ASSOCIATION OF RESPIRATORY TECHNICIANS AND PHYSIOLOGISTS


ISSUE NO. 25 JULY 1985

# BREATH

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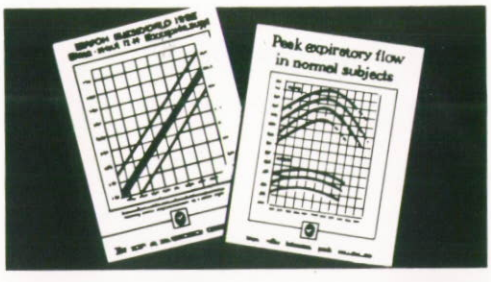


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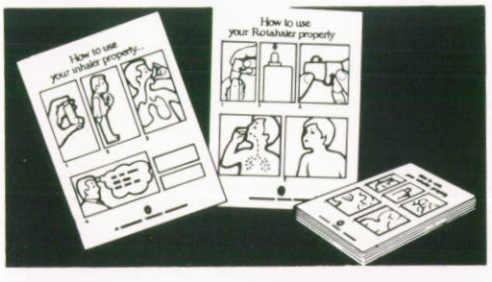


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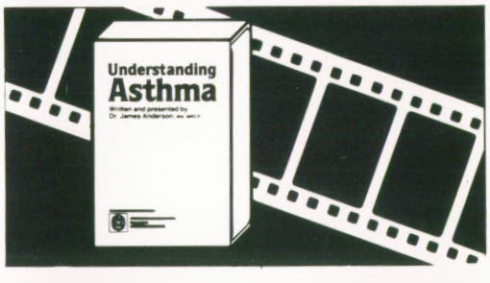


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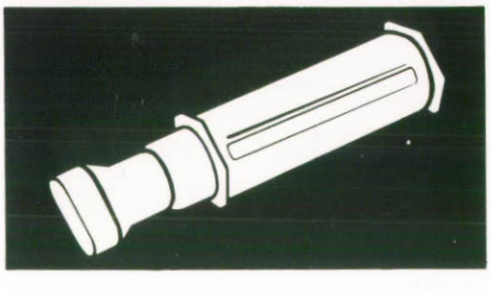

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# TENTH ANNIVERSARY CELEBRATION

## AGM 4th and 5th October 1985

## Papworth / Hinchingbrooke Hospitals

### Friday (Papworth)

4.00 pm: Tea followed by computer workshop.

Evening: Sherry, poster display and commercial demonstration followed by dinner with guest speaker.

### Saturday (Hinchingbrooke)

9.30-12.30: Exciting programme of scientific papers.

12.30-2.00: Lunch and commercial exhibitions.

2.15-4.00: A.G.M. followed by tea.

Limited hospital accommodation is available on Friday night. Early reservations are recommended. Contact Sally Gough in Respiratory Physiology, Papworth Hospital, Papworth Everard, Cambs. Tel: 0480 830541.

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# LEGIONNAIRES' DISEASE

*Gisella Borzone*
Department of Thoracic Medicine, King's College School of Medicine, London SE5.

In the year 1976, a severe form of pneumonia due to a previously unidentified organism struck a number of people attending a conference of the American Legion in Philadelphia. Because of this association and its predilection for the lung, the new bacteria was called *Legionella pneumophila*; after analysis of stored sera and environmental samples, the organism was subsequently found to be the cause of a number of previously obscure outbreaks of disease.

Legionella has again been in the news after the recent epidemic in Stafford, where there was a substantial mortality rate. The source of the outbreak has not been positively identified in spite of considerable investigation, but suspicion rests on the cooling tower of a hospital air-conditioning system, from which the organism has been isolated.

### Clinical Features

The organism may manifest itself in a number of ways (1):

a) *Asymptomatic infection*: In certain areas elevated antibody titres may be found among healthy individuals.

b) *Mild febrile illness*: The disease may present as a self-limiting illness without pneumonia, as in the case of the 1968 outbreak in Pontiac, Michigan, which was characterized by an acute short-lived moderately severe influenza-like syndrome, with fever, myalgia and headache.

c) *Severe pneumonia with involvement of other systems: (Legionnaires' disease).* The incubation period is between two and three days and the disease begins with prodromal symptoms such as malaise, diffuse myalgia, headache and lethargy. 12-48 hours later, there is a sudden onset of high non-remittent fever (39-40°C), recurrent shaking chills and severe prostration. About 20% of the patients have gastrointestinal symptoms including nausea, vomiting and watery diarrhoea. During the second or third day, a dry cough develops and 20-40% of patients may have a minor haemoptysis or pleuritic pain. Some have confusion and disorientation out of proportion to the degree of fever or hypoxaemia, with no localizing neurological signs or CSF abnormality. In the chest, only fine inspiratory rales can be heard at the onset, but as the disease progresses, all the signs of consolidation may develop (2).

The prognosis in the severe form is variable. In most cases, the fever continues until the institution of therapy or spontaneous resolution, which occurs at 8-10 days. In a small number of patients, the condition progresses to a chronic inflammatory phase with respiratory failure and some become chronically disabled, though whether as a result of the disease itself or of long term artificial ventilation is not known. The mortality rate among those severely affected enough to be admitted to hospital, is about 15% but may reach 50% among patients whose immune response is compromised.

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## Laboratory findings

There is a mild to moderate leukocytosis and elevated erythrocyte sedimentation rate. Some patients have mild abnormalities in liver function, about 10% have microscopic haematuria and 15-40% have a small pleural effusion. Hyponatraemia and hypophosphataemia may also be found in some patients (2).

## Radiological findings

In 70% of cases, the initial involvement is unilateral, commonly with poorly marginated round opacities, either centrally or peripherally located and diffuse patchy bronchopneumonia. As the disease progresses, peripheral shadows enlarge and become lobar in extent. Radiological resolution usually lags behind clinical recovery. There is radiographic improvement within two weeks of treatment, but clearing is usually delayed.

## Morbid anatomy

The pathological features of Legionnaires' disease are limited to the lungs, which show different degrees of consolidation and fibrinous pleuritis with or without pleural effusion.

Microscopically there is an extensive exudation of proteinaceous fluid and inflammatory cells into the alveoli (polymorphonuclears and macrophages) (1). A distinctive feature of this pneumonia is the extensive lysis of inflammatory cells with accumulation of nuclear debris and fibrin. 50% of cases show 'hyaline' membranes.

## Microbiology

Legionella pneumophila is a Gram negative slowly growing obligatory aerobe that was difficult to identify because of its growth requirements in culture and its failure to stain adequately with the usual techniques. It has a narrow optimal pH range (6.9-7) and temperature requirement (35°C) and grows best in an atmosphere of 2.5% $CO_2$. The organism requires cysteine and ferric salts and the best culture medium is charcoal yeast extract agar, with the plates incubated at 35°C in 5% $CO_2$ or in candle jars. Microscopically, it is a small rod 2-3$\mu$m in length producing in culture a soluble brown pigment that fluoresces under a Wood's lamp. It is catalase positive and causes a weakly positive oxidase reaction.

Several exotoxins as well as an endotoxin have been described, but none appears to be potent enough by itself to produce such a severe disease or to explain its multisystemic manifestations.

There are eight known pathogenic serogroups of Legionella pneumophila and six additional species of the same genus have been discovered (Table 1) (3). These are also Gram negative bacteria resembling Legionella pneumophila in cultural characteristics and ecology, but with distinct antigenic and genetic properties. These newly discovered species can also cause respiratory infection in humans and have been isolated during searches for L. pneumophila, but little else is known about them.

