Pulmonary function studies in bird breeder's lung

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Pulmonary function studies were performed at the time of diagnosis in 10 patients, aged between 11 and 51 years, with bird breeder's lung due to exposure of between 9 months and 42 years. The studies were repeated after a varying interval in nine of these patients, following removal from exposure. In initial tests, diffusing capacity was impaired in eight patients and the arterial oxygen tension at rest was below 80 mm Hg in seven. Five patients had a low vital capacity and three showed evidence of airway obstruction. One patient continued to breed pigeons, against medical advice, and his pulmonary function, repeated after nine months, showed further deterioration. The others with abnormal pulmonary function showed significant improvement on repeat studies. Pulmonary function studies appear to be helpful in the diagnosis, irrespective of radiographic changes, and in the follow-up of patients.

Since the description of three patients with acute pneumonitis following exposure to pigeons which they bred as a hobby (Reed, Sosman, and Barbee, 1965), there have been a number of reports of interstitial pulmonary diseases in non-atopic subjects which appear to have been due to hypersensitive reactions to the excreta of both pigeons and budgerigars, similar to those due to other inhaled organic dusts.

The clinical features are characterized by an acute or insidious onset of dyspnoea, tachypnoea, fever, cough, malaise, loss of weight, and crepitations at the lung bases. Radiographs may appear normal or show micronodular shadowing in the early stages, but continued exposure often leads to further mottling, diffuse reticulation, and fibrosis (Leading article, 1967). In the series of affected patients, specific precipitins have been demonstrated to antigens derived from both pigeon and budgerigar material (Reed et al., 1965; Hargreave, Peps, Longbottom, and Wraith, 1966; Stiehm, Reed, and Tooley, 1967; Boyd, Dick, Lorimer, and Moran, 1967), though the presence of serum precipitins in bird breeders without any pulmonary symptom is not uncommon (Hargreave et al., 1966; Maloney, 1967). In most patients, intracutaneous tests with extracts of serum or droppings give a late Arthus-type response with an occasional immediate reaction. Aerosol challenge with appropriate antigen has been used as a diagnostic aid in adults, but such provocative tests do not seem to be an essential procedure (Stiehm et al., 1967).

The purpose of the present study is to present data of some of the main lung function changes in bird breeder's lung and to demonstrate the effect of removal from the source of exposure on these changes.

MATERIALS AND METHODS

Ten patients with bird breeder's lung were studied. Details of their age, sex, exposure to birds, radiological features, serum precipitin, and skin tests are shown in Table I. All had typical clinical features and positive precipitin tests against avian antigens, and five out of nine patients had a positive delayed reaction to intracutaneous skin tests with extracts from feathers.

Pulmonary function studies were performed at the time of diagnosis in all patients and were repeated several times in all except one (whom we were not able to trace) following removal from exposure. Only the results of the first and the last tests are discussed here.

The vital capacity (V.C.), forced expiratory volume in 1 second (F.E.V._1), functional residual capacity (F.R.C.), total lung capacity (T.L.C.) and residual volume (R.V.) were determined, using the closed-circuit helium dilution method (Gilson and Hughes-Jones, 1949) in a twin spirometer with helium analyser (Godard Pulmotest). Predicted values were obtained from the standard nomograms (Miller, Johnson, and Wu, 1959; Goldman and Becklake, 1959). The ratio F.E.V._1/V.C. was calculated and values below 70% were regarded as abnormal. The nitrogen washout time was measured by breath to breath analysis with 100% oxygen breathing, recorded in a single channel recorder; expired nitrogen was analysed by the Nitromed Analysrer (Lundin, 1953).

