Differentiation Between Aberrant Ventricular Conduction and Ventricular Ectopy in Atrial Fibrillation Using RR Interval Scattergram

Akiko Chishaki Suyama, MD; Kenji Sunagawa, MD; Masaru Sugimachi, MD; Tsuyoshi Anan, MD; Kensuke Egashira, MD; Akira Takeshita, MD

Background. Differentiation between aberrant ventricular conduction and ventricular ectopy during atrial fibrillation (AF) is of etiologic, prognostic, and therapeutic importance. We developed a noninvasive technique to diagnose aberrant ventricular conduction and ventricular ectopy in AF.

Methods and Results. We studied the Holter ECGs of 34 patients with paroxysmal AF and 62 patients with chronic AF. In all the patients, frequent wide QRS complexes were observed, and 32 patients were shown by electrophysiological examination to have ventricular ectopies or aberrant ventricular conductions. We obtained the RR interval scattergrams by plotting sequential pairs of RR intervals. Each point has the (n)th RR interval as its x value and the (n+1)th RR interval as its y value. The irregularity of the RR intervals in AF resulted in widely scattered points delineated by the envelope along the axes. The y value of the envelope along the x axis indicates the shortest coupling interval to the preceding RR interval. Therefore, this curve defines the functional refractory period of atrioventricular conduction. The scattergram of the RR interval pairs immediately preceding the aberrant conduction (coupling points of aberrant conduction) specifically distributed along the envelope. In contrast, the coupling points of ventricular ectopies showed different distributions that had no relation to the envelope. That is, it included three typical patterns, ie, linear distribution below the envelope, linear distribution partially overlapped in the area of normal AF conduction, and chaotic distribution in the AF area. None of the scattergrams of ventricular ectopies showed curvilinear distribution along the envelope as aberrant conduction did. The specific distribution of the aberrant conduction on the RR interval scattergram suggested that aberrant conduction in AF could result from the difference of refractory periods between the AV node and bundle branch block.

Conclusions. We conclude that the RR interval scattergram makes it possible to differentiate between aberrant ventricular conduction and ventricular ectopy in atrial fibrillation, and thus, it is a useful noninvasive clinical tool. (Circulation. 1993;88[part 1]:2307-2314.)

KEY WORDS • atrioventricular node • electrocardiography

We frequently observe wide QRS complexes in atrial fibrillation. Differentiation between aberrant ventricular conduction and ventricular ectopy is of etiologic, prognostic, and therapeutic importance. The distinction, however, is more difficult in atrial fibrillation than in sinus rhythm, since the diagnostic relation of discrete atrial activity to the wide QRS complex is lost. Although ECG criteria have been reported to help differentiate aberrant ventricular conduction from ventricular ectopy during atrial fibrillation, their usefulness and reliability remain to be established.1-3 The introduction of intracardiac electrophysiological techniques has made it possible to definitely differentiate aberrant ventricular conduction from ventricular ectopy.4-5 With these techniques, surface ECG criteria have been extensively reevaluated, resulting in the advancement of newer criteria for differentiation of wide QRS complexes during atrial fibrillation.6,7 Definite diagnosis, however, still depends on the technique of intracardiac electrograms. Intracardiac electrophysiological studies may not be justifiable only to diagnose aberrant ventricular conduction or ventricular ectopy.

The aim of this investigation was to develop a noninvasive technique to diagnose aberrant ventricular conduction and ventricular ectopy in atrial fibrillation. For this purpose, we used the successive RR interval scattergram with Holter ECG.8-10 Since aberrant ventricular conduction and ventricular ectopy distribute differently on the RR interval scattergrams, we could differentiate these two wide QRS complexes in atrial fibrillation.

Methods

Principle of the RR Interval Scattergram

The principle of the successive RR interval scattergram has been described elsewhere.8-10 Briefly, in reference to Fig 1, the first two RR intervals (d1, d2) in

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From the Research Institute of Angiocardiology and Cardiovascular Clinic, Kyushu University Medical School, Fukuoka, Japan, and the National Cardiovascular Center Research Institute (K.S., M.S.) Osaka, Japan.
Reprint requests to Akiko Chishaki Suyama, MD, Research Institute of Angiocardiology and Cardiovascular Clinic, 3-1-1 Maidashi, Higashiku, Fukuoka 812, Japan.

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curve reasonably represents the functional refractory period of the atrioventricular node.8,9

We defined on the RR interval scattergram the coupling point of a wide QRS complex as follows, in reference to panel D of Fig 1: Coupling point (x, V1-V2 interval; y, V2-V3 interval) V1, V2, preceding successive QRS complexes followed by a wide QRS complex (V3).

