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Original Article

Long-term effect of cardiac pacing on sleep-disordered breathing in patients with conventional indications for a permanent pacemaker

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ABSTRACT

Background: The effect of cardiac pacing on sleep-disordered breathing is controversial. We investigated the long-term effect of cardiac pacing on sleep disordered breathing in patients with conventional indications for permanent pacemakers

Methods: Subjects comprised 40 patients (29 men; mean age 69 ± 9 years, mean left ventricle ejection fraction 69 ± 8%, and body mass index 23.6 ± 3.5 kg/m^2) who were diagnosed with indications for permanent pacemakers (sick sinus syndrome in 23 patients, atrioventricular block in 15, and brady atrial fibrillation in 2). All patients received polysomnographic evaluations before implantation of permanent pacemakers. After implantation of permanent pacemakers, all patients received polysomnographic evaluations during use of the pacemaker settings (AAI/DDD/VVI at 70 beats per minute).

Results: The mean follow-up period was 35 ± 13 months. Before implantation, the distribution of sleep-disordered breathing was as follows: 93% had apnea hypopnea index > 5, 58% had apnea hypopnea index > 15, and 20% had apnea hypopnea index > 30. The mean apnea hypopnea index for all patients was 20 ± 15, for those with obstructive type apnea was 4.9 ± 5.3, and for those with central type apnea was 3.0 ± 4.5. The mean Epworth Sleepiness Score was 5.9 ± 4.0. No patient received continuous positive airway pressure therapy or any other therapy for sleep-disordered breathing during the follow up period. The mean apnea hypopnea index at 1 week after implantation of permanent pacemakers was 21 ± 14 (P=0.8) and the mean apnea hypopnea index at end of follow-up was 11 ± 7 (P < 0.0001).

Conclusion: Long term cardiac pacing significantly reduces the number of episodes of sleep apnea in patients with conventional permanent pacemaker indications.

1. Introduction

Garrigue et al. reported that atrial overdrive pacing reduces the number of sleep apnea episodes in pacemaker patients without heart failure [1]. However, this finding has not been replicated in subsequent investigations [2–5], although some meta-analysis studies [6,7], reported some effect of pacing therapy on sleep apnea. All of these studies investigated relatively short periods of pacing. Therefore, the effect of cardiac pacing on sleep-disordered breathing (SDB) is still controversial. We investigated the long-term effect of cardiac pacing on SDB in patients with conventional indications for permanent pacemakers (PPM).

2. Materials and methods

Consecutive patients with conventional indications for pacemakers in our hospital were included. Subjects comprised 40 patients (29 men; mean age 69 ± 9 years; mean left ventricle ejection fraction 69 ± 8%, and body mass index 23.6 ± 3.5 kg/m^2) who were diagnosed with indications for PPM (sick sinus syndrome in 23 patients, atrioventricular block in 15 patients, brady atrial fibrillation in 2 patients).

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All patients received polysomnographic evaluations using the Morpheus C® (Teijin, Japan) before implantation of PPM. This ambulatory cardiorespiratory polygraph records nasal airflow, chest and abdominal wall movements, oxygen saturation, heart rate, and Holter ECG. An apnea event was defined as an absence of airflow for ≥10 s. Central-type sleep apnea (CSA) was defined as an apnea event with absence of chest and abdominal wall motion for more than one-third of its duration. Obstructive-type sleep apnea (OSA) was defined as an apnea event with presence of chest and abdominal wall motion for more than one-third of its duration. Mixed-type sleep apnea (MSA) was defined as an apnea event of other than the central or obstructive types. A hypopnea event was defined as a reduction in respiratory airflow of >50% for 10 s associated with desaturation of >3%.

After implantation of PPM, all patients received polysomnographic evaluations during use of the pacemaker settings (AAI/DDD/VVI at 70 beats per minute) using the Morpheus C® during the in-hospital-period (acute phase) and using the SAS 2100® (Teijin, Japan) during the out-patient period (chronic phase).

The SAS 2100® records nasal airflow, oxygen saturation, and pulse rate.

No patient received continuous positive airway pressure therapy or any other therapy for SDB during the follow-up period. All subjects signed informed consent as approved by our hospital ethics committee.

Continuous variables are expressed as means ± standard deviation (SD) and categorical variables as counts or percentages. Comparisons between the 2 study groups were performed using Pearson's s^2 test for categorical and Student t test for continuous variables, respectively. Comparisons between baseline and follow-up data were performed by paired t test. A P value <0.05 was considered significant. SPSS software, v.19.0.0, was used for all statistical analyses (SPSS Inc., IL, USA).

