Fast-slow and slow-slow form of atrioventricular nodal reentrant tachycardia sustained by the same reentrant circuit: a case report

Francesco Cantù, Paolo De Filippo, Roberto Rordorf, Gaetano M. De Ferrari, Folco Frattini, Barbara Petracci, Giovanni Russo, Marina Cerrone, Maurizio Landolina

Department of Cardiology, IRCCS Policlinico San Matteo, Pavia, Italy

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It has been suggested that a reentrant circuit confined to the posterior extensions of the atrioventricular node underlies both fast-slow and slow-slow types of atrioventricular nodal reentrant tachycardia (AVNRT). According to this hypothesis the fast-slow reentrant circuit would be formed by two slow pathways, located in the rightward and leftward posterior extension of the atrioventricular node. Thus, the fast pathway would act as a bystander with respect to the reentrant circuit.

We describe the case of a 40-year-old woman with several episodes of palpitations unresponsive to antiarrhythmic drugs. The ECG during symptoms showed a narrow QRS tachycardia with a long ventriculo-atrial interval and a negative P wave in the inferior leads. Electrophysiological study showed the inducibility of a slow-slow AVNRT which rapidly shifted to a fast-slow AVNRT without any change in the duration of the tachycardia cycle. Our observation is in agreement with the hypothesis that the fast-slow reentrant circuit consists of two slow pathways with the fast pathway acting as a bystander.

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Address:

Dr. Francesco Cantù

Dipartimento di Cardiologia IRCCS Policlinico San Matteo Piazzale Golgi, 2 27100 Pavia E-mail: elettrofisiologia@smatteo.pv.it

Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is a common form of paroxysmal supraventricular tachycardia, based on a substrate characterized by two functionally and anatomically distinct atrioventricular (AV) nodal pathways. The exact boundaries of the reentrant circuit in AVNRT have been subject of debate for many years: while some investigators supported the idea that the circuit was confined within the AV node, others suggested that atrial tissue and transitional cells formed the upper part of the reentrant circuit[1]. The role of the atrio-nodal connections and atrial tissue in the AVNRT circuit has been definitely proven following the introduction of radiofrequency catheter ablation: as a matter of fact, discrete lesions applied to the posterior AV junction (slow pathway), far from the compact AV node, may prevent the occurrence of AVNRT[2,3]. It has been conclusively shown that the reentrant circuit of the common form of AVNRT (slow-fast) consists of antegrade conduction over the slow pathway and retrograde conduction over the fast pathway. The atrio-nodal connections representing the fast pathway are located in the antero-septal region, superior to the tendon of Todaro, whereas the slow pathway is formed by transitional cells situated in the postero-septal area.

Less is known about the reentrant circuit in the uncommon forms of AVNRT (slow-slow and fast-slow). Multiple slow nodal pathways, confined to the posterior extensions of the AV node, are the suggested anatomical substrate of the slow-slow form of AVNRT[4]. The reentrant circuit consists of two different slow pathways, while the His bundle is activated by a lower common pathway.

With regard to fast-slow AVNRT, it has been traditionally thought that this arrhythmia uses the fast pathway for antegrade conduction and the slow pathway for retrograde conduction; i.e. the same circuit of slow-fast AVNRT but in the opposite direction. Accordingly, it has been shown[5] that a retrograde pathway extending from the His bundle to the area below the coronary sinus os is an essential limb of the reentrant circuit in fast-slow AVNRT, confirming that this arrhythmia shares the same circuit with slow-fast AVNRT.

On the other hand, Jackman and colleagues[6] have recently suggested that the

fast-slow AVNRT circuit is similar to the slow-slow circuit; this hypothesis is based on the existence of a relatively long lower common pathway in fast-slow AVNRT. Actually they were able to demonstrate that during fast-slow AVNRT, ventricular extrastimuli have to advance the timing of His bundle activation by 30-60 ms or more before advancing atrial activation and resetting the tachycardia. According to this hypothesis, the fast-slow AVNRT circuit would be confined within the posterior extensions of the AV node (similar to the slow-slow one).

We describe a case of an uncommon form of AVNRT which provides support to this latter theory.

