Ventriculo-atrial Conduction in Patients with Normal and Impaired Atrio-ventricular Conduction

Tomoo Inoue, M.D., Katsuya Kobayashi, M.D., and Hisashi Fukuzaki, M.D.

Summary

Ventriculo-atrial (VA) conduction was studied in 133 patients with various kinds of arrhythmias using intracardiac electrograms and programmed stimulation. One-to-one VA conduction was observed during RV pacing at the rate just above the sinus rate in 6 of 31 patients (19.4%) with advanced AV block, in 7 of 26 patients (26.9%) with impaired AV nodal conduction, in 25 of 71 patients (35.2%) with normal AV nodal conduction and 3 of 5 patients (60%) with enhanced AV nodal conduction. However, the differences between these groups were not significant. There was no significant difference in either the AH block rate during RA pacing or the antegrade functional refractory period (FRP) of the AV node in patients with or without VA conduction, and the VA block rate during RV pacing was not significantly correlated with the AH block rate or the FRP of the AV node. VA conduction time (S-HRA) also showed no significant differences between these groups. The mean VA conduction time during RV pacing at rates of 60 to 80 bpm was 208±87 msec, ranging from 100 to 395 msec. In conclusion, AV conduction disturbances may influence VA conduction, but VA conduction cannot be predicted from antegrade conductivity.

Additional Indexing Words:
Ventriculo-atrial conduction time Atrio-ventricular conduction disturbance Atrio-ventricular block Sick sinus syndrome Enhanced AV nodal conduction

The presence or absence of ventriculo-atrial (VA) conduction is not a significant problem in normal subjects. However, VA conduction is observed clinically in patients with ventricular tachycardia or occasionally during ventricular pacing, as shown in Fig. 1. The existence of VA conduc-
Fig. 1. Ventriculo-atrial conduction observed in patients with ventricular tachycardia and during ventricular pacing.

tion causes a problem of differential diagnosis of ventricular arrhythmias from supraventricular arrhythmias, and VA conduction can unfavorably affect hemodynamics in patients with ventricular pacing.\textsuperscript{1-3)} Furthermore, the development of physiologic pacing in recent years has introduced the problem of "endless loop tachycardia" in patients with atrial synchronous ventricular pacing.\textsuperscript{4-6)}
Electrophysiologic studies using intracardiac electrograms and programmed stimulation have provided useful clinical information about atrioventricular (AV) conduction. Although a substantial number of papers have examined AV conduction, there are relatively few reports of systematic studies of VA conduction. The present study was undertaken to evaluate VA conduction in patients with various kinds of arrhythmias and compare retrograde conductivity with antegrade conductivity.

**SUBJECTS AND METHODS**

VA conduction was evaluated in 133 patients who underwent electrophysiologic study to assess arrhythmias or symptoms of palpitation, syncope or dizziness. Patients with manifest and concealed WPW syndrome and AV nodal re-entrant tachycardia, who had anomalous pathways as well as the usual AV conduction system, were excluded from this study. Patients were 91 males and 42 females, aged 10 to 81 years (mean 53.4 ± 18.9). Patients were divided into 4 groups according to clinical diagnosis and AV conductivity after incremental right atrial (RA) pacing. Group I consisted of 31 patients with advanced AV block, including 21 patients with complete AV block. Group II consisted of 26 patients with impaired AV nodal conduction who showed AH Wenckebach block during RA pacing at the rate lower than 130 bpm, including 16 patients with I° or II° Wenckebach type AV block. Group III had 71 patients with normal AV nodal conduction who showed AH block at the rate from 130 to 200 bpm, including 24 patients with infranodal abnormalities, such as HV prolongation, bundle branch block, hemiblock and intraventricular conduction disturbances associated with myocardial infarction, cardiomyopathy, or other organic heart diseases and 47 patients with normal infranodal conduction. Finally, group IV consisted of 5 patients with enhanced AV nodal conduction who showed 1:1 AV conduction until 200 bpm. Nine and 22 patients with sick sinus syndrome belonged to groups II and III, respectively.

All patients were studied in the unmedicated, postabsorptive state in the cardiac catheterization laboratory. At least three multipolar electrode catheters were percutaneously introduced through the femoral vein under fluoroscopic guidance. One quadripolar catheter was positioned in the high right atrium near the sinus node for RA pacing and recording of high right atrial electrogram (HRA). One bipolar catheter was positioned in the right ventricular (RV) apex for RV pacing and the other bipolar catheter was positioned across the tricuspid valve for recording the His bundle electrogram. Intracardiac electrograms and surface electrograms were simul-
taneously recorded at a paper speed of 100 mm/sec.

The site of block was determined in patients with AV block and the AH interval and HV interval were measured in other patients from the His bundle electrogram in the control state. Incremental RV pacing was initiated using a programmable stimulator at the rate just above the sinus rate (mean±SD, 77.9±17.4 bpm). The presence of VA conduction was determined when atrial electrograms, with low to high sequence, followed paced ventricular electrograms constantly. When VA conduction was present, the VA conduction time was measured from the onset of spike of RV pacing to the beginning of the HRA deflection (S-HRA) and the pacing rate was increased by 10 bpm until second degree VA block occurred (VA block rate).

In group II, III and IV patients, incremental RA pacing was performed in the same manner. The pacing rate inducing AH Wenckebach block (AH block rate) was determined. The RA extrastimulus technique with a mean basic cycle length of 702±103 msec was also performed and the functional refractory period (FRP) of the AV node was measured.

