COGNITIVE FUNCTIONING IN ANOREXIA AND BULIMIA.

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Cognitive Functioning in Anorexia and Bulimia.

I declare that the study described herein is my own composition.

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This study compared cognitive test performance between matched controls and participants with anorexia (N=30) and bulimia (N=30). The results revealed that both of the eating disorder groups demonstrated a considerable number of neuropsychological deficits. However, between-group significance was not established and the deficits were accounted for by a small subgroup of poorer test performers. On analysing group results on a wide range of cognitive tests, both clinical groups performed more poorly than controls on attentional, learning and visuo-spatial/constructional tasks. There was however, no evidence of executive difficulties in either group. On partialling-out the effects of anxiety and depression, between-group significance was removed on a considerable proportion of the test results. However, significance remained for the anorexia group on a digit coding task, two auditory-verbal learning tests, a measure of incidental visual learning and a measure of visuo-spatial/constructional functioning. These results suggested mild difficulties in cognitive functioning across neuropsychological domains which were not fully explained by the influence of emotional and other psychological factors.

In the bulimia group, significance remained on a digit coding task and an omnibus attentional measure together with two dependent measures of auditory-verbal learning. There was also a trend for poorer performance on tests of visuo-spatial/constructional functioning. However, the results were largely accountable by the effects of emotional variables on attentional processes.

The results of cognitive test performance in anorexia and bulimia were discussed with reference to previous studies and with reference to clinical practice.
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INTRODUCTION.

Anorexia nervosa is a complex eating disorder in which the individual refuses to maintain body weight at 85 per cent or above the expected level for age and height; or fails to achieve appropriate weight gain during a period of growth. A body mass index of 17.5 or below is a diagnostic feature of the disorder and amenorrhoea a feature in post-menarcheal females. The core psychopathology of anorexia is the presence of overvalued ideation concerning the individual's body shape and weight. Typically, a morbid fear of becoming fat is expressed in association with a drive for thinness. Binge eating and compensatory purging behaviours may occur in 50 per cent of cases and excessive exercise is a common mode of additional weight control (Fairburn, 1993).

The heterogeneity of anorexia nervosa is recognised by the inclusion of two subtypes in DSM IV. A restricting type of anorexia is characterised by dietary restraint without regular binge eating or purging behaviours; whereas a binge-eating / purging type of anorexia is characterised by regular binge eating and purging. The latter includes self-induced vomiting, the use of laxatives and diuretics. The subtypes of anorexia are differentially associated with personality and affective variables (Casper, Eckert, Halmi, Goldberg & Davies, 1980). For example, in comparison to the relative stability and self-control of the restricting subtype of anorexia; the binge / purging form has been associated with increased impulsivity, anxiety, depression, substance abuse, deliberate self-harm and suicide. Binge-purging anorexia is also associated with a poorer treatment outcome (Casper et al, 1980; Vitousek and Manke, 1994). However, such characteristics may also be viewed along a continuum.

Anorexia is also associated with obsessive-compulsive symptomatology (Solymon, Freman & Miles, 1982; Jarry and Vaccarino, 1996) and may share underlying serotonergic dysfunction with obsessive-compulsive disorder (Hsu, Kaye & Weltzin, 1993). Cognitive processing in anorexia is associated with a personality-perceptual style which has been described as lacking both 'ego' development and Piagetian 'formal operational thinking' (Garfinkel and Garner, 1982). However, immaturity of thought as a testable hypothesis has not been established (Swift, Camp, Bushnell & Bergman, 1984; Kowalski, 1986) although cognitive distortion and ego dysfunction are apparent in anorexia. For example, there is considerable evidence of reality distortions, dichotomous thinking and increased logical errors in anorexia nervosa when compared with control groups and normal weight individuals with bulimia nervosa (Small, Madero, Teagno & Ebert, 1983; Strauss and Ryan, 1988; Butow, Beumont & Touyz, 1993).

The annual incidence of anorexia ranges from 0.234 to 14.6 per 100,000 of the female population and the disorder is relatively rare in males. The onset of anorexia is usually in adolescence with a later onset being associated with poorer outcome. Up to 50 per cent of anorexics may make a full recovery although there is a widespread variation in the rates of recovery and there is no consensus as to the most effective treatment regime (Fairburn, 1993; Gillberg, Rastam & Gillberg, 1994). The disorder may be intractable in 20 per cent of cases and co-exists with depression and other
psychopathology (Solyom, Freman & Miles, 1982). The mortality rate is high with a range of 15 - 20 per cent due to both suicide and the severe medical complications which accompany the disorder (Ratnasuriya, Eisler, Szmuckler & Russell, 1991). Nutritional status is inevitably compromised and metabolic disturbances are particularly common in those anorexics with vomiting and other purging behaviours (Treasure and Szmuckler, 1995).

The aetiology of anorexia is generally considered to be multifactorial and includes hypotheses concerning sociocultural influences, individuation-separation conflict, dysfunctional family systems, childhood trauma, genetic vulnerability, obstetric complications at birth and other biological factors (Morley & Blundell, 1988; Fairburn, 1993; Wren & Lask, 1993; Gillberg & Rastan, 1998; Cnattingius, Hultman, Dahl & Sparen, 1999). There is support for genetic susceptibility to anorexia and a consensus that anorexia is strongly associated with an endocrine disorder. However, the aetiology remains unclear (Treasure and Szmuckler, 1995; Pieri and Campbell, 1999).

There is consistent evidence of structural brain abnormalities in anorexia nervosa which are typified by enlarged ventricles and widened sulci and which generally normalise on weight restoration (Ellison and Foong, 1998). There is also a limited body of evidence which suggests neuropsychological impairment across several domains of cognitive functioning (Touyz & Beumont, 1994; Treasure and Szmuckler, 1995). However in contrast to the neuropsychology of depression (Crews and Harrison, 1995) and obsessive-compulsive disorder (Tallis, 1995), there are no neuropsychological models of anorexia (Touyz & Beumont, 1994).
Bulimia Nervosa was also described in the 18th century although its nosological status was unclear until relatively recently. The disorder is characterised by 'normal weight' and recurrent episodes of binge eating within a specified time period (an average of two binges per week over a period of at least three months). Binge eating is typically associated with feelings of loss of control in relation to eating behaviours. Bulimia is also diagnosed on the basis of two other cardinal features - overvalued ideation concerning body weight and size, and the excessive control of weight by fasting, exercising or purging.

The existence of subtypes is recognised by DSM IV. - a purging subtype involving regular self-induced vomiting or the use of purgatives and a non-purging subtype involving fasting or excessive exercise as compensatory mechanisms.

Bulimia is also predominantly a disorder of the female population with a prevalence of between one and two per cent in America and the U.K. (Fairburn and Beglin, 1990). Community sample surveys however suggests that the prevalence may be underestimated (Halmi, Falk & Schwartz, 1981). The onset of bulimia is usually later than that of anorexia and peaks at around the age of twenty years. Bulimia may also be associated with physical complications due to purging behaviours and intermittent starvation. Psychiatric comorbidity is high with an increased risk for lifetime prevalence for major depression (Crow, Zander, Crosby & Mitchell, 1995).

Bulimia is associated with personality characteristics of obsessionality and impulsivity. Multiple substance abuse is common and the symptomatology is often secondary to anxiety and dissociative disorders (Fairburn, 1993; Everill, Waller & MacDonald, 1995). The treatment outcome is optimistic in the short-term, although
chronicity and a high incidence of relapse are common (Keller, Herzog, Lavori, Bradburn & Mahoney, 1991). The aetiology of bulimia nervosa is generally unclear. Up to 25 per cent of bulimics may have previously suffered an anorexic episode and premorbid weight is reported as having been overweight in many cases. There is limited evidence of a seasonal affective disorder (Gruber & Dilsaver, 1996) and of familial factors related to increased prevalence of depression and substance abuse in relatives (Fairburn, 1993). There is evidence of childhood trauma with hypothesised relationships to purging behaviours (Waller, 1992; Connon & Treasure, 1998; Brewerton, Dansky, Kilpatrick & O'Neil, 1999).

That hyperphagia has been found in neurological conditions and ventromedial hypothalamic and frontal lobe lesions, has been postulated as providing some evidence of organic aetiology in bulimia nervosa (Erb, Gwirtsman, Fuster & Richeiner, 1989). There is also evidence of structural brain changes which are similar to those found in anorexia and which are therefore not attributable to body weight (Touyze & Beumont, 1994). Such structural changes are proposed to be a result of the starvation-binge-purging cycle rather than a precursor to the disorder (Lankenau, Swigar & Bhimani et al. 1985).

It is the neuropsychological dimension to both anorexia nervosa and bulimia nervosa which is the focus of the present study. The literature will be reviewed and preceded by an outline of the relevant neurobiological factors in eating disorders.
PART 1: NEUROBIOLOGICAL CORRELATES OF EATING DISORDERS.

1a) EEG and epileptiform phenomena in anorexia.

Anorexia is associated with a generalised slowing of the dominant frequency and dysrhythmia in EEG recordings. The occurrence of such abnormalities is however variable with both high and low frequencies reported (Braun & Chouinard, 1992). Rothenberger, Blanz and Lehmkuhl (1995) reported that 56 per cent of a sample of adolescents with anorexia demonstrated abnormal electrical brain activity when compared with 15 per cent of a control group comprising emotionally disturbed adolescents. The results however failed to establish correlations with either Body Mass Index or CT brain scan findings. Miyamoto, Sakuma, Kumagai, Ichikawa and Koizumi (1992) investigated auditory brain stem responses in twenty participants with anorexia nervosa and ten controls. The results found significantly smaller amplitude ratios in those with anorexia and the authors concluded that "some dysfunction might exist in the region of the brain stem" (page 673).

Where epileptiform phenomena are observed, the abnormal activity is usually associated with electrolyte imbalance due to purging behaviours. However, the incidence of epileptiform activity in anorexia is apparently rare and the causal factors are uncertain (Kohlmeyer, Lehmkuhl & Poutsha, 1983). Signer and Benson (1990) suggest that anorexia is caused by a primary dysfunction of the limbic system. This speculation is based upon only three cases of anorexia which demonstrated body image delusions and affective psychosis following temporal lobe epilepsy. Similarly, based on a meta-analysis of single-case studies, Braun and Chouinard (1992) suggest
that a predominance of right-sided temporal-occipital foci is evidence for right hemispheric dysfunction in anorexia. However, Artman, Grau, Adelmann and Schleiffer (1985) found that only six per cent of a group of participants with anorexia demonstrated such focal abnormalities. Kohlmeyer, Lehmkuhl and Poutska (1983) have also noted that the slight EEG abnormalities evident in the majority of a sample of teenagers with anorexia, were not present however after refeeding. The participants in the latter study were nutritionally stable and demonstrated normal blood chemistry. In Bradley, Taylor, Rovet, Goldberg, Hood, Wachsmuth, Azcue and Pencharz’s (1997) controlled study however, abnormalities of event related potentials only partially normalised following weight restoration in 20 adolescents with anorexia. The abnormalities in Bradley et al’s (1997) study were localised to the central parietal region and correlated with cognitive inefficiency on specific verbal and nonverbal cognitive tasks. However, cognitive inefficiency was not evident on a wider battery of cognitive tests and the clinical significance of Bradley et al’s (1997) results is not clear. Such evidence appears to suggest that where EEG abnormalities or epileptiform phenomena exist in anorexia, the phenomena are generally consequential to rather than as a precursor of the disorder. In most cases, the abnormalities appear to resolve on weight restoration.

1b) Neuroimaging studies in anorexia.

A considerable number of computed tomography (CT) and magnetic resonance imaging (MRI) studies have reported enlarged external cerebral spinal fluid (CSF) spaces, ventricular enlargement and loss of viscera (parenchyma) in anorexia nervosa (Enzman and Lane, 1977; Lankenau, Swigar, Bhimani, Luchins & Quinlan, 1985;
Kreig, Bachmund & Pirke, 1986; Palazidou, Robinson & Lishman, 1990; Kingston, Szmukler, Andrews, Tress & Desmond, 1996). Whilst the majority of the early studies were not controlled or reported on small numbers of participants, these findings have been confirmed in fairly large controlled studies. For example, Kreig et al (1986) found that 50 per cent of a group of participants with anorexia demonstrated slight enlargement, with a further 36 per cent demonstrating marked enlargement of external CSF spaces. Artman, Grau, Adelmann and Sclieiffer (1985) also reported an association between anorexia and sulci widening with 90 per cent of those with anorexia demonstrating sulci widening and 49 per cent demonstrating cerebellar atrophy. There is also limited evidence of reduced thalamus and mid-brain areas in comparison with both individuals with bulimia nervosa and normal controls (Husain, Black, Doraiswamy, Shah, Rockwell, Ellinwood & Krishnanet, 1992) and smaller pituitary gland size in anorexia when compared to control groups (Doraiswamy, Krishnan, Figeil, Husain, Boyko, Rockwell & Ellinwood, 1990). Lambe, Katzman, Mikulis, Kennedy and Zipursky (1997) found greater CSF volumes and smaller grey matter volumes in 12 participants with early-onset anorexia who were 'weight-recovered'. Whilst the results of Lambe et al's (1997) study suggests irreversible structural brain changes in anorexia, some of the participants continued to have a relatively low Body Mass Index. Further, the inclusion of children in the study introduced the confounding variable of maturation effects. Other studies have found that that structural abnormalities changes found in the underweight state may normalise following weight restoration in a considerable number of individuals with anorexia (Herzholz, Kreig, Emrich, Pawli, Beil, Pirke, Pahl, Wagner, Wienhard, Ploog & Heiss 1987: Kingston et al, 1996, Golden, Astari, Kohn, Patel, Jacobson, Fletcher & Shenker, 1996). For example, Golden et al's (1996) re-feeding study of
twelve female adolescents with anorexia, found cerebral ventricular enlargement to correlate with the degree of malnutrition and the abnormalities were resolved at one year post-weight gain.

The possibility of caudate nucleus dysfunction in anorexia nervosa has been postulated as representing an organic link with obsessive-compulsive disorder, although the evidence from functional imaging studies is limited and contradictory. Herholtz, Kreig, Emrich et al (1987) and Kreig, Hotloff, Schreiber, Pirke and Herholz (1991) both reported caudate nuclei hypermetabolism involving studies of five and seven participants respectively. In contrast, Delevenne, Lotstra, Goldman, Biver, De Maerttelauer, Appelboom-Fondu, Schoutens, Bidaut, Luxen and Mendlewicz (1995) found significant global glucose hypometabolism in twenty underweight participants with anorexia, particularly in the frontal and parietal areas. No significant correlations were found however between regional cerebral glucose metabolism, Body Mass Index and affective variables. Similarly, Rastam, Bjure & Vestegen, Uvebrant and Gillberg (1998) found hypofrontality and hypoperfusion in the parietal lobes with some degree of hypoperfusion of temporal regions in weight restricting anorexia. The possibility of hypometabolism in parietal areas may have correlates with neuropsychological evidence of a visuospatial deficit in anorexia. (Kingston et al,1996). However, the results of studies which have combined neuroradiological measures with cognitive tests have generally failed to establish associations between scan or imaging abnormalities and test performance (Laessle, Kreig, Fichter, & Pirke,1989; Palazidou, Robinson & Lishman,1990, Kingston et al, 1996). Whilst, Palazidou et al (1996) found a negative correlation between enlarged external CSF spaces and poorer performance on a perceptual-motoric coding task in anorexia, the functional significance of this finding is unclear.
There have been several hypotheses suggested to explain the general finding of radiological brain abnormalities in anorexia. These include dehydration, loss of intercellular protein, the presence of elevated cortisol, gliosis and perinatal brain damage (Ellison & Foong, 1998). The last hypothesis is based upon limited evidence of increased obstetric complications in anorexia at birth (Halmi, 1984; Artman, Grau, Adelmann & Schleiffer, 1985; Cnattingius, Hultman, Dahl & Sparen, 1999). Halmi (1984) reported associations in anorexia between perinatal injury and older parental age, and further relationships with both low and high birth weights. Artman et al (1985) attributed the abnormalities found in a CT brain scan study of anorexia to early acquired brain lesions. However, the relationship between obstetric complications at birth and brain abnormalities remains unclear. Perinatal injury has been excluded from some studies and is not reported in others. Where perinatal injury is reported as a significant factor however, no significant correlations have been established between the number of obstetric complications at birth and neuropsychological test performance (Hamsher, Halmi and Benton, 1981). For example, Lankenau, Swigar & Bhimai, Luchins and Qunlan (1985) found that whilst 36 per cent of participants with anorexia were found to have a history of CNS insult, no significant relationship was evident between perinatal complications and CT brain abnormalities.

1c) Neuroimaging studies in bulimia.

Some of the structural changes observed in anorexia have also been found in normal weight bulimics (Kreig, Bachmund and Pirke, 1987; Kreig, Lauer & Pirke, 1989; Hoffman, Ellinwood, Rockwell et al, 1990). For example, Kreig et al (1987),
found that over one third of participants with bulimia nervosa demonstrated enlarged external CSF spaces; although 40 per cent of the sample had a history of anorexia. The latter participants were observed to have more frequent sulci widening than those without a history of anorexia, although there was no evidence of higher rates of abnormal ventricular brain ratio (VBR). These findings were confirmed in a later study by Kreig et al. (1989), who suggested that the abnormal VBR's and sulci widening found in eating disorders may have different aetiologies. Laeselle, Fichter, Pirke & Kreig (1989) compared eating disorder groups with matched controls and found that 27 per cent of those with bulimia nervosa and 76 per cent of those with anorexia demonstrated abnormal VBR's. Both clinical groups also performed significantly poorer than the control group on a vigilance task. No correlations were established however, between VBR and test performance although a subgroup of those with bulimia nervosa and metabolic disturbance were found to produce the lowest scores on the vigilance task.

In functional imaging studies of bulimia nervosa, there is limited evidence of reduced right hemispheric metabolism, frontal lobe changes and temporal hypermetabolism (Wu, Hagman, Buchsbaum, Blinder, Derrfler, Tai, Hazlett & Sicotte, 1990; Ellison & Foong, 1998). Wu et al's (1990) controlled PET scan study involving a vigilance task, found that hemispheric equivalence in metabolism was evident for those participants with bulimia nervosa. In contrast, the participants in the control group demonstrated increased right hemispheric metabolism during a vigilance task. Reduced right hemispheric metabolism in bulimia was also reported by Hagman, Buchsbaum, Rao, Reynolds and Blinder (1990), lending some support perhaps to a right hemispheric dysfunction hypothesis in eating disorders. However,
Andreason, Altemus, Zametkin and King (1992) found bulimia nervosa to be associated with increased temporal metabolism in both hemispheres.

In conclusion, the 'brain mapping' of bulimia nervosa is far from definitive and several studies have demonstrated equivocal or contradictory results.

The causal explanations for the radiological brain abnormalities found in bulimia nervosa are hypothesised to be due to the complex metabolic and endocrine changes which result from both rapid weight loss and purging behaviours. Beatty, Bailly and Fisher's (1989) single-case study of an association between persistent vomiting and amnesia also suggests sub-cortical brain damage and is consistent with empirical evidence of vomiting-induced Korsakoff's syndrome (Lezak, 1995). However, the general effects of purging behaviours on brain morphology have yet to be established. As is the case with anorexia, the functional significance and aetiology of brain abnormalities in bulimia nervosa remains unclear. On weight restoration in anorexia, the normalisation of brain abnormalities is accompanied by normalisation of vasopressin (antidiuretic hormone) release and thus vascular permeability in the brain. A similar process involving vasopressin may be involved in bulimia nervosa (Hoffman, Ellinwood, Rockwell, Herfkens, Nishita & Guthrie, 1989).

**1d) Neuroimaging in eating disorders - summary.**

In summarising the evidence of neuroimaging studies in eating disorders, it is apparent that both anorexia and bulimia nervosa are associated with morphological brain changes in the form of enlarged ventricular spaces and/or sulci widening.
In anorexia, up to 80 per cent of participants in some studies are recorded as showing such abnormalities. However, the relationship between brain abnormality and Body Mass Index is unclear, with some studies reporting inverse associations (Artman, Grau, Adelmann & Scleiffer, 1985; Datloff, Coleman, Forbes & Kreipe 1986; Kingston, Szmuckler, Andrews, Tress & Desmond, 1996) and other studies failing to establish significant relationships (Dolan, Mitchell & Wakeling, 1988; Delevenne et al., 1995).

In bulimia, up to 30 per cent of subjects have been reported as having similar brain structural changes with intermittent starvation or purging behaviours being attributed as the likely causal factors. (Krieg, Backmund & Pirke, 1989). The fact that normal weight bulimics demonstrate similar structural abnormalities to those found in anorexia suggests that body weight is not the primary causal factor.

The functional significance of radiological brain abnormalities in eating disorders remains unclear partly due to methodological questions. In reviewing the literature on neuroimaging in anorexia nervosa, Gillberg and Rastam (1998) conclude that the formulation of coherent hypotheses is lacking due to the scarcity of published studies with sound methodology. Imaging studies may reflect both causal and consequential factors in eating disorders and may not necessarily reflect major pathology (Black and Botterton, 1997; Palmer, 1998). That brain abnormalities may normalise following weight restoration in anorexia, does suggest that the brain changes are a result of the anorexic behaviours rather than a precursor to the disorder. However, some participants with anorexia fail to demonstrate normalisation of brain abnormalities following weight restoration and this appears to suggest that anorexia may be associated with a permanent, albeit mild state of brain damage. An alternative
explanation is that the participants in some studies may not have been fully restored to their premorbid weight. This may have been the case in Kingston et al's (1996) study, in which the BMI's of those participants with weight-restored anorexia remained relatively low.

There is also limited evidence for the presence of perinatal complications as a causal factor in anorexia although this is unclear and may be regarded as a nonspecific precursor to the disorder (Artman, Grau, Adelmann & Schleiffer, 1985). Lambe, Katzman, Mitulis, Kennedy and Zipursky (1997) suggest that where structural brain changes are identified, then those individuals may be more vulnerable with regard to the persistence of anorexia and more susceptible to relapse. However, to date this hypothesis has not been tested.

The brain scanning procedures involved in the above studies are also fraught with various methodological problems. Early CT studies had considerable methodological shortcomings with regard to measurement of ventricular volume (Ellison & Foong, 1998). That ventricular volume appears to be genetically determined also raises questions about interpretation of abnormality (Reveley, Reveley, Chikara & Clifford, 1984). Kingston et al (1996) reported that the MRI abnormalities which were observed in anorexic subjects would have probably been disregarded as abnormal on routine radiological screening. It is also known that such abnormalities may be demonstrated in other psychiatric disorders including schizophrenia (Shenton, Wible & McCarley, 1997); bipolar and psychotic depression (Trimble, 1996); substance abuse and affective disorders (Palazidou, Robinson & Lishman, 1990). Further individuals with Cushing's syndrome and those taking steroids, may have similar structural brain abnormalities (Kreig, Backmund & Pirke, 1987).
Whilst functional imaging studies have the potential to offer powerful insights into brain functioning in eating disorders, the procedures and analyses are highly complex and there are many inconsistencies in methodology and reported results (Brodie 1996; Ellenson & Foong, 1998). Age and gender effects have only been established for whole brain blood flow, making regional comparisons highly problematic (Madhukar, Husain & Devous, 1997). Where regional comparisons have been attempted, most studies have used ratio measures with specific brain structures as a relative proportion of overall brain size. Black and Botterton (1997) argue that this approach has led to spurious conclusions due to normalisation of data based upon the assumption of between-group normality. Black and Botterton (1997) also suggest that the use of qualitative ratings of images (or area measured) are insensitive to the measurement of structural volume and further, are not easily reproducible. There are also potential difficulties for re-scanning procedures where correct head positioning is essential and the thickness of 'slice' is required to be constant (Black and Botterton, 1997).

Brodie (1996) notes that the complex technology involved in PET and SPECT imaging is open to misuse and spurious interpretation of findings. Similarly, whilst acknowledging the potential for functional imaging to reveal insights into psychiatric disorders; Nemeroff, Clinton & Berne (1999) note that -

"There are also risks and limitations: statistical analysis of data from multiple brain regions in a limited number of subjects with the use of multiple comparisons in the absence of priori hypotheses can potentially result in both false positive and false negatives findings that may redirect the field in spurious directions" (page 673).

In anorexia several brain scan studies have used child and adolescent populations with problems in establishing normal control measurements (Kornreich, Shapira,
There is also a serious methodological flaw in studies which include both child and adult participants. A recent functional imaging study by Casey (1999) demonstrates the confound of brain maturation. On comparing children and adults on a response inhibition task, Casey (1999) found that child participants demonstrated an increased activation in specific brain areas when compared to adults; thus suggesting cognitive efficiency in the mature brain. Parks, Loewenstein, Dodrill, Barker, Yoshii, Chang, Emran, Apicella, Shermaya and Duara’s (1988) PET study of normal adults also found a 'cognitive efficiency' factor with participants who scored high on a verbal fluency task, demonstrating the lowest level of cerebral activation. Parks et al (1988) suggest therefore that the relationship between behavioural task and metabolic cerebral activation is likely to be an inverted -U -shaped curve; similar to that found in anxiety related test performance. Similarly Haier, Siegel, Nuechterlein, Hazlett, Wu, Paek, Browning and Buchsbaum (1988) demonstrated that brain glucose rate was inversely correlated with intelligence with total glucose consumption decreasing following training on a computer game. This study also found that the more intelligent participants showed the greatest decrement on total glucose volume after training, a result which underlines the need for IQ matching in between-participant designs.

