S24

Outcomes following major OSA surgery, including quality of life analysis

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This talk will focus on three parts

1 A review of quality of life (QOL) outcomes following contemporary airway reconstruction surgery compared to CPAP (Sam Robinson/ Michael Chia published data 2009)
2 Analysis of QOL and polysomnogram outcomes following those surgeries specifically involving Submucosal Lingualplasty (Sam Robinson unpublished data)
3 Presentation of early outcomes following major airway reconstruction surgery (Stuart MacKay)

The talk aims to present the perspective that contemporary upper airway reconstruction should be discussed with patients with poor CPAP compliance/CPAP failure.

S25

Lies, damned lies, and statistics: different surgical outcomes according to different scoring systems

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This paper elaborates on the Ruehland concept of different polysomnogram scoring systems. Differences in these systems can markedly influence the interpretation of outcomes following airway reconstructive surgery for snoring and OSA.

Forty consecutive cases of adult OSA with AHI Chicago > 15, who underwent surgery, will be presented. The outcomes of AHI Chicago and AHI recommended will be discussed, as will different criteria to judge 'success' and how outcomes can vary widely according to where the 'goalposts' are placed.

It is believed that symptomatic control and measuring quality of life outcomes should be considered highly relevant, rather than focusing purely on a single surrogate marker of outcome such as AHI.

S26

Surgery for paediatric OSA

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Adenotonsillectomy is widely regarded as the gold standard treatment in paediatric patients with sleep disordered breathing and adenotonsillar hypertrophy. Controversies such as the role of pre and post polysomnography, persistent OSA after surgery, and the role of subsequent treatments such as CPAP versus further surgery will be presented as discussion points.

S27

Circadian rhythms and ambient light

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This is the first talk in a symposium session designed to address the known factors that precipitate and perpetuate circadian rhythm sleep disorders, such as delayed sleep phase disorder. The master circadian pacemaker in humans is located in the suprachiasmatic nuclei in the hypothalamus and it regulates circadian rhythms throughout the body. In most people, the endogenous circadian clock has a period ('tau') slightly > 24 h, which produces a natural tendency to phase delay or drift slightly later each day (on average about 12 min per day). The timing of the endogenous circadian clock is also regulated by external light. Light exposure via intrinsically photosensitive ganglion cells in the retina phase shifts the circadian clock daily. As illustrated by the light phase response curve, light in the subjective morning shifts the circadian clock earlier ('phase advance') and light in the subjective evening shifts the circadian clock later ('phase delay'). When the circadian clock is optimally timed relative to the desired sleep/wake schedule ('circadian alignment'), it promotes wakefulness during the desired wake time and promotes sleep during the desired sleep times. However, when the circadian clock is mistimed relative to the desired sleep/wake schedule ('circadian misalignment'), wakefulness can be significantly impaired and sleep significantly disturbed. Notably, even subtle shifts in circadian alignment can affect wellbeing. The general western trend towards later and shorter sleep episodes is typically a result of delayed bedtimes, with relatively stable wake times due to employment and/or childcare responsibilities. Regular late bedtimes lead to repetitive daily exposures to ambient light in the evening. Recent evidence indicates this light exposure in the home environment can negatively impact optimal circadian alignment in two ways. First, repetitive exposure to this evening light can significantly phase delay the circadian clock, thereby adding to the pre-existing tendency to drift later (phase delay). Second, this evening light can also reduce corrective phase advances to morning light, which many of us require to maintain optimal circadian misalignment. Thus the ambient evening light that many of us receive every night may predispose many of us to the negative consequences of circadian misalignment. Dr Burgess is supported by National Institutes of Health grant R01 HL083971.

S28

Melatonin and melatonin agonists: implications for treatment of DSPD

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Delayed Sleep Phase Disorder (DSPD) is a Circadian Rhythm Sleep Disorder characterised by markedly delayed sleep onset and wake times that are intractably later than desired. Our studies and those of others demonstrate that melatonin treatment may shift the timing of the endogenous circadian pacemaker, with the direction of the shift (advance or delay) depending on the time of administration. Melatonin also promotes sleep and increases sleep propensity, in particular during the biological day when endogenous melatonin is low. These properties have led to the suggestion that melatonin and its agonists could provide an effective treatment approach for DSPD. A significant barrier to developing and testing effective treatments for DSPD has been the misclassification of patients; misdiagnosis of DSPD as insomnia, and failure to assess endogenous circadian phase to support diagnosis and inform treatment. We are developing an online questionnaire to identify those at risk for DSPD, and to assess the impact of DSPD symptoms on self-reported absenteeism and functioning in work or school, social and family life. We screened 13 844 individuals recruited by an internet survey provider. Of the eligible participants, 5.1% were at high risk for DSPD and a further 5.3% were at high risk for DSPD and/or insomnia. Compared to those not at high risk for DSPD, those at high risk showed significantly higher odds of missing school or work and having reduced productivity at school or work. High risk DSPD was also associated with increased odds of at least moderate level of disruption to work or school, social or leisure activities and family life or home responsibilities. These findings suggest that DSPD risk can be initially assessed by an online