have been estimated to occur in 11 to 65 percent of the patients with acute Q fever. When liver-function tests are considered, 70 to 85 percent of patients have abnormal values. We reviewed 38 patients with acute Q fever cared for at our hospital in the last seven years. We found that 18 patients (47.3 percent) presented with pneumonia, 16 (42.1 percent) presented with hepatitis without evidence of pulmonary involvement, and four (10.6 percent) presented with a febrile, self-limited illness without evidence of pulmonary or liver involvement. Furthermore, among patients with pneumonia seven (38.8 percent) had liver and/or spleen enlargement, seven (38.8 percent) had elevated levels of AST, six (33.3 percent) elevated levels of ALT, five (27.7 percent) elevated levels of alkaline phosphatase, nine (50 percent) elevated levels of GCPT, and six (33.3 percent) elevated levels of LDH. However, the level of hepatic transaminases was significantly higher in those patients without pneumonia when compared with those with pneumonia, (p = .0001) as was the level of direct bilirubin (p = .004). On the other hand, mean interval between the beginning of the symptoms and hospitalization was significantly higher in patients without pneumonia (4.5 ± 2.3 vs 11.2 ± 8.8 days, p = .004).

Lack of data referring to liver dysfunction in the article by Marrie et al is surprising, especially if we consider that 14 patients were at some time admitted to a hospital. Although Marrie's article focuses mainly on epidemiologic aspects of Q fever, it could be interesting to know how many of their patients presented clinical and/or laboratory evidence of liver involvement and also if there were patients with only hepatitis. This could be important to further define the spectrum of organ involvement in acute Q fever and may document differences between epidemic and sporadic cases of acute Q fever.

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REFERENCES

To the Editor:

Our study (Chest 1988;93:98-103) was an epidemiologic one. Patients were admitted to a community hospital and did not have liver function tests performed.

However, since we have begun the study of Q fever in 1979, we have identified 170 patients with Q fever in Nova Scotia. None of these patients have presented with hepatitis. We have also studied 20 patients with severe pneumonia due to Q fever who were hospitalized at the Victoria General Hospital in Halifax. All of these patients had serial liver function tests carried out. Alkaline phosphatase was above the upper limit of normal in 57 percent, AST was elevated in 52.6 percent and ALT in 42.8 percent. These figures are similar to the data quoted by Domingo et al. We have also studied 40 patients with Mycoplasma pneumoniae and 14 with Legionella pneumophila pneumonia. We found that 47.5 percent of the Mycoplasma patients and 64 percent of the Legionella patients had an elevated alkaline phosphatase level. AST was elevated in 37.5 and 57 percent of these two groups respectively, and ALT in 27.5 and 30.7 percent.

In most instances, patients with Q fever pneumonia had very mild elevation of the various liver function tests. Mean AST was 54 (upper limit of normal is 29) and ALT 48.1 (upper limit of normal 41).

None of the 21 patients had hepatosplenomegaly. Indeed, only two of 50 patients with Q fever pneumonia that I personally examined had splenomegaly.

These data would suggest that indeed there may be something different about Q fever in Nova Scotia compared with Q fever in Spain—this could reflect strain or host differences. It is important to realize, however, that abnormal liver function tests do not necessarily mean involvement of the liver by Q fever since mild abnormalities of liver function tests are common in various atypical pneumonias.

Thomas J. Marrie, M.D., Dalhousie University, Victoria General Hospital, Halifax, Nova Scotia

Diagnostic Value of Bronchography

To the Editor:

I read with great interest Dr. Cervantes-Perez's recent letter illustrating the use of bronchography in the diagnosis of lobar torsion.1 It is both nostalgic and refreshing for those of us who performed countless bronchographies in the past to see that it still has a place in diagnostic bronchography. I hope that the young generation of radiologists will continue to take advantage of this excellent diagnostic modality in carefully selected cases.

It must be noted, however, that Dr. Cervantes-Perez's claim to be the first to diagnose lobar torsion by bronchography is upset by a few years by Huang and Cho.2 Also, a similar case was illustrated by Felson3 not too long ago.

Yahya M. Berkmen, M.D. Department of Radiology, New York Hospital, Cornell Medical Center, New York

REFERENCES

To the Editor:

I wish to answer some of Dr. Berkmen's questions.

My basic objective in informing your readers about our case of lobar torsion was to demonstrate the usefulness of bronchography as a diagnostic procedure prior to surgery in some cases of pulmonary torsion, and to relate this to the report of Shorr and Rodriguez (Chest 1987; 91:297-30).

From this point of view, the matter of whether our case was or wasn't the first to be preoperatively diagnosed by means of bronchography is irrelevant. However, the additional information Dr. Berkmen provided confirms our impression that our case was the
first to be recorded because we presented the case before the Sociedad Mexicana de Neurología-Cirugía de Torax in 1976, three years before Huang and Cho published their case. At that time we sent a report to *Chest*; however, the report was not published.

