weakness, primitive defence operations, and problematic object relations. He cautioned however, that descriptive symptoms were not sufficient for a definitive diagnosis. Instead he believed that diagnosis rested on a sophisticated structural analysis that revealed four key features: (1) non-specific manifestations of ego weakness; (2) shift toward primary process thinking; (3) specific defensive operations; and (4) pathological internalised object relations.

By 1980 the construct was strong enough to earn inclusion in DSM-III (APA, 1980 in Paris, 1990). Prior to DSM-III, borderline patients were usually diagnosed with the DSM-II category of schizophrenia, latent type (APA, 1968 in Paris, 1990). In developing diagnostic criteria for BPD, an effort was made both to include the descriptive features identified by Gunderson and to reflect the
structural analysis of Kernberg. Recently, the definition has been refined and updated in the DSM-IV (APA, 1994).

We are now approximately a century down the track and although the previously termed “wastebasket” category has now been refined and has a new label - *Borderline Personality Disorder* - it is hardly in its finality as regards aetiology. What has happened is that BPD has moved away from sitting under the umbrella of schizophrenia, and is now a separate clinical entity.

### 3.2.2. Current status of PBD

Currently DSM-IV diagnostic criteria for Borderline Personality Disorder are the most widely used in the field. The utility and credibility of DSM-IV require that it focus on its clinical, research, and educational purposes and be supported by an extensive empirical foundation. DSM-IV is used by clinicians and researchers of many different orientations (for example, biological, psychodynamic, cognitive, behavioural, interpersonal, family systems), and by psychiatrists, other physicians, psychologists, social workers, nurses, occupational and rehabilitation therapists, counsellors, and other health and mental health professionals. Furthermore, it is a multiaxial system involving assessment on several axes, each of which refers to a different domain of information that may help the clinician plan treatment and predict outcome. There are five axes included in the DSM-IV multiaxial classification (see Table 3.2.2A).

The use of the multiaxial system aids the clinician in making a comprehensive and systematic evaluation of the patient with regard to mental disorder, general medical condition, psychosocial and environmental problems, and level of functioning. In addition, this multiaxial system provides a standardized format for organizing and communicating clinical information, for capturing the complexity of clinical situations, and for describing the heterogeneity of individuals presenting with the same diagnosis.
TABLE 3.2.2.A

DSM-IV Multiaxial Classification

<table>
<thead>
<tr>
<th>Axis</th>
<th>Description</th>
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<tbody>
<tr>
<td>Axis 1</td>
<td>Clinical Disorders</td>
</tr>
<tr>
<td></td>
<td>Other Conditions that may be a focus of clinical attention</td>
</tr>
<tr>
<td>Axis 11</td>
<td>Personality Disorders</td>
</tr>
<tr>
<td></td>
<td>Mental Retardation</td>
</tr>
<tr>
<td>Axis 111</td>
<td>General Medical Conditions</td>
</tr>
<tr>
<td>Axis IV</td>
<td>Psychosocial and Environmental Problems</td>
</tr>
<tr>
<td>Axis V</td>
<td>Global Assessment of Functioning</td>
</tr>
</tbody>
</table>

(APA, 1994)

3.3 DEFINITION AND DIAGNOSTIC CRITERIA

Borderline Personality Disorder falls in the dramatic cluster (Cluster B) of personality disorders and is classified on Axis 11 of DSM-IV (APA, 1994). The distinguishing feature of BPD is an instability of personality which affects all aspects of living. It is further characterised by a pervasive pattern of stormy interpersonal relationships, unstable affect, and behavioural dyscontrol (Dulit, Marin & Frances, cited in Dunner, 1993).

Persons in the BPD category tend to be impulsive and unpredictable in their behaviour, leading quiet lives one week and then gambling, driving at high speeds, overdosing on drugs or alcohol, or running up huge bills at another time. Their emotions tend to be erratic and marked by abrupt shifts, culminating in outbreaks...
of anger, bouts of anxiety and spells of depression, emptiness and boredom. Their relationships are also subject to frequent highs and lows. Severe identity disturbance may accompany their instability manifesting with problems in self-image, gender identity, career choice, and long-term goals (Dunner, 1993; Stone, 1990).

The DSM-IV classification bases its diagnosis on a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

**TABLE 3.3A**

**Diagnostic criteria for 301.83 Borderline Personality Disorder**

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<table>
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</tr>
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<tbody>
<tr>
<td>1.</td>
<td>Frantic efforts to avoid real or imagined abandonment. Note: do not include suicidal or self-mutilating behaviour covered in Criterion 5.</td>
</tr>
<tr>
<td>2.</td>
<td>A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation.</td>
</tr>
<tr>
<td>3.</td>
<td>Identity disturbance: markedly and persistently unstable self-image or sense of self.</td>
</tr>
<tr>
<td>4.</td>
<td>Impulsivity in at least two areas that are potentially self-damaging (e.g. spending, sex, substance abuse, reckless driving, binge eating. Note: Do not include suicidal or self-mutilating behaviour covered in Criterion 5.</td>
</tr>
<tr>
<td>5.</td>
<td>Recurrent suicidal behaviour, gestures, or threats, or self-mutilating behaviour.</td>
</tr>
<tr>
<td>6.</td>
<td>Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).</td>
</tr>
<tr>
<td>7.</td>
<td>Chronic feelings of emptiness.</td>
</tr>
<tr>
<td>8.</td>
<td>Inappropriate, intense anger or difficulty controlling anger (e.g. frequent displays of temper, constant anger, recurrent physical fights.</td>
</tr>
<tr>
<td>9.</td>
<td>Transient, stress-related paranoid ideation or severe dissociative symptoms.</td>
</tr>
</tbody>
</table>

(APA, 1994, p.654)
3.4 DIAGNOSTIC FEATURES

Gunderson and Zanarini (1987, p.6) liken the problem of the initial selection criteria for Borderline Personality Disorder to “packing a suitcase and then being surprised to find what is in it when it is opened”. However, despite conflicting views among and within analytic, biological, and social learning schools of thought concerning the aetiology, dynamics and treatment of BPD, there are key borderline features that contemporary clinicians judge as salient and valid regarding the clinical description of the “typical” borderline patient (Clarkin, Marzioli & Munroe-Blum, 1992).

These patients have complex clinical presentations comprising a kaleidoscope of emotions including diverse combinations of anger, sarcasm, anxiety, intense and labile affect, brief disturbances in consciousness (for example, depersonalization, dissociation), chronic loneliness, boredom, a chronic sense of emptiness, unstable and volatile interpersonal relationships, identity confusion, impulse behaviour (including self-injury - cutting and self-mutilation, recurrent suicide attempts, death by suicide in 8%+), and a hypersensitivity to abandonment (Tomb, 1995).

It is pertinent at this stage to discuss the clinical features of BPD in detail in order that the reader may get a ‘feel’ for the complex presentation of this disorder. Individuals with BPD classically display some or all of the following features: abandonment fears; unstable interpersonal relationships; identity disturbance; impulsivity; suicidal gestures; affective instability; feelings of emptiness; inappropriate, intense anger; transient paranoid ideation or dissociative symptoms.
Abandonment fears: Individuals with BPD tend to have an exaggerated fear of impending separation and rejection which may be related to an intolerance of being alone and a perception that they are ‘bad’. This can have a serious effect on the self-image, affect, cognition, and behaviour, and can lead to impulsive actions such as self-mutilating or suicidal behaviours (Paris, 1994).

Unstable interpersonal relationships: Pervasive instability and ambivalence intrude constantly into the everyday lives of individuals with BPD, resulting in fluctuating attitudes, erratic or uncontrolled emotions, and a general capriciousness and undependability. Because these individuals are impulsive, unpredictable and often explosive, it is difficult for others to be comfortable in their presence and relatives and friends often feel ‘on edge’ in anticipation of this volatility and contrariness. In addition, they tend to distort their present relationships by putting every person into either an all-good or an all-bad category, seeing people as either nurturant and attachment figures or hateful and sadistic. As a result of the use of this primitive defence mechanism, called splitting, the good person is idealized and the bad person is devalued (Kaplan et al., 1994).

Identity disturbance: An immature, nebulous, or wavering sense of self-identity is marked by a precarious sense of being and a lack of inner harmony which is expressed in segmented, fragmented, contradictory attitudes and enigmatic actions. This diminished sense of wholeness is reinforced by erratic and conflicting impulses generating new experiences, and may be due to defective psychic structures where there is a failure to develop internal cohesion and hierarchical priorities.

Impulsivity: The tendency to act on one’s emotions (impulsivity) is a defining feature of BPD. These individuals tend to become labile and cope inappropriately with feelings of dysphoria by abusing substances, binge-eating, driving recklessly, engaging in promiscuous and/or unsafe sexual activity, gambling irresponsibly, or
participating in chaotic interpersonal relationships as a means of damping down the dysphoria. Both the dysphoria and the impulsive acting-out tend to reinforce each other, and a negative feedback loop develops (Paris, 1994). These constant upsets in their equilibrium are subject to emotional eruptions and uncontrollable behaviour and thoughts. Once these feelings are discharged, they regain a modicum of psychic balance until their tensions mount to uncontrollable proportions (Clarkin et al., 1992).

**Suicidal gestures:** Schmideberg (in Preston, O'Neal & Talaga, 1994), described the course of BPD as “stably unstable” and this is demonstrated in the repetitive self-destructive acts, for example, wrist-cutting and burning, which are common in the BPD group of individuals, and are usually precipitated by threats of separation or rejection. A form of expressing anger, a numbing of overwhelming affect, relief in the form of reaffirming the ability to ‘feel’, or by making amends for the individual’s sense of being evil, is often the justification given for these self-mutilative acts. On the other hand, self-mutilation may occur during dissociative experiences (DSM-IV, APA, 1994). Although these parasuicidal gestures are sometimes manipulative in nature, the reality is that there is a high rate of completed suicide in BPD (Paris, 1994). Kjelsberg et al. (1991) report a suicide rate of 8%, whereas other recent studies report a rate as high as 10% (Aarkrog, 1993; Silver & Cardish, 1991 in Kjelsberg et al., 1991; DSM-IV, APA, 1994).

**Affective instability:** This is displayed in marked shifts in mood and exhibited by periods of dejection and apathy interspersed with spells of anger, anxiety, or excitement. Moods and threats are the vehicles used to ‘get back at’ or ‘teach a lesson to’ those who have failed to be emotionally nurturant. Anguish and despair are covert means of expressing the hostility and frustration whereas self-destructive acts are more overt expressions of their perceived dejection (Clarkin et al., 1992).
Feelings of emptiness: Chronic feelings of boredom and inner emptiness pervade the lives of these individuals who constantly seek ways to fill this 'void'. Coupled with a very low tolerance for anxiety, frustration, and being alone, plus a lack of suitable sublimatory channels, they may resort to impulsive and inappropriate behaviour, such as accepting a stranger as a friend or engaging in promiscuous behaviour (Bootzin & Acocella, 1988).

Inappropriate, intense anger: Sarcasm, irritability, and bitterness often culminate in verbal or physical outbursts in response to feelings of perceived neglect or abandonment by a relative or lover. These feelings of real or imagined abandonment exacerbate the shame and guilt experienced, and a cycle of dysphoria, dejection and inner emptiness is propagated (Kaplan et al., 1994).

Transient paranoid ideation or dissociative symptoms: When under great stress, or in reaction to drugs or alcohol, BPD individuals may experience brief, transient psychotic symptoms (for example, hallucinations, body-image distortions, ideas of reference, and hypnagogic phenomena). Although reality testing is usually intact, brief lapses in reality testing occur under particular forms of stress (Grinker et al., 1968; Johnson, 1988; Kaplan et al., 1994). Although cognitively capricious, as exhibited in rapidly changing and often antithetical thoughts about themselves and others, the BPD individual usually shows ordinary reasoning abilities on structured tests, such as the Wechsler Adult Intelligence Scale and deviant processes only on unstructured projective tests, such as the Rorschach test (Kaplan et al., 1994). Primitive defence mechanisms such as denial, depersonalisation, derealisation, projection, splitting, devaluation and idealisation are commonly used, and these strategies highlight the difficulty that BPD individuals have with reality perception (Masterson, 1978).

Preston et al. (1994) summarise the core symptoms of BPD as:

* Generalised ego impairment
* Chronic emotional instability
* Chaotic interpersonal relations
* Feelings of emptiness
Most theorists agree that BPD is clearly a chronic illness with formidable morbidity and mortality. Although improvement over the long term is the most common outcome, it is by no means certain. In this vein, the current research proposes to investigate BPD from a neuropsychological testing perspective to find out whether there are deficits in cognition which may shed some light on the clinical presentation of these subjects. This may pave the way for addressing the distortions in information processing which form a part of the clinical picture in BPD.

3.5 DIFFERENTIAL DIAGNOSIS

As mentioned previously, the term 'borderline' originated from its overlap with schizophrenia (in old terminology, the overlap between neurosis and psychosis). Subsequently, BPD has been seen to overlap with disorders of impulse control, affective disorders, anxiety disorders, and certain other personality disorders such as Schizotypal, Paranoid, Histrionic and Antisocial (Kaplan et al., 1994; Silk, 1994).

Affective disorder is particularly common in BPD, with rates ranging from 24 to 74% for Major Depression, 4 to 20% for Bipolar Disorder, and 3 to 14% for Dysthymia (Widiger & Rogers, 1989). BPD has been reported to occur in 25% of bulimics (Levin & Hyler, 1986), and up to 67% of BPD patients may meet criteria for at least one substance use disorder diagnosis (Dulit, Fyer & Haas, 1990).

It is imperative that these disorders are excluded when making a diagnosis of BPD. In some instances the differences (particularly amongst the other personality disorders and BPD) are subtle, and in others, two or more personality disorders can co-exist and both or all can be diagnosed.
In general, assessment by a multidisciplinary team, a comprehensive account of medical, psychiatric, personal and family history, 'collateral' from relatives, and psychological tests, form the basic outline for forming a differential diagnosis of BPD. It is the researcher's explicit viewpoint that a basic battery of neuropsychological tests should also form part of the initial assessment for diagnosis of BPD.

3.6 DEMOGRAPHIC FEATURES

According to Gunderson and Zanarini (1987), BPD is by far the most commonly used Axis I diagnosis, with a prevalence somewhere between fifteen and twenty percent in the clinical population. Although prevalence rates vary substantially across studies because of differences in diagnostic criteria and setting, it appears that BPD is one of the more common psychiatric diagnoses with estimates in the general population of between two and four percent (Baron et al., 1985; Gunderson & Zanarini, 1987). In addition, BPD diagnosis is the most prevalent of the personality disorder diagnoses in both inpatient and outpatient settings (Widiger & Rogers, 1989).

There also appears to be an increased prevalence of major depression, alcoholism, and psychoactive substance abuse in first-degree relatives of persons with BPD (Kaplan et al., 1994).

The legitimacy of the diagnostic category of BPD has been substantiated by long-term follow-up studies demonstrating that, over time, borderline patients continue to manifest consistent clinical symptomatology; and in the vast majority of cases, they do not shift into other major psychiatric disorders (Gunderson & Zanarini, 1987).

The behaviour pattern in BPD transcends cultural barriers, and has been identified in many settings around the world. In addition, BPD is more commonly diagnosed
in adolescents and young adults of which females make up about seventy-five percent of the BPD population, and it is estimated to be five times more common among first-degree biological relatives of those with the disorder than in the general population (DSM-IV, APA, 1994).

The epidemiology of BPD is unknown but patients with this type of disorder are being diagnosed more commonly. It is not clear whether this is due to a change in diagnostic patterns or whether the frequency of the disorder is increasing as a consequence of social changes (Beumont & Hampshire, 1989). As at least two-thirds of patients who are diagnosed with BPD are female (Gunderson, 1984), this finding may be due to cultural biases stemming from sex-role stereotypes, because male patients who have features of BPD are often diagnosed as having Narcissistic or Antisocial personality disorder (Gabbard, 1990).

The prevalence of BPD is currently estimated to be:

* about 2% of the general population (DSM-IV, APA, 1994; Baron et al., 1985; Gunderson & Zanarini, 1987);
* about 10% among individuals seen in outpatient mental health clinics (DSM-IV, APA, 1994; Gunderson, 1984);
* about 20% among psychiatric inpatients (Widiger & Francis, 1989); and
* about 30-60% among clinical populations with personality disorders (Dulit et al., in Dunner, 1993; DSM-IV, APA, 1994; Widiger & Francis, 1989).

With a prevalence rate for BPD of somewhat between fifteen and twenty percent in the clinical population, and a rate of between two and four percent in the general population, it would appear that BPD is on the increase, and as such is clearly an urgent and critical challenge to mental health professionals.
3.7 AETIOLOGY OF BPD

The aetiology of BPD is essentially unknown although it is likely that multiple pathways exist, and that this complex disorder is shaped by biological vulnerability, brought on by psychological experiences, and influenced by social conditions (Paris, 1994). This discussion examines BPD and its empirical links in a multidimensional framework of biological, psychological and social risk factors. It is acknowledged that there is individual variation as to which risk factor is predominant, and that each risk factor is a necessary but not sufficient condition for development of BPD.

A Table depicting the major orientations to BPD (adapted from Treatment of the Borderline Personality by P.M. Chatham, 1985; [see Table 3.5A]) will precede a discussion of risk factors and most important theoretical frameworks pertaining to the aetiology of BPD.

3.7.1 Risk factors

It is acknowledged that risk factors (biological, psychological and social) are not necessarily proven facts in the aetiology of BPD. However, as the aetiology of BPD is clouded in a multiplicity of unique and over-riding variables which make it very difficult to separate the various aetiological factors, risk factors will be discussed as markers and empirical links in the development of BPD.

Biological risk factors include genetic transmission and changes in neurotransmitter activity; psychological risk factors include trauma in the form of physical and/or sexual abuse, separation and loss, parental psychopathology, and lack of parental bonding; whereas social risk factors include the social structure and environmental influences.
3.7.1.1 Biological risk factors

Evidence of the role of biological factors in the aetiology of BPD has been shown to be indirect but suggestive. Biological vulnerability seems to be linked to underlying personality traits rather than disorders, although no specific biological risk factors that could specifically predispose an individual to BPD have as yet been identified. (Personality traits are clusters of characteristic behaviours which are influenced by genes, show variance between populations, and are shaped by natural selection) (Paris, 1994). A number of studies using different methodological approaches, none of which are conclusive, have been undertaken to investigate biological vulnerability in BPD:

To investigate whether there is a genetic pattern of inheritance in BPD, methods employed involve family pedigree studies and twin studies. In a family pedigree study, Zanarini (1993 in Paris, 1993) found that relatives of BPD patients are more likely to have other impulse disorders such as Antisocial personality disorder (ASPD) or substance abuse; and that there is a greater frequency of affective disorders in the relatives of BPD patients with comorbid depression. This study is supported by Silverman, Pinkham and Horvath’s (1991) study where they examined personality traits in the relatives of BPD probands and found these traits to be characterized by either impulsivity or affective instability, the core dimensions of BPD. In Torgensen’s 1984 twin-study, there was no evidence of monozygotic-dizygotic differences, and thus far, genetic studies of BPD do not show that BPD is heritable.

