CASE REPORT

An Unusual Narrow QRS Complex Tachycardia: What Is the Mechanism?

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

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

The electrocardiogram of a 72-year-old woman showed episodes of nonsustained narrow QRS complex tachycardia. Tracing analysis suggested that the arrhythmia was due to interpolated atrial extrasystoles occurring in bigeminal rhythm. Interpolation of atrial extrasystoles is a rare phenomenon. In this condition, a premature atrial beat is "sandwiched" between 2 normal sinus beats, and sinus PP interval containing the extrasystole is often longer than unaffected sinus cycles. Alternative mechanisms for the arrhythmia are discussed, such as: (1) sinus node reentry; (2) 1:2 response to atrial ectopy over the fast and the slow atrioventricular nodal pathways; and (3) couplets of atrial extrasystoles.

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CASE PRESENTATION

The electrocardiogram (ECG) of Figure 1 (noncontinuous strips of lead II with an enlarged section of the top strip) has been recorded from a 72-year-old healthy woman complaining of palpitation. The tracing shows 2 episodes of nonsustained narrow QRS complex tachycardia followed by sinus rhythm restoration. During tachycardia, the RR intervals are "regularly irregular," resulting in a trigeminal grouping of the beats with average rate of 110/minute. Sinus rhythm restarts after a relatively long pause (1340 ms) in the bottom episode, whereas no pause is seen at tachycardia termination in the top event. Several QRS complexes are preceded by a clearly visible P wave, that is at times positive (e.g., the P waves marked with dots in the upper strip of Figure 2) and on other occasions negative (those labeled with arrows). The above findings seem to be inconsistent with any of the known types of supraventricular tachycardia. What is, then, the most likely mechanism underlying this unusual arrhythmia?

COMMENTARY

The positive P waves of tachycardia are very likely of sinus origin since they are identical to P waves observed during regular sinus rhythm. The negative P waves, in contrast, are consistent with atrial extrasystoles. PP intervals between consecutive manifest upright (sinus) P waves during tachycardia are exactly twice the PP intervals during sinus rhythm: this suggests that the regular sinus node discharge is not affected during tachycardia. It is therefore very likely that postextrasystolic QRS complexes not preceded by any manifest P wave (e.g., those marked with asterisks in the top strip of Figure 2) are of sinus origin, being a sinus P wave totally hidden within the T wave of the preceding ventricular complex. Such a hypothesis is supported by the observation that the 9th QRS of the top strip is preceded by an evident sinus P wave that is superimposed upon the T wave following the 8th QRS complex. This T wave is distorted by the nearly simultaneous P wave, as shown in the detail of Figure 2 (arrow).

Address for correspondence: Dr. Vincenzo Carbone, M.D., via Europa, 84 80047 S. Giuseppe Vesuviano (Naples), Italy. E-mail: vcarbone@cardionet.it Fax number: +39 0815287237

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Figure 1. Noncontinuous strips of lead II. The section within the dashed rectangle has been enlarged to allow ECG analysis. See text.

The intervals between a manifest sinus P wave and the assumed “invisible” (or barely visible) P wave coinciding with a T wave, however, measure about 1020 ms, being markedly longer than the intervals separating these hidden P waves from the ensuing manifest sinus P wave, that measure 690 ms (see the bottom strip of Figure. 2). At first glance, such variability in sinus PP cycle length speaks against the persistence of a regular sinus rhythm;

this condition, however, can still be assumed postulating the occurrence of interpolated atrial extrasystoles in bigeminal rhythm. Interpolation is relatively common in ventricular extrasystoles, but extremely rare in atrial ones. Whenever a premature beat of atrial origin is “sandwiched” between 2 normal sinus beats, the sinus PP interval containing the extrasystole is often slightly longer than the unaffected sinus cycle whereas,

Figure 2. Part of the rhythm strips as shown in Figure 1. The box at the top is a detail from the upper strip (enlargement of a section including beats 7th to 9th). The arrow highlights a sinus P wave that is superimposed on the T wave following the 8th QRS complex. Numbers express PP intervals in milliseconds (ms). See text.

