Dr. Michael Berthon-Jones has been closely involved in the field of respiratory medicine and the development of CPAP devices for most of his working career.

After completing his medical degree at Sydney University, Dr Berthon-Jones spent two years working with Dr Russel Vandenberg at Royal North Shore Hospital, in the area of respiratory medicine.

He was working on a PhD thesis studying the control of breathing in sleep, both in normal subjects and in obstructive sleep apnea, when his supervisor, Professor Colin Sullivan, first proposed using nasal CPAP to treat obstructive sleep apnea. Dr Berthon-Jones worked on the first take-home CPAP devices and the first take-home customized CPAP masks.

Later he moved on to carry out research on animal models of sudden infant death syndrome, then spent several years teaching respiratory physiology, before moving into the development of software for respiratory function laboratories and sleep laboratories.

In 1990, he joined the newly established ResMed company where he is currently Chief Scientific Officer.

Here Dr Berthon-Jones speaks about the work behind the development of CPAP devices.

What inspired the concept of an “intelligent” automatic clinical CPAP device?

It was Professor Colin Sullivan who came up with the original concept of an intelligent CPAP device. He was working with patients who were unable to get to sleep on high levels of CPAP. He felt the problem could be resolved by a device that would keep the pressure low until the patient was asleep, and then quickly put the pressure up until he was well treated.

That is where the idea of a CPAP machine that would increase pressure if there was snoring, was born. I was asked to help study how well it worked at Colin’s sleep laboratory at Royal Prince Alfred Hospital and to suggest improvements.

Later we realised that an intelligent CPAP device has perhaps three uses. Firstly it can keep the pressure low while you are waiting to go to sleep. Secondly, it can find the right pressure for you without having to wait months, sometimes many months, for a night in a sleep laboratory where your pressure can be titrated. Thirdly, by only giving you high pressure when you need it, you get less pressure-related side effects, such as mouth leak and blocked nose. Some people only need high pressures when they are on their back, or dreaming, or if they have a stuffy nose or a cold coming on.

What sort of problems did you encounter in developing it?

The biggest problem was getting a comfortably sealing mask. In those days we used to make custom individualized masks, at great expense. And we’d glue the mask on with a silicone adhesive. This was a very messy, time consuming and expensive business, but it produced a superb seal.
If you are running a busy sleep laboratory, you’re probably faced with a growing pile of medical journals, all containing numerous ‘must read’ research articles. Here are some suggestions that can help you make the most of precious reading time by identifying a good quality article.

1. Journals and Authors

Journals that carry important scientific research are always peer reviewed, that is scrutinized by independent experts in the field.

Authors should usually be experts on the subject being researched. While they often supervise post-graduate students whose names also appear as authors, experts in a field will not put their name to a piece of research that does not stand up to critical review. It is also important to ask whether an author has a particular interest in a subject that may influence how the results are interpreted and presented and note whether any other interest, such as funding, is involved.

2. Abstract

A well-presented article begins with an abstract that gives an overview of the study. There should be a brief discussion of methodology, including sample size followed by the results and conclusion.

3. Literature Review

This should give a thorough and objective overview of the issues involved and demonstrate the purpose of the research being reported. The literature should be reviewed in an objective and critical fashion, including discussion of literature that does not support the author’s proposals. Ideally, the literature reviewed should not be more than ten years old but substantive works in the field should be included.

4. Methodology

Having established the questions to be asked by the research, the report should then explain how these questions were answered. In the field of sleep medicine the methodology used is usually quantitative, where data is collected in such a way as to be measurable and subject to statistical analysis. Another way of collecting data is through qualitative methods, where the emphasis is placed on the type of data being collected. This provides less information about sampling but more on the context of the study and how the data was collected and analysed.

5. Sample size

The methodology should also include an explanation of the sample and population, ie all subjects who are eligible for inclusion in the study. It is a sample of this population that is taken to test the research question.

