

Computed Tomography of Anorexia Nervosa

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Computed tomographic studies were performed in patients with anorexia nervosa to confirm the observations of other authors on so-called reversible cerebral atrophy. In 21 of 23 cases a marked enlargement of the cortical sulci and the interhemispheric fissures was observed, which was reversed in a second computed tomographic study in 11 patients 4 weeks after they had reached normal weight. Psychological tests were carried out at the same time as the computed tomographic studies to correlate the changes in the brain tissue with cerebral function. Data obtained in each group of tests for both the initial and the follow-up studies were analyzed using the Student *t*-test. The differences were found to be statistically significant ($p = 0.01$ in most cases). The results indicate that anorexia nervosa is not only a psychodynamic problem, but also one in which an organic brain lesion plays an important role during the course of the illness.

There are several communications about findings in cranial computed tomography (CT) of patients with anorexia nervosa [1–3]. A so-called reversible cortical cerebral atrophy occurs in a small percentage of patients, while ventricular dilatation seems rare. Similar findings have resulted from pneumoencephalographic studies of patients with anorexia nervosa, which have also indicated ventricular enlargement in some cases [4]. However, none of these authors has correlated the morphologic cerebral findings with cerebral function as shown by psychological testing.

Subjects and Methods

CT was used to study 23 patients (22 girls, one boy) with anorexia nervosa treated as inpatients. Weight loss in each case was more than 2 standard deviations below normal weight related to height. Mean data are given in table 1.

Because the cortical sulci and the interhemispheric fissure are not visible in CT of normal adolescents of this age, we counted the visible cortical sulci in three slices from the temporal, frontal, and parietal convexity of the brain. Slice 1 was at the level of the frontal horns, slice 2 was at the level of the cella media, and slice 3 was 9 mm above the cella media. The numbers of sulci counted were added and then divided by 23 to get a mean value for the group. The mean in this first study was 43.1 (SD, 21.8).

To determine the width of the inner cerebrospinal fluid (CSF) spaces the Huckman number and the cella media index were used. The third ventricle was measured in millimeters at the level of its

widest diameter. The degree of enlargement of the interhemispheric fissure was then graded as 0–3, with 0 being not visible, and 1, 2, and 3 being assigned for mild, moderate, and severe, respectively. In the first study the mean interhemispheric fissure grade was 2.2 (SD, 0.9).

In 11 patients CT was repeated 4 weeks after a normal and stable body weight had been achieved. The values obtained by the first and second studies were compared, and the differences analyzed statistically according to the Student *t*-test. Electroencephalographic (EEG) data were recorded at both CT studies. Psychological tests were also administered to obtain values for concentration, reaction time, intelligence quotient (IQ), and perceptual speed each time. These test values were compared and the differences analyzed statistically by the Student *t*-test. Other appropriate tests were conducted each time to determine the levels of blood cells, hemoglobin, hematocrit, electrolytes, enzymes, serum albumins, creatinine, and urea.

Results

Widening of the cerebellar sulci was observed in two cases. The lateral ventricles were slightly dilated in six cases, including the third ventricle in three cases. In one case, dilatation of the third ventricle occurred only with enlarged cortical sulci. The cases presenting with enlarged inner and outer CSF spaces and with wide cerebellar sulci were those with the most severe and longest course of illness. Conversely, there were two patients with normal CT, and they had the shortest duration of illness of all our subjects (2 months with the mean of 15 months).

Table 2 reports the mean values of the outer CSF spaces and the psychological tests in the 11 patients studied twice. A complete normalization of the sulci and the interhemispheric fissures occurred in four cases. In this group there was one patient with additional ventricular dilatation whose CT scan was unchanged from the first to the second study, despite a marked improvement of the outer CSF spaces. The two patients with initially wide cerebellar sulci showed normal sulci in the second study. The values of the ventricular system were too few to be analyzed statistically. Comparing the mean values of the first and second assessments we found a highly significant improvement of concentration and reaction time, a significant improvement of perceptual speed, and no change of the IQ. Laboratory data were normal except for gamma glutamate transferase. Serum albumin and the hematocrit were unchanged in each patient.

EEG recordings were slightly abnormal (e.g., generalized abnor-

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TABLE 1: Anorexia Nervosa: Clinical Findings in 23 Patients at Initial CT

	Mean (SD)
Age (years)	15.2 (2.1)
Duration of illness (months)	15.3 (10.2)
Body weight (kg)	36.5 (6.2)
Weight loss from onset of illness (kg)	16.5 (5.0)

TABLE 2: Means of Cortical Sulci Counted, Hemispheric Fissure Grades, and Values of Performances in Psychological Tests in 11 Patients Studied Twice by CT and Tests

	First Study	Second Study	Significance
	Mean (SD)	Mean (SD)	
No. of cortical sulci	47.2 (17.6)	7.6 (7.2)	$p = 0.01$
Grade of interhemispheric fissure	2.3 (0.7)	0.6 (0.6)	$p = 0.01$
Concentration	52.6 (14.2)	66.3 (9.2)	$p = 0.01$
Reaction time	179.3 (8.6)	194.6 (4.6)	$p = 0.01$
IQ	110.6 (8.3)	114.3 (9.3)	Not significant
Perceptual speed	4.3 (0.9)	5.8 (0.9)	$p = 0.05$

malities and dysrhythmias) in most cases at the time of the first CT study. EEG was normalized in the second study in those patients with diminution or appearance of the enlarged outer CSF spaces.

