A MOTOR PERFORMANCE DEFICIT IN

WOMEN WITH BULIMIA:

A NEUROPSYCHOLOGICAL INVESTIGATION

A thesis
submitted in partial fulfillment
of the requirements for the degree
of
Master of Arts in Psychology

by

Emma C. Brinded

University of Canterbury

February 1995

TABLE OF CONTENTS

ACKNOWLEDGEMENTS	vii
List of Tables	viii
List of Figures	ix
ABSTRACT	xi
Chapter 1 INTRODUCTION	1
1.1 Bulimia Nervosa	1
1.2 Correlates of Bulimia Nervosa	1
1.3 Some Common Methodological Problems	3
1.4 Right Hemispere Dysfunction	4
1.5 Pre-Existing Trait or Consequence	5
1.6 Research on Neuropsychological	
Functioning in Eating Disorders	6
1.7 Bulimia and Impulsivity	20
1.7.1 Definitional Difficulties	21
1.7.2 Findings on Bulimia and	
Impulsivity	22
1.8 The Present Study	27
1.8.1 Hypotheses	28

Chapter 2 METHOD		31
2.1	Subjects	31
	2.1.1 Bulimic Subjects	31
	2.1.2 Control Subjects	32
2.2	Materials and Apparatus	32
	2.2.1 Diagnostic Information	32
	2.2.2 Eating Disorders Inventory	32
	2.2.3 State Anxiety Inventory	33
	2.2.4 Beck Depression Inventory	33
	2.2.5 Barratt Impulsivity Scale	34
2.3	Neuropsychological Testing	34
	2.3.1 Pro-rated Intelligence Quotients	34
	2.3.2 Wecshler Adult Intelligence	
	Scale - Digit Span Subtest	35
	2.3.3 Trail Making Test	35
	2.3.4 Finger Tapping Test	35
	2.3.5 Reaction Time Test	36
	2.3.6 Go No Go Test	36
	2.3.7 Stroop Test	37
2.4	Procedure	38

Chapter 3	RE	ESULTS	40
	3.1	Subject Characteristics	40
	3.2	Neuropsychological Measures	44
		3.2.1 Barratt Impulsivity Scale	44
		3.2.2 Go-No-Go Test	45
		3.2.3 Stroop Test	46
		3.2.4 Trail Making Test	46
		3.2.5 Digit Span	47
		3.2.6 Finger Tapping Test	47
		3.2.7 Reaction Time Test	47
Chapter 4 DISCUSSION		SCUSSION	60
	4.1	Future Research	67
	4.2	Conclusion	67
REFERENCES		69	
APPENDICES			77
	1.1	Barratt Impulsivity Scale	77
	1.2	Beck Depression Inventory	79
	1.3	The Stroop Test	82

ACKNOWLEDGMENTS

I would like to thank the people who have helped in the preparation of this thesis, and those who have provided emotional support. Thanks go to Cindy Bulik for her encouragement and supervision, and Pat Sullivan, Leslie Livingston and the crew at the Bulimia Treatment Study. Thanks to the support team; Nishi Parkhill, Wayne Parkhill and Sandie Browne, and to John Townsend and Annette Searle. Thanks for technical help to John Barton and Jenny Fear.

A special thanks to the women who participated in the study, who freely gave their time and without whom this would not have been possible.

LIST OF TABLES				
Table 1:	Subject Characteristics	41		
Table 2:	Neuropsychological Test Results	42		
	For Bulimic and Control Subjects			
Table 3:	EDI2 Subscale Scores For Bulimic	43		
	and Control Subjects			
Table 4:	Neuropsychological Test Results	44		
	For Bulimic and Control Subjects			

LIST OF FIGURES		
Figure 1:	BIS-Total Means For Bulimic and Control	50
	Subjects Pre- and Post-Treatment.	
Figure 2:	BIS-Non Planning Means For Bulimic and	51
	Control Subjects Pre- and Post-Treatment.	
Figure 3:	BIS-Motor Means For Bulimic and Control	52
	Subjects Pre- and Post-Treatment.	
Figure 4:	BIS-Cognitive Means For Bulimic and	53
	Control Subjects Pre- and Post-Treatment.	
Figure 5:	Stroop Interference Index Means For	54
	Bulimic and Control Subjects Pre-	
	and Post-Treatment.	
Figure 6:	TMT-A Means For Bulimic and Control	55
	Subjects Pre- and Post-Treatment.	
Figure 7:	TMT-B Means For Bulimic and Control	56
	Subjects Pre- and Post-Treatment.	
Figure 8:	Digit Span Age-Scaled Score Means	57

For Bulimic and Control Subjects

Pre- and Post-Treatment.

Figure 9:	Finger Tapping Means For Bulimic	58
	and Control Subjects Pre- and	
·	Post-Treatment.	
Figure 1:	Reaction Time Means For Bulimic	59
	and Control Subjects Pre- and	
	Post-Treatment.	

ABSTRACT

Hypothesised neuropsychological causes for an observed deficit in motor performance on a computer game in women with bulimia were tested. Subjects were 19 normal weight women with bulimia nervosa participating in a cognitive-behavioural treatment trial, and 19 healthy controls matched for age and IQ in a yoked pre- and post-treatment design. Measures of global impulsivity, motor impulsivity and response inhibition failed to show bulimic women as more impulsive than controls, and failed to support motor impulsivity as a cause of the observed deficit. Results failed to support an attention deficit, slowed motor speed or a visuo-spatial-motor deficit as responsible for the observation, but reaction time was found to be significantly slowed in the women with bulimia. Differences in reaction time were primarily a function of depression and not pathognomonic of bulimia per se.

Chapter 1 INTRODUCTION

1.1 BULIMIA NERVOSA

The DSM III-R characterises bulimia nervosa as a disorder involving binge eating behaviour accompanied by the fear of not being able to stop. These binges are associated with purging in the form of self-induced vomiting, laxative use, diuretic use, excessive exercise, or food restriction. Some women with bulimia may also have a past or current history of anorexia nervosa. Vandereycken and Pierloot (1983) quote that up to 45% of anorexia nervosa patients also have bulimic symptoms. Other problems often associated with bulimia are alcohol and drug abuse, depression, suicidality, personality disorder and stealing (Bulik, 1987; Hatsukami, Mitchell, Eckert &Pyle, 1986; Vandereycken & Pierloot, 1983).

1.2 CORRELATES OF BULIMIA NERVOSA

The abnormal eating, binging, starving and purging that occurs in bulimia may produce changes in most systems in the body including dermatological, cardiovascular, gastrointestinal, endocrine, musculoskeletal, metabolic, neurochemical, cognitive and neuropsychological systems (Kaplan & Woodside, 1987). Bulimic

patients are often biologically starved even when they are not apparently underweight, because they may be maintaining their body weight under their natural set point (Garfinkel, Moldofsky & Garner, 1980).

The initiative for this investigation arose from incidental observations made during experiments involving women with bulimia (Bulik & Brinded, 1993, Bulik & Brinded, 1994). In these experiments the reinforcing value of certain drugs under conditions of food deprivation was being tested by the use of a computer game, Applepicker (Norman & Jongerius, 1985). Points were scored by positioning the cursor on numerous goal "trees" with a joystick and "picking" apples by pressing a button on top of the joystick. I observed that bulimic subjects tended to require longer practice periods before targets could be consistently, efficiently landed on, and results showed a generally lower hit rate for bulimic subjects compared to controls.

On closer observation it appeared that their difficulty was in failing to stop the movement of the cursor fast enough once it was initiated. This phenomenon could have had a number of neuropsychological causes. It may have been due to slowed motor speed, slowed reaction time, an attention deficit, a visuo-spatial deficit,

interference due to anxiety or a deficit of motor impulse control.

The literature addressing neuropsychological functioning in eating disorders is sparse, and in bulimia alone, even more sparse.

1.3. SOME COMMON METHODOLOGICAL PROBLEMS

There are a number of problems that are common in this literature. The delineation of the eating disorders is still progressing, and as such diagnostic criteria have tended to vary among researchers before DSM, and have undergone changes with each revision of DSM. This is problematic in that some studies have combined subgroups of eating disorders (i.e. restrictor anorexics with bulimic anorexics) which have different characteristics (Garfinkel et al., 1980), and this has the potential to obscure group differences.

Another difficulty lies in the fact that varying methods have been used across the studies to define impairment on neuropsychological tests. Some use percentile cutoffs from established norms and some use idiosyncratically defined deviations from the mean of control group performance, with some control groups being various psychiatric patients and some being healthy individuals. These two problems make direct comparison of findings in the literature difficult.

All of the studies which have used eating disordered subjects have

been run from clinic samples and as such are biased to that severe subgroup of eating disordered people who present for treatment.

Beglin & Fairburn (1992) suggest that only a small percentage of eating disordered patients ever reach treatment, thus the skew in research samples constrains the generalisability of findings.

Difficulties arise for research in this area as well, from the imprecise and indirect nature of neuropsychological testing, where tests tap many different functions simultaneously and some tests are used to test more than one function. For example the Trail Making Test is a test of attention-concentration-visual-spatial-motor function, and it is used as a test of attention or a test of visuomotor tracking or a test of dual conceptual tracking. Imprecision like this makes attribution of effect difficult.

1.4 RIGHT HEMISPHERE DYSFUNCTION

The biggest contention in this literature has been over the possibility of a right hemisphere dysfunction involvement in eating disorders. Conventionally the right hemisphere has been determined to mediate non-verbal and spatial processing and the left hemisphere verbal processing, however this is an oversimplification. Lateralisation of verbal processing is more variable and is also related to handedness

(Lezak, 1983), a variable which only the Jones, Brouwers & Mirsky (1991) study mentions (see below). A right hemisphere dysfunction involving spatial functioning is intuitively appealing in its possible explanatory value for the body image distortion which occurs in eating disorders. Eating disordered patients commonly make distorted judgments of their body size (Thompson & Spana, 1991).

1.5 PRE-EXISTING TRAIT OR CONSEQUENCE

Another issue is the degree to which any neuropsychological impairments reflect a pre-existing trait entity which may predispose to development of an eating disorder or may be a consequence of abnormal eating patterns involved in the disease. As such, studies which test pre- and post-treatment functioning or use weight recovered subjects in addition to acute subjects will have superior explanatory value. However, the possibility of metabolic/ neuropsychological "scarring" makes absolute causal attributions impossible to make. Only three of the studies reported below have this pre and post treatment design advantage; Jones et al., (1991) Szmukler, Andrews, Kingston, Chen, Srargatt & Stanley, (1992) and Hamsher, Halmi & Benton, (1981).

1.6 RESEARCH ON NEUROPSYCHOLOGICAL FUNCTIONING IN EATING DISORDERS

The following studies represent the main research on neuropsychological functioning in eating disordered subjects.

Fox (1981) conducted one of the early studies to discover whether neuropsychological deficits exist in anorexia nervosa. Her sample consisted of 14 females and 1 male anorexic admissions to an evaluation unit, and a psychiatric control group of 8 females and 7 males. Anorexic subjects met the specific criteria outlined by Halmi, Goldberg, Eckert, Casper & Davis, (1977); (1) onset between 10 and 30 years; (2) Loss of 20% of original body weight constituting a weight that is at least 15% below normal for age and height; (3) refusal to maintain body weight within normal limits; (4) disturbance of body image with inability to perceive body weight accurately; (5)intense fear of becoming obese; (6) no medical illness to account for weight loss; (7) amenorrhea; (8) no other research psychiatric diagnosis. Foxs' failure to match the controls for sex is problematic in that there are sex differences in cognitive functioning which could confound comparisons. Females perform better on symbol substitution and less well on visuospatial tasks than males (Lezak, 1993).

All subjects were given a battery of tests on admission which

consisted of the Wecshler Adult Intelligence Scale-Revised (WAIS-R) or Wecshler Intelligence Scale for Children-Revised (WISC-R), Mattis Organic Mental Screening Examination (MOMSE), which has considerable overlap with the WAIS-R, the Trail Making Test (TMT) and the Benton Visual Retention Test (BVRT). Eleven of the anorexic sample were also given the Wide Range Achievement Test (WRAT), but this was without control comparison.

