Original Article

Obstructive Sleep Apnoea in Singapore: Polysomnography Data From a Tertiary Sleep Disorders Unit

Li Ling Lim,[1,2] *MBBS, MRCP (UK), ABSM (Sleep Medicine) (USA)*, Kwang Wei Tham,[3,4] *MB,BCh, BAO*, Stephanie MC Fook-Chong,[5] *BSc, MSc (McGill), CStat*

Abstract

Introduction: Comprehensive sleep architecture and respiratory event data in local patients with suspected obstructive sleep apnoea (OSA) from overnight polysomnography (PSG), the gold standard for the evaluation of sleep-related breathing disorders, are not widely available. We present 1 year retrospective PSG data with the objective of describing PSG characteristics of patients evaluated for OSA in Singapore. Materials and Methods: PSG data of patients evaluated for OSA in 1 year (January through December 2005) in the Sleep Laboratory of a public tertiary hospital were retrospectively reviewed. Results: Five hundred and eighty-four diagnostic PSG studies were performed in patients with symptoms suggestive of sleep-disordered breathing, including snoring, excessive daytime sleepiness, unrefreshing sleep, or recurrent unexplained awakenings. There were 449 male patients (76.9%) and 135 female patients (23.1%), with a mean age of 47.5 years (SD 12.7). Men were on average younger than women, 46.1 years versus 52.0 years (*P* <0.0005). The mean body mass index (BMI) was 27.9 (SD 6.7), with no significant difference between genders. An association was shown between apnoea-hypopnoea index (AHI) and BMI (Pearson correlation index r = 0.362). Men had overall significantly higher AHI (16.5 vs 9), shorter mean sleep onset latency (11 vs 16.5 minutes), more light sleep (65.5% vs 58.9%), less deep sleep (17.7% vs 23%), and more respiratory event related arousals per hour of sleep (11.6 vs 5.1) (*P* <0.0005). Severity was classified: AHI <5 ("Normal Overall AHI") (28.3%), AHI 5-15 ("Mild") (22.3%), AHI >15-30 ("Moderate") (18.3%), AHI >30 ("Severe") (31.2%). There was no significant age difference among the 4 groups. More severe OSA patients were significantly heavier, and had more light sleep, less deep sleep, less REM sleep, more respiratory event related arousals and lower levels of oxygen desaturation. Conclusion: OSA is predominant in middle-aged, overweight Singapore males and much less common in females who tend to be older. A majority of patients have moderate to severe OSA, which significantly disturbs normal sleep architecture. The relatively lower BMI compared to Caucasian OSA populations may be related to local craniofacial characteristics and/or higher percentage of body fat for BMI which has been described in Singaporeans.

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Key words: Body-mass index, Obstructive sleep apnoea (OSA), Polysomnography (PSG), Sleep architecture

Introduction

Overnight attended polysomnography (PSG) in the sleep laboratory has been the gold standard to confirm the presence and severity of obstructive sleep apnoea (OSA).[1] Standard PSG scoring rules using the criteria of Rechtschaffen and Kales[2] have recently been revised and updated by the American Academy of Sleep Medicine.[3] There are few published reports of comprehensive sleep architecture and respiratory event data from PSG using standard scoring criteria for OSA patients in Singapore.

OSA is a polysomnographic finding, characterised by episodes of partial (hypopnoea) or complete (apnoea) upper airway obstruction during sleep. When hypopnoeas and apnoeas occur frequently, and are associated with

[1] Singapore Neurology & Sleep Centre, Gleneagles Medical Centre, Singapore
[2] Sleep Disorders Unit, Singapore General Hospital, Singapore
[3] Department of Endocrinology, Singapore General Hospital, Singapore
[4] Obesity Unit, Singapore General Hospital, Singapore
[5] Department of Clinical Research, Singapore General Hospital, Singapore
Address for Correspondence: Dr Lim Li Ling, Singapore Neurology & Sleep Centre, Gleneagles Medical Centre #04-19, 6 Napier Road Singapore 258499.
Email: drlimliling@gmail.com

symptoms such as excessive daytime sleepiness, the terms *obstructive sleep apnoea syndrome (OSAS)* or *obstructive sleep apnoea-hypopnoea syndrome (OSAHS)* are applied.[4] The number of apnoeas and hypopnoeas on PSG are quantified as the apnoea-hypopnoea index (AHI). Typically, a patient with OSAS may present with snoring, daytime somnolence, choking or gasping during sleep, and witnessed apnoeas.[5]

