



CLINICAL REVIEW

# The management of simple snoring

Paul Counter<sup>a,\*</sup>, Janet A. Wilson<sup>b,1,2</sup>

<sup>a</sup>*Department of Otolaryngology, Head and Neck Surgery, Sunderland Royal Hospital, Kayll Road, Sunderland SR4 7TP, UK*

<sup>b</sup>*Head and Neck Surgery, University of Newcastle, Freeman Hospital, Newcastle upon Tyne NE7 7 DN, UK*

## KEYWORDS

Snoring; Diagnosis;  
Differential therapy

**Summary** Simple snoring is a very common problem that presents a number of different challenges. The initial difficulty is in confirming the presence of snoring, next one must exclude any other nocturnal respiratory pathology and finally a decision as to an appropriate treatment must be made. There are many different ways of achieving these objectives, but no one-way has a clear advantage in terms of both accuracy and cost effectiveness. In this review the authors do not intend to give a didactic method for the management of simple snoring but to discuss the pros and cons of various different options in order to help physicians make a choice based on local priorities.  
© 2004 Elsevier Ltd. All rights reserved.

Snoring is not a new phenomenon. It has been recognised as a social problem for thousands of years, and is regularly referred to in classical literature. In these early references there is often little distinction between the words for snoring or for sleep, which may well have the same origin in ancient Greek. Some of the predisposing factors to snoring were also frequently noted in ancient literature, but probably none more succinctly than in Virgil's Aeneid where three—obesity, alcohol and position—are mentioned in a single line

Who, gorged, and drunk with wine, supinely snore.

A potential, albeit radical, solution is also mentioned

With his drawn sword... him and his sleeping slaves he slew.

The treatment of snoring has, happily, moved on somewhat since Virgil's day.

The recognition of sleep apnoea, of which snoring is the cardinal symptom, has led to one of the major priorities in the modern treatment of snoring, the exclusion of pathological respiratory disturbance. It is still not entirely clear whether simple snoring in itself is the direct cause of any adverse health consequences.

The prevalence of snoring in the population of the UK has been estimated at around 25-40%.<sup>1</sup> In mid 2002 the population of the UK stood at 59,206,700. Eighty percent of these were over the age of 16 and approximately 70% were cohabiting. This means that approximately 10 million UK adults are likely to experience problems because of snoring. The equivalent figures for the United States are a population of 285 million and approximately 47 million sufferers from snoring. Obviously a large proportion of these people will not be sufficiently disturbed by snoring to seek medical

\*Corresponding author. Tel.: +44-7808-766409.

E-mail addresses: [paul@paulcounter.demon.co.uk](mailto:paul@paulcounter.demon.co.uk) (P. Counter); [j.a.wilson@ncl.ac.uk](mailto:j.a.wilson@ncl.ac.uk) (J.A. Wilson).

<sup>1</sup> The authors declare no conflict of interest.

<sup>2</sup> Tel.: +44-191-284-3111x26628; fax: +44-191-223-1246.

### Nomenclature

SDB	sleep disordered breathing
OSAHS	obstructive sleep apnoea hypopnoea syndrome

$P_{\text{crit}}$	critical closing pressure of pharynx
$R_{\text{up}}$	flow resistance upstream of pharynx
UARS	upper airways resistance syndrome
ESS	Epworth sleepiness score

help and indeed it is not clear from current research what proportion of people would seek treatment if it were both successful and easily available.

The intention behind this review is to summarise the current state of knowledge in diagnosing simple snoring and to examine the currently available solutions.

### Definition of simple snoring

A useful working definition of snoring is the production of sound by of the upper aerodigestive tract during sleep. The diagnosis of simple snoring, however, is one of exclusion. It requires that the presence of any other nocturnal respiratory pathology be excluded. It is therefore important for any physician treating snoring to understand the pathophysiology behind sleep disordered breathing (SDB).

Snoring is part of the spectrum of SDB, from obstructive sleep apnoea/hypopnoea syndrome (OSAHS) at one end to simple snoring at the other. The entire spectrum is characterised by changes in the physical conformation, structural properties and neuromuscular function of the pharynx. The spectrum has also been characterised in respect of the pressure required to collapse the upper airway.<sup>2</sup> Thus, despite qualitative differences in pathology, the pathophysiological difference between simple snoring and OSAHS may be merely quantitative.

A mathematical model<sup>3</sup> of snoring has been created in terms of airflow through an elastic tube. If the geometry, elasticity, resistance and flow are adjusted correctly then the tube can be made to open and close repetitively. This leads to production of sound that is acoustically very similar to snoring. If the parameters are altered further complete collapse of the tube can be created, simulating apnoea. Unfortunately this technique does not reliably locate exact level of sound production within the pharynx, as the airway instability may occur at any level. Similarly, clinical observations<sup>4</sup> confirm vibration anywhere from the soft palate to the epiglottis. This mathematical model does, however, suggest the mechanism by which variations in muscle tone, airway dimensions and route of breathing (nasal or oral) may cause snoring.

