Mechanisms of Spontaneous Alternation between Reciprocating Tachycardia and Atrial Flutter-Fibrillation in the Wolff-Parkinson-White Syndrome

RUEY J. SUNG, M.D., AGUSTIN CASTELLANOS, M.D., STEPHEN M. MALLON, M.D., MARTIN G. BLOOM, M.D., HENRY GELBAND, M.D., AND ROBERT J. MYERBURG, M.D.

SUMMARY In a group of 36 consecutive patients with the Wolff-Parkinson-White (WPW) syndrome undergoing electrophysiological studies because of paroxysms of reciprocating tachycardia (RT) and/or atrial flutter-fibrillation (AF), 7 patients (19%) had repeated episodes of spontaneous alternation between RT and AF. Electrophysiological studies demonstrated left-sided anomalous pathways (AP) in all 7 patients. Atrial vulnerability, as evidenced by the occurrence of repetitive atrial responses or a paroxysm of AF following a single atrial prematurity stimulus, was also noted in all. Invariably, spontaneous conversion of RT to AF (7 patients) was triggered by an atrial prematurity depolarization which resulted in atrial asynchrony during the atrial vulnerable phase. In contrast, spontaneous conversion of AF to RT (3 of the 7 patients) required the presence or the development of antegrade unidirectional block in the AP prior to the cessation of AF.

The demonstration of atrial vulnerability in association with the phenomenon of spontaneous alternation between RT and AF provides further information pertaining to the understanding of the mechanisms of tachyarrhythmias in the WPW syndrome. It is suggested that the occurrence of this electrophysiological phenomenon may be more common than is generally appreciated, and optimal medical treatment should be directed toward controlling both RT and AF in this group of Wolff-Parkinson-White patients.

TACHYARRHYTHMIAS, predominantly supraventricular tachycardia and atrial flutter-fibrillation, are a key feature of the clinical syndrome described by Wolff, Parkinson, and White (WPW). The frequent episodes of supraventricular tachycardia are generally a form of reciprocating tachycardia utilizing both normal and anomalous atrivo-ventricular (A-V) pathways. On the other hand, the occurrence of atrial flutter-fibrillation in association with this syndrome is not well understood and may, in some instances, be related to coexisting sinus nodal and intraatrial disease. Spontaneous conversion between reciprocating tachycardia and atrial flutter-fibrillation has also been described in patients with the WPW syndrome; however, little information is available concerning its underlying mechanisms.

In seven of the 36 consecutive patients with the WPW syndrome undergoing electrophysiological studies, spontaneous conversion between reciprocating tachycardia and atrial flutter-fibrillation was repeatedly observed. The purpose of this paper is to describe the mechanisms responsible for this unique electrophysiological phenomenon and to discuss its possible clinical implications.

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TABLE 1. Clinical Data

<table>
<thead>
<tr>
<th>Case No.</th>
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<th>ECG documented arrhythmias</th>
<th>Associated cardiovascular diseases</th>
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<td>1 (H.J.)</td>
<td>20M</td>
<td></td>
<td>AF</td>
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</tr>
<tr>
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<td></td>
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<td>71F</td>
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<td></td>
<td>RT</td>
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<td>AF &amp; RT</td>
<td>Rheumatic heart disease (mitral stenosis)</td>
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<td>7 (R.A.)</td>
<td>33M</td>
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<td>AF</td>
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Abbreviations: AF = atrial flutter-fibrillation; RT = reciprocating tachycardia.

