

VARIATIONS IN ABDOMINAL ELECTRICAL POTENTIALS IN BULIMIC AND  
NORMAL HUMAN SUBJECTS

by

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## ABSTRACT

The research on the gastrointestinal function of individuals with eating disorders has tended to focus on the gastric emptying of individuals with anorexia nervosa. As a result, little is known about the gastric activity of bulimics. This study extended the research by monitoring the gastric activity of bulimics using electrogastrography (EGG), the recording of electrical activity from cutaneous electrodes placed on the abdomen.

The gastric potentials were recorded from 13 female bulimics and 13 controls after an overnight fast. Recordings were conducted for 30 minutes prior to and 90 minutes immediately following the consumption of a liquid meal.

Spectral analysis (FFT) of selected segments of the EGG data revealed no overall differences between the bulimics and the control group. However, it did indicate significant differences in postprandial activity between the two groups. Specifically, while the control group was characterized by increased power in the 2 cpm to 6 cpm frequency range approximately 12 minutes into the postprandial phase, a similar increase in activity was not found in the bulimics. These results suggest that electrical control activity (ECA) and/or electrical response activity (ERA) in bulimics may be disordered, and as such may contribute to the gastrointestinal complications experienced by many bulimics.

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**PART A**  
**INTRODUCTION**

Bulimia is an eating disorder characterized by a pattern of episodic binge eating (Pyle, Mitchell & Eckert, 1981; Russell, 1979). Yet, bingeing is not unique to bulimia, as it occurs through a continuum of weight disorders including both anorexia nervosa and obesity (Wardle & Beinart, 1981). In fact, binge eating occurs in many people, whatever their weight, who are attempting to exercise dietary restraint.

While it is frequently difficult to make sharp distinctions between the normal spectrum of eating behavior and 'true' eating disorders (Garrow et al., 1976), it is apparent that the feeding and weight regulatory mechanisms inherent in normal eating patterns are altered when abnormal eating habits are adopted. These, in turn, may not only jeopardize a person's physical and/or psychological health (Harris, 1983), they may also lead to 'disease and disability' (Garrow et al., 1976). For example, as bulimia involves the "uncontrollable rapid ingestion of large amounts of food over a short period of time" (Gandour, 1984, p. 3), it is not surprising that a number of complications related to gastrointestinal function have been noted. Specifically, gastric dilatation (Mitchell, Pyle, & Miner, 1982) and gastric rupture (Evans, 1968; Matikainen, 1979), both of which can be fatal, have been reported. Furthermore, since some form of purging behavior (typically vomiting or laxative abuse) frequently follows the binge episode, the more typical complaints of abdominal fullness or bloating, nausea and pain (Abraham & Beumont, 1982) may be related to alterations in

gastrointestinal function. In fact, research indicates that there is a relation between nausea and/or vomiting and abnormalities in gastric electrical activity in certain subgroups of patients (Geldof, van der Schee, van Blankenstein, & Grashius, 1983a, 1983b).

Since the research on the gastrointestinal function of individuals with eating disorders has tended to focus on the gastric emptying of individuals with anorexia nervosa, very little is known about the gastric activity of bulimics. Furthermore, since bingeing with or without vomiting can be an associated feature of anorexia nervosa, information on the gastric function of any particular type of eating disordered individual is limited. Accordingly, this study extended the research by monitoring the gastric activity of bulimics using electrogastrigraphy (EGG), the recording of electrical activity from cutaneous electrodes placed on the abdomen.

## The Stomach

Anatomically, the smooth muscle cells of the stomach are arranged in three layers, none of which completely envelops the entire stomach. The outer longitudinal layer is sparse or absent on the anterior or posterior surfaces, while the middle circular layer is minimal in the para-oesophageal region, and the inner oblique layer, being the least complete, is formed by two bands of muscle lying on the anterior and posterior surfaces (Johnson, 1981; Weisbrodt, 1984). The stomach is also richly innervated with both intrinsic and extrinsic nerves. The intrinsic nerve cell bodies lie in various plexuses, the most prominent being the myenteric plexus, and form a portion of the enteric nervous system. Extrinsicly, the stomach is innervated by branches of the vagus nerve and by fibres originating in the coeliac plexus of the sympathetic nervous system (Johnson, 1981; Weisbrodt, 1984).

In terms of basic control mechanisms, the smooth muscle cells in the longitudinal muscle of the greater curvature of the stomach (approximately halfway between the fundus and pylorus) have a membrane potential that fluctuates rhythmically with cyclic depolarizations and repolarizations. Known as slow waves, pacesetter potentials, the basic electrical rhythm (BER) and/or electrical control activity (ECA), these rhythmic oscillations have two components - an initial upstroke and a secondary plateau potential (Johnson, 1981). If this plateau potential is

sensitive to the generation of a second potential, electrical response activity (ERA) occurs. Consisting of one or more spikes superimposed on the decline phase of the ECA, this second potential (also known as fast activity or spike burst) is time-locked to the ECA, i.e., it always follows the BER and precedes contractile activity (Stern, Ray, & Davis, 1980).

ECA is always present in the stomach, regardless of the presence or absence of contractions. Its frequency is believed to be constant and in humans is estimated at about 3 cycles per minute (cpm) (Davenport, 1977, 1982; Johnson, 1981).

Gastric motility (the mechanical activity of the gut) is directly involved in three stomach functions: First, the gastric musculature allows the stomach to accommodate the large quantities of material ingested while eating. Second, the contractile activity emulsifies the gastric contents in preparation for intestinal digestion and adsorption. Third, gastric peristalsis propels the partially digested gastric contents, known as 'chyme' into the duodenum (the first segment of the small intestine.)

Following the ingestion of a meal, the peristaltic or contractile activity in the stomach is very weak: thereafter the waves increase in intensity and slightly in frequency, eventually returning to the resting rate (Vander, Sherman, & Luciano, 1975). Since peristaltic waves follow the ECA of the stomach, the basic frequency, velocity, and direction of gastric

peristalsis is paced by the slow waves (Mountcastle, 1980). In humans, the duration of these contractions ranges between 2 and 20 seconds, with the maximum frequency being between 3 and 5 contractions per minute (Johnson, 1981). Because of the relation between the size of the stomach and the frequency and rate of peristalsis, two or three waves are usually present at any one time (Davenport, 1977, 1982).

Immediately after the ingestion of a meal, the stomach may contain over a litre of contents. The rate at which these contents leave the stomach and empty into the small intestine is determined by the balance between the force and frequency of gastric peristalsis and the resistance to flow offered by the pylorus (Mountcastle, 1980). The most thoroughly studied determinant of this rate of gastric emptying is gastric volume, i.e., the stomach empties at a rate proportional to the volume of material in it at any given time. However, volume is not the most important factor controlling gastric emptying. In actuality, it is the chemical and physical properties of the gastric contents themselves: "These, operating through nervous and humoral pathways, regulate the rate of emptying so that delivery of chyme is adjusted to duodenal motility and emptying" (Davenport, 1977, p. 41). The physical properties of chyme affecting the rate of emptying include the fineness of division of its particles, osmotic pressure, acidity and volume. Chemically, the rate of emptying is greatly influenced by the presence of fat, i.e., fatty acids entering the duodenum tend to

inhibit gastric motility and impede the rate of emptying. Finally, all the established gastrointestinal hormones - gastrin, secretin and cholecystokinin (CCK) - are reported to slow gastric emptying (Cooke & Christensen, 1978).

Motility may also be affected by "adventitious influences such as pain, nausea and emotional disturbances" (Davenport, 1977, p. 41). Some researchers report that emotions such as depression, sadness and fear tend to decrease gastric motility, while feelings of aggression and anger often increase it (Vander, Sherman & Luciano, 1975). Such relationships are unpredictable, however, as people show different responses to apparently similar emotional states (Davenport, 1982). Furthermore, these purported rate changes are based on studies using invasive recording techniques.

During intergestive periods, a "specific integrated pattern" (Koch, 1985, p. 15) is evident in several organs of the gastrointestinal tract. Known as the migrating motility (or motor or myoelectric) complex (MMC), this pattern consists of several phases of cyclic activity. Specifically, following the series of strong peristaltic contractions which signify the end of the digestive state, a phase of motor quiescence (referred to as phase I, basal, and inactivity) begins. In humans, this period of quiescence lasts approximately 60 to 110 minutes. Gastric contractions (which are more clearly seen in the small bowel) then appear (phase II, intermediate, preburst and irregular activity) and rapidly build to a 10 to 25 minute phase

of intense peristaltic contractions (phase III, burst, activity front and regular activity). Quiescence then returns and the cycle repeats. The ingestion of food disrupts the MMC (in humans and dogs at least) through as yet unknown mechanisms (Weisbrodt, 1984). Due to the "stripping waves" (Koch, 1985, p. 16) which completely empty the stomach and bowel during phase III activity, the MMC has been called the 'housekeeper' of the gut (Code & Schlegel, 1973).

Gastrointestinal factors also play a role in the regulation of eating behavior. In fact, interest in gastrointestinal determinants of feeding behavior can be traced to the early experiments of Cannon and Washburn (1912) and Carlson (1916), where it was found that strong gastric contractions inevitably preceded sensations of hunger (i.e., abdominal sensations, often described as emptiness, pangs or growls (Stunkard & Fox, 1971)) in humans. Furthermore, it was found that these contractions appeared to become stronger and more frequent as the length of time without food increased (Carlson, 1916). While the majority of contemporary researchers no longer support the notion that hunger sensations arise from changes in stomach contractions (Davenport, 1982; Russell & Stern, 1967), others such as Stunkard and Fox (1971) still maintain that gastric motility exercises an influence on hunger, albeit a weak one. An alternate formulation is provided by Grundy and Stratcherd (1984), who speculate that the intense gastric contractions associated with the MMC probably correspond to the hunger



contractions first described by Carlson.

In addition to gastric hunger contractions, gastric distension and the gastric or duodenal secretion of hormones have been implicated as regulators of eating behavior. Research suggests that stretch receptors sensitive to changes in intragastric food volume may mediate the satiety associated with gastric distension (Lytle, 1977). With regard to the major gastrointestinal hormones involved in food digestion and absorption, only CCK has been shown to produce a specific satiety effect in animals (Smith, 1984). However, since its effects in humans are mixed (Garfinkel & Coscina, 1982), and other gut peptides such as bombesin, glucagon and somatostatin may also be involved in satiety regulation, it is clear that the "relationship between CCK and other gut hormones to hunger and satiety requires further investigation" (Garfinkel & Coscina, 1982, p. 250).

#### Recording Gastric Activity

Although first identified by Alvarez (1922) in 1921, electrogastrography has just recently come into prominence as a method for recording abdominal potentials. Known also as the EGG, this non-invasive technique involves recording gastric electrical activity from cutaneous electrodes. In contrast to the invasive techniques frequently used to record gastrointestinal function, the EGG does not disturb the

physiological process being measured (for example, through the stimulating effect of a probe placed in the stomach) or place undue psychological stress on the individual being tested.

Since 1922, a limited number of investigators have published studies on the EGG (Davis, R. C., Garafolo, & Gault, 1957; Davis, R. C., Garafolo, & Kveim, 1959) and while agreement now exists on the sinusoidal configuration of the EGG signal and on its frequency in humans - about 3 cpm - it has only been in recent years that direct evidence of the gastric origin of the signal has been provided. Specifically, while Brown, Smallwood, Duthie, and Stoddard (1975) maintained that only electrical control activity (ECA) was reflected in the EGG signal, Smout, Van Der Schee and Grashius disagreed, postulating that "in electrogastrography phasic contractile activity [i.e., ERA] is measured" (Smout et al., 1980, p. 179). In fact, when they compared the electrical signals simultaneously derived from serosal and cutaneous electrodes in conscious dogs, they found that not only was ECA activity evident in the surface records of over 91% of the 17/minute blocks of EGG data, but that this frequency was the same as that recorded from the serosal electrodes. In essence, their recording proved the gastric origin of the electrogastrogram (Smout et al., 1980).