Neurophysiological changes in the form of changes in neurotransmitter activity have been observed in BPD. For example, Coccaro, Siever and Owen (1990) compared personality disorders and affective disorders by examining serotonin activity as measured by the fenfluramine challenge test. Their findings evidenced a significant flattening in response in BPD patients, reflecting a sluggishness in serotonin activity, giving way to the possibility of trait impulsivity. On neuropsychological testing, Andrulonis et al. (1981) found “soft” neurological
### TABLE 3.5A

#### MAJOR ORIENTATIONS TO BPD

<table>
<thead>
<tr>
<th>Dimensions</th>
<th>Psychoanalytic</th>
<th>Biological</th>
<th>Eclectic</th>
<th>Biosocial</th>
<th>Cognitive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major theorists</td>
<td>Adler, Kernberg, Masterson, Meissner, Rinsley</td>
<td>Akiskal, Adrulonis, Cowdry, Gardner, Hoch, Kasuin, D. Klein, Kety, Polatin, Soloff, Stone, Wender</td>
<td>Frances; Grinker; Gunderson; Spitzer's DSM-III, DSM-III-R, DSM-IV</td>
<td>Linchan, Millon, Turner</td>
<td>Beck, Pretzer, Young</td>
</tr>
<tr>
<td>2. <strong>What is meant by “borderline”</strong></td>
<td>Psychostructural level or psychodynamic conflict</td>
<td>Mild variant of one of the major disorders</td>
<td>A specific personality disorder</td>
<td>A specific personality disorder</td>
<td>A specific personality disorder</td>
</tr>
<tr>
<td>3. <strong>Data on which diagnosis is based</strong></td>
<td>Symptoms, inferred intrapsychic structures, transference</td>
<td>Clinical symptoms, familial-genetic history treatment response, and biological markers</td>
<td>Combination of symptoms and behavioral observations, psychodynamics and psychological test data (WAIS, Rorschach)</td>
<td>Behavioral observation, structured interviews, behaviorally anchored test data</td>
<td>Behavioral observation, structured interviews, behaviorally anchored test data</td>
</tr>
<tr>
<td>4. <strong>Etiology of disorder</strong></td>
<td>Nurture, nature, fate&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Nature&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Unspecified</td>
<td>Nature, nurture</td>
<td>Nurture</td>
</tr>
<tr>
<td>5. <strong>Composition of borderline</strong></td>
<td>Homogeneous: Intrapsychic Structure</td>
<td>Heterogeneous: total sample</td>
<td>Heterogeneous</td>
<td>Heterogeneous: Unspecified</td>
<td></td>
</tr>
<tr>
<td>6. <strong>Importance of diagnostic subtyping</strong></td>
<td>Not important, except Meissner</td>
<td>Important</td>
<td>Somewhat important</td>
<td>Important</td>
<td>Unspecified</td>
</tr>
<tr>
<td>7. <strong>Basis on which subtyping made</strong></td>
<td>Etiology</td>
<td>Grinker and Gunderson: clinical; DSM: clinical and etiological</td>
<td>Behavioral patterns</td>
<td>Unspecified</td>
<td></td>
</tr>
<tr>
<td>8. <strong>Recommended Treatment</strong></td>
<td>Modified psychoanalysis, confrontive Psychotherapy</td>
<td>Chemotherapy</td>
<td>Unspecified</td>
<td>Modified behavior/ cognitive-behavior therapy</td>
<td>Modified cognitive therapy</td>
</tr>
</tbody>
</table>

*Note. Adapted from *Treatment of the Borderline Personality* by P.M. Chatham, 1985, New York: Jason Aronson.

<sup>a</sup> Cognitive components can play a role, as can fate; most theorists except Kernberg consider nurture a major cause.

<sup>b</sup> Stone (1981) believes that 10-15% of all cases of BPD in adults are purely psychogenic in origin.
signs in BPD patients, whereas O'Leary and colleagues (1991) found that some of the clinical features in BPD are associated with problems in the recall of complex learned material (O'Leary, Brouwers, Gardner & Cowdry, 1991). Stone (1993) suggests that abnormalities in limbic activity and/or cortical modulation of the limbic system may account for the behavioural abnormalities of BPD patients.

3.7.1.2 Psychological risk factors

There is some evidence that early childhood experiences such as trauma, early separation or loss, and abnormal parenting play a role in the development of BPD. However, there are some methodological problems in the research conducted thus far: retrospective data (for example, asking patients to recall their childhood experiences) have been measured and this poses questions of validity. As BPD patients tend to have distorted perceptions of their relationships in adulthood, it is likely that their childhood memories could also be distorted (Paris, 1994).

A number of the long-term effects of childhood sexual abuse resemble borderline pathology. Symptoms such as depression, suicide attempts, substance abuse, revictimisation, and problems with intimate relationships predominate in BPD and have also been observed in some individuals with histories of childhood sexual abuse. Efforts have been made to determine the mechanisms whereby abuse leads to psychopathology. Parameters currently used in research include frequency of abuse, duration of the abusive relationship, the relationship of the child to the perpetrator, the age of the child at the onset of the abuse, the age of the offender, the type of sexual act, whether force was used, whether there was disclosure, and what the parental reaction was to that disclosure. To date, all research conducted on childhood sexual abuse has been retrospective. Nine empirical studies (Briere and Zaidi, 1989; Byrne, Velamoor & Cernovsky, 1990; Herman, Perry & Van der Kolk, 1989; Links & Van Reekum, 1988; Ludolph, Westen & Misle, 1990; Ogata, Silk & Goodrich, 1990; Shearer, Peters & Quaytman, 1990; Westen, Ludolph & Misle, 1990; Zanarini, Gunderson, Marino, Schwartz & Frankenburger, 1989) have shown that childhood sexual abuse is frequent in BPD - about 70% in most.
reports. More recently, Paris, Zweig-Frank and Guzder (1994) examined multivariately the psychological risk factors in abuse. This study is the first of its kind to examine the relationship between childhood sexual abuse and BPD by measuring all the parameters. Results indicate that childhood sexual abuse is common in BPD, and that the more severe the childhood sexual abuse, the higher the risk of BPD.

Frequency, duration and severity are the parameters which predispose children to later psychopathology when exposed to physical and verbal abuse, and family violence. Browne and Anderson (1991), show that physical abuse is more common in males than females, and that there is a correlation between physical abuse and substance abuse and/or suicidality, two features of BPD.

There is evidence that borderline patients have a high frequency of early separation and loss from their parents during childhood (Paris et al., 1988; Soloff & Millward, 1983; Zanarini et al., 1989). However, it is in the interaction with other risk factors that separation and loss can contribute to BPD.

In addition, there are a number of lines of evidence which suggest that the parents of BPD patients have significant psychopathology. Links, Steiner and Huxley (1988), conducted a systematic study on the parents of BPD patients and found that the disorders with the greatest morbid risk in first degree relatives were recurrent unipolar depression (27%), alcoholism (21%), BPD (15%), and ASPD (10%). It is noteworthy that all of the parental disorders suggest underlying deviations in impulsive and affective personality traits, two core features of BPD.

Finally, terms such as “invalidating environment” (Linehan, 1993), “affectionless control” (Parker, 1983), and “oscillations in attachment” (Melges and Swartz, 1989), have been used to describe a combination of maternal overprotection and inconsistency as being predictive of BPD. Studies conducted on parental neglect and abnormal parental bonding as a risk factor for BPD (Frank & Paris, 1981; Soloff and Millward, 1983) have shown that in females, maternal affection was significantly lower in the BPD group, whereas in males there were higher levels of
control from their fathers. Abnormal parental bonding appears to be a risk factor for the development of BPD.

3.7.1.3 Social risk factors

The role of social risk factors in borderline pathology is difficult to research empirically. The general method for examining social risk factors in the major psychiatric disorders is epidemiological research, the rationale being that if the prevalence of a mental disorder is known, then it can be determined if there are variations under different social conditions. According to Paris (1994), there are three mechanisms which could link social factors with the development of BPD: Firstly, social factors may influence personality dimensions underlying BPD (for example, culture shaping the structure of personality by positively reinforcing some traits and negatively reinforcing others). Secondly, social factors may influence family structure and function; and thirdly, social factors may interfere with the development of social roles.

To date, there is no direct epidemiological evidence concerning changes in the prevalence of BPD over time. However, indirect evidence has linked social factors with symptoms associated with BPD. The increase in parasuicides among women, in suicide among young men, in substance abuse, and in ASPD are findings which show that the symptoms of BPD are becoming more prevalent, and this is suggestive of an increase in the prevalence of BPD. Paris (1994) hypothesizes that social integration is a protective factor against BPD and that social disintegration is a risk factor for BPD. In this connection, it has been found that impulsivity increases when social containment for deviant behaviour decreases. Societies can either proscribe characteristically “borderline” behaviours such as self-multilation, recurrent parasuicide, and substance abuse, or create an unstructured and permissive environment in which these behaviours are more likely to occur. Millon (1987) suggests that anomie that characterizes the rapid social change in contemporary society is having a particular effect on youth and is increasing the
risk for borderline psychopathology by interfering with intergenerational transmission of values and reduced influence of the extended and social community.

3.7.2 Theoretical Viewpoints

With a few exceptions, empirical studies into the aetiology of BPD have only been conducted since its recognition as a valid diagnostic entity in DSM-III in 1980 (Frank & Paris, 1981; Paris, 1994). Prior to this, the most persuasive postulates regarding the aetiology of the borderline syndrome have come from applications of psychodynamic models of personality development and reconstructions from therapy (Clarkin et al., 1992). In the last decade, clinical investigators have challenged the psychodynamic theories by proposing neurobehavioural (Taylor & Zaparniuk, 1991), family-dynamic (Millon, 1985; Soloff & Millward, 1983) and cognitive-behavioural (Linehan, 1987; Turner, 1987) theories to explain the aetiology of BPD.

3.7.2.1 Psychodynamic perspective

It is under this heading that most of the literature on the borderline can be found, although the reports are not well documented by empirical reference. In psychoanalysis, inferences are made from observations of the patient, reported symptoms, and interview material which includes recollections of early life experiences with caregivers (Gabbard, 1990; Clarkin et al., 1992). Although various psychoanalytic theorists are at odds as to the specific factors contributing to the development of BPD, most attribute them to developmental failures in the first two years of life (Kernberg, 1975; Masterson and Rinsley, 1975; Gunderson, 1984).
Contemporary psychodynamic theorists follow one of three broad psychoanalytic frameworks:

1. Ego psychology, derived from the psychoanalytic theory of Freud;
2. Object relations theory, derived from the work of Melanie Klein and other theorists such as Fairburn, Winnicott, and Balint; and
3. Self-psychology derived from Sullivan’s interpersonal theory, and formulated and elaborated on by Heinz Kohut (Gabbard, 1990).

The object-relations theorists point to the early mother-child relationship in shaping the personality structure. Much of the early work is based on Mahler and her colleagues’ (1975) writings on the “separation-individuation” process, which if successfully resolved results in the child’s attainment of “a sense of self and of separateness distinct from the mother” (cited in Dawson & MacMillan, 1993, p.12). The rapprochement subphase of the separation-individuation occurs between 16 and 25 months, when the infant begins to realise that he is separate from the mother. The conflict, which now faces the child, is a need for independence plus a need for symbiosis with mother. If the child receives “good enough mothering”, the rapprochement crisis is resolved. However, if not resolved, “fixation” can result in a psychic structure (ego or superego) that will later emanate into borderline symptomatology (Winnicott, 1955 in Dawson & MacMillan, 1993).

Kernberg’s concept of borderline personality organisation is structural in approach. He focuses on certain constitutional phenomena combined with deficiencies in the environment which contribute to failure of the infant to develop an integrated self-concept, and the formation of early developmental conflicts that fail to be adequately resolved. He also suggests that this developmental failure results in a split between good and bad self and object representations. He viewed borderline patients as having successfully traversed Mahler’s symbiotic phase so that self and object can be clearly distinguished, but also as having become fixated during the separation-individuation phase. Kernberg targeted the rapprochement
subphase between approximately 16 and 30 months, as the chronological site of this developmental crisis. At this stage, the child becomes alarmed about the potential for its mother to disappear, and at times displays a frantic concern as to her whereabouts. From this developmental standpoint, BPD patients repeatedly relive an early infantile crisis in which they fear that separation from their mother will result in her disappearance and abandonment of them. In the adult form of this crisis, individuals are unable to tolerate periods of being alone and fear abandonment from significant others. Kernberg also implicates drives into his aetiological theory and maintains that a failure on the part of the child to integrate good and bad self-other object representations is due either to an excessive aggressive drive plus inability to neutralise aggression and/or lack of anxiety tolerance. Primitive defences such as denial, projection, and splitting are used to keep the conflicted perceptions of the self and other separate. Kernberg does not blame the mother for the pathological outcome of the borderline’s identity formation, but rather focuses on the progressively integrative aspects of ego development. He presumes that the borderline has acquired the cognitive capacity but not the emotional capacity for object constancy (Clarkin et al., 1992; Gabbard, 1990).

Masterson and Rinsley (1975) also use Mahler, Pine and Bergman’s (1965) rapprochement subphase of separation-individuation to locate the conflict which they believe stems from the mother’s withdrawal of libidinal supplies when the child attempts to separate from her in search of his/her own identity. They further describe the mother of the borderline as having a pathological need to cling to her child in order to perpetuate the gratification she experienced when her infant’s survival was symbiotically bound to her. Masterson and Rinsley (1975), found that the mothers of BPD patients (whom they viewed as typically borderline themselves), were highly conflicted about their children growing up. As a result, the child receives a message from the mother that growing up and becoming one’s own person will result in the loss of maternal love and support. A key corollary of this message is that remaining dependent constitutes the only available means of maintaining the maternal bond.
Adler (1985) and Gunderson (1984) based their understanding of the aetiology of BPD on a deficit or “insufficiency” model which was influenced by the self-psychological theories of Kohut. They believe that the borderline personality results from a failure to develop sufficient resources for “holding-soothing” and thus is unable to develop a stable identity in relation to a perception of an independent other. This could be due to the absence of “good enough” mothering during the separation-individuation phases. Because the mother is emotionally unavailable, the child fails to achieve “evocative memory” (Piaget’s sixth stage of cognitive development, age 18 months). Arrested at this crucial stage of development the borderline is unable to restore an integrated memory of the object and regresses to the earlier stage of “recognition memory” (age 8 months). This creates feelings of emptiness, depressive tendencies, and clinging dependency (Dawson & MacMillan, 1993; Gabbard, 1990).

Although most psychodynamic hypotheses about borderline pathology draw on Mahler’s observational, longitudinal studies of mothers and their children, Mahler (1971 in Dawson & MacMillan, 1993) cautioned against drawing inferences about adult psychopathology from observations of childhood developmental phenomena. There may be some link between ego fixation and developmental conflicts during the rapprochement subphase of separation-individuation but this hypothesis is not specific to BPD. Similarly, Kernberg’s constitutional view of the aetiology of borderline personality organisation is not specific to BPD but applies also to Schizotypal, Narcissistic, Histrionic, and Antisocial personality disorders (Dawson & MacMillan, 1993).

In summary, Kernberg (1975), Masterson & Rinsley (1975), and Adler (1985) all postulate some variation of a developmental disaster around the separation-individuation phase of childhood. Essentially, the psychopathology of the mother does not allow healthy separation and individuation to occur, nor does it allow the development of a self-soothing interject, with the result that the libidinal and aggressive components of self and object (mental) representations are not
integrated and negative emotions cannot be tolerated. This leads to a fragile sense of identity and the primitive ego defences (splitting and projective identification) into adult life (Paris, 1993).

Although Kernberg (1975), and Masterson and Rinsley (1975), have presented the most complete psychodynamic developmental formulations for the aetiology of BPD, no evidence has been adduced to support such complex constructs or to bolster the underlying metapsychological assumptions. The problem lies in the absence of empirical validation (Gabbard, 1990), and the lack of evidence between clinical observation and explanatory hypotheses (Paris, 1993).

3.7.2.2 Neurobehavioural Viewpoint

The neurobehavioural model suggests a connection between the negative developmental effects of childhood brain dysfunction and the development of borderline symptomatology. The neurologically impaired child characteristically displays symptoms such as hyperactivity, short attention span, distractability, mood oscillation, and high impulsivity resulting in a behavioural syndrome which is similar to that of BPD including problematic social interaction, academic difficulties, and low levels of achievement (Clarkin et al., 1992).

Various research studies using electroencephalograms (EEG's), computed tomography scans (CT scans), P300 auditory event-related potentials, and soft-sign neurological examinations have tentatively identified a large subgroup of patients diagnosed with BPD who have histories of developmental or acquired brain insults. These studies have demonstrated a possible biological correlation between the severity of BPD and the number of previous brain insults. The possibility of frontal system cognitive dysfunction in BPD has been raised (Andrulonis et al., 1981; Cowdry, Picker & Davis, 1985; Kutcher & Blackwood, 1987; Gardner, Lucas & Cowdry, 1987; Lucas, Gardner, Cowdry & Pickar, 1989; Schulz, Koller, Kishore, Hamer, Gehl & Friedel, 1983; Snyder, Pitts & Quinton,
Additionally, strong evidence is beginning to emerge from studies employing neuropsychological testing. Most studies have consistently found that subjects with BPD, as a group, show evidence of cognitive impairment, and most commonly these deficits seem to reflect frontal system impairment (Burgess, 1990; Judd & Ruff, 1993; O'Leary, Brouwers, Gardner & Cowdry, 1991; Van Reekum, 1993).

A review of the literature on neurological investigations using current technology follows:

There are two reports of an increased prevalence of non-specific EEG abnormalities in BPD. Snyder & Pitts (1984) compared BPD subjects with normal controls and found an increased incidence of slow-wave activity in BPD subjects, whereas Cowdry et al. (1985) found bilateral posterior sharp activity in BPD subjects when compared with unipolar depressed subjects. These non-lateralized EEG abnormalities suggest a subtle dysfunction in the central nervous system (CNS) rather than an abnormal cortical focus.

Three studies to date have sought structural brain abnormalities by CT scan (Lucas et al., 1989; Schulz et al., 1983; Snyder et al., 1983). Snyder et al. (1983) found no evidence of anatomical changes whereas Schulz et al. (1983) found slightly larger ventricular-brain ratios in BPD subjects. Although Lucas et al. (1989) found no evidence of frontal lobe atrophy nor abnormal ventricle-brain ratios in BPD subjects, they did find a narrower third ventricle in BPD which could be accounted for by a narrower third ventricle observed in female subjects overall.
Kutcher & Blackwood (1987) used P300 and other long-latency auditory event-related EEG potentials to demonstrate that BPD subjects share a “dysfunction of auditory neurointegration” with schizophrenic patients.

Gardner et al. (1987) used a battery of soft neurological signs to compare 17 BPD females with 22 normal controls. They found that the BPD group had significantly more soft signs than the normal controls.

Van Reekum and colleagues’ (1990) case-controlled, retrospective, chart review study examined the prevalence and significance of brain insults in BPD. Their findings indicated that a large subgroup of BPD subjects (54%) had at least one insult and that the cognitive functioning of this cohort with BPD was very similar to that of a cohort with traumatic brain injury (TBI). In addition, they demonstrated that a biological gradient exists between the severity of BPD and both neurodevelopmental or acquired brain insult history and neurocognitive functioning.

