



Inspire

The Official Journal of The Association of Respiratory Technicians and Physiologists

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FIRST WORD

In this edition of INSPIRE an account of the ARTP Winter Meeting in Pontefract, including abstracts and a report of the AGM, a major article on transcutaneous blood gas analysis, news of a brand new service for all ARTP members—developed and run by the Association, Health & Safety News, review of recent articles, calendar of forthcoming events and much, much more!

The ARTP—the Association for the Millennium?

Time for a change of image and name? The ARTP Executive Committee believes it is. Over the years the Association has worked hard to improve the image of the profession at the Department of Health, throughout the NHS and with other professional bodies. We have an extremely useful working relationship with the British Thoracic Society (BTS), which has led to a number of successful on-going ventures, namely the provision of post basic advanced training courses, the publication of the National Guidelines for the Measurement of Respiratory Function, and the yearly joint seminar at the BTS summer meeting. The Association has also looked beyond its National borders with involvement in the European Respiratory Society (ERS). The ARTP has been a major player in the creation of a new assembly within the ERS, the Respiratory technology and health care Section.

The days when technicians were seen as the 'hand-maidens', or 'yes men' to the consultant are long gone (thank goodness!). Technicians are the experts, and as such should advise on the most appropriate tests for a patient, undertake their own research, identify needs, manage service delivery and construct future developments and organisational planning.

At the end of the 1980's (88/89) people employed on the Whitley Council B pay scales (physiological measurement, medical physics, pharmacy, dental and medical photograph technicians) underwent a name change to Medical Technical Officers (MTO's). 'Equivalent' grades in pathology, biochemistry and other 'medical laboratories' had long been known as medical laboratory scientific officers. Now we believe it is time the Association reflected the change and removed 'technician' from its own name. Do you think the generic name 'technician' has misplaced and out-of-date connotations associated with it, and does this lead to misrepresentation of the profession? Please let us know your views, and if you think a new name is appropriate make a few suggestions.

For starters

- Association for Respiratory Physiology (ARP)
- or Association for Respiratory Function (ARF)
- or Association for Respiratory Measurement (ARM)

Write to me with your opinion and your own suggestions:

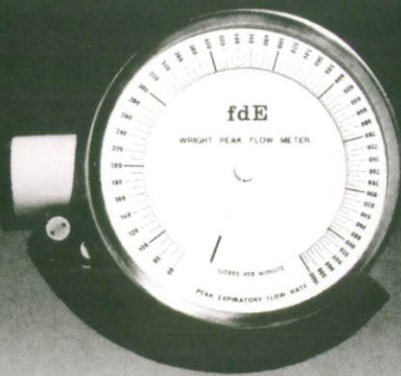
Miss Sue Revill (Editor)
Department of Respiratory Medicine
Glenfield Hospital
Leicester LE3 9QP

DATES FOR YOUR DIARY

See page 3 for more details

15th–19th April	Short Course in Advanced Respiratory Physiology Coventry University (HNC Specialist Option)
18th–19th April 1996	ARTP/BTS Joint Advanced Course Diagnosis and Treatment of Sleep Disordered Breathing Organisers Mrs Sue Bradbury and Dr Martin Allen Department of Respiratory Medicine City General Hospital, Stoke-on-Trent
22nd–25th April 1996	ARTP Short Course on Basic Respiratory Function Measurement Queen Elizabeth Hospital Birmingham
3rd July (pm) 1996	ARTP Summer Meeting Programme to be announced
4th–5th July 1996	University of Warwick Coventry British Thoracic Society Summer Meeting including ARTP/BTS Joint Seminar
7th–11th September 1996	European Respiratory Society Annual Congress (The ERS) Venue: Stockholm, SWEDEN
16th–20th September 1996	Short Course in Advanced Respiratory Physiology Coventry University (HNC Specialist Option)
9th–11th December 1996	British Thoracic Winter Meeting Queen Elizabeth 11 Conference Centre London

Continued on Page 2



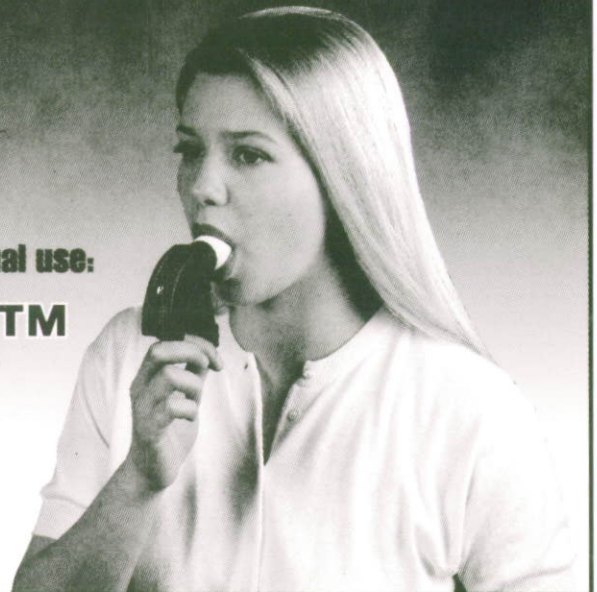
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FIRST WORD Continued from front page

ARTP OFFICERS

Mrs Julie McWilliam has recently left the Executive Committee. The birth of Julie's first child is imminent. Our thanks go to Julie, and the Cardio-Thoracic Measurement Department at the Derbyshire Royal Infirmary, for all their commitment and hard work for the ARTP over many years.

Dr Roger Carter (Glasgow Royal Infirmary) takes over as Secretary to the Association, and Mr Steve Scholey (Pontefract General Infirmary) becomes membership secretary.

Following a postal vote Miss Julie Lloyd (Goodhope Hospital, Sutton Coldfield) takes over as Association Treasurer. Our thanks go to Mrs Evelyn Smith who has filled this post admirably over the last few years.

All enquiries concerning membership should be addressed to:-

Mr Steve Scholey
 Chest Unit, General Infirmary
 Pontefract
 West Yorkshire WF8 1PL
(WRITTEN ENQUIRIES ONLY)
THANK YOU

Continued from front page

REMINDER: Return of grading questionnaire

DEADLINE: February 28th

There is still a large number of questionnaires to be returned, please spare us 20 minutes of your time and complete the questionnaire and return it to Dr Brendan Cooper, City Hospital Nottingham.

Thank You

ARTP/BTS NATIONAL ASSESSMENT

Call for candidates
 The assessment for 1996 will be taking place in August and September.
 Application forms are enclosed with this copy of INSPIRE
CLOSING DATE 28TH MARCH

These should be sent to our Examination Secretary along with all other enquiries

Claire Thomas
 (Education Secretary)
 Clinical Measurement Unit
 Manor Hospital
 Moat Road
 Walsall WS2 9PS

Tel: 01922 656583

CALL FOR ABSTRACTS

Abstracts are invited for the ARTP Summer Meeting (3rd July 1996). The abstract for a short presentation (15 minutes) should be typed on one side of A4 paper, with single spacing, and include title, authors and places of work. Abstracts for posters are also welcome.

Please Send To

Dr Sue Hill (before 12th April 1996)
 Lung Investigation Unit
 Nuffield House
 Queen Elizabeth Hospital
 Edgbaston
 BIRMINGHAM

Calendar of Forthcoming Events

MEETINGS

27th-28th FEBRUARY

2nd National Conference on Asthma Education and Management

Kensington New Town Hall
London

This conference is aimed at a multi-disciplinary audience, for anyone working in the field of asthma care

For more information contact
The National Asthma Campaign
Tel: 0171 226 2260

3rd JULY (pm)

ARTP Summer Meeting

University of Warwick
Programme to be announced

4th-5th JULY

University of Warwick
Coventry

British Thoracic Society Summer Meeting

Including the Joint ARTP/BTS seminar to which all ARTP members are invited. In celebration of the 150th anniversary of Hutchinson making the first measurement of FVC the topic will be dynamic lung volumes. ARTP members (& non-members) can register for one or both days through the BTS office, or register on the day at the meeting venue.

7th-11th SEPTEMBER 1996

European Respiratory Society Annual Congress (The ERS)

Venue: Stockholm, SWEDEN
(Bursaries are available for this meeting. Please see page 11 for details.)

NB all bursary applications must be received before 1st July 1996).

This meeting will contain a number of sessions organised by the Respiratory Technology and Health Care Section. The sessions are aimed at

technicians, scientists and other associated health care workers. There will be two post-graduate workshops on Saturday 7th Sept., and in the main body of the Congress the section has been awarded 2 major symposia, an assembly symposium as well as poster sessions.

A business meeting of the section will also take place to which all members of the ARTP are invited. Please support this very important European venture.

9th-11th DECEMBER

British Thoracic Winter Meeting

Queen Elizabeth 11
Conference Centre
London

A call for abstracts for this National meeting will be appearing later in the year, and the final programme will be circulated to BTS members in December. There are usually several sessions of interest to respiratory function technicians. ARTP members are able to attend as guests. The fee is usually around the £15 per day level.

COURSES

15th-19th APRIL

Short Course in Advanced Respiratory Physiology.

Coventry University
Course is still recruiting

Target audience: Technicians working in Respiratory Function wishing to update and extend their knowledge, as well as students following the National BTec HNC.

Topics include:

Respiratory exercise testing, and interpretation

Sleep studies, CPAP and nasal ventilation

Inhalation therapy

Ventilatory control

Bronchial challenge testing

FEE: £125 for the whole week.
Day fee £25.

DISCOUNT FOR ARTP MEMBERS

Reduced fee for ARTP members-£112.50 (£22.50 day fee). Please state your ARTP number on the application form to claim the reduced fee. Please note the application form for the Coventry Course is enclosed with this copy of INSPIRE

Participants may attend for the whole week, part of the week or just for one day.

INTERESTED? Post your application form NOW!

18th-19th APRIL

Diagnosis and Treatment of Sleep Disordered Breathing

Organises Mrs Sue Bradbury and Dr Martin Allen

Department of Respiratory Medicine
City General Hospital
Stoke-on-Trent

This is a must for all technicians and medics involved with the diagnosis and treatment of sleep disordered breathing. As well as lectures and workshops, there is hands-on experience with a wide range of diagnostic equipment, as well as nasal ventilators and CPAP equipment with real-life patients. This course was run in 1995, and heavily over subscribed, so hurry and book your place now.

PRICE: £190 (includes course fee, overnight accommodation (4 star hotel), conference meal and lunch)

Contact:

Mrs L Farnsworth
01782 741251

to book your place NOW
(numbers limited to 30)

22nd-25th APRIL

Queen Elizabeth Hospital,
Birmingham

ARTP Short Course Basic Respiratory Function Measurement

This course complements the ARTP/BTS National Assessment and is a must for all technicians registering for the National Assessment this year.

It is also extremely valuable for ALL technicians training in respiratory function. The course includes anatomy, physiology, measurement of dynamic lung volumes and flows including flow volume curves, reversibility testing, transfer factor and static lung volumes.

Applications forms will be posted to all ARTP members during February.

16th-20th SEPTEMBER

Short Course in Advanced Respiratory Physiology.

Coventry University (HNC Specialist Option)

Topics include:

Flow volume curves

Respiratory Muscle Function Measurement

Blood Gas Analysis

Lung Volume Measurement

CO Gas Transfer

Skin Testing

FEE: £125 for the whole week.
Day fee £25.

DISCOUNT FOR ARTP MEMBERS

Reduced fee for ARTP members-£112.50 (£22.50 day fee).

Participants may attend for the whole week, part of the week or just for one day.

APPLICATION FORM ENCLOSED



REPORT OF THE ARTP WINTER SCIENTIFIC MEETING

PONTEFRACT GENERAL INFIRMARY NOVEMBER 1995

The meeting was held in the Post Graduate Centre at Pontefract General Infirmary. It began lunch time on Friday 24th November with a trade exhibition which was well supported by many of the manufacturers. Each session was attended by approximately 60 people.

The afternoon session began with Dr W Biernacki giving a talk on the use of CT scanning in the diagnosis and quantification of emphysema and chronic bronchitis. He discussed the advantages—namely it is non-invasive and the data obtained is very specific regarding location and quantity of the emphysema. However, as a procedure it is expensive, not widely available and causes problems with claustrophobic patients. Dr Biernacki questioned the usefulness of scanning as the result has very little influence on the patients management or treatment.

Sue Revill talked about her own research into the selection of treadmill or cycle ergometry to measure exercise capacity in severe COAD. Ms Revill outlined her study protocol. The results indicated that despite there being advantages and disadvantages to both methods the most suitable can not be predicted and a decision should be made on the information required from the study and the individual patient.

Dr Peake then gave a very informative talk on interstitial lung disease, its causes, the expected results of lung function tests and X-ray which could be expected in different forms of the disease, including pneumoconiosis and extrinsic

from CLAIRE THOMAS, WALSALL MANOR HOSPITAL

allergic alveolitis. He then discussed prognosis, treatment and management. It was felt that lung function tests are a useful method for the diagnosis and monitoring of treatment.

Dr Brendan Cooper then lead a lively open floor discussion on the draft grading guidelines. The results of the recent survey were tabled, as were the comments which had been received on the draft proposals. A revised set of guidelines will be sent to everyone who responded to the survey.

After the Chairpersons, Education and Treasurers report a drinks reception was held with the manufacturers followed by a lively dinner at the Kings Croft Hotel. Many of the conference delegates only reluctantly retired to bed in the early hours after the Hotel management forced the bar to close!

Saturdays session began with Dr Johnson outlining guidelines for assessing the suitability of patients with severe COAD to fly. Dr Johnson explained why flying can have such serious repercussions. He highlighted the contraindications related to pressure, hypoxia and infection. Dr Johnson then suggested some useful methods of assessment including simple walk test, prediction formula and hypoxic challenge test.

He summarised by saying that the airline should be advised immediately. The patient should check whether there are any financial implications or any stop over points on the flight.

Dr Elliot, from Leeds, gave a comprehensive talk on Nasal Ventilation, the type of patients who may benefit and how it can be useful in avoiding intubation. He explained that it is easy to manage at ward level, it is easier to wean than endotracheal ventilation and it can in some instances reduce hospital stay. On the down side however he did point out that it is less efficient and not as easy to control.

Dr Martin Allen of Bradford gave an informative talk on the worlds biggest killer—TB. He explained why it is on the increase again, how treatment is altered and how patients are currently being managed in Bradford which has a particularly high incidence of TB.

Professor Chrystn from the School of Pharmacy at Bradford University presented data from several studies investigating the efficiency of different types of inhaled drug delivery method. Data was also presented on how patient technique affects delivery dose.

ANNUAL GENERAL MEETING

Chairwoman's Report 1995

Dr Sue Hill,
Lung Investigation Unit,
Queen Elizabeth Hospital,
Birmingham

This has been a year of consolidation for the Association, particularly with a change in personnel undertaking some of the activities and difficulties with schedules and changing job roles. However we have made progress and I will highlight some of the salient activities.

