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BREATH

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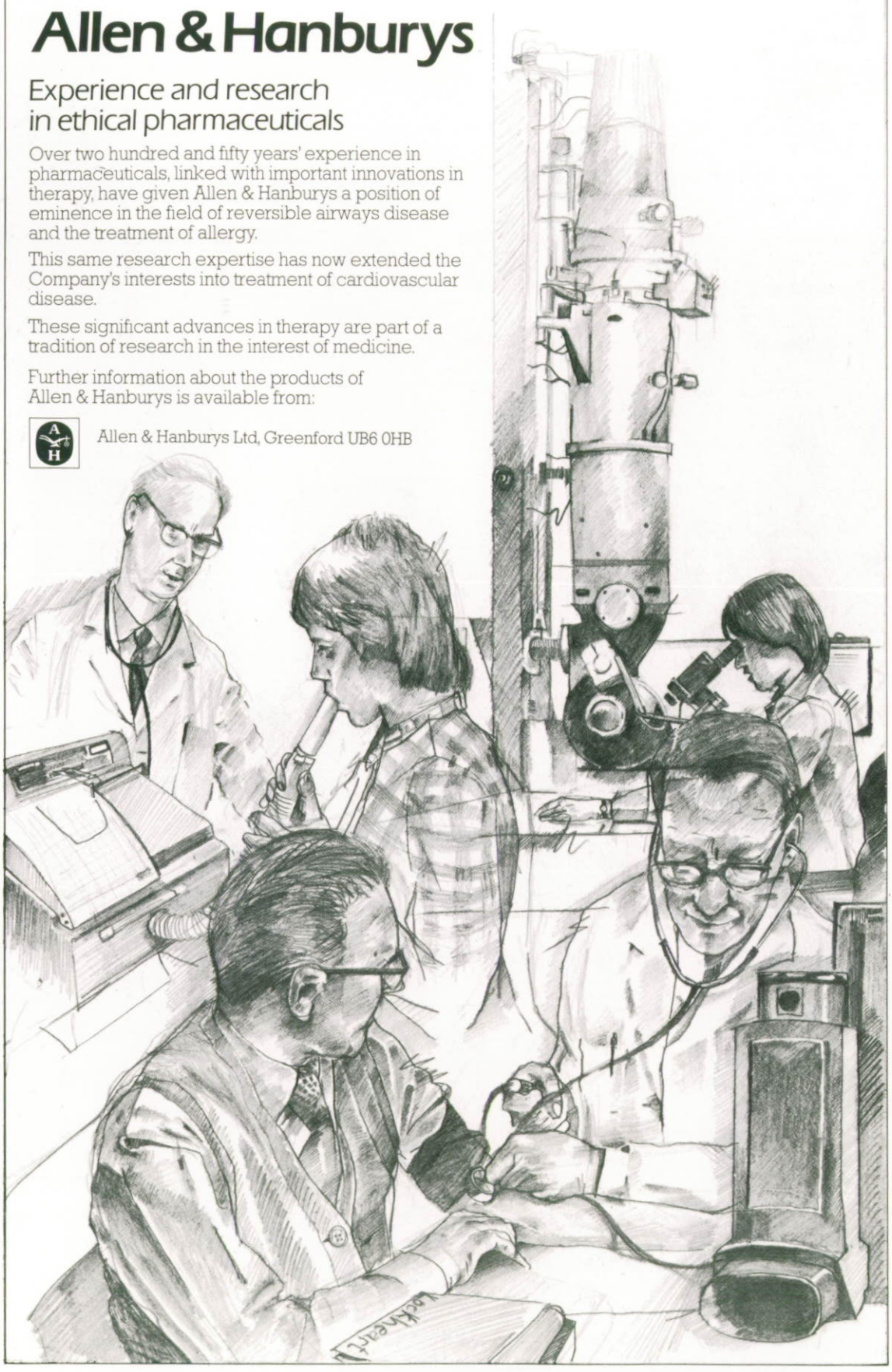

ANNUAL GENERAL MEETING

6th OCTOBER, 1984

AT

SUITE B, CONFERENCE CENTRE
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SHEFFIELD

PROGRAMME

10.30 – 11.00	**Coffee and Registration**	
11.00 – 11.30	**How the Pulmonary Function Unit can help in diagnosing Occupational Chest Diseases.**	**Dr. J. Dornan**
11.30 – 12.00	**Patient associations – trials and tribulations**	**Dr. S. Brennan**
12.00 – 12.30	**High dose bronchodilators**	**Dr. P. B. Anderson**
12.30 – 2.30	**Lunch**	
2.30 – 3.30	**Annual General Meeting.**	
3.30 –	**Tea.**	

Registration forms will be forwarded before the meeting. Overnight accommodation can be arranged as can transport from the Railway Station.

COR PULMONALE

R. J. D. Winter
The London Chest Hospital

Introduction

Pulmonary heart disease and cor pulmonale are terms that can be used synonymously to describe the effects of lung disease on the right ventricle. A World Health Organisation expert committee defined cor pulmonale as: *"Hypertrophy of the right ventricle resulting from diseases affecting the function and structure of the lung, except when these pulmonary alterations are a result of diseases that primarily affect the left side of the heart or congenital heart disease".*[1] It follows that before a diagnosis of cor pulmonale can be made it is essential to exclude congenital heart disease or an independent disorder affecting the left ventricle.

Aetiology

Cor pulmonale results from any severe disease process affecting the lungs; the principal causes are shown in Table 1. These can be grouped into conditions affecting primarily the airways such as chronic bronchitis and emphysema, diseases affecting the pulmonary vasculature such as primary pulmonary hypertension, and disorders causing impaired function of the thoracic cage.

Prevalence

Cor pulmonale is difficult to diagnose unless right ventricular failure has supervened because physical signs are frequently minimal or absent. As a result cor pulmonale is probably underdiagnosed and estimates of prevalence may be inaccurate. Careful autopsy studies on the other hand have shown an increase in right ventricular weight in 40% of patients with chronic bronchitis.[2] Clinical

TABLE 1

Classification of cor pulmonale according to principal causative diseases.

1. Diseases affecting the air passages of the lungs or alveoli.
   a) Chronic bronchitis and emphysema
   b) Pulmonary fibrosis due to:
      tuberculosis, sarcoidoisis, malignant infiltration, bronchiectasis, cystic fibrosis, cryptogenic fibrosing alveolitis or berylliosis.
   c) Pulmonary resection.
2. Diseases affecting the pulmonary vasculature
   a) Primary arterial disease, e.g.
      Primary pulmonary hypertension, polyarteritis and other forms of arteritis.
   b) Thrombotic and embolic disorders, e.g.
      Pulmonary embolism, chronic pulmonary thrombo-embolic disorders.
   c) High altitude hypoxia.
3. Upper airways obstruction.
   a) Obstructive sleep apnoea.
   b) Tracheal stenosis.
4. Disorders affecting movement of the thoracic cage.
   a) Chest wall deformity, e.g.
      Kyphoscoliosis or thoracoplasty.
   b) Impaired ventilatory drive, e.g.
      Central sleep apnoea or primary alveolar hypoventilation.

practice suggests that cor pulmonale frequently complicates chronic bronchitis and emphysema, which affects men more often than women, probably as a reflection of occupational factors as well as patterns of tobacco smoking. Cor pulmonale also occurs in cystic fibrosis[3] and may be seen in cryptogenic fibrosing alveolitis. Very exceptionally it may complicate poorly controlled asthma.

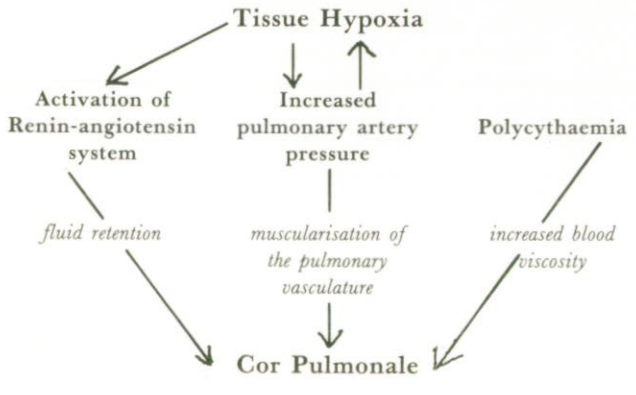
Mechanism

The pulmonary vasculature in health is a highly distensible structure and large increases in blood flow can occur, for example during exercise, with only a small increase in pulmonary artery pressure. Neural control is believed to be minimal and there is no counterpart to the renin-angiotensin system in the pulmonary vasculature. Obliteration of part of the pulmonary vascular bed as might occur in emphysema or in thromboembolic disease would be expected to lead to an increase in mean pulmonary artery pressure and eventually to right heart failure. Experimental studies have shown however that a very substantial part of the pulmonary vasculature needs to be obliterated before there is any appreciable increase in pulmonary artery pressure,[4] suggesting that this does not play an important role in the pathogenesis of cor pulmonale. This is supported in man by the observation that there is very little rise in pulmonary artery pressure following lung resection.

