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Obstructive sleep apnoea

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Cite this as: *BMJ* 2014;348:g3745
doi: 10.1136/bmj.g3745

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Although snoring and occasional apnoeic breath holding in sleep (obstructive sleep apnoea) is common, the diagnosis of obstructive sleep apnoea syndrome (OSAS) requires the presence of repetitive apnoeas and symptoms of sleep fragmentation, most commonly excessive daytime sleepiness. With increasing awareness of OSAS and portable diagnostic equipment, OSAS is no longer an esoteric diagnosis made only in specialist sleep centres but an easily recognised condition for which effective treatment is widely available. Obstructive sleep apnoea syndrome is also important from a public health perspective because of the increased risk of cardiovascular morbidity[1] and road traffic incidents.[2] This review is intended for non-specialists and describes the physiology and diagnosis of OSAS, the practicalities of treatment, and the identification of those patients who are likely to benefit.

SOURCES AND SELECTION CRITERIA

We based this review on articles found by searching PubMed and the Cochrane Database of Systematic Reviews. Search terms included "obstructive sleep apnoea or obstructive sleep apnoea syndrome" and "driving or treatment". The search was limited to articles in English and to studies carried out in adults. We gave priority to data from meta-analyses, reviews, and randomised controlled trials. We also included relevant reports and national guidelines.

What are apnoeas and how common is obstructive sleep apnoea?

Conventionally, an apnoea is a cessation of airflow for 10 seconds and is often associated with oxygen desaturation, whereas a lesser reduction in airflow is termed a hypopnoea. Sleep studies measure the apnoea/hypopnoea index (AHI), which is the number of respiratory events an hour. Community studies have used questionnaires and overnight monitoring to estimate the prevalence of obstructive sleep apnoea; in North American and Chinese cohorts the prevalence was 24% and 18.8% (AHI >5) and 9% and 5.3% (>15), respectively, in men and 9% and 3.7% and 4% and 1.2%, respectively, in women.[3 4 5] Excessive sleepiness becomes more prevalent once the AHI exceeds five events an hour, and this value has become a lower cut-off for the diagnosis of OSAS. The prevalence of OSAS (AHI >5 and symptoms of excessive daytime sleepiness) is surprisingly and consistently high, at 4% in men and 2% in women.

Why do obstructive apnoeas occur and why do they cause excessive daytime sleepiness?

In sleep the loss of pharyngeal dilator muscle tone results in a narrowing of the airway, and, if combined with retropositioning of the lower mandible or narrowing of the lateral pharyngeal wall by fat pads, may predispose to complete collapse of the upper airway. As the inspiratory respiratory muscles attempt to overcome the obstruction, autonomic activation re-establishes tone and terminates the apnoea but at the expense of a neurophysiological arousal,[6] an abrupt lightening of sleep stage that can be detected by electroencephalography. Patients with OSAS spend insufficient time in REM (rapid eye movement) and restorative slow wave sleep. During an obstructive apnoea event, swings in intrathoracic pressure cause both systemic and pulmonary arterial hypertension and sympathetic activation. No single genetic basis for OSAS has been identified, but a hereditable component is thought to play a part, perhaps mediated through craniofacial morphology and ventilatory control.

SUMMARY POINTS

- Obstructive sleep apnoea syndrome (OSAS) is a common and under-recognised cause of excessive daytime sleepiness
- It can be easily and effectively treated
- General practitioners play an important role in identifying symptoms and referring appropriately
- The risk of road traffic incidents is increased in untreated patients with OSAS
- OSAS may be a modifiable factor for vascular disease

When should OSAS be suspected?

A history of snoring and witnessed apnoeas with symptoms of sleep fragmentation, such as excessive daytime sleepiness, suggests OSAS. Sleepiness is most conveniently assessed using the Epworth sleepiness scale (fig 1). Investigation is usually recommended when scores are greater than 10, but as about 10% of the population have a score of 11 or more,[7] a high score should be complemented by a narrative confirmation of intrusive somnolence. Screening tools such as the Berlin questionnaire[8] and STOP-Bang[9] identify sleepiness as well as snoring, apnoeas, and hypertension, and they have sensitivities of about 85% in primary care and preoperative settings. Box 1 lists important aspects of the sleep history and relevant examination findings if obstructive sleep apnoea is suspected. Occasional witnessed apnoeas are common in the general population, particularly when people assume a supine position after alcohol consumption, and they are a potent source of spousal anxiety and referral. The presence of uncomplicated snoring or occasional apnoeas without a history of excessive sleepiness does not require investigation, and reassurance can be given that such apnoeas are self terminating and do not require treatment. Inadequate sleep time, a common cause of unrefreshing sleep or daytime sleepiness,[10] can be identified from the history and usually does not require further investigation. Box 2 lists the other presentations that would indicate a need for referral to a sleep study.

