ASSOCIATION OF RESPIRATORY TECHNICIANS AND PHYSIOLOGISTS

ISSUE NO 17 OCTOBER 1982

BREATH

CONTENTS

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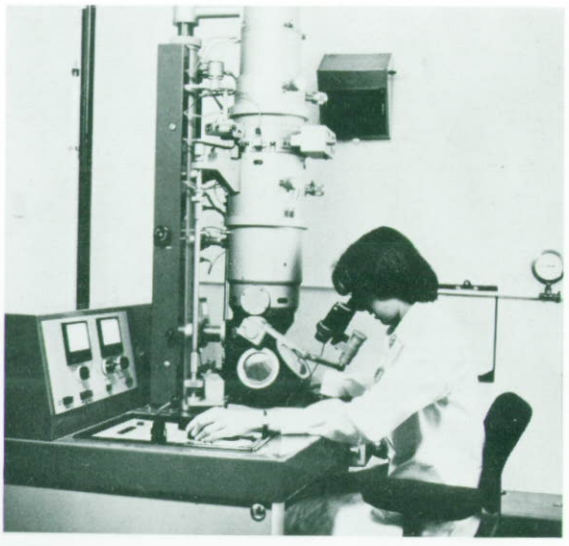
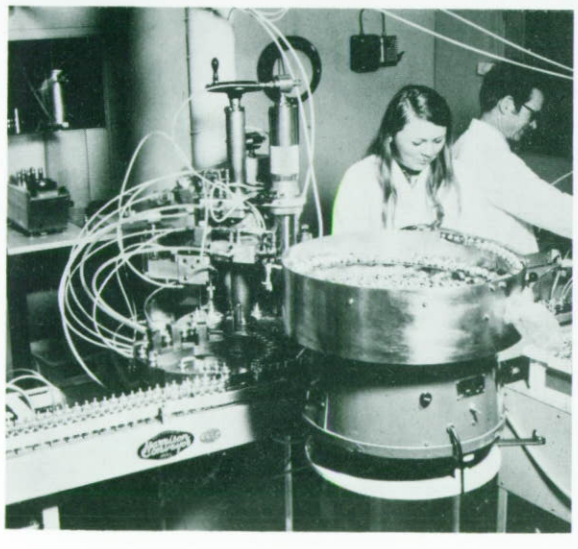
Leaders in the field of reversible airways obstruction



VENTOLIN – The bronchodilator no asthmatic need be without.

BECOTIDE – For the asthmatic who needs more than a bronchodilator.

BECONASE – Effective control of allergic rhinitis – both seasonal and perennial.



Significant advances in therapy from Original British Research

Ventolin Inhaler

(Salbutamol BP)

Uses
Routine control of bronchospasm in bronchial asthma, bronchitis and emphysema, or as required to relieve attacks of acute bronchospasm. Doses may also be taken before exertion to prevent exercise-induced asthma or before exposure to a known unavoidable challenge.
Dosage and administration
As single doses for the relief of acute bronchospasm, for managing intermittent episodes of asthma and to prevent exercise-induced bronchospasm.
Using Ventolin Inhaler—*Adults:* one or two inhalations.
Children: one inhalation increasing to two if necessary.
Using Ventolin Rotahaler—*Adults:* one Ventolin Rotacap 200mcg or 400mcg.
Children: one Ventolin Rotacap 200mcg.

For chronic maintenance or prophylactic therapy.

Using Ventolin Inhaler—*Adults:* two inhalations three or four times a day.
Children: one inhalation three or four times a day increasing to two inhalations if necessary.
Using Ventolin Rotahaler—*Adults:* one Ventolin Rotacap 400mcg three or four times a day.
Children: one Ventolin Rotacap 200mcg three or four times a day.
For optimum results in most patients inhaled Ventolin should be administered regularly.
Contra-indications
Ventolin preparations should not be used for the prevention of threatened abortion during the first or second trimester of pregnancy.
Precautions
If a previously effective dose of inhaled Ventolin fails to give relief lasting at least three hours, the patient should be advised to seek medical advice. Ventolin should be administered cautiously to patients suffering from thyrotoxicosis.
Unnecessary administration of drugs during the first trimester of pregnancy is undesirable.
Side effects
No important side effects have been reported following treatment with inhaled Ventolin.
Presentation and Basic NHS cost
Ventolin Inhaler is a metered-dose aerosol delivering 100mcg Salbutamol BP per actuation. Each canister contains 200 inhalations. Basic NHS cost £3·00.
Ventolin Rotacaps 200mcg and 400mcg, each contain a mixture of the stated amount of microfine Salbutamol BP (as sulphate), and larger particle lactose in light blue/colourless or dark blue/colourless hard gelatine cartridges, respectively. Containers of 100. Basic NHS cost £5·29 and £7·15, respectively.
Ventolin Rotahaler for use in conjunction with Ventolin Rotacaps. Basic NHS cost 78p.
Product Licence numbers

Ventolin Inhaler	0045/5022
Ventolin Rotacaps 200mcg	0045/0116
Ventolin Rotacaps 400mcg	0045/0117

Becotide Inhaler

(Beclomethasone Dipropionate BP)

Uses
Bronchial asthma especially in patients whose asthma is not adequately controlled by bronchodilators and patients with severe asthma who would otherwise be dependent on systemic corticosteroids or adrenocorticotrophic hormone (ACTH) or its synthetic equivalent.
Dosage and administration
Using Becotide Inhaler—*Adults:* two inhalations three or four times a day is the usual maintenance dose.
Alternatively, the total daily dose may be administered as two divided doses. In severe cases dosage may be started at twelve to sixteen inhalations per day and subsequently reduced when the patient begins to respond. *Children:* one or two inhalation, two, three or four times a day according to the response.
Using Becotide Rotahaler—*Adults:* one 200mcg Becotide Rotacap three or four times a day is the usual maintenance dose. Alternatively, the total daily dose may be administered as two divided doses. *Children:* one 100mcg Becotide Rotacap two, three or four times a day according to the response.
For optimum results inhaled Becotide should be administered regularly.
Contra-indications
No specific contra-indications to inhaled Becotide are known but special care is necessary in patients with active or quiescent pulmonary tuberculosis.
Precautions
The maximum daily intake of Beclomethasone Dipropionate BP should not exceed 1mg. Inadequate response after the first week of inhaled Becotide therapy suggests that excessive mucus is preventing penetration of inhaled drug to the target area.
A short course of systemic steroid in relatively high dosage should be given and therapy with inhaled Becotide continued.
Unnecessary administration of drugs during the first trimester of pregnancy is undesirable. When transferring patients to Becotide from systemic steroid therapy the possibility of adrenocortical suppression should be considered and patients given a supply of oral steroids for use during periods of stress. Please refer to the detailed procedure described in the data sheets for Becotide Inhaler and Becotide Rotacaps.
Side effects
Occasional candidiasis of the mouth and throat (thrush) occurs in some patients, particularly those with high blood levels of *Candida precipitins*. Topical therapy with antifungal agents usually clears the condition without withdrawal of Becotide.
Presentation and Basis NHS cost
Becotide Inhaler is a metered-dose aerosol delivering 50mcg Beclomethasone Dipropionate BP per actuation. Each canister contains 200 inhalations. Basic NHS cost £4·77.
Becotide Rotacaps 100mcg and 200mcg, each contain a mixture of the stated amount of microfine Beclomethasone Dipropionate BP and larger particle lactose in buff or chocolate-brown/colourless hard gelatine cartridges, respectively. Containers of 100. Basic NHS cost £7·26 and £9·67 respectively.
Becotide Rotahaler, for use in conjunction with Becotide Rotacaps. Basic NHS cost 78p.
Product Licence numbers

Becotide Inhaler	0045/0089
Becotide Rotacaps 100mcg	0045/0119
Becotide Rotacaps 200mcg	0045/0120

Beconase Nasal Spray

(Beclomethasone Dipropionate BP)

Uses
The prophylaxis and treatment of perennial and seasonal allergic rhinitis, including hay fever and vasomotor rhinitis.
Dosage and administration
The recommended dosage is two applications into each nostril twice daily. Alternatively, a single application may be given into each nostril three or four times a day.
Not for use in children under six years of age.
Contra-indications, warnings, etc.
There are no specific contra-indications but any infections of the nasal passages and paranasal sinuses should receive the appropriate treatment.
Care must be taken while transferring patients from systemic steroid treatment to Beconase if there is any reason to suppose that adrenal function is impaired.
Unnecessary administration of drugs during the first trimester of pregnancy is undesirable.
No major side effects attributable to Beconase have been reported, but occasionally sneezing attacks have followed immediately after use of the aerosol.
Presentation and Basic NHS cost
Beconase Nasal Spray is a metered-dose aerosol delivering 50mcg Beclomethasone Dipropionate BP per actuation into a special nasal applicator. Each canister provides 200 applications. Basic NHS cost £4·77.
Product Licence number
0045/0093

Further information on Beconase, Becotide, Rotacap, Rotahaler and Ventolin (trade marks) is available from:
Allen & Hanburys Limited, Greenford UB6 0HB

EDITORIAL

Killer dust or vital commodity?

Asbestos is in the news again, which is not a little surprising since workers in the asbestos industry were known to be affected by pulmonary fibrosis as long ago as 1906! The current resurgence of interest has in part been stimulated by the recent Yorkshire TV programme 'Alice: a fight for life'. Programmes of this sort are often criticized for lack of objectivity or just plain sensationalism (perhaps valid comment on occasions) but they nevertheless present the personal tragedy in a manner which statistical analysis can never reveal.