The sample size needs to be a true representation of the population under study and big enough to allow the findings to be generalized across the population. The authors should explain how the sample size was calculated. A number of statistical tests can be done to calculate sample size. The power calculation simply demonstrates the ability of a research design and sample size to detect a relationship between variables. Basically, the nearer to 1.0 the power calculation is, the better chance the sample size has of providing a significant result. Most studies aim for a power of about 0.80. In studies that do not include a power calculation there may have been a precedent for the size of the sample set in previous similar studies.

6. Study Design

In quantitative studies the design is usually either experimental or non-experimental. An experimental study takes one or more variables, known as the independent variables, and manipulates them in order to see the effect on another, dependent variable. Commonly subjects are randomized into two groups. One group receives an intervention while the control group does not.

In non-experimental quantitative studies the researcher is looking for relationships among already occurring phenomena. There is no attempt made to intervene. Therefore, a sleep researcher might be interested in whether a relationship exists between an independent variable such as the subject’s BMI and a dependent variable such as CPAP pressure required to eliminate snore. The researcher is not interested in manipulating the BMI to change the CPAP pressure but is simply looking for a relationship between the two.
The explanation of the study should be simple and easy to follow. There should be no point where you are left thinking ‘Now why did they do that?’ Finally the report should discuss any ethical issues that might be involved and whether approval was sought from the appropriate bodies.

7. Results

In medical journals the results are usually of a statistical nature and can be divided into two groups. All will have descriptive data such as mean age and height, gender and BMI. The other data will be inferential and will attempt to find significant relationships between the independent and dependent variables. The researcher should give a significance expressed as p value, which is an indication of how strong the relationship is. The more zeros after the decimal point, the stronger the relationship is and the less likelihood that this result occurred due to chance.

Graphs and tables illustrating results should be easy to interpret.

8. Discussion

The discussion of the findings and the conclusion will refer to the original research question and include the significance of the findings and what new questions the study raises. Other observations should also be discussed and could include information such as the qualitative data collected. Finally, the researcher should not be afraid to criticize the study design and suggest ways in which it could be improved.

9. Conclusion

Finally, the conclusion should refer back to the original research question and include flaws in the research design and further questions raised by the study. There may have been details omitted since many of the sections will have been curtailed to fit in with a specified word limit. It is important therefore that, as readers, we approach journal articles with a degree of subjectiveness. The report’s purpose is to add to an existing body of research and will inevitably lead to further questions being raised.

From Fenella Connell RN, Scotland
Use of ResMed CPAP equipment on airlines

1. Can a CPAP flow generator be used on a commercial airline flight?
   Not always. Some airlines are helpful with patients wanting to use their CPAP devices on airliners and others are not.

2. What airlines allow the use of flow generators on their aircraft? Why do some allow them and some don’t?
   Few airlines have a formal policy regarding the use of CPAP devices on aircraft. Airlines including United and American have proven helpful and most tend to deal with requests from customers on a case by case basis. Qantas and Air New Zealand are active in assisting patients who wish to use a flow generator on their aircraft. British Airways’ policy is that they do not allow such devices on board at all.
   The best advice is to check with the airline directly.

3. What do your patients need to know when they enquire about a flight and booking a seat?
   You should first find out from the airline concerned if they generally allow passengers to use CPAP equipment on their aircraft. They should also obtain the following:
   1. A letter from their doctor stating the need for CPAP treatment.
   2. A US socket adaptor to operate the flow generator on the aircraft supply of 110 volts AC. ResMed’s flow generators use switch mode power supplies that can handle any voltage ranging from 110 – 240 VAC; however a US style pin is required because outlets on aircraft use the US style sockets. Other devices may not have switch mode power supply so in these cases you may need to contact the manufacturer for advice.
   They will also have to arrange to book a seat close to a power outlet on the aircraft. The number and availability varies from one plane to the next and from one airline to the next. They may also have to explain to the flight crew that your flow generator will work on the aircraft’s power supply of 110 volts 400Hz despite the rating plate on the flow generator indicating 50-60Hz. The most important thing is to be prepared and make their arrangements well in advance. If required, copies of approval letters are available from ResMed Technical Services (techs@resmed.com.au)

4. Is a transformer needed to operate the ResMed flow generator in another country?
   No. All ResMed flow generators will operate from any mains voltage anywhere in the world. All you will need is an adapter to fit the mains socket.

