Monitoring Oxygenation

To the Editor:

Ghows et al conclude their recent report by saying that a transcutaneous oxygen electrode can reliably monitor oxygenation in patients undergoing bronchoscopy. Their data do not support this contention.

With normal arterial oxygen levels (PaO₂) of 85 to 95 mm Hg, transcutaneous oxygen levels (tcPO₂) ranged from 45 to 90 mm Hg, while with a tcPO₂ near 40 mm Hg, PaO₂ ranged from 50 to near 80 mm Hg — not a very helpful guide to normoxia or hypoxia! Consider the internist who reads that a transcutaneous oxygen sensor (type not named) "correctly detected all instances when PaO₂ was less than 65 mm Hg. The sensor was noninvasive and caused no adverse effects." It would seem reasonable for him to assume that such a device would reliably distinguish between normoxia and hypoxia in his patients undergoing bronchoscopy, and avoid the expense and hazards of arterial blood gas analysis. Wrong!

Their results may have been affected by 1) ignoring the response time of the electrode, 2) local pressure on the electrode reducing capillary perfusion, or 3) taking pre-bronchoscopy readings before tcPO₂ was stable.

In vivo 90 percent response time (90 percent of the time taken to reach a new steady state after a step change in oxygenation) of most transcutaneous electrodes is on the order of minutes, so tcPO₂ might not detect rapid oscillations in PaO₂ at all! Ghows et al fail to mention this problem in the design of their study. It should be appreciated that upon applying an electrode to the skin, tcPO₂ values will rise, reaching a plateau at 10 to 20 min; this value will depend on PaO₂, the electrode operating temperature, the position of the electrode, skin perfusion, local tissue oxygen consumption, skin thickness, and response time of the electrode, not to mention interference from anesthetic agents. Nothing is said of the monitor's characteristics in the report.

The authors state that "measurements of tcPO₂ correlated well with PaO₂." They did not. The correlation coefficient of 0.69 proves this point, as it indicates that less than 50 percent of the variability in tcPO₂ was accounted for by changes in PaO₂ values (variance r² = 0.476). They further quote a significance value of p < 0.001, indicating that the two methods are related; however, it would be amazing if two methods (blood gas analyzer and transcutaneous electrode) designed to measure the same quantity were not related.

Although measurements of tcPO₂ were statistically significantly correlated with PaO₂, the degree of correlation reported tells us little about the accuracy of the transcutaneous monitor: A plot of the differences between tcPO₂ and PaO₂, against the mean of the two estimates of arterial oxygen would be a more helpful guide, and would allow the calculation of the mean difference and 90 percent confidence intervals for the difference between tcPO₂ and PaO₂.

The implication in the authors' concluding sentence is neither safe nor cautious, as it suggests that a cutaneous oxygen sensor "affords a safe and reliable means of monitoring oxygenation in hemodynamically stable adults" — without reference to arterial blood gas measurement. Yet well-conducted studies of transcutaneous monitoring in adults always warn of the necessity to check tcPO₂ values against PaO₂ values, even if excellent correlation between the two is present.

Colin Lanigan, M.B., M.R.C.P.I., and Jose Ponte, M.D., Ph.D., F.F.A.R.C.S.,
King's College School of Medicine and Dentistry,
King's College of London,
London, England

References
1. Ghows MB, Josen MJ, Chuang MT, Sacks HS, Tierstin AS.
2. Rithalia SV, Booth S. Factors influencing transcutaneous oxygen tension. Intens Care World 1985; 2:126-31

To the Editor:

Drs. Lanigan and Ponte correctly point out that a given value of transcutaneous oxygen tension (tcPO₂) may be associated with a range of arterial PaO₂ values, the precise relationship dependent upon several variables. In our study, the transcutaneous sensor (Biochem Lifespan 100) was placed on each patient at least 30 min before the first arterial blood gas analysis was performed, and tcPO₂ measurements were stable in each instance.

They also remind us that there are several ways to analyze agreement between two measurements. We thank them for their reference 4, which had not yet been published when we submitted our paper. By that method, the mean difference between tcPO₂ and PaO₂ was 25.3 torr, with a 90 percent confidence interval of 21.2 to 29.4. This is a more general way of expressing our finding that tcPO₂ below 40 indicates that PaO₂ was below 65 mm Hg.

