patients with diffuse interstitial pulmonary disease shown by chest x-ray film, three patients had not only endobronchial disease, but also metastatic cancer of the breast involving the lymphatic vessels, as determined by transbronchial biopsy. As in the case report of DeBeer et al, exophytic masses were seen in two of our biopsy-proven cases, while the remaining five abnormal specimens from biopsy were taken from bronchial mucosa diffusely involved by nodular lesions.

With the new advances in the treatment of cancer of the breast, the clinical and diagnostic recognition of the multiple patterns of metastatic involvement of the lung becomes increasingly important. Our experience indicates that a systematic approach to the symptoms, x-ray films, and subsequent bronchoscopic evaluation of patients with cancer of the breast will identify endobronchial metastatic cancer of the breast as an increasingly common event.

To the Editor:

Tenholder et al astutely recognized and reviewed their relevant experience in patients with cancer of the breast who had endobronchial lesions, and these investigators identified seven biopsies showing malignant disease. The exophytic masses in two of their biopsy-proven cases seem to confirm our experience. We appreciate their review and agree with their conclusion that a systematic approach to symptoms, x-ray films, and subsequent bronchoscopic evaluation in this population of patients will identify metastatic endobronchial lesions with increasing frequency.

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To the Editor:

In their article entitled "Endobronchial Metastasis from Cancer of the Breast" (Chest 73:94-96, 1978), DeBeer and associates comment on the rarity of endobronchial metastasis from cancer of the breast. Indeed, this metastasis must be rare for carcinoma of the breast. Peribronchial metastasis on the ipsilateral side must not be nearly so rare. I have seen three such cases in my five years of practice in thoracic surgery. The metastases do not occur endobronchially, although they produce intense cough with a great deal of bronchial edema and inflammation. Such metastases are usually diagnosed by a high index of suspicion and aggressive techniques of biopsy at mediastinoscopic examination.

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Eosinophilia Caused by Rifampin

To the Editor:

Although therapy with rifampin (rifampicin) has been considered a possible cause of eosinophilia, this has never been definitely established, as the drug is usually given in combination with isoniazid, which itself may result in eosinophilia. The present case is reported because eosinophilia that developed following treatment with antituberculous drugs regressed on stopping therapy with rifampin, despite continuation of therapy with isoniazid and streptomycin.

CASE REPORT

A 62-year-old man was admitted to the hospital on Aug 9, 1977, with a four-year history of cough with expectoration of grey sputum and, over the previous six months, a loss of weight of about 9 lb (4 kg). He had been treated for pulmonary tuberculosis in 1967 and again in 1972. On both occasions the patient had defaulted from follow-up, and treatment had been limited to five months and two months, respectively.

On examination the patient was thin and unwell, with a regular pulse rate, blood pressure of 120/80 mm Hg, and scattered ronchi throughout the chest, but no other signs of note. The hemoglobin level was 17.0 gm/100 ml, and the white blood cell count (WBC) was 8,800/cu mm (eosinophil count, 200/cu mm). A smear of sputum was negative for acid-fast and alcohol-fast bacilli. The chest x-ray film showed a soft infiltrate in both upper zones, with multiple cavities on the right.

A diagnosis of pulmonary tuberculosis was made, and treatment commenced on Aug 22, 1977, with streptomycin (0.75 gm daily), rifampin (450 mg daily), and isoniazid (300 mg daily). On a subsequent culture of sputum, there was a scanty growth of Mycobacterium tuberculosis. On Sept 15, the WBC was 11,700/cu mm, with an eosinophil count of 4,800/cu mm. Therapy with rifampin was replaced on Sept 19 by administration of ethambutol (800 mg daily). Therapy with streptomycin and isoniazid was continued as before. Blood cell counts over the succeeding month were as follows: on Sept 26, WBC of 10,100/cu mm and eosinophil count of
2,300/cu mm; and on Oct 17, WBC of 8,600/cu mm and eosinophil count of 900/cu mm. Therapy with ethambutol, isoniazid, and streptomycin was continued throughout this period. A provocation test with rifampin was not thought to be justified. The patient's clinical condition improved considerably, and there was an accompanying improvement in the appearance of the chest x-ray film.

**DISCUSSION**

Although eosinophilia is described by Martindale as a side effect of therapy with rifampin, there have been no previous published case reports, and neither the British Committee on Safety of Medicines (data from Register of Adverse Reactions, Finsbury Square, London) nor the manufacturers have received written reports of this complication. According to the manufacturers, no causal relationship between therapy with rifampin and eosinophilia has previously been established. In the present case a causal relationship seems probable in view of the appearance of eosinophilia on commencing therapy with rifampin and the regression of eosinophilia on stopping therapy with the drug. It seems unlikely that administration of streptomycin or isoniazid could have been responsible, as therapy with both drugs was continued uninterrupted and without change in dosage.

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**REFERENCE**


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**The Management of Sick Sinus Syndrome in Children**

**The Use of Exercise Tolerance Testing and Standardized Valsalva’s Test**

To the Editor:

We recently studied a child with sick sinus syndrome of unknown etiology in whom exercise testing and standardized Valsalva's tests were utilized as part of the determination of the suitability of implantation of a pacemaker.

**CASE REPORT**

A 12-year-old girl had initially been seen at the age of 4 years with easy fatigability. An electrocardiogram had shown long sinus pauses with occasional junctional escape beats. When the patient was 6 years old, the sinus pauses were as long as 1.7 seconds, and a 3.0-second paroxysm of atrial flutter had been documented. At 8 years of age, she had a questionable syncopal episode. During the next four years the patient had experienced no episodes of fainting or dizziness, although periods of sinus arrest up to 3.0 seconds, overall heart rates of 45 beats per minute, and junctional escape rhythm were documented (Fig 1).

Cardiac catheterization in 1977 demonstrated no abnormality. Electrophysiologic studies revealed junctional rhythm with occasional conducted sinus beats. The P-A and His-ventricle (H-V) intervals were normal, but the atrio-His (A-H) interval was prolonged to 120 msec. Cessation of atrial pacing after two minutes at 152 beats per minute resulted in an asystolic interval of 3,000 msec, followed by a junctional escape rhythm. Similar pacing after infusion of atropine resulted in a recovery time of 1,150 seconds and a junctional escape rhythm. Administration of a single bolus of atropine increased the heart rate to 80 beats per minute.

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**Figure 1.** Electrocardiographic tracings (leads 1, 2, and 3) from 12-year-old girl with sick sinus syndrome. Long sinus pauses of up to 3.0 seconds with junctional escape, echo, and blocked atrial beats were noted. Overall heart rate was 45 beats per minute.

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