To the Editor:

We appreciate the comments of Hiyama and colleagues on our article in CHEST (August 2000). They stated that the percentage of crypto
genic hemoptysis (42%) was particularly high in our study compared with other previously published studies and with their own results (12%). The distribution of causes of hemoptysis differed greatly in different studies, depending on geographic setting, time, and design of the study, and patient inclusion (or exclusion) criteria. Hiyama and colleagues studied a clinical series of 51 patients hospitalized for hemoptysis during a 6-year period, with detailed information and prospective follow-up. In contrast, we performed a retrospective epidemiologic study, covering a broad population (6,349 subjects who had, during a 3-year period, received a discharge diagnosis of spontaneous hemoptysis, with less detailed information collected from a large electronic discharge data file. This also explains why we had no information on smoking history or on the amount of expectorated blood. Furthermore, in our study, patients were not selected, and our data file included all kinds of patients, regardless of their age, the completeness of the etiologic investigation, and the amount and/or duration of bleeding. It is possible that a small number of the cases could have been erroneously classified as cryptogenic hemoptysis, but it is noteworthy that, in the study by Adelman et al., cryptogenic hemoptysis was identified in approximately 30% of all patients.

In the second part of their letter, Hiyama and colleagues commented about smoking, but they did not specify whether they considered tobacco an etiologic factor or a triggering factor of cryptogenic hemoptysis. Indeed, as with coronary atherosclerosis, tobacco use could, with a long latent period, lead to the creation of an underlying lesion, or it could transform this underlying lesion to clinically detectable bleeding. Due to the small number of patients with cryptogenic hemoptysis (only six patients), any conclusions about the etiologic factors should be drawn very cautiously. Further, among the 51 hospitalized patients described by Hiyama and colleagues, the percentage of patients with hemoptysis who smoked was low, and the status of smokers (history of smoking or current smoking) was not precisely specified. So it is possible that some of the nonsmoking patients may have stopped smoking in reaction to an underlying lesion, or it could transform this underlying lesion to clinically detectable bleeding. Due to the small number of patients with cryptogenic hemoptysis (only six patients), any conclusions about the etiologic factors should be drawn very cautiously. Furthermore, among the 51 hospitalized patients described by Hiyama and colleagues, the percentage of patients with hemoptysis who smoked was low, and the status of smokers (history of smoking or current smoking) was not precisely specified. So it is possible that some of the nonsmoking patients may have stopped smoking in reaction to an underlying lesion, or it could transform this underlying lesion to clinically detectable bleeding.

Forcing residents into different subspecialty training programs to amass, they cannot possibly how great their intuition, and no matter how much data or how much periodic evaluations they can amass, they cannot possibly work, for reasons that have been recognized for many years. Nonetheless, the elite group of planners, no matter how great their intuition, and no matter how much data or how many periodic evaluations they can amass, they cannot possibly substitute successfully for the function of the free market. Forcing residents into different subspecialty training programs to ensure their future ability to “obtain meaningful and gainful” employment is doomed to failure. Workforce and trainee planning should be abandoned immediately as an idea that is outdated, unworkable, and out of touch with reality.

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To the Editor:

I appreciate the letter of Drs. Arnett and Orient regarding the COMPACCS, Committee on Manpower of Pulmonary and Critical Care Societies.1 I enthusiastically agree that it would be a mistake, indeed it would be impossible, to “force residents into different subspecialty training programs. . . . ” That was never the intent or goal of manpower planning nor the point of my editorial. It is absolutely clear to all practicing pulmonologists and intensivists that, right now, there are more jobs than trained physicians to fill those spots. I believe that the primary-care training initiatives that were mandated several years ago are partially responsible for our current subspecialty shortage. Those far-reaching initiatives were based on decisions made essentially with no hard data, then “written in stone,” with very little confirmatory or refutatory data collected.

In order to not repeat that scenario, we must make manpower decisions based on data and then, I believe, recheck our data and therefore our assumptions. We would never administer warfarin without checking the prothrombin-time frequently. Why would we obtain a snapshot picture of physician manpower needs and never recheck the data in the future?

I disagree completely with the authors regarding the value of workforce assessment. As the COMPACCS article has shown, it is possible to obtain a detailed, methodologically rigorous analysis of manpower needs that, best of all, is completely in touch with our current reality.

Periodic workforce assessment should be a part of our response to the dilemma of increased physician demand, which is occurring earlier and more rapidly than even the COMPACCS paper suggested. There is no question that we need to develop strategies to meet these demands, not only now, but more importantly, in the not-too-distant future, when the baby-boomer generation will need more critical care services. Increased numbers of clinical trainees, new strategies of “virtual” intensive care, and evaluation of alternative physician providers in the ICU should be considered and evaluated to solve this important problem.

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A Diagnostic Dilemma of Syncope

To the Editor:

We read with interest the article in CHEST by Zayd Eldadah et al (June 2000),1 illustrating the first known case report of a patient with pulmonary embolism presenting as syncope, due to high-grade atrioventricular node dysfunction. We describe a similar case of a previously healthy 76-year-old man who presented with syncope and transient sinus node dysfunction secondary to chronic bilateral thromboemboli of the main pulmonary arteries.

A 76-year-old man presented with a witnessed syncopal episode of 2-min duration. There were no symptoms suggestive of a seizure. On transport to the hospital, marked bradycardia with sinus pauses were noted by the paramedics on the telemetric monitor. On arrival, the patient was afebrile, normotensive, with a normal heart rate. The cardiorespiratory examination revealed findings compatible with pulmonary hypertension. There was no calf or thigh swelling or tenderness.

The cell blood count, electrolytes, coagulation parameters, urinalysis, and chest radiography were noncontributory. The ECG demonstrated first-degree atrioventricular block with no evidence of ischemia. As the possibility of a cardiac etiology for syncope was entertained, the patient was admitted to the hospital for continuous telemetry and serial cardiac enzymes, the results of both of which were negative. The patient underwent two-dimensional echocardiography that confirmed moderate-to-severe tricuspid regurgitation, with an estimated pulmonary systolic pressure of 90 to 99 mm Hg. Bilateral compression ultrasound of the legs revealed no evidence of deep venous thrombosis. The patient subsequently underwent an infused spiral CT of the chest, which demonstrated bilateral chronic pulmonary emboli in the main pulmonary arteries with acute pulmonary embolism in the left segment (Fig 1). The patient was later identified as heterozygous for the factor V Leiden mutation. The patient received anticoagulation with unfractionated heparin, and warfarin therapy was initiated for life.

Our case is notable for transient sinus node dysfunction as a cause of syncope in the setting of acute pulmonary embolism on chronic thromboembolic disease, similar to the Bezold-Jarisch vasodepressor reflex described by Eldadah et al.1 The patient’s complete lack of symptoms for either acute or chronic pulmonary embolism, aside from syncope, is unique. Although pulmonary thromboendarterectomy offers patients with chronic pulmonary hypertension an improvement in their functional status, the utility of this option in our patient with a single episode of syncope is unknown.

Both of these cases remind us that one should entertain the diagnosis of pulmonary embolism, acute and chronic, in any patient presenting with syncope.

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Figure 1. Infused spiral CT of the chest demonstrating an acute on chronic pulmonary embolus in the left main pulmonary artery (arrow).

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