BRIEF COMMUNICATION

Dieting and Pain Sensitivity: A Validation of Clinical Findings

S. LAUTENBACHER, K. BARTH, E. FRIESS, F. STRIAN, K. M. PIRKE AND J.-C. KRIEG[1]

Max Planck Institute of Psychiatry, Clinical Institute
Department of Psychiatry, Kraepelinstr. 10, D-8000 Munich 40 Germany

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LAUTENBACHER, S., K. BARTH, E. FRIESS, F. STRIAN, K. M. PIRKE AND J.-C. KRIEG. *Dieting and pain sensitivity: A validation of clinical findings.* PHYSIOL BEHAV **50**(3) 629–631, 1991.—To validate findings of a reduced pain sensitivity in anorexia and bulimia nervosa, the effects of dieting on somatosensation (especially pain sensitivity) were investigated in healthy young women. One group of subjects (n = 11) received a calorically reduced balanced diet for 21 days, while the other group (n = 14) continued to eat normally. The fasting state induced in the dieting subjects was comparable to that of eating disorder patients, since the dieters showed a reduction of the body mass index, a decrease in triiodothyronine and an increase in β-hydroxybutyric acid plasma levels. However, neither the thresholds of pain, warmth, cold and vibration sensitivity nor the peripheral skin temperature changed systematically under the diet. Therefore, the reduced pain sensitivity in eating disorder patients is apparently not a mere effect of fasting, but a true pathological feature.

Somatosensory thresholds Pain Fasting state Eating disorder Anorexia nervosa Bulimia nervosa

IN a series of studies, we observed a reduced heat pain sensitivity in patients with anorexia and bulimia nervosa (6, 7, 9). Reports on an altered activity of the endogenous opioid system (2,10) prompted us to conduct a naloxone-placebo experiment in a subsample of eating disorder patients, which resulted in the finding that naloxone did not normalize the reduced pain sensitivity (7). Thus it is unlikely that the decreased pain sensitivity observed in our patients was due to an opioid-mediated mechanism. We also tested the hypothesis that a subclinical polyneuropathy, due to malnutrition, is responsible for the reduced pain sensitivity. However, the pattern of somatosensory deficits which we observed in the eating disorder patients was not pathognomonic for a polyneuropathy (9).

One similarity between anorexia and bulimia nervosa is the fact that both types of eating disorders exhibit metabolic and endocrine indices of starvation; thus emaciated patients with anorexia nervosa as well as normal-weight bulimic patients display a reduced plasma level of triiodothyronine and an increased serum concentration of β-hydroxybutyric acid (11). Therefore, it might be possible that this pathophysiological state produces the alteration of pain sensitivity. Hence a validation study is necessary to test whether the reduced pain sensitivity is indeed a pathological feature of anorexia and bulimia nervosa or merely a consequence of prolonged or intermittent fasting. For this purpose we initiated a diet study in which healthy females without any signs of an eating disorder either received a calorically reduced balanced diet or served as control subjects. Pain, warmth, cold and vibration thresholds were measured to evaluate the diet effects on the somatosensory system.

METHOD

Subjects

Twenty-five normal-weight (body mass index between 19 and 24) women (11 in the diet group with an age of 24.4 ± 3.4 years, 14 in the control group with an age of 24.8 ± 2.3 years) participated in the study (see Table 1). They had no signs of an eating disorder, substance abuse, major health problems, recent stressful life events, current dysmenorrhea or pregnancy, and no history of neurological or dermatological diseases. All subjects gave written informed consent; the protocol was approved by an ethics commission. The subjects were randomly assigned to the diet or control group. For three weeks the dieters daily received a balanced 1000 kcal diet, which consisted of 50% carbohydrate, 30% fat and 20% protein. The selection of the diet was guided by the consideration that the food consumed by eating disorder patients usually does not deviate from standard values with respect to the nutrients' composition (5,12). The subjects of the

[1]Requests for reprints should be addressed to PD Dr. med. Jürgen-Christian Krieg, Max Planck Institute of Psychiatry, Kraepelinstr. 10 D-8000 Munich 40 Germany.

