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A method for the control of eating rate: A potential intervention in eating disorders

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A method for the control of eating rate gave subjects feedback from a computer screen on how much and at what rate to eat during a meal. The method also allowed us to record the development of satiety during the meal. Linear eaters—that is, women selected for eating at an approximately constant rate—underate when challenged to eat at a lower rate and overate when challenged to eat at a higher rate, thereby modeling the eating behavior of patients with anorexia nervosa and binge eating disorder, respectively. In both cases, the women's postmeal perception of satiety mimicked that of the respective patient group. The results provide support for the notion that linear eaters have the capacity to exhibit disordered eating.

A recent review reported that recovery from eating disorders takes a long time; only 11% of patients with anorexia nervosa and only 10% of those with bulimia nervosa are free of symptoms in 10 years (Von Holle et al., 2008). As the authors pointed out, however, criteria for remission have not been universally accepted; with less strict criteria, more patients remit, but then the majority relapse within a year (Von Holle et al., 2008). The same conclusions had been reached before—for example, by Ben-Tovim et al. (2001), who also pointed out that existing treatments have little, if any, effect. In the years after the review of Ben-Tovim et al., about 3,500 articles have been published on "anorexia" and "bulimia" (PubMed search), but there has been no significant improvement in outcome (Von Holle et al., 2008) or in treatment efficacy (Striegel-Moore & Bulik, 2007).

The incidence of obesity is escalating (Bovbjerg, 2008). Alas, with only one exception, available treatments are only marginally effective (Bergh et al., 2008). That exception is restrictive gastric surgery, but patients relapse into their preoperative pattern of eating within 30 min after relief of the gastric constraint imposed 15 months earlier (Bergh et al., 2008).

Suggested Point of View

Although eating disorders are thought to be a consequence of predisposing mental problems, treatment of these problems has not significantly improved outcome (Striegel-Moore & Bulik, 2007). Similarly, although human biology clearly permits the development of obesity, candidate genetic predisposing factors may be on the order of thousands (Emilsson et al., 2008), making genetic-evidence-based clinical intervention in the near future unlikely.

To improve this situation, we suggested a neurobiologically plausible framework for eating disorders (Södersten, Nergårdh, Bergh, Zandian, & Scheurink, 2008), based on observations of enforced (Burger, Sandstead, & Drummond, 1948) and experimental (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950) starvation. There can be no doubt that all the symptoms of eating disorder patients emerge from starvation (Södersten et al., 2008), including the perceptual biases and mental problems once thought to be specific for anorexic patients (Crow, 2004). These symptoms are reversible and include many changes, which have emerged during evolution as beneficial adaptations to the all too common challenge of starvation (Södersten et al., 2008).

Obesity is the extreme opposite of anorexia on the same behavioral and biochemical continuum (Bergh et al., 2008). Because of these "similarities," we have postulated that anorexia and obesity can be similarly treated (Bergh et al., 2008).

Eating Behavior As Cause and Intervention

When eating behavior normalizes, body weight and the other symptoms of anorexia, including the mental symptoms, normalize (Bergh, Brodin, Lindberg, & Södersten, 2002). Although normalization of body weight is an obvious criterion for remission, it is probably not the cause of the symptomatic relief, because normal weight bulimics and overweight patients with binge eating disorder (BED) have mental problems similar to those of anorexics (Södersten et al., 2008). Instead, we have hypothesized that disordered eating behavior causes the mental problems; relearning how to eat is essential for restoration of body weight and symptomatic relief (Södersten et al., 2008; Zandian, Ioakimidis, Bergh, & Södersten, 2007). Treatment based on this assumption has been demonstrated to be effective in randomized controlled trials with eating disorders (Bergh et al., 2002).

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In this study, we present our refined methodology for recording and analyzing eating behavior data from single meals. Current innovations include advances in the data analysis. Our in-house algorithm for data filtering and validation automates the procedure and facilitates its use for an increased number of meals. We also present evidence supporting our hypothesis connecting eating behavior characteristics with eating disorders. Data from anorexic and BED patients have not been collected and analyzed under this scope before. Moreover, there is no prior description of healthy individuals' behavior mimicking that of patients, in a controlled environment.

