NEUROPSYCHOLOGICAL PERFORMANCE IN ADOLESCENT ANOREXIA NERVOSA PATIENTS: A LONGITUDINAL STUDY

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SUMMARY

Background. *Impairments of neurocognitive performance in patients with anorexia nervosa (AN) have been reported in many studies, but no consensus has been reached. The purpose of our study was to develop a neuropsychological profile of adolescent AN patients and to determine whether or not the neuropsychological deficits are reversible with weight gain.*

Material and methods. *23 female AN patients who satisfied DSM-IV diagnostic criteria and 25 healthy female controls, matched for age, estimated IQ and educational level, participated in the study. 16 female subjects who attained psychiatric recovery participated in the follow-up study (longitudinal data). A battery of neuropsychological assessment methods was used, including: Vocabulary and Digit Span (WAIS-R), Auditory Verbal Learning Test, subtest for long-term memory of the Choynowski Memory Scale, the Rey-Osterrieth Complex Figure Test, the Benton Visual Retention Test, Diagnosis of Brain Damage, the Beck Depression Inventory, and the Memory Questionnaire.*

Results. *Several neuropsychological deficits were observed in AN subjects in both the verbal and nonverbal domains. Neuropsychological functioning improved after weight recovery, with significant changes on tests of verbal and nonverbal learning, long-term visual memory and psychomotor speed. In addition, cognitive performance did not significantly correlate with depressive symptoms or BMI in either the baseline or follow-up assessments.*

Conclusions. *AN patients exhibit several neuropsychological deficits, which tend to resolve at least partially after treatment. However, this improvement does not appear to be associated with an increase in BMI, and cannot be explained by the decreased severity of self-reported depressive symptoms.*

INTRODUCTION

Anorexia nervosa (AN) is a serious psychiatric illness characterized by significant weight loss, exaggerated fear of gaining weight or becoming fat, disturbances in the perception of one's weight or shape, and reproductive endocrine dysfunction. In addition to these symptoms, other psychological and somatic disorders and signs are often present. Neither the restricting nor the binge eating/purging AN subtypes are unaffected by the patient's biological and psychosocial condition and functioning.

Current hypotheses regarding the etiology of eating disorders (ED) are based on a biopsychosocial model. There is growing evidence that dysfunctions within the central nervous system (CNS) could be associated with abnormal eating behaviors and patterns (Braun & Chouinard 1992). AN has been the focus of intense neuropsychological research in recent years. Impairments of neurocognitive performance in adolescent AN patients have been reported in many studies. Relative to healthy unaffected controls individuals with AN present with deficits in several different neuropsychological domains, including verbal and nonverbal memory, verbal and nonverbal learning, attention, visuospatial ability, psychomotor speed, and executive functioning (Thompson 1993, Mathias & Kent 1998, Tchanturia et al. 2001, Koba et al. 2002, Murphy et al. 2002, Moser et al. 2003, Murphy et al. 2004, Ohrmann et al. 2004, Tchanturia et al. 2004, Steinglass et al. 2006). To date, preliminary evidence indicates that there may be some degree of improvement in cognition with weight recovery (Katzman et al. 2001, Kerem & Katzman 2003, Moser et al. 2003). However, several other recent studies have suggested that not all of these neuropsychological deficits resolve with weight recovery (Tchanturia et al. 2002, Tchanturia et al. 2004).

Despite the use of a wide range of neuropsychological assessment methods, some of the existing findings are contradictory. The nature of the neurocognitive deficits is not entirely clear. One possibility is that these disturbances in neuropsychological performance could arise as a result of a significant energy deficit caused by the restricted caloric intake. Additionally, the presence of co-morbid depression is likely to lead to poor neuropsychological functioning. On the other hand, neurocognitive deficits could represent a potential risk factor for AN onset and hence play a significant role as both a risk factor and a precipitating factor in the clinical manifestation of the disorder. There is no general consensus on the origin, nature, or degree of neurocognitive impairments in the clinical symptomatology of these EDs. Therefore, it is essential to determine whether the neuropsychological characteristic profile of patients with AN represent "state or trait" characteristics, and in consequence whether or not the deficits in different cognitive domains should be regarded as reversible with weight recovery. Further questions as to whether the neuropsychological impairments in AN patients are antecedents for the disordered eating behavior or a consequence of the ill-

ness should also be addressed. Additionally, it remains unclear whether the cognitive deficits are primary, directly caused by the dietary restrictions, or secondary, i.e. resulting from malnutrition.