Case report

We present the case of a 40-year-old female with recurrent episodes of palpitations not responding to antiarrhythmic therapy (sotalol and amiodarone). The ECG recorded during symptoms showed a narrow QRS tachycardia with a long ventriculo-atrial interval and a negative P wave in the inferior leads (Fig. 1). After informed consent, the patient underwent electrophysiological study with our standard set-up: a decapolar deflectable catheter positioned inside the coronary sinus, an octapolar deflectable catheter in the His bundle region, and a quadripolar catheter in the high lateral right atrium (subsequently moved into the right ventricle for para-Hisian pacing). Ventricular incremental pacing showed retrograde nodal conduction both over the fast and slow pathways. A slow-slow AVNRT (cycle length 540 mc, HA 276 ms) was induced after a jump in the HA interval during ventricular programmed stimulation and was interrupted by a spontaneous ventricular extrasystole. In the premature ventricular beat inducing the tachycardia (S2), a retrograde His potential and a shift in retrograde atrial activation were recorded. The atrial retrograde sequence both in the S2 beat and during tachycardia was identical to that observed after retrograde conduction over the slow pathway, with the first atrial potential recorded at the proximal coronary sinus. The interruption by a ventricular extrasystole ruled out the possibility that the tachycardia was an atrial tachycardia originating from the postero-septal area.

The slow-slow tachycardia was also reproducibly inducible during programmed atrial stimulation following a jump in the AH interval (120 ms abrupt AH lengthening). After a few beats of tachycardia, a shortening of the AH interval (from 264 to 140 ms) with no change in the tachycardia cycle length (540 ms) (Fig. 2) occurred. This phenomenon was repeatedly observed following induction. To confirm the diagnosis, late ventricular extrastimuli were delivered beginning 50 ms after the His bundle and then advanced in 5 ms increments without retrograde activation of the His bundle. None of the extrastimuli was capable of advancing the following atrium. Para-Hisian pacing was also performed showing retrograde nodal conduction both in the presence and in the absence of His bundle capture[7].

We carefully mapped the retrograde atrial activation sequence during tachycardia: the first atrial activation was recorded in the postero-septal region and was preceded by evidence of a sharp potential (highly suggestive of a slow pathway potential) which, in contrast, followed atrial activation in sinus rhythm (Fig. 3). Retrograde coronary sinus activation mapping during tachy-

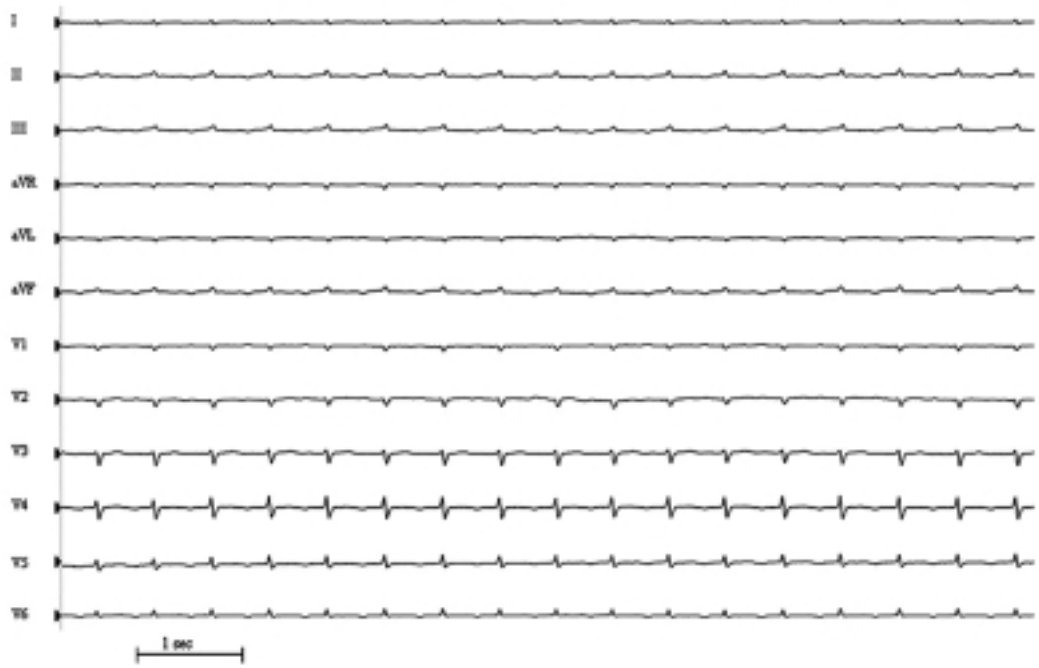

Figure 1. *ECG recording during symptoms: narrow QRS tachycardia with a long ventriculo-atrial interval and a negative P wave in the inferior leads suggesting a fast-slow atrioventricular nodal reentrant tachycardia.*

