Introduction

The United Kingdom has one of the fastest growing rates of obesity in the developed world (1). In 2013, approximately a quarter of adults (26% of men and 24% of women) were obese and 2% were morbidly obese (2). Analysis by the UK government estimates that over half of the UK population could be obese by 2050, posing a significant challenge to the National Health Service (NHS). Obesity causes an increased load on the respiratory system (3,4) leading to breathlessness and is associated with a number of respiratory conditions including asthma, pulmonary embolism, pneumonia and obstructive sleep apnea (OSA) (5).

OSA is the most common sleep-related breathing disorder, characterized by recurrent episodes of upper airway occlusion leading to apneas and hypopneas whilst the patient is asleep (6). These episodes cause intermittent hypoxia, arousal from sleep and lead to increased...
sympathetic activity, increased respiratory effort (3,4) and sleep fragmentation (7). OSA causes daytime sleepiness and has been associated with co-morbidities (8) including type 2 diabetes mellitus (T2DM) (9), hypertension (10), and increased cardiovascular risks, including stroke (11).

The prevalence of OSA is increasing, it affected 4% of middle aged men and 2% of middle aged women in the United States in the early 1990’s (12), but with the obesity epidemic the prevalence is now at 10% for 30–49-year-old men and at 3% for 30–49-year-old women in the US (13). In obese subjects, prevalence can be as high as 60-70% (14) and most patients remain undiagnosed (15) and untreated (8).

Continuous positive airway pressure (CPAP) is the best available treatment for OSA (16), the National Institute of Health and Clinical Excellence (NICE) recommends the use of CPAP as a treatment option for adults with moderate to severe OSA and mandibular advancement devices for mild conditions (17). In obese patients, weight loss can improve sleep-disordered breathing (18). In severe obesity which is refractory to diet, physical exercise and bariatric surgery effectively support weight loss (19). However, weight loss due to bariatric surgery has, so far, failed to prove its superiority compared to conventional weight loss strategies with respect to its impact on the severity of OSA, as measured by the apnea-hypopnea-index (20).

Patients who are eligible for bariatric surgery may suffer from undiagnosed OSA (21) potentially putting them at risk when undergoing general anesthesia (22-27). A meta-analysis investigating the association between OSA and post-operative complications of any intervention found that the incidence of post-operative desaturations, respiratory failure, cardiac events and transfers to the intensive care unit was higher in patients with untreated OSA (23).

The standard to diagnose OSA is overnight polysomnography (PSG), but this is an expensive and time-intensive procedure and not widely available (8,28). In contrast, a case-finding screening tool often used to classify patients at risk of OSA is the STOP-BANG questionnaire. STOP-BANG is the acronym for snoring (‘does the patient snore loudly (loud enough to be heard through closed doors or your bed-partner elbows you for snoring at night?)’), tiredness (‘does the patient often feel tired, fatigued, or sleepy during the daytime?’), observed apnea (‘has anyone observed the patient stop breathing or choking/gasping during sleep?’), blood pressure (BP) (‘does the patient have or is the patient being treated for high BP?’), body mass index (BMI >35 kg/m²), age (>50 years), neck circumference (>40 cm), and gender (male). Each item scores either ‘0’ (if the answer is no) or ‘1’ (if the answer is yes) point. Those who scored >4 points are likely to be at risk of OSA and were referred to the Sleep Disorder Centre for a comprehensive evaluation. A STOP-BANG score of >4 points has been found to have a sensitivity of 88% for the diagnosis of OSA (32).

As part of the evaluation expert sleep physicians assessed the patients’ history of the sleep complaint, brief examination, recorded weight, height, BP and other co-morbidities and measured the epworth sleepiness scale (ESS) (33).