Such results have considerable implications for both neuropsychological studies and combination studies involving brain scans. To date the latter is limited and concordance between brain scan abnormality and test performance has not been been established (Laeselle, Kreig, Fichter, & Pirke, 1989; Kingston, Szmuckler, Andrews, Tress & Desmond, 1996).
Further problems with brain scan research arise when studies include inappropriate controls or involve participants with a previous history of bulimia. The latter was examined in two studies and the results failed to find any differences on brain scan measures between anorexia and those anorexics with a previous history of bulimia nervosa (Kreig, Backmund & Pirke, 1987; Krieg, Lauer & Pirke, 1989). Thus whilst the results of structural and functional brain scanning in eating disorders have provided insights into potential brain changes in eating disorders, these results have to be viewed with a degree of caution, particularly in the emerging field of brain mapping.

Drevets, Videen, MacLeod, Haller and Raichle (1992) demonstrated how easily false positive findings can occur using PET methodology. The authors cite a PET study of panic disorder by Benkelfat and colleagues (1991) which contrary to previous results, found that the greatest amount of cerebral blood flow observed during panic attack was extra rather than inter-cranial. Drevets et al (1992) explored this further in a PET 'teeth clenching' study and found similar results. The authors concluded that the results of previous PET studies of panic disorder may have been confounded by temporalis and masseter muscle contraction. Similarly, Kertesz (1994) has argued that cerebral blood flow may not be truly representative of the stimulation-inhibition nature of brain processes and therefore the foci of activation in imaging studies may reflect only a part of a complex processing network. Indeed, Vanzeta and Grinvald (1999) recently reported that the brain's initial response to stimuli is highly localised with regard to oxygen activity. They argue that MRI methodology does not measure this initial localised activity and the methodology therefore may have resulted in numerous brain mapping errors.
1e) The effects of starvation on cognitive functioning.

The effects of starvation on cognitive functioning has not been addressed beyond the clinical situation due to the ethical issues involved (Jones, Duncan, Brouwers & Mirsky, 1991). There is evidence from studies of malnourished children that intellectual development is clearly affected by nutritional variables. For example, Gunston, Burkimsher, Malan and Sive (1992) reported significant cerebral atrophy in children with acute Kwashiorkur (protein energy malnutrition) which was demonstrated by widened cortical sulci, enlarged ventricles, widened inter-hemispheric fissure and cerebellar folia. The structural brain changes resolved however after refeeding. There is conflicting evidence to link the age of onset of malnutrition and subsequent developmental problems and there are only a few studies which address the duration of malnutrition. However there is evidence from longitudinal studies of an association between severe childhood malnutrition and poorer visuo-kinesthetic integration (Cravioto and Arrieta, 1984).

There is no clear evidence for a similar effect in adults. The results of the post-war Minnesota study on experimental malnutrition in adults concluded that intellectual functioning was not unduly affected by prolonged semi-starvation (Keys, Brozek, Henschel, Mickelsten & Taylor, 1950). Although psychological and behavioural changes were marked in the male subjects involved, the results of formal intellectual testing failed to reveal significant deficits. Several studies have also examined the delayed effects of malnutrition in concentration camp survivors (Prisoners of War). For example, Abalan, Achminov and Pinsolle (1985) reported an increased risk for dementia in a POW population. In a similar vein, Sutker, Allain, Johnson and
Butters (1992) found that POWs who had sustained trauma-induced weight loss of more than 35 per cent of pre-confinement weight, performed significantly worse than veteran controls on four of five Wechsler Memory Scale (Revised) indices. Sutker et al (1992) suggested that the deficits found in the POW population were similar to the deficits associated with Korsakoff’s syndrome and speculate therefore on the possible link between thiamine deficiency and memory loss. However anorexia nervosa is not always accompanied by thiamine deficiency and many people with anorexia may often supplement a meager dietary intake with vitamins.

In contrast to Sutker et al's (1992) findings, Sulway, Broe, Creasey, Dent, Jorm, Kos and Tennant (1996) examined over one hundred former prisoners of war with veteran controls and found no significant differences on both neuropsychological testing and CT scanning. Sulway et al (1996) pointed out that many of the previous POW studies have questionable methodology and failed to control for the long term psychological effects of imprisonment in a variety of traumatic conditions.

There is also little known about the effects of short-term or intermittent starvation on cognitive functioning. Green and Rogers (1995) in a study of self-reported dieting behaviour, found that dieting was associated with lowered vigilance and slower cognitive processing. Green, Elliman & Rogers (1995) examined the effects of deprivation over three experimental conditions - missing one meal, missing two meals and 24 hour fast. Green et al (1995) found no significant differences among the experimental conditions with the exception of a poorer performance on a motoric vigilance test in the 24 hour fasting condition. Green et al (1995) also reported that 'dieters' demonstrated poorer working memory span although significant differences were not established between dieters and a group of non-dieting low to medium food
intake restrainers. The study suggest that dieting is associated with smaller working memory capacity due to preoccupation with food and eating behaviours, rather than global attentional deficit. In Kretsch, Green, Fong, Elliman and Johnson's (1997) controlled prospective study of obese dietiers, participants were found to have significantly slower reaction times which did not readily recover on weight restoration. However, contrary to the results of studies on short-term dieting, no significant differences were established for measures of sustained attention, motoric performance and immediate memory recall.

The actual content of diet is also a subject of which little is known in terms of cognitive functioning. Lloyd, Green and Rogers (1993) have demonstrated that the macronutrient content of food may have significant effects on both mood and cognitive functioning. Martin and Stevenson (1997) also found that hunger may precipitate headache although this is mediated by caffeine withdrawal and sleep variables. An hypothesised association between cholesterol reduction and serotonergic activity has been given tentative support from animal studies (Muldoon, Kaplan, Manuck & Mann, 1992) although such research is at an early stage of formulation. There is however, substantial evidence of a relationship between hyperglycaemia and memory dysfunction and further evidence of a modulating effect on cognitive function in the elderly due to peripheral ingestive hormones (Morley, Flood & Silver, 1992)

1f) **The influence of cortisol levels on cognitive functioning.**

It has been established that stress results in increased levels of blood cortisol concentrations acting upon glucocorticoid receptors in the brain. The result is severe
mood changes which is demonstrated in 75 per cent of those with Cushing's Disease and in those with prolonged use of corticosteroids (Kelly, Checkley, Bender & Mashiter, 1983; Brown, Koob & Rivier, 1991). There is also some evidence from animal studies, that elevated cortisol concentrations cause dehydration and hypoglycaemia with subsequent neuronal damage in the hippocampus region (Sapolsky, 1992). However, in a recent review, O'Brien (1997) argued that the relationship between prolonged hypothalamic-pituitary axis activation and neuronal damage is limited and the research requires to be replicated.

There is some evidence that increased blood cortisol has a negative effect on cognitive testing. For example, in a recent study involving normal volunteers, Newcomer, Selke, Melson, Hershey, Craft, Richards and Alderson (1999) found that high-dose cortisol treatment resulted in verbal (declarative) memory decrements using the Logical Memory subtests (Wechsler Memory Scales-Revised). However, no significant effects were established for recognition memory for geometric line drawings; visuo-spatial memory, executive functioning and measures of both sustained and selective attention. Wolkowitz, Reus, Weingarter, Thompson, Brier, Doran, Rubinow and Pickard (1990) in three independent studies, also found an association between dexamethasone administration and significantly poorer performance relative to controls on verbal memory tasks.

Hypercortisolism is particularly associated with anorexia in the acute phase with normal levels evident on weight restoration. The increased levels of cortisol in anorexia is hypothesised as being due to a regulatory defect at, or above the level of the hypothalamus (Casper, 1998). Gillberg and Rastam (1998) have suggested that hypercortisolism in anorexia may be responsible for the observed pseudo atrophy found in brain scan studies and the some of the deficits evident on
cognitive testing. The latter is supported by the evidence from studies on normal healthy volunteers and by the results of one study which found poorer vigilance performance to be associated with cortisol levels (Laessle, Fischer, Fichter, Pirke & Krieg, 1992). However, recent evidence reported by Seed (2000) suggests that cognitive problems in anorexia may not be related to cortisol levels. Seed (2000) found no difference between levels of cortisol secretion in participants with anorexia and matched controls. Whilst no between-group difference was found on a recall task, the introduction of a distractor resulted in poorer recall in the participants with anorexia. Seed (2000) concluded that anorexia may be associated with a heightened sensitivity to distraction on cognitive test.

1g) The influence of serotonin in eating disorders.

Serotonergic abnormalities are associated with eating disorders and a wide range of psychiatric disorders. In anorexia, serotonergic dysfunction may be secondary to elevated levels of cortisol, poor nutritional status, oestrogen deficiency or may co-exist with depressive symptoms. As is the case with hypercortisolism, weight restoration is accompanied by normalisation of serotonin levels (Connon & Treasure, 1998). In bulimia nervosa however, there is evidence for persistent abnormalities in both the hypothalamic-pituitary axis and serotonergic pathways following weight stabilisation. Whether or not these abnormalities are present prior to the onset of the disorder is unclear (Connon & Treasure, 1998). There is some evidence from animal studies which suggest a sex differentiation with female rats having higher serotonin potential (Arato, Freeska, Tekes, & Macrimmon, 1991). There is also some preliminary evidence of increased serotonergic binding sites in
females which may partly explain the high incidence of eating disorders and
depression in females (Arato et al 1991). However, the role of serotonin in pathology
and its interaction with other monoaminergic systems remains unclear (Herbert,
1997).

In considering eating behaviours generally, serotonin appears to have a role in
normal satiety mechanisms although the influence on poor impulse control found in
bulimia nervosa is uncertain (Palmer, 1998).

Increased levels of cortisol and serotonin dysfunction are of course not the only
biochemical factors involved in the stress response and recent research has
highlighted the possible role of peptides as a further modulating factor
(Herbert, 1997). In anorexia for example, the levels of the peptide vasopressin
stabilise with weight gain. However brain expression of vasopressin may be acute in
response to persistent stress and may act with other peptides to increase levels of
arousal and thus anxiety (Gold, Kaye, Robertson & Ebert, 1983; Herbert, 1997).
PART 2. NEUROPSYCHOLOGY AND EATING DISORDERS.

2a) The evidence for neuropsychological impairment in eating disorders.

In one of the first reported studies of neuropsychological functioning in anorexia, Fox (1981), administered a battery of cognitive tests to 14 participants with anorexia. The experimental group included two adolescents and one male. The tests included the Wechsler Adult Intelligence Scales-Revised (WAIS-R), the Wechsler Intelligence Scales for Children-Revised (WISC-R), the Benton Visual Retention Test (BVRT), and the Reitan Trail Making Tests. Eleven of the participants were also administered the Wide Range Achievement Test (WRAT). Fox (1981) found that 73 per cent of the participants scored at below two standard deviations on Trail Making Test - B (a test of visuo-motor tracking and set-shifting) and 47 per cent demonstrated visual memory impairment on the BVRT. However, these scores were comparable with a psychiatric control group with 80 per cent and 67 per cent respectively. Fox (1981) also found that 40 per cent of the anorexia group had a Verbal IQ of more than one standard deviation above their Performance IQ and that those who completed the WRAT, demonstrated a significant deficit on Arithmetic. In analysing these results, Fox (1981) cited the assumed link between arithmetical ability and visuo-spatial synthesis to speculate that the observed deficits may represent right hemispheric dysfunction. More specifically, Fox (1981) proposed that such dysfunction may be linked to observed perceptual disturbance of body size found in anorexia. However, later studies failed to find similar deficits on the WRAT and the evidence for a right hemispheric dysfunction in eating disorders is contentious (Touyzé, Beumont & Johnstone, 1986).
Fox's (1981) study had considerable methodological shortcomings. The comparison group of eight male and seven female psychiatric patients included affective disorders, hyperactivity disorder and one subject with schizophrenia. Given the established finding of cognitive deficits and structural brain changes in these disorders and of course gender effects, the results of this study should be interpreted with caution (Lezak, 1995).

In a study of outcome prediction, Small, Madero, Teagno & Ebert (1983) found that the Wechsler Adult Intelligence Scales (WAIS) subtests of Arithmetic and Digit Span explained almost 50 per cent of the variance. In contrast, the Rorshach scales measuring personality and perceptual variables failed to yield any outcome factors. (Klopfer, Ainsworth, Klopfer & Holt, 1954). Small et al (1983) found the latter result surprising given the clinical observation of difficulty in 'accessing' the thought processes of anorexics. Small et al (1983) noted that both the WAIS subtests of Arithmetic and Digit Span loaded highly on a 'Freedom from Distractibility' factor (Crawford, 1992). Small et al (1983), therefore proposed that the problem of therapeutic 'accessibility' in anorexia, may be more a related to difficulty in sustaining attention and organisation of thought, than a feature of personality in those with anorexia. Such a proposal is given support from experimental evidence which suggests that whilst anorexia is associated with poor performance on tasks of automatic (incidental) learning, cognitive efficiency is however demonstrated on structured tasks (Strupp, Weingartner, Kaye & Gwirtsman, 1986). However, there is no clear evidence that structured forms of therapy are more effective than other approaches in the treatment of anorexia and the question of 'accessibility' therefore remains unresolved.
In a study involving only three participants with anorexia and twenty four matched psychiatric controls, Maxwell, Tucker and Townes (1984) found that the anorexia group scored at more than one standard deviation below the mean of the comparison group on tests of "spatial reasoning" and on the Tactile Performance Test. However this study has considerable shortcomings with regard to the use of psychiatric controls. Further the use of a control participant aged 69 years is questionable.

Gordon, Halmi and Ippolot (1984) administered a variety of cognitive tests to inpatient adolescent females with anorexia and reported significant subtest scatter and cognitive test dysfunction in six from ten participants. Seven of the group scored relatively low on the WAIS/ WISC-R Information Subtest (a test of general knowledge) which Gordon et al (1984) found surprising, given the level of academic achievement reported in this population. Only one participant had difficulty on the verbal memory test. Gordon et al (1984) discussed their findings in terms of Bruch's (1979) conceptualisation defect in anorexia which proposed that those with anorexia may have difficulty integrating isolated facts; whereas they perform better in well structured situations with rote learning a more satisfactory strategy. However, Gordon et al's (1984) study used a female control group of adolescents with conduct disorders which limits the usefulness of their study. Further several of the participants with anorexia also had either a DSM III Axis II diagnoses of Borderline Personality Disorder or Schizotypal Personality Disorder both of which may have differential neuropsychological difficulties (Kemperman, Silbersweig, Stern & Russ, 1997).

Thompson (1991, 1993) administered a test battery to ten adults with anorexia and found that the anorexia group performed significantly more poorly than a control group on 16/40 neuropsychological test measures. However when depression was
partialled out, significant differences remained on only two test measures - the 3rd trial of the Rey Auditory Verbal Learning Test and the delayed recall trial of the Rey Complex Figure Test (RCFT-Delayed). Thompson (1991) concluded that the residual results suggested both an auditory-verbal memory impairment and visuospatial difficulties in anorexia. He further speculated that the poorer performance on the RCFT (Delayed) may be indicative of right hemispheric dysfunction. However, performance on the delayed recall component of this test may be influenced by conceptual strategy in copying and subvocal verbalisation, both of which are likely to involve bilateral hemispheric processing (Lezak 1995). Further the narrow focus of the results and the low number of participants would suggest that caution be exercised when drawing any conclusions from Thompson's (1991) study.

In a controlled study of cognitive functioning in bulimia, McKay, Humphries, Allen and Clawson (1986) found significant test differences when comparing 30 participants with bulimia (including participants with anorexia) with a control group. McKay et al (1986) administered the 269 item Luria-Nebraska Battery to reveal a poorer performance by the clinical group on the Motor and Pathognomonic Scales of the test battery. Poorer performance by the clinical group was also observed on a Design Fluency subtest which involved drawing geometric designs spontaneously and within a set time period. The results on Design Fluency accounted for much of the variance on the Luria-Nebraska Right Hemispheric Localisation Scale. McKay et al (1986) had hypothesised a right hemispheric deficit hypothesis in eating disorders and concluded that their results suggested focal right anterior hemispheric dysfunction in bulimia.
Whilst McKay et al's (1986) conclusion may be given tentative support by the finding of reduced right frontal glucose metabolism in bulimia (Wu et al, 1990; Andreason et al, 1992), impaired design fluency may also be associated with lesions in both hemispheres (Reitan & Woolfson, 1994; Kirk & Kertese, 1994). A major confounding factor in McKay et al's (1986) study is the inclusion of mixed eating disorder participants. Over a third of the group had anorexia and a third were depressed. An attempt was however made to control for the latter by comparing the test results of depressed and non-depressed participants. However, whilst no significant difference was found on this comparison, a trend towards poorer performance was observed in those who were depressed on the majority of the scales. McKay et al (1986) concluded therefore that the poorer performance by those with bulimia could not be attributed entirely to the influence of affective variables.

In a fairly large study Bowers (1994) found that a large group of participants with anorexia demonstrated significantly more cognitive deficits than a group of participants with bulimia nervosa. Both groups were in the acute phase of disorder and medication-free. Surprisingly perhaps, none of the participants met DSM III criteria for depression. A comprehensive test battery including the WAIS was administered pre-treatment and within three days of admission to hospital. However only the Category Test (DeFillipas et al 1979) and the Canter Interference Procedure for the Bender Gestalt (Canter, 1975) were computed for neuropsychological assessment. A significant difference was found between the clinical groups with 62.5 per cent of the anorexia group demonstrating one test score deficit compared to 30 per cent of the bulimia nervosa group. Bowers (1994) concludes that the results provide evidence for right hemispheric dysfunction in the acute phase of anorexia.
However, the study failed to report details such as Body Mass Index. IQ Scores and was self-limiting in not using a control group.

2b) Prospective neuropsychological studies on refeeding in anorexia.

A practical approach to establishing the aetiology of neuropsychological impairment found in anorexia is to re-assess cognitive functioning following weight restoration. Hamsher, Halmi and Benton (1981) found that 60 per cent of a group of participants with anorexia continued to demonstrate impairment on cognitive tests following weight restoration. Defining impairment as occurring at two standard deviations below the mean for 'normal' score results, Hamsher et al (1981) found that 70 per cent of their sample demonstrated impairment on at least one measure at pre-treatment. Impairment was noted in tests of reaction time, short term memory, general knowledge and arithmetical ability. However, only one participant demonstrated impairment on a visuospatial task and a further participant showed impairment on a constructional task. The lack of visuospatial impairment found in this study is in contrast to the results of other studies (Jones, Duncan, Brouwers & Mirsky, 1991; Szmuckler, Andrews, Kingston, Chen, Stargatt & Stanley, 1992; Kingston, Szmuckler, Andrews, Tress & Desmond, 1996). At post-treatment, 75 per cent of the participants in Hamsher et al's (1981) study maintained or increased their weight whilst the remainder actually lost weight. The number of cognitive impairments demonstrated at post-treatment was considerably reduced, although 60 per cent of the participants continued to demonstrate at least one impairment. It is worth noting that at post-treatment, these deficits were primarily found in the attention domain and predictive of outcome as measured by interview at one year
follow up. For example, 71 per cent of those participants having two or more
cognitive deficits at post-treatment had an unfavourable outcome at follow-up.
Although 19 of the 20 participants reported a history of perinatal complications, no
significant association was found between the number of complications and
neuropsychological measures. Whilst the presence of an anxious mood was
associated with two or more cognitive deficits at pre-treatment, no association was
found between depression and a more than one deficit. The importance of the effect
of anxiety was however not fully addressed by the authors, particularly in the light of
the attention deficits remaining at post-treatment.

Hamsher et al's (1981) study is flawed in not having a control group, although the
use of neuropsychological tests with published norms do allow for comparative
deficit analysis. A further possible methodological flaw is the use of both adults and
children as subjects and the inherent problems in partialling out the effects of
developmental status in the latter (Reitan and Woolfson, 1994).

In a controlled refeeding study, Szmukler et al (1992) administered a cognitive
test battery to twenty-one inpatients with anorexia (four with bulimic
symptomatology) at both pre and post-treatment periods. Szmuckler et al (1992)
found significant differences between the anorexia group and a control group at pre-
treatment on several neuropsychological domains. In the attention domain,
significance was established largely due to the anorexia group's poorer performance
on the Reitan Trail Making Tests A and B. Significant differences were also found on
tests of visuospatial/constructional functioning and problem solving ability.
However, in the domain of learning there were no significant differences established.
Following refeeding, eighteen participants with anorexia were retested to reveal
significant between-group differences in the visuo-spatial / constructional domain. Overall, the anorexia group demonstrated a greater variation in test scores than the control group. Those participants with a deficit scores (at two standard deviations) were analysed to reveal that at pretreatment, 62 per cent had demonstrated at least one deficit whilst 29 per cent had two or more deficits. At weight gain, 32 per cent of the participants in the anorexia group continued to have at least one test score deficit although Smuckler et al (1992) acknowledged that many of those who were considered to be weight-restored continued to have a relatively low Body Mass Index (B.M.I.). The authors also failed to establish significant associations between neuropsychological measures and age of onset and duration of eating disorder. A significant negative correlation was however found between B.M.I. and poorer performance on the Austin Maze although this result was confounded by slower processing speed. The study involved administration of the Beck Depression Inventory (BDI - Beck and Steer, 1987) and a measure of state anxiety - the State-Trait Anxiety Inventory (STAI-S - Spielberger 1983). However, no correlations were established between the BDI or STAIS-S and neuropsychological test scores. Thus mood was not deemed to be an influential variable.

More recently Szmuckler and colleagues reported on a controlled 'refeeding' study which combined neuropsychological test measures with MRI brain scans (Kingston, Szmuckler, Andrews, Tress & Desmond, 1996). In Kingston et al's (1996) study, 46 participants with anorexia and forty matched controls were administered an extensive neuropsychological battery which was repeated in 78 per cent of the anorexia group on refeeding. MRI scans were also carried out on all participants at pre-treatment and on 72 per cent of the anorexia group at post-treatment. The BDI and STAI-S were given at the end of the test battery. Participants with perinatal complications were
included in this study (12 per cent of those with anorexia and 25 per cent of controls) although no significant correlations were subsequently established between perinatal injury and cognitive test performance. The results of Kingston et al's (1996) study found significant between-group differences on an omnibus attentional score with poorer performance by participants with anorexia on the following measures: the Stroop Test, Reitan Trail Making Test A and the Digit Symbol Subtest from the WAIS. The participants with anorexia also performed significantly more poorly on the WAIS visuospatial subtests of Block Design and Picture Completion and on the copy score of the Rey Complex Figure Test. No significant differences were found on the serial learning tasks, although a significant difference was established in the memory domain due to poor performance on a measure of immediate text recall. Analyses of post-treatment results revealed, that whilst participants with anorexia improved on all domains excepting attention, they continued to perform significantly worse than controls on visuospatial and memory domains. A deficit analysis of the test scores revealed that at pre-treatment, 17 per cent of the participants with anorexia and 17 per cent of the controls scored at deficit level on one domain, with 26 per cent of those with anorexia demonstrating impairment on two or more domains. The difference between groups at pre-treatment was significant on all domains excepting memory. At post-treatment, significant differences remained in the learning domain only. However, the number of deficits in the anorexia group were not significantly reduced.

The results of MRI analyses in Kingston et al (1996) confirmed previous findings of abnormalities in anorexia. However, the authors pointed out that where present, radiological abnormalities were slight and may not be regarded as important on routine screening. As in previous studies, no correlations were established between
imaging MRI results and cognitive test scores. However, significant relationships were found between memory function, cognitive flexibility and BMI on admission to treatment. Trends were also noted in the other neuropsychological domains for low BMI and poorer performance. Interestingly, Kingston et al (1996) suggest that analysis of covariance is an inappropriate statistical procedure where significant differences are established between groups. Whilst this is questionable, Kingston et al (1996) therefore analysed the influence of affective variables using within-group correlational procedures. The results found no significant relationship between cognitive test performance and either STAIS or BDI scores. No significant correlation was found between psychotropic drug use (33 per cent of the anorexia group) and cognitive test or radiological measures. The authors note however that the use of medication may be a confounding factor, with medication use being associated with significantly lower BMI's and a significantly longer duration of eating disorder.

The importance of controlling for medication in such studies is unclear. Whilst antidepressant medication may both enhance and interfere with test performance (Sweet, Newman & Bell, 1992; Lezak, 1995); the use of benzodiazepines is more clearly associated with lowered cognitive performance (Dealberto, Sauron, Derouesne, Boyer, Mayeux, Piette, Kohler, Lubin & Alperovitch, 1994).

In a small controlled study of cognitive performance tests in anorexia, Green, Elliman, Wakeling and Rogers (1996) found recurrent poorer performance in the anorexia group as measured by a task battery previously employed in several studies.
of non-eating disordered dieters. Over the course of twelve weeks, the test battery was administered on three occasions with a measure of state anxiety presented at the beginning of each session. The results found that participants with anorexia demonstrated slower simple reaction time, poorer immediate recall of words and fewer finger tapping responses. Fatigue was evident reflecting poor nutritional status. Although the participants with anorexia made therapeutic progress and weight gain, there was no improvement on test performance over the twelve week period. The authors conclude that the results suggested working memory difficulties and this was discussed in terms of possible frontal lobe impairment. However, the study failed to find significant between group differences on other measures of attention and which suggested intact working memory.

