reports emanating from the Third ACCP Consensus Conference on Antithrombotic Therapy, which is a valuable reference issue. I was particularly interested in the article on antithrombotic therapy in atrial fibrillation and would like to know the authors' opinion on the role of warfarin in paroxysmal atrial fibrillation. Since the publication of the new recommendations on anticoagulation therapy for chronic atrial fibrillation, local physicians have been recommending warfarin in the treatment of paroxysmal atrial fibrillation. It seems to me that one cannot extrapolate the relevant studies to paroxysmal atrial fibrillation. If we do, are we not opening a can of worms?

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To the Editor:

The role of warfarin in preventing thromboembolic stroke in patients with chronic nonrheumatic atrial fibrillation is now well established. However, in patients with paroxysmal atrial fibrillation, this role is less clearly defined. Of the five randomized, controlled trials recently published, the Boston Area Anticoagulation Trial for Atrial Fibrillation1 and the Stroke Prevention in Atrial Fibrillation Trial2 included patients with both chronic and intermittent atrial fibrillation. Patients with transient, self-limited atrial fibrillation occurring after trauma or with an acute medical illness, such as pneumonia, were excluded from these trials. In the Boston trial, both chronic and intermittent nonrheumatic atrial fibrillation had similar risks of stroke. Furthermore, both studies showed a statistically significant benefit of warfarin therapy in reducing thromboembolic stroke, with a low incidence of major bleeding complications. Therefore, as Dr. Thuyagaraj stated, warfarin therapy may be implied to be beneficial in paroxysmal nonrheumatic atrial fibrillation.

The duration of atrial fibrillation and the presence of coinciding medical conditions influence the decision to anticoagulate. For example, patients with conditions that may preclude anticoagulation (eg, alcoholism, bleeding disorders, history of falls) are at higher risk from warfarin therapy. Conversely, patients with previous thromboembolism, cardiomyopathy, valvular heart disease, or prosthetic heart valves should receive anticoagulant therapy whether the atrial fibrillation is chronic or paroxysmal.

It is thought that thrombus forms within the left atrial appendage in patients who have atrial fibrillation for as short a time as 2 days. It is also known that the incidence of embolization is greater following electrical, pharmacologic, and spontaneous cardioversion in patients who are not receiving anticoagulant therapy than in those who are.14 It is postulated that restoration of normal atrial contractions may be the mechanical factor responsible for dislodging thrombus from the left atrial appendage. Since patients with "lone" atrial fibrillation are at low risk for thromboembolic stroke, one may question the need for anticoagulation in this group. However, the rates of survival and incidence of cerebrovascular accident were the same regardless of type (isolated, recurrent, or chronic) of lone atrial fibrillation.9 Therefore, if the atrial fibrillation is "lone" and of 48-h duration and requires cardioversion, we would still recommend anticoagulation.

Since low-intensity anticoagulation with warfarin is relatively safe, with a low incidence of major bleeding complications, it is our recommendation that patients with paroxysmal atrial fibrillation whose episodes persist for more than 2 days be anticoagulated with warfarin adjusted to an international normalized ratio between 2.0 and 3.0. Also, the anticoagulant should be given for 3 weeks before and continued 4 weeks after elective cardioversion.

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Percutaneous Lung Biopsy in the Diagnosis of Bronchiolitis Obliterans Organizing Pneumonia

To the Editor:

We read with interest the supplement to the July 1992 issue of Chest, containing the proceedings of the International Congress on Bronchiolitis Obliterans Organizing Pneumonia (BOOP), in which the diagnostic procedures for this disorder are discussed. We present the case of a patient with idiopathic BOOP, diagnosed by means of percutaneous lung biopsy performed with use of a Tru-cut biopsy kit (Baxter Healthcare Corp, McGaw Park, IL).

A 40-year-old woman presented with fever, cough, and yellowish sputum. She had not been exposed to toxic substances or drugs, nor were there clinical data indicating systemic disease. Examination revealed a temperature of 38°C and crepitant pulmonary rales. The hemoglobin level was 9.5 g/dl, and the erythrocyte sedimentation rate was 122 mm/h. Urinary sediment and liver and kidney laboratory results were normal. Chest radiography revealed condensation in the middle lobe. Microbiologic and immunologic studies were negative. Treatment with erythromycin produced no improvement. Radiography showed a decrease in the condensation in the middle lobe, but condensation appeared in the right upper and lower lobes. The results of bronchoscopy were normal, and a transbronchial biopsy specimen measuring 0.4 × 0.3 × 0.1 cm was not diagnostically relevant. Percutaneous Tru-cut biopsy was performed to obtain a 2.1 × 0.1-cm cylinder, the histologic study of which led to a diagnosis of BOOP. The patient was treated with prednisone, and rapid clinical and radiologic improvement followed.