The Centre for Disease Control in Atlanta, USA, has reported (4) that of the known Legionella species, L. pneumophila is the most common cause of pulmonary infection in the group of patients studied; all the L. pneumophila serogroups combined are responsible for 80-85% of the Legionella infections diagnosed by the direct fluorescent antibody technique.

**TABLE 1**

**Species of the genus Legionella (3)**

| *Species* | *Number of serogroups* |
|---|---|
| L. pneumophila | 8 |
| L. micdalei | 1 |
| L. bozemani | 1 |
| L. dumoffi | 1 |
| L. gormanii | 1 |
| L. longbeacheae | 1 |
| L. jordanis | 1 |

## Diagnosis

When the diagnosis is suspected on clinical grounds, it can be confirmed by several methods(6, 7):

a) *Culture* of the bacterium is selective media, from sputum, transtracheal aspirate, bronchoscopic washings or pleural fluid. This takes approximately 10 days.

b) *Immunological methods:*

i. Demonstration of a fourfold or greater rise in serum antibodies to Legionella, using the indirect fluorescent antibody technique, with polyvalent antigens. Because antibody titres rise slowly in some patients, two samples are needed, one at presentation and the other 21 days later.

ii. The bacterial antigen can be detected in respiratory secretions or tissues by the highly specific direct immunofluorescent antibody technique. A different antibody is needed for each serogroup.

Although individual clinical features may not be sufficiently characteristic to distinguish Legionnaires' disease from other types of acute pneumonia, the clinical profile shown in Table 2 may be helpful (1).

**TABLE 2**

**Main clinical features of Legionnaires' disease**

1. High fever with rigor and relative bradycardia
2. Early gastrointestinal symptoms
3. Severe myalgia
4. Microscopic haematuria and liver function abnormalities
5. Toxic encephalopathy
6. Non-productive cough with absence of bacterial pathogens on Gram stain or culture by conventional methods.
7. Progression from patchy bronchopneumonia to lobar and multilobar consolidation
8. Prompt and dramatic response to treatment with erythromycin.

## Treatment

Legionella pneumophila produces a beta-lactamase which inactivates cephalosporins and penicillins (1). Retrospective analysis and clinical experience support erythromycin as the drug of choice in this disease. Its empirical use in the Philadelphia outbreak and in other epidemics, lowered the mortality rate considerably in patients who were treated with erythromycin (13% mortality) compared with those who did not receive this treatment (55% mortality) (1). In culture and in animal studies, several other drugs are active against L. pneumophila but erythromycin and rifampin are the most effective.

Erythromycin should be given intravenously for the first few days to severely ill patients and should be changed to the oral route when there is evidence of clinical response. For the less ill patients, oral therapy is sufficient and the recommended dose is 500 mg to 1 g 6 hourly. Generally the patient feels better within a few days of the start of the therapy although pulmonary infiltrates on the chest xray may progress during that period. The treatment should be given for at least three weeks to prevent relapses.

## Epidemiology

Although Legionella has been isolated from different types of water and soil in several epidemics, knowledge of its distribution in nature is limited. There is no evidence that ingestion of the organism causes disease, but aerosolization of contaminated water during showering for example, leads to inhalation of potentially infectious droplets. There is good epidemiological evidence that airborne spread of L. pneumophila from the environment is a major mode of dissemination. No person-to-person transmission has been documented. Contaminated water has been found in air conditioning cooling towers, evaporative condensers, shower heads and nebulizers.

*Predisposing factors (5)*

*a) Environmental risk factors*

1. Outbreaks of the disease are more likely to occur in large buildings such as hotels and hospitals.
2. The common modes of spread from the environment are contaminated water fixtures and heat-exchange apparatus.
3. The cases cluster in summer, but can occur all the year round.

*b) Host risk factors:*

1. The disease is commoner in middle aged and elderly people and in those with underlying chronic cardiopulmonary disease, chronic renal failure or diabetes. Underlying chronic disease has been present in 86% of patients in several epidemics.
2. The disease is commoner in men than women, but is rare in children.
3. People with immunosuppression from primary disease or drugs that affect cellular immunity are greatly predisposed.

Several epidemics have been brought under control by:
— repairing defective evaporative condensers in air conditioning systems;
— changing shower fixtures;
— temporary hyperchlorination of the water system. When cooling tower water has been treated with chlorine containing compounds, bacteria have disappeared in 24-48 hours.
— intermittent heating of water to levels above those usually maintained.

Serological surveys using the direct fluorescent antibody technique have shown that less than 5% of healthy individuals in the USA have high titres against Legionella. However, some more geographically restricted surveys have given different results; in Michigan, for instance, where the microhemagglutination technique was used, there was a 22% overall prevalence of antibodies in asymptomatic subjects, suggesting that the infection may be endemic in certain areas.

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# TRANSCUTANEOUS OXYGEN MEASUREMENTS IN THE ADULT PATIENT

*B J Gray*
Department of Thoracic Medicine, King's College Hospital Medical School, London SE5

The measurement of arterial oxygen tension ($PaO_2$) is a widely used and important test of oxygenation both in clinical patient monitoring and in the pulmonary function laboratory. Arterial blood is obtained by arterial puncture or indwelling cannula, but measurement of $PaO_2$ obtained in this discontinuous fashion has the serious disadvantage that critical changes in $PaO_2$ occurring over short periods of time may be missed. Arterial cannulation is, furthermore, an uncomfortable procedure and not without the risk of long term complications (1). A keen interest has therefore been taken in the development of safe and accurate non-invasive methods of monitoring arterial $PaO_2$.

Two non-invasive methods of assessing $PaO_2$ have received the most attention. Oxyhaemoglobin saturation ($SaO_2$) can be monitored using a fibre-optic ear oximeter, and transcutaneous oxygen tension, $tcPO_2$ (or more correctly skin surface oxygen tension) can be monitored using oxygen sensitive electrodes.

The ear oximeter is a rapidly responding non-invasive measure of oxygen saturation but $PaO_2$ has to be derived from the oxy-haemoglobin dissociation curve, by measuring (or assuming) arterial pH and $CO_2$ tension. Its accuracy in monitoring trends in $PaO_2$ above $SaO_2$ of 80% is limited because the oxy-haemoglobin dissociation curve in this area is relatively flat (2). It is also inaccurate when the blood contains excess bilirubin or carboxyhaemoglobin.

## Measurement of transcutaneous oxygen tension

Transcutaneous oxygen electrodes measure the oxygen tension at the skin surface. A flux of oxygen across the skin was first described by Von Gerlach in 1851 (3). Skin surface oxygen is almost zero at normal body temperature, but because the skin is a thermoregulatory organ, its complex capillary circulation can become greatly over-perfused in relation to the skin's metabolic needs. Heating of the skin results in opening of many previously closed capillaries, 'arterialisation' of blood within the capillaries and an increased flux of oxygen across the epidermis, so that the skin surface oxygen tension approaches arterial $PO_2$ (4).