Two of the most important parameters in determining where on the spectrum of SDB a patient lies are the pressure at which the pharynx collapses ( $P_{\text{crit}}$ ) and the resistance to airflow upstream of the pharynx ( $R_{\text{up}}$ ).<sup>5</sup> Variations in  $R_{\text{up}}$  have a profound effect on the pressures generated within the pharynx during respiration.

In normal sleep resting muscle tone acts against gravity to keep the airway patent, however a relative muscular hypotonia still occurs in the upper airway in comparison to the awake state. The muscle tone increases somewhat during maximal airflow, helping to maintain airway patency. If the resting muscle tone is too low or the external forces too great this can lead to partial collapse of the upper airway. In turn this provokes increased inspiratory negative pressure, further exacerbating the collapse. Studies comparing snorers and non-snorers have confirmed that snorers generate more negative inspiratory pressures,<sup>6</sup> prolonged inspiratory time,<sup>6</sup> and limitation of respiratory flow.<sup>7</sup> These changes are pathological exaggerations of the normal respiratory changes in sleep. They lead to unstable, turbulent airflow within the upper airway and tissue vibration, causing the production of noise; 'the snore'. It has been consistently observed that snoring is worse in slow wave sleep and during the early part of the night, whilst it is relatively rare during REM sleep.<sup>8</sup>

### Clinical significance of snoring

It has been clearly shown that systemic blood pressure fluctuates transiently with snoring.<sup>9</sup> The search for a link between snoring and hypertension, cerebrovascular disease and coronary artery disease has yielded conflicting results. None of the studies linking simple snoring and cardiovascular disease included polysomnography (PSG).<sup>10</sup> Those studies that have excluded patients with sleep apnoea found no such association.<sup>11</sup> In other words, uncomplicated snoring appears to have no long-term effect on the coronary, systemic or cerebral circulation.

There is unresolved debate as to whether snoring alone, without significant sleep apnoea, can interfere with daytime functioning. It is now known that

in some individuals the resistance to inspiration increases repetitively, leading to brief arousals without any significant arterial hypoxaemia—the Upper Airways Resistance Syndrome (UARS).<sup>12</sup> Once sufferers from UARS are identified and excluded from the population of nonapnoeic snorers, it becomes even less clear whether snoring itself has any fragmentary effect on sleep.

There is some evidence that academic performance may be affected by snoring,<sup>13</sup> but there are problems with the exclusion of OSA in the study populations. This point cannot therefore be considered proven.

In summary then, it has not been clearly shown that there is any significant long-term medical effect from snoring. However, the fragmentation of sleep that may be caused, both by the snoring and by the bed partner's attempts to stop it, need further investigation.

## Assessment of the snoring patient

The assessment of a patient presenting with snoring is mainly directed at three things:

1. Confirmation of the presence of snoring.
2. Excluding other causes of sleep disturbance, e.g. OSAHS.
3. Identifying the optimum treatment.

### Confirming and quantifying the presence of snoring

Simple snoring is a very unusual medical problem in that it is most commonly not complained of by the patient, but by their bed partner. It is therefore important that the patient be seen along with their partner. Failing this it is very useful to ascertain if anybody else, e.g. family or friends, has noticed the snoring. The partner may inform on the following:

- frequency of snoring (i.e. how many times per night),
- position sensitivity (i.e. supine, side or prone),
- witnessed apnoeic episodes,
- duration of disruptive snoring (i.e. over how many years),
- degree of disruption (i.e. number of nights per week sleeping separately or disturbed).

Patients often attend clinic without their partner, which means much of this information must be reported second hand. Unfortunately there is a large degree of disagreement between partner

and self-reporting of snoring<sup>14</sup> (Cohen's kappa  $-0.01-0.59$ ). This is not merely due to the long-term emotional strain of sleeping with a snorer as another study comparing a sleep lab technician with the snorer<sup>15</sup> also showed very poor agreement.

The patient may be asked to supply a tape recording of their snoring. For example, prior to surgery, to avoid operating on a 'snorer' when in fact the problem lies with an insomniac bed partner being disturbed by essentially normal nocturnal breathing noise. The recording may be on a sound activated dictaphone by the side of the bed, but it is uncertain at what volume the recorder will be triggered. More sophisticated, purpose designed instruments have been developed, which are somewhat simpler for the patient to operate and give a more consistent result, along with other analysis of the snoring noise. Unfortunately, however sophisticated the measuring device, the sound intensity of the snores is probably not as important as the amount of social disturbance caused by the snoring.

