

Biatrial Pacing for Prevention of Lone Atrial Fibrillation

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Summary

40 patients with frequent episodes of drug refractory atrial fibrillation/flutter related to interatrial conduction disturbances (defined as possessing the P-wave duration exceeding 120 ms) received a pacing system for permanent biatrial stimulation. All patients had a normal sinus rate and had no structural heart disease (lone atrial fibrillation). Biatrial pacing was effected by a dual-chamber pacemaker using a standard right atrial lead and a specially designed coronary sinus lead connected to the "ventricular" channel of the pacemaker. The AV-delay of the pacemaker was shortened to 0 ms, providing right-atrial-triggered left atrial pacing with simultaneous excitation of both atria. This stimulation pattern results in a shortening of the atrial impulse propagation as documented by a P-wave shortening of 36.3 ± 20.7 ms. 29 patients had no recurrences of atrial fibrillation during the follow-up period (4 of them with antiarrhythmic drug therapy), in 5 patients recurrences were reduced, and atrial fibrillation was not influenced in 5 patients. In one case, a coronary sinus electrode could not be implanted.

Key Words

Antiarrhythmic therapy, biatrial pacing, interatrial conduction block, resynchronization

Introduction

The development of biatrial pacing has led to an improvement of tachyarrhythmia prevention. It is reported that pacing providing an atrial contribution (AAI/DDD mode) and rate normalization in sick sinus syndrome, particularly the bradycardia-tachycardia syndrome, has an antiarrhythmic effect on atrial fibrillation [4-7]. These investigations favored studies on biatrial pacing. In 1972, Dayem and co-workers [1] described the connection between interatrial conduction disorders and atrial tachyarrhythmias. Since 1990, Daubert et al. [2] have investigated arrhythmia prevention through permanent atrial resynchronization, and more recently [3] they have summarized their clinical experience with biatrial synchronous pacing in patients with atrial conduction block. They used a single-chamber pacemaker (AAT mode) and a Y-bifurcated connector to connect two leads for simultaneous atrial stimulation in patients with normal AV conduction. In cases where AV conduction disorders were present, a dual-chamber pacemaker was implanted with the ventricular port taken for sensing and stimulation. This study was performed with a standard dual-chamber pacemaker that was used only for atrial resyn-

chronization. The ventricular port was connected to the left atrium. Therefore, the atria could be paced independently, offering the possibility of right atrial stimulation when there is no sinus node activity.

Materials and Methods

40 patients (15 female, 25 male) with a mean age of 64.6 ± 7.8 years and at least one occurrence of atrial fibrillation per month were treated with biatrial pacing. When selecting patients for the presented study, we deliberately restricted our choice to those presenting aggressive, drug-resistant paroxysmal atrial fibrillation and atrial conduction block. Sinus bradycardias (monitored by Holter-ECG/L-ECG) and additional heart diseases (examined by clinical thoracic X-rays, echocardiography, TSH levels) were excluded. All patients had an interatrial conduction disturbance with P-wave durations exceeding 120 ms.

Furthermore, the left atrial diameters (LA < 40 mm) and the hemodynamic functions (EF \geq 50%; documented by Echo) of the patients were normal.

A standard dual-chamber (DDD) pacemaker (LOGOS,

BIOTRONIK) that permits shortening the AV-delay to 0 ms was implanted. Right atrial pacing was done with a commercially available, standard bipolar screw-in lead (YP-BP, BIOTRONIK).

After dislocation problems and unstable pacing conditions with conventional leads, the aim of the study (approved by the Ethics commission in October 1997) was to develop a coronary sinus lead which could be securely fixated and easily handled. As a result, left atrial pacing was performed with a custom-made coronary sinus lead (BIOTRONIK) with an electrically inactive, silicone-threaded end distal to the ring electrodes (Figure 1). This silicone thread is advanced as far as possible into a side branch of the coronary sinus and fixated by turning (Figure 2, thoracic X-ray). By connecting the right atrial lead to the atrial channel of the pacemaker and the coronary sinus (CS) lead to the "ventricular" channel, sensing of a right atrial potential leads to immediate left atrial pacing via the coronary sinus when the AV-delay is programmed to 0 ms.