In man the principal pressor mechanism has been identified as hypoxia and this appears to be the most potent stimulus to pulmonary arterial vasoconstriction. The mechanism by which this occurs is not fully understood at present, but one possibility is that certain vasoactive substances are released as a result of hypoxia.[5] Hypoxic pulmonary vasoconstriction due to this mechanism may be modified by extrapulmonary reflexes, but it is known that hypoxia can induce vasoconstriction in isolated lung preparations devoid of all nervous connections. The proposed role of vasoactive substances is further strengthened by the observation in experimental animals that the development of pulmonary hypertension in response to prolonged hypoxia may be prevented by methyl dopa.[6]

Tissue hypoxia also leads to activation of the renin-angiotensin system with resultant retention of salt and water. Erythropoietin may be released from the kidney in response to hypoxia with the result that red cell formation is stimulated. If hypoxia is sustained then polycythaemia (an increase in the red cell mass) will develop, leading to an increase in whole blood viscosity. Both these factors lead to a further load on the right ventricle and will enhance the development of pulmonary hypertension (fig. 1).

Fig. 1. *Factors in the pathogenesis of pulmonary hypertension and cor pulmonale.*



Clinical Diagnosis

The diagnosis depends on demonstrating signs of right ventricular enlargement, having first excluded congenital or left sided heart disease.

A resting tachycardia and ankle oedema for which no other cause can be found, are valuable physical signs. Cyanosis is likely to be present but mild degrees are often difficult to detect. The jugular venous pressure may be raised and there may be a palpable right ventricular impulse. All too frequently the auscultatory findings of a loud pulmonary component of the second heart sound and a right ventricular third heart sound are inaudible because of loud breath sounds or interposed lung tissue.

Lung function tests may indicate the nature of the underlying disease. Signs of right ventricular strain may be seen on the electrocardiogram and radionuclide ventriculography is likely to show a reduced right ventricular ejection fraction.

Prognosis

Until recently the prognosis in cor pulmonale was poor and a five year survival of less than 50% is frequently quoted.[7] Recent developments in treatment make it possible to predict some improvement in prognosis over the next decade; two important studies have demonstrated the value of long term oxygen in reducing pulmonary artery pressure and also in increasing survival in some patients.[8,9] Recently an orally active respiratory stimulant, almitrine, has been shown to produce an improvement in arterial oxygen saturation coupled with a reduction in signs of right heart failure.[10] There is also renewed interest in the use of vasodilators.[11]

Management of cor pulmonale

Treatment of cor pulmonale is aimed at relieving symptoms and lowering pulmonary artery pressure, thereby reducing the load on the right ventricle. Oxygen will lower pulmonary artery pressure acutely[12] and will relieve breathlessness.[13] Both the British MRC trial[8] and the American nocturnal or continuous oxygen trial[9] have shown a significant reduction in pulmonary artery pressure in patients treated with oxygen for more than 15 hours each day. Unfortunately oxygen is both expensive and inconvenient and may not be suitable for patients with chronic hypercapnia. However, experience with oxygen concentrators, which enable cheaper and more convenient delivery of oxygen, is encouraging[14] and it is probable that they will be more extensively used in the future. When a diagnosis of cor pulmonale has been made, a formal trial of oxygen should be undertaken to help decide whether the patient would benefit from long term domiciliary therapy.

More recently an orally active respiratory stimulant has been developed. This drug, almitrine, has been shown to produce a sustained increase in chemoreceptor afferent nerve discharge at a given level of hypoxia, thereby stimulating respiration.[15] Clinical trials are still in progress but preliminary studies are encouraging. The drug has been shown to produce a sustained improvement in PaO_2 and a reduction in the signs of right ventricular failure in patients with chronic bronchitis.

Activation of the renin-angiotensin system leads to fluid retention, which is conventionally treated with diuretics. Spironolactone, an aldosterone antagonist, is a logical choice. However, it should be remembered that patients with pulmonary hypertension and impaired right

ventricular function require a high right ventricular filling pressure and thus the use of potent diuretics needs to be tempered with caution. The use of loop diuretics can result in a metabolic alkalosis, which will impair the ventilatory response to CO_2. There is little evidence to suggest that digoxin confers any benefit when the patient is in sinus rhythm and patients with cor pulmonale appear to be especially susceptible to its toxic effects.

Secondary polycythaemia can be treated with venesection but unless the procedure is performed isovolaemically, it may result in a potentially hazardous acute reduction in right heart filling pressure, and later a rebound increase in platelet count. Erythrapheresis is an isovolaemic method of treating polycythaemia[15]; by this technique the red cells are removed and the plasma and other blood constituents are then returned to the patient. However, the equipment used is expensive and an acceptable alternative is isovolaemic venesection, where the volume of blood removed is replaced by dextrose or normal saline.

Considerable improvement in cor pulmonale is frequently seen with treatment of the underlying lung disease. Treatment of infection, nebulised bronchodilator drugs and professional physiotherapy in patients with chronic bronchitis and emphysema frequently produce a substantial improvement in right heart failure and a reduction in pulmonary artery pressure.

Summary

Cor pulmonale is an important cause of disability in patients with lung disease. Physical signs are frequently minimal until right ventricular failure has supervened. Recent interest has focussed on the use of long term oxygen, the role of respiratory stimulants and the value of vasodilators in treatment. These developments may result in improved treatment of the disease and better prognosis.

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SLEEP DISORDERED BREATHING: Its Causes and Management

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The London Hospital and The London Chest Hospital

In a previous article[1] I detailed the various ways in which respiration may be studied during sleep. Sleep studies in which respiration is monitored allow the diagnosis of sleep disordered respiration, which may have been suspected on clinical grounds, to be confirmed. The purpose of this paper is to describe the common disorders of respiration which occur during sleep and to discuss their clinical presentation and management.

These common disorders are central and obstructive apnoeas, and nocturnal hypoventilation with normal respiratory rhythm. For each of these conditions adequate assessment is necessary to make the diagnosis and decide on the best line of treatment[2].

Obstructive Apnoea Syndrome

Obstructive apnoea (Table 1) is defined as a period during which airflow ceases despite continuing respiratory effort; it is characterised by indrawing of the chest in a paradoxical manner during attempted inspiration. Obstruction is commonly produced by collapse of the pharynx, when further inspiratory effort leads to collapse of the upper chest. Straining against an inspiratory obstruction leads to an increase in vagal tone, and hence bradycardia, which may be exacerbated by the accompanying hypoxia and associated with ventricular ectopic beats or other dysrhythmias. Nasal obstruction, an enlarged tongue or tonsils, narrowing of the pharynx caused by fat or myxomatous deposits in myxoedema and acromegaly, may produce partial obstruction to airflow which may become complete during sleep[3].

TABLE 1

Features of Obstructive Sleep Apnoea

Narrow upper airway Sleep Muscle hypotonia	*Leading to:*	
PHARYNGEAL COLLAPSE		
INCOMPLETE:	*Leading to:*	Snoring
COMPLETE:	*Leading to:*	Hypoxia Dysrhythmia Arousal and poor sleep

Muscle Tone

During sleep there is a reduction in the tone of the muscles that hold open the pharynx. The genioglossus muscle normally acts to pull the tongue forward preventing it from slipping back and causing airway obstruction. With the onset of sleep there is a reduction in tone of this and other pharyngeal dilator muscles, and during REM sleep, and dreaming, hypotonia may be profound. This has the effect of changing the pharynx from a firm muscular tube, to a floppy 'bag', which can easily collapse. In the awake subject, any collapse of the airways can be rapidly sensed by the upper airway receptors and the response to hypoxia and hypercapnia is likewise brisk, leading to enhanced muscle tone and an increase in ventilation. During sleep, however (and particularly during REM sleep) these reflexes are much less active so that the protective mechanism is partly lost; muscle relaxants such as diazepam or alcohol can also increase the possibility of obstruction.

Symptoms

Obstructive apnoea most commonly presents with severe snoring. During sleep there is always some degree of obstruction to airflow in these patients, which leads to noisy breathing. From time to time the obstruction becomes complete and silence ensues. Often the obstruction leads to arousal; the patients wake with a start, fling out their arms and legs and make a loud noise as the obstruction is relieved. Although the patients may report these symptoms themselves, it is often their wives or other family members who are the best witnesses, having observed obstruction with waking for a considerable time. Frequent obstructive apnoea leads to very poor sleep, with often no more than two or three minutes of sleep between obstructive episodes and arousals. The constant sleep deprivation makes the patients very sleepy during the day and they may fall asleep at inconvenient times, when driving, at work or whenever they sit down; they are then woken by an obstructive apnoea. This extreme daytime hypersomnolence associated with loud snoring is characteristic of the obstructive apnoea syndrome. Depression, anxiety and impotence are common in this group of patients.

