Family Functioning and Metabolic Control of School-Aged Children With IDDM

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The relationship of two aspects of family life to metabolic control were examined as part of a longitudinal study of school-aged children with newly diagnosed insulin-dependent diabetes mellitus (IDDM). Glycosylated hemoglobin level was the primary index of metabolic control; weight-adjusted insulin dosage served as an indirect index. Neither the quality of family life nor aspects of the parents' marriage predicted the child's metabolic control over the next 3–4 mo, and they were also unrelated to concurrent weight-adjusted insulin dosage. Longitudinal data spanning a 6-yr period of the child's diabetes also failed to reveal an association between aspects of family life and metabolic control. The significance of the findings are discussed in light of the sample's characteristics and possible methodological constraints. *Diabetes Care* 12:409–14, 1989

The family life of children with insulin-dependent diabetes mellitus (IDDM) has been considered to be important in the management and course of the illness (1,2). There has been particular interest in how the psychological functioning of the family and the marital relationship of the parents affect children's metabolic control. A well-functioning family can facilitate a child's well-being by providing emotional support, advice, and practical help (3,4). A good marital relationship can increase the likelihood that family resources and attention are focused on the offspring with IDDM as needed (3). Theoretically, family problems or marital distress could deleteriously affect metabolic control of the diabetic child by interfering with self-care or by causing stress-related physiological dysregulation (5).

There is clinical literature that appears to document the effects of family characteristics on diabetic control (for review, see refs. 2,3,6,7). However, much of this literature, including the only longitudinal study of this issue (8), has been criticized on methodological grounds. For instance, it has been noted that family characteristics were typically ascertained by subjective or clinical judgment and that the judges of family life were not blind to the children's medical status (2).

Recent studies have used improved research designs, but the results have not been conclusive. For example, Anderson et al. (9) reported that as rated by parents, only one dimension of a multiscale family measure differentiated children in good and poor control, whereas Waller et al. (10) found that several diabetes-specific family dimensions were related to control. Moreover, in one study, cohesive, emotionally expressive, and conflict-free families had children in better metabolic control than problem families (11). However, in another study, the more rigid families had children with better control than the highly adaptive families (12). Such discordant findings may partly reflect the fact that various instruments were used to assess family life. In addition, medical and demographic variables, which may influence metabolic control, were not uniformly taken into account by different studies. Moreover, patients and families were typically assessed on only one occasion, which precluded the examination of predictive hypotheses or time-dependent aspects of family life and metabolic control.

Because we initiated in 1978 a naturalistic longitudinal study of a sample of school-aged children with

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newly diagnosed IDDM, we have been in the position to monitor temporally evolving relationships between certain psychosocial factors and aspects of the medical disorder. Here we report on the interface between family life and metabolic control. To examine this problem area, we posed two research questions: *1*) Does the quality of family environment predict metabolic control? *2*) Does the quality of family environment and metabolic control covary over the years?

MATERIALS AND METHODS

Subjects. Our sample was recruited from among consecutive admissions to the Pediatric Endocrinology inpatient unit of the Children's Hospital of Pittsburgh (CHP) who agreed to participate in the study and met the following inclusionary criteria: newly diagnosed with classic acute-onset ketosis-prone IDDM, no evidence of mental retardation, no major systemic illness other than IDDM, 8–13 yr of age, and living with parent(s) or legal guardian(s) within commuting distance of Pittsburgh. The age cutoff was dictated by the nature of several instruments used in the study, which were not part of the analyses for this article.

Based on data for 1982 and 1983, 76% of suitable families were successfully recruited. Patients who consented and those who refused did not significantly differ in sex distribution, age, race, and caretaker arrangement (1- vs. 2-parent households). Signed consent forms were obtained from parent(s) and children for an initial 5-yr follow-up and, on its completion, for a second 5 yr. Families received a monetary reimbursement for expenses at each assessment.

We report on the first 85 children in our sample, the first of whom entered the study in May 1978, and the last in July 1984. At study entry, children's ages ranged from 8.3 to 13.9 yr (mean 11.1 yr). There were 48 girls and 37 boys; most were White (93%) and the remainder were Black (7%). Ten cases dropped out, and one died before completing the first phase of the project (follow-up range 0–47 mo). Because the cases were accumulated over a span of 6 yr, follow-up for the nondropouts ranged from 27 mo (entered in 1984) to 97 mo (entered in 1978) by the November 1986 cutoff used in these analyses.