The complex social impact of snoring is difficult to quantify. It is usually addressed by means of questionnaires directed at both the snorer and their bed partner. Most questionnaires have been designed to help exclude OSAHS, and are therefore not particularly useful for quantifying the severity of the snoring. The actual problems of which snorers complain centre around disturbing others' sleep, poor sleep quality, lethargy and sore throat.<sup>16</sup> This is in contrast to otolaryngologists' views on snoring, which tend to be more focused on relationship difficulties, poor sleep quality, daytime somnolence and, finally, disturbance of others' sleep. A recent, specifically targeted questionnaire<sup>17</sup> is therefore helpful and will be useful for future comparisons of snoring outcome.

### Excluding other causes of sleep disturbance

The concern foremost in most physicians' minds when presented with any case of apparently simple snoring is that this may actually be the presenting symptom of more severe SDB, i.e. OSAHS or UARS. The next step in management is therefore aimed at excluding this.

#### Clinical assessment

Although history and physical examination are unreliable in diagnosing SDB,<sup>18</sup> the following risk factors must be assessed:

- *Male sex.* This has been very clearly shown in almost all epidemiological studies. The reasons are not entirely clear but probably relates to differences in laryngo-pharyngeal anatomy

and the arrangement of body fat. It is also possible that women perceive and report snoring more frequently than men.<sup>19</sup>

- **Obesity.** Obesity has also been clearly shown to be linked to SDB. It seems likely that this is in part a proxy marker for neck circumference,<sup>1,20</sup> although the two do have a small degree of independent effect. Therefore a history of a recent change in weight and/or collar size, particularly if coincident with a worsening of snoring, is important.
- **Alcohol/tranquillisers.** Most patients are well aware of the association between snoring and sedatives. This is due to relaxation of the pharynx and therefore increased compliance.
- **Smoking.** This association has been noted in some epidemiological studies,<sup>1</sup> but not clearly explained. It is possibly due to mucosal oedema and therefore nasal and pharyngeal narrowing.
- **Nasal obstruction.** As mentioned above, there is evidence that, for some snorers, nasal obstruction is a critical factor.<sup>3,5</sup> Unfortunately there is no way of determining who falls into this subgroup. Figures for resolution of snoring following septal surgery vary between 69<sup>21</sup> and 15%.<sup>22</sup> Resolution has even been reported following sinus surgery alone.<sup>23</sup> Unfortunately none of these studies are particularly conclusive and therefore the real figure is still unknown.
- **Daytime sleepiness.** This obviously correlates with sleep disturbance, but unfortunately also with a number of other non-sleep related problems, e.g. depression, hypothyroidism, etc. This is often quantified by means of the Epworth Sleepiness Scale (ESS).<sup>24</sup> The ESS also helps monitor changes over time within the same patient.
- **Witnessed apnoeas.** As mentioned above in the commentary on partner reported snoring, this is notoriously unreliable as marker for excluding significant apnoeic events, although partner reports of its presence can increase the physician's suspicion of OSA.
- **Abnormal nocturnal motor activity.** This includes sleepwalking and the nocturnal restlessness of periodic limb movement disorder.
- **Other nocturnal events.** Night sweats, reduced libido and enuresis are all more common in SDB.

Different combinations of these symptoms, along with various others, have been used in scoring systems to predict the likelihood of OSAHS/UARS. None has proved particularly useful in isolation, although when combined with ESS and body mass index (BMI) they may be of some value.<sup>20</sup>

Examination is nonspecific, but directed at the head and neck. The following should be included:

- BMI and neck circumference have been linked strongly to OSA.<sup>1</sup>
- The nose should be examined to exclude causes of obstruction, e.g. polyps, septal deviation, turbinate hypertrophy.
- Direct examination of oral cavity and oropharynx.
- Laryngoscopy, with a mirror or endoscope, will help exclude any laryngeal pathology as a cause of snoring. Approximately 3 in 200 of all patients with OSAHS will have such a lesion.<sup>25</sup> The proportion in snorers is not known.
- Blood pressure: as uncontrolled hypertension may be a marker for OSAHS.

### Investigation

The gold standard for excluding other causes of SDB remains multichannel PSG. Debate continues about which variables need to be measured during PSG, and even if it is the gold standard at all.<sup>26</sup> It is also the most expensive. PSG on snorers with no additional features suggestive of OSAHS will be normal in up to 80% of studies.<sup>27,28</sup> As healthcare costs rise across the globe this has become more of an issue. Current guidelines from the American Thoracic Society state that snoring alone is not an indication for a sleep study. The Thoracic Society of Australia and New Zealand, the American College of Chest Physicians and the Association of Sleep Disorders Centres have echoed this in slightly less categorical form. The question then is who should have a PSG, or whether PSGs are necessary at all. The answer to this will probably depend on the availability of suitable alternatives and the economic situation where the physician is working. A PSG may well be performed if enough of the above factors have been found to raise the possibility of another cause of SDB, as this is commonly perceived to be the most reliable test. There is some evidence that this might not actually be true and that home based testing could be just as accurate. Arguments against it are:

