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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The effect of atrial pacing site on electrophysiological properties of the atrioventricular junction and induction of atrioventricular nodal reentry in patients with typical atrioventricular nodal reentrant tachycardia

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Abstract

Background: Clinical studies in humans have shown the site of atrial stimulation to influence atrioventricular (AV) conduction times and refractory periods, the demonstration of dual AV nodal (AVN) pathways, and induction of AVN reentry. These studies often found conflicting results. Moreover, among enrolled patients a minority of them were found to have AVN reentrant tachycardia (AVNRT).

Aim: The purpose of this study was to investigate the effect of right and left atrial pacing on the electrophysiological properties of the AV junction in the typical AVNRT population.

Methods: Ninety-two consecutive patients with typical AVNRT were included. Atrial pacing was performed from the high right atrium (HRA) and the left atrium via the proximal coronary sinus (CS).

Results: Stimulation from either the HRA or the CS could result in dual AVN physiology and AVNRT. No site-dependent differences in the ease of induction of dual AVN pathways with variability of initiation from either site were found. However, AVNRT was easier to induce from the HRA. With CS pacing the leftward but not the rightward AVN approaches were the entry point to the AV node because of significantly shorter AH conduction times compared to HRA pacing. Conduction over the leftward AVN extensions could initiate the tachycardia with significantly shorter critical AH interval compared to conduction over the rightward AVN extensions; however, the AH interval during AVNRT and its cycle length were not significantly different.

Conclusions: Rightward and leftward AVN extensions are regular features of the AV node. Their different electrophysiological properties lead to variation in the demonstration of discontinuous AVN conduction and AVNRT during right and left atrial pacing. Despite the observation that the left AVN extensions could compose the entry point to the reentrant circuit, there is no evidence that they constitute the critical component of sustained typical AVNRT.

Key words: atrioventricular node, atrioventricular nodal extensions, atrioventricular nodal reentrant tachycardia

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INTRODUCTION

Clinical studies in humans have shown the site of atrial stimulation to influence atrioventricular (AV) conduction times and refractory periods, the demonstration of dual atrioventricular nodal (AVN) pathways, and induction of AVN reentry [1–8]. These studies often found conflicting results that could be explained by different atrial pacing sites and the electrophysiological study (EPS) protocol applied. Moreover, among enrolled patients the minority of them were found to have AVN reentrant tachycardia (AVNRT). The purpose of this study was to investigate the effect of right and left atrial pacing on the electrophysiological properties of the AV junction and induction of AVN reentry in the typical AVNRT population.

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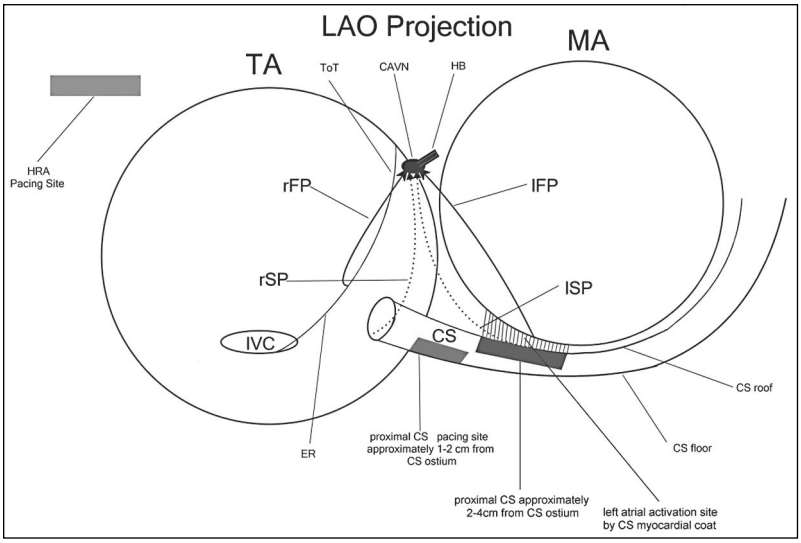

Figure 1. A schematic diagram of the AV node with postulated anterograde AV nodal (AVN) transmission during different atrial pacing sites in the left anterior oblique (LAO) projection. During high right atrium (HRA) pacing the atrial wavefront propagates toward the compact AV node (CAVN) and then the His bundle (HB) via the rightward fast AVN pathway (rFP), until it is refractory, with following conduction via the rightward slow AVN pathway (rSP). During proximal coronary sinus (CS) pacing the atrial wavefront propagates toward the CAVN via the leftward fast AVN pathway (lFP) with following conduction via the leftward slow AVN pathway (lSP). The rightward AVN anterior extension, considered as an electrophysiological substrate for the FP, travels along the tendon of Todaro (ToT), which is a continuation of the Eustachian ridge (ER) of the inferior vena cava (IVC). The rightward AVN posterior extension, which represents the electrophysiological substrate for the SP, travels along the septal leaflet of the tricuspid annulus (TA). The activation of the left atrium during proximal CS pacing takes place by the CS myocardial coat at the site located 2–4 cm from the CS ostium. The leftward posterior AVN extension travels within the roof of the proximal CS along the mitral annulus (MA), in the left inferoseptal region. Clinically, the leftward inferior nodal extension can behave as the SP