In a controlled study of event-related potentials and neuropsychological functioning in 20 adolescents with anorexia, Bradley, Taylor, Rovet, Goldberg, Hood, Wachsmuth, Azcue and Pencharz (1997) found evidence for localised brain dysfunction which was not fully resolved on weight restoration. Bradley et al's (1997) study was discussed earlier in considering the neurobiological correlates of eating disorders and the focus here is on the neuropsychological aspects of the study. A variety of cognitive measures were used including the Peabody Picture Vocabulary Test to measure baseline IQ and the WISC-R at post treatment. Various test were used to assess attention, memory and hemispheric specialisation. The results revealed no significant group differences at both test periods, other than two post -treatment measures in favour of the participants with anorexia. Subsequent re-testing at eight months however, involved only eight pairs of participants and demonstrated an improvement on test scores in both groups. The results of Bradley et al's (1997)
study are of some interest in that whilst event-related potentials were found to be abnormal on verbal and non-verbal memory tasks, such abnormality was clearly not reflected by cognitive impairment.

In the first prospective study of both anorexia and bulimia nervosa, Lauer, Gorzewiski, Gerlinghoff, Backmund and Zihl (1999) administered cognitive tests to small groups of participants with either anorexia or bulimia nervosa. Assessment occurred at various treatment phases: pre-treatment, early treatment, twelve weeks treatment and at sixteen weeks follow-up. The results found no significant group difference at pre-treatment and both clinical groups demonstrated mild to moderate deficits on attentional and executive tasks. There were no problems revealed on verbal or visual memory tasks and no significant correlations established between test scores and affective variables. At seven months, speed of cognitive processing and problem solving ability were improved in both groups. The authors discussed the initial poor performance in both clinical groups on the attentional and executive tasks with reference to the possibility of motoric slowing due to nutritional status and/or 'cognitive overload'. Whilst Lauer et al (1999) observed cognitive improvements in parallel to treatment, the authors note that improvement in those participants with initial impaired performance (35 per cent), occurred in only 11 per cent of that group. This is consistent with Hamsher, Halmi and Benton's (1981) study which also found attentional difficulties to be sustained in some participants with anorexia at post-treatment.
2c) Equivocal neuropsychological findings in eating disorders.

Whilst the above studies do suggest neuropsychological dysfunction in eating disorders, there are several studies which have either failed to establish significant differences between clinical groups and controls or have found equivocal results (Witt, Ryan and Hsu, 1985; Palizidou, Robinson & Lishman, 1990). One study failed to establish differences between the scores of participants with eating disorders and published norms (Touyze, Beumont and Johnstone, 1986). A recent study by Mathias and Kent (1998) also found minimal differences between participants with anorexia and a control group on a wide range of cognitive tests. A further study of cognitive functioning in bulimia nervosa suggested that observed impairment was largely attributable to affective variables (Beatty, Wanderlich, Statton & Ternes, 1990).

In a study of adolescents with anorexia, Witt, Ryan and Hsu (1985) attempted to partial out the possible effects of affective variables and chronic medical illness by using three matched control groups comprising, participants with a depressive disorder, participants with diabetes and normal controls. The participants were matched for premorbid intelligence on the WISC-R or WAIS-R Information subtest despite Fox's (1981) finding that anorexia is associated with a poorer score on this test. The results of Witt et al's (1985) study, found no significant differences between groups on the WISC-R / WAIS-R Information, Digit Span and Digit Symbol Subtests. Further, no differences were evident on the Reitan Trail Making Test-B and the Visual Reproductions test from the Wechsler Memory Scales. These results are
in contrast to the results reported by Fox (1982), Szmuckler et al (1992) and Kingston et al (1996). Witt et al (1985) did however find that participants with anorexia performed significantly more poorly on a paired-associate task involving unrelated visual material (Symbol Digit Learning Test). The possibility of a processing speed deficit was excluded however, by the observation of equivalence between groups on the Reitan Trail Making Tests and the WAIS-R / WISC-R Digit Symbol Subtest.

Palazidou, Robinson and Lishman (1990) in a combined study of neuroradiological and cognitive test measures found no significant differences between seventeen adults with anorexia and nine matched controls on a computerised test battery. Further the mean scores of the anorexia group were within normal limits. A negative correlation was however established between low scores on a symbol digit coding test and enlarged external CSF spaces. Palazidou et al (1990) suggested that this result implied the presence of psychomotor slowing - a finding reported by others and possibly due to fatigue (Szmuckler et al, 1992; Green, Elliman, Wakeling & Rogers, 1996). Palazidou et al (1990) also found that a number of participants with anorexia scored below two standard deviations from the mean: 23 per cent on tests of visuo-spatial ability, 18 per cent on digit coding, 18 per cent on visual perceptual analysis and 18 per cent on a card-sort test. Interestingly, Palazidou et al (1990) revealed that a third of the control group had also scored at these levels. Similar equivocal test results obtained from controls was observed by Smuckler et al (1992) and Mathias and Kent (1998). Neither Palazidou et al (1990) nor Szmuckler et al (1992) however commented upon such results in terms of the normal population. It is possible that in Palazidou et al's (1990) study, the influence of test anxiety was considerable although this was not measured. It is may also be the
case that the computerised battery employed may require further validation as a neuropsychological instrument (Kane, 1992).

Mathias and Kent (1998) administered a battery of cognitive tests to thirty-four participants with anorexia and matched controls. The influence of affective variables was measured by the BDI and the STAI-S. Routine haematological results established for the anorexia group were analysed for correlation with test scores. Significant between-group differences were revealed with poorer performance by participants with anorexia on the WAIS-R, the General, Verbal and Delayed Indexes of the Wechsler Memory Scales -Revised (WMS-R) and the delayed recall element of the RCFT. The groups were however unmatched for premorbid intellectual ability and this measure was therefore entered as a covariant. The results of a partialling out procedure demonstrated significant between-group difference on one result only - Logical Memory I (WMS-R). A consideration of normal performance on other cognitive tests however, suggested impaired encoding for auditory-verbal prose independent of attention. Mathias and Kent's (1998) study also revealed that few of the participants with anorexia scored at a level of cognitive impairment with the highest number of deficits occurring on a measure of verbal fluency (Controlled Oral Word Association - Benton & Hamsher, 1989). However, a considerable number of the control group also scored at a defective level on this measure. The study also found no relation between cognitive functioning and depression. Further no relationship was established between cognitive functioning and BMI with the exception of low BMI and a longer time to complete the Reitan Trail making Tests. An analysis of haematological levels failed to demonstrate any significant relationship with test scores despite the presence of haematological abnormalities.
Mathias and Kent (1989) attributed the latter to the possibility of a high intake of mineral and vitamin supplements in the anorexia group. The authors concluded that the excessive weight loss and nutritional imbalance which characterises anorexia, appears to have minimal impact on measures of cognitive functioning.

In analysing cognitive functioning in a group of participants with anorexia and participants with bulimia nervosa, Touyz, Beaumont and Johnstone (1986) failed to establish significant between-group differences. More interestingly, on comparing the test scores with published norms, Touyz et al (1986) found that the scores to be within the normal range with the exception of ten per cent of the participants with anorexia who were impaired on a test of visual memory. The tests used in this study included many of those tests previous reported. For example the WAIS, the Benton Visual Retention Test, the Reitan Trail Making Test B and the Wide Range Achievement Test.

In a well crafted and controlled neuropsychological study, Jones, Duncan, Brouwers and Mirsky (1991) compared groups of participants with anorexia, participants who were weight-restored, participants with bulimia nervosa and matched controls. For the purpose of analysis, the authors used principle component analysis to reduce the considerable test variables to five neuropsychological domains - Vigilance, Focusing / Execution, Verbal, Memory and Visuospatial. The results established significant between group differences on four from five of those domains. Both the underweight anorexia group and the bulimia nervosa group performed at a level significantly below control participants in the domain of Focusing / Execution. The underweight anorexia group also scored below all groups in the Verbal Domain
based on the WAIS-R Subtests scores on Similarities, Comprehension and Vocabulary. The authors found the latter finding surprising given the evidence of a preserved Verbal IQ in anorexia. (Horne, Van Vactor & Emerson, 1990; Ransheen and Humphries, 1991). In the both the domains of Memory and Visuospatial Functioning, the underweight anorexia group performed significantly below the level of the control group only. In covarying for the possible effects of anxiety and depression, the authors found anxiety rather than depression to be an influential variable. However the latter was not discussed at length and the study failed to account for the possible effects of state anxiety. In concluding, the authors note that "The absolute differences in test scores between the eating disorder groups and normal controls were small. For the most part, the eating disorder groups mean scores differed little from published norms, suggesting subtle rather than frank neuropsychological dysfunction in the eating disorder groups." (page 721)

In a comparative study of bulimia nervosa and depression, Beatty, Bailly & Fisher (1990) found significant differences between groups on several neuropsychological tests, although concluded that the results were largely accounted for by affective variables. The authors compared fourteen participants suffering from depression with thirty two participants with bulimia nervosa and over forty controls on tests of short-term memory, a coding task and several verbal and non-verbal fluency tasks. The participants were also administered the BDI and STAI-S prior to cognitive testing. Beatty et al (1990) found significant differences between the clinical groups and controls on all four trials of a free recall memory test, the Famous People Fluency Test (Beatty et al, 1990) and in the number of rule violations committed in a Design Fluency Test (Jones-Gotman & Milner, 1977). However, differences between participants with bulimia nervosa and those with depression were significant on one
measure only - the number of rule violations. Beatty et al (1990) suggested that the latter may be a neuropsychological index of impulsivity. To control for the effects of depression, BDI scores in the bulimia group were also analysed to reveal that those participants with bulimia nervosa who were also depressed, performed at an impaired level on a free recall task. On comparing questionnaire scores between clinical groups, those participants with clinical depression scored significantly higher than those in the bulimia group on the BDI although not on the STAI-S. Beatty et al (1990) discussed these results in relation to the evidence for impaired retrieval difficulties in depression and concluded that the cognitive deficits found in bulimia may largely be accounted for by affective variables.

2d) Neuropsychological functioning in eating disorders - summary.

The available evidence suggests that anorexia is associated with clinically significant neuropsychological impairment. Where established, impairments have been found in attention, memory and visuospatial / constructional domains and do not appear to be correlated with brain scan abnormalities, Body Mass Index, duration of eating disorder and reported obstetric complications at birth. The presence of anxiety and depression appears to have had a minimal effect on neuropsychological test performance although only a few recent studies have used a measure of state anxiety. The occurrence of impairment is however variable with cognitive deficits reported between 17 and 70 per cent of those with anorexia. Prospective studies suggest an overall improvement on test scores on weight restoration although group results may mask individual levels of impairment. The longer term follow-up of
neuropsychological difficulties in eating disorders is yet to be reported and there is evidence that a liberal definition of weight restoration has been applied in some studies. Many of the studies lack appropriate controls although more recent studies have employed sound methodology. However there may be methodological problems inherent in testing participants with differential BMI's and at differing levels of weight restoration and therapeutic progress.

In bulimia nervosa, there are too few studies to draw definitive conclusions. Whilst there is evidence of impairment in attention in this population, the available evidence suggests that where evident, impairment is a result of affective variables. There is therefore a need to clarify whether or not bulimia nervosa is definitively associated with cognitive impairment and if so to what degree.

2e) The 'Right Hemispheric Dysfunction' hypothesis in eating disorders.

Several studies have been concerned with testing the hypothesis that anorexia and bulimia nervosa are associated with a right hemispheric dysfunction. This hypothesis stems in part from Bruch's (1962) clinical observations of perceptual disturbances in anorexia, and from neurological evidence of body image and appetitive disturbances in right hemispheric lesions. Bruch's (1962) observations promoted a considerable volume of research on body image distortion. However, the results of body image research have been remarkably inconsistent, due to methodological differences in estimation of body size (Strober, Goldenberg, Green & Saxon, 1979; Slade, 1985) and a host of extraneous environmental variables such as lighting effects and demand
characteristics (Reilly, 1994). Whilst Slade's (1985) review of the evidence suggested that over-estimation of body size is not unique to anorexia and may be widely found in the normal population, Smeets, Smit, Panhuysen and Ingelby's (1997) meta-analysis found considerable evidence of body shape distortion in anorexia.

The evidence for an association between eating disorders and right hemispheric dysfunction is also based upon body schema distortions associated with right-hemispheric pathology, brain tumors, traumatic brain injury and epileptiform activity (Braun & Chouinard, 1993; Reilly, 1994; Joseph, 1996). It is known that the right hemisphere is responsive to bilateral tactile stimuli whereas the left hemisphere is lateralised to the right side of the body. The right parietal region appears to maintain a somesthetic image of the entire body and lesions in this area are associated with body image disturbances (Joseph, 1988). However body schema distortions appear to be a rare occurrence relative to the incidence of anorexia and bulimia and the association may therefore be regarded as tenuous. It is conceivable that in eating disorders, depression may play a mediating and possibly causal role in body image distortion through elaboration of depressive body shape schemata. This hypothesis is given some support by the association between depression and reduced metabolism in the right posterior hemisphere (Heller, 1993). However, Kulbartz-Katt, Florin and Pook (1999) found body size estimation in a sample of bulimic women to be related to fluctuations in mood, rather than level of depression. This may suggest that body image is a more transient process than that inferred by neurological findings.

Some support for a state effect of body over-estimation in bulimia was provided by Lovell, Williams and Hill's (1997) emotional stroop study which found that
participants who had recovered from bulimia nervosa performed at the level of normal controls. However, this finding was not evident in a group of participants who had suffered from anorexia and were considered to be weight-restored. In this group, impaired colour-naming for anorexic-related words was clearly demonstrated and suggests an enduring concern with body shape and food in this population.

Limited support for a right hemispheric dysfunction hypothesis is evident from Casper and Heller's (1990) small laboratory study of eight participants with restrictive anorexia. The participants were measured at pre and post-treatment on an experimental task involving hemispheric differentiation. Casper and Heller (1990) found a non-significant trend for increased left hemispheric activation (as measured by the Chimeric Faces Test) and lower body BMI. On weight restoration, the participants demonstrated greater over-estimation of body size which was associated with increased right hemispheric activation and emotional processing. Although hemispheric differentiation failed to reach significance, the finding is consistent with the clinical finding of increased concern about body image in anorexia on weight restoration. The latter was also inferred from the results of Lovel et al's (1997) emotional stroop study. Casper and Heller (1990) speculated that anorexia is associated with decreased right hemispheric functioning and suppressed negative affect and this speculation is given tentative support by the considerable degree of alexithymia observed in anorexia (Bourke, Taylor, Parker & Bagby, 1992). However, Casper and Heller's (1990) results may have been too crude and were possibly confounded by intra-hemispheric dissociations between functional regions. For example, Mayberg, Liotti, Brannan, McGinnis, Mahurin, Jerabek, Silva, Tekell and Martin (1999) recent PET scan studies have found that feelings of sadness to be
associated with a decrease in right frontal cortical metabolism together with an increase in limbic metabolism (subgenua cingulate). The reverse was found on recovery from depression suggesting a functional reciprocity between these regions in the mediation of affect and attention. This evidence along with Kulbartz-Katt et al's (1999) finding of depression related body image distortion in bulimia, may suggest that body image distortion in bulimia is related to transient decreases in right prefrontal metabolism.

Horne, van Vactor & Emerson (1991) reported that body size over-estimation in a large sample of participants with an eating disorder, was significantly correlated with reduced Performance IQ scores relative to Verbal IQ scores (of more than one standard deviation). Other studies have also reported significant discrepancies between Verbal IQ and Performance IQ scores in eating disorders (Fox, 1981; Touyz, Beaumont & Johnstone, 1986; Blanz, Detzer, Lay, Rose & Schimdt, 1997). A significant Verbal-Performance IQ discrepancy is also associated clinically with brain lesions ipsilateral to a reduced Verbal or Performance IQ score (Lezak, 1995).

The WAIS Verbal and Performance IQ Scales were validated as differential measures of hemispheric functioning by Chase, Fedio, Norman, Brooks, Chiro and Mansi's (1984). Chase et al's (1984) widely reported PET study found that the WAIS Verbal IQ Subtests were associated primarily with glucose metabolic activity in the left parasylvian area, whilst the WAIS Performance IQ Subtests were reflected by activity in the right posterior parietal region. It is worth noting however, that the study was based on a small number of participants with Alzheimers disease with generalised cerebral atrophy on brain scanning and only five 'normal' control participants. The results of Chase et al's (1984) study should therefore be interpreted
with some caution. Others have suggested that the validity of using Performance IQ score as a measure of right-hemispheric impairment is questionable given the considerable cross-lateralisation involved in performance on the various WAIS Subtests (Crawford, 1992; Lezak, 1995). Nonetheless, some performance subtests are associated with right hemispheric activity. For example, the WAIS Block Design and Object Assembly Subtests. On factor analysis, these subtests have been found to load heavily on Perceptual Organisation which is associated with right hemispheric functioning (Walsh, 1993; Lezak, 1995).

There is some evidence that anorexia may be associated with difficulties on those WAIS Subtests which load heavily on perceptual organisation. For example, Kingston et al (1996) reported that some of their participants with anorexia were unable to complete the difficult patterns on the Block Design Subtest and suggested that this may represent a visuo-spatial impairment at a deeper level. Gillberg, Gillberg, Rastam and Johansen (1996) also found lower mean scores in the WISC-R Object Assembly subtest in a mixed group of under-weight and weight restored participants with anorexia. The authors attributed the participants poorer performance on the Object Assembly Subtest to a lack of gestalt in perceptual processing.

Other studies have failed to find a Performance IQ reduction relative to Verbal IQ in eating disorders. Blanz et al (1997) administered standard IQ tests to 190 adolescents with an eating disorder and found Full Scale IQ scores with a mean of 116.5 points for the participants with anorexia, and 114 points for those with bulimia nervosa. Whilst, the results failed to reveal a Verbal-Performance IQ differential in the group with anorexia, such a discrepancy was established for the group with
bulimia nervosa and in favour of Performance IQ. This study also compared psychometric performance and academic achievement in the eating disorder adolescents relative to psychiatric controls. The results confirmed empirical evidence that eating disorder adolescents achieve academically without being overstrained at school.

Other smaller scale studies have also failed to establish differential WAIS or WISC patterns in anorexia. (Wilbur and Colligan, 1981; Ransheen and Humphries, 1991). Wilbur and Colligan's (1981) psychometric study for example, failed to reveal a Verbal-Performance IQ discrepancy in thirty participants with anorexia. The WAIS / WISC IQ scores in this study were found to be approximately 0.6 standard deviations above the mean on the dimensions of Verbal Comprehension / Expressive Ability, Perceptual Organisation and general intelligence / academic aptitude.

It may be the case that where Verbal IQ - Performance IQ discrepancies have been found (in either direction), such results may be reflect discrepancies in the normal population and therefore may not be indicative of cognitive dysfunction (Matarazzo & Herman, 1985; Crawford, 1992; Lezak, 1995). Where a substantial proportion of those with an eating disorder do demonstrate Verbal-Performance IQ discrepancies, the results may possibly reflect the established finding of structural brain changes. However, given the high incidence of reported brain abnormalities in eating disorders, it would be expected that a higher incidence of Verbal - Performance IQ discrepancy is reported in the studies. An alternative explanation for the observed discrepancy may be that poorer performance on the WAIS Performance IQ Scale is attributable to both affective variables and slower processing speed. Slower processing speed has been demonstrated in both anorexia and bulimia (Beatty, Wandeerlich, Statton & Ternes, 1990; Szmuckler et al, 1992; Kingston et al,
1996; Green, Elliman, Wakeling & Rogers, 1996; Lauer et al, 1998) and the result may be confounded by depression, fatigue, distractibility or possibly a function of personality / cognitive processing style. The WAIS Performance IQ Subtests are timed with bonuses given for speedier responses in several subtests. There is an acknowledged speed versus accuracy trade-off in psychometric testing (Dennis & Evans, 1996), which may be more prominent in clinical groups associated with obsessive or perfectionist tendencies.

In summary, the evidence for a right hemispheric dysfunction in eating disorders is limited and a relationship between body image disturbance and localised right hemispheric activity has not been established. There is a growing body of evidence of perceptual asymmetry in neuropsychological models of emotions which may eventually help to clarify the lateralisation issue. For example, whilst cheerfulness is associated with left frontal activation, sadness and depression are associated with right frontal activation and depression with both reduced left frontal activity and posterior right hemispheric dysfunction (Davidson, 1992; Heller, 1993, Heller, Etienne & Miller, 1995; Crews and Harrison, 1995). The evidence for asymmetry in anxiety states is however less clear, with anxiety found to be associated with both increased right and left hemispheric activity (Heller et al, 1995). Recently Heller et al (1995) have suggested that when anxiety and depression are comorbid features, the resultant effect on brain activity may be confounded on EEG or imaging studies. There is also a sex issue related to lateral shift during the menstrual cycle (Coates, 1996). Functional imaging also promises to provide insights into hemispheric specialisation with studies demonstrating discrete emotional responses such as disgust being represented by left frontal activation. (Paradiso, Robinson, Andreaon, Downhill, Davidson, Kirchner, Watkins, Boles Ponto & Hichwa, 1997). It is perhaps
only a matter of time before a functional imaging study reports on cerebral activation occurring during eating disorder-related body image disturbance!

2f) The influence of emotional variables on cognitive test performance.

In reviewing the literature on cognitive functioning in eating disorders, the impact of both anxiety and depression on cognitive test performance appears to have been minimal. Thomson (1991) found that partialling out the effects of depression removed the majority of significant between group differences in a controlled study of cognitive functioning in anorexia. Beatty, Wanderlich, Statton and Ternes (1989) also found depression as measured by the Beck Depression Inventory (BDI) to be a major factor in the test battery performance in participants with bulimia nervosa. In contrast, Jones, Duncan, Brouwers and Mirsky (1991) found anxiety to be more influential than depression. However, in Szmukler et al's (1992) 'refeeding' study in anorexia, no significant correlations were established between cognitive test performance and measures of depression (BDI) and state anxiety (STAIS). Similarly Mathias and Kent (1998) found no significant relationships between test scores and the BDI and STAIS.

That only a few neuropsychological studies have reported emotional variables as influential or indeed have included a state measure in the methodology; may seem surprising given the evidence for emotion as a confounding variable (Lezak, 1995). However, in considering attention, Deary, Ebmeir, MacLeod, Dougall, Hepburn, Firer and Goodwin (1994) argue that it may be difficult to measure anxiety and attention as if they were independent variables. Anticipatory anxiety appears to elevate regional cerebral metabolism generally and is therefore difficult to control for
its effect on functional imaging (Drevets, Videen & MacLeod, 1992; Lucey, Costa & Blanes, 1995). Anxiety is also likely to be an important confounding variable, particularly on demanding cognitive tests. In clinical neuropsychology, cognitive testing does not take place in an emotional vacuum and the results of any form of testing have to be considered in light of affective variables and test anxiety (Lezak, 1995). This is perhaps particularly so in the case of eating disorders where anxiety and depression are co-morbid factors.

It is well established that anxiety produces selective processing effects on a wide range of laboratory measures with participants high in trait anxiety, consistently demonstrating selective attentional bias, interpretive bias and negative memory bias (Eysenck 1997). Arousal is associated with both positive and negative influences on learning and memory with high and low arousal associated with poorer test performance. High emotional arousal is generally associated with an explicit memory bias (Eysenck, 1997; Williams, Watts, MacLeod and Mathews, 1997) although in some cases, for example during severe trauma, heightened arousal may lead to repressive or dissociative mechanisms (Drugan, 1999). There is some evidence that encoding in severe trauma is essentially visual-motoric rather than verbal which provides some explanation of the phenomenon of flashbacks in post traumatic stress disorder (van der Kolk, McFarlane & Weisaeth, 1996). Joseph (1990) suggests that severe emotional, physical or sexual trauma experienced in early childhood may be processed visually and somatosensory in the right hemisphere only; with verbal encoding inhibited due to poor development of the corpus callosum. At the preconscious level Williams et al (1997) suggest that prior emotional material and possibly biologically primed processes are 'tagged' with a threat or loss value which is activated (in neuromodulatory terms) by perceived threat. There is considerable
evidence that threat is processed pre-attentively in anxiety disorders and which appears to effect emotional arousal as a signal for reallocation of cognitive resources (Williams et al, 1997).

In the emotional stroop paradigm, pre-attentive processes are likely to be triggered by motivationally significant stimuli with subsequent impairment on colour-naming performance. In many cases, the presentation of motivationally significant material also elicits an explicit memory bias (Wallace & Newman, 1992). In anorexia, impairments in colour-naming of anorexic-related words has been a consistent finding (Channon, Hemsley and de Silva, 1988; Ben-Tovim, Walker, Fok and Yap, 1988; Perpina, Hemsley, Treasure and de Silva, 1992; Lovell, Williams & Hill, 1997; Green, Wakeling, Elliman and Rogers, 1998).

