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DISORDERED EATING BEHAVIOR AND MICROVASCULAR COMPLICATIONS IN YOUNG WOMEN WITH INSULIN-DEPENDENT DIABETES MELLITUS

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ABSTRACT

Background Insulin-dependent diabetes mellitus (IDDM) and eating disorders are relatively common among young women in North America. Their coexistence could lead to poor metabolic control and an increased risk of the microvascular complications of IDDM.

Methods We studied 91 young women with IDDM at base line and four to five years later to determine the prevalence and persistence of disordered eating behavior (on the basis of self-reported eating and weight-loss practices, including the intentional omission or underdosing of insulin to control weight) and the association of such eating disorders with metabolic control, diabetic retinopathy, and urinary albumin excretion. At base line, the mean age of the young women was 15 ± 2 years and the duration of diabetes was 7 ± 4 years.

Results At base line, 26 of 91 young women (29 percent) had highly or moderately disordered eating behavior, which persisted in 16 (18 percent) and improved in 10 (11 percent). Of the 65 women with normal eating behavior at base line (71 percent), 14 (15 percent) had disordered eating at follow-up. Omission or underdosing of insulin to lose weight was reported by 12 of 88 young women (14 percent) at base line and 30 (34 percent) at follow-up ($P=0.003$). At base line, the mean (\pm SD) hemoglobin A_{1c} value was higher in the group with highly disordered eating behavior (11.1 ± 1.2 percent) than in the groups whose eating behavior was moderately disordered (8.9 ± 1.7 percent) or nondisordered (8.7 ± 1.6 percent, $P<0.001$). Disordered eating at base line was associated with retinopathy four years later ($P=0.004$), when 86 percent of the young women with highly disordered eating behavior, 43 percent of those with moderately disordered eating behavior, and 24 percent of those with nondisordered eating behavior had retinopathy.

Conclusions Disordered eating behavior is common and persistent in young women with IDDM and is associated with impaired metabolic control and a higher risk of diabetic retinopathy. (N Engl J Med 1997; 336:1849-54.)

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INSULIN-dependent diabetes mellitus (IDDM) is one of the most common chronic illnesses of childhood and adolescence in North America.¹ Although most young patients with IDDM are healthy, up to 40 percent eventually have diabetes-related microvascular complications.^{2,3} The risk is greater in those whose diabetes is poorly controlled.⁴

Up to one third of young women with IDDM have eating disturbances,⁵⁻⁸ which may affect the management of diabetes. The coexistence of eating disorders and diabetes is associated with noncompliance with treatment for diabetes,^{7,9} omission or underdosing of insulin to induce glycosuria and promote weight loss,^{7,10-13} and impaired metabolic control^{7,8,11,14-17}; however, the long-term effects of disordered eating on complications of diabetes are not known. We performed this study to determine the natural history of disordered eating behavior and its association with microvascular complications in young women with IDDM.

METHODS

Study Design

From June to December 1988 (base line), we invited 121 girls and women 12 to 18 years old, who had previously diagnosed IDDM and were being followed in the diabetes clinic of the Hospital for Sick Children in Toronto, to participate in a self-reported survey of eating attitudes and behavior.^{7,18} This represented all girls and women in this age group who attended the clinic during this period, except for one patient with cerebral palsy. The clinic is the main treatment center for more than 800 children and adolescents with IDDM, providing treatment for 60 to 70 percent of all patients in this age group in metropolitan Toronto. We attempted to contact all the participants four to five years later, be-

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tween July 1992 and January 1994. Approximately one third were still attending the diabetes clinic, and the remainder had been referred to an adult-treatment setting. Our research group did not have contact with the study participants between study entry and follow-up, except that one of us provided routine medical care for some of the patients at the clinic and another saw several patients for psychiatric assessment.