Materials and Methods

Of the 36 consecutive WPW patients undergoing electrophysiological studies in our laboratory, the phenomenon of spontaneous conversion of reciprocating tachycardia to atrial flutter-fibrillation, or vice versa, was observed in seven (19%). Clinically, all seven patients presented with recurrent tachyarrhythmias (table 1); three experienced paroxysms of atrial flutter-fibrillation, two reciprocating tachycardia, and two both atrial flutter-fibrillation and reciprocating tachycardia. There were four males and three females ranging in age from 20 to 71 years. Subsequent electrophysiological evaluation suggested that all seven patients had anomalous A-V pathways (Kent bundle) connecting the left atrium and the left ventricle.10-12 One (case 4) of these patients has been recently reported.9

After obtaining an informed consent, all cardiotonic and antiarrhythmic medications were discontinued 48 to 72 hours prior to the study. The study was performed in a postabsorptive, nonsedated state. High right atrial (HRA) activity was recorded through the proximal pair of electrodes of a hexapolar electrode catheter, and the left atrial (LA) electrogram was recorded with a quadripolar electrode catheter placed in the coronary sinus or in the left atrium through a patent foramen ovale. Using a conventional technique,14 the His bundle electrogram (HBE) was obtained through a tripolar electrode catheter, from which low septal right atrial (LRA) activity was recorded as well. The intracardiac electrograms were then displayed simultaneously with standard ECG leads I, II, and V1 on a multichannel oscilloscopic photographic recorder (Electronics for Medicine, DR-16) at a paper speed of 100 or 150 mm/sec, using filter setting between 40 and 500 Hz.

Through the remaining pairs of electrodes of the hexapolar and quadripolar electrode catheters, both the atria and the right ventricular apical endocardium were stimulated at one or two cycle lengths (A1-A2 or V1-V2) via a programmed digital stimulator which delivered stimuli (S1 and S2) of 2.0 msec duration at approximately twice diastolic threshold.13 Following every eighth spontaneous or paced beats (A1 or V1), single premature atrial or ventricular beats (A2 or V2) were delivered at progressively shorter coupling intervals until the effective refractory period (ERP) of the atrium or the ventricle was encountered.

The presence of a left-sided anomalous pathway in each patient was suggested when the following observations occurred:15-18 1) programmed left atrial (LA) stimulation induced a tall R wave in lead V1 during maximal ventricular pre-excitation and/or 2) the sequence of retrograde atrial activation during reciprocating tachycardia and/or induced by programmed ventricular stimulation indicated that left atrial (LA) activation preceded low septal right atrial (LRA) (in HBE) and high right atrial (HRA) activation.

For the purpose of this study, the following pertinent electrophysiological data were analyzed (table 2):

1) Effective refractory period of the atrium (ERP_A1): the longest S1-S2 interval that failed to generate an atrial response.

2) Functional refractory period of the atrium (ERP_A0): the shortest attainable A1-A2 interval during programmed atrial stimulation.

3) Antegrade effective refractory period of the anomalous pathway (ERP_A):18,19 the longest A1-A2 interval near the anomalous pathway which failed to conduct with a ventricular pre-excitation pattern.

4) Atrial vulnerability: an atrial premature stimulus (S2), delivered close to the ERP of the atrium (ERP_A) (within its relative refractory period), may elicit short-lived repetitive atrial responses (mean atrial cycle length of 300 msec or less) or a paroxysm of atrial flutter-fibrillation (mean atrial cycle length of less than 250 msec) (fig. 1A).15-18 The occurrence of this phe-

**Table 2. Pertinent Electrophysiological Data**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>AP loc</th>
<th>DCL (mph)</th>
<th>ERP_A1 (msec)</th>
<th>ERP_A0 (msec)</th>
<th>Atrial vulnerability LA</th>
<th>Antegrade ERP_A (msec)</th>
<th>RT induction during PAS</th>
<th>AF induction during PAS</th>
<th>Spontaneous conversion RT → AF</th>
<th>AF → RT</th>
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<td>-</td>
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<td>+</td>
<td>-</td>
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<td>275</td>
<td>+</td>
<td>UDB</td>
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<td>220</td>
<td>290†</td>
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<td>+</td>
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</table>

+Paroxysm of atrial flutter-fibrillation induced by single atrial premature stimuli.
†Sinus cycle length.

