sentative of all IDDM individuals. This diabetes clinic is a major referral center for diabetes, and patients admitted to this unit tend to have more severe cases of diabetes. Moreover, only 65% of all clinic patients admitted during the study period were screened for neuropathy, and smoking information was obtained on only 69% of these patients.

In recent years, there has been renewed interest in the potential role of vascular factors in the pathogenesis of diabetic neuropathy (6,7). It may be speculated that cigarette smoking may contribute to tissue hypoxia and subsequent injury to the neural microvasculature. The association between smoking and neuropathy, however, was restricted to subjects with IDDM. This may be due to the much lower overall level of smoking in the NIDDM patients in this sample. Alternatively, the pathogenic features of neuropathy may differ between IDDM and NIDDM.

In summary, a strong association was observed between cigarette smoking and diabetic neuropathy among these IDDM clinic patients. The hypothesis that cigarette smoking is a risk factor for neuropathy should be investigated both prospectively and in a more representative population of diabetic individuals.

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Hyperphosphaturia and Hypermagnesuria in Children With IDDM

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Urinary excretion of calcium, inorganic phosphorus, magnesium, glucose, and creatinine was measured in first-void spot urine samples collected 4 days apart in 220 insulin-dependent diabetic (IDDM) children (mean age 11.9 yr) attending a summer camp. A single control urine sample was obtained from 33 healthy nondiabetic siblings (mean age 11.2 yr). Mean ± SD urinary calcium-creatinine ratios ($U_{Ca/Cr}$) did not significantly differ between IDDM and control subjects (0.14 ± 0.09 vs. 0.12 ± 0.09, respectively, $P = 0.156$). Mean urinary magnesium-creatinine ratios ($U_{Mg/Cr}$) were elevated in IDDM compared with control subjects (0.15 ± 0.06 vs. 0.08 ± 0.03, respectively, $P = 0.0001$). Similarly, mean urinary phosphorus-creatinine ratios ($U_{P/Cr}$) were significantly increased over those in control subjects (1.12 ± 0.33 vs. 0.40 ± 0.22, respectively, $P = 0.0001$). $U_{Ca/Cr}$, $U_{Mg/Cr}$, and $U_{P/Cr}$ were correlated with increasing mean urine glucose content ($P = 0.0001$). No correlations were found when $U_{Ca/Cr}$, $U_{Mg/Cr}$, or $U_{P/Cr}$ were compared with patient age, duration of diabetes, glycosylated hemoglobin, or insulin dosage. Urine losses of phosphorus and magnesium were present even when glycemic control was considered good by several methods (glycosylated hemoglobin, short-term glycemic index, or urinary glucose content). Glomerular hyperfiltration was unable to account for increased urinary mineral content. In conclusion, the data indicate that urinary excretion of phosphorus and magnesium is elevated in children with IDDM, regardless of glycemic control. In the presence of glucosuria, this loss is further enhanced. Urinary calcium excretion is significantly higher only during periods of glucosuria. The data suggest that children with IDDM could be at risk for mineral deficiencies in the absence of intensive insulin management. *Diabetes Care* 13:437–41, 1990

The glucosuria that accompanies the diabetic state is believed to impair renal tubular reabsorption of several cations and anions from the glomerular filtrate (1–6). Urinary losses of calcium and phosphorus in the patient with diabetes have been shown to be significantly reduced by a 7- to 14-day period of intensive insulin management in adults with insulin-dependent diabetes mellitus (IDDM) (7). The mechanisms for these effects are postulated to be due to a reduction of glucosuria, minimizing concomitant mineral losses. It is often difficult to maintain strict glycemic control in diabetic children, because most children are on a 2-injection/day insulin regimen. The absence of strict control may place children at risk for significant urinary losses of calcium, inorganic phosphorus, and magnesium.

The purpose of this study was to measure urinary calcium, inorganic phosphorus, magnesium, glucose, and creatinine in a large group of children attending a diabetes summer camp. Thus, a typical cross-sectional population of children with IDDM was studied, representing varying levels of glycemic control. Relative losses of these minerals in the urine of IDDM children were assessed. Comparisons were made between urinary mineral content, duration of disease, daily insulin dosage, and level of glycemic control.

