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In Reply:—In August of 1996 Life-Tech's instrument testing procedures determined that instruments of the lot that was being tested was found to have the problem Dr. Hadzic describes. The engineering team was alerted and the problem was quickly eliminated with an addition of a capacitor change.

It was determined that a recall was not necessary because the problem posed no danger to patients. We have and continue to add the modification to instruments returned to Life-Tech. All instruments with this defect, whether or not it is returned for this complaint, have been modified at no charge to the customer.

This inconsistency was only in the DualStim Deluxe, model NS-2CA/DX and was eliminated in instruments manufactured after August 16, 1996. Due to the inability to no longer obtain metal cases for the DualStim, it has since been replaced with newly introduced MaxiStim and EZStim.

Customers experiencing this the problem described by Dr. Hadzic can call Life-Tech's service department at 1-800-231-9841.

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Area under the Compound A Concentration Curve (Compound A AUC) Analysis

To the Editor:—We read with interest the article by Kharasch *et al.*[1] In particular, we found their use of an AUC analysis to quantify compound A exposure to be an interesting approach. We, therefore, re-analyzed the data from our own publication[2] using a similar approach and found the following.

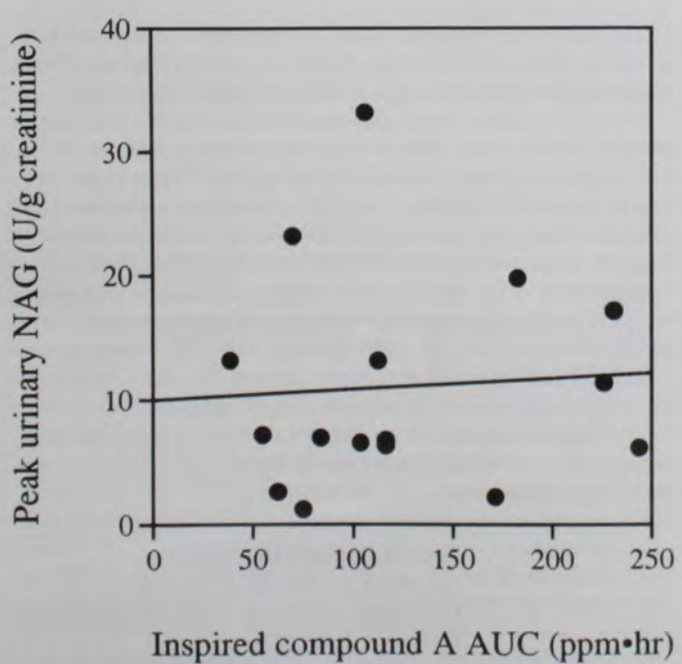

Fig. 1. Relationship between postanesthesia peak urinary NAG excretion and compound A exposure (inspired compound A AUC) in the low-flow sevoflurane group ($r^2 = 0.004, P = 0.821$).

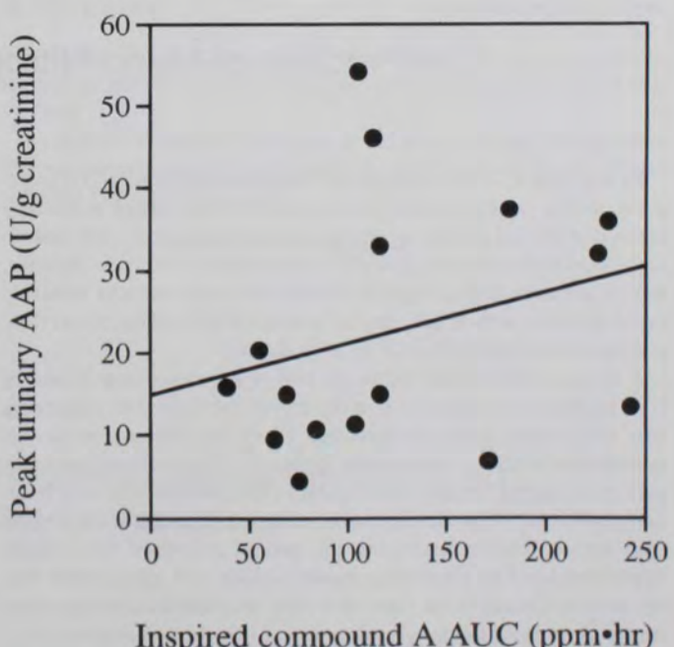

Fig. 2. Relationship between postanesthesia peak urinary AAP excretion and compound A exposure (inspired compound A AUC) in the low-flow sevoflurane group ($r^2 = 0.074, P = 0.313$).