- Although it is the only reliable way of ensuring that the patient slept during the test, only about 3% of patients with OSAHS sleep less than three hours and repeat PSG has not changed the diagnosis in any of these.
- PSG is the only reliable way of determining the apnoea/hypopnoea index, but there is no evidence that this is a good predictor of morbidity in OSAHS. Better predictors of

response to CPAP therapy are nocturnal oxygen saturation and movement during sleep.<sup>29</sup>

- PSG cannot reliably exclude narcolepsy.<sup>30</sup>
- There is some confusion over whether periodic limb movement disorder is a significant cause of daytime sleepiness.<sup>31</sup>

An alternative is to use home studies in varying degrees of complexity. This has a number of advantages

- Most patients sleep better at home.
- There is no restriction on physical space for the tests, although there is obviously a financial restriction on the amount of equipment any one department can afford. This will tend to keep the waiting time much shorter.
- There is no capital cost for the hospital rooms, thus making the home studies significantly cheaper.<sup>26</sup>

There are many different sensors that may be used in a home study. The arguments as to which are best able to exclude OSAHS is still very active at present. A useful summary of the argument can be seen in reference 26. It does need to be borne in mind that in this group of patients we are not trying to make an accurate diagnosis of the degree of OSAHS, but to rule it out as a cause of the patient symptoms. This probably makes home studies an even more attractive option.

It is entirely appropriate to use home studies as a means of identifying which patients need a full PSG.

### Deciding on a treatment option

Once simple snoring has been confidently diagnosed, the choice of treatment then needs to be addressed. The initial step in this is to try to localise the level at which the snoring noise is being generated. The commonly used ways of doing this are:

- *Fibre-optic nasal endoscopy with Müller's manoeuvre.* There are a number of drawbacks to this test. The patient is awake and therefore the pharyngeal muscular tone is much higher than in sleep; inspiratory effort may vary from patient to patient, thus varying the degree of collapse;<sup>32</sup> there is a lot of inter-observer variation in determining the level of collapse.
- *Sleep nasendoscopy.* The technique of assessing the level of sound production/pharyngeal collapse by fiberoptic nasendoscopy while the patient is under sedation was described in 1991.<sup>4</sup> This technique is reasonably reliable at

demonstrating the site of snoring/obstruction.<sup>4</sup> There is little debate that the cross-sectional area of the pharynx, can be estimated to within 10% by this method, when linked to a computer.<sup>33</sup>

The difficulty lies in knowing whether any observed snoring source and/or reduction in pharyngeal diameter under sedation also occur during natural sleep—more people may snore/experience apnoeas under sedation than in natural sleep.<sup>34</sup> This picture is further complicated by the unstandardised sedation protocol, and evidence that most palatal surgery failures occur in any case at the level of the palate.<sup>35</sup> In other words the poor prediction of outcome of laser uvulopalatoplasty<sup>36</sup> by sleep nasendoscopy probably reflects the inherent limitations of both the procedure and the test.

- *Upper airway pressure recordings during sleep.* It has been shown that using several pressure transducers within the oesophagus during sleep produces very similar results to a full PSG.<sup>37</sup> It is also significantly cheaper. There is, however, some evidence that the invasive nature of this test influences the duration and stage of sleep. Also, only the lowest limit of the obstructed airway is determined and the method may not be able to localise the level of snoring as opposed to the airway collapse.
- *Radiography.* Cephalometry, somnoflouroscopy, CT and MRI all provide some information about the upper airway, but suffer from limited availability, high cost, high radiation exposure or lack of dynamic imaging. They are all therefore used very rarely in the assessment of snoring.
- *Acoustic analysis.* This technique has come to prominence fairly recently. It is based on the premise that snoring noises generated at different levels have different acoustic characteristics. If this is true, it should be possible to mathematically analyse the sound produced in a snore and from this derive the level at which the sound has been produced. This has been done by comparison with sleep nasendoscopy and a difference has indeed been found.<sup>38</sup> Unfortunately acoustic analysis, like sleep nasendoscopy appears a poor predictor of the outcome of snoring surgery. It has also been suggested that acoustic analysis can help diagnose OSAHS.<sup>39</sup> These techniques seem to hold some promise as a screening test, but still require further refinement.

Given the present level of uncertainty in the prediction of the level at which snoring is produced,



it is not unreasonable to perform none of the above techniques, but rather to base decisions on a 'best guess' derived from the history and examination, in conjunction with patient preference.

## Treatment of snoring

- Patient administered interventions—weight loss, posture adjustment, over-the-counter medication, nasal dilators, stopping smoking.
- Nonsurgical physician interventions—tongue retaining devices, mandibular advancement devices, CPAP.
- Surgical interventions—nasal surgery, palatal shortening surgery, intra-palatal surgery, bariatric surgery.

