# Variation of P-QRS Relation During Atrioventricular Node Reentry Tachycardia

Yasuhiro Taniguchi, MD, San-Jou Yeh, MD, Ming-Shien Wen, MD, Chun-Chieh Wang, MD, Fun-Chung Lin, MD, Delon Wu, MD, FACC

Taipei, Taiwan, and Gunma, Japan

OBJECTIVES	The main objective of this study was to characterize the phenomenon of variation in the P-QRS relation during atrioventricular node reentry tachycardia.
BACKGROUND	Variation of P-QRS relation during tachycardia has been observed occasionally in atrioven- tricular node reentry tachycardia. However, the incidence, the characteristics and the mechanisms of this phenomenon have not been investigated previously.
METHODS	Retrospective analysis was performed in 311 consecutive patients with slow-fast form and 108 patients with atypical or multiple form of atrioventricular node reentry tachycardia to examine whether variation of P-QRS relation with changes in AH, HA and AH/HA (A = atria; H = His bundle) ratio occurred during tachycardia.
RESULTS	A total of 28 patients, 8 with slow-fast and 20 with atypical or multiple tachycardias, were found to manifest this phenomenon. There were 6 males and 22 females, with an average age of $38 \pm 16$ years. In 10 patients, this phenomenon occurred transiently following electrical induction of the tachycardia. In 15 patients, changes in AH, HA and AH/HA ratio were associated with the occurrence of Wenckebach or 2:1 block proximal to the His bundle (H) recording site without interruption of the tachycardia. In nine patients, three with nonsustained tachycardia and six after administration of adenosine triphosphate, this phenomenon was observed at the termination of the tachycardia. This phenomenon was usually accompanied by a mild lengthening of the tachycardia cycle length.
CONCLUSIONS	Variation of P-QRS relation with or without block may occur during atrioventricular node reentry tachycardia, especially in atypical or multiple-form tachycardias. It was postulated that decremental conduction in the distal common pathway, which exists between the distal link of the reentry circuit and the H, is primarily responsible for this phenomenon. (J Am Coll Cardiol 1999;33:376–84) © 1999 by the American College of Cardiology

The common slow-fast form atrioventricular node reentry tachycardia (AVNRT) is characterized by a stable cycle length with a fixed P-QRS relation (1–4). Variation of the P-QRS relation is occasionally seen in patients with atypical or multiple-form AVNRT (5,6). Some of the later tachycardias also manifest with atrioventricular (AV) block without termination of the tachycardia. However, there have been no studies concerning the incidence, the characteristics and the mechanisms of variation of the P-QRS relation with or without AV block during AVNRT. Thus, we retrospectively analyzed a large group of consecutive patients with the common slow-fast form as well as the atypical or multiple form of AVNRT in order to evaluate

the incidence, the characteristics and the mechanisms of variation P-QRS relation during tachycardia. Our results have provided new insight into the understanding of the AV node reentry.

### **MATERIALS AND METHODS**

**Patients.** Retrospective analyses of 311 consecutive patients with slow-fast form and 108 consecutive patients with atypical or multiple-form AVNRT who underwent radio-frequency ablation therapy in this institution were performed to examine whether variation of P-QRS relation with changes in (A = atria; H = His bundle) interval, HA interval and AH/HA ratio occurred during tachycardia. Twenty-eight patients, 8 with the slow-fast form and 20 with the atypical or multiple-form tachycardia, were found to display variation of P-QRS relation during tachycardia. These 28 patients were the study population of this report. There were 6 males and 22 females, with an average age of  $38 \pm 16$  years (range, 8 to 67 years).

From the Second Section of Cardiology, Department of Medicine, Chang Gung Memorial Hospital, Chang Gung University College of Medicine, Taipei, Taiwan. This work was supported in part by grants from the National Science Council (NSC 87-2314-B-182-005 and NSC 87-2314-B-182-059) of the Republic of China, Taipei. Dr. Taniguchi is an International Fellow in Electrophysiology, Gunma University School of Medicine, Gunma, Japan.

Manuscript received February 27, 1998; revised manuscript received September 1, 1998, accepted October 15, 1998.

