A brief history of OSA

Is 30 years a long time or a short time in medical science? Have we come a long way, or is our understanding of OSA still in its infancy? Whichever way you look at it, Sullivan et al’s 1981 paper brought a revolutionary treatment to light.

OSA has been observed since ancient times, and there are records of its symptoms, such as heavy snoring, dating back over 2,000 years. The term ‘Pickwickian syndrome’ was adopted in the late 19th century to describe apneic symptoms, but research concentrated on the patients’ obesity rather than on disordered breathing during sleep. In 1965 the first polysomnograph recorded apneas during sleep. Further research in the 1960s established that obesity was not essential for OSA, but that there were other comorbidities associated with sleep-disordered breathing.1

In 1970 the first sleep clinic was established at Stanford University, California, USA by William Dement. In 1972 Christian Guilleminault joined the clinic, concentrating on respiratory disorders during sleep. The period 1975–80 saw an intense amount of research into sleep and apnea, with 319 articles appearing in medical literature. In 1978 JE Remmers et al resolved the key question of where airway obstruction occurred during an apnea, showing that the locus of airway closure lay in the oropharynx, not the larynx.2

In 1979 Sullivan returned to Sydney and devised a mask that would fit over a dog’s snout to deliver air or an experimental gas.2

In Toronto, Canada, Eliot Phillipson started investigating respiratory control in dogs in 1970. He was joined in 1976 by Colin Sullivan on a post-doctoral research fellowship from Sydney University, Australia. Sullivan worked with the dogs in the laboratory, studying effects such as hypoxia, hypercapnia, control mechanisms during REM and non-REM sleep, arousal and laryngeal stimulation. In 1979 Sullivan returned to Sydney and devised a mask that would fit over a dog’s snout to deliver air or an experimental gas.

Until this time the experiments on the dogs had been conducted via a tracheotomy. Similarly, people who were severely affected by OSA were given tracheotomies to bypass the blockage in the upper airway and allow unimpeded breathing. However, this was a dramatic solution for the problem, and could in itself have serious consequences for the patient. Sullivan was moving towards an alternative non-invasive treatment for OSA.

He applied his experimentation on dogs to humans, using masks that were created for each patient. A plaster cast would be made of their nose and fibreglass moulded over the cast. The resulting fibreglass shape would be fitted with air inlets and outlets and attached to the patient’s face each night with silicone adhesive. Tubes to the therapy device were attached to the mask, providing the patient with a source of continuous positive airway pressure that could be regulated.

Sullivan used his observations of five patients using this technology for his 1981 paper. He describes the patients as having long histories of noisy snoring and excessive daytime sleepiness to the point where their lives were seriously affected. Two had lost their jobs as a result of falling asleep at work, and one, a 13-year-old boy, was unable to stay awake at school and had consequently been categorised as ‘mentally retarded’. Sullivan notes that three of the five had been offered, but refused a tracheotomy. He conducted three all-night sleep studies on each patient, using CPAP on the third night. He writes: ‘Continuous positive airway pressure completely prevented the upper airway occlusion in each of the five patients. The upper airway occlusion could be turned off and on simply by increasing or reducing the level of positive airway pressure.’

Sullivan had shown that the occurrence of obstructive sleep apnoea could be reversed by the application of CPAP to provide ‘a pneumatic splint for the nasopharyngeal airway’. Acceptance of this treatment did not come immediately, and application of his findings to a wide audience was even slower.

From the Editor

It is 30 years since Colin Sullivan et al’s groundbreaking paper, ‘Reversal of obstructive sleep apnoea by continuous airway pressure applied through the nares’ was published in The Lancet.1 To mark its anniversary, in this edition of ResMedica we have interviewed one of the sleep technicians from Dr Sullivan’s first sleep unit; a long-time practitioner in sleep-disordered breathing; a long-time user of continuous positive airway pressure (CPAP); and one of the people deciding the future direction of CPAP. These interviews take us from the source of Dr Sullivan’s paper to the present day and beyond.

The global significance of Professor Colin Sullivan’s work was acknowledged when he was profiled in The Lancet in April this year.2

We thank everyone interviewed for this edition of ResMedica for generously giving us their time and sharing their thoughts and passion about the treatment of obstructive sleep apnea (OSA).

Once again we extend an invitation to you to contribute your thoughts to ResMedica by sending an email to theeditor@resmed.com.au

We look forward to your comments.

Sleep well,
Alison Hansford,
Global Editor

A BRIEF HISTORY OF OSA
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Twelve years later the first major epidemiologic study of the prevalence of OSA was published by Young et al, finding OSA to be present in 2% of middle-aged women and 4% of middle-aged men. Seven years after that, in 2000, four separate papers were published that demonstrated associations between OSA and hypertension (see Key research articles p14-15). This was a turning point in sleep apnea studies. They were the first full studies involving sleep apnea effects that were well-designed with a large patient base, which found a significant OSA health effect. That the four separate papers were all published within the same year gave a lot of impact to the result.

From then on, treatment of OSA was not just about relieving daytime sleepiness—there was the probability that there was a mortality benefit as well. In 2001 further research showed an increased prevalence of coronary heart disease, heart failure, and stroke at levels of an apnea-hypopnea index equal to or greater than five per hour.3

The connections between OSA and a range of comorbidities continue to grow. The medical consequences of OSA are currently considered to arise from a complex interaction between five factors: disturbed sleep, intrathoracic pressure swings, intermittent hypoxia, sympathetic activation and the mechanical consequences of snoring. Figure 1 shows the symptoms resulting from these five factors.


Figure 1: Medical Consequences of Obstructive Sleep Apnea

[Diagram showing medical consequences of obstructive sleep apnea]
Bron Lehrhaft completed her nursing training in 1967 and followed up with a cardiothoracic diploma in 1968 at Royal Prince Alfred Hospital (RPAH), Sydney. In 1984 she started working with Dr Colin Sullivan in his sleep unit at RPAH and has worked with patients on continuous positive airway pressure (CPAP) therapy ever since. She has worked in education for sleep technologists and is now a Nurse Consultant, Senior Technologist at a private clinic. She pioneered follow-up techniques after sleep studies to improve compliance rates.

How did you start working with OSA patients?

