

Precipitous fall of the forced expiratory volume

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Two patients are described who showed a precipitous loss of forced expiratory volume from normal to low levels within a few years. They did not have bronchial asthma and we suggest that this pattern of deterioration may occur occasionally in patients before they attend hospital with the symptoms of chronic bronchitis.

Patients attending hospital on account of chronic airways obstruction usually present with breathlessness. Most, at this stage, are found to have a severe reduction of the forced expiratory volume (F.E.V.), but little is known about the nature of the deterioration in ventilatory function in earlier phases of the illness. Two patients are described to show how precipitous this deterioration may be. The observations illustrate that the step-wise drop in function reported in some patients (Howard, 1967) may be sufficiently large to plunge a few individuals from normal levels of ventilatory capacity into severe disability within a few years.

CASE REPORTS

CASE 1 A. P. was first seen in 1956 at the age of 54 years, giving a 10-year history of early morning cough and winter bronchitis necessitating 1-2 weeks off work annually. Effort dyspnoea of steadily increasing severity had been present for one year. On physical examination the only abnormal signs were found in the respiratory system. Chest expansion was poor, the lung fields were enlarged to percussion, the breath sounds were generally reduced, particularly at the bases, and a few scattered wheezes were audible.

A partial gastrectomy had been performed in 1947, but his previous health had been otherwise good. His father died of bronchitis at the age of 57 years. For seven years before being seen he had been employed as a welder, but had worked at the coal face for 18 years from the age of 15: he had also had a spell in the Army. He had smoked 25 cigarettes per day until about one year before attending outpatients.

His breathlessness had increased until 1959, when he was forced to give up work. Routine examination revealed an abdominal mass which proved to be an aortic aneurysm. A chest infection followed the resection, which was otherwise uneventful. There was

never any evidence of heart failure and he was not cyanosed. He remained dyspnoeic until the autumn of 1964, when he suffered a severe chest infection from which he never adequately recovered: there was radiographic evidence of bronchopneumonia. He was not seen again before his death in November 1965, which was said to have been due to acute cor pulmonale. The electrocardiogram was consistently normal and the chest film demonstrated enlarged lung fields, suggesting generalized emphysema, and a fine nodulation suggestive of pneumoconiosis.

The Table shows the measurements of F.E.V. and forced vital capacity (F.V.C.), and Fig. 1 shows a plot of the values against time. The F.E.V. on initial referral to the clinic was normal for his age and height (predicted value 2.5 litres (Baldwin, Courman, and Richards, 1948)) and considerably higher than that observed for most of our patients. The subsequent course was one of a precipitous fall until 1961, when it levelled out at about 0.5 litre. The F.V.C. also declined, but more slowly than the F.E.V. The sputum was observed to be intermittently purulent and *Streptococcus pneumoniae* was isolated on occasions. Microscopic examination did not reveal an excess of eosinophils, although specific counts were not made. There was no eosinophilia in the peripheral blood. In 1962 0.25 g. of aminophylline injected intravenously caused a small improvement in the F.E.V. from 0.3 litre to 0.8 litre and of the F.V.C. from 2.8 litres to 3.0 litres. It was not felt on clinical grounds nor as a result of these investigations that the precipitous fall of the F.E.V. could be due to bronchial asthma. The severity of cough, phlegm, and winter chest illnesses did not appear to be any different before, during, or immediately after the steep fall of ventilatory capacity. The breathlessness, however, increased rapidly.

The total lung volume (T.L.C.) and residual volume were measured by a closed-circuit helium technique (Gilson and Hugh-Jones, 1949) in 1959 and 1961. The values of T.L.C. were 7.38 litres and 7.81 litres

respectively, with corresponding residual volumes of 52% and 56%. Arterial blood withdrawn in 1962 showed a saturation for oxygen of 94.5% and a carbon dioxide tension of 42 mm. Hg.

CASE 2 J.G. was first seen in 1951 at the age of 47, when he volunteered to take part in a long-term follow-up study of the symptoms of bronchitis. He was entirely symptom-free and considered himself to be in good health. He was still symptom-free in 1956, when measurements of the F.E.V. were begun,