In view of the uncertainties, further empirical studies on this problem are needed. The value of neuropsychological research involving AN patients seems obvious, in order not only to increase our theoretical understanding of the problem, but also to provide adequate preventive and neurocognitive rehabilitation programs for individuals with EDs. The main goals of our study, then, were as follows:

– to develop a neuropsychological profile of AN adolescent patients;
– to determine whether or not the cognitive deficits observed in their neuropsychological functioning are reversible with weight gain;
– to examine the relationship between BMI, self-reported depressive symptoms, and cognitive functions in individuals with eating disorder, both at baseline and in a follow-up study.

MATERIAL AND METHODS

23 female AN adolescent patients who satisfied DSM-IV diagnostic criteria and 25 healthy female controls, matched for age, estimated IQ and educational level, participated in the study (baseline data). 16 female subjects who had attained psychiatric recovery participated in the follow-up study (longitudinal data). The patients had been admitted for inpatient or outpatient treatment of severe eating disorders in the Child and Adolescent Unit of the Developmental Psychiatry Department at the Medical University of Gdansk, Poland, in the Child and Adolescent Clinical Hospital in Bialystok, Poland, or the Psychiatric Hospital in Suwalki, Poland. The treatment in the inpatient or outpatient setting was predominantly focused on individual and family therapy, which in consequence made it possible to compare the patients across both treatment settings. Furthermore, psychiatric comorbidity included depression (all of the participants had symptoms of mild to severe depression), obsessive-compulsive disorder (13% of the patient sample), and a history of childhood sexual abuse (13% of the patient sample). The control sample consisted of high school and college students. None of the participants had a known history of perinatal complications, psychotic or somatic disorders, or significant neurological insult, nor did they present with another medical condition that might have affected neurocognitive functioning. Participation in the study was voluntary. Consent to participation was obtained from the parents, the adolescent patients themselves, and their treating physicians. The most pertinent demographic and epidemiological data are shown in Table 1.

A variety of measures was used to collect background information and assess attention, memory and learning (both verbal and nonverbal), psychomotor speed, visuospatial ability, depression, and self-evaluated memory functioning.

Table 1. Basic descriptive data for the patient and control groups

		Patients (n I = 23, n II = 16)		Controls (n = 25)	
		M	SD	M	SD
Age (years)		17.02	2.00	18.13	2.17
Weight (kg)		45.64	5.16	60.12	5.61
Height (m)		1.67	.04	1.69	.04
BMI (kg/m²)		16.28	1.65	20.84	2.12
Duration of illness prior to admission (months)		25.26	23.40		
Weights (kg) at assessment II		50.56	10.36		
BMI (kg/m²) at assessment II		18.00	3.44		
Interval between assessments I and II (months)		6.51	1.28		
Handedness	Right	87.0 %		92.0 %	
	Left	13.0 %		8.0 %	
Assessment I	Hospitalization	60.9 %			
	Ambulatory	39.1 %			
Assessment II	Hospitalization	12.5 %			
	Ambulatory	87.5 %			

Note: BMI = Body Mass Index; M = mean; SD = standard deviation.

Background information was collected from the participants by using a questionnaire which covered basic demographic details. The medical history, clinical condition and BMI values used in the study were obtained from medical records and from a semi-structured clinical interview. A battery of standard neuropsychological assessment methods was used, including:
– the Vocabulary and Digit Span subtests of the Wechsler Adult Intelligence Scale-Revised (WAIS-R, Brzeziński et al. 1996);
– the Auditory Verbal Learning Test (AVLT, Choynowski & Kostro 1980);
– the subtest for long-term memory of the Choynowski Memory Scale (Radziwiłłowicz 2001);
– the Rey-Osterrieth Complex Figure Test (CFT, Strupaczewska 1990);
– Benton Visual Retention Test (BVRT, Sivan 1996);
– Diagnosis of Brain Damage (DBD, Weidlich & Lamberti 1996);
– Beck Depression Inventory (Beck & Steer 1984);
– Memory Questionnaire (Squire & Zouzounis 1988).

The vocabulary subtest of the WAIS-R was used to provide estimates of premorbid ability. The Digit Span subtest of the WAIS-R provides a measure of attention and immediate verbal memory. Additional measures of verbal memory and verbal learning were provided by the AVLT and the subtest of the Choynowski Memory Scale. Immediate and delayed nonverbal memory, psychomotor speed and visuospatial ability were assessed using the CFT. The BVRT also provides a measure of nonverbal memory, and the DBD was used in order to provide estimates of nonverbal memory and nonverbal learning.