I’d left Australia after my training and worked with my husband in Germany and England for many years. We returned to Sydney in 1984. I was looking at the ads in the paper for nurses and one was at RPAH—it was the most interesting ad, obviously written by Dr Sullivan, and one of the requirements was a cardiothoracic background. You also had to have had experience in ICU because the patients that we were seeing were almost knocking at death’s door. I’ve had some really interesting work as a nurse, and I’ve always loved what I did and given 100%. But with this job I just found my niche. It was just the most incredible experience working with Dr Sullivan and his little unit.

Can you describe the work at the sleep unit?

We were the only unit in Australia that was doing sleep studies. In the first year of CPAP, 1981–82, there were six patients on CPAP. In 1982–83 there were a further 14, then in 1983–84 in the first year of CPAP, 1981–82, there were six patients on CPAP. They didn’t do very well—they hated it because they didn’t have the bed for him at the time, so he had a chart on heat sensitive paper that shows the obstructions as the oxygen level goes down and back up again. SaO₂, oxygen saturation, is an indirect way of reading what the PaO₂, the arterial oxygen level, is doing, so 80 SaO₂ is actually about 60 PaO₂. PaO₂ normal is about 80. Now this guy dropped to 10 SaO₂ then partially woke himself up, then went up to 30 or 40 SaO₂ and stayed there then started going down again. And the way he woke himself up—he was almost moribund and choking—was to slap himself on the leg repeatedly. Then he’d go off to sleep and the whole cycle would start again. I watched this for two hours, sitting by the bed, then I rang Colin Sullivan and said, "We’re going to have to start him on CPAP. I’m absolutely terrified that the next breath he draws will be his last." So he was started on CPAP—the highest pressure I could get him on, to stop the desaturations. He had a PSG the night after and started on CPAP. After that he was like a new person.

What was the treatment like when you started?

At that stage it was very custom-built! Masks were made by just one person—Jim Bruderer in the University of Sydney Biomedical Engineering Department. He would take a plaster cast of the nose and make a mask from that. The masks were glued on to the face with a Silastic® type compound that had to be mixed. But on the first night of the study with CPAP I would have to search through a box of spares and find one that fitted, as close as possible, and glue it onto the person’s face. And it might mean that five or six times during the night I’d have to reglue it because we wouldn’t want to waste that time, or money, in having the patient not using the CPAP.

What were the machines like?

The very earliest machine was a paint compressor, with the motors reversed, with thick white tubing, which was pool tubing. An attachment connected this tubing to double-ribbed, throwaway, respiratory tubing that fitted to the mask. This machine delivered anywhere between 4 and 20 cm H₂O CPAP pressure. It was noisy as anything [and had] tremendous turbulence, which made breathing against it more difficult.

How did you do the studies?

We used to do two sleep studies a night with a third person just on oxygen. The two people having studies were being tracked by monitors that were very tall, almost above the door, and each of the 16 channels was nearly the size of a current PSG. These units were very complicated, and I don’t have a wonderful relationship with complicated machinery, so that was a big steep learning
THE FIRST SLEEP TECHNOLOGIST IN AUSTRALIA: AN INTERVIEW WITH BRON LEHRHAFT
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curve, but I became very adept at it. You didn’t have anything else, and the one that was just on oxygen had an SaO2 monitor, with nothing else to go by. We didn’t have a digital readout or anything. We would go around and see the patients and check the masks and see if we needed to adjust the pressure—usually taking it up—depending on what was happening.

The patients would be there for three or four days, then they would take the machine home with them. They would have their own mask by then, from the plaster cast.

How did the treatment evolve?

When I started, patients would come back after three or four months. They weren’t followed really closely but they were followed. I asked permission then to have a problem clinic in about April 1986. And I found the patients would do better if they came to the clinic, because they were being seen more regularly. Originally I would give them my telephone number or my pager number and they would ring me if they were having problems.

Then that developed into the system where they would ring two weeks after they had their sleep study and make an appointment with me. I was running three CPAP clinics at RPAH at one stage. Two were with respiratory failure patients and the other was in the sleep unit. I discovered that if you left them longer than two weeks without any follow-up support then you had a greater chance of losing them. If you couldn’t deal with those problems in the first 2–3 weeks most of the patients would just give up and say—‘It’s too hard.’

“It was a really exciting time. Nurses do not usually get to work at the cutting edge of a whole new area of medicine.”

Did this work?

In 1986–88 I did a two-year follow-up. I knew that I had seen most of these patients—it was when I had sole command, I didn’t have anyone else seeing them beforehand or afterwards—and I found we had an 89% adherence to CPAP. It was really quite remarkable. But this wasn’t accepted because we didn’t have any monitors, and critics said, oh the patients are just telling you what you want to hear. There were some people who’d fallen by the wayside and never returned, or who lived too far away and didn’t come back, but the ones that came back were still using CPAP. And it had made an enormous difference in their lives.

What improvements did you see?

Improvements in how they were during the day, in their personality; less depression; just simply able to start thinking what they were going to do. There were blood gas improvements. In every case where I knew that they were on CPAP and not just fiddling around, the pulmonary artery pressure was stabilised and came almost to normal, or if not, then very close. Sleeping through the night; their skin was fine; diabetes improved. We had noticed when we were doing the rounds in 1987–88 we had people in for all sorts of reasons and we noted that there were three diabetics in at about the same time and each of them was waking up round about 3 am, 4 am with hypoglycaemia—really low blood sugars. That started a study into the effect of CPAP on diabetics and it was discovered that CPAP will actually improve their condition and improve the glucose levels in the blood and obviously insulin levels as well.

How did the masks evolve?

After a couple of years there was one mask for everyone—just one size, with elastic around the sides and over the head. If their nose was any different from the small norm there was a pressure area over the bridge of the nose where people would get pressure sores, so I would treat that—just like I used to treat patients with pressure area care in the ‘60s—massaging and making sure that the blood flowed so they didn’t have a chance for the bruise to form and the skin to die.

Then ResMed started to develop some masks. I still think that ResMed masks are, if not the best in the world, then among the best in the world. They seem to suit the greatest number of faces. With those early masks I found that if I just pressed lightly on either side of the nose it helped because it stopped air coming in. So then I made a shape from fencing wire which came around the nose, and I would just squeeze it in lightly. Then I said to ResMed, I’m making these at the rate of knots is there any way we can design a mask like this? It was a really exciting time. Nurses do not usually get to work at the cutting edge of a whole new area of medicine.