If respiration during sleep is severly affected by obstructive episodes then respiratory failure may be produced, with a reduction in arterial pO_2 and increase in pCO_2 occurring overnight. As the frequency and severity of the obstructive episodes become still worse, there may be a diminution of respiratory drive even when awake and the patient may be in continuous respiratory failure. If respiratory failure only occurs at night, the main symptom is morning headache caused by the increased pCO_2.

Management (Table 2)

In the majority of patients with obstructive sleep apnoea, the diagnosis can be established from the history of severe snoring and daytime hypersomnolence. In those few severe enough to be in respiratory failure the snoring may have diminished in intensity; in these patients respiratory failure may be associated with reasonably normal lung function and thus apparently unexplained. The diagnosis may be confirmed by questioning the relatives on the patients' sleeping habits, and the severity can be assessed by a formal sleep study.

TABLE 2

Treatment of Obstructive Sleep Apnoea

Removal of upper airway obstruction
Tracheostomy
Pharyngeal reconstruction
Weight loss
Continuous positive airway pressure

Such patients were originally treated with a permanent tracheostomy, thus bypassing the pharynx and abolishing the problem. An obvious mechanical obstruction in the pharynx or nose can be removed. In the USA operative reconstruction of the pharynx has become popular but has achieved only moderate success. In obesity, general weight loss can improve the situation by reducing peripharyngeal fat. Most recently a simple mechanical method of treatment has been found to be very successful. The treatment, nasal continuous positive airway pressure (CPAP), consists of air directed through the nose into the pharynx at a pressure of 5-15 cm H_2O. The air is delivered through nasal cannulae or via an anaesthetic mask attached over the nose. This effectively dilates the pharynx, preventing its collapse and stimulating the upper airway receptors to increase upper airway muscle tone[4].

Nasal CPAP is a highly effective and simple method for the alleviation of obstruction in patients with obstructive sleep apnoea. When the obstructive episodes have been relieved, a more normal night-time sleep returns with loss of the daytime hypersomnolence and an increase in respiratory drive so that respiratory failure may disappear. If nasal CPAP is used in obesity it allows time for weight to be lost, and as the pharyngeal fat decreases the treatment may be discontinued. Thus a simple non-surgical approach to this problem may be all that is necessary and tracheostomy can be avoided. (Fig 1).

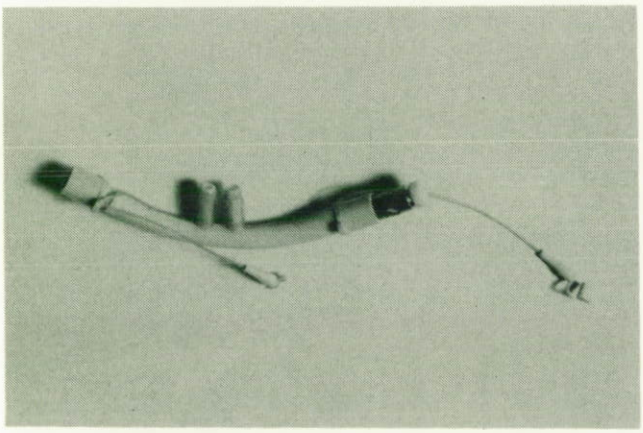

Fig. 1. *STRADLING – PORTEX NASAL CPAP TUBE*
Continuous positive airway pressure delivered through the nose.

Central Sleep Apnoea

Central apnoeas are seen much more commonly than obstructive apnoeas. A central apnoea is said to occur if there is cessation of airflow at the mouth and nose or larynx, with no evidence of respiratory effort. Central apnoea occurs because of failure or diminution of the central drive to respiration, and may be part of a periodic waxing and waning respiratory pattern (Table 3). If the apnoea is short there is often little change in the blood gases, as the subsequent increase in ventilation corrects for any underventilation at the time of the apnoea. If the apnoea is longer than 30 seconds there may be moderate desaturation; often such long apnoeas are terminated by arousal and waking. Central apnoeas are much less often associated with dysrhythmia then the central type, both because they are shorter and because there is no increase in vagal activity as would occur on attempting to inspire against an obstructed pharynx. Rarely are central apnoeic episodes as frequent during the night as obstructive episodes, so that even if the long central apnoeas are associated with arousal and waking, they seldom cause sleep deprivation and daytime hypersomnolence although they may be associated with lethargy, depression and anxiety[5].

TABLE 3

Features of Central Sleep Apnoea

1 Apnoea a) Self-terminating
b) Arousal and waking
c) Hypoxia if prolonged

2 Irregular respiration without apnoea:
a) Without hypoventilation
b) With hypoventilation and hypoxia.

The diagnosis of significant central apnoea is made more difficult by the fact that minor episodes are very common. In any individual over the age of forty, residing at high altitude, or suffering from congestive cardiac failure, waxing and waning of respiration on going to sleep is common. Periodic variations in respiratory amplitude, with periods of cessation of respiration are known as Cheyne-Stokes or periodic respiration. If the apnoeas are short there may be neither arousal nor desaturation and the respiratory rhythm is then of no pathological significance. It is rare for this "normal" irregularity to be so marked as to lead to long apnoeas and frequent waking, or significant hypoxaemia.

Management

As with obstructive apnoeas, it is often the spouse who has noted the central apnoeas and, worried by their frequency and duration, has sought medical attention. The diagnosis may be confirmed by a sleep study which will allow assessment of the frequency, duration and severity of the problem and will show whether the apnoeas are associated with waking or significant hypoxaemia.

Treatment of this condition depends upon its severity (Table 4). If the apnoeic episodes are short and unaccompanied by hypoxia or sleep disturbance, no action is required but if hypoxia or frequent waking are major problems, treatment will be necessary. A variety of drugs have been used as respiratory stimulants in these patients. Theophyllines and medroxyprogesterone have been tried but with limited success though acetazolamide, a carbonic anhydrase inhibitor, has been found to be more effective[6]. Acetazolamide inhibits carbonic anhydrase, an enzyme central to the control of acid base balance. The drug renders the CSF more acidic, increasing hydrogen ion concentration by reducing the degree of buffering available. Increased hydrogen ion concentration acts on the brain stem chemosensitive areas and leads to an increase in respiratory drive which can superimpose a more regular respiratory pattern. Acetazolamide has been used successfully to abolish the Cheyne-Stokes respiration seen at high altitude and has been used on a limited number of patients with central apnoeas, abolishing them and returning sleep and respiration to normal.

TABLE 4

Treatment of Central Sleep Apnoea

Mild form: no treatment required
Acetazolamide
Diaphragm pacing
Mechanical ventilation.

