Prevalence of sleep disordered breathing in paroxysmal and persistent atrial fibrillation patients with normal left ventricular function

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Aims
Recent studies have suggested an emerging link between sleep apnoea and atrial fibrillation (AF). These studies included patients with reduced left ventricular (LV) function which may cause both AF and sleep disordered breathing (SDB). We examined the prevalence of SDB in a population of patients with AF and normal LV function.

Methods and results
Ninety patients with paroxysmal or persistent AF and 45 controls were prospectively enrolled and matched 2:1 for age (AF 56 ± 12 years; controls 54 ± 11 years) and sex. All patients had normal LV function. SDB was diagnosed using all-night portable polysomnography. Apnoea–hypopnoea index (AHI) in AF patients was higher than in controls (23.19 ± 19.26 vs. 14.66 ± 12.43, P = 0.01). The proportion with significant SDB (AHI > 15) was also greater in AF patients (62 vs. 38%, P = 0.01). After adjustment for relevant covariates, the odds ratio for the association between AF and SDB (AHI > 15) was 3.04 (95% CI 1.24–7.46, P = 0.02). The paroxysmal AF group was classified as either ‘low-frequency AF’ (< 6) or ‘high-frequency AF’ (> 6) episodes in the past year. High-frequency AF was associated with a higher prevalence (75 vs. 43%, P = 0.012) and severity (mean AHI 28.08 ± 22.94 vs. 16.69 ± 15.06, P = 0.028) of SDB when compared with those with low-frequency AF.

Conclusion
A high prevalence of SDB is found in relatively young patients with both paroxysmal and persistent AF with normal LV function. This AF population warrants careful consideration for the presence of SDB.

Keywords
Atrial fibrillation • Sleep apnoea • Normal left ventricular function

Introduction
Atrial fibrillation (AF) is the most frequently encountered arrhythmia in clinical practice, with a prevalence increasing with advancing age to > 9% in those over 80 years. In fact there has been a marked rise in the prevalence of AF over the past three decades. This trend is of particular concern given the known morbidity, mortality, and economic burden associated with the condition. Despite the high prevalence of this arrhythmia, in many patients, the cause of AF is unknown.

In recent years, there has been an increasing awareness of the prevalence and clinical significance of sleep disordered breathing (SDB) in a range of cardiovascular conditions. These conditions, which include congestive cardiac failure, coronary artery disease, and other forms of structural heart disease, may also play an important pathophysiological role in AF. Against the background of these common associations, there has been an increasing awareness of the potential relationship between AF and SDB.2 Studies demonstrating the increased prevalence of sleep apnoea in heart failure also observed an increase in SDB in the small subgroup with AF.3–6 Similarly, patients with sleep apnoea undergoing coronary artery bypass surgery were found to have a higher incidence of post-operative AF.7 Recently, two studies have evaluated the prevalence of sleep apnoea in patients with a primary diagnosis of AF with divergent results. Porthan et al.8 found a similarly high prevalence of sleep apnoea in lone AF patients as in a control group of controls. However, Bhat et al.9 found a lower prevalence of sleep apnoea in lone AF patients compared with controls.
group and concluded that sleep apnoea was not more common in AF than in control patients. In contrast, Gami et al.,9 using a questionnaire to detect sleep apnoea, found a significantly higher prevalence of sleep apnoea in AF patients, many of whom also had structural heart disease, than in age-matched cardiology controls. These discordant results, which might in part be explained by differences in the patient populations studied and in methodology, create uncertainty as to the precise nature of the relationship between SDB and AF.

In the present study, we used overnight sleep studies to prospectively investigate the prevalence of SDB in patients with AF and compared this with an age- and sex-matched control cardiology population. In particular, we included only patients without structural heart disease or left ventricular (LV) dysfunction in order to focus on the potential independent relationship between SDB and AF.

