TECHNICAL REPORT

Validation study of a portable monitoring device for identifying OSA in a symptomatic patient population

KIN-WANG TO,[1] WING-CHI CHAN,[2] TAT-ON CHAN,[1] ALVIN TUNG,[1] JENNY NGAI,[1] SUSANNA NG,[1] KAH-LIN CHOO[2] AND DAVID S. HUI[1]

[1]*Respiratory Medicine Division, Department of Medicine and Therapeutics, Prince of Wales Hospital, The Chinese University of Hong Kong, and* [2]*Department of Medicine, North District Hospital, Hong Kong, China*

ABSTRACT

Background and objective: **Obstructive sleep apnoea syndrome (OSAS) is a common disorder associated with early atherosclerosis, diabetes mellitus, ischaemic heart disease and cerebrovascular disease. The gold standard for confirming OSAS is based on an attended overnight polysomnography (PSG) in a sleep laboratory; however lack of health-care resources creates long waiting times for patient access to this diagnostic test. This study evaluated the ability of a portable sleep-monitoring device to identify patients in Hong Kong with suspected OSAS.**

Methods: **Patients with symptoms of OSAS were invited to use the ARES (apnoea risk evaluation system) concurrently with an attended inpatient PSG. Several sets of AHI were generated by the ARES provider based on different oxygen desaturation criteria and surrogate parameters of arousal. The results were compared against PSG to determine the optimal sensitivity and specificity.**

Results: **There were 141 patients who completed the study successfully. Results of AHI from the ARES study were presented in the order of different scoring criteria—4% oxygen desaturation alone, obstructive events with 3% oxygen desaturation and obstructive events with 1% desaturation plus surrogate arousal criteria. The sensitivity was 0.84 (95% confidence interval (CI): 0.77–0.90), 0.89 (95% CI: 0.89–0.94) and 0.97 (95% CI: 0.94–0.99), respectively. The specificity was 1, 1 and 0.63 (95% CI: 0.55–0.71), respectively. The receiver operating curve had an area of 0.96, 0.97 and 0.98, respectively. The kappa coefficient varied from 0.24 to 0.55 for agreement of severity between PSG and ARES. The likelihood ratio positive and the likelihood ratio negative were 2.61, infinity, infinity and 0.16, 0.11, 0.05, respectively, in the order of oxygen desaturation described earlier.**

Conclusions: **The ARES device has reasonable sensitivity and specificity for diagnosing severe OSAS in symptomatic Chinese patients. There is moderate agreement between ARES and PSG in the diagnosis of severe disease, but less agreement in patients with mild/moderate disease.**

Key words: ambulatory monitoring, diagnosis, obstructive sleep apnoea, polysomnography, sensitivity and specificity.

INTRODUCTION

Obstructive sleep apnoea syndrome (OSAS) is a common disorder causing sleep fragmentation, daytime sleepiness, impaired cognitive function and poor health status.[1] OSAS is equally common among the middle-aged male Caucasian and Hong Kong Chinese populations, with prevalence rates of at least 4%.[2–4] There are growing data showing an association between OSAS and early atherosclerosis, diabetes mellitus, ischaemic heart disease and cerebrovascular disease,[5–11] constituting part of the metabolic syndrome.[12–15] The resultant public health impact is tremendous.[16,17] The gold standard for confirming OSAS is based on an attended overnight polysomnography (PSG) in a sleep laboratory.[18] This diagnostic procedure is labour-intensive and depends on the availability of hospital beds, the multichannel PSG equipment and the night attendants.[19] As a result of limited health-care resources in most countries, the waiting time for a sleep study is often lengthy.[20,21] Currently there are four types of diagnostic devices for suspected OSAS. Type I is the fully attended PSG (seven channels or more) in a laboratory setting. Type II consists of a minimum of seven channels, including electroencephalogram, electrooculography, chin electromyography, ECG or heart rate, airflow, respiratory effort and pulse oximetry. Type III consists minimally of four channels, including at least two channels of respiratory movement, or respiratory movement and airflow, heart rate or ECG and oxygen saturation. Type IV consists of a single parameter or two parameter devices.[22] The type III portable