The area under the compound A concentration curve (com-

pound A AUC) was calculated as the product of inspired compound A concentration and duration of exposure, determined at 1-h intervals, using the rhomboid rule. In the low-flow sevoflurane group, the correlations between inspired compound A AUC and both postanesthesia peak NAG and AAP were evaluated.

The mean compound A AUC values in the low- and high-flow sevoflurane groups were 124.4 ± 66.1 ppm · h (mean ± SD; range, 38.4–243.0) and 1.4 ± 0.6 ppm · h (0.6–2.5), respectively ($P < 0.01$). There was no significant correlation between compound A AUC and either postanesthesia peak urinary excretion of NAG or AAP in the low-flow sevoflurane group (figs. 1 and 2).

The mean compound A AUC value in the present study was 1.5 times higher than that in the study of Kharasch *et al.* Nevertheless there was no correlation between inspired compound A AUC and postanesthesia NAG excretion, which is in agreement with the study of Kharasch *et al.* These data suggest that the exposure dose of compound A was not related to the increase in postanesthesia urinary excretion of NAG or AAP and these increases in NAG and AAP were therefore not due to compound A exposure. These results emphasize that low-flow anesthesia with sevoflurane, in which compound A is formed by the degradation of sevoflurane, does not appear to be associated with renal tubular injury.

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The Practice of Using Nitroglycerin for Uterine Relaxation

To the Editor:—The practice of using nitroglycerin (NTG) to reduce uterine smooth muscle tension acutely, has gained popularity in the past several yr. Unfortunately, we still lack reproducible quantitative data demonstrating that this intervention is effective. Further, the intravenous administration of NTG definitely reduces maternal blood pressure which may impact negatively on uterine blood flow at times when fetal oxygenation is challenged.

A recent Letter to the Editor by Bell *et al.*, describing a case of NTG induced acute relaxation of the uterus, included data suggesting that NTG might have a measurable effect on uterine contraction quantifiable with an intrauterine catheter.[1] The maximal pressure generated during uterine contraction in this patient was clearly reduced after the NTG administration. Although frequency and resting tone appeared to be unaltered, the patient described was initially hyperstimulated by pitocin (probable half-life 3–5 min) which was discontinued at about the same time NTG was administered clouding the cause of this effect.

Work in gravid rabbits and ewes in our laboratory has failed to document a reduction in the frequency of contraction, resting tone, or the maximal force generated during spontaneous or induced labor.[2] In-vitro studies by Shin demonstrated that uterine smooth muscle harvested from term pregnant rats responded to NTG only at pharmacologic concentrations.[3] A study by Kumar in 1965 which examined the effect of amyl nitrate on uterine tension as examined by tocodynometry similarly failed to demonstrate a measurable response to nitrosovasodilators.[4] In that study, intrauterine pressure was also monitored in women induced to labor with pitocin.

Recent data from a very different in-vitro model using uterine smooth muscle strips from primigravida rabbits at term show that the compliance of the uterus is altered by NTG.[5] Prior to Dr. Bell's case all reports of the efficacy of NTG involved the application of an external force to the uterus by the obstetrician. In a hypercontractile state, the sustained generation of active tension would be analogous to application of an external force. Hence, a change in compliance would be expected to appear as a decrease in maximal tension during sustained contraction. Dr. Bell's findings then are consistent with this recent data from our laboratory. Although Dr. Bell's report only describes a single patient, it presents an appealing model to consider (ie, the hyperstimulated uterus may be a model in which a change in compliance resulting from the administration of NTG can be reproducibly demonstrated).

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