
To the Editor:

It’s amazing to me how in this age of computers that an article as specific as yours was somehow missed in our personal review and in our automated computer search. However, I have had the occasion recently to review it and agree with you that your report on the effects of angiotensin on left ventricular ejection fraction in both man and dog was well described. Although your methods of determining left ventricular ejection fraction and your reduced levels of infused angiotensin with their resultant lower increments of pressure elevation differed from our current study, I would agree that your conclusions were appropriate. In both studies, angiotensin infusion had an effect on depressing left ventricular contractility in both the normal and diseased heart. The discrepancy in the depressed left ventricular ejection fraction found in both normal and diseased hearts of man may be explained, as you suggest, by methods of ejection fraction measurement and by levels of angiotensin infused.

We both conclude that angiotensin infusion at either level used appears to depress cardiac function in man, whether normal or diseased. I appreciate your having brought this to our attention.

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Diffusing Capacity in Alcoholics

To the Editor:

The editorial by Dr. Banner in the April, 1980 issue of Chest (77:460-461) addressing the interaction of “Alcohol and the Lung,” and the accompanying article by Peavy et al in the same issue (77:488-492), both make reference to reports of reduction in the single breath diffusing capacity for carbon monoxide (DLco) in alcoholics. They correctly note that this has not been accounted for by cigarette consumption alone and that it seems to be reversible since the diffusing capacity is normal in former alcoholics. We wish to point out though, that when the DLco is also corrected for anemia in these individuals, the DLco is not significantly reduced. This information comes from a study in which the DLco was measured in 20 consecutive patients with a physician diagnosis of excessive alcohol consumption. The mean observed DLco was significantly reduced to 70 percent of predicted (± SD 15.8) and was less than 70 percent of predicted in 12 of the 20 patients. However, when the DLco in anemic individuals was corrected to a hemoglobin concentration of 14.7 g/dl the mean DLco was then 82.3 percent of predicted (± SD 12.9) and was abnormal in only 4 of the 20 patients. In anemic subjects, the mean DLco adjusted for hemoglobin was in fact no different from the DLco in subjects with a normal hemoglobin. Furthermore, when the DLco prediction is corrected for the smoking history there is no difference (P > 0.2) between predicted and observed DLco adjusted for hemoglobin.

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REFERENCES
3 VanCanse WF, Ferris BG, Coles JE. Cigarette smoking and pulmonary diffusing capacity (transfer factor). Am Rev Respir Dis 1972; 106:30-41

To the Editor:

The data presented by Fisher is at variance with my own work and that of Emirgil et al which found that diffusing capacity is impaired in alcoholic patients. Fisher attributes this difference to our failure to correct for anemia. I believe the different results can be attributed to other factors. I did not correct for anemia in my patients since none was significantly anemic (all had a hematocrit > 40 percent). The hematocrits of the patients in the study by Emirgil et al ranged from 36 to 47 percent. I believe Dr. Fisher’s conclusion can be attributed to the use of a regression equation with large scatter and to the matter of the definition of alcoholism. The mean diffusing capacity of our patients was less than his, 62 percent vs 70 percent of predicted. All of our patients were drawn from a group of individuals admitted for alcoholic detoxification, confirming the severity of their addiction. The patients of Fisher were judged alcoholic if their physicians felt they consumed excessive quantities of alcohol. This is a subjective judgment and may relate more to the individual physician’s concept of alcoholism rather than to the amount of alcohol actually consumed.

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REFERENCES

Diagnosis of Sarcoidosis by Transbronchial Lung Biopsy

To the Editor:

The article by Roethe et al (Chest 1980; 77:400-09) deserves some comment. While the authors have attempted to determine the optimal number and sites of transbronchial lung biopsies necessary to diagnose sarcoidosis, their data fall short of providing conclusive answers.

CHEST, 79: 1, JANUARY, 1981

COMMUNICATIONS TO THE EDITOR
In the evaluation of transbronchial biopsy for the diagnosis of diffuse parenchymal lung disease, the presence of adequate alveolar tissue in each specimen must be assessed, as individual specimens consisting mostly of bronchial wall may be insufficient for diagnostic purposes. Providing these data would have permitted clarification of the authors' "negative" biopsies.

It would also be interesting to note which of the five biopsies (ie-1st, 2nd, 3rd, etc) from each lobe were positive, thus permitting a more precise statement regarding the number of biopsies actually necessary.

The site of each biopsy should also have been more specifically noted. Information as to whether the same or different subsegments were biopsied might have yielded data about the distribution of disease within each lobe and whether it is useful to vary the position of the bronchoscope before each biopsy.

The authors' conclusion that five to ten biopsies are needed to provide adequate tissue for diagnosis is not supported by our data. We have found that three to four biopsies, when serially sectioned and examined for granulomatous, will be diagnostic of sarcoidosis in almost all cases. Ninety-seven percent of our last 89 patients with proven sarcoidosis were diagnosed using the above technique.

While we are pleased to note the absence of serious complications in Roethe's series, we do not believe that adherence to their criteria for number of biopsies should be generally accepted at this time.

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To the Editor:

Roethe and coworkers provide valuable clinical informa-

tion in their article suggesting the optimal number of trans-
bronchoscopic biopsies for diagnosing sarcoidosis. If their hypothesis that the ability to make a diagnosis is related to sampling error is true, application of the geometric distribu-
tion1 allows calculation of the exact probability of making the diagnosis for a given number of biopsies.

The table shows the results of these calculations for three situations. "A" is based on their overall data, in which 153 of 370 samples (41.4 percent) were diagnostic; "B" is based on the results in stage I patients, from whom 21 of 100 specimens (21.0 percent) were positive. "C" is based on stage II and III patients, considering biopsies from the predominantly involved lobe or either lobe in patients without predomi-
nance; 114 of 200 biopsies (57.0 percent) were positive. For each of these situations, the first column shows the probability (in percentages) that a particular biopsy will be the first positive one, and the second column shows the cumulative probability of making a diagnosis if that number of biopsies is taken. For example, for a stage I patient, the probability of making a diagnosis if five biopsies are taken is 69.2 percent, there is an 8.2 percent probability that the fifth in a series of biopsies will be the first one to allow the diagnosis. In a stage I patient, taking a seventh biopsy will increase the chance of making a diagnosis by 5.1 percent, while a seventh biopsy from an involved lobe in a stage II patient will only provide a 0.4 percent increase in the probability of making a diagnosis.

Looking at the results in this manner allows one to think quantitatively about the risk-benefit relationships in performing transbronchoscopic biopsies.

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REFERENCE


<table>
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<th>Situation</th>
<th>A. Overall</th>
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*Biopsies from predominantly involved lobe or any lobe if none is predominant;
**The "marginal" probability is the probability that this will be the first positive specimen, and the "total" probability is the probability of having at least one positive biopsy out if this number of biopsies are taken.