

- neuropathy: assessment and comparison of clinical examination and quantitative sensory testing. *Diabetes Care* 12:270–75, 1989
2. Rydel A, Seiffer W: Untersuchungen über das Vibrationsgefühl oder die Sog: "Knochensensibilität" (Pallästhesie). *Arch Psychiatr Nervenkr* 37:488–536, 1903
 3. Assal J Ph, Liniger C, Albeanu A, Moody JF: The tuning fork revisited: detection of potentially threatening sensation loss with Rydel-Seiffer's tuning fork (Abstract). *Diabetologia* 32:462A, 1989
 4. Boulton AJM, Kubrusly DB, Bowker JH, Gadia MT, Quintero DM, Becker JS, Skyler J, Sosenko M: Impaired vibratory perception and diabetic foot ulceration. *Diabetic Med* 3:335–37, 1986
 5. Guy RJC, Clark CA, Malcolm PN, Watkins PJ: Evaluation of thermal and vibration sensation in diabetic neuropathy. *Diabetologia* 28:131–37, 1985
 6. American Diabetes Association, American Academy of Neurology: Consensus statement: report and recommendations of the San Antonio Conference on Diabetic Neuropathy. *Diabetes Care* 11:592–97, 1988

Extreme Hyperglycemia and Hyperosmolarity

We report the occurrence of hyperosmolar hyperglycemic nonketotic coma (HHNKC) in a 29-yr-old man with new-onset type I (insulin-dependent) diabetes mellitus who showed extreme hyperglycemia and hyperosmolarity of unreported magnitude and made a complete recovery. The patient, with no significant past medical history, was found unconscious in his apartment. Paramedics infused 50 ml of 50% dextrose along with naloxone and thiamine. On arrival in the emergency room ~30 min later, the patient was deeply comatose. After blood was drawn for routine laboratory work, including serum glucose, he was given another 50 ml of 50% dextrose. The physical examination was unremarkable apart from coma and signs of dehydration. His temperature was 101°F, blood pressure was 100/70 mmHg, and pulse rate was 120 beats/min. His chest X ray was normal, and an electrocardiogram revealed sinus tachycardia with nonspecific S-T segment changes. He was intubated and placed on a mechanical ventilator. Shortly thereafter, he developed focal seizures that became generalized.

Laboratory data showed a serum glucose of 311.3 mM confirmed by repeat analysis with the glucose oxidase–oxygen electrode method. The patient's serum sodium level was 105 mM, potassium was 5.3 mM, chloride was 77 mM, bicarbonate was 12 mM, blood urea nitrogen was 17.5 mM, and creatinine was 259.42 $\mu\text{mol/dl}$. Serum and urine acetone were negative. Calculated serum osmolarity was 541 mM. Lactic acid was 5.3 mM, hemoglobin was 17.3 g/dl, hematocrit was 56, white blood cell count was $9400 \times 10^9/\text{L}$, and mean corpuscular volume was 107 fl. He was given 20 U of regular insulin intravenously and rapid infusion of normal saline with 10 U/h of regular insulin in the emergency room.

On arrival in the intensive care unit, his blood pressure was 70/30 mmHg by arterial line inserted in the femoral artery, and his pulse rate was 144 beats/min. Pulmonary capillary wedge pressure was 4 mmHg. Arterial blood gases revealed pH 7.25, PCO_2 36 mmHg, and PO_2 200 mmHg on 50% inspired O_2 . Over the next 6 h, he was managed aggressively with normal saline solution until he was hemodynamically stable. Insulin was infused at the rate of 1U/h. Wedge pressure after 6 h of fluid therapy in the intensive care unit was 17 mmHg. Urine output averaged 40 ml/h. Fifteen hours after admittance to the emergency room, the patient had received a total of 79 U of insulin and 10 L of fluids. Serum glucose after 13 h was lowered to 37.67 mM.

The day after admission, decerebrate posturing on painful stimuli was noted. Intravenous fluids and insulin were continued with close monitoring of blood glucose and hemodynamic profile. The patient regained consciousness on the 3rd day. Oral feedings were started along with NPH insulin. The patient admitted to having polyuria and polydipsia beginning the day before admittance to our hospital. He had consumed 12 L of Coca-Cola, 6 cans of pineapple juice with sugar, several liters of orange juice, and sugared water. He had attributed his thirst to hot weather. He was discharged on 35 U of NPH insulin and 10 U of regular insulin in the morning, and 20 U of NPH insulin and 8 U of regular insulin in the evening. Fasting serum glucose was 8.25 mM. The physical examination, including a neurological examination, was normal.

