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BREATH

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EDITORIAL

Terminological inexactitudes

"It is one of the prequisites of clear thought that basic concepts must be clearly defined."

J G Scadding[1]

The terminology of the group of disorders that give rise to airflow obstruction has been the subject of argument for some considerable time. Many of us may feel that we understand quite well what is meant by such commonplace terms as chronic bronchitis, but as so often happens it may mean quite different things to different people. Under these circumstances, discussion becomes somewhat unproductive and further thought and action may be severely inhibited. For example, it appeared early on in this debate, that there were striking differences between American and British terminology. Thus in the United States 'chronic bronchitis' was regarded as a mild nuisance rather than a disease, but in Britain as a serious progressive disorder. Fletcher and colleagues[2] in a comparative study found however, that patients attending a British 'bronchitis' clinic and an American 'emphysema' clinic were very similar.

The CIBA symposium of 1959[3] was set up to provide some basic definitions for bronchitis, emphysema and asthma, the major sources of semantic confusion. The panel suggested the name 'chronic non-specific lung disease' for the whole group adding the comment that 'this cumbersome phrase will seldom be used in clinical practice'. This forecast, or hope perhaps, was unfortunately not fulfilled and the expression (often abbreviated to CNSLD) has frequently appeared in the literature, alongside its equally unlovable relatives, chronic obstructive lung (substitute pulmonary or airways) disease — COLD, COPD and COAD.

Chronic bronchitis

Chronic bronchitis seems to be the term which has caused most difficulty. The most widely used definition[4] divided this disorder into three main categories:

1. *Simple chronic bronchitis:* This essentially means persistent production of sputum, though other conditions which might result in expectoration would have to be excluded (eg bronchiectasis).
2. *Chronic or recurrent mucopurulent bronchitis:* The same as simple chronic bronchitis with additional infection.
3. *Chronic obstructive bronchitis:* This is defined as chronic bronchitis in which there is persistent narrowing of the airways.

A number of difficulties arise out of these definitions. It is widely believed (though this was perhaps not the original intention) that simple chronic bronchitis inevitably or commonly proceeds to the obstructive form and even as far as the syndrome of airflow obstruction, hypercapnia, hypoxaemia and recurrent cardiac failure (the so-called 'blue bloater' syndrome). On the contrary, it now seems likely that this sequence is followed in only a minority of cases but the erroneous concept has been reinforced by the common habit of referring to *all* such patients as 'chronic bronchitics'. Patients with obvious emphysema likewise get lumped together under the same heading, though it is quite clear that many emphysematous patients produce no sputum. Many smokers have a persistent productive cough, which is often accepted simply as part of normal existence and not something to bother a doctor about unless shortness of breath also occurs.

Emphysema

Emphysema was defined at the CIBA symposium as "increase beyond the normal in the size of airspaces distal to the terminal bronchiole either from dilatation or from destruction of their walls". The inclusion of 'dilatation' in the definition, however, would allow the inclusion of asthma or the simple enlargement of advancing age, both quite different conditions from what the clinician sees as severe emphysema. One would therefore prefer the American Thoracic Society definition[5] from which dilatation was omitted.

The definition of emphysema is based on morbid anatomical appearances and it would therefore hardly ever be possible to make the diagnosis during life if one kept rigidly to this approach. Chest radiographs and physiological tests could now be used for this purpose.

Emphysema and chronic bronchitis frequently occur together, but there is no evidence that chronic bronchitis leads per se directly to emphysema. It seems that the two conditions are more likely to be related to a third factor, namely cigarette smoking. We see after all, many emphysematous patients who have no sputum, or who develop it later in life; when the smoking habit is abandoned moreover, sputum may clear up. The expression 'primary emphysema', which may be used to indicate emphysematous patients who have no bronchitis therefore seems to have no useful place in our terminology.

The somewhat jocular expressions 'pink puffer' and 'blue bloater' (attributed to A C Dornhorst[1]) refer on the one hand to emphysema and on the other to chronic bronchitis with cor pulmonale and heart failure. These graphic terms fulfil a useful rôle in drawing attention to particular disorders so long as they do not become so firmly fixed in our minds that we cannot dislodge them when necessary.

Our terminology and classification systems, if they are to serve us really well, must reflect as far as possible the current state of our knowledge. We must therefore be sure to examine our definitions from time to time and to be quite ready to discard those terms that have outlived their usefulness.

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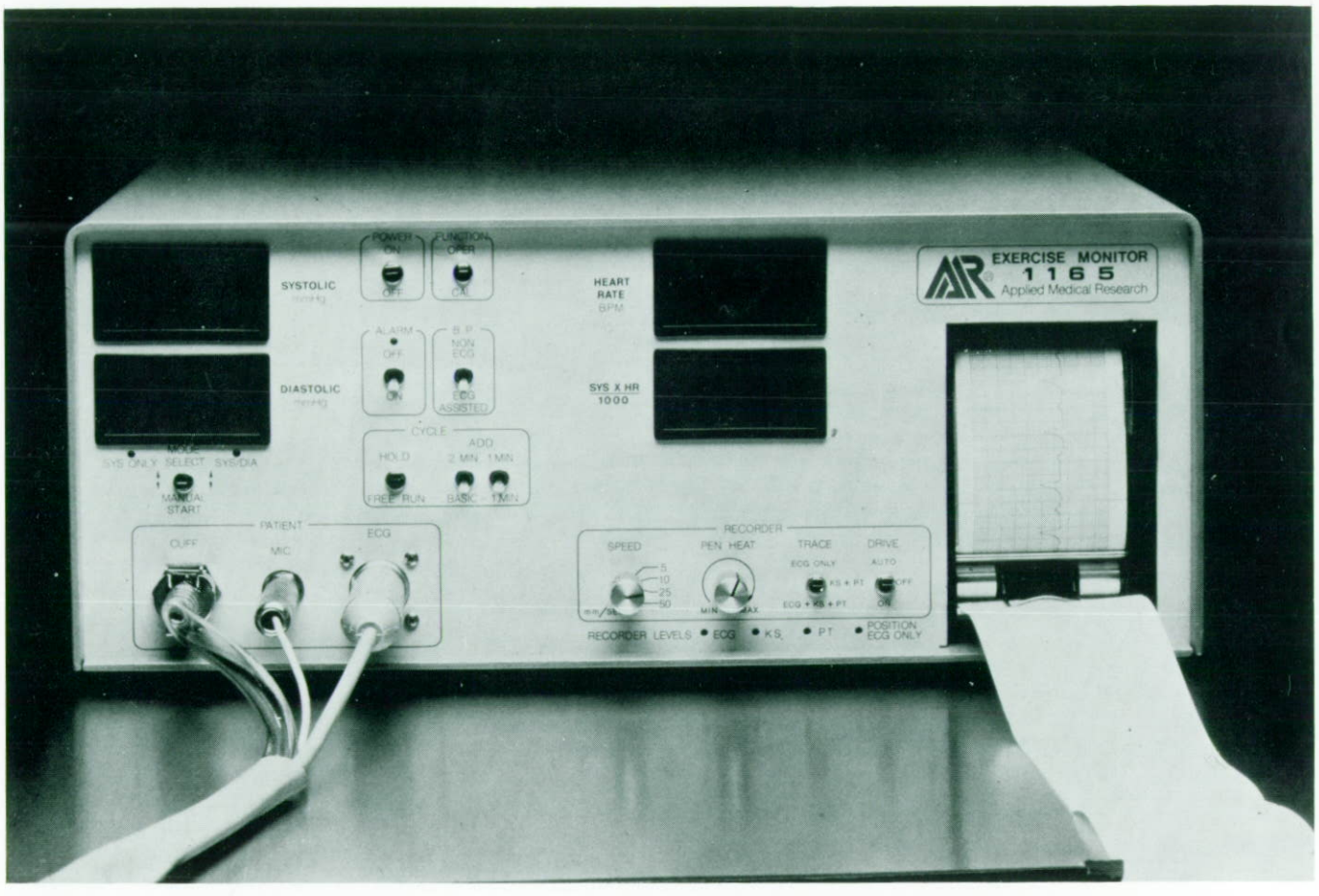