She concluded that the anorexic group showed impairment of concentration and attention, as evidenced by impaired performance on the TMT as defined by norms. Although some subtests in the WAIS-R are traditionally tests of attention (digit-span, arithmetic), Fox does not report specific results of these subtests. The performance of the anorexic group was not statistically different from the psychiatric control group and Fox interprets the attention effect to be a correlate of having a psychiatric disorder, and not specific to eating disorders. However, she fails to discuss the significance of the anorexics' "abnormally high" (p289) WAIS-R digit-symbol performance compared to overall Performance scores by control comparison. Sustained attention and concentration are necessary to achieve this, which contradicts her main finding. This control comparison result is in the direction that one would expect given the sex ratios and gender

differences in performance of the two groups and as such may be confounded. Fox also fails to report on or control for handedness, which is problematic if one is to make inferences about relative functioning of hemispheres using comparisons of verbal and other functions.

The second finding she discusses is that of significantly impaired Wide Range Achievement Test- Arithmetic performance in comparison with reading and spelling performance. She links this with accompanied impaired figure copying presumably from the Construction scale of the MOMSE although she neither states this or presents results. From this she poses the possibility of impaired visual-spatial synthesis underlying arithmetic impairment. Unfortunately she only states that deficient arithmetic performance was "frequently accompanied by impaired ability to copy complex geometric designs" (p289) and presents no results linking the arithmetic and figure drawing performance.

This possibility of the visuo-spatial or right hemisphere weakness in anorexia nervosa is unsupported by the failure of Touyz, Beumont and Johnstone (1986) to replicate Fox's findings with a larger sample.

They repeated the same tests with a group of 35 DSM-III defined anorexic and 15 bulimic subjects. They compared subjects' performance

with norms rather than a control population. They found no significant difference between anorexic and bulimic subjects on any measure. In contradiction to Fox, they found low incidence of impairment on the TMT (0% for AN compared to Foxs' 73% for AN), and also on the BVRT (10% for AN compared to 47% for Fox). Touyz et. al., (1986) also failed to find exceptional performance on the Digit Symbol Subtest of the WAIS-R, and they found no significant deviations of full scale WAIS-R IQ scores from theoretical norms. Unfortunately they do not report similar comparisons for individual subscales. They also failed to find the arithmetic weakness reported by Fox, concluding that there was no sign of impaired neuropsychological or intellectual functioning in the anorexia nervosa subjects.

Hamsher et al., (1981) conducted a study to test the hypothesis that a subtle brain dysfunction exists in anorexia nervosa and that it predicts poor treatment outcome. Their subjects were 20 consecutive female admissions to a treatment unit who fitted the Halmi et al. (1977) criteria for anorexia nervosa. Subjects were given a neuropsychological battery at admission, again just before discharge at normal weight and a progress assessment was made one year after discharge. The battery consisted of 15 tests including: 6 subtests from the WAIS-R (Information, Comprehension, Arithmetic, Digit Span, Block Design

and Digit Symbol), the Shipley-Hartford Scale for verbal skills, a serial digit learning test for short term memory, the Benton Visual Retention Test for short term visual memory, the Judgement of Line Orientation Test for spatial orientation, the Test of Facial Recognition for visuoperceptive ability, the Test of Three Dimensional Constructional Praxis for constructional ability, the Controlled Oral Word Association Subtest of the Multilingual Aphasia Examination for verbal ideational fluency and two reaction time tests. They found 14 of the 20 subjects (70%) had impairment on at least one of the measures and 35% of subjects had impairment on two or more measures. Twelve subjects (60%) had impairment at post-treatment assessment on at least one measure. At follow-up one year later, there was a significant association between these measures and treatment outcome. Eightyfive percent of subjects with one or less impairment showed maintenance of weight, and 71% of the subjects with two or more impairments did not maintain their weight. Reaction time was one of the measures that showed improvement after treatment and they found reaction time at post treatment to be the only variable to be reliably predictive of outcome at follow up.

Overall they found no pattern to the impairments and concluded similarly to Fox that the anorexic subjects had an impairment of attention, which was non specific and similar to attention impairment in other psychiatric disorders. In contradiction to Fox, they conclude that there is no specific impairment, and as such no right hemisphere impairment in anorexia nervosa. However the "possibility of constructional dyspraxia"(p289) that Fox observed does seem to be supported by the test of Three Dimensional Constructional Praxis performed by Hamsher et al. even though they do not discuss this. Six out of twenty of Hamshers' subjects performed below the fifth percentile on norms before treatment and 3/20 performed below it after treatment.

Maxwell and Townes (1984) administered the WAIS-R, Weschler Memory Scale, the Wide Range Achievement Test, and the Halstead-Reitan Neuropsychological Test Battery to three atypical anorexic inpatient subjects, by Feighner's criteria, one of whom was at normal weight. These criteria do not distinguish bulimia, and use bulimic symptoms as a criteria for diagnosis of anorexia. It is likely that the normal weight subject was bulimic, but this is not specified. The remaining two subjects were deemed atypical due to onset after 25 years. The ages of these subjects were 19, 30, and 69 years. They compared performance with 24 psychiatric control subjects who were matched for age, sex and IQ, and found that the anorexic sample performed significantly better on verbal than performance sections of

the WAIS-R, and better than controls on 10/11 verbal measures and significantly worse on 9/10 measures of spatial reasoning. Maxwell et al. suggest that these findings support the notion of a right hemisphere dysfunction in eating disorders. However the sample size is so small in this study and sample selection problematic that no meaningful generalisations about either anorexia or bulimia can be made.

McKay, Allen and Clawson (1986) used the Luria Nebraska Neuropsychological Battery (LNNB) to assess the functioning of 28 women and 2 men with bulimia as diagnosed by DSM-III criteria, of which 10 also met RDC criteria for anorexia nervosa, and a healthy control group. The LNNB consists of 269 items which group into 14 function areas; motor, rhythm, tactile, visual, receptive language, expressive language, writing, reading, arithmetic, memory, general intelligence, a pathognomic brain-damage sensitive scale and a left hemisphere and a right hemisphere scale. McKay et al. (1986) found that the bulimic sample performed significantly worse than the control group on the motor performance scale, mostly due to slow performance on the Speed of Drawing Scale. On the Localisation Scales, the bulimic sample scored significantly differently on the Right Frontal Localisation Scale, with drawing speed again being the main contributor. McKay et al. (1986) conclude that there is in fact a right

hemispheric dysfunction involved in eating disorders, and locate it even more specifically in the right frontal cortex which mediates speed of initiation and execution of action. However, the number of subjects to the number of tests of significance leaves unacceptable possibility of Type I error (Moses & Maurish, 1988) such that findings may be due to chance alone.

Jones et al. (1991) conducted a wide-ranging and well designed neuropsychological study which lends support for attentional difficulties but not for right hemisphere dysfunction in women with eating disorders. They had a large sample of 30 underweight anorexics, 38 normal weight bulimics, 20 long term weight restored anorexics and 39 normal controls. This design enabled the separation of acute effects of emaciation from other possible neuropsychological effects of eating disorder. The patient groups were defined by DSM III criteria, and were inpatients and outpatients. Subjects were matched for age, education and handedness. Each subject was given a battery of 30 neuropsychological tests which, via principle component analyses, were amalgamated to five factors; vigilance and focusing/execution (attention factors), a verbal factor, a mixed memory/ comprehension factor, and a visuospatial factor. As the variables for analysis, they chose the fifteen variables with the highest loadings for the five factors.

They then transformed all scores for each subject into z-scores and averaged the three z-scores for each of the five factors into a single score which was analysed across the four groups. This statistical procedure circumvents problems with increased Type I errors, but it also leads to a loss of specific information and possible test differences within areas. They found that underweight anorexics performed significantly worse than controls in four areas; focusing/execution, verbal, memory, and visuospatial. Normal weight bulimics performed worse than controls in focusing/ execution tasks as did the underweight anorexics, but not the long term weight restored anorexics. This suggests that an attentional deficit exists and is related to acute disease effects. There were no group differences in vigilance. The underweight anorexics scored lower than controls in the visuo-spatial domain, but this was also the case in the verbal and memory domains, which mitigates against a specific right hemisphere dysfunction. Jones et al. (1991) also highlight that the level of impairment in the eating disordered groups was small and reflected only subtle neuropsychological changes. A drawback for these findings resulted from a MANCOVA for anxiety measures which revealed that removal of the covariance due to anxiety would remove the statistical significance of group differences. Consequently this study must be interpreted with caution.

De Witt and George (1985) examined memory and learning

performance in a well controlled study, but with a small sample size. Sixteen adolescents with anorexia nervosa by DSM-III criteria were compared with a chronically ill (diabetic) group of 16, a depressed group (by DSM-III) of 16, and a healthy control group of 16. They were matched on age, education, and WAIS-R Information, and tested at admission. Each subject was administered the Symbol Digit Leaning Test for associative learning, the Visual Reproductions Subscale from the Weschler Memory Scale for short and long term figural memory, the Information, Digit Span and Digit Symbol subtests from the WAIS-R, for premorbid IQ, attention/ immediate memory and motor persistence/ response speed/sustained concentration/visuomotor coordination respectively, and the Trail Making Test examine subtle dysfunction. The anorexic group performed significantly worse than all of the other groups on the Symbol Digit Learning Test, implying a learning impairment. Scores on this test correlated significantly with duration of illness, but not with the percentage the subject was underweight, inferring that the longer a subject was underweight, the more her associative learning would be impaired. There were no significant differences found between groups on the measures of figural memory, attention, immediate memory, or the motor measures. This finding is at odds with that of Hamsher et al.(1981) who did find impaired concentration/attention.

In an attempt to clarify this inconsistency, Szmukler et al. (1992) conducted a further study with the advantage of a pre- and posttreatment design. They administered attentional-perceptual-motor tests; the Trail Making Test and the Digit Symbol Subtest of the WAIS-R, learning tests; The Rey Auditory Verbal Learning Test (RAVLT) and the Serial Digit Learning Test (SDT), and visuospatial construction and problem-solving tests; the Block Design subtest of the WAIS-R and the Austin Maze Test to 21 inpatient anorexics by DSM III criteria and a healthy control group. Four of the anorexic group also had "bulimic symptoms". They found the anorexic group at low weight to be significantly slower than the control group on the Trail Making Test, but not after refeeding and they found performance of the anorexic group to be significantly poorer on the Austin Maze test and Block Design, which also improved on refeeding. No significant differences were found on the Serial Digit Learning Test or the Rey Auditory Learning Test. These results fail to confirm the learning deficit observed by De Witt et al. (1985), and Szmukler et al. (1992) interpret them as supporting visuospatial deficit. This is because concentration is necessary for both the learning tasks and the TMT, Block Design and Austin Maze, so the poor performance must be due to a visuospatial

deficit rather than a concentration/attention one. A visuospatial deficit lends support to a right hemisphere dysfunction hypothesis.

Thompson and Spana (1991) conducted a study designed to directly explore any link between incorrect estimation of body size and deficits in visuospatial ability. Subjects were 69 college women, but unfortunately sample selection specifically excluded anyone with a DSM III-R diagnosis of an eating disorder as this was part of a larger normative study. Subjects were required to estimate the size of their waist, hips and thighs using an adjustable light beam projecting onto a wall, a method with reported reliability and validity. Visuospatial ability was assessed using the Benton Visual Retention Test (BVRT) for short term visual memory and the Differential Aptitude Test - Spatial relations scale in which subjects have to match a 2-dimensional stimulus figure to a choice of four 3-dimensional figures. Bulimic behaviour was assessed by The Bulimia Test (Smith & Thelen, 1984). The only significant correlation was a .33 correlation between error scores on the BVRT and overestimation of thigh size. This finding is interesting and may prompt further research, but too weak to draw conclusions from. Replication with eating disordered subjects and controls might provide more information on the involvement of visual memory deficits in body image distortion, but findings

concerning impairments in eating disordered subjects thus far have been mixed.

