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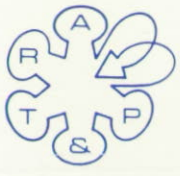

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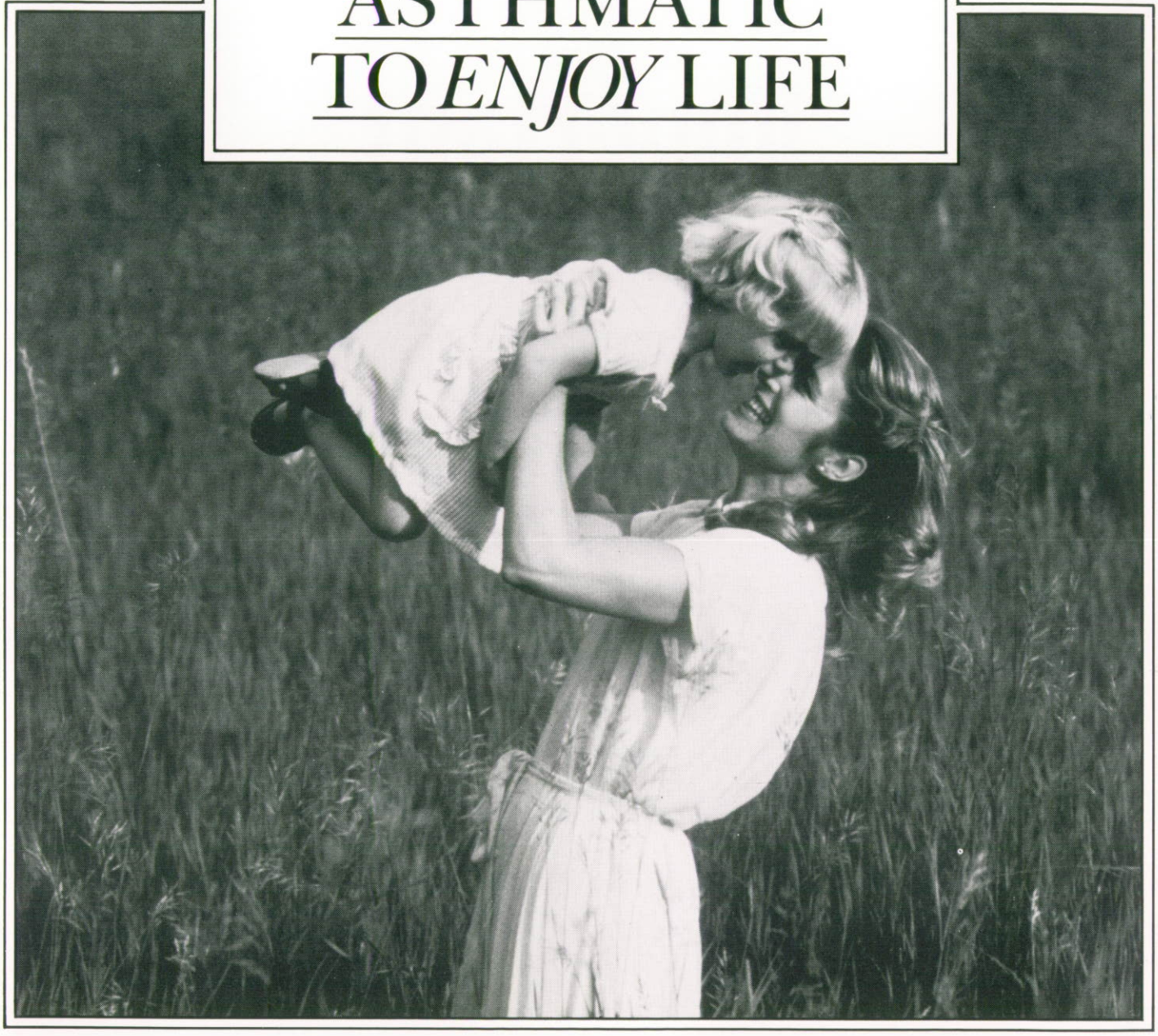

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EDITORIAL

To Standardize or Not to Standardize .....?

A H Kendrick
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Lung function tests are used clinically in a number of ways: a) to identify an abnormality, b) to quantify the severity of the abnormality, c) to locate the main site of disease and d) to follow the course of a disease and/or its treatment. The tests employed, whether simple ones such as spirometry or more complicated ones such as airways resistance, require considerable co-operation from an untrained subject, who must perform unnatural, and sometimes complex manoeuvres so that the measurements may be made.

Within a given laboratory, the performance of the tests, the calculation and presentation of the results and the clinical reporting of the results should, in practice, be consistent. This will, with regular equipment quality control, ensure that serial measurements on a given patient are comparable, thus allowing the results to follow accurately the course of the disease or treatment.

In an ideal world, different laboratories should be able to obtain the same result and a similar clinical interpretation on the same patient. To do this a number of criteria need to be satisfied. Firstly the test procedures need to be identical. Secondly, the specification and characteristics of the apparatus should be the same. Thirdly, the results should be calculated in the same manner and should be expressed in standard units. Fourthly, if the clinical interpretation of the results is to be similar, the reference values and the limits of normality should be the same in each laboratory. On these grounds, there is a clear need for standardization of lung function testing.

It is quite clear from the number of publications over the last 25 years relating to standardization of testing procedures and definitions, that considerable efforts, by many workers, have already been made in this direction (1-10). Of particular importance, is the document from the European Community for Coal and Steel (9). This provides recommendations on the performance of the tests and on reference values. The consequences of employing these recommendations have been critically reviewed by Laszlo (11).

The purpose of this editorial is to discuss some of the arguments for and against the need for standardization, and to highlight some of the problems of current methodology.

Arguments for Standardization

Standardization of lung function testing potentially has three principal benefits. Firstly, uniform methodology would allow clinicians to interpret and compare test results from different units with greater ease. This would obviate the need for the patients to have repeated measurements at a different laboratory, therefore allowing earlier tests to be used as the baseline measurements. In turn, this would reduce the costs of lung function testing.

Secondly, if clear standards were provided for equipment designers and manufacturers, the development of future equipment capabilities would follow the same lines. In addition to the equipment specifications, the role and application of computers, both for data collection and for data analysis would need to be clearly defined.

Thirdly, if all equipment was standardized, the training of technical and other specialist staffs involved in lung function testing would be much simpler.

Arguments against Standardization

There are a number of arguments against standardizing methodology. Firstly, for too many of the tests there is disagreement amongst the experts as to what the standard method should be and, until a consensus of opinion is achieved, standardization will be impossible. As recommendations for standardization often take many years to develop, particularly when several states or countries are involved, standardization of even the simplest routine procedures would be difficult.

Secondly, as it takes so long to produce recommendations, any valid improvements in methodology or equipment will require a considerable length of time (and money!) before being incorporated into standardized procedures. This would lead to the stifling of creativity, because the cost in human and financial terms is too great.

Thirdly, testing procedures that are designed for screening purposes may not always be appropriate when diagnosis of a subtle disease process is required and in research, precision and accuracy of measurement may need to be even greater. This therefore reduces the usefulness of the measurement on a large scale because it is too difficult or complex to be made on large numbers in a reasonable space of time.

Fourthly, in the present economic climate, standardization may involve excessive expense if major modification or even replacement of existing equipment is needed simply to satisfy the standards. These costs, which may seem justifiable to workers within the field, would doubtless be scorned by other measurement disciplines, and by those who administer the finance.

Fifthly, many physicians feel that the choice of methods should be left to them, and not dictated by an outside group of physicians. However, where a number of methods are available, as for the transfer factor, very varying results can be obtained on the same patient, resulting in considerable confusion.

Finally, the problem arises as to who is responsible for ensuring that a laboratory is performing the tests according to the standards. This could ultimately lead to the suggestion that laboratories would need to be licensed to perform the tests and to have their equipment and procedures checked on a regular basis. Many people would find this wholly unacceptable.