Hartocollis (1977) postulates an association between the distorting effects of minimal brain dysfunction (MBD) and the child’s perceptions of his/her own behaviours and interactions with caregivers. The suggested outcome is confused cognition, affect regulation and impulse control which ultimately leads to borderline ego development and behaviour.

Andrulonis and Vogel (1984) examined neurological factors specific to the development of BPD. They identified four subcategories of BPD, two of which included organicity factors: “attentional deficit/learning-disabled”, and “organic”. The results showed that 40% of the males, compared with only 14% of the females, suffered from an attentional deficit and/or learning disabilities. Also, 52% of the males, compared with 28% of the females, had either a current or past history of organic insults (e.g. head trauma, encephalitis, or epilepsy). Andrulonis and Vogel (1984) concluded that borderlines with MBD are predominantly male
and have an earlier onset of emotional and functional difficulties, based in part on a constitutional deficit.

Soloff and Millward (1983) tested several aetiological hypotheses in a cohort of BPD patients. Included was a test of a neurobehavioural model of borderline personality style, the results of which showed that there were more complications of pregnancy reported in the prebirth histories of BPD patients than in the other two groups for major depressive disorder and schizophrenia. In addition, the BPD group had more childhood psychopathology, including temper tantrums, rocking, and head banging whereas the schizophrenic group had a greater prevalence of learning difficulties.

Van Reekum (1993) conducted chart reviews on 48 borderline and 50 nonborderlines. The results showed that the borderline group had a statistically greater prevalence of developmental and acquired brain insults. The neurological markers included developmental delay, epilepsy, traumatic brain injury, and other central nervous system illnesses. Marziali (in Clarkin et al., 1992) suggests that there is a parallel between symptoms associated with frontal system dysfunction and borderline symptoms (impulsivity, cognitive inflexibility, poor self-monitoring, and perseveration).

In patients with developmental disturbances, other family members, notably parents, may also suffer from developmental disturbances. Thus, the children of these parents are more frequently exposed to aberrant behaviours, such as substance abuse, erratic parenting, marital discord and physical and/or sexual abuse. Exposure to these behaviours may directly affect the behavioural development of the child, resulting in a BPD profile of symptomatology (Gardner et al., 1987).

On the other hand, impulse dyscontrol - a feature of BPD - may be the result of a genetic predisposition to development disturbance. This may lead to a higher risk of developing TBI and substance abuse, both of which exacerbate the existing
impulse control disorder and contribute to cognitive limitations. Gardner et al. (1987) hypothesize that impulsively formed cognitions (including a lack of self-monitoring and modulation) result in impulsive behaviour leading to repeated personal failures and depressed or angry affects.

The above review indicates that a subtle form of neurological dysfunction may exist in a subgroup of the BPD population as a result of developmental or acquired brain insults. This underscores the need for clinicians and researchers to routinely examine the neuropsychological functioning of patients with BPD in order to establish whether underlying brain dysfunction is a feature of BPD.

3.7.2.3 Family Dynamics Viewpoint

Family dynamics theory points toward abuse and parental psychopathology, mediated biologically and environmentally, as likely contributors to the aetiology of BPD. The family environment and/or family history of psychopathology are purported to play a role. Family experiences of children who later develop BPD have been described and researched from various standpoints, such as, early separation and loss of a primary caretaker (Soloff & Millward, 1983; Akiskal et al., 1985; and Links, Steiner, Offord & Eppel, 1988); sexual and physical abuse in early childhood (Zanarini et al., 1989; Ogata et al., 1990; Stone, 1990); biparental failure (Frank & Paris, 1981; Soloff & Millward, 1983; Akiskal et al., 1985) and from a family systems theoretical viewpoint (Minuchin, 1974; Jones, 1987).

Numerous empirical studies have been conducted in the sphere of early separation and loss, and several studies have found empirical evidence that supports the concept of neglect or deprivation resulting from early separation from, or loss of, a primary caretaker. Links et al. (1988) found that early separation in BPD was more often due to marital separation of the parents whereas in the comparison group, early separation was more often due to death of a parent. They concluded that early parental loss that is due to separation rather than death may predispose a
child to BPD. However, the magnitude of this impairment may be partly
determined by the protection conferred on the child by the remaining parent.

Davis and Akiskal (1986) hypothesized that for some borderlines “early
separation and loss may permanently affect the neural pathways of biogenic
amines and endorphins that underlie the reinforcement mechanisms. A disruption
of such reinforcement mechanisms is an appealing etiology for much of the self-
destructive and socially inept behaviours that characterize borderline patients”
(Davis & Akiskal, 1986, p.680).

According to Clarkin et al. (1992), the association of early loss with
psychopathology, particularly BPD, may be too simplistic. Thus, more complex,
comprehensive, and testable aetiological models are needed.

Recent studies support sexual and physical abuse in early childhood as part of the
aetiological chain of events in the development of BPD (Zanarini et al., 1989;
Ogata et al., 1990; Stone, 1990). It appears that BPD stems in part from the early
and prolonged effects of physical and sexual abuse on the child’s developing
personality. In addition, the interaction between parental over-involvement and
malevolence may be the mechanisms by which these traumas act.

Biparental failure has also been levelled as an aetiological factor in BPD. In
studies of BPD patients’ early family experiences, a significant proportion of both
mothers and fathers show significant impairment and failure to carry out their
parental functions (Frank & Paris, 1981; Soloff & Millward, 1983; Akiskal et al.,
1985). Parental psychopathology may create a home environment that impairs a
child’s learning or development, thus placing the child at risk for school failure
and adult psychopathology, especially BPD.
A number of investigators have carried out family history studies to examine whether BPD is more frequently found in the families of BPD patients. Goldman, D’Angelo & DeMaso (1993) demonstrated in their study that the families of the patients with BPD had significantly greater rates of psychopathology, particularly in the areas of depressive, substance abuse, and antisocial disorders. Their findings support the hypothesis that a history of significant family psychopathology is associated with BPD. In Goldman’s words “What appears to shine as a beacon through the foggy nature of this disorder is its association with abuse and parental psychopathology” (Goldman et al., 1993, p.1835).

It would appear that abuse and parental psychopathology is not a specific factor in the aetiology of BPD, but is rather a more generic type of critical environmental failure when combined with biological vulnerability across a wide range of developmental circumstances.

Disturbance in family structure and relationships has been a point of departure in studying the aetiology of BPD. A structural approach emphasizes concepts such as parental coalitions and intergenerational boundaries (Minuchin, 1974). As the early therapists struggled to understand a patient’s disturbed behaviour, they became aware that its roots and meaning might be found in the context of the family system. In 1931, Sullivan emphasized the importance of studying symptoms in context. More recently, Mandelbaum (1977) stated that the overall “symptom picture ... the patient presents, whether neurotic, borderline, or ... psychotic ... calls for an examination of those developmental factors in family life that have produced dysfunctional structures and roles” (Mandelbaum in Jones, 1987, p.285).

Family experiences, relationships, and family structure as described above, may be aetiological in the BPD patient’s experience of extreme emotionality, invalidation, and fear of abandonment. The disruption of the parent-child relationship may lead to pathological attachment patterns in adulthood, such as the borderline’s characteristic unstable, intense relationships.
3.7.2.4 Cognitive behavioural viewpoint

Until very recently, behaviourists and cognitive-behaviourists have generally neglected BPD in both theoretical writings and research investigations. This is not surprising given the fact that the borderline construct was developed within the psychoanalytic community (Paris, 1993). This neglect is fast disappearing with the work of Linehan (1987), Millon (1987), and Young, (1983); Young and Swift (1988).

Linehan’s (1987) biosocial-behavioural theory delineates “dysfunction in emotion regulation” as the core characteristic of BPD (in Beck & Freeman, 1990, p.184). Emotional dysregulation is a product of an oversensitive and overreactive emotional response system and an inadequate emotion regulation system - that is, the core pathology is a combination of emotion vulnerability and the inability to regulate affect. She believes that emotion dysregulation is probably physiologically based and responsible for the BPD individuals’ dramatic over-reactions to events and for their impulsive acts. She also hypothesizes that significant others tend to discount the emotional experiences and distress which the developing child is expressing. The child in turn learns to take a disparaging, punitive attitude to her own emotions. There is a constant failure by the child to learn adaptive ways of regulating emotional life or of handling interpersonal problems. The continuing emotional dysregulation leads eventually to reliance upon destructive, particularly self-destructive, ways of coping with emotional pain (Barley, Buie, Peterson, Hollingsworth, Griva, Hickerson, Lawson & Bailey, 1993).

The basic assumption of Linehan’s dialectical theory is that psychological disorders represent systemic dysfunctions which are mediated by the individual and the environment, thus constituting a process of reciprocal influence (Paris, 1993). The theory proposes that the defining characteristics of BPD (intense, labile emotions and interpersonal relationships, suicidal and other self-injurious
behaviours, and the disturbance of the self and identity), are sequelaes of the
effects of this fundamental interaction between emotion dysregulation and
invalidating environment (Linehan & Heard in Clarkin et al., 1992).

Millon (1987) provides a view based on social learning theory in which he
attributes the lack of a clear sense of identity characteristic in BPD, to a
combination of biological, psychological and sociological factors acting in
concert. This lack of a clear sense of identity leads to poorly coordinated actions,
poorly controlled impulses, and a lack of consistent accomplishment. As a result
of this inconsistent strategy for dealing with problems, the BPD patient looks to
others for protection and reassurance while at the same time rejecting these
sources of support. Thus, intense conflicts arise regarding dependency and
assertion.

Young (1983 in Young and Swift, 1988) have developed a cognitive-behavioural
model for the aetiology of BPD in which he postulates that “early maladaptive
schemas” (stable and enduring patterns of thinking) can develop during childhood
and result in maladaptive behaviour patterns that reinforce the schemas. He sees
nine maladaptive schemas as being characteristic of BPD: abandonment/loss;
unlovability; dependence; subjugation/lack of individuation; mistrust; inadequate
self-discipline; fear of losing emotional control; guilt/punishment; and emotional
depprivation. Unfortunately, Young does not describe how these schemas produce
BPD.

The three preceding perspectives focus on different aspects of BPD. Linehan
hypothesizes that a defect in emotion regulation is the core of BPD; Millon
emphasizes the individual’s identity disorder as playing a central role in BPD; and
Young sees BPD as being based on strongly held assumptions that are acquired
early in development and play an important role throughout life (Beck & Freeman,
1990).
3.8 SUMMARY

From the research it appears that both researchers and clinicians tend to differ both in their practical orientation and theoretical viewpoints regarding the development and manifestation of BPD. Some accord biological factors such as genetic transmission or changes in neurotransmitter activity as being the primary pathway to the development of BPD. Others accord psychological factors such as physical and/or sexual abuse or deleterious childhood experiences; and still others accord social factors such as the social structure or environmental influences. The orientation one chooses influences the type of research conducted (empirical or qualitative) and also the treatment selected. The accompanying symptoms of BPD are targeted and treated variously by using a host of pharmacological agents such as antidepressants, minor tranquillizers, anticonvulsants, mood stabilizers, and neuroleptics (Cowdry & Gardner, 1988; Soloff et al., 1986) and/or the symptoms are treated with psychodynamic, cognitive-behavioural, supportive, or family-systems therapy or a combination of these treatment modalities. However, to date, BPD poses a problem as it does not seem to respond to any type of treatment.

In addition, the BPD concept itself has undergone changes in its meaning and continues to evolve as evidenced in revision of the diagnostic criteria in DSM-III, III-R and IV. However, despite these problems there is growing evidence that the behavioural sequelae manifest in BPD, (for example, impulsivity, affective instability, deviant thought and communication patterns, and difficulty in drawing logical inferences) may be indicative of cognitive impairment. As BPD is a chronic illness with formidable morbidity and mortality, and as neither a single factor nor a component “syndrome” seems to be satisfactory in explaining the aetiology, it demands a different approach. Thus, when the psychosocial factors of the biopsychosocial model have been exhausted – to no avail – it would be prudent to turn to the “biopsycho” side of BPD in search of a neuropsychological substrate.
The next chapter will discuss organic brain dysfunction and the neuropsychological concomitants of BPD with regard to research conducted in this sphere. Research conducted on frontal system dysfunction, cerebral structure, possible role of the limbic system, neurological dysfunction, brain imaging techniques, and neuropsychological testing results will be evaluated.
CHAPTER FOUR

ORGANIC BRAIN DYSFUNCTION AND BORDERLINE PERSONALITY DISORDER (BPD)

4.1 INTRODUCTION

Although research findings are not totally convincing, there is reason to suspect that a subtle form of organic brain dysfunction underlies the development of BPD. Attention has more recently focused on the neurological and biological underpinnings of some of the cardinal symptoms of BPD, such as cognitive distortions, affective instability and impulsivity. Although numerous investigations have been conducted, it would seem that most clinicians do not have the time, funds or facilities to evaluate BPD patients "biologically" - thus this form of investigation rests in the hands of researchers. However, psychologists who form part of the multidisciplinary team at mental institutions are conducting neuropsychological assessment more frequently. This form of investigation is relatively economical and may contribute to a different aetiological pathway being found in BPD.

In this chapter, research conducted and hypotheses postulated will be discussed with regard to frontal system dysfunction; cerebral structure, possible role of the limbic system; neurological dysfunction; and neuropsychological testing results in BPD.

4.2 BPD AND FRONTAL SYSTEM DYSFUNCTION

Borderline Personality Disorder is characterized by dysregulation of the stress response; emotional instability, including dysphoria, anxiety, and anger;
impulsive, maladaptive behaviour; and, as a result, disordered interpersonal relations. These elements are all associated with frontal lobe disease. In fact, neuropsychological deficits characteristic of frontal lobe dysfunction have been reliably demonstrated in the “dramatic personality disorders” (Burgess, 1992). It is proposed that a deficit in the functional system regulating experiential learning (that is, integration and assimilation of reinforcement) is central to the maladaptive behaviours of BPD.

Impulsivity theory suggests that dysfunction in the frontal regions of the brain (specifically, the prefrontal cortex) combined with impulsivity, could predispose a person to violent behaviour via loss of inhibition normally exerted by the frontal cortex on subcortical structures that facilitate aggression, impulsivity, loss of self-control, and poor social judgement; behavioural changes, such as emotional and aggressive outbursts and argumentative behaviour; loss of intellectual flexibility and concept formation skills; and poor sustained attention, concentration, and reasoning ability (Stuss & Benson, 1986).

The frontal lobes are most closely identified with the following cognitive abilities: forming of concepts, executive abilities, attention and arousal, perseverance, awareness, and empathy. The clinical literature on BPD contains frequent references to problems these patients have with attention/concentration, memory, learning, confusion, and perceptual distortions. These difficulties, which probably contribute to the problems encountered by borderlines in daily living as well as in treatment, have led to increased interest in neuropsychological functioning in this disorder.

Specifically, efforts have been made to discern whether the cognitive problems encountered by BPD patients are secondary, reflecting defensive ego operations, or whether they are primary, organically based deficits (Swirsky-Sacchetti, Gorton, Samuel, Sobel, Genetta-Wadley & Burleigh, 1993). Recent studies have demonstrated significant levels of cognitive impairment in persons with BPD, particularly in tests of planning/sequencing cognitive functions (Burgess, 1992).
Andrulonis, Glueck, Stroebel, Vogel, Shapiro and Aldridge (1980) postulated that an “organic” subgroup exists amongst BPD. They also suggest that BPD is a syndrome: “a constellation of behaviours that frequently coexist rather than a disease state with a single etiology” (Andrulonis et al., 1980, p.127). It is with this in mind that frontal system functioning and consequent behaviour will be discussed.

Conceptual thinking comprises the ability to draw abstractions from perceptual experience and to manipulate abstract ideas in an organized and effective way. Conceptual ability and cognitive flexibility (which refers to the ability to move from one operating principle to another as circumstances require), are central to the ability to function effectively, especially in social interactions. The “executive functions” of the prefrontal cortex refer to the capacity for autonomous behaviours beyond the structures of external guidance, and include activity-related behaviours that are necessary for appropriate, socially responsible, and self-serving adult conduct. Included in these functions is the capacity for initiative, motivation, spontaneity, planning, judgement, insight, goal-directed behaviour, the ability to operate in favour of a remote or an abstract reward, the capacity for self-monitoring, and the flexibility required for self-corrections. Individuals with frontal lobe dysfunction are incapable of developing a conceptual framework for complex, goal-directed behaviour without the provision of external structure. They tend to lapse into inactivity, or they pursue idle, self-defeating impulses - a prominent feature in BPD.

Arousal is the ability to be awakened and to maintain wakefulness, and the ability to follow stimuli or respond to commands. The frontal lobes participate in the functions of arousal and attention as the rostral pole of a brainstem-frontal lobe axis that includes the thalamus and the ascending reticular activating system. The brainstem-frontal lobe system provides tonic levels of arousal and alertness through the reticular apparatus; phasic levels of alertness through the diffuse thalamic projection system; and selected and directed attention through the frontal-thalamic gating system which enables conscious, directed behaviour. It
appears to be under the influence of ascending reticular projections and
descending impulses from the frontal cortex. Thus, afferent and efferent impulses
can be integrated, interpreted, and used to control sensory pathways. Pathology in
this system results in disorders of more complex behaviours, such as planning,
selection of behaviour, and monitoring of performance (Stuss & Benson, 1984).
Although on the one hand, there may be difficulty in sustaining attention, on the
other, these patients are capable of intense concentration on matters that are novel,
stimulating, or intensely gratifying - a term known as “limbic tagging”. It is
thought that input from the frontal lobe may be protected from abnormally rapid
decay by limbic reinforcement because of the projection of several areas of the
prefrontal cortex onto the limbic system via the cingulate, and from there to the
hippocampus (Gualtieri in Ratey & Fogel, 1995). If there is not a high level of
limbic activation to begin with, preservation of a prefrontal activity, such as
concentration or maintaining a set, may be blunted (Gualtieri in Ratey & Fogel,
1995).

Affective and emotional disorders are a consequence of the regulatory activity of
the frontal lobes and are also the most dramatic signs of frontal lobe disease. The
limbic system influences the frontal lobes and modulates externally directed
higher forms of drives whereas the prefrontal cortex influences internally directed
limbic drive states.

Reduced frontal lobe influence over subcortical drive states results in behavioural
disinhibition. Lesions in the orbitofrontal cortex which has extensive connections
to the septal area and the amygdala, produces disinhibition in these areas, and
behaviours such as impulsivity, excitability, euphoria, inappropriate irritability,
anger or rage are demonstrated (Gualtieri in Ratey & Fogel, 1995). These
behaviours are characteristically seen in BPD, and may indicate a subtle form of
dysfunction in the orbitofrontal cortex.
4.3 CEREBRAL STRUCTURE IN BPD

Findings of increased neurological soft signs (Gardner et al., 1987), electroencephalographic (EEG) abnormalities (Snyder & Pitts, 1984; Cowdry et al., 1985), and histories of neurological dysfunction, for example, attention deficit hyperactivity disorder (ADHD), seizures, and head trauma (Andrulonis et al., 1980), in patients with BPD has raised the possibility that structural brain abnormalities occur with increased frequency in this population.

