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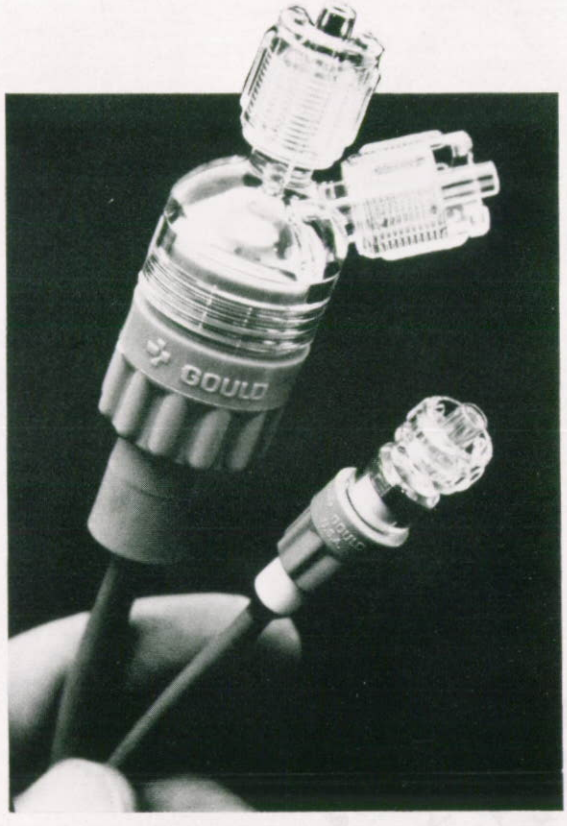

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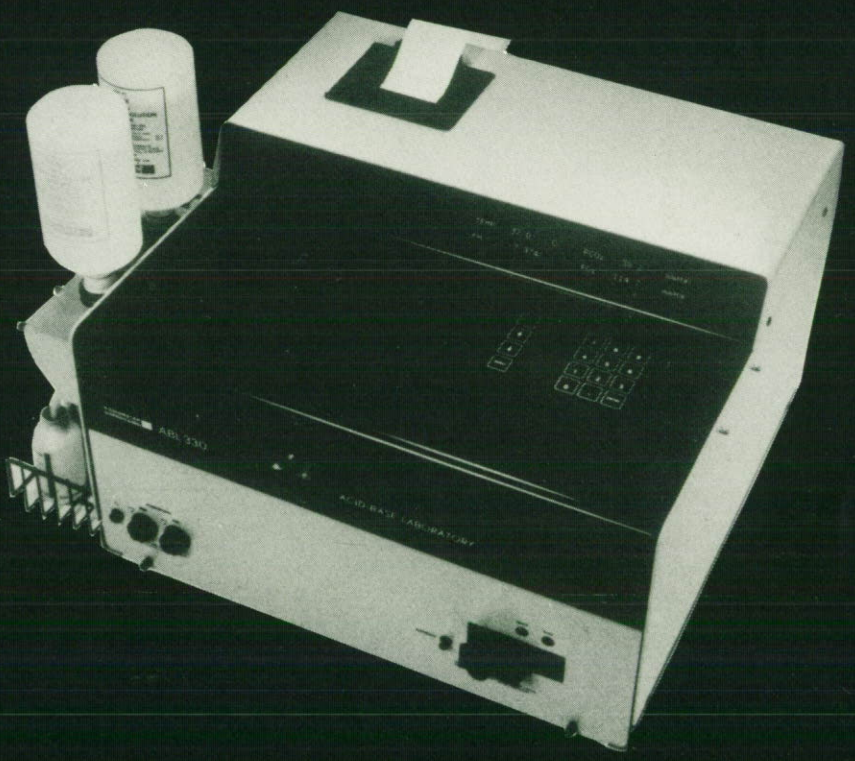

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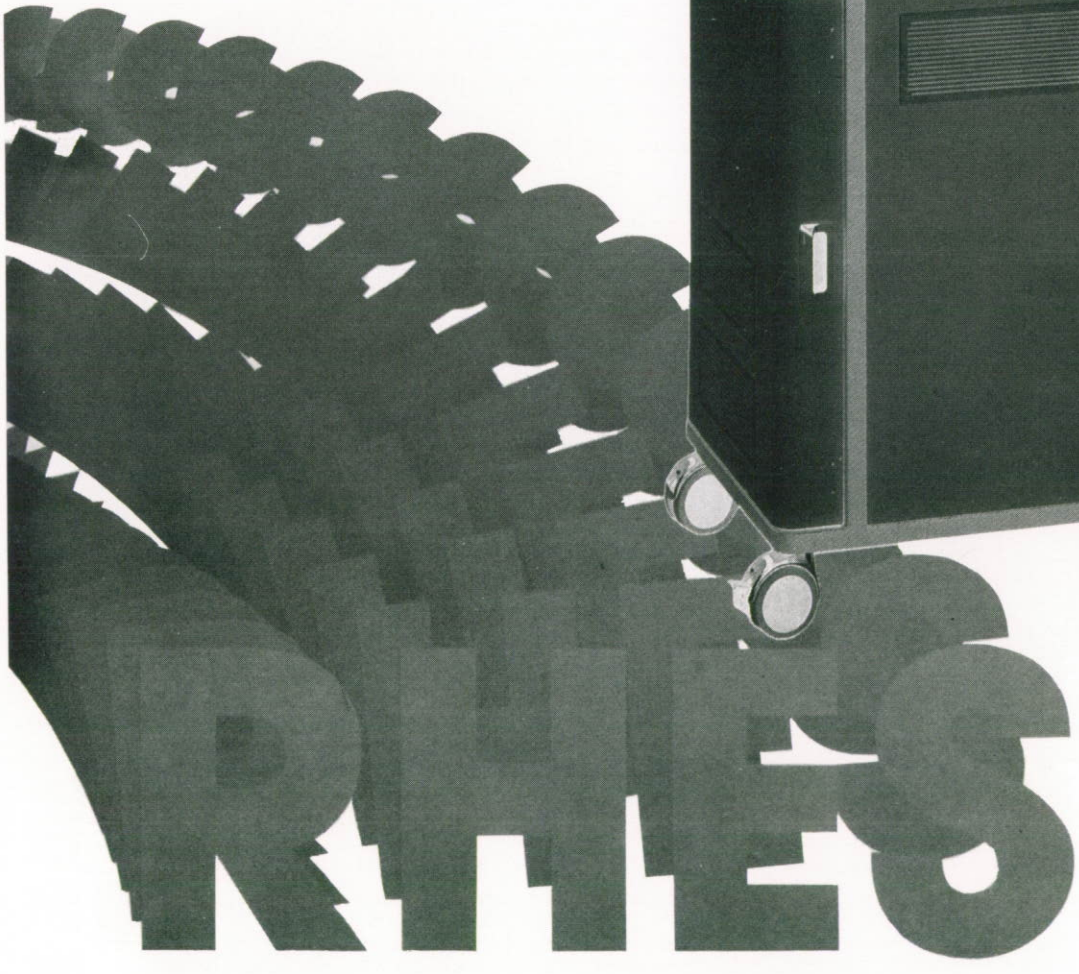

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EDITORIAL

How normal is your lung function?