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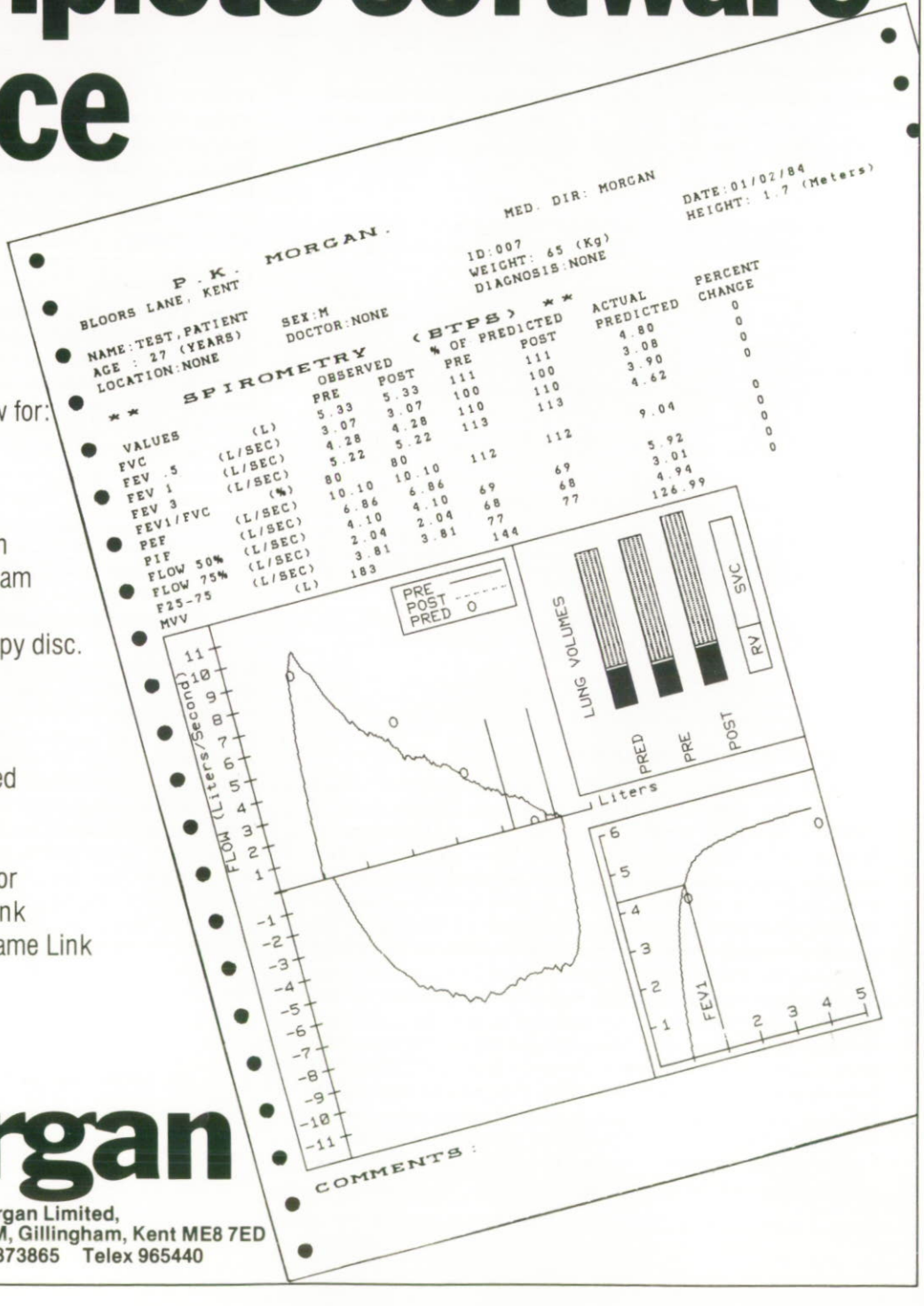

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Hypoventilation

In all subjects there is a reduction of respiratory drive, and in the responsiveness to hypoxia and hypercarbia during sleep; there is a reduction in arterial pO_2 and a small increase in pCO_2. The reduction in respiratory drive is moderate during non-REM sleep, but is much more profound in REM sleep when there is generalised muscular hypotonia, and in particular the contribution of the intercostal muscles to respiration is almost abolished. In any patient who is already hypoxic when awake, as a result of respiratory or cardiac disease, there will be a further fall in pO_2 during sleep with a worsening of any existing respiratory failure[7].

Patients who underventilate to an abnormal extent during sleep are said to suffer from nocturnal hypoventilation. The arterial pO_2 may fall precipitously on going to sleep and the pCO_2 will rise. Characteristically they feel worse after a good night's sleep than when they went to bed, with morning headache and lethargy. These patients may present with the consequences of hypoventilation, daytime respiratory failure, polycythaemia and cor pulmonale, but may have reasonably normal lung function tests and lung mechanics. A number have nocturnal hypoventilation associated with a disorder of respiratory rhythm such as obstructive apnoea or central apnoea, but many have a normal respiratory rhythm with inadequate ventilation.

In a few patients diaphragm weakness or paralysis is the cause of nocturnal hypoventilation. Awake and in the erect position they can breathe adequately but lying flat they lose the advantage of gravity to aid diaphragm action; their respiratory muscle power decreases with sleep and they become hypoxic. In REM sleep they are particularly likely to develop hypoxia as they depend upon intercostal muscle activity for inspiratory power, and in REM sleep there is profound hypotonia of the intercostal muscles, making respiration dependent upon the action of the diaphragm[8].

Management

The diagnosis of nocturnal hypoventilation depends upon the demonstration of a reduced arterial pO_2 and increased pCO_2 during the night or in the early morning. It is necessary to compare day and night-time blood gases to assess whether there is a greater fall at night than would be expected. Lung function tests, blood count, ECG and chest X-ray may reveal evidence of hypoxic lung disease, polycythaemia or cor pulmonale. If the nocturnal hypoventilation is severe, early morning blood gases will be abnormal, but in less severe cases, nocturnal monitoring of haemoglobin oxygen saturation, and pCO_2 may be necessary.

Diaphragm function should be assessed; this can most easily be done by performing spirometry in the lying and standing positions. In the normal subject there is little fall in vital capacity lying down, but in diaphragm paralysis it may fall dramatically, possibly to below 1 litre. Inspiratory muscle power can also be assessed by measurement of the static mouth inspiratory maximum pressure, or by assessment of transdiaphragmatic pressure changes using oesophageal pressure measurements[8].

Treatment of nocturnal hypoventilation depends both on its severity and cause. A respiratory rhythm disorder needs specific treatment. If there is no rhythm disorder and only moderate hypoxia, drugs such as medroxyprogesterone, or theophylline can stimulate respiration in certain cases. In diaphragm failure, but with intact phrenic nerves, diaphragm pacing has been used with considerable success[9]. In some patients continuous nocturnal oxygen has been effective in reducing nocturnal hypoxia, but this may lead to an increase of pCO_2 if unduly high flows of oxygen are used. As a final option for the treatment of patients with hypoventilation, either with normal respiratory rhythm or with central apnoea, it is possible to use a negative pressure tank or cuirass ventilator to take over ventilation at night. This method of treatment has been used successfully for many years in a large number of patients with neuromuscular disorders and diaphragm weakness. Many of these patients may have evidence of bronchospasm or congestive cardiac failure, and obviously these causes of hypoxia need to be corrected as far as possible before any other techniques are utilized.

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THE ARTP EDUCATION COMMITTEE

Kelvin Houston
Llandough Hospital, Penarth
(Adapted from a communication to an ARTP Workshop, March 1984)

The Education Committee of the ARTP was formed in 1982 to deal with the numerous questions which were arising in the education and training of physiological measurement technicians; business of this nature was occupying an increasingly large amount of the time of the main Executive Committee.

Lines of Communication

Before discussing the activities of the Education Committee it is necessary to consider the relationship between the ARTP Membership, the DHSS and other bodies. This somewhat complex interrelationship is illustrated in Figure 1. The ARTP and its committees have three main lines of communication:

(1) *Department of Health and Social Security (DHSS)*
The DHSS, situated at the Elephant & Castle in South London is essentially concerned with management and control. Matters relating to the training of all grades of technical staff have now been devolved to the National Health Service Training Authority (NHSTA) and all the Regional Education and Training Officers (RETO's) report to this body. There is one RETO in each region and they are usually assisted by a Professional and Technical Trainer whom ARTP members may have had dealings with if they sit on their College MPPM Advisory Panel. This Panel consists mainly of the College MPPM lecturers and those heads of MPPM departments who are involved in teaching at the College. The trainers communicate with each other through the National Network of Scientific Professional and Technical Officers.

(2) *The ARTP itself*
The membership of the ARTP may communicate directly with the Education Committee and the Executive Committee. The business of the Education Committee is channelled through the Executive Committee, an important matter if the Association is to have a united front. Two representatives from the Executive Committee sit on the Federated Associations of Medical Technology (FAMT) and the latter body receives two members from each of the other technical associations. These representatives make up the FAMT Central Council which communicates with the DHSS to produce, one hopes, a common policy. They meet representatives of the MPPM National Training Committee where important decisions on technician training are likely to take place.

