whole of the way down the respiratory tract, right to the alveolar walls. What is known of the pathology of chronic bronchitis and of emphysema has certainly improved in recent years.

THE PHYSIOLOGICAL BACKGROUND—NORMAL AND DISTURBED

Dr P. A. Emerson (*Consulting physician, Westminster Hospital*)

It is my task to give you a theoretical background to the more pragmatic information that you will have from the later speakers about the various pieces of apparatus and the various ways of assessing patients with chronic bronchitis. This is a ' second M.B.' on respiratory physiology.

Definition of ' early chronic bronchitis '

The definition of chronic bronchitis is a clinical one and it seems easy enough to say, " We will pick out a number of patients who have a chronic or recurrent cough with expectoration, which cannot be attributed to some other cause ", and then to say, " You have chronic bronchitis. We will study your pulmonary function and see what we find ". The difficulty, however, is that one cannot really know whether or not these patients also have some significant degree of emphysema. By general consensus of opinion now, the degree of emphysema can only be accurately diagnosed on morphological grounds, such as you have just seen in those wonderful slides that Dr Heard has shown you. It is relatively easy to make a diagnosis in these circumstances though not of course so easy to prepare the specimens and slides.

But for the clinician it is much more difficult; if the chest x-ray shows gross evidence of emphysema, he can make an intelligent guess and say, " There is a lot of emphysema here ", and he will usually be right in these circumstances, so that such patients can usually be excluded from the studies. But if we are considering less severe emphysema it is very much more difficult. It is well recognized that even in random autopsies a significant degree of emphysema may be found in patients who have not complained of a cough, and indeed some people would almost regard this as part of a normal ageing process. So the point I want to make is that when I speak of chronic bronchitis I mean chronic bronchitis diagnosed on the basis