Assessment schedule and definition of variables. The first research evaluation (intake) took place 2–3 wk after the child's discharge from the hospital for IDDM diagnosis. In the 1st yr of study participation, there were three to four additional research evaluations; subsequently, the protocol specified one assessment approximately every 8–10 mo. As is usual with longitudinal studies, the actual number of and time intervals between assessments varied among the cases.

At each assessment, demographic and psychosocial data were gathered by research clinicians in interviews with the parents. Sociodemographic information was elicited according to a standardized general information sheet (GIS). Life events for the children were recorded as per Coddington's scales (13). Parents completed self-rated questionnaires independent of the clinicians' evaluations.

Family functioning. Two aspects of family functioning were assessed: *1*) parental perception of overall quality of family life and *2*) quality of the parents' marriage.

The Family Concept Inventory of van der Veen and Olson (14) was used to assess overall quality of family life. The construct of Family Effectiveness (FAMLIFE) is quantified by a subset of 48 of the 80 items on the inventory. The resultant score, which can range from a low of 0 to a high of 48 (reflecting a very effective family), mirrors the extent to which, in the respondent's view, his/her family contains qualities that professional clinicians consider to be important for a good family life. Thus, the overall score taps areas such as openness of communication, ability to resolve conflict, family loyalty and satisfaction, and closeness of familial relations (e.g., "We can usually depend on each other," and "We can adjust well to new situations") (14). Because with time, mothers predominated as informants, their reports served as data for this article.

As summarized by van der Veen and Olson (14), the FAMLIFE score has been shown to have excellent test-retest reliability (r = .87 and .94) in studies with a 4-wk intertest interval. It correlates with overall levels of marital adjustment and children's socialization, can differentiate families with well-adjusted versus disturbed children, and discriminates between families with children exhibiting aggressive versus withdrawn behavior (14).

To assess the internal consistency of the FAMLIFE scale in our sample, we selected the first test for each parent. As reflected by coefficient α, the scale had high internal consistency (for 82 mothers, α = .90 and for 57 fathers, α = .94) (15). To estimate parental agreement on family perception, we selected the first assessment when both parents of a child completed the scales. Scores of mother-father pairs were significantly correlated (r = .46, P = .001, n = 49).

To assess the quality of the parents' marriage, four variables were used; one variable was abstracted from the GIS, and three were derived from the Coddington life-events scales. The GIS item on current marital problems/strife among caretaking parents was coded as 0, does not apply (single-parent household); 1, no strife; 2, yes, as before or somewhat less; and 3, yes, new discord or definite exacerbation of previous strife.

To estimate the validity of this item as an index of marital problems, we examined its relationship to the independently completed Locke Wallace Short Marital-Adjustment Questionnaire (16). For 67 mothers in our cohort, we found a correlation of −.71 (P < .0001) between the two indexes; for 46 fathers, the correlation was −.43 (P < .003). Because this questionnaire was introduced into our assessment several years after the

study started, these data were not available at many evaluation points, thereby precluding their use in these analyses.

Additional information about marital adjustment was provided by three items from the Coddington life-events scales (i.e., presence or absence of increased arguments between parents, separation of parents, and divorce of parents). At study intake, these data covered the preceding year; at each follow-up, the data referred to the time interval since the last evaluation.

Metabolic control. As part of the diabetic clinic visits, scheduled at ~4-mo intervals, medical and laboratory data were collected independently by clinic staff who were blind to the results of our research assessments. Two variables were extracted from this data base: *1*) percentage of glycosylated hemoglobin in whole blood (HbA_1) and *2*) current insulin dosage and weight to compute weight-adjusted insulin dosage.

HbA_1 reflects the average level of blood glucose over the 3 mo or so preceding the day of sampling and has been found to be the most useful clinical index of metabolic control (17). The assaying method at CHP has been described elsewhere (18). In this laboratory, HbA_1 for normal children ranged from 4.9 to 7.3% (mean ± SD 6.1 ± 0.6%); for 622 diabetic clinic cases, HbA_1 figures were 11.6 ± 2.1% (D. Becker, personal communication; 18).

In previous analyses of our HbA_1 data, we found a relationship between the calendar year during which the blood was sampled and control. Since 1979, when these assays became routine, mean HbA_1 levels have declined (for cases in 1st yr with IDDM entering the study between 1979 and 1985, $F[6,252] = 4.92$, $P < .0001$). We also found a tendency for HbA_1 to increase during adolescence and then decline. This suggests a quadratic relationship of HbA_1 and age. Therefore, both age and age squared (allowing for quadratic relationship) are used as covariates in the longitudinal analyses of HbA_1 data.