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THE SITE OF AIRWAY NARROWING

M. R. Partridge
The London Chest Hospital

Much of the clinical practice of Respiratory Medicine is concerned with narrowing of the airways, yet most of the commonly used lung function tests give no information about the site of this narrowing. While this may not be important clinically, from the research viewpoint tests yielding this information would be valuable. Measurements of FEV_1, PEFR, etc., are relatively insensitive to changes affecting the peripheral airways alone; hence further tests are needed to permit early detection of the effect on these airways of smoking and possibly of diseases such as sarcoidosis and rheumatoid arthritis. Of arguably greater importance is the need in patients with overt airway narrowing (such as asthma and emphysema), for a test which can apportion whether the narrowing is affecting mainly the larger central airways, or predominantly the smaller peripheral airways. Such information may be helpful in understanding the pathogenesis of these diseases and in directing therapeutic efforts.

There are thus two requirements for any test:

1) Can the test detect changes in the small peripheral airways in the absence of disease of the large airways?
2) In the presence of obvious airway narrowing, can the test determine whether it is mainly the large central, or mainly the smaller peripheral airways which are affected?

What tests are available and what are their indications and limitations?

Airways resistance (R_{aw})

Measurement of airways resistance is a sensitive reflection of airway dimensions. In normal subjects about 80% of the total airways resistance is located in the central intra- and extra-thoracic airways, and peripheral airways of less than 2 mm in diameter contribute little. Measurement of airways resistance is therefore relatively insensitive to changes in small airways in normal subjects and largely reflects large airway calibre. In a patient with abnormal airways resistance however, it is impossible to say whether this is due to a relatively small change in the calibre of the large airways or to a relatively large change in the calibre of the small airways. Measurement of airways resistance therefore fills neither *requirement 1* nor *2* referred to above. It is included here because it is a common misconception that even in disease it is a measure of large airway narrowing.

Frequency dependence of compliance

In a normal subject dynamic and inspiratory static compliance over a given volume range are the same and do not vary with respiratory frequency, because the time constants for all pathways in normal subjects are the same or nearly so. However, in the presence of patchy airway narrowing, areas of lung fed by narrowed airways will receive a normal distribution of ventilation during slow breaths, but as breathing frequency increases these areas will receive less and less ventilation. Under these circumstances dynamic compliance will decrease and the lung will appear to get stiffer. Thus if conventional indices of airway calibre are normal but there is narrowing of peripheral airways (and hence inequality of time constants), a reduced dynamic compliance with increased frequency of breathing will demonstrate the abnormality. Its ability to locate the site of airway narrowing is essentially restricted to cases of narrowing of the peripheral airways in the absence of disease of the large airways. It thus appears to fill *requirement 1* but not *requirement 2*; the measurement has major drawbacks in that it requires oesophageal intubation and good patient co-operation and even with care reproducibility is poor.

Closing volume

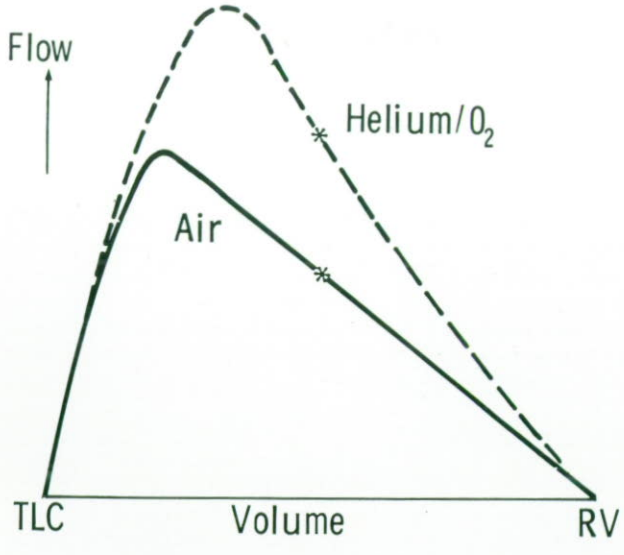
The closing volume is that lung volume at which airways begin to close in the dependent zones of the lung. Using 100% oxygen and the resident gas technique it is easy to measure the closing volume (phase 4), and the gradient of the preceding phase 3 is also a valuable measurement and thought to be a good indicator of inequality of ventilation. Closing volume is increased when there is a loss of lung elasticity or when airway closure occurs at an increased transpulmonary pressure. It is believed to be a sensitive indicator of disease in small airways and thus fulfils *requirement 1*. However, once overt airway narrowing is present the gradient of phase 3 becomes much steeper and the closing volume is often not discernible. *Requirement 2* is not met by this measurement.

Flow volume curves breathing air

Maximum expiratory flow volume curves can be recorded on an X-Y plotter, volume being derived from a spirometer or from integration of the flow signal, and flow fram a pneumotachograph or from differentiation of the volume signal. The recording is relatively easy to make and part of the curve at least, is relatively independent of patient effort. Near to TLC flow increases with driving pressure but as volume decreases dynamic compression of airways produces a plateau effect whereby greater pressures no longer achieve higher flow rates — at low lung volumes, as long as modest effort is applied the resulting curve is independent of patient effort. (In those with airway obstruction this plateau effect persists even near to TLC.)

Measurements made from the curve are usually the maximal flow rate ($\dot{V}_{max}$ which corresponds to PEFR), and flow rates at 50% and 25% of vital capacity ($\dot{V}_{max\ 50}$ and $\dot{V}_{max\ 25}$).

What does the maximum expiratory flow volume curve tell us about the site of airway narrowing? In normal subjects the main resistance to flow at large lung volumes is due to convective acceleration and disturbed flow in large airways and there is relatively little frictional resistance to flow in the peripheral airways near to TLC. The early part of the flow volume curve (usually the left hand side of the curve) predominantly reflects events in large central intrathoracic airways, while flow at low lung volumes (usually the right-hand side of the curve) reflects what is happening in the smaller peripheral airways. In patients with early isolated disease of the peripheral airways this may be reflected in reduced flow rates at low lung volumes (eg. $\dot{V}_{max\ 25}$ and $\dot{V}_{max\ 50}$) and *requirement 1* is fulfilled although a rather wide predicted normal range diminishes its value in individuals. In the presence of overt airway narrowing however, a situation analogous to that of measurement of airways resistance arises; the left hand side of the curve thus may no longer represent large, nor the right side the small airways and *requirement 2* is not wholly fulfilled. For this reason further refinements of the technique have been made using gases of differing densities.



An example of flow volume curves performed whilst breathing air and a helium/oxygen mixture in a 'responder' (see text).

Flow volume curves breathing He/O_2

In the larger central airways linear velocity of the airstream is high due to the inverted funnel effect of cross-sectional area and flow is turbulent. In the small peripheral airways however, velocity is less, total cross-sectional area is large and a laminar flow regime prevails.