Correspondence: David S. Hui, Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Prince of Wales Hospital, 30-32 Ngan Shing Street, Shatin, Hong Kong, China. Email: dschui@cuhk.edu.hk

Received 17 April 2008; invited to revise 16 May 2008, 18 June 2008; revised 2 June 2008, 24 June 2008; accepted 24 June 2008 (Associate Editor: Peter Eastwood).

doi: 10.1111/j.1440-1843.2008.01439.x

monitoring device is simple to set up and may reduce health-care costs and shorten the waiting time for reaching a diagnosis.[19,23] A recent joint systematic review and practice parameters by three professional societies in the USA have recommended future research focusing on developing different algorithms for managing patients with suspected OSAS.[21,22] This study compared the accuracy of a portable type III monitoring device against PSG for diagnosing OSAS in patients in Hong Kong.

METHODS

All patients with suspected OSAS in this study were initially referred by general practitioners or physicians for assessment at a specialist respiratory clinic. The study, which took place in 2007, was approved by the local ethics committee and registered in http://ClinicalTrials.gov, numbered NCT00628511. In-hospital sleep studies were arranged for those with significant sleepiness that interfered with their daily activities or for patients with two of the following symptoms: choking or gasping during sleep, recurrent awakenings from sleep, unrefreshed by sleep, daytime fatigue and impaired concentration.[24] During admission for PSG in the sleep laboratory, patients were invited to undergo concurrent type III diagnostic device assessment. Informed consent was obtained. Pregnant women, patients who declined to participate in the study or could not comply with the set-up of the device were excluded. The concurrent use of a type I and a type III device served as a validation for the type III device.

Type III device: ARES

The type III device being tested was an apnoea risk evaluation system (ARES, Advanced Brain Monitoring, Carlsbad, CA, USA).[25] The ARES received Food and Drug Administration clearance in October 2004. This device was chosen because it was feasible to perform concurrent PSG and ARES for validation. The ARES is a wireless physiological recorder that acquires nocturnal data. According to the service provider, the ARES Unicorder used in this study was a different version from the previous model described by Westbrook *et al*. that did not assess airflow and respiratory effort.[25,26] The ARES was worn on the forehead and measured blood oxygen saturation, pulse rate, airflow and respiratory effort, snoring levels, head movement and head position. After the patient had finished the concurrent study, the raw data were uploaded to the website of the service provider and then analysed by ARES Insight software. According to the service provider, this software applied pattern recognition algorithms to quantify obstructive respiratory events followed by manual editing by the service provider. Investigators in this study were unable to edit the ARES data and results. The service provider was blinded to the PSG results.

Patients also had to answer the ARES questionnaire that assessed the risk factors for OSAS, including age, gender, BMI, neck circumference, daytime sleepiness, frequency of snoring, observed apnoeas and history of hypertension. It estimated the pretests probability of OSA and identified any discrepancy between the physical data and questionnaire risk level, which implied the need for further confirmatory tests.[25] The final analysis combined the data of the physiological signals with the results of the ARES questionnaire to generate an overall risk level for OSAS. Accordingly, apnoea was defined as cessation of airflow for ≥10 s. Hypopnoea was defined as ≥50% reduction in airflow with either ≥4% or ≥3% desaturation from the baseline. One per cent desaturation was included if the obstructive event was accompanied by changes in pulse rate, head positions or snoring sounds, which implied arousals. Four sets of AHI values were presented in the ARES diagnostic report using the criteria: (i) 4% desaturation solely; (ii) both 4% and 3% desaturation; (iii) 4%, 3% and 1% desaturation; and (iv) all of (i)–(iii) including events suggestive of arousal even without oxygen desaturation. The principles behind the chosen percentage of desaturation were described in detail by Westbrook *et al*.[25] The ARES provider adopted a different severity grading for OSAS in which an ARES AHI of 0–5/h was labelled as normal, 6–20/h as mild OSAS, 21–40/h as moderate OSAS, 41–60/h as severe OSAS and over 61/h as very severe OSAS.