RESEARCH DESIGN AND METHODS

Two hundred twenty IDDM children attending the Texas Lions Camp for children with diabetes were enrolled in the study. Control urine samples were collected from 33 healthy siblings who had no overt signs of diabetes. A first-void urine sample was collected from each child on the 2nd (S1) and 6th (S2) days of camp. All samples were collected in the fasting state. Because dietary mineral intake undoubtedly affects urinary mineral excretion, all children were sampled in the fasting state to minimize this source of variability. In addition, control samples were obtained from siblings so that any differences in dietary mineral intakes would be minimized. Control samples were obtained as a single sample on the 1st day of camp registration. Samples were immediately examined for the presence of ketones, blood, protein, and pH (Chemstrip 6L, Boehringer Mannheim, Indianapolis, IN), then acidified with HCl (1 N) and frozen at −20°C.

Demographic data were collected on each child, including duration of diabetes, age, sex, and total daily insulin dose. Heights were measured with a wall-mounted stadiometer, and weights were measured on a balance scale. A glycemic index was calculated from 5 days of self-monitoring of blood glucose with the scoring system described by Ellis et al. (8). Each child measured his/her blood glucose visually before breakfast, supper, and bedtime (Chemstrips bG, Boehringer Mannheim). Additionally, 47 patients had a hemoglobin A_{1c} (HbA_{1c}) measurement (Bio-Rad, Richmond, CA) obtained at the conclusion of camp as part of routine patient care (normal values 3–6%).

Urinary and serum creatinine were measured with the Beckman Creatinine Autoanalyzer II (Fullerton, CA). All other assays were performed by accepted spectrophotometric methods (9–12). Urine mineral content was expressed as a ratio to the urine creatinine content in milligrams per deciliter and was therefore unitless. Urinary glucose was also expressed in milligrams per deciliter.

The Wilcoxon rank-sum statistic for nonparametric data was used to determine whether significant differences existed between the means of the various groups described. Significance was established if $P < 0.05$. The mean of the S1 and S2 urinary mineral-creatinine ratios was calculated for each child. Univariate linear regressions were performed for measured variables (HbA_{1c}, mean urine glucose, age, duration of diabetes, glycemic index, or daily insulin dosage) with each mean mineral-excretion–creatinine ratio as the dependent variable. This protocol was approved by the Institutional Review Board, University of Texas Medical Branch.

RESULTS

Mean age of 33 sibling control subjects (13 girls, 20 boys) was 11.2 yr (range 6–16 yr). Results of urine dipstick for ketones, glucose, protein, and hematuria were negative. Values for mean urinary calcium-creatinine ratio ($U_{Ca/Cr}$), phosphorus-creatinine ratio ($U_{P/Cr}$), and magnesium-creatinine ratio ($U_{Mg/Cr}$), from the control group are shown in Fig. 1.

Descriptive statistics of the diabetic children studied are listed in Table 1; there were 112 boys and 108 girls. Height and weight percentiles decreased as a function of duration of diabetes ($r = -0.28$, $P = 0.0001$, for height, and $r = -0.19$, $P = 0.004$, for weight). No children were clinically acidotic, although 8% in S1 and 9.3% in S2 had measurable urine ketones greater than trace.

Mean $U_{Ca/Cr}$ did not significantly differ from control values ($U_{Ca/Cr}$ ± SD 0.14 ± 0.09 for IDDM children and 0.12 ± 0.09 for control subjects, $P = 0.156$; Fig. 1). When mean $U_{Ca/Cr}$ was expressed as a function of urinary glucose content, a positive association was detected ($r = 0.36$, $P = 0.0001$). $U_{Ca/Cr}$ was also positively correlated with increasing glycemic index ($r = 0.33$, $P = 0.0001$; Fig. 2).

Mean $U_{P/Cr}$ of IDDM children differed significantly from that of control subjects (1.12 ± 0.33 and 0.40 ± 0.22, respectively, $P = 0.0001$; Fig. 1). $U_{P/Cr}$ of IDDM children was significantly elevated over that of control subjects at all levels of glycosuria. However, increasing urine glucose was positively associated with increasing $U_{P/Cr}$ ($r = 0.31$, $P = 0.0001$; Fig. 2). $U_{P/Cr}$ was significantly increased when expressed as a function of glycemic index ($r = 0.37$, $P = 0.0001$; Fig. 2).