We analyzed all points composed of successive pairs of RR intervals associated with normal conduction and the coupling points of wide QRS complexes. We can operate to plot scattergrams for wide QRS complexes of a single morphology or all wide QRS complexes of different morphologies. In all patients, we first plotted all wide QRS complexes of different morphologies and then separately plotted each morphology for further analysis.

Subjects

We studied 34 patients with paroxysmal atrial fibrillation (PAF) and 62 patients with chronic atrial fibrillation (CAF). In all patients, frequent wide QRS complexes were observed. We selected all the PAF patients who satisfied the condition that the wide QRS complexes during PAF had waveforms identical to aberrancy of the supraventricular ectopies, for reasons discussed later. Clinical characteristics are shown in the Table. Twelve patients with PAF and 20 patients with CAF underwent His bundle recordings and were shown to have ventricular ectopy or aberrant ventricular conduction. We divided the patients into two groups, ie, 32 patients with His recordings (group 1) and 64 patients without them (group 2). In 22 PAF patients in group 2, we diagnosed aberrant ventricular conduction if the waveforms of the wide QRS complexes were identical to the aberrant forms of supraventricular premature beats during sinus rhythm, and these waveforms were analyzed. In 42 CAF patients of group 2, we analyzed all waveforms according to the RR interval scattergram. All patients were taking digitalis, with serum levels within the normal therapeutic range. None had clinical evidence of toxicity. No ß-blockers were taken by any patients.

ECG Recording and Analysis

Holter electrograms were recorded with a portable tape recorder (model 445, Del Mar Avionics, Irvine, Calif) and were played back with an automatic cardioscanner (DCG VII, Del Mar Avionics) at a rate 120 times faster than real time. We modified the scanner so that the RR interval and waveform signals could be downloaded to a slave microcomputer (IBM 5550, IBM Corp, Purchase, NY) at this accelerated rate.

Clinical Characteristics of Patients With Paroxysmal Atrial Fibrillation and Chronic Atrial Fibrillation

<table>
<thead>
<tr>
<th>Total No. of Patients</th>
<th>No. of Female Patients</th>
<th>Age, y</th>
<th>Atrial Fibrillation Alone</th>
<th>RHD</th>
<th>IHD</th>
<th>CM</th>
<th>SSS</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAF</td>
<td>34</td>
<td>22</td>
<td>56±14</td>
<td>8</td>
<td>8</td>
<td>9</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>CAF</td>
<td>62</td>
<td>33</td>
<td>58±10</td>
<td>0</td>
<td>38</td>
<td>6</td>
<td>9</td>
<td>0</td>
</tr>
</tbody>
</table>

RHD, rheumatic heart disease; IHD, ischemic heart disease; CM, cardiomyopathy; SSS, sick sinus syndrome; PAF, paroxysmal atrial fibrillation; CAF, chronic atrial fibrillation.
We studied these patients first to establish the assumption that different patterns on the RR interval scattergrams can differentiate aberrant conduction from premature ventricular ectopies.

**PAF patients.** All the wide QRS complexes in the PAF patients accompanied His waves before the ventricular waves and thereby confirmed aberrant conduction. The distributions of the coupling points of the aberrant conduction were concordant with the envelope of widely scattered points of normal atrial fibrillation conduction without exception. Fig 2A shows the RR interval scattergram of normal conduction (left) and the coupling points of aberrant conduction shown in the inset electrogram (right) during atrial fibrillation. The identical waveform of aberrant conduction of supraventricular ectopies appeared in sinus rhythm. We analyzed an electrogram obtained for 5 hours. These coupling points distributed along the envelope of widely scattered points of normal atrial fibrillation conduction. Fig 2B shows another example of PAF that had two types of waveforms of aberrant ventricular conduction, ie, right bundle branch block (RBBB) and left bundle branch block (LBBB) types. Both waveforms were also indistinguishable from those of the aberrant ventricular conduction of supraventricular ectopies observed during sinus rhythm in this patient. The coupling points of both types also distributed along the envelope of the RR interval scattergram of atrial fibrillation. The coupling points of
LBBB type (middle panel), however, distributed more closely to the envelope than did those of the RBBB type (right panel). The RBBB type distributed slightly but consistently upward relative to the envelope (average shift, 0.10 second). The x values of the coupling points of the LBBB type varied between 0.30 and 2.00 seconds, whereas those of the RBBB type varied between 0.52 and 1.48 seconds. Three other patients had different kinds of waveforms of aberrant ventricular conduction.

