ASSOCIATION OF RESPIRATORY TECHNICIANS AND PHYSIOLOGISTS




ISSUE NO 13 JUNE 1981

# BREATH

## CONTENTS

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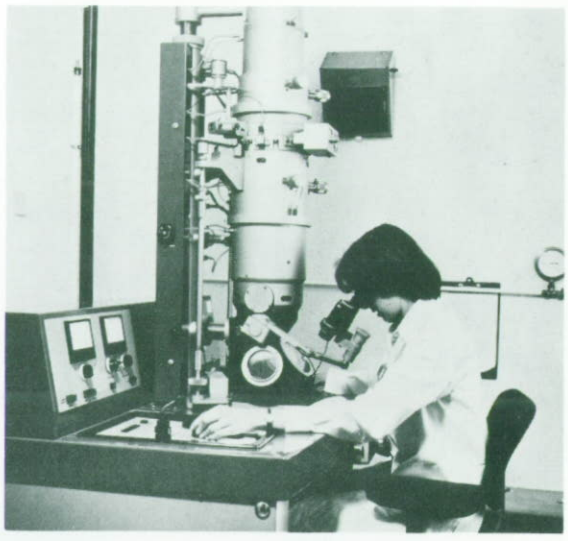

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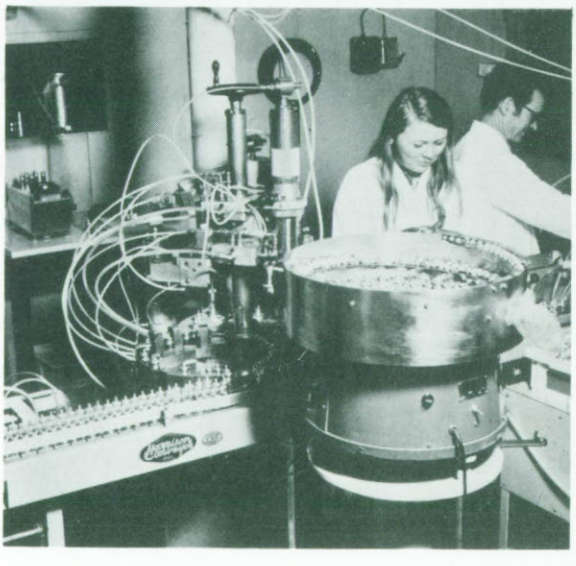

## Significant advances in therapy from Original British Research

# EDITORIAL

## Victims of Respiratory Disability

Chronic lung disease with all its resulting disabilities has been part of the life of this country for very many years. The killers of the past such as tuberculosis, air pollution and pneumoconiosis, if they have not been eliminated have at least come under some degree of control. One might therefore have hoped that disabling lung disease would have become a thing of the past, but in spite of the massive NHS budget this has not occurred. The 20th-century syndrome of chronic airflow obstruction is of course quite largely due to the epidemic of cigarette smoking which has figured rather prominently in our columns of late. While prevention of this group of diseases must remain our basic goal, the care of those who have already become victims, engages much of our efforts.

The recent report[1] by a Working Party of the Royal College of Physicians is therefore of interest. The usual daunting statistics are first outlined — 26 million working days lost per year, 57,000 on the Disabled Persons' Register because of respiratory disease and so on — and the report then goes on to devote its main attention to treatment and the problems of rehabilitation and employment.

Provision of suitable employment for those with respiratory disease has always been difficult and the report gives a good short account of the services which are available to provide employment for the disabled, and which we perhaps take for granted. The Employment Medical Advisory Service (a branch of the Health and Safety Executive) is concerned with prevention of occupational disease and with advising those with health problems on suitable work. The Manpower Services Commission runs a number of Employment Rehabilitation Centres to help the handicapped to return to work or to obtain alternative employment and it also supports 'Sheltered Workshops' for the severely disabled. To a cynical observer, these worthy bodies look like imminent victims of future 'cuts in public expenditure'.

An interesting new suggestion which again would need funding is for a 'Respiratory Health Worker' rather on the lines of the Tuberculosis Health Visitors whose numbers are now much reduced. Such a person would have many possible duties such as promoting health education, providing support and assessment in employment and assisting in domicilary care. Such a post they say, could

be filled by a nurse, health visitor, occupational therapist or *lung function technician* (our italics)! Just a thought — perhaps the ARTP should take an interest in this proposal.

The knotty problem of oxygen therapy again receives an airing. We have in a previous editorial[2], drawn attention to the difficulties of obtaining oxygen in the domiciliary setting. While oxygen must be made available in the home for emergency use in spite of the expense, a new dimension has been brought to this problem by the recommendations for long-term continuous or semi-continuous use of domiciliary oxygen. Two large-scale studies in patients with severe chronic airflow obstruction have been carried out and it is worth considering these in more detail.

The first study is a multi-centred trial conducted in this country by a Working Party of the Medical Research Council[3] and compares the mortality rate in two groups — a *treated* group who received oxygen at about 2 litres/min by nasal prongs for at least 15 hours a day and a *control* group who received no oxygen; the 5-year survival was rather better in the treated group.

In a second study carried out in the United States[4], patients were allocated *either* to continuous oxygen therapy *or* to 12-hour nocturnal therapy, those in the continuous group having the lower mortality rate. A number of the patients contrived to carry on smoking during the trials (even in the groups taking oxygen) but it was a little surprising to find that there was scant analysis of this aspect (none at all in the American study). Naively perhaps, one would imagine that the longer the patients were on oxygen, the less they would be likely to smoke. While the 'Oxygen Concentrator'[5] involves much less expense than the provision of cylinders, prolonged oxygen therapy is still a costly form of treatment. Before prescribing it therefore one would like to feel that there is a good chance of really benefitting the patient.

The Royal College report has many good points and is well worth some detailed study. For all this, we return to the old saying (with a present-day slant), 'Prevention is better than cure and miles better than palliation.'

**References**


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2. Editorial (1978). Breath No. 8, p 2.
3. Medical Research Council (1981). Long-term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. Lancet 28 March, 681-686.
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# OCCUPATIONAL ASTHMA

*A. Newman Taylor*
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Occupational asthma may be defined as variable airway narrowing causally related to agents inhaled in the working environment. It affects only a minority of those exposed and develops only after an initial symptom-free period of exposure, which can vary between individuals from weeks to years. Asthmatic reactions tend to recur on re-exposure to the causal agent at atmospheric concentrations which do not affect others similarly exposed. Although occupational exposure is a relatively infrequent cause of asthma, it is important to recognise it, as avoidance of further exposure can be followed by complete remission of symptoms and restoration of normal lung function.

## CAUSES

A large number of agents to which exposure occurs at work have been reported as causing asthma. Those of major importance in the United Kingdom are listed in Table 1. New materials are continually being introduced into industry, so that the number of agents causing occupational asthma is likely to increase.

## CLINICAL FEATURES

The characteristic feature of occupational asthma is episodic breathlessness which may be associated with coughing, wheezing or chest tightness and which is temporally related to occupational exposures. Not uncommonly those affected may themselves recognize that their symptoms develop during the working week, often increasing in severity as the week progresses, improve when away from work, at weekends and on holiday and recur on return to work. Symptoms can develop either within minutes of exposure or more usually several hours after the onset of exposure, starting during the latter part of the working day or even in the evening after returning home.

On occasions the recognition of occupational asthma may be difficult. In some patients breathlessness occurs without wheezing or chest tightness, varies little in severity from day to day and can persist for several days, or even weeks after exposure has ceased. In such patients symptoms can easily be attributed to the effects of cigarette smoking. Patients with occupational asthma also find that, in addition to occupational exposures, stimuli such as exercise, upper respiratory tract infections and inhalation of cold air may also provoke asthmatic reactions.

