

THE IMPACT OF PACING MODE AND THE ANATOMICAL POSITION OF PACING LEAD ON THE INCIDENCE OF HEART FAILURE¹

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Abstract: *In Romania in the last decade, pacing is playing an increasingly important role in the management of cardiac disease. If, at first, attention of the cardiologists and researchers was focusing on the electrical rather than functional effects of pacing, the fact that pacing the RV may initially improve cardiac function but may induce heart failure over time, has led to a change in direction.*

This study evaluates comparative the clinical outcome as well the incidence and predictors of heart failure in 38 patients with VVIR pacing, VDDR and DDDR pacing implanted in "Sf. Ioan" Hospital, Bucharest, over a period of 2 years. We also intended to evaluate the long-term effects of alternative right ventricular pacing sites on LVEF.

Key words: *right ventricular pacing; pacemaker syndrome; RV pacing sites; VVIR; VDDR; DDDR pacing modes*

1. Introduction

VVI pacing alone has shown a higher risk of sudden death compared with non-paced patients with a similar degree of heart failure [1]⁶. A retrospective study of long-term follow-up between VVI and DDD pacing showed that DDD enhances survival compared with VVI in patients with heart failure and AV block [2]. Nielsen et al. demonstrated that VVI pacing for sinus node disease was associated with the development of congestive heart failure over a 5 year follow-up period.

On the other side the expectation that the hemodynamic benefits of atrioventricular synchrony would lead to a reduction in cardiac mortality, a reduced risk of heart failure, and a better quality of life were not proven by all the clinical trials. The MOST study which followed for three years the cardiovascular mortality and morbidity in patients with DDDR cardio stimulation toward patients with VVIR cardio stimulation showed no statistical differences between the two groups. In exchange, concerning the heart failure episodes and the quality of life, the study proved the superiority of the DDDR stimulation.

During ventricular pacing the asynchronous ventricular activation may lead to abnormal regional myocardial blood flow and metabolic disturbances which can reduce systolic and diastolic left ventricular (LV) function [3,5]. These functional abnormalities seem to have enhanced effects over time. Some studies have shown that long-term right ventricular apical (RVA) pacing induces abnormal histologic changes and asymmetrical LV hypertrophy and thinning [6,7,9]

The choice of pacing site in the right ventricle is an other important issue. No recommendation could be made so far concerning the location of the right ventricular pacing site.

The right ventricular apex, does not seem to lead to best haemodynamic results, although it is easy accessible and best for electrode stability. [10,11,12]. While acute haemodynamic studies find that outflow tract or dual-site pacing are best for haemodynamic reasons, most of the controlled studies with permanent pacing found no significant difference to right ventricular apical pacing

In a previous clinical study, on 547 patients we have shown that the relation between VVIR pacing and the development of the pacemaker syndrome is likely to be complex. Age, comorbidity and haemodynamic status before pacing are factors that influence the appearance of the pacemaker syndrome. The patient group over 85 years had a higher incidence of worsening heart failure than the other age groups. The patients with EF > 40% before pacing had a better outcome than those with impaired left ventricular systolic function.

The data of our previous study has shown that VVIR pacing may not induce directly heart failure but may increase the risk of developing atrial fibrillation, an important precipitant of heart failure.

In animal studies, pacing at the right ventricular outflow tract (RVOT) has been shown to decrease the asynchrony of activation so that it seems to ameliorate the reduction in LV function and prevent the wall motion abnormalities and the impair of the LV function.

2. Methods

The study, included 38 patients, men and women, which needed permanent pacing, hospitalized in the Internal Medicine And Cardiology Department of "Sfantul Ioan" Hospital, over a two and a half years period, between January 2007 and June 2009. Patients who refused to sign the written consent and those with serious (severe) coagulation disorders, chronic patients with dialysis or with cancer in terminal stages were excluded.

