proper intraoperative positioning, this usually is recognized, and preventive measures are implemented. Stoelting conjectures that "unavoidable events associated with anesthesia and surgery" might cause injury.[2] Prolonged recuperation after major surgery can occur and is associated with significant changes in personal habits and levels of consciousness.

Case Report. I (age 37–39 yr, ASA physical status 1, 85 kg) underwent two major laparotomies *via* xiphoid-suprapubic midline incision with hospitalization of 1 week each time. Customarily, I sleep prone. Postoperatively, I found that I slept exclusively supine with my hands positioned across my lap, causing me to wake frequently with numbness and paresthesia of the fourth and fifth digits, at times bilaterally. Arousal and active motion of the hands resolved this transient problem without permanent lesions. After the third postoperative day, the skin over both olecranons was chaffed and painful (because of supine movement using the elbows), and I noticed the symptoms occurring during the day, generally while sitting in an armchair, as I was positioning my elbows on the medial epicondule to avoid pain from the raw skin over the olcranons. I began to sleep laterally for the same reason. At home, I ingested 0.25 mg triazolam HS (instead of 25 mg diphenhydramine used in the hospital) and awoke the next day with deep pain over the left greater trochantor, as if I had bruised my hip during sleep. The next night, after the same dose of triazolam, the same pain over my right hip developed. I attribute the pain to deep pressure scores secondary to benzodiazepine central nervous system depression. Since the surgery, I cannot sleep prone and, frequently, am awakened supine with ulnar paresthesias, even while sleeping on the padded "egg crate" mattress, provided while I was "on call" in the hospital.

Discussion. Intraoperative ulnar nerve protection routinely is discontinued with placement of the patient onto the gurney and throughout subsequent convalescence. The search for causative mechanisms of ulnar neuropathy during this period in previous studies has not occurred.[1,3–5] Prolonged supine positioning alone may result in cumulative and definitive injury, because personal habits including elbow-leaning have been proposed as causative mechanisms.[6] Clearly, my convalescence was associated with significant elbow-leaning.

Our inability to understand this process to date may be a result of examining the problem only as an anesthetic complication. Contemporary ulnar neuropathy may occur because of absent concerns for postoperative protection, compounded by frequent administration of sedatives, analgesics, or neuromuscular blocking agents. I hope Warner *et al.* will reexamine the available data in this new light, given their unique database.

Paul Martin Kempen, M.D., Ph.D.
Associate Professor
Co-Director of Obstetric Anesthesia
Department of Anesthesiology
Louisiana State University Medical Center
P.O. Box 33932
Shreveport, Louisiana 71130-3932

References

1. Warner MA, Warner ME, Martin JT: Ulnar neuropathy: Incidence, outcome, and risk factors in sedated or anesthetized patients. ANESTHESIOLOGY 81:1332–1340, 1994

2. Stoelting RK: Postoperative ulnar nerve palsey—is it a preventable complication? Anesth Analg 76:7–9, 1993

3. Wey JM, Guinn GA: Ulnar injury with open heart surgery. Ann Thorac Surg 39:358–360, 1985

4. Roy RC, Stafford MA, Charlton JE: Nerve injury and musculoskeletal complaints after cardiac surgery: Anesth Analg 67:277–279, 1988

5. Alvine FG, Schurrer ME: Postoperative ulnar-nerve palsy: Are there predisposing factors? J Bone Joint Surg 69:255–259, 1987

6. Miller RG: The cubital tunnel syndrome: Diagnosis and precise localization. Ann Neurol 6:56–59, 1979

(Accepted for publication March 21, 1995.)

Anesthesiology
82:1539–1540, 1995

In Reply:—We appreciate Kempen's interest in our study and enjoyed reading his anecdotal report. We believe that his case is not unusual and presents several patient care issues that may be associated with the development of ulnar neuropathies.