We selected the PAF patients who satisfied the condition that the wide QRS complexes during PAF had waveforms identical to aberrant forms of supraventricular ectopies. These wide QRS complexes in group 1 accompanied the His waves before the ventricular waves during PAF and thereby confirmed aberrant conduction. This result also validates the assumption that the wide QRS complexes during PAF having waveforms identical to the aberrant forms of supraventricular ectopies would be the aberrant conduction.

CAF patients. In 20 patients with CAF as shown by electrophysiological study, 6 patients had only ventricular ectopies, 7 patients had only aberrant ventricular conduction, and the remaining 7 patients had both. In 7 patients who had only aberrant conduction, the distribution of the coupling points showed specific curvilinearity along the envelope.

Fig 3 shows three cases, including three different typical distributions of the coupling points of ventricular ectopies, ie, linear distribution below the envelope, linear distribution partially overlapped in the area of normal atrial fibrillation conduction, and chaotic distribution in the atrial fibrillation area. In panel A, the coupling points were clearly separated below the envelope, and the curvilinearity observed in the aberrant ventricular conduction no longer existed, ie, the coupling intervals of the ventricular ectopies were virtually constant irrespective of the x values and were smaller than those of the envelope. Since the y values of the envelope represent the functional refractory periods of atrioventricular conduction (see discussion for details), the wide QRS complexes whose coupling intervals are shorter than the functional refractory period of atrioventricular conduction thus indicate ventricular ectopies. Therefore, this distinct distribution would allow diagnosis of the wide QRS complexes as ventricular ectopies without proof by electrophysiological study. Panel B shows the scattergram with two kinds of ventricular ectopies, whose coupling intervals were different from each other (mean value, 0.48 and 0.72 seconds) and had more variability compared with those of the ventricular ectopies shown in panel A. The distributions of the coupling points of both ventricular ectopies were not clearly separated below the envelope, but the curvilinearity along the envelope observed in aberrant ventricular conduction no longer existed. This example had two kinds of coupling points respective to the different morphologies of ventricular ectopies. Single linear distribution partially overlapping in the area of normal atrial fibrillation conduction was common. Panel C shows the other case having different kinds of ventricular ectopies. One of the coupling points was rela-
tively constant, and the other was markedly variable. The constant point was separated below the envelope, but the variable one was chaotically overlapped in the area of normal atrial fibrillation conduction. Only chaotic distribution was observed in the atrial fibrillation area. None of the coupling points of the ventricular ectopies showed curvilinearity along the envelope.

Seven patients had both aberrant conduction and ventricular ectopies. Fig 4 shows the RR interval scattergrams of the surface ECG (V2 lead) and the simultaneous recording of intracardiac ECG (right ventricle and His bundle). The wide QRS complexes judged as aberrant ventricular conduction from the intracardiac ECG (Fig 4A) showed curvilinear distribution along the envelope (Fig 4D). Conversely, the wide QRS complexes that had no His waves before the ventricular waves (Fig 4B) distributed linearly without any relation to the envelope (Fig 4E).

In a total of 13 patients with ventricular ectopies (6 with only ventricular ectopies and 7 with ventricular ectopies and aberrant conduction), single-linear and clearly separated distributions were observed in 5 patients, overlapped linear distributions were observed in 3 patients, chaotic distributions were seen in 2 patients, and a combination of linear and other distributions were seen in the remaining 3 patients.

**Group 2**

We studied wide QRS complexes in 22 PAF and 42 CAF patients on the basis of the principle obtained from the results in the definite group (group 1).

**PAF patients.** We analyzed the wide QRS complexes whose waveforms were identical with the aberrant forms of supraventricular ectopies during sinus rhythm. The results obtained from group 1 suggested that these wide QRS complexes during PAF would be aberrant conduction. The coupling points of the wide QRS complexes had curvilinear distributions along the envelope of normal atrial fibrillation conduction without exception. These distributions were characteristic of the aberrant conduction observed in group 1.

**CAF patients.** The coupling points of the wide QRS complexes in 16 of 42 CAF patients distributed along the envelope of normal atrial fibrillation conduction. Namely, the wide QRS complexes were estimated to be aberrant conduction.

The distribution of 11 cases had no relation to the envelope of atrial fibrillation conduction. This suggests that these wide QRS complexes were of ventricular origin.