Further advances in this method of gas analysis were limited until the introduction of the Clark polarographic electrode for the direct measurement of $O_2$ tension (5). The polarographic electrode (Fig. 1) is an electrical cell, consisting of a metal cathode (usually platinum) and a metal anode (usually silver), separated by an electrolyte solution and separated from the gas under investigation by a gas permeable membrane. A constant polarisation voltage of about -650mv is applied across the cell, and current is generated by the diffusion of molecular oxygen across the permeable membrane into the cell electrolyte and its reduction at the platinum cathode. The currents generated are extremely small ($10^{-11}$ A per mm Hg) but are proportional to the concentration or tension of oxygen present. Huch et al (6) developed a miniaturised Clark cell with a thermostatically controlled heating coil built into the anode and this is the basic form of all oxygen skin electrodes used today. Fig. 2 shows the Radiometer E5243 transcutaneous oxygen electrode, which consists of a thin platinum electrode (25$\mu$ in diameter) and a ring shaped silver anode into which is incorporated a thermostatically controlled heater, capable of heating the underlying skin to a pre-selected temperature up to 45°C. The electrode is covered by a thin oxygen permeable polypropylene membrane and is applied to the skin on a thin layer of contact fluid consisting of deionised water. The heater within the electrode heats the underlying skin, causing an enormous increase in dermal capillary flow. Oxygen diffusion across the skin increases, and oxygen diffuses across the membrane into the electrode where reduction at the cathode produces a current which is proportional to the $tcPO_2$. The current is amplified and displayed continuously in digital form or on a chart recorder.

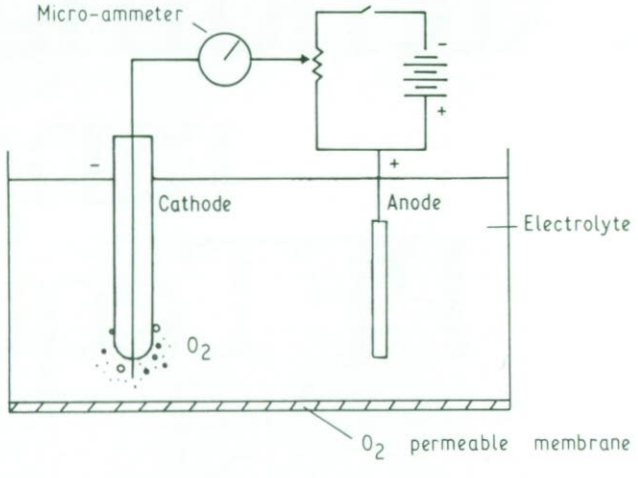


***Fig. 1.*** *Diagram of a polarographic oxygen electrode. A voltage of -650 mv is applied across the cell and the current generated is proportional to the oxygen tension.*

The initial work using this apparatus was almost exclusively in newborn infants and $tcPO_2$ was shown to be closely related to $PaO_2$ in these patients (7). In adults, the increase in dermal and epidermal thickness together with greater oxygen consumption within the skin, change the relationship between $tcPO_2$ and $PaO_2$. While excellent correlation between $tcPO_2$ and $PaO_2$ has been reported in adults (8,9), the scatter of results in these studies show that $tcPO_2$ cannot predict $PaO_2$ with consistent accuracy when in vitro methods are used to calibrate the skin electrode, especially in patients with circulatory insufficiency.

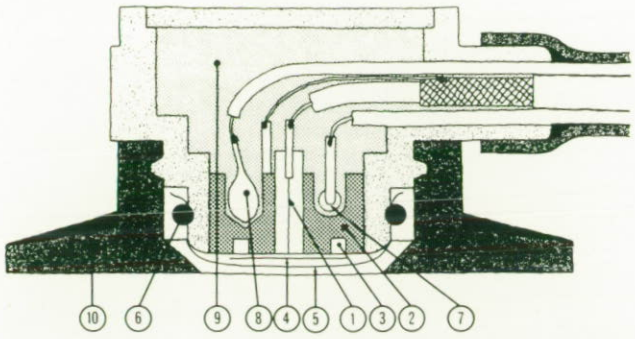


*Fig. 2. Cross section of Radiometer E5243 $tcPO_2$ electrode. 1. 25μ platinum cathode. 2. Silver anode. 3. Electrolyte chamber. 4. Cuprophane spacer. 5. Oxygen permeable membrane. 6. O-ring securing membrane. 7. Heating element. 8. NTC resistor, for measurement and control of electrode temperature. 9. Epoxy resin. 10. Fixation ring; the electrode is attached to the skin by means of a self-adhesive ring. Reproduced by kind permission of Radiometer Company, Copenhagen.*

## In vivo calibration

We have investigated the in vivo characteristics of a Radiometer E5243 oxygen electrode using an in vivo method of calibration (10). This type of electrode is rapidly responding, the in vitro 90% response time being about 11 seconds. We studied 14 patients in an intensive care unit and calibrated the electrode in vivo, that is to say, after an initial period of stabilisation of about 15 minutes we adjusted the $tcPO_2$ value to the $PaO_2$ of a single arterial blood sample by altering the sensitivity gain on the oxygen monitor. By altering the inspired oxygen concentration in each patient we were able to obtain a range of $PaO_2$ values between 50 and 120 mm Hg. There was an excellent corelation between the $tcPO_2$ and $PaO_2$ (Fig. 3), the relationship being:

$$tcPO_2 = 0.98\ PaO_2 + 1.6 \text{ mm Hg.}$$

($r = 0.98$: $P<0.001$: 95% confidence limits ± 6.6).

This study showed that $tcPO_2$ could accurately and consistently be used to predict $PaO_2$ over a wide range of values when this in vivo calibration technique was used.

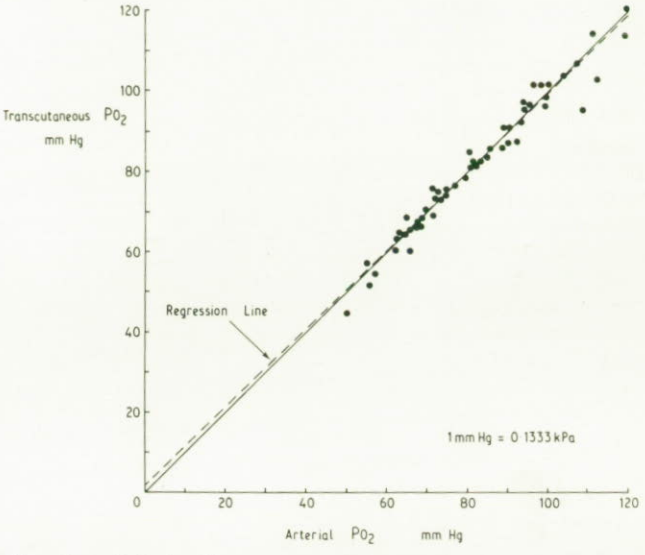


*Fig. 3. Relationship between $tcPO_2$ and $PaO_2$ in patients in an intensive care unit. An in vivo calibration procedure was used (see text).*