After experiencing initial fixation problems with conventional leads for the coronary sinus electrodes, we implanted biatrial pacemakers with the CS lead described above in 40 patients from October 1997 to October 1998.

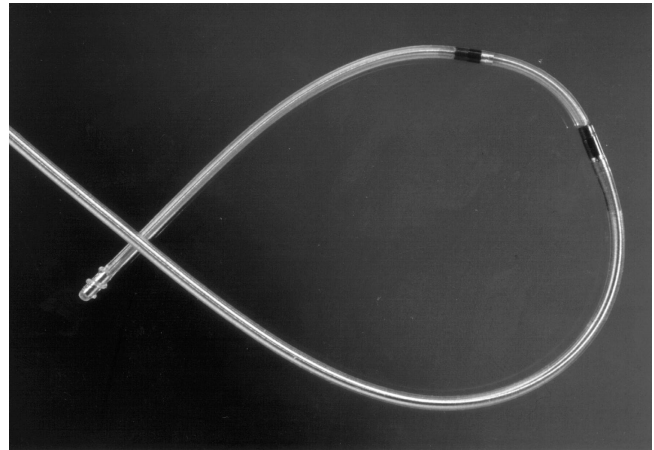


Figure 1. Coronary sinus lead for left atrial pacing.

Results

The newly developed lead could be positioned and fixated without problems in 36 patients. In the remaining 3 patients, the CS lead could not be fixated after two attempts, and in one patient an approach to the coronary sinus was not possible. Within 36 hours, dislocation occurred in 4 patients and a high threshold (> 4 V) was observed in 3 patients. Reoperation was performed to achieve a correct CS lead position.

The following electrophysiological results were obtained: The pacing threshold measured in the right atrium was

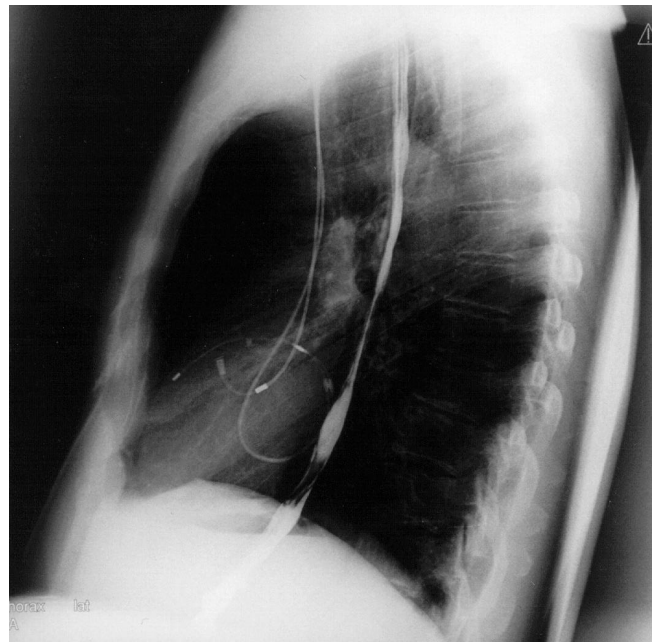
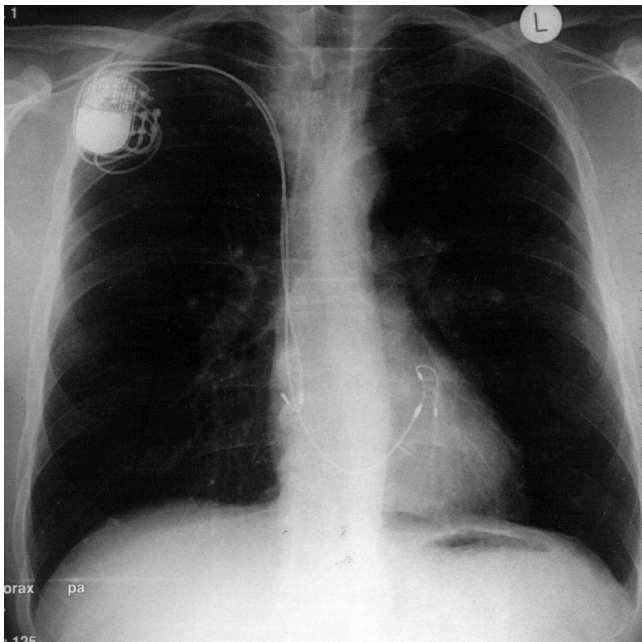


Figure 2. X-rays of thorax with CS lead fixed in a side branch of the coronary sinus.