We also computed for each clinic visit each child's weight-adjusted insulin dosage (total units of insulin divided by weight in kilograms). This variable served as an alternative index of control to account for the fact that, in clinical practice, one response to high HbA_1 is to increase the patient's insulin dosage.

Statistical analyses. To answer the first research question of whether family variables predict metabolic control, the data were tabulated by levels of independent and dependent variables. The distributions (e.g., number of cases by levels of family effectiveness and metabolic control) were examined by χ^2-tests. Then, regression analysis was used to relate the independent family variable (with or without covariates) and the outcome of interest (e.g., HbA_1).

The second research question necessitated longitudinal data analysis. This was accomplished by means of a modification of regression analysis that may be used with repeated measures even when they vary in number across the cases and are unequally spaced over time (19). For our data, we used the simplest form of this technique, referred to as the random-intercepts model, in which the average level of HbA_1 is allowed to depend on the individual. The results of the analyses are coefficients and their standard errors, which are interpreted in the same way as those in linear regression.

RESULTS

Family characteristics of the cohort. Most children were living in intact families at study entry, were of middle-class backgrounds, and had a working head of household (Table 1). The average family size was consistent with national norms (20).

At study entry, there was no significant association between the FAMLIFE score and children's socioeconomic status (SES), sex, age, race, or household size (r from −.02 to .22, $n = 70$; reduced n due to missing intake scores). Quality of family life, as perceived by mothers, was comparable to that reported for families with a handicapped child but fell short of the scores reported for controls. That is, our mothers' initial mean ± SD FAMLIFE score was 35.5 ± 8.7, whereas van der Veen and Olson (14) reported a score of 36 ± 8.5 for mothers of 123 handicapped children. For non-

TABLE 1
Characteristics of families at entry into study

Variable	Statistic*
Family composition (%)	
Intact (both biological parents present)	77
Biological and stepparent	7
Adoptive parents	2
Single parent/grandparent	13
Other relative(s)	1
People in household (*n*)	4.8 (2–11)
Socioeconomic status of head of household by Hollingshead index (%)	
1 (Highest)	9
2–4	84
5	7
Employed head of household (%)	91
Parents' ages (yr)	
Mother/mother surrogate	38 ± 6.1
Father/father surrogate	40 ± 6.7
Family Effectiveness score†	35.5 ± 8.7
Current marital distress (%)‡	26
Divorced in past year (%)	2
Separated in past year (%)	5
Increased parental arguments in past year (%)	11

$n = 85$. Values are means ± SD, and range is in parentheses.
*On psychometric family data, percentages adjusted for missing information.
†Mothers as respondents.
‡Excluding single-parent homes.

clinic control mothers (n = 99), van der Veen and Olson (14) reported a score of 39.5 ± 5.8.

To evaluate whether family effectiveness and parental marriage remained stable, we compared intake information with figures at two subsequent assessment points, covering approximately the initial 2.5 yr of IDDM. FAMLIFE did not appreciably change over that time interval. For example, at 24–30 mo after intake, the mean score was still 35.5 ± 9.7. Likewise, marital distress was consistently reported by ~30% of the sample (for baseline data, see Table 1). Subsequent to intake, only 3% more of the remaining couples divorced, and 13% reported increased arguments at the 24- to 30-mo point. Such cross-sectional examination of the data was not practical for later years because, with time, many cases were off schedule by >3 mo.

Do family variables predict metabolic control? To answer this research question, we excluded the data covering the 1st yr with IDDM because of the likelihood of partial pancreatic β-cell functioning or honeymooning (21). We reasoned that a test of psychosocial factors as predictors of metabolic control would be confounded by honeymooning during which exogenous events may be less potent in their effects. Then, we approached the question from two perspectives.

First, we sought to assess whether family variables at a given time predicted subsequent HbA_1. Therefore, we selected for each case the first (time 1) FAMLIFE score and the indexes of parental marriage that could be paired with an HbA_1 assay 60–140 days after that assessment (time 2). Second, as an alternative approach, we sought to determine the association between weight-adjusted insulin dosage and contemporaneous family factors. Thus, we selected for each subject the first assessment point for family data along with the corresponding weight-adjusted insulin dosage from the clinic visit on or near (±17 days) the selected research assessment. Note that for either strategy, cases were deleted from the analysis for whom data could not be paired within the specified interval (e.g., FAMLIFE with later HbA_1 and FAMLIFE with concurrent weight-adjusted insulin dosage).

