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Sleep medicine – prevalent and relevant

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ABSTRACT – There is an increasing awareness of different sleep disorders among the public and healthcare professionals, and the impact they can have on an individual. This conference was organised jointly with the British Thoracic Society to discuss some of these pertinent conditions, issues around driving and around service planning to accommodate an increasing specialty.

KEY WORDS: Obstructive sleep apnoea (OSA), continuous positive airway pressure (CPAP), obesity hypoventilation syndrome, driving

Normal sleep from young to old and the consequences of sleep disruption

Sleep requirements change across a lifetime, but most adults require on average 7–9 hours per night. It is well recognised that sleep patterns are affected by age, circadian rhythm disturbance, sleep deprivation, alcohol, caffeine and medication, as well as with sleep disorders, of which obstructive sleep apnoea (OSA) is one of the most common seen in sleep clinics. Sleep loss of whatever cause has many different impacts: daytime sleepiness, mood change, worsening cognitive function, memory impairment, changes in metabolic rate and temperature, changes in growth hormone, melatonin and testosterone levels, and changes to food intake and food selection have all been shown. Older adults with sleep-disordered breathing have lower levels of daytime sleepiness as measured by the Epworth Sleepiness Score (ESS) compared with younger counterparts with equivalent-severity sleep-disordered breathing, but the reason or the significance of this fact is unclear.[1]

Obesity-related sleep disordered breathing and treatments

Increasing obesity was highlighted as the cause of much of the increasing prevalence of sleep-disordered breathing, with huge public health implications. Obesity is also common in non-respiratory sleep disorders. Not all those individuals who are obese will have sleep-disordered breathing, but it is not yet clear which factors lead some individuals who are obese to have obesity hypoventilation syndrome (OHS) and other same-weight counterparts not to. Upper airway resistance syndrome (frequent arousals owing to increasing respiratory effort, but no desaturation), OSA (recurrent upper airway obstruction, apnoea, desaturation and subsequent arousal) and OHS (awake hypercapnia and a body mass index >30 kg/m^2), represent different phenotypes of a spectrum of disease in which weight loss remains a key component of the management. However, this is not often achieved by such patients with dietary modification alone and other weight-loss strategies, such as bariatric surgery, have been used with good effect.[2] Continuous positive airway pressure (CPAP) remains the mainstay of treatment for symptomatic moderate to severe OSA, or mild symptomatic OSA in which conservative treatments have failed.[3] Those with OHS might require bilevel non-invasive ventilation (NIV) if they are in decompensated type 2 respiratory failure, or if CPAP alone is inadequate at reversing the daytime hypercapnia. Mandibular advancement devices are used for those with mild OSA or snoring, or who are intolerant of CPAP. Novel treatments, such as neuromuscular stimulation, nasal valves and tongue protrusion devices, await further evaluation before they can be widely recommended, but might in time be useful therapies for those patients with milder sleep-disordered breathing.

Clinical consequences of obstructive sleep apnoea – brain, heart and vessels

Large epidemiological studies have shown OSA to be associated with increased rates of hypertension, cardiovascular disease and stroke. There are many confounders in these observational cohorts and, therefore, the randomised controlled trial of treatment for a single variable has emerged as a robust way to establish cause and effect. If the treatment for OSA, namely CPAP, improves blood pressure, then OSA can be implicated as the cause. Many randomised controlled trials and subsequent meta-analysis have established this cause-and-effect link for hypertension,[4] but the direct causality of OSA itself on other markers of cardiovascular risk is harder to establish. Longer-term randomised controlled trials are needed to demonstrate a change in cardiovascular health with the intervention and only minimally symptomatic patients with OSA are ethically able to be randomised. Therefore, although many ascribe a direct link of OSA to cardiovascular risk, this has not yet been unequivocally demonstrated. Most of the vascular consequences of OSA seem to result from increased sympathetic nervous system activity, probably as a result of recurrent arousal and oxidative stress. Randomised controlled trial data prove a causal relation between OSA, endothelial dysfunction and arterial hypertension, but there are still no data proving a causal relation between OSA and cardiovascular events. A novel investigative technique, CPAP withdrawal in patients with established OSA, might lead to further understanding in this area.[5] The MOSAIC trial found that those patients recruited with minimally symptomatic OSA and randomised to CPAP rather than control (no CPAP) experienced

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a clinically significant fall in ESS, despite not having a raised ESS initially.[6] Therefore, it seems reasonable to give patients with significant OSA on sleep study but minimal daytime sleepiness an empirical trial of CPAP to see whether they derive a subjective benefit and want to continue.

