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Research paper

Symptom profile of undiagnosed obstructive sleep apnoea in hypertensive outpatients in primary care: a structural equation model analysis

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ABSTRACT

Background Obstructive sleep apnoea (OSA) has been linked to hypertension in sleep clinic populations, but little is known about the symptom profile of undiagnosed OSA in hypertensive outpatients in primary care.

Aim To explore characteristics associated with undiagnosed OSA in hypertensive primary care patients.

Methods Cross-sectional design, including 411 consecutive patients (52% women), mean age 57.9 years (standard deviation [SD] 5.9 years), with diagnosed hypertension (blood pressure >140/90 mmHg) from four primary care centres. All subjects underwent a full-night, home-based, respiratory recording to establish the presence and severity of OSA. Clinical variables, medication and comorbidities, as well as data from self-rating scales regarding symptoms/characteristics, insomnia, excessive daytime sleepiness, depressive symptoms and health were collected during a clinical examination. Factor analyses and structural equation modelling (SEM) were used to explore the relationships between self-rated symptoms, clinical characteristics and objectively verified diagnosis of OSA.

Main outcome Measures symptom profile of undiagnosed OSA (as measured by the Apnoea/Hypopnoea Index [AHI]) in hypertensive outpatients in primary care.

Results Fifty-nine percent of the patients had an AHI $\geq$ 5/hour indicating OSA. An exploratory factor

analysis based on 19 variables yielded a six-factor model (anthropometrics, blood pressure, OSA-related symptoms, comorbidity, health complaints and physical activity) explaining 58% of the variance. SEM analyses showed strong significant associations between anthropometrics (body mass index, neck circumference, waist circumference) (0.45), OSA-related symptoms (snoring, witnessed apnoeas, dry mouth) (0.47) and AHI. No direct effects of OSA on comorbidities, blood pressure, dyssomnia or self-rated health were observed.

Conclusion OSA was highly prevalent and was directly associated with anthropometrics and OSA-related symptoms (snoring, witnessed apnoeas and dry mouth in the morning). When meeting patients with hypertension, these characteristics could be used by general practitioners to identify patients who are in need of referral to a sleep clinic for OSA evaluation.

Keywords: depression, health perception, hypertension, insomnia, obstructive sleep apnoea, sleep

How this fits in with quality in primary care

What do we know?
Obstructive sleep apnoea (OSA) is common and a growing problem in the general population. It is linked to cardiovascular disease. Treatment of OSA with continuous positive airway pressure can reduce blood pressure, morbidity and mortality, especially in severe OSA associated with daytime symptoms. However, general practitioners (GPs) have difficulties identifying patients in primary care who might have OSA and benefit from OSA treatment.

What does this paper add?
Undiagnosed OSA was highly prevalent among hypertensive outpatients in primary care. A total of 59% of the patients was identified as having an Apnoea/Hypopnoea Index $\geq$ 5/hour. Undiagnosed OSA had no direct association with comorbidities (ischaemic heart disease, diabetes, hypercholesterolemia), blood pressure, dyssomnia or self-rated poor health, as often described in sleep clinics populations. Undiagnosed OSA was directly associated with anthropometrics (body mass index, neck circumference, waist circumference) and OSA-related symptoms (snoring, witnessed apnoeas, dry mouth). These characteristics can be used by GPs to identify patients who should objectively be evaluated for OSA.

Background

Obstructive sleep apnoea (OSA) is a common sleep-related breathing disorder characterised by apnoeas and/or hypopnoeas.[1,2] Apnoeas and hypopnoeas are defined as total (apnoeas) or partial (hypopnoeas) obstruction of the upper airway leading to a cessation of airflow over the nose and mouth despite continued respiratory movements. The length of these events should be at least 10 seconds and hypopnoeas (but not apnoeas) have to be associated with blood oxygen desaturation. The severity of OSA is graded using the Apnoea/Hypopnoea Index (AHI) which is the average number of apnoeas and hypopnoeas per hour of sleep.[1] Dominating symptoms are loud snoring and witnessed breathing interruptions. Sleep fragmentation may cause daytime symptoms, such as excessive sleepiness, in which case the disease OSA syndrome (OSAS) occurs.[3] Insomnia, as well as depression have, however, also been linked to the presence of OSA. The prevalence of mild OSA (AHI $\geq$ 5 without daytime symptoms) has been estimated to be as high as 28%, and 4% of men and 2% of women in the general North American population suffer from OSAS.[1] OSA has been linked to hypertension[4] and cardiovascular disease (CVD).[5] A proposed mechanism is the sympathetic activation and increased levels of catecholamines causing inflammation, arterial stiffness and atherosclerosis,[6] due to apnoea-related oxygen desaturations. Another link between hypertension and OSA is the shared risk factor of obesity.[4] OSA can negatively affect the treatment of hypertension,[7] and has been shown to increase morbidity and mortality.[8] A previous study on men with therapy-resistant hypertension showed that as many as 56% had OSA,[9] compared with 19% of successfully treated hypertensive patients matched for age and gender. Continuous positive airway pressure (CPAP) is the treatment of choice and may reduce blood pressure[10] and cardiovascular morbidity and mortality in patients with severe OSAS.[11]

