MULTIPLE FORMS OF ATYPICAL ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA WITH DIFFERENT RIGHT- AND LEFT-SIDED RETROGRADE SLOW PATHWAYS

SHORT TITLE: MULTIPLE DIFFERENT RETROGRADE SLOW PATHWAYS IN ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA

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ABSTRACT

A 56-year-old man was admitted for the treatment of supraventricular tachycardia. After successful ablation of the left concealed accessory pathway, 4 forms of fast-slow forms of atrioventricular nodal reentrant tachycardia associated with different right- and left-sided retrograde slow pathways were induced. The locations of retrograde slow pathway were observed at the left inferior paraseptum, left mid-septum, right inferior paraseptum and coronary sinus ostium, respectively. These retrograde slow pathways formed the integral limb of each tachycardia because conduction block of each slow pathway by catheter ablation was associated with the termination of tachycardia or abrupt change in the atrial activation sequence.

Key words: atrioventricular node, ablation, electrophysiology.

INTRODUCTION

Radiofrequency catheter ablation of the slow pathway is an established treatment for atrioventricular nodal reentrant tachycardia (AVNRT) (1). Slow pathway conduction can be eliminated by radiofrequency energy application to the right inferior paraseptum in most slow-fast and fast-slow forms of AVNRT; however, it has been reported that standard right-sided ablation was ineffective for ablation of the slow pathway and required left-sided catheter ablation in rare cases with the slow-fast and fast-slow forms of AVNRT (2-4).We report a case of multiple fast-slow forms of AVNRT associated with multiple different right- and left-sided retrograde slow pathways that were successfully eliminated by right- and left-sided approaches.

CASE REPORT

A 56-year-old man was admitted to our hospital for catheter ablation for paroxysmal supraventricular tachycardia. No structural heart disease was detected on physical

examination or transthoracic echocardiography. A 12-lead surface electrocardiogram during sinus rhythm showed no abnormal findings. Electrophysiological study demonstrated AV reciprocating tachycardia (AVRT) using a left concealed accessory pathway with a cycle length of 350 msec (Figure 1, AVRT) (Figure 2a). Tachycardia was reset by single right ventricular extrastimulation delivered during the refractory period of His bundle and showed a V-A-V pattern following entrainment by right ventricular stimulation. Atrial mapping was then performed using a 7-Fr large tip deflectable quadripolar electrode catheter, which was advanced into the left atrium by the transseptal approach. The earliest atrial activation during AVRT was observed at the superolateral mitral annulus (2:30 time position in the left anterior oblique view) (Figure 1, site K in the right panel) (Figure 2a). Radiofrequency energy application to the earliest atrial activation site successfully eliminated ventriculo-atrial conduction via the concealed left superolateral accessory pathway. Subsequently, the fast-slow form of AVNRT was induced (Figure 1, AVNRT-1) (Figure 3, AVNRT-1). A diagnosis of AVNRT was made by previously established criteria (5). Mapping of the right and left atrium revealed that the earliest atrial activation was at the left inferior paraseptum (Figure 1, site 1 in the right panel) (Figure 3, AVNRT-1). Application of radiofrequency energy to the left inferior paraseptum terminated tachycardia immediately after the onset of energy application (Figure 2b), suggesting that the retrograde slow pathway at this ablation site was an integral limb of the circuit. Although AVNRT-1 was rendered non-inducible after energy application to the left inferior paraseptum, a different fast–slow form of AVNRT (AVNRT-2) was then induced (Figure 1, AVNRT-2) (Figure 3, AVNRT-2). Intra-atrial mapping revealed that the earliest retrograde atrial activation site during AVNRT-2 was at the left mid-septum (Figure 1, site 2 in the right panel) (Figure 3, AVNRT-2). As shown in Figures 1 and 3, the morphology of the P wave of the surface 12-lead electrocardiogram and the atrial activation sequence on intracardiac electrograms in AVNRT-2 differed from those in AVNRT-1 (Figures 1 and 3). The negative deflection of P wave in leads II, III and aVF during AVNRT-2 was slightly shallower than those in AVNRT-1 and the positive deflection of the P wave in lead V1 during AVNRT-2 was