How was sleep apnea viewed in those early days?

There was opposition to the idea of sleep apnea at the time. I can remember in 1986 one respiratory physician said that he thought sleep apnea was just a money-making fad.

How was sleep apnea treated previously?

Tracheotomy. Some of the early patients that I studied had had a tracheotomy which had been closed, but they had had terribly severe respiratory failure.

Did the tracheotomy help?

Yes. From the oropharynx, or down from behind the nose, down to your voicebox or epiglottis, it’s just a muscular passageway, which can narrow in sleep, so that air is restricted in and out of the lungs. You have a high-level sensor system that stops you...
from suffocating in sleep—as soon as the oxygen level starts dropping it cuts in and wakes you up. But that dampens over time, and during REM sleep you’re virtually paralysed so you haven’t got muscle control over that part of the pharynx. By bypassing that upper airway, with a tracheotomy down into the trachea, you don’t have the problem. But you can’t swim, can’t have a bath, you’ve got the risk of infections because you haven’t got the air being warmed or moistened. And it was performed on a lot of people. Not in the very mild cases but in the severe cases.

So the revolution was creating a mask to use positive airway pressure, so patients didn’t have to have a tracheotomy.

Yes. Of course the people with sleep apnea who had a tracheotomy just breathed through that—they weren’t using a ventilator or positive airway pressure at all before the invention of CPAP devices and masks.

But there were people with diseases like muscular diseases and neuromuscular diseases who were permanently on a ventilator with a tracheotomy. They had to live in hospital. There was no such concept as ‘weaning them off’ a ventilator to send them home.

There had to be a way of treating respiratory failure that didn’t involve spending the rest of your life on a ventilator in hospital. It was really about supporting their breathing during the night. A person with one of the neuromuscular disorders gets into the most dreadful state at night, but their breathing during the day is reasonably stable. For people with neurological conditions such as Duchenne’s syndrome, using CPAP can improve their quality of life, and extend their life.

How has treatment evolved over the years?

There have been enormous changes to CPAP devices, and the masks. When you think that the original CPAP was heavy, cumbersome, with massive tubing, and went into a heavy box... and now the newest one is tiny! Basically you need a CPAP machine that is quiet, will continue working, is not going to stop working after two weeks, and you need the comfortable interface.

What would your ideal CPAP device be like?

ResMed’s latest, the S9™, fulfils just about every promise it makes. I use one myself and it’s an outstanding machine, a totally new concept. The climate control on the tubing, the pressure delivery, all the other features. I don’t know how they’re going to better it. I’m not paid to say this—it’s what I’ve experienced.

How are sleep studies conducted now?

In sleep studies, the gold standard is that you do a PSG sleep study and then once the diagnosis is made, you know what their oxygen levels have dropped to, you know what their breathing is like, you know what you’re treating. Then you do a CPAP titration study, which is set up exactly the same way as the diagnostic was, and you titrate the pressure they need for that night. Sometimes there will need to be some changes to the pressure when they go home, usually upwards rather than downwards, because it’s only on the night of the study that you’ve got the problems of them sleeping in a strange bed, in a strange room, with 22 electrodes on and all the rest of it.

What has your long experience with CPAP shown you about its current use?

There are people using CPAP machines now who haven’t been properly assessed. They come to us on anything between 4 and 20 cm H2O, and might have been using the machine for years, but haven’t been shown how to use it, or fit a mask or anything. They might have a ramp that they can use to increase the pressure until they drop off to sleep, but they don’t know that. They’re not using the machine properly so they think it doesn’t work and it’s not improving their condition. If the pressure starts off too high you’ll struggle against it, and every time you swallow you take in a bolus of air, which inflates your stomach, making you very uncomfortable by the morning. So you won’t keep the machine on all night. If we know that someone is on a manual titration and they need 12 cm H2O during the night, it needs to start at 8 or 9 cm H2O so that as they’re getting off to sleep they’re able to stay asleep until the pressure starts moving up. On the other hand you don’t want it starting too low, because it only goes up 0.2 cm H2O every 70 seconds and they’ll be waking up choking before it’s reached the necessary pressure.

“I still get excited conducting a sleep study, watching the REM marching across the page then watching the obstructions and knowing that I’m holding in my hands something that will make them better.”

Where do you think the treatment for OSA is heading?

There have been such enormous strides in the development of CPAP machines that you don’t need so much knowledge to operate them. But you still need skilled sleep technologists to conduct sleep studies, to read the PSG and understand what it’s telling you. I’m worried that too many people are being given CPAPs without proper diagnosis, and without proper advice and follow-up. The area needs to be tightened up, with registration tests for sleep technologists and set standards for sleep labs.

What work are you involved in now?

I’m still in CPAP. I’m still in sleep. I still get excited conducting a sleep study, watching the REM marching across the page then watching the obstructions and knowing that I’m holding in my hands something that will make them better. I’m the longest serving sleep technologist in Australia—I don’t know if it’s in the world, but certainly in Australia. Since 1984 to now. And it has been just wonderful.
Steve Creighton is a professional driver who lives in Western Sydney with his wife Kate and their three teenage children.

Kate Creighton is a registered nurse, so she’s always mindful of health issues. When she was pregnant with their first child in 1994, she and her husband Steve went to see a respiratory specialist. Kate’s sister had lost a baby to sudden infant death syndrome (SIDS) and Kate was concerned that there may be a genetic link that would predispose her baby to the same risk. After speaking to the specialist about the risk of SIDS, Kate added, “By the way, Steve’s got sleep apnea.”

“This was news to me,” Steve says, recalling that day nearly 17 years ago. “I didn’t think that there was a problem, but she said when I’m sleeping I stopped breathing and she had to give me a nudge—I had no idea. But I went to see a sleep specialist and they said yes, you’ve got sleep apnea; it’s mild, you have 20 episodes an hour. I remember thinking, that’s every three minutes, which seemed like a lot. They put me on a CPAP trial with a Sullivan 4 and the effect was fairly dramatic. I felt, wow I’ve got energy now. It was really good. I understood why I needed treatment. Life was good again.”