METHODS

Patient characteristics

Ninety-two consecutive patients (59 women and 33 men with a mean age of 46.6 ± 17.6 years) with invasive diagnosis of slow-fast AVNRT were included in the study. 28% of patients had a history of ischaemic or hypertension heart disease; however, they had normal atrial dimensions and function, and no enlargement of the tricuspid and mitral annulus was found. All patients displayed no evidence of prolonged intra-atrial as well as AV conduction times at the time of EPS and were studied before radiofrequency (RF) catheter ablation. The occurrence of accessory pathways was excluded by classical manoeuvres [5, 9]. The study complies with the Declaration of Helsinki. All patients provided written informed consent.

Electrophysiological study

Patients were studied in a drug-free state. Three quadripolar catheters (5-mm inter-electrode distance) were introduced via the right femoral vein and positioned in the high lateral right atrium (HRA) at the junction of the superior vena cava and right atrium (RA), the right ventricular apex (RVA), and His bundle region to record the His bundle electrogram (HBE). The atrial electrogram (AE) on the HBE showed sufficient amplitude and stable morphology with clearly visible His bundle deflection to precisely measure the AH intervals during both sinus and atrial pacing. The measurement of the AH interval was taken from the earliest reproducible rapid deflection of the AE to the onset of the His deflection, defined by the earliest deflection from baseline. HV interval ≥ 30 ms excluded right bundle branch recording [10]. A decapolar catheter (5-mm inter-electrode distance) was advanced into the coronary sinus (CS) via the left subclavian vein, with the most proximal pair of electrodes positioned 1–2 cm from the CS ostium. Localisation of the ostium was facilitated by: (1) observing, in the left anterior oblique projection, the entry point into the CS of the diagnostic catheter; and (2) stimulation from the proximal CS was associated with inverted P waves in inferior leads [10–12]. The distal pair of electrodes of quadripolar catheters and the proximal pair of electrodes of the decapolar catheter were used for pacing. The stimulus was 1 ms in duration and delivered at twice the diastolic threshold. Atrial pacing was performed from the HRA and low posterior left atrium (LA) via the proximal CS (Fig. 1). In each patient alteration of pacing site was associated with a change of the AE morphology on the HBE (Fig. 2, 3). The study protocol consisted of incremental pacing (IP) and single extrastimulus testing (SET). IP was initiated at a cycle length (CL) just shorter than a sinus rate and progressively decreased (10–50 ms steps) until Wenckebach block occurred. Before

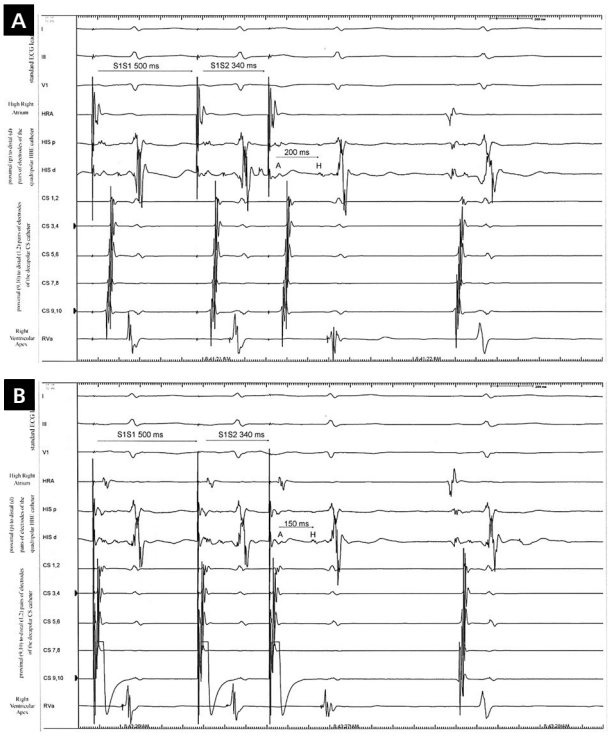

Figure 2. The effect of atrial pacing site on atrioventricular (AV) nodal conduction. Both panels show atrial single extrastimulus testing from the right (**A**) and left (**B**) atrium performed at the same patient. Right atrial pacing was performed from the high right atrium (HRA), and the left atrium pacing via the proximal coronary sinus (CS). The measurement of the AH interval was taken from the earliest reproducible rapid deflection of the atrial electrogram (A) to the onset of the His deflection (H), recorded on the distal pair of electrodes of the quadripolar catheter positioned in the His bundle region (His d). At the same drive cycle length (S1S1), the same number of driven stimuli, and the same extrastimulus coupling interval (S1S2), CS stimulation caused a significantly shorter AH interval (150 ms) than HRA stimulation (200 ms). We support the concept that the different atrial pacing sites resulted in AV nodal activation via the different right and left atrial inputs. During HRA pacing the atrial wavefront propagated toward the AV node via the rightward AV nodal pathway(s) but during proximal CS pacing via the leftward AV nodal pathway(s) because of significantly different AH conduction times

