VIA MEDICA

ORIGINAL ARTICLE

The influence of atrioventricular and atrioventricular nodal re-entrant tachycardia on left ventricular systolic and diastolic function

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Abstract

Background: *Incessant tachycardia may result in left ventricular (LV) systolic dysfunction. Little is known about the influence of atrioventricular tachycardia (AVRT) and atrioventricular nodal re-entrant tachycardia (AVNRT) on LV systolic and diastolic function. The aim of the study was to assess LV systolic and diastolic function and factors affecting LV systolic and diastolic performance in patients with AVRT and AVNRT.*

Material: *The study group included 90 patients (40 male, 50 female, mean age 40 ± 12 years) with symptomatic narrow-QRS tachycardia. After an invasive electrophysiology study (EPS) had been performed, these were divided into two groups: one (AVNRT) of 25 patients with AVNRT and the other (WPW) of 65 patients with AVRT. The control group consisted of 50 healthy volunteers (23 male, 27 female, mean age 39 ± 13 years). In all three groups transthoracic echocardiography was performed in order to assess LV systolic and diastolic function.*

Results: *LV fractional shortening (FS) (WPW: 32 ± 6%, AVNRT: 36 ± 4%, p < 0.005) and ejection fraction (EF) (WPW: 54 ± 7%, AVNRT: 55 ± 5%, p < 0.001) were significantly decreased in the study groups compared to the control group (FS: 40 ± 5%, EF: 62 ± 4%). FS was significantly increased in the AVNRT compared to the WPW patients (p < 0.005). LV diastolic dysfunction was found in 20 (80%) of the AVNRT and 55 (83%) of the WPW group and in none of the patients from the control group. A positive correlation was found between diastolic dysfunction and the scale of recurrence of AVRT (R = 0.67, p < 0.05) and AVNRT (R = 0.52, p < 0.05).*

Conclusions: *AVNRT and AVRT may lead to development of LV systolic and diastolic dysfunction. LV diastolic dysfunction is related to the frequency of AVRT or AVNRT recurrence.* (Cardiol J 2007; 14: 160–166)

Key words: AVRT, AVNRT, systolic dysfunction, diastolic dysfunction

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Introduction

Incessant tachycardia may lead to the development of dilated cardiomyopathy with enlargement and deterioration of left ventricular (LV) systolic function [1–4]. Although the influence of atrioventricular tachycardia (AVRT) and atrioventricular nodal re-entrant tachycardia (AVNRT) on LV systolic and diastolic function has not been clearly defined, it is known that low-frequency long-lasting AVRT with slow retrograde conduction in some patients with Wolff-Parkinson-White syndrome (WPW) may produce tachyarrhythmic cardiomyopathy [5, 6]. The aim of the study was to assess LV systolic and diastolic function and factors affecting LV systolic and diastolic performance in patients with AVRT and AVNRT.

Methods

In this study we conducted a prospective analysis of 90 patients (40 male, 50 female, mean age 40 ± 12 years) selected from 158 subjects with symptomatic narrow-QRS tachycardia resistant to pharmacological treatment. These patients underwent transthoracic echocardiography (TTE) followed by invasive electrophysiology study (EPS) and radiofrequency catheter ablation (RFCA) in our division between March 1998 and December 2000. The control group consisted of 50 healthy volunteers (23 male, 27 female, mean age 39 ± 13 years) selected from 79 subjects. The study population was divided into two groups: the first, AVNRT, consisted of patients with atrioventricular node dissociation and AVNRT induced during electrophysiology study (EPS) and the second, WPW, of patients with pre-excitation syndrome and AVRT stimulated during EPS. Inclusion criteria for the study groups were the presence of an accessory pathway (AP) or atrioventricular node dissociation and AVRT or AVNRT induction during EPS. Patients were excluded from the study group if more than one AP was present (7 patients) or AP and atrioventricular node dissociation co-existed in the same subject (2 patients). The presence of concomitant diseases, such as coronary artery disease (2 patients), myocarditis (1 patient), hypertension (22 patients), hypothyreosis (2 patients) or valve dysfunction (29 patients), conduction disturbances other than WPW (LBBB — 1 patient), obesity (BMI ≥ 30 — 7 patients), drug intake (5 patients) or a poor TTE image (14 patients) were also defined as exclusion criteria in both study and control groups.

