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Glucose Metabolism in the Caudate Nuclei of Patients with Eating Disorders, Measured by PET

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Summary. Regional cerebral glucose metabolism was measured with ^{18}F-2-fluoro-2-deoxyglucose and positron emission tomography in nine patients with bulimia nervosa and in seven patients with anorexia nervosa. Relative caudate glucose metabolism (caudate glucose metabolism divided by global cerebral glucose metabolism) was significantly higher in anorexia nervosa than in bulimia nervosa, suggesting that caudate hyperactivity is characteristic of the anorexic state. Whether increased caudate function is a consequence of anorexic behaviour or whether it is directly involved in the pathogenesis of anorexia nervosa is an issue still to be clarified.

Key words: Bulimia nervosa – Anorexia nervosa – Caudate nuclei – Glucose metabolism – Positron emission tomography

Introduction

Recently we reported on the regional cerebral glucose metabolism in patients with anorexia nervosa as assessed by positron emission tomography (PET). The main finding was a significant increase in the metabolic rates in both caudate nuclei. A re-examination of the patients after weight gain revealed a normalization of caudate glucose hypermetabolism (Herholz et al. 1987). Meanwhile, our finding of an increased glucose metabolism in the caudate nuclei of anorexic patients has been confirmed by others (Delvenne et al. 1990).

As patients with anorexia nervosa and bulimia nervosa display numerous psychopathological, neuroendocrine, metabolic and neuroradiological similarities (e.g. Garner et al. 1985; Pirke et al. 1985; Krieg et al. 1989), we raised the question of whether patients with bulimia nervosa also show increased glucose metabolism in the caudate nuclei as an abnormality characteristic of both types of eating disorders.

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Patients and Methods

After giving written informed consent, nine female patients with the DSM-III-R (1987) diagnosis of bulimia nervosa took part in the study. The mean age ± SD of the patients was 23.7 ± 4.9 years, the mean body weight 98 ± 5% of the ideal body weight (IBW)[1] and the mean duration of the eating disorder 6.4 ± 5.1 years. Patients were checked for normal plasma levels of ketone bodies to rule out interference with glucose metabolism.

The measurement of regional cerebral glucose metabolism, using ^{18}F-2-fluoro-2-deoxyglucose as a tracer, was carried out in the same way as was described in detail in the study on anorexic patients (Herholz et al. 1987). The metabolic rates of 15 defined brain structures (for specification, see Fig. 1) were computed as relative rates (i.e. regional metabolic rate divided by global brain metabolic rate). The metabolic rates of the patients with bulimia nervosa were compared with those previously assessed in seven female patients with anorexia nervosa (mean age: 20.0 ± 2.0 years; mean body weight: 72 ± 6% IBW, mean duration of the eating disorder: 3.7 ± 2.6 years) (Herholz et al. 1986). Radiation protection rules prohibited the establishment of a control group of young healthy females. However, as the main aim of the PET study was the comparison of bulimia nervosa with anorexia nervosa patients, we deemed it acceptable to use eight healthy males (mean age: 27.0 ± 3.1 years) as an additonal reference group.

Results

The measurement of global cerebral glucose metabolism (μmol/100 g/min) revealed similar rates in patients with bulimia nervosa (mean ± SD: 39.7 ± 3.0), anorexia nervosa (36.3 ± 4.8) and control subjects (39.1 ± 3.0) (ANOVA, $P = 0.22$, NS). However, as shown in Fig. 1, the mean relative rate of caudate glucose metabolism was significantly higher in the patients with anorexia nervosa (1.32 ± 0.08) than in those with bulimia nervosa (1.22 ± 0.06) and in the control subjects (1.21 ± 0.08) (ANOVA, $P = 0.01$; with Tukey's studentized range test for comparison of multiple means: significant for anorexia nervosa vs bulimia nervosa and anorexia nervosa vs controls, not significant for bulimia nervosa vs controls). In all other

[1]According to the tables of the Metropolitan Life Insurance Company (1959) Stat Bull 40:1–17