A. H. Kendrick, Respiratory Department, Bristol Royal Infirmary.

Normal values of pulmonary function may be compared with the measured values when estimating impairment of lung function, and are therefore a prerequisite for clinical and epidemiological studies of lung disease. Usually the results of lung function tests are employed in conjunction with a physical examination, patient history and other laboratory tests before a positive diagnosis is made. Furthermore, very valuable information may be obtained from serial measurements, which may assist in diagnosis or may reveal a subsequent abnormal decline in lung function although the actual values remain within the normal expected range.

The European Community for Coal and Steel (ECCS) have reviewed the multitudinous number of reference equations available as part of standardization of lung function (1). Though not exhaustive, this extensive review provides great insight into the normal values presently available, and in particular the problems faced by anyone trying to choose equations for their own laboratory.The ECCS found, not surprisingly, that there was great variability in predicted values for all the commonly used lung function indices. This variability may have been due to the selection of subjects, methodological and technical differences or the type of population studied. The purpose of this editorial is to highlight some of the problems encountered and to describe the production and use of predicted equations.

The word "normal" implies that "a particular person or thing does not exceed certain limits, or does not deviate far from an average or standard established for a group, class or species" (2). The problem with defining "normal" in the context of the lung function indices lies firstly in the definition of the population and secondly, of the selection criteria. Many equations have been derived from specific groups of subjects such as coal miners, factory workers or medical personnel, rather than from a general population. Where a general population survey has been carried out, it has generally been within a specific region of a country, and it must be questioned whether the lung function of individuals in "an essentially pollution free area" (3) is representative of the population as a whole.

The choice of population group, to some extent determines the selection criteria applied (4). With a general population survey, the selection of normal individuals should be made after completion, whereas for occupational groups and hospital populations, the recruitment and rejection criteria need to be precisely defined beforehand.

Theoretically, a general population survey should provide the best way of obtaining predicted equations, though in practice, this is not the case. For reasons of logistics, cost and convenience, only well defined population groups are actually studied, which can provide useful information (such as disease prevalence) in addition to the data used to derive the equations. On completion of the survey, individuals having conditions which may impair the function of the lung can be excluded to provide the "healthy" population.

For occupational groups and hospital populations (staff and patients), the recruitment and rejection procedures should include at least a clinical history in a standardized form (ie questionaire). An electrocardiograph, clinical examination and chest x-ray may be added depending on facilities and time available, and of course funds! It should be borne in mind that increasing the number of recruitment criteria will ultimately lead to the rejection of more subjects, which although probably narrowing the limits of normality, may make the study very difficult to complete.

The object of recruitment and rejection criteria is the elimination of persons with clinical conditions which may influence pulmonary function or its measurement. These conditions include lung disease, gross obesity, neuromuscular disorders, cardiac disease, hyperthyroidism and all debilitating disorders. Also of importance is the motivation of the subject to perform the tests, and to provide technically satisfactory data. Poor motivation usually leads to poor results, especially for tests requiring maximal effort, and these need to be excluded.

One particular problem relating to the use of occupational and hospital staffs as the study population, is that the equations derived from these groups are really only applicable to people at work (that is, up to 60 or 65 years) as after retirement inactivity and weight gain will tend to reduce ventilatory function (5) in addition to ageing effects (6).

Having decided on the population to be studied, the next important step is to choose the methods and measurements to be made. Recommendations for the test procedures have been made (1,7) and common sense dictates that these should be followed as far as possible. Of equal importance is the choice of equipment, which should conform to the minimum recommended standards, be regularly calibrated and serviced. Intra- and inter-observer variability should be assessed where necessary, but this may be reduced to some extent by the use of computers for data collection and analysis.

On completion of the study, the data remaining after exclusion of technically unsatisfactory results and subjects who have not met the recruitment criteria need to be analysed to produce the predicted equations. The procedure for deriving normal values entails the determination of both the prediction formulae and the upper and lower limits between which values will be considered as normal. To obtain the prediction equation requires the selection of the predicting variables and the construction of a model for the prediction formulae (8). The distribution of the observations about the predicted equation influences the choice of model since it is desirable that the differences between the predicted and measured values have a normal distribution function.

The model used by many of the predicted equations, including those from the ECCS, is somewhat dubious. A number are not accurate for young adults, or have few subjects over the age of 70. Those that do, seemed to have disregarded the effects of ageing on lung function (6). Thus, the present equations, with few exceptions, will not adequately describe the general population over the age range 16 to over 90 years. As an increasing proportion of society is over 60 years, there is clearly a need for more appropriate equations.

In deriving equations, most workers have used simple regression equations to describe the relations of lung function to height and/or age. Body mass, as noted by Hutchinson (9) in 1846, affects most lung function measurements: lung function increases with mass - the muscularity effect, and decreases with further increase in mass - the obesity effect. Few workers have taken this into account.

To derive optimal equations for describing the data, a comprehensive statistical analysis is required using multiple linear regression analysis (10). The lung function index is the dependant variable, the independant variables being height (H), mass (M), and age (A); quadratic and logarithmic functions of these variables: interaction terms (AxM) between 2 or all 3 of these variables; and other combinations of variables such as the obesity indices $H/\sqrt{M}$ and M/H (11). Having calculated these variables, all possible regression equations should be examined (12) using an efficient algorithm (13). To obtain the ''best'' equation to describe the data, selection criteria need to be applied to determine the degree to which each variable contributes to the equation. These criteria should include 1) all independant variables contributing significantly to the regression; and 2) the standard error of the estimate decreases as a variable is added. Furthermore, it is important to check the residuals of the ''best'' equations (observed minus predicted) for normality and to check that the 'homoscedasticity' (the variation of y for a fixed value of x) is constant. If heteroscedasticity is found (the opposite) then transformation of the data should be applied to correct for this. This thorough analysis may result in rather complex predicted equations (14,15), but with the advent of the departmental computer, this should not cause very great difficulty.

