RESPIRATION AND THE AIRWAY

Proportion of surgical patients with undiagnosed obstructive sleep apnoea

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Editor’s key points

- Obstructive sleep apnoea (OSA) is associated with perioperative morbidity but is under-diagnosed in the community.
- In this study of Canadian surgical patients, both anaesthetists and surgeons often failed to diagnose OSA.
- Preoperative diagnosis was poor, even in patients with symptoms of moderate-to-severe OSA.

Background. Obstructive sleep apnoea (OSA) affects ~9–24% of the general population, and 90% remain undiagnosed. Those patients with undiagnosed moderate-to-severe OSA may be associated with an increased risk of perioperative complications. Our objective was to evaluate the proportion of surgical patients with undiagnosed moderate-to-severe OSA.

Methods. After research ethics board approval, patients visiting preoperative clinics were recruited over 4 yr and screened with the STOP-BANG questionnaire. The 1085 patients, who consented, subsequently underwent polysomnography (PSG) (laboratory or portable) before operation. Chart review was conducted in this historical cohort to ascertain the clinical diagnosis of OSA by surgeons and anaesthetists, blinded to the PSG results. The PSG study-identified OSA patients were further classified based on severity using the apnoea–hypopnoea index (AHI) cut-offs.

Results. Of 819 patients, 111 patients had pre-existing OSA and 58% (64/111) were not diagnosed by the surgeons and 15% (17/111) were not diagnosed by the anaesthetists. Among the 708 study patients, PSG showed that 233 (31%) had no OSA, 218 (31%) patients had mild OSA (AHI: 5–15); 148 (21%) had moderate OSA (AHI: 15–30), and 119 (17%) had severe OSA (AHI >30). Before operation, of the 267 patients with moderate-to-severe OSA, 92% (n=245) and 60% (n=159) were not diagnosed by the surgeons and the anaesthetists, respectively.

Conclusions. We found that anaesthetists and surgeons failed to identify a significant number of patients with pre-existing OSA and symptomatic undiagnosed OSA, before operation. This study may provide an impetus for more diligent case finding of OSA before operation.

Keywords: diagnosis; perioperative period; screening; sleep apnoea, obstructive; surgery

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Obstructive sleep apnoea (OSA) is a common sleep disorder, characterized by episodes of apnoea or hypopnoea during sleep, resulting in hypoxaemia and hypercapnia. The obstructive apnoea or hypopnoea is caused by a complete or partial closure of the pharyngeal walls, requiring a confirmatory polysomnography (PSG) for assessment.1

Evidence from epidemiological studies suggests that the prevalence estimates for OSA range from 9% to 24% of the general population.2,3 Nearly 80% of men and 93% of women with moderate-to-severe sleep apnoea are undiagnosed in the community, and the extent of undiagnosed moderate-to-severe OSA in surgical patients needs to be determined.6 Among the general population, undiagnosed OSA may be associated with increased morbidity and mortality.5 The adjusted hazard ratio for all-cause mortality in patients with moderate-to-severe OSA is three- to six-fold higher compared with those without OSA.6,7 Undiagnosed OSA patients may present a variety of perioperative concerns: they have a higher incidence of difficult intubation,8 postoperative complications, increased admissions to intensive care unit (ICU), and longer duration of hospital stay.9,10

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The prevalence of OSA in the surgical population is higher than the general population and varies with the different surgical populations such as bariatric surgery.\textsuperscript{11,12} The disparity between a high prevalence of undiagnosed OSA in the population and the low level of clinical recognition has been recognized in the general population.\textsuperscript{13} We suspected that this discrepancy also exists in the surgical population and thus hypothesized that a significant proportion of surgical patients with moderate-to-severe OSA remains undiagnosed. We conducted a historical cohort study to test this hypothesis.