A similar 'stroop' effect has been found in bulimia (Ben-Tovim et al, 1988; Fairburn, Cooper, Cooper, McKenna & Anastasiades, 1991; Cooper, Anastasiades & Fairburn, 1992; Perpina et al, 1992; Lovell et al, 1997) although one study has reported non-significant findings (Black, Wilson, Labouvie and Hefferman, 1996). Lovell et al (1997) also demonstrated that participants who had recovered from anorexia continued to demonstrate impaired colour-naming suggesting an enduring concern with anorexic issues. However, several studies have found that the stroop effect for food and body concerns is also evident in student population and in controls who have restrained eating habits / high drive for thinness (King Pollivy & Herman, 1991; Huon and Brown, 1995). Such results are likely to reflect the increasing preoccupation with body shape and food in the normal population and may provide some evidence for a continuum in eating disorders.
Perpina et al (1992) also found a differential effect on the stroop between anorexia and bulimia. Whereas anorexia was found to be associated with food related words, bulimia was associated more with body and shape words. Perpina et al (1992) discussed this result in terms of Cooper and Fairburn's (1992) clinical differentiation between anorexia and bulimia. Cooper and Fairburn (1991) have suggested that whilst a concern with food and eating is central to anorexia; a concern with weight and shape is associated with bulimia nervosa.

In the case of anorexia, cognitions about eating are often accompanied by vivid imagery of an invasive nature and beliefs that food will retain its mass within the body (Goodsitt, 1997). It may therefore be the case that different phases in the anorexic process may reveal different levels of concern about food, weight and body shape. In contrast to other studies, Green, Corr and De Silva (1999) argued that food concerns are not central to anorexia as measured by the Stroop paradigm. Rather a concern with food in anorexia may simply reflect a state of hunger. In support of this view they cite evidence from Channon and Hayword (1990) and Green, Elliman and Rogers, (1996) of impaired colour-naming as a result of experimental manipulation of hunger. However, hunger is essentially a transient variable which many anorexics are able to suppress and perhaps the significant factors which require hypothesis testing are the occurrence of enduring anorexic schemata and their emotional correlates. For example, Power and Dalgleish (1997) have suggested that disgust is a central component of anorexia, although this has not been tested experimentally.

The hypothesised influence of self-control as a causal factor in eating disorders was given some support by Ogden and Grenville's (1993) study of dieting. In a high 'preloaded' condition, Ogden and Grenville (1993) found that restrictive eaters
demonstrated increased activity, feelings of rebellion and poorer performance on the Stroop Test. The stroop test included eating related words (food and shape) and self-control related words (active and passive). Ogden and Grenville (1993) discussed their results in terms of the violation of diet through the consumption of forbidden food with subsequent emotional backlash. However, Green, Elliman and Rogers, (1996) carried out a preloading experiment with non-dieting participants in a food deprived and non-deprived conditions. Whilst the study found a significant effect for colour-naming of food words, the stroop effect for body/shape words was not evident. There was also no significant interaction effect between semantic content and level of hunger. The authors suggested therefore that subjective ratings of hunger rather than actual feelings of hunger appear to be the mediating variable in redirection of attentional resources.

In an alternative approach to pre-attentive investigation, Herman, Pieters & Eelen (1998) attempted to control for the confound of threat related arousal and current schematic concerns about food in anorexia, by ensuring that anorexic-related words were not significantly different from neutral words in affective valence. The study used word stimuli rated as pleasant or unpleasant in conjunction with a instruction to create word-related images. On the basis of the emotional stroop findings, the study tested the hypothesis that an implicit memory bias would be evident in anorexia. Cued-recall words were used to test explicit memory bias and a word-stem completion tasks was employed to measure implicit memory bias. The results found that compared to a normal control group, participants with anorexia demonstrated a strong explicit memory bias for cue-recalled anorexic-related words. The results of the stem completion task (implicit memory) however failed to yield a significant
between-group difference. It is worth noting that Herman et al (1998) also controlled for the possible confounding variable of hunger by using participants with anorexia who were not fasting, thus presumably controlling for current food related concerns.

On reviewing the evidence of the emotional stroop effect in general, Williams et al (1997) concluded that the data was best explained by a parallel distributed processing (PDP) model (Rumelhart, Smolensky, McClelland & Hinton, 1986), where prior experience and elaborative mechanisms such as current concerns (worry) increase input sensitivity and therefore responsiveness at pre-attentive levels. In the PDP approach there is no requirement for stimuli to be explicitly linked and therefore a variety of apparently unconnected stimuli may provoke a threat response (Williams et al, 1997).

Depression is also associated with selective cognitive processing bias with selective recall for negative material an established finding (Beck, Rush, Emery & Beck, 1979). A pre-attentitive bias has however not been established in depression and it is unclear as to what processes are involve in transient mood shifts and where anxiety is a component of the depressive symptomatology. Depression is also often accompanied by attentional deficits, impaired short term memory recall for both verbal and visuo-spatial material and psycho-motor slowing (Williams et al, 1997). Where memory deficits are established, they appear to be due to retrieval rather than learning difficulties and may be a result of weak encoding (Lezak 1995). However, some studies have failed to establish differences between participants with depression and control participants on cognitive testing and differentiation may only be evident in subsets of depressive states.
The results of cognitive testing in depression may also be influenced by poor sustained attention and catastrophic response to failure. Such a response was evident in Elliot, Sahakian, McKay, Herrod, Robbins & Paykel's (1996) study which found that participants with depression demonstrated an apparent over-sensitivity to failure with subsequent test failure. The trend was found to be correlated with level of depression and may be explained by Williams et al's (1997) priming versus elaboration model. In Williams et al's model, depression is posited as being maintained by the active elaboration of negative stimuli; followed by reduced attention to neutral or ambiguous stimuli.

In contrast to the catastrophic effect described by Elliot et al (1996) in depressive disorders, DiBartolo, Brown and Barlow (1996) found that negative test feedback promoted a positive response in anxiety. In a controlled study of attention allocation, the effect of including a negative feedback condition (on task performance) led to the unexpected result of enhanced performance for participants with anxiety compared to non-anxious controls. DiBartolo et al (1996) reported that improved test performance in anxiety disorders is linked to increased levels of worry and possibly a relocation of additional attentional resources.

There are also several other self variables which may have an influence on cognitive test performance. For example, anorexia is associated with perfectionism and self-directed goal achievement and where such characteristics occur, it would be reasonable to predict test anxiety, fear of failure and slower test response times. In bulimia nervosa, an association with poor impulse control and variable attention may suggest the possibility of distractibility and poor motivation on test performance. These variables are difficult to control for in a clinical neuropsychological study and
therefore require experimental laboratory approaches to tease out the various influences on cognitive functioning in eating disorders.

2g) The influence of the menstrual cycle on cognitive test performance.

Since eating disorders are primarily associated with the female population it is worth examining the potential influence of the menstrual cycle on cognitive test performance. Broverman, Klaiber, Kobayashi, and Vogerl (1968) found that colour naming performance was performed better during the follicular than the luteal phase (post-ovulatory) of the menstrual cycle. In a study of cognitive functioning over the duration of the menstrual cycle Hampson and Kimura (1988) found differential results according to menstrual phase. Using the rod and frame test to measure perceptual -spatial processing and several tests of manual coordination (Purdue pegboard, finger tapping and manual sequencing) Hampson and Kimura (1988) found that better perceptual-spatial performance was evident during the menstrual phase compared to the luteal phase. In contrast, the reverse result occurred on the manual coordination tests. Hampson and Kimura (1988) argued that the high levels of 'female' hormones present in the luteal phase appear to enhance skills for which women tend to excel, whilst apparently compromising skills generally associated with males. However, Ho, Gilger and Brink (1986) found that a mental rotation task (associated with better performance by males) was performed quicker near the time of ovulation compared to the menstrual phase when female hormones are at their lowest level.

Heister, Landis, Regard and Schroeder-Heister's (1988) study of twelve healthy adult females confirmed an established left hemispheric advantage for verbal
processing and right hemispheric advantage for face perception. However 83 per cent of the participants also demonstrated a smaller or reversed asymmetry for face recognition during the premenstrual phase. Similarly, Chiarello, McMahon & Schaefer (1989) found that performance on the Judgement of Line Orientation Test (a measure of angular relationships considered to measure right hemispheric functioning - Benton & Hamsher, 1983); varied with menstrual phase. Peak performance was found during the follicular phase and poorest performance during the menstrual phase. The latter result is consistent with Ho et al's (1986) finding of poorer visuospatial processing during the menstrual phase. In contrast, a small study by Gordon, Corbin and Lee (1986) failed to find any significant difference between menstrual cycle phases on visuo-spatial and verbal fluency tasks.

In reviewing the above studies, there does appear to be some albeit limited evidence for differential effects on cognitive function during the menstrual cycle. However, it is difficult to draw any conclusions and the studies failed to control for emotional variables and other possible confounding factors. Walker (1997) in a comprehensive review of the evidence, concluded that differential cognitive function during the menstrual cycle is not established and where it is evident; the effect is unlikely to have any marked influence on intellectual ability.
PART 3: COGNITIVE FUNCTIONING IN ANOREXIA AND BULIMIA.

The aim of the present study was to determine the presence and extent of neuropsychological dysfunction in adult eating disorders. Specific hypotheses were tested in line with the available evidence which suggests that whereas anorexia nervosa is associated with mild impairment across all neuropsychological domains, bulimia nervosa is associated primarily with attentional deficits.

3a. HYPOTHESES.

Hypothesis 1.

In comparison to a matched control group, a group of participants with anorexia nervosa will demonstrate significantly poorer performance on cognitive tests in each neuropsychological domain tested.

Hypothesis 2.

In comparison to a matched control group, a group of participants with bulimia nervosa will demonstrate significantly poorer performance on cognitive tests in the neuropsychological domain of attention only.
Hypothesis 3.

Where significantly poorer performance is established for participants with bulimia nervosa, test scores will be found to have been influenced significantly by affective variables.

3b. METHOD.

A between-subjects experimental design method was used involving self-report questionnaires and the administration of a range of cognitive tests.

3b (i). Participants:

30 female participants with anorexia nervosa, 30 female participants with bulimia nervosa and 30 females without an eating disorder were recruited using a postal opt-in procedure.

Participants with anorexia are referred to below as Group AN (Anorexia) or Group AN. Participants with bulimia nervosa are referred to below as Group BN (Bulimia) or Group BN. Participants without an eating disorder are referred to below as Group C (Controls), Group C or 'controls'. Participants in Group C were administered the experimental test battery and may therefore be regarded as an experimental group. However, for the purpose of clarity, the term 'controls' is used below. All participants were matched for age, years of education and premorbid intelligence (Table 1: page 62).
Group AN (Anorexia Nervosa):

Twenty three adult female subjects with a DSM IV diagnosis of Anorexia Nervosa were recruited from two specialist out-patient treatment centres. At the time of testing, four further participants with anorexia nervosa were psychiatric in-patients. However, the results of blood chemistry and physical examination indicated relative physical stability and nutritional intake. Three further participants were recruited via local self-help groups.

Group BN (Bulimia Nervosa):

Twenty five adult females with a diagnosis of Bulimia Nervosa were recruited from two specialist treatment centres. Five further participants were recruited from a local self-help group.

Group C (Control):

Thirty adult females with normal body weight were recruited as controls. The participants in the Control Group were screened for eating pathology using self-report questionnaires and on the basis of a brief pre-test interview.

Exclusion criteria:

Pregnancy, epilepsy, substance abuse, traumatic brain injury and disease with established correlates of cognitive impairment (eg Diabetes Mellitus) were defined as
exclusion criteria (Strachan, Deary, Ewing & Frier, 1997). Psychiatric disorder was a further exclusion factor for Group C participants.

Two participants in the Group BN (Bulimia) were left-handed and matched with two left-handed participants in the control group. Such matching was deemed desirable given the evidence for handedness-dependency in the performance of specific cognitive tasks (Jones and Martin, 1997; Karapetas and Vlachos, 1997).

**Group Characteristics (Table 1):**

The mean age of Group AN (Anorexia) was 25.1 years with a range of 17 to 48 years. Twenty three per cent of this group were above the age of 30 years and 7 per cent were aged 40 years or over (Table 1).

The mean age of Group BN (Bulimia) was 27.1 years with an age range of 18 to 43 years. Thirty seven per cent of this group were above the age of 30 years and 10 per cent were aged 40 years or over.

The mean age of Group C (Control) was 27.4 years with a range of 17 to 47 years. Twenty seven per cent of this group were aged 30 years or more with 10 per cent aged 40 years or over.

As would be expected, significant between-group differences were established for Body Mass Index ($F_{2,87} = 57.39, p<0.01$) with Group AN (Anorexia) having a mean BMI of 15.5 with a range of 14 to 17 (Table 1).
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group AN (Anorexia) N = 30</th>
<th>Group BN (Bulimia) N = 30</th>
<th>Group C (Control) N = 30</th>
<th>F(df) / Chi-Sq</th>
<th>p value</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>25.1</td>
<td>27.1</td>
<td>26.7</td>
<td>F(2.87)0.091</td>
<td>p = 0.449</td>
<td>n.s.</td>
</tr>
<tr>
<td>Range (yrs)</td>
<td>17 - 48</td>
<td>18 - 43</td>
<td>17 - 47</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education (yrs)</td>
<td>12.6</td>
<td>12.2</td>
<td>12.7</td>
<td>F(2.87)0.87</td>
<td>p = 0.423</td>
<td>n.s.</td>
</tr>
<tr>
<td>Premorbid IQ (NART - R)</td>
<td>sd 2.34</td>
<td>sd 1.67</td>
<td>sd 1.29</td>
<td>F(2,87)1.037</td>
<td>p = 0.359</td>
<td>n.s.</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>15.5</td>
<td>23.4</td>
<td>23.1</td>
<td>F(2,87)57.39</td>
<td>p = 0.000**</td>
<td>AN &lt; BN, C</td>
</tr>
<tr>
<td>Duration of Eating Disorder (yrs)</td>
<td>7.55</td>
<td>6.57</td>
<td>n/a</td>
<td>U 414.392</td>
<td>p = 0.598</td>
<td>n.s.</td>
</tr>
<tr>
<td>Duration - Range (yrs)</td>
<td>0.5 - 26</td>
<td>1.5 - 20</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Purging Behaviours</td>
<td>23%</td>
<td>87%</td>
<td>n.a.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antidepressant Medication</td>
<td>30%</td>
<td>33%</td>
<td>n.a.</td>
<td>Chi-Sq 0.341</td>
<td>p = 0.559</td>
<td>n.s.</td>
</tr>
<tr>
<td>Birth Complications</td>
<td>13%</td>
<td>13%</td>
<td>16.70%</td>
<td>Chi-Sq 0.480</td>
<td>p = 0.786</td>
<td>**p &lt; 0.010</td>
</tr>
</tbody>
</table>

Two-thirds of Group BN (Bulimia) demonstrated a BMI within the normal range of 20-24. The remaining third demonstrated a BMI within the overweight category (BMI: 25 to 29).

Group C (Control) demonstrated a mean BMI of 23.1 and a range from 21 to 27. Thirteen per cent were overweight with BMIs ranging from 25 to 27.
The mean duration of eating disorder in Group AN (Anorexia) was 7.55 years with a range of 6 months to 26 years. The mean duration of eating disorder in Group BN (Bulimia) was 6.6 years with a range of 1.5 to 20 years. Between-group significance was not demonstrated on duration of eating disorder (Table 1).

Twenty three per cent of Group AN (Anorexia) and 83 per cent of Group BN (Bulimia) reported purging behaviours (Table 1). However, DSM IV subtypes were not applied for separate statistical analysis.

Thirty per cent of Group AN (Anorexia) and 33 per cent of Group BN (Bulimia) were taking prescribed anti-depressant medication and between-group differences failed to reach statistical significance (Table 1). However, due to the possible confounding effects of psychotropic medication on cognitive test scores, separate analyses were carried out using anti-depressant medication as an independent variable. The results of this analysis will be discussed in the Results section which follows.

Thirteen per cent of Group AN (Anorexia) and 13 per cent of Group BN (Bulimia) reported possible birth complications. Seventeen per cent of Group C (Control) also reported possible obstetric complications at birth. Group differences failed however to reach statistical significance (Table 1).
3b (ii). Materials:

(a). Psychometric Questionnaires:

Eating Disorder Inventory - 2. (Garner 1991)

The Eating Disorder Inventory (EDI-2) is a widely used and validated self-report instrument which taps into anorexic and bulimic symptomatology. The results provide standardised subscale scores for 8 scales - Drive for Thinness, Bulimia, Body Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust, Interoceptive Awareness and Maturity Fears. The EDI 2 also provides three provisional scales - Asceticism, Impulse Regulation and Social Insecurity.

The Bulimic Investigatory Test Edinburgh (Henderson and Freeman, 1987).

The Bulimic Investigatory Test Edinburgh (BITE) is a 36-item measure of bulimic symptomatology yielding both a symptom score and severity score. The psychometric properties of the BITE appear to be adequate, although the questionnaire has not been subjected to extensive research and the cut-off scores may not adequately detect binge-purging anorexics (Waller, 1992: Nathan & Allison, 1998)

The Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983)

The Hospital Anxiety and Depression Scales (HADS) is widely used as a measure of both anxiety and depressive symptomatology. The 14 items were selected to avoid the confounding effects of somatic symptoms prevalent in anorexia nervosa.
Delusions Symptoms States Inventory / Neurotic Symptoms - Revised
(Foulds and Bedford 1978).

The Delusions Symptoms States Inventory / Neurotic Symptoms (DSSI / NS) measures frequency and severity in 5 symptom categories - Conversion, Compulsion, Phobic, Rumination and Dissociation. The DSSI/NS is regarded as a comprehensive screening measure and may be deemed to encompass many of the psychological symptoms which are associated with eating disorders (Solyom, Freman & Miles, 1982; Everill, Waller & Macdonald, 1995).

State-Trait Anxiety Inventory: State Anxiety Scale/ Form Y-1 (Speilberger, 1983).

The State-Trait Inventory: State Anxiety (STAIS-S) is a 20 item scale measuring apprehension, tension and worry. It is regarded as a reliable and valid measure of state anxiety and has been used extensively in psychotherapeutic and quasi-experimental situations.

(b). Cognitive tests.


The National Adult Reading Test - Revised (NART-R) requires the participant to read aloud two columns of phonetically irregular words. An error score yields estimated Full Scale, Verbal and Performance IQ's related to the Wechsler Adult Intelligence Scale - Revised (WAIS-R) As a measure of premorbid intelligence, the NART-R is based on the knowledge that vocabulary correlates best with overall
intellectual ability level. Vocabulary is also more resistant to brain damage and
cognitive decline than other intellectual abilities. The NART-R IQ Score also
correlates significantly with education and social class. However, whilst accounting
for 72 per cent of Verbal IQ Score on the WAIS-R, it accounts for only 33 per cent
of Performance IQ and the NART-R Performance IQ is regarded therefore as a poor
predictor of Performance IQ (Crawford, 1992; O'Carroll, 1995).

Attention Domain:

Whilst it is argued that there are no true tests of attention, there are various
neuropsychological tests which tap into attentional processes and the following are
examples of widely used tests of attention:

Wechsler Memory Scale -Revised - Attention-Concentration Index:

The Attention-Concentration Index summates five individual tests - Mental
Control, Digit Span (Forwards and Backwards) and Visual Memory Span (Forwards
and Backwards). The index has sufficient validity (Elwood, 1991) and adequately
separates out the attentional components of the Wechsler Memory Scale -Revised
battery (Lezak, 1995).

Mental Control is a simple mental tracking task comprising three brief tasks -
counting backwards from 20, repeating the alphabet and counting from 1 to 40 in
threes. Each test is timed and scored and individual scores are summated to yield a
Mental Control score. Factor analytic studies have consistently revealed Mental
control as a test of attention (Lezak, 1995).
**Digit Span** measures immediate verbal recall for numeric material presented verbally and is primarily a test of attention. Digits Forward and Digits backwards involve different mental activities and are affected differentially by brain damage. This questions the use of Digit span as a unitary factor when summated with Digits Backward (Lezak 1995). Digits Span Forwards is affected by anxiety which may inhibit the number of digits recalled (Mueller 1979). The test is more vulnerable to left than right hemispheric dysfunction although Chase et al (1984), found that glucose metabolism during Digits Forwards increases bilaterally (and principally in the anterior dorsal regions). However, D'Esposito and Postle (1999) in a review of eleven studies of pre frontal lesions and digit span deficits, failed to find evidence of significant differences from normal control scores.

**Digit Span Backward** is a mental-tracking test which essentially measures verbal working memory. However, the cognitive processes involved may include visual scanning and factor analyses suggests the involvement of both verbal and visual processes. The test is sensitive to brain damage particularly in the left hemispheric where visual field defects are associated with shortened digit span.

**Visual Memory Span** is a variant of the block tapping test devised by Corsi to measure topographical / visual memory. The task involves tapping out a series of visual spans demonstrated by the test administrator. The result may be confounded by response consistency (Lezak, 1995)
In this study, the raw results of these individual tests were used to analyse group differences in cognitive deficits. However, for the purposes of statistical analysis only the computed transformed score for the Attention-Concentration Index was used to reduce the amounts of dependent variables processed.

**Paced Auditory Serial-Addition Task** (Gronwall and Sampson 1974; Gronwall 1997)

The Paced Auditory Serial-Addition Task (PASAT) is a measure of sustained attention and perseverance. The task involves listening to an audiotaped presentation of several trials of 60 digits in trials of increasing speed. The subject is required to mentally add the first number to the second; provide the answer verbally; add the second number to the third, and so on. The version used here presents trials at intervals of 2.4 seconds; 2.0 seconds, 1.6 seconds and 1.2 seconds.

Gronwall and Wrightson (1981) indicated that performance on the PASAT is neither related to general intelligence nor arithmetic ability. However, Egan (1988) found that the PASAT did in fact correlate highly significantly with general mental ability. Similarly, Deary, Langan, Hepburn & Frier (1991), in a study of 94 healthy young adults with Type 1 insulin dependent diabetes, found PASAT scores to correlate significantly with WAIS-R subtests. Differential effects were also revealed by presentation speed. Further, Deary et al (1991) found that the PASAT loaded highly on 'Freedom from Distractibility' which is summated by the WAIS-R Subtests: Digit Span, Arithmetic and Digit Symbol (Crawford, 1992). In agreement with Gronwall and Wrightson (1981) Deary et al (1991) also found that the Four Second PASAT scores also correlated significantly with a measure of auditory-verbal learning (Rey Auditory-Verbal Learning Test). The Two second PASAT scores were
also correlated with perceptual processing speed. With regard to 'brain mapping', Deary, Ebmeir, MacLeod, Dougall, Hepburn, Frier and Goodwin's (1994) SPECT study involving diabetics and controls, found that PASAT performance was related to relative decreases in left posterior cingulate and right anterior cingulate areas (areas considered to be central to arousal). The established IQ decrement found in participants with severe hypoglycaemia was controlled for in Deary et al's (1994) study with no significant effects reported. Deary et al (1994) suggest that the association revealed between the PASAT and decreased cingulate activity reflects differential aspects of attention. They suggest that the PASAT requires active suppression of internal information in working memory and this indeed is consistent with the results of other studies requiring attention to internal information (Eysenck, 1997). The PASAT appears to be sensitive to subtle information processing deficits or abnormally slow processing ability (Ponsford and Kinsella 1992).

The PASAT is often experienced as anxiety provoking due to a sense of pressure and participants may interpret failure despite sustained attention and satisfactory responses (Lezak 1995). Deary et al (1994) also found anxiety to be a confounding variable as measured on the STAIS questionnaire (for state anxiety).

Analyses of cognitive deficits in the present study was provided by the results of the third and fourth PASAT trials. However, to reduce the number of dependent variables, the results of the four trials were summated to provide a PASAT Mean Score.
Digit Symbol.

Digit Symbol (WAIS-R) is a well used coding test involving complex attention, motor persistence and visuo-motor co-ordination. It is relatively unaffected by intelligence, learning and memory; although the task may involve an incidental learning component. It is regarded as the most sensitive WAIS-R subtest to minimal brain dysfunction. Although non-specific with regard to regional brain damage, glucose metabolism studies have revealed Digit Symbol to be associated with bilateral increase in posterior areas, more right than left (Chase et al, 1984). The test is also subject positively to the effects of aerobic exercise (Dustman, Ruhling, Russell, Shearer, Bonekate, Shingoka, Wood & Bradford, 1984).

Trail Making Test A.

The Reitan Trail Making Test A (TMT-A) is a timed pencil and paper test of visuo-motor tracking which has been widely used since its development in the 40's. A longer time denotes poorer performance and may be influenced by motor slowing, poor coordination, and visual scanning problems. It is likely to be affected by affective variables (Gass and Daniels 1990) and possibly by poor impulse control. Trail Making Test performance correlates highly with caudate atrophy in Huntington's disease and is associated with frontal lobe function.

The Stroop Test (Modified).

The Stroop Test is a widely used measure of attention. There are several variations employed in presentation and materials. The modified version used in this study involves two trials -
Trial 1. The participant is required to read aloud and as quickly as possible, several columns of colour name words printed in black ink. This serves as a priming task for the second trial which also involves reading aloud columns of colour word names which are however printed in colours which do not correspond to the words being read. A total score is derived from the time taken to read the colour word trial minus any mistakes made without correction. The Stroop task used here is modified and was therefore not used in collating levels of cognitive deficit.

**Learning and Memory Domain.**

**WMS-R: Logical Memory I and II.**

Logical Memory I is an immediate free recall test of auditory-verbal prose material. Logical memory II is a delayed recall task presented after 30 minutes. Both Logical memory I and II are associated primarily with verbal processing, possibly due to the need to organise the material verbally with syntax required for repeating the prose. There is some evidence for poor performance being associated with left hemispheric lesion (Ivnik, Sharbrough & Laws, 1988)

**WMS -R: Verbal Paired Associates I and II.**

Verbal Paired Associates I requires the subject to learn pairs of word associations over three trials, although six trials may be given if necessary in order that the pairs are learned. A recall test (Verbal Paired Associates II) is administered after 30 minutes. There is a significant verbal learning component to this task with left
hemispheric lesions associated with significant lower scores when compare to right hemispheric lesions. However, some of the word pairs are easily encoded visually and the task may therefore involve dual encoding (Lezak, 1995).