The way in which the equations are used to define the normal limits is also important. For some reason, it has been the tradition to quote a mean predicted value and to assume that the range of normality is ±20% of this value. There is no logical explanation for this, and it certainly has neither physiological or statistical validity.

The implication of the 80% predicted value as the lower limit of normal is that all pulmonary function indices have a variance about the predicted value, which is a fixed percentage of predicted. If a predicted value minus 1 SD, 1.64 SD or 2 SD and the 80% predicted value are close, this occurs by chance and need not imply validity. Generally agreement is not found.
The use of this 80% predicted value is further complicated when lung function measurements are expressed on the report as % predicted. For an individual who is tall and young, a result of 70% predicted will (and should) be regarded as abnormal. For a small old man, 70% predicted is often within the normal limits, and is therefore not abnormal. To compound the problem, if the predicted equation uses two or more variables to obtain the regression line, the 80% lower limit will deviate from statistical validity due to the scatter about the regression line, and the steepness of the line. The 80% lower limit applied to regression lines will result in the limits of normal for short and elderly people deviating more readily from the limits of normal than for tall and young people. The statistically valid limits of normal are parallel to the regression line, whilst the limits of normal using % predicted will deviate from the line as the values increase. This latter situation has been shown to be false in practice (16).

Although this practice is invalid, it is ingrained in many. Although there are better alternatives, Sobol (17) has suggested correcting the 80% lower limit and the use of % predicted to be more statistically valid. This does not appear to have caught on fortunately!

The statistically and physiologically more correct approach is to report a normal range obtained from the predicted value plus and minus 1 SD or more. The choice of 1 SD, 1.64 SD or 2 SD to define the upper and lower limits is difficult. The use of 1 SD has been suggested, since values outside of these ranges will, on statistical grounds, be abnormal. This assumes a normal distribution, and would result in the probability of an incorrect interpretation of 1 in 6. Alternatively, 2 SD will give a better probability of being correct (40:1). This covers approximately 96% of the population, but may result in a reduction in the sensitivity of the tests, thereby failing to identify patients with abnormalities.

The provisionally recommended limits of normal are ±1.64 SD about the mean (1). This range will encompass 90% of a normally distributed population, such that the probability of a result being abnormal approaches close to the accepted level of statistical significance ($P < 0.05$). Thus, on the report form, the normal range for a specific lung function index, for a particular subject can be clearly quoted (18).

In conclusion, the present state of predicted values is such that it is difficult to choose a particular set of equations to suit a given population. In the short term, there appears to be little to be gained from choosing one set over another, although the attempts of the ECCS (1) should not be overlooked, and should probably be used until better equations are available. There is clearly a need for properly conducted, comprehensive, nationwide studies of lung function to be carried out. These should encompass all types of environment (rural and urban), use standardized methods and equipment, and should include ethnic groups. Guidelines are given as to the minimum requirements for such studies (1). On completion, a comprehensive statistical analysis should be performed to derive the best equations to describe the data. Although a multicentre, nationwide study would be a major undertaking, there is most definitely a need for this.

The way of reporting the results does need reconsidering. Whatever index is chosen, the interpretation will only be accurate if those using it know what is meant by sensitivity. If the residual SD is quoted,there is less chance of misinterpretation. Therefore, the ECCS recommendations of ±1.64 SD as setting the upper and lower limits of normal should be employed. The practice of 80% predicted as the lower limit of normal and of % predicted to define the degree of abnormality should be abandoned. Physicians who use these should be encouraged (gently at first) to change, having had the (statistical) error of their ways pointed out to them.

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ASBESTOS INDUCED DISEASE

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Asbestos is a term for a number of naturally occurring fibrous, mineral silicates. Its fire resistant qualities have been recognised for thousands of years. In the 19th century, with the discovery of Canadian and South African deposits, asbestos began to be used in industry on a large scale. Its full potential was realised when methods were developed for spinning and weaving it on textile machinery. Unfortunately, although its hazards to health were suspected at a very early stage, little was done to protect workers from these hazards until quite recently.

Pliny the Younger (61-114 AD) is said to have remarked on the illnesses afflicting slaves who worked with asbestos, but it was not until 1900 that the first case of "asbestosis" was described at Charing Cross Hospital. No case histories appeared in the literature until 1924, but in succeeding years several more reports appeared and in 1930 a review by Wood and Gloyne (1) of 37 cases seen at the City of London Hospital for Diseases of the Heart and Lungs (later to become the London Chest Hospital) resulted in general acceptance of asbestosis as a pathological entity. It was later realised that asbestos caused not only asbestosis of the lungs but also several other diseases, including benign and malignant pleural disease, cancer of the lung and probably cancer of the larynx and gastrointestinal tract.

Imports of raw asbestos fibres into the United Kingdom reached their peak in 1973 but did not decline greatly until after 1979 when the Advisory Committee on Asbestos (2) recommended the gradual substitution of asbestos, a ban on the use of sprayed asbestos and licensing of the asbestos removal industry. Because the latent period between the first exposure to asbestos and the onset of asbestos induced disease is usually more than 20 years in the case of asbestosis and 30-40 years in the case of mesothelioma, (a malignant tumour of the pleura and peritoneum), new cases can be expected to appear in the United Kingdom until well past the turn of the century.

The number of deaths from asbestosis averaged 113 each year from 1978 to 1982 with little variation from year to year. The number of deaths from mesothelioma has been steadily increasing for more than a decade and exceeded 500 in 1983 (Fig. 1). Asbestos induced diseases are under-diagnosed and the true figures are probably considerably higher. Moreover, the role of asbestos in the causation of lung cancer is frequently not recognised and the number of deaths from lung cancer due to asbestos is probably at least twice the number of deaths from mesothelioma.

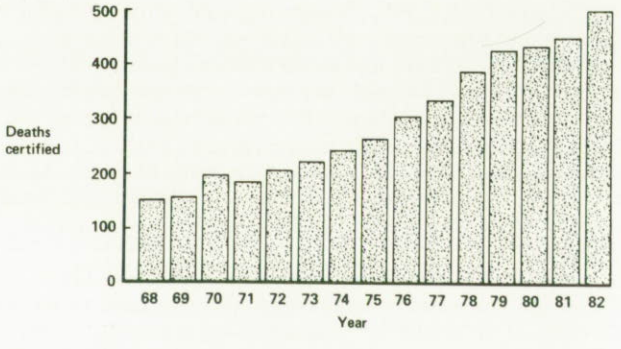

Fig 1. *Deaths due to mesothelioma: a steady increase has occurred.*

Diseases caused by asbestos

Asbestosis

This is a type of interstitial fibrosis of the lungs. It usually presents with breathlessness, initially only on exertion. Cough is occasionally a prominent feature and may reflect involvement of the airways by the disease process. Crackles heard over the lower zones of the lung fields are the most reliable physical sign though they are occasionally absent in early disease. Clubbing of the fingers occurs in about 50% of patients.