What are the tests for OSAS?

Overnight oximetry is widely available but oxygen desaturation is an inexact surrogate for apnoeic events, and the

Epworth Sleepiness Scale

Name: _____ Today's date: _____

Your age (Yrs): _____ Your sex (Male = M, Female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the **most appropriate number** for each situation:

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

It is important that you answer each question as best you can.

Situation	Chance of dozing (0-3)
Sitting and reading	
Watching TV	
Sitting, inactive in a public place (e.g. a theatre or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	

Fig 1 | Epworth sleepiness scale

Box 1 | Key history taking and examination findings in the assessment of suspected obstructive sleep apnoea

Questions to elicit a history

- Do you snore?
- Does your snoring occur when you are in a particular position?
- Have you ever had upper airway surgery?
- Has anyone ever witnessed you stop breathing or have choking episodes in your sleep?
- For how long do you sleep?
- Do you feel refreshed on waking?
- Do you experience daytime sleepiness, poor concentration, or poor memory?
- Have you ever had any road traffic incidents or near misses when driving?
- Do you have to use the toilet in the night?
- Do you have headaches in the early morning?
- Do you have loss of libido?

Examination

- Body mass index, neck circumference
- Blood pressure
- Thyroid assessment
- Assessment for nasal obstruction and retrognathia
- Oral examination for tonsillar enlargement and oropharyngeal crowding

Additional assessment should be made regarding drugs, comorbidities, and alcohol consumption

Box 2 | Indications for referral to a sleep study

- Symptoms of excessive sleepiness or an Epworth sleepiness score of ≥11*
- Recurrent witnessed apnoeas
- Nocturnal choking, gasping, or "dyspnoea"
- Headache in the morning
- Unrefreshing sleep despite adequate sleep time and continuity
- Near miss events or incidents caused by reduced vigilance while driving*
- Screening before bariatric surgery or upper airway surgery for snoring
- Otherwise unexplained polycythaemia, pulmonary hypertension, or ventilatory failure

*Urgent referral is necessary if the Epworth sleepiness score is >18 or the patient has had a road traffic incident or near miss event

ideal frequency and depth of desaturation events is still debated. An oxygen desaturation of 4% is conventionally used to indicate apnoea. The mean sensitivity and specificity of overnight oximetry is only 87% and 65%, respectively.[11] The false positive and negative results are partly explained by the shape of the oxygen dissociation curve, which means that obese patients with chronic obstructive pulmonary disease on the steep part of the curve may have numerous desaturations without many apnoeas, whereas young non-obese patients with major apnoeas can have a drop in partial pressure of oxygen without showing desaturation.

The SIGN (Scottish Intercollegiate Guideline Network) guidelines recommend that people with a typical history and more than 10 desaturations of 4% an hour can proceed to treatment without more detailed studies. If patients have fewer than five desaturations an hour and no sleep fragmentation then no further testing is required. If patients have excessive daytime sleepiness then a more detailed study is indicated, as the negative predictive value of "normal" oximetry is not sufficient to exclude OSAS.[12] The use of single channel testing (oximetry) alone is not, however, considered adequate in North America[13] or western Europe[14] and more variables are measured.

Domiciliary diagnostic systems, such as respiratory multichannel recording, can measure snoring, nasal airflow, position, oximetry, and pulse rate (fig 2) and can detect apnoeas and hypopnoeas. Thoracic and abdominal binders register reductions in chest movement (hypopnoeas) that fall short of an apnoea and can differentiate between obstructive and central events. The AHI is a summation of the number of events each hour, and SIGN guidelines use the AHI to stratify severity of OSAS: >5-14/h (mild), 15-29/h (moderate), and >30/h (severe).

Because of the non-linear correlation of the AHI with symptoms, the results of testing must be interpreted with the history. Arbitrary stratification can result in highly symptomatic patients with mildly abnormal studies being deprived of effective treatment, or those with more abnormal studies but minimal symptoms being asked to persist with a treatment from which they feel no benefit. The ideal method for diagnosis is full polysomnography, which involves overnight admission for supervised multichannel recording, including electroencephalography.

