ASSOCIATION OF RESPIRATORY TECHNICIANS AND PHYSIOLOGISTS

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BREATH

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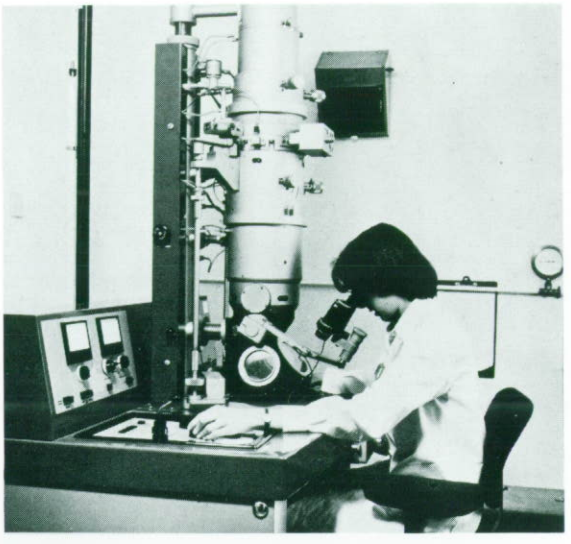

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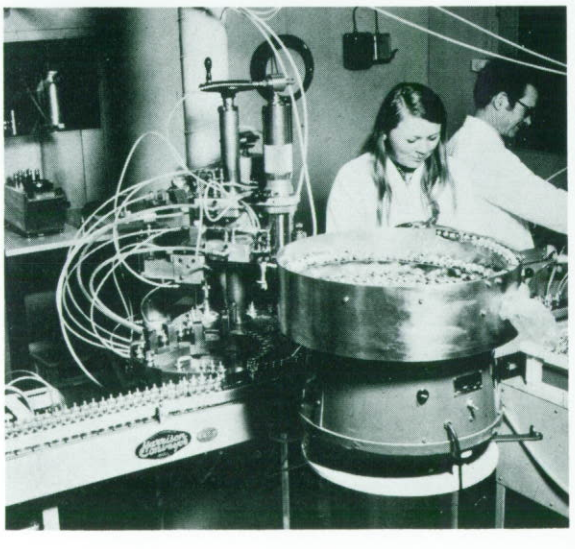

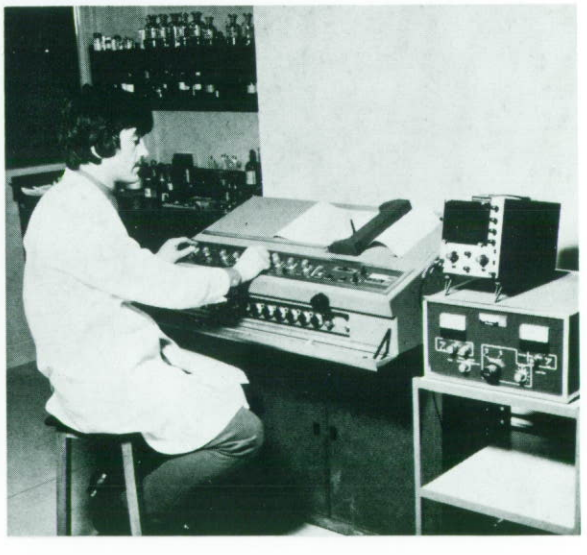

Significant advances in therapy from Original British Research

WHY WE HAVE A TRAINING MANUAL

Sally Gough,
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We have a training manual for the simple reason that student technicians need to be trained; stating the obvious, one might think, but a look backwards in time will demonstrate that, not so long ago, the idea of technician training in Respiratory Physiology barely existed.

Many technicians in this field will remember when our discipline was quite unrecognised. We were graded on a number of different scales or were paid in an ad hoc manner; some were working as medical laboratory technicians while others came from the nursing profession; and our profession was not even recognised by the Whitley Council.

In 1974, in response to the Zuckerman Report, a meeting of the various technical disciplines involved in Medical Physics and Physiological Measurement met in Manchester; a number of these groups had already formed themselves into individual associations and societies and the members felt that they could best be represented by combining to form a single unit which was to become the body known as the 'Federated Associations of Medical Technology' (the FAMT). The FAMT was then made up of Medical Physics, Cardiology, Neurophysiology, Renal, Perfusionist, Dental and Anaesthetic technicians and was joined soon after its foundation by the newly-formed ARTP. Among its main aims, the FAMT offered to advise on the education and training of staff in any of its constituent disciplines, to establish equivalent grading levels and generally to uphold professional and ethical standards. It soon became clear that if credibility was to be given to these aims, it was essential that the training and education of student technicians would have to be one of its very first tasks.

At about this time, the Colleges of Further Education were changing courses from ONC to O TEC and there was a good deal of discussion about this between the FAMT and the Technician Education Council (TEC). It was hoped that technician training could be included in the new O TEC course, but it was soon found that the TEC would only recognise an academic syllabus. We then discussed at length how a recognised in-service training programme could best be achieved and it was felt that this could be done by converting the O TEC certificate into a Diploma course. A great deal of work from interested parties in all disciplines was then put into the formulation of a log book with the appropriate academic material to cover the extra hours needed for a Diploma. The outcome was the "Orange Report" of October 1979 (in full — The Report of the DHSS Working Party on the Training of Medical Physics and Physiological Measurement Technicians within the TEC System). This was discussed in Harrogate in August 1981 but to our disappointment a Diploma appeared to be quite unacceptable, nor was there any agreement to recognise a national training scheme.

The *Whitley Council* conditions of service were changed at about this time and it is important to note in detail the conditions for entry into employment as a Physiological Measurement Technician. (It was, by the way, of great interest to note the recognition by the Council of the ARTP's existence!)

> Physiological Measurement Technicians are employed in a department of audiology, cardiology, neuro-physiology, or an allied discipline and are engaged mainly in recording, measuring and monitoring physiological changes in patients and in routine maintenance of the equipment. Entry to the Technician Grade is open to those who have followed a two-year course of *in-service training* during which time they have obtained the ONC in Physiological Measurement or the O TEC Certificate in Medical Physics and Physiological Measurement or have obtained an appropriate equivalent qualification.

So we are presented with a condition of service that cannot be fulfilled! After the Harrogate meeting therefore, a number of the Working Party members together with interested Regional Training Officers decided to get together and produce a Training Manual for all disciplines.

So far as we are concerned, this task was completed in April 1982. The DHSS undertook to cover the cost of printing and a second meeting took place in Harrogate in June 1982 to launch the manuals and obtain the opinions of those who would actually be using them in the instruction of students. The numbers attending necessarily had to be limited and we anticipated that Regional Training Officers and Scientific Officers would be able to introduce the manual to a wider range of staff throughout the country. The response to this was rather less than we had hoped for, so in March 1983 the ARTP arranged a Workshop (reported elsewhere in this issue of 'Breath') to reach those not yet familiar with the Manual.

The Manual

The manual itself contains sections on training of student technicians in Audiology, Cardiology, Neurophysiology, Perfusion and Respiratory Physiology. Each section is divided into 'Foundation' and 'Basic' Training Units and for Respiratory Physiology the details are as follows:

Foundation Unit

This section is an introduction to some of the routine measurements. The student is given an opportunity to perform the simpler tests on normal subjects and to learn the principles of each test, the equipment used and the calculations needed.

By the end of the foundation training the student would be expected to understand the rules of laboratory safety, elementary lung physiology and to be able to perform and calculate simple lung function measurements and to have observed other routine tests such as gas transfer and lung volume measurements. A minimum of four weeks is recommended for the foundation training.

Basic Training Unit

The aim of the basic training period is to establish the theoretical knowledge and skills required for a basic grade technician. By the end of training the student should understand the function and organisation of a respiratory laboratory, understand the structure and function of the respiratory system, be able to perform the basic lung function tests unsupervised and to relate the results to the respiratory disorder and finally to understand the principles of the equipment used and to be able to perform routine maintenance.

The Education Committee believes that practical work would be the most appropriate teaching method for achieving the majority of the objectives and that *only those measurements made during routine testing* should form the basis of practical training; student technicians would not be confronted during training with equipment that would not otherwise be readily accessible to them. The student would be expected to have observed some of the more sophisticated methods of measurement of lung function testing and they might well have to leave the home base to do this.

Doubtless there will be disagreements on the details of the Manual but after very lengthy discussions, we believe that it now forms the basis for a recognised training programme. It must be stressed that there is as yet no agreement on a policy for national assessment of the Manual, though some regions have moved a long way in this direction.