For there's no shortage of statistics! The extraction and processing of asbestos and its conversion into a multitude of products form a major industry employing many thousands of workers and the world consumption of asbestos is now in the region of 5 million tons per year. The substance derives its value from its strength and resistance to heat and corrosion — the word literally means 'indestructible'. There are said to be over one thousand different applications: for fire- and heat-resistant materials, in insulation and in cement, the bulk of the material being used in the building industry.

One can hear dramatically opposing views on how useful a substance this is: from a manufacturer we might hear that asbestos is an essential commodity, which with the present regulations can be processed with complete safety and that there are in any case, no effective substitutes for many applications. From a trade unionist we might hear that the regulations are of no value and that asbestos should no longer be used.

The term 'asbestos' refers to a number of silicates which have a unique fibrous structure giving the material its much valued properties. The main types are: *Chrysotile* or 'white asbestos': this is far the commonest form in nature and the most widely used. It is probably the 'safest' of the three types perhaps because the fibres are curly and thus do not penetrate into the lung so far as other types of fibre. *Crocidolite* or 'blue asbestos': this seems to be far the most dangerous form having very long fibres which penetrate deeply into the lungs. It has been banned from import into the UK for the last 10 years, but has already been widely used in sprays for insulation and lagging with obvious hazards to demolition workers. *Amosite* or 'brown asbestos': this is intermediate in hazard between chrysotile and crocidolite but its use is now increasing considerably.

The diseases associated with the inhalation of asbestos dust rank among the more unpleasant afflictions. *Pulmonary fibrosis* was recognised over 70 years ago and is a progressive, irreversible and ultimately fatal condition. *Bronchial carcinoma* was proved to be associated with asbestos by Doll in 1955 and the risk is increased many times in cigarette smokers. *Mesothelioma* is a malignant tumour of

the pleura; it is much less common than bronchial carcinoma but occur rarely if at all outside the context of asbestos exposure. It is particularly associated with crocidolite though probably all forms of asbestos can cause it.

Mesothelioma is an appalling disorder and the long-suffering Alice died of it at the age of 47 after working for no more than 9 months at Acre Mill (now closed) in Hebden Bridge, Yorkshire; she eventually managed to obtain £36,000 in compensation. There is no effective treatment for mesothelioma at present and surgical intervention seems to make matters worse, if possible. The time lapse between exposure and the appearance of the disease can be as long as 40 years which creates great difficulty in establishing the facts and in making compensation claims. It has indeed been shown that the death certificates of many people known to have had an asbestos-related disease, do not contain any such information and that many sufferers (or more likely, their widows) have received little compensation or none at all.

It is still believed that these harrowing case histories simply reflect the working practices of the 'bad old days' and that current hygiene standards should protect workers in this industry. The Advisory Committee on Asbestos was formed in 1976 to look into the whole question and has made a number of recommendations to the Government: none of these have apparently passed into law, though some have been put into practice on a voluntary basis. One such recommendation is the so-called 'one-fibre standard' which means a maximum level for chrysotile of 1 fibre per ml of air respired by the workers. The corresponding figure for amosite is 0.5 and for crocidolite 0.2, the latter being in any case subject to a voluntary ban. Many experts now believe the chrysotile standard should be reduced to half the present level but we are still left with the question of how any standard can readily be maintained other than by the major producers and manufacturers. Many areas could be left quite free of restraint, for example, those industries concerned with the repair or demolition of asbestos containing structures, often undertaken by small contractors or the self-employed, where the workers may have little information on the materials which they are handling.

Asbestos dust may moreover affect people not working directly in the industry, such as the wives of asbestos workers or those living in the immediate neighbourhood of asbestos factories. A hidden problem is that products of asbestos are now accumulating in enormous quantities from indiscriminate and unrecorded dumping of waste building materials and in many countries, regulations fall very far short of our own and there may well be no surveillance whatsoever.

Plain logic dictates that from the health point of view, asbestos should cease to be used and that alternative materials should be developed to take its place; indeed a certain amount of research has already taken place along these lines. A further stimulus is no doubt arising from a massive increase in litigation against asbestos companies, especially in the US and a record award of £800,000 was recently paid to the widow of a mesothelioma victim. We just hope that the new products will be less toxic!

THE ASSESSMENT OF TRAINEE TECHNICIANS
The Trent Regional Scheme

R. Douglas Moore and Anne E. Perry
Trent Regional Health Authority, Sheffield

Background to the Scheme

The provision of adequate training for technical staff of all disciplines has always been of importance in the Trent Region and this Region was the very first in the country to set up a separate training section. In the disciplines of Medical Physics and Physiological Measurement, a Regional supernumerary training scheme for technicians has been in operation since 1973, largely due to the foresight of Professor Harold Miller, formerly Regional Physicist and Head of Medical Physics in Sheffield. The scheme was initially confined to South Yorkshire, but over the years (particularly since the changeover from ONC to TEC) has grown to encompass the whole Region. Although not all student technicians are employed on a supernumerary basis, all (both supernumerary and in-post) have followed similar training programmes at Colleges of Further Education in Sheffield or Nottingham.

Production of Training Modules

The training arrangements for Medical Physics and Physiological Measurement Technicians have been supervised by a *Steering Committee*. During the past two years, this Committee, and its specialist working groups, have put considerable effort into the production of practical in-service training modules which were intended to form the basis of a structured in-service training programme that would ultimately take over from the 'apprenticeship' type of training in the practical skills which was previously in operation.

The first modules to be produced were for those disciplines with the largest number of technicians. The remaining modules have only become available in recent weeks, in fact at the same time as those prepared by the respective professional groups in association with the Regional Training Officers. This may be unfortunate in some ways, but we do not consider our efforts to have been wasted and hope that the two sets of modules will eventually be fused and the best points of each adopted. We did not complete our work on all the modules we intended to prepare, and those for the Perfusion and Anaesthetics technicians remain outstanding. These will not be pursued for the time being firstly because the national training manual for Perfusion is now available and secondly because of the very small prospective number of trainees in Anaesthetics and uncertainties on a suitable training programme.

Having published the modules, the Steering Committee was concerned that they should not only be accepted in principle, but actually put into practice. It was felt that their validity and acceptability to the professional groups would only be clearly established by arranging for an assessment of the adequacy of the trainee's performance and, by implication, the adequacy of the training programme itself. The scheme we have produced follows, we hope, quite simple lines. It has been accepted by the Regional Scientific Committee and the Regional Team of Officers and arrangements are now being made to implement it as soon as possible.

Assessment Procedure

We propose a panel of assessors for each discipline, such as Respiratory Physiology, Audiology or Neurophysiology, each panel consisting of two clinicians or non-clinical scientists and two technical members. Assessors would be selected by the Steering Committee from nominations by the appropriate professional (including clinical) groups in the Region. They would serve for three years in phased rotation, thus providing a degree of continuity by ensuring that not all assessors retire at the same time.

The panel of assessors would prepare protocols for the assessment consisting primarily of oral and practical tests, but not excluding written tests if these were considered essential. Protocols would be approved by the Steering Committee who would also coordinate them to ensure a uniform standard and would establish a liaison with the DHSS and with the national professional bodies both on test protocols and the selection of assessors.

Two assessors from the appropriate panel (one clinician or scientist and one technical member) would conduct assessments at a small but sufficient number of centres in the Region to minimise travelling and absence from duty. In order to ensure impartiality assessors would not conduct tests at their own centres or on their own students. Successful completion of the assessment would be acknowledged by a certificate from the Regional Health Authority over the signatures of the assessors.

Certain points have yet to be clarified: for example, no final decision has yet been made as to how many attempts each candidate would be allowed at the assessment, though we anticipate that they would be allowed at least one re-sit. It will be the task of the Steering Committee to resolve this and other matters such as the selection of the assessment centres and whether suitable accommodation facilities can be provided, in consultation with departments and District Health Authorities. In some ways the fewer centres used the better as long as travelling is not excessive. In terms of the accommodation to be provided, we have to consider the disruption to the host department and the need to provide suitable examination conditions for the student. We still have to establish that the equipment for practical tests will be available at a sufficient number of centres and this will obviously have to be a factor in selection. Keeping the number of assessment sessions to a minimum is more economical in time and expense, but we would not want the number of candidates to be so large that it was not possible to give each a fair hearing. Finally, there is the confidentiality of questions to consider. It seems sensible to ask all candidates in one speciality the same questions, but if we cannot assess them all on the same day at the same centre we need to devise some kind of security system or to have different, but equivalent, questions. On many of these points we will be looking to our education colleagues on the Steering Committee for advice since they have more experience in matters of this kind.

The costs of the scheme requiring explicit funding are estimated at £1,600 per year which can be absorbed in the Regional training budget. However, the total cost of the scheme has been estimated at £6,500 per year which includes for example, allowances for absences from duty of both candidates and assessors.

Acceptability of the Scheme

Reaction from District Health Authorities has been encouraging in that most support the assessment scheme in principle. However, there are areas for concern, most notably the extent to which each District can mount an assessment in view of their dependence on the resources available, such as equipment, staff and space. Not all staff have time to take students through the specialist training so that an assessment can be made and we must also acknowledge a desire not to conflict with any national scheme.

This then is the situation as we begin to put the assessment scheme into practice. We hope to get it into operation in May or June, 1983 for those trainees who will have completed their specialist training during the academic year 1982/3. When the first assessments have been completed we hope to make the results of our experience available so that others may both criticise and make use of them in any similar plans they may have.

HARROGATE '82

IN-SERVICE TRAINING OF STUDENT TECHNICIANS

To follow up the first DHSS meeting on this topic which was held in August, 1981, (See Editorial, *Breath* October, 1981), a second meeting was held at the NHS Training and Studies Centre, Harrogate on 3rd and 4th June, 1982.

