criteria for raised blood pressure. The table also shows the average systolic and diastolic blood pressures at which each doctor decided that a patient had hypertension. These ranged from 173 to 206 mm Hg systolic and 103 to 116 mm Hg diastolic. In addition, not all patients labelled hypertensive were treated (see table), although most of those not treated had had only one reading that suggested high pressure.

Discussion

The widespread variation in the labelling of hypertension by the individual general practitioners at the start of the screening programme reflected differing attitudes towards the management of hypertension, and this was largely maintained throughout the screening period.

Although the screening programme seemed to help some doctors in detecting patients with raised blood pressures, most of the doctors who had labelled more of their patients as having raised blood pressure at the start of screening identified more as a result of screening than those who started with fewer. On the other hand, those who identified fewer patients over the five years of screening were more likely to fulfil the agreed criteria. But the low overall proportion of patients detected during the screening period who actually fulfilled the criteria for hypertension (24/187, 12.8%) suggests that the general practitioners were unhappy

about not treating patients with more borderline blood pressures and perhaps took other factors into consideration. Interestingly, this figure of 12.8% of patients fulfilling the criteria is very close to the 12.5% found by Patterson in his study of the diagnosis of hypertension in general practice, in which the same criteria were used.⁴

In conclusion, the results suggest that the screening helped some of the general practitioners to detect patients with significantly raised blood pressure but individual general practitioners seemed to adhere to their own criteria for labelling hypertension rather than to those formally agreed.

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Relation between systemic hypertension and sleep hypoxaemia or snoring: analysis in 748 men drawn from general practice

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Abstract

Objective—To establish whether a history of snoring or the degree of overnight hypoxaemia is an important independent predictor of systemic blood pressure.

Design—Prospective community based study of blood pressure in relation to overnight oxygen saturation, height, weight, and a questionnaire assessment of snoring, smoking, and alcohol consumption. Analysis was by multiple linear regression techniques and analysis of variance.

Setting—Small town outside Oxford, served by one group general practice of four partners. All measurements were made at home.

Subjects—The names of 836 men aged 35-65 were drawn at random from the general practitioners' age and sex register and the men then asked to participate; 752 (90%) agreed.

Main outcome measures—Systolic, mean, and diastolic blood pressures and their association with age, obesity, alcohol consumption, cigarette consumption, snoring, and overnight hypoxaemia.

Results—Though systemic blood pressure correlated significantly with overnight hypoxaemia, this was due to the cross correlation with age, obesity, and alcohol consumption. No independent predictive effect of overnight hypoxaemia was found. Snoring was correlated with systemic blood pressure but not significantly so and also was not an independent predictor once age, obesity, and alcohol consumption had been allowed for.

Conclusions—It is unlikely that snoring and sleep hypoxaemia from occult sleep apnoea are important causes of diurnal systemic hypertension when compared with age, obesity, and alcohol consumption.

tion. The increased prevalence of cardiovascular complications reported in snorers may be due to the confounding variable of obesity or to nocturnal rises in blood pressure that are not reflected in the daytime figures.

Introduction

In most patients with arterial hypertension there is no apparent cause for the condition,¹ except that age and body weight are known to be significant partial predictors of actual blood pressure.^{2,3} Much interest from various sources has been expressed in the possibility that occult sleep apnoea might be the cause of a substantial proportion (25-30%) of this "essential hypertension."⁴⁻¹² The evidence, however, is conflicting, and two recent studies failed to find an increased prevalence of sleep hypoxaemia in an untreated group of newly diagnosed hypertensive men compared with a matched control group.^{13,14} We have looked at 752 men aged 35-65 drawn from one group general practice and compared the power of overnight hypoxaemic episodes and snoring as partial predictors of arterial blood pressure with that of other known predictive factors such as age and obesity.

