Inspire

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FIRST WORD

The venue for the ARTP Winter Meeting 2000 is Daventry in the West Midlands, date 10th - 12th February. The Executive Committee is in the process of putting together the programme for the event. We have taken into account all the useful comments from the last winter conference and hope to make the meeting even more interesting, relevant and enjoyable than the last one. New for the meeting will be a poster session. You will find an abstract form for the poster session on page 3 in this edition of INSPIRE. We urge as many members as possible to submit an abstract - the session does not have a theme (other than respiratory measurements!) therefore we hope to display a varied and diverse selection of subjects. The deadline date for abstract submission is 10th January - so start writing.

The results of the recent postal vote concerning the new constitution, disciplinary code and code of conduct are as follows:

New constitution: 96 voted to accept 2 voted to reject

Code of conduct: 96 voted to accept 2 voted to reject

Disciplinary code: 96 voted to accept 2 voted to reject

As greater than 2/3rds of the vote were in favour of the changes they were formally accepted.

The deadline for articles, letters and any other contributions for the next issue of INSPIRE is 20th November 1999. Please write to me:

Sue Revill
Department of Respiratory Medicine
Glenfield Hospital
Leicester LE3 9QP

DATES FOR YOUR DIARY

13th - 15th September
Conquering airway inflammation in the 21st century
London

13th - 17th September 1999
Advanced Course for Respiratory Physiology
Coventry University

6th October
Allergy in laboratory workers
London

9th - 13th October 1999
European Respiratory Society Annual Congress
Madrid

18th - 19th November
Occupational and environmental lung diseases; recent advances
London

10th - 12th February 2000
ARTP Winter Meeting
Daventry
West Midlands

See page 19 for more details

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CHARITY NEWS

National Asthma Campaign

London H/Q tel no for enquiries or leaflets 0171 226 2260
For fund raising activities and national events contact Beck Bayram 0171 226 2260 ext 342, e-mail bbayram@asthma.org.uk

Ad-hoc fund raising packs are available for one-off events such as a child's sponsored silence for the weekend, sitting in a tub of baked beans, or any other wacky past-time you can imagine!

GREAT BRITISH SWIM starts 11th October - packs from London H/Q. Last year 13,000 swimmers took part and the total distance covered was just 300 miles short of the target 6,629 miles - the length of the British coastline.
LONDON TRIATHLON, 19th Sept places are available free if you can raise £600. Enter as a team of three with each person doing a leg each.
NEW YORK MARATHON, 7th November, raise £2000 and have your fee, flight and accommodation refunded.
4th and 5th November Barbados half and full marathons. Run a race, raise £2000 and stay in Barbados for 2 weeks for free !

The Asthma Agenda can be found on the charity's web site at **http//www.asthma.org.uk.** The web site carries all the latest up to date news, statistics and research concerning asthma.

British Lung Foundation

For tickets and information contact The BLF London office on 0171 831 5831.

21st - 28th November. Cycle 220 miles from N to S Jordan, raise £2000, pay £195 entry fee towards flight, accommodation and back-up.

UPDATE ON STATUTORY REGULATION OF PRACTICE

Dr Sue Hill, *Department of Respiratory Medicine, Queen Elizabeth Hospital, Birmingham*

Earlier this year you were updated on the position relating to statutory regulation of practice for scientific and technological staff under the aegis of the Council for Professions Supplementary to Medicine (CPSM). At that time 3 of the 12 possible 'Boards' to undertake the process of state registration for different professional groupings were vacant with 3 petitions awaiting approval (Ambulance paramedics, Speech and Language Therapists and Clinical Scientists). All of these petitions were from coherent staff groupings who could be represented by a single professional umbrella organisation. In June of this year Privvy Council approval was granted for these 3 petitions under the 1960 Act for Professions Supplementary to Medicine meaning that no other 'Boards' could be formed under this old legislation. However the 1960 Act is due to be repealed soon and in its place secondary legislation will be crafted with the aim to modernise and update the current system including a requirement for CPD as well as to allow other groups of staff to be included if they fulfill the requirements and their practice subsequently regulated.

Against this backdrop a forum for Clinical Physiology with representation from audiology and hearing therapy, cardiology, gastroenterology, neurophysiology and respiratory physiology (with renal technology as an observer) was established last year. This forum has been working on a common agenda of putting together a case for consideration by CPSM for statutory regulation of practice under the 'new' secondary legislation and of developing an infrastructure for voluntary registration in the first instance and has been fulfilling the role of the necessary umbrella professional organisation. The case will be a submission from Clinical Physiology as a whole staff grouping (which would potentially be the protected title for the group of staff) comprising of a number of distinct specialities (for example respiratory physiology) since it became evident that single specialities would stand little chance of becoming state registered due to the small numbers involved- the combined group represents approximately 10,000 staff members in the UK.

We are optimistic that the case will be approved and that a system for voluntary registration can be put into place with all the necessary requirements for eventual approved statutory regulation of practice, in much the same way as the Clinical Scientists have done. Part of the process involves the setting of the educational entry to the independent practitioner level (i.e. the level which requires statutory regulation of practice) which is likely to be set at degree level or equivalent and the establishment of grandfather clauses for individuals who may have been in post for many years working as an independent practitioner and who may not hold the recognised qualifications but who can provide evidence of competent practice in all of the specified areas. To develop the academic courses that are required a liaison group between the Clinical Physiology Forum and colleges and higher education institutes has been established and will be meeting on a regular basis in the presence of the Chief Scientific Officer of the Department of Health who is supportive of the developments overall. It is currently impossible to provide any more detail until the case has been submitted to CPSM and a decision is known. It is hoped that this will be achieved by the end of September. Following this outcome the ARTP plans to hold a series of meetings in different geographical locations to which a representative from each respiratory physiology laboratory will be invited to discuss the specific details relating to statutory regulation of practice (as well as other items of business). It is critical that issues and problems are raised and discussed early in the process and where possible integrated into the process.

For Clinical Scientists employed in respiratory physiology laboratories there will be two options available. The first will be to become state registered via the newly approved Clinical Scientists Board which should be up and running formally next year after which time the title of Clinical Scientist will be protected and only open to state registered individuals who fulfill the criteria. The Clinical Scientists petition recognised a number of specialities including clinical physiology and under this heading a number of sub-specialities including respiratory physiology. To date although the criteria against which evidence will need to be provided of competent practice is available in a generic form there is a need for clear pathways to be established in respiratory physiology. To help this process a higher degree is being developed (MSc) as a potential exit academic qualification from Clinical Scientist training programmes in the discipline together with vocational training outcomes and an assessment of competence. Until the new Board is fully established the Registration Council for Scientists in Health Care will continue to operate and the ARTP is keen to help Clinical Scientists to become voluntary registered under this system. Again the exact requirements are not well defined for respiratory physiology but a working model is available (contact me for details plus registration forms). The second option if the criteria for acceptance onto the register is not fulfilled which does include evidence of scientific research would be state registration under the proposed system for Clinical Physiology as a protected title as outlined above. It is worth noting that some currently employed MTO's may fulfill the criteria for registration as a clinical scientist. Since it is important that an infrastructure for clinical scientists is established in the near future the proposed ARTP meetings for representatives of respiratory physiology laboratories to be held in the Autumn will include a session both about the clinical scientists state registration and specifically for clinical scientists to discuss pertinent issues.

Please do not hesitate to contact me on these important issues. When more news is available this will be communicated to you in an attempt to keep you fully informed of the process. This is an exciting time for science and technology within health care and we hope that statutory regulation of practice will be one that you welcome.

ABSTRACT FORM

ARTP WINTER MEETING

DAVENTRY, 10th -12th FEBRUARY 2000

Name ..... Grade .....

Dept. Address .....

Tel No ..... Fax No ..... e-mail .....

Please type within the box.

Return with 3 photocopies to Dr S. Revill, Dept of Respiratory Medicine, Glenfield Hospital, Leicester LE3 9QP

Deadline: **10th January 2000**

Abstract Instructions

Deadline: this abstract must be received together with 3 photocopies before 10th January 2000.

PLEASE NOTE:

1. Trade names should not be mentioned in the title. However, trade names in brackets, will be accepted in the body of the text.
2. References (max 2) can be included in the body of the text (e.g. Wilson, M.J. et al. Nature 1996; 67: 24-30).
3. Abbreviations should be defined.
4. Avoid a sweeping or potentially unwarranted final statement.
5. The form MUST be completed by the presenting author.
6. Email and FAX submissions WILL NOT be accepted.

TYPING INSTRUCTIONS

1. Use an electric typewriter or word processor and type within the space outlined.
2. Symbols not available in typeface should be drawn in black ink only.
3. Type within the large square your entire abstract including title in capital letters, name(s) of author(s), institution and address, free of smudges and errors. Leave one line between the address and the abstract text. Practice first on a photocopied abstract form. Abstracts which do not conform will be returned and not accepted.
4. Underline the name of the presenting author.
5. Abstracts should contain not more than 200 words excluding the title and authors.

Mail the unfolded abstract and 3 good photocopies to:

Dr Sue Revill, ARTP 2000 Abstracts
Dept of Respiratory Medicine, Glenfield Hospital, Leicester LE3 9QP.

NOTE:
THE PRESENTING AUTHORS OF ACCEPTED ABSTRACTS WILL HAVE THEIR FULL CONFERENCE FEE WAVERED

. . . BURSARY REPORT . . .

OXYGEN SATURATION

The highs and the lows

LAURA WATSON – ***Senior Chief Technician, Lung Function Laboratory, QMC, Nottingham***

HOW IT ALL BEGAN

The use of pulse oximetry as a method of assessing levels of oxygenation in blood is becoming increasingly popular, but the technology behind it has been around for nearly 70 years[1].