Maximal flow ($\dot{V}_{max}$) is dependent on the upstream resistance, R_{us} (the resistance of the airways from alveoli to the equal presure point, or EPP) and the static recoil pressure of the lungs (P_{el}) viz:

$$\dot{V}_{max} = R_{us}/P_{el}$$

During forced expiration the pressure drop from alveolus to EPP ($\Delta P_A - EPP$) must be accounted for by pressure drops due to resistance to laminar (ΔP_{la}) flow, to turbulent (ΔP_{tu}) flow and to convective acceleration (ΔP_{ca}), ie.

$$\Delta P_A - EPP = \Delta P_{ia} + \Delta P_{tu} + \Delta P_{ca}$$

If the equal pressure points are in large airways and most of the upstream resistance is due to convective acceleration and turbulence, the pressure losses will be dependent on gas density and if this is reduced, maximum expiratory flow will increase. If on the other hand the small airways are narrowed the EPPs will be nearer the alveoli and here resistance to laminar flow (which is independent of gas density) may make up more of R_{us} and maximum flow rates will not be affected by variation in gas density. Thus by comparing flow rates at the same lung volume on gases of differing densities, the relative contributions of convective acceleration and laminar and turbulent flow to R_{us} can be determined and the major site of airway resistance inferred.

The gas mixture most commonly used for these studies is one of 80% Helium to 20% O_2. This has a density of 0.429 $Kg.m^{-3}$ compared to that of 1.286 $Kg.m^{-3}$ for air (and a slightly greater viscosity). A patient's response to breathing He/O_2 is usually assessed by the change in $\dot{V}_{max\ 50}$ breathing He/O_2 compared to that breathing air (see Figure) as:

$$\Delta \dot{V}_{max\ 50}\% = 100 \times (\dot{V}_{max\ 50}He/O_2 - \dot{V}_{max\ 50}Air)/\dot{V}_{max\ 50}Air$$

Patients who increase their flow rates by more than 20% are called 'responders' and are thought to have their major site of resistance in the large airways; those with no improvement in $\dot{V}_{max\ 50}$ breathing He/O_2 are called 'non-responders' and the major resistance to flow is thought to be in the smaller peripheral airways. Thus normal subjects whose major resistance is in the large airways increase flow rates by 30-50% on He/o_2. Patients with asthma may be responders or non-responders depending upon which airways bear the brunt of the disease while patients with chronic obstructive pulmonary disease are more likely to be non-responders.

The test is easy to perform but reproducibility is not good and attempts to improve this by additionally verifying the response by breathing a gas of high density (SF_6/O_2), or by assessing response in terms of change in mean transit time (instead of $\dot{V}_{max\ 50}$) have not proved fruitful. *Requirement 1* is only partially fulfilled because the normal range of response to He/O_2 breathing is wide, but smokers have been shown (as a group) to have lower He/O_2 responses than non-smokers. Despite the poor reproducibility the comparison of flow volume curves on air and He/O_2 fulfils *requirement 2* but as with many physiological tests, observation of a phenomenon sometimes precedes satisfactory explanation. The exact rôle of gas viscosity remains unclear, as does the size of airways in which laminar flow regimes may predominate (although it is almost certainly larger than the anatomically defined small airways).

Moment analysis of the forced expiratory spirogram

Measurements made from the flow volume curve usually reflect the state at one lung volume only (eg. flow rate at 50% of VC). Ingenious attempts to improve extraction of data from the curve by assessing the slope or by calculating maximum mid-expiratory flow rate have been made. However a further drawback remains in that the events of end expiration are compressed into the terminal portion of the curve and information about terminal events may be obscured. For this reason, moment analysis of the forced expiratory spirogram has obvious attractions, as it reflects in a single number events occurring throughout expiration and restores to importance the time factor; prolongation of expiration is after all the cardinal feature of airway obstruction.

The mean transit time (MTT) calculation is well-known to cardiologists from dye dilution techniques and higher moment analysis is a standard statistical technique. Essentially moment analysis of the spirogram is a mathematical description of the curve and the mean transit time is derived from the 1st moment about the origin and indicates the average time taken by gas molecules in the lungs to reach the mouth. Derivatives of the second moment about the mean transit time indicate the spread or variation of transit times between the initial part of the vital capacity which is expired quickly and the slower terminal portion of the VC. Derivatives of the 3rd moment indicate whether the variation occurs mainly in the initial or terminal part of the distribution. These techniques are new and await further evaluation but MTT certainly fulfils *requirement 1* although it has not so far been shown to be significantly superior to existing methods. Despite early claims derivatives of 2nd and 3rd moment analysis do not fulfil *requirement 2* although the direction of change of these parameters may be helpful in assessing site of action of drugs.

Other methods

Bronchography has been used with success to determine the site of action of antigen challenge in dogs but the technique is impractical for widespread use in patients or for serial study. Newer techniques of radio-aerosol and isotope imaging require further validation before they can be used in this context.

It can thus be seen that no technique is ideal for determining the site of narrowing within the airways in patients with airflow obstruction. If 'best buys' were to be chosen, flow rates at low lung volumes, mean transit time and closing volume would best fulfil *requirement 1*. *Requirement 2* is still not satisfactorily achieved although at the present time the use of flow volume curves breathing air and He/O_2 best dertimine whether the narrowing is predominantly affecting the larger central airways or the smaller more peripheral airways. In this context it should be recalled that small airways are defined differently by each technique. Air and He/O_2 flow volume curves define 'small airways' as airways in which a laminar flow regime prevails. These will not correspond with the anatomical small airways (those with an internal diameter of less than 2 mm), nor necessarily with the small airways detected by closing volume measurement (which are those which close on expiration), nor with the 'small airway' in the sense of airways peripheral to the equal pressure point. What is being determined is whether the predominant source of increased resistance to airflow in any one patient, at a given time, is in smaller peripheral airways or in the larger more central bronchi.

Acknowledgement
It is a pleasure to acknowledge the help of Professor Ken Saunders with my work on this subject.

Further reading
Most of these techniques are fully explained in:
Cotes: Lung function — assessment and application in medicine: 4th Edition.

For the newer techniques the interested reader is referred to:
Permutt, S., Menkes, H. A., (1979) Spirometry: analysis of forced expiration within the time domain. In: 'The lung in the transition between health and disease'. Chapter 6 (Pp 113-152), (Lung biology in health and disease series, Vol 12), edited by P. T. Macklem and S. Permutt, Marcel Dekker, New York.

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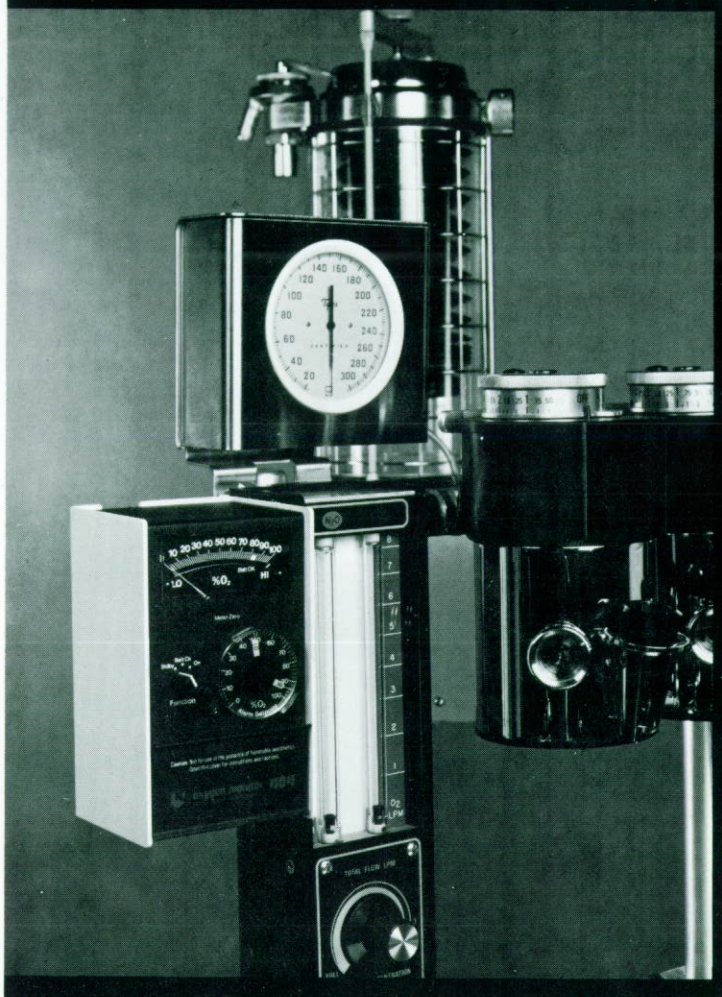