Subjects and methods

The town of Wheatley (8 km east of Oxford) is served mainly by one group general practice (four partners). The age and sex register of the practice was used to identify men whose birth dates put them between 35 and 65 years of age at the time of the study. They were then selected for study by year of birth with the year order randomised. Each subject received a

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TABLE I—Overall results of continuous variables from 748 of 752 subjects in whole study population*

Continuous variables	Mean	SD	Median	5-95% Range	0-100% Range
Age (years)	47.6	8.1	48	36-62	35-65
Height (cm)	176	67	177	165-187	148-198
Weight (kg)	78.9	11.2	78.0	63.0-99.5	53-130
Body mass index (weight (kg)/(height (m)) ²)	23.3	3.2	25.1	20.9-30.8	15.3-38.8
Systolic blood pressure (mm Hg)	135.8	18.4	134	109-170	94-221
Mean blood pressure (mm Hg)†	103.9	12.2	103	86-126	75-160
Diastolic blood pressure (mm Hg)	88.1	11.2	88	70-108	58-130
>4% SaO ₂ dips/h‡	1.64	1.63	1.09	0.25-4.8	0-12.2
Median overnight SaO ₂ (%)	94.3	1.5	95	92-96	87-98

*Details incomplete in four cases.

†Two thirds of diastolic pressure plus one third of systolic.

‡See text.

letter of explanation from his own doctor, followed by a telephone call from one of us (JC). Eight hundred and thirty six subjects were invited to take part, of whom 752 (90%) agreed. Table I gives the details of these subjects.

TECHNIQUES

Blood pressure was measured in the patient's own home by a nurse (JC). Measurements were taken in either arm after at least 15 minutes' sitting quietly in a chair and were always done at least in duplicate. A third reading was taken if the first two readings differed by more than 15 mm Hg in either systolic or diastolic values. The final figure used was the mean of the readings. In addition to systolic and diastolic values, mean blood pressure was calculated as two thirds of diastolic pressure plus one third of systolic. All measurements were with an electronic sphygmomanometer (Omron Tatsei Electronics, Japan), which was calibrated weekly throughout the study against a standard mercury sphygmomanometer. The device incorporates a microphone which detects the Korotkoff sounds simultaneously with an electronic measurement of cuff pressure, the rate of fall of which was roughly 3 mm Hg/s. The area of the bladder was 23 × 13 cm, which is appropriate for arm circumferences of 24-34 cm. No subject's arm was too big or too small for the cuff used, but in four subjects measurements could not be made owing to a break in the microphone wire, leaving 748 measurements for analysis.

Each subject completed a questionnaire asking about alcohol consumption (categories 1-4: <10 g/day, 10-39 g/day, 40-70 g/day, >70 g/day; cigarette consumption (categories 1-4: nil, 1-5/day, 6-15/day, >15/day); snoring (categories 1-4: never, rarely, sometimes, often); and whether currently taking any medicines (category 1 or 2: yes, no). Table II gives the details. Height was measured with a steel rule and weight by portable scales (SECA, West Germany), which were checked monthly. The same instruments were used throughout. The age of the subject at the time of study and whether the subject's wife was present at the interview were also recorded.

Overnight oxygenation (eight hours) was measured in the subject's home using an Ohmeda Biox 3700 pulse oximeter (version J) with a flexible finger probe.¹⁵ The machine's internal memory stores a two digit value at the end of every 12 seconds which represents the lowest percentage arterial oxygen saturation (SaO₂) detected during the 12 seconds. Thus dips in SaO₂ are not missed but recovery between dips is underestimated. These data from the eight hour memory were offloaded by means of an RS232 interface to a BBC B computer. The 2400 points were then analysed for median SaO₂ and dips in excess of 4%. Dips are counted when the SaO₂ falls more than 4% below the previous updated high. No further dip can be scored unless the SaO₂ subsequently rises by more than 3%. When this happens a constantly updated maximum SaO₂ is stored until another value more than 4% lower than this occurs, which is again scored as a dip. Thus

no assumptions are made about baselines but any new fall greater than 4% is scored as a dip. The number of these dips is then divided by the length of the tracing and reported as >4% SaO₂ dips/h. This value correlates closely with the apnoea index.¹⁶⁻¹⁹

PROTOCOL

After the preliminary letter and phone call the subjects were visited in the evening at home by JC. The study was explained and the questionnaire completed. If the subject's wife was present they were allowed to discuss the answer before the reply was noted. Blood pressure was then measured followed by height and weight. The subjects were shown how to use the oximeter, and the flexible probe was left ready to slip on to the finger, using the disposable sticky tapes supplied by Ohmeda. The subjects applied the probe and switched the machine on when retiring to bed and switched it off on waking next morning ready for collection and return to the hospital. All 752 patients were interviewed by the same person over three and a half years.