Jones et al. (1991), as previously mentioned, found underweight anorexic, long term weight restored anorexics and bulimic subjects to perform no differently than control subjects on a test of vigilance using the Continuous Performance Task (CPT), involving recognition of a degraded stimulus, but the acute eating disordered groups to be relatively impaired on the focusing/execution aspect of attention. Laessle, Hank, Hahlweg and Pirke (1990) used the same CPT task to compare attention in bulimic subjects and a healthy control group. Their rationale for the study was that deficits in attention in anorexic subjects had been observed by Hamsher et al. (1981), and his aim was to assess this possibility in bulimics. However the study by Jones et al. (1991) suggests that Laessle et al. (1990) were testing the wrong aspect of attention. The aspect of attention found to be impaired by Hamsher et al. (1981) was that of focusing/execution, which Jones et al. (1991) also found. However the CPT measures the vigilance aspect of attention, which Hamsher did not test, and Jones found not to be impaired in either underweight anorexics or long term weight recovered anorexics. Subjects were 30 inpatients with DSM III-R diagnoses of bulimia, 15 of whom had a history of anorexia, and 23 healthy controls. The

measures used were; signals detected, responses to non-signals, accuracy of discrimination (perceptual sensitivity) and the amount of perceptual evidence needed to recognize the stimulus (response criterion level). They measured Betahydroxybutyric acid levels (BHBA) as an indicator of metabolic signs of starvation. They found signals detected and perceptual sensitivity to be significantly lower, and response criterion level to be significantly higher in the bulimic subjects, with no difference in response to non-signals. They also found of the bulimic group, subjects with indications of intermittent starvation (by BHBA levels) had significantly lower signals detected and accuracy of discrimination than bulimic subjects without signs of starvation. This indicates a deficit in vigilance performance in bulimia, which correlates with metabolic changes due to abnormal eating patterns. This is in opposition to the finding by Jones et al. (1991).

Research findings on the issue of a right hemisphere weakness in eating disorders have been mixed. Five studies have results that are suggestive of such a weakness [Fox (1981), Maxwell et al. (1984), McKay et al. (1986), Thompson (1991) and Szmukler et al. (1992)]. The McKay et al. (1986) study suggests a specific deficit in speed of initiation and execution of action in bulimic women. However, three of these studies [Fox (1981), Maxwell et al. (1984), Mckay et al. (1986)] have questionable validity due to methodological weaknesses. Three studies find no

evidence to support a right hemisphere dysfunction [Touyz et al. (1986), De Witt et al. (1985), Jones et al. (1991)] all having adequate methodology.

Smukler et al. (1992) and Jones et al. (1991) both found that neuropsychological deficits improved on refeeding, or were not present in long term weight restored anorexics, and Hamsher et al. (1981) also found a lessening of deficits after refeeding, but additionally that the presence of neuropsychological deficits after refeeding was a predictor of outcome at one year follow-up.

Factors have been discussed which may contribute to the inconsistency of neuropsychological findings in this field. There is a need for further research to resolve these issues which will be helped by a consolidation of diagnostic criteria, and by attempts to make methods comparable, but precision in neuropsychological research findings is dependent partly on progress in the science of neuropsychological testing.

1.7 BULIMIA AND IMPULSIVITY

One of the main features of bulimia is the binge and its associated feelings of loss of control. Bulimia is often considered a disorder of impulse control.

1.7.1 Definitional Difficulties

Impulsivity is described in the clinical literature as a feature of many different syndromes; Attention Deficit Hyperactivity Disorder, borderline personality disorder, intermittent explosive disorders, kleptomania, pyromania and bulimia, but its definition varies and suffers a lack of clarity. It is variously considered to involve the explosion of primitive affects, speed of response, risk taking behaviour or a lack of social responsibility. There are a number of self-report instruments designed to measure impulsivity, which tend to correlate moderately or highly, but correlations of these with psychometric tests of impulsivity such as the MFFT or the Porteus Maze test tend to be low (Barratt and Patton, 1985). This suggests that they are measuring different constructs. Barratt and Patton (1985) suggest the solution to this lack of correlation is that impulsivity consists of a motor component and a cognitive component, which are differentially addressed by questionnaire and psychometric test measures. The Barratt Impulsivity Scale (Barratt, 1985) is based on this hypothesis, and has a motor scale, a cognitive scale and a non-planning scale.

1.7.2 Findings on Bulimia and Impulsivity

A number of studies report clusters of other so called impulsive behaviours associated with bulimia. These have included drug and alcohol abuse (Bulik, 1987), suicide attempts, self injurious behaviour (Weiss & Ebert, 1983), aggressive outbursts (Vandereycken & Pierloot, 1983), and stealing (Mitchell, Hatsukami, Pyle, & Eckert, 1987).

Mitchell et al. (1987) indicate that their data on stealing suggests its existence before the eating disorder, so necessity due to high food or laxative consumption is not its only explanation.

Lacey & Evans (1986) examined stealing, drug abuse and alcohol abuse, self harm and promiscuity in 112 patients at an eating disorder clinic, and concluded that there exists a subgroup of bulimics who show a failure of impulse control in multiple areas, and who have a particularly bad prognosis. They coined the term "multi-impulsive personality disorder" to describe this group, but fail to distinguish it from Borderline Personality Disorder, which also occurs at a high rate in bulimia, sufficiently to give the concept validity.

In addition to these observations of greater behavioural impulsivity in bulimics, experimental evidence for greater cognitive impulsivity in bulimics comes from Toner et al. (1987). She reported that bulimic anorexics made significantly more errors on the Matching

Familiar Figures Test (MFFT) than weight restored restrictor anorexics. Errors on this test demonstrate cognitive impulsivity.

Bulimics have been found to score higher than restrictor anorexics on the psychoticism scale of the Eysenck Personality Questionairre, which indicates greater impulsivity. However, restrictor anorexics scored higher on the Lie scale than bulimics, which suggests caution in interpretation (de Silva & Eysenck, 1987).

Brewerton, Hand & Bishop, (1993) examined the results of Cloningers' Tridimensional Personality Questionairre (TPQ) in a large sample of 110 bulimics, 27 anorexics and 10 subjects with bulimia and anorexia and 350 controls. They found that bulimics had significantly higher scores on Novelty Seeking, and specifically on the Impulsivity subscale than controls.

Similar results were found by Bulik, Sullivan, McKee, Weltzin & Kaye (1994) who compared bulimic women with and without alcohol abuse on the TPQ. They also found bulimic women to be higher than general population norms on Novelty Seeking, whether alcohol abuse was present or not. They found the group with alcohol abuse to be significantly higher on Novelty Seeking than the group without, but not on the Impulsiveness subscale.

Sohlberg, Norring, Holmgren & Rosmark (1989) compiled an impulsivity index from the presence of binge eating, stealing, alcohol

or drug abuse and suicide attempts. They found scores on this to be the best predictor of long-term outcome at follow up two and a half years later. High scorers were found to have more disordered eating at follow-up by information gathered in a semi-structured interview. They discuss their results in terms of the high rates of personality disorder found in eating disorder populations. Impulsive behaviours are involved in the borderline-histrionic spectrum of personality disorders and this population can also be expected to have poorer outcome in treatment.

Piran, Lerner, Garfinkel, Kennedy & Brouillette (1988) used unstructured interviews to study personality disorder distribution in eating disorder populations. They found 66% of their bulimic sample had Cluster B, dramatic-erratic personality disorders, compared to 0% of anorexics. Seventy-seven percent of the anorexic sample had Cluster C, anxious, personality disorder diagnoses compared to 29% in the bulimic sample. Cluster B personality disorders include borderline, histrionic, narcissistic and antisocial personality disorders. Impulsivity is a diagnostic feature in all of these disorders.

Levin and Hyler (1986) used consensus decisions by two
psychiatrists after semi-structured interviews with 24 normal-weight
bulimics to diagnose personality disorders. They suggested their results

indicate a heterogeneity of personality disorder diagnoses in eating disordered groups. Sixty-three percent of the bulimic sample qualified for personality disorders in the borderline histrionic spectrum, while 29% qualified for diagnosis in the anxious spectrum.

Newton, Freeman, & Munro (1993) examined a clinic sample of 58 normal-weight bulimics and 27 controls with the Barratt Impulsivity Scale (BIS) before treatment. They found BIS total scores to be significantly higher in bulimics compared to controls. They found the BIS motor subscale to be significantly higher in bulimics than controls, but no other subscale showed significant differences between groups.

However, evidence has not always supported the concept of greater impulsivity in bulimics.

Fahy and Eisler (1993) used Eysenck & Eysencks' Impulsivity

Questionnaire to test a sample of 29 anorexics, 23 bulimic anorexics and

44 bulimics. The Impulsivity Questionnaire is designed to measure

impulsivity in terms of decision making without reference to risk.

They found that bulimic subjects had higher impulsivity scores than

anorexics, but their scores were not significantly different to norms for

healthy women. They illustrate that the so-called impulsive

behaviours contributing to the description of bulimia as impulsive

may not necessarily be so. Suicidal behaviour, substance abuse and

stealing are not always performed without planning and consideration of risk. They conclude that there is not evidence enough to attribute the constellation of these behaviours to an underlying impulse control disorder.

Gartner, Marcus, Halmi & Loranger, (1989) studied personality disorders in eating disordered populations with structured interviews, a methodological advantage over the unstructured interviews of Piran et al. (1988) and Levin & Hyler (1986). They found anxious Cluster C personality disorders to be equally common in bulimic and anorexic samples.

Wonderlich, Swift, Slotnick & Goodman (1990), also using structured interviews, reported that Cluster B dramatic-erratic personality disorders were equally common in anorexic and bulimic samples.

Feldman and Eysenck (1986) tested the hypothesis that bulimics lack impulse control with the Impulsiveness Questionnaire (Eysenck, 1985). There were no significant differences on the Impulsiveness Questionnaire between bulimics and controls thus the hypothesis was not supported.

Laessle, Kreig, Fechter, and Pirke (1990) using a degraded stimulus continuous performance test, failed to find support for increased

impulsivity in bulimics. In this study, impulsivity was measured by response criterion level, ie. amount of perceptual evidence before recognition of a target. It was found that bulimic subjects had a significantly higher response criterion than healthy controls which means they showed more caution before making the decision that a target signal had occurred.

Thus far evidence supporting the description of bulimics as more impulsive than healthy women is mixed and further research is necessary to clarify the issue.

1.8 THE PRESENT STUDY

The neuropsychological literature suggests several explanations for the poor experimental performance originally discussed in this paper. Reaction time has been showed to be slowed in anorexic subjects, an effect which improves with treatment. This has not been tested in bulimic subjects. There is evidence that motor functioning, especially in speed of initiation and execution is impaired in bulimic women. The existence of spatial deficits is still under debate and could be a contributor. An impairment in attention functions is well established, and could be a contributor. If impulsivity is a significant

factor in bulimia, it could extend to motor functioning. Thus deficits in motor impulse control could also explain the observed effect that is the focus for this investigation.

The purpose of this investigation was to explore the difference in observed experimental performance between bulimic and control women. A second purpose is to examine any change in neuropsychological functioning before and after treatment in a sample of bulimic women. This design allows the discrimination between acute disease effects and a possible trait that may predispose to the development of bulimia. A test has been chosen to assess each of the following neuropsychological functions; self-report of impulsivity (BIS), reaction time, visuo-spatial organisation/visuomotor tracking (Trail Making Test), motor speed (Finger Tapping), motor impulse restraint (Go No Go), verbal response inhibition (Stroop Test), and concentration/attention (Digit Span).

1.8.1 Hypotheses

It was hypothesised that tasks not impaired by impulsive performance would display a pre-treatment impairment in bulimic subjects compared to control subjects, which would disappear after treatment.

If, as some literature suggests, impulsivity in bulimic subjects

takes the form of a trait, then tests affected by impulsivity should be impaired in bulimics before and after treatment.

It is hypothesised that bulimic subjects will score higher than controls on the Barratt Impulsivity Scale. This is not expected to change after treatment. This would suggest a trait of impulsivity in bulimic subjects.

It is hypothesised that Go No Go errors of commission will be higher in bulimic subjects before treatment, and that this will not change after treatment. This would show motor impulsivity and support a trait of impulsivity in bulimic subjects.