MEETINGS

I am pleased to report that this has been a year of meetings. We held two successful ARTP meetings. The first was in Nottingham which preceded the Summer meeting of the British Thoracic Society, where an excellent symposium on Exercise Testing took place. At our own Summer meeting we had a good attendance with a varied programme and much lively discussion. Our thanks to Brendan Cooper and Sue Revill for all their hard work.

Our Winter meeting was held in Pontefract with an excellent attendance. There were a number of topics covered in the programme from physiological assessment of patients with lung disease flying at altitudes to an update on tuberculosis. At this meeting we re-introduced a format that we used successfully during the early eighties, where the meeting started on Friday lunchtime, was concluded by a reception with the Manufacturers and then followed by a delegates Dinner. The second part of the scientific meeting being held on Saturday morning.

We are continually looking at the format of all our meetings to try and find one that is more successful and acceptable. At the Winter meeting we circulated an evaluation form which we hope will give us the feedback to direct the planning of future meetings.

The other meetings held this year have been organised in conjunction with the British Thoracic Society. We held a very successful Workshop at the National Exhibition Centre

in March 1995 to discuss the Lung Function Guidelines published the previous year in Respiratory Medicine. I am sure that all those of you who attended this workshop were amazed that the Atrium at the National Exhibition Centre was such an inviting venue. We

I have now taken over the chair from Duncan Hutchison who I would like to thank formally for doing such an excellent job, particularly with regard to the publication of the Lung Function Guidelines. There have been a number of other retirements from this

own Summer meeting will be held on 3rd July.

CLINICAL SCIENTISTS IN RESPIRATORY MEDICINE

The ARTP is now officially represented at the Department of Health Liaison Committee for Clinical Scientists in the NHS and has been a founder member in the formation of a national Conference of Clinical Scientist Organisations. As mentioned in previous articles, clinical scientists are a staff group which have been specifically targeted by the NHS Management Executive for their potential involvement in research and development within the NHS. The production earlier this year of the Culyer Report gives even greater emphasis for research and development in the NHS, particularly since Trusts can benefit from research monies from the R component of SIFTR monies.

I would like to see the opportunity for more technicians to convert to clinical scientist grades and to undertake more specific areas of activity in Respiratory Medicine. As part of our ART/BTS joint activities we are continuing to develop a MSc level qualification in respiratory physiology which we hope will be available in the near future.

EUROPEAN RESPIRATORY SOCIETY

As many of you are aware in 1994 I was elected as chairman of the new section within the European Respiratory and Society entitled "Respiratory and Technology and Health Care". This section has 3 scientific groups which represent health care workers allied to respiratory medicine (Respiratory Measurement, Respiratory Physiotherapy and Respiratory Nursing). I am pleased to report that membership to this section is increasing and that there were excellent submissions to the section at the recent 1995 ERS Congress held in Barcelona. During the programme of this congress a section symposium was held on Lung Volumes where two ARTP members

gave excellent presentations. I would like to formally thank Angela Evans and Adrian Kendrick for their contribution. In addition from the abstracts submitted to the section, presentations were given in an oral session and also in a poster session. Grateful thanks to all contributors and to those of you who supported the activities of this section at the meeting.

The ERS Annual Congress in general is an excellent meeting for all people working in respiratory medicine with much to interest lung function staff. The 1996 Annual Congress will be held in Stockholm between September 7th and 11th. An exciting programme has been planned starting with postgraduate courses on Saturday 7th on Practical Aspects of Body Plethysmography and Respiratory Physiotherapy—practical aspects and theoretical basis. In the main programme of the congress the section has major symposia on Respiratory Nursing, Mucus transport and physiotherapy and Assessment and monitoring of pulmonary diseases; lung function impairment versus perception of disease together with an assembly symposium on Practical Aspects of Clinical Exercise Testing. There will also be free communication sessions based on abstracts submitted for presentation to the section. Can I please encourage you to submit an abstract which may be a case study, preliminary results from a clinical study, evaluation of a new technique/equipment and comparison of testing procedures. Abstract submission forms are available from me and the deadline for receipt of abstracts is February 19th. I would be more than happy to advise or help anyone. Can I also ask you to encourage your respiratory nursing and physiotherapy colleagues to participate.



Trade Exhibition

had a terrific response in terms of numbers wishing to attend and we now have a waiting list of people who would like to attend a second workshop. We apologise, however, for the alteration in format which meant the Afternoon practical session was not as user friendly and informative as planned; this was due to circumstances beyond our control. We are pleased that the manufacturers have agreed to sponsor a number of other workshops on topic specific areas, such as the Measurement of Dynamic Lung Volumes and Flows, Static Lung Volumes etc. so watch this space and remember to book early. We also held an extremely successful ARTP/BTS Short Course on The Interpretation of Lung Function Tests. Again we received an excellent response and we have enough people on the waiting list to run a further 2 courses. This short course resulted in a generous profit for the British Thoracic Society which we hope will be used to pursue other joint activities.

LIAISON WITH BTS

We continue to go from strength to strength with the British Thoracic Society and we are now seen as the reference point for all aspects relating to respiratory physiology laboratories and the measurement of lung function.

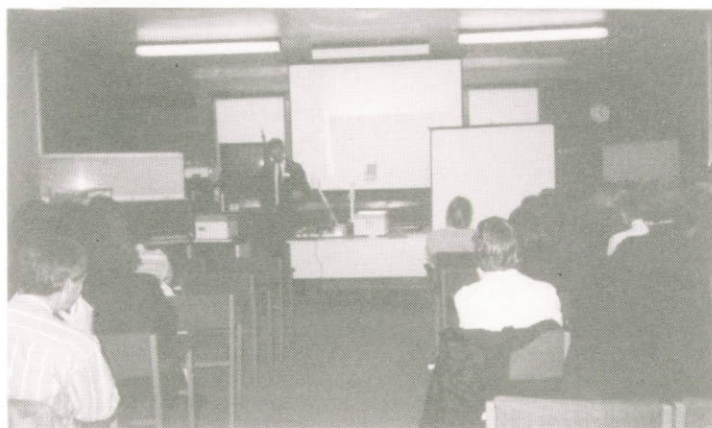
committee, most notably Sue Revell the Secretary who I would like to thank formally for her contribution. New ARTP additions to the committee are Steve Scholey (Pontefract) and Evelyn Smith (Bristol).

We continue to look at producing and hopefully publishing guidelines for other measurements of lung function and for other issues that relate to lung function laboratories such as infection control. We are also looking at whether we should set up a system of accreditation for lung function laboratories in a similar way to that used for pathology laboratories. This obviously has enormous implications for issues such as staffing and equipment levels within departments which need to be fully discussed and explored with all interested parties.

We are currently planning a joint ARTP/BTS symposium to celebrate the 150th anniversary of the first measurement of vital capacity by Hutchinson at the summer meeting of the BTS which is to be held at the University of Warwick on 4th/5th July. Please support this symposium and celebrate this important milestone in the history of Respiratory Function. Our

To encourage attendance there is a reduced congress fee for attendees associated with section 9 which is a considerably reduced fee and equates to 1 year's free membership. This reduced tariff applies whether or not you are an ERS member. All non-member respiratory health care professionals registering for the congress will automatically receive the last 3 issues of the 1996 European Respiratory Journal, and become Associate members until December 31, 1996. Registration forms are also available from me.

Another important development is the agreement by the ERS Executive Committee to offer reduced membership fees for people who wish to join section 9. This reduced membership fee will entitle members to all the benefits of full membership including reduced congress



Dr B Cooper

fees and newsletter/publications, with the exception of receiving the European Respiratory Journal. If you are interested in joining, or if any of your colleagues in Respiratory Medicine are interested in joining, please contact me for an application form.

The ERS is very much a people driven society so help me develop the practice of Respiratory Technology and Health Care throughout Europe.

MANUFACTURERS

I would like to thank the manufacturers for their continuing support particularly in a rather difficult financial climate. I am pleased to say that we seem to have much more support for our meetings at present and therefore perhaps this is an encouraging sign that things are not as gloomy for our industrial colleagues as they were in the past. I am pleased to announce that to pursue issues from both sides of the fence we have appointed a manufacturer's liaison officer. Dr. Brendan Cooper (City Hospital, Nottingham) has kindly agreed to take on this role where any specific points that you would like raised with the manufacturers which may be relevant to other users can be discussed. We also hope that this will provide a forum for the manufacturers to bring to us new developments or ideas/suggestions where they would like professional input.

We hope also to gain their support for our educational objectives and for the development of our bursary scheme.

MTO GRADING GUIDELINES

Even though this may appear rather late in the day we have developed draft guidelines to help with the grading of Medical Technical Officers. The official guidelines, as many of you will be only too familiar, have lacked the specific detail required to help the internal grading process. I would like to thank Brendan Cooper and Steve Scholey for all the work that they have put

in devising the questionnaire to accompany the guidelines and for analysing the results. I thank you all for completing this questionnaire and for your comments on the guidelines. Please complete them if you have not already done so as the data will provide a valuable source of information that we are unable to obtain elsewhere. We hope to use this information to direct both ARTP and other relevant national policies in the future.

PSM ACT

The Professions Supplementary to Medicine Act 1964 is currently under review. At present this act generally covers the therapy professions and this has not encompassed staff groups making measurements on patients. I am pleased to report that both myself on behalf of the ARTP and the BTS have replied suggesting that Respiratory Technologists and Clinical Scientists working in respiratory medicine should be included under the umbrella body which would help improve our status and recognition within the NHS. We will keep you all informed of the outcome of the review.

INSPIRE

On behalf of the ARTP I would like to thank Sue Revill for all her hard work in producing an extremely news-worthy and user friendly magazine for the Association. She is always very happy to receive contributions so please help her to fill the space and make it your journal. This can include HNC projects, case studies or a review of a new/old technique.

CONCLUDING REMARKS

I would like to thank all members of the Executive and Education Committees. There have been a number of changes in the past year. Julie McWilliam has resigned due to an impending arrival in the New Year. Julie has worked for many years on behalf of the Association, being secretary, membership secretary and the person responsible for circulating the job vacancy

bulletin as well as many other jobs, which are too numerous to mention. I would like to thank Julie, on behalf of the ARTP for her dedication, hard work and invaluable contribution to our activities. This will be the first time that there will be no representative from the Derbyshire Royal Infirmary on the Executive Committee since the formation of the Association in the early seventies.

Joan Ashley from Bristol has also resigned as Meetings Officer and I would like to thank Joan for all the hard work that she put in to organising our meetings. I am pleased to say that the job of memberships secretary and the job vacancy bulletin has been kindly taken over by Steve Scholey (Pontefract), and the position of secretary by Roger Carter (Glasgow). As mentioned earlier Brendan Cooper has taken on the role of the manufactures liaison officer. I would like to thank Evelyn Smith for keeping us on the straight and narrow and for all the hard work she has put into the treasurers role over the years. This position was due for renewal and following a postal vote conducted after the AGM Julie Lloyd from Good Hope Hospital, Sutton Coldfield will become the new treasurer. I would like to thank Sue Revill for chairing the Education Committee and for production of Inspire, and Clare Thomas from Walsall Manor Hospital for taking on the position of Examinations Secretary and secretary of the Education Committee. I am pleased to report that we have 2 new faces on the Executive Committee, Jane Benson from Rotherham and Pat Mitchell from Fazackerley Hospital, Liverpool.

To conclude, these are changing times for everyone working in the health care arena. People's perception of the changes often is one of fear. For all of my working life in respiratory physiology I have had a laboratory in one room but recently after much business planning and negotiating I now have a suite of 12 rooms in a

new department at the Queen Elizabeth Hospital. So remember to keep fighting your corner, it sometimes can work out in your favour.

Finally I would like to leave you something to reflect upon. The name of the Association was created in the early 1970s and reflected the staff groups who performed lung function measurements. In the 1990s we sometimes find this can be counterproductive for our recognition and status. Perhaps it is time to change the name to reflect what is done and to ensure that everyone's contribution throughout the country is recognised. This could take the Association into the 21st century and give you all the true recognition that you deserve.

Education Report 1995

Miss Sue Revill, Dept of Respiratory Medicine, Glenfield Hospital, Leicester

Along with the ARTP/BTS National Assessment OTEC still remains the cornerstone of basic training. The BTEC Higher National Certificate course should be regarded as

Respiratory Specialist Option held at Coventry University. In addition the course aims to develop scientific communication skills - both written and verbal, greater powers of analysis and critical evaluation.

Following the Higher National Certificate the ARTP has offered a number of post basic courses during the past year. Some of these have been held in conjunction with the BTS, further cementing our links with this Society. The courses have been one or two days in length. This year the sleep and ventilation course was held at Stoke. This was a re-run of this very successful course, and is due to be held again in 1996, possibly late April, early May. Feedback, again was very encouraging, and this course is a must for anyone with an existing sleep service or due to develop one in the future. Our thanks go to Mrs Sue Bradbury and Dr Martin Allen for the excellent organisation of this course.

There was a one day course on the interpretation of lung function, held in Sutton

On to the ARTP/BTS National Assessment, again very successful this year with 9 candidates. A basic course was run at Bristol as an adjunct to the assessment, this also attracted a wide range of practitioners who found the course very useful, reflecting the high quality of the course. Our thanks go to Dr Adrian Kendrick who continues to organise this event. It is envisaged the course will run again in 1996. Applications for the National Assessment will be circulated to members early 1996.

This year the standard in the Assessment was particularly high, which is gratifying for the ARTP and especially the work-based supervisors of all the students who participated. I would like to thank all the clinical and medical assessors who travelled the length and breadth of the UK to assess students. And a special thanks to our new examination secretary Miss Claire Thomas from Walsall Manor Hospital, who took over the position at very short notice, and has done an excellent job.

I would like to pause now and present the certificates to the successful candidates who are with us this afternoon.

Thank You.

ABSTRACTS

Treadmill or cycle exercise to measure peak oxygen consumption in severe COPD
Speaker:

Sue Revill
Department of Respiratory Medicine, Glenfield Hospital, Leicester

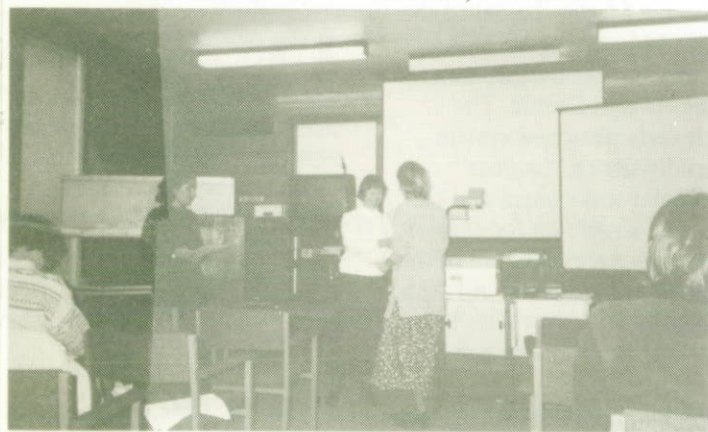
In normal subjects treadmill exercise usually produces the greatest maximum oxygen consumption (VO_2max). This may not be true in severe COPD where bicycle exercise, which offers support for the shoulder girdle, may produce a higher oxygen consumption than treadmill exercise. We have recently examined maximal exercise capacity on the bicycle

ergometer and the treadmill in 8 patients with COPD (FEV_1 3 SR below predicted) to determine which mode of exercise produced the greatest oxygen consumption.