Lucas et al. (1989) conducted computed tomographic (CT) scans of brains of 31 BPD patients and 28 normal controls. They analyzed the scans for ventricle-brain ratios, third ventricular size, and evidence of frontal atrophy, and found no significant differences between the two groups on any of the measures except for a narrower third ventricle in BPD patients. Their findings indicated that while BPD patients may show signs of subtle neurological dysfunction, they do not show evidence of structural brain pathology. This is consistent with Snyder et al.’s (1983) report of the absence of ventricular enlargement by clinical evaluation in 26 BPD patients and in contrast to the frequent findings of enlarged lateral or third ventricles in schizophrenic patients (Lucas et al., 1989).

4.4 POSSIBLE ROLE OF THE LIMBIC SYSTEM IN BPD

The limbic system (incorporating the hippocampus, amygdala, limbic striatum and prefrontal cortex) is thought to influence memory, learning, emotional states and responses, and aggressive behaviour. Symptoms such as intense, unstable interpersonal relationships, affective instability, aggression, frequent suicidal thoughts or attempts, and self-destructive behaviour, are the hallmarks of BPD. It is now hypothesized that these symptoms may be due to limbic system irritability (Andrulonis, Glueck & Stroebel, 1981) or due to serotonin dysregulation (Coccaro, Siever & Klar, 1989).
Studies conducted by Stone (1981); Herman, Perry and Van der Kolk (1989); and Ogata, Silk and Goodrich (1990), have revealed a strong association between early abuse and the development of BPD. The connection between early abuse and the limbic system seems due in part, to a phenomenon known as “kindling” in which repeated, intermittent stimulation results in long-term alterations in neuronal excitability. Van der Kolk and Greenberg (1987) have proposed that repeated traumatization such as child abuse may lead to limbic kindling and to the emergence of neurological abnormalities which can then lead to inappropriate aggression and sexual activity. It is noted that the “kindling” may have been used too loosely in the context of abuse, as the situation in “kindling” is subthreshold.

Although the research on early abuse, limbic system dysfunction and BPD has been inconclusive, it nevertheless provides a platform from which to further investigate BPD in the future.

4.5 NEUROLOGICAL DYSFUNCTION IN BPD

In the late 1980’s it was postulated that a subgroup of BPD’s probably had some type of organic brain impairment. Various researchers have investigated this possibility by conducting EEG studies, neurodevelopmental studies, history of trauma studies, and by neurological examination.

In a chart review, Andrulonis et al. (1981) found that 14% of their non-schizotypal borderline patients had a history of head trauma, encephalitis, or epilepsy, and that 26% had a history of attention-deficit disorder (ADD), and/or a learning disability (LD). They also found that BPD patients were more likely than affective disorder patients, to have a history of organic disturbance.

In 1983, Soloff and Millward assessed the neurodevelopmental histories of BPD. They retrospectively compared the histories of 45 BPD, 32 depressed, and 42 schizophrenic patients. They found that the BPD subjects reported more
complications of pregnancy and had a greater incidence of learning difficulties as children.

EEG studies have been conducted in BPD patients due to the similarity of symptoms reported in both BPD and Temporal Lobe Epilepsy (TLE). These symptoms include depersonalization, derealization, impulsivity, transient psychosis, and affective instability. Snyder and Pitts (1984) compared the EEG records of 37 male BPD patients with 31 male dysthymic subjects. They found that 19% of BPD had marginally abnormal EEG’s and another 19% had definitely abnormal EEG’s. The combined results (marginally and definitely abnormal EEG’s) were significantly higher in BPD’s than in dysthymic controls. In addition, slow-wave activity was significantly more common in BPD subjects.

Gardiner, Lucas & Cowdry (1987), administered a “soft sign” neurological examination to 17 BPD and 22 normal controls. All subjects were female. They found that BPD’s had a significantly greater number of soft sign neurological abnormalities, indicating abnormal neurological development or a disturbance in brain organization.

In Zanarini et al. (1989) study, their findings indicated that it was quite common for BPD’s to have some form of subtle neurological dysfunction, and this dysfunction was not specific to BPD’s as it was equally common in Axis I control subjects.

The above discussion has mentioned only a few of the studies conducted on neurological dysfunction in BPD. Yet, despite the numerous studies conducted in this field, very little empirical work has been done to test the validity.
4.6 BRAIN IMAGING AND BPD

Brain imaging is a relatively new technique which attempts to “visualize” and “measure” processes and structures within the cranium. Although these measures have been used with increasing frequency in Axis I disorders, they have not been applied to BPD (an Axis II disorder) until recently because of a lack of reliable and consistent diagnostic criteria for BPD and also because the aetiology was thought to be primarily psychological. In view of the overlap of BPD symptomatology with other personality disorders, such as Schizotypal, Antisocial, and Histrionic, and given the continuing dialogue concerning the relationship of BPD to mood disorders, it is useful to compare the structural brain images of BPD patients with “Other” personality disorders, Major Depressive Disorder, and Schizophrenia. Over the last decade the few studies that have used brain imaging indicate that this technique may become a powerful tool in the search for a pathophysiology of Borderline and/or Schizotypal personality disorders. The results of structural imaging studies seem to indicate that BPD patients do not have the same parameters as patients with schizophrenia or with delusional depression in that BPD patients do not appear to have enlarged ventricles (Schulz et al., 1983; Snyder et al., 1983). However, this is stated with caution as no known studies have employed the P.E.T. (Position Emission Tomography) scan, which is a more sensitive instrument and fundamentally functional. Although the results to date are not striking, future brain imaging studies should include pharmacological and/or cognitive activation probes during functional brain imaging scans in order to elicit a better understanding of BPD.

4.7 NEUROPSYCHOLOGICAL CONCOMITANTS OF BPD

“It is by testing that we discern fine gold.”

Leonardo da Vinci

The literature on BPD is replete with examples of memory impairment and cognitive distortions occurring in psychotherapy and in daily life. Psychological testing dives below the surface of ordinary experience, tapping layers of mental
functioning not necessarily accessible to the clinician. However, in the past testing was limited to the WAIS and Roschach tests. In 1975 Gunderson and Singer drew attention to the observed good performance of BPD patients on the structured Wechsler Adult Intelligence Scale (WAIS), and poor performance on projective techniques such as the Rorschach, where structure is low, suggesting that BPD patients show ordinary reasoning and communication in highly structured situations but demonstrate flamboyantly deviant reasoning and thought processes in unstructured situations (Singer in Hartocollis, 1977; Stone, 1980). This leaves unaddressed a vast area of neuropsychological functioning in BPD (O'Leary, Brouwers, Gardner & Cowdry, 1991), and the effects of brain dysfunction on BPD which may be better discerned by examining the neuropsychological functioning of these patients (Paris, 1993).

Cornelius, Soloff and George (1989) evaluated the significance of selected neuropsychiatric abnormalities in the aetiology of BPD, comparing BPD subjects with historical controls. They found that the BPD subjects performed normally on measures of memory, language, motor, and visuospatial functioning, and concluded that neuropsychiatric abnormalities are "at most an uncommon etiology" of BPD. Paris (1993) is critical of this study and maintains that the behavioural pattern exhibited in BPD (impulsivity, self-mutilation, and affective disinhibition) is suggestive of dysfunction in limbic and frontal sites.

In 1991, O'Leary et al., subjected 16 BPD outpatients and 16 normal volunteers to a battery of neuropsychological tests which assessed general intelligence and problem solving; memory; and visual perception with the aim of detecting whether there were cognitive deficits in areas of perception, learning, and memory. They found that the BPD subjects demonstrated impairment on memory tests requiring uncued recall of complex, recently learned material, and on visual perceptual tasks requiring discrimination and filtering. They concluded that BPD subjects display dysfunction in the non-dominant and dominant temporal lobes.
Burgess (1990) used tests sensitive to information-processing and found that frontal system impairment in the form of deficits in attention and memory correlated with self-mutilation. A second study confirmed that self-injury in BPD subjects was highly correlated with attentional testing and memory testing, implying impairment in frontal system functioning.

Stone (1992) found a peculiarity in the memory capacity of BPD subjects in that they remain at the mercy of the latest sensory impression in personal relationships. He concluded that the links between short- and long-term memory were severed.

Van Reekum (1993) studied Cornelius et al's (1989) cohort of ten BPD patients with a neuropsychological screening battery and found that seven of the nine patients who completed interpretable testing showed evidence of frontal and possibly primarily orbital-frontal system dysfunction in the form of impulsivity, cognitive inflexibility, poor self-monitoring, and perseveration. According to Cummings (1985), there is a striking similarity between the behavioural sequelae of orbital-frontal lesions and the behavioural pattern of patients with BPD: “Lesions in this region appear to divorce frontal monitoring systems from limbic input, resulting in a disinhibited behavioural syndrome where impulses are acted on without consideration of consequences, antisocial actions occur, and emotional lability is marked” (p.63). These deficits were noted most often in the Wisconsin Card Sorting Test (Berg, 1948), the Trails B (Army Individual Test Battery, 1944) and the Rey-Osterreith Complex Figure (copy recall) (Osterreith, 1944). Performance on other neuropsychological tests was normal. Although this pattern of cognitive deficits is consistent with the behavioural disturbance that defines BPD and with the poor response to psychotherapy in most instances, the data suggest that larger controlled assessments utilizing further measures of frontal functioning should be made (Paris, 1993).

Judd and Ruff (1993) compared 25 BPD subjects with 25 matched archival controls. They based their study on Luria’s theory of higher cortical functions, using the San Diego Neuropsychological Battery to test visuospatial and verbal attention; concentration; learning and memory; motor ability; cognitive flexibility
and fluency; and logical thinking and planning. They found BPD subjects to be impaired on visuospatial tasks requiring learning and recall of novel, complex information; and also on tasks measuring visuospatial discrimination, processing speed and fluency. In particular, they suggested that BPD subjects displayed odd reasoning; poor concept formation; difficulty drawing logical inferences about relationships between people and events; dichotomous thinking; quasi-psychotic thinking; an integrative deficit; deviant thought and communication patterns; an inability to maintain or shift cognitive set; disruptions of boundaries between concepts; and lapses in logical thinking on tasks requiring extensive use of language.

4.8 CONCLUSION

It appears from the literature that BPD is a clinical problem of great significance because of the high prevalence, high morbidity, and high mortality rates. Specifically, BPD warrants attention in terms of aetiology and treatment.

The nature of BPD has been clearly defined according to the DSM-IV diagnostic criteria. However, the problems lie in the aetiology and treatment. As no clear-cut aetiological modality exists, there can be no clear-cut treatment modality.

Thus far, biological, psychological and social risk factors have rendered little in the way of clarity as to the aetiology of BPD as there are inherent flaws in methodology and a dearth of systematic research techniques.

Psychological research into the aetiology of BPD has involved measurement of psychological risk factors through retrospective designs (for example, asking patients to remember their childhood experiences). Because BPD patients tend to have distorted perceptions of their relationships in adulthood, their memories of childhood could be equally unreliable. Herman et al. (1989) found that childhood experiences account for about a third of the variance in borderline
symptomatology, leaving two thirds to be explained in other ways. It would seem
that the explanation for the lack of specificity of the psychological risk factors to
BPD might not lie with childhood experiences at all. It may be that psychological
factors can lead to BPD only in the presence of nonpsychological risk factors. The
retrospective nature of research into psychological factors causing BPD is a
limitation, and as yet there have been no prospective designs where a cohort of
children is followed longitudinally into adulthood.

Social risk factors are probably the most difficult to examine empirically. As with
biological and psychological variables, social stressors can be associated with the
development of specific disorders only in interaction with other risk factors. The
general method for examining social risk factors is epidemiological research.
While it is possible to determine the prevalence of BPD in different social classes,
over time and across cultures, it is difficult to establish the direction of causality.

Thus, no clear aetiology leading to adequate treatment exists. It is also clear that
no adequate biochemical model has been established. Possibly the problem lies
therein that no exclusive domain for aetiology can be found, or perhaps it lies in
the interaction between physiological and psychological variables.

There is sufficient preliminary evidence to suspect that a subtle form of organic
brain dysfunction underlies the development of BPD. Research conducted thus far
indicates that dysfunction in the orbito-frontal cortex (Andrulonis et al., 1981;
Gardner et al., 1987; and Soloff & Millward, 1983); or in the limbic system
(Herman et al., 1989; Ogata et al., 1990; and Stone, 1981) exists in at least a
subgroup of BPD individuals.

Recent neuropsychological studies have identified impairment in attention,
memory, visuospatial and visuoperceptual functioning (Burgess, 1990; O'Leary et
al., 1991; Stone, 1992; and Van Reekum, 1993) which has been variously
correlated with behavioural sequelae such as impulsivity, cognitive inflexibility,
poor self-monitoring, (Van Reekum, 1993), inappropriate anger, (Van der Kolk & Greenberg, 1987) and peculiarities in memory capacity (Stone, 1992).

Although there is considerable individual variability in the course of BPD, it is the impairment from the effects of the accompanying symptomatology (for example, substance abuse and/or risk-taking behaviour) coupled with the high risk of suicide, which underscores the urgency of finding a more definitive aetiological pathway to BPD.

Thus, a comprehensive neuropsychological investigation needs to be attempted in order to solve the BPD aetiological and treatment dilemma, as it is probable that neuropsychological deficits account for the problem and set it aside from other Axis II Personality Disorders.

It was therefore the purpose of this study to firstly determine the nature and extent of neuropsychological deficits in BPD by utilizing a comprehensive neuropsychological evaluation, encompassing all relevant dimensions in the neuropsychological substratum of human behaviour. In order to differentiate neuropsychological deficits in BPD from those shared by BPD with other groupings of Personality disorders, a control group of OTHER PD's was used. A NORMAL volunteer group was used to differentiate BPD and OTHER PD's from the general population. More specifically the following hypotheses were investigated:

**Hypothesis I**

The experimental group of subjects with Borderline Personality Disorder (BPD) will show significantly greater neuropsychological deficits in construction; orientation and attention; memory; perception; and concept-formation and reasoning, than the control group of subjects with Personality Disorders from DSM-IV Clusters A or C (OTHER PD) and the control group of NORMAL volunteer subjects.
Hypothesis II

The experimental group of subjects with Borderline Personality Disorder (BPD) and the control group of subjects with personality disorders from DSM-IV Clusters A or C (OTHER PD) will show significantly greater neuropsychological deficits overall when subjected to a battery of neuropsychological tests than the control group of NORMAL volunteer subjects.
CHAPTER FIVE

METHODOLOGY

5.1 INTRODUCTION

Borderline Personality Disorder (BPD) is a baffling and serious disorder that severely restricts the life of its sufferers. It is a disorder that despite the tremendous strides achieved by modern science, is still not fully understood. Furthermore, it has been treated with derision by virtue of its designated label “borderline”, and although there is now sufficient evidence to assert that the interaction of genetic, constitutional, neurological, and environmental factors impact in the aetiology of borderline pathology, the research findings in this area are frequently divergent and inconclusive.

Currently there is a need for well-chosen comparison groups from the spectrum of other Personality Disorders as it is not known from the studies conducted thus far, whether the pattern of impairment on memory and visuospatial tasks is particular to BPD or whether it also features in other Personality Disorders.

The present study included both an inpatient BPD group; an inpatient psychiatric control group (subjects diagnosed with Cluster A or Cluster C Personality Disorder); and a control group of normal volunteers.

Specifically the hypotheses were:

Hypothesis I

The experimental group of subjects with Borderline Personality Disorder (BPD) will show significantly greater neuropsychological deficits in construction; orientation and attention; memory; perception; and concept-formation and reasoning, than the control group of subjects with Personality Disorders from
DSM-IV Clusters A or C (OTHER PD) and the control group of NORMAL volunteer subjects.

**Hypothesis II**

The experimental group of subjects with Borderline Personality Disorder (BPD) and the control group of subjects with personality disorders from DSM-IV Clusters A or C (OTHER PD) will show significantly greater neuropsychological deficits overall when subjected to a battery of neuropsychological tests than the control group of NORMAL volunteer subjects.

### 5.2 SUBJECTS

The sample consisted of sixty subjects as follows:

Twenty subjects diagnosed with BPD formed the experimental group; twenty subjects diagnosed with Cluster A or C personality disorders formed the first control group (OTHER PD); and twenty subjects who were free of any psychiatric illness formed the second control group (NORMAL).

#### 5.2.1 Source of Subjects

The 20 persons from each of the two groups of voluntary inpatients (BPD and OTHER PD) were drawn from the psychotherapy unit (Wards 4 and 5) at Tara the H. Moross Centre (Tara Hospital), Gauteng. Every person admitted to the ward during the period January 1996 to December 1996 was assessed for suitability for inclusion in the study. This was done by the multi-disciplinary team working on the ward. Selection of subjects was further determined by inclusion and exclusion criteria so as to avoid overlap between categories. The remaining 20 persons (NORMAL) were drawn by self-selection from a group of volunteers most of whom were working on a temporary basis at Tara Hospital. This sample included
final year nursing students, 5th and 6th year medical students, undergraduate
university students, and a marketing agent.

The experimental group to be described consisted of a total of 20 subjects (17
females and 3 males) with a primary diagnosis of Borderline Personality Disorder,
aged between 20 and 50 years with an average age of 19.6 (BPD). They were
clinically diagnosed according to the DSM-IV (APA, 1994) criteria, by a team
comprising psychiatrists, psychologists, social workers, psychiatric nurses, and
occupational therapists. Selection of subjects was also determined by exclusion
criteria so as to avoid any overlap between categories. The sample was thus free
of complicating disorders such as other Axis II personality disorders and other
Axis I disorders, such as Major Depressive Disorder (MDD). Additionally, a
history of Learning Disorder (LD), Attention Deficit Hyperactivity Disorder
(ADHD), brain trauma, encephalitis, and/or Temporal Lobe Epilepsy (TLE) was
also exclusionary.

The first control group consisted of 20 subjects (13 females and 7 males) with a
primary diagnosis of Personality Disorder from either Cluster A or Cluster C
according to the DSM-IV (APA, 1994), aged between 22 and 56 years, with an
average age of 38.2 years (OTHER PD).

The second control group consisted of 20 subjects (13 females and 7 males) aged
between 18 and 46 years with an average age of 23.35 years. These subjects were
free of any psychiatric illness and consisted of one 4th year medical student, six
5th year medical students, five 6th year medical students, five final year student
nurses, two 1st year university students (Bachelor of Arts; Bachelor of
Commerce), and one marketing agent (NORMAL).

Although the three groups of subjects would not meet stringent criteria for
comparative purposes with regards to age (BPD average age 29.6; NORMAL
average age 23.4; OTHER PD average age 38.2), and gender (BPD 85% female;
OTHER PD and NORMAL 65% females), it was decided to use these subjects as
they consisted of the total available subjects meeting stringent inclusion and exclusion criteria at one of the major psychiatric centres in Gauteng at the given time. Moreover, as regards gender, BPD subjects tend to be more often diagnosed amongst the female population. The duration of this study was one year (from January 1996 to December 1996).

5.2.2 Biographic Profile of the Sample Groups

Listed below is a profile of the biographical composition of the three research groups, BPD, OTHER PD, and NORMAL (see also Table 5.2.1A, p.72).