### Patient administered interventions

The partners of some snorers may give a very definite history of snoring only being a problem in one position (typically supine). This has been confirmed by nocturnal sound intensity recordings.<sup>40</sup> A number of different interventions are available to prevent supine posture, including bolsters for the bed, specially designed pillows and hard objects attached to the back of nightclothes. Indeed, most patients who present to a secondary care physician have already tried such fairly basic measures themselves, although there is a small group in whom they may still succeed.<sup>41</sup> The same argument applies to the use of homoeopathic tablets. These have been shown to be effective in reducing snoring in approximately 80% of people,<sup>42</sup> versus 45% of a placebo group. Success rates of around 30% have been obtained for oropharyngeal sprays.<sup>43</sup>

Weight loss is very effective in both snoring and OSAHS.<sup>40</sup> This is not surprising given the strong epidemiological links between weight, and neck circumference and snoring.<sup>1</sup> Despite short-term successes, however, weight loss is very rarely achieved by patients in the long-term. That said, the success rate of surgical interventions is much lower in obese patients and therefore some degree of weight loss is often viewed as a prerequisite for surgical intervention. Alcohol reduction is clearly advisable, but the exact benefit of stopping smoking is currently unclear.

The occasional snorer in whom nasal obstruction is a critical factor is hard to identify, but, if found,

may respond to a nasal splint. The first of these was described in 1905, but more modern versions are now available, both internal reusable and external disposable versions. The internal dilators have been shown to significantly reduce nasal resistance,<sup>44</sup> snoring volume<sup>45</sup> and snoring frequency.<sup>46</sup> External dilators have a similar effect.

### Nonsurgical physician interventions

The most popular nonsurgical therapy is the mandibular repositioning device (MRD), ranging from the simple 'boil and bite' to the more complicated, dentally fitted, patient adjustable devices. All are designed to advance the mandible a varying degree relative to the skull. A typical value would be approximately 75% of maximum advancement or around 5-6 mm. There is no clear advantage of one design over another in terms of efficacy, but the different designs offer advantages over each other in terms of cost, ease of fitting, longevity, comfort and adjustability. The right combination of these will vary from patient to patient. There are a large number of studies looking at their application in OSAHS, but well designed studies including data on snoring are less common.

One study<sup>47</sup> has shown that, in patients with an apnoea hypopnoea index <10, use of an MRD reduces the proportion of snores greater than 50 dB from 28 to 9% and the number of snores per minute from 8 to 6. A further study<sup>48</sup> compared an MRD with advancement to one without. Advancement had a significantly greater effect on snoring with a self-reported reduction in snoring volume in 84% and frequency in 76% of patients. A number of crossover designs between MRDs and various other treatments have been performed. In summary, in comparison to placebo, MRDs seem to be significantly more effective. In comparison to somnoplasty, there was no significant difference, but the numbers in this study were very small (10 in each arm).<sup>49</sup> In comparison to CPAP MRDs are significantly less effective.<sup>50</sup> One uncontrolled study suggested that the overall effectiveness of MRDs may be improved by fitting a device that allows the patients to adjust the degree of advancement,<sup>51</sup> but the remarkably high overall success rate (96%) may reflect the method of outcome assessment more than the success of treatment.

The side effects of MRDs include excess salivation, xerostomia, temporo-mandibular joint pain, dental pain, myofascial pain and bite change. One or other of these effects will occur in around 50-80%<sup>52</sup> of patients. The vast majority settle within four weeks, but overall compliance is around 50-75%.<sup>48,52</sup> Overall, MRDs are effective in between

50 and 75% of patients. They rarely eliminate snoring (about 5%), but they do reduce the frequency, intensity and duration of snores. The effect of MRDs is complex—the obvious effect of an increased antero-posterior diameter of the oropharynx may be augmented by increased genioglossus activity.<sup>53</sup>

Tongue retaining devices similarly work by holding the anterior part of the tongue forward by the application of a soft cup to it. These have not achieved a great deal of popularity and therefore studies are limited to anecdotal evidence. The only patients in whom they would seem to offer any advantage are those with insufficient dentition to retain an MRD.

Nasal CPAP is undoubtedly very effective, reducing the number of snores per hour from 154 to 3, but the well-known problems with compliance due to the discomfort of wearing the device, and to some extent the noise of the machine itself lead, to a very low compliance when used for snoring alone. One study indicated that of 59 patients offered CPAP for snoring, only 11 took up the offer and after six months they were all using it for fewer than three hours per night.<sup>54</sup> Although CPAP is approximately 100% effective, therefore, approximately 70% of patients prefer the slightly less effective MRD, compared with 25% preferring CPAP.<sup>48</sup>

### Surgical interventions

Surgery for nasal obstruction has similar drawbacks to nasal dilators, in that there is a small group of patients in whom this is undoubtedly effective, but there is no way of identifying prior to surgery whether any individual patient is in this group.