## IMPORTANCE OF DIAGNOSIS

Any patient with occupational asthma should be advised to avoid further exposure to the causal agent. Relocation within the same firm is sometimes possible but often further exposure can only be avoided by leaving current employment. No physician would wish to give such advice without being as confident as possible that the patient's symptoms are due to specific occupational exposure. This usually requires evidence of a temporal relationship between asthma and occupational exposure. Inhalation testing with the specific agents suspected of causing disease was developed for this purpose but such tests are potentially hazardous and should only be conducted by those experienced in their techniques. They are very demanding on resources and the patient must be admitted to hospital for observation by medical staff for at least twenty-four hours after testing. Other methods of investigation have therefore been increasingly employed, of which the most important is the measurement of lung function during periods of occupational exposure.

## TABLE 1: SOME CAUSES OF OCCUPATIONAL ASTHMA

| CAUSAL AGENTS | CIRCUMSTANCES OF EXPOSURE |
|---|---|
| **BIOLOGICAL AGENTS** | |
| B Subtilis enzymes (Alcalase, etc) | Enzyme detergent industry |
| Small mammal urine proteins (rats, mice, etc) | Animal laboratory workers |
| Grain, flour and their contaminants | Food industry (millers, bakers, etc), farmers |
| Wood dusts (Western red cedar/Iroko, etc) | Wood mills, carpenters |
| Colophony (as soft solder flux) | Electronics industry |
| Antibiotics (penicillins, etc) | Pharmaceutical industry |
| **CHEMICAL AGENTS** | |
| Di-isocyanates: toluene (TDI), diphenylmethane (MDI), hexamethylene (HDI), and naphthalene (NDI) | Polyurethane foam manufacture<br>Printing industry<br>Synthetic paints and rubber adhesives |
| Epoxy resin curing agents (phthalic anhydride, trimellitic anhydride, triethylene tetramine, etc) | Surface coating<br>Adhesives |
| Complex salts of platinum (particularly ammonium hexachlorplatinate) | Platinum refining |
| Formaldehyde | Laboratory workers |

## PEAK FLOW RECORDS

Away from their place of work many patients with occupational asthma have either normal lung function or evidence of airflow limitation which fails to improve after inhalation of bronchodilator. Single measurements of lung function made before and after a working shift may provide evidence of occupational asthma but are often found to be unhelpful and may be misleading. The most useful method of demonstrating work-related asthma is regular measurement of peak expiratory flow rate made by the patient over several weeks including periods both at and away from work.

The patient is provided with a peak flow meter (most conveniently the Mini-Wright Peak Flow Meter) and asked to measure peak flow rate every two hours from waking to sleeping and to record on each occasion the best of three measurements. Those who have a normal peak flow rate require a minimum period of two weeks at work with two two-day rest periods with less than 15% diurnal variation for the records to be considered normal. Those with a decreased peak flow rate require a similar period at work, followed by at least ten days away from work and a further two-week period at work to give sufficient time for improvement to occur.

Prophylactic drugs such as sodium cromoglycate and corticosteroids should not be taken during the period of the record as they can mask significant changes. The frequency with which other forms of treatment such as bronchodilators are taken should be recorded and if possible kept constant during periods at and away from work. It is convenient to plot the arithmetic mean of the peak flow rates for each day, together with the maximum and minimum values recorded for that day.

## PATTERNS OF OCCUPATIONAL ASTHMA

Such records show that the patterns of asthmatic reaction which occur in those with occupational asthma are quite different from those reported following exposure to specific occupational agents in inhalation tests (see below) where the exposure is single and of short duration. The different patterns of asthmatic reaction which occur during and following occupational exposure depend particularly on the time taken for normal lung function to be restored and on the cumulative effect of repeated exposure. Three separate patterns of asthmatic reaction have been distinguished: —

(1) *Equivalent deterioration in peak flow rate during each day at work* with restoration of normal lung function on the first day away from work. This pattern is unusual and occurs in those whose recovery after work is sufficiently rapid for their peak flow rate to return to normal overnight.

(2) *Progressive deterioration in peak flow rate during each day of the working week* with restoration of previous lung function within three days. Restoration of lung function is however complete by the end of a weekend away from work.

(3) *Progressive deterioration in peak flow rate week by week.* This occurs in those whose period of recovery takes longer than a weekend away from work. Recovery may not start for a week or more after exposure to the causal agent has ceased and can continue for up to three months. This pattern is particularly common in occupational asthma due to diisocyanates.

## SKIN TESTS

Further confirmation of the diagnosis of occupational asthma is obtained by skin prick testing with extracts of the causal agent which in some patients will elicit an immediate reaction. Skin testing however is limited to soluble allergens which provoke an IgE mediated reaction. Such allergens include the Bacillus subtilis enzyme, alcalase, the complex platinum salt, ammonium hexochloroplatinate and urine from laboratory animals such as rats, mice and guinea-pigs.

## INHALATION TESTS

There remain circumstances in which it is necessary to undertake inhalation testing: —

(1) Where an individual is suspected of reacting to an agent not yet recognised as a cause of occupational asthma.

(2) Where an individual with work-related asthma is exposed to more than one recognised cause of the disease.

(3) Where the symptoms experienced at work are of such severity that it is not thought justifiable for the patient to be further exposed in his working environment.

(4) Where genuine doubt remains about the diagnosis of occupational asthma after other appropriate investigations, including work records of peak flow measurements have been completed.

The aim in an inhalation test is to expose the affected individual to the agent or agents thought to be the cause of asthma in circumstances which resemble as closely as possible the conditions of exposure at work. Unlike exposures at work, exposures in inhalation tests are of short duration, using individual agents in carefully controlled conditions. The exposure concentrations in inhalation tests should be as close as possible to the exposures experienced at work; the same applies to the physical conditions of exposure at work such as the temperature to which materials are heated and the size of dust particles.

Several different methods for inhalation testing with occupational agents have been developed which depend primarily on the physical state of the different materials. Inhalation of nebulised extracts is used for exposure to extracts of *soluble allergens* such as urine and serum proteins. This is the traditional method of inhalation testing but is not applicable to the majority of occupational agents. Liquids such as *toluene di-isocyanate* and *formaldehyde* which are volatile at room temperature can be painted onto a flat surface in an enclosed space. The atmospheric concentration achieved can be varied by using different concentrations of the material in solution and is measured in an appropriate monitor. Exposure to dust such as *wood-dust*, the complex *platinum salts* and *pharmaceutical agents* such as antibiotics can be achieved by asking the patient to tip the material between two trays in an enclosed chamber. Wood-dusts can be used without dilution, but antibiotics and complex platinum salts require dilution in dried lactose powder to avoid exposures greater than those experienced at work. Materials which are heated at work such as the soft solder flux, *colophony*, are heated to the same temperatures during the inhalation testing so that exposure to similar degradation products occurs.

## ASTHMATIC REACTIONS DURING INHALATION TESTS

At least four patterns have been distinguished: —

(1) *Immediate reactions* which develop within minutes and improve spontaneously over one to one and a half hours.