**Methods**

**Study cohort**

The study was conducted in the preoperative clinics of Toronto Western Hospital and Mount Sinai Hospital, Toronto, Ontario, Canada. Institutional Review Board approvals were obtained from both institutions (MSH: 06-0143-E and 07-0183-E; UHN: 06-0135-AE and 07-0515-AE). Patients visiting two preoperative clinics were approached to undergo screening for OSA and followed by a PSG for research purposes. Adult patients (>18 yr), ASA physical status I–IV who were undergoing elective procedures (general surgery, gynaecology, orthopaedics, urology, plastic surgery, ophthalmology, spine, or neurosurgery) were approached. Patients who were unwilling or unable to give informed consent or patients who were expected to have abnormal EEG findings (e.g. brain tumour, epilepsy surgery, patients with deep brain stimulator) were excluded. Study patients were recruited from the preoperative clinics at Toronto Western Hospital and Mount Sinai Hospital over 4 yr (October 2005 to November 2009).

**Preoperative screening and PSG**

After informed consent was obtained, patients were asked to complete the STOP-BANG questionnaire (Appendix).\textsuperscript{14–16} The STOP-BANG questionnaire is an acronym of eight independent elements where three are OSA-related symptoms, three are physiological measurements (BMI, neck circumference, and high arterial pressure), and two are patient characteristics (age and gender). The patients answered the questions on loud snoring, observed apnoea, daytime sleepiness, and history of high arterial pressure. A research assistant documented the data on BMI, age, neck circumference, and gender. The OSA-related symptoms (snoring, daytime tiredness, and observed apnoea) were determined from the patient’s response to the STOP-BANG questionnaire. Patients were invited to undergo an overnight PSG study at a laboratory PSG\textsuperscript{15} during the first 2 yr of study or a portable PSG at home with Embletta X-100 (Embletta X-100, Embla Systems, Inc., Broomfield, CO, USA) during the third and fourth year of study.\textsuperscript{17} The portable PSG device has been validated with laboratory PSG in our institution.\textsuperscript{17}

The PSG recordings were manually scored by a qualified PSG technician, and later reviewed by a sleep physician. The apnoea–hypopnoea index (AHI) was calculated as the number of abnormal respiratory events (apnoea or hypopnoea) per hour of sleep based on the American Academy of Sleep Medicine (AASM) criteria.\textsuperscript{18} The severity of OSA was defined by using AHI cut-offs as follows: no OSA (AHI ≤5), mild (AHI 5–15), moderate (AHI 15–30), or severe (AHI >30) based on the AASM criteria.\textsuperscript{19} The STOP-BANG questionnaire and the PSG study results were for research purposes only. The results were not available to the anaesthetists or surgeons taking part in the clinical care of the patients. At the time of the study, systemic screening for OSA by anaesthetists or surgeons was not part of standard preoperative assessment.

**Clinical diagnosis of OSA**

In this historical cohort study, charts of patients who underwent PSG before operation as part of the study cohort were selected and reviewed to ascertain the clinical diagnosis of OSA by the physicians. The research fellows who reviewed the charts were blinded to the results of the STOP-BANG questionnaire and PSG. The OSA diagnosis made by the surgeon at admission and the OSA diagnosis made by the anaesthetists in the preoperative assessments were noted. For patients with pre-existing OSA, the clinical diagnosis of OSA was classified as either ‘Documented OSA’ or ‘Suspected OSA’. Documented OSA was defined as OSA diagnosis based on a previous laboratory or portable PSG, or on the prescription of continuous positive airway pressure (CPAP) for OSA. Suspected OSA was defined as histories or features suggesting the presence of OSA based on a documentation on chart as ‘at-risk’, ‘probable’, or ‘possible’ OSA. Patients using CPAP therapy at home were also noted. Patients were considered to have undiagnosed OSA if there was no evidence found of a diagnosis of OSA under the ‘documented’ or ‘suspected’ OSA categories above.

