results entirely on the assumption that a "v" wave in the left atrial pressure trace is suggestive of mitral regurgitation. We do not disagree with this assumption, but we do not agree that the so-called "v" waves in their Figures 2 and 3 are caused by mitral regurgitation. They are, in our interpretation, actually large "a" waves due to atrial contractions against a closed mitral valve. In oscillographic records of Figures 2 and 3, the "P" wave of the ECG follows the "R" wave, and hence, atrial contractions during ventricular systole. In their Figure 4, the "P" wave precedes the "R" wave, and the atrium does not contract during ventricular systole; so there is no apparent "v" wave. Thus, without data which indicate the existence of mitral regurgitation or position of the mitral valve, the measurement of papillary muscle length cannot be used to infer its role in valve closure, since there is no way of determining whether length changes are passive or active.

An oscillographic record (Fig 1) from our laboratory clearly illustrates the correct interpretation of the data. In panel A, the three "a" waves occur when the mitral valve is open, while in panel B, the second "a" wave occurs during ventricular systole and could be mistaken for a "v" wave but for three facts: 1) it occurs at the time we would expect the atrium to contract; 2) it is not sustained as is the "v" wave of mitral regurgitation; and 3) it is clear from the record of electromagnetically measured mitral flow (encircled) that there is no mitral regurgitation. Other oscillographic records which support this interpretation can be found in previous publications from our laboratory.2,3 We do not wish to suggest that the authors' conclusions are in error; but we do wish to point out that their data do not substantiate the conclusion.

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References


To the Editor:

Drs. Yellin and Frater do not disagree that a "v" wave in the left atrial pressure is suggestive of mitral regurgitation. They disagree with our interpretation, however, that the recorded atrial pressure in Figures 2 and 3 shows "v" waves during premature ventricular contractions. Rather, they interpret the atrial pressure to show large "a" waves.

The criteria that Drs. Yellin and Frater use for an "a" wave are: 1) it occurs at the expected time, and 2) it is not sustained. These criteria fit the description of their "a" waves in the illustration that they have submitted. The criteria, however, do not apply to the atrial pressure waves in Figures 2 and 3 in our manuscript. Criterion 2 clearly is not applicable. The apparent "v" waves in Figures 2 and 3 are sustained and markedly longer in duration than the "a" waves that precede and follow the "v" waves.

Criterion 1 is more difficult to assess; but careful measurement shows that it does not apply to our recorded pressure. The apparent "v" waves in Figures 2 and 3 start slightly sooner than the "a" waves would be expected. This result from the QRS occurring slightly before the P wave. The difference of timing is more apparent in Figure 3 because it was recorded at a faster paper speed.

Additional criteria for distinguishing "v" waves from "a" waves that were not mentioned by Drs. Yellin and Frater may be applied. Following closure of the mitral valve, it billows backward producing the "o" wave.1 In both panel A and panel B of Drs. Yellin and Frater's illustration, a "o" wave is coincident with the apex of backward
flow that they recorded with an electromagnetic flow-transducer. During sinus rhythm, the "c" wave closely followed the "a" wave. With premature ventricular contractions, if the mitral valve closes, a "c" wave precedes the "a" wave. Both "a" waves recorded by Drs. Yellin and Frater in panels A and B show "c" waves preceding the "a" wave in premature ventricular contractions. During sinus rhythm, in our Figures 2 and 3, "c" waves were clearly recorded. No "c" wave preceded the "v" waves, however, in Figures 2 and 3. The reason is that the mitral valve was open.

In a previous study, we showed that the mitral valve is open when the papillary muscle achieves its shortest length. There was no implication regarding whether the changes in length of the papillary muscle were active or passive. The question of active or passive changes of length raised by Drs. Yellin and Frater, therefore, is irrelevant.

Even though the P wave may follow the QRS complex during premature ventricular contractions, it does not imply that the atrium contracts against a closed mitral valve. If the papillary muscle can prevent closure of the mitral valve, then the atrium may contract against an open mitral valve and mitral regurgitation will occur. The sequence of a P wave during or following a QRS does not require that the recorded wave in the left atrium be an "a" wave.

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REFERENCES

Benefits of Open Lung Biopsy in Patients with Previous Nondiagnostic Transbronchial Lung Biopsy

To the Editor:

The recent article by Toledo-Pereyra et al (Chest 1980; 77:647-50) not only is misleading, but their conclusions are based upon inappropriate data. In their abstract, they conclude that "in immunosuppressed patients who previously had a nondiagnostic fiberoptic (italics mine) transbronchial biopsy of the lung," an open lung biopsy should be performed due to the high percentage of false-negative results with the transbronchial procedure. However, their technique of transbronchial biopsy, as they described in their methods section, does not utilize the fiberoptic bronchoscope. Rather, a fiberoptically guided flexible catheter under fiberoptic control is used to reach the lesion. This presumed oversight in their communication misleads the reader, as well as diminishes the validity of their conclusion, since a comparison of this technique with the routine fiberoptic transbronchial biopsy was not performed.

Another criticism of their study is the quality of the transbronchial biopsy specimens which they analyzed. In 5 of 13 nondiagnostic biopsies, either bronchial wall or peribronchial tissue was obtained. This suggests that one major reason for their high percentage of false-negative biopsies is a technical problem in obtaining an adequate alveolar specimen. As well, in another 4 of the 13 patients with nondiagnostic biopsies, they report that “no diagnostic abnormality” was obtained. I am unclear how to interpret this description—if it means that normal lung parenchyma was biopsied, then it is obvious that the biopsy could not have been taken from the diseased area of the lung. Therefore, if I were to reinterpret their data, I would say that in 9 out of 13 nondiagnostic biopsies, inappropriate tissue was obtained to diagnose a pathologic parenchymal process.

Finally, the authors do not mention the number of biopsies that were taken in each patient. We have recently reported that in a diffuse parenchymal disease such as sarcoidosis, the diagnostic yield of the transbronchial biopsy technique increases logarithmically with the number of biopsies. Therefore, extrapolating this concept to other diffuse diseases, if Toledo-Pereyra et al only took one biopsy per patient, it is expected that a low diagnostic yield would result.

We feel that the problem of a nondiagnostic transbronchial biopsy in the immunosuppressed patient brings up a critical patient management problem— to proceed to open lung biopsy or not. We agree with Toledo-Pereyra et al that with the results of transbronchial biopsy using their technique, an open lung biopsy should be performed. However, we would reserve generalized recommendations about this problem until more precise studies are carried out.

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2 Gilman M, Wang KP. Transbronchial biopsy in sarcoidosis (abstract). III World Congress on Bronchoesophagology, April, 1980

To the Editor:

I am happy to answer comments of Drs. Gilman and Wang in hope that further explanation may either clarify a misimpression or, where we differ, provide support for our position.

There was no oversight in our communication regarding the use of a fiberoptically-guided flexible catheter rather than the fiberoptic bronchoscope as the means of obtaining the biopsy. Since most of these lesions represent an alveolar process and not an endobronchial process, it is difficult to locate the area of maximum radiographic pathology to be biopsied without fiberoptic. Our pulmonary radiologists perform the transbronchial lung biopsy by positioning the biopsy catheter under fiberoptic control into the region of maximal abnormality. They believe that simple observation of the endobronchial passages with a fiberoptic bronchoscope without fiberoptic does not lead with any as-