А	=	atria
$A_2A_3$	=	interval between atrial responses to the first and the second extrastimulus
$A_3A_3$	=	AH interval of the second atrial extrastimulus
$A_1A_2$	=	interval between atrial responses to basic stimulus and extrastimulus
AVNRT	=	atrioventricular node reentry tachycardia
ATP	=	adenosine triphosphate
ECG	=	electrocardiogram
Н	=	His bundle
V	=	ventricles

Electrophysiologic studies. Electrophysiologic study was conducted after discontinuance of cardioactive drugs for at least 5 half-lives and after securing informed written consent. Two 6F quadripolar electrode catheters were, respectively, positioned in the high right atrium and the right ventricular apex for recording of intracardiac electrograms and pacing. A third 7F quadripolar electrode catheter was placed across the tricuspid valve for His bundle (H) recording, and a fourth quadripolar electrode catheter was placed inside the coronary sinus for recording of local electrograms. The proximal electrode of this later catheter was positioned at the orifice of the coronary sinus. The interelectrode spacing of electrode catheters was 0.5 or 1 cm. Electrocardiogram (ECG) leads I, aVF, V<sub>1</sub> as well as intracardiac electrograms were simultaneously displayed and recorded on a multichannel oscilloscopic recorder (Electronics for Medicine, model VR-16 or Midas-2500, PPG Industries). The pacing stimuli were approximately twice the diastolic threshold in strength and 2 ms in duration, and were provided by a digital programmable stimulator (Bloom and Associates, DTU-200).

Anterograde and retrograde conduction properties were evaluated with incremental pacing and extrastimulus testing techniques. Diagnosis of AVNRT was made using the previously described criteria (1,3). Intravenous boluses of adenosine triphosphate (ATP) (3.5 to 10 mg in graded increments) were given to 16 of the 28 study patients during tachycardia. The exclusion criteria for atrial tachycardia or atrial flutter included the combination of 1) retrograde induction of the tachycardia showing a sequence of V-A-H-V; 2) the interval of atrial responses (AA or  $A_1A_2$ ) interval; the interval between atrial responses to basic stimulus and extrastimulus) to the initiating ventricular beat being longer than that of the noninitiating ventricular beat; 3) termination of the tachycardia by a ventricular premature beat that was not conducted to the atria; 4) paradoxical delay of the atrial activation following a premature ventricular beat during tachycardia; 5) entrainment of the tachycardia by overdrive ventricular pacing with a similar atrial activation sequence of the entrained beat to that during tachycardia; and 6) successful ablation of the tachycardia with delivery of the radiofrequency current to the inferiorposterior aspect of Koch's triangle using inferior approach (1,5,7–9). The possibility of AV reentry tachycardia incorporating a slow paraseptal accessory pathway was excluded by demonstration of 1) AV block without termination of tachycardia, and 2) no resetting of the atrial activation by a premature ventricular beat delivered within 50 ms of the expected QRS complex during tachycardia while the His bundle (H) was refractory (5,8).

**Definitions.** The slow-fast form of AVNRT is defined as the tachycardia with an AH/HA ratio >2 and the atrial activation (P wave) occurring slightly before, simultaneously with, or immediately after, the QRS complex during tachycardia (1–4). The earliest atrial activation may be registered from the H recording site, the orifice of coronary sinus, or simultaneously from the H recording site and the orifice of the coronary sinus. The atypical form of AVNRT is defined as tachycardia with an AH/HA ratio <0.6 (fast-slow form) or  $\approx$ 1 (fast-intermediate form). The earliest atrial activation is usually registered from the orifice of the coronary sinus in atypical AVNRT (10,11). The multiple form of AVNRT is defined as multiple tachycardias with a combination of the slow-fast form, the fast-slow form, and the fast-intermediate form of tachycardias.

Variation of P-QRS relation during tachycardia is defined as a change of AH and HA interval >20 ms accompanied by a change in AH/HA ratio without changes in atrial activation sequence. The cycle length of the tachycardia may remain stable or change slightly. Change in AH interval or HA interval alone is not sufficient for a diagnosis of variation P-QRS relation during tachycardia. For example, prolongation of AH interval and, therefore, the tachycardia cycle length without a change in HA interval may occur following intravenous bolus of ATP in slow-fast AVNRT but it will not result in variation of P-QRS relation.

**Data analysis.** Data are reported as mean value  $\pm$  SD, unless otherwise indicated. Chi square and Student *t* test were used for statistical analysis. A p value <0.05 was considered significant.

