Oesophageal and gastric motor activity in patients with bulimia nervosa

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Abstract
Previous studies showed that symptoms of oesophageal motor disorders can be misinterpreted as indicating anorexia nervosa and that in primary anorexia nervosa gastric motility is frequently impaired. We investigated in 32 women with bulimia nervosa whether symptoms of oesophageal motor disorders could be obscured by or be mistaken as forming part of bulimic behaviour, and whether impaired gastric motility was frequent as well. Oesophageal motility was normal in 18 of 26 patients studied, another four had incomplete lower eosophageal sphincter relaxation. Two patients had vigorous achalasia and each one achalasia and diffuse oesophageal spasm, all of whom experienced two types of vomiting: one selfinduced and one involuntary, in which the vomit was non-acidic and tasted as the preceding meal. Gastric emptying of a semisolid meal was studied in all patients except of the eight with oesophageal motor abnormalities. Emptying was significantly slower than in healthy controls and grossly delayed in nine of 24 patients. Antral contraction amplitudes were lower and increased less postcibally than in controls. In conclusion (i) bulimic behaviour can obscure symptoms of oesophageal motor disorders and (ii) gastric emptying is frequently delayed in bulimia nervosa.

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Bulimia nervosa is an eating disorder related to primary anorexia nervosa, typically beginning in adolescence or early adult life. The disorder is diagnosed increasingly frequent, and bulimic behaviour has been reported to be increasingly prevalent especially on college campuses.[1,2] Clinically significant bulimia, however, seems to be rare.[3,4] Bulimia is characterised by recurrent episodes of binge eating – that is, the rapid consumption of a large amount of food in a discrete period of time, a feeling of lack of control over eating behaviour during the eating binges, and, in order to prevent weight gain, the engagement of the affected individual in either selfinduced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise. Induced vomiting decreases the pain of abdominal distension and allows either continued eating or termination of the binge, but may lead to electrolyte imbalance and dehydration. The recently revised diagnostic criteria require a minimum average of two binge eating episodes a week for at least three months and a persistent over-concern with body shape and weight.[5] In contrast with primary anorexia nervosa, the concern with weight does not lead to a fall below a minimal normal weight, although the weight loss may be substantial. Most research on bulimia has dealt with psychosocial and therapeutic aspects of the disorder, whereas relatively few work has been done in the field of bodily functions. In particular, the functioning of organs likely to be affected by the bulimic behaviour, that is, the stomach and the oesophagus, has received little attention. This is surprising all the more, as symptoms indicative of disordered gastrointestinal function, such as postprandial fullness, early satiety, bloating and epigastric pain, are encountered in a substantial proportion of patients.[6,7] That such symptoms in fact often reflect grossly disordered gastric motor activity is suggested by studies on patients with primary anorexia nervosa, which revealed that delayed gastric emptying[8–17] and motor activity[13–15] are frequent features in that disorder. In patients categorised as having primary anorexia nervosa it was found that on primary evaluation, symptoms of vomiting and weight loss were mistaken in several instances as indicating primary anorexia nervosa, whereas the reevaluation of the patients' history and subsequent fluoroscopic, manometric, and pH-metric studies revealed that they in fact suffered from oesophageal motor disorders such as achalasia, diffuse oesophageal spasm and gastro-oesophageal reflux.[10,12] Studies of gastric motor function in patients with bulimia yielded contradictory results: gastric emptying was found to be delayed in part of the patients[18] or to be within the normal range,[16,17] and the motor activity of the gastric antrum was reported to be normal.[16] The present study was aimed at investigating, in consecutive patients diagnosed as having bulimia nervosa, whether symptoms of disordered oesophageal motor function, such as vomiting and weight loss, were misinterpreted at primary evaluation as forming part of the bulimic behaviour or even led to a misdiagnosis of bulimia, and, whether gastric emptying and antral motor activity were similarly affected in bulimia as in primary anorexia nervosa.