Patients and methods
Screening of bariatric patients
Patients undergoing bariatric surgery at Guy’s & St Thomas’ NHS Foundation Trust, London, UK between 12/2013 and 12/2014 were screened as part of the pre-operative assessment using the STOP-BANG questionnaire. STOP-BANG is the acronym for snoring [‘does the patient snore loudly (loud enough to be heard through closed doors or your bed-partner elbows you for snoring at night?)’], tiredness [‘does the patient often feel tired, fatigued, or sleepy during the daytime?’], observed apnea [‘has anyone observed the patient stop breathing or choking/gasping during sleep?’], blood pressure (BP) [‘does the patient have or is the patient being treated for high BP?’], body mass index (BMI >35 kg/m²), age (>50 years), neck circumference (>40 cm), and gender (male). Each item scores either ‘0’ (if the answer is no) or ‘1’ (if the answer is yes) point. Those who scored >4 points are likely to be at risk of OSA and were referred to the Sleep Disorder Centre for a comprehensive evaluation. A STOP-BANG score of >4 points has been found to have a sensitivity of 88% for the diagnosis of OSA (32).

As part of the evaluation expert sleep physicians assessed the patients’ history of the sleep complaint, brief examination, recorded weight, height, BP and other co-morbidities and measured the epworth sleepiness scale (ESS) (33).

Patient data
Patient demographics included age, sex, BMI, ESS score, comorbidities including hypertension, T2DM, depression and ischemic heart disease (IHD).

Nocturnal pulse oximetry
Patients underwent nocturnal pulse oximetry (Pulsox 300i, Konica Minolta sensing Inc., Hachioji, Tokyo, Japan) for two consecutive nights at home. The following data were recorded and calculated:

- Mean peripheral capillary oxygen saturation (SpO₂)
- 4% and 3% oxygen desaturation index (ODI, h⁻¹)
- Total time with oxygen saturation <90% (percent of the total night)
- Mean pulse rate (h⁻¹)
- Pulse rises [>6 beats per minute (bpm); h⁻¹]

The recorded traces were reviewed and interpreted by
an experienced sleep physician. Based on the results of the tests the sleep physician provided an interpretation. OSA was defined as a 4% ODI of \( >5 \text{ h}^{-1} \). Severity of OSA was classified as mild (4% ODI 5-15 h\(^{-1}\)), moderate (4% ODI 15-30 h\(^{-1}\)) and severe (4% ODI >30 h\(^{-1}\)) (34).

Treatment decisions for the patient, based on the results of the above assessment, included (Figure 1A,B):

(I) No significant sleep-disordered breathing, no action required;

(II) Mild OSA, if the patients were symptomatic it was recommended to consider a mandibular advancement device for symptom management independent of the surgical intervention;

(III) Moderate-severe OSA, trial of CPAP;

(IV) Extubation onto CPAP advised post-surgery due to significant desaturations;

(V) Suggested admission to the sleep center for further respiratory assessment, if the results of the pulse oximetry were inconclusive.

**Statistical analysis**

Continuous variables are presented as mean (standard deviation) and categorical variables as percentages. For unadjusted comparisons between OSA and non-OSA patients, continuous variables were compared using an unpaired two-tailed \( t \)-test. All other categorical variables were compared using the Chi-square test. For unadjusted comparisons between mild, moderate and severe OSA, continuous variables were compared using the Kruskal-Wallis one-way ANOVA with Dunn parisons between multiple comparisons. All other categorical variables (class II or III obesity, hypertension, T2DM, depression and IHD) were compared using the Chi-square test. In the univariate analysis we compared the dependent variables 4% ODI h\(^{-1}\) (and 3% ODI h\(^{-1}\)) with other independent variables (gender, age, BMI, ESS, class II/III obesity, hypertension, diabetes, IHD, SpO\(_2\), Time with oxygen saturation <90% (%), pulse rate (h\(^{-1}\)) and pulse rise (h\(^{-1}\)) using a Spearman correlation test. We finally performed a multiple linear regression analysis to identify the variables that significantly correlated with the dependent variables; 3% ODI h\(^{-1}\) and 4% ODI h\(^{-1}\). A \( P \) value <0.05 was assumed to represent statistical significance.

**Results**

**Demographics**

The studied cohort included a total of 141 patients awaiting bariatric surgery who scored \( >4 \) points on STOP-BANG in their pre-operative assessment [70% female, age 49 (±12) years, BMI 48.7 (±7.3) kg/m\(^2\); Table 1]. Systemic hypertension was prevalent in 40%, T2DM was present in 35%, and 25% of the patients had depression. The majority of the patients were not excessively sleepy [ESS 7.3 (±4.9) points].

