Rapidly Recurring Supraventricular Tachycardia

A Manifestation of Reciprocating Tachycardia and an Indication for Propranolol Therapy

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SUMMARY

Reciprocating tachycardias were diagnosed on the basis of previously established ECG criteria in seven patients with recurrent tachycardia but without WPW. In each patient we could record the onset of arrhythmia because tachycardia recurred within minutes of its termination. Treatment with propranolol alone or with digitalis, or digitalis and quinidine, prevented tachycardia in five patients and slowed it in one. Our cases suggest the following: (1) Recurrent supraventricular tachycardia in patients without the WPW syndrome may frequently be caused by a reciprocal mechanism. (2) The diagnosis of reciprocating tachycardia should be suspected when (a) the tachycardia recurs within minutes of its termination, (b) the onset follows prolongation of the P-R or R-P interval, (c) the P wave during the tachycardia is superiorly directed and different from the P wave which initiates the tachycardia, and (d) at least one other criterion for the diagnosis is met. (3) Propranolol is the drug of choice for the treatment of recurrent supraventricular tachycardias which satisfy the criteria for reciprocating tachycardia.

Additional Indexing Words:
Antiarrhythmic therapy Arrhythmia Beta blockers Dual pathways
ECG Echo beats Re-entry phenomenon Digitalis Quinidine

Reciprocating tachycardia of sinus or atrial origin occurs when an impulse arising in the sinus node or in an ectopic atrial focus traverses the atrioventricular (A-V) connection in an orthograde direction to excite the ventricles and is reflected in a retrograde direction at some point within the A-V junction or common His bundle to reactivate the atria. This type of tachycardia depends upon the existence of functionally discrete A-V pathways. It is now generally accepted that at least some of the supraventricular tachycardias associated with the Wolff-Parkinson-White (WPW) syndrome are reciprocating and occur when a supraventricular stimulus travels in an orthograde direction through the normal A-V junction and returns in a retrograde direction through the anomalous pathway to reactivate the atria and re-enter the A-V junction.1-9 The frequent occurrence of atrial and ventricular echo (reciprocal) beats9-14 as well as recent anatomic15, 16 and electrophysiologic studies17, 18 suggest that a separate anomalous pathway is not necessary for this type of tachycardia. Isolated cases of reciprocating tachycardia in patients without the WPW syndrome have been reported,10, 19-25 and electrocardiographic criteria for the diagnosis of reciprocating tachy-
cardia of sinus or atrial origin have been described. These include: (1) lengthening of the P-R or R-P interval at the onset of tachycardia,4 23 25 (2) a difference between the configuration of the P wave which initiates the tachycardia and the P waves recorded during the tachycardia;23 (3) the presence of retrograde P waves having the same configuration as the P waves recorded during the tachycardia;23 (4) the presence of atrial or ventricular echoes;9 10 11 (5) the resetting of the tachycardia by a ventricular ectopic beat;25 and (6) the termination of the tachycardia with both a retrograde P wave and with a QRS complex.4

On the basis of these criteria, we have diagnosed reciprocating tachycardia of sinus or atrial origin in seven patients without the WPW syndrome who were referred because of increasingly frequent attacks of paroxysmal supraventricular tachycardia which were refractory to treatment with digitalis and usually quinidine or procainamide. In each patient the tachycardia recurred within minutes of being terminated by carotid sinus massage or the intravenous infusion of pressor amines, and we were, therefore, able to record the onset and termination of the arrhythmia. Treatment with orally administered propranolol prevented the attacks of tachycardia in five of

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**Figure 1**

Leads I, II, and III of patient S.E. Paper speed, 25 mm per sec. A diagrammatic representation is below the ECG. Abbreviations in this and in subsequent figures: s = sinus node; a = atrium; a = atrioventricular junction; v = ventricle. The points of reflection within the A-V junction are schematic and are intended only as illustrations of the reciprocating mechanism rather than the actual re-entrant points. The second QRS complex is followed by an ectopic atrial P wave which is inverted in I and upright in II and III. The interval between the preceding sinus P wave and the ectopic atrial P is 0.58 sec, the P-R interval of the ectopic beat is 0.20 sec. The fourth QRS complex is followed by a similar ectopic P wave. The interval between the preceding sinus P and the ectopic P is 0.42 sec, the P-R interval of the ectopic P is 0.24 sec, the QRS complex is followed by a P wave which is inverted in I and isoelectric in III (superiorly directed), and tachycardia occurs. The first four QRS complexes during the tachycardia have a right bundle-branch block configuration and each QRS complex during the tachycardia is followed by a superiorly directed P wave.

