ASSOCIATION OF RESPIRATORY TECHNICIANS AND PHYSIOLOGISTS

ISSUE NO. 21 FEBRUARY 1984

BREATH

CONTENTS

SPRING MEETING OF THE ASSOCIATION and TRAINING WORKSHOP

Post-graduate Medical Centre,
Stoke Mandeville Hospital,
Aylesbury, Bucks.

April 6th and 7th, 1984

Preceding the Spring Meeting of the Association at Stoke Mandeville Hospital, there will be a follow-up to the training workshop held last year. We hope that those of you who are involved in supervising or teaching on the in-service training programme will be able to attend. The Education Committee will be very interested in your comments and experiences.

EDITORIAL

Pollution, pollution...

...wear a gas mask and a veil,
then you can breathe, as long as you don't inhale.
Tom Lehrer.

Many of the agents which cause human disease enter the body by way of the respiratory tract and we may include among their number the large variety of micro-organisms, allergens and occupational dusts or fumes. The term air pollution, however, usually refers to a more general contamination of the atmosphere by smoke from burning fuel and has, in modern times, been applied to a variety of less visible but often more hazardous substances. We do not, curiously enough, generally include our own personal form of air pollution, brought about by inhalation of the smoke of smouldering, dried tobacco leaves.

In medieval times, 'sea coal' brought to London by ship from Tyneside became increasingly used for domestic heating, in part because of a growing shortage of wood and charcoal. In the 13th century a law was passed in an attempt to limit the serious air pollution which had already made itself obvious through this cause. The situation dragged on with intermittent but largely ineffective public debate until the culminating event, the great 'smog' of 1952. The term 'smog', used to indicate a toxic mixture of smoke and fog, has certainly been in use for over 70 years and has been an accepted feature of London life for centuries and of other large cities since the start of the Industrial Revolution. The 1952 smog lasted for no more than five davs but in that short space of time brought about a serious epidemic of respiratory disease which caused, over the next few weeks, between four and five thousand deaths in the London population, mainly among the elderly and those with chronic lung disease. The ensuing public outcry resulted in the Clean Air Act of 1956 which forbade the burning of soft coal in open grates for domestic heating. We naturally still see the ordinary type of fog now and again, London weather being what it is, but the 'killer' smog happily seems to have disappeared. Along with this the mortality and morbidity of chronic obstructive bronchitis and related disorders has fallen steadily over the last fifteen years. London is now a reasonably pleasant place to live in (some Londoners claim).

Is all well then? Not quite — since the Clean Air Act much of the coal burned in this country has been used for industrial purposes and in order to minimise local air pollution the smoke is dispersed through chimney stacks which may reach a height of 800 feet. The smoke (containing a substantial quantity of sulphur dioxide which is too expensive to remove) is then carried by the prevailing wind to fall as acid rain in Scandinavia, to the severe detriment of animal and plant life in that region and the impotent chagrin of the inhabitants.

A new and different form of smog has more recently been created by the fumes emitted from motor vehicles. The problem results from the action of ultraviolet light on oxides

of nitrogen and unburnt hydrocarbons, producing ozone and other irritant compounds which can exacerbate and perhaps even initiate chronic airflow obstruction. This 'photochemical smog' is particularly severe in Los Angeles; strong sunlight and stable weather conditions are basic requirements but even so this type of air pollution has appeared in London on occasions.

Petrol engines also produce a substantial quantity of carbon monoxide, a potentially toxic compound because of its strong affinity for haemoglobin. However, the blood levels encountered in pedestrians and drivers do not constitute a hazard and are, in any case, far lower than those achieved by the average cigarette smoker.

Organic lead compounds are usually added to petrol to improve engine performance and most of this emerges in the exhaust fumes as inorganic lead. Now lead in high concentrations is an extremely toxic substance, causing (among other effects) intellectual deterioration and behaviour disorders in children. The issue, which has caused keen public debate, is whether the lower quantities of lead derived from exhaust fumes and ingested by the oral or respiratory routes can produce these symptoms. The most recent investigation (but surely not the last) is the large joint study on lead levels in children's teeth from the Institute of Child Health in London and Southampton University. Only weak relationships between tooth lead and behaviour or intelligence could be demonstrated and the lead level was more closely related to 'social' factors such as education, environment and parental attitudes. A Royal Commission has recommended the removal of lead from petrol, which seems sensible advice in the light of public concern. The main danger in fact probably comes from lead in old paint, which often contains high concentrations.

A similar scene has developed in the nuclear industry where there is the potential for release of even more insidious toxins into the environment. The radioactive element plutonium and related nuclides somehow find their way into the atmosphere and have been isolated from soil and household dust near nuclear plants in the United States and at Sellafield — the name disguises our old friend Windscale. Plutonium, as an alpha particle emitter is a potent carcinogenic agent; cancer of the bone marrow (leukaemia) is a particular hazard since plutonium is readily deposited in bone and may stay there for life. Reports have indeed emerged which claim to link various forms of bone cancer with environmental levels of plutonium but again opinion is divided on the question.

The problem about the postulated lead and plutonium risks is that it difficult to evaluate what is occurring when the disease may not develop until many years after exposure to the original stimulus. We have seen the same sequence of events in relation to asbestos, as recently discussed in these columns. The situation was quite different in the 1952 smog epidemic where one could see the damage occurring before one's very eyes.

How should one deal with these difficult problems? One thing is clear — the public must be informed and there can be no question of treating the issues solely as a series of academic debates; one should not have to wait years for the results of these social experiments to yield statistically significant results. In the case of lead pollution, the right decision has been taken but only after acrimonious debate. The fact is that we have not yet devised a method for providing the public with information in a form which it can readily assimilate and which, above all, originates from a body it can trust.

Perhaps Tom Lehrer's nightmare hasn't quite come true, but *Breath* researchers have already hinted that on the day plutonium starts to appear in their own household dust, they'll start looking for other accommodation.

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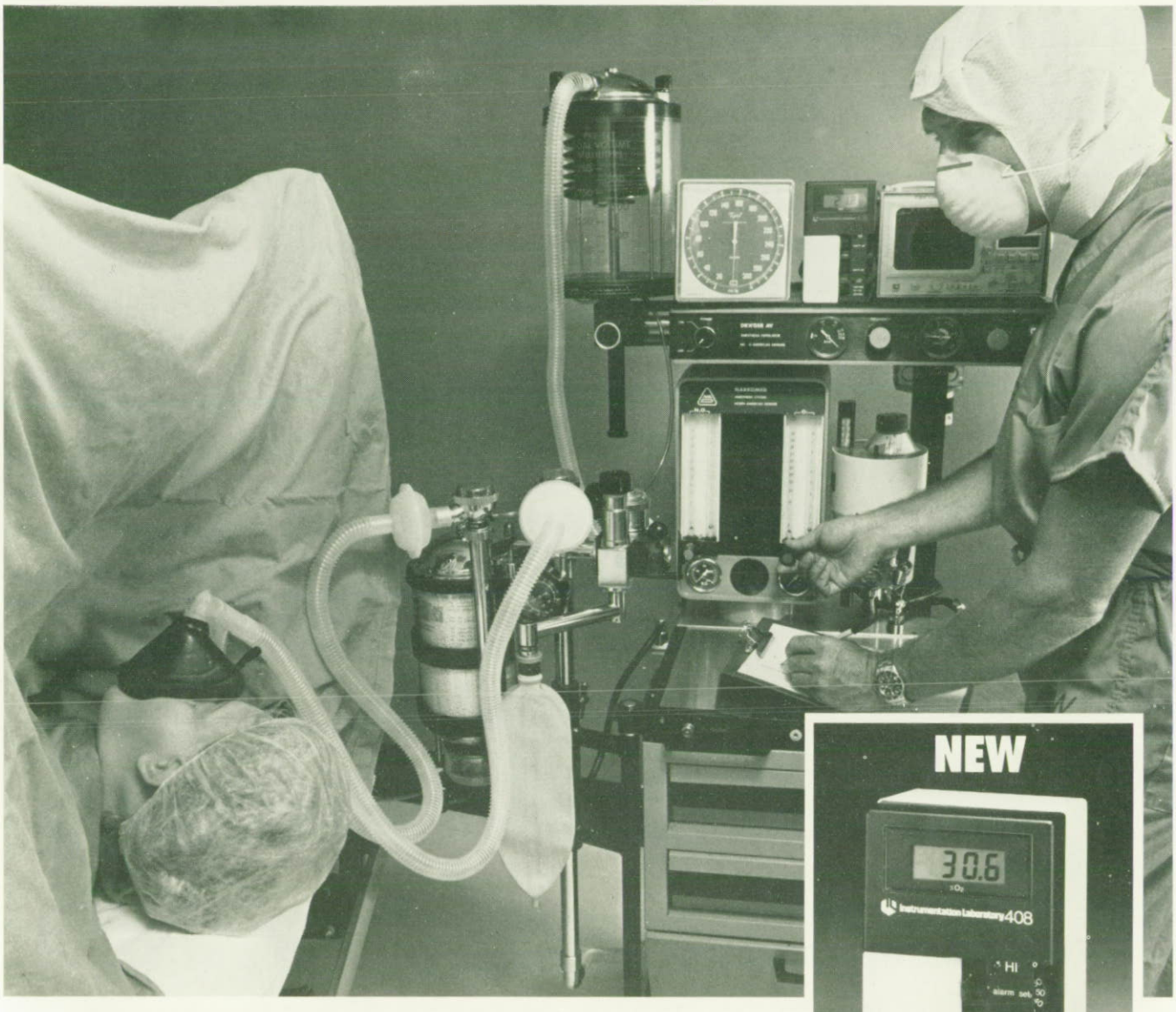

