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Original article

Prevalence and predictors of ventriculo-atrial conduction in structurally normal hearts

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ABSTRACT

Background: The prevalence of ventriculo-atrial (VA) conduction varies from 20% to 90%, depending on the population studied (Militianu et al., 1997; Inoue et al., 1985; Kazmierczak et al., 1993; Ciemniewski et al., 1990; Hayes and Furman, 1983; Westveer et al., 1984). This wide range is mostly based on studies done in patients with implanted devices or impaired atrioventricular conduction. However, the prevalence of VA conduction in structurally normal heart has not been well documented till date.
Objective: To study the prevalence and identify predictors of retrograde conduction via the His-Purkinje system and AV node in structurally normal hearts.
Methods: We included 54 consecutive adults without structural heart disease who underwent electrophysiological (EP) study for various tachycardias. The basic parameters including PR, AH and HV intervals, atrioventricular Wenckebach point (AVWP) and anterograde effective refractory period (ERP) of atrioventricular node (AVNERP), were measured after ablation. The VA conduction was assessed basally and if absent, after isoprenaline. The VA Wenckebach point (VAWP) and retrograde ERP(VAERP) were recorded in patients showing VA conduction.
Results: The mean age was 37.1 ±12.6years. Twenty five (46%) of the patients were men. VA conduction was present in 30 (55%) patients at baseline. Of the remaining 24 patients, 18 (34%) showed VA conduction after isoprenaline. Only 6 (11%) patients failed to reveal VA conduction even after adequate response to isoprenaline. Amongst all clinical and EP variables analysed, only the HV interval was shorter ($p < 0.01$) in patients with VA conduction.
Conclusion: In structurally normal hearts, VA conduction was present at baseline in 55% of patients. Isoprenaline unmasked VA conduction in an additional 34% of the subjects. The HV interval was longer in patients without VA conduction.

1. Introduction

The prevalence of ventriculoatrial (VA) conduction varies widely from nearly 20% to 90%, depending on the population studied.[1–6] Most of these studies were performed in patients with implanted cardioverter-defibrillators or pacemakers. There is scanty data on the prevalence of VA conduction in structurally normal hearts with normal atrioventricular conduction. Hence the current study was designed to address this issue.

2. Method

Between December 2013 and December 2015, adults scheduled for electrophysiological study (EPS) were included for this study. Patients with structural heart disease, atrioventricular nodal reentrant tachycardia (AVNRT) and septal accessory pathways (APs) were excluded.

The study was approved by the Institutional Bioethics Committee, and informed written consent was obtained from all the patients. The electrophysiological study was performed with CardioTek, EP-Tracer software version 0.85 under local anaesthesia. No general anesthesia or sedation used. All the patients had discontinued anti-arrhythmic drugs for at least 5 half-lives. Using the femoral vein approach, a quadripolar electrode catheter (Supreme Electrophysiology Diagnostic Catheter, St Jude Medical) was inserted in the His bundle area and a decapolar catheter

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Table 1
Electrophysiologic parameters in patients with and without VA conduction.

Parameters(ms)	VA conduction present; N = 48 mean ± SD	VAconduction absent; N = 6 mean ± SD	p value
PR	133.5 ± 16.4	139.2 ± 22.1	0.56
QRS	85.1 ± 17.0	81 ± 12.4	0.49
AH	65.1 ± 14.2	70.3 ± 20.3	0.56
HV	36.9 ± 10.1	47 ± 7.5	0.01
AVWP	293.8 ± 57.7	303 ± 35.6	0.5
AVNERP	260 ± 37.8	273.3 ± 35	0.41

(Inquiry steerable diagnostic catheter, St Jude Medical) was inserted in the coronary sinus (CS). Intra-cardiac conduction intervals were recorded at a paper speed of 100–300 mm/s simultaneously with ECG data from 12 surface leads. Catheter ablation was performed in the same sitting after the initial EP study. Post-ablation basic intervals were measured. The presence of VA conduction during ventricular pacing at a cycle length of 400–600 ms was evaluated.[7] The EP study was repeated during isoprenaline infusion (1–3 μg/min) if VA conduction was absent basally. Adequate response to isoprenaline was considered as a rise in heart rate by 25% above the baseline.[8] Incremental atrial and ventricular pacing was commenced at rates just above the sinus rate and continued until AtrioventricularWenckebach point (AVWP) or VA Wenckebach point (VAWP) were achieved or till the paced cycle length reached 200 ms. The extra stimulus technique was used to measure the antegrade effective refractory period (ERP) of the AV node (AVNERP), retrograde ERP (VAERP) and ventricular effective refractory period (VERP).[9] Retrograde pathway was detected as fast pathway if the His bundle tracing showed earlier atrial activation than CS channels and slow pathway if the His bundle tracing showed atrial activation wave later than that in the CS channels.