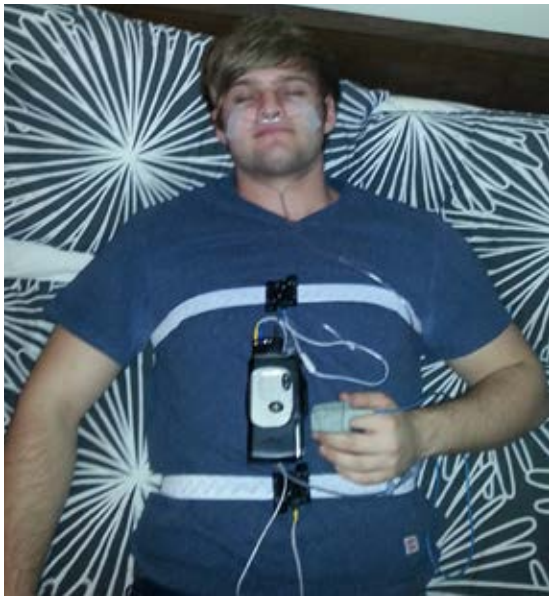

Fig 2 | Patient set up for respiratory multichannel recording, with sensors for nasal airflow, thoracic and abdominal movement, and pulse oximetry

Restricted availability of polysomnography and the cost mean that oximetry and limited respiratory monitoring are more widely used; these are just as effective.[12] The role of preliminary investigation in primary care is currently under evaluation.

What treatments are available for OSAS?

Intervention is usually indicated for patients with moderate to severe OSAS and some patients with mild OSAS with intrusive sleepiness. The main treatment options are essentially physical solutions to narrowing of the upper airway—namely, continuous positive airway pressure (CPAP), oral appliances, and upper airway surgery. Weight loss and bariatric surgery may also be appropriate interventions. Currently, the primary aim of treatment is to improve symptoms, particularly sleepiness, rather than the attenuation of putative vascular risks.

Lifestyle measures and weight loss

Patients with mildly abnormal sleep studies and minor sleep fragmentation may not require intervention other than general advice and reassurance. There may be scope to stop sedative drugs that can exacerbate OSAS or cause excessive sleepiness in their own right. Alcohol predisposes to apnoea, but the long term efficacy of abstinence is unknown. Positional treatment may be suitable when most apnoeic events occur with the patient in the supine position, but the available devices are not consistently effective or are so uncomfortable as to limit sleep in any position. Treatment with tricyclic and serotonergic drugs might have favourable effects on the musculature of the upper airway or sleep architecture, but such treatment is largely ineffective. A Cochrane review identified 25 studied drugs and 30 trials but a total of only 516 participants.[15] The studies were generally short term and any small reductions in AHI were often dissociated from improvement in sleepiness. The authors concluded that evidence was insufficient to recommend drug treatment but that in future there might be a role for targeted treatment if the mechanisms behind a patient's OSAS could be characterised.

The rising prevalence of obesity, which affects up to 70% of patients with OSAS, has contributed to the increased awareness of the condition. In a year-long randomised study of 72 patients with mild obstructive sleep apnoea (AHI 5-15) and minor symptoms (Epworth sleepiness score 10) a very low energy diet caused a 40% reduction in AHI, and in two thirds of patients the sleep study reverted to normal results.[16] A prospective observational study in 63 people who had previously managed a mean weight loss of 18 kg on a very low energy diet found that after a year the mean weight gain was 6 kg but that 30 out of 63 patients were still managing without CPAP.[17]

Although a Cochrane review in 2001[18] found no randomised trials on the effect of weight loss on the severity of sleep apnoea, a review of nine before and after studies[19] found evidence of an effect, with a mean reduction in body mass index of 4.8 kg/m^2 leading to a pooled decrease in the AHI from 53 to 28 events an hour. As sleep disordered breathing was diminished rather than abolished, the authors concluded that weight loss was adjunctive rather than curative in most patients. Bariatric surgery may be dramatically effective and offer permanent cure for some patients with OSAS. A systematic review found that the mean body mass index decreased from 55 to 38 kg/m^2 and the AHI from 55 to 16, but that residual symptoms requiring treatment were still present in over half of the study population.[20] Clearly, advice about weight loss should be given when indicated, but it is inappropriate to withhold other treatment from symptomatic patients in the over-optimistic hope of eventual sustainable weight loss.

