ASSOCIATION OF RESPIRATORY TECHNICIANS AND PHYSIOLOGISTS

ISSUE NO 20 OCTOBER 1983

BREATH

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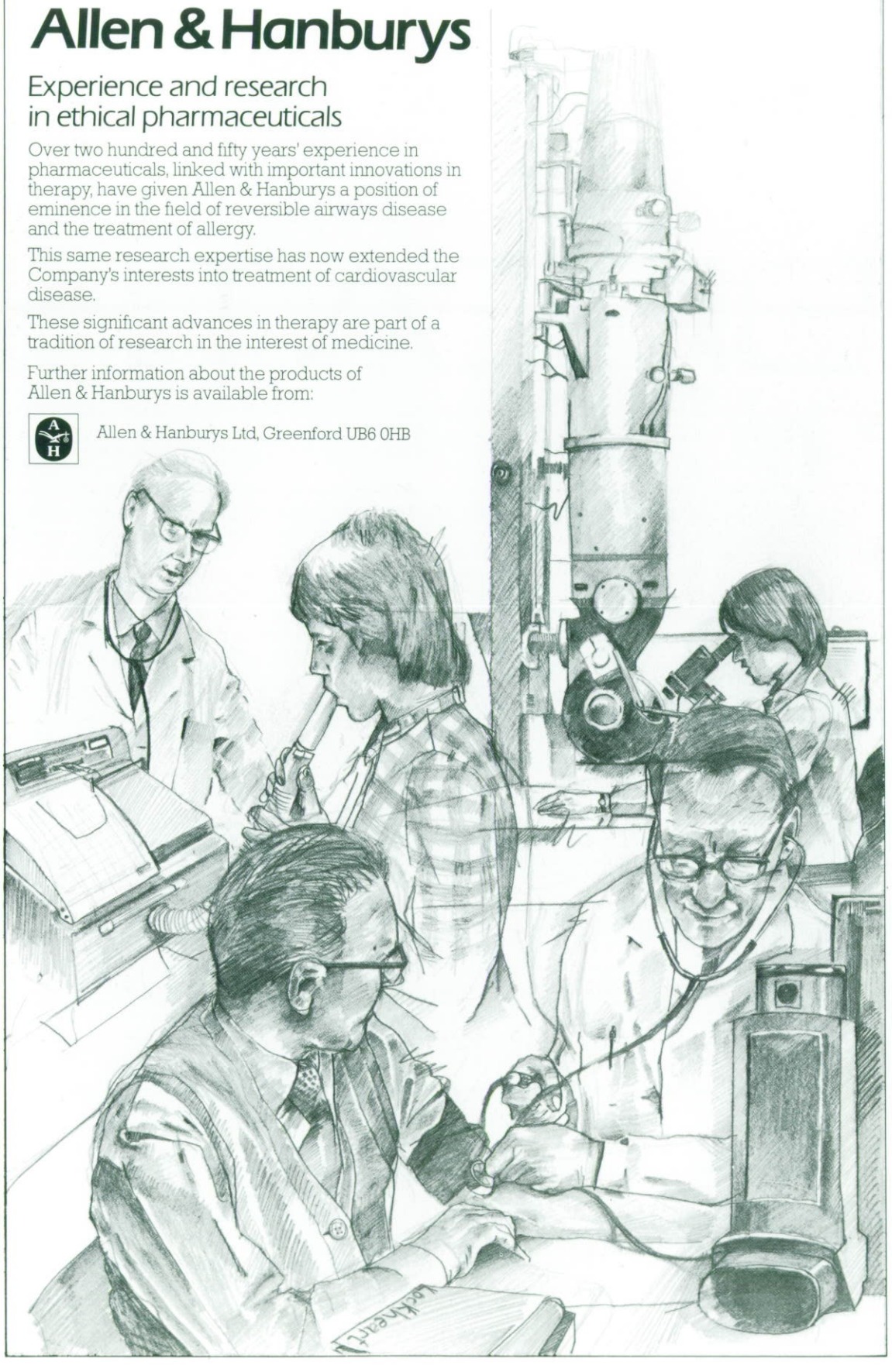

ARTP — PAST AND FUTURE

This month the autumn meeting of our Association takes place at King's College Hospital, an establishment in South London where, old-timers may recall, the foundation meeting of the Association was held nearly eight years ago. At that time there were powerful reasons why this had to happen; the changes envisaged in the Zuckerman report made it essential for all technicians to belong to some professional organisation, though much of the revolutionary fervour of that time was in fact directed against the Zuckerman proposals rather than in their favour. But now that some of the fervour has worn off (perhaps inevitably), this might be the moment to take stock of what has been achieved, where we now stand and what the future might hold.

The Association first and foremost is a professional body, representing a skilled body of technical staff who are undertaking an essential public service. Its constitution, as constitutions will, make all the right noises about promoting advances firstly, in the care of patients with respiratory disease and secondly, in the education and training of ARTP members. These two objectives in many ways are complementary and plainly the first objective cannot be fulfilled without dedicated attention to the second. The Association's scientific meetings and workshops provide a valuable forum where those with common interests can meet. Such activities being voluntary, reach a limited audience (for financial and other reasons) and thus go only part of the way towards fulfilling the above objectives.

It was clear at the outset that the establishment of Education and Training Programmes would be one of the main tasks of the Association and so it has proved. Substantial changes have taken place in this area; supervision of the National Certificate system of technical education has been taken over by the new Techician Education Council (TEC) and students will now take the O TEC and H TEC examinations. Unfortunately there are barely a dozen colleges in the country where the TEC approved syllabus can be taken, hardly any of them being in the South where much of the demand is. Although it is nice to see the North being favoured for a change, the imbalance is certainly not going to be rectified in the short term and we may well have to explore some of the alternative educational techniques such as the 'distance learning' scheme recently outlined by D. J. Keller[1].

Even larger problems loom up when we come to consider the question of In-Service Training. The Association gives this equal standing to the college-based courses but this view is unfortunately not shared by the TEC, who have not so far agreed to accept validation of in-service training courses on the grounds that they are not 'educational'. We commented on such attitudes in these columns[2] some two years ago and we can only repeat what was said then: full education must mean the integration of theoretical knowledge with practical experience. No professional body within the health service (and doctors certainly observe this principle) could afford to let its members loose upon the public without such an educational programme.

Undeterred by such obstacles, the Association has pressed ahead. A comprehensive training manual has been produced and the reasons for introducing this have been spelled out in detail by Sally Gough[3]. The training programme itself can only be finally acceptable when a formal assessment of the performance of the trainees can be established; in other words a practical examination is required. Fortunately a good deal of work on this has already been done in the Trent Regional Health Authority[4]. The organisation of such an examination and the selection of assessors and of the assessment centres would be a substantial task and one which the Association has been reluctant to undertake, though it may yet have to do so. More work for the future!

The physiological measurement technician has, we all recognise, a special role, often providing the first contact with the patient in a complex and even frightening environment. The technician must gain the patient's confidence, explain the nature and necessity of the procedures with clarity and obtain valid and reproducible results in tests requiring maximal efforts or complicated respiratory manoeuvres.

But when all's said and done, even the most saintly devotion will fail to 'promote advances in diagnosis and treatment' (vide Constitution again) unless accurate and reproducible results can be made available. The mysteries of drift, noise, linearity and so on have recently been discussed in *Breath* by A. C. Newton[5] and the rapidity with which some modern instruments pour out printed results can easily dull our appreciation of such errors. The problems are enhanced by the multiplicity of instruments now on the market. The possibility of standardization of lung function tests has been discussed much more fully in the United States[6] than in this country and the arguments for and against have been elaborated by Clausen[7]. Standardization across the country, if it could be achieved, would offer certain benefits. Thus the results for any patient transferring from one laboratory to another would be directly comparable and the advantages in multicentre studies would be considerable; it might however result in a rigid system that would be difficult to change. Clausen[7] suggests an alternative approach based on the premise 'we don't care how you get results, but they must be comparable to the results obtained by a specific reference method'. On this system the different types of spirometer, for example, would remain in use but would conform to a given standard. This approach would be more flexible but difficult to implement. Our meetings and workshops provide an excellent means of making personal contact with instrument manufacturers and we should be able to guide them on the instrument characteristics that we require. A task for the next decade perhaps.