Despite knowledge of this high prevalence, difficulties in identifying patients with OSA have been described in primary care, causing low referral rates to sleep clinics.[12,13] Guidelines that describe flow charts

to identify patients in need of sleep evaluation and potential treatment have been published by the American Academy of Sleep Medicine (AASM).[14] The primary step of this flow chart is based on a routine health examination, patient complaints (e.g. sleep history, characteristics of OSA), as well as an evaluation of the occurrence of comorbidities associated with high risk of having OSA. Increased knowledge regarding new or unknown clinical characteristics and symptoms that are easy to collect and measure in a primary care setting may help to identify those who are in need of OSA evaluation/treatment. Early identification of undiagnosed OSA in newly diagnosed hypertension patients may prevent further development of atherosclerosis and future morbidity and mortality.[6] To the best of our knowledge, no studies have evaluated the association of characteristics included in the initial step of the AASM guidelines with the occurrence of undiagnosed OSA in hypertensive primary care patients. The aim of this study was therefore to explore characteristics associated with undiagnosed OSA in hypertensive primary care patients.

Material and methods

Design and selection criteria

A cross-sectional design was used (Figure 1). All patients, 18–65 years of age, with diagnosed hypertension (140/90 mmHg) at four primary care centres in Sweden received written and oral information about the study from a researcher without clinical contact with the patient, and those who gave informed consent were screened. Exclusion criteria were terminal disease, ongoing treatment for OSA or OSAS, severe psychiatric disease, dementia, alcohol or drug abuse, and difficulties reading or understanding the Swedish language. All data were collected during face-to-face interviews or examinations performed by an ear, nose and throat nurse and physician.

Clinical variables

Data regarding clinical variables (e.g. blood pressure), anthropometrics (weight, height, neck circumference, waist circumference), sleep (self-rated total sleep time and estimated sleep need), medication, OSA symptoms and comorbidities were collected. Diagnosis of diabetes mellitus was based on a history of diabetes, current treatment with antidiabetic drugs or repeated measures of fasting blood glucose values $\geq$ 7 mmol/l. Ischaemic heart disease (IHD) was defined as a history of angina pectoris and/or myocardial infarction and/or coronary angioplasty and/or coronary bypass surgery. Respiratory disease was defined as a history of asthma or chronic obstructive pulmonary disease, or patients who were on current treatment (β^2 agonists and/or inhaled corticosteroids). Transient ischaemic attack (TIA)/stroke was defined as a history of TIA and/or stroke. Snoring, morning headache and dry mouth were measured by 10-point scales (1–10, higher scores indicated more symptoms) used at the study site.

Self-rating scales

One question from the Berlin Sleep Apnoea Questionnaire (BSAQ) was used to measure witnessed apnoeas.[15] The respondent rates the frequency of witnessed apnoeas on a five-point scale (almost every night, 3–4 nights/week, 1–2 nights/week, 1–2 nights/month, never or almost never). The Minimal Insomnia Symptoms Scale (MISS) was used to measure insomnia.[16] The respondent was asked to rate difficulties initiating sleep, difficulties maintaining sleep and difficulties with non-restorative sleep on a five-point scale (0–4). The Epworth Sleepiness Scale (ESS) was used to measure daytime sleepiness.[17] The respondent was asked to assess the chance of falling asleep in eight different situations on a four-point scale (0–3). The Hospital Anxiety and Depression Scale (HAD) was used to measure depressive symptoms.[18] Seven of the items concern depressive symptoms and were scored on a four-point scale (0–3). The first question concerning current health status from the SF-36 was used to measure perceived health.[19] The participants ranked their health as: (1) excellent, (2) very good, (3) good, (4) fair or (5) poor.

Recordings of sleep-disordered breathing

Full-night respiratory recordings with monitoring of nasal airflow, pulse oximetry, respiratory movements and body position were performed in the patients' homes using polygraphy[14] (Embletta, ResMed AB, Trollhättan, Sweden). Apnoeas and hypopnoeas were manually scored by one researcher (OS) who was blinded with regard to other data. An apnoea was scored if the nasal pressure signal amplitude dropped $\geq$ 90% for $\geq$ 10 seconds and 90% of the event met amplitude reduction criteria. A hypopnoea was scored if the nasal pressure signal amplitude dropped $\geq$ 30%, oxygen-saturation dropped $\geq$ 4% for $\geq$ 10 seconds and 90% of the events met amplitude reduction criteria. Sleep time was estimated from patient's sleep-log and respiratory movement patterns. The total number of apnoeas and hypopnoeas was divided by the estimated sleep time giving the AHI. An oxygen-desaturation index (ODI) was calculated in the same manner based on desaturations of $\geq$ 4%. Patients were