The Wisconsin Sleep Cohort Study, the largest reported prevalence study with PSG among middle-aged adults, has reported an estimated prevalence of OSAS (AHI ≥5 and excessive daytime sleepiness) of 2% in women, and 4% in men. In this study, male gender and obesity were strongly associated with the presence of sleep-disordered breathing.[6] Male gender as a risk factor for OSAS may be related to the differential fat distribution in men (mainly in the neck and abdomen) compared to women (fat distribution in the hips and legs). Obesity is another major risk factor for OSAS. The World Health Organization (WHO) defines normal weight as a body mass index (BMI) of 18.5 to 24.9, overweight as a BMI of 25.0 to 29.9, and obesity as a BMI of 30.0 to 39.9.[7] In one Australian population study, a BMI in the obese range (>30) was associated with up to 18 times the risk of OSA compared to a BMI of <25.[8] In Singapore, one recent local study of severely obese candidates for bariatric surgery reported a very high prevalence of OSA of 72%.[9]

Our goal was to study anthropomorphic and PSG (including sleep architecture and respiratory event data) characteristics in local patients undergoing evaluation for suspected OSA. We have observed that OSA patients in Singapore are less frequently obese than in western populations, and that similar to large prevalence studies elsewhere, OSA appears much less commonly in women. We sought to examine the effects of OSA on sleep architecture, the correlation between OSA and BMI, and to compare sleep and respiratory parameters between genders, and between the various categories of severity in the spectrum of sleep-disordered breathing i.e. Normal Overall AHI (AHI <5), and those with Mild (AHI 5-15), Moderate (AHI >5-30) and Severe (AHI >30) OSA.

Materials and Methods

This was a retrospective analysis of PSG data in a large group of patients with suspected OSA who underwent comprehensive diagnostic PSG evaluation in the Sleep Laboratory of a public tertiary hospital. This study was approved by the participating hospital's Institutional Review Board. Patients were recruited from a consecutively encountered patient population seen over 1 year period at the Singapore General Hospital Sleep Disorders Unit. This is a 6-bedded Sleep Laboratory of a tertiary hospital, staffed by a multi-disciplinary team of sleep physicians, including neurologists, respiratory physicians, ENT surgeons and a psychiatrist. PSG was performed for patients with clinically suspected OSA who experienced any one of the following sleep-related symptoms: Excessive daytime sleepiness, snoring, witnessed apnoeas, choking or gasping in sleep, sleep maintenance insomnia, or unrefreshing sleep.

A standard polygraph (Compumedics E Series) was used to record the electroencephalogram (EEG), electro-oculogram (EOG), electromyogram (EMG) of the chin and bilateral tibialis anterior muscles, electrocardiogram (ECG), airflow measurement, chest wall and abdominal movements. Airflow was measured using nasal and oral thermistors, and a nasal pressure transducer. Respiratory effort was monitored with piezo chest and abdominal belts. Oximetry was measured using a disposable finger probe placed on the index finger. Snoring was recorded using snore microphones attached to the neck. Audiovisual information was obtained using simultaneous video recording.