Oximetry depends upon measurement of the colour change in blood. In 1862 haemoglobin was identified and named by Felix Hoppe-Seyler, and it was George Gabriel Stokes who demonstrated that when transporting oxygen haemoglobin changed its colour. The spectroscope was being used from the 1860s in Bunsen and Kirchoff's laboratory in Heidelberg. In 1874 Karl von Vierordt had effectively invented the first oximeter when he spectroscopically measured the change in red light penetrating his hand when he tourniqueted his arm. This discovery was not pursued for over 55 years, and it was not applied to the routine monitoring of oxygen levels for more than 100 years.

In 1929 an American physiologist called Glen Alan Milikan went to Cambridge to work in Barcroft's laboratory where Roughton supervised the optical measurement of the speed of combination of oxygen with haemoglobin. During World War 11 Lord Adrian asked Millikan for help with problems encountered from hypoxia during military training at high altitudes. Millikan had by then returned to Philadelphia, where he and John Pappenheimer developed a lightweight optical device which could be placed on the earlobe and would provide a non-invasive estimation of SaO_2. He called this device an 'oximeter', and it was designed primarily to be used in aviation research to assess the SaO_2 of pilots flying at high altitudes[2].

In 1947 Julius Comroe used an early ear oximeter to conduct a study demonstrating the unreliability of clinical signs in detecting cyanosis in 3673 patients. The 1950's saw an increase in developmental work on the oximeter world wide, and by 1964 a surgeon called Robert Shaw in San Francisco began the design and construction of a self-calibrating, 8 wavelength ear oximeter. This was marketed by Hewlett Packard in 1970, when the 'modern era' of oximetery really began. This oximeter was expensive ($10,000), but had improved accuracy and gave a reliable indication of arterial oxygen saturation (except at low levels). By the late 1970s it became the 'gold standard' for non-invasive oximetry.

In the mid 1970s Takuo Aoyagi (an engineer working for the Nihon Kohden Corporation in Tokyo) developed what was to be known as a 'pulse oximeter', and so revolutionised the field of non-invasive oximetry. Following this work in the late 1970s, Scott Wilber (an engineer in Boulder, Colorado) made two important improvements on the pulse oximeter which allowed it to develop into a clinically relevant monitoring device. Firstly, he used solid state LEDs and photodetectors to produce a much smaller and lightweight probe. Secondly, he incorporated a microprocessor into the monitor to allow more complex analysis of the pulsatile data and so significantly improving the accuracy of the oximeter. He started Biox (now known as Ohmeda) based on his patented computation of saturation from the red to infra-red ratio of the AC to DC ratios of transmitted light.

The anaesthetist William New of Stanford realised the advantages of pulse oximetry in clinical medicine, and with engineer Jack Lloyd, founded Nellcor. The response of pulse oximeters to hypoxaemia (i.e. saturations 40 - 70%) was studied by Severinghaus and Niafeh.[3] They found that pulse oximeters were inaccurate at low saturations, and were unable to accurately track rapidly developing hypoxaemia. From this data manufacturers adjusted the response of the pulse oximeter to improve performance at low saturations.

The pulse oximeters available today are precise and accurate when measuring saturations from 70 - 100%,[4,5] but may not be reliable at lower oxygen saturations. Today there are many pulse oximeters available for a variety of applications in clinical medicine both in and out of the hospital setting.

CLINICAL APPLICATIONS OF OXIMETRY

The recently published COPD guidelines[6] suggest that blood gases need not be measured unless the SpO_2 < 92%. Here at the QMC we have been using a similar regime for the past year or so, in order to prevent patients with normal oxygen levels from having unnecessary blood gas measurements performed, assuming that if the SpO_2 is higher than 92% then the PaO_2 will be greater than 8 kPa and supplemental oxygen would not be required.

We had noticed during this time that occasionally when the SpO_2 > 92% the PaO_2 could be < 8 kPa. This posed the question - were we missing some hypoxic patients by only measuring blood gases if the SpO_2 < 92%? In order to investigate this hypothesis we decided to perform a small study.

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STUDY PROTOCOLS

Study 1

20 consecutive patients requiring blood gas measurements had their SpO_2 measured using a finger probe connected to an Ohmeda Biox 3700e oximeter (Ohmeda, Hatfield, Herts), and capillary blood gases measured on a Ciba Corning 840 Blood gas machine (Ciba Corning Diagnostics Corp, Medfield, Mass, USA). The Ciba calculates the SaO_2 using a relationship described by Kelman and Thomas. Since oxygen saturation also depends upon the level of carbon monoxide and 2,3 diphosphoglycerate (2,3 DPG) in the blood, the calculated value for SaO_2 may not be equal to the measured value in patients with abnormal levels of 2,3 DPG or carbon monoxide, but this did not occur with any patients included in the studies.

The calculated SaO_2 was compared with the SpO_2 measured using the finger probe, and the difference between the calculated SaO_2 and the measured SpO_2 was recorded. The SaO_2 calculated from the capillary blood was taken as the true saturation, with the non-invasive SpO_2 compared to this value. Capillary blood gases were collected and measured according to the procedure outlined in Appendix 1.

It was noted that out of the 20 patients studied 4 (20%) had a finger probe saturation of 92% or greater but the $PaO_2 < 8$ kPa. It was also noted that there was a wide variability between the calculated SaO_2 and the measured SpO_2, ranging from +18% to –6%.

The results illustrated that hypoxic patients may be missed if relying on SpO_2 of 92% or above. In order to examine if errors in SpO_2 were due to the site of measurement (i.e. finger probe), we decided to look at the SaO_2 calculated from the blood gas measurements to see if the PaO_2 was ever less than 8 kPa when the calculated SaO_2 was less than or equal to 92%.

Study 2

Taking a larger sample size we looked at 100 consecutive capillary blood gas measurements comparing the calculated SaO_2 and the measured PaO_2. We found that only 1 out of the 30 patients who had a PaO_2 of less than 8 kPa had an SaO_2 greater than 92%. In a third study the method of measuring SaO_2 was examined.

Study 3

SaO_2 was measured on 20 consecutive patients using a finger probe, an ear probe on a hyperaemic ear (pre warmed using a vasodilator cream) as recommended, and also by placing the ear probe directly onto the ear lobe at normal body temperature (not the recommended procedure, however the method used by medical/nursing staff if an ear probe is available). The probes were attached to the finger and ear lobe as described in Appendix 2. The SaO_2 calculated from the blood gas data, and the PaO_2 (kPa) was also measured. From this data the difference between the measured SpO_2 for each method and the calculated SaO_2 was recorded, (table 1).

TABLE 1

SpO_2 Finger probe	Difference from BG calculated	SpO_2 Ear lobe (body temp)	Difference from BG calculated	SpO_2 Ear lobe (warmed	Difference from BG calculated	PaO_2 kPa	SaO_2 BG calculated
88	–5	90	–3	93	0	8.26	93.1
90	–1	92	+1	92	+1	8.11	90.7
95	+2	94	+1	91	–2	8.76	92.8
94	–2	92	–4	94	–2	10.78	95.6
98	+3*	96	+1	97	+2*	9.67	95.2
94	0	96	+2*	94	0	9.20	94.4
88	–8*	94	–2	91	–5*	10.19	96.2
92	–4	96	0	96	0	10.76	96.0
94	0	94	0	96	+2*	9.18	94.3
94	–1	94	–1	93	–2	10.09	95.0
82	–1	74	–9*	79	–4	5.85	83.0
91	–5	87	–9*	92	–4	9.61	95.9
90	+1	89	0	87	–2	7.30	88.6
96	+3*	91	–2	93	0	8.85	93.4
100	+2	95	–3	97	–1	12.33	97.7
91	–5	94	–2	94	–2	10.41	95.7
97	0	94	–3	94	–3	11.49	97.4
97	+1	94	–2	97	+1	10.68	96.2
94	–2	96	0	94	–2	10.21	96.2
86	–4	85	–5	86	–4	7.21	89.9

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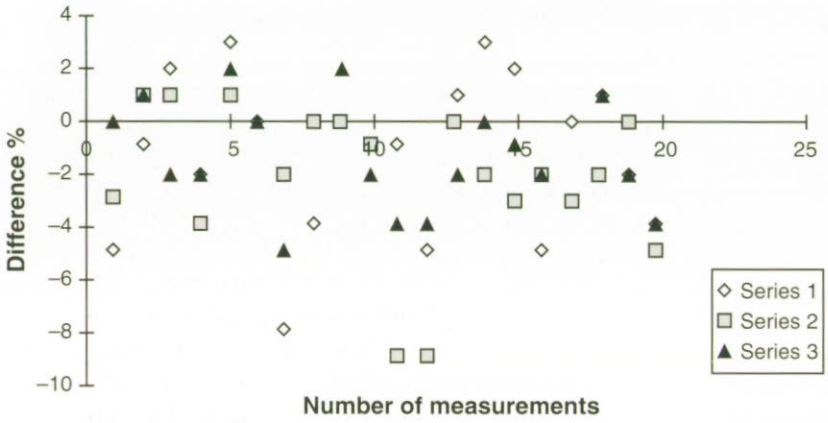
There was wide variation between the calculated SaO2 and the SpO2 measured from the finger or ear probe e.g. measured values of 2 or 3% higher than the calculated values (finger probe 3% higher, ear probe 2% higher), and 9% lower than the calculated SaO2 (finger probe and un-vasodilated ear lobe underestimated by approximately the same amount, i.e. 8% and 9% lower respectively), with the hyperaemic ear probe underestimating the SpO2 by the least amount (5%).