lower than that during AVNRT-1 (Figure 1). Also, the intra-atrial activation interval between CS 9-10 and HRA 7-8 during AVNRT-2 was shorter than during AVNRT-1 (45 vs. 55 msec) (Figure 3). The atrial electrogram at CS 7-8 was observed earlier than that at CS 9-10 during AVNRT-1. However, the atrial electrogram at CS 9-10 was observed earlier than that at CS 7-8 during AVNRT-2. In addition, atrial activation time in the CS during AVNRT-1 was shorter than that during AVNRT-2 (13 vs. 16 msec) (Figure 3). Radiofrequency energy application to the left mid-septum (site 2 in Figure 1) during AVNRT-2 produced an abrupt change in the cycle length and intra-cardiac activation sequence (Figure 2c). The tachycardia cycle length was abruptly prolonged from 360 to 390 msec accompanied by delayed activation of the atrial electrogram at the ablation site, suggesting a shift in the retrograde limb of tachycardia and transition of the tachycardia circuit to a different form of tachycardia by ablation (Figure 2c). After elimination of AVNRT-2, we remapped the left atrium during subsequent fast–slow AVNRT (AVNRT-3); however, the earliest atrial electrogram was not in the left atrium, but was observed at the right inferior paraseptum during AVNRT-3 (Figure 1, site 3 in the right panel) (Figure 3, AVNRT-3). The morphology of the P wave on the surface 12-lead electrocardiogram and the atrial activation sequence on intracardiac electrograms in AVNRT-3 were different from those in AVNRT-1 and -2 (Figures 1 and 3). Although the atrial electrograms at CS 9-10 during AVNRT-1 and -2 were observed 55 and 45 msec earlier than those at HRA 7-8, the atrial electrogram at CS 9-10 was observed 70 msec earlier than that at HRA 7-8 during AVNRT-3 (Figure 3). In addition, atrial activation time in the CS during AVNRT-3 was longer than those during AVNRT-1 and -2 (32 vs. 13 and 16 msec, respectively) (Figure 3). Furthermore, atrial activation sequence from HRA 1-2 to 9-10 during AVNRT-3 was different from that during AVNRT-1 and -2 (Figure 3). Subsequent application of radiofrequency energy to site 3 terminated AVNRT-3 and eliminated retrograde slow pathway conduction at site 3; however, another different fast–slow AVNRT (AVNRT-4) was induced (Figures 1 and 3). The earliest atrial electrogram was observed at the coronary sinus ostium during AVNRT-4 (Figure 1, site 4 in the right panel). P wave morphology on the surface

12-lead electrocardiogram and intracardiac activation sequence during AVNRT-4 differed from those during AVNRT-1, -2 and -3 (Figures 1 and 3). Also, the atrial activetion time in the CS during AVNRT 4 differed from those during AVNRT-1, -2 and -3 (Figure 3). Radiofrequency energy application to the coronary sinus ostium (site 4 in Figure 1) terminated AVNRT-4 and eliminated retrograde slow pathway conduction. After this energy application, tachycardia was rendered non-inducible.

DISCUSSION

Our case presented multiple forms of fast-slow AVNRT using different multiple right- and left-sided retrograde slow pathways. Indeed, change in the retrograde slow pathway conduction via the retrograde right- and left-sided slow pathways was associated with the change in the intracardiac activation sequence. These different retrograde slow pathways formed the integral limb of each tachycardia. This was confirmed by the finding that the conduction block of each slow pathway by radiofrequency energy application was associated with the termination of tachycardia or shift in the atrial activation sequence accompanied by a change in the tachycardia cycle length. Of interest, the difference in the tachycardia cycle length among tachycardias was mainly due to the difference in the HA interval. This may reflect the different retrograde conduction time among different retrograde slow pathways or it might be caused by the change in the autonomic tone.

Our observations confirmed the previous case of the common form of AVNRT, in which the successful ablation site was in the left mitral annulus (2). Left-sided ablation of the retrograde slow pathway in the fast–slow form of AVNRT has also been reported previously (3, 4). However, the successful ablation site of the eccentric atypical AVNRT was in the coronary sinus ostium and, to our knowledge, it has never been shown in the left atrial mitral annulus. Previously, Inoue and Becker provided histologic evidence of both rightward and leftward inferior extensions and they speculated that these extensions may form the substrate of the slow pathway in AVNRT (6). Katritsis et al. demonstrated the presence of the left inferior extensions electrophysiologically (7). Based on these findings, they suggested that a right or left circuit may occur in AVNRT (7). Thus, these multiple extensions might be the substrate of the right- and left-sided retrograde slow pathways observed in our case.