What led you to think about developing an automatic CPAP device for home use?

That was really the goal from the outset. However, it was much easier to come up with a device that worked in the hospital only, because we didn’t have to worry about cost, or how big it was, or how ugly it looked. We could concentrate on making it work superbly. We ended up with AutoSet Clinical, of which I am still very fond. It was too big and expensive to go home with a patient, but it did a very good job at automatically determining how much pressure the patient needed. They could then go home on a conventional CPAP machine set at that pressure. Over time, we came up with AutoSet T, the take home device, which is much smaller and simpler for a patient to use.

What led you to incorporate the idea of flow limitation into the algorithm, years ahead of its time?

The initial device (based on responding to snoring alone) could only respond to reasonably loud snoring. This is because it had to ‘hear’ the snoring through the roar of the motor and the mask. That sort of approach could get the pressure reasonably close reasonably quickly, but it turned out that it was generally insufficient. We now know why. If the pressure is just enough to eliminate snoring, the airway will still narrow to the point where it is very difficult to breathe. This is called silent inspiratory airflow limitation.

While I was doing my PhD, I noticed that normal subjects, while they were asleep, showed a characteristic flattening of the inspiratory flow-time curve, which went away the instant they awoke from sleep. I noticed that if you put obstructive sleep apnea patients on CPAP, and then lowered the pressure a bit so that it was enough to prevent snoring, they would also show this flattening of the inspiratory flow-time curve. Indeed, if you put them on just adequate CPAP and pushed them a bit harder by giving them some carbon dioxide to breathe, this would also produce this characteristic flow limitation, so that even though they tried harder and harder to breathe, they didn’t get any more air for their efforts.

So I thought here was the way to ‘fine tune’ an automatic CPAP machine: increase the pressure if you see apneas, or if you hear snoring, but increase it that last little bit if you see this characteristic flattening of the flow time curve.
Why is it important for an automatic CPAP device to respond to flow limitation, snore and apnea?

The characteristic flattening of the flow-time curve caused by flow limitation is the very best signal for fine-tuning the pressure, once you have eliminated apneas and snoring. But if you are just falling asleep, you can go very quickly from having a totally open airway to snoring very loudly, in a way that produces somewhat chaotic or messy flow-time curves, without seeing the characteristic flattening. So the best approach is to respond very quickly to loud snoring, and then fine tune using flattening. Rarely, you can go straight from awake and unobstructed to asleep and apneic, and so it can be useful to increase pressure in response to apnea as well. However, actual apnea is pretty rare on AutoSet, because in most cases the responses to snoring and flattening get the pressure up quickly enough to prevent apneas.

Why doesn't ResMed's AutoSet respond to hypopnoea?

When you are lying quietly awake, or when you first go to sleep, or when you are dreaming, you can have hypopneas (reductions in the depth of breathing) which are nothing to do with the state of the airway. For example if you sigh, which you do every few minutes, you usually have a hypopnea immediately afterwards. This can also happen if you have just rolled over and are getting settled, or if you are dreaming. And the annoying thing is that when you are on CPAP, this tendency to have what are called central hypopneas - hypopneas that are nothing to do with the state of the airway - is increased. If you make an automatic CPAP device that responds to hypopneas, you will put the pressure up to the maximum while the patient is awake.

Do you think there is a misconception clinically that all hypopneas should be treated?

For simple obstructive sleep apnea, central hypopneas should not be treated. They are not a disease. Everyone has them. And they don’t go away with CPAP.