The patients were admitted for pacemaker implantation due to following diseases:

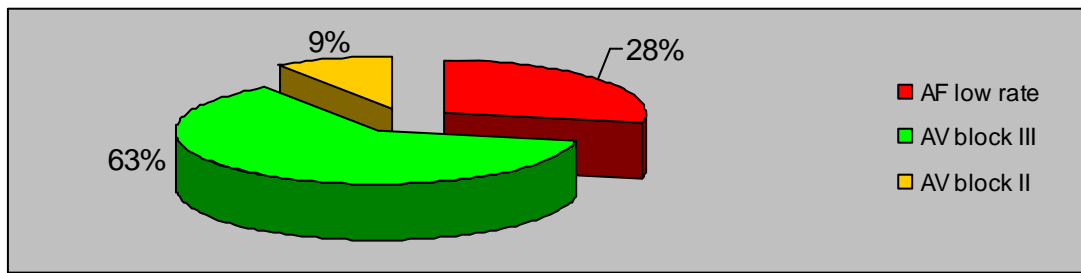


Figure 1. Patients' history

The follow up after the implant was planned at 1 month, 3 month, 12 month and 48 month.

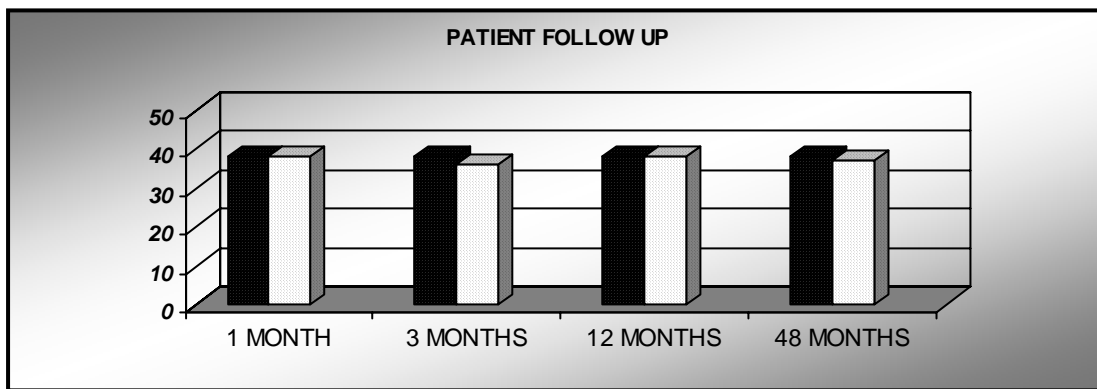


Figure 2. Patient follow up

We selected the pacing type, following a simple algorithm that regarded the aethiology and the anatomical and functional status of the atria (see figure 3).

All patients underwent implantation of a single chamber or a dual-chamber pacemaker using one active fixation atrial lead and one active fixation bipolar ventricular lead.

The ventricular pacing lead was inserted into the RV through subclavian vein puncture. Under fluoroscopic guidance, the ventricular lead was positioned in the right ventricular apex or in the RVOT (by advancing the lead through the tricuspid valve and then withdrawing it and positioning the tip against the interventricular septum and verifying the position using multiple fluoroscopic views).

