Value of the Paced QRS Duration

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ABSTRACT

Background: The value for paced QRS duration (pQRSd) to detect left ventricular (LV) dysfunction in right ventricular apex (RVA)−paced patients has not been evaluated.

Methods and Results: A total of 272 RVA-paced patients, including 99 with LV systolic dysfunction (LVSD) and 173 without LVSD, were enrolled in this study. The pQRSd, echocardiographic variables, and plasma N-terminal pro-brain natriuretic peptide (NT-proBNP) levels were measured. Relationships between pQRSd and echocardiographic variables, NT-proBNP levels, and New York Heart Association (NYHA) functional classification were analyzed. pQRSd was correlated with LV end-diastolic and end-systolic dimensions ($\beta = 1.59$ and 1.54, respectively; all $P < .001$), NT-proBNP levels ($\beta = 12.98$, $P < .001$) and LV ejection fraction ($\beta = -109.25$, $P < .001$). There was a stepwise increase in pQRSd with increasing NYHA Class (all $P < .001$). The pQRSd cutoff value of 200 ms, derived from the receiver operator characteristic curve, had sensitivity of 71.72% and specificity of 86.71% to detect LVSD. pQRSd ≥ 240 ms gave a positive predictive value of 100%, whereas <180 ms excluded >97.3% of patients with LVSD.

Conclusions: In RVA-paced patients, pQRSd is correlated with left ventricular structures and function and pQRSd of 200 ms is a satisfactory cutoff value in terms of sensitivity and specificity for detecting LVSD. (*J Cardiac Fail 2009;15:347−352*)

Key Words: Pacing, systolic dysfunction, left ventricular ejection fraction, QRS duration, heart failure.

The QRS duration (QRSd) represents the time required for the 2 ventricular chambers to be activated. It has been used to select patients with congestive heart failure (CHF) for cardiac resynchronization therapy (CRT). QRSd ≥120 ms is one of the indications for CRT in patients with CHF recommended by American College of Cardiology/American Heart Association/Heart Rhythm Society (HRS) 2008 guidelines.[1] In patients with CHF, there is a negative correlation between QRSd and left ventricular (LV) ejection fraction (LVEF)[2] and prolonged QRSd is associated with worse prognosis.[2−5] The Framingham studies showed that QRSd was positively correlated with LV dimension and LV mass, and prolonged baseline QRSd was a risk factor of HF over a mean follow-up of 12.7 years, even after adjusting for LV mass in subjects without HF.[6,7] In patients with pacemaker implantation, prolonged baseline paced QRS duration (pQRSd) is associated higher risk for HF.[8−12] The QRSd was also used to detect LV systolic dysfunction (LVSD).[13,14] Das et al found that prolonged QRSd (QRSd ≥170 ms) and left axis deviation in the presence of left bundle branch block was a marker for poor left ventricular systolic function.[15] Previous studies had showed that pQRSd could be a useful indicator for impaired LV function in patients with right ventricular pacing.[8−12] However, the relationship between pQRSd and ventricular structures and function in pacing patients was only partially investigated in a few small studies.[9,10] The value for pQRSd to detect cardiac dysfunction has not yet been evaluated. Accordingly, in this study, we investigated the value of pQRSd for detecting LVSD and observed the association between pQRSd and LV structures and function parameters in a large right ventricular apex (RVA)−paced patient cohort.

Methods

Patient Selection

Consecutive patients referred to our department for routine pacemaker interrogation between December 2006 and May 2008 were enrolled in this case control study, including 99 patients

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with LVSD (LVEF <0.55, case group) and 173 patients without LVSD (LVEF ≥0.55, control group). The inclusion criteria were as follows: (1) the ventricular pacemaker lead should be placed at the RVA and the pacing duration should be more than 1 year; (2) paced QRS complex could be seen in each lead in standard electrocardiography (ECG) for correct pQRSd measurement; (3) patients with VVI(R) pacemakers or patients implanted with DDD(R) pacemakers having high or third-degree atrioventricular block. Patients younger than 18 years of age or using class I antiarrhythmic drugs or with severe hyperkalemia or hypokalemia (<3.0 or >6.5 mmol/L) were excluded. The study conformed to the principles outlined in the Declaration of Helsinki. The local ethics committee approved the study and all patients provided written informed consent to participate in the study.

Measurement of pQRSd

In patients with DDD(R) pacemaker implantation, the atrioventricular delay was at factory setting (130-170 ms) during the ECG examination. Standard 12-lead ECG was acquired at a paper speed of 50 mm/second and a scale of 10 mm/mV. The pQRSd was defined as the length of time from the beginning of the pacing spike to the end of the QRS complex. It was measured from the ECG using the widest QRS complex from 12 leads by 1 experienced observer who was blinded to the clinic and echocardiography data. To assess the intraobserver reproducibility of pQRSd measurement, 60 randomly chosen ECGs were measured twice. The intraobserver variability was 4.69 ms (2.55%).