Eight patients exercised to a symptom limited maximum on a bicycle and on a treadmill, on separate days. The workload on the bicycle was increased by 10 watts each minute, and the treadmill gradient was increased by 2.5% alternate minutes whilst the speed remained constant. Measurements of oxygen consumption (VO_2), ventilation (VE), heart rate and oxygen saturation were made, and capillary blood gases were measured before and immediately after exercise. Lactate concentration was measured before and 4 minutes post exercise.

There were no differences at peak exercise between the two forms of exercise for VO_2 (median 11.7 and 12.2 ml.min⁻¹kg for bicycle and treadmill respectively), for VE (median 26.6 and 25.0 l.min⁻¹ respectively), and for heart rate (median 119 and 115 beats.min⁻¹ respectively). The median post exercise lactate for bicycle exercise was higher than that for the treadmill (2.42 vs 0.94 mmol.l⁻¹).

Although individual variability was large, there was no clear difference between the two forms of exercise. Regular bicycle exercise was unfamiliar to this group of patients, and generated the greatest lactate response. The results do not support the hypothesis that bicycle exercise will produce a better performance in patients with severe COPD, but the two modes of exercise cannot be used interchangeably.



Presentation of Awards

essential, following on from the National Certificate. This course offers a more in-depth understanding of human physiology, pathology and respiratory measurements. The aim is to develop more advanced technical understanding of the basic measurements and introduce the more complex investigations e.g. exercise testing, sleep studies and respiratory muscle function studies are covered on the

Coldfield, and our thanks go to Dr Sue Hill and Dr Martin Miller for the organisation. Again this course was very successful, with more people applying than could be accommodated. For 1996, invasive and non-invasive blood gas measurement is planned, and possibly an advanced practical exercise testing course judging by the demand following the recently circulated ARTP questionnaire.

Continued on Page 18

ARTP BURSARIES

Application Form

Meeting/Course.....

Date..... ARTP membership No.....

Applicant Name..... Technical Grade.....

Departmental address.....

..... Post Code.....

Tel. No..... Ext.....

Reasons for attending meeting/course.....

Details of funding required.....

..... Total £.....

I the undersigned certify that a) I would like to attend the above meeting/course for the reasons given b) I am unable to receive the full funding from any other source c) understand that I am required to submit to INSPIRE a written report of the event/or detailed report of one session, before receipt of any reimbursement.

Signed..... Date.....

Head of Dept. signed..... Date.....

Please return form to:

Ms J Lloyd (Treasurer)
Respiratory Department
Goodhope Hospital
Rectory Road
Sutton Coldfield B75 7RR

This form may be photo-copied.



TECHNICAL UPDATE—LATEST EQUIPMENT

Open Circuit Lung Function and Single Breath Diffusion for Clinical Use

The measurement of diffusion studies has been widely documented in the literature. An editorial discussion entitled 'The single-breath carbon monoxide transfer test 25 years on: a re-appraisal' recently appeared in *Thorax* (*Thorax* 1993; 38: 1-9). In his concluding paragraph Dr Robert E Forster reported, "As I admitted earlier, I originally doubted whether the single-breath DLCO method would provide data of sufficient reproducibility to be of clinical value. I am therefore especially delighted that the technique is in such wide use today".

It is this same reproducibility that today makes the single-breath transfer factor the preferred diagnostic tool of the Respiratory Clinic. With this in mind P.K. Morgan Ltd developed a 'new' and stylish instrument to assist your studies, The Transflow™ uses 'Pneutek' technology to provide 'open circuit' studies of clinical lung function.

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Lung volume by open circuit techniques is achieved by the use of work taken from the pages of Cotes Lung Function, with new input based on the works of G.C. Cumming, Jr E.P. Radford and H.K. Chang. These authors offered ways of looking at lung mixing, the physical relationship of gases in contact with each other and gas distribution.

Cotes stated that alveolar volume can be calculated from information obtained during a single breath test, and derived the following equation

$$V_A = \frac{V_I, F_{A,N_2} - (V_E, N_2)(V_d/(V_E - V_d))}{F_{A,N_2} - (V_E, N_2)/(V_E - V_d)} \text{ (litres)}$$

F_{A,N_2} = fractional alveolar concentration before test

V_E, N_2 = volume of nitrogen expired

THIS WAS THE STARTING POINT, TO FIND OUT MORE CONTACT

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CHARITY NEWS

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The National Asthma Campaign
Providence House
Providence Place
London N1 ONT

The National Asthma Campaign has set up a Health

Professional Subscription Scheme to help keep health professionals up-to-date with developments in therapies and care provisions, leaflets, events organised locally and nationally. More information may be obtained by phoning the NAC on 071 226 2260 or Fax 071 704 0740, or writing to the address above.

Fund raising events for the British Lung Foundation:

The 10K Lung Run is scheduled for the end of April. The event will take place in Sutton Park, Sutton Coldfield. Substantial prizes for anyone who breaks the course record. More details from Dr Sue Hill, Queen Elizabeth Hospital, Birmingham. Tel: 0121 472 1311, ext 3233.

The official launch of 'Sow a Little Hope' was 18th October 1995. Embroidery from patients' wishes. This now forms a travelling exhibition. More information from the London HQ—Susan Kay on 0171 371 7704

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Environmental carbon monoxide pollution levels and the carbon monoxide single breath transfer factor test

A perspective

Louise Phillips, Lung Function Laboratory, Queens Medical Centre, Nottingham

Whenever I have performed a carbon monoxide (CO) single breath transfer factor test ($TLCO_{SB}$) the thought that we are using carbon monoxide has never really bothered me. I will often re-assure the patient with the comment "you inhale more carbon monoxide walking down a busy high street than performing this test". But is this fact or fiction?

Determined to find out I set off to the library in search of any studies that connect CO in the local high street and the amount of CO involved in the $TLCO_{SB}$. There were no papers from 1987 onwards with any relevant information. Even in some of the most recent environmental books there was very little up to date information. One of the most recent books was 1969, not really very relevant. The books covering respiratory function measurement did not relate inspired CO during testing to any environmental indices.

Where to next? Media reports concerning pollution and cars have been very topical, perhaps I needed to look in another direction. I telephoned the environmental health department at the local county council office and spoke to a very nice man about car exhausts. He seemed interested in my cause and promised help. True to his word, within a week he turned up at the lung function laboratory carrying a published report on the air pollution in the UK in 1993/4 (1). This report contained measurements on all pollutants from Nitrogen Oxide/Dioxide, Benzene, Butadiene and carbon monoxide. These figures were very interesting. The measurements were recorded from automatic air monitoring networks funded by the Department of the Environment (DoE) in 20 centres around the UK. These centres include:- Cromwell Street, London, Sheffield, Belfast, Leeds and Bristol.

Maximum hourly average, the highest results are:-

CROMWELL STREET LONDON	SHEFFIELD	BELFAST	LEEDS	BRISTOL
12.6 ppm	6.7 ppm	14.5 ppm	12.8 ppm	8.7 ppm

The exact locations for each site can be found at the end of this report.

The World Health Organisation (WHO) guidelines for safe limits are as follows:-

15 minute mean	87 ppm or 0.0087%
30 minute mean	50 ppm or .00050%
1 hour mean	5 ppm or 0.0025%
8 hour mean	10 ppm or 0.0010%

Most moderate to heavy smokers will be exceeding some of these 'safe' limits most of the time. Commercial drivers and other outdoor workers are exposed to the upper safe limits in our busy cities, although environmental pollution fluctuates dramatically during the day, and is seasonal. Additionally what are the consequences of environmental or cigarette CO on the foetus?

In the lung function laboratory patients are exposed to very high levels in a transient manner. Performing two $TLCO_{SB}$ 5 minutes apart the patient is exposed to a total of 5600 ppm of CO (assuming the CO inspire is 0.28%). However the average exposure over 30 minutes in the laboratory will be very different, as patients only inhale the test gas twice during this period. As stated previously, patients are exposed to high levels in a transient fashion, the majority of patients do not have full pulmonary function tests on a daily basis! As for the physiological consequences of the $TLCO_{SB}$ this has been studied in more detail, where the background CO level has been measured after performing a single test, then two tests etc up to a total of 10 (verbal communication with Dr A. Kendrick).

Additionally there are correction factors for the $TLCO_{SB}$ measurement for smokers who have a high back tension of CO which acts to lower the transfer factor.

So my quest was complete, with persistence I finally managed to establish the levels of CO in our city streets. I am not sure whether I will use the analogue again, as our chronic exposure to the pollutant is very different from a very brief transient exposure in the laboratory.

Continued on Back Page

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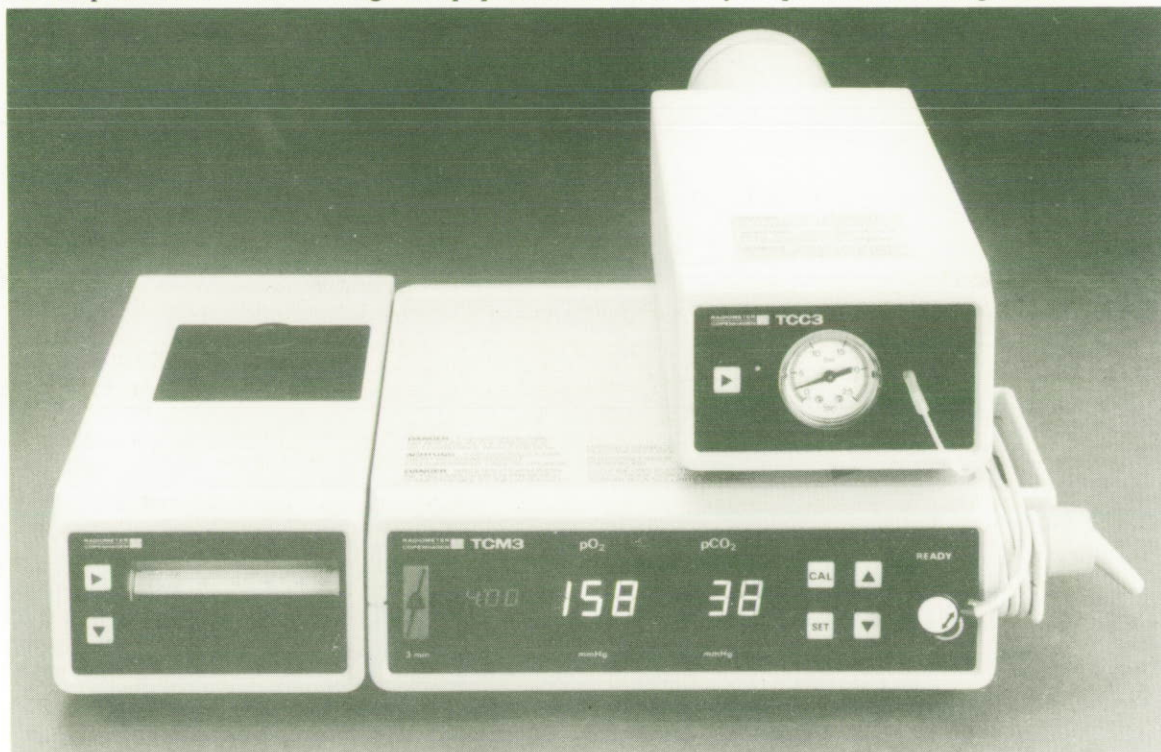
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(Reference: Comparison of peak oxygen consumption during cycle and treadmill exercise in severe chronic obstructive pulmonary disease.

RS Mathur, SM Revill, DD Vara, R Walton and MDL Morgan. *Thorax* 1995; 50; 829-833)

PRE-FLIGHT ASSESSMENT AND COAD

Speaker:

DR Owen Johnson
CONSULTANT
PHYSICIAN, PONTEFRAC
GENERAL INFIRMARY

Commercial aircraft's are pressurised but it is important to realise that they are not pressurised to sea level pressures and passengers will be exposed to the equivalent pressure that would be found at an altitude of 2450 metres. At this altitude barometric pressure is around 72 kilopascals and inspired oxygen pressure about 15 kilopascals. Passengers with impaired gas exchange may become profoundly hypoxaemic.

As well as hypoxaemia induced by altitude other perils of flying include gas expansion and the enclosed cabin environment. Contra-indications to flying include those related to pressure including pneumothorax, pneumomediastinum and scuba diving as well as the relative contraindications of recent surgery and upper respiratory tract infections. Contraindications related to hypoxaemia include severe respiratory failure or any condition which will be aggravated by hypoxaemia such as unstable ischaemic heart disease or sickle-cell anaemia.

Pre-flight assessment is based on three methods. The traditional method is by asking the question whether the passenger can walk 50 metres on the flat without breathlessness. Others advise

using prediction formulae which are based on ground level pO₂ with or without FEV₁. The more physiological assessment is by hypoxic challenge test. The hypoxic challenge test uses an inspired pO₂ of 15 kilopascals stimulated at ground level by using 15% oxygen in nitrogen. The patient is given this gas mixture to breath for 30 minutes and oxygenation assessed. These 3 methods have been compared in cryptogenic fibrosing alveolitis and only the hypoxic challenge test was found to be a useful predictor of hypoxaemia as well as symptoms of hypoxaemia. Pre-flight assessment in COAD is much more difficult as changes in oxygenation due to altitude are much more unpredictable in patients with this condition.

The conclusions are that if a patient undergoes pre-flight assessment it should be by hypoxic challenge test. It is probably safest to test all respiratory patients who are hypoxaemic, those who have a very low FEV₁, those that are concerned about flying and those who have co-existent angina. However, the practical limitations are obvious.

TUBERCULOSIS - AN INCREASING PROBLEM

Speaker:

Dr. Martin Allen
Dr Suleman Moreea
Chest Clinic
St Lukes Hospital
Bradford. BD5 0NA

Tuberculosis remains a global health problem with estimates from the World Health Organisation of 1.7 billion (1/3 of the world's population) having been infected with TB, 8 million per year have active TB with 3 million dying annually.

Apart from small increases during the world wars tuberculosis has declined steadily in the western world since 1860. However, over recent years there has been a world-wide upsurge in TB.

