The study design, however, does not support the conclusion of the authors that "there was no evidence of tuberculosis transmission within the institutions." Their record review of 4,744 inmates showed that only 347 (7.3 percent) had documented repeat skin tests during incarceration. The extent to which this small percentage of inmates, some of whom had been released from prison between skin tests, represents the entire inmate population is not known. Because of the absence of data on serial tuberculin testing on 92.7 percent of the prison population, the authors' conclusion cannot be supported that inmates who stayed in the prison system did not experience skin test conversions. Of the 345 inmates with repeat skin tests, there were three identified converters, all of whom were identified after a break in incarceration and reimprisonment. Similarly, in the absence of serial testing of inmates it cannot be concluded, as the authors claim, that the readmitted inmates were infected while outside the prison.

I applaud the efforts of the authors to measure the cost and some of the benefits derived from a large admission screening program. However, what is also needed from future evaluations is a measure of the extent to which screening and case-finding activities are protecting inmates from tuberculosis infection and disease. Results of retests on a representative sample of inmates for whom the number of years spent in prison is known would partially address this need. Incarceration can provide an ideal setting for transmission of tuberculosis, and the lack of reported cases in a prison system does not preclude the existence of unrecognized and unreported disease.14 Infected prison inmates may be at higher risk of disease than nonincarcerated individuals because reported TB risk factors such as IV drug abuse, alcoholism, and HTLV-III/LAV infection have been shown in some studies to be disproportionately represented in prison populations.4

The prison population consists mainly of young adults, most of whom will return to the community. Inmates are a relatively accessible and potentially high-risk group. We need to assure that all feasible approaches are being taken to prevent this group from becoming (or perhaps continuing to be) a focus for unnecessary future tuberculosis morbidity and mortality. Thorough evaluation of existing programs will determine which approaches can most effectively achieve this purpose.

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Differences in EPAP and CPAP

To the Editor:

We read with great interest the recent study by Layon et al. (Chest 1986; 89: 517-21) in which the authors observed no difference between the effects of CPAP and EPAP in anesthetized dogs. As discussed by the authors, these findings are not in agreement with our findings1 that FRC with CPAP was higher than that with EPAP at both 5 and 10 cm H2O pressure in human subjects. Layon et al argue that we did not directly measure end-expiratory pressure, relying instead on the expiratory pressure valve to quantify expiratory pressure. If this were true, expiratory pressure during EPAP would have been spuriously high, possibly accounting for the differences in CPAP and EPAP we observed. However, as stated clearly in the methods section of our study, we measured airway pressures directly with appropriately calibrated pressure transducers. As shown in our study, end expiratory pressures between EPAP and CPAP were equivalent. Therefore, we do not think technical problems in our study account for the differences between the results of Layon et al and our findings. Although the explanation for the discrepancy between the findings of Layon et al, Gherini et al,2 and our results is not completely clear, there are significant methodologic differences among the studies. We proposed in our study that alterations in chest wall elastic recoil between EPAP and CPAP may have been responsible for differences between EPAP and CPAP. These alterations may not be manifested in anesthetized experimental animals. Therefore, while we agree that possible differences between EPAP and CPAP may be related to effects of conscious alterations in breathing, we cannot accept the hypothesis that they are related to technical problems in our study. Furthermore, since positive end-expiratory pressure during spontaneous breathing is usually applied in unanesthetized patients, the probability that CPAP more effectively increases FRC and PaO2 and results in less work of breathing than EPAP should be considered in choosing between these two modes of applying end-expiratory pressure.

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To the Editor:

We thank Quan et al for clarifying the methods by which they measured airway pressure in their study comparing CPAP and EPAP.1 We do appear to have misinterpreted their "Method" section and, for this, we are pleased to be corrected.

The disparate findings in our study (Chest 1986; 89:517-31) and those of Schlobohm and colleagues2 are due to several factors. First of all, there were differences in the subjects studied: Schlobohm and colleagues studied 18 intubated, spontaneously breathing, supine patients in the intensive care unit. Twelve of 18 were comatose and six of 18 had recently been weaned from mechanical ventilation. Three of Schlobohm's patients had a history of chronic obstructive pulmonary disease.1 We studied ten healthy mongrel dogs who were anesthetized and spontaneously breathing. Schlobohm and colleagues found a statistically significant difference in the functional residual capacity (FRC) at 5 and 10 ml of water CPAP and EPAP. Yet
they also noted appreciable variability of response, since the difference in response between the two modes of positive pressure was greater in the first group of ten supine patients (908 ml at 10 cm H$_2$O) than in their second group of eight individuals (253 ml at 10 cm H$_2$O) in Fowlers' position (head of bed elevated at least 2 R).