Driving and obstructive sleep apnoea

UK Department of Transport figures show that the cost of road traffic accidents, fatal and non-fatal, is enormous. A fatal accident cost was estimated to be approximately £1.8 million in 2009. It is estimated that 20% of motor vehicle accidents are the result of sleepiness; only small numbers of these are because of untreated OSA, yet this is the condition that seems most linked in popular consciousness with road traffic accidents.

There continues to be anxiety and discomfort among general practitioners and physicians looking after patients with possible and confirmed OSA about clear driving advice. UK law is clear that all drivers have a legal duty of care to ensure they are fit to drive and are responsible for their own driving performance. This is useful to convey to patients when discussing driving issues with them. Although the UK Driver and Vehicle Licensing Authority (DVLA) guidelines are relatively black and white on the issue, real life is often harder. There are real concerns that if patients think they will be stopped from driving by the sleep clinic (albeit temporarily until successful treatment is started), they will not want to come forward with their sleepiness problems. It is much better to be able to offer these patients an accelerated 'fast-track pathway' or a 'one-stop shop' for investigation and treatment, prioritising professional drivers, and giving sensible driving guidelines, than to inhibit them from presenting because of anxieties about driving advice. It is also much safer to diagnose such patients and treat them, than to frighten them underground. Those patients who do not have daytime sleepiness with OSA, that is the OSA syndrome, are now clearly excluded from the DVLA regulations and do not need to notify the DVLA of their diagnosis.

Sleep physicians could be expected to be precise regarding driving advice, but this might not be realistic, given the limitations of the tests available. A test to predict individual driving performance and differentiate those likely to crash before CPAP treatment is started would be ideal (akin to a breathalyser), but is not yet possible. There is a poor correlation between measures of OSA severity, sleep fragmentation, daytime symptoms (ESS or partner-scored ESS) and driving simulator performance in many different studies. Any test performed under research conditions does not reflect real-life driving conditions, even though the newer advanced driving simulators are more realistic. Therefore, it is difficult to argue that poor performances on these tests should prohibit driving, when supportive data that these tests predict crash likelihood do not exist. The ESS, although subjective and, therefore, prey to a range of errors, including denial of sleepiness by the person completing it, or a low score owing to drive and desire to stay awake despite significant sleepiness, remains the most evaluated, widely used test of daytime sleepiness. Objective tests, such as multiple sleep latency tests or the maintenance of wakefulness test, also correlate poorly with OSA severity. They might be useful clinically in individual patients to give the latter information and education about their personal wakefulness performance, but cannot be used in individuals to validly predict crash risk.

At present, although it is acknowledged that the relation between OSA, sleep fragmentation and driving performance is complex, expert opinion suggests that those patients with untreated OSA who are at most risk of RTAs are those with excessive daytime sleepiness with high ESS (>17), confirmed by family history, plus previous sleep, fatigue or inattention-related crashes or near-misses. These are the patients to whom it should be strongly suggested that they suspend driving while awaiting treatment for OSA. The American Thoracic Society published guidelines in June 2013 offering this advice.[7] The guidelines are also likely to state that there is no compelling evidence to restrict driving in untreated OSA if there is no history of crash or near-miss attributable to sleepiness, fatigue or inattention. Certainly, CPAP therapy in those with OSA appears to decrease the number of crashes per driver per year, to bring crash levels to the same as those of a control population.[8]

