Our patients, being Japanese, are considerably smaller in height or weight than average American patients.

2) The 6 ml we used is within the doses of 5–8 ml used by Dr. Bauman. Considering again the difference in size of patients of the two groups (Dr. Bauman's and ours), our doses may be more than the average he used. We do not believe that our technique of performing epidural block is substantially poorer than his.

3) We did give about 10 ml of 1% mepivacaine, as it is our routine to use a somewhat larger volume for caudal epidural block than for lumbar epidural block. We omitted this in the manuscript, because we considered it to be common knowledge, and stating it in detail complicates the matter unnecessarily.

4) For stellate ganglion block, we generally check for the presence of Horner's syndrome. For other blocks, we do not obtain any "scientific evidence" of sympathetic block to individual patients. We simply assume that we obtain the sympathetic block as long as we block it properly.

5) Giving daily blocks to patients with herpetic disease has two aspects: one is relieving the patient from pain, the other is the prophylaxis against postherpetic neuralgia. We gave daily blocks with the belief that such prophylaxis would occur, but failed to substantiate this, as stated in our article. We have no intention to insist, especially after analyzing our own data, that the patient of herpes zoster should be blocked on a daily basis. We respect Dr. Bauman's opinion of treating this disease, but request that he substantiate his opinion with scientific data. So far, he has done so only vaguely with a one-page abstract.

6) We do not believe that there is a way of preventing the postherpetic neuralgia with 100% certainty. Incidence may decrease by certain treatment or treatments, but patients still develop postherpetic neuralgia. We hope that Dr. Bauman substantiates his conviction by scientific data, writes a good full-length paper, and publishes it in a respectable journal.

We are not sure if we feel grateful to Doctor Bauman for supporting the last two lines of our table 4. It is not fair to split somebody's data and to use it to his liking. We can turn his comment around by focusing on that one patient of the very last line, who ended up with postherpetic neuralgia despite the block started at day 10, which is contrary to what Dr. Bauman is insisting.

HISASHI YANAGIDA, M.D.
Chairman
Department of Pain Clinic and Anesthesia
Tokyo Kosei Nenkin Hospital
Tokyo, Japan, and
Permanent Visiting Professor of Anesthesiology
Erasmus University
Rotterdam, The Netherlands

KUNIO SUWA, M.D.
Associate Professor
Department of Anesthesiology
Faculty of Medicine
University of Tokyo
Tokyo, Japan

(Accepted for publication April 20, 1987.)

Anesthesiology
67:442–443, 1987

Prolongation of QT Interval in Association with the Administration of Large Doses of Opiates

To The Editor:—Studies in our laboratories indicated that the opioids, fentanyl and sufentanil, produce significant prolongation in canine cardiac action potential duration.[1] Changes found are consistent with a delay in repolarization similar to antiarrhythmic drugs, such as quinidine. The association between action potential duration and the QT interval led us to evaluate the electrocardiogram of a patient who received sufentanil for induction and maintenance of anesthesia.[2] We present a case of prolongation of Q-Tc in association with the administration of a high-dose narcotic anesthetic. A 61-yr-old male with a history of angina presented for coronary artery bypass surgery. Current medications include metoprolol 50 mg po bid, diltiazem 60 mg po tid, nitroglycerine patch 2 inches q 6H. Anesthesia was induced with an iv infusion of sufentanil 24 μg/kg over 90 min. Electrocardiogram recordings were made 24 h prior to surgery, prior to induction, and at 10-min intervals for the first 90 min prior to bypass. QTc was calculated according to the standard formula of QT interval divided by the square root of the preceding R-R interval. Ten minutes after the infusion was begun, we found the QTc prolonged to .410 s at a total dose of 35 μg (0.4 μg/kg, fig. 1). Twenty minutes after induction and at a dose of 7 μg/kg, Q-Tc was prolonged to .440 s, which is considered the upper limit of the acceptable range.[3] QTc did not change significantly until the 50-min interval. At a dose of 16 μg/kg, the Q-Tc was measured at .470 s. Following a total dose of 24 μg/kg, Q-Tc at 90 min was .483 s.