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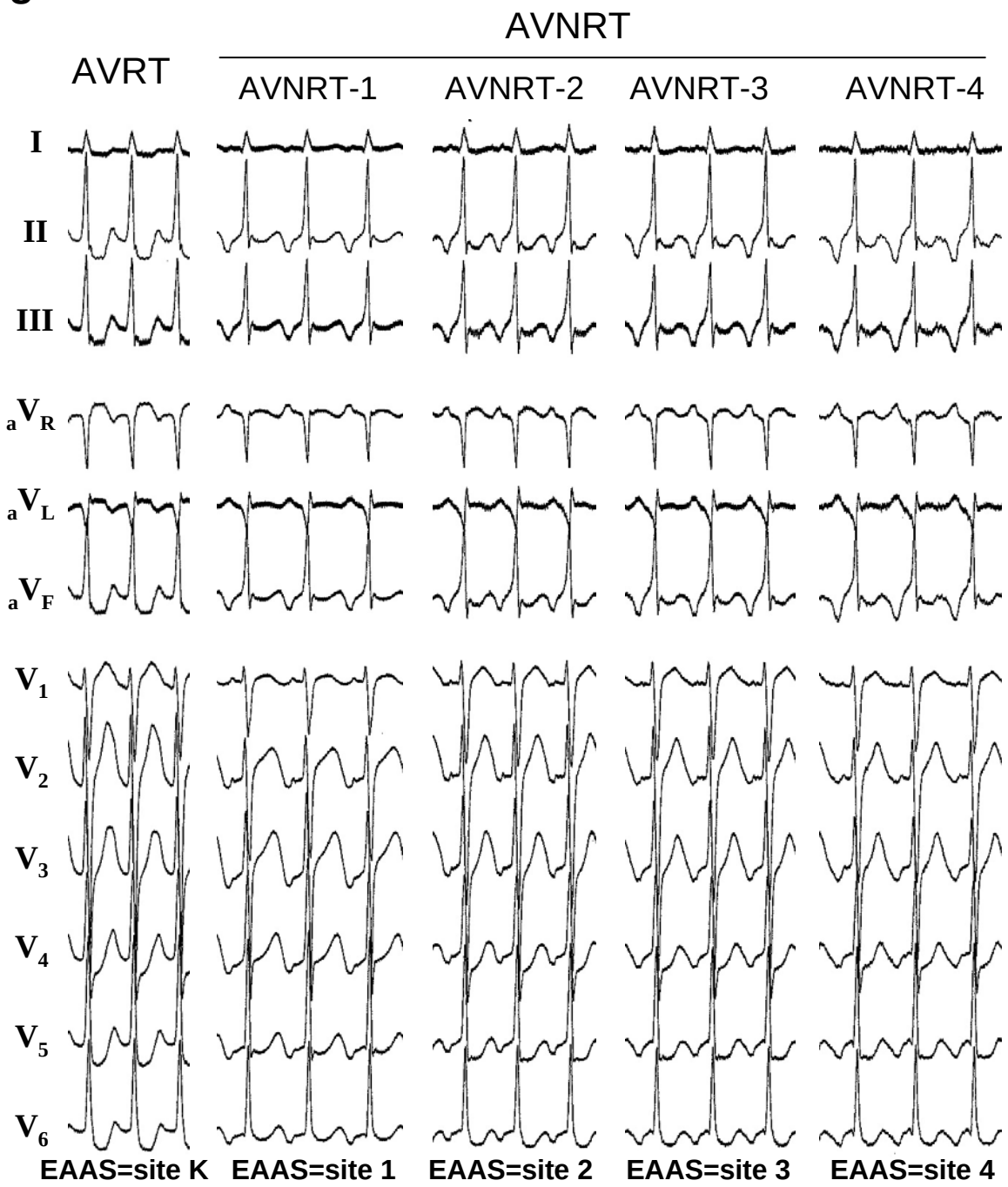
FIGURE LEGENDS