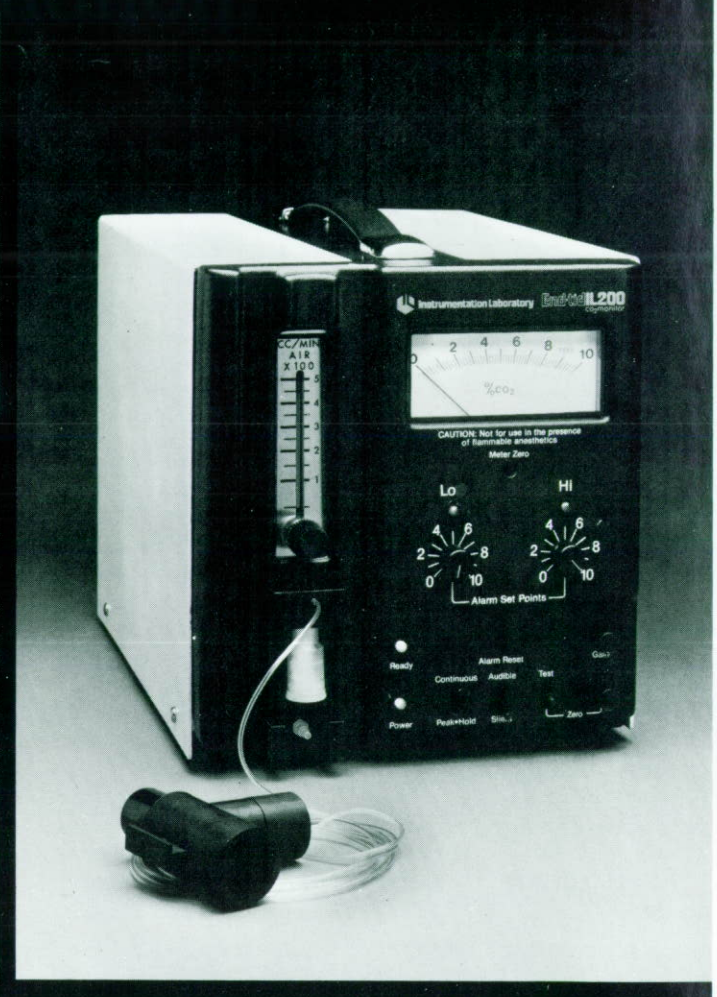

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RESPIRATORY FUNCTION TESTS IN THE SELECTION OF PATIENTS FOR BRONCHIAL CARCINOMA SURGERY.

Peter Lockwood
Respiratory Physiology Department, Harefield Hospital

In 1964 Simonsson and Malmberg pointed out that the rôle of lung function tests in the assessment of patients before thoracotomy for bronchial carcinoma, should be to demonstrate the presence of any generalised airway obstruction due to chronic bronchitis, when there is already localised airway obstruction due to the tumour. Traditionally, bronchial cancers had been regarded as predominantly space occupying lesions but in fact this is rarely the case, unless they cause complete occlusion of major airways. It was recognised that bronchitis was the most important source of post-operative complications even in general surgery and it was therefore obvious that when surgery involved the structures of the chest, bronchitis would take on an extra dimension. The group of patients most prone to bronchial carcinoma (cigarette-smoking males over the age of 50 years) are exactly that group most prone to bronchitis. These investigators recommended the nitrogen washout test as the best indicator of generalised airway obstruction.

In fact, it is not sufficient simply to demonstrate the presence of generalised obstruction in these cases. It is necessary to assess the severity of generalised obstruction and from that the likelihood of post-operative complications. Thus the concept arises that, as the degree of bronchitis increases, the risk of complications also increases.

Cardiopulmonary Complications

It is relevant at this stage to decide what we mean by a 'post-operative cardiopulmonary complication'. We have defined this as 'a condition affecting the heart or lungs, arising as a direct result of the operation, of an extent requiring alteration of the treatment and of a nature known to lead to death in some instances' (Lockwood 1980). This means that we include any condition disturbing the tranquil passage of the patient from operation to convalescence which affects the heart and lungs, including atrial fibrillation, sputum retention and similar conditions which are usually easily controlled by alert post-operative care. Equally, they are conditions which could degenerate into thorough-going illnesses resulting in death if they are neglected or treated with insufficient vigour.

Only certain cardiopulmonary complications have been demonstrated to be related to the pre-operative tests (Lockwood 1973a). These include respiratory insufficiency, sputum retention and infection, primary cardiac conditions, bronchopleural fistula and possibly haemothorax, but not pulmonary embolism, pleurisy or empyema.

Assessment of airflow obstruction

How are we to determine what constitutes a significant degree of generalised airway obstruction? Few tests on their own, taken with clinical assessment, can make any contribution to this problem (Lockwood, Lloyd and Williams 1980). It has been shown that the application of the "normal/abnormal" principle in this context is inappropriate, although the forced ventilation tests (FEV_1, MVV, flow volume values) show a significant difference between the patients considered fit for operation and those not operated on. The lung function test results, though, are needed in this context because they are the only available means by which a quantitative assessment can be made; therefore we need a method using the tests for this purpose.

Arbitrary limits applied to two or more test results taken together can be used to divide patients into different categories of risk of cardiopulmonary complications (Lockwood 1973b). These limits involve the use of either the FEV_1, MVV and FVC results together, or three out of the five TLC, RV, RV/TLC ratio, MVV and helium mixing time. The recently published modification of this scheme adds the use of the MMFR and whether or not the operation is to be a right pneumonectomy (where more than half the lung mass is removed).

Nowadays, we use a simplified version of this, based upon discriminant analysis where the general lung function picture is presented in terms of the weighted RV value plus the weighted MVV value and also involves the MMFR at 1.2 litre/second and right pneumonectomy as arbitrary limits. The RV is an adverse volume influence (related to airway obstruction) whilst the MVV is an advantageous volume and flow influence (also affected by airway obstruction). The balance between these two factors is expressed in the discriminant.

Results of selection

As an example of the effectiveness of this selection procedure we have shown that, in a sample of 240 patients assessed prior to thoracotomy, 58 subsequently developed post-operative complications.

Table I

	Risk category: Above average	Average	Below average
Complications:	17	24	17
No complications:	22	70	90
Risk	1 in 2.3 43.6%	1 in 3.9 25.5%	1 in 6.3 15.9%

It can be seen from Table I that 107 were correctly forecast, that is, 17 patients were considered to be above average risk and developed complications and 90 were considered to be below average risk and did not develop complications. 39 were incorrectly forecast, i.e. 17 patients were considered to be below average risk yet still developed complications and 22 were considered above average risk but did not develop complications. The 94 patients in the average risk category showed an approximately 1 in 4 risk of complications which was about the average for the sample. These were the patients not classified into the other two categories so that they could be considered to represent the failure of the system. Nevertheless, the demonstration that they were not bad enough for the above average risk category and not good enough for the below average risk category was clinically important.

