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BREATH

CONTENTS

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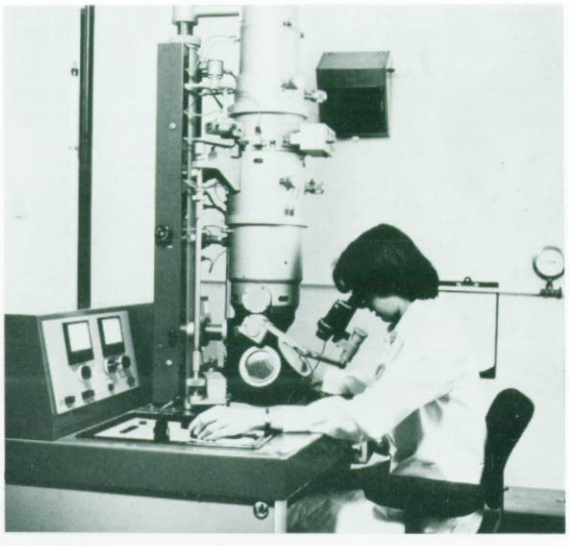

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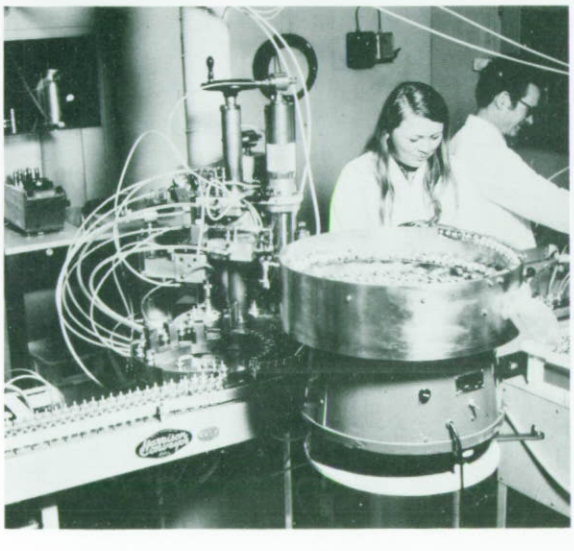
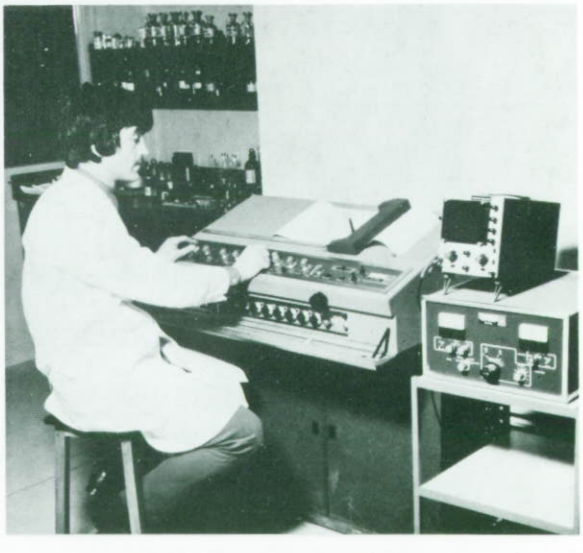




Significant advances in therapy from Original British Research

EDITORIAL

Tuberculosis — a Centenary

One hundred years have passed since the identification of the tubercle bacillus by the German scientist Robert Koch, but tuberculosis still remains a world-wide and formidable problem.

This is an ancient disease; signs of it have been found in Stone Age skeletons, in Egyptian mummies 5000 years old and it is described in the writings of Hippocrates. In this country the history of the disease dates back many centuries when it took the form known as 'scrofula' which refers to the tuberculous lymph node enlargement in the neck, often associated with bone disease. In the 5th century the Kings of France were reported to be endowed with the power to heal scrofula by 'touching' — a favour not granted by English kings until the 11th century: thousands flocked to be cured and the practice continued until the end of the 18th century. According to Boswell, Samuel Johnson was 'touched' by Queen Anne in 1712, but records of any controlled trials of this therapy do not appear to have survived.

During the 18th century the disease increased in its incidence along with the urbanisation of the Industrial Revolution and at its peak the incidence rose to 700 deaths per annum for every 100,000 in the population. We have a unique continuous statistical record in London from the year 1631 to the present day. These records started as the 'Bills of Mortality' which continued up to the beginning of Death Registration in 1838 when the records were formally taken over by the Registrar. There were obviously diagnostic shortcomings as bacteriology was still a science of the future, but the clinical features in the majority of cases must have been plain enough, because by this era the disease was recognised mainly in its pulmonary form, commonly known as 'consumption'. The poet Keats died of it at the age of 26, no doubt helped on his way by the blood-letting and semi-starvation which was the customary treatment at the time. Since the year 1800, the incidence of the disease has declined steadily, presumably due to the gradual improvement in living conditions, with reduction in over-crowding and better nutrition.

And so to Robert Koch's discovery: Pasteur only twenty years earlier by his research in the wine-making industry had completely disposed of the theory of spontaneous generation of micro-organisms which was widely held at that time. Tuberculosis was known to be an 'infective' process in fact, although no organism had been isolated. Koch's essential step was to move from the liquid media for growing organisms to a new technique; he developed the use of solid media, based on agar gel, so that the bacteria would remain stationary and form colonies of a single strain. For his discovery of this organism, which was named *mycobacterium tuberculosis*, Koch received the Nobel prize for Medicine in 1905.

Koch went on to extract a protein known as 'tuberculin' from the culture of the organism and announced prematurely that the extract was of therapeutic value. This news was of course sensational but unfortunately administration to humans with the disease soon showed that there was no substance in the claims. Nevertheless tuberculin injected intradermally (as in the Mantoux test) was subsequently found to be of great value as a method of distinguishing persons who have been infected with tuberculosis.

In the present century we have seen further great advances in the containment of this disease. Vaccination was introduced in France in 1922, using a weakened strain of the organism known as Bacille Calmette Guerin (or BCG) after the two French scientists who developed it. This proved to be highly effective in preventing the disease and was widely used in Europe after the 1939-45 War when TB showed a sudden increase; it is still the policy to administer BCG to school children and to groups at special risk, such as workers in the health industry.

But curative treatment for tuberculosis did not really arrive until 1943. The discovery of penicillin had alerted microbiologists to the possibility of producing other antibacterial substances from moulds and Selman Waksman in the USA then isolated a substance from a mould of the Streptomyes family which was active against many organisms where penicillin was ineffective. The new antibiotic was named *Streptomycin* and this was the first to be effective against the tubercle bacillus. Waksman, like Koch before him, received the Nobel prize for this work.

Other good drugs such as isoniazid and para-aminosalicylic acid were developed shortly afterwards and it soon became clear that the drugs had to be given in combination because of the remarkable propensity of the organisms to become resistant to any drug given alone. Much time and energy has gone into the design of clinical trials to determine the correct combination and duration of therapy and to-day with powerful new drugs like rifampicin, a patient newly diagnosed in this country can be reasonably certain of being cured in 6 to 9 months. The correct administration of these drugs requires a good deal of experience if resistance is to be avoided and in some parts of the world serious problems have arisen through the appearance of drug resistant strains of the bacillus which can then be passed on to others.

One hundred years after the discovery of the organism, the World Health Organization calculates that there are still 10 to 20 million cases of TB in the world, with a mortality rate of 1 to 2 million per annum, the majority in developing countries. The eradication of the disease will require the expenditure of enormous resources, but perhaps in another 100 years, this ancient scourge will have gone the way of smallpox. Well, perhaps!

PHYSIOLOGICAL ADAPTATIONS TO EXERCISE

J. W. Reed
University of Newcastle-upon-Tyne

Exercise is the most common and important physiological stress that man must endure. An exercising muscle may exhibit an enormous increase in metabolic activity (up to 50 times the resting level), which presents major problems in terms of fuel supply, elimination of waste products and maintenance of cell and body homeostasis. The study of exercise physiology provides basic information about the nature and range of the functional capacity of virtually every organ system in the body, and the complexity of the interactions and adaptations involved provide a considerable intellectual challenge. Apart from academic considerations, there is an important practical side; the capacity of any organ or system can be properly determined only when it is subjected to a representative functional load. This is particularly true in the case of the respiratory and cardiovascular systems. At rest, ventilation is about 8 litres/minute but can exceed 100 litres/minute during exercise; similarly resting cardiac output is about 6 litres/min whereas during exercise it can exceed 20 litres/min so there is considerable reserve capacity and a significant proportion of this capacity must be encroached upon before normal daily activity becomes limited. Tests of cardiopulmonary function at rest may reveal a great deal, but may not on the other hand be sensitive enough to detect any abnormality in the patient who complains of exertional dyspnoea but who is symptom-free at rest. By stressing the system and demanding a high output, it may be possible to detect early disease by assessing the response elicited by the extra demand. It may also be possible to quantify any reduction in working capacity brought about by that disease.

