and often supported by NIH training grants, cannot reduce the trainee time devoted to research training. For other programs, the issue will be funding of the fourth year.

If a reduction of pulmonary-critical care medicine training positions is mandated by new workforce analyses and the reality of funding, how will they be distributed? I do not support uniform reductions of all medical subspecialty programs, nor uniform reductions within subspecialties. It has been proposed that a national workforce commission assign training slots based on prior training records, academic strength, and academic performance of the trainees of the various programs, as well as on geographic needs. A political solution to the workforce issue seems a bit draconian and is not supported by leading academicians. The quality of the educational and scientific training should be the primary reason for program retention. Centers of academic excellence should be established, where both the best clinical and scientific training can be obtained. When acceptable to both parties, community programs should consider affiliating with academic medical centers. The academic centers must be receptive to these affiliations and capitalize on the strengths of community programs. The character of the community program must be maintained. When it is feasible due to geographic proximity, academic medical centers should consider merging and thereby strengthening their subspecialty programs.

If there really is an increased need for generalists, can pulmonary-critical care medicine specialists pick up the slack, particularly in underserved areas? I suggest that the pulmonary-critical care division directors in concert with department heads establish programs whose purpose is primarily subspecialty training but with a component of continued generalist training in the form of outpatient clinics, etc. This would be ideal for those pulmonary-critical care medicine trainees who wish to enter the private sector or for those who prefer to be clinician-teachers. The issue of retraining established practicing pulmonary-critical care physicians is a viable one for our specialty and one which has been addressed by others.

The United States must maintain its role as the world leader in medicine. Academic medical institutions must remain the focal points for education and research training. The biotechnology revolution must not stop, and similarly, the quality and access to medical care afforded to our population cannot diminish. I hope the day never comes when we are compelled to send our trainees abroad for advanced research training or American citizens feel compelled to seek medical care overseas. Regardless of what workforce changes are dictated after more specific specialty-directed workforce analyses, it will fall upon the federal government as well as the public recipients of healthcare dollars to support graduate medical education and continued scientific investigation.

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Myocardial Sarcoidosis

A Wolf in Sheep’s Clothing

Pulmonary sarcoidosis is relatively easy to diagnose, follows mostly a benign and predictable course, and when treated responds satisfactorily to treatment with corticosteroids and other anti-inflammatory drugs. Myocardial sarcoidosis, however, is difficult to diagnose, follows a treacherous course that may lead to death, and responds poorly and randomly to treatment. The occurrence of myocardial sarcoidosis is generally accepted, but its true incidence is not known, and the accompanying grave consequences are not fully appreciated.

How Common Is Myocardial Sarcoidosis?

In 1929, Bernstein and associates first described sarcoidosis of the heart. More than four decades had
slipped by before Gozo et al\(^2\) gave us the clinical and pathologic features of cardiac sarcoidosis. Hagemann and Wurm\(^3\) estimated that 5 percent of the patients with chronic sarcoidosis had cardiac involvement. The incidence, however, tends to be higher in autopsy studies. In an autopsy review from Johns Hopkins Hospital, Baltimore, myocardial granulomas occurred in 27 percent of the patients. In 37 percent of the patients with myocardial disease, there were no clinical signs or symptoms.\(^4\) Cardiac involvement was a feature in 24 (19.5 percent) of 123 patients with sarcoidosis who had autopsy at LAC-USC Medical Center, Los Angeles.\(^5\) The literature now contains more than 600 references to myocardial sarcoidosis.

**Who Gets Myocardial Sarcoidosis?**

Iwai et al\(^6\) compared autopsy data from the Mayo Clinic in Rochester, LAC-USC Medical Center in Los Angeles, and the Japanese Society of Pathology in Japan. The frequency of cardiac sarcoidosis in Japanese was significantly higher than that seen in whites in Rochester or individuals of African-American origin in Los Angeles. Curiously, pulmonary sarcoidosis was the major cause of sarcoid death in patients of African-American heritage, whereas Japanese patients had a higher rate of mortality due to myocardial sarcoidosis. Elderly Japanese women suffered more from cardiac sarcoidosis than men.\(^5\)

**How to Diagnose Sarcoidosis of the Heart?**

Clinical evidence of myocardial involvement is present in only 5 percent of the patients with sarcoidosis. Cardiac involvement may precede or follow the disease or occur at any time during its course. The symptoms and signs are varied and include benign arrhythmias, heart block, intractable heart failure, intense chest pain, and sudden death. If cardiac manifestations occur in a patient with multisystem disease, the diagnosis, although circumstantial, is relatively clear. However, when cardiac dysfunction is the sole manifestation of sarcoidosis, the diagnosis is often not entertained, and even if the diagnosis is contemplated, it is usually not confirmed because of the unavailability of specific diagnostic tests.

Electrocardiogram may be normal or reflect every degree of heart block and every form of arrhythmia along with nonspecific ST-T changes.\(^6\) In this issue of Chest (see page 1021), Suzuki et al emphasize the usefulness of 24-h Holter monitor in identifying cardiac involvement in patients with sarcoidosis with or without myocardial symptoms and signs. Thirty-eight consecutive biopsy specimen-proved cases of sarcoidosis, 12 with known cardiac sarcoidosis and 28 with no evidence of myocardial sarcoidosis, had 24-h Holter monitoring using a dual-channel recorder with two bipolar monitor leads. The patients also received M-mode echocardiographic and thallium 201 myocardial single photon emission computed tomographic imaging. Holter monitoring revealed \(>10\) supraventricular ectopic beats (SVEB) in 8 (66.7 percent) of 12 patients with cardiac sarcoidosis and in 7 (26.9 percent) of 26 patients with no clinical evidence of myocardial disease. SVEB also occurred in 16 of 58 normal controls. Ventricular ectopic beats (VEB) of greater than 100 beats/day were more common in patients with myocardial sarcoidosis (\(p<0.01\)).

