Thorax

Editorials

Who should receive treatment for sleep apnoea?

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The presence of obstructive sleep apnoea is a common finding, occurring in 5–20% of adult men, although only about one in five of these individuals will have any symptoms potentially attributable to this sleep apnoea (sleep apnoea syndrome).[1] Conventionally, sleep apnoea has been quantified in terms of the respiratory disturbance during sleep, with an arbitrary number of apnoeas or hypopnoeas per hour (usually 15) required to make the diagnosis of sleep apnoea, the apnoea/hypopnoea index (AHI).[2 3] Unfortunately this index correlates poorly with subjective or objective measurements of sleepiness[4] and, as this is the predominant symptom of sleep apnoea, such measurements are not ideal for defining sleep apnoea and for grading its severity. As our understanding of the sleep apnoea syndrome has improved, it is recognised that this condition has a wide spectrum of severity and that it is no longer appropriate to select an arbitrary AHI value to define the severity of sleep apnoea and therefore the suitability for treatment. Any clinical management decisions for patients at the two extremes of this spectrum are not usually difficult, but there is some uncertainty knowing how best to treat the large number of patients in the middle or towards the lower end of it. The paper by Engleman *et al*[5] in this issue of *Thorax* shows that some patients in the traditionally classified "mild sleep apnoea" group (AHI 5–15) do benefit from treatment with nasal continuous positive airways pressure (nCPAP), but there is difficulty in identifying which patients in this "mild" group will best respond and comply with treatment thereafter. This editorial discusses the uncertainties in this area.

The predominant symptom of sleep apnoea is excessive daytime sleepiness due to recurrent arousal from sleep[6] causing sleep fragmentation. This symptom is the main indication for treatment with nCPAP and it responds excellently to treatment.[7 8] The most important stimulus to this recurrent arousal from sleep is increasing respiratory effort[9] which occurs as a consequence of the upper airway obstruction. It is now recognised that increases in upper airway resistance alone, without an apnoeic or hypopnoeic event, can cause recurrent arousal from sleep.[10] We also now know that not all apnoeas cause the same degree of sleep disruption when assessed from the electroencephalogram (EEG).[11] These last two points are likely to be a major part of the explanation for the poor correlation between respiratory disturbance indices and daytime symptoms. Given these problems it seems, in theory, logical that our assessment of these patients should be aimed at quantifying the recurrent arousal from sleep, rather than respiratory disturbance as it is currently measured. This can be done in several ways – for example, by monitoring body movements.[12] Alternatively, the EEG can be manually scored for visible changes which reflect brief arousals from sleep (microarousals).[13] Unfortunately, previous attempts to correlate microarousals with daytime function in patients with sleep apnoea have shown that they are little or no better than respiratory indices such as AHI.[4] The paper in this issue by Engleman *et al*, however, shows that in the "mild" group the microarousal index is the factor best able to differentiate poor nCPAP compliers from better compliers.[5] This is likely to be due to episodes of upper airway resistance during sleep causing sleep disruption but remaining undetected by standard respiratory monitoring. A very different approach to assessing sleep disruption uses the rises in blood pressure that occur with each arousal from sleep. Such "autonomic arousals" can occur in response to stimuli even without changes in the EEG,[14] and there are some preliminary data to suggest that these may also cause daytime sleepiness.[15] However, at present there is insufficient evidence to decide which measure of sleep apnoea and its consequent sleep disturbance is the best guide to determine the severity of sleep apnoea and thus to assess the likely benefit from treatment. This is clearly an important area of research which is being studied in several laboratories.

While new approaches to the assessment of the sleep apnoea syndrome are being explored, we need to accept that treatment decisions for these patients should not be based solely on the results of sleep studies. We need to adopt an approach similar to the management of asthma, for instance, where a combination of symptoms and objective measurements are used to initiate and vary treatment. For example, it would be correct to increase the dose of inhaled steroid of an asthmatic patient complaining of nightly episodes of bronchospasm even if the morning peak flow dip did not reach an arbitrary threshold, and to reduce it again if it made no difference. In the same way patients who complain of daytime sleepiness with perhaps only mild objective respiratory disturbance on the sleep study (such as loud snoring and restless sleep) should be offered a trial of nCPAP to see whether it improves their daytime symptoms sufficiently to be worth continuing the treatment. We have shown that pretreatment subjective sleepiness using the Epworth Sleepiness Scale (ESS) and Sa_{O_2} dip rate in a sleep clinic population does not correlate with acceptance of long term treatment but improvement in ESS on nCPAP does,[16] which suggests that we cannot identify with confidence all those patients who will respond to nCPAP from their pretreatment investigations. Treatment obviously needs to be appropriate to the severity of the symptoms, and if patients do not have a real problem with daytime sleepiness then they are unlikely to be prepared to tolerate the treatment long term.[17] With this approach patients will select for themselves whether or not they stay on the treatment indefinitely.

There may be some patients with sleep apnoea who have improvement in their daytime symptoms while on nCPAP but in whom these improvements do not outweigh the disadvantages and unpleasantness of such a cumbersome form of treatment. Newer and simpler treatments for sleep apnoea are being developed – for example, removable mandibular advancement splints which aim to keep the upper airway clear by holding the tongue and/or jaw forward during sleep.[18] Although they are unlikely to be satisfactory in severe sleep apnoea, there is some preliminary evidence that they help in mild to moderate cases and should enable us to offer a more flexible treatment approach than has previously been available.[19]

The main indication for treatment of the sleep apnoea syndrome is therefore the disabling symptom of excessive daytime sleepiness and this usually responds well to treatment. The relationship between daytime sleepiness and objective severity of sleep apnoea is complex and traditional indices of sleep apnoea severity do not correlate well with symptoms. Future improvements in our understanding of sleep fragmentation may enable us to predict from sleep study parameters which patients will best respond to treatment. While research into these areas is continuing, a sensible and practical approach to the management of sleep apnoea is to offer a trial of nCPAP treatment to patients who have both an objective sleeping respiratory disturbance due to upper airway obstruction and symptomatic sleepiness. The decision on long term treatment is then usually made by the patient on the basis of improvement in daytime symptoms on nCPAP.

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