Assessment of potential factors affecting LV performance was based on data analysis of the histories. To reduce error in the precise frequency of occurrence and duration of AVRT or AVNRT, scales were introduced for AVRT or AVNRT duration (3 — more than five years, 2 — one to five years, 1 — less than one year) and recurrence (3 — at least once a week, 2 — at least once a month, 1 — less than once a month).

Transthoracic echocardiography

TTE was performed without any information on the patient or clinical data, using ACUSON 128XP with a 2.5–3.5 MHz transducer. All measurements were made according to established standards [7]. Recordings were taken with the patients in the left lateral decubitus position. The M-mode traces and Doppler signals were recorded at a speed of 50 mm/s. An average of three consecutive cycles was calculated for each parameter. Measurements of LV end-diastolic, end-systolic and left atrial dimensions were made on M-mode traces recorded from the parasternal long axis view. LV end-diastolic and end-systolic volumes were acquired from the apical four-chamber view. Both diameters and volumes were adjusted for body surface area. LV ejection fraction (EF) by the bi-plane Simpson method and mass were also calculated. Doppler parameters of mitral and right upper pulmonary vein (RUPV) flow, reflecting LV filling, were obtained from the apical four-chamber view with a sample volume of 2–4 mm. Mitral flow measurements included peak early transmitral filling velocity during early diastole (E), peak transmitral atrial filling velocity during late diastole (A), deceleration time (DT — the time elapsed between peak E velocity and the point where the extrapolation deceleration slope of the E velocity crosses the zero baseline), isovolumic relaxation time (IVRT — the time between the aortic valve closure and mitral valve opening) and the duration of A wave (tA). Assessment of RUPV flow involved measurement of systolic (S), diastolic (D) and atrial reversal (AR) peak velocities and duration of the AR (tAR) wave. The ratio of E and A (E/A) and S and D (S/D) waves and the difference between the duration of the AR and A waves (ΔT) were calculated. Measurements of mitral and RUPV flows enabled LV diastolic function to be assessed [8]:
— normal;
— slow isovolumic relaxation [IVRT > 92 ms (< 30 years), IVRT > 100 ms (30–50 years), IVRT > 105 ms (> 50 years)];

— slow early LV filling [E/A < 1 and DT > 220 ms (< 50 years), E/A < 0.5 and DT > 280 (> 50 years), S/D > 1.5 (< 50 years), S/D > 2.5 (> 50 years)];
— mitral flow pseudonormalisation (E/A > 1, DT and IVRT normal adjusted for age and complying with at least three of the following criteria [9–11]: S < D, E < A and increase in A velocity during Valsalva manoeuvre, AR ≥ 0.35 m/s, $\Delta T = tAR-tA \geq 20$ ms);
— mitral flow restriction (E/A > 2, DT < 140 ms, S/D < 0.5, AR > 0.35 m/s, $\Delta T > 30$ ms).

In the study groups TTE was performed up to 24 hours before EPS and RFCA and in the control group during recruitment to the study.

Reproducibility

All measurements were made by two independent observers and repeated in 10 randomly chosen patients at least one month from the baseline TTE in order to assess intra- and inter-observer variability calculated as the difference of two values and their arithmetical mean quotient. Intra- and inter-observer variability was found as follows: for LV diameters 1.7 ± 2.9% and 3.3 ± 2.1, LV volumes 3.5 ± 3.2% and 7.7 ± 2.8, fractional shortening (FS) 2.3 ± 2.2% and 4.4 ± 2.6, EF 4.5 ± 3.1 and 6.3 ± ± 3.4, mitral and RUPV flow velocities 2.6 ± 2.8 and 1.3 ± 2.5%, DT and IVRT 7.1 ± 5.4 and 9.1 ± 4.6%.