Methods

SUBJECTS AND PROCEDURE
Thirty two female patients diagnosed as suffering from bulimia nervosa and presenting consecutively to the Division of Psychosomatic Medicine for hospitalisation and treatment participated in this study. The age of the patients ranged from 17 to 40 years ($\bar{x}$=24·6 yr (0·9) SEM) and their percentage of desirable weight from 77 to 113% ($\bar{x}$=90·8% (1·6) SEM). None of the patients had any evidence of diabetes mellitus, connective tissue disease or neuropathy,

and none had undergone gastric surgery or vagotomy. The diagnosis of bulimia nervosa was made according to the specific criteria outlined in the DSM-III[19] and DSM-III R,[5] respectively. Treatment included hospitalisation of six to eight weeks and psychotherapy according to accepted psychiatric practice as well as goal oriented weight programmes. Thirteen patients had a history of primary anorexia nervosa, of whom, however, none had fulfilled diagnostic criteria for that disease during the preceding three years. None of the patients was on any psychotropic medication or on drugs, which could have affected autonomous nervous system or smooth muscle function. Within the first week after admission to the hospital, oesophageal motor activity could be recorded manometrically in 26 patients. Subsequently, gastric emptying and antral motor activity were studied in all patients except of eight, in whom disordered oesophageal motor function was revealed at manometry. Gastric motility data of the patients were compared with those of a group of 24 healthy female volunteers studied earlier.[13] The volunteers ranged in age from 20 to 39 years ($\bar{x}=$ 24·8 yr (0·9) SEM) and in their percentage of desirable weight from 82 to 120% ($\bar{x}=96·2\%$ (2·2) SEM). None of the healthy control subjects took any drugs at the time of investigation, except for oral contraceptives in the case of eight volunteers. All studies were carried out between 0800 and 1100 h. The subjects were instructed to have their usual meal on the evening preceding the studies but to refrain from eating after 2200 h and to eat and drink nothing before coming to the laboratory on the study day. Informed consent was obtained from all patients and control subjects. The investigation was approved by the Institutional Committee on Studies Involving Human Beings.

MEASUREMENT OF OESOPHAGEAL MOTOR ACTIVITY

Oesophageal pressures were recorded by means of a Konigsberg model 31 probe (Konigsberg Instruments Inc, Pasadena, CA) fitted with three strain gauge force transducers spaced at 5 cm intervals and oriented radially 120° apart. The signals were processed by Beckman 9853A couplers and recorded by a R-411 Dynograph (Sensormedics, Anaheim, CA). The probe was passed into the stomach through the nose and slowly withdrawn at 5 mm steps and one minute intervals. During each one minute period, the patients were requested to take one wet swallow. The resting pressure within the lower oesophageal sphincter and the pressure level obtained during swallow induced relaxation were measured, in millimeters of mercury (mmHg), using end-expiratory pressure with the mean fundic pressure as the zero reference. Lower oesophageal sphincter resting pressures were expressed as the mean value of the recordings from each of the three transducers as obtained before a swallow from the zone of maximal pressure. For each swallow induced sphincter relaxation, the difference between the resting pressure and the pressure level during relaxation was calculated and expressed as percent relaxa-

tion from resting pressure. At each transducer site, at least three sphincter relaxations upon swallowing were recorded. Amplitude of contractions within the oesophageal body was measured from the oesophageal base line pressure to the peak of the complex. Duration of contractions was measured, in seconds (s), from the intersection of lines for mean resting intra-oesophageal pressure and the upstroke of the complex to its return to mean resting pressure. Propagation velocity was measured as the time elapsing from the onset of a swallow contraction at the oral transducer to the onset of contraction at the intermediate and from the intermediate to the aboral transducer. Mean values were determined from at least 10 swallow induced contractions in the lower, middle, and upper third of the oesophageal body, respectively. The resting pressure of the upper oesophageal sphincter as well as its relaxation upon swallowing were determined from at least three swallows at each transducer site and expressed as mean values. All recordings were scored independently and blindly by two investigators. The initiation of swallowing was recorded by a surface electromyogram of the submental muscles. Two Beckman biopotential electrodes were attached 1 cm from the right and left mandibular bow, respectively. The signal was integrated by a Beckman 9852A coupler.

