Radiological distribution of pulmonary emphysema

Clinical and physiological features of patients with emphysema of upper or lower zones of lungs

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Martelli, N. A., Hutchison, D. C. S., and Barter, C. E. (1974). Thorax, 29, 81–89. Radiological distribution of pulmonary emphysema: clinical and physiological features of patients with emphysema of upper or lower zones of lungs. Pulmonary emphysema exists in two main pathological forms, centrilobular and panlobular (panacinar) emphysema, the lesions predominantly affecting the upper and lower zones of the lungs respectively. There is disagreement among authors as to the clinical and physiological differences between these two forms, and direct evidence of the pathological type is seldom available during life. Patients with emphysema can, however, be divided on radiological criteria into an 'upper zone' and a 'lower zone' group, and it can be argued that these groups relate respectively to the centrilobular and panlobular forms of the disease. The evidence is far from conclusive but it was thought that a comparison of the two radiological groups would be of value. Patients in whom there was no obvious zonal preponderance were not included in the study.

Fifty patients with definite radiological evidence of pulmonary emphysema have been studied, those with $\alpha_1$-antitrypsin deficiency being excluded. Thirty-one patients (62%) had emphysema which predominantly affected the upper zones of the lungs; the lower zones were the more severely affected in the remainder. Bullae were found in approximately equal proportions in each group. All the patients were, or had been, cigarette smokers. There was no significant difference between the mean ages of the two groups; only seven patients were free from exertional dyspnoea, all being in the upper zone group. Chronic bronchitis occurred with equal frequency in the two groups but started on average about 10 years earlier in those with lower zone disease; the latter patients had rather more severe airflow obstruction and more severe blood-gas abnormalities. The presence or absence of chronic bronchitis per se, however, did not appear to have any significant effect upon the common respiratory function tests. No data emerged from this study which suggested that there were differing aetiological factors in the two groups.

Pulmonary emphysema is generally considered to occur in two main pathological forms (Gough, 1952; McLean, 1956; Leopold and Gough, 1957) now commonly known as centrilobular emphysema and panlobular (or panacinar) emphysema; the former type is found mainly in the upper zones of the lungs and the latter in the lower zones, particularly in the advanced case (Thurlbeck, 1963; Wyatt, Fischer, and Sweet, 1964). A precise classification is, however, difficult in many cases (Thurlbeck et al., 1969; Mitchell et al., 1970), and not all authors agree about the clinical, radiological, and physiological features, particularly of centrilobular emphysema (Sweet, Wyatt, Fritsch, and Kinsella, 1961; Snider, Brody, and Doctor, 1962; Reid, 1967; Thurlbeck, Henderson, Fraser, and Bates, 1970). In one type of emphysema, however, that associated with $\alpha_1$-antitrypsin deficiency, there seems little doubt that the lower zones are predominantly affected (Eriksson, 1965; Guenter et al., 1968; Hutchison et al., 1971).

Direct evidence of pathological type is seldom
available during life, and in view of differing opinions which have been expressed, we felt that there would be reasonable grounds for studying the relationship between clinical and physiological findings and radiological distribution of the lesions. We therefore selected two groups of patients for special study, one group having radiological evidence of emphysema predominantly affecting the upper zones and the other group (omitting those with \( \alpha_1 \)-antitrypsin deficiency) with evidence of lower zone disease. The arguments for and against this approach will be discussed later in this report.

**PATIENTS AND METHODS**

The patients included in this study were referred to the Chest Unit from chest clinics, and other medical or surgical units, in or outside the King's College Hospital Group. Seventy-eight patients attending for the first time during the period 1964 to mid-1971 had definite radiological evidence of pulmonary emphysema (Laws and Heard, 1962) which occurred either alone or in association with bronchitis. Patients with asthma or severe pulmonary fibrosis and those who had undergone previous thoracic surgery were not considered. No patients were included unless obvious and extensive pulmonary abnormalities were seen (Figs 1 and 2).

In the 50 patients who form the main study, emphysema predominantly affected either the upper half or the lower half of the lung fields as seen on the postero-anterior chest radiograph. A detailed case history was obtained by the authors and special emphasis was placed on the presence and age of onset of permanent unremitting exertional dyspnoea and of chronic bronchitis as defined by the Medical Research Council (1965). The total lifetime cigarette consumption was expressed in grams, as previously described (Hutchison et al., 1971). The body weight was expressed as a percentage of the mean value for subjects of equivalent height and age (Society of Actuaries, 1959).