Our patient showed extreme hyperglycemia and hyperosmolarity previously unknown to be compatible with human life. His serum glucose level was substantially higher than the world record of 264 mM reported in the past (1). The serum sodium of 105 mM was unusually high for the degree of hyperglycemia. With the formula that 5.6 mM elevation of serum glucose decreases serum sodium by 1.5 mM, sodium should have been 58 mM. This relative hypernatremia may be explained by severe dehydration with water loss in excess of sodium loss. This is supported by the observation that, after 2 h of saline infusion and a decrease in serum glucose by 121 mM, serum sodium rose to only 111 mM. Volume depletion was substantiated by the patient's hemodynamic profile, and after volume replacement, hematocrit decreased from 56 to 37%, and mean corpuscular volume of erythrocytes decreased from 106 to 90 fl in 4 h. The extreme severity of our patient's hyperglycemia was contributed to by drinking large quantities of sugared beverages, administering intravenous dextrose before the diagnosis of HHNKC, and impaired renal function due to marked prerenal azotemia.

Because of hemodynamic instability, large amounts of fluid in the form of normal saline were given rapidly, which resulted in an undesired precipitous drop in osmolarity. Although unnecessarily large doses of insulin were administered in the emergency room, the reduction of serum glucose was largely caused by volume expansion. Serum glucose fell from 311.3 to 37.67 mM

in 13 h. This rapid reduction did have a short-term adverse affect, manifested by the appearance of decerebrate posturing 24 h after initiation of therapy. However, this resolved quickly, and at the time of discharge, no neurological deficits were detectable.

Unquestionably, hyperglycemia and hyperosmolarity in our patient were aggravated by the injudicious administration of 50% dextrose, which in a more fragile individual may have exceeded the tolerable limit. Given the ease and rapidity of glucose estimation by reagent strips, the practice of indiscriminate administration of dextrose to comatose patients should, in our opinion, be discouraged.

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REFERENCES

1. Knowles HC Jr: Syrupy blood (Editorial). *Diabetes* 15:760–61, 1966

Deception in Diabetes Research

Recently, Ziegler et al. (1) used deception with their research subjects to examine the reliability of patient-generated data from self-monitoring of blood glucose (SMBG). In their study, they actively misled 14 patients with type I (insulin-dependent) diabetes into thinking their reflectance meters (Glucometer I) were malfunctioning and then used this opportunity to replace the patients' meters with experimental memory meters: "During a routine visit of the subjects to the outpatient clinic of our department, we took advantage of an alleged malfunctioning of their device to lend them a memory-reflectance meter for a period of 21 days" (1). Patients were not informed that their blood glucose measurements, along with the time and date, were being stored in the memory meters, and they were told to continue to monitor their blood glucose and record the measurements in their logbooks. Ziegler et al. examined the reliability of the patients' recorded measurements by comparing the logbook and memory-reflectance meter recordings.

The use of deception by the physicians in this study concerns me for two reasons. First, deception appears to compromise the patients' privacy. Privacy in this context refers to "the freedom of the individual to pick and choose for himself the time and circumstances under which, and most importantly, the extent to which, his attitudes, beliefs, behavior and opinions are to be shared with or withheld from others" (2). In Ziegler et al.'s study, the measurements in patients' logbooks represent what

patients choose to present to the physician. By using covert observations, the physicians took away the patients' ability to control their self-presentation and intruded into their privacy without their consent. This intrusion reduces the patients' autonomy and could be construed as a lack of respect for the person. Second, deception has the potential of undermining the physician-patient relationship. Patients who participated in the study, and those who may become acquainted with the research, may lose trust in their doctors as a result of the deception and, consequently, may be less willing to share significant information with their physicians, or they may actively misrepresent information about their adherence to the therapeutic regimen. These consequences may ultimately lead to poorer patient care.

My concerns about the use of deception in the study by Ziegler et al. are heightened because the presentation of misleading information by the researchers may not have achieved its intended goal—the reduction of measurement reactivity. One of the arguments for using deceptive practices in research studies such as Ziegler et al.'s is that if people know they are being studied (e.g., if patients' SMBG measurements are electronically stored and evaluated by researchers), they may change their behavior and act unnaturally. Researchers using deceptive practices generally argue that deception creates the necessary conditions that allow them to observe patients' natural behavior. The potential flaw in this argument is that some patients may see through the deception or attempt to guess the purpose of the study, which may have occurred in the study by Ziegler et al. if any of the 14 patients were in contact with each other and talked about the sudden "breakdown" and replacement of their meters. The implication is that we cannot be sure that the results of the study represent patients' spontaneous behavior under natural conditions. Ziegler et al. indicate that the "patients were unaware of the storage capacity of the new memory-reflectance meters," but they do not describe the procedures they used to make this determination (e.g., were patients probed to determine if they were suspicious about the true purpose of the study?). Not surprisingly, Ziegler et al. did not consider the patients' level of naiveté as a potential factor explaining the differential level of reliability in the SMBG data.

My concerns about Ziegler et al.'s study are further heightened because the weakness of their design precludes the possibility of arriving at any firm conclusions about the reliability of patient-generated data. Ziegler et al. treated the number of SMBG readings in the memory-reflectance meters as the "true" number of patient readings and viewed discrepancies between the logbooks and memory meters as evidence that the patient was overreporting ("addition of phantom values in logbook") or underreporting ("omission of SMBG measurement from logbook"). The problem with using the memory meter as the true measure of patients' self-monitoring behavior is that individuals other than the patients could have used the memory meter, and/or the