Clearly, the stakes are potentially higher for those group 2 licence holders, including heavy goods vehicle (HGV) drivers. There is greater potential for a catastrophic accident, in terms of fatalities and expense, owing to the size and weight of the vehicle involved. Approximately 35% of all sleep-related crashes involve HGVs and they are the most common cause of an HGV driver being killed at work. By the nature of the work, such drivers are driving long distances, often dull motorway driving, for longer periods of time at the wheel and with other potential sleep disruptions, such as early starts, late nights, sleeping uncomfortably in the cab of the vehicle during breaks or at night. Many HGV drivers are obese and OSA is also likely. Studies in the UK and Australia have sought to quantify how common OSA is in this population, with prevalence figures of approximately 4–41%.[9] Given how many HGV drivers there are in the European Union, this is a huge public health concern. There are many who argue (including some trade unions) that it would be beneficial to screen these drivers for OSA, perhaps as part of their regular 5-yearly fitness-to-drive medicals, so that if they are found to be at high risk for OSA, this can be diagnosed with sleep studies and treated promptly to enable improved well being, safe driving, improved road safety and decreased crash-related insurance costs.

Screening tools for obstructive sleep apnoea

The British Lung Foundation estimates that 80% of individuals with OSA are undiagnosed. A publicity campaign is educating healthcare professionals in primary care and members of the public about the symptoms of OSA to encourage them to seek help. Effective screening programmes would aim to detect OSA in those individuals who had not presented clinically and in whom treatment (usually CPAP) would be effective. Therefore,

Table 1. The STOP-BANG screening tool. Reproduced with permission (British Lung Foundation[11] and Chung *et al*[12]).

S	Does the patient **snore** loudly (louder than talking or loud enough to be heard through closed doors)?	Y/N
T	Does the patient often feel **tired**, fatigued or sleepy during the day?	Y/N
O	Has anyone **observed** the patient stop breathing during their sleep?	Y/N
P	Does the patient have, or is the patient being treated for, high blood **pressure**?	Y/N
B	Does the patient have a **BMI** of more than 35 kg/m^2?	Y/N
A	**Age**. Is the patient older than 50 years old?	Y/N
N	Is the patient's **neck** circumference greater than 40 cm?	Y/N
G	**Gender**. Is the patient male?	Y/N

BMI = body mass index; N = no; OSA = obstructive sleep apnoea; Y = yes.
Scoring: Y≥3 = high risk of OSA; Y<3 = low risk of OSA.

possible populations to screen include: those with obesity (such as a weight-management clinic), those with hypertension, professional drivers and, increasingly, pre-operative patients. The latter are presumed to have more potential complications from unknown and untreated OSA, such as airway management issues, intensive care admission, post-operative complications and prolonged length of stay, although evidence of the effect of screening in this area is lacking. The STOP-BANG screening questionnaire (Table 1) is now widely used to predict probable OSA and to highlight who should go on to have screening oximetry or multi-channel sleep studies.[10]

Non-obstructive sleep apnoea hypersomnolence

OSA is only one of many sleep-impacting conditions; thus, other conditions might need to be considered in patients presenting with hypersomnolence and in patients with OSA and hypersomnolence not improved after initial management. Narcolepsy, restless leg syndrome, periodic limb movements during sleep and rapid eye movement (REM) sleep behaviour disorder are all recognised, in addition to a variety of parasomnias. Vigilance is essential to prevent underdiagnosis, with a thorough sleep history being the cornerstone of assessment, and a 2-week sleep diary an additional useful tool. Tertiary sleep clinics have a greater experience within this area, and are an invaluable resource for smaller services to optimise patient management.

The future

Finally, the National Health Service (NHS) Atlas of Variation of 2010 acknowledged geographical differences in the rate of sleep studies undertaken per population by primary care trusts (PCTs). Local centres vary in the services they offer, such as oximetry vs polysomnography, plus/minus CPAP and NIV provision. Clearly, given that sleep disorders are becoming increasingly prevalent, sleep medicine is incredibly relevant. It is crucial for commissioners to continue to work with local and national sleep networks to ensure that services enable easy referral, prompt sleep studies and diagnosis, effective treatment and provision of long-term care.

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