PSG

Standard full-night PSG (Siesta, Compumedics, Australia) was performed according to the recommendations of the American Academy of Sleep Medicine (AASM) Task Force. This included electroencephalogram, electrooculography, ECG, oxygen saturation, chest wall and abdominal strain gauges, submental and anterior tibialis electromyography. An airflow pressure transducer (Pro-Tech, PTAF2, Mukilteo, WA, USA) was used to measure airflow and snoring. All PSG were manually staged according to the criteria of Rechtschaffen and Kales.[27] The technician responsible for scoring PSG was blinded to the ARES results. For the PSG, obstructive apnoea was defined as a complete cessation of airflow lasting for ≥ 10 s with respiratory effort. Central apnoea was defined as cessation of airflow lasting for ≥ 10 s without respiratory effort. Mixed apnoea was defined as the cessation of airflow lasting for ≥ 10 s with both components of obstructive and central apnoea. Hypopnoea was defined as a reduction of airflow > 50% from baseline or a discernible reduction in respiratory airflow accompanied by a decrease of ≥ 3% in oxygen saturation and/or an arousal, lasting for ≥ 10 s.[24] The conventional grading of severity was adopted for reporting OSAS in the PSG: AHI 5–15/h = mild, 15–30/h = moderate and > 30/h = severe.

Statistical analysis

The *K* coefficient was used to assess the agreement between ARES AHI and PSG AHI, under the assumption that PSG with a clinical cut-off of AHI > 5 is

diagnostic of OSA. The discrepancy between the two diagnostic procedures was analysed using the method described by Bland and Altman.[28] Receiver operating characteristic curve was used to analyse different cut-off values of oxygen desaturation criteria for using the ARES as a diagnostic tool in the population of the study. All results are presented as mean (SD) unless stated otherwise.

RESULTS

A total of 184 patients were invited to participate in the study; nine refused to participate, so 175 patients underwent concurrent PSG and ARES. There were 21 failures (12%) in the ARES (with recording time < 4 h), 8 failures (4.5%) in PSG and 5 failures (2.9%) with both PSG and ARES. The most common cause for ARES failures was poor signal output. Other causes included very short total sleeping time and machine failure. The characteristics of the patients are described in Table 1. There were more men than women, but a similar proportion of each gender had comorbid conditions.

All patients had predominant OSAS. The number of obstructive apnoeas/h was 21.4 (22.7), central apnoeas/h was 1.1 (1.9) and mixed apnoeas/h was 2.5 (7.5). The false-negative rate was 73% using oxygen desaturation ≥ 4% as the only scoring criterion, but there were no false positives. The *K* coefficient between ARES AHI and PSG AHI was 0.24, $P < 0.01$. When oxygen desaturation ≥ 3% was adopted as the scoring criterion, the false-negative rate became 65.2%, although there were no false positives. The *K* coefficient between ARES AHI and PSG AHI was 0.3, $P < 0.01$. When 1% desaturation was included as the scoring criterion, the false-negative rate became 44% whereas the false-positive rate became 37.5%. The *K* coefficient between ARES AHI and PSG AHI was 0.55, $P < 0.01$. The concordance between ARES AHI and PSG AHI across different oxygen desaturation criteria is shown in Table 2. Bland–Altman plot showed that the ARES AHI was lower than the corresponding PSG AHI. The difference between the two AHI was reduced when the 1% scoring criterion was used in the ARES (Figs 1,2). The sensitivity and specificity, with different levels of oxygen desaturation as scoring criteria, varied from 0.84 to 0.97 and 0.63 to 1, respectively. The likelihood ratio positive (LR+) and likelihood ratio negative (LR–) varied from 1.15 to infinity and 0 to 0.16, respectively (Table 3). The area under curve of the receiver operating curve was 0.96 to 0.98 (Fig. 3).