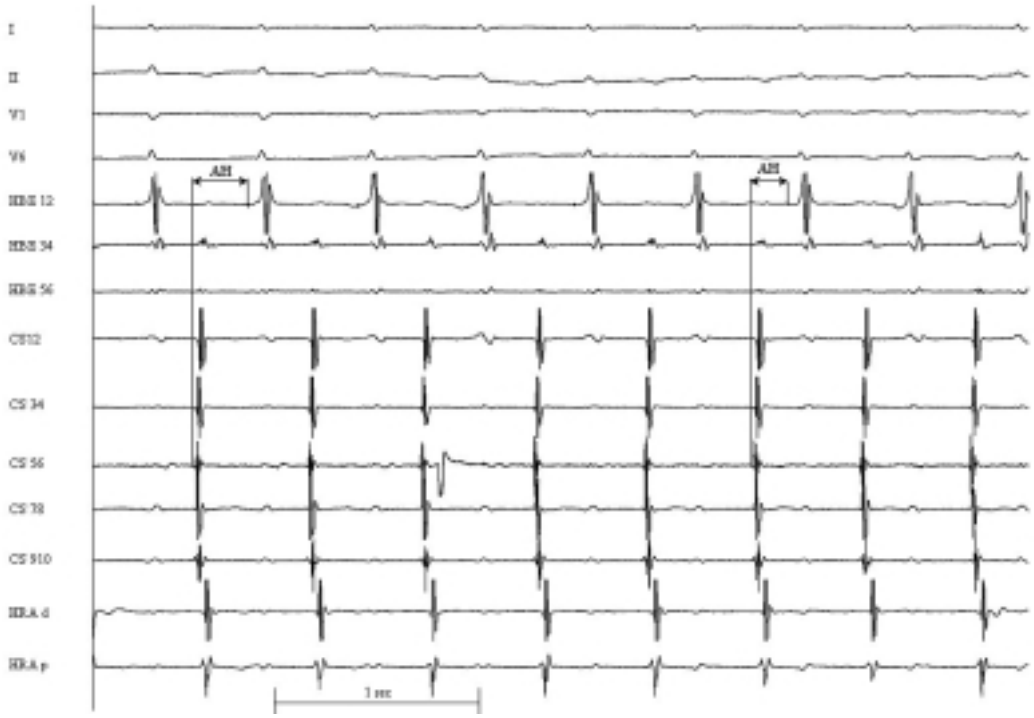

Figure 2. *Bipolar electrograms (filter 30 to 250 Hz) during tachycardia induced by programmed atrial stimulation with a jump in the AH interval: after a few beats of tachycardia a shortening of the AH interval occurs (4th, 5th, and 6th beat) with no change in the tachycardia cycle length. CS = coronary sinus; HBE = His bundle electrogram; HRA = high right atrium.*

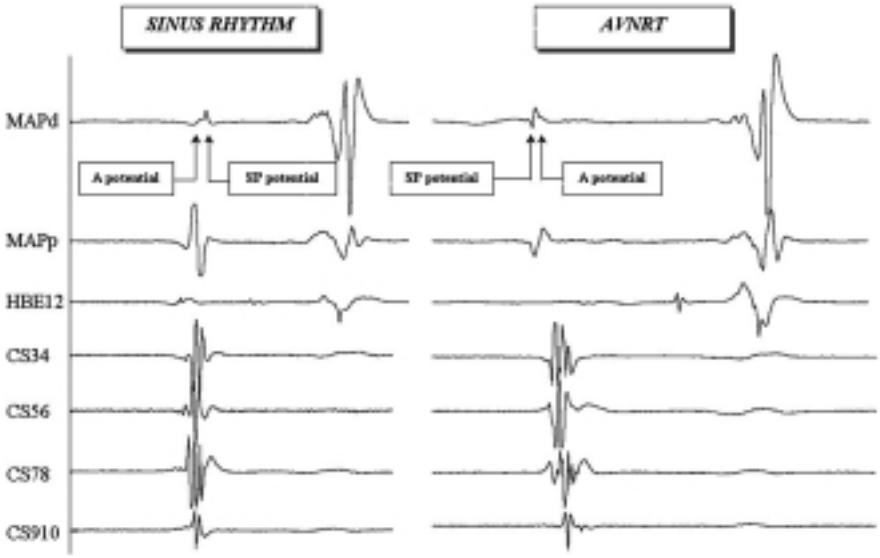

Figure 3. *Bipolar electrograms (filter 30 to 250 Hz) recorded during sinus rhythm and during atrioventricular nodal reentrant tachycardia (AVNRT) with a mapping catheter positioned in the postero-septal region. During sinus rhythm, the atrial (A) potential precedes the supposed slow pathway (SP) potential while during tachycardia the sequence is reversed. CS = coronary sinus; HBE = His bundle electrogram; MAP = mapping potential.*

cardia showed late activation of the coronary sinus musculature and confirmed that the first atrial activation was in the postero-septal area.