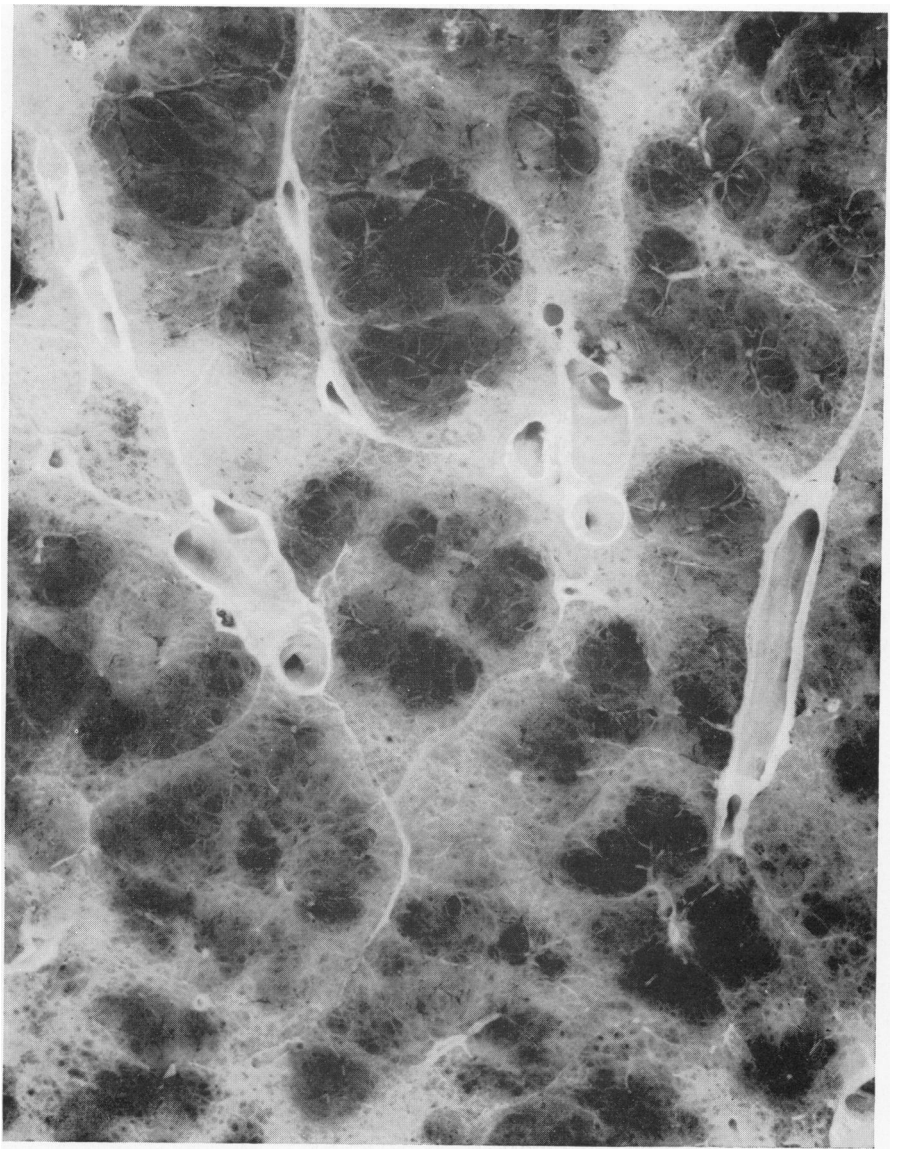

Figure 8
Centrilobular emphysema. The pale areas represent surviving lung at the peripheries of the secondary lobules. The dark areas are severely damaged lung, conveniently dust-pigmented, occupying the centres of the secondary lobules (x 3, barium sulphate impregnation)

facing page 20

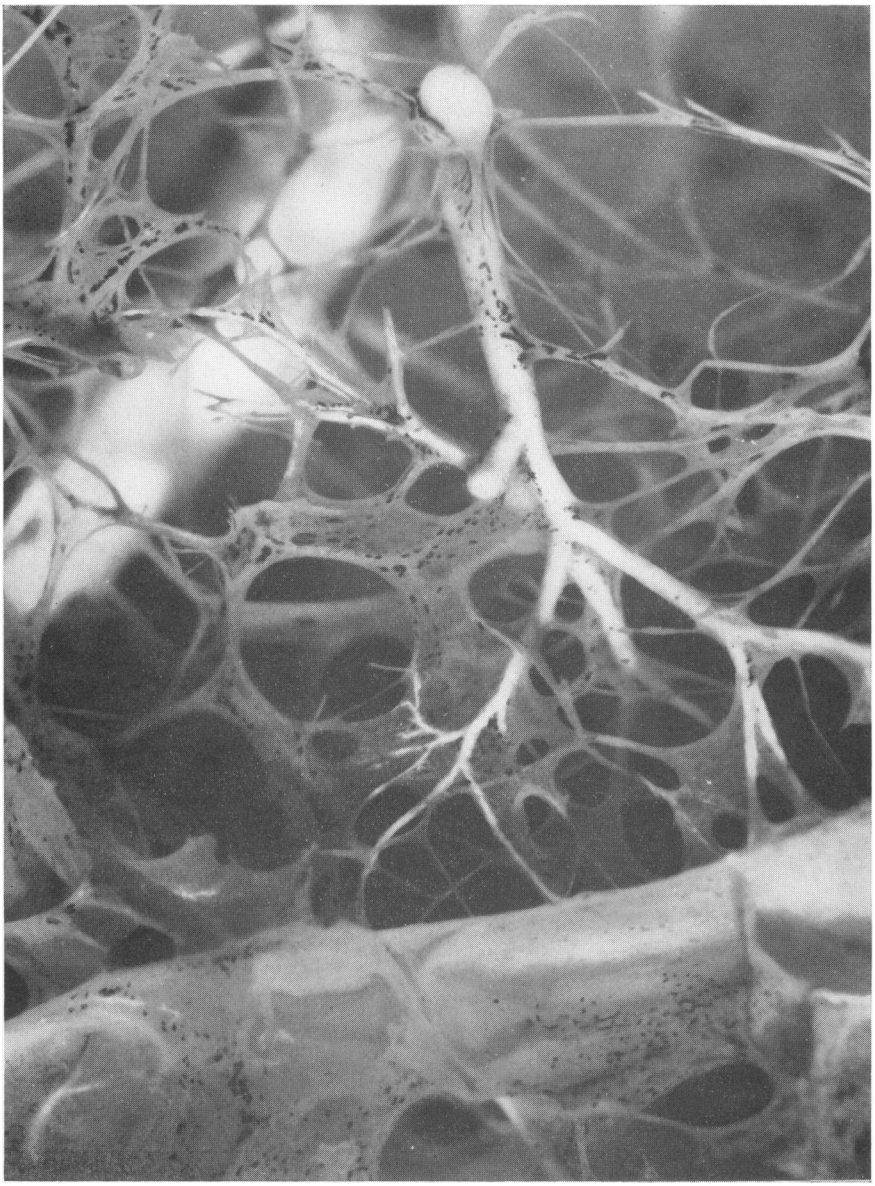

Figure 9

Severe panacinar emphysema. Most of the fine structure of the lung is destroyed leaving a few remnants of connective tissue attached to surviving pulmonary arteries which have been injected with barium sulphate-gelatin mixture (white). The septum at the periphery of the secondary lobule runs down the left side of the photograph (x 20, barium sulphate impregnation)

facing page 21

of the clinical definition, and I do not pretend to know whether or not the patients have associated slight or moderate emphysema.