But enough of moralising — continue to supply us with good food and wine at our meetings and all will be well.

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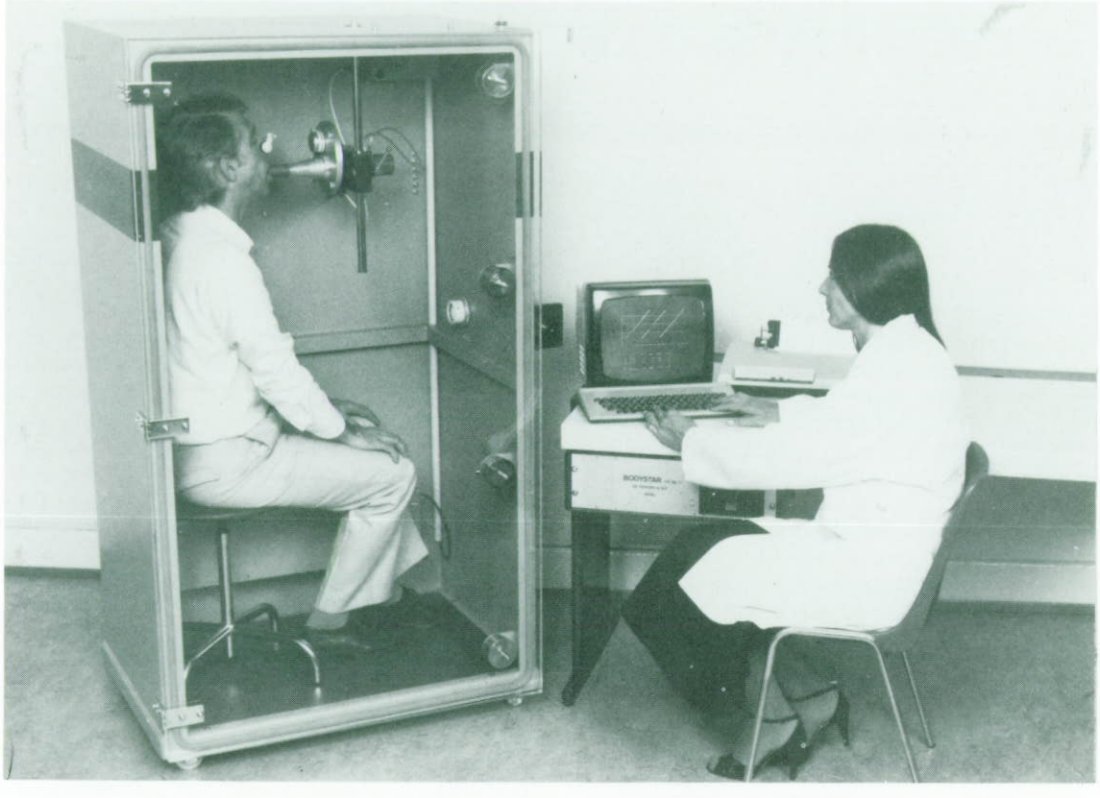

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ASSESSMENT OF RESPIRATION DURING SLEEP

M. C. P. Apps
The London Hospital and The London Chest Hospital

Introduction

Over the last few years interest in respiratory patterns and ventilation in the sleeping patient has increased[1, 2]; this has led to the development of methods for non-invasive monitoring of respiration and sleep.

Abnormalities of respiration have been reported in a large number of disorders and range from the minor where there may be irregularity of respiration with little or no hypoxia, to the serious where the respiratory pattern may be disorganised with long periods of apnoea, severe hypoxia, cardiac arrhythmias, pulmonary hypertension and the development of cor pulmonale. Sleep apnoea syndromes have been described and treatments for them are being developed. It is the purpose of this paper to discuss the various methods that are available for monitoring respiration during sleep, so that sleep disordered breathing may be diagnosed and adequately assessed.

Sleep Apnoea Syndromes (Table 1)

Apnoea is defined as a cessation of airflow, measured at the nose, mouth, or larynx.

Central apnoea. An apnoea is said to be 'central' in nature if the cessation of airflow is accompanied by absence or considerable diminution in the movement of the chest wall and abdomen. This corresponds to failure or diminution of central drive to respiration, and may be part of 'periodic' respiration associated with fluctuating respiratory drive. If the apnoeic episode is very short there is no change in the blood gases but if prolonged there may be severe hypoxia and reflex pulmonary hypertension. For practical purposes only cessation of respiration for longer than 10 seconds is considered as 'Apnoea'.

TABLE 1

Causes of Obstructive Sleep Apnoea

Nose	Deviated nasal septum
	Nasal packing
	Allergic rhinitis
Pharynx	Obesity
	Myxoedema
	Acromegaly
	Micrognathia
	Macroglossia
	Enlarged tonsils and adenoids
	Retropharyngeal masses
Larynx	
	Shy Drager syndrome

Causes of Central Apnoea

Normal periodic respiration
High altitude
Encephalitis
Brain stem disease
Intracranial tumour
Ondine's curse
Myxoedema
Congestive cardiac failure

Obstructive apnoea; there is no airflow at the nose or mouth but there is continuing respiratory effort. One can see paradoxical movement of the chest and abdomen accompanied by increasing respiratory effort until the obstruction is relieved. As the obstructive episode is prolonged so hypoxia develops; straining against an obstructed airway leads to an increase in vagal activity and bradyarrhythmias are very common. The site of obstruction is usually pharyngeal, as the pharyngeal walls collapse inwards at a time of decreased muscle activity. In a few patients with vocal cord paralysis obstruction occurs at the level of the larynx due to inward apposition of the vocal cords.

'Obstructive sleep apnoea syndrome' is defined as the occurrence of more than 30 obstructive episodes, each lasting longer than 10 seconds, during a single night of sleep.

'Mixed apnoeas' show the characteristics of both central and obstructive apnoeas. They start as a central apnoea followed by increasing evidence of respiratory muscle activity with obstruction, until this is relieved and free airflow returns.

Hypoxia and Hypoventilation (Table 2)

In all individuals there is a slight decrease in arterial oxygen saturation and rise in CO_2 tension during sleep, with decrease in the ventilatory response to hypoxia and hypercarbia. Any patient with respiratory disease may already have a degree of hypoxaemia, in which case the nocturnal fall in arterial O_2 saturation may lead to profound hypoxia. Elevation of arterial CO_2 tension also occurs, indicating hypoventilation (which may also be present during waking hours).

TABLE 2

Causes of Nocturnal Hypoxia

Hypoxic lung disease	Obstructive airways disease
	Pulmonary fibrosis
	Kyphoscoliosis
	Cystic fibrosis
	Asthma

Congestive cardiac failure
Diaphragmatic paralysis
Poliomyelitis
Myasthenia gravis
Guillain-Barre syndrome
Sleep apnoea syndromes

Assessment of Sleep

To study respiration during sleep it is necessary to provide conditions conducive to sleep! This may seem obvious but many of the techniques used to monitor respiration make it more difficult for the subject to sleep and any study done on an open ward or in a noisy Intensive Therapy Unit may be fraught with difficulties. Sleep may be assessed subjectively by asking the subject to report on the quality and quantity of sleep in the morning, or the sleep may be

staged quantitatively (Figs. 1 and 2). This requires the recording of at least one channel of the electro-encephalogram (and preferably more), at least one channel of the electro-oculogram (to record eye movements), and the electromyogram, which is usually recorded from submental electrodes. The signals are recorded on paper or tape and studied so that formal staging of sleep can be performed (Fig. 3).