**Methods**

**Study population**

Subjects: Patients with paroxysmal or persistent AF and no structural heart disease who consented for the assessment of SDB. Patients were prospectively enrolled over an 18 month period.

Controls: Patients referred to the same tertiary referral arrhythmia centre during the same time period but without AF who consented for the assessment of SDB.

All study patients had a normal LV ejection fraction of >50% with no evidence of wall motion abnormality on transthoracic echocardiography.

Patients were excluded if they had: a prior diagnosis of SDB, age <18 years or >80 years, impaired LV function, active myocardial ischemia, ECG or echocardiographic evidence of prior myocardial infarction or valvular heart disease. Patients with prior AF ablation or AF due to a transient or reversible cause (post-operatively following cardiac or non-cardiac surgery, lung disease, or hyperthyroidism) were also excluded.

**Recruitment procedure**

i. A consecutive list of eligible patients (as described earlier) with AF was generated.

ii. Patients were approached and recruited from this list for consent to participate as cases (90 of 135 consented).

iii. Cases were paired on the basis of sex and age within 5 years.

iv. Controls were selected from a list of patients satisfying criteria matching paired cases (sex, age <5 years, inclusion criteria)

v. Selected controls were approached for consent; if not willing, the next patient satisfying criteria was approached (45 of 67 patients consented)

The study design was therefore a matched case–control study with a 2:1 casecontrol ratio and 45 matched sets. For each set of three patients (two AF and one control), age was matched to within a range of 5 years.

The study protocol was approved by the Melbourne Health Clinical Research and Ethics Committee at the Royal Melbourne Hospital and all participants gave written informed consent.

AF was defined as either paroxysmal or persistent on the basis of current American College of Cardiology, American Heart Association, and European Heart Society guidelines.15

**Diagnosis of sleep disordered breathing**

The presence of SDB was determined by means of at-home polysomnography using a portable Holter monitor-like device (Somteé data acquisition system, Compumedics, Melbourne, Australia).

This ambulatory sleep study continuously monitors an electroencephalogram (C3/A2), thoracic and abdominal effort (respiratory inductive plethysmography), nasal airflow (nasal pressure signal), arterial oxygen saturation (oximetry), limb movement (leg piezo), body position, pulse rate and waveform, and ECG. This system has been validated against 12-channel ‘in-hospital’ polysomnography for quantifying SDB.11–11 The electrodes and sensors for the portable device were attached by a trained and experienced sleep nurse in a hospital outpatient setting. The patients returned to the hospital the subsequent morning and the system was removed by the same nurse.

Sleep studies were analysed and scored by an experienced registered polysomnographic technologist. Definitions of respiratory events were as per recommendations of the American Academy of Sleep Medicine.14 An apnoea was defined as cessation of airflow of >10 s and hypopnoea as a >50% reduction of airflow lasting >10 s. An event was also considered to be a hypopnoea when there was a reduction in airflow which did not reach the 50% criteria, but was associated with either an arousal or an arterial oxygen desaturation of >3%. The apnoea–hypopnoea index (AHI) was calculated as the number of apnoeas and hypopnoeas per hour of sleep. Total sleep time was scored in minutes for each study using the Rechtschaffen and Kales criteria. We prospectively defined an AHI of >15 as ‘significant SDB’ because of the previously documented increased sensitivity of nasal pressure particularly in the detection of hypopnoeas when compared with nasal thermistor measurements.15–17 We have also demonstrated previously in our laboratory a median AHI of 7.7 in a young, non-obese control population when using the same methodology as was used in this study.20

All patients with paroxysmal AF were asked to characterize the number of (symptomatic) AF episodes in the 12 months prior to enrolment. AF burden was arbitrarily defined as low-frequency AF if patients estimated six or more episodes in the past year and high frequency if more than six episodes in the past year.

Patients were also asked whether episodes had predominant nocturnal onset (defined as either being woken at night time with symptoms of AF or waking in the morning in AF after having gone to bed in sinus rhythm).

