Acute Effects of Dual-chamber Pacing on the Left Ventricular Systolic Function and Relaxation in Patients with Advanced AV Block and Sick Sinus Syndrome

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Background: Abnormal activation of the ventricles via right ventricular apical pacing deteriorates cardiac function, which may explain the increased mortality of patients with congestive heart failure receiving permanent pacemakers. We hypothesized that pacing at alternative sites may cause less detrimental effects on the cardiac function.

Methods: Five symptomatic patients with either advanced AV block (n = 4) or sick sinus syndrome with normal left ventricular (LV) function (n = 1) were studied. During cardiac catheterization, LV pressure was recorded with a high-fidelity catheter-tipped transducer. Baseline rhythms were sinus rhythm or VVI pacing. Sequential VDD pacing with variable AV intervals was performed at the right ventricular apex (RVA), right ventricular septum (RVS), right ventricular outflow tract (RVOT) and coronary sinus (CS). LV systolic function was assessed by calculating dP/dt_{max} and LV diastolic function was indexed by calculating the exponential isovolumic relaxation constant (Tau). Percentage changes (mean ± SE) from baseline to pacing were measured.

Results: RVA pacing reduced dP/dt_{max} (–0.8 ± 8.4%) and prolonged Tau (7.0 ± 5.6%); RVS pacing enhanced dP/dt_{max} (20.7 ± 15.3%) and shortened Tau (–10.4 ± 9%); RVOT pacing reduced dP/dt_{max} (–8.0 ± 20.0%) and shortened Tau (–6.0 ± 12.2%); CS pacing reduced dP/dt_{max} (–11.7 ± 13.0%) and prolonged Tau (10.5 ± 11.9%). Our results demonstrated that different pacing sites have different effects on LV contractility and relaxation in patients with normal LV function.

Conclusion: Since pacing at the RVS preferably increased LV dP/dt_{max} and shortened Tau, it may be a better alternative than the RVA. [*J Chin Med Assoc* 2006;69(1):32–36]

Key Words: left ventricular function, left ventricular relaxation, pacing therapy

Introduction

The right ventricular apex (RVA) has been the preferred site for ventricular stimulation since the introduction of permanent transvenous cardiac pacing. It is well known that electrical depolarization of the left ventricular (LV) myocardium via the normal conducting system is rapid and synchronous. In contrast, apical stimulation alters the timing and sequence of the LV electrical depolarization and of mechanical contraction.[1] Beats produced by right ventricular apical pacing eject less blood compared with beats produced by right atrial, LV septal, or LV apical pacing.[1] Right ventricular apical pacing acutely lowers peak LV systolic blood pressure and reduces cardiac output, maximal rate of LV pressure (LVP) increase (dP/dt_{max}) and decrease (dP/dt_{min}) and end-systolic elastance, and compromises coronary blood

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flow.[2–4] Chronic right ventricular apical pacing can produce myofibrillar disarray, and regional changes in tissue perfusion and heterogeneity between perfusion and sympathetic innervation.[5,6] Both DDD and VVI pacing significantly reduce peak systolic blood pressure, dP/dt_{max}, dP/dt_{min}, stroke volume, ejection fraction and cardiac output.[7] In contrast to the RVA, pacing at the right ventricular proximal septum may produce a favorable effect on LV performance.[3] From the findings of the MOde Section Trial (MOST), ventricular pacing induced ventricular dysfunction even with preserved AV synchrony to increase the risk of heart failure and atrial fibrillation in sinus node disease with normal baseline QRS duration.[8] Thus, we hypothesized that pacing at alternative sites other than the RVA may cause less detrimental effects on the LV performance. The purpose of this communication is to describe the preliminary results of the pacing effects on the LV systolic function and relaxation.

Methods

Patients

Five patients with symptomatic bradycardia, who were indicated for diagnostic catheterization and placement of permanent pacemaker, were enrolled. Four patients had advanced AV block and one patient had sick sinus syndrome. All patients had normal LV function (mean ejection fraction = 69.8 ± 7.9%) and normal coronary arteries as revealed by coronary angiography and left ventriculography during cardiac catheterization. Table 1 provides the clinical characteristics of the study group.