As well as concluding that both ECA and ERA are measured in the EGG, Smout et al. (1980) also found that the amplitude of the EGG increased when ERA occurred. Consistent with this observation, researchers now maintain that when contractions are

present, the amplitude of the slow wave reflects the strength of the contraction (Koch, 1985; Koch & Stern, 1985). However, while there is typically a one-to-one relationship between slow waves and gastric contractions (Smallwood & Brown, 1983), contractions are not present all the time. As well, changes in the EGG have been observed in the absence of significant changes in motility (Koch & Stern, 1985). Such discrepancies, however, may be due to inadequate in vivo recording devices (i.e., intraluminal pressure transducers). Specifically, the lack of correlation "may reflect eccentric contractions of the antrum or contractions that failed to form or migrate over a transducer" (Koch & Stern, 1985, p. 129).

In summary, while refinements in recording electrical and mechanical activity of the stomach should continue to enhance the interpretation of the EGG, research to date has shown that this technique provides an accurate measure of the frequency of gastric electrical activity (Grashius, van der Schee, & Geldof, 1985; Holzl, 1985; Smallwood & Brown, 1983; Smout et al., 1980).

## Disturbances in Gastric Activity

Disorders of gastric motility are generally manifested by a rate of gastric emptying that is either too slow or too fast and are assumed to reflect an abnormality in one or all of the major motor functions of the stomach (Weisbrodt, 1981). For example, rapid gastric emptying is common in duodenal ulcer patients and is also a major factor in the pathogenesis of the dumping syndrome (Minami & McCallum, 1984). When gastric emptying is delayed, however, the usual symptoms are nausea, vomiting (especially postprandial vomiting), abdominal fullness, and weight loss (Minami & McCallum, 1984; Schiller, 1983). Delayed gastric emptying characterizes a wide variety of clinical situations including gastric carcinoma, gastric ulcer disease, hypothyroidism and anorexia nervosa (Minami & McCallum, 1984).

Furthermore, abnormalities in gastric electrical activity have been observed in individuals with unexplained nausea and/or vomiting (Geldof et al., 1983a, 1983b; You & Chey, 1984; You, Lee, Chey, & Menguy, 1980). Using running spectrum analysis, Geldof et al. (1983a, 1983b) identified an abnormal EGG in 11 out of 28 patients with unexplained nausea and vomiting who were normal by endoscopic examination. Unstable frequency and/or a decrease in amplitude in the postprandial phase was seen in 10 patients, two of whom had tachyarrhythmia (increased frequency of slow wave activity - 5 or more cpm - occurring at irregular intervals, followed by a compensatory pause) in the fasting

state. Similar findings were reported by You et al. (1980). Using a peroral suction electrode to record antral myoelectric activity, they observed tachygastria (increased frequency of slow wave activity - 5 or more cpm occurring at regular intervals, followed by a compensatory pause) in patients with unexplained nausea, epigastric bloating and vomiting. Unfortunately, it is not known whether gastric dysrhythmia affects gastric emptying thereby causing nausea, bloating and/or vomiting (You & Chey, 1984). As Minami and McCallum (1984) note, "further investigations are needed to delineate the electrical correlates of both gastric dysmotility and impaired function, namely gastric emptying" (p. 1602).

### Bulimia

Literally meaning 'ox hunger' or voracious appetite, bulimia is characterized by binge eating followed by various methods of purging, including vomiting and abuse of cathartic or diuretic drugs (American Psychiatric Association, 1980). Occurring in individuals exercising dietary restraint, bulimia tends to be more common in women than men (Katzman, Wolchik & Braver, 1984). The diagnostic criteria for bulimia, as described in the Diagnostic and Statistical Manual of Mental Disorders (1980) are included in Appendix A.

Although first identified as a distinct eating disturbance by Stunkard (1959) in his analysis of the eating patterns of

obese individuals, binge eating has since been described in cases of anorexia nervosa (Beumont, George, & Smart, 1976; Bruch, 1973; Casper, Eckert, Halmi, Goldberg & Davis, 1980; Garfinkel, Moldofsky & Garner, 1980) as well as in normal weight individuals (Bruch, 1973, Palmer, 1980; Boskind-Lodahl, 1976; Russell, 1979). In fact, since the symptoms of bulimia occur along a continuum of weight disorders the DSM-III criteria for bulimia are controversial. Specifically, the stipulation that the bulimic episodes not be due to anorexia nervosa is problematic as it is not at all clear that bulimia and anorexia nervosa are separate, dicotomous syndromes. Not only do some individuals fulfill both sets of criteria at different times (Abraham & Beumont, 1982; Russell, 1979), but the bulimic activity itself may have more diagnostic utility than information related to weight history (Johnson, Lewis, & Hagman, 1984). For example, Garner, Garfinkel and O'Shaughnessy (1985) report that bulimics at normal weight closely resemble anorexics with bulimic symptoms, and that these two groups can be distinguished from restricting anorexics (those who lose weight by rigidly restricting their food intake) in many ways, including age of onset, highest and lowest premorbid weight, duration of illness, histories of substance abuse, mood fluctuations, sexual activity, body distortion and family characteristics. In other words, "there may be greater heuristic as well as empirical justification for differentiating eating disorder groups in terms of the presence or absence of bulimia or specific indices of severity of illness rather than in terms

of a history of emaciation" (Garner et al., 1985, p. 586). Finally, while both anorexics and bulimics may share the "vigorous pursuit of a thin body, regardless of weight" (Garfinkel & Garner, 1982, p. 52), it is only the anorexics who are successful at reducing their food intake and body weight" (Schlesier-Stropp, 1984). Not only do bulimic individuals have a history of being overweight (Fairburn, 1984; Fairburn & Cooper, 1982; Garner et al., 1985; Halmi, Falk, & Schwartz, 1981; Russell, 1979), they frequently maintain a body weight within the normal range for their height, weight, and sex (Abraham & Beumont, 1982). Moreover, Lacey (1982) maintains that "what marks out patients in the bulimic syndrome is that the development of progressive and massive obesity which would otherwise have been their destiny, is prevented by either abstinence or laxative abuse or psychogenic vomiting, or by a combination of methods" (p. 60).

If some form of binge eating frequently occurs in individuals exercising dietary restraint, what distinguishes the clinically pathologic levels of bulimic behavior from the binge eating in otherwise normal eating patterns? Wardle & Beinart (1982) suggest that it is not dietary restriction, craving for food and/or eating binges, although the frequency may be higher in clinical populations (Wardle & Beinart, 1982). Rather, it may be the "salience" (Wardle & Beinart, 1982, p. 102) or meaning of the dieting and bingeing which provokes the personal distress associated with bulimic activity. For example, Katzman et al.

(1984) found that binge eaters (those having at least eight binge eating episodes per month, yet not meeting DSM-III criteria) in their nonclinical sample viewed their eating habits as only mildly disruptive, while the bulimics (DSM-III criteria) found their habits disruptive, with many reporting an interest in professional treatment. In fact, research indicates that bulimics are obsessed with thoughts of food, eating, ridding themselves of the ingested food and weight (Fairburn, 1980; Fairburn & Cooper, 1982; Pyle et al., 1981, Russell, 1979). No longer able to eat normally, they often oscillate between severe dieting or fasting and bingeing followed by vomiting. Moreover, when vomiting becomes established as the 'escape response' it becomes the "driving force that sustains binge eating, not visa versa" (Rosen, Leitenberg, Fondacaro, Gross, & Willmuth, 1985, p. 60). In other words, the anticipation of vomiting frees the bulimic individual from any 'normal' inhibitions against overeating (Rosen et al., 1985). Not suprisingly, depression and anxiety are common, as are intense feelings of guilt, shame and self-contempt (Schlesier-Stropp, 1984).

In summary, while bulimics often describe themselves as having strong appetites or urges to eat (Johnson & Larson, 1982; Mitchell, Hatsukami, Eckert, & Pyle, 1985; Pyle et al., 1981; Russell, 1979) and difficulty in knowing when they are full (Pyle et al., 1981; Russell, 1979), increased feelings of inadequacy and/or dyscontrol, rather than hunger, often precipitate binge eating (Johnson & Larson, 1982). Moreover, not



only does the degree of over-eating necessary to cause distress vary amongst individuals (Lacey, 1982), it is often the phenomenological experience of being out of control, rather than the amount of food eaten, than defines the binge (Gormally, 1984). It may be, as Katzman et al. (1984) suggest, that a cognitive criterion, i.e., the awareness that the eating behavior is abnormal, most clearly discriminates individuals who binge eat from those with bulimia. In other words, binge eating and self-induced vomiting are regarded as a problem only when the attitudes characteristic of bulimia are present to a "severe degree" (Fairburn & Cooper, 1983, p. 66).

While bulimia may be an example of disordered eating, it is not an unregulated activity, especially with regard to weight management. Moreover, the type of purging behavior used may influence the effectiveness of the weight control. For example, 'vomiters' tend to eat more in comparison to 'purgers' (laxative users) and weigh significantly less (Lacey & Gibson, 1985). Unfortunately, however, bulimia tends to increase in severity and intensity after vomiting commences, i.e., after the initial delight at being able to eat and not gain weight, an increase in food consumption is common (Abraham & Beumont, 1982; Johnson & Larson, 1982; Johnson et al., 1984). In fact, Johnson et al. (1984) maintain that the purging behavior eventually becomes the primary reinforcer for the loss of control inherent in binge eating. Specifically, "many bulimic patients who originally purged so that they could binge eat, began to binge eat so that

they could purge" (Johnson et al., 1984, p. 262).

While binge eating, in conjunction with purging behavior appears to resolve the the dilemma posed by the simultaneous 'wish to eat' and 'fear of fat' (Johnson & Larson, 1982), researchers disagree as to whether the binge episode relieves the dysphoria and anxiety characteristic of the pre-binge state (Abraham & Beumont, 1982) or exacerbates it (Davis, R., Freeman, & Solyom, 1985). On the other hand, it may be that the "negative mood preceding binges is qualitatively different from that which follows" (Davis, R., et al., 1985, p. 335).

Since bulimia is commonly precipitated by the intention to diet in individuals who are not only predisposed to obesity (Abraham & Beumont, 1982; Fairburn & Cooper, 1982, 1984; Garner et al., 1985), but who also appear to covet weights lower than are constitutionally comfortable for them (Cooper, Waterman, & Fairburn, 1984; Weiss & Ebert, 1983), the notions of set point and restrained eating (Polivy & Herman, 1985; Polivy, Herman, Olmsted, & Jazwinski, 1984; Wardle, 1980) have been invoked to explain the mechanisms involved in binge eating. For example, Garner et al. (1985) suggest that while bulimics may maintain a body weight within the normal range, they may actually be battling a regulated weight or set point for body weight that is considerably higher than their current weight and/or ideal preference. In other words, the "term 'normal-weight bulimic' maybe misleading, since many of these individuals have suppressed their weight below a regulated weight that is much

higher than the statistical average" (Garner et al., 1985, p. 586). Consequently restrained eating may increase susceptibility to 'loss of control' binge episodes, thereby suggesting that binge eating might represent the body's attempt to restore weight to a more "biologically appropriate level" (Polivy & Herman, 1985, p. 196). Needless to say, this 'biologically appropriate level' may not correspond to the cultural or personal aspirations of the dieter. Furthermore, research suggests that sex differences in binge eating and in labeling one's behavior as binge eating mirror sociocultural norms. For example, not only are women more likely than men to label similar patterns of eating behavior as binge eating (Katzman et al., 1984), but Leon, Carroll, Chernyk and Finn (1985) found that positive, rather than negative, affect frequently follows binge episodes in men suggesting that "gluttonous behavior can actually be seen as a sign of masculine prowess and may increase self-esteem in males" (Leon et al., 1985, p. 55).