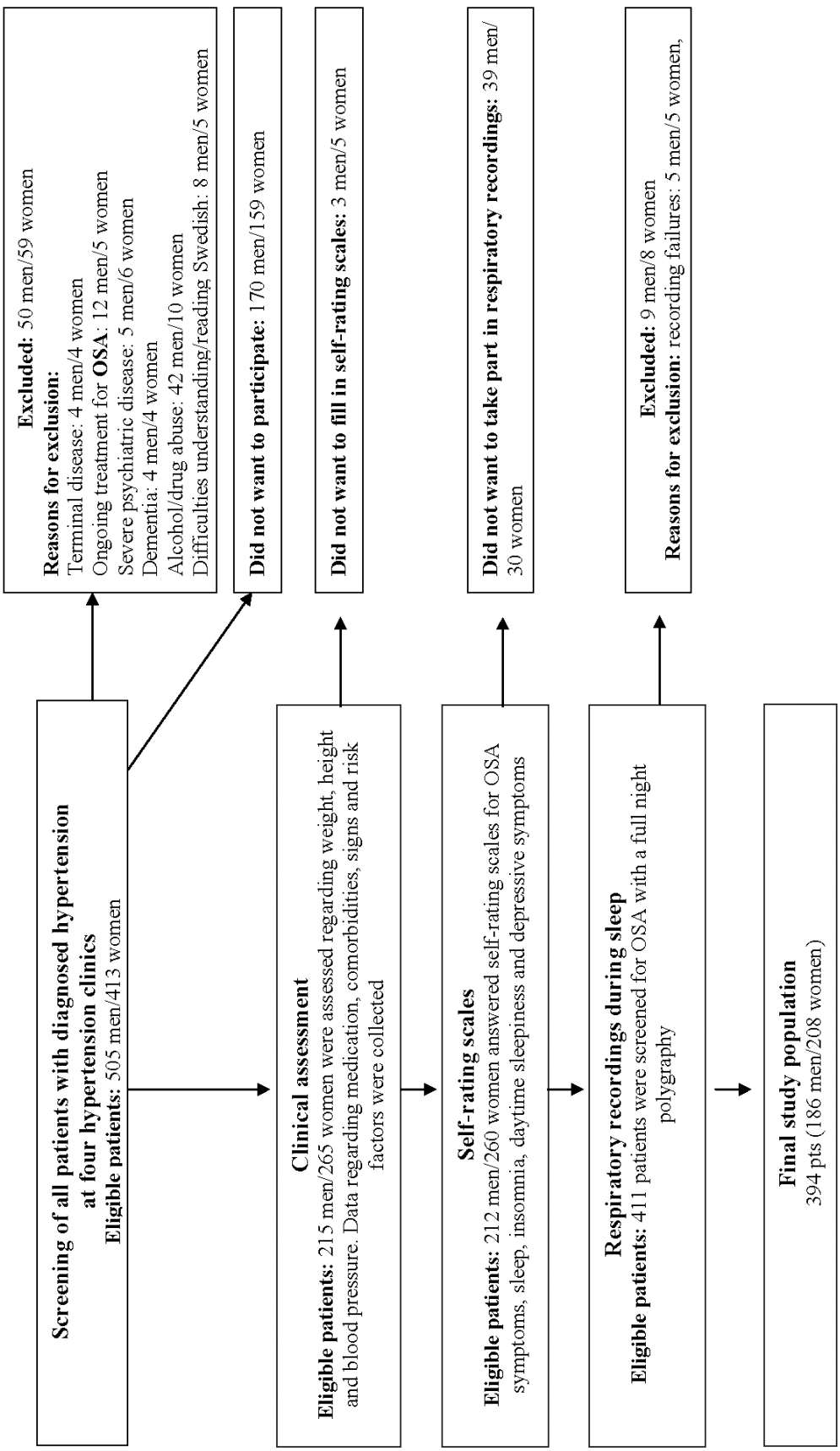

Figure 1 Description of design, number of eligible and excluded patients in the study

defined as having mild, moderate or severe OSA if they had AHI scores of 5–14.9, 15–29.9 or $\geq$ 30, respectively.