STATISTICS

All data were entered into the Oxford University Vax computer and analysed by the SAS statistical package.²⁰ The interrelation among the continuous variables was first examined with Pearson's correlation coefficients and the relation between snoring and blood pressure (systolic, mean, and diastolic) examined by using simple analysis of variance alone. The data were then examined together by multiple linear regression techniques with either systolic, mean, or diastolic blood pressure as the dependent variable. The stepwise procedure (with $p=0.15$ as the significance level for entry or retention) was used initially, but exactly the same results were obtained when using either the backward or forward method to enter variables. To deal correctly with both continuous variables (table I) and discontinuous variables (table II)—for example, alcohol consumption and snoring—the general linear models procedure was also used with the classes option.^{20, 21} All these analyses gave the same values of r^2 for each variable, and we report the p values from the

TABLE II—Overall results of discontinuous variables from whole study population

Discontinuous variables	No (%) of subjects
Alcohol consumption (g/day):	
<10	375 (49.9)
10-39	268 (35.7)
40-70	96 (12.8)
>70	12 (1.6)
All subjects	751 (100.0)
Cigarette consumption (No/day):	
0	522 (69.4)
1-5	71 (9.4)
6-15	71 (9.4)
>15	88 (11.7)
All subjects	752 (100.0)
Snoring, wife not present at interview:	
Never	68 (19.0)
Rarely	64 (17.9)
Sometimes	187 (52.2)
Often	39 (10.9)
All subjects	358 (100.0)
Snoring, wife present at interview:	
Never	56 (14.5)
Rarely	85 (22.1)
Sometimes	152 (39.5)
Often	92 (23.9)
All subjects	385 (100.0)
Currently taking medicines?	
Yes	190 (25.3)
No	562 (74.7)
All subjects	752 (100.0)

general linear models procedure. All these statistical tests were carried out on the whole study population, the subset of 562 subjects currently not taking medicines of any type, and the subset of 385 subjects whose wives were present at the interview.

Results

Single Pearson correlation coefficient analysis between blood pressure and $>4\%$ SaO₂ dip rate, age, body mass index (weight (kg)/(height (m))²), and median overnight SaO₂ showed that all the variables were significantly correlated with each other. For example, the correlation of $>4\%$ SaO₂ dip rate with mean blood pressure was $r=0.17$ (95% confidence interval 0.1 to 0.24), $p<0.0005$. This apparent correlation, however, was probably due to the primary correlation of $>4\%$ SaO₂ dip rate with age or obesity, or both.²² After allowance for age and obesity the $>4\%$ SaO₂ dip rate accounted for only 0.12% of the mean blood pressure variation ($p>0.3$) as compared with 12.5% from obesity, age, and alcohol consumption (table III). Systolic blood pressure correlated best with the age of the subject, an average rise of 6.7 mm Hg being recorded with each decade. Diastolic blood pressure, on the other hand, correlated best with obesity, an average rise of 0.9 mm Hg being recorded with each digit increase in the body mass index. Alcohol consumption had a small effect on blood pressure. Very approximately the mean blood pressure showed an average rise of 1 mm Hg for every 12 g of alcohol consumed a day. Neither the multiple general linear models procedure nor the simple analysis of variance (systolic, mean, and diastolic blood pressures: $p=0.3$, 0.26, and 0.40 respectively) assigned any significance to the history of snoring as a predictor of blood pressure. As expected, the $>4\%$ SaO₂ dip rate was significantly related to snoring category (analysis of variance, $p<0.0001$).