Table 1 Characteristics of the 175 patients involved in the apnoea risk evaluation system validation

	Men ($n = 132$)	Women ($n = 43$)
Age, years (mean ± SD)	47.8 ± 9.8	52.3 ± 12.2
BMI (mean ± SD)	28.5 ± 4.9	29.2 ± 6.0
Epworth sleepiness scale (mean ± SD)	9.8 ± 5.3	12.2 ± 5.0
PSG AHI (mean ± SD)	41.6 ± 26.9	32.2 ± 22.9
Major co-morbid condition,† *n* (%)		
Hypertension	62 (47.6)	23 (54.8)
Diabetes mellitus	20 (15.1)	7 (16.7)
Hyperlipidaemia	20 (15.1)	5 (11.9)
Fatty liver	12 (9)	6 (14.3)
Cerebrovascular accident	9 (6.8)	2 (4.8)

† Premorbid conditions did not add up to 100% because some patients had multiple conditions.

PSG, polysomnography.

DISCUSSION

This study has shown that the ARES device has a reasonable sensitivity and specificity for diagnosing OSAS in symptomatic patients when compared with conventional PSG. When examining the change in agreement and determining which of the oxygen desaturation scoring criteria would give the best sensitivity and specificity, the 3% oxygen desaturation criterion was seen to yield the most optimal sensitivity and specificity. This is inherently expected as the same oxygen desaturation criterion was used in PSG. In general, the sensitivity and specificity of the present study were similar to other studies of type III portable sleep devices, which were between 85% and 100%.[21] Both the high positive predictive value and LR+ of ARES results, based on the 3% oxygen desaturation criterion, reflected that a positive result was likely to confirm the diagnosis. No patient in the group of severe OSA was misdiagnosed as normal

Table 2 The apnoea risk evaluation system (ARES) AHI versus the polysomnography (PSG) AHI with different levels of oxygen desaturation as scoring criteria

	PSG AHI				
ARES AHI†	Normal	Mild	Moderate	Severe	Total
Normal (0–5)	8/8/5	14/11/2	8/4/2	0/0/0	30/23/9
Mild (6–20)	0/0/3	8/11/20	20/22/12	11/3/0	39/36/35
Moderate (21–40)	0/0/0	0/0/0	5/7/18	25/31/23	30/38/41
Severe (41 or above)	0/0/0	0/0/0	0/0/1	42/44/55	42/44/56
Total	8	22	33	78	141

† ARES AHI with 4% oxygen desaturation only/3% oxygen desaturation/1% oxygen desaturation with head movement included as scoring criteria.

Table 3 Prevalence of OSA determined by the ARES AHI at various oxygen desaturation levels using polysomnography AHI ≥ 5 for comparisons

Desaturation criteria (%)	Sensitivity (95% CI)	Specificity (95% CI)	PPV	NPV	LR+	LR–	AUC (95% CI)
4	0.84 (0.77–0.90)	1.00 (NA)	1.00	0.27	Infinity	0.16	0.96 (0.93–0.99)
3	0.89 (0.84–0.94)	1.00 (NA)	1.00	0.35	Infinity	0.11	0.97 (0.95–1.00)
1	0.97 (0.94–0.99)	0.63 (0.55–0.71)	0.98	0.56	2.61	0.05	0.98 (0.95–1.00)

ARES, apnoea risk evaluation system; AUC, area under curve; CI, confidence interval; LR+, likelihood ratio positive; LR–, likelihood ratio negative; NA, not applicable; NPV, negative predictive value; PPV, positive predictive value.