Spectrum of Standardization

Standardization of testing procedures means, in essence, that certain aspects of equipment performance and test methodologies would need to be in agreement with written standards. The spectrum of standardization is quite broad. At one end is the formulation of exact specifications encompassing all aspects of the test, so that all equipment, methods and reference values are identical in all laboratories. This would be the simplest approach, and the one which would result in uniform testing procedures in the shortest period of time. This is also the approach that would stifle innovations and improvements (real or perceived), and require a large-scale modernization or replacement

programme of the equipment. (Presumably, all functioning, but non-standard items of equipment would become museum pieces!) This approach is not realistic to a multicentre country-wide standardization programme, unless standardization were to take place over many years, but may be applicable to a small number of laboratories which have a vested interest in co-operating in a standardization programme.

At the other end of the spectrum, is the view that "I don't care how the results are actually obtained, as long as they are comparable to those results obtained from a specific reference method." This view may well satisfy workers who feel they need the freedom to pursue new tests and new types of equipment, but unfortunately does not simplify the task of standardizing lung function tests. The approach itself is difficult to develop, because it initially requires a consensus on what the reference methods should be, and may require a considerable effort and expenditure in comparing every piece of equipment with the accepted reference method.

To some extent, some of the problems which can arise from this view were investigated during the preparation of the United States document (6), in that new work was undertaken to solve some of the problems that had emerged. This work took many years and a large amount of money to complete, and some would question whether the work was really necessary at all.

Current problems

The commonly used lung function tests all have their problems in terms of standardization. Recommendations have been made for spirometry, lung volumes, flow volume curves and transfer factor (9).

The measurement of transfer factor for carbon monoxide appears to be the test which has caused most concern. Most of the problems centre around the calculation involved in the single-breath method, which is the method recommended for routine use. The effects of breath-hold time derivation, alveolar volume calculation and CO back tension were reviewed by Leech et al (12), while a more extensive review of the calculations was presented by Morris and Crapo (13). In the latter study, it was shown that transfer factor could be in error by up to 41%, depending on the methodology and corrections applied. Their suggestions were in all cases, sensible and should be employed routinely.

One particular methodological problem, which has plagued the transfer factor measurement, is the calculation of the alveolar volume. This may be calculated as volume inspired plus residual volume, or by the dilution of an inert, insoluble gas. (In both cases, the volume inspired should be greater than 95% of the total lung capacity (9), otherwise transfer factor will be underestimated.) In normal subjects, and in patients with a restrictive ventilatory defect, the values obtained by either calculation are very close. However, in chronic obstructive lung disease, the values can be rather different, leading to confusion. The matter can be simplified by stating whether the alveolar volume or effective alveolar volume has been calculated. This is certainly necessary, as different physicians in the same unit may request the alveolar volumes to be calculated in two different ways!

The application of various correction factors to the calculation may cause further difficulty. For instance, where lung volumes are quoted as in transfer coefficient (TI/VA), it is often unclear whether VA is at ATPS, STPD or BTPS. It would therefore be helpful if all workers actually stated whether and in what way the volumes have been corrected, eg. TI/VA(BTPS).

Yet another problem involving transfer factor, and many other lung function tests, relates to the units in which they are expressed. We have, fortunately (or unfortunately, depending on your point of view), the SI system of units. Despite being introduced many years ago, many workers still continue to use the traditional units in their lung function reports and in their publications. Thus the units of pressure may be expressed in mm Hg, cm H_2O or kPa — rather confusing. The situation is perpetuated by the fact that many European journals have either not adopted SI units, or require the authors to state both SI and traditional units. Although it is fairly simple to convert from one unit to another, confusion and irritation does nevertheless result. Therefore everyone, including manufacturers of peak flow meters, should be encouraged to start using SI units, otherwise one might as well scrap the system completely.

Once the methodology and the equipment specifications have been sorted out, the remaining major problem will be the reference values. The multitudinous number of reference values available make the decision about "normal" or "abnormal" a difficult one. Many of the reference equations available are of poor quality, or have been derived from highly selected groups of subjects, or else it is unclear how the values have been obtained. In their review of reference equations, the European group (9) had great difficulty in comparing the different versions. There is a clear need for better equations, but until such studies are properly set up, the European summary equations are likely to remain the best available for many years.

Conclusion

Standardization is regarded as important for epidemiology and for research work, though is argued by some that it is less important for clinical practice. It is clear that there will be many problems in achieving national, let alone international standardization of the various lung function tests. There is, in my opinion, a definite need for technicians, physiologists, physicians and manufacturers to concern themselves with this topic. Old attitudes and old practices need to be updated and brought into line with current thinking. The recommendations of the European document need careful consideration and discussion, but should be employed by all, until further improvement in standards becomes available. Workers should never be discouraged from devising new tests and improving current methods and, eventually, if constant review of the recommended standards takes place (every five years, perhaps), new tests and improvements will be incorporated. I hope that those who believe in standardization will encourage others to follow suit, encourage their physicians to perhaps rethink their practices and continue to encourage manufacturers to produce the right type of equipment.

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Survey of Respiratory Methods

The Education Committee of the ARTP is at present circulating a questionnaire on Respiratory Methods now in use in Lung Function Laboratories in this country. Even for relatively simple tests, there are wide inter-laboratory variations in the equipment used, the procedures, the methods of reporting and the reference values with which the results are compared. These differences are particularly important in the standardization of student training.

A number of completed questionnaires have already been returned for which we are most grateful. Not all laboratories have yet been sent the questionnaire but they should receive these in the next few weeks. The questionnaire is necessarily somewhat complex but we look forward to as large a return as possible and hope that the results will be published in due course.

ARTP Education Committee

PULMONARY COMPLICATIONS OF KIDNEY DISEASE

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Summary

This review deals with the diseases of the lung which may be associated with or complicate various types of kidney disease; the effects upon the lung of the various forms of treatment administered to patients with renal disease are also considered. With increasing length of survival, such patients are becoming more likely to suffer not only from the effects of treatment but also from the consequences to the lungs of prolonged metabolic derangement. With improving treatment for chronic renal disease, the pulmonary complications may become more important in determining prognosis.

Introduction

Untreated progressive kidney failure invariably results in an unpleasant death with intractable nausea, vomiting, itching of the skin and drowsiness progressing to coma. Terminal pulmonary oedema is usual, but the clinical picture is dominated by the features of loss of kidney function. This gloomy picture has been changed by the availability of renal replacement therapy, particularly since the mid-1970s. Such treatment, including haemodialysis ("kidney machines", Fig 1), peritoneal dialysis and renal transplantation, has transformed the outlook for these patients. Long-term survival is now common, but this means that many complications of kidney failure and its treatment have been brought to light (1). This paper reviews some of the pulmonary complications seen in patients with kidney disease, including a brief account of diseases and drugs that affect the lungs and the kidneys and of the complications of the various forms of renal replacement therapy.

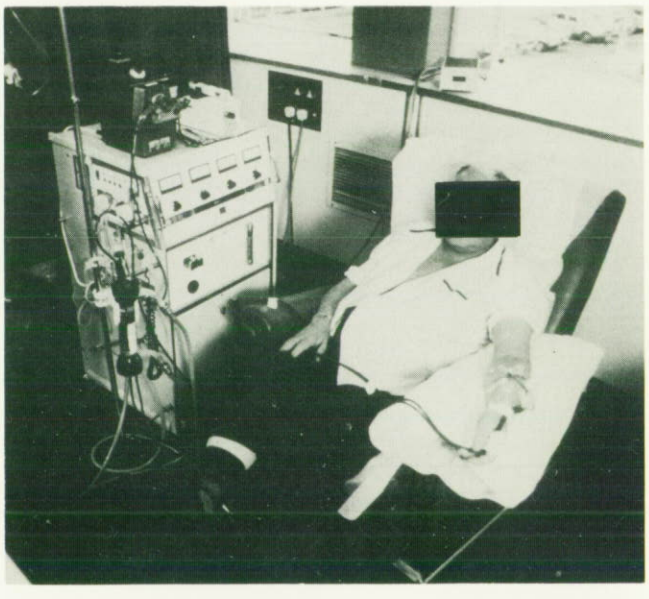

Fig 1. *Patient undergoing treatment with a kidney machine. All this apparatus can be installed in the home, and prolonged survival with a good quality of life is common.*

*British Heart Foundation Research Fellow

Diseases affecting both the lungs and the kidneys

a. Auto-immune diseases

This group of conditions includes systemic lupus erythematosus (SLE), polyarteritis nodosa and its variants, mixed connective tissue disease (MCTD) and progressive systemic sclerosis. All these conditions can cause renal inflammation (glomerulonephritis) which presents with proteinuria (which in SLE and MCTD may be so severe as to cause hypoalbuminaemia and oedema), haematuria and severe hypertension (2). The renal disease can dominate the illness, but all can cause severe lung complications. Polyarteritis nodosa, and particularly its variant, the Churg-Strauss syndrome, can cause asthma. The other three conditions are associated with progressive pulmonary fibrosis (Fig 2), and SLE can also cause severe diaphragm weakness.

b. Granulomatous diseases

Tuberculosis, sarcoidosis and Wegener's granulomatosis may all cause granulomas (discrete collections of chronic inflammatory cells) in the lungs and kidneys. The pulmonary complications of tuberculosis and sarcoidosis are very familiar, but both can affect and even totally destroy the kidneys without any overt lung involvement; this is particularly true of tuberculosis. Wegener's granulomatosis is a progressive condition of unknown cause that was invariably fatal, with destructive upper and lower respiratory tract lesions and glomerulonephritis (Fig 3). It can now often be successfully treated with cyclophosphamide and plasma exchange, which involves filtering off aliquots of the patient's plasma and replacing it with another fluid, usually a solution of albumin.