It is hypothesised that Stroop colour-word naming times will be will be higher in bulimic subjects before treatment, and that this will not change after treatment. Longer colour-word naming times are caused by more impulsive errors and more interference from the attempt to inhibit the overlearned correct response, and thus are suggestive of higher impulsivity.

It is hypothesised that bulimic subjects will have poorer TMT performance than controls, and that this difference will disappear after treatment. This would support the notion of a right hemisphere visual-spatial deficit in bulimia.

It is hypothesised that impaired attention will be displayed by

bulimic subjects before treatment, by lower Digit Span Scores than controls. This difference is expected to disappear with treatment. This would support previous findings of attention impairment due to specific disease effects.

It is hypothesised that bulimic subjects will have slower reaction times than control subjects before treatment, and that this difference will disappear after treatment. This would support previous findings of reaction time impairment due to specific disease effects.

It is hypothesised that bulimic subjects will have lower finger tapping scores than control subjects before treatment, and that this difference will disappear after treatment. This would support the previously discussed finding of impairment of right frontal functions of speed of initiation and execution.

Depression may have an effect on neuropsychological performance. The hypothesis that any group differences in performance are effected by depression rather than bulimia will be tested with analysis of covariance.

Chapter 2 METHOD

2.1 SUBJECTS

Nineteen women with bulimia nervosa and 19
healthy female controls participated in this study. Probands
and controls were matched for age and pro-rated IQ level. Ethical
approval for this investigation was received from the Human Ethics
Committee of the Canterbury Area Health Board, from the University
of Canterbury Ethics Committee, and all subjects gave written informed
consent.

2.1.1 Bulimic Subjects

Subjects with bulimia nervosa were participants in an outpatient cognitive-behavioural clinical trial. Recruitment to the study was community based, and the main sources were self referrals. Subjects with current anorexia nervosa, obesity (i.e. BMI greater than 30), significant medical illness effecting the eating disorder, or medications effecting the eating disorder, such as anti-depressants were excluded from the study. The ideal programme length was 12 weeks, but some subjects remained in treatment for up to 24 weeks.

2.1.2 Control Subjects

Control subjects were recruited by a snowball method starting with associates of the author. Subjects were screened with the Eating Disorders section of the Structured Clinical Interview for DSM-III-R (SCID) to exclude an eating disorder.

2.2. MATERIALS AND APPARATUS

2.2.1 Diagnostic Information

In the first two days of the intake phase bulimic subjects were assessed by a clinical psychologist or a psychiatrist using the SCID.

Control subjects were assessed by the author using the SCID-Eating Disorders section to exclude the presence of an eating disorder. The following self-report measures were completed by all subjects during the assessment phase and again at the completion of treatment, or at a similar interval for the controls.

2.2.2. Eating Disorders Inventory (EDI2)

All subjects completed the EDI2 as a measure of severity. This is a commonly used 91 item self-report scale. It has 11 subscales; Drive for thinness, Bulimia, Body dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal distrust, Interoceptive awareness, Maturity fears, Ascesticism, Impulse regulation and Social insecurity.

which are provisional. It correlates with other eating disorder scales and distinguishes anorexia nervosa and bulimia nervosa.

2.2.3 State Anxiety Inventory (SAI)

The State Anxiety Inventory is a 20 item self report measure of current anxiety. The subject is requested to answer questions based on "how you feel right now". The scale has good reliability (Cronbach alpha=0.93) and New Zealand norms. (Knight, Waal-Manning & Spears, 1983)

Due the effects of anxiety on test performance, all subjects completed the SAI to measure of anxiety in the testing situation. Total scores were used as the dependent measure.

2.2.4 Beck Depression Inventory (BDI)

The Beck Depression Inventory is a 21 item self report measure for depressive symptoms in the last week. It has a scoring range from 0-63, with 0-9 indicating none or mild depression, 10-18 mild-moderate, 19-29 moderate-severe and 30-63 indicating severe depression. It has acceptable test-retest reliability (r= 0.48-0.86, Beck, Steer & Garbin, 1988)

Due to the effects of depression on concentration, memory and

motor speed all subjects completed the Standard Form of the Beck Depression Inventory. Total scores were used as the dependent measure.

2.2.5 Barratt Impulsivity Scale (BIS)

The Barrett Impulsivity Scale-10 is a 30-item report scale which measures impulsivity (Barratt, 1985). There are three subscales: cognitive, non-planning and motor. The cognitive subscale measures impulsivity in terms of quick cognitive decision making. The non-planning subscale measures impulsivity in terms of a lack of forward planning in problem solving, and the motor subscale measures impulsiveness involved in acting without thinking. It has been shown to correlate with non-questionnaire measures of impulsivity such as the Porteous Maze Test and the MFFT.

2.3 NEUROPSYCHOLOGICAL TESTING

The neuropsychological battery consisted of the following tests.

2.3.1 Pro-Rated Intelligence Quotients

Due to the confounding effect of IQ in some of the

neuropsychological tests, Silversteins' Short Form of the Weschler Adult Intelligence Scale- Revised (Silverstein, 1982) was used to equate the proband and control groups. This uses the vocabulary and block design subtests to pro-rate IQ levels. Standard instructions and procedures were used. The dependent measure used was the age adjusted pro-rated IQ score. IQ assessment was not repeated.

2.3.2 Weschler Adult Intelligence Scale- Digit Span Subtest

The Digit Span subtest was given as a measure of concentration. Standard instructions were used and the dependent measure was the age adjusted scaled score.

2.3.3 Trail Making Test (TMT)

The Trail Making Test was given as a measure of visuomotor tracking. Standard instruction were given. Reitans' method of administration was used whereby the examiner points out errors as they occur and the scoring is based on the time taken. The dependent measure was the time taken to complete each part.

2.3.4 The Finger Tapping Test (FTT)

The Finger Tapping Test was used as a measure of motor speed. A

standard board with tap counter was used. Three 10 second trials were given with the preferred hand allowing short rests between trials. The dependent measure was the mean number of taps across the three trials.

2.3.5 Reaction Time

A standard no-choice reaction time task was used. Warned trials were given, using a verbal "ready" cue preceding the visual stimulus by 2 seconds. The time between the visual stimulus and the lifting of a button was the reaction time. Five practice trials were given and this was followed by 10 actual trials. The mean of the ten trials was the dependent measure.

2.3.6 Go No Go Test

As a test of motor impulsivity a computerised adaptation of Lurias' Go No Go test was created. The stimuli were a single or a double auditory tone emitted from a Macintosh Powerbook 100.

Subjects were requested to respond to a single tone with a single space bar press, and to respond to a double tone with no reaction. In such a way the subject was required to make a motor response to one cue and withhold response to another, thus displaying impulsivity in errors of

commission. The test consisted of a practice trial of 5 single and double tones randomly ordered, and repeated until responses were correct. This was followed by three sets of 10 tones randomly ordered, each tone separated by .5 second. A five second gap between each of the three trials was given. All subjects received the same order of presentations. The subjects response was automatically recorded. The dependent measures used were the number of errors of commission.

2.3.7 Stroop Test

The Stroop colour naming task was used to assess the inability to inhibit immediate but inappropriate responses. It has previously been used for this purpose by Heilbrun and Bloomfield (1986) and Boucugnani and Jones (1989). Three different cards were presented. Each had 100 items printed in 10 rows of 10 with an extra row at the top separated from the others to provide a practice. Card A had randomised colour names; red, blue, green printed in black ink. Card B had randomised colour names; red blue and green, printed in conflicting coloured inks of red blue or green. Card C had randomised square blocks of coloured ink in the same red blue or green. For card A subjects were requested to read the words, for card B subjects were requested to ignore the words and read the colour of the ink, and for card C they were requested to name the colours of the squares. They

were requested first to read the practice line to habituate to the task, and then instructed to complete the task "as fast as possible" and to self correct any mistakes. The experimenter pointed out mistakes as they were made. Dependent measures used were the time taken in seconds to complete each card. Errors were not recorded directly as they were incorporated into total time taken. An interference index (time for Card B - time for Card C) was calculated to detect interference while controlling for varying colour naming speeds.

2.4 PROCEDURE

Subjects with bulimia were tested at the Clinical Research Unit at The Princess Margaret Hospital, and later at its' new site at Terrace House, between April 1993 and July 1994. Control subjects were tested between December 1993 and December 1994 in the Psychology Department at the University of Canterbury.

Neuropsychological testing of bulimic subjects was conducted on the third day of the assessment phase. A booklet of the self report measures was given to the women with bulimia on the first day of assessment to complete at home. Testing and self report measures were repeated at the end of treatment. Control subjects were assessed and tested on the same day and also given the self report measures to complete at home. Control subjects received their post-test at intervals yoked to the subjects with bulimia. All neuropsychological tests were completed in one session in a 1.25 hour period.

Chapter 3 RESULTS

3.1 SUBJECT CHARACTERISTICS

Bulimic and control subjects were matched for age and IQ and did not differ significantly on these variables [age; t(37)= 1.51, p=0.14] IQ; t(37)= 1.56, p=0.13)], as shown in Table 1. There was no significant difference between bulimic and control subjects on Body Mass Index [BMI; t(37)= 0.45, p=0.65], as shown in Table 1. Bulimic subjects had significantly higher EDI2 scores than controls on all subscales except Perfectionism and Maturity Fears; [Drive for Thinness; t(35)=8.15, p=0.00, Bulimia; t(37)=5.30, p=0.00, Body Dissatisfaction; t(35)=5.34, p=0.00, Ineffectiveness; t(35)=3.19, p=0.00, Perfectionism; t(37)=1.67, n.s., Interpersonal Distrust; t(35)=4.21, p=0.00, Interoceptive Awareness; t(35)=5.09, p=0.00, Maturity Fears; t(35)=1.33, n.s., Asceticism; t(35)=4.91, p=0.00, Impulsiveness; t(35)=3.25, p=0.00, Social Insecurity; t(35)=4.14, p=0.00]. Means and standard deviations are displayed on Table 3.

Before treatment, scores on the Beck Depression Inventory showed bulimic subjects to be significantly more depressed than control subjects, [BDI; t(36)=3.68, p=0.0008]. Bulimics were less depressed after treatment, although still significantly more so than control subjects, [BDI; t(36)=2.06, p=0.047] as shown in Table 2.

Table 1.

<u>Subject Characteristics</u>

	BULIMIC	CONTROL	
	n=20	n=19	
AGE			
mean	27.10	29.63	
s.d.	5.72	4.68	
I.Q.			
mean	107.95	114.90	
s.d.	14.69	12.99	
B.M.I.			
mean	23.00	23.70	
s.d.	5.04	6.83	

4

TABLE 2: NEUROPSYCHOLOGICAL TEST RESULTS FOR BULIMIC AND CONTROL SUBJECTS

	BULIMIC		CONTROL		p. VALUES				
	pre	post	<u>pre</u>	post			<u>Dx</u>	<u>time</u>	Dx x time
	mean+SD	mean+SD	mean+SD	mean+SD	<u>Pre</u>	<u>Post</u>	<u>D</u> X	<u>D</u>	<u>DX X CITIE</u>
BDI	16.4 <u>+</u> 9.7	9.5 <u>+</u> 8.9	5.4 <u>+</u> 10.8	4.0 <u>+</u> 4.4	0.0008	0.05			
SAI	45.4 <u>+</u> 12.3	35.4 <u>+</u> 9.9	33.1 <u>+</u> 11.4	33.9 <u>+</u> 11.5	0.03	ns			
STROOP WORD	44.5 <u>+</u> 8.1	43.2 <u>+</u> 8.5	46.4 <u>+</u> 5.8	46.0 <u>+</u> 7.9			ns	ns	ns
STROOP COLOUR BLOCK	57.3 <u>+</u> 12.5	54.5 <u>+</u> 13.9	61.5 <u>+</u> 6.7	60.6 <u>+</u> 9.7			ns	ns	ns
STROOP COLOUR WORD	96.1 <u>+</u> 31.8	86.7 <u>+</u> 24.2	116.2 <u>+</u> 28.3	115.3 <u>+</u> 28.8			ns	ns	กร
STROOP INTERFERENCE INDEX	38.8 <u>+</u> 21.1	32.2 <u>+</u> 13.0	54.7 <u>+</u> 24.5	54.7 <u>+</u> 21.4			0.03	ns	ns

