accuracy of blood glucose self-monitoring techniques by comparing the results obtained with laboratory values (1–3). However, factors other than technique have been poorly studied and may contribute significantly to erroneous results, especially under the particular conditions found in intensive-care units.

We recently discovered wide discrepancies between nurse-obtained blood glucose values and laboratory results in a patient in our intensive-care unit who was in hepatic failure and was receiving intravenous insulin boluses every 6 h based on Chemstrip bG (Boehringer Mannheim, Indianapolis, IN) results. After ensuring that the reagent strips were accurate and that the procedure was being performed correctly and in accordance with the limitations set forth in the package insert, we requested assistance from the strips' manufacturer.

Among the patient's many abnormal blood chemistries and medications, he was receiving dopamine in a continuous intravenous drip. Boehringer Mannheim's investigation determined that dopamine [4-(2-aminoethyl) brenzcatechin-HCl] inhibited the chemical reaction on the strip. In fact, the dopamine interference, although not as great, also occurred with laboratory glucose oxidase procedures (D. Dunn, personal communication). We then discovered similar results with Glucostix (Ames, Elkhart, IN) in a diabetic patient who was receiving dopamine.

Because all the commonly used whole-blood glucose reagent strips are based on the glucose oxidase reaction, it seems reasonable to assume a similar interference with all manufacturers' strips. This interference can be easily demonstrated in vitro with various concentrations of dopamine either in whole blood or in glucose solutions.

Further research is indicated to determine the actual blood levels of dopamine that interfere with blood glucose readings

Retinopathy in Tropical Pancreatic Diabetes

This letter is in reference to those of Davidson and Smith (1) and Couet and Drouin (2). Davidson and Smith refer to Geevarghese's book published in 1968 (3), which mentions the low prevalance of retinopathy in tropical pancreatic diabetes (TPD) in southern India. We have recently shown that both sight-threatening forms of retinopathy, i.e., proliferative retinopathy and maculopathy, are seen in patients with TPD when these patients are followed for long periods (4). Neuropathy (5) and nephropathy (6) are also seen in these patients. The frequency of microvascular complications was not significantly different from that of a matched group of non-insulin-dependent diabetic patients (6). Note that in Geevarghese's more recent book (7), there is a separate chapter on diabetic retinopathy in TPD. The subject of microvascular complications in TPD has been reviewed by us recently (8,9). Macrovascular complications are less common in this entity (6). This could be related to the relative youth, leanness, or low lipid levels of these patients (6). Prospective studies are needed to determine the natural history of vascular complications in TPD because of the special characteristics of this form of diabetes, which include association with protein-calorie malnutrition and relative infrequency of ketosis despite requiring insulin for stabilization of diabetes (10).

VISWANATHAN MOHAN, MD
REMA MOHAN, MBBS, DO
AMBADY RAMACHANDRAN, MD
CHAMUKUTTAN SNEHALATHA, DPhil
MOOPIL VISWANATHAN, MD

From the Diabetes Research Centre and M.V. Hospital for Diabetes, Royapuram, Madras, India.
Address correspondence and reprint requests to Viswanathan Mohan, MD, Diabetes Research Centre and M.V. Hospital for Diabetes, 5 Main Road, Royapuram, Madras 600 013, India.

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extreme hyperkalemia associated with severe hyperglycemia and ketoacidosis.

A 62-yr-old diabetic male, insulin dependent since age 52 yr and complicated by proliferative retinopathy, peripheral neuropathy, and mild renal failure, had been admitted to our hospital for diabetic ketoacidosis and discharged a week before the episode reported herein. His only medications were 12 U regular insulin in the morning and 18 U before dinner and 30 U NPH insulin at night.

He was admitted to the emergency room in a stupor. Table 1 summarizes the main laboratory findings. The ECG showed lack of P-waves and marked widening of the QRS complexes. Endovenous $CaCl_2$, $NaHCO_3$, and regular insulin were administered, but he developed ventricular fibrillation and, finally, despite cardioversion, cardiac arrest. Intracardiac epinephrine was injected and resuscitative maneuvers continued over 45 min without success. In that critical situation an intracardiac injection of regular insulin (0.5 U/kg) was administered. Two minutes later he started a sinusal rhythm with subsequent hemodynamic normalization, and consciousness was restored. His hyperkalemic and ketoacidotic state was resolved in a few hours with conventional treatment. Later, the patient revealed that he abandoned insulin therapy 3 days before admission.

Hyperkalemia is a well-known initial finding in untreated diabetic ketoacidosis and is thought to be a major cause of death (3). Deleterious effects on electrical activity of the heart are by far the most important consequences of hyperkalemia. The electrically active tissues of the heart are particularly sensitive to changes in the extracelluar concentration of K^+. Thus, hyperkalemia may result in dysrhythmia due to increased automaticity, reflecting repetitive depolarizations. At higher concentrations of K^+ (8–9 meq/L), there is profound depression in impulse generation and conduction in all cardiac tissues, widening of the QRS complex, and