The chest x-ray (Fig 2.) commonly shows small, often irregular, opacities most obvious in the lower zones. The pattern is quite variable and sometimes short linear shadows, or diffuse shadows occur. Pleural thickening and/or calcification is present in more than 50% of cases. Radiological changes are an insensitive indicator of the presence of early interstitial lung disease. Broncho-alveolar lavage fluid obtained during fibre-optic bronchoscopy often demonstrates inflammatory activity in the lungs of men with asbestos exposure but no clinical or radiological evidence of asbestosis.

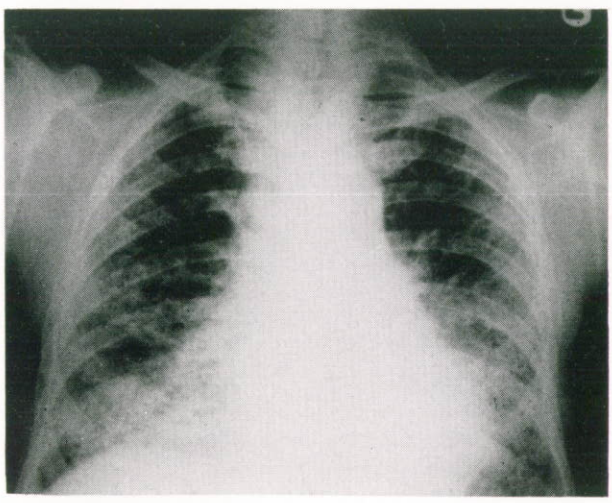

Fig 2. Chest x-ray in asbestosis: irregular opacities are present in both lower zones.

The clinical, radiological and pathological features are very similar to those of cryptogenic fibrosing alveolitis (CFA) and it can be difficult to distinguish between these conditions. The history of substantial asbestos exposure is the most important diagnostic feature. The presence of pleural disease on the chest x-ray is another useful sign because this is often associated with asbestosis but rarely occurs with CFA. Asbestosis is on average less rapidly progressive than CFA, though the rate of progression is variable and this is not a reliable diagnostic feature. If there is doubt about the occupational history, broncho-alveolar lavage may be helpful because asbestos bodies and uncoated asbestos fibres can be identified and quantitated by light and electron microscopy (Fig.3). If there is doubt about the diagnosis a drill or open lung biopsy may be necessary. Trans-bronchial biopsies are usually too small to give useful diagnostic information in this situation.

Asbestosis is usually unresponsive to treatment. There are theoretical reasons for believing that treatment might be effective if it could be instituted at an early stage of inflammatory activity in the lung, before fibrosis has become established. There are anecdotal reports of occasional good responses to corticosteroids and this form of treatment is worth attempting in the minority of patients who appear to have rapidly progressive disease.

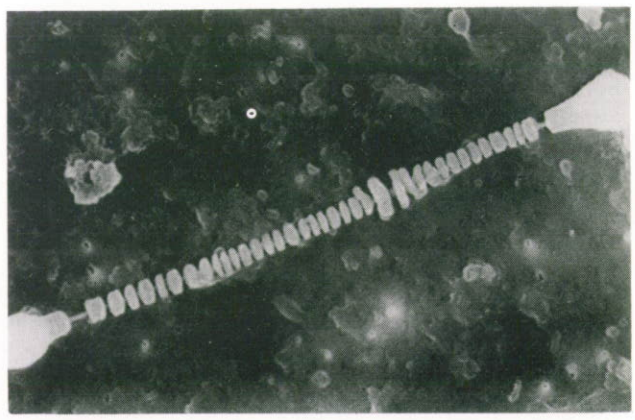

Fig 3. Electron micrograph of an asbestos body showing the coating building up in segments on the asbestos fibre.

Mesothelioma

This is a malignant tumour which affects the pleura, the peritoneum or both. It usually presents with pain in the chest or with pleural effusion causing breathlessness associated with loss of weight and general debility. Chest x-ray (Fig.4) shows irregular pleural thickening often associated with effusion. The diagnosis may occasionally be confirmed by cytology of aspirated pleural fluid and more often by percutaneous pleural biopsy. Where the diagnosis remains in doubt, thoracoscopy under local anaesthetic with biopsies under direct vision may be helpful.

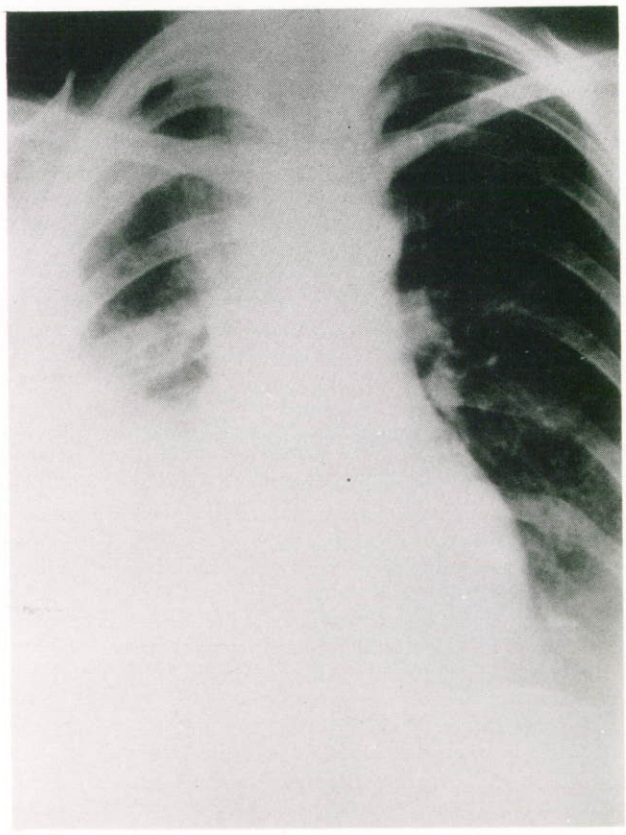

Fig 4. Mesothelioma: malignant disease of the pleura.