Statistical analysis

Descriptive statistics were presented in terms of means and standard deviations, or in numbers and percentages. Factor analytical techniques including structural equation modelling (SEM) were used for data reduction and modelling. The rationale for using factor analytical techniques and SEM was to reduce the complexity of data, and analyse complex relational schemes (e.g. look at relationships, direct and/or indirect effects between several variables) in a way that cannot be done with simple multivariate analysis.[20] First, an exploratory principal component analysis with oblique rotation was used to reduce the complexity of a large number of observed variables to create a simpler factor model. Variables easy to collect and measure in a primary care setting were entered in the explorative factor analysis: body mass index (BMI), neck circumference, waist circumference, diagnosis of diabetes, diagnosis of IHD, diagnosis of hypercholesterolaemia, systolic blood pressure, diastolic blood pressure, moderate physical activity, vigorous physical activity, disturbing snoring, dry mouth on awakening, morning headache, global perceived health, depressive symptoms, witnessed apnoeas, excessive daytime sleepiness, difficulties initiating sleep, difficulties maintaining sleep, early morning awakenings and non-restorative sleep. Criteria for a variable to be retained in a factor were that they had to achieve a factor loading of at least 0.3. To determine the number of factors, Eigenvalues > 1, Scree tree plots, as well as a theory-based selection (i.e. that the factors are meaningful and logical) were used.[21] In a second step the factors from the final exploratory factor analysis were incorporated into a measurement model using a confirmatory factor analysis ad modum LISREL.[22] This was done to examine and test the extent to which the data collected could be represented by the factor model. Finally, SEM analyses[20,23] were performed to test and compare a theoretically sound (as judged by the authors based on the existing literature) model of the structural relationships to a dependent continuous variable, in this study AHI (Figure 2). The reason for using AHI as a continuous numerical variable was to avoid effects of position dependent OSA,[24] as well as losing power in the analyses. Associations between the factors were derived using maximum likelihood and are described with their standardised coefficients. Standardised effects found between 0.10 and 0.30 were considered to be small, effects found between 0.30 and 0.50 were considered moderate, and effects > 0.50 were considered strong. 'Goodness of fit' tests were reported as the χ^2 value including degrees of freedom (df), root mean square error of approximation (RMSEA) and the comparative fit index (CFI). An overall RMSEA < 0.06 and a confidence interval range from 0.00 to 0.08 indicated a good fit. A CFI value $\geq$ 0.95 was considered a very good fit.[25] A level of $P < 0.05$ was regarded as significant.

Descriptive and exploratory factor analysis was performed with SPSS version 16.0. Confirmatory factor analysis and SEM analyses were performed with LISREL software.[22]

Results

Study population

Sixty-nine (39 men and 30 women) of the 480 patients who participated in the clinical examination declined respiratory recordings. No significant differences were seen regarding comorbidities and medications compared with those accepting to participate. Of the 411 performed recordings, 17 were lost due to technical problems. Thus, the final study population consisted of 394 patients (186 men/208 women). Fifty-nine percent of the patients had an AHI $\geq$ 5/hour indicating undiagnosed OSA. Mild (mean AHI 8.8, SD 2.8), moderate (mean AHI 21.8, SD 4.4) and severe OSA (mean AHI 49.3, SD 19.2) occurred among 29, 16 and 14% of the patients, respectively. Population characteristics, comorbidities and medications are given in Table 1.

Exploratory and confirmatory factor analysis

Initially a seven-factor model was extracted explaining 64.4% of the variance. Five factors consisting of anthropometrics, blood pressure, comorbidity, health complaints and physical activity were theoretically sound. The two other factors, however, both described OSA-related symptoms with morning headache and dry mouth in one factor and snoring and witnessed apnoeas in the other. Therefore, data were forced into a six-factor solution. Table 2 describes the final six-factor model with the four variables describing OSA-related symptoms in one factor. The model explained 58.3% of the variance. Four variables, diagnosis of diabetes, global perceived health, excessive daytime sleepiness and morning headache, showed difficulties with factor loadings > 0.3 in two factors. Morning headache loaded 0.49 in health complaints, but also 0.38 in OSA-related symptoms. The other three loaded strongest in the factor to which they logically belonged.