The pioneering operation for OSAHS, and by extension snoring, was the uvulopalatopharyngoplasty (UPPP). This was first described in 1964<sup>61</sup> and then popularised in the West in 1981<sup>62</sup>. There are many studies looking specifically at the question of the response of snoring to UPPP. A useful summary of palatal approaches up to 1996 can be found in reference 40. There have been few studies adding additional quality to the volume of information after that date although an objective measure of outcome in a small group of patients<sup>55</sup> confirms the overall findings that snoring is indeed reduced in between 75 and 100% of patients in the short-term. The long-term efficacy would appear to fall to around 50%.<sup>56</sup> This dramatic decline may represent long-term changes in the pharynx following surgery, or perhaps more likely, changes in self-reporting over time as the placebo effect diminishes. When measured objectively the snoring volume does not, however, seem to change significantly over time following UPPP.<sup>55</sup>

Laser assisted uvulopalatoplasty was first described by Kamami in 1990.<sup>57</sup> It has been widely used in several modifications ever since. Unfortunately at that stage they all suffered from similar problems, which were that no objective measure of snoring was employed and in some cases OSAHS was not excluded. Since that date objective or well-validated studies have been published,<sup>58</sup> which confirm success rates of around 50-95% in the short-term. Over the long-term their success rates declined to 45-75%.<sup>56</sup> It is worth noting that there is a marked disparity between subjective decline in benefit over a five-year period and maintenance of a small, but statistically significant fall in snoring volume (4 dB). This is true both for LAUP and UPPP.<sup>55</sup> This same study also highlights extremely well the poor correlation between objective decrease in snore intensity and subjective improvement. This poor association has been confirmed in other studies.<sup>59</sup>

Intra-palatal surgery has recently become popular, due to its markedly lower morbidity<sup>60</sup> in comparison with the more traditional types of surgery. Indeed, because of this lower morbidity, it is commonly performed in the outpatient setting, under local anaesthetic. A number of different techniques of performing this have been developed, including monopolar diathermy, bipolar diathermy and coblation. These techniques have also been applied to the tongue base. The relatively recent introduction of these techniques means that there are few studies looking at their success specifically in snoring, those that there are suggest a success rate of around 30-60%<sup>60</sup> in the short-term, i.e. significantly less than for the more destructive forms of surgery. The best results appear to be in those with a BMI <25.<sup>60</sup> The long-term results also declined significantly and remain approximately half those of UPPP/LAUP, at around 20%. Despite these significantly lower success rates there are two significant advantages of this type of surgery:

- It is repeatable, although exactly how many times it may be repeated has not yet been determined.
- If it does not prove to be successful then UPPP/LAUP may still be performed.

### Practice points

- A large number of available treatments are effective in different sub-groups of snorers.
- No reliable prediction of the individual success is available.

- No economically attractive method to reliably exclude obstructive sleep apnoea exists, although increasing work on domiciliary studies is in progress.
- Full PSG should be reserved for those with increased suspicion of OSAHS, either clinically or on the basis of a screening test.
- There is also no gold standard for the measurement of snoring, and an extremely poor correlation between the subjective and objective assessments.
- Both are advised in any future studies on snoring.
- The selection of an appropriate treatment for any given patient remains problematic. Snoring is probably often multilevel in origin and fixing one level will merely reveal sound production at another.
- Identification of the snoring level is not enough—are failures due to a failure of the assessment procedure, or of the therapy?
- There is a major placebo effect, and so uncontrolled trials are of limited value.
- There have been very few studies of the effects of snoring on his/her partner, which is somewhat surprising, given that these are the people who actually present with the problem.

### Research agenda

- Influence of partner's noise tolerance during sleep on perceived snoring intensity.
- Further examining the best techniques for home-based exclusion of OSAHS.
- Agreement on a standard technique for measuring subjective snoring levels.

