technique we describe as “normal”—that is, manoeuvre 1—should be universally adopted.

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Adventitia

Déjà vu: tuberculosis and AIDS

Much has been written in the last decade about the fear of occupational infection as a factor in the reluctance of physicians to care for patients with AIDS. The fear of occupational infection was well known to pulmonary physicians who began their careers in the middle of this century, but then the infectious agent was the tubercle bacillus.

Late in 1948, during my final year of training in internal medicine, I made the decision to undertake subspecialty training in pulmonary disease. Soon after it became known in the hospital where I worked that I was going to the Trudeau sanitarium to study, I was approached by a fellow resident who had been treated in the sanitarium for tuberculosis. He tried to convince me that I was very unwise to go to work in a tuberculosis sanitarium because of the high risk of developing the disease.

His response was understandable. The tuberculosis death rate in the USA in 1948 was 48 per 100 000 living persons, compared with a rate in 1989 of 0.7. When tuberculosis was diagnosed in patients who were in general hospitals during that time, prompt isolation and transfer to a sanitarium followed as quickly as possible. Nurses and physicians had limited personal experience with the disease and were fearful of being infected themselves. Although tuberculosis was a definite occupational hazard, patients with undiagnosed disease in general hospitals presented a greater risk than patients in tuberculosis sanitarium who were trained to protect staff.

Treatment options were limited and stays of one to two years in a sanitarium were common. Streptomycin, the first effective drug available for treating the disease, was discovered in 1944. Used in short courses the effects of the drug on symptoms and the chest radiograph were dramatic, but all too often they were shortlived. The emergence of resistant mycobacteria with loss of effectiveness of the drug was frequent. Para-aminosalicylic acid was found to be a weakly effective

antimycobacterial agent in 1946. By 1949 in vitro and animal work, followed by studies in patients with tuberculosis, had confirmed that combined treatment with streptomycin and para-aminosalicylic acid greatly delayed the emergence of streptomycin resistant tubercle bacilli.

In July 1949 I arrived at the Trudeau sanitarium in Saranac Lake, New York, with my wife who was three months pregnant with our first child. The first question I was asked was: “Where and when did you cure?” When I answered that I had not had tuberculosis the follow up question was: “Oh, then your wife must have cured.” It was then that I learned that most pulmonary physicians and thoracic surgeons at that time had themselves had tuberculosis. Small wonder that the sanitarium director was shocked to find that my wife was pregnant.

The introduction of isoniazid as an anti-tuberculosis agent in 1954 ushered in the modern era of effective chemotherapy of tuberculosis. Within a few years mortality and morbidity decreased dramatically, and the fear of tuberculosis as an occupational hazard for healthcare givers faded.

The emergence of AIDS in 1981, followed later in the decade by a rising incidence of tuberculosis and the emergence of multiresistant strains of *M tuberculosis*, changed the complacency of nurses and physicians regarding the risks of on-the-job infection. As had been true when I started in pulmonology, the possibility of exposure to microorganisms for which there was ineffective treatment made the taking of appropriate precautions a part of the day's work. Once again physicians were dealing with a disease that commonly kills young people, and for which treatment is relatively ineffective. In the words of Yogi Berra, the great catcher for the New York Yankees baseball team: “It was déjà vu all over again.”

GORDON L SNIDER

This column is now open to allcomers for suitable contributions (maximum 700 words). We would like to keep this column running.—SGS