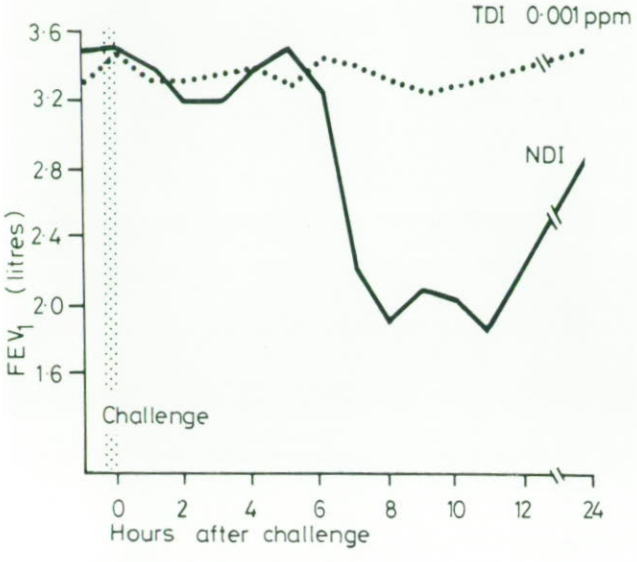


***Fig 1.*** *Patient 2. Bronchial provocation test by exposure to NDI vapour for 60 s showing an early non-immediate reaction. No reaction to TDI. These are curing agents used in rubber manufacture.*

(2) *Late reactions* (Fig 1) which develop an hour or more after exposure, most commonly after an interval of 3 to 4 hours, and which persist for 24 to 36 hours before resolving spontaneously.

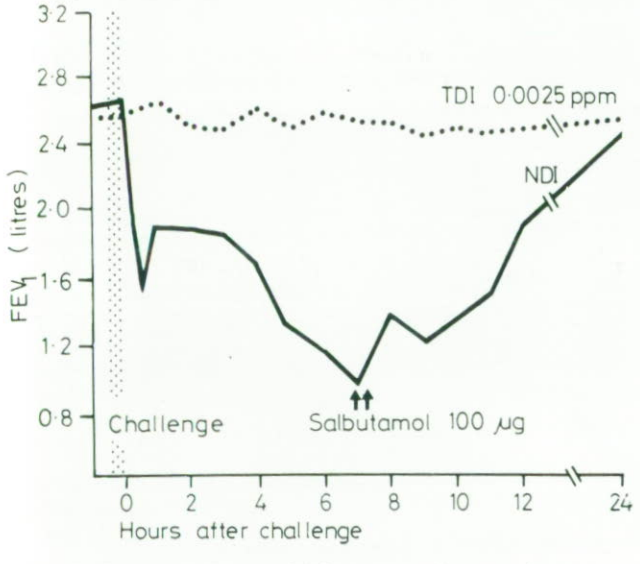


***Fig 2.*** *Patient 1. Bronchial provocation test by exposure to 1,5-naphthylene di-isocyanate (NDI) vapour for 60 s showing both immediate and non-immediate (dual) reactions. No reaction to toluene di-isocyanate (TDI).*

(3) *Dual reactions* (Fig 2): a late reaction preceded by an immediate reaction.

(4) *Recurrent nocturnal reactions* which wake the patient from sleep with complete or partial improvement in lung function during the day. Such reactions follow a single exposure of short duration. Because of these recurrent reactions it is essential that sufficient time is left between exposures to different agents to ensure that their effects can be distinguished.

Figures 1 and 2 reproduced from: Harries et al (1979) Isocyanate Asthma. Thorax *34* 762. Reproduced by kind permission of the authors and the Editor of Thorax.

## CONCLUSION

The diagnosis of occupational asthma is based not only on a history of work-related symptoms but also on evidence of change in lung function related to occupational exposure. Accurate diagnosis of occupational asthma is important for two reasons; firstly, to allow those with the disease to avoid further exposure to the causal agent and secondly, to prevent those without occupational asthma being advised unnecessarily to change their place of work.

# MEDICAL MEMO

## Sarcoidosis

*D. C. S. Hutchison*
King's College Hospital Medical School

Patients with pulmonary sarcoidosis are common visitors to the lung function laboratory and repeated attendance may be required in chronic cases. Sarcoidosis can affect almost all the organs of the body, though the earliest manifestations are usually seen in the thorax.

### CLINICAL FEATURES

#### *The acute stage*

Intrathoracic lymph gland enlargement is the commonest feature of sarcoidosis and the radiological picture is very characteristic (Fig 1). In about half the cases, there are no symptoms and the patient comes to the attention of the chest specialist simply because of the X-ray abnormality; other patients develop mild shortness of breath, cough or chest pain. Lung function tests are usually quite normal at this stage and in 90% of the cases the condition clears up without any further trouble.

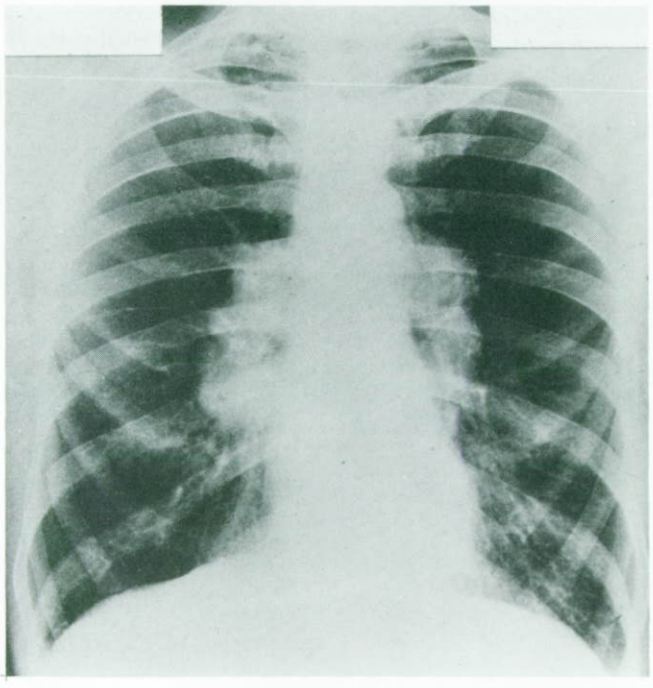

***Fig 1.*** *Gross bilateral hilar gland enlargement; there are also some line shadows and nodules at the bases.*

Hilar gland enlargement may also be associated with erythema nodosum (red tender swellings on the shins), fever and migratory joint pains.

#### *Pulmonary infiltration*

In the acute stage, about one-third of patients develop nodular or larger patchy X-ray opacities in the lung fields (Fig 2) and this can be associated with moderate shortness of breath, though again there may be virtually no symptoms. Most of these cases resolve spontaneously or after steroid treatment.

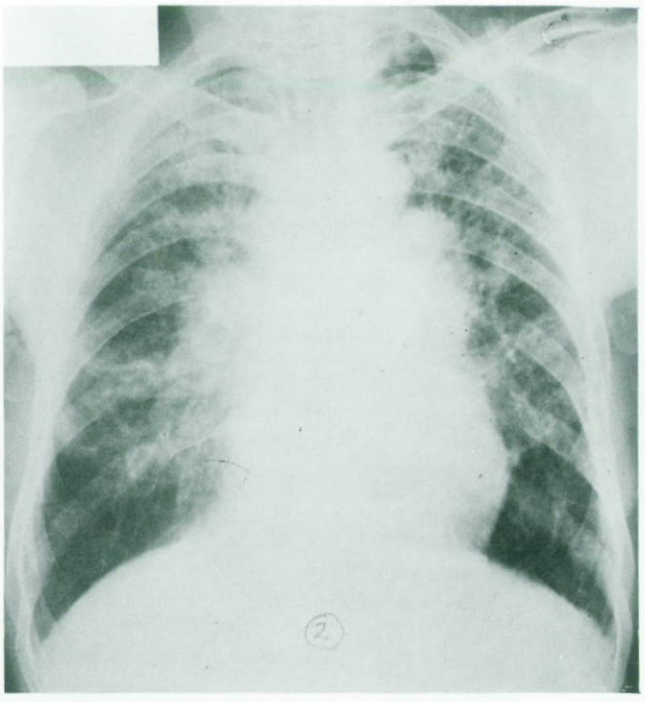

***Fig 2.*** *Hilar gland enlargement with more severe pulmonary infiltration.*

#### *Pulmonary fibrosis*

In a minority, the stage of pulmonary infiltration progresses to irreversible pulmonary fibrosis (Fig 3), with increasingly severe shortness of breath. The disease may 'burn itself out' leaving the patient in a stable state, but with impaired lung function; in some cases there is a steady downhill course, the patient eventually dying in cor pulmonale.