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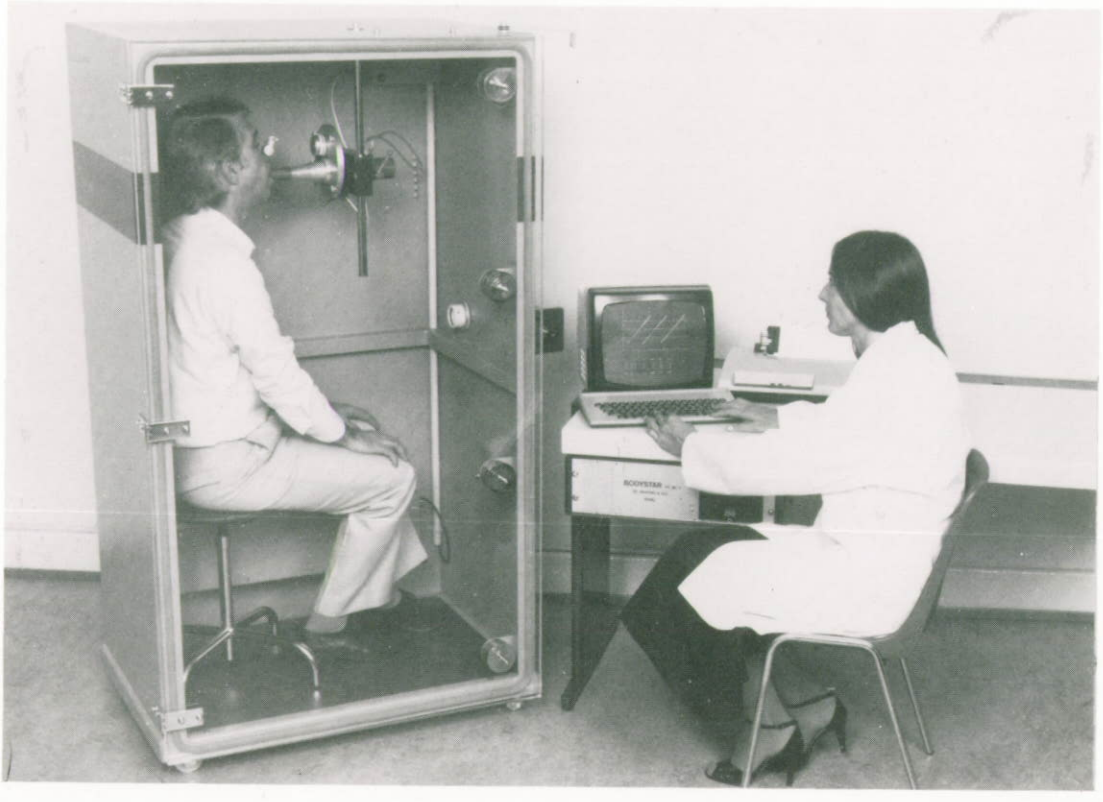

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AN EVALUATION OF THE LA3 – MK 2 LUNG FUNCTION ANALYSER: A new portable electronic spirometer.

K A Gunawardena and A P Smith
Llandough Hospital, Penarth, South Glamorgan

Summary

A compact, British-made electronic spirometer, which measures in a single expiration the PEFR, FVC, FEV_1 and FEV % (FEV_1/FVC) was compared with the Vitalograph and the Wright peak flow meter (PFM) in 58 subjects — (FVC range 0.85 to 5.8 litres; FEV_1 range 0.4 to 4.95 litres; PEFR range 80 – 630 litres/min.). For FEV_1 and FVC the correlations were high (r = 0.992 and 0.991 respectively) and the line of identity was within the 95% confidence limits. For PEFR correlation was high (r = 0.982) but the slope of the regression line (0.825) deviated significantly from unity, with a positive intercept of 35.8 litres/min; this meant that the LA3 under-read by over 15% at high flow rates. Judged by the American Thoracic Society standards for spirometry the resistance of the LA3 was too high and this probably accounts for the low readings for PEFR at high flow rates. The FEV % displayed by the machine showed a systematic error, tending to over-read at low values. Both these faults could be overcome with slight modifications to the design of the instrument.

Introduction

Indices derived from a forced expiration, such as FVC, FEV_1, FEV % and PEFR are the commonest tests of pulmonary function used in clinical and epidemiological practice today and are often used in clinical trials of bronchodilator drugs. A compact portable instrument which is easy to operate and yields the commonly used indices in a single breath would therefore serve a useful function . The LA3 – Mk. 2 Lung Function Analyser *(Mercury Electronics, Glasgow)* is such an instrument and we compared the performance of this with the Vitalograph and the Wright PFM, which are widely used and whose reliability has been established.[1,2,3]

Operation and description of the instrument

The LA3 (Fig 1) weighs 4.5 kg, measures 39 × 48 × 16 cm and is mains powered. The instrument is allowed to warm up initially for about 10 mins and is zeroed by pressing "RESET" button. The subject is given the tube and the mouth-piece and is instructed to perform a forced expiratory manoeuvre, while the operator presses and holds the "TEST" button. The PEFR (range 0-1000 litres/min), FVC and FEV_1 (range 0-10 litres) and FEV % (range 5-100%) are then read off the digital meter by pressing the appropriate button.

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Fig 1. The LA3 Mk 2 Lung Function Analyser.

The instrument measures flow through an unheated pneumotachograph and integrates this flow to provide a volume measurement. The flow first passes through a dust filter and then through a fine wire gauze held in plastic rings. The pressure developed across the gauze, which is proportional to flow, is measured with a transducer. The output from the transducer is also taken to a sensitive switching circuit which connects the flow output to a peak sensing device and to an integrator. A flow as low as 15 litres/min is sufficient to switch this circuit. During the first second of expiration a "sample and hold circuit" follows the integrator output and then disconnects from it leaving a signal equivalent to the volume expired during the first second. A divider circuit continuously computes the output of the "sample and hold circuit" as a percentage of the total volume expired. The PEFR, FEV_1, FVC and FEV % are stored ready to be read off the digital meter.

The machine also provides output sockets for flow and volume and a switch to allow inspiratory flows to be recorded. By connecting these outputs to a fast response XY recorder a full flow volume loop can be obtained. Alternatively, the volume output can be connected to a YT recorder to give a spirographic curve. The instrument can be easily calibrated by the potentiometer provided at the back of the unit. As the relationship between flow and volume is fixed only one needs to be set. The unit costs £760.

Methods

The LA3 – Mk 2 Lung Function Analyser (loaned by the manufacturers) was compared with two Model S Vitalographs and two standard Wright Peak Flow Meters that were in routine use in the lung function laboratory. Volume calibration was effected with a 1.0 litre syringe so that the volume displayed on the LA3 agreed to within 5% of the volume indicated on the ATPS scale of the Vitalographs. The two peak flow meters used gave readings agreeing to within ± 5% when tested on several normal subjects.

The subjects tested were drawn from the staff or from among the out-patients attending the chest clinics at Llandough Hospital. After the subjects had become familiar with the instruments each provided three readings with the LA3 and three with either the Vitalograph or the PFM. FVC, FEV_1 and FEV % data were obtained from 34 subjects while PEFR data were obtained from 24 subjects. In each study the order of using the instruments was alternated so that the results were not biased by the effects of training or fatigue. The best as well as the average, of three satisfactory readings were used for comparisons. For FVC and FEV_1 comparisons, volumes indicated on the BTPS scale were used.

Results

Among the subjects tested the FVC ranged from 0.85 to 5.8 litres, FEV_1 from 0.4 to 4.95 litres, FEV % from 33% to 96% and PEFR from 80 to 630 litres/min. The best of three satisfactory readings correlated slightly better than the average of the three readings. The following regressions and correlations therefore refer only to the best readings.