Recovered mild head injury has consistently shown slightly lower performance on delayed recall. Delayed recall scores are apparently affected by depression. Separate WMS-R norms are not available for this test and thus the scores were not processed for levels of cognitive deficit.

**Rivermead Behavioural Memory Test - Picture Recognition**

The Picture Recognition Test is a component of the Rivermead Behavioural Memory Test. This component measures delayed visual recognition following presentation of line drawings of familiar objects. The Extended Version used here was devised by De Wall, Wilson and Baddely (1994).

**Rey Complex Figure Test - Recall.**

The Rey Complex Figure Test - Copy is a visuospatial and graphic task involving copying a complex 2D figure drawing. Delayed free recall at 3 minutes yields a measure of incidental visual memory. The participant is not informed that a recall trial will be given. The scoring system has been found to be reliable (Tupler, Welsh, Asare-Aboagye & Dawson, 1995). Recall scores may be influenced by conceptual copying and lateralisation of lesions. Left hemispheric lesions may give rise to defective copy due to poor organisational strategy yet show improvement on recall. Right hemispheric lesions may produce poor or impaired copy together with poor recall (Walsh, 1994: Lezak, 1995).
Executive Functioning Domain.

Trail Making Test B.

Reitan's Trail Making Test B is a visuo-motor tracking task with a conceptual shift component. The test immediately follows administration of Trail Making A and involves a ascending and alternating numeric/alphabetic sequence. A longer time denotes a poorer performance. Trail Making Test B is regarded as both a complex test of attention and of executive function. A defective score in either Trail Making Test A or B suggests global impairment. Impulsivity as demonstrated by brain trauma is revealed by the omission of Letter Item '12' between Number Items '12' to '13' although this measure was not analysed in the present study. Depression is associated with slowing on Trail Making B (Lezak, 1995).

WISC- III - Mazes subtest.

The Mazes subtest from the Wechsler Intelligence Scales for Children - III (1991) is a pencil and paper test of planning and perceptual organisation. The most difficult maze is regarded as equivalent to the items of the Porteus Maze test for adults (Lezak, 1995). Mazes is a brief timed test and is included here in preference to the considerably longer Porteus Maze.

Verbal Fluency (Benton and Hamsher, 1989).

The Controlled Oral Word Association Test (COWA) is a simple test of cognitive flexibility which is sensitive to brain dysfunction. The COWA measures the quantity of words produced by the subject over three one minute trials, each with a designated letter (C,F and L). Frontal lesions on either hemisphere decrease fluency
scores generally although left frontal lesions may have a more profound effect than right hemispheric lesions (Perret 1974). This is given support from functional brain imaging studies which demonstrate that verbal fluency is associated with predominantly frontal activation and lower scores being associated with higher metabolic rate (Parks, et al, 1988). This would intuitively suggest that higher cerebral metabolism is the result of increased effort as a response to poorer verbal fluency although Parks et al (1988) also found that the verbal fluency task activated a wide network of cerebral regions on PET scan and the influence of general arousal and anxiety are therefore potential confounding factors. Performance on a verbal fluency test may also reflect organisation of thought with the use of initial consonant providing more words e.g. father, fathom, far (on the FAS version), or through the use of themes or variations on a word. Poor scores may also reflect cognitive inflexibility and poor retrieval.

**Visuo-spatial / constructional functioning.**

**WAIS-R Block Design.**

Block Design is a 2D measure of visuospatial/constructional ability. It also reflects gestalt, problem solving, planning and general ability. Factor analysis has demonstrated that Object Assembly loads highly in the domain of perceptual organisation (Crawford, 1992) and is regarded as the best measure of visuo-spatial functioning in the WAIS battery (Lezak 1995). The test is timed and influenced by visuo-motoric speed and dexterity with high scores dependent upon speed and gestalt perceptual ability. Some designs patterns are harder than others with design number
'7' being particularly more difficult to construct due to the use of split block patterns only and is therefore less amenable to trial and error construction.

Poor performance on Block Design is associated with post-parietal dysfunction (Warrington, James & Maciejewski, 1986) particularly in the right hemisphere; and with parietal dysfunction in the left hemisphere (McFie, 1975). Both right and left hemispheric damage is associated with more errors made on the side of the design contralateral to the side of the lesion. Errors made on the top and bottom of the designs may reflect upper visual field and temporal dysfunction and lower visual field and parietal dysfunction respectively.

**WAIS -R Object Assembly.**

Object Assembly is a jigsaw test of perceptual organisation requiring the ability to synthesise parts into whole quickly and translate via rapid hand movements. It is a relatively simple timed task which does not rely on general ability. It reflects visual organisation and motor response as much as it reflects visuo-spatial ability.

The test is regarded as being sensitive to parietal lesions, particularly in the right hemisphere. Differentiation between hemispheric lesion has been found by noting responses to matching. Right hemispheric lesions are associated with the individual matching by surface detail whereas left hemispheric lesions are reflected by joining jigsaw pieces according to edge contours. Such observations were not made in the present study.
Rey Complex Figure Test - Copy.

The copying component of this test discussed above provides qualitative measure of conceptualisation and gestalt with differentiation demonstrated between hemispheric lesions. Lesions which are associated with poor spatial relations reflect parieto-occipital dysfunction and associated with poor copying reflect frontal lobe dysfunction. The participant's method of drawing may be observed and recorded by the use of different coloured pencils or directions taken. Left hemispheric lesions is associated with fragmented design which may be smaller than normal on recall. Right hemispheric lesions are associate with omissions and neglect.

WAIS-R Picture Completion.

The Picture Completion subtest from the WAIS-R is a brief test of visual reasoning and knowledge.
Ethical approval for this study was sought and granted from both Grampian Health Board / University of Aberdeen Ethics Committee and Lothian Health Board Ethics Committee. Following a postal opt-in procedure (Appendices) participants in the clinical groups were sent the following questionnaires-

- Eating Disorder Inventory-2 (EDI2).
- Bulimic Investigatory Test Edinburgh (BITE).
- Hospital Anxiety and Depression Scales (HADS).
- Delusions -Symptoms -States- Inventory / Neurotic Symptoms (DSSI/NS).

Participants in Group C (Control) were given the above questionnaires with the exception of the BITE questionnaire. Following receipt and analysis of the questionnaire responses, suitable participants were invited to attend for testing. Diagnostic criteria in the clinical groups were established through liaison with clinicians and from the results of self-report questionnaires.

The results of the EDI-2 scores for the participants recruited from self-help groups were compared with the EDI-2 clinical norms. The BITE results were compared with the results of a large eating disorder sample collated by Grampian Eating Disorder Service.

To avoid the confounding factors of alcohol and other recreational drugs, participants were asked not to drink alcohol nor take recreational drugs in the 24 hour
period before testing. Where appropriate, participants were also requested to refrain from strenuous exercise and purging behaviours in the 12 hour period prior to testing.

All participants were requested to have eaten a light snack prior to testing although this was not controlled for during the study. Participants in the clinical groups were also invited to keep a 48 hour diary of eating and related clinical behaviours.

Cognitive testing took place in one session lasting approximately 1 hour and 20 minutes. Testing was carried out at various times of the day and the possibility of differential effects is acknowledged. The testing environments were quiet and free from interruptions or distractions. All participants were required to read and complete a standardised consent form prior to testing (Appendices). Each participant was also asked to provide the following details:

1. Weight and height (for the computation of Body Mass Index).
2. The use of psychotrophic medication or other relevant medication.
3. Awareness of birth complication.

The limitations of ascertaining potential perinatal factors through retrospective report is acknowledged.

In light of the evidence for differential cognitive test performance during the menstrual cycle (Coates, 1996), participants were also asked to provide the current phase of their menstrual cycle. However, the data was confounded by amenorrhoea
in the majority of participants with anorexia and with menstrual irregularities in participants with bulimia nervosa. Insufficient data was available for analysis.

**Presentation order of cognitive tests:**

The cognitive tests, including a mid-test measure of state anxiety (STAIS-S) were presented in a fixed order as follows:

National Adult Reading Test - Revised (NART-R)
Logical Memory I. (WMS-R)
Reitan Trail Making Tests A and B.
Block Design (WAIS-R).
Mental Control (WMS-R).
Rey Complex Figure Test (RCFT) - Copy.
Verbal Fluency (COWA).
RCFT - 3 min Delay Trial.
Digit Span and Visual Memory Span (WMS-R).
Object Assembly (WAIS-R).
Logical Memory II (WMS-R)
Verbal Paired Associates I (WMS-R).
Modified Stroop Test
Digit Symbol (WAIS-R).
Rivermead Picture Recognition subtest- Presentation Trial (Extended version)
STAI-S Questionnaire
Rivermead Picture Recognition subtest - Recognition Trial (Rivermead).
 Mazes (WISC III).
Paced Auditory Serial Addition Test.
Verbal Pairs Associates II (WMS-R)
Picture Completion (WAIS-R).

Following test administration all subjects were given the opportunity to discuss their test performance and given feedback where appropriate.
3d. RESULTS.

Descriptive data, test scores and questionnaire results were analysed using SPSS 6.1 release for windows. Due to the considerable number of comparisons involved in multivariate analyses, alpha was set at 0.01 for non-predicted results. However, for Hypothesis Numbers 1 and 2 alpha was set at 0.05.

For within-group correlational analyses and the analysis of questionnaire data, alpha was set at 0.01. Following Parry (1989), two-tailed tests were used throughout.

Although the setting of directional hypotheses suggested a priori comparisons and thus individual t tests, univariate analyses was carried using analysis of variance with post hoc testing carried out by the Scheffe Test for multiple comparisons. This approach avoided numerous univariate analyses and thus the possibility of Type I errors (Howell, 1992; Bryman and Cramer, 1997).

On screening test scores for dispersion, several test scores were found to be skewed and failed to conform to the normal distribution. Whilst non-parametric tests are usually regarded as the most appropriate medium for analysis of such data, this rule of thumb is not without dissenting voices. Several authors suggest that parametric tests may be used for non-normally distributed data provided that the group sample sizes are equal and that the principles of normal distribution and homogeneity of variance between group means are not seriously compromised (Howell, 1992; Kinnear & Gray, 1996; Bryman & Cramer, 1997). Bryman and Cramer (1997) further suggest that running both parametric and non-parametric test
will identify any trend towards Type I error. When both parametric and non-parametric tests were carried out on the relevant test score results, no differences were established in outcome for significant and non-significant results. The results presented therefore are based on the more powerful parametric testing with Pillais $F$ value used for its robustness for mild violations of the principles of analyses of variance (Norusis, 1994). For questionnaire score analyses and for categorical data however, non-parametric tests were used. Non-parametric tests were also used when analysing the use of antidepressant medication as an independent variable where samples sizes were small and unequal.

Similarly, non-parametric tests were used for the post hoc analyses of two non-standard test measures: the number of tracking errors made on both the Reitan Trail Making Tests and the number of design failures on the WAIS-R Block Design Subtest.
Results of BITE Questionnaire (Table 2):

Thirteen per cent of Group AN reported binge eating and purging behaviours and reached the cut-off score for the BITE Symptom Score for Bulimia (Table 2: below). All participants in Group BN reached the cut-off level for the BITE Symptom Score and 93 per cent of this group reached the cut-off level for the BITE Severity Score. Only 13 per cent of Group BN were not involved in purging behaviours.

Table 2. BITE Questionnaire - Clinical Groups.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>MW / Chi-Sq.</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Binge eating frequency (per week)</td>
<td>1.458</td>
<td>4.892</td>
<td>U 100.5</td>
<td>AN &lt; BN</td>
</tr>
<tr>
<td>Purging behaviours</td>
<td>23%</td>
<td>87%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Purging - Vomiting (per week)</td>
<td>1.892</td>
<td>4.958</td>
<td>U 143.5</td>
<td>AN &lt; BN</td>
</tr>
<tr>
<td>Purging - Laxatives (per week)</td>
<td>0.492</td>
<td>0.383</td>
<td>U 437</td>
<td>n.s.</td>
</tr>
<tr>
<td>Purging - Diuretics (per week)</td>
<td>0.083</td>
<td>0.17</td>
<td>U 435</td>
<td>n.s.</td>
</tr>
<tr>
<td>BITE Symptom Score</td>
<td>13.458</td>
<td>24.929</td>
<td>U 43.5</td>
<td>AN &lt; BN</td>
</tr>
<tr>
<td>% with cut-off score for pathology</td>
<td>13%</td>
<td>100%</td>
<td>Chi-Sq 41.097</td>
<td>AN &lt; BN</td>
</tr>
<tr>
<td>BITE Severity Score</td>
<td>4.667</td>
<td>10.714</td>
<td>U 116.000</td>
<td>AN &lt; BN</td>
</tr>
<tr>
<td>% with cut-off score for pathology</td>
<td>13%</td>
<td>93%</td>
<td>Chi-Sq 33.830</td>
<td>AN &lt; BN</td>
</tr>
</tbody>
</table>

p"p <0.010

p**p <0.001
Results of HADS and DSSI/NS Questionnaires (Tables 3 and 4):

Descriptive data for the three groups on reported levels of depression and general anxiety as measured by the HADS are illustrated in Table 3 below.

Group scores on the HADS were not significant between the clinical groups with 70 per cent of Group AN and 57 per cent of Group BN scoring at the level of 'caseness'. A further 13 per cent and 20 per cent respectively scored in the range of subclinical anxiety cases (Table 3). On the HADS Anxiety Subscale the Control group reported significantly lower levels of general anxiety than both clinical groups (Chi Sq 2.87: 58.126, p<0.01).

Table 3. Group Scores: Hospital Anxiety and Depression Scales.

<table>
<thead>
<tr>
<th>Scale</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
<th>Chi-Sq</th>
<th>p value</th>
<th>M W</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS Anxiety</td>
<td>12.821</td>
<td>12.862</td>
<td>2.367</td>
<td>58.126</td>
<td>AN, BN &gt; C</td>
<td></td>
</tr>
<tr>
<td></td>
<td>sd 3.55</td>
<td>sd 4.414</td>
<td>sd 1.326</td>
<td>p = 0.000**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subclinical cases</td>
<td>13%</td>
<td>20%</td>
<td>0%</td>
<td>68.116</td>
<td>AN, BN &gt; C</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p = 0.000**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical cases</td>
<td>70%</td>
<td>57%</td>
<td>0%</td>
<td>37.405</td>
<td>AN, BN &gt; C</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p = 0.000**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HADS Depression</td>
<td>8.679</td>
<td>7.759</td>
<td>0.633</td>
<td>54.55</td>
<td>AN, BN &gt; C</td>
<td></td>
</tr>
<tr>
<td></td>
<td>sd 4.408</td>
<td>sd 4.293</td>
<td>sd 0.890</td>
<td>p = 0.000**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subclinical cases</td>
<td>27%</td>
<td>16%</td>
<td>0%</td>
<td>23.87</td>
<td>AN, BN &gt; C</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p = 0.000**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical Cases</td>
<td>33%</td>
<td>27%</td>
<td>0%</td>
<td>10.33</td>
<td>AN, BN &gt; C</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>p = 0.000**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < 0.010
A considerable number of Group AN (33 per cent) and of Group BN (26.7 per cent) scored on the *depression* subscale of the HADS for 'caseness' (Table 3). A further 27 per cent of Group AN and 10 per cent of Group BN scored at a subclinical level. Nonetheless, scores at this level may represent dysthymia and thus a degree of depressive symptomatology. Significant between-group differences in the clinical groups were neither established for levels of depression nor as noted above, for the numbers of participants prescribed anti-depressant medication. The prescription of anti-depressant medication was associated with higher HADS Depression scores. The Control group as would be expected, reported zero caseness for depression.

Analyses of the subscale scores from the DSSI/NS (Table 4: overleaf) revealed no significant group differences between the clinical groups, although significance between the clinical groups and Group C was established on each of the DSSI/NS subscales. Whilst Group AN and Group BN means suggest relatively low levels of pathology, 27 per cent of Group AN and 23 per cent of Group BN scored for caseness in the Rumination Symptom subscale (cut-off score = 4 or >). Further, 37 per cent of Group AN reached clinical levels of pathology on the Rumination Severity subscale (Table 4).
## Table 4. Group Scores on the DSSI / Neurotic Symptoms.

<table>
<thead>
<tr>
<th>DSSI/NS Subscale</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
<th>Chi-Sq</th>
<th>MW p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conversion Symptom Score</td>
<td>(Cvs)</td>
<td>1.25</td>
<td>1.069</td>
<td>0.1</td>
<td>19.804</td>
</tr>
<tr>
<td></td>
<td>sd 1.296</td>
<td>sd 1.252</td>
<td>sd 0.305</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Severity Score</td>
<td>(Cvt)</td>
<td>1.536</td>
<td>1.621</td>
<td>0.233</td>
<td>17.098</td>
</tr>
<tr>
<td></td>
<td>sd 1.795</td>
<td>sd 1.741</td>
<td>sd 0.43</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Compulsion Symptom Score</td>
<td>(Cps)</td>
<td>1.536</td>
<td>1.621</td>
<td>0.233</td>
<td>17.098</td>
</tr>
<tr>
<td></td>
<td>sd 1.795</td>
<td>sd 1.741</td>
<td>sd 0.43</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Severity Score</td>
<td>(Cpt)</td>
<td>1.926</td>
<td>2.103</td>
<td>0.103</td>
<td>15.99</td>
</tr>
<tr>
<td></td>
<td>sd 2.495</td>
<td>sd 2.682</td>
<td>sd 0.521</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Phobic Symptom Score</td>
<td>(Ps)</td>
<td>1.179</td>
<td>1.172</td>
<td>0.067</td>
<td>25.891</td>
</tr>
<tr>
<td></td>
<td>sd 1.249</td>
<td>sd 1.311</td>
<td>0.254</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Severity Score</td>
<td>(Pt)</td>
<td>1.815</td>
<td>2.035</td>
<td>0.138</td>
<td>22.934</td>
</tr>
<tr>
<td></td>
<td>sd 2.167</td>
<td>2.035</td>
<td>0.516</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Ruminaton Symptom Score</td>
<td>(Rs)</td>
<td>2.86</td>
<td>2.621</td>
<td>0.233</td>
<td>30.21</td>
</tr>
<tr>
<td></td>
<td>sd 2.652</td>
<td>1.953</td>
<td>sd 0.430</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Severity Score</td>
<td>(Rt)</td>
<td>3.778</td>
<td>3.897</td>
<td>0.241</td>
<td>28.792</td>
</tr>
<tr>
<td></td>
<td>sd 3.916</td>
<td>3.529</td>
<td>sd 0.435</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Dissociation Symptom Score</td>
<td>(Ds)</td>
<td>0.893</td>
<td>1</td>
<td>0</td>
<td>18.88</td>
</tr>
<tr>
<td></td>
<td>sd 1.423</td>
<td>sd 1.254</td>
<td>sd 0</td>
<td></td>
<td>p = 0.000''</td>
</tr>
<tr>
<td>Severity Score</td>
<td>(Dt)</td>
<td>1.482</td>
<td>1.828</td>
<td>0</td>
<td>18.677</td>
</tr>
<tr>
<td></td>
<td>sd 2.440</td>
<td>2.234</td>
<td>sd 0</td>
<td></td>
<td>p = 0.000''</td>
</tr>
</tbody>
</table>

**p < 0.010**
On the EDI-2, score differences between the clinical groups and Group C were significant on all subscales (Table 5: below).

Table 5. Group Scores: Eating Disorder Inventory 2.

<table>
<thead>
<tr>
<th>EDI 2 Subscale</th>
<th>Group AN (Anorexia) N = 28</th>
<th>Group BN (Bulimia) N = 23</th>
<th>Group C (Control) N = 30</th>
<th>Chi-Sq</th>
<th>MW</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drive For Thinness (DT)</td>
<td>14</td>
<td>17.217</td>
<td>2.3</td>
<td>51.667</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 6.237</td>
<td>sd 4.101</td>
<td>sd 2.366</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulimia (B)</td>
<td>2.75</td>
<td>13.035</td>
<td>0.167</td>
<td>59.088</td>
<td>BN &gt; AN, C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 2.465</td>
<td>sd 3.764</td>
<td>sd 1.172</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Dissatisfaction (BD)</td>
<td>18.43</td>
<td>21.93</td>
<td>5.9</td>
<td>53.419</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 7.260</td>
<td>sd 5.110</td>
<td>sd 0.481</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ineffectiveness (I)</td>
<td>12.857</td>
<td>12.345</td>
<td>1.033</td>
<td>49.814</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 8.445</td>
<td>sd 6.852</td>
<td>sd 1.791</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perfectionism (P)</td>
<td>8.321</td>
<td>6.172</td>
<td>2.267</td>
<td>25.13</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 4.091</td>
<td>sd 5.036</td>
<td>sd 3.129</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interpersonal Distrust</td>
<td>5.429</td>
<td>5.552</td>
<td>1.3</td>
<td>26.34</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 3.872</td>
<td>sd 4.188</td>
<td>sd 1.878</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interoceptive Awareness</td>
<td>10.393</td>
<td>12.069</td>
<td>0.233</td>
<td>57.27</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 7.249</td>
<td>sd 6.216</td>
<td>sd 0.774</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maturity Fears (MF)</td>
<td>4.214</td>
<td>4.379</td>
<td>0.4</td>
<td>39.883</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 4.333</td>
<td>sd 3.866</td>
<td>sd 0.814</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascetism (A)</td>
<td>9.143</td>
<td>8.621</td>
<td>0.933</td>
<td>57.582</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 5.053</td>
<td>sd 6.621</td>
<td>sd 1.173</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impulse Regulation (IR)</td>
<td>6.5</td>
<td>6.855</td>
<td>0.3</td>
<td>43.236</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 6.04</td>
<td>sd 5.79</td>
<td>sd 0.794</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social Insecurity (SI)</td>
<td>9.786</td>
<td>8.899</td>
<td>1.033</td>
<td>50.748</td>
<td>AN, BN &gt; C</td>
<td>p = 0.000**</td>
</tr>
<tr>
<td></td>
<td>sd 5.36</td>
<td>sd 4.354</td>
<td>sd 1.671</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**p < 0.010
Significant between-group difference in the clinical groups was evident for only one subscale from the EDI-2 Questionnaire - *Bulimia*, with mean scores being predictably higher for Group BN ($U = 59, p<0.01$). The relatively low score for group AN is of some interest given that 23 per cent of this group reported purging behaviours and 13 per cent reached the cut-off level for pathology on the BITE Symptom and Severity Scores. The equivalence of group means between the clinical groups on the EDI 2 Perfectionism subscale is notable given the clinical association between anorexia nervosa and personality traits of perfectionism. Both group means however, fall within the range of the EDI 2 norms although Group BN means fell at the bottom end of the range and are equivalent to female college norms.

**Cognitive deficits (Tables 6 and 7).**

The analysis of test scores to yield group frequencies of cognitive deficits was based upon individual score comparisons with published norms on 73 per cent of the cognitive tests administered. Individual test scores at between one and two standard deviations below the published norms were collated to illustrate differential group performances on individual tests (Table 6:overleaf). However test scores were analysed principally to determine the presence of test scores at two standard deviations below the published norm for the individual's age and educational background (Table 7). Test deficits at this level are regarded by neuropsychologists as clinically significant and may represent neuropsychological impairment when considered in conjunction with a clinical history of cognitive dysfunction.
Table 6. Group Levels of Cognitive Deficit.

<table>
<thead>
<tr>
<th></th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
<th>Chi-Sq</th>
<th>Mann Whitney p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficit 1-2 sd</td>
<td>2.033</td>
<td>2.333</td>
<td>0.4</td>
<td>16.998</td>
<td>AN, BN &gt; C</td>
</tr>
<tr>
<td>sd</td>
<td>2.426</td>
<td>2.20</td>
<td>0.62</td>
<td>p = 0.002**</td>
<td></td>
</tr>
<tr>
<td>% deficit 1-2 sd</td>
<td>66.70%</td>
<td>70%</td>
<td>33%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficit 2&gt; sd</td>
<td>0.467</td>
<td>0.367</td>
<td>0.056</td>
<td>5.605</td>
<td>n.s.</td>
</tr>
<tr>
<td>sd</td>
<td>0.900</td>
<td>0.850</td>
<td>0.254</td>
<td>p = 0.061</td>
<td></td>
</tr>
<tr>
<td>% deficit 2&gt; sd</td>
<td>30%</td>
<td>23.30%</td>
<td>6.70%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**p < 0.010

A test score performance of between one and two standard deviations below published norms was demonstrated by 67 per cent of Group AN, 70 per cent of Group BN and 33 per cent Group C (Table 6). That a third of the control group demonstrated this level of test performance may raise questions concerning matching variables, although closer scrutiny of the data reveals that 50 per cent of the test scores at this level were accounted for by one cognitive test result (Verbal Fluency).