METHOD

The Mandometer is a scale connected to a computer. The patient places a plate on the scale and puts food on the plate, and the computer records the weight loss of the plate during the meal. This yields a curve of cumulative food intake (Jordan, Wieland, Zebley, Stellar, & Stunkard, 1966; Kissileff, Thornton, & Becker, 1982; Pudel, 1971; Westerterp-Plantenga, 2000; Zandian, Ioakimidis, Bergh, Brodin, & Södersten, 2009). During treatment, patients are provided with feedback on the computer screen on how much and at what rate to eat (Zandian, Ioakimidis, Bergh, et al., 2009). Thus, the method allows control of eating rate. In addition, a rating scale is presented on the screen during the meal; patients rate their feeling of fullness, and the Mandometer generates a curve of the development of satiety. Feedback for the perception of satiety during meals can also be provided (Zandian, Iokimidis, Bergh, et al., 2009).

Tested this way, eating behavior can be described by a quadratic equation: Food intake $y = ax^2 + bx + c$, where a = change in eating rate over time, b = initial eating rate, and $c = 0$—that is, food intake at start of the meal (Kissileff et al., 1982; Pudel, 1971; Westerterp-Plantenga, 2000; Zandian, Iokimidis, Bergh, et al., 2009). Most often, eating decelerates during the meal (i.e., $a \leq 0$); accelerating eating rates have been reported only in individual patients with bulimia (Kissileff, Walsh, Kral, & Cassidy, 1986) or binge eating (Westerterp-Plantenga, 2000). Linear eaters—that is, individuals with $a \approx 0$—overeat when eating rate is experimentally increased and undereat if it is decreased; this may put them at risk of developing the disordered eating behaviors of obesity and anorexia (Zandian, Iokimidis, Bergh, et al., 2009).

In the present study, we selected linear eaters, challenged them to eat slowly or quickly, and compared their behavior with that of anorexic and BED patients to validate our method.

Subjects

Twenty-nine female students were recruited by advertisement on a nearby university campus. They filled in questionnaires to ensure that they were nonsmoking and nonvegetarian and had no food-specific allergies, no medical problems, no medication, and no history of anxiety or eating disorders. They first were screened (see below) and were included in the study if their eating rate $a \geq -1.5$, their age was between 18 and 24 years, and their body-mass index (BMI, weight/height squared, kg/m^2) was between 19 and 25. Sixteen women fulfilled these criteria; their median age was 19.8 years (range, 17.8–24.3) and their BMI = 21.7 (18.5–24.6).

Sixteen female patients with anorexia nervosa who were 14.6 years (12.3–26.7) old, had a BMI = 15.3 (13.4–16.9), had been ill for 2.6 years (0.3–9.5), had had 1.5 (0–4) previous treatments, and were consecutive referrals to our clinic during a period of 6 months participated. Twelve female BED patients who were 32.2 years (17.3–63.3) old, had a BMI = 39.5 (31.2–62.8), had been ill for 15.1 years (2.2–45.5), had been obese for 16.3 years (2.2–57.3), and who had had 3 (0–6) previous treatments were similarly recruited. Both groups of patients fulfilled DSM–IV diagnostic criteria. We studied women because, ultimately, our aim was to understand eating disorders, which mainly affect women.

Materials

The preprogrammed eating curve that provides feedback on the screen of the Mandometer (Mikrodidakt, Lund, Sweden) has no numerical values; subjects are unaware of the amount of food that they consume. Further feedback during the meal is provided by text and sound when eating deviates up to 15 g from the presented curve ("You are eating too quickly/too slow").

While eating, subjects indicate their perceived satiety on a revised Borg CR10 Scale (Borg, 1982), which appears on the Mandometer screen. It is a vertical line, with categories ranging from *no feeling* to *maximum feeling*. Any point along the scale can be selected by pressing on the touch screen, and values ranging from 0 to 100 are generated.