While there are a number of medical complications associated with bulimia, most result from purgative abuse rather than from binge eating (Russell, 1979). Repeated vomiting results in sore throats (Fairburn, 1980; Pyle et al., 1981; Russell, 1979), parotoid gland enlargement (Herzog, 1982; Pyle et al., 1981; Russell, 1979) and dental hygiene problems, including cavities and enamel erosion due to acid in the vomitus (Harris, 1983; Herzog, 1982; Pyle et al., 1981). Furthermore, vomiting and

purging lead to fluid and electrolyte disturbances (Fairburn, 1980; Herzog, 1982; Stunkard, 1959), most notably, hypokalemia (potassium deficiency), the symptoms of which include muscle weakness, constipation, polydipsia (excessive ingestion of water), headaches, palpitations, abdominal pain and easy fatigability (Brotman, Rigotti, & Herzog, 1985; Harris, 1983). With regard to binge eating, typical complaints include abdominal fullness, pain, nausea, fatigue and headaches (Mitchell et al., 1982).

### Gastric Activity in Individuals with Eating Disorders

While the issue of gastric motility in anorexic individuals has been addressed in the literature (Silverstone & Russell, 1967), most of the research on the gastrointestinal function of eating disordered individuals has been concerned with gastric emptying. In fact, these studies have consistently shown delayed gastric emptying following the ingestion of a test meal in individuals with anorexia nervosa (Dubois, Gross, Ebert, & Castell, 1979; Holt, Ford, Grant, & Heading, 1981; Russell et al., 1983; Saleh & Lebwohl, 1979, 1980). Furthermore, subsequent prokinetic drug treatment, i.e., with either metoclopramide (Moldofsky, Jeuniewicz, & Garfinkel, 1977, Saleh & Lebwohl, 1980) or domperidone (Russell et al., 1983), frequently normalizes this altered rate of gastric emptying. While results such as these suggest that ECA and/or ERA in anorexics may not lie within the characteristic 3 per minute range, it must be noted

that bingeing with or without purging was frequently a presenting symptom in these studies.

Most reported cases of gastric dilatation associated with eating disorders have involved individuals in an emaciated condition undergoing refeeding during treatment for anorexia nervosa (Bossingham, 1977; Brook, 1977; Evans, 1968; Jennings & Klidjian, 1974; Keane, Fennell, & Tomkin, 1978; Russell, 1966; Saul, Dekker, & Watson, 1981; Scobie, 1972). In cases such as these, the "sudden swelling of the stomach is out of proportion to that expected from simply ingesting a large volume of food" (Harris, 1983, p. 803). Consequently, symptoms of gastric dilatation include abdominal distension and severe pain, nausea, and occasionally vomiting. In very severe cases, gastric rupture, a condition associated with an 80% mortality rate, may occur (Evans, 1968; Matikainen, 1979).

While the malnourished clinical state of these individuals has been cited as a possible reason for the development of gastric dilatation (Russell, 1966), such is not always the case. For example, Mitchell et al. (1982) report an instance of acute gastric dilatation in conjunction with bulimia. In this case, the woman was not emaciated at the time the dilatation developed. She did, however, have a previous history of anorexia nervosa. Due to the abdominal pain experienced by both anorexics on refeeding programs and bulimics following binge eating, Harris (1983) suggests that a "physician encountering a bulimarexic patient complaining of severe abdominal pain after

binge eating should not discount the symptom as just a bellyache from overeating" (p. 803).

Not only do these reports of gastric dilatation and/or rupture and the incidence of delayed gastric emptying present evidence of gastrointestinal differences between individuals with eating disorders and normal eaters, they also indicate some abnormality in gastric motility. In an attempt to clarify the relationship between one particular type of eating disorder and gastrointestinal function, the present study examined gastric activity in a bulimic population using electrogastrigraphy. Specifically, abdominal electrical potentials were recorded in bulimics and in control subjects in both fasted and fed states.

**PART B**

**METHOD**

## Subjects

The 13 female bulimic subjects were volunteer clients from the Anorexia Nervosa and Bulimia Clinic at Shaughnessy Hospital. All had been diagnosed as bulimic according to DSM-III criteria (see Appendix A). The mean age of the sample was 24.38 years ( $SD=4.86$ ). Average illness duration from the onset of bingeing was 4.78 years ( $SD=3.03$ ). Previous weight history indicated that the majority of the bulimics had been both overweight and underweight in the past. Specifically, the mean highest past weight ( $n=11$ ) was substantially greater than the Matched Population Mean Weight (119.09% of MPMW,  $SD=12.32$ ) as predicted by the 1983 Metropolitan Height and Weight Tables (Whitney & Cataldo, 1983). In contrast, the reports of lowest past weight ranged from 82.73% to 91.37% of MPMW, with the average ( $n=12$ ) being 88.08% ( $SD=4.10$ ). As well, two subjects experienced amenorrhea in excess of three months, which is indicative of a prior history of anorexia nervosa. At the time of data collection, the average weight of the sample was 101.48% of MPMW ( $SD=10.81$ ). In addition, three subjects disclosed at the time of testing that they were taking prescribed medication<sup>1</sup>.

The control group consisted of 13 female volunteers from the University population. While their mean age (26.15 years,  $SD=4.03$ ) was comparable to that of the bulimics, their average weight was lower (95.01% of MPMW,  $SD=9.36$ ). This weight difference was not significant (Table 1).



Table 1

Group Comparisons on Demographic Variables

Variable	Group		Statistic
	Bulimic (n=13)	Control (n=13)	
Age			
<u>M</u> ( <u>SD</u> )	24.38 (4.86)	26.15 (4.03)	<u>t</u> (24)=-0.92
Weight (Kgs)			
<u>M</u> ( <u>SD</u> )	137.94 (10.95)	126.68 (19.17)	<u>t</u> (24)= 0.10
Weight (%MPMW)			
<u>M</u> ( <u>SD</u> )	101.48 (10.81)	95.01 (9.36)	<u>t</u> (24)= 0.16

%MPMW refers to the Matched Population Mean Weight as predicted by the 1983 Metropolitan height and weight tables (Whitney & Cataldo, 1983). The subject's bodyweight is expressed as a percentage of matched population mean weight using the upper weight in the 'small frame' range for height.

## Apparatus

The abdominal recording took place in the psychophysiological laboratory at Simon Fraser University. Developed over the last decade, this laboratory is equipped with an electrically shielded, acoustically insulated subject chamber and a control room (Figure 1). The main amplifiers are locally designed, computer controlled, automatically resetting, D.C. amplifiers with high input impedance designed to be used in continuous data collection. The primary data store is a Nova Computer, 3/D, 96kW, with an RDOS operating system and a ten megabyte disk.

During experimentation, the electrical potentials are transmitted continuously from the subject chamber to the control room. Following amplification, the data are digitized and then both written on magnetic tape and buffered back to the control room where they are displayed, in digital form, on a display oscilloscope. Offline, selections of the data may be reviewed on an oscilloscope, plotted on an x,y plotter, or the digits displayed on a line printer. The data may also be treated by a number of analysis programs (including Fast Fourier Transform).

In this study, all signals were collected at a rate of 100 samples/second, with the cut-off frequencies of high- and low-pass digital filters (attenuating at 40 db/decade) at .2 and 50 Hz, respectively.

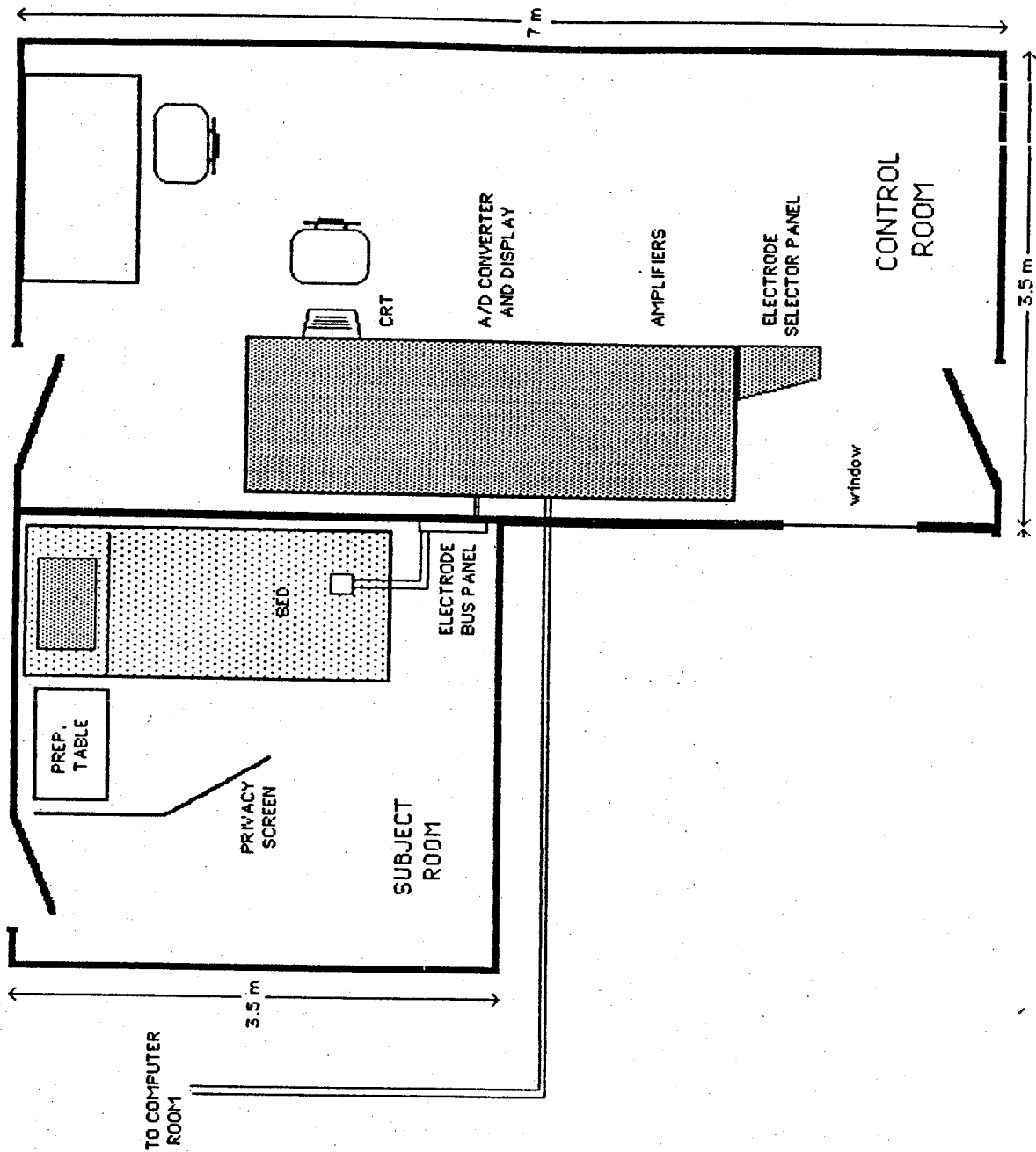


Figure 1. Laboratory layout

## Procedure

Subjects were asked to arrive at the laboratory after an overnight fast of approximately 12 hours. The experimental procedure was explained and informed consent obtained (Appendix B). Each subject then completed the Binge Scale (Hawkins & Clement, 1980), a nine-item self-report questionnaire designed to measure severity of bulimic symptoms in accordance with DSM-III criteria<sup>2</sup> (Appendix C).