Assessment of disability implies a knowledge of the 'normal' response, and of the factors which limit that response.

A. THE NORMAL RESPONSE TO EXERCISE

Exercise means increased work for the muscles involved, which in turn means an increased energy requirement. Muscle obtains energy for mechanical work from an 'energy-rich' compound called adenosine triphosphate (ATP), which is stored in the muscle itself. Unfortunately the amount of ATP in muscle is limited and very quickly used up; for the exercise to continue the stores of available ATP must be continually replenished. The synthesis of ATP depends upon a second high-energy compound, creatine phosphate (CP), which is itself in short supply. Regeneration of CP depends upon energy derived from the breakdown of foodstuffs, mainly carbohydrate and fat. These reactions may be summarised as:

(1) Creatine (C) + Phosphate (P) + Energy $\rightleftharpoons$ CP

(2) CP + ADP (adenosine diphosphate) $\rightleftharpoons$ ATP + C

(3) ATP $\rightleftharpoons$ ADP + P + Energy

The major steps in the breakdown of foodstuffs are given in Fig 1. The initial stages take place in the cytoplasm of the cell and culminate in the production of pyruvate. The

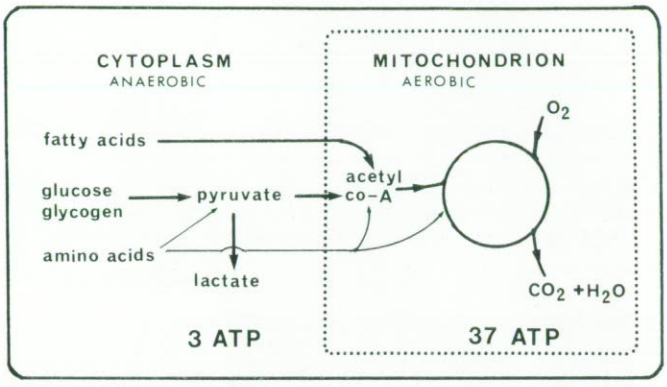


Fig 1. *The main metabolic pathways. Amino acids (from proteins) can enter pathways at different sites depending upon structure, but are not an important source of ATP.*

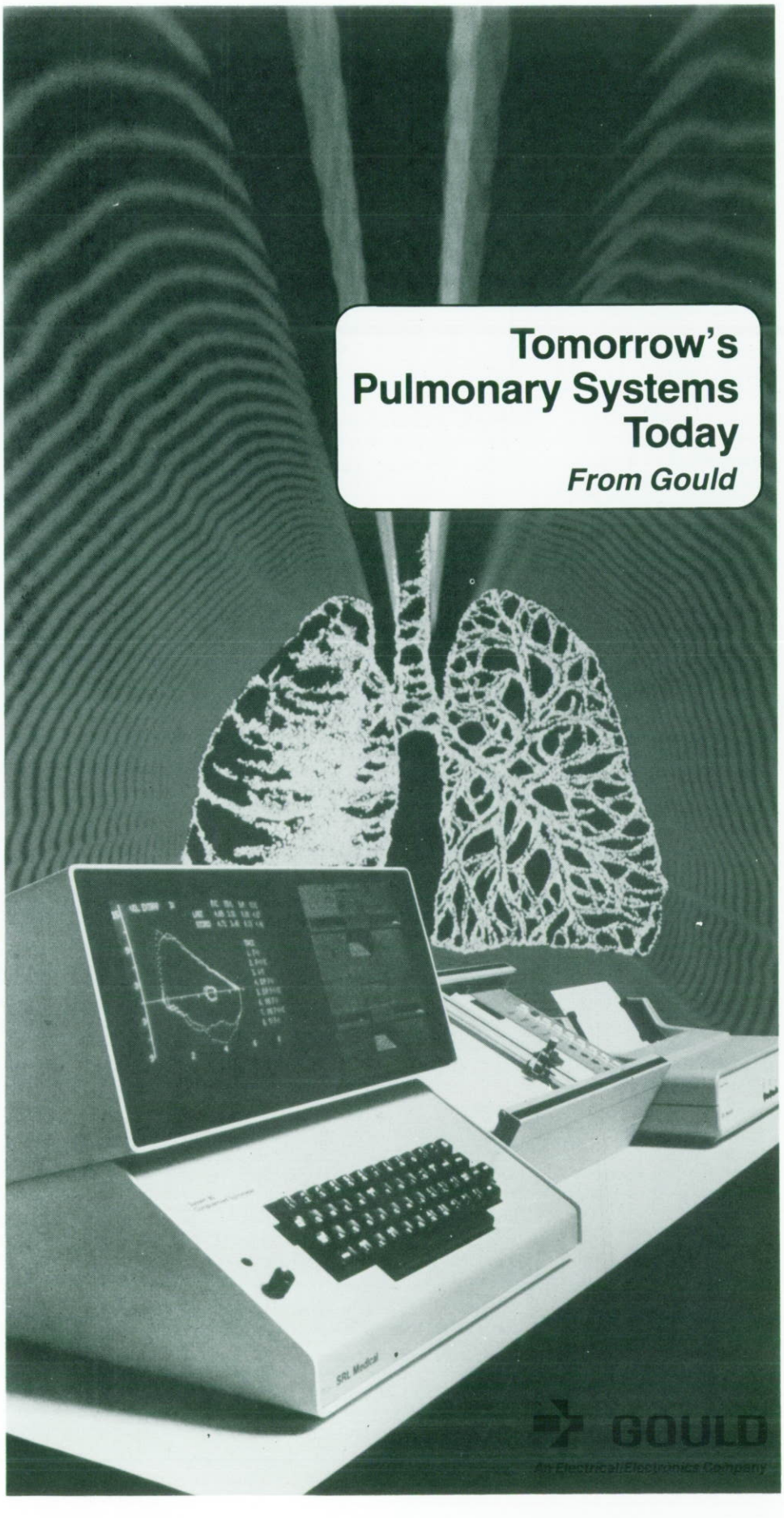
fate of the pyruvate then depends upon the supply of oxygen; if oxygen is available, then pyruvate can enter the mitochondria of the cell to form acetyl Co-enzyme A (Acetyl Co-A). Acetyl Co-A then is oxidised to produce a net yield, for carbohydrate, of 37 molecules of ATP. However, if oxygen is unavailable, or the supply is severely limited, then the pyruvate will be converted to lactate, with a net yield of only 3 ATP molecules. It is thus possible for energy to be gained for muscle contraction by anaerobic metabolism but the amount so produced is relatively small and the end product, lactate, is a toxic compound that will eventually 'poison' the cell. The large majority of the energy for muscle contraction comes therefore, from ATP derived from aerobic metabolism and the ability to perform work of more than a very short duration depends upon oxygen delivery.

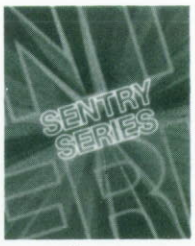
The Oxygen Transport System

The dynamics of muscle oxygen consumption during work are shown graphically in Fig 2a. During fixed-load exercise, oxygen uptake measured at the mouth increases from the resting value until a plateau is reached. This indicates that 'steady-state' conditions have been attained and that the oxygen uptake is sufficient for the external work load. If the external work load is increased, then the oxygen uptake will increase to an appropriate level.

If now the steady-state oxygen uptake is plotted against external load (Fig 2b), it can be seen that there is a linear relationship over most of the range — in other words, oxygen uptake is directly related to work done. Only at the very top of the work range does the oxygen uptake fail to increase with increasing load; this level is the true maximal oxygen uptake and is widely regarded as being the most acceptable measure of maximal working capacity.

Because oxygen uptake is linearly related to external work, and because it is a better measure of *total* work (which includes work done moving the legs, maintaining posture, etc.), oxygen uptake is usually regarded as the preferred measure of work done.





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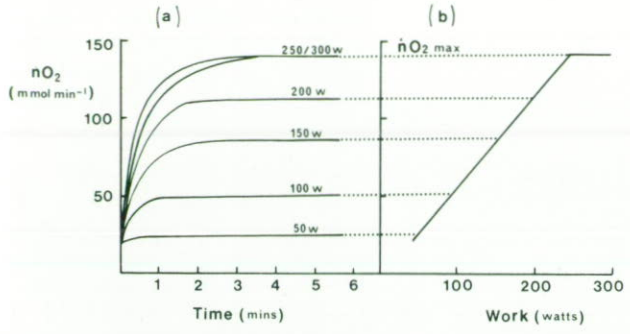

Fig 2. *(After Astrand, 1977)*

(a) to show the attainment of steady-state values of oxygen uptake (nO_2) at different exercise levels. Note that the greater the workload, the longer the time necessary to reach a steady-state.

(b) Oxygen uptake expressed directly in relation to external work. The small amount of work that can be performed after the nO_2 plateau has been reached is funded by anaerobic metabolism.