Abbreviations: AP loc = anomalous pathway location; DCL = driving cycle length; ERP = effective refractory period; FRP = functional refractory period; AT = atrium; RA = right atrium; LA = left atrium; PAS = programmed atrial stimulation; PVS = programmed ventricular stimulation; RT = reciprocating tachycardia; AF = atrial flutter-fibrillation; + = present; - = absent; UDB = unidirectional block; L = left-sided.
nomenon was referred to herein as atrial vulnerability.16-18

Following the completion of the above study protocol, reciprocating tachycardia or atrial flutter-fibrillation was induced by a single premature stimulus (S<sub>1</sub>) on either programmed atrial or programmed ventricular stimulation study. Observations on the arrhythmia were then continued for approximately five minutes, during which time the patient's blood pressure was monitored at one minute intervals. Electrical interventions such as atrial or ventricular premature stimulation, continuous atrial or ventricular pacing, and electrical countershock (if necessary) were applied to terminate the arrhythmia when the patient became symptomatic or at the end of each observation period. Two patients (cases 1 and 7) required electrical countershock because of sustained atrial flutter-fibrillation (>5 min duration) with a rapid ventricular rate. The course during the study was otherwise uneventful in all patients.

Results
Atrial Vulnerability and Atrial Flutter-Fibrillation

During programmed atrial stimulation, repetitive atrial responses induced by single premature stimuli could be demonstrated in all seven patients (table 2). In general, the right atrium appeared to be more vulnerable than the left atrium. Only one patient (who had rheumatic mitral stenosis) had clinical evidence of atrial enlargement (case 5, table 1). Paroxysms of atrial flutter-fibrillation, triggered by

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**Figure 1.** A) Initiation of a paroxysm of atrial flutter-fibrillation (AF) following an atrial premature stimulus (case 1). S = stimulus; A = atrial electrogram; H = His bundle potential; HRA = high right atrial lead; LA = left atrial lead; HBE = His bundle electrographic lead. At an atrial driving cycle length (S<sub>2</sub>-S<sub>1</sub>) of 475 msec, an atrial premature stimulus (S<sub>1</sub>) with a coupling interval (S<sub>1</sub>-S<sub>2</sub>) of 230 msec (corresponding A-A interval of 270 msec) elicits a paroxysm of AF (mean atrial cycle length = 210 msec). During AF, the anomalous pathway maintains antegrade conduction for most or all propagated beats. Note the ventricular electrograms in HBE lead are clearly discernible; however, the atrial electrograms are fractionated in all three intracardiac leads during AF. These abbreviations will be used in the subsequent figures. B) Spontaneous conversion of reciprocating tachycardia (RT) to atrial flutter-fibrillation (AF) triggered by an atrial premature depolarization (same patient as in figure 1A). The high right atrial depolarization (A-A interval of 270 msec) following the fifth QRS complex (solid arrow) is premature with respect to the expected reciprocating rhythm (A-A interval of 330 msec) during RT. The corresponding A-H interval is lengthened from 120 to 200 msec following the atrial premature depolarization. A paroxysm of AF with a mean atrial cycle length of 230 msec is then initiated. The anomalous pathway maintains antegrade conduction for most propagated beats during AF. Note left atrial activation precedes low septal right atrial (in HBE lead) and high right atrial activation during RT, suggestive of the presence of a left-sided anomalous pathway. The atrial electrograms during AF are fairly regular in the LA lead but are fractionated in both HRA and HBE leads.
single premature atrial stimuli, occurred in five patients (cases 1, 4–7). Four of these five patients had at least one episode of atrial flutter-fibrillation documented on ECG prior to the study (table 1). Paroxysms of atrial flutter-fibrillation were more easily elicited at shorter atrial driving cycle lengths (400–600 msec) (table 2). The induction of a paroxysm of atrial flutter-fibrillation following a single premature stimulus (S₂) is exemplified in figure 1A (case 1). At an atrial driving cycle length (S₁–S₁) of 475 msec, a premature stimulus (S₂) with a coupling interval (S₁–S₂) of 230 msec (corresponding A₂–A₂ interval = 270 msec) initiated the onset of an atrial flutter-fibrillation (mean atrial cycle length = 210 msec). During atrial flutter-fibrillation, the atrial electrograms were fractionated and the anomalous pathway maintained antegrade conduction for most or all propagated beats. In no instance could repetitive atrial responses or atrial flutter-fibrillation be elicited during programmed ventricular stimulation in these seven patients.