In our study, there were small differences in FRC when CPAP and EPAP were applied to our model at end expiratory pressures of 5 to 20 ml of water; these differences were not statistically significant. Most importantly, we agree with Schlobohm et al$^1$ that the chest wall and expiratory muscle tone provide the clue to all of the interstudy variability and discrepancies. Patients and animals alike tend to resist inflation pressure. They differ only in the degree of resistance and in the degree with which they oppose CPAP or EPAP. We tried to quantify the effect of expiratory muscle tone by comparing the volume increase during CPAP and EPAP with the relaxation volume. The volume increase with CPAP and EPAP, eg, at 10 cm H$_2$O, was 56 percent for both. Thus, expiratory muscle tone prevents inflation to the volume predicted by the elastic properties of the system. This, presumably, is also the case in patients. We observed and measured the same phenomenon in awake healthy adults (unpublished data).

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Eosinophilic Alveolitis?

To the Editor:

We agree with Davis and co-workers that there are therapeutic implications of eosinophilic bronchoalveolar lavage (BAL).$^1$ We recently evaluated a 69-year-old white woman with a nonproductive cough of four weeks' duration. She was a nonsmoker and took no medications. She had no history suggesting atopy, asthma or any hypersensitivity pneumonitis. Physical examination was remarkable for tachypnea at rest and bilateral diffuse rales. Cardiac examination results were unremarkable. Her CBC showed no eosinophilia. Arterial blood gas levels (FiO$_2$ 21 percent) showed PO$_2$ of 49 and PCO$_2$ of 25. Screening spirometry demonstrated FVC 1.05 (47 percent) and FEV$_1$.98 (50 percent). Chest roentgenographic film revealed bilateral lower lobe infiltrates (Fig 1).

After nondiagnostic sputum evaluation, she was empirically started on intravenous erythromycin therapy. Progressive respiratory failure requiring mechanical ventilatory support rapidly ensued (four days). Bronchoscopy with BAL and transtracheal biopsy (TBBx) was performed. This lung biopsy revealed a lymphocytic interstitial infiltrate without eosinophilia. Special stains were negative for infectious organisms, but BAL fluid demonstrated 30 percent eosinophils. She was treated with intravenous methylprednisolone and was extubated within 48 hrs. At three months, pulmonary function tests showed normal volumes and flows with a normal chest roentgenographic film.

Davis' did not comment on the course of steroid therapy in his two cases. Our patient was tapered over four months from an initial oral dose of prednisone 50 mg daily. Two months later she relapsed with identical roentgenographic and clinical findings. Bronchoscopy with BAL again showed eosinophilia (25 percent) in lavage fluid with the same interstitial histology on TBBx.

We cannot agree with Davis that eosinophilic alveolitis is not part of the spectrum of chronic eosinophilic pneumonias or pulmonary infiltrates with eosinophilia. With a subacute presentation, progression to respiratory failure, steroid-responsive infiltrates, and subsequent relapse, our case is entirely consistent with a chronic eosinophilic pneumonia.$^2$ Interesting is the discrepancy between our BAL and TBBx results in demonstrating eosinophilia. Other series have shown eosinophilic BAL fluid and lung tissue eosinophilia in chronic eosinophilic pneumonias.$^3$ A recent review of the applications of BAL suggests that eosinophilic fluid is distinctly unusual in other pulmonary disease. $^4$ However, the correlation between BAL and histologic interstitial cells is variable in other diseases and will likely show such variable correlation in eosinophilic syndromes.

The similarity of these three cases to previously identified syndromes does not seem to justify describing a new entity based on BAL.$^5$

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REFERENCES


To the Editor:

The authors wish to thank Drs. Whitlock and Tenholder for their interest in our article. They raise several important issues. First, could the cases we described represent chronic eosinophilic pneu-