Race: The BPD group and the OTHER PD group consisted exclusively of whites whereas the NORMAL group consisted of 75% whites, 10% Indian and Chinese, and 5% Coloured subjects.

Age: The three groups were clearly different with respect to their general level of age. The mean age of the BPD group was 29.6 years while that of the NORMAL group was 23.4 and that of the OTHER PD group was 38.2 years.

Marital status: As the NORMAL group are students, it comes as no surprize that most are single. It is unclear whether the married/divorced pattern for the BPD and OTHER PD groups are significantly different but it appears as if there were more married patients in the OTHER PD group. Of course, the OTHER PD group members were substantially older which might account for the higher percentage of married subjects.

Gender: The BPD group was represented by a higher percentage of females (90%) compared to the OTHER PD and NORMAL groups which were both represented by 65%. This is understandable when one considers the higher prevalence of BPD in females.

Education: It seems that the NORMAL group is more educationally trained than the other groups. Yet as is shown in Table 5.2.1A, they are on average significantly younger than the patients in the other two groups are. This can be put down to the fact that the NORMAL group was all students.
# Table 5.2.1A

## Characteristics of the Three Groups: BPD, Other PD, and Normal

<table>
<thead>
<tr>
<th>CHARACTERISTICS</th>
<th>BPD %</th>
<th>OTHER PD %</th>
<th>NORMAL %</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RACE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>100</td>
<td>100</td>
<td>75</td>
</tr>
<tr>
<td>Black</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Indian</td>
<td>0</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Coloured</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Chinese</td>
<td>0</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td><strong>AGE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-20 years</td>
<td>20</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>21-25 years</td>
<td>30</td>
<td>15</td>
<td>80</td>
</tr>
<tr>
<td>26-30 years</td>
<td>15</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>31-35 years</td>
<td>5</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>36-40 years</td>
<td>10</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>41-45 years</td>
<td>15</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>46-50 years</td>
<td>5</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>51-55 years</td>
<td>0</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>56 years</td>
<td>0</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td><strong>MARITAL STATUS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>5</td>
<td>45</td>
<td>10</td>
</tr>
<tr>
<td>Separated</td>
<td>20</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Divorced</td>
<td>10</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>Single</td>
<td>65</td>
<td>20</td>
<td>90</td>
</tr>
<tr>
<td><strong>GENDER</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>10</td>
<td>35</td>
<td>35</td>
</tr>
<tr>
<td>Female</td>
<td>90</td>
<td>65</td>
<td>65</td>
</tr>
<tr>
<td><strong>EDUCATION</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>More than Std. 10</td>
<td>40</td>
<td>55</td>
<td>85</td>
</tr>
<tr>
<td>Std. 10</td>
<td>50</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>Less than Std. 10</td>
<td>10</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>(but not less than Std.8)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>HANDEDNESS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right-handed</td>
<td>85</td>
<td>80</td>
<td>95</td>
</tr>
<tr>
<td>Left-handed</td>
<td>15</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td><strong>RELIGION</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jewish</td>
<td>10</td>
<td>5</td>
<td>15</td>
</tr>
<tr>
<td>Christian</td>
<td>85</td>
<td>80</td>
<td>70</td>
</tr>
<tr>
<td>Hindu</td>
<td>0</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Secular</td>
<td>0</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Non-believer</td>
<td>5</td>
<td>10</td>
<td>5</td>
</tr>
</tbody>
</table>

**Handedness**: Only 5% of the NORMALS group were left-handed whereas 15% of the BPD group and 20% of the OTHER PD group were left-handed. It is unclear whether this is of any significance.

**Religion**: Subjects in all three groups came from all religious affiliations and no discernible differences seem to exist between the research groups except for the fact that the majority of subjects in all three groups belonged to the Christian religion.
5.2.3 **Number of Subjects**

The total sample size of 60 subjects was drawn from all race groups and included both males and females. Although the total sample included predominantly white subjects this was not intentionally designed but rather related on the one hand to the level of education required for inclusion in the study, and on the other to the fact that not all of the neuropsychological tests included in the battery were culture-fair.

5.2.4 **Inclusion Criteria**

- Male and female subjects aged 18 years and over;
- satisfaction of the DSM-IV diagnostic criteria for Borderline Personality Disorder (301.73);
- a minimum level of education of Std. 8 or equivalent;
- persons from whom informed consent was obtained.

5.2.5 **Exclusion Criteria**

- Lack of fluency in English (unable to understand and complete the MCMI-II);
- presence of psychosis;
- use of alcohol, illicit drugs, or sedative-hypnotics within 48 hours of testing;
- any history of cocaine, amphetamine, or alcohol dependence, or any other chemical dependence within the past two months;
- Electro Convulsive Therapy (ECT) in the past three months;
- judgement clouded by heavy doses of psychotropic medication;
- a previous history of Temporal Lobe Epilepsy;
- a previous history of Learning Disorder and/or Attention Deficit Hyperactivity Disorder;
- head trauma with history of documented or known brain injury;
- neurologic or medical conditions known to affect cognitive function;
- any acute Axis I diagnosis, including major depression.

5.2.6 Assessment Instruments

On the basis of both DSM-IV criteria and MCMI-II (Millon, 1987) scores, 40 subjects were selected: 20 subjects who met both DSM-IV criteria for BPD and achieved a score over 100 on the Borderline Scale of the MCMI-II comprised the Borderline Personality Disorder (BPD) group, and 20 subjects who had DSM-IV Axis II diagnoses other than BPD, who did not meet the DSM-IV criteria for BPD and scored less than 85 on the Borderline Scale of the MCMI-II served as a comparison group (OTHER PD).

5.2.6.1 Millon Clinical Multiaxial Inventory - II (MCMI-II)

The MCMI-II is a 175 item, forced-choice, true-false, computer scored, psychodiagnostic instrument developed to assess a wide range of clinically relevant behaviours and personality characteristics. The purpose of the MCMI-II is both to provide information to clinicians regarding diagnosis and treatment of patients, as well as to generate standardized, theory-grounded individual scale scores and profile patterns for research purposes. It is an instrument developed exclusively for clinical populations, and as such is not applicable as a general assessment of personality in the "normal" population.
Both the original MCMI and its refinement, the MCMI-II, were constructed as operational measures of syndromes derived from a theory of personality and psychopathology (Millon, 1983). As such, they measure theory-derived variables directly and quantifiably. The 22 scales of the MCMI-II have been constructed in line with the DSM-III-R (APA, 1987) multiaxial diagnostic system. The MCMI-II is an attempt to strengthen the relationship of the original MCMI to DSM-III-R categories. It is important to note that there is not an exact correspondence between the MCMI-II and DSM-III categories, as Millon's personality typology differs in some ways from empirical methods used to develop the DSM-III-R criteria (Chick, Sheaffer, Goggin & Sison, 1993; Reich, 1985; Widiger & Francis, 1987).

The MCMI-II distinguishes between Axis II (enduring personality characteristics) and Axis I (acute clinical disorders). Scales that distinguish syndromes in terms of their levels of psychopathologic severity have also been constructed. In order to keep pace with the rapidly growing interest in personality disorders, two new personality scales have been added (6B: Aggressive/Sadistic Personality, and 8B: Self-Defeating Personality). Among other changes made are the addition of modifications in the procedures for correcting distortion effects (random responding, faking, denial, complaining), and the replacement of 45 items found to be unhelpful in discriminating between their referenced scales (Millon, 1987).

Literature and research data on the MCMI and MCMI-II suggest sufficient generalizability, dependability, and accuracy of diagnostic scale cutting lines and profile interpretation to justify their use (Millon, 1983; Reich, 1985). The usefulness of the MCMI-II is enhanced by its efficiency and cost-effectiveness (Skodol & Oldham, 1991). The inventory is small and convenient enough to facilitate use in all types of diagnostic and treatment settings, but at the same time does not sacrifice any clinically relevant information. The MCMI-II is geared to
the equivalent of a South African Standard 6 reading level. It is quick to use, and the majority of patients can complete it in 30 minutes.

The MCMI-II was used in this study as a preliminary diagnostic screening device for BPD and also to provide evidence of co-morbid Axis I and II disorders in both the BPD experimental group and the OTHER PD control group. Scale scores above 85 were considered to be suggestive of the presence of a DSM-IV disorder. The information obtained from the MCMI-II was coded and then analyzed by the researcher as further support for inclusion or exclusion from the BPD experimental group and the OTHER PD control group.

5.3 SELECTION OF THE TEST APPARATUS

The tests used in this study do not constitute a major battery such as the Luria-Nebraska or the Halstead-Reitan. Specifically, the tests and subtests have been chosen for their ability to reflect on several categories of neuropsychological functioning such as attention/concentration, simple and complex visual memory, verbal and non-verbal memory, visuo-perceptual skills, concept-formation and cognitive flexibility, abstract reasoning, problem solving, planning, hypothesis formation, speed of processing, and general psychomotor performance. In most cases the tests have scoring criteria, allowing for both quantitative empirical and qualitative study at a later stage. The tests seem to be able to illuminate in a varying degree the dysfunction thought to be present.

5.3.1 Validity in Neuropsychological Assessment

The internal and external validity of the tests used in neuropsychological assessment is subject to many criticisms. Internal validity is affected by uncontrolled historical factors such as educational level, premorbid level of
functioning, age, handedness, gender and socioeconomic status (Franzen, 1989). Testing effects such as duration of testing days, repeated measures, and fatigue are also problematic. The use of tests normed on other populations irrelevant to the testee, or that do not allow for normal human variation in terms of their ceilings are also liable to yield invalid results (Franzen, 1989).

External validity is threatened by the use of medication, the sequence of the tests used, and the problem of interpersonal interaction (Franzen, 1989). It is thus clear that the difficulty in validating neuropsychological tests must be accounted for in the research design, and statistically and operationally controlled for.

Either the researcher intends to use the tests to hypothesize areas of brain dysfunction, or otherwise to describe the behaviour observed in testing, and the comparison of those observations with the performance of others without conclusions as to underlying brain conditions. Franzen expresses his opinion on this issue:

... the ultimate purpose of validating a test is to inform the clinician or the consumer about the kinds of decisions that may be made, based on the results of the procedure in question ... the ultimate evaluative-validational demonstration of a test is its clinical utility (Franzen, 1989, p.51).

The idea given is that tests live or die in the pragmatic value of the information they give on the individual, as well as on the average. The purpose of evaluating the individual with Borderline Personality Disorder (BPD) is to define a picture that differs both from the individuals with other personality disorders (OTHER PD), and from normal individuals (NORMAL), in order to shed light on the difficulties experienced in treatment and rehabilitation. The information thus gleaned can lead to more suitable handling of the patient, and more effective rehabilitation of these notoriously difficult therapy candidates.
5.4 THE NEUROPSYCHOLOGICAL BATTERY

A description of the neuropsychological battery which was administered to 20 BPD subjects, 20 OTHER PD subjects and 20 NORMAL control subjects follows in this subsection. The reliabilities and validities of the applicable tests are explored and the methodological or measurement problems of specific tests are discussed.

5.4.1 Description of the Measuring Instruments

The following measures were administered to all of the subjects:

* Rey Complex Figure (A. Rey, 1941; Osterrieth, 1944)
* Logical Memory subtest of the Wechsler Memory Scale - Revised (D. Wechsler, 1945, 1987)
* Wisconsin Card Sorting Test (F.A. Berg, 1948; D.A. Grant and Berg, 1948)
* Gottschaldt Embedded Figures Test (Gottschaldt, 1928).

In most cases the tests have scoring criteria allowing for both quantitative empirical and qualitative study at a later stage. The tests also seem to illuminate in varying degrees the dysfunction thought to be present.

5.4.2 Constructional Functions

5.4.2.1 Rey-Osterreith Complex Figure Test (RCF)

The Rey-Osterreith Complex Figure Test was developed by Rey in 1941, and standardized by Osterreith in 1944 (Lezak, 1995; Spreen and Strauss, 1998). This test is both a test of frontally-based planning as well as being able to evaluate non-
verbal memory function of the right temporal lobe (Kolb & Whishaw, 1996). It assesses constructional functioning, visuographic copying and visuographic memory. As such it is devised to investigate both perceptual organisation and visual memory.

The procedure involves having the subject copy a complex visual figure and then, without prior warning, reproduce it from memory at 3 minute (immediate) and 45 minute (delayed recall) intervals. As the patient is not told that he/she will have to remember the drawing, recall after interference comes as a surprise, with right temporal lobe lesion patients suffering most (Beaumont, 1983). The delayed recall is thought to be more sensitive than the 3-minute recall to the presence of memory deficits (Loring, Lee, Martin & Meador, 1988). Performance on the two recall trials (after 3 minutes and after 45 minutes) helps the examiner sort out different aspects of constructional and memory disabilities that might contribute to defective recall of the complex figure. The recall task is an indicator of a high level of nonverbal memory. Constructional performance combines perceptual activity with motor response and always has a spatial component (Lezak, 1995).

The test material consists of a complex geometric drawing, which the subject is told, to copy in detail using a series of coloured pens. The coloured pens are given so that the steps taken in the sequence of copying can be later analyzed. Osterreith’s sequences can be used to determine the most common procedure for copying the figure (Lezak, 1983).

Scoring criteria for the RCF are strictly applied for both the correctness of the details and their placement, because slight drawing errors have been found to be significant in differentiating groups of patients with varying cerebral lesions. Allowance is made for the fact that it is difficult to draw a straight line without the use of a ruler, so that if there is a waver in a line or if the line descends or ascends slightly, there is no penalty for these slight inaccuracies. Copy and memory trials are scored in the same manner. The figure is typically broken down into 18 scoreable elements, 0.5 - 2.0 points being awarded for each element, depending on
accuracy/distortion, and also location of its reproduction (Lezak, 1995): 2 points are awarded if the unit is correct and placed properly; 1 point is awarded if the unit is correct and placed poorly; 1 point is given if the unit is distorted but placed correctly; ½ point is given if the unit is distorted and placed poorly; and 0 point is awarded if the unit is absent or not recognizable. The highest possible score is 36. One can calculate a percentage recall score \((\text{CF recall} / \text{CF copy}) \times 100\) in order to remove the effects of the level of performance on the copy administration from the memory performance (Lezak, 1995).

Interpretation of the RCF should consider not only the actual score but also qualitative aspects of performance such as distortion and misplacement, approach or style, and level of organization. A disorganized piecemeal approach to the copy of the figure may result in an accurate production, but recall tends to be poor. In the memory production, a piecemeal strategy is very rare after age nine. In older children and adults, errors or distortions are quite common in the memory condition but are rare in the copy condition. A piecemeal approach to the copying of the RCF is characteristic of patients with either left or right hemisphere lesions (Binder, 1982). However, the drawings by the right-brain damaged patients tend to be less accurate and more distorted than those of their left-sided counterparts. Differences between patients with parietal-occipital lesions and patients with frontal lobe lesions have also been noted on the copy trial (Lezak, 1995). Patients with posterior lesions are more likely to have difficulty with the spatial organization of the figure whereas patients with frontal lobe lesions are more likely to have difficulty planning their approach to the task (Kolb & Whishaw, 1996). According to Walsh (1991), dementing patients have the greatest difficulty in copying the figure. Lezak (1995) also reports on findings by Messerli (1979) who found that frontal patients made more errors 75% of the time. Moses (1983) reports that patients with left hemisphere lesions may reproduce the outline, and lose the internal working, and those with right hemisphere lesions may lose the outline on recall.
The RCF is often perceived to be a test of memory. However, the test is more complex, and interpretation of the recall score must consider whether the initial copy is performed adequately. There is a tendency for patients with right hemisphere lesions to perform more poorly on the recall trial than do patients with left hemisphere disturbances. However, the test does not provide a perfect predictor of the side of the lesion.

Analysis of qualitative features (for example, distortion of overall configuration, major mislocating) may be helpful in distinguishing laterality of dysfunction. When the initial copy is performed satisfactorily, mislocation and distortion on the recall trial tend to be characteristic of patients with right, as opposed to left, hemisphere dysfunction (Loring, Martin & Meador, 1990). Finally, information from the test may also be useful in differentiating among different memory disorders (Bigler, 1983).

No information is available regarding the validity of the test (Franzen, 1989). Females reportedly perform at a higher level than males, and the test is sensitive to age. Inter-rater reliability may be as high as 0.94. The test has indicated some need for an intact right temporal lobe, in investigation of the RCF’s construct validity (Franzen, 1989).

There are few normative studies based on large samples of healthy people. Kolb and Whishaw (1985) provide normative data derived from Canadian samples of school children, ages 6-15, and healthy adults, aged 16-44, and Spreen and Strauss (1998) give data for healthy, well-educated (mean = 13.2 years) people, aged 50-85. Both age and intellectual level contribute to performance on the RCF. There is little decrement in copy scores with advancing age. Delayed-recall trials show little decline until the eighth decade. Finally, scores on the RCF show a modest correlation (r=23 - r=47) with measures of general intellectual ability.

The most useful aspect of the RCF is its value in determining the strength of visual memory, as well as perceptual organization. Research has shown that
delayed visual memory procedures are more valuable than the Wechsler visual memory design method, or the Benton visual retention method (Larrabee, Kane, Schuck & Francis, 1985). When using the RCF, there is thus no need for Wechsler memory scale designs, nor geometric reproduction designs, as these become redundant. Furthermore, correlations with RCF are of the low order of 0.28 (Franzen, 1989). Although the scoring criteria provide an objective measure of non-verbal memory, the test has the drawback that depressed or poorly motivated subjects may perform poorly not because there is right temporal lobe damage, but because they refuse to try to recall the figure. There is no easy solution to this problem, since all tests of non-verbal memory are subject to this complication (Kolb & Whishaw, 1996).

5.4.3 Orientation and Attention

5.4.3.1 Digit Symbol subtest of the Wechsler Adult Intelligence Test - Revised

The Digit Symbol subtest of the WAIS-R is a multifactorial task which requires a number of preserved skills. In order to rapidly transcribe symbols that are paired with digits in a reference key, the respondent must have at least adequate motor speed; paired associate learning; and the ability to scan back and forth and to and from the reference key. The test measures clerical motor speed and processing, the ability to adapt to a novel task, incidental memory, concentration and attention, ability to monitor errors, visual acuity, psychomotor performance and visual discrimination and filtering, and has a considerable attentional component. In addition, Digit Symbol may measure information-processing capacity and speed of non-verbal learning (Royer, 1971; Lezak, 1995). It is considered to be an indicator of sustained attention but also measures partly response speed and visuomotor coordination. It is the most sensitive among the WAIS-R subtests to brain damage even when damage is minimal (Hirschenfang, 1960; Lezak, 1995). The test illuminates deficits in motor persistence, sustained attention, response speed, immediate visual memory and visuomotor coordination (Berg, 1983), and is thus very sensitive to multiple aspects of brain dysfunction.
In essence the test is a coding task: numbers have to be transferred ("coded") into simple symbols. The working sheet contains a key for pairing each number (from 1-9) with a different symbol. Following the test instructions, the respondent is given a practice trial ("examples") in which he/she has to code a few numbers. The test working sheet consists of 100 small blank boxes each of which is paired with a number. The numbers are given in a random order. The respondent’s task is to fill in correctly the blank boxes with the symbol that belongs to the respective number. This has to be done as quickly as possible (i.e. within 90 seconds).