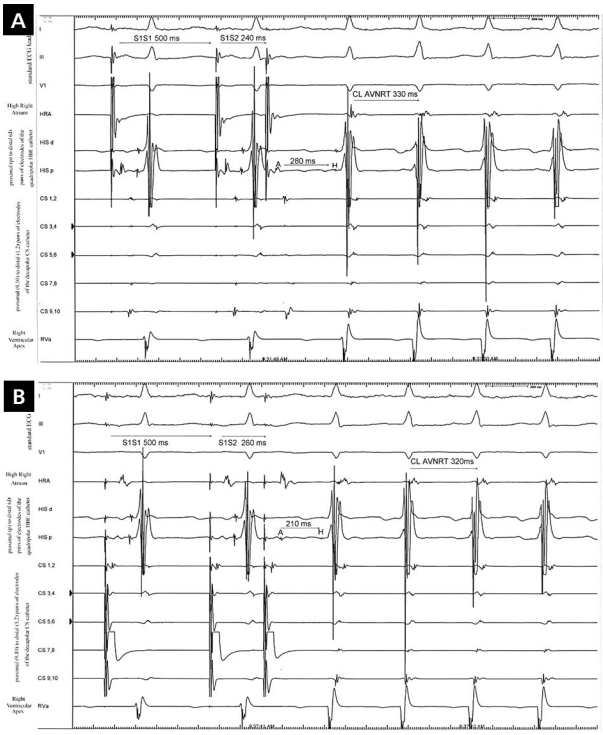

Figure 3. The effect of atrial pacing site on the electrophysiological properties of atrioventricular nodal (AVN) reentrant tachycardia (AVNRT). Both panels show initiation of AVNRT with the atrial single extrastimulus testing from the right (**A**) and left (**B**) atrium in the same patient. Right atrial pacing was performed from the high right atrium (HRA), and the left atrium pacing via the proximal coronary sinus (CS). The measurement of the AH interval was taken from the earliest reproducible rapid deflection of the atrial electrogram (A) to the onset of the His deflection (H), recorded on the proximal pair of electrodes of the quadripolar catheter positioned in the His bundle region (His p). At the same drive cycle length (S1S1), the same number of driven stimuli, and the similar extrastimulus coupling interval (S1S2), CS stimulation caused a significantly shorter critical AH interval (210 ms) required for AVNRT induction than HRA stimulation (280 ms), followed by similar tachycardia cycle length (CL AVNRT). It seems that the leftward AVN extension composed the entry point to the reentrant circuit during the first reentrant beat when the CS was paced and the rightward AVN extension when the HRA was stimulated because of significantly different critical AH conduction times required for AVNRT induction. During the following reentrant beats, regardless of AVNRT induction pacing site, anterograde conduction must have been over the same AVN pathway because the AH interval during AVNRT and its cycle length were not significantly different

changing pacing, CL sinus rhythm was allowed to return to baseline. SET was performed with 500 ms drive CL. Each extrastimulus was introduced after every eighth paced beat successively earlier until the atrium was refractory. Prematurity of the test atrial impulse was reduced in 10-ms decrements. If AVNRT was not induced during these manoeuvres (31 patients) the whole protocol was repeated during isoprenaline infusion that increased gradually until a stable sinus rate 25% higher than the initial basic rate but of at least 100 bpm was achieved [11, 12]. Finally, all patients had inducible AVNRT, diagnosed using the standard criteria [5, 13]. After changing the atrial pacing site IP was started at the same CL and SET at the same coupling interval. Standard definitions for Wenckebach CL (WBCL), effective refractory period (ERP) of the AV node, the

fast (FP) and slow pathways (SP), as well as the atrium were used [10]. Both atrial extrastimulus testing (an increase in the A2H2 interval of > 50 ms in response to a decrease in the A1A2 coupling interval of < 10 ms) [5, 9] and rapid atrial pacing (similar beat-to-beat change in the AH interval of 50 ms and the PR interval that exceeds the RR interval) [9, 14] were used to demonstrate discontinuous AVN conduction. The earliest retrograde atrial activation (ERAA) during RVA pacing was observed on the HBE in all patients. All intervals were measured at a display sweep speed of 100 ms.

Catheter ablation

We used a stepwise technique for SP ablation [15]. In all patients RF ablation between the CS ostium and tricuspid annulus eliminated AVNRT.

Statistical analysis

All measurements are reported in milliseconds (ms) and expressed as mean value ± standard deviation. Comparisons between groups were performed with the McNemar's test or the Wilcoxon signed-rank test. A p value < 0.05 was accepted as statistically significant.