Treatment for patients at the diabetes clinic during this interval included routine quarterly visits and conventional IDDM management in a multidisciplinary setting. It is clinic practice to recommend a psychosocial assessment for patients with persistently elevated hemoglobin A_{1c} values or disturbed eating attitudes and behavior. Twenty-one patients reported that during the follow-up interval they had been assessed or treated for one or more of the following: an eating disorder (nine patients), depression (nine patients), family difficulties (two patients), and other psychosocial problems (four patients). At follow-up, patients with a possible eating disorder were offered referral. The study protocol was approved by the scientific and human-subjects review committees at the University of Toronto and Toronto Hospital. Informed, written consent was obtained from each subject or from a parent, for those less than 16 years of age.

At base line and follow-up, demographic and clinical information was collected, height and weight were measured, and body-mass index was calculated. Self-reported episodes of ketoacidosis and severe hypoglycemia in the preceding year were documented. Behavior related to eating and weight psychopathology was assessed at base line and follow-up with the Diagnostic Survey for Eating Disorders.¹⁹ This self-administered questionnaire, which elicits information about eating habits during the past three months, was modified to include diabetes-related items, including omission or underdosing of insulin to promote weight loss. The patients either completed the questionnaire during their clinic visits or completed it at home and returned it by mail. Despite reminder calls, some questionnaires (eight at base line and nine at follow-up) were not returned.

Hemoglobin A_{1c} was measured at base line and follow-up by high-performance liquid chromatography after removal of the labile fraction (normal range, 4 to 6 percent). The intraassay and interassay coefficients of variation were 2.3 and 2.9 percent, respectively.²⁰

The urinary albumin excretion rate, a predictor of the risk of diabetic nephropathy, was determined at follow-up in both 1- and 24-hour urine collections by double-antibody radioimmunoassay (Pharmacia, Uppsala, Sweden)²¹ and calculated from the albumin concentration and the volume and duration of the urine collections. Microalbuminuria was defined as an albumin excretion rate of at least 15 but less than 200 μg per minute, and macroalbuminuria as a rate of at least 200 μg per minute.²¹ If only one measurement was available (18 percent of the patients), it was used to make the classification. The values for the two samples were discrepant in 18 percent of the patients; in these cases, the results from the 24-hour collection were taken as the more reliable measurement.²²

Assessment of Retinopathy

Diabetic retinopathy was detected at follow-up by indirect ophthalmoscopy and slit-lamp biomicroscopy (after pupillary dilation) and grading of seven-field stereoscopic color fundus photographs by a retinal specialist who did not know the patient's eating habits, hemoglobin A_{1c} value, or urinary albumin excretion rate. Retinopathy was graded on the basis of the fundus photographs according to an extension of the modified Airlie House classification,^{23,24} as in the Diabetes Control and Complications Trial.⁴ The level of retinopathy was derived by giving greater weight to the eye with the higher level. With this classification, level 10 indicates no diabetic retinopathy; level 20, very mild retinopathy (microaneurysms, but no other lesions); level 30, mild nonproliferative retinopathy (microaneurysms plus one or more of the following: retinal hemorrhage, hard exudates, soft exudates

possibly present, intraretinal microvascular abnormalities possibly present, or venous beading possibly present); levels 40 to 55, moderate-to-severe nonproliferative retinopathy (microaneurysms plus one or more of the following: soft exudates, hard exudates, intraretinal microvascular abnormalities, or venous beading definitely present); and level 60 or higher, mild-to-high-risk proliferative retinopathy (including proliferation of new vessels, macular edema, photocoagulation scars, and preretinal or vitreous hemorrhages).

Classification of Eating Behavior

Eating behavior at base line and follow-up was categorized on the basis of data from the Diagnostic Survey for Eating Disorders.¹⁹ Three mutually exclusive, hierarchical categories were used. Highly disordered eating was defined as the occurrence of one or more of the following forms of disordered behavior at least twice per week during the preceding three months: binge eating, omission or underdosing of insulin to promote weight loss, self-induced vomiting, or use of laxatives. Moderately disordered eating was defined as the occurrence of one or more of these forms of disordered behavior at least twice per month, but less than twice per week, during the preceding three months. Nondisordered eating was defined as the absence of disordered behavior or its occurrence less than twice per month during the preceding three months.