FIGURE 1

Difference between calculated SaO_2 and measured SpO_2



Where: Series 1 from the finger probe
Series 2 from the ear probe (cold)
Series 3 from the vasodilated ear probe

The mean differences (95% confidence intervals) in SaO2, between the measured values and the calculated values were: Finger probe - 1.3% (-2.7, 0.12); Ear probe (no vasodilatation) - 2.0% (-3.4, -0.6); Ear probe (hyperaemic) - 1.4% (-2.4, -0.44).

CONCLUSION

The results show that there is very little difference between the finger and the hyperaemic ear lobe as sites of measurement, but the finger probe underestimates the SpO_2 to a lesser degree. The underestimation is probably not as great a clinical problem as it may seem, as any underestimation of the SpO_2 would result in a blood gas measurement in order to check the PaO_2. An overestimation of the SpO_2 is a much greater problem as a hypoxic patient may be missed if the SpO_2 is overestimated.

In some cases a SpO_2 of 92% may give a PaO_2 of 8 kPa or less, thus if there are clinical signs of cyanosis the blood gases should be checked. In our laboratory we are now using a saturation of 94% as the cut-off limit, and any saturations below 94% will automatically have blood gas analysis. We are continuing to collect SpO_2 and PaO_2 data from patients presenting to the laboratory for blood gas measurement, and hope to re-evaluate a larger sample of data in the future.

APPENDIX 1

Capillary blood gas sampling

In adults capillary blood is taken from the ear lobe. Correct sampling procedures will give values very close to arterial levels[7,8,9]. All blood is mixed with an anti-coagulant (the capillary tubes are usually coated with Lithium Heparin) before analysis to prevent clot formation.

Equipment required: Pre-heparinised glass capillary tubes and end caps, rubber bung, sterile lancet, paper towel, vasodilator cream (Algipan), gauze swabs, cotton wool balls.

Procedure:

Ear rings should be removed, and hair pinned back away from the ear. A paper towel is placed on the patients shoulder to protect clothing.

The circulation to the ear is stimulated by vigorous rubbing with a gauze swab. This is assisted by the application of a vasodilator cream (Algipan), which is applied to the ear lobe approximately 15 minutes before sampling.

The cream is thoroughly removed and the ear rubbed vigorously again.

The ear lobe is held firmly, and supported by placing a rubber bung behind the ear lobe. A sterile lancet is used to quickly stab the ear about 3 mm from the edge of the earlobe, and to a depth of about 3 mm. Blood flow from the puncture site should be rapid. The earlobe should not be squeezed, as this will contaminate the sample with tissue fluid and will result in a lower pH, PaO_2 and SaO_2, and an increase in $PaCO_2$. The sample should be discarded if the flow of blood is slow, as it may not reflect true arterial blood gas levels.

The blood sample (approximately 0.1 ml) is collected in a pre-heparinised glass capillary tube. The tube will fill by capillary action when one end is placed into the well of blood.

The capillary tube is capped in order to maintain anaerobic conditions before analysis. The sample must be well mixed before analysis.

Cotton wool is placed on the patients ear lobe, and the patient asked to apply pressure until the bleeding has stopped.

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APPENDIX 2

Attachment of the probes

The probe comprises a light source and a sensor (photodetector) which are placed across a pulsatile vascular bed (ear lobe or finger tip). The light source consists of two LEDs that emit light at known wavelengths - 660nm (red) and 940nm (infra red). These wavelengths are used as the absorption characteristics of HbO_2 and HbO are significantly different at these two wavelengths. By comparing the ratio R of pulsatile and baseline absorption at these two wavelengths, the ratio of oxyhaemoglobin to reduced haemoglobin is calculated.

The oxygen saturation is calculated from the ratio based on experimental data; there is no known mathematical relationship between oxygen saturation and R[10].

Finger probe

Proper coverage of the photodetector is essential, and so the finger used for monitoring should be chosen as the one which best covers the photodetector and seats correctly in the lower half of the probe housing. Any nail varnish should be removed before placing the probe on the finger, and the probe cleaned before and after each use. The patients finger is inserted into the probe housing until it touches the raised finger stop inside the probe, being sure that the surface of the finger covers the detector window on the lower inside surface of the probe, and the light source is shining through the nail bed. The hand should be relaxed.

Ear probe

The surface of the probe was cleaned before and after use. The ear lobe had either been previously warmed using a vasodilator cream (capillary blood gases had been previously measured from this vasodilated ear lobe), or the probe was placed on the other ear lobe (at normal body temperature). The ear probe was centred with the light source side towards the head on the lower, fleshy part of the lobe, being certain that the detector window was fully covered by the tissue and not exposed to external light sources. The probe was not positioned where cartilage was present, and care was taken not to press it against the side of the head. In general, signals from the ear are weaker (unless peripheral vasoconstriction is present, or hypotension limits finger perfusion), but the responses are much faster and at low saturations are said to be more accurate than from a finger probe[11].

With both the finger and ear probes, the signal validity was checked in order to determine correct positioning of the probe. Three complete passes of the plethysmographic waveform were identified (the plethysmographic waveform corresponds to the arterial pressure waveform). Noise spikes present on the waveform were indicative of poor probe placement, the detector not flush with the test site, or the thickness of the tissue sample is too great, and so the probe was re-sited.

The signal strength indicator bar was close to full scale (assuring a strong signal strength). If the signal strength was seen to be 50% or less a test site with a shorter distance between the emitter and the detector was found. The SpO_2 readings also had to be stable before they were accepted as reliable.

REFERENCES

1) Schapp LM. Pulse Oximetry - Uses and abuses (Review). Chest 1990;98(5): 1244-1250

2) Severinghaus JW, Astrupt BT. History of blood gas analysis. International anaesthesiology clinics 25, No 4, 1987. Boston: Little Brown and company

3) Severinghaus J, Niafeh K. Accuracy of response of six pulse oximeters to profound hypoxia. Anesthesiology 1987;67:551-58

4) Mendelson Y, Kent J, Saharian A, Welch G, Giasi R. Evaluation of the Datascope Accusat pulse oximeter in healthy adults. J Clin Monit 1988;4:59-63

5) Mackenzie N. Comparison of a pulse oximeter with an ear oximeter and an in-vitro oximeter. J Clin Monit 1985;1:156-160

6) BTS Guidelines for the management of Chronic Obstructive Pulmonary Disease. Thorax December 1997;52 Supp 5

7) Pitkin AD, Roberts CM, Wedzicha JA. Arterialised earlobe blood gas analysis: an underused technique. Thorax 1994; 49: 364-366

8) MacIntyre J, Norman JN, Smith G. Use of capillary blood in measurement of arterial PO2. BMJ 1968; 3:640-643

9) Spiro SG, Dowdswell IRG. Arterialised ear lobe blood samples from blood gas tensions. Br J Dis Chest 1976;60:263-268

10) Dawson A. How should we report the oxygen saturation measured on a CO-oximeter? Cal Thoracic Society ABG Newsletter 1989; June:6-7

11) Barker SJ, Tremper KK. Pulse oximetry: applications and limitations. Int Anesthesiol Clin 25: 155-75, 1987

"ON THE BLOWER" – Manufacturers News

1. Equipment Comparisons

The ARTP are continually asked for advice on which spirometer we would recommend for drug trials, GPs, practice nurses, research departments, etc. As the UK professional body for respiratory technology and physiology, we have a responsibility to inform all users of spirometers and other related equipment as to what are the acceptable standards and functions recommended for clinical spirometry. Furthermore, it is also our responsibility to either independently evaluate or else to report the results of independent scientifically sound evaluations of this equipment. We also feel it is important that we work with manufacturers to improve their equipment in terms of ease of use, accuracy and reliability.

Therefore, in the light of the demand for spirometry in general practice, we feel it is time that independent scientific data is made available to purchasers of spirometers. We know that lung function departments around the country regularly do comparisons of different spirometers. These trials may not always have an ideal scientific design, and may be open to operator bias. The ARTP Executive Committee has pledged to produce standard scientific protocols for members to follow when doing comparative trials on a range of spirometers. We would like the results of those trials (together with quality control and calibration data) to be registered centrally with ARTP so that we can build up a library of reports on equipment that can then be made available to potential purchasers. This all fulfills our basic remit which is to be the Association **for** Respiratory Technology and Physiology. We will keep you informed of any trial results. Any members wishing to be involved in multi-centre trials of equipment, please contact this column by post or fax so we can register your interest.

2. Trade Stand

Pharmaceuticals

I have received assurances from Boehringer, Astra-Zeneca, Glaxo-Wellcome and Baker-Norton that they will be supporting our winter meeting at Daventry in February (10th-12th). We are grateful for their support and I am sure they will receive a good response from the delegates who will include targeted specialist registrars.

Lung function equipment

I have still not received official verification of the arrangement formed between **Erich Jaeger** and ThermoNeutron the owners of **EME/SensorMedics** at the ATS meeting in San Diego *(St God?? ... bit full of themselves aren't they?).* I was led to believe that the arrangement would be a bit like Ford owns Jaguar, but you'd never see a Ford logo on an XK8!! The deal sounds like the kind of thing shareholders get ecstatic about but I feel will take a long time to make a difference to the market place. Any official comment beyond this speculation would be gratefully received.

All the three major lung function kit companies have little new equipment to show off although I am getting rumours of new software to be launched for the **Ferraris Collins** system in the Autumn. Also, **Jaeger** are planning to launch their latest software Version 4.5 in the late summer. It is being tested at a few Beta sites around Europe to iron out any bugs. We'll keep you posted.

I recently treated myself to a trial of the **Cosmed K4** portable full exercise system. We ran a couple of subjects on the K4 and a SensorMedics 2900 and got reasonable comparisons for all ventilation and metabolic parameters. Their telemetry option, the neat software and the user options available leave me singing its praises. Is it the replacement for my Year 2000 obsolete 2900?? Watch this space!

