findings of Sonnenblick et al of the greatest positional changes in PaO2 that were found with two patients with cardiac failure were because of occult contralateral effusions, even though the diagnosed effusions were reported to be small. Indeed, cardiac failure is most often associated with bilateral effusions if more sensitive diagnostic techniques are performed.6-8 The two reported patients with ascites may also have had an occult contralateral effusion. Nearly 30 percent of patients have been found to have bilateral effusions with ascites when demonstrated with radioactive labeled albumin techniques.4 Even though it has recently been reported that sensitive findings on routine roentgenogram such as the thorn sign9 and displacement of a sharp costophrenic angle9 suggest effusions, as much as several hundred milliliters of loculated pleural fluid may remain undetected without the use of other diagnostic testing.7 In conclusion, although the significance of the study by Sonnenblick and associates is noteworthy, it is possible that over half the patients had bilateral effusions, and not unilateral as reported.

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To the Editor:

The purpose of our study was to answer the question: in which lateral decubitus position is gas exchange better in patients with unilateral pleural effusion. We chose common clinical situations where unilateral pleural effusion is seen in plain chest roentgenograms obtained during the routine work-up of such patients. We did not intend to detect minute pleural effusions, which are not demonstrated in the plain chest roentgenograms. Moreover, even the lateral decubitus films of our patients were obtained just to make sure that the pleural fluid was free.

However, the remarks of Drs. Montiel, Hashemian and Brandstetter are very interesting since they raise the question: where is the transitional zone between health and disease? Small amounts of pleural fluid (15-50 ml) are found in normal individuals. What is the amount which is not physiologic and when does impairment of ventilation and perfusion start are questions that should be answered in further studies.

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Infected Mural Thrombus Caused by Bacteroides fragilis

To the Editor:

I read the case report by Dr. Esteban and his colleagues (Chest 1983; 84:104) with great interest. The patient was a 54-year-old man who had an infected mural thrombus with multiple abscesses. There was no culture of this mural thrombus and Gram-stain was not done. The patient also had healing ulcerations consistent with colitis. Antemortem blood cultures grew Bacteroides fragilis.

I submit that the title of this article, "Endocarditis caused by B fragilis," is very misleading. First, there was no microbiologic evidence that the infected mural thrombus was caused by B fragilis. The source of positive blood cultures for B fragilis could well have been from the colonic ulcerations and not from the infected mural thrombus. Second, if the infected mural thrombus was indeed due to B fragilis, the title should have been "Infected mural thrombus caused by B fragilis." This article will most likely be quoted in the future as endocarditis caused by B fragilis, when, in fact, it was not a case of endocarditis at all.

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To the Editor:

As stated in the case report, postmortem cultures were not obtained of the thrombus in the left ventricle. There is a notorious lack of correlation between the results of cultures obtained at postmortem examination and the results of cultures obtained ante-mortem.12 For this reason, postmortem cultures are not obtained routinely at either of our institutions. Gram stain performed on material from the thrombus and from the myocardial abscess revealed the presence of numerous Gram-negative bacilli. No other microorganism was noted on Gram stain.

It is most unlikely that the antemortem blood cultures which yielded Bacteroides fragilis were the result of the colonic lesions. As stated in the case report, at autopsy the colonic ulcerations were noted to be healing. Moreover, these lesions were not extensive. Histopathologically, the mucosa in these areas was regenerating and there was no evidence of active infection noted.

As stated in the case report, the infection extended from the thrombus through the endocardium into the myocardial wall and even perforated through the apex of the left ventricle which communicated with an abscess cavity. Contrary to the assertion in Dr. Watanakunakorn's letter, the infectious process clearly involved the endocardium as well as the myocardium.

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