5.4.4 Memory

5.4.4.1 Logical Memory subtest of the Wechsler Memory Scale - Revised

The WMS-R grew out of the original Wechsler Memory Scale designed by David Wechsler in 1945 and revised in 1987. The Logical Memory subtest consists of two brief stories that are read to the subject. After each one, the subject immediately retells the story from memory (Logical Memory I). Following a delay of 20 minutes the subject is asked to relate each story, as a measure of delayed recall (Logical Memory II). The Logical Memory subtest is an auditory-verbal memory task which is designed to evaluate learning and memory for complex novel verbal information. In fact, it is a verbal analogue to the RCF. Specific memory deficits such as problems with initial learning, retention, or recall of material can be elucidated using this subtest which is sensitive to impairment in the dominant temporal lobe. The test is scored both for exact repetitions, as well as for semantically correct ones, as long as the meaning remains essentially unchanged.

The WMS-R scale is intended for individuals aged 16-74 years, with each index having a mean of 100 and a standard deviation of 15. Clinical investigations using the WMS-R have shown that the test is sensitive to memory disturbances and may characterize the learning and memory disorders in a number of different patient
groups including those with Alzheimer's disease, Huntington's disease, multiple sclerosis, Korsakoff's long-term alcoholism, neurotoxic exposure, schizophrenia and depression (Lezak, 1995).

Normative data is included in the WMS-R manual which provides norms for individuals aged 16.0 to 74.11 years. The test was standardized on a large sample considered representative of the United States population. Level of education is highly correlated with all five indices (The Psychological Corporation, 1987).

5.4.5 Perceptual Functions

5.4.5.1 Gottschaldt Embedded Figures Test

Designed by Gottschaldt in 1928, the purpose of this test is to examine visual search and tracing of a simple figure embedded in a complex background pattern. The Gottschaldt Embedded Figures Test requires the subject to identify the hidden figure by marking the outline of the simple figure embedded in the more complex one. At the most difficult levels, the subject has to determine which of two intricate designs contains the simpler figure. Successful performance on this task is strongly associated with "...the ability to form a perceptual closure against some distraction ...and the ability to hold a closure against distraction" (L.L. Thurstone, 1944 in Lezak, 1995, p.414). This is an example of selective perception as the combination of a few relevant lines has to be seen against the background of a multitude of irrelevant ones. Crawford, Parker and McKinlay (1992), stated that successful performance in normals was related to the ability to form a perceptual closure against some distraction. The test has been shown to be sensitive to brain lesions in a variety of locations, and caused by various aetiologies (Lezak, 1995). According to Spreen and Strauss (1998), patients with right hemisphere lesions do more poorly than patients with left hemisphere lesions on tests of this type, and those with anterior lesions do better than those with posterior lesions if no time limit is imposed, although most patients with brain damage have some difficulty with the test, dependent on the size of the lesion. As visual synthesis is an
important aspect of perception, deficient synthesis may be revealed in tests which involve incomplete, mutilated or embedded figures.

Embedded figures have been used in many experimental studies since the time of Gottschaldt (1928), for example, the figures by Witkin, Oltman and Karp (1971) have been used in studies of field dependence and independence. Witkin’s version is designed mainly for adults, although norms for children down to the age of 10 years and reliability coefficients between .9 and .61 have been reported. The normative data show that differences in performance between males and females are negligible, and Strauss and Spreen’s (1998) recently compiled data show that healthy adults make very few errors on this test.

5.4.6 Concept Formation and Reasoning

5.4.6.1 Wisconsin Card Sorting Test (WCST)

Perhaps the most commonly observed trait of frontal lobe patients is the difficulty they have in using information (feedback) from environmental cues to regulate or change their behaviour. The Wisconsin Card Sorting Test (WCST) was originally developed to assess abstract reasoning ability and the ability to shift cognitive strategies in response to changing environmental contingencies (Berg, 1948; Grant & Berg, 1948). Revised and expanded by Heaton in 1981, the WCST can be considered a measure of “executive function”, requiring the ability to develop and maintain an appropriate problem-solving strategy across changing stimulus conditions in order to achieve a future goal. Similar to other measures of executive function, the WCST requires strategic planning, organized searching, utilizing environmental feedback to shift cognitive sets, directing behaviour toward achieving a goal, and modulating impulsive responding. However, unlike other measures of abstract reasoning, the WCST provides objective scores not only of overall success, but also for specific sources of difficulty on the task (for example, inefficient initial conceptualization, failure to maintain cognitive set, perseveration, and inefficient learning across stages of the test).
While it was developed and has been used as a measure of abstract reasoning among normal adult populations, the WCST has increasingly been employed as a clinical neuropsychological instrument. Much of its current popularity among clinicians stems from its reported specific sensitivity to brain dysfunction affecting the frontal lobes. Because of its apparent sensitivity to the effects of frontal lobe lesions, the WCST is often referred to as a measure of “frontal” or “prefrontal” functioning. However, this labelling represents an oversimplification. The frontal lobes are highly complex structures and subserve a far wider variety of cognitive functions than those assessed by the WCST alone. Conversely, while several cognitive dimensions assessed by the WCST are thought to be particularly vulnerable to neurologic conditions affecting the frontal regions of the brain, any medical or psychological disorder that disrupts executive functions, in whole or in part, can result in impaired performance on the WCST (Heaton, Chelune, Talley, Kay & Curtiss, 1993).

The WCST consists of four stimulus cards and 128 response cards that depict figures of varying forms (crosses, circles, triangles, or stars), colours (red, blue, yellow, or green) and numbers of figures (one, two, three, or four). Four stimulus cards with the following characteristics are placed before the subject in left-to-right order: one red triangle, two green stars, three yellow crosses, and four blue circles. The subject is then handed a deck of 64 response cards and instructed to match each consecutive card from the deck with one of the four stimulus cards, whichever one he/she thinks it matches. The subject is told only whether each response is right or wrong and is never told the correct sorting principle (or category). Once the subject has made a specified number of consecutive “correct” matches to the initial sorting principle (to Colour), the sorting principle is changed - to Form or Number - without warning, requiring the subject to use the examiner’s feedback to develop a new sorting strategy. The WCST proceeds in this manner through a number of shifts in set (i.e. sorting principle) among the three possible sorting categories (Colour, Form, and Number).
Normative data are now provided for individuals 6.5 through 89 years of age, and additional corrections for education are provided for individuals 20 years of age and older. WCST normative data were derived from a total group of 899 normal subjects aggregated from six distinct samples. Performance on the WCST has been examined in relation to subject's age, stress, anxiety, psychiatric disorder, and neurological status.

The WCST is regarded as a reliable measure of executive function, and overall, the evidence from studies of children, adolescents, and adults also suggests that it is a valid measure of executive function in neurologically impaired populations. Research studies examining the WCST performance of adults in such diverse clinical groups as seizure disorders, multiple sclerosis, Parkinson's disease, structural brain lesions of other aetiologies, and schizophrenia find impaired performance levels compared with those of normal adults. In addition, studies of physiological correlates of WCST-R performance and the performance of groups of focal brain-lesioned subjects suggest that the WCST is sensitive to frontal lobe dysfunction in particular. Findings from a recent study of children and adolescents with focal brain lesions also support these conclusions. However, data exist which are contradictory to the specificity of the WCST to frontal lobe lesions, and the frontal-nonfrontal differences that have been reported do not appear to be sufficiently robust to warrant using the WCST as a "frontal lobe sign" when diagnosing individuals. Yet, Kolb and Whishaw (1996) maintain that the WCST is the best available test of dorsolateral frontal cortex function.

5.5 PROCEDURE

Twenty research inpatients diagnosed according to DSM-IV criteria with Borderline Personality Disorder (BPD), twenty research inpatients diagnosed according to DSM-IV criteria with Cluster A or Cluster C personality disorders, (OTHER PD) and twenty normal volunteers (NORMAL), completed a battery of neuropsychological tests. Both the BPD subjects and the OTHER PD subjects had been referred to Tara Hospital by mental health professionals in the community and were diagnostically
re-assessed by the psychiatrist and multidisciplinary team comprising psychologists, social workers, nursing staff, and occupational therapists on Wards 4 and 5 at Tara Hospital. A psychiatric interview which included medical history, substance abuse history, past/present neurological history and review of symptoms, developmental history (including learning disabilities and hyperactivity), current and past medications, handedness, family history of medical/neurological/psychiatric illness or substance abuse, and risk factors for AIDS or documented positive serology for HIV, was conducted on the BPD and OTHER PD groups of subjects. The selected patients were subjected to a battery of neuropsychological tests towards the end of their six-week stay in hospital. The reason for this was to ensure that all the selected patients were substance-free and alcohol-free (as confirmed by urine-cannabis tests and gamma-globulin blood tests) and further, that they were free from medication which was likely to interfere with cognitive functioning.

The selected BPD subjects met the following criteria:

1. DSM-IV criteria for BPD;
2. criteria for BPD as assessed by the Millon Clinical Multiaxial Inventory-II;
3. showed clear behavioural evidence of dyscontrol: suicide attempt, self-injury, violence towards persons or property, or any combination of these;
4. had no cardiovascular, renal, hepatic or seizure disorder;
5. had no history of drug or alcohol dependence according to DSM-IV criteria within the past two months;
6. no evidence of a concurrent diagnosis of Axis I disorder.

After a statement of informed consent was obtained, the subjects from each group, namely BPD, OTHER PD and NORMAL were tested individually on the aforementioned neuropsychological tests at approximately the same time of day (i.e. early afternoon), and in respect of the BPD experimental group and OTHER PD control group, at the end of their stay in hospital (i.e. in their sixth week after admission) at which time they were medication-free or on minimal doses of psychotropic medication which was not expected to affect their cognitive functioning during the testing.
Testing followed an invariate sequence for all subjects beginning with a difficult and presumably arousing memory task which would require frontally-based organization and planning, at the same time assessing non-verbal memory: the Rey Complex Figure Test (RCF). This would be closely followed by the Digit Symbol subtest (DS), a 90-second test of concentration and attention, motor-processing speed, and visual acuity. A 3-minute delayed recall of the RCF follows the DS. The Logical Memory subtest I from the Wechsler Memory Scale Revised (LOG MEM I), an auditory verbal learning test, would then be administered, followed by the Wisconsin Card sorting Test (WCST), a more complex test of frontally-based planning and organization. After administration of the WCST, the 20-minute delayed recall of the Logical Memory subtest II (LOG MEM II) will proceed. A short break follows LOG MEM II, and thereafter the 45-minute delayed recall of the RCF would ensue. The final test of the battery would be the Gottschaldt Embedded Figures test (GEF), a test of visuospatial discrimination and filtering.

The tests were administered in the following invariate order:

1. Rey Complex Figure Test (RCF) - copy design.
2. Digit Symbol Substitution subtest of the Wechsler Adult Intelligence Scale Revised (DS).
3. Rey Complex Figure Test (RCF) - 3 minute recall.
4. Logical Memory subtest of the Wechsler Memory Scale Revised (LOG MEM I):
   (a) Paragraph Memory No. I and immediate recall
   (b) Paragraph Memory No. II and immediate recall (LOG MEM I).
6. Wechsler Memory subtest of the Wechsler Memory Scale Revised (LOG MEM II).
   (a) Paragraph Memory No.I and 20-minute delayed recall
   (b) Paragraph Memory No.II and 20-minute delayed recall (LOG MEM II).

---------- short break ----------
7. Rey Complex figure (RCF): 45-minute delayed recall.
8. Gottschaldt Embedded Figures Test (GEF).

As variations of the sequence would result in a greater interindividual variance in performance, the sequence of testing was selected in such a way that major interferences between performance required by the different tests were avoided.

5.6 EXPERIMENTAL DESIGN AND STATISTICAL ANALYSIS

Determination of the significant differences between the experimental and control groups was undertaken by multivariate analysis of variance for the determination of major vectors in the study and univariate analysis of variance. In the event of a significant F-statistic, Scheffe post hoc tests were conducted to determine significant differences in the cell means. Because of the intrinsic nature of the Rey Complex figure Test (RCF) and the Logical Memory subtests (LOG MEM I and II), they were subjected to a repeated measures univariate analysis of variance. In the next chapter, the analysis of results is reported.
CHAPTER SIX

RESULTS

6.1 INTRODUCTION

This study was conducted in order to assess whether Borderline Personality Disorder subjects (BPD) differed from Other Personality Disorder subjects (OTHER PD) and from normal subjects (NORMAL) in their performance on a battery of neuropsychological tests. It was expected that BPD subjects would perform less efficiently than both the OTHER PD subjects and the NORMAL subjects on the selected neuropsychological tests. In particular, it was anticipated that BPD subjects would experience problems with frontally-mediated functioning (planning, self-monitoring, arrangement of material, problem solving, cognitive inflexibility, impulsiveness); axial-based difficulties (poor recall); dorsolateral frontal cortex functioning (forming abstract concepts, shifting and maintaining set); and temporal lobe functioning (perceptual, motor and memory functioning).

As described in Chapter Five, data has been analysed statistically using univariate and multivariate analysis. In the case of the univariate analysis, one-way Anova was used. Where the overall F-ratio was significant, either the Dunnet or the Scheffe test (post hoc tests) was conducted to test for the differences between the cell means of the groups.
6.2 HYPOTHESES

Hypothesis I

The experimental group of subjects with Borderline Personality Disorder (BPD) will show significantly greater neuropsychological deficits in construction; orientation and attention; memory; perception; and concept-formation and reasoning, than the control group of subjects with Personality Disorders from DSM-IV Clusters A or C (OTHER PD) and the control group of NORMAL volunteer subjects.

Hypothesis II

The experimental group of subjects with Borderline Personality Disorder (BPD) and the control group of subjects with Personality Disorders from DSM-IV Clusters A or C (OTHER PD) will show significantly greater neuropsychological deficits overall when subjected to a battery of neuropsychological tests than the control group of NORMAL volunteer subjects.

6.3 INFERENTIAL STATISTICAL ANALYSIS OF DATA

The subjects were required to perform five neuropsychological tasks which formed the neuropsychological battery assessing constructional functions (visuo perceptual); orientation and attention (motor-sequencing and information-processing plus speed of processing); auditory memory (immediate and delayed); perceptual functions; and concept-formation and reasoning (abstract/conceptual).

In order to determine, on an inferential basis, the significant difference between experimental and control groups, inferential statistics were completed. The statistical measures described for each grouping will firstly concern a grouping of the data and analysis by means of a multivariate procedure for the significance of differences between the three groups. In the event of significant multivariate differences, each contributor to the variance will be analysed by means of
univariate analyses of variance with post hoc analyses for determination of significance of difference in group means.

The results will be described in four sections. The first section will focus on multivariate analysis of variance, the second section will focus on univariate analysis of variance and post hoc tests; the third section will focus on a repeated measure design and post hoc tests, and the fourth section will focus on descriptive statistical analysis.

The first section on inferential statistics will address two major groupings of the data. The first grouping will address a combined multivariate analysis of variance on the Digit Symbol subtest and the Logical Memory subtest, and the second grouping will address multivariate analysis of variance on the Gottschaldt Embedded Figures test and the Wisconsin Card Sorting test.

6.3.1 Multivariate Analysis of Variance

In order to determine the difference between groups of subjects classified as BPD, OTHER PD, and NORMAL, subjects were grouped and subjected firstly to a multivariate analysis of variance. In the event of groups significantly differing from each other they were then subsequently subjected to univariate analyses of variance.

6.3.1.1 Multivariate Analysis of Variance: The group effect of the experimental and control groups on the grouped data of the Digit Symbol subtest of the WAIS-R (DS) and the Logical Memory subtest of the WMS-R (LOG MEM)

In order to determine the differences between groups of subjects classified as BPD, OTHER PD, or NORMAL, on general measures of attention, concentration and memory, assessed by the Digit Symbol subtest of the WAIS-R, and the Logical Memory subtest of the WMS-R, they were subjected to a multivariate analysis of variance. This revealed a significant main effect due to the groups (p<0.01) (see Table 6.3.1.1A).
6.3.1.2 Multivariate Analysis of Variance: The group effect of the experimental and control groups on the grouped data of the Gottschaldt Embedded Figures test (GEF) and the Wisconsin Card Sorting test (WCST)

In order to determine differences between groups of subjects classified as BPD, OTHER PD, and NORMAL on general measures of visual discrimination and filtering, planning, problem-solving, adaptability and reasoning, assessed by the Gottschaldt Embedded Figures Test and the Wisconsin Card Sorting Test, they were subjected to a multivariate analysis of variance. This revealed a significant main effect due to the groups ($p<0.0001$) (see Table 6.3.1.2A) indicating that the tests differentiated between the three groups of subjects.

**TABLE 6.3.1.1A.**

**MULTIVARIATE ANALYSIS OF VARIANCE**

<table>
<thead>
<tr>
<th>TEST</th>
<th>VALUE</th>
<th>F-RATIO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilks' Lambda</td>
<td>.749</td>
<td>4.352</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Pillai's Trace</td>
<td>.252</td>
<td>4.106</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Hotelling's T2</td>
<td>.334</td>
<td>4.590</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>
TABLE 6.3.2A.

MULTIVARIATE ANALYSIS OF VARIANCE

The group effect of the experimental and control groups on the grouped data of the Gottschaldt Embedded Figures Test (GEF) and the Wisconsin Card Sorting Test (WCST)

<table>
<thead>
<tr>
<th>TEST</th>
<th>VALUE</th>
<th>F-RATIO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilks' Lambda</td>
<td>.486</td>
<td>3.771</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pillai's Trace</td>
<td>.541</td>
<td>3.272</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hotelling's T2</td>
<td>1.006</td>
<td>4.273</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

6.3.2 Univariate Analysis of Variance and Post Hoc Tests

Because of the significance of findings on the multivariate analyses of variance (p<0.01, see Table 6.3.1.1A; and p<0.0001, see Table 6.3.1.2A), univariate analyses of variance and post hoc tests were performed on the relevant tests for determination of significance of difference in group means.

6.3.2.1 Univariate Analysis of Variance and post hoc test: The effect of the experimental and control groups on values for The Digit Symbol subtest of the WAIS-R (DS)

An analysis of variance performed to establish the significance of differences between the experimental and control groups for values obtained from the raw data of the Digit Symbol subtest of the WAIS-R showed a significant difference due to the groups (p<0.0001) (see Table 6.3.2.1A)
### TABLE 6.3.2.1A.

UNIVARIATE ANALYSIS OF VARIANCE

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>SS</th>
<th>Df</th>
<th>MS</th>
<th>F-RATIO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digit Symbol</td>
<td>2293.2</td>
<td>2</td>
<td>1146.6</td>
<td>9.133</td>
<td>p&lt;0.0001</td>
</tr>
<tr>
<td>Error Variance</td>
<td>7156.0</td>
<td>57</td>
<td>125.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>9449.2</td>
<td>59</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In order to determine the significance of the differences in cell means for the significant main effect due to the groups (p<0.0001) (see Table 6.3.2.1A), a Scheffe post hoc test was performed. A significant difference was found between the NORMAL group and the BPD experimental group, where the BPD experimental group showed significantly more errors (Mean = 57.65) than the NORMAL control group (Mean = 68.15); (p<0.05) (see Table 6.3.2.1B). In addition, the OTHER PD control group showed significantly more errors (Mean = 53.45) than the NORMAL control group (Mean = 68.15); (p<0.01) (see Table 6.3.2.1B). There was no significant difference in cell means between the BPD and OTHER PD groups.
TABLE 6.3.2.1B.

