a report by Hakim and Milstein is mentioned as the only negative article on bronchial stapling.1 We have also had negative experiences with the hinged stapling devices; these were also published in 1985. In our small series, we had three cases of bronchopleural fistulas in eight consecutive pneumonectomies, but none after lobectomies.2 Before and after this period we used parallel firing staplers (TA and TA II 30, 4.8 mm staples). With these instruments, we performed more than 400 bronchial closures (more than 100 of them on main stem bronchi) without a single bronchopleural fistula.

We therefore strongly support the recommendation of the authors that only parallel firing screw-down types of stapling devices should be used at the present time.

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Sarcoidosis and the Diaphragm

To the Editor:

In sarcoidosis, involvement of the diaphragm has rarely been mentioned. We report a case of systemic sarcoidosis in which noncaseating granulomas were found in the diaphragm.

CASE REPORT

A 66-year-old man died of gastrointestinal bleeding. Autopsy showed a duodenal ulcer with a small blood vessel projecting from it. Microscopic examination revealed multiple noncaseating granulomata in lungs, lymph nodes, gastroesophageal junction, duodenum (from ulcer area), liver, spleen, bone marrow and diaphragm (Fig 1). Acid-fast bacilli and fungi stain results were negative.

DISCUSSION

In view of affinity of sarcoidosis for skeletal muscles, it would not be surprising to find non-caseating granulomas in other muscles, including intercostal muscles and the diaphragm.1 Still, diaphragmatic involvement in sarcoidosis has not been reported previously. Diaphragmatic biopsies are not routinely obtained, nor is the examination of the diaphragm always included in autopsy studies. Skavlem et al3 briefly mentioned the diaphragmatic involvement in a patient on whom an autopsy examination showed asbestosis and sarcoidosis.

We reviewed 19 diaphragmatic sections from 19 autopsy cases of multisystem sarcoidosis; only one had noncaseating granulomas in the diaphragm. This cannot be considered as a true prevalence, particularly when one considers the 50 to 80 percent rate of granulomatous involvement of skeletal muscles. In diagnosis of asymptomatic sarcoidosis of muscle, many serial sections of a generous specimen are advisable because granulomas may be sparse and difficult to find.

Discrepancies between subjective, radiologic and physiologic assessment in patients with sarcoidosis is frequently noted. It is not known at present if diaphragm involvement in sarcoidosis is a possible contributory factor in dyspnea in sarcoid patients. A prospective study is required to find out the true prevalence in diaphragm involvement and its possible clinical significance.

The observation is, however, of some significance because diaphragmatic granulomas were observed in the patient who showed no dyspnea, tachypnea, or abnormality of diaphragmatic function (eg, flutter or paradoxic movement.)

In cases where discrepancies between subjective, radiologic and physiologic assessment are noted, one should include the diaphragmatic involvement in the evaluation.

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Ring Calcification of Coronary Artery Aneurysms

To the Editor:

We read with interest the article by Doi and colleagues (Chest 1987; 92:1118-20) describing an adolescent with a ring of calcification in a coronary artery aneurysm.1

We have been caring for a 10-year-old boy who was diagnosed in infancy as having infantile polyarteritis nodosa, which may be identical to Kawasaki disease.2 Cardiac catheterization at 17 months of age demonstrated an aneurysm in the proximal anterior descending coronary artery. At six years of age, a 6 mm ring of calcification was noted on chest roentgenogram (Fig 1). Coronary angiography at 10 years of age confirmed that this calcification is in the wall of an aneurysm of the anterior descending coronary artery. There was mild diffuse disease of the distal branches of the left coronary artery. The proximal right coronary artery was completely obstructed, but the distal right coronary artery was opacified via collateral vessels.

Both Dr. Doi’s patient and our patient had unusually severe coronary arteritis, as demonstrated by late obstruction of a coronary artery contralateral to the aneurysm. We agree that the plain chest roentgenogram should not be overlooked in following patients who had Kawasaki disease or infantile polyarteritis nodosa. Although it is difficult to draw conclusions from only two cases, a calcified coronary artery aneurysm may suggest severe coronary artery disease and warrant coronary angiography.