TABLE
MEASUREMENTS OF F.E.V. AND F.V.C. MADE DURING FOLLOW-UP STUDIES

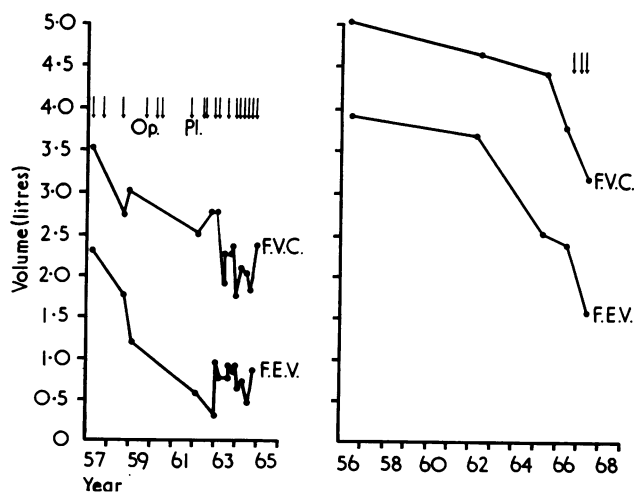
Date	F.E.V. _{0.75} (l.)	F.V.C. (l.)
<i>Patient A.P. Case 1</i>		
2. 5.57	2.4	3.5
7.10.58	1.9	2.8
30. 1.59	1.2	3.0
20. 3.62	0.6	2.5
7.11.62	0.3	2.8
20.11.62	0.9	2.8
12. 2.63	0.7	2.8
9. 4.63	0.9	1.9
4. 6.63	0.8	2.2
30. 7.63	0.7	2.3
22.10.63	0.8	2.3
14. 1.64	0.5	1.8
10. 3.64	0.6	2.1
5. 5.64	0.6	2.0
12. 5.64	0.5	1.8
9. 6.64	0.6	1.9
29. 8.64	0.9	2.4
Predicted ¹	2.5	3.6
<i>Patient J.G. Case 2</i>		
1956	3.9	5.0
1962	3.7	4.6
1965	2.5	4.4
1966	2.4	3.8
1967	1.6	3.2
Predicted	3.0	3.8

¹ The predicted values were calculated according to the formulae of Baldwin, Cournand, and Richards (1948).

and remained so until 1966, when he experienced his first attack of winter bronchitis. In the past 18 months he has had two further chest illnesses necessitating time off work, but he has denied a productive cough between attacks. A progressive effort dyspnoea has developed during this period, and he is now limited to walking at his own pace on the level. There is no hypertension nor recognizable cardiac defect.

The past health has been excellent, with no major illness. He is a non-smoker, and worked as a turner for 20 years before becoming a tool-room foreman. The Table shows the measurements of F.E.V. and F.V.C. and Fig. 1 a plot of the values against time. The F.E.V. fell in two steps, one between 1962 and 1965 and the other between 1966 and 1967. The 1967 figure has been checked on three occasions during a period of one month. The F.V.C. has also been falling steadily, but the decline began after that for the F.E.V. and even now is still 89% of his predicted value (Baldwin *et al.*, 1948). He was investigated in some detail in November 1967. The T.L.C. measured by the closed-circuit helium technique was normal at 6.478 ml. The F.V.C. at this point was 3.2 litres, making the residual volume 51% of the total lung volume. The specific airways conductance measured by the whole body plethysmograph (DuBois, Botelho, and Comroe, 1956) was 0.07 sec.⁻¹cm.H₂O⁻¹ (normal values in this laboratory: 0.16–0.31 sec.⁻¹cm.H₂O⁻¹). An arterial blood gave values for Po₂ of 75 mm. Hg, for PCO₂ of 35 mm. Hg and SaO₂ 93.5%. The A-aDO₂ was raised (36 mm. Hg) and the three-compartment analysis of lung distribution (Penman and Howard, 1966) gave values for the alveolar dead space fraction (Fup.v) of 36.1%, for the alveolar blood shunt fraction (Fuv.p) 6.3%, and for the effective fraction (Fvp) 63.6%. Our normal values for these compartments in the sitting position are: Fup.v 25%, Fuv.p 3%, and Fvp 72%. The static recoil pressures of the lung (P_{st}) were measured by the method of Milic-Emili, Mead, Turner, and Glauser (1964). Figure 2

FIG. 1. The decline of the F.E.V._{0.75} and F.V.C. in (left) Case 1 and (right) Case 2. The arrows indicate acute chest illnesses for which antibiotics were given. Op=operation. Pl=pleurisy.



shows a plot of the values of P_{st} at various levels of the vital capacity which, judging by the work of Mead, Turner, Macklem, and Little (1967), would appear normal. Dynamic compliance at a respiratory frequency of 11/min. was also normal at 0.18 l./cm. H_2O . Inhaled isoprenaline improved the F.E.V. from 1.6 litres to 1.8 litres and the F.V.C. from 3.2 litres to 3.6 litres. At the time of study he could not produce any sputum. Thus, in summary, this patient shows a marked reduction of F.E.V. minimally affected by isoprenaline, a decrease of airways conductance, normal overall elastic properties of the lung as reflected in the measurements of dynamic compliance and static recoil pressure, and evidence of ventilation-perfusion inequality.