MEASUREMENT OF GASTRIC EMPTYING AND ANTRAL CONTRACTILE ACTIVITY

Gastric emptying and antral motor activity were recorded by means of an isotope technique.[12][13][20] A semisolid test meal labelled with a dose of 74 MBq (2 mCi) 99m-Technetium sulphur colloid diluted in isotonic saline (0·15 M) was used. The meal was prepared from 250 ml whole milk (8·75 g protein, 8·75 g fat, 12·5 g carbohydrates), 15 g sugar, 14 g maize starch (Maizena[R], Knorr, Wels, Austria; 11·9 g carbohydrates) and, for flavouring, cinnamon. It had a caloric content of 1150 kJ. The meal was cooked slowly under continuous stirring until a homogenous, rather stiff, semisolid consistency was reached. After cooling to a temperature at which it could be ingested, it was mixed thoroughly with the radioisotope. Thirty minutes after drug administration, the subjects ate the test meal with a spoon (six patients with bulimia) or sucked it through a wide lumen polyethylene tube (18 patients and all controls). During the latter procedure, no air swallowing was noted and belching after meal ingestion was very rare in both patients and control subjects. Recording was started with the beginning of meal ingestion. A dual-headed gamma scintillation camera (ROTA-CAMERA, Siemens AG, Erlangen, West Germany) coupled to a computer system (Digital Equipments Corporation, System GAMMA-11) with one camera in an anterior and the other in a posterior position recorded the radioactivity over the stomach and over the remaining abdomen for 50 minutes. In minutes seven to 10, 27 to 30 and 47 to 50 after the start of recording, data were acquired in frame mode with 80 serial images over three seconds each and in the remaining time with serial images of one

minute frame time. To avoid possible overprojection of the stomach and the small intestine, the patients were seated in an armchair tilted at an angle of 30 degree backwards.

QUANTIFICATION OF GASTRIC EMPTYING

The one-minute frames were used for the generation of gastric emptying curves. Background activity was calculated and subtracted from the counts of the anterior and posterior camera, respectively. Thereafter, the arithmetic mean of the counts obtained from the two cameras was calculated, so that a falling count rate was representative of the marker remaining in the stomach. By using a dual-headed camera, artefacts caused by movement and absorption, as occurring in measurements with one camera only, could be avoided. The percent emptying rates for the first 10 minutes and for the entire 50 minutes after the start of meal ingestion were calculated from the emptying curves. In addition, the half emptying time (t½) was calculated from the regression line of the count rate plotted on a logarithmic scale against time on a linear scale.[21]

QUANTIFICATION OF ANTRAL MOTOR ACTIVITY

The serial images with three seconds frame time recorded in the three periods seven to 10, 27–30 and 47–50 min after the start of meal ingestion were used for measuring antral motor activity. The stomach was displayed on an oscilloscope and three small regions of interest were positioned at right angles to the axis of the antrum. To control for movements of the subject or her stomach, these regions were adjusted for every period of measurement. By using a dual headed camera, again, artefacts arising from variations in depth of radioactivity could be avoided. The amplitude of antral contractions was calculated from the more or less sinusoidal variations of radioactivity in each of the regions positioned across the antrum. These curves were high pass filtered by transforming them into the Fourier space, applying a filter with a lower frequency cut-off at 1·5 Hz, and back transformation. The algorithm of the fast Fourier transform was used for these computations. The modulation depth of the oscillations of the curve was calculated as a measure of the amplitude of antral contractions

and expressed as mean modulation depth over each recording period.[13 20] After this procedure, the curve was low pass filtered with an upper cut-off frequency of 5·5 Hz using the method described above. Then the autocorrelation function of the curve was computed and used to determine the frequency of antral contractions. Based on this frequency, a 'gated' study[22] was generated by adding all images of the three second frames belonging to the same state of contraction of the antral wall. An amplitude as well as a phase image were obtained by applying a Fourier analysis of the first component – that is, by fitting only the first sinusoid of the Fourier expansion to the time activity curve for each pixel of the gated study. Since in the phase image regions of equal phase appear in the same colour, the distances between points of equal contraction state could be calculated. These distances were used, together with the contraction period, to compute the propagation velocity of antral contractions.[13]