Electrophysiology study and ablation procedure

EPS and RFCA were performed according to a previously described procedure [12]. All anti-arrhythmic drugs were discontinued at least three half-lives before the study, except for amiodarone, which was withdrawn two months before. EPS was performed using quadripolar diagnostic electrodes introduced under fluoroscopic guidance. Three of the electrodes with a 0.5 cm interpolar distance were introduced via the femoral veins into the right atrial appendage (HRA), His bundle area and right ventricle apex. A fourth quadripolar diagnostic electrode recording unipolar and bipolar signals from the coronary sinus was introduced via the right internal jugular vein. The electrode was positioned in a standard location, identical for each patient, in order to obtain reproducible measurements. The right atrium and right ventricle were paced with impulses generated by Biotronic (Quinton Electrophysiology Corp., Seattle, WA, USA), recorded and displayed with the help of the 1993 Quinton EPAmp electrophysiological monitoring system. Two stimulation protocols were performed during the study:

— programmed stimulation of the HRA with a train of basic stimuli, a subsequent single stimulus and afterwards double extra stimuli with a gradually (20-ms step) shortened coupling interval;
— incremental pacing protocol. RFCA was performed with the ablation catheter placed at the site of earliest activation, using commercial ablation electrodes (Daig, Cordis, Medtronic). Radiofrequency energy was delivered at 40 W, with temperature up to 60°C for 60 s. In cases of left-sided AP the radiofrequency catheter was placed in the left atrium using a previously described technique of trans-septal puncture [13]. In the presence of atrioventricular node dissociation and AVNRT induction RFCA was performed from the right atrium [14].

Statistical analysis

All values were expressed as mean ± SD. Parametric demographic data and LV systolic and diastolic parameters were compared with a two-tailed Student's test for unpaired variables with normal distribution and with the Mann-Whitney test for variables without normal distribution. Non-parametric variables were assessed with the χ^2 test. Spearman's regression analysis was used to correlate potential factors affecting LV performance with parameters of LV systolic and diastolic function. P value < 0.05 was required to fulfil statistical significance.

Results

Study and control group characteristics

After EPS 25 patients with atrioventricular node dissociation and AVNRT (mean frequency 181.3 ± 25.4/min) were included in the AVNRT group: the typical (22 patients, 88%), the atypical (2 patients, 8%) and the slow-slow type (1 patient, 4%). A total of 65 (72.2%) patients with AP and AVRT (mean frequency 183.2 ± 25.4/min) were included in the WPW group. During EPS a left-sided AP was found in 37 (56.9%) patients and right-sided in 28 (43.1%). In 29 (44.6%) patients the AP was localised within the intraventricular septum and in 36 (55.4%) within the left or right ventricular free wall. Orthodromic AVRT was stimulated in 62 (95.4%) and antidromic in 3 (4.6%) patients. The control group consisted of 50 healthy volunteers (23 male, 27 female, mean age 39 ± 13 years). No difference was found in relation to age, sex, BMI, body surface area or systolic and diastolic blood pressure values. AVNRT was

Table 1. Study and control group characteristics.

Parameter	AVNRT (n = 25)	WPW (n = 65)	Control (n = 50)	p*	p**	p***
Age	42 ± 11	39 ± 11	39 ± 13	NS	NS	NS
Gender	7 M (28%)	33 M (50.8%)	23 M (46%)	0.053	NS	NS
BSA [m²]	1.77 ± 0.15	1.8 ± 0.15	1.75 ± 0.19	NS	NS	NS
SBP [mm Hg]	123 ± 11	125 ± 12	121 ± 10	NS	NS	NS
DBP [mm Hg]	77 ± 8	75 ± 6	76 ± 8	NS	NS	NS

*Comparison between AVNRT and WPW groups; **comparison between AVNRT and control groups; ***comparison between WPW and control groups; BSA — body surface area; SBP — systolic blood pressure; DBP — diastolic blood pressure

more frequent in women on the verge of statistical analysis (Table 1). Diameters and volumes were presented as body surface area indices.

