Scuba Diving and Diabetes

Practical guidelines

We have been asked to sign medical release forms for diabetic patients who wish to scuba dive. With minimal personal experience and ambivalent literature (1–4), we developed a questionnaire to gather information about individuals with diabetes who dive that has been completed by 18 active divers with insulin-dependent diabetes mellitus (IDDM). This information has helped us develop safety tips for such people. Nine women and nine men responded to our questionnaire. Their ages ranged from 14 to 46 years, with a mean of 33 years. Duration of diabetes was 2–27 years, with a mean of 12 years.

Seventeen individuals took two to four insulin injections daily, and one patient used an insulin pump. All patients monitored their blood glucose; 17 monitored more frequently the day of and the day after scuba diving. Of the 18 patients, 16 increased their carbohydrate intake the day of a dive and ate within 60 min of their dive. All patients avoided alcohol use for 24 h before diving and carried a liquid form of glucose to treat hypoglycemia on the day of the dive. Thirteen of the patients carried glucose with them when diving. No patients reported hypoglycemia during the dive. Three individuals reported a successful trial of ingesting liquid glucose during the dive.

Of the 18 patients, 17 were certified divers with from 1 month to 19 years of diving experience. Three of these individuals were certified before the onset of diabetes. Five had received a medical release stating that they had diabetes. Seventeen of the 18 patients always dive with a buddy. The adverse experiences reported by these individuals included one episode of indigestion, one episode of ear congestion, and one report of being lost under water, unrelated to hypoglycemia. The remaining 15 individuals have had no adverse experiences while diving.

We believe that individuals should be accountable for their own personal safety. In accentuating the safety of a person with diabetes who chooses to scuba dive, consider the following guidelines.

1. Blood glucose should be measured and be >150 mg/dl before the dive, and the dive should follow a meal.
2. If blood glucose before the dive is <150 mg/dl, 5 g of glucose for every 25 mg under 150 mg/dl should be ingested. Carbohydrates in the form of simple sugars should be ingested, such as fruit juice, milk, or glucose tablets/liquid.
3. The diver should carry liquid glucose in the wet suit during the dive and use as needed.
4. The diver should measure the blood glucose after the dive and ingest glucose as needed.
5. The diver should always dive with a companion who understands how to recognize and treat a hypoglycemic reaction. When under water, divers should have a pre-arranged means to communicate the likely presence of hypoglycemia.
6. Glycemic control should be stable during the days of planned diving.
7. Alcohol should not be used 24 h before or during diving activities.

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References

1. Browhard B, Travis LB, Schreiner B, Henderson S, McMahon P: Scuba diving and diabetes. *Am J Dis Child* 141:605–606
2. Brown C: Diabetes and diving. *NAUI News*. May 1978, p. 6
3. Lin L: Scuba divers with disabilities challenge medical protocols and ethics. *Physician Sportsmed* 15:224–225
4. Neuman T: Diving with diabetes. *Skin Diver*. October 1994, p. 83

Measurement of D-*chiro*-Inositol in Clinical Studies

A recent report by Suzuki et al. (1) in *Diabetes Care* relates urinary *chiro*-inositol excretion to insulin sensitivity. Since D-*chiro*-inositol may be involved in the mediation of insulin action, this is an important research area, but the conclusions depend critically on the method of measurement for D-*chiro*-inositol. The authors (1) use gas chromatography/mass spectrometry (GC/MS), a technique that is highly specific but prone to errors in quantitation under some circumstances. Their method, given by citation of previous work (2), requires that before analysis *chiro*-inositol be partially purified with quantitative recovery and that the analyzed samples produce equivalent ionization in the mass spectrometer ion source. In our own assays of D-*chiro*-inositol using several derivatives and both positive and negative ion GC/MS, we have noted that incomplete purification and/or recovery of samples often results in reduction or even complete loss of our ability to detect D-*chiro*-inositol. For this reason, we routinely add hexadeuterated *chiro*-inositol to all samples as an internal standard and we compare our results to a standard curve also containing deuterated *chiro*-inositol (3). As described in Kennington et al. (2), the authors did not use any internal standard. Thus, reduction in the amount of D-*chiro*-inositol that they observed in diabetic urine relative to that in control samples may be due to either a reduced *chiro*-inositol level, as they

suggest, or an experimental artifact for which we compensate by the use of an internal standard. In addition, the data are difficult to interpret, since the authors do not report whether they measured total *chiro*-inositol (the sum of D and L optical isomers) or the desired D enantiomer. Both occur in urine.