The subjects ate normal Swedish food (Findus Foodservices AB, Malmö, Sweden; 400 kJ, 4.5 g protein, 18 g fat, and 15 g carbohydrate/100 g), and the patients were provided with standard restaurant food of their choice, with a similar proportion of macronutrients and caloric density.

Procedure

The healthy women were first accustomed to the procedure during an initial visit with a meal; no data were collected.

Eating session setup. Meals were served at 11:30, 12:00, or 12:30 h, and the subjects, who were asked to refrain from snacking and drinking (except water) after breakfast, were escorted into a dining area 5 min before the meal. No social contact, reading, or listening to music were allowed. Great care was taken in keeping the composition, preparation, and presentation of each meal constant. Thus, the food was weighed before it was served, and food not consumed was also weighed to calculate the amount consumed. The duration of each meal was calculated as the interval between the moment when food was first removed from the plate and the moment when the fork and knife were placed on the table. Food intake and meal duration measured in this way overlapped 98.3% and 96.5%, respectively, with Mandometer-generated values.

Control test. The women first ate a meal on the Mandometer without constraints (Figure 1A). A bowl with 1,300 g of food was presented, and the women could eat as much as they wanted, adding food to their plate at any point and eating at their own pace. Out of the 29 women, 16, who ate with $a \geq -1.5$, were selected for further study. Their $a \approx 0$ on average, and they therefore will be referred to as *linear eaters* in the following. It is sufficient to test subjects once; the food intake curve is intraindividually stable (Kissileff et al., 1982; Pudel, 1971; Westerterp-Plantenga, 2000; Zandian, Ioakimidis, Bergh, et al., 2009).

Decreasing and increasing eating rate. The women ate two experimental meals in which eating rate was either decreased (ER−) or increased (ER+). This was achieved by placing 30% less (ER−) or 40% more (ER+) food on the plate, as compared with the amount consumed during the control test (Figure 1B). The women followed a curve/line with the a value that they had generated in the control test displayed on the Mandometer during the meal. The tests were given in random order and separated by 7–20 days. The women were informed about the general procedures, but meal-specific details were not disclosed.

Similarly, the patients were first accustomed to the procedure and then tested as in the control procedure; this is part of their diagnostic examination at admission.

The procedures were approved by the ethics committee of the Karolinska Institute, and the women gave written consent to participate; they were free to drop out of the study any time.

Data Analysis

Data collection and filtering. During each meal, the weight on the scale was recorded every 4 sec with an accuracy of 1 g, and the