Table 6 illustrates that 30 per cent of Group AN, 23 per cent of Group BN and seven per cent of Group C demonstrated test score deficits at an impaired level. The deficits computed for Group C were accountable by impaired performance on the Rey Complex Figure Copy Test (Table 7:overleaf).
### Table 7. Group Levels of Cognitive Deficit by Domain

<table>
<thead>
<tr>
<th>Domain and Cognitive Test</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1-2 sd</td>
<td>≥2 sd</td>
<td>1-2 sd</td>
</tr>
<tr>
<td><strong>ATTENTION:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digit Span Forwards</td>
<td>7%</td>
<td>3%</td>
<td>10%</td>
</tr>
<tr>
<td>(Attention - Conc Index)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digit Span Backwards</td>
<td>10%</td>
<td>0%</td>
<td>7%</td>
</tr>
<tr>
<td>(Attention - Conc Index)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual Memory Span Forwards</td>
<td>17%</td>
<td>0%</td>
<td>17%</td>
</tr>
<tr>
<td>(Attention - Conc Index)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual Memory Span Backwards</td>
<td>17%</td>
<td>3%</td>
<td>26%</td>
</tr>
<tr>
<td>(Attention - Conc Index)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PASAT Trial No. 3</td>
<td>13%</td>
<td>0%</td>
<td>27%</td>
</tr>
<tr>
<td>PASAT Trial No. 4</td>
<td>13%</td>
<td>7%</td>
<td>30%</td>
</tr>
<tr>
<td>Trail Making Test A</td>
<td>10%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>10%</td>
<td>0%</td>
<td>10%</td>
</tr>
<tr>
<td><strong>LEARNING AND MEMORY:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Logical Memory I</td>
<td>7%</td>
<td>3%</td>
<td>7%</td>
</tr>
<tr>
<td>Logical Memory II</td>
<td>17%</td>
<td>0%</td>
<td>7%</td>
</tr>
<tr>
<td>RCFT- Delayed Recall</td>
<td>3%</td>
<td>10%</td>
<td>20%</td>
</tr>
<tr>
<td><strong>EXECUTIVE FUNCTIONING:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verbal Fluency</td>
<td>13%</td>
<td>0%</td>
<td>13%</td>
</tr>
<tr>
<td>Trail Making Test B</td>
<td>0%</td>
<td>3%</td>
<td>0%</td>
</tr>
<tr>
<td><strong>VISUOSPATIAL/CONSTRUCT:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Object Assembly</td>
<td>20%</td>
<td>0%</td>
<td>27%</td>
</tr>
<tr>
<td>Block Design</td>
<td>20%</td>
<td>0%</td>
<td>17%</td>
</tr>
<tr>
<td>Picture Completion</td>
<td>7%</td>
<td>3%</td>
<td>10%</td>
</tr>
<tr>
<td>RCFT - Copy</td>
<td>10%</td>
<td>13%</td>
<td>0%</td>
</tr>
</tbody>
</table>
Table 7 also illustrates that the highest number of deficits occurred on the final and quickest PASAT Trial (Trial 4) with almost a third of Group BN demonstrating a deficit test score.

It is worth noting that many of the test score deficits established were accountable to a small number of participants in the clinical groups with impaired test performance demonstrated over the range of neuropsychological domains. Indeed one participant accounted for 54.5 per cent of the anorexic group deficits (at two standard deviations). Whilst it would be informative to explore this further by way of individual case analyses, such a procedure was outside the remit granted by ethical approval for this study.

Test scores analyses.

A multivariate analysis of variance (MANOVA) was computed to reveal significant overall group differences (Pillais $F_{36, 140}: 2.247, p < 0.01$). The results of univariate analyses for each neuropsychological domain are illustrated in Tables 8 to 16 in the following pages. In general, the mean scores for the control group were higher (or on some test scores, appropriately lower) than those of the eating disorder groups. A wide variation in scores was observed in both clinical groups. Overall, significant group differences on cognitive test results were found between the control group and the clinical groups on eight from eighteen (44.4 per cent) in relation to Group AN and on seven from eighteen (38.8 per cent) in relation to Group BN. Significant group difference between the clinical groups was established on only one test result (Verbal Pairs Associates I - Learning & Memory Domain - Table14).
Further post-hoc analyses were carried out on several test measures - the frequency of tracking errors made on the Reitan Trail Making Tests (as a measure of speed versus error); the number of design failures on the Block Design Test and the percentage of prose material retained between groups on Logical Memory II. The results of these analyses will be discussed in the relevant sections below.

Because significant between-group differences were found, correlational analysis was carried for within-groups using Pearson's Coefficient and with alpha set at $p<0.01$. Only those correlations found to be significant at this level will be discussed. The strength of correlation used is defined by Cohen and Holliday's (1982) as follows: Pearson $r < 0.4 = \text{weak}$; Pearson $r$ 0.4 to 0.69 = modest; Pearson $r$ 0.70 to 0.89 = high).

The cognitive test results are shown by neuropsychological domain as follows.
Test Results - Attention Domain (Table 8):

In the domain of Attention, Group C performed better than both clinical groups on the majority of the test measures.

Table 8. ANOVA: Cognitive Tests in the Attention Domain.

<table>
<thead>
<tr>
<th>Test</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
<th>F (df)</th>
<th>p value</th>
<th>Post Hoc</th>
<th>Scheffe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attention-Concentration Index (WMS-R)</td>
<td>98.3 sd 13.789</td>
<td>98.4 sd 13.377</td>
<td>105.567 sd 10.183</td>
<td>F(2,87) 4.456</td>
<td>p = 0.014*</td>
<td>BN &lt; C</td>
<td></td>
</tr>
<tr>
<td>PASAT Mean Score</td>
<td>35.179 sd 9.233</td>
<td>32.724 sd 7.225</td>
<td>41.527 sd 4.870</td>
<td>F(2.86) 11.573</td>
<td>p = 0.000**</td>
<td>AN, BN &lt; C</td>
<td></td>
</tr>
<tr>
<td>Modified Stroop Test</td>
<td>110.13 sd 2.732</td>
<td>109.93 sd 2.532</td>
<td>111.43 sd 1.005</td>
<td>F(2.87) 4.001</td>
<td>p = 0.022*</td>
<td>BN &lt; C</td>
<td></td>
</tr>
<tr>
<td>Digit Symbol (WAIS-R)</td>
<td>61.333 sd 10.263</td>
<td>61.8 sd 9.900</td>
<td>67.8 sd 6.900</td>
<td>F(2.87) 4.617</td>
<td>p = 0.012*</td>
<td>AN, BN &lt; C</td>
<td></td>
</tr>
</tbody>
</table>

Significant between-group differences were established on four from five test results:

1. On the Attention-Concentration Index (WMS-R) significance was established between Group C and Group BN (F2,87 : 4.46 , p<0.01).
2. On the Paced Auditory-Serial Addition Test, significance was established between Group C and both the clinical groups \((F_{2,86}: 11.57, p < 0.01)\).

3. On the Modified Stroop Test, significance was established between Group C and Group BN \((F_{2,87}: 4, p < 0.05)\)

4. On the WAIS-R Digit Symbol Subtest, significance was established between Group C and both clinical groups \((F_{2,87}: 4.62, p < 0.05)\).

The highest number of test score deficits in the clinical groups occurred on the PASAT Trial Number 4 where seven per cent of Group AN demonstrated a defective test score (Table 7).

In Group AN, there were no significant correlations between test scores and affective variables (Table 9:overleaf). However, both the Reitan Trail Making Test A and the Modified Stroop Test were correlated with duration of eating disorder \((\text{Pearson } r = 0.5 \text{ and Pearson } r = -0.5 \text{ respectively: Table 10})\).

Within-group correlational analysis of Group BN (Tables 11 and 12) revealed only one significant relationship, where the use of laxatives was modestly and inversely associated with the Modified Stroop Test \((\text{Pearson } r = -0.48: \text{ Table 12})\).

Analysis of the relationships between variables in Group C scores (Table 13) revealed only one significant relationship with Trails Making Test A being modestly associated with HADS Anxiety Subscale scores \((\text{Pearson } r = 0.47)\).
Summary of results in the Attention Domain:

In the neuropsychological domain of attention, Group C performed better than both clinical groups on all test measures excepting Trail Making Test A. Statistical significance was accepted between Group C and Group AN on two from five test results (PASAT Mean Score and Digit Symbol). These results support Hypothesis Number 1.

Significant group differences between Group C and Group BN were established in four from five results (Attention-Concentration Index, PASAT Mean Score, Digit Symbol and the Modified Stroop Test). Group BN also demonstrated the highest number of deficits (n=38) compared to Group AN (n=29 deficits). These results provide strong evidence for attentional difficulties in Group BN and this evidence supports Hypothesis Number 2.

Correlational analysis revealed few relationships and where established, occurred between test scores and clinical variables rather than affective variables. Thus support for Hypothesis No.3 was not evident on correlational analyses.
Table 9. Group AN (Anorexia): Correlations between cognitive test scores and state anxiety, HADS, and DSS/INS Questionnaire scores.

<table>
<thead>
<tr>
<th>Test</th>
<th>STAIS-S</th>
<th>HADS</th>
<th>HADS</th>
<th>DSS/INS</th>
<th>DSS/INS</th>
<th>DSS/INS</th>
<th>DSS/INS</th>
<th>DSS/INS</th>
<th>DSS/INS</th>
<th>DSS/INS</th>
<th>DSS/INS</th>
</tr>
</thead>
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<td>-0.25</td>
<td>-0.33</td>
<td>-0.01</td>
<td>0.25</td>
<td>-0.35</td>
<td>-0.18</td>
<td>-0.15</td>
<td>-0.04</td>
<td>-0.07</td>
<td>-0.14</td>
</tr>
<tr>
<td>PASAT</td>
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<td>-0.04</td>
<td>-0.11</td>
<td>-0.22</td>
<td>-0.19</td>
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<td>-0.02</td>
<td>-0.2</td>
<td>-0.2</td>
<td>-0.14</td>
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</tr>
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<td>0.32</td>
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<td>0.2</td>
<td>0.06</td>
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<td>0.24</td>
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<td>0.3</td>
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<tr>
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<td>-0.18</td>
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<td>0.13</td>
<td>0.11</td>
<td>-0.33</td>
<td>-0.17</td>
<td>-0.32</td>
<td>-0.19</td>
<td>-0.44*</td>
<td>-0.62**</td>
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<tr>
<td>Verbal Pairs Ass. I</td>
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<td>0.06</td>
<td>-0.12</td>
<td>-0.19</td>
<td>-0.03</td>
<td>0.11</td>
<td>0.18</td>
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<td>0.32</td>
<td>-0.67**</td>
<td>-0.42*</td>
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<td>-0.44</td>
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<td>-0.38</td>
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<td>-0.39*</td>
<td>0.4</td>
<td>-0.59*</td>
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<td>0.48**</td>
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<td>-0.67**</td>
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<td>-0.03</td>
<td>0.07</td>
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<td>0.02</td>
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<td>-0.24</td>
</tr>
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<td>-0.54**</td>
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<td>0</td>
<td>-0.08</td>
<td>-0.24</td>
<td>-0.4*</td>
<td>-0.26</td>
<td>-0.1</td>
<td>-0.17</td>
</tr>
<tr>
<td>Trail A &amp; B Errors</td>
<td>0.2</td>
<td>0.13</td>
<td>0.07</td>
<td>0.24</td>
<td>0.1</td>
<td>-0.18</td>
<td>-0.02</td>
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<td>0.21</td>
<td>-0.02</td>
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<td>Block Des. Failures</td>
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<td>0.66**</td>
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<td>0.41**</td>
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*p<0.05  **p<0.01
Table 10. Group AN (Anorexia): Correlations between cognitive test scores, clinical variables, EDI-2 and BITE Questionnaire scores.

<table>
<thead>
<tr>
<th>Test</th>
<th>EDI-2 Perfectionism</th>
<th>EDI-2 Social Ins.</th>
<th>EDI-2 Impulse Reg.</th>
<th>BITE Symptom</th>
<th>BITE Severity</th>
<th>BMI</th>
<th>Duration of disorder</th>
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<td>-0.2</td>
<td>0.04</td>
<td>-0.46*</td>
<td>-0.41*</td>
<td>-0.18</td>
<td>0</td>
</tr>
<tr>
<td>PASAT Mean</td>
<td>0</td>
<td>-0.18</td>
<td>-0.04</td>
<td>-0.19</td>
<td>-0.22</td>
<td>-0.22</td>
<td>-0.18</td>
</tr>
<tr>
<td>Trails Test A</td>
<td>0.05</td>
<td>0.42*</td>
<td>0.11</td>
<td>0.14</td>
<td>0.15</td>
<td>-0.14</td>
<td>0.6**</td>
</tr>
<tr>
<td>Stroop</td>
<td>0.27</td>
<td>-0.18</td>
<td>0.03</td>
<td>0.17</td>
<td>0.09</td>
<td>0.25</td>
<td>-0.06*</td>
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<td>-0.45*</td>
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<td>-0.12</td>
<td>0.01</td>
<td>0.13</td>
<td>-0.04</td>
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<td>Logical Memory I</td>
<td>0.13</td>
<td>0.05</td>
<td>0.05</td>
<td>-0.04</td>
<td>-0.15</td>
<td>-0.08</td>
<td>0</td>
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<tr>
<td>Logical Memory II</td>
<td>0.16</td>
<td>0.04</td>
<td>0.01</td>
<td>-0.13</td>
<td>0</td>
<td>0</td>
<td>-0.11</td>
</tr>
<tr>
<td>Verbal Pairs Ass. I</td>
<td>0.26</td>
<td>-0.08</td>
<td>0.00</td>
<td>0.22</td>
<td>0.21</td>
<td>0.21</td>
<td>0.6**</td>
</tr>
<tr>
<td>Verbal Pairs Ass. II</td>
<td>0.28</td>
<td>-0.16</td>
<td>0.05</td>
<td>-0.14</td>
<td>-0.1</td>
<td>0.16</td>
<td>-0.34</td>
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<td>0.22</td>
<td>0.14</td>
<td>0</td>
<td>0.26</td>
<td>0.01</td>
<td>-0.01</td>
</tr>
<tr>
<td>RCFT Recall</td>
<td>-0.27</td>
<td>-0.39*</td>
<td>-0.35</td>
<td>-0.44*</td>
<td>-0.06</td>
<td>0.12</td>
<td>-0.17</td>
</tr>
<tr>
<td>Verbal Fluency</td>
<td>0.23</td>
<td>-0.35</td>
<td>-0.03</td>
<td>-0.07</td>
<td>-0.33</td>
<td>0.41*</td>
<td>-0.01</td>
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<tr>
<td>Trails Test B</td>
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<td>0.26</td>
<td>-0.02</td>
<td>0.00</td>
<td>-0.07</td>
<td>-0.12</td>
<td>0.29</td>
</tr>
<tr>
<td>Mazes</td>
<td>-0.23</td>
<td>-0.52</td>
<td>-0.1</td>
<td>-0.05</td>
<td>0.19</td>
<td>0.12</td>
<td>-0.62**</td>
</tr>
<tr>
<td>Object Assembly</td>
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<td>-0.33</td>
</tr>
<tr>
<td>Picture Completion</td>
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</tr>
<tr>
<td>RCFT Copy</td>
<td>0.23</td>
<td>-0.39*</td>
<td>-0.08</td>
<td>-0.14</td>
<td>0.27</td>
<td>-0.12</td>
<td>-0.39*</td>
</tr>
<tr>
<td>Trail Errors A &amp; B</td>
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<td>0.15</td>
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<tr>
<td>Block Design Failures</td>
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<td>0.03</td>
<td>0.62**</td>
<td>0.37</td>
<td>0.07</td>
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*p<0.05  **p<0.01
Table 11. Group BN (Bulimia): Correlations between cognitive test scores and state anxiety, HADS and DSSI/NS Questionnaire scores.

<table>
<thead>
<tr>
<th>Test</th>
<th>STAIS-G</th>
<th>HADS Anxiety</th>
<th>HADS Depression</th>
<th>DSSI/NS Convers.</th>
<th>DSSI/NS Compuls</th>
<th>DSSI/NS Phobic</th>
<th>DSSI/NS Reminist</th>
<th>DSSI/NS Dissoc.</th>
<th>DSSI/NS Dissoc.</th>
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<td>-0.36*</td>
<td>-0.22</td>
<td>-0.13</td>
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<td>-0.13</td>
<td>-0.24</td>
<td>-0.29</td>
<td>-0.34</td>
</tr>
<tr>
<td>PASAT Mean</td>
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<td>-0.16</td>
<td>-0.06</td>
<td>-0.3</td>
<td>-0.29</td>
<td>-0.22</td>
<td>-0.11</td>
<td>-0.16</td>
<td>-0.29</td>
</tr>
<tr>
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<td>-0.01</td>
<td>0.08</td>
<td>-0.05</td>
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</tr>
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<td>-0.34</td>
<td>-0.05</td>
<td>0.07</td>
<td>-0.21</td>
<td>-0.14</td>
<td>-0.25</td>
<td>-0.15</td>
<td>-0.36</td>
</tr>
<tr>
<td>Digit Symbol</td>
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<td>-0.27</td>
<td>-0.03</td>
<td>-0.38</td>
<td>-0.29</td>
<td>-0.29</td>
<td>-0.27</td>
<td>-0.34</td>
</tr>
<tr>
<td>Logical Memory I</td>
<td>-0.12</td>
<td>-0.65**</td>
<td>-0.37</td>
<td>-0.50</td>
<td>-0.29</td>
<td>-0.32</td>
<td>-0.29</td>
<td>-0.38</td>
<td>-0.16</td>
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<td>-0.34</td>
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<tr>
<td>Verbal Fluency</td>
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<td>-0.33</td>
<td>-0.35</td>
<td>-0.36</td>
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<td>0.15</td>
<td>0.24</td>
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<td>0.13</td>
<td>-0.25</td>
<td>-0.26</td>
<td>0.07</td>
<td>0.12</td>
<td>0.1</td>
<td>0.16</td>
</tr>
<tr>
<td>Object design</td>
<td>-0.33</td>
<td>-0.38*</td>
<td>-0.04</td>
<td>-0.15</td>
<td>-0.18</td>
<td>0.06</td>
<td>0.01</td>
<td>0.04</td>
<td>0.06</td>
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<td>-0.31</td>
<td>-0.23</td>
<td>-0.34</td>
<td>-0.33</td>
<td>-0.07</td>
<td>-0.04</td>
<td>-0.19</td>
</tr>
<tr>
<td>Picture Completion</td>
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<td>-0.06</td>
<td>0.05</td>
<td>-0.07</td>
<td>-0.01</td>
<td>0.02</td>
<td>0.12</td>
<td>-0.1</td>
<td>-0.09</td>
</tr>
<tr>
<td>RCFT Copy</td>
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<td>-0.29</td>
<td>-0.05</td>
<td>-0.33</td>
<td>-0.26</td>
<td>-0.05</td>
<td>-0.03</td>
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<tr>
<td>Trails A &amp; B Errors</td>
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<td>0.08</td>
<td>0.04</td>
<td>0.05</td>
<td>0.07</td>
<td>0.07</td>
<td>0.05</td>
<td>0.04</td>
</tr>
<tr>
<td>Block Des. Failures</td>
<td>0.66**</td>
<td>0.57*</td>
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<td>0.25</td>
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<td>0.32</td>
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*p<0.05 **p<0.1
Table 12. Group BN (Bulimia): Correlations between cognitive test scores, clinical variables, EDI-2 and BITE Questionnaire scores.

<table>
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<th></th>
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*p<0.05 **p<0.01
Table 13. Group C (Control): Correlations between cognitive test scores and state anxiety, HADS and DSSI/NS Questionnaire scores.

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<th>HADS</th>
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<th>DSSI/NS</th>
<th>DSSI/NS</th>
<th>DSSI/NS</th>
<th>DSSI/NS</th>
<th>DSSI/NS</th>
<th>DSSI/NS</th>
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*p<0.05  **p<0.01
Test results - Learning and Memory Domain (Table 14):

In the domain of Learning and Memory, Group C performed better than both clinical groups on all test measures with significance established on five of the six test results.

Table 14.

<table>
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<tr>
<th>Test</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
<th>F(df)</th>
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<td>22.583</td>
<td>28.333</td>
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<td>sd 6.057</td>
<td>sd 5.934</td>
<td>sd 4.196</td>
<td>p = 0.000**</td>
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<td>20.567</td>
<td>19.333</td>
<td>26.133</td>
<td>F(2.87) 11.37</td>
<td>AN, BN &lt; C</td>
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<tr>
<td></td>
<td>sd 6.323</td>
<td>sd 6.541</td>
<td>sd 4.599</td>
<td>p = 0.000**</td>
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<td>20.367</td>
<td>20.867</td>
<td>F(2.87) 6.974</td>
<td>AN &lt; BN, C</td>
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<td>2.622</td>
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<td>p = 0.002**</td>
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<td>7.8</td>
<td>8</td>
<td>F(2.87) 5.024</td>
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<td>sd 0.774</td>
<td>sd 0.464</td>
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<td>F(2.87) 4.64</td>
<td>n.s.</td>
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<td>sd 2.662</td>
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<td>F(2.87) 9.21</td>
<td>AN, BN &lt; C</td>
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<td></td>
<td>sd 7.452</td>
<td>sd 6.553</td>
<td>sd 5.832</td>
<td>p = 0.000**</td>
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</table>

On Logical Memory I, significance was established between Group C and both clinical groups ($F_{2,87}: 8.61, p<0.01$).

On Logical Memory II, significance was established between Group C and both clinical groups ($F_{2,87}: 11.37, p<0.01$).
On Verbal Pairs Associates I, significance was established between Group C, Group BN and Group AN (F2,87: 6.97, p<0.01).

On Verbal Pairs Associates II, significance was established between Group C and Group AN (F2,87:5.02, p<0.05).

On the Rey Complex Figure Test (Delayed), significance was established between Group C and both clinical groups (F2,87: 9.2, p<0.01).

The highest number of test deficits in this domain occurred on the Rey Complex Figure Test - Delayed Recall with 10 per cent of Group AN and seven per cent of Group BN scoring at an impaired level. Seven per cent of Group BN also scored at an impaired level on Logical Memory I (Table 7).

Correlational analysis of Group AN scores revealed very few statistical associations (Tables 9 and 10). In this group, scores on the HADS Anxiety Subscale and DSSI/NS Rumination (Severity Subscale) were inversely associated with Logical Memory II (Pearson r = -0.49, Pearson r = -0.53 respectively). The DSSI/NS scores also revealed correlations between the Conversion Severity Subscale and Verbal Pairs Associates Learning II (Pearson r = -0.57); between the Compulsion Symptoms Subscale and the Rey Complex Figure Test - Delayed Recall (Pearson r = 0.48). Only one of the clinical variables, 'duration of eating disorder', was associated with test scores - Verbal Pairs Associates Learning I (Pearson r = -0.52) and Picture Recognition (Pearson r = -0.51).

Correlational analyses within Group BN found the HADS Anxiety scores to be related to Logical Memory Tests, I and II (Pearson r = -0.55; Pearson r = -0.58).
respectively - Table 11). Similarly scores on the the DSSI/NS Rumination subscale were also associated with Logical Memory Tests I and II (Pearson r = -0.54; Pearson r = -0.51 respectively). Only one clinical variable demonstrated a strong and rather odd positive association between the use of diuretics and Verbal Pair Associates Learning II (Pearson r = 0.73 - Table 12).

Correlational analysis of the control Group scores revealed no significant relationships (Table 13).

**Summary of results in the Learning and Memory Domain.**

Group AN demonstrated significantly poorer test performance relative to Group C on five from six tests in this domain with 10 per cent of the group scoring at a defective level on the delayed recall component of the RCFT (Tables 7 and 14). These results are consistent with Hypothesis No 1.

Group BN demonstrated significantly poorer test performance relative to Group C on three from six tests with seven per cent of the group scoring at a defective level on both Logical Memory I and the RCFT - Delayed Recall (Tables 7 and 14). The unexpected finding that Group BN performed significantly poorer than the control group in the Learning and Memory Domain provides evidence that bulimia nervosa may be associated with neuropsychological difficulties in a domain other than attention. However, modest inverse correlations on both of the Logical Memory Tests provides some support for the hypothesis that test scores in this population are influenced both by affective variables and attention variables as predicted by Hypotheses Numbers 2 and 3.
Test Results - Executive Functioning Domain (Table 15):

The control group performed better than both eating disorder groups on two from three test results in this domain (Table 15). However between group significance failed to be established on any test result. Three per cent of Group AN scored at an impaired level on Trail Making Test B.

Table 15. ANOVA: Cognitive Tests in the Executive Domain.

<table>
<thead>
<tr>
<th>Test</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
<th>F(df)</th>
<th>p value</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal Fluency</td>
<td>46</td>
<td>42.4</td>
<td>45.433</td>
<td>F(2,87) 1.18</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>sd 9.653</td>
<td>sd 7.407</td>
<td>sd 11.702</td>
<td></td>
<td>p = 0.311</td>
<td></td>
</tr>
<tr>
<td>Trail Making Test B</td>
<td>56.9</td>
<td>51.233</td>
<td>47.233</td>
<td>F(2,87) 2.46</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>sd 19.794</td>
<td>sd 18.878</td>
<td>sd 10.724</td>
<td></td>
<td>p = 0.914</td>
<td></td>
</tr>
<tr>
<td>Mazes (WISC III)</td>
<td>22.867</td>
<td>24.5</td>
<td>24.6</td>
<td>F(2,87) 2.69</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.863</td>
<td>sd 3.908</td>
<td>sd 2.872</td>
<td></td>
<td>p = 0.073</td>
<td></td>
</tr>
</tbody>
</table>

Correlational analyses within Group AN found only two significant relationships (Tables 9 and 10). Verbal Fluency was inversely associated with the HADS Depression subscale score (Pearson r = -0.57; Table 9) and the Mazes test was inversely associated with the duration of eating disorder (r = -0.51; Table 10).
In Group BN, inverse correlations were also established between Verbal Fluency and the HADS Depression subscale (Pearson $r = -0.53$). Verbal Fluency was also inversely related to the DSSI/NS Rumination Severity subscale (Pearson $r = -0.52$; Table 11). The latter was also correlated with a longer response time on the Trail Making Test B (Pearson $r = 0.49$). A similar finding was revealed by a correlation between Trail Making Test B and Body Mass Index (Pearson $r = 0.53$; Table 12).

