Incessant junctional reciprocating tachycardia caused by dual atrioventricular nodal pathways and atrio-nodal bypass tract

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SUMMARY A case is described with clinical and electrocardiographic findings of incessant junctional reciprocating tachycardia. Electrophysiological study showed that longitudinal dissociation of the atrioventricular node into two pathways was responsible for the maintenance of the arrhythmia. The two intranodal pathways had different refractory periods but reciprocally related and overlapping conduction times (anterograde fast, retrograde slow, and vice versa). Induction and termination of the arrhythmia was related to the presence of a partial atrio-nodal bypass tract.

Atrioventricular re-entry is the most common mechanism of paroxysmal sustained supraventricular tachycardia in man.¹ Atrioventricular nodal re-entry is the result of a functional or anatomical dissociation of the atrioventricular node into two pathways with different conduction times and refractory periods.²⁻⁴ In the usual type of atrioventricular nodal re-entry, the slow pathway is used for anterograde conduction and the fast pathway for retrograde propagation of the re-entrant impulse.³ Induction of atrioventricular nodal re-entry is dependent on the achievement of a critical atrioventricular nodal delay (AH interval).⁴ Wu *et al.*⁵ described an unusual type of atrioventricular nodal re-entry in which there is an intranodal anterograde fast and a retrograde slow pathway. They also showed that both types of atrioventricular nodal re-entry, usual and unusual, may be present in the same patient, that is the slow and fast pathways can be used in both anterograde and retrograde direction at different times.⁵ Coumel *et al.*^{6,7} described an incessant form of junctional reciprocating tachycardia in children. Induction of the incessant form of tachycardia is not dependent on a critical conduction delay but requires a critical atrial coupling interval or rate.^{6,7} Similar observations have also been made in patients with the Wolff-Parkinson-White syndrome.^{8,9}

We describe a case with clinical presentation of incessant junctional reciprocating tachycardia. Initiation of the supraventricular tachycardia was not dependent on critical atrioventricular conduction delay but required a critical atrial coupling interval or

rate. The electrophysiological study suggested the presence of dual atrioventricular nodal pathways and a partial atrio-nodal bypass tract. The intranodal pathways (alpha and beta) had different refractory periods but overlapping conduction times.

Case report

A 56-year-old woman was admitted to the hospital because of a long standing history of palpitation (lasting from minutes to several days) and chest pain. The patient had a past history of hypertension. She had received propranolol and quinidine in the past without any improvement in her symptoms. Physical examination was normal. The chest x-ray film and M-mode echocardiogram showed nothing abnormal. The electrocardiogram during sinus rhythm showed left atrial abnormality and was otherwise normal. During supraventricular tachycardia the P waves preceded the QRS complexes with PR intervals of 0.16 s. The rate of the tachycardia was 140/min. Spontaneous initiation and termination of the tachycardia on rhythm strips was related to the occurrence of a premature atrial beat without significant lengthening of the PR interval.

The electrophysiological study was performed after informed consent was obtained. Electrode catheters were positioned under fluoroscopic control in the high right atrium, coronary sinus, and His bundle region. At the time of the study, the patient was in supraventricular tachycardia. During the initial intracardiac recording three different types of supraventricular

tachycardia with identical retrograde activation sequence and cycle length, but with varying anterograde (A_c -H) conduction times, were observed (Fig. 1). Tachycardia number one had an A_c -H interval of 240 ms and an H- A_c of 220 ms. The H- A_c / A_c -H ratio was 1:1.1 (Fig. 1A). Tachycardia number two had an A_c -H interval of 190 ms and an H- A_c of

270 ms. The H- A_c / A_c -H ratio was 1:0.7 (Fig. 1B). Tachycardia number three had an A_c -H interval of 140 ms and an H- A_c of 320 ms. The H- A_c / A_c -H ratio was 1:0.4 (Fig. 1C). The three types of tachycardia were sustained; transition from one type of tachycardia to another was observed on the monitor to be abrupt, and could not be recorded. Coupled atrial

Fig. 1 Spontaneous paroxysmal supraventricular tachycardia (PSVT). Electrocardiographic leads aVL, V1, III, and intracardiac high right atrial (HRA), proximal coronary sinus (CSP), distal coronary sinus (CSD), His bundle (HBE) electrograms are shown. A_c represents the atrial echo beat and H the His bundle electrogram. The paper speed in this and subsequent illustrations is 100 mm/s and the time lines are at 40 ms. Measurements are expressed in ms. (A) PSVT No. 1. There is low to high atrial depolarisation sequence (HBE→CSP→HRA→CSD) the cycle length of the tachycardia is 460 ms, with H- A_c of 220 ms and A_c -H of 240 ms. The H- A_c / A_c -H ratio is 1:1.1. (B) PSVT No. 2. The atrial depolarisation sequence and cycle length of the tachycardia is identical to PSVT No. 1. The H- A_c however, is 270 ms and A_c -H is 190 ms and the ratio is 1:0.7. (C) PSVT No. 3. The atrial depolarisation sequence and cycle length of tachycardia are identical to PSVT No. 1 and 2. The H- A_c however, is 320 ms and the A_c -H is 140 ms. The H- A_c / A_c -H ratio is 1:0.4.

