with ST-segment changes and the latter more often the group to which Spodick is referring. Nevertheless, we have witnessed significant ST-segment changes infrequently in those in whom we were unable to establish anything other than a uremic cause for the pericarditis.

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REFERENCES

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Respiratory Failure and Pulmonary Tuberculosis

To the Editor:

The conclusions concerning the frequency of respiratory failure and the types of abnormalities of pulmonary function that were reached in the article entitled "Respiratory Failure in Pulmonary Tuberculosis" by Agarwal et al are premature and are not supported by adequate data. Fifteen of the 18 patients had hepatic disease or alcoholism which might have contributed to the hypoxemia noted. Only those cases with alveolar hypoventilation manifested by hypercapnea can be attributed unquestionably to respiratory failure secondary to pulmonary disease. All of the other patients could have had all or part of their hypoxemia secondary to shunting, which has been well documented in hepatic disease. Studies of shunting using 100 percent oxygen have eliminated absolute shunting (both intrapulmonary and extrapulmonary) as a significant factor in the reported cases with respiratory failure. In the absence of such information, the conclusions about the frequency and mechanism of respiratory failure in patients with pulmonary tuberculosis are merely speculative.

Similarly poor documentation exists concerning the conclusions reached about abnormalities of pulmonary function in patients with respiratory failure and pulmonary tuberculosis. Agarwal et al stated that only five patients had tests of pulmonary function performed, yet these investigators extrapolated the results to the 11 survivors of respiratory failure with tuberculosis. If all of the surviving patients had been evaluated with complete tests of pulmonary function, the conclusions might have been different from those reported and perhaps more in line with those abnormalities previously reported in the literature.

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To the Editor:

We thank Kaplan for his interest in our article. Kaplan suggests that the cause of our patients' severe hypoxemia was the shunting which has been described in patients with cirrhosis, and he refers to his own interesting article entitled "Cirrhosis of the Liver: An Unusual Cause of Severe Hypoxemia;" however, this is not pertinent, because none of our patients clinically had cirrhosis, and cirrhosis was not found at the autopsies of those who died. Several other reports of hypoxemia and cirrhosis have emphasized that the oxygen tension of such patients is not very low:

abnormal functioning communications may exist between the portal and pulmonary venous systems through the paraesophageal mediastinal, azygos and bronchial venous systems in patients with portal hypertension. Except in rare cases, the arterial oxygen unsaturation is slight and of little clinical significance.

Alcoholism per se has not been associated with hypoxemia. It would be most unusual, if not impossible, to attribute the severe hypoxemia (arterial oxygen pressure below 50 mm Hg in all of our patients on admission) to either mild or moderate hepatic disease. Furthermore, all of the patients who survived demonstrated improvement in oxygenation, as shown in Figure 1 of our article, again excluding hepatic disease as a major cause of their hypoxemia.

We disagree that a study of shunting using 100 percent oxygen would have resolved the mechanism of hypoxemia, because it has been demonstrated that in cirrhotic patients the hypoxemia is primarily on the basis of an imbalance of diffusion and perfusion.

There was no evidence of obstruction of the airways in the five patients in whom we performed tests of pulmo-
Dilatation of the Aortic Root in Marfan's Syndrome

To the Editor:

In the report of Come et al1 entitled "Echocardiographic Recognition of Silent Aortic Root Dilatation in Marfan's Syndrome," a vivid description of progressive aortic dilatation over an 18-month period was given. Two months before the patient's death, the diameter of his aortic root measured 7.5 cm. Using the normal laboratory value of Come et al1 for measurement of the aortic root as 2.1 to 4.4 cm, we have a 42 to 72 percent increase in the size of the ascending aorta over that period of time. With the knowledge that this patient had Marfan's syndrome and that his aorta was continuing to dilate, despite therapy with propranolol hydrochloride (Inderal) of up to 240 mg per day, I would be very interested in the recommendations of Come et al1 as to how often a patient with Marfan's syndrome and progressive dilatation of his aorta should be examined with echocardiograms and at what point a cardiovascular surgical consultation would be indicated to consider prophylactic replacement of the ascending aortic arch. Although aortic dissection in patients with Marfan's syndrome cannot be predicted, it would seem that the cardiovascular surgeons, along with the echocardiographers, could delineate for us through their collective experience a diameter above which prophylactic replacement would be indicated. This approach has been used in abdominal aneurysms and may be a worthwhile study in patients with Marfan's syndrome and its variants.

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REFERENCE


To the Editor:

At the present time, data are not available to answer the several important questions raised by Liss. How often a patient with Marfan's syndrome should be examined with echocardiograms is not known, as the natural history and progression of aortic dilatation in these patients have not yet been defined. Until these factors are known, it will be difficult to say at what diameter one should intervene prophylactically to replace or buttress the dilating aorta. Only with large-scale longitudinal studies of these patients will these answers be forthcoming; one such study is in progress at our institution. Although the studies will take time, with techniques of one-dimensional and two-dimensional echocardiography, the answers are at least attainable.

Patricia C. Come, M.D.; Bernadine H. Bulkley, M.D.; Victor A. McKusick, M.D.; and Nicholas J. Fortuin, M.D. Johns Hopkins Hospital, Baltimore

Mycotic Aneurysm of the Right Subclavian Artery
A Complication of Heroin Addiction

To the Editor:

We have studied a young patient with a mycotic aneurysm of the right subclavian artery that followed repeated injections of heroin into the neck with development of an abscess.

CASE REPORT

A 24-year-old male heroin addict was admitted because of a painful swelling on the right side of his neck, fever, and respiratory distress. A chest x-ray film disclosed a large, well-circumscribed homogeneous mass in the anterior superior mediastinum.

An emergency transfemoral catheterization of the thoracic aorta was performed, which revealed a 2 × 1.5-cm aneurysm arising near the origin of the right subclavian artery. At