The paired data were first subjected to preliminary analyses. Specifically, the sample was divided into low, medium, and high groups with respect to HbA_1, weight-adjusted insulin dosage, and FAMLIFE in such a way that ~33% of the subjects fell into each group. In addition to cross-tabulations with FAMLIFE scores, distributions were also derived for whether there were increased arguments among the parents. Because of the low rates of separation and divorce in our study, these variables were not used in cross-tabulations.

Children with low, medium, or high levels of HbA_1 (≤11.7, 11.8–12.9, and ≥13%, respectively) were similarly distributed across the three categories of FAMLIFE, indicating no evidence of an association (χ^2 = 6.3, df = 4, $P > .10$). For example, of the 20 cases who had very effective families (scores ≥42) at the initial time point, 35% had low but 20% had high levels of HbA_1 3–4 mo later. Of 20 cases with poorly functioning families at time 1, 40% had low and 20% had high levels of HbA_1 at time 2. (We are aware that the obtained cutoff for low HbA_1 does not approximate clinically desirable values.) We also found no association between levels of metabolic control and marital problems (χ^2 = 6.7, df = 6, $P > .30$). For instance, 29% (10 of 35) of children whose parents did not have marital problems had high HbA_1 (poor control) later on, whereas 28% (5 of 18) of the cases whose parents had marital problems had high levels of HbA_1. The results were similar regarding the role of parental arguments in metabolic control (χ^2 = 0.45, df = 2, $P > .70$).

The distribution of cases by levels of FAMLIFE, marital problems, and levels of weight-adjusted insulin dosage was also examined (χ^2 = 0.5, df = 4, $P > .95$ and χ^2 = 4.7, df = 6, $P > .40$, respectively). Insulin dosage levels were not found to be associated with either psychometric or self-reported indexes of family life. For example, a high weight-adjusted insulin dose (≥1.01 U/kg) was just as likely to be associated with ineffective or highly effective families (36 and 32% of high weight-adjusted insulin-dosage cases, respectively).

We also used regression analyses to assess the relationship between our predictors and the quantitative (as opposed to categorical) distributions of our medical outcome measures and to take into account the possible effects of demographic factors. No relationship was found between FAMLIFE at time 1 and HbA_1 several months later or between FAMLIFE and weight-adjusted insulin dosage at about the same point in time, regardless of the model that was used. In the simplest linear-regression models, with HbA_1 or weight-adjusted insulin dosage, the magnitude of the regression coefficient for FAMLIFE was less than its standard error (e.g., for percentage HbA_1 and FAMLIFE, the coefficient ± SE was .016 ± .025), and R^2 was <.01. Introducing into the analyses demographic covariates (sex, race, and SES) did not alter the lack of evident relationship between the outcome variables and FAMLIFE score. (Only race was related to the outcome variables, but this was based on only 4 non-White subjects.)

Separate regression analyses were also performed with each option of the marital problem item (no marital problem, stable marital problems, and new or worse marital problems) and arguments item, together with the demographic covariates. The results were similar to those obtained with FAMLIFE: neither HbA_1 nor weight-adjusted insulin dosage appeared to be related to any of the responses of the marital problem item or to marital arguments. For all regression analyses, R^2 remained small. (The largest occurred for the regression with the no marital problem item choice, with weight-adjusted insulin dosage as the criterion; here R^2 = .176, but this was based almost entirely on the race covariate.)

Is there a relationship between family environment and metabolic control over the years? To examine this question, we used longitudinal data on cases with two or more FAMLIFE scores, each of which could be

paired with a concurrent HbA_1 value (n = 51). For some children, the time span with usable data ranged over 6 yr. We chose not to use weight-adjusted insulin dosage in these longitudinal analyses because alterations in dosage that depend on age and puberty, among other variables, do not permit a straightforward statistical approach.

Several alternative models were examined that adjusted for the possible effects of important covariates. As covariates in our random-intercepts models, we considered these background variables: age (at testing), age squared (allowing for a quadratic relationship), sex, the sex-by-age interaction, intake age, and intake age squared. In some models, we also included SES, race, and calendar date of HbA_1.