## RESULTS

Variation of P-QRS relation with changes in AH, HA and AH/HA ratio during tachycardia was observed in 8 of the 311 (3%) patients with slow-fast AVNRT and in 20 of the 108 (19%) patients with atypical or multiple AVNRTs. In 14 patients with both forms of AVNRT, variations of the P-QRS relation were confined to the atypical fast-slow form only (Table 1). The occurrence of variations of P-QRS relation was significantly more common in patients with atypical or multiple form of AVNRT (p < 0.0001). Patients with variation of P-QRS relation were younger than those without (age,  $38 \pm 16$  vs.  $46 \pm 17$  years; p = 0.0155). There was no significant difference in sex, basic sinus cycle length,

Case No.		Type of AVNRT	Variation of P-QRS During Tachycardia								
	Age/ Sex		AVNRT	Earliest Atrial Activation Site	Initiation of AVNRT	Wenckebach or 2:1 AVB	Termination of AVNRT	ATP	Maximum Δ-AH (ms)	Maximum Δ-HA (ms)	Maximum Δ-CL (ms)
1	36/F	SF	SF	His	No	Yes	No	No	85	100	20
2	22/F	SF	SF	His	No	Yes	No	(-)	50	20	30
3	30/M	SF	SF	His	No	Yes	No	(-)	80	55	35
4	33/M	SF	SF	His	No	Yes	No	(-)	100	80	40
5	62/F	SF	SF	His	No	Yes	No	(-)	70	50	20
6	52/M	SF	SF	His	Yes	No	No	(-)	55	40	25
7	22/F	SF	SF	OS	Yes	No	No	(-)	60	50	50
8	33/F	SF	SF	OS	Yes	Yes	Yes	Yes	35	60	20
9	53/F	Aty (FS)	FS	OS	No	Yes	No	No	70	65	10
10	41/F	Aty (FS)	FS	OS	No	No	Yes	Yes	70	55	25
11	67/M	Aty (FS)	FS	OS	No	Yes	No	(-)	110	120	10
12	65/M	Aty (FS)	FS	OS	No	No	Yes	Yes	205	160	60
13	57/F	Aty (FI)	FI	OS	No	Yes	No	No	85	85	45
14	27/F	Aty (FI)	FI	Simultaneous	No	No	Yes	No	85	70	15
15	24/F	Mul (SF, FS)	SF	OS	Yes	No	No	(-)	25	40	40
16	53/F	Mul (SF, FS)	SF	OS	Yes	No	No	No	90	140	60
17	62/F	Mul (SF, FS)	SF	OS	Yes	No	No	(-)	140	110	30
18	27/F	Mul (SF, FS)	SF	OS	No	Yes	No	(-)	80	85	20
19	43/F	Mul (SF, FS)	FS	OS	No	No	Yes	Yes	40	40	10
20	36/F	Mul (SF, FS)	FS	OS	No	Yes	No	No	45	55	10
21	51/F	Mul (SF, FI)	FI	OS	Yes	No	No	(-)	80	40	40
22	39/F	Mul (SF, FI)	FI	OS	Yes	Yes	No	No	80	115	40
23	20/F	Mul (SF, FI)	FI	OS	No	No	Yes	Yes	185	135	50
24	35/F	Mul (SF, FI)	FI	OS	No	Yes	Yes	No	200	160	40
25	8/F	Mul (SF, FI)	FI	OS	No	Yes	No	No	130	120	10
26	26/F	Mul (SF, FI)	FI	Simultaneous	Yes	Yes	Yes	Yes	60	35	65
27	27/F	Mul (FS, FI)	FI	OS	No	No	Yes	No	55	40	15
28	16/M	Mul (SF, FS, FI)	SF	Simultaneous	Yes	No	No	(-)	40	80	35

#### Table 1. Clinical and Electrophysiology Findings

ATP, adenosine triphosphate; Aty, atypical form of AVNRT; AVB, atrioventricular block; AVNRT, atrioventricular node reentry tachycardia; CL, cycle length; F, female; FI, fast-intermediate form AVNRT; FS, fast-slow form AVNRT; His, His bundle recording site; M, male; Maximum  $\Delta$ -AH, Maximum  $\Delta$ -HA and Maximum  $\Delta$ -CL: maximum changes in AH interval, HA interval and tachycardia cycle length; Mul, multiple form AVNRT; OS, orifice of the coronary sinus; SF, slow-fast form AVNRT; (-), data not available.