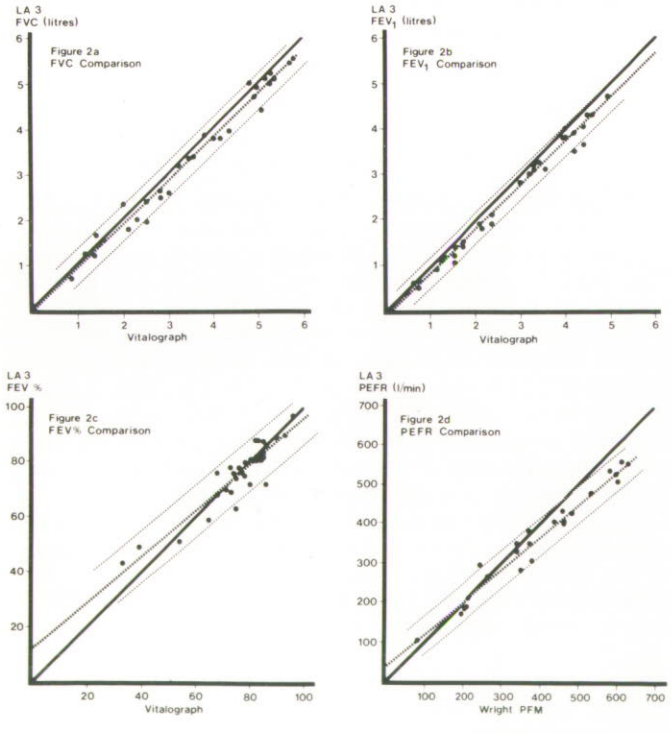

Fig 2. Comparisons of the measurements of (a) FVC – LA3 versus Vitalograph, (b) FEV_1 – LA3 versus Vitalograph, (c) FEV % – LA3 versus Vitalograph and (d) PEFR – LA3 versus Wright's peak flow meter. Continuous lines: line of identity; dotted lines: Regression line and 95% confidence limits: 95% limits: FVC 0.41 l. FEV_1 0.33 l. FEV % 9.5%. PEFR 49 l/min.

FVC and FEV_1: (Figs 2a and 2b). Linear correlations were high (r = 0.991 and 0.992) with the regression coefficients close to unity (0.962 and 0.956) and intercepts approximately zero (-0.014 and -0.116). The regression equations were calculated from the results of only 33 patients as one asthmatic who clearly developed bronchospasm between the two tests was excluded from statistical analysis.

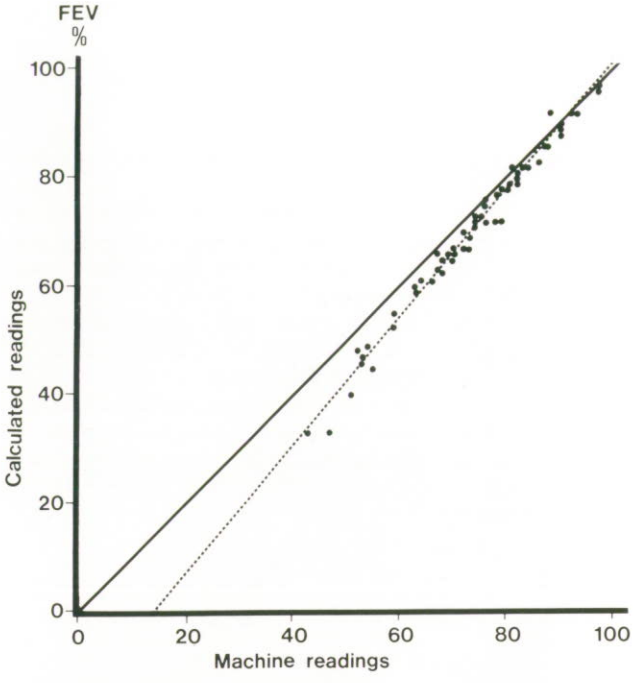

Fig 3. Comparison of the FEV % shown on the machine with FEV % calculated from FVC and FEV_1 displayed by the machine.

FEV %: This was obtained from the test that gave the best values for FVC and FEV_1. Fig 2c shows the FEV % displayed by the LA3 and the FEV % derived from the Vitalograph tracing. Although the correlation was good (r = 0.919) the slope (0.836) and the intercept (+ 12.11) deviated widely from the line of identity. The FEV % calculated independently from the FVC and FEV_1 readings displayed by the LA3 was closer to the line of identity (slope 1.015, intercept -4.973, correlation coefficient 0.911), demonstrating the inability of the machine to process its own results correctly. Fig 3 shows the relation between the FEV % shown on the machine and the FEV % calculated from the FVC and FEV_1 displayed by the machine. There appears to be a systematic error, the LA3 tending to over-read by almost 15% at the lower values of FEV %.

PEFR: (Fig 2d). Although the correlation was high (r = 0.982), the slope (0.825) and the intercept (+ 35.8) were unacceptable, with the line of identity lying beyond the 95% confidence limits at peak flow rates over 500 litres/min. At these values the LA3 under-read by 10-15%.

Comments

While electronic spirometers offer considerable advantages over water-filled or bellows spirometers in terms of portability and ease of operation, they have often been found to be seriously inaccurate. Fitzgerald et al[4] evaluated four electronic spirometers and found that none were accurate enough to be recommended for routine use. Two previous versions of LA3 were also found to be unsuitable[5].

The present version of the LA3 is a considerable improvement on the previous models. When compared with the Vitalograph its performance in a clinical setting was good as regards the FVC and FEV_1, but the FEV % displayed by the machine was still not sufficiently accurate. The machine does not compute the FEV % mathematically from the final FVC and FEV_1 readings but instead uses a "divider circuit" which probably accounts for the error. This fault could be easily rectified by using a microprocessor but this would increase the cost of the instrument.

Table 1

Back pressures developed by the LA3 at different flow rates

Flow Rate (litres/min)	Pressure without filter (mm H_2O)	Pressure with filter (mm H_2O)
100	5	18
200	13	44
300	22	76
600	62	220

(Figures supplied by the manufacturer)

The peak flow rates obtained with the LA3 correlated well with the Wright PFM readings and their repeatability was good but as with the previous versions[5], LA3 under-read by 10-15% at high flow rates. This could be due to the high resistance offered by the dust filter which is made of a fine mesh identical to the screen used in the pneumotachograph. Table 1 shows the back pressures developed at various flow rates with and without the dust filter. The filter almost trebles the resistance of the instrument. The "minimal spirometry standards" laid down by the American Thoracic Society[6] requires the resistance to be less than 1.5 cm of water/litre/second at a flow of 12.0 litres/sec. This level is exceeded by the LA3 even at a flow rate of 10 litres/sec. The high resistance of the LA3 is also commented upon by Shaw and Fisher[7] who found it impossible to generate a flow greater than 8.0 litres/sec into the instrument with the flow calibration they used. Clearly, the instrument should have a dust filter of lower resistance or have the peak flow readings re-calibrated to give about 15% higher readings without affecting the volume calibration. Either or both of these alterations should not be too difficult for the manufacturers.

Conceptually, the design of the LA3 is good. In effect, it combines a Vitalograph (with a function analyser), a standard and a low reading Wright PFM. It could easily be used as a desk-top instrument in a busy clinic. With extra attachments its use can be extended to graphic recordings of the volume-time curve or the flow-volume curve. However, it still has a few irritating drawbacks and if these were rectified it could be recommended for wider use.

Acknowledgements

We thank the staff of the Lung Function Laboratory, Llandough Hospital, Penarth, for their help.

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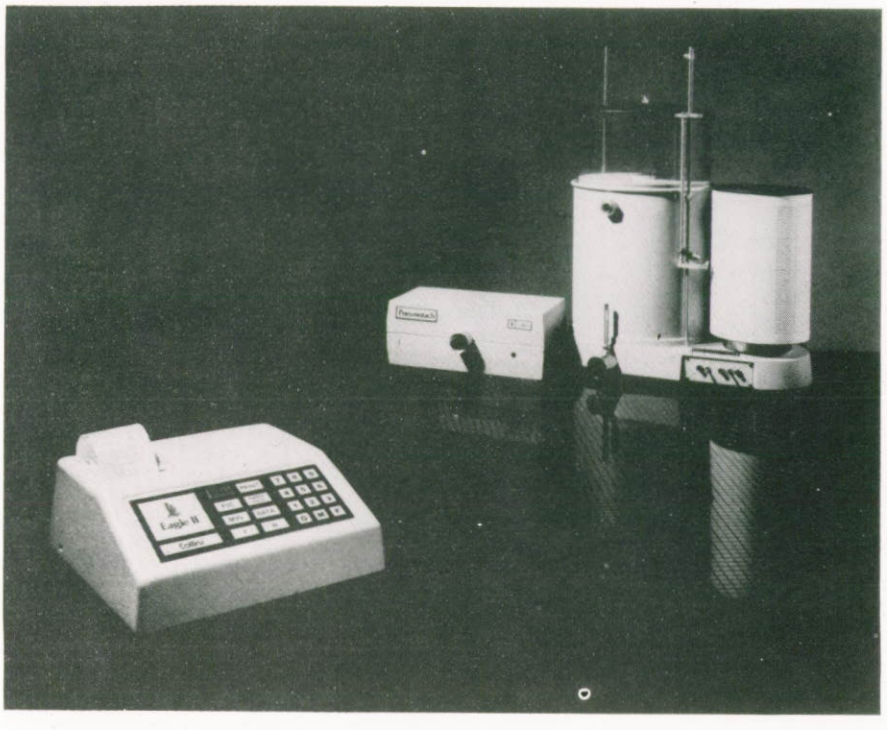

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BRONCHIAL REACTIVITY: its assessment by the histamine challenge test

M. J. Walshaw

Royal Liverpool Hospital

Introduction

Bronchial hyper-reactivity is defined as 'the abnormally increased response of bronchial smooth muscle to stimuli'. A stimulus such as cold air or dust which would produce no detectable airway changes in a normal individual, may produce a marked degree of bronchospasm in an individual with increased bronchial reactivity. Such hyper-reactivity occurs in most asthmatic patients (both extrinsic atopic and intrinsic non-atopic types), less commonly in those with chronic bronchitis and transiently in normal individuals.