Oxygen uptake and delivery have historically been accepted as being dependant on lung ventilation (to bring oxygen to the gas exchanging surface) and cardiac output (to deliver the oxygenated blood to the exercising muscles).

Ventilation

The demand for increased oxygen uptake is met initially by the ventilatory system. One might therefore expect ventilation to be related to work load, and indeed it is. (See Fig 3a). Note however, that at higher work loads, ventilation does not reach a steady value but continues to rise throughout the exercise period. If the ventilatory response to the exercise is plotted in a slightly different way (Fig 3b), it is apparent that ventilation increases linearly at first and can therefore be regarded as linked to

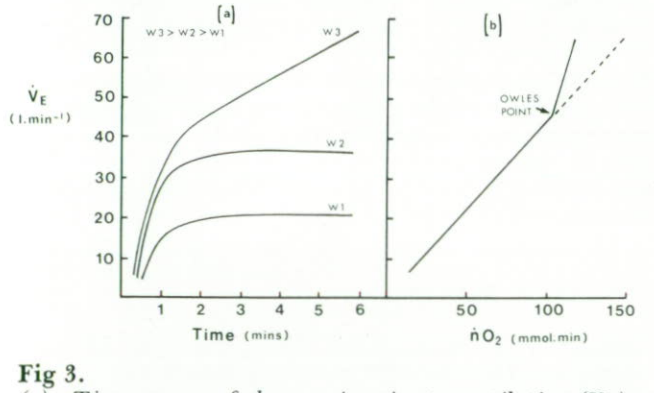

Fig 3.

(a) Time course of changes in minute ventilation (V_E) at different levels of steady-state work (W). At higher workloads (W_3) a steady-state ventilation cannot be achieved because of increased stimulation arising from aerobic metabolism.

(b) The same data re-plotted to show the relationship between V_E and oxygen uptake (nO_2). The relationship is essentially the same during progressive exercise.

metabolic demand. At higher work levels ventilation increases disproportionately; the point at which this relative hyperventilation occurs has been somewhat inappropriately called the 'anaerobic threshold' — it would be more accurate to use the original term 'Owles' Point', after Owles who first demonstrated the link between work rate and the lactate production which is thought to be the causative factor (see A, below: *The Anaerobic Threshold*).

The increase in ventilation is brought about by changes in the breathing frequency and tidal volume. The initial rise is almost entirely due to an increase in tidal volume, with little or no change in respiratory frequency. The tidal volume increases progressively to a value which is normally about half the vital capacity; thereafter, the respiratory frequency is increased and is primarily responsible for the increased ventilation. An understanding of the control of the pattern of breathing during exercise requires a detailed analysis of the relationships between tidal volume and duration of inspiration and expiration, but sufficient information for most purposes can be obtained from a graph of ventilation against tidal volume, the so-called 'Hey-plot' (Fig 4). A knowledge of the pattern of breathing is important clinically, because the balance between frequency and tidal volume appears to be affected by the work of breathing, which is altered in various diseases. The tidal volume is also important in that it affects gas mixing and therefore the efficiency of ventilation in gas exchange.

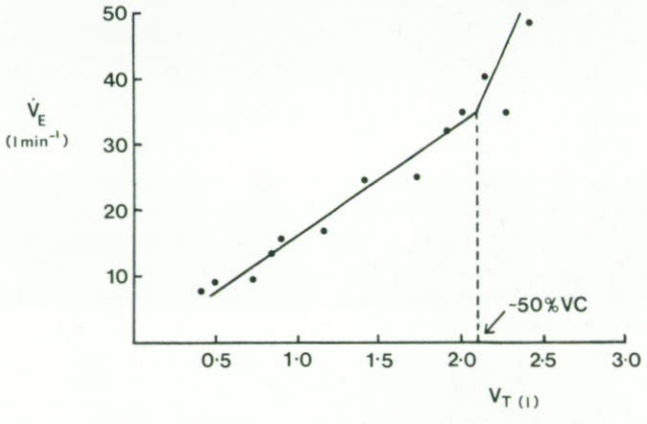

Fig 4. *Relationship of minute ventilation (V_E) to tidal volume (V_T) during progressive exercise — Hey-plot. At a tidal volume equal to approximately 60% of vital capacity, respiratory frequency becomes the major determinant of increases in ventilation.*

Cardiac Output

Cardiac output is also directly related to work and the relationship is linear over the whole range (Fig 5). As with ventilation, cardiac output is the product of volume (stroke volume) and frequency (cardiac frequency). Stroke volume very quickly reaches a maximal value during exercise whereas cardiac frequency increases progressively over nearly the whole range. Because stroke volume does not change materially after the early increase, changes in cardiac output are faithfully mirrored by changes in cardiac frequency, so this very important facet of cardiovascular function may be easily monitored (but see B.3).

The absolute level of cardiac output is not the only important factor; it is the muscle blood flow which is critical for oxygen delivery. To augment the increase in local muscle blood flow produced as a result of increased cardiac output, blood is diverted away from non-active regions such as the gut. The actual uptake and utilization of the oxygen by the muscle is then governed by muscle fibre type, the number of mitochondria and the quantity of oxidative enzymes present in the muscle cell.

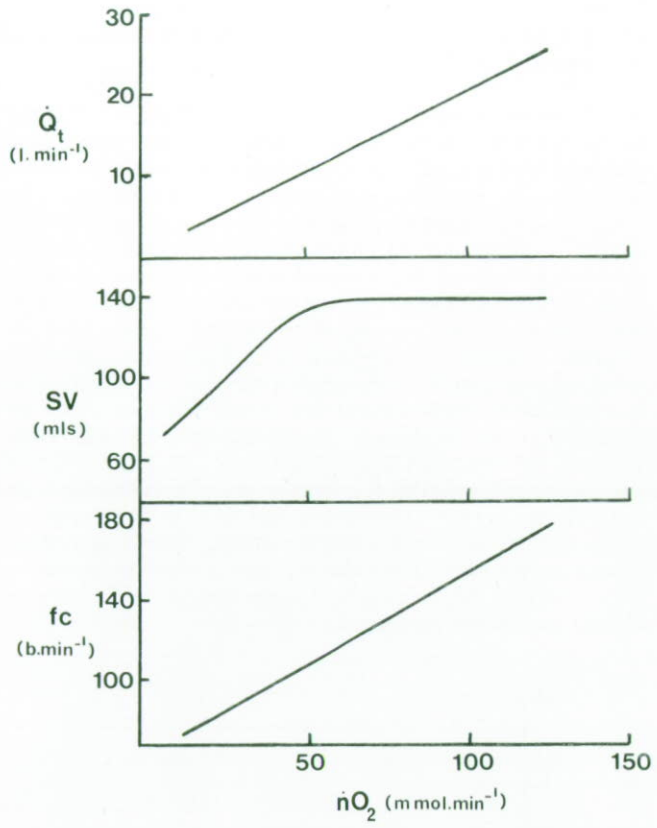

Fig 5. *Changes in cardiac output (Qt), stroke volume (SV) and cardiac frequency (fc) during exercise.*

Both Qt and fc increase linearly to maximal values but stroke volume reaches a plateau at about 40-50% of nO_2 max. — this is equivalent to a heart rate of about 120 breaths per min.

The Anaerobic Threshold

At a certain intensity of work, lactate is produced faster than it can be metabolised and blood lactate levels start to rise. This point has been called the 'anaerobic threshold', although in fact there is good evidence that oxygen is still available to the muscle. An acceptable explanation for the increase in lactate production has yet to appear. The *effects* of lactate however, are obvious. It provides an additional stimulus to breathe, so ventilation increases relative to work performed; as a result, end-tidal oxygen increases and end-tidal CO_2 decreases. Lactate is buffered by bicarbonate to produce CO_2 and as a consequence carbon dioxide output increases relative to oxygen uptake, and the respiratory exchange ratio increases. Interest in the 'anaerobic threshold' has increased in recent years since it is apparently related to efficiency of the oxygen transport system and can presumably be utilized as an index of physical fitness.

B. FACTORS AFFECTING THE NORMAL RESPONSE

These factors are numerous, complex and interrelated. The most commonly allowed for are:

1. Age — Maximal oxygen uptake increases during childhood and adolescence as the components of the O_2 transport chain grow, but declines steadily thereafter. This is thought to be due to the observed reduction in maximal heart rate which occurs with the ageing process.

2. Sex — The variability, in exercise response associated with sex difference can be largely attributed to differences in body size, but there is some residual variation.

3. Size — The bigger the person, the more muscle and hence the greater ability to perform work. In addition, a large heart will have a proportionately bigger stroke volume and hence a greater potential to increase cardiac output. Fortunately, heart size is closely correlated with the amount of body muscle, so with appropriate measurements, the influence of heart size on cardiac performance can be allowed for.

4. Habitual Activity — This is effectively training, which improves oxygen transport. The effects are mainly on the cardiovascular system, (increasing stroke volume: increased peripheral flow) and the muscle (increased number of mitochondria and more oxidative enzymes). The net result is delayed anaerobiasis, Owles' point occurs later, and maximal oxygen uptake is increased.