Left ventricular systolic parameters in the study and control groups

The results of a comparison of LV systolic parameters between the study and control groups are shown in Table 2. Statistically significant differences in LV end-systolic diameters and FS were found between the AVNRT and WPW groups. In the WPW group end-systolic diameter was significantly increased and FS reduced compared to the AVNRT group. LVEF and FS in patients from the AVNRT group were reduced in comparison with the control group. An EF below 55% was found in 11 patients. In the WPW group a statistically significant increase in end-systolic diameter and volume and a reduction in LV FS and EF and in intraventricular septal thickening was observed. In the WPW group an EF below 55% was found in 35 patients, including one patient with tachyarrhythmic cardiomyopathy and an EF of 27%. A left or right-sided localisation of

the AP had no influence on LVEF or FS. AP localisation within or outside the intraventricular septum had an impact on intraventricular septal thickening. In the case of AP localisation within the intraventricular septum the thickening of the intraventricular septum was increased compared to a free-wall localisation and similar to the control group (50 ± ± 14 *vs.* 40 ± 13, p < 0.005). If the AP was situated within the free wall, the intraventricular septal thickening was significantly decreased compared to the control group (40 ± 13 *vs.* 52 ± 17, p < 0.001).

Diastolic function parameters in the study and control groups

A comparison of LV diastolic function parameters between the study and control groups is shown in Table 3. In 5 (20%) of the AVNRT patients LV diastolic dysfunction was not found, slow isovolumic relaxation was observed in 6 (24%), slow early LV filling in 9 (36%) and mitral flow pseudonormalisation in 5 (20%) patients. In 11 (17%) patients from the WPW group there were no LV diastolic function abnormalities. However, slow isovolumic

Table 2. LV systolic function parameters in the study and control groups.

Parameter	AVNRT (n = 25)	WPW (n = 65)	Control (n = 50)	p*	p**	p***
HR [1/min]	74 ± 9	74 ± 17	71 ± 7	NS	NS	NS
EDD [cm/m²]	2.66 ± 0.22	2.63 ± 0.26	2.70 ± 0.30	NS	NS	NS
ESD [cm/m²]	1.62 ± 0.38	1.79 ± 0.24	1.65 ± 0.24	0.05	NS	0.005
LAD [cm/m²]	1.91 ± 0.26	1.88 ± 0.35	1.98 ± 0.25	NS	NS	NS
M [kg/m²]	108 ± 24	100 ± 24	106 ± 33	NS	NS	NS
FS [%]	36 ± 4	32 ± 6	40 ± 5	0.005	0.005	0.001
IVSTh [%]	46 ± 16	44 ± 14	52 ± 17	NS	NS	0.05
PWTh [%]	63 ± 18	57 ± 18	62 ± 15	NS	NS	NS
EDV [ml/m²]	41 ± 7	44 ± 12	45 ± 12	NS	NS	NS
ESV [ml/m²]	19 ± 4	21 ± 8	17 ± 5	NS	NS	0.005
EF [%]	55 ± 5	54 ± 7	62 ± 4	NS	0.001	0.001

*Comparison between AVNRT and WPW groups; **comparison between AVNRT and control groups; ***comparison between WPW and control groups; HR — heart rate; EDD — LV end-diastolic diameter; ESD — LV end-systolic diameter; LAD — left atrial end-diastolic diameter; M — LV mass; FS — LV fraction shortening; IVSTh — intraventricular septal thickening; PWTh — LV posterior wall thickening; EDV — LV end-diastolic volume; ESV — LV end-systolic volume; EF — LV ejection fraction

Table 3. LV diastolic function parameters in the study and control groups.