3. Results

Baseline characteristics and co-existing diseases are shown in Tables 1 and 2, respectively. Before implantation, the mean Epworth sleepiness score (ESS) was 5.9 ± 4.0 points. The apnea hypopnea index (AHI) of all patients was 20 ± 15/h, OSA index was 4.9 ± 5.3/h, and CSA index was 3.0 ± 4.5/h. The prevalence of SDB was 93% (using a cut-off of AHI > 5), 58% (using a cut-off of AHI > 15) and 20% (using a cut-off of AHI > 30) respectively. When SDB was defined as AHI > 15 there were no significant differences in baseline characteristics except for body mass index (BMI) (P=0.0345, Fig. 1). Seventeen per cent of patients were only in the OSA group (OSA only) (n=4). Eighty-three per cent of patients were included in the CSA group (CSA(+)) (n=19), while 39% of patients were in a group that predominantly expressed CSA characteristics (CSA > OSA) (n=9) (Table 3). When SDB was defined with an AHI cut-off level >15, there were no significant differences in baseline characteristics or co-existing diseases (Tables 1 and 2).

A representative case is illustrated in Fig. 1. The patient was a 73 year-old man with sick sinus syndrome. Before implantation of PPM, his BMI was 24.9 kg/m^2, his cardio-thoracic ratio (CTR) was 53%, his brain natriuretic peptide level (BNP) was 271.2 pg/mL, and his ESS was 4 points. His AHI was 38.4/h, his apnea index (AI) was 20.7/h, his OSA index was 15.7/h, his CSA index was 2.7/h, and his hypopnea index (HI) was 4.8/h (Fig. 1A). At the end of follow-up (962 days (32 months) after implantation of PPM), his AHI was 7.6/h, his AI was 6.4/h, and his HI was 1.2/h (Fig. 1B). He was not given continuous positive airway pressure therapy or any other treatment for sleep apnea.

The mean follow-up period of the 40 patients was 35 ± 13 months. The mean AHI at 1 week after implantation of PMM was 21 ± 14/h (P=0.8850) and the mean AHI at the end of the follow-up period was 11 ± 7/h ($P<0.0001$) (Table 3). The AHI and incidence of desaturation >3% were improved in all patient groups (all patients, AHI > 15, OSA only, CSA (+), and CSA > OSA) at the end of follow-up (Table 3). Mean BMI at end of follow-up was 23.3 ± 3.5 kg/m^2 in all patients (P=0.2427, vs. baseline), 22.2 ± 2.9 kg/m^2 in the SDB(−) group (P=0.9359, vs. baseline) and 24.0 ± 3.8 kg/m^2 in the SDB(+) group (P=0.1041, vs. baseline), respectively. We did not vary the pacing rate from 70 bpm. The average heart rate of end of follow-up was derived from detected pulses using the SAS 2100. These data are potentially affected by occurrence of premature beats (Table 3). AHI changes in individual patients are shown in Fig. 2.

Table 1
Patients' baseline characteristics.

	All (n=40)	SDB(−) (n=17)	SDB(+) (n=23)	(−) vs. (+) P
Age (y)	69.0 ± 9.3	68.0 ± 10.8	69.7 ± 8.3	0.5528
BMI (kg/m^2)	23.6 ± 3.5	22.2 ± 2.9	24.6 ± 3.7	0.0345
CTR (%)	52.7 ± 6.0	52.2 ± 6.8	53.0 ± 5.6	0.6796
LVEF (%)	69.3 ± 8.2	67.8 ± 8.7	70.3 ± 7.8	0.3554
BNP (pg/mL)	225 ± 366	215 ± 356	232 ± 382	0.9078

SDB=Sleep disordered breathing; SDB(+)=Apnea-Hypopnea Index >15; BMI=body mass index; CTR=cardio-thoracic ratio; LVEF=left ventricular ejection fraction; BNP=brain natriuretic peptide; and ESS=Epworth Sleepiness Score.

Table 2
Patients' co-existing diseases.

	All (%) (n=40)	SDB(−) (%) (n=17)	SDB(+) (%) (n=23)	(−) vs. (+) P
Hypertension	55	41.20	65.20	0.2343
Diabetes mellitus	27.50	29.40	26.10	>0.9999
Dyslipidemia	22.50	5.90	34.80	0.0749
Gout or high uremic acid	20	11.80	26.10	0.4717
Ischemic heart disease	2.50	5.90	0	0.8779

SDB=Sleep disordered breathing and SDB(+)=Apnea-Hypopnea Index >15.

4. Discussion

Young et al. reported that prevalence of SDB (defined using a cut-off of AHI≧5) was 9% in women and 24% in men in the general population [8]. In this study, prevalence of SDB was very high in patients with conventional indications for PPM. The prevalence of SDB was 58% when the cut-off level was defined as AHI > 15 and 20% when the cut-off level was defined as AHI > 30, respectively. In a previous study, the majority of patients with implanted PPM suffered from unrecognized SBD [9]. In that study, the prevalence of SDB was 59% when the cut-off level was defined as AHI > 10 and 27% when the cut-off level was defined as AHI > 30, respectively.

In our study, the mean AHI was lowered significantly at the end of the follow-up period and these findings were seen in all patient categories, occurring without changes in BMI. We used different polysomnographic systems for evaluation in the acute and chronic phases. There could be some differences in sensitivity for AHI between these systems. However, there was good correlation between AHI and the index of desaturation >3% determined by the polysomnographic systems. The index of desaturation <3% was also improved in all patient groups (all patients, AHI > 15, OSA only, CSA (+), and CSA > OSA) at the end of follow-up.