The delegates from each Association were asked to form a Working Party to discuss practical problems in the future use of the training manuals. A statement was made to the meeting on behalf of the ARTP delegates and the delegates and the following is a summary:

Training of Student Technicians in Respiratory Physiology.

The delegates considered that the Regional Scientific Officers should hold the primary responsibility for distribution of the Training Manuals. They should work closely with the Regional Training Officer who would have details of all students in the Region.

The Regional Training Officers should inform the senior technicians in each department (if necessary, by calling a meeting) that the Manuals were now available and should be put into effect forthwith. The senior technicians would be asked to indicate any areas where teaching on a specific topic could not be carried out, either through lack of equipment or knowledge. Courses of instruction in teaching methods for potential supervisors should be available.

The Training Manual would be continuously assessed by the Education Committee of the Association of Respiratory Technicians and Physiologists (ARTP) and the manual should be up-dated annually.

A number of possible centres for training of student technicians have been named, but at present there are no 'Approved Centres'. The standards for these centres will be set and maintained by the ARTP and it follows that a delegation from the ARTP would visit and assess each laboratory.

The ARTP is in favour of the O TEC course, but consider that practical in-service training should be incorporated into the course. The ARTP does not wish to establish its own professional examinations. Examinations in all disciplines should be standardised through the FAMT.

PLEURAL ULTRASOUND — Its practical value

J. W. Hadfield
Derbyshire Royal Infirmary

Disease of the pleura is a common clinical problem; it is often of great importance to establish whether there is an effusion (collection of fluid) in the pleural space, but standard chest radiographs are often unhelpful in localising the fluid and in distinguishing it from a solid mass.

The standard approach to this problem is the time-honoured trial of aspiration with a needle and syringe. Not only is this unpleasant for the patient but is likely to miss significant quantities of pleural fluid particularly if it is loculated. Two new techniques may help in this situation. *Computerised axial tomography (CAT)* has been shown to be superior to standard radiography in defining pleural disease[1], but it is expensive and not widely available. Since CAT scanning can only be performed with the patient in the supine position, there may still be difficulties in obtaining fluid at aspiration. Conventional *ultrasound* machines are also expensive, cannot be taken to the bedside and have hitherto been unhelpful in thoracic disease.

The use of echocardiographic equipment for pleural ultrasound was first described in 1967[2], but this has not gained wide acceptance despite its superiority over plain radiographs in the detection of pleural fluid[3]. A recently completed trial at Papworth Hospital[4] has shown pleural ultrasound to be 92% accurate compared to 68% for standard radiography, the combined results being correct

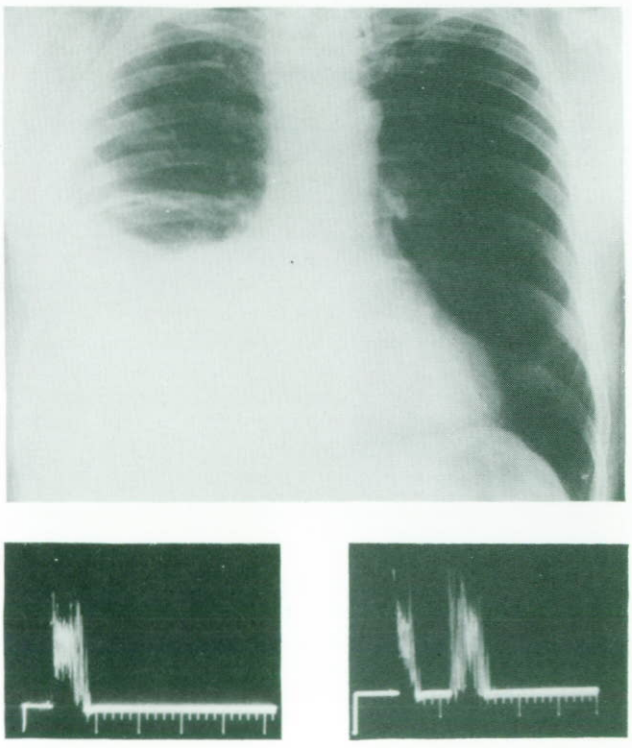


Fig 1. *(A) Chest radiograph showing a right sided pleural effusion, confirmed by ultrasound. (B) Normal ultrasound appearance. (C) A clear echo-free zone demonstrates the presence of a pleural effusion.*

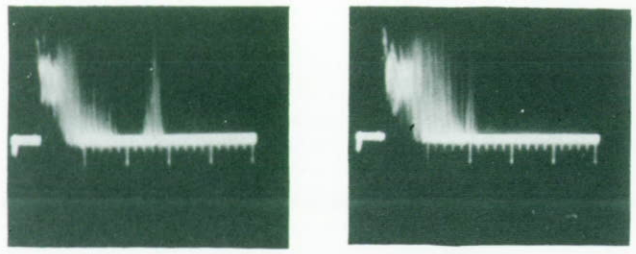


Fig 2. *Ultrasound picture taken at the level of the normal diaphragm on (A) inspiration and (B) expiration. A relatively echo-free zone can be seen but the edge of this space is not clear cut.*

in 98% of cases. In this series of 50 patients, even when radiographs correctly predicted the presence of fluid, the first attempt at aspiration by experienced clinical staff failed in 40% of cases. The correct site for aspiration was then identified in all patients by ultrasound. The conclusion from this study was that pleural ultrasound made a significant contribution to patient management in 50% of cases, either by clarifying the diagnosis where radiographs had been equivocal or by aiding aspiration and biopsy. The diaphragm was identified in the normal hemithorax in all patients; on the abnormal side it was identified in all patients with effusions but not in 5 of the patients with pleural thickening or tumour.

The Technique

Ultrasound examinations are performed using the A-mode (eg Ekoline 20, Smith Kline Instruments Incorporated) with a 2.25 MHz transducer. The transducer has a diameter of 1.5cm and is applied to the chest wall in the rib spaces.

To keep the technique simple, we have found it best to use the machine with the controls set to the same point each time. The settings will largely depend on the type of machine used. On the Ekoline apparatus the 'Damp' and 'Reject' controls are set to give mild damping at No 2. The Ramp is removed from the screen because detailed imaging from one particular depth is not required and this enables the Near Gain control to be ignored. The Course Gain control is then set at No 3 (scale 0 to 10) and only rarely are adjustments to the Course Gain control then required.

Just as in conventional echocardiography, it is important to angulate the transducer within a rib space since an echo-free zone will only be demonstrated if the sound is reflected directly back to the transducer.

Typical Ultrasound Findings

Since an air filled lung transmits sound very poorly, the pattern obtained from the normal chest consists of a cluster of echoes to a depth of 3 to 5cm. These arise from the chest wall and outer few centimetres of the lung (fig 1).

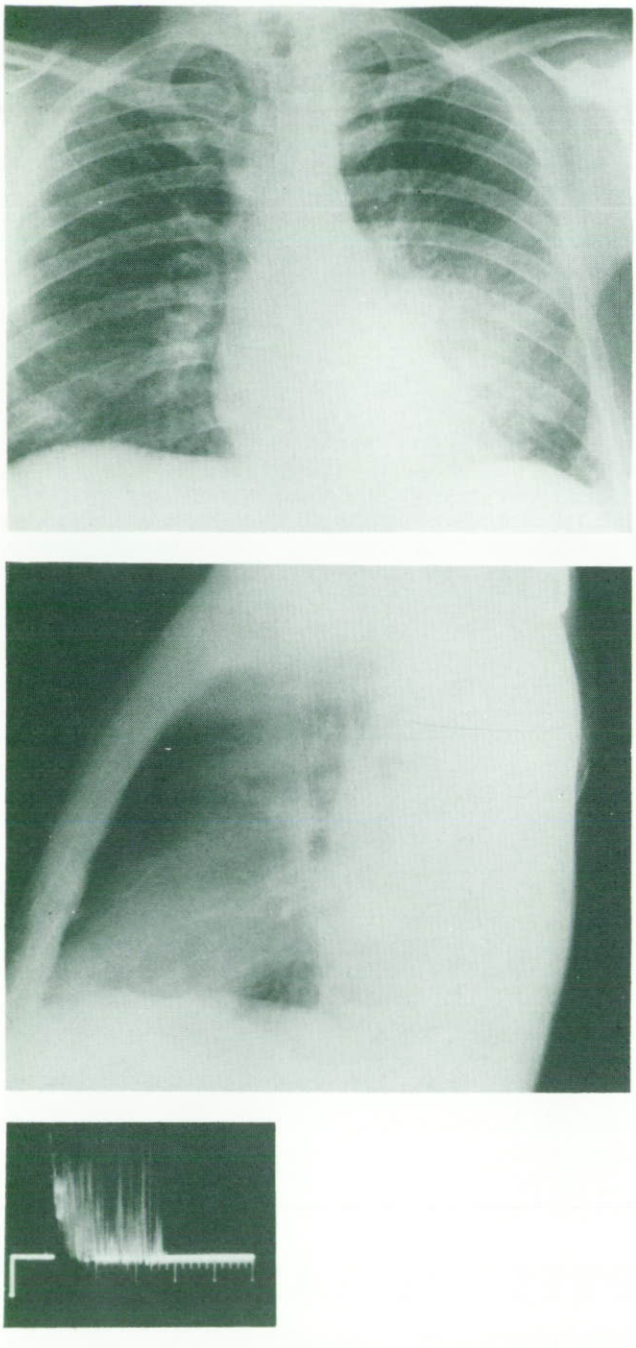


Fig 3. *(A) The chest radiograph of a 35-year-old man with left lower lobe pneumonia. (B) The lateral film suggested a loculated empyema which was also suspected clinically because of a swinging fever. (C) Ultra-sound did not detect fluid but confirms consolidated lung, also confirmed by tomography.*

If a *pleural effusion* is present the sound will be transmitted through the fluid and is reflected back by the lung deep to this fluid. Two sets of echoes will be produced, one from the chest wall and one from the underlying lung, with an echo-free space in between corresponding to the depth of the fluid. The quantity of fluid present can be gauged both by the depth and by the area over which this echo-free space can be found (fig 1).