STATISTICAL ANALYSIS

The percentage of marker remaining in the stomach and the data on antral motor activity were subjected to analyses of variance for repeated measures[23] accounting for the fixed factors 'group' (patients, controls) and 'time' (periods of measurement), and of the random factor 'subjects'. To evaluate differences between mean values in each period of time, a sequentially rejective multiple test procedure was used.[24] In this procedure, which was based on the analysis of variance, directional tests were carried out and an overall significance level of $\alpha = 0.05$ was adopted. The t½ values were analysed by a nonparametric test – that is, the Mann-Whitney U-test for differences between independent samples.[25]

Results

OESOPHAGEAL MOTOR ACTIVITY

Oesophageal manometry revealed that 18 of the 26 patients studied had an entirely normal oesophageal motor activity. In four patients, the lower oesophageal sphincter relaxed incompletely upon swallowing (64%, 47%, 46% and 33%, respectively), the swallow induced contractions in their oesophageal body, however, were propulsive. Two of these patients with lower oesophageal sphincter 'hypochalasia' had high amplitude (>200 mmHg), long duration (>8 s) contractions in the lower third of their oesophageal body. None of the four patients showed an increased incidence of nonpropulsive, tertiary contractions or of nonpropulsive reactions to swallows, and none of them had presented any symptoms prior to the manometric investigation, which could have been indicative for a disordered oesophageal motor function (Table I).

In four patients, distinct disorders of oesophageal motility were found to be present in manometric and fluoroscopic studies: one had an achalasia of the lower oesophageal sphincter and non-propulsive contractions of low amplitude in

TABLE I Gastrointestinal symptoms in bulimia patients with and without demonstrated oesophageal motor disorders

n	Achalasia 1	Vigorous achalasia 2	DOS 1	'Hypochalasia' 4	Normal motor activity 18
Dysphagia for solids	1	2	–	–	–
Dysphagia for solids and liquids	–	–	1	–	–
Vomiting spontaneous non-acidic	1	2	1	–	–
Vomiting selfinduced non-acidic	1	2	1	–	–
Vomiting selfinduced acidic	1	2	1	4	18
Excessive saliva regurgitation	1	1	–	–	–
Chest pain	1	1	1	–	–
Heart burn	–	–	–	–	1
Postprandial fullness and bloating	1	1	1	4	14
Epigastric pain	1	–	–	2	8
Belching	–	–	1	3	11
Constipation	1	2	1	2	13

DOS, diffuse oesophageal spasm; 'hypochalasia', incomplete relaxation of lower oesophageal sphincter but propulsive swallow induced contractions in the oesophageal body.
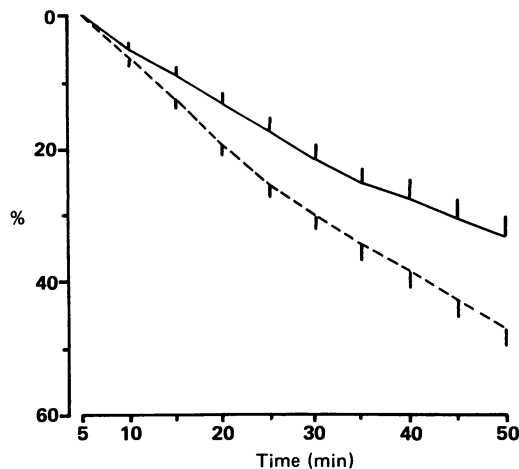
Figure 1: Mean percent rate of gastric emptying (SEM) of the test meal in the 24 patients with bulimia nervosa (——) and in the 24 healthy control subjects (————).
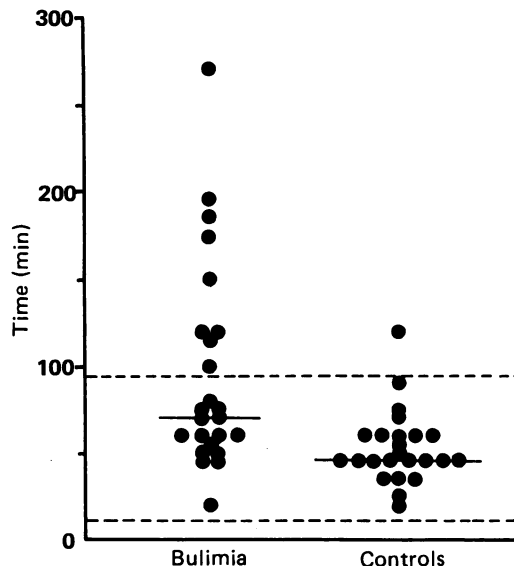