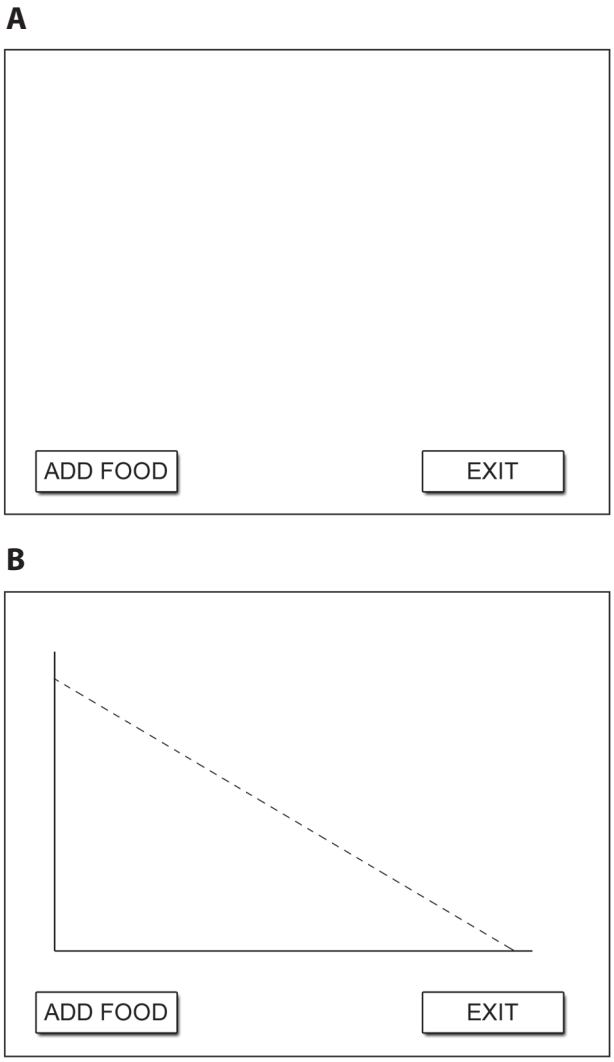

Figure 1. Schematic representations of the on-screen instructions for the Mandometer. (A) Blank screen during the control meals. (B) Subjects are asked to adjust their eating rate to a reference line. The axis scales are modified to a decreased (ER−) or increased (ER+) eating rate, but the reference line always appears the same.

data were transferred to a PC. An automated algorithm for data filtering, developed using MATLAB (The MathWorks, Natick, MA), excluded noise—for example, increases of weight due to pressure on the plate or placement of utensils on the plate or inactive periods at the beginning and end of the meal. In-meal incidents—for example, addition of food to the plate—in the data series were treated as periods with zero intake. The data were then transformed into curves, starting with zero until the end of the meal, with the time scale transformed into minutes.

Curve fitting. The food intake curve, $y = ax^2 + bx$, was calculated using least squares best curve fitting (Sigmaplot, Systat Software, San Jose, CA). Only fits with $r^2 \geq .9$ were analyzed; 5 out of 76 fits were excluded.

Statistical analysis. Results are presented as box plots in the figures and as medians (range) in the text. Normality for the distribution of continuous variables was verified by the Kolmogorov–Smirnov goodness-of-fit test, and ANOVAs for repeated or independent measures were used, as appropriate, to analyze the data. Post hoc comparisons for ANOVAs were performed with the Tukey HSD tests, and differences between the control and experimental measurements were evaluated by two-sided Dunnett tests. The analyses were performed using Statistica 8.0 (StatSoft, Tulsa, OK).

RESULTS

Control Meals

We first compared the food intake of the linear eaters in the control meal with that of the anorexic and BED patients (Figure 2).

There was a significant difference among the groups in the rate of deceleration of eating [$F(2,41) = 6.34$, $p < .004$]; the a values of the BED patients were significantly more negative than those of the anorexics, but there were no other significant differences. In anorexics, $a \approx 0$ with very little variation.

The initial eating rate differed significantly among the groups [$F(2,41) = 53.91$, $p < .001$]; anorexics showed significantly lower b values than did both linear eaters and BED patients, and BED patients had higher b values than did the linear eaters.

There were significant differences among the groups in food intake [$F(2,41) = 30.14$, $p < .001$]; anorexic patients ate significantly less than did linear eaters and BED patients, who ate more than did the linear eaters.

Also, there was a significant group effect on meal duration [$F(2,41) = 50.8$, $p < .001$]. Linear eaters completed their meals in a significantly shorter time than did anorexic patients, but the difference between the linear eaters and the BED patients was not statistically significant.

Effect of Decreasing or Increasing Eating Rate in Linear Eaters

Next, we compared the food intake of the linear eaters in the experimental meals with the control meal (Figure 2).

There was no significant difference in the rate of deceleration, but there was a significant difference in initial eating rate [$F(2,30) = 69.44$, $p < .001$]; b values were significantly lower in the ER− condition and higher in the ER+ condition than in the control condition. Since the linear eaters were asked to decrease or increase their food intake and follow intake curves with a values that they had generated in the control test, these results are not surprising; a and b were not strictly outcome variables. Even so, the results show that linear eaters are able to decrease or increase their eating rate.