Echoes arising from the boundary between the fluid and lung are always at their maximum height immediately after the echo-free space. It is important to note this since

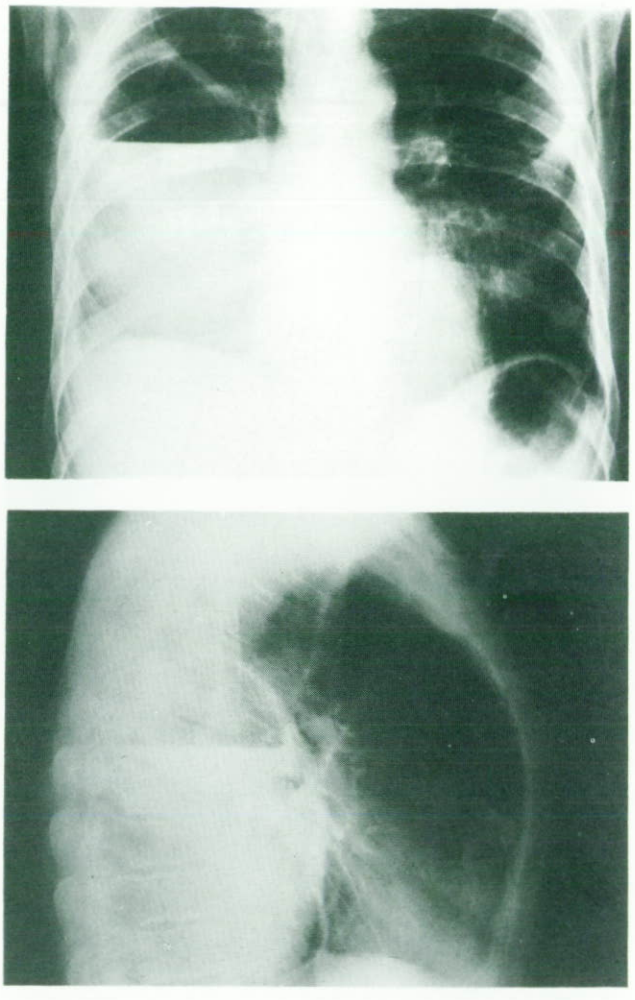


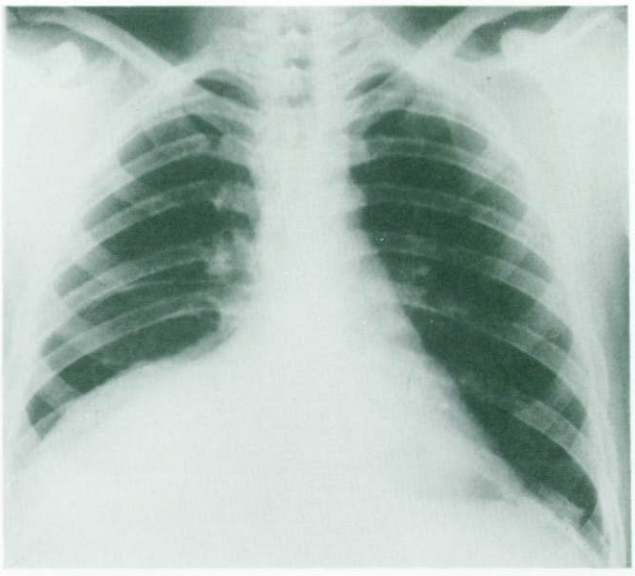
Fig 4. *(A) PA chest radiograph and (B) lateral showing a large loculated fluid-filled cavity situated posteriorly in the right lung. Three attempts at aspiration failed before ultrasound was used to identify the correct site for aspiration.*

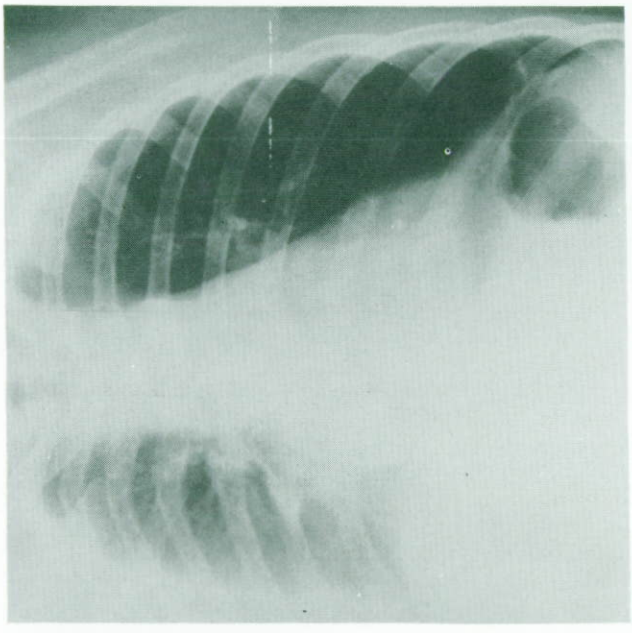
it is possible to find a relatively echo-free zone at the level of the normal diaphragm (fig 2). It can be seen that the boundary between the echo-free zone and the echoes is not clear cut. However, this phenomenon is useful in identifying the position of the diaphragm.

When *pleural thickening* is present the depth of echoes obtained is increased, by an amount corresponding to the thickness of the pleura. A similar appearance will be obtained if the lung immediately adjacent to the pleura is consolidated. (fig 3).

We have found pleural ultrasound to be very helpful in selecting the best sites for pleural aspiration since many effusions are loculated (fig 4) and the position of the diaphragm is not always clear. This particularly applies to *empyema* (a collection of pus) and avoids the necessity for frequent attempts at aspiration in patients with unidentified pleural disease. If no fluid is detected by ultrasound a decision can then be taken as to whether a formal biopsy with a cutting needle or drill is indicated.

A common clinical situation is the patient whose chest radiograph shows a completely opaque hemithorax. This is usually due to a combination of pleural effusion and





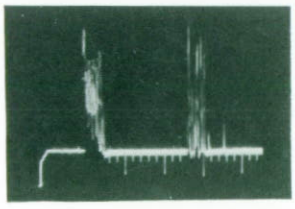


Fig 5. *(A) Chest radiograph showing what could be either collapse or subpulmonary effusion. (B) The film is taken with the patient lying on the R side. The effusion has moved to the dependant part of the R hemi-thorax and is demarcated by the horizontal line. (C) The effusion was clearly identified by ultrasound.*

collapsed lung. Little fluid may be present however, and attempts at aspiration often fail. Ultrasound will predict whether there is a significant effusion and where to aspirate and will quickly show whether a bronchoscopy would be more appropriate.

Subpulmonary effusions may pose both diagnostic and therapeutic problems as illustrated in fig 5. Although special views such as supine or decubitus films will often confirm the presence of fluid, this does not always help the clinician who needs to know the correct site for aspiration.

Conclusion:

Pleural ultrasound is a simple bed-side investigation using widely available low-cost equipment. Essentially the technique will answer the 3 main questions. Is the opacity seen on the radiograph solid or fluid? How much of the opacity is fluid, and where is the most appropriate site for pleural aspiration? Hitherto these questions have only been answered by trial and error, not a pleasant procedure for patients subjected to frequent attempts at aspiration.

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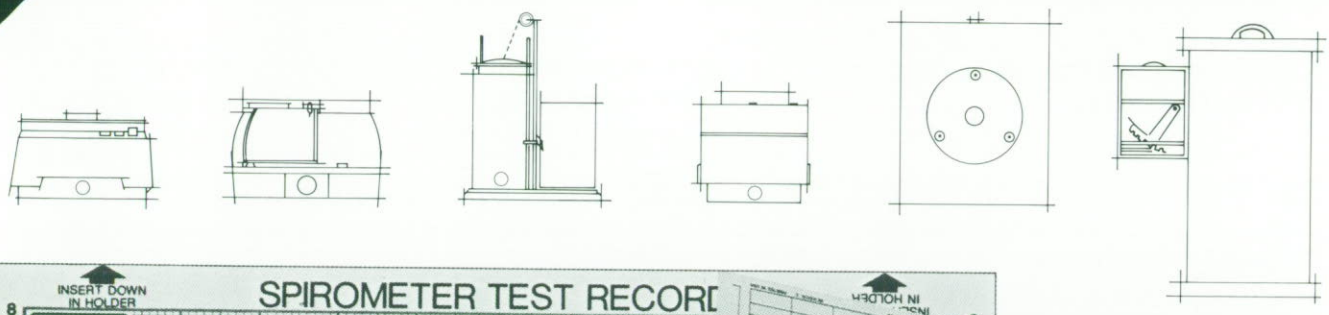