SCHEFFE POST HOC TEST

<table>
<thead>
<tr>
<th></th>
<th>MEAN</th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD</td>
<td>57.65</td>
<td></td>
<td></td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>OTHER PD</td>
<td>53.45</td>
<td></td>
<td>p&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>68.15</td>
<td></td>
<td></td>
<td>p&lt;0.01</td>
</tr>
</tbody>
</table>

6.3.2.2 Univariate Analysis of Variance: The effect of the experimental and control groups on the values for the Logical Memory subtest of the WMS-R (LOG MEM)

An analysis of variance performed to establish the significance of differences between the experimental and control groups for values obtained from the raw data of the Logical Memory subtest of the WMS-R revealed no significant difference due to the groups (see Table 6.3.2.2A). Because no significant difference was found, no post hoc test was performed.
TABLE 6.3.2.2A.

ANALYSIS OF VARIANCE

The effect of the experimental and control groups on the values for the Logical Memory subtest of the WMS-R (LOG MEM)

<table>
<thead>
<tr>
<th>VARIANCE</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F-RATIO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Logical Memory</td>
<td>3.7</td>
<td>2</td>
<td>1.8</td>
<td>.1</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Error Variance</td>
<td>907.1</td>
<td>57</td>
<td>15.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>910.8</td>
<td>59</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6.3.2.3 Univariate Analysis of Variance and post hoc test: The effect of the experimental and control groups on values for the Gottschaldt Embedded Test (GEF)

An analysis of variance performed to establish the significance of differences between the experimental and control groups for values obtained from the raw data of the Gottschaldt Embedded Figures test showed a significant difference due to the groups (p<0.0001) (see Table 6.3.2.3A).
### TABLE 6.3.2.3A.

**ANALYSIS OF VARIANCE**

The effect of the experimental and control groups on values for the Gottschaldt Embedded Figures test (GEF)

<table>
<thead>
<tr>
<th>VARIANCE</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F-RATIO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gottschaldt</td>
<td>3580.0</td>
<td>2</td>
<td>1790.0</td>
<td>20.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Error Variance</td>
<td>4866.7</td>
<td>57</td>
<td>85.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>8446.7</td>
<td>59</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In order to determine the significance of the differences in cell means for the significant main effect due to the groups (p<0.0001) (see Table 6.3.2.3A), a Scheffe post hoc test was performed. A significant difference was found between the BPD experimental group and the NORMAL control group, where the BPD group showed significantly more errors (Mean = 17.55) (p<0.0001) (see Table 6.3.2.3B). There was also a significant difference between the NORMAL control group (Mean = 35.80) and the OTHER PD control group (Mean = 22.35); (p<0.0001; see Table 6.3.2.3B). There was no significant difference between the BPD experimental group (mean = 17.55) and the OTHER PD control group (mean = 22.35); (p>0.05; see Table 6.3.2.3B).
TABLE 6.3.2.3B.

SCHEFFE POST HOC TEST

<table>
<thead>
<tr>
<th></th>
<th>MEAN</th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD</td>
<td>17.55</td>
<td></td>
<td></td>
<td>p&lt;0.0001</td>
</tr>
<tr>
<td>OTHER PD</td>
<td>22.35</td>
<td>&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>35.80</td>
<td></td>
<td></td>
<td>p&lt;0.0001</td>
</tr>
</tbody>
</table>

6.3.2.4 Univariate Analysis of Variance and post hoc test: The effect of the experimental and control groups on values for The Wisconsin Card Sorting test (WCST)

An analysis of variance performed to establish the significance of differences between the experimental and control groups for values obtained from the raw data of the Wisconsin Card Sorting test showed a significant difference due to the groups only on “failure to maintain set” (p<0.05; see Table 6.3.2.4A). The values for “total correct”, “total error”, “total perseverative errors”, “categories complete”, and “completion of first category” were not significant (see Table 6.3.2.4A).
### TABLE 6.3.2.4A.

#### ANALYSIS OF VARIANCE

The effect of the experimental and control groups on values for the Wisconsin Card Sorting Test

<table>
<thead>
<tr>
<th>VARIANCE</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F-RATIO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Correct</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST</td>
<td>120.9</td>
<td>2</td>
<td>60.4</td>
<td>.8</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Error Variance</td>
<td>4018.7</td>
<td>57</td>
<td>70.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>4139.6</td>
<td>59</td>
<td>70.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total Errors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST</td>
<td>120.9</td>
<td>2</td>
<td>60.4</td>
<td>.8</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Error Variance</td>
<td>4018.7</td>
<td>57</td>
<td>70.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>4139.6</td>
<td>59</td>
<td>70.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total Perseverative Errors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST</td>
<td>18.6</td>
<td>2</td>
<td>9.3</td>
<td>.3</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Error Variance</td>
<td>1412.3</td>
<td>57</td>
<td>24.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>1430.9</td>
<td>59</td>
<td>24.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Categories Complete</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST</td>
<td>13.3</td>
<td>2</td>
<td>6.6</td>
<td>2.7</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Error Variance</td>
<td>135.5</td>
<td>57</td>
<td>2.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>148.8</td>
<td>59</td>
<td>2.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>1st Category</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST</td>
<td>436.6</td>
<td>2</td>
<td>218.3</td>
<td>.9</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Error Variance</td>
<td>12564.3</td>
<td>57</td>
<td>220.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>13000.9</td>
<td>59</td>
<td>220.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Failure to Maintain</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WCST</td>
<td>5.7</td>
<td>2</td>
<td>2.8</td>
<td>4.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Error Variance</td>
<td>33.9</td>
<td>57</td>
<td>.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Variance</td>
<td>39.6</td>
<td>59</td>
<td>.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
In order to determine the significance of the differences in cell means for the significant main effect due to the groups (p<0.05; see Table 6.3.2.4A), a Dunnett T3 post hoc test was performed. A significant difference was found between the NORMAL group and the BPD group, where the BPD experimental group showed significantly more difficulty in “maintaining set” (see Table 6.3.2.4B).

**TABLE 6.3.2.4B.**

**DUNNETT T3 POST HOC TEST**

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>MEAN</th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failure to Maintain Set</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BPD</td>
<td>1.05</td>
<td></td>
<td></td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>OTHER PD</td>
<td>.60</td>
<td>&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>.30</td>
<td></td>
<td></td>
<td>p&gt;0.05</td>
</tr>
</tbody>
</table>

**6.3.3 Repeated Measures Design**

A Repeated Measures Design was selected to establish the significance of differences between the experimental and control groups for the values obtained from the raw data of the Rey Complex Figure: RCF I (immediate copy), RCFII (3-minute recall), and RCF III (45-minute delayed recall). The same procedure was followed for the Logical Memory subtest: LOG MEM I (immediate recall); and LOG MEM II (20-minute delayed recall).
6.3.3.1 Multivariate Analysis of Variance: The group effect of the experimental and control groups on the Rey Complex Figure I, II and III (RCF I, II and III)

In order to determine differences between groups of subjects classified as BPD, OTHER PD, and NORMAL on general measures of constructional functioning, visuographic copying and visuographic memory, as well as perceptual organization and visual memory, assessed by the Rey Complex Figure Test, they were subjected to a repeated measures multivariate analysis of variance. This revealed a significant main effect due to the groups (p<0.05) (see Table 6.3.3.1A).

**TABLE 6.3.3.1A.**

MULTIVARIATE ANALYSIS OF VARIANCE

<table>
<thead>
<tr>
<th>TEST</th>
<th>VALUE</th>
<th>F-RATIO</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilks' Lambda</td>
<td>.828</td>
<td>2.767</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Pillai's Trace</td>
<td>.175</td>
<td>2.735</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Hotelling's T2</td>
<td>.203</td>
<td>2.796</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

6.3.3.2 Repeated Measures Analysis of Variance and post hoc tests: The effect of the experimental and control groups on values for the Rey Complex Figure Test (RCF)

Because of the significant findings on the multivariate analysis of variance (p<0.05; see Table 6.3.3.1A), a univariate analyses of variance was performed. This showed a significant difference between the experimental and control groups for values obtained from the raw data of the Rey Complex Figure Test (p<0.0001; see Table 6.3.3.2A).
TABLE 6.3.3.2A.

REPEATED MEASURES ANALYSIS OF VARIANCE

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F-RATIO</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCF I</td>
<td>7194.7</td>
<td>2</td>
<td>3597.3</td>
<td>383.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Variance</td>
<td>178.6</td>
<td>4</td>
<td>44.6</td>
<td>4.7</td>
<td></td>
</tr>
<tr>
<td>Variance</td>
<td>1068.4</td>
<td>114</td>
<td>9.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In order to determine the significance of the differences in cell means for the significant main effect due to the groups (p<0.05; see Table 6.3.3.1A), a Dunnett T3 post hoc test was performed. (Because the variances were unequal, the Dunnett T3 was used instead of the Scheffe.) Although no significant differences were found between the three groups on the immediate copy recall (RCF I; see Table 6.3.3.2B(i)), a significant difference was found on the 3-minute recall (RCF II) between the NORMAL control group (Mean = 25.40) and the BPD experimental group (Mean = 19.88) where the BPD group showed significantly more errors than the NORMAL group (p<0.01; see Table 6.3.3.2B(ii)). There was also a significant difference between the NORMAL control group (Mean = 25.40) and the control group of OTHER PD (Mean = 19.84) where the OTHER PD control group showed significantly more errors than the NORMAL control group on the 3-minute recall (p<0.01; see Table 6.3.3.2B(ii)). There were no significant differences between the BPD experimental group and the OTHER PD control group on the 3-minute recall (see Table 6.3.3.2B(ii)).
### TABLE 6.3.3.2B(i)

**DUNNETT T3 POST HOC TEST**

Significance of differences in cell means of the experimental on the control groups for the dependent variable Rey Complex Figure Immediate copy (RCF I)

<table>
<thead>
<tr>
<th></th>
<th>MEAN</th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD</td>
<td>34.50</td>
<td></td>
<td></td>
<td>p&gt;0.05</td>
</tr>
<tr>
<td>OTHER PD</td>
<td>34.75</td>
<td></td>
<td>p&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>35.80</td>
<td></td>
<td></td>
<td>p&gt;0.05</td>
</tr>
</tbody>
</table>

### TABLE 6.3.3.2B(ii)

**DUNNETT T3 POST HOC TEST**

Significance of differences in cell means of the experimental on the control groups for the dependent variable Rey Complex Figure 3-minute recall — (RCF II)

<table>
<thead>
<tr>
<th></th>
<th>MEAN</th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD</td>
<td>19.88</td>
<td></td>
<td></td>
<td>P&lt;0.01</td>
</tr>
<tr>
<td>OTHER PD</td>
<td>19.84</td>
<td></td>
<td>p&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>25.40</td>
<td></td>
<td></td>
<td>P&lt;0.01</td>
</tr>
</tbody>
</table>

In order to determine the significance of the differences in cell means for the significant main effect due to the groups (p<0.05) (see Table 6.3.3.1A), a Dunnett T3 post hoc test was performed on the Rey Complex Figure 45-minute delayed recall (RCF III). A significant difference was found between the BPD experimental group (Mean = 19.98) and the NORMAL control group (Mean = 25.28), where the BPD group showed significantly more errors than the NORMAL control group (p<0.05; see Table 6.3.3.2B(iii)). In addition, a significant difference was found between the OTHER PD control group (Mean = 19.27) and the NORMAL control group (Mean = 25.28), where
the OTHER PD group showed significantly more errors than the NORMAL group (p<0.01; see Table 6.3.3.2B(iii)). There were no significant differences between the BPD experimental group and the OTHER PD control group.

**TABLE 6.3.3.2B(iii)**

**DUNNETT T3 POST HOC TEST**

<table>
<thead>
<tr>
<th></th>
<th>MEAN</th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD</td>
<td>19.98</td>
<td></td>
<td></td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>OTHER PD</td>
<td>19.27</td>
<td>p&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>25.28</td>
<td></td>
<td></td>
<td>p&lt;0.01</td>
</tr>
</tbody>
</table>

6.3.3.3 Multivariate Analysis of Variance: The group effect of the experimental and control groups on the Logical Memory subtest (LOG MEM I and II)

In order to determine differences between groups of subjects classified as BPD, OTHER PD, and NORMAL on general measures of auditory-verbal learning and memory for complex novel verbal information, assessed by the Logical Memory subtest of the WMS-R, they were subjected to a multivariate analysis of variance. There was no significant main effect due to the groups (p>0.05; see Table 6.3.3.3A).
### TABLE 6.3.3.3A

MULTIVARIATE ANALYSIS OF VARIANCE

<table>
<thead>
<tr>
<th>TEST</th>
<th>VALUE</th>
<th>F-RATIO</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilks’ Lambda</td>
<td>.996</td>
<td>.116</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Pillai’s Trace</td>
<td>.004</td>
<td>.116</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Hotellings T2</td>
<td>.004</td>
<td>.116</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

6.3.3.4 Repeated Measures Analysis of Variance and post hoc tests: The effect of the experimental and control groups on values for the Logical Memory subtest (LOG MEM)

Although the multivariate analysis of variance conducted on the Logical Memory subtest revealed no significant differences overall between the groups, a repeated measures analysis of variance revealed significant differences between the experimental and control groups for values obtained from the raw data of the Logical Memory I and II (p<0.0001; see Tables 6.3.3.4A and 6.3.3.4B).
### TABLE 6.3.3.4A

**REPEATED MEASURES ANALYSIS OF VARIANCE**

The effect of the experimental and control groups on values for the Logical Memory subtest I (LOG MEM I)

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F-RATIO</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOG MEM I</td>
<td>261.075</td>
<td>1</td>
<td>261.075</td>
<td>32.809</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Total Variance</td>
<td>1.850</td>
<td>2</td>
<td>.925</td>
<td>.116</td>
<td></td>
</tr>
<tr>
<td>Error Variance</td>
<td>453.575</td>
<td>57</td>
<td>7.957</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### TABLE 6.3.3.4B

**REPEATED MEASURES ANALYSIS OF VARIANCE**

The effect of the experimental and control groups on values for the Logical Memory subtest No.II (LOG MEM II)

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F-RATIO</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOG MEM II</td>
<td>125906.408</td>
<td>1</td>
<td>125906.408</td>
<td>1453.656</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Error variance</td>
<td>4936.975</td>
<td>57</td>
<td>86.614</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total variance</td>
<td>1101.1</td>
<td>2</td>
<td>550.558</td>
<td>6.356</td>
<td></td>
</tr>
</tbody>
</table>
In order to determine the significance of the differences in cell means for the
significant main effect due to the groups (p<0.0001; see Tables 6.3.3.4A and
6.3.3.4B), a Dunnett T3 post hoc test was performed on Logical Memory I and II.
A significant difference was found on both the Logical Memory I (immediate
recall) and on the Logical Memory II (20-minute delayed recall) between the BPD
experimental group and the NORMAL control group, where the BPD group
showed significantly more errors (p<0.05; see Tables 6.3.3.4A(i) and 6.3.3.4B(i)).
A significant difference was found on both the Logical Memory I (immediate
recall) and the Logical Memory II (20-minute delayed recall) between the OTHER
PD group and the NORMAL group where the OTHER PD group showed
significantly more errors (p<0.01 and p<0.05 respectively (see Tables 6.3.3.2A(i)
and 6.3.3.4B(i)). There were no significant differences between the BPD
experimental group and the OTHER PD control group on both Logical Memory I
and II.

**TABLE 6.3.3.4A(i)**

DUNNETT T3 POST HOC TEST

<table>
<thead>
<tr>
<th></th>
<th>MEAN</th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD</td>
<td>32.60</td>
<td></td>
<td></td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>OTHER PD</td>
<td>30.90</td>
<td>p&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>38.10</td>
<td></td>
<td></td>
<td>p&lt;0.01</td>
</tr>
</tbody>
</table>
### TABLE 6.3.3B(i)

DUNNETT T3 POST HOC TEST

<table>
<thead>
<tr>
<th></th>
<th>BPD</th>
<th>OTHER PD</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MEAN</strong></td>
<td>30.00</td>
<td>p&lt;0.05</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>BPD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OTHER PD</td>
<td>27.80</td>
<td>p&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>NORMAL</td>
<td>34.95</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Significance of differences in cell means of the experimental on the control groups for the dependent variable Logical Memory subtest II (LOG MEM II)

6.4 DESCRIPTIVE STATISTICAL ANALYSIS

In order to provide a qualitative analysis of the information, a descriptive statistical analysis was conducted.

6.4.1 Average and variability distribution for the Digit Symbol subtest for the three groups: BPD, NORMAL and OTHER PD

A descriptive analysis of the score distribution for the Digit Symbol subtest for the BPD experimental group and OTHER PD and NORMAL control groups showed the three groups to have similar distributions in terms of high and low scores and standard deviations. The means seemed to favour the NORMAL group to show the highest amount of digit symbols produced. There was no difference between the BPD and OTHER PD group of subjects (see Figure 6.4.1A).
6.4.2 Average and variability distribution for the Logical Memory subtest for the three groups: BPD, NORMAL and OTHER PD

A descriptive analysis of the score distribution for the Logical Memory subtest for the BPD experimental group and OTHER PD and NORMAL control groups showed the BPD group and OTHER PD group to have similar distributions in terms of high and low scores, standard deviations and means. On the other hand, the NORMAL control group showed a greater distribution of subjects who performed above the mean on the Logical Memory subtest (see Figure 6.4.2A).
A descriptive analysis of the score distribution for the Logical Memory subtest for immediate recall and for 20-minute recall revealed the following differences amongst the groups: the BPD experimental group and the OTHER PD group showed similar distributions in terms of high and low scores and standard deviations in immediate recall and 20-minute recall but the 20-minute recall was lower in both groups. In addition, the OTHER PD group performed more poorly overall whereas the NORMAL group showed the highest immediate recall with a
marginal variability in overall scores and also the highest amount of logical memory scores on 20-minute recall (see Figure 6.4.2.1A).

![Logical Memory I and II](image)

Figure 6.4.2.1A: Average and variability distribution for the Logical Memory subtest (LOG MEM I and II) for the three groups: BPD, NORMAL and OTHER PD

6.4.3 Average and variability distribution for the Rey Complex Figure (RCF I, II, III) for the three groups: BPD, NORMAL and OTHER PD

A descriptive analysis of the score distribution for the comparison between Rey Complex Figure Test copy recall (RCF I), 3-minute recall (RCF II), and 45-minute delayed recall (RCF III) showed that all three groups were able to accurately copy the figure. The NORMAL group in its entirely managed to copy the RCF with no mistakes whereas there were minimal mistakes amongst a few subjects in both the BPD and OTHER PD groups. On both the 3-minute recall and 45-minute recall the NORMAL group once again surpassed the BPD and OTHER
PD groups who showed greater variability in terms of high and low scores and standard deviations (see Figure 6.4.3A).

