We speculated[1] that anesthesia-induced hepatotoxicity observed in hyperthyroid rats may be hypoxic damage to hypermetabolic centrilobular cells resulting from anesthetic-induced depression of splanchnic blood flow. This speculation is consistent with the report[3] cited by Dr. Carmichael indicating the presence of a hypermetabolic state of the liver induced by thyroxine, not T_3 as stated by Dr. Carmichael.

Smith *et al.*† have observed that the administration of cysteamine and N-acetylcysteine potentiate, whereas diethylmaleate, which depletes glutathione, reduces halothane-induced liver damage in hyperthyroid rats. Others[4] have shown that N-acetylcysteine reduces halothane hepatotoxicity in T_3 pretreated rats. Thus, the role of free radical scavangers in this setting is uncertain.

Table 4 in our article[1] shows that 36 hyperthyroid rats anesthetized with pentobarbital (not phenobarbital) and 36 nonanesthetized hyperthyroid rats (air control) did not have hepatic lesions develop. We think this is significantly different from isoflurane-, enflurane-, and halothane-anesthetized hyperthyroid rats that did not have hepatotoxicity develop.

† Smith AC, James RC, Berman ML, Harbison RD: Paradoxical effects of pertubation of intracellular glutathione on halothane-induced hepatotoxicity in hyperthyroid rats. Fundamental and Applied Toxicology: In press.

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Preoperative Management of Atrial Fibrillation

To the Editor:—Our goal as anesthesiologists is to determine if adequate rate control in atrial fibrillation has been achieved preoperatively and to maintain that control throughout the operative and postoperative period. This may require therapeutic intervention with drugs that slow atrioventricular conduction or avoidance of agents that facilitate atrioventricular conduction. The clinical report by Kopman[1] of two cases presenting for cardiac surgery with atrial fibrillation and inadequate rate control stresses these points.

Both patients presented by the author were receiving digoxin and had apparently normal digoxin levels; however, frequently a high to subtoxic level is required for adequate rate control.[2] Indeed, in past years digitalization of patients in atrial fibrillation consisted of digitalis administration until nausea and vomiting occurred, then the dosage was reduced slightly. Present day success is evaluated clinically by a ventricular response consistently less than 100 beats/min, by the absence of a significant pulse deficit, and by the lack of a rapid ventricular rate during stress testing.

Preoperative anxiety with the release of catecholamines may increase atrioventricular conduction and the ventricular response in inadequately digitalized patients. Traditionally, additional digitalis or combination therapy with propanolol is administered to these patients. More recently, the calcium channel blocker, verapamil, has been shown to be effective in controlling the rate in patients with atrial fibrillation.[3] Furthermore, a reassuring preoperative visit with an adequate preoperative sedative to allay anxiety along with the administration of digitalis the morning of surgery should facilitate rate control.

Although Kopman[1] was successful in controlling the ventricular response with verapamil, these extremely rapid ventricular rates (180–190 beats/min) strongly suggest inadequate preoperative control. The rise in blood pressure that occurred with the decrease in heart rate clearly shows the necessity in allowing for an adequate diastolic filling time to maintain cardiac output in patients with mitral valve disease who have an elevated left atrial pressure and central blood volume. In these cases, this effect more than compensated for the negative inotropy and vasodilation of verapamil. Nonetheless, caution must be used if significant hypotension is to be avoided with verapamil in the critically ill, anesthetized, or hypovolemic patient with atrial fibrillation without mitral valve disease. Adequate preoperative rate control in atrial fibrillation is desirable to avoid complications of further required therapeutic interventions.

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Questionable Partial Cure for a Minor Clinical Problem

To the Editor:—The recent paper by Loeser *et al.*[1] concerning postoperative sore throat again draws attention to the obvious. There are multifactorial mechanisms involved in the occurrence and severity of sore throat after tracheal intubation. Cuff geometry, tube design, lubricants, nasogastric tube, humidification of anesthetic gases, anticholinergic drugs, succinylcholine, patient sex, operation, and questioning method are just some of the factors known to effect incidence and severity of this relatively minor clinical problem.[1–5] Finally, interaction between factors is another important yet unknown entity.

We looked at 94 anesthetized patients intubated with unlubricated NCC hi–low cuffed tracheal tubes. Vocal cords and trachea were sprayed with 4 ml 4% lidocaine solution without preservative. Mild sore throat occurred postextubation in 28.7% of patients. Our incidence and severity are less than Loeser *et al.* report following use of hi–low cuffs lubricated with lidocaine jelly (90%)[2] and 5% lidocaine ointment (58%).[3] But, we failed to control rigidly other factors known to cause sore throat and, therefore, hesitated to publish our data. Keeping only the cuffed tracheal tube the same between groups does not provide sufficient factor control to allow definitive conclusions. Capan *et al.*[4] were able to draw conclusions from their study on succinylcholine and sore throat because other factors were controlled. If we compare our data with those of Loeser *et al.* it appears we have a partial cure for a mild problem. But, this conclusion is questionable because of study design.

In 1941, Murphy suggested use of a "neutral water soluble tube lubricant"[6] and Menias reported in 1977 that postoperative sore throats were uncommon when nonanesthetic lubricants such as Lubrifax were used, regardless of cuff design or duration of endotracheal intubation.[7] If one feels that lubrication facilitates intubation, why not spray the vocal cords and trachea with 4% lidocaine solution without preservative. For tube/cuff lubrication, I recommend use of a water soluble bland lubricant. Loeser reports 30% incidence of postoperative sore throat using Surgilube on hi–lo tracheal tubes and 26% without tube/cuff lubricant.[8] I personally prefer to lubricate the vocal cords and trachea with 4 ml 4% lidocaine without preservative in an effort to minimize sore throat, bucking on the tube, and intubation hypertension.[9]

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