More interesting, there was a significant difference in food intake [$F(2,30) = 42.23$, $p < .001$]; the linear eaters ate significantly less food in the ER− condition and more food in the ER+ condition than in the control condition (Figure 2). Equally interesting, there was also a significant difference in meal duration [$F(2,30) = 60.46$, $p < .001$], which, however, went in the other direction. Thus, although the linear eaters ate less food in the ER− condition, they completed this meal in a significantly longer time than the control meal, and although they ate more food in the ER+ condition, they completed this meal in a significantly shorter time than the control meal (Figure 2).

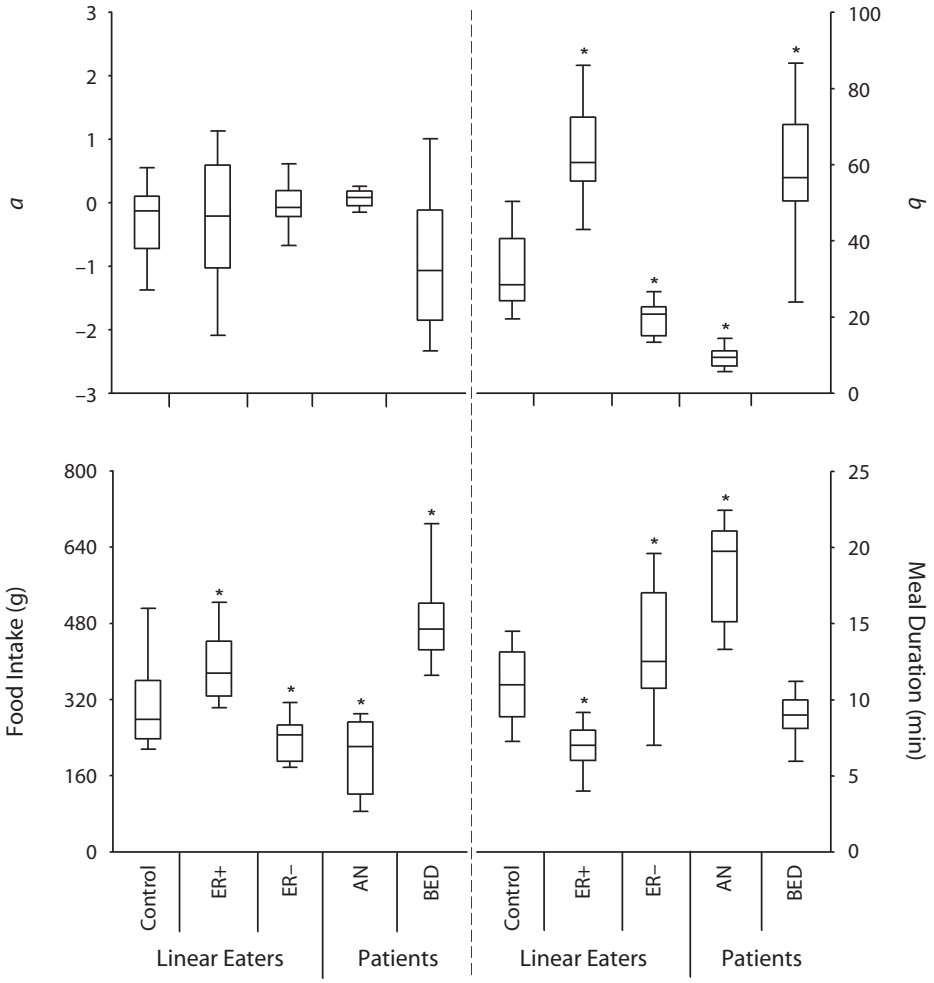

Figure 2. Rate of deceleration (*a*), initial eating rate (*b*), food intake, and meal duration for women who ate at a constant rate (linear eaters, $n = 16$) and female patients with anorexia nervosa (AN, $n = 16$) or binge eating disorder (BED, $n = 12$). Linear eaters were challenged to eat at an increased (ER+) or decreased (ER−) rate. $^{*}p < .005$, as compared with control.