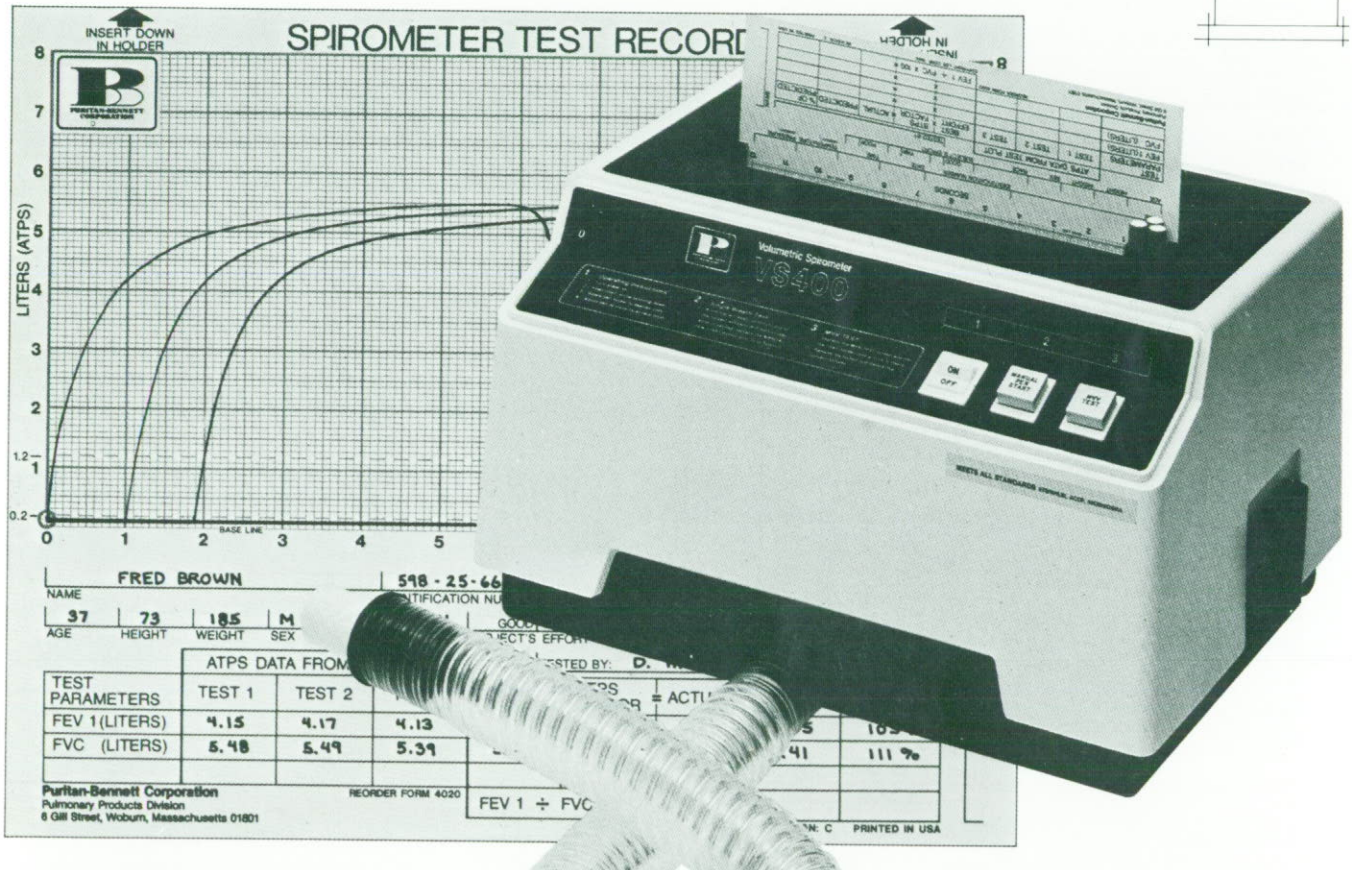

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DYNAMIC SPIROMETRY							
FVC	Lts:	5.02	92.65	5.41	7.54	139.16	150.19
FEV 1	Lts:	3.52	78.61	4.47	5.61	125.34	159.37
FEV1/FVC	% :	70.11	85.00	82.47	74.40	90.20	106.11
PEFR	Lts/Secs:	4.61	59.11	7.79	11.07	142.01	240.13
PIFR	Lts/Secs:	3.27			13.22		251.08
PIFR/PEFR	% :	70.93			119.42		168.36
MMF	Lts/Secs:	3.38	68.21	4.95	5.32	107.36	157.39
FEF 25%	Lts/Secs:	4.26			9.47		222.30
FEF 50%	Lts/Secs:	3.91	51.64	1.55	6.13	60.85	156.77
FEF 75%	Lts/Secs:	2.86	80.57	3.54	2.97	83.67	103.84
FET	100% :	1.68			3.22		191.66
MTT	Secs:	8.47					
FEV 0.5	Lts:	1.49			3.45		231.54
FEV 2	Lts:	4.96			6.95		140.12
FEV 3	Lts:						
MVV	Lts:	120.24	62.08	193.66	250.80	129.50	208.58

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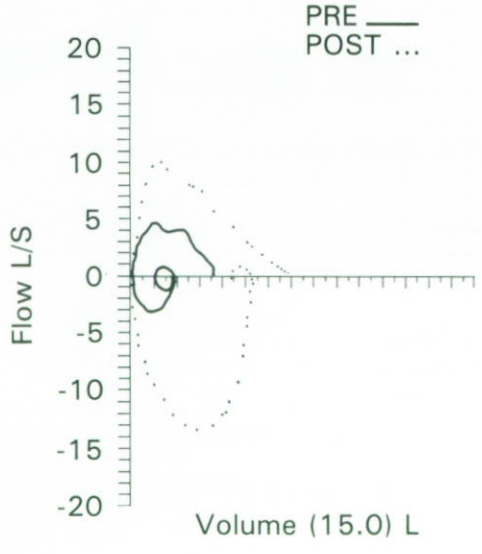
Erv	Lts:	1.00
Rv	Lts:	3.52
Svc	Lts:	5.00
Tlc	Lts:	8.52
Rv/Tlc	% :	41.00
DIFFUSION		
Tl (eff)	mL/min/mmHg:	
Va	Lts:	8.47
Kco	:	1.45
Dm	mL/min/mmHg:	21.91
Vc	mL:	88.69
Tl (Prime)	mL/min/mmHg:	42.36
HB	:	14.80
DLCO	:	



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EXERCISE, COLD AIR AND ASTHMA

Richard Heaton
Chest Unit, King's College Hospital Medical School, London, SE5

"Whatsoever therefore makes the blood boyl, or raises it into an effervescence as violent motion of the body or mind, excess of extern cold or heat... doth cause asthmatical assaults to such as are predisposed."

Thus, Thomas Willis writing in the seventeenth century clearly recognised that attacks of asthma could be precipitated by exercise or by changes in environmental conditions. Different forms of exercise were also known to differ in their potential for inducing attacks of asthma, arm exercise being more likely to provoke an attack than leg exercise. Despite the long recognition of exercise as a powerful, naturally occurring, precipitant of asthma, it is only in recent years that the relationship between stimulus and response has been quantified in terms of respiratory heat exchange. This theory allows much of the apparently conflicting evidence on exercise and asthma to be reconciled.

Since the days of Willis, numerous theories have been proposed to explain how muscular work produces bronchoconstriction. These have included changes in blood CO_2 levels; increased lactic acid in the blood from anaerobic metabolism in the exercising muscles; increased minute ventilation stimulating airway receptors or the release of pre-formed chemical mediators. Attempts to confirm the various theories have proved difficult; frequently the response to exercise is poorly reproducible, either with the same subject in the same laboratory or in different groups of subjects studied by different investigators.

Different types of exercise moreover produce different airway responses, even though the actual work done is the same. Thus, free running will produce bronchoconstriction to a greater degree and in more subjects than will running for the same length of time on a laboratory treadmill, and this in turn is more effective than exercise on a cycle ergometer. Performing a fixed work-load using the arms is a more powerful stimulus than doing the same task using the leg muscles, but surprisingly, swimming is a relatively weak stimulus to bronchoconstriction.[1] Environmental conditions also affect the response to an exercise task; many asthmatics will report that exertion that is easily performed on a summer day may be followed by an episode of wheezing if done on a frosty winter morning. And finally, failure to control environmental conditions may explain the poor reproducibility of the exercise responses recorded by some workers.

Airway heat exchange

Observations such as these led McFadden, Ingram, and their colleagues[2] in Boston to study the effects of environmental changes on the response to exercise in more detail. They found that bronchoconstriction following exercise was greatly enhanced if the air breathed during the test was cooled to sub-freezing temperatures, whereas it was abolished completely if the inspired air was prewarmed to body temperature and fully saturated with water vapour. Breathing cold air at rest had little effect on the airways, but if the levels of ventilation were increased to those seen with mild exercise, then increasing bronchoconstriction was recorded.

Now it had previously been thought that air entering the tracheo-bronchial tree was fully conditioned by the upper air passages, but by passing a thermocouple down a fibre-optic bronchoscope directly into the lower bronchial tree it was shown that significant changes in temperature do occur during deep breathing of room air, especially if the inspired gas is cooled to sub-freezing temperature.

On these results, the Boston workers put forward the hypothesis that respiratory heat exchange (RHE) is the initiating stimulus to exercise-induced bronchoconstriction.[3] They proposed that exercise hyperventilation overwhelms the capacity of the upper airways to condition the inspired air so the air reaching the tracheo-bronchial tree then needs to be warmed and saturated with water-vapour; both these processes require heat to be given up by the respiratory mucosa. On expiration some of the heat is regained as water condenses out of the air once more, but the net effect is a loss of heat from the respiratory mucosa, and it is this cooling effect that triggers bronchoconstriction. By measuring inspired and expired air temperatures and water content it is possible to calculate the RHE during a challenge test, and to demonstrate a linear relationship between RHE and the degree of subsequent bronchoconstriction.