The credibility of our profession to provide a competent service depends on our being able to train technicians to a nationally recognised level; we must take the responsibility *now* for the quality of care which patients will receive in future years.

BREATHLESSNESS IN CHRONIC LUNG DISEASE

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Breathlessness is the most frequently reported symptom among patients with chest disease; it causes much suffering and disability in those with chronic bronchitis and emphysema which occur so commonly in the U.K. Despite this, breathlessness is rarely measured and its occurrence in some patients and not in others with apparently equivalent functional impairment is unexplained[1]. The airways obstruction in these patients may be only partially responsive to conventional treatment with bronchodilators and steroids, and there is a need for a safe symptomatic remedy for breathlessness[2]. Much interest has recently been focused on the actual measurement of breathlessness and on treatments aimed not at improving lung function but at the symptom itself.

What is breathlessness?

The terms 'breathlessness' and 'dyspnoea' are commonly used interchangeably and indiscriminately. Etymologically, 'dyspnoea' means 'difficulty in breathing', but the patients complain of breathlessness and not of dyspnoea. The term breathlessness has the advantage of being understood by both patients and doctors and does not invoke arguments on whether the sensation is really appropriate to the loss of lung function.

Measurement of disability

a) *Dyspnoea Grade*

This scale was first introduced by Fletcher and modified by McGavin et al[3] (Table 1). Patients are asked to circle the grade which most accurately describes the severity of their disability. This grading scale is useful in the initial assessment of patients with chronic airways obstruction.

Table 1
DYSPNOEA GRADE

THIS WEEK HAVE YOU BEEN:

5. Breathless at rest or on mimimal effort.
4. Able to walk about 100 yards on the level.
3. Able to walk for 1 mile on the level at own pace but unable to keep up with people of similar age.
2. Able to keep up with people of similar age on level but not on hills or stairs.
1. Normal.

Exercise tolerance limited by other factors — Yes. No.
If YES list factors .....

b) *Oxygen Cost Diagram*

In the oxygen cost diagram everyday activities are ranked according to their cost in terms of oxygen consumption along a 100 mm line (Fig. 1).

c) *Walking Test*

A twelve minute walking test was introduced by McGavin et al[3] as a method of assessing disability in chronic bronchitis and it has been useful as a method of monitoring treatment and disease progress without the complexities of conventional exercise testing. It may also confirm symptomatic improvement in the absence of spirometric improvement. Instructions are standardised and the subjects are asked to cover as much ground as possible in 12 minutes in a covered hospital corridor. The test is reproducible after two practice walks. A shorter six minute walk which is more convenient and less distressing to the patients has been shown to be equally reliable[4].

d) *Cycle Ergometer*

Complex physiological changes during exercise are monitored by conventional exercise testing on a cycle ergometer. The ventilatory response to exercise, oxygen consumption and CO_2 production, A-a O_2 gradient and arterialised ear lobe blood gases all provide useful information in the initial assessment of patients. This technique is however too cumbersome for patient follow-up and cycling is unlike most everyday activities.

e) *Pedometer*

This is a device for measuring the distance walked and is the only way of measuring mobility objectively as an out-patient. The pedometer is attached at the waist and detects the vertical movements occurring with each step; it can compute the distance walked when programmed with the stride length.

f) *Breathlessness on Treadmill Testing*

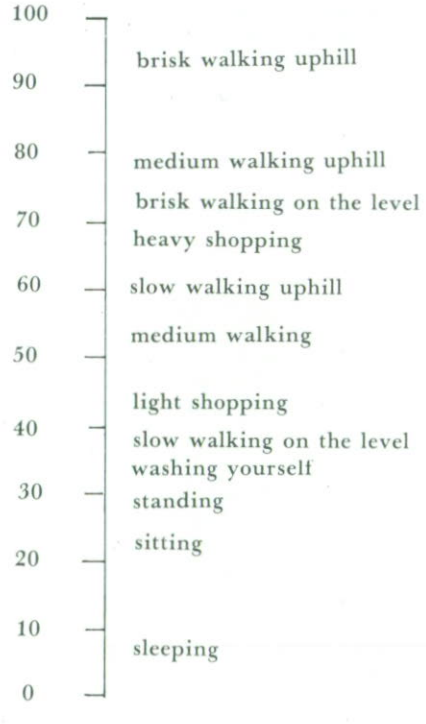
We have recently validated the use of visual analogue scales to assess breathlessness during treadmill exercise in both normal subjects and patients with chronic lung disease. Subjects walk on a treadmill on the flat, with logarithmic increase in speed each minute starting at the lowest speed (1.3, 1.8, 2.5, 3.3, 4.4, 5.9, 8.0 and 10.8 km/hr). This enables subjects with a wide range of disabilities to be stressed and made breathless. At the end of each minute, subjects score the severity of breathlessness on a 10 cm visual analogue scale ranging from *not breathless* to *extremely breathless*. Instructions are standardised for each patient ('How breathless are you now?'). By fixing the speed at which the subject walks, we are able to compare breathlessness in studies of drugs which might relieve this symptom. We do not monitor ventilation during this test since the use of a mask increases the sensation of breathlessness and may obscure any benefits.

Treatments for breathlessness

Bronchodilation reduces breathlessness on treadmill exercise but since most patients with chronic lung disease are still breathless on exertion despite maximal bronchodilator and steroid therapy, other approaches have been devised.

a) *Diazepam*

A preliminary study suggested that diazepam relieved breathlessness in 'pink and puffing' patients with chronic airflow limitation. In a subsequent double-blind crossover trial[5] in eighteen patients diazepam had no effect on breathlessness and noticeably reduced exercise tolerance. One patient moreover died whilst taking the drug during an exacerbation of breathlessness.



The above tasks are ranked according to their degree of difficulty. Please mark the most difficult task you are able to perform without your breathlessness stopping you.

Fig. 1. *Oxygen cost diagram.*

b) *Portable Oxygen*

The Royal College of Physicians Thoracic Advisory Committee recently pointed out the need for further study of the effects of oxygen on breathlessness, including the efficiency and safety of oxygen systems. A double blind crossover study[6] of portable oxygen during exercise showed that both breathlessness and exercise tolerance were improved. The benefits were most obvious during the treadmill test, when there were considerable reductions in visual analogue scores of breathlessness. Pre-dosing with oxygen before exercise was also effective in reducing breathlessness; pre-dosing with oxygen for 1 minute was insufficient, but pre-dosing for 5 or 15 minutes was significantly better. Strategically placed oxygen supplies for pre-dosing (eg. at the bottom of the stairs) may improve mobility in the home. Individual patients can be tested in a blind manner, comparing portable oxygen and air on a treadmill test.

c) *Alcohol*

Many pink and puffing patients claim that they are less breathless after consumption of spirits. This was confirmed by an increase on exercise tolerance and a reduction in breathlessness (during treadmill testing) following alcohol consumption in a double-blind study[7] (double measure of Vodka). This was not due to an alteration in central sensitivity to breathlessness, but alcohol acted as a brochodilator with a small but significant improvement in forced vital capacity.

d) *Dihydrocodeine*[7]

Opiates have a profound effect on the symptom of breathlessness and have been used since the late 19th century to relieve breathlessness in asthma, pneumothorax and emphysema though we now realise that the use of these drugs can be hazardous. Morphine reduces the ventilatory response to a number of stimuli and reduces oxygen consumption in normal subjects both at rest and on exercise. Dihydrocodeine, which can conveniently be given acutely to the 'pink puffer' with chronic airflow limitation, reduces breathlessness and increases exercise tolerance. This is due to an effect of dihydrocodeine on metabolic rate since it can be shown that exercise ventilation and oxygen consumption are both reduced by approximately 10% after a dose of 1 mg/kg. When this dose was given on a long-term basis, severe side effects of nausea and vomiting were encountered but a recent trial of low dose dihydrocodeine[8] (15 mgs) before exercise (used in a similar manner to glyceryl trinitrate for angina) has shown significantly improved breathlessness on a treadmill test and improved mobility at home assessed by pedometer readings, without side effects.

e) *Cycles*

Cycling is at least twice as efficient as walking in terms of oxygen consumption for a given speed. Patients can cycle on a light weight tricycle almost 4 times the distance they can walk in six minutes, and are less breathless doing so[9]. A tricycle is ideal since patients can adopt a good breathing posture and if they become too breathless can stop without dismounting; supplemental oxygen can be carried at virtually no cost.

