CASE REPORT

Supraventricular Tachycardia Initiated by Couplets of Atrial Extrasystoles but Not by Single Premature Atrial Beats: What Is the Mechanism?

Vincenzo Carbone, M.D.,* Vincenzo Marafioti, M.D.,† Lilia Oreto, M.D.,* and Giuseppe Oreto, M.D.*

From the *Department of Clinical and Experimental Medicine, University of Messina, Messina, Italy; and †Cardiovascular and Thoracic Department, University Hospital of Verona, Verona, Italy

The reported electrocardiogram shows several atrial extrasystoles (AEs) sometimes occurring in couplets. The former beat of each couplet is nonconducted, whereas the latter triggers a supraventricular tachycardia with negative P waves in inferior leads and $RP > PR$. This suggests an atypical atrioventricular nodal reentrant tachycardia involving the fast pathway anterogradely and the slow pathway retrogradely. The tachycardia is never precipitated by single AEs. The blocked AE of each pair is pivotal in tachycardia initiation, allowing the subsequent impulse to conduct down the fast pathway. A concealed slow pathway penetration during the blocked AE is invoked as the key mechanism.

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atrial extrasystoles; couplets; dual AV nodal pathways; atypical AV nodal reentrant tachycardia; fast pathway; slow pathway

CASE PRESENTATION

The electrocardiogram (ECG) in Figure 1 has been recorded from a hypertensive 62-year-old woman suffering from recurrent palpitations. It reflects a sinus rhythm at a rate of 75 beats/min and several atrial extrasystoles (AEs) with varying coupling intervals. The premature P waves are flat in lead II and biphasic (+/–) in lead V₁. Some of these AEs are not conducted to the ventricles, giving rise to prolonged RR intervals. Such a phenomenon is apparent in the strips 1 and 2: in lead II, the QRS complex starting the long cycle is followed by a peaked T wave, the expression of a premature P wave superimposed on the preceding T wave. Sometimes, the AEs occur in couplets. The former extrasystole of every couplet is blocked, whereas the latter invariably starts a narrow QRS complex tachycardia at a rate of 120 beats/min (strips 3 and 4). Surprisingly, such

a tachycardia is never elicited by a single AE. The first QRS of each tachycardia episode is wide, displaying a complete right bundle branch block configuration. This is due to ventricular aberrant conduction caused by the long–short cycle sequence. During the tachycardia, the QRS complexes are followed at a considerable distance (i.e., closely the end of the corresponding T wave) by a P wave that is negative in leads II, III, aVF and positive in V₁. These P waves, indicating a retrograde atrial activation, occur nearer to the ensuing beat than to the preceding one so that the resulting RP interval is longer than the subsequent PR interval. Figure 2 illustrates three different episodes of supraventricular tachycardia (SVT) showing the above features. The tachycardia terminates either spontaneously (A) or following a carotid sinus massage (B) or as a consequence of a couplet of AEs (C). The episode A ends with a retrograde P wave, whereas both

Address for correspondence: Vincenzo Carbone, M.D., via Europa, 84, 80047 S. Giuseppe Vesuviano (Naples), Italy. Fax: +39 0815287237; E-mail: carbovincenzo61@gmail.com

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Figure 1. Electrocardiogram on admission. Selected noncontinuous strips showing several atrial extrasystoles (AEs), labeled with dots. Strips 1 and 2: single AEs in standard leads II and V₁. The AEs occurring with a very short coupling interval are blocked. The nonconducted premature P waves are superimposed on the preceding T wave, which in lead II assumes a peaked configuration. Strips 3 and 4: two episodes of supraventricular tachycardia started by couplets of AEs, recorded in leads II and V₁, respectively. The former AE of each pair is nonconducted.

episodes B and C terminate with a narrow QRS complex. The tachycardia episodes ranged from 27 to 116 seconds and alternated with periods of regular sinus rhythm lasting approximately 1–3 minutes.

COMMENTARY

An SVT with negative P waves in inferior leads and RP intervals longer than PR intervals may be caused by the following mechanisms: (1) atrial tachycardia arising from the region surrounding the coronary sinus; (2) permanent form of atrioventricular (AV) junctional reciprocating tachycardia (PJRT), maintained by anterograde conduction over AV node/His bundle and retrograde conduction over a posteroseptal, slowly-conducting accessory pathway; and (3) AV nodal reentrant tachycardia (AVNRT) with anterograde conduction down the fast pathway and retrograde conduction up the slow pathway (the so-called uncommon, atypical, or fast-slow AVNRT).^{1,2} The above arrhythmia features rule out the diagnosis

1. The AEs precipitating the tachycardia strongly differ from the P waves seen during the tachycardia. In contrast, in the automatic atrial tachycardia, the first P wave shows the same configuration as the subsequent P waves. In addition, automatic atrial tachycardia usually accelerates shortly after its initiation, whereas the episodes in Figures 1 and 2 all show constant cycle lengths, since the beginning. Finally, the reported tachycardia is interrupted by carotid sinus massage or by pairs of AEs, and these instances are inconsistent with the diagnosis of automatic atrial tachycardia, because (1) enhanced vagal activity usually does not affect the focal automaticity, while it results in appearance or worsening of an AV block without modifying the sequence of the ectopic P waves; and (2) premature atrial impulses cannot stop an automatic atrial tachycardia, but they can merely reset its timing. An SVT elicited by a premature P wave with a contour differing from that of the subsequent P waves could theoretically reflect an