Induction of Reciprocating Tachycardia

During programmed atrial stimulation, reciprocating tachycardia could be elicited in four (cases 2, 3, 4 and 6) of these seven patients. Two (cases 3 and 4) of these four patients manifested antegrade unidirectional block in the anomalous pathway. At short premature atrial coupling intervals (S₂–S₁ of 190 to 310 msec at cycle lengths of 400 to 780 msec), the development of repetitive atrial responses or atrial flutter-fibrillation (atrial vulnerability) prevented initiation of reciprocating tachycardia in these four patients, owing to the atrial cycle length of repetitive atrial responses (< 300 msec) or atrial flutter-fibrillation (≤ 250 msec) being shorter than those of reciprocating tachycardia (330–290 msec). In the remaining three patients (cases 1, 5 and 7) in whom reciprocating tachycardia could not be elicited by atrial extrastimuli, the antegrade effective refractory period (ERP) of the anomalous pathway was shorter than the functional refractory period (FRP) of the atrium and its precise measurement was limited by atrial refractoriness (the ERP of the atrium). However, induction of reciprocating tachycardia was possible during programmed right ventricular stimulation in the latter three patients (table 2). During programmed ventricular stimulation, ventriculo-atrial conduction in all seven patients was exclusively or predominantly through the anomalous pathway and the initiation of reciprocating tachycardia was possible in all but one patient (case 4).

Spontaneous Conversion of Reciprocating Tachycardia to Atrial Flutter-Fibrillation

During periods of observation, spontaneous conversion of reciprocating tachycardia to atrial flutter-fibrillation was noted in all patients (table 2). Invariably, the precipitating event was an atrial premature depolarization which resulted in the abrupt transition of a reciprocating tachycardia to a paroxysm of atrial flutter-fibrillation. One example is shown in figure 1B (case 1). Following the fifth QRS complex, a reciprocating tachycardia with a cycle length of 330 msec suddenly converted to a paroxysm of atrial flutter-fibrillation with a mean atrial cycle length of 230 msec. During the reciprocating tachycardia, the sequence of retrograde atrial activation was the left atrium (LA) followed by the low septal right atrium (LRA) (in HBE) and the high right atrium (HRA), indicative of the presence of a left-sided anomalous pathway. The abrupt conversion of reciprocating tachycardia to atrial flutter-fibrillation was apparently triggered by a premature depolarization of the right atrium following the fifth QRS complex. Note appearance of high right atrial (HRA) electrogram 60 msec earlier than it would have been expected to occur (A–A interval of 270 msec vs 330 msec during reciprocating tachycardia). The appearance of low septal right atrial (LRA) electrogram (in HBE) was also premature. The premature activation of the right atrium was accompanied by sudden prolongation of the corresponding A–H interval (A–V nodal conduction time) from 120 to 200 msec. During atrial flutter-fibrillation, the left atrial (LA) electrogram remained fairly regular, and the high right atrial (HRA) and low septal right atrial (LRA) (in HBE) electrograms became fractionated. Further examples of such a phenomenon are illustrated in figures 2 and 3, in which spontaneous conversion of reciprocating tachycardia to atrial flutter-fibrillation was related to a premature depolarization originating in the left atrium (LA) and in the low septal right atrium (LRA) (in HBE), respectively. The abrupt transition from a reciprocating tachycardia to an atrial flutter-fibrillation precipitated by an atrial premature depolarization resembled the onset of repetitive atrial responses or atrial flutter-fibrillation induced by a single atrial premature stimulus as illustrated in figure 1A. Therefore, it was postulated that the occurrence of an atrial premature depolarization which resulted in atrial asynchronous during the atrial vulnerable phase was the triggering mechanism responsible for conversion of reciprocating tachycardia to atrial flutter-fibrillation.