CPAP treatment

CPAP consists of a motor unit that pushes out filtered air through a variety of facial mask interfaces at a positive pressure that is adjusted for each patient individually (fig 3). The pressure inside the mask remains positive throughout the respiratory cycle, splinting open the collapsible upper airway while the patient self ventilates. CPAP differs from non-invasive ventilation where cyclical positive pressure augments inspiration to inflate the lung and improve gas exchange in patients with ventilatory failure. CPAP is

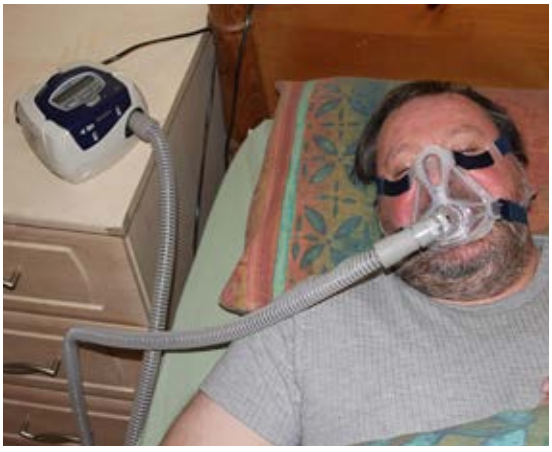

Fig 3 | Continuous positive airway pressure in a patient, whose nose and mouth is covered by a full face mask. Mouth breathers should be switched from a nasal to a full face mask if they develop intolerable nasal symptoms as a result of air leak from the mouth

the treatment of choice for symptomatic moderate or severe OSAS. A Cochrane review[21] identified 36 trials involving 1718 adults comparing CPAP with placebo or an oral appliance in participants with obstructive sleep apnoea compared with controls in parallel or first arm crossover studies. CPAP effected large improvements in sleepiness (mean decrease in Epworth sleepiness score 3.8, P<0.001) and various quality of life indicators. Adherence to treatment—that is, the number of hours used—is recorded by the CPAP unit and can be regularly monitored, although the minimum effective usage time is unknown. Although symptom resolution is often dramatic, at least 20% of patients eventually discontinue treatment because of lack of efficacy or poor tolerance.[22] If compliance is defined as more than four hours' use each night then this is not achieved by 46-83% of patients.[23] An experienced team familiar with machines, masks, and patients' anxieties is an essential part of any sleep service and helps to maximise the effectiveness of CPAP (table). Improvement in symptoms and the pattern of adherence is normally established within the first week, but some patients take longer to acclimatise or eventually decide that the inconvenience of treatment outweighs any symptomatic benefit. Failure to improve should prompt consideration of an alternative explanation (box 4).

Box 4 | Potential causes of persistent excessive daytime sleepiness despite continuous positive airway pressure

- Wrong or additional diagnosis
- Poor sleep hygiene, particularly inadequate sleep time
- Poor adherence (minimum 4 hours each night)
- Inadequate control of obstructive sleep apnoea (unrecognised mask leak, inadequate pressure)
- Comorbidities or sedating drugs
- Complex sleep disordered breathing (central sleep apnoea or additional obesity hypoventilation)

Oral appliances

Oral appliances include devices that are used to advance the mandible or tongue, which work by enlarging the upper airway or reducing upper airway collapsibility by increasing muscle tone. They should not be recommended as the preferred treatment for severe OSAS or in patients with severe sleepiness or noticeable nocturnal desaturation.[11 24] They can, however, be considered in patients who are intolerant of CPAP and can be useful for patients with mild to moderate OSAS; a meta-analysis of available studies suggests that sleepiness and apnoeic episodes are reduced in about 50% of such patients.[24] The devices may be bought off the shelf or custom made by dentists. They are not suitable for patients who are edentulous, and because of the possibility of tooth movement a preliminary dental assessment is advisable. A well tolerated device can only be presumed efficacious if symptoms improve, and ideally efficacy should be confirmed by a repeat diagnostic study.