Furthermore, I really do not know what ' early chronic bronchitis ' is. Presumably in any really early chronic bronchitic patient there is no change in the respiratory function. If I take this as my definition, I have to sit down, and there is nothing more to say. So what I am going to take as my definition of early bronchitis is chronic bronchitis diagnosed on a clinical basis in patients in whom there is no reason to suppose that there is any disturbance of arterial blood gases, either because these have been measured and found to be normal, or because it has not been thought worthwhile to measure them because the condition was so mild.

Disturbance of respiratory function

In any consideration of pulmonary function it is essential to consider both the ventilation of the lungs with air and the perfusion of the lungs with blood. It is much easier to measure the ventilation of the lungs with air and so there is much more information available about this. Unhappily, there is much less information about the perfusion of the lungs with blood.

The lungs are ventilated as a result of work done by the muscles of respiration during inspiration and expiration is a passive affair. There have been one or two studies done with the U.S.A. by Cherniak and Hodgson, and by Lyons and Ting who produced some evidence to show that in patients with chronic bronchitis the chest wall becomes stiffer than usual. Why exactly this is, we do not know. It is probable that this is simply a secondary change, the result of the lung changes resulting in hyperinflation of the lungs, to which I shall presently refer.

I. Airway resistance and ventilation

Figure 10 compares a normal spirogram with one obtained in a patient with obstructive lung disease. The amount of air that is left in the lungs at the end of a full expiration is called the residual volume. The circular figures represent imaginary sections taken through the subject's chest horizontally; they are not to scale because they have to match up with the projections from the spirograms but they give an idea of the situation in the normal chest and in obstructive lung disease. In patients with obstructive lung disease what happens is that, although they are able to get air into the chest, the bronchi narrow on expiration and the patient is unable to blow out as much air, so that more is trapped in the lungs, and the residual volume is increased. A normal person may have a residual volume of about 1,500 c.c. whereas in a patient with chronic bronchitis it may be as high as 3,400 c.c. This is why these patients have barrel-

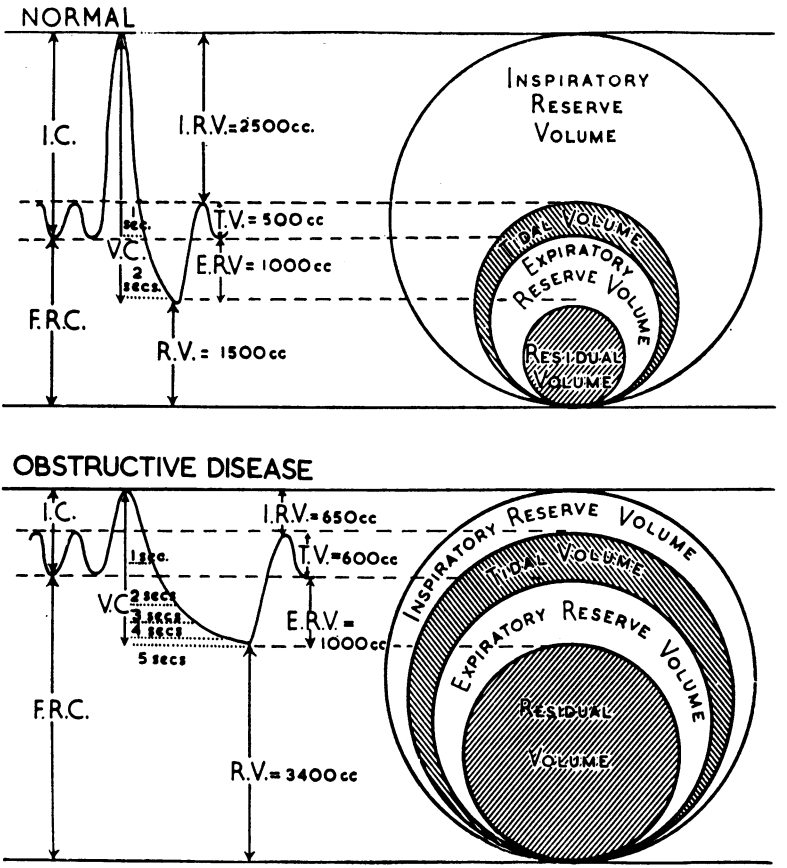

Figure 10
A comparison of a normal spirogram with one obtained from a patient with obstructive lung disease

shaped chests. The ratio of the residual volume to the total volume of the chest is a useful measurement that can be related to the degree of disability. Normally the ratio of the residual volume to the total volume of the chest is less than 35 per cent and when it is over 50 per cent there is nearly always a considerable respiratory disability.