All studies were analysed by trained PSG technicians and sleep physicians using the criteria of Rechtschaffen and Kales, and in close concordance with scoring updates given by the American Academy of Sleep Medicine .[2,3] The traditional Rechtschaffen and Kales terminology for the 5 sleep stages (i.e. stages 1, 2, 3, 4, and REM sleep, with stages 1 and 2 collectively referred to as "light sleep", stages 3 and 4 collectively referred to as "deep sleep") were used in this report. Apnoeas were scored when there was a complete cessation of airflow or ≥90% drop in the peak thermal sensor excursion for at least 10 seconds. Hyponoeas were scored when there was a drop in nasal pressure signal excursion by ≥30% of baseline lasting at least 10 seconds with a ≥4% desaturation from pre-event baseline, or when there was a drop in nasal pressure signal excursion by ≥50% of baseline lasting at least 10 seconds with or without a ≥4% desaturation from pre-event baseline.

Statistical Analysis

The PSG data were analysed collectively, and then grouped according to gender for a comparison between males and females, as well as grouped according to severity of sleep-disordered breathing for a comparison of sleep architecture between the groups. Comparisons of sleep parameters between gender and severity groups were tested by the non-parametric Kruskal-Wallis test, with statistical significance taken as P <0.05. Pearson correlation coefficient was used to assess the association between AHI and BMI. All analyses were done using SPSS® for Windows.

Table 1. Summary Statistics: Polysomnography Data in Patients Evaluated for Obstructive Sleep Apnoea

Demographics/Sleep parameters (N = 584)	Median	Range
BMI (kg/m^2)	26.8	14.0, 63.3
Age (y)	49.0	14.0, 90.0
Sleep latency (min)	11.5	0, 182.5
REM latency (min)	104.0	0, 605
Sleep efficiency (%)	82.6	0, 98.3
Light sleep (%) (stages 1, 2)	64.0	15.4, 100
Deep sleep (%) (stages 3, 4)	18.6	0, 62.5
REM sleep (%)	16.9	0, 75.1
Total arousal index	25.1	0, 43.1
Respiratory arousal index	9.2	0, 106.7
PLM arousal index	0.0	0, 64
AHI	14.7	0, 150.6
Total sleep time in supine position (%)	75.2	0, 100
Supine AHI	16.9	0, 150.6
REM AHI	16.7	0, 134
NREM AHI	12.9	0, 207
Minimum oxygen saturation	85.0	33, 98
PLM index	0.0	0, 140.5

AHI: apnoea-hypopnoea index; BMI: body mass index; PLM: periodic limb movement; REM: rapid eye movement

Results

Our findings are summarised in Tables 1, 2 and 3, and Figure 1.

Collective Dataset (Table 1)

A total of 584 patients with suspected OSA underwent comprehensive diagnostic PSG in 2005 in the Sleep Laboratory of a tertiary hospital. There were 449 male patients (76.9%) and 135 female patients (23.1%), with a median age of 49.0 years (SD 12.7; range, 14 to 90). Male patients were on average younger than the female patients, 46.1 years old in men compared to 52.0 years in women (P <0.0005). The mean BMI was 27.9 kg/m^2 (SD 6.7; range, 14 to 63.3). The BMI was not significantly different between the male and female patients (P = 0.145).

Sleep architecture was analysed. The following sleep parameters of sleep onset latency, rapid eye movement (REM)-sleep onset latency, sleep efficiency (including light sleep, deep sleep and REM sleep), AHI, lowest oxygen saturation, supine AHI, REM AHI, non-rapid eye movement (NREM) AHI, percentage back time (sleeping in the supine position), periodic limb movement (PLM) index, arousal index, and PLM-arousal index are summarised in Table 1.

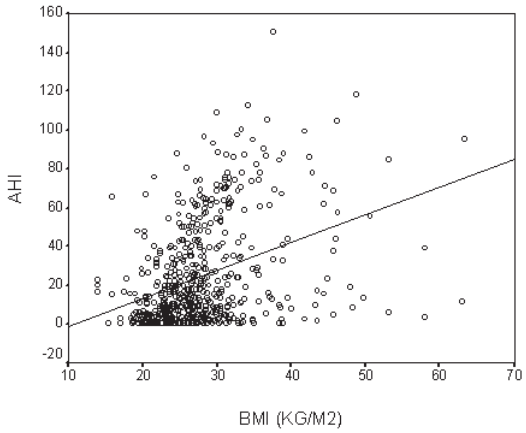

Fig. 1. Correlation between AHI and BMI.