There can be no intention of making a surgical decision on the basis of the test findings alone. Nevertheless, demonstrating that a patient has a higher or a lower degree of risk of function-related complications has a role in influencing the surgical decision and therefore in maintaining life and alleviating suffering.

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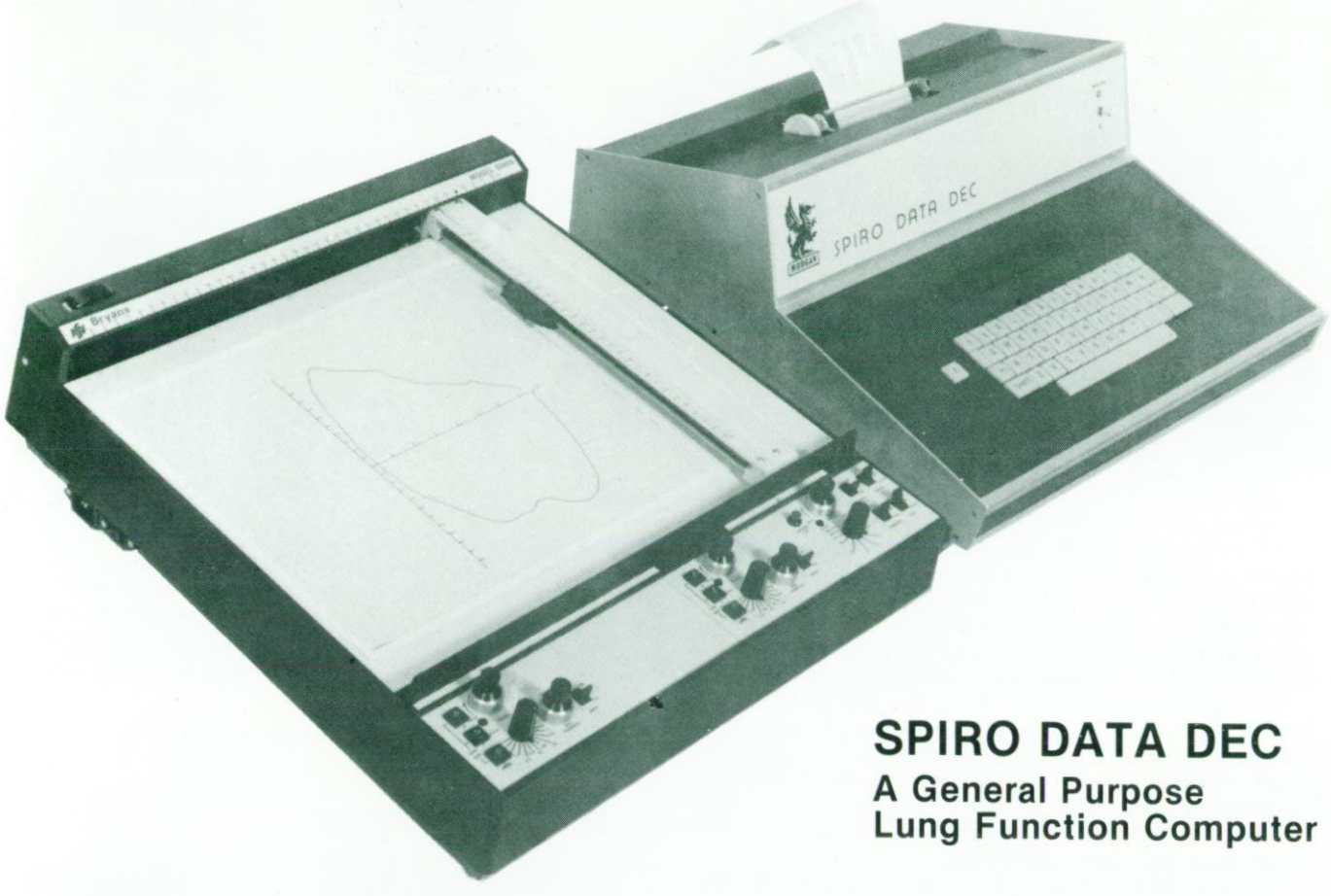

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MEDICAL MEMO

Pulmonary Emphysema

D. C. S. Hutchison
King's College Hospital Medical School

Clinical features

The destruction of the lung substance that occurs in pulmonary emphysema results in progressively severe shortness of breath and disability. Shortness of breath is, at first, seen only on heavy exertion, but a very gradual decline in exercise tolerance occurs as the disease progresses. The manual worker may thus be obliged to undertake lighter work (if available); those in sedentary occupations may be able to continue in work for a longer period but even they may be forced into early retirement and chronic invalidism.

Emphysema is predominantly a disease of cigarette smokers and over 90% of sufferers have indulged in this habit at some time. Nevertheless many heavy smokers seem to 'get away with it' and the idea has arisen that there may be individuals who are particularly susceptible to the inhalation of cigarette smoke.

There is also a striking preponderance of males, who form some 90% of the cases. The explanation commonly advanced for this, that men smoke more than women, does not account for the facts, since the ratio of male to female tobacco consumption is no more than 1.5:1. Other possible explanations (neither of which seem quite adequate) are that men are more heavily exposed to atmospheric pollution or simply that they complain far more readily to their doctors!

Sputum production is a common feature in these patients though it is probably not directly connected to the emphysematous process. Sputum production and emphysema are apparently both due to a common factor, namely cigarette smoking. Sputum in fact often clears up on cessation of smoking though the damage caused by emphysema is largely irreversible.

Severe weight loss may be a feature of the advanced disease and can often be related to loss of appetite and a feeling of distension after meals; t! is is perhaps due to the low position of the diaphragm. Peptic ulceration is rather commoner in patients with emphysema than among the general population and both disorders are related to cigarette smoking.

The patients themselves may present no obvious physical abnormalities until the disease is quite advanced, when the over-expanded chest and characteristic 'pursed lips' breathing are seen. The breath sounds may be faint or even absent, though an expiratory wheeze may appear if infection is present. Cyanosis occurs only at a fairly late stage.

Radiographic appearances

Abnormalities in the chest radiograph may be seen at any stage though are generally late features. Typical appearances are shown in Fig 1. Large bullae (gas-containing cysts) may also be seen (Fig 2).

Lung function tests

Typical lung function results are shown in Table 1.
Forced expiratory volume (FEV_1) and equivalent indices are usually much reduced by the time the patient seeks advice. The limitation of expiratory flow is brought about by airway collapse due to loss of elastic recoil.