5. Motivation and psychological factors — a somewhat grey area which is now being studied using the subject's own estimation of the severity of "breathlessness".

The clinical importance of exercise testing is now widely accepted, and will be discussed in a following article. As expected in such a complex topic the literature is extensive; the references cited below will give a good introduction and can be used as source material for more detailed reading.

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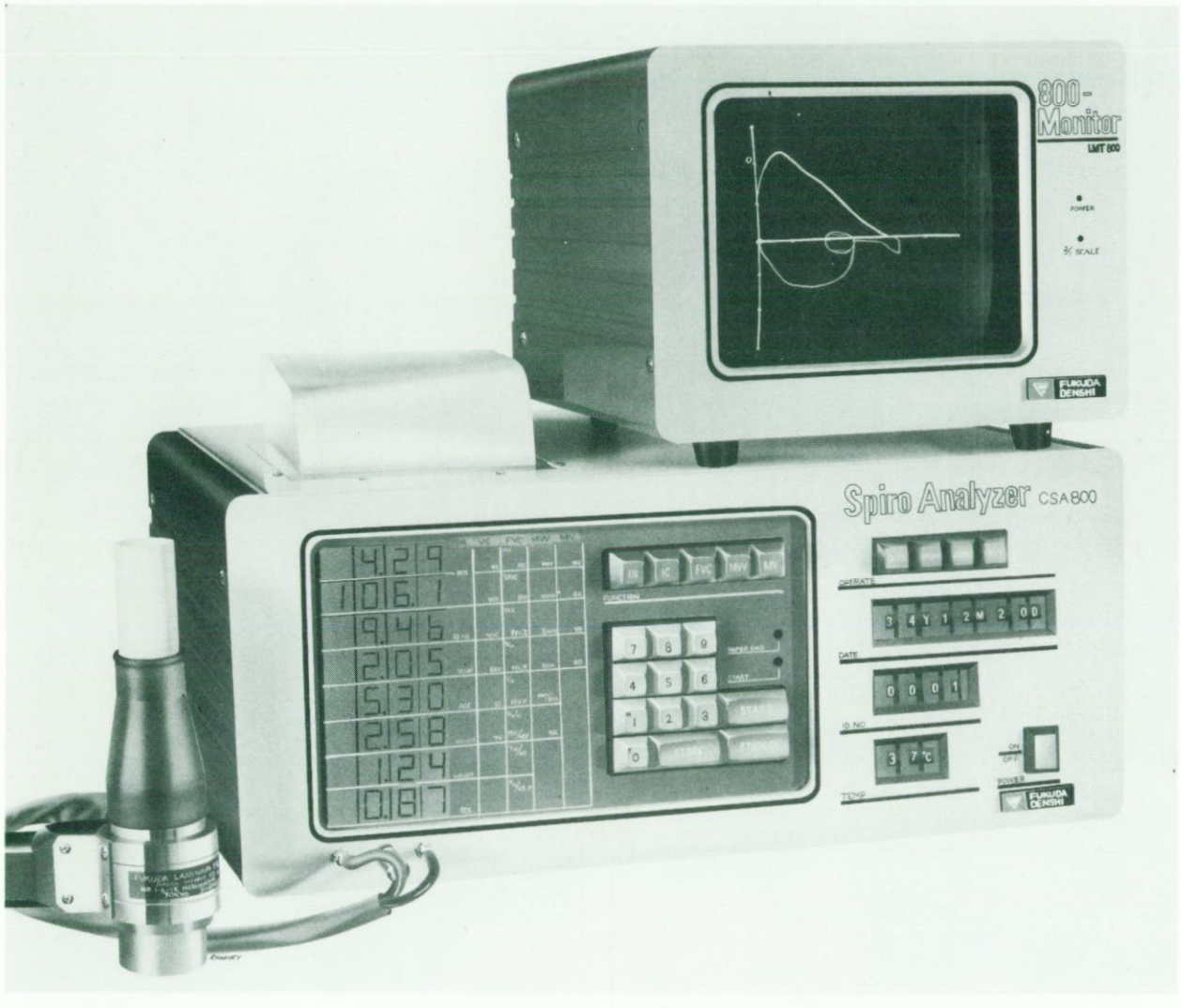

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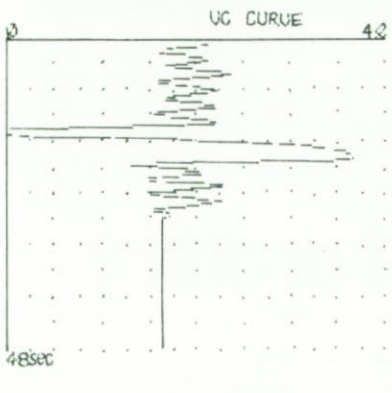

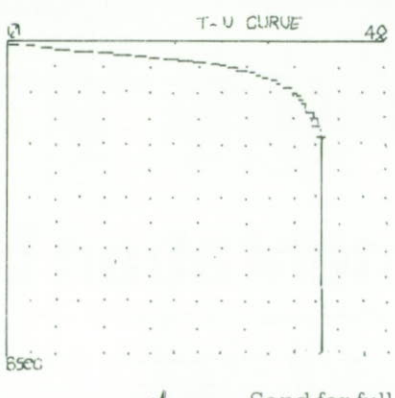

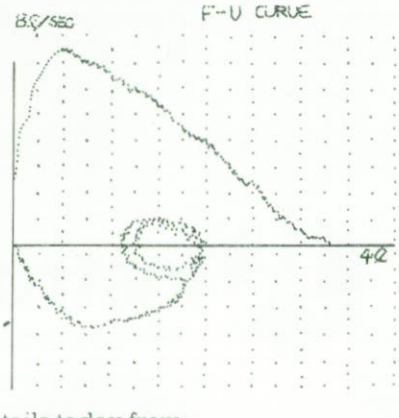

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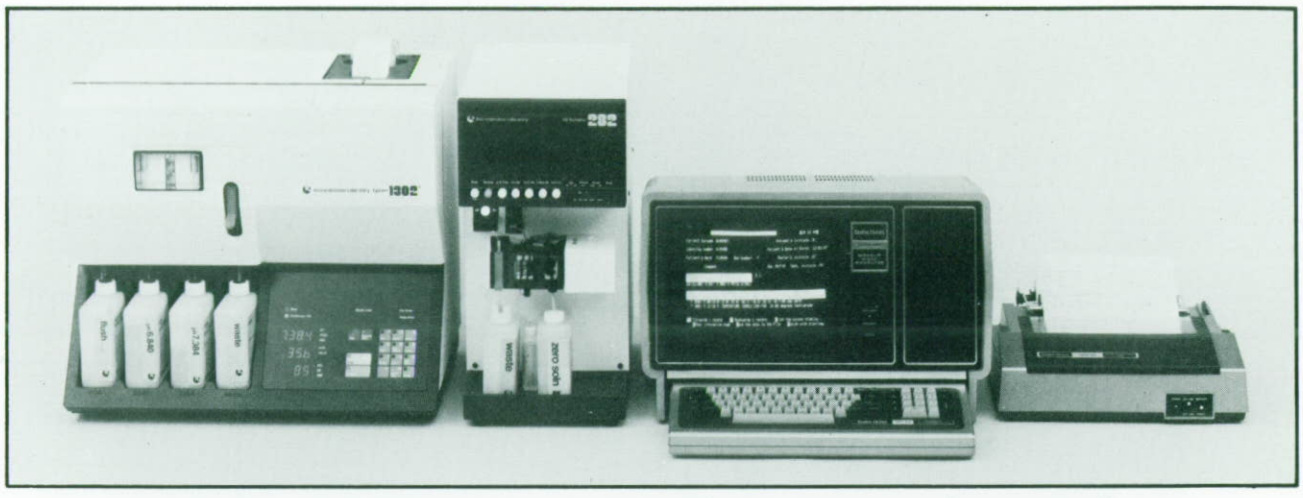

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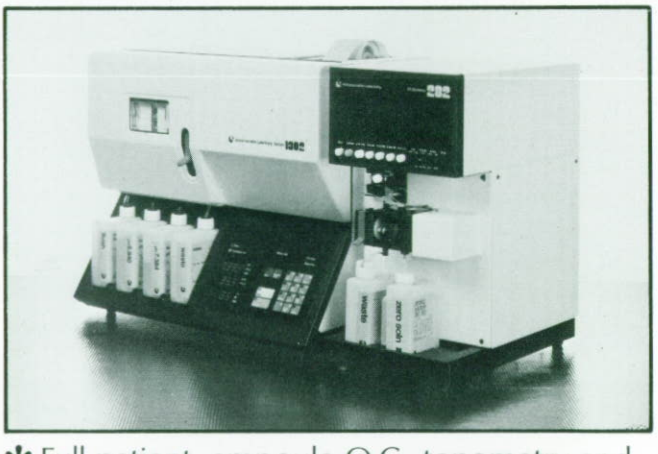

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OESOPHAGEAL MANOMETRY AND pH TESTING - A METHOD OF ASSESSING OESOPHAGEAL FUNCTION

Margaret Marples
Department of Surgery, Hope Hospital, Salford

INTRODUCTION

The most common method of assessing oesophageal function is by barium meal and oesophagoscopy. Over the last few years, more departments have been using oesophageal manometry and pH testing in addition to conventional methods. These tests are of value to the gastroenterologist, to the surgeon for pre- and post-operative assessment and to the chest consultant, who should be aware of the relationship between gastro-oesophageal reflux and asthma[1] and may need to determine whether chest pain is oesophageal or cardiac in origin.[2]

The technique described in this paper is used in the Surgical Investigation Unit, at Hope Hospital, for both clinical and research purposes.