Spontaneous Conversion of Atrial Flutter-Fibrillation to Reciprocating Tachycardia

In three (cases 3, 4, and 6) of the seven patients, spontaneous conversion of atrial flutter-fibrillation to reciprocating tachycardia was observed (table 2). Of these three patients, two (cases 3 and 4) had anomalous pathways capable only of retrograde conduction. The presence or the development of antegrade unidirectional block in the anomalous pathway prior to the cessation of atrial flutter-fibrillation was a condition for its conversion to reciprocating tachycardia (figs. 4 and 5). For example, in figure 4 (case 6), a premature depolarization of the left atrium following the fourth QRS complex converted a reciprocating tachycardia (cycle length of 375 msec) to a short run of atrial flutter-fibrillation (mean atrial cycle length of 240 msec), which then spontaneously converted back to reciprocating tachycardia. During atrial flutter-fibrillation, two fusion beats (the 7th and 8th QRS complexes) (antegrade conduction over both normal and anomalous pathways) were noted. However, prior to the cessation of atrial flutter-fibrillation, the last three QRS complexes (the 9th, 10th, and 11th QRS complexes) were conducted exclusively through the normal pathway as evidenced by normalization of the H–V interval and disappearance of ventricular pre-excitation pattern (note complete left bundle branch block pattern during reciprocating tachycardia). Under these circumstances, the development of antegrade unidirectional
block in the anomalous pathway allowed the impulse of the last atrial flutter-fibrillation conducted beat (11th QRS complex) to enter the anomalous pathway in a retrograde direction and subsequently to reactivate the left atrium initiating an onset of reciprocating tachycardia. A decrease in the retrograde refractory period of the anomalous pathway and/or the atrium as a result of cycle length shortening, and a decrease in the depth of antegrade penetration of the anomalous pathway during atrial flutter-fibrillation might also be expected to facilitate retrograde excitation of the anomalous pathway and reactivation of the corresponding atrium. As expected, this phenomenon occurred in both patients (cases 3 and 4) with anomalous pathways capable only of retrograde conduction (fig. 5).

Discussion

Atrial Vulnerability and Atrial Flutter-Fibrillation

Atrial flutter-fibrillation occurs in association with a variety of disease states. In reference to its association with the WPW syndrome, Scherf and Cohen stated that a reciprocating rhythm involving both normal and anomalous
A-V pathways could not explain the occurrence of atrial flutter-fibrillation in patients with the WPW syndrome, while Castillo and Castellanos suggested that it might be related to a co-existing atrial disease. Dreifus et al. subsequently disclosed the findings of widespread destruction and a marked increase in connective tissue fibers in the sinus node and the atria in a patient with the WPW syndrome who manifested atrial flutter-fibrillation prior to his sudden death. The observation that atrial vulnerability could be demonstrated in these seven patients, in whom five (cases 1, 2, 5-7) experienced paroxysms of atrial flutter-fibrillation clinically (table 1), and all developed spontaneous conversion of reciprocating tachycardia to atrial flutter-fibrillation (table 2 and figs. 1-4) was noteworthy. Whether the presence of atrial vulnerability can be used as an index for identifying intraatrial disease is only conjectural. Further clinical and experimental studies are necessary.

**Conversion of Reciprocating Tachycardia to Atrial Flutter-Fibrillation**

Spontaneous conversion of reciprocating tachycardia to atrial flutter-fibrillation, as demonstrated in the present study, is triggered by an atrial premature depolarization which results in atrial asynchrony during the atrial vulnerable phase (figs. 1-4). Recently Wyndham et al. have demonstrated that atrial vulnerability is related to the atrial cycle length in man; shortening of the atrial cycle length potentiates atrial vulnerability. Our observation that it was easier to induce a paroxysm of atrial flutter-fibrillation by a single atrial premature stimulus at short atrial driving cycle lengths (400-600 msec) (table 2 and fig. 1) is in keeping with their findings. Furthermore, the cycle lengths during the paroxysms of reciprocating tachycardia were generally shorter than those chosen for programmed atrial stimulation (330-390 msec vs 400-780 msec in this study).

