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Little Risk of Hyperosmolar Coma Following Hyperglycemia during Cardiopulmonary Bypass

To the Editor:—In their review, Hirsch *et al.*[1] suggest that hyperglycemia during cardiac operation can lead to postoperative hyperosmolar coma. In the accompanying editorial, Alberti[2] states that a deleterious effect of intraoperative hyperglycemia is hyperosmolarity but does not further define the adverse consequences of hyperosmolarity. The references cited by Hirsch *et al.* fail to support a relationship between intraoperative hyperglycemia and postoperative hyperosmolar coma, and we have been unable to identify an adverse consequence of intraoperative hyperosmolarity.[3]

Seki[4] searched the world's literature and his personal experience to find twelve instances of hyperosmolar coma following cardiac operation. In his reported cases, an average of 10 days of unrelenting postoperative drug-induced diuresis coupled with a high osmolar load produced dehydration, hyperosmolarity (average 383 mOsm/l), and coma. We[3] recently reported osmolarity data from 107 nondiabetic patients undergoing elective coronary artery bypass grafting, half receiving glucose-containing intravenous and priming solutions and half receiving glucose-free solutions. Despite glucose concentrations greater than 800 mg/dl during cardiopulmonary bypass in the glucose group, the maximum osmolarity observed during operation was 313 mOsm/l. Hyperglycemia produced intraoperative diuresis, and by the end of operation, mean osmolarity was 300 mOsm/l. The highest osmolarity values during operation did not approach the values reported in hyperosmolar coma (370–390 mOsm/l) for the medical and surgical patients who suffered sustained fluid losses for days to weeks before developing coma.[5,6] In patients undergoing cardiac operations with marked intraoperative hyperglycemia, it is highly unlikely that glucose-induced osmotic diuresis could be so severe as to produce dehydration and coma within 24 h. Seki's inability to find more than 12 instances of postoperative hyperosmolar coma suggests that this complication in cardiac patients is exceedingly rare. Our own experience of approximately 70,000 diabetic and nondiabetic patients undergoing cardiopulmonary bypass with glucose-containing priming and intravenous solutions confirms this rarity, since in no patient who failed to awaken after operation could the diagnosis of hyperosmolar coma be made.

Although hyperosmolar coma is a well-known complication of uncontrolled diabetes, neither the authors of the review article nor the editorial documents any hazard of intraoperative hyperosmolarity or even postulates how transient intraoperative hyperglycemia could lead to hyperosmolar coma in the early postoperative period.

SAMUEL METZ, M.D.
Division of Cardiovascular Anesthesiology
Texas Heart Institute
P.O. Box 20466
Houston, Texas 77025

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In Reply:—We thank Metz for his thoughtful comments regarding the risks of perioperative hyperglycemia and hyperosmolar coma in patients undergoing elective coronary artery bypass grafting. However, we must take issue with his statement that our references do not support a relationship between intraoperative hyperglycemia and postoperative hyperosmolar coma in *diabetic* patients. Brenner *et al.*[1] noted ten patients with hyperosmolar coma during a 2-yr period following surgery. Interestingly, three of these patients had coronary artery bypass surgery, and one of the conclusions from their study was that this surgical procedure is a risk factor for postoperative hyperosmolar coma.

With careful monitoring of diabetic patients during and after surgery as recommended in our review,[2] we would agree with Metz that hyperosmolar coma is indeed a rare event following coronary artery bypass surgery. While profound hyperglycemia did not produce a hyperosmolar state in *nondiabetic* patients undergoing elective coronary artery surgery,[3] our review article[2] and the accompanying editorial by Alberti[4] discussed potential complications in patients *with diabetes mellitus.* How high would the blood glucose concentration and serum osmolality have risen in a diabetic population receiving a similar glucose load during cardiac surgery? Secondly, as Metz correctly points out, his patients were only slightly hyperosmolar and most certainly not dehydrated (the serum sodium was calculated to be 133 mM based on a plasma glucose of 836 mg/dl and a serum osmolality of 313 mOsm/kg).

While we agree with Lanier's comment that "there is a large body of convincing data that glucose worsens outcome from cerebral ischemia,"[5] hyperglycemia should be considered separately from hyperosmolarity. Although osmolarity cannot predict mortality in hyperosmolar coma,[6,7] previous studies suggest that hyperosmolarity may contribute to neurologic dysfunction.[8,9] Since adequate free water was administered intraoperatively to the patients studied by Metz and Keats,[3] it is not surprising that they did not observe hyperosmolar coma. However,

in insulinopenic patients with intraoperative hyperglycemia and the resultant diuresis, a hyperosmolar state could develop during the early postoperative period.

IRL B. HIRSCH, M.D.
Department of Medicine
University of Washington
Seattle, Washington 98195
PAUL F. WHITE, PH.D., M.D.
Department of Anesthesiology
Washington University School of Medicine
St. Louis, Missouri 63110

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In Reply:—Dr. Metz has done a valuable service with his careful analysis and review of hyperosmolarity during surgery. He shows very clearly that in the nondiabetic patient, hyperosmolar coma is extremely rare, and this is, of course, reassuring. There are two problems, however, that remain to be addressed. The first is the situation in patients with diabetes and the second, and more important, are the possible adverse effects of hyperosmolarity that fall short of hyperosmolar coma and that in turn represent the tip of a potential iceberg.

We have certainly seen hyperosmolar nonketotic coma in postoperative diabetic patients, particularly following orthopedic surgery—always the result of gross mismanagement of the diabetes as well as diabetic ketoacidosis. The difference from the nondiabetic patient is that the latter in general will compensate for hyperglycemia by hypersecretory insulin and eventually will cope metabolically. The diabetic patient cannot. What, however, of the lesser effects of hyperosmolarity. These are less well documented but remain a theoretical—and perhaps real—risk, and increased blood glucose will cause loss of fluid from cells and extracellular dilution (as evidenced by a lower sodium), which will compensate in part for the increase in osmolarity. In a compromised surgical patient this could lead to problems if there was any impairment of renal function or hypotension or if there were other electrolyte disturbances. An increase in osmolarity also has clear adverse metabolic effects on the liver.

These are, of course, largely theoretical problems and as such warranted *one word* in my editorial![1] Nonetheless, the possible problems are avoidable by sensible use of insulin and are another reason, albeit small, for avoiding hyperglycemia in surgery in the diabetic patient who cannot compensate in the same way as the nondiabetic patient.

K. G. M. M. ALBERTI, B.M., B.CH., D. PHIL., F.R.C.P.
Professor and Head of Department of Medicine
The University of Newcastle upon Tyne
The Medical School
Newcastle upon Tyne NE2 4HH United Kingdom

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Opioid Analgesics and the Burning Pain of Guillain-Barré Syndrome

To the Editor:—Recently Connelly *et al.*[1] reported on the effective treatment of deep "muscular"-type pain using epidural opioids in a patient with acute Guillain-Barré Syndrome (GBS). Pain is a common characteristic of GBS.[2] In their case report, the authors found that while deep muscle pain was effectively controlled by their intervention, the patient's burning pain, associated with areas of hyperesthesia, was not. Based on this outcome, the authors concluded that these two types of pain in GBS most likely result from two different mechanisms. The underlying assumption in drawing this conclusion is that this patient's clinical response to opioid analgesics is characteristic of GBS patients with similar pain.

Recently, a 37-yr-old man, suffering from long-standing disabling low back pain and a recurrent form of GBS,[3] came to surgery. One year after his first acute attack of GBS he suffered a recurrence. The