The article by Felson does not contain an additional similar case; the related reference concerns the case published by Huang and Cho.

I remain in complete agreement with Dr. Berkmen that bronchographic study continues to be an excellent diagnostic resource in selected cases of bronchopulmonary pathology.

**Porfirio Cervantes-Peres, M.D., F.C.C.P.,
Hospital Central Militar,
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Thus, while we agree that different reference equations may yield slightly different results, we continue to believe the final recommendation of our study: "... all patients with chronic obstructive lung disease who have exercise intolerance and a resting arterial oxygen tension above 55 mm Hg should undergo exercise testing if their diffusing capacity is 55 percent of predicted or less." We suspect that Dr. Ries and colleagues would agree.

**Gregory R. Owens, M.D., F.C.C.P.;
Frank C. Scurba, M.D.; and
Robert M. Rogers, M.D., F.C.C.P.,
University of Pittsburgh,
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**References**


**To the Editor:**

We read with interest the recently published article, "Pulmonary Function Tests Cannot Predict Exercise-induced Hypoxemia in Chronic Obstructive Pulmonary Disease." This is the second article published in the past year which has questioned the findings of our 1984 study of arterial desaturation in patients with COPD. Careful scrutiny of the three articles, however, reveals no significant contradictions in the data presented and analyzed. Sue et al. noted that a decrease in the single breath carbon monoxide diffusing capacity (DLco) was associated with abnormalities of gas exchange during exercise. However, they also found gas exchange abnormalities in 32 individuals with relatively normal values for DLco and suggested that arterial blood gas studies were necessary during exercise testing. Abnormalities noted in this study, however, included not only significant changes in arterial oxygen tension or saturation, but rather any change in these parameters or an abnormality in VD/VT or a-ET CO2. We agreed with Sue et al. that subtle abnormalities of gas exchange may be found during exercise testing, but pointed out that clinically significant changes (or lack of change) in oxygen saturation would still be predicted by DLco.

The title of the current study implies that pulmonary function tests are not helpful clinically in determining who will and will not experience desaturation during exercise. However, analysis of the data presented reveals that the findings of this study are virtually identical to ours. These data confirm that DLco is helpful in predicting arterial desaturation since no patient in their study desaturated who had DLco values greater than 20 ml/min/mm Hg. We did not state in our article that there was a linear relationship between pulmonary function test results and exercise desaturation, nor that all patients with abnormal values of DLco (<55 percent of predicted in our paper) desaturated, but rather that as a screening test DLco was helpful in deciding who should undergo exercise testing to evaluate arterial desaturation.

One of the caveats raised by Ries and colleagues was the wide range of reference values for DLco, implying that this fact may account for the apparently different findings in their study. However, two other studies evaluating exercise responses in patients with interstitial lung disease and one study of patients with cystic fibrosis found results virtually identical to ours even though different testing equipment and different reference values for DLco were utilized. This suggests to us that the use of reference values, validated for a specific laboratory, are indeed useful. Use of an absolute value, as suggested by Ries and colleagues, completely ignores the fact that these reference values were designed to normalize a population for age, sex, height, and sometimes weight. Utilizing their approach, a small, elderly woman and a tall young man would have the same cut-off values. This approach is clearly nonsensical.

**To the Editor:**

We appreciate the comments of Owens and colleagues regarding the comparison of our recently published study with theirs. We agree that the data in both studies are similar and stated in our article that "careful examination of their data reveals little discrepancy." We believe that the apparent differences between the studies are more in emphasis than in substance. Both studies demonstrated that pulmonary function tests are most useful in excluding COPD patients with less severe disease who are unlikely to develop arterial oxygen desaturation with exercise and who, therefore, do not need to undergo exercise testing for this purpose.

Although the conclusion that the threshold criterion for DLco is 55 percent of predicted for laboratories which validate their normal values is an interesting hypothesis, we disagree that it can currently be accepted as fact. As indicated in our article, we have previously confirmed that the normal reference values of Miller and coworkers for DLco are most appropriate for our laboratory. Use of these reference values gave a threshold criterion of 70 percent of predicted for the patients we tested.

We also do not agree that different reference equations for DLco yield results that are only slightly different. As indicated in Table 2 in our publication, mean DLco for our patients expressed as a percentage of predicted ranged from 53 to 79 when different predictive equations were utilized. We think these differences could have substantial clinical impact if diagnostic criteria for DLco from one laboratory and reference equation are used inappropiately in another laboratory.

Interlaboratory comparisons have indicated excessive differences in DLco measurements, whether expressed as absolute values or as percent of predicted. This suggests that neither standardization of