Catheterization protocol

After routine diagnostic catheterization, including coronary angiography, left ventriculography and electrophysiologic study to assess sinus node function or AV block levels, a 2F catheter incorporated with a catheter-tipped transducer was advanced through a pig-tailed catheter to record high-fidelity LVP during the pacing study. A quadrapolar electrode catheter was positioned across the tricuspid annulus to record the His bundle electrocardiogram as well as to serve as pacing at the right ventricular septum (RVS). A second quadrapolar electrode was positioned at the RVA or right ventricular out-flow tract (RVOT), with a third catheter in the right atrium for atrial sensing and/or pacing. For pacing at the coronary sinus (CS), an electrode catheter was introduced via the right internal jugular vein and advanced into a lateral marginal vein. Once all catheters were positioned, pacing in the VDD mode was initiated at either RVA, RVS, RVOT or CS. Because of technical problems, not every site was studied in every patient. At each site, 3–4 different AV intervals were studied. Each AV interval differed by 40 ms, with the longest delay being 20–70 ms shorter than the intrinsic AV interval, assuring inclusion of an interval near 120 ms in each patient. Data were recorded for 3 minutes in steady state, with the order of pacing sites and AV intervals at each site fully randomized. Data were re-measured in normal sinus rhythm (NSR) before and after each pacing intervention. These data were used to determine the normal variance in various hemodynamic parameters. Changes in hemodynamics for each pacing condition were determined relative to the immediately preceding NSR data. In patients who were dependent on pacing due to profound bradycardia, VVI at RVA served as the baseline for comparison. LVP was used to derive peak and diastolic pressure (pressure at time when dP/dt exceeded 10% of maximal value), maximal and minimal pressure derivatives (dP/dt_{max} and dP/dt_{min}, derived digitally from a running 5-point slope), and time constant of pressure relaxation (monoexponential fit employing a non-zero decay asymptote). LV systolic function was assessed by dP/dt_{max} and LV diastolic function was indexed by the relaxation time constant.

Statistical analysis

Because of the very limited number of cases studied, only descriptive statistics are presented. Data are expressed as mean ± SE.

Table 1. Clinical characteristics of the patients

Patients	Age (yr)	Sex	LVEF (%)	Diagnosis
Case 1	73	M	82	2° AV block + CAVB, infra-His block
Case 2	71	M	69	Trifascicular block + SSS
Case 3	75	M	65	CAVB, infra-His block
Case 4	77	M	61	2° AV block, supra-His block
Case 5	72	M	72	2° AV block, infra-His block

CAVB = complete atrioventricular block; LVEF = left ventricular ejection fraction; SSS = sick sinus syndrome; 2° AV block = secondary atrioventricular block.

Table 2. Summary of hemodynamic results at baseline and after pacing

	SBP (mmHg)	LVEDP (mmHg)	dP/dt_{max} (mmHg/s)	Tau (ms)
RVA (n = 5)				
Baseline	165 ± 15	10 ± 2	1,606 ± 335	66 ± 10
Pacing	163 ± 19	10 ± 1	1,580 ± 336	72 ± 10
RVS (n = 5)				
Baseline	157 ± 10	10 ± 2	1,511 ± 194	72 ± 19
Pacing	164 ± 19	9 ± 1	1,724 ± 208	59 ± 6
CS (n = 3)				
Baseline	158 ± 7	10 ± 1	1,588 ± 154	59 ± 11
Pacing	159 ± 10	10 ± 2	1,484 ± 253	70 ± 18
RVOT (n = 2)				
Baseline	159 ± 1	9 ± 1	1,597 ± 237	69 ± 24
Pacing	154 ± 22	11 ± 1	1,649 ± 199	63 ± 11

CS = coronary sinus; dP/dt_{max} = maximal rate of LV pressure increase; LVEDP = left ventricular end-diastolic pressure; RVA = right ventricular apex; RVOT = right ventricular outflow track; RVS = right ventricular septum; SBP = peak left ventricular systolic pressure; Tau = constant of left ventricular relaxation.