Parameter	AVNRT (n = 25)	WPW (n = 65)	Control (n = 50)	p*	p**	p***
E [cm/s]	69 ± 17	74 ± 19	88 ± 15	NS	0.001	0.001
A [cm/s]	68 ± 13	68 ± 19	63 ± 11	NS	NS	NS
E/A	1.09 ± 0.41	1.15 ± 0.34	1.42 ± 0.3	NS	0.001	0.001
ΔT [ms]	8 ± 39	9 ± 27	–23 ± 18	NS	0.001	0.001
DT [ms]	223 ± 34	217 ± 37	175 ± 24	NS	0.001	0.001
IVRT [ms]	104 ± 15	104 ± 14	82 ± 12	NS	0.001	0.001
Ev [cm/s]	55 ± 16	59 ± 14	70 ± 13	NS	0.001	0.001
Av [cm/s]	60 ± 0.13	61 ± 16	55 ± 11	NS	NS	0.05
E/Av	0.93 ± 0.35	1.0 ± 0.3	1.30 ± 0.23	NS	0.001	0.001
S [cm/s]	58 ± 11	59 ± 12	59 ± 8	NS	NS	NS
D [cm/s]	49 ± 10	53 ± 11	55 ± 8	NS	0.01	NS
S/D	1.18 ± 0.30	1.16 ± 0.30	1.08 ± 0.18	NS	0.05	NS
AR [cm/s]	27 ± 7	27 ± 8	14 ± 12	NS	0.001	0.001

*Comparison between AVNRT and WPW group; **comparison between AVNRT and control group; ***comparison between WPW and control group; E — early mitral filling velocity; A — atrial filling velocity of mitral flow; E/A — ratio of E to A; DT — deceleration time of E wave; IVRT — isovolumic relaxation time; Ev — early mitral filling velocity during Valsalva manoeuvre; Av — atrial filling velocity of mitral flow during Valsalva manoeuvre; E/Av — ratio of E to A during Valsalva manoeuvre; S — systolic velocity of right upper pulmonary vein flow; D — diastolic velocity of right upper pulmonary vein flow; S/D — ratio of S to D; AR — atrial reversal of systolic velocity of right upper pulmonary vein flow; ΔT — difference in duration of A and AR waves

Table 4. Factors affecting LV systolic function in patients from the AVNRT and WPW groups.

Parameter	AVNRT (n = 25)		WPW (n = 65)	
	R	p	R	p
Scale	–0.29	NS	–0.27	0.05
Frequency [1/min]	0.07	NS	0.19	NS
Duration [years]	0.14	NS	0.05	NS

R — Spearman's correlation index; scale — scale of occurrence AVRT or AVNRT; frequency — frequency of AVRT or AVNRT; duration — time from the first ever episode of AVRT or AVNRT

Table 5. Factors affecting LV diastolic function in patients from the AVNRT and WPW groups.

Parameter	AVNRT (n = 25)		WPW (n = 65)	
	R	p	R	p
Scale	0.52	0.01	0.67	0.001
Frequency	0.25	NS	0.07	NS
Duration	–0.08	NS	0.09	NS

R — Spearman's correlation index; scale — scale of occurrence AVRT or AVNRT; frequency — frequency of AVRT or AVNRT; duration — time from the first ever episode of AVRT or AVNRT

relaxation was found in 19 (29.2%), slow early LV filling in 24 (36.9%), mitral flow pseudonormalisation in 10 (15.4%) and restriction in 1 (1.5%) patient. There were no differences in type of LV diastolic dysfunction between the AVNRT and WPW groups (p > 0.05). No LV diastolic function abnormality was found in any subject from the control group.

The relations between potential factors influencing LV performance and systolic and diastolic function parameters are presented in Tables 4 and 5. The frequency of AVRT or AVNRT and the scale of AVRT or AVNRT recurrence and history duration were similar in the two groups (p > 0.05). The only factor affecting LVEF was the scale of recurrence of AVRT in the WPW group. However, the patient with tachyarrhythmic cardiomyopathy and

an EF of 27% having been excluded from the analysis, no such correlation was found (R = –0.23, p = 0.068) in the WPW group. No relation was observed between the scale of AVNRT recurrence and LV systolic function in the AVNRT group. The scale of AVRT or AVNRT recurrence affected LV diastolic function. This positive correlation was stronger in the WPW group.

Discussion

Chronic supraventricular tachycardia produces a dilated cardiomyopathy. After one week of pacing at 400/min, the left atrium booster pump for the LV is affected and congestive heart failure starts to develop with moderate increases in left atrial pressure and LV volumes. After 6 weeks of rapid

pacing severe congestive heart failure is usually present with a marked increase in LV end-diastolic pressure, volumes and stiffness. LV dilation is accompanied by wall thinning in the absence of significant changes in cardiac weight. On the cellular level, myocyte loss or elongation, myofibril misalignment and disruption of the extracellular matrix architecture are reported [2–4, 15–17].