The participants were tested in one session, lasting approximately 1 to 11 hr. Verbal and nonverbal tasks were presented randomly and in alternation. To minimalize the practice effect during the second assessment, equivalent alternative forms of the diagnostic methods were used. All tests were administered and scored by a trained psychologist.

RESULTS

An independent-sample t test was performed to determine whether the AN and control group were comparable in terms of the matching variables. The two groups were found to be comparable in terms of age, estimated IQ and educational level. As predicted, individuals with AN and healthy participants differed in terms of their BMI ($t(46) = -8.24$, $p < 0.001$), level of depression ($t(31) = 7.77$, $p < 0.001$), and level of self-evaluated memory functioning ($t(34) = 4.15$, $p < 0.001$).

Our primary interest was in the neuropsychological profile of female AN adolescent patients. An independent-sample t test revealed significant differences between participants with ED and unaffected controls on the following measures:

- attention;
- immediate and delayed verbal memory and verbal learning;
- immediate and delayed nonverbal memory;
- nonverbal learning;
- psychomotor speed;
- visuospatial constructional abilities (see Table 2).

In addition, individuals with AN displayed higher error rates compared to the control group, particularly in respect to confabulation (Choynowski Memory Scale, AVLT, DBD), perseveration (AVLT, DBD), and misplacement errors (BVRT).

Furthermore, a six-month follow-up study was conducted, then a paired-sample t test was used to determine whether neurocognitive functioning in the AN group was significantly different from baseline. Neuropsychological functioning improved after weight recovery with significant changes on tests of verbal and nonverbal learning, long-term visual memory and psychomotor speed (see Table 3).

Additionally, individuals with AN displayed high error rates, particularly in respect to confabulation (Choynowski Memory Scale). Nevertheless, the error rate on the DBD was lower (perseveration errors, inversion errors).

The average BMI value increased, with a mean increase of 1.88 (SD = 3.28), while the severity of self-reported depressive symptoms decreased, with a mean decrease of 5.56 (SD = 8.33), for all affected participants during second assessment, which is both clinically and statistically significant ($t(15) = -2.28$, $p = 0.037$; $t(15) = 2.67$, $p = 0.017$, respectively). In addition, cognitive performance did not significantly correlate with depressive symptoms or

Table 2. Neuropsychological functioning in AN adolescent patients (baseline)

	Patients	Controls	Comparison of groups (level of significance)	
	M (SD)	M (SD)	t	p
Digit Span (WAIS-R)				
Digit Span Forward	5.22 (1.31)	7.48 (1.78)	-5.03	**<.001**
Digit Span Backward	5.09 (1.78)	6.36 (1.38)	-2.78	**.008**
Total Score	8.26 (2.59)	11.52 (2.16)	-4.73	**<.001**
Choynowski's Memory Scale				
Correct	10.78 (3.05)	13.52 (2.23)	-3.55	**.001**
Errors	6.22 (3.05)	4.12 (3.45)	2.21	**.032**
Confabulation Errors	1.65 (1.72)	<.01 (<.01)	4.60	**<.001**
AVLT				
Trial 1	6.74 (2.09)	7.88 (1.45)	-2.17	**.036**
Trial 2	10.00 (2.08)	11.36 (1.77)	-2.43	**.019**
Trial 3	11.17 (2.12)	13.12 (.92)	-4.05	**<.001**
Trial 4	12.26 (1.73)	13.72 (1.06)	-3.47	**.001**
Trial 5	12.70 (1.86)	14.04 (.88)	-3.13	**.004**
Trials 1 to 5	52.87 (7.78)	60.12 (4.74)	-3.85	**<.001**
Errors	8.65 (7.03)	.04 (.20)	5.86	**<.001**
Recognition Trial	12.87 (1.32)	14.04 (1.02)	-3.44	**.001**
Perseveration Errors (Immediate Recall)	5.48 (6.57)	<.01 (<.01)	3.99	**.001**
Confabulation Errors (Immediate Recall)	2.57 (2.46)	<.01 (<.01)	4.99	**<.001**
Copy Trial Time	2.73 (.88)	2.12 (.59)	2.79	**.008**
Total Trials Time	5.21 (1.88)	4.10 (1.18)	2.41	**.021**
BVRT				
Correct	6.87 (1.14)	7.76 (1.09)	-2.76	**.008**
Errors	5.17 (2.44)	3.40 (1.80)	2.87	**.006**
Misplacement Errors	.74 (1.05)	.20 (.57)	2.17	**.037**
DBD				
Errors	7.73 (4.44)	3.84 (3.07)	3.52	**.001**
Confabulation Errors	1.64 (1.76)	.68 (.85)	2.32	**.027**
Perseveration Errors	3.91 (3.51)	2.04 (2.33)	2.17	**.035**