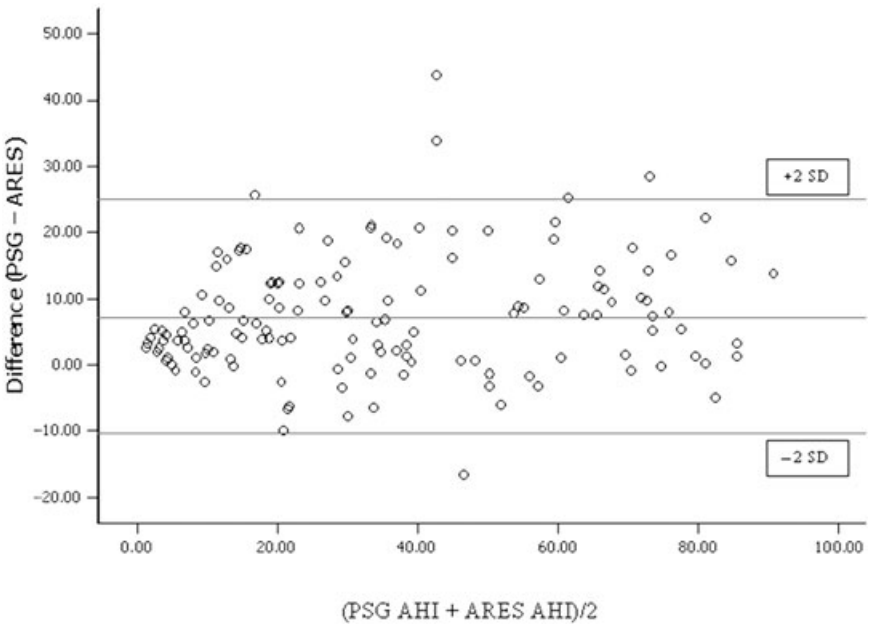

Figure 1 Bland–Altman plot using 3% desaturation as the only scoring criterion. ARES, apnoea risk evaluation system; PSG, polysomnography.

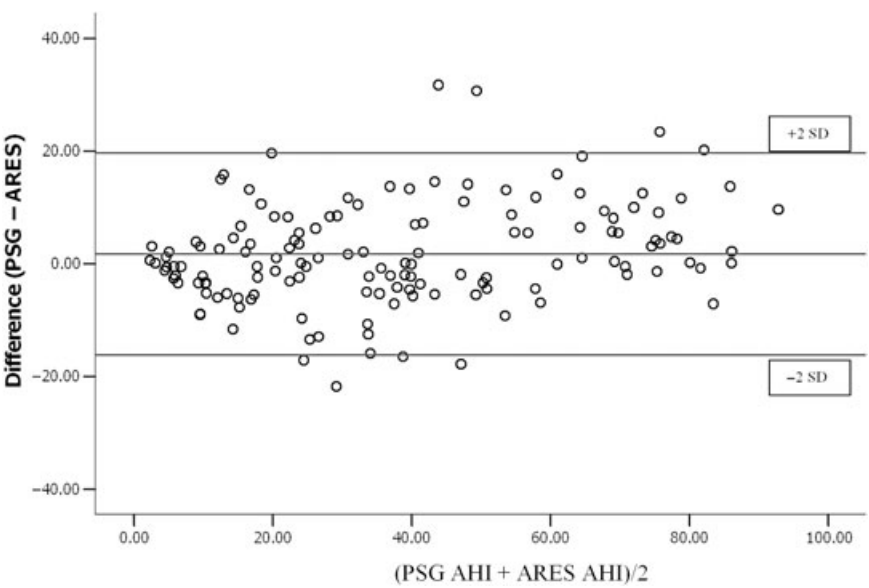

Figure 2 Bland–Altman plot with 1% desaturation and head movement included as the scoring criterion. ARES, apnoea risk evaluation system; PSG, polysomnography.

(Table 2). However, the relatively low negative predictive value at the 3% oxygen desaturation level implies that there was only 35% chance of excluding OSAS in a patient with negative ARES result in this population. Conversely, a negative ARES result still carried a 75% chance that the patient actually had OSAS. This was illustrated in Table 2 where 50% and 12% of the patients with mild and moderate OSA diagnosed by

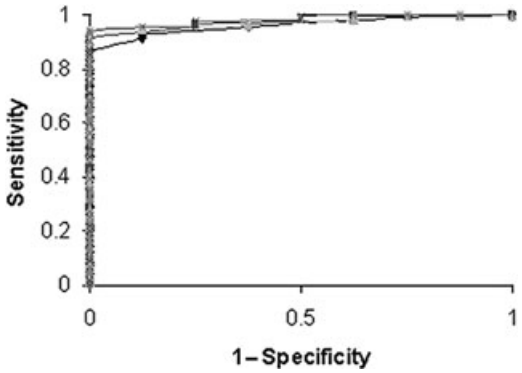

Figure 3 Receiver operating characteristics curve using different oxygen desaturation criteria. (–◆–) 4% desaturation; (–■–) 3% desaturation; (–▲–) 1% desaturation; (–×–) 0% desaturation.