There is a rare and important exception: central hypopneas due to heart disease. This is called Cheyne-Stokes breathing. CPAP does help with that.

Why doesn’t ResMed’s AutoSet respond to apnea above 10 cmH2O in pressure?

I mentioned before that the higher the pressure, the more central hypopneas you will have. At a pressure somewhere around 10 cmH2O, the central hypopneas become central apneas. On the other hand, the vast majority of obstructive apneas are already well controlled by 10 cmH2O, and we are only fine tuning using snoring and flattening. So it is a pretty good bet that if the pressure is already above 10 cmH2O, any apneas are most likely central, and you should leave them alone (except in patients with central apneas due to heart failure). But if the pressure is below 10 cmH2O, most apneas will be obstructive and you should put the pressure up. There’s nothing magical about 10 cmH2O, it’s just a good place to put the line in the sand.

Can you over-treat apnea?

You can’t over-treat obstructive apnea. You really don’t want the patient having unresolved obstructive apneas. And we want not just to prevent apnea - we also want to keep the airway sufficiently open for the subject to breathe easily and regularly and stay asleep.

But you can use too much pressure. The higher the pressure, the greater the side effects. Although this has never been proven, it is rather obvious - no pressure, no side effects! So you want to use the lowest pressure possible while keeping the airway nicely open.

Likewise can a device that responds to hypopnea over-treat it?

The funny thing is that it can both over-treat and under-treat. It will put the pressure up through the roof in some subjects, who have lots of central hypopneas. And it can completely miss repetitive severe silent inspiratory flow limitation that is totally disturbing the patient’s sleep without there being any hypopneas. If this occurs without CPAP, it is called upper airway resistance syndrome. It is just as bad for you as obstructive sleep apnea. But a CPAP machine that responds only to hypopneas will treat your obstructive sleep apnea, and give you upper airway resistance syndrome instead.

How can Automatic CPAP devices help optimise treatment?

CPAP devices, whether automatic or not, can tell us - the clinician, the technician - about what is going on when we are not there. Is the patient using the device? Is there a leak, and if so, when and how much? If it is an automatic device, what is the pressure doing? How well is the patient breathing? How steadily, how much? This might be particularly important if the patient also has heart disease or lung disease, or has had a stroke, and has other reasons, apart from sleep apnea, for having abnormal breathing during sleep.
Flow Limitation

Q. How important is flow limitation for the diagnosis of OSA?
A. In patients with mild disease, measurement of flow limitation is crucial for an appropriate diagnosis. Using an accurate method to measure the shape or morphology of the flow-time curve will allow partial upper airway closure to be detected. Although EEG arousals - without a known cause - may help to identify the syndrome, some flow limitation resulting in increased negative intrathoracic pressure swings may not lead to arousals.1,2

Q. Isn't it necessary to measure esophageal pressure to detect inspiratory flow limitation?
A. No. While some consider esophageal pressure the “gold standard” for detection of inspiratory flow limitation, this invasive technique is generally not necessary. The non-invasively determined contour of the flow-time curve indicates flow limitation and has a very good correlation to the esophageal pressure. 2,3

Q. Can thermistors / thermocouples measure flow limitation?
A. No, thermistors or thermocouples cannot detect the full range of flow related respiratory disorders. This limitation results from the relatively long time-constant of this technology (it takes too long to measure the minute temperature changes that occur during the respiratory cycle). Thermistors do not allow analysis of the shape of the inspiratory flow-time curve, therefore they will underestimate subtle changes in nasal flow and will not detect flow limitation. In addition, ambient room temperature can also impact on the heat detection of thermistors.

Q. How does a nasal cannula and pressure transducer measure flow limitation and why are they more accurate than thermistors?
A. The capability of nasal prongs to detect flow is based on the sensing of pressure changes associated with turbulence at the nostrils. This combination functions as a reasonably accurate type of flow sensor. The excellent time response of nasal prongs with a pressure transducer allows precise detection of each component of, or events within, the breathing cycle by providing a very high quality signal. Snoring and silent partial airflow limitation can be accurately detected, recorded and analyzed.

