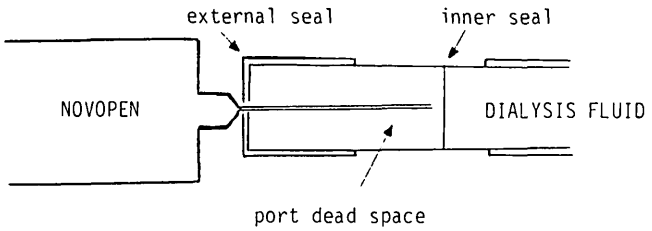

FIG. 1. NovoPen injection into continuous ambulatory peritoneal dialysis device.

The use of NovoPen to add insulin to dialysis bags for CAPD diabetic patients should be advised only when longer injection needles are used and after proper testing to assure that the insulin is not retained in the dead space of the dialysis bag used by the CAPD patient.

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Alcohol Abuse and Diabetic Ketoacidosis

Alcohol abuse can seriously complicate the management of diabetes mellitus. Moreover, excessive alcohol intake, especially in binges, is believed to sometimes precipitate ketoacidosis (1). A few patients admit such drinking, and others are suspected of it. To learn how often recent excessive alcohol use occurred in patients with diabetic ketoacidosis (DKA), we questioned them and measured their serum ethanol concentrations. We particularly wanted to learn whether recent drinking might be an antecedent in patients with otherwise unexplained episodes of DKA, especially those with recurrences.

We analyzed the findings in 97 consecutive episodes of DKA in adults admitted to the Medical Service of the Bronx Municipal Hospital Center during a 19-mo period (January 1986–July 1987). Serum ethanol was measured in 88 (47 men, 41 women) of the 97 episodes; the patients had mean ± SD age of 44.0 ± 18.2 yr (range 18–79). All patients had a blood pH <7.37 (mean 7.20 ± 0.14), serum glucose >200 mg/dl (mean 808 ± 389 mg/dl), and serum bicarbonate <20 mM (mean 9.8 ± 5.2 mM). Sixteen patients (7 men, 9 women) had recurrent episodes (31 total episodes during 19 mo).

Biochemical measurements were performed in the hospital clinical laboratories. During the first 8 mo of the survey (involving 34 episodes), serum ethanol was measured manually according to the alcohol dehydrogenase enzymatic method of Bonnichsen (2). During the second half of the study (54 episodes), serum ethanol was measured with the same chemical method but with a Du Pont ACA IV (Wilmington, DE). With the manual method used earlier, moderately lipemic sera gave a "blank" reading for ethanol, which did not occur with the automated method. Eleven patients stated that they had been drinking mildly, and 1 heavily, during ≤1 wk before hospitalization. Ethanol was detected in the serum of only 3 of those patients, at levels of 13, 32, and 292 mg/dl.

In the 34 episodes in which serum ethanol was measured manually, 6 patients who denied drinking had moderate lipemia. Their serum ethanol concentrations artifactually were 8–13 mg/dl. Two other patients who did not have lipemia had serum ethanol levels of 13 and 25 mg/dl. In the 54 episodes in which serum ethanol was measured with the automated method, only 2 patients had detectable levels (32 and 292 mg/dl). Thus, only 4 of the 82 patients without visible lipemia had ethanol detectable in their serum, at concentrations of 13, 25, 32, and 292 mg/dl. Three of them readily admitted recent ethanol use, and the patient with a level of 292 mg/dl had a history of recent heavy drinking and was clinically intoxicated.

Of the 16 patients who had recurrent episodes of DKA, only 2 had ethanol detectable in their blood. Only 1 admitted recent drinking and had an ethanol level of 13 mg/dl. The other patient with recurrent episodes had lipemic serum and a very low serum ethanol level (12 mg/dl) measured manually.

During this survey we also studied 28 ketotic diabetic patients who were not acidemic (mean ± SD serum bicarbonate 19.3 ± 3.3 mM, but blood pH ≥7.37). Of these, 5 had ethanol detectable in their serum, but in 1, with a level of 34 mg/dl, this was probably due to lipemia. The other 4 had ethanol levels of 5, 16, 30, and 236 mg/dl, and the latter 2 admitted recent drinking.

Ethanol is metabolized at a rate averaging ~100 mg · kg^{-1} body wt · h^{-1} (3). Therefore, a person who drank enough to have a blood level of 100 mg/dl could dissipate that in ~6 h, and a very intoxicated person with a level of 200 mg/dl might not have ethanol detected after ~12 h. Nevertheless, in this series of patients seen in a municipal hospital, whose population otherwise includes many ethanol abusers, evidence of ethanol intoxication was rare in the patients with DKA. Indeed, only 4 of the 88 acidemic patients had any de-

tectable serum ethanol that could not be explained by the lipemia artifact. This does not necessarily mean that prior drinking may not have contributed to the episodes of DKA, but there was no evidence of very recent heavy drinking in most of the patients, thus confirming their histories. In particular, 14 of the 16 patients with recurrent episodes had neither a history of drinking nor detectable blood ethanol.

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