Clinical Validation of the Method

Finally, the hypothesis that anorexic and BED eating behavior can be mimicked in linear eaters by experimentally decreasing and increasing eating rate was tested (Figure 2).

All parameters for the eating behavior of linear eaters tested in the ER− condition were statistically indistinguishable from those for anorexic patients, although the duration of the meal was somewhat longer for the anorexics. Similarly, the eating behavior of the linear eaters tested in the ER+ condition was statistically indistinguishable from that of the BED patients, although the BED patients ate somewhat more food.

Figure 3 shows that the experimental manipulations for the linear eaters modeled the eating behavior of anorexic and BED patients well.

Satiety

Although there were no significant group differences in the rating of satiety before the meal [$F(2,41) = 1.66$, n.s.], there were significant differences by the end of the meal [$F(2,41) = 4.79$, $p < .001$; see Figure 4]. It is noteworthy that even though anorexic patients rated their satiety significantly higher than did linear eaters, they ate less food, and although BED patients ate more food than did linear eaters, they did not significantly increase their postmeal rating of satiety. Interestingly, the satiety ratings of the linear eaters approached those of anorexic patients in the ER− condition. Equally interesting, they ate less food in this condition. The rating of satiety did not increase in the ER+ condition in linear eaters, although they ate more food.

DISCUSSION

Attempts have previously been made to describe patterns of eating within patient groups (e.g., Kissileff et al., 1986; Westerterp-Plantenga, 2000). This strategy assumes that eating is an outcome of factors endogenous to the patient. We take a different approach in hypothesizing that eating behavior is a cause of some of the problems as-

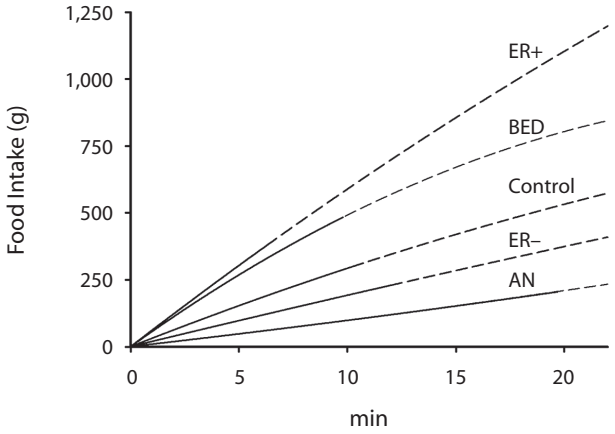

Figure 3. Cumulative food intake in women who ate at a constant rate (linear eaters, $n = 16$) and female patients with anorexia nervosa (AN, $n = 16$) or binge eating disorder (BED, $n = 12$). Linear eaters were challenged to eat at an increased (ER+) or decreased (ER−) rate. The end of the uninterrupted lines indicates the point in time when the subjects stopped eating; the dashed continuation of the line indicates the extrapolation of the modeled curve.

sociated with weight regulation (Södersten et al., 2008; Zandian, Ioakimidis, Bergh, et al., 2009; Zandian et al., 2007). On the basis of this hypothesis, we use eating behavior as an intervention in treating patients who have these problems (Bergh et al., 2002). The present method, Mandometer, was developed to test the hypothesis and to be used for the intervention; it extends the original methods of Jordan et al. (1966), Pudel (1971), and Kissileff, Klingsberg, and Van Itallie (1980) for continuous recording of intake. Pudel first adapted measures obtained with the method to a quadratic equation, which has since been confirmed (Kissileff et al., 1982; Westerterp-Plantenga, 2000; Zandian, Ioakimidis, Bergh, et al., 2009). However, the effect of experimental manipulation on the coefficients of the food intake curve has been reported in only three studies (Kissileff et al., 1982; Muurahainen, Kissileff, Lachaussée, & Pi-Sunyer, 1991; Zandian, Ioakimidis, Bergh, et al., 2009). Hence, in most studies, the cause–effect relationship between eating pattern and the symptoms of the patients remains unclear.