![Rey Complex Figure](image)

**Figure 6.4.3A:** Average and variability distribution for the Rey Complex Figure (RCF I, II and III) for the three groups: BPD, NORMAL and OTHER PD

### 6.4.4 Average and variability distribution for the Gottschaldt Embedded Figures Test for the three groups: BPD, NORMAL and OTHER PD

A descriptive analysis of the score distribution for the Gottschaldt Embedded Test for the experimental and control groups showed the greatest variability in scores between the three groups. The BPD group performed most poorly with less variability in high and low scores than the OTHER PD group and lower means and standard deviations. The NORMAL control group surpassed both the BPD...
and OTHER PD groups in terms of means, standard deviations and high and low scores (see Figure 6.4.4A).

![Gottschaldt Embedded Figures](image)

**Figure 6.4.4.A:** Average and variability distribution for the Gottschaldt Embedded Figures Test for the three groups: BPD, NORMAL and OTHER PD

### 6.4.5 Average and variability distribution for the Wisconsin Card Sorting Test for the three groups: BPD, NORMAL and OTHER PD

The Wisconsin Card Sorting Test was evaluated according to various subcategories as follows: Total Correct (Figure 6.4.5A); Total Error (Figure 6.4.5B); Total Perseverative Errors (Figure 6.4.5C); Categories Completed (Figure 6.4.5D); First Category (Figure 6.4.5E); and Failure to Maintain (Figure 6.4.5F).
In the first five subcategories there seems to be no major differences between the groups (see Figures 6.4.5A, 6.4.5B, 6.4.5C, 6.4.5D, and 6.4.5E). However, in the subcategory of Failure to Maintain the BPD group performed significantly more poorly than both the NORMAL control group and the OTHER PD control group. The BPD experimental group showed the greatest variability in terms of high and low scores, means and standard deviations (see Figure 6.4.5F).
Wisconsin Card Sorting: Total correct

Figure 6.4.5.A: Average and variability distribution for the three groups on the Wisconsin Card Sorting test: Total correct

Wisconsin Card Sorting: Total error

Figure 6.4.5.B: Average and variability distribution for the three groups on the Wisconsin Card Sorting test: Total error
Figure 6.4.5.C: Average and variability distribution for the three groups on the Wisconsin Card Sorting Test: Total Perseverative Errors.

Figure 6.4.5.D: Average and variability distribution for the three groups on the Wisconsin Card Sorting Test: Categories Completed.
Figure 6.4.5E: Average and variability distribution for the three groups on the Wisconsin Card Sorting Test: First category

Figure 6.4.5F: Average and variability distribution for the three groups on the Wisconsin Card Sorting Test: Failure to maintain
CHAPTER SEVEN

DISCUSSION

7.1 INTRODUCTION

BPD has become a reasonable working description, assessed with acceptable objective criteria, allowing systematic investigation of its contents and of its limits (Tarnopolosky & Berelowitz, 1987). Although clinical evidence for possible organic impairment in BPD has been anecdotal and non-specific, it is possible that many of the manifestations of BPD, including the neuropsychological findings, derive from a fundamental organic impairment.

The present study aimed at drawing a neuropsychological profile of subjects diagnosed with BPD. For this purpose, a sample of inpatient BPD subjects was compared with a control group of inpatient OTHER PD subjects, and with a control group of NORMAL volunteers. The three groups were assessed by means of a battery of neuropsychological tests specifically selected to detect the presence and nature of neuropsychological deficits. Data gathered by the research and statistical analysis of this information are reported in Chapter 6.

7.2 HYPOTHESES

Hypothesis 1

The experimental group of subjects with Borderline Personality Disorder (BPD) will show significantly greater neuropsychological deficits in construction; orientation and attention; memory; perception; and concept-formation and reasoning, than the control group of subjects with Personality Disorders from
Hypothesis II

The experimental group of subjects with Borderline Personality Disorder (BPD) and the control group of subjects with Personality Disorders from DSM-IV Clusters A or C (OTHER PD) will show significantly greater neuropsychological deficits overall when subjected to a battery of neuropsychological tests than the control group of NORMAL volunteer subjects.

7.3 DIFFERENCES IN NEUROPSYCHOLOGICAL FUNCTIONING BETWEEN THE BPD GROUP, OTHER PD GROUP AND NORMAL GROUP

Five areas of neuropsychological functioning were assessed: constructional functioning; orientation and attention; auditory verbal learning and memory; perceptual functioning; and concept-formation and reasoning.

Firstly, the hypothesis for significant differences between BPD and NORMAL subjects was confirmed. However, the hypothesis for significant differences between BPD and OTHER PD was not significant. The reason for this would seem to be that both the BPD and OTHER PD subjects represent a neuropsychologically impaired group which is at risk for the development of a personality disorder. The specific early childhood experiences will determine the type of personality disorder which manifests in early adolescence/adulthood.

7.3.1 Constructional Functioning

Firstly, there appears to be dysfunction in complex visual and constructional memory in the area of visuospatial discrimination and filtering. This was observed
on the Digit Symbol subtest of the WAIS-R, Rey Complex Figure 3-minute and 45-minute delayed recall, and on the Gottschaldt Embedded Figures Test.

The observed memory deficits seem to be partly a factor of the complexity of the material. The performance of the BPD subjects was impaired on a complex memory task (RCF: 3-minute and 45-minute delayed recall) and they also experienced significant difficulty on a test requiring visual filtering and discrimination (in the form of filtering out extraneous stimuli and selecting relevant visual details from a complex field).

Impaired performance on the Gottschaldt Embedded Figures Test (GEF) and the Rey Complex Figure (RCF) may be attributable to the tests' memory components, but impaired performance on the Digit Symbol subtest of the WAIS-R (DS) suggests a problem in speed of motor-processing, information-processing capacity and speed of non-verbal learning.

The apparent difficulties with visual discrimination and filtering in BPD tend to mirror the reported difficulties these patients have in observing and recalling details of a complex figure or story and may suggest an hysterical cognitive style, consistent with clinical descriptions of the BPD cognitive style and with the classification of BPD in the dramatic cluster of personality disorders.

On the contrary, the OTHER PD group also had difficulties with visual discrimination and filtering and one may question the reason for this. In the BPD group this could be attributed to a processing style characterised by "chaos" and in the OTHER PD group this may be due to "slowness" of information processing. According to Muller (1992), difficulties in visual discrimination and filtering may reflect a fundamental neurological disturbance in information processing. BPD patients tend to take the cognitive path of least resistance, resorting to simple categorization that allows only 'either/or', 'black/white' decisions when faced with perceptual confusion or impairment.
7.3.2 Orientation and Attention

Secondly, there appears to be dysfunction in attention and processing speed as observed on the Digit Symbol subtest of the WAIS-R. According to Kroll (1988, p.71) the general cognitive style of the BPD patient shows “a tendency towards global perceptions with a loss of attention to detail, distortion of the meaning of an event, patterns of confusion and spotty amnesias”.

In clinical practice it is observed that BPD patients exhibit types of attentional difficulties: they tend towards under-inclusion and miss details and facts; or they focus exclusively on one small aspect of a situation and miss the gestalt or context. In this research the BPD subjects tended to miss the gestalt, and this was seen particularly on the Gottschaldt Embedded Figures Test (GEF).

7.3.3 Memory

Thirdly, there appears to be dysfunction in both visual and auditory memory as observed in performance on the Rey Complex Figure Test (RCF) and the Logical Memory subtest (LOG MEM).

With regard to dysfunction in visual memory, the BPD group displayed impairment on both the 3-minute and 45-minute free recall, but not on the copy. As no indication is given that the subject must remember the figure as he/she will be asked to recall it later, one would assume that the BPD group performed poorly due to merely copying the figure and not encoding it into memory. This may be due to a fundamental difficulty in encoding uncued, novel and complex information, and may be also indicate a lack of planning as the RCF is also a test of frontally-based planning. Dysfunction in visual memory applied to both the BPD experimental group and the OTHER PD control group but not to the NORMAL control group.
Dysfunction in auditory memory was revealed on the 20-minute delayed recall of the Logical Memory subtest. Apparent memory deficits can result from problems with initial learning, retention, or recall of material. The BPD experimental group and the OTHER PD control group did not experience problems in initial learning as immediate recall of the stories was satisfactory. However, the 20-minute delayed recall showed impairment which is suggestive of difficulty in longer-term retention of material. In addition, 20-minute delayed recall of stories by the BPD group revealed a distortion of the facts — it was as if they learned one set of facts and regurgitated another.

7.3.4 Perceptual Functioning

Fourthly, there appears to be dysfunction in perceptual functioning as observed on the Gottschaldt Embedded Figures Test (GEF). According to L.L. Thurstone (1944, in Lezak, 1995, p.414), successful performance on this task is “strongly associated with the ability to form a perceptual closure against some distraction ... and the ability to hold a closure against distraction”. Visual synthesis is also an important aspect of perception. In this research BPD subjects were unable to hold a closure against distraction which might be attributable to problems in perceptual shifting and focussing and their reported tendency to process information impulsively. There was no evidence of deficient visual synthesis on this task. Whereas the NORMAL group performed optimally on this task, the OTHER PD group also experienced difficulties in perceptual functioning.

7.3.5 Concept-Formation and Reasoning

Finally, there appears to be dysfunction in maintaining cognitive set (that is, inability to maintain an appropriate problem-solving strategy across changing stimulus conditions in order to achieve a future goal) as observed on the Wisconsin Card Sorting Test (WCST). This problem has been expounded in the literature on BPD and may explain the impulsivity, tendency to jump from one
topic to another, and frequent incorrect perceptions of a situation that are so characteristic of the BPD profile.

### 7.3.6 Summary

Two central characteristics of BPD, namely, affective instability and impulsivity may reflect a difficulty in cognitive processing. Lane and Swartz (1987) suggest that emotional awareness is a form of cognitive processing that undergoes its own development. BPD subjects have particular difficulty verbalizing emotions and placing them in an interpersonal context. Furthermore, the dramatic behaviours which are characteristic of BPD patients may serve as the language of unintegrated cognitive-affective schemas.

These results may not be typical of all patients with BPD according to DSM-IV criteria, although this sample of BPD subjects had firmly established diagnoses. Patients with a history of ADHD, TLE, brain trauma, or encephalitis, were excluded from the study, as were patients with recent alcohol/drug dependence (in the last two months), and there were no subjects in the BPD group with a concurrent Axis I diagnosis, such as Major Depressive disorder.

### 7.4 LIMITATIONS OF THIS STUDY

It should be noted that there were limitations in this research study. The major limitations are the small sample size and the lack of matched controls. The relative small number of subjects could have skewed the results in a direction not recognized within the confines of this study. The findings would need replication in larger groups in order to give them scientific validity.

As highlighted in Chapter Four, most studies conducted to date on neuropsychological deficits in BPD have been characterized by a number of limitations. An attempt was made in this study to address these limitations by including a psychiatric control group as well as a normal volunteer control group,
by using an inpatient sample of subjects diagnosed with BPD, and by using stringent inclusion/exclusion criteria for all three groups.

However, this study is limited by the following shortcomings:

1. The small sample size (20 in each group) renders it difficult to make inferences about the general population of Borderline and other personality disordered subjects since those subjects who underwent the neuropsychological tests may not have been representative of the general BPD and OTHER PD population.

2. The absence of matched control subjects according to age, gender, culture, ethnic group and IQ renders it impossible to compare the groups equitably. In addition, no formal IQ testing was conducted prior to the neuropsychological testing, although a proviso that each subject had attained a minimum education level of Std. 8 was adhered to.

3. The “self-selection” of normal volunteers resulted in a sample group of highly educated subjects and this may have skewed the results.

4. The majority of the BPD subjects had a prior history of polysubstance and/or alcohol abuse and although they were substance and alcohol free for six weeks prior to neuropsychological testing, this factor may have influenced the results.

5. The examiner was not blind to BPD or OTHER PD diagnoses, although every attempt was made to adhere to strict, quantitative scoring procedures that minimize tester effects.

Consequently it seems safest to say that the findings represent a tentative exploration into the relationship between neuropsychological dysfunction and BPD.
7.5 SUGGESTIONS FOR FUTURE RESEARCH

Research into the biological components of BPD is on the cutting edge, so a replication with a larger sample of borderlines that includes a proportionate number of males is needed. Future research ideally would include neurometabolic studies as well, which have proven useful in isolating specific regional deficits in other forms of subtle organicity, such as attention deficit disorder with or without hyperactivity (Zametkin et al., 1990). Already, preliminary findings using positron emission tomography (PET) in BPD are consistent with the presence of frontal and perhaps parietal lobe dysfunction (Goyer & Andreason, 1991).

Whether or not brain dysfunction also qualitatively shapes behaviour cannot be addressed by the data in this study. Brain dysfunction must interact with environmental and psychodynamic factors in producing behavioural change. Determining how this interaction occurs will await prospective studies that incorporate measures of environmental and psychodynamic factors and brain dysfunction.

These preliminary results suggest several questions for future research: Are the observed visuospatial impairments due to poor psychological differentiation, an hysterical cognitive style, impulse dyscontrol or poor perceptual functioning? Will psychological testing suggest any specific anatomical regions of interest? For example, poor performance on tasks that may be particularly sensitive to impairment in both the non-dominant temporal lobe (RCF and GEF) was observed in BPD subjects in this study.

Ultimately, confirmation of specific neuropsychological abnormalities in some patients with BPD may have important implications for clinical work with these patients. It is therapeutically valuable to know that some BPD patients may have genuine impairment in filtering information and recalling complex material, in addition to their selective forgetting and defensive cognitive styles. While not
suggesting that neuropsychological deficits are the cause of BPD, deficits in visuospatial discrimination and filtering and in recalling complex material may contribute to the cognitive difficulties observed in BPD.

It is hoped that past efforts combined with future informed research will provide us with a clearer idea of what occurs within the maze of these patients' cognitive experiences.

7.6 IMPLICATIONS FOR CLINICAL TREATMENT

The results of this study encourage the use of neuropsychological testing in the assessment of persons with Borderline Personality Disorder. Such testing provides information about the nature of information processing in the brain and can lead a clinician to detect whether the dysfunction lies in arousal, encoding and storage, or planning and problem solving.

In addition, the results of this study support the findings of a strong association between BPD and the presence of neuropsychological deficits. The clinical implications of these findings suggest that clinicians should look for evidence of brain pathology in their patients with BPD. Further treatment of neurobehavioural disorders may serve to improve the subjective experience and objective behaviour of this debilitating and difficult disorder. It is hoped that current research will contribute ultimately to the development of new treatment modalities of BPD.

If psychotherapy is to be efficacious with borderline patients, we need to find pharmacological as well as psychological "holding environments" so that therapeutic work can proceed more calmly and more constructively. We also need to tailor the treatment program uniquely and individually to the patient concerned.

The relationship found between neuropsychological deficits and BPD suggests important practical considerations for current clinical care and treatment planning.
for BPD. Neuropsychological assessment should be a standard procedure in evaluating psychiatric patients, particularly BPD patients whom are inordinately difficult to treat. In treatment, focusing on the particular neuropsychological deficit(s) present, might contribute to a more comprehensive understanding by the patient of his/her particular problem(s), thus leading to greater motivation to use this information as a remedial tool rather than as a condemning label.

However, it is important to remember that borderline patients are often highly suggestible as to the causes of their psychopathology (Ogata, 1990) and therefore it is necessary to be cautious in over-interpreting neuropsychological dysfunction as the primary aetiological factor in patients whose biological and environmental histories are often pathogenic in multiple respects. Rather, BPD patients should be alerted to the fact that by taking some responsibility for the remediation of deficits, they can effectively lessen the symptomatology of BPD.

7.7. CONCLUSION

The intriguing feature of the neuropsychological deficits found in BPD in this study is that they occurred in a relatively affect-neutral situation (that is, the test battery was devoid of affect-laden evocative material). This observation may suggest that there is a fundamental cognitive disability in BPD that may reflect an underlying neurophysiological dysfunction.

Although this study found no difference between the BPD group and the OTHER PD group on a variety of indices of neuropsychological dysfunction, this seems to indicate that neuropsychological deficits are not specific to BPD but occur in other personality disorders as well.

It is probable that borderline pathology is best considered a final common outcome of many aetiological factors. There is still a great deal of research that remains to be carried out into the aetiology and development of BPD. It is hoped that the emergence of different, refined definitions of the disorder, new
assessment techniques, and improved research designs will facilitate extensive advances in our understanding, diagnosis and treatment of BPD.

7.8 SUMMARY

In summary, the BPD and OTHER PD patients presented a markedly dysfunctional profile compared to the NORMAL group, performing in the impaired range on four out of five neuropsychological measures. These results suggest that deficits in perception, cognition, complex visual memory and visuospatial ability may be a common feature in both BPD and OTHER PD patients.

It is appropriate to end with a quote by Hippocrates which confirms that even two thousand years ago there was a rudimentary recognition of the brain as being the site of cognition and emotion. We have come a long way since then and it is hoped in the near future that the understanding of specific areas of dysfunction in the brain will embellish our knowledge of mental disorders in general and BPD in particular.

Men ought to know that from the brain, and from the brain only, arise our pleasures, joys, laughter, and jests, as well as our sorrows, pains, griefs, and fears ... It is the same thing which makes us mad or delirious, inspires us with dread and fear, whether by night or day, brings sleeplessness, inopportunite mistakes, aimless anxieties, absentmindedness, and acts that are contrary to habit.

- Hippocrates
REFERENCES


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## ADDENDUM

### ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ADD</td>
<td>Attention Deficit Disorder</td>
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<tr>
<td>ADHD</td>
<td>Attention Deficit Hyperactivity Disorder</td>
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<tr>
<td>AIDS</td>
<td>Acquired Immunodeficiency Syndrome</td>
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<td>APA</td>
<td>American Psychiatric Association</td>
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<tr>
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<td>BPD</td>
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<tr>
<td>CNS</td>
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<td>CT</td>
<td>Computed Tomography</td>
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<td>DF</td>
<td>Degrees of freedom</td>
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<td>DS</td>
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<td>DSM</td>
<td>Diagnostic and Statistical Manual of Mental Disorders</td>
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<tr>
<td>ECT</td>
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<tr>
<td>EEG</td>
<td>Electroencephalograph</td>
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<td>EMD</td>
<td>Eye movement dysfunction</td>
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<tr>
<td>GEF</td>
<td>Gottschaldt Embedded figures Test</td>
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<td>HIV</td>
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<tr>
<td>ICD</td>
<td>International Classification of Diseases</td>
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<tr>
<td>IQ</td>
<td>Intelligence Quotient</td>
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<tr>
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<td>MAOI</td>
<td>Monoamine oxidase inhibitor</td>
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<td>Minimal brain dysfunction</td>
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<tr>
<td>MS</td>
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<tr>
<td>p</td>
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<td>PET</td>
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<tr>
<td>RCF</td>
<td>Rey Complex Figure</td>
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<td>SS</td>
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<tr>
<td>TLE</td>
<td>Temporal Lobe Epilepsy</td>
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<tr>
<td>WAIS-R</td>
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<tr>
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