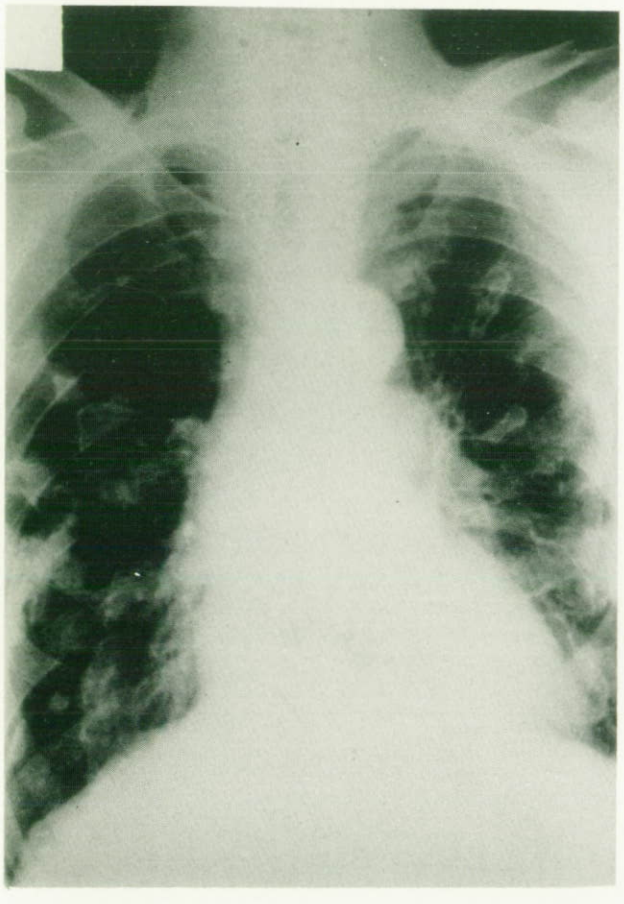

Fig 5. Bilateral pleural plaques.

There is no effective treatment for mesothelioma. Chemical pleurodesis may be helpful if pleural effusion is recurrent. Chemotherapy with presently available agents is almost always useless and its general use is not recommended, though investigations of new regimes in special centres can be justified. Radiotherapy occasionally relieves chest wall pain.

Benign Pleural Disease

The most common manifestation of asbestos exposure is the development of pleural plaques which often become calcified and which are visible on the chest x-rays. These are usually said not to be associated with any symptoms but a few patients with extensive calcified plaques complain of a grating sensation and discomfort in the chest and if plaques are very extensive they can be associated with minor impairment of lung function.

Asbestos also causes diffuse pleural thickening which is usually, but not always, bilateral. This appears on the chest x-ray as a rim of thickened pleura which may eventually cover the whole surface of the lungs. The pleural thickening gradually constricts the lungs causing impairment of their function (Fig.5).

Asbestos can also cause benign pleural effusions which are often recurrent. This entity is frequently not recognised and the patient is said to be suffering from an "idiopathic" pleural effusion if the history of asbestos exposure has not been obtained.

Cancer of the lung and other organs

The risk of lung cancer is increased in people who have had substantial asbestos exposure whether or not they also smoke. It used to be believed that the risk of lung cancer was only present in patients who already had asbestosis and this view is still sometimes put forward. However, in the light of modern knowledge of carcinogenesis it appears more likely that the risk of cancer is a consequence of asbestos exposure per se rather than of lung fibrosis secondary to asbestos exposure.

Evidence is accumulating that asbestos exposure also increases the risk of development of laryngeal carcinoma and possibly carcinoma at all sites within the gastrointestinal tract.

Effects of Asbestos induced diseases on lung function

Asbestos leads to increased stiffness of the lungs resulting in decreased compliance. This leads to impairment of the vital capacity, and reduction in the residual volume, functional residual capacity and total lung capacity. There is usually also a reduction in the gas transfer factor and the gas transfer coefficient. Evidence of airflow limitation is occasionally present. It is usually attributed to cigarette smoking which appears to have been almost universal among asbestos workers, but asbestos can cause disease of the small airways and this may make some contribution to airflow limitation though it is probably minor.

Exercise testing is probably the most sensitive indicator of abnormality in lung function. Even when routine lung function tests give normal results exercise testing may show an increase in ventilation relative to oxygen consumption (Fig.6) and an increase in the alveolar to arterial oxygen tension difference on exercise.

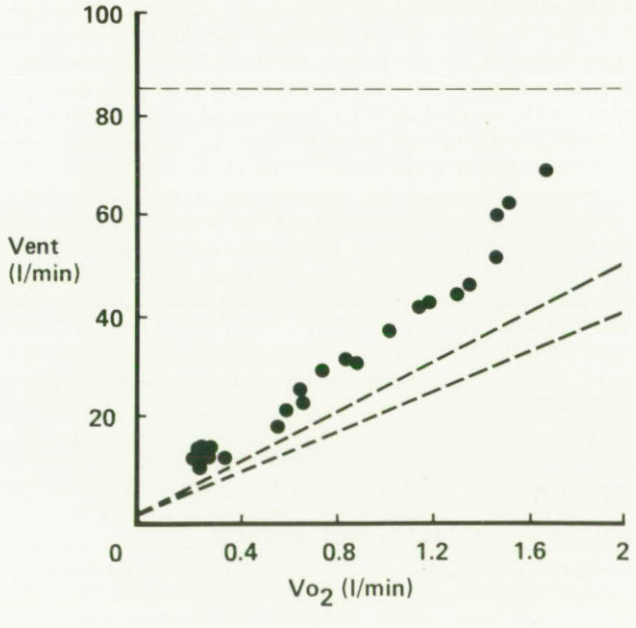

Fig 6. A plot of ventilation against oxygen consumption (VO_2) during exercise by a patient with early asbestosis. Lung volumes and gas transfer were within the predicted ranges. Horizontal dashed line: Maximum predicted ventilation on exercise. Other dashed lines: normal limits.

Diffuse pleural disease also produces a restrictive ventilatory defect with a reduction in residual volume and total lung capacity. If the disease is extensive the gas transfer factor is reduced but the gas transfer coefficient is normal or increased, reflecting constriction of the lungs by the pleural disease rather than disease of the lung tissue itself.

Arrangements for compensation

There are two methods by which a sufferer from an asbestos induced disease can obtain compensation in the United Kingdom. The first is by application to the Department of Health and Social Security through the Pneumoconiosis Medical Panel, recently renamed the Medical Boarding Centre (Respiratory Diseases). The claimant will be examined by doctors working for the DHSS who arrive at a decision as to whether it is more likely than not that the claimant has a prescribed disease. If a prescribed disease is diagnosed compensation is awarded according to the estimated level of disability stated in percentage terms. Conditions for which compensation can be awarded are asbestosis of the lungs; diffuse pleural thickening but only if it is bilateral and causing disability; lung cancer but only if asbestosis or bilateral diffuse pleural thickening is also present; and mesothelioma.

