giographic examination is needed for accurate detection of earliest retinal damage.

These data do not conflict with those previously reported by Aspinall and co-workers. They showed the predicting value of color vision disturbance in diabetic patients but not a correlation with early stages of retinopathy. Periodic evaluation of color vision may be efficient in order to detect diabetic subjects with high risk of retinopathy. But angiographic examination is necessary to confirm early stages of retinopathy in those subjects who have color vision disturbances. The need of angiographic examination in diabetic subjects without color vision disturbance is now being investigated.

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Acuphobia in a Long-standing Insulin-dependent Diabetic Patient Cured by Hypnosis

A 42-yr-old white woman who had had extremely poor glycemic control due to unexplained, frequent, severe hypoglycemia throughout her 33 yr of diabetes was admitted to our diabetic service for complete evaluation. From her history and from our clinical pathology results she appeared to be an excellent candidate for a trial of continuous subcutaneous insulin infusion. She had been hospitalized on several previous occasions for adjustment of insulin therapy, but had always been discharged with less than optimal glycemic control. She had been on multiple insulin injections, was practicing home glucose monitoring, demonstrated a good level of diabetic education, and was compliant with her diet. Her diabetes was complicated by distal symmetrical polyneuropathy and background retinopathy.

After admission she was started on continuous subcutaneous insulin infusion and within 48 h was in excellent control. At this stage it was noted by the nurses that she refused to insert the needle of the pump catheter into the abdominal subcutaneous tissue and it was thought that she had an aversion to using the abdominal site. On further questioning she admitted that she had never been able to give herself an insulin injection or to prick her finger for home glucose monitoring and that insulin injections and her home glucose monitoring were performed by her husband. Moreover, when she was a child her father had performed these tasks. We felt that unless her acuphobia was corrected, she would not be able to perform the frequent home glucose monitoring required to minimize the dangers of external insulin pump therapy.

Before hypnotic evaluation and induction the patient was asked by the therapist to inject herself, either in the thigh or the stomach, with a standard insulin syringe containing 0.25 ml sterile saline. Upon receipt of the needle and syringe, the patient became diaphoretic and appeared to be extremely distressed. She then developed tremors and said she could not inject herself. After observation of these symptoms the patient was assessed for possible hypnotherapy. She was initially typed as a moderate-to-poor candidate for hypnosis by standard rating methods. However, after the first induction she did manifest a light trance, with arm levitation. During the second session she was instructed to inject the needle into her abdomen and that in so doing she would feel a slight cool sensation at the injection site but no pain. She complied. During this session and during three subsequent sessions she was given the posthypnotic suggestion that she would feel no pain or discomfort on needle insertion or when doing her own finger punctures. Thereafter, she had no difficulty with insulin injections or finger sticks and 1 mo after discharge she continued to perform these tasks.

We conclude that when severe and chronic acuphobia occurs in insulin-requiring diabetic individuals, hypnotherapy may be helpful in overcoming the acuphobia, and that once this phobia has been overcome, it need not recur.

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Ketoacidosis Without Hyperglycemia During Self-Monitoring of Diabetes

Ketoacidosis remains a hazard for patients with insulin-dependent diabetes (IDDM).[1] Self-monitoring of capillary blood glucose provides a means of adjusting insulin therapy during such difficult periods.[2] We describe two patients who presented in severe ketoacidosis despite maintaining relatively normal blood glucose concentrations until shortly before admission.

Case 1: A 36-yr-old male hospital administrator, with a 25-yr history of IDDM, had been managed for 2 yr by continuous subcutaneous insulin infusion in combination with self-monitoring of blood glucose with a reflectance meter (Dextrostix/Glucometer, Ames Division, Miles Laboratories, Inc., Stoke

Poges, Slough, England). Three days before admission he experienced abdominal pain and nausea after dining out. He maintained his preprandial capillary blood glucose below 10 mmol/L with his usual insulin schedule and a light equivalent diet. Twenty-four hours before admission he began vomiting but was still able to take equivalent amounts of oral glucose. On the morning of admission, capillary blood glucose was 9.0 mmol/L, the patient having taken his normal basal insulin infusion rate (0.6 U/h) for the previous 12 h. He was now feeling thirsty and telephoned the ward for advice. He wanted to continue managing the condition at home; however, his speech sounded "thick" on the telephone and he was advised to come to the ward. On admission 3 h later, he was profoundly dehydrated and ketotic; plasma glucose was 21.7 mmol/L, serum bicarbonate 7.9 mmol/L, serum urea 11.8 mmol/L, and arterial pH 7.1. He was managed with intravenous fluids and additional hourly doses of insulin, and recovered uneventfully.