The reasons for this vary greatly, depending upon country and population. In the UK poverty and deprivation appears to be the main reason; 29% of the TB occurring in the poorest 10% of the population. Significant associations with other indicators of poverty, including the Jarman index, Townsend index and free school meals have recently been confirmed. An additional factor in some areas is immigration, individuals from areas of high incidence bringing the disease with them. Although of less importance in the UK, infection with the human immunodeficiency virus (HIV) accounts for many cases of TB in the developing world. Of the 8 million individuals with TB in 1990 about 4.2% are related to HIV, in the year 2000 13.8% of the estimated 10.2 million individuals with TB will have both infections. The mechanism for the association being the reduced immunity allowing reactivation of latent TB and easier acquisition of new infection.

Tuberculosis is spread by the airborne droplet route so examination of sputum gives a very good indication of how infectious an individual with TB may be. If smear positive (acid alcohol fast bacilli, AAFB, identified on a direct examination of sputum) the risk of passing infection on is very high, especially to close contacts. Whether an individual exposed to an aerosol containing AAFB develops TB depends on several factors especially the hosts immunity, e.g. have they prior sensitisation to AAFB (previous infection, exposure to environmental Mycobacteria or BCG vaccination) and are they immunocompetent (effected by HIV, immunosuppressive therapy or chronic illness). The reduction in cellular immunity with age explains the increased incidence in the elderly with reactivation of previously acquired infection.

The management of TB relies on prompt identification,

notification and treatment of the index case to prevent further spread. Notification, a legal requirement, allows limited central information to be collected and initiates the contact procedure where contacts of the index case are identified and screened by chest x-ray and Heaf test to ensure they do not have active disease.

Treatment of active TB is with three drugs (or four if resistant Mycobacteria are suspected) for two months, changing to two drugs to which the AAFB are sensitive for a further 4 months. With good compliance and regimens using Rifampicin and Isoniazid a 100% cure can be expected. In patients with multi-drug resistant TB (resistance to Rifampicin and Isoniazid) treatment is with less active second line drugs for a longer period; alternative therapies using surgical excision or immunotherapy with *M vaccae* may be needed. Treatment of environmental or atypical Mycobacteria often needs a longer course of treatment and relapse in fully compliant patients may occur.

By remaining vigilant and suspecting TB in patients with pulmonary problems smear positive (infectious) cases will be identified. Prompt and appropriate treatment will render such individuals non-infectious within a few weeks and cure the infection with a low relapse rate. As *Mycobacterium tuberculosis* has no host other than man reduction in the number of new cases should be possible in the developed world by following the established treatment and screening guidelines and attention to public health issues.



RECENT ARTICLES

The following summarise recently published papers/articles appearing in medical journals which may be of interest to ARTP members

INVESTIGATION OF SLEEP APNOEA AND TREATMENT

Efficacy of non-invasive CPAP in COPD with acute respiratory failure

P Goldberg, H Reissmann, F Maltais, M Ranieri, SB Gottfried. *Eur Respir J* 1995, 8, 1894-1900.

The purpose of this study was to determine the effects of graded amounts of continuous positive airway pressure (CPAP) on the degree of inspiratory effort, pattern of breathing, gas exchange, and level of dyspnoea in a group of spontaneously breathing, nonintubated COPD patients in acute hypercapnic respiratory failure. The effect of CPAP application on the 10 patients studied was to decrease inspiratory effort and dyspnoea whilst improving breathing pattern. The authors suggest the early institution of CPAP may conceivably obviate the need for intubation and conventional mechanical ventilation.

A provocative thought! This certainly requires further investigation. Anyone out there to take up the challenge?

Use of pulse transit time as a measure of inspiratory effort in patients with obstructive sleep apnoea.

DJ Pitson, A Sandell, R van den Hout, JR Stradling. *Eur Respir J* 1995; 8: 1669-1674

The aim of this study was to determine if the inspiratory rises in pulse transit time could provide a quantitative measure of inspiratory effort in patients with obstructive sleep apnoea. Pulse transit time is the time taken for the arterial pulse pressure wave to travel from the aortic valve to a peripheral site, and is conventionally measured from the ECG R wave to pulse wave arrival in a finger. In the

normal situation the transit time increases during pulsus paradoxus (transient blood pressure fall during inspiration).

Eight patients with OSA were studied. Recordings of oesophageal pressure and pulse transit time were recorded throughout sleep whilst airway pressure was varied to produce a range of inspiratory efforts. The results yielded a very good correlation between pulse transit time and oesophageal pressure ($r=0.94$), leading the authors to suggest that pulse transit time may be a clinically useful, non-invasive and quantitative measure of inspiratory effort.

Choosing a negative pressure ventilation pump: are there any important differences.

IE Smith, MA King, JM Shneerson. *Eur Respir J* 1995; 8: 1792-1795

These workers assessed five negative pressure ventilators to assess any differences in performance. Characteristics assessed were pressure waveform, tidal volume generated, stability of performance over 8 hour period and the response to a change in leak.

PHYSIOLOGICAL MEASUREMENT TECHNIQUES

Detection of flow limitation during tidal breathing by the interrupter technique

R Hage, JGJV Aerts, AFM Verbraak, B van den Berg, JM Bogaard. *Eur Respir J* 1995, 8, 1910-1914

In this study the flow interrupter technique was applied to detect flow limitation during tidal breathing in a group of patients with airflow obstruction, with and without airflow limitation. The results are compared to those obtained with the body plethysmograph.

The interrupter technique assumes the mouth pressure measured at the end of a short interruption of expiratory flow to be equal to alveolar pressure, and the ratio between this mouth pressure and flow before the interruption is used to estimate airways resistance. The collapsibility of the airways can also be established from the interrupter technique.

There was a highly significant difference between the mean spike area for patients with airflow limitation and the control group and the patients without airflow limitation, leading the authors to conclude that this is a useful technique for the assessment of airflow limitation during quiet breathing.

In vivo assessment of diaphragm contraction by ultrasound in normal subjects.

Ueki J, De Bruin PF, Pride NB. *Thorax* 1995; 50:1157-1161.

Accurate quantification of respiratory muscle strength is important in assessing patients with respiratory muscle dysfunction. The simplest method of assessing respiratory muscle strength is by measuring mouth pressure during maximum inspiratory effort against a closed airway (P_{Imax}) but a wide range of normal values has been reported. Ultrasound allows observation of the thickness of the diaphragm in the zone of apposition in vivo during relaxation and maximum respiratory efforts. The technique is non-invasive and was found to be repeatable in the same subject. The thickening ratio (TR) was measured.

TR = Thickness during P_{Imax} manoeuvre at FRC mean thickness while relaxing at FRC

The thickness ratio at FRC may be a good indicator of diaphragm strength. The investigation takes about 15

minutes and can be done at the bedside and may prove useful in the intensive care unit.

ASTHMA AND INHALERS

Metered-dose Inhalers and CFC's : What respiratory physicians need to know.

Partridge MR.

Respiratory Medicine 1995; 88:645-647.

In this editorial the effect of CFC's on the environment, the use of alternative inhaler devices, alternative propellants in MDI's and ultimately the effect of any change in medication or device on the patient are discussed.

Evaluation of the effectiveness of four different inhalers in patients with chronic obstructive pulmonary disease.

Van der Palen J, Klein JJ, Kemchhoff AHM, Van Herwaarden CLA.

Thorax 1995; 50:1183-1187.

The inhalers investigated were MDI's and three dry powder devices, Turbohaler, Diskhaler and Rotahaler. 152 patients with COPD aged between 18 and 65 years took part in the study. Disease and inhaler variables were assessed by means of a check list. Many patients were found to use their inhaler ineffectively. Fewest errors were found with the diskhaler, while most patients using MDI's made crucial mistakes.

Bioequivalence of inhaled drugs

E Derom, R Pauwels (Editorial) *Eur Respir J* 1995; 8: 1634-1636

This useful editorial briefly reviews the past research and the current situation for the need for standardisation criteria to determine the exact therapeutic equivalence of drugs generated by the newly developed inhalers.

Continued Overpage

It also reviews a paper addressing bioequivalence of steroid inhalers in the same journal.

Differences in output from corticosteroid inhalers used with a volumatic spacer

MR Miller, P Bright Eur Respir J 1995; 8: 1637-1638

Beclomethasone dipropionate inhalers manufactured by three different companies were investigated. A sample of 20 canisters from each manufacturer was investigated by firing into a volumatic spacer in random and mixed order. The amount of drug deposited onto a filter was measured using high performance liquid chromatography.

There were significant differences between the output from each of the different types of inhaler, with a 36% difference between the highest and the lowest outputs. The authors conclude substitution of one device for another will not necessarily give equivalent doses

Comparing methods for assessing bronchial responsiveness in children: single step cold air challenge, multiple step cold air challenge, and histamine provocation.

M Modl, E Eber, B Steinbrugger, E Weinhandl, MS Zach. Eur Respir J 1995; 8: 1742-1747.

Three types of bronchial challenge were performed over

separate days in a group of children and adolescents with asthma. The challenges were: single step cold air challenge—hyperventilation at 75% of maximal voluntary ventilation (MVV) for 4 minutes with dry air at -10 deg C. CO₂ was added to inspire to maintain eucapnia.

Multiple step cold air challenge—hyperventilation for 3 minutes using same cold dry air, but the level of hyperventilation increasing in step wise progression from 10% of MVV to 80% MVV. FEV₁ measured at 30, 90 and 180 seconds after each step, and a dose response curve plotted. Histamine challenge—as the European standardisation, multiple step protocol consisting

of 2 minute inhalation by quiet tidal breathing. The first aerosol was the diluent, followed at 5 minute intervals by doubling concentrations of histamine from 0.03 to 8.0 mg/ml.

The results of the study showed there was no difference in the response activated by the single or the multi step cold air challenges, therefore allowing the less time consuming single step challenge to be substituted for the longer multi step challenge. However the cold air challenge appeared to provoke a different type of bronchial responsiveness to the histamine challenge, and were not therefore interchangeable.

REFERENCES

Air Pollution in the UK: 1993/4 Prepared by the National Environmental Technology Centre at the request of the Department of the Environment.

SITE LOCATIONS AND INFORMATION:-

Cromwell Road, London

Site Type

Description

Month highest result recorded

Curbside

Located at the curbside of a busy arterial road in central London. Traffic density approximately 60,000 vehicles per day.

November 1993

Sheffield

Site Type

Description

Month highest result recorded

Urban

Near community centre in a mixed residential/industrial area 200 m from the M1

October 1993

Belfast

Site Type

Description

Month highest result recorded

Urban

Pedestrianised street (Lombard Street) 25 m from major road

November 1993

Leeds

Site Type

Description

Month highest result recorded

Urban

Open area (Queens Court) 30 m from major road

January 1994

Bristol

Site Type

Description

Month highest results recorded

Urban

Pedestrianised walkway (Lower Castle Street to Bond Street) 43 m from major road

January 1994



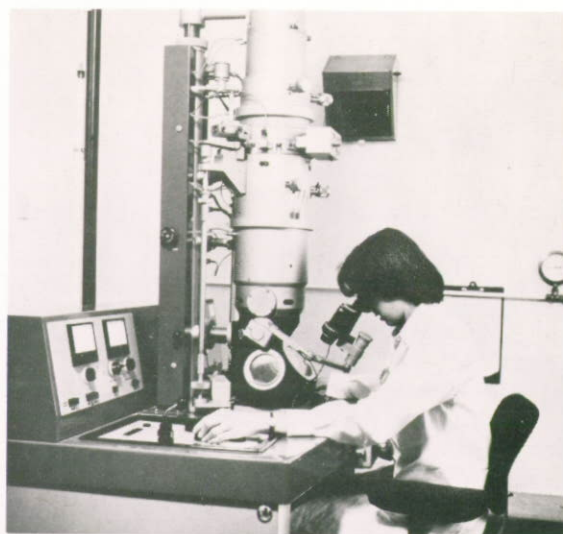
BREATH

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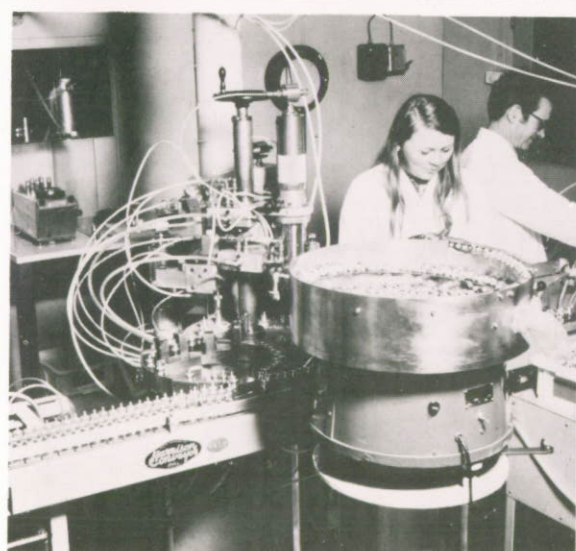
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a bronchodilator.

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Ventolin Inhaler (salbutamol BP)

Uses. Routine control of bronchospasm in bronchial asthma, bronchitis and emphysema, or as required to relieve attacks of acute bronchospasm. Doses may also be taken before exertion to prevent exercise-induced asthma or before exposure to a known unavoidable challenge.

Dosage and administration. As single doses for the relief of acute bronchospasm, for managing intermittent episodes of asthma and to prevent exercise-induced bronchospasm. **Using Ventolin Inhaler** - Adults: one or two inhalations. **Children:** one inhalation increasing to two if necessary. **Using Ventolin Rotahaler** - Adults: one Ventolin Rotacap 200mcg or 400mcg. **Children:** one Ventolin Rotacap 200mcg. For chronic maintenance or prophylactic therapy. **Using Ventolin Inhaler** - Adults: two inhalations three or four times a day. **Children:** one inhalation three or four times a day increasing to two inhalations if necessary. **Using Ventolin Rotahaler** - Adults: one Ventolin Rotacap 400mcg three or four times a day. **Children:** one Ventolin Rotacap 200mcg three or four times a day. For optimum results in most patients inhaled Ventolin should be administered regularly.

Contra-indications. Ventolin preparations should not be used for the prevention of threatened abortion.

Precautions. If a previously effective dose of inhaled Ventolin fails to give relief lasting at least three hours, the patient should be advised to seek medical advice. Ventolin should be administered cautiously to patients suffering from thyrotoxicosis. Unnecessary administration of drugs during the first trimester of pregnancy is undesirable.

Side effects. No important side effects have been reported following treatment with inhaled Ventolin.