We also analysed separately the subgroup of men whose wives were present at the interview ($n=385$), which may have provided a more accurate snoring history. When the wife was present significantly more men were recorded as snorers ($p<0.0001$) (table II). Using this subset improved the predictive value of snoring (analysis of variance) to nearly significant levels (p values for systolic, mean, and diastolic blood pressures 0.08, 0.06, and 0.07 respectively). Nevertheless the multiple general linear models procedure entering all the variables failed to assign any significance to the history of snoring. The actual mean blood pressure values in the four snoring categories (never, rarely, sometimes, often) were 102.6, 100.0, 104.7, and 103.0 mm Hg, respectively, as compared with the

values predicted from the subjects' ages, body mass indices, and alcohol consumption, which were 101.9, 102.6, 104.2, and 105.8 mm Hg. Thus the slightly higher mean blood pressure values in the "sometimes" and "often" snoring groups were entirely accountable by their greater age, obesity, or alcohol consumption.

Analysis of the subset of men not taking drugs of any kind ($n=562$; table III) did not materially alter any of the relations and produced only minor changes in the actual values of r^2 .

Discussion

We have shown in a randomly selected group of 748 men aged 35-65 that neither snoring nor nocturnal $>4\%$ SaO₂ dip rate contributed to explaining the variance in systolic, mean, or diastolic blood pressure. These findings were obtained in a data set in which age and obesity were highly significant predictors with combined r^2 values of 13.6% for systolic pressure and 8.9% for diastolic pressure. The r^2 values and the slopes of the relations with age and obesity were similar to data published by others.²³

The accuracy of our blood pressure measurements is unlikely to have been a reason for the failure to detect a relation on several accounts. The electronic blood pressure measuring device was used because of its portability, because it has no terminal digit preference, and to guard against the possibility of a change in personnel during the study. As the electronic device was used throughout (and its pressure calibration checked weekly), any bias in measuring systolic or diastolic pressure should have been constant. The diastolic pressure measured by the device approximates most closely to phase IV. The 3 mm Hg/s fall in cuff pressure is slow enough not to underestimate the diastolic pressure. Though the median diastolic pressure was slightly higher than expected (88 mm Hg; table I), it was only 2 mm Hg higher than predicted from the data of Miall and Lovell,² presumably owing to the different type of sphygmomanometer used or the slightly high median body mass index (25.1) in our population. Finally, if the quality of the readings had been poor then we would have got poorer relations with age and obesity than reported by others, which was not the case.²³ Physiologically a subject having his blood pressure measured by a nurse after 15 minutes' sitting quietly and talking to her is probably ideal and avoids the "white coat effect" and the transient effects of exercise.²³

We found that the self reported snoring history differed when the subject's wife was present at interview. Her presence increased the prevalence of often snoring from 10.9% to 23.9% (table II). We (and others) have also noticed that some subjects with irrefutable histories of heavy snoring inexplicably do not snore very much when studied in a sleep laboratory. Thus the report of snoring by the spouse is probably the best way to quantify the amount of snoring other than with domiciliary snoring monitors. Had we predicted the importance of the wives' presence at interview we would have made a specific point of asking them the relevant questions.

The Ohmeda 3700 oximeter accurately monitors SaO₂, and falls of more than 4% closely reflect apnoeas of more than 10 seconds.^{16,19} We shall report separately the prevalence and cause of dips in SaO₂ in these men, which is mainly supine obstructive sleep apnoea for short and variable periods during the night.²² The reproducibility of overnight SaO₂ tracings done at home has been reported and is adequate for epidemiological studies.²⁴

Though daytime hypertension has invariably been referred to in studies of obstructive sleep apnoea, it is far from clear that obstructive sleep apnoea is the main

TABLE III—Multiple linear regression results for blood pressure (743 subjects)

Variable	r^2 (95% confidence interval)	Slope (95% confidence interval)	p Value
<i>Systolic blood pressure</i>			
Age	8.8% (5.3 to 13.0)	0.67 (0.51 to 0.83)	<0.0001
Body mass index	3.8% (1.4 to 6.8)	1.11 (0.72 to 1.50)	<0.0001
Alcohol consumption	1.0% (0.1 to 2.9)	2.23 (0.57 to 3.91)	0.038
Total	13.6% (9.3 to 18.4)		
<i>Mean blood pressure</i>			
Body mass index	7.6% (4.3 to 11.6)	0.95 (0.69 to 1.21)	<0.0001
Age	3.7% (1.1 to 5.9)	0.33 (0.23 to 0.44)	<0.0001
Alcohol consumption	1.2% (0.1 to 3.0)	1.72 (0.61 to 2.83)	0.018
Total	12.5% (8.4 to 17.2)		
<i>Diastolic blood pressure</i>			
Body mass index	6.9% (3.8 to 10.8)	0.87 (0.26 to 0.63)	<0.0001
Age	1.0% (0.1 to 2.9)	0.16 (0.06 to 0.26)	0.007
Alcohol consumption	1.0% (0.1 to 2.9)	1.47 (0.43 to 2.51)	0.040
Total	8.9% (5.2 to 13.0)		