The results in table II were taken from 83 patients with early chronic bronchitis as already defined. The figures represent the mean values expressed as percentages of the predicted lung volumes. This then is the

TABLE II
MEAN PREDICTED VALUES FROM 83 CHRONIC BRONCHITICS

Vital capacity	*Residual volume*	*Total lung capacity*
Per cent 69	*Per cent* 176	*Per cent* 100

functional profile of a typical early chronic bronchitic. His vital capacity is reduced to 69 per cent; his residual volume is increased 176 per cent—almost double the normal; and his total lung capacity works out at 100 per cent, as would be expected; the fundamental change is the increase in the residual volume.

Table III shows the mean results in 25 patients with early chronic bronchitis who were selected because they showed relatively normal or at least low normal values for the FEV_1 and peak expiratory flow rate. Again the mean lung volumes are expressed as percentages of the predicted values. As expected the total lung capacity is again about 100 per cent, and again they show this change of an increase in the residual volume (143 per cent)—nearly half again as much as it should be—and their vital capacity is reduced to 80 per cent.

TABLE III

THE MEAN RESULTS IN 25 PATIENTS WITH EARLY CHRONIC BRONCHITIS AT RELATIVELY LOW F.E.V.[1] VALUES

Mean F.E.V.[1]. 0 per cent and peak expiratory flow rate	*Mean predicted values*		
	Total lung capacity	*Residual volume*	*Vital capacity*
Per cent 71 370L/min.	*Per cent* 98	*Per cent* 143	*Per cent* 80

So the conclusion here is that one of the earliest changes in function in early chronic bronchitis is an increase in the residual volume. This measurement of the residual volume is a laboratory test and, because it takes some cumbersome apparatus to measure, it is necessary for practical field work to use some more simple and direct tests.

To describe the various methods that can be used for measuring the effects of the narrowing of the bronchial airways we must be a little theoretical for a moment. Poiseuille's Law states that the pressure required to maintain laminar flow along a tube such as an airway is directly proportional to the flow rate and inversely proportional to the fourth power of the radius of the airway. So the most important factor is the radius of the airway; any minor decrease in the radius makes a tremendous difference to the pressure that is required to keep the air going up and down the airway, and as even normal bronchial airways decrease in diameter during expiration, in chronic bronchitis the increased airway resistance is most apparent and best measured during expiration.

It is possible to measure the airway's resistance directly by various methods requiring expensive and complicated apparatus but for practical purposes it is much easier to assess this indirectly by measuring various aspects of the expiratory flow rate. The expiratory flow rate depends, not only on the radius of the airways, but also

on the tendency of both normal and abnormal airways to collapse when the intrapleural pressure becomes sufficiently high during a forced expiration and this plays an important part in limiting expiratory flow.

Fundamental to all the different methods of assessing the expiratory flow rate is the forced vital capacity spirogram. The patient takes a maximum inspiration and then expires as rapidly and fully as possible into a spirometer with a fast revolving drum as shown in figure 13.

There are a number of different ways of expressing the information given by this expirogram.

(1) *Fast expiration volume* (*FEV*) The actual value of the fast expiratory volume itself has not been used very much, but it is probably of considerable value; recently Segal, in an article in *The Lancet*, has related various parameters of the FEV to the partial pressure of carbon dioxide in the patient's arterial blood and has found that the best correlation was with the whole of the FEV itself. This was probably because the most important part of the FEV is the later part of the curve which is the part most related to the mechanical properties of the lung, whereas the upper part of the curve is related much more to the amount of effort and enthusiasm the patient has for doing the test.

This fact is important because when the airways resistance is assessed by some of the other techniques, such as the peak expiratory flow rate, the meter is picking out the point of peak flow high on the curve, and it does not reflect the mechanical properties of the lung as well as those measurements which do take into account the lower part of the curve of the fast expiratory volume.

(2) *Fast expiratory volume in one second* ($FEV_{1\cdot0}$). The actual volume of air that the patient is able to blow out in one second is measured. In general, anything below 1,500 c.c. is definitely abnormal, and down at 1,200 c.c. the patient is very short of breath. There are also tables and normograms to show the range of normality and Kory (1961) has produced a regression formula i.e.—$FEV_{1\cdot0}$= .094 (ht. in ins.)—.028 (age in years)—1.59.

