

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

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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

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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

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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.

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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

sequelae of these two episodes were mild motor impairment of the intrinsic muscles of the hands and feet, as well as significantly distressing burning pain and deep muscle pain affecting all four extremities in a diffuse distribution.* Following lumbar spinal fusion to correct the mechanical derangement causing his back pain, parenteral opioids were administered *via* a continuous intravenous infusion for postsurgical pain control. Not only did the parenteral opioids effectively manage the postoperative pain, but as a secondary outcome, they also abolished the deep muscle pain and burning pain associated with the GBS.

In light of this brief case report, Connelly *et al.*'s underlying assumption, that their patient's response to opioid analgesics was typical of GBS patients, might be in error. A case control experimental design, as used by these authors, does not allow generalization from its results. The outcome of such studies simply points the way for further research.

*Ennis JH: The physician-patient relationship: A patient-physician's view. *Canadian Family Physician* 36:2215-2220, 1990

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In Reply—We appreciate Ennis's interest in our case report, and certainly recognize individual variations in patients' responses to therapy. In general, burning, hyperesthetic pain is often believed to be neurogenic in origin, and it is well accepted that neurogenic pain responds poorly to opioids.

We certainly agree that this topic warrants further investigation.

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A Palatable Gelatin Vehicle for Midazolam and Ketamine

To the Editor—The medical community continues to search for an ideal sedative medication for patients undergoing office and surgical procedures and in the intensive care unit. When we choose a route of administration for sedative medications in patients of low chronologic or mental age, the amount of associated pain can be especially important.

Two agents increasingly used for oral sedation are midazolam and ketamine.¹⁻⁴ Unfortunately, neither of these drugs is available in oral formulation in this country, and the intravenous forms of both of these medications are quite unpalatable unless mixed with a flavoring. A recent letter to the editor proposed one formulation.² We would like to present some alternatives.

During our 5-yr experience with these medications, we have used melted PopSicle[®], orange juice, apple juice, flavoring extracts (cherry and banana), Hershey's chocolate syrup, crème de maraschino (cherry syrup), cola, and flavored gelatin with and without sugar. Although all these formulations can be used to deliver the drug, they meet with variable acceptance by the patient. In addition, nausea and vomiting have been noted in some of these patients if they are not anesthetized

after becoming sedated. Vomiting, when it occurs, occurs most often in outpatients after the sedation has begun to resolve.

At present we prefer flavored gelatin, sweetened with sugar, as the vehicle for delivery. It is used for sedation in the pediatric intensive care unit, the operating rooms, and the clinics. We have chosen the sugared form because of the problems of administering aspartame (NutraSweet[®]) to children with phenylketonuria. Our dose is 0.4-0.8 mg/kg for midazolam and 4-8 mg/kg for ketamine. Onset time is as rapid as with other flavorings (10-20 min). The duration of sedation varies from 20 min to 3 h.

The gelatin mixture is made in ice cube trays. It is prepared by adding ¼ cup of boiling water to a small package of flavored gelatin (one that makes 2 cups) and allowing this to cool at least to 40°C. The liquid gelatin is then added to the drug in a ratio of at least 1.3 ml gelatin to every 1 ml drug. Cubes are made containing 5, 10, or 15 mg midazolam or 100 or 250 mg ketamine, and the mixture is allowed to set in a refrigerator. Once set, the gelatin may be administered as prepared or may be cut into portions if fractional doses are needed (for example, a 5-mg cube is cut in two to provide 2.5 mg). The pH

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