In BOOP, the definitive diagnostic procedure is biopsy involving thoracotomy.14 It has recently been reported14 that, in cases of strong clinical suspicion, transbronchial biopsy indicating BOOP is diagnostic proof. We propose that when transbronchial biopsy is not conclusive, percutaneous Tru-cut biopsy may be a valid alternative, since frequently the disease is peripheral,26 and it is usually possible to obtain a larger specimen than that afforded by transbronchial biopsy.25 Percutaneous Tru-cut biopsy has been widely used to obtain samples of numerous organs. In the lung, it has shown a high diagnostic yield in both focal and diffuse pathologic processes, with low rates of morbidity and mortality when compared with thoracotomy.
We have found only one reference in the literature to Tru-cut lung biopsy in a case of BOOP.1

We consider that when the clinical situation suggests BOOP and transtracheal biopsy has not led to a definitive diagnosis, the performance of percutaneous Tru-cut biopsy may make thoracotomy unnecessary.

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Perivascular Fibrosis of Muscular Pulmonary Arteries in COPD

To the Editor:

The article by Andoh and colleagues,1 which appeared in the December 1992 issue of Chest, is a useful addition to the literature on cor pulmonale in COPD. Its contribution to the larger understanding of COPD would be considerably enhanced if some additional information could be provided.

What is the frequency in the investigators' population of chronic bronchitis patients similar to those reported? My own experience and the literature suggest that such cases are rare. It would be helpful to know the time taken to accumulate these six cases and the total number of cases of chronic bronchitis and emphysema studied over this time period.

Three of the six subjects with chronic bronchitis were nonsmokers. Did these subjects have other risk factors for COPD, such as a dusty occupation or onset of symptoms after acute viral illness? Were lifetime smoking histories similar in the three ex-smokers in the chronic bronchitis group and the six in the emphysema group?

The authors say that autopsy "confirmed the absence of emphysematous changes" in the chronic bronchitis cases. It would be helpful if some objective evidence of this were given, such as a semiquantitative estimate of the amount of emphysema observed in the inflation-fixed specimens or a mean linear intercept measurement on the histologic sections.

The investigators say that all subjects died of cor pulmonale and respiratory failure. However, no information is given from the autopsies about right ventricular anatomy. It would be helpful to know the ratio of right ventricular to left ventricular plus septal weight or, if that measure is not available, the heart weight and right and left ventricular wall thicknesses.

It is only from the information requested that one can make a judgment as to whether these cases are rare examples of "pure" chronic bronchitis with perivascular fibrosis and cor pulmonale or whether they fall into the group of "increased-marking emphysema." 2,3

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To the Editor:

In Japan, chronic bronchitis with severe obstruction and without emphysema is found in nonsmoking adults. This is a progressive lung disease with a poor prognosis and is frequently accompanied by Pseudomonas aeruginosa infection, showing diffuse linear and reticulonodular shadows on the chest radiograph. It has been called diffuse panbronchiolitis by Homma et al1 and has not been reported in white patients.

The clinical features resemble those found in adult patients with mild cystic fibrosis (CF), an inherited disorder in white persons but with minimal or little pancreas dysfunction and intermediate-range sweat chloride concentration.2 Although Sugiyama et al2 have implicated a genetic background for this disease, our results based on delta F508 mutation analysis of the CF gene indicate that the chronic bronchitis may represent a disease different from CF.3

Thus, in Japan, pure chronic bronchitis with severe obstruction and without pulmonary emphysema is very common. The pathologic findings of this type of chronic bronchitis are well described in many previous reports in English that show the absence of emphysematous changes in spite of severe obstructive impairment and right ventricular hypertrophy.1,3,7 As pointed out by Dr. Snider in his comment on our report, such pure chronic bronchitis is rare in the United States and European countries. However, this chronic bronchitis is a good clinical model for understanding the bronchial or airway lesions in COPD because of the absence of pulmonary emphysema. The obtained findings will also be useful for the management of COPD patients in the United States and European countries.

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