Three radiofrequency application pulses (35-40 W, 45-50°C, 60 s) were delivered on the slow pathway potential eliciting a junctional rhythm. Afterwards, atrial and ventricular programmed stimulation, both with and without isoproterenol, did not induce the tachycardia. Retrograde slow pathway conduction was also abolished.

Discussion

The observed phenomenon is quite unusual and its interpretation may provide interesting insights into the

pathophysiology of a fast-slow AVNRT circuit. Different mechanisms underlying the present finding may be suggested.

First, the tachycardia could be a slow-slow AVNRT with a rapid progressive acceleration of conduction over the lower common pathway during the first beats. This interpretation is very unlikely given the short antegrade conduction time during the short AH beats of tachycardia (AH 140 ms). Such a short time is not consistent with conduction over both the antegrade slow pathway and the lower common pathway.

Second, the tachycardia could, from the very beginning, be a fast-slow AVNRT with a conduction delay confined to the antegrade pathway during the first beats. This second possibility appears even more unlikely given the fact that the arrhythmia was reproducibly induced, with programmed atrial stimulation, following a 120 ms jump in AH conduction, suggesting antegrade conduction over a slow pathway.

A third explanation could be that the observed phenomenon was due to a shift from a slow-slow to a fast-slow AVNRT with a jump of antegrade conduction from a slow to a fast pathway. This possibility seems more likely even though some aspects are not totally consistent with this interpretation. The cycle length is exactly the same in the first and following beats of tachycardia, whereas in case of a shift to an alternative limb of reentry a change in the tachycardia cycle length would be expected. Also, the AH interval shortening is accompanied by an identical and very marked HA interval lengthening. This is difficult to explain since given the fact that the retrograde conduction over the slow pathway does not change. Finally, the described phenomenon was reproducibly observed following atrial stimulation and the two arrhythmias were never observed separately; this is quite curious if we assume the existence of two different reentrant circuits.

A fourth possibility is that of a circuit formed by a decremental concealed postero-septal accessory pathway as the retrograde limb and, alternatively, a fast and slow nodal pathway as the anterograde limb. However, several findings militate against this option. Indeed, both para-Hisian pacing and the use of late ventricular extrastimuli during His-bundle refractoriness were not consistent with an accessory pathway. Specifically, this latter maneuver was conducted by delivering extrastimuli beginning 50 ms after the His potential and then advancing them in 5 ms increments: in the presence of a decremental postero-septal accessory pathway, at least some of the delivered extrastimuli would be expected to advance the following atrial activation, but this was never the case. Also, the stability of the tachycardia cycle length suggests a single reentrant circuit underlying both the observed arrhythmias. Finally, both the clinical presentation and the response to radiofrequency ablation favor the hypothesis of AVNRT rather than that of permanent junctional reentrant tachycardia.

A possible and reliable interpretation of the described findings derives from the suggestion by Jackman and colleagues[6] that the fast-slow circuit might be very closely related to the slow-slow circuit. According to this hypothesis, the fast-slow AVNRT circuit could be formed by two slow pathways with the fast pathway