The discrepancies explained

This theory explains many of the earlier observations in exercise-induced asthma. The importance of muscular work is only as a stimulus to increased minute ventilation, which in turn leads to airway cooling. The large muscle groups of the legs can accomplish a given work-load at a lower ventilatory cost than the arm muscles; airway cooling is therefore greater with arm exercise and the degree of subsequent bronchoconstriction is also greater. The air above the surface of a swimming pool is warm and fully saturated with water vapour so that RHE during swimming is less than during running. Further confirmation that airway cooling is the stimulus to bronchoconstriction after exercise came from experiments in which the RHE during an exercise challenge was calculated; an identical degree of airway cooling was then achieved by voluntary hyperventilation *without* exercise and identical increases in airways resistance produced. By using dry air at sub-freezing temperatures, airway cooling can be achieved at lower levels and with shorter periods of hyperventilation, thus making the challenge easier and less unpleasant for the patient. By using such a system, the challenge is more easily controlled and the results more reproducible than when relying on air at ambient temperature and humidity. In addition, by using sub-freezing air at very high levels of ventilation, much higher degrees of airway cooling can be achieved than with ambient-air exercise alone, and by using challenges of this sort, "normal" subjects will also show significant bronchoconstriction.

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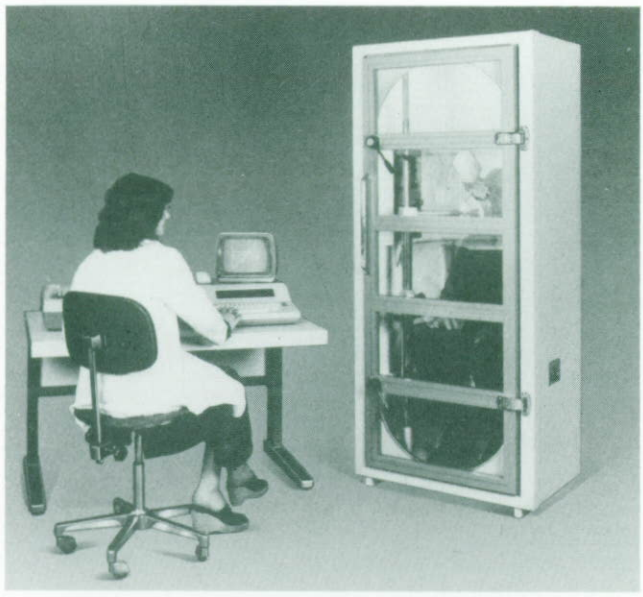
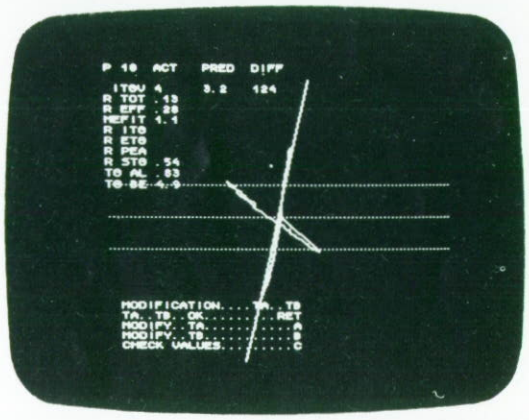
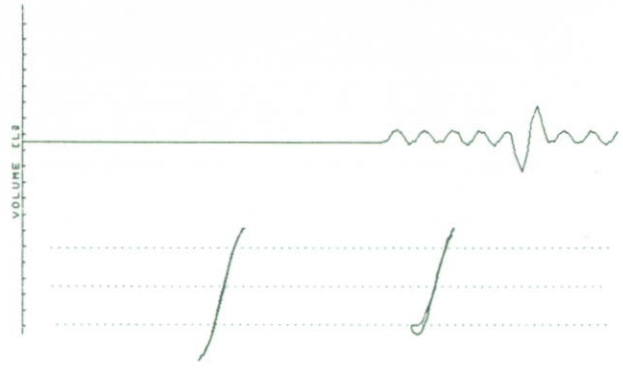




ID. # --235--
NAME EICHLER
FIRST M.RUEDIGER

AGE [YEARS] 32
HEIGHT [CM] 195
WEIGHT [KG] 98
SEX M

DATE 05.07.82
TEMP.[DEG C] 21
BAR.PR.[MB] 1012
HUMID. [%] 56



BODYPLETHYSMOGRAPHY

PARAMETER # 11	PRED	1.ACT	%PRED	2.ACT	%PRED	%2/1
R AW.....KPA/L/S		.22		.3		136
SR AW.....KPA/S		.92		1.2		130
G AW.....L/S/KPA		4.4		3.3		74
SG AW.....S/KPA		1		.82		81
CORR.....%..		.87		.86		98
ITGV.....L..	3.2	4	124	4	124	100
RV.....L..	1.8	2.1	116	2.1	116	99
TLC.....L..	8	6.7	83	6.7	83	100
ITGV % TLC.....%..		60.7		59.7		98
RV % TLC.....%..		31.8		32		100
VC IN.....L..	6.2	4.5	72	4.5	72	100

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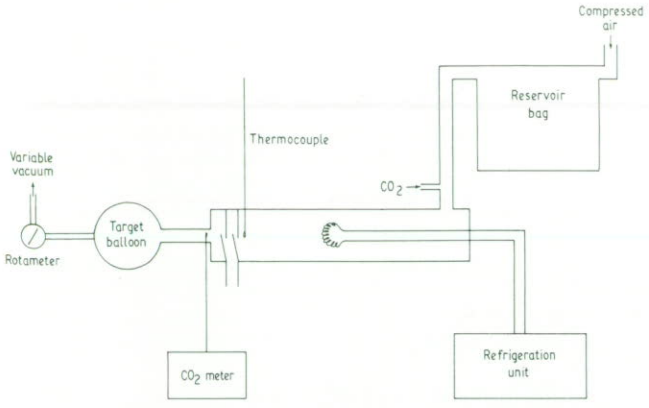

Fig. 1. Schematic representation of the apparatus used in a cold air challenge. Air from the reservoir bags passes across the cooling coil before reaching the subject via a valve system to separate the inspired and expired air.

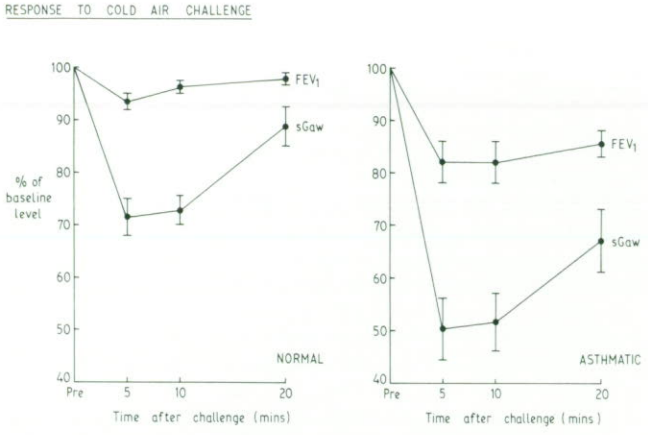

Fig. 2. Changes in FEV_1 and airway conductance (SGAW) following a cold air challenge in a group of normal (left) and asthmatic (right) subjects. Mean ± S.E.

The cold air challenge test

The apparatus used for delivering a cold air challenge in our laboratory is shown in Fig. 1. There is a central, heavily insulated PVC cylinder, inside which is the cooling coil of a refrigeration unit which operates in the same way as a domestic refrigerator. Compressed dry air is fed into the cooling chamber via a reservoir bag and this passes over the cooling coil before reaching the valve-box which contains two one-way valves to separate the inspired and expired gas. To maintain the sub-freezing temperatures, it is important to continue the insulation up to the subject's lips. Inspired air temperature is measured by a thermocouple situated just upstream of the inspiratory valve. End-tidal CO_2 concentration is analysed continuously and CO_2 is fed into the inspired air to maintain eucapnia. The target balloon in the expiratory circuit is filled prior to the start of the challenge and the level of ventilation is set by instructing the subject to keep the balloon inflated, whilst it is being evacuated through a rotameter at a set rate by a variable vacuum pump. The degree of cooling of the inspired air is highly reproducible at a given flow-rate and challenging the same subject with the same flow-rate on different occasions produces comparable results. Inspired air temperatures are of the order of -20°C and using such a test, all asthmatics (not only those who wheeze on exercise) will develop bronchoconstriction following three minutes of breathing at a minute ventilation of fifteen times their FEV_1. Normal subjects will bronchoconstrict less markedly after breathing at twice this rate, which approaches 80% of their maximal breathing capacity.

Fig. 2 shows the effects of a cold air challenge in a group of normal and asthmatic subjects. Measurements of FEV_1 and airways conductance using the body plethysmograph have been made before and for twenty minutes after the challenge. In the normal subjects FEV_1 falls by 6-10% and conductance by 30% at 5 and 10 minutes after the challenge. By 20 minutes both parameters have returned to normal. Both parameters fall more severely in asthmatics and have not completely recovered by 20 minutes after the challenge, though most are back to their starting point by 45 minutes.