**Figure 1.** Recordings from case 8 showing variation of P-QRS relation with changes in AH, HA and AH/HA ratio at the initiation of slow-fast atrioventricular node reentry tachycardia following delivery of two atrial extrastimuli during sinus rhythm. The sinus cycle length was 655 ms and the coupling interval of the first extrastimulus was 320 ms. (A) The coupling interval of the second extrastimulus was 280 ms, and was 260 ms in (B). Note that the HA interval of the first echo beat was the longest and a progressive shortening of HA interval associated with changes in AH interval, AH/HA ratio and the cycle length of the tachycardia was observed after initiation of the tachycardia. The atrial activation sequence was unchanged, and the earliest atrial activation was registered from the orifice of the coronary sinus. Note also that the HA interval of the first echo beat was longer in (A) when the tachycardia was induced at a longer coupling interval (A<sub>2</sub>A<sub>3</sub>) with a shorter AH interval (A<sub>3</sub>H<sub>3</sub>) as compared to that in (B). I, aVF and V<sub>1</sub> = ECG leads I, aVF and V<sub>1</sub>; HRA, CSd, CSp and HBE = bipolar electrograms recorded from the high right atrium, the distal two electrodes of coronary sinus catheter, and the His bundle recording catheter; CL = cycle length; S<sub>2</sub> and S<sub>3</sub> = stimulus artifact of the first and the second extrastimulus; A<sub>1</sub>, A<sub>2</sub> and A<sub>3</sub> = atrial responses to the basic sinus or driven beat, the first extrastimulus and the second extrastimulus; A<sub>1</sub>, A<sub>2</sub> and A<sub>3</sub> = atrial responses to the basic sinus or driven beat, the first extrastimulus and the second extrastimulus; Ae and He = atrial and His bundle responses to the basic sinus or driven beat, the first extrastimulus and the second extrastimulus; Ae and He = atrial and His bundle responses during atrioventricular node reentry echoes or tachycardia. The cycle lengths of the tachycardia, AeHe and HeAe intervals are listed.

AH interval, HV interval, the longest atrial paced cycle length that sustained 1:1 anterograde fast-pathway conduction, the longest atrial paced cycle length that induced AV nodal block, the effective refractory period of the anterograde fast or slow pathway, and the tachycardia cycle length in patients with and without variation of P-QRS relation during tachycardia. Although the longest ventricular paced cycle length that induced VA block was not different in patients with or without variation of P-QRS relation, 24 of the 28 patients with variation of P-QRS relation during tachycardia displayed a retrograde Wenckebach periodicity with an increment of VA (ventriculo-atrial) interval  $\geq$ 50 ms. In contrast, only 60 of 303 patients with slow-fast (p < 0.0001) and 32 of 88 patients with atypical or multiple AVNRT (p < 0.0001) demonstrated retrograde Wenckebach periodicity with increment of VA interval  $\geq$ 50 ms.

**Case illustration.** A 33-year-old female had induction of slow-fast AVNRT by delivery of two atrial extrastimuli during

sinus rhythm or by rapid atrial pacing, when anterograde atrial impulse was blocked in the fast pathway and conducted through the slow pathway. The tachycardia had a cycle length of 450 ms, an AH of 360 ms, and an HA of 90 ms. The earliest atrial activation was registered from the ostium of the coronary sinus and the P wave was recorded immediately after the QRS complex during tachycardia. Variation of P-QRS relation with changes in AH, HA and AH/HA ratio was noted at the initiation of the tachycardia. The HA interval of the first echo beat was the longest, shortened rapidly and then became stable in the subsequent beat. An inverse relationship of the HA interval and the coupling interval of the initiating atrial extrastimulus was also noted. Figure 1 shows induction of the tachycardia after two atrial extrastimuli during sinus rhythm. The coupling interval of the first extrastimulus was 320 ms. In Figure 1,A, the  $A_2A_3$  (interval between atrial responses to the first and the second extrastimulus) coupling interval was 280 ms, the A<sub>3</sub>H<sub>3</sub> (AH interval of the second atrial



Figure 2. Continuous recording from case 8 showing variation of P-QRS relation with block proximal to the His bundle recording site (beat with a star) without interruption of the tachycardia after intravenous bolus of 4 mg adenosine triphosphate (see text for discussion).

extrastimulus) was 510 ms, and the HA interval of the first echo beat was 150 ms. The HA interval of the second echo beat was 105 ms, the third 95 ms, the fourth and the subsequent echo beats 90 ms. In Figure 1B, the coupling interval was shortened to 260 ms, and the  $A_3H_3$  was 570 ms. The HA interval of the first echo beat was 120 ms, much shorter than that in Figure 1,A.