In addition, all patients completed the Berlin sleep apnoea questionnaire.21

**Statistical analysis**

Study population characteristics were expressed as means (with standard deviation), medians (with interquartile range), and counts (with percentages). Conditional logistic regression analysis was used to compare cases and controls on relevant attributes and to determine the odds ratios (ORs) and 95% confidence intervals for the associations between significant SDB and explanatory variables, with adjustments for relevant covariates for the association between AF and SDB. Where zero counts prevented this, Fisher’s exact test was used. For continuous variables, analysis of variance was used to compare cases and controls, using the matched set as a factor in the analysis, to take account of the matching. Differences within the AF group were assessed using unpaired two-tailed t-test for continuous variables
Results

Characteristics of the study population

The AF and control groups were matched with respect to age and gender (Table 1). Ninety of 135 (67%) eligible AF patients and 45 of 67 (67%) eligible control patients consented to participate in the study. None of the patients had a known history or prior management of SDB.

As mentioned previously, all patients had an LV ejection fraction of >50% with no evidence of wall motion abnormality on transthoracic echocardiography. There were no significant differences in the prevalence of hypertension or in mean LV posterior wall thickness, the latter being within the normal range in both groups (Table 1). The AF group had a trend towards a slightly greater body mass index (BMI) (27.9 vs. 26.6 kg/m², \( P = 0.08 \)) and neck circumference (42.5 vs. 41.3 cm, \( P = 0.05 \)). The AF group had a significantly greater left atrial size (4.13 vs. 3.86 cm, \( P = 0.01 \)). There was a trend towards higher usage of beta blockers (AF 24% vs. control 11%, \( P = 0.05 \)), flecainide (AF 13% vs. control 2%, \( P = 0.08 \), and amiodarone (AF 8% vs. control 0%, \( P = 0.09 \)) in the AF patients and a significantly increased prescription of sotalol in the AF group (AF 24% vs. control 4%, \( P = 0.01 \)). There was no difference in the prescription of other antiarrhythmic medications, sedatives, or antidepressants between groups. There was no significant difference in alcohol consumption between the groups (AF median 0–14 standard drinks per week vs. control median 4, IQR 0–12 standard drinks per week, \( P = 0.3 \)).

Polysomnography data

No patients were excluded from the study due to poor quality recordings (Table 2). The average total hours of available recording were similar in both groups. The sleep period time (from the time of the first 30 s epoch of sleep to the final 30 s epoch of sleep), total sleep time (actual time EEG recorded sleep within the total sleep period), and the sleep efficiency (total sleep time/sleep period time, %) were also similar in the two groups. The mean oxygen saturation nadir was significantly lower in the patients with AF (86.90 ± 5.19) compared with controls (88.49 ± 3.62, \( P = 0.04 \)). None of the control patients had AF during overnight polysomnography. Twenty seven of the 90 AF patients (30%) had AF documented during the recording period. Five of the 63 paroxysmal AF patients (8%) and 22 of the 27 persistent AF patients (81%) had AF recorded during the sleep study recording period.

Atrial fibrillation group

In the AF group, the mean duration of AF symptoms was 6.21 ± 4.45 years. There were 63 (70%) patients with paroxysmal AF and 27 (30%) with persistent AF. Of the 63 patients with paroxysmal AF, 17 (27%) had predominantly nocturnal onset of episodes; their mean estimated number of AF episodes in the preceding 12 months was 31 and ranged from 0 episodes to daily episodes.