TABLE 3: EDI2 SUBSCALE SCORES FOR BULIMIC AND CONTROL SUBJECTS

SUBSCALE	BULIMIC	CONTROL	Б
Drive for Thinness	<u>mean+SD</u> 14.4 <u>+</u> 4.9	<u>mean+SD</u> 2.7 <u>+</u> 3.4	0.00
Bulimia	8.5 <u>+</u> 5.7	1.2 <u>+</u> 2.0	0.00
Body Dissatisfaction	18.5 <u>+</u> 7.0	5.7 <u>+</u> 7.5	0.00
Ineffectiveness	8.4 <u>+</u> 5.9	2.7 <u>+</u> 4.6	0.00
Perfectionism	5.2 <u>+</u> 5.0	3.2 <u>+</u> 1.8	ns
Interpersonal Distrust	5.8 <u>+</u> 4.5	0.8 <u>+</u> 0.9	0.00
Interoceptive Awareness	7.7 <u>+</u> 5.0	1.2 <u>+</u> 1.6	0.00
Maturity Fears	3.0 <u>+</u> 3.3	1.8 <u>+</u> 1.6	ns
Asceticism	7.1 <u>+</u> 3.9	2.1 <u>+</u> 1.6	0.00
Impulsiveness	2.8 <u>+</u> 2.6	0.5 <u>+</u> 1.2	0.00
Social Insecurity	6.3 <u>+</u> 3.7	1.8 <u>+</u> 2.8	0.00

4
7

	TABLE 4: NEUROPSYCHOLOGICAL TEST RESULTS FOR BULIMIC AND CONTROL SUBJECTS BULIMIC CONTROL						
	<u>pre</u> mean+SD	post mean+SD	pre mean+SD	post mean+SD	p. VALU Dx p	ES time p	Dx x time p
DIGIT SPAN	11.16 <u>+</u> 2.3	11.95 <u>+</u> 2.6	10.47 <u>+</u> 2.6	11.42 <u>+</u> 2.6	ns	0.002	ns
REACTION TIME	28.17 <u>+</u> 4.5	26.40 <u>+</u> 4.7	23.65 <u>+</u> 1.8	22.95 <u>+</u> 2.9	0.01	ns	ns
FINGER TAPPING	44.31 <u>+</u> 7.5	46.00 <u>+</u> 7.0	47.76 <u>+</u> 4.8	47.53 <u>+</u> 5.3	ns	ns	ns
TMT PART A	23.95 <u>+</u> 7.4	18.53 <u>+</u> 5.5	22.95 <u>+</u> 5.1	21.26 <u>+</u> 4.8	ns	0.0001	0.03
TMT PART B	48.30 <u>+</u> 15.5	46.05 <u>+</u> 17.4	47.32 <u>+</u> 11.4	43.40 <u>+</u> 15.5	ns	ns	ns
BIS-TOT	68.83 <u>+</u> 12.3	62.28 <u>+</u> 11.1	63.05 <u>+</u> 13.3	62.00 <u>+</u> 10.9	ns	0.05	ns
BIS-NON-PLANNING	26.94 <u>+</u> 5.7	24.67 <u>+</u> 5.2	26.11 <u>+</u> 6.0	26.28 <u>+</u> 4.9	ns	ns	0.10
BIS-MOTOR	26.72 <u>+</u> 5.5	23.83 <u>+</u> 3.9	22.78 <u>+</u> 4.7	23.00 <u>+</u> 4.4	0.06	ns	0.11
BIS-COGNITIVE	15.17 <u>+</u> 3.5	14.39 <u>+</u> 3.1	14.61 <u>+</u> 4.2	14.00 <u>+</u> 3.5	ns	ns	ns

Before treatment, scores on the State Anxiety Inventory showed bulimic subjects to be significantly more anxious than control subjects, [SAI; t(20)=2.42, p=0.025]. Bulimics were less anxious after treatment, showing no significant difference from control subjects. Means are reported in Table 2.

3.2 NEUROPSYCHOLOGICAL MEASURES

To avoid violation of the normality assumption in MANOVA, all variables were assessed for normality using the Shapiro-Wilks test. A repeated measures MANOVA for one between factor (Diagnosis) and one within factor (Time) was performed using JMP (SAS Institute, 1994) for all approximately normally distributed variables.

Means and significance values for the following variables are presented in Tables 3 and 4, and means are displayed in graph form in Figures 1-10.

3.2.1 Barratt Impulsivity Scale

The BIS Total scores displayed a significant main effect for time [F(1,34)=4.3, p=0.05], with scores lower at post-testing. There was no significant difference between the bulimic and control group, and the group x time interaction was also not significant. Means are graphed in

Figure 1.

The Non-Planning subscale of the BIS showed no significant main effects for group or time and no significant group x time interaction.

Means are graphed in Figure 2.

The Motor subscale of the BIS showed no significant main effect for group or time and no significant group x time. Means are graphed in Figure 3.

The Cognitive subscale of the BIS showed no significant main effects for group or time and no significant group x time interaction. Means are graphed in Figure 4.

3.2.2 The Go-No-Go Test

Scores on this test showed an extreme non-normal distribution. As there is no standard non-parametric significance test for repeated measures with data of this distribution, the dependent variable was converted into a dichotomous variable (0-no errors, 1-one error, 2-more than one error) and a chi square comparison was performed on the pre-test. The post-test data were converted into a dichotomous variable related to the pre-test (i.e. 0-decrease, 1-no change, 2-increase), and chi-square performed again. There were no significant differences in go-no-go errors before or after treatment.

3.2.3 The Stroop Test

Card One and Card Two of the Stroop test (ie. the colour block page and the word page) showed no significant main effects for time or group, nor significant interaction effects. On the third card, the interference colour word card, the main effect for group approached significance with the bulimic group tending to have faster reading times than the control group [F(1,18)=4.12, p=0.057]. When the interference index was analysed it revealed a significant group effect, with the bulimic group showing significantly faster time [F(1,18)=5.82, p=0.03]. Means are graphed in Figure 5.

3.2.4 The Trail Making Test

The time taken to complete the Trail Making test Part A showed a significant decrease over time [F(1,36)=18.75, p=.0001]. There was no significant difference between the control group and the bulimic group, but there was a significant time x group interaction [F(1,36)=5.19, p=0.03], with the bulimics times reducing more after treatment than the controls. Means are graphed in Figure 6. The time taken to complete the Trail Making test Part B showed no significant difference between the bulimic or the control group or over time. The interaction between

time and group was also not significant. Means are graphed in Figure 7.

3.2.5 Digit Span

Digit span for both the bulimic and control group showed a significant increase over time [F(1,36)=11.20, p=0.002]. The main effect for group was not significant. The diagnosis x time interaction was also not significant. Means are graphed in Figure 8.

3.2.6 Finger Tapping

Finger tapping scores did not differ significantly by diagnosis or by time. The diagnosis x time interaction was also not significant. Means are graphed in Figure 9.

3.2.7 Reaction Time

Control subjects showed significantly faster reaction times than bulimic subjects [F(1,20)=7.90, p=0.01]. There was no effect for time and the time x group interaction was not significant. Means are graphed in Figure 10.

To determine whether the difference in reaction time was affected by the presence of depression, an analysis of covariance was performed using pre-treatment BDI scores as the covariate. The whole model was significant [F(2,21)=7.86, p=0.003]. The effect of BDI score was significant [F(1,21)=3.76, p=0.07]. The independant effect of diagnosis when controlling for depression was reduced to non-significance [F(1,21)=1.08, p=0.31].

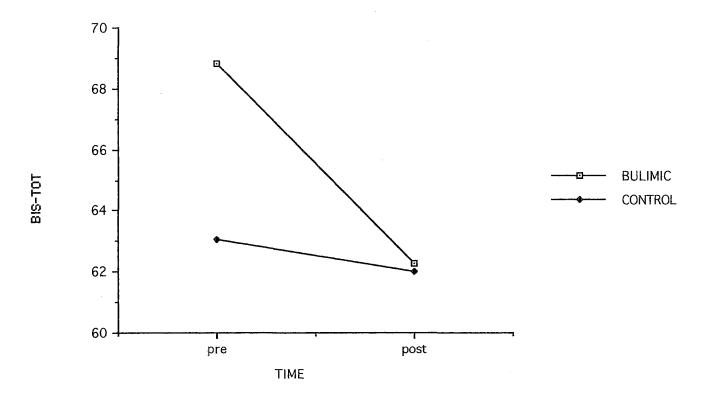
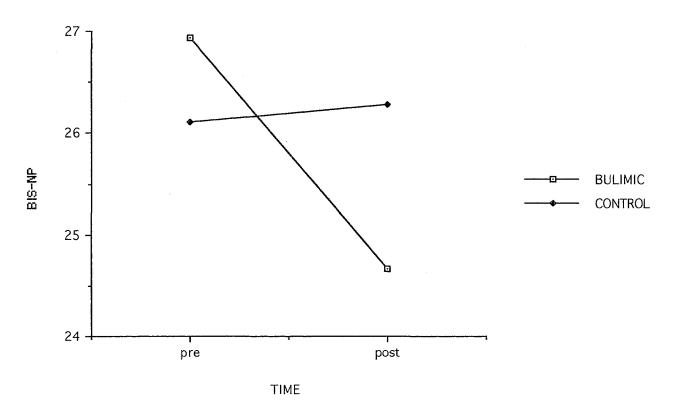
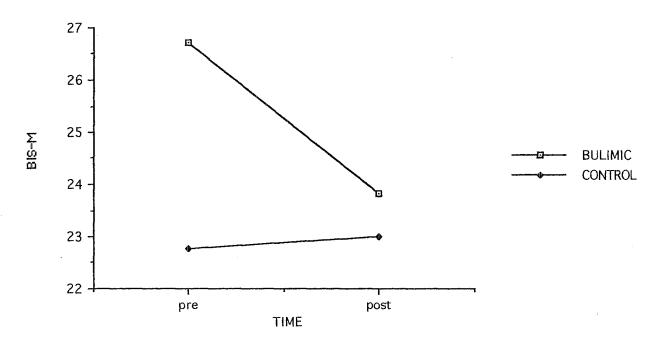


FIGURE 2: BIS-Non Planning Means for Bulimic and Control Subjects Pre- and Post-Treatment.



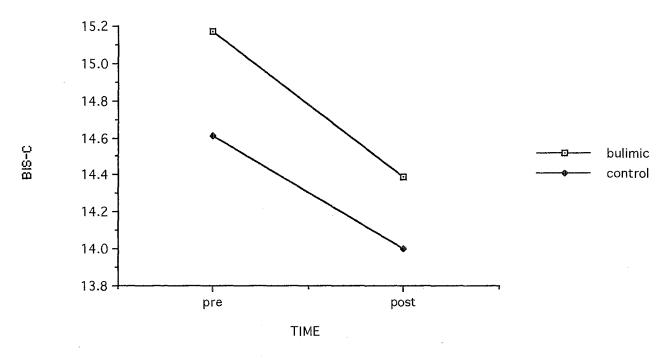
7

FIGURE 3: BIS-Motor Means for Bulimic and Control Subjects Pre- and Post-Treatment.



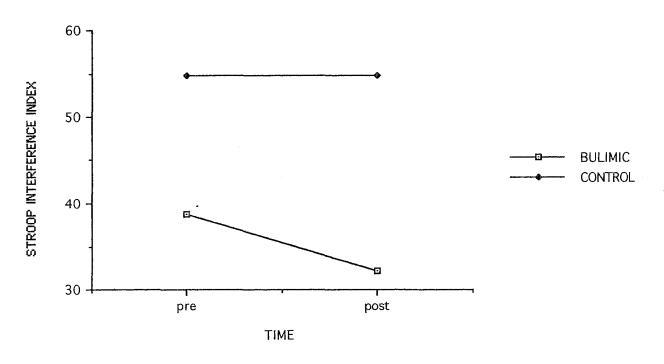
Û

FIGURE 4: BIS-Cognitive Means for Bulimic and Control Subjects Pre- and Post-Treatment.