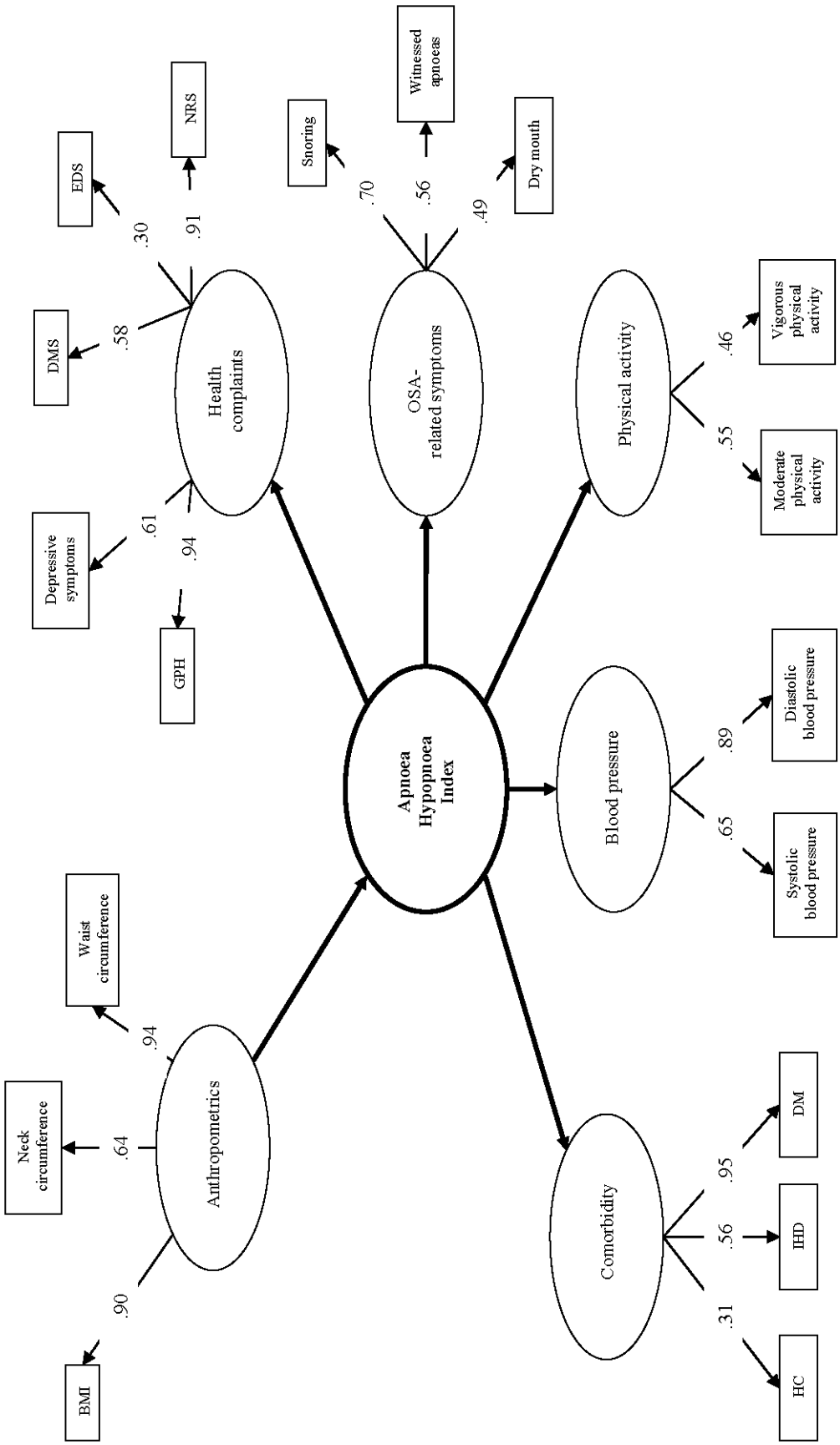

Figure 2 Theoretical pathways (bold arrows) between the six factors (circles) derived in the factor analysis and undiagnosed obstructive sleep apnoea (AHI) in hypertensive outpatients. The confirmatory factor analysis describing the factors including manifest variables (squares) and factor loadings are also shown

Table 1 Characteristics, medication and comorbidities for the whole sample (n = 394) as well as those with and without obstructive sleep apnoea

Characteristics	Total sample (n = 394)	No OSA (AHI < 4.9/hour) n = 161 (41%)	OSA (AHI ≥ 5/hour) n = 233 (59%)	P
Gender: Men, n (%)	61 (38)	60 (38)	108 (46)	0.05
Age: mean (SD)	57.8 (6.7)	57.3 (6.6)	58.1 (6.7)	NS
Blood pressure, mean (SD):				
Systolic blood pressure	140.5 (17.3)	138.8 (17.4)	141.6 (17.3)	NS
Diastolic blood pressure	87.2 (10.5)	86.5 (9.6)	87.7 (11.1)	NS
Anthropometrics, mean (SD):				
BMI	28.8 (4.9)	27.0 (4.2)	29.9 (5.0)	0.001
Neck circumference	39.2 (5.6)	37.8 (6.1)	40.0 (5.0)	0.001
Waist circumference	101.0 (13.2)	96.2 (11.5)	104.0 (13.3)	0.001
Medication, n (%):				
CA-blockers	80 (20)	27 (17)	53 (23)	NS
B-blockers	185 (47)	72 (46)	113 (48)	NS
ACEI/ARB	227 (58)	90 (57)	137 (59)	NS
Digoxin	1 (0)	0 (0)	1 (0)	NS
Diuretics	130 (33)	50 (32)	80 (34)	NS
Number of hypertensive drugs, mean (SD)	1.6 (0.9)	1.5 (0.8)	1.6 (0.9)	NS
Comorbidities, n (%):				
DM	67 (17)	20 (12)	47 (20)	NS
IHD	286 (73)	112 (70)	174 (74)	NS
HC	114 (29)	40 (25)	74 (32)	NS
TIA/stroke	8 (2)	3 (2)	5 (2)	NS
Self-rated sleep, mean (SD):				
Sleep, hours	6.8 (1.1)	6.7 (1.0)	6.8 (1.2)	NS
Sleep need, hours	7.8 (0.9)	7.6 (0.7)	7.8 (0.9)	NS
Insomnia symptoms, n (%):				
Difficulties initiating sleep	112 (28)	36 (11)	76 (33)	0.01
Difficulties maintaining sleep	199 (50)	73 (45)	126 (54)	NS
Difficulties with non-restorative sleep	188 (48)	68 (42)	120 (51)	NS
Daytime sleepiness, mean (SD):				
Total ESS score	7.9 (4.3)	7.8 (4.4)	8.1 (4.6)	NS
Symptoms of OSA:				
Witnessed apnoeas, n (%)	77 (19)	13 (8)	64 (27)	0.000
Snoring, mean (SD)	5.2 (3.3)	4.1 (3.4)	6.2 (2.9)	0.000
Dry mouth, mean (SD)	4.6 (3.2)	3.8 (3.1)	5.1 (3.2)	0.000
Morning headache, mean (SD)	2.6 (2.6)	2.6 (2.6)	2.5 (2.6)	NS
Sleep disordered breathing, mean (SD):				
AHI	14.0 (17.6)	2.1 (1.5)	21.9 (18.9)	0.000
ODI	12.8 (16.8)	2.1 (1.5)	20.1 (18.4)	0.000
No OSA, n (%)	161 (41)	161 (41)	–	–
Mild OSA, n (%)	113 (29)	–	113 (29)	–
Moderate OSA, n (%)	64 (16)	–	64 (16)	–
Severe OSA, n (%)	57 (14)	–	57 (14)	–