Note: WAIS-R = Revised Wechsler Adult Intelligence Scale; AVLT = Auditory Verbal Learning Test; Rey-Osterrieth CFT = Rey-Osterrieth Copy Figure Test; BVRT = Benton Visual Retention Test; DBD = Diagnosis of Brain Damage. Bold numbers represent level of significance (p < 0.05); M = mean; SD = standard deviation.

Table 3. Neuropsychological functioning in AN adolescent patients (follow-up)

	Assessment I	Assessment II	Significant differences between assessments I and II	
	M (SD)	M (SD)	t	P
Choynowski's Memory Scale – Confabulation Errors	1.13 (1.25)	2.19 (1.55)	-2.22	**.042**
AVLT – Trial 3	11.38 (2.06)	12.44 (1.89)	-2.86	**.012**
AVLT – Trial 4	12.56 (1.71)	13.56 (1.09)	-2.58	**.021**
AVLT – Trial 5	12.63 (1.66)	13.63 (1.25)	-2.28	**.037**
AVLT – Trials 1 to 5	53.69 (7.71)	58.63 (6.63)	-3.47	**.003**
Rey-Osterrieth CFT – Copy Trial Time	2.71 (.90)	1.88 (.63)	4.09	**.001**
Rey-Osterrieth CFT – Delayed Recall Trial Time	2.50 (1.38)	1.67 (.60)	3.14	**.007**
Rey-Osterrieth CFT – Total Time	5.31 (2.07)	3.72 (1.29)	4.58	**<.001**
Rey-Osterrieth CFT – Delayed Recall Trail	23.21 (5.33)	26.18 (3.39)	-2.19	**.044**
DBD – Trials 1 to 6	33.75 (7.32)	39.81 (8.40)	-2.67	**.017**
DBD – Errors	8.81 (4.49)	3.81 (3.33)	3.49	**.003**
DBD – Inversion Errors	1.81 (1.72)	.50 (.81)	2.89	**.011**
DBD – Perseveration Errors	4.94 (3.43)	1.56 (2.15)	3.32	**.005**

Note: AVLT = Auditory Verbal Learning Test; Rey-Osterrieth CFT = Rey-Osterrieth Copy Figure Test; DBD = Diagnosis of Brain Damage. Bold numbers represent level of significance (p < 0.05); M = mean; SD = standard deviation.

BMI in either baseline or follow-up studies. No significant difference was found between baseline and follow-up assessment on measures of self-evaluation of memory functioning.

DISCUSSION

Disordered eating behavior and weight regulation patterns with disturbances in attitudes and perceptions towards body shape and weight, which characterize individuals with AN, are not unrelated to the functioning of the central nervous system. Neuropsychological functions, such as attention, memory, learning, visuospatial abilities or psychomotor speed are generally considered to be mediated by cerebral cortex. Development of the frontal lobes is known to continue throughout late adolescence, contrary to the earlier maturation of other cortical regions (Davies & Rose 1999, Romine & Reynolds 2005). The psychopathological symptomatology that appears during adolescence, including EDs, is due not only to the complex interaction among biopsychosocial risk factors, but also to neurodevelopmental changes that could interfere with the phenomenological expression of the illness.

Therefore, inadequate daily caloric intake could be a hindrance to the developmental processes and have a significant impact upon neurocognitive performance in AN adolescents.

Our results suggest that several neurocognitive deficits can be identified in participants with AN in both the verbal and nonverbal domains. The impairments we found in attention, immediate and delayed verbal memory and verbal learning in individuals with AN are consistent with previous data (Thompson 1993, Mathias & Kent 1998, Seed et al. 2001, Murphy et al. 2002, 2004, Ohrmann et al. 2004). However, a number of investigations failed to find deficits in the attentional and verbal abilities of participants with eating disorders (Touyz et al. 1986).