While the subject was lying supine on a bed in the recording chamber, five electrodes were taped to her abdomen in the following array: Electrode 2 was centered above the navel at the intersection of the midline and epigastric line (a line joining the lowest ribs on the left and right sides.) Electrodes 1 and 3 were then placed 5 centimeters on either side of electrode 2. Finally, electrodes 4 and 5 were affixed 5 centimeters above electrodes 2 and 3, respectively (Figure 2). Two electrodes were also placed on the right and left ankles, serving as the reference and ground, respectively.

In addition to the abdominal recording, heart rate and respiration were also monitored. Specifically, EKGs were recorded from electrode pairs affixed to the skin over the sternum and right lower back, respectively. Following amplification, the EKG served as the input to a cardiometer<sup>3</sup> in the control room. Respiration was recorded via a strain-gauge respiratory transducer<sup>3</sup> attached to a belt

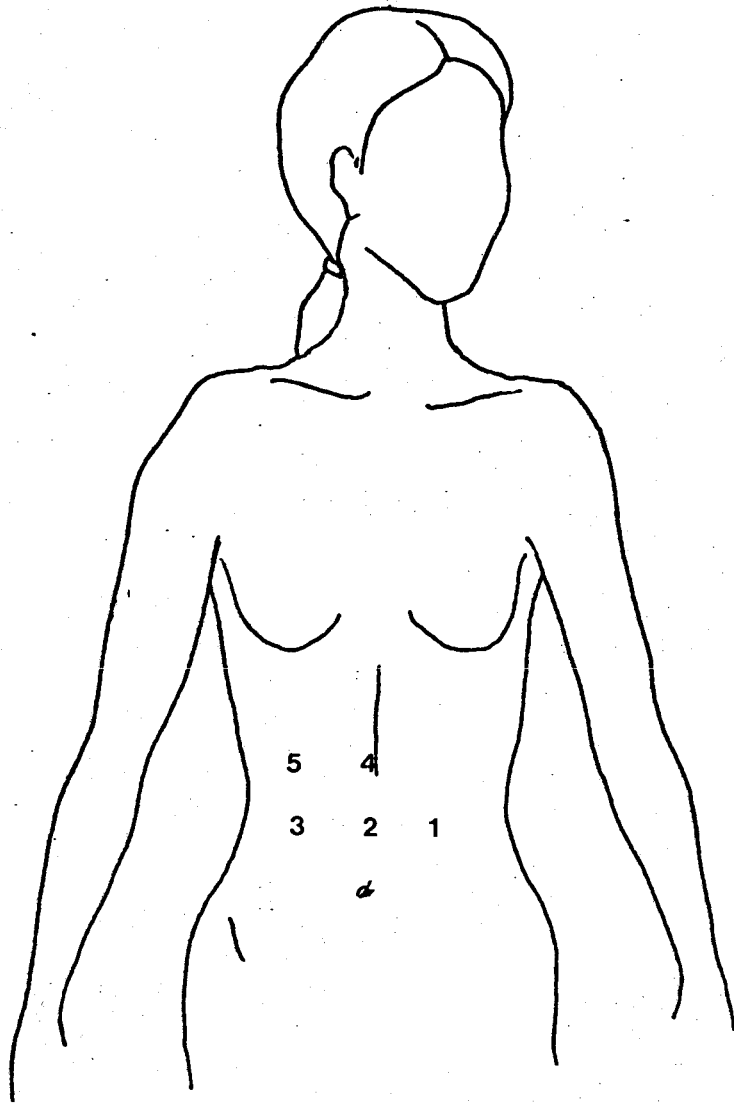


Figure 2. Electrode placement

and strapped around the thorax. Comprised of two strain gauges mounted on a piece of flexible spring steel, this device is sensitive to changes in thoracic circumference.

All direct recordings were made using Beckman Ag-AgCl electrodes filled with Beckman electrode electrolyte paste and affixed with adhesive collars. Impedances were kept to 10 KOhms, or below, in all subjects through the use of skin abrasion.

After the electrode placement had been completed, 30 minutes of fasting potentials were recorded. A liquid meal consisting of 235ml/250kcal of ENSURE (a nutritional supplement marketed by Ross Laboratories) was then served. Following the consumption of the beverage, data collection resumed for 90 minutes. Recording was then terminated and the subject debriefed and excused.

### Data Analysis

The continuous data from one stomach electrode or channel per subject record were chosen for Fourier analysis. Currently the most common method of analyzing the EGG, Fast Fourier Transform (FFT) decomposes the recorded signal into a series of trigonometric functions (sines and cosines) representing the frequency domain of the sample. Their relative amplitudes (root power) are then plotted with respect to frequency (Holzl, 1983; Koch, 1985). As spectral analysis is "adversely affected by low frequency activity or electrode drift" (Stern et al., 1980, p. 168), and computer controlled resets occurred frequently

during data collection, data segments from different electrode channels within a single record were often used for the analysis. Using different stomach channels was not considered problematic, however, as research indicates that the frequency of slow wave activity is identical across multiple electrode sites (Brown et al., 1975; Davis, C. M., McIntosh, M., & Murray, 1985; Smallwood & Brown, 1983). Finally, it was occasionally necessary to vary the starting time of selected data epochs due to amplifier resets.

Four epochs of data per subject were chosen for FFT analysis<sup>4</sup>, one approximately 13 minutes before the consumption of the meal (Pre), and three following at approximately 12 minutes (Post 1), 42 minutes (Post 2) and 72 minutes (Post 3). A 5.7 minute sample of data (1024 data points) was used for each FFT calculation<sup>5</sup>.

The frequencies lying between 2.148 and 6.834 cpm (.0358 and .1139 Hz, respectively) in each power spectrum were then chosen for further analysis. The average magnitude (root power) was then calculated for each particular frequency band (2 cpm, 3 cpm, 4 cpm, 5 cpm, 6 cpm) and these data values (five averaged values per time epoch) served as cell entries in an ANOVA with repeated measures generated by the BMDP-P2V software program. Following a visual inspection of the initial results, a logarithmic transformation was applied to the original data to correct for apparent correlations between the cell means and standard deviations. Accordingly, all reported data reflect this

transformation.

Heart rate and respiration were not analyzed at this time and as such are not included in the reported findings.



**PART C**  
**RESULTS**

An examination of the self-reports of binge eating tendencies revealed that there were significant differences overall between the bulimics and controls (Table 2). While 92% of the bulimic sample binged either weekly ( $n=6$ ) or daily ( $n=6$ ), only 8% of the control group did<sup>6</sup>. During these episodes, 38% ( $n=5$ ) of the bulimics ate until their stomachs were full, while others (31%,  $n=4$ ) experienced painfully full stomachs and some ate until they could consume no more (31%,  $n=4$ ). This contrasts with the control group who typically ate until they felt satisfied (84%,  $n=11$ ).

During the binge episode, feelings of dyscontrol were common (77% of the bulimic group reported feeling completely out of control), as were reports of self-contempt following the binge (69% ( $n=9$ ) of the bulimics reported that they hated themselves, while 23% ( $n=3$ ) felt moderately upset). The self-reports of depression following a binge episode varied, with 8% ( $n=1$ ) reporting no depression, 15% ( $n=2$ ) indicating mild depression, 31% ( $n=4$ ) experiencing moderate levels, and 46% ( $n=6$ ) being very depressed. Furthermore, 92% of the bulimic group vomited after a binge, the frequency varying from sometimes ( $n=4$ ), to usually ( $n=4$ ), or always ( $n=4$ ). In summary, while the self-reports indicated variations in both the frequency of bingeing and vomiting and in accompanying emotional distress, 84% of the bulimics ( $n=11$ ) felt that their binge eating was a major concern.

Table 2

Group Comparison on the Binge Scale

Measure	Group		Statistic	
	Bulimic	Control		
	( <u>n</u> = 13)	( <u>n</u> = 13)	<u>t</u> (24)	<u>p</u>
	<u>M</u> ( <u>SD</u> )	<u>M</u> ( <u>SD</u> )		
Binge Scale	16.53 (2.6)	2.23 (4.53)	25.16	.001

Fourier analysis of selected segments of the EGG data revealed no overall differences between the bulimics and the control group. There was, however, a significant effect with regard to time ( $p < 0.01$ ), with increased power in the 2 cpm to 6 cpm frequency range characterizing the postprandial phase (Figure 3). Moreover, there were significant differences in postprandial activity between the two groups ( $p < 0.01$ ). While the control group was characterized by increased power in the selected frequency range approximately 12 minutes into the postprandial phase, a similar increase in power was not found in the bulimics (Figure 4). In fact, it was only the first postprandial epoch which significantly differentiated between the two groups ( $p < 0.05$ , see Table 3).

Selected segments of continuous raw data which illustrate these changes in electrical activity from pre- to postprandial conditions are presented in Figures 5 and 7. Their corresponding power spectra are included in Figures 6 and 8. While a change in amplitude is evident in the EGGs of the control subject (Figure 5), a similar alteration in electrical activity is not apparent in the bulimic record (Figure 7). As well, the power spectra for this time epoch (Figure 8B) indicate little power at any particular frequency. This contrasts with the postprandial increase in power in the 3 cpm frequency range in the control subject (Figure 6B).