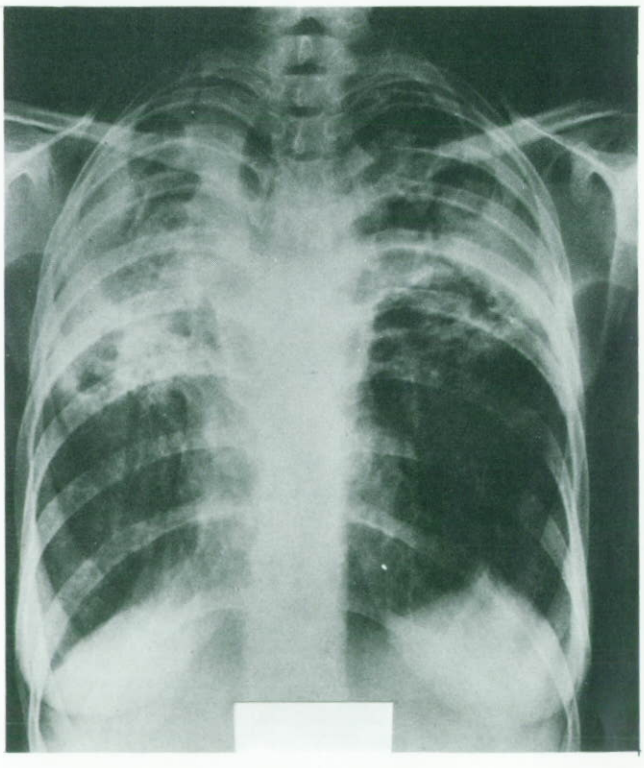

***Fig 3.*** *Severe pulmonary fibrosis: there is consolidation and shrinkage in both upper zones, particularly on the right, the trachea being deviated to that side. The diaphragms are pulled up and distorted.*

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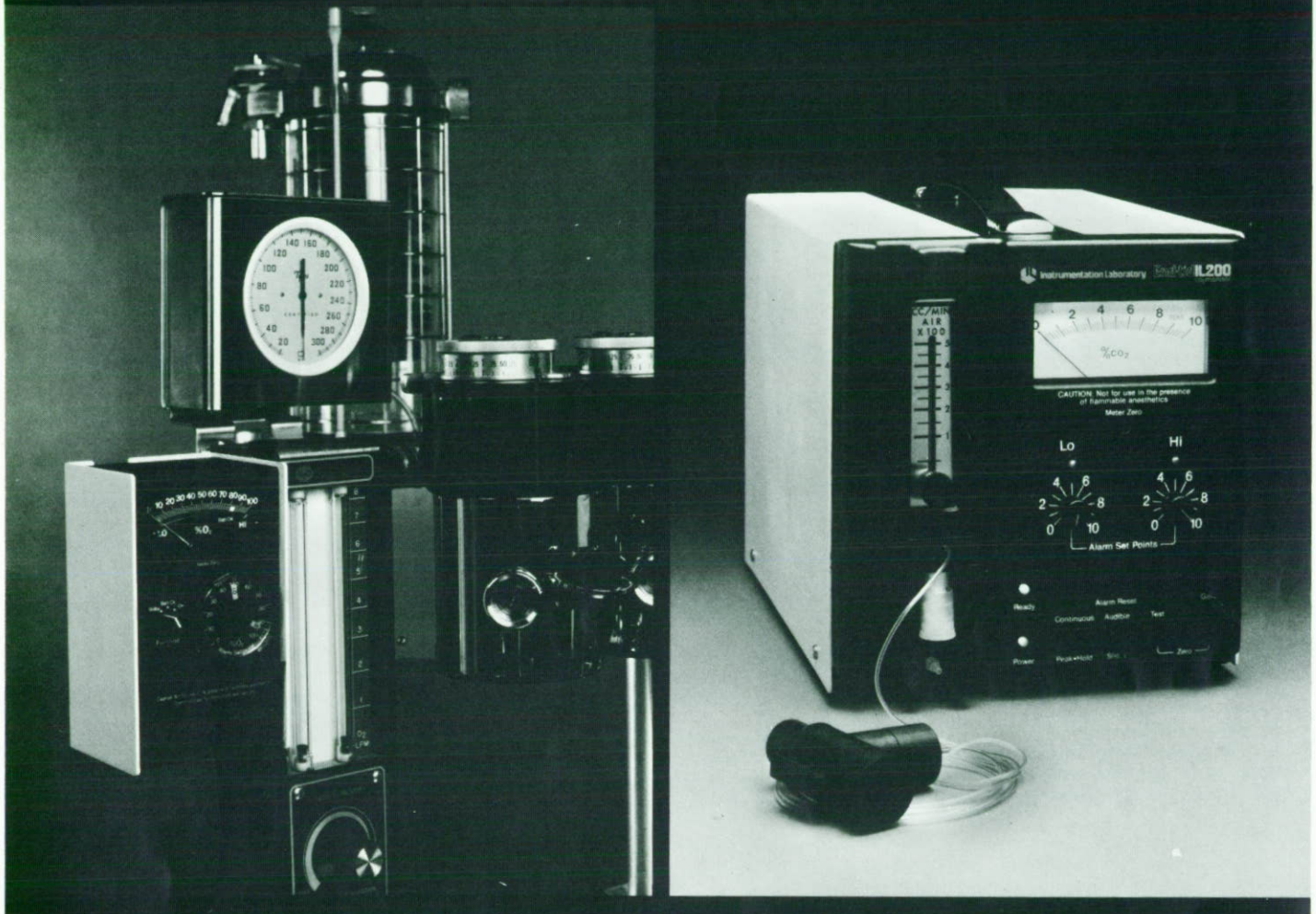


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## PATHOLOGY

The basic pathological lesion is the 'granuloma'; this is a rounded collection of chronic inflammatory cells (the 'epithelioid' cells) whose characteristic appearance is easily recognisable under the light microscope (Fig 4). They can be seen in biopsy specimens of lung, bronchial mucosa, lymph nodes, liver or spleen.

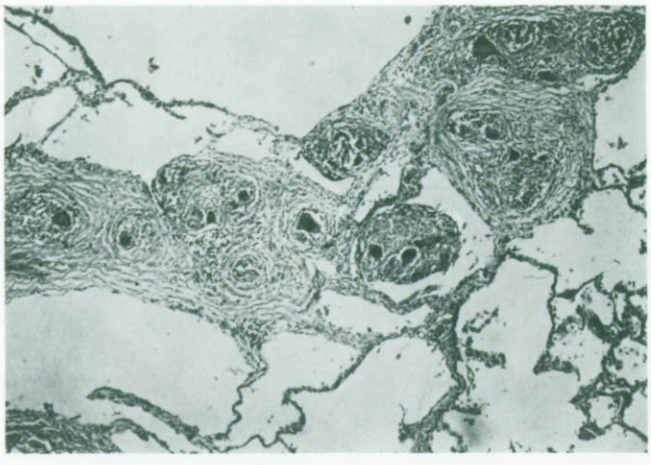

*Fig 4. Lung biopsy in sarcoidosis. There is obvious thickening of the interstitial spaces between the alveoli with fibrous tissue and characteristic granuloma formation. Multinucleated giant cells appear at the centre of the granuloma.*

### *The Kveim test*

The Kveim-Siltzbach test (often referred to simply as the Kveim test) is a useful aid to diagnosis. 0.1 ml of a sarcoid tissue extract is injected intradermally; a nodule forms which is biopsied four to six weeks later and on histological examination the characteristic granulomas can be recognised. A positive result is obtained in 80 to 90% of the acute cases but much less often in the chronic form.