Summary

Patients with lung disease find it extremely difficult to describe the sensation of breathlessness which is much more complex than respiratory sensations produced experimentally. Pain is an equally complex sensation but, unlike breathlessness, attempts have been made over the years to quantify and treat it. Recently interest has focused on the measurement of breathlessness by visual analogue scales and other simple estimates of disability. The developments outlined in this paper indicate that symptomatic relief of breathlessness is a realistic aim which can be achieved and should be pursued further.

Acknowledgements

I am indebted to Drs. Duncan Geddes and Margaret Johnson for help in these studies, to Jane Jones, Derek Cramer, Liz Gross, Renza Craddock and Meg Lunt for technical help, to the Pharmacies of the Brompton Hospital and the London Chest Hospital and to Lesley Bryant, Trish Harris, Vicky Rawlinson and Angela Betchley for secretarial help.

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CYSTIC FIBROSIS: Clinical features, pathogenesis and treatment

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This article is divided into three sections: Section A is concerned with presentation and treatment and outlines some of the reasons for the improved survival of patients with cystic fibrosis. In Section B current research into the mechanisms of lung damage is discussed and in Section C we move on to the recent changes in antibiotic therapy.

A. Presentation, Treatment and Prognosis of Cystic Fibrosis

Presentation:

Cystic fibrosis (CF) is the commonest genetically inherited disorder in the United Kingdom and causes significant morbidity. The incidence is about 1 in 1,700 live births; there are 650,000 births per annum in the UK so that about 400 children with the condition are born each year. Even with today's remedies only about 70% live to the age of 17 years, though this figure appears to be improving all the time. CF is inherited as an autosomal recessive disorder but there is no proven method of detecting the condition before birth or of detecting the carrier state at any stage. Despite extensive research, the specific biochemical defect is not known.

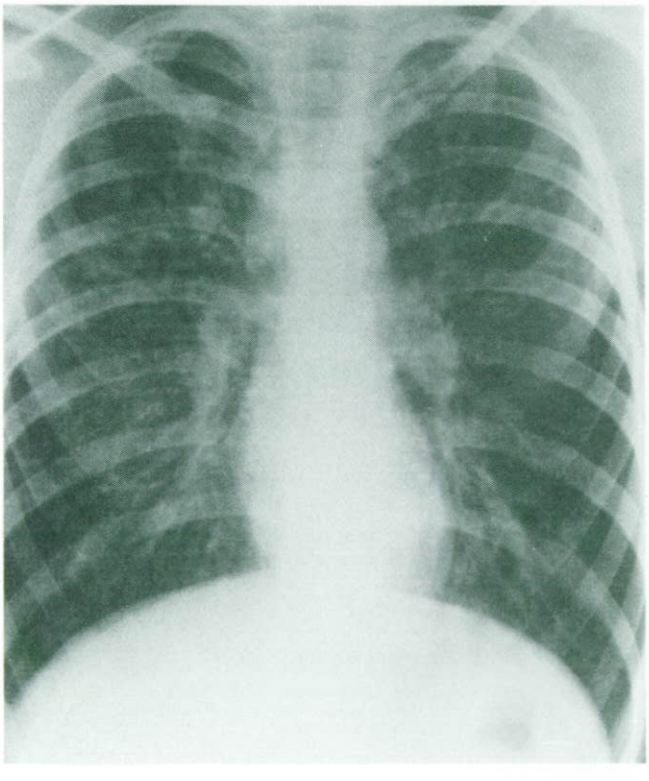

Fig. 1. *Chest radiograph from a patient with cystic fibrosis; the typical appearances resulting from bronchiectasis and inflammatory change are shown.*

The term 'cystic fibrosis' is an abbreviation for 'cystic fibrosis of the pancreas' but both are poor names for there are no true anatomical cysts and other organs (lungs and liver) are frequently affected. Mucoviscidosis is an older name seldom used today.

Cystic fibrosis has three major manifestations: 1) Progressive lung damage, (Fig. 1) 2) maldigestion and malabsorption of food and 3) excessive excretion of sodium and chloride in the sweat. The pulmonary damage is responsible for much of the morbidity and mortality in childhood and adolescence, and the excessive loss of sodium and chloride in the sweat provides the basis for the diagnostic *'Sweat Test'*, so important in diagnosis of affected children.

The age of presentation varies considerably. The diagnosis is most commonly made in infancy or early childhood but some do not present until adult life. A list of typical symptom complexes and their incidence at time of diagnosis is shown in Table 1.

TABLE 1

Presenting symptoms of cystic fibrosis.

	INCIDENCE %
Meconium ileus	11
Pulmonary and gastro-intestinal symptoms	52
Gastro-intestinal symptons only	16
Pulmonary symptoms only	17

Based on a survey of 700 cases (1952-58)

Meconium ileus affects newborn infants; the bowel is filled with thickened meconium (gastro-intestinal secretions) leading to intestinal obstruction. Children with meconium ileus or those presenting with both gastro-intestinal and pulmomary symptoms have the highest mortality rates.

In addition to varying modes of presentation, there is a wide spectrum of disease severity, ranging from a four year old child dying from cor pulmonale secondary to lung disease, to a patient living a normal life until the third decade. There is no known cause of this variation in severity in a disease which is apparently a single genetic defect. However, certain predictive factors concerning prognosis are recognised (Table 2) though the reason for the sex difference is not known.

TABLE 2

Prognostic factors in cystic fibrosis

GOOD PROGNOSIS	POOR PROGNOSIS
1) Male	Female
2) Maintains appropriate weight	Poor weight gain or loss of weight
3. Single system involvement	Gastro-intestinal and lung involvement
4) Normal chest x-ray within first year of diagnosis	Abnormal chest x-ray in first year of diagnosis

Anatomically, the lungs usually appear normal at birth though hyperplasia and obstruction of the submucosal glands of major airways may be found in the absence of pulmonary infection. The lung damage progresses with time, bronchiolitis occurring first with the later appearance of bronchiolectasis, mucous plugging, bronchitis, bronchiectasis, obstruction and chronic infection. However, all these changes may be present simultaneously at an early age.

Treatment:

Treatment is aimed at control of firstly, the gastro-intestinal symptoms by using pancreatic enzyme supplements to decrease malabsorption of fat and secondly, the chest disease by daily physiotherapy and postural drainage together with administration of regular antibiotics, especially in childhood.

Prognosis:

This appears to have improved over the last 30 years. In a survey of 550 cases diagnosed between 1939 and 1958, only 85 cases were found to have lived for longer than 10 years, and the average life expectancy of a cystic fibrosis patient diagnosed in 1950 was only 4 years. Now, 70 to 80% of patients can expect to live to the age of 18 or more.

There are several possible reasons for improved survival and these include (1) a greater awareness of the diagnosis and clinical manifestations, (2) the recognition of milder forms of the disease, (3) improved diagnostic techniques in particular the discovery in 1954 of the *Sweat Test* (measuring the sodium and chloride content of sweat) and (4) the introduction of effective antibiotics.

B. Possible mechanisms of lung damage

Many bacteria have been implicated as a cause of acute infection of the chest in CF but *Staphylococcus aureus, haemophilus influenzae* and *pseudomonas aeruginosa* are particularly common pathogens.

Pseudomonas aeruginosa is especially important because its presence in the sputum is frequently associated with a progressive downhill course. It is difficult to eradicate and we know that it is capable of producing many enzymes, some of which (protease and elastase) have been implicated in the damage to body tissues seen in patients with pseudomonas septicaemia, ocular infection and invasion of the body from a burn injury infected with pseudomonas. Antibodies to enzymes produced by pseudomonas have been found in the serum of patients with CF indicating production of such enzymes within the lung and furthermore elastase originating from other sources has been implicated in the pathogenesis of chronic bronchitis and emphysema.

We therefore undertook a study of sputum samples from CF patients to discover whether an elastase was present and if so, to determine its probable origin, i.e. whether it was produced by the pseudomonas or whether it originated from polymorphonuclear leucocytes. The idea of leucocytes producing enzymes which are detectable in sputum and which are capable of causing lung damage may seem strange, but is readily explained in the following way. When leucocytes phagocytose bacteria, enzymes are released within the cell to destroy the bacteria. However, some of the enzyme produced inevitably leaks to the outside of the cell where, to prevent it causing damage, it is 'mopped-up' by naturally occurring inhibitors. In severe inflammation however such as in purulent sputum or an abscess there may be excess of these digestive enzymes over the inhibitors when free enzymic action may readily be detected.

Thus, in sputum from CF patients there are two potential sources of elastase, bacteria and polymorphs. (Macrophages can also produce elastase but in small quantities and there few macrophages in CF sputum.) Elastase may be a particularly important enzyme because it can cause decreased mucociliary clearance, damage to ciliated epithelium and damage to types of connective tissue other than elastin. Some patients with CF have been shown to have decreased mucociliary clearance so that enzyme induced epithelial damage could lead to retention of secretions allowing conditions suitable for bacterial growth and the creation of further lung damage.