Limitations of the theory

Attractive as the RHE theory is in accounting for exercise-induced asthma, the response to airway cooling does not entirely mimic the response to exercise. A well-recognised feature of exercise induced asthma is the existence of a refractory period after exercise during which a repeated exercise test fails to elicit a further episode of wheezing. No such refractory period exists to cold air challenge. A rise in circulating levels of mediators such as histamine can be detected in the blood of asthmatics after an exercise challenge, but when the same subjects undergo a cold air challenge that produces the same degree of broncho-constriction no such rise in circulating mediators can be detected. Clearly we are still some way from a complete understanding of what occurs in the lungs of an asthmatic during exercise.

Assessment of asthma

Apart from its research applications, the cold air challenge also has important clinical potential in the diagnosis of asthma and in assessing appropriate treatment for the patient. A characteristic feature of asthma is increased bronchial reactivity, and it is useful to be able to detect such hyper-reactivity in a patient who complains of episodes of shortness of breath or chest tightness and in whom there is doubt about the diagnosis of asthma. Usually this involves inhaling increasing doses of histamine or methacholine until wheezing is produced. For safety reasons, it is necessary to start at very low concentrations which are gradually increased, a time-consuming procedure. Inhaling histamine in concentrations that produce wheezing can also be rather unpleasant and as well as producing cough, can give rise to flushing, skin irritation and headache. A diagnostic challenge using cold air can be a much shorter procedure and is better tolerated by the patient. Sensitivity to cold air and to pharmacological agents is very similar[4] and so this is a valid technique for confirming a diagnosis of asthma.

Control of exercise-induced asthma

Exercise-induced wheezing can be a difficult symptom to control satisfactorily, particularly in the young person who wishes to participate in vigorous sport. Standard doses of cromoglycate or beta-sympathomimetic agents may appear

effective during an exercise test in the laboratory, but may be unable to control symptoms when the patient is pursuing his sporting interests. By using cold air as a challenge, the effects of airway cooling can be studied in a dose-response fashion that is not possible with exercise-testing; it then becomes apparent that these agents do not block the response in an all-or-none manner, but the protection afforded can be overcome if the RHE is increased.[5] One or two puffs of a beta-agonist may control the wheezing induced by a brisk walk on a summer day, but cross-country skiing may require four or eight puffs of the same agent. By devising appropriate treatment regimes in this way it should be possible to free the asthmatic from any limitations imposed by exercise-induced symptoms.

The cold air generator has made an important contribution to our understanding of exercise-induced asthma. Besides its research application it also has potential as a useful clinical tool in respiratory function testing.

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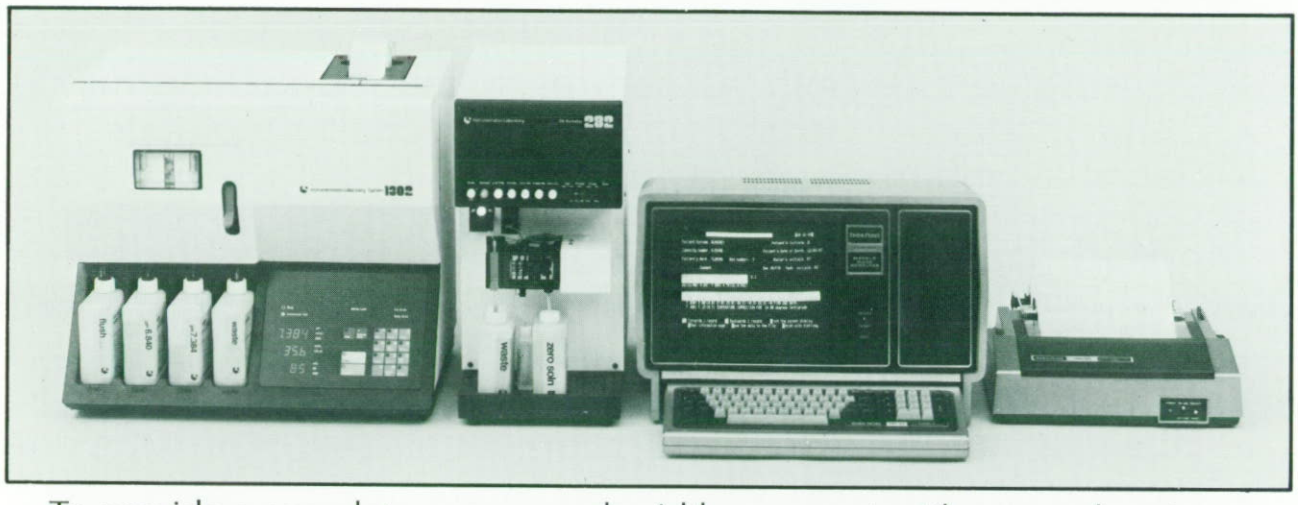

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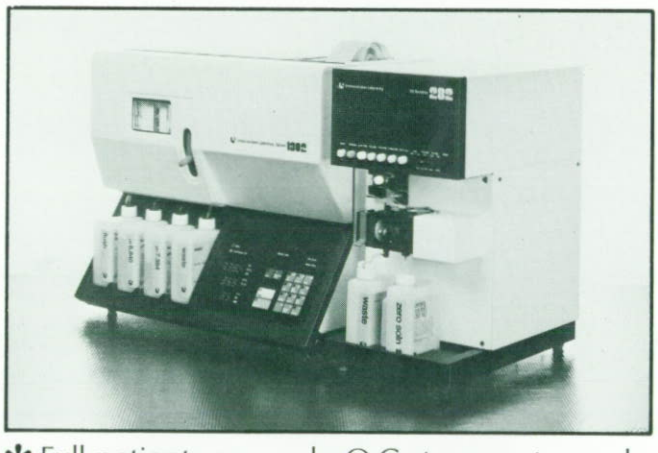

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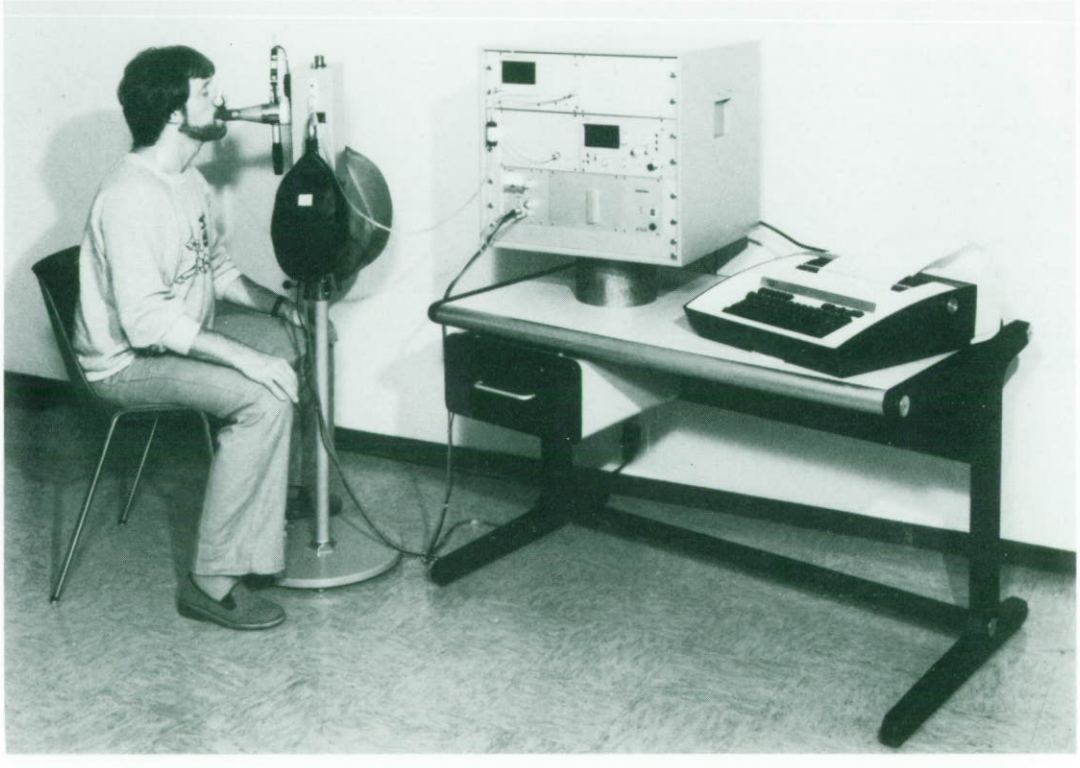

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COCCIDIOIDOMYCOSIS: An Exotic Disorder

Richard Coulden
Whipps Cross Hospital, London, E.11

This disease with the long and unpronounceable* name is limited entirely to the Western Hemisphere of this planet. It is a fungus infection which was first reported in Argentina in 1892, though the majority of cases are found in the South-Western United States and Northern Mexico and especially in the San Joaquin valley of central California.

In spite of this limited endemic area the disease should be of considerable interest to us in the Old World; the chest is involved in the majority of cases and chronic infection may develop which has similarities to tuberculosis. With the wide travel facilities now available, we may well see such cases from time to time.

Coccidioidomycosis rarely arises outside the specific endemic areas, but within those areas is a common disease; it accounts for some 100,000 cases and 70 deaths per year in the US. The fungus (known as *Coccidioides immitis*) is a saprophytic soil organism and the disease has this very limited endemic area because of the exacting requirements of its life cycle; the San Joaquin valley provides a semi-arid climate with a short rainy season which are just the conditions this fungus needs to thrive. The human species is not a necessary stage in the life cycle and man is affected quite accidentally. Outbreaks of the disease usually follow dust storms or man-made disturbances of the infected soil and the so-called 'valley fever' is caused by the parasitic phase of the life cycle, the infecting component being known as the 'arthrospore'. The life cycle is illustrated in Fig. 1.