Table 1  Study population characteristics

<table>
<thead>
<tr>
<th></th>
<th>AF patients (n = 90)</th>
<th>Control patients (n = 45)</th>
<th>P-value</th>
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</thead>
<tbody>
<tr>
<td>Male gender, n (%)</td>
<td>74 (82)</td>
<td>37 (82)</td>
<td></td>
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<tr>
<td>Age, years</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Mean ± SD</td>
<td>56 ± 12</td>
<td>54 ± 11</td>
<td></td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>58 (48–63)</td>
<td>56 (48–62)</td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>27.9 ± 4.5</td>
<td>26.6 ± 3.0</td>
<td>0.08</td>
</tr>
<tr>
<td>Median (IQR)</td>
<td>27 (25–30)</td>
<td>26 (24–29)</td>
<td></td>
</tr>
<tr>
<td>Neck circumference, cm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>42.5 ± 3.8</td>
<td>41.3 ± 3.8</td>
<td>0.05</td>
</tr>
<tr>
<td>Median IQR</td>
<td>42 (40–45)</td>
<td>41 (39.5–44)</td>
<td></td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>33 (37%)</td>
<td>12 (27%)</td>
<td>0.14</td>
</tr>
<tr>
<td>LV posterior wall thickness, cm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>0.95 ± 0.15</td>
<td>0.97 ± 0.14</td>
<td>0.41</td>
</tr>
<tr>
<td>Left atrial size, cm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>4.14 ± 0.55</td>
<td>3.86 ± 0.50</td>
<td>0.01</td>
</tr>
<tr>
<td>AF rhythm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paroxysmal, n (%)</td>
<td>63 (70)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Nocturnal, n (% of paroxysmal)</td>
<td></td>
<td>17 (27)</td>
<td>NA</td>
</tr>
<tr>
<td>Persistent, n (%)</td>
<td>27 (30)</td>
<td>NA</td>
<td></td>
</tr>
</tbody>
</table>

IQR indicates interquartile range; NA, not applicable.
Control population

The control population consisted of 19 patients undergoing ablation for SVT, five patients being investigated for syncope, eight patients with bradycardia requiring pacemaker insertion, six patients undergoing ablation for idiopathic VT with no structural heart disease, four patients with palpitations, two patients referred for genetic testing because of a family history of sudden death, and one patient with asymptomatic pre-excitation.

Symptoms related to sleep disordered breathing

When using the Berlin Questionnaire alone to diagnose SDB, 46% of patients with AF were classified as ‘high risk’ compared with 18% in the control patients. The mean AHI in patients diagnosed as high risk with the Berlin Questionnaire was 26.33 ± 19.91, which was higher than the mean AHI of 16.94 ± 15.43 in those at ‘low risk’. The median AHI was also higher in the high risk (20.6) as opposed to the low risk group (14.0). However, there was considerable overlap over a wide range of AHIs between patients diagnosed as having high or low risk on the Berlin Questionnaire (Figure 1).

Association of atrial fibrillation with sleep disordered breathing

The mean AHI in patients with AF was significantly higher than in the control group (23.19 ± 19.26 vs. 14.66 ± 12.43, P = 0.01). This observation extends to the median AHI, which is greater in the AF group (18.65, IQR 8.58–31.78 vs. 9.8, IQR 6.1–19.2). Furthermore, the proportion of patients with significant SDB (AHI > 15) was also significantly higher in the AF group than in the control population (62 vs. 38% respectively, P = 0.01, Figure 2).

Univariate analysis demonstrated an unadjusted OR of 2.69 (95% CI 1.27–5.68, P = 0.01) for the association of AF with SDB (AHI > 15) (Table 3). Univariate analysis also demonstrated an association of SDB (AHI > 15) with neck circumference (OR 1.19; 95% CI 1.03–1.38, P = 0.02, Table 3) and BMI (OR 1.24; 95% CI 1.06–1.45, P = 0.01, Table 3). Using conditional logistic regression analysis, after adjustment for hypertension, BMI, and neck circumference, there was a significant relationship between SDB (AHI > 15) and AF (OR for AF = 3.04, 95% CI 1.24–7.46, P = 0.02) (Figure 3).

Analyses in which AHI was treated as a continuous variable and accounting for matching demonstrated a P-value of 0.01 for the association between AHI and AF.