Sputum collection:

For our studies, sputum was collected from CF patients over a 3 hour period and ultracentrifuged at 50,000g to separate the *sol* phase (used for analysis and containing all the diffusible proteins) from the *gel* phase (containing unwanted cells, bacteria and mucus and which was discarded).

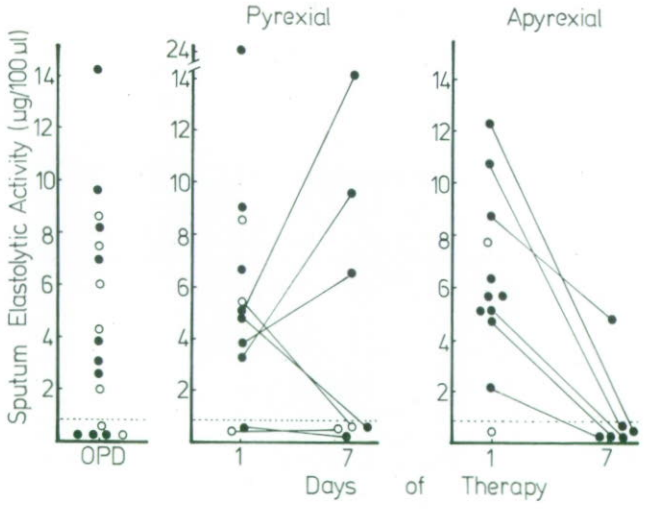

Fig. 2. *Elastase activities for out-patient and in-patient sputum samples. The dotted line indicates the lower limit of measurement. Closed circles: samples of sputum from which pseudomonas was isolated. Open circles: other organisms. The solid lines join pairs of samples (where available for analysis) collected from the same patient at day 1 (day of admission) and on the seventh day of treatment. OPD: values obtained from out-patient samples. Pyrexial and Apyrexial refer to the patients admitted to hospital for pulmonary exacerbation. Pyrexia refers to the presence of fever at the time of admission.*

The elastase activity of the sol phase was measured using a simple diffusion technique and compared to known standards of porcine pancreatic elastase. Elastase activity was expressed as $\mu g/100\mu l$ of sol phase.

We have examined the results from two groups of patients:- (A) Out-patients, i.e. samples from CF patients when seen routinely in the clinic in the apparently quiescent state, and (B) Samples from CF patients admitted to hospital with pulmonary exacerbation, with or without pyrexia at the time of admission.

The results of the analyses are shown in Figure 2. The following features are notable:- (1) The range of elastase activity was similar for both out-patient and in-patient groups. (2) Elastase activity was present in most sputum samples, whether or not pseudomonas was isolated on culture. (3) In the group admitted to hospital without a pyrexia, treatment with antibiotics over a period of 7 days was associated with a decline in enzyme activity.

In addition to the elastase activity of the sputum samples, we measured the albumin content in an attempt to assess the degree of inflammation in the bronchial tree, since albumin is believed to pass into the bronchi by passive diffusion. The sputum/serum albumin ratios were calculated to allow for any differences in serum albumin level and the results are shown for the in-patient groups in Figure 3. There was a significant difference between the pyrexial and apyrexial groups at presentation, the pyrexial groups having higher ratios than apyrexial groups suggesting a greater degree of inflammation. In the pyrexial groups there was a tendency to lower levels with 7 days of treatment.

To summarise, elastase activity is often present in sputum from both in-patients and out-patients and this is potentially harmful. We noted no difference between our groups at presentation but there was a fall in enzyme activity with treatment in the apyrexial group and this is potentially beneficial to the patient.

C. Aerosol Antibiotics

Nebulised inhaled antibiotics have been used in the treatment of CF for many years though opinions differed on their value. No good trial was undertaken until the Brompton Hospital published their results in the Lancet in 1981. This was a double blind controlled trial of twice daily inhalation of 1 gram of Carbenicillin and 80 mg of Gentamicin. The treatment period was 6 months and was compared with an equal placebo period. (The placebo was identical in taste and colour to the active preparation). Twenty patients entered the trial and seventeen completed it. FEV_1, FVC and PEFR were measured at monthly intervals; 15 patients showed improvement in these measurements when on active treatment and this was significant in eight. No patient improved when on placebo. During this trial, 7 patients were admitted to hospital whilst taking placebo but only 3 whilst inhaling the active preparation.

In the light of these results, we tried similar treatment in one of our patients with CF who was going through a particularly troublesome period. The patient aged 20, was diagnosed in infancy as a result of failure to thrive, recurrent chest infection, fatty stools and a positive sweat test. He had been treated in the customary manner with antibiotics and pancreatic enzyme supplements. However by 1977, he had a persistent cough and sputum and in 1978 pseudomonas was isolated for the first time from his sputum. Admissions now became more frequent. His illness is charted in Figure 4, which indicates the

relationship between weight, FEV_1 and admissions to hospital. Weight and FEV_1 are the most useful and readily obtainable indices of progression of the disease or well-being of the patient.

Between May 1980 and July 1981, he required seven admissions to hospital for pulmonary exacerbation. Although he often showed a little improvement in terms of weight gain and improvement in FEV_1 with each admission, overall there was an inexorable downhill progression. In desperation, we followed the lead of the Brompton Hospital and started him on aerosol antibiotics. To our delight by January, 1982, his weight had returned to its ideal value and his FEV_1 had improved significantly fro 1.9 to 2.4 litres.

Why should this treatment appear to work? We do not know and logically it should not, since only about 10% of the inhaled dose actually reaches the lungs; the dose would be small even if it were given intravenously. In addition, the drug would not be delivered to the areas which presumably most needed it, the obstructed airways. Disadvantages are the cost of about £7.30 per day, though this is much cheaper than hospital admission; secondly, resistant strains may be selected out by such therapy, though this has not yet been proved to be a problem.

It should be emphasised that not all patients with CF respond to aerosol antibiotics in such a gratifying manner and we suggest that such treatment should be restricted to those needing frequent hospital admission or to those not fit enough to lead normal lives. Clearly, their use need further careful monitoring.

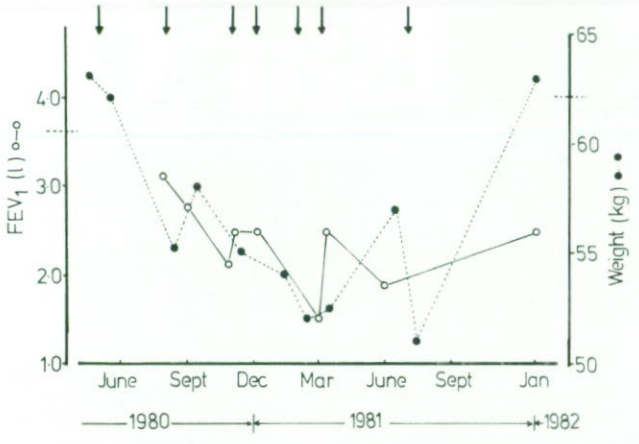

Fig. 4. *The relationship between FEV_1, weight and hospital admissions for one patient who started aerosol therapy in July, 1981. Each hospital admission is indicated by an arrow. The vertical axes show FEV_1 and weight, the ideal values for his height being indicated by the dotted lines crossing the axes. Weight is indicated by closed circles. FEV_1 by open circles. Time in months is indicated on the horizontal axis.*

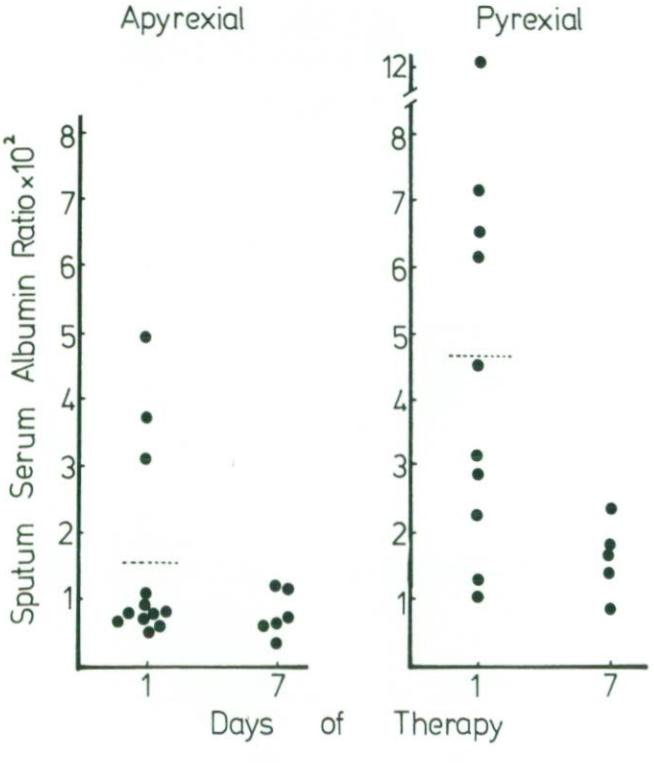

Fig. 3: *Shows sputum/serum albumin ratios for the two in-patient groups (see Fig. 2.). The dotted lines indicate means for each group.*

Further Reading

Wood R E, Boat T F, Doenshuk C F (1976). Cystic fibrosis. Am Rev Resp Dis *113* 833-878

Dann L G (1982) Cystic fibrosis: current research into antenatal diagnosis. Hospital Update *8* 131-144

Lung function in young adults with cystic fibrosis Br J Dis Chest *76* 35-43.