Fig. 2 Initiation and termination of PSVT. Abbreviations similar to Fig. 1. S represents the pacing stimulus, A_s the atrial depolarisation, and H_s the His bundle depolarisation in response to the atrial extrastimulus. (A) During sinus rhythm, PSVT is induced with a single atrial extrastimulus (S) at a coupling interval of 400 ms. During sinus rhythm the AH interval is 80 ms; the A_s-H_s interval is 100 ms. The cycle length of the tachycardia is 460 ms, with an H-A_e and A_e-H of 330 ms and 130 ms, respectively. (B) A premature atrial impulse at a coupling interval of 260 ms terminates the PSVT by blocking in the retrograde pathway. Only slight prolongation of the A_s-H_s interval (140 ms) compared with the preceding A_e-H interval (130 ms) is seen. (C) Atrial pacing at a cycle length of 410 ms initiates the arrhythmia.

pacing during tachycardia number three showed reproducible termination of the arrhythmia at a coupling of 260 ms (Fig. 2B). Initiation of tachycardia number three was dependent on a critical atrial coupling interval (Fig. 2A) or atrial paced cycle length (Fig. 2C). Coupled atrial pacing during tachycardia number three induced only minimal lengthening of the atrioventricular nodal conduction times (A_s-H_s) of the premature impulse (Fig. 3A, B, and C). In contrast, lengthening of retrograde conduction time (H_s-A_s) was observed, with increasing prematurity of the atrial impulse (Fig. 3A, B, and C). The plot of anterograde conduction time during coupled atrial pacing (A_s-H_s) and during supraventricular tachycardia (A_s-H) against the atrial coupling interval ($H-A_s$) and the retrograde conduction time ($H-A_s$) is shown in Fig. 3C. There is a distinct difference in anterograde conduction times at close coupling intervals. Specifically, at $H-A_s$ and $H-A_s$ intervals of 220 ms the anterograde conduction time during tachycardia (A_s-H) measured 240 ms (Fig. 1A, Fig. 3C), and during coupled atrial pacing (A_s-H_s) it was 140 ms (Fig. 3B and C).

During sinus rhythm the basic conduction intervals were as follows: PA was 30 ms, AH was 80 ms, and the HV was 40 ms. During coupled atrial pacing the atrioventricular nodal conduction curves ($A_1A_2-H_1H_2$) were continuous. During right ventricular pacing no ventriculoatrial conduction was present. The patient was discharged on amiodarone (600 mg/day) and remained free of symptoms a year later. The electrophysiological study was not repeated.

Comments

Induction and termination of the tachycardia in our patient with a single atrial extrastimulus suggest the presence of a re-entrant mechanism (Fig. 2).¹⁻⁷ The absence of ventriculoatrial conduction excludes the participation of a Kent bundle in the re-entry circuit.¹ The differentiation between atrial tachycardia, with different A_s-H and $H-A_s$ intervals resulting from changes in autonomic tone, and atrioventricular nodal re-entrant tachycardia is more difficult; in our case the incessant form of the arrhythmia and the changes in AH intervals, however, which were abrupt and sustained, argue against atrial tachycardia and make atrioventricular nodal re-entry the most likely mechanism.^{1 10}

We propose that the supraventricular tachycardia seen in our patient is the result of the presence of dual atrioventricular nodal pathways (alpha and beta) and, in addition, a partial atrionodal bypass tract. During sinus rhythm, the impulse propagates through the atrioventricular nodal pathways (alpha and beta) and through the bypass tract (Fig. 3D). An atrial prema-

ture beat or a critical increase in sinus rate results in an anterograde block in the beta pathway with conduction through the alpha pathway and the atrionodal bypass tract. Conduction through the bypass tract propagates with faster velocity than in the intranodal alpha pathway and engages the beta pathway in a retrograde direction. Retrograde conduction through the beta pathway occurs with sufficient delay to enable the alpha pathway to recover and complete the re-entrant circuit in an anterograde direction (Fig. 3D). Once the tachycardia is induced, the anterograde conduction occurs through the alpha pathway, the retrograde conduction through the beta pathway, and the depolarisation of the atria through the proximal common pathway (Fig. 3D). During tachycardia, the impulse conducts through the alpha pathway rather than the bypass tract because of its closer anatomical location to the retrogradely conducting beta pathway. Retrograde conduction occurs through the beta pathway rather than the bypass tract because of retrograde block in the bypass tract resulting from concealed anterograde conduction of the atrial echo beat (Fig. 3D). Termination of the arrhythmia is observed when a premature atrial beat at a critical coupling interval propagates through the bypass tract with a faster conduction velocity than through the alpha pathway, rendering the alpha and beta pathways refractory (Fig. 3D).