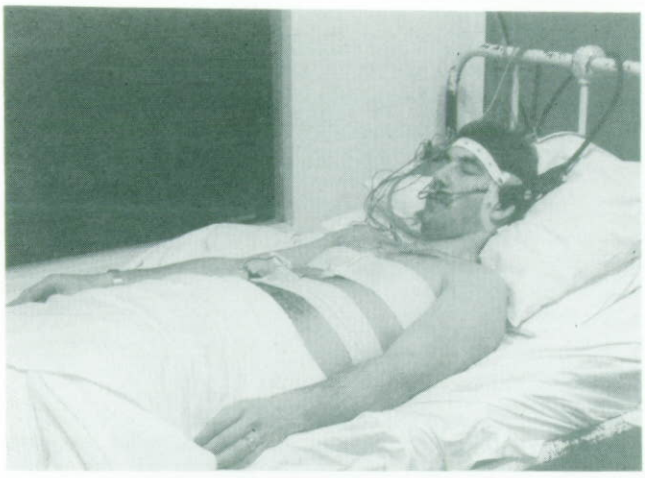

Fig. 1. *Patient undergoing sleep study with 'Respitrace' inductance bands to monitor movement of chest and abdomen, thermistors on nasal cannulae for airflow and oximeter on left ear. The electro-oculogram electrode can be seen on the left cheek.*

Fig. 2. *Investigation room: The patient's room is behind the blackout curtain. Leads from the ear oximeter, 'Respitrace' and other transducers enter through the wall. Data is being monitored on a 16 channel recorder.*

Breathing during Sleep

If a patient is having frequent obstructive or central apnoeic episodes during sleep it is often possible to make the diagnosis by direct observation. In a sleeping patient who is snoring loudly, paradoxical movement of the chest and abdomen may be seen from the end of the bed and central apnoeas may also be diagnosed. Observation is limited by the perseverance of the observer, and does not give quantitative data on arterial oxygenation which is necessary for the planning of treatment.

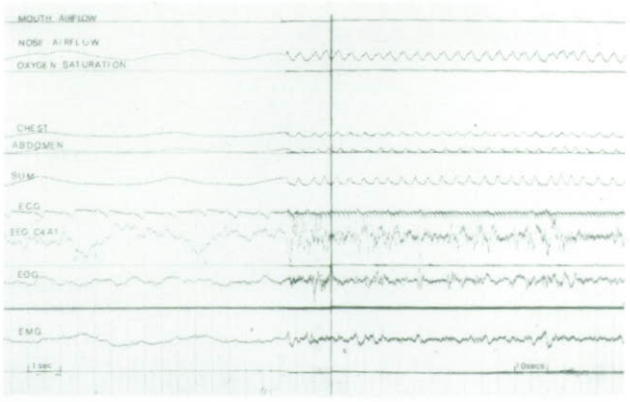

Fig. 3. *Continuous recording obtained from a sleeping subject. Signals from above downwards indicate: Mouth and nose airflow from thermistors; oxygen saturation from ear oximeter; chest and abdominal movement and their sum from 'Respitrace'; electrocardiogram (ECG); electroencephalogram (EEG); electro-oculogram (EOG); electromyogram (EMG).*

Assessment of Air Flow (Table 3)

Absence of airflow signifies apnoea. Early studies of respiration during sleep measured both flow and volume of inspired and expired air by using a noseclip and mouthpiece or a tightly fitting mask. These are very effective but few patients can sleep whilst wearing them. Today more indirect methods tend to be used to sense airflow and accuracy of volume is sacrificed for the ease and comfort of the wearer. By monitoring the temperature of the air in the anterior nares and just outside the mouth using thermistors or thermocouples, it is possible to sense airflow. With this method it is necessary to record from both the nose and mouth since some patients are mouth breathers, some are nose breathers and others alternate between the two. One difficulty of the nasal thermistors is that if they become lodged any distance up the nose the environment is continuously warm so that there is no change in temperature with respiration. These methods are qualitative rather than quantitative, allowing us to diagnose apnoea but telling us nothing about whether it is central or obstructive, or about its effect on oxygenation. A small microphone attached to the neck is a cheap, comfortable method by which the breath sounds can be recorded[3]. A small probe in the anterior nares, attached to a rapidly responding CO_2 analyser or mass spectrometer, has also been used successfully to sense airflow and to diagnose apnoea.

TABLE 3

Assessment of Airflow

Thermistors and thermocouples
Laryngeal microphone
End expired CO_2 tension
Face-mask, mouthpiece and noseclip

Assessment of Chest Movement (Table 4)

Monitoring of airflow simply allows apnoea to be diagnosed. If there is no chest movement however then central apnoea is present. If on the other hand there is increasing paradoxical movement of the chest then the apnoea is obstructive in nature. Some patients have marked periodicity in their respiratory pattern, with hypoventilation but without any apnoea. The recording of chest and abdominal movement allows these states to be assessed.

TABLE 4

Assessment of Chest Movement

Observation
Strain gauges
Impedance method
Inductance coils
Magnetometers

Movement of the chest and abdomen can be measured using mercury filled strain gauges which are easy to make but can be temperamental. Movement can also be assessed by measuring the impedance between electrodes on the front and the back of the chest. These two methods are however only semiquantitative.

For more accurate assessment of movement, and hence of ventilation volume, the equipment necessary is more complex and expensive. Magnetometers, with one coil producing a magnetic field and a second sensing the field, can accurately measure the distance between the two coils and thus assess ventilation.[4] A recent development is the use of inductance coils[5], which give an output dependant upon the volume of the cylinder inside the coil; this technique is proving increasingly popular and at least in patients with a normal chest can give an estimate of ventilation volume within 5-10% of that obtained by spirometry or pneumotachography.

Assessment of Respiratory Efficiency (Table 5)

No matter what the respiratory rhythm, or the number of apnoeas, it is the resulting hypoxia and hypercarbia that lead to the serious complications of pulmonary hypertension, cor pulmonale and polycythaemia. It is necessary therefore to have some method of assessing pO_2 and pCO_2 in the sleeping patient. If an arterial line is in place, blood gases can be regularly assessed. For many years this was the only accurate method of studying the sleeping patient but the arterial line carries certain risks, may be difficult to sample without waking the patient and the sampling frequency is limited.

TABLE 5

Assessment of Respiratory Efficiency

Arterial O_2 and CO_2 tensions
Transcutaneous O_2 and CO_2 electrodes
Analysis of expired gases
Ear oximeter for arterial O_2 saturation

Ear oximeter. For the last five years, use of the Hewlett Packard ear oximeter for continuous monitoring of ear oxygen saturation has been the preferred method in many centres[6]. With this method light is passed through the ear and the oxygen saturation assessed from light absorption at a range of frequencies. The probe is comfortable and fits easily onto the ear, so that it does not disturb sleep. The method has certain limitations; readings become inaccurate if the ear is not sufficiently arerialized or if O_2 saturation falls below 60% (though tables of correction factors are now available) and with excessive movement the oximeter may become detached. The method cannot discriminate between oxyhaemoglobin and

carboxyhaemoglobin and to obtain pO_2 from the O_2 saturation requires knowledge of pH and pCO_2 which would have to be measured in other ways.

Transcutaneous blood gas measurement. Accurate transcutaneous pO_2 and pCO_2 electrodes have recently become available[7]. These incorporate a heater in order to arterialize the circulation in the underlying skin and have to be moved in order to avoid minor burns. Later models operate at a lower temperature and thus need moving less often. The results obtained correlate with direct blood gas measurements.