Statistical analysis: Fisher's exact test was applied to test the relationship of categorised independent and dependent variables. For quantitative data, the Mean, Standard deviation, Standard error and 95% Confidence intervals were calculated. The unpaired 't' test was used to compare quantitative variables individually with VA Conduction. Stata SE 13.1 was used to analyse data. A p value (significance) of <0.05 was deemed statistically significant and $p < 0.01$ as highly significant.

3. Results

Of 121 patients with various arrhythmias who underwent EP study, 54 patients met the inclusion criteria. The mean age was 37.1 ± 12.6 years, ranging from 15 to 66 years; 25 (46%) patients were men. Forty-seven patients had an AP, 4 patients had fascicular VT, 1 patient had atrial tachycardia and 2 had outflow tract VT. The overall prevalence of VA conduction was in 30 (55%) patients at baseline. Of the remaining 24 patients, 18 (34%) showed VA conduction after isoprenaline. Only 6 (11%) patients failed to reveal VA conduction even after isoprenaline. 25 patients were tested for the conduction through slow or fast pathway, of which 22 (88%) showed retrograde conduction through fast pathway, 2 through slow pathway and 1 with both fast and slow pathway. There was no significant difference between the VA conductive and VA non-conductive patients in terms of age and gender.

Of the variables studied, only the HV interval was significantly shorter (36.9 ± 10.1 vs 47 ± 7.5 ms, $p < 0.01$) in patients with VA conduction (Table 1). Most parameters in patients with baseline VA conduction were similar to those having VA conduction only after isoprenaline (Table 2); only the QRS duration was significantly longer in patients with baseline VA conduction(88.3 ± 20.1vs79.7 ± 8.2 ms, $p = 0.04$).

4. Discussion

The prevalence of VA conduction in this study was 55% at baseline, which increased to 89% with isoprenaline. This finding was different from the studies by Dehghani et al. and Westveer et al., in which the overall prevalence of VA conduction was 38.9% and 40% respectively.[6,10] This difference could be attributed to the difference in study population, as almost all patients included in the quoted studies had structural heart disease. The prevalence of VA conduction in our study was similar to the Goldreyer et al. study which showed VA conduction in 23 of 26 patients with normal AV conduction.[11] In that study, normal AV conduction was defined as PR interval <200, irrespective of the structural abnormality of the heart.

Our study did not find any correlation between VA conduction and clinical parameters like age and gender, which is in agreement to the Westveer study; however, Dehghani et al. reported that VA conduction was more prevalent in men.[6,10] Several electrophysiological parameters in the study of Dehghani et al. were prolonged compared to our study; this could again be because they had a population of ICD recipients.[10] They found the PR interval, AH interval and AVNERP to be significantly longer in patients with absent VA conduction. As against this, our study did not find PR, AH and AVNERP as predictors of VA conduction; the HV interval was found to be longer by us in patients without VA conduction. But the mechanism for this is difficult to explain.

The findings of our study add to core knowledge and understanding of VA conduction physiology. Moreover, the presence of VA conduction in 89% of structurally normal hearts

Table 2
Parameters in patients with VA conduction at baseline and only after isoprenaline.

Parameters(ms)	VA conduction(+) at baseline N = 30 mean ± SD	VAconduction (+) after isoprenaline N = 18. mean ± SD	p value
PR	132.7 ± 15.8	134.7 ± 17.7	0.69
QRS	88.3 ± 20.1	79.7 ± 8.2	0.04
AH	64.4 ± 14.8	66.2 ± 13.5	0.66
HV	37.2 ± 11.9	36.3 ± 5.7	0.73
AVNERP	258.3 ± 30.6	262.7 ± 48.5	0.72
AVWP	294.5 ± 53.9	292.7 ± 64.9	0.91
VAERP	296.8 ± 60.8	274.4 ± 72.1	0.31
VAWP	320.9 ± 84.3	331.9 ± 71.9	0.67