Statistical Analysis

Statistical analysis was performed with the Statistical Package for the Social Sciences,²⁵ except for the repeated-measures analysis of variance, which was performed with the BMDP statistical program.²⁶ The results for eye examinations and urinary albumin excretion were dichotomized into normal and abnormal. For the purposes of data analysis, any diabetic retinopathy was defined as mild retinopathy (level 20)²⁴ or worse. Paired and independent t-tests were used for two-group comparisons of continuous variables. Comparisons of participants with nonparticipants (those participating at base line but not at follow-up), and of mean hemoglobin A_{1c} values according to disordered-eating status at base line, were made with one-way analyses of variance for continuous variables and chi-square analyses for data presented as proportions. Chi-square analyses with Yates' correction for continuity were used to test the association of disordered-eating status at base line with microvascular complications and disordered-eating status at follow-up (persistence). Fisher's exact test was used to test the hypothesis regarding the persistence of disordered behavior. The association between the persistence of disordered-eating status and metabolic control was tested with a repeated-measures analysis of variance examining the interaction of group and time. McNemar's test for paired proportions (with the chi-square statistic) was used to test for increased prevalence of disordered-eating symptoms from base line to follow-up, against the null hypothesis of equal prevalence at each time. Stepwise discriminant-function analyses (with the minimization of Wilks' lambda criterion) were used to predict the presence or absence of complications at follow-up. All statistical tests were two-tailed.

RESULTS

One hundred seven (88 percent) of the 121 eligible girls and women participated at base line, 8 did not return their questionnaires, and 6 refused to participate. Ninety-one (85 percent) of these 107 girls and women participated at follow-up, 2 refused to participate, 9 did not return their questionnaires, and 5 could not be located. The mean (\pm SD) follow-up interval was 4.4 ± 0.3 years (range, 3.7 to 5.3). The characteristics of the patients at base line and follow-up are shown in Table 1.²⁷

TABLE 1. CHARACTERISTICS OF 91 YOUNG WOMEN WITH IDDM STUDIED AT BASE LINE AND FOLLOW-UP.*

CHARACTERISTIC	BASE LINE	FOLLOW-UP
Age (yr)		
Mean	15±2	19±2
Range	12–18	16–22
Age at onset of IDDM (yr)		
Mean	8±4	—
Range	0.3–16	—
Duration of IDDM (yr)		
Mean	7±4	11±4
Range	0.6–16	4–20
Unmarried (%)	100	93
Living with parents (%)	100	75
Employment status (%)†		
Student	100	82
Working part time	—	37
Working full time	—	8
Homemaker	—	3
Unemployed	—	2
Body weight (kg)‡		
Mean	56.5±10.2	64.4±11.5
Range	36–98	40–116
Body-mass index‡§		
Mean	22.3±3.1	24.7±3.7
Range	16.4–33.1	17.2–38.7
Hemoglobin A _{1c} (%)‡		
Mean	9.0±1.7	8.7±1.9
Range	4.9–13.4	4.8–14.2
Injections of insulin per day (%)		
1	20	13
2	78	75
3	0	7
4	2	4
5	0	1

*Plus-minus values are means ±SD. IDDM denotes insulin-dependent diabetes mellitus.

†Employment status was not assessed at base line; all the girls and women were students (range, grade 6 to college or university). The total percentage was greater than 100 at follow-up because some patients were also working part time.

‡Fourteen patients refused follow-up medical evaluations, which included measurements of height, weight, and hemoglobin A_{1c}; the follow-up values therefore reflect data on 77 patients.