The remaining 15 patients showed a combination of curvilinear distributions along the envelope, ie, charac-

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**Fig 4.** RR interval scattergram and simultaneous recording of intracardiac ECG. A. Wide QRS complex (*) had His wave before V wave (ie, aberrant ventricular conduction). The distribution of the coupling points of these wide QRS complexes are shown in D. B. Wide QRS complex (*) that had no His wave before V wave was diagnosed as ventricular extrasystole. Distribution of the coupling points is shown in E. C shows all points. V2, surface electrogram of V2 lead; RV, His, intracardiac ECGs of right ventricle and His bundle; LVP, left ventricular pressure.
teristic for aberrant conductions, and distribution without any relation to the envelope, ie, ventricular ectopies. The distributions without any relation to the envelope included 10 separated, 8 overlapped, 5 chaotic, and 3 mixed patterns.

Discussion

Differentiation of Aberrant Conduction From Ventricular Ectopy in Atrial Fibrillation

We have demonstrated in PAF patients that the coupling points of aberrant conduction shown by intracardiac His recording distributed along the envelope of the RR interval scattergram. Similarly, the coupling points of the wide QRS complexes, which had waveforms identical to those seen in aberrant conduction after proven supraventricular premature contraction, also distributed along the envelope of the RR interval scattergrams. In the study patients with PAF, there were no exceptions. Furthermore, the distribution of the proven ventricular ectopies never formed a curvilinearity along the envelope of atrial fibrillation. Thus, this particular distribution appeared to be characteristic of aberrant ventricular conduction.

The RR interval scattergram of wide QRS complexes, which had no preceding His activity, either scattered widely in the area of normal atrial fibrillation conduction, distributed clearly below the envelope, or formed a linear band overlapping in the atrial fibrillation area. Therefore, one could differentiate aberrant conduction from ventricular ectopy in atrial fibrillation using the RR interval scattergram obtained from Holter ECG.

Envelope in the RR Interval Scattergram: Significance as the Refractory Period of the AV Node

In this study, we considered that the envelope of the RR interval scattergrams of atrial fibrillation represents the functional refractory period of atroventricular conduction; this was based on our previous study. In the previous study, we simulated atrial fibrillation by pacing the right atrium in isolated cross-perfused canine hearts and obtained RR interval scattergrams. The functional refractory period has been defined as the shortest attainable interval between two impulses traversing that tissue and measured at a point distal to the tissue. The value of the envelope corresponding to each value, ie, the preceding RR interval, was the shortest RR interval and then stood for the functional refractory period of the atroventricular node at the preceding RR interval. The estimated functional refractory periods determined by the RR interval scattergram correlated well with those measured by the conventional electrophysiological method. Once we know the refractory period of the atrioventricular node, any beats with wide QRS complexes distributed below the envelope indicate true ventricular origin. Therefore, the wide QRS complexes distributed clearly below the envelope would be diagnosed as ventricular ectopies without proof by electrophysiological study.

Influences of Autonomic Tone on the Scattergram

Changing autonomic tone is well known to affect atroventricular nodal conduction. Our previous study using isolated, cross-circulated canine hearts indicated that the envelope of the scattergram shifted upward by vagal nerve stimulation. It is conceivable that the envelope provides a tool to identify the effect of changes in autonomic tone on atroventricular conduction. Obviously, further investigation is needed to establish the usefulness of the RR interval scattergram in a clinical setting.

Mechanisms of Aberrant Ventricular Conduction and Relation to the RR Interval Scattergram

Physiological mechanisms of aberrancy. The mechanisms generally contributory to aberration with changing cycle length include (1) premature excitation before completion of repolarization; (2) unequal refractoriness of conducting tissue resulting in local delay of conduction; (3) prolongation of the action potential duration because of the prolonged preceding cycle length; (4) failure of the refractory period to shorten in response to acceleration of the heart rate; (5) a reduced takeoff potential secondary to diastolic depolarization; and (6) specifically in atrial fibrillation, concealed conduction of atrial fibrillatory impulses. If the stimulus falls during the effective or the functional refractory period of the conduction system, conduction will be physiologically delayed. Duration of the refractory period depends to a great extent on the basic heart rate and on the duration of the immediately preceding RR intervals. Normally, the refractory period shortens with acceleration of the basic heart rate and lengthens with slowing of the basic heart rate. This phenomenon of the atrioventricular node was reflected in the curvilinearity of the envelope. Consequently, with a relatively accelerated heart rate, sudden prolongation of the immediately preceding RR interval may result in aberration. This relation of aberrancy to changes in the preceding RR interval is known as the Ashman phenomenon. As we demonstrated, all aberrant conductions distributed along the envelope over the x axis. This is to say that whenever the aberrant conductions took place, the preceding interval was always longer than the coupling interval. But there was no absolute length of the coupling interval above which aberrant conduction always occurred. This is the generalized expression of the Ashman phenomenon on the RR interval scattergram. The common determinant through these mechanisms is the timing between the premature impulse conducting the atrioventricular node and the refractoriness of the conduction system.