When intravenous (IV) bolus of 4 to 6 mg ATP was given during episodes of tachycardia, termination of the tachycardia in the anterograde direction was noted with progressive lengthening of the tachycardia cycle length accompanied by shortening of the HA intervals. Occasionally, block proximal to the H recording site was noted without termination of the tachycardia. Figure 2 is a continuous recording during tachycardia showing nodo-H block after ATP. The HA interval shortened from 100 to 50 ms and the AA interval lengthened from 450 to 465 ms before nodo-Hisian block proximal to the H recording site occurred; the tachycardia continued without termination. Following the blocked beat, variation of AH, HA interval and AA interval were noted before stable tachycardia was resumed. During stable tachycardia, overdrive ventricular pacing entrained the tachycardia with an atrial activation sequence similar to that during the tachycardia (Fig. 3).

In the entire group of 28 patients, variation of P-QRS relation occurred either at the initiation of tachycardia (Fig. 1) or during Wenckebach periodicity with termination of the tachycardia (Figs. 2, 4 to 6). In 10 patients, variation of P-QRS relation with changes of AH, HA and AH/HA ratio occurred transiently following electrical induction of tachycardia; it lasted for a few beats and the AH, HA and AH/HA ratio then became stable (Fig. 1). The maximum change in tachycardia cycle length was  $41 \pm 14$  ms (range,

20 to 65) in these 10 patients. In 15 patients, variation of P-QRS relation with changes in AH, HA and AH/HA ratio was associated with the occurrence of Wenckebach or 2:1 block proximal to the H recording site without interruption of the tachycardia (Figs. 2 and 6). The maximum change in tachycardia cycle length in these 15 patients was  $28 \pm 16$  ms (range, 10 to 65); 8 of these 15 patients had a cycle length change of equal to or less than 20 ms.

In 9 patients, variation of P-QRS relation with changes in AH, HA and AH/HA ratio occurred during spontaneous termination of the tachycardia, three had nonsustained tachycardia (Figs. 4 and 5) and six received IV bolus, of ATP (Figs. 2 and 6). In the latter six patients, a total of 19 episodes of variation of P-QRS relation were noted following administration of various doses of ATP (3.5 to 10 mg). Changes in AH, HA and AH/HA ratio was usually associated with a progressive prolongation of the tachycardia cycle length resulting in termination of the tachycardia; however, it occurred before major changes in the tachycardia cycle length in 10 of the 19 episodes. Furthermore, progressive forward shifting of the P wave toward the QRS complex frequently made a fast-slow or fast-intermediate tachycardia appearing as slow-fast tachycardia before termination.

#### DISCUSSION

**Nature of atrioventricular node reentry.** The reentry circuit in AVNRT consists of an anterograde limb, a retrograde limb, a proximal link, and a distal link. In the common slow-fast form tachycardia, the anterograde limb is a slow pathway and the retrograde limb is a fast pathway (3,4,12). The retrograde fast pathway has little or no ability of decremental conduction,



**Figure 3.** Continuous recordings from case 8 showing entrainment of the tachycardia by overdrive ventricular pacing. The tachycardia displayed a cycle length of 435 to 445 ms. The ventricular pacing was conducted from the right ventricular apex at a cycle length of 400 ms. The last three paced beats entrained the tachycardia. Note the atrial activation sequences of the entrained beats are similar to that during tachycardia.

and its atrial exit is usually registered from the H catheter at the apex of Koch's triangle. A variant form of slow-fast AVNRT utilizes a slow pathway for anterograde and an intermediate pathway for retrograde conduction (10,13). The retrograde intermediate pathway is capable of a certain degree of decremental conduction, and its atrial exit is usually registered from the orifice of the coronary sinus.