I am aware that **Morgan Medica**l are currently racing around the country installing MDAS 4.0 on Benchmark and Autolink systems as quickly as possible. Most installations have run fairly smoothly and I'd be grateful if users of the new program would let me have their comments on the changes. Morgan users who have not heard about the upgrade and have a service contract with the company need only phone Chatham and arrange a date for the upgrade. Remember, this new software will require at least a pentium PC. (N.B. It's worth trying to wangle a PC upgrade out of your Year 2000 budget!!).

Sleep study and associated equipment

Medic Aid (the sole agents for Respironics in the UK) have announced that they will be the UK agents for **Healthdyne** equipment and services which was formerly held by SLE. They state that this is part of a Respironics restructuring process and does not reflect on SLE.

ResMed have launched their AutoSet T (for therapy) which is an auto-titration system coming in at the £800 (excl VAT) mark. Also the Mirage fullface Mask is now available costing £115 to hospitals. I've not tried one yet, but if Gary Ward could loan me one for the weekend, I could tell you how good they were as a fashion accessory!! I believe **DeVilbiss** are having a re-vamp of their AutoAdjust titration system called the AutoAdjust LT. It uses a pneumotachograph to monitor patient airflow, and has a fancy Windows-like software log of performance, but I am unsure of any prices as yet, but I have heard rumours of it costing around £700.

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B&D Electromedical, the company who actually build all the NIPPY ventilators have sent me their price list for NIPPY spares. They are remarkably cheaper than the prices I have been quoted in the past!!! Peripherals such as a Battery Back-up inclusive of charger is only £670 (incl VAT). For a price list contact Peter Bachelor on Tel: 01789 293460. For technical/medical support a useful contact is Noel Davis on Tel: 01789 721577. We hope to see B&D at the Winter meeting in Daventry.

I also had a interesting demonstration of the Medivent RTX Respirator. This Intermittent Negative Pressure Ventilator operates via a cuirass. The sensation of having your breathing controlled for you like a vice around your chest is very weird. It is also weird sitting like a giant aphid in the middle of the department!! My advice is do not try the vibration mode after a full lunch or fizzy drinks!!!! Linked to a PC this machine monitors its delivery and will make a very useful option for those acute COPD patients who cannot tolerate a nasal mask. I am informed they may cost around £12K - £15K and will be available in the mid/late autumn.

Miscellaneous

We are grateful to all the companies who sponsored our successful winter meeting in Doncaster. Their commitment and participation added to the success and enjoyment of all participants. However, members should be aware that **InterMedical Ltd** pulled out of supporting the meeting at the very last minute and nearly caused the meeting to run at a loss. Would they pull out of an important delivery at the last minute? Would they fail to turn up to an appointment at your department? Consider these points when you plan to place an order for some ventilator equipment. Would **Mallinkrodt, Rusch** and other competitors let you down?

Moan of the month: Generally, have you seen the declarations in handbooks of equipment such as CPAP machines, nebulisers, and other equipment targeted for the United States which says that only a physician or healthcare professional can buy or issue the device? How many patients buy equipment through newspaper and magazine advertisements or directly from manufacturers when there is no objective evidence of assessment and benefit? Let's hope the "Stop the rip-off Britain" campaign covers this loop hole for our patients.

3. Complaints Database and WatchDog

Owners of **SensorMedics** VMax lung function testing kit should be aware of the fact that using small cylinders of calibration gases from the company not only proves to be more expensive but also is more likely to lead to leaks as the O-ring gets damaged more easily from regular cylinder changes. Contacting one of the reputable specialist medical gas companies for a quote (e.g. **BOC Special Gases** who attend our winter meetings regularly!) is always a good idea. I would be interested to know whether users of VMax systems have noticed any improvement in service in the last 4-6 months? Please write and let me know.

We have been working with **Jaeger** to sort out the problems we have been having with the transfer factor test. Compared to a Morgan system, the Jaeger MasterScreen system had lower TLCO, VA and KCO values. This was confirmed by customers in Nottingham, Dublin and Dorchester. Some of the fault was a problem with PC controlled balloon-valve opening, which effected VA, but has been solved with a software fix. The slightly lower gas transfer values (by only 10%) may be caused by different techniques and is being investigated further. Helium dilution lung volumes and spirometry values are generally comparable on the 2 systems. An upgrade for the MasterScreen software is due out by the end of the summer. I don't know when installation will be nationwide, but we will let you know.

I have heard mixed stories about the quality of service from **Morgan Medical.** The good news is that they are gradually installing the latest version of MDAS software (4.0), and upgrading numerous systems (Walsall, Derby, Sheffield, Nottingham, Burton, Leicester, to name but a few). However, these upgrades are still causing some problems in several centres (Nottingham, Burton, Leicester). Are we being unreasonable wanting the upgrades to be perfect? I believe not. The MDAS 4.0 software has been developing for at least 2 years when ARTP members views/suggestions were expressed at ARTP/Morgan Users Groups meetings. We expect to have many of the niggles ironed out, and we are paying for the privilege of the upgrade. I would appreciate letters from users saying what they think of the new software.

Finally, I am shortly to meet with representatives of the Medical Devices Agency to discuss whether they should turn their efforts to the lung function market. I will be taking along the Watchdog File of your letters and complaints and will discuss on your behalf whether formal trials should be undertaken by the MDA.

*When writing to the Complaints Database and WatchDog, please state **(i) exact dates, (ii) names of people you dealt with and (iii) state clearly your grievance.** Also, give a **summary account** of the history of your complaint (a maximum of one page of A4). There is no need to send photocopies of correspondance at this stage.*

Dr Brendan Cooper (ARTP Manufacturer's Liaison Officer)
Lung Function Department
Nottingham City Hospital
Nottingham NG5 1PB
DDI/FAX (24 hours):
0115 840 2615

The relationship between changes in FEV_1 and SG_{aw} following bronchodilator administration and its implication for assessing bronchodilator response in patients with airflow obstruction.

Carter R and Banham SW

Department of Respiratory Medicine, Glasgow Royal Infirmary University NHS Trust

KEY WORDS: SPECIFIC AIRWAYS CONDUCTANCE, BRONCHODILATOR RESPONSE, COPD

ABSTRACT

Background

There is a continuing debate concerning the measurement of FEV_1 as the most appropriate method of assessing a bronchodilator response due to the mechanical challenge of forced expiratory manoeuvres in patients with airflow limitation. We have therefore assessed changes in specific airways conductance (SG_{aw}), measured during a panting manoeuvre, to establish its relationship to changes in FEV_1 and the level of a significant change in patients who showed a significant bronchodilator response based on forced spirometry. To assess the sensitivity of this measurement, compared to FEV_1, we have applied the measurement of SG_{aw} in a group of patients who would be considered to have irreversible airways obstruction based on forced spirometry.

Methods

Retrospective analysis of the pulmonary function test results of 150 patients (mean age 54.2, range 17-78 years, 85 males) who showed an increase of >200 ml in the FEV_1 following bronchodilator administration (Responders). This was repeated in a subgroup of 70 patients (mean age 61.9, range 45-76 years; 40 males) who did not show a significant change in FEV_1 following bronchodilator administration (Non-Responders). All patients had performed forced spirometry and SG_{aw} measurements in a constant volume body plethysmograph (Sensormedics V6200, Yorba Linda, California, USA) which were repeated at 15 minutes post bronchodilator administration (Salbutamol MDI 3 x 100 mcg via Spacer).

Results

Mean baseline FEV_1 in the Responders was 1.48 (SD 0.54) litres; FEV_1 % predicted 51.9 (15.6); mean SG_{aw} was 0.44 (0.20) $s^{-1}.kPa^{-1}$. There was a significant relationship between FEV_1 % predicted and baseline SG_{aw} in the Responders ($r = 0.75$ $p<0.001$). Following bronchodilator administration the response as assessed by the absolute change in FEV_1 (litres) and the absolute change in SG_{aw} was significantly related ($r = 0.74$ $p<0.001$) as was absolute change in FEV_1 and percentage change in SG_{aw} ($r = 0.74$ $p<0.001$). The minimum change in SG_{aw} associated with a significant change in FEV_1 was 0.1 $s^{-1}.kPa^{-1}$ and 35%. In the Non-Responders [Mean FEV_1 0.96 (0.45); FEV_1 % predicted 39.9 (13.4), SG_{aw} 0.29 (0.26)] there was a similar relationship between baseline % predicted FEV_1 and SG_{aw} ($r = 0.69$ $p<0.001$). There was no relationship between the bronchodilator response as assessed by absolute change in FEV_1 and SG_{aw} or percentage change in FEV_1 and percentage change in SG_{aw}. However, 25 patients showed an increase in SG_{aw} of more than 35% and 0.1 (range 36-189 %, absolute change 0.1-0.84) in the absence of a significant change in FEV_1.

Conclusion

These results suggest that SG_{aw} may be a useful additional measurement assessing bronchodilator response in patients with COPD.

INTRODUCTION

The change in airway calibre that results from the administration of a bronchodilator drug is clearly of great importance in respiratory medicine. The most commonly used test to assess the response to a bronchodilator is the forced expiratory volume in the first second (FEV_1). For a change in FEV_1 to be correctly assessed, the natural variability of routine FEV_1 measurements has to be ascertained for the type of patient in question. A number of reports have appeared on this topic and there has been some debate on whether an absolute or percentage change should be used as the criteria for a significant bronchodilator response[1-4]. We have previously reported a retrospective analysis of the best three FEV_1 measurements performed in 327 patients selected for various levels of FEV_1 and with varying degrees of airways obstruction and in 41 healthy hospital volunteers[5]. The results were divided into groups according to the level of the FEV_1 to allow comparison of variability at different levels. The coefficient of variation decreased as the baseline FEV_1 increased but there was no change in the absolute variability, expressed as the standard deviation, or trend towards larger absolute variability as the baseline FEV_1 increased. This allowed the establishment of a single absolute value of 186 ml or, in practical term's 200 ml, which would be valid at any level of the FEV_1 to reflect significant change following bronchodilator administration.