The other method of obtaining compensation is by action at common law against former employers. This generally stands a greater chance of success and produces much larger rewards. A claim can usually be successfully pursued for any asbestos related condition.

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NEBULISER USAGE IN DOMICILIARY PRACTICE

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Abstract

A questionnaire was sent to 250 chest clinics in England, Wales and Scotland to determine the usage of domiciliary nebuliser therapy, the conditions treated and the drugs and apparatus employed. Eighty-three replies were received, Sixty-two physicians used domiciliary nebuliser therapy, 97% of them using it for treatment of asthma, 66% for chronic bronchitis and emphysema, 38% for cystic fibrosis and 23% for bronchiectasis. The drug most commonly used was salbutamol, the dose varying from 1.25 to 50mg. The total volume of solution varied from 1 to 10ml and the nebulisation time from 1 to 20 minutes. Few physicians knew the flow pressure characteristics either of the compressors or the gas cylinders they employed and less than half had a maintenance service. We have therefore found wide variations in the usage of domiciliary nebuliser services, the drugs and doses employed and amount of drug delivered to the patient.

Introduction

Nebuliser therapy is commonly used in hospitals for the treatment of acute exacerbations of airways obstruction. (1) Its use in the home also appears to be increasing both for the management of acute attacks and for regular maintenance therapy. A recent study of hospital usage showed a wide variation in dosage, volume nebulised and flow rates used. (2) We suspected a similar lack of uniformity when nebulisers are used in the home, and we therefore undertook a postal survey of 250 chest clinics in an attempt to determine the extent of the variation.

Method

Questionnaires were sent to 250 consultants with an interest in chest medicine at addresses obtained from "A Handbook of Chest Clinics in England, Wales and Scotland" (British Thoracic and Tuberculosis Association 1977). For convenience we used a marketing company (Boston Consulting

Table 1
Domiciliary Nebuliser Survey

Conditions treated	% of physicians using domiciliary nebulisers	Substances nebulised as % use by physicians for each condition				
		Antibiotics	Disodium cromoglycate	Salbutamol	Terbutaline	Ipratropium bromide
Asthma	97	1.6	40	100	55	40
Chronic bronchitis and emphysema	66	2.3	2.3	100	71	69
Cystic fibrosis	38	65	0	96	52	13
Bronchiectasis	23	69	0	100	84	61

Group Ltd) with a covering letter from one of us (RSEW). We hoped to determine:—

1. How commonly domiciliary nebuliser therapy was used,
2. What drugs were administered and in what quantity and solution,
3. What type of apparatus was used and what knowledge that the physician had of it,
4. What advice was given to the patient regarding the use of nebuliser therapy. (Appendix)

Results

We received only 83 replies (a 33% response rate) and of these six were letters from physicians who felt unable or unwilling to complete the questionnaire. 14 physicians did not use domiciliary nebuliser therapy and one had only two compressors. 62 physicians returned completed questionnaires stating they used nebuliser therapy in the home.

These 62 physicians estimated that in their clinics they saw a total of 52,000 patients with chronic airway obstruction, of which 27,000 had asthma, 23,000 had chronic bronchitis and emphysema and 2,000 had cystic fibrosis or bronchiectasis. Of these patients about 2% used domiciliary nebuliser therapy. This figure correlated well with the physicians' estimate of the number of nebulisers available to them (1097). In 54 instances the physicians' use of domiciliary nebuliser therapy had increased and 50 felt the need for more nebuliser units to optimise their service.

Table 2
Physicians Using Nebuliser Units and Compressors

Nebuliser Units	% of replies
Minineb	53
Acorn	37
Hudson	13
Sandoz	5
Bird	5
Wright	3
Others	16
Compressors	
Medic-aid RTU 4	37
Medix Mini	34
Inspiron	11
Pulmosonic	9
Aerolyser CBI	6
Unknown	6
Others	6

The widest indication for domiciliary nebuliser therapy was asthma (Table 1). The drug most commonly nebulised was salbutamol in all conditions but antibiotics, cromoglycate, terbutaline, atropine and ipratropium bromide were also used (Table 1).

A wide range of nebuliser units and compressors were used (Table 2); 14% of physicians used mouthpieces, 37% used facemasks and 49% used both as a method of administration.

Seventeen physicians used oxygen or compressed gas cylinders instead of compressors, 31 used them on occasions and 14 used compressors only for nebulisation. The flow rates with oxygen cylinders varied from 2 to greater than 10 litres/min and when questioned on the characteristics of the compressor used as a driving source, 56 physicians were unaware of the flow rate and 58 of the no-flow pressure. 31 did not know the flow rate or pressure of the oxygen or gas cylinders they used.

A wide variation was also found in the quantity of drug and solution used and also in the time for which nebulisation was employed (Fig 1). The doses most frequently used were 5mg of salbutamol and either 5 or 10mg of terbutaline. Most commonly 2ml of solution was used but there was a wide variation from 1 to 10ml. Twenty-one physicians advised nebulisation to dryness and 24 advised times between 1 and 20 minutes. Seventeen suggested 10 minutes. The frequency advised varied from 1 to 8 times a day.

Twenty-seven physicians had a maintenance service and 22 an emergency service for the compressors used at home.

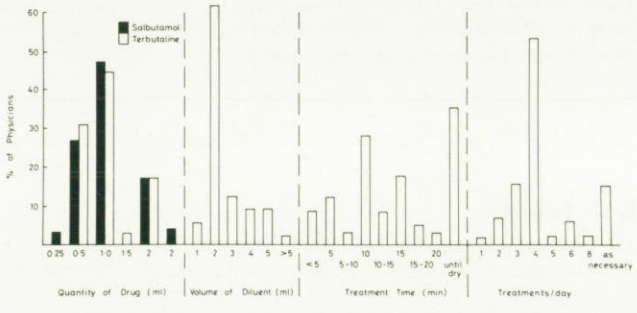

Fig 1: Percentage of physicians (total 62) prescribing: Quantity of drug (ml), volume of diluent (ml), nebulisation time (min) andnumber of treatments/day.

Discussion

The disappointing response rate may have been due to the use of a marketing company, to the length of the questionnaire and the nature of the questions. The low response may bias the results in that the non-responders may well not have used domiciliary nebulisation therapy. As an epidemiological study the results are non-valid, but reveal some interesting points. As in the recent study in hospitals (2), our main finding was the marked variation in dose, solution, administration time and frequency of use. If our respondents are representative of chest physicians in the whole country, the use of domiciliary nebuliser therapy has increased in the last two years and is likely to increase further, at least in the short term. There is also the implication that about 2% of patients with chronic airflow obstruction are being treated with nebuliser therapy in the home. Although most commonly used in asthma and in chronic bronchitis and emphysema, we were somewhat surprised at the frequency of use in bronchiectasis.

