cuff inflation is seldom seen, and an increase of 1 ml or less in cuff volume can markedly increase LWP.

What can be done to minimize the possibility of cuff-induced tracheal damage? First, select the largest endotracheal tube that can be insertedatraumatically. A greater cross-sectional area ratio between the tube and the trachea means less air must be injected into the cuff to effect a seal. Second, use a tube with the greatest cuff length and resting cuff volume. As our report showed, the variability between different cuffs, even from a specific manufacturer, can be significant. Third, measure ICP, even though the absolute pressure may not reflect the true LWP. A progressive increase of ICP necessary to maintain a cuff seal, particularly when high peak inflation pressure is necessary, may indicate a need to change the tube, reposition it, or consider alternatives, such as intermittent cuff inflation.12 As we stated in our study, the ultimate solution awaits new and improved cuff design.

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Figure 1. Typical micrograph of airborne tungsten oxide fibers.

Possible Role of Tungsten Oxide Whiskers in Hard-Metal Pneumoconiosis

To the Editor:

One of the key processes in tungsten technology is the reduction of tungsten trioxide with a gas phase to yield the tungsten metal. Whiskers of slightly reduced tungsten trioxide are a characteristic reaction product during the process.1 Hard metal is manufactured by a process of powder metallurgy from tungsten metal and carbon, with cobalt as a binder. Dust generated during the process of manufacturing hard metals causes clinical and pathologic changes in the lungs of workers, which have occasionally proved fatal. Such industrial airborne dust, which can cause severe lung fibrosis, is of a heterogeneous, multielement type.

In a communication to the editor of Chest in April 1985, Dr. Cullen2 emphasized that the cause of lung disorders that have been associated with exposure to hard metal (tungsten carbide with binder, including cobalt) remains uncertain. By contrast, Dr. Abraham3 argued in the same issue that the causal agent has been clearly identified and that the pathologic condition observed in hard-metal industry employees is produced by cobalt. No previous report has shown or suggested the presence of airborne fibers in the work environment in the hard-metal industry.

In a study conducted as part of a project on optimizing the sample transfer method for analysis of airborne fibers by transmission electron microscopy, airborne tungsten oxide fibers were observed in such an environment. Figure 1 shows a typical photomicrograph of the observed fibers. It is not, however, within the scope of this letter to give more elaborate details of the results. Nevertheless, all fibers were respirable, and about 80 percent of the fibers were 0.3 μm or less in diameter. This suggests that most of these fibers are below the practical resolution limit of the optical microscope.

Inhalation of asbestos fibers has long been associated with a variety of malignant and nonmalignant respiratory diseases. In recent years, however, the category of possibly etiologic fibers has been broadened to include other durable inorganic fibers. Fibers with chemical characteristics, structure, and dimensions different from those of asbestos are suspected to have caused various biologic effects.4,5 In this respect, our observation is compatible with the skeptical view of Dr. Cullen, that occupational exposure to such fibers never has been accounted for in discussions of hard-metal pneumoconiosis. It should also be noted that tungsten makes up a major part of hard-metal production, about 95 percent.

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Bronchoscopically Induced Bleeding

To the Editor:

We read with interest in the October 1991 issue of Chest the excellent review by Cordasco et al1 on the prevention and management of bleeding induced by bronchoscopic biopsy. We would like to comment on another technical aspect of transbronchial biopsy that seems of importance in our experience.

We have encountered severe bleeding (more than 100 ml) after transbronchial biopsy in three patients with sarcoidosis or diffuse lung disease. On each occasion, the sequence of events had been as follows: After closing the cups, a greater than usual resistance had been felt in withdrawing the biopsy forceps. At the same time,
fluoroscopy had shown the bronchovascular markings being drawn quite far in the direction of the hilus. Immediately after tearing off the fragment, profuse bleeding had occurred. Aspiration and the “bleeding lung down” maneuver were able to handle the situation until the bleeding spontaneously subsided. Recovery was uneventful. In one case, microscopy showed a medium-sized pulmonary artery.