Curves displayed on the monitor of the Mandometer make experiment and clinical intervention feasible with this method. In a previous experiment, we found that decelerated female eaters, with $a < -2$, had difficulty increasing their rate of eating and so consumed less food in response to this challenge (Zandian, Ioakimidis, Bergh, et al., 2009). However, they had no difficulty reducing their eating rate and did not change their intake of food in this condition (Zandian, Ioakimidis, Bergh, et al., 2009). By contrast, linear female eaters, with $a \approx 0$, ate more food when challenged to eat at an increased rate and less food when challenged to eat at a reduced rate (Zandian, Ioakimidis, Bergh, et al., 2009). These results for linear eaters, confirmed in the present study, suggest that they have difficulty maintaining their intake when eating deviates from their control level. These experimental results may be clinically relevant because the eating behavior of anorexic patients was conspicuously similar to that of the linear eaters challenged to eat at a reduced rate and the eating behavior of BED patients was very similar to that of linear eaters facing the opposite challenge—that is, eating at an increased rate. Interestingly, ratings of satiety after the meal among the patients were also similar to those of the linear eaters in the corresponding experimental challenge. These similarities support the clinical validity of the present method.

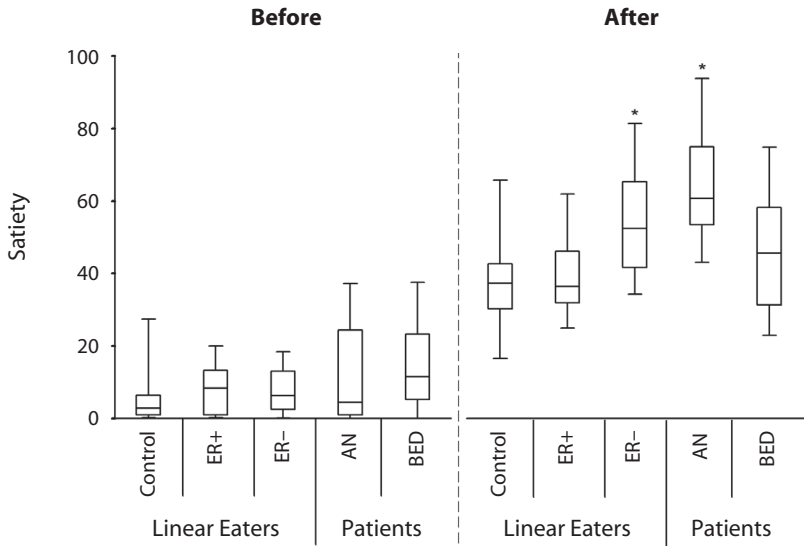

Figure 4. Satiety rating before and after a meal for women who ate at a constant rate (linear eaters, $n = 16$) and female patients with anorexia nervosa (AN, $n = 16$) or binge eating disorder (BED, $n = 12$). Linear eaters were challenged to eat at an increased (ER+) or decreased (ER−) rate. *$p < .005$, as compared with control.

An analysis of the cause–effect relationship between eating behavior and clinical phenotype is essential; unless the present hypothesis that the pattern of eating causes problems of weight regulation is verified, the Mandometer may not be clinically useful. In a recent test of the hypothesis, we found that linear eaters who practiced eating at a decelerated rate maintained the decelerated pattern of eating when tested without feedback from the Mandometer, resisted overeating when challenged to eat at an increased rate, and showed a reduction of some cognitive factors believed to be risk factors for both anorexia and obesity (Zandian, Ioakimidis, Bergh, & Södersten, 2009). Further testing of the hypothesis is underway, including the question of whether a new pattern of eating behavior can be maintained over time and whether problems of weight regulation can be prevented by learning to eat at the proper rate. In these tests, we have improved the method by implementing an algorithm that generates the proper values of *a* and *b* and automatically displays the corresponding training curves on the monitor when the patient places food on his or her plate. This opens the possibility of using the Mandometer in large-scale, hands-off clinical trials.

AUTHOR NOTE

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