EQUIPMENT

Oesophageal pressures are recorded using an eight lumen, micro-miniature multi-lumen catheter (Arndorfer Medical Specialties), with single side openings at 10 cm and 7.5 cm and three side openings placed at 120°, 5 cm from the tip of the catheter. The catheter is water perfused by a low compliance pneumo-hydraulic capillary infusion system (Arndorfer Medical Specialties). Strain gauges (Statham P23 transducers), are coupled between the infusion system and the catheter. Pressure recordings are displayed on an eight channel chart recorder (Grass Polygraph 7D).

Respiration is monitored with a nasal thermistor (Grass Instruments) coupled to the chart recorder (Fig. 1).

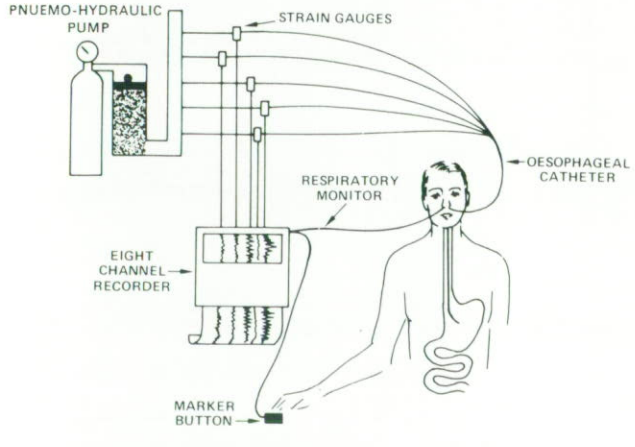

Figure 1. System for oesophageal manometry.

pH measurements are made using a stomach pH electrode (Radiometer GK282/C), joined to a reference electrode (Radiometer K4018) by a salt bridge and connected to a pH meter (Radiometer PHM62). The pH meter is linked to the chart recorder.

A single manometry line, with a side opening 4 cm from the tip of the probe, is fused to the pH electrode thus enabling accurate positioning in the oesophagus. The line is water perfused by an infusion pump (Braun).

TECHNIQUE

The patient is admitted to the Surgical Investigation Unit in the mid-morning, having had an early light breakfast. Drugs such as cimetidine are not taken on the day of admission. On admission the patient is given nothing by mouth and asked to refrain from smoking. The test procedure is fully explained to the patient and informed consent obtained.

With the patient sitting on the edge of the bed, the manometry catheter is passed nasally, without anaesthetic (K-Y jelly is used to lubricate the tube) into the stomach. Sips of water are given to aid swallowing the tube. The patient is then placed in the supine position, given a marker button to press on swallowing and the nasal thermistor is positioned. The infusion system is switched on and while the bubbles clear from the line, the patient relaxes and tries to control breathing and swallowing.

When the system is clear and the patient relaxed, the catheter is withdrawn from the stomach using the 'station pull-through technique'[3], one cm at a time, holding each position for 10-20 seconds. The trace is monitored on the pre-calibrated chart recorder, paper speed 100 mm/min. Each new position is marked on the paper.

Gastric pressure, lower oesophageal sphincter pressure, relaxation of the sphincter to gastric pressure levels on the arrival of a swallow wave, oesophageal motility, peristalsis and crico-pharyngeal sphincter pressures are measured. The catheter is then withdrawn.

The pH electrode is passed into the stomach, using the same technique as for the manometry, and withdrawn using the station pull-through, until the tip of the probe is 5 cm above the lower oesophageal sphincter. The line is taped to the patient's nose and cheek.

Stress tests are then performed by the patient, to try to provoke acid reflux. The tests consist of three Valsalva manoeuvres, three sit ups, three leg raises to 45° and three stomach pushes. A drop of two pH units on any three manoeuvres indicates a positive test[4].

Finally, an acid perfusion test (Bernstein test)[5], is performed. Normal saline and 0.1N Hydrochloric acid are infused alternately via the manometry line. The acid should provoke the patient's usual symptoms.

The system is then prepared for overnight monitoring. The paper speed is slowed to 5 mm/min. and the water infusion is slowed to 1 ml/hr. The patient is allowed to eat and drink normally, with the exception of drinks with a pH value below 5. The probe is removed at 0800 hours.

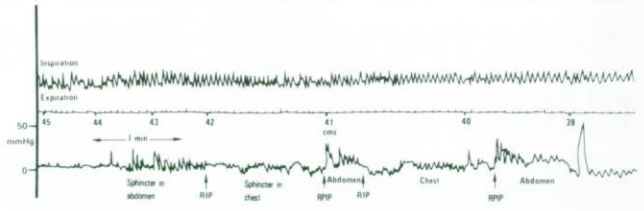

Figure 2b. Reduced lower oesophageal sphincter pressure (RPIP) with second respiratory inversion points (RIP).

INTERPRETATION OF RESULTS

Like most physiological tests, interpretation of the tracings comes with time and experience. The basic figures calculated from the trace are as follows:

1. Lower oesophageal sphincter pressure is calculated as mm Hg above gastric pressure, measured at end-expiration, in an attempt to eliminate respiratory effects[6] (Figs. 2a and 2b).

2. Sphincter length is measured in cm.

3. The number of respiratory inversion points (the point where the catheter passes from the abdomen into the chest, thus changing from positive to negative pressure) are noted. More than one point indicates a hiatus hernia, because the sphincter slides backwards and forwards through the hiatus (Figs. 2a and 2b).

4. Peristalsis and motility in the body of the oesophagus are studied for abnormalities (Figs. 3a and 3b).

5. The crico-pharanyx is graded as coordinate or incoordinate, depending on whether pharyngeal contraction coincides correctly with sphincter relaxation[7] (Figs. 4a and 4b).

6. On the overnight trace the number of episodes during which pH falls below 4 for 30 seconds or longer are counted and also expressed as percentage of the total monitoring time[8]. The number of episodes longer than 5 minutes and the longest episode is also reported (Fig. 5).

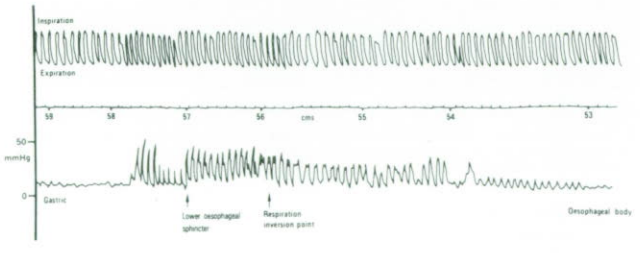

Figure 2a. Normal lower oesophageal sphincter pressure and respiratory inversion point.

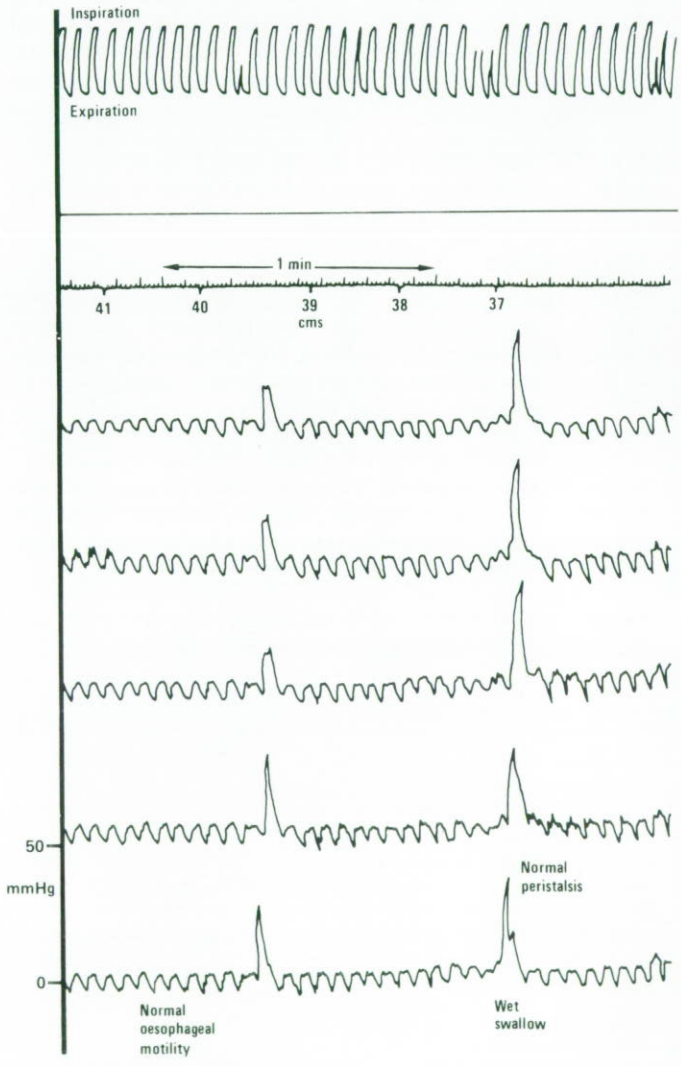

Figure 3a. Normal oesophageal motility and peristalsis.

