treatment, social rejection, overprotectiveness by parents. In general, children cope well with these problems, but problems still exist.” The report from the San Diego (Calif) group is a well-controlled study using standard psychologic measuring instruments that extend this sort of work, with similar conclusions, to an older population.

Pulmonologists as a group are not noted for enthusiasm about the psychology literature. The value of this article is that it demonstrates that this new population does not need to be approached with kid gloves. While some of the medical problems may be unique, the patients are not “forever young,” and they can be treated as perfectly competent individuals. Ten years ago, critics of this article would have pointed out that this population of CF adults probably were those who had experienced minimal disease early in their childhood or were those whose conditions had been diagnosed relatively late in life. I do not think that is the case for most CF adults today. Most have had health problems throughout their lives and still manage to function well. It is entirely possible that they are functioning well because the health care system (and now society) expects them to do so. That makes for interesting speculation that the authors were polite enough to avoid.

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Elimination of Tuberculosis in the United States

A campaign is underway to eliminate tuberculosis from the United States.1,2 The goal of reducing the number of new cases to less than one per million population per year by the year 2010 has action committees working hard. The decline in the incidence of tuberculosis has reversed. We pulmonary physicians, who have our heritage in tuberculosis, must ensure the success of this elimination program. Because of the enormity of the problem and the complexity added by acquired immunodeficiency syndrome (AIDS), some say this aim is impossible. Because of the chance for national calamity and the complexity added by AIDS, others say it is imperative. All agree the time must be now. The effort must be complete. This dream can be realized even though it has failed so far. Patients, physicians, and society have failed, but we cannot let failure continue.

Patients fail to seek medical attention, attend clinics, and take medicines because of social stigma, expense, inconvenience, indifference, or ignorance. They may not perceive the rewards of curing tuberculosis to be as great as the nuisance of following a medical program. Patients often fail to realize they have to continue to take medicine after their symptoms are gone. Some may not care if they contract or spread this disease. Drug abuse and mental deficiency contribute to the problem. We must anticipate and prevent noncompliance. We must remind, reward, supervise, and persist.

Physicians often fail to diagnose and properly treat tuberculosis and tuberculous infection (positive purified protein derivative [PPD]). They may be indifferent, ignorant, or inexperienced. They may perceive that there is ambiguity or a lack of a standard of care. They may not act on a tuberculin test if they are confused by information about the booster effect, different cut points for the size of the skin reaction, and different times the test should be read.

Assuring compliance is a responsibility of physicians. We must check compliance, cajole compliance, demand compliance. Test with tuberculin. Treat positive skin tests, but follow closely to avoid a drug reaction. The best way to counter those who would derail tuberculosis elimination by saying the drugs are too toxic is to monitor carefully. We must teach, motivate, and enjoin other physicians in the elimination strategy.

The public and its health officials also have failed. Many people think of tuberculosis as a disease of the past. The problem has not been perceived to be urgent and many people are not aware that the tools to accomplish the task are available. Others think of tuberculosis as a disease of the disadvantaged. Expressions such as “As long as there is poverty, there will be tuberculosis” are antithetic to this cause. We must publicize and influence the allocation of resources. We must show the public that the least action is the most costly one.

AIDS makes tuberculosis more prevalent and its treatment more difficult. Immigrant and migrant populations make tracking and contact follow-up harder. Alcoholism and homelessness frustrate all aspects of tuberculosis elimination. The psychiatrically ill and
flagrantly noncompliant patients can barely be treated if there is no place for them. The courts have to be enlisted in this crusade.

With broader commitment, the obstacles are resolvable. If we, whose tradition is in tuberculosis control, work on the eradication of tuberculosis as much as our predecessors did on the health care of patients with tuberculosis, we may not have to wait until 2010. When treating our patients, if we do not rest until every tubercle is dead, every tubercle may be dead before we finally rest.

This effort is only trying to do in the United States what other nations have already done. If Cuba could decrease tuberculosis more than sixfold by a national campaign, can we not do more? Why just the United States? Are we not the leader among nations? Then, why not the world?

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Renal Sarcoidosis; A Reminder

Although, sarcoidosis affects almost every organ in the body, kidney involvement is less frequent. The incidence of renal sarcoidosis varies from 7 to 22 percent.1-3 Renal manifestations of sarcoidosis can be broadly divided into the following groups:

Nephrocalcinosis and Nephrolithiasis due to Deranged Calcium Metabolism

Hypercalcemia is present in about 10 percent of patients with sarcoidosis, whereas hypercalcuria is about three times more common.4-7 These abnormalities of calcium metabolism are due to an increased ability of the sarcoid tissue to synthesize dihydroxyvitamin D which then leads to an enhanced absorption of calcium by gastrointestinal tract.6,9

Clinically, nephrocalcinosis occurs in about a third of the patients with hypercalcemia. Nephrocalcinosis in sarcoidosis is similar to that observed in primary hyperparathyroidism, vitamin D intoxication, and milk alkali syndrome. Renal calcifications, although predominant in the medulla, are also found in the interstitium, the tubules and surrounding area, and the distal nephron. Tubular atrophy, interstitial scarring, and glomerular hyalinization are the frequent complications. Renal failure is rare but when present it is due to a number of mechanisms that include a direct effect of hypercalcemia, dehydration, nephrocalcinosis, and tubulointerstitial disease.

Hypercalcemia in patients with sarcoidosis manifests in many ways that largely depend on the duration of the metabolic abnormality, the severity of nephrocalcinosis, and the coexistence of granulomatous involvement. It may be minimal and may present with polyuria with or without azotemia. There may also be a severe impairment of glomerular filtration rate due to the hypercalcemia that is reversible with appropriate therapy.10 In chronic sarcoidosis severe nephrocalcinosis may lead to progressive renal failure and hypertension. In a few patients, nephrolithiasis is the chief complaint causing recurrent episodes of hematuria, pyelonephritis, partial or complete hydronephrosis, and renal insufficiency.

Direct Granulomatous Involvement

The incidence of granulomatous infiltration of the kidneys is not known, but significant functional renal insufficiency is uncommon and overt renal failure is rare. An asymptomatic patient may escape recognition unless a kidney biopsy is performed. In one series, granulomas were found in 40 percent of renal biopsy specimens obtained in presumably selected cases of sarcoidosis.8 At autopsy 7 to 27 percent of patients with sarcoidosis were found to have renal granulomata.11 Clinically, these patients may have sterile pyuria, proteinuria, impaired acidification and concentrating ability, and renal insufficiency.

The granulomas are found principally in the interstitium. They are usually discrete, well localized, and composed of epithelioid cells. Lymphocytes and plasma cells are often scattered throughout the renal parenchyma. Occasionally, asteroid and Schaumann bodies are also present. Glomerulosclerosis, tubular atrophy, interstitial fibrosis, and vascular changes may be present. The latter include subintimal hyaline deposition, and intimal thickening of arterioles and arteries. In the absence of granulomatous involvement, immunofluorescence may reveal IgM and IgA deposits in the blood vessels.

Glomerulonephritis

The glomerular lesions that occur in sarcoidosis include membranous glomerulonephropathy, endocapillary proliferative glomerulonephritis, and crescentic proliferative glomerulonephritis.12 Prominent glomerular hyalinosis is common; complete hyalinization of the glomeruli may also occur. Rarely, renal amyloidosis has been observed.13