Table 2 The final six-factor solution of the exploratory factor analysis. Loadings given in bold describe those variables included in the specific factor. The total explained variance of the model is 58.3%

Variables	Anthropo-metrics	Blood pressure	OSA-related symptoms	Comorbidity	Health complaints	Physical activity
BMI	**0.861**	0.11	0.080	0.095	0.116	–0.010
Neck circumference	**0.791**	–0.008	0.106	–0.088	–0.034	–0.004
Waist circumference	**0.910**	0.056	0.081	0.171	0.031	–0.050
Systolic blood pressure	0.18	**0.880**	0.002	0.046	–0.003	0.071
Diastolic blood pressure	0.063	**0.857**	0.045	–0.103	–0.031	–0.123
Snoring	0.103	0.013	**0.831**	0.093	0.008	0.095
Witnessed apnoeas	0.027	0.054	**0.733**	0.065	0.133	–0.177
Dry mouth	0.178	–0.03	**0.592**	–0.110	0.251	0.026
Morning headache	–0.193	0.073	0.380	–0.142	**0.490**	0.145
Diagnosis of IHD	–0.053	0.11	0.046	**0.554**	–0.012	0.286
Diagnosis of DM	0.345	–0.14	0.074	**0.583**	–0.022	–0.187
Diagnosis of HC	0.076	–0.05	0.010	**0.736**	0.044	–0.078
Global perceived health	0.342	–0.043	0.143	0.090	**0.632**	–0.167
Depressive symptoms	0.223	–0.138	–0.036	–0.174	**0.555**	–0.160
Excessive daytime sleepiness	–0.207	–0.049	0.111	0.313	**0.406**	–0.071
Difficulties maintaining sleep	0.015	0.06	0.035	0.075	**0.706**	0.176
Difficulties with non-restorative sleep	–0.059	0.013	0.195	0.052	**0.823**	0.006
Moderate physical activity	0.007	0.067	–0.086	–0.004	–0.097	**0.736**
Vigorous physical activity	–0.058	–00.12	–0.105	–0.010	0.095	**0.710**
Eigenvalue	3.42	2.52	1.89	1.41	1.28	1.19
Variance %	17.99	13.38	9.96	7.42	6.76	6.25

The confirmatory factor analysis established the six factors of the exploratory factor analysis with an acceptable goodness of fit (χ^2 = 153, df 122, P = 0.03; RMSEA = 0.029 [0.011–0.042]; CFI = 0.98). However, after scrutinising the results, minor changes in the included variables were deemed necessary. Morning headache still loaded in two factors and showed high standard residual values and was therefore excluded, which improved the fit (χ^2 = 147.5, df 124, P = 0.074, RMSEA = 0.026). Figure 2 describes the final confirmatory factor analysis and the theoretical paths from the factors to undiagnosed OSA.

The structural model and associations to undiagnosed OSA

The theoretical model was not fully confirmed in the SEM analyses. No associations were found between undiagnosed OSA and comorbidity, blood pressure or physical activity (Figure 3). Moreover the analysis revealed a negative association (–0.31) between health complaints and undiagnosed OSA, implying that fewer health complaints were associated with undiagnosed OSA. A factor analysis of the factor 'health complaints' revealed that the dyssomnia variables (excessive daytime sleepiness, difficulties maintaining sleep, non-restorative sleep) and the variables describing poor health (global perceived health and depressive symptoms) represented two separate clusters. The factor, 'health complaints', was therefore separated into two factors named dyssomnia and poor health. After several options had been tested, the final SEM model (Figure 3) showed moderate significant associations between anthropometrics (0.45), OSA-related symptoms (0.47) and undiagnosed OSA. OSA had no direct effect on dyssomnia or poor health. Indirect significance effect of 0.16, 0.15 and –0.19 were seen on dyssomnia, poor health and decreased physical activity, respectively, mediated by OSA-related symptoms.