ý

FIGURE 5: Stroop Interference Index Means for Bulimic and Control Subjects Pre- and Post- Treatment.



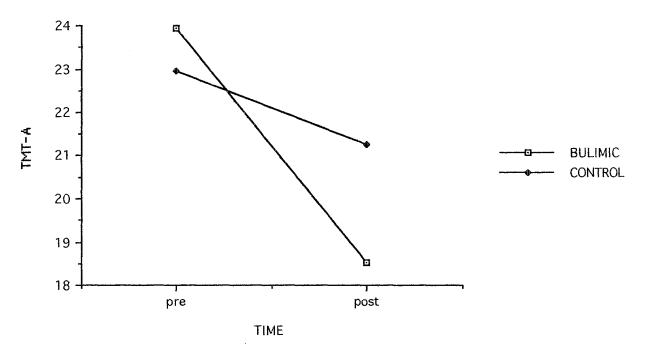


FIGURE 7: Trail Making Test-B Means for Bulimic and Control Subjects Pre- and Post-Treatment.

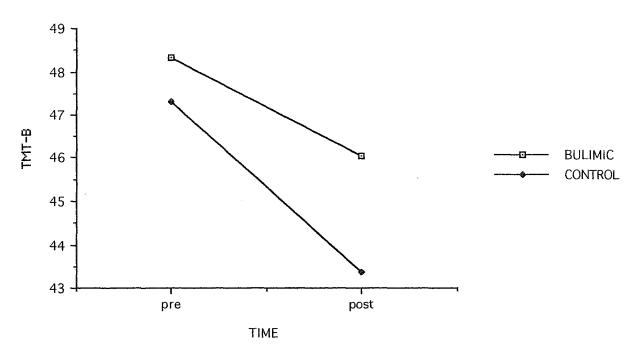
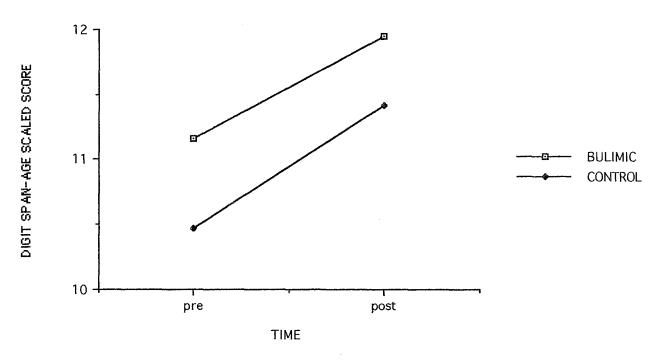
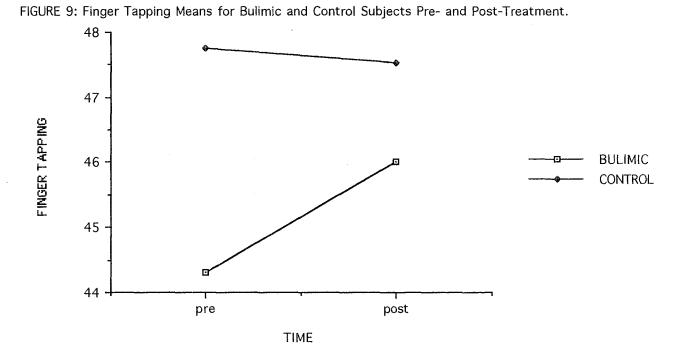


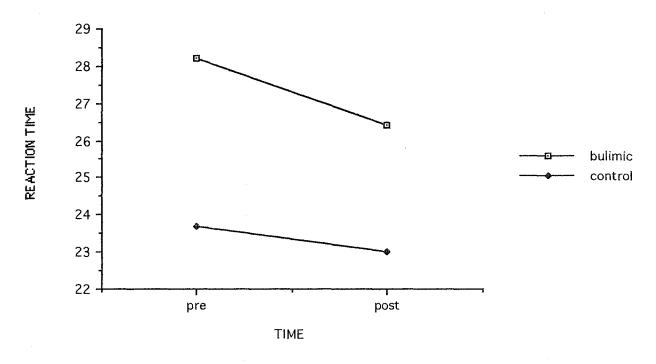
FIGURE 8: Digit Span Age-Scaled-Score Means for Bulimic and Control Subjects Pre- and Post-Treatment.





59

FIGURE 10: Reaction Time Means for Bulimic and Control Subjects Pre- and Post-Treatment.



Chapter 4 DISCUSSION

The aim of this investigation was to explore the cause of the poor motor performance in computer games observed in women with bulimia. I observed that bulimic subjects tended to require longer practice periods before targets could be consistently, efficiently landed on, and results showed a generally lower hit rate for bulimic subjects compared to controls. It appeared that their difficulty was in failing to stop the movement of the cursor fast enough once it was initiated.

A number of hypotheses which could account for this effect were tested in this study.

First, it was hypothesised that bulimic subjects would display a trait of impulsivity by higher scores than control subjects on the BIS before treatment and after treatment. However, this hypothesis was not confirmed. Total BIS scores displayed no difference between bulimic and control groups. There was a significant reduction in BIS-total scores from pre- to post-treatment, which was mainly caused by reduction in the bulimic subjects scores. This can be explained by the fact that treatment involved a cognitive behavioural programme with a relapse prevention component, of which one specific goal was control over urges to binge. This is counter to the findings of Newton et al.

(1993), who found bulimic subjects to have higher scores on the BIS than healthy controls.

Second, it was also hypothesised that women with bulimia would display motor impulsivity marked by a higher number of errors of commission on the Go No Go Test than the control subjects, before treatment and after treatment. This hypothesis was also not confirmed. Control subjects made significantly more errors on the Go No Go Test pre-treatment than the bulimic subjects. There was no significant difference between bulimic and control subjects post-treatment. According to this result bulimic subjects actually displayed less impulsivity than control subjects. This was the opposite of what was predicted. Results on this test across all subjects showed a low rate of errors, (0-3). The distribution of errors suggests it to be a test able to detect gross deficits of motor impulse control, but too insensitive to detect more subtle deficits. Bulimic subjects' scores show no significant change over treatment, but the concept of a trait of impulsivity was not supported by the absence of the main effect.

Third, it was hypothesized that bulimic subjects would display a trait of impulsivity by showing less response inhibition, marked by higher scores on the Stroop Interference Index before and after treatment. This hypothesis was also not confirmed. In fact, the reverse

was so, with control subjects showing significantly more interference than bulimic subjects. There were no significant changes over treatment, but the concept of a trait of impulsivity was not supported.

Although The Stroop Test is used as a test of response inhibition, it is a non-specific test. It is also variously used to test ability to change perceptual set, to test the effects of perceptual interference and as a test of concentration. However a deficit in any one of these areas would be expected to impair performance, thus although the test is non specific, with unimpaired perormance, it can be determined if a particular deficit is absent.

Results from three separate measures, the Barratt Impulsivity

Scale, the Go No Go Test and the Stroop Test converge in failing to

characterise the bulimic sample in this study as more impulsive than

control sample. This contrasts with some of the clinical literature

(Lacey & Evans, 1986) and general clinical opinion. However, this

result agrees with the findings of Fahey and Eisler, (1993) Feldman and

Eysenck, (1986) and Laessle et al., (1990). There are a number of possible

reasons for this finding. Although none of the women in the present

sample had current anorexia nervosa, a history of anorexia nervosa

was not an exclusion criterion for entry into the trial. Previous authors

(Laessle et al., 1990) have found different impulsivity characteristics in

bulimic women with a history of anorexia and bulimic women without a history of anorexia. Bulimic women with a history of anorexia tend to have more rigid-controlled characteristics than those without a history of anorexia. It may be that partition of the proband group according to history of anorexia may have shown different results. However partitioning on this dimension would have lead to unacceptably small subsample size and inadequate statistical power.

An alternative explanation may lie in the sampling method. Most previous studies have relied on clinic samples from tertiary referral centres. These clinic samples are likely to have a sample bias towards more extreme disorder and co-morbidity. It may be that impulsivity is a feature in more extreme disorder. Depression, frequently co-morbid with bulimia, is associated with impulsivity (Jimerson, Lesem, Kaye & Brewerton, 1990). The current study used a largely self-referred clinical sample thus reducing the likelihood of this bias in severity. These results suggest that it may be inaccurate to characterise women with bulimia as globally impulsive.

A secondary hypothesis with regard to impulsivity was that a difficulty in motor impulse restraint was responsible for the observation under study. There was no evidence found to support this hypothesis, but, as discussed, this test was too insensitive to rule out a

fine-grained deficit in motor impulse restraint.

In a state of starvation glucose energy supplies are exhausted and energy in the form of ketone bodies is used for metabolism. Ketone bodies do not reach all areas of the brain, which can be expected to have consequences for cognitive functioning (Laessle et al., 1990). Normal weight bulimic women are often in a state of metabolic starvation, thus may be expected to have resulting impaired cognitive functioning.

The current study had no measures of metabolic starvation in the bulimic subjects and thus it is not possible to partition the biochemical effects of disordered eating habits.

Fourthly, the hypothesis was tested that a visuo-spatial-motor deficit was responsible for the observation under study. Confirmation of this hypothesis would lend support for previous findings of a right hemisphere deficit in anorexia before re-feeding by Szmukler et al. (1992) and Fox (1981). However, results from the TMT revealed no evidence to support this hypothesis. A number of other authors have also failed to find impairment on the TMT. Touyz, Beumont and Johnson(1986) found no impairment in bulimic and anorexic subjects compared to norms on the TMT. Maxwell et al. (1984) and De Witt et al. (1985) also found no difference between psychiatric controls and women with anorexia on the TMT.

Fifth, the hypothesis was tested that a deficit in concentration or

attention was responsible for the observation under study. This was predicted to disappear with treatment. Confirmation of this hypothesis would lend support for the previous findings which have been attributed to non-specific disease effects associated with having a mental disorder (Fox, 1981 and Hamsher, 1981). This hypothesis was not supported, with no differences found between bulimics and controls on the test of digit span before treatment, and an increase in both groups after treatment. It is likely that a practice effect or a reduction of anxiety caused by novelty contributed to this increase. Converging support for the reliability of this result comes also from the results of the Stroop test, a test which is impaired by deficient concentration.

Sixth, the hypothesis was tested that a motor speed or dexterity deficit was responsible for the observation under study. Confirmation of this hypothesis could lend support for the reported findings of impairment in right frontal functions of speed of initiation and execution. However results of the finger tapping test provide no support for this, with no differences evident between bulimic and control groups or over treatment.

Seventh, the hypothesis was tested that a slowing of reaction time was responsible for the observation under study. This hypothesis was

supported with bulimic subjects displaying significantly slower reaction times than control subjects. Both bulimic and control groups showed a decrease in reaction time by the second testing, which suggests a practice effect.

To further explore the reaction time differences, the possibility that group differences in neuropsychological performance might be caused by depression rather than bulimia was tested. Analysis of covariance of reaction time scores with BDI scores revealed a reduction of the significance of group differences to non significance. This confirms that depression was a significant factor in the slower reaction times of bulimic women. However, the sample size for the reaction time test was only 10, raising the possibility that a more powerful design may have produced a significant result.

Neuropsychological testing in this study has revealed little impairment in the neuropsychological functioning of women with bulimia. The only neuropsychological function found to be impaired with respect to the control group was that of reaction time. On the Stroop test, a measure of response inhibition and concentration, bulimic subjects performed significantly better than control subjects. It is plausible that reaction time has the lowest threshold for impairment, as Hamsher et al. (1981) found deficits in reaction time to be the only

reliable predictor of outcome of all the neuropsychological functions they tested.

4.1 FUTURE RESEARCH

The phenomenon of increased impulsivity in bulimia is brought futher into question by the findings of this study. Further investigation is neccesary to clarify whether global impulsivity is a characteristic of women with bulimia, or just of a subset of bulimic women with more severe disorder and co-morbidity. Other subgroups within the bulimia disorder which require comparitive investigation with regard to impulsivity characteristics are women with and without a history of anorexia.

The phenomenon of motor impulsivity in bulimia was only grossly tested in this study, and future research should include finer testing of this function.