RESULTS

Demonstration of discontinuous AVN conduction

At the basic state, without isoprenaline infusion, demonstration of discontinuous AVN conduction was seen in 95% of cases. We observed no significant differences in the ease of demonstration of AVN pathways from the right and left atrial pacing site ($p > 0.05$); however, we noted variability of initiation from either site. In most cases discontinuous AVN conduction was revealed with stimulation from both sites (79%). In rare cases, it was accomplished only from one site (the HRA in 9% and the CS in 7%). In 31 patients in whom EPS protocol was repeated after isoprenaline infusion we observed discontinuous AVN conduction in 80% of cases. In this group AVN pathways were easier to reveal from the HRA than from the CS ($p = 0.03$). Discontinuous AVN conduction was observed with stimulation from both sites in 48% and exclusively from the HRA in 29% and from the CS in 3% of cases.

AVN conduction times and refractory periods

The data were analysed in three different groups. Group A consisted of patients who presented discontinuous AVN conduction from both the HRA and CS (Table 1). Group B consisted of patients who did not present AVN pathways from the HRA or from CS (Table 2). Group C was considered as the entire group of patients (Table 3). We did not analyse the subgroup of patients who demonstrated discontinuous AVN conduction only from the single pacing site.

Effectiveness in inducing AVNRT

We observed that AVNRT was easier to induce from the HRA than from the CS ($p = 0.02$) with variability of initiation from either site. In most cases AVNRT was induced with stimulation from both sites (70%). Demonstration of AVNRT was accomplished only from the HRA in 22% and only from the CS in 8% of cases.

Electrophysiological properties of AVNRT

The data were analysed only in patients who presented AVNRT induction from both the HRA and CS (Table 4). In all patients the ERAA during the tachycardia was recorded on the HBE.

DISCUSSION

Anterograde AVN transmission during pacing

Several clinical studies have shown the site of atrial stimulation to influence AVN transmission of impulses. This finding was usually interpreted as the result of AVN activation via the different right and left atrial inputs. However, the obtained outcomes cannot be reliably referred to AVNRT patients because of their marginal participation in those studies. Moreover, different right (high, mid, or low) and left atrial pacing sites (via different CS sites, a persistent foramen ovale or a transseptal puncture) and EPS protocols seem to be the possible reason for conflicting results [1–8]. In all mentioned studies LA stimulation resulted in shorter AH intervals than did RA stimulation. Moreover, Suzuki et al. [8] and Gonzalez et al. [3] documented (although not consistently) that AVN conduction depends on CS pacing site. Suzuki et al. [8] reported that the AH interval was the longest with stimulation from the HRA, followed by the distal, mid, and proximal CS; however, in some patients the AH interval was identical or longer when measured during proximal vs. distal CS pacing. The AH interval was significantly different among the pacing sites except the mid and proximal CS. They indicated that a wavefront from the distal CS propagates toward the AV node mainly via the anterior LA approach, from the mid and proximal CS via the posterior LA approach, and from the HRA via the anterior RA approach [8]. Gonzalez et al. [3] found that the AH interval prolonged as the site of pacing was moved from the distal to the proximal CS, but it was always shorter compared with RA stimulation. They explained this finding by a gradual shift between two atrial inputs: from the mitral annulus (MA) that could represent the functional counterpart of the leftward posterior extension to the rightward posterior approach [3].

Our observations are consistent with findings that LA stimulation via the CS results in shorter AH intervals than RA stimulation (Fig. 2). It is clear to us that during proximal CS pacing the rightward AVN approaches did not provide the input to the AV node because of the significantly shorter AH conduction times compared with HRA pacing. Moreover, the LA activation during CS pacing propagated from this area toward the septum via the leftward fast or the slow pathway. The significantly shorter AH conduction times over the fast and slow pathway during CS than HRA pacing, among the patients with discontinuous conduction curves, strongly support this

Table 1. The effect of atrial pacing site on the electrophysiological properties of the atrio-ventricular junction obtained during single extrastimulus testing and incremental pacing in group A

Variable	HRA	CS	p	HRA	CS	p
	At the basic state (n = 44)			After isoprenaline infusion (n = 9)		
Single extrastimulus testing (SET)						
AH_{min} FP	151 ± 59	120 ± 54	< 0.0001	102 ± 21	69 ± 24	0.01
AH_{max} FP	182 ± 67	159 ± 63	< 0.0001	152 ± 28	121 ± 32	0.02
ΔAH FP	33 ± 27	39 ± 31	0.3	50 ± 30	52 ± 23	0.9
ERP FP	362 ± 73	340 ± 66	< 0.0001	291 ± 33	260 ± 41	0.07
AH_{min} SP	303 ± 93	273 ± 90	0.0005	264 ± 37	222 ± 50	0.02
AH_{max} SP	360 ± 100	312 ± 96	< 0.0001	297 ± 54	233 ± 48	0.01
ΔAH SP	57 ± 59	39 ± 46	0.04	33 ± 46	11 ± 15	0.2
ERP SP	310 ± 70	306 ± 58	0.5	266 ± 34	252 ± 36	0.5
ERP A	288 ± 81	288 ± 67	0.7	264 ± 36	246 ± 37	0.2
	At the basic state (n = 46)			After isoprenaline infusion (n = 11)		
Incremental pacing (IP)						
AH_{min}	104 ± 28	78 ± 20	< 0.0001	78 ± 15	62 ± 12	0.008
AH_{max}	292 ± 57	254 ± 58	< 0.0001	256 ± 68	232 ± 31	0.08
ΔAH	192 ± 61	174 ± 56	0.005	175 ± 60	170 ± 29	0.4
aWBCL	379 ± 45	371 ± 45	0.01	327 ± 35	317 ± 25	0.3