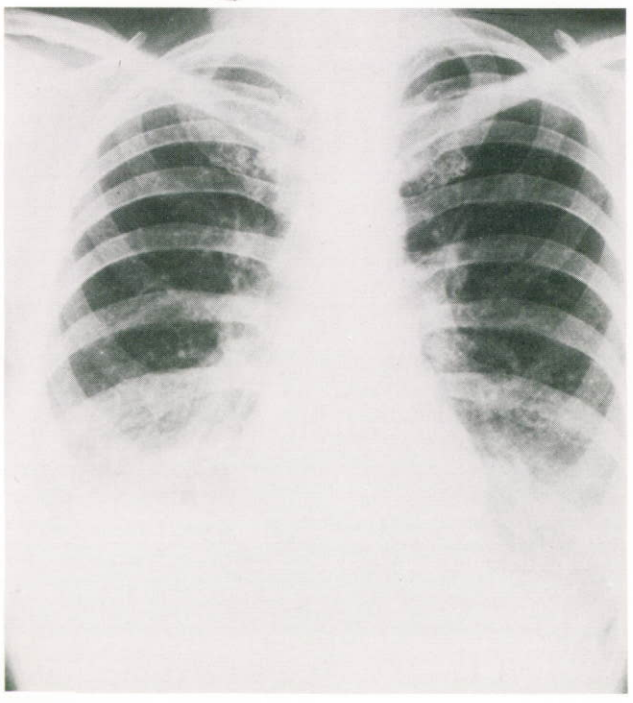

Fig 2. *Fibrosing alveolitis complicating progressive systemic sclerosis. There are bilateral lower zone infiltrates. This disease also presents with accelerated hypertension and acute irreversible renal failure.*

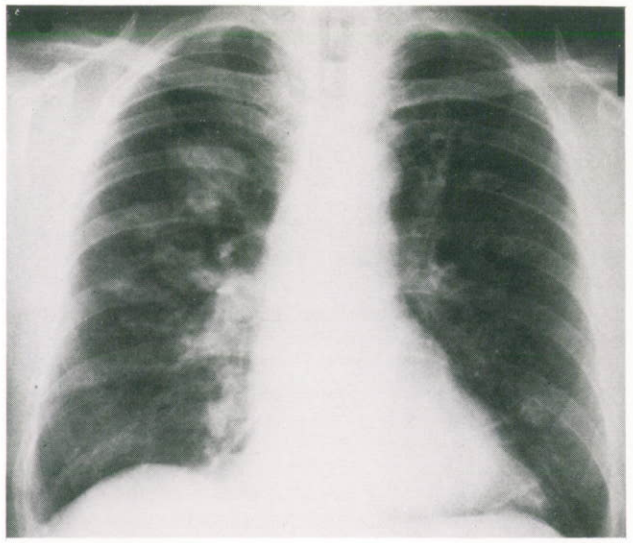

Fig 3. *Wegener's granulomatosis. There are cavitating nodules in both lung fields.*

c. The alveolar haemorrhage syndromes

These rare diseases usually present as rapidly progressive kidney failure due to glomerulonephritis, with generalised diffuse bleeding from the alveolar membranes. Some of these cases are associated with an autoantibody to glomerular basement membrane. The extent of the alveolar haemorrhage can be monitored by serial determination of carbon monoxide transfer (TCO), which is elevated in the presence of active bleeding. In remission, TCO is normal or may be reduced if there is associated pulmonary fibrosis. If the onset of this syndrome is acute, intensive treatment with high doses of methyl prednisolone and plasma exchange is often successful. Relapses can be provoked by cigarette smoking or solvent inhalation (glue-sniffing). A few chronic cases are associated with coeliac disease (intolerance to wheat protein) (3).

d. Miscellaneous

Pulmonary infections with, for example, Pneumococcus, Klebsiella or Mycoplasma may rarely cause an inflammation of the kidneys. A similar picture is a rare complication of oat cell carcinoma of the bronchus. This may be because circulating immune complexes (antibody bound to antigen from the organism or tumour) become trapped in the kidney and provoke a destructive inflammatory response and later scarring (4). Amyloidosis is a condition in which insoluble proteins are deposited in the tissues; it can complicate chronic inflammatory disease or more rarely may occur without any underlying cause. Renal failure may result, and also diffuse pulmonary disease or localised airway obstruction (5). Thrombosis of the renal veins may complicate some forms of glomerulonephritis and the occluded veins may act as a source of pulmonary thromboemboli.

Side-effects of drugs

Under this heading are considered drugs used in the treatment of kidney disease which can damage the lung, drugs used in lung disease which can cause renal side-effects and drugs used to treat a variety of conditions which may damage both the lung and the kidneys. Many drugs are excreted by the kidneys, so, if kidney failure is present, the doses have to be reduced; thus drug toxicity is much more common than in healthy people (6).

a. Drugs used in the treatment of renal disease

The main class of drugs used in the treatment of renal disease which may affect the lungs are the immunosuppressive agents such as steroids, cyclophosphamide and azathioprine, which are often used to treat glomerulonephritis. The lungs receive the entire cardiac output, at least 7000 litres of blood per 24 hours and about 9000 litres of air during the same period. They are thus likely to be major target organs for airborne or bloodborne infection, particularly if the host immune system has been impaired by drugs (7). Pulmonary infections with bacteria, viruses, Pneumocystis carinii and fungi can be fatal in the immunosuppressed host (Fig 4). Immunosuppression also permits the development of cancer, usually of the skin but occasionally resulting in primary and secondary lung tumours. An important side-effect of cyclophosphamide is pulmonary fibrosis. Azathioprine has only rarely been implicated as a direct cause of pulmonary toxicity. Antibiotics used in the treatment of renal infection (nitrofurantoin, sulphonamides) can cause pulmonary eosinophilia (8).

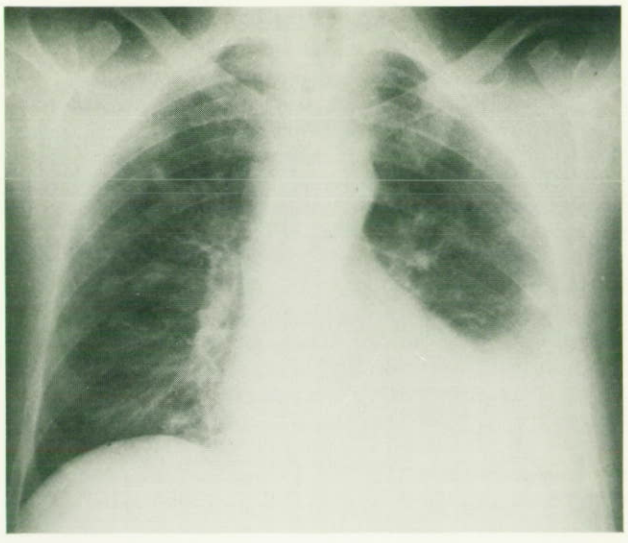

Fig 4. *Nocardia pneumonia in a renal transplant recipient on immunosuppressive drugs. There is a left pleural effusion and infiltrates in both lung fields.*

b. Drugs used in the treatment of lung disease

The normal healthy kidney concentrates drugs within the medullary tubules, hence the liability to kidney drug-induced damage. Antibiotics are usually safe, but the relatively less soluble sulphonamides can crystallise in the urine or cause acute renal damage. Other antibiotics that cause acute renal toxicity include aminoglycosides (such as gentamicin), the tetracyclines, cephalosporins and high-dose penicillin. Most other drugs used in the treatment of chest disease are free of important renal side-effects.

c. Drugs with toxicity for lungs and kidneys

Systemic lupus erythematosus (leading to lung and kidney damage) can be caused by drugs such as isoniazid (used in the treatment of tuberculosis), anticonvulsants, antibiotics and hydralazine, used for the treatment of high blood pressure. Penicillamine, used in the treatment of rheumatoid arthritis, may cause glomerulonephritis and has been implicated in the development of obliterative bronchiolitis. Heroin abusers may develop acute nephritis and pulmonary oedema or pulmonary granulomas.