Results

Table 2 shows a summary of hemodynamic results at baseline and after pacing. As compared with baseline, dual chamber pacing at RVA decreased dP/dt_{max} and prolonged Tau. A typical example is shown in Figure 1A. Overall, peak systolic blood pressure reduced by 0.9 ± 3.8%, LV end-diastolic pressure by 4.0 ± 6.8%, dP/dt_{max} by 0.8 ± 8 .4%, and Tau prolonged by 7.0 ± 5.6% with RVA pacing. As compared with baseline, dual chamber pacing at RVS increased dP/dt_{max} and shortened Tau. A typical example is shown in Figure 1B. Overall, peak systolic blood pressure increased by 2.0 ± 4.9%, dP/dt_{max} increased by 20.7 ± 15.3%, LV end-diastolic blood pressure decreased by 17.9 ± 4.6%, and Tau shortened by 10.4 ± 9.0% with RVS pacing.

As compared with baseline, dual chamber pacing at CS decreased dP/dt_{max} and prolonged Tau. A typical example is shown in Figure 1C. Overall, peak systolic blood pressure increased by 1.2 ± 2.1%, LV end-diastolic pressure increased by 1.1 ± 17.1%, dP/dt_{max} decreased by 11.7 ± 13.0%, and Tau prolonged by 10.5 ± 11.9% with CS pacing.

As compared with baseline, dual chamber pacing at RVOT decreased dP/dt_{max} but shortened Tau. A typical example is shown in Figure 1D. Overall, peak systolic blood pressure reduced by 4.6 ± 10.3%, LV end-diastolic pressure increased by 17.0 ± 0.2%, dP/dt_{max} decreased by 8.0 ± 20.0%, and Tau shortened by 6.0 ± 12.2% with RVOT pacing.

The percentage changes of hemodynamic variables from baseline for various pacing sites are summarized in Figure 2.

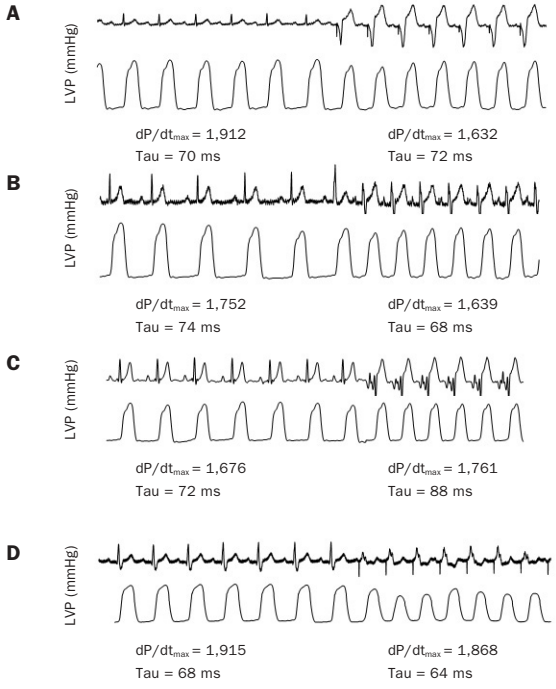

Figure 1. (A) Right ventricular apical (RVA) pacing and LVP recording. (B) Right ventricular septum (RVS) pacing and LVP recording. (C) Coronary sinus (CS) pacing and LVP recording. (D) Right ventricular outflow tract (RVOT) pacing and LVP recording.