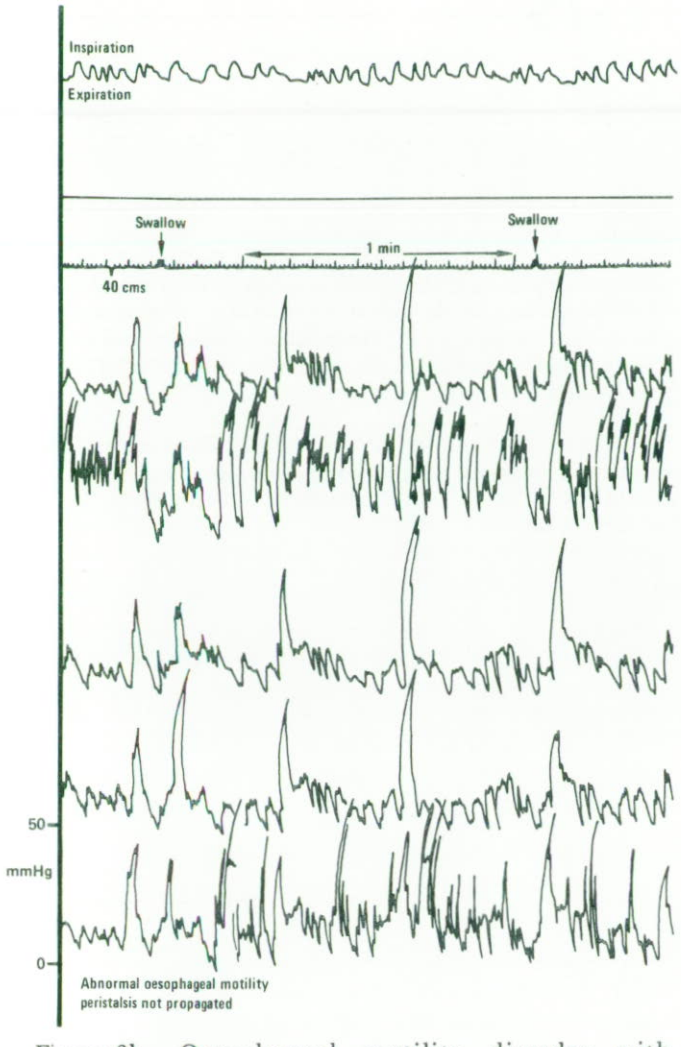

Figure 3b. Oesophageal motility disorder with abnormal peristalsis.

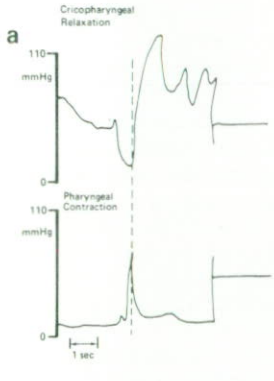

Figure 4a.

Coordinate cricopharynx.

Figure 4b.

Incoordinate cricopharynx.

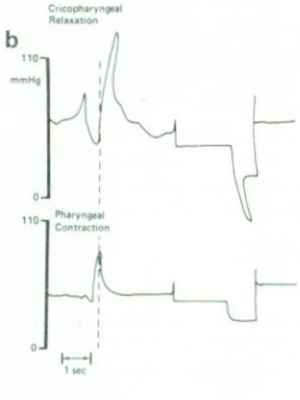

Figure 5. Section of overnight pH trace, showing gastro-oesophageal reflux.

CONCLUSION

1. We have found this method of oesophageal assessment suitable for all types of patient, whether for clinical or research studies.

2. It has been used for a wide range of age groups; our youngest patient was 18 months (he underwent pH testing only), and the oldest 90 years.

3. Though the initial passing of the tubes can be quite traumatic and distressing for the patient, with patience and understanding from the staff, most patients can be persuaded to undergo the tests and indeed, many return for further testing.

ACKNOWLEDGEMENTS

This paper is written with grateful thanks to the following, without whom the work could not be done: Snr. SEN. A. Ross and SEN. S. Froggatt, who patiently pass the catheters and have the tiring job of withdrawing the tubes whilst keeping the patients relaxed: Mrs G. Cloherty, student physiological measurement technician, for assisting with the measurements: Mr J. Bancewicz, Senior Lecturer and consultant surgeon, for his help and comments on this paper.

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SERIAL LUNG FUNCTION TESTS

P. Lockwood
Harefield Hospital, Middlesex

Any chest disease can be studied from two major aspects: firstly, we can consider the cellular or structural changes which are the fundamental basis of the condition and which we can detect by such means as radiography, bronchoscopy and microscopy. Secondly, we can assess the functional effects of these diseases by quantitative tests of respiratory function which can be used to demonstrate changes occurring with the passage of time or as a result of treatment. Indeed some clinicians hold that this is the most valuable way to use these tests.

Short-Term Response Tests

Most workers in our field know the value of simple peak flow or spirometric tests in measuring the effect of treatment on bronchospasm. There have been two excellent talks at recent meetings of the Association on this topic, by Dr. J. E. Stark at Papworth Hospital and Dr. G. Jones at Walsgrave Hospital, Coventry. Repeated testing on a single occasion, as in exercise response or bronchial challenge testing[1] is excluded from discussion here.

It is important to record the test results in a graphical form so that the overall changes can be seen, and to test at least three times during a 24-hour period to display diurnal variation. The exact time at which the test should be carried out varies from patient to patient but it is particularly important to discover if a nocturnal or early morning 'dip' in the peak expiratory flow rate is occurring in asthmatics (and some bronchitics). The patient will often awaken with acute dyspnoea or cough and should be encouraged to record the peak flow rate at this time, preferably both before and after a bronchodilator inhalation.

Whatever test is used, it is important that it should be repeated a number of times so as to give a more accurate assessment (whether the maximum or the average of a given set of recordings is finally obtained). The PEFR, FEV_1 or maximal mid-expiratory flow rate are more significant in this context than the forced vital capacity (FVC) or FEV_1/FVC ratio. The test should be carried out at the same time interval after administering the bronchodilator on each occasion, 5-10 minutes after a short-acting agent such as rimiterol, or 10-30 minutes after salbutamol, terbutaline, or similar longer-acting substances. Two inhalations from the aerosol dispensers should be given, or 1 ml diluted with 2 ml sterile water from a nebuliser (gas flow: 2 litres/min) in the case of salbutamol Respirator Solution. It is essential to see that a patient using an aerosol inhaler is using it properly; in our experience the terbutaline 'Spacer-Inhaler' is much easier to use and avoids too rapid an inhalation which causes the droplets to impinge upon the posterior wall of the pharynx instead of being inhaled into the bronchial tree.

Misleading results can occur when forced expiration produces tracheo-bronchial collapse[2]. At one time the presence of this phenomenon, with its distinctive appearance on the spirogram (Figure 1), was considered indicative of emphysematous changes affecting the major airways. It is now recognised that there is a variation in the normal structure of the upper respiratory tract which can give rise to collapse when the transpulmonary pressure rises. Because this abrupt collapse occurs within the time of the recording of the peak flow rate, the gas thereby expressed contributes to the result, which is thus falsely high. From personal experience the possible error is of the order of 50-60 litres/min. Although it is important to be aware of this factor, it may actually have little significance in serial testing, except to explain discrepancies between the findings from different tests (eg, PEFR and FEV_1).

Full respiratory function tests should be done before the short-term response tests and repeated after the course of inhalation or nebulisation treatment is completed.