The association between the overall AHI and BMI was analysed. Pearson correlation coefficient indicated a fair degree of correlation between AHI and BMI (r = 0.362, Fig. 1).

Comparison by Gender (Table 2)

Comparing respiratory events between genders, male patients had overall significantly higher AHI (16.5 vs 9; P <0.0005), higher supine AHI (20.4 vs 9.0; P <0.0005), and higher NREM AHI (15.9 vs 7.1; P <0.0005) than females. Comparing sleep architecture, male patients had shorter median sleep onset latency (11 vs 16.5 minutes; P <0.0005), more light sleep (65.3% vs 59.3%; P <0.0005), less deep sleep (17.7% vs 23%; P <0.0005), higher mean (total) arousal indices (26.9 vs 19.9; P = 0.001) and more respiratory event related arousals (11.6 vs 5.1; P <0.0005).

There was no significant difference in the overall sleep efficiency, REM sleep onset latency, percentage of REM sleep, REM AHI, percentage back time (sleeping time in supine position), PLM-index or PLM-arousal index comparing male and female patients.

Comparison by Severity: Comparison between Normal Overall AHI, Mild, Moderate and Severe OSA (Table 3)

The 584 patients were compared according to severity of sleep-disordered breathing: AHI <5 ("Normal Overall AHI"), AHI 5-15 ("Mild OSA"), AHI >15-30 ("Moderate OSA"), AHI >30 ("Severe OSA"). There was no difference in age among the 4 groups, but BMI was significantly higher with greater severity of OSA (P <0.0005; BMI for the 4 OSA groups as in Table 3). Patients with normal overall AHI comprised 28.3% of the patients evaluated, the remainder of the patients were categorised as 22.3% mild, 18.3% moderate and 31.2% severe OSA. The more severe

Table 2. Comparison of Polysomnography Data Between Male and Female Patients

Demographics/Sleep parameters	Statistics	Gender		*P* value
		Female (N = 135)	Male (N = 449)	
BMI (kg/m^2)	Median	26.2	26.9	0.145
	Minimum	14.0	15.5	
	Maximum	63.3	58.1	
Age (y)	Median	54.0	47.0	<0.0005
	Minimum	23.0	14.0	
	Maximum	83.0	90.0	
Sleep onset latency (min)	Median	16.5	11.0	<0.0005
	Minimum	0.0	0.0	
	Maximum	141.0	182.5	
REM onset latency (min)	Median	109.0	104.0	0.206
	Minimum	0.0	0.0	
	Maximum	605.0	458.5	
Sleep efficiency (%)	Median	81.9	82.7	0.315
	Minimum	0.0	2.0	
	Maximum	97.3	98.3	
Light sleep (%) stages 1, 2	Median	59.3	65.3	<0.0005
	Minimum	23.8	15.4	
	Maximum	100.0	100.0	
Deep sleep (%) stages 3, 4	Median	23.0	17.7	<0.0005
	Minimum	0.0	0.0	
	Maximum	52.6	62.5	
REM sleep (%)	Median	18.1	16.5	0.099
	Minimum	0.0	0.0	
	Maximum	75.1	55.0	
Total arousal index	Median	19.9	26.9	0.001
	Minimum	0.0	0.9	
	Maximum	95.4	43.1	
Respiratory arousal index	Median	5.1	11.6	<0.0005
	Minimum	0.0	0.0	
	Maximum	92.2	106.7	
PLM arousal index	Median	0.0	0.0	0.608
	Minimum	0.0	0.0	
	Maximum	47.4	64.0	
AHI	Median	9.0	16.5	<0.0005
	Minimum	0.0	0.0	
	Maximum	100.4	150.6	
Total sleep time in supine position (%)	Median	74.4	75.4	0.996
	Minimum	0.0	0.0	
	Maximum	100.0	100.0	

Table 2. Contd.