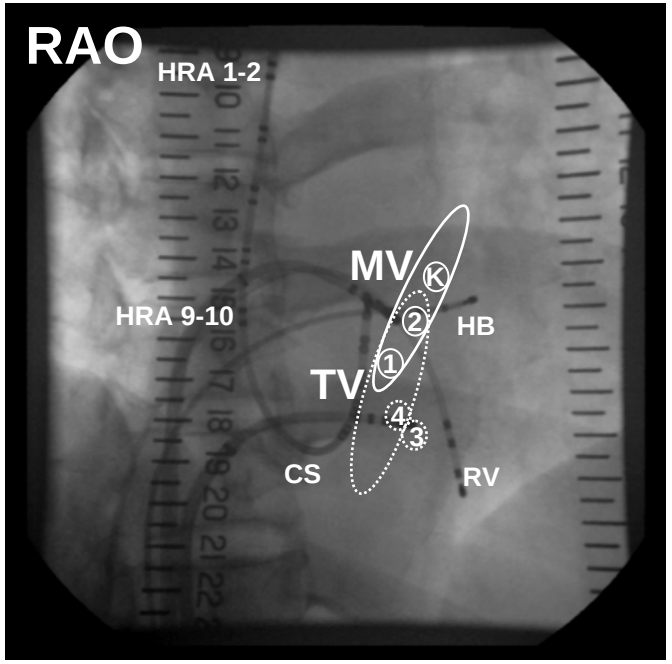
Figure 1. **Left panel:** Surface 12-lead electrocardiograms of each tachycardia. **Right panel:** Location of the earliest atrial activation site (EAAS) in each tachycardia on bi-plane fluoroscopic images. AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reciprocating tachycardia; CS, coronary sinus; HB, His bundle; HRA, high right atrium; LAO, left anterior oblique view; MV, mitral valve; RAO, right anterior oblique view; RV, right ventricle; TV, tricuspid valve.

Figure 2. **Panel a:** Surface electrocardiogram I, II and V1 and intracardiac electrograms during atrioventricular reciprocating tachycardia. The ablation catheter (ABL) is located at the earliest atrial activation site (2:30 time position in the left anterior oblique view; MV 2:30). **Panel b:** recording during AVNRT-1 at the onset of radiofrequency energy application. The ablation catheter (ABL) is located at the left inferior paraseptum, where the earliest atrial electrogram was recorded during AVNRT-1. **Panel c:** Recording during radiofrequency energy application to the mid-septum of the left atrium, where the earliest retrograde electrogram was observed during AVNRT-2. Abbreviations are the same as in Figure 1.

Figure 3. Surface electrocardiograms I, II and V1 and intracardiac electrograms during AVNRT-1, -2, -3 and -4 are shown. The ablation catheter (ABL) is located at the earliest atrial activation site (EAAS) in each tachycardia. See text for discussion. Abbreviations are the same as in Figure 1.

Figure 1


AVNRT
AVRT
AVNRT-1
AVNRT-2
AVNRT-3
AVNRT-4
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
EAAS=site K
EAAS=site 1
EAAS=site 2
EAAS=site 3
EAAS=site 4