Mean $U_{Mg/Cr}$ values for IDDM children were signifi-

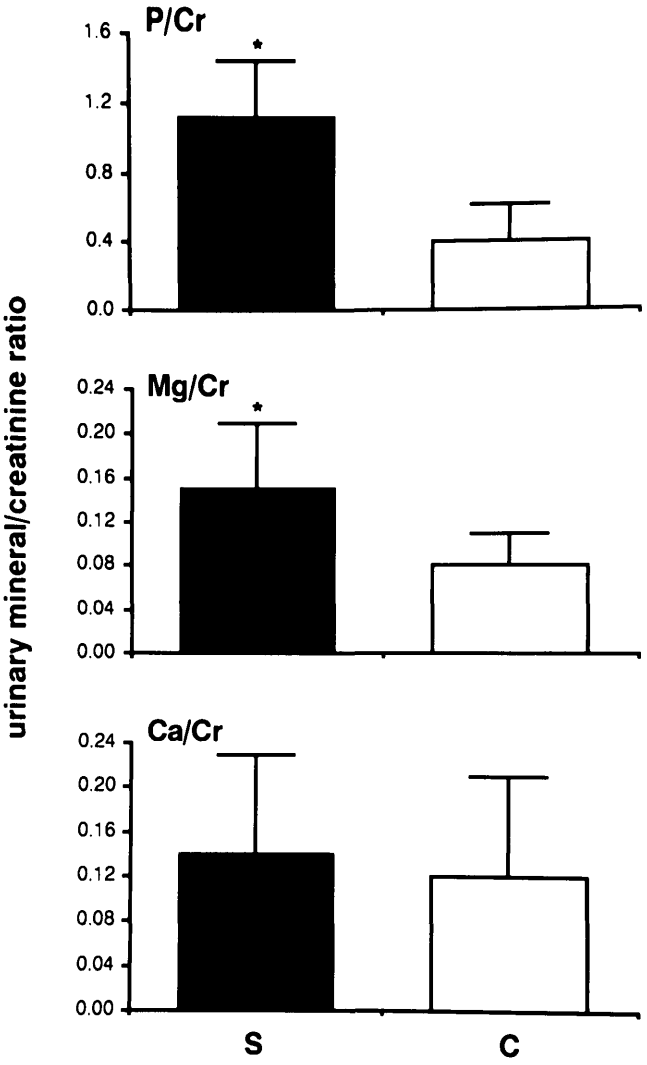

FIG. 1. Mean ± SD of urinary phosphorus-creatinine (P/Cr), magnesium-creatinine (Mg/Cr), and calcium-creatinine (Ca/Cr) ratios for 220 children with insulin-dependent diabetes mellitus (IDDM). Ratios are expressed as mineral-creatinine content (mg/mg). S, mean of 2 samples measured 4 days apart in all children shown. Control (C) values in 33 healthy nondiabetic siblings are shown on *right*. *Statistical significance from control subjects ($P < 0.0001$) by nonparametric analysis. IDDM children have significant elevation in urinary phosphorus and magnesium content relative to control subjects.

cantly higher than for control subjects (0.15 ± 0.06 and 0.08 ± 0.03, respectively, $P = 0.0001$; Fig. 1). Similar to $U_{Ca/Cr}$ and $U_{P/Cr}$, $U_{Mg/Cr}$ varied directly with the mean urine glucose content ($r = 0.33$, $P = 0.0001$) and the glycemic index ($r = 0.32$, $P = 0.0001$; Fig. 2).

Mean urine glucose content was categorized into low (0–100 mg/dl, $n = 64$), medium (100–1000 mg/dl, $n = 56$), and high (1000–10,000 mg/dl, $n = 100$) levels of glycosuria. $U_{P/Cr}$ and $U_{Mg/Cr}$, but not $U_{Ca/Cr}$ (except in the high group), were significantly elevated over control values at all levels of glycosuria ($P = 0.0001$).

TABLE 1
Descriptive statistics

Variables	Range	Mean ± SE
Age (yr)	7–18	11.9 ± 2.2
Duration	1 mo–15.6 yr	3.4 ± 2.9 yr
Height (%)	0–100	44.5 ± 2.0
Weight (%)	0–100	55.8 ± 1.9
Glycemic index	0–75	17.7 ± 1.1
HbA_{1c} (%)*	4–11.8	7.6 ± 0.3
Insulin ($U \cdot kg^{-1} \cdot day^{-1}$)	0.03–1.8	0.82 ± 0.02

$n = 47$ for HbA_{1c}; $n = 220$ for all other variables.
*Normal = 3–6%.

There were no correlations between HbA_{1c} and $U_{Ca/Cr}$, $U_{Mg/Cr}$, or $U_{P/Cr}$. Similarly, duration of disease or daily insulin dosage did not correlate with any mean mineral-excretion ratios.