Results are presented as the mean ± standard deviation. Statistical significance of the results was tested by one-way analysis of variance and unpaired t-test.

Results

Presence of VA conduction:

Both AV conduction and VA conduction in each group are summarized in Table I. The antegrade FRP of the AV node in groups II, III and IV were 642±120 msec, 422±150 msec and 329±57 msec, respectively, and there were significant differences between these groups. VA conduction was present in 6 of 31 patients (19.4%) with advanced AV block, 7 of 26 patients (26.9%) with impaired AV nodal conduction, 25 of 71 patients (35.2%) with normal AV nodal conduction and 3 of 5 (60.0%) with enhanced AV nodal conduction. The better the AV conductivity of the group was, the higher the positive rate of VA conduction tended to be. However, the differences in occurrence between these groups were not statistically significant.

In group I, 4 of 21 patients (19.0%) with complete AV block showed VA conduction. VA conduction was present in 1 of 4 patients with AH block, none of 7 patients with intra-His bundle block and 5 of 20 with HV block. In group II, VA conduction was present in only 2 of 16 patients with I° and/or II° Wenckeback AV block; the sites of block were all in the AV node (Table II). In group III, VA conduction was present in 8 of 24 patients (33.3%) with infranodal conduction disturbances and 17 of 47 (36.1%) with
normal infranodal conduction (n.s.). In 31 patients with sick sinus syndrome, 8 patients (25.8%) showed VA conduction: 2 patients (22.2%) and 6 patients (27.3%) belonged to groups II and III, respectively (n.s.) (Table II).

*AV and VA conductivity:*

Antegrade and retrograde conductivity were compared in group II, III and IV patients. The antegrade AH block rate and FRP of the AV node were 148±33 bpm (n=33) and 464±130 msec (n=31) in patients with VA conduction, and 140±35 bpm (n=64) and 479±119 msec (n=66) in patients without VA conduction. The differences were not statistically significant (Table III). In patients with VA conduction, the VA block rate was not significantly correlated with either AH block rate or FRP of the AV node (Fig. 2).
VA conduction time:

The VA conduction times in group I to IV patients with VA conduction were 193±49 msec, 208±94 msec, 213±76 msec and 150±240 msec, respectively (n.s.). When RV pacing rate was limited from 60 to 80 bpm, the VA conduction time in groups I to III were 193±49 (150-285) msec (n=6), 213±112 (125-395) msec (n=5) and 216±98 (100-375) msec (n=10), respectively (n.s.). The distribution of VA conduction time in these 21 patients is shown in Fig. 3 and the mean VA conduction time was 208±87 msec.

Fig. 2. Correlation of VA block rate with AH block rate and functional refractory period (FRP) of the AV node in patients with VA conduction.

Fig. 3. VA conduction time (S-HRA) during RV pacing at the rate of 60 to 80 bpm.
DISCUSSION

The existence of VA conduction in patients with implanted pacemakers for bradyarrhythmias cannot be clinically disregarded because of unfavorable hemodynamic effects and pacemaker mediated tachycardia.1)-6)

As previously reported,3),8),10)-15) VA conduction was observed in 8-40% of patients with high degree or complete AV block, in 60-77% with sick sinus syndrome and in 65-90% with normal AV conduction. In this study, subjects were divided into 4 groups according to AV conductivity. The incidence of VA conduction was relatively low, but the incidence of VA conduction tended to be higher in groups with higher AV conductivity.

The criteria for enhanced AV nodal conduction proposed by Gallager et al16) were (1) AH interval ≤60 msec during sinus rhythm, (2) 1:1 conduction from atrium to His bundle at cycle length ≤300 msec during RA pacing, (3) AH prolongation ≤100 msec between sinus cycle length and a cycle length of 300 msec. In this study, 1 of 5 patients in group IV satisfied all these criteria and the other 4 patients satisfied 2 of 3 criteria. Even in these 5 patients, 2 patients did not display VA conduction, whereas VA conduction was observed in 19% of cases of complete AV block. Furthermore, VA conduction was observed in 19% of cases of complete AV block. Furthermore, VA conductivity was not correlated with AV conductivity in this study. Thus, VA conduction may exist independent of antegrade conductivity.

It was previously reported that the site of retrograde block is generally in the AV node of patients without AV conduction disturbances.8) This was supported by the fact that VA block was not observed in the infranodal conduction system at a high rate of RV pacing in patients with AV nodal re-entrant tachycardia who had retrograde pathways in the AV node. In the present study, it was noted that the incidence of VA conduction was extremely low, when common AV pathways (AV node, His bundle) were severely injured (in patients with advanced AH, BH block and I° and/or II° AV nodal block). Organic or functional disturbances in a part of the AV conduction system may impair retrograde conduction in the same location.

VA conduction time was not correlated with AV conductivity. In many patients, it did not exceed 200 msec, but in some patients it was approximately 400 msec at the usual pacing rate. These results were essentially same as the report by Hayes et al.14) To prevent endless loop tachycardia in patients with atrial synchronous ventricular pacing units (VAT, VDD or DDD mode), the atrial refractory period of the pacemaker would be usually set about 350 msec (200 msec + AV delay) and needed to be programmed up to 500 msec or more. In conclusion, the existence of antegrade conduction disturbances
may influence VA conduction, but VA conduction cannot be predicted from antegrade conductivity. Thus, it is recommended that VA conduction and VA conduction times should be evaluated in each patient with bradyarrhythmia who will be a candidate for pacemaker implantation.

REFERENCES

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