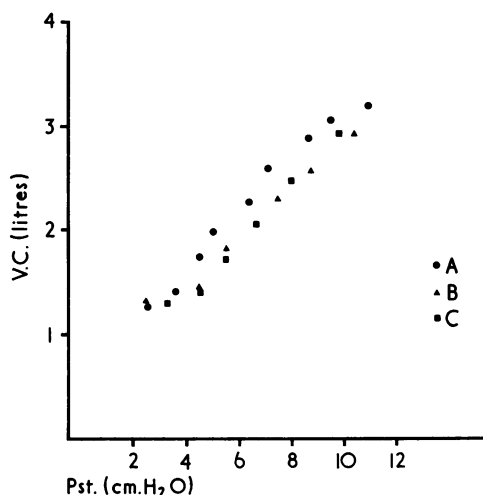


FIG. 2. The relationship between the static-recoil pressure of the lung (P_{st}) and vital capacity (V.C.). Three series of measurements were made (A, B, and C) under the same conditions.

His chest radiographs in 1965 showed a normal heart and lung fields except for some peaking of the right diaphragm and a pleural adhesion to the sixth rib anteriorly.

DISCUSSION

Hospital patients with chronic bronchitis are a selected group of those who have chronic airways obstruction. The F.E.V. is usually much reduced from normal levels on first presentation, in our experience to about 1.0 litre. Most patients give a history of many years' productive cough and winter illnesses before the onset of breathlessness, but in some patients the previous history is shorter and breathlessness might be the initial complaint. Hospital patients probably include the more severely disabled, but little is known about them before they achieve this level of disability.

There is considerable variation in the rate of deterioration of the F.E.V., but in some patients it may be no greater than that reported for normal populations even when the F.E.V. reaches low levels (1.0 litre). Presumably, therefore, in these latter individuals the F.E.V. must have been falling at a greater rate in previous phases of the disease. The decline of the F.E.V. occurs in two ways—by a steady deterioration and by sudden drops (Howard, 1967). These cases are presented because they illustrate how rapidly ventilatory function as measured by the F.E.V. may deteriorate, and that one type of disease preceding severe disability may involve a precipitous decline of function from normal levels. In the first patient the sudden drop of function occurred after many years of productive cough, winter illnesses, heavy smoking, and an occupational hazard. There was no indication as to why the sudden deterioration should occur at that moment. The acute chest illness recorded before the precipitous fall in 1958 seemed to be similar to his many previous winter illnesses. The importance of the relation of bronchial infection (as manifested by symptoms of phlegm and acute chest illnesses) to the decline of the F.E.V. has been questioned. In a study by the Medical Research Council (1966) on early chronic bronchitis in which the F.E.V._{1.0} was greater than 1.46 litres, chemoprophylaxis or treatment of acute exacerbations as they occurred had no effect on the decline of the F.E.V. Howard (1967), in a follow-up study of more severely disabled individuals, could find no more than a weak correlation between the decline of the F.E.V. and the number of acute chest illnesses per year. Cigarette smoking has been repeatedly associated with lower F.E.V. levels and also with an increased rate of decline of the F.E.V. (Higgins and Oldham, 1962; Fletcher, 1968). This patient had stopped smoking by the time the precipitous decline occurred. If these factors are important to the decline of the F.E.V. then it must be presumed that some other factor combined to achieve the precipitous deterioration in this patient.

In the second patient, the fall of F.E.V. was equally dramatic and for the most part asymptomatic. He did not have bronchial asthma, there was no smoking history and no occupational hazard. Thus the commonly recognized causes of obstructed airways were not in evidence. Mead *et al.* (1967) and Macklem and Mead (1967) have pointed out that expired flow is dependent upon the elastic recoil of the lung and the mechanical properties of the larger airways. Obstruction of a large number of smaller bronchioles in a normal dog lung was observed to have little effect upon

the F.E.V. In our patient the overall elasticity of the lung was normal: the fall in F.E.V. was probably due to a disturbance in the larger airways and was associated with a moderate decrease in airways conductance. Disease of the small airways (<2 mm. in diameter) is considered to be an important cause of ventilation-perfusion abnormality and this was clearly abnormal. There is evidence of disorder in both small and large airways, but it is not clear from these studies whether the progress of disease in these different parts of the bronchial tree can develop independently.

About 200 patients regularly attend the bronchitis clinic, but only 25 had an F.E.V. of 2.25 litres or greater when first seen. The first patient was drawn from this group of 25. The second patient was taken from a long-term follow-up study of 290 industrial workers, most of whom were initially healthy individuals. This man was the only one to show such a precipitous fall of ventilatory function although many others had step-wise losses of less severity. It is difficult to assess from these figures how frequently a precipitous loss of F.E.V. might occur in an urban

community, but it is suggested that this is one type of deterioration that occurs in patients with obstructed airways before they attend hospital.

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