Q. How do ResMed flow generators detect flow limitation?
A. By monitoring the shape of the inspiratory flow-time curve. If the upper airway is patent, the shape of the inspiratory flow-time curve is rounded. As the upper airway begins to obstruct, the mid portion of the inspiratory flow-time curve begins to flatten. This has been measured and compared with esophageal pressures in published studies 2,3

References
ADEP is a very large home care company in France, responsible for 7018 home care patients. Of these, 3580 are on CPAP. Fifty-five percent of these patients are treated with Autotitrating CPAP devices, with 70-80% using humidification.

When did you begin using AutoSet T?

In July 1998 ADEP organized an internal meeting with the intention of learning more about the various CPAP devices on the market and what compliance data they offered. We invited various manufacturers to present their equipment, one of which was ResMed. Jerome Otter from ResMed France presented the AutoSet T and the efficacy data provided by the device. The AutoSet T clearly met our needs for monitoring patients on CPAP. We were also very intrigued by the AutoSet T algorithm and felt that responding to flow limitation was very important. We had reviewed the literature and it was clear that flow limitation was important for titration and we felt it was equally important for treatment of OSA.

How are you using AutoSet T?

Some of our prescribers use the AutoSet T for CPAP titration. After several days of use, we download the data and our prescribers use the 95th centile pressure for the fixed CPAP prescription. Because many of their patients preferred the AutoSet T to fixed CPAP for treatment, some physicians prescribe AutoSet T directly without the titration step. Today, nearly half of our 3500 CPAP patients use AutoCPAP for treatment.

How do you see the future for Automatic CPAP devices?

We think there will be more and more patients directly placed on Automatic CPAP for treatment after diagnosis without a titration step. The price of Automatic CPAP devices are decreasing and with the time we save, the overall savings are very interesting.

How do you use the efficacy data provided by the AutoSet T and do you find it useful?

We find the information provided by the AutoSet T very useful, especially the leak information. All our homecare nurses are equipped with portable computers. They download the efficacy data during their home visits. We ask our prescribing physicians what information they want to receive for their patient and then we create a specific report form for them. We also send some basic information, such as usage data and 95th centile pressure, to all our physicians.

How has accessing this information changed clinical practice?

I’m not sure. There are probably more full face masks used now than in the past. Our nurses do not have the right to make this change themselves, but with the information we send, it sensitizes the prescriber to what changes are needed.
Fatigue is a contributing factor in up to 50% of heavy vehicle accidents in Australia, at an estimated cost of 300 million dollars [1] [2]. Major factors causing fatigue or excessive sleepiness include inadequate sleep, circadian rhythm (body clock) effects and sleep disorders. Sleep-disordered breathing or SDB is the most common sleep disorder. It affects 24% of men in the general population with 4% of all men having significant associated fatigue [3]. In this disorder, recurrent brief obstruction to the upper airway during the night causes the sufferer to stop breathing, reduces blood oxygen levels and disrupts sleep. In moderate to severe cases these events occur from 20 to as many as 100 times per hour during sleep. This results in daytime sleepiness, impaired vigilance and slow reaction times [4]. SDB sufferers have a threefold increased risk of motor vehicle accidents and are also seven times more likely to have had multiple accidents [5] [6]. They are also at higher risk of stroke, heart attacks and impotence. Treatment of SDB with CPAP (continuous positive airway pressure) reduces the accident risk of sufferers back towards normal. Treatment with CPAP involves wearing a small mask over the nose at night. The mask is connected to a pump that delivers a continuous pressure of air to hold the throat open.

Studies in America and Canada have suggested that SDB is more common in truck drivers than the general population, with rates of up to 78% of drivers [7]. This has led to an Australian study to assess the size of the problem in Australian drivers.