**Sleep apnea prevalence and treatment recommendations**

Sleep-disordered breathing of any degree was evident in 103 of 141 patients (73%). 13 of 141 patients (9%) were found to have severe OSA, 19 of 141 (13%) had moderate OSA and 34 of 141 (24%) had mild OSA. 12 of 141 (9%) suffered with moderate to severe OSA with significant periods of the night below SpO\(_2\) of 90%, potentially indicating co-existing obesity hypoventilation syndrome (OHS) (35).

Based on these findings, 34 of 141 (24%) patients who were excessively sleepy were given CPAP prior to surgery and in 5 of 141 (4%) patients’ extubation to CPAP post-surgery was recommended. Fifteen of 141 patients (11%) were admitted for further respiratory assessment to the sleep laboratory, and in 13 of 141 patients (9%) the use of a mandibular advancement device was recommended (Figure 1A,B).

**Nocturnal pulse oximetry**

There were no significant differences in parameters between the nocturnal pulse oximetry data on night 1 vs. night 2 (Table 2). Bariatric patients who had OSA were older (\( P <0.004 \)), more obese (\( P <0.008 \)), and more sleepy (\( P <0.05 \)). They had more co-morbidities with hypertension (\( P <0.05 \)) and T2DM (\( P <0.05 \) (Table 3)). A multiple linear regression analysis revealed that time with SpO\(_2\) saturation <90% (\( P <0.001 \)), pulse rise index (\( P <0.001 \)) and ESS (\( P <0.001 \)) were independently associated with the oxygen desaturation indices (Table 4).

**Discussion**

Patients undergoing bariatric surgery who score >4 on the STOP-BANG questionnaire have a high prevalence of OSA with almost three quarters of patients having some abnormal nocturnal breathing pattern. Approximately one quarter of the patients had moderate-severe OSA, while 1/10 had a recording that was consistent with combined OSA/OHS (35). In more than one quarter of all patients CPAP was employed for peri-operative management. 11% of the patients were invited for further respiratory assessment for an inpatient sleep study in the
Patients referred by the bariatric service [2014]

N=141
STOP BANG score >4

Pulse oximetry study (ODI)

ODI 0-5
No OSA/minor SDB (n=63)

ODI 5-15
Mild OSA (n=34)

ODI 15-30
Moderate OSA (n=19)

ODI >30
Severe OSA (n=13)

OSA and OHS (n=12)

A

B

ODI 0-5
No OSA/minor SDB (n=63)

No treatment (n=57)
Mandibular assisted device (n=3)
Extubation onto CPAP advised post-surgery (n=1)
Continuous positive airway pressure (n=2)

ODI 5-15
Mild OSA (n=34)

No treatment (n=19)
Mandibular assisted device (n=10)
Continuous positive airway pressure (n=1)
Extubation onto CPAP advised post-surgery (n=4)

ODI 15-30
Moderate OSA (n=19)

Continuous positive airway pressure (n=18)
Admission to the sleep centre for further respiratory assessment (n=1)

ODI >30
Severe OSA (n=13)

Continuous positive airway pressure (n=8)
Admission to the sleep centre for further respiratory assessment (n=5)

OSA and OHS (n=12)

Continuous positive airway pressure (n=5)*
Admission to the sleep centre for further respiratory assessment (n=9)

Figure 1 Flow diagram of decisional management of bariatric cohort. *, two patients given CPAP and admitted. ODI, oxygen desaturation index; SDB, sleep disordered breathing; OSA, obstructive sleep apnea; OHS, obesity hypoventilation syndrome; CPAP, continuous positive airway pressure.
Table 1 Baseline data of the cohort of 141 patients studied

<table>
<thead>
<tr>
<th>Variables</th>
<th>Participants (n=141)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (M:F)</td>
<td>42:99</td>
</tr>
<tr>
<td>Age (years)</td>
<td>48.8±(12.2)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>48.7±(7.3)</td>
</tr>
<tr>
<td>ESS (/15)</td>
<td>7.3±(4.9)</td>
</tr>
</tbody>
</table>

Data are presented as mean ± (SD). BMI, body mass index; ESS, epworth sleepiness scale.