PSG respectively were classified as normal by ARES. The false-negative rate was even higher when 4% oxygen desaturation was used as the diagnostic criterion. Thus, ARES did not accurately identify patients with mild/moderate OSA.

As both positive predictive value and negative predictive value depend on the prevalence of OSAS, the LR– is a better index to reflect the performance of a negative ARES result. In this case, LR– of 0.11 was only marginally powerful to alter the pretest probability. The LR– became 0.05 in the case of 1% oxygen desaturation as the scoring criterion. This implied that at this level of oxygen desaturation, a negative ARES result was more likely to be true even in patients with symptoms of OSAS. However, the drawback of using the 1% result was that the LR+ was only 2.61, which implied a positive ARES result would only be marginally more conclusive than clinical judgment in such patients. The corresponding high sensitivity and low specificity with this criterion were undesirable as 37.5% of normal subjects were then labelled as mild OSA by ARES (Table 2). Thus, these patients might be advised to try CPAP or a dental device unnecessarily. In addition, diagnoses other than OSA, which might cause daytime sleepiness, could be missed. Therefore, ARES was more useful in confirming the diagnosis in patients with severe OSA and was far less accurate in patients with mild/moderate OSA. The LR– value of the present study was comparable to that reported by Ayappa *et al*, although the specificity and LR+ values in the present study were higher whereas the sensitivity was lower, probably because these subjects were more homogenous.[26]

The agreement between ARES AHI and PSG AHI, as reflected by the *K* coefficient, was just fair to moderate. The ARES AHI tended to be lower than the PSG AHI in this study. One possible reason was that the ARES measured recording time, whereas PSG measured total sleep time. This could reduce the sensitivity and artificially increase the specificity.[21] The discrepancy between ARES and PSG was the greatest for patients with mild or moderate OSA, as shown in Table 2. This could be due to interrater differences in the interpretation of oxygen desaturation.[29,30] Using a similar device, Westbrook *et al*. reported the *K* coefficient as 0.77–0.85, which was much higher,[25] but the PSG in their paper was not considered as the 'gold standard'. In the present study, the *K* coefficient was determined by comparing the severity level between ARES and PSG, using the predefined severity of the service provider and the conventional PSG grading, which differed from each other. A discernible flow limitation followed by an arousal in PSG would be considered as hypopnoea even without a significant oxygen desaturation of ≥3% according to the AASM. Many obstructive events would have been excluded in ARES when only the 4% or 3% desaturation criterion was used. Therefore the agreement increased as the ARES scoring criteria became less stringent.

The *K* coefficient varied between 0.2 and 0.6 in different brands of type III device.[30] In one study focusing on the accuracy of a type III device, the *K* coefficient was found to be only 0.23.[31] Yet in another type III device using different AHI as cut-off, the *K* coefficient was found to be 0.5–0.6.[32] Thus the result of the present study was comparable to other studies. The drawback of the underestimation of ARES AHI is that if preferential treatment is offered according to the severity of ARES AHI, then some patients will be jeopardized, as illustrated by the 27–55% false-negative results and about 60% of moderate OSA patients would be labelled as mild OSA by ARES (Table 2). The optimal AHI cut-off for ARES diagnosing OSA was not determined in the present study because this has already been described by Westbrook *et al*.[25]

There were no significant side-effects related to application of the ARES. The most common complaint after application of ARES was mild redness of the forehead skin due to pressure of the sensor, which subsided spontaneously. A few patients complained of discomfort at the nostrils due to the simultaneous application of ARES and PSG flow sensors. Otherwise there was no report of other safety issues. Most patients could tolerate the simultaneous application of ARES and PSG.