This study demonstrates that recurrent episodes of AVRT or AVNRT affect LV function, both systolic and diastolic. The results of LV systolic function analysis in patients with AVRT or AVNRT show that LVEF, FS and also, in the WPW group, LV end-systolic diameters differ significantly from those of healthy subjects and are compatible with other data [5, 6] (Table 2). In agreement with the results of Fishberger et al. [18] but in contrast to other studies [1–5, 19], no significant differences were found between the study and control groups with regard to LV end-diastolic diameters. However, the number of subjects and the duration of tachycardia in our study differed from other studies and, in addition, patients with concomitant diseases were excluded.

The localisation of AP within the free wall resulted in a reduction of intraventricular septal thickening. The probable mechanism of this finding is LV wall asynergy during systole as a consequence of a resting pre-excitation, which was present in 54 (83.1%) of WPW patients. Where there was AP free-wall localisation, intraventricular septal contraction was delayed in relation to the free wall. Posterior wall thickening was not decreased in the presence of AP within the intraventricular septum, which could be expected. However, the precise localisation of the AP within the anterior or posterior part of the intraventricular septum has not been taken into account.

The mechanism of LV systolic dysfunction development in patients with WPW syndrome and orthodromic AVRT and patients with AVNRT is probably similar and resembles the model of rapid atrial stimulation suggested by Spinale et al. [3]. It was shown that atrial stimulation 240/min for three weeks does not affect myocardial blood flow in the first instance. Deterioration of myocardial perfusion is the result of the development of heart failure. Myocardial perfusion may be different in patients with orthodromic and antidromic AVRT. Helmer et al. proposed an experimental model of regional LV stimulation and impaired myocardial blood flow within the stimulated myocardium, resulting in myocyte damage [16].

Such analysis was not carried out in our study as there were only 3 (4.6%) patients with antidromic AVRT.

It was shown in the present study that recurrent AVRT or AVNRT lead to LV diastolic function disturbances, mainly slow LV relaxation or slow early LV filling. More advanced types of LV diastolic dysfunction, such as mitral flow pseudonormalisation or restriction, were observed in 20% of AVNRT and 14% of WPW patients. Quantitative analysis of LV diastolic function parameters revealed significant differences between the study and control groups (Table 3). However, RUPV systolic velocities did not differ between the study and control groups. RUPV systolic velocity is related to mean left atrial pressure and LV end-diastolic pressure. An increase in mean left atrial or LV end-diastolic pressure will result in a reduction of RUPV systolic velocity and therefore mean RUPV systolic velocity in patients with all types of LV diastolic dysfunction may not differ between the study and control groups. Analysis of potential factors influencing LV systolic function showed that only the scale of AVRT recurrence affects LVEF (Table 4). The patient with tachyarrhythmic cardiomyopathy having been excluded from the WPW group, this correlation was no longer found. No factors influencing LV systolic performance were found in the AVNRT group. The scale of AVRT or AVNRT recurrence was proved to affect LV diastolic function. This positive correlation was more significant in the WPW group, which might be the result of the number of subjects. The scale of AVRT or AVNRT recurrence reflects the duration of exposure to tachycardia. The more frequent the episodes of AVRT or AVNRT, the longer the total exposure to tachycardia and the more advanced the LV diastolic dysfunction. No relation between frequency of AVRT or AVNRT and LV systolic or diastolic dysfunction was observed. Such correlations have been described in other studies [1–4, 19, 20], although in our study the AVRT or AVNRT frequency only ranged from 160 to 200/min in 87% of patients.

Conclusions

Atrioventricular tachycardia and atrioventricular nodal re-entrant tachycardia may lead to development of left ventricular systolic and diastolic dysfunction. Left ventricular diastolic dysfunction is related to the frequency of recurrence of the atrioventricular tachycardia or atrioventricular nodal re-entrant tachycardia.

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