Their first baby, a boy, was born and monitored for any breathing irregularities, but although the monitor was triggered on a few occasions there were no crises. “We’d learnt the resuscitation techniques but didn’t have to use them—we’d just go in and check on him and he was never blue, no problems.” Kate and Steve went on to have two more children, both girls, in 1996 and 1998. Both were monitored but didn’t even trigger the alarms. “I’ve never had a problem using CPAP,” Steve says. “OK,” he adds, “the first time I tried it, it was a bit confronting but I thought, yes there’s a mask but let’s get over that; yes there’s air flowing, but I could put the machine on ramp so it would start off soft and gradually come up to the required pressure. One of my sleep studies a few years ago showed that I’m actually borderline narcolepsy. I’ve always been able to go to sleep very easily and now I know why. So sleeping with the mask has never been a problem.”

He takes his CPAP with him when he travels, including an overseas trip in 2005. “No problems,” he says.

When he was diagnosed with borderline narcolepsy, Steve was prescribed dexamphetamine—15 mgs daily, which he takes in three lots of 5 mgs throughout the day. He notices that it adds another dimension to the treatment of his condition. “Before I started CPAP I’d be tired all the time,” he says. “Once I started on CPAP I’d be fine, but if I had a big lunch I’d be falling asleep. Now that I’m staggering the dexamphetamine I can take it after lunch and it takes that edge off the tiredness. So you need the combination—it’s not like one thing will fix you.”

Another part of his treatment was a rhinoseptoplasty because his septum was crooked. His nose had been broken when he was younger, blocking his left nostril. This meant he had to work harder to breathe. The procedure didn’t affect his sleep apnea, but has let him breathe more easily in general.

Steve has noticed that a number of people he has worked with are also on CPAP, but he’s baffled by the people who say they can’t stay on it. “You mention it in a group and there’s bound to be someone else who’s on it or knows someone who’s on it. It’s fairly widespread. Some people say they have trouble staying on the treatment. I can understand if they get claustrophobic, but if it’s just that it feels uncomfortable then they should just get through that.”

“But the first time you ever wore a hat it might have felt uncomfortable but if you don’t wear a hat and you go out in the sun you end up with skin cancer. CPAP is something that’s going to improve your quality of life and it’s not just the tiredness, it’s the long-term effects that sleep apnea has, the stress levels and the heart and the rest of the body. I’ve been on it now for 16–17 years and I’m glad. If I was just starting on it my health would have been compromised by now. I’m fit. I’m a few kilos overweight now, 82 kilos, should be 75, but not at all obese.”

“OSA doesn’t just affect you, it affects your whole family—your partner, your kids, how you react and interact with them, your whole quality of life.”

As Steve is a professional driver his use of CPAP is reportable—it’s marked on his drivers licence and his government authorisation to drive a bus. He has to see a specialist every 12 months to check on his compliance and to ensure that the treatment is working. If Steve is involved in a driving accident...
he would probably be asked to give a blood sample to make sure that he had taken his dexamphetamine, even if he wasn’t at fault. His CPAP use may also be checked, by downloading his usage record from his flow generator.

Steve takes this responsibility very seriously. “Even after a couple of days off CPAP I feel the difference,” he explains. “If I’ve got a cold, I get a blocked nose, and I might pull the mask off during the night. But I wouldn’t do that two nights in a row. I’ll go and get something for the cold so that when I get to bed I know I’ll be able to sleep.”

“It’s being proactive I suppose. We do the occasional trip to visit my family in Newcastle and I usually take my CPAP machine with me, but sometimes I’ll get there and realise I’ve forgotten the lead or something. Overnight, one night, is not a problem, I know what’s happening and I can usually compensate. But if it’s more than two nights I feel tired, wrung out. It raises my stress levels. So it’s a vicious cycle, because the stress makes it harder to relax.”

He explains that he wouldn’t drive in those circumstances. “I’d know I wouldn’t be fit, wouldn’t have the judgment. Because of being a professional driver I’ve got to take ownership of it.”

Recently Steve upgraded from the Sullivan 4 that he had been using since 1994. It was still working effectively, but Kate had heard about a new generation of flow generators. As Steve describes it, “Whereas the old machine was continuous air pressure the newer one will start off at a low pressure, sense your breathing and then only increase the pressure when it registers that you’re having an apnea. It’s not continuous air pressure, it only gives you the pressure when you need it.”

Steve is also trialling a humidifier at the moment as his throat is a bit husky, possibly through using the CPAP. He had tried using a humidifier in the past on the Sullivan 4 but the mask and the tubing became wet with condensation. He’s finding that humidification with the new machine is trouble-free.

Kate’s interest in the area of sleep motivated her to seek work at the Sleep Disorders Centre at Concord Repatriation General Hospital, Sydney, and she has been there for the past 12 years. This has helped Steve to keep up to date with improvements in technology and information about sleep apnea. He finds this access to the latest news helps him to stay involved with his treatment. “If you don’t keep up to date or have regular check-ups you don’t know what’s happening,” Steve says. “You might find out that what you’re doing is good but that something new is a lot better. That’s part of the treatment—making sure you keep yourself informed.”

Steve is a very experienced and thoughtful CPAP user and he has some advice for people who have just been diagnosed with OSA: “Think about the long-term effects, what sleep apnea can do to you if you’re not treated. You only get one life so it’s up to you to say, ok if this is what I’ve got I’m going to look at all the options. Apart from CPAP what other options are there? Is there a drug that’s an option for me? Surgery is invasive but is there a possibility?—removing adenoids or tonsils, or fixing a broken nose like I did. Have a look at your diet and exercise. Obviously! And realise that having OSA doesn’t just affect you, it affects your whole family—your partner, your kids, how you react and interact with them, your whole quality of life.”

“So, if you try CPAP and it’s a bit uncomfortable—yes it might be, but it’s going to be more uncomfortable if you don’t persist and see what sorts of benefits you might be able to get from it. I would say hang in there and even if you don’t like it, try it for at least a fortnight and within that time you should be able to see the benefits.”
Road safety laws and OSA

Australia

Road safety laws in most parts of Australia require all drivers, whether they are driving a private car or a commercial vehicle, to report to their Driver Licensing Authority any permanent or long-term illness that is likely to affect their ability to drive safely. Health professionals may then be required to make an assessment of a person’s health to assess their ability to drive safely. Health professionals should also advise patients if a medical condition impacts on their ability to drive safely, whether in the short or long term. Criteria for medical conditions that affect a person’s driving, including sleep disorders, alcohol dependency, heart diseases and respiratory disorders, are listed in the publication, Assessing Fitness to Drive, published by Austroads, the association of Australian and New Zealand road transport and traffic authorities, and the Australian National Transport Commission. Assessing Fitness to Drive has recently undergone a review period, and a new edition is expected in mid-2011. See www.austroads.com.au for more information.