4.2 CONCLUSION

Of several different neuropsychological parameters investigated, the only significant difference that could have accounted for the deficit in bulimic womens' performance on a computer task was reaction time. Bulimic women were found to have significantly slower mean

reaction times compared to the control group. This supports a slowed reaction time in women with bulimia as causal in the observed phenomenon, but analysis suggests this is caused more by depression than bulimia.

REFERENCES

- Barratt, E. (1959). Anxiety and impulsiveness related to psychomotor efficiency. <u>Perceptual and Motor Skills</u>, 9, 191-198.
- Barratt, E. (1983). The biological basis of impulsiveness: The significance of timing and rhythm disorders. <u>Personality and Individual Differences</u>, 4(4), 387-391.
- Barratt, E. (1987). Impulsiveness and anxiety: Information processing and electroencephalograph topography. <u>Journal of Research in Personality</u>, 21, 453-463.
- Barratt, E., Patton, J., Olsson, N., & Zucker, G. (1981). Impulsivity and paced tapping. <u>Journal of Motor Behavior</u>, 13(4), 286-300.
- Barratt, E., Pritchard, W., Faulk, D., & Brandt, M. (1987). The relationship between impulsiveness subtraits, trait anxiety, and visual N100 augmenting/reducing: A topographic analysis.

 Personality and Individual Differences, 8(1), 43-51.
- Barratt, E. S. Patton, J. H. (1985). Impulsivity: Cognitive, Behavioral, and Psychophysiological Correlates. In C. D. B. Spielbeger J. N. (Eds.), <u>Advances in Personality Assessment</u> (pp. 77-122). Hillsdale, N.J.:Lawrence Earlbaum Associates.
- Barratt, E. Impulsivness subtraits, arousal and information processing. In: Motivation, emotion and personality Spence, Izzard eds.

 Amsterdam: Elsevier Science Publications. 1985: 137-146.
- Beck, A. T., Steer, R. A. & Garbin, M. G. (1988). Psychometric Properties of the Beck Depression Inventory: Twenty-five years of evaluation. <u>Clinical Psychology Review</u>, 8, 77-100.
- Boucugnani, L., & Jones, R. (1989). Behaviors analogous to frontal lobe dysfunction in children with attention deficit hyperactivity disorder. <u>Archives of Clinical Neuropsychology</u>, 4, 161-173.
- Brewerton, T. (1988). Serotonin dysregulation in bulimia nervosa: Relationship to anorexia nervosa, affective illness, and migraine. In Maryland: <u>National Institute of Mental Health</u>.

- Brewerton, T. D., Hand, L. D., Bishop, E. R. (1993). The Tridimensional Personality Questionaire in eating disorder patients. <u>International Journal of Eating Disorders</u>, 14(2), 213-218.
- Buckholtz, N., George, D., Davies, A., Jimerson, D., & Potter, W. (1988). Lymphocyte beta-adrenergic receptor modification in bulimia. Archives of General Psychiatry, 45, 479-482.
- Bulik, C. M. (1987). Drug and alcohol abuse by bulimic women and their families. <u>American Journal of Psychiatry</u>, 144, 1604-1606
- Bulik, C. M. & Brinded, E. C. (1993). The effect of food deprivation on alcohol consumption in bulimic and control women. <u>Addiction</u>, 88, 1545-1551.
- Bulik, C. M. & Brinded, E. C. (1994). The effect of food deprivation on the reinforcing value of food and smoking in bulimic and control women. <u>Physiology and Behavior</u>, 55, 665-672.
- Caplan, B., & Schechter, J. (1990). Clinical applications of the matching familiar figures test: Impulsivity vs. unilateral neglect. <u>Journal of Clinical Psychology</u>, 46(1), 60-67.
- Classen, W., & Fritze, J. (1989). Ventricular size, cognitive and psychomotor performance, and laterality in schizophrenia. Psychiatry Research, 29, 267-269.
- Coccaro, E., Harvey, P., Kupsaw-Lawrence, E., Herbert, J., & Bernstein, D. (1991). Development of neuropharmacologically based behavioral assessments of impulsive aggressive behavior. <u>Journal of Neuropsychiatry</u>, 3(2), 544-551.
- Coppen, A., Gupta, R., Eccleston, E., Wood, K., Wakeling, A., & de Sousa, V. (1976). Plasma-tryptophan in anorexia nervosa. <u>The Lancet</u>, 1, 961.
- Corulla, W. (1988). A further psychometric investigation of the Sensation Seeking Scale Form-V and its relationship to the EPQ-R and the I.7 Impulsiveness Questionnaire. Personality and Individual Differences, 9(2), 277-287.

- de Silva, P. & Eysenck, S (1987). Personality and addictiveness in anorexic and bulimic patients. <u>Personality and individual differences</u>, 8(5), 749-751.
- De Witt, E. D., Ryan, C., George, L. K. (1985). Learning deficits in adolescents with anorexia nervosa. <u>Journal of Nervous and Mental Disease</u>, 173(3), 182-184.
- Elliott, P., Walsh, D., Close, S., Higgins, G., & Hayes, A. (1990). Behavioural effects of serotonin agonists and antagonists in the rat and marmoset. Neuropharmacology, 29(10), 949-956.
- Eysenck, H. (1983). A biometrical-genetical analysis of impulsive and sensation seeking behavior. In M. Zuckerman (Eds.), <u>Biological Bases of Sensation Seeking</u>, <u>Impulsivity</u>, and <u>Anxiety</u> (pp. 1-36). Hillsdale: Lawrence Erlbaum Associates.
- Eysenck, H. (1987). The place of anxiety and impulsivity in a dimensional framework. <u>Journal of Research in Personality</u>, 21, 489-492.
- Fowles, D. (1987). Application of a behavioral theory of motivation to the concepts of anxiety and impulsivity. <u>Journal of Research in Personality</u>, 21, 417-435.
- Fox, C. F. (1981). Neuropsychological correlates of anorexia nervosa. <u>International Journal of Psychiatry in Medicine</u>, 11(3), 285-290.
- Fuller, R. (1986). Pharmacologic modification of serotonergic function: Drugs for the study and treatment of psychiatric and other disorders. <u>Journal of Clinical Psychiatry</u>, 47(4), 4-8.
- Garfinkel, P., Warsh, J., & Stancer, H. (1979). Depression: New evidence in support of biological differentiation. <u>American Journal of Psychiatry</u>, 136(4B), 535-539.
- Garfinkel, P. E. Moldofsky., M. D. Garner, D. M. (1980). The heterogeniety of anorexia nervosa. <u>Archives of General Psychiatry</u>, 37, 1036-1040.
- Goldbloom, D., Hicks, L., & Garfinkel, P. (1990). Platelet serotonin uptake in bulimia nervosa. <u>Biological Psychiatry</u>, 28, 644-647.

- Grebb, J., Yingling, C., & Reus, V. (1984). Electrophysiologic abnormalities in patients with eating disorders. <u>Comprehensive Psychiatry</u>, 25(2), 216-224.
- Gwirtsman, H., Roy-Byrne, P., Yager, J., & Gerner, R. (1983). Neuroendocrine abnormalities in bulimia. <u>American Journal of Psychiatry</u>, 140(5), 559-563.
- Halmi, K. A. Goldberg, S. C. & Ekcker, E. (1977). <u>Pretreatment</u> Evaluation in Anorexia Nervosa. New York: Raven Press.
- Hamsher, K. de S., Halmi, K.A. & Benton, A.L. (1981). Prediction of outcome in anorexia nervosa from neuropsychological status.

 <u>Psychiatry Research</u>, 4, 79-88.
- Hatsukami, D., Mitchell, M.D., Eckert, E.D. and Pyle, R. (1986).

 Characteristics of patients with bulimia only, bulimia with affective disorder, and bulimia with substance abuse problems.

 Addictive Behaviors, 11, 399-406.
- Heilbrun, A. J., & Bloomfield, D. (1986). Cognitive differences between bulimic and anorexic females: Self-control deficits in bulimia.

 <u>International Journal of Eating Disorders</u>, 5(2), 209-222.
- Jelsma, O., & Van Merrienboer, J. (1989). Contextual interference: Interactions with reflection-impulsivity. <u>Perceptual and Motor Skills</u>, 68, 1055-1064.
- Jimerson, D., Lesem, M., Kaye, W., & Brewerton, T. (1992). Low serotonin and dopamine metabolite concentrations in cerebrospinal fluid from bulimic patients with frequent binge episodes. <u>Archives of General Psychiatry</u>, 49, 132-138.
- Jimerson, D., Lesem, M., Kaye, W., Hegg, A., & Brewerton, T. (1990). Eating disorders and depression: Is there a serotonin connection? <u>Biological Psychiatry</u>, 28, 443-454.
- Jones, B. P. Duncan, C. C. Brouwers, P. & Mirsky, A. F. (1991). Cognition in eating disorders. <u>Journal of Clinical and Experimental</u>
 <u>Neuropsychology</u>, 13(5), 711-728.

- Kaplan, A. S. W.& Woodside, D. B. (1987). Biological aspects of anorexia nervosa and bulimia nervosa. <u>Journal of Consulting and Clinical Psychology</u>, 55(5), 645-653.
- Kaye, W., Ballenger, J., Lydiard, R., Stuart, G., Laraia, M., O'Neil, P., Fossey, M., Stevens, V., Lesser, S., & Hsu, G. (1990). CSF monoamine levels in normal-weight bulimia: Evidence for abnormal noradrenergic activity. <u>American Journal of Psychiatry</u>, 147(2), 225-229.
- Kaye, W., Ebert, M., Gwirtsman, H., & Weiss, S. (1984). Differences in brain serotonergic metabolism between nonbulimic and bulimic patients with anorexia nervosa. <u>American Journal of Psychiatry</u>, 141, 1598-1601.
- Kaye, W., Jimerson, D., Lake, C., & Ebert, M. (1985). Altered norepinephrine metabolism following long-term weight recovery in patients with anorexia nervosa. <u>Psychiatry Research</u>, 14, 333-342.
- Kaye, W., & Weltzin, T. (1991). Neurochemistry of bulimia nervosa. <u>Journal of Clinical Psychiatry</u>, 52(10), 21-28.
- Kent, T., Candace, S., Bryant, S., Barratt, E., Felthous, A., & Rose, R. (1988). Blood platelet uptake of serotonin in episodic aggression: Correlation with red blood cell proton T1 and impulsivity. Psychopharmacology Bulletin, 24(3), 454-457.
- Knight, R. G. Waal-Manning, H. J. & Spears, G. F. (1983). Some norms and reliability data for the Stait-Trait Anxiety Inventory and the Zung Self-Rating Depression Scale. <u>British Journal of Clinical</u> <u>Psychology</u>, 22, 245-249.
- Lacey, J. H. &. Evans, D. H. (1986). The impulsivist: a multi-impulsivist personality disorder. <u>British Journal of Addiction</u>, 81, 641-649.
- Laessle, R., Schweiger, U., & Pirke, K. (1988). Depression as a correlate of starvation in patients with eating disorders. <u>Biological Psychiatry</u>, 23, 719-725.
- Laessle, R. G. B., S. Hank, G. Hahlweg, K. Pirke, K.M. (1990). Cognitve performance in patients with bulimia nervosa: Relationship to intermittent starvation. <u>Biological Psychiatry</u>, 27, 549-551.

- Lezak, M. D. <u>Neuropsychological Assessment</u> (2nd. ed.) Oxford University Press, New York, 1983.
- Li, T., Lumeng, L., McBride, W., & Murphy, J. Alcoholism: Is it a model for the study of disorders of mood and consummatory behavior?

 <u>Annals New York Academy of Sciences</u>, 239-249.
- Maxwell, J. K., Tucker, D.M., Townes, B.D. (1984). Asymmetric cognitive function in anorexia nervosa. <u>International Journal of Neuroscience</u>, 24, 37-44.
- McElroy, S., Keck, P., Harrison, G., & Hudson, J. (1989). Pharmacological treatment of kleptomania and bulimia nervosa. <u>Journal of Clinical Psychopharmacology</u>, 9(5), 358-360.
- McKay, S. E., Humphries, L. L., Allen, M. E. Clawson, D. R. (1986). Neuropsychological test performance of bulimic patients. <u>International Journal of Neuroscience</u>, 30, 73-80.
- Mitchell, J., & Bantle, J. (1983). Metabolic and endocrine investigations in women of normal weight with the bulimia syndrome.