Presentation and Basic NHS cost (exclusive of VAT). Ventolin Inhaler is a metered-dose aerosol delivering 100mcg salbutamol BP per actuation. Each canister contains 200 inhalations. Basic NHS cost £3.00. Ventolin Rotacaps 200mcg and 400mcg, each contain a mixture of the stated amount of microfine salbutamol BP (as sulphate), and larger particle lactose in light blue/colourless or dark blue/colourless hard gelatine cartridges, respectively. Containers of 100. Basic NHS cost £5.29 and £7.15, respectively. Ventolin Rotahaler for use in conjunction with Ventolin Rotacaps. Basic NHS cost 78p.

Product Licence numbers. Ventolin Inhaler 0045/5022. Ventolin Rotacaps 200mcg 0045/0116. Ventolin Rotacaps 400mcg 0045/0117.

Becotide Inhaler (beclomethasone dipropionate BP)

Uses. Bronchial asthma especially in patients whose asthma is not adequately controlled by bronchodilators and patients with severe asthma who would otherwise be dependent on systemic corticosteroids or adrenocorticotrophic hormone (ACTH) or its synthetic equivalent.

Dosage and administration. Using Becotide Inhaler - Adults: two inhalations three or four times a day is the usual maintenance dose. In severe cases dosage may be started at twelve to sixteen inhalations per day and subsequently reduced when the patient begins to respond. **Children:** one or two inhalations, two, three or four times a day according to the response. **Using Becotide Rotahaler** - Adults: one 200mcg Becotide Rotacap three or four times a day is the usual maintenance dose. **Children:** one 100mcg Becotide Rotacap two, three or four times a day according to the response. For optimum results inhaled Becotide should be administered regularly.

Contra-indications. No specific contra-indications to inhaled Becotide are known but special care is necessary in patients with active or quiescent pulmonary tuberculosis.

Precautions. The maximum daily intake of beclomethasone dipropionate BP should not exceed 1mg. Inadequate response after the first week of inhaled Becotide therapy suggests that excessive mucus is preventing penetration of inhaled drug to the target area. A short course of systemic steroid in relatively high dosage should be given and therapy with inhaled Becotide continued. Unnecessary administration of drugs during the first trimester of pregnancy is undesirable. When transferring patients to Becotide from systemic steroid therapy the possibility of adrenocortical suppression should be considered and patients given a supply of oral steroids for use during periods of stress. Please refer to the detailed procedure described in the data sheets for Becotide Inhaler and Becotide Rotacaps.

Side effects. Occasional candidiasis of the mouth and throat (thrush) occurs in some patients, particularly those with high blood levels of Candida precipitins. Topical therapy with antifungal agents usually clears the condition without withdrawal of Becotide.

Presentation and Basic NHS cost (exclusive of VAT). Becotide Inhaler is a metered-dose aerosol delivering 50mcg beclomethasone dipropionate BP per actuation. Each canister contains 200 inhalations. Basic NHS cost £4.77. Becotide Rotacaps 100mcg and 200mcg, each contain a mixture of the stated amount of microfine beclomethasone dipropionate BP and larger particle lactose in buff or chocolate-brown/colourless hard gelatine cartridges, respectively. Containers of 100. Basic NHS cost £7.26 and £9.67 respectively. Becotide Rotahaler, for use in conjunction with Becotide Rotacaps. Basic NHS cost 78p.

Product Licence numbers. Becotide Inhaler 0045/0089. Becotide Rotacaps 100mcg 0045/0119. Becotide Rotacaps 200mcg 0045/0120.

Beconase Nasal Spray (beclomethasone dipropionate BP)

Uses. The prophylaxis and treatment of perennial and seasonal allergic rhinitis, including hay fever and vasomotor rhinitis.

Dosage and administration. The recommended maximum dosage is one application into each nostril, four times daily. Not suitable for children under six years of age. Full therapeutic benefit requires regular usage and the absence of any immediate effect should be explained to the patient to facilitate compliance with the regular dosage schedule.

Contra-indications, warnings, etc. There are no specific contra-indications but any infections of the nasal passages and paranasal sinuses should receive the appropriate treatment. Care must be taken while transferring patients from systemic steroid treatment to Beconase if there is any reason to suppose that adrenal function is impaired. Eye symptoms may necessitate additional topical therapy. Unnecessary administration of drugs during the first trimester of pregnancy is undesirable. No major side effects attributable to Beconase have been reported, but occasionally sneezing attacks have followed immediately after use of the aerosol.

Presentation and Basic NHS cost (exclusive of VAT). Beconase Nasal Spray is a metered-dose aerosol delivering 50mcg beclomethasone dipropionate BP per actuation into a special nasal applicator. Each canister provides 200 applications. Basic NHS cost £4.77.

Product Licence number. 0045/0093.



Further information on Beconase, Becotide, Rotacap, Rotahaler and Ventolin (trade marks) is available from:
ALLEN & HANBURY LIMITED, LONDON E2 6LA.

EDITORIAL

The Harrogate File

The education of Physiological Measurement Technicians (the PMTs of official jargon) has been reviewed in this journal from time to time, some may think quite adequately enough. Further discussion of this topic (for which we make no apology) is now occasioned by an interesting conference which was held in Harrogate during August at the 'NHS Training and Studies Centre'. In this comfortable establishment overlooking a pleasant park we heard the very latest on 'Assessment of the Practical Ability of Student PMTs'.

A variety of experts and others had been invited; there were DHSS representatives, Regional Scientific and Training Officers, college teachers and members of the FAMT and of its constituent associations including our very own ARTP. This provided an opportunity to meet people in different spheres (a process assisted by the well-stocked bar) and to hear other views without being obliged to change one's own. Some participants may have been disappointed that no hard and fast decisions were made but the conference was not designed with this in mind.

As background reading, we have already seen Jim Reed's account of the position (*Breath*, July 1978) and some further candid thoughts from Sally Gough in the same journal

(February 1980). The whole subject is treated at length in the so-called 'Orange Report' (because it's got an orange cover), which presents the findings of a working party set up to monitor the college-based TEC teaching programmes and to develop 'in-service' training schemes in collaboration with the technician associations; it is on this last issue that we have heard great argument. The necessity for teaching in lecture theatre and practical classroom would appear to be generally accepted but when we turn to the 'in-service' question, the principle itself creates substantial divisions. We are told for instance, that the TEC is not prepared to accept the validation of the in-service training courses, on the grounds that they are not 'educational' in the strict sense; this seems a curious philosophy in the world of today.

Full education in any field must surely mean the integration of theoretical 'text-book' knowledge with practical experience. One can think of no professional body within the health industry which can let its members loose on the public without such an educational programme and so far as we, the ARTP, are concerned this practical knowledge will have to be gained directly in the departments of respiratory medicine in our main teaching centres and nowhere else. It is proposed that PMTs should be

trained as 'supernumerary' students who, for the first 15 months of a two-year course, would rotate through the various specialist departments. The advantage of such a scheme is perhaps self-evident; the students would gain experience on a much broader base than could be achieved by spending their time in a single department. If this is to take place, there will have to be, in each region, at least one centre willing and able to accept the responsibility; the teaching load would fall fairly and squarely on the senior staff who have been known to wonder what would be gained by spending their precious time teaching successive groups of students who, for the most part, would afterwards simply disappear into limbo. The good teacher would nevertheless attract students into the speciality. It might help if the term 'supernumerary' could itself be dropped from our vocabulary — it always seems to have a ring of someone unwanted, an outsider, as well as being virtually unpronounceable. Let's just call them 'students'!

If this system seems unsatisfactory it is worth glancing at the situation which exists in other Health Service disciplines. In our highly revered medical profession for example, the students are intended to receive and in some cases do receive, a balanced programme of theoretical knowledge with practical knowledge acquired by rotation through the specialised departments, learning their trade at first hand in wards, operating theatres and laboratories. There is no question of a student being 'apprenticed' so to speak, to a cardiologist or a chest physician, but during their brief stay in these departments they may get an inkling of their eventual goal. Although the bulk of medical teaching (for better or worse) takes place in the hospital setting the majority of students are eventually employed outside the

hospital. Similarly, for student PMTs, there could be openings in industrial medicine, health education, the pharmaceutical industry and with equipment manufacturers. Indeed, the Orange Report Working Party, considered that PMTs should have qualifications suitable for use *outside* the NHS.

All the specialist groups of the FAMT have now submitted in-service training schemes, which appear in a large Appendix to the Orange Report; it must be said, however, that the amount of material which some students would be expected to learn in the time available would give headaches to a Mastermind finalist! And yet more headaches will arise from the problems of putting the plans into practice. A supernumerary scheme has in fact been operating in the West Midlands for some time and apparently works well; no doubt a good deal of energy and persistence has been required to make it a success.

The FAMT is one body to which we should be able to turn in order to have our Association training programme put into effect, for it alone has the fighting weight we need. The individual associations can, in this respect, achieve relatively little in isolation, though they can and do provide useful pressure groups within the larger organisation. Some associations, moreover have been established for many years and already have their own training programmes and examinations; their members understandably may see little need for change but nevertheless might benefit from it. The ARTP is a newcomer on the scene and its members, particularly those in the more junior grades have every reason to welcome the principles which are embodied in the new training programmes however much we may disagree about the details.

LUNG SOUNDS

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INTRODUCTION

The modern methods of recording and analysis of lung sounds, coupled with the advances in respiratory physiology have revived interest in auscultation of the lung. Systematic auscultation of the chest began with the invention of the stethoscope by Laennec in 1816. He described and classified lung sounds and attempted to correlate what he heard in life with the pathological findings in the lung. In this way certain lung sounds came to be associated with well understood pathological conditions; the presence of 'bronchial breathing' in pneumonia is one example. Although the mechanism of sound production was poorly understood, it did not preclude the clinicians of the 19th century from exploiting auscultation as an important means of diagnosing respiratory disorders. With the introduction of the chest radiograph in the early 20th century, interest in lung auscultation declined because pathological entities like fibrosis, collapse and consolidation could readily be visualised.

By contrast auscultation of the heart progressed to a more precise science. The introduction of electrocardiography, phonocardiography and cardiac catheterisation allowed precise timing of the heart sounds and murmurs in relation to the pressure and flow changes in the heart. A similar functional approach to lung sounds was not

attempted until Paul Forgacs showed the advantages of linking the lung sounds with the physiological events of respiration. The result was a better understanding of lung sounds and a wealth of new information of clinical and research interest.

RECORDING OF LUNG SOUNDS

A microphone, an amplifier incorporating a variable high pass filter, a tape recorder and a recording system are the basic requirements for recording lung sounds. Physiological parameters such as the flow rate, lung volume and transpulmonary pressure can all be measured simultaneously with the lung sounds. Depending upon one's particular interest, further sophistication can be added to the basic recording system and the facilities offered by a computer have made it possible to undertake a wide range of analytical work on lung sounds.

CLASSIFICATION

The human lung is capable of generating only a limited repertoire of sounds. These sounds may be classified as normal and abnormal breath sounds, wheezes and crackles.

BREATH SOUNDS (Fig. 1)

Breath sounds are a form of noise which contain oscillations of random amplitude and frequency predominantly in a range of 200 to 2000 Hz (white noise).

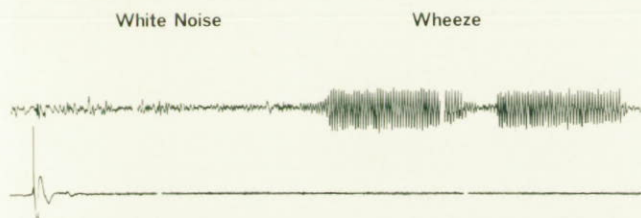


Fig 1. Tracing of breath sounds and expiratory wheeze. Random variations in amplitude of the breath sounds (white noise) contrast with the regular wave form of the wheeze.

The site and the generation of breath sounds is closely related to the nature of gas flow in the human airways. In the upper airways, the trachea and the main bronchi, gas flow remains turbulent at physiological rates of breathing. As the bronchi divide and sub-divide, the rate of gas flow progressively diminishes with the increase in total cross-sectional area of the airways. The turbulent airflow in the main bronchi, however, is not replaced by laminar flow until the most peripheral airways are reached. The gas flow in the 'intermediate' airways (up to the 15th generation of bronchi) remains non-laminar from disturbance to gas flow at the branching site with the formation of turbulent eddies. Thus the gas flow in the airways may be considered in three phases; 1. *turbulent airflow* in the upper airways, trachea and the main bronchi; 2. *non-laminar disturbed flow* in the intermediate airways and 3. *laminar flow* in the terminal airways and alveoli.

Breath sounds are generated only within those airways where the flow conditions favour sudden oscillations of gas particles thus converting kinetic energy of gas flow into heat and sound. The predominant source of breath sounds is in the turbulent zone of the airways with a further contribution from airways in the non-laminar zone where turbulent eddies are produced at the branching sites. In the terminal airways and alveoli the sluggish rate of gas flow is incompatible with sound production in a gas phase.

TRANSMISSION OF BREATH SOUNDS

Sound waves travel well in wide tubes containing air medium and so the breath sounds transmitted from the proximal airways to the mouth are accompanied by little loss of frequency or of sound energy. Breath sounds heard at the chest wall are transmitted through the lobar and segmental bronchi and across the lung and the chest wall. During their transmission they are attenuated as the higher frequencies are selectively filtered out. The spectrum of breath sounds at the mouth contains a range of frequencies from about 200 to 2000 Hz; breath sounds recorded over the upper chest contain frequencies from 200 to 800 Hz while over the lower chest there is a narrower band, ranging from 200 to 400 Hz. Thus the lung and the chest wall act as a low pass filter with the greatest attenuation of frequencies at the lung bases.

CLINICAL APPLICATION

Breath sounds at the mouth

In stenosis of the major airways at the level of the larynx, trachea or the main bronchus, the formation of turbulent eddies at the site of stenosis is intensified and the breath sounds at the mouth become disproportionately loud. Similarly breath sounds in diffuse airway obstruction due to asthma and chronic bronchitis are frequently louder than normal. In these patients loud breathing arises from turbulence, which is intensified in the narrowed proximal bronchi by a high velocity of gas flow. The more severe the airways obstruction, the louder the breath sounds at the mouth; bronchodilator drugs can make the breath sounds quieter. By contrast, inspiratory breath sounds at the mouth in primary emphysema are normal although the peak expiratory flow rate is very low. In this disorder the inspiratory calibre of the larger airways is normal while the low expiratory flow rate results from the loss of elastic recoil and premature dynamic compression of the bronchi.

Breath sounds transmitted through the chest wall

Breath sounds are attenuated during their transmission through the lung and chest wall. When fluid or air intervenes between the lung and the chest wall the breath sounds are absent. In diffuse airways obstruction, the loudness of the breath sounds varies with the regional variations of air-flow and the thickness of the chest wall. Air distended lung, as in emphysema, is a poor transmitter of sound and so the breath sounds become quieter. By contrast, a consolidated lung, as in pneumonia, improves transmission of the higher frequencies and the breath sounds resemble sounds generated over the main bronchi; this is known as 'bronchial breathing'.