Data from 18 severely affected patients have been included in a previous study (Hutchison et al., 1972); 28 of the original 78 patients were excluded from the main study:

(a) eight patients with \( \alpha_1 \)-antitrypsin defi-

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**FIG. 1.** Upper zone emphysema: 45-year-old man with normal exercise tolerance. FEV\(_1\) 53% predicted value; TLco 113% predicted value.
Radiological distribution of pulmonary emphysema

FIG. 2. Lower zone emphysema: a severely disabled 63-year-old man. FEV₁ 24% predicted value. TLCO 37% predicted value.

ciency previously described (Hutchison et al., 1971);

(b) eight patients in whom there was no definite predominance of emphysema in either the upper or the lower half of the lung fields. In one patient a large bulla occupied the central part of the right lung field;

(c) 12 patients for whom data were incomplete; eight had upper zone emphysema and four had lower zone emphysema.

RADIOLOGY Standard postero-anterior and lateral chest radiographs were obtained in all patients, and bilateral whole-lung tomograms in all but two. The tomograms were taken with the patient supine, at intervals of 3–4 cm, from approximately 6 to 20 cm from the table top. Emphysema was recorded as present when the peripheral vasculature was attenuated or destroyed (Laws and Heard, 1962). The localization of bullae, if present, was also noted. A decision was made as to whether emphysema was more severe in the upper half of the lung fields ('upper zone emphysema') or in the lower half ('lower zone emphysema'). The abnormalities were obvious in all cases, but no attempt was made to match the two groups in respect of the apparent severity of the radiological lesions, nor were these lesions required to be distributed symmetrically between the two lungs.

BIOCHEMICAL METHODS Starch gel electrophoresis and assay of trypsin inhibitory capacity were carried out as previously described, the values for trypsin inhibitory capacity in 279 healthy unrelated subjects being used as controls (Hutchison et al., 1971). The nomenclature of the α₁-antitrypsin variants is given by Fagerhol (1968).

PULMONARY FUNCTION Forced expiratory volume in one second (FEV₁), vital capacity (VC), functional residual capacity (FRC), carbon monoxide transfer factor (TLCO), permeability constant (kCO), and arterial oxygen and carbon dioxide tensions (Pao₂, Paco₂) were measured by methods previously described (Hutchison et al., 1971). Normal values for FEV₁, VC, FRC, and TLCO were obtained from Cotes (1968) and the results were expressed as a percentage of the predicted value. Normal values for Pao₂ were obtained from the equation of Raine and Bishop (1963).
N. A. Martelli, D. C. S. Hutchison, and C. E. Barter

**TABLE I**

**CLINICAL DATA**

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<tr>
<th></th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Height (m)</th>
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<th>% of Predicted</th>
<th>Dyspnoea on Exertion (age of onset-yr)</th>
<th>Chronic Bronchitis (age of onset-yr)</th>
<th>Cigarette Smoking (total-g x 10^3)</th>
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TIC = trypsin inhibitory capacity (mg trypsin inhibited/ml plasma)
The four subjects with MS or FM variants (Fagerhol, 1966) have been omitted
NS = not significant at the 5% level

**TABLE II**

**MEASUREMENTS OF RESPIRATORY FUNCTION**

<table>
<thead>
<tr>
<th></th>
<th>FEV&lt;sub&gt;1&lt;/sub&gt; (% pred)</th>
<th>VC (% pred)</th>
<th>FRC (% pred)</th>
<th>TLC (% pred)</th>
<th>TLco (ml/min/mmHg)</th>
<th>kco (min-1)</th>
<th>PaO&lt;sub&gt;2&lt;/sub&gt; (mmHg)</th>
<th>PaCO&lt;sub&gt;2&lt;/sub&gt; (mmHg)</th>
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<td>0-94</td>
<td>9-7</td>
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<td>19</td>
<td>19</td>
<td>19</td>
<td>18</td>
<td>18</td>
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<tr>
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<td>111-2</td>
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<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
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</table>

NS = not significant at the 5% level

**RESULTS**

The criteria for entry into the study were fulfilled in 50 patients, of whom 31 (62%) had upper zone emphysema and 19 (38%) had lower zone emphysema. Clinical and physiological data are shown in Tables I and II.