**CONFERENCE UPDATE**

In November last year, Dusseldorf hosted the annual NeuroREHA conference, which followed on from the MEDICA conference.

The conference is organised by the German Society of Neurological Rehabilitation (DGNR), where Dr. Alfred Thilmann, Rhein-Ruhr-Klinik Essen, is a member of the ‘widened’ board.

This small but important event attracts international and local neurologists and physiotherapists.

This year the scientific program included papers on the topic ‘Sleep and Neurological Rehabilitation’, with the sessions chaired by Dr. Alfred Thilmann and Dr. Helmut Teschler, both from Essen.

**Papers included:**

**SBD: Basics, prevalence and clinical relevance** from Claudio Bassetti, Zurich;

**Treatment of SDB in neurological diseases** from Helmut Teschler, Essen;

**Sleep apnea: effects of apnea and hypopnea on thrombocyte function, vascular regulation, intracranial pressure and cerebral hemodynamics** from Ludger Grote, Göteborg;

**Interaction between sleep and immune system** from Thomas Pollmächer, Munich;

**Classification & Diagnosis of SDB** from Volker Töpfer, Essen;

**Treatment concepts in sleep apnea** from Thomas E. Wessendorf, Essen; and

**Neuromuscular Diseases** from Uwe Mellies, Essen.

**Australian Sleep-Disordered Breathing Study**

A large study of SDB in Australian road transport drivers is nearing completion. It involved overnight sleep studies of 300 drivers and a survey of 6,000 drivers. Fifty-five percent of drivers had at least mild SDB compared to twenty-six percent of adult males in the general Australian population [8] [9]. Seven percent of drivers had severe SDB, with more than 40 episodes per hour when their breathing stopped and their sleep was disrupted. These drivers had a much higher level of chronic sleepiness or fatigue than other drivers. Treatment
A common and preventable cause of fatigue and accidents in Australian drivers

for these drivers is clearly indicated, and would reduce their accident risk. Overall fifteen percent of the drivers involved in the project have had follow-up for fatigue and/or sleep disorders, with two-thirds of these proceeding to treatment.

There are several reasons for SDB being more common among professional drivers. Most drivers are male, and men have a higher rate of SDB. Our study showed that the drivers are more likely to be overweight (41%) or obese (39%) and this is another risk factor. Finally inadequate sleep is a common problem amongst drivers and can make SDB worse. Forty percent of drivers averaged less than six hours of sleep per day while working.

Diagnosis of SDB requires an overnight study to monitor sleep and breathing. This Australian study shows that a simple questionnaire can be used to identify drivers at high risk of having SDB who would benefit from further assessment and treatment if required. This would result in reduced accidents, injuries and absenteeism due to fatigue. Cost benefit analysis shows significant potential cost savings from screening and treatment of SDB in professional drivers.

Bibliography


Acknowledgements:

Authors: Dr Mark Howard and Professor Robert Pierce
Fatigue, Sleep-Disordered Breathing and Accidents

Institute for Breathing and Sleep, Austin and Repatriation Medical Centre, Melbourne
Royal Prince Alfred Hospital, Sydney
Princess Alexandra Hospital, Brisbane
Transport Workers Union of Australia
Clinicians frequently have questions about the use of humidifiers in conjunction with, or as part of, CPAP machines.

One of the most common concerns raised is that heated humidifiers may become contaminated and place patients at risk of respiratory infections.

However according to Dr Glenn Richards, despite long term use of humidifiers in both intubated patients and with CPAP machines, there is no evidence that patients develop excess numbers of respiratory infections or infections due to unusual organisms.

The first is what he describes as a misconception that hot water humidifiers act as a culture medium so that any pathogens reaching the humidifier chamber encounter an environment that supports rapid growth and multiplication. In fact the environment in the humidifier chamber under most operating conditions is such that the majority of pathogens are rapidly killed. He cites research carried out by Goularte et al, that found that four species of Gram -ve pathogens and S. aureus were rapidly killed by the conditions found in hot water humidifiers at temperatures above 44°C (111.2°F).\(^1\)

While bacteria were not killed at 37°C (98.6°F) they did not increase in numbers. While bacteria can survive in water at normal body temperature, it does not contain the nutrients required for multiplication.