Discussion

This study performed on primary care patients with hypertension confirms the associations between undiagnosed OSA, OSA-related symptoms (snoring, witnessed apnoeas, dry mouth) and anthropometrics (BMI, neck circumference, waist circumference). In contrast, there was no direct association between undiagnosed OSA, comorbidities, blood pressure, dyssomnia, physical activity or self-rated poor health.

Treatment of OSA, especially in patients with severe symptomatic OSA, may be of importance before irreversible vascular damage has taken place.[6] Treatment can also decrease morbidity and mortality.[8,10,11] Despite this, epidemiological studies suggest that the disease is underdiagnosed.[1,3] We found that 59% of the study population had an AHI $\geq$ 5/h. A previous Swedish study found in a stratified sample of hypertensive men that 37% had AHI $\geq$ 10.[26] Furthermore, another study found a prevalence of 83% (AHI $\geq$ 10) in patients with both hypertension and diabetes.[27] One cause for OSA being underdiagnosed might be due to non-specific symptoms, and the fact that patients themselves are unable to describe apnoeas, as they occur during sleep. Another cause might be that the assessments of patients with sleeping problems in clinical practice tend to focus more narrowly on physiological parameters concerning sleep, breathing and circulation. Because a definite diagnosis requires an objective respiratory recording (performed with expensive and technically advanced equipment), and the disorder is fairly common, there is a need in daily clinical practice for methods that may facilitate better detection of patients where such a recording is warranted. Earlier studies have focused on individual risk factors for OSA, such as anthropometric measures, hypertension, daytime symptoms, nighttime symptoms, depressive symptoms and the metabolic syndrome.[14,28] However, most of these studies have also been performed on sleep clinic populations (where patients have already been identified as likely suffering from OSAS) which might have affected the predictive value of the different variables. A multimodal approach, taking into account both biometric and psychometric aspects easy to assess and collect in a primary care setting may be needed to identify the patients.

Our model demonstrated that anthropometrics (BMI, neck circumference and waist circumference) as well as OSA-related symptoms (snoring, witnessed apnoeas and dry mouth) may be helpful in identifying undiagnosed OSA among hypertensive primary care patients. Furthermore, biometric and psychometric aspects were equally important in the present model. Anthropometrics and OSA-related symptoms showed beta values of 0.45 and 0.47 respectively. Anthropometrics are known associates of OSA and $\sim$ 70% of those with OSA are obese and higher BMI values tend to be associated with a more severe OSA.[4] Mean BMI, neck and waist circumference for patients with OSA (AHI $\geq$ 5) was 29.9 kg/m^2 (SD 5.0 kg/m^2), 40.0 cm (SD 5.0 cm) and 104.0 cm (SD 13.3 cm), respectively, significantly higher than in those without OSA (P < 0.001). From a mechanistic perspective, obesity causes increased fatty deposits that contribute to narrowing of the upper airway, and also leads to an altered shape.[29] Anatomical factors (e.g. maxillo-mandibular retrognathia, enlarged tonsils) can also compromise the size of the upper airway and increase the risk for OSA and other comorbidities over time, but are difficult to assess for a nurse or GP without specific