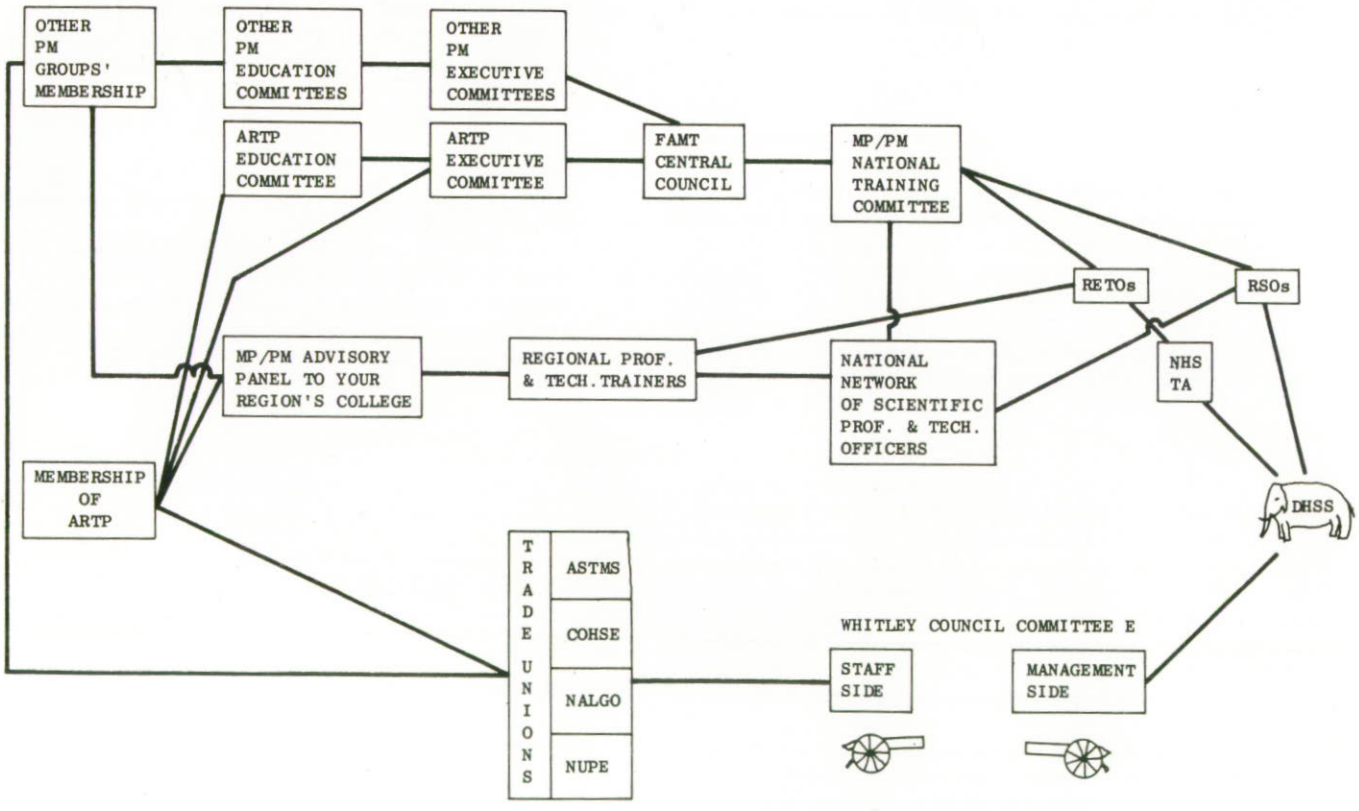

Fig 1 Interrelationships between the ARTP, the DHSS and other negotiating bodies or committees.

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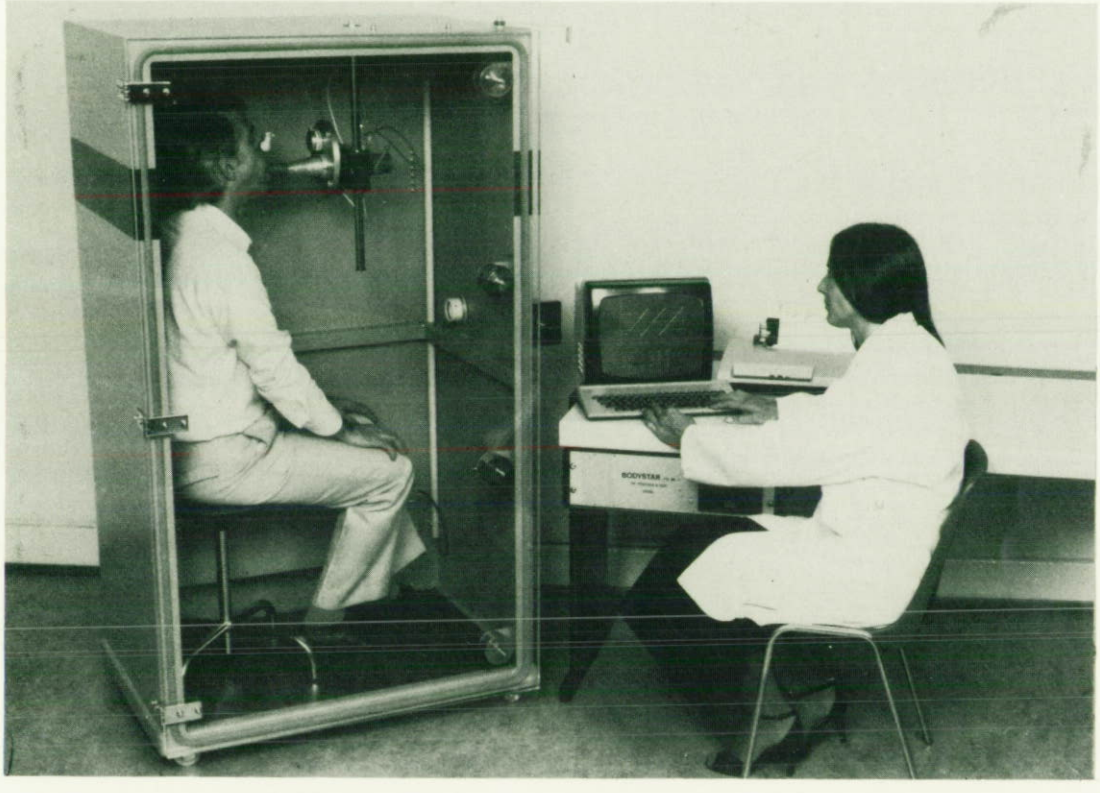

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(3) *Trade Unions*
We also have links with four trade unions which represent our interests; they are ASTMS, NALGO, NUPE and COHSE. Two representatives from each Union make up the staff side of Committee E of the Whitley Council and there are also ten management side representatives. This is where our pay and conditions of service are considered, but it is worth noting that the existence of respiratory physiology technicians was only recognised by this Committee as recently as 1981 when the conditions of service were amended in order to include members in our discipline.

RECENT ACTIVITIES OF THE EDUCATION COMMITTEE

Review of O TEC and H TEC Curricula

For the sake of accuracy it should be pointed out that the full name of O TEC is now the "Business and Technician Education Council Certificate in MP/PM (BTEC Certificate)" and H TEC is now known as the BTEC Higher Certificate. This comes about because of the merger of the Business and Technician Education Councils but the shorter and more familiar names will be used in this article. Figure 2 shows the relatively small number of colleges in England and Wales at present offering these courses. The courses themselves are disappointing in that there are very wide variations in the respiratory physiology content. With the change from ONC and HNC to O TEC and H TEC, it was to be hoped that much of this variation would have been removed as all the courses had been vetted by a TEC Committee on which the ARTP was represented. This unfortunately did not occur and we now have a situation in which some colleges run O TEC courses with virtually no respiratory physiology in the PM Unit while others include topics which are much more suitable to an H TEC course. The ARTP Education Committee is currently reviewing the available information in order to formulate a common curriculum and clearly much more liaison with the Technician Education Council is required if the physiological measurement units are to be standardised.

Fig 2
The distribution of colleges in England and Wales running MPPM O TEC and H TEC courses. Open circles: O TEC only, triangles: H TEC only, closed circles: both.

Content of H TEC Curriculum

Topics which should perhaps be included at this level are advanced sections on anatomy (with embryology), respiratory mechanics and gas exchange, the common diseases including morbid anatomy, radiology and therapeutic procedures and respiratory physiology in unusual or hazardous environments.

Practical Procedures for the H TEC Course

We consider that candidates for the H TEC certificate should have detailed knowledge of post-basic test procedures, possibly some of those listed in Table 1. It must be pointed out here that there are one or two people at the DHSS and NHSTA who do not consider that H TEC is a necessary qualification for senior and chief technicians and that O TEC would be quite sufficient. The ARTP Education Committee strongly disagrees with this view and has spent some time preparing a case supporting the need for H TEC; our views have now been forwarded to the FAMT who are presenting the case for H TEC to the DHSS.

TABLE 1

H TEC IN-SERVICE TRAINING PROGRAMME
SUGGESTED PROCEDURES

Airway Resistance and Conductance
Lung Compliance
Components of CO Transfer Factor
Alveolar – Arterial Oxygen Tension Difference
Dead Space – Tidal Volume Ratio
Shunt Fraction
Transcutaneous O_2 & CO_2 Measurement
Closing Volume
Ventilatory Response to CO_2
Bronchial Challenge Testing
Radionuclide Lung Scans

Training Workshop

The Education Committee clearly cannot work in isolation from the ARTP Membership and some time was spent in preparation of this training workshop so that the views of the Membership could be sought on a number of important points, such as the content of the O TEC syllabus, the content of the In-service Training Manual which is due for review at the end of this year, the implementation of the Manual and how it should be assessed. We are amazed to see that there are still heads of respiratory departments who have failed to realise that the In-service Training Programme is now mandatory; in fact one or two appear to be quite unaware of its existence!