Figure 3. Average Magnitude of EGG Frequency Components Per Time Epoch

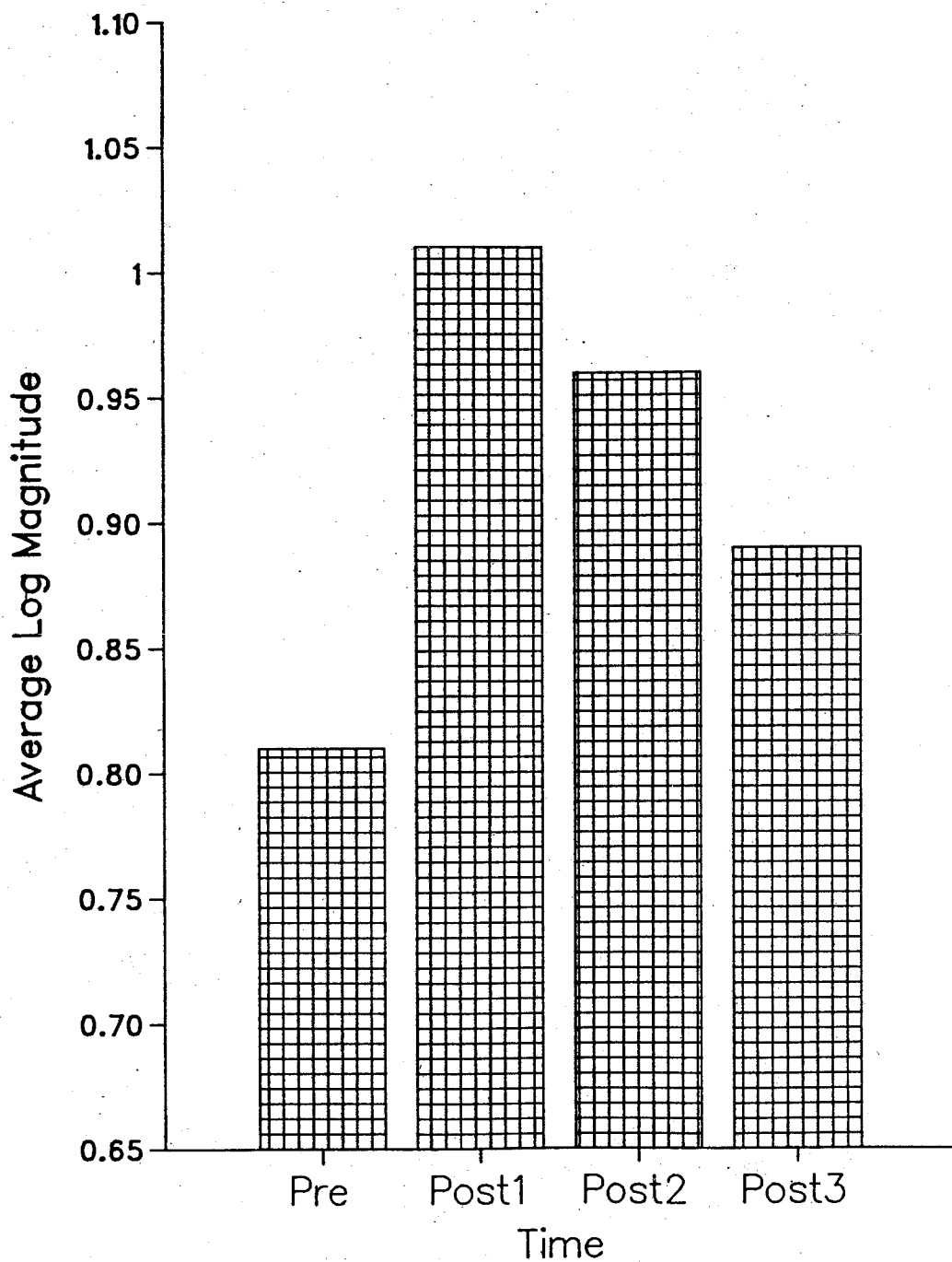


Figure 4. Average Magnitude of EGG Frequency Components For Each Group Per Time Epoch

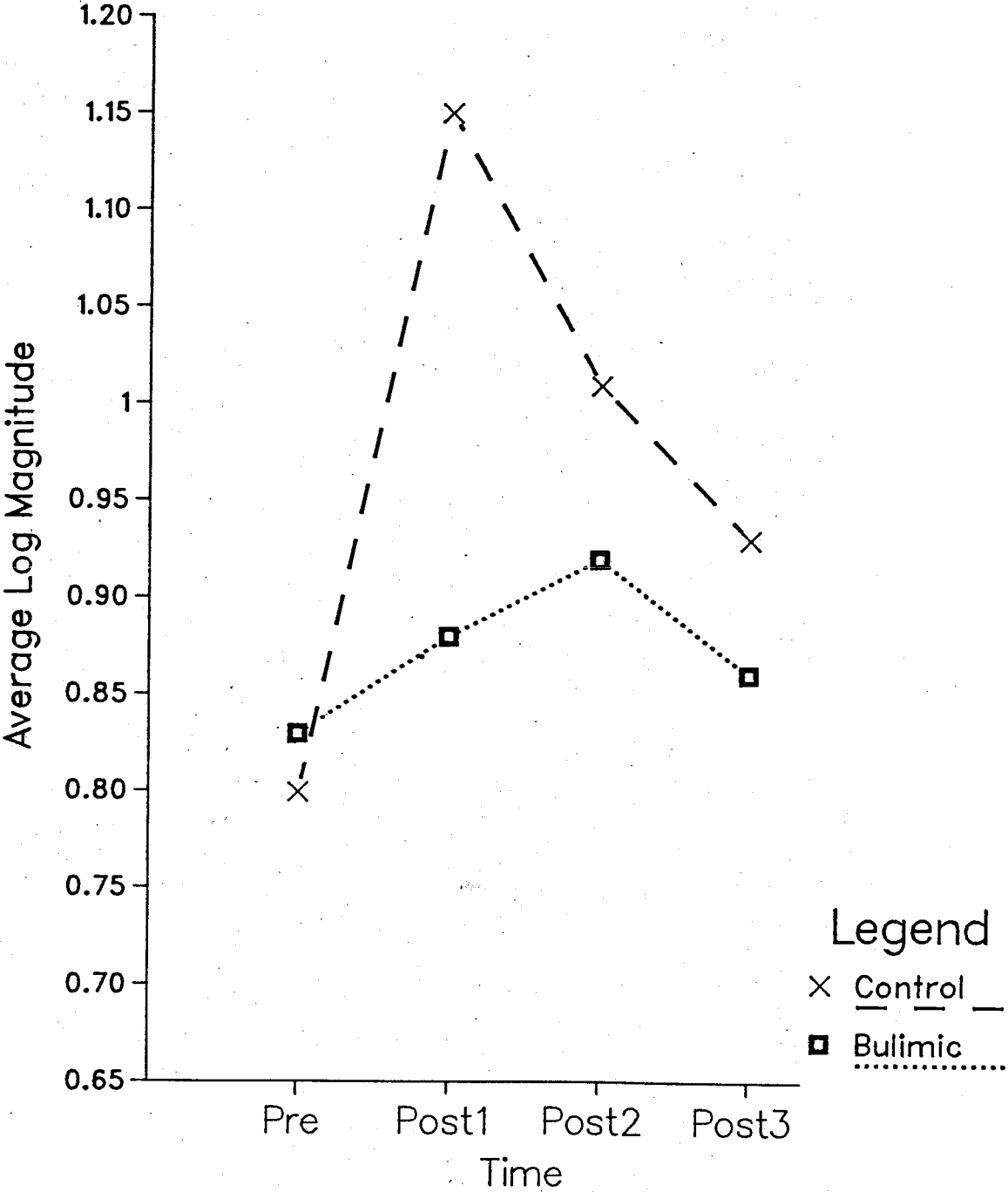


Table 3

Multiple Comparisons Between Group Means Per Time Epoch

Comparisons		Pre	Post 1	Post 2	Post 3
k=8	<u>q</u>	-0.53	4.78	1.59	1.24
<u>df</u> =65*	<u>p</u>	n.s.	<0.05	n.s.	n.s.

\*Satterthwaite correction

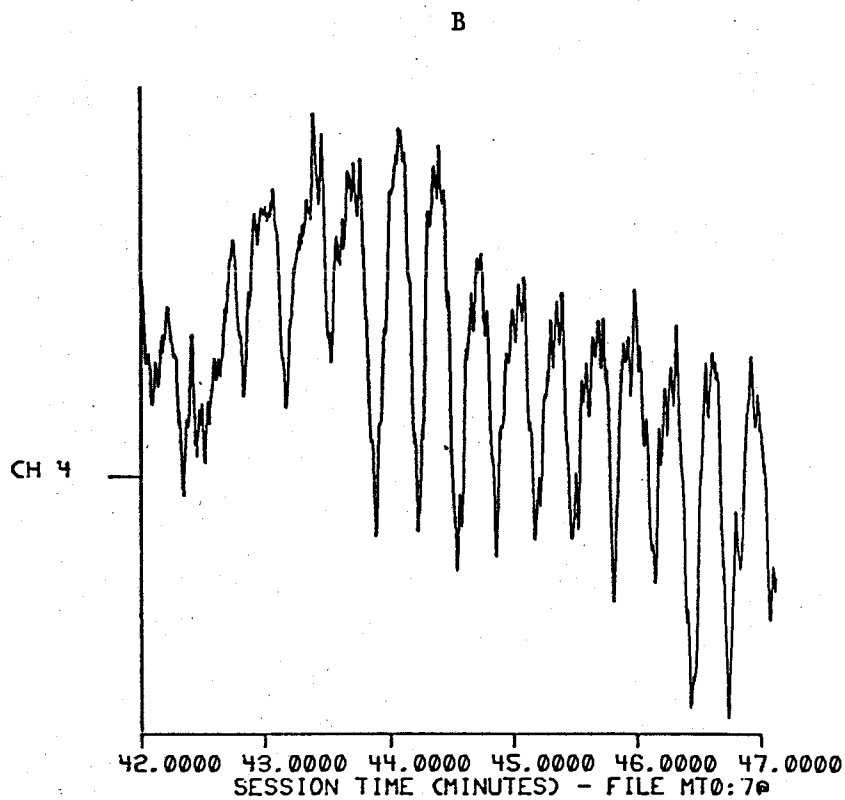
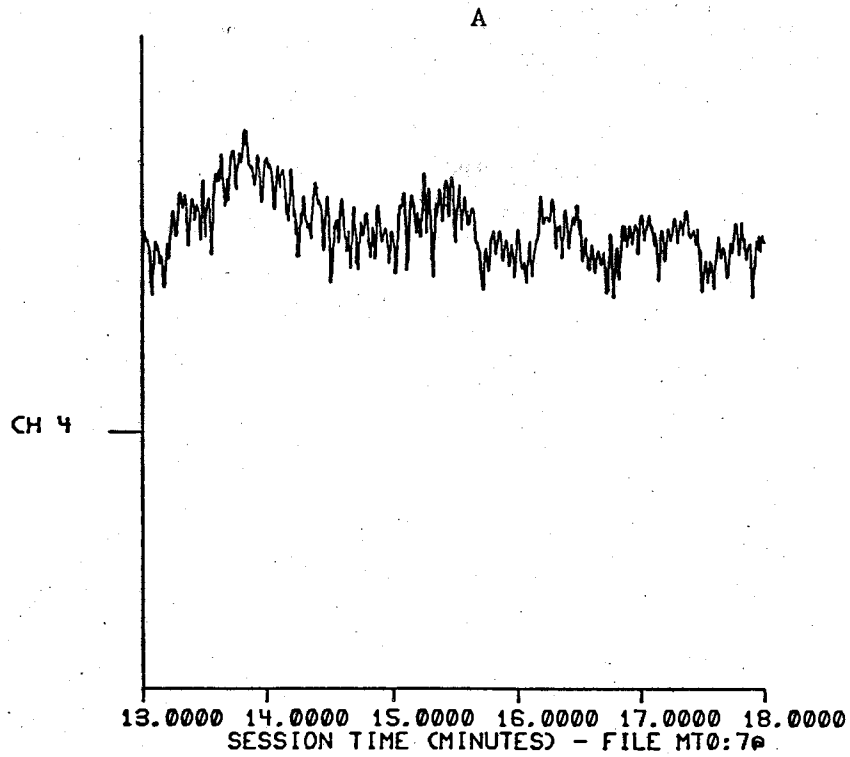
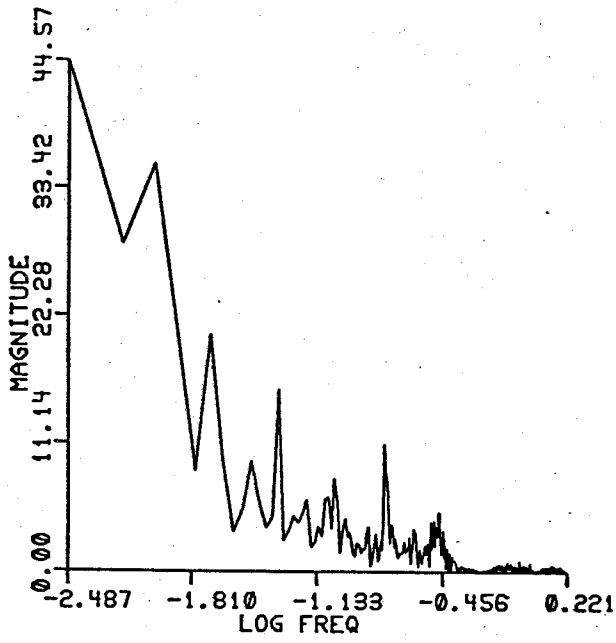