Parameters measured during SET: $AH_{min\ FP}$ — the shortest AH interval recorded with conduction over the fast pathway (FP); $AH_{max\ FP}$ — the longest AH interval recorded with conduction over the FP; ΔAH_{FP} — the difference between the minimal and maximal AH interval; ERP FP — the effective refractory period of the FP; $AH_{min\ SP}$ — the shortest AH interval recorded with conduction over the slow pathway (SP); $AH_{max\ SP}$ — the longest AH interval recorded with conduction over the SP; ΔAH_{SP} — the difference between the minimal and maximal AH interval; ERP SP — the effective refractory period of the SP; ERP A — the effective refractory period of the atrium. Parameters measured during IP: AH_{min} — the shortest AH interval measured during the longest pacing cycle length (CL); AH_{max} — the longest AH interval measured just before the onset of AVN Wenckebach phenomenon; ΔAH — the difference between the minimal and maximal AH interval; aWBCL — anterograde Wenckebach CL; CS — coronary sinus; HRA — high right atrium

Table 2. The effect of atrial pacing site on the electrophysiological properties of the atrioventricular junction obtained during single extrastimulus testing and incremental pacing in group B

Variable	HRA	CS	p	HRA	CS	p
	At the basic state (n = 26)			After isoprenaline infusion (n = 10)		
Single extrastimulus testing (SET)						
AH_{min}	191 ± 86	155 ± 86	< 0.0001	96 ± 28	69 ± 20	0.008
AH_{max}	279 ± 99	245 ± 89	0.006	183 ± 45	155 ± 30	0.02
ΔAH	88 ± 55	90 ± 54	1.0	85 ± 43	81 ± 33	0.5
ERP AVN	294 ± 59	294 ± 64	0.9	235 ± 52	226 ± 24	0.3
ERP A	262 ± 42	275 ± 67	0.6	225 ± 36	223 ± 35	0.9
	At the basic state (n = 18)			After isoprenaline infusion (n = 15)		
Incremental pacing (IP)						
AH_{min}	98 ± 23	73 ± 18	0.0002	81 ± 16	62 ± 13	0.001
AH_{max}	218 ± 68	173 ± 66	0.0003	173 ± 61	158 ± 70	0.06
ΔAH	123 ± 56	99 ± 55	0.007	95 ± 69	96 ± 69	0.9
aWBCL	351 ± 56	349 ± 58	0.6	306 ± 37	316 ± 29	0.1

Parameters measured during SET: AH_{min} — the shortest AH interval measured during the longest coupling interval, AH_{max} — the longest AH interval measured just before the onset of ERP AVN, ΔAH — the difference between the minimal and maximal AH interval, ERP AVN — the effective refractory period of the AV node, ERP A — the effective refractory period of the atrium. Parameters measured during IP: AH_{min} — the shortest AH interval measured during the longest pacing cycle length (CL), AH_{max} — the longest AH interval measured just before the onset of AVN Wenckebach phenomenon, ΔAH — the difference between the minimal and maximal AH interval, aWBCL — anterograde Wenckebach CL; CS — coronary sinus; HRA — high right atrium

Table 3. The effect of atrial pacing site on the electrophysiological properties of the atrioventricular junction obtained during single extrastimulus testing and incremental pacing in group C

Variable	HRA	CS	p	HRA	CS	p
	At the basic state (n = 92)			After isoprenaline infusion (n = 31)		
Single extrastimulus testing (SET)						
AH_{min}	163 ± 76	134 ± 73	< 0.0001	95 ± 24	75 ± 37	< 0.0001
AH_{max}	327 ± 108	283 ± 100	< 0.0001	247 ± 72	207 ± 63	0.002
ΔAH	163 ± 91	147 ± 78	0.06	152 ± 73	129 ± 62	0.04
ERP AVN	301 ± 68	298 ± 60	0.7	241 ± 33	239 ± 31	0.7
ERP A	278 ± 70	280 ± 63	1.0	234 ± 40	233 ± 33	0.8
	At the basic state (n = 92)			After isoprenaline infusion (n = 31)		
Incremental pacing (IP)						
AH_{min}	102 ± 28	77 ± 22	< 0.0001	80 ± 15	63 ± 13	< 0.0001
AH_{max}	271 ± 72	224 ± 69	< 0.0001	223 ± 83	185 ± 68	0.007
ΔAH	169 ± 72	145 ± 61	< 0.0001	143 ± 82	122 ± 66	0.2
aWBCL	371 ± 57	362 ± 53	0.006	316 ± 38	318 ± 32	0.8