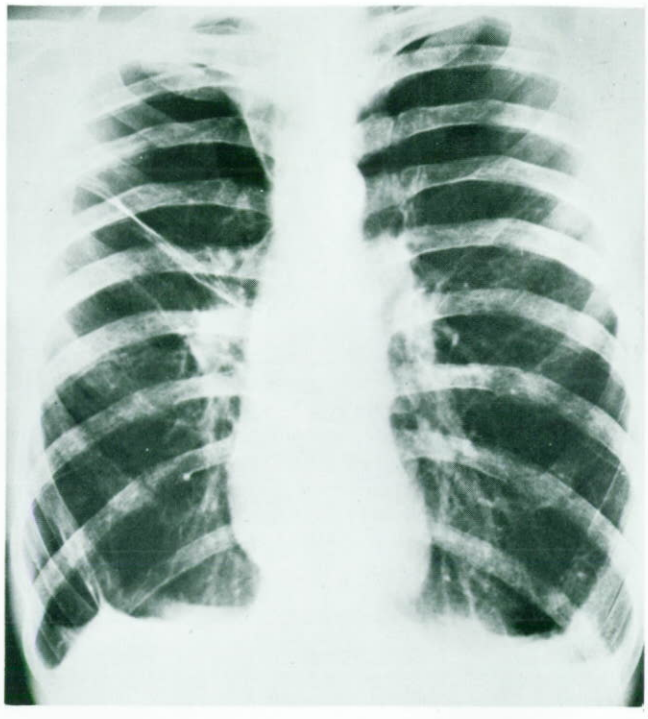

Figure 1: *Chest radiograph of a patient with very severe emphysema. Note the low flattened diaphragm and elongated heart shadow. The vascular markings are reduced in most areas and the margin of a bulla is seen in the right upper zone.*

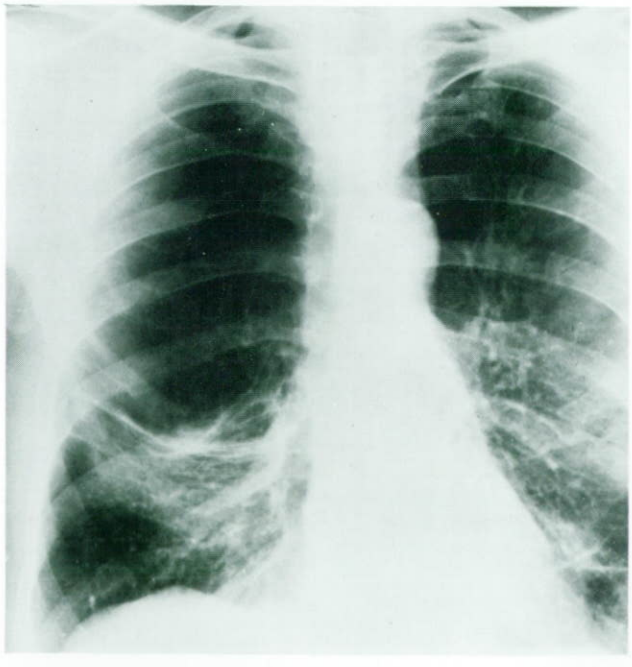

Figure 2: *A large emphysematous bulla compressing the right lower zone.*

Table 1: Lung function tests in a patient with advanced emphysema.

	Before bronchodilator	After bronchodilator	Expected normal value
FEV_1 (litres)	0.7	0.8	3.8
VC (litres)	3.1	3.8	5.0
FEV_1/VC %	23	21	75
TLC (litres)	9.5	9.4	7.5
RV (litres)	6.4	5.6	2.5
CO transfer (mmol/kPa/min)	3.0	—	10.7
Arterial pCO_2 (kPa)	5.5	—	5 to 6
Arterial pO_2 (kPa)	8.5	—	11 to 12.5

Increase in bronchomotor tone plays little part here and thus inhaled bronchodilators have little effect on FEV_1; some would argue however that an increase of 0.1 litres in the patient of Table 1 represents a response of 14%. Looking at it another way, it means only a change from 18% to 21% when the patients' results are expressed as a percentage of the *normal* value.

Vital capacity (VC) In contrast to the FEV_1, the VC response to bronchodilator is often remarkable and relates to the patients' subjective sense of relief much more closely than does the FEV_1. The bronchodilator is evidently capable of opening up previously closed lung units and this is done largely at the expense of the RV, TLC undergoing little change.

An increase in the FEV/VC ratio is often used as a measure of bronchodilator responsiveness but this is clearly misleading in emphysema if there is a substantial increase in VC. It is not generally realised furthermore, that the 'forced' VC can underestimate VC because of premature airway closure and the 'slow' VC should always be used in emphysema.

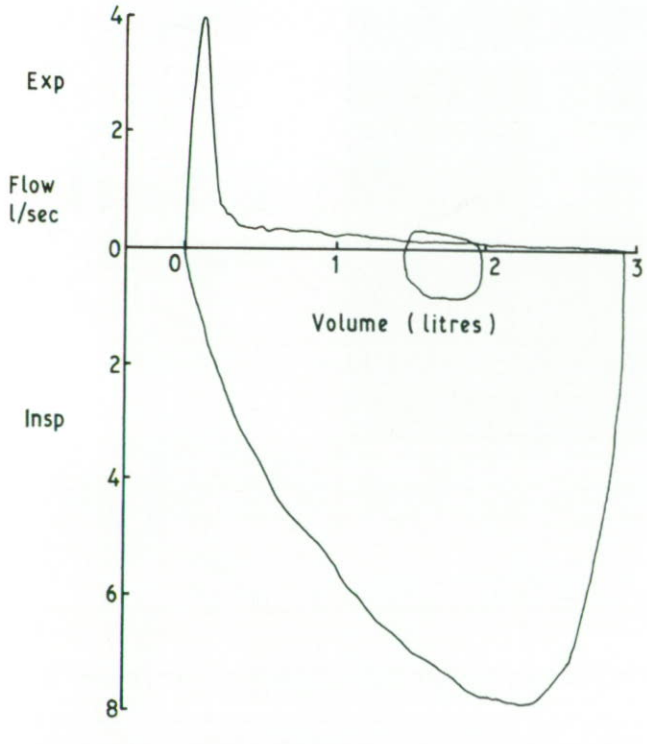

Figure 3: *Tracing of maximal flow-volume curve in severe emphysema: note rapid reduction of flow immediately after the peak. (Tidal breath at centre)*

CO transfer The loss of surface area for gas exchange results in a much reduced CO transfer factor.

Arterial blood gases Arterial pO_2 usually remains at a reasonable value only falling sharply late in the disease. Arterial pCO_2 remains normal or even low for many years, only rising above normal at a very late stage.

Maximal flow-volume curves These curves present a very characteristic appearance in emphysema (Fig 3). The inspiratory portion of the curve is normal; in contrast the expiratory portion shows a reduced peak flow which is followed by a rapid fall-off in flow to very low levels during the latter part of the vital capacity.

Pathogenesis of emphysema

Cigarette smoking plays an important part in the development of emphysema; only a minority of smokers (perhaps 20%) actually develop the disease however, suggesting that those who do, may have some special predisposition. The destruction of the elastic tissue is probably brought about by the release of the powerful enzyme elastase from circulating polymorphs or from alveolar macrophages; cigarette smoke seems in some way to stimulate release of the enzyme. Release of elastase is usually a slow continuous process and the serum contains enzyme inhibitors whose function is to protect the individual's tissues against digestion by its own enzymes; in emphysema the enzyme inhibitors are evidently not functioning adequately and fail to prevent the enzyme from attacking the lung elastin.

One of the most important serum inhibitors is $alpha_1$-antitrypsin and in the hereditary disorder, $alpha_1$-antitrypsin deficiency, the serum inhibitor level is only 10-20% of normal. This condition is strongly associated with emphysema, but accounts for 5% of the cases at most. In emphysematous patients with normal serum $alpha_1$antitrypsin, the nature of any predisposition remains quite obscure.

Treatment

Any patient who continues to smoke should be advised to abandon the habit entirely and if successful, there is a good prospect that sputum production, if present, will diminish and that chest infections will become fewer and less severe. The lung damage that has already occurred cannot of course be reversed to any extent, but there is evidence that the rate of deterioration will be slowed up.

Bronchodilator therapy

Drugs such as salbutamol, terbutylene or ipratropium bromide are usually effective given as aerosols (see lung function tests and table 1).