This study sends a clear message that every patient with sarcoidosis during the initial workup should receive a 24-h Holter monitoring. Any ECG or Holter abnormality should be further investigated by a two-dimensional (2-D) echocardiography that can reveal abnormalities due to fibrosis and granulomatous reaction.\(^7\) Since sequential contraction may be missed on a 2-D echocardiography, the next step is to perform a thallium 201 imaging. The fibrogranulomatous involvement of the myocardium results in segmental areas of decreased thallium 201 uptake. Unfortunately, focal thallium perfusion defects at rest are not specific to sarcoidosis and may occur with myocardial ischemia due to coronary artery diseases or other cardiomyopathies. During exercise, the perfusion defect seen on thallium scanning may disappear or decrease in size. This feature, called "reverse distribution," helps to differentiate the granulomatous nature of the injury from the one caused by the coronary artery disease.\(^8\) However, a coronary angiogram is often needed. In the presence of normal coronary arteries, the perfusion defects on thallium 201 imaging in a patient with known systemic sarcoidosis strongly suggest cardiac involvement. The combination of thallium 201 and gallium 67 scanning has been reported to have provided more information, but additional studies are needed.\(^9\) Gated cardiac magnetic resonance imaging is promising and needs further investigation.\(^10\) It is desirable to have an early myocardial biopsy, but because of the inhomogeneous distribution of the granulomatory process, normal results of a biopsy will not exclude cardiac sarcoidosis.\(^11\)

Corticosteroids benefit myocardial sarcoidosis, resulting in clinical improvement and regression of lesions. The dose should be high, 80 mg of prednisone daily or higher, and continued as long as it is necessary and can be safely tolerated. In intractable cases and in individuals with intolerable side effects due to corticosteroid therapy, chloroquine and methotrexate should be added.\(^12,13\) A permanent endocardial pacemaker should be used early in patients with unstable or complete heart block. Corticosteroids are needed in patients who have pacemakers. Cardiac
transplant remains a possibility for younger patients with severe, intractable heart failure.\textsuperscript{15} However, transplantation can be avoided by diagnosing the disease early and treating with corticosteroids.\textsuperscript{15}

To deal successfully with the menace of myocardial sarcoidosis, one must first learn to think of the entity not only in patients with multisystem sarcoidosis but in any patient with a complex and undiagnosed arrhythmia, conduction disease, myocardioopathy, or unexplained congestive heart failure in a young or middle-aged person.\textsuperscript{16} Once the presence of the wolf is suspected, further diagnostic studies should be aggressively pursued to establish the extent and severity of the illness. Finally, the beast needs to be tamed with high-dose corticosteroids and immunosuppressive drugs. Surgical intervention and cardiac transplantation are the last resort.

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\textbf{Chemotherapy and Survival in Non-Small Cell Lung Cancer: Three Years Later

Locally advanced or metastatic non-small cell lung cancer (NSCLC) is, notoriously, a therapeutic dilemma to solve. Although radiotherapy has a palliative effect and cytotoxic combination chemotherapy produces a definite number of objective responses, their overall clinical value remains uncertain.\textsuperscript{1} A controversy, in particular, already strong in the early 1990s,\textsuperscript{2,4} is still topical today.\textsuperscript{5-7} It pertains to the suitability of encouraging widespread use of chemotherapy at the community hospital level.

Three years ago, arguments against such use were essentially of two types: one was basically scientific, the other rather moral and philosophical. The first argument, moved in the most cautious commentaries,\textsuperscript{2,3} was that the scientific evidence suggesting a beneficial effect of chemotherapy on survival was scarce or incomplete. Is such an objection still valid?

By the end of 1991, at least ten trials of chemotherapy vs supportive care alone had been peer-reviewed and published.\textsuperscript{8,17} These studies are quite inhomogeneous as to years of publication, drugs and regimens used, planned duration of chemotherapy, and characteristics of the populations studied. For example, one study was limited to patients with metastasis,\textsuperscript{14} whereas in other studies, locally advanced disease (stage IIIb) was usually included and consisted of varying proportions of the sample.\textsuperscript{8,13,15,17} Only one element, ie, the prolongation of the median survival recorded after chemotherapy, was constant in each of the studies. One of them, the Canadian trial by Rapp et al,\textsuperscript{12} showed significantly improved survivals after two different programs of chemotherapy; the study was large enough, used active drugs (cisplatin, vindesine), and had a study design good enough to be convincing. Three older studies, dating back to the 1960s and early 1970s, had shown a similar significant difference,\textsuperscript{6} or suggested a trend toward a prolongation of survival in the group of patients treated with chemotherapy.\textsuperscript{9,10} However, minimally active drugs (alkylating agents, methotrexate) and only single-agent chemotherapy regimens were used in these studies. Two other trials showed statistically significant survival differences favoring chemotherapy with MACC (methotrexate,