It has more recently been reported that the performance of forced expiratory manoeuvres can cause bronchoconstriction during both a deep inspiration to total

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lung capacity and also by forced expiration to residual volume[6,7]. In addition, patients with more severe airways obstruction may not be able to tolerate the mechanical challenge of forced expiratory manoeuvres due to flow limitation mechanisms. This has led to the search for an alternative measurement of bronchodilator response in patients with airflow obstruction which does not rely on a forced manoeuvre to assess airways calibre. Previous studies have assessed the reversibility of airflow obstruction based on forced oscillation[8] or interrupter techniques of airways resistance measurement during normal tidal breathing at functional residual capacity[9] and plethysmographic airways resistance and conductance measured during a panting manoeuvre[10,11]. These studies have shown these techniques to be more sensitive to change than measurements based on forced manoeuvres but have not established a significant level of response in these parameters compared to the change in FEV_1.

In our own laboratory we routinely assess the response to a bronchodilator by measuring lung volumes and airways conductance in a constant volume plethysmograph as well as measuring indices of forced expiration. We have therefore performed a retrospective analysis of these indices in patients who would be considered responders (change in FEV_1 >200 ml) to assess the relationship between changes in FEV_1 and specific airways conductance (SG_{aw}) as a means of establishing a significant change in SG_{aw}. In addition we have assessed the changes in specific airways conductance in a group of patients who would be considered non-responders based on FEV_1 criteria to assess the sensitivity of specific airways conductance as a measurement of bronchodilator response in patients with COPD.

METHODS

Study Population

A retrospective analysis of pulmonary function results was performed in 150 patients (mean age 54.2 years, range 17-78, 85 males) who had attended the Department of Respiratory Medicine for the assessment of bronchodilator response from January 1997. All these patients had a >200 ml increase in FEV_1 following bronchodilator administration (Responders). The pulmonary function test results of a separate subgroup of 70 patients (mean age 61.9, range 45-76 years, 40 males) were also analyzed. These patients did not show a significant change in FEV_1 following bronchodilator administration (Non-Responders).

Pulmonary function tests

Dynamic spirometry, with the best FEV_1 of a minimum of three attempts reported as representative for an individual patient, was performed before and a minimum of 15 minutes after 3 x 100 micrograms of salbutamol delivered from an MDI through a spacer device. Reproducible values for spirometry were obtained according to ERS guidelines[12]. The level of agreement between the best two FEV_1 being a maximum of 100ml. Specific airways conductance was measured as the mean of a minimum of four flow/box pressure loops with centre line fitting (+/- 0.5 l /s) and thoracic gas volume loops (mouth pressure/box pressure) in a constant volume plethysmograph (Sensormedics V6200) prior to and following bronchodilator administration. All testing was performed according to the recommendations of European Respiratory Society (ERS)[12]. Quality control and procedures of lung function testing were performed according to formal guidelines published by ERS[12,13]. Specific airways conductance measurements were always performed after dynamic spirometry with resting period of 2-3 minutes. Predicted normal values of lung function tests were determined using the European Community for Steel and Coal equations[12,13]

Data presentation and analysis

Unless stated otherwise, values are expressed as mean +/- standard deviation (SD). Comparisons between results for Responders and Non-Responders were performed using the independent samples Student's *t*-test. The within-subject variability for baseline measurements was expressed as mean coefficient of variation (CoV). The relationship between variables was assessed by simple linear regression and correlation coefficient. A level of $p < 0.05$ was considered significant.

RESULTS

Table 1 shows the baseline pulmonary function data for Responders and Non-Responders. The Non-Responders showed significantly lower baseline FEV_1; % predicted FEV_1 and SG_{aw} reflecting the more pronounced level of airways obstruction in this group. The mean coefficient of variation for FEV_1 in the Responders (average of the individual CoV, obtained by dividing the within-subject standard deviation by mean of the baseline values) was 7.9% and in the Non-Responders 10.3%. For SG_{aw} these values were 14.7% and 17.0%, respectively.

TABLE 1

	Responders (N=150)	Non-Responders (N=70)	P-Value
FEV_1 (litres)	1.48 (0.54)	0.96 (0.45)	p<0.01
FEV_1 % predicted	51.9 (15.6)	39.9 (13.4)	p<0.01
Mean CoV	7.9%	10.3%	
SG_{aw} $s^{-1}.kPa^{-1}$	0.44 (0.20)	0.29 (0.26)	p<0.01
Mean CoV	14.7%	17.0%	

Baseline pulmonary function test results and within-subject mean coefficient of variation (CoV) in Responders and Non-Responders.

Values are Mean (SD).

SG_{aw}: Specific airways conductance.

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Table 2 shows the absolute and relative changes in FEV_1 and SG_{aw} following a bronchodilator in Responders and Non-Responders. There is a greater change in the relative conductance measurements than volume measurements in both Responders and Non-Responders.

Figure 1 is a plot of simple linear regression between FEV_1 % predicted and baseline SG_{aw} $s^{-1}.kPa^{-1}$ in Responders showing a significant relationship between these two variables ($r = 0.75$ $p<0.001$). Following bronchodilator administration the response, as assessed by the absolute change in FEV_1 and absolute change in SG_{aw}, was significantly related ($r = 0.74$ $p<0.001$) as was absolute change in FEV_1 and percentage change in SG_{aw} (Figure 2: $r = 0.74$ $p<0.001$). The minimum change in SG_{aw} associated with a significant change in FEV_1 of 200 ml or more was 0.1 $s^{-1}.kPa^{-1}$ and 35%.

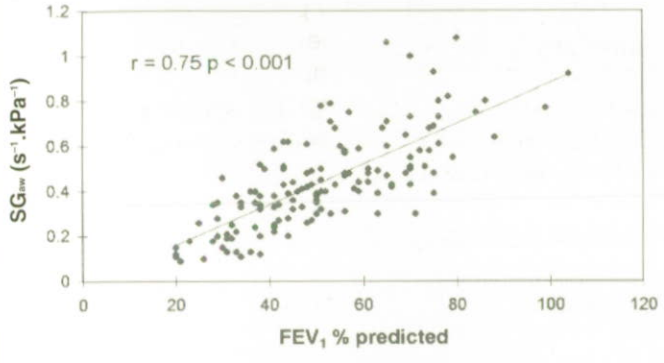
TABLE 2

	Responders		Non-Responders	
	Absolute	***% Change***	***Absolute***	***% Change***
FEV_1 (Litres)	0.39 (0.20-1.12)	26 (9-65)	0.11 (0-0.19)	10 (0-27)
SG_{aw} $s^{-1}.kPa^{-1}$	0.51 (0.10-2.10)	105 (35-262)	0.19 (0-0.84)	39 (0-159)

Mean values (Range) of change in pulmonary function tests following bronchodilator administration in Responders and Non-Responders.

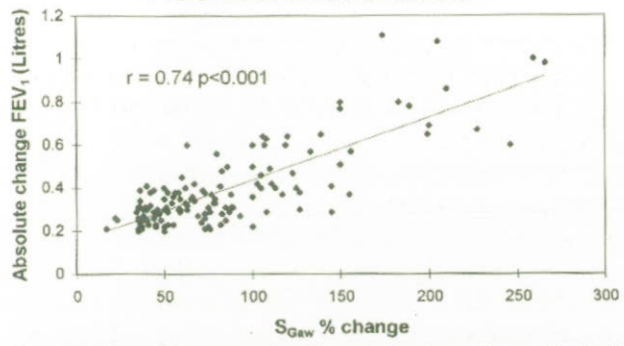
In the Non-Responders there was a similar significant relationship between baseline % predicted FEV_1 and SG_{aw} (Figure 3: $r = 0.69$ $p<0.001$). In this group of patients there was no relationship between the bronchodilator response as assessed by percentage changes in FEV_1 and SG_{aw} or absolute change in FEV_1 and percentage change in SG_{aw}. However, 25 patients showed an increase in SG_{aw} of more than 35% and 0.1 $s^{-1}.kPa^{-1}$ (range 36-229%, absolute change 0.1-0.84) in the absence of a significant change in FEV_1 .

FIGURE 1: RESPONDERS



A scatterplot with regression line of baseline SG_{aw} ($s^{-1}.kPa^{-1}$) against baseline % predicted FEV_1 in Responders (n = 150).

FIGURE 2: RESPONDERS



A scatterplot with regression line of absolute change in FEV_1 (litres) against relative change in SG_{aw} (%) following bronchodilator administration in Responders (n = 150).

Table 3 shows changes in Functional Residual Capacity by body plethysmography (FRC_{PL}) in the Non-Responders separated into those who showed a significant change in SG_{aw} of >35% and 0.1 $s^{-1}.kPa^{-1}$(n=25) and those who did not show a significant change in SG_{aw}. In the first group there is a significant fall in FRC_{PL} following bronchodilator administration [Mean change FRC_{PL} -11.5% (5.9) $p<0.001$; Mean Change SG_{aw} 74.8% (30.2)]. In the second group (N=45) there was no significant change in FRC pre and post bronchodilator [Mean Change FRC_{PL} -2.0% (4.0); Mean Change SG_{aw} 13.5% (10.7)].