Consistent with cross-sectional analyses, we found no associations in the analyses of longitudinal data; the regression coefficients of FAMLIFE were always smaller than their standard errors and not significantly different from zero. Without covariates (beyond the correction for subject effects introduced by random intercepts), the coefficient of FAMLIFE was $-.004 \pm .019$. When age, age squared, sex, and the interaction of age with sex were introduced as covariates, the coefficient of FAMLIFE was essentially unchanged at $-.005 \pm .020$. When SES, race, intake age, intake age squared, and date of sampling were added, the coefficient of FAMLIFE was $-.007 \pm .020$.

We also examined whether marital problems, divorce, or separation, reported over the years at time points where HbA_1 data were available, were helpful in understanding metabolic control. We used various manipulations of the marital problem item alone or with covariates (SES, race, date of HbA_1). Results were nonetheless similar and suggested no evident association among the family variables and metabolic control over time. For example, when any marital problem or only new marital problems were used with the background variables and covariates to assess the temporally evolving relationship to metabolic control, their respective coefficients were $.81 \pm .46$ and $-.83 \pm .54$.

DISCUSSION

With data from a sample of juveniles whom we have followed prospectively since diagnosis of their IDDM, we examined the relationship between family factors and metabolic control in two ways. First, we wanted to ascertain whether family variables at one point in time predict metabolic control 3–4 mo later as reflected by percentage of HbA_1 (excluding, however, the 1st yr with IDDM because of partial pancreatic β-cell functioning). Second, we wanted to establish whether family factors and metabolic control covary over the years in a meaningful manner. Weight-adjusted insulin dosage was also used as an alternative index of control, and the possible effects of demographic variables were statistically taken into account. With both sets of approaches, our indexes of family functioning were not found to be associated with metabolic control during the first several years of IDDM.

Our results must be interpreted in light of the possibility of a type 2 error (i.e., accepting the null hypothesis of no difference when, in fact, a difference exists), the measures we used, our sample selection criteria, the age range of our subjects, and our methodology. First, it must be emphasized that our failure to reject the null hypothesis could be due to our small sample size. That is, given the magnitude of variability both within and across our subjects, a considerably larger sample would be necessary to detect small effects.

Second, metabolic control of children may be affected by aspects of family functioning that are too subtle to have been captured by the measures of general functioning used in this study. It is equally possible that entirely different family domains are at play than the ones on which we focused. The fact that this report was based only on mothers as respondents also means that a certain reporting bias may have been introduced. However, the importance of the maternal viewpoint is underscored by the fact that mothers are the primary care givers of children with IDDM (22).

The fact that our cases were not preselected on IDDM-related variables may also have played a role in the results. For instance, in a recent study of children repeatedly hospitalized for diabetic ketoacidosis, it was found that family dysfunction and distress affected blood glucose regulation above and beyond compliance (23). Certain subject selection criteria may therefore identify a homogenous subgroup of patients who are deviant on several dimensions. Also, our study only included youngsters who were 8–13 yr old when they were first diagnosed with IDDM. Therefore, our findings may not be generalizable to children who develop IDDM at younger ages.

In addition, because our methodology did not specify or quantify the variables that could mediate the relationship of family life to metabolic control, possible associations may have been obscured. Although previous studies of family effects on diabetic control did not assess mediating variables either (2), one link could be compliance with IDDM regimen, as suggested by a recent study (24).

Our study differs from previous investigations in several ways, one of which is that subjects entered it at the same baseline (i.e., IDDM onset). Because our cases were not preselected on other dimensions, they may be more representative of the general population of children with newly diagnosed IDDM than samples in other studies (2). However, this population may also be intrinsically heterogenous. Repeated-measures longitudinal analysis can be effective in reducing such variability and in detecting long-term effects. Future studies may be more successful than ours by increasing both the number of subjects and the number of observations per case.

In conclusion, in our sample, knowing the quality of

family life or the degree of marital harmony did not allow us to predict the level of metabolic control of a child. But even if there is no association between these variables, there still seems to be a subset of children characterized by both metabolic and family problems. For example, in our sample, 6.7% (4 of 60) of the cases whose data could be cross-tabulated had both poor family environment and subsequent poor control (high HbA_1). Similarly, 8.3% (5 of 60) of the children had parents with marital problems and were later found to have poor control. Perhaps such a group with coexisting family and medical problems is so striking in clinical practice that it accounts for the persistence of beliefs concerning the causal relationship between aspects of the family and metabolic control. This possibility could also be examined by future studies within the context of a large sample-based investigation.