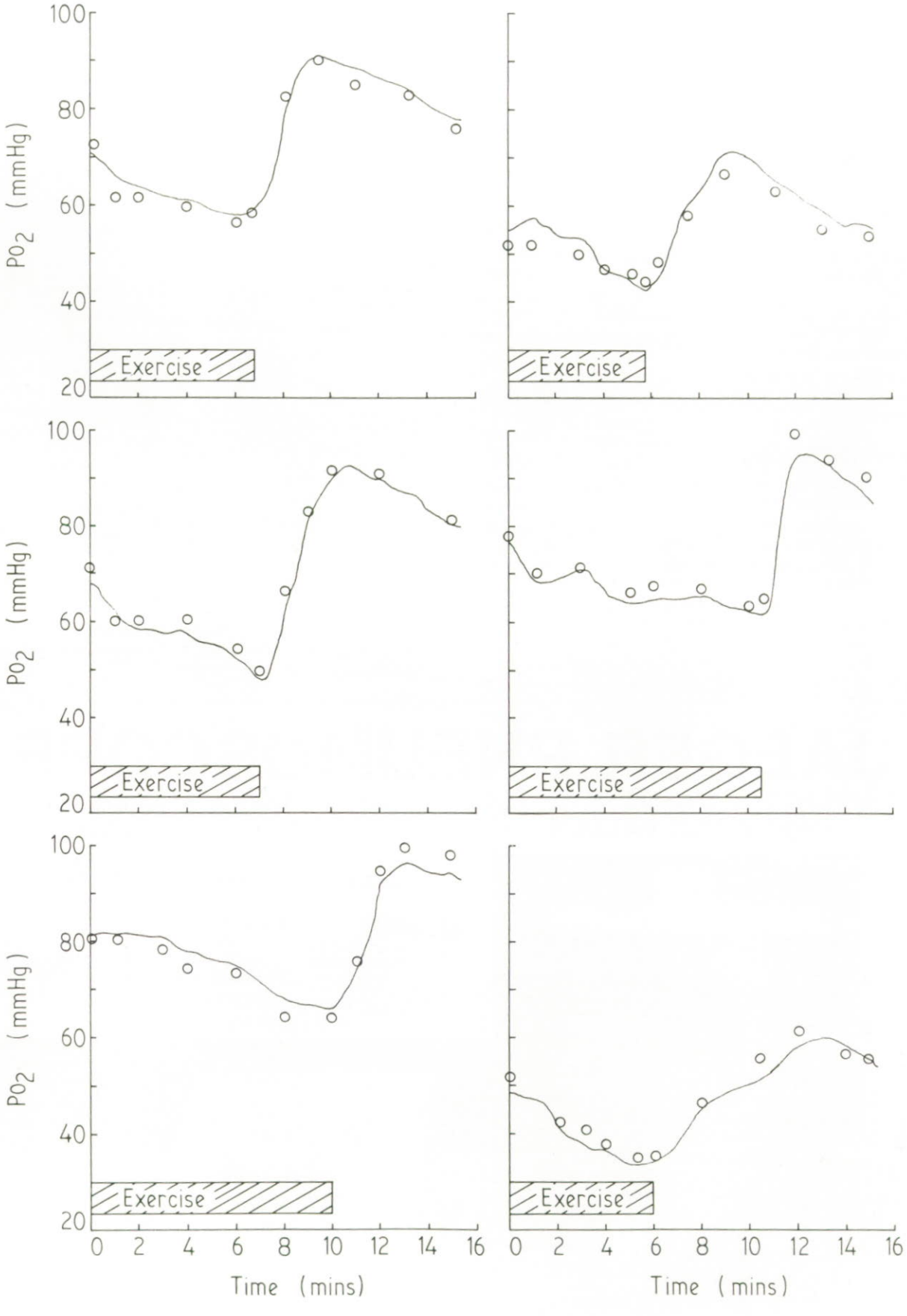


***Fig. 4.*** *Simultaneous measurements of $tcPO_2$ and $PaO_2$ in six emphysematous patients during progressive exercise tests. The workload was increased to maximum by 10 watts every minute. Continuous line: $tcPO_2$. Circles: $PaO_2$.*

## Exercise

A great deal of useful information can be obtained from the changes in $PaO_2$ that occur during exercise, particularly in patients with chronic airflow obstruction or interstitial lung disease (11). We investigated the ability of the Radiometer transcutaneous oxygen electrode to monitor changes in $PaO_2$ accurately during unsteady state exercise in six patients with chronic airflow obstruction (12). The electrode was calibrated in vivo before each study and changes in $tcPO_2$ (measured continuously) gave an accurate picture of $PaO_2$ changes in exercise (Fig. 4). Sixty two simultaneous measurements of $tcPO_2$ and $PaO_2$ were made in six patients with emphysema and $tcPO_2$ and $PaO_2$ were shown to be closely related by the equation:

$$tcPO_2 = 0.975\ PaO_2 + 1.1 \text{ mm Hg.}$$

$$r = 0.98{:}95\% \text{ confidence limits} \pm 3 \text{ mm Hg.}$$

This study showed that in intrinsically unstable conditions such as exercise, $tcPO_2$ responses could accurately reflect the rapidly changing $PaO_2$.

## In Vivo Response

The allegedly slow response characteristics of the skin electrode has been seen in the past as one of its major disadvantages when compared with the ear oximeter. The previously described exercise study refutes this argument but we have formally compared the response characteristics of the ear oximeter, the Radiometer transcutaneous electrode and end-tidal gas tensions, in normoxic and hypoxic conditions, at rest and during exercise in normal subjects. In a study comparing the response times of a $tcPO_2$ electrode and ear oximeter, it was shown that both responded to changes in inspired $O_2$ significantly more slowly than end-tidal measurements, but that during exercise there was no significant difference between the response characteristics of the ear oximeter and $tcPO_2$ electrode (13).

## Limitations

Transcutaneous oxygen monitoring has ideal characteristics for non-invasive assessment of oxygenation. What then are the major limitations of this technique? There is no doubt that there are serious discrepancies between $tcPO_2$ and $PaO_2$ in shocked or hypothermic patients (14) and $tcPO_2$ is therefore an unsatisfactory indicator of $PaO_2$ in patients with poor peripheral perfusion, whether due to haemorrhagic shock (15) or to cardiogenic shock (16). The other limitation of the method is the need to heat the underlying skin to between 43 and 45°C in order to ensure maximum hyperaemia and arterialisation of blood. At 45°C the electrode may only be left in situ for 4 to 5 hours on adult skin before thermal damage begins, resulting in inaccurate results and blistering. It may be possible to limit these effects by moving the electrode frequently or using in vivo calibration techniques at lower electrode temperatures, but further work is required in these areas. Use of the electrode in lower temperature ranges would allow continuous attachment to the skin for twelve hours or more without the risk of thermal damage, and thus $tcPO_2$ measurements would become useful in more prolonged studies, such as the detection of hypoxaemia during sleep.

## The Future

Little mention has been made in this short review of transcutaneous $CO_2$ measurements and indeed this subject requires a review of its own to do it justice! Suffice it to say that if accurate information about $PaCO_2$ could be obtained from transcutaneous $PCO_2$ ($tcPCO_2$) measurements, then this would add enormously to the value of simultaneously obtained $tcPO_2$ measurements in the physiological laboratory. It has been shown that $tcPCO_2$ can accurately reflect $PaCO_2$ (17) and that it is feasible to combine both a $tcPO_2$ and a $tcPCO_2$ sensor in a single compact electrode head (18). The main areas of advance in the future will be in the direction of continued reduction in weight and size of monitoring and recording equipment with a consequent increase in its portability, opening up the exciting prospect of continuous ambulatory transcutaneous gas monitoring.

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# COMPUTERISED ANALYSIS OF VITALOGRAPH CURVES

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## Summary

The analysis of forced expiratory volume (FEV) curves obtained on a 'Vitalograph Model S' spirometer is subject to certain inaccuracies of measurement; in particular, it can be difficult to obtain reliable measurements of the peak expiratory flow rate (PEFR) and forced mid-expiratory flow (FMF). By connecting a BBC microcomputer to the Vitalograph, it is possible to obtain these and other parameters quickly and accurately.

Our objective was to produce a low-cost system which was nevertheless as useful as the 'Spirotrac II' (the commercially available computerised version of the Vitalograph) and we consider that our system has distinct advantages with possibilities for further improvement. The use of a standard, readily available microcomputer means that the hardware can be used for a wide variety of other purposes.

## Method

The Vitalograph Model S spirometer has an input/output port, ending in a 15-way 'D' socket at the rear of the machine. Some of the connections from this port lead to a potentiometer mounted inside the machine next to one of the pivots for the bellows. The voltage dropped across this potentiometer is a measure of the bellows expansion and is linearly related to the volume of gas within the spirometer.

