Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Use of Chi Square

To the Editor:

We believe that the article, “Intracranial Metastases in the Initial Staging of Bronchogenic Carcinoma,” by Mintz et al (Chest 1984; 86:951-53) is a valuable addition to our appreciation of computed tomography (CT) of the brain in the initial staging of bronchogenic carcinoma. We congratulate the authors on their observations.

Using tables, the authors show the effectiveness of the medical history, physical examination, and EEC relative to CT for preoperative evaluations. We would like to point out that the chi square values (corrected for continuity) calculated in Tables 2 and 4 are incorrect. The correct values should be 15.0 and 1.28, respectively. We are happy to see the relative efficacies with chi square values, but we emphasize the need to correct these values.

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Acknowledgments

To the Editor:

It was recently brought to my attention that a speech, “Pregnancy and Tuberculosis,” I presented at the American College of Chest Physicians (ACCP) Annual meeting and published in Chest 1984; 86:105-35 contained information and quotes from a paper by Weinberger et al (Am Rev Respir Dis 1980; 121:559) which were not acknowledged in the published manuscript. I want to apologize to Weinberger et al and to the editors and readers of Chest and American Review of Respiratory Disease for this oversight.

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Pulmonary Involvement in Sjögren’s Syndrome

To the Editor:

I reviewed with interest the paper by Constantopoulos and associates entitled, “Respiratory manifestations in primary Sjögren’s syndrome—a clinical, functional, and histologic study” (Chest 1985; 88:226-29.)

This study presents a limited series of 26 patients with primary Sjögren’s syndrome who were evaluated for pulmonary manifestations using clinical, functional, roentgenologic and histologic criteria. The conclusion was that 27 of 36 patients (75 percent) had evidence of pulmonary involvement. In eight of these patients, the only functional abnormality was a minor decrease in the MEF_50 on the flow volume curve, suggestive of small airways disease. An additional six patients had normal functional evaluation results but had a dry cough. Bronchoscopic examination of these patients found “dry mucosa of the tracheobronchial tree,” and the term “xerotrachea” was used to describe the combination of dry cough and dry respiratory mucosa. There appeared to be minimal changes of significance in these 14 patients.

We previously reported a much larger series of patients (Chest 1976; 70:354-61); specifically, 343 patients with classic Sjögren’s syndrome. Our series included patients with both primary and secondary Sjögren’s syndrome; however, the majority of our patients had primary Sjögren’s syndrome. We eliminated those patients who had no pulmonary symptom, or had normal chest roentgenographic or pulmonary function study results. These patients were virtually without evidence of pulmonary involvement. We found that only 9 percent, or 31 patients in our series, had clinically significant pulmonary disease associated with Sjögren’s syndrome. At that time we classified the pulmonary manifestations of Sjögren’s syndrome more frequently than previously reported. We feel that this is the true incidence of clinically significant pulmonary disease in Sjögren’s syndrome. The majority of our patients with primary Sjögren’s syndrome had minimal or no pulmonary symptoms.

When comparing the present study of Constantopoulos and associates to our much larger series of patients, it is our impression that the conclusion that pulmonary involvement is present in such a high percentage of patients with Sjögren’s syndrome is misleading. This disparity regarding the clinical incidence of pulmonary involvement in Sjögren’s syndrome should be discussed.

C. Vaughn Strimlan, M.D., F.C.C.P.
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To the Editor:

We wish to thank Dr. Strimlan for his very productive comments on our paper, "Respiratory manifestations in primary Sjögren's syndrome." He does not appear to disagree with our findings and the high rate of evidence of pulmonary involvement in primary Sjögren's syndrome. However, comparing them to the results of his paper of 1976,¹ the first significant work on pulmonary involvement in Sjögren's syndrome, he correctly points out that many of our patients "had minimal changes of significance" and that "clinically significant pulmonary disease associated with Sjögren's syndrome" must be approximately 9 percent. Therefore, Dr. Strimlan concludes that the high percentage of pulmonary involvement in our paper is misleading.

The question is, "What is clinically significant?" For instance, as we emphasize in our paper, many of our patients with dry cough (xerotrachea) as the only manifestation had been treated for years for chronic bronchitis, asthma or even tuberculosis before the diagnosis of Sjögren's syndrome was made. None of these patients had any functional impairment. Does this fact classify their problem as "clinically insignificant?" We think that pulmonary specialists should
know this entity and include Sjögren’s syndrome in their differential diagnoses of cough of undetermined origin.