The present study has several limitations. First, the ARES arousal was determined by head movement with pulse rate changes following flow limitation and oxygen desaturation. This could be a possible limitation in patients with heart diseases, autonomic neuropathy or those on beta blocker, who might not have pulse rate changes after arousal. Moreover, microarousal without head movement could have been missed. Second, this study was performed in patients with symptomatic OSAS. The results are not applicable if ARES is to be used as a screening tool in asymptomatic populations.[25,26] Therefore it is more appropriate to apply the ARES device in a clinical setting where the majority of patients are symptomatic, as in a tertiary centre, rather than using the device as a screening tool in the general population. As a tertiary centre receiving referrals from other physicians, the population in the present study was highly selected and did not represent the general population. Finally, the failure rate was higher in the present study, probably because two nasal cannulae were used and this might have caused discomfort and poor signal collection in some cases. Nevertheless,

the flow signal quality was satisfactory in the majority of cases and posed no significant problems for scoring respiratory events in the PSG. This study was performed in an attended setting. The accuracy would be expected to reduce whereas the failure rate would be higher in an unattended setting such as the home, as no technician would be available to correct potential problems.

A portable monitoring device with simple applicability such as the ARES is an alternative to PSG for confirming the diagnosis in symptomatic patients suspected of severe OSA and has the potential to reduce health-care resource use. The poor negative predictive value and LR– could not rule out OSA in symptomatic patients and a negative or mild result would require a standard PSG to rule out the condition. On the contrary, a significant positive result (moderate or severe) is likely to identify patients with severe OSA, but not patients with mild/moderate OSA given the poor accuracy in this group.

ACKNOWLEDGEMENTS

This study was funded by the Hong Kong Lung Foundation and the Respiratory Research Fund of The Chinese University of Hong Kong.