Echocardiographic Examination and N-terminal Pro-Brain Natriuretic Peptide Assays

Echocardiography was performed with GE Vivid 7 (GE Vingmed, Horton, Norway) using 1.7-3.4 MZ probe. LV end-systolic diameter (LVDS), LV end-diastolic diameter (LVDD), interventricular septum thickness (IVST) and LV posterior wall thickness (LVPWT) were obtained from parasternal long-axis view of LV. LVEF was measured with Simpson's method. LVSD was defined as LVEF <0.55 in our echocardiography lab. EDTA whole blood sample (5 mL) was obtained in 163 patients (59.93%). N-terminal pro-brain natriuretic peptide (NT-proBNP) levels were determined using a commercially available enzyme linked immunoassay (Roche Diagnostics, Basel, Switzerland) by an automatic electrochemical analyzer (Elecsys 2010, Roche) and reported in ng/L.[16] The pQRSd, echocardiographic variables, NT-proBNP levels and New York Heart Association (NYHA) class were simultaneously determined within a time interval of 72 hours.

Statistical Analysis

Continuous variable and categorical variables were presented as mean value ± SD and%, respectively. A 95% confidence interval (CI) was reported with the sensitivity, specificity, and predictive value. The logarithm of NT-proBNP level was presented as log (NT-proBNP). The data were compared using *t*-test or 2-sample Wilcoxon rank-sum (Mann-Whitney) test or chi-square test. A 1-way analysis of variance followed by Bonferroni's post hoc test was performed to evaluate the differences of pQRSd among patients with different NYHA Class ratings. Relationships between pQRSd and other variables were assessed by linear regression model. Then, stepwise multivariables linear regression was performed to study these relationships, adjusting for potential confounders. The dependent valuable was pQRSd and independent valuables used for regression analysis included LVEF, NYHA Class, LVPWT, IVST, LVDD, LVDS, and ischemic heart disease. Because the effects of pacing mode,[17] atrial fibrillation,[18] or age[19] on NT-proBNP level could be independent of cardiac function, they were chosen as covariates in the multivariate model to study the relationship between NT-proBNP level and pQRSd. Criteria of $P < .10$ for entry and a $P \geq .05$ for removal were imposed in this procedure. Receiver operator characteristic curve was used to examine the performance of pQRSd to detect LVSD. The curve represented a plot of sensitivity vs. 1-specificity. The area under the curve was derived from the receiver operator characteristic curve. A statistically derived value, based on the Youden index,[20] maximizing the sum of the sensitivity and specificity, was used to define the optimal cutoff value for pQRSd to detect LVSD. $P < .05$ was considered to represent statistical significance. Statistical analyses were performed using the SPSS 13.0 software package (SPSS, Inc, Chicago, IL).

Results

Patient Characteristics

A total of 272 patients (171 men, mean age: 69.9 ± 12.7 years) were enrolled in this study. Fifty-nine (21.69%) and 213 (78.31%) patients were implanted with DDD(R) and VVI(R) pacemakers, respectively. The underlying cardiovascular diseases were as follows: 34 (12.5%) ischemic heart disease, including 14 patients with myocardial infarction, 24 (8.82%) dilated cardiomyopathy, 13 (4.78%) hypertrophic cardiomyopathy, 3 (1.10%) viral myocarditis, 26 (9.56%) rheumatic or valvular heart disease, 124 (45.59%) hypertension, 22 (14.15%) diabetes, 4 (1.47%) congenital heart disease, and 66 (24.26%) without defined cardiovascular diseases. The underlying bradyarrhythmias were as follows: 87 (27.36%) sick sinus syndrome, 125 (56.60%) atrioventricular block, and 80 (16.38%) atrial fibrillation with long R-R interval. The patients' clinical characteristics in case group and control group are shown in Table 1. Patients in case group were significantly younger than patients in control group ($P < .05$). There were significantly more male, higher NYHA Class, lower LVEF, larger LVDD and LVDS, and thicker LVPWT patients in the case group compared with those in the control group (all $P < .05$). pQRSd and log (NT-proBNP) were significantly increased in the case group ($P < .001$) and number of patients with ≥200 ms was significantly higher in case group compared with the control group ($P < .001$). There was no significant difference of pacing duration between the 2 groups (3.94 ± 3.88 vs. 3.28 ± 3.16 years, $P > .05$). LVEF in the case group and control group before pacemaker implantation was 0.52 ± 0.07 and 0.70 ± 0.06 ($P < .05$), respectively. The baseline pQRSd (determined several days after pacemaker implantation) in the case group and control group was 198.56 ± 20.14 ms and 176.02 ± 17.39 ms ($P < .01$), respectively. Sixty-seven patients (67.68%) in the case group had LVSD before RVA pacing.

Relationships between pQRSd and Left Ventricular Structures and Function

pQRSd was positively correlated with LVDD ($\beta = 1.59$, $P < .001$), LVDS ($\beta = 1.54$, $P < 0.001$), IVST ($\beta = 3.10$,