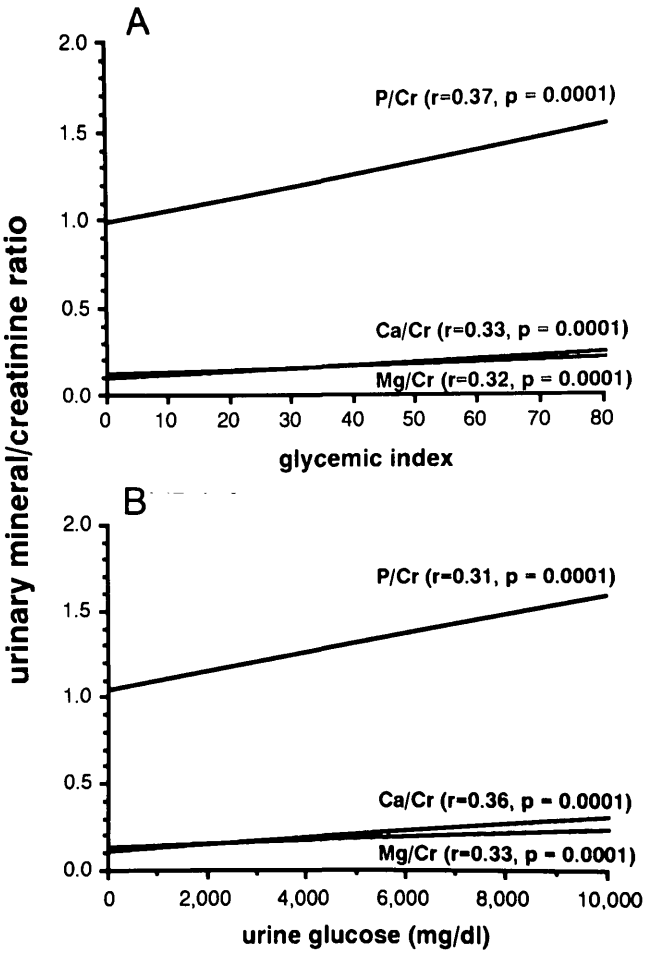

FIG. 2. Linear regression analyses for mean urinary mineral-creatinine content (phosphorus-creatinine [P/Cr], calcium-creatinine [Ca/Cr], and magnesium creatinine [Mg/Cr]) as dependent variable (expressed as mineral-creatinine content mg/mg) and glycemic index (unitless; *A*) or urinary glucose content (*B*) as independent variable in 220 children with insulin-dependent diabetes mellitus. Correlation coefficients and significance are shown.

Urinary calcium, phosphorus, magnesium, and glucose were measured in two age- and sex-matched groups (n = 40) to determine whether glomerular hyperfiltration was associated with elevated urinary mineral content. Two groups were formed. The elevated glomerular filtration rate group was defined by the presence of a creatinine clearance (CrCl) ≥ 2.38 ml $\cdot$ s^{-1} $\cdot$ 1.73 m^{-2}, a commonly accepted definition of glomerular hyperfiltration (13). The comparison group consisted of children with CrCl <2.38 ml $\cdot$ s^{-1} $\cdot$ 1.73 m^{-2}. The CrCl was calculated by the standard formula from a timed 4-h urine collection and venipuncture. Mean ± SE CrCl in the hyperfiltrating group was 2.78 ± 0.12 ml $\cdot$ s^{-1} $\cdot$ 1.73 m^{-2}. The nonhyperfiltrating group mean CrCl was 1.83 ± 0.06 ml $\cdot$ s^{-1} $\cdot$ 1.73 m^{-2} (P = 0.001). There were no significant differences between the two paired groups for calcium, phosphorus, magnesium, or glucose content.

DISCUSSION

The data indicate that the population of IDDM children in this study have normal urinary calcium indices in the absence of glucosuria. However, urinary calcium excretion varies directly with urinary glucose excretion. In contrast, both urinary phosphorus and magnesium were elevated in aglucosuric diabetic children compared with control subjects. Increasing excretion of urinary glucose was positively correlated with further increases in urinary excretion of phosphorus and magnesium.

Both groups demonstrated positive correlations between urinary mineral-creatinine ratios and between the glycemic index and urine glucose content. However, there were no correlations observed in a subgroup of 47 children who had a HbA_{1c} measurement taken at the conclusion of camp. This suggests that short-term glycemic control (assessed by the glycemic index) is linked with urinary mineral excretion to a greater extent than glycemic control over a 2- to 3-mo period. The previous studies by Gertner et al. (7) support this statement. They showed a significant reduction in urinary calcium and phosphorus excretion in association with a 1-wk period of intensive insulin therapy and concomitant reduction in mean serum glucose levels.