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these patients and slowed the heart rate during the attack in one. Two of these cases have been previously reported. The purpose of this report is to demonstrate that recurrent supraventricular tachycardia in patients without the WPW syndrome may frequently be reciprocating and to stress the usefulness of propranolol in the treatment of this arrhythmia.

**Report of Cases**

**Case 1**

S.E. is a 25-year-old housewife with a 13-year history of paroxysmal tachycardia which was not prevented by treatment with digitalis and quinidine. Two weeks before admission the episodes of tachycardia increased in frequency and at the time of admission were almost continuous. Physical examination was normal, but roentgenologic examination of the chest revealed an enlarged heart. The electrocardiogram (ECG) revealed frequent atrial ectopic beats and repeated episodes of supraventricular tachycardia which began when an atrial ectopic beat occurred during the preceding T wave and was conducted to the ventricles with a P-R interval greater than 0.19 sec (figs. 1 and 2). Frequently, the first several beats of the tachycardia were conducted with aberration (fig. 1). The P waves during the tachycardia were inverted in leads I and II and flat in lead III (superiorly directed) and differed from the P wave which initiated the tachycardia. Termination of the tachycardia by carotid sinus massage was associated with a superiorly directed P wave or with a QRS complex (fig. 3). Treatment with digoxin, 0.25 mg per day, and quinidine sulfate, 200 mg every 6 hours, was not effective, but the addition of propranolol, 10 mg every 6 hours, resulted in a decreased frequency of both the episodes of tachycardia and the ectopic atrial beats. Episodes of tachycardia and ectopic atrial beats increased in frequency when propranolol was discontinued and were again suppressed when propranolol was reinstituted. The patient has had only rare, short episodes of tachycardia in the last 11 months.

**Case 2**

M.B. was a 52-year-old male in whom the diagnosis of paroxysmal tachycardia was first made in October 1968, 2 months prior to the replacement of a malfunctioning mitral valve prosthesis. The patient had been maintained on 0.25 mg of digoxin per day for several years. Postoperatively, junctional rhythm with superiorly directed P waves and paroxysms of supraventricular tachycardia were observed (fig. 4). The tachycardia began when an atrial ectopic beat occurred during the preceding T wave and was conducted with a P-R interval of 0.40 sec (figs. 4B and 5). The P waves during the tachycardia were superiorly directed, as were the P waves which followed the junctional beats. These P waves differed in configuration from the atrial ectopic P waves which initiated the tachycardia. Termination of the tachycardia by carotid sinus massage was associated with a superiorly directed P wave (fig. 6A) and with a QRS complex (fig. 6B), but sinus rhythm could not be maintained. Following the intravenous administration of 2 mg of propranolol, tachycardia did not recur for more than 2 hours although the atrial ectopic beats continued. The patient was treated with orally administered propranolol, 5 mg every 6 hours,

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**Figure 2**

*Scattergram of ectopic atrial premature beats followed by tachycardia (open squares), and those not followed by tachycardia (closed circles) in patient S.E. The interval from the preceding sinus P to the ectopic P (P'-P) is on the vertical axis and the interval from the ectopic P to the following QRS complex (P'-R) is on the horizontal axis. Note the clear separation between the two types of beats. Those followed by tachycardia occur earlier and/or are conducted with a longer P'-R interval than those not followed by tachycardia.*

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and 0.25 mg of digoxin per day, and remained free of tachycardia. Propranolol was discontinued 6 weeks later without recurrence of tachycardia.

**Case 3**

H.D., a 54-year-old retired miner, gave a history of shortness of breath and paroxysmal tachycardia of 1 year’s duration which was not prevented by treatment with 0.25 mg of digoxin per day and quinidine sulfate, 200 mg four times per day. The tachycardia was associated with chest pain and was often precipitated by exercise. The patient slept on two pillows and complained of paroxysmal nocturnal dyspnea and ankle swelling. Physical examination, x-ray examination of the chest, and pulmonary function tests were compatible with obstructive airway disease, but examination of the heart was normal. The ECG showed atrial and ventricular ectopic beats (fig. 7A and B) and atrial echoes or paroxysms of supraventricular tachycardia which occurred.
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when an atrial ectopic beat arose during the previous T wave and was conducted to the ventricles with a P-R interval of 0.20 sec (fig. 7A and C). The retrograde P waves of the atrial echoes and those occurring after the ventricular ectopic beats were superiorly directed and similar to the P waves which occurred during the tachycardia. These P waves differed from the atrial ectopic P waves which initiated the tachycardia. Sinus rhythm could be restored by carotid sinus massage and by the intravenous infusion of metaraminol but could not be maintained. Following the intravenous administration of 2 mg of propranolol, the tachycardia did not recur for 4 hours. The oral administration of 10 mg of propranolol four times daily completely suppressed the tachycardia and the atrial echoes although the atrial ectopic beats persisted. Tachycardia recurred when the dose of propranolol was decreased to 10 mg three times daily. The patient has been free of attacks for 12 months.