**Statistical analyses**

Data were entered into a specifically designed Microsoft Access database and checked for possible errors. SAS 9.2 for Windows (SAS Institute, Cary, NC, USA) was used for data analysis. Categorical data were presented as frequency and percentage. The bootstrap resampling method was used to calculate the confidence interval (CI) of the percentage of undiagnosed OSA. The statistical significance was checked by the $\chi^2$ test or Fisher exact test. Continuous data with normal distribution are presented as mean ($\mu$); continuous data with skewed distribution are presented as median (25th, 75th percentile). The association between the OSA severity and the OSA-related symptoms, the number of undiagnosed OSA patients, and the number of OSA-related symptoms were tested with the Cochran–Armitage trend test. Bootstrap resampling method was used to adjust the $P$-value for multiple comparisons involved. Differences were considered statistically significant if $P<0.05$ or adjusted $P<0.05$ for multiple comparisons.
Sample size calculation
In the middle-aged general population, the prevalence of OSA has been found to be 9% in women and 24% in men. Since women accounted for 52% in their study population, the prevalence for whole population was calculated as 16.2% (9%×0.52+24%×0.48=16.2%). If at least 80% of them remain undiagnosed, then the calculated prevalence of undiagnosed OSA in the general population would be 13% (16.2×0.8). The majority of surgical patients are middle aged, and 27.5% surgical patients were identified as high risk of OSA by the STOP-BANG questionnaire. The positive predictive value of the STOP-BANG questionnaire has been shown to be 78.4%, and the estimated prevalence of OSA in surgical patients to be 21.6%. If we assume that the proportion of undiagnosed OSA in the surgical patient is the same as 80%, thus the calculated prevalence of undiagnosed OSA in the surgical patients would be 17.2%. With an α-error of 0.05 and a power of 0.9, the sample size was estimated to be 610.

Results
The study cohort
Over the study period, 5884 patients visiting the preoperative clinics were approached. A total of 1085 patients gave their consent. Two hundred and sixty-six patients withdrew from the study protocol, and 819 patients were able to complete a PSG study (Fig. 1). Of the 819 patients, 111 patients had pre-existing OSA diagnosis with 76 patients on home CPAP therapy.

Clinical diagnosis of OSA by anaesthetists and surgeons
The characteristics of 111 patients with pre-existing OSA and 708 patients with no previous diagnosis of OSA are summarized in Table 1. Among the 111 patients with pre-existing OSA, 85% (n=94) patients were identified as having OSA by the anaesthetists and 42% (n=47) patients were identified as having OSA by the surgeons. In 708 patients, the pre-operative PSG showed that 233 (31%) had no OSA, and 465 (69%) had PSG newly identified OSA. Of the 465 patients, 218 (31%) patients had mild OSA (AHI: 5–15); 148 (21%) had moderate OSA (AHI: >15–30), and 119 (17%) had severe OSA (AHI>30).

A significant proportion of the PSG study-identified OSA patients were not recognized by the physicians. As shown in Figure 2, 76% (95% CI: 67–79) of mild OSA, 65% (95% CI: 53–70%) of moderate, and 53% (95% CI: 38–58%) of severe OSA patients were not identified by the anaesthetists. The numbers of patients not identified by the surgeons were 97% (95% CI: 94–99%) of mild OSA, 93% (95% CI: 87–97%) moderate, and 90% (95% CI: 82–95%) severe OSA patients. In 267 (38%) patients diagnosed as moderate and severe OSA by the preoperative PSG (AHI>15), 60% (95% CI: 49–62%) and 92% (95% CI: 87–94%) were missed by the anaesthetists and the surgeons, respectively.

OSA-related symptoms in PSG study-identified patients
The frequency of loud snoring and observed apnoea increased with the increasing severity of OSA (Table 2). Sixty-
Table 1 Patient characteristic data of study population. CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CPAP, continuous positive airway pressure; GERD, gastroesophageal reflux disease; NYHA, New York Heart Association; OSA, obstructive sleep apnoea

