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 56 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 overdrive 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 antiarhythmic 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,2,3 the recurrent ventricular tachycardia ultimately responded to overdrive pacing.

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REFERENCES
3 Koster KW, Wellens HJJ: Quinidine induced ventricular flutter and fibrillation without digitalis therapy. Am J Cardiol 38:519-523, 1976

CELL-MEDIATED REACTIONS OF LYMPHOCYTES IN PLEURAL FLUID

To the Editor:

We read with interest the article by Pettersson and colleagues5 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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It has been shown that the effector cells in antibody-dependent cell-mediated cytotoxicity belong to the population of “null cells.”

We wish to contribute additional data on the reactivity of lymphocytes in the pleural effusions of patients with pulmonary malignant neoplasms. Tests were performed in order to study T lymphocyte-associated reactions and antibody-dependent cell-mediated cytotoxicity.

**Materials and Methods**

Five patients with metastatic pulmonary malignant neoplasms and malignant pleural effusion were studied. Patients were investigated before any oncologic treatment; none had clinical tuberculosis. Histologic studies disclosed two oat cell carcinomas, one squamous cell carcinoma, one undifferentiated carcinoma, and one malignant mesothelioma.

Lympocytes from the pleural fluid and blood, which were isolated on a gradient (Ficoll-Hypaque), were cultivated according to the method of Hartzmann et al.\(^1\) Blastogenesis following activation in unidirectional mixed lymphocytic culture by normal irradiated allogeneic lymphocytes and the response to phytohemagglutinin (25 μg/100 μl) and to purified protein derivative of tuberculin (PPD; 0.01 tuberculin units/100 μl) were assessed by the uptake of tritiated thymidine. Indices of stimulation (ie, the ratio of experimental value over autologous controls) were calculated.

Antibody-dependent cell-mediated cytotoxicity was investigated by the system of assay described by MacDonald and Bonnard.\(^4\) Lytic activity mediated by “null cells” was measured by the release of radioactive \(^{51}\)chromium from rabbit antibody-coated murine lymphoma target cells.

**Results**

**Blastogenesis**

Results obtained for the lymphocytes in the pleural fluid of patients, for lymphocytes in their peripheral blood, and for the lymphocytes in the peripheral blood of control subjects are listed in Table 1. The distribution of indices of stimulation is illustrated in Figure 1. It can be seen that the lymphocytes in the pleural fluid react normally in mixed lymphocytic culture and to phytohemagglutinin; however, the lymphocytes in the peripheral blood of patients had an impaired reactivity in mixed lymphocytic culture, while they reacted normally to phytohemagglutinin. Moreover, in two patients, lymphocytes in the pleural fluid and peripheral blood reacted strongly to PPD.

**Antibody-Dependent Cell-Mediated Cytotoxicity**

Studies on the lymphocytes in the pleural fluid and the peripheral blood were made in two patients. In one patient, lymphocytes from both the pleural fluid and the peripheral blood exhibited a normal antibody-dependent cell-mediated cytotoxicity, as shown by lysis of 50 percent of the target cells with a lymphocyte-target cell ratio ranging from 3:1 to...
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30:1 (Fig 2). Lysis of 50 percent of the target cells was indeed observed with these ratios in 100 normal individuals. In the other patient the lymphocytes in the pleural fluid had depressed but existent antibody-dependent cell-mediated cytotoxicity, while the lymphocytes in his peripheral blood reacted normally in this test.

DISCUSSION

According to their reactivity in mixed lymphocytic culture, to phytohemagglutinin, and to PPD, it appeared that the lymphocytes in the pleural fluid of patients with metastatic pulmonary malignant neoplasms had a normal thymus-dependent function. Moreover, functional “null cells” were found in the pleural effusion of two patients, as assessed by antibody-dependent cell-mediated cytotoxicity. Therefore, these data strongly support the results of Pettersson et al., who reported that the pleural fluid of patients with cancer of the lung contained a “normal” number of T lymphocytes (ie, rosette-forming cells), as well as a significant number of “null cells” (ie, lymphocytes which are the effector cells in antibody-dependent cell-mediated cytotoxicity).

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REFERENCES


Positive-Pressure Breathing and Induction of Cough

To the Editor:

We would like to report findings that are in direct disagreement with those of Shim et al in their article entitled “The Effect of Inhalation Therapy on Ventilatory Function and Expectoration” (Chest 73:798-801, 1978). In a study of ten male hospitalized patients with an exacerbation of chronic obstructive bronchitis (mean forced expiratory volume in one second equalled 47 percent of predicted, and mean arterial oxygen pressure equalled 9.6 KPa), an intermittent positive-pressure nebulizer (Bird Mark 7) was compared with a compressed-gas simple nebulizer (Inspiron Mini-Neb) for their effectiveness in producing cough and sputum. The study was performed on four consecutive days, with the two forms of nebulization being alternated between morning...