The heterogeneity of devices and patient populations and the differing criteria of success make it difficult for studies to be compared. The consensus is that oral appliances are less effective than CPAP in patients with severe OSAS or with a high body mass index, but overall they are equally efficacious at reducing the AHI.[11 25]

Upper airway surgery

Surgical procedures may be considered for patients for whom CPAP or oral appliances have failed, or if such treatments are contraindicated by claustrophobia or dental disease, respectively. Surgical options include tonsillectomy, laser palatoplasty, uvulopalatopharyngoplasty, radiofrequency ablation of the tongue base, and suspension of the hyoid bone. Exceptionally, maxillofacial surgery can be considered to achieve advancement of the mandible. Operative interventions have not been as rigorously evaluated as "medical" treatments, but in the only randomised study of operative intervention, uvulopalatopharyngoplasty reduced the mean AHI by 60% at six months compared with only 11% in the controls who did not have surgery. To be eligible for the trial the 65 participants had to have failed treatment with CPAP or an oral appliance, an AHI >15, a body mass index <36 kg/m^2, and a specific palatal phenotype. Postoperative complications were unusual (pain or bleeding in six) and described as mild, but the effects on daytime sleepiness were not reported.[26]

Is there a link between obstructive sleep apnoea, OSAS, and vascular risk?

In prospective studies, obstructive sleep apnoea and OSAS have been associated with an increased risk of hypertension[27] and stroke[28] but less convincingly with coronary artery disease.[29] Community based studies show that as the AHI increases so does blood pressure,[30] and obstructive sleep apnoea is common in patients with hypertension that is difficult to treat.[31] Causation is, however, unproved and confounders such as age and obesity weaken the association.[1] The question arises as to whether treatment of obstructive sleep apnoea or OSAS will attenuate the

Pragmatic problem solving in patient receiving continuous positive airway pressure (CPAP)

Problem	Solution
Unable to tolerate pressure	Set machine to gradually ramp up pressure or auto-adjust CPAP
Difficulty falling asleep with mask on	Avoid daytime naps, use ramp function
Dry nose or mouth	Use humidification, if mouth leak try full face mask or chin strap
Mask removal in sleep	Exclude mouth leak (sore throat), mask leak (rush of air from side of mask). Adjustment of pressure may be required
Aerophagy (air swallowing)	Reduce pressure or tell patient to sleep propped up
Dry eyes or excess lacrimation	Adjust mask
Skin irritation	Recommend mask care (hot water and dilute washing-up liquid, well rinsed) or liners
Mask claustrophobia	Use nasal cushions, and gradually acclimatise patient during day with machine switched off
Nasal obstruction	Prescribe topical steroid or ephedrine, if severe consider surgery

QUESTIONS FOR FUTURE RESEARCH

Can the vascular risk associated with obstructive sleep apnoea be attenuated by treatment?

Would treatment for vascular risk be equally efficacious in all age groups?

How cost effective are preliminary investigations in primary care?

What is the optimal diagnostic test in primary care?

How practical and feasible would it be in secondary care to treat large numbers of patients who require continuous positive airway pressure?

ADDITIONAL EDUCATIONAL RESOURCES

Resources for healthcare professionals

Avidan AY, Zee PC. Handbook of sleep medicine. Lippincott Williams and Wilkins, 2011—overview of sleep medicine, including sleep apnoea, insomnia, parasomnias, and paediatrics

Douglas NJ. Clinician's guide to sleep medicine. Arnold, 2002—very readable text on sleep disorders, with particularly useful information on obstructive sleep apnoea

McNicolas WT, Bonsignore MR, eds. Sleep apnoea. *Eur Resp Monogr* 2010, No 50—detailed and exhaustive series of review articles on all aspects of sleep apnoea, including physiology, epidemiology, diagnosis, and management

Resources for patients

Sleep Apnoea Trust Association (www.sleep-apnoea-trust.org/)—advice for patients and relatives on all aspects of sleep apnoea and treatment

Driving and Vehicle Licensing Agency. Obstructive sleep apnoea syndrome and driving. 2013 (www.gov.uk/obstructive-sleep-apnoea-and-driving)—practical advice on sleep apnoea and driving and notification requirements

putative vascular consequences. Blood pressure is the most easily studied surrogate for vascular risk, and CPAP reduces blood pressure during both sleep and wakefulness,[32] suggesting a correlation between OSAS and hypertension. Patients with higher AHIs seem to derive the greatest benefit, but the fall in blood pressure also occurs in non-sleepy patients with obstructive sleep apnoea.[33] However, several meta-analyses have shown that the size of the blood pressure lowering effect of CPAP is small, at between 0.95 and 2.5 mm Hg (summarised in Parati and colleagues[34]). If only placebo controlled studies utilising ambulatory blood pressure monitoring are considered, then the mean fall in both 24 hour systolic and diastolic pressures was only about 1.5 mm Hg. The prevalence of obstructive sleep apnoea in patients with type 2 diabetes is approximately 20%,[35] and several studies have shown an independent effect of AHI on various aspects of glucose metabolism.[36] Intermittent hypoxia and oxidative stress predispose to insulin resistance, but CPAP did not alter insulin resistance in most studies.[37] The intriguing possibility that CPAP might reverse or at least stabilise diabetic retinopathy is currently being prospectively examined in the ROSA (Continuous positive airway pressure (CPAP) in patients with impaired vision due to diabetic Retinopathy and concurrent Obstructive Sleep Apnoea (OSA)) trial.

