spirit of cooperation and the appropriate use of technology, primary care providers and TB specialists can make a very effective team for preventing nosocomial TB transmission while reducing costs at the same time.

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Diagnosing Malignant Pleural Mesothelioma

Exposure to asbestos causes important benign and malignant intrathoracic disease. Disabling, often fatal, pulmonary fibrosis was a recognized disease of insulation workers and asbestos miners in the early 1930s. Nicholson and colleagues reported benign pleural thickening, plaques, and calcification not only in exposed workers but also among family members exposed to a worker’s clothing. With the interdiction of the use of asbestos in construction and shipbuilding in the United States, severe fatal pulmonary fibrosis has greatly diminished. However, workers and the general population are still exposed when old buildings and ships are renovated. Despite strict laws regarding asbestos abatement, the green dumpsters overflowing with debris that dot our streets at renovation sites continue to be a hazard. The malignant manifestations of asbestos exposure, lung cancer, and malignant mesothelioma usually do not appear until 20 to 40 years after asbestos exposure. Understandably, they continue to be an increasing medical problem. More than 1,000 pleural mesothelioma deaths were reported in England and Wales in 1995. Feto and coworkers estimated that the number will increase to more than 3,000 per year within 25 years.

While controversy continues to rage about the potential cocarcinogenic role of cigarette smoking and asbestos exposure in lung cancer, no disagreement exists as to causation of pleural malignant mesotheliomas. Asbestos exposure, even for relatively short periods of time, causes malignant pleural mesothelioma. When physicians encounter patients with pleural disease and a history of exposure to asbestos, they must determine if a patient has a benign or malignant process. Benign pleural plaques, thickening, and calcifications are readily identified on routine chest radiographs. Confirmation is available by CT scanning. Except in rare instances where the pleural thickening encases the lungs, causing impaired pulmonary function, most of the benign asbestos-related pleural changes require no further study, even though they may progress slowly with time. The question of malignant pleural involvement arises when a patient presents with chest pain, pleural effusion, pleural nodules, masses and scallops, and involvement of the mediastinal pleura. Pleural effusions may occur in benign and malignant pleural disease. CT scans and MRI are of great aid in delineating these manifestations of neoplasia. However, in some patients, routine radiographs, CT scans, and MRI cannot differentiate between benign and malignant pleural disease. Positive scans suggest the presence of cancer. The diagnosis of malignant pleural mesotheliomas frequently is difficult. In the absence of a thorough occupational history that includes remote employment, clinicians often omit the disease from the differential diagnosis. If a history of asbestos exposure is obtained from a patient with pleural abnormalities, radiologists and pulmonologists must decide whether the radiographic pleural changes are benign or malignant. Thoracic CT scanning and MRI have greatly enhanced radiographic accuracy. The
finding of pleural masses, exudative effusion, and mediastinal pleural involvement usually are indicators of malignant disease. Pleural fluid cytology and closed needle biopsy of the pleura are usually inadequate for diagnosis. A cytologist may report malignant cells or metastatic epithelial malignancy. In most instances, open pleural biopsy or thorascopic biopsy are necessary for diagnosis. Special stains are required, especially when differentiating adenocarcinoma from malignant pleural mesothelioma. On occasion, even these studies leave doubt about the specific diagnosis.

The report by Benard and colleagues in this issue of CHEST (see page 713) describes the use of 18-fluoro-2-deoxyglucose positron emission tomography (FDG-PET) scanning scores in differentiating malignant mesothelioma from benign pleural disease. Twenty-two of 24 patients with malignant pleural disease were identified by positive FDG-PET scan. Two of these were adenocarcinoma, not mesothelioma. Two patients with malignant mesothelioma had false negative FDG-PET scans. There was one false positive in 4 patients with benign pleural disease. The distribution of radioactivity roughly paralleled the anatomic pattern and extent of disease. Metastatic mediastinal lymph nodes were identified in 12 patients. These results support the authors’ proposal that FDG-PET scanning is a valuable aid in the evaluation of patients with asbestos exposure. Unfortunately, neither FDG-PET scans nor 67Ga scans can differentiate malignant mesothelioma from metastatic carcinoma involving the pleura.

Although the prognosis for both malignant mesothelioma and metastatic pleural carcinoma is poor, some patients with malignant mesothelioma have lived for as long as 7 years with palliative therapy or none. Currently, there are several therapeutic trials that use surgical resection (usually with extrapleural pneumonectomy), chemotherapy, and gene therapy for mesothelioma, whereas metastatic carcinoma to the pleura is usually treated with palliative therapies, pleurodesis, or both. Malignant pleural mesothelioma in an occupationally exposed patient is a compensable disease, but the role of asbestos exposure in the etiology of lung cancer continues to be debated. Therefore, it is necessary to differentiate mesothelioma from carcinoma. In most patients, this can be accomplished only by examining pleural tissue obtained by surgical biopsy, applying special stains and routine histologic examination, and, in a few patients, electron microscopy. At best, FDG-PET scanning can suggest the presence of malignancy before a biopsy, but this does not obviate the need for tissue diagnosis. FDG-PET scans may find a role as a supplement to CT scans and MRI in identifying which patients require biopsy and which sites are best for biopsy and in delineating the extent of the disease.

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Tissue Capnometry as a Monitoring Strategy for Critically Ill Patients
Just About Ready for Prime Time

According to the English dictionary on my bookshelf, a tonometer is a device for measuring the pitch of a tone or, alternatively, a pressure, such as intraocular pressure or the vapor pressure (ie, partial pressure) of a gas. In 1964, Bergofsky introduced the concept of using a hollow viscus, such as the urinary bladder or the gallbladder, as a "convenient in vito tonometer in which to equilibrate liquids until they assume the gas tensions of the surrounding tissues."1 Hollow viscus tonometry was later used by Dawson and colleagues2 to measure mucosal gas (oxygen and carbon dioxide) tensions in the small intestine. Subsequently, in a series of pioneering studies, Fiddian-Green and colleagues3-5 promulgated the idea that tonometric measurements of tissue PCO2 in the stomach or sigmoid colon could be used to estimate (intra) mucosal pH (pH) and thereby to monitor visceral perfusion in critically ill patients.

On theoretical grounds, measuring tissue pH to assess the adequacy of perfusion is an extremely attractive concept. As a consequence of the stoichiometry of the reactions responsible for the substrate-level phosphorylation of adenosine diphosphate to