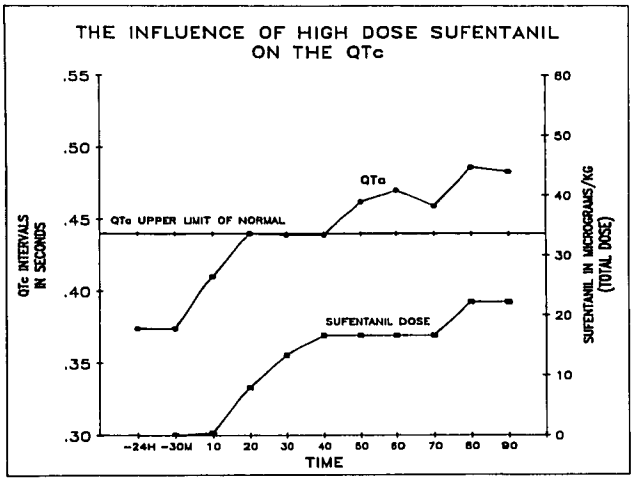

FIG. 1. Values for the QTc are shown on the left vertical axis, values for total sufentanil dose are shown on the right vertical axis. −24H = QTc 24 h prior to surgery; −30m = QTc 30 min prior to induction. Measurements 10 through 90 are minutes post-induction. Upper limit of normal for QTc is 0.440 s.

This case illustrates that narcotics in clinical doses may produce prolongation in QT interval. This patient clearly displays lengthening in Q-Tc after administration of the sufentanil during the anesthetic induction and maintenance period prior to cardiopulmonary bypass. We speculate that the observed change in QTc resulted from a prolongation of ventricular repolarization produced by the high-dose narcotic anesthetic. The electrophysiologic activity of the narcotic anesthetic described in this case appears to be antiarrhythmic in character, and might be beneficial. It is our concern that narcotics used in high doses may have an additive effect in patients with prolonged QT interval (*e.g.*, patients on quinidine therapy). We would suggest that one should be cautious when using high doses of narcotics in patients with congenital or required abnormalities of QT interval who may be predisposed to premature contractions and subsequent R on T phenomenon.

JOHN R. BLAIR, D.M.D., M.D.
Assistant Professor

JACK K. PRUETT, PH.D.
Professor and Senior Scientist

ROBERT S. CRUMRINE, M.D.
Professor and Chairman

JEFFREY S. BALSER, B.S.

Department of Anesthesiology
Medical College of Georgia
Augusta, Georgia 30912

REFERENCES

1. Blair JR, Pruett JK, Adams RJ, Crumrine RS: The electrophysiologic effects of opiates in canine cardiac purkinje fibers. ANESTHESIOLOGY 65:A406, 1986
2. Biggerm JT, Hoffman BF: Antiarrhythmic drugs. The Pharmacological Basis of Therapeutics, 7th edition. Edited by Gilman AG, Goodman LS, Rall TW, Murad F. New York, MacMillan. 1985, p 761
3. Moss AJ: Prolonged QT-interval syndromes. JAMA 256:2985–2987, 1985

(Accepted for publication April 29, 1987.)

Anesthesiology
67:443–444, 1987

Midazolam in Obstetric Anesthesia

To the Editor:—In their recent Letter to the Editor, Heyman and Salem[1] recommended postponement of midazolam administration during regional analgesia for cesarean section until after the newborn baby had been shown to the mother, to prevent complaints of amnesia for the birth experience. Modern obstetrics, however, favors conscious participation of the mother during the entire peripartum period. Today's new mothers want to remember more than a brief glimpse of the baby in the delivery room. They desire a clear recollection of their first intimate interaction with the newborn, which, in our institution, takes place approximately 3 h after completion of surgery. At this time, breast feeding may be initiated.

The amnesic effects of midazolam can be profound and prolonged.[2,3] The use of midazolam in obstetrics should, therefore, be limited to special indications. With satisfactory regional analgesia, comfort of the mother during closure of uterus and abdomen can be provided by conversation, music, or a sedative without prominent amnesic properties.

STEVEN F. SEIDMAN, M.D.
Assistant Professor of Anesthesiology

GERTIE F. MARX, M.D.
Professor of Anesthesiology

Department of Anesthesiology, J-1226
Albert Einstein College of Medicine
1300 Morris Park Avenue
Bronx, New York 10461