The potentiometer requires a reference voltage which is most readily obtainable from the nominal 5 volt output on the analogue port of the BBC, dropped via a 2.7k resistor. The connections are shown in Table 1. It is best to use screened cable, and to use the screening for (i). No other hardware is required.

**Table 1**

**Electrical connections from Vitalograph to BBC**

| | Vitalograph | | BBC |
|---|---|---|---|
| i | Pin 5 | to | Pin 8 |
| ii | Pin 6 | to | Pin 1 (via Resistor) |
| iii | Pin 7 | to | Pin 3 |
| iv | Pin 8 | to | Pin 15 |

The software (see below) for recording the data was written in BBC BASIC; the speed of machine code is not important since the analogue-to-digital conversion takes approximately 10 ms. The programme was developed and tested in floppy-disc format but once working satisfactorily, it was transferred to a ROM chip which now resides inside the computer housing.

When it is RUN, the programme asks for the ambient temperature to be INPUT for conversion of measured volumes to BPTS, since predicted values are given under these conditions. The user is then asked to calibrate the system by emptying a 1 litre gas syringe into the Vitalograph, which gives the system a reference mark. The user then records the FEV curve exactly as done with the ordinary Vitalograph.

The system is in a 'waiting state'; no data are recorded until the programme detects that the patient has started to exhale. As soon as this occurs a short 'blip' sounds, which also enables the user to identify false starts caused by any large fluctuations in the inevitable inherent random noise in the system.

The recording capacity of our system is almost 13 seconds and therefore over twice as long as that of the standard Vitalograph. This is particularly useful in the case of patients like asthmatics who cannot empty their lungs quickly. At the end of the recording, the curve of expired volume against time is plotted on the screen. The volume axis is self-scaling so that the maximum forced vital capacity (FVC) uses the whole height of the screen. The user is then asked whether a repeat is required and if so, the programme reverts to its 'waiting state'.

When the user indicates that no further tests are required, the programme uses the highest figure recorded to calculate the printout. The PEF is calculated from the largest change found over any period of 50 ms; this was found to be a good compromise between the inaccuracies caused by extrapolating differences over a shorter period (with their associated noise) and those caused by fitting a chord to two more widely separated points on the curve, rather than a tangent at one point. The FMF is found by calculating the maximum average flow rate between 25% and 75% of FVC.

We have extended the programme to include the patient's personal data (age, height, etc.), and use these in conjunction with the prediction equations (1) to give a printout of observed and expected values for the five parameters (PEF, FMF, FVC, FEV at 1 second and $FEV_1/FVC\%$). In addition, the programme deals with data obtained from helium dilution and transfer factor tests, and produces a rapid printout of all the test results on any patient, in a form suitable for direct filing. A typical set of results obtained by the method described above is shown in table 2, together with the results given by the Spirotrac II for the same FEV curve.

## Conclusions

This method seems to be a highly satisfactory way of using the full capabilities of the Vitalograph spirometer in a way which can be appreciated by personnel not trained in the interpretation of FEV curves. It requires no expensive equipment and leaves both spirometer and microcomputer in an unmolested condition. We are investigating the possibility of using a flow meter (perhaps of the Bernoulli type) and integration programme to obtain the same parameters. This would have the advantage of greater portability as there would be no need to collect the expired gas.

**Table 2**

**Comparison of lung function results in a healthy subject**

| Parameter | BBC | Spirotrac II |
|---|---|---|
| $FEV_1$ (litres) | 4.56 | 4.55 |
| FVC (litres) | 5.22 | 5.16 |
| $FEV_1$/FVC (%) | 87.5 | 88 |
| PEF (litres.$s^{-1}$) | 13.51 | 10.62 |
| FMF (litres.$s^{-1}$) | 5.87 | 6.40 |

## Software

The software described in this article for evaluating Vitalograph data is available as a ROM chip for the BBC microcomputer (model B only). For NHS hospitals, this software is available free of charge apart from the cost of the chip itself; private hospitals wishing to purchase the ROM will be required to pay an additional fee for the software. In either case, enquiries should be sent to Mr. P. Jenkinson at the above address; potential purchasers should specify whether they require a printed output and if so, whether the printer is driven via the RS423 port or the printer port. (In all cases the FEV curve and the numerical data are displayed on the monitor).

## Reference

1. Cotes, J. E. Lung Function: Assessment and Application in Medicine (fourth edition), Blackwell, Oxford, 1979.

## Acknowledgement

My grateful thanks are due for the assistance and time given by our senior physiological measurement technician, Ken Link.

# SPRING SCIENTIFIC MEETING

The Spring Meeting of the Association took place on 20 April 1985 at Leeds General Infirmary.

We owe grateful thanks to Geoffrey Wade for organising the meeting and to the speakers for their excellent papers.

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

P. K. Morgan
Gould Medical
Vitalograph
Cardiokinetics
Bearwell International Analytical Machines Limited.

The following scientific papers were given:

1. Mechanisms of respiratory failure. S Pearson, *Killingbeck Hospital, Leeds.*
2. Limitations of physiological testing in the evaluation of breathlessness. M Muers, *St. James and Killingbeck Hospitals, Leeds.*
3. Effect of exercise on respiratory function in patients with obstructive airways disease. R Hainsworth, *Dept. of Cardiovascular Studies, Univ. of Leeds.*
4. The mass spectrometer in respiratory gas measurement. M Buckman, *Cardiothoracic Institute, Midhurst.*
5. Accuracy of gas analysis — an inter-laboratory study. DJ Chinn, Y Naruse, JE Cotes. *Univ. Dept. Occupational Health, Newcastle-upon-Tyne.*
6. Measurement of transfer factor for carbon monoxide during progressive exercise in healthy smokers and non-smokers. AH Kendrick, J Cullen, H Green, M Papouchado, G Laszlo. *Bristol Royal Infirmary.*
7. The Gould 2800 Autobox and the Morgan computerized plethysmograph compared. G. Wade, Susan Baker and Rosemary Bunting. *Killingbeck Hospital and Dept. of Cardiovascular Studies, University of Leeds.*

## ABSTRACTS

### The Mass Spectrometer in Respiratory Gas Measurement. *Maureen Buckman*

The respiratory mass spectrometer is a device for continuous simultaneous measurement of the partial pressures of several components in a gas mixture.

Technical improvements over the last thirty years have enabled the manufacture of mass spectrometers that are not only more sensitive and stable, but are also simpler in operation and maintenance and are therefore applicable to routine respiratory function. Present day respiratory mass spectrometers are capable of measuring with accuracy the rapidly changing gas concentrations of up to eight components of a gas mixture simultaneously, while sampling at flow rates of 25 ml/minute or less.

Uses of the respiratory mass spectrometer include regional gas sampling of the lung during bronchoscopy, monitoring artificially ventilated patients, measurement of oxygen consumption and carbon dioxide production and measurement of pulmonary blood flow using a non-invasive technique. At Midhurst a mass spectrometer is used to measure gases during the multi-breath nitrogen washout. From this test an index of the efficiency of ventilation is derived, the alveolar mixing efficiency (1). In a cross sectional study of 134 smokers and 162 never-smokers, alveolar mixing efficiency was measured and then plotted against age. The results show that smokers have a decline in alveolar mixing (0.42% per annum) which is greater than that of never smokers (0.22% per annum).

