Communications for this section will be published as space and priorities permit. The comments should not exceed 500 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Hypoxemia during Fiberoptic Bronchoscopy

To the Editor:

The article by Shadrer and Lakshminarayan entitled “The Effects of Fiberoptic Bronchoscopy on Cardiac Rhythm” (Chest 73:821-824, 1978) rightfully focuses attention on the problem of hypoxemia during the fiberoptic bronchoscopic procedure. This excellent article stimulates further thought on the possible causes and cures for this hypoxemia.

One cause often overlooked is the removal of oxygen-enriched air from the lungs by suctioning. This air is replaced by room air enriched with oxygen in a more dilute fashion because of the higher inspiratory flow rate. The higher the rate of removal of pulmonary air by suctioning, the lower is the fractional concentration of oxygen in the oxygen-enriched air which replaces it.

To help correct hypoxemia during suctioning, the following steps would be beneficial: (1) low vacuum settings on the suctioning device should be used; (2) only intermittent suctioning for short intervals should be applied; and (3) high-flow oxygen enrichment should be employed if a nasal cannula or catheter is used (6 to 9 L/min).

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Lidocaine for Quinidine-Induced Ventricular Arrhythmias

To the Editor:

In two places in his editorial entitled “Intoxication with Quinidine” (Chest 73:129-131, 1978), Luchi stated that therapy with lidocaine is useful in controlling ventricular arrhythmias occurring in the course of intoxication with quinidine and particularly in cases of quinidine syncope. Endorsement of therapy with lidocaine under these circumstances is not justified by the rather scanty literature on this subject. Although one case report claimed that therapy with lidocaine was effective,1 in two others,2,3 it was ineffective.

I was recently involved in the care of yet another patient who had quinidine-induced recurrent ventricular tachycardia with syncope which failed to respond to therapy with lidocaine. As with other cases of quinidine syncope,4 the dosage of quinidine was relatively low and had only been given for a few days. The patient weighed 58 kg (123 lb), was normokalemic, and was not in congestive heart failure. Lidocaine was administered by bolus (80 mg) and infusion (4 mg/min), yet paroxysmal ventricular tachycardia recurred until the condition abated spontaneously as the quinidine was excreted.

As Luchi indicated, defibrillation is important for ventricular fibrillation, and both electrical and pharmacologic overdriving may be very important in preventing recurrent ventricular tachycardia and fibrillation caused by quinidine. Ultimately, though, treatment is only supportive to tide the patient over the acute crisis of a self-limited condition.4 Unless Luchi has additional evidence indicating that therapy with lidocaine is effective for intoxication with quinidine, it would seem to me that the use of another antiarrhythmic drug having an uncertain interaction with quinidine is only likely to complicate the situation and not improve it.

Anderson and Mason4 also reported a patient with quinidine syncope due to ventricular tachycardia that failed to respond to therapy with lidocaine. Measurement of the concentration of lidocaine in the serum confirmed that the drug was given in effective dosage. As in our previously reported patient,5,6 the recurrent ventricular tachycardia ultimately responded to overdrive pacing.

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References

Cell-Mediated Reactions of Lymphocytes in Pleural Fluid

To the Editor:

We read with interest the article by Pettersson and colleagues1 entitled “T and B Lymphocytes in Pleural Effusions.” Six of the patients whom they described had pulmonary malignant neoplasms; they found a normal number of T lymphocytes in the blood from these patients and a similar number of T lymphocytes in their pleural fluid. On the other hand, a decreased number of B lymphocytes was observed in the pleural fluid of these patients with malignant neoplasms, suggesting the presence of a high percentage of “null cells” (ie, lymphocytes bearing neither T nor B surface receptors).

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