Standardisation of Lung Function Tests

Even for simple procedures a number of different methods are in use in lung function laboratories and we propose to circulate a questionnaire on this topic. The variations could be important in multi-centred surveys for instance and from the point of view of training, students may have to be familiar with a number of different procedures. Furthermore, assessors of the training manual will need to be aware of the accepted practices in each Department.

Careers Leaflet

We considered that it was of the utmost importance to attract good potential students into our profession and we have drawn up a careers leaflet which can be circulated in secondary schools and elsewhere. We have included information on our role in patient care, the entry qualifications and career prospects.

PROGRESS

The progress that has been made in training during the last fifteen years can be illustrated by reference to the situation as it was in about the year 1970. At that time a certain student underwent training on a device known as the La Mer Sinclair Aerosol Generator which was used to produce particles of varying sizes for research into pneumoconiosis. At the end of two years' training the student was a master of this technique, duly qualified as a technician and moved to a post at a well-known London teaching hospital. On arrival an embarrassing situation arose: it appeared that this 'Technician' had no idea of the nature of the FEV_1, Vital Capacity, Total Lung Capacity, Residual Volume or Transfer Factor, nor any idea of how to set about measuring them. To make matters worse the hospital had no training scheme of any kind and the 'Technician' simply had to pick up the requisite knowledge as best he could.

In 1984 we have a BTEC certificate in MPPM matched with an In-Service Training Programme and with the support of the ARTP members for this Training Scheme the experience of this student need never be repeated. Was it really as bad as that, one may ask? *Well, I should know, I was that student.*

ASSESSMENT OF THE TRAINING MANUAL

Sue Hill
The General Hospital, Birmingham
(Adapted from a communication to an ARTP Workshop, March 1984)

The impetus for a National Assessment programme stems from the production in 1982 of the National Training Manuals for the In-Service Training of Student Physiological Measurement Technicians (PMT's). The production of these manuals followed the 1979 Orange Report on Training of MPPM Technicians within the TEC system and in 1981 the Whitley Council ruled that a basic grade PMT should not only have gained the TEC certificate but should also have undergone an approved period of in-service training.

Assessment of the Manuals

There are two main reasons for undertaking assessment of the Training Manuals. In the first place, assessment is an integral part of the teaching process and recurs throughout the whole tuition period. It allows the students' progress to be monitored, can have a strong influence on their motivation and can provide an important two-way communication between them and their teachers and supervisors.

Secondly, assessment is the means by which our professional standards can be maintained and by which they can be established on a nationwide basis.

The Form of the Assessment

It is generally agreed that this will fall into two main parts:

1 **Continuous assessment** will be carried out by the supervisor within the hospital department. It is likely to consist of two elements:
   (a) Diagnostic – a check on the student's understanding and progress before deciding on the next phase.
   (b) Terminal – the final assessment in each of the objectives in the Manual.

2 **Final assessment** should be a final check at the end of the training period and should be done by persons other than those responsible for the day to day teaching of the students.

Continuous Assessment

Here we are concerned with the student's ability to master the objectives in the Training Manual. These objectives have been written in 'behavioural terminology' so that a logical teaching programme can be developed which can ultimately be assessed (Table 1). By using this terminology we can specify precisely what the student should have achieved at the end of each stage; we can then carry out the assessment in order to ensure that these objectives have been attained.

TABLE 1

Behavioural Terminology

General objectives
(prefixed by a non-decimal number)

General objectives give the training goals:
eg 9 Understands the basic principles of the flow-volume curve.

Specific objectives
(prefixed by a decimal number)

Specific objectives are the means by which the student demonstrates the attainment of the general objective:
eg 9.4 Can measure a flow-volume curve with the subject breathing air.

TABLE 2

Behavioural Objectives

		'Action Verbs'
Low level	1 Knowledge	Define, state, measure
	2 Comprehension	Identify, explain, understand
	3 Application	Use, compute, demonstrate, perform
Medium level	4 Analysis	Analyse, compare, identify, break down
	5 Synthesis	Summarise, discuss, describe, organise, derive
High level	6 Evaluation	Recognise, determine, select, appreciate

The objectives can be further classified to indicate the level of difficulty (Table 2). This classification may help one to determine at what level the students should be operating in order to meet the objectives and in turn may help one to choose the appropriate method of assessment. The 'action verbs' are those words used to describe the behavioural objective and which appear in the training manual under the specific objective.

Expanding on each category:

1 Knowledge: involves remembering facts, terms and principles in the form in which they were learned.
2 Comprehension: understanding the material studied without necessarily relating it to other material.
3 Application: using generalisations or other abstractions appropriately in specific situations.
4 Analysis: breakdown of material into its constituent parts.
5 Synthesis: combining elements into a new structure.
6 Evaluation: judging the value of material for a specified purpose.

Assessment will often involve the simultaneous achievement of different levels of behavioural objective.

Methods of Assessment

1 **Written Assessment:** this may involve objective tests where the question requires the student to choose an answer from a number of options; closed questions (usually requiring the answer yes or no); open questions requiring a sentence or paragraph; essays, reports or prior notice tests.

2 **Log Book Report:** this could be a very important form of assessment. The student is expected to keep evidence of all practical and reading work carried out. This would enable a balance to be maintained between theoretical knowledge and practical experience and capability.

3 **Oral Assessment:** this would be of greater importance in testing comprehension than written assessment. The options here could include a two to five minute talk on specific objectives or on some practical activity. A topic could be introduced gradually through discussion of an item from the log book which the assessor will already have seen. They key point is to avoid making the test one of confrontation.

4 **Practical Assessment:** here the student will be observed undertaking a practical skill or a series of short practical tests. This section could include oral and/or written tests together with a practical to assess knowledge of techniques, the logical choice of steps to take in an experiment and the ability carry them out in practice. This would therefore combine different behavioural levels. The opportunity to assess the students may occur naturally during the course of training and the supervisor may be satisfied as to their competence without any formal assessment procedure.

Standard of Performance

The criteria for a minimum level of performance are not easy to determine and it may actually be easier to decide on criteria for failure. Assessment implies the collection of information about the students' progress, on the assumption that a comparison is to be made with some pre-determined standard. Unfortunately no such standard exists at the present time though we should ideally have a *national* standard for continuous assessment. This may be difficult to achieve and in practice it is more important to standardise the teaching material so that all students are taught to the same level in each of the specific objectives; the ARTP intends to produce information leaflets to accompany the in-service training manual. A plan for assessment is outlined in Table 3.

TABLE 3

Planning for Assessment

1 Define the behavioural objectives to be assessed.

2 Break down the specific objectives into:
  a Action to be performed.
  b Conditions under which performance will take place.
  c Criteria for minimum level of performance.

3 Choose an appropriate method of assessment.

Final Assessment

It has long been recognised by the ARTP that a "Final Assessment" on a national basis will be required. This will ensure that professional standards are maintained and that national uniformity is achieved by the training manual. All disciplines represented on the FAMT agree that the same format for final assessment should be adopted throughout. A number of questions however need to be answered:

1 Would individual regions be able to set up a final assessment protocol to examine all students within that region? We should note that the Trent Regional Health Authority have pointed the way be establishing their own In-Service Training and Assessment Programme.

2 Could the professional examinations already organised by some disciplines be used for the final assessment? Are they of the required standard and do they cover all aspects of the Manual?

3 Should the disciplines still without professional examinations set them up and use them as the final assessment?

4 Could the TEC validate the in-service training alongside issuing the O TEC Certificate? No, the old TEC was, as we know, not interested in validating vocational training. However with the introduction of the B TEC this year, one hopes this may be possible since they are already involved in vocational training for the business side.

A National Policy

What is the current position in 1984 for a national policy? At a recent working party meeting of the Professional and Technical Trainers, representatives of FAMT and the NHS Training Authority, it was agreed to give formal recognition to this working party and to rename it the "National Training Committee in MPPM". It was suggested that final assessment should be administered on a similar basis to the Trent RHA scheme with the professional associations making the final assessment. This would utilise some of the current professional practical examinations but would require other professions, including respiratory physiology, to introduce an assessment protocol.

TABLE 4

The Practical Assessment – Organisational Requirements

Registration of students.

Issuing of guidelines for a) students.
b) supervisors.
c) examiners.

Appointment of examiners.

Arrangements for assessment.

Examination paper.

Marking scheme.

Results.

Organisation of the Practical Assessment

The ARTP will have to consider a number of points before introducing the Final Assessment (Table 4). The ARTP believes that this examination should test only the practical tasks required by a basic grade technician and that the successful student should be considered as having reached a National Standard in Respiratory Physiology as assessed by the ARTP (though open both to members and non-members).

In conclusion, it is vital that we assess our students to an agreed National Standard to ensure that all technicians have equal opportunities for future employment in any part of the country.