In analysing relationships between test scores and other variables in the control group, a modest significant correlation was evident between Trail Making Test B and HADS Anxiety Subscale (Pearson $r = 0.47$; Table 13).

**Summary of results in the Executive Functioning Domain.**

On the three sets of test results processed in this domain, there was no evidence of significant between-group differences. Hypothesis Number 1 is therefore not supported.

The weak to modest significant correlations found on analysis of Group BN scores, suggest that test performance was negatively influenced by affective variables due to attentional processes. This result is consistent with Hypothesis Number 3.
Test Results - Visuospatial / Constructional (Table 16).

In the domain of Visuospatial / Constructional Functioning, Group C demonstrated the highest scores throughout, although between-group significance was established on one test result between group C and Group AN: Object Assembly ($F_{2,87} = 4.56, p<0.5$).

<table>
<thead>
<tr>
<th>Test</th>
<th>Group AN (Anorexia)</th>
<th>Group BN (Bulimia)</th>
<th>Group C (Control)</th>
<th>F(df)</th>
<th>p value</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Object Assembly</td>
<td>30.167 sd 7.705</td>
<td>29.9 sd 6.022</td>
<td>33.933 sd 2.033</td>
<td>$F(2.87)$</td>
<td>$p = 0.017^*$</td>
<td>AN &lt; C</td>
</tr>
<tr>
<td>Block Design</td>
<td>32.9 sd 9.053</td>
<td>31.867 sd 8.792</td>
<td>37.667 sd 4.536</td>
<td>$F(2.87)$</td>
<td>$p = 0.15^*$</td>
<td>n.s.</td>
</tr>
<tr>
<td>Picture Completion</td>
<td>15.433 sd 2.897</td>
<td>16.133 sd 2.097</td>
<td>16.833 sd 1.289</td>
<td>$F(2.87)$</td>
<td>$p = 0.052$</td>
<td>n.s.</td>
</tr>
<tr>
<td>Rey Complex Figure Test Copy</td>
<td>33.4 sd 3.973</td>
<td>34.433 sd 1.870</td>
<td>34.933 sd 2.149</td>
<td>$F(2.87)$</td>
<td>$p = 0.106$</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

In comparing the levels of cognitive deficits (Table 7: page 89), 13 per cent of both Group AN and Group BN scored at an impaired level on the Copy score of the RCFT. However, seven per cent of Group C also scored at an impaired level on this test.
Correlational analyses for Group AN found a modest association between the HADS Depression subscale score and the Rey Complex Figure Test - Copy (Pearson r = -0.54; Table 9: page 95).

Correlational analyses for Group BN found State Anxiety to be inversely associated with Picture Completion (Pearson r = -0.50) and Block Design (Pearson r = -0.52; Table 11: page 97). Of the clinical variables (Table 12: page 98), Laxative use was inversely associated with Object Assembly (Pearson r = -0.47).

Correlational analysis of control group scores revealed no significant relationships (Table 13: page 99).

**Summary of results in the Visuospatial / Constructional Domain.**

One from four results demonstrated between-group significance in this domain (WAIS-R : Group C v Group AN). This is consistent with Hypothesis No 1.

**Post hoc analyses: Trail Making Test Errors and WAIS-R Block Design Failures (Table 17).**

On post hoc scoring of the total number of tracking errors made on both the Reitan Trail Making Tests (A and B), Group C made fewer errors than both of the the clinical groups (Table 17: overleaf). However, between-group significance was established between Group C and Group AN only with Chi-Sq 2 : 6.857, p < 0.05. Within-group correlational procedures failed to reveal any significant relationships on this measure in either Group C or Group AN. In Group BN however, one modest
inverse relationship was demonstrated between tracking errors and BMI (Pearson r = -0.69; Table 12: page 98).

On analysing the number of Block Design failures, Group AN scored a significantly higher rate of failure than Group C with between-group significance at Chi-Sq 2 : 7.71, p < 0.05. Block Design failures in this group tended to occur on the more difficult designs patterns (5,7,8 & 9; Lezak, 1995). However, there was no evidence of impairment on Block Design Scores (Table 7).

<table>
<thead>
<tr>
<th>Table 17.</th>
<th>ANOVA: Trail Making Tests Errors and Block Design Failures.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test</td>
<td>Group AN (Anorexia)</td>
</tr>
<tr>
<td>-----------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Trail Making Tests A &amp; B: Errors</td>
<td>0.466</td>
</tr>
<tr>
<td>Block Design Failures</td>
<td>1.2</td>
</tr>
</tbody>
</table>

Correlational analyses for Group AN found modest relationships between Block Design Failure and State Anxiety (Pearson r = 0.55; Table 9) and the BITE Symptom Score (Pearson r = 0.53; Table 10).

Correlational analyses for Group BN found one modest relationship between Block Design Failures and State Anxiety (Pearson r = 0.56; Table 11).

No statistical correlations at p <0.01 were evident for within-group analysis of Group C.
On post hoc analyses of these two measures there was evidence of attentional difficulties in Group AN which is consistent with Hypothesis Number 1. There was also evidence of visuo-spatial / constructional difficulties in Group AN which is also consistent with Hypothesis Number 1.

The influence of anti-depressant medication.

To analyse the possible effects of antidepressant use on cognitive test scores, the participants who reported using antidepressants were compared to those not using antidepressant medication. Thirty per cent of the Group AN and 23 per cent of Group BN were using anti-depressant medication at the time of testing. A non-parametric test for independent samples (Kruskal-Wallis) was performed on the test variables due to unequal sample sizes. Of 22 test results, between group significance set at $p<0.01$ was obtained on only one measure (Verbal Fluency - Executive Functioning Domain). Subsequent Mann-Whitney analysis sustained significance at this level between the two groups with a diagnosis of bulimia nervosa and in favour of the non-antidepressant subgroup.

On analysing between-group differences for affective variables, no significant difference was found on measures of State Anxiety and clinical anxiety (HADS). However, a significant between-group difference was found on the HADS Depression subscale ($U = 23, p <0.01$) between the groups diagnosed with anorexia nervosa, with the anti-depressant subgroup having higher scores for depression. Although the physical side effects of antidepressant medication may be a confounding factor, it would appear to be the case that the major contributing factor
to a trend for lower scores in the antidepressant subgroups is the presence of depression.

The influence of state anxiety on test scores (Table 18).

The control group scored significantly lower than both clinical groups on the mid battery test of state anxiety (STAIS-S Questionnaire). Between-group difference was established at $F(2,87): 24.18, p<0.01$. The highest state anxiety scores were recorded by the Group BN although there was no significant difference found between the clinical groups (Table 18: below). The mean STAIS-S score for Group C of 32.53 is equivalent to the mean scores published for American females (Spielberger, 1983); which range from working adults norms (mean 35.20) to Student norms (mean 38.76). The high state anxiety mean score found in Group AN was not reflected however by significant relationships with cognitive test scores.

In Group BN, state anxiety was also evident in the Visuo-Spatial / Constructional Domain with Block Design and Picture Completion inversely correlated (Pearson $r = -0.52$ and Pearson $r = -0.50$ respectively; Table 11). State anxiety was also evident on Block Design Failures (Pearson $r = 0.56$; Table 11). These significant correlations for Group BN are consistent with Hypotheses Number 3.

| Table 18. ANOVA: State anxiety (STAIS-S Questionnaire). |
|---------------------------------|----------------|----------------|----------------|----------------|----------------|
|                                | Group AN (Anorexia) | Group BN (Bulimia) | Group C (Control) | F(df) | Post-Hoc |
| STAIS-S mean score            | 44.633 (9.57)     | 47.167 (8.60)     | 32.533 (7.37)     | 24.18 (2,87) | AN, BN > C |
| **p < 0.01**                  | **p < 0.01**      | **p < 0.01**      | **p = 0.000**     | **p = 0.000** | **p = 0.000** |
Summary of the effects of partialling-out procedures.

It was predicted by Hypothesis Number 3 that Group BN test results would be influenced by affective variables. The results of within-group correlational analyses generally supported this hypothesis. There was also evidence for the influence of affective variables in both clinical groups and across neuropsychological domains. Therefore state anxiety (STAIS) and both the HADS Subscales (Anxiety and Depression) were entered as covariates in a multi-analysis of covariance (MANCOVA).

On considering the analysis of covariance generally, it is accepted that the number of covariates entered should be kept to a minimum in order to avoid unnecessary reduction of the test power. There is also no need to covary variables which are strongly related, ie with a correlation coefficient of 0.8 or more (Bryman and Cramer, 1997). Correlational analysis had found that State Anxiety was related to the HADS Anxiety and Depression subscales (Pearson r = 0.62, Pearson r = 0.50 respectively). Similarly, the HADS Anxiety Subscale was found to be highly correlated with the HADS Depression Subscale (Pearson r = 0.8). However, correlational analysis within the anorexic group established weak correlations with the HADS Anxiety Subscales (Pearson r = 0.35) and HADS Depression subscale (Pearson r = 0.35). Further, the relationship between the HADS subscales was modestly correlated at Pearson r = 0.45. In Group BN, the HADS Subscales were however also modestly correlated at Pearson r = 0.60 and State Anxiety was found to be weakly correlated with the HADS Anxiety and Depression Subscales at Pearson r = 0.26 and Pearson r = 0.11, respectively (Tables 11 and 12).
Given that these correlations are generally weak, it was deemed reasonable to enter State Anxiety and both the HADS subscales as covariates. However, the DSSI/NS Subscale scores were rejected as potential covariants due to apparent minimal impact on the test results as suggested by correlational analyses. Whilst analysis of the DSSI/NS scores revealed that between 7 per cent and 40 per cent of the participants in both clinical groups had scores which reached 'caseness', analysis of group means failed to reflect this pathology. Where correlational analysis revealed significant correlations on subscales and test results, for example the Rumination subscales, subscales were generally found to correlate highly with the HADS Anxiety Subscale.

The results of partialling out affective variables was significant between groups with Pillais $F_{30, 219} = 2.285, p < 0.01)$. Univariate analyses revealed significant between group differences on five from eight test results in Group AN and four from seven results in Group BN. The results will be discussed with reference to each neuropsychological domain where ANCOVA was carried out (Tables 19 to 21).
Results of ANCOVA: Attention Domain (Table 19).

Table 19 below illustrates that a partiaiing-out procedure removed significant between-group differences on two from four test measures: the PASAT Mean Score (Group AN, Group BN v Group C) and the Modified Stroop Test (Group BN v Group C). Significant between-group differences remained on the following two test scores:

1. Attention-Concentration Index : $F(3, 80): 3.12, p<0.05$ (Group BN v Group C)
2. Digit Symbol : $F(3, 80): 2.94, p<0.05$ (Group AN, Group BN v Group C).

<table>
<thead>
<tr>
<th>Test</th>
<th>ANOVA</th>
<th>ANCOVA</th>
<th>Covariate</th>
<th>Covariate</th>
<th>Covariate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F(df)</td>
<td>F(df)</td>
<td>STAIS-S</td>
<td>HADS</td>
<td>HADS</td>
</tr>
<tr>
<td></td>
<td>p value</td>
<td>p value</td>
<td>State Anx.</td>
<td>Anxiety</td>
<td>Dep.</td>
</tr>
<tr>
<td>Atten-Conc. Ind. (WMS-R)</td>
<td>$F(2.87) 4.46$</td>
<td>$F(3.80) 3.12$</td>
<td>$t 2.219$</td>
<td>$t 0.41$</td>
<td>$t 1.07$</td>
</tr>
<tr>
<td></td>
<td>$p = 0.01^{**}$</td>
<td>$p = 0.03^{*}$</td>
<td>$p = 0.03^{*}$</td>
<td>$p = 0.68$</td>
<td>$p = 0.29$</td>
</tr>
<tr>
<td></td>
<td>BN &lt; C</td>
<td>BN &lt; C</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
</tr>
<tr>
<td>PASAT Mean Score</td>
<td>$F(2.86) 11.57$</td>
<td>$F(3.80) 1.132$</td>
<td>$t 0.55$</td>
<td>$t 0.89$</td>
<td>$t 0.68$</td>
</tr>
<tr>
<td></td>
<td>$p = 0.00^{**}$</td>
<td>$p = 0.34$</td>
<td>$p = 0.58$</td>
<td>$p = 0.38$</td>
<td>$p = 0.49$</td>
</tr>
<tr>
<td></td>
<td>AN, BN &lt; C</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroop (mod)</td>
<td>$F(2.87) 4.00$</td>
<td>$F(3.80) 0.719$</td>
<td>$t 0.01$</td>
<td>$t 1.21$</td>
<td>$t 0.03$</td>
</tr>
<tr>
<td></td>
<td>$p = 0.02^{*}$</td>
<td>$p = 0.54$</td>
<td>$p = 0.99$</td>
<td>$p = 0.23$</td>
<td>$p = 0.98$</td>
</tr>
<tr>
<td></td>
<td>BN &lt; C</td>
<td>n.s.</td>
<td>n.s.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digit Symbol (WAIS-R)</td>
<td>$F(2.87) 4.62$</td>
<td>$F(3.80) 2.943$</td>
<td>$t 0.01$</td>
<td>$t 1.31$</td>
<td>$t 1.24$</td>
</tr>
<tr>
<td></td>
<td>$p = 0.02^{*}$</td>
<td>$p = 0.38^{*}$</td>
<td>$p = 0.37$</td>
<td>$p = 0.19$</td>
<td>$p = 0.22$</td>
</tr>
<tr>
<td></td>
<td>AN, BN &lt; C</td>
<td>AN, BN &lt; C</td>
<td>AN, BN &lt; C</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p<0.05  **p<0.01
Results of ANCOVA: Learning and Memory Domain (Table 20).

Table 20 below illustrates that a partialling-out procedure removed significant between-group differences on one from five measures. In Group AN one from five significant results was removed (Verbal Pairs Associates I: Group AN v Group C) and in Group BN, one from three significant results was removed (RCFT-Delayed: (Group BN v Group C).

<table>
<thead>
<tr>
<th>Test</th>
<th>ANCOVA: Cognitive Tests in the Learning and Memory Domain.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ANOVA</td>
</tr>
<tr>
<td></td>
<td>F(df)</td>
</tr>
<tr>
<td>Logical Memory I</td>
<td>F(2.87) 6.61</td>
</tr>
<tr>
<td></td>
<td>AN, BN &lt; C</td>
</tr>
<tr>
<td>Logical Memory II</td>
<td>F(2.87) 11.37</td>
</tr>
<tr>
<td></td>
<td>AN, BN &lt; C</td>
</tr>
<tr>
<td>Verbal Pairs</td>
<td>F(2.87) 6.96</td>
</tr>
<tr>
<td>Associate I</td>
<td>AN &lt; BN, C</td>
</tr>
<tr>
<td>Verbal Pairs</td>
<td>F(2.87) 5.08</td>
</tr>
<tr>
<td>Associate II</td>
<td>AN &lt; C</td>
</tr>
<tr>
<td>Rey Complex</td>
<td>F(2.87) 9.21</td>
</tr>
<tr>
<td>Fig. Test Delayed</td>
<td>AN, BN &lt; C</td>
</tr>
</tbody>
</table>

*p<0.05  **p<0.01
However, between-group significance was sustained on the following tests:

1. Logical Memory I: $F_{3,80}: 5.25, p < 0.01$ (Group AN, Group BN v Group C).
2. Logical Memory II: $F_{3,80}: 7.89, p < 0.01$ (Group AN, Group BN v Group C).
3. Verbal Pairs Associates II: $F_{3,80}: 3.78, p < 0.05$ (Group AN v Group C).
4. Rey Complex Figure Test-Delayed: $F_{3,80}: 2.91, p < 0.05$ (Group AN v Group C).

**Results of ANCOVA: Visuo-spatial / Constructional Domain (Table 21).**

Table 21 below illustrates that following a partialling-out procedure for affective covariables, the significant mean score difference between Group AN and Group C was sustained on Object Assembly ($F_{3,80}: 2.789, p < 0.05$).

<table>
<thead>
<tr>
<th>Test</th>
<th>ANOVA</th>
<th>ANCOVA</th>
<th>Covariate STAIS-S</th>
<th>Covariate HADS Anxiety</th>
<th>Covariate HADS Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Object Assembly (WAIS-R)</td>
<td>$F(2,87) 4.56$ $p = 0.01^*$</td>
<td>$F(3,80) 2.789$ $p = 0.46^*$</td>
<td>$t 1.75$ $p = 0.08$</td>
<td>$t 1.55$ $p = 0.12$</td>
<td>$t 1.64$ $p = 0.09$</td>
</tr>
</tbody>
</table>

$^*p < 0.05$
ANCOVA: Summary.

Thus following partialling-out procedures, two from eight significant group differences related to Group AN were removed with significance remaining on one attentional measure (WAIS-R Digit Symbol); three related tests of auditory-verbal learning (WMS-R Logical Memory Tests I and II and Verbal Pairs Associates Test II); a test of visual memory for a 2D complex figure (Rey Complex Figure Test - Delayed) and a test of visuo-spatial / constructional functioning (WAIS-R Object Assembly).

In Group BN partialling-out procedures resulted in three from seven significant between-group differences being removed. Significance was sustained between Group BN and Group C on two attentional measures (WMS-R Attention-Concentration Index and the WAIS-R Digit Symbol) and two related tests of auditory-verbal memory (WMS-R Logical Memory Tests I and II).

Results of Hypotheses Testing.

Hypothesis 1.

In comparison to a matched control group, participants with anorexia nervosa (Group AN) will be found to have demonstrated significantly poorer results on cognitive tests over all the neuropsychological domains tested.
Result: The participants with anorexia nervosa (Group AN) demonstrated significantly poorer performance than Group C (Control) in three from four neuropsychological domains - Attention, Learning and Memory and Visuo-spatial / Constructional. The results of partialling out affective variables sustained between-group significance on one of the two significant results in the Attention Domain, four of the five significant results in the Learning and Memory Domain and one significant result in the Visuo-spatial / Constructional Domain. However, no significant between-group differences were established on any of the three test measures in the Executive Functioning Domain and Hypothesis Number 1 is therefore rejected.

Hypothesis 2.

In comparison to a matched control group, a group of participants with bulimia nervosa will be found to have demonstrated significantly poorer results on cognitive tests in the neuropsychological domain of attention only.

Result: The participants with bulimia nervosa (Group BN) revealed significantly poorer performances compared to Group C (Controls) on four from five measures in the Attention Domain and three from six measures on the Learning and Memory Domain. Following partialling-out procedures for affective variables, significant group differences remained for two of the four significant results in the Attention Domain and two of the three significant results in the Learning and Memory Domain. Hypothesis Number 2 is therefore rejected.
Hypothesis 3.

Where significantly poor test results are established for participants with bulimia nervosa, these results will be found to have been significantly influenced by affective variables.

Result: Using anxiety and depression as covariables in a partialling-out procedure, significant group differences between Group BN (Bulimia) and Group C (Control) were removed on two from four significant results in the Attention Domain and one from three significant results in the Learning and memory Domain.

On the two remaining significant between-group differences in the Learning and Memory Domain (Logical Memory Tests I and II); within-group correlational analysis revealed significant albeit modest and inverse relationships with scores on the HADS Anxiety Subscale and DSSI/NS Rumination Subscale. Therefore, Hypothesis Number 3 is accepted.
3e. DISCUSSION.

This study tested three hypotheses related to cognitive functioning in eating disorders. Although the participants with anorexia nervosa demonstrated significantly poorer test performance than the control group on three of the four domains tested, the hypothesis of overall poorer performance across neuropsychological domains was rejected. However, rejection of this hypothesis should not be viewed as evidence for a failure to establish neuropsychological dysfunction in anorexia per se. On the contrary, the evidence from this study supports previous studies in finding significant between group-differences when participants with anorexia are compared to normal controls across neuropsychological domains (Hamsher, Halmi & Benton, 1981; Jones, Duncan, Brouwers & Mirsky, 1991; Kingston, Szmuckler, Andrews, Tress & Desmond, 1996; Mathias & Kent, 1998).

The finding of attentional difficulties in bulimia nervosa is consistent with the few studies which have investigated attentional impairment in bulimia (Beatty, Wanderlich, Statton & Ternes, 1990; Jones et al, 1991; Bowers, 1994; Lauer, Gorzewski, Gerlinghoff, Backmund & Zihl, 1999). The finding of an auditory-verbal memory difficulty in the present study was not predicted having previously been reported by only one study (Beatty, Wanderlich, Statton & Ternes, 1990). The finding of a non-significant trend for difficulties in the domain of visuo-spatial / constructional functioning was also not predicted although McKay, Humphries, Allen & Clawson (1986) reported poor performance on measures of right hemispheric
functioning. However, the finding in the present study that cognitive functioning in bulimia nervosa is strongly influenced by affective variables questions the hypothesis that cognitive deficits in this population are a reflection of neuropsychological dysfunction. Indeed, both correlational analyses and partialling-out procedures established that affective variables were influential in several test domains and for both clinical groups.

Explaining the results:

Overall, both clinical groups performed more poorly than matched controls with between-group differences reaching significance level on 44.4 per cent of the test measures. Both clinical groups also demonstrated more cognitive deficits (at two standard deviations below published norms) and significantly more (at between one and two standard deviations) than the control participants. Group AN (Anorexia) scored significantly poorer than the control group on 44.4 per cent of the test results with Group BN (Bulimia) scoring significantly poorer than the control group on 39 per cent of these measures. After partialling-out the effects of anxiety and depression, 25 per cent of the significant results were removed suggesting that affective variables may account for a considerable amount of the variance found between groups on cognitive test results.

On considering the results of Group AN's test performance in the Attention Domain, the mean scores were significantly worse than controls on two of the five tests of attention (PASAT Mean Score and WAIS-R Digit Symbol). Both of these tests are sensitive to attentional deficits. However, it is arguable that Group
AN’s poorer performance on the PASAT could be accounted for by fatigue and/or other variables. The PASAT requires speed in cognitive processing along with accuracy in response and sustained concentration (Deary, Ebmeir, MacLeod, Dougall, Hepburn, Freir & Goddwinl, 1994). The PASAT was the second last test to be administered and after a hour of testing, the participants in Group AN may have been subject to fatigue, particularly in light of low Body Mass Index. Ward (1997) has also noted an age effect on the PASAT with younger participants paradoxically, performing more poorly compared to older age groups. However, in the present study the participants were matched for age and this variable is therefore rejected as an explantory factor.

Digit Symbol also requires speed and accuracy in processing and both attentional tests may have been subject to performance anxiety. In contrast, the WMS-R Attention-Concentration Index does not require speed and on this test, Group AN failed to demonstrate a significantly poorer result. It is reasonable therefore to suggest that the participants with anorexia had difficulty with both the PASAT and Digit Symbol due to either affective or clinical variables. This suggestion is given some weight by the fact that the partialling-out procedure for affective variables removed significance on the PASAT Mean Score. However, partialling-out failed to removed significance on Digit Symbol and within-group correlational analysis failed to yield strong correlations between test performance in this domain and affective variables. One possible explanation is that the Digit Symbol scores were influenced negatively by the physical side-effects of antidepressant medication. One third of Group AN (Anorexia) were taking antidepressant medication and a trend was evident for better test scores from those participants who were not taking antidepressants.
Digit Symbol requires motor ability, visuo-spatial skills and incidental learning memory. It is therefore not purely a test of attention and may have been influenced by other factors. However, correlational analyses however, failed to suggest any strong associations with clinical variables.

A neurobiological explanation would favour the possibility of mild global brain damage. Digit Symbol is regarded as a sensitive measure of neuropsychological dysfunction and one of the few cognitive test found to correlate with radiological brain abnormality in anorexia (Palazidou, Robinson and Lishman, 1990). However, on scrutinising the levels of cognitive deficits found in this group, there was no evidence of impaired test performance on Digit Symbol. The minimal brain damage hypothesis is therefore not well supported.

In the Learning and Memory Domain, significant between-group differences prior to partialling-out procedures were established between the Group AN and the control group on five out of six test score results (Logical Memory Test I and II, Verbal Pairs Associates Learning Test I and II; and the Rey Complex Figure Test - Delayed Recall).

Group AN's poorer performance on the visual memory component of the RCFT may have been due to the incidental learning nature of this free recall task. The copy task of the Rey Complex Figure Test tests visuo-spatial and graphic abilities and no instruction is given to learn the 2 D complex figure. Subsequent free recall therefore requires good incidental learning, which one study has reported as being defective in anorexia (Strupp, Weingarter, Kaye & Gwitsman, 1986). Strupp et al (1986) also found that when visual attention was a requirement of the task; satisfactory learning was evident in participants with anorexia. In the present study, Group AN
demonstrated satisfactory learning on the Extended Picture Recognition subtest (Rivermead Behavioural Memory Test). This result suggests that visual memory is not compromised in anorexia where a recall task is made explicit.