Assessment of Respiration and Sleep

In any study in which indices of respiration and sleep are being recorded, a major problem is posed by the storage and analysis of the large volume of data produced. EEG is conventionally recorded onto paper; if possible all other data should be recorded on the same trace, and selected data recorded onto tape for further analysis. No single department of chest medicine or electrophysiology tends to have all the equipment and expertise necessary for recording or analysing this data, so close interdepartmental cooperation is essential for obtaining satisfactory results. The studies are carried out at night so that the availability of staff who can understand and operate the equipment is often a problem. If the study is carried out on a normal ward, this may be noisy and the patient may not sleep; if it is carried out in a quiet EEG department then nursing supervision of the patient is necessary and it may be difficult to obtain staff or funding for this. Unless a reasonable number of studies are being performed it is difficult to develop standard methods of performance, and with the large number of separate types of data to be obtained, equipment failures may be unavoidable unless only a limited number of experienced staff are involved.

The Need for Sleep Studies

It is possible by simple history-taking and observation of the sleeping patient, with the judicious measurement of early morning blood gases, to identify most severe cases of sleep apnoea or of nocturnal hypoventilation. For fuller assessment formal sleep studies are needed. At present there are few units in the United Kingdom which are able to undertake full assessments of respiration during sleep, but if obstructive apnoea or severe nocturnal hypoventilation are being considered as possible diagnoses, then patients should be referred for assessment in such units.

There is an increasing range of therapeutic options available in these patients, ranging from specialised surgery to the pharynx or the use of nocturnal continuous raised airway pressure delivered through nasal cannulae for patients with obstructive apnoea; diaphragm pacing and a variety of drugs can be used in patients with hypoventilation. Full assessment of respiration during sleep allows diagnosis of the particular sleep associated respiratory disorder to be made and treatment can then be tailored to the patient and to the condition.

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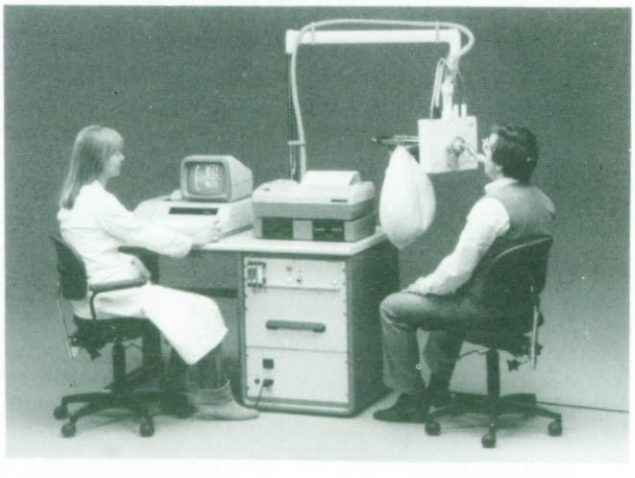

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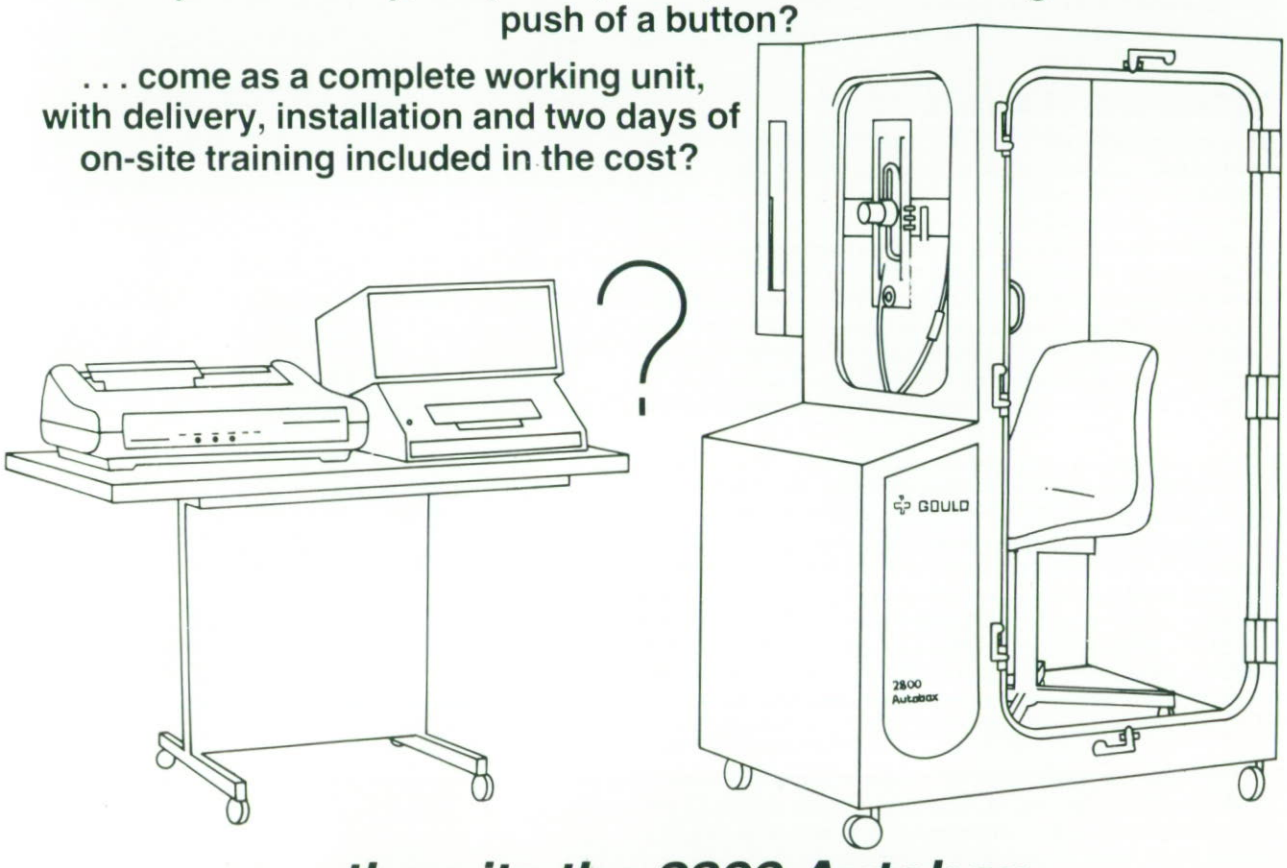

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BRONCHIAL ASTHMA: Clinical features and management

Robin Rudd
The London Chest Hospital

Asthma was recognised by the ancient Greeks and was thought to be a disorder caused by blocking of the air passages to the lungs by an excess of phlegm, one of the principal humours of the body. Hippocrates ascribed the disorder to a combination of constitutional and environmental factors, a view very much in accordance with current ideas. The importance of genetic factors is suggested by the variable incidences of asthma in different populations and the increased incidence of asthma seen in relatives of patients with the disease. Many environmental factors influence asthma, including climate, pollen count, house dust mite levels and the prevalence of upper respiratory infections.

Pathogenesis of Asthma

Asthma is currently defined in functional terms as widespread airways obstruction reversible over short periods of time, either spontaneously or as a result of treatment. Anatomically the airways obstruction is caused by contraction of bronchial smooth muscle, oedema and inflammation of the bronchial wall and plugging of the airways by intraluminal secretion and debris. Physiologically, the airways are abnormally sensitive to a variety of stimuli, the condition known as bronchial hyper-reactivity, which implies that environmental stimuli which do not cause symptoms in the normal person can provoke bronchoconstriction in an asthmatic patient. Cooling and drying of the bronchi by hyperventilation at rest or during exercise and exposure to irritant particles such as tobacco smoke are common examples of such stimuli. In the lung function laboratory bronchial hyper-reactivity may be noted when forced expiration produces coughing or decreasing expiratory flow with repeated measurements. Hyper-reactivity can be demonstrated by finding increased sensitivity to inhaled histamine; asthmatics, but not normal subjects, develop a fall in forced expiratory volume in one second (FEV_1) of 20% or more after inhaling histamine in concentrations of 8 mg/ml or less[1].