Case 4

The patient, W.W., was a 58-year-old male, with a 1-year history of paroxysmal tachycardia which increased in frequency and severity in the 2 weeks prior to his evaluation. The attacks were associated with shortness of breath and were not prevented by treatment with 0.25 mg of digoxin per day and quinidine sulfate, 200 mg four times per day. Physical examination and x-ray examination of the chest were normal. The glucose tolerance test was characteristic of diabetes mellitus, and pulmonary function studies were consistent with obstructive airway disease. The ECG showed frequent atrial ectopic beats and paroxysmal tachycardia which began when an atrial ectopic beat occurred within the previous T wave and was conducted with a P-R interval longer than 0.20 sec (fig. 8). The P waves observed during the tachycardia were superiorly directed and differed from the atrial ectopic P waves which initiated the tachycardia. Superiorly directed P waves were also observed following ventricular ectopic beats. After institution of therapy with orally administered propranolol, 10 mg four times per day, the attacks of tachycardia ceased and quinidine was discontinued. Subsequent ECGs showed atrial ectopic beats. This patient has been free of attacks of tachycardia for 24 months.

Case 5

J.P., a 28-year-old male, had a 19-year history of recurrent paroxysmal tachycardia which was unresponsive to treatment with various combinations of digitalis, quinidine sulfate, procainamide, diphenylhydantoin, prostigmine, atropine sulfate, reserpine, and phenobarbital. The tachycardia occurred both at rest and after exercise and was associated with shortness of breath and occasional

Figure 5

Lead V1 and esophageal lead (VES) of patient M.B. recorded at onset of tachycardia. Paper speed, 50 mm per sec. An ectopic atrial P wave distorts the T wave of the first QRS complex, and is conducted to the ventricles with a P-R interval of 0.40 sec. The second QRS complex is followed by a retrograde P wave which, in turn, is conducted to the ventricles and the reciprocating tachycardia is established.

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Figure 6

Lead V1 and esophageal lead VES of patient M.B. recorded on two occasions during the termination of the tachycardia by carotid sinus massage. Paper speed, 50 mm per sec. In A, the tachycardia terminates with a retrograde P wave. The fifth and sixth complexes are junctional beats and are followed by retrograde P waves. In B, the tachycardia terminates with a QRS complex. The fourth and fifth QRS complexes are junctional beats but are not followed by a retrograde P wave. These tracings indicate that in A, orthograde conduction is blocked and in B, retrograde conduction is blocked.

syncope. Although there was no history of heart disease, the first episode of tachycardia occurred shortly after the patient had had pertussis. Physical examination and x-ray examination of the chest were normal, but the ECG was abnormal (fig. 9). Atrial echoes and supraventricular tachycardia followed the prolongation of the P-R interval which occurred as the result of abortive Wenckebach periods (figs. 10 and 11). The tachycardia could be terminated by carotid sinus massage but recurred almost immediately. The intravenous administration of 10 mg of propranolol slowed the heart rate from 160 to 120 beats per min by prolonging the P-R interval from 0.20 to 0.28 sec but did not suppress the tachycardia. Tachycardia also continued to occur after treatment with orally administered propranolol in combination with digitalis and other antiarrhythmic drugs. However, we were unable to administer more than 60 mg of propranolol per day because of paresthesias and vertigo.

Cases 6 and 7

These cases have been previously reported. In case 6 (F.P.), atrial echoes occurred when the P-R interval was normal (0.14 sec) and tachycardia occurred when the R-P interval of the atrial echo was 0.16 sec or longer. Junctional and ventricular beats followed by retrograde P waves identical to the P waves during the tachycardia were also observed. The intravenous administration of 10 mg of propranolol did not terminate the tachycardia but slowed the heart rate from 200 to 160 beats per min by prolonging the P-R interval.
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A.