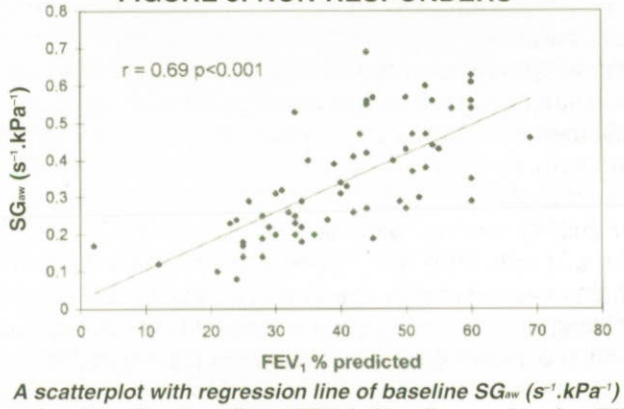
TABLE 3: NON-RESPONDERS

	FRC_{PL} Pre (Litres)	FRC_{PL} Post (Litres)	% change
SG_{aw} % change >35 N=25	5.05 (0.98)	4.46 (0.87)	-11.5 (5.9)
SG_{aw} % change <35 N=45	4.89 (1.41)	4.76 (1.34)	-2.0 (4.0)

Mean values (SD) and percentage change following bronchodilator administration of Functional Residual Capacity measured by body plethysmography (FRC_{PL}) in Non-Reponders separated into significant (% change SG_{aw} >35%) and non-significant (<35%) reponses.

Figure 4 shows the relationship between the percentage change in SG_{aw} and FRC_{PL} in those patients who showed a change in SG_{aw} >35%. This shows a significant relationship between a fall in FRC_{PL} and rise in SG_{aw} in these patients [correlation coefficient of -0.67 ($p<0.001$)]. There is no relationship between these variables in the group of Non-Responder patients who showed a change in SG_{aw} of <35% [correlation coefficient 0.13 (NS)].

FIGURE 3: NON-RESPONDERS



A scatterplot with regression line of baseline SG_{aw} ($s^{-1}.kPa^{-1}$) against baseline %predicted FEV_1 in Non-Responders (n = 70).

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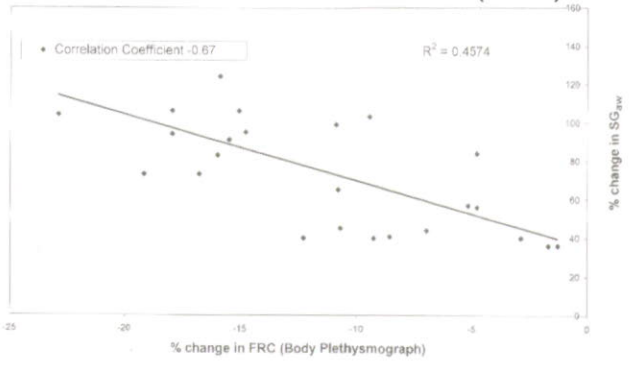
FIGURE 4: NON-RESPONDERS (N=25) WITH SIGNIFICANT CHANGE IN SGAW (>35%)



A scatterplot with regression line of relative change in SG_{aw} against relative change in FRC in patients who showed a >35% change in SG_{aw} in the Non-Repsonders (n=25).

DISCUSSION

The use of forced expiratory manoeuvres to assess the bronchodilator response is an established method with an increase of 200 ml and 15% being considered significant. The applicability of forced manoeuvres to assess bronchodilator response, however, may be complicated by their ability to alter bronchomotor tone causing bronchoconstriction not only by the deep inspiration to total lung capacity but also by forced expiration to residual volume[6,7]. For these reason other methods of assessing airway calibre which do not rely on a forced expiratory manoeuvre have been used to assess bronchodilator response[8-10].

In this study we have shown a significant relationship between baseline FEV_1 and SG_{aw} in a group of patients with airway obstruction. Following bronchodilator administration there was a significant relationship between absolute change and relative change in SG_{aw} and absolute change in FEV_1 in the patients who showed a significant increase in FEV_1 based on previously set criteria. This suggests that SG_{aw} is useful in assessing changes in airway calibre with a sensitivity that is at least as good as standard methods in this group of responders. In a previous study comparing forced oscillation, forced expiration and plethysmographic measurements of airways resistance, Van Noord et al[11] showed that the most sensitive indices for detecting a change following a bronchodilator in 125 asthmatic patients were plethysmographic measurements of resistance and conductance, followed by forced vital capacity , oscillometry and then FEV_1. In a study by Bridge et al[9] the measurement of airways resistance by the interrupter technique was shown to be correlated with changes in FEV_1 following bronchodilator administration in 25 asthmatic schoolchildren. However, the interrupter technique was found to be more sensitive in assessing a significant change following a bronchodilator (22 of 25 patients) compared to FEV_1 (16 of 25). Based on this evidence the methods of assessing the bronchodilator response in asthmatics, which do not rely on a forced expiration, are more sensitive than the FEV_1.

If forced expiratory manoeuvres have some limitations in asthmatics they may be of even less value in patients with COPD where patients whose FEV_1 fails to improve by 200 ml or 15% are labelled as irreversible. However, many patients with COPD who fail to show a significant change in FEV_1 on treatment report clinical improvement and with an increase in exercise tolerance. In the present study we have established a significant change in SG_{aw} which is compatible with a significant change in FEV_1 in a group of responders. In the group of 70 patients considered to have irreversible airways obstruction based on forced expiratory manoeuvres, 25 of these patients showed a change in SG_{aw} similar that that in the responder group (absolute change of 0.1 $s^{-1}.kPa^{-1}$ and 35%) in the absence of a significant change in FEV_1. This 35% threshold in SG_{aw} is very similar to the 34.9% threshold response for significance at the 95% probability based on the coefficient of variation of repeat measurements of SG_{aw} found in the study of Van Noord et al[11]. Consistent with data from the literature, volume measurements demonstrated better reproducibility, based on the coefficient of variation, compared to measurements of specific airways conductance. However, relative change after salbutamol was much greater for conductance measurements than for FEV_1 supporting its greater sensitivity in identifying reversibility in these patients.

Specific airways conductance contains an element of airways resistance and an element of thoracic gas volume and can therefore assess changes in both the calibre of the airways and changes in air trapping following bronchodilator administration. One reason for a change in SG_{aw} in the absence of a change in FEV_1 is suggested by Chrystyn et al[14]. With an increasing degree of airway obstruction there is the parallel development of hyperinflation in COPD which helps to maintain the patency of the airways by increasing the elastic forces tending to open the airways but at the expense of mechanical advantage in the thorax leading to dyspnoea. As treatment relieves obstruction there is less need to remain at the higher lung volume to maintain airway patency and tidal respiration can return to a more comfortable level. Because the overall sum of forces on the airways has not altered the FEV_1 may change little in these patients due to expiratory flow limitation mechanisms which may remain unaffected. Chrystin et al showed that in patients with COPD following theophylline treatment there were no significant changes in FEV_1 (12.5%) but that there was a 64% fall in thoracic gas volume as measured in a body plethysmograph suggesting significantly lower levels of air trapping. These patients also showed a 20% improvement in walking distance in the absence of a significant change in FEV_1. The authors showed that the fall in thoracic gas volume was associated with a rise in the relaxed vital capacity (RVC) which changed much more than FVC. This suggests that since RVC is much more easily measured than TGV this measurement should probably used more widely in patients with COPD when assessing bronchodilator responses.

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The finding of a reduction in thoracic gas volume following bronchodilator administration measured by plethysmography in patients with expiratory flow limitation is supported by the recent studies of Pellegrino and Brusasco[15,16]. The present study has also confirmed the effect of a significant change in FRCPL in the absence of a significant change in FEV_1 in patients with more severe airflow obstruction. There may also be a change in airway calibre as assessed by airway resistance measurements in the absence of a change in FEV_1. In a study by Khan et al[17] of 27 patients with COPD in which a bronchodilator was given there was no significant change in FEV_1 (10.2%), however, this group showed a significant fall in airways resistance (30%) measured by oscilloresistometry. The measurement of FEV_1 may, therefore, not be the most appropriate measurement of bronchodilator response in patients with COPD as they may be unable to tolerate the mechanical challenge of repeated forced expiratory manoeuvres due to flow limitation mechanisms. The present study therefore supports the findings of previous studies in suggesting that measurements of airway calibre and thoracic gas volume based on normal tidal breathing or panting manoeuvres may be more sensitive indicators of a bronchodilator response. The findings of this study suggest that the measurement of SG_{aw}, with its component measurements of thoracic gas volume and airway resistance, may be a useful additional measurement assessing bronchodilator response in patients with COPD.

REFERENCES

1) Hruby T, Buller J.Variability of routine pulmonary function tests. Thorax 1975; 30:548-553.

2) Nickerson BG, Lemen RJ, Gerdes CB, Wegmann MJ, Robertson G.Within subject variability and percent change for significance of spirometry in normal subjects and patients with cystic fibrosis. Am Rev Respir Dis 1980; 122:859-866.

3) Goldman AL.Influence of tracing selection technique on daily variation of FEV_1 and FVC. Am Rev Respir Dis 1979; 119:218.

4) Tweedale PM, Merchant S, Leshe M, Alexander I, McHardy GJR.Short term variability in FEV1: relation to pretest activity, level of FEV_1, and smoking habits. Thorax 1984; 39:928-932.

5) Carter R. Variability in a set of FEV_1 measurements in relation to bronchodilator response. Breath 1987; 32:6-9.

6) Suzuki S, Miyashita A, Matsumoto Y, Okubo T. Brochoconstiction induced by spirometric manoeuvres in patients with bronchial asthma. Ann Allergy 1990; 65:315-320.

