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-sweeping 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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Reference

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 afibrile, 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 ultrason sound 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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Nicotine Reduction and Bupropion

To the Editor:

In a recent article in CHEST, Dale and colleagues (May 2001) reported on factors that are predictive of successful smoking cessation and noted that the first 2 weeks of treatment were the “most crucial.” We also noticed this time period and found no significant immediate, 3-month, or 6-month reduction or quit rate differences between 1 month and 2 months of use of bupropion SR, 300 mg/d. The results of the study of smoking cessation by Dale et al., as well as the work of others in antidepressant therapy, suggest very limited differences between bupropion SR dosing schedules of 150 and 300 mg/d.

We recently completed a treatment program evaluation to compare the relative effectiveness of 150 mg/d vs 300 mg/d bupropion SR as part of nicotine reduction therapy with older American veterans. Seventy-three patients completed all project requirements and were the focus of this study. Treatment condition assignment (150-mg group, 54 patients; 300-mg group, 39 patients) was random, with patients in the 150-mg group having the option to receive 1 month of bupropion therapy at 300 mg daily (our standard dosage) after the 2-month follow-up. All patients signed consent forms and received the same behavioral treatment during the first session. Patients returned after 2.5 weeks to discuss their progress and difficulties, to receive more behavioral change information, and to receive bupropion SR. Subjects were followed-up by mail 2 months after the end of the second session.

The typical veteran was a white male, aged 57 years. There were no significant differences between groups regarding demographic factors, treatment motivation variables, or health habits. About 26% of patients reported quitting all nicotine use at follow-up, and 80% of those still using tobacco claimed nicotine reduction. Neither the group that quit (χ² test, 1.35; p = 0.25) showed significant differences; however, side effect profiles tended to be different. In response to a yes-no question concerning the presence of side effects, a higher percentage of patients in the 300-mg group complained of side effects than did patient in the 150-mg group (χ² test, 3.37; p = 0.06), with the total number of side effects averaging 2.6 per patient in the 300-mg group vs 1.2 per patient in the 150-mg group (t test, 1.92; p = 0.06). Weight change did not vary significantly between groups.

Larger studies with fewer methodological limitations are needed. However, this program evaluation indicates that 1 month of bupropion SR therapy at 150 mg/d may provide the maximum benefit for minimal expense and risk.

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Significant Omission in Antithrombotic Supplement

To the Editor:

In reviews of the treatment of venous thromboembolism, Hyers and colleagues and the researchers in the 263 articles that they referenced neglected to mention a randomized trial by Nielsen et al. comparing heparin and a phenprocoumon anticoagulation therapy with phenylbutazone therapy in patients with deep venous thrombosis (DVT). This is the only published randomized controlled trial comparing the treatment of DVT patients using standard anticoagulation therapy with control subjects who had not received anticoagulation therapy. It was a negative study with 1 of 45 anticoagulated patient dying of pulmonary emboli (PEs) and 0 of 42 patients receiving phenylbutazone therapy experiencing fatal PEs.

In the 1995 review, Hyers et al. state, “Patients with DVT or PE should be treated with IV heparin or adjusted-dose subcutaneous heparin sufficient to prolong the activated partial thromboplastin time to a range that corresponds to a plasma heparin level of 0.2 to 0.4 U/mL. This grade A recommendation is based on level 1 studies in patients with PE and DVT.

The placebo-controlled randomized trial by Barritt and Jordan of patients with PE is too old, small (n = 35), and flawed to be considered as proof of the efficacy of anticoagulant therapy. Barritt and Jordan made the diagnosis based on clinical suspicion. We now know that only about 27% of patients suspected of having PEs actually have the diagnosis confirmed by angiogram or lung scan. Of the five deaths in the control group, cases 1 and 4 had chronic septic cavitation of the lungs, with no recent embolization. The patient in case 2 had thrombophlebitis and thrombosis of the hepatic veins but no documented autopsy evidence of PE. In the patient in case 5, the cause of death was cerebral infarction. Consequently, only case 3 would meet a modern definition of fatal PE: “massive fresh emboli present in the main pulmonary artery, or in at least two lobar arteries, demonstrated post mortem in patients in whom no other cause of death was found.”

None of the other randomized trials cited in the later reviews concerning DVT or PE had control subjects who were not anticoagulated. Therefore, they do not provide “level 1” evidence of the efficacy of anticoagulant therapy in patients with PEs or DVT.

Heparin and vitamin K antagonists became the standard treatment for DVT and PE patients in the 1940s before the...