Management of Pleural Effusions in Cancer of the Breast

To the Editor:

In the article entitled “Management of Pleural Effusions in Breast Cancer” (Chest 75:51-53, 1979), Lees and Hoy report their experience in treating pleural effusions associated with carcinoma of the breast. While their study is a retrospective analysis, the numbers are large, and their technique for instillation of sclerosing agents into the pleural space following drainage via a chest tube appears adequate.1 Since it is recognized that carcinoma of the breast is a malignant neoplasm producing malignant effusions in which the prognosis is not so ominous, successful management of these effusions is extremely important.

We were surprised by the results of Lees and Hoy showing no significant difference between the four groups of therapeutic manipulations, which included (1) thoracentesis alone, (2) thoracentesis plus instillation of an alkaling agent, (3) drainage via a chest tube plus instillation of an alkaling agent, and (4) drainage via a chest tube plus instillation of tetracycline. Their report rate of successful obliteration of the pleural space with tetracycline is much lower (approaching 50 percent) than has been reported previously. Both Wallach2 and Rubinson and Bolooki3 report intrapleural therapy with tetracycline to be effective in 83 to 100 percent of the patients with malignant pleural effusions; our rate of successful treatment approaches 90 percent.4 In addition, studies in animals have confirmed the effectiveness of tetracycline in producing pleural symphysis and have suggested that there is a dose-related response.5 Perhaps one explanation for the lower rate of successful tetracycline-induced pleural symphysis reported by Lees and Hoy was more extensive pleural involvement with tumor in their patients.

We have noted that patients who have a malignant effusion with a low level of glucose and a low pH have a poor rate of successful pleurodesis, when compared to those who have effusions with a normal pH and a normal level of glucose. We think that this is due to extensive pleural infiltration by the tumor, which makes the likelihood of pleurodesis low, and the development of a “trapped lung” due to visceral pleural carcinomatosis more likely. If successful reexpansion of the lung cannot be accomplished, it is impossible to obliterate the space with instillation of the sclerosing agent, and this should probably not be attempted.

We think that properly selected patients with malignant pleural effusions should have a high rate (>80 percent) of successful pleural symphysis following drainage via a chest tube and intrapleural instillation of 15 to 20 mg of tetracycline per kilogram of body weight.

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To the Editor:

The comparison of results of the management of pleural effusion in cancer of the breast is difficult unless the criteria for judging the rate of response are the same.5 Our results from Edmonton, Alberta (Chest 75:51-53, 1979), apply only to cancer of the breast, were calculated by actuarial methods, and refer to greater numbers and longer periods of follow-up than those of Wallach2 and of Rubinson and Bolooki3 and, therefore, cannot be directly compared. The report by Sahn et al5 of 90 percent obliteration is impressive, but as the report is in press at the time of this writing, there is no indication as to whether this refers only to cancer of the breast and whether this result was calculated on an actuarial basis.

An effect related to dosage of tetracycline was sought but not found in our study.

There is no way of telling whether our patients had more extensive pleural involvement than those in the other reports.5,4 The collaborating thoracic surgeons have suggested

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Coronary Arterial Disease and Arrhythmias in Chronic Obstructive Pulmonary Disease

To the Editor:

The recent article by Flick and Block1 entitled “Nocturnal vs Diurnal cardiac arrhythmias in patients with chronic obstructive pulmonary disease” (COPD) and the accompanying editorial by Senior et al4 discuss the causes of arrhythmias in obstructive disease of the airways. A point briefly mentioned, but which I believe deserves further emphasis, is the role of coronary arterial disease.

A significant number of patients with COPD have coronary arterial disease.2-3 Steele et al4 have shown that in both the stable and acutely decompensated patient with chronic pulmonary disease, the presence of coronary disease may depress left ventricular function, even without clinical evidence for its presence. Ten of 14 patients who died with acute respiratory decompensation had extensive coronary obstruction at necropsy. It is important to know the role of coronary arterial disease in arrhythmogenesis in patients with obstructive disease of the airways, as this would have important therapeutic implications. The use of coronary vasodilator drugs and myocardial scintiscans is a new method that may be applied to this area.5

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To the Editor:

We would agree with Kachel that coronary arterial disease probably is an etiologic factor of arrhythmias in some patients with chronic obstructive pulmonary disease (COPD). Certainly, the coexistence of clear-cut coronary arterial disease with COPD would seem to heighten the likelihood of arrhythmias, since poor left ventricular function is present in such patients.3 Unfortunately, there are no data relating arrhythmias in patients with COPD to a careful evaluation of their coronary arteries. In our published experience, serious arrhythmias occurred commonly in patients with advanced COPD but without clinical evidence of coronary arterial disease.2 This is noteworthy, since in the majority of the patients with poor left ventricular function and coronary arterial disease at autopsy in the study of Steele et al,3 the diagnosis of coronary arterial disease was clinically evident. It would appear that in patients with COPD, the prevalence of arrhythmias exceeds the prevalence of advanced coronary arterial disease (the latter incidence being about 25 percent),2 so that, as we suggested, factors other than coronary arterial disease probably have a more important role in most patients; however, clearly, this entire issue requires study, since, as Kachel points out, the results may have therapeutic importance.

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Sleep-Related Apnea

To the Editor:

The article by Riedy et al entitled “Sleep Apnea Syndrome: Practical Diagnostic Method” (Chest 75:81-83, 1979) focuses attention on the fact that sleep-related apnea is difficult to diagnose in community hospitals. Riedy et al reported the use of a Swan-Ganz thermocatheter thermistor positioned in the anterior nares as the indicator of airflow. We have used this type of thermistors in the anterior nares and have found it to be very unreliable in a large number of patients. We have found that when individuals breathe only through the mouth, the thermistor placed in or in front of the nares may not indicate any airflow. This, in the presence of motion of the chest wall, may be falsely interpreted as obstructive sleep-related apnea. To solve this problem, we use two thermistors in series (one in front of the nose and one in front of the mouth) as the indicators of airflow.

A more fundamental issue is what type of data should be used to decide whether tracheostomy is required. When does snoring or the apneas so common in elderly sleeping individuals indicate sleep-related apnea? It is only when the apneas are associated with disturbed physiologic findings (disrupted sleep, hypoxemia, or cardiac arrhythmia) that one