§Body-mass index was calculated as the weight in kilograms divided by the square of the height in meters. The normal range (2nd to 98th percentiles) is 14.0 to 28.5 in adolescent girls and young women 12 to 18 years of age, and 16.0 to 29.5 for those 16 to 22 years of age.²⁷

The 16 patients who participated at base line but not at follow-up did not differ from the 91 who completed both assessments, in terms of age, age at onset of IDDM, duration of IDDM, hemoglobin A_{1c} values, body-mass index, and eating status at base line. Among the 11 patients who refused to participate or who failed to return their questionnaires at follow-up, 1 was in the group with highly disordered eating at base line and 2 were in the group with moderately disordered eating. Among the five patients lost to follow-up, one was in the group with highly disordered eating at base line.

Prevalence and Persistence of Disordered Eating Behavior and Disordered-Eating Status

The prevalence and persistence of disordered eating behavior are shown in Table 2. Intentional omission or underdosing of insulin and dieting for weight loss increased in prevalence from base line to follow-up. Binge eating, self-induced vomiting, and dieting for weight loss tended to persist at follow-up if they were present at base line.

At base line, 9 of the 91 young women met the criteria for highly disordered eating, 17 met the criteria for moderately disordered eating, and 65 met the criteria for nondisordered eating. The nine patients with highly disordered eating did not differ from the others in age, but they had a longer mean duration of diabetes (9±4 vs. 6±4 years, $P=0.03$). Disordered-eating status tended to persist over time ($P=0.007$): of the 26 patients with highly or moderately disordered eating at base line, 16 remained in these categories and 10 improved. Of the 65 patients with nondisordered eating at base line, 14 had disordered eating at follow-up.

Disordered-Eating Status and Metabolic Control

At base line, the patients with highly disordered eating had a significantly higher mean hemoglobin A_{1c} value (11.1±1.2 percent) than those in the groups with moderately disordered eating (8.9±1.7 percent) and nondisordered eating (8.7±1.6 percent) ($P<0.001$). Among the 14 patients who had persistently disordered eating behavior and whose hemoglobin A_{1c} values were measured at follow-up, the values were similarly high at both assessments (9.5±1.8 and 9.9±2.2 percent). Among the nine patients who had improved eating status and whose hemoglobin A_{1c} values were measured at follow-up, the mean value decreased from 9.7±2.2 to 7.6±1.4 percent ($P=0.002$). With a repeated-measures analysis of variance examining the interaction of group and time, there was a significant association between disordered-eating status and metabolic control measured at base line and four years later ($P=0.01$).

Of the 77 patients for whom there were data regarding ketoacidosis and severe hypoglycemia in the year preceding follow-up, 6 reported one to three episodes of ketoacidosis and 14 reported one to four episodes of hypoglycemia. One patient, who reported 20 episodes of hypoglycemia, was in the group with highly disordered eating at follow-up. Chi-square analyses revealed no significant differences between the disordered-eating groups with respect to either variable.

Diabetes-Related Microvascular Complications at Follow-up

Diabetic Retinopathy

Seventy-one of the 91 young women had ophthalmologic examinations (including fundus pho-

TABLE 2. PREVALENCE AND PERSISTENCE OF DISORDERED EATING BEHAVIOR IN YOUNG WOMEN WITH IDDM.

BEHAVIOR*	BASE LINE	FOLLOW-UP	BOTH TIMES	P VALUE FOR PREVALENCE†	P VALUE FOR PERSISTENCE‡
	number (percent)				
Binge eating (n=87)	39 (45)	48 (55)	31 (36)	0.11	<0.001
Omission or underdosing of insulin for weight loss (n=88)	12 (14)	30 (34)	5 (6)	0.003	0.53
Self-induced vomiting (n=89)	7 (8)	15 (17)	4 (4)	0.06	0.01
Laxative use (n=88)	2 (2)	7 (8)	1 (1)	0.13	0.15
Dieting for weight loss (n=90)	34 (38)	49 (54)	26 (29)	0.01	0.002

*Only patients who responded to each item on both the base-line and follow-up questionnaires were included in each analysis.

†The P values are for the comparison of the prevalence of each form of behavior between base line and follow-up, by McNemar's test.

