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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Preferred Treatment of Fentanyl-induced Postoperative Rigidity

To the Editor:—Dr. Christian and co-workers appropriately identified the problem of postoperative rigidity after fentanyl anesthesia.[1] This occurrence, as well as that of biphasic respiratory depression, is an important aspect to be aware of in regard to the usage of high-dose fentanyl. All of the author's patients had significant chest wall rigidity develop 5–7 hours after the induction of anesthesia with fentanyl. The last sentence of the article indicated that the rigidity may be attenuated by naloxone or neuromuscular blocking agents. In the period following high-dose fentanyl anesthesia (usually used in patients for coronary bypass surgery or those with an unstable cardiovascular system), a sudden antagonism of the narcotic effect may lead to undesirable changes in cardiovascular hemodynamics secondary to sudden increases in catecholamine activity. Naloxone does have significant CNS impact as evidenced by elevated concentrations of dopamine in brain tissue within minutes of naloxone-precipitated withdrawal in morphine-dependent mice and rats.[2,3] Furthermore, several authors have noted adverse clinical hemodynamic effects with even low doses of naloxone given to reverse narcotic anesthesia in both humans and dogs.[4–6] These studies demonstrate a significant increase in heart rate, cardiac index, blood pressure, left ventricular stroke work, and myocardial oxygen consumption (60–70% increase from prenaloxone values). Therefore, we strongly advocate the use of neuromuscular blockers to treat this problem rather than naloxone. Specifically, we recommend metacurine because it produces minimal histamine release and clinically insignificant changes in hemodynamic parameters.

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