Figure 7

Lead V1 of patient H.D. Paper speed, 50 mm per sec. In A, an upright ectopic atrial premature P wave distorts the T wave of the first QRS complex but is not conducted to the ventricles. An upright ectopic P wave also distorts the T wave of the third QRS complex. It occurs later than the first ectopic P wave and is conducted to the ventricles with a P-R interval of 0.20 sec. An inverted P wave (atrial echo) follows fourth QRS complex, but tachycardia does not occur. In B, the second QRS complex is ectopic and is followed by an inverted (retrograde) P wave. In C, an upright ectopic atrial P, similar to the ectopic P wave in A, distorts the T wave of the first QRS complex and is conducted to the ventricles with a P-R interval of 0.20 sec. The QRS complex after the ectopic P (second QRS) is followed by an atrial echo which is conducted to the ventricles and the tachycardia is established. Note the inverted P waves during tachycardia.

and prevented the recurrence of tachycardia after its termination by the intravenous injection of 2 mg of phenylephrine. The tachycardia was prevented by the oral administration of 10 mg of propranolol four times per day, and except for brief periods when propranolol has been temporarily discontinued, the patient has been free of attacks for 5 years.

In case 7 (N.T.), supraventricular tachycardia began when a normally conducted sinus beat (P-R = 0.16 sec) was followed by a superiorly directed P wave with an R-P interval of 0.32 to 0.36 sec. In this patient, the tachycardia was reset by ventricular ectopic beats.25 Intravenously and orally administered propranolol did not prevent the tachycardia but slowed the heart rate from 120 to 90 beats per min by prolonging both the P-R and R-P intervals.

Discussion

We have reviewed two previously reported cases and presented five additional cases with recurrent paroxysmal supraventricular tachycardia but without the WPW syndrome, in which a diagnosis of reciprocating tachycardia of sinus or atrial origin was made on
the basis of previously established ECG criteria. In each of the seven patients we were able to record the onset and termination of tachycardia because, in each, tachycardia recurred within minutes of being stopped by carotid sinus massage or by the intravenous administration of pressor amines. In four of our patients, the episodes of tachycardia predated the first evaluation at our hospital by more than 10 years, and in the other three patients, by more than 1 year. Underlying heart disease was diagnosed in only two patients (M.B. and J.P.), but was suspected in two others (S.E. and H.D.). Two of the patients (H.D. and W.W.) had moderately severe obstructive airway disease, and two (W.W. and F.P.) had diabetes mellitus. All of our patients had been treated unsuccessfully with digitalis and, in five, treatment with quinidine or procainamide, or both, had been either poorly tolerated by the patient or had not prevented the tachycardia. Two patients had not been treated with quinidine or procainamide.

In table 1, the six criteria for the diagnosis of reciprocating tachycardia have been divided into three categories signifying: (1) the unmasking of a second A-V junctional pathway and recovery of the atrial connection

**Table 1**

<table>
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<tr>
<th>Case no. (patient)</th>
<th>Category I*</th>
<th>Category II†</th>
<th>Category III‡</th>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>4</td>
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<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>4</td>
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<tr>
<td>6 F.P.</td>
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<td>Total</td>
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</table>

*Category I: Criterion suggesting the unmasking of a second A-V junctional pathway and recovery of the atrial connection.
†Category II: Criteria suggesting that tachycardia due to repetitive ectopic focus is unlikely, but that retrograde atrial activation during tachycardia is likely.
‡Category III: Criteria suggesting the presence of two A-V junctional pathways.
§VEB = ventricular ectopic beat.
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Figure 9
ECG of patient J.P. The rhythm is sinus with a prolonged (0.22 sec) P-R interval, and the P wave is notched in leads II, V3, V5, and V6. There is left axis deviation, an S wave in leads V5 and V6, and a Q wave in leads I, AVL, V5, and V6. The tracing is interpreted as showing an interatrial conduction disturbance, first degree A-V block, and an intraventricular conduction disturbance of the left anterior hemiblock type. It is also suggestive of a lateral infarction.