Figure 5. A. A 5.7 minute sample of data from the preprandial phase (Control subject 7)

B. A 5.7 minute sample of data from the post 1 time epoch (Control subject 7)



A



B

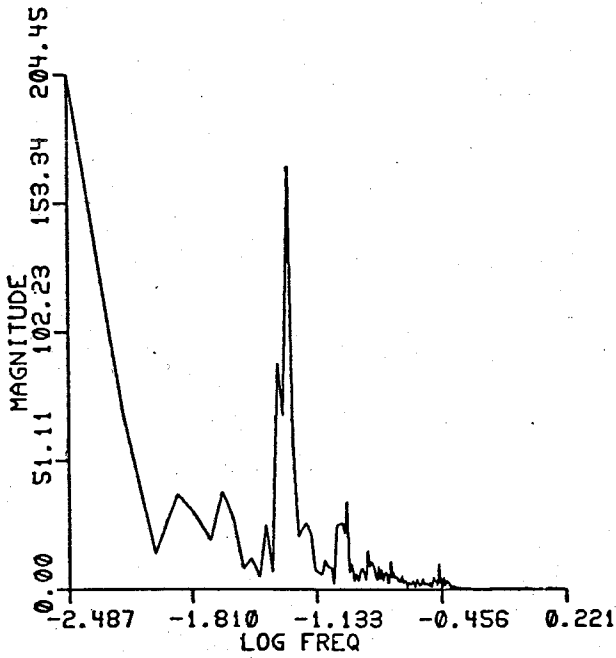


Figure 6 A. Power spectrum of the data from Figure 5A  
B. Power spectrum of the data from Figure 5B

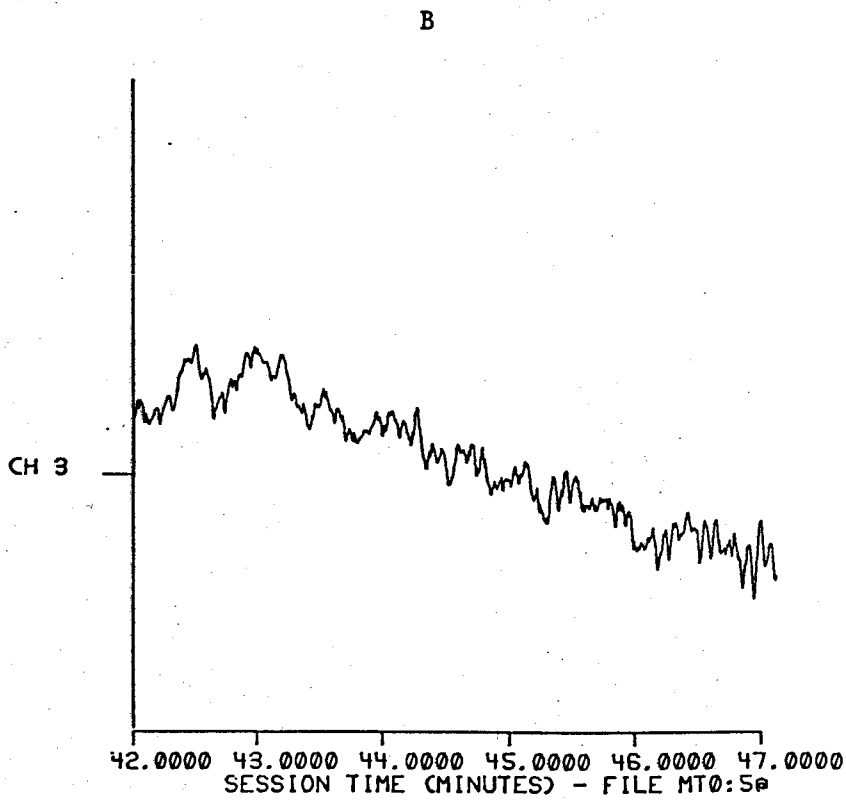
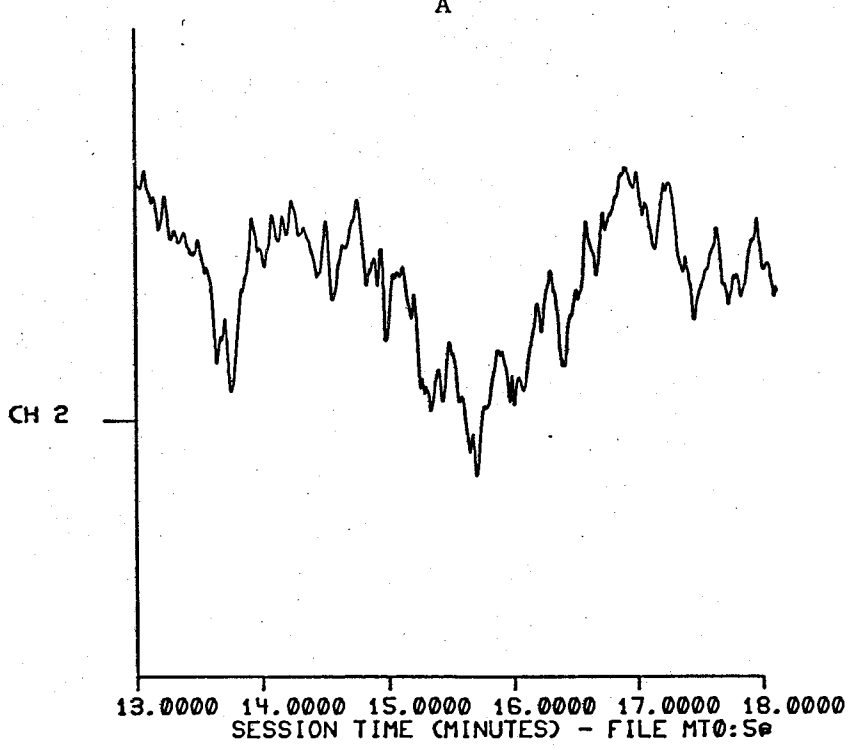
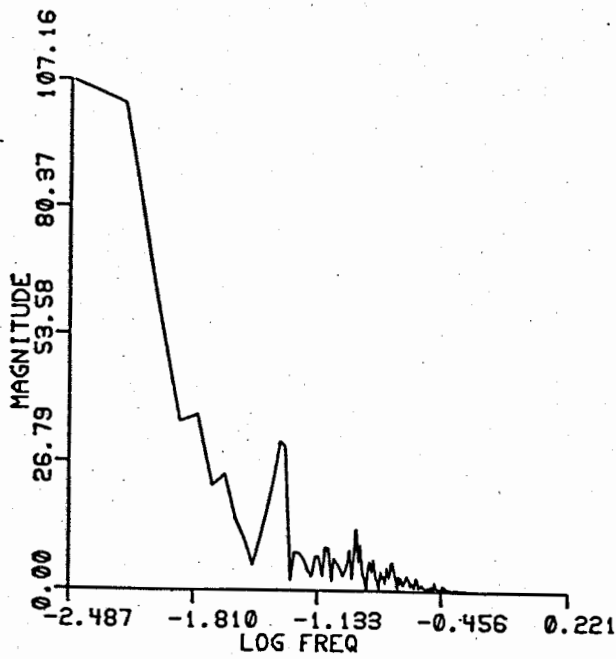


Figure 7 A. A 5.7 minute sample of data from the preprandial phase (Bulimic subject 8)

B. A 5.7 minute sample of data from the post 1 time epoch (Bulimic subject 8)



B

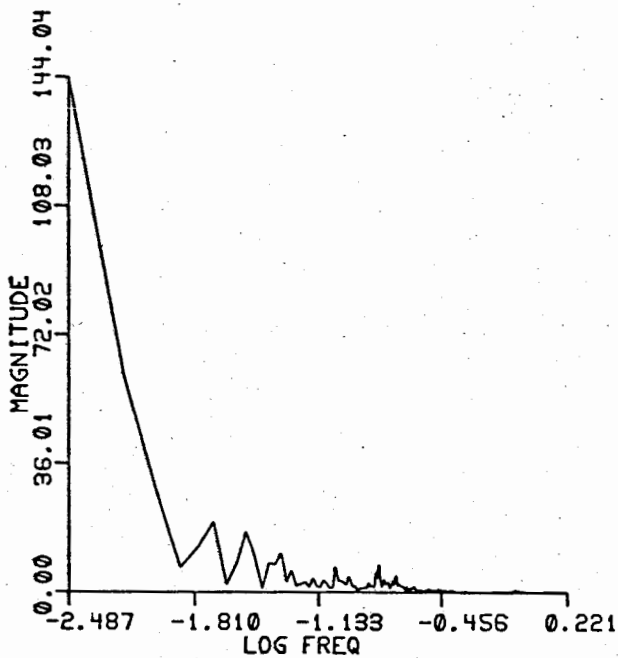


Figure 8 A. Power spectrum of the data from Figure 7A  
 B. Power spectrum of the data from Figure 7B

There were significant differences in power between frequency bands ( $p < 0.001$ ), with the most power being displayed in the ranges of 2 to 3 cpm. Frequencies in the range of 4 to 6 cpm were less powerful (Figure 9). In addition, there was a significant frequency  $\times$  group interaction ( $p < 0.05$ ), with more power being evidenced in all but one frequency band by the control group. In contrast, the magnitude of activity was essentially the same for both groups in the 5 cpm range (Figure 10). Post hoc t-tests comparing the group means at each frequency band were not significant (Table 4). Such an uninterpretable interaction could be due to a lack of statistical power, (i.e., more data are needed), or it may be that the relationships among the various frequency bands are more complex than that tapped by pairwise comparisons between the means.

In summary, while the frequency of gastric electrical activity was within the range typical for ECA-ERA, the results suggest that postprandial activity may be impaired in bulimics.