The bronchial hyper-reactivity seen in asthma is produced by alterations in the tone of bronchial smooth muscle. Tone is regulated by the autonomic nervous system, the cholinergic and alpha-adrenergic systems producing bronchoconstriction and the beta-adrenergic system causing bronchodilation. In asthma there is evidence for impaired beta-responsiveness and increased responsiveness to cholinergic and alpha-adrenergic stimuli. As well as affecting bronchial smooth muscle directly the autonomic nervous system can influence the ease with which histamine and other chemical mediators are released from mast cells. This can occur as a result of the interaction of an antigen with IgE antibody on the surface of the mast cell and as a response to other stimuli such as exercise. These chemical mediators stimulate mucus secretion and bronchial smooth muscle contraction and increase vascular permeability, leading to oedema and inflammation of the bronchial wall.

Physiological Changes in the Lung in Asthma

During an attack of asthma there is widespread narrowing of airways causing increased resistance to airflow. Consequently, the rate at which gas can be expelled from the lungs is reduced and this is reflected in measurements of expiratory flow such as FEV_1 and the peak expiratory flow rate (PEFR). The chest is held in an inspiratory position in which the total lung capacity (TLC) is increased and this posture, by increasing traction exerted on the airways, tends to oppose the airways narrowing. The residual volume (RV) increases, probably because of a greater tendency for the already narrowed airways to close during expiration. The functional residual capacity (FRC) also increases because inspiration begins before the previous prolonged expiration is complete. The vital capacity (VC) is usually reduced because RV increases to a greater extent than TLC (VC = TLC – RV) (fig. 1).

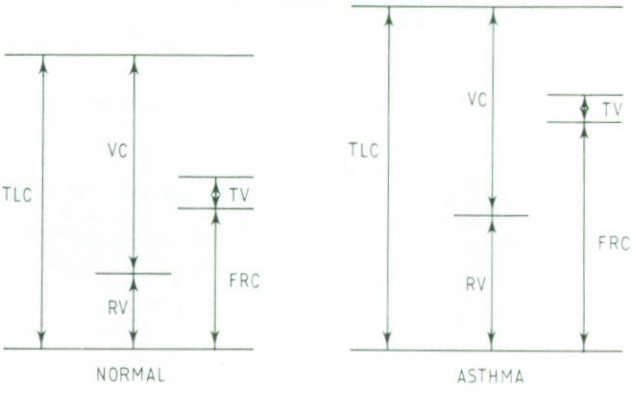


Fig. 1. *Changes in static lung volumes in asthma. TV: Tidal volume. TLC: Total lung capacity. VC: Vital capacity. FRC: Functional residual capacity. RV: Residual volume. Note the enlargement of RV and FRC in asthma.*

In asthma, gas exchange is impaired because of mismatching of ventilation and perfusion. In the normal lung most alveoli are well ventilated and well perfused but in asthma a considerable proportion of the alveoli are underventilated because of disturbances in the distribution of inspired gas, caused by airway narrowing and blockage. Perfusion of underventilated areas is reduced to some extent by vasoconstriction in response to local hypoxia, but not sufficiently to match the degree of underventilation (fig. 2)[2]. Overall hyperventilation allows increased elimination of carbon dioxide in well ventilated areas to compensate for decreased elimination in poorly ventilated areas and arterial carbon dioxide tension is usually normal or low. In contrast, because of the sigmoid shape of the oyxhaemoglobin dissociation curve, well ventilated areas cannot increase the oxygen content of the blood sufficiently to compensate for the low oxygen content of the blood passing through underventilated areas; the result is arterial hypoxaemia.

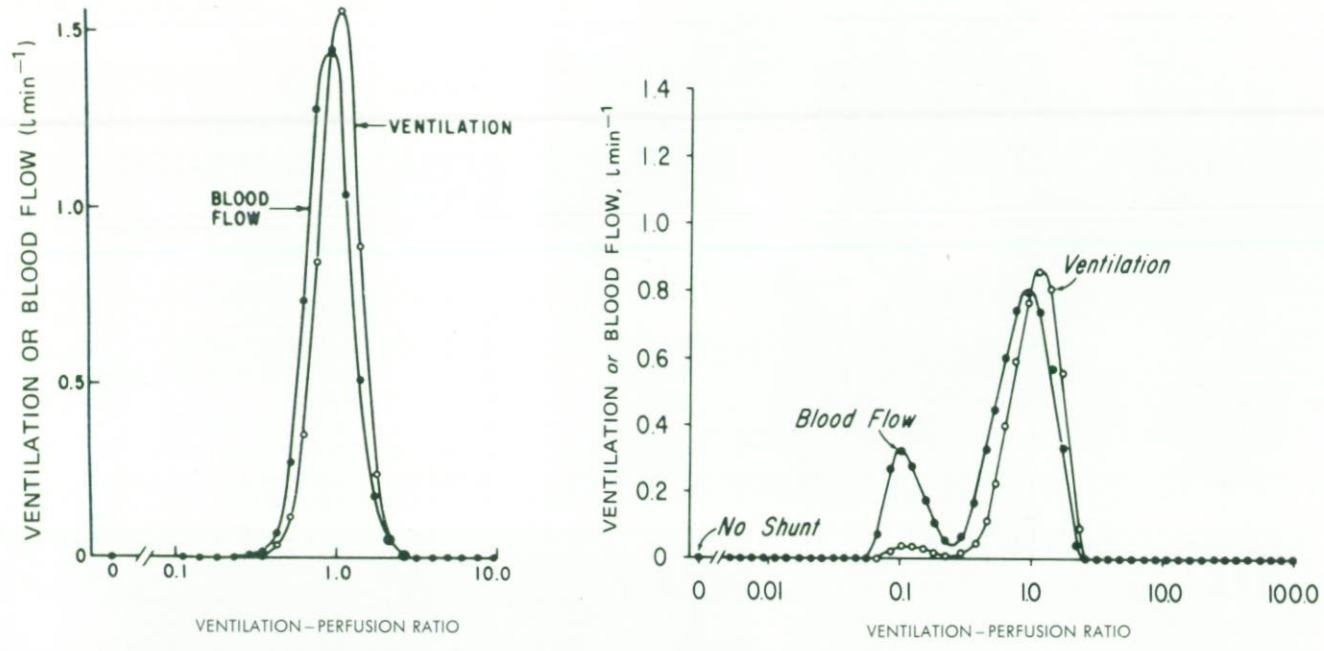


Fig. 2. *Ventilation/perfusion relationships in (LEFT) a normal subject and (RIGHT) an asthmatic. In asthma the proportion of units with a low ventilation/perfusion ratio is increased (West[2]). Reproduced by courtesy of William Heinemann Medical Books Ltd.*

Clinical Types of Asthma

Patients with asthma suffer from episodic breathlessness with wheezing. There is wide spectrum of severity, frequency and persistence of symptoms and a variety of initiating factors. Some patients have occasional mild wheezing or may notice a cough or chest tightness while others have sudden life threatening attacks or are chronically disabled by severe breathlessness.

Some clinical types of asthma are identified on the basis of causative factors and others on the basis of a clinical description. Because of this a patient may be included in more than one category: for example, a patient may have asthma primarily caused by allergic mechanisms but may also have attacks provoked by exercise, bronchial infection or emotion.