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**Figure 4.** Spontaneous conversion between reciprocating tachycardia (RT) and atrial flutter-fibrillation (AF) (same patient with a left-sided anomalous pathway as in figure 2). The left atrial depolarization (A-A interval of 280 msec) following the fourth QRS complex (solid arrow) is premature with respect to the expected reciprocating rhythm (A-A interval of 375 msec) during RT. The corresponding A-H interval is lengthened from 120 msec to 180 msec following the atrial premature depolarization. A short run of AF with a mean atrial cycle length of 240 msec is then initiated. During AF, the atrial electrograms are fairly regular in the HRA lead but are fractionated in both LA and HBE leads. The seventh and eighth QRS complexes are fusion beats (f) as evidenced by the inscription of delta wave (ventricular pre-excitation) before the His bundle potential (H). The eleventh QRS complex is the last AF conducted beat which then enters the left atrium (hollow arrow) initiating an onset of RT. Note prior to the conversion of AF to RT, the last three AF conducted beats (ninth, tenth and eleventh QRS complexes) are antegrade blocked in the anomalous pathway as evidenced by disappearance of the delta wave and normalization of the H-V interval (50 msec). A complete left bundle branch block pattern is present during RT.

**Figure 5.** Spontaneous conversion of atrial flutter-fibrillation (AF) to reciprocating tachycardia (RT). (Same patient with a left-sided anomalous pathway as in figure 3). The sixth QRS complex is the last AF conducted beat which then enters the left atrium (hollow arrow), initiating an onset of RT. A complete left bundle branch block pattern with a H-V interval of 55 msec is present during both AF and RT. Persistent antegrade unidirectional block in a left-sided anomalous pathway has been demonstrated during an electrophysiological study in this patient.
Therefore, it is conceivable that atrial vulnerability may be somewhat enhanced during reciprocating tachycardia. It has been demonstrated that atrial premature depolarizations may occur spontaneously during paroxysmal tachycardia. The genesis of these atrial premature depolarizations during reciprocating tachycardia in the WPW patients may be due to several possibilities: 1) spontaneous sinus nodal or an ectopic atrial or A-V junctional activity not suppressed by the reciprocating tachycardia; 2) presence of multiple anomalous A-V pathways allowing ventriculo-atrial conduction over different pathways during reciprocating tachycardia; 3) occurrence of sinus nodal or intra-atrial re-entry during reciprocating tachycardia; and 4) mechanical atrial irritation induced by electrode catheter movement during reciprocating tachycardia. Regardless of the mechanism of the atrial premature depolarization, it undoubtedly precipitates abrupt conversion of a reciprocating tachycardia to an atrial flutter-fibrillation.

Conversion of Atrial Flutter-Fibrillation to Reciprocating Tachycardia

Spontaneous conversion of atrial flutter-fibrillation to reciprocating tachycardia requires the presence or the development of antegrade unidirectional block in the anomalous pathway prior to the cessation of atrial flutter-fibrillation; the impulse conducting antegrade through the normal pathway enters the anomalous pathway in a retrograde direction and then reactivates the atrium establishing the circuit for a reciprocating tachycardia. As it would be expected, this phenomenon occurs more commonly in patients with anomalous pathways which are capable only of retrograde conduction. Under these circumstances, the depth of antegrade penetration of the anomalous pathway, the refractory period of the atrium and the anomalous pathway in a retrograde direction during atrial flutter-fibrillation will influence the occurrence of this phenomenon. A decrease in the depth of antegrade penetration of the anomalous pathway associated with shortening of the retrograde refractory periods of the anomalous pathway and the atrium favors the conversion of atrial flutter-fibrillation to reciprocating tachycardia following its spontaneous termination.

