

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*

REFERENCES

1. Brenner WI, Lansky Z, Engelman RM, Stahl WM: Hyperosmolar coma in surgical patients: An iatrogenic disease of increasing incidence. *Ann Surg* 178:651-654, 1973

Anesthesiology
75:913, 1991

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

Anesthesiology
75:913-914, 1991

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

2. Hirsch IB, McGill JB, Cryer PE, White PF: Perioperative management of surgical patients with diabetes mellitus. *ANESTHESIOLOGY* 74:346-359, 1991
3. Metz S, Keats AS: Benefits of a glucose-containing priming solution for cardiopulmonary bypass. *Anesth Analg* 72:428-434, 1991
4. Alberti KGMM: Diabetes and surgery (editorial). *ANESTHESIOLOGY* 74:209-211, 1991
5. Lanier WL: Glucose management during cardiopulmonary bypass: Cardiovascular and neurologic implications. *Anesth Analg* 72:423-427, 1991
6. Khardori R, Soler NG: Hyperosmolar hyperglycemic nonketotic syndrome. *Am J Med* 77:899-904, 1984
7. Rimailho A, Riou B, Dadez E, Richard C, Auzepy P: Prognostic factors in hyperglycemic hyperosmolar nonketotic syndrome. *Crit Care Med* 14:552-554, 1986
8. Maccario M: Neurological dysfunction associated with nonketotic hyperglycemia. *Arch Neurol* 19:525-534, 1968
9. Vastola EF, Maccario M, Holman R: Activation of epileptogenic foci by hyperosmolality. *Neurology* 17:521-526, 1967

(Accepted for publication August 2, 1991.)

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*

REFERENCES

1. Alberti KGMM: Diabetes and surgery (editorial). *ANESTHESIOLOGY* 74:209-211, 1991

(Accepted for publication August 2, 1991.)

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