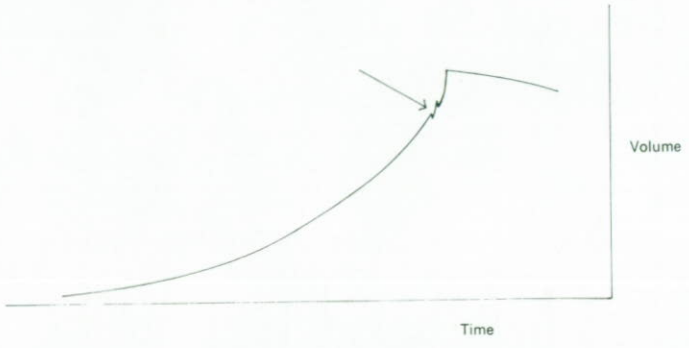

Fig 1. *Forced vital capacity tracing showing the effect of tracheobronchial collapse (arrowed).*

Lung Function Tests in Bronchospasm

It is worth carrying out a number of independent tests of air flow and the forced expiratory and inspiratory vital capacity should be recorded both spirometrically and as the flow volume trace. This gives a set of values against which the PEFR or other repeated tests can be assessed and the long-term changes noted. In conjunction with the forced breathing tests the lung volume measurement shows the influence of airway obstruction on the VC and the residual volume (RV). As airway obstruction is relieved not only do the tests of expiratory airflow show improvement but the VC increases and the degree of hyper-inflation, as revealed by the RV, decreases.

In spite of modern fashion, it is useful to record the direct maximum voluntary ventilation (MVV) over a 15-20 second period. Although this test is affected by factors other than the state of the airways it is a valuable assessment of the total possible ventilation. It has wide clinical application, displaying how the patient's motivation may improve along with a general sense of well-being. Contrary to often-expressed opinion, it is not as difficult as the FVC for the dyspnoeic patient to perform and in 21 years of using the test the present writer has never noted any detrimental effects on a patient. It is salutary to see how often the indirect value calculated from the FEV_1 can differ from the direct MVV. Critics of the test on the basis of its non-specificity should ponder the fact that few discount the value of the transfer factor on similar grounds.

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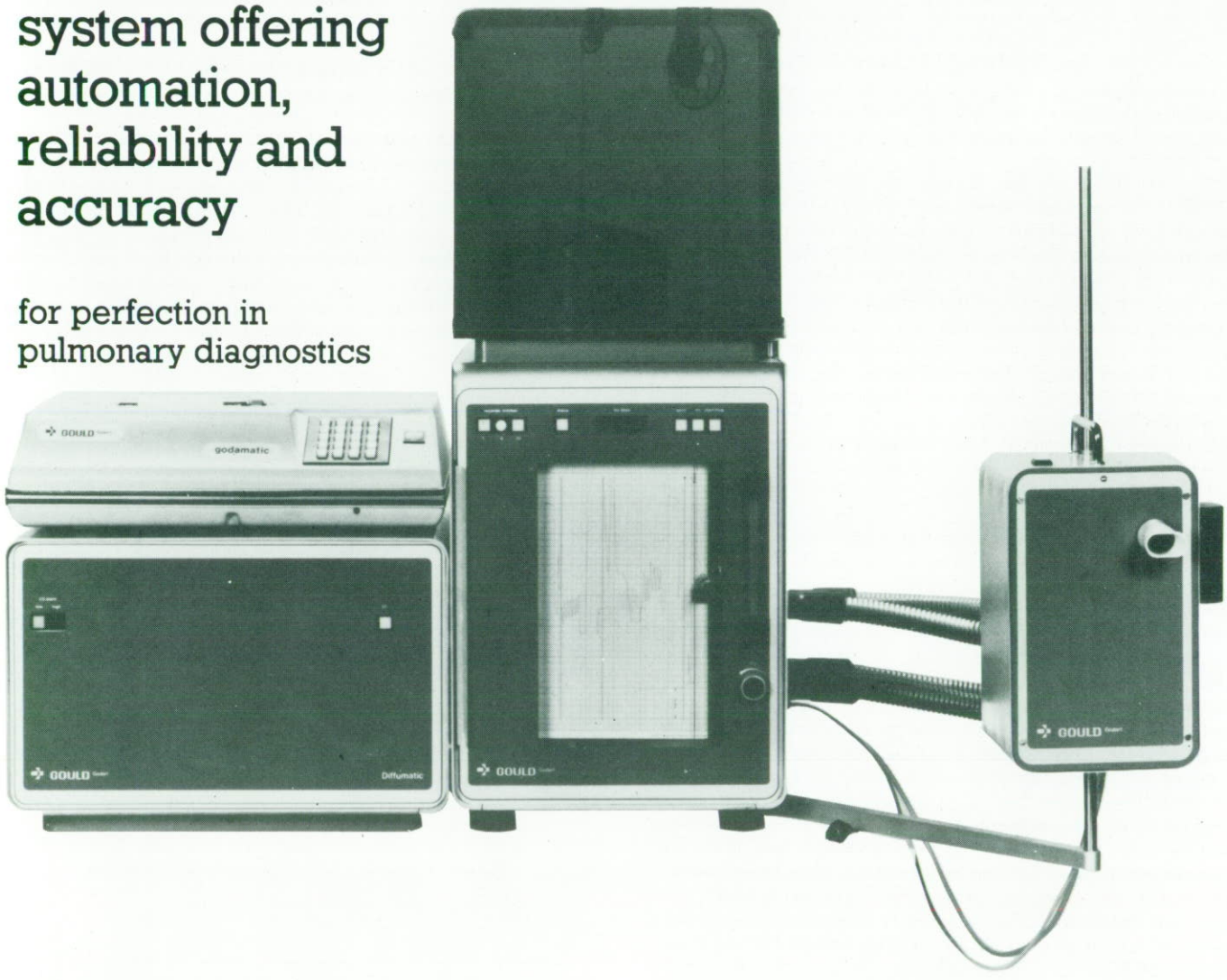

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If the lung volumes are measured by the helium dilution method, a decrease in the time taken for the helium to reach equilibrium (i.e. the intrapulmonary gas mixing time) is an indication of improved evenness of the alveolar ventilation and thus of increased ventilatory efficiency.

Serial Lung Function Tests in Chronic Obstructive Airways Disease

Similar changes should be sought in chronic bronchitis and emphysema. An important observation here is that once a patient is under effective medical control the function need not deteriorate significantly over many years. We have instances where over 10 years or more the lung volume and ventilation findings have not changed more than could be attributed to increasing age.

Once the situation has stabilised, repeating the tests annually (at the same season each year) is usually sufficient, and we may observe changes in the degree of airway obstruction (with the bronchodilator response) and in the respiratory reserve (i.e. the difference between the MVV and the resting minute volume). But equally telling is a changing degree of hyperinflation and/or loss of volume into bullae and deterioration in the diffusing capacity or arterial blood gases[3]. Refinements in the assessment of gaseous exchange, such as measurement of the ventilation/perfusion relationship, can add enormously to the understanding of what changes are taking place.

Fibrotic Lung Disorders

Serial lung function tests are essential in the management of fibrosing alveolitis especially when treatment with steroids is instituted, because of the time difference between changes in function and changes in radiographic and clinical findings. Furthermore, the effects of steroid treatment on the psyche invalidates subjective impressions of improvement or deterioration.

During initial assessment and stabilisation on steroids, weekly tests are required, but three-monthly testing on an out-patient basis is sufficient after this. Once the condition enters the chronic, stable phase, annual tests are all that are necessary. The greatest value in any repeated series of tests is gained by identifying the aspects of lung function which show most abnormality in each individual subject and then by watching how these change. It is not always the transfer factor which is most affected in fibrosing alveolitis. Hyper-support of the airway walls (where lung stiffness produces abnormally fast expiratory flow rates), airway crowding (increased closing volume and other evidence of airway obstruction in the dependent areas of the lung), arterial desaturation, and motility (reduced MVV) can be important.

Exercise Tests

Exercise tests have their value in all conditions and a useful exercise/D_{CO} method has recently been introduced[4].

The test procedure imposed upon a patient must always have strict relevance to the condition and it is equally important that the patient should appreciate this. Most of the common static tests fill this requirement (with a word of explanation from the operator). Difficult or distressing procedures can be counter-productive by causing resentment, discomfort and confusion in the subject.

Some exercise procedures do not lend themselves to serial use because of these effects. The 12-minute walking test[5], however, seems to be well tolerated and is gaining wider and wider acceptance, just because it has so much direct relevance to everyday life. It is easy for the patient to understand the requirements of the test and reassurance is created by the knowledge that the walking speed is optional (with some encouragement from the operator!) and that the test can be stopped at will. The test also lends itself to use in various treatment regimens, such as portable oxygen.

One particular instance of which the present writer has experience concerns a patient who underwent a right pneumonectomy. The static tests showed considerable bronchitis yet the patient, who was a man in his middle fifties, was active and well. Before operation he attained a walking speed of about four miles per hour over the 12 minutes. The test was repeated three and six months after operation. Although on the first post-operative occasion the walking distance was reduced, three months later the distance walked was 100 yards more than before operation.

General Rules

It is therefore most important to understand the reasons for doing repeated tests and thus good liaison between the laboratory staff and the clinician is essential. After that we require informed interpretation of the findings by the reporting officer and careful assimilation of the data in the ward or clinic.