Figure 9. Average Magnitude of EGG Frequency Components Per Frequency Band

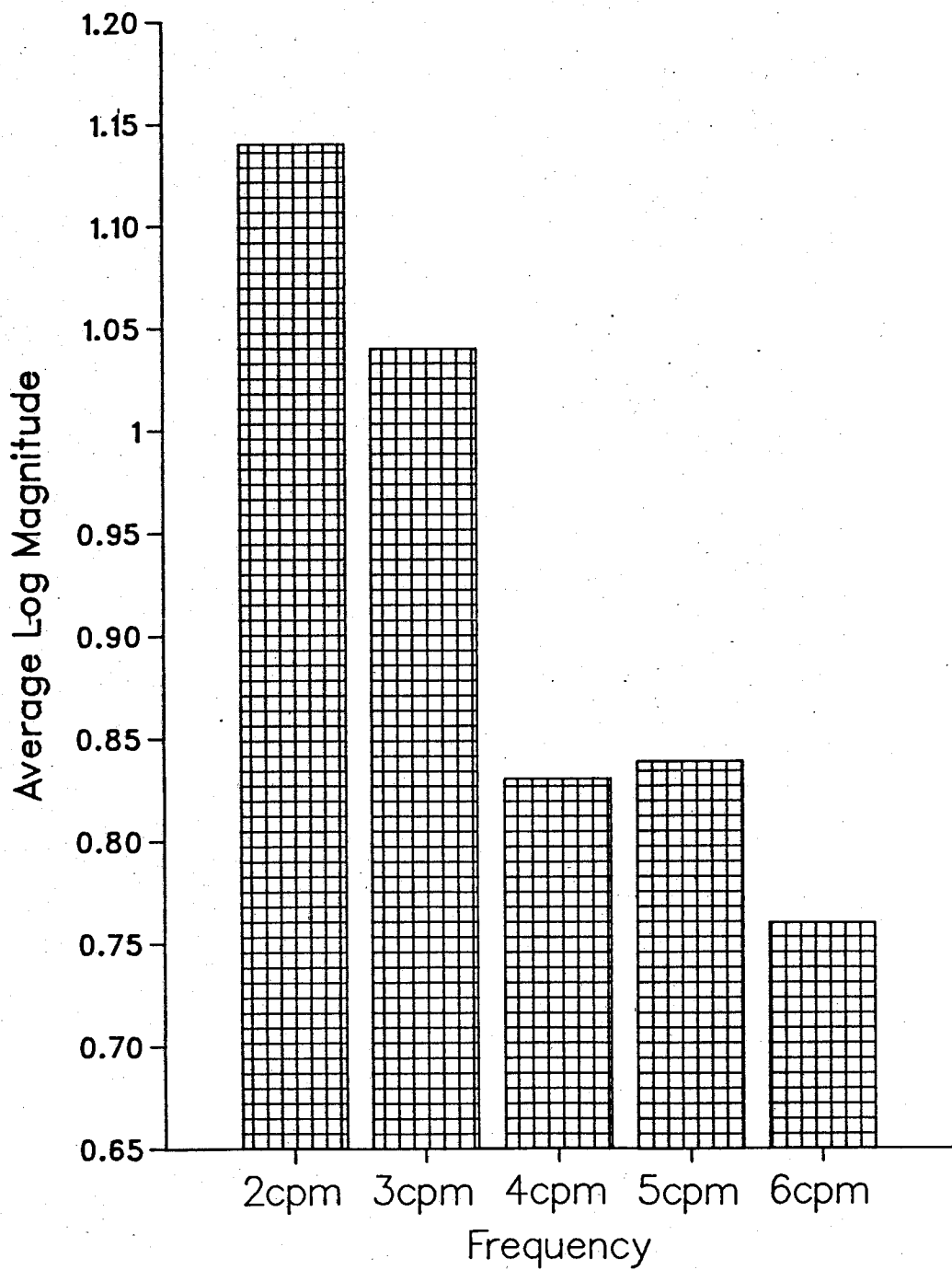


Figure 10. Average Magnitude of EGG Frequency Components For Each Group Per Frequency Band

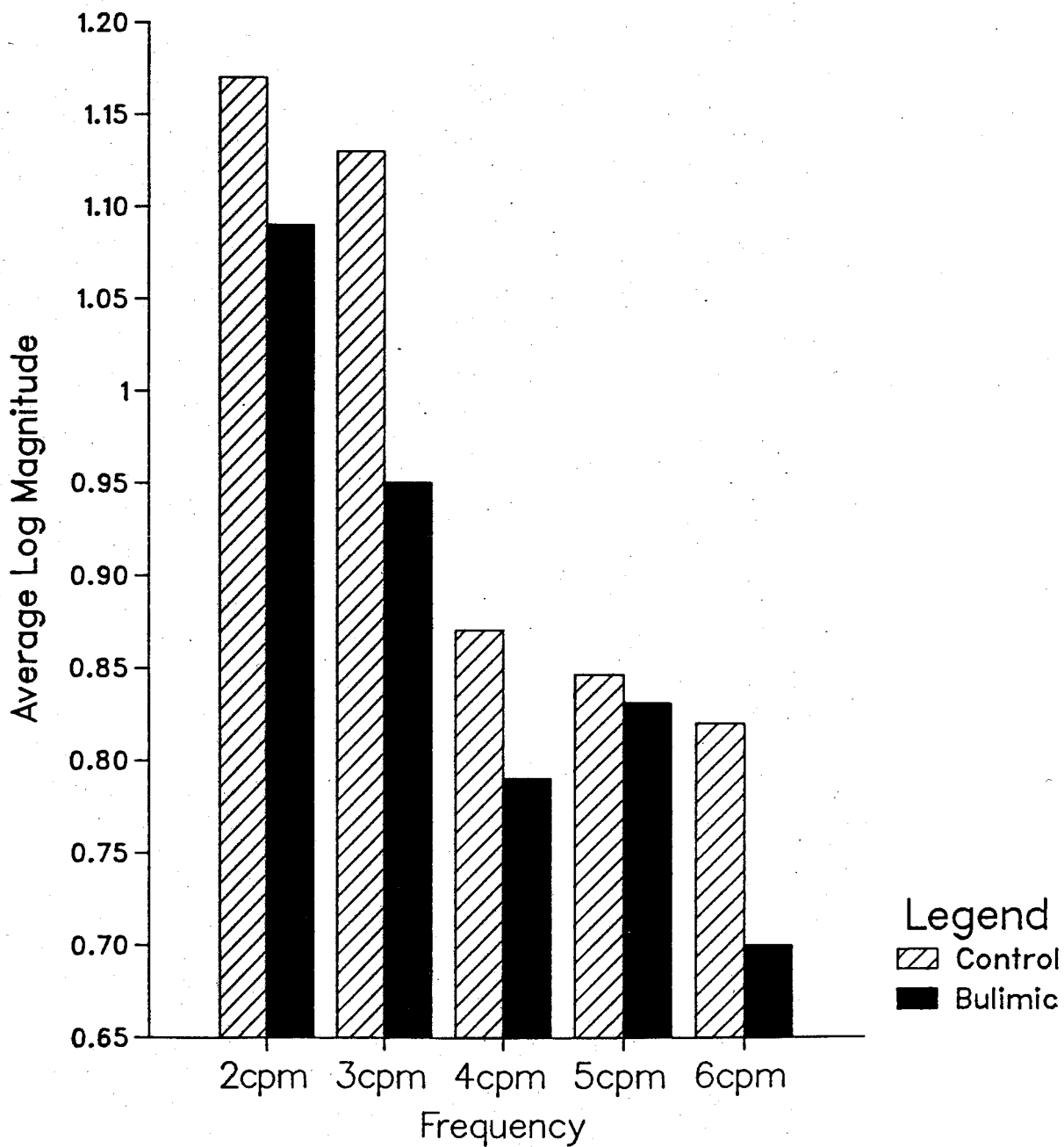


Table 4

Multiple Comparisons Between Group Means Per Frequency Range

Comparisons		2cpm	3cpm	4cpm	5cpm	6cpm
k=10	<u>q</u>	1.67	3.75	1.67	0.21	2.50
<u>df</u> =40*	<u>p</u>	n.s.	n.s.	n.s.	n.s.	n.s.

\*Satterthwaite correction

**PART D**  
**DISCUSSION**



After eating, the EGGs of the bulimics exhibited less power than the controls in the 2 to 6 cpm frequency range. Moreover, such alterations in electrical activity were found in individuals who not only met DSM-III criteria for bulimia, but who also reported that binge eating was a major concern. Feelings of dyscontrol, self-contempt and depression were also common. It is of interest that the one control subject who binged (see reference note 6) reported being moderately concerned about her binge eating, a view shared by only two bulimics. Perhaps the level of concern also discriminates individuals who binge eat from those with bulimia.

Overall, the frequency of gastric electrical activity for both the bulimics and the control group was within the range characteristic for ECA-ERA. At the same time, however, the most dominant frequency was in the range of 2 cpm rather than 3 cpm. While the reason for this is unclear, it may be related to the analysis regime. Specifically, collapsing all frequencies within a certain band into a mean frequency value precludes an examination of variability around any specific frequency value. Furthermore, there was wide variability in the preprandial EGG records with respect to observable 3 cpm activity. In fact, in some records there was no discernable 3 cpm preprandial activity (see Figure 5A). This is in contrast to Smout et al. (1980) who observed a 3 cpm component in all fasting EGGs. Possible reasons for this failure to find 3 cpm activity include excessive fat between the stomach and the electrode, improper electrode

location and/or too little amplification. Finally, while the nature of the relationship between frequency and group type was not revealed, Figure 10 shows that more power was evident in all but one frequency band by the control group. In fact, by visual comparison, more power was exhibited by the control group in the 3 cpm range than in the other frequency bands.

While increased power in the 2 to 6 cpm frequency range characterized the postprandial phase overall, there was an absence of variation in frequency, which is contrary to the well-documented 'dip' in slow wave frequency following feeding (Smallwood & Brown, 1983). One reason for this lack of alteration in frequency could have been methodological. For example, Jones and Jones (1985) have demonstrated that the 3 cpm activity of the EGG signal is slowed significantly during the first ten minutes after eating. Accordingly, since the Post 1 time epoch began 12 minutes into the postprandial phase, early variation in frequency could have been missed. Also, research indicates that the type of food ingested during the test meal can have a speeding or slowing effect on frequency (Jones & Jones, 1985). As it is not known how ENSURE affects gastric electrical activity (although its effects with respect to gastric emptying are documented), it may have contributed to this lack of variation in frequency.

Since normal EGGs are characterized by frequencies in the range of 3 cpm and increased postprandial EGG amplitude (Davis, C. M., et al. 1985; Smout et al., 1980; Stern, 1985), it may be

concluded that the electrogastrograms of the control group were 'normal' with respect to frequency. At the same time, it is also clear that there were disturbances in the postprandial EGGs of the bulimics<sup>7</sup>. While the control group was characterized by increased power in the 2 to 6 cpm frequency range approximately 12 minutes into the postprandial phase, a similar increase in activity was not found in the bulimics. Moreover, these differences were no longer significant 42 minutes into the postprandial phase. As well, any differences in electrical activity between groups were minimal in the fasting state (see Figure 4). These findings suggest an explanation for the lack of overall differences between the bulimics and the control group. Specifically, bulimics may resemble normal eaters in the fasted state. It is only with the ingestion of food that differences in abdominal electrical potentials arise. These then diminish as the food empties from the stomach into the duodenum.

Furthermore, these differences in postprandial electrical activity suggest that ECA and/or ERA may be impaired in bulimics. Since Fourier analysis yields information regarding frequency, it is not appropriate to relate the power of certain frequency bands with changes in amplitude. However, considering that postprandial EGGs are characterized by increases in wave amplitude and that the FFTs of the control group exhibited increased power in the postprandial state, it is likely that changes in waveform amplitude contributed to the increased power in the postprandial phase. By comparison, the rather flat power

spectrum of the bulimic group suggest not only minimal differences between pre- and postprandial EGGs, but small variations in postprandial waveform amplitude. This, in turn, suggests disturbances in ERA. Furthermore, it may be that the mechanical activity of the stomach is also impaired, although such an interpretation is tentative as the relationship between EGG amplitude and contractile force is not strong (van der Schee, Smout, & Grashius, 1982). For example, Brown et al. (1975) found a clear increase in amplitude of 3 cpm waves in surface EGGs following eating. While they attributed this to the dislocation of the stomach (i.e., the result of closer electrode proximity in the distended postprandial stomach) rather than stronger waves, Smout et al. (1980) maintained that amplitude increases were indicative of ERA, especially since they observed increased amplitudes during isolated contractions in an empty stomach. However, while variations in amplitude of the EGG should indicate variations in contractile motility, firm conclusions cannot be drawn (Jones & Jones, 1985). Consequently, researchers suggest that a "change in amplitude, particularly in combination with certain changes in waveform denotes the presence of ERA" (Davis, C. M. et al., 1985, p. 37). As well, such changes provide information about variation in contractile activity (Jones & Jones, 1985).

