replacement. After the first day of hospitalization, the patient deteriorated very rapidly, with progressive hepatomegaly, jaundice, and infebrile diaphragmatic edema. There was also complicating thrombocytopenia with bleeding. The patient died on the seventh day of hospitalization.

On postmortem examination, there was hepatocellular carcinoma with multiple intra-abdominal metastases and thrombosis of the inferior vena cava. The heart weighed 275 gm and was normal, with normal thickness of the septum and left ventricular free wall. Microscopic study showed normal orientation of the myofibers and no evidence of hypertrophy.

**DISCUSSION**

A systolic anterior motion of the mitral valve has been known to occur in hypertrophic cardiomyopathy, and it is evident for outflow obstruction; its mechanism is still not well understood. We have also observed that ectopic chordae tendineae can also produce an abnormal systolic anterior motion of the mitral valve with functional outflow obstruction in the absence of hypertrophic cardiomyopathy (unpublished data). Bulkley and Fortuin reported a case of typical echocardiographic mitral systolic anterior motion in a patient with a normal heart. They postulated that hypovolemia and catecholamine stimulation were responsible for such an abnormality.

Several investigators have shown that hemorrhagic shock and infusion of catecholamines may produce functional intraventricular obstruction. In our case the combination of hypovolemia and anemia seem to have caused the echocardiographic abnormality. Although in our case, as in the one reported by Bulkley and Fortuin, there were no pressure recordings made, the association of a loud systolic murmur and the systolic anterior motion in the echocardiogram suggests an outflow gradient. The absence of left ventricular hypertrophy, the finding of a perfectly normal mitral leaflet with no thickening, and the history of a recent murmur all suggest that the functional obstruction was indeed related to the circumstances of terminal illness, namely, hypovolemia and anemia. I would like to subscribe to the speculation of Bulkley and Fortuin that a small, underfilled left ventricle with increased contractility due to infusion of catecholamines or, as in our case, to anemia may produce a systolic murmur and abnormal anterior systolic motion of the mitral valve. The physiologic change in the left ventricle simulates hypertrophic cardiomyopathy with obstruction and suggests that the approximation of the mitral valve to the interventricular septum, with increased contractility and high velocity of the ejection flow, are responsible for the functional obstruction, as has been suggested previously.

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**Coronary Arterial Aneurysm vs Poststenotic Dilatation**

To the Editor:

I enjoyed the article entitled "Coronary Artery Aneurysm: A Review of the Literature with a Report of 11 New Cases" by Falsetti and Carroll in the May issue (Chest 69:630-636, 1976). Nevertheless, I was a bit disturbed by the classification of some of the abnormalities illustrated as aneurysms. In spite of the definition of aneurysms as


To the Editor:

Dr. Rainer points out the difficulty in distinguishing poststenotic dilatation from coronary arterial aneurysm. When we first became interested in this problem, we reviewed the literature and talked to other angiographers about the criteria to be used. It became clear that coronary arterial aneurysms are easy to identify if there is no associated coronary atherosclerosis. In the face of coronary atherosclerosis, if the aneurysm is poststenotic for a considerable length and distance, there is no question. If the aneurysm occurs after an atherosclerotic obstruction, the criteria depend on the diameter of the vessel and the length of the dilatation.

From our reading of the literature, as well as discussions with other cardiologists, we chose the following criterion to separate coronary arterial aneurysm from poststenotic dilatation: an increase in the vessel’s diameter two times its usual diameter over a length of at least 2 cm. In a prospective study of 742 patients, this criterion seemed to easily separate poststenotic dilatation from coronary arterial aneurysm. In fact, coronary arterial aneurysms usually have dilatation of the coronary artery for the majority of the vessel’s length. Until other criteria become available, we believe this is still the best one.

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Open Pulmonary Biopsy

To the Editor:

The 11 authors of “Open Pulmonary Biopsy: Nineteen-Year Experience with 416 Consecutive Operations,” which appeared in the January 1976 issue (Chest 69:43-47, 1976), said that their indication for biopsy was the presence of a clinical and radiologic diagnosis of diffuse pulmonary disease undiagnosed by indirect methods. In the one tabulation contained in the article, the 39 patients with bronchogenic carcinoma are difficult for me to fit into their protocol. Similarly, the inclusion of eight cases of pulmonary infarction diagnosed by lung biopsy makes one think that the adjective, “diffuse,” was employed rather broadly. Since it seems that there was not strict adherence to the protocol, the 11 cases of histoplasmosis arouse curiosity. One wonders if included in the 11 were examples of (1) acute infiltration of one or two pulmonary segments, (2) acute diffuse reticulonodular disease, (3) acute multifocal nodular densities, and (4) single or multiple calcified lesions, all of which are usually self-limited processes requiring no specific treatment.

As in histoplasmosis, rarely is it necessary to resort to lung biopsy to establish a diagnosis of farmer’s lung. In the report by Ray et al, 64 instances of farmer’s lung are listed as having been diagnosed by lung biopsy.

It is also a little unsatisfying, considering the fact that Ray et al tout the specificity of biopsy information, that in the tabulation, two of the neoplastic diagnoses were listed as “other,” while nine of the occupation-related diagnoses, four of the diagnoses of specific infections, and 38 of the specific histologic diagnoses were described as “miscellaneous.” Furthermore, if you count diagnoses of “chemical inhalation,” “acute bronchiolitis,” and “arteriolar occlusion” as specific, the reader can still discern only 241 specific diagnoses (58 percent) among the 416 cases mentioned in the tabulation.

Even when some details of individual cases are given, one is kept in suspense about the diagnosis established. I would wager that even those “few internists” who “still pursue the elusive diagnosis of diffuse pulmonary disease down the winding avenues of lupus erythematosus preparations, endless sputum cultures, and repeat chest x-ray films” would want to read the diagnosis in one of the patients who died within 24 hours after biopsy. He was the 80-year-old man who had malignant hypertension, a recent myocardial infarction, and renal failure, as well as diffuse pulmonary disease.

In the 19 patients (4.5 percent) who died after biopsy, only four had their therapeutic management altered because of the histologic diagnosis provided by the biopsy. Ray et al state that it was of value to the family to know, rather than not know, a specific histologic diagnosis. Because of probable misapplication of their protocol and the incomplete presentation of data, I cannot gauge whether the quality of information gained by biopsy verifies such rhetoric as that and the following: “open-lung biopsy is a true bargain . . .” and “. . . it allows and promotes early and definitive diagnosis and prompt therapy.” Nevertheless, Ray et al should be given credit for a responsive interdisciplinary approach to a difficult problem and for the wisdom of the final sentence in their article (the Latin word, “ultimare,” means “to come to an end, be last”).

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