Demographics/Sleep parameters	Statistics	Gender		*P* value
		Female (N = 135)	Male (N = 449)	
Supine AHI	Median	9.0	20.4	<0.0005
	Minimum	0.0	0.0	
	Maximum	103.4	150.6	
REM AHI	Median	11.6	17.5	0.141
	Minimum	0.0	0.0	
	Maximum	120.0	134.0	
Non-REM AHI	Median	7.1	15.9	<0.0005
	Minimum	0.0	0.0	
	Maximum	105.6	207.0	
Minimum oxygen saturation	Median	86.0	85.0	0.067
	Minimum	38.0	33.0	
	Maximum	98.0	97.0	
PLM-Index	Median	0.0	0.0	0.510
	Minimum	0.0	0.0	
	Maximum	71.0	140.5	

AHI: apnoea-hypopnoea index; BMI: body mass index; PLM: periodic limb movement; REM: rapid eye movement

OSA patients were also significantly heavier, and had more severely affected sleep architecture: median (range) for BMI in Normal, 24.5 (15.5, 58.1), vs Mild, 26.2 (19.0, 62.9), vs Moderate, 26.4 (14.0, 47.9), vs Severe OSA, 29.3 (15.8, 63.3), (P <0.0005). While sleep efficiency, sleep onset latency and REM onset latency were similar in all groups, there was significantly more light sleep (P <0.0005, Table 3), less deep sleep (P <0.0005, Table 3), less REM sleep (P <0.0005, Table 3), more respiratory event related arousals (P <0.0005, Table 3) and lower oxygen desaturation in the more severe OSA patients (P <0.0005, Table 3). Of note, the more severe OSA patients spent more time in the supine position (P <0.0005, Table 3).

PLM-indices were significantly higher in the "normal" AHI group (P <0.0005, Table 3), also associated with a significantly higher PLM-arousal index in the same group, albeit still within the normal range: median (interquartile range) for PLM-arousal index in Normal Overall AHI, 0 (0, 2.1), vs Mild, 0 (0, 2.0), vs Moderate, 0 (0, 0.9), vs Severe OSA groups, 0 (0, 0), (P <0.0005).

PSG data from patients with normal overall AHI are summarised in Table 3. This group which did not meet the PSG cut-off criteria for OSA (i.e. overall AHI ≥5) was categorised as either "Normal", or "Primary Snoring" if snoring was recorded from the snore microphone, or as REM sleep related or positional OSA if the REM AHI or supine AHI, respectively, were ≥5. Briefly, the following median values for this category of patients are summarised below and compared to the sleep architecture of the entire group overall: sleep onset latency 11 vs overall 11.5 minutes, REM sleep onset latency 98 vs overall 104 minutes, sleep efficiency 82.9% (with 56.6% light sleep, 23.9% deep sleep and 19.1% REM sleep), AHI of 1.6 (median oxygen saturation 92%), supine AHI 1.7 (range, 0 to 24.4), REM AHI 2 (range, 0 to 35), NREM AHI 1.2 (range, 0 to 207), percentage back time 70.4% (range, 0 to 100%), PLM index 0.7 (range, 0 to 82.6), arousal index 13, respiratory arousal index 0.8 and PLM arousal index 0.

Discussion

These findings from a large public tertiary hospital in Singapore seeing predominantly local patients indicate that symptoms suggestive of OSAS are seen more commonly in middle-aged, overweight men than in women, who tend to be older. The male predominance is a consistent finding across OSAS populations, likely partly related to android-type fat distribution. Older women are also at increased risk because of weight gain and the post-menopausal status, which is known to confer increased risk of OSA.[10,11] The majority of patients (49.5%) presenting to our tertiary facility had moderate to severe OSA with AHI >15. This higher respiratory disturbance index (>20) has been shown in one study of cumulative survival to increase mortality compared to OSA patients with an index below 20.[12]

Overall, the median BMI for all categories of severity in

Table 3. Comparison of Polysomnography Data Between the Severity Spectrum of OSA Patients