Figure 3. Ladder diagram to explain the mechanism of tachycardia. S = sinus node; SA = sinoatrial junction; A = atrium; AV = atrioventricular junction; V = ventricle. Numbers in the section S express the sinus pacemaker cycle lengths; numbers at the bottom are the sinus PP intervals on the ECG (ms). Explanation in the text.

accordingly, the ensuing PP interval is shorter than the undisturbed sinus cycle.¹⁻³ Such a behavior occurs pronouncedly in this ECG, where the bigeminal distribution of the atrial ectopy results in perpetuating the interpolation-induced rhythm pattern, thus generating the unusual tachycardia. The ladder diagram of Figure 3 shows that any premature atrial impulse penetrates partially into the sinoatrial (SA) junction and does not reach the sinus pacemaker, whose rhythmic discharge is not disturbed. The ensuing sinus impulse thus finds a partially refractory SA junction so that the conduction time from the sinus pacemaker to the atrium is increased, and the corresponding P wave is postponed, resulting in PP interval prolongation. The next sinus impulse, in turn, occurs on schedule, and since its conduction time to the atrium is normal, the PP interval is shorter than the basic sinus cycle, due to delayed SA conduction of the previous impulse. The shortening of the sinus PP cycle that does not contain an atrial extrasystole is compensatory for the preceding PP prolongation so that the sum of 2 consecutive sinus PP intervals during tachycardia (1020 ms + 690 ms = 1710 ms) is exactly twice the undisturbed sinus cycle (860 ms × 2 = 1720 ms). Although interpolation of bigeminal atrial extrasystoles is the most likely mechanism for this tachycardia, alternative explanations may also be invoked, such as: (1) sinus node reentry provoked by the atrial premature impulse; (2) 1:2 atrioventricular (AV) conduction of the atrial extrasystole, namely, separate conduction over the fast and the slow AV nodal pathways, resulting

in 2 QRS complexes for 1 premature negative P wave; and (3) couplets of atrial extrasystoles. The mechanism (1) can be ruled out because in SA reentry, the sinus PP interval immediately following the atrial extrasystole should be longer than the basic sinus cycle;⁴ in this ECG, by contrast, the interval from the postextrasystolic hidden sinus P wave to the ensuing manifest one (690 ms) is considerably shorter than the normal PP interval (860 ms). The detection of a sinus P wave before the 9th QRS complex in the top strip (Figure 2) allows to exclude the mechanism 2), since in this circumstance, the 2nd QRS complex after the atrial extrasystole is not preceded by any P wave.⁵ Hypothesis 3) is also unlikely because in the reported ECG, the PP interval that follows 2 coupled beats is markedly shorter than the basic sinus cycle, as opposed to what one would expect in the presence of atrial couplets. Usually, the PP interval that is started by the 2nd of a pair of atrial extrasystoles is far longer than the normal PP interval since it results from the sum of: (1) retrograde SA conduction time of the 2nd ectopic impulse; (2) sinus cycle length; and (3) anterograde SA conduction time of the 1st sinus impulse following the couplet. In addition, the inherent intersinus interval occurring after an atrial pair may be lengthened, for repeated premature discharge of the sinus pacemaker tends to depress temporarily its automaticity, thereby prolonging even further the postextrasystolic PP interval.¹ It should be noted that the last 2 beats of tachycardia in the lower strip are a true couplet of atrial extrasystoles, as suggested by

the morphology and timing of the P waves (close inscription of 2 consecutive negative P waves) and by the subsequent sinus pause: the last finding implies that now interpolation does not occur at all, so that the ectopic impulses are able to reach the sinus node and reset it closely and prematurely, thus affecting its spontaneous rhythmic discharge.¹⁻³

In this report, interpolated atrial extrasystoles occurring with a bigeminal distribution can be regarded as a novel mechanism underlying a supraventricular tachycardia.

REFERENCES

1. Schamroth L. The Disorders of Cardiac Rhythm, 2nd Edition. Oxford, Blackwell, 1980, pp. 353-357.
2. Chung EK. Electrocardiography. Hagerstown, Harper & Row, 1974, pp. 185-223.
3. Langendorf R, Lesser ME, Plotkin P, et al. Atrial parasystole with interpolation: Observations on prolonged sinoatrial conduction. *Am Heart J* 1962;63:649-658.
4. Strauss HC, Geer MR. Sinoatrial node re-entry. In Kulbertus HE (ed.): *Re-entrant Arrhythmias: Mechanisms and Treatment*. Lancaster, MTP Press, 1977, pp. 27-38.
5. Fraticelli A, Saccomanno G, Pappone C, et al. Paroxysmal supraventricular tachycardia caused by 1:2 atrioventricular conduction in the presence of dual atrioventricular nodal pathways. *J Electrocardiol* 1999;32:347-354.