TABLE 1

BODY MASS INDEX (BMI; HEIGHT/WEIGHT^2), T3 AND β-HBA VALUES, SOMATOSENSORY THRESHOLDS (PAIN, WARMTH COLD, VIBRATION) AND PERIPHERAL SKIN TEMPERATURE FOR SESSIONS 1 AND 2 IN THE DIET GROUP (n = 11) AND IN THE CONTROL GROUP (n = 14) (MEAN ± SD)

		Session 1	Session 2	Test
BMI	Diet	22.1 ± 1.2	20.9 ± 1.2	"group": $F=0.2$, $p=0.65$
(kg/m^2)	Control	21.3 ± 1.4	21.2 ± 1.2	"session": $F=86.4$, $p<0.01$
				"g × s": $F=72.8$, $p<0.01$
T3	Diet	1.24 ± 0.26	1.00 ± 0.23	"group": $F=5.0$, $p=0.04$
(ng/ml)	Control	1.31 ± 0.18	1.33 ± 0.26	"session": $F=11.7$, $p<0.01$
				"g × s": $F=16.7$, $p<0.01$
β-HBA	Diet	0.02 ± 0.02	0.38 ± 0.36	"group": $F=9.4$, $p<0.01$
(μmol/ml)	Control	0.06 ± 0.06	0.03 ± 0.03	"session": $F=11.1$, $p<0.01$
				"g × s": $F=16.0$, $p<0.01$
Pain	Diet	43.2 ± 1.8	43.1 ± 2.0	"group": $F<0.1$, $p=0.89$
(°C)	Control	43.3 ± 1.5	42.7 ± 1.4	"session": $F=5.7$, $p=0.03$
				"g × s": $F=2.9$, $p=0.11$
Warmth	Diet	4.2 ± 2.0	4.3 ± 2.6	"group": $F<0.1$, $p=0.86$
(°C)	Control	4.3 ± 2.4	3.9 ± 2.1	"session": $F=0.3$, $p=0.61$
				"g × s": $F=0.5$, $p=0.47$
Cold	Diet	0.8 ± 0.3	0.8 ± 0.2	"group": $F=1.4$, $p=0.25$
(°C)	Control	1.1 ± 0.8	1.0 ± 0.6	"session": $F=0.4$, $p=0.56$
				"g × s": $F=0.2$, $p=0.63$
Vibration	Diet	0.8 ± 0.6	0.7 ± 0.5	"group": $F<0.1$, $p=0.77$
(μm)	Control	0.7 ± 0.5	0.6 ± 0.5	"session": $F=0.9$, $p=0.35$
				"g × s": $F<0.1$, $p=0.78$
Skin Temp.	Diet	27.1 ± 1.5	26.5 ± 2.6	"group": $F=0.3$, $p=0.62$
(°C)	Control	26.2 ± 1.8	26.7 ± 1.9	"session": $F<0.1$, $p=0.88$
				"g × s": $F=2.7$, $p=0.12$

The results (F and p values) of the two-way MANOVA-analysis [df: (23,1)] for the factors "group" and "session" and the resulting interaction ("g × s") are presented.

control group were instructed not to change their usual eating habits.

Procedure and Apparatus

The sensory variables were tested in two sessions which were separated by a three-week interval and, in the case of the dieters, were performed immediately before and at the end of the diet. The experimental procedure was identical in both sessions.

The sessions for dieters and controls started at 7:30 a.m. with the collection of a blood sample for the biochemical analyses. Triiodothyronine (T3) was assessed as an indicator for prolonged fasting, and β-hydroxybutyric acid (β-HBA) to gain information about the patients' acute fasting state at the time of the experiment [for details see Pirke et al. (11)]. From 8:15 a.m. on, thresholds for pain, warmth, cold and vibration were measured in this sequence on the right foot. Pain and thermal thresholds were obtained with a PATH-Tester MPI 100 [Phywe Systeme GmbH, Göttingen; for details see Galfe et al. (3)]. The thermode was attached to the lateral dorsum pedis. Vibratory thresholds were assessed by a VIBRA-Tester (Phywe Systeme GmbH, Göttingen). The site for threshold determination was the dorsomedial aspect of the first metatarsal bone. For determination of the pain threshold, 8 heat stimuli were applied with a rate of temperature change of 0.7°C/s, beginning at 38°C. The subjects were instructed to press a button as soon as they felt pain. Each time they pressed the button, the temperature returned to the base value. The pain threshold was calculated as the mean of the peak temperatures of the last 5 stimuli. To measure the warmth and cold threshold, 7 warmth stimuli and then 7 cold stimuli were administered, starting at a temperature of 32°C. The rate of the temperature change was again 0.7°C/s. The subjects had to press a button as soon as they noticed a change in temperature. Thereupon, the temperature returned to the base value. The mean differences between the base temperature and the peak temperature in the 2 sets of 7 trials were the measures of the warmth and cold thresholds. For the assessment of the vibration threshold, the vibration amplitude was increased from zero with a rate of change of 0.2 μm/s until the subject felt the vibration and pressed a button (vibration perception threshold, VPT). There were 3 trials. Then, in another 3 such trials, the vibration amplitude was decreased with the same rate of change from a clearly suprathreshold value until the sensation disappeared (vibration disappearance threshold, VDT). The average of the VPTs and VDTs, measured in the 6 trials, was taken as the vibration threshold (VT). Skin temperature was assessed at the dorsal side of the same foot by a PT-100 sensor in 4 readings, from which the average was taken.