Aetiology

The mechanisms governing increased bronchial reactivity are not well understood: many theories have been formulated[1] but none have achieved popular acceptance.

In 1921, Alexander and Paddock[2] showed that pilocarpine injections caused 'asthmatic breathing' in asthmatic patients, but not in normal controls; adrenaline relieved the symptoms. Pilocarpine is a parasympathetic nervous system stimulator and adrenaline opposes its action by stimulating the sympathetic system; this work led to the suggestion that increased bronchial reactivity could be related to autonomic nervous system imbalance. In 1932, Weiss and his colleagues[3] showed that histamine infusions could produce bronchospasm in asthmatic patients but that even quite large doses had no effect on normal individuals.

Histamine is not thought to have any action on the autonomic nervous system, but is known to be a powerful vasodilator produced by the body as part of the acute inflammatory response. However, its action at the bronchiolar level is not thought to be inflammatory in nature since the concentrations needed are very small and the onset of bronchospasm is very rapid, features which are much more in keeping with a nervous reflex action than with chemical inflammation. Many similar substances have now been shown to produce bronchospasm. Examples bring serotonin, prostaglandin F_2 and bradykinins.

Other factors may also play a part in altering bronchial reactivity. Under conditions of laminar flow, airway resistance is inversely proportional to the fourth power of the radius. Thus, halving the radius will increase the airway resistance proportionally by a factor of 16. It is suggested that some asthmatic patients (and certainly most chronic bronchitic patients) have a degree of bronchial oedema and smooth muscle hypertrophy which will decrease resting bronchiolar calibre. A small degree of bronchospasm will therefore produce a large increase in airway resistance but this cannot explain the increased bronchial reactivity found in asthmatics with no pre-existing airway narrowing, nor that seen in normal subjects.

Bronchiolar epithelial damage has been suggested as a contributory factor, in that it might allow subepithelial vagal nerve endings and other receptors to become sensitised. This is analogous to a cavity in a tooth; exposure of the nerve root causes it to become hypersensitive, and then any stimulus (a cup of tea for instance) will cause severe pain. However, when there is no cavity the same stimulus produces little reaction. It is not clear whether bronchiolar epithelial damage does occur in all asthmatics but any epithelial gaps will certainly allow ingress of inhaled materials giving easier access to subepithelial target areas.

Alteration in bronchiolar smooth muscle may be important; those patients who do have smooth muscle hypertrophy will experience a greater degree of contraction for a given stimulus since the muscle bulk is larger. However, the main factor in producing bronchial hyper-reactivity is the way in which bronchial smooth muscle reacts to a stimulus, though it is not clear whether this reaction is governed by autonomic nervous system imbalance, nerve ending sensitisation, alteration in bronchiolar calibre, increased muscle bulk or a combination of these factors.

Measurement

Whatever the cause of bronchial hyper-reactivity, its measurement is now well defined. In principle, the patient inhales a known dose of an irritant substance via a nebuliser and the effect on airflow is measured. The most commonly used irritant is histamine acid phosphate and the FEV_1 is used to assess its effect on airflow. Histamine acid phosphate is used in doubling concentrations (starting at 0.025 mg/ml), dissolved in phosphate buffered saline to maintain a constant pH since pH itself can alter bronchial reactivity. The definitive procedure for histamine challenge testing accepted by most centres has been devised by Hargreave et al[4], and the following description briefly outlines the procedure:

Before starting the test, it is necessary to standardise the inhaled dose of histamine; this depends upon nebuliser output and also upon the patient's breathing technique[5]. Nebuliser output is influenced by the driving gas flow rate and also by the construction of the particular nebuliser; it is thus necessary to calibrate each nebuliser so that the same output is obtained. Alternatively, the same nebuliser can be used for each histamine concentration. Within a fairly wide range, aerosol particle size does not seem to influence the test[5]. Breathing technique is important; there is a tendency for patients to hyperventilate when given a facemask and this will increase the inhaled dose. The patient is therefore asked to breathe gently through the mouth using normal tidal volume only.

The patient first inhales plain phospate buffered saline solution for two minutes and FEV_1 is measured at 30 seconds, 90 seconds and at every minute until the lowest value is reached; this is taken as the baseline value.

Solutions of phospate buffered saline containing doubling concentrations of histamine acid phosphate are then used, until there is a reduction in FEV_1 of 20% or more. The results can be expressed on a suitable graph (fig. 1), with the histamine concentration plotted as a logarithmic value. From this it is possible to interpolate the PC_{20}, the 'Provocation Concentration' of histamine which would cause a 20% reduction in FEV_1. Alternatively, the following equation can be used:

$\log PC_{20} = \log C + \log 2 [(20-A) / (B-A)]$

where A is % reduction in FEV_1 at histamine concentration immediately before 20% reduction in FEV_1, B is % reduction in FEV_1 at histamine concentration immediately after 20% reduction in FEV_1 and C is histamine concentration immediately before 20% reduction in FEV_1. In Fig. 1, A = 13, B = 35 and C = 0.2 from which PC_{20} = 0.25 mg/ml.

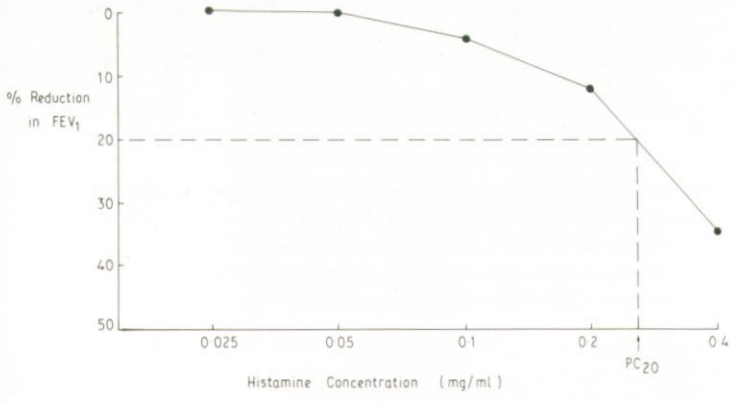

Fig. 1. *A typical Histamine challenge test, showing estimation of PC_{20}. PC_{20} = 0.25 mg/ml. (For calculation see text).*

The PC_{20} is used as an index of bronchial reactivity; the lower its value, the less histamine is needed to produce a given degree of bronchospasm and therefore the greater is the degree of bronchial hyper-reactivity. Fig. 2 shows the range of PC_{20} recorded in a group of asthmatic and non-asthmatic subjects at this centre; while there is a little overlap, the asthmatic patients tend to have a much lower PC_{20} (and hence greater bronchial reactivity) than the non-asthmatics.

Pitfalls

The technique of histamine challenge testing is quite straight-forward but there are several factors which can influence the results and have to be taken into account:

1. Time of day[6]: There is a diurnal variation in bronchial reactivity with an increase in the small hours of the morning; this is why asthmatic patients often present as 'morning dippers'. For repeat tests to be comparable, they must be performed at the same time of day.

2. Drug therapy[7]: Bronchodilators such as salbutamol and aminophylline oppose the action of histamine and will thus interfere with the test. Oral bronchodilators should be withheld for two days and the inhaled drugs for eight hours. Antihistamines should be withdrawn for two days. The effect of sodium cromoglycate and steroids is not clear but they are usually continued.

3. Cold air[8, 9]: This will increase bronchial reactivity probably by altering respiratory heat exchange. Patients attending for the test should be allowed to 'warm up' thoroughly, especially if they have entered the laboratory straight from outside on a cold day.

4. Viruses: Influenza vaccinations[10] have been shown to increase bronchial reactivity transiently in normal subjects and upper respiratory tract infections[11] seem to have a similar effect. It is therefore important to take a history of such episodes before performing the test.

Precautions

All our therapy is aimed at making asthmatic patients wheeze less but this test is specifically designed to make them wheeze more; for this reason the patient should be aware of the possible consequences of the test, and informed signed consent should be obtained. The side effects of histamine acid phosphate are few and are related to dosage; they are not usually a problem in the small concentrations used for asthmatic patients but can occur with the higher doses used for normal controls. Headaches, facial flushing, nasal discharge and a hoarse voice can occur and rarely hypotension due to generalised vasodilation in very large doses. The main potential danger is excessive broncho-spasm but if the test is carefully carried out, this problem should not arise. Resuscitative equipment should always be on hand however and the test should only be carried out under the close supervision of qualified medical personnel.