REFERENCES

1 Engleman HM, Douglas NJ. Sleep. 4: sleepiness, cognitive function, and quality of life in obstructive sleep apnoea/hypopnoea syndrome. *Thorax* 2004; **59**: 618–22.
2 Young T, Palta M, Dempsey J, Skatrud J, Weber S *et al.* The occurrence of sleep-disordered breathing among middle-aged adults. *N. Engl. J. Med.* 1993; **328**: 1230–5.
3 Bearpark H, Elliott L, Grunstein R, Cullen S, Schneider H *et al.* Snoring and sleep apnea. A population study in Australian men. *Am. J. Respir. Crit. Care Med.* 1995; **151**: 1459–65.
4 Ip MSM, Lam B, Tang LCH, Lauder IJ, Ip TY *et al.* A community study of sleep-disordered breathing in middle-aged Chinese women in Hong Kong: prevalence and gender differences. *Chest* 2004; **125**: 127–34.
5 Suzuki T, Nakano H, Maekawa J, Okamoto Y, Ohnishi Y *et al.* Obstructive sleep apnea and carotid-artery intima-media thickness. *Sleep* 2004; **27**: 129–33.
6 Shamsuzzaman ASM, Gersh BJ, Somers VK. Obstructive sleep apnea: implications for cardiac and vascular disease. *JAMA* 2003; **290**: 1906–14.
7 Wolk R, Somers VK. Cardiovascular consequences of obstructive sleep apnea. *Clin. Chest Med.* 2003; **24**: 195–205.
8 Dart RA, Gregoire JR, Gutterman DD, Woolf SH. The association of hypertension and secondary cardiovascular disease with sleep-disordered breathing. *Chest* 2003; **123**: 244–60.
9 Lattimore J-DL, Celermajer DS, Wilcox I. Obstructive sleep apnea and cardiovascular disease. *J. Am. Coll. Cardiol.* 2003; **41**: 1429–37.
10 Bray GA. Medical consequences of obesity. *J. Clin. Endocrinol. Metab.* 2004; **89**: 2583–9.
11 Hui DS, Choy KL, Wong KS, Ko WS, Li ST *et al.* Prevalence of sleep-disordered breathing and continuous positive airway pressure compliance: results in Chinese patients with first-ever ischemic stroke. *Chest* 2002; **122**: 852–60.
12 Vgontzas AN, Bixler EO, Chrousos GP. Sleep apnea is a manifestation of the metabolic syndrome. *Sleep Med. Rev.* 2005; **9**: 211–24.
13 Punjabi NM, Polotsky VY. Disorders of glucose metabolism in sleep apnea. *J. Appl. Physiol.* 2005; **99**: 1998–2007.
14 Sasanabe R, Banno K, Otake K, Hasegawa R, Usui K *et al.* Metabolic syndrome in Japanese patients with obstructive sleep apnea syndrome. *Hypertens. Res.* 2006; **29**: 315–22.
15 Punjabi NM, Ahmed MM, Polotsky VY, Beamer BA, O'Donnell CP. Sleep-disordered breathing, glucose intolerance, and insulin resistance. *Respir. Physiol. Neurobiol.* 2003; **136**: 167–78.
16 Visscher TL, Seidell JC. The public health impact of obesity. *Annu. Rev. Public Health* 2001; **22**: 355–75.
17 Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am. J. Respir. Crit. Care Med.* 2002; **165**: 1217–39.
18 Kushida CA, Littner MR, Morgenthaler T, Alessi CA, Bailey D *et al.* Practice parameters for the indications for polysomnography and related procedures: an update for 2005. *Sleep* 2005; **28**: 499–521.
19 Chervin RD, Murman DL, Malow BA, Totten V. Cost-utility of three approaches to the diagnosis of sleep apnea: polysomnography, home testing, and empirical therapy. *Ann. Intern. Med.* 1999; **130**: 496–505.
20 Ng DK, Kwok K-L Chow P-Y, Cheung M-Y. Diagnostic access for sleep apnea in Hong Kong. *Am. J. Respir. Crit. Care Med.* 2004; **170**: 196; discussion 197.
21 Flemons WW, Littner MR, Rowley JA, Gay P, Anderson WM *et al.* Home diagnosis of sleep apnea: a systematic review of the literature. An evidence review cosponsored by the American Academy of Sleep Medicine, the American College of Chest Physicians, and the American Thoracic Society. *Chest* 2003; **124**: 1543–79.
22 Chesson AL Jr, Berry RB, Pack A, American Academy of Sleep Medicine, American Thoracic Society. Practice parameters for the use of portable monitoring devices in the investigation of suspected obstructive sleep apnea in adults. *Sleep* 2003; **26**: 907–13.
23 Reuven H, Schweitzer E, Tarasiuk A. A cost-effectiveness analysis of alternative at-home or in-laboratory technologies for the diagnosis of obstructive sleep apnea syndrome. *Med. Decis. Making* 2001; **21**: 451–8.
24 Flemons W, Buysse D, Redline S. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The report of an American academy of sleep medicine task force. *Sleep* 1999; **22**: 667–89.
25 Westbrook PR, Levendowski DJ, Cvetinovic M, Zavora T, Velimirovic V *et al.* Description and validation of the apnea risk evaluation system: a novel method to diagnose sleep apnea-hypopnea in the home. *Chest* 2005; **128**: 2166–75.
26 Ayappa I, Norman RG, Seelall V, Rapoport DM. Validation of a self-applied unattended monitor for sleep disordered breathing. *J. Clin. Sleep Med.* 2008; **4**: 26–37.
27 Rechtschaffen A, Kales A. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Brain Information Service, UCLA, 1968.
28 Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; **1**: 307–10.
29 Ramsey R, Mehra R, Strohl KP. Variations in physician interpretation of overnight pulse oximetry monitoring. *Chest* 2007; **132**: 852–9.
30 Collop NA, Anderson WM, Boehlecke B, Claman D, Goldberg R *et al.* Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. Portable monitoring task force of the American academy of sleep medicine. *J. Clin. Sleep Med.* 2007; **3**: 737–47.
31 Liesching TN, Carlisle C, Marte A, Bonitati A, Millman RP. Evaluation of the accuracy of SNAP technology sleep sonography in detecting obstructive sleep apnea in adults compared to standard polysomnography. *Chest* 2004; **125**: 886–91.
32 Dingli K, Coleman EL, Vennelle M, Finch SP, Wraith PK *et al.* Evaluation of a portable device for diagnosing the sleep apnoea/hypopnoea syndrome. *Eur. Respir. J.* 2003; **21**: 253–9.