These patients may experience ulnar neuropathic symptoms during the perioperative period. Third, many patients do not notice or complain of ulnar neuropathic symptoms until more than 24 h after their surgical procedures.1″ We suggested several reasons for this in our report; one may be the use of postoperative sedatives in patients resting for prolonged periods in a supine position, a reason similar to that noted by Kempken.

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Should Acidosis during Liver Transplantation Be Treated?

To the Editor.—Shangraw et al. described the use of dichloracetate during liver transplantation (OLTX).1 Although we agree with the authors’ concerns that too much NaHCO3 is potentially problematic, we have taken a different approach to lactic acidosis during liver transplantation.

There is conflicting evidence in the literature concerning the potential harm of acidosis.2 Much evidence exists that there are no significant enhancements to myocardial performance and responsiveness to catecholamines as long as the pH is greater than 7.1.3 Additionally, we believe that the acidosis in OLTX differs from most lactic acidoses in that it usually arises as a result of inadequate clearance by the diseased or excised liver rather than a situation where excessive production from tissue hypoperfusion or hypoxia overwhelms a normal liver. Although lactic acidosis during OLTX may result from tissue hypoperfusion, much of the lactic acid load results from administration of banked blood.4 With this in mind, we have elected not to treat acidosis except in the rare cases when the patient has significant cardiac rhythm disturbances or severe fulminant liver disease for which bicarbonate infusion was started preoperatively in the intensive care unit.

We have cared for more than 250 liver transplant patients without correcting acidosis and have not made any attempts to correct pH with ventilation. Blood pH normally decreases to less than 7.30, which is the threshold for treatment in other centers and was used in Shangraw et al.’s study.5 In those rare instances (about six cases) when bicarbonate was given to treat acidosis, we noted no subsequent changes in hemodynamics. This is consistent with observations of others treating lactic acidosis in nontransplant settings.6,7 We therefore eliminated the potential problems of hypernatremia and metabolic alkalosis intraoperatively and any contribution that intraoperative bicarbonate therapy makes toward abnormalities in the postoperative period.

In summary, we agree with Shangraw et al. that the administration of large doses of sodium bicarbonate should be avoided, but we believe the goal usually can be achieved by simply resetting our setpoints for the lower limits of tolerable pH. In our experience, a pH greater than 7.10 is tolerated by the majority of patients undergoing OLTX without significant hemodynamic instability.

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In Reply:—Bogdonoff and Speiermann raise an interesting point regarding the use of NaHCO3 in the operating room, particularly during liver transplantation. They rarely use NaHCO3, employing a less aggressive protocol than that used in our study.1 The data fully support a less aggressive approach, and since our original study, we have relaxed our criteria for the administration of NaHCO3 as well. We found there are no apparent hemodynamic consequences to allowing the arterial pH to decrease to 7.2, although liver transplantation is not the ideal setting in which to evaluate the effects of incremental changes in arterial pH. We also noticed that hyperkalemia may be exacerbated at lower arterial pH. On the other hand, the use of dichloroacetate has continued to ameliorate the acid-base changes during liver transplantation, in part by stimulating pyruvate dehydrogenase activity in the native liver before heparectomy.2 Thus, dichloroacetate still may be beneficial in moderating the severity of lactic acidosis during liver transplantation.

We agree that the mechanism of lactic acidosis during liver transplantation does not necessitate reflect tissue hypoperfusion but more typically represents an exogenous lactic acid challenge and excess nonanabolic peripheral lactic acid production in response to a glucose challenge.1 Two animal studies on the effect of metabolic acid challenge on cardiac function are germane.3,4 First, Teplinsky et al. induced lactic acidosis in anesthetized dogs by infusing 0.5 N lactic acid to obtain arterial pH of 7.5, 7.2, or 7.1.3 They found that, compared to controls maintained at pH 7.4, cardiac output decreased at pH 7.3 (by 14%), 7.2 (by 20%), and 7.1 (by 29%), which occurred secondary to decreased stroke volume and impaired contractility (dp/dtmax). Further, there was an accompanying pulmonary hypertension at pH 7.2 and 7.1 and an increase in right atrial pressure at pH 7.1, indicating that the lactic acid-induced decrease in cardiac output is not the result of hypovolemia but rather a direct effect on pump function.

Zhou et al. expanded on these findings in an isolated perfused rat heart model.1 These authors induced metabolic acidosis by employing an HCO3-depleted perfusate (pH 6.8, HCO3 6; analogous to our transfused reservoir blood) and used nuclear magnetic resonance spectroscopy to evaluate metabolic function coincidentally with hemodynamic measurements. They noted a marked decrease in dp/dt (by 70% by 30 min) and intracellular pH, followed by decreases in creatine phosphate and oxygen consumption and a 50% increase in inorganic phosphate. Further, reperfusion of acidic hearts with "normal" perfuse led to recovery of dp/dt and intracellular pH, followed by repletion of creatine phosphate from inorganic phosphate. Similar changes were observed when the acidic perfuse pH was neutralized with Carbicarb (International Medication Systems, South El Monte, CA). Thus, correction of an acidic extracellular pH has a direct, positive isotropic effect on the myocardium.

The reasoning behind the treatment of pH 7.5 is that, until the graft liver is reperfused, the acidosis is not self-limited and is likely to get worse before it improves. This approach is comparable to treating hypotension after spinal anesthesia at a mean arterial pressure above that considered critical for end-organ perfusion. We agree that it is not necessary to choose pH 7.5 as the value to begin NaHCO3 therapy. However, our experience has been that, regardless of the pH used as the threshold at which NaHCO3 therapy is begun, dichloroacetate decreases NaHCO3 utilization because it stabilizes arterial pH.

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