Extrinsic Asthma

Asthma is described as 'extrinsic' if there is a history of external factors provoking symptoms and this form is further subdivided into atopic and non-atopic forms. The *atopic state* indicates the capacity to develop an immediate allergic reaction to common environmental substances and in clinical practice is usually demonstrated by *skin tests* in which a fine needle is passed into the skin through a drop of liquid containing the suspected substance; a positive response consists of redness and swelling developing at the site within fifteen minutes. If an asthmatic has one or more positive skin tests to common environmental allergens he is said to have *extrinsic atopic asthma* and allergic mechanisms are probably at least partly responsible for the asthma. (It is important however, to note that a positive skin test to a particular substance does not necessarily mean that allergy to that substance is important in causing asthma.) Patients with extrinsic atopic asthma usually develop the disease in childhood and often have eczema and hay-fever as well. Symptoms are often seasonal increasing for example, in late spring and early summer when allergy to grass pollen is involved. There may be a remission around puberty but asthma may return in the third and fourth decade.

If an external agent provokes attacks but the patient does not have any positive skin tests the condition is called *extrinsic non-atopic asthma.* This type is less common and usually develops in adult life; some types of occupational asthma fall into this category.

Intrinsic asthma

Symptoms normally begin in middle life explaining the alternative term 'late onset asthma'. There is a tendency for symptoms to persist with fluctuations in severity rather than for acute attacks and remisssions.

In most patients with asthma transient bronchodilation occurs during strenuous exercise, followed by bronchoconstriction about a minute after stopping (fig. 3).

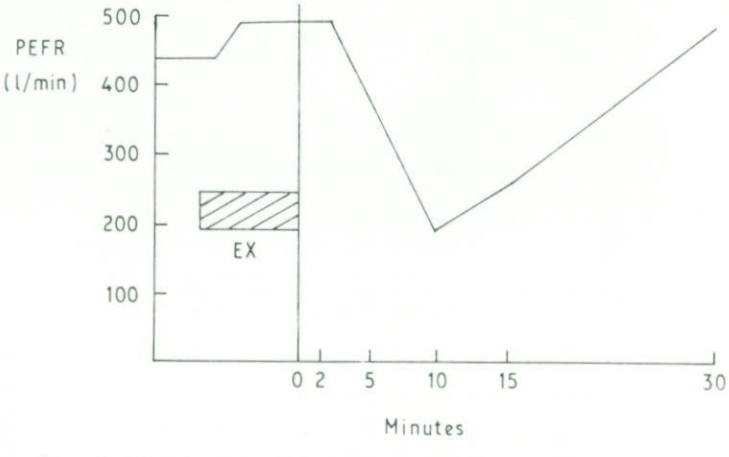
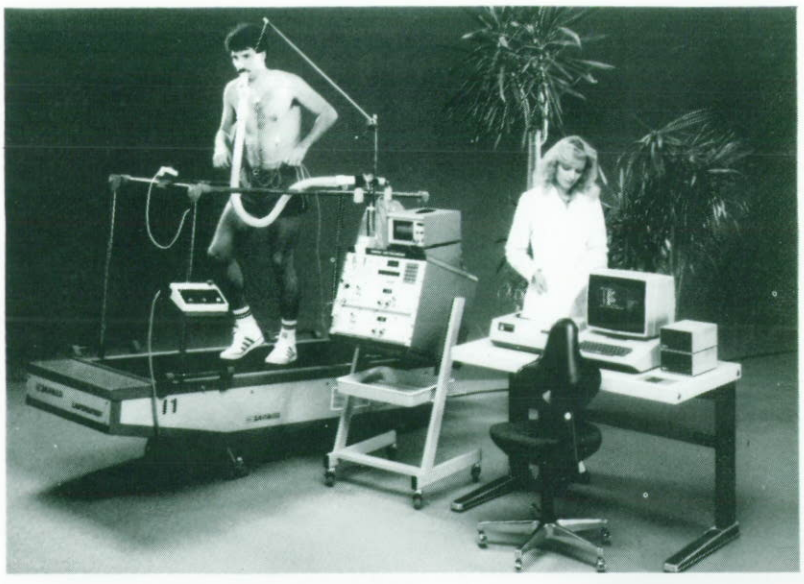


Fig. 3. *Exercise induced asthma: a substantial fall in PEFR is observed after the cessation of exercise (Ex).*



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When this response is an important cause of symptoms the patient may be said to have *exercise induced asthma*. This is more common in children and young adults, perhaps because older patients tend not to engage in vigorous exercise. This post-exertional wheezing must be distinguished from the simple breathlessness during exertion which occurs with most pulmonary and cardiac causes of dyspnoea. Exercise-induced asthma can be prevented by inhalation of salbutamol, other bronchodilator or disodium cromoglycate (DSCG or 'Intal') before exercise.

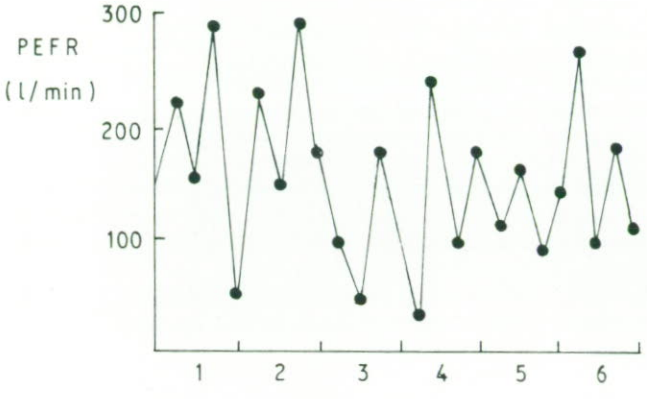

Fig. 4. *Brittle asthma: wide variations in PEFR are seen.*

In *chronic asthma* airflow obstruction varies with time in a number of ways and recognition of these patterns is important in assessing the response to treatment. The 'brittle' asthmatic has severe symptoms with rapid onset and remission (fig. 4). Attacks respond to bronchodilators but may not be prevented by maintenance therapy with bronchodilators, DSCG or corticosteroids. These patients are at an increased risk of life threatening asthma[4]. In the 'morning dipper' the diurnal variation of airway narrowing seen in all asthmatics is greatly exaggerated (fig. 5). The patient may be woken by severe wheezing in the early hours of the morning, which is not usually prevented by long acting bronchodilators taken at bedtime. Some patients thought to have 'irreversible' airways obstruction may gradually improve with a long course of high dose prednisolone, thereby revealing the presence of chronic asthma (fig. 6).

Diagnosis of Asthma

When the patient complains of intermittent breathlessness and is found to have wheezes on auscultation with reduced PEFR or FEV_1 increasing after bronchodilator inhalation, the diagnosis of asthma is easy. Often the history is less typical and the complaint may be of cough, chest tightness, poor exercise tolerance or just lack of energy. Because airflow obstruction is intermittent the patient may have no abnormality in physical signs or lung function at the time of consulting the doctor. It is then that other methods of diagnosis are of value.

Home monitoring of the PEFR is useful in confirming a diagnosis of asthma[5] and it has been shown that patients can reliably keep their own PEFR record[6]. The mini-Wright peak flow meter is suitable for home use because it is easily portable and cheap. The patient should be asked to record the PEFR at least three times daily for several days preferably on waking, at 4 pm and at bedtime. This

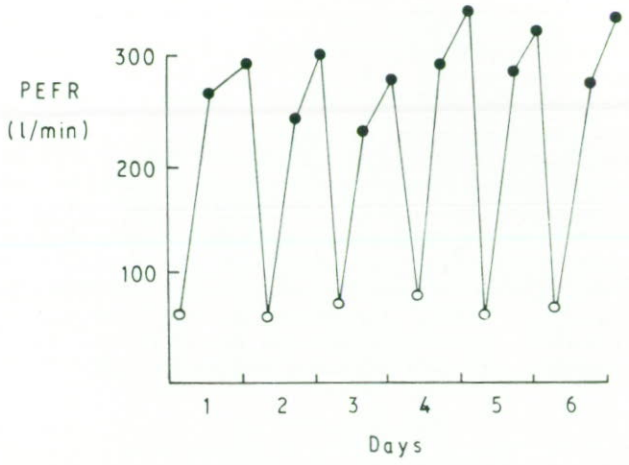

Fig. 5. *'Morning dips'. Note profound fall in PEFR on waking (open circles).*

is because the highest value usually occurs at about 4 pm and the lowest at one of the other two times. There is slight variation with time in the PEFR in most normal subjects but in asthmatic subjects the variation is greatly exaggerated, often leading to nocturnal and early morning symptoms; in asthmatics the amplitude of the variation in PEFR may exceed 20% of the mean value[7]. If there is a suspicion that symptoms may be related to exposure to allergens or irritants at work serial recording of the PEFR at work and at home may be extremely valuable in helping to confirm or refute the diagnosis of occupational asthma[8].The period of recording may need to include a period away from work as improvement after cessation of exposure may be slow.