<table>
<thead>
<tr>
<th>Type of surgery [n (%)]</th>
<th>Pre-existing OSA</th>
<th>No pre-existing OSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ear, nose, and throat</td>
<td>0 (0.0)</td>
<td>31 (4.4)</td>
</tr>
<tr>
<td>General</td>
<td>20 (18.2)</td>
<td>131 (18.5)</td>
</tr>
<tr>
<td>Gynaecology</td>
<td>1 (0.9)</td>
<td>37 (5.3)</td>
</tr>
<tr>
<td>Ophthalmology</td>
<td>3 (2.7)</td>
<td>31 (4.4)</td>
</tr>
<tr>
<td>Orthopaedic</td>
<td>59 (53.6)</td>
<td>343 (48.5)</td>
</tr>
<tr>
<td>Plastic</td>
<td>3 (2.7)</td>
<td>7 (1.0)</td>
</tr>
<tr>
<td>Spine</td>
<td>14 (12.7)</td>
<td>56 (7.9)</td>
</tr>
<tr>
<td>Urology</td>
<td>5 (4.6)</td>
<td>49 (6.9)</td>
</tr>
<tr>
<td>Other</td>
<td>5 (4.6)</td>
<td>22 (3.1)</td>
</tr>
</tbody>
</table>

Discussion

This historical cohort study shows that among the 111 patients with pre-existing OSA, 15% of patients were not identified as having OSA by the anaesthetists and 58% of patients were not identified as having OSA by the surgeons. Thirty-eight per cent (n=267) of the surgical patients who underwent PSG (n=708) had study-identified moderate-to-severe OSA (AHI>15). Surgeons and anaesthetists did not diagnose 93% and 65% of patients with moderate OSA and 90% and 53% of patients with severe OSA, respectively. The results indicate that the proportion of surgical patients with undiagnosed moderate–severe OSA remains very large and is similar to what is seen in the general population and that the anaesthetists and surgeons failed to identify obviously symptomatic OSA patients.

It is important to emphasize that OSA should be considered in the preoperative assessment, and anaesthetists and surgeons together should systematically evaluate the possibility of OSA in their patients as there are potential hazards in not doing so. Overall, 36.3% of patients with PSG-study-identified OSA had one or no cardinal symptoms. We should recognize the fact that these patients may have been beyond the reach of the clinical diagnosis as surgeons and anaesthetists cannot be expected to clinically detect ‘silent’ or asymptomatic OSA. These cases may have contributed to the high proportion of undiagnosed OSA.

Although the distinction between mild, moderate, and severe OSA with AHI ranges of 5–15, >15–30, and >30 is arbitrary, it is still the most widely accepted OSA severity classification. At present, there is no conclusive proof that mild OSA merits active treatment with CPAP and these patients may not have increased perioperative complications. Patients with moderate-to-severe OSA may carry increased perioperative risk. Increased awareness of the diagnosis of OSA could potentially decrease the proportion of undiagnosed moderate-to-severe OSA in the surgical patients, thus reducing perioperative risk.

The under-diagnosis of OSA has been highlighted in the perioperative medicine literature. Recently, the collaboration of the European Cooperation in Science and Technology (COST) Action B26 Group has been set up to determine sleep medicine service delivery with reference to OSA in Europe.
In our study, more than 60% of PSG study-identified moderate and severe OSA patients had reported at least two symptoms suggestive of OSA including either snoring, daytime sleepiness, or witnessed apnoea. Daytime sleepiness was the most common symptom, followed by witnessed apnoea and snoring. It is possible to greatly decrease the proportion of undiagnosed OSA by implementation of an OSA screening tool. In our patients with moderate and severe OSA, obstructive sleep apnoea; PSG: polysomnography.
OSA and not identified by the anaesthetists or surgeons, 93% were classified as at risk of OSA by the STOP-BANG questionnaire. This suggests that had our study patients been screened before operation with the STOP-BANG questionnaire, the majority of undiagnosed moderate and severe OSA would have been identified. The sensitivity and the specificity of the STOP-BANG questionnaire to identify patients with moderate-to-severe OSA is 93% and 43% and the positive predictive value and the negative predictive value is 52% and 90%, respectively.14 A higher STOP-BANG score has been shown to indicate a higher probability of moderate-to-severe OSA and may help identify these patients.16 The specificity for a STOP-BANG score of 5, 6, and 7 to predict severe OSA is 74%, 88%, and 96%, respectively.28 In a recent series of 135 patients presenting with MI, 74% of those with confirmed MI had a STOP-BANG score suggestive of high risk of OSA.29