RESULTS

The effectiveness of our diet regimen is demonstrated in Table 1. The body mass index was clearly reduced after 3 weeks in the diet group only (both the factor "session" and the interaction "group" × "session" were highly significant in a two-way MANOVA-analysis, which was computed for all variables). The corresponding mean weight reduction was 3.8 kg. The same clear diet effects were found for the measures T3 and β-HBA with the well-known decrease in T3 and increase in β-HBA after a period of prolonged dieting. However, no major differences

between the sessions were observed for the somatosensory thresholds in either the diet group or the control group. An exception was the pain threshold, where small decreases in both groups resulted in a significant effect of the factor "session"; but the "group" × "session" interaction was not significant even in that case. Significant results could also not be obtained for the skin temperature.

DISCUSSION

The findings of the present study suggest that our diet regimen was effective. The subjects lost approximately 4 kg of their body weight and the indicators of intermittent (β-hydroxybutyric acid) and prolonged (triiodothyronine) fasting became comparable to those observed in our eating disorder patients (see Table 2).

With respect to the main concern of this study, i.e., to clarify whether the reduced pain sensitivity in anorexia and bulimia nervosa is a mere starvation effect, the result was negative. No change of pain sensitivity or of the additional somatosensory variables due to the process of dieting could be demonstrated. The pain thresholds of the dieters, assessed after the regimen, were more similar to those of the nondieting control subjects than to those of the patients in our former study (see Table 2). This validation attempt was necessary because so far no other study has addressed the consequences of dieting or starvation on pain sensitivity in humans. In accordance with our findings, no long-term effects of food restriction on pain sensitivity could be observed in animal studies; only in the onset of a total or partial food deprivation was there a transient reduction of pain sensitivity (1, 4, 8). Therefore, the statement seems to be justified that prolonged dieting is not a sufficient condition to change pain sensitivity. Thus it is very likely that a reduced pain sensitivity is a pathological feature of anorexia and bulimia nervosa which is independent of the dieting state, the cause of which still remains to be clarified. So far, these statements have been proven to be valid for the pain threshold with heat stimuli, which guarantees a nonartificial and only slightly painful stimulation; the validity for, e.g., mechanical or more intense pain stimuli has yet to be tested.

TABLE 2

PAIN THRESHOLDS, T3 AND β-HBA LEVELS IN ANORECTIC AND BULIMIC PATIENTS, IN NONDIETING (ND) CONTROL SUBJECTS [VALUES FROM LAUTENBACHER ET AL. (6)] AND IN DIETING (D) CONTROL SUBJECTS AFTER A THREE-WEEK CALORICALLY REDUCED DIET

	Pain (°C)	T3 (ng/ml)	β-HBA (μmol/ml)
Anorexia nervosa (n = 19)	44.5 ± 2.4	0.92 ± 0.18	0.25 ± 0.64
Bulimia nervosa (n = 20)	44.4 ± 1.5	1.11 ± 0.26	0.25 ± 0.32
Control ND (n = 21)	42.2 ± 1.6	1.48 ± 0.31	0.04 ± 0.05
Control D (n = 11)	43.1 ± 2.0	1.00 ± 0.23	0.38 ± 0.36

Values from the present study (mean ± SD).

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