The "Compensatory Pause" of Atrial Fibrillation

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SUMMARY A long pause after an abnormal beat during atrial fibrillation has been called a "compensatory pause" and has been used to identify premature ventricular complexes (PVCs) and to differentiate them from supraventricular beats with aberration. The diagnostic value of the compensatory pause is controversial and has not been tested systematically with programmed stimulation and intracardiac recordings. In this study we used these methods to determine if PVCs induced during atrial fibrillation were followed by compensatory pauses.

Five patients were studied who had ECGs with a normal PR interval and a normal QRS duration and morphology during sinus rhythm. Atrial fibrillation was induced by rapid atrial pacing. PVCs were induced by coupling a ventricular extrastimulus to every eighth or tenth QRS complex during atrial fibrillation. The coupling interval of the PVC was changed in 10- or 20-msec increments until the entire cardiac cycle was scanned.

The mean duration of recorded atrial fibrillation was 15.7 minutes. Fifty-seven to 163 PVCs were induced in each patient. The mean cycle after the induced PVC was calculated and compared with the mean control cycle. The mean cycle after the PVC was 107-136 msec longer than the mean control cycle (p < 0.001 in every patient). This study confirms the presence of a compensatory pause after stimulated PVCs in atrial fibrillation.

AN ABNORMAL QRS COMPLEX ON AN ECG recorded during atrial fibrillation may be a premature ventricular complex (PVC) or a supraventricular beat with aberrant intraventricular conduction. Clinical cardiologists are faced with the difficult problem of distinguishing between these abnormal beats in order to prescribe the correct drugs to control them. Patients with atrial fibrillation are usually treated with digitalis, yet PVCs may result from digitalis toxicity. The decision to alter a patient's drug therapy may depend on separating PVCs from supraventricular beats with aberration.

Langendorf suggested that in atrial fibrillation the cardiac cycle after a PVC was longer than the cardiac cycle after a supraventricular beat with aberration. He called this longer cycle the "compensatory pause" of atrial fibrillation. His explanation for it was that the PVC penetrated the atrioventricular (AV) node in the retrograde direction and increased AV nodal conduction time of the next supraventricular impulse. He demonstrated a compensatory pause after PVCs induced by a fixed-rate ventricular pacemaker in a patient with atrial fibrillation.

Although its diagnostic value has been questioned, the compensatory pause is cited frequently in textbooks as indicating a ventricular origin for an abnormal beat. In this study we used intracardiac recordings and programmed stimulation to demonstrate the compensatory pause and to study its application to clinical electrocardiography.

Methods

The five patients reported here were referred to the Clinical Electrophysiology Laboratory of Duke University between July 1978 and June 1979 for the treatment of tachycardia. All patients had ECGs that showed a normal PR interval and normal QRS duration and morphology during sinus rhythm. The research nature of the study was explained to each patient, and written consent was obtained before study. Antiarrhythmic medicines were discontinued 5 half-lives before study. Patients were studied after fasting and without sedation.

Four multipolar electrode catheters were used to record simultaneously from the right atrium, right ventricular apex, bundle of His and coronary sinus. Five ECG leads and the intracardiac leads were recorded at 100 mm/sec using a Mingograph (model 1605) ink-jet recorder. The type of conduction in the retrograde direction was determined by endocardial mapping during ventricular pacing. Atrial fibrillation was induced by rapid atrial stimulation.

A ventricular extrastimulus (S₂) was coupled to every eighth or tenth QRS complex during atrial fibrillation. The coupling interval of the extrastimulus was held constant until five PVCs (V₂) had been induced, so that each coupling interval followed a range of variable cardiac cycles. The coupling interval was then changed in 10- or 20-msec increments until the entire cardiac cycle was scanned. Fifty-seven to 163 PVCs were induced. In general, fewer PVCs were induced in patients with faster ventricular rates.

The two beats immediately preceding the induced ventricular beat (V₂) were designated V₁ and V₀ (fig. 1A). The beat after V₂ was designated V₃. These beats were used to define the following cycles and intervals:
V_0V_1 = control cycle; V_1V_2 = coupling interval; V_2V_3 = compensatory cycle.

These intervals were measured from the onset of one QRS complex to the onset of the succeeding QRS complex using a sonic X-Y digitizer and an interactive computer program. This system measures intervals of the cardiac cycle with interobserver variability of less than 2 msec. If V_0, V_1, or V_2 was abnormal or had a prolonged HV interval, the series was discarded from the analysis. Measured intervals were kept in disk files for later analysis. The sum of the coupling interval and the compensatory cycle for each PVC (V_1V_2) was calculated and the relative prematurity of each PVC was determined using the ratio V_1V_2/V_0V_1.

Spontaneous Abnormal Beats During Atrial Fibrillation

In all patients the analog records were searched for spontaneous abnormal QRS complexes, which were then placed into one of the following categories:

1. Supraventricular beat with aberration consistent with the Ashman phenomenon if the QRS morphology was right bundle branch block, if the cycle preceding the abnormal beat was short and followed a relatively long cycle, and if a His bundle electrogram preceded the QRS complex.

2. Supraventricular beat with aberration not caused by the Ashman phenomenon if a His bundle electrogram preceded the abnormal beat but the other two conditions specified in (1) were not satisfied.

3. Catheter-induced premature ventricular beat if the QRS of the abnormal beat was similar to V_2 but was not stimulated.

4. Repetitive ventricular response if the QRS of the abnormal beat was similar to and immediately followed V_2.

5. Spontaneous PVC if the abnormal QRS was not preceded by a His bundle electrogram and did not resemble V_0.

6. Unknown if the abnormal QRS did not resemble the induced premature beat and a stable His bundle electrogram was not recorded at the time of the abnormal beat.