Figure 2: Gastric half-emptying time, in minutes, of the isotopically labelled test meal in the 24 patients with bulimia nervosa and in the 24 healthy control subjects. ——: median times. Area between ————: mean half emptying time ±2 standard deviations in the 24 health control subjects.

the oesophageal body, two had a vigorous achalasia and non-propulsive, repetitive contractions in the lower third of the oesophageal body, and the fourth patient had diffuse oesophageal spasm as defined by the manometric criteria proposed by Richter and Castell.[26] Subsequent fluoroscopic studies yielded results compatible with the manometric diagnoses. At investigation before manometry, all of these four patients had indicated to experience two different types of vomiting: one selfinduced, in which the vomited material tasted either acidic or non-acidic and like the preceding meal, and one involuntary, in which the vomited material always was nonacidic and of the same taste as the preceding meal (Table I). The patients with achalasia and vigorous achalasia also had dysphagia for solid food and two of them reported chest pain and a series of events, in which food stuck behind the sternum and could not be swallowed down. The patient with diffuse spasm had dysphagia for solid and liquid food as well as chest pain. The second, involuntary type of vomiting disappeared after mechanic dilatation in the patient with achalasia, and under nifedipine (Adalat-retard[R], Bayer, Leverkusen, West Germany) medication in the three patients with vigorous achalasia and diffuse spasm, respectively. These therapeutic measures, however, had no influence on the bulimic behaviour of the patients, who all continued their binge eating and selfinduced vomiting. One of the patients was concerned before the initiation of nifedipine medication that this drug could interfere with her ability to vomit selfinduced. The presence or absence of disordered oesophageal motor activity was in no discernible relationship with the frequency of

self-induced vomiting and the duration of the history of bulimia.

GASTRIC EMPTYING

Percentage of marker remaining in the stomach
The emptying curves over time followed closely a mono-exponential pattern. No initial lag phase as reported to occur after the ingestion of solid meals was observed. In the patients with bulimia, gastric emptying was significantly slower than in the 24 healthy control subjects (Fig 1). This difference was not present in the first 10 minutes after the start of meal ingestion (Table II).

Half emptying time
Gastric half emptying time was grossly prolonged – that is, longer than the mean t½ of the control subjects plus two standard deviations (SD) in nine patients. The respective t½s of these patients ranged from 99 to 272 minutes (Fig 2). As a group, the patients had significantly longer t½s (median: 74 minutes) than the control subjects (median: 47 minutes; Mann-Whitney U Test: $p<0.001$), and only one patient had a t½ shorter than the median t½ of the control subjects. Long t½s were associated with low serum potassium levels (r (23)=0.427, $p<0.05$). Long t½s tended also to be associated with a low percentage of desirable body weight and with long illness duration, statistical significance, however, was not reached. There were no relationships between the length of t½ and the frequency of vomiting per week, the abuse of laxatives and the presence of a history of primary anorexia nervosa, respectively.

ANTRAL MOTOR ACTIVITY
In all patients with bulimia as well as in all of the healthy control subjects, phasic antral contrac-

TABLE II Emptying rate in the first 10 minutes and during the 50 minutes after the start of meal ingestion in healthy control subjects and in patients with bulimia nervosa

		Emptying rate (%/min)	
	n	First 10 min	Entire 50 min
Control subjects	24	0·61 (0·10)	0·98 (0·05)
Bulimia patients	24	0·51 (0·08)	0·67 (0·06)*

Values are means (SEM). *Significantly different (t-test for paired observations) from values of control group.