Acceleration-dependent aberrancy. At certain critical heart rates, impaired intraventricular conduction results in aberrancy. This aberrancy differs in a number of respects from the physiological aberrancy observed in a normal heart. Differences include (1) appearance of aberrancy at relatively slow heart rates, (2) predominance of LBBB morphology, (3) independence from the immediately preceding cycle length, (4) occasional appearance without or with only a slight change in cycle length, and (5) association with heart disease. This aberrancy may persist at an RR interval considerably longer than the interval that initiated the aberrancy. The acceleration-dependent aberrancy, however, may be rare in atrial fibrillation. This type of aberrancy was not observed, at least in our study patients whose aberrancy was shown by His bundle recording.

Aberration may result when any of the above mechanisms alter conduction in the bundle branches and...
atrial impulses pass the atroventricular node. Therefore, the critical differences between the functional refractory period of the atroventricular node and that of the bundle branches contribute to aberration. Normally, at slow heart rates, the right bundle has the longest refractory period, whereas the left bundle and the atroventricular node are somewhat shorter and the His bundle the shortest. With increasing heart rates, the refractory period of the right bundle is shortened.\textsuperscript{13} The relation that explains our results is schematized in the RR interval scattergram in Fig 5. If the refractory period of either bundle is longer than that of the AV node, atrial pulses conduct through the atroventricular node and hit the timing of the refractory period of either blocked bundle. Then, aberration caused by the blocked bundle will take place. Therefore, the distribution of aberrant conduction in the RR interval scattergram overlaps with the area between the refractory curve of the atroventricular node and that of either blocked bundle branch. Thus, the distribution of the coupling points of aberrant conduction was curvilinear along the envelope. The distribution of aberrant conduction along the envelope of the RR interval scattergram of atrial fibrillation explains the physiological mechanisms of aberrancy.

\textbf{Limitations}

In this investigation, we could not perform electrophysiological examination for all patients; therefore, we described a gold standard for differentiating aberrant conduction from ventricular ectopies in group 1 and then evaluated our method in group 2. To supplement the limited numbers of group 1, we selected only patients with PAF, which frequently accompanied the wide QRS complexes having morphologies similar to the supraventricular premature complexes with aberrant conduction. These identical wide QRS complexes were likely to be aberrant conduction, for the following reasons. The functional refractory periods of the bundle branches in these patients would be cycle dependent longer than those of the atroventricular node, since supraventricular premature complexes with aberrant conduction were observed. Once the patients had attacks of PAF with rapid ventricular rate, the probability of hitting the period between the refractory periods of bundle branches and that of the atroventricular node would be increased, and this could result in frequent aberrant conduction. In this investigation, all patients who underwent intracardiac His recording validated this assumption. Thus, we thought that the criterion we used to judge the wide QRS complexes in the PAF of group 2 as aberrant conduction would be reasonable.

In patients with ventricular ectopies, further analyses would result in more different patterns of scattergrams. We also need to accumulate more cases to definitely diagnose aberrant conduction by the distribution along the envelope.

Acceleration-dependent aberrancy is a phenomenon that has no relation to the functional refractory periods of the atroventricular node and bundle branches. Therefore, if this aberrancy should take place, it could not be diagnosed by the RR interval scattergram. At least in our study patients confirmed by His recordings, this type of aberrancy was not observed.

This method analyzes many RR intervals and then diagnoses the wide QRS complexes by the shape of scattergram; therefore, it requires frequent occurrences of wide QRS complexes to compose the figure. Single or very rare occurrences of wide QRS complexes cannot be diagnosed by this method. But in clinical situations, these cases would not need a strict diagnosis.

In this investigation, we did not derive quantitative criteria or indices for diagnosing the wide QRS complexes during atrial fibrillation by the RR interval scattergram. This would require future study.

We did not perform a comparative study regarding the sensitivity, specificity, and predictive value of this technique versus any other presently applied methods. Since the aim of this study was primarily to introduce a new technique for diagnosing wide QRS complexes in atrial fibrillation, this comparative study would also be a future goal.

\textbf{Conclusions}

In chronic atrial fibrillation, wide QRS complexes could be either aberrancy or ventricular ectopy. Although definitive diagnosis of these complexes requires intracardiac His bundle recording, this is not always feasible. We have developed an RR interval scattergram using the Holter ECG. The specific distribution of aberrant conduction in the RR interval scattergram allowed us to differentiate aberrant conduction from ventricular ectopy in atrial fibrillation with noninvasive means. This method would be useful in diagnosing the origins of wide QRS complexes in atrial fibrillation.

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\textbf{References}


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