REFERENCES

1 Gurevitch MJ. Pressure-controlled inverse ratio ventilation: what have we learned? Chest 1993; 104:664-65
3 von Pohle WR, Anholm JD, McMillan J. Carbon dioxide and oxygen partial pressure in expiratory water condensate are equivalent to mixed expired carbon dioxide and oxygen. Chest 1993; 101:1601-04

To the Editor:

The “standardized criteria” proposed by Dr. Rothfeld in his table are no doubt tried and true, but it would seem to have more application in regards to weaning and “outcomes data” which are of great importance.

They, however, do not necessarily address implementation standards and criteria, which were our concerns in the editorial. As new modes of ventilation continue to “pop up” on newer ventilators along with increasing prices, it may be more important than ever to develop standards so that we can more meaningfully decide whether the added “bells and whistles” justify the significantly increased costs.

The rapidly approaching “new era” in medicine will continue to apply close scrutiny to those of us in critical care and mandates that these efforts begin and continue.

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Increased Frequency of Obstructive Airway Abnormalities With Long-Term Tracheostomy

To the Editor:

I read with interest the article by Law et al, which appeared in the July 1993 issue of Chest.1 We have had the opportunity to care for patients admitted to a 30-bed long-term ventilator unit. In the process of removing tracheostomy tubes from patients who were successfully weaned from mechanical ventilation after prolonged periods, we have noticed six patients with upper airway obstruction as described by Dr. Law. All six had granulation tissue originating from the anterior tracheal wall at the proximal margin of the tracheostomy site. This granulation tissue obstructed the fenestrations of the tracheostomy tube, preventing adequate airflow when the tube was plugged, which required us to perform a bronchoscopy on the patient for evaluation.

We found that performing a bronchoscopy from above with the tracheostomy tube in place overestimates the degree of obstruction. When the tracheostomy tube is withdrawn, the granulation tissue falls anteriorly, into the tracheostomy defect, and no longer significantly interferes with airflow. The tracheostomy tube itself pushes the granuloma into the lumen of the trachea; when the tracheostomy tube is removed, the degree of obstruction falls. The granuloma frequently rests on the greater curve of the tracheostomy, over the fenestrations.

Before realizing how variable the degree of obstruction may be, we initially treated these patients with surgical resection, using electrocautery rather than a laser. In two recent cases, bronchoscopy with the tracheostomy tube removed showed that the granulation tissue fell into the tracheal defect and no resection was done. In both patients, decannulation was successful, the tracheostomy site healed, and no clinical evidence of upper airway obstruction developed. One patient underwent bronchoscopy months later for an endobronchial lesion and there was no significant tracheal obstruction.

We think that before resection of granulomatous tissue at this site, bronchoscopic evaluation should include observation with the tracheostomy tube removed.

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REFERENCE


Asbestos-Related Pleural Plaques and Lung Cancer

To the Editor:

In his review in the June 1993 issue of Chest, Dr. Weiss1 concluded that the risk of lung cancer is not increased in the absence of parenchymal asbestosis among persons with asbestos-related pleural plaques. This conclusion is questionable.

Exposure to asbestos dust can cause asbestosis, pleural abnormalities, and lung cancer. Thus fibrogenic and oncogenic effects in the human lung have a common causal agent, and an association, therefore, exists between the occurrence of plaques and the observed risk of lung cancer. Recently, the question has arisen of whether asbestosis or pleural plaques are related to the occurrence of lung cancer. Although pleural plaques are not a precursor of lung cancer, whether asbestosis is an intermediate stage in the causal process from asbestos exposure to the development of lung cancer is a matter of contention.

Dr. Weiss argued that if the risk of lung cancer is not increased for persons who have pleural plaques but not asbestosis relative to the risk for unexposed persons or for the population at large, then evidence would exist that supports the hypothesis of asbestosis being a necessary precursor for asbestos-related lung cancer.

There are two counterclaims to this hypothesis. First, asbestosis may be a precursor of asbestos-related lung cancer, but, even if it is, it would not have to be a necessary condition. In asbestos-exposed workers, lung cancer can appear without parenchymal fibrosis and vice versa.2,3 Second, the validity of pleural plaques as a marker of asbestos exposure is, in most circumstances, far from perfect, and very large populations would be needed in a valid epidemiologic study before a statistical relation between the occurrence of pleural plaques and an excess risk of lung cancer would be detected.

For example, in one study, the proportion of the Finnish male population exposed to asbestos was estimated to be 13.4 percent.4 Among asbestos-exposed men, the population fraction with pleural plaques was 13.0 percent, whereas only 4.6 percent of the unlikely exposed men had signs of plaques. In two other studies, the mortality risk for lung cancer among asbestos-exposed men was about twofold.2,3 It follows, therefore, that the mortality risk ratio of lung cancer associated with the presence of pleural plaques is as low as 1.11. With a significance level of 5 percent and a power of 80 percent, the cohort size required to detect a risk ratio

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