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Asbestos-Related Pleural Plaques and Lung Cancer

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A M E R I C A N C O L L E G E O F



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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 my 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 When the tracheostomy tube is withdrawn, the granulation tissue falls anteriorly, into the tracheostomy defect, and no longer significantly interferes with airflow.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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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

of this magnitude would comprise several thousands of lung cancer cases and would be unrealistically large.

Despite the fact that some "negative" studies on asbestos-exposed populations have turned out to be of questionable quality or based on inadequate population-time experience, the logic of inference from epidemiologic evidence to the probability of causation should be flawless before a conclusion is drawn, because, in tort suits, the drawing of hasty conclusions can affect compensation decisions.

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To the Editor:

Drs. Nurminen and Tossavainen are correct that there is some contention as to whether asbestosis (parenchymal) is a necessary condition to diagnose asbestos-related lung cancer. Most of the current literature on this subject in my introduction, however, provide data and opinions that asbestosis is necessary.

In their first counterclaim, they state that lung cancer can appear in asbestos-exposed workers without asbestosis and cite their references 2 and 3 to support this. Their reference 2, however, by Anttila et al, is not an epidemiologic study. It is merely a collection of *resected* cases of lung cancer and is, therefore, biased by the exclusion of cases with substantial asbestosis since this condition would obviate resection because of poor pulmonary function.

Their reference 3 by Nurminen was an epidemiologic mortality study using death certificate information on both lung cancer and asbestosis in a cohort of asbestos factory workers. He observed 13 cases of lung cancer and expected only 6 based on national rates by age and sex, giving a relative risk of 2.2 with no adjustment for differences in smoking habits between the cohort and the Finnish population. Tables 4 and 5 in the Nurminen article reveal that 6 of the 13 cases of lung cancer also had diagnoses of asbestosis on the death certificates. Therefore, the relative risk for those without asbestosis, assuming that asbestosis was always recorded on the certificates, would be seven observed and six expected or 1.2, a value which is easily the result of chance. Since smoking is the dominant causal factor for lung cancer and high proportions of asbestos-exposed workers have been smokers, it is obvious that some lung cancers among such workers would have occurred without asbestos exposure.

In their second counterclaim they say that the validity of pleural plaques as a marker of asbestos exposure is far from perfect. I agree with this since plaques are sometimes not visible on chest roentgenograms and this may bias the results. The three autopsy studies I described, however, also yielded insignificant estimates of relative risk. Even the pooled data for the autopsy

studies, not presented in my review, gave a statistically insignificant odds ratio of 1.38 with 95 percent confidence limits of 0.81 to 2.37.

I agree that it would be necessary to do very large studies to validate a very low elevated relative risk for lung cancer among people with pleural plaques who do not have asbestosis, and this may be unrealistic. It is difficult to prove a negative. However, their example is only speculation. We must make reasonable decisions based on the best hard data, and there is no such thing as flawless epidemiologic evidence of an observational nature.

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Survival of Patients Undergoing Nd-YAG Laser Therapy Compared With Nd-YAG Laser Therapy and Brachytherapy for Malignant Airway Disease

To the Editor:

I read with interest the article by Shea et al¹ in the April 1993 issue of *Chest*. The authors describe their experience with endobronchial therapy for exophytic inoperable squamous cell carcinoma of the lung with Nd-YAG plus or minus low dose brachytherapy. Their conclusions about longer survival of patients receiving combination therapy may be related to the deficiencies that exist in their study rather than a true benefit. I cite various factors that would affect survival of inoperable lung cancer and that are not addressed in their study.

First, this was a retrospective study and patients were not randomized to each arm. Second, the patients had inoperable squamous cell carcinoma. Were they inoperable because of poor pulmonary reserve, stage IIIB disease, or stage IV disease? Patients in each group will have a marked difference in survival irrespective of the therapy administered.

Third, why was endobronchial therapy given? Just because an endobronchial exophytic lesion existed or because these patients had postobstructive pneumonia? They mention that patients were symptomatic, but the exact severity of their symptoms and complications before therapy were not mentioned. Did more patients in the laser therapy group have postobstructive pneumonia? What was the performance status, P(A-a)O₂ gradient, FEV₁, etc, of the patients in the two groups?

Endobronchial brachytherapy should not be administered to all patients with an exophytic lesion. Without any kind of systemic therapy, it is hard to realize survival benefits in patients with metastatic nonsmall cell lung cancer. Patients with problems likely to lead to imminent death, *ie*, gas exchange abnormalities from major bronchial obstruction or postobstructive pneumonia are likely to benefit from immediate Nd-YAG laser resection of exophytic tumors. It would make sense to follow this with endobronchial brachytherapy or subsequent laser resection on a timely basis to keep the airway open, depending on the overall condition of the patient.

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