1. Cumming G and Guyatt AR (1982) Alveolar gas mixing efficiency in the human lung. Clinical Science *62,* 541-547.

## Measurement of the transfer factor for carbon monoxide during progressive exercise in healthy smokers and non-smokers

*Kendrick A. H., Cullen J., Green H., Papouchado M., Laszlo G.*

There is good evidence that the transfer factor for carbon monoxide (TL, CO) rises with increasing levels of work, but uncertainty remains as to the exact relationship of TL,CO and transfer coefficient ($K_{CO}$) to oxygen uptake ($VO_2$). We have studied the effects of increasing levels of work on TL,CO and $K_{CO}$ in 22 normal male subjects using the single-breath technique and a standardized protocol (Neville et al Thorax 1984; 39: 823-827). Additionally, we have investigated the effects of age, the need for carboxyhaemoglobin corrections in current smokers and non-smokers, and the variation of cardiac frequency during breath holding. Our results show that TL,CO and $K_{CO}$ increase in a curvilinear fashion up to a maximal $VO_2$. Age had no significant effect on the relationships. There was no significant increase in the carboxyhaemoglobin levels and therefore this correction is unnecessary. Cardiac frequency showed significant variation during the breath-holding manoeuvre only at rest and at low levels of exercise.

## Comparison of Two Plethysmographs.

*Wade, Baker and Bunting*

Two modern body plethysmographs were assessed. One was found to be less suitable for making measurements on patients than the other and both had problems giving results on patients with low ERV. Specific airway conductance (sGaw) and thoracic gas volume at end expiration (Vtg) was measured on normal subjects and patients in both boxes. Significant differences in sGaw were shown between the boxes in normal subjects and in patients. There was less difference of Vtg between the boxes in normal subjects and patients. Reproducibility was similar in the boxes in sGaw in normal subjects, but significantly different in patients. Reproducibility of Vtg was similar in each box in normal subjects but was significantly different in patients.

## Other papers: report by G. Wade.

*Dr. S. Pearson* discussed the control of breathing in chronic respiratory failure and showed that $CO_2$ and hypoxic respiratory drive are both reduced in 'blue bloaters'. He discussed the effects of oxygen therapy on such patients, the mechanisms by which it produced hypercapnia and whether the hypercapnia was related to loss of respiratory drive or deterioration of VQ mismatch.

*Dr. M. Muers* showed that lung function tests are useful in the assessment and management of demonstrable lung diseases such as asthma but may be of limited value in patients whose only symptom is breathlessness. He explained that the sensation of breathlessness was generated by several complex processes including psychological factors. Lung function studies do not elucidate problems of breathlessness in pregnancy, hyperventilation syndrome, or changes due to physical training and referred to Dr. Hainsworth's paper on studies in patients with COAD. Because of the difficulty in assessing "true" responses, measures of breathlessness such as the MRC scale, time of walks, visual analaogue scales, and formal exercise tests were useful.

*Dr. R. Hainsworth* showed that large variations in lung function tests such as $FEV_1$, FVC, TLCO and TLC can be expected in repeated tests on the same patient with COAD in the absence of any treatment. He spoke of the difficulties these patients have of undertaking an exercise training programme to increase their 12 minute walking distance and showed that patients who trained increased their 12 minute walking distance significantly over the first 3 weeks.

*Dr. D. J. Chinn* discussed the results of the recent survey of gas analysis carried out by the Newcastle group. Variations of 3-5% for CO and He, 1-8% for $O_2$ and 5-8% for $CO_2$ were shown between the laboratories. Calculation of the respiratory exchange ratio using the results of the $CO_2$ and $O_2$ analysis showed a coefficient of variation of 10%, larger than would be expected from biological variation.

# BOOK REVIEWS

## Pulmonary Function Testing — Guidelines and Controversies

Ed. Jack L Clausen

*Grune & Stratton Inc, 1984. 368 pages.*
*ISBN 0-8089-1647-5. Price £14.50 (soft cover).*

This book has arisen from a project of the California Thoracic Society to develop recommendations for methods of pulmonary function tests in pulmonary function laboratories, and is primarily aimed at technicians, physiologists, and physicians who are responsible for the clinical application of pulmonary function testing. It covers aspects of equipment, technique, and normal values for a wide range of common tests. A further volume will follow covering indications for tests, interpretation of results and clinical usefulness of the tests.

The first six chapters cover general aspects, appropriate to all laboratories, including infection control, laboratory safety and instrumentation. The remaining 22 chapters cover specific testing procedures for the more common pulmonary function tests. Each of these chapters is generally subdivided into sections on equipment, quality control, test procedures, calculations, normal values, reproducibility and controversial issues.

Unfortunately, the detail and range of referencing given for each chapter is somewhat variable, and the inclusion of many more illustrations throughout the text would have been appropriate.

Despite the origin of this book, its contents are also applicable to European laboratories, but it should be regarded as a critical review on the technical aspects of pulmonary function tests, and not as a methodological handbook. It should provide a useful supplement to Cotes' *Lung Function.*

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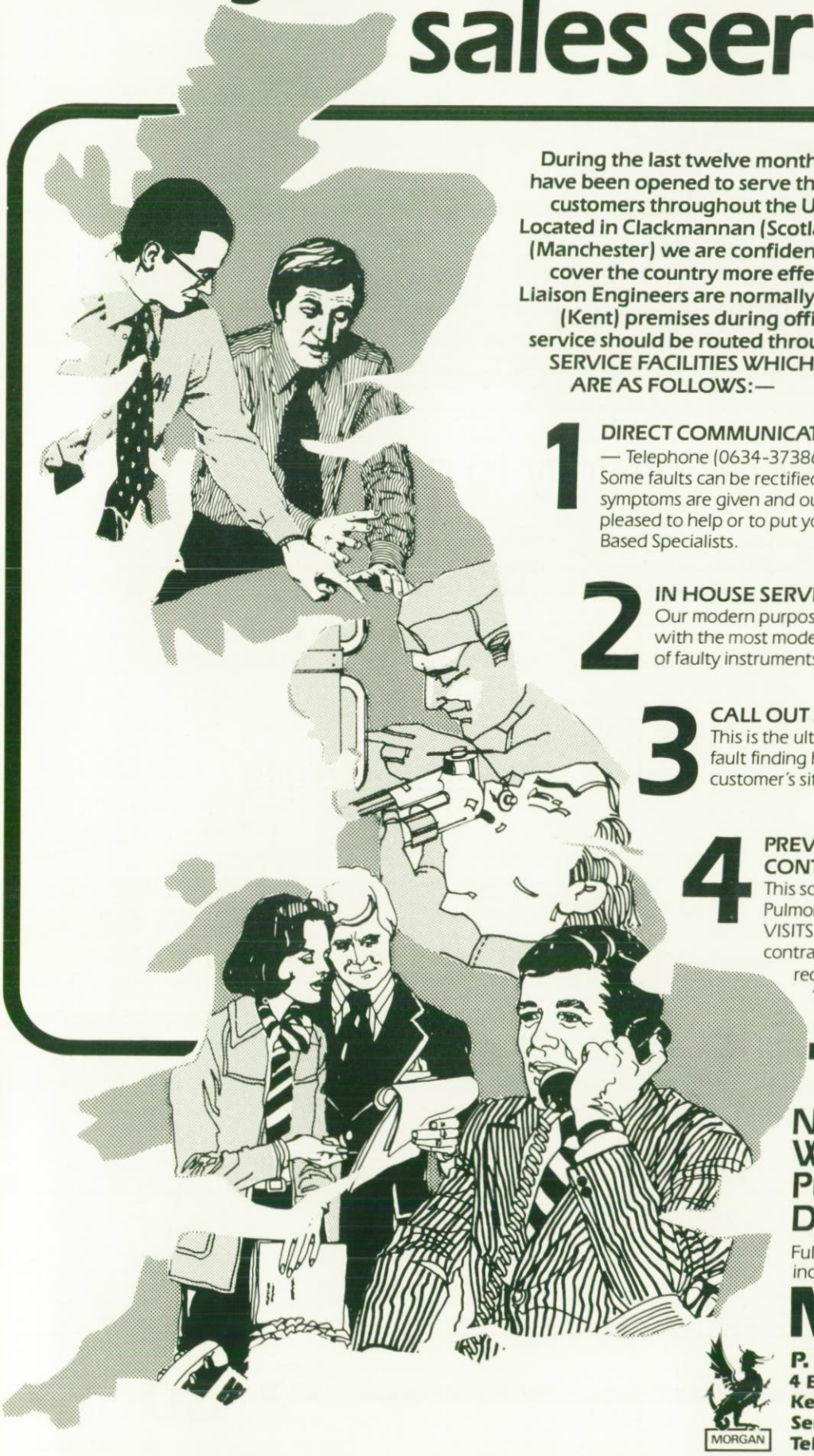