Undiagnosed OSA has been associated with an increased risk of perioperative complications.30 36 Recently, Kaw and colleagues10 found that in patients with OSA undergoing non-cardiac surgery, there was a higher incidence of postoperative hypoxaemia, overall complications, ICU transfer, and a longer hospital length of stay. In a large population-based study, Memtsoudis and colleagues31 demonstrated that OSA was associated with a significantly higher adjusted odds ratio (OR) of pulmonary complications after orthopaedic and general surgical procedures. Initiation of positive airway pressure therapy for patients with OSA can significantly decrease healthcare costs in the general population.32 The association between OSA and the incidence of postoperative delirium has been established recently.33 34 With the timely preoperative CPAP therapy and the appropriate monitoring in the perioperative period, the incidence of postoperative complications could be reduced in a bariatric surgical population with OSA.35 Anaesthetists could potentially provide long-term health benefit to the patients with undiagnosed OSA by the implementation of screening for OSA, initiation of CPAP therapy perioperatively, and ensuring follow-up by the sleep physician after operation.24 36

There are some limitations to this study. One limitation could be the self-selection bias with an increased prevalence of OSA in our study population.2 4 There was a high refusal and a large drop-out rate among patients to complete a PSG possibly because of preoperative anxiety and patients with symptoms of OSA may have been more likely to give consent for PSG. This potential selection bias would influence the conclusions drawn from our study if our objective was to estimate the prevalence of OSA in the surgical patients as a whole. However, this was not the intent of our study. Our objective was to determine the proportion of surgical patients with PSG-determined moderate-to-severe OSA, who were identified as having OSA by their responsible surgeons and anaesthetists. The potential selection bias in our sample does not invalidate our finding that the responsible physicians often failed to identify patients with formal diagnoses of moderate-to-severe OSA. Indeed, it could be reasonably argued that since this sample had characteristics that may increase physicians’ suspicion of OSA (e.g. high BMI), our estimate of the proportion of ‘missed’ OSA diagnoses may be an underestimate of the true proportion in a general sample of surgical patients.

Secondly, the retrospective nature of the analysis has some inherent limitations. The high proportion of undiagnosed moderate–severe OSA in the surgical patients may be a combination of multiple factors: under-recognition, failure to consider OSA as a pre-existing medical disease, poor record keeping, and deference to another team member. Also, we were unable to identify specific predictors used by the responsible physicians to help diagnose OSA. A recording bias may exist, as the data were abstracted from the clinical charts of the patients after their surgeries. Thus, specific symptoms or signs that the physicians may have used were not available unless it was documented on the chart.

In conclusion, anaesthetists and surgeons failed to identify a significant number of patients with a pre-existing OSA diagnosis and undiagnosed OSA patients with obvious symptoms of OSA before operation. This study may provide an impetus for more diligent case finding of OSA before operation. Future studies should focus on ways to determine barriers to implementation of screening tools, and increased recognition of moderate-to-severe OSA among the perioperative team.

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Declaration of interest
None declared.

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Surgical patients with undiagnosed sleep apnoea


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**Appendix: STOP-BANG questionnaire**

1. Snoring: Do you snore loudly (loud enough to be heard through closed doors)?
   - Yes
   - No

2. Tired: Do you often feel tired, fatigued, or sleepy during daytime?
   - Yes
   - No

3. Observed: Has anyone observed you stop breathing during your sleep?
   - Yes
   - No

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Blood pressure: Do you have or are you being treated for high blood pressure?
Yes No

BMI: BMI more than 35 kg m$^{-2}$?
Yes No

Age: Age over 50 years old?
Yes No

Neck circumference: Neck circumference greater than 40 cm?

Gender: Male?
Yes No

Low risk of OSA: Yes to less than 3 questions.
At risk of OSA: Yes to 3 or more questions.
High risk of OSA: Yes to 5 or more questions.
Modified from Chung and colleagues.$^{14, 28}$