Table 2 Nocturnal pulse oximetry data of the patients studied, comparison of night 1 vs. night 2

<table>
<thead>
<tr>
<th>Nocturnal oximetry</th>
<th>Night 1</th>
<th>Night 2</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SpO₂ (%)</td>
<td>94 (2.8)</td>
<td>93.9 (2.9)</td>
<td>0.79</td>
</tr>
<tr>
<td>4% ODI (h⁻¹)</td>
<td>17.1 (25.3)</td>
<td>19 (27.5)</td>
<td>0.09</td>
</tr>
<tr>
<td>3% ODI (h⁻¹)</td>
<td>21.6 (27.2)</td>
<td>21.9 (28.1)</td>
<td>0.54</td>
</tr>
<tr>
<td>Time with O₂ sats &lt;90% (%)</td>
<td>8.9 (17.1)</td>
<td>8.3 (16.3)</td>
<td>0.84</td>
</tr>
<tr>
<td>Pulse rate (h⁻¹)</td>
<td>35.3 (12)</td>
<td>35.5 (11.4)</td>
<td>0.91</td>
</tr>
<tr>
<td>Pulse rise (h⁻¹; &gt;6 bpm)</td>
<td>75.4 (21.1)</td>
<td>74.9 (20.4)</td>
<td>0.37</td>
</tr>
</tbody>
</table>

There were no significant differences between the nights. The results indicated moderate sleep-disordered breathing with almost 1/10 of the night below oxygen saturations of 90%, and an increased pulse rise index caused by arousal from sleep. All data are presented as mean (SD). SpO₂, peripheral capillary oxygen saturation; ODI, oxygen desaturation index; bpm, beats per minute.

Table 3 Comparison of patients with OSA and those without

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>OSA</th>
<th>No OSA</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48.5 (12.3)</td>
<td>42.0 (10.6)</td>
<td>0.004</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>49.6 (7.8)</td>
<td>46.0 (5.7)</td>
<td>0.008</td>
</tr>
<tr>
<td>BMI score 35-39</td>
<td>97.0 (68.7)</td>
<td>35.0 (82.1)</td>
<td>0.65</td>
</tr>
<tr>
<td>ESS (/15)</td>
<td>7.6 (5.1)</td>
<td>6.0 (4.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>Nocturnal oximetry</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SpO₂ (%)</td>
<td>93.4 (2.9)</td>
<td>96.0 (1.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>4% ODI (h⁻¹)</td>
<td>22.3 (27.8)</td>
<td>3.0 (12.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3% ODI (h⁻¹)</td>
<td>27.9 (29.5)</td>
<td>4.0 (18.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time with O₂ sats &lt;90% (%)</td>
<td>12.1 (19.0)</td>
<td>0 (6.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulse rate (h⁻¹)</td>
<td>75.4 (11.7)</td>
<td>74.0 (8.1)</td>
<td>0.38</td>
</tr>
<tr>
<td>Pulse rise (h⁻¹; &gt;6 bpm)</td>
<td>37.5 (23.0)</td>
<td>31.0 (15.2)</td>
<td>0.06</td>
</tr>
<tr>
<td>Co-morbidities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>46.0 (44.6)</td>
<td>10.0 (26.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>T2DM</td>
<td>40.0 (38.8)</td>
<td>8.0 (21.1)</td>
<td>0.05</td>
</tr>
<tr>
<td>Depression</td>
<td>24.0 (23.3)</td>
<td>11.0 (28.9)</td>
<td>0.49</td>
</tr>
<tr>
<td>IHD</td>
<td>2.0 (1.9)</td>
<td>1.0 (2.6)</td>
<td>0.73</td>
</tr>
</tbody>
</table>

Hypertension ≥140/90 mmHg. Patients with OSA were older, more obese, sleepier and had more comorbidities with hypertension and T2DM. BMI, body mass index; ESS, epworth sleepiness scale; SpO₂, peripheral capillary oxygen saturation; ODI, oxygen desaturation index; T2DM, type 2 diabetes mellitus; IHD, ischemic heart disease; OSA, obstructive sleep apnea.