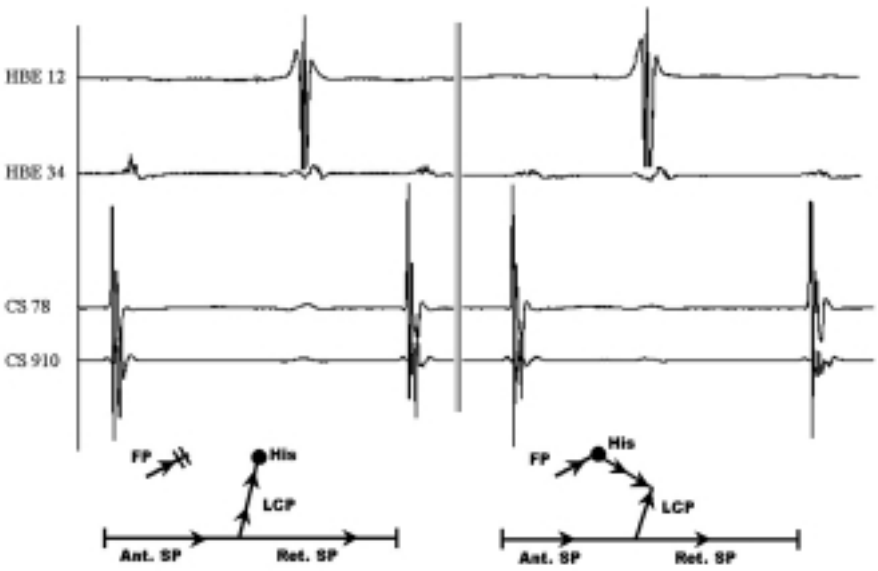

Figure 4. *In the* upper part *of the figure four bipolar electrograms (filter 30 to 250 Hz) recorded at the beginning* (left panel) *and after a few seconds* (right panel) *of the same episode of tachycardia are shown. Note the clear difference in the AH value between the two beats in spite of the fact that the tachycardia cycle lengths are identical. The* lower part *of the figure shows the proposed electrophysiological mechanism underlying the described phenomenon. During the first beats of tachycardia* (left)*, the His bundle (His) is activated by the lower common pathway (LCP) of the slow-slow circuit while the fast pathway (FP) is refractory and cannot be activated by the atrial septum musculature. A few beats later* (right)*, the AH interval has shortened due to antegrade conduction over the FP which however plays a bystander role with respect to the reentrant circuit. A progressive shortening of refractoriness allows conduction over the FP; two wavefronts collide in the LCP which is no longer responsible for His activation. Ant SP = antegrade slow pathway; CS = coronary sinus; HBE = His bundle electrogram; Ret SP = retrograde slow pathway.*

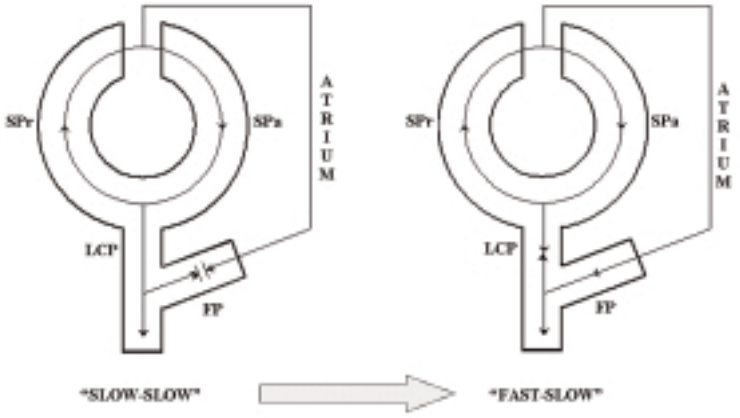

Figure 5. *Outline of the proposed electrophysiological mechanism underlying the described phenomenon. FP = fast pathway; LCP = lower common pathway; SPa = antegrade slow pathway; SPr = retrograde fast pathway.*

responsible for His activation but not directly involved in the reentrant circuit. Indeed, the shift within a few beats from a slow-slow to a fast-slow AVNRT, with no change in the tachycardia cycle length, favors the hypothesis that these two arrhythmias share the same reentrant circuit formed by two slow pathways. If this is the case, during a fast-slow type AVNRT, the ventricle is activated by the fast pathway which, however, is not an integral part of the circuit. In the present case, the tachycardia started as a slow-slow type AVNRT with the first atrial retrograde activation recorded in the postero-septal region. During the first beats of tachycardia the fast pathway was still refractory and the His bundle was activated by the lower common pathway of the slow-slow circuit. After a few seconds, a progressive shortening of refractoriness – probably due to a rate-dependent shortening of the action potential of the transitional cells – allows conduction over the fast pathway which in turn results in earlier activation of the His bundle. Thus, the fast pathway plays a bystander role with respect to the reentrant circuit, while two opposite wavefronts collide in the lower common pathway that is no longer responsible for His bundle activation (Figs. 4 and 5).

The possibility that in fast-slow AVNRT a slow pathway is the antegrade limb of reentry, was recently confirmed in a large cohort of AVNRT patients[8]. In this description all fast-slow and the majority of slow-slow AVNRT cases showed a long lower common pathway, while no evidence of a lower common pathway was demonstrated in most patients with slow-fast AVNRT. This finding confirms that a fast-slow AVNRT circuit is unlikely to be the reverse of a slow-fast circuit and could be more closely related to a slow-slow circuit. The authors report that many episodes of fast-slow AVNRT, in their cohort, were initiated following a sudden AH prolongation which shortens in the ensuing beats, but they do not elaborate further on the cycle length and on the implications of their findings. This latter observation favors the hypothesis that a slow pathway is the antegrade pathway in fast-slow AVNRT. Moreover, the observation of an AH shortening in the beats following induction could be explained by the same mechanism presumed as being the culprit in the present case report.

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