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REFERENCES

1. Drash AL, Becker D: Diabetes mellitus in the child: course, special problems, and related disorders. In *Advances in Modern Nutrition*. Vol. 2. Katzen HM, Mahler RJ, Eds. Washington, DC, Hemisphere, 1977, p. 615–43
2. Anderson BJ, Auslander WF: Research on diabetes management and the family: a critique. *Diabetes Care* 3:696–702, 1980
3. Newbrough JR, Simpkins CG, Maurer H: A family development approach to studying factors in the management and control of childhood diabetes. *Diabetes Care* 8:83–92, 1985
4. Compas BE: Coping with stress during childhood and adolescence. *Psychol Bull* 101:393–403, 1987
5. Barglow P, Hatcher R, Edidin DV, Sloan-Rossiter D: Stress and metabolic control in diabetes: psychosomatic evidence and evaluation of methods. *Psychosom Med* 46:127–44, 1984
6. Hansen CL, Henggeler SW: Metabolic control in adolescents with diabetes: an examination of systemic variables. *Fam Syst Med* 2:5–16, 1984
7. Wishner WJ, O'Brien MD: Diabetes and the family. *Med Clin N Am* 62:849–56, 1978
8. Koski ML, Kumento A: The interrelationship between diabetic control and family life. *Pediatr Adolesc Endocrinol* 3:41–45, 1977
9. Anderson BJ, Miller JP, Auslander WF, Santiago JV: Family characteristics of diabetic adolescents: relationship to metabolic control. *Diabetes Care* 4:586–94, 1981
10. Waller DA, Chipman JJ, Hardy BW, Hightower MS, North AJ, Williams SB, Babick AJ: Measuring diabetes-specific family support and its relation to metabolic control: a preliminary report. *J Am Acad Child Adolesc Psychiatry* 25:415–18, 1986
11. Marteau TM, Bloch S, Baum JD: Family life and diabetic control. *J Child Psychol Psychiatry* 28:823–33, 1987
12. Evans CL, Hughes IA: The relationship between diabetic control and individual and family characteristics. *J Psychosom Res* 31:367–74, 1987
13. Coddington RD: The significance of life events as etiologic factors in the diseases of children. II. A study of a normal population. *J Psychosom Res* 16:205–13, 1972
14. Van der Veen F, Olson RE: *Manual and Handbook for the Family Concept Assessment Method*. Encinitas, CA, 1983
15. SPSS: *SPSS User's Guide*. 2nd ed. New York, McGraw-Hill, 1986
16. Locke HJ, Wallace KM: Short marital-adjustment and prediction tests: their reliability and validity. *Marriage Fam Living* 21:251–55, 1959
17. Nathan DM, Singer DE, Hurxthal K, Goodson JD: The clinical information value of the glycosylated hemoglobin assay. *N Engl J Med* 310:341–46, 1984
18. Daneman D, Wolfson DH, Becker DJ, Drash AL: Factors affecting glycosylated hemoglobin values in children with insulin-dependent diabetes. *J Pediatr* 99:847–53, 1981
19. Laird NM, Ware JH: Random-effects models for longitudinal data. *Biometrics* 38:963–74, 1982
20. U.S. Bureau of the Census: *State and Metropolitan Area Data Book*. Washington, DC, U.S. Govt. Printing Office, 1982
21. Drash AL, Laporte R, Becker DJ, Singh B, Fisbein H, Goldstein D: The natural history of diabetes mellitus in children: insulin requirements during the initial 2 years. *Acta Pediatr Belg* 33:66–69, 1980
22. Kovacs M, Finkelstein R, Feinberg TL, Crouse-Novak M, Paulauskas S, Pollock M: Initial psychologic responses of parents to the diagnosis of insulin-dependent diabetes mellitus in their children. *Diabetes Care* 8:586–75, 1985
23. White K, Kolman ML, Wexler P, Polin G, Winter RJ: Unstable diabetes and unstable families: a psychosocial evaluation of diabetic children with recurrent ketoacidosis. *Pediatrics* 73:749–55, 1984
24. Schafer LC, Glasgow RE, McCaul KD, Dreher M: Adherence to IDDM regimens: relationship to psychosocial variables and metabolic control. *Diabetes Care* 6: 493–98, 1983