USA

In the US the Federal Motor Carrier Safety Administration (FMCSA) publishes guidelines for medical examiners who are determining a driver’s medical qualifications pursuant to Section 391.41 of the Federal Motor Carrier Safety Regulations (FMCSRs). According to their website, “The medical examiner may, but is not required to, accept the recommendations. Section 390.3(d) of the FMCSRs allows employers to have more stringent medical requirements.” Recommendation 391.41(b)(5) in regard to respiratory dysfunction states that: “There are many conditions that interfere with oxygen exchange and may result in incapacitation, including emphysema, chronic asthma, carcinoma, tuberculosis, chronic bronchitis and sleep apnea. If the medical examiner detects a respiratory dysfunction, that in any way is likely to interfere with the driver’s ability to safely control and drive a commercial motor vehicle, the driver must be referred to a specialist for further evaluation and therapy.”

UK

UK law requires drivers to tell the Driver and Vehicle Licensing Agency (DVLA) about any condition that may affect their ability to drive safely. Notifiable conditions, for holders of any drivers licence, include sleep apnea. The DVLA website warns that a driver who is involved in an accident where their health condition was a contributing factor may be prosecuted and their insurance may not be valid.\(^2\)

Canada

The Canadian Lung Association reports that: “Doctors have a duty to tell the motor vehicle board [of their province or territory] about patients who are not safe to drive. If you have sleep apnea and you refuse treatment, your doctor is required to report you. The motor vehicle board could suspend your license until you get your sleep apnea treated.”\(^3\)

Europe

A study of 25 European countries published in 2008\(^4\) found that: “Excessive daytime sleepiness is mentioned in nine [driving licence regulations], whereas sleep apnoea syndrome is mentioned in 10 countries. A patient with untreated sleep apnoea is always considered unfit to drive. To recover the driving capacity, seven countries rely on a physician’s medical certificate based on symptom control and compliance with therapy, whereas in two countries it is up to the patient to decide (on their doctor’s advice) to drive again.”

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An all-round view of sleep disorders: An interview with Professor Barbara Phillips

How was research into SDB/OSA viewed when you started out?
The field of sleep medicine has been multi-disciplinary from the outset, and thus has not been considered to be mainstream by pulmonary, neurology, psychiatry or any of its other constituent branches. Sleep research was neither fish nor fowl. Unfortunately, that is still partly true.

With increasing literature showing an association between OSA and other comorbidities such as cerebro- and cardiovascular disease and depression, do you think sleep medicine will become more mainstream?

Probably. And the recognition that sleep apnea has significant comorbidities may shift its management away from sleep specialists who haven’t done a very good job of promptly, efficiently, inexpensively managing this very prevalent, deadly disease. For example cardiologists, who live in a culture of urgency, are among those who are getting involved in the diagnosis and management of sleep apnea.

Can you describe what the treatment for OSA was like when you started out?
Continuous positive airway pressure (CPAP) treatment was rarely used in the early ‘80s. We tried (with the same kind of success that is still the norm today) to get people to lose weight. But folks who were seriously afflicted with OSA were best treated with a tracheotomy.

How has the treatment for OSA evolved over the years?
Well, there is no question that CPAP is the treatment of choice. Much peer-reviewed literature demonstrates that it effectively treats most of the secondary consequences of sleep-disordered breathing. Oral appliances are clearly gaining in stature and popularity, because they are easier to use, and the dental community has done a good job of proving their efficacy. And I think that most clinicians now realize that upper airway surgery is not a good choice.

Do you think the treatment for OSA is going in the right direction?
No. Until or unless we figure out how to get people to lose weight and maintain weight loss, CPAP will simply be a bandaid on the much bigger problem.

Would you like to see the diagnosis/treatment of OSA alter in any way?
Get insurance rules and regulations out of the picture! Let clinicians manage patients. Stop requiring a sleep study in order to have CPAP paid for. Stop pretending that a CPAP titration (or an in-lab PSG, for that matter) is some kind of magical test. Is the requirement that a sleep study must be carried out in order for CPAP to be paid for likely to change anytime soon? Beats me. Probably not if those who profit by sleep apnea testing continue to loudly proclaim that it is necessary in every single case.
What would your ideal therapy device be like?
It would be a very simple blower with flawless, non-condensing humidity, and a pressure knob that patients could adjust between about 6 and 16 cm H2O. It would come with a dozen different easy-to-use masks.
Would patients in the US healthcare system accept this or would they still want more?
Most of my patients want things to be simple and straightforward.
What has your ‘all-round view’ of SDB/OSA shown you about current treatment methods and attitudes toward SDB/OSA?
We have made it way too complicated and expensive for patients to get effective treatment.
Where do you think the treatment for OSA is heading?
Hopefully, simpler and more mainstream.
What can the sleep medicine fraternity do to make this happen?
More primary care education. Getting sleep apnea into the news media (or anything that increases patient awareness). Reduce some of the barriers (in-lab studies, sometimes twice, delays, unnecessary expenses) currently associated with the care of this disease.

“Sleep apnea is linked to very, very common medical problems such as diabetes and hypertension.”

In a recent talk at ResMed, Sydney, one of the points that you made was that ‘the prevalence and importance of sleep apnea are attracting attention’. Many researchers have been looking at sleep apnea for many years—why do you think it is receiving more attention now?
Because OSA harms not only the afflicted person, but also those on the road and in the car with them. And because we now realize it is linked to very, very common medical problems such as diabetes and hypertension.
Is the prevalence of sleep apnea increasing? If so, why is that?
Yes. We are getting heavier and older.
Do you think there is any difference in the treatment of SDB/OSA in the US compared to other parts of the world?
Yes. I think it is more expensive here, partly because of insurance rules and partly because there is a financial incentive to test, rather than treat, the patient. We do way too many sleep studies and not nearly enough clinic visits in the management of sleep apnea patients in the US.