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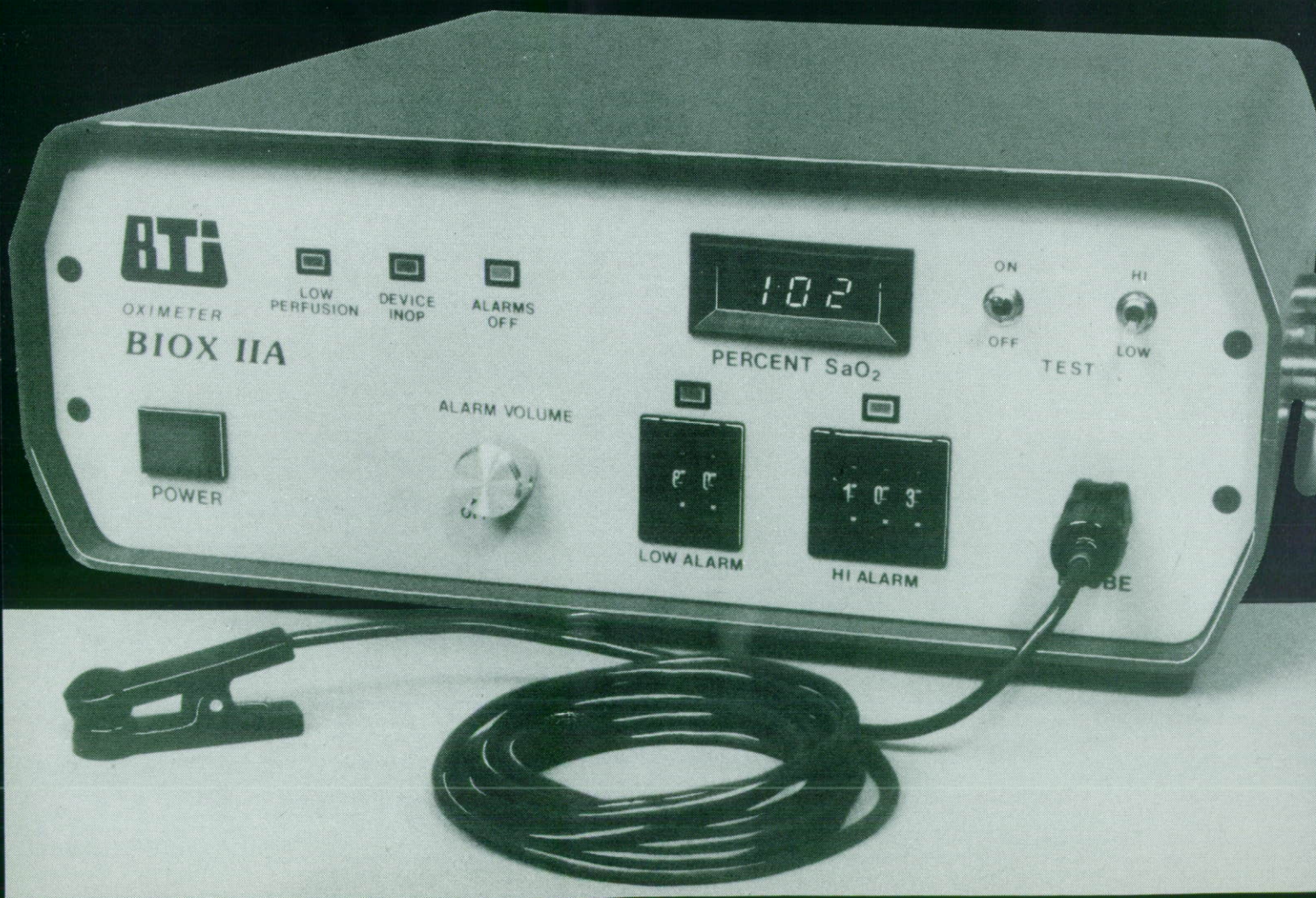

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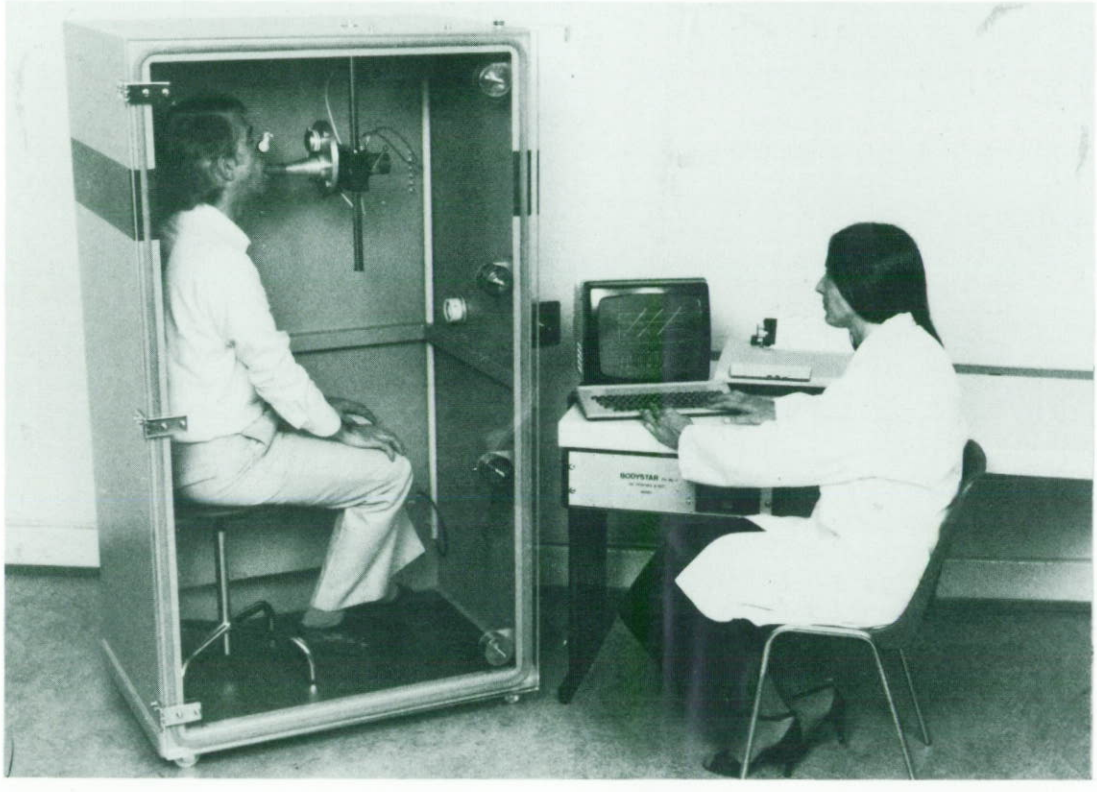

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RESPIRATORY MUSCLE FATIGUE

C. D. Shee
The London Chest Hospital, London E2

Muscle fatigue can be defined as 'a failure to sustain a given force'; we are all familiar with fatigue of the leg muscles after prolonged exercise. The muscles of respiration are also skeletal muscles and the question arises whether they too can undergo fatigue. In patients with chronic obstructive lung disease the work of breathing is greatly increased and this work is predominantly borne by the inspiratory muscles. These muscles are at a mechanical disadvantage because of hyperinflation of the chest, which adversely affects their length tension characteristics. If these muscles fatigue and fail as force generators, alveolar hypoventilation will follow leading to a fall in PaO_2 and a rise in $PaCO_2$.