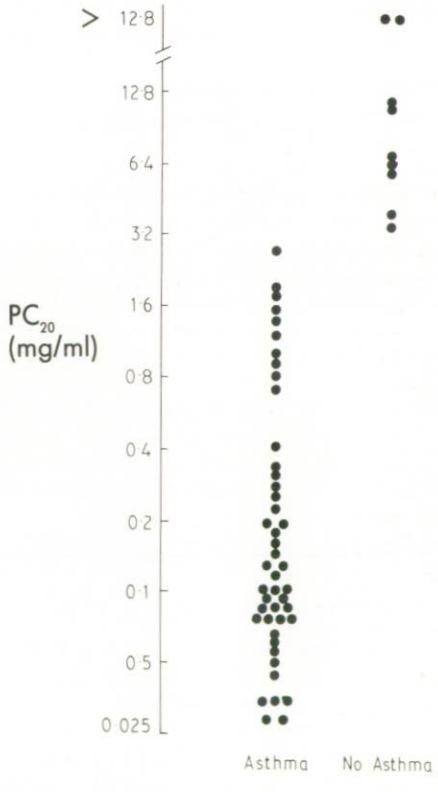

Fig. 2. *Range of PC_{20} in 46 asthmatic and 12 non-asthmatic subjects.*

Practical uses

Histamine challenge testing can be used to support the diagnosis of asthma in the patient who presents with unexplained cough or dyspnoea; if there is no clue from the history, physical examination is unremarkable and simple pulmonary function tests are normal, then the demonstration of increased bronchial reactivity makes the diagnosis of asthma likely. It is however, important to point out that a PC_{20} within the normal range does not rule out the diagnosis of asthma. Histamine challenge testing is more commonly used as a research tool in order to obtain serial measurements of PC_{20} on patients who are undergoing clinical trials which are designed to improve their bronchial reactivity.

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THE MAXIMUM VOLUNTARY VENTILATION

P. Lockwood
Harefield Hospital, Middlesex

The year 1983 sees the fiftieth anniversary for the introduction of the Maximum Breathing Capacity as a respiratory measurement[1]. In the early 1950s it gained in favour and at one time was recommended as the best single index of ventilatory function[2].It was considered necessary, in addition to the vital capacity, for showing the effects of lung resection[3] which was becoming more common at that time as pulmonary tuberculosis yielded to treatment.

Standardisation

There were several attempts to standardise the conditions under which the test should be carried out, in particular stipulating various rates of breathing to be adopted. The method eventually used most widely was that of Gilson and Hugh-Jones[4], in which the subject was instructed to breathe 'as deeply as you can, as fast as you can'. For this favoured technique, Bernstein and Kazantzis[5] recommended the term 'Maximum Voluntary Ventilation' (MVV) and this was adopted by the joint committee of the American College of Chest Physicians and the American Thoracic Society on nomenclature in respiratory physiology.

Many workers came to consider the procedure, if carried on for the required 15 to 20 seconds, to be too exhausting for many patients. Yet wide practical experience shows that patients will stop any procedure that they find too demanding. For subjects with chronic airflow obstruction the MVV can be less tiring than the forced vital capacity (FVC) which in fact may take longer to complete and being virtually a Valsalva manoeuvre may cause dizziness, flushing of the face and venous distension. Distress with the MVV is more often seen in severe fibrotic syndromes, where the gain in oxygen on hyperventilating may be offset by the increased oxygen requirement of the work of breathing maximally. Even so, however, recovery is very rapid after the completion of the test.

Substitutes for the MVV

From 1938 onwards, successive groups of workers sought to find a suitable substitute for the MVV, usually based upon measurement of sections of the FVC recording. From the first, it was recognised that such measurements were indeed substitutes, simply methods of estimating what the MVV would have been if it had been measured directly. The value of recording the MVV itself was not disputed; these doubts were to occur later.

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The situation was reviewed in depth by Kennedy in 1953[6] who introduced the FEV_{40}, (or $FEV_{0.75} \times 40$) which is the substitute most widely used today. Its logical basis is as follows; firstly, provided the respiration rate is not too fast the recording of the MVV resembles sections of the inspiratory and expiratory FVC curves measured near to the TLC position and included a variable portion of the FVC. The actual 'swept volume' or amount of the VC traversed depends upon the respiration rate, the 'summits' of each breath approaching nearer to the TLC the slower the rate[5].

Secondly, when allowed to choose the rate and depth of breating quite freely, most individuals breathe at about 40 breaths per minute during the MVV manoeuvre. Assuming that inspiration and expiration are of the same duration, each expiration at this rate will take about 0.75 sec. Thus the FVC expired in 0.75 sec multiplied by 40 gives a value approximating to the MVV.

Even at 40 breaths per minute however the swept volume does not reach TLC and inspiratory and expiratory times are rarely exactly the same. The FEV_{40} therefore usually underestimates the MVV[5]. Increasing the rate of breathing increases the value of the MVV, reaching a maximum between 70 and 120 breaths per minute[7], when the FEV_{40} obviously underestimates the MVV even more.

Reliability of the MVV

Bernstein and Kazantzis[5], the discussion section of whose paper is a joy to read, showed that the MVV was correlated with $FEV_{0.75}$ and also with the FEV_1 (and some authorities use the FEV_1, multiplying it by 30 to arrive at the indirect MVV). They even found a good correlation with the FVC, but even a perfect correlation, does not justify replacing one measurement with another. It is valuable to review the factors that led to the wide acceptance of the FEV_{40} as a replacement for the MVV. Even back in the days when the investigation of ventilation prior to surgery for pulmonary TB was commonplace, Little[8] stressed that the FEV_1 was a more reliable measurement than the MVV on the grounds that the FEV_1 was more reproducible. As in many instances where this argument is applied, the significance of better reproducibility is suspect. Some measurements with a reproducibility of (say) 10% are clinically more valuable than others with a reproducibility of 2%. It is very easy to lose sight of the extent of the information that is being conferred in the quest for refinements in measurement.

Since the publication of the classical paper on lung mechanics by Fry and Hyatt in 1960[9] there has been a trend towards expressing the mechanical state of the lungs in terms of the three basic dimensions, pressure, volume and flow. Abnormalities have come to be expressed in terms of the changes in the relationships between these three aspects. The increased sensitivity of the flow-volume loop compared with the volume-time spirogram is an example of one of the advantages that have arisen from this approach.

The introduction of the more refined techniques does not necessarily replace the older, more easily executed and often more widely understood procedures. Further, it is now recognised that there are clinically important aspects of the performance of the patient as the whole which may be lost if the investigation is confined to the more specific tests.

McHardy and co-workers[10] introduced the 12-minute (and subsequently the 6-minute) walking test to display the overall capability of dyspnoeic patients under conditions directly related to their everyday lives. It is recognised that two patients of the same age and dimensions and apparently the same degree of lung dysfunction can attain surprisingly different distances in a timed walk. Thus other influences are revealed by this exercise procedure which are not shown by the standard tests, and a simple walking test can in no way be described as a specific procedure.

The same can be shown to apply in the case of MVV. The performance of the MVV may show how patients compensate for the limitations imposed by disease. The recommendation by Cotes[11] that high respiratory rates (not less than 80 breaths/minute) should be used to obtain the highest values for the MVV is very much borne out in patients with moderate degrees of airflow obstruction. Here the directly recorded MVV may be more than 30% greater than the FEV_{40} because the patient uses the small, relatively fast uppermost part of the FVC at a high respiratory rate, the chest held in a hyperinflated position throughout. Thus the swept volume is less than the $FEV_{0.75}$ and the respiratory rate is greater than 40 breaths per minute (See Table 1).

Table 1

	Patient	Predicted
MVV (litres/min)	105	120
FEV_{40} (litres/min)	74	120
FEV_1 (litres)	2.1	2.8
FVC (litres)	3.5	4.0
VC (litres)	4.4	4.0
RV (litres)	2.0	1.4
TLC (litres)	6.4	5.3
Flow/volume curve		
PEFR (litres/sec)	8.8	8.1
Flow (litres/sec) at:		
75% VC	3.9	7.6
50% VC	1.2	4.7
25% VC	0.5	1.6

Lung function indices in a male patient of 51 years with bronchiectasis. By using the upper faster part of the FVC the MVV is increased beyond the value indicated by the FEV_{40}.