Because of added patient discomfort, cost and inconvenience, arterial blood gases are not usually monitored in hemodynamically stable patients during fiberoptic bronchoscopy. Transcutaneous measurements are both noninvasive and continuous. We continue to believe that these advantages, coupled with an acceptable level of agreement between PaO₂ and tcPO₂ measurements, justifies the conclusion that transcutaneous PO₂ monitoring is safe and reliable in stable patients undergoing bronchoscopy.

Mark J. Rosen, M.D., F.C.C.P., and Henry S. Sacks, Ph.D., M.D.,
Department of Medicine,
Mount Sinai Medical Center
New York

BAL in Asbestosis

To the Editor:

Robinson et al reported their experience with bronchoalveolar lavage (BAL) studies in patients with asbestosis. As expected, they found significant neutrophilic and eosinophilic alveolitis. An unexpected finding was the absence of asbestos bodies (AB) in the BAL fluid of one-third of their patients, all the more surprising since 20 of 27 patients had been exposed to crocidolite, an asbestos type which easily forms asbestos bodies. Those results disagree with our own experience. Indeed, we found AB in the BAL fluid of 58 of 59 patients with asbestosis (defined by similar clinical and radiologic criteria) and in 131 of 138 patients with asbestos-related benign pleural disease.

The reason for such a large discrepancy between the studies is most probably of a technical nature. Indeed, Robinson et al examined cytospin preparations of BAL fluid with 10³ cells per slide, ie, only about 0.2 percent of the total amount of fluid recovered (mean recovery 49.10³ cells), so that low AB concentrations can easily be overlooked. By contrast, we examine at least 20 ml of fluid after hypochlorite digestion and filtration, with our technique, low concentrations of AB (less than 10/ml fluid) were found in nine of 59 patients with asbestosis.

We conclude that usual methods of examining BAL fluid are not
sensitive enough to detect low concentrations of AB as can be found in patients with chest x-ray results suggestive of interstitial lung disease but with ill-defined exposure to asbestos; in such cases, large amounts of fluid have to be processed and examined.

J. C. Yernault, M.D., F.C.C.P.; P. De Vuyst, M.D., and P. Dumortier, Université Libre de Bruxelles, Hospital Erasme, Brussels, Belgium

REFERENCES

An Error of Omission

To the Editor:

We read with great interest a recent article entitled "Posterior Mediastinal Sarcoidosis" by Rosseel et al (Chest 1986; 90:462-64). As the authors indicate, posterior mediastinal lymphadenopathy in sarcoidosis has rarely been recorded. It may be expected that frequent use of CT in these cases may change our current concept about the incidence of such involvement in the disease, and additional well-documented cases are valuable contributions.

It appears, however, that there may be an inadvertent error in the selection of illustrations reproduced in the article. While Figure 1A shows right paratracheal and Figure 2B demonstrates right paratracheal, anterior tracheal, prevenous and prearterial lymph nodes, the sections illustrating posterior mediastinal nodes are omitted. Their inclusion would markedly increase the intrinsic value of the paper.

Sheila D. Davis, M.D., and Yahya M. Berkmen, M.D., Department of Radiology, Division of Pulmonary Radiology, New York Hospital-Cornell Medical Center, New York

To the Editor:

We read with interest the comments of Drs. Davis and Berkmen on our recent article.

As they expect, with the frequent use of the CT-scan some authors have already reported an increased incidence of posterior mediastinal lymph node enlargement with bilateral hilar lymph node enlargement and various extrathoracic manifestations of sarcoidosis.1-4 (Schabel SI, et al. Radiology 1978, 129:591-93, Kutty CPK, Varkey B. Postgrad. Med. 1982, 71:64-6). However, we report two cases of posterior mediastinal lymph node involvement without any other extramediatinal disease.

In the first patient, the mass was dissected between the azygos vein and the esophagus, as is documented on Figure 1 (with right paratracheal extension at a more cranial level).

In the second patient, the mass was located again between the azygos vein and esophagus. To clearly demonstrate this, copies of the CT scan film at a lower level are inclosed (Fig 2).

B. Rosseel, M.D., and B. Cham, M.D., Academisch Ziekenhuis, University of Brussels, Brussels, Belgium

REFERENCES

Reprint requests: Dr. Cham, Université of Brussels, Academisch Ziekenhuis, 1090 Brussels, Belgium