Demographics/ Sleep parameters	Statistics	Severity				*P* value
		Normal Overall AHI (N = 165)	Mild AHI (N = 130)	Moderate AHI (N = 107)	Severe AHI (N = 182)	
BMI (kg/m^2)	Median	24.5	26.2	26.4	29.3	<0.0005
	Minimum	15.5	19.0	14.0	15.8	
	Maximum	58.1	62.9	47.9	63.3	
Age (y)	Median	47	51	49.5	48.5	0.114
	Minimum	14	20	20	19	
	Maximum	82	74	90	79	
Sleep latency (min)	Median	11	13.5	13.25	10.75	0.069
	Minimum	0	0	0.5	0	
	Maximum	142	182.5	141	93.5	
REM latency (min)	Median	98	103.5	102.5	111	0.580
	Minimum	0	2	11	0	
	Maximum	458.5	605	352	417	
Sleep efficiency (%)	Median	82.9	81.65	82.1	83.5	0.325
	Minimum	0	29.6	0	2	
	Maximum	97.3	97.3	96.9	98.3	
Light sleep (%)	Median	56.6	60.9	63.6	76	<0.0005
	Minimum	17.4	15.4	23.8	32.8	
	Maximum	100	94.8	100	100	
Deep sleep (%)	Median	23.9	21	18.4	9.3	<0.0005
	Minimum	0	0.1	0	0	
	Maximum	62.5	47	42.4	33	
REM sleep (%)	Median	19.1	18.35	16.6	14.3	<0.0005
	Minimum	0	2.1	0	0	
	Maximum	43.3	55	75.1	41.8	
Total arousal index	Median	13	17.95	27.3	54.8	<0.0005
	Minimum	0	2.8	0	1.3	
	Maximum	187	79.9	61.1	43.1	
Respiratory arousal index	Median	0.8	5.75	15.3	45.15	<0.0005
	Minimum	0	0	0	0	
	Maximum	4	33.3	27.7	106.7	
PLM arousal index	Median	0	0	0	0	<0.0005
	Minimum	0	0	0	0	
	Maximum	36.1	37	22.7	64	

both genders fell in the overweight (by WHO international guidelines) rather than the obese range. There was a fair degree of correlation between AHI and BMI, with more severe OSA patients tending to have a higher BMI. The relatively lower range of BMI in local OSA patients parallels the differences in BMI cut-off points recommended in Asian patients for a range of metabolic and cardiovascular disorders. Asian populations have been found to have higher percentages of body fat at a given BMI (about 3% to 5% higher for the same BMI) compared to Caucasians.[13] Local studies have shown that for the same amount of body fat as Caucasians with a BMI of 30 kg/m^2 (the cut-off BMI

Table 3. Contd.

Demographics/ Sleep parameters	Statistics	Severity				*P* value
		Normal Overall AHI (N = 165)	Mild AHI (N = 130)	Moderate AHI (N = 107)	Severe AHI (N = 182)	
AHI	Median	1.6	9.25	21.1	57.9	< 0.0005
	Minimum	0	1.2	14.9	30.4	
	Maximum	4.9	15.0	29.8	150.6	
Total sleep time in supine position (%)	Median	70.4	71.85	73.9	83.6	0.012
	Minimum	0	0	0	0	
	Maximum	100	100	100	100	
Supine AHI	Median	1.7	10.85	27.45	63	< 0.0005
	Minimum	0	0	0	0	
	Maximum	24.4	47.7	87.5	150.6	
REM AHI	Median	2	14.5	22.8	52.4	< 0.0005
	Minimum	0	0	0	0	
	Maximum	35	62.3	75.5	134	
NREM AHI	Median	1.2	7.95	20.3	60.4	< 0.0005
	Minimum	0	0.9	0	20.2	
	Maximum	207	20.3	37.7	150.6	
Minimum oxygen saturation	Median	92	87	82	71.5	< 0.0005
	Minimum	69	40.4	38	33	
	Maximum	98	95	94	92	
PLM index	Median	0.7	0	0	0	< 0.0005
	Minimum	0	0	0	0	
	Maximum	82.6	101	140.5	82.3	

