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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(Accepted for publication May 12, 1997.)

Anesthesiology
1997; 87:716–7

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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(Accepted for publication May 21, 1997.)

Anesthesiology
1997; 87:717

In Reply:—Dr. Langevin correctly points out that the animal data do not consistently support the clinically observed phenomenon of uterine relaxation resulting from administration of nitroglycerin in human parturients. Dr. Shin *et al.*[1] found a dose-dependent reduction of contractility in isolated human uterine muscle segments exposed to nitroglycerin in vitro, but the mean dose to abolish spontaneous contractions was 4.5×10^{-4} M (250 μg in the 12 ml bath), far exceeding commonly used clinical doses. This may be a result of the scarcity of vascular endothelium present in these preparations.

Dr. Langevin also states that administration of nitroglycerin definitely reduces maternal blood pressure. In our experience, the administration of 50-100 μg of nitroglycerin intravenously or 800 μg sublingually has not resulted in decreased maternal blood pressure consistently, or even frequently, in urgent clinical settings. These include tetanic uterine contraction, extraction of a breech twin, or manual extraction of placenta. I suspect that the anxiety engendered by rapid interventions such as maternal position changes, increased intravenous fluid administration, application of supplemental oxygen, and summoning the obstetrician ameliorate the vasodilatory effects of the nitroglycerin. This is in contrast to our recent trial using nitroglycerin spray in the setting of elective external version of breech position.[2] We saw a high percentage of patients respond with decreased blood pressure (4 of 10 patients experienced a decrease of 20% or greater). It is unclear whether this was in response to the nitroglycerin or mechanical compression from vigorous attempts, and certainly deserves further investigation.

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(Accepted for publication May 21, 1997.)

Anesthesiology
1997; 87:717-8

Conversion of a Nasal to an Orotracheal Intubation Using an Endotracheal Tube Exchanger

To the Editor:—A 57-yr-old woman presented to the emergency room with confusion, weakness and cyanosis. Her history included a muscle biopsy and contracture test consistent with malignant hyperthermia, confirmed hypertrophic obstructive cardiomyopathy, and an undiagnosed neurologic condition characterized by progressive, episodic confusion, somnolence, dysarthria, and headache. She had been started on amiodarone and sotalol by a cardiologist for paroxysmal atrial fibrillation.

Progressive somnolence and hypercapnic acidosis led to a decision to intubate. Airway evaluation revealed mild micrognathism but no other abnormalities. Monitoring consisted of continuous electrocardiography, oximetry, and invasive arterial blood pressure. Lidocaine spray was applied to the oropharynx and hypopharynx, but direct laryngoscopy was poorly tolerated. After preoxygenation, sleep was induced with propofol. Bag and mask ventilation was easily provided, and vecuronium was administered. With appropriate positioning, di-