**RADIOLOGY**

One or more bullae were observed in 32 of the 50 patients; the proportions with bullae in the upper zone (19 out of 31 cases) and the lower zone (13 out of 19 cases) groups did not differ significantly. There was no association between bullae and chronic bronchitis; bullae were found in 12 of the 21 upper zone patients who also had chronic bronchitis, and in 7 of the 10 who did not (P=0-25). One patient with bullae in both upper zones also had a bulla in a lower zone, and one with bullae in both lower zones also had a bulla in an upper zone.

**CLINICAL FEATURES**

Only four patients were females, all of whom had upper zone emphysema.

There was no significant difference in age between the upper zone and the lower zone groups.

Considering the male patients only, those with upper zone emphysema were significantly taller and heavier than those with lower zone emphysema; the difference in weight as a percentage of the predicted value, however, did not reach the 5% level of significance (see Table I).

Permanent exertional dyspnoea was the commonest major complaint and occurred in all members of the lower zone group and in 24 of the 31 members of the upper zone group (77%), the difference between the two groups being significant (P=0-027). The mean age of onset of dyspnoea, when present, was similar in the upper and lower zone groups (P>0-9). The seven patients who were free from exertional dyspnoea were all in the upper zone group, and five of these were also free from chronic bronchitis.

Chronic bronchitis was present in 38 patients (76%), and although the incidence was higher in the lower zone group (89%) than in the upper zone group (68%), this difference fell short of a significant level (P=0-065). The onset of chronic
Radiological distribution of pulmonary emphysema

![Graph showing relationship between onset of exertional dyspnoea and chronic bronchitis in patients with 'upper zone emphysema' (UZE) and 'lower zone emphysema' (LZE). Horizontal line through zero indicates the simultaneous onset of chronic bronchitis and dyspnoea.]

bronchitis, when present, occurred at a significantly younger age in the lower zone group (mean 34.9 years) than in the upper zone group (mean 45.3 years) (0.02>P>0.01). Chronic bronchitis and exertional dyspnoea started, on average, at about the same age in patients with upper zone emphysema, though a wide variation was observed in the time-relationship of the two symptoms, some patients developing dyspnoea many years before bronchitis and vice versa (Fig. 3). In the lower zone patients, on the other hand, chronic bronchitis either started at the same time as dyspnoea or preceded it, dyspnoea being noticed before chronic bronchitis in only one case. To assess the relationship between these two symptoms, the age of onset of chronic bronchitis was subtracted from the age of onset of dyspnoea in each case, negative values being assigned where appropriate, a similar procedure to that carried out by Hutchison et al. (1972).

The mean difference between the ages in the lower zone group (+10.9 years SD 13.5) was significantly greater (t=2.35; 0.025>P>0.01) than in the upper zone group (+1.5 years: SD 10.1).

All 50 patients were, or had been, cigarette smokers. Among the upper zone patients, those with chronic bronchitis (who were older) had smoked significantly more than those without (0.05>P>0.025).

RESPIRATORY FUNCTION Data from the upper zone and lower zone groups are compared in Table II. The mean FEV₁ as a percentage of the predicted value and the Pao₂ were significantly more reduced in the lower zone group and the Paco₂ was significantly greater. Otherwise there was no difference between the two groups. The mean Paco₂ in the lower zone group was significantly greater than an assumed normal value of 40 mmHg (P<0.01); Paco₂ was normal in the upper zone group. All other measurements shown in Table II differed very significantly from normal in both groups (P<0.001).

The respiratory measurements in general showed rather greater impairment in those with chronic bronchitis although there was no significant difference for the individual tests within the upper zone group (Tables III and IV). A similar comparison in the lower zone group was not possible as only two patients were free from chronic bronchitis.