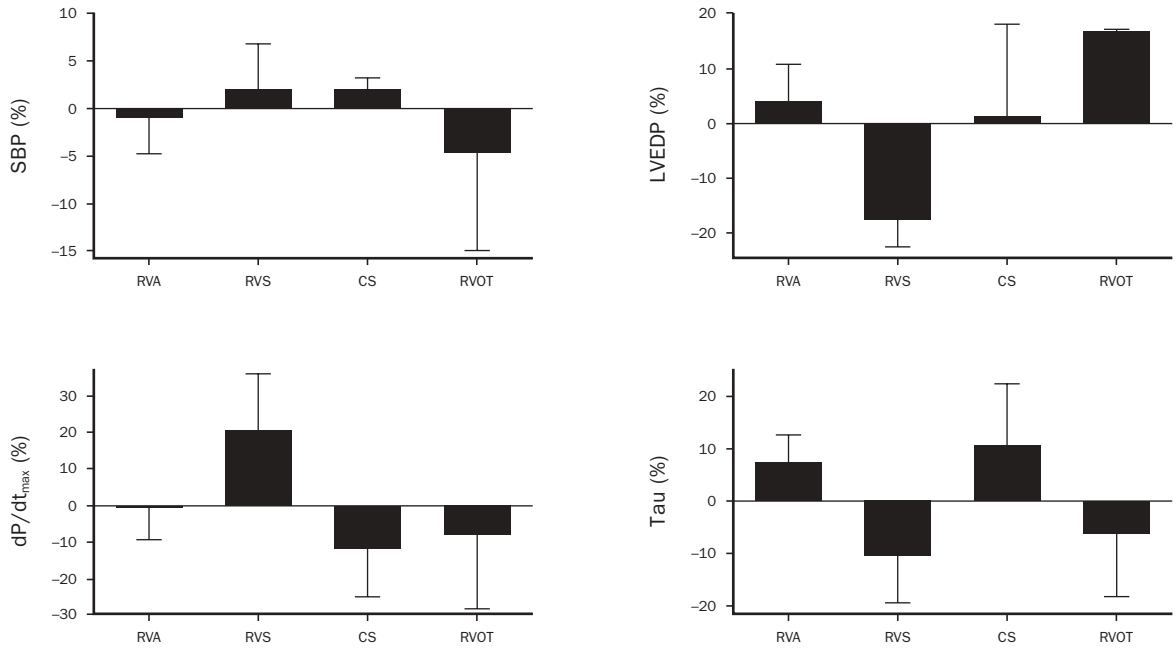

Figure 2. Summary of percentage changes of hemodynamic variables from baseline for various pacing sites. CS = coronary sinus; dP/dt_{max} = maximal rate of LV pressure increase; LVEDP = left ventricular end-diastolic pressure; RVA = right ventricular apex; RVOT = right ventricular outflow tract; RVS = right ventricular septum; SBP = peak left ventricular systolic pressure; Tau = constant of left ventricular relaxation.

Discussion

The preliminary results obtained were in accord with our hypothesis: pacing at alternative sites other than the RVA, e.g. the RVS, might cause less detrimental effects on the LV performance.

RVA is the conventional pacing site for permanent pacemaker implantation because it is easily approached. We used His bundle pacing as RVS pacing because it allows simple catheter placement, instead of right proximal septal pacing that was previously used. In the clinical setting, His bundle is more easily approached but the fixing of the pacing lead is difficult. The technical difficulties in the positioning of the electrode under the septal leaflet of the tricuspid valve and high pacing thresholds are problems that need to be studied further. The CS was paced at the distal CS and CS pacing was used to simulate LV pacing. This position was difficult to approach due to anatomic variation and small vessel size, so only 2 patients were paced at the CS. RVOT pacing could be easily performed without difficulty in most patients.

A normal ventricular activation pattern seems to be of crucial importance for optimal systolic and diastolic function.[2,7] Both positive and negative dP/dt significantly decreased during AV synchronous pacing as compared with atrial pacing. Right ventricular stimulation produced an abnormal pattern of ventricular contraction, to depress the LV function,[7] and a significant deterioration in regional LV systolic performance was noted. The observed differences were related to changes in afterload caused by asynchronous ventricular contraction.[2] In contrast, preliminary studies of proximal septal pacing in patients are promising, showing hemodynamic improvement.[3] In agreement with the results of these preliminary studies, our preliminary results consistently showed that RVS pacing results in the best systolic function among the different pacing sites.

Some studies reported that an abnormal ventricular activation also causes abnormal relaxation, reflecting impaired diastolic function.[9,10] We used Tau as an index of diastolic function. Similar to the systolic function, we observed significantly shorter Tau during RVS pacing than other sites. Shorter Tau means better diastolic function. Cardiac function may be preserved by pacing at RVS instead of the traditional site (i.e. the RVA). Whether this beneficial effect persists long enough in patients with advanced AV block remains to be established.

In conclusion, our results demonstrated that different pacing sites had different effects on LV contractility and relaxation in patients with normal LV function. Based on our preliminary results, it appears that pacing at the RVS preferably increases LV dP/dt_{max} and shortens Tau, and, thus, may be a better alternative to conventional pacing at the RVA. The relative beneficial effects from pacing at the RVS may be due to the relatively preserved synchronous contraction between the left and right ventricles. Further studies on more patients are required to confirm this hypothesis.

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