There was a wide variation in the dose of salbutamol and terbutaline advised but it is worth noting that the commonest dose of salbutamol was 5mg, which is the same dose most commonly used in hospitals (2). The total volume of solution was also variable although 2ml was the most common and the length of time suggested for nebulisation varied from 1 to 20 minutes. This variation in drug dose, volume of solution and nebulisation time implies a wide

variation in the amount of drug delivered to the patient. This is compounded by the variation in the nebuliser units used, along with the variation in the types of driving source employed.

The majority of our respondents were unaware of the flow and pressure characteristics of the driving source employed. Without this knowledge the most efficient nebuliser cannot be matched to it (3) and the physician will need to rely upon the advice of the distributing company which in our experience is not always appropriate.

The lack of maintenance service for compressors reported by over half our respondents would seem to be a matter of concern. To ensure safe and efficient running of any electrical equipment, some form of maintenance is necessary and the necessary technology should be available in most hospitals (4).

Our survey, despite the poor response, demonstrates the lack of uniformity when nebuliser therapy is used in the home. This means that when nebuliser therapy is used in one area of the country a completely different amount of drug is delivered to the patient than in another area. This lack of standardisation leads to inefficient use of equipment and drugs as shown by our study.

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Appendix — The Questionnaire

1.1. Approximately how many patients with the following diseases have you seen in the past year: asthma, chronic bronchitis/emphysema, cystic fibrosis, bronchiectasis, other diseases resulting in airways obstruction?

1.2. Has the use of nebulisation therapy increased or decreased in the last 2 years? In hospital ..... at home .....

1.3. How many compressors does the unit have in stock?

1.4. As far as you know are there any compressors elsewhere in the hospital?

1.5. Would you prefer to have more nebulisers in order to provide optimum service?

2.1. Do you use domiciliary nebuliser therapy?

2.2. Approximately how many patients use such therapy?

2.3. Of the following patient types which do you treat with domiciliary nebulisation: asthma, chronic bronchitis/emphysema, cystic fibrosis, bronchiectasis, others?

2.4. Which drugs do you use in nebulisation therapy of the above diseases: Antibiotics, Intal, Ventolin, Bricanyl, Atrovent, Atropine?

2.5. Which of the following nebuliser units do you use: acorn, oem, sandos, minimeb, other?

2.6. What make of compressor(s) do you use?

2.7. Do you use oxygen or compressed gas cylinders:—
instead of compressors,
in some cases,
not at all?

2.8. If in use what are the flow/pressure characteristics of such cylinders?

2.9. What are the flow/pressure characteristics of the compressors that you use?

2.10. Do you use facemasks or mouthpieces or both?

2.11 What quantity of solution do you advise: 2ml, other?

2.12. What quantity of the following do you advise: ventolin, bricanyl?

2.13. How long an administration period do you instruct your patients to take?

2.14. How frequently do you advise your patients to use the nebulisers?

2.15. Do you have a maintenance service and/or an emergency service?

BOOK REVIEWS

Best and Taylor's PHYSIOLOGICAL BASIS OF MEDICAL PRACTICE

Editor — *J B West* 11th edition
Williams and Wilkins, 1985. 1340 pages
ISBN 0-683-08944-7. Price £35.50 (hard-back)

The majority of patients tested in lung function laboratories are those with diseases of the respiratory system. There are, however, occasions when patients with disorders of other systems need to be tested. Although many texts on respiratory physiology will indicate the likely or possible respiratory pattern, no text has the space to explain fully the physiological mechanisms by which the disorders of other systems affect lung function. Indeed, some respiratory texts do not explain the physiological mechanisms occuring in respiratory diseases. There is, therefore, a need for texts which explain medical conditions of any system, in terms of the changes in the physiology.

Best and Taylor's text was first published in 1939, with the aim of serving the link between the laboratory and the medical clinic. The principal aim of the book is to emphasize the clinical relevance of physiology, whilst at the same time provide the appropriate scientific background.

The text is divided into nine sections — 1) General Physiological Processes, 2) Cardiovascular System, 3) Blood and Lymph, 4) Body Fluids and Renal Function, 5) Respiration, 6) Gastro-intestinal system, 7) Metabolism, 8) Endocrine system and 9) Neurophysiology. The section editors, and other contributors, are all experts (from the States) in their respective fields. Each of the sections follows the broad outline of covering the anatomy of the system, including its blood and lymph supply, thus providing the necessary background to the understanding of the physiological and biochemical processes of the system which follows.

The sections vary in length, section 9 not surprisingly being the longest. Each of the sections is presented in a clear and concise manner, and within each section, the chapters follow logically. Throughout, the text is liberally interspersed with appropriate illustrations, either as line drawings, or as photographs. At the end of each section, there is a considerable reference list, for those who wish to delve a little deeper into particular aspects. The index is well laid out, and there is little difficulty in finding specific points. This is rather important for a book of this size.

The editors have done a superb job in bringing together this excellent book, and in a manner that makes it readable by expert and non-expert alike. At its extremely reasonable price, it will hopefully find its way onto many departments' bookshelves as a necessary and useful reference guide to physiology and medicine.

Adrian Kendrick

VENTILATION/BLOOD FLOW AND GAS EXCHANGE

J B West 4th edition
Blackwell Scientific Publications, 1985.
119 pages
ISBN 0-632-01504-7. Price £6.50 (soft cover)

This short monograph is the second in the series of books on respiratory physiology by Professor West, and follows on from the text — Respiratory Physiology — the essentials.

Its purpose is to explain how the inequality of blood flow and of ventilation in the lung interferes with gas exchange, and so how the lung is able to take up oxygen and excrete carbon dioxide. The mechanisms are important to the understanding of arterial hypoxaemia — a problem often encountered on medical wards.

The first chapter opens with a discussion of the movement of oxygen from the atmosphere to the tissues via the lungs, and briefly introduces the various causes of arterial hypoxaemia. Chapter 2 then goes on to discuss the distribution of blood flow and ventilation in the normal upright lung, and shows how the pattern of ventilation-perfusion ratio inequality is derived. Chapter 3 uses this information to deduce the differences in regional gas exchange, by means of a simple oxygen-carbon dioxide diagram. Chapter 4 compares normal lung to impaired lung, thus showing how overall gas transfer may be impaired. Methods for the measurement of the ventilation-perfusion ratio inequality are discussed in chapter 5. The final chapter — a new addition — discusses recent work on ventilation-perfusion ratios in various diseases.