Challenge tests

If home monitoring of the PEFR is not practical a challenge test can be performed to provoke bronchospasm. The two most commonly used procedures are exercise testing and histamine challenge. For *exercise testing* the subject is asked to run on a treadmill or pedal on a cycle ergometer at a work load that produces a heart rate of about 160 beats per minute; this is maintained for six minutes or until exhaustion. The PEFR or FEV_1 is recorded before exercise and at intervals of 2, 5, 10, 15 and 30 minutes after. A fall of 20% at any time compared with the base line value is significant.

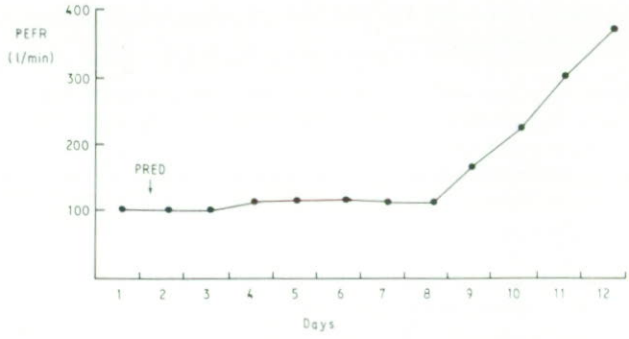

Fig. 6. *Reversal of chronic asthma with prolonged steroid treatment. PRED: start of treatment with prednisolone 40 mg/day.*

In the *histamine challenge test* the patient first inhales saline, followed by increasing concentrations of histamine starting at 0.03 mg/ml and doubling in stages to a maximum of 8 mg/ml. The PEFR or FEV_1 is recorded before and at intervals of 30, 90 and 180 seconds after each dose. If the PEFR or FEV_1 falls by 20% or more compared with the post-saline level inhalations are stopped. The concentration of histamine producing a 20% fall in FEV_1 (PC_{20}) is determined by linear interpolation on the log dose response curve (fig. 7). In normal subjects the PC_{20} is more than 8 mg/ml while in asthmatic subjects the value is nearly always less than 8 and often less than 0.25 mg/ml. Histamine induced asthma can be inhibited by pre-treatment with bronchodilators but not by DSCG.

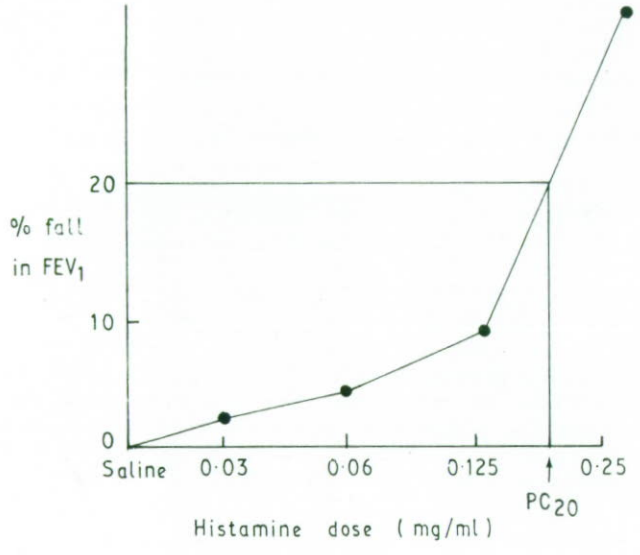

Fig. 7. *Determining the dose of inhaled histamine (PC_{20}) required to produce a 20% reduction in FEV.*

Treatment of Asthma

The *bronchodilators* are the most important group of drugs used to treat asthma. Their principal effect is thought to be the reversal of bronchial smooth muscle spasm but they also inhibit allergen induced mediator release. Selective beta-adrenergic agonists are the most useful agents examples being salbutamol (Ventolin) and terbutaline (Bricanyl). The anti-cholinergic agent ipratropium bromide (Atrovent) and the theophyllines produce bronchodilation by different pathways.

Beta-agonists and ipratropium can be administered by inhalation from a metered dose pressurised aerosol inhaler or from a nebuliser. Inhalation allows a therapeutic effect to be achieved without producing high enough blood levels to cause unwanted systemic affects though this can occur if high doses are inhaled causing, in the case of beta-agonists, tachycardia, tremor and eventually cardiac dysrhythmias. Patients who have difficulty coordinating inhalation with actuation of the aerosol, can use spacing devices such as the Bricanyl 'Spacer' where coordination is not essential or breath-actuated inhalers such as the Ventolin 'Rotahaler'.

Patients with occasional symptoms are usually prescribed inhaled bronchodilators to use as required. If symptoms occur on most days the patient may be advised to use the bronchodilator regularly whether or not symptoms are present. If this is not sufficient to control symptoms a prophylactic inhaled drug, such as the cortiscosteroid beclomethasone (Becotide) or occasionally DSCG is added. This should be used on a regular basis, usually four times daily to prevent attacks occurring. DSCG is chosen for children, in whom allergic mechanisms nearly always play some part and in adults with a clear history of an allergic provoking factor for their asthma. If symptoms are still not controlled a higher dose of inhaled corticosteroid can be used and if necessary prednisolone by mouth.

In severe acute asthma treatment is begun with high doses of inhaled beta-agonists, usually administered by nebuliser together with intravenous hydrocortisone. Sometimes an intravenous infusion of aminophylline is administered but this should be reserved for patients who do not respond to inhaled bronchodilators because of its greater potential for toxicity. Oxygen is administered to correct hypoxaemia and intravenous fluids may be needed if under-hydration occurs as a result of fluid loss from hyperventilation or inability to drink because of breathlessness. If the attack does not respond to initial treatment and the patient becomes exhausted, mechanical ventilation may be necessary until intensive drug treatment becomes effective in relieving bronchospasm.

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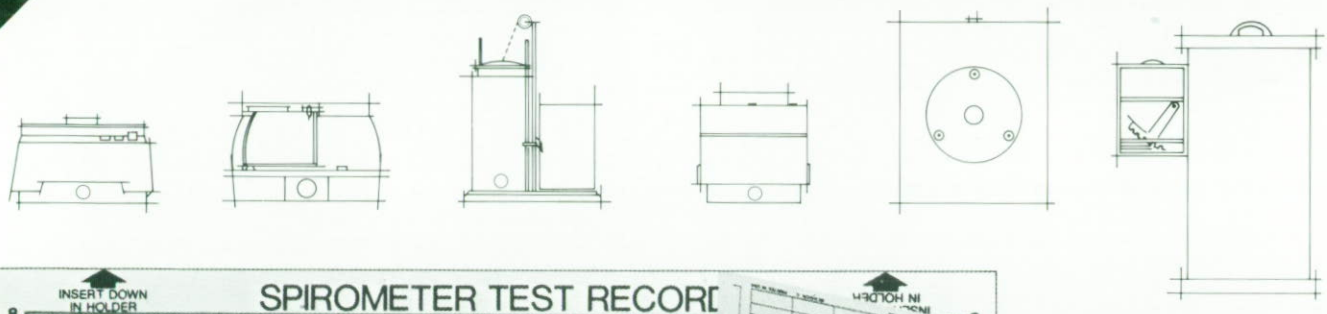

