Gastrointestinal Transit is Delayed in Patients with Bulimia

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Orocecal transit time was measured in 13 patients with bulimia and in 15 healthy age- and sex-matched controls. Diagnosis was made according to DSM-III. Bulimic patients had a signficantly longer transit time. Patients with a history of anorexia nervosa (n = 4) had a longer transit time than patients without such a history (n = 8). These results indicate that gastrointestinal transport remains disturbed when anorexia nervosa develops into bulimia. The indicator intermittent dieting did not show significant correlations with transit time.

Bulimic behavior may cause gastrointestinal disturbances. Vomiting can cause erosions and ulcera in the esophagus (Bailey, 1984; Cuellar & Van Thide, 1986). Binge attacks, during which often huge amounts of food are ingested, have been reported to cause gastric rupture and intestinal infarction, which may become life threatening (Edwards, 1985; Breslow et al., 1986; Mitchell, 1986). Laxative abuse is often reported by bulimic patients (Mitchell et al., 1985; Lacey & Gibson, 1985). All three behaviors (vomiting, binging, and laxative abuse) may disturb intestinal transport and consequently secretion and absorption functions that are closely linked to it (Ruppin, 1985).

We report here the measurement of the gastrointestinal transit in bulimic patients. Correlations with eating behavior and metabolic state were studied.

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SUBJECTS

Thirteen female patients with bulimia and 15 controls matched for sex and age participated in the study. The age of the bulimics was 22.3 ± 1.8 years (controls 23.5 ± 1.8 years). Body mass index (BMI) was calculated according to the formula weight/height^2 (kg/cm^2) and was 21.4 ± 2.8 kg/cm^2 (controls: 21.1 ± 1.9). Diagnosis was made using the DSM-III criteria (American Psychiatric Association, 1980). Four patients had a history of anorexia nervosa. Laxative abuse was reported in one patient. The duration of the illness in all cases was longer than 1 year. More than two bulimic attacks occurred per week. The patients participated in the study prior to entering an outpatient treatment program in our clinic. Control subjects were carefully studied. Subjects with psychiatric and somatic diseases, particularly gastrointestinal and eating disorders, were excluded.

METHODS

Food Diary

The bulimic patients kept in a food diary a standardized record of eating and drinking over a 7-day period preceding the transit study. The diaries were evaluated according to Souci et al. (1981/1982).

Measurement of Triiodothyronine (T3) and retro T3

On the morning of the transit study (8:00 a.m.) 5 ml blood was drawn from an antecubital vein. T3 and retro T3 were measured by radioimmunoassay (Serono, Freiburg, FRG). The interassay variability was 6.2% at an average concentration of 1.5 ng/ml for T3 and 8.3% at an average concentration of 120 pg/ml for retro T3.

Orocecal Transit Time

The orocecal transit time was measured by a hydrogen breath test. Lactulose, a carbohydrate which is neither metabolized nor absorbed in the small intestine, was given orally. Upon reaching the cecum it is metabolized by bacteria, whereupon hydrogen (H_2) is liberated, appearing in the breath 4–5 minutes after lactulose has reached the cecum (Bond & Levitt, 1975). Hydrogen is measured in the breath by electrochemical detection.

Subjects were advised to avoid carbohydrate-rich food on the day preceding the test. After an overnight fast (smoking prohibited) the test began at 8:30. The subjects drank 15 ml lactulose syrup consisting of 13 g lactulose dissolved in 150 ml water. Starting 10 minutes before the application and at 10-minute intervals thereafter, two measurements of H_2 were conducted over a 3-hour period. Air was exhaled into a syringe through a T-shaped plastic piece. The air was analyzed for H_2 in a GMI-H2 Monitor. (GMI Medical LTD, Inchinnan Estate, Renfrew, Scotland). Since no standard definition for the increase of H_2

Table 1. Number of successful transit measurement during the first and second session.

	Transit Determined in the First Session	Transit Determined in the Second Session	Transit Not Measurable
Bulimia (n = 13)	9 (69%)	3 (23%)	1 (8%)
Controls (n = 15)	13 (87%)	2 (13%)	0 (0%)
Whole group (n = 28)	22 (79%)	5 (18%)	1 (3%)

marking the transit time has been used up to now (Armbrecht et al., 1986; Korth et al., 1984), we determined the transit time as measured by H_2 by means of three different algorithms described elsewhere in detail (Galfe et al., in preparation). Since each of the algorithms occasionally may yield erroneous results, transit time was calculated only when at least two produced identical results. When no transit time could be determined in the first study, a second study over 4 hours instead 3 hours was conducted.

Statistical Evaluation

Groups were compared by the Mann-Whitney U-test. The Moses test was used to search for significant differences in interindividual variability between groups. Pearson correlation coefficients were calculated.