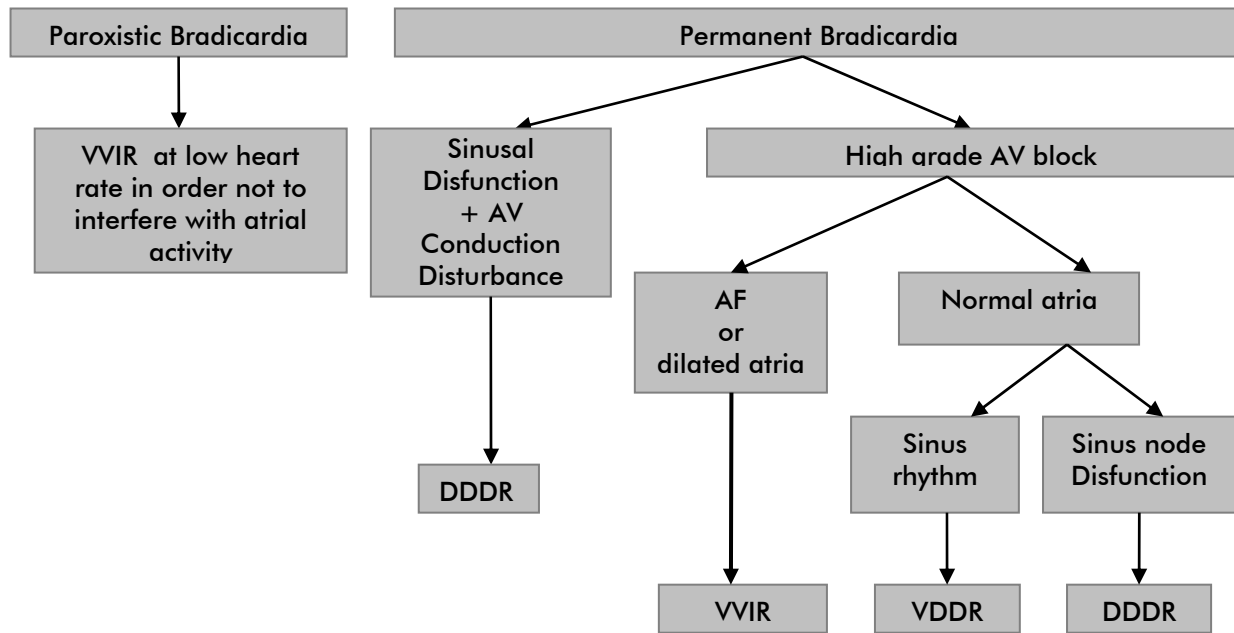


Figure 3. Pacing's type selection algorithm

The ventricular pacing leads were positioned at a stable position to obtain a satisfactory pacing threshold value (mean 1.1 ± 0.2 V) at a pulse width of 0.5 ms and R-wave sensing value (mean 12.3 ± 0.1 mV). Atrial leads were positioned to the right atrial lateral wall. After implantation, optimization of the AV delay was performed by using pulsed Doppler echocardiography of the transmitral blood flow.

Patients were evaluated before implant by a complete clinical examination. Cardiac risk factors, cardiac and associated non cardiac pathology were identified and concomitant medication was recorded.

For a proper evaluation of heart failure a special attention was given to include the patients in different NYHA classes according with their symptoms. The symptom screening, prior to the clinical examination and echocardiogram was made by the physician by asking the same questions in order to evaluate symptoms of heart failure.

The real effort capacity was estimated by standard 6 minutes walking test.

Before and after the implant, the end systolic and end diastolic volumes of the left ventricle and the ejection fraction (Simpson method in two and four chambers incidence) were measured.

Echocardiographic measurements were made in M mode and two-dimensional echocardiography (2DE). Measurements of left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), and EF were obtained using the software installed on the ultrasound equipment, with LVEDV measurements at the time of mitral valve closure and LVESV measured on the image with the smallest LV cavity. The papillary muscles were excluded from the volumes. Biplane Simpson's rule volumes were obtained from the apical four- and two-chamber views.

M mode parameters were measured according to the American Society of Cardiology.

The severity of MR was appreciated from Doppler color-flow in the conventional parasternal long-axis and apical four-chamber images. Mitral regurgitation was

characterized as: *mild* (jet area/left atrial area <10%), *moderate* (jet area/left atrial area 10% to 20%), *moderately severe* (jet area/left atrial area 20% to 45%), and *severe* (jet area/left atrial area >45%).

3. Results

As the result of the pre-procedural evaluation, the patients were included into three groups with different pacing mode:

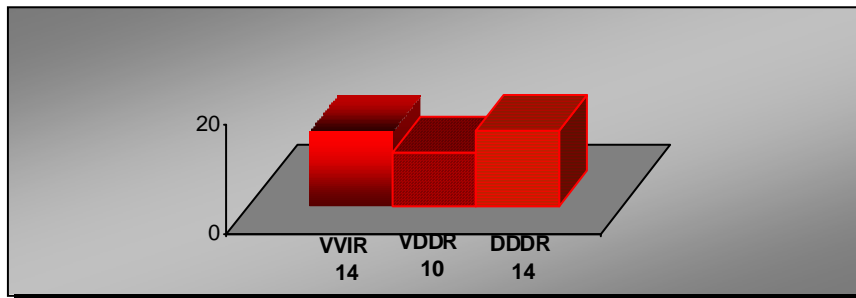


Figure 4. The distribution of the patients by the pacing mode

The incidence of heart failure at screening, at 3 months and at 12 months was the following:

Table 1. The incidence of heart failure

Pacing mode	Number of patients	Number with heart failure Screening	Number with Heart failure At disclosure	Number with heart failure 12 month
VVIR	14	2	1	5
VDDR	10	3	0	2
DDDR	14	3	0	1
Total	38	8	1	8

At pre-discharge echocardiography, there was no significant difference from baseline values in LVEF, cardiac output, as measured by Doppler echocardiography, or transmitral A- and E-wave ratio in all the three groups.