The electrophysiological characteristics of both anterograde and retrograde intranodal pathways (alpha and beta) need further discussion. The retrograde (beta) pathway showed a progressive lengthening of its conduction time (H_s-A_s) at decreasing $H-A_s$ coupling intervals (Fig. 3C). The anterograde conduction time through the alpha pathway showed pronounced and reciprocal variations with the conduction time present in the beta pathway. In addition, unexpected differences were observed in anterograde conduction times between spontaneous tachycardia and coupled atrial pacing. In fact, as shown in Fig. 3C, at similar $H-A_s$ and $H-A_s$ intervals, the anterograde conduction time (A_s-H) during spontaneous tachycardia was considerably longer than the conduction time (A_s-H_s) observed during coupling atrial pacing. This finding suggests that the pathway used for anterograde conduction during supraventricular tachycardia (alpha pathway) was different from the pathway used when premature atrial extrastimuli were delivered (atrionodal bypass tract). The latter pathway failed to show significant increases in its conduction time at close coupling intervals ($H-A_s$), suggesting the presence of a partial atrionodal bypass tract. The absence of ventriculoatrial conduction in our case can be explained by a unidirectional block in the distal common pathway (Fig. 3D).

Akhtar *et al.*¹¹ showed that three patterns of parox-

Fig. 3 Atrial coupled pacing during PSVT. Abbreviations similar to Fig. 1. (A) During PSVT, a single premature atrial stimulus is delivered at a coupling interval of 360 ms. The coupling interval to the preceding H is 330 ms. The anterograde conduction time of the premature impulse (A_s-H_s) is shorter (120 ms) than the anterograde conduction time (A_e-H) during the PSVT (130 ms). (B) The coupling interval of the premature atrial impulse is decreased (270 ms). The H-A_s interval measures 220 ms; a minimal prolongation of the A_s-H_s interval is observed (140 ms). (C) The anterograde conduction time of the premature atrial impulse (A_s-H_s), the retrograde conduction time of the resulting echo beat (H_s-A_e), and the anterograde conduction time during PSVT (A_e-H) are plotted against the atrial coupling interval (H-A_s). H_s-A_e shows the expected lengthening with increasing shortening of H-A_s. At equivalent H-A_s intervals (330 ms and 220 ms), the conduction through the anterograde pathway during PSVT (A_e-H) shows distinct prolongation when compared with the anterograde conduction during coupled atrial pacing (A_s-H_s). (D) Schematic representation of the proposed mechanism of the PSVT. PCP, proximal common pathway; BT, bypass tract; DCP, distal common pathway; PAB, premature atrial beat; Cross hatched areas, zone of retrograde block. See text for discussion.

ysmal atrioventricular junctional tachycardia can be identified on the basis of the relation between retrograde and anterograde conduction intervals (H-A_e/A_s-H) and that these ratios could offer a simple and reproducible method for determining the site of re-entry during supraventricular tachycardia. They also concluded that the ratios are fairly constant and the intermediate relations are only seen during changes of the cycle length of the tachycardia.¹¹ The varying H-A_e/A_s-H ratios (1:1.1 to 1:0.4) during spontaneous tachycardia observed in our case demonstrate the limited usefulness of this classification.¹¹

Wu *et al.*⁵ described three patients with atrioventricular nodal re-entrant tachycardia in whom the fast pathway was used for anterograde and the slow path-

way for retrograde conduction (unusual type of atrioventricular nodal re-entry). They also showed that the same pathways could be used in the opposite direction, that is the fast for retrograde and the slow for anterograde conduction (usual type of atrioventricular nodal re-entry).⁵ Though in our case both usual and unusual type of atrioventricular nodal re-entry were observed, our electrophysiological findings differ from the observations of Wu *et al.*⁵ The alpha pathway was always used for anterograde conduction and the beta pathway was always used for retrograde conduction. There was a reciprocal relation between the alpha and beta pathway conduction times, that is

when one was slow the other was fast and vice versa. Thus, in the present report, the anterograde and retrograde intranodal pathways could not be classified as either "slow" or "fast". For this reason we labelled them as alpha and beta pathways, according to the original description of Moe *et al.*² The varying and reciprocal changes of the conduction times in the two intranodal pathways recorded in this case are interesting in that they have not previously been reported in man, but observed only in the experimental animal models.¹²

The presence of partial atrio-nodal bypass fibres has been suggested to explain the lack of AH interval prolongation observed during initiation of incessant junctional reciprocating tachycardia.¹³ The postulated partial atrio-nodal bypass tract, however, could also have been an intranodal structure.¹³ Our case provides electrophysiological evidence for the presence of a partial atrio-nodal bypass tract separate from the atrioventricular node.

Finally, the therapeutic implications of the described mechanism deserve further comments. In our case, the induction and the maintenance of the tachycardia were critically dependent on the presence of an atrio-nodal bypass tract and of dual atrioventricular nodal pathways, respectively. Either a "myocardial" or a "nodal" drug, affecting the electrophysiological properties of the bypass tract or of the atrioventricular node, might have been considered in combination or alone, as previously described.^{8,9} Amiodarone has been proved to exert significant electrophysiological effects (slower conduction velocity, increased refractory periods) on both the accessory pathway and the atrioventricular node.^{14,15} Amiodarone may be the drug of choice in cases where multiple re-entrant pathways are responsible for the occurrence of supraventricular tachycardia.

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