7) Brusasco V, Pellegrino R, Rodarte JR. Vital capacities in acute and chronic airways obstruction: dependence on flow and volume histories. Eur Respir J 1997; 10:1316-1320.

8) Mazurek HK, Marchal F, Derelle J, Hatahet R, Moneret-Vautrin D, Monin P. Specificity and sensitivity of respiratory impedance in assessing reversibility of airway obstruction in children. Chest 1995; 107:996-1002.

9) Bridge PD, Lee H, Silverman M. A portable device based on the interrupter technique to measure bronchodilator response in schoolchildren. Eur Respir J 1996; 9:1368-1373.

10) Smith HR, Irvin CG, Cherniak RM. The utility of spirometry in the diagnosis of reversible airways obstruction. Chest 1992; 10:1577-1581.

11) Van Noord JA, Smeets J, Clement J, Van de Woestijne KP, Demedts M. Assessment of reversibility of airflow obstruction. Am J Respir Crit Care Med 1994; 150:551-554.

12) Report Working Party-Standardisation of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society. Eur Respir J Suppl 1993; 16.

13) Quanjer PH, Tammeling GJ, Cotes JE, Pederson OF, Peslin R, Yernault JC. Lung Volumes and forced ventilatory flows: Official statement of the European Respiratory Society. Eur Respir J 1993; 6:5-40.

14) Chrystyn H, Mulley BA, Peake MD. Dose response relation to oral theophylline in severe chronic obstructive airways disease. BMJ 1988; 297:1506-1510.

15) Pellegrino R, Brusasco V. Lung hyperinflation and flow limitation in chronic airways obstruction. Eur Respir J 1997; 10:543-549.

16) Pellegrino R, Brusasco V. On the causes of lung hyperinflation during bronchoconstriction. Eur Respir J 1997; 10:468-475.

17) Khan MA, Zaidi SR. Bronchodilator response in pulmonary disease at two different states of respiratory mechanics. Respiration 1996; 63;288-291.

MEASUREMENT MATTERS (1)

Sue Revill

Dept of Respiratory Medicine, Glenfield Hospital, Leicester

The value of any measurement depends on the degree of confidence that can be placed on the result. Calibration is a process of establishing how the response of a measurement process varies with respect to the substance being measured. The usual way to perform calibration is to subject known amounts of the substance, for example using a reference material, to the measurement process and monitor the response. When any equipment is positioned and used in a chosen environment, that environment will immediately begin to act on the equipment causing a change that will ultimately result in an unacceptable level of degradation in performance. This degradation is called drift, and causes unreliable results that are no longer fit for purpose. While drift cannot be eliminated it can be detected and contained through the process of calibration.

The credibility of test results depends on accuracy, precision (repeatability) and reproducibility. In turn, these depend on the competence of the tester and the validity of the methods used. Calibration using standards or reference materials is an important factor in obtaining accurate results that are consistent with measurements made in other laboratories. Calibration is at the centre of the quality assurance systems that should be in place for any clinical measurement department.

DEFINITIONS:

Accuracy:
how well the measurement reflects the true or correct value.

Bias:
the extent to which a measurement will differ more or less constantly and in the same direction from the true value.

Precision (or repeatability):
the variation of results obtained by the method when the same sample is run repeatedly. Accuracy and precision are independent of one another.

Random errors:
errors that occur without prediction or regularity, they tend to decrease precision.

Systematic errors:
errors within the test system or methodology, they will tend to produce bias.

Test sensitivity:
Number of disease **and** test positives divided by the true number of disease positives i.e. the proportion of the disease positives who are also test positive.

Test specificity:
Number of disease **and** test negatives divided by the true number of disease negatives i.e. the proportion who are disease negative and also test negative.

Accuracy is usually tested by comparison with reference techniques that are known to be accurate i.e. 'gold standards'. Since a very precise measurement is one that has nearly the same value each time that it is measured, it's magnitude can be evaluated from the standard deviation of repeated measurements on the same biological sample or repeated measurements within a subject. The smaller the SD the greater the precision of the measurement. A rule of thumb for a single operator performing a technique or biological measurement is that the coefficient of variation (i.e.SD/mean x 100) is < 5%. A simple exercise to assess CoV for a measurement technique would involve serial measurements on a single biological sample e.g. 10 serial blood gas determinations on a single sample of blood i.e. obtain 10 ml of arterial blood, make serial measurements of PaO_2, $PaCO_2$ and pH. The measurements should be made one after another, immediately the blood gas machine is ready to analyse the next sample. After each sample is drawn-up by the machine, the air should be expelled from the syringe to minimise contamination and then placed on ice to reduce metabolic effects. Calculate the mean, SD and CoV for each variable.

Precision, or repeatability, may also be assessed from the SD of the difference between pairs of measurements e.g. 20 healthy and practiced subjects each make two peak flow manoeuvres using the same peak flow meter. The difference between the two measurements is calculated for each subject, then the mean and SD of the differences is calculated. In order to assess the variability in the mean value you have calculated for

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the sample group, the 95% confidence intervals should be calculated from the standard error of the mean difference (SE = SD / square root of n, where n= number of observations e.g. in this example n=20). The 95%CI is calculated by multiplying the SE by the 't-value'* for n-1 degrees of freedom (in this case 19). For arguments sake let us say the mean difference is 2 l/min and the 95%CI is 10 litres/min, we can now be sure that for 95 times out of 100 the 'true population mean' of the difference between repeated measurements will lie between 2 ± 10 l/min (i.e. +12 to -8 l/min). How we can visualise the magnitude of the individual differences between repeat measurements and graphical methods for quality control purposes will be discussed in the next issue of INSPIRE.

*t tables may be found in any standard statistics book.

For a more detailed description of measurement theory the following references are strongly recommended.

An introduction to medical statistics. Martin Bland 1995. Oxford Medical Publications (ISBN 0-19-262428-8). (Chapter 15, Clinical Measurement).

Statistics with confidence. MJ Gardner & DG Altman 1989. The Universities Press (Belfast) Ltd. ISBN 0-7279-0222-9.

Ranges, confidence intervals, and related quantities: what they are and when to use them. S Chinn Thorax 1991; 46: 391-393.

Repeatability and method comparison. S. Chinn Thorax 1991; 46: 454-456.

URGENT !!! TO ALL ARTP MEMBERS

As promised in the last edition of INSPIRE, **ResMed (UK) Ltd** have kindly offered a £300 bursary to fund an ARTP member to attend the Winter Meeting of the BTS in London, 13th - 15th December 1999.

To apply for the bursary you should send a letter and brief essay (no more than two sides of A4) which addresses the following questions:

'What are the difficulties in obtaining good patient compliance with CPAP machines, and what are your suggestions to improve this?'

The winner will be selected jointly by the ARTP Committee and **ResMed (UK) Ltd** and will be expected to write a short report on two research presentations (specified by ResMed) from the winter meeting. The report should be at least 500 words in length.

Applications (covering letter and essay) should be sent before ***19th November 1999,*** to
Gill Butcher (Bursary Secretary)
Cardio-Respiratory Unit, Queens Hospital,
Belvedere Road, Burton-on-Trent DE13 ORB

Additionally, bursaries are available from the ARTP Education Fund to support ARTP members to attend the **ARTP Winter Meeting (**10th - 12th February 2000**).** In order to qualify you should write a report for INSPIRE on ONE of the following topics: the implications of clinical governance for the respiratory function laboratory; evidence based practice in the respiratory function laboratory; risk assessment in the respiratory function laboratory; clinical audit in the respiratory function laboratory. Additionally you will be expected to write a short report for INSPIRE critically appraising one session at the ARTP meeting.

Deadline for submission for the ARTP bursary is 20th December 1999. Applications should be sent to Gill Butcher.

MEMBERSHIP UPDATE

Report from Steve Scholey

At the time of going to press we have 426 members of the ARTP. However, of these only 265 have renewed their membership this year, with 161 outstanding fees. The deadline for renewal was July 31st. Renewals after the 1st August incur a late registration fee of £5. A reminder letter and application form will be posted to those who have failed to renew. Please note that renewing membership through your hospital ordering system can take up to 3 months therefore we would be grateful if you could take this into account next year.

At the risk of overstating the state registration line, all practitioners of respiratory function will have to show membership of the appropriate controlling body for the profession i.e. the ARTP. So if you know of people who are not members please encourage them to contact me. It is not all take - there is a lot to gain from ARTP membership, the cost of membership can easily be recouped by attending a couple of meetings a year at the concessionary rates

FEES – FULL: £25 (+£5 for late renewal) • STUDENT: £15 • CORPORATE: £45

The fees for departmental membership will be calculated on a sliding scale depending on the number of members in the department and each member will receive a copy of INSPIRE.

Enquiries to: **Steve Scholey, ARTP Membership Secretary, Chest Unit, General Hospital, Pontefract, West Yorkshire, WF8 1PL. FAX: 01977 606401**

RECENT ARTICLES

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

NITRIC OXIDE

Sampling of exhaled nitric oxide in children: end-expiratory plateau, balloon and tidal breathing methods compared.

Q Jobsis, SL Schellekens, A Kroesbergen, WCJ Hop, JC de Jongste. Eur Respir J 1999; 13:1406-1410.

The method of choice for measuring exhaled nitric oxide is during controlled slow exhalation. This study compared the accepted technique with two techniques which were simpler and easier for children - blowing air into a balloon and tidal breathing through a mouthpiece. All the children in the study were allergic asthmatics but in a stable condition. All the children performed the two simpler manoeuvres without difficulty whilst 29 children from the group of 101 children were unable to perform the slow exhalation method. There was no significant difference between the NO concentration using the balloon method and the accepted slow exhalation method. However the tidal breath method yielded much higher NO concentrations suggesting the possibility of nasal air contamination. The authors suggest the balloon method might be considered an alternative to the slow exhalation and has the potential to offer sampling that is remote to the NO analyser i.e. for field trials.