‡The P values are for the persistence of disordered eating behavior, by Fisher's exact test.

tography) at follow-up; 24 of the 71 were found to have some degree of retinopathy. Sixteen had mild retinopathy, with 1 to 11 microaneurysms per eye. Among the eight patients with more severe retinopathy, six had nonproliferative retinopathy (three with hard exudates and two with venous beading); one had advanced preproliferative retinopathy in one eye and early proliferative retinopathy in the other; and one had proliferative retinopathy and macular edema bilaterally, for which she received laser therapy.

Urinary Albumin Excretion

Urinary albumin excretion was measured in 72 of the 91 patients at follow-up. Twelve had microalbuminuria (range, 15 to 66 μg per minute), and three had macroalbuminuria (range, 222 to 427 μg per minute).

Of the 74 patients for whom there were medical data at follow-up, 30 had evidence of at least one complication of diabetes. These 30 patients were slightly older than the 44 patients without complications at follow-up (20 ± 2 vs. 19 ± 2 years, $P=0.04$), had a longer mean duration of diabetes (12 ± 4 vs. 10 ± 4 years, $P=0.02$), and had a higher mean hemoglobin A_{1c} value at base line (9.6 ± 1.6 percent vs. 8.4 ± 1.6 percent, $P=0.002$).

Disordered-Eating Status at Base Line and Microvascular Complications at Follow-up

The association between disordered-eating status at base line and diabetes-related microvascular complications at follow-up is shown in Table 3. Retinopathy was significantly more common in patients with disordered eating at base line ($P=0.004$), but abnormal urinary albumin excretion was not ($P=0.32$).

Predictors of Microvascular Complications at Follow-up

Stepwise discriminant-function analyses were used to predict the presence or absence of retinopathy and abnormal urinary albumin excretion at follow-up. We controlled for the duration of diabetes by entering this variable in the first step. We accounted for 22 percent of the variance in predicting retinopathy ($P=0.003$) with the following predictor variables measured at base line (in order of inclusion in the stepwise model): duration of diabetes (which accounted for 5 percent of the variance), highly disordered eating (an additional 10 percent), the hemoglobin A_{1c} value (an additional 5 percent), and moderately disordered eating (an additional 2 percent). Duration of diabetes accounted for all 7 percent of the explained variance in predicting abnormal urinary albumin excretion ($P=0.03$). Hemoglobin A_{1c} values and disordered-eating status were not significant predictors of abnormal urinary albumin excretion.

DISCUSSION

The most striking finding of this study is that some degree of retinopathy was present at follow-up in more than 85 percent of young women with IDDM who had highly disordered eating at base line, as compared with 43 percent of those with moderately disordered eating and only 24 percent of those with nondisordered eating. Furthermore, disordered-eating status accounted for more of the explained variance in a model predicting retinopathy than did duration of diabetes, an established risk factor for microvascular complications.⁴ The mean duration of diabetes in these patients (11 ± 4 years)

TABLE 3. DISORDERED-EATING STATUS AT BASE LINE AND DIABETES-RELATED MICROVASCULAR COMPLICATIONS AT FOLLOW-UP.

DISORDERED-EATING STATUS AT BASE LINE	DIABETIC RETINOPATHY AT FOLLOW-UP*	ABNORMAL URINARY ALBUMIN EXCRETION AT FOLLOW-UP†
	no. with complication/total no. (%)	
Highly disordered	6/7 (86)	3/7 (43)
Moderately disordered	6/14 (43)	3/15 (20)
Nondisordered	12/50 (24)	9/50 (18)

*Seventy-one of the 91 patients had ophthalmologic examinations at follow-up. $P=0.004$ by the chi-square test for the association of disordered-eating status at base line and diabetic retinopathy at follow-up.

†Seventy-two of the 91 patients had measurements of urinary albumin excretion at follow-up. $P=0.32$ by the chi-square test for the association of disordered-eating status at base line and abnormal urinary albumin excretion at follow-up.

may not have been long enough for incipient nephropathy to occur in enough patients to reveal an association with disordered-eating status. The limitations of our study include incomplete participation in the follow-up medical evaluations, assessment of eating behavior on only two occasions four to five years apart, limited established reliability of the self-report measure, and absence of base-line evaluations of microvascular complications.