(3) *Percentage of FEV expired in one second* ($FEV_{1\cdot0}$ *per cent*). The volume of air blown out in the first second is expressed as a percentage of the total fast expiratory volume. In normal subjects more than 80 per cent of the vital capacity is expired in the first second. The percentage measurement does little to discriminate between moderate and severe airways obstruction—the value being usually about 50 per cent in bronchitic patients.

(4) *Maximum expiratory flow rate* (*MEFR*). The maximum expiratory flow rate can be obtained by measuring the slope of the

curve after the first 200 c.c. for the next 1,000 c.c. The MEFR is more influenced by the subject's effort than is the maximum mid flow rate (MMFR) which is obtained by measuring the slope of the middle part of the curve which, as has already been explained, is less dependent on the subject's effort and more directly dependent on the mechanical properties of the lungs. Normal values vary between 1.8 to 4.9 litres/sec. In obstructive lung disease values of the order of only 1.0 litre/sec. are obtained.

(5) *Peak expiratory flow rate* (*PEFR*). Another single expiration measurement is the PEFR measured with the Wright peak expiratory flow meter. This makes a spot measurement on the slope of the curve at its steepest point. It has therefore the same objections as the MEFR in that it is dependent on effort. While admitting this disadvantage, the ease and convenience of the test makes it an extremely valuable one in clinical and survey work. There are two models—a large one with a range of 60—1,000 litres/min. for adults and a smaller one with a range of 20—200 litres/min. for children.

(6) *De Bono whistle.* Moving further down the scale of simplicity we come to the de Bono whistle. The instrument is simple but not very satisfactory.

(7) *The Snider match test.* The simplest apparatus is required for the Snider match test; just a box of matches. A match is struck and held 6 inches in front of the patient's open mouth; if he can blow it out with his mouth open his respiratory function is not too bad; a successful blow indicates that the patient has a $FEV_{1.0}$ of about 1,200 c.c., or would be able to achieve something like a peak expiratory flow rate of 130 litres/min. or more on the peak expiratory flow meter.

(8) *Forced expiratory time* (*FET_c*). Lastly, it is possible, without even a match, to assess the airway resistance by listening with a stethoscope over the patient's trachea, and measuring the time it takes him to blow out his vital capacity. An FET_c of less than five seconds indicates that there is no significant airway obstruction and that the $FEV_{1.0}$ percentage is something better than 60 per cent. An FET_c of over six seconds indicates some airway obstruction and that the $FEV_{1.0}$ percentage is likely to be less than 50 per cent.

II. Unequal distribution of ventilation

So far we have been considering the methods of detecting and assessing the increased airways obstruction which occurs in chronic bronchitis.

This obstruction is not evenly distributed throughout all the bronchioles and so the ventilation of the lungs, which is not completely uniform even in normal lung, becomes very unequal in

chronic bronchitis and other obstructive lung disease. It is not even a simple static problem, because time enters into the matter as well.

If there are two systems of alveoli, one of which is relatively stiff or non-compliant and one elastic or compliant, the elastic system will inflate more than the stiff one. If the airway to the elastic system is narrowed and the airway resistance thereby increased it will still inflate more than the stiff or non-compliant system as long as the flow rate is low, i.e. during slow breathing. In rapid breathing, however, the more rapid flow rate results in a disproportionate increase in the airway resistance to the partly obstructed elastic system so that it is no longer preferentially ventilated as it was at low flow rates. This is the situation that exists in the lungs. When a patient is breathing quietly the distribution of ventilation to the many alveoli may be unequal but not nearly so unequal as when the patient starts to breathe rapidly. Then the whole system changes and the uneven airway resistances contribute much more to the inequality of the distribution of ventilation and the lungs become even less efficient. The unequal distribution of air in the lungs can be assessed only by relatively complicated methods; the simplest of these is the single breath nitrogen washout test, but even this cannot be done in the field and is limited to the pulmonary function laboratory.

I hope I have now given you a brief theoretical outline of the problems concerned in the ventilation of the lungs. I have said nothing about the perfusion of the lungs with blood because I think that this probably is not disturbed in the early stages of chronic bronchitis, about which I was supposed to talk to you.

USE OF PULMONARY TESTS IN GENERAL PRACTICE

Dr George Gomez (*Member of the College of General Practitioners*)

I am going to show you some cases which are the kind seen by many family doctors.

This is an ordinary patient (figure 11). He is a company director up for an annual check-up. He eats a little too much, drinks a little too much, smokes. He is complaining of shortness of breath; he cannot chat as he did on the walk up from the station, and he has a cough. He is using a vitalograph (figure 13).