The MVV in pulmonary fibrosis

Conversely, the MVV can display the added influence of non-conductive elements in reducing ventilation. Factors in the lung and chest wall which limit motibility cause the MVV to be less than the FEV_{40}. (In these cases it is easy to dismiss the reduced result as showing a lack of effort on the patient's part though direct observation may show that the patient is highly motivated and cooperating well.) Such a situation is sometimes found in the various types of fibrosis: restricted lung volume with high flow rates may give a near-normal FEV_{40}, yet the MVV may be reduced. The mechanism seems to be that as neuromuscular activity dies away at the end of expiration the chest movement (and hence airflow) dies away sooner than in the normal. Equally, as activity builds up for the subsequent inspiration there is a lag in the chest and air

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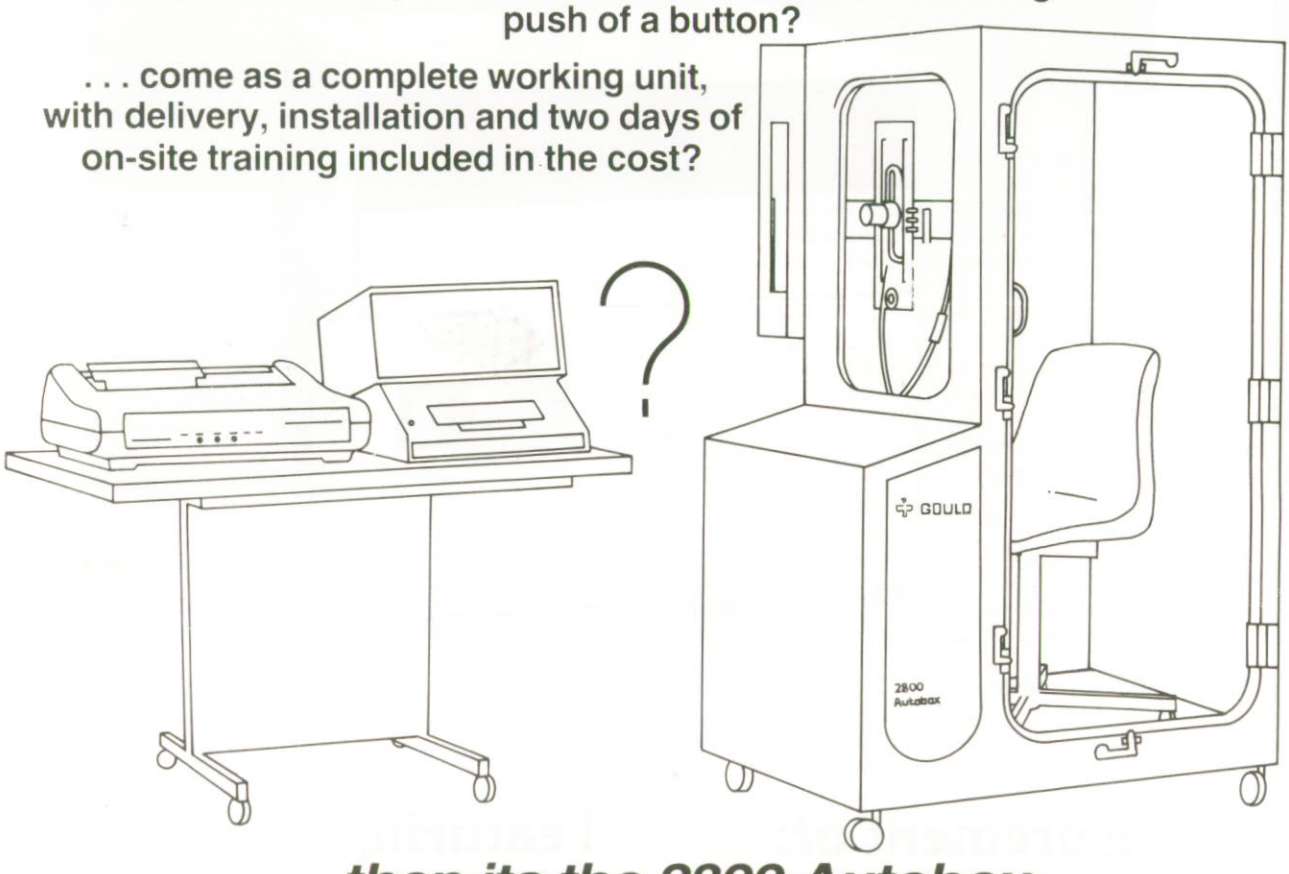

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ASTHMA CURED BY SURGERY: a case report

Olwen Cloudsley and Jean Elmes
St. Martin's Hospital, Bath

A 55 year old ex-miner was referred to the Chest Unit at St. Martin's in October 1982 with the following history:-

In June 1982, the patient complained to his General Practitioner of increasing breathlessness and was treated with antibiotics, 'Becotide' and prednisolone. The symptoms persisted and he was again admitted to hospital under the care of a General Physician where he became very breathless and cyanosed and on one occasion had a fit. He was treated with hydrocortisone, co-trimoxazole, salbutamol and aminophylline. His condition improved and he was discharged after four days though he remained short of breath. He was thought to be suffering from late onset asthma and was referred to the Chest Unit because of his failure to improve. When seen at the clinic, he was thought to have *stridor* rather than asthma and was admitted to St. Martin's Hospital for further investigation.

Past history. He had a hyperextension injury to his neck in January 1968 which led to cervical myopathy. This was later treated by surgical excision of the protruding discs. In March 1981 he was admitted to hospital under the care of a General Physician having become cyanosed and unconscious while watching television. No specific diagnosis was made.

Investigations

Preoperative lung function tests (Table 1) show marked reduction in FEV_1 and PEFR with normal VC and TLCO. The vitalograph recording was noted to be abnormal (Fig. 1), the striking feature being the straight line from the origin in contrast to the usual convex curve, suggestive of major airway obstruction. The preoperative flow-volume curve (Fig. 2) shows a relatively normal inspiratory section but marked blunting of the expiration section characteristic of variable intrathoracic obstruction of a major airway.

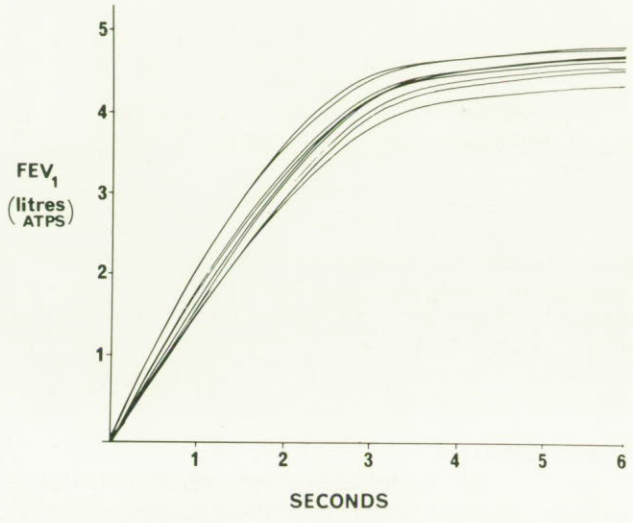

Fig. 1. *Preoperative vitalograph recording. Note the abnormal straight line from the origin. (compare Fig. 3).*

Table 1
Lung Function Tests

	Pre-operative		Post-operative	Predicted Values
	Before broncho-dilator	After broncho-dilator	Before broncho-dilator	
FEV_1 (litres)	2.0	2.2	3.1	3.5
VC (litres)	5.2	5.3	3.9	4.6
PEFR (litres/sec)	3.2	3.7	9.8	9.5
TLCO (mmol/min/kPa)	12.5	—	9.6	10.3

Bronchoscopy revealed an intra-tracheal polyp 5 cms above the carina which almost occluded the trachea on expiration. In October 1982 he was transferred to the Thoracic Surgery Unit at Frenchay Hospital. A *right thoractomy* was performed and a benign lipoma with adjacent trachea was successfully removed. There were no post operative complications.

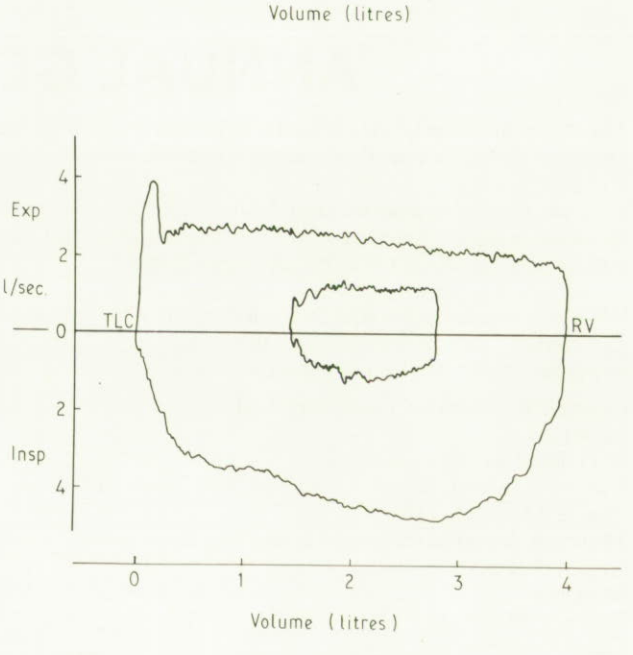

Fig. 2. *Preoperative flow-volume curve. Note the marked blunting of the expiratory section (compare Fig. 4).*

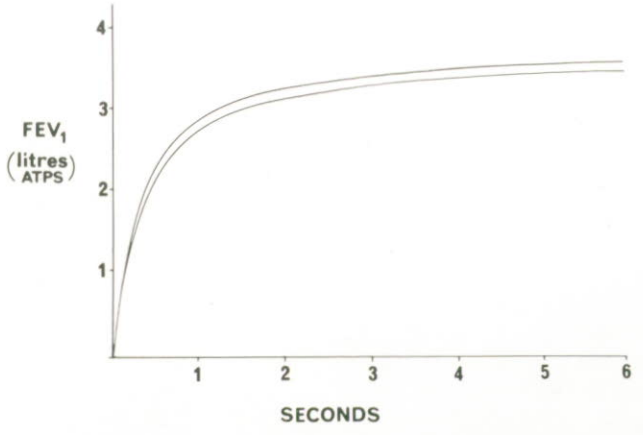

Fig. 3. Postoperative vitalograph recording. The curve has returned to normal.