Figure 10
Lead V1 of patient J.P. Paper speed, 25 mm per sec. The P-R interval after the first, third, and fifth P waves is 0.18 sec and after the second, fourth and sixth P waves, 0.24 sec. An inverted (echo) P wave follows the second, fourth, and sixth QRS complexes.

between the pathways; (2) a greater likelihood that the tachycardia was the result of a reciprocal mechanism than the result of a repetitive ectopic pacemaker and that the atria were activated in a retrograde direction; and (3) the presence of functionally discrete pathways within the A-V junction. In the first category is the initiation of tachycardia following prolongation of the P-R or the R-P interval; in the second, the difference between the P waves initiating the tachycardia and those recorded during the tachycardia, and the similarity of the P waves recorded during the tachycardia to retrograde P waves; and in the third, the presence of atrial or ventricular echoes, the resetting of the tachycardia by a ventricular premature beat, and the termination of tachycardia with both a retrograde P wave and with a QRS complex in the same patient. Table 1 shows that in all patients, tachycardia followed prolongation of the P-R or R-P interval and the P waves recorded during the tachycardia were superiorly directed and different from the P waves which initiated the tachycardia. These criteria would, therefore, appear to be the sine qua
non for the diagnosis of reciprocating tachycardias of sinus or atrial origin. Table 1 also shows that in each patient at least one other criterion was met, and that in all but one patient at least one criterion in each of the three categories was satisfied.

In four of our patients, tachycardia was initiated by an atrial ectopic beat which occurred during the previous T wave and was conducted to the ventricles with a long P-R interval. In this way, these cases are similar to previously reported cases and experimental studies in which reciprocal beats and rhythms occurred spontaneously or followed premature electrical stimulation of the atria. In the other three patients, the arrhythmia followed a sinus beat. In one of these patients, as in previously reported cases, tachycardia occurred when the P-R interval was prolonged, while in the other two patients, tachycardia occurred when the R-P interval was prolonged and in this way resembled the case reported by Codina-Altes and Pijoan de Beristain.

The intravenous administration of 2 to 10 mg of propranolol in three patients did not terminate the tachycardia but resulted in prompt cessation of attacks. The oral administration of 20 to 60 mg of propranolol per day prevented the tachycardia in all four patients in whom tachycardia followed an atrial ectopic beat and in one of the three patients in whom it followed a sinus beat. In four of these five patients, propranolol may have prevented conduction through the retrograde pathway because in three, the tachycardia, but not the ectopic beats, was suppressed and in one (in whom tachycardia followed a sinus beat), echo beats were also prevented. In the fifth patient, propranolol suppressed both the tachycardia and the ectopic beats. Propranolol did not prevent the tachycardia in two patients whose tachycardia followed a sinus beat, but in one it slowed the rate of the tachycardia by slowing both orthograde and retrograde conduction and in so doing decreased the symptoms associated with the arrhythmia. In two of the six patients in whom the tachycardia was either prevented or slowed, propranolol alone was effective. In the other four patients, propranolol was administered in combination with digitalis (three

**Figure 11**

*Lead V₁ and esophageal lead Vₑₛ of patient J.P. recorded at the onset of an episode of tachycardia. Paper speed, 50 mm per sec. The P-R interval preceding the second QRS complex is 0.22 sec. This QRS complex is followed by an inverted (retrograde) P wave with an R-P interval of 0.16 sec which, in turn, is conducted to the ventricles with a P-R interval of 0.30 sec. The third QRS complex is also followed by a retrograde P wave and the tachycardia is established.*
patients) or with digitalis and quinidine (one patient). In these, as in patients with other types of arrhythmias,20,31 the combination of drugs may have been more effective than therapy with propranolol alone. However, in each of these patients prior therapy with digitalis or digitalis and quinidine was not effective, and the arrhythmia was prevented only after the administration of propranolol.

Our patients are similar to patients with the WPW syndrome in that in both groups, the tachycardias are recurrent, are frequently refractory to treatment with digitalis and quinidine, may be attributed to a reciprocating mechanism, and are often suppressed by propranolol therapy.24,32,33 The effectiveness of propranolol in both groups of patients supports our earlier suggestion24 that reciprocating tachycardias may be a specific indication for propranolol therapy.

Addendum

Since this manuscript was submitted for publication, Goldreyer and Bigger presented eight patients with recurrent supraventricular tachycardia without WPW (Circulation 40 [suppl. III]: III-92, 1969, abstr.) in whom a diagnosis of reciprocating tachycardia of atrial origin was confirmed by recording His bundle activity. In these patients tachycardia could be induced by a single atrial premature stimulus from a pacing catheter. These patients were similar to ours and confirm the hypothesis that a reciprocating mechanism may frequently be the cause of recurrent supraventricular tachycardia.

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