WHEEZES (Fig. 2)

Wheezes are continuous musical sounds with a definite pitch. Their musical quality has naturally been compared with the sounds of orchestral instruments and in particular, the wind instruments. The choice of the right model, however, is important to the understanding of wheeze production.

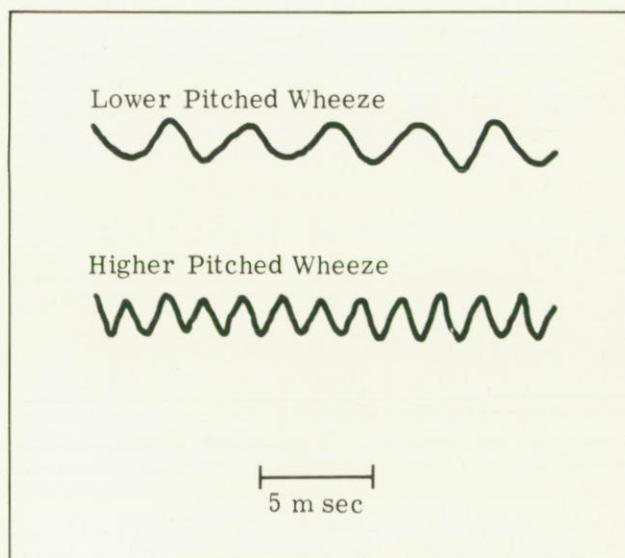


Fig 2. Wheeze from an asthmatic patient at two inspiratory flow rates. The pitch of the wheeze rises as the flow rate is increased.

Wind instruments may be considered in three groups; the first group is represented by the recorder and the flue pipes of the organ where a hiss is generated by the impact of air on a sharp edge which is changed to a musical note in the body of the instrument. The second group is represented by the clarinet and the oboe which have in common a single or double reed. The reed opens and closes when air is blown, generating a musical note which is set in resonant oscillation by the gas column in the body of the instrument. As with the organ pipe, the pitch of the note depends upon the length of the pipe and the density of the gas. The pitch of the note can be raised by blowing with helium, a low density gas in which the sound waves travel faster. By contrast the pitch of a wheeze is unchanged when a subject breathes a low density gas. The pitch of a wheeze however, can be varied by the velocity of air flow so that an airway of given calibre can generate a high or a low pitched wheeze. In this respect the mechanism of wheeze production resembles closely a simple *toy trumpet* rather than the orchestral wind instruments. A toy trumpet generates a musical note when air flow sets the reed in oscillation. The decorative paper attachment to the toy trumpet is entirely ornamental and serves no acoustic function. As with the wheeze, the pitch of the note of a toy trumpet can be raised or lowered by varying the velocity of airflow and is independent of the density of the blowing gas.

Production of the wheeze

A wheeze is produced when the airway is narrowed to the point of closure so that the opposing walls, lightly touching, function as a reed. When the velocity of airflow reaches a critical value, the reed-like mechanism is set in oscillation generating the musical sound of a wheeze. The pitch of the wheeze depends upon the mechanical properties of the reed and the velocity of airflow and is independent of the gas density. In an excised bronchus a wheeze can be generated only when the airway is narrowed to the point of closure.

Even in the most severe airways obstruction only a few wheezes are generated, indicating that despite widespread narrowing, only a few airways at a given time are on the point of closure. When a bronchus is narrowed by a tumour mass, breathing is usually noisy. It can be brought to the point of closure by rapid inspiration when the 'Venturi' effect comes into play setting the mass of tumour in slow oscillation and generating a low pitched wheeze. In severe asthma wheezes may be absent even in the presence of severe airflow obstruction; in these exhausted patients, the gas velocity may be too low to set up oscillations in a bronchus on the point of closure.

CRACKLES

Crackles are explosive non-musical sounds, generated by the lung or the pleura. Major groups of lung crackles can be identified by their timing in inspiration and by their related features.

Late Inspiratory Crackles (Fig. 3)

Late inspiratory crackles are so named because the crackles continue to the end of inspiration though they may begin in any part of the inspiratory cycle. The crackles are typically basal in distribution but may extend to the mid or upper zones in more severe cases; they are not modified by cough or transmitted to the mouth. They are a feature of a wide range of lung disorders characterised by lung deflation due to pulmonary oedema, infiltration or fibrosis. They may also be heard in healthy subjects during the first few inspirations after prolonged shallow

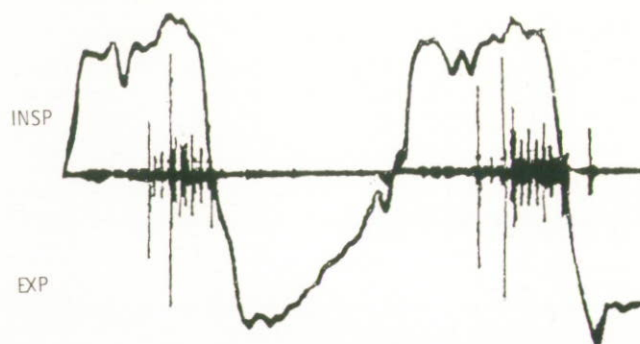


Fig 3. *Fibrosing alveolitis: Inspiratory crackles recorded simultaneously with the flow rate. The crackles are seen in the mid and the late phase of inspiration.*

breathing or when the movements of the diaphragm are restricted. The crackles are characteristically *posture dependent*; when the patient bends forwards, the basal crackles are reduced or completely silenced, returning as the subject regains the upright position. (Fig. 4)

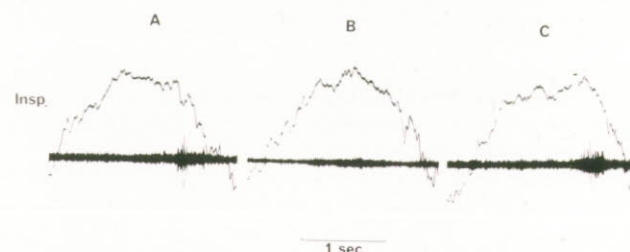


Fig 4. *Late inspiratory crackles recorded at the lung base from a patient with fibrosing alveolitis in three body positions. (a) The patient is standing, (b) bending forwards and (c) upright again.*

In widely differing pathological conditions like fibrosing alveolitis, asbestosis and pulmonary oedema, the lungs share a common functional abnormality; they become smaller and stiffer which is reflected in the reduced functional residual capacity and low lung compliance. Lung deflation leads to narrowing of peripheral airways and due to the effects of gravity this effect is particularly marked in the basal regions of the lung. As a result, the basal airways may close completely early in expiration and not open until the next inspiration is well advanced. As the inspiration proceeds, the force acting on the walls of the airways increases until the pressure gradient between the open and closed section of the airway reaches a critical value; the closed airway then abruptly opens.

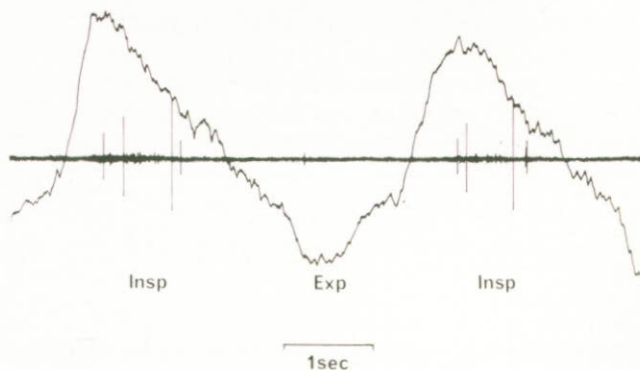


Fig 5. *Repetitive inspiratory crackles in two successive breaths.*

The opening of each airway is accompanied by a crackle. It is important to note that the crackles do not occur at random but repeat from breath to breath *in the same sequence* suggesting that the pressure and volume changes during each breath determine their timing (Fig. 5). The measurements confirm that an individual crackle identified by its timing and amplitude in successive breaths recurs at a similar lung volume and transpulmonary pressure.

Early Inspiratory Crackles (Fig. 6)

Early inspiratory crackles are so named because they are confined to the early phase of inspiration and do not continue to the end of inspiration. They are low pitched sounds, few in number, usually heard at one or both lung bases and are well transmitted to the mouth. They are not modified by cough or by a change in body position.

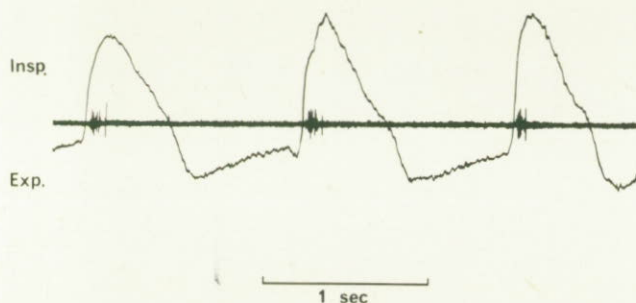


Fig 6. Chronic bronchitis. The crackles are confined to early inspiration.

Early inspiratory crackles are a feature of widespread air-flow obstruction due to chronic bronchitis, asthma and emphysema. In a study of patients with early inspiratory crackles, it was found that they were present only in severe airways obstruction (FEV_1/VC ratio less than 45 per cent). By contrast, the FEV_1/VC ratio of patients with late inspiratory crackles is 70 per cent or more.

As with the late inspiratory crackles, the early inspiratory crackles repeat from breath to breath in the same sequence suggesting that their occurrence is also determined by the mechanical events of breathing. They are believed to originate in the larger airways because they are few in number and are well transmitted to the mouth. In severe airways obstruction, the larger airways are already narrowed and will tend to close prematurely in expiration because the compliance of the airway wall is increased. In the subsequent inspiration they open early as the critical opening pressure required is low.

Early and Mid Inspiratory Crackles (Fig. 7)

In patients with *bronchiectasis*, the inspiratory crackles show a distinct pattern, different from the early crackles of chronic bronchitis and from the late crackles of fibrosing alveolitis. In bronchiectasis, the crackles start early in inspiration, continue to the middle of inspiration and fade by the end of inspiration. Crackles are typically coarse in quality and expiratory crackles are commonly present.

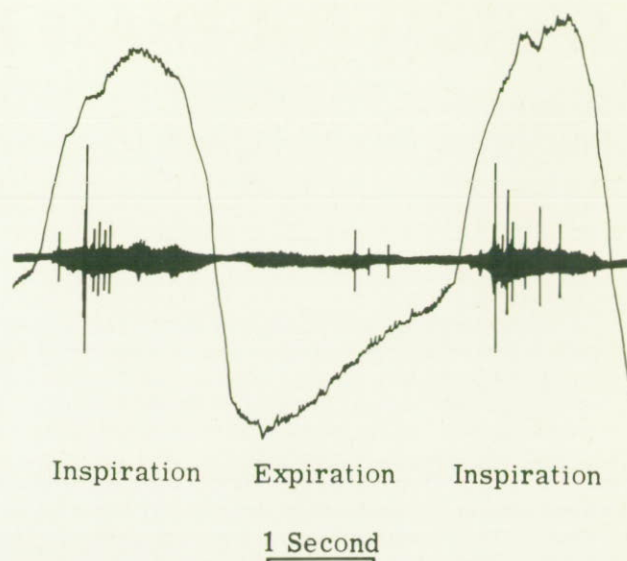


Fig 7. Bronchiectasis: Early and mid inspiratory and expiratory crackles.

As in chronic bronchitis, the lung crackles of bronchiectasis are well transmitted to the mouth suggesting their origin in the proximal bronchi. This observation is in keeping with the bronchographic findings which show abnormalities of the larger and the medium sized bronchi. By contrast the crackles which originate in the peripheral airways are rarely heard at the mouth. It is likely that the crackles relate to retained secretions in the damaged and dilated bronchi. They are typically present in inspiration and expiration and become less abundant after expectoration. Bubbling in retained secretions however is probably only one of the causes of these crackles. The damaged and dilated bronchi with high compliance which close in expiration, may generate crackles when they open in subsequent inspiration.

Pleural Crackles (Pleural Rub)

When the pleural membrane is inflamed or infiltrated, the movement of the adjacent surfaces becomes jerky in a manner similar to the bow of a stringed instrument. Pleural crackles are coarse and non-musical but may acquire a musical quality if the chest wall is set in resonant oscillation. They are usually present in both phases of respiration.

SUMMARY

Some of the lung sounds discussed should be of interest to all those concerned in the evaluation of respiratory disorders. Their interpretation, based on pathological and simultaneous functional changes in the lung has improved our understanding and enhanced the value of auscultation of the chest.

SPUTUM SOL PHASE PROTEINS

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Introduction

The secretions of the tracheobronchial tree are complex fluids containing cells, together with a mixture of substances produced by a variety of cell types lining the walls of the respiratory tract. The secretions are important in controlling humidification of inspired air, trapping and transporting inhaled foreign particles and in protecting the mucosal surface. 95% of this is free and bound water, the remaining 5% being macro-molecular material consisting of high molecular weight glycoproteins (2-3%), soluble proteins (0.1-0.5%) and fats (0.3-0.5%).

In normal individuals the tracheobronchial secretions reach the larynx, mix with the upper respiratory tract secretions and are then swallowed. In diseases of the airways, however, these secretions can be altered in quantity and composition. Chronic bronchitis, asthma, bronchiectasis and cystic fibrosis are all characterised by hyper-secretion of mucus, the quantity of which increases dramatically and can overload mucociliary transport with secretions expectorated as sputum. Since bronchial secretions from healthy subjects are difficult to obtain in sufficient quantity for detailed study, chemical analysis has largely been carried out on sputum from patients with these chronic diseases.

Methods

The analysis of whole sputum has revealed the presence of soluble components although the viscous nature of whole sputum has limited detailed analysis. Various techniques have been used to separate the soluble components including freezing and thawing of whole sputum, ultrasonification or mucolytic digestion followed by dialysis and/or low-speed centrifugation; the simplest and potentially least damaging is the method of Ryley and Brogan¹ using ultracentrifugation (50,000 g for 90 minutes) to separate sputum into "sol" and "gel" phases. The sol phase is of low viscosity and can be removed and stored for analysis leaving the gelatinous phase of high viscosity which may be discarded.

This separation then allows easier analysis of the sol phase of sputum and the presence of several plasma proteins can be determined including: albumin, haptoglobin, transferrin, orosomucoid, the immunoglobulins A, G and M (IgA, IgG and IgM) and the protease inhibitors — α_1 antitrypsin, α_1 antichymotrypsin, α_2 macroglobulin and inter- α -trypsin inhibitor.