The chain of command affecting respiratory muscle activation is shown in Fig. 1 and abnormalities at any level of this chain could cause ventilatory failure. Barbiturates for instance, may depress respiration by a central effect on the brain or polyneuritis by affecting the nerves supplying the muscle. I would like to concentrate on changes beyond the neuromuscular junction that can impair force generation. Experimentally, a part of such 'peripheral fatigue' can be prolonged and has been referred to as 'low frequency fatigue' as it preferentially reduces muscle force at low frequency stimulation rates (the sort of nerve firing rates that normally control the diaphragm).

Apart from severe attacks of bronchitis or asthma are there any other conditions in which respiratory muscles might become fatigued? It is possible that the immature respiratory muscles of newborn infants may fatigue when they are faced with a superadded stress such as the 'Respiratory distress syndrome'. Similarly during assisted ventilation in adults disuse atrophy of the respiratory muscles may occur which may in part account for the difficulty in weaning certain patients off ventilators. Experimentally low frequency fatigue of the diaphragm can be produced when volunteers breath through a respiratory resistance for as long as possible (Moxham et al 1981).

Factors predisposing to inspiratory muscle fatigue

One can list a number of factors that might predispose to respiratory muscle fatigue. As interest in this topic is fairly recent, a number of these suggestions are speculative.

1. Increased work of breathing e.g. airways obstruction or 'stiff lungs'.
2. Muscles working at a mechanical disadvantage e.g. hyperinflation.
3. Disuse atrophy e.g. after prolonged assisted ventilation.
4. Decreased muscle strength e.g. premature babies or poor nutrition.
5. Disorders of the chest wall e.g. obesity or kyphoscoliosis.
6. Decreased delivery of oxygen or chemical substrates for contractile processes e.g. reduced cardiac output or anaemia.
7. Severe hypoxia or acidosis.
8. Pre-existing neuromuscular disorder e.g. poliomyelitis or myasthenia gravis.

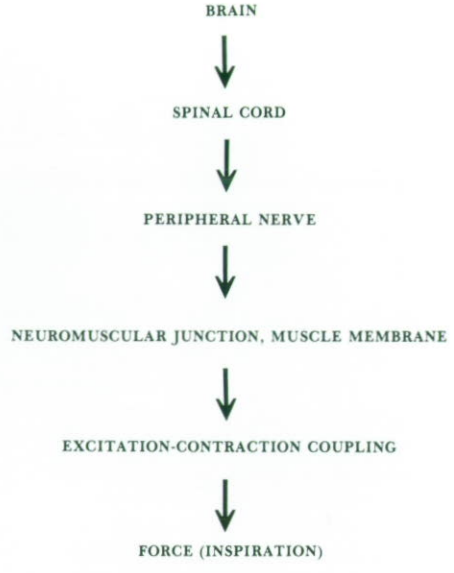

Fig. 1. *The chain of events involved in respiratory muscle function.*

Factors protecting against inspiratory muscle fatigue.

The diaphragm contracts throughout life and considering the large loads with which it may have to cope it is perhaps surprising how rarely fatigue becomes a serious matter. I have listed a few factors that may enhance resistance to fatigue.

1. The respiratory muscles contract intermittently and so have a chance to recover between breaths. Fatigue is more likely to occur when muscles contract continuously.
2. These muscles have an excellent blood supply which increases exponentially with increasing work. Blood flow limitation of contractile effort has not been shown in normal animals.
3. The diaphragm has a flat radial distribution of fibres and so during vigorous contraction the fibres do not compress each other thereby causing ischaemia as may occur in the quadriceps muscle.
4. Animal experiments suggest the diaphragm is relatively resistant to anaerobic metabolism and only produces lactate in large quantities when severely hypoxic. In isolated muscle strips I have shown that like most other skeletal muscles and unlike the heart, the diaphragm is resistant to the depressive effects of a respiratory (hypercapnic) acidosis (Fig. 2).
5. When the work of breathing is greatly increased, respiration may alternate between predominantly thoracic and predominantly abdominal breathing; this 'cycling' of the respiratory muscles allows a relative rest period for particular muscle groups and may therefore protect against the progression of fatigue. Such thoraco-abdominal incoordination has been observed in patients and it may represent a useful clinical sign of respiratory muscle fatigue.

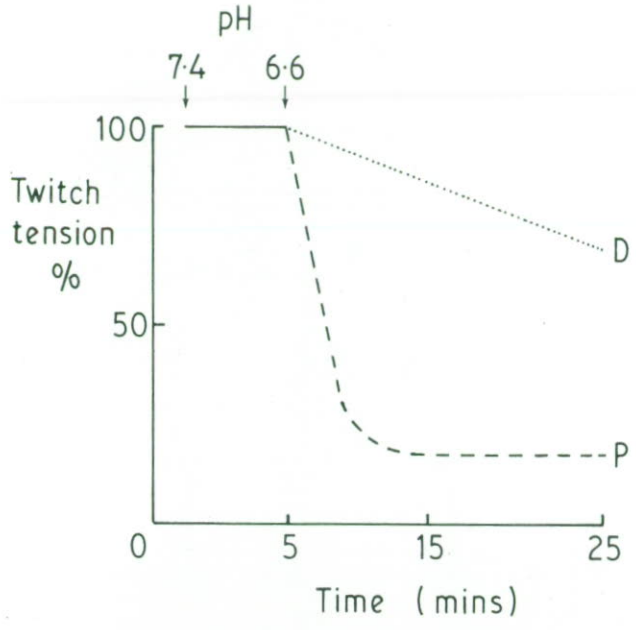

Fig. 2. The effects of hypercapnic acidosis on twitch tension of isolated rat diaphragm (D) and of cardiac papillary muscle (P). Acidosis causes a rapid and marked fall in cardiac muscle contractility; diaphragmatic contractility is relatively well preserved.

Detection of fatigue and weakness

There are certain well recognised clinical signs that respiratory failure is developing but it is obviously important to be able to detect and measure fatigue at an early stage. Weak muscles are naturally more likely to fatigue with a given load than strong ones.

A. *Decrease in Vital Capacity*

Providing patients have relatively normal lungs and perform maximally, a decrease in vital capacity may indicate muscle weakness. This test can be used in hospital to detect deterioration in patients with diseases that affecting respiratory muscle strength such as acute polyneuropathy.

B. *Maximal Mouth Pressures (P max)*

Maximal inspiratory or expiratory mouth pressures can easily be measured using a simply modified pressure gauge, as described by Black and Hyatt in 1969 (Fig. 3). Two diaphragm gauges are connected to a metal cylinder by rigid plastic tubing; one gauge records negative pressure and the other positive pressure. The distal end of the cylinder is closed except for a small opening that prevents the facial muscles from producing significant pressures. Maximal expiratory pressure is measured near total lung capacity and maximal inspiratory pressure near residual volume. Maximum inspiratory mouth pressure is of particular importance because it is the inspiratory muscles that are most vital to ventilation. P max involves a complicated mixture of different respiratory muscles and values can increase due to a learning effect and depend very much on the subject's motivation. Despite these limitations, P max can be a useful guide to respiratory muscle strength.

C. *Transdiaphragmatic pressure (Pdi)*

This technique allows us to measure the strength of diaphragmatic contraction. Two balloons are passed through the nose, one into the stomach and one into the lower oesophagus. Pdi is derived by subtracting oesophageal from gastric pressure. Pdi (max) is measured during a maximal inspiratory manoeuvre. In one individual Pdi is fairly reproducible but there is a large variation between subjects. The technique is slightly uncomfortable for the patient, and careful positioning of the balloons is essential for a useful result. At the Brompton Hospital Pdi measurement during a maximal sniffing manoeuvre is currently being assessed and it is possibly a better measurement of diaphragm strength than the static manoeuvre (Miller et al 1983).