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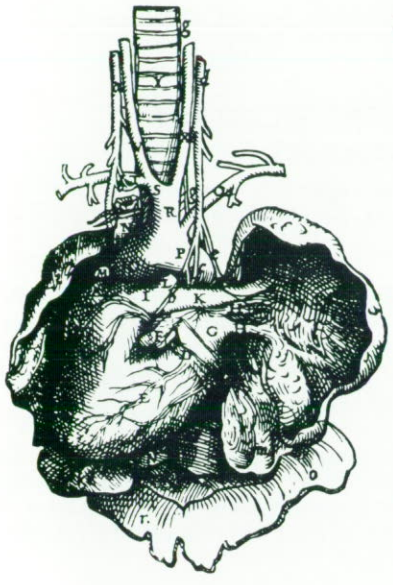

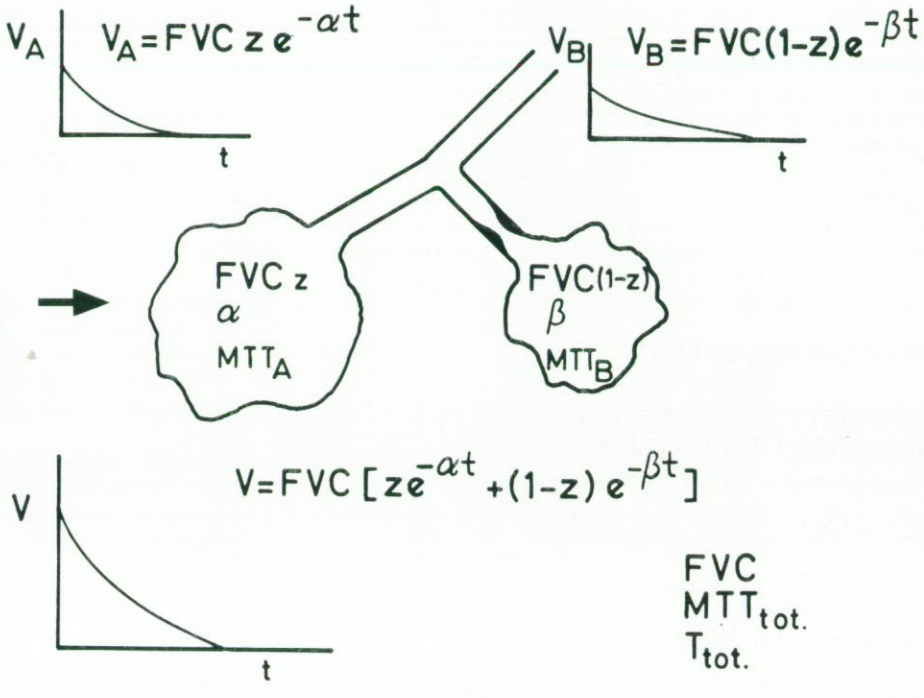

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ARTP NEWS

The Section of Measurement in Medicine of the Royal Society of Medicine has approached us to see if we would be interested in an affiliation with them. All the other technicians' societies have the backing of a larger and more influential medical group and the Executive Committee felt that it was worthwhile investigating the possibility. It was suggested that we might consider having an annual meeting with them, but it was emphasised that this was not an attempt at a take-over bid. There are, of course, other societies that we could consider an affiliation with, such as the Thoracic Society, but these tend to be much larger and more impersonal, and they have not approached us. If any members feel strongly either one way or the other, could they write to the editor of BREATH and express their opinions before any further action is taken.

FORTHCOMING EVENTS

THE SOCIETY OF ANAESTHETIC LABORATORY TECHNICIANS

The next scientific meeting will be held on the 24th and 25th September 1982 at the Institute of Naval Medicine, Alverstoke, Gosport, Hampshire.

Further details of this meeting are available from:—
Mr. R. Howard,
Royal Naval Hospital,
Haslar,
Gosport,
Hampshire.

THE ASSOCIATION OF RESPIRATORY TECHNICIANS AND PHYSIOLOGISTS ANNUAL GENERAL MEETING

The Annual General Meeting of the Association will be held on Saturday October 16th 1982 at Harefield Hospital, Harefield, Uxbridge, Middlesex. Coffee and registration will be at 10.30 am and the morning session will start at 10.45 am. The chairman will be Miss A. L. Morgan. There will be four very interesting talks in the morning, followed by lunch and demonstration. The Annual General Meeting will be in the afternoon.

Peter Lockwood is organising the meeting and those of you who went to Harefield a few years ago will remember what a very enjoyable and informative meeting it was. You will be receiving further details nearer to the time.

Remember that if you want to make any amendments to the Constitution, you must inform the secretary at least eight weeks before the AGM.

1983 SPRING MEETING OF THE ASSOCIATION

We soon have to decide on a meeting place for the 1983 Spring Meeting. Much more interest is generated if meetings are held in different regions, and attendance seems to be greater outside London. We are therefore looking for volunteers to arrange this meeting. Anyone who would like to do this, is asked to contact Gillian Lowe as soon as possible, at:—
Cardiothoracic Unit,
Derbyshire Royal Infirmary,
London Road,
Derby.
'Phone: 0332 47141

SUBSCRIPTIONS FOR 1982-1983

Members are reminded that their subscriptions are now due. Junior membership is £5.00 and Full and Associate membership is £7.00. Please send your fee to: The Treasurer, Gloria Holbrook, Lung Function Lab., Nevill Hall Hospital, Abergavenny, Gwent.

MESSAGE FROM THE SECRETARY REGIONAL ORGANISERS

Elections for regional organisers are now due and your representatives should be contacting you in the near future.

It is vital that the regional organisers play an active part in the Association. This involves attending two council meetings a year, (usually held at the time of the Annual General Meeting and Spring Meeting) and passing on information within your region.

The idea of having regional organisers is to ensure that the Association is not run by a small number of people which is what so often happens. So please let me have the name of the regional organiser in your area by July 31st, 1982.

AMENDMENTS TO CONSTITUTION

The Annual General Meeting is to be held in October 1982. Any person wishing to propose amendments to the Constitution should let me have them in writing by August 1982.

Gilliam Lowe,
Secretary

SPRING MEETING OF THE ASSOCIATION

The Spring Meeting took place at The General Hospital, Birmingham, on 3rd April, 1982. We are much indebted to Susan Hill for arranging a very interesting meeting and providing an excellent lunch.

We are most grateful to the following firms who sponsored the meeting: a number also put on demonstrations of their products.

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The following scientific papers were given:

Variations in response to the cytotoxicity of cigarette smoke.

J. Hopkins, Queen Elizabeth Hospital, Birmingham

Cigarette smoke is lethal stuff, but for the precise explanation of how it produces its effects (in emphysema particularly) we have to get right down to the cellular

level. The viability of polymorphs can be tested by their ability to take up dye particles; cigarette smoke even in low concentrations can kill a proportion of these cells, so that the bronchial defences against bacteria are lowered. The effect seems to be more marked in emphysema but is it cause or effect? (Smokers need not wait for an answer to this question!)

Advances in cystic fibrosis

A. H. Jackson, Selly Oak Hospital

Cystic fibrosis is a hereditary disorder which affects mucus production throughout the body. In childhood it presents with malabsorption of foodstuffs from the gut, but the most severe effects are upon the lungs; many patients die prematurely from respiratory failure in their teens or in early adult life. In this interesting talk we heard how the life-expectancy has improved dramatically over the last two decades both from greater awareness of the condition and from better treatment. Pseudomonas is one of the most dangerous invaders, but administration of specific antibiotics was shown to result in fewer hospital admissions and an improvement in lung function — a most encouraging advance.

Proteases and antiproteases in lung disorder

R. A. Stockley, The General Hospital, Birmingham.

Proteolytic enzymes released from circulating polymorphs can attack the elastic tissue of the lung; normally the tissues should be protected against these enzymes by specific inhibitors such as alpha$_1$ antitrypsin, which can be found in serum, in interstitial fluid and in the bronchial secretion. So there is normally a 'balance' between enzymes and inhibitors and Dr. Stockley reviewed the many ways in which this balance can be upset. In emphysema, the polymorphs contain considerably more enzymes than normal; on the other side of the balance the inhibitors can be damaged by oxidants derived from cigarette smoke, from bacteria or even from the polymorphs themselves during phagocytosis. A very complicated subject, but again smokers should not wait for the answers!

Acute Mountain Sickness

A. D. Wright, The General Hospital, Birmingham.

Ascent into high mountains without time for acclimatization can lead to the condition of 'acute mountain sickness', where the victim complains of headache, tiredness, fluid retention and of feeling generally terrible. This is partly related to the low inspired oxygen tension and partly to the fact that this low tension stimulates respiration and lowers the body CO_2 stores. Dr. Wright recently led an expedition to Mount Kenya to an altitude of 16,000 ft. He and his colleagues took the drug acetazolamide (which inhibits carbonic anhydrase) with very beneficial results. How this works is not really understood, but does it really matter, when you can observe the beautiful scenery?

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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