Our data support the conclusions of a number of previous studies that have reported impairments in the immediate and delayed nonverbal memory, nonverbal learning, psychomotor speed and visuospatial capacities of participants with AN (Mathias & Kent 1998). Contrary to these findings, some researchers have failed to find differences between the performance of individuals with AN and a control group on neurocognitive measures of nonverbal functioning (Touyz et al. 1986).

Subsequent improvements after weight recovery occurred across different neuropsychological domains, with significant changes on tests of verbal and nonverbal learning, long-term visual memory and psychomotor speed. Our results support the conclusions of some studies (Moser et al. 2003), in contradistinction to evidence from others (Tchanturia et al. 2002, 2004).

Furthermore, consistent with the available literature, we found that depressive symptoms were not significantly associated with neuropsychological functioning (Mathias & Kent 1998, McDowell et al. 2003, Moser et al. 2003). The complete lack of a significant relationship between depression and cognitive ability, both in baseline and in follow-up, suggests that neurocognitive deficits are independent of the severity of self-reported depressive symptoms and that improvement in depression would not necessarily be associated with significant neuropsychological improvement.

Moreover, our data confirm, consistent with previous findings (Mathias & Kent 1998, Fassino et al. 2002, Moser et al. 2003), that there is no significant relationship between BMI and neuropsychological performance, either at baseline and or in follow-up. It is possible that BMI cannot be considered an adequate indicator of nutritional rehabilitation.

It is essential to point out that that studies of the quantitative aspects of neuropsychological performance in adolescent patients with AN remain rare. Our preliminary results suggest that visuospatial construction abilities measured by the Rey-Osterrieth CFT are impaired. The Gestalt approach, which tends to be the most mature and perceptually well-organized method of strategic performance on the Rey-Osterrieth CFT, for copy and delayed recall respectively, characterized 43.5% and 60.9% at baseline, as compared to 68.8% and 75% in follow-up. Furthermore, the specific dysfunctional charac-

teristics of AN patients' families tend to have an immense impact upon the adolescents' neurocognitive functioning. In this context it may be worth noting that the anorexic families tend to present patterns of enmeshment, overprotectiveness, rigidity, and lack of conflict resolution. One of the words provided by the AVLT word list is parents. 48% of all confabulation errors demonstrated by the participants at baseline was family, and 9% children; in the follow-up assessment, 30% of all confabulation errors were family and 6% child. The boundaries that keep anorectic family members overinvolved with each other and separate them from the world, the impeded separation-individuation process (Minuchin et al. 1978), seem to have a strong reflection in an abnormal confabulation pattern, and could act as a significant indicator of the psychopathological symptomatology.

The fact that individuals with AN display several deficits in their neuropsychological performance and the cognitive-behavioral aspects of the food-related psychopathology could be associated with the fundamental role of the frontal lobes in the cerebral manifestation of AN. Recent neuropsychological investigations have confirmed poor performance in AN participants on frontal lobe tests, such as the Trail Making Test (Tchanturia et al. 2004, 2004, Holliday et al. 2005), the Brixton Test (Tchanturia et al. 2004, 2004, Holliday et al. 2005) and the Wisconsin Card Sorting Test (Koba et al. 2002, Steinglass et al. 2006). Moreover, a significant reduction in regional cerebral blood flow (rCBF) and its association with impaired neurocognitive performance tend to be linked to the cortical and subcortical components of the presented disordered eating pattern (Stamatakis & Hetherington 2003, Ohramann et al. 2004, Lask et al. 2005). We hypothesize that these deficits could be interpreted in terms of limbic system functioning. This particular set of structures seems to be responsible for long-term memory, which is both structurally and functionally connected with short-term memory. Individuals with AN have been shown to perform particularly poorly on tasks of focused attention, memory and learning. In our estimation these data could reflect a specific reduction in working memory capacities.

CONCLUSIONS

The findings presented here suggest that patients with AN exhibit several neuropsychological deficits, which tend to resolve at least partially after treatment. However, this improvement does not appear to be directly correlated with an increased BMI, and cannot be explained by a decrease in the severity of self-reported depressive symptoms. Therefore, it would be important to continue neuropsychological research in the AN population in order to eliminate some of the methodological limitations of our study. A more homogenous and larger sample, adequate definition of psychiatric recovery (not only in terms of weight recovery and decreased depressive symptoms), data obtained from long-term recovered patients and additional data from struc-

tural and functional neuroimaging study techniques would provide important background knowledge to help identify the underlying mechanisms in the etiology and persistence of EDs.

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