Do you have a particular area of SDB/OSA research that you are currently involved in? Could you tell us a little about that, and how you came to be involved in it?
I am interested in sleep-disordered breathing in older patients. It ‘looks’ different to sleep-disordered breathing in younger patients, and appears to be much more prevalent. How should it be defined? How should it be treated? Is there a benefit to treatment of OSA in older people? Stuff like that. Why am I interested? Well, someday, if I’m lucky, I’ll be old too ...

Tregear S; Reston J; Schoelles K; Phillips B. Continuous positive airway pressure reduces risk of motor vehicle crash among drivers with obstructive sleep apnea. Sleep 2010;33(10):1373-1380.

Context: OSA is associated with an increased risk of motor vehicle crash.

Objective: We performed a systematic review of the literature concerning the impact of CPAP treatment on motor vehicle crash risk among drivers with OSA. The primary objective was to determine whether using CPAP could reduce the risk of motor vehicle accidents among drivers with OSA. A secondary objective involved determining the time on treatment required for CPAP to improve driver safety.

Data Sources: We searched seven electronic databases (MEDLINE, PubMed (PreMEDLINE), EMBASE, PsycINFO, CINAHL, TRIS, and the Cochrane library) and the reference lists of all obtained articles.

Study Selection: We included studies (before/after, case/control, or cohort) that addressed the stated objectives. We evaluated the quality of each study and the interplay between the quality, quantity, robustness and consistency of the evidence. We also tested for publication bias.

Data Extraction: Data were extracted by two independent analysts. When appropriate, data were combined in a fixed or random effects meta-analysis.

Results: A meta-analysis of nine observational studies examining crash risk of drivers with OSA pre- vs. post-CPAP found a significant risk reduction following treatment (risk ratio = 0.278, 95% CI: 0.22 to 0.35; P < 0.001). Although crash data are not available to assess the time course of change, daytime sleepiness improves significantly following a single night of treatment, and simulated driving performance improves significantly within two to seven days of CPAP treatment.

Conclusions: Observational studies indicate that CPAP reduces motor vehicle crash risk among drivers with OSA.
The evolution of CPAP:
An interview with Mark Bertinetti

Mark Bertinetti has worked at ResMed for almost 10 years on a range of flow generators and accessories. He is now the Senior Industrial Designer for Sleep Product Development.

How did you become involved in designing flow generators?
I was studying industrial design at the University of New South Wales, Sydney, and ResMed presented a case study on the design of one of the earlier devices. One of my university friends then went on to create an oxygen device for ResMed as her final year thesis project, so I became quite familiar with ResMed and its work. After university I started my own design consultancy and worked for another design company for a number of years before deciding I really wanted to be in a company structure where you get to interact on a daily basis with all the major stakeholders in the design. I work at ResMed with the marketing team and with some very talented mechanical, electrical, software and system engineers on a daily basis. The other thing that attracted me to this job is that it's very fulfilling to know that your work is making a difference to people's lives.

Could you describe your job?
As the Senior Industrial Designer I see myself as a translator between marketing and engineering. I understand a little bit of everything. I have studied design, marketing, human factors, ergonomics, web design, materials and a range of manufacturing processes. This means I can use the inputs from all directions plus the market research to create a product that is easy to use, aesthetically pleasing and can be produced cost-effectively while meeting the electrical, software, and mechanical requirements. The key to success is to create a product that touches the product during its lifecycle—from the people assembling it on the assembly line, to a physician or sleep technician using it to titrate in a sleep lab, to a dealer setting it up for a patient, to the patient and partner’s experience with it in their bedroom.

Then there are all the considerations about manufacturing the product, shipping it, packaging it, distributing it around the world, having storage facilities for it and making sure it fits the business model for everyone, as well as the usage model for those who are actually using it.

Flow generators are constantly evolving. How do you decide what needs to be changed when you create a new model?
As I've mentioned, we're catering for sleep technicians, distributors, patients, physicians and assemblers so we have to understand all of their needs and how they each interact with the product. We recently ran some focus groups looking at what's working and what's not working for people who use the S9™ sleep therapy device. Twenty-five patients came in and spoke about what they liked about it, what difference it has made to their life and how they used it, to give me some input for future designs. Some were people who've been using CPAPs for a long time, and for others, the S9 was their first CPAP. We did a similar thing with S8™ before designing the S9. It's great to speak with patients directly to hear about the issues they may be having, the ways they think it can be improved, and to hear about their basic daily routine—whether they wash their masks every day, whether they pull off the tube, how they store things, what settings they use and what feedback they would like.

Other important feedback comes to us from sleep technicians, physicians, distributors and ResMed staff in regions including US, Germany, France, UK, Japan, Asia-Pacific. The regions sometimes have quite different requirements and it is my job to create a design that best satisfies all those needs.

We are always looking into new technologies and this is often what allows us to make significant improvements in CPAP design and comfort. For example, the new ClimateLine™ heated tube delivers optimal temperature and humidification all the way to the mask while virtually eliminating rainout. This was only possible thanks to some incredible engineering and a new process that enables us to wind wires into the tubing's helix and capture tiny sensors in the cuff. Prior to these technologies it wasn't possible to cost effectively make an automatically controlled heated tube. As technology changes, we're constantly able to innovate and fix problems that we couldn't fix before.

From the market research, people were saying that they didn't want something in their bedroom with a big tub and a big hose coming out of it. Visitors who saw their flow generators would say, “Are you ok, are you sick or something?” So we created a product that met all the stringent quality, safety, and durability requirements of a medical device while looking like a contemporary consumer product. The S9's more inviting appearance allows users to be more accepting of it, creating one less barrier to treatment.

Is the development of the devices linear, building on previous devices, or is it a whole new development every time?
Every device we've done has been a totally new development. Nothing has borrowed parts from previous models. I think the only thing that's common between the S8 and the S9 is some screws and a spring. Everything else is totally new. We apply all the latest in technology and make everything the best it
THE EVOLUTION OF CPAP: AN INTERVIEW WITH MARK BERTINETTI

CONTINUED >

can be to deliver a new device. Even the AutoSet CS2™, that looks similar to the S7™, was completely different. It just looks similar so it could fit with the H2™ humidifier, but the only thing they had in common was the handle.