In the atypical fast-slow form tachycardia, the circuit is reversed utilizing a fast pathway for anterograde and a slow pathway for retrograde conduction (11,14). The retrograde slow pathway displays marked decremental conduction, and its atrial exit is registered from the orifice of the coronary sinus. Rarely, the reentry circuit may utilize a fast pathway for anterograde and an intermediate pathway for retrograde conduction (10). Under such a circumstance, the tachycardia is very fast and may mimic atrial tachycardia or flutter. Multiple AVNRTs with combination of the above-described tachycardias may occur in the same patient (8,10). The exact nature of the AV node reentry circuit is unclear. The proximal link is likely to involve the perinodal transitional tissue and perhaps also a portion of the atrial tissue, while the distal link is likely to involve the nodo-Hisian transitional tissue (the NH zone) and a distal common pathway is likely to exist between the distal link and the H recording site (3,8,15,16). The retrograde fast pathway and the retrograde intermediate pathway are likely to reflect conduction through a portion of the anterior input of the perinodal transitional tissue; however, the intermediate pathway is located at a more proximal and inferior position relative to the fast pathway (9,10,17). The anterograde fast pathway is likely to reflect normal anterograde conduction. The slow pathway is likely to reflect conduction through the posterior input of the perinodal transitional tissue and the compact node, and is located inferiorly and posteriorly in Koch's triangle. The atrioventricular junction is a 3-dimensional stereo-type structure and, therefore, may show a certain degree of variation, especially in data obtained from surface mapping of Koch's triangle (17–22).

Variation of P-QRS relation during tachycardia. The present study demonstrates that variation of P-QRS relation with changes in AH, HA and AH/HA ratio during tachycardia may be observed in patients with AVNRT, especially in atypical or multiple-form tachycardias. This phenomenon usually occurs when the conduction system and/or reentry circuit are unstable during initiation or termination of the tachycardia. The mechanisms responsible for this phenomenon are proposed schematically in the ladder diagrams of Figure 7. As a common pathway exists between the distal link of the reentry circuit and the H recording site in AVNRT, changes in AH interval, HA interval, AH/HA ratio as well as block proximal to the H recording site without concomitant changes in the tachycardia cycle length or termination of the tachycardia may be observed when decremental conduction occurs at the distal common pathway.



**Figure 4.** Continuous recordings from case 12 showing induction of nonsustained fast-slow form atrioventricular node reentry tachycardia with variation of P-QRS relation. The tachycardia was induced by a ventricular extrastimulus, which was coupled to a ventricular driven cycle length of 500 ms at a coupling interval of 230 ms. Note there were trivial changes in AH, HA and AH/HA ratio at the beginning of the tachycardia. It was followed by a progressive lengthening of AH interval (from 105 to 310 ms) and a progressive shortening of HA interval (from 210 to 50 ms) concomitant with a progressive lengthening of the tachycardia cycle length (from 300 to 360 ms). The AH/HA ratio was reversed before termination of the tachycardia. S<sub>1</sub> = artifact of the basic driven beat.

However, because the distal common pathway shares similar electrophysiologic properties with the distal portion of the slow pathway and perhaps also the intermediate pathway or even the fast pathway, decremental conduction is likely to involve all these structures, and a concomitant lengthening of the tachycardia cycle length



**Figure 5.** Recordings from case 24 showing induction of a nonsustained atypical atrioventricular node reentry tachycardia. The tachycardia was induced by a ventricular extrastimulus, which was coupled to a ventricular driven cycle length of 480 ms at a coupling interval of 240 ms. At the beginning, the tachycardia appeared to be of the fast-intermediate form with an AH of 150 ms and HA of 150 ms. A progressive shortening of the HA interval (from 150 to -10 ms) and a progressive lengthening of the AH interval (from 150 to 350 ms) concomitant with a lengthening of the tachycardia cycle length (from 300 to 340 ms) were observed. The AH/HA ratio was reversed before termination of the tachycardia. Note that the atrial activation of the last beat was registered before the His bundle potential.