The steady-state carbon monoxide diffusing capacity (D.L.CO) was estimated at rest and on exercise. The apparatus and method used were as described by
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TABLE I
CHARACTERISTICS AT TIME OF DIAGNOSIS

<table>
<thead>
<tr>
<th>No.</th>
<th>Age/ Sex</th>
<th>Bird</th>
<th>Duration of Contact</th>
<th>Duration of Symptoms</th>
<th>Chest Radiograph</th>
<th>Precipitin Test</th>
<th>Skin Test (delayed reaction)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pigeon</td>
<td>Budgerigar</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Serum Dropping Extract</td>
<td>Serum Dropping Extract</td>
</tr>
<tr>
<td>1</td>
<td>11 M</td>
<td>Budgerigar</td>
<td>18 mths</td>
<td>1 yr</td>
<td>Heavy vascular markings in both lower zones</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>2</td>
<td>30 M</td>
<td>Pigeon</td>
<td>5 yrs</td>
<td>18 mths</td>
<td>Miliary mottling all over</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>3</td>
<td>42 M</td>
<td>Pigeon</td>
<td>35 yrs</td>
<td>2 yrs</td>
<td>Normal</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>4</td>
<td>43 M</td>
<td>Budgerigar</td>
<td>5 yrs</td>
<td>1 yr</td>
<td>Normal</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>5</td>
<td>44 M</td>
<td>Pigeon</td>
<td>20 yrs</td>
<td>6 mths</td>
<td>Bilateral diffuse reticulo-nodular shadowing in lower zones</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>6</td>
<td>46 M</td>
<td>Budgerigar</td>
<td>2 yrs</td>
<td>18 mths</td>
<td>Normal</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>7</td>
<td>47 M</td>
<td>Budgerigar</td>
<td>15 yrs</td>
<td>6 mths</td>
<td>Bilateral diffuse reticulo-nodular shadowing in lower zones</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>8</td>
<td>51 M</td>
<td>Pigeon</td>
<td>42 yrs</td>
<td>5 yrs</td>
<td>Bilateral diffuse reticulo-nodular shadowing in lower and mid zones</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>9</td>
<td>13 F</td>
<td>Budgerigar</td>
<td>13 yrs</td>
<td>3 yrs</td>
<td>Minimal miliary mottling in mid and lower zones on both sides</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>10</td>
<td>37 F</td>
<td>Budgerigar</td>
<td>9 mths</td>
<td>6 mths</td>
<td>Normal</td>
<td>Negative</td>
<td>Positive</td>
</tr>
</tbody>
</table>

National Academy of Sciences *Handbook of Respiration* (1958)

**RESULTS**

Details of the pulmonary function tests are shown, with dates, in Table II and in the Figure. We found a higher degree of repeatability in the parameters measured.

**VENTILATORY FUNCTION AND STATIC LUNG VOLUME**

In initial tests the vital capacity was less than 80% of the predicted value in five patients. The F.E.V./V.C. ratio was below 70% in only three patients and the response to isoprenaline inhalation...
tion was slight. Five patients had a minute ventilation of more than 9 litres per minute.

Low values were found for the total lung capacity in two patients, for the residual volume in two patients, and for the functional residual capacity in four patients. In patient 6, the residual volume was raised. The R.V./T.L.C. ratio corresponded closely to the figures obtained for R.V. The nitrogen washout time, to detect any impairment of pulmonary gas mixing, was within normal range in all.

CARBON MONOXIDE DIFFUSING CAPACITY Eight of the 10 patients showed impairment of diffusing capacity at rest and on exercise. The results were normal in patient 3 and border-line in patient 6.

BLOOD GAS ESTIMATIONS These were performed in nine patients with arterialized capillary blood. In seven, Po2 was below 80 mm. Hg at rest. The Pco2 level was below normal in seven, and in none was it elevated. The blood pH was normal and the plasma bicarbonate slightly low in all patients.

REPEAT PULMONARY FUNCTION STUDIES (Table II) Nine patients were available for repeat pulmonary function studies following cessation of exposure to avian antigens. All had subjective improvement, except patient 8, whose symptoms were originally not severe and who continued to breed pigeons. This patient's pulmonary function, repeated after nine months, showed further deterioration. The others showed improvement in almost all of the previously abnormal parameters of pulmonary function, except patients 3 and 6 whose pulmonary functions were normal at the time of diagnosis. The transfer factor showed good correlation with the Dlco.