The uraemic state and the lung

As kidney function progressively falls below 50% of normal, waste products and toxic substances start to accumulate. There is controversy over whether any of these chemicals can cause damage to the lungs independent of the effects of treatment (9).

a. Effects of chronic renal failure prior to treatment

A major function of the kidneys is to excrete water and as renal function falls to less than 5% of normal, pulmonary and peripheral oedema may occur for which reason many patients are given diuretics. This tendency to water retention means that any abnormality found on pulmonary function testing could be due to the effects of increased lung water rather than accumulation of toxins, which may account for the surprisingly severe impairment of carbon monoxide transfer (TCO) found in the only large study so far carried out (10); the impairment of TCO correlated only poorly with the degree of impairment of renal function.

b. Effects of haemodialysis

In a recent review of post mortems of 46 patients who had died while on haemodialysis, only one subject had normal lungs, 19 patients had pulmonary oedema, and pulmonary fibrosis and metabolic calcification were also common (11). There was no control group and so it is possible that some of these abnormalities were chance findings. Possible causes of lung disease related to treatment of haemodialysis are discussed below and elsewhere (9).

Soon after the start of a period of treatment by haemodialysis, there is a sharp drop in the white blood cell count, associated with arterial hypoxaemia. There are several possible explanations; white cells may accumulate in and block small pulmonary blood vessels, or microemboli may form within the membranes of the dialyser and be carried to the lungs (12). Both might be expected to cause acute reduction in TCO and ventilation perfusion mismatch, both of which have been found during the course of haemodialysis. It is possible that hypoventilation occurs during haemodialysis, related to carbon dioxide elimination by the dialyser or to the metabolism of acetate used in the process of dialysis. Particles of silicone may be carried from the dialysis machine to the lung, and may cause an acute alveolitis. Rare complications of haemodialysis include acute anaphylactic shock and the precipitation of asthma. In between periods of haemodialysis, studies have shown that patients have a reduced TCO, even after correction for the almost inevitable anaemia (13).

c. Effects of peritoneal dialysis

Peritoneal dialysis requires that 1.5-3 litres of sterile fluid are placed in the peritoneal cavity and left there, often for several hours, and then drained out. The procedure can be done in hospital, usually in intensive care units for acute renal failure, or patients can be trained to do it for themselves outside hospital allowing them to lead nearly normal lives. This latter technique is known as continuous ambulatory peritoneal dialysis (CAPD).

Early studies of the effects of this fluid on respiration were done in patients with acute renal failure, often gravely ill with multi-organ failure, where the excess fluid resulted in pulmonary infection and lower lobe collapse. This led to fears that pre-existing lung disease would be a contra-indication to CAPD (14). We studied 29 patients on CAPD, and reviewed the findings of other workers (15). We found no acute effect of the fluid on lung function or respiratory muscle power. We also studied three patients with pulmonary fibrosis who were also on CAPD, and found that the fluid caused no deterioration in lung function. Possible long-term effects of the fluid have not been studied.

A rare complication of CAPD is the leakage of fluid through minor defects in the diaphragm, causing massive pleural effusions. This is treated by thoracotomy and pleurectomy to obliterate the pleural cavity.

d. Effects of renal transplantation

There is some evidence that carbon monoxide transfer may remain low for at least three years after a successful transplant (10). All transplant patients require lifelong immunosuppression which until recently was usually with prednisolone and azathioprine; pulmonary complications of this treatment have already been discussed (and see Fig 4). A new immunosuppressant, Cyclosporin A, appears to have no pulmonary toxicity, and this drug is being increasingly used to prevent rejection of renal transplants.

Acknowledgements

I am grateful to DR I H Kerr, Consultant Radiologist, for the radiographs used in Figs 2-4, and to Dr Roger Gabriel, Consultant Nephrologist, for Fig 1 and for much helpful teaching and advice.

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SYMPTOMATIC OXYGEN

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"A daily charge of oxygen has enabled a man previously virtually immobilised by breathlessness to move about the house and to walk out to sit in the garden." On portable oxygen therapy (1).

Summary
Symptomatic oxygen can benefit certain patients with severe disability and although there is no hard and fast relationship, those in whom there is substantial desaturation on exercise are likely to benefit most. Otherwise, in chronic airflow obstruction there is no clinical predictor of which patients will benefit. Clearly patients must be stable and on maximum conventional therapy before symptomatic oxygen is considered and under these circumstances there should be an objective assessment using simple measurements of endurance and breathlessness which convince both patient and doctor that symptomatic oxygen is worth pursuing. Ideally the patients should then have a trial period of one to two months using oxygen in the home where they will have the opportunity of testing its value in the context of their normal lives.

Introduction
It is commonly believed by the public that difficulties with breathing can universally be relieved by oxygen (O_2). It is certainly true that the vast majority of patients receiving domiciliary O_2 use it as a temporary relief for breathlessness and, indeed, less that 5% are said to use it for more than 2-3 hours per day (2). Is this a rational and scientific way of using O_2 or is one merely providing an expensive placebo for relieving breathlessness? In this article I will consider the evidence and justification for symptomatic O_2 and simple measures for assessing its effectiveness.

Rationale for using oxygen during exercise
The whole area of supplementary O_2 therapy was first examined by Haldane in 1917 (3) when its use was considered for those who were hypoxaemic. This work was extended and the principle established in 1960 by Campbell (4). It was not until the 1950s that O_2 was tried expressly as a means of improving exercise tolerance in patients with debilitating lung disease (1).

There are a number of reasons for expecting oxygen to improve exercise tolerance. Hypoxaemia is known to reduce exercise capacity and oxygen consumption in normal subjects (5). In addition, oxygen is known to reduce minute ventilation for a given oxygen consumption both in normal subjects and in those with airflow obstruction (6). The reasons for this are obscure and the evidence somewhat contentious, but in patients who desaturate during exercise the effect may be due in part to a reduction in hypoxic respiratory drive (7).

A number of studies have suggested that supplementary oxygen may increase the potential maximum oxygen consumption of the patient, but the evidence is conflicting and difficult to interpret due to the technical difficulties of measuring O_2 consumption in patients breathing O_2-enriched air (7). In addition, the favourable effects of O_2 upon haemodynamics and upon pulmonary hypertension in particular, are well documented (8). Finally, it has been suggested that the provision of oxygen acts merely as a placebo (9) and it is interesting in this respect that compressed air delivered by mask, by cooling the face, is capable of reducing minute ventilation significantly, presumable due to a change in the breathing pattern.

The evidence for symptomatic O_2
Cotes and Gilson (1), working amongst the miners of South Wales, examined the effects of portable O_2 and found that many patients were able to cover up to four times their normal walking distance before being incapacitated by breathlessness when receiving oxygen at 4 litres per minute; the mean endurance was improved by 213% in a group of 29 patients with severe airflow obstruction. Although this study has been criticised on the grounds that the patients knew that they were receiving O_2, it set the scene for the prescription of domiciliary oxygen as symptomatic relief for breathless patients.

Since that time, the benefits and effects of oxygen upon exercise capacity have been studied in some detail. On cursory examination the results seem to show a striking variability of benefit in terms of exercise tolerance and understandably this may have brought symptomatic oxygen into disrepute. However, the disparity is due largely to experimental technique and it is important for one to appreciate that the assessment of exercise capability is complex and findings vary considerably depending on the methods.