**Figure 6.** Recordings from case 19 showing induction of sustained fast-slow atrioventricular node reentry tachycardia and variation of P-QRS relation with block proximal to the His bundle recording site following intravenous bolus of 3.5 mg adenosine triphosphate. (A) A sustained fast-slow atrioventricular node reentry tachycardia was induced following rapid ventricular pacing at a cycle length of 300 ms. Note the tachycardia was induced after the fourth ventricular paced beat during an atypical retrograde Wenckebach periodicity when conduction shifted from the fast to the slow pathway. The cycle length of the tachycardia varied between 290 to 310 ms, AH 70 to 90 ms and HA 220 ms. (B) After adenosine triphosphate, a prolongation of AH between 90 to 130 ms, a variation of the tachycardia cycle length between 310 to 320 ms and a shortening of HA between 230 to 190 ms were noted before a blocked beat (indicated by a star) occurred. This was then followed by an episode of 3:2 block proximal to the His bundle recording site (indicated by stars) with progressive lengthening of the tachycardia cycle length and, then, termination of the tachycardia. s = stimulus artifact.

is likely to occur simultaneously. The fact that the tachycardia cycle length varies during variation of P-QRS in practically every patient, suggests a constant involvement of either one or both of the limbs of the reentry

circuit. It is possible that patients with atypical or multiple-form AVNRTs may have a larger or longer distal common pathway capable of decremental conduction. This may in part explain why variation of P-QRS



**Figure 7.** Ladder diagram showing the hypothetical model of variation P-QRS relation during atrioventricular node reentry tachycardia. In this diagram, the atypical form of tachycardia using a fast pathway for anterograde and an intermediate pathway for retrograde conduction is presented. The first beat represents a beat during stable tachycardia. The second beat represents a beat with changes in AH, HA and AH/HA ratio due to decremental conduction in the distal common pathway; the conduction time in anterograde limb and the retrograde limb and, therefore, the cycle length of the tachycardia remain unchanged. The third beat represents a beat with block proximal to the His bundle recording site due to block in the distal common pathway. The fourth beat represents a beat with variation of P-QRS relation due to simultaneous prolongation of the conduction time in the distal common pathway and the retrograde limb. The fifth beat represents a beat with variation of P-QRS relation due to prolongation of the tachycardia is increased in the fourth and the fifth beats. Note that in the fifth beat the AH/HA ratio is that of the slow-fast form. A = atria; N = compact atrioventricular node; NH = nodo-Hisian zone; H = His bundle. Solid line represents stable conduction; interrupted lines represent decremental conduction.

relation is observed more often in these patients. If this is the case, then at least in patients with both forms of AVNRT, the fast-slow reentry circuit is not the reversed form of the slow-fast circuit and the anterograde fast and slow pathway are not exactly the same pathway as their retrograde counterparts.

Alternatively, the concealed penetration of the initiating stimuli may result in different conduction times and degrees of recovery of the rest of the circuit and, thus, this may affect AH/HA intervals at the initiation of the tachycardia and for a few beats until the tachycardia is stabilized. As the retrograde fast pathway has shown an "all or none" behavior to anterograde concealed penetration by the initiating atrial extrastimuli, variation of P-QRS relation could be expected to occur less often in the slow-fast form AVNRT.

**Conclusions.** Variation of P-QRS relation with or without block though it occurs rarely in the common slow-fast form AVNRT, it occurs not infrequently in atypical or multipleform AVNRTs. This phenomenon is primarily the result of decremental conduction in the distal common pathway, frequently involving one or both limbs of the reentry circuit and resulting in tachycardia cycle length variations as well. The ECG manifestation of P-QRS variations with or without AV block during tachycardia, especially at initiation of tachycardias or in cases of nonsustained tachycardias, should not be misdiagnosed as atrial tachycardias while they could indeed be atypical or rarely, typical forms of AVNRT. Moreover, the variations could be of such magnitude that a long RP tachycardia can masquerade for brief periods of time as short RP tachycardia.

Reprints requests and correspondence: Dr. Delon Wu, Chang Gung Memorial Hospital, 199 Tung Hwa North Road, Taipei, Taiwan. E-mail: dw0917@cguaplo.cgu.edu.tw.