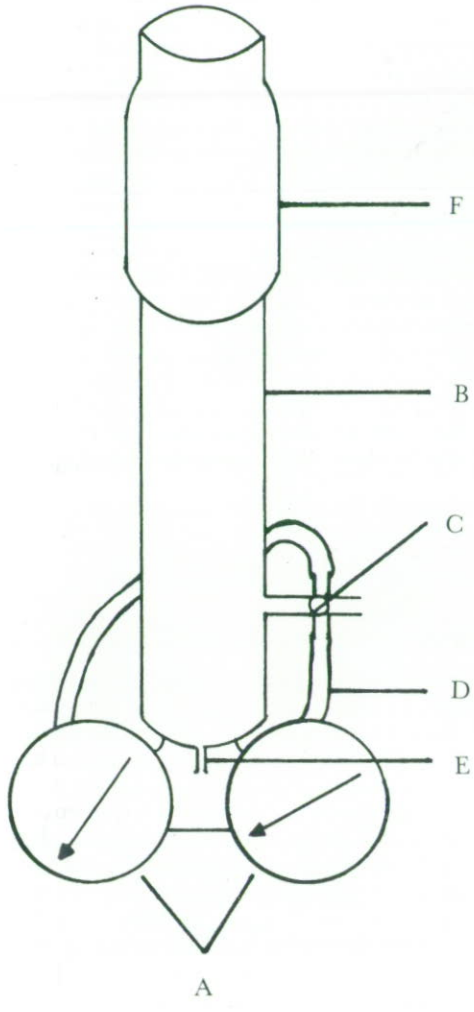

Fig. 3. Instrument used to measure maximal inspiratory and expiratory mouth pressures (after Black and Hyatt, 1969). A: Inspiratory and expiratory pressure manometers which are alternately connected to cylinder B via three-way Tap C. D: rigid plastic tubing. E: small outlet (internal diameter 2 mm). F: mouthpiece.

D. *Pdi following phrenic nerve stimulation*

Reduction in Pdi (max) may be due to fatigue of the muscles themselves or to failure of central drive, where the patient is unable to exert maximal effort. To bypass the effect of motivation on performance, Pdi can be measured during phrenic nerve stimulation (Moxham et al 1981). The phrenic nerve is stimulated transcutaneously at the root of the neck and to ensure that these nerve impulses are reaching their target, a diaphragm EMG can be measured with surface

electrodes near the costal margin (Fig. 4). Magnetometers are used to ensure that the shape of the chest is constant. Pdi can be measured at different phrenic nerve stimulation rates and thus 'low frequency fatigue' can be detected. This technique is obviously complicated, slightly uncomfortable and is currently only used in a few specialised centres.

E. *Diaphragm EMG*

Some workers have suggested that a sophisticated analysis of the diaphragm EMG ('frequency spectrum analysis') can predict patients who are about to develop respiratory muscle fatigue but there is disagreement on the meaning of these EMG alterations and whether they necessarily foreshadow development of fatigue. In low frequency fatigue there is probably a failure of excitation-contraction coupling within the cell which would not be directly reflected in the EMG.

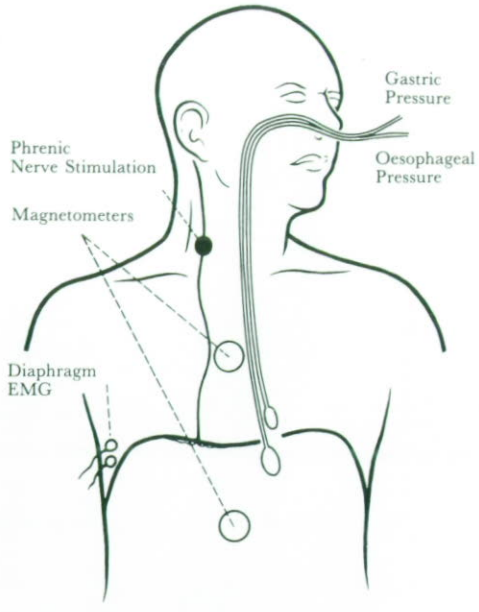

Fig. 4. *Method of recording transdiaphragmatic pressure during stimulation of the right phrenic nerve (see text). Moxham et al (1981). Reproduced by kind permission of the authors and of the Editor of Thorax.*

Conclusion

If respiratory muscle fatigue does occur, the muscles need rest to recover. In some patients this can be provided by relieving bronchospasm or treating pulmonary oedema but in others assisted ventilation must be considered. Whether training of respiratory muscles can increase their strength or endurance is uncertain and results so far are conflicting. Similarly the use of drugs such as methyl-xanthines in reversing fatigue is being investigated. Under what circumstances do the respiratory muscles fatigue? Can one detect fatigue in the early stages? Widespread interest in this topic is relatively recent but the importance of respiratory muscle fatigue in clinical ventilatory failure will only become clear when accurate and easily applied techniques for detecting fatigue become available.

Acknowledgement

I would like to thank Dr. John Moxham for his constructive advice and for his permission to publish Fig. 4.

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ARTP NEWS

ARTP Training Manual

Report on a Workshop held at Derbyshire Royal Infirmary on 4 March 1983.

The Chair was taken by Sue Hill, Chairman of the ARTP Education Sub-Committee and after a welcome by Derek Cramer (Chairman of the ARTP), the following papers were given:

Why We Have a Training Manual
Mrs. S. Gough
Papworth Hospital
Cambridge

The DHSS and the Training Manual
Dr. P. Bourdillon
Senior Medical Officer
DHSS

The Training of Supervisors
Mr. J. Baird
Senior Training Officer
North East Thames Regional Health Authority

Implementation and Assessment
Mrs. S. O'Shea
Training Adviser
DHSS

A Clinician's Viewpoint
Dr. D. C. S. Hutchison
Senior Lecturer
Chest Unit
King's College Hospital Medical School

In the afternoon the participants divided into small discussion groups who were asked to comment on a number of topics. We report briefly the general opinions which were expressed.

1. *The content of the Manual in relation to the in-service training of student technicians.*
   In general, the content of the Manual was thought to be satisfactory though some considered that the 'Basic' unit was too detailed. Further guidelines on the use of the Manual were required and the completion of the training programme should be mandatory. The Manual will need regular review to make sure that it is up to date and always meets the needs of the students.

2. *Will there be any special difficulties which the students might face in completing the in-service training?*
   It was felt that a supernumerary training scheme might be the most advantageous. Some of the equipment required would not be available in all centres, so that travelling facilities should be made available. Students in joint departments might well have difficulty in completing (for example) both the Cardiology and the Respiratory Physiology Training Manuals. A Regional Coordinator will be needed to organise the training of Physiological Measurement Technicians.

3. *The role of the Supervisor.*
   The supervisors are those designated to oversee the training of the students and as such will need to be highly motivated persons who have a detailed knowledge of their own training manuals and are familiar with those of other disciplines. They may need specific instruction in teaching; their training courses should be organised on a regional basis and they should attend regular refresher courses.

4. *How should a student's in-service training be assessed?*
   During the training programme, continuous assessment should take place in the student's own Department and this could be written, oral or practical as required. The general feeling was that the Final Assessment should take place in the student's own Department but the minority felt that it should be conducted at a different Centre in the presence of the student's own Supervisor. The Final Assessment should be primarily practical and oral but should include a written element.

5. *How might the in-service training of students affect the routine work of a Department?*
   The Introduction of the Training Manual will mean extra work for both student and Supervisor; additional staff will be needed to cover the routine work when students or supervisors are attending training courses. It is to be hoped that the student's training will not be hindered by undue preoccupation with routine service requirements.

We are grateful to the following firms who supported the meeting and put on demonstrations.

Simonson & Weel, Coats Pacesetter, Gould Medical, Cardiokinetics, Mercury Electronics (Scotland), Medic-Aid, Garrick Equipment Co. and P. K. Morgan.

We welcome comments at any time on the Training Manual and how it is being put into effect; comments can be sent to Sue Hill, Gillian Lowe or to the Editor of 'Breath' for inclusion in the correspondence column.

We hope that a follow-up workshop can be held next year.

Spring Meeting of the Association

The Spring Meeting took place at the Royal Liverpool Hospital on 16 April 1983. We owe grateful thanks to Mrs. Barbara Peattie and her colleagues for making us all very welcome and for arranging the programme, and to the speakers for their interesting and varied contributions.

We are also very grateful to the following firms who were kind enough to support the meeting:

Deva Medical
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Coats Pacesetter
Medic-Aid (represented by Sorsky's).

The following papers were given:

Histamine challenge tests. Martin Walshaw.
Respiratory effects of endocrine disease.
Acromegaly. Doreen Russell.
Diabetes. John Williams.
Hyperventilation — does it exist? Can it be cured?
Michael Pearson, Barbara Peattie and Sheila Wild.
Squawks. John Earis.
A case of floppy trachea. Linda Savage.

In the afternoon an open discussion was held on:
Integration of staffing structure in the scientific services.
The chair was taken by Derek Cramer.

The meeting ended on a light-hearted note with a dissertation on Liverpool Football Club by Dr. Christopher Evans.