The ability to exercise may be assessed in three main ways: work capacity, speed and endurance.

1. **Work capacity** is the ability to achieve a given level of energy expenditure. It is conventionally measured by some form of progressive exercise test where the subject performs (either on a treadmill or cycle ergometer) at increasing workloads until exhaustion. The main purpose of this type of test is to evaluate cardiopulmonary function and determine the limiting factor in the individual's exercise capacity, be it respiratory or cardiac.

2. **Speed** is related to work capacity and is most simply measured by the distance covered within a given time either on a treadmill or as a corridor walk. Currently the most common test is the six- or twelve-minute walking distance. Clearly this test may be limited by peripheral vascular disease, neuromuscular disease or orthopaedic problems before a patient is constrained by heart or lung disease.

3. **Endurance** is the ability to sustain a level of energy expenditure and this is usually measured by the length of time that a subject can exercise at a given workload, or the distance a patient can walk to exhaustion, independent of time.

O_2 has different effects upon each of these measurements. In interpreting exercise tests or drawing conclusions about the benefits of oxygen, therefore, one must be mindful of the type of assessment used.

In the initial study done by Cotes and Gilson (1) the main assessment of exercise capability was the absolute distance

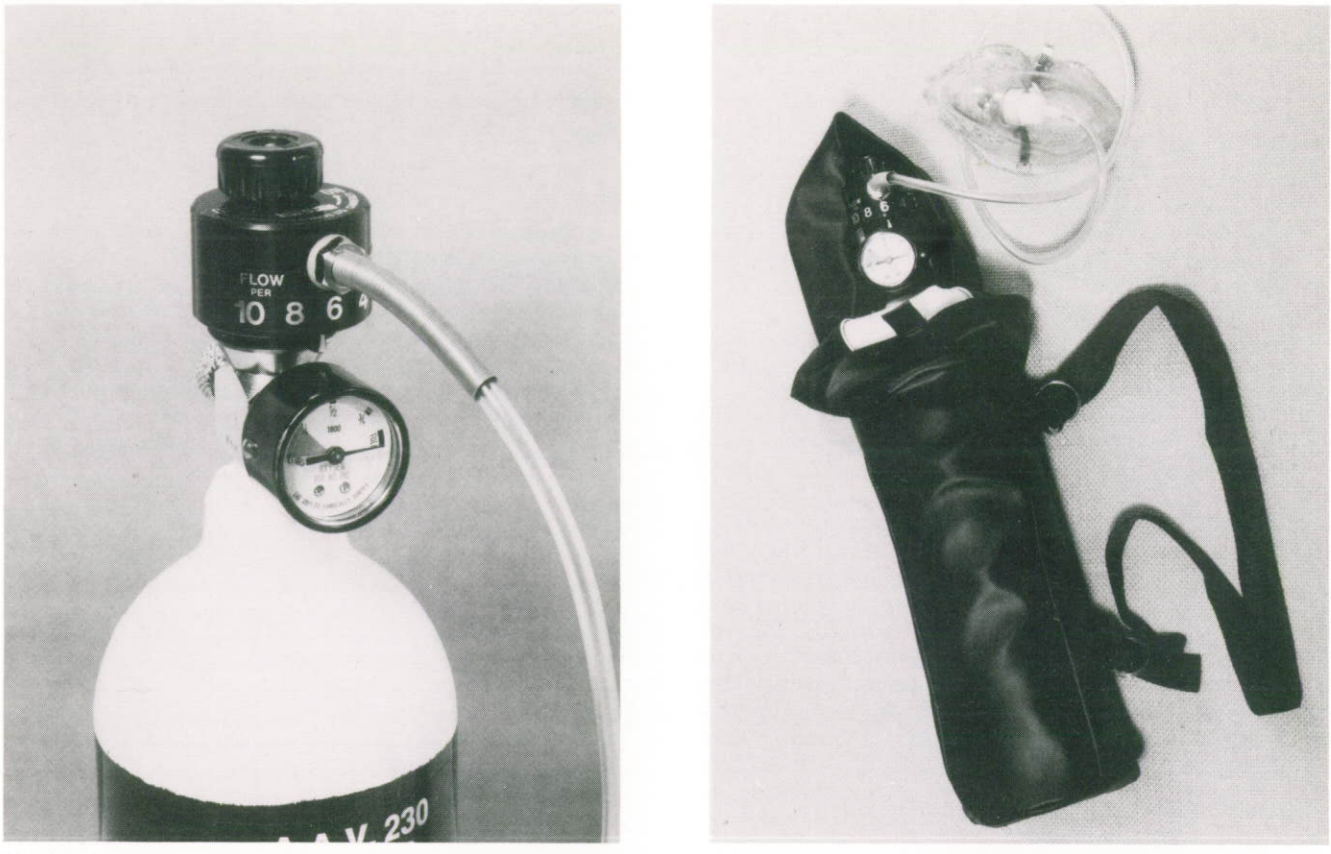

Fig 1. *Portable oxygen inhaler, showing valve, mask and carrying case. Approx. weight 6lb (2.7kg); overall length 18½" (47cm). By courtesy of Sabre-AAV Ltd (previously Air Apparatus and Valve Co Ltd).*

a patient could cover to exhaustion. After O_2, a small but significant number increased this distance four-fold, and overall the patients were able to cover more than twice their normal walking distance. Subsequently Bradley and colleagues (10) studied a similar group of patients with severe obstructive lung disease and found that, while maximum work rate was uninfluenced by oxygen, the patients' endurance at constant workload was increased by 60%. In addition, there was a reduction in blood lactate implying that the benefit was due in some measure to improved oxygenation.

The effects of portable oxygen upon speed has also been examined in some detail. In patients with chronic airflow obstruction and cor pulmonale, the twelve-minute walking distance may be improved by up to 25% (11) though this benefit is offset by the exertion of carrying a portable oxygen supply. Woodcock and colleagues (12,13) observed that O_2 brought about an improvement in six-minute walking distance of the order of 10% and a reduction of breathlessness by between 20 and 30%. They made one other interesting and important observation, namely that the use of oxygen just prior to short periods of exercise was almost as effective as continuous oxygen supplements (12).

In some interesting studies from the United States, oxygen has been used in a slightly different way. With the 'transatlantic' emphasis upon training in pulmonary rehabilitation, oxygen may be used in patients who desaturate significantly upon exercise as a safety measure and as a means of allowing training at workloads that would otherwise be constrained by hypoxaemia. The combination of oxygen and exercise was first considered by Pierce (14) who showed that, in terms of safety, the use of supplementary oxygen was wise. Conflicting data have appeared in other studies, however, where it is difficult to divorce the benefits of training from that of exercise with oxygen (10). However, recently Zach and colleagues (15) have found oxygen to increase maximum workload per se and this seemed unchanged by subsequent rehabilitation. The benefit also seemed independent of desaturation. Furthermore, they also found endurance to be increased by an additional 40% over and above the effects of training, and the twelve-minute walking distance which was improved by 50% with training rose by an impressive 72% when combined with oxygen therapy.

In summary, patients' endurance may improve by between 50 and 200% and at best may increase by four-fold with the use of symptomatic oxygen, whereas speed is only increased modestly (up to 20%); the maximum exercise capacity of an individual is probably uinfluenced by supplementary oxygen.

The provision of symptomatic O_2

1. Requirements

In the home For symptomatic use, the majority of patients will require supplementary oxygen for less than two hours per day, which should be provided as cylinders. For use around the home, patients should ideally be able to use oxygen during exercise or at least be able to use oxygen just prior to exercise. On this basis patients will require a number of cylinders which should be placed in strategic positions (eg, bedroom, bottom of the stairs, main living room) and enough tubing should be provided to allow mobility. In addition, all patients should be advised that pre-dosing with oxygen may be just as effective as using it during exercise and that they should also continue using the oxygen until breathlessness has resolved. It should be emphasised that oxygen is not addictive, not is it responsible for any deterioration in lung function.

Portable oxygen Portable oxygen (not prescribable under the NHS) is available in small rechargeable cylinders either as compressed gas or as liquid oxygen. The cylinders weigh about 3kg but only contain sufficient gas for 45 minutes use at a flow of 4 litres per minute. They must be refilled from the conventional home cylinders which are only capable of generating sufficient pressure for 2-3 fillings. Liquid oxygen carriers hold sufficient gas for about four hours use, but the logistics and expense of refilling these is probably prohibitive for the majority of patients (Fig 1).