Surgery

The best results are obtained where there is a large bulla (or collection of bullae) compressing other parts of the lung (see Fig 2). The bulla can be removed completely or, in the more disabled patients, decompressed by insertion of a tube connected to an evacuation pump. A successful result depends on re-expansion of the compressed part of the lung and if this occurs, the patient may obtain considerable relief from shortness of breath.

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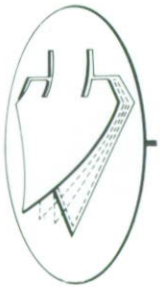

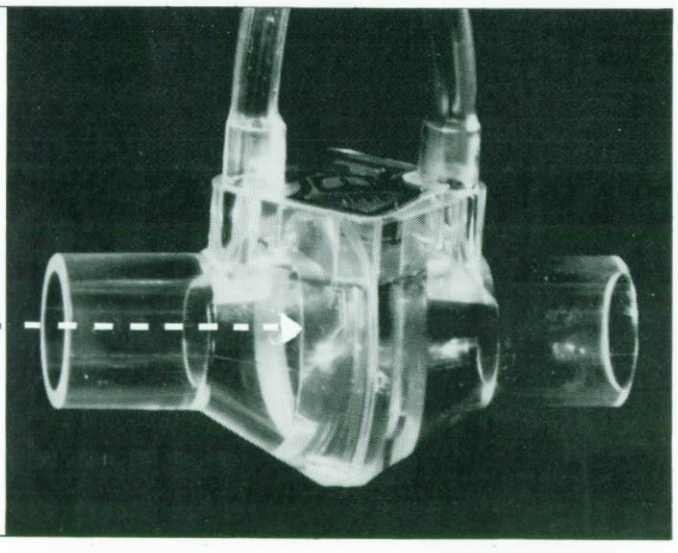

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ANNUAL GENERAL MEETING OF THE ASSOCIATION

The Annual General Meeting took place on Saturday, 4 October 1980 at the Walsgrave General Hospital, Coventry.

We are most grateful to Lynne Clarke who organised the meeting, to the speakers for their interesting papers and to Dr W E Zundel who kindly took the chair for the scientific session.

We are much indebted to the following firms for generous sponsorship of the meting and for putting on demonstrations of their products:

Beckman RIIC Limited
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The following scientific papers were given during the morning session:

1. Bronchial reactivity to inhaled histamine in asthmatic patients.

Dr G Jones Chest Physician, Walsgrave Hospital

Bronchial hyper-reactivity is one of the main features of the asthmatic patient and narrowing of the airways may result from a wide variety of stimuli, including allergens, cold air, dust, smoke, anxiety, coughing, overbreathing and exercise. The histamine challenge test might be regarded as rather a specialised procedure with limited applications but Dr Jones and the laboratory staff gave us an excellent demonstration of how simple this can be — with the kind cooperation of one of their patients. Dr Jones suggested that this could be used as an addition to our routine tests in selected cases, for diagnosis and for assessment of drug requirements.

2. The intra-breath behaviour of radioactive tracer gas in the lung.

David Hamilton Physicist

Isotope imaging of the lung has been greatly improved since the introduction of Krypton-81m, a gamma-emitter with a half-life of only 13 seconds. The method is generally used simply to obtain a visual display of the distribution of ventilation within the lung but Mr Hamilton demonstrated with the aid of some elegant mathematics, how a breath-by-breath analysis of the activity in sub-sections of the lung would yield very much more information. Not a routine method just yet!

CHAIRMAN'S REPORT

Once again, I am making my yearly contribution to Breath.

Yes, it does take me nearly a year to put pen to paper and produce something in writing, but I hope that all members will agree that over the last few months, the Executive Committee has tried to keep you all informed of the business of the Association via the "new-look" Breath and the broadsheet 'In-between Breaths'.

How far forward, in the last year, has the Association moved towards its objectives as set out in the Constitution?

I hope that we all strive to promote advances in the diagnosis and treatment of patients with respiratory disease, and to further the aims of research in this field.

The Association has continued to hold meetings for its members, free from a registration fee, due to the goodwill of many firms dealing in respiratory equipment and the continuing generosity of hospital caterers and administrators. I would also like here, to take the opportunity of thanking those members concerned with the organisation of the Scientific Meetings over the last four years.

Promotion of educational and training programmes for members of the Association must, I think, be one of the most time-consuming and difficult tasks as well as one that takes up a considerable amount of room in the filing cabinets of those concerned with it. The paper work accumulating on Education is now reaching phenomenal quantities, all of which has to be mulled over, digested, discussed and then, in some cases, discarded to the back of the filing cabinet and a fresh start made. But beware of destroying it, as some Committee or another will refer to a two-year-old document at some future date.

What is happening in Education at the moment?

O Tec syllabus is starting in some colleges, but without recognition of the in-service training programme for students. Discussions are still taking place on the most appropriate way of assessing students' abilities to perform basic routine measurements — should this be during the course of in-service training on a log-book basis, or at the end of training by a practical examination? For members interested in educational developments a report can be obtained from Regional Scientific Officers:

Department of Health and Social Security Working Party: Training in Medical Physics and Physiological Measurement within the Tec System.
Final Draft Report — October, 1979.

Higher Education is still very much in the early discussion stages (this does not mean any less paper work), but consideration must now be given to what is required for the good of the patients, the service and the technicians. Should it be a higher academic qualification or a more technical, professional one?

What is required from the membership now is feedback of ideas and views on the way in which they would like to see Higher Education develop. Without some indication from the membership, those dealing with Education or for that matter, Association policies, can only assume that they are carrying out the wishes of the members, and continue to follow the initial path chosen (ideas and views change — bounce back and forth, and your Executive needs to know how to express the opinions of the majority as well as giving careful consideration to the minority point of view).

How far has the Association moved over the last year towards improving career opportunities and conditions of service for its members?

Up until the last few weeks — not very far at all, but during the early summer the DHSS approached the Association with regard to the various grades in which technicians in Respiratory Physiology are employed. The results of the survey are listed below and on behalf of Margaret Marples, I would like to thank all the members who returned the questionnaire. As a result of the survey,

two other members of the Executive Committee — Margaret Marples and Derek Cramer — and myself attended a meeting on 30th September, 1980 at Hannibal House to meet DHSS representatives. The DHSS members were Dr. Bourdillon, Professional Adviser to the DHSS for Physiological Measurement, Mr. Godffrey, a representative for Medical Physics and two Whitley Council representatives.

Some discussion took place on the appropriate grading structure for Respiratory Physiology technicians, and the general opinion of the DHSS was that the appropriate grade for Respiratory Physiology technicians was Physiological Measurement. ARTP representatives were then asked for their views on the developments for the future. The answer from the ARTP representatives was in three parts:

1. Recognition for Respiratory Physiology technicians, i.e. the Physiological Measurement grade should *either* include Respiratory Physiology, *or* should refer to no disciplines by name.
2. Abolish the so-called Certificate of Competence and put in its place a nationally recognised education qualification for entry to technician grade.
3. Parity on grading and pay with the Medical Physics structure.

These views were received by the DHSS representatives sympathetically, but it was pointed out that there would be difficulties with some of the other disciplines in implementing them. In fact, discussions have been held up on the Senior Chief Grade due to disagreement with one discipline.

The general impression brought away from the meeting was that for both sides it had been a good public relations exercise, but for our part, it was up to us to push forward to achieve the recognition we required for our members.

How can this be done?