Throughout, the text has been updated from the previous edition, and is highly readable. Mathematical formulae are kept to the minimum. The two most important equations — the ventilation-perfusion ratio equation and the alveolar gas equation are presented in an appendix.

This small book is an important text for all technicians to read, in order to understand the concepts of ventilation/blood flow and gas exchange.

Adrian Kendrick

GUIDELINES FOR CONTRIBUTORS TO BREATH

Breath is the Journal of the Association of Respiratory Technicians and Physiologists (ARTP) and is published three times a year. Both members of the Association and non-members are invited to submit original articles, reviews, or case reports in the field of respiratory medicine, physiology or technology; articles relating to other disciplines are also welcome. Two copies should be submitted to the Editor, Dr. D C S Hutchison, Department of Thoracic Medicine, King's College School of Medicine, Denmark Hill, London SE5 8RX. Articles are accepted on the understanding that they may undergo editorial revision.

The Editor would be grateful if contributors would observe the following guidelines:

Material should be typed in double spacing on one side of the paper only and authors should keep one copy. Original articles should follow the usual format of (in order), a summary of 200 to 250 words, introduction, methods, results and discussion. Each table should be typed on a separate sheet and numbered in order of appearance. Figures should be submitted as original art-work in black on a white background or as half-plate glossy prints, all marked on the reverse with the name of the first author and the figure numbers in order of appearance. Legends to figures should be typed on a separate sheet.

Units and Symbols: This journal uses SI units only (blood pressure should be given in mm Hg). Abbreviations and symbols should be defined at first appearance. Acceptable symbols, units and abbreviations for lung function indices are given in Bull Europ Physiopath Respir, Suppl 5, 19, 52-61, 1983.

References: References should be numbered in the text in brackets in the order in which they first appear eg. (12). The reference list should be typed in the same order on separate sheets. References should be typed as in the following examples (Abbreviations as given in Index Medicus):

Original Article:

1. Ogilvie C M, Forster R E, Blakemore W S, Morton J W (1957). A standardized breathholding technique for the clinical measurement of the diffusing capacity of the lung for carbon monoxide. J Clin Invest 36 1-17.

Book:

2. Crofton J, Douglas A (1981). Respiratory diseases: 3rd Edn. Chap 15, 265-77. Blackwell Scientific Publications. Oxford.

Section in Edited Book

3. Morgan P K (1983). Physical Gas Analysers. In: Measurement in Clinical Respiratory Physiology. Eds. Laszlo G, Sudlow M F. Academic Press, 113-30.

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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 arises in job advertisements appearing in *Breath*.

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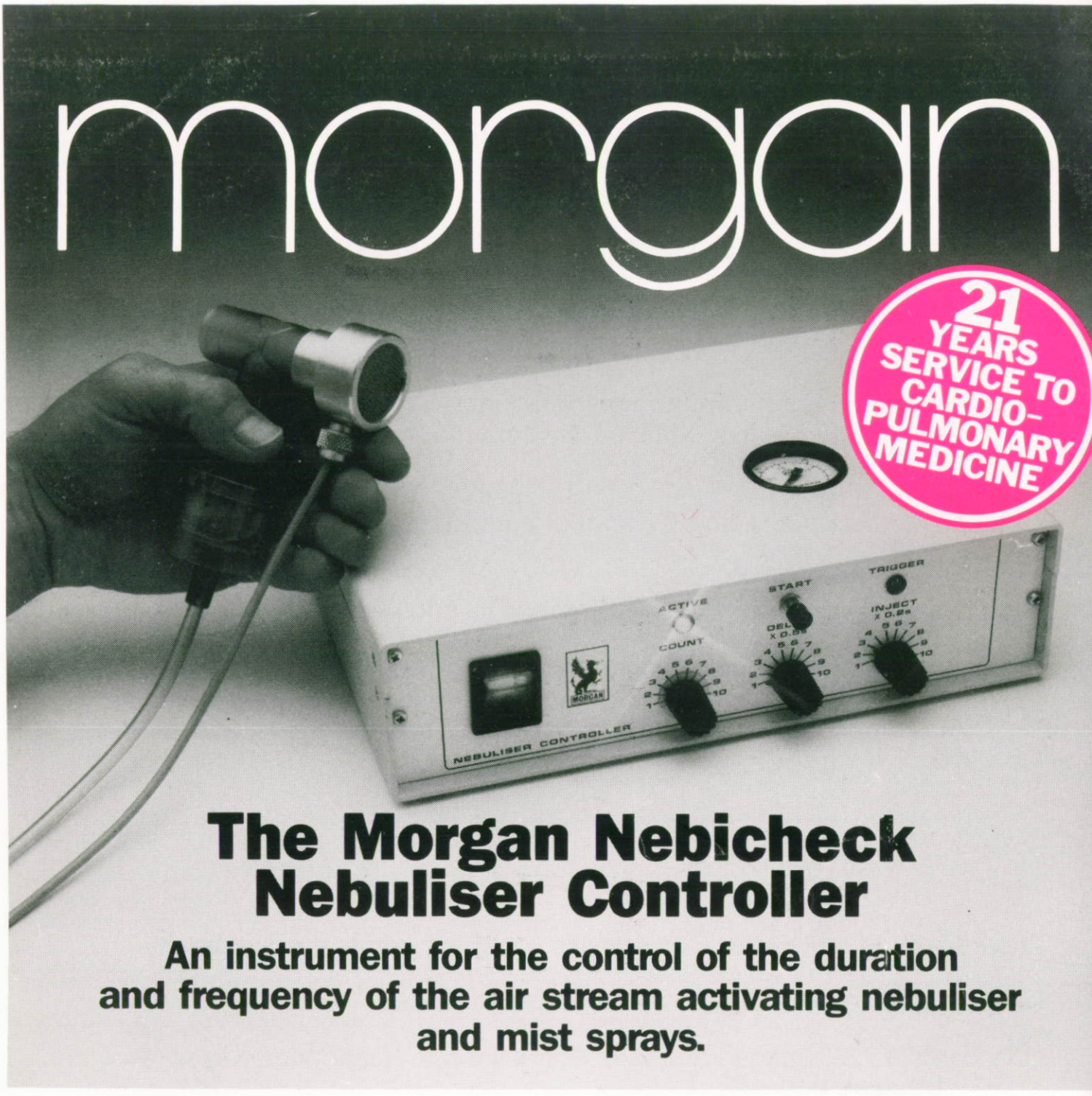

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