#### REFERENCES

384

- Wu D, Denes P, Amat-y-Leon F, et al. Clinical, electrocardiographic and electrophysiologic observations in patients with paroxysmal supraventricular tachycardia. Am J Cardiol 1978;41:1045–51.
- 2. Akhtar M, Damato AN, Ruskin JN, et al. Anterograde and retrograde conduction characteristics in three patients of paroxysmal atrioventricular junctional reentrant tachycardia. Am Heart J 1978;95:22–42.
- 3. Wu D. Dual atrioventricular nodal pathways: a reappraisal. Pacing Clin Electrophysiol 1982;5:72–89.
- Akhtar M, Jazayeri MR, Sra J, Blanck Z, Deshpande S, Dhala A. Atrioventricular nodal reentry: clinical, electrophysiological, and therapeutic considerations. Circulation 1993;88:282–95.
- Yeh SJ, Yamamoto T, Lin FC, Wu D. Atrioventricular block in the atypical form of junctional reciprocating tachycardia: evidence supporting the atrioventricular node as the site of reentry. J Am Coll Cardiol 1990;15:385–92.

- Lerman BB, Greenberg M, Overholt ED, et al. Differential electrophysiologic properties of decremental retrograde pathways in long RP' tachycardia. Circulation 1987;76:21–31.
- McGuire MA, Yip AS, Lau KC, et al. Posterior ("atypical") atrioventricular junctional reentrant tachycardia. Am J Cardiol 1994;73:469–77.
- 8. McGuire MA, Lau KC, Johnson DC, Richards DA, Uther JB, Ross DL. Patients with two types of atrioventricular junctional (AV nodal) reentrant tachycardia: evidence that a common pathway of nodal tissue is not present above the reentrant circuit. Circulation 1991;83:1232–46.
- Wu D, Yeh SJ, Wang CC, Wen MS, Chang HJ, FC Lin. Nature of dual atrioventricular nodal pathways and the tachycardia circuit as defined by radiofrequency ablation technique. J Am Coll Cardiol 1992;20:889–95.
- Wu D, Yeh SJ, Wang CC, Wen MS, Lin FC. Double loop figure-of-8 reentry as the mechanism of multiple atrioventricular node reentry tachycardias. Am Heart J 1994;127:83–95.
- Wu D, Denes P, Amat-y-Leon F, Wyndham CRC, Dhingra R, Rosen KM. An unusual variety of atrioventricular nodal reentry due to retrograde dual atrioventricular pathways. Circulation 1977;56:50–9.
- Denes P, Wu D, Dhingra RC, Chuquimia R, Rosen KM. Demonstration of dual A-V nodal pathway in patients with paroxysmal supraventricular tachycardia. Circulation 1973;48: 549–55.
- Ross DL, Johnson DC, Denniss AR, Cooper MJ, Richards DA, Uther JB. Curative surgery for atrioventricular junctional ("AV nodal") reentrant tachycardia. J Am Coll Cardiol 1985; 6:1383–92.
- Sung RJ, Waxman HL, Sakseua S, Juma Z. Sequence of retrograde atrial activation in patients with dual atrioventricular nodal pathways. Circulation 1981;64:1059–67.
- Iinuma H, Dreifus LS, Mazgalev T, Price R, Michelson EL. Role of the perinodal region in atrioventricular nodal reentry: evidence in isolated rabbit heart preparation. J Am Coll Cardiol 1993;2:465–73.
- Miller JM, Rosenthal ME, Vassalo JA, Josephson ME. Atrioventricular nodal reentrant tachycardia: studies on upper and lower 'common pathway.' Circulation 1987;5:930–40.
- Becker AE, Anderson RH. Morphology of the human atrioventricular junctional area. In: HJJ Wellens, KI Lie, MJ Janse, editors. The Conduction System of the Heart. Leiden, The Netherlands: H.E. Stenfert Kroese BV, 1976:263–86.
- James TN. Morphology of the human atrioventricular node, with remarks pertinent to its electrophysiology. Am Heart J 1961;62:756-71.
- 19. Truex RC, Smythe MQ. Reconstruction of the human atrioventricular node. Anat Rec 1967;101:17–57.
- 20. Radker DK. Atrioventricular node and input pathways: a correlated gross anatomical and historical study of the canine atrioventricular junctional region. Anat Rec 1989;224:336–54.
- Ho SY, McComb JM, Scott CD, Anderson RH. Morphology of the cardiac conduction system in patients with electrophysiologically proven dual atrioventricular nodal pathways. J Cardiovasc Electrophysiol 1993;4:504–12.
- 22. McGuire MA, Bourke JP, Robotin MC, et al. High resolution mapping of Koch's triangle using sixty electrodes in humans with atrioventricular junctional (AV nodal) reentrant tachycardia. Circulation 1993;88:2315–28.