Paroxysmal atrial fibrillation

In patients with paroxysmal AF, AF burden was associated with both a higher prevalence and severity of SDB. For those patients defined as having low-frequency AF (n = 35) (six or less episodes in prior year), the median number of AF episodes in the prior year was 1.0. In this group, the mean AHI was 16.69 ± 15.06. For those patients defined as having high-frequency AF (n = 28) (more than

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Table 2 Polysomnography data

<table>
<thead>
<tr>
<th></th>
<th>AF patients (n = 90)</th>
<th>Control patients (n = 45)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean AHI(^a)</td>
<td>18.7 (8.5–32.2)</td>
<td>9.8 (6.1–19.2)</td>
<td>0.01</td>
</tr>
<tr>
<td>Obstructive apnoea index(^a)</td>
<td>0.63 (0–2.4)</td>
<td>0 (0–1.3)</td>
<td>0.14</td>
</tr>
<tr>
<td>Central apnoea index(^a)</td>
<td>0 (0–0.58)</td>
<td>0 (0–0.19)</td>
<td>0.03</td>
</tr>
<tr>
<td>Hypopnoea index(^a)</td>
<td>15.7 (7.6–21.9)</td>
<td>9.3 (6.1–17.9)</td>
<td>0.05</td>
</tr>
<tr>
<td>Total time available (min)(^b)</td>
<td>509.0 ± 74.39</td>
<td>498.10 ± 67.7</td>
<td>0.39</td>
</tr>
<tr>
<td>Sleep period time (SPT) (min)(^b)</td>
<td>481.13 ± 70.91</td>
<td>470.20 ± 70.00</td>
<td>0.38</td>
</tr>
<tr>
<td>Total sleep time (TST) (min)(^b)</td>
<td>402.11 ± 63.47</td>
<td>397.70 ± 68.50</td>
<td>0.70</td>
</tr>
<tr>
<td>Sleep efficiency: TST/SPT x 100(^b)</td>
<td>83.81 ± 10.00</td>
<td>84.70 ± 8.77</td>
<td>0.65</td>
</tr>
<tr>
<td>Mean oxygen saturation (%)(^b)</td>
<td>95.41 ± 1.44</td>
<td>95.30 ± 1.32</td>
<td>0.61</td>
</tr>
<tr>
<td>Oxygen saturation nadir (%)(^b)</td>
<td>86.89 ± 5.19</td>
<td>88.49 ± 3.62</td>
<td>0.05</td>
</tr>
</tbody>
</table>

\(^a\)Median (interquartile range).
\(^b\)Mean ± standard deviation.
In those with paroxysmal AF, patients with a predominant nocturnal onset did not show a worse severity of SDB than patients with episodes that did not demonstrate diurnal variation (AHI 17.69 ± 12.4 nocturnal vs. 23.25 ± 21.65 non-nocturnal, P = 0.21). The proportion of patients with significant SDB (AHI > 15) was 65% (nocturnal) vs. 54% (non-nocturnal) (P = 0.57).

Discussion

This study demonstrates a high prevalence of SDB in a relatively young AF population (mean age 55 years) with normal LV function and no structural heart disease. This was present in patients with paroxysmal as well as those with persistent AF. Over half of the AF patients have significant SDB compared with a significantly lower prevalence in an age- and sex-matched control population with other (non-AF) cardiac arrhythmias. This observation was made in a group of patients without many of the traditional risk factors for sleep apnoea and who had limited other co-morbidities. Furthermore, in the group with paroxysmal AF, the likelihood of significant SDB was coupled to the AF burden with high-frequency AF associated with a higher prevalence of significant SDB. These findings suggest a causative link between SDB and AF which extends beyond the established association with other AF risk factors such as LV dysfunction. Hypertension, which was present in approximately one-third of patients, was observed in a similar proportion in both groups and thus could not account for the different rates of SDB. There was a higher prescription of sotolol in the AF patients (24 vs. 4%). Although there is no specific data relating to sotolol, there is some evidence showing that beta blockers improve central sleep apnoea and have no effect on obstructive sleep apnoea. On multivariable analysis, the strongest independent predictor for the development of AF was significant SDB.

