subsequent postoperative right ventricular pressure tracings did not show the characteristic dip and plateau of pericardial constriction. For the third patient with right ventricular dysplasia, the diastolic pressure tracing was unremarkable except for the elevated pressures and the fourth patient was not catheterized.

The second suggestion that the early diastolic sound might represent an early right ventricular third heart sound (S₃) was considered in more detail. Completely ruling out this possibility with the data available was impossible, but the findings made it unlikely. The right ventricle is particularly difficult to assess noninvasively, but on the echogram in all four patients, the early diastolic sound occurred at or before the E point of tricuspid valve opening. Although the timing of a right ventricular third heart sound relative to tricuspid motion is unknown, we would expect it to occur with the same time relation as a left ventricular S₃ (ie, on the downstroke of the valve closure). On that basis, the sound occurred too early. Recording a right ventricular displacement curve rather than the apexcardiogram was considered, but this is an unreliable technique and was impractical when doing simultaneous echo recordings. We realized too late that a phonocardiogram recorded simultaneously with the intraventricular pressure tracing would have been helpful, but could not justify repeat invasive studies on that basis.

Overall, the sound occurred consistently coincident with the abnormal septal motion and we would therefore expect it to be found occasionally in other disease states, since the proposed mechanism should continue to apply.

Derek R. Boughner, M.D.
Professor, Department of Medicine; Cardiac Investigation Unit, University Hospital, London, Canada

Alveolitis in an Asymptomatic Pigeon Breeder

To the Editor:

Recently Costabel et al (Chest 1984; 85:514-18) studied T-lymphocytosis in bronchoalveolar lavage (BAL) fluid of patients with hypersensitivity pneumonitis (HP). This study revealed that lymphocytosis in BAL of HP is predominated by suppressor T-lymphocytes.

We studied BAL and peripheral blood of a pigeon breeder who had contact with pigeons for 18 years, without clinical symptoms of pigeon breeder’s lung. Total IgE in peripheral blood was 60 IU/ml (normal). Circulating serum precipitins to pigeon antigens were demonstrated. Pulmonary function tests and diffusing capacity (Dco) were normal. Bronchomucosal biopsies showed chronic inflammation with infiltration of predominantly plasma cells and lymphocytes. We applied fluorescein isothiocyanate (FITC) labelled monoclonal antibodies to study T-lymphocyte subsets in BAL and peripheral blood. Analysis of BAL fluid demonstrated 30 percent lymphocytes, 65 percent macrophages and 5 percent granulocytes. Immunologic characterization revealed that the BAL lymphocytes were T-lymphocytes with a helper/suppressor (H/S) ratio of 0.7. This H/S ratio is depressed as compared to patients with sarcoidosis and T-lymphocytosis in BAL. About 60 percent of the mononuclear cells in the peripheral blood were lymphocytes with a H/S ratio of 1.8, which is in the normal range. Although this patient had no clinical symptoms of pigeon breeder’s lung, our results demonstrated that an alveolitis was present.

Previous observations by others showed that in patients with antigen exposure but without clinical evidence of HP and without radiologic or lung function abnormalities, alveolitis can exist. Costabel et al clearly demonstrated that after cessation of antigen exposure signs of an alveolitis were still present in BAL fluid, even in otherwise normal patients in whom hypersensitivity pneumonitis alveolitis could be demonstrated. Our findings demonstrate alveolitis in an asymptomatic pigeon breeder. Patients with pigeon breeder’s lung and asymptomatic pigeon breeders both can demonstrate T-lymphocytosis with a depressed H/S ratio in BAL fluid.

Academic Hospital Dijkzigt/Erasmus University, Rotterdam, The Netherlands

REFERENCES
1 Solal-Celigny Ph, Laviolette M. Hébert J, Cornier Y. Immune reactions in the lungs of asymptomatic dairy farmers. Am Rev Respir Dis 1982; 126:964-67
2 Leatherman JW, Micheal AF, Kronenberg RS, Schwartz BS, Hoidal JR. Evaluation of cell-mediated immunity (CMI) in the lung by monoclonal antibodies in hypersensitivity pneumonitis (HP) and asymptomatic pigeon breeders (APB). Am Rev Respir Dis 1983; 127(suppl) 62

Prevention from Recurrence of Summer-Type Hypersensitivity Pneumonitis

To the Editor:

Recently, Kawai et al reported summer-type hypersensitivity pneumonitis as a unique disease in Japan (Chest 1984; 85:311-17). Although they did not discuss recurrences in detail, this is very important because this disease has a high recurrence rate during the following summer season, if untreated. We have been successful in preventing the recurrence of summer-type hypersensitivity pneumonitis through improving housing environment. We have examined 31 cases (15 men, 16 women) of summer-type hypersensitivity pneumonitis since 1974. All these patients became symptomatic during the summer with fever, dry cough and dyspnea. Chest x-ray films showed diffuse nodular opacities. Restrictive change on pulmonary function tests, arterial hypoxia and impairment of diffusing capacity were also noted. The diagnosis in 24 cases was confirmed histologically by biopsy. Anti-cryptococcal antibody was positive in all 14 cases examined.

As to environment, these patients reside in generally very humid climate, so we recommended renovation of their housing environments to decrease humidity in the house as much as possible and to obtain better ventilation and better exposure to the sun. Nineteen of the 31 patients cleaned their homes thoroughly without any renovation. Five of remaining 12 patients moved to other houses in different locations. The other seven renovated their houses, such as making a new window to have better ventilation and more exposure to the sun, or fixing inadequate and leaky water drainage in the kitchen and bath room. Recurrence rarely occurs in autumn, so we reexamined the patients during the following summer to look for recurrence.

The recurrence rate was as high as 68 percent—13 of 19 among those who did not renovate their homes. No recurrence was noted among 12 patients who had renovated their home (seven patients) or moved to new locations (five patients). The overall recurrence rates in our cases (41.9 percent) appears to approximate what Kawai et al have reported (35.9 percent), though they did not indicate the means of prevention.

Recurrences of summer-type hypersensitivity pneumonitis have been prevented effectively by improving housing environment,