For speed, ease and reliability we have adopted the Collier\textsuperscript{4} technique using a mobile unit designed for the purpose.\textsuperscript{4} The technique requires only a 30-sec period of rebreathing, which is more easily tolerated by patients than the 1½ min first-stage of the two-stage technique. The rebreathing bag is filled to about 1½ times the patient's tidal volume with a suitable mixture of oxygen and carbon dioxide (not pure oxygen, as stated by Drs. Burki and Albert) adjusted to give a bag \textit{P}co\textsubscript{2} about 10 mm Hg above the expected PVCO\textsubscript{2} but neither the volume nor the initial Pco\textsubscript{2} need be exact, and the procedure is repeated if an equilibrium plateau is not obtained. The technique is applicable to patients breathing spontaneously whether intubated or not, and also to patients on artificial ventilation.

The statement that an elevated PaCO\textsubscript{2} invalidates the use of the formula PVCO\textsubscript{2} = 0.8 × Pco\textsubscript{2} is also incorrect. This relationship, determined empirically\textsuperscript{6} and theoretically,\textsuperscript{5,4} takes into account the nonlinear characteristic of the CO\textsubscript{2} dissociation curve and automatically corrects for the level of PaCO\textsubscript{2}. The relationship is altered by reductions in cardiac output, and decreased arterial oxygen saturation, anemia and polycythemia all have minor effects. The alteration in the relationship of PFCO\textsubscript{2} to Pco\textsubscript{2} by these factors is predictable\textsuperscript{6} and can be used to determine reductions in cardiac output without central catheterization. Further, increases in respiratory quotients do not invalidate measurements of cardiac output using the PVCO\textsubscript{2} : Pco\textsubscript{2} relationship, but may affect subsequent estimates of mixed venous oxygen saturation from the cardiac output.\textsuperscript{6,4}

In our Intensive Care Unit, arterial oxygenation and alveolar ventilation are routinely monitored by ear oximetry and PVCO\textsubscript{2} measurements, reducing the need for arterial blood gas analysis by at least 50%, and the number of arterial lines by about 75%. If a reduction in cardiac output is suspected, both PFCO\textsubscript{2} and Pco\textsubscript{2} are measured, and the reduction in cardiac output quantified using either a graphic approach\textsuperscript{8} or a programmable pocket calculator. The PFCO\textsubscript{2} : Pco\textsubscript{2} relationship may be altered by extreme disturbances in the acid base balance, or change in body temperature. These factors have been taken into account in a more complex program which may also be used in exercise testing.\textsuperscript{6}

Finally, we would emphasize that severe lung disease does not prevent the attainment of good rebreathing CO\textsubscript{2} records and accurate values of Pco\textsubscript{2}.

Our experience has led us to conclude, as Drs. Burki and Albert do, that the measurement of PVCO\textsubscript{2} by rebreathing has the advantages of noninvasiveness, speed and simplicity.

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To the Editor:

Dr. Powles and colleagues have made some important points regarding our article and we would like to clarify some of these points.

We have not described "two rebreathing techniques for measurement of PFCO\textsubscript{2}"; we have described in detail only the Campbell and Howell technique and have not described the Collier technique in our article.

Powles et al raise an important point concerning our statement regarding the accuracy of the relationship PFCO\textsubscript{2} = 0.8 × Pco\textsubscript{2} at high levels of Pco\textsubscript{2}. It is important to note that McEvoy et al (Br Med J 1974; 4:687-90) derived this formula as the best approximation in a group of 19 patients, of whom 9 patients had an SaO\textsubscript{2} < 90%, 3 patients had right heart failure, and 4 patients had respiratory exchange ratios > 1.0. As McEvoy et al noted, and as indicated in our article, the Pco\textsubscript{2}, the cardiac output, SaO\textsubscript{2}, and respiratory exchange ratio, affect the relationship of PFCO\textsubscript{2} to Pco\textsubscript{2}. Thus, the relationship PFCO\textsubscript{2} = 0.8 × Pco\textsubscript{2} is an approximation, although it is sufficiently accurate for most clinical purposes. In clinical practice, an increase in Pco\textsubscript{2} is almost invariably associated with a decrease in SaO\textsubscript{2} and frequently with right heart failure. It was our attempt to caution the reader to take these factors into account in relating the PFCO\textsubscript{2} to the Pco\textsubscript{2} in these clinical circumstances.

Our paper did not discuss the measurement of cardiac output by the use of these noninvasive techniques. Hence, the comments regarding the respiratory quotient and cardiac output, although interesting, are not apropos.

Nevertheless, we are in agreement with the letter from these authors in that the noninvasive measurement of PFCO\textsubscript{2} due to its speed and simplicity, is of major clinical value—provided that the observer is aware of the factors affecting the relationship between PFCO\textsubscript{2} and Pco\textsubscript{2}.

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Body Positional Effect on Gas Exchange in Unilateral Pleural Effusion

To the Editor:

We read with interest the article by Sonnenblick et al (Chest 1983; 83:784-85) where blood gases were analyzed in patients with unilateral pleural effusions. It was found that when the affected side was dependent, there was worsening gas exchange, as reflected in a lower PaO\textsubscript{2} when compared to gas exchange when the affected side was in the upright decubitus position. We believe, however, that the value of the study was compromised, as the authors did not rule out an accompanying "occult" contralateral effusion by more sensitive techniques than routine roentgenogram. Five of their eight patients had diseases that may have been associated with a contralateral effusion if M-mode ultrasonic examination was performed.\textsuperscript{1} Even if only bilateral decubitus chest roentgenograms were obtained, the three patients reported with cardiac failure may have been found to have bilateral effusions.\textsuperscript{1} It is therefore quite possible that the

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findings of Sonnenblick et al of the greatest positional changes in PaO\textsubscript{2} 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.\textsuperscript{4,3} 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.\textsuperscript{4}

Even though it has recently been reported that sensitive findings on routine roentgenogram such as the thorn sign\textsuperscript{5} and displacement of a sharp costophrenic angle\textsuperscript{6} suggest effusions, as much as several hundred milliliters of loculated pleural fluid may remain undetected without the use of other diagnostic testing.\textsuperscript{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 comments 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. Antemortum 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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REFERENCE


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.\textsuperscript{14} 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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