intensive care units. By and large, the respondents who offered additional comments believed that critical care should be an inclusive rather than an exclusive discipline, one open to qualified practitioners from many fields.

This stance is in keeping with that taken by the specialty boards in anesthesiology, medicine, pediatrics, surgery, and neurosurgery that are designing their own critical care certification procedures. For example, the American Board of Internal Medicine apparently does not desire to create a new specialty or subspecialty in critical care. Nevertheless, it does want to recognize the special qualifications of certain internists who have significant training or experience in critical care and can demonstrate their knowledge on a written examination, the first of which is scheduled for November, 1987. Because some diplomates of the Board have little formal training in critical care, the first three examinations will be open to certified internists with at least four years of commitment (involvement of at least 50 percent of their practice) in critical care and to certified pulmonologists, cardiologists, and other subspecialists with at least two years of similar commitment. Future examinations (in addition to the first three) will be open to diplomates who are also certified in a medical subspecialty and have completed a one-year fellowship in critical care medicine, diplomates who have completed a two-year critical care fellowship, and diplomates who have completed a two-year fellowship in general internal medicine which includes six months of critical care training along with another one-year fellowship in critical care. The American Board of Internal Medicine will not examine physicians who are not internists; information about the examination for anesthesiologists, surgeons, and pediatricians are available from their own boards. At present the American Board of Anesthesiology is scheduling an examination for the fall of 1986.

The American Board of Internal Medicine stresses that participation in its examination is entirely voluntary, that certification in critical care medicine is not a requirement to practice critical care, and that certification by the Board does not confer privilege to practice. Nevertheless, we suspect that in these competitive times, many internists will seek such certification, as will anesthesiologists, pediatricians, and surgeons through their own boards. The demographic information obtained by our questionnaire suggests that many members of the ACCP Council on Critical Care will qualify for the first examinations by virtue of their critical care experience. For fellows currently in a two-year subspecialty training program which includes critical care, one year of additional critical care training will be required. (Currently, the American Board of Internal Medicine is discussing whether a three-year training program with a year of disbursed critical care work will qualify.) It is not clear where this training will be obtained or who will pay for it, given the recent cutbacks in support for fellowships. It also is not apparent yet what the curriculum for critical care trainees will be.

Nevertheless, it is clear that our membership includes many practitioners and leaders in critical care medicine and that the ACCP must respond to their needs. We do not intend to take a stand on whether or not critical care certification is necessary; our Council members clearly are divided on this question, and certification soon will be a fait accompli regardless of how they feel. However, this editorial will serve to communicate the sentiments of our members to the American Board of Internal Medicine and other specialty organizations. Furthermore, we will continue to fulfill a major purpose of the College: postgraduate medical education. Critical care medicine will be the focus of the College's 52nd Annual Scientific Assembly in San Francisco, September 22-26, where a distinguished faculty will discuss issues such as nutrition, mechanical ventilation, ethics, and cost containment that were requested by the respondents to our questionnaire. Over the months ahead, the Critical Care Council will prepare publications on these and other topics also requested by our respondents. In 1987, the College will present two Critical Care Examination review courses under the leadership of D. Robert McCaffree, M.D., FCCP. The first will take place June 7-10, 1987, in San Diego at the Hotel Del Coronado. The second, an identical course, will be given at The Shoreham in Washington, D.C., September 14-17. These and other efforts reflect our belief that critical care is indeed here to stay.

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Slow Response to Treatment of Acute Bronchial Asthma

Initial therapy for hospitalized asthmatic patients most often includes intravenous aminophylline, intravenous corticosteroids, inhaled and oral beta agonist therapy, and supplemental oxygen. Pulmonary function status is monitored by repeated measurements of peak expiratory flow rate. Though the initial therapy may be identical, patients hospitalized for treatment of acute asthma respond to therapy at different rates.1-5 Patients with a rapid response achieve their maximum pulmonary function values within three days, whereas those with a slow response often
require more than seven days. At present there is no clear description of the recovery rates for a hospitalized asthma patient and only a few attempts have been made to predict the rate of response. As a result, the importance of understanding the individual patient's response to therapy and the need for predicting that response early in the course of hospitalization have been understated. The ability to identify the patient with a slow response and insight regarding the pattern of response to therapy help predict the length of hospitalization, an important consideration in this age of DRGs.

The criteria established by Benfield and Smith to provide a useful means for predicting who will be a slow responder: (1) age greater than 40 years; (2) lack of atopy; (3) duration of attack greater than seven days; (4) three admissions in the previous 12 months; (5) maintenance oral corticosteroids; and (6) pulsus paradoxus greater than 25 mm Hg. Furthermore, our previous data suggest a consistent recovery pattern for individuals as reflected by simple pulmonary function measurements. Recovery approaches baseline pulmonary function status in a nearly asymptotic manner.

However, it is important to note that, even in a “stable” patient, peak flow measurements vary considerably and it could take successive measurements over a three- to four-day period to establish a good estimate of baseline pulmonary function status. A patient with a rapid response to asthma therapy may manifest a complete recovery within this time period. Problems arise in the management of slow responders who may not be identified as such. A single simple measurement such as peak expiratory flow rate done on a twice daily basis during hospitalization and then on a weekly follow-up basis to document continued improvement are more helpful in the management of a slow responder than a full set of pulmonary function tests done less frequently.

With the current pressures for cost containment, there may be a tendency to discharge a patient prematurely from the hospital or emergency room before the patient has attained an acceptable peak expiratory flow rate. Caution needs to be exercised with the slowly responding patient. Such a patient may require a lengthy hospitalization or at least frequent outpatient monitoring to document an acceptable recovery. Furthermore, premature discharge can result in reexacerbation necessitating additional hospitalization. Hospital admissions that are characterized by a slow response in an adult asthmatic patient may require greater than two weeks of hospitalization to attain an acceptable level of recovery. The current DRG guidelines regarding hospitalization for acute asthma may be too restrictive for the slowly responding patient.

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Prevalence of Congestion in Chronic Heart Failure

The causes and manifestations of heart failure have elicited considerable discussion in the past two centuries. Largely through the influence of MacKenzie at the turn of the century, "heart failure" became an accepted clinicopathologic entity. Prior to his writings, practitioners and anatomists were mostly concerned about the anatomic factors causing the disease. The mechanics of the heart as a pump were largely ignored. The signs of heart failure were grouped together by Austin Flint (1870) as cardiac dropsy, which he described as occurring mainly in individuals with valvular lesions. Osler, too, attributed dilatation of the heart primarily to valvular lesions. The term, "congestive failure," was introduced by Hamilton in 1924 to replace terms like chronic myocardial insufficiency and congestive cardiac decompensation.

In 1936, Harrison, in an attempt to differentiate the various forms of circulatory failure, defined congestive heart failure as a "syndrome characterized by dyspnea, rales at the lung bases, venous distention, engorgement of the viscera and accumulation of fluid in the subcutaneous tissue and body cavities." He also appreciated that "backward failure" could occur in the absence of valvular insufficiency due to "diminished distensibility of the ventricular musculature." Paul Wood refined the definitions of heart failure based on the patients' symptoms and clinical findings. He separated left from right ventricular failure and also concluded that left-sided problems, as well as conditions affecting the heart as a whole, could result in congestive heart failure. Although several authors recognized that cardiac dysfunction could occur without