Clinical Implications

These observations have certain clinical implications. The occurrence of spontaneous conversion between reciprocating tachycardia and atrial flutter-fibrillation in the WPW patients may be more common than it is generally realized. Second, the electrophysiological phenomenon of conversion of reciprocating tachycardia to atrial flutter-fibrillation with a rapid ventricular response further identifies a potentially high risk group of patients with the WPW syndrome. Third, the optimal therapeutic approach in this group of patients should be directed toward controlling both reciprocating tachycardia and atrial flutter-fibrillation. To achieve this goal, combinations of antiarrhythmic drugs, such as quinidine (to prolong the refractory period of the anomalous pathway) and propranolol (to lengthen the refractory period of the A-V node) may be necessary. Digitalis alone may be contraindicated as it may shorten the refractory periods of both the atrium and the anomalous pathway. Lastly, pacemaker therapy is considered less desirable in this group of WPW patients, as either atrial or ventricular pacing may trigger an onset of atrial flutter-fibrillation in an attempt to control reciprocating tachycardia in WPW patients. Patients with anomalous pathways capable only of retrograde conduction are not likely to develop life-threatening, rapid ventricular rate during atrial flutter-fibrillation, however, induction of atrial flutter-fibrillation during pacemaker therapy may result in conversion to a reciprocating tachycardia following its termination, producing recurrent attacks of reciprocating tachycardia.

Acknowledgment

We express our appreciation to Francisco Garcia-Montes, B.S., for outstanding technical assistance, and to Mrs. Michelle Enriquez and Mrs. Patricia Zenoz for excellent secretarial work.

References

Serial Electrocardiograms in Hypertensive Cardiovascular Disease

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SUMMARY A graphic method for depicting serial changes in electrocardiograms is described and demonstrated for patients being treated in an antihypertension clinic. For these patients, the diagnostic categories, normal, left ventricular hypertrophy, and biventricular hypertrophy, are of primary interest. For each electrocardiogram, 14 measurements are used to compute posterior probabilities for each of the three categories. A triangular grid is used to plot each set of probabilities for an electrocardiogram as one point, which by its position in the triangle can be related to the three diagnostic categories simultaneously. Points representing successive electrocardiograms can be plotted in the same triangle, giving a pattern of change with time. This pattern of change has been corroborated with associated clinical information on a number of patients. This display, which can be produced quickly and efficiently on a computer graphics terminal, should be considered as a possible tool in evaluating the status of individual hypertensive patients in terms of increase or decrease of ventricular hypertrophy and the efficacy of therapeutic measures.

COMPARISONS between old and recent electrocardiographic records represent an essential part of electrocardiography. The lack of computer programs to perform such comparisons has often been quoted as a serious shortcoming of computerized electrocardiographic analysis. In recent years, however, several such programs have been reported.1,4 A major difficulty in their development has been the normal variability of the electrocardiogram from day to day or from year to year. For this reason, only changes in final diagnostic statements are being reported in some comparison programs. In others, changes in electrocardiographic measurements are added to the diagnostic statements if they exceed normal day-to-day variability. The extent of such variability has been well documented for orthogonal leads both in normals and abnormals.** In our Veterans Administration comparison program,4 changes in diagnostic statements are reported together with electrocardiographic measurement changes.

In certain types of heart disease, particularly those associated with ventricular hypertrophies, it would be desirable and helpful if serial changes in the electrocardiogram could be used as an indicator of the course of the disease where the patient's latest record is viewed in relation to previous ones. Based on observations by Poblete and coworkers,10 this should be feasible in cases with hypertensive cardiovascular disease (HCVD). When comparing records from patients on antihypertensive therapy with those in a placebo group, they found that the electrocardiographic changes in the treated patients showed a tendency toward normalization which was seen rarely in records of untreated patients who had either increased or unchanged signs of left ventricular hypertrophy (LVH). Results of this study and many other observations on dynamic changes of LVH signs in the electrocardiogram re-emphasize the fact that the electrocardiographic diagnosis of LVH is not an all-or-none statement. Most observers have felt that a relationship between the degree of LVH and its electrocardiographic characteristics must exist. However, as reviewed by Holt et al.,11 quantitative correlations have met with only moderate success. Of the many factors which may limit quantitative correlations, two appear most obvious. In patients with persisting left ventricular overload, hypertrophy develops, not

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