TABLE III Antral contractile activity in healthy control subjects and in patients with bulimia nervosa. Amplitude, frequency and propagation velocity of contractions at seven to 10, 27–30 and 47–50 minutes after the start of meal ingestion

	n	7–10 min	27–30 min	47–50 min
Amplitude (modulation depth in %)				
Control subjects	24	24·2 (1·9)	26·5 (1·7)	29·7 (2·2)
Bulimia patients	19	20·8 (1·7)	20·4 (1·6)*	21·2 (1·7)*
Frequency (cycles/min)				
Control subjects	24	3·08 (0·05)	3·03 (0·03)	2·95 (0·03)
Bulimia patients	19	3·06 (0·06)	3·01 (0·05)	3·01 (0·06)
Propagation velocity (mm/s)				
Control subjects	24	3·02 (0·29)	2·75 (0·23)	2·74 (0·21)
Bulimia patients	19	3·04 (0·26)	3·04 (0·22)	3·17 (0·24)

Values are means (SEM) measured in the three regions of interest positioned across the antrum.
*Significantly different (t-test for treatment means) from values of control group.

tions were present all the time and no 'silent' phases were observed in any individual.

Amplitude of antral contractions

The contraction amplitudes were lower in the patients with bulimia than in the 24 healthy control subjects during all of the three recording periods – that is, seven to 10 minutes, 27–30 minutes and 47–50 minutes after the start of meal ingestion (Table III). In the healthy subjects, contraction amplitudes increased steadily over the postprandial 50 minutes of recording. In the bulimia patients no or only slight increases occurred and the contraction amplitudes remained more or less unchanged over the 50 minutes of recording. In the periods 27–30 minutes and 47–50 minutes after the start of meal ingestion, amplitudes were significantly lower in bulimia patients than in the healthy individuals (Table III).

Frequency of antral contractions

In the patients with bulimia having received no drug or placebo, the frequency decreased slightly less over the 50 minutes after meal ingestion than in the healthy control subjects (Table III), in whom this decrease correlated with the simultaneous increase in contraction amplitude. The mean contraction frequency averaged over the three postprandial recording periods was within the range of the mean frequencies of the control subjects in all of the patients.

Propagation velocity of antral contractions

The propagation velocity was slightly higher in the bulimia patients than in the healthy subjects (Table III). No retropulsive waves could be detected in any patient with bulimia and in any control subject.

Discussion

The results of the present study show that symptoms of oesophageal motor disorders can be obscured by coexisting bulimic behaviour. Other than reported to have occurred in patients categorised on primary evaluation as having primary anorexia nervosa however,[10 12] such symptoms in no instance gave rise to a misdiagnosis of bulimia nervosa. That the symptoms of oesophageal motor disorders were overlooked or

misinterpreted by the patients' physicians and psychiatrists has to be attributed not so much to an inadequate history taking, but to the fact that the symptoms were misinterpreted by the patients themselves. Directed questioning, however, will yield information indicative of the presence or absense of a swallowing disturbance. This was also the case in the present study in which, at the investigation before manometry, all of the four patients, in whom subsequently the presence of achalasia, vigorous achalasia and diffuse oesophageal spasm, respectively, was documented, reported to experience two different types of vomiting: one selfinduced, in which the vomited material tasted either acidic or non-acidic and like the preceding meal, and the other involuntary, in which the vomited material was always non-acidic and of the same taste as the preceding meal. Although the involuntary emesis occurred also in situations in which the patients not at all intended to get rid of the ingested food, all of them thought that this type of vomiting and the symptoms of dysphagia and retrosternal pain, as well, were consequences of their selfinduced vomiting. This seemed the more plausible to them, as all had had no such symptoms before the onset of their bulimic behaviour. There is no reason to assume, however, that the oesophageal motor disorders detected could have been induced or their onset facilitated by the vomiting behaviour. Self-induced emesis, however, may cause oesophageal perforation, which, with its frequently ensuing mediastinitis, is a life threatening event. In patients with primary anorexia nervosa, incidences of mediastinal emphysema as a consequence of selfinduced vomiting have been described.[27 28] In one paper[29] it has been reported that a patient with bulimia and chronic alcohol abuse suffered from 'mechanical dysphagia' – that is, 'solids or large bolus-sized foods would get 'stuck' at an area under the xiphisternum'. Endoscopically, 'an area 4 cm above the gastro-esophageal junction' was found that 'contracted (and relaxed) like a second sphincter'. On manometry, however, no high pressure zone above the lower oesophageal sphincter was seen and the resting pressure of the sphincter was low and acid infused into the mid-oesophagus resulted in 'lower retrosternal pain'. A fluoroscopic investigation was not performed. Thus, the description yields no information as to the nature or even the mere presence of the presumed 'mechanical dysphagia'. The high incidence of oesophageal motor disorders in our group of patients with bulimia nervosa in relation to the incidence to be expected in the general population certainly cannot be extrapolated to other patient groups. There seems also to be no reason to assume an underlying disorder accounting for both bulimia and oesophageal contraction abnormalities, although one group of workers has reported an association between contraction abnormalities and criteria defined psychiatric illness in diabetic[30] and non-diabetic patients.[31] The results of the present study show that the clinical evaluation of patients with bulimia nervosa should always include the taking of a thorough history regarding swallowing and vomiting and the nature as well as the taste