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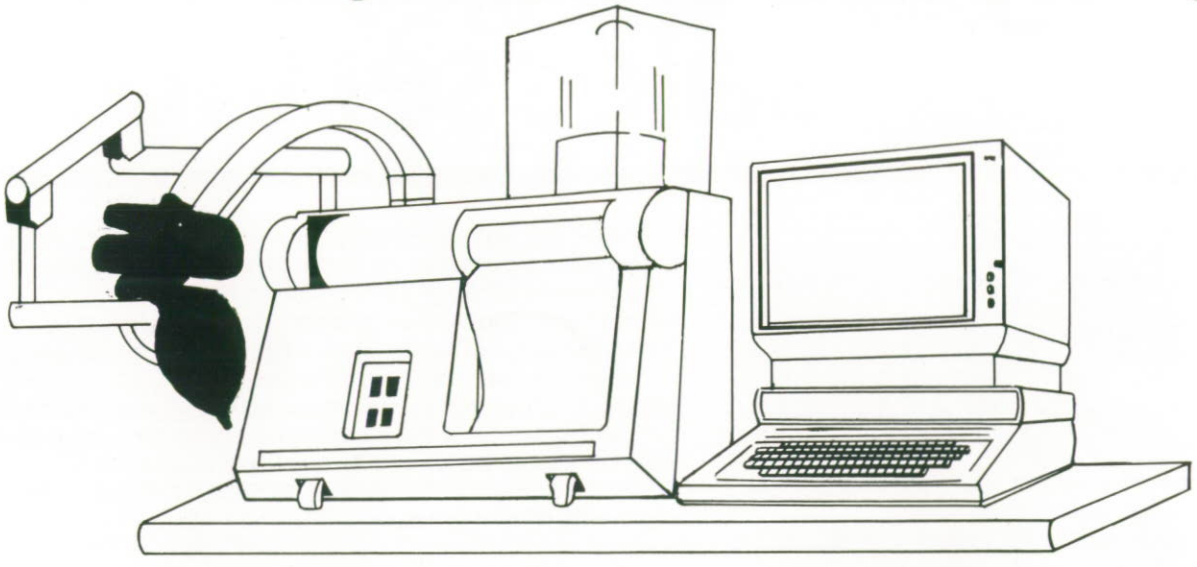

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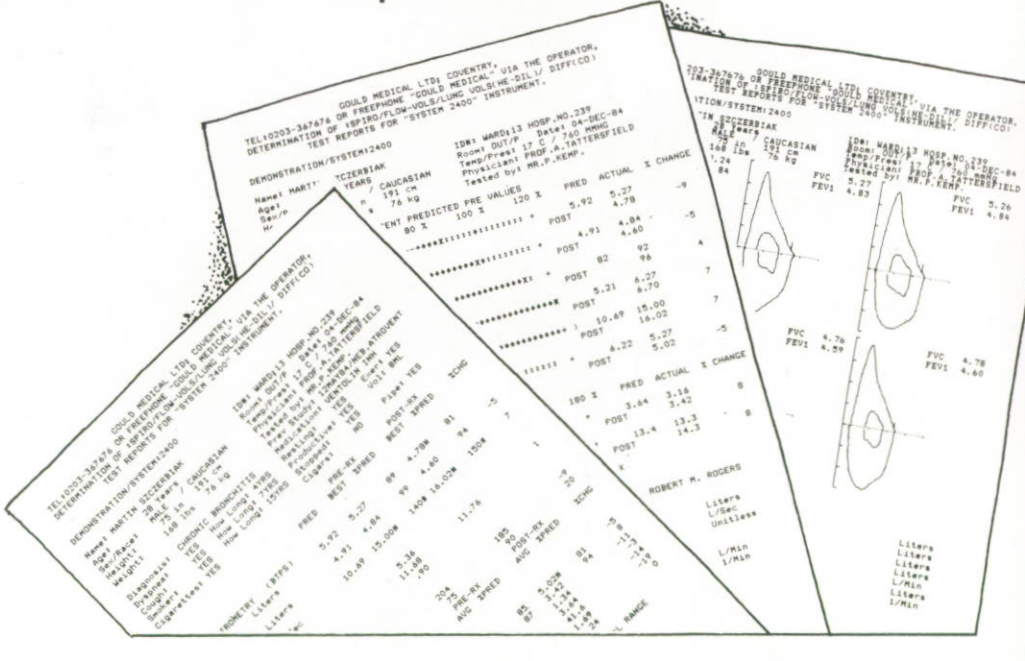

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## The Pathway for Oxygen: Structure and Function in the Mammalian Respiratory System.

E. R. Weibel

*Harvard University Press, 1984. 425 pages.*
*ISBN 0-674-65790-X. Price £19.95 (soft cover).*

This book is based on a series of lectures delivered by the author at Harvard University in 1979. Its aim is to explore the mechanisms by which an efficient pathway for oxygen — from lung to blood, to the tissues, to the cells and to the molecular respiratory chain in the mitochondria — is established and maintained in man and animals, and how these different mechanisms are dependant on each other. Although the bias is towards morphology, the physiological and biochemical aspects of the processes are given good coverage.

The book has 13 chapters, with a very brief discussion of SI units, particularly of their problems. Chapter 1 discusses "oxygen and the history of life", which gives an excellent opening for the rest of the book. The next 10 chapters cover cellular respiration, mitochondria, oxygen transport mechanisms, design and development of the lung, lung cell biology, and the mechanical support of the lung. Chapter 12 is of particular interest, covering the role of the lung as a gas exchanger, and includes sections on the physiology and morphology of pulmonary diffusing capacity (transfer factor). The final chapter presents overviews of the respiratory system.

Throughout, the text is lucid, concise, very well illustrated, and a pleasure to read. Each chapter is well referenced, with both recent works, and important "classic" works, which allow the reader to explore further particular aspects in detail outside the scope of this book. This excellent book is highly recommended, both for technicians and for physiologists.

## Measurement in Clinical Respiratory Physiology

Eds. Gabriel Laszlo; Michael F Sudlow.

*Academic Press, 1983. 337 pages.*
*ISBN 0-1243-7030-2. Price £35.00 (hardback).*

This book forms part of the Medical Physics series from Academic Press, and provides a useful additional source of information. Its aim is to be a practical handbook, specifically for the hospital physicist, technician or physiologist responsible for setting up and running a respiratory physiology department. All the contributors are active workers in the field of respiratory physiology.

The book does not, however, provide a comprehensive review of all the measurements of respiratory function, but instead concentrates on the more important aspects of measurements in current practice. Additionally, there is no systematic discussion of respiratory physiology, interpretation of respiratory function tests, or their clinical relevance.