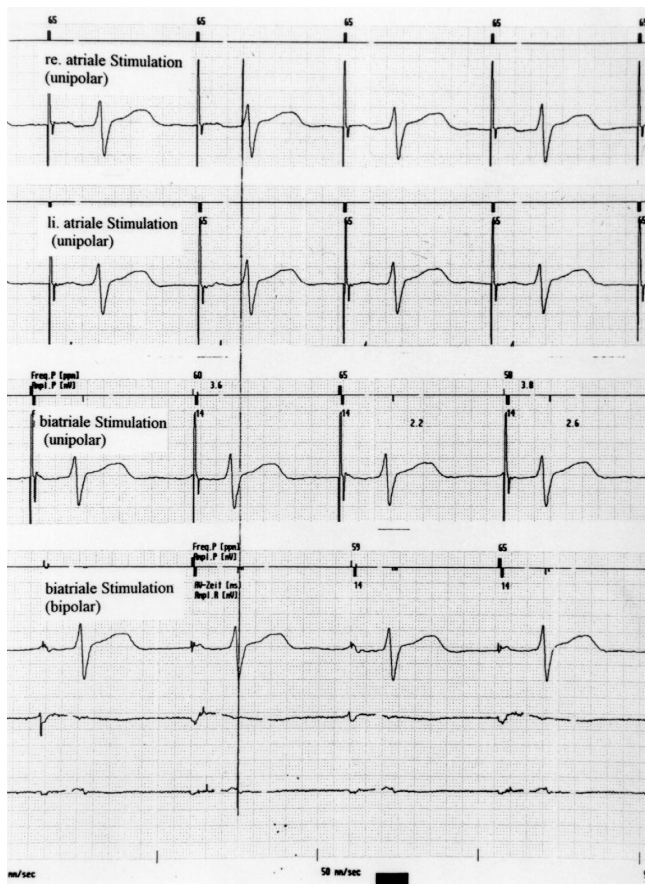


Figure 3. ECG during biatrial pacing; the shortened P wave and the earlier occurrence of the previously delayed left atrial depolarization and contraction is visible.

0.95 ± 0.75 V (0.5 ms pulse width); the atrial signal amplitude, 2.46 ± 0.88 mV. The pacing threshold in the coronary sinus assumed a value of 1.57 ± 0.86 V (0.5 ms pulse width) and a signal amplitude was 3.58 ± 1.39 mV. The spontaneous P-wave duration, obtained from surface ECG (lead II) was 121.2 ± 26.7 ms and the shortening of the P-wave duration during biatrial pacing was determined to be 36.3 ± 20.7 ms. The data are listed in Figures 4 and 5 and in Table 1. Consequently, the duration of the P-wave is shortened because the previously delayed left atrial depolarization and contraction occurs earlier (Figure 3).

This investigation demonstrates that biatrial pacing leads to an inhibition of atrial fibrillation: 29 patients had no recurrences of atrial fibrillation; with 4 of them being administered antiarrhythmic drugs. Recurrence was reduced in 5 patients, and the treatments did not have any influence on the prevalence of atrial fibrillation in 5 patients.

Pacing threshold RA	0.95 ± 0.75 V (0.5 ms pulse width)
Signal amplitude RA	2.46 ± 0.88 mV
Pacing threshold CS	1.57 ± 0.86 V (0.5 ms pulse width)
Signal amplitude CS	3.58 ± 1.39 mV
P-wave duration (II)	121.2 ± 26.7 ms
P-wave shortening during biatrial stimulation	36.3 ± 20.7 ms

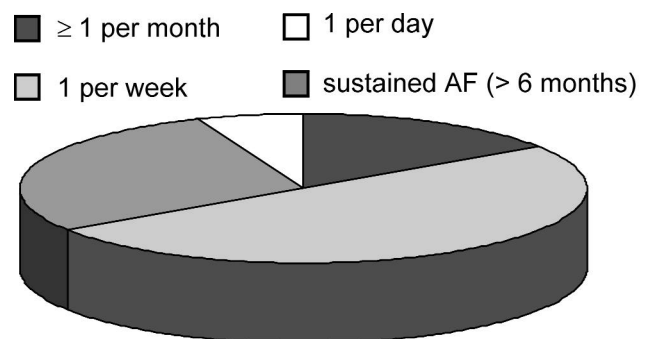
Table 1. Patient data and AF prevalence prior to implantaion.