In an attempt to prolong the effective life of portable cylinders, devices for conserving oxygen have recently come onto the market. The simplest amongst these are nasal spectacles with some form of reservoir device (eg, Oxymiser). These allow adequate oxygen supplement at approximately half the flow rate ordinarily required and therefore have the potential of doubling the lifespan of a portable cylinder (16). More sophisticated valve devices have also been investigated but these are probably not a tenable proposition for widespread use (17). In a limited number of patients, direct transtracheal delivery of oxygen has met with some success, though this method is more suitable for patients requiring long-term oxygen treatment (18).

2. Assessments

It is clear from the literature that in chronic airflow obstruction there is no single clinical index that will single out the patient likely to benefit from symptomatic oxygen treatment. It is therefore important that some attempt is made to quantify a patient's response to oxygen. The investigations performed at the London Chest Hospital are fairly exhaustive but we would recommend the following as a minimum in the assessment of patients.

Exercise capabilities These should be assessed using a six-minute walking distance for speed and some measure of endurance, either in the form of a distance walk to exhaustion independent of time or a formal exercise test at 50% maximum work capacity to exhaustion. In all the investigations the patients should be taught the technique and given at least one preliminary trial, and should always be encouraged to perform at their best (19). The results of both six-minute distance and endurance are surprisingly reproducible after one or two attempts. Thirty minutes rest is usually sufficient between walking tests.

Breathlessness Breathlessness is probably best measured using a visual analogue scale. This is a simple technique where a patient is asked to mark a blank line 10 cm in length, the extremes of which have been labelled 'not breathless' and 'extremely breathless'. The patient should be instructed to gauge their symptoms and make a mark at an appropriate distance. This is also a reproducible test and is simple to perform.

More sophisticated tests, whilst interesting, are more of academic interest than of practical benefit in the assessment of patients for symptomatic oxygen. The final arbiter should be an improvement in breathlessness and/or exercise capability.

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SPRING SCIENTIFIC MEETING

The Spring Meeting of the Association took place on 19 April 1986 at Frenchay Hospital, Bristol.

We owe grateful thanks to Sonia Jackson for organising the meeting and to the speakers for their interesting and varied contributions.

We are most grateful to the following firms who generously contributed towards the cost of the meeting:

BMS Critical Care Ltd*
Cardiokinetics Ltd
Cobe Laboratories Ltd*
Cranlea and Co Ltd
Graphic Controls Ltd
Gould Electronics Ltd*
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Medic-Aid Ltd
Micro Medical Instruments Ltd
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*Also exhibited at the meeting.

The following scientific papers were given:

1. Thymectomy in myasthenia gravis. P S Simpson, *Consultant Anaesthetist, Frenchay Hospital, Bristol.*
2. Trans-tracheal oxygen treatment. M H Lloyd, *Respiratory Physiology Department, Harefield Hospital, Harefield.*
3. Bronchiectasis. S Hill, *Respiratory Physiology, The General Hospital, Birmingham.*
4. Bird fanciers' lung. R White, *Consultant Physician, Frenchay Hospital, Bristol.*
5. Temperature corrections in routine spirometry. D Cramer, A Peacock, D Denison, *Lung Function Unit, Brompton Hospital, London.*
6. Peak expiratory flow measurements: a comprehensible approach. A Henman, D Parry, *Respiratory Physiology Department, Guy's Hospital, London.*
7. Effect of Freon-22 on the analysis of CO by an infrared analyser. A Kendrick, G T R Lewis, *Respiratory Physiology Department, Bristol Royal Infirmary, Bristol.*
8. Effects of domiciliary nebuliser therapy. M Ward, J W Hadfield, A Tattersfield and A Bebbington, *Respiratory Physiology Departments, Derby City Hospital, Derbyshire Royal Infirmary and Nottingham City Hospital.*
9. The management of equipment in a respiratory laboratory. G Manning, *Cardiothoracic Measurement Department, Derbyshire Royal Infirmary.*

ABSTRACTS

Thymectomy in Myasthenia Gravis

P S Simpson

Myasthenia gravis is a condition characterised by altered transmission at the skeletal neuromuscular junction. The main presenting symptom is that of weakness which is gradually progressive throughout the day and may affect different muscle groups to a variable extent. Some patients, for example, may have drooping eyelids and difficulty in swallowing, while others may have difficulty in breathing and yet have quite normal power in their limbs. Considerable research has shown that the main lesion is an autoimmune one, whereby the patients produce antibodies to their own neuromuscular junctions. Although symptoms can be relieved by the taking of anticholinesterase drugs, a total cure necessitates the prevention of antibody production.

Antibodies are produced and modified in a number of sites, particularly the thymus gland, situated in the anterior mediastinum of the chest and many patients with myasthenia gravis have a benign thymoma. At Frenchay, we have had a considerable number of referrals of patients with myasthenia gravis for elective thymectomy and in this study 30 such patients are reviewed, 20 females and 10 males, whose ages ranged from 14 to 75 years.

The duration of symptoms was anything from one month to seven years (commonly 18 months to two years); the main presenting symptoms were double vision and drooping eyelids, together with dysphagia. All the patients had been receiving anticholinesterase drugs preoperatively, together with a variety of other medications which included steroids, azathioprine and, more recently, preoperative plasmapheresis. In all cases, operative thymectomy was performed through a median sternotomy, since this provided the best access to the gland with minimum trauma.

Of the 30 patients studied, only 8 had a normal thymus gland and a further 15 had either mild thymic hyperplasia or a distinct thymoma. One had a malignant thymic tumour and two suffered from Hodgkin's disease of the thymus gland. In addition, two of the patients were thyrotoxic and one was diabetic, both factors commonly associated with myasthenia. 18 patients had an uneventful postoperative course, while a further 10 needed intensive care at some stage, of whom 6 were electively ventilated. The commonest causes for this were difficulty in managing the postoperative anticholinesterase regime resulting in relative overdosage and weakness. It appeared that the chief time of risk was not immediately after operation but rather 2/3 days into the postoperative period when the patients had been restarted on their drugs. The long-term results of thymectomy are encouraging. Of all the myasthenic patients studied, 12 ultimately had reduced symptoms and reduced anticholinesterase requirements, two had a complete cure and four were unchanged. None were made worse.

Trans-Tracheal Oxygen Treatment

M H Lloyd

Over the course of the last two years trans-tracheal oxygen via a micro-catheter, in patients with Chronic Obstructive Airways Disease, has been introduced at Harefield Hospital. This treatment has been investigated by Dr J R Govan, who first saw the system in use in the U.S.A.in the early '80s. A Teflon catheter, diameter 17mm and length 13cm, is inserted percutaneously under local anaesthetic into the trachea between the second and third rings. The patients are then supplied with two portable oxygen cylinders, and the appropriate regulators and connecting devices, and a domestic source of oxygen is also arranged. The system has been in use continuously for up to eighteen months with only minor complications, and the catheters are routinely changed at three-month intervals.

Each patient is carefully assessed prior to the insertion of the system. Lung function tests are performed, including forced ventilation, flow volume loops, lung volumes by

helium dilution, bronchodilater effect and transfer factor measurements. Arterial blood gases are taken with the patient breathing air and repeated after a suitable interval, with the patient breathing oxygen at 3l per minute via nasal cannulae. Further to this two six-minute walks are performed, one whilst the patient is breathing air, and after a recovery period a repeat walk is performed with the patient breathing oxygen at 3l per minute via nasal cannulae.

Over the course of two to three days, after the insertion of the catheter, several arterial blood gas samples are taken at differing oxygen flow rates and the optimum level derived. The patient is then discharged home.

Arterial blood gas results indicate that using the transtracheal oxygen system relieves hypoxaemia with 50% less oxygen than is required using nasal cannulae. The system is more comfortable and cosmetically more acceptable than the face mask or nasal cannulae and has lead to improved patient compliance when continuous oxygen therapy was prescribed.

Bronchiectasis

S Hill

Bronchiectasis is a chronic lung disorder characterised by the presence of dilated bronchi, bronchitis, loss of connective tissue and mucosal ulceration in addition to reduced muco-ciliary clearance. The term covers a wide spectrum of clinical disorders ranging from limited pathological changes associated with few symptoms (except for the occasional infective episode) to gross pathological changes and persistent symptoms such as a cough productive of copious secretions (often purulent in nature), permanent low-grade ill-health and in some cases progressive deterioration.