Variables entered: $>4\%$ SaO₂ dip rate, age, body mass index, median overnight SaO₂, alcohol consumption, cigarette consumption, snoring history. No variable other than those in table reached $p<0.15$ in any of the modelling.

cause. A report looking at hypertension in 372 snorers (194 with apnoea-hypopnoea indices >10/h) found that in a stepwise multiple linear regression analysis 8% of the variance in diastolic blood pressure was accounted for by body mass index, 4% by age, and only 1.7% by the apnoea-hypopnoea index.²⁵ In the non-apnoeic snorers blood pressure was correlated significantly only with body mass index. When sleep apnoea is treated effectively the blood pressure does not fall by very much.²⁶

Reports linking disorders of breathing during sleep and hypertension or cardiovascular complications have essentially fallen into two groups. The first consists of several papers describing a high (about 20-30%) prevalence of mild to moderate sleep apnoea in patients with previously diagnosed essential hypertension.⁴⁻⁷ These studies either examined hypertensive subjects receiving treatment or did not include a control group appropriately matched for weight. A fuller criticism of these studies appeared with a report from this unit which failed to find a higher prevalence of sleep hypoxaemia in newly diagnosed, untreated hypertensive subjects than in identically matched control subjects.¹³ Though that paper looked only at sleep hypoxaemia, a later report investigating apnoeic episodes as well also failed to detect a higher prevalence in moderately obese hypertensive subjects than in controls. More overweight hypertensives had higher degrees of apnoea but they were not compared with a control group.¹⁴

The other group of studies linking disorders of breathing during sleep and cardiovascular abnormalities were epidemiological surveys.⁸⁻¹² They suggested either that any significant relation between snoring and cardiovascular consequences was due to cases of occult sleep apnoea among the snorers or that snoring itself could directly provoke these complications. It is difficult to set up a hypothesis for this second link. Nevertheless, snoring may be attended by powerful inspiratory effort against a semiclosed pharynx, with large swings in pleural pressure, thereby transiently affecting both preload and afterload to the heart. There is no doubt that arterial blood pressure oscillates in heavy snorers, and more so in obstructive sleep apnoea,²⁷ but how this could lead to sustained hypertension is unclear.

These epidemiological surveys linking snoring and hypertension or cardiovascular complications (angina and stroke) have invariably had to deal with the confounding variable of obesity. This has usually been partialled out by multiple regression techniques using body mass index (weight (kg)/(height (m))²) or percentage of ideal body weight. Several studies, however, have shown that hypertension, ischaemic heart disease, and stroke are better correlated with upper body obesity—for example, waist to hip ratio or abdominal girth—than with general obesity (as measured by weight (kg)/(height (m))²).²⁸⁻³⁰ We and others find that obstructive sleep apnoea and snoring correlate better with neck obesity (neck circumference) than with general obesity,²²⁻³¹ and thus in these epidemiological surveys snoring may be acting as a marker of upper body obesity rather than being an independent risk factor itself.¹³ There is also evidence that smoking and alcohol consumption are independent risk factors for snoring, as they are for cardiovascular disease.³² This further confuses the issue whether snoring is an independent predictor or merely a marker of other factors.

We concur with a recent review of epidemiological reports that a causal link between snoring and cardio-

vascular disease is unproved and that any association is probably a consequence of confounding variables such as obesity (particularly of the upper body), smoking, and alcohol consumption.³³ If a truly independent link between snoring and the sequelae of hypertension can be shown in further studies, then harmful cardiovascular changes actually during sleep would be the more likely explanation rather than operating through sustained, diurnal systemic hypertension.

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