Research also suggests that there is a relationship between nausea and/or vomiting and tachygastria, tachyarrhythmia (Geldof et al., 1983a, 1983b; You & Chey, 1984; Vantrappen, Schippers,

Janssend, & Vandeweerd, 1984) and decreased waveform amplitude in the postprandial state (Geldof et al., 1983a, 1983b). While abdominal complaints such as bloating, nausea and vomiting are common in bulimia, similar disturbances in gastric activity were not found in this study. One reason for this may have been methodological as the frequencies characteristic of tachygastria and tachyarrhythmia ( $>7$  cpm) were not considered for analysis (the range was restricted to those frequencies typical for ECA-ERA). It may be that reduced power in the 2 to 6 cpm frequency range simply reflects increased power at the higher frequencies. Another possibility could be the nature of the subject population under investigation. Perhaps individuals with unexplained nausea and/or vomiting are an inappropriate comparison group for bulimics, even if their symptomatology is similar. On the other hand, it was not known if the bulimics in the current research tend to experience nausea or bloating and/or if they did so in the testing situation. In summary, perhaps what is distinctive about bulimia is a minimal increase in the power of postprandial electrical activity (possibly indicative of impaired ERA) in the absence of unstable frequency.

It is clear that the exact nature of the disturbance in abdominal electrical potentials cannot be discerned from the present research. As well, if bulimics tend to binge when they are not extremely hungry, as Davis, R., et al. (1985) suggest, then the alterations in electrical activity shown may or may not

occur during the binge episode. At the same time, however, these results do suggest that the abdominal complaints experienced by many bulimics may be related to disturbances in gastric function. Perhaps, diminished gastric activity in the postprandial phase precipitates gastric retention which in turn leads to experiences of abdominal fullness and bloating.

While it is impossible to know if changes in gastrointestinal function are primary or secondary to the development of bulimia, it may be that bulimics with a history of anorexia nervosa (the reported incidence ranges from 5% (Russell, 1979) to 28% (Abraham & Beumont, 1982)) are more at risk for developing complications. Specifically, the increased incidence of delayed gastric emptying among anorexics as well as the success of prokinetic drug treatment suggest that the mechanical activity of the stomach may be impaired in anorexic individuals. Perhaps, bulimics who have been previously anorexic are more susceptible to, or have already developed, disturbances in gastrointestinal function.

In an attempt to more fully understand the nature of the alterations in gastric electrical activity particular to bulimic individuals future research should incorporate more rigorous subject selection criteria. Not only would this minimize possible confounding influences (i.e., drug effects), it would permit an EGG analysis of bulimics with particular associated features, i.e., with or without a previous history of anorexia nervosa. As well, a more comprehensive assessment of abdominal

complaints such as epigastric bloating and/or nausea is necessary, for it may be that their severity and/or chronicity are related to progressive changes in gastric function. Finally, the Binge Scale should be used as a method for screening out subjects who binge and purge in the nonbulimic comparison sample.

With regard to methodology, a wider range of frequencies should be incorporated into the analysis format. Also, a recently developed method of Fourier analysis known as Running Spectrum Analysis (RS) should be considered (van der Schee et al., 1982). As this technique provides overlapping power spectra displayed as a function of time, it offers the advantage of yielding continuous time and frequency information. As van der Schee et al. (1982) note, RS analysis "allows the recognition and quantitative analysis of gastric (and duodenal) frequency patterns, relatively uninfluenced by the appearance of short-lasting motion artefacts" (p. 249). As a result, this technique may be particularly useful for detecting repeatedly occurring episodes of tachygastrica and tacharrhythmia of short duration. In addition, with RC analysis, more data (i.e., increased sample size) and/or a revised analysis format it may be possible to clarify the nature of relationship between frequency changes and group type.

Finally, future research should consider investigating changes in abdominal electrical potentials at different stages in the binge cycle. Perhaps there are particular alterations in

electrical activity prior to and subsequent to bingeing. As well, it is not known how the anticipation of vomiting - or the prevention of it - affects EGG recordings. Not only would this provide a more accurate assessment of changes in gastric activity in bulimic individuals, but it would provide additional support for the clinical usefulness of the surface electrogastrogram.



**APPENDIX A**

DSM-III Criteria for Bulimia

1. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time, usually less than two hours).
2. At least three of the following:
  - a. consumption of high-caloric, easily ingested food during a binge
  - b. inconspicuous eating during a binge
  - c. termination of such eating episodes by abdominal pain, sleep, social interruption, or self-induced vomiting
  - d. repeated attempts to lose weight by severely restrictive diets, self-induced vomiting, or use of cathartics or diuretics
  - e. frequent weight fluctuations greater than ten pounds due to alternating binges and fasts
3. Awareness that the eating pattern is abnormal and fear of not being able to stop eating voluntarily.
4. Depressed mood and self-deprecating thoughts following eating binges.
5. The bulimic episodes are not due to Anorexia Nervosa or any known physical disorder.

**APPENDIX B**

## Subject Information

### Gastric Motility and Bulimia

Conducted by C. Barker and Dr. L. Solyom

In this experiment, we are recording the movements of your stomach before and after you have eaten. This is done by attaching an array of five electrodes to your abdomen above and near your navel. These electrodes are sensitive to any stomach movement and will transfer the electrical potentials to the recording device in the next room. Before these electrodes can be placed, however, the skin surface beneath the electrode sites must be cleaned with alcohol and gently rubbed with electrode jelly. This cleaning preparation is necessary as it removes oils and loose skin tissue, thereby enhancing the quality of the recorded signal. Finally, your heart rate and respiration will also be monitored. More specifically, an electrode will be placed on your sternum to record your heart beat and an elastic belt equipped with a small transducer will be fastened around your chest to register your breathing.

After you have completed a brief questionnaire and the electrodes have been attached (approximately one hour), you will be asked to lie quietly for 30 minutes. At this time, you will be fed a liquid meal, after which recording will continue for another 90 minutes. A \$10.00 payment for your participation will be presented to you at this time.

In order to assure confidentiality, a code number will be assigned to all documents pertaining to you. In addition, the questionnaire will be shredded upon completion of this study.

It is hoped that if you have any questions regarding this procedure or queries about the laboratory itself, you will make them known. Once again, be assured that this study may be terminated by you at any time

Finally, since the lights will be dimmed during data collection, you may sleep if you wish.

I have read this description \_\_\_\_



SIMON FRASER UNIVERSITY, BURNABY, B.C., CANADA, V5A 1S6  
CONSENT FORM DEPARTMENT OF PSYCHOLOGY; 291-3354

This experimental procedure has been requested by

\_\_\_\_\_  
Carol Barker and Dr. L. Solyom

I have read the procedures specified in the document  
entitled:

\_\_\_\_\_  
Subject Information: Gastric Motility and Bulimia

I understand the procedures to be used in this experiment  
and also understand that I am free to withdraw at any time prior to  
the completion of this study.

Furthermore, if I am currently undergoing treatment for  
bulimia, my withdrawal will, in no way, jeopardize my opportunity  
to receive continued medical care.

My signature below certifies that I consent to the  
experimental procedures described in the document stipulated above  
and which are to be conducted on the following date:

\_\_\_\_\_  
in the following place:

\_\_\_\_\_  
CC4205, Simon Fraser University

and designated in the following manner:

\_\_\_\_\_  
Gastric motility and bulimia

DATE \_\_\_\_\_ NAME \_\_\_\_\_

SIGNATURE \_\_\_\_\_



APPENDIX C

B Scale

NAME: \_\_\_\_\_

DATE: \_\_\_\_\_

DIRECTIONS: Please read each item carefully and circle the number beside the statement that best applies to you.

1. Binge eating is the rapid consumption of a large amount of food in a discrete period of time. How often do you binge eat?
  - 0 seldom.
  - 1 once or twice a month.
  - 2 once a week.
  - 3 almost every day.
2. What is the average length of a binge eating episode?
  - 0 less than 15 minutes.
  - 1 15 minutes to one hour.
  - 2 one hour to four hours.
  - 3 more than four hours.
3. Which of the following statements best applies to your binge eating?
  - 0 I eat until I have had enough to satisfy me.
  - 1 I eat until my stomach feels full.
  - 2 I eat until my stomach is painfully full.
  - 3 I eat until I can't eat anymore.
4. Do you ever vomit after a binge?
  - 0 never.
  - 1 sometimes.
  - 2 usually.
  - 3 always.
5. Which of the following best applies to your eating behaviour when binge eating?
  - 0 I eat more slowly than usual.
  - 0 I eat about the same as I usually do.
  - 1 I eat very rapidly.
6. How much are you concerned about your binge eating?
  - 0 not bothered at all.
  - 1 bothers me a little.
  - 2 moderately concerned.
  - 3 a major concern.
7. Which best describes your feelings during a binge?
  - 0 I feel that I could control the eating if I chose.
  - 1 I feel that I have at least some control.
  - 2 I feel completely out of control.
8. Which of the following describes your feelings after a binge?
  - 0 I feel fairly neutral, not too concerned.
  - 1 I am moderately upset.
  - 2 I hate myself.
9. Which most accurately describes your feelings after a binge?
  - 0 not depressed at all.
  - 1 mildly depressed.
  - 2 moderately depressed.
  - 3 very depressed.



**APPENDIX D**

Summary of the Analysis of Variance (Overall)

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Source	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Group (G)	1	1.21	2.64	n.s.
Error	24	0.46		
Time (T)	3	0.97	7.95	0.0001
T x G	3	0.50	4.08	0.0098
Error	72	0.12		
Frequency (F)	4	2.63	76.71	0.0
F x G	4	0.09	2.82	0.0481
Error	96	0.03		
T x F	12	0.01	0.84	n.s.
T x F x G	12	0.01	0.91	n.s.
Error	288	0.01		

---

Note: All probabilities are Huynh-Feldt

## REFERENCE NOTES

1. The prescribed medications were Sinequan (Doxepin) (75 mg daily), Ludiomil (300 mg daily) and Pondimin (Fenfluramine) (60 mg daily).

2. The Binge Scale was originally given exclusively to the bulimic subjects. However, due to self-disclosure on the part of one control subject and information gleaned through a review of the literature, it became apparent that a change in format was necessary. Consequently, the BS was incorporated into the experimental procedure and those control subjects who had not completed this questionnaire at the time of data collection ( $n=9$ ) did so retrospectively.

3. The cardiometer and strain-gauge respiratory transducer were designed for the psychophysiological laboratory at Simon Fraser University by Howard F. Gabert, P.Eng.

4. Prior to data collection, the proposed method of frequency analysis was Peak Detection. Utilizing a program recently developed by H. F. Gabert, P.Eng., this technique detects periodicity in the data by counting peaks and specifying inter-peak distances. These values are then averaged and standard deviations computed. Since the parameters for analysis in the PDP are controlled by the software user, it was hoped that this technique would be particularly sensitive to the waveform pattern characteristically recorded by the EGG.

Unfortunately, such was not the case. Not only was 3 cpm activity inconsistent across records, there was great variability in waveforms between subjects (H. F. Gabert, personal communication, November 14, 1985). Such variability, along with a lack of identifiable waveform activity, necessitated abandoning the PDP in favor of the most commonly used method of analyzing the EGG, Fast Fourier Transform.

5. Raw EGG data was smoothed prior to FFT analysis with a low-pass filter window of 6 points (-20 db/decade), producing a .266 Hz. cutoff. In addition, since the spectral analysis was computed from time-limited epochs of continuous data, 'windowing' (using a ramp of 70 points) was employed to minimize the unwanted effects created by discontinuities in the record segment at the beginning and end of each data snapshot.

6. One control subject scored within the bulimic range on the Binge Scale. Specifically, her score of 16 was coincident with the mean score of 16.53 in the bulimic sample. However, since subsequent statistical analysis of her EGG record (multiple t-tests) revealed that her data were within acceptable limits, they were included in the data file for the control group. As expected, her scores on the BS elevate the overall response statistics reported for the controls.

7. Although it was impossible to discern if, or how, the EGGs of the subjects taking medications were affected, the fact that significant differences appeared postprandially and not

pre-prandially suggests that ECA and/or ERA may be disordered in bulimics.

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