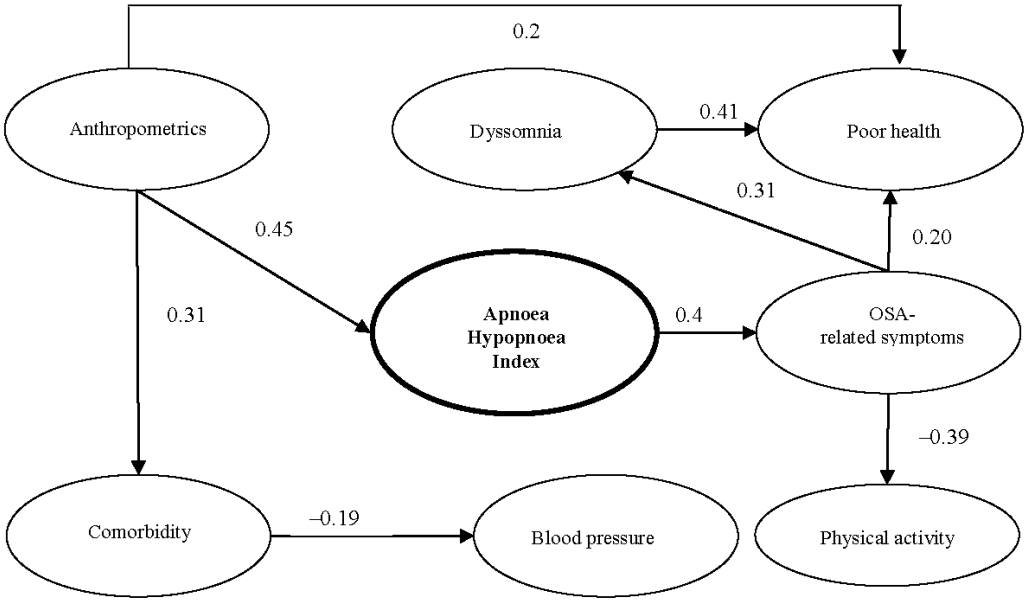

Figure 3 SEM of characteristics associated with undiagnosed obstructive sleep apnoea (i.e. AHI). Only significant effects are described with arrows. Anthropometrics and OSA-related symptoms were the factors directly associated with undiagnosed obstructive sleep apnoea. The goodness of fit values for the model are: Chi-square = 151.2, df 124 ($P = 0.048$); RMSEA = 0.026 (0.0022–0.004); CFI = 0.98

competence. Furthermore, central and upper body fat correlates with occurrence of OSA. Young *et al*[30] found that every increase of 13–15 cm in waist or hip circumference increased the risk for having OSA by a factor of 4. The use of easy assessable anthropometric measures other than BMI (i.e. neck and waist circumference) may therefore be of importance when identifying patients with OSA. The BSAQ[15] (a validated tool used to categorise patients as 'low' or 'high' risk for OSA) may, together with information from partners and simple inexpensive two-channel recording devices,[14] be suitable for use at an early follow-up appointment (after diagnosis of hypertension has been established) to detect undiagnosed OSA.

Comorbidities (IHD, diabetes, hypercholesterolemia), blood pressure, dyssomnia (excessive daytime sleepiness, difficulties maintaining sleep, non-restorative sleep) and poor health (perceived health and depressive symptoms) were not directly associated with AHI in the present model. In contrast to our findings, Vgontzas showed that patients with OSA had higher fasting blood glucose, insulin resistance and glycated haemoglobin (HbA1c) than weight-matched controls,[31] and that the severity correlated with severity of AHI. Others have supported the metabolic syndrome as one of the best predictors for OSA.[28] Depression and poor self-rated health are often associated with OSA.[32] The use of a non-sleep clinic population with less severe OSA (41% having no OSA and 29% having AHI 5–15 indicating mild OSA), a low level of daytime symptoms (excessive daytime sleepiness) and already treated and controlled hypertension in the present model might explain these findings. Furthermore, we used SEM analyses that focus on associations of the derived factors with AHI as a continuous numerical variable, not on OSA as indicated by, for example, AHI ≥ 5 (mild OSA) or AHI ≥ 15 (moderate OSA). Identifying the relevant patient is, however, of critical importance because CPAP treatment can decrease morbidity and mortality, thus reducing consumption of healthcare resources.

Limitations

Despite its relatively large sample size, this study has some limitations. A cross-sectional design was used, which limits conclusions of cause and effect in the proposed theoretical model. We performed full-night respiratory recordings with polygraphic equipment in patients' homes. Guidelines describe polysomnography[14] as a preferred method, but polygraphy can be used as a comprehensive sleep evaluation to decrease costs and minimise inconvenience for patients.[33] Well validated self-rating scales[15–19] were used to collect self-rated variables in the theoretical model. A limitation, how-

ever, was that the model was based on well known characteristics from the existing AASM guidelines[14] and did not explore new, unknown characteristics in hypertensive primary care patients. Furthermore, aspects, such as cognitive function (e.g. memory loss, decreased concentration), decreased libido and irritability were not measured and included in the model. Another limitation was the lack of clear cut-offs for the dependent variable (AHI), anthropometrics, or OSA-related symptoms that we used in the SEM analyses. If data regarding 24-hour blood pressure were collected we might have found different results (i.e. significant differences) for the association between blood pressure and AHI. Furthermore, the results might have been different if only patients with moderate or severe undiagnosed OSA suitable for treatment with CPAP had been included in the SEM model. Further studies are needed to evaluate sensitivity and specificity of this model to identify patients with undiagnosed OSA of different severity levels. Such studies should use a gender perspective to identify men and women that would show high cost benefit for referral to sleep clinics.

Conclusion

Undiagnosed OSA was directly associated with OSA-related symptoms (snoring, witnessed apnoeas, dry mouth) and anthropometrics (BMI, neck circumference, waist circumference). These characteristics could be used by GPs to identify patients who are in need of referral to a sleep clinic for OSA evaluation. No direct associations were found for comorbidities, blood pressure, insomnia or poor health.

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PEER REVIEW

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CONFLICTS OF INTEREST

None.

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