Figure 2. Three different episodes of supraventricular tachycardia in leads II (episodes A and B) and V₁ (episode C). The upper strip of each event shows tachycardia initiation. The lower strip of each event shows tachycardia termination. Dots indicate the premature P waves. The episode A ends spontaneously. The episode B is interrupted by carotid sinus massage (CSM). The event C is stopped by a couplet of AEs.

intraatrial reentrant mechanism. This hypothesis, however, does not account for tachycardia termination with a nonconducted P wave (Figure 2, event A), as such a circumstance indicates the need for the AV conduction in the arrhythmia mechanism. In addition, the response of an intra-atrial reentry to vagal maneuvers is quite unusual.¹⁻³ A PJRT may be induced by AEs, although it is usually elicited by a slight shortening in sinus cycle length. For this reason, such a tachycardia occurs in a permanent form or in closely recurrent episodes interposed to brief periods of sinus rhythm, being therefore defined as incessant. In the present case, however, the tachycardia is initiated only by couplets of AEs and never starts following a single AE or a simple

sinus cycle shortening. In addition, the tachycardia occurs in recurrent paroxysmal episodes between long periods of sinus rhythm, rather than in a chronic or permanent form.^{1,2} The most tenable explanation for this ECG, thus, is the mechanism 3. The so-called fast-slow AVNRT is much less common than the slow-fast variety of the same tachycardia. The unifying mechanism is a reentry within the AV node in the presence of dual AV nodal pathways. As the refractory period of the fast pathway is usually longer than that of the slow pathway, a premature atrial beat readily finds the former refractory, being conducted to the ventricles over the slow pathway, with a long PR interval. Once the impulse reaches the lower common AV nodal pathway, it returns to the atria

Figure 3. Ladder diagram to explain the tachycardia mechanism. Solid red lines indicate the conduction over the fast nodal pathway. Broken blue lines indicate the conduction over the slow nodal pathway. Horizontal dark bar represents the slow pathway refractory period. Horizontal green bar indicates the fast pathway refractory period (the terminal gray area expresses a relative refractoriness). See text for explanation.

traversing the fast pathway in a retrograde direction, thus perpetuating the reentrant loop underlying the tachycardia.¹⁻⁴ In some circumstances, a reverse reentry (fast anterograde–slow retrograde) occurs, but in these cases, the most likely precipitating event is an extrasystole of ventricular, rather than atrial, origin. This occurs because the ventricular premature impulse finds the fast pathway refractory and conducts retrogradely over the slow pathway, and then it returns down to the ventricles through the previously blocked fast pathway.^{2,3} However, a single AE may sometimes trigger a fast–slow AVNRT, but such an event requires that the slow pathway refractoriness is longer than the fast pathway refractoriness. This instance, however, is uncommon and explains the rareness of such a form of tachycardia.²⁻⁴ In the present case, an SVT with the features of an atypical AV nodal reentry is invariably precipitated by couplets of AEs whose first beat is nonconducted, and this does not seem to be an haphazard finding. As illustrated in the ladder diagram in Figure 3, the former premature atrial impulse of each couplet blocks in both fast and slow nodal pathways. However, a concealed anterograde conduction down the slow pathway

occurs as far as its distal end. As an alternative explanation, the impulse proceeding down the fast pathway could concealedly reenter the slow pathway, before blocking in the lower common pathway. Such concealed (anterograde or retrograde) conduction over the slow pathway results in prolongation of its refractoriness so that this lasts longer than that of the fast pathway. This allows the latter AE to travel anterogradely over the partially recovered fast pathway, with a slight delay due to conduction during the relative refractory period. The delayed conduction down the fast pathway of the second AE (expressed by a small PR interval prolongation) gives a little more time for recovery of the slow pathway from the effects of concealed penetration, making it available for retrograde conduction. Finally, therefore, the impulse returns to the atria through the slow pathway, thereby initiating and perpetuating the fast–slow AVNRT. We assume that in this case, as it usually occurs, the refractory period of the slow pathway is shorter than that of the fast pathway, but this difference is so small that a typical AVNRT is unlikely to be induced despite the high number of AEs. Moreover, the longer refractory period of the fast

nodal pathway should prevent AEs from initiating an atypical form of AV nodal reentry.¹⁻⁴ Nevertheless, episodes of fast-slow AVNRT are easily started by couplets of AEs, whereas never this occurs by single AEs. The former AE of each couplet, thus, despite being blocked in the AV junction, seems to play a pivotal role as it changes the electrophysiological properties of the AV node so as to help the following AE to trigger an atypical AVNRT. The nonconducted AE results in postponement of the refractory period of the slow pathway whose recovery, therefore, occurs later than the fast pathway. This also explains why a single premature atrial beat is always unable to initiate a tachycardia episode. To the best of our knowledge, such initiating mechanism for fast-slow AVNRT has never been reported previously. However, it has been shown by using double atrial extrastimuli that in the presence of dual AV nodal physiology a blocked atrial extrastimulus obscures the anterograde slow pathway conduction favoring the fast pathway conduction of the subsequent atrial impulse. The authors speculate that this may occur owing to a concealed

anterograde penetration of the nonconducted beat more deeply in the slow nodal pathway than in the fast one.⁵ The mechanism we invoked to explain the reported ECG and depicted in Figure 3 is strongly consistent with the above electrophysiological findings.

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