AHI: apnoea-hypopnoea index; BMI: body mass index; PLM: periodic limb movement; REM: rapid eye movement

for obesity as defined by WHO), the BMI cut-off points for obesity should be about 27 kg/m^2 for Chinese and Malays, and 26 kg/m^2 for Indians.[14] In 2000, the WHO convened an expert consultation which concluded that there is a substantial proportion of Asian people who have a high risk of type 2 diabetes and cardiovascular disease at BMIs lower than the existing WHO BMI cut-off point for overweight of ≥25 kg/m^2. The panel recommended that for some Asians, BMI of 23 kg/m^2 or higher marks a moderate increase in risk for these diseases while a BMI of 26 kg/m^2 or more represents high risk.[15] Based on these local studies and the WHO Expert Consultation, the Singapore Ministry of Health (MOH) in 2005 has recommended that the BMI cut-off points for public health action and clinical interventions be revised to 23-27.4 ("Moderate Risk") and 27.5 ("High Risk"), with a BMI of 18.5-22.9 being the healthy range.[16] Our finding of moderate to severe OSA in a majority of local patients screened for sleep-disordered breathing with BMI ranges in the moderate to high risk categories as defined by the MOH (i.e. in the WHO overweight rather than the obese range) supports this view. American studies which have compared Asian and white OSA patients have found that Asians are younger with a lower mean BMI (26.6 ± 3.7 in the Asians vs 30.7 ± 5.9 in the whites).[17] The mean respiratory disturbance index was similar in both groups, being in the severe range (56.6 ± 34.9 vs. 55.6 ± 26.9). In comparison, data from regional Asian populations have shown similar clinical OSA features to Caucasian data.[18,19] In Hong Kong Chinese, obesity was found to be a risk factor for OSA – with a mean BMI of 30.4, higher than the average local population, but also lower than Caucasian OSA patients. Comparing severity of sleep-disordered breathing, Asian patients with OSA have been found to have greater severity of their illness compared to Caucasian patients matched for age, gender and BMI.[20]

Besides differences in body fat for BMI between Asian and Caucasians, Asian OSA patients also have been found to have distinct craniofacial features based on cephalometric data, such as maxillomandibular protrusion, narrower cranial base angle, larger posterior airway space, and more

superiorly positioned hyoid bone compared with Caucasians.[17] In Singapore, a local prospective study of 106 Southeast Asians found some similar anatomical characteristics which may contribute to the upper airway obstruction of OSA.[21] These unique features included a longer lower-facial length, narrower skull base, smaller and receding mandible, smaller posterior airway space, narrower retropalatal space, longer and thicker soft palate, smaller hard and soft palate angles, longer tongue length and more inferiorly displaced hyoid. Overall, the unique Asian body fat and craniofacial characteristics are likely to contribute to the greater severity of sleep-disordered breathing at a relatively lower range of BMI in OSA patients in Singapore.

In Singapore, one study has suggested that OSAS is much more common locally than the oft quoted 2% to 4% prevalence figure from the landmark Wisconsin Sleep Cohort Study.[6,22] This study has estimated that OSAS affects as many as up to 15% of the population. In our local context, therefore, the burden of OSA is expected to be large, and probably an overlooked health problem. The markedly disturbed sleep architecture we have documented contributes to chronic partial sleep deprivation, with consequent well-recognised impaired neurocognitive function, increased risk for metabolic and cardiovascular diseases and overall diminished quality of life. This abnormal sleep architecture is worse with greater severity of OSA, and includes diminished deep sleep and REM sleep, more light sleep and recurrent arousals.

In summary, OSA represents a considerable and likely under-recognised health problem in Singapore, which is also reversible with behavioural, non-invasive and surgical treatments. Considering the increased morbidity and mortality associated with OSA, greater awareness of and a high index of suspicion for OSA is needed in local, including non-obese, patients with suggestive symptoms, with a view to early diagnosis and intervention to improve outcomes.

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