We also observed a similar prevalence of SDB in patients with paroxysmal AF whose episodes showed nocturnal onset compared with patients in whom there was no diurnal variation.
Previous studies
In recent years there has been an increasing awareness of a relationship between AF and sleep apnoea. Initial studies demonstrating the increased prevalence of sleep apnoea in heart failure also observed an increased occurrence of AF in those with sleep apnoea.1–5 Javaheri et al.3 studied 81 patients with heart failure and found that 51% had sleep apnoea. The patients with sleep apnoea also had a significantly higher prevalence of AF (22 vs. 5%). Similarly, patients with an AHI ≥ 5 undergoing coronary artery bypass surgery were found to have a higher incidence of post-operative AF (32 vs. 18% in patients without SDB). Other studies of patients with sleep apnoea have observed a higher prevalence of nocturnal arrhythmias compared with control patients who did not have sleep apnoea.2,7,25 In the sleep heart health study, the prevalence of AF was higher in 228 subjects with SDB than in 338 subjects without (4.8 vs. 0.9%; \( P = 0.003 \)).

Against this background, two recent studies have looked at the prevalence of sleep apnoea in a population of patients presenting for clinical management of AF, in order to determine whether this may be an important factor in the broader population of AF patients. These studies have yielded seemingly divergent results. Porthan et al.,6 using overnight sleep studies, found a similarly high prevalence of sleep apnoea in 59 lone AF patients (32%) as in 56 control patients (29%, \( P = 0.67 \)) and concluded that sleep apnoea was not more common in AF than in control patients. However, the control group was derived from respondents to a letter, 43% of which were not answered. As the authors point out, this raises the possibility of bias in the control group towards those with symptoms. Furthermore, although not statistically significant, the proportion of AF patients with moderate or severe SDB was double that of the control group. The landmark study by Gami et al.,9 observed a significantly higher prevalence of sleep apnoea in 151 patients with AF referred for cardioversion (49%) than in a control population of 373 general cardiology patients (32%). The investigators used the Berlin Questionnaire to diagnose sleep apnoea. In this study, the mean patient age was 71 years, 33% had coronary artery disease, 19% congestive heart failure, and all patients had persistent AF (referred for cardioversion). Our study is consistent with the seminal results of Gami et al. and extends them to a different population recruited from an arrhythmia outpatient setting. In the current study, mean patient age was much younger (55 years), no patients had heart failure or coronary artery disease, and the majority (70%) had paroxysmal AF. In addition, by diagnosing sleep apnoea utilizing the Berlin Questionnaire, Gami et al. have selected a population of patients that is symptomatic with regard to their SDB. This is an important point, as our study reports objective data regarding SDB irrespective of whether or not the patients had symptoms relating to their SDB.

The prevalence of SDB in our control population was high (38%) but comparable with previous reports.8,9 A higher AHI is also attributable to the use of nasal pressure rather than the nasal thermistor technique.15–19

We also found an association between symptomatic AF burden and severity of SDB with a higher AF burden linked to worse SDB.

Sleep apnoea and atrial fibrillation: possible mechanistic link
Although this study did not address the possible mechanisms underlying the association between SDB and AF, it is worthy of brief speculation. SDB can result in recurrent episodes of hypoxemia and hypercapnia together with marked haemodynamic and autonomic fluctuations. We certainly found a significantly lower oxygen saturation nadir in the AF group when compared with the control patients. Resultant pulmonary or systemic hypertension, catecholamine, and stretch-mediated channel activation may also contribute in varying degrees to a tendency towards AF. In addition, sleep apnoea may cause elevations of potent pro-inflammatory factors such as C-reactive protein,26,27 which may also be elevated in patients with AF.18