RAO
HRA 1-2
MV
K
HRA 9-10
2
HB
1
TV
4
3
CS
RV

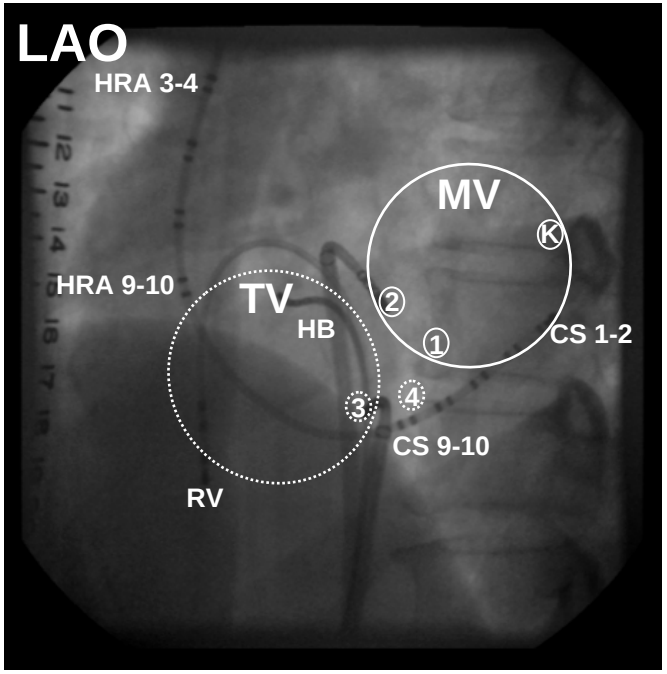

LAO
HRA 3-4
MV
K
HRA 9-10
TV
2
HB
1
CS 1-2
3
4
CS 9-10
RV

Figure 2

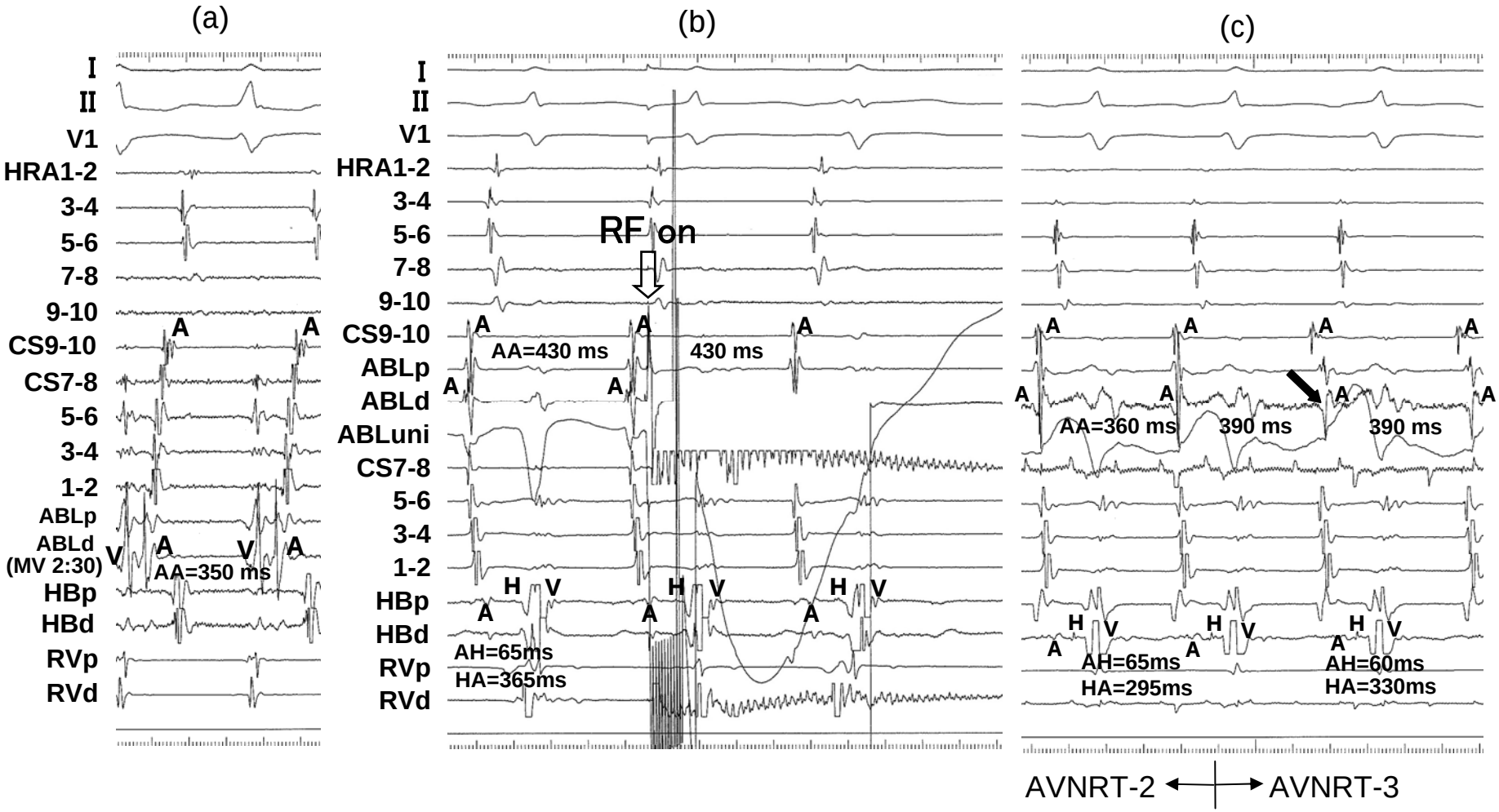
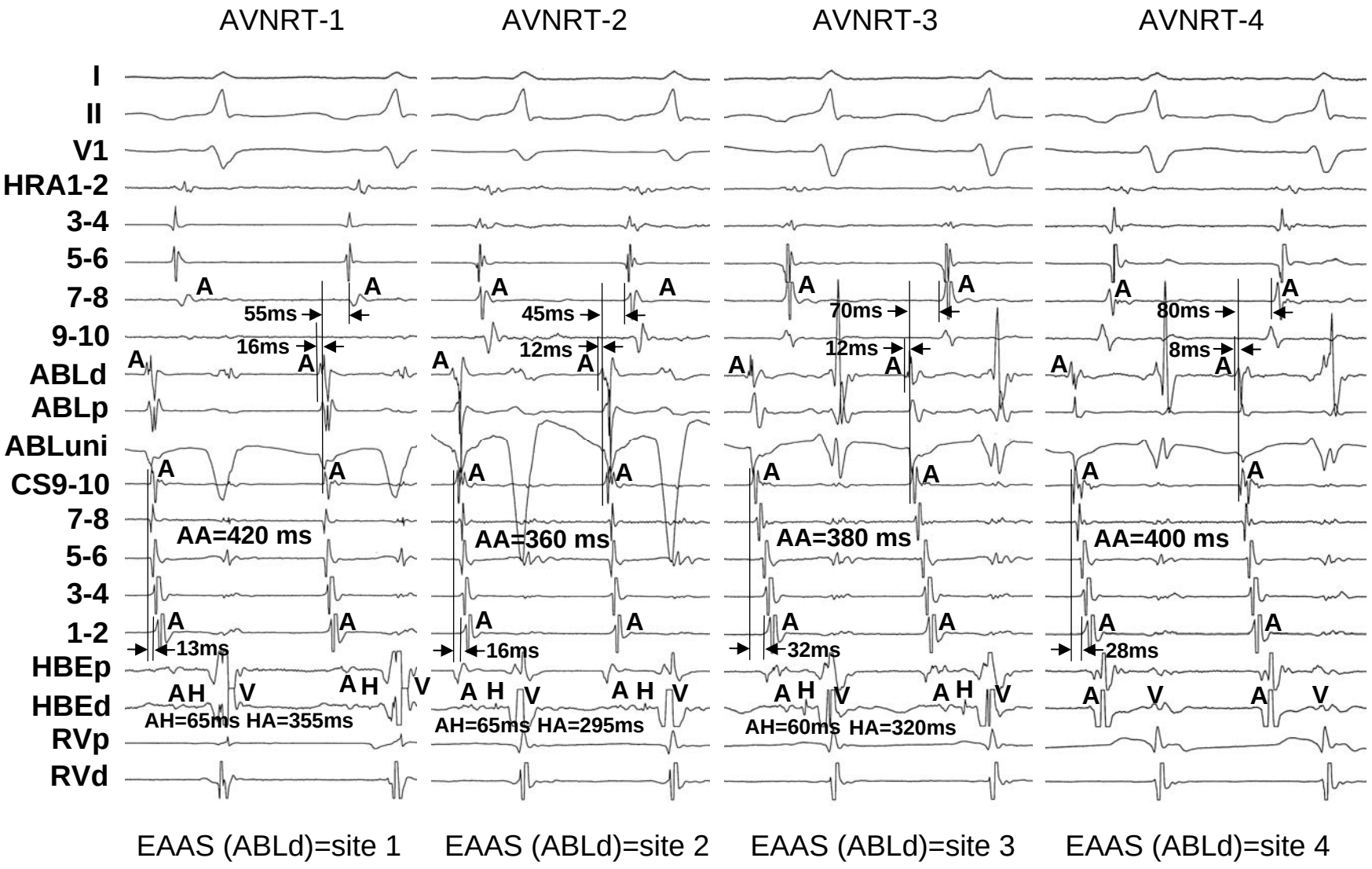

(a)
(b)
(c)
I
II
V1
HRA1-2
3-4
5-6
7-8
9-10
CS9-10
CS7-8
5-6
3-4
1-2
ABLp
ABLd
(MV 2:30)
HBp
HBd
RVp
RVd
A
V
AA=350 ms
RF on
AA=430 ms
430 ms
ABLuni
H
AH=65ms
HA=365ms
AA=360 ms
390 ms
AH=65ms
HA=295ms
AH=60ms
HA=330ms
AVNRT-2
AVNRT-3

Figure 3


AVNRT-1
AVNRT-2
AVNRT-3
AVNRT-4
I
II
V1
HRA1-2
3-4
5-6
7-8
9-10
ABLd
ABLp
ABLuni
CS9-10
7-8
5-6
3-4
1-2
HBEp
HBEd
RVp
RVd
55ms
16ms
AA=420 ms
13ms
AH=65ms HA=355ms
45ms
12ms
AA=360 ms
16ms
AH=65ms HA=295ms
70ms
12ms
AA=380 ms
32ms
AH=60ms HA=320ms
80ms
8ms
AA=400 ms
28ms
EAAS (ABLd)=site 1
EAAS (ABLd)=site 2
EAAS (ABLd)=site 3
EAAS (ABLd)=site 4