## LUNG FUNCTION TESTS

In the stage of hilar gland enlargement, lung function tests usually remain normal. When pulmonary infiltration occurs, a moderate fall in CO transfer factor may be seen which usually reverts to normal as the condition resolves. In pulmonary fibrosis, a 'restrictive' pattern is seen (Table 1), where the lung volumes are reduced, often to a severe degree. The $FEV_1/VC$ ratio is often above normal unless there is an accompanying disorder causing airflow obstruction. The CO transfer factor is reduced, partly due to the loss of lung volume and partly to the loss of surface area for gas exchange. Due to mismatching of ventilation and perfusion, arterial $pO_2$ is reduced which is, at least in part, responsible for stimulation of ventilation so that arterial $pCO_2$ is also below normal. Pulmonary compliance is reduced, reflecting the increased stiffness of the lungs brought about by fibrosis.

Measurement of lung function in sarcoidosis may help to confirm the diagnosis, though other conditions, such as fibrosing alveolitis may produce a similar pattern. In patients with pulmonary involvement, it is particularly important to follow the changes in lung function with time so that the effects of treatment (eg with steroids) can be assessed.

## TREATMENT

No treatment is generally required for hilar gland enlargement alone and spontaneous resolution can be expected. When pulmonary infiltration is present, steroids are commonly given, though it is not at present clear how effective this is, bearing in mind that a number of cases resolve without treatment. Prednisolone is given in a fairly large dose to begin with (say 40 gm per day orally); the dose is then gradually reduced over several weeks, while keeping the chest X-ray and lung function tests under careful observation. The eventual aim is to stop steroids but if there is any deterioration the treatment must be restarted. Once pulmonary fibrosis has set in, steroid treatment is of little value.

## TABLE 1: LUNG FUNCTION TESTS IN A PATIENT WITH SEVERE PULMONARY SARCOIDOSIS

| | *Patient* | *Expected normal value* |
|---|---|---|
| $FEV_1$ (litres) | 2.4 | 3.5 |
| VC (litres) | 2.6 | 4.4 |
| $FEV_1/VC\%$ | 92 | 75 |
| TLC (litres) | 4.3 | 7.0 |
| CO transfer (mmol/kPa/min) | 6.5 | 9.6 |
| Arterial $pCO_2$ (kPa) | 4.1 | 5 to 6 |
| Arterial $pO_2$ (kPa) | 7.3 | 11 to 12.5 |

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# SARCOIDOSIS

## A Case Report

*Roland Guy*
London Chest Hospital

The patient, a 29-year-old housewife, who had been born in Malaya, came to this country nine years ago from Singapore. Apart from intermittent abdominal pain over the last fifteen years, she had been well except for the last two months. During this time she had developed bilateral ankle swelling with pain on touching the anterior tibial shafts. She had a painful right eye, described variously as itching or burning and she felt she had to keep rubbing it. She had had a productive cough, some right posterior chest pain and right temporal headaches. During this time she had lost two stone in weight. There was no family history of tuberculosis and the patient thought she had had a BCG as a child, although no scar was visible. She was a non-smoker and on no medications.

*On examination* she looked well and no abnormalities were detected in the cardiovascular, respiratory, abdominal or neurological systems.

A small right scalene node was palpable and her left ankle was swollen with small violacious areas about one cm in diameter which were slightly thickened over both anterior tibial shafts. These were tender on touching and consistent with erythema nodosum.

## INVESTIGATIONS

*Chest X-ray showed right hilar lymphadenopathy and tomograms showed possible enlargement of the left hilum as well.*

*The Biochemistry* including serum calcium was normal as was the haemoglobin, white cell count and ESR.

*The tuberculin test* (Mantoux 1/1,000) was negative.

*Respiratory function* tests: —

| | *Patient* | *Expected* |
|---|---|---|
| $FEV_1$ (litres) | 2.0 | 2.5 |
| VC (litres) | 2.4 | 2.7 |
| tCO (ml/min/mm Hg) | 15 | 26 |

*Bronchoscopy* showed no visible endobronchial lesion and numerous bronchial biopsies showed no evidence of granulomata.

*An ophthalmic opinion* from Moorfields Eye Hospital was that she had bilateral anterior uveitis and small nodules on her left iris.

The differential diagnosis in this immigrant woman with right hilar enlargement and erythema nodosum lay between sarcoidosis and tuberculosis. The eye involvement favoured sarcoidosis although it was disappointing that the bronchial biopsies were not helpful.

*A Kveim Test Biopsy* was therefore performed which showed a 6 mm papule with 6 mm of erythema at the time of biopsy. The histology showed sarcoid granuloma. Her serum angiotensin converting enzyme was raised at 89 nmol/ml/min (normal 16-52 nmol/ml/min).

The patient was treated with Betnesol and Atropine eye drops and discharged to be followed as an out-patient. She returned three weeks later complaining that her mouth had suddenly felt "odd", with difficulty in eating and saliva dribbling out of the left side of her mouth; she also felt that the right side of her body was weak.

*On examination* she had developed a partial left-sided lower motor-neurone facial palsy and her spleen was now palpable. There was no enlargement of her parotid glands and no abnormal neurological signs on the right side.

However, in view of her symptoms of right-sided weakness, in order to exclude a co-existing basal granulomatous meningitis, the patient was referred for a neurological opinion. Apart from the facial palsy, there were no other clinical signs in the CNS and the patient had normal cerebro-spinal fluid and C.T. Scan with enhancement. The conclusion was that there was no evidence of intracranial sarcoidosis.

Within two weeks her left facial palsy had resolved spontaneously and she felt better. However, three weeks later (fourteen weeks after her initial admission), she developed a lower motor neurone *right* facial palsy and was started on Prednisolone (40 mgs/day) by her General Practitioner. She remained on this alone for a few weeks and the dose was then decreased. Her facial palsy by then had started to improve slightly, and her right hilar glands were smaller.

## CONCLUSION

This case illustrates the diffuse nature of sarcoidosis, some neurological complications and the difficulty in assessing the value of steroids in treatment. Without treatment, spontaneous recovery of her *left* facial nerve palsy occurred within two weeks; with steroids partial recovery of the *right* facial palsy only occurred after two weeks. This does not necessarily mean that steroids delayed recovery, but would we have claimed the efficacy of steroids if it had been the other way round?

I would like to thank Dr. D. M. Geddes for permission to report on his patient.

# REVIEW

## Why Smoking is a Human Issue

*Martin Jarvis*
Addiction Unit, Institute of Psychiatry, London SE5

***The Ladykillers: Why smoking is a feminist issue***
***By Bobbie Jacobson  Pluto Press 1981***
***£1.95***

When cigarette smoking first came under scrutiny as a cause of disease and death the focus was almost entirely on male smoking. This was understandable. Many more men than women were smokers, and men started smoking cigarettes some thirty years before women. It was, by and large, the men who were dying! More recently, while the prevalence of cigarette smoking in men has begun to decline, women have been recruited to smoking in increasing numbers. In the youngest age groups, as many women as men now smoke cigarettes, and indeed among teenagers in the United States girls are now smoking more than boys.

For a while it seemed possible that women were less susceptible than men to the health effects of smoking. It is now, alas, becoming clear that the lower prevalence of smoking-related diseases among women, merely reflected the differing smoking histories of men and women in the older age groups. Lung cancer, for example, is related not only to how heavily people smoke, but also, more importantly, to how long they have been doing it. It is only now that substantial numbers of women with 30-year smoking histories are beginning to accumulate. Lung cancer rates in women are rising steeply and it is estimated that by 1983 it will have overtaken breast cancer as a cause of death in American women.