### References

- \*1. Stradling J, Crosby J. Predictors and prevalence of obstructive sleep apnoea and snoring in 1001 middle-aged men. *Thorax* 1991; **46**: 85–90.
2. De Backer W et al. Intraluminal pressures during central apnea compared to critical closing pressures. *Sleep Research* 1996; **25**: 231.
3. Gavriely N, Jensen O. Theory and measurement of snores. *Journal of Applied Physiology* 1993; **74**: 2828–2837.
- \*4. Croft C, Pringle M. Sleep nasendoscopy: a technique of assessment in snoring and obstructive sleep apnoea. *Clinical Otolaryngology* 1991; **16**: 504–509.
5. Schwartz A-R et al. Effect of positive nasal pressure on upper airway pressure–flow relationships. *Journal of Applied Physiology* 1989; **66**: 1626–1634.
6. Stoohs R, Guilleminault C. Snoring during NREM sleep: respiratory timing, esophageal pressure and EEG arousal. *Respiratory Physiology* 1991; **85**: 151–167.
7. Skatrud J, Dempsey J. Airway resistance and respiratory muscle function in snorers during NREM sleep. *Journal of Applied Physiology* 1985; **59**: 328–335.
8. Perez-Padilla J, West P, Kryger M. Snoring in normal young adults: prevalence in sleep stages and associated changes in oxygen saturation, heart rate and breathing pattern. *Sleep* 1987; **10**: 249–253.
9. Lugaresi E et al. Breathing during sleep in man in normal and pathological conditions. *Advances in Experimental Medicine and Biology* 1978; **99**: 35–45.
10. Waller P, Bhopal R. Is snoring a cause of vascular disease: an epidemiological review. *Lancet* 1989; (1): 143–146.
11. Schmidt-Nowara W et al. Snoring in a Hispanic-American population: risk factors and association with hypertension and other morbidity. *Archives of Internal Medicine* 1990; **50**: 597–601.
- \*12. Exar E, Collop N. The upper airway resistance syndrome. *Chest* 1999; **115**(4): 1127–1139.
13. Urschitz M et al. Snoring, intermittent hypoxia and academic performance in primary schoolchildren. *American Journal of respiratory and critical care medicine* 2003; **168**: 464–468.
14. Wiggins CL et al. Comparison of self and spouse reports of snoring and other symptoms associated with sleep apnea syndrome. *Sleep* 1990; **13**: 245–252.
15. Hoffstein V, Mateika S, Anderson D. Snoring: is it in the ear of the beholder? *Sleep* 1994; **17**: 522–526.
- \*16. Scott S et al. A comparison of physician and patient perception of the problems of habitual snoring. *Clinical Otolaryngology* 2003; **28**(1): 18–21.
17. Douglas S et al. Development of a snoring symptoms inventory. *Otolaryngology, Head and Neck Surgery* 2003; **129**: 200.
18. Viner S, Szalai M, Hoffstein V. Are history and physical examination a good screening test for sleep apnoea. *Annals of Internal Medicine* 1991; **115**: 356–359.
19. Norton P, Dunn E, Haight J. Snoring in adults: some epidemiological aspects. *Canadian Medical Association Journal* 1983; **128**: 674–675.
20. Lim P, Curry A. The role of history, Epworth sleepiness scale score and body mass index in identifying non-apnoeic snorers. *Clinical Otolaryngology* 2000; **25**: 244–248.
21. Fairbanks D, Mickelson S, Woodson B. *Snoring and obstructive sleep apnoea*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins 2003.
22. Illum P. Septoplasty and compensatory inferior turbinate hypertrophy: long-term results after randomised turbino-plasty. *European Archives of Otorhinolaryngology* 1997; **254**(Suppl 1): S89–S92.
23. Low W. Can snoring relief after nasal septal surgery be predicted? *Clinical Otolaryngology* 1994; **19**: 142–144.
24. Johns M. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991; **14**: 540–545.
25. Sher A, Schechtmen K, Piccirillo J. The efficacy of surgical modification of the upper airway in adults with obstructive sleep apnoea syndrome. *Sleep* 1996; **19**: 156–177.

\*The most important references are denoted by an asterisk.