Discussion

Our findings on CT agree with those of other researchers [1-3], who reported enlargement of the cortical hemispheric sulci in five cases of anorexia nervosa, as well as widening of the interhemispheric fissure. These are normally not identified on CT of adolescents at the age of 15, which was the mean age of our subjects and those in the above-mentioned studies. The appearance of such changes in the cerebral sulci supposes a duration for the illness of several months or more. Our two cases with normal CT had a duration of 2 months only, and the single case in the literature with normal CT had a course of but 6 months. The amount of weight loss is important also; all our patients showed a dramatic weight loss over 6-20 months. Enlargement of the lateral ventricles, the third ventricle, and some cerebellar sulci in a few very severe cases have been reported.

Enlargement of the cortical sulci and of the interhemispheric fissures with normal-size ventricles and basal cisterns is a typical CT finding in patients with anorexia nervosa. The typical changes in the brain are generally denoted as cortical cerebral atrophy. This is either a static or a progressive condition according to its pathological definition [2].

But the "cortical atrophy" of patients with anorexia nervosa is not the most common atrophy. In seven of our 11 patients studied by CT a second time after achieving normal weight, and in a few other cases reported, a marked reversibility of the enlarged cortical sulci and the interhemispheric fissures was observed, as was the widening of the cerebellar sulci in two cases. In four cases repeat CT findings were completely normal. A reversibility of enlargement

of the lateral ventricles occurring in one controlled case was not evident despite a favorable remission of the cortical sulci. A similar case reported by Heinz et al. [2] also showed a marked decrease in width of the enlarged inner CSF spaces in a repeat CT study.

Such observations should remind the radiologist to use care in diagnosing cerebral atrophy by CT unless the static or progressive condition is confirmed. "Reversible atrophy" seems to us to be an inadequate designation, and we prefer to speak in diagnostic terms which do not anticipate a traditionally defined entity of illnesses. "Diminution of the brain volume" could be severe as an initial or preliminary CT diagnosis, for such changes occur in a similar way in chronic alcoholism, in patients treated with corticosteroids, or those undergoing chemotherapy because of malignant illness. Pseudoatrophy [3] and cerebral dystrophy [5] have also been used to denote these findings in CT.

But there is no explanation for the etiology of these changes in the brain, especially of the cortical tissue. There were no signs of dehydration, and the normal values of serum albumin indicated that malnutrition was not severe. The physical state of our patients with anorexia nervosa did not compare with that of patients with malabsorption or malnutrition following disorders of the gastrointestinal tract, or with that of chronically starving people. (It would be interesting to obtain information on cranial CT findings and on their course in such other patients to compare them with those found in anorexia nervosa.) The slight abnormalities of the gamma glutamate transferase in our patients, indicating a dysfunction of the liver, cannot serve as an explanation. Except for their influence on EEG, the significance of these morphological changes in the brain for cerebral function has not been discussed in the literature.

But EEG is only one parameter able to show normality or disorders of cerebral cortical function, and an uncertain one at that. Therefore, it is remarkable that nearly every patient we studied presented with EEG abnormalities at the time of the first CT, even though these bioelectrical disorders were not severe enough to suggest morphologic cerebral changes. We believe a more confident way of determining these is the assessment of a patient's performance in a standardized test battery. Our results indicate an improvement of some cerebral function corresponding to the improvement of the morphologic state of the cortex, but further studies must be done to confirm these preliminary findings.

REFERENCES

1. Enzmann DR, Lane B. Cranial computed tomography findings in anorexia nervosa. *J Comput Assist Tomogr* 1977;1:410-414
2. Heinz ER, Martinez J, Haeggeli A. Reversibility of cerebral atrophy in anorexia nervosa. *J Comput Assist Tomogr* 1977;1:415-418
3. Sein P, Searson S, Nicol AR, Hall K. Anorexia nervosa and pseudo-atrophy of the brain. *Br J Psychiatr* 1981;139:257-258
4. Heidrich R, Schmidt-Matthias H. Encephalographische Befunde bei Anorexia nervosa. *Arch Psychiatr Nervenkr* 1961;202:183-201
5. Artmann H, Grau H, Schleiffer R. Dystrophische Hirnveränderungen bei Anorexia nervosa. Presented at the meeting of the German Society of Neuroradiology, Hamburg, November 1982