Case 2: A 24-yr-old female arts graduate with IDDM for 11 yr, on twice-daily subcutaneous insulin, had been self-monitoring with a reflectance meter (Glucometer, Ames) for 2 yr. Two weeks before admission she had a mild abdominal upset with vomiting lasting 4 days, which she managed successfully at home, taking glucose drinks and extra short-acting insulin. Only one capillary blood glucose reading during that time exceeded 10 mmol/L. Four days before the present admission, the patient had further abdominal discomfort with intermittent diarrhea and vomiting. She maintained relatively normal blood glucose readings (only one reading greater than 10 mmol/L) until the morning of admission. By then the reflectance meter read "high" and she was feeling thirsty. After taking an increased morning insulin dose she came to the hospital emergency department. On admission, she was dehydrated and ketotic; plasma glucose was 36.4 mmol/L, serum bicarbonate 11.3 mmol/L, serum urea 13.8 mmol/L, and arterial pH 7.23. After intravenous fluids and hourly supplementary insulin she made a full recovery.

Both of the patients described were intelligent; they tested blood glucose at least four times daily and were regular clinic attenders. It might be expected that such patients would be well equipped to manage intercurrent illness themselves and to avoid hospital admission. Our two patients, while able to manage the early part of their illness adequately, ultimately delayed seeking formal advice until dangerous acidosis had developed.

Patients who are self-monitoring must not forget that severe acidosis may co-exist with normal blood glucose concentrations.[3] Clouding of consciousness may not be prominent in ketoacidosis without hyperglycemia and such patients may appear surprisingly well. Neither of our patients had tested urine for ketones, which might have given early warning of a situation beyond their control. A new generation of patients (and doctors) instructed in the disadvantages of testing urinary rather than blood glucose concentrations needs to be reminded of the value of testing urine for ketones. The development of a reagent strip suitable for quantitative estimation of blood ketone concentrations would be useful for patients attempting to monitor intercurrent illness at home.

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Urinary Cortisol-Creatinine Ratio and Nocturnal Hypoglycemia

Recent reports suggest varying views on the relationship between hypoglycemia and the urinary cortisol-creatinine ratio (UCCR) and the latter's usefulness in monitoring nocturnal hypoglycemia (NH) in insulin-treated diabetic individuals (ITD). Asplin et al.[1] and Seaworth et al.[2] report findings that suggest that the UCCR might be useful in detecting NH, but Scott and Scandrett[3] and Darlow et al.[4] found no clear relationship between episodes of NH and elevated UCCR.

We report the results of a study to find the frequency of NH in a small group of stable and unstable ITD. Seven unstable (either newly diagnosed or known diabetic subjects with poor control) and 20 apparently stable (asymptomatic and normal urine test records and normal and near-normal spot blood glucose levels) diabetic subjects were studied. The patients' characteristics are summarized in Table 1.

Urine was collected as an early morning sample on four consecutive days. Capillary blood on filter paper was obtained from the same subjects at 3 a.m. Details of UCCR and capillary blood glucose determination are described elsewhere.[5]

The mean ± SEM UCCR of the stable and unstable ITD were 11.27 ± 0.90 and 16.71 ± 2.81, respectively (normal controls 8.5 ± 0.40). The normal range of UCCR for our laboratory is considerably lower than other reported values.[2–4,6] The difference is most likely to be attributable to methodology employed in the determination of urinary free cortisol.

Interpreting values of UCCR greater than the normal mean +2 SD as indication of NH, five of the seven unstable diabetic patients had at least one episode of NH in 4 days and 8 of the 20 stable ITD had NH by the same criterion.