- \*26. Douglas N. Home diagnosis of the obstructive sleep apnoea/hypopnoea syndrome. *Sleep Medicine Reviews* 2003; **7**(1): 53–59.
27. Bliwise D, Nekich J, Dement W. Relative validity of self-reported snoring as a symptom of sleep apnoea in a sleep clinic population. *Chest* 1991; **99**: 600–608.
28. Hoffstien V, Szalai J. Predictive values of clinical features in diagnosing obstructive sleep apnoea. *Sleep* 1993; **16**: 118–122.
29. Bennett L et al. Sleep fragmentation indices as predictors of daytime sleepiness and nCPAP response in obstructive sleep apnoea. *American Journal of Respiratory and Critical Care Medicine* 1998; **158**: 778–786.
30. Aldrich M. Diagnostic aspects of narcolepsy. *Neurology* 1998; **50**: S2–S7.
31. Douglas N. 'Why am I sleepy **163**: 1310–1313.
32. Ritter C et al. Quantitative evaluation of the upper airway during nasopharyngoscopy with the Müller manoeuvre. *Laryngoscope* 1999; **109**: 954–963.
33. Isono S et al. Static mechanics of the velopharynx of patients with obstructive sleep apnoea. *Journal of Applied Physiology* 1993; **75**: 148–153.
34. Dolly F, Block A. Effect of flurazepam on sleep disordered breathing and nocturnal oxygen saturation in asymptomatic subjects. *American Journal of Medicine* 1982; **73**: 239–243.
35. Woodson B, Wooter M. Manometric and endoscopic localisation of airway obstruction after uvulopalatopharyngoplasty. *Otolaryngology, Head and Neck Surgery* 1994; **111**: 38–43.
36. El Badawey M et al. Predictive value of sleep nasendoscopy in the management of habitual snorers. *Annals Otolaryngology and Rhinology* 2003; **112**(1): 40–44.
37. Tvinneim M et al. Diagnostic airway pressure recording in sleep apnoea syndrome. *Acta Otolaryngologica* 1995; **115**: 449–454.
- \*38. Agrawal S et al. Sound frequency analysis and the site of snoring in natural and induced sleep. *Clinical Otolaryngology* 2002; **27**: 162–166.
39. McCombe A, Kwok V, Hawke W. An acoustic screening test for obstructive sleep apnoea. *Clinical Otolaryngology* 1995; **20**: 348–351.
- \*40. Hoffstien V. Snoring. *Chest* 1996; **109**(1): 201–222.
41. Berry R, Block A. Effect of nasal spray, positional therapy, and combination thereof in the asymptomatic snorer. *Sleep* 1994; **17**: 516–522.
42. Lipman D, Sexton G, Schlessner J. A randomised double-blind placebo-controlled evaluation of the safety and efficacy of a natural over-the-counter medication in the management of snoring. *Sleep and Breathing* 1999; **3**(2): 53–56.
43. Series F, Marc I. Effects of protriptyline on snoring characteristics. *Chest* 1993; **104**: 14–18.
44. Metes A, Cole P, Hoffstein V. Nasal airway dilation and obstructed breathing in sleep. *Laryngoscope* 1992; **102**: 1053–1055.
45. Hoijer U et al. The effect of nasal dilation on snoring and obstructive sleep apnoea. *Archives of Otolaryngology, Head and Neck Surgery* 1992; **118**: 281–284.
46. Hoffstein V, Mateika J, Metes A. Effect of nasal dilation on snoring and apnoeas during different stages of sleep. *Sleep* 1993; **16**: 360–365.
- \*47. O'Sullivan R et al. Mandibular advancement splint: an appliance to treat snoring and obstructive sleep apnoea. *American Journal of Respiratory and Critical Care Medicine* 1995; **151**: 194–198.
48. Johnston C et al. Oral appliances for the management of severe snoring: a randomised controlled trial. *European Journal of Orthodontics* 2001; **23**: 127–134.
49. Cartwright R et al. Treatments for snoring: a comparison of somnolasty and an oral appliance. *Laryngoscope* 2000; **110**: 1680–1683.
50. Ferguson K et al. A randomised crossover study of an oral appliance versus nasal continuous positive airway pressure in the treatment of mild-moderate obstructive sleep apnoea. *Chest* 1996; **109**: 1269–1275.
51. Pancer J et al. Evaluation of variable mandibular advancement appliance for treatment of snoring and sleep apnoea. *Chest* 1999; **116**: 1511–1518.
52. McGown A et al. Long-term use of mandibular advancement splint for snoring and obstructive sleep apnoea: a questionnaire survey. *European Respiratory Journal* 2001; **17**: 462–466.
53. Tsuki S, Ono T, Kuroda T. Mandibular advancement modulates respiratory related genioglossus electromyographic activity. *Sleep and Breathing* 2000; **4**(2): 53–57.
54. Rauscher H, Formanek D, Zwick H. Nasal continuous positive airway pressure for non-apnoeic snoring? *Chest* 1995; **107**: 58–61.
- \*55. Osman E et al. Snoring assessment: do home studies and hospital studies give different results? *Clinical Otolaryngology* 1998; **23**(6): 524–527.
56. Hicklin L, Tostevin P, Dasan S. Retrospective survey of long-term results and patient satisfaction with uvulopalatopharyngoplasty for snoring. *Journal of Laryngology and Otology* 2000; **114**: 675–681.
57. Kamami Y-V. Laser CO<sub>2</sub> for snoring: preliminary results. *Acta Oto-rhino-laryngologica Belgica* 1990; **44**: 451–456.
58. Osman E et al. Uvulopalatopharyngoplasty versus laser assisted uvulopalatoplasty for the treatment of snoring: an objective randomised clinical trial. *Clinical Otolaryngology* 2000; **25**: 305–310.
59. Reda M et al. Can we improve patient selection for snoring surgery? *Otolaryngology Head and Neck Surgery* 2003; **129**: 117.
60. Trotter M, D'Souza A, Morgan D. Medium-term outcome of palatal surgery for snoring using the Somnus unit. *Journal of Laryngology and Otology* 2002; **116**: 116–118.
61. Ikematsu T. Study of Snoring, 4th report: therapy. *J Jpn Otol Rhinol Laryngol* 1964; **64**: 434–435.
62. Fujita S, Conway WA, Zorik F, et al. Surgical corrections of anatomical abnormalities in obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. *Otolaryngol Head Neck Surg* 1981; **89**: 923–934.