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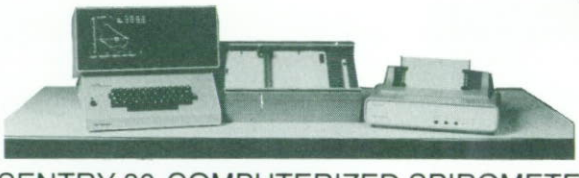

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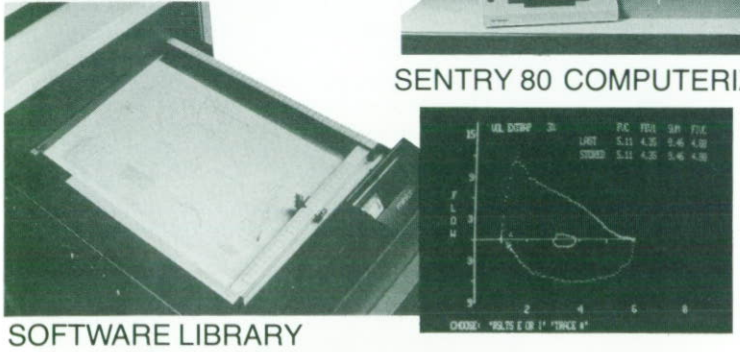

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Annual General Meeting of the ARTP

The Annual General Meeting will be held on 7th and 8th October 1983 at King's College Hospital, London SE5.

The Scientific Programme will start at 7.00 p.m. on the Friday and will be followed by a buffet supper. The Scientific Programme will continue on Saturday followed by the Annual General Meeting.

The details will be announced at a later date.

University of Manchester Department of Anaesthetics

Last year the Physiological Measurement Technicians in the Salford Health Authority held a one day seminar on all aspects of physiological measurement. This was such a success that it is being repeated again this year but on a national basis.

The meeting will be held on Thursday, September 8th, 1983 in the Postgraduate Centre, Hope Hospital, Eccles Old Road, Salford, Manchester. Lunch and registration will cost £5.00 and programme details are available from Margaret Marples of the Department of Anaesthesia of Hope Hospital. Telephone number 061-789 7373. Ext.115.

OBITUARY

GLORIA GESSEY

Mrs. Gloria Gessey was Senior Chief Physiological Measurement Technician in Cardiology and Respiratory Physiology to the Derbyshire Royal Infirmary and Derby City Hospital. She died on 19 January 1983 after a long illness.

Gloria started her technical career in 1948 in the Radiotherapy Department at Derbyshire Royal Infirmary. In 1951 she became a Cardiological Technician at Derby City Hospital and in 1957 took charge of the ECG Department at the Royal Infirmary. With great personal effort and hard work she developed this Department into the large, purpose-built Cardio-thoracic Unit it is today.

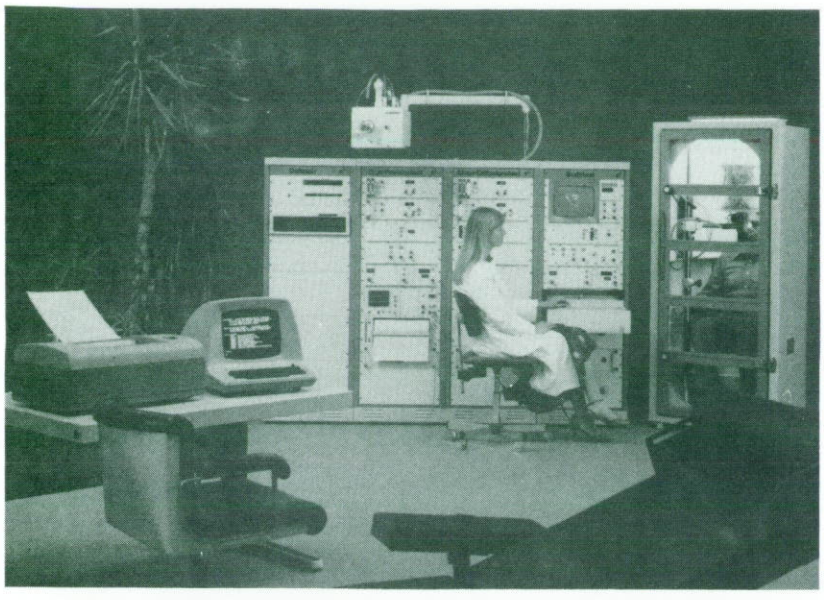
After the appointment of a Respiratory Physician at the Royal Infirmary in 1973, the Department began lung function assessment and now carries the full range of procedures in respiratory physiology; in 1982 respiratory investigations were started at the City Hospital as well.

Gloria was a founder member of the ARTP and served on the Executive Committee from October 1977 to October 1979. She was also a founder member of the Trent Region Physiological Measurement Group and represented the Group on the ONC Steering Committee of the Technician Education Council. She organised the writing of the Trent Region Specialist Training Modules in Cardiology and Respiratory Physiology which were introduced in September 1981 and was currently involved in the assessment of this training programme. Much of this was achieved in spite of her sadly failing health.

Gloria was a far-seeing leader in our field. She was one of the first to recognise the importance of training technicians in cardiology and respiratory physiology and encouraged all her staff to attend meetings and courses and to take higher examinations. Her influence at local, regional and national level will be badly missed. She leaves us with a tremendous example of what can be achieved by sheer hard work and determination.

Gillian Lowe

IMPORTANT ANNOUNCEMENT



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Scientific Services: Integration of Staffing Structure

from the Executive Committee of the ARTP

An integrated pay and grading structure for the various groups of workers in the NHS scientific services is proposed in a discussion paper recently prepared by Regional Scientific Officers. This paper was considered at a Council Meeting of the ARTP in February of this year; in the light of the conclusions of this Meeting and of correspondence from individual ARTP members, the Executive Committee of the ARTP has issued the following statement:

The Executive Committee believes the integration of staffing structure to be in general a desirable objective, but considers that a number of important points need to be made:

1. The discussion paper does not make clear how far integration is to go. The possibilities seem to be:
   (a) total integration of the scientific services
   (b) limited integration between disciplines with overlapping interests, for example:
      (i) medical physics and physiological measurement
      (ii) radiography and medical physics
      (iii) all of the physiological measurement disciplines.

   Integration might be advantageous where joint departments such as Cardiology and Respiratory Physiology already exist and this could be extended to include Clinical Perfusion. But the first step towards integration must be the recognition of equivalent pay and grading structures for all disciplines, together with a common title.

2. It is recognised in the discussion paper that there is a definite need for higher education within the technical disciplines, a principle already accepted within the MLSO grades. Expansion of a developing service depends to a large extent on those employed within it and it is essential that the service is supported, not only by graduates but also by technicians with academic and practical training who can meet the needs of the future. The Executive Committee consider that H TEC is the qualification for which all technicians should aim.

3. There are five grades in the present structure, namely Student, Technician, Senior Technician, Chief Technician and Senior Chief Technician. It is proposed in the discussion paper that the Technician and Senior Technician grades should be amalgamated, thereby removing one grade. The Executive considers, however, that a Technician who undertakes further study should be able to advance to level 2.

4. Grade entry to an integrated structure is possible but it is recognised in a number of disciplines that practical experience is required. Provision should be made for this and with the recognition of higher qualifications for technicians it would be possible to open up a common core structure allowing for open competition within the grades. (In any case, whatever the career structure, personnel have to satisfy an Appointments Committee.)

5. The proposed document may alter the role of the designated head of department who holds the departmental budget; it is unlikely that many of them would rise above level 3 in the new grading structure and as such would no longer be designated head of department.

6. The introduction of a parallel grade may cause friction within a department, although it is recognised that in the future every member of the profession will have obtained an H Tec qualification and thus be graded as a Scientific Officer.

7. Satisfactory completion of a practical in-service training course is essential to the physiological measurement profession and it should be continued in parallel with academic training and extended if possible.

8. The Medical Consultant in charge of a physiological department is an integral part of the department and this should remain the case should integration occur.

Copies of the discussion paper are available from Regional Organisers.

DERBYSHIRE ROYAL INFIRMARY, LONDON ROAD, DERBY DE1 2QY

SENIOR CHIEF PHYSIOLOGICAL MEASUREMENT TECHNICIAN

Applicants are invited from suitably qualified and experienced officers for the post of Senior Chief Physiological Measurement Technician based in the Derbyshire Royal Infirmary but also serving the Derby City Hospital, Derwent, Manor and Derbyshire Children's Hospital.

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Further detail, job description and application form are available from the Personnel Division, Derbyshire Royal Infirmary, London Road, Derby. Telephone Derby (0332) 47141 Ext. 425.

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EXECUTIVE COMMITTEE OFFICERS for 1982-83

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The Brompton Hospital,
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