Improper anesthetic care and patient malpositioning have been implicated as causative factors in the development of ulnar neuropathies since reports by Büdinger[1] and Garriques[2] in the 1890s. These factors likely play an etiologic role for this problem in some surgical patients. However, other determinants may contribute to the development of postoperative ulnar neuropathies. In a series of 12 inpatients with a newly acquired ulnar neuropathy, Wadsworth and Williams[3] determined that external compression of an ulnar nerve during surgery was contributory in only two patients. At the Mayo Clinic, a number of nonsurgical patients experience ulnar neuropathies during inpatient and outpatient treatment. We are investigating the incidence and outcomes of these neuropathies. Although our investigation is not complete, it is clear that both surgical *and* medical patients may experience ulnar neuropathies during or after an episode of care.

Many factors may be associated with the development of ulnar neuropathy. Although the final pathologic event usually is nerve ischemia or trauma (*e.g.,* myelin sheath or nerve fiber disruption), etiologic mechanisms may include external nerve compression or stretch, generalized or site-specific hypoperfusion, or metabolic/genetic predisposition to neuropathy.[4,5]

Typically, anesthesia-related ulnar nerve injury is thought to be associated with external nerve compression or stretch caused by malpositioning. Although this implication may be true for some patients, three considerations suggest that other factors may contribute. First, we found several patient-related characteristics[6] to be associated with these ulnar neuropathies. Second, a high incidence of contralateral ulnar nerve conduction dysfunction in patients with postoperative ulnar neuropathies suggests that many of these patients likely have abnormal ulnar nerves before their anesthetics but are asymptomatic.[7]

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.[3,6] 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 Kempen.

Mark A. Warner, M.D.
Associate Professor of Anesthesiology
Mary E. Warner, M.D.
Assistant Professor of Anesthesiology
Mayo Clinic
Rochester, Minnesota 55905

John T. Martin, M.D.
Professor Emeritus
Department of Anesthesiology
Medical College of Ohio at Toledo
3000 Arlington Avenue
Toledo, Ohio 43614

References

1. Büdinger K: Ueber Lähmungen nach chloroform-narkosen. Arch f Kin Chir 47:121–145, 1894

2. Garriques HJ: Anaesthesia-paralysis. Am J Med Sci 113:81–89, 1897

3. Wadsworth TG, Williams JR: Cubital tunnel external compression syndrome. Br Med J 1:662–666, 1973

4. Dawson DM, Krarup C: Perioperative nerve lesions. Arch Neurol 46:1355–1360, 1989

5. Lupski JR, Chance PF, Garcia CA: Inherited primary peripheral neuropathies: Molecular genetics and clinical implications of CMT1A and HNPP. JAMA 270:2326–2330, 1993

6. Warner MA, Warner ME, Martin JT: Ulnar neuropathy: Incidence, outcome, and risk factors in sedated or anesthetized patients. ANESTHESIOLOGY 81:1332–1340, 1994

7. Alvine FG, Schurrer ME: Postoperative ulnar-nerve palsy: Are there predisposing factors? J Bone Joint Surg 69:255–259, 1987

(Accepted for publication March 21, 1995.)

Anesthesiology
82:1540–1541, 1995

J. B. Lippincott Company, Philadelphia

Should Acidosis during Liver Transplantation Be Treated?

To the Editor:—Shangraw *et al.* described the use of dichloroacetate during liver transplantation (OLTX).[1] Although we agree with the authors' concerns that too much $NaHCO_3$ 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 *p*H 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.[1] 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 *p*H with ventilation. Blood *p*H commonly decreases to less than 7.30, which is the threshold for treatment in other centers and was used in Shangraw *et al.*'s study.[1] 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.[4,5] 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 *p*H. In our experience, a *p*H greater than 7.10 is tolerated by the majority of patients undergoing OLTX without significant hemodynamic instability.

David L. Bogdonoff, M.D.
Assistant Professor of Anesthesiology and Surgery
Burkhard F. Spiekermann, M.D.
Assistant Professor of Anesthesiology
Box 238
Department of Anesthesiology
University of Virginia Health Sciences Center
Charlottesville, Virginia 22908