On the auditory-verbal memory subtests (WMS-R), Group AN performed significantly poorer than controls on Logical Memory I and II and Verbal Pairs Associates Tests I and II. Whilst the test format of Logical Memory suggests a requirement for immediate recall, there is no explicit requirement for delayed recall. Indeed most participants in this study seemed surprised and a few perplexed when given the free recall task. It may therefore be argued that Group AN's poorer performance in this domain was attributable to a verbal component of incidental learning similar to that described above for visual memory recall. It may also be the case that the test results may have been influenced by a degree of cognitive rigidity or narrow focusing of attention in this population.

Cognitive rigidity has been suggested as a characteristic of anorexia nervosa and therefore a possible confounding variable on cognitive testing. Cognitive rigidity may also be associated with the personality trait of perfectionism and features of obsessive-compulsive disorder, both of which are associated with anorexia nervosa (Small, Camp, Bushnell & Bargman, 1984; Hsu, Kaye & Weltzin, 1993). The evidence suggests that attention capacity may be compromised or distracted due to these factors. For example, in comparing a group of normal healthy adults rated as 'dysfunctional perfectionists' with a group rated as 'functioning' perfectionists, Rheaume, Freeston, Ladouceur, Bouchard, Gallant, Talbolt and Vallieres (2000) found that the 'dysfunctional perfectionist' group demonstrated significantly longer times to complete a precision task. This group also demonstrated more intrusive
thoughts related to performance as opposed to problem solving. The 'dysfunctional perfectionist' group also scored higher on a measure of obsessive-compulsive indices. Some evidence for the influence of such personality traits in the present study may be gleamed from Group AN's poorer test performance on a visuo/constructional task (WAIS-R Block Design). Although between-group significance on this test was not established, post-hoc analysis of the number of design failures did reach significance. Further, the participants with anorexia tended to fail on the more difficult design patterns - a failure which is associated with cognitive rigidity (Lezak, 1995). However, the fact that Group AN performed satisfactorily on a measure of verbal fluency which is also regarded as a measure of cognitive flexibility is not consistent with this hypothesis. It does not however, exclude perfectionism as a possible explanatory factor in Group AN test poorer results.

A measure of perfectionism was available from the Eating Disorders Inventory (2) Perfectionism Subscale and Table 5 illustrates that Group AN scored significantly higher on this subscale relative to the control group. However, within-group correlational analysis failed to yield any significant correlations between the EDI 2 Perfectionism Subscale and cognitive test scores.

It is possible that ruminative anxiety was a further confounding variable on the auditory-verbal learning tasks with 27 per cent of Group AN scoring for 'caseness' on the DSSI/NS Rumination Symptoms subscale (Table 4). Thirty seven per cent also scored for 'caseness' on the severity subscale. Whilst ruminative anxiety was found to be weakly correlated with Logical Memory II, no further significant correlations were established for ruminative anxiety. Similarly, whilst Group AN scored highly on the EDI2 subscale, Interceptive Awareness this was not correlated significantly with cognitive test measures. A further possible explanation for Group AN's poor
performance on Logical Memory may be the fact that Logical Memory was the second test administered and therefore particularly vulnerable to test anxiety. This would explain both the poor immediate recall task and delayed recall due to weak encoding of the stimulus material. Although the STAIS-S was used as a measure of test anxiety, its presentation in the latter half of the testing session may have insufficiently measured early test anxiety. Qualitative observations did suggest early anxiety responses in many of the participants including the control group, although this was not measured objectively.

The influence of affective variables on the anorexic group's test performance in the Learning and Memory Domain was not compelling with only one modest significant correlation revealed between Logical Memory II and the HADS Anxiety subscale. Further, the effects of partialling-out affective variables was limited with group significance being removed on only one significant test result in the Learning and Memory Domain (Verbal Pairs Associates I). That significant between-group differences remained on both the incidental visual memory test (Rey Complex Figure Test - Delayed Recall), Logical Memory Tests I and II and Verbal Pairs Associates II, suggests that participants with anorexia had working memory difficulties not accountable by emotional variables. To explore this further the percentage of material retained at Logical Memory II was subjected to post-hoc analysis. The results found that whilst Group C (Control) retained 92.5 per cent of the text on delayed recall, Group AN (Anorexia) retained 82.8 per cent with between-group difference found to be significant. Given that the participants with anorexia also performed significantly more poorly than controls on Logical Memory I, this would
suggest that anorexia is associated with both auditory-verbal encoding and retrieval difficulties. Such difficulties are not readily explainable by affective or other variables and may in the final analysis may be a product of mild brain damage. If this is indeed the case, then there appears to be little prospect of localising any such neuropsychological deficit given that episodic memory retrieval has been found to involve a network of brain regions on functional imaging studies (Buckner & Barch, 1999).

There is one further explanatory factor in Group AN's performance on auditory-verbal learning tests. As noted above, Logical Memory II was was modestly and inversely correlated with HADS Anxiety scores and DSSI/NS Rumination Severity score. Although partialling-out HADS Anxiety scores failed to remove between-group significance, nonetheless there may be a selective processing bias evident in both Logical Memory and Verbal Pairs Associates which involves a threat component. Both of the prose stimuli in the Logical Memory tests have themes of robbery and pathos. The logic behind such themes is of course that the stimuli should be sufficiently interesting to facilitate encoding and thus retrieval on delayed recall. For example:

'She had been held up...and robbed of fifty six dollars'... 'His truck skidded off the road into a ditch...He was thrown against the dashboard and was badly shaken...and he doubted that help would come'.

The first of the Logical Memory prose stimuli also includes a food variable:
'She had four small children...and they had not eaten for two days.'
It is conceivable that such stimuli may have posed some threat to participants in the clinical groups. Some evidence for this hypothesis comes from the results of King, Polivy and Herman's (1991) study, which found that both participants with anorexia and restrictive dieters recalled weight and food items disproportionately to other items when presented with prose material involving shape and dietary concerns. A similar effect may have arisen in the present study and may have negatively influenced Group AN's test performance. Similarly, Group AN's poorer performance on Verbal Pairs Associates I and II may have been influenced by word pairs with a possible threat valence. For example - 'School - Grocery'; 'Fruit - Apple' and 'Cabbage - Pen'.

Such a hypothesis could be tested by analysis of verbatim responses in both Logical Memory and Verbal Pairs Associates. It would also be relatively easy to adapt these tests to control for both affective and anorexic valence.

In the domain of executive functioning, Group AN demonstrated slower completion time scores than both Group C and Group BN. Whilst between-group difference was not significant, the trend is consistent with several studies which have found poorer test performance by participants with anorexia on a computerised maze task (Jones, Duncan, Brouwers & Mirsky, 1991; Kingston, Szmuckler, Andrews, Tress & Desmond, 1996). In the present study, the Mazes task required planning ability and speed, both of which were computed to yield a final score. Violation of the rules are made explicit, for example; that the maze lines should not be crossed. In general, qualitative observation suggested a trend for participants in Group AN to plan ahead and execute a strategy in completing the task. Whilst this led to fewer errors and penalties, this appeared to be at the expense of longer completion times. It
may be the case that in anorexia, whilst executive functioning is not clearly compromised, executive test performance may be subject to personality variables such perfectionism, a tendency to over-planning or obsessional slowness. To date such hypotheses have not been investigated.

As discussed above, in the Visuo-spatial / Constructional Domain the mean score for Block Design in Group AN was considerably, yet not significantly lower than the control group. On examining the frequency of Block Design failure however, a significant group difference was established with a noted failure on those designs comprised largely of diagonal features. Such failure is associated with right hemispheric lesions and concrete mindedness (Lezak, 1995). Gillberg, Gillberg, Rastam and Johansson (1996) also found similar processing errors in Block Design in a large sample of children with anorexia nervosa. Unfortunately the authors did not report which designs were problematic for the sample. In the present study, participants with anorexia also scored significantly lower than controls on the WAIS-R Object Assembly Subtest. Object Assembly is a test of perceptual organisation which correlates strongly with Block Design (Lezak, 1995). Within-group correlation revealed that the two test were indeed highly correlated. Partialling-out the influence of affective variables on Object Assembly failed to remove between-group significance and this provides further evidence of perceptual organisational difficulties in anorexia nervosa.

On considering the influence of affective variables on test performance in the participants with anorexia, there was evidence that anxiety based factors were
influential on test scores. However, partialling-out the effects of such variables did not provide compelling evidence that affective variables may account for poorer test performance in this population.

The influence of clinical variables was also not compelling with surprisingly few significant correlations evident between test performance and eating disorder pathology. In particular, Body Mass Index was not found to correlate with any of the cognitive test measures and the duration of eating disorder was only modestly correlated with Trail Making Test A.

The duration of eating disorder was found to correlate with Verbal Pairs Associates I and this is consistent with the significant between-group difference found on univariate analyses. However, that significance was removed when partialling-out anxiety suggests that the duration of the eating disorder appears to have a minimal impact on test performance.

On considering the results of Group BN (Bulimia), it is clear that attentional difficulties were highly influential on test performance. Compared to the control group, participants with bulimia nervosa performed more poorly on four from five test measures of attention and which cluster around processing speed (Modified Stroop, Digit Symbol) sustained attention (PASAT) and digit and visual memory span (Attention-Concentration Index). Whilst the possible influence of affective variables was not clearly demonstrated by correlational analyses, partialling-out procedure removed between-group significance on two of the four significant results. The removal of the significance on the PASAT and the Modified Stroop suggests that speed of cognitive processing was strongly influenced by affective variables. However that partialling-out procedures failed to remove significance between
groups on Digit Symbol suggests a pure attentional deficit. That Group BN also continued to demonstrate between-group significance following partialling-out procedures on the Attention-Concentration Index, where speed is not a test requirement suggests that attentional difficulty is the most likely explanation.

Whilst the Group BN had a shorter completion time compared to the other groups on the visuo-motor tracking test (Reitan Trail Making Test A) this was at the expense of a higher error rate than that of the control group. This may suggest impulsive behaviour rather than attentional deficits, a characteristic associated with bulimia nervosa and manifested as 'rule violation' in Beatty, Wanderlich, Statton & Ternes's (1990) study. However, whilst Group BN scored significantly higher than the control group on the Impulse Regulation Scale, within-group correlational analysis failed to reveal significant relationships between Impulse Regulation and test scores.

Prior to partialling-out procedures, Group BN demonstrated significantly poorer performance compared to the control group on three of six measures in the Learning and Memory Domain (Logical Memory Tests I and II and the Rey Complex Figure Test -Delayed Recall). Within-group correlational analyses revealed that the Logical Memory Tests I and II were inversely correlated with the HADS Anxiety subscale and inversely associated with DSSI/NS Rumination severity subscale. This would suggest that anxiety played a considerable role in Group BN's performance on Logical Memory. However no significant association was established with either state anxiety and HADS Depression scores and partialling-out for affective variables failed to remove between-group significance. The test results may have also been influenced by personality characteristics such as perfectionism and obsessive-compulsive traits. However, whilst between-group significance was not established for the eating disorder groups on the EDI 2 Perfectionism subscale, the bulimic group
demonstrated lower scores and Perfectionism was not evident as an influential variable on correlational analysis.

The bulimic group's score on the Maze Test was also lower than the control group although again, not at a significant level. However poor impulse control may be a reasonable explanation for poorer test performance on executive tasks. Group BN scored significantly higher than controls on the EDI-2 Impulse Regulation Subscale and qualitative observations on Mazes test performance suggested impulsivity with shorter completion times at the expense of accuracy and therefore a higher error rate. However, correlational analysis between Maze test score and EDI-2 Impulse Regulation failed to yield a significant relationship.

Impulsivity may also explain Group BN's faster time on the Trail Making Test A when compared to the control group although between-group means were not significant and there were no relationships established on correlational analysis.

One further possible explanatory factor in Group BN's test performance is the varying BMI in this clinical group. Bulimia nervosa is associated with recurrent cycles of restrictive dieting, binge eating, purging. On correlational analyses, a significant, albeit modest correlation was established between BMI and a longer time on Trail Making Test B. This may suggest that those bulimics with higher BMI's (and therefore overweight) may be associated inversely with poorer test performance. However, Kretsch, Green, Fong, Elliman and Johnson's (1997) study of dieting in obesity failed to reveal problems with sustained attention. In the present study, no
measure was taken of BMI variations although this may be a useful variable to control for in a prospective study.

Whilst Group BN did not demonstrate significant between-group differences relative to the control group in the visuo-spatial domain, poorer test scores were evident on Object Assembly and Block Design. Group BN also demonstrated a higher rate of Block Design Failure although between-group difference failed to reach significance. As was the case with Group AN, participants in Group BN had difficulties with designs based largely on diagonal lines. However, these tests are timed and scores are therefore vulnerable to both affective variables and poor impulse control. Whilst no evidence for the latter was found on correlational analyses, state anxiety was found to be modestly and inversely correlated with Block Design. The poorer scores may have also been due to clinical variables such as sudden weight loss and bulimic behaviours. However correlational analysis found only one weak relationship between Object Assembly and the use of laxatives. Interestingly very little relationships were found between clinical variables, particularly in relation to purging behaviours which have been hypothesised as a causal factor in the structural brain changes found in bulimia (Touyz and Beumont, 1994)

Methodological issues:

In the this study it was not possible to control for the potential effects of unstable nutritional or biochemical status. For those participants recruited via the specialist treatment centres, liaison with clinicians and screening of blood test results allowed unsuitable opt-ins to be excluded from the study. However, several of the
participants had not undergone routine physical examination and where results were available, it was not always possible to arrange a testing appointment within a week or so of examination. Several of the participants were no longer in therapy and those recruited from a self-help group had not been in contact with specialist services. Therefore it is possible that the results of the clinical groups in this study may have been confounded by unstable nutritional or biochemical status. It is worth noting however, that Mathias and Kent's (1998) controlled neuropsychological study included analysis of biochemical markers of anorexia. The study reported that between 20 per cent and 30 per cent of the clinical group group revealed blood test results outwith normal limits and reflecting possible medical problems such as anaemia, immuno-deficiency disorder, electrolyte disturbance and liver damage. However, correlational analysis revealed few significant associations between cognitive test performance and these biochemical markers.

A related methodological issue is the extent to which it is possible to assess cognitive functioning in a clinical group at differing levels of treatment and with potentially differing levels of motivation and fatigue effect. The present study did not attempt to measure aspects of motivation nor attempt to employ participants at initial contact with specialist services. Although, to an extent the use of pre-test questionnaires and correlational analysis does allow for some measurement of the relationship between clinical variables and cognitive test scores.

A further methodological factor not controlled for is the limited evidence of birth injury in anorexia (Artman, Grau, Adelmann & Schleiffer (1985; Cnattingius, Hultman, Dahl & Sparen, 1999). In the present study a crude measure of self-reported
birth complications was attempted although the limitations of retrospective self-report data are considerable. The evidence from studies which have reported case-note assessment of perinatal injury, has however revealed a lack of significant correlations with cognitive test scores or brain scan results (Kingston et al, 1996). Nonetheless, the possibility of birth injury cannot be ruled out as an explanatory factor.

Another methodological consideration is the measurement of the affective variables which were an important element of this study. On reflection it may have been prudent to use more measures of state anxiety particularly with regard to immediate pre-test anxiety. Alternatively it may have been useful to control for the potential effects of early test anxiety by counter-balancing test administration for half the participants in each group.

Finally, despite having discussed the issue in some detail, insufficient data was available to analyse the potential effects of the menstrual cycle phase on cognitive test performance.

**Relevance to clinical psychology:**

Neuropsychological impairments were found in a 30 per cent of participants with anorexia and in 23 per cent of those with bulimia nervosa. These results were attributable to a subgroup of poor performers and may be partially explained by the role of affective variables and personality characteristics. There is a case however, for routine neuropsychological screening in these clinical populations (Hamsher, Halmi & Benton, 1981). Given the finding of attentional deficits and problems in the recall
of auditory-verbal logically related material, there may be also case for shorter therapeutic sessions and an increase in the use of non-verbal communication. For example, by increased use of written or diagrammatic formulations or by the use of memory enhancing materials to explore therapeutic issues. Whilst the use of visual approaches to therapy in anorexia may appear counter-intuitive with a client group who have been reported to have visuo-spatial deficits, it is reasonable to speculate on the possible benefits of 'visual therapy' in improving possible hemispheric asymmetry. Crews and Harrison (1995) have speculated that cognitive therapy may result in increased left hemispheric activation and thus have a counterbalancing effect on the right-hemispheric activation associated with depression. There is also some evidence of decreased right hemispheric functioning in bulimia (Wu, Hagman, Buchsbaum, Blinder, Derrfler, Tai, Hazlett & Sicotte, 1990), and the use of visual material may therefore help to redress a possible asymmetry in this disorder.
3f. SUMMARY.

This cross-sectional quasi-experimental study attempted to clarify the nature and extent of neuropsychological impairment in anorexia and bulimia nervosa. A comprehensive battery of neuropsychological tests was administered to 30 adult females with anorexia nervosa, 30 adult females with bulimia nervosa and 30 matched controls. The results found that 30 per cent of participants with anorexia nervosa, 23 per cent of participants with bulimia nervosa and seven per cent of controls demonstrated at least one cognitive deficit at a level of neuropsychological impairment. However, between-group differences failed to reach significance and a large number of the deficits were attributable to a small subgroup of poor performers.

On analysis of the results of 18 cognitive tests, participants with anorexia were found to perform significantly more poorly than controls on 44 per cent of test measures. Similarly, participants with bulimia nervosa were found to perform significantly more poorly on 39 per cent of test measures. Correlational analyses revealed that anxiety and depression were influential variables, particularly with regard to test performance in the population with bulimia nervosa. Partialling-out procedures resulted in the removal of significance between the control group and participants with anorexia in two from eight results. Partialling-out procedures also removed significance between the control group and participants with bulimia nervosa in three from seven results. Significance on test score results remained between the control group and participants with anorexia on one measure of attention, four measures of learning and memory (auditory-verbal and visual) and one
measure of visuo-spatial/constructural ability. Although between-group differences were not established on measures of executive functioning, the overall results suggest mild neuropsychological difficulties across all domains. The results were not fully explained by affective or other psychological variables.

Significance on test score results remained between the control group and participants with bulimia nervosa on two tests of attention and two measures of auditory-verbal learning. The significant results in the domain of learning and memory were not predicted although may be explained with reference to attentional difficulties and the considerable influence of affective variables.

Correlational analyses revealed very few relationships between cognitive test performance and clinical variables.

These results are consistent with the literature on cognitive functioning in anorexia nervosa and the limited reports of cognitive functioning in bulimia nervosa.

In order to make any definitive conclusions about the incidence, severity and nature of cognitive impairments in eating disorders, further research should be focused on testing specific hypothesis rather than explore further between-group differences on cognitive test batteries. Mathias and Kent (1998) have suggested that it would be useful to explore the nature of impairment demonstrated by participants whose results are often 'lost' to quantitative analysis. Such an exploration would be admirably suited to a neuropsychological case-by-case analysis in conjunction with a functional imaging study. However, for those clinicians who wish to explore further the relationship between group scores, there is a need to focus upon studies which attempt to control for some of the biological and dietetic variables evident in the eating disorder population. There is also a need for further prospective studies which
measure cognitive functioning over a longer period of time and which may determine if impairment is progressive in those who do not respond to treatment. Such research may best be carried out from a multi-disciplinary perspective thus reflecting the psychological, psychiatric and dietetic complexity of eating disorders.
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APPENDICES.

Appendix 1: Consent Form

Appendix 2: Research Information Sheet - Eating Disorder
          Research Information Sheet - Control Group

Appendix 3: Opt-In Form.
Appendix 1:

Consent Form (Grampian Health Board/ University of Aberdeen).

CONSENT BY PATIENT/VOLUNTEER TO PARTICIPATE IN:

A controlled study of the effects of eating disorders on attention, learning, memory and problem solving.

NAME OF PATIENT/VOLUNTEER: .................................................................

NAME OF STUDY: Cognitive Functioning in Anorexia and Bulimia.

PRINCIPLE INVESTIGATOR: James I. Isles, Chartered Clinical Psychologist.

I have read the patient/volunteer information sheet on the above study and have had the opportunity to discuss the details with James Isles and ask questions. Mr. Isles has explained to me the nature and purpose of the tests to be undertaken. I understand fully what is proposed to be done.

I have agreed to take part in the study as it has been outlined to me, but I understand that I am completely free to withdraw from the study or any part of the study at any time I wish and that this will not affect my continuing treatment in any way.

I understand that this study is a research project designed to promote medical knowledge, which has been approved by the Joint Ethical Committee, and may be of no benefit to me personally.

I also understand that, where appropriate, my General Practitioner will be informed that I have taken part in this study.

I hereby fully and freely consent to participate in the study which has been fully explained to me.

SIGNATURE OF PATIENT/VOLUNTEER .....................................................

DATE ........................................

I confirm that I have explained to the patient/volunteer named above, the nature and purpose of the tests to be undertaken.

SIGNATURE OF INVESTIGATOR .................................................................

DATE .........................................................................

THIS FORM MUST BE KEPT IN SECTION A OF THE PATIENTS NOTES.
Appendix 2: Research Information Sheet

Cognitive Functioning in Eating Disorders - Information Sheet

This letter is to inform you about a research study which I am undertaking in the area of eating disorders. My study aims to examine whether or not processes such as attention, memory and learning are influenced by restrictive eating habits and other behaviours which associated with having an eating disorder. If you are female and you have an eating problem, I would ask you to consider being involved in this study. I will emphasise that should you not wish to participate, this will not effect your contact with the Eating Disorders Service. I hope that the following information answers any questions which you may have about the study, although I may be contacted at the address below to discuss the study further.

“What is involved and how do I participate?”

Should you decide that you would like to participate, then please complete the enclosed ‘opt-in’ form and return it to me in the stamped addressed envelope. Once you have had your initial appointment with a clinician at the Eating Disorders Service and if appropriate, I will send you several questionnaires to complete and return to me by post. I will then contact you to arrange a suitable time to ask you several questions concerning your background and give you approximately a dozen brief test items. The tests measure processes such as attention, memory and learning. Feedback on your test performance will be given on request. You will also be asked to complete a food intake and activity level diary for a period of 24 hours prior to testing. You will also require to refrain from taking alcohol in the 12 hour period prior to testing (where applicable).

“What happens to the results of my questionnaires and tests?”

All results will be gathered together for analysis and a report will be submitted to The University of Edinburgh as part fulfilment of a higher degree. If suitable, the study may be submitted for publication. However, neither your name nor any details which would make you identifiable will be recorded.

“What if I change my mind and decide to withdraw from the study?”

You can withdraw at any point during the study without explanation.

Jim Isles, Chartered Clinical Psychologist

CLINICAL PSYCHOLOGY SERVICES
Block A, Royal Cornhill Hospital, Aberdeen AB25 2ZH
Tel (01224) 663131 Extension 57532 Fax 01224 404045
Cognitive Functioning in Eating Disorders - Information Sheet

(Volunteer - comparison group)

This letter is to inform you about a research study which I am undertaking in the area of eating disorders. My study aims to examine whether or not processes such as attention, memory and learning are influenced by restrictive eating habits and other behaviours associated with having an eating disorder. If you are female, I would ask you to consider being involved in this study as a volunteer. (The use of a comparison group of people who do not have an eating disorder, allows me to establish whether any results found in the eating disorder group may also be found in the wider population and therefore may not be attributable to the eating disorder itself).

“Are there any exclusions to the study?” - Yes, those who are pregnant, or have epilepsy, head injury, or other physical problems which may influence the test results.

“What is involved and how do I participate?” - Should you decide that you would like to participate, then please complete the enclosed 'opt-in' form and return it to me in the stamped addressed envelope. Alternatively you may contact me at the address below. I will then contact you to arrange a suitable time to give you approximately a dozen brief test items. The tests measure processes such as attention, memory and learning. Feedback on your test performance will be given on request. Prior to testing, I will briefly ask you some questions about your current weight, height and other details related to the study. You may be required to complete a food intake and activity level diary for a period of 24 hours prior to testing. You will also be required to refrain from taking alcohol in the 12 hour period prior to testing (where applicable).

Following testing, I will give you several questionnaires to complete at your leisure and return to me in a stamped addressed envelope. The results of these questionnaires and your test results will be coded to preserve your anonymity.

“What happens to the results of my questionnaires and tests?” -

The results will be gathered together for analysis and a report will be submitted to The University of Edinburgh as part fulfilment of a higher degree. If suitable, the study may be submitted for publication. However, neither your name, nor any detail which would make you identifiable will be recorded.

“What if I change my mind and decide to withdraw from the study?” - You can withdraw at any point during the study without explanation.

Jim Isles, Chartered Clinical Psychologist.

CLINICAL PSYCHOLOGY SERVICES,
Block A, Royal Cornhill Hospital, Aberdeen AB25 2ZH.
Tel. 01224 663131 Ex. 57602 Fax. 01224 404045
Appendix 3: Opt-In Form.

NAME: ........................................................

I am willing to participate in this study

☐

I am NOT willing to participate in this study

☐

I require further information before I decide to participate in this study

☐

I may be contacted at the following address:-

........................................................

........................................................

........................................................

Postcode .................................................

Telephone number ................................................

May I leave a message if you are not available YES / NO

May I contact you at your work YES / NO

If yes, what is your work telephone number ................................................

May I leave a message should you not be available YES / NO

Thank you for completing the above.

PLEASE RETURN THIS FORM IN THE PRE-PAID ENVELOPE PROVIDED