We have confirmed that in young women with diabetes, eating disturbances tend to persist, to increase in frequency through adolescence, and to be highly predictive of poor metabolic control and subsequent complications. The increased prevalence of behavior designed to foster weight loss at follow-up is not surprising, because more of the patients had reached the age of higher risk for eating disturbances. Apart from dieting to lose weight, intentional omission or underdosing of insulin (reported by one third of the patients at follow-up) was the most common means of inducing weight loss. The availability of this method of preventing weight gain or promoting weight loss may account for the relatively low prevalence of other purging behaviors in our sample.^{7,12,13}

Prevention and early treatment of eating disorders in young women are important to prevent long-term morbidity and mortality. The health risk of these conditions is increased when they are associated with diabetes, because of their effect on metabolic control and microvascular complications. Diabetic women with eating disorders may present with less well recognized features of an eating disturbance,²⁸ including noncompliance with treatment for diabetes,^{7,9} unstable metabolic control,^{6-8,15-17,29} hyperglycemia and recurrent ketoacidosis,²⁹ and an earlier onset of microvascular complications.^{6,30,31} As compared with women who are seen in psychiatric settings, diabetic

women with eating disorders may have less florid symptoms and are more likely to be of normal weight or overweight, rather than underweight.³² Our study suggests that routine screening for eating disturbances may be indicated in young women with diabetes and that some intervention should be undertaken, if eating disorders are detected, to minimize the risk of later complications.^{33,34}

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REFERENCES

- Drash AL. The epidemiology of insulin-dependent diabetes mellitus. *Clin Invest Med* 1987;10:432-6.
- Krolewski AS, Warram JH, Christlieb AR, Busick EJ, Kahn CR. The changing natural history of nephropathy in type I diabetes. *Am J Med* 1985;78:785-94.
- Krolewski AS, Warram JH, Rand LI, Christlieb AR, Busick EJ, Kahn CR. Risk of proliferative diabetic retinopathy in juvenile-onset type I diabetes: a 40-yr follow-up study. *Diabetes Care* 1986;9:443-52.
- The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* 1993;329:977-86.
- Rodin GM, Johnson LE, Garfinkel PE, Daneman D, Kenshole AB. Eating disorders in female adolescents with insulin dependent diabetes mellitus. *Int J Psychiatry Med* 1986-87;16:49-57.
- Steel JM, Young RJ, Lloyd GG, Clarke BF. Clinically apparent eating disorders in young diabetic women: associations with painful neuropathy and other complications. *BMJ* 1987;294:859-62.
- Rodin G, Craven J, Littlefield C, Murray M, Daneman D. Eating disorders and intentional insulin undertreatment in adolescent females with diabetes. *Psychosomatics* 1991;32:171-6.
- Vila G, Nollet-Clemencon C, Vera L, Crosnier H, Robert J-J, Mouren-Simeoni M-C. Étude des troubles des conduites alimentaires dans une population d'adolescentes souffrant de diabète insulino-dépendant. *Can J Psychiatry* 1993;38:606-10.
- Wing RR, Nowalk MP, Marcus MD, Koeske R, Finegold D. Subclinical eating disorders and glycemic control in adolescents with type I diabetes. *Diabetes Care* 1986;9:162-7.
- Stancin T, Link DL, Reuter JM. Binge eating and purging in young women with IDDM. *Diabetes Care* 1989;12:601-3.
- Fairburn CG, Peveler RC, Davies B, Mann JI, Mayou RA. Eating disorders in young adults with insulin dependent diabetes mellitus: a controlled study. *BMJ* 1991;303:17-20.
- Polonsky WH, Anderson BJ, Lohrer PA, Aponte JE, Jacobson AM, Cole CF. Insulin omission in women with IDDM. *Diabetes Care* 1994;17:1178-85.
- Biggs MM, Basco MR, Patterson G, Raskin P. Insulin withholding for weight control in women with diabetes. *Diabetes Care* 1994;17:1186-9.
- Szmukler GI, Russell GFM. Diabetes mellitus, anorexia nervosa and bulimia. *Br J Psychiatry* 1983;142:305-8.
- Birk R, Spencer ML. The prevalence of anorexia nervosa, bulimia, and induced glycosuria in IDDM females. *Diabetes Educ* 1989;15:336-41.
- Steel JM, Young RJ, Lloyd GG, Macintyre CCA. Abnormal eating attitudes in young insulin-dependent diabetics. *Br J Psychiatry* 1989;155:515-21.
- Friedman S, Vila G, Timsit J, Boitard C, Mouren-Simeoni MC. Troubles des conduites alimentaires et équilibre métabolique dans une population de jeunes adultes diabétiques insulino-dépendants. *Ann Med Psychol* 1995;153:282-5.
- Littlefield CH, Craven JL, Rodin GM, Daneman D, Murray MA, Ry-