Although not acknowledged in the paper (1), the reader should be aware that we have found increased D-*chiro*-inositol in urine from predominantly Caucasian non-insulin-dependent and insulin-dependent diabetes subjects using quantitative GC/MS techniques with an internal standard (3). Our finding is thus exactly the opposite of the authors, who reported decreased urinary levels in non-insulin-dependent diabetes patients (1,2). In our work, elevated urinary excretion of D-*chiro*-inositol by diabetic subjects was strongly related to urinary glucose, plasma glucose, and glycated hemoglobin levels. Diabetic urine contains greatly increased quantities of many polyols and sugars (4), making trace analyses very difficult without extensive prepurification and the use of an internal standard. Thus, it is possible that the reduced urinary levels of D-*chiro*-inositol reported by the authors in diabetic subjects from both Japanese (1) and Caucasian (2) populations may have resulted from a GC/MS artifact. It would be important to resolve this question by cross-comparison of results and validation of the authors' assays. Future work published in this area should include detailed documentation of the analytical methods used.

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References

1. Suzuki S, Kawasaki H, Satoh Y, Ohtomo M, Hirai M, Hirai A, Hirai S, Onoda M, Matsumoto M, Hinokio Y, Akai H, Craig J, Larner J, Toyota T: Urinary *chiro*-inositol excretion is an index marker of insulin sensitivity in Japanese type 2 diabetes. *Diabetes Care* 12:1465–1468, 1994
2. Kennington AS, Hill CR, Craig J, Bogardus C, Raz I, Ortmeyer HK, Hansen BC, Romero G, Larner J: Low urinary *chiro*-inositol in non-insulin-dependent diabetes mellitus. *N Engl J Med* 323:373–378, 1990
3. Ostlund Jr RE, McGill JB, Herskowitz I, Kipnis DM, Santiago JV, Sherman WR: D-*chiro*-inositol metabolism in diabetes mellitus. *Proc Natl Acad Sci USA* 90:9988–9992, 1993
4. Pitkanen E: The serum polyol pattern and the urinary polyol excretion in diabetic and in uremic patients. *Clin Chim Acta* 38:221–230, 1972

The Effect of Hyperglycemia on Glipizide Absorption in NIDDM Patients

Recently, it has been described that glucose-induced hyperglycemia impairs the absorption of the sulfonylurea agent glipizide in healthy young subjects (1). Therefore, the absorption and therapeutic activity of this drug in diabetic patients could be affected by metabolic state. The current study examined whether the absorption rate of glipizide in non-insulin-dependent diabetes mellitus (NIDDM) subjects would be increased following insulin-induced euglycemia.

Seven patients (five female and two male) with NIDDM who started insulin therapy because of sulfonylurea failure were studied. Their mean age was 46.0 ± 3.0 years, duration of diabetes was 13.5 ± 4.2 years, body mass index (BMI) was 30.2 ± 1.9 kg/m^2, and HbA_{1c} was 10.1 ± 0.3%. The existence of upper gastrointestinal surgery, peptic ulcer disease, or autonomic neuropathy were considered exclusion criteria. No patient had clinical signs or symptoms of renal or hepatic disease, and serum creatinine and liver enzyme levels were normal in every case. Two patients presented background retinopathy, and one patient had microalbuminuria (urinary albumin excretion: 30–300 μg/min).

The study was initiated after subjects were hospitalized for two days and received an isocaloric diet as the only diabetic treatment. Glipizide absorption was measured on two consecutive days, in hyperglycemic and euglycemic conditions, respectively. On day 1, after a sample for measurement of baseline plasma glucose and glipizide was obtained, a 5-mg tablet of glipizide was given with 200 ml of water. Thereafter, samples for measurement of plasma glucose were taken every 15 min and for plasma glipizide at 15, 45, 90, and 150 min. When plasma glucose decreased to <250 mg/dl, a 20% glucose infusion was started through a contralateral antecubital venous catheter adjusted to maintain hyperglycemia (250–350 mg/dl). After the study, insulin infusion was started to maintain the euglycemic state for 24 h (80–120 mg/dl). With the subjects in this condition, the study was repeated on day 2. Serum glipizide concentration was measured by high-performance liquid chromatography (2).

On day 1, mean blood glucose during the study was 271 ± 18 mg/dl, and during the second day it was 94 ± 3 mg/dl ($P < 0.001$). Peak concentrations of glipizide were 0.59 ± 0.30 (hyperglycemia) and 0.77 ± 0.20 (euglycemia) μg/ml. During euglycemia, glipizide appeared earlier in serum (47 ± 8 vs. 90 ± 18 min, $P < 0.05$) (Table 1). In both glycemic conditions, a plateau of the glipizide level was achieved between 90 and 150 min after ingestion of the drug (Table 1). There was no relationship between age, duration of diabetes, HbA_{1c}, or BMI and glipizide absorption.

Our results indicate that hyper-