LUNG FUNCTION and POLLUTION

Ozone-induced lung function decrements do not correlate with early airway inflammatory or antioxidant responses.

A Blonberg, IS Medway, C Nordenhall, H Hedenstrom, FJ Kelly, AJ Frew, ST Holgate, T Sandstrom. Eur Respir J 1999; 13: 1418-1428.

Ozone is a common summertime pollutant which frequently exceeds recommended safety levels throughout Europe. This study sought to clarify early events in the lung occurring after ozone challenge. Thirteen healthy subjects undertook intermittent rest and exercise over a two hour period whilst breathing filtered air contaminated with 0.2 ppm ozone. Lung function was assessed pre and post-exposure. Additionally bronchoscopies were performed 1.5 hours after exposure. Spirometry showed significant reductions in FEV1 and FVC and evidence of small airway narrowing with reduced expiratory flows. Analysis of the BAL and the biopsy material revealed a range of inflammatory markers were elevated. There was no relationship between the changes in the inflammatory markers and the lung function measurements. The authors suggest the lack of a relationship indicates that lung function measurements are not good indicators of subsequent ozone induced lung injury.

MEASUREMENT OF AIRWAY FUNCTION

What are the minimal important changes for asthma measures in a clinical trial?

Santanello NC, Zhang J, Seidenberg, Reiss TF, Barber BL. Eur Respir J 1999; 14: 23-27.

This research group has attempted to relate how patients feel with measurements of airway function in order to find the minimally significant clinical change in FEV1 and PEF. The study was a randomised placebo-controlled trial of a leukotriene agonist in 281 asthmatic patients. Patients rated their symptoms from a 7 point scale ranging from 0 to 6, and using the descriptors of very much worse to very much better. The average minimal patient perceivable improvement in symptom scores was -0.31 from the baseline score, +0.23 l for FEV1, +18.79 l/min for PEF, and -0.81 puffs/day for beta-2 inhalers. The authors state that these changes cannot be applied universally since there may be differences between other treatment groups and other types of patient populations. However the results are a pointer to assess meaningful changes reported in other clinical trials.

The effect of patient technique and training on the accuracy of self-reported peak expiratory flow.

Gannon PFG, Belcher J, Pantin C, Burge PS. Eur Respir J 1999, 14: 28-31.

The aim of this study was to investigate the difference between encouraged self-recorded PEF with unobserved readings and to investigate any long-term changes in PEF self recording. In this arduous study 41 patients were asked to record PEF every 2 hours until their next clinic visit. A subgroup

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of patients were examined at a third clinic visit. Significant differences between the unobserved and encouraged PEF were detected. The mean difference was 21 l/min, however the limits of agreement were as great as 60 l/min. The authors recommended that PEF technique should be re-evaluated at each clinic visit, and that re-training should might be necessary.

Repetitive hyperpnoea causes peripheral airway obstruction and eosinophilia.

Davis MS, Freed AN. Eur Respir J 1999; 14: 57-62.

The background to this study is the increased incidence of asthma in winter sports athletes. These athletes routinely experience hyperpnoea during training in very cold conditions. The hypothesis for the study was that repeated episodes of hyperpnoea with dry air may predispose individuals to the development of asthma. A canine modal was used to test the hypothesis with repeated dry air challenge over a period of two weeks. Cells and soluble mediators were examined from the bronchial lavage solutions. Compared to the control airways the airways receiving the dry air challenge had marked eosinophilic inflammation, increased prostaglandin's and leukotrienes. The authors concluded that repeated dry air challenge in dogs causes persistent airway obstruction and inflammation not unlike that found in humans, with the data supporting the hypothesis that repeated cold air hyperpnoea contributes to the development of asthma.

Forced oscillation total respiratory resistance and spontaneous breathing lung resistance in COPD patients.

Farre R et al. Eur Respir J 1999; 14: 172-178.

Forced oscillation resistance measurements may be unreliable in airways obstruction. This study examines a method which acts to minimise the errors in COPD. The results demonstrated the modified method was more reliable than previous methods in severe airways obstruction.

A comparison of a new transtelephonic portable spirometer with a laboratory spirometer.

Izbicki G. Et al Eur Respir J 1999; 14: 209-213.

This new transtelephonic portable spirometer records the slow and forced expiratory vital capacity tests. Data can be transmitted via the telephone to a remote receiving centre where a volume-time and a flow-volume curve are displayed on a screen in real time. The "spirophone" was compared to a laboratory spirometer which conformed to ATS standards in 30 patients and 15 healthy subjects. The new device was found to be comparable to the laboratory spirometer for slow VC, FVC, FEV_1 and PEF.

BLOOD GAS MEASUREMENT

Accuracy of the i-stat bedside blood gas monitor.

Sediame S et al. Eur Respir J 1999; 14 : 214-217.

The i-stat is a portable blood gas machine. This study compared its performance to the bench top ABL520 Radiometer. Ninety-two samples were analysed in duplicate covering a wide range of blood gas and pH values. There was good agreement between the two machines. However the portable machine remained in the same laboratory for the duration of the study, therefore the reliability of the machine in the context in which it would be used i.e. at the bedside and with regular movement remains to be examined.

CALENDAR OF FORTHCOMING EVENTS

13th - 15th September
Conquering airway inflammation in the 21st century
National Heart and Lung Institute, London
Course organiser D Rogers
Tel: 0171 351 8172

13th - 17th September 1999
Advanced Respiratory Physiology
Coventry University
Topics:- Bronchial challenge and skin testing, Gas transfer and measurement of lung volumes, respiratory muscle measurement and flow-volume loops, invasive and non-invasive blood gas measurement. Respiratory and cell physiology.
Course FEE - **£150** for week (or **£30 /day**)
10% reduction for ARTP members
Quote ARTP membership number on application form.
Contact Anna Kovalchuk (Biology Office) for application form on 01203 631313

6th October
Allergy in Laboratory Workers
National Heart and Lung Institute
Course organiser S Gordon
Tel: 0171351 8172

9th - 13th October 1999
European Respiratory Society
Madrid, Spain
Information ERS HQ, Lausanne
Tel: 00 41 21 617 2865

1st - 2nd November
Respiratory Medicine 1999
The Royal Marsden Conference Centre London
For course programme and application form contact Sarah Powell on 0181 678 5322

18th - 19th November
Occupational and environmental lung diseases
National Heart and Lung Institute
Course organiser AJ Newman Taylor
Tel 0171 351 8172

10th - 12th February 2000
ARTP Winter Meeting
Daventry, West Midlands

COMPETENCY IN DRUG ADMINISTRATION

In 1995 The Royal Devon & Exeter Hospital NHS Trust reviewed its policy on drug administration within the hospital and produced a new/revised document on who should give drugs, how it should be done and what documentation is required to be completed. When this document was brought out it was assumed that only doctors and nurses administered drugs. It was oblivious of the work technicians in many disciplines do that involves drug administration, mainly for assessment/evaluation purposes. Our department decided to meet this challenge. Therefore we devised our own policy and protocol, along the lines of the general hospital policy but with a specific interest in the drugs used in the department.

Every staff member who administers drugs, from the lowly MDI to putting lignocaine up some poor patients' nose, has to be competent to give the drug and be able to cope with any unexpected side effects. To ensure this happens we all complete an in-house study day and have a theoretical and practical assessment annually. The study day is very comprehensive led by the Sister of our department and with speakers from the respiratory clinic and pharmacy.

The respiratory nurse specialist explains about the different aspects of assessing and delivering bronchodilators, the different techniques available to us and the best way to deliver the dose required. Although this sounds like information we as technicians should know already, it is good to have it reinforced and also given from a slightly different perspective. If the reversibility tests indicate a patient requires bronchodilators the work of the nurse to train patients with the inhaler equipment is easier if we all use the same technique.

The Chief Pharmacist introduces the Medicine Act 1968 and talks on the typical drugs used in the laboratory, what the likely side effects are and the pathways in the body these drugs use. He highlights the need for using the correct generic names, the necessary documentation and the precautions to be taken when under 16s come for testing. In addition it ensures that we are kept updated of any new device or drug preparation.

The Sister gives guidance on the information that needs to be checked before the patient is given any drug (i.e. name, DOB, doctors signature, drug and dosage required); how we prepare for the test by instructing the patient and what to document in the notes and on the results such as time the drug is given, dosage and signature. There is also advice if an emergency happens and the procedure for reporting incidents.

Having completed the study day, we are given a multi-choice questionnaire and a practical assessment, when the Sister watches us perform a reversibility test with a patient. If we pass all this we are deemed competent to administer the drug and we can relax for another year! Supported by the chest consultants, every procedure has been substantiated by standing orders, protocols, references and general information for each drug used.

This is a basic outline of the procedure followed in our Department and how it has evolved. As a new member I had not experienced this kind of assessment in other Departments I had worked in which makes me wonder if a Departmental drug policy is a rarity. As we progress towards a state registered profession we will be required to standardise our procedures of drug administration to patients to ensure legal protection or should we rely on our own local protocols? It also raises the question of other substances we may use within the pulmonary laboratory such as histamine or allergens used in skin testing. May be we will eventually need patients to sign consent forms before we proceed with any tests.

I hope that this Department is not unique in this type of assessment and it makes me wonder how other laboratories approach this problem or even if they perceive it as a problem?

Ed: There are some interesting issues raised in this article. The policy sounds comprehensive and an example of 'good clinical governance' to ensure safety of the patient and knowledgeable practice from the technician.