When we started designing the S9, we literally did hundreds of sketches as a design team and pinned them all up on the wall. Then we looked at them all and said, “I like the button in that one, and the tube in that one; that one solves the humidifier issue.” As you look across the wall you start to converge them into a handful of ‘superconcepts’ that draw on all the best ideas and eliminate many issues. To help give it a more attractive look, we teamed up with an external design consultancy from the consumer design world. This combination of expertise resulted in a safe, reliable medical product in consumer ‘clothing’.

“As technology changes we’re constantly able to innovate and fix problems that we couldn’t fix before.”

Is there any difference between the old machines and the new ones? They both deliver air pressure.

The new devices are very different compared to the earliest ones, mainly because of the science behind the algorithms. The algorithms are now much smarter than they ever were, while the earliest devices were very basic—they just blew a constant air pressure the whole night through. The algorithms can now detect the difference between a central apnea and an obstructive apnea. The motor sends tiny little pulses through the airway and looks for the pulses coming back so it can determine whether the airway is open or closed. If the airway is closed it’s an obstructive apnea so it increases the pressure; if the airway is open it’s a central apnea so it won’t change the pressure.

The blower is also quieter than ever before, with conducted noise reduced by 78% (8 decibels). When ResMed was formed, the blower design was done almost on pencil and paper. Today, with the computer aided design (CAD) software that we have, we can run simulations of the airflow through the blower and optimise every little detail in the design.

Today’s devices have many differences in terms of materials and processes compared with earlier machines. Now we can use ‘two-shot moulding’ to mould two different types of materials into one so we can improve assembly, ease of use, sealing and noise. For example, the top cases have a hard structural plastic with a softer material moulded over the top of it to create the waterproof keypad.

Additionally, there are all the environmental considerations that we design for now. Because the blower is so much quieter, we can use less foam than we used to. The size of the device is optimised in terms of how it fits onto a pallet, and how a pallet fits into a shipping container, so we can ship up to 75% more S9 units in a 40-foot container than we could with S8 units. We’ve tried to think about the whole lifecycle of the product. All of the materials used are ROHS (Removal of Hazardous substances) compliant where possible, so when you dispose of it and it breaks down it doesn’t release hazardous substances.

Do current devices deliver better treatment?

Yes, definitely. We can make things seal better and be quieter and do things that weren’t possible before. I’ve already spoken about how the algorithms deliver more targeted treatment, and people in the focus groups find the therapy easier to use. We’ve improved the overall experience with the product—its ease of use, comfort, the aesthetic look and feel. This all affects how it makes you feel in terms of your self-esteem—that’s critical to initial acceptance. Once people get past the psychological barrier of acceptance and start using it enough to feel the difference, they won’t go back because it becomes a natural part of their life. They feel better, they’re more active, their whole quality of life improves—they generally don’t want to give up their CPAP once they feel its impact on their health.

The impact on health goes beyond just getting a better night’s sleep and feeling more awake. Research has shown that there is a high mortality risk associated with untreated OSA.1 There is also an increased likelihood of hypertension, cardiovascular disease and stroke.2 Treating the OSA can actually help the comorbid conditions as well. Take people with type 2 diabetes, for example. The prevalence of OSA is higher in this group than in the general population3 and CPAP use has beneficial effects on insulin sensitivity.4 People with heart failure have improved blood pressure and left ventricular function when their OSA is treated with CPAP5.

Are the devices treating different conditions now?

The range of devices available now means that we can effectively treat a wide range of conditions, from basic OSA through to Cheyne-Stokes respiration and respiratory failure. We’re also catering for a broader population. There’s been a definite shift towards treating much younger users. As medical
practitioners and the public become more aware of the effects of sleep-disordered breathing, as youth obesity and lifestyle stress increase, and as diagnosis methods improve, the market includes many more socially active, younger, working people (including a higher proportion of females) than ever before. Quite recently, research has shown that women were under-diagnosed because their sleep apnea symptoms present quite differently to those of men. The first sign of OSA for women is often insomnia, or symptoms such as fatigue or mood disturbance.6

So, we are treating people with different needs, meaning that the design must evolve to accommodate a greater range of lifestyles, medical conditions, aspirations, gender differences and social pressures.

What direction are CPAPs going in now? What’s your ultimate aim?

I think the biggest challenge is to change the initial perceptions of CPAP by really improving the experience that the user has with it. Portability and cost are probably the next big issues because I think the market dynamic is shifting. People no longer want a machine that just sits in their bedroom—most of the users in the focus groups have active lives and go travelling with their CPAP for business, camping trips and holidays. The S9 allows them to maintain their active lives because it’s small and light enough to travel with and it can be run using low voltage power, even on a plane. Men over 40–50 years of age used to be the typical CPAP user but now it’s not uncommon for men and women in their mid to late 30s to be using them.

"As the Senior Industrial Designer I see myself as a translator between marketing and engineering."

The future CPAP must suit a broader market and meet people’s desires to be more social and mobile, by giving them the freedom and peace of mind to travel, or even just go to a friend’s place and get a good night’s sleep without feeling embarrassed about their treatment. The ultimate aim is to improve the experience of using CPAP to the point that it is easily accepted into everyday life and seen as a best friend, not an enemy.

Thirty years of CPAP: Key research articles


SUMMARY: Five patients with severe obstructive sleep apnoea were treated with continuous positive airway pressure (CPAP) applied via a comfortable nose mask through the nares.

Low levels of pressure (ranging from 4.5 to 10 cm H2O) completely prevented upper airway occlusion during sleep in each patient and allowed an entire night of uninterrupted sleep. CPAP applied in this manner provides a pneumatic splint for the nasopharyngeal airway and is a safe, simple treatment for obstructive sleep apnoea syndrome.


