Atrio-Ventricular and Ventriculo-Atrial Conduction in Patients with Symptomatic Sinus Node Dysfunction

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Summary: Van Mechelen, R. et al.: Atrio-ventricular and ventriculo-atrial conduction in patients with symptomatic sinus node dysfunction. In 26 patients (pts) with symptomatic sinus node dysfunction (SND) electrophysiological studies were performed before pacemaker implantation. Patients were divided into two groups: Group I pts (18) with intact antegrade AV conduction (AVNW ≥ 130/min); Group II pts (8) with impaired antegrade AV conduction (AVNW < 130/min).

VA conduction was present in 20/26 pts (77%) of the total patient population. In 17/18 pts (94%) of Group I and in 3/8 pts (37%) of Group II VA conduction could be demonstrated (p < 0.01). In regard to a pacemaker syndrome, VVI pacing is therefore not the optimal pacemaker therapy in SND patients with VA conduction. However, the presently available alternatives (AAI, DVI, DDD pacemakers) have their imperfections too.

Introduction

Some patients with symptomatic sinus node dysfunction are not suitable candidates for permanent ventricular demand pacing (1–6). In these patients retrograde conduction to the atria during ventricular pacing results in atrial contraction against closed mitral and tricuspid valves. The symptoms of this so called “pacemaker syndrome” are in some patients so disabling that their demand unit has to be explanted (6).

Three years ago we started to perform electrophysiological studies (EPS) in these patients to demonstrate the presence or absence of retrograde conduction during ventricular pacing. In addition sino-atrial, AV nodal and infranodal conduction were evaluated, in order to select the most appropriate permanent pacing mode.

Methods

The decision to implant a pacemaker in patients with sinus node dysfunction was based upon symptoms of dizziness, dyspnea and/or syncope in relation with sinus bradycardia, sinus arrest, or sinoatrial block, documented by telemetry or by holter monitoring. Patients on drug therapy that might impair sino-atrial function were not included in this study.

Electrophysiological studies (EPS)

Following informed consent, EPS were performed in patients who met the selection criteria. The patients received no medication prior to the study. Catheters were placed for
both stimulation and recording of intracardiac electrograms at the right ventricular (RV) apex, tricuspid valve ring, and high right atrium (HRA). Patterns of AV and VA conduction, refractory periods, were determined using the extrastimulus technique during pacing from the HRA and RV apex at several basic cycle lengths of pacing (7). Incremental ventricular pacing was initiated at a rate 10–15% faster than the spontaneous sinus rate or AV junctional rate, up to a rate at which second degree VA block occurred. Pacing rate was increased in steps of 10 beats/min; each pacing period lasted one minute. Criteria for the acceptance of 1:1 VA conduction were the following:

1. atrial activation sequence from the low right atrium to the high right atrium;
2. identical ventricular and atrial cycle lengths at the ventricular paced rate;
3. constant VA conduction time at one and the same pacing rate.

Incremental atrial pacing was performed up to a rate at which second degree AV block occurred. A normal response to atrial pacing at incremental rates was defined as the development of AV nodal Wenckebach at a heart rate of least 130 beats/min (7). The development of second degree AV block at heart rates below 130/min suggested impaired AV conduction. Patients were divided into two categories: Group I patients with intact antegrade AV conduction; Group II patients with impaired antegrade AV conduction.

An estimate of cardiac automaticity was obtained by pacing the atrium at approximately 120/min for a minimum of 30 seconds. Upon cessation of pacing, the interval from the last pacing impulse to the first sinus node recovery beat was measured (SNR T). The corrected sinus node recovery time (CSNR T) was calculated by subtracting the basic sinus rhythm cycle length form the sinus node recovery time. If asystole persisted for 5 seconds without escape rhythm, atrial pacing was resumed.

Autonomic tone was assessed by carotid sinus stimulation and the intravenous administration of atropine, starting with 0.5 mg up to a maximum dose of 2 mg. A normal response to atropine was defined as an increase of sinus rate to greater than 90/min, and an increase over the spontaneous rate of 20–50%.

**Results**

The clinical and electrocardiographic data are listed in Table 1. Table 2 and 3 show the results of EPS. Sinus node recovery time ranged from 1400–5500 msec in Group I, and from 1100–6400 msec in Group II. Corrected sinus node recovery time ranged from 250–600 msec in Group I, and from 250–1100 msec in Group II. These differences were not statistically significant. The response to atropine was blunted in 9 patients of Group I, and in 4 patients of Group II. VA conduction was present in 20/26 or 77% of the total patient population. The incidence of VA conduction in patients of Group I was 17/18 (94%); the incidence of VA conduction of Group II patients was 3/8 (37%). The difference between the two groups was a statistically significant difference associated with a p-value of < 0.01 (8). Therefore this difference might be of clinical importance. The rate up to which VA conduction remained intact during incremental ventricular pacing varied considerably in both groups, and was not related to the atrial rate up to which 1:1 AV conduction was present. However, in the majority of patients in both groups antegrade AV conduction remained intact up to higher rates during incremental atrial pacing, than retrograde conduction did during incremental ventricular pacing.