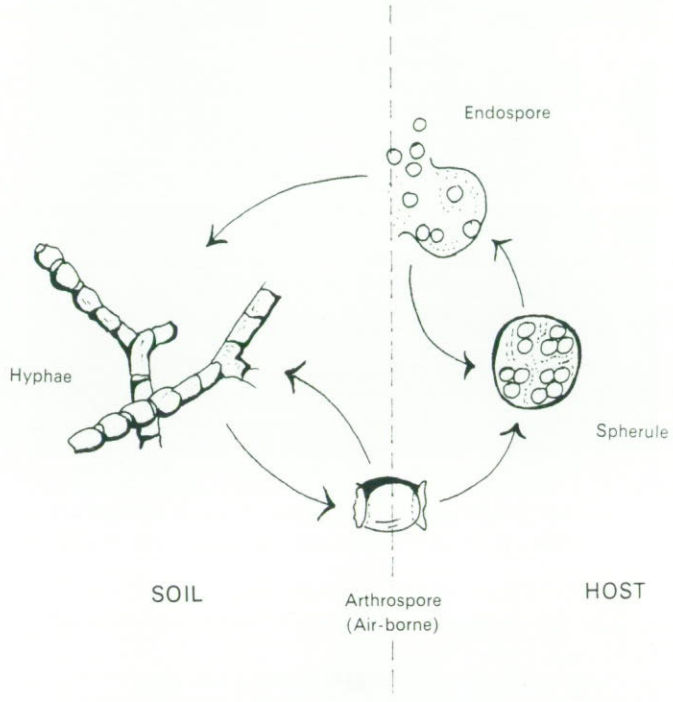

Fig. 1: Life cycle of coccidioidomycosis: the fungus resides in soil as septate hyphae. The hyphae give rise to spores (arthrospores) which are thick walled, resist drying and easily become airborne. The arthrospore gives rise to the spherule which reproduces itself as an endospore. The endospore may short-circuit the cycle within the living host to give rise to spherules or may, if placed in suitable soil, develop into the saprophytic phase. This means that the disease cannot be transferred from man to man, since only the arthrospore is infective.

Clinical Presentation

The disease may be subdivided into *primary* and *disseminated* forms; the majority of the primary cases arise from the inhalation of arthrospores, giving respiratory tract infections but accidental percutaneous inoculation giving local disease can occur.

Following inhalation of the arthrospore and its subsequent development, a granulomatous host reaction arises (rather like the more familiar tubercle). This cell mediated response limits the extent of the illness and provides life long immunity under ordinary circumstances. About 80% of the cases are sub-clinical but the remainder may develop respiratory symptoms such as cough or chest pain, or extra-pulmonary symptoms such as joint pains, conjunctivitis or 'erythema nodosum', the painful swellings on the lower leg produced by allergic vasculitis and also seen in TB or sarcoidosis. Opacities on the chest radiograph can occur in any part of the lung fields and are often seen in association with lymph gland enlargement on the same side of the thorax. These opacities usually clear in 3 to 5 weeks, but persistent pneumonic activity may indicate impending dissemination of the disease. Fig. 2.

The following syndromes of persistent pneumonic activity have been recognised:

1. Persistent lymphadenopathy, particularly peritracheal or mediastinal node involvement: this is a pre-dissemination syndrome.
2. Chronic pulmonary coccidioidomycosis progressive over years, especially in the upper lobes. Defective cellular immunity or chronic airflow obstruction are predisposing factors.
3. Cavitation within the areas of previous consolidation. The cavities are coccidioidal abscesses containing fungal elements, and may be complicated by bronchiectasis, pneumothorax and empyema. Haemoptysis occurs in two thirds of cases.
4. Coccidioidal nodules: granulomata 1-4cm in diameter which may readily be mistaken for secondary neoplastic deposits.

Extrapulmonary spread accounts for less than 1% of cases but all the deaths. The susceptibility to dissemination is unpredictable, but the black and brown races are more susceptible than whites. Immunosuppression, pregnancy and diabetes are all factors favouring dissemination. Any organ can be affected, but meningitis is one of the most dangerous complications.

* Try: Cox-idi-oido-mycosis.

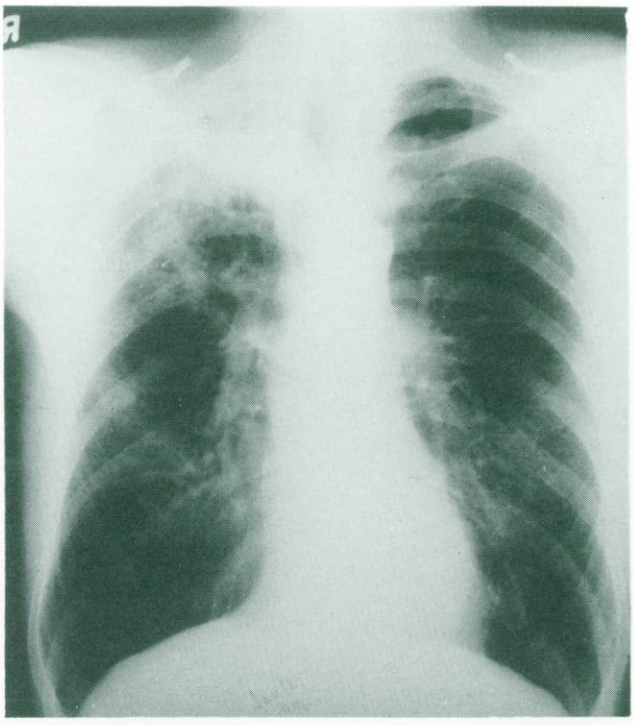

Fig. 2: Coccidioidomycosis, clearly showing diffuse infiltration of the right upper zone with contraction of the right upper lobe, and small nodular opacities in the right mid-zone.

Organ involvement is listed in order of decreasing frequency:
— Skin: granulomatous ulcers, subcutaneous abscesses with sinus formation
— Bone and Joint: osteomyelitis, synovitis, etc.
— Meningitis: meningitis and hydrocephalus
— Lymph nodes
— Urogenital tract.

Dissemination may give secondary involvement of the lung, haematogenous spread of organisms giving rise to a miliary appearance on the chest radiograph, again similar to tuberculosis.

Diagnosis

The diagnosis of primary coccidioidomycosis must be considered in any patient from the endemic area with radiographic changes and cough, fever, arthralgia or rash. Serology gives precipitins (IgM) in the first 2 to 4 weeks, and complement-fixing antibodies (IgG) after that. A skin test similar to the Mantoux with intradermal injection of 'coccidioidin' (an extract of the organism) is also available. The test is only significant if shown to convert from negative to positive within 2 weeks. Reversal of skin test (positive to negative) is an indication of poor prognosis and suggests dissemination. Diagnosis can also be made by isolation or microscopy of sputum, pus or biopsy specimen.

Treatment

Symptomatic treatment only is required for primary infection, although surgery may be required for pulmonary complications such as prolonged haemoptysis, ruptured cavity or secondary infection resistant to therapy. The only effective chemotherapeutic agent availab. is amphotericin B which is used in all disseminated and pre-disseminated states. Immunosuppressed patients, pregnant women and diabetics are included for treatment in the pre-disseminated group. Treatment is also given in primary coccidioidomycosis with serological titres, in chronic pulmonary forms and to cover surgery of infected lung.

Coccidioidomycosis is rare outside the endemic areas, but its similarity in pathology, symptomatology and radiographic changes to TB and some other granulomatous diseases means that it is a diagnosis which can easily be missed. In patients from the endemic area or patients known to travel, not all that looks like TB is TB.

RESPIRATORY PHYSIOLOGY DEPARTMENT
PAPWORTH HOSPITAL, PAPWORTH EVERARD, CAMBRIDGE

BASIC GRADE PHYSIOLOGICAL MEASUREMENT TECHNICIAN

A basic grade physiological measurement technician with experience in respiratory physiology is required for the above department to perform routine lung function tests.

Salary — £4,204 - £5,381

For further information contact Mrs. S. E. Gough:—
Telephone number Huntingdon 830541, Extension 310

Job description and application forms from:—
The Administrator, Papworth Hospital, Papworth Everard, Cambridge.

MID-STAFFORDSHIRE HEALTH AUTHORITY

SENIOR PHYSIOLOGICAL MEASUREMENT TECHNICIAN

Applications are invited for a vacancy in the Lung Function Laboratory of the Staffordshire General Infirmary and the new Stafford District General Hospital, a recently completed, newly equipped D.G.H. The Lung Function Laboratory serves a population of approximately 300,000 people. Stafford is the county town, set in attractive countryside within easy reach of the Peak District National Park. Local industries include farming, manufacturing and coal mining. Pneumoconiosis assessments are also done for the local pottery industry.

Facilities at the new D.G.H. include a screening out-patient lung function laboratory with computerised spirometry, blood gas analysis and treadmill exercise testing. The main Lung Function Laboratory is at the Staffordshire General Infirmary and is equipped with Gould 5000 IV computerised equipment for spirometry, flow volume loops, nitrogen washout and helium dilution lung volumes and gas transfer factor measurements; blood gas analysis; exerise stress testing; compliance; fibreoptic bronchoscopy; bronchial challenge tests.

The successful candidate would join a team of two technicians already in post. Candidates should have two years' experience as a technician. Previous experience in research would be of value.

Salary scale: £5193 at age 18, £5740 at age 21, rising to £6506 p.a.

Application forms and job descriptions available from Mr. G. Nuttall, Deputy Admnistrator, Staffordshire General Infirmary, Foregate Street, Stafford. Tel. Stafford 58251.

Closing date: 5th November 1982.
Please quote reference B2 in all correspondence.

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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