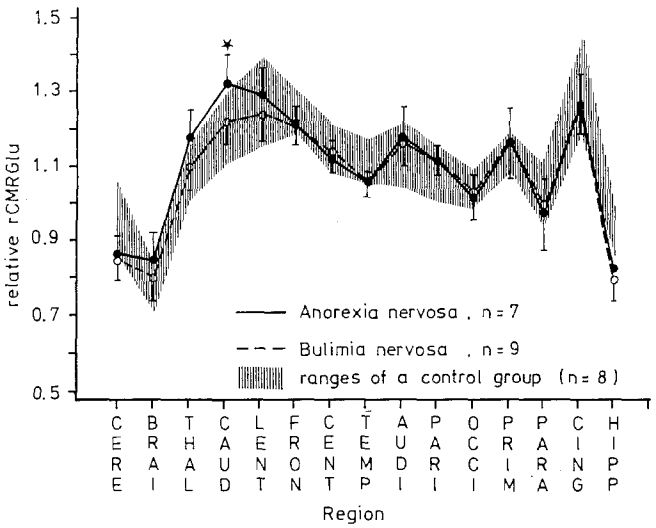

Fig. 1. Relative regional glucose metabolism *(rCMRGlu)* in patients with anorexia nervosa and bulimia nervosa. *CERE,* Cerebellum; *BRAI,* brain stem; *THAL,* thalamus; *CAUD,* caudate nucleus; *LENT,* lentiform nucleus; *FRON,* frontal cortex; *CENT,* central sensorimotor cortex; *TEMP,* temporal association cortex; *AUDI,* primary auditory cortex; *PARI,* parietal cortex; *OCCI,* occipital cortex; *PRIM,* primary visual cortex; *PARA,* paravisual cortex (mainly cuneus); *CING,* cingulate; *HIPP,* hippocampal structures. * $P = 0.01$

supratentorial regions assessed, no significant differences in the metabolic rates could be detected among the three groups studies (Fig. 1).

Discussion

This appears to be the first study comparing regional cerebral glucose metabolism in patients with bulimia and anorexia nervosa and the first to reveal significant differences in caudate glucose metabolism between these two types of eating disorders. The finding that patients with bulimia nervosa do not exhibit as high rates for caudate glucose metabolism as anorexic patients disproves the assumption that caudate glucose hypermetabolism is typical of both types of eating disorders. The results rather indicate that neurobiological alterations, which are present in anorexia but not bulimia nervosa, are responsible for caudate hypermetabolism. Our study also supports the finding of Wu et al. (1990), who, however, only compared bulimia nervosa patients with control subjects and could detect no significant differences between these two groups with regard to their basal ganglia metabolism.

Abnormalities in caudate glucose metabolism, as a sign of altered neuronal activity, have not only been observed in patients with anorexia nervosa but also in patients with other psychiatric disorders. Thus patients with schizophrenia, affective and obsessive compulsive disorders have been described as displaying a reduced caudate glucose metabolism in comparison with control subjects (Buchsbaum et al. 1982, 1986; Baxter et al. 1985; Martinot et al. 1990). The reason for altered caudate activity in these psychiatric disorders is still far from clear, and has given rise to various speculations. As is well known, the caudate nuclei, as part of the extrapyramidal motor system, contain mainly dopaminergic neurons. Experiments in rats suggest that increased motor activity enhances dopamine turnover (Broocks et al. 1989). Since, in contrast to bulimic patients, most of the anorexic patients are hyperactive (Kron et al. 1978), we speculate that this behaviour increases caudate metabolism in anorexia nervosa patients. As caudate nuclei also play an important role in cognitive processing (Teuber 1976; Cools et al. 1984), one could further hypothesize that caudate hyperactivity in anorexia nervosa is an expression of an altered cognitive state or function. However, in a vigilance performance test we could find no significant differences between patients with anorexia and bulimia nervosa (Laessle et al. 1989).

It is very likely that in anorexia nervosa caudate hyperactivity, which normalizes after weight gain (Herholz et al. 1987), is a consequence of the anorexic state or behaviour. However, one also has to consider the possibility that caudate hyperactivity is directly involved in the pathogenesis of anorexia nervosa, which, in our opinion, finds no convincing support in the current literature on the neurophysiology of feeding (e.g. Rolls 1984). Until future studies have clarified this issue, we remain more reserved than Wu et al. (1990) in interpreting the differences in caudate activity in anorexic and bulimic patients as a hint that anorexia and bulimia nervosa are aetiologically two distinct psychiatric disorders.

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