DISCUSSION

Bird breeder's lung exemplifies a large group of interstitial pulmonary diseases, occurring as a result of inhaled antigens. This develops after a period of exposure to pigeon or budgerigar, the interval varying from a few months to over 30 years (Hargreave et al., 1966), and has also been observed in the present series. Chest radiographs may be normal, as they were in four of our patients, whereas abnormalities in pulmonary function are more frequent in this condition. In our series, five patients showed demonstrable radiographic changes and eight patients had impairment of pulmonary function. Two patients with a typical history of symptoms developing repeatedly after exposure to birds had normal radiographs and pulmonary function. On removal
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from exposure there was no recurrence. Had they been examined at the height of their symptoms, it is likely that the pulmonary function tests would have been abnormal at that time.

Reported pulmonary function studies in bird breeder's lung have shown moderate to severe reduction in vital capacity, little or no airway obstruction, moderate reduction in diffusing capacity, arterial hypoxia, and small alterations in the lung volumes (Reed et al., 1965; Hargreave et al., 1966; Stiehm et al., 1967). These abnormalities are compatible with the pathological changes in the peripheral gas-exchanging tissue of the lungs where the main changes are interstitial inflammation and alveolar exudation, without significant involvement of the bronchial tree or blood vessels.

Our findings are similar to those previously reported. Half of the patients had a reduction of vital capacity while the total lung capacity was below the predicted value in only two patients. One, patient 6, had an abnormally high residual volume, though his total lung capacity, pulmonary gas mixing efficiency, and diffusion studies were within normal limits. On subsequent examination of the same patient, however, the residual volume was found to be near normal. The diffusing capacity was impaired in the majority of patients and was the most common abnormality found in our group.

Seven patients had low arterial oxygen tension at rest. This, we believe, is due to increased venous admixture, possibly caused by a reduction in the rate of gas transfer. As pulmonary capillary volume was not measured, alterations of the pulmonary vascular bed cannot be excluded.

Hyperventilation is a common feature of 'stiff lung' where this is due to alteration at the alveolar region. This has been reported in patients with diffuse fibrosing alveolitis and other interstitial pulmonary disorders. In our series, this pattern has been observed, and in the majority of the patients the Pco₂ remained low; the highest reading obtained was 42 mm. Hg.

Except for one patient of Stiehm et al. (1967), all cases reported previously by different authors had no evidence of airway obstruction, though wheezing was a feature in two patients in one series (Hargreave et al., 1966). In the same series, however, few of the patients showed a significant reduction of F.E.V₁ after inhalation tests. In our group, two patients had mild and one (patient 1) moderate airway obstruction. There was no evidence of airway obstruction on repeat pulmonary function testing in these patients. The airway obstruction was not reversed completely by isoprenaline inhalation. This finding, along with impaired diffusing capacity, contrasts with bronchial asthma, where usually airways obstruction is reversible and diffusing capacity is normal. Mild airway obstruction in some of our cases may be due to bronchial obstruction. Similar findings have been reported in some cases of farmer's lung (Williams, 1963).

All our patients were advised to dispose of their pigeons or budgerigars. One, patient 8, failed to do this and showed further deterioration of his pulmonary function during the continued exposure. All the other patients, in whom pulmonary function was abnormal during the initial testing, showed improvement on repeat testing following a lapse of varying periods. In those patients who showed initial impairment of diffusing capacity, the DLCO, although improved, remains low. This requires further study. Two patients, 5 and 10, were treated with corticosteroids for a period of 3–12 months and, although they did not show a greater improvement in lung function than other patients, subjective improvement was more rapid.

The changes in pulmonary function in bird breeder's lung are not specific, as similar changes are found in other diseases which involve the pulmonary alveolar tissue, such as farmer's lung, sarcoidosis, scleroderma, diffuse fibrosing alveolitis, and other interstitial pulmonary diseases. However, with pulmonary symptoms and a history of exposure to pigeon or budgerigar, pulmonary function studies appear to be helpful in the diagnosis and assessment of progress.

We wish to thank Dr. J. Miles Walker for patients 3 and 5, and Dr. J. D. Allan for patients 1 and 9; all the technicians of our respiratory laboratory for additional work and inconvenience; Mrs. L. Denison for secretarial help; and Professor J. Pepys, of the Brompton Hospital, for carrying out the precipitin tests.

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REFERENCES


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