In response to these developments smoking among women is now receiving attention as a phenomenon in its own right. In 1980 Doll and his colleagues published a female counterpart to their British doctors study, and in the United States the Surgeon General's "Health Consequences of Smoking for Women" appeared. Bobbie Jacobson's book represents a less academic, but nonetheless welcome, addition to this emerging literature.

Ms Jacobson makes some claims about women's smoking which if they could be substantiated would be of considerable importance. She asserts that women's smoking is increasing while men's is decreasing, and that women have more difficulty than men in giving up. She plays down pharmacological explanations of dependence on cigarettes and sees women's difficulties more in terms of sexual politics. It is worth examining each of these claims in some detail.

It is probably somewhat pessimistic to say that smoking in women is still on the increase. Certainly during the 'sixties and early 'seventies the consumption of cigarettes by women was increasing steadily at a time when there had already been a downturn in male smoking. But the data suggest that in this country female smoking prevalence peaked several years ago and is now beginning to decline, albeit very slowly. Part of the increase in female prevalence was due to older cohorts with very low smoking rates dying off and being replaced by younger ones with much higher rates. In the youngest age group, there now seems to be no difference between the proportions of men and women taking up smoking.

Most controversial is the suggestion that women find it harder to give up smoking than men. There is a considerable amount of evidence that can be interpreted in this way, particularly the outcome of a number of treatment studies in smoking clinics. However, those attending smoking clinics are self-selected groups, and a number of possible biases might operate. It is more illuminating to look at rates of quitting in the population as a whole. And here again, at first glance, it might seem that women *do* find it harder or at least give up in smaller numbers. A recent Government survey indicated that 33% of women who had ever smoked cigarettes were now ex-smokers while the corresponding figure for men was 42%.

This figure deserves closer examination, since it is important to make sure that one is comparing like with like. It is only the younger women who have had the same smoking careers as men, and below the age of 35 there is no difference between the sexes in the proportion of ex-smokers. It is the older women, particularly middle-aged women, who have given up in smaller numbers. These women as a group started smoking somewhat later than their male counterparts and their motivation to stop may therefore be less. There is a further complication to comparing proportions of ex-smokers between the sexes. About 10% of men smoke pipes and cigars, while less than 1% of women do. Since many of these pipe and cigar smokers will have previously smoked cigarettes, the male figure for ex-smokers will over-state the proportion who are now true non-smokers; the magnitude of this effect is not known.

The U.K. data do not therefore lend clear-cut support to the idea that women find it harder to give up smoking. If anything they suggest that women and men with similar smoking careers have a similar propensity to give up. Data from the United States tend further to undermine the contention. The recent Surgeon General's report, again reviewing population rather than clinic evidence, concluded that "with respect to the probability of attempting to quit and the success rate, adult men and women cigarette smokers are now indistinguishable".

The claimed sex difference in ease of giving up is one important plank in Bobbie Jacobson's argument. The other related idea is that men and women smoke for different reasons. There is a large and growing body of evidence that points to the overwhelming importance of pharmacological factors, particularly nicotine, in the maintenance of smoking and no evidence that this factor operates differentially between men and women. (Although women smoke slightly fewer cigarettes per day than men and are more likely to smoke low-tar cigarettes with a low nicotine content, recent data indicate that they derive similar nicotine intakes from their smoking.) How can this evidence be reconciled with the book's argument?

Ms Jacobson is aware of the nicotine hypothesis of smoking, but notes that the evidence in its favour is not totally impregnable. She also notes that pharmacological factors cannot explain the large class differential in smoking that has emerged over the past twenty years, and she moves from this to a position where she sees addiction in social rather than pharmacological terms, and pays scant regard to the rôle of nicotine. She is evidently one of that school who are unwilling to concede an important pharmacological dimension to dependence on the grounds that it leads to the smoker's defensive rationalization, "I'm an addict, so I can't stop." This is a pity because there is no necessary conflict between the view that most smokers smoke because they are pharmacologically dependent (addicts, if you like), and the assertion that every smoker is responsible for his or her own smoking and has the power to decide to stop. Addiction should be seen as one input to the decision-making process, not as pre-empting it.

She tells us that 'every woman who discussed her smoking problem with me was convinced she was hooked on nicotine.' She dismisses this introspection, and then goes on to say that the best way to find out why women smoke is to look at "how women themselves understand their need to smoke" — forgetting that she has just summarily dismissed the one factor on which all her interviewees were agreed. What we are then treated to is a series of anecdotal accounts of little value other than that they allow the development of an argument that is couched in terms of sexual politics.

This book left me unconvinced of the existence of any important sex differentials in smoking. This is not to say that there are not ways in which smoking is different for men and for women, and some of these may provide links to feminism more generally. For example, the way in which many women consciously use smoking as a way of regulating their weight clearly has to do with ideas of female attractiveness that are bound up with sex rôle stereotypes. There have been encouraging developments recently in helping people stop smoking, but the book's section on stopping is clearly written by someone who has no practical experience in this area. Self-help groups, though fine in principle, have never been shown in practice to have much contribution to make in smoking cessation. However, the use of nicotine chewing gum, mentioned only in passing in the book, if combined with appropriate instructions and expectations, will I am sure help many motivated women (and men) to become ex-smokers, and put an end to their shared exploitation by tobacco companies.

## PRESS RELEASE

From 1st May Cardiokinetics Limited of Salford will be working in conjunction with Instrumentation Laboratory (UK) Limited as dealers for I.L.'s range of cardiopulmonary equipment in Central and Northern England and Scotland. Amongst the products are a series of oxygen alarms and monitors, a carbon dioxide monitor, cardiac output computer, colloid pressure oncometer and cardiac Q.C. kit.

For further details contact Ken Lee, Cardiokinetics Ltd., 218-220 Regent Road, Salford, Manchester 5 (Tel. 061-872-8287) or I.L. (UK) Ltd. (Tel. 0925-810141).

***CORRECTION TO***

***Page 12, Breath Issue 12, February, 1981***

## DR. FENYVES & GUT

We wish to apologise to Dr. Fenyves & Gut for publishing the incorrect telephone number on their advertisement for Lung Function Testing Equipment for Paediatrics.

***THE CORRECT TELEPHONE NUMBER IS***

***(061) 22 17 21***

# ARTP EDUCATION UPDATE

*Dr. J. Reed*
Department of Physiology, University of Newcastle-upon-Tyne

Since my last note on education appeared in *Breath* (July 1978) virtually every college of further education which offered ONC courses in Medical Physics and Physiological Measurement has changed to the Technician Education Council (TEC) award, the O-TEC Certificate.

In general, the new courses have been quite favourably received, though some colleges have taken the opportunity to restructure their curricula completely, whereas others have done little more than change the name. Perhaps the most serious criticism levelled against O-TEC is that it is too general, providing neither enough specialist teaching nor adequate training.

The DHSS working party had originally recognised this problem and had included in their recommendations to TEC that an additional 'In-Service' module be added to the O-TEC course. Although TEC approved of the general principle, they would not, unfortunately, accept the incorporation of such a module into the O-TEC syllabus, on the grounds that the award is strictly academic and cannot include anything classified as training rather than educational. Eventually, after much discussion, it was agreed that one approach to the problem would be to convert the award from a Certificate to a Diploma.