An alternate hypothesis is that AF may contribute to the development of sleep apnoea perhaps by elevation of left atrial pressures leading to pulmonary congestion. Certainly in the case of patients with systolic heart failure, AF is primarily seen in those patients with central rather than obstructive sleep apnoea.3 Also, recently, Garrigue et al. demonstrated that atrial overdrive pacing reduced the amount of both central and obstructive sleep apnoea in patients with a permanent pacemaker implanted for sinus node dysfunction. However, other studies have failed to demonstrate any improvement in obstructive SDB with atrial overdrive pacing, and any possible role of AF in the development of this condition is speculative.30–32

Limitations
It has been demonstrated that up to 50% of patients with paroxysmal AF have episodes which are asymptomatic and thus it is difficult to assess the true AF burden without continuous or repeated monitoring. Future studies using more stringent assessment of AF burden are required to ascertain whether the association between AF burden and significant SDB is upheld with smaller gradations in AF burden.

A limitation of current technology is the difficulty in clearly differentiating obstructive vs. central SDB. There is significant overlap, and classification particularly of hyperpnoeas is not recommended. This is reflected in the most recent AASM guidelines for classification and scoring of sleep events.33 Thus, we have not formally categorized patients as having either obstructive or central SDB, as the majority of events were hypopnoeas.

Conclusion and clinical implications
The current study describes a high prevalence of SDB in young patients with both paroxysmal and persistent AF and with normal LV function. Thus this population warrants careful consideration of the diagnosis of SDB. An important study by Kanagala et al.34 demonstrated that treatment of obstructive sleep apnoea in patients undergoing cardioversion of persistent AF reduced the rate of AF recurrence. Whether treatment of SDB in patients with paroxysmal AF will lead to a reduction in episodes is an intriguing question worthy of further study.
Conflict of interest: none declared.

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References

A 61-year-old man with atypical angina pectoris showed inferolateral ischaemia on exercise single-photon emission computed tomography (SPECT) myocardial perfusion imaging. Electrocardiogram (ECG)-gated multi-slice computed tomography (MSCT, Aquilion ONE, Toshiba Medical Systems) with 320 simultaneous detector rows (each 0.5 mm wide) was performed at 120 kV and 450 mA after intravenous injection of 80 mL of an iodinated contrast agent (iobitridol, Xenetix 350, Guerbet). This very recently developed MSCT scanner covers 16.0 cm (Panel A, frontal view) in a single rotation and heart beat thus virtually eliminating artefacts arising from breathing or irregular cardiac activity. The volume acquired with a single X-ray shot looks like a cylinder (insets in Panel B, the latter being a lateral view, Ao=aorta) bounded by two circular cones (the angle of the X-ray beam is 15°, Panels A and B). Therefore, this new CT scanner that will become commercially available in 2008 is also referred to as ‘cylindrical wide-area detector CT’. In our patient, three-dimensional volume-rendered reconstructions (Panels C–E) as well as curved multi-planar reformations along the vessel centrelines (insets in Panels C–E) clearly demonstrate significant stenoses (arrows) in all three main coronary arteries. The stenosis in the posterolateral artery (PLA) was caused by a purely non-calcified plaque (inset in Panel C, arrowhead), whereas the stenoses in the left anterior descending (LAD) and obtuse marginal artery (OM) were caused by plaques with calcified and non-calcified components (insets in Panels D and E, arrowheads). There was excellent correlation with subsequently performed conventional coronary angiography (arrows in Panels F–H), during which the patient immediately underwent stent placement for the OM artery stenoses (responsible for the ischemia on SPECT). Further interventional procedures are scheduled. It is important to note that 320-slice coronary CT angiography dispenses with overscanning and overranging and thus has the potential to reduce the radiation exposure by a factor of 2–4 to <5 mSv as in the patient presented here.