Significant changes between the three pacing modes were found at the 3 months and 12 months follow up, as shown in the table:

Table 2. Changes found at follow up checks

	VVIR	VDDR	DDDR	p value
NYHA class				
▪ Baseline	2.8 ± 0.3	2.5 ± 0.2	1.3 ± 0.2	< 0.01
▪ 12 months	3.2 ± 0.3	2.4 ± 0.3	3.1 ± 0.3	< 0.05
QRS (ms)				
▪ Baseline	136 ± 22	130 ± 26	129 ± 27	< 0.01
▪ 12 months	147 ± 26	132 ± 27	125 ± 18	< 0.01
LVEF (%)				
▪ Baseline	35 ± 6	31 ± 7	33 ± 6	< 0.01
▪ 12 months	38 ± 5	30 ± 6	30 ± 7	< 0.05
Severe MR				
▪ Baseline	3	2	3	NS
▪ 12 months	4	2	2	NS

Mitral regurgitation improved by at least one grade in 8 of 14 (57,14%) patients with severe regurgitation.

The mean QRS duration was significantly longer in VVIR paced patients than in VDDR or DDDR pacing.

We subdivided the patients in two different groups, those with right ventricular apex pacing (RVA) and those with right ventricular outflow tract (RVOT) pacing.

The differences between the 2 subgroups are seen in the following table:

Table 3. Subgroups' particularities

	Right ventricular apex pacing	RVOT/RS pacing	p Value
Baseline echocardiography			
LVEF	54 ± 11%	55 ± 12%	0.72
E/A ratio	0.9 ± 0.3	1.1 ± 0.2	0.30
Optimal atrioventricular interval(ms)	140 ± 39	146 ± 36	0.47
Discharge echocardiography			
LVEF	55 ± 2%	58 ± 11%	0.36
E/A ratio	1.0 ± 0.3%	1.1 ± 0.3%	0.25
Optimal atrioventricular interval (ms)	141 ± 38	145 ± 38	0.49
6 months echocardiography			
LVEF	47 ± 3%	56 ± 12%	0.38
E/A ratio	0.9 ± 0.3%	1.0 ± 0.3%	0.28
Optimal atrioventricular interval (ms)	140 ± 38	146 ± 38	0.49

4. Discussion

Although VVIR pacing is effective in preventing symptomatic bradyarrhythmias, it has been demonstrated to be associated with a significant negative inotropic effect and with an increased rate of congestive heart failure. This pacing modus has led also to a greater increase in the grade of mitral regurgitation than the bicameral pacing modes.

RV apical pacing frequently produces an LBBB pattern with alteration in myocardium depolarization and contraction which results in a reduction of the LVEF, an prolongation of conduction intervals. Our study reveals that these changes are more important over longer periods of time than immediately after pacing.

RVOT or RS pacing can improve cardiac performance, over that obtained with RV apical pacing, despite the presence of AV synchrony. This improvement appears in every pacing mode, but is more efficient in dual chamber pacing.

5. Conclusions

Even if for over 4 decades of cardiac pacing, the right ventricular apex (RVA) has been the main site for right ventricular lead placement. during RVA pacing, larger QRS duration, impairment of LV diastolic function with significant reduction in global LV function is present.

Pacing at the RVOT is associated with more synchronous ventricular activation with a narrower QRS duration and with lower incidence of deterioration in global LV systolic and diastolic function.

Over long-term follow-up the clinical benefit seems to be greater.

RVOT pacing for routine pacemaker implantation might be the answer for preventing and treating congestive heart failure in paced patients.

Further studies may be necessary in order to compare the benefits of RVOT pacing compared with classical RVA pacing in patients with risk for heart failure.

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