Parameters measured during SET: AH_{min} — the shortest AH interval measured during the longest coupling interval, AH_{max} — the longest AH interval measured just before the onset of ERP AVN, ΔAH — the difference between the minimal and maximal AH interval, ERP AVN — the effective refractory period of the AV node, ERP A — the effective refractory period of the atrium. In the cases where clear demonstration of discontinuous AVN conduction curves on extrastimulus testing was observed AH_{min} was measured as the shortest AH interval recorded with conduction over the fast pathway, AH_{max} was measured as the longest AH interval recorded with conduction over the slow pathway, and ERP AVN was measured as the effective refractory period of the slow pathway. Parameters measured during IP: AH_{min} — the shortest AH interval measured during the longest pacing cycle length (CL), AH_{max} — the longest AH interval measured just before the onset of AVN Wenckebach phenomenon, ΔAH — the difference between the minimal and maximal AH interval, aWBCL — anterograde Wenckebach CL; CS — coronary sinus; HRA — high right atrium

Table 4. Electrophysiological properties of atrioventricular nodal reentrant tachycardia (AVNRT) induced from different atrial pacing sites during single extrastimulus testing (SET) and incremental pacing (IP).

Variable	SET (n = 39)			IP (n = 35)		
	HRA	CS	p	HRA	CS	p
$S2_{crit}$	319 ± 66	308 ± 62	0,03	–	–	–
CL_{crit}	–	–	–	367 ± 42	354 ± 44	0.01
AH_{crit}	289 ± 73	248 ± 72	< 0.0001	270 ± 60	237 ± 51	< 0.0001
AVNRT CL	362 ± 64	352 ± 62	0.15	363 ± 54	351 ± 50	0.19
AVNRT AH	293 ± 62	282 ± 61	0.11	289 ± 50	277 ± 50	0.24

$S2_{crit}$ — the critical coupling interval for AVNRT induction; CL_{crit} — the critical pacing cycle length for AVNRT induction; AH_{crit} — the critical AH interval for AVNRT induction; CS — coronary sinus; HRA — high right atrium

theory. It seems to us that the leftward extensions could stand for the preferential access to the compact AV node during CS pacing. We support the concept that during HRA pacing the atrial wavefront propagated toward the AV node via the rightward fast AVN pathway, until it was refractory, with following conduction via the rightward slow AVN pathway if dual AVN pathways were revealed. Similarly, during proximal CS pacing the atrial wavefront propagated toward the AV node via the leftward fast AVN pathway with following conduction via the leftward slow AVN pathway.

While AVN transmission during RA pacing is well established and takes place via the rightward AVN extensions located in the region of the triangle of Koch (ToK) [5, 9, 10, 13], AVN transmission during LA pacing remains controversial. The rightward anterior extension, considered as an electrophysiological substrate for the FP, travels from the anterior limbus of the fossa ovalis along the tendon of Todaro with the ERAA usually recorded at the same site recording the His bundle potential, in the apex of the ToK [4, 9, 13, 16–19]. The rightward posterior extension, which represents the electrophysiological substrate for the SP, travels within the ToK along the septal leaflet of the tricuspid annulus (TA) with the ERAA usually recorded between the TA and CS ostium, in the base of the ToK [9, 12, 13, 19]. It has been known for some time

that the leftward posterior extension can provide the LA input to the AV node, showing a preferential access from the left inferior septum [3–5, 8, 10, 11, 13, 20, 21]. The ERAA over the leftward posterior AVN pathway can be recorded along the posterior MA, in the left inferoseptal region or along the roof of the proximal CS, usually 2–4 cm (as much as 5 to 6 cm) from the ostium [11, 13]. It means that the leftward posterior nodal extension travels within the myocardial coat of the proximal CS [13], which is consistent with anatomical studies that shown that the LA myocardium is continuous with the CS musculature in its proximal portion and terminates beyond orifices of the coronary veins [20]. Clinically, the leftward inferior nodal extension can behave as the SP.

However, exact atrial insertions of the leftward AVN approaches can potentially be identified by locating the site of the ERAA during selective retrograde conduction over these pathways [12, 13]; it was not the key of this study, especially that during RVA pacing retrograde conduction was observed exclusively over the rightward FP. Nonetheless, it seems to us that the activation of the LA during proximal CS pacing took place by the CS myocardial coat at the site located close to the CS ostium. Postulated anterograde AVN transmission during different atrial pacing sites can be seen in Figure 1.

