ATSUO F. FUKUNAGA, M.D., Ph.D. *Professor of Anesthesiology*

YOSHIO TANIGUCHI, M.D. YOSHINORI KIKUTA, M.D. Research Fellows in Anesthesiology Department of Anesthesiology UCLA School of Medicine Harbor/UCLA Medical Center 1000 W. Carson St. Torrance, California 90509

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A Pain in the Neck—and Shoulder

To the Editor:—The phrenic nerve has been injured and anesthetized accidentally on several occasions during central venous cannulation. Permanent injury to the phrenic nerve during internal jugular vein cannulation was reported first in 1980.1 In 1982, we reported a case of transient diaphragmatic paralysis² similar to that reported by Schiessler et al., sexcept that our patient experienced respiratory distress and required therapy with continuous positive airway pressure. This patient was our fourth personally observed case of diaphragmatic weakness following internal jugular vein cannulation. Subsequently, we have treated another such case. It is possible that other patients have had similar results but were asymptomatic, thus preventing detection. Perhaps the complication is much more frequent than previously suspected. Both in our report and during other nonreported occasions, most patients have complained of shoulder pain prior to the injection of local anesthetic. Therefore, when patients complain of pain during the cannulation of the internal jugular vein, they should be asked to locate the pain. If the pain is identified in the shoulder, it is likely that the phrenic nerve is being stimulated or injured by the tip of the needle, which should be redirected. Further, no local anesthetic should be injected until the needle is repositioned and the patient is free of shoulder pain.

M. CHRISTINE STOCK, M.D. Assistant Professor of Anesthesiology

Department of Anesthesiology Emory University School of Medicine 1365 Clifton Road, N.E. Atlanta, Georgia 30322

JOHN B. DOWNS, M.D.
Professor and Chairman
Department of Anesthesiology
University of South Florida
12901 Bruce B. Downs Boulevard
Tampa, Florida 33612-4799

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Cardiac Arrest in a Day Surgery Patient

To the Editor:—In a recent Case Report, Dr. Hanson suggested that a presumed cardiac arrest during induction of general anesthesia in the Day Surgery Unit (DSU) at the University of Pennsylvania might have been avoided by a more thorough preoperative evaluation.

First, from the description provided in the Case Report, it is not clear that the patient even experienced a cardiac arrest. Both the automated blood pressure cuff and the pulse oximeter are unreliable in the presence of acute hypotension and bradycardia. Second, to suggest

that a combination of thiopental, 600 mg (6 mg/kg) iv, and lidocaine, 400 mg (4 mg/kg) iv is a "standard" induction dosage regimen for unpremedicated outpatients is inappropriate in our opinion. The cardiovascular depressant effects of this drug combination could produce profound hypotension and bradycardia in otherwise healthy fasted outpatients. Finally, it is not clear how "admitting this patient to the hospital on the night prior to surgery and ordering a routine chest film, electrolyte panel, complete blood count, urine analysis, and electrocardiogram" would have prevented the problem. We are not convinced that a "borderline" chest film and electrocardiogram would have led to a more exhaustive evaluation of an apparently healthy, active young man. However, if one chooses to eliminate routinely ordered preoperative screening laboratory tests, it is essential that a thorough history and physical exam be performed prior to ambulatory surgery.

Apparently the "moderate" mitral regurgitation that was noted on the echocardiogram was not detected during cardiac auscultation. In addition, the history of alcohol usage was not elicited, and consequently, the search for alcoholic cardiomyopathy or hepatic insufficiency was not instituted. Were the two best questions to search for alcohol usage²—"Have you had a drink in the last 24 hours?" and "Have you ever had a drinking problem"—even asked? Were these questions included on the health survey this patient was given? We would recommend that these questions be included on every health survey designed specifically for preoperative purposes. While being thorough is often difficult in the brief time we spend preoperatively with surgical outpatients, either a check-off form or an automated system can be used to improve our efficiency.

In summary, it seems to us that the problem described might have been avoided had either an adequate preoperative history been taken (and testing indicated by it pursued) or more usual doses of the iv anesthetics been administered (e.g., 3–4 mg/kg of thiopental with or without 1–2 mg/kg of lidocaine). After all, didn't this problem result from an anesthetic overdosage rather than inadequate preoperative laboratory testing?

PAUL F. WHITE, PH.D., M.D.
Professor of Anesthesiology
Director of Clinical Research
Department of Anesthesiology

Washington University School of Medicine St. Louis, Missouri 63110

MICHAEL F. ROIZEN, M.D.

Professor and Chairperson Department of Anesthesia University of Chicago Chicago, Illinois 60637

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Cardiac Effects of Anabolic Steroids

To the Editor:—I read with great interest the case reported by Hanson1 wherein a presumably healthy individual harboring a significant cardiomyopathy that becomes evident at the time of induction of anesthesia. By all appearances a more rigorous work-up preoperatively would not be called for based upon the present accepted standards of care. There is, however, one element here that may require greater investigation. Historically this patient is a fit individual suggested by the history of semiprofessional ice hockey play and continued weekend softball games. Recent literature has documented the prevalence of use of anabolic steroids among participants of many age groups in sports.^{2,3} The effects of anabolic steroid use on the heart include increased risk of coronary artery disease and hence infarction secondary to changes in lipid metabolism and concentric myocardial hypertrophy with decreased ventricular volume possibly altering pressure-volume relationships within the heart.3-5 There is no information I am aware of regarding long-term changes in myocardial function after discontinuation of these drugs. As part of the preoperative history and physical

examination evidence of involvement (current or past) in organized sports or weight lifting should suggest the question of androgenic steroid use; if this is elicited further, cardiac work up may be needed such as an ECG and chest x-ray or even echocardiogram if the former are suspicious. There is no mention of steroid use in this instance so we have no way of knowing if this is the case; the patient refused follow up leaving several unanswered questions. I bring this to the attention of the reader as a history of sports activity may not only indicate health and fitness but disease as well.

ZVI HERSCHMAN, M.D. Director, SICU
B-1 SICU
Elmhurst Hospital Center
79-01 Broadway
Elmhurst, New York 11373