Life-threatening bleeding has been attributed to biopsy of a sizable pulmonary or bronchial artery.2,3 Although it has been stated that such an event is unpredictable,4 our experience shows that at least some of those bleedings can be avoided.

Transthoracic biopsy should be performed in the outer portion (cortex) of the lung parenchyma. Advancing the forceps too far can lead to severance of the pleura, resulting in pneumothorax; even advancing the forceps not far enough can lead to biopsy of the larger bronchovascular bundles in the more central (medullar) part of the lung, resulting in severe bleeding. The resistance felt when withdrawing the forceps (which, of course, demands experience) and the fluoroscopic findings can give a warning sign that such an event could occur; it would then seem prudent to release the grasp of the forceps and to biopsy at another place. This could also be a reason for the routine use of fluoroscopy during transthoracic biopsy.

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Effect of Oxygen Therapy on Increasing PaO2 in Hypoxemic Patients with Stable COPD While Breathing Ambient Air

To the Editor:

I read with great interest the article by Dr O’Donohue,1 which appeared in the October 1991 issue of Chest. Over the past 16 years, as director of the Pulmonary Medicine Department, I have followed up several hundred patients with severe chronic obstructive pulmonary disease and secondary cor pulmonale who were receiving supplemental oxygen. Like Dr O’Donohue, I have observed that the PaO2 improves to adequate levels over a three- to six-month period in about 20 percent to 30 percent of patients. The improvement in oxygenation appears to be coincident with the objective improvement of the cor pulmonale, as evidenced by the resolution of peripheral edema and hepatomegaly. Because the bronchial veins, which drain the bronchi and bronchioles, enter the aygoses vein and then the vena cava, it seems reasonable to presume that the respiratory tree is edematous during the presence of the right-sided heart failure and that the edema improves as the cor pulmonale resolves, thereby improving ventilation. It has also been my experience that once a patient is able to discontinue the use of supplemental oxygen, barring an acute exacerbation of the obstructive lung disease, the hypoxemia and cor pulmonale do not recur. I therefore disagree with Dr O’Donohue’s conclusion that the supplemental oxygen should not be discontinued when oxygenation improves.

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REFERENCE
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To the Editor:

We recently read the article by O’Donohue,1 describing the improvement in baseline arterial blood oxygenation in 20 patients receiving transtracheal oxygen therapy (TTOT). The author found an increase in PaO2 and a reduction in alveolar-arterial oxygen gradient after six months of TTOT. The patients had previously been receiving oxygen by nasal cannula for at least six weeks.

We prospectively studied ten patients who were receiving chronic oxygen therapy (12 ± 6 months) by nasal prongs for 18 h a day and who accepted TTOT.2 The patients followed a rehabilitation program before placement of a transtracheal catheter (TTC), but were not permitted to participate in any other rehabilitation program during the one-year follow-up. As expected, pulmonary function test results did not improve, and some parameters even deteriorated. (The FEV1 decreased from 0.78 to 0.62 L [p<0.01]). In our series, PaO2 with the same oxygen flow through the TTC had increased from 66.8 to 71 mm Hg (p<0.04) at the end of the study.2

Several mechanisms have been proposed to explain the clinical improvement observed in patients receiving TTOT, among them a positive end-expiratory pressure effect or decreased inspired minute ventilation. O’Donohue points out the possibility that uninterrupted oxygen therapy for 24 h a day by TTC may have additional therapeutic benefits. We have observed that nocturnal pulse oximetry values are more stable when patients are oxygenated through a TTC,3 and that sustained hemodynamic benefits4 are achieved when 24-h oxygen therapy is administered.

We agree with O’Donohue’s hypothesis that improvement in PaO2 after oxygen therapy may be due to the beneficial effects of oxygen therapy. Uninterrupted long-term oxygen therapy for 24 h a day can be accomplished only by TTC. Thus, this oxygen delivery device should probably be more frequently recommended.

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