of the vomited materials. In cases in whom symptoms are suggestive of an oesophageal motor disorder, radiographic and manometric studies should be performed. Gastric emptying of the semisolid test meal was grossly delayed in more than one third of the studied patients, and antral contraction amplitudes were markedly lower than in the healthy control subjects and the normally occurring postprandial increase in contraction amplitude was not present. This shows that gastric motor function is impaired not only in a high proportion of patients with primary anorexia nervosa as reported previously,[8-17] but also in many patients with bulimia. Our results confirm those of a recent study,[18] in which gastric emptying of cream of wheat was found to be delayed in 12 of 20 bulimia patients. The observation made in that study that the remaining eight patients, by contrast, had rapid emptying, does, however, not correspond with the results of the present investigation, in which only one patient had a shorter t½ than the median t½ of the healthy controls. Other authors found gastric emptying of a solid meal to be delayed in only one of 10 patients with bulimia[17] or to be normal.[16] In the latter study,[16] however, emptying data of the bulimic patients were compared with those of a very heterogenous control group of 16 premenopausal women, which included patients with morbid obesity and inflammatory bowel disease and whose t½s for solid meal components were highly scattered. In the same study it was reported that, in three patients with bulimia, antral motor activity as recorded manometrically was normal and even higher than in the controls.[16] The latter findings have to be viewed with caution regarding the very small number of patients studied and the recording technique used, which is prone to detect no more than 50% of actually occurring contractions.[32] As there is no information available on the rate of gastric emptying in patients hospitalised for psychiatric diseases without eating disorders, the question of how emptying in such patients relates to that in patients hospitalised for bulimia cannot be answered.

The mechanisms underlying the impaired gastric motor function in patients with bulimia are unknown. As revealed in the present study, the absence or presence of a history of primary anorexia nervosa, the absence or presence of laxative abuse and the frequency of vomiting per week bore no relationship to the rate of gastric emptying. There was, however, a significant inverse relationship between t½s and serum potassium concentrations, which were below the normal range in four of the 24 patients studied and near the lower borderline of normal in another eight. There was also a tendency of low percentages of desirable weight being associated with long t½s, which, however, did not attain statistical significance. Incidences of dilatation, infarction and rupture of the stomach upon the ingestion of large quantities of food, which were observed not only in depleted patients with primary anorexia nervosa[33-37] but also in patients with bulimia,[38 39] suggest that weakness and atrophy of gastric smooth muscle, caused by malnutrition and/or electrolyte depletion resulting from the repetitive self-induced vomiting,

might play a crucial role. This also could have been the case in a number of our patients, who were substantially underweight by up to 23%, although the average weight of the patients was only about 10% below their calculated desirable weight. Disordered gastric motor function may also be the result of abnormal autonomic function[15] or of peripheral neurophathy as found to impair skeletal muscle function in patients with primary anorexia nervosa and bulimia.[40 41] There was no evidence of such changes, however, in any of our patients.

It is concluded that bulimic behaviour can obscure symptoms of oesophageal motor disorders and that delayed gastric emptying, low antral contraction amplitudes and an impaired contractile response of the antrum to the ingestion of a meal are frequent not only in primary anorexia nervosa but also in bulimia nervosa.

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