 <u>Biological Psychiatry</u>, 18(3), 355-365.
- Morley, J. (1989). An approach to the development of drugs for appetite disorders. Neuropsychobiology, 21, 22-30.
- Moses, J. A. Maurish, M. E. (1988). A critical review of the Luria-Nebraska Neuropsychological Battery literature: V. Cognitive deficit in miscellaneous psychiatric disorders. <u>International Journal of Clinical Neuropsychology</u>, 10(2), 63-73.
- Netter, P., & Rammsayer, T. (1989). Serotoninergic effects on sensory and motor responses in extraverts and introverts. <u>International Clinical Psychopharmacology</u>, 4(1), 21-26.
- Norman, R., & Jongerius, J. L. (1985). Apple Picker: Computer software for studying human responding on concurrent and multiple schedules. <u>Behavioral Research Methods Instruments and Computers</u>, 17.

- Pirke, K., Fichter, M., Schweiger, U., Fruth, C., Streitmatter, A., & Wolfram, G. (1987). Gonadotropin secretion pattern in bulimia nervosa. <u>International Journal of Eating Disorders</u>, 6(5), 655-661.
- Rau, J., & Green, R. (1975). Compulsive eating: A neuropsychologic approach to certain eating disorders. <u>Comprehensive Psychiatry</u>, 16(3), 223-231.
- SAS Institute Inc. (1994). <u>IMP Users Guide (Version 3</u>). Cary, NC: SAS Institute, Inc.
- Silverstein, A. (1982). Two- and four-subtest short forms of the Wechsler Adult Intelligence Scale-Revised. <u>Journal of Consulting</u> and <u>Clinical Psychology</u>, 50(3), 415-418.
- Smith, M. C. & Thelen, M. H. (1984) Development and validation of a test for bulimia. <u>Journal of Consulting and Clinical Psychology</u>, 52, 863-872
- Sohlberg, S. N., Norring, C., Holmgren, S. & Rosmark, B. (1989). Impulsivity and long-term prognosis of psychiatric patients with anorexia nervosa/bulimia nervosa. <u>Journal of Nervous and Mental Disease</u>, 177(5), 249-258.
- Soubrie, P. (1986). Reconciling the role of central serotonin neurons in human and animal behavior. The Behavioral and Brain Sciences, 9, 319-364.
- Strauss, J., & Ryan, R. (1988). Cognitive dysfunction in eating disorders. International Journal of Eating Disorders, 7(1), 19-27.
- Szmukler, G. I. Andrews, D., Kingston, K., Chen, L., Stargatt, R., Stanley, R. (1992). Neuropsychological impairment in anorexia nervosa: Before and after refeeding. <u>Journal of Clinical and Experimental Neuropsychology</u>, 14(2), 347-352.
- Thompson, J. K. & Spana, R. E. (1991). Visuospatial ability, accuracy of size estimation and bulimic disturbance in an noneating disordered college sample: A neuropsychological analysis.

 <u>Perceptual and Motor Skills</u>, 73, 335-338.

- Touyz, S. W. Beaumont, P. J. V. and Johnstone, L. C. (1986).

 Neuropsychological correlates of dieting disorders. <u>International Journal of Eating Disorders</u>, 5(6), 1025-1034.
- Vandereycken, W. &. Pierloot, R. (1983). The significance of subclassification in anorexia nervosa: a comparative study of clinical features in 141 patients. <u>Psychological Medicine</u>, 13, 543-549.
- Weiss, S. R. & Ebert, S. (1983). Psychological and behavioural characteristics of normal-weight bulimics and normal weight controls. <u>Psychosomatic Medicine</u>, 45(4), 293-303.
- Wu, J., Hagman, J., Buchsbaum, M., Blinder, B., Derrfler, M., Tai, W., Hazlett, E., & Sicotte, N. (1990). Greater left cerebral hemispheric metabolism in bulimia assessed by positron emission tomography. <u>American Journal of Psychiatry</u>, 147(3), 309-312.
- Zuckerman, M. (1983). A biological theory of sensation seeking. In M. Zuckerman (Eds.), <u>Biological Bases of Sensation Seeking</u>, <u>Impulsivity</u>, and <u>Anxiety</u> (pp. 37-75). Hillsdale: Lawrence Erlbaum Associates.

Appendix 1.1

B.I.S.

DIRECTIONS: People diff	er in the ways they	y act and think in	ID# 1 - 1 - different situations. This
is a survey that measures statement and place in th			and think. Read each
1 = Rarely/Never 2 = Occasionally		3 = Often 4 = Almos	t Always/Always
Do not spend too much tinand honestly.	ne on any statemen	t. Answer quickly	1
		Rarely/Never Occasionally	
1. I plan tasks carefull	у		
2. I do things without	thinking		
3. I am happy-go-luck	y		
4. I have "racing" the	oughts		
5. I plan trips well ahea	ad of time	. 	
6. I am self-controlled.		· · · · · · · · · · · · · · · · · · ·	
7. I concentrate easily.	· · · · · · · · · · · · ·		
8. I save regularly		· · · · · · · · · · · · · · · · · · ·	
9. I find it hard to sit s			
10. I am a careful think			
11. I plan for job securi			
12. I say things withou13. I like to think about			

		1 = Rurely/Never 2 = Occasionally	3 = Often 4 = Almost	Always/Always
14.	I change jobs			17
15.	I act "on impulse"			. 📙
16.	I get easily bored when solving	g thought problems		. 🔲
17.	I have regular medical/dental c	check ups		. 🔲
18.	I act on the spur of the moment.	.		. 📙
19.	I am a steady thinker			. 🔲
20.	I change where I live			
21.	I buy things on impulse			. []
22.	I finish what I start			
23.	I walk and move fast			
24.	I solve problems by trial-and-e	error		
25.	I spend or charge more than I	earn		
26.	I talk fast			
27.	I have outside thoughts when	thinking		
28.	I am more interested in the pro-	resent than the future		
29.	I am restless at lectures or talks	s		
30.	I plan for the future			
five transfer				

Appendix 1.2

		BDI	7
		ID#]
have stat app	efully. e bee emen ly eq	questionnaire are groups of statements. Please read each group of state Pick the one statement in each group which best describes the way en feeling the past week; including today. Place the number of the you picked in the box. If several statements in the group seem ually well, circle each one. Be sure to read all the statements in each or each all the statements in each or each or each or each or each or each all the statements in each or e	you B (Ö
1.	0	I do not feel sad	
	1 2 3	I feel sad. I am sad all the time and I can't snap out of it I am so sad or unhappy that I can't stand it	4
2.	0 1 2 3	I am not particularly discouraged about the future. I feel discouraged about the future. I feel I have nothing to look forward to I feel that the future is hopeless and that things cannot improve	
3.	0 1 2 3	I do not feel like a failure. I feel I have failed more than the average person. As I look back on my life, all I can see is a lot of failures I feel I am a complete failure as a person	
4.	0 1 2 3	I get as much satisfaction out of things as I used to I don't enjoy things the way I used to. I don't get real satisfaction out of anything anymore I am dissatisfied or bored with everything	
5.	0 1 2 3	I don't feel particularly guilty. I feel guilty a good part of the time. I feel quite guilty most of the time I feel guilty all of the time	
6.	0 1 2 3	I don't feel I am being punished I feel I may be punished. I expect to be punished I feel I am being punished	
7.	0 1 2 3	I don't feel disappointed in myself I am disappointed in myself. I am disgusted with myself I hate myself	
8.	0 1 2 3	I don't feel I am any worse than anybody else I am critical of myself for my weaknesses or mistakes. I blame myself all the time for my faults I blame myself for everything had that happens	
9.	0 1 2	I don't have any thoughts of killing myself I have thoughts of killing myself, but I would not carry them out. I would like to kill myself	

10.	0 1 2 3	I don't cry any more than usual. I cry more now that I used to. I cry all the time now I used to be able to cry, but now I can't cry even though I want to.
11.	0 1 2 3	I am no more irritated now than I ever am. I get annoyed or irritated more easily than I used to. I feel irritated all the time now I don't get irritated at all by the things that used to irritate me
12.	0 1 2 3	I have not lost interest in other people. I am less interested in other people than I used to be. I have lost most of my interest in other people I have lost all of my interest in other people
13.	0 1 2 3	I make decisions about as well as I ever could. I put off making decisions more than I used to. I have greater difficulty in making decisions than before I can't make decisions at all anymore
14.	0 1 2 2 3	I don't feel I look any worse than I used to I am worried that I am looking old or unattractive. I feel that there are permanent changes in my appearance that 'make me look unattractive. I believe that I look ugly
15.	0 1 2 3	I can work about as well as before. It takes an extra effort to get started at doing something. I have to push myself very hard to do anything. I can't do any work at all
16.	0 1 2 3	I can sleep as well as usual. I don't sleep as well as I used to. I wake up 1-2 hours earlier than usual and find it hard to get back to sleep. I wake up several hours earlier than I used to and cannot get back to sleep.
17.	0 1 2 3	I don't get more tired than usual. I get tired more easily than I used to. I get tired from doing almost anything. I am too tired to do anything.
18.	0 1 2 3	My appetite is no worse than usual. My appetite is not as good as it used to be. My appetite is much worse now. I have no appetite at all anymore
19.	0 1 2 3	I haven't lost much weight, if any, lately. I have lost more than 5 pounds.(about 2.2 kilograms) I have lost more than 10 pounds.(about 4.5 kilograms) I have lost more than 15 pounds.(about 6.8 kilograms)
		m purposely trying to lose more by eating less. Yes 2 = No

20.	1	I am worried about physical problems such as aches and pains; or upset stomach; or constipation.	
	2	I am very worried about physical problems and it's hard to think of much else.	
	3	I am so worried about my physical problems that I cannot think about anything else	2
21.		I have not noticed any recent change in my interest in sex.	
	I	I am less interested in sex than I used to be.	
	2	I am much less interested in sex now.	1 1
	3	I have lost interest in sex completely	
	· ·	Monthus	

:

Appendix 1.3 The Stroop: Card A

SLUE RED BLUE GREEN BLUE GREEN RED BLUE GREEN RED

ED GREEN BLUE GREEN RED BLUE RED GREEN RED GREEN BLUE GREEN BLUE RED GREEN Q.F RED **BLUE GREEN** RED ED **GREEN** BLUE RED GREEN RED BLUE **GREEN** RED ED GREEN BLUE GREEN RED BLUE GREEN RED BLUE **GREEN** }ED GREEN RED BLUE RED GREEN BLUE GREEN BLUE RED GREEN RED BLUE RED **BLUE** RED GREEN BLUE RED **GREEN** BLUE RED BLUE BLUE RED GREEN RED BLUE GREEN BLUE GREEN BLUE RED BLUE RED GREEN RED BLUE GREEN RED GREEN RED BLUE GREEN **RED BLUE GREEN** RED BLUE RED BLUE GREEN RED BLUE RED GREEN BLUE RED

The Stroop: Card B

RED GREEN BLUE GREEN RED BLUE RED GREEN RED GREEN

BLUE GREEN RED BLUE RED GREEN BLUE RED BLUE RED RED GREEN RED RED BLUE GREEN BLUE BLUE GREEN RED RED GREEN BLUE RED GREEN RED **BLUE** GREEN RED GREEN RED BLUE GREEN **BLUE** GREEN RED **BLUE** GREEN GREEN RED RED GREEN BLUE GREEN RED GREEN BLUE RED BLUE GREEN BLUE GREEN BLUE RED BLUE RED GREEN BLUE RED RED BLUE RED GREEN BLUE RED BLUE GREEN GREEN BLUE **BLUE** GREEN BLUE RED BLUE GREEN RED RED BLUE GREEN BLUE GREEN BLUE RED GREEN RED BLUE GREEN RED RED BLUE GREEN RED BLUE GREEN BLUE RED BLUE