The effect of treatment on mortality is unknown. In a landmark observational study, untreated patients with severe OSAS (AHI >30) had a higher risk of fatal and non-fatal cardiovascular events than did patients with treated severe OSAS or untreated moderate OSAS.[38] A large follow-up study of mildly somnolent patients with OSAS[39] found that those who complied with CPAP were less likely to develop hypertension than untreated patients or those who were poorly compliant. Although undoubtedly an association exists between obstructive sleep apnoea, OSAS, and adverse vascular outcomes, evidence from well controlled studies is as yet insufficient to justify recommending CPAP purely to attenuate vascular risk in the absence of symptoms of sleep fragmentation.

What advice should patients with OSAS be given about driving?

Patients with OSAS should be assessed for their ability to drive safely and perform occupational activities requiring vigilance. Patients with intrusive sleepiness should be advised to cease driving until control of the symptoms has been achieved. In the United Kingdom, the Driver and Vehicle Licensing Agency requires patients with symptoms of sleepiness to notify the diagnosis if their ability to remain alert when driving is impaired, and information on the requirements and legislation for patients with sleep disorders, including OSAS, is available on its website.[40] Vocational drivers usually require regular follow-up and confirmation of treatment adherence and effectiveness.

Contributors: MG and MH contributed equally to the writing of this review and are the guarantors.

Competing interests: We have read and understood the BMJ group policy on declaration of interests and declare the following interests: none.

Provenance and peer review: Not commissioned; externally peer reviewed.

Patient consent: Obtained.

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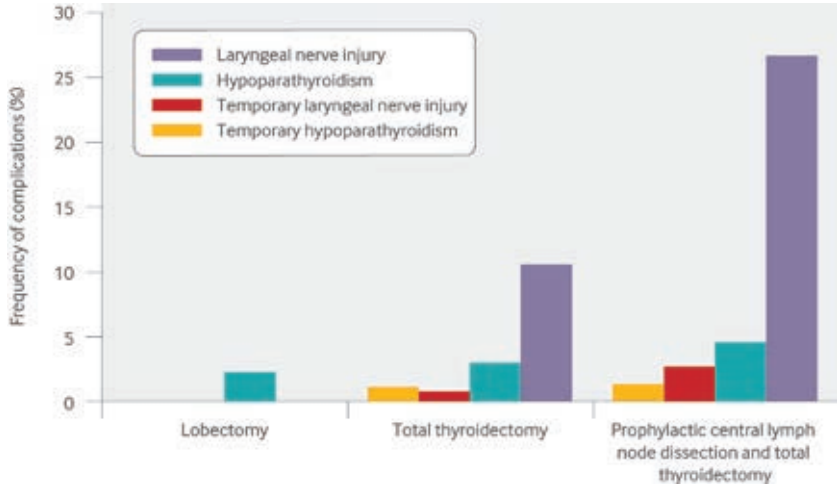
State of the Art reviews

Low risk papillary thyroid cancer



Frequency of complications of different surgical procedures for low risk papillary thyroid cancer calculated from relevant comparative cohorts

This week our State of the Art review is low risk papillary thyroid cancer (http://www.bmj.com/content/348/bmj.g3045). Thyroid cancer is one of the fastest growing diagnoses; more cases of thyroid cancer are found every year than all leukaemias and cancers of the liver, pancreas, and stomach.

Patients have a 99% survival rate at 20 years yet treatment is often aggressive. Although surgery is traditionally viewed as the cornerstone treatment there is less agreement about the extent of surgery (lobectomy *v* near total thyroidectomy) and whether prophylactic central neck dissection for removal of lymph nodes is needed.

Many of these tumors are treated with radioactive iodine ablation and thyrotropin suppressive therapy, which—although effective for more aggressive forms of thyroid cancer—have not been shown to be beneficial in the management of these lesions.

The review provides an evidence based approach to managing low risk papillary thyroid cancer. It also looks at the future of promising alternative surgical techniques, non-surgical minimally localized invasive therapies (ethanol ablation and laser ablation), and active surveillance.