THE TRENT REGIONAL EXPERIENCE OF PRACTICAL ASSESSMENT

Gillian Lowe
Derbyshire Royal Infirmary
(Adapted from a communication to an ARTP Workshop, March 1984)

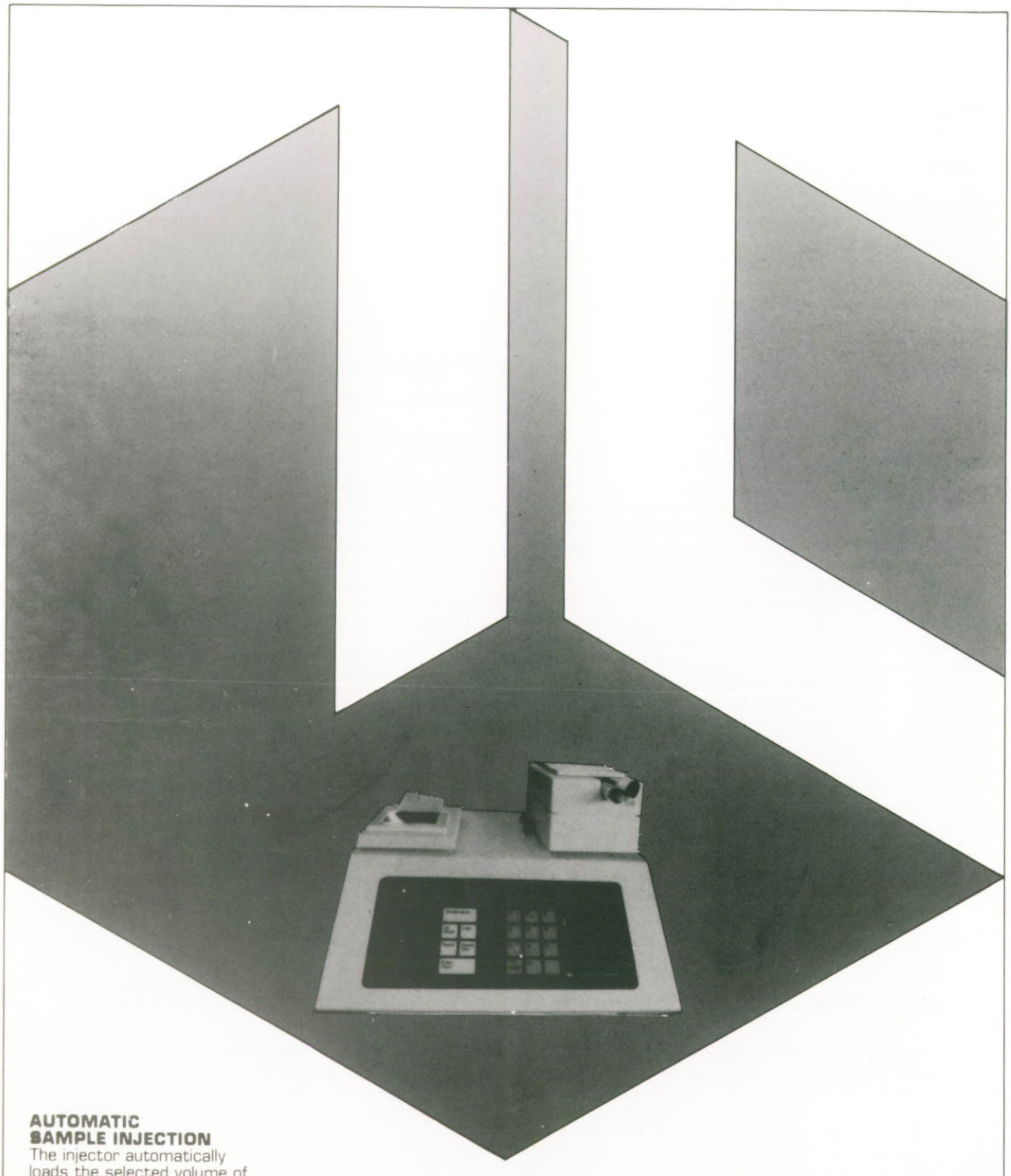
The Trent Regional Health Authority has for many years placed great importance on the training of technical staff in all disciplines. The Trent Regional Scheme and the plans for the future were reviewed two years ago by Moore and Perry[1]; at that time it was hoped that an assessment scheme could be introduced by mid-1983 and this hope was fulfilled to the extent that in 1983 the Trent Region was able to conduct practical assessments in Audiology, Cardiology, Neurophysiology and Respiratory Physiology.

A great deal of work had to be done to make this possible. During the years 1980 and 1981, practical in-service training modules were written by the Specialist Panels in each Physiological Measurement discipline. The modules were intended to form the basis of a structured in-service training programme for both in-service and supernumerary students and the Respiratory Physiology Manual was the basis for the National Training Manual which was produced in the following year. There would have been little point in all this effort without some means of assessing the students to ensure that they were trained to a satisfactory and uniform standard.

In the year 1982, therefore, a Working Group was established to discuss the formation of an Assessment Programme in Medical Physics and Physiological Measurement. The Working Group consisted of:

The Regional Scientific Officer.
The Regional Education and Training Officer for Medical Physics and Physiological Measurement.
Representatives from each of the four Physiological Measurement disciplines.
Two representatives from the Medical Physics discipline.

The Working Group in conjunction with the Specialist Panels created a general protocol for the Assessment. It was agreed that the Audiology and Neurophysiology students would sit the examinations set by their own professional societies, while the Cardiology, Respiratory Physiology and Medical Physics students would undergo an assessment written by the Specialist Panels. The following guidelines were accordingly drawn up and issued to the appropriate Heads of department, supervisors and students:



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Application for Assessment

Students are required to complete an application form endorsed by the Supervisor and by the Head of Department, and submitted to the Regional Education and Training Officer by 30th April in each year.

Eligibility for Assessment

Students presenting themselves for assessment should have:

a) Successfully completed or expect to have completed their O TEC certificate prior to the date of assessment.
b) Substantially completed their in-service training.
c) Completed a log book and training manual prior to the Assessment.

The log book should contain three sections:

1. An account of practical tests carried out.
2. All lecture notes.
3. Departmental procedures.

Outline of the Assessment

The assessment is divided into three parts, the total time allowed being two to three hours:

Section A: Mandatory Practical
Section B: Optional Practical
Section C: Oral (15-30 mins)

The students must complete Section A successfully before proceeding to Sections B and C. Students will be notified of the results of the Assessment by post and certificates will be issued to all successful candidates. In 1984 the assessment is to be based on the Trent Region Specialist In-Service Training Manual and in 1985 will be based on the National Training Manual.

Specific arrangements for the Assessment

After the registration of candidates, two Assessors (one technical and one clinical or scientific) are appointed by the Specialist Panel. The Assessors visit each department before the Assessment, decide which tests need to be made available and inspect the candidates' log-books.

The Assessment then takes place; the pass mark is 60% with a distinction at 85%. Each assessor fills in a marks sheet independently and these are then collated by the assessor, forwarded to the Regional Education and Training Officer who notifies the candidates of the result. Successful candidates are issued with a Certificate; those unsuccessful are allowed to resit the assessment on one further occasion, but in the event of a second failure, readmission to the examination would be subject to the approval of the Working Group. (In 1983 two students sat the assessment in Respiratory Physiology and in 1984 we have five students registered.)

Future Recommendations

With the introduction of the new system, a few problems arose and the Working Group's recommendations for the future are:

1. More detailed consultation should take place between the Assessor and the departments.
2. The log books should be included as part of the Assessment and more detailed instructions need to be issued about their completion.
3. A different marking scheme needs to be devised.

The arrangements for 1984 are well in hand. Following the last assessments the supervisors, assessors and the Working Group met to discuss the necessary changes and we are confident that the minor problems encountered will be solved this year.

Reference

Moore R D, Perry A E (1982). The assessment of trainee technicians; The Trent Regional Scheme. Breath *No. 17* 4-5.

ARTP News

Training Workshop and Spring Meeting of the Association

The Spring Meeting of the Association took place at Stoke Mandeville Hospital, Aylesbury, Bucks on 7 April 1984. A workshop on Training in Physiological Measurement was held on the previous day.

We owe grateful thanks to Marion Geary and colleagues for making all the necessary arrangements, to the speakers for their very interesting papers and to the many firms who provided exhibitions and joined in sponsoring the meeting.

Training Workshop

The following papers were given:

Your Education Committee	Mr K Houston
National Assessment	Miss S Hill
The Trent Experience in Practical Assessment	Miss G Lowe

After the above papers, the members participated in group discussions on a number of questions related to training and the Training Manual. Each group then reported its conclusions and this was followed by a general discussion.