AVN transmission during AVNRT

Consistent with Josephson [5], we found that the critical AH interval required for AVNRT induction was shorter from the CS (Fig. 3), achieved at a significantly shorter coupling interval during SET and a significantly shorter CL during IP. It seems to us that during LA pacing conduction over the leftward AVN extensions can initiate the tachycardia. In contrast to Josephson [5], the present study demonstrated that the AH interval during AVNRT and its CL (measured immediately after the initiation as well as during the sustained tachycardia) were not significantly shorter when AVNRT was initiated from the CS. These observations, supported by the fact that in all patients' ablation at the right inferoseptal region eliminated AVNRT, suggest that the rightward posterior AVN extensions were the critical component of sustained AVNRT. However, the leftward AVN extensions composed the entry point to the reentrant circuit during the first reentrant beat when the CS (but not the HRA) was paced. During the first and following reentrant beats, regardless of the site of stimulation, the ERAA was always detected on the HBE, which suggests that retrograde conduction was over the rightward FP. During the second and the following reentrant beats, regardless of AVNRT induction pacing site, anterograde conduction must have been over the rightward SP with retrograde conduction over the rightward FP.

However, whether the left-sided atrionodal connection constitutes a critical component of the reentrant circuit or is only an innocent bystander is unclear. In some cases, direct left-sided ablation to the ERAA site within the CS rendered the tachycardia non-inducible, which suggests that the left-sided SP may constitute the critical part of the reentrant circuit (left variant slow-fast AVNRT, 5% of patients) [9, 11–13, 22]. In these cases, the leftward inferior extension serves as the SP conducting anterogradely, with leftward FP conducting retrogradely. Alternatively, the entire circuit may be located on the left side of the heart (left atrial slow-fast AVNRT, 1% of patients), and to eliminate the tachycardia there is a necessity of ablating along the MA [11, 12]. In our study, during sustained AVNRT, the left-sided atrionodal connection did not constitute a critical component of the reentrant circuit.

Electrophysiological properties of the AV junction and induction of AVNRT

Previous reports have documented that the site of atrial stimulation can affect AVN function and the ability to initiate dual AVN pathways and AVNRT [1–8].

No site-dependent differences in the ease of induction of dual AVN pathways and AVNRT were found by Ross et al. [7]; however, they noted variability of initiation from either site. The differences were noted by Josephson [5]. Dual AVN pathways and reentry were easier to reveal from the HRA than from the CS. In the majority of cases the tachycardia was induced with stimulation from both sites although in rare cases it was accomplished only from the CS. In contrast to those authors we found no site-dependent differences in the ease of induction of dual AVN pathways, with variability of initiation from either site, but we found them during AVNRT induction. AVNRT was easier to reveal from the HRA. The different atrial pacing sites and EPS protocols seem to be a possible reason for the conflicting results with those studies.

Ross et al. [7] showed no significant alteration of refractoriness of the fast and slow pathway. In other studies, Aranda et al. [1], Gonzalez et al. [3], and Leon et al. [6] determined that right and left atrial stimulation resulted in a similar AVN refractory period. In contrast to the others, Josephson [5] found site-dependent differences in the refractory periods of the fast and slow pathways. The refractory period of the fast pathway was shorter during CS than HRA stimulation. Also, Batsford et al. [2] documented that the refractory period of the AV node was shorter during CS stimulation. We observed that HRA and CS pacing generally resulted in the similar AVN function. There were no significant site-dependent differences in the ERP of the AV node as well as the SP in patients with discontinuous conduction curves. However, the ERP of the FP was shorter during CS than during HRA stimulation.

Gonzalez et al. [3] and Leon et al. [6] determined that right and left atrial stimulation resulted in similar WBCL. In contrast, Batsford et al. [2] and Aranda et al. [1] found that WBCL was shorter during CS pacing. We also observed that anterograde WBCL was significantly shorter during CS than

HRA stimulation, with the exception of the subgroup with continuous AVN conduction.

The effect of isoprenaline administration

The effect of isoprenaline on both the fast and slow AVN pathway varies interindividually and cannot be predicted. Anterograde and retrograde conduction times over the fast and slow pathway and their ERPs could be prolonged, shortened, or not changed, and as a consequence, induction of AVNRT could be facilitated, prevented, or not altered. AVNRT induction only during isoprenaline administration could be seen in between 19.8% and 41.6% of cases [23]. In our study it was observed in 34%. The effect of isoprenaline administration on the leftward AVN approaches is almost unknown.

We found site-dependent differences in the ease of induction of dual AVN pathways. It was harder to induce them from the CS when isoprenaline was administered. The site-dependent ease of AVNRT induction was not altered. It was still harder to reveal AVNRT from the CS.

However, site-dependent differences in the AH conduction times (shorter with CS pacing) as well as the ERP of the AV node and the SP (no difference) remained unchanged, and there was a distinct effect of isoprenaline infusion on AVN properties. Site-dependent differences in the ERP of the FP as well as antegrade WBCL were not noticed anymore.