One of our patients with transbronchial lung biopsy specimen showing lymphocytic infiltrates with follicular pattern is another example. She had normal pulmonary function, including DLCO. We included her in the diffuse interstitial disease group on the basis of this biopsy result, but if we only had pulmonary function study results she would have been considered normal. What is the clinical significance of these lymphocytic infiltrates? We consider them quite significant since they show involvement of pulmonary interstitium, and this fact alone classifies this patient as a case of the extraglandular form of Sjögren’s syndrome, with everything that accompanies it. Close follow-up would determine if she develops clinically and functionally obvious interstitial lung disease. So far, two years later, she has not. Could this suggest that the patchy lymphocytic infiltrates of Sjögren’s syndrome in the pulmonary interstitium do not interfere with diffusion, at least in early stages?

What is the significance of small airways involvement? Probably limited, we think, and thus the six flow/volume curves shown in our paper are only mildly deformed. Still, this involvement has been previously emphasized as indicating that “human airways should be considered another target organ of Sjögren’s syndrome” in small series of patients1 and we thought that we should evaluate its significance in a larger series. Lack of specificity of the tests for small airways is one of the reasons why it is better to evaluate patients with autoimmune rheumatic diseases in comparison with normal control subjects and not with predicted values alone.

Finally, we think that if we consider pulmonary disease “clinically significant” only in the presence of incapacitating symptoms or very heavily impaired pulmonary function, the percent of pulmonary involvement in Sjögren’s syndrome is probably lower than the 9 percent reported by Dr. Strimlan in 1976. In our report, though, we did not comment on whether the pulmonary involvement was clinically significant. We only wanted to define this involvement using as many parameters as possible exclusively in patients with primary Sjögren’s syndrome. Most studies do not make a clear distinction between primary and secondary Sjögren’s syndrome with the result that many respiratory manifestations attributed to Sjögren’s syndrome may, in fact, be the result of rheumatoid arthritis in patients with secondary Sjögren’s syndrome. For example, pleural effusion, mentioned in most pulmonary textbooks4 as a frequent manifestation of Sjögren’s syndrome, was absent in all 36 patients of our series. Our conclusions remain the same: pulmonary involvement is frequent in primary Sjögren’s syndrome and it extends from the pharynx and the trachea to the pulmonary interstitium. Whether it is of “clinical significance” remains to be elucidated with follow-up studies involving patients with primary Sjögren’s syndrome alone or in comparison with patients with other rheumatic disorders and healthy controls. Certainly, from our results we do not suggest that Sjögren’s syndrome produces the incapacitating pulmonary manifestations of progressive systemic sclerosis or the life-threatening complications of systemic lupus erythematosus.

S. H. Constantopoulos, M.D.,
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REFERENCES
4 Myers AR. Pulmonary manifestations of collagen-vascular dis-

Spacer Devices

To the Editor:

I appreciated Dr. König’s excellent review of spacer devices which appeared in Chest (1985; 88:277). I would like to make two additional points. I have seen several patients who, because of local oropharyngeal and laryngeal irritation, could never tolerate inhaled beclomethasone but are now able, with spacer devices, to tolerate it without difficulty. The second point is that, as Dr. König has shown so well, the studies are contradictory. This may be because all patients are not equal and some may respond better to spacer devices than others. Therefore, in the difficult-to-manage asthmatic patient, I strongly recommend a trial of a tube spacer device.

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To the Editor:

I agree with Dr. Stulbarg’s comments, but his strong recommendation of tube spacers for the difficult-to-manage asthmatic patient should consider other types of spacers like pear, cone or plastic reservoirs, which are equally good and possibly better than tube spacers.

Peter König, M.D., Ph.D.,
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University of Missouri, Columbia

Contraindications for Chest Physiotherapy

To the Editor:

The excellent review entitled “Does chest physical therapy work?” (Chest 1985; 88:436-44) by Kirilloff and colleagues was noted with great interest. This comprehensive and authoritative review goes far to debunk many of the myths surrounding this often overused modality.

Unfortunately, steadfast resistance is often encountered by those who attempt to limit the use of chest physiotherapy to only those patient groups in which it has been shown to be efficacious. When individual clinicians are approached to solicit their reasons for ordering chest percussion, they often express their belief that, even if chest percussion might not add to the therapy of their patients, at least it does not detract (except from the patients’ pocketbooks). No one, including patients, should be required to pay for needless therapy. Of course, with the advent of DRG’s, administrators are becoming much more vigilant in their effort to minimize or eliminate useless and costly activities. Furthermore, clinicians should not blithely assume that chest percussion might merely be neutral in its effects. As pointed out by Kirilloff et al, chest percussion and/or vibration has been shown to produce bronchoconstriction in chronic bronchitis patients.