Secondly, if they are to cause disease, pathogens must be transported from the humidifier chamber to the patient. Modern humidifiers produce molecular humidity only and molecules of water are too small to transport bacteria or other contaminants. Pathogens reaching the chamber are trapped there.

Finally, unlike intubated patients, those on nasal CPAP have their normal respiratory defences intact. Dr Richards says, ‘Any pathogens carried in the inspiratory airflow are subject to the same defences as during normal breathing. Heated humidifiers do not encourage bacterial growth and do not provide a method of transport for bacteria, so normal physical defences are sufficient to prevent respiratory infections.’ Hetzel in 1996 concluded that there was no increase in infections of the upper or lower respiratory system associated with humidification therapy.\(^2\)

Further to this, an article published by Sanner et al in 2001 suggested that proper cleaning of the humidifiers is also important in preventing infection.\(^3\)

Another common question concerns nasal symptoms such as congestion, dry nose and throat, and sore throat, which affect approximately 40% of patients using CPAP, and may be so severe as to reduce compliance with treatment.

Mouth leak is the likely mechanism by which CPAP causes or increases nasal symptoms. Many patients with OSA are chronic mouth breathers and on CPAP they are prone to the development of mouth leaks, which lead to unidirectional nasal airflow at high rates. Normally the nasal mucosa heats and humidifies inspired air but this can be overwhelmed at high airflow rates and particularly under conditions of unidirectional flow.

At low temperatures, the nasal mucosa dries out, provoking nasal airway resistance (NAR), symptoms of nasal congestion and nasal discharge. This reaction can be prevented by humidification of the inspired air to above 92% relative humidity. This has been reinforced by numerous published articles indicating that heated humidification does reduce symptoms and improve compliance.\(^4\&5\)

References
Recent Research Articles


Dr JR Stradling and colleagues from the Oxford Centre for Respiratory Medicine have critically reviewed the evidence on OSA and hypertension, to conclude that 'it now seems beyond all reasonable doubt that OSA is an independent risk factor for diurnal hypertension, and that this has more than trivial consequences at a public health level'.

2. Inspiratory flow limitation during sleep in pre-eclampsia: comparison with normal pregnant and nonpregnant women. Connolly G; Razak A R; Hayanga A; Russell A; McKenna P; McNicholas W T Depts of Obstetrics and Gynaecology, Rotunda Hospital, Dublin, Ireland. European respiratory journal Oct 2001, 18 (4) p672-6

Self-reported snoring is common in pregnancy, particularly in females with pre-eclampsia. The prevalence of inspiratory flow limitation during sleep in preeclamptic females was objectively assessed and compared with normal pregnant and nonpregnant females. Fifteen females with pre-eclampsia were compared to 15 females from each of the three trimesters of pregnancy, as well as to 15 matched nonpregnant control females (total study population, 75 subjects). All subjects had overnight monitoring of respiration, oxygen saturation, and blood pressure (BP). No group had evidence of a clinically significant sleep apnoea syndrome, but patients with pre-eclampsia spent substantially more time (31+/−8.4% of sleep period time, mean+/−SD) with evidence of inspiratory flow limitation compared to 5.5+/−2.3% in third trimester subjects and <5% in the other three groups (p=0.001). In the majority of preeclamptics, the pattern of flow limitation was of prolonged episodes lasting several minutes without associated oxygen desaturation. As expected, systolic and diastolic BPs were significantly higher in the pre-eclamptic group (p<0.001), but all groups showed a significant fall (p< or =0.05) in BP during sleep. Inspiratory flow limitation is common during sleep in patients with pre-eclampsia, which may have implications for the pathophysiology and treatment of this disorder.