BACKGROUND: Limited data has suggested that sleep-disordered breathing (SDB), a condition of repeated episodes of apnea and hypopnea during sleep, is prevalent among adults. Data from the Wisconsin Sleep Cohort Study, a longitudinal study of the natural history of cardiopulmonary disorders of sleep, was used to estimate the prevalence of undiagnosed SDB among adults and address its importance to public health. METHOD: A random sample of 602 employed men and women years 30–60 was studied by overnight polysomnography to determine the frequency of episodes of apnea and hypopnea per hour of sleep (the apnea–hypopnea score). We measured the age- and sex-specific prevalence of SDB in this group using three cutoff points for the apnea–hypopnea score (≥5, ≥10 and ≥15). We used logistic regression to investigate risk factors. RESULTS: The estimated prevalence of sleep-disordered breathing, defined as an apnea–hypopnea score of 5 or higher, was nine per cent for women and 24 per cent for men. We estimated that two per cent of women and four per cent of men in the middle-aged work force meet the minimal diagnostic criteria for the sleep apnea syndrome (an apnea–hypopnea score of 5 or higher and daytime hypersomnolence). Male gender and obesity were strongly associated with the presence of SDB. Habitual snorers, both men and women, tended to have a higher prevalence of apnea–hypopnea scores of 15 or higher.

CONCLUSION: The prevalence of undiagnosed SDB is high among men and is much higher than previously suspected among women. Undiagnosed SDB is associated with daytime hypersomnolence.


BACKGROUND: SDB is prevalent in the general population and has been linked to chronically elevated blood pressure in cross-sectional epidemiologic studies. We performed a prospective, population-based study of the association between objectively measured SDB and hypertension (defined as a laboratory-measured blood pressure of at least 140/90 mm Hg or the use of antihypertensive medications). METHOD: We analysed baseline data on SDB, blood pressure, habitus and health history, and after four years analysed follow-up values in 709 participants of the Wisconsin Sleep Cohort Study (and after eight years, follow-up values in the case of 184 of these participants). Participants were assessed overnight by 18-channel polysomnography for SDB, as defined by the apnea–hypopnea index (the number of episodes of apnea and hypopnea per hour of sleep). The odds ratios for the presence of hypertension at the four-year follow-up study according to the apnea–hypopnea index at baseline were estimated after adjustment for baseline hypertension status, body-mass index, neck and waist circumference, age, sex, and weekly use of alcohol and cigarettes. RESULTS: Relative to the reference category of an apnea–hypopnea index of 0 events per hour at baseline, the odds ratios for the presence of hypertension at follow-up were 1.42 (95 percent confidence interval, 1.13 to 1.78) with an apnea–hypopnea index of 0.1 to 4.9 events per hour at baseline as compared with none, 2.03 (95 percent confidence interval, 1.29 to 3.17) with an apnea–hypopnea index of 5.0 to 19.9 events per hour, and 2.89 (95 percent confidence interval, 1.46 to 5.64) with an apnea–hypopnea index of 15.0 or more events per hour. CONCLUSIONS: We found a dose-response association between SDB at baseline and the presence of hypertension four years later that was independent of known confounding factors. The findings suggest that SDB is likely to be a risk factor for hypertension and consequent cardiovascular morbidity in the general population.


CONTEXT: SDB and sleep apnea have been linked to hypertension in previous studies, but most of these studies used surrogate information to define SDB (eg. snoring), or were based on small clinic populations, or both. OBJECTIVE: To assess the association between SDB and hypertension
in a large cohort of middle-aged and older persons. **DESIGN AND SETTING:** Cross-sectional analyses of participants in the Sleep Heart Health Study, a community-based multicenter study conducted between November 1995 and January 1998. **PARTICIPANTS:** A total of 6132 subjects recruited from ongoing population-based studies (aged ≥ 40; 52.8% female). **MAIN OUTCOME MEASURES:** Apnea–hypopnea index (AHI), the average number of apneas plus hypopneas per hour of sleep, with apnea defined as a cessation of airflow and hypopnea defined as a ≥30% reduction in airflow or thoracoabdominal excursion, both of which are accompanied by a ≥4% drop in oxyhemoglobin saturation (corrected), obtained by unattended home polysomnography. Other measures include arousal index, percentage of sleep time below 90% oxygen saturation, history of snoring and presence of hypertension, defined as resting blood pressure of at least 140/90 mm Hg or use of antihypertensive medication.

**RESULTS:** Mean systolic and diastolic blood pressure and prevalence of hypertension increased significantly with increasing SDB measures, although some of this association was explained by body mass index (BMI). After adjusting for demographics and anthropometric variables (including BMI, neck circumference and waist-to-hip ratio), as well as for alcohol intake and smoking, the odds ratio for hypertension, comparing the highest AHI category (≥30 per hour) with the lowest category (<1.5 per hour), was 1.37 (95% confidence interval [CI], 1.03-1.83; P for trend = .005). The corresponding estimate comparing the highest and lowest categories of percentage of sleep time below 90% oxygen saturation (≥12% vs <0.05%) was 1.46 (95% CI, 1.12-1.88; P for trend <.001). In stratified analyses, associations of hypertension with either measure of SDB were seen in both sexes, older and younger people, all ethnic groups and among normal-weight and overweight individuals. Weaker and non-significant associations were observed for the arousal index or self-reported history of habitual snoring.

**CONCLUSION:** Our findings from the largest cross-sectional study to date indicate that SDB is associated with systemic hypertension in middle-aged and older individuals of different sexes and ethnic backgrounds.


**OBJECTIVE:** To assess whether sleep apnoea syndrome is an independent risk factor for hypertension. **DESIGN:** Population study. **SETTING:** Sleep clinic in Toronto. **PARTICIPANTS:** 2,677 adults, aged 20–85 years, referred to the sleep clinic with suspected sleep apnoea syndrome. **OUTCOME MEASURES:** Medical history, demographic data, morning and evening blood pressure, and whole night polysomnography.

**RESULTS:** Blood pressure and the number of patients with hypertension increased linearly with severity of sleep apnoea, as shown by the apnoea–hypopnoea index. Multiple regression analysis of blood pressure levels of all patients not taking antihypertensives showed that apnoea was a significant predictor of both systolic and diastolic blood pressure after adjustment for age, body mass index and sex. Multiple logistic regression showed that each additional apnoeic event per hour of sleep increased the odds of hypertension by about 1%, whereas each 10% decrease in nocturnal oxygen saturation increased the odds by 13%. **CONCLUSION:** Sleep apnoea syndrome is profoundly associated with hypertension independent of all relevant risk factors.
# 2011 Calendar of Events

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<td>ATS 2011</td>
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<td>12–16 November</td>
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## Global Offices

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