Glomerular hyperfiltration (creatinine clearance ≥ 2.38 ml $\cdot$ s^{-1} $\cdot$ 1.73 m^{-2}) is frequently present in patients with IDDM (13); however, it does not explain the calciuria, phosphaturia, and magnesuria of IDDM.

Chronic renal wastage of calcium, phosphorus, and magnesium in diabetic children suggests that deficiencies of these elements are possible. Based on these data, mineral losses are present early in the clinical course of IDDM and are unrelated to insulin dosages in children with nonrigid glycemic control. The data suggest that phosphaturia and magnesuria are constitutive in some children in the absence of strict glycemic control. Because the reservoir for body minerals lies in the skeleton, relative deficiencies of calcium, phosphorus, or magnesium may be related to the reduced pubertal growth spurt in height or decreased bone mineral content observed in some IDDM children (14,15).

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Impact of Social Support and Stress on Compliance in Women With Gestational Diabetes

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Compliance with medical recommendations is especially important for women with gestational diabetes because of the health implications for both mother and fetus. This study examined compliance with two daily self-management tasks, diet and insulin administration, in 98 women with gestational diabetes. Furthermore, the influence of stress and regimen-related social support on compliance was investigated. Results indicate that the level of reported compliance was high for both insulin administration and diet. Fewer minor stressors and greater social support were associated with greater compliance. *Diabetes Care* 13:441–43, 1990

Although gestational diabetes occurs in up to 4% of pregnant women (1), no studies have investigated the degree of compliance or the influences of psychosocial factors, e.g., stress and social support, on compliance in women with gestational diabetes. Compliance with medical recommendations is important in women diagnosed with gestational diabetes because of the presence of a second "patient," the fetus, who is at risk for complications associated with inadequate metabolic control. Therefore, this study was designed to investigate the effects of stress and regimen-related social support on self-care compliance. Because the diagnosis of gestational diabetes is sudden, and management tasks are new for these women, we hypothesized that women with gestational diabetes are highly compliant, and increased stress and low social support are associated with poorer compliance with daily regimen tasks.

RESEARCH DESIGN AND METHODS

The participants in this study were 98 English-speaking pregnant women (85% White, 60% married) with gestational diabetes ranging in age from 17 to 41 yr (mean ± SD 27.6 ± 5.6) who were interviewed at 30–40 wk gestation (34.6 ± 3.2). The Hollingshead social class breakdown of the women was as follows: I, 23%; II, 20%; III, 25%; IV, 25%; and V, 7% (2). The diagnosis of gestational diabetes was based on a standardized 3-h oral glucose tolerance test administered at ~28 wk gestation with plasma glucose and the hexokinase method (3). The criteria used for diagnosis were >95 mg/dl at fasting, >180 mg/dl at 1 h postprandial, >155 mg/dl at 2 h postprandial, and >140 mg/dl at 3 h postprandial. All glucose-intolerant patients were treated with diet, whereas 31 patients were treated with insulin in addition to diet. Dietary recommendations were given by the dietitian at the initial clinic visit after the diagnosis and were based on the American Diabetes Association's *Exchange Lists for Meal Planning* (4). Specific dietary prescriptions included 125 g protein and 30–35 kcal · kg^{-1} · day^{-1} (5). Furthermore, recommendations for total daily calorie intake were generally between 1800 and 2600 kcal, except in a few patients (e.g., morbidly obese patients) in whom higher levels were needed. The specific content and timing of meals were tailored for the individual. Each patient's whole-blood glucose levels were monitored weekly at the clinic, and in addition, insulin-treated patients were required to monitor their blood glucose daily.

One to 2 wk after their first clinic appointment, all patients were asked to complete a checklist of 17 major life events (e.g., death of a close relative) and a measure of minor stressors (hassles scale; 6). The Diabetes Compliance Questionnaire (Table 1) was also administered, and responses were analyzed to assess two areas of diabetic management and control applicable to pregnant women with diabetes, i.e., insulin administration and diet (7). The Diabetes Social Support Questionnaire (Table 2) was used to assess the patients' perception of social support from family and friends regarding diabetes care for the same two areas of diabetic management (7).

RESULTS

In general, the level of reported compliance with both diet and insulin was high, with 66 and 71% of patients reporting that they frequently or always comply with these regimen tasks, respectively. The degree of re-