We have been interested in the role of proteolytic enzymes and in particular neutrophil elastase in the pathogenesis and progression of this disease. There is experimental evidence showing that purified human neutrophil elastase can damage bronchial epithelium and reduce ciliary beat frequency in vitro, both features of bronchiectasis. In addition this enzyme is frequently present in bronchiectatic secretions. Thus we were interested in the effects of antibiotic therapy on elastase in order to clarify the role of these agents in the treatment of these patients.

The assessment and classification of secretions from these patients have shown that enzyme activity is only usually present in purulent secretions and thus potentially harmful to the lung. These secretions were shown to be associated with greater physiological evidence of lung damage, greater inflammation in the lung and reduced ciliary beat frequency in vitro. However, the potentially harmful nature of these secretions could often be reversed following conventional broad spectrum antibiotic therapy given for 14 days.

It appears therefore that purulent (enzyme-positive) secretions may contribute towards a vicious circle in bronchiectasis, leading to inflammation, further tissue damage and progressive deterioration in some patients. Antibiotic therapy can reverse the potentially pathogenic nature of the secretions, break the vicious circle and thus may have a crucial role to play in the management of such patients.

Bird Fancier's Lung

R J White

A number of cases of bird fancier's lung were presented. The main symptom is of breathlessness but cough can be prominent and, in younger patients, weight loss may occur. The usual signs in the lungs are of fine inspiratory crackles at the bases. The X-ray in the early stages shows a hazy, ground-glass appearance and, in later stages, actual lung fibrosis develops. Lung function testing shows restricted ventilatory function with reduced gas transfer and all of the physiological changes are completely reversible providing the diagnosis is made early. Confirmation of the diagnosis is obtained by the finding of specific avian antibodies in the blood. Treatment with corticosteroids in addition to removal of the bird is recommended.

Temperature Corrections in Routine Spirometry

D Cramer, A Peacock, D Denison

Forced expiratory volume and forced vital capacity were measured in nine normal subjects with three Vitalograph and three rolling seal spirometers at three different ambient temperatures (4, 22 and 32°C). When the results obtained with the rolling seal spirometer were converted to BTPS the agreement between measurements in the three environments improved, but when the Vitalograph measurements obtained in the hot and cold rooms were converted, an error of up to 13% was introduced. The error was similar whether ambient or spirometer temperatures were used to make the conversion. In an attempt to explain the behaviour of the Vitalograph spirometers the compliance of their bellows was measured at the three temperatures. Compliance was higher at the higher temperature (32°C) and lower at the lower temperature (4°C) than at the normal room temperature. These changes in instrument compliance could account for the differences in measured values between the two types of spirometer. It is concluded that the ATPS-BTPS conversion is valid and necessary for measurements made with rolling seal spirometers, but can cause substantial error if it is used for Vitalograph measurements made under conditions other than normal room temperature.

Peak Expiratory Flow Measurements — a Comprehensible Approach

A Henman, D Parry

There are many reasons for carrying out domiciliary peak flow measurements. The usefulness of such measurements depends on their validity and interpretation; our aim was to improve both of these.

We have assumed responsibility for the issuing of all domiciliary peak flow meters, designed a computer programme for the processing of the results and designed an uncomplicated diary card for patients to complete. This has resulted in consistent patient instruction, standard processing and a comprehensible report. Results are reported as an A4-sized graph showing 28-day 'blocks' of results and the predicted range.

We feel that graphic display of such measurements facilitates quick and easy interpretation. Our programme also allows some analysis such as calculation of diurnal variation. The system uses a BBC B microcomputer and is written in BASIC. Data is stored on floppy disc, up to 100 days per file, with three readings per day.

Effect of Freon-22 on the Analysis of CO by an Infra-Red Analyser

A H Kendrick, G T R Lewis

Infra-red gas analysers are used in respiratory departments principally to measure carbon monoxide (CO). Water vapour, oxygen and carbon dioxide concentrations are known to affect the selectivity of the infra-red analyser for CO. Water vapour and carbon dioxide are usually removed chemically, and oxygen only becomes important at high concentrations.

The effects of Freon-22 have not been previously investigated, although other workers have observed that the presence of Freon-22 in a mixture containing CO seriously affects the analysis of CO using an infra-red analyser.

We have investigated the effects of differing concentrations of Freon-22 (range 0-5%) in a mixture of CO (range 0-0.3%) and nitrogen. Freon-22 seriously interferes with the selectivity of a commercially set up analyser. Inspection of the infra-red spectrum reveals a Freon-22 peak at a wave number of approximately 2200, overlapping the principal CO peak.

To make any measurements of CO with Freon-22 present, the gain of the instrument must be reduced, and the analyser should be calibrated specifically for a CO/Freon-22 mixture. However, correction of the CO for the presence of Freon-22 is still required. We have therefore derived a correction equation for 2 different commercial analysers, which accounts for the level of Freon-22 present. Mixtures of CO and Freon-22 in nitrogen (n = 68) were obtained using Wosthoff pumps in the ranges given above. Multiple linear regression equations compared. There was no significant difference between the equations.

Measurements of the single-breath carbon monoxide transfer factor (Tlco) were made in 10 subjects with and without Freon-22 present. The analysers were calibrated specifically for each mixture. Where Freon-22 was present, the equations were applied. No significant differences were found between the Tlco(Freon) and Tlco(no Freon).

We conclude that when Freon-22 is present in a mixture containing CO, which is to be analysed on an infra-red analyser, that 1) the gain of the analyser should be decreased, 2) a correction equation should be applied to correct the CO concentration for the Freon-22 concentration, and 3) the equation appears to be applicable to any correctly set up CO infra-red analyser.

Cardiac Effects of Domiciliary Nebulisers

J W Hadfield, A Tattersfield, A Bebbington, M Ward

Study 1: Eleven patients were studied at home by ambulatory ECG monitoring. Their age range was 34-67 years and all had chronic severe asthma. Salbutamol was used by all patients (dose range 5-60mg daily); six patients also used ipratropium (0.5-3.0mg daily). Nebulisers were driven by both air and oxygen. A record of heart rate was made before and at five-minute intervals for the hour following nebuliser use. Heart rate increased after nebuliser therapy from 89±4.4 beats/min to 100±5.9 at twenty minutes. Arrhythmias occurred and in one patient appeared to be related to bronchodilator therapy. Individual heart rate changes varied considerably, though the patient with the most severe airways obstruction developed the greatest tachycardia. These results are in contrast to previous work.

Study 2: Eighteen patients (age range 43-75 years) were studied in the laboratory. There were three treatment periods in which patients received nebulised salbutamol in doses of 200μg, 1mg and 5mg four times daily. In each treatment period a 5-minutes step test was performed together with 24-hour ECG monitoring. Patients completing the crossover study entered a further 2-week period of 2.5mg nebulised salbutamol. Heart rate immediately before nebuliser use was 94, 91 and 95 beats/min on the 200μg, 1mg and 5mg dose. Twenty minutes after the start of nebulisation the changes in heart rate were −2%, +0.6% and 1.7%. These differences were not significant.

A laboratory dose response assessment was performed in an attempt to identify the patients needing higher doses. However we found laboratory assessment does not identify these patients. Some patients do benefit from high dose beta agonists. Large differences in dose produce small differences in overall response. Many patients currently using domiciliary nebulisers could probably be managed with lower dose inhaler devices. No nebuliser-related cardiac dysrhythmias were identified.

The Management of Equipment in a Respiratory Physiology Department

Gillian Manning

In January 1982, The Health Equipment Information Bulletin No 98 entitled "Management of Equipment" was produced following the investigation of a number of serious accidents which revealed shortcomings in the management of equipment in hospitals.

The aim of the document was to "recommend a system of equipment management," the recommended system affecting all users of equipment including respiratory physiology departments.

The main areas covered in this document are:—

1. To define the responsibilities for equipment management.
2. To define a procedure for:—
   a. equipment selection
   b. acceptance of new equipment
   c. training of staff
   d. servicing of equipment
   e. replacement/disposal of equipment.
3. To recommend that a complete up-to-date inventory of equipment is maintained.