Protein Measurements

These proteins are quantitated using immunoprecipitation techniques. The principle is that most plasma proteins are antigenic and, if mixed with a specific antiserum containing antibody against the single protein to be measured, an antigen/antibody precipitation occurs. The reaction can be visualised in gels because of the large size of the precipitating complexes. This can be achieved by allowing the antigen to diffuse into the antibody passively (radial immunodiffusion) or actively using an electric current (immunoelectrophoresis).

a) Radial Immunodiffusion (Mancini et al)²

This technique is for large proteins with little negative charge such as the immunoglobulins A, G and M. The protein (antigen) is allowed to diffuse passively into the antibody radially from the point of application. Diffusion stops when the antigen/antibody reaction reaches equivalence and the limits can be seen with protein staining (Fig. 1a). The square of the diameter of this diffusion ring is proportional to concentration and can be compared with standard samples of known concentration on the same plate.

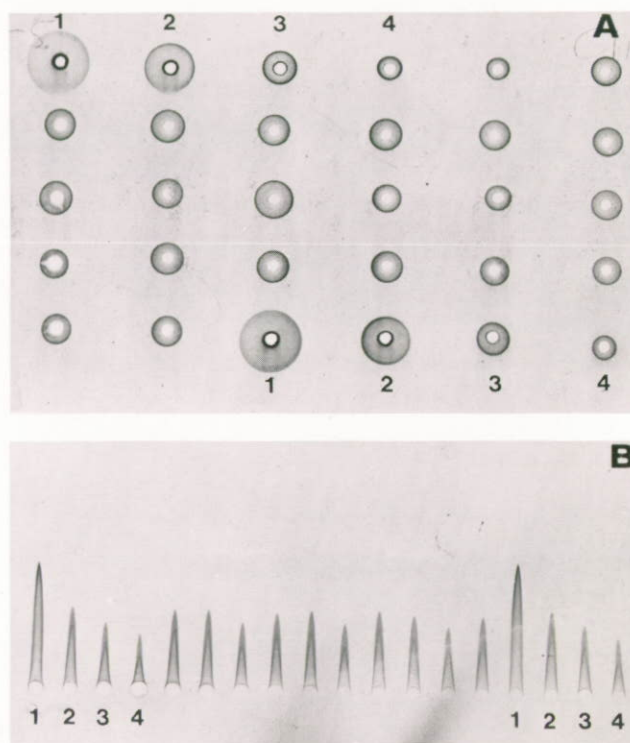


Figure 1:

A) "Mancini" radial immunodiffusion plate.

B) "Laurell" monorocket immunoelectrophoresis.

Unknowns are placed in the wells and compared with standard samples of known concentration on each plate (1, 2, 3, 4).

b) Rocket Immunoelectrophoresis (Laurell)³

This method is faster and depends on the fact that many proteins are negatively charged. Thus, when a current is applied to the sample the proteins move to the anode. This principle can be used to move the proteins into the antibody more rapidly than by diffusion. Precipitation again occurs at the point of antigen/antibody reaction with movement ceasing at the point of equivalence and the limits can be visualised with protein staining (Fig. 1b). The test samples are compared with standard samples of known concentration on the same plate.

Interpretation of Results

Concentration of Proteins in Secretions

Many of the proteins are serum-derived and enter secretions by a process of ultrafiltration. The secretion concentration is, at least in part, dependent on the serum concentration. This is particularly important for proteins such as α_1 antitrypsin where serum deficiency or high concentration due to "acute phase" response during infection will result in similar changes in the secretion. This problem can be overcome by taking sputum/serum ratios of proteins so that individual subjects can be compared, though this is only true for proteins that are entirely serum-derived. This is thought to be the case for albumin and thus the sputum/serum albumin ratio can be used as a standard with which to compare other sputum proteins. Thus proteins of a similar size to albumin will have a similar sputum/serum concentration ratio if they enter the secretion by diffusion alone (Table 1). Proteins of a larger size will be impaired in their diffusion compared to albumin and will thus have a lower sputum/serum ratio (Table 1).

If, however, any protein is locally produced in the lung or has a special transport mechanism to facilitate its movement into secretions, it will be present in a concentration above that determined by diffusion alone. Thus the sputum/serum concentration ratio will be much higher than albumin confirming this "local mechanism", IgA being an example (Table 1).

The sputum/serum albumin ratio can also be used as a guide to the degree of lung inflammation. When the lung is inflamed, an increase in protein transudation will occur and this will be reflected in a raised sputum/serum albumin ratio (Table 1).

Although sample contamination with saliva is a variable problem in the study of sputum, its main effect is dilutional since the concentrations of protein are very low. This problem can be overcome by studying large numbers of patients to balance out the dilutional effect between patient groups. On the other hand, in an individual patient, all proteins in sputum will be diluted by saliva to the same extent. Thus the practice of standardising protein X, by obtaining the ratio of protein X per ratio of albumin, has become an accepted means of comparing individual subjects.

Implications

Much work has been done on the quantitation of sol phase sputum plasma proteins in the various chronic respiratory diseases. In recent years interest has focused on the immunoglobulins, especially IgA, and the protease inhibitors, since it is thought that they are particularly important in protecting the lung.

The protease inhibitor α_1 antitrypsin is the main serum and alveolar inhibitor of proteolytic enzymes; serum deficiency of this protein is associated with early onset of panacinar emphysema (Eriksson, 1965)⁴. This observation together with the fact that instillation of proteolytic enzymes into the respiratory tract of animals produces lesions resembling human emphysema, has led to the concept of the "protease-antiprotease" theory for the pathogenesis of emphysema.

The *proteases* or proteolytic enzymes implicated are the *elastases* which are present in alveolar macrophages and neutrophils. These are the only enzymes capable of destroying the elastin component of the elastic fibre, thereby producing experimental emphysema.

The *anti-proteases* are those inhibitors which possess anti-elastase activity, such as α_1 antitrypsin and

α_2 macroglobulin which are mainly serum-derived, although others may be important. In theory, it appears that a balance between elastases and anti-elastases is required for the maintenance of normal alveolar structures. Factors which upset this balance either intermittently or continuously and allow the lung parenchyma to be exposed to the unimpeded action of elastase potentially result in the subsequent development of emphysema.

The balance may be tipped in favour of the enzymes for a variety of reasons, including the presence of infection when increasing numbers of elastase-containing neutrophils may enter the lung; cigarette smoking not only increases the lung neutrophil count and hence the elastase burden but interferes with the functioning of protease inhibitors. By identifying these factors and understanding them, it may be possible to interfere therapeutically and halt the progress of the disease or detect susceptible individuals in the presymptomatic stage.

Immunoglobulin A (IgA) on the other hand is believed to be the major immunoglobulin which protects epithelial surfaces from bacterial invasion; serum deficiency of this protein is associated with recurrent infections. IgA is regularly found within the bronchial secretions and most is "locally produced". Recent studies suggest, however, that local deficiency of IgA in the secretion can occur in the presence of *normal* serum IgA (Stockley, 1981)⁵ and this may be associated with morbidity and mortality.

Most patients with chronic bronchitis and bronchiectasis regularly produce sputum, develop emphysema and have repeated infective exacerbations and are thus ideal for study in terms of the "protease-antiprotease" theory, IgA production and the general bronchial biochemical environment.

Drug therapies, such as antibiotics, mucolytics and steroids, may affect the local nature of lung secretions. The result may be harmful or beneficial in the long term but an understanding of the nature of these responses is necessary before the implications can be determined.

Considerable information above and beyond bacterial culture can thus be gained from the analysis of sputum in relationship to the bronchial environment. Although most of this work is still in the research and development stage, perhaps you will now think twice about discarding into the waste bin the sputum your patient has just produced during the course of respiratory function testing! You may be losing important information about the pathogenesis of the patient's disease.

Acknowledgment

My grateful thanks to Dr. R. A. Stockley for his help and guidance during the compiling of this article.

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TABLE 1

**SPUTUM/SERUM PROTEIN RATIOS IN CHRONIC
BRONCHITIS**

	Protein	Sputum/serum ratio (n = 20)	
		Mean	Standard error
Standard protein	Albumin	0.75	0.14
Proteins of similar size to albumin	Alpha ₁ antitrypsin	1.13	0.20
	Transferrin	0.81	0.06
Proteins larger than albumin	Alpha ₂ macroglobulin	0.28	0.09
Protein "locally produced"	IgA	13.50	0.53
Inflamed lung	Albumin	5.51	1.69

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ARTERIAL OXYGENATION DURING ONE LUNG ANAESTHESIA FOR THORACIC SURGERY

The effect of different anaesthetic regimes

E. G. Bradshaw and E. E. M. Thompson

Department of Anaesthetics, University of Manchester

SUMMARY

110 patients who underwent thoracic surgery were studied using four different techniques of anaesthesia. All patients received oxygen and nitrous oxide supplemented by one of the following: *either* halothane *or* a continuous infusion of althesin, *or* fentanyl with droperidol *or* a continuous infusion of etomidate. The four groups were each sub-divided into those patients having surgery for 'lung' lesions and those who had surgery for 'non-lung' conditions.

Patients who had surgery for 'non-lung' conditions were found to have significantly lower arterial oxygen tensions during one lung anaesthesia than those who had operations for 'lung' lesions. There was no significant difference in arterial oxygenation between the four anaesthetic techniques.

INTRODUCTION

The reduction in arterial oxygen tension (PaO_2) which has been reported during one lung anaesthesia (OLV) is presumably due to continued perfusion of the non-ventilated (upper) lung as a result, in part, of the failure of the pulmonary hypoxic vasoconstrictor reflex. Animal studies have shown that this reflex may be depressed by various inhalational anaesthetic agents but the evidence for these effects in humans ventilated with halothane or ether is inconsistent (Bjertnaes et al 1976). These investigators showed that the pulmonary hypoxic vasoconstrictor reflex seems to be unaffected by intravenous techniques such as neuroleptanaesthesia (NLA). Because inhalational agents may have a direct effect on the pulmonary vasculature the purpose of this study was to investigate the effect of an inhalational agent, halothane, and three different intravenous anaesthetic techniques on PaO_2 during endobronchial anaesthesia and OLV in patients undergoing (a) 'lung' surgery and (b) surgery not involving the lung, such as cardio-oesophagectomy and hiatus hernia repair.

METHODS

Adult patients who presented for anaesthesia for thoracic surgery were allocated to one of eight groups (Table 1). As premedication, the patients usually received either diazepam 10 mg or lorazepam 3.5 mg orally. Groups I and II: anaesthesia was induced with althesin and maintained with halothane and nitrous oxide and oxygen. Groups III and IV: anaesthesia was induced with a sleep dose of althesin and maintained with an infusion of 25 ml althesin which was administered continuously during the operation and discontinued ten minutes prior to skin closure. Most patients required only one infusion of 25 ml of althesin but, if necessary, this was repeated. Groups V and VI: the patients received NLA, fentanyl 250 to 500 μg and droperidol 0.2 to 0.4 mgs per kg to induce anaesthesia, and nitrous oxide and oxygen with increments of 50 to 200 μg of fentanyl to maintain anaes-

TABLE 1

Groups	Surgery	Anaesthetic
I	Lung	Halothane
II	Non-lung	
III	Lung	Althesin infusion
IV	Non-lung	
V	Lung	Fentanyl and Droperidol
VI	Non-lung	
VII	Lung	Etomidate infusion
VIII	Non-lung	

thesia. Groups VII and VIII: received 250 to 500 μg of fentanyl followed by a sleep dose of etomidate; anaesthesia was maintained with nitrous oxide and oxygen and a continuous infusion of etomidate at a rate of 10 to 40 $\mu\text{g}/\text{kg}/\text{min}$ administered by a syringe pump. Muscle relaxation was maintained in all patients by intermittent doses of pancuronium. The majority of patients were intubated using appropriately sized left or right double-lumen Robertshaw endobronchial tubes; the remainder were intubated using a Carlens double-lumen tube. The patients were ventilated to normocarbida with not less than 50% oxygen in nitrous oxide using a Blease ventilator. The ECG and body temperature were monitored throughout the operation and the fluid balance maintained with crystalloids and blood when required.

Arterial blood samples were taken at the following times: The *first* was immediately prior to OLV with 50% oxygen/nitrous oxide, a second at 20 minutes after the start of OLV with 50% oxygen/nitrous oxide and a third, ten minutes after OLV with 100% oxygen. Provided that the first oxygen tension on OLV was acceptable, the patient returned to a further period of ten minutes of ventilation with 50% oxygen/nitrous oxide. Further samples were taken if clinically indicated. In patients who had not undergone pneumonectomy another sample was taken after two lung ventilation (TLV) had been restored. The results were analysed statistically with the Mann-Whitney U test.

RESULTS

PaO_2 results for the patients undergoing 'lung' surgery are shown in Fig. 1, and those for 'non-lung' surgery in Fig. 2. There was a significant fall in oxygen tension in all patients on OLV with 50% oxygen. The fall was significantly greater in the 'non-lung' patients ($P < 0.05$) although there were some dangerously low oxygen tensions in the 'lung' patients. Changing to 100% oxygen, PaO_2 increased in all cases to at least minimally acceptable levels. The overall improvement was significantly greater in the patients undergoing 'lung' surgery ($P < 0.002$). Despite the different anaesthetic techniques used no significant differences in PaO_2 between the groups could be demonstrated. There were no differences in the arterial carbon dioxide tensions and acid-base status between TLV and OLV or between the groups.

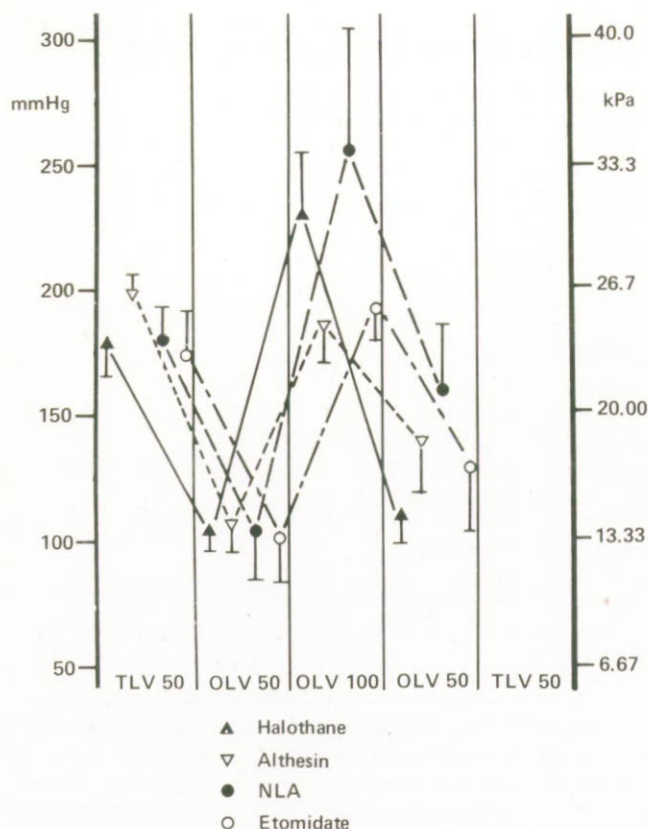


Fig 1. Changes in PaO_2 during anaesthesia for lung disorders.