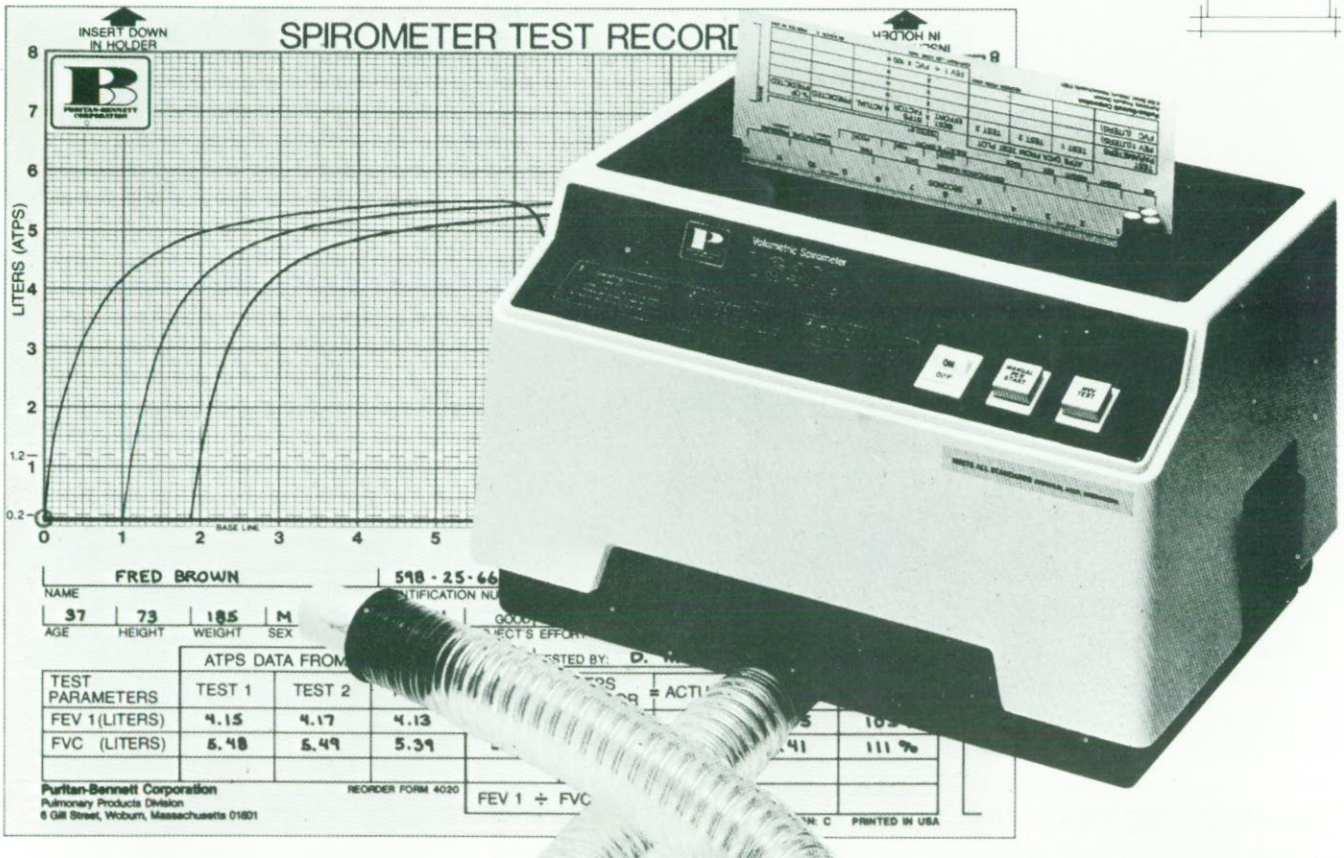

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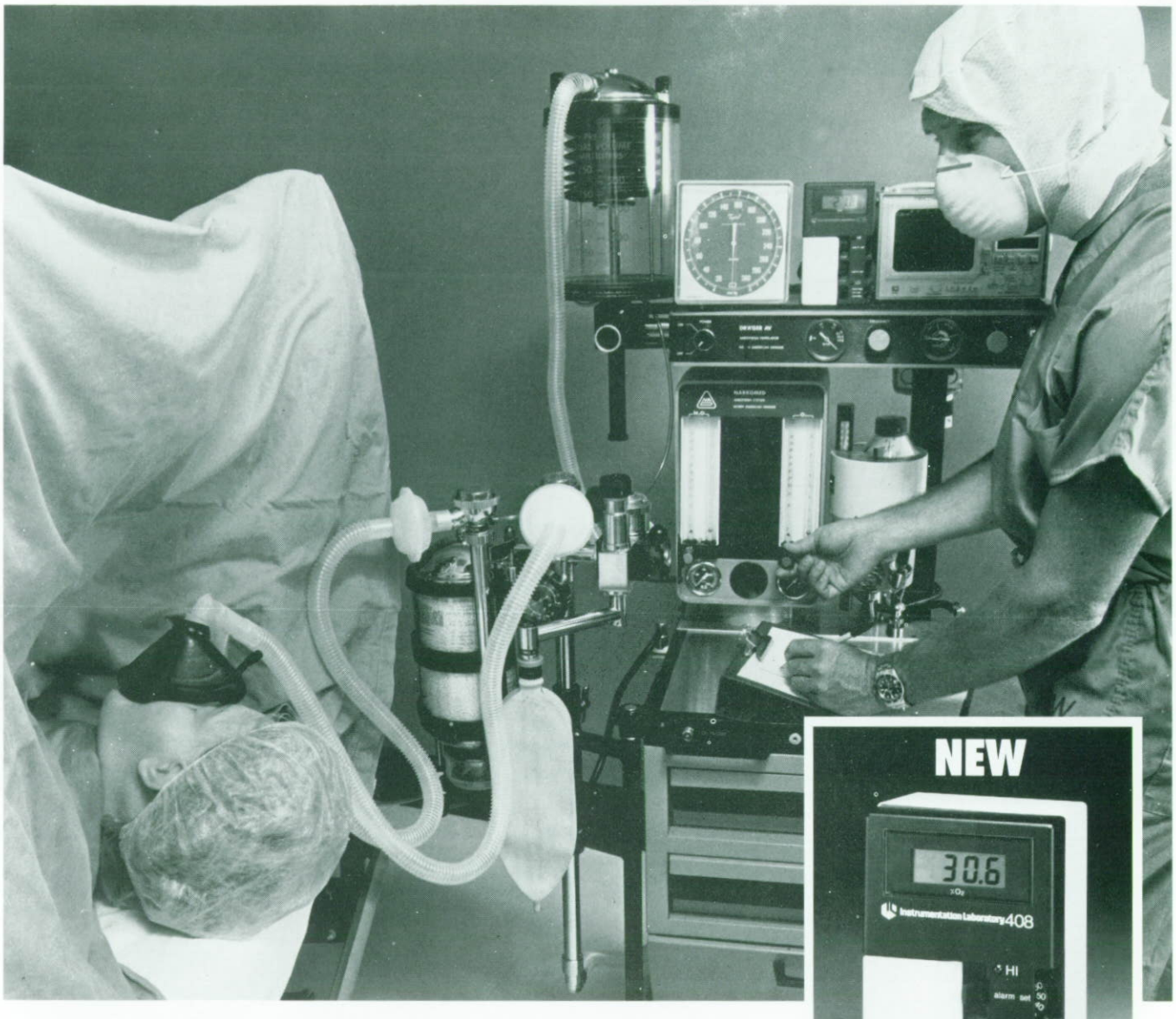

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