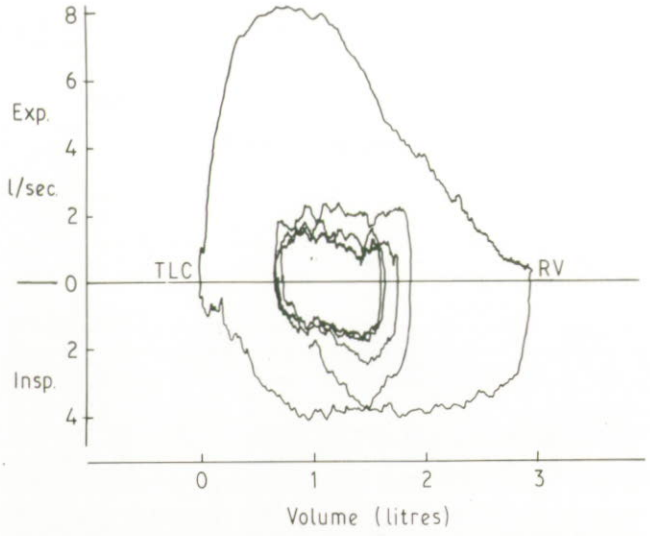

Fig. 4. Postoperative flow-volume curve. The form of the expiratory section has returned to normal.

Lung function tests were repeated and showed a substantial increase in FEV_1 and PEFR although the FVC was reduced perhaps due to the thoracotomy. The vitalograph trace and flow volume curve had returned to normal (Figs. 3 and 4). The patient has had no further episodes of wheezing or dyspnoea.

Discussion

A lipoma of the trachea is extremely uncommon and we have found only one other reported case. Bronchial lipomata are more common and make up 0.1% of all pulmonary tumours and about 13% of benign tumours. They occur more frequently in men than in women and the mean age at diagnosis is reported to be 52 years[1]. Although lipomata arise from fat cells in the peribronchial tissue they may be associated with the presence of lipomata elsewhere in the body, but not usually with obesity. Our patient had no other lipomata although he was appreciably obese. This case illustrates the usefulness of a simple spirometer tracing in that the abnormal shape of the expired curve drew immediate attention to the likely site of the abnormality.

Acknowledgment

We would like to thank Dr. A. R. Tanser for permission to report on this patient.

Reference

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ANNUAL GENERAL MEETING

The Annual General Meeting took place on 7th and 8th October 1983, at King's College Hospital.

We owe grateful thanks to Anne Watson and her colleagues for organising the meeting and for providing refreshments and to the speakers for their excellent papers.

We are particularly grateful to the following firms who most generously sponsored the meeting and put on demonstrations of their products:

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The following scientific papers were given by speakers from the Chest Unit, King's College Hospital:

1. Phototherapy for lung cancer. Dr P Hugh-Jones.
2. Sleep apnoea syndromes. Dr. J Price.
3. Respiratory muscle fatigue. Dr J Moxham.
4. Marathon running: madness or medicinal? Dr J Costello.

Chairman's Report — Derek Cramer

I should like to express my thanks on behalf of all Association members to Barbara Peattie and colleagues for organising the Spring Meeting at the Royal Liverpool Hospital and to Anne Watson and colleagues for organising the present Autumn Meeting at King's College Hospital. We are also most grateful to the speakers, to the caterers and to all the firms who provided sponsorship and demonstrated their equipment; all contributed to the success of these two meetings. This Annual General Meeting was the first occasion on which our proceedings have been extended to the Friday evening in addition to our usual Saturday; this was an excellent innovation.

Royal Society of Medicine

The Royal Society of Medicine has continued to show considerable interest in our organisation. We are particularly grateful to Dr. Martin Partridge who has organised a series of five lectures specifically for respiratory technicians; these are being held at the Royal Society of Medicine during October and November 1983.

British Thoracic Society

We have also been in communication with the British Thoracic Society which represents physicians and surgeons in the field of thoracic medicine. Dr. Hutchison and I have written to the secretary (Dr. Ian Campbell) to keep him informed about all the activities of the ARTP, including the present position of technicians in Thoracic Medicine and of changes envisaged for the future. The question of a joint meeting in some form has been raised but the British Thoracic Society is, at present, undergoing a considerable reorganisation of its own scientific meetings; a joint meeting might nevertheless be possible at some future time. Meanwhile the Society has assured us of its moral support and of its readiness to give us any advice we may need.

Executive Committee

Our long negotiations with the tax authorities have reached a satisfactory conclusion and the Association has been granted exemption from income tax. A considerable amount of time has been taken up in discussion and preparation of a report on the Integration of Scientific Services and on the education and training programme.

Gloria Gessey

All the members of the Association will regret the death of Gloria Gessey at the beginning of the year after a long illness. This is a profound loss to the Association (Obituary, *Breath,* June 1983).

Treasurer's Report — Mrs. G. Holbrook

The chief expenditure as in previous years is on travel expenses and postage. The meetings/exhibitions are now making a small profit and I would like to thank the firms for continuing to support our meetings and Miss G. Lowe for organising the meetings.

Breath continues to make a profit thanks to the hard work of Dr. D. C. S. Hutchison and Ms. J. Jones. Binders for *Breath* are still available at virtually cost price.

The Association has been granted exemption from the 1982 Income and Corporation Tax Act but we are still pressing for Charitable Status. Our request is still under consideration and we have been asked to make minor alterations to our constitution which will be discussed later on the agenda.

Finally, we still rely on subscriptions as our major source of income and I would like to remind members that subscriptions are due by 1st May each year.

STATEMENT OF INCOME AND EXPENDITURE

1st April 1982 to 31st March 1983

EXPENDITURE		INCOME	
Travel	1,205.09	Subscriptions	1,196.44
Catering	632.93	Donations	957.00
Postage & Stationery	484.44	Folders	150.70
Breath and Miscellaneous	2,800.55	Advertising and Miscellaneous	3,457.15
Excess Income over Expenditure	693.54	Deposit Account Interest	55.26
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Bank Balance 31.3.83	1,265.09	Balance Brought Down	693.54
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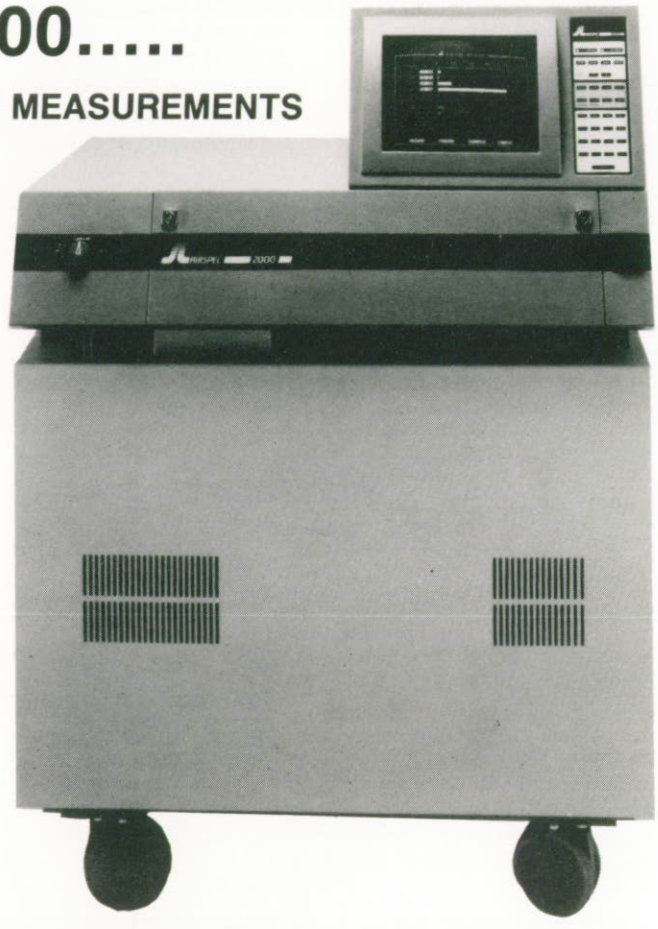

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BREATH: Editor's Report

The Editors of *Breath* are most grateful to all who have helped in the publication of the Journal. We owe thanks to our many contributors for their interesting articles, to Margaret Rusbridge for preparing many of the diagrams with her usual expertise, to Catherine Barton for invaluable typing help, to our advertisers for their continuing and welcome support and to Mr. T. Boughton and staff at Stevens Brothers, our printer, for their co-operation in the production of the Journal.