Discussion

Atrial fibrillation is the most frequently occurring arrhythmia. Antiarrhythmic drug therapy is often characterized by ineffectiveness, side effects or proarrhythmia. The risk of embolism originating from the fibrillating atria necessitates permanent anticoagulation treatment, which has been associated with bleeding complications in the elderly.

There are hemodynamic reasons for the development of atrial fibrillation, such as atrial dilatation resulting from enddiastolic pressure increase, ventricular relaxation disturbances, and cardiomyopathy in cardiac failure. In such cases, a prophylactic therapy may be attempted only when antiarrhythmic drug therapy is combined with treatment of the underlying disease.

In contrast, some cases of atrial fibrillation are primarily of rhythmological origins and are apparently also triggered by an increase in atrial wall stress. Atrial tachycardia can be triggered by frequent retrograde atrial depolarizations with atrial contractions against closed AV valves in the presence of a long-lasting AV nodal rhythm or bigeminy. Treating supraventricular bradycardia in the presence of ventriculoatrial conduc-



n = 40 (15 female, 25 male) age = 64.6 ± 7.87 years

Figure 4. Patient data and AF prevalence prior to implantaion.

tion through inappropriate ventricular pacing therapy (VVI/VVIR pacing in sick sinus syndrome) has become the classic clinical model of how this form of atrial fibrillation develops [9].

With AAI/DDD pacing, which provides the atrial contribution, artificial stabilization of the atrial rate and maintenance/restoration of AV synchrony have been proven to have a long-lasting preventative effect [5]. However, this effect appears to be limited in the presence of additional interatrial conduction disturbances. Disturbed conduction between the atria may be the cause of atrial fibrillation that occurs during DDD pacing when the AV-delay is inadequate for the individual patient [10].

Another form of atrial fibrillation/flutter is related to a prolongation of the interatrial impulse propagation [3]. To a great extent, it is refractory to drug therapy; the disturbance of the interatrial conduction manifests itself in the ECG in prolonged P-wave durations, often exceeding 120 ms. Accompanying bradycardias, heart dilatation or other heart diseases are usually not observed.

Biatrial pacing methods are obvious therapeutic options for treating these cases of atrial fibrillation. The methods aim to shorten the disturbed impulse propagation by triggering the left atrial contraction earlier. Clinical studies with permanent pacemakers - mainly in patients with sick sinus syndrome and interatrial conduction blocks - have been presented by Saksena [8] (dual-site atrial pacing) and by Daubert [1] (right atrial pacing by means of conventional leads and left atrial pacing via the coronary sinus). Both stimulation patterns resulted in reduced fibrillation episodes.

In this study, the use of two separate channels for the right and left atrium improves patient safety with respect to atrial sensing, as left atrial pacing can be performed independently of right atrial pacing.

The new silicone-threaded lead did not cause any complications (e.g., coronary sinus thrombosis, perforations, or similar). Problems with high or greatly fluctuating thresholds, as encountered in three patients, are expected to be prevented by improved lead design in the future.

Our pacing method - left atrial pacing initiated with the beginning of right atrial excitation mediated by a "DDD" pacemaker with a 0 ms delay time - has resulted in a reduction in the P-wave duration by about 36 ms and in a (for us surprising) total absence of episodes in 29 of 40 patients. The number of fibrillation

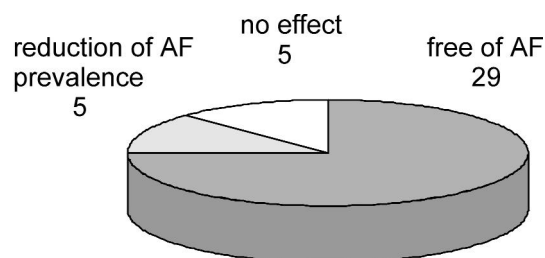


Figure 5. Clinical results.

episodes was significantly reduced. However, the long-term effectiveness of this preventative treatment cannot be assessed at present.

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