3. Comparison of the effects of sleep deprivation, alcohol and obstructive sleep apnoea (OSA) on simulated steering performance. Hack MA; Choi SJ; Vijayapalan P; Davies RJ; Stradling JR Oxford Centre for Respiratory Medicine, Oxford Radcliffe Trust, UK. Respiratory medicine (England) Jul 2001, 95 (7) p594-601

Patients with obstructive sleep apnoea (OSA) are reported to have an increased risk of road traffic accidents. This study examines the nature of the impairment during simulated steering in patients with OSA, compared to normal subjects following either sleep deprivation or alcohol ingestion. Twenty-six patients with OSA and 12 normal subjects, either deprived of one night’s sleep or following alcohol ingestion [mean (SD) alcohol blood level 71.6 mg dl(-1) (19.6)], performed a simulated steering task for a total of 90 min. Performance was measured using the tendency to wander (SD), deterioration across the task, number of ‘off-road’ events and the reaction time to peripheral events. Control data for OSA, sleep deprivation and alcohol were obtained following treatment with nasal continuous positive airway pressure (nCPAP), after a normal night of sleep, and following no alcohol, respectively. Patients with untreated OSA, and sleep-deprived or alcohol-intoxicated normal subjects performed significantly less well, compared to their respective controls (P<0.01 for all tests), with untreated OSA lying between that of alcohol intoxication and sleep deprivation. Alcohol impaired steering error equally throughout the whole drive, whilst sleep deprivation caused progressive deterioration through the drive, but not initially. Untreated OSA was more like sleep deprivation than alcohol, although there was a wide spread of data. This suggests that the driving impairment in patients with OSA is more compatible with sleep deprivation or fragmentation as the cause, rather than abnormal cognitive or motor skills.

Watch out for future issues of the ResMedica Clinical Newsletter where we will be highlighting the theme of hypertension and stroke.

A web site of interest: Sleep Home Pages, hosted by the University of California: http://bisleep.medsch.ucla.edu/
2002 calendar of events

April 22-23 Las Vegas, NV, USA Medtrade Spring
May 5-9 Sydney, Australia 14th World Congress of Cardiology
May 7-10 Brisbane, Australia 10th Annual Scientific Meeting of the Australasian Faculty of Rehabilitation Medicine
May 9-11 Sydney, Australia International Cardiac Rehabilitation Meeting
May 17-22 Atlanta, GA, USA ATS 2002, 98th International Conference (American Thoracic Society)
May 28-31 Brighton, UK 13th European Congress of Physical Rehabilitation Medicine
June 29 - June 1 Geneva, Switzerland 11th European Stroke Congress
June 3-7 Reykjavik, Iceland 16th Congress of the European Sleep Research Society
June 10-12 Seattle, WA, USA APSS 16th Annual Meeting (Associated Professional Sleep Societies)
August 14-17 Mexico City, Mexico North American Stroke Meeting (National Stroke Association)
September 14-18 Stockholm, Sweden 12th ERS (European Respiratory Society) Annual Congress
September 10-12 South Stradbroke Is., Queensland, Australia 4th Milano International Symposium on Sleep Stroke Society of Australasia Annual Scientific Meeting
October 5-8 Tampa, FL, USA AARC (American Association for Respiratory Care)
October 29-31 Atlanta, GA, USA Medtrade 2002
November 5-7 San Diego, CA, USA CHEST 2002 (American College of Chest Physicians)
November 17-20 Chicago, IL, USA AHA Scientific Sessions 2002 (American Heart Association)

2003 forward planner

June Chicago, IL, USA APSS 17th Annual Meeting (Associated Professional Sleep Societies)
June 30 - July 3 Helsinki, Finland 7th World Congress in Sleep Apnea
November 21-23 Zuhai, China 4th ASRS Congress (Asian Sleep Research Society)
October 7 - 9 Atlanta, GA, USA Medtrade 2003

AN IMPORTANT NOTE TO YOU THE READER

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