One way is through the FAMT. Twice over the last four weeks the Federation has been referred to by members of the DHSS as the professional body for Physiological Measurement/Medical Physics Technicians in the NHS with whom they wish to negotiate. But again, the FAMT needs to be fed with ideas from the ordinary membership of each of the eight individual Societies and Associations. It is time for the younger members to come forward and serve on the Executive Committees. Otherwise, I fear, that your futures will be decided for you by others who are more than half-way through their careers and who are looking for a comfortable niche in which to end it. Your future professional careers should be in your own hands and you should all be giving some consideration to the way in which you wish them to develop and be making these views widely known.

SURVEY OF TECHNICIANS' GRADING 1980

PHYSIOLOGICAL MEASUREMENT TECHNICIANS

Chief Technician	15
Senior Technician	36
Basic Grade Technician	57
Student Technician	23
TOTAL	131

MEDICAL PHYSICS TECHNICIANS

Grade I	1
Grade II	3
Grade III	9
Grade IV	17
TOTAL	30

OTHERS

Chief M.L.S.O.	1
Senior M.L.S.O.	2
Senior Scientific Officer	2
Research Officer	2
Principal Physiologist	1
TOTAL	8

Over the next year, I would like members to give some careful thought to the Rules of the Constitution. It becomes increasingly difficult to observe these rules with reference to the Council of the Association, due to lack of full representation of Council Members at the twice yearly meetings required by the Constitution. The Executive Committee has in some cases broken the Rules of the Associaion. This has occurred with Rule 4 (a) which states that all applications for membership shall be subject to the approval of the Council; this approval has been granted over the last two years by the Executive Committee, due to lack of full representation at Council Meetings. I feel that the time has come to give close consideration to the various rules governing the Association, and to look how the business of the Association can best be served. The Constitution has hitherto served us well, but the Association is developing and it could possibly even be hindered in this development by its own Constitution.

At the Council Meeting before this year's Annual General Meeting at which the stated quorum of eight was satisfied, the Council agreed to the request of the Executive Committee to invite *Philip Morgan* to become the first Honorary Member of the Association. It was felt that this was the most appropriate way in which the Association could express its thanks to Philip Morgan and his staff for the continuing support he gives to Scientific Meetings, and his generosity over the last four years to the Association.

I felt the time had arrived for me to step down as an Officer of the Association and to take a supporting role. *Derek Cramer*, who has for a number of years taken an active part in the business of the Association, and has worked in the NHS for many years (most of the time in Respiratory Physiology) agreed to stand and was elected the new Chairman of the Association.

I would like to take the opportunity in this report of thanking *Margaret Marples* for her support as Secretary. Margaret is resigning as Secretary for personal reasons, but like myself, will continue on the Executive Committee, mainly because of lack of impetus from other members of the Association.

At the time of writing this report, the Executive has one new member, *Sue Hill* from Birmingham, and one vacancy, but otherwise it is just a re-shuffle from last year. There must be, amongst our 150 members, one or more who wishes to work for the good of the Association. This does involve you giving up some time, but it is the Association policy to try to ensure that Executive Members are not financially embarrassed by accepting office.

It is always difficult to find the right words or sentence to end a report. I will end this one with best wishes for the forthcoming year to Derek Cramer, Gillian Lowe as the new Secretary, and to Janie Jones, Treasurer, who I hope will continue to keep the Association's finances secure, and a vote of thanks to members who continue to support the ARTP, and to the Association itself for the many new friends I have made over the last four years.

TREASURER'S REPORT

AGM 4.10.80

I should like to present the Treasurer's Report for the period 1.9.79 up to date. The usual procedure is to present the report from April to April, but as I have decided to have the accounts audited, I thought you would like to see how our financial position is to date.

As you can see our account is fairly healthy. Membership subscriptions are up on last year, and our income from advertisements, either in BREATH, or from circulation to members, has increased considerably. We have also increased the amount received from firms for exhibiting at our meetings.

Our chief expenditure is in postage and stationery and travelling expenses to Executive Committee meetings. Unfortunately this may necessitate increasing the membership fee for next year. I am sorry to have to request this but we have to keep up with rising costs and the subscription has not increased since the Association started.

The Bank Charges were incurred because BREATH was paid for before all the advertising fees were paid to us. It was decided at the Executive Meeting to leave the deposit account alone as it was accruing interest.

The Accounts are now being audited, and when I receive the auditors' report, this will be circulated to all members.

May I also finally ask all members to attempt to pay their subscriptions on time.

Jane Jones, Treasurer, ART&P

INCOME AND EXPENDITURE ACCOUNT FOR THE PERIOD 1.5.79 to 30.9.80

EXPENDITURE		INCOME	
Travelling Expenses	£ 565.04	Membership Subscriptions	£1108.00
Catering Costs	£ 438.27	Exhibitors Donations	£ 425.00
Stationery & Postage	£ 232.29	Advertisements	£ 795.95
FAMT Subscription (two years)	£ 40.00		
BREATH (two issues)	£ 832.72		
Bank Charges	£ 20.30		
Badges	£ 132.37		
Miscellaneous	£ 10.00		
TOTAL	£2271.99		£2328.95
Excess of income over expenditure	£ 56.96		
BALANCE	£2328.95		£2328.95

ASSETS IN BANK AT 3.10.80

Deposit Account	£ 507.14
Current Account	£ 416.47
TOTAL	£923.61

ASSISTANT EDITOR'S REPORT

I am still pushing for articles for BREATH. If you have noticed a reponderance of articles from the London Chest Hospital, this is not entirely a coincidence! I think that some people feel shy about asking the medical staff to write for them, but I have found that far from being refused, the doctors I have asked have been very interested and have also suggested colleagues who would provide interesting material. In fact I had the pleasure the other day of seeing an article in BREATH on a curriculum vitae. I felt very proud!

So please, if every member provided one article, we would have enough material for a lot of issues. I also think it would be interesting to have material from people such as physiotherapists, nurses and anyone else concerned with the care of respiratory patients.

A FEW EXCERPTS TO BOOST MY EGO! (and maybe encourage you)

"I find the news bulletins and job vacancies posted to me as a member of ARTP very useful, and always enjoy reading BREATH."

Y. Nichols, Bury General Hospital

"Although I do not manage to attend meetings, I want to remain a member in order to keep in touch with training schemes, etc., and to receive BREATH. I hope in future H. Tec will be available at more polytechnics. I'm sure many people would do the course if it were nearer to them."

M. Smyllie, Doncaster Royal Infirmary.

And continuing on this more serious note is a letter from Dublin.

"The situation in Ireland with regard to Pulmonary Function Technicians is akin to the type of situation technicians in England experienced some years ago. We are anxious to be recognised in our own right and to be given some type of educational system, which would suit our needs. At present these are much neglected.

Geraldine Lawless, St. Vincent's Hospital, Dublin

As the next issue of BREATH will probably not be until January, I shall take the opportunity of wishing you all a very premature Merry Christmas and a Happy New Year.

REMEMBER — JUST ONE ARTICLE EACH!

Jane Jones,
Assistant Editor

ARTP NEWS

Jane Jones

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Jane Jones,
Assistant Editor

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Closing date — 21st November, 1980

Breath is the journal of the Association of Respiratory Technicians and Physiologists. Original articles, reviews, correspondence or comment on subjects of scientific or general interest may be submitted to the Editor: D C S Hutchison, Chest Unit, King's College Hospital, London SE5 8RX. Material should preferably be typed on one side of the paper only, in treble spacing throughout. Photographs should be of good contrast, printed on glossy paper and unmounted. Tables and legends to figures should be typed on separate sheets.

Applications for advertisement space and for rates should be addressed to: Jane Jones, Respiratory Laboratory, London Chest Hospital, Bonner Road, London E2.

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