Following a brief introduction from the editors discussing the work of the respiratory function laboratory, the book is divided into two parts. The first discusses in detail the instrumentation used in respiratory laboratories. Most of the information given, particularly of calibration procedures, is not easily found elsewhere. In the second, discussion is centred on the assembly of equipment for specific respiratory function tests. Throughout the book, the text is generally clear, concise and very informative, with each chapter generally well referenced.

This book should be a useful and welcome addition to the shelves of a departmental library, and is recommended to all physiological measurement and medical physics technicians involved in respiratory function testing, as well as technicians from other disciplines who are seeking academic qualifications.

## Clinical Tests of Respiratory Function.

G J Gibson.

*MacMillan Press Ltd. 1984. 334 pages.*
*ISBN 0-3333-2568-0. Price £30.00 (hardback).*

This book, principally aimed at physicians, sets out to provide a clear understanding of the role of respiratory function testing in the clinical diagnosis of both respiratory and non-respiratory disorders, and to aid in the interpretation of tests of respiratory function.

Following a brief description of the types of apparatus used in respiratory function testing, chapters 2 to 6 describe the theoretical and practical aspects of the commonly applied tests of mechanical and gas exchange function, of respiratory control and exercise, together with the causes of their variations in normal subjects. These chapters present many of the difficult physiological concepts, particularly of lung mechanics, in a clear and understandable form. Chapters 7 to 10 deal with diseases of the respiratory system, and again provide a clear and concise view of the use and expected findings for the various measurements of lung function. Chapters 11 to 17 deal with diseases primarily of non-respiratory origin, including areas of cardiac disease and neuromuscular disorders. Finally, in chapter 18, the author gives some guidelines on the everyday application of the tests. Each chapter is well referenced, providing excellent sources for further reading.

For respiratory technicians, this book provides a very lucid and concise theoretical guide to the use and role of respiratory function tests, and should give technicians a greater understanding of the results of the tests in the many disorders that are referred for testing. This excellent book is strongly recommended, and is an essential book for every respiratory department.

## Recent articles from Thorax, British Journal of Diseases of the Chest and American Review of Respiratory Diseases — Oct. 1984 to March 1985. Selected by Derek Cramer.

**Thorax 1984 Vol. 39**

| | |
|---|---|
| Oct. (No. 10) | Temperature corrections in routine spirometry. D. Cramer, A. Peacock and D. Denison. p 771-774. |
| | Abnormalities in the flow-volume loop in obstructive sleep apnoea — sitting and supine. E. Shore, R. Millnam. p 775-779. |
| Nov. (No. 11) | A standardized method of estimating KCO on exercise. E. Neville, A. Kendrick. p 823-827. |
| | Effect of encouragement on walking test performance. G. H. Guyatt, S. Pugsley et al. p 818-822. |
| Dec. (No. 12) | Pulmonary vascular resistance in children with congenital heart disease. N. Davies, E. Shinebourne et al. p 895-900. |
| | Pulmonary function in adolescents with mild idiopathic scoliosis. R. Smyth, K. Chapman et al. p 901-904. |
| | Short term variability in $FEV_1$ and smoking habit. P. Tweeddale, S. Merchant et al. p 928-932. |

**1985 Vol. 40**

Jan. (No. 1) Bronchial reactivity to inhaled histamine and annual rate of decline in $FEV_1$ in male smokers and ex-smokers. R. G. Taylor, H. Joyce et al. p 17-22.

Dose response effect of Sodium Cromoglycate pressurised aerosol in exercise induced asthma. W. M. Tullett, K. M. Tan et al. p 41-44.

Lung function in the elderly. M. L. Burr, K. M. Phillips, D. N. Hurst. p 54-59.

Effects of pnuemothorax or pleural effusion on pulmonary function. J. J. Gilmartin, A. J. Wright, G. J. Gibson. p 60-65.

Feb. (No. 2) Effects of mitral valve surgery on static lung function and exercise performance. K. M. Rhodes, K. Every et al. p 107-112.

March (No. 3) Nebulised salbutamol without oxygen in severe acute asthma: How effective and how safe? J. G. Douglas, P. Rafferty et al. p 180-183.

**Br J Dis Chest**
**1984. Vol. 78.**

No. 4 Home nebulisers in severe chronic asthma. D. McGivern, M. Ward et al. p 376-382.

The efficacy of drug delivery by a pear shaped spacer and metered dose inhaler. J. Morris, J. Milledge et al. p 383-387.

The 100 metre walk: A simple and reproducible exercise test. A. Morice and T. Smithies. p 392-394.

**1985. Vol. 79.**

No. 1. How well do asthma clinic patients understand their asthma? M. E. Ellis and J. A. R. Friend. p 43-48.

**Amer Rev Resp Dis**
**1984. Vol. 130.**

Oct. (No. 4) Evaluation of single breath helium dilution — total lung capacity in obstructive lung disease. G. Burns and J. Scheirhorn. p 580-583.

Dec. (No. 6) Mechanics and gas distribution in normal and obstructed lungs during tidal breathing. K. R. Lutchen, G. M. Saidel and S. P. Primiano et al. p 974-979.

**1985. Vol. 131**

Jan. (No. 1) Determination of cardiac output at rest and during exercise by $CO_2$ rebreathing method in obstructive airway disease. D. A. Mahler, R. A. Matthay, P. E. Snyder et al. p 73-78.

Feb. (No. 2) Pulmonary function and respiratory symptoms in polyvinylchloride fabrication workers. M. E. Baser, M. S. Tockman and T. P. Kennedy. p 203-208.

# ARTP News

The ARTP revised leaflet "A Career in Respiratory Physiology" is now available from Dena Muirhead at the Cardiothoracic Unit, Derbyshire Royal Infirmary. Telephone: 0332 47141.

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### 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 arises in job advertisements appearing in *Breath*.**

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

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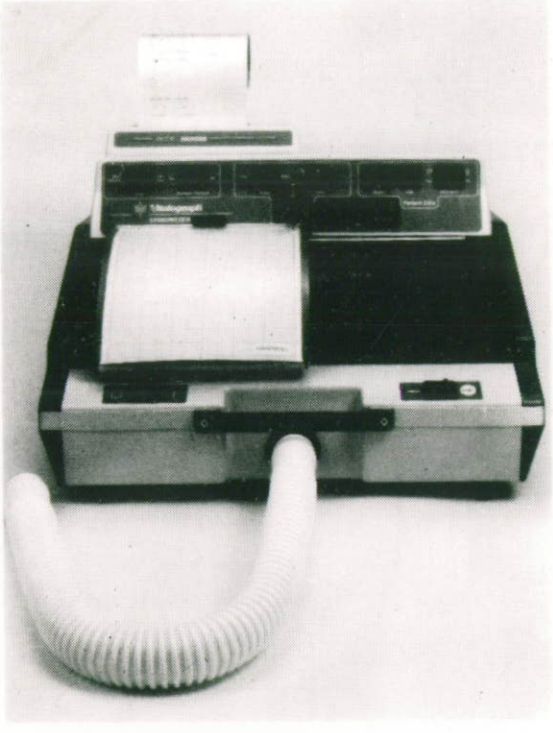

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| FVC | 5.18 | 4.10 | 79 | 4.60 | 89 | + 12 |
| FEV 1 | 4.18 | 2.65 | 63 | 3.78 | 90 | + 42 |
| FEV 1/VC | 81 | 59 | -22 | 83 | + 2 | + 24 |
| FEV 1/FVC | 81 | 65 | -16 | 82 | + 1 | + 17 |
| FMEF | 5.13 | 2.05 | 40 | 3.69 | 72 | + 80 |
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