Table 4 Spearman correlation between the dependent variables (4% ODI and 3% ODI) and other independent variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>4% ODI</th>
<th>3% ODI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>0.26</td>
<td>0.29</td>
</tr>
<tr>
<td>Age</td>
<td>0.17</td>
<td>0.18</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>0.30</td>
<td>0.28</td>
</tr>
<tr>
<td>ESS (score)</td>
<td>0.28</td>
<td>0.28</td>
</tr>
<tr>
<td>Class II obesity</td>
<td>0.06</td>
<td>0.07</td>
</tr>
<tr>
<td>Class III obesity</td>
<td>0.06</td>
<td>0.07</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.18</td>
<td>0.19</td>
</tr>
<tr>
<td>T2DM</td>
<td>0.16</td>
<td>0.16</td>
</tr>
<tr>
<td>Depression</td>
<td>0.05</td>
<td>0.04</td>
</tr>
<tr>
<td>IHD</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>SpO₂</td>
<td>0.64</td>
<td>0.65</td>
</tr>
<tr>
<td>Time with O₂ saturation &lt;90% (%)</td>
<td>0.89</td>
<td>0.87</td>
</tr>
<tr>
<td>Pulse rate (h⁻¹)</td>
<td>0.28</td>
<td>0.29</td>
</tr>
<tr>
<td>Pulse rise (h⁻¹)</td>
<td>0.35</td>
<td>0.36</td>
</tr>
</tbody>
</table>

Hypertension ≥140/90 mmHg. †, P<0.05, ‡, P<0.001. BMI, body mass index; ESS, epworth sleepiness scale; SpO₂, peripheral capillary oxygen saturation; ODI, oxygen desaturation index; T2DM, type 2 diabetes mellitus; IHD, ischemic heart disease.

Clinical significance of findings

Almost half of the patients were given some recommendation for further respiratory management, no further action was required for 54% of patients. CPAP is a common and effective treatment for OSA (36). It reduces symptoms like daytime sleepiness, but it has also been found to improve cognitive function (37) and cardiovascular risks (38). As our understanding about prevalence in bariatric surgery cohorts improves, it is important that we diagnose and treat OSA in these patients to reduce peri-operative complications. Our study also found that patients with OSA compared to those without OSA were older, heavier and sleepier, they were also more likely to have hypertension and T2DM. Although this is not causal evidence in this small cohort, it supports the suggestion that OSA is a risk factor for cardiovascular and metabolic conditions (13).
Review of literature

Our results are consistent with previous studies describing the prevalence of OSA to be 78.3% in patients who have bariatric surgery (39), while another study found that 77% of patients for bariatric surgery had OSA (40). It has been reported that patients with OSA who received CPAP therapy before surgery have less peri-operative complications compared to patients with undiagnosed and untreated OSA (25). This underlines the importance to diagnose and treat OSA in these patients to reduce the risk of complications during and following bariatric surgery.

Data from the national bariatric surgery registry in the UK show that in 2006 there were fewer than 500 bariatric surgery procedures and that this increased to over 6,000 bariatric surgery procedures in 2013 (41), a trend that is mirrored the world over. Therefore the number of patients at risk of peri-operative complications is increasing and diagnosis and treatment of patients with OSA using CPAP could optimize peri-operative management.

Limitations of the study

Patients were from a selected cohort, screened using the STOP-BANG score, and final outcomes and complications after surgery was not recorded, peri-operative risks were not assessed. In order to investigate the effectiveness of CPAP treatment future studies would need to randomize identified cases of patients with OSA undergoing bariatric surgery to a trial of CPAP or sham-CPAP peri-operatively. It would also be of interest to extend this research to other surgical non-bariatric cohorts. Further, the absolute prevalence of OSA in all bariatric surgery patients may differ due to the exclusion of patients who scored less than four points on the STOP-BANG questionnaire.

Conclusions

There is a high prevalence of sleep-disordered breathing in patients undergoing bariatric surgery who score >4 points on STOP-BANG. Screening these patients for OSA prior to bariatric surgery using nocturnal pulse oximetry and offering treatment with CPAP could reduce peri-operative risks.

Acknowledgements

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

References