- dall AC. Relationship of self-efficacy and bingeing to adherence to diabetes regimen among adolescents. *Diabetes Care* 1992;15:90-4.
19. Johnson C. Initial consultation for patients with bulimia and anorexia nervosa. In: Garner DM, Garfinkel PE, eds. *Handbook of psychotherapy for anorexia nervosa and bulimia*. New York: Guilford Press, 1985:19-51.
20. Ellis G, Diamandis EP, Giesbrecht EE, Daneman D, Allen LC. An automated "high-pressure" liquid-chromatographic assay for hemoglobin A_{1c}. *Clin Chem* 1984;30:1746-52.
21. Sochetti E, Daneman D. Screening tests to detect microalbuminuria in children with diabetes. *J Pediatr* 1988;112:744-8.
22. McKenna MJ, Arias C, Feldkamp CS, Whitehouse FW. Microalbuminuria in clinical practice. *Arch Intern Med* 1991;151:1745-7.
23. Diabetic Retinopathy Study Research Group. Report 7: a modification of the Airlie House classification of diabetic retinopathy. *Invest Ophthalmol Vis Sci* 1981;21:210-26.
24. Early Treatment Diabetic Retinopathy Study Research Group. Grading diabetic retinopathy from stereoscopic color fundus photographs — an extension of the modified Airlie House classification: ETDRS report number 10. *Ophthalmology* 1991;98:Suppl:786-806.
25. Norusis MJ, ed. *SPSS-PC+ base system user's guide, version 5.0*. Chicago: Statistical Package for the Social Sciences (SPSS), 1992.
26. Dixon WJ, ed. *BMDP statistical software manual*. Vol. 1. Berkeley: University of California Press, 1988.
27. Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. *Arch Dis Child* 1995;73:25-9.
28. Rodin GM, Daneman D. Eating disorders and IDDM: a problematic association. *Diabetes Care* 1992;15:1402-12.
29. Hudson JI, Hudson MS, Wentworth SM. Self-induced glycosuria: a novel method of purging in bulimia. *JAMA* 1983;249:2501.
30. Colas C, Mathieu P, Tehobrousky G. Eating disorders and retinal lesions in Type I (insulin-dependent) diabetic women. *Diabetologia* 1991;34:288.
31. Ward A, Troop N, Cachia M, Watkins P, Treasure J. Doubly disabled: diabetes in combination with an eating disorder. *Postgrad Med J* 1995;71:546-50.
32. Fairburn CG, Welch SL, Norman PA, O'Connor ME, Doll HA. Bias and bulimia nervosa: how typical are clinic cases? *Am J Psychiatry* 1996;153:386-91.
33. Peveler RC, Fairburn CG. The treatment of bulimia nervosa in patients with diabetes mellitus. *Int J Eat Disord* 1992;1:45-53.
34. Lawson ML, Rodin GM, Rydall AC, Olmsted MP, Daneman D. Eating disorders in young women with IDDM: the need for prevention. *Eat Disord J Treat Prev* 1994;2:261-72.