TLV 50: Two lung ventilation with 50% O_2

OLV 50: One lung ventilation with 50% O_2

OLV 100: One lung ventilation with 100% O_2

NLA: Neuroleptanaesthesia (fentanyl and droperidol)

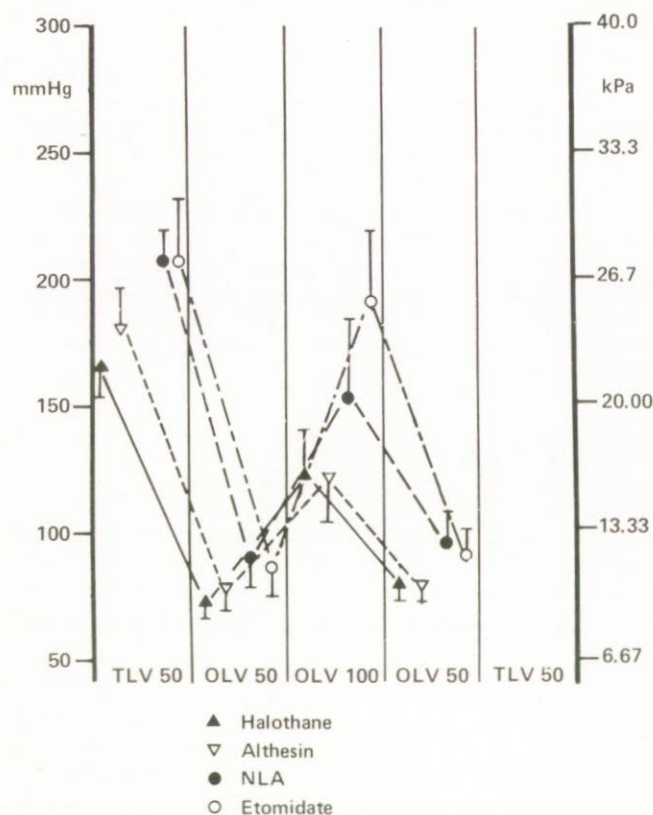


Fig 2. Changes in PaO_2 during anaesthesia for non-lung disorders. Abbreviations as Fig 1.

DISCUSSION

The findings of significant falls in PaO_2 during periods of OLV particularly in patients undergoing 'non-lung' surgery agree with those of Kerr et al (1974) who found the mean shunt in this group of patients to be over 40%. In certain patients in this group there were such extreme falls in PaO_2 (50–60 mm Hg) as to suggest that there may be shunting in the ventilated lung as well as in the collapsed upper lung. This might have been due to misplacement of the double-lumen tube so that the right upper lobe was inadequately ventilated or to compression of the lung in the lateral position by the weight of the mediastinum and the pressure of the abdominal contents on the diaphragm. There was no evidence of a hypoxic pulmonary vasoconstrictor reflex progressively diverting blood away from the non-ventilated lung during endobronchial anaesthesia. Results of earlier animal studies have reported that the pulmonary hypoxic pressor response is depressed reversibly after ventilation with volatile anaesthetic agents such as 1 to 1.5% halothane in the isolated lung (Sykes et al 1973), although this is not so in the intact dog anaesthetised with halothane 0.75 to 1.0% (Sykes et al 1978). It could be argued that one-lung ventilation in 'non-lung' surgical cases should be abandoned because of the dangerously low oxygen tensions that may occur but Thompson and Campbell (1973) demonstrated that heavy surgical retraction, which would be necessary especially in middle third oesophageal surgery, may produce even lower oxygen tensions than one lung anaesthesia. While the use of 100% oxygen ensures adequate oxygenation in nearly all patients it is unnecessary in many, and makes the maintenance of anaesthesia without the risk of awareness more difficult. Recent studies in patients (Capan et al 1980) suggest that the best arterial oxygenation can be obtained using oxygen insufflation of the upper deflated lung at 10 cm of water pressure while a lower lung is ventilated with zero end-expiratory pressure.

ACKNOWLEDGMENTS

We wish to thank Ms. M. Marples for her invaluable assistance in this project.

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MEDICAL MEMO

Fibrosing alveolitis

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Chest Unit, King's College Hospital Medical School

The term 'fibrosing alveolitis' includes a group of disorders in which there is an accumulation of macrophages and other cells in the interstitial space and within the alveoli; this is associated with progressive fibrosis of the alveolar walls.

The disease has a number of causes and it is convenient to divide it into two main classes:

- 1. Extrinsic or allergic alveolitis.
- 2. Intrinsic or cryptogenic alveolitis.

EXTRINSIC ALVEOLITIS

This disorder is brought about by the inhalation of a variety of organic dusts, the commonest cause in the UK being 'farmers' lung', which results from the inhalation of fungal spores from mouldy hay; inhalation of other dusts leads to disorders such as 'bird-fancier's lung', 'mushroom-worker's lung', 'malt-worker's lung' and so on.

CRYPTOGENIC FIBROSING ALVEOLITIS

The basic cause of this type of alveolitis, is unknown as the name implies but there is increasing evidence that auto-immune processes may play a part.

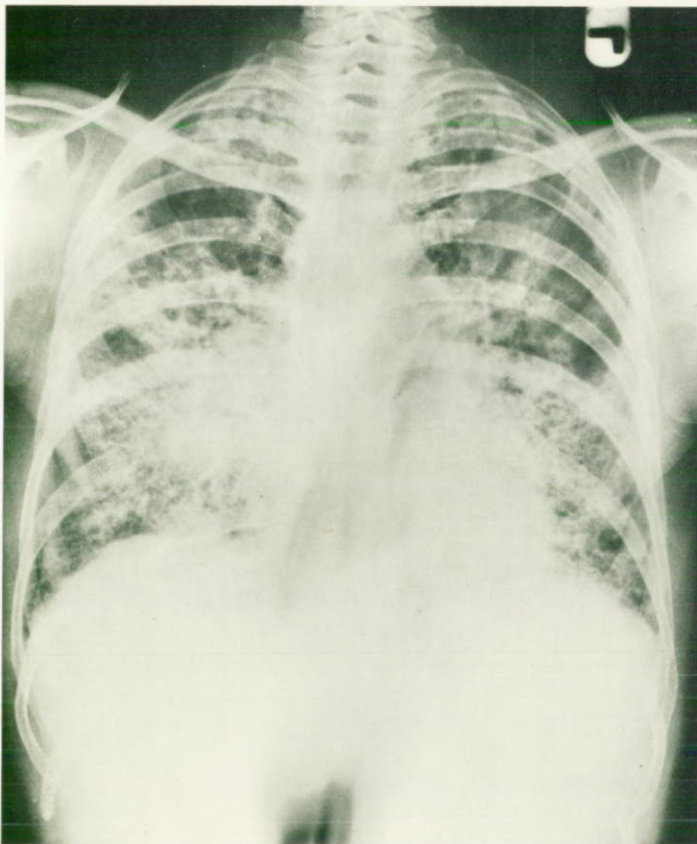


Fig 1. Chest radiographs in fibrosing alveolitis. There are multiple nodular opacities in both lung fields.

SYMPTOMS

Allergic form

Acute exposure: Some three to six hours after exposure, the patient develops shortness of breath, cough and fever which can last for up to twenty-four hours.

Chronic exposure: There is progressive shortness of breath and weight loss.

Cryptogenic form

Progressive shortness of breath is the usual feature.

SIGNS

Obvious *crackles* at the lung bases can be heard with the stethoscope in nearly all cases.

Finger clubbing: curvature of the nails and enlargement of the finger ends are seen in the majority.

Chest radiograph (Fig 1): multiple small nodular or confluent shadows are seen and in the advanced case this leads to the appearance known as 'honeycomb lung'.

PATHOLOGY

Cellular phase: in the early stages there is infiltration of the alveolar spaces with polymorphs, lymphocytes or macrophages. In the chronic phase, there is gross thickening of the alveolar walls by fibrous tissue (Fig 2).

PULMONARY FUNCTION

The characteristic abnormality is a 'restrictive' defect with much reduced lung volumes and a low CO transfer factor (Table 1). Severe arterial hypoxaemia is usually present, which stimulates ventilation and leads to reduction in arterial pCO₂. If compliance is measured, the lungs are found to be much stiffer than normal.

TABLE 1: LUNG FUNCTION TESTS IN FIBROSING ALVEOLITIS

	Patient	Expected normal value
FEV ₁ (litres)	1.2	2.6
VC (litres)	1.4	3.6
FEV ₁ /VC %	86	75
TLC (litres)	3.5	6.0
CO transfer (mmol/kPa/min)	2.5	7.7
Arterial pCO ₂ (kPa)	3.8	5 to 6
Arterial pO ₂ (kPa)	6.5	11 to 12.5

TREATMENT AND PROGNOSIS

In the allergic form, exposure to the responsible antigen ideally should cease at once.

The outlook in the chronic cases is variable. Corticosteroids in large dosage are the usual first line of treatment and some improvement can be expected in cases where there is an extensive cellular component. Once fibrosis has set in however, the condition is irreversible and death from respiratory failure with cor pulmonale is the likely outcome.

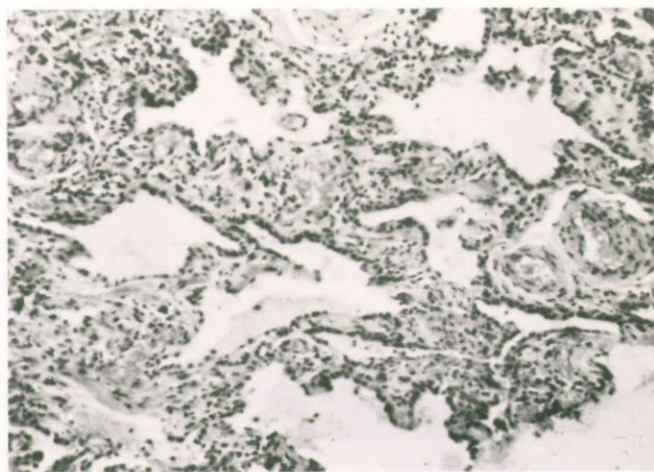
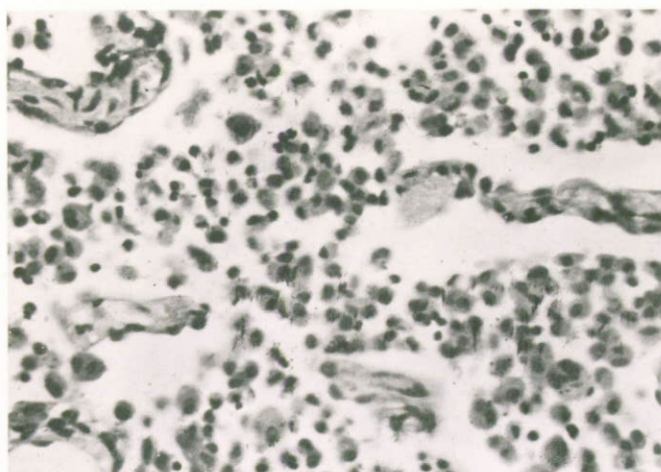


Fig 2. Pathological features illustrating the two ends of the range of appearances.

a) Cellular form: The alveolar spaces contain numerous macrophages and chronic inflammatory cells.

b) Fibrotic form: The alveolar walls are grossly thickened with oedema, inflammatory cells and fibrous tissue.

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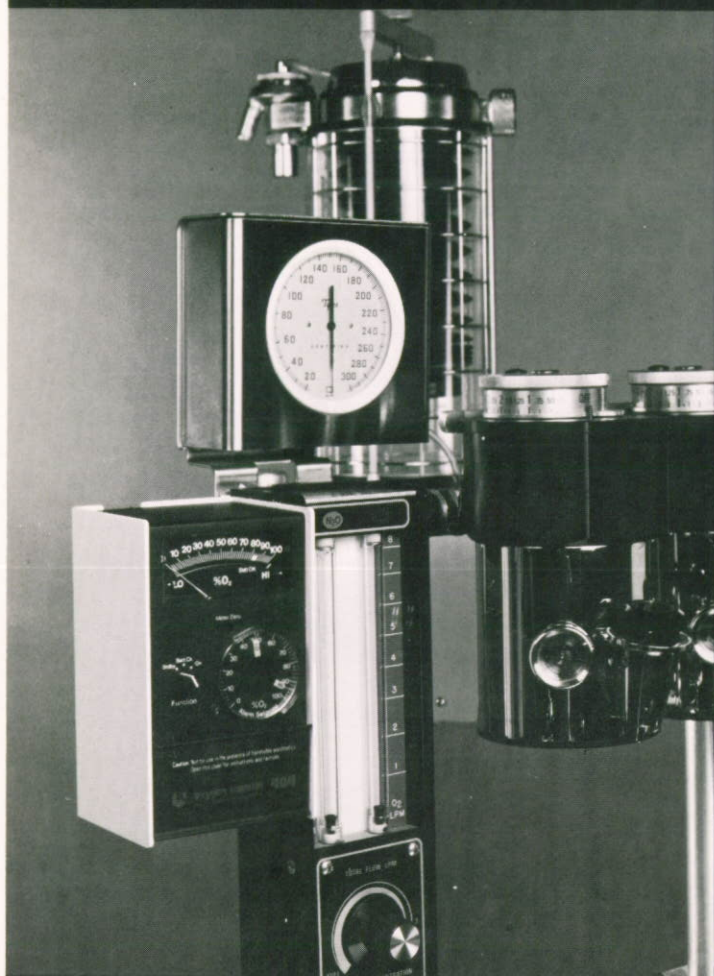
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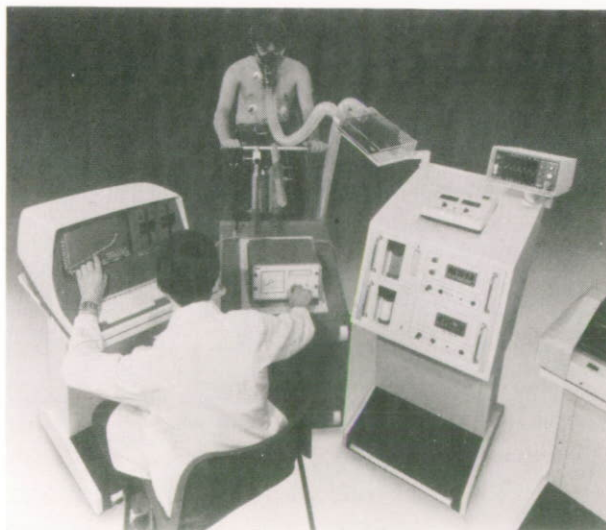
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