The aim of this paper is to summarise the document to provide a basis for a comprehensive equipment management policy in a respiratory physiology department.

Note: Health Equipment Information Number 98 available from: Mr C W Crisp, Department of Health and Social Security, Health Services Supply Branch, 14 Russell Square, London, WC1.

BOOK REVIEW

PULMONARY FUNCTION TESTING — Indications and Interpretations

Editor — *Archie F Wilson*

Grune & Stratton Limited, 1985. 353 pages

ISBN 0-8089-1692-0. Price £39.50 (hard-back)

Like the preceding volume, "Guidelines and Controversies", this book is a product of the California Thoracic Society. It is primarily aimed at physicians requesting pulmonary function tests, although it may be of some use to the technician wishing to gain greater knowledge of interpretation.

The first chapter provides an overview of the problems associated with interpreting data, such as inaccuracies of measurement and definitions of abnormality. The next eleven chapters discuss routine pulmonary function tests. Each chapter provides a brief summary of the methodology of testing, indications for performing the test and examples of interpretation. The remaining chapters cover the more advanced techniques or specific areas. Chapter 16, covering computer applications, seems out of place and is very poor in content. Chapter 15 attempts to bring together all the routine tests and this should have been enlarged considerably with more examples of the variations found in common diseases.

As with most books written by a number of authors, the standard varies considerably from chapter to chapter. In summary, the book provides an introduction to the interpretation of pulmonary function testing, but one cannot recommend all technicians to purchase a copy in order to obtain an authoritative work on this topic.

Trefor Watts

SELECTED ARTICLES

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NOTE FROM THE EDITORS — Vacancies advertised in *Breath*

We are glad to accept advertisements for job vacancies in *Breath* but regret that we are unable to take responsibility for verifying that the conditions of the post are as advertised. We strongly advise applicants for any post to check on the terms and conditions of service and particularly on special items such as equipment or training facilities. We would be glad to hear of any such errors that arise in job advertisements appearing in *Breath*.

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For even more flexibility, real-time monitoring can be added to the system using the V1000 Digital Storage Monitor.

This provides a continuous, multi-channel display of all the signals and annotation at the ES1000 writing head, even with no paper running! The display can be frozen and hard copies made on to ES1000.

If required, both the ES1000 and V1000 can be controlled from a remote station equipped with a slave screen monitor.

- 1-40 channels
- 250mm chart width
- Overlapping traces
- Resolution to 0.1% F.S.
- 10^{-4}mm/s to 25m/s effective chart speed
- 40μs peak capture – 3db at 15kHz
- IEEE 488 compatible

Write or phone for full data to
Gould Electronics Ltd.
Instrument Systems,
Roebuck Road, Hainault,
Ilford, Essex IG6 3UE
Telephone 01-500 1000 Telex 263785

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BODYSCREEN II By JAEGER—

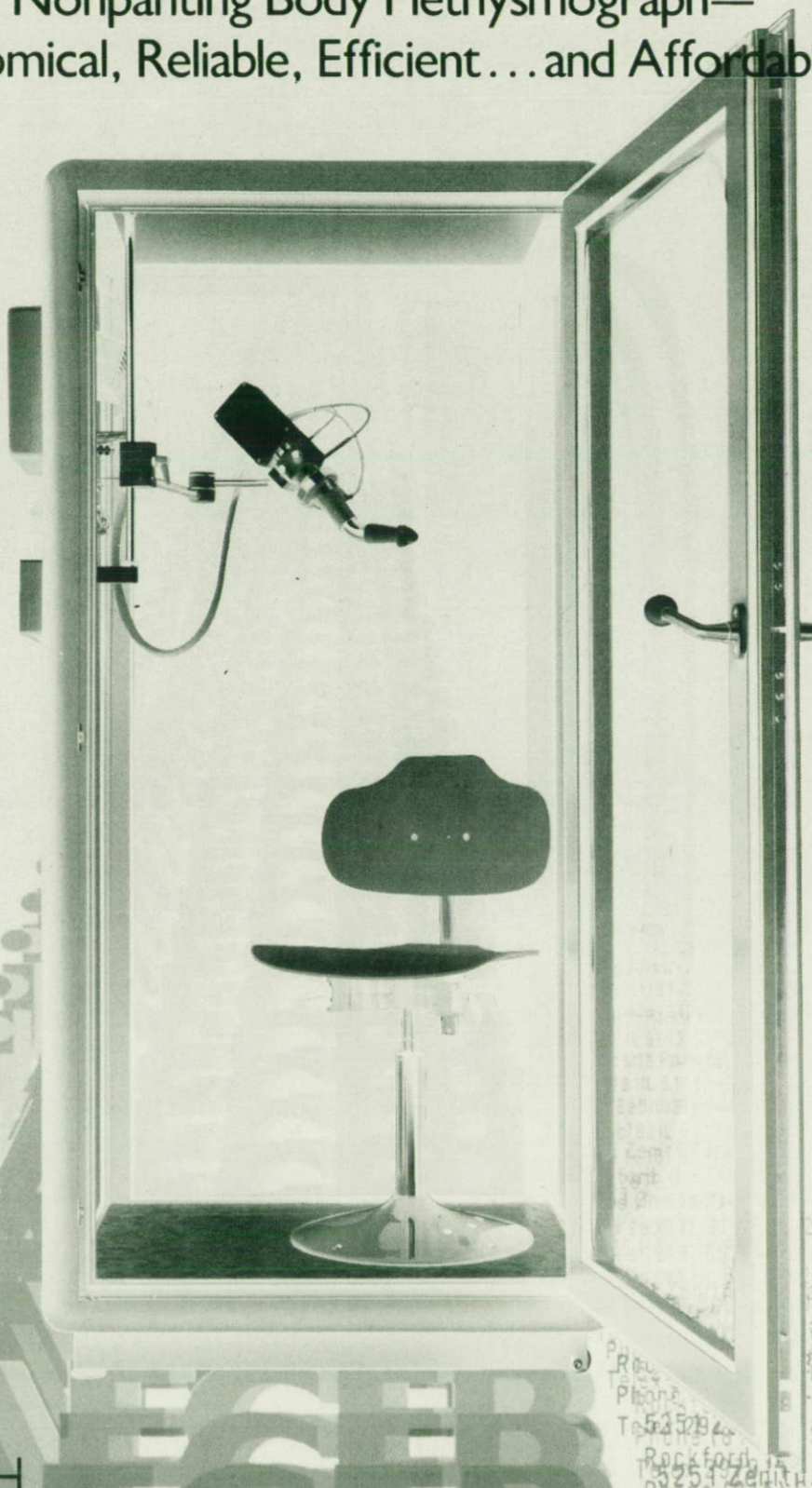
Nonpanting Body Plethysmograph— Economical, Reliable, Efficient . . . and Affordable

Jaeger built the very first commercial nonpanting body plethysmograph in the early 60's, and we are still leading in advanced technology, design, and simplified operation. BODYSCREEN II® represents our commitment to remain the leader in pulmonary diagnostics.

The use of real time computing for quick, accurate study of pulmonary function is another first from Jaeger. Our 35 years of experience and research, document the effectiveness of our specially designed microcomputer system to record and analyze the data generated in body plethysmography.

Programs available include spirometry, flow-volume, breathing mechanics, dynamic and static compliance, provocation, and more. View loops on the CRT, then generate a hard copy of your results along with predicteds or other data of your choice.

BODYSCREEN II is simple to use. Select the program, then concentrate on the patient. Operation is virtually automatic as the patient breathes normally and comfortably (NO PANTING IS NEEDED FOR ANY PORTION OF THE TESTING). In just five minutes, testing is complete and the system is ready for the next procedure.



- Floppy disk drive for the BODYSCREEN II provides additional flexibility for storage and other user applications.
- Jaeger quality is unequalled, providing years of trouble-free operation. When needed, however, parts and service are readily available.
- Expert installation and in-service training and seminars for your staff demonstrate Jaeger's commitment to our customers.
- BODYSCREEN II is just one of a full line of cardio-pulmonary diagnostic instrumentation by Erich Jaeger, Inc. ranging from simple pulmonary function testing to a multi-user lab. Call or write for complete information.

ERICH JAEGER

Pulmonary Function Diagnostic Systems

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