**TABLE III**

EFFECTS OF CHRONIC BRONCHITIS: CLINICAL DATA IN UPPER ZONE EMPHYSEMA

<table>
<thead>
<tr>
<th></th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Height (m)</th>
<th>Weight</th>
<th>Dyspnoea on Exertion (age of onset-yr)</th>
<th>Cigarette Smoking (total in g X 10⁶)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without chronic bronchitis</td>
<td>8</td>
<td>2</td>
<td>10</td>
<td>47.2</td>
<td>8</td>
<td>45.6</td>
</tr>
<tr>
<td>Mean</td>
<td>8</td>
<td>2</td>
<td>47.2</td>
<td>1.727</td>
<td>8</td>
<td>45.6</td>
</tr>
<tr>
<td>SD</td>
<td>5.9</td>
<td>61.2</td>
<td>1.067</td>
<td>6.3</td>
<td>8</td>
<td>45.6</td>
</tr>
<tr>
<td>With chronic bronchitis</td>
<td>19</td>
<td>2</td>
<td>21</td>
<td>54.9</td>
<td>19</td>
<td>47.4</td>
</tr>
<tr>
<td>Mean</td>
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<td>54.9</td>
<td>1.724</td>
<td>19</td>
<td>47.4</td>
</tr>
<tr>
<td>SD</td>
<td>8.1</td>
<td>64.1</td>
<td>0.076</td>
<td>10.4</td>
<td>19</td>
<td>47.4</td>
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<tr>
<td>P (difference of means)</td>
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<td>NS</td>
<td>NS</td>
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<td>0.01-0.025</td>
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</table>

Male patients only.

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TRYPSIN INHIBITORY CAPACITY  The mean trypsin inhibitory capacity of the upper zone group (Table I) did not differ significantly from that of the lower zone group (0.8>P>0.7); the mean values in both groups, however, were significantly greater than that of the controls. Four heterozygotes with MS or FM variants (Fagerhol, 1968) were omitted from these calculations.

There was no significant difference in the mean trypsin inhibitory capacity between patients who had chronic bronchitis and those who did not (0.7>P>0.6).

DISCUSSION

The reports available in the literature on the relative proportions of upper zone and lower zone emphysema among patients with radiological evidence of the disease are far from consistent. Fraser and Bates (1959) and Bentivoglio et al. (1963), who studied patients considered to have emphysema on clinical and physiological grounds, found that the lower zones were the most severely affected in the great majority of cases in whom there was a definite zonal predominance.

Where patient selection was based on the presence of radiological bullae, however, rather different results were obtained. Thus Stone, Schwartz, and Feltman (1960), Laurenzi, Turino, and Fishman (1962), Viola and Zuffardi (1966), and Boushy, Kohen, Billig, and Heiman (1968) found that in almost all their patients the main lesions were in the upper zones. Ogilvie and Catterall (1959) and Simon (1964), on the other hand, found bullae to be more evenly distributed, as in the present study. These discrepancies must be presumed to arise from differing selection criteria.

Permanent dyspnoea at rest or on effort was a feature of 86% of our patients and was present in all patients with lower zone emphysema, of whom the youngest was only 42 years of age. A number of the younger patients with upper zone emphysema, however, were free from this symptom, and similar cases were also observed by Viola and Zuffardi (1966), Boushy et al. (1968), and Colp, Park, and Williams (1970). There is little information on the relationship of dyspnoea to lower zone emphysema apart from the patients of Ogilvie and Catterall (1959) who all complained of severe dyspnoea.

Chronic bronchitis occurred in 76% of the patients, a similar percentage to that found in previous studies (Burrows, Niden, Fletcher, and Jones, 1964; Hutchison et al., 1972). It was rather more common in the lower zone than in the upper zone group though the difference did not attain a significant level (P=0.065); a somewhat unexpected finding was the very much earlier onset (by some 10 years) in the lower zone patients. Again, the only other comparable information on this matter comes from the study of Ogilvie and Catterall (1959) who, like ourselves, found chronic bronchitis in many of those with lower lobe disease; it was virtually absent from all of their five patients with upper lobe disease, whose mean age was similar to that of our upper zone group who did not have chronic bronchitis.

The relationship between the age of onset of chronic bronchitis and that of permanent exertional dyspnoea is also of some interest. In patients with upper zone emphysema we have observed (Table I) that these two features originate on average at about the same age; in lower zone emphysema, chronic bronchitis preceded dyspnoea by over 10 years. There were wide variations in the time-relationship of the two symptoms in individual patients (Fig. 3). In some the symptoms develop simultaneously, but in others they may be separated by an interval of many years. Statistical analysis of these times...
intervals showed that there was a significant difference between the upper and lower zone groups (0.025>P>0.01). Chronic bronchitis, being commoner in those with lower zone disease, might, at first sight, have appeared to be responsible for the differences between the two groups in respect of clinical and physiological data, but on analysis of the upper zone group alone, no such effect could be demonstrated.