## TEC DIPLOMA

You will recall that the TEC awards have a modular structure; that is, they are composed of 'units' of study, each unit being of 60 hours — the O-TEC Certificate has 10 units (600 hours) to be completed in 2 years. The Diploma will require a further 10 units but these will be essentially 'Work Experience' units, very similar in structure to the 'In-Service' modules originally prepared for the DHSS Working Party. In this respect, the Diploma seems to circumvent the problems of the Certificate; the first 15 months would be spent gaining multi-disciplinary hospital experience, the next 9 months being devoted to a single elective discipline. This emphasis on practicality and the more specialist aspects of Medical Physics and Physiological Measurement will necessitate specialist teaching and equipment which in turn will require specialist lecturers and the use of laboratories and other facilities away from the colleges. In the present economic climate, the unhappy result of such a system will be staffing and funding problems. All these facets of the proposed Diploma course are presently being discussed by DHSS, TEC and FAMT representatives and you will be kept informed of any developments.

Advantageous as the Diploma may appear to be, it has not been universally welcomed. There are those who fear that such a work-orientated course will be seen as an end in itself, and will hinder if not stop completely, progression to a more advanced level of study. This view has some justification as it is well-known that the DHSS are not entirely in favour of a higher qualification for Medical Physics and Physiological Measurement technicians.

## THE H-TEC AWARD

The H-TEC, an advanced course of study leading to a higher award, *will* however be available. About 18 months ago TEC set up a committee to look into the need for a higher qualification for technical staff in the NHS. This committee found in favour and went on to delineate the aims, objectives and eventual structure of such a programme. The guidelines for Higher Certificate Programmes were produced a short time ago, and are now being circulated. To summarise, the 'H'-TEC course is aimed at those who hold or are aiming at, senior posts in *Medical Physics*, *Physiological Measurement* or *Medical Instrumentation*. The award will probably be the final qualification achieved by most students, but TEC expect that it will be recognised as contributing towards a professional qualification or a degree for those with the ability and inclination to carry on. In content it is structured in a similar way to the O-TEC, in that it is modular and has a common core curriculum with specialist subject options. These are listed in Tables 1 and 2.

As you can see, the academic programme is not particularly specialised, nor is there any practical bias. For this reason a project has been included, of approximately one unit in length, which will presumably be in the candidate's specialist field.

To date, two colleges of further education have submitted H-TEC courses to the TEC validation committees, and have had their submissions provisionally accepted. This means that it is virtually certain that these colleges will be offering an 'H'-TEC course by the end of this year.

The onus is now on individual members to persuade their Heads of Departments that such a qualification is advantageous and is worth the time and money spent. Good Luck!

### TABLE 1: STRUCTURE OF THE H-TEC

| *Common Core Units* | *Unit Value* |
|---|---|
| Physiological Measurements | 0.5 |
| Human Physiology | 1.0 |
| Principles of Radiation | 0.5 |
| Biomedical Uses of Radiation | 0.5 |
| Mathematics, Computing and Data handling | 1.5 |
| Biomedical Electronics (A) | 1.0 |
| Biological Science (A) | 0.5 |
| Plus | |
| *either* Biomedical Electronics (B) | 0.5 |
| *or* Biological Science (B) | 0.5 |

### TABLE 2: SPECIALIST SUBJECT OPTIONS FOR THE H-TEC

| **Physiological Measurement Options** | *Unit Value* |
|---|---|
| Physiological Measurements | 1.5 |
| Physiological Measurements Instrumentation | 0.5 |
| Clinical Measurement Studies | 1.0 |
| Project | 1.0 |
| **Medical Physics Options** | |
| Radiation Technology | 1.5 |
| Radiation Technology Instrumentation | 0.5 |
| Clinical Measurement Studies | 1.0 |
| Project | 1.0 |
| **Medical Instrumentation Options** | |
| Physiological Measurements Instrumentation | 0.5 |
| Radiation Technology Instrumentation | 0.5 |
| Electronics | 2.0 |
| Project | 1.0 |

# SPRING MEETING OF THE ASSOCIATION

The Spring Scientific Meeting took place at Hope Hospital, Manchester on Saturday, April 4, 1981. We are most grateful to Margaret Marples and her colleagues for arranging a stimulating meeting, to the speakers for their interesting papers and to Mr. J. Bancewicz for so kindly acting as chairman. A very reasonable attendance was achieved despite the rival attractions of the Boat Race and the Grand National.

We owe grateful thanks to the following firms who sponsored the meeting and put on demonstrations: —

Gould Medical
Instrumentation Laboratories
Beckman RIIC
Boehringer Ingelheim
P. K. Morgan

The film 'Airways Control' was shown by courtesy of Boehringer Ingelheim.

The following papers were given: —

## THE RELATIONSHIP BETWEEN GASTRO-OESOPHAGEAL REFLUX AND ASTHMA

Dr. D. Cooper. Department of Thoracic Medicine, Hope Hospital.

Dr. Cooper reported on his detailed investigations into this relationship. It was found for instance that intubation of the lower oesophagus could, by itself, produce a fall in expiratory peak flow and that medical treatment of reflux reduced the clinical incidence of asthma. The precise mechanisms are not yet worked out and clearly this interesting subject merits further exploration.

## ARTERIAL OXYGENATION DURING ONE-LUNG ANAESTHESIA

Dr. E. Bradshaw. Department of Anaesthetics, Hope Hospital.

Dr. Bradshaw discussed the difficult problems that may arise during thoracic surgery when anaesthesia is maintained through one lung alone. Large shunts can develop and even on 50% oxygen the patient may be hypoxaemic. The site of these shunts appears to be unknown; half the audience favoured the upper lung and the other half favoured the lower.

## EDUCATION UPDATE

Dr. J. Reed. Department of Physiology, University of Newcastle-upon-Tyne.

Dr. Reed reviewed the lengthy negotiations about technical education in the field of Respiratory Physiology. The curricula and examination standards now seem to have been worked out, but how it's all going to be organised and who is going to organise it, is not yet entirely clear! (See article elsewhere in this issue.)

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# CORRESPONDENCE

There seems to be a gross misunderstanding of the policy of the FAMT with regard to the T.E.C. This is a statement to make matters clear, once and for all!

## "O" TEC

At the moment, after two years' study at student grade, a T.E.C. Examination is taken and if the student passes it, he/she is awarded a TEC *Certificate*.

*However*, the certificate does not give any credit for the considerable amount of *academic* content of the *in-service* work during these two years.

*Therefore*, negotiations are going on at the present time to convert the award from a *certificate* into a *diploma*, but a decision has not yet been made as to whether this is possible. *If it does happen, the diploma will be instead of a certificate.*

*If* the diploma is awarded instead of the certificate, this will in *no way* take the place of "H" T.E.C.

## "H" TEC

Negotiations are going on at the moment with the TEC representatives and the colleges of further education and the F.A.M.T. towards formulating a suitable course, which will be at a considerably higher level than the "O" TEC courses and will be comparable to the old HNC.

I hope this is now quite clear. If there are any further queries, please contact me.

DOROTHY BATTYE
01-703 6333 ext 33

*The following letter was sent to Gillian Lowe by the Honorary Secretary of SALT. Anyone who would like to attend should contact Mr. Ball at the address given below.*

Division of Anaesthesia,
Clinical Research Centre,
Watford Road,
Harrow,
Middlesex, HA1 3UJ.

28th April, 1981

Dear Mrs. Lowe,

Sally Gough, as Secretary of the Central Council of the F.A.M.T., has suggested that I notify the secretaries of the member societies and associations of the F.A.M.T. of our forthcoming scientific meetings.

The next scientific meeting of the Society of Anaesthetic Laboratory Technicians will be held in Plymouth on Friday and Saturday, 11th and 12th September, 1981. This will also be the Annual General Meeting.

Further details of the meeting are available from Mr. J. D. Ball, Department of Anaesthetics, Royal Naval Hospital, Plymouth, Devon.

Yours sincerely,

Anne M. Rhodes (Mrs)
Hon. Sec. S.A.L.T.