Spring Meeting

The following papers were given:

Inhaled Treatment of Severe Asthma	Dr S Williams
Rheumatoid lung – fact or fiction?	Dr M Webley
Chest Medicine in Central Africa	Dr S Fisher
Optical Mapping of the Thoraco-Abdominal Wall of Spinal Patients	Dr M Morgan

The following firms sponsored the meeting and put on exhibitions of their products:

Airspec
Cardiokinetics
Cranlea & Co
Deva Medical Electronics
Garrick Equipment Co
Gould Medical
Instrumentation Laboratory
Intersurgical
Micromedical Instruments
P K Morgan
Vitalograph

Representatives from a number of the above firms gave short talks on their products and on new developments.

If any member is willing to offer a venue for the 1985 Spring Meeting, would they please contact the secretary, **Dena Muirhead** at the Derbyshire Royal Infirmary. Telephone: 0332 47141.

ANNUAL SUBSCRIPTIONS are now due. Would anyone who has not renewed their membership send their subscriptions to the membership secretary, **Sonia Jackson**: Lung Function Lab, Frenchay Hospital, Frenchay, Bristol. Full Members £7 Junior Members £5

Correspondence

The LA3 Mark 2 Lung Function Analyser

We read with interest Dr. Gunawardena's report on our Lung Function Analyser, type LA3. We appreciate the care he has taken in comparing this instrument with the Vitalograph and the Wright Peak Flow Meter (with both of which we are familiar) and would like to thank him for the kind remarks he has made about certain aspects of our own instrument. While in no way disagreeing with the data he has obtained, we would like to make some further comments.

1. *Calibration.* Dr. Gunawardena mentioned that there is a single external calibration control on the instrument and that he set this by using a 1 litre syringe as a standard volume. This, in fact, is the procedure we ourselves follow in production. In addition, we calibrate the instrument for flow against a rotameter at three points. This adjustment however is internal, as it is not felt necessary to alter the volume/flow relation once it is correct, and a volume calibration using a syringe is more convenient.

2. *Computation.* We read with interest the suggestion that the FEV 1% computation could be improved by using a microprocessor. We use this method in all our other spirometers but we were endeavouring to keep the price down as far as possible in this, which is the smallest of our range.

3. *Air Flow Resistance.* We accept the fact that the resistance when using a lint filter is somewhat higher than that recommended by the American Thoracic Society but we should point out that this filter is only provided to prevent fluff from cardboard mouthpieces collecting on the flowhead. If washable mouthpieces – such as those which we supply – are used, then this filter can safely be removed permanently. Alternatively, we can now supply a low resistance filter: in either case the resistance is below the figure recommended by the American Thoracic Society. With regard to recalibrating the flow to compensate for resistance, this would be difficult to do in any useful manner, as the instrument is calibrated against continuous flow and to apply an arbitrary correction for resistance would not give meaningful results. We think it may be of interest to append values of resistance we have measured for peak flow meters and have added those for LA3 with the new filters as well as without a filter. We would also draw attention to the linearity curves Mr. Shaw obtained for the Wright Peak Flow Meters in the paper to which Dr. Gunawardena refers.

Resistance Comparison Table (at 12 1/sec flow)

	Resistance (cm H_2O/l/sec)
LA3 (Standard)	2.9
LA3 (without Filter)	0.7
LA3 (with low loss Filter)	1.4
Mini-Wright Peak Flow Meter	2.3
Standard Wright Peak Flow Meter	0.8
Palrod Peak Flow Meter	1.4
Amer. Thor. Soc. (recommended maximum)	1.5

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A STUDY DAY
on
'DISEASES OF THE AIRWAYS'
WEDNESDAY, 14th NOVEMBER, 1984
10.30 a.m. – 4.30 p.m.
at the
Royal Society of Medicine
1 Wimpole Street
London W1

This study day is designed to provide background clinical, pathological and pharmacological information which should be of interest and value to those working in lung function laboratories.

Subjects to be covered will include: –

The structure and function of the airways; How airways may be narrowed; The assessment of airway narrowing; Bronchodilators; Asthma; Occupational asthma; Lung cancer; Chronic Bronchitis and emphysema; Respiratory failure and Sleep apnoea syndromes.

Fee – £5 to include coffee, lunch and tea.

Speakers will include Dr. H. R. GRIBBIN (London Chest and University College Hospitals), Dr. A. D. BLAINEY (St. Bartholomew's Hospital), Dr. M. R. PARTRIDGE (Whipps Cross Hospital) and Dr. M. RUDOLPH (Hammersmith and Ealing Hospitals).

Applications (which must be received by October 1st, 1984) should be sent to: –

Secretary to Dr. M. R. Partridge
Chest Clinic
Whipps Cross Hospital
Whipps Cross Road
London E11 1NR

Receipt will not be acknowledged until that date when final programme and instructions will be sent.

I should like to attend the Study Day on Wednesday, 14th November, 1984.

NAME.....

ADDRESS FOR CORRESPONDENCE.....

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HOSPITAL ADDRESS (IF DIFFERENT FROM ABOVE) .....

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I enclose a cheque for £5 payable to The Royal Society of Medicine.

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

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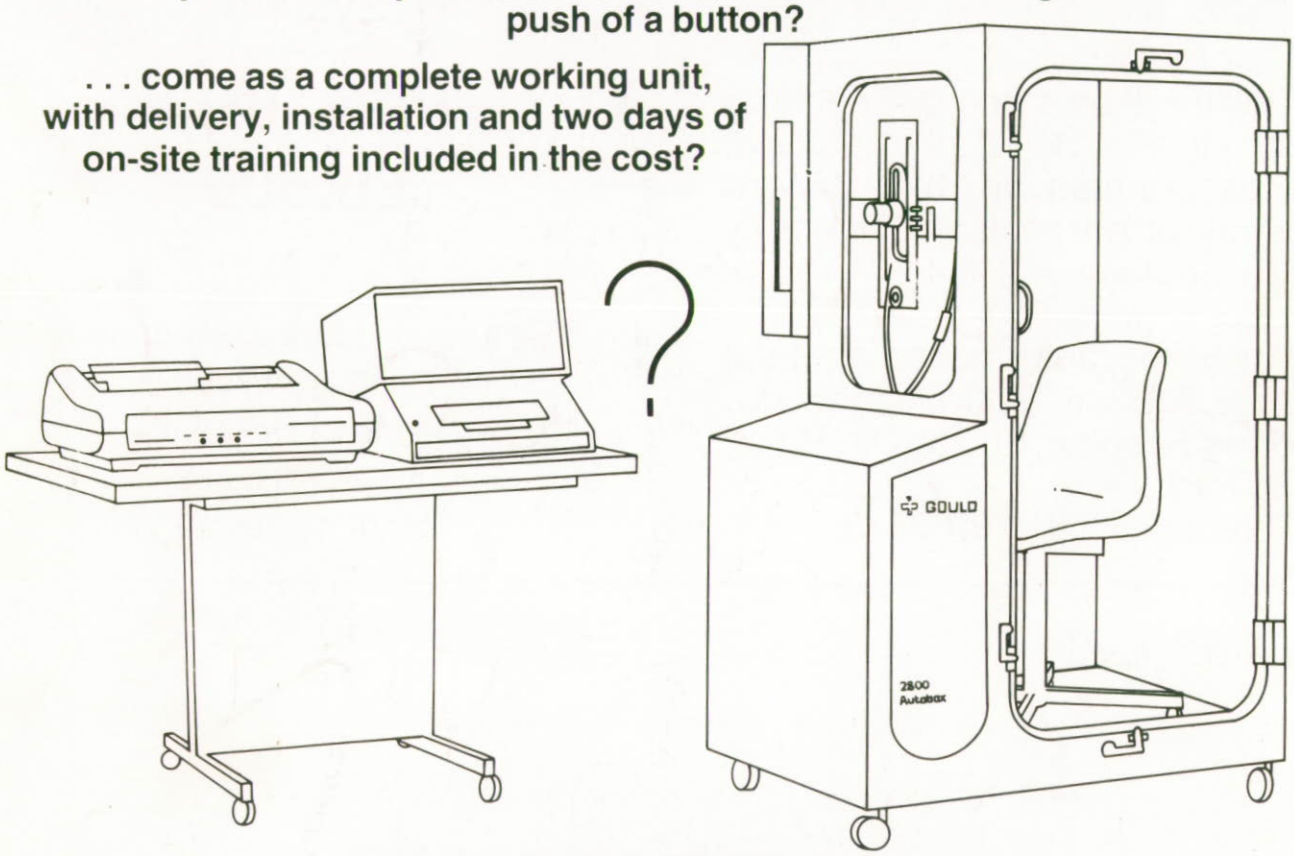

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