Lieberman (1969) and Kueppers, Fallat, and Larson (1969) have suggested that a high proportion of cases of emphysema are associated with the heterozygous form of α1-antitrypsin deficiency. In the present study, however, the mean trypsin inhibitory capacity in both the upper and lower zone groups was significantly greater than that observed in 14 obligatory heterozygotes (parents or children of subjects homozygous for α1-antitrypsin deficiency), confirming the previous observations (Hutchison et al., 1971, 1972) that there is apparently no excess of heterozygotes among patients with emphysema. Kueppers (1968) has suggested that an inflammatory process such as chronic bronchitis may elevate trypsin inhibitory capacity, thus masking the heterozygote; in the present report, it was found that the presence or absence of chronic bronchitis had no significant effect upon the trypsin inhibitory capacity.

The important question now arises as to whether the radiological distribution of the lesions in the cases reported here has any pathological significance. By the nature of the study there is, of course, no evidence bearing directly on this point, but a certain amount of relevant information can be gained from the literature. It is known, for instance, that the radiological abnormalities in α1-antitrypsin deficiency are almost always at their most severe in the lower zones (Eriksson, 1965; Guenter et al., 1968; Hutchison et al., 1971) and that panlobular emphysema was the dominant lesion in most of the cases where the histology was available (Eriksson, 1965; Schlesener, Talamo, Paré, and Thurlbeck, 1968). In random necropsies, the lesions of severe panlobular emphysema have a similar basal distribution, whereas those of centrilobular emphysema are particularly found in the upper zones (Thurlbeck, 1963; Wyatt et al., 1964).

There has been some disagreement as to whether centrilobular emphysema can produce either radiological abnormalities or physical disability. Snider et al. (1962) found that even quite advanced lesions had shown no ill-effects during life. Thurlbeck et al. (1970), on the other hand, found obvious radiological vascular attenuation in a number of the more severe cases of centrilobular emphysema, though in many others this pattern was not seen. Wyatt et al. (1964) and Heard (1969) demonstrated lung sections where such cases were associated with extensive bullous formation, and it is hard to believe that such lesions as these would often escape detection by chest radiography, particularly if tomograms were also done. Differences in interpretation may have contributed in part to the controversy; when the centrilobular lesion has resulted in the destruction of a large part of the lobule, it may be difficult to distinguish this from the panlobular form. Some investigators have preferred to classify all such lesions as panlobular emphysema (Burrows et al., 1966; Reid, 1967) and thus the very cases of centrilobular emphysema most likely to cause symptoms or to be visible on the chest radiograph would presumably not have been classified as such. Dunnill (1969) considered that the typical appearance could always be recognized. A further difficulty is posed by the fact that many patients have both types of emphysema together (Laws and Heard, 1962; Thurlbeck, 1963; Gough, 1965), and serious disagreements between observers on the type present in a given lung are not uncommon (Thurlbeck et al., 1969; Mitchell et al., 1970). The latter authors attempted to make a distinction between the two types on the basis of clinical or physiological features. The only difference that they were able to detect was in the location of the lesions, and they expressed some doubt as to whether they were in fact observing two quite separate disorders. In this study, we found rather greater differences between two groups defined on radiological than on pathological criteria, but the major disparities between centrilobular and panlobular emphysema observed by Sweet et al. (1961) have no counterpart here.

One further point might be made in favour of considering our cases as two separate groups; we were for the most part able to place the lesions in one or other half of the lung fields with some confidence. (Doubtful cases usually had advanced generalized disease, and the upper and lower lung fields appeared equally damaged.) If pulmonary emphysema were the result of a single pathological process, one would perhaps expect to find an equivalent number of patients in whom the mid-zones were preferentially affected. In fact, only one of the patients whose cases were reviewed prior to selection for this study would have been classified as a ‘mid-zone’ subject; his chest radiograph showed a large bulla occupying the right mid-zone with apparently...
normal lung above and below it. It is unfortunate that a pathological assessment of the type of emphysema is seldom available during life, since adequate biopsy specimens can be obtained only at open thoracotomy.

In spite of the conflicts of evidence which have already been mentioned, it appears to us that there are still some grounds for believing in the existence of the two separate pathological types and that these types may correspond to the two radiological groups considered in this report.

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Radiological distribution of pulmonary emphysema


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