Lieutenant of the Men of Death

A little over 75 years ago, Sir William Osler borrowed the phrase "Captain of the Men of Death" from John Bunyan and applied it to pneumonia, which he described as one of the most widespread and fatal of all acute diseases. He considered the Pneumococcus to be the sole etiology of lobar pneumonia, but postulated that other organisms might contribute to this infection as secondary invaders. Osler also knew that the Pneumococcus as well as the other bacteria were commonly found as part of the normal oral and pharyngeal flora, and that pneumonia probably resulted from colonization with a particularly virulent strain or from a breakdown in normal host defenses. Although antibiotics dramatically changed the mortality rate, pneumonia remains the seventh leading cause of premature death in the United States. In addition, pulmonary infections are common in patients with other serious illnesses and are frequent contributing factors to death in critical care units. There continues to be a need for research in this illness. If pneumonia is no longer a Captain, it at least seems to deserve a commissioned rank. Perhaps Lieutenant is appropriate.

Today, it seems clear that most bacterial pneumonias are caused by organisms that have previously colonized the upper airway. Under normal circumstances, most of the upper respiratory flora consist of relatively nonvirulent aerobic and anaerobic bacteria. "Pathogenic" bacteria (Streptococcus pneumoniae, Hemophilus influenzae, and occasionally gram-negative rods) are present in many normal people, but are usually present in small numbers. Most people are able to aspirate small volumes of this "normal flora" on a daily basis without developing pneumonia. If larger volumes of normal pharyngeal secretions are aspirated (usually because of altered consciousness, seizures, or pharyngeal dysfunction), the resultant pneumonia is due to a mixture of aerobic and anaerobic organisms. A traditional respiratory pathogen may or may not be present in this mixture.

Pneumonias due to traditional pathogenic bacteria probably occur because of increased bacterial counts of these organisms in the pharynx or because of alterations in host defense as suggested by Osler. Viral upper respiratory infections increase the numbers of bacteria in pharyngeal secretions and decrease alveolar macrophage function. Gram-negative bacteria occur with increased frequency in elderly and alcoholic patients and are particularly common in critically-ill patients. It is not uncommon to recover more than one organism from the airways of patients with typical pneumonia. Although traditional thought has been that these additional organisms represent contamination of the specimen, the data suggest that polymicrobial pneumonia with two or more traditional pathogens is not uncommon. Bacterial synergism may play a role in establishing some of these infections.

Most community-acquired pneumonias respond well to empiric antibiotics. The epidemiologic features of the illness and the careful evaluation of the host for associated conditions are usually the most important considerations determining therapy. Sputum gram stains and cultures are useful largely to safeguard against unexpected virulent bacteria which might not respond to empiric therapy. Accurate bacteriologic study of these infections is impossible with sputum, and our current knowledge is partially due to the development of "invasive" techniques such as tracheal aspiration, direct needle aspiration of the lung, and bronchoscopy with the protected brush catheter (PBC). There has been some disagreement in the literature concerning the relative accuracy of these techniques, but useful information has been obtained with each. The most accurate results have been obtained when well designed protocols have been carefully followed. These techniques are very useful from a research standpoint in accurately defining the bacteriology of various respiratory conditions. Although the techniques are clearly not indicated for routine clinical use, they offer a means of obtaining accurate bacteriologic studies in circumstances where such results are important in determining therapy.

One circumstance in which an accurate bacterial diagnosis may be helpful is pneumonia in critically-ill ventilated patients. In this issue of Chest, Villers and coauthors describe the use of the PBC in such patients. The results indicate that the central airways rapidly become colonized in all intubated patients, usually with two or more "pathogenic" bacteria. The central airways thus become the bacterial equivalent of the pharynx in nonintubated critically-ill patients and harbor several organisms which may potentially invade the lung. In the patients who develop...
oped pneumonia, the PBC was useful in determining which of the organisms from the central airways were responsible for the infection. The use of the PBC was similar in purpose to its use in nonintubated patients, i.e., it allowed the investigators to traverse a colonized area and obtain an uncontaminated specimen from a distal site. The authors suggest that the PBC was "contaminated" in two patients whose PBC and tracheal cultures yielded identical results. A more likely explanation is that the PBC obtained an uncontaminated specimen, and that the bacteria recovered were actually present at the distal site despite lack of confirmation by the "reference" methods. Unfortunately, the authors were not able to perform quantitative cultures, which might have determined which of the organisms were pathogens.8,9

The excellent results indicate that the PBC is capable of obtaining accurate samples from the distal airways in intubated patients. The patients would have been appropriately managed with antibiotic selection based on tracheal culture results plus careful observation of response so that the procedure may not be indicated in all intubated patients with pneumonia. Nevertheless, it appears that accurate samples may be obtained in these complicated patients when they are needed for research or for planning therapy.

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REFERENCES

Alterations of Pulmonary Epithelial Permeability Caused by Smoking and Other Injuries to the Lungs*

The study by Minty et al in this issue of Chest (see page 531) on the effect of smoking low yield cigarettes upon the permeability of the pulmonary epithelium provides one more piece of evidence that use of these cigarettes will do little to decrease lung injury associated with smoking. Both Russell et al10 and Ashton et al11 have shown that nicotine levels do not fall as much as would be anticipated when subjects smoke low-nicotine cigarettes. Smokers appear to compensate for changes in nicotine content by smoking more cigarettes or altering their pattern of smoking. It was therefore not surprising that Minty and colleagues found that carboxyhemoglobin levels remained high when commercially available low tar, low nicotine cigarettes were used. However, when special cigarettes which contained a high nicotine content but generated less carbon monoxide were smoked, carboxyhemoglobin levels unexpectedly remained elevated. Although the pattern of smoking might alter CO yield relative to nicotine content, it is not obvious why subjects would choose to keep their CO levels constant.

Regardless of what motivates smokers to keep nicotine and carbon monoxide levels constant, the present study indicates that the abnormally rapid clearance of 99mTc-DTPA aerosol from the lungs does not decline when low yield cigarettes are smoked. The advantage of utilizing this approach for evaluating the effect of smoke is the rapidity with which this functional test responds when smoking is discontinued.4,5 Because elevated clearances have been associated with a variety of both chronic and acute lung injuries in experimental animals and man, it seems likely that this effect of smoking is also related to damage of the pulmonary parenchymal membranes.

Since its introduction eight years ago,6 the radiolabeled aerosol procedure has proved increasingly useful for detecting pulmonary epithelial abnormalities in a wide variety of illnesses. The initial observations that the clearance of 99mTc-DTPA from the exchange surfaces of the lung is accelerated in a variety of chronic interstitial lung diseases suggests that the permeability of the epithelium might be increased.6 These abnormalities have focused attention upon the morphologic changes which occur in the alveolar epithelium under such diverse conditions as idiopathic pulmonary fibrosis,