ARTP

Newly Elected Officers

CHAIRMAN	Miss S. Hill
SECRETARY	Mrs. S. Gough
TREASURER	Mrs. G. Holbrook
Executive Members	Mrs. D. Muirhead
	Mrs. S. Jackson
	Mr. D. McDonald
	Mr. D. Cramer

Public Relations Officer

Miss G. Lowe

F.A.M.T. Representatives

Mrs. S. Gough
Dr. J. Reed

Miss S. Hill
(to attend in the absence of either of the representatives)

Education Committee

Miss S. Hill, Chairman
Mr. K. Houston, Secretary
Mrs. S. Gough
Mr. P. Lockwood
Dr. D. C. S. Hutchison
Miss G. Lowe
Ms. D. Roberts
Dr. J. Reed

Editor and Sub-Editor of *Breath*

Dr. D. C. S. Hutchison and Ms. J. Jones

CORRESPONDENCE

Do our references values need up-dating?

I have thought for a long time that the reference values in use currently have need of standardisation and up-dating — a view also held by the physicians at this hospital. Most studies in the past have concentrated on one aspect of lung function, so that reference values for diffusion are calculated using a different population from that used for lung volumes, for example.

Would it not be possible for the Association as a whole, to prepare a new set of results? A good cross-section of the population can be obtained as members of the Association come from all over the country. Computer analysis would obviously be required, which my department is unable to provide. It would mean co-operation between departments in standardising techniques and providing data.

A great deal more thought and discussion, either through *Breath* or at future meetings, is needed on the subject but it is one way, I thought, that the Association could be more constructive.

Miss A. L. Morgan,
Senior Technician, Respiratory Physiology,
Tehidy Hospital, Camborne, Cornwall.

The Department of Cardiovascular Studies
The University of Leeds.

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Anyone interested should write to G. Wade, Departmental Superintendent, Department of Cardiovascular Studies, University of Leeds, Leeds LS2 9JT to reserve a place on the course and book accommodation if required.

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.

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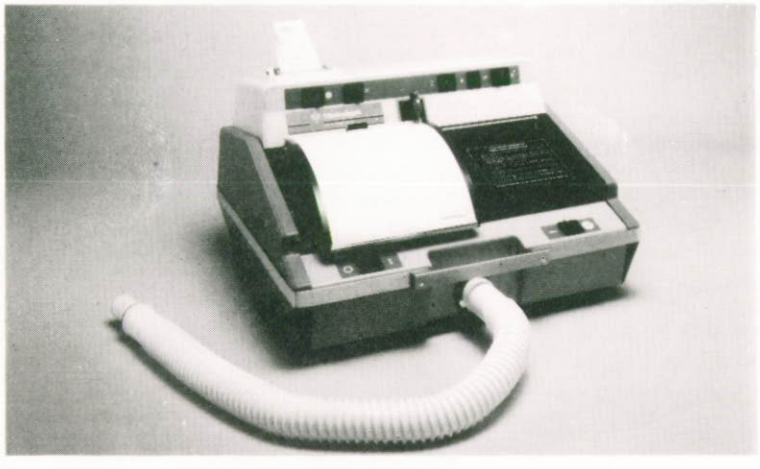

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Programme for the SPRING MEETING and TRAINING WORKSHOP

Post-graduate Medical Centre,
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Friday 6th April 1984

TRAINING WORKSHOP

12.30-2.20	Registration, Exhibition and Buffet	

SESSION 1. Chairman: Dr. D. C. S. Hutchison (King's College Hospital Medical School)

2.30	Your Education Committee	Mr. K. Houston Hon. Secretary, Education Sub-Committee
2.55	National Assessment	Miss S. Hill Chairman, Education Sub-Committee
3.20	The Trent Experience in Practical Assessment	Miss G. Lowe
3.45	Tea	

SESSION 2. Chairman: Miss S. Hill

4.00	Group Discussions	
5.30	Group Reports and Panel Discussions	
7.00	Buffet/Wine/Exhibition	

Saturday 7th April

SPRING MEETING

10.00-11.00	Coffee/Registration/Exhibition	

SESSION 1. Chairman: Dr. S. Williams, Chest Physician, Stoke Mandeville Hospital.

11.00	Inhaled Treatment of Severe Asthma	Dr. S. Williams Chest Physician Stoke Mandeville Hospital
11.30	Rheumatoid Lung — Fact or Fiction?	Dr. M. Webley Consultant Rheumatologist Stoke Mandeville Hospital
12.00	Chest Medicine in Central Africa	Dr. S. Fisher Chest Physician Stoke Mandeville and Milton Keynes Hospitals

SESSION 2. Chairman: Miss S. Hill, A.R.T. & P. Chairman

PRODUCT DEVELOPMENTS

12.30	Gould Medical Cranlea & Company (Warren Collins) P. K. Morgan Instrumentation Laboratory (UK) Ltd Intersurgical Ltd. Air-Spec Limited	Mr. I. Sloan Mr. D. Pollock Mr. C. Phillips Mr. G. Hart Mr. S. Williams Mr. Avery
1.00	Open Discussion	
1.15	Lunch/Exhibition	

SESSION 3

2.45	Optical Mapping of the Thoraco-Abdominal Wall of Spinal patients	Dr. M. Morgan Research Fellow Brompton and Stoke Mandeville Hospitals

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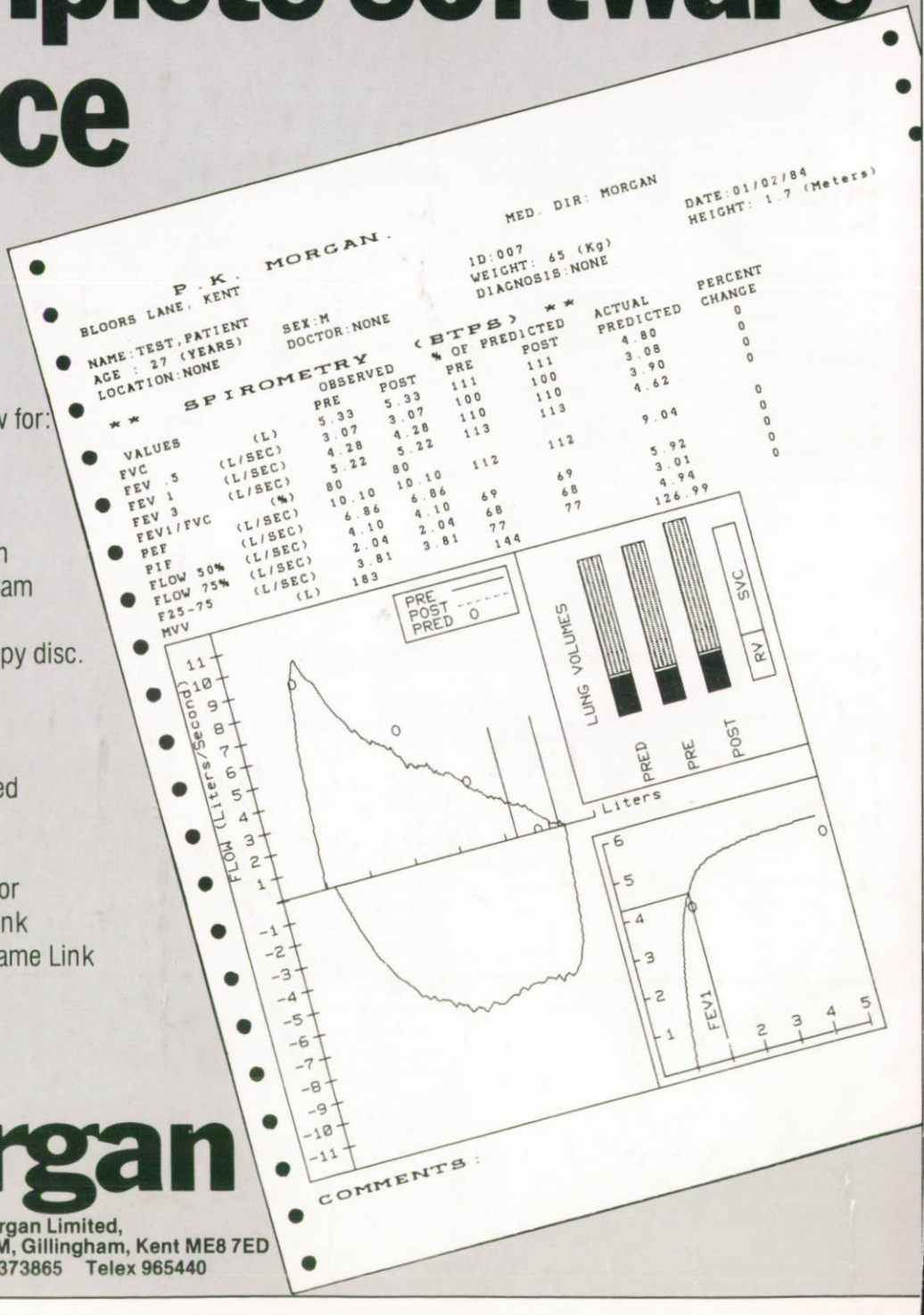

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