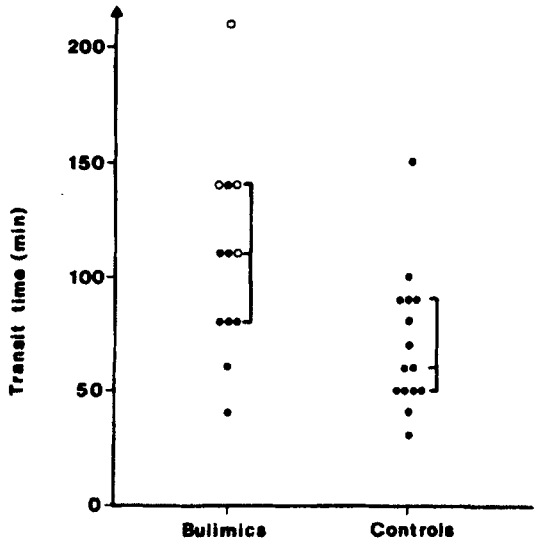

Figure 1. Transit time in bulimic patients and controls. The open circles mark bulimic patients with a history of anorexia nervosa.

Table 2. Triiodothyronine (T3), retro T3, body mass index (BMI), number of binge attacks (NOBA), number of calories eaten during binge attacks in one week (BACAL), and number of calories eaten outside of binge attacks during one week (SUMCAL)

Variable	$\bar{X} \pm$ SD	Correlation with Transit	p	n
T3 (ng/ml)	1.38 ± 0.25	0.49	0.11	12
R-T3 (pg/ml)	267.70 ± 129.56	0.48	0.16	10
Q R-T3/T3	198.29 ± 86.30	0.12	0.75	10
BMI (kg/m^2)	21.44 ± 2.80	−0.39	0.21	12
NOBA	8.30 ± 9.31	0.19	0.60	10
BACAL (kcal/week)	18934.70 ± 28723.07	0.27	0.45	10
SUMCAL (kcal/week)	9353.90 ± 2982.17	−0.43	0.21	10

RESULTS

As indicated in Table 1, a transit time was determined in 69% of the bulimic patients and in 87% of the control subjects in the first session. An additional 23% of the bulimics and 13% of the controls were measured during the second, longer study. In one bulimic patient no positive result was obtained in either session. This patient was also the only one who reported laxative abuse. Figure 1 lists the transit times in bulimics and controls. No significant differences were found in variability. There was a broad overlap between patients and controls. The bulimics had significantly longer transit times ($p < .025$). When bulimic patients with a history of anorexia nervosa (open circles, Fig. 1) were compared with patients without such a history, a significant difference ($p < .025$) was found. When the bulimic patients without a history of anorexia nervosa were compared with controls, no significant difference remained.

Table 2 lists the correlations between the transit time and T3, retro T3, BMI, the number of bulimic attacks per week, the total amount of calories consumed during binge attacks in one week, and the total amount of calories consumed outside of binge attacks during one week. No significant correlations were obtained in the patient group.

DISCUSSION

The orocecal transit time was measurable in all control subjects and in 12 out of 13 bulimic patients. In one patient with laxative abuse no transit time was measureable. Since some persons do not have H_2-producing bacteria (Lembke & Caspary, 1983), it remains unclear whether this bulimic patient belonged to this group or had destroyed the H_2-producing bacteria by laxative abuse.

Although the overlap of the transit time between bulimics and controls is high, the groups are significantly different. This finding indicates that at least some bulimic patients have pathologically prolonged orocecal transit. From this fact we can conclude (Lübke & Wienbeck, 1985; Ruppin, 1985) that prolonged digestion and resorption periods occur in some bulimic patients. Similar obser-

vations have been made in anorexia nervosa. It has been shown that as a consequence of permanent dieting, the gastric emptying is prolonged (MacCallum et al., 1985; Lautenbacher et al., 1986; Stacher et al., 1986; Hölzl et al., 1984).

The method used here does not allow separate consideration of gastric and intestinal transit. The regulation of both phases of the transit correlates only weakly (Read, 1984). Since the assumption that the gastric phase of the transit can be neglected when fluid meals are consumed was proven incorrect (Korth et al., 1984), we must assume that some bulimic patients show either a prolonged gastric or intestinal transit or a combination of both.

Many bulimic patients indulge in intermittent dieting, accompanied by large fluctuations in body weight (Pirke et al., 1987). Weight reducing diets in bulimic patients are accompanied by a decreased conversion of thyroxine (T4) to T3 and an increased conversion to the biologically inactive retro T3. (Pirke et al., 1985). Transit times in the bulimic group did not correlate to T3, retro T3, or the ratio of both. BMI also did not correlate to transit. The sum of calories eaten per week outside the binge attacks, which may also serve as an indicator of intermittent dieting (Pirke et al.,1987), also did not correlate to transit time. The observations do not support the hypothesis that intermittent dieting causes a prolonged orocecal transit in bulimic patients.

Patients with a history of anorexia nervosa had longer transit times than patients without such a history. This indicates that disturbances of gastrointestinal transport acquired while suffering from anorexia nervosa do not normalize when anorexia nervosa develops into normal-weight bulimia.

In conclusion, our data suggest that neither binging, vomiting, nor intermittent dieting causes prolonged transit in bulimia. It appears that the disturbed transit which develops in anorexia nervosa does not normalize when anorexia develops into bulimia once normal weight has been achieved.

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