Clinical implications

The study addresses the important problem of non-inducibility of clinically manifest paroxysmal supraventricular tachycardia (pSVT) during EPS. Of the 92 patients only 70% were inducible by SET from both atrial sites. 22% were only inducible from the HRA, while 8% were only inducible from the proximal CS catheter. Transferring this into the clinical context, an AVNRT would be missed in 22 out of 100 patients if CS stimulation only were used, and an AVNRT would be missed in 8 out of 100 patients if only pacing from the HRA were used. While some studies have addressed the effect of anatomically different pacing sites on AVN conduction physiology [1–8], to the best of our knowledge there is no previous study with relevant patient numbers having systematically addressed the issue of AVNRT induction in correlation to pacing site. The observations presented in our study may thus motivate electrophysiologists to choose an additional pacing site when a clinically documented pSVT cannot be induced during EPS. This additional manoeuvre would not expose the patient to increased risk and would be cost and almost time neutral and would increase the number of patients who could be effectively treated for AVNRT by RF ablation.

Limitations of the study

We could not exclude variations of electrophysiological properties of the AV junction during pacing and AVNRT due to variability and susceptibility of the autonomic tone.

Nearly one third of the patients had evidence of mild structural heart disease that could have caused slightly anatomical changes in the region of the AV junction not precisely visible during the standard echocardiography. We also could not exclude changes in the functional properties of the AV conduction system in these patients.

The applied pacing protocol could potentially not reveal all cases of dual AVN pathways and AVNRT from a particular pacing site. If SET and IP fail multiple extrastimuli, different driving rhythm, pharmacological provocation, or finally another pacing site could presumably uncover these phenomena [5, 11, 12].

CONCLUSIONS

This study shows that not only rightward but also leftward AVN extensions are a regular feature of AV node in the typical AVNRT population. Their different electrophysiological properties lead to variation in the demonstration of discontinuous AVN conduction and AVNRT during right and left atrial pacing. That is why, in any case, it is important to stimulate from both the left and right atrium. Despite the observation that the left AVN extensions could compose the entry point to the reentrant circuit, there is no evidence that they constitute a critical component of sustained typical AVNRT. In such cases standard RA ablation will render AVNRT non-inducible.

Conflict of interest: *none declared*

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Wpływ miejsca stymulacji przedsionka na właściwości elektrofizjologiczne łącza przedsionkowo-komorowego i powstawanie węzłowych pobudzeń nawrotnych u pacjentów z typowym częstoskurczem węzłowym nawrotnym

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Streszczenie

Wstęp: Wyniki badań klinicznych przeprowadzonych u ludzi wykazały, że miejsce stymulacji przedsionka wpływa na czas przewodzenia przez węzeł przedsionkowo-komorowy (AV), jego okres refrakcji, ujawnienie dualizmu przewodzenia i pojawienie się węzłowych pobudzeń nawrotnych. W badaniach tych często uzyskiwano jednak sprzeczne rezultaty. Ponadto wśród włączonych pacjentów u mniejszości rozpoznawano częstoskurcz węzłowy nawrotny (AVNRT).

Cel: Celem pracy była ocena wpływu stymulacji prawo- i lewoprzedsionkowej na właściwości elektrofizjologiczne łącza AV w populacji pacjentów z typowym AVNRT.

Metody: Do badania włączono 92 kolejnych pacjentów z typowym AVNRT. Stymulacje przeprowadzano z górnej części prawego przedsionka (HRA) oraz lewego przedsionka poprzez proksymalny odcinek zatoki wieńcowej (CS).

Wyniki: Stymulacja zarówno z obszaru HRA, jak i CS może prowadzić do ujawnienia dualizmu przewodzenia przez węzeł AV i prowokacji AVNRT. Nie stwierdzono zależności między miejscem stymulacji a łatwością w ujawnieniu dualizmu przewodzenia AV, rejestrując zależną od miejsca stymulacji zmienność występowania tego zjawiska, chociaż AVNRT łatwiej było wywołać stymulacją z HRA. Podczas stymulacji CS to dostępy lewostronne, a nie prawostronne stanowiły punkt wejścia do węzła AV, z powodu istotnie krótszego odstępu AH niż podczas stymulacji HRA. Przewodzenie lewostronnym przedłużeniem węzła AV może inicjować częstoskurcz z istotnie krótszym czasem AH w porównaniu z przewodzeniem prawostronnym przedłużeniem węzła AV, jakkolwiek odstęp AH podczas AVNRT i długość cyklu arytmii nie różnią się w obu przypadkach istotnie.

Wnioski: Obecność prawo- i lewostronnych przedłużeń węzła AV stanowi jego stałą cechę. Ich odmienne właściwości elektrofizjologiczne prowadzą do zmienności w ujawnieniu dualizmu przewodzenia AV i prowokacji AVNRT podczas stymulacji prawego i lewego przedsionka. Mimo że lewostronne przedłużenia węzła AV mogą stanowić punkt wejścia do pętli re-entry, nie ma dowodów na to, iż stanowią krytyczny element utrwalonego typowego AVNRT.

Słowa kluczowe: węzeł przedsionkowo-komorowy, przedłużenia węzła przedsionkowo-komorowego, częstoskurcz węzłowy nawrotny

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