Thoracic Out-patients Department
Llandough Hospital
Penarth
CF6 1XX
Tel.: 0222 705411

Dear Editor

**O TEC Physiological Measurement**

With reference to my dispute with South Glamorgan Health Authority's Area Training Officer, South Glamorgan Institute of Higher Education and the Technician Education Council, I am happy to say that on the 11th March 1981 at the Welsh Office, the Institute's representatives agreed to reinstate the original respiratory physiology section in the second revised submission. I did, however, have to concede to their wish, and that of the Welsh Office Scientific Adviser, that the section also include content relevant to work in Theatre and Anaesthetic Departments. However, I was able to negotiate that the complete section be allocated approximately twice as much time as the others, so we have not lost out in any way.

I do hope that I do not experience so much difficulty with the H. T.E.C. submission!

Yours sincerely,

KELVIN HOUSTON,
Regional Organiser,
Association of Respiratory Technicians and Physiologists.

# PLEASE NOTE

## Job Evaluation of Operating Department Assistants with Reference to Physiological Measurement Technicians

Dear Colleagues,

A number of you have been asked by the DHSS to take part in a survey with reference to the above.

Questionnaires have been sent out and these are not to be regarded lightly. Please give careful thought to your answers as these could be used again in the future should the DHSS ever wish to compare our work with, say, that of a Medical Laboratory Scientific Officer.

Members of the team of Officers from the DHSS will be visiting the Departments in the Hospitals concerned. Again the onus will be on us to make our work as varied and as technically absorbing as possible on these visits. It is not just Respiratory Physiology that is being assessed, but all branches of Physiological Measurement.

Should you be having any difficulty with this survey, do not hesitate to contact me.

S. E. Gough,
Respiratory Physiology Department,
Papworth Hospital.

**The FAMT**

I read with interest the article on the FAMT in the February issue of 'Breath' and would like to reply to the criticism of general apathy among its members. I have to agree that attendance at FAMT general meetings is poor, but what steps have been taken by the FAMT or its individual associations to find out why? If the members were asked why they do not come, they would probably, like myself, reply 'pressure at work, finance or lack of knowledge of the topics on the agenda'. I would find it hard to attend even one meeting a year.

I feel that most of the work done at general meetings could be done through the regional organisers, who could obtain the members' reactions on subjects due for debate. The FAMT should have a page in the journals of all member societies and associations. Intending speakers at FAMT meetings could have their text published in advance in the journals; votes on each motion could be invited, which I am sure would meet with a better response than 57 out of 1,500. I would like to know for instance, more details of the proposed changes in education.

Just lecturing the members on their failings won't help!

BRIAN McHUGH
Pulmonary Function Laboratory
Forster Green Hospital
Saintfield Road
Belfast BT8 4HD

**A reply from Sally Gough, Hon Sec FAMT**

I am grateful to Brian McHugh for his comments and will bring these forward at the next Council meeting.

I have already stressed to the FAMT Central Council the importance of keeping the membership informed and feel that individuals on the Central Council with special interests should publish reports in the journals of the eight constituent disciplines. My letter was in fact, the first such report and I hope that it will encourage others!

# ARTP NEWS

## AMENDMENTS TO THE CONSTITUTION

I have written to the Secretary and suggested the following amendments to the Constitution of the Association.

Rule 5. **COUNCIL OF THE ASSOCIATION**

5a) For 24 Full or Junior members read 21.
For Scotland 4 read 1.

This is not discriminating against the Scots, it is simply because we have only one member for Scotland!

We also find it very difficult to hold a council meeting because there are generally less than 8 council members present at the meetings. I therefore propose an amendment to Rule 5c so that it reads: "A quorum of the Council shall be seven." This would seem to make sense as we are cutting down on our Scottish representatives.

Rule 8 **ASSOCIATION NEWS BULLETIN**

8b) I propose that sub-editor be inserted so that it reads Editor/Sub-Editor.

Rule 10 **RULES**

It was proposed to amend 10b but I think this should be left as it is. An amendment can therefore be passed provided that two-thirds of the actual votes cast are in favour of the amendment. This means that members who do not vote do not affect the decision.

Rule 11 **FINANCE**

I propose that 11a should read as follows: "The annual subscription shall be £7.00 for full members and £5.00 for junior members, or such sums as may be subsequently decided by the Council of the Association. The subscription shall be payable in advance and shall be due on the 1st May each year."

11b) I propose that 11b be deleted.

Because of the financial situation it may be necessary to alter the annual subscription at short notice. The Council may have to make a quick decision on the Treasurer's advice.

These are the amendments that I propose. If any other members have proposals will they please write to the Secretary by the 1st August.

## THE ANNUAL GENERAL MEETING

The AGM will be held during the first two weeks in October on a Saturday. Offers are invited from members for a venue for this meeting. The Executive Committee feels that it is more interesting to hold meetings in different parts of the country and is anxious to avoid the Association becoming London-based.

If anyone is interested in offering their hospital for the meeting, would they please contact the Secretary, Gillian Lowe, who will give you details of numbers to cater for, etc.

Gillian Lowe,
Cardio-Thoracic Unit,
Derbyshire Royal Infirmary,
London Road, Derby.
Tel: 0332 47141.

## P. K. MORGAN LTD.

P. K. Morgan recently held a very interesting seminar for their agents abroad, to which both Kelvin Houston and I were invited. Jim Reed was also there but he was working! For each aspect of Respiratory Function – for example Exercise Testing, Body Plethysmography, Dm and Vc – there was a technical explanation from one of P. K. Morgan's staff followed by a physiological and clinical lecture by a doctor. After this we were all let loose on the appropriate machines!

Those of us who went, felt that not only had it been very enjoyable but also extremely useful in presenting a whole picture of each aspect of lung function and its measurement. We suggested to Philip Morgan that members of ARTP would benefit from this type of seminar (and also many junior thoracic doctors). He felt that the Association should avoid any alliance with a commercial firm for its annual meetings, but said that he would be happy to consider such an idea for a special teach-in if members were interested.

He also thought we could arrange similar seminars with other commercial firms at a later date if it were a success. If anyone would be interested or has ideas on the subject, could they write to me, or to Kelvin Houston at Llandough Hospital.

## MESSAGE FROM THE SECRETARY

Elections for regional organisers are now due and your representatives should be contacting you in the near future.

It is vital that the regional organisers are prepared to play an active part in the Association. This involves attending Council Meetings approximately 2-3 times a year and passing on information to your region.

The original idea of having a Council was to avoid a minority running the Association which is what often happens and is in danger of happening now. Please make sure the person you elect is prepared to represent you properly. Why not take on the job yourself?

The Annual General Meeting is to be held during the first two weeks of October. The date and venue has still to be decided.

If any member wishes to propose any constitution amendments they must let me have them in writing by 1st August 1981 AT THE LATEST.

Gillian Lowe
Secretary

And finally

## AN EXCERPT FROM A REFERRAL LETTER!

On examination he was anxious but denied it. There were very poor breath sounds over the left lung which is not surprising bearing in mind the old operation but nothing in the right lung. Breathing tests seemed typically erotic to me with lots of pausing during the expiratory phase and there was no response again to Ventolin. However he vigorously denies any feeling of anxiety and persists in his story.

# EXECUTIVE COMMITTEE OFFICERS for 1980-81

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Tel: 061 789 7373 Ext. 115

*Temporary Stand-In:*
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   Cardio/Respiratory Lab.,
   Nevill Hall Hospital,
   Abergavenny.

3. Mrs. B. Bateman,
   E.C.G. Dept.,
   Singleton Hospital,
   Swansea,
   West Glamorgan.

*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.

Applications for advertisement space and for rates should be addressed to: Jane Jones, Respiratory Laboratory, London Chest Hospital, Bonner Road, London E2.

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