Statistical Methods

Several comparisons were used to determine the best application of the compensatory pause. For each patient histograms of the intervals V_0V_1, V_1V_2, V_2V_3, and V_1V_3 were drawn. Mean V_0V_1 was compared to mean V_2V_3 using a t test. Maximum V_0V_1 was compared to maximum V_2V_3. The effect of the coupling interval (V_1V_2) on the compensatory cycle (V_2V_3) was assessed in each patient by calculating the correlation coefficient for V_1V_2 and V_2V_3. The correlation coefficient for V_1V_2/V_0V_1 and V_2V_3 was also calculated.

Results

A clinical description of each patient is given in table 1. The mean duration of atrial fibrillation was 15.7 minutes.
**TABLE 1. Clinical Data**

<table>
<thead>
<tr>
<th>Pt (years)</th>
<th>Sex</th>
<th>Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>33</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>3</td>
<td>33</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chaotic supraventricular tachycardia</td>
</tr>
<tr>
<td>4</td>
<td>38</td>
<td>F</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PSVT due to reentry in AV node</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>F</td>
</tr>
<tr>
<td></td>
<td></td>
<td>AV reentrant tachycardia*</td>
</tr>
</tbody>
</table>

*Accessory pathway conduction only in the retrograde direction.

Abbreviations: AV = atroioventricular; PSVT = paroxysmal supraventricular tachycardia.

**Effect of Induced PVCs on the Succeeding Cardiac Cycles**

The type of conduction in the retrograde direction that was determined by pacing the ventricles while the atria were not fibrillating did not affect results. When

**TABLE 2. Cardiac Cycles During Atrial Fibrillation**

<table>
<thead>
<tr>
<th>Pt</th>
<th>n</th>
<th>V_2V_1 Max</th>
<th>V_2V_1 Min</th>
<th>V_2V_1 Mean ± SD</th>
<th>V_1V_2 Max</th>
<th>V_1V_2 Min</th>
<th>V_1V_2 Mean ± SD</th>
<th>V_2V_3 Max</th>
<th>V_2V_3 Min</th>
<th>V_2V_3 Mean ± SD</th>
<th>V_1V_3 Max</th>
<th>V_1V_3 Min</th>
<th>V_1V_3 Mean ± SD</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>57</td>
<td>708</td>
<td>312</td>
<td>457 ± 102</td>
<td>465</td>
<td>319</td>
<td>382 ± 34</td>
<td>925</td>
<td>319</td>
<td>564 ± 130</td>
<td>1274</td>
<td>702</td>
<td>946 ± 142</td>
</tr>
<tr>
<td>2</td>
<td>152</td>
<td>1159</td>
<td>348</td>
<td>651 ± 164</td>
<td>765</td>
<td>262</td>
<td>476 ± 155</td>
<td>1110</td>
<td>419</td>
<td>768 ± 151</td>
<td>1786</td>
<td>705</td>
<td>1244 ± 231</td>
</tr>
<tr>
<td>3</td>
<td>163</td>
<td>1069</td>
<td>373</td>
<td>610 ± 123</td>
<td>671</td>
<td>269</td>
<td>417 ± 116</td>
<td>1125</td>
<td>409</td>
<td>742 ± 136</td>
<td>1676</td>
<td>791</td>
<td>1160 ± 163</td>
</tr>
<tr>
<td>4</td>
<td>99</td>
<td>938</td>
<td>308</td>
<td>475 ± 104</td>
<td>553</td>
<td>235</td>
<td>344 ± 79</td>
<td>923</td>
<td>367</td>
<td>592 ± 123</td>
<td>1254</td>
<td>686</td>
<td>938 ± 123</td>
</tr>
<tr>
<td>5</td>
<td>119</td>
<td>734</td>
<td>310</td>
<td>463 ± 83</td>
<td>421</td>
<td>230</td>
<td>326 ± 57</td>
<td>902</td>
<td>335</td>
<td>599 ± 117</td>
<td>1262</td>
<td>681</td>
<td>928 ± 111</td>
</tr>
</tbody>
</table>

Abbreviations: Max = maximum; Min = minimum.

**Figure 2. Histogram of cardiac cycles, patient 3. (A) The control cycle, V_0V_1. (B) The coupling interval, V_1V_2. (C) The compensatory cycle, V_2V_3. (D) The sum of V_1V_2 and V_2V_3. Note that mean V_2V_3 is slightly larger than mean V_0V_1.**
tervals in patient 2 and in only one of 99 coupling intervals in patient 4.

The compensatory cycle (V₂V₃) did not correlate with the coupling interval (V₁V₂) in a consistent way. There was no significant correlation between these variables in any patient. Similarly, the correlation coefficient of V₂V₅/V₅V₁ and V₂V₃ was calculated for every patient, and no significant correlation was found. Scatter plots of data from patient 3 are shown in figure 3.

Spontaneous Abnormal Beats During Atrial Fibrillation

All five patients had some spontaneous abnormal beats during atrial fibrillation. Supraventricular beats with aberration consistent with the Ashman phenomenon were recorded in all patients, but no other type of supraventricular beat with aberration was recorded in any patient. That is, there were no supraventricular beats with left bundle branch block aberration, and aberrant conduction was recorded only when a short cycle followed a relatively long cycle.

PVCs were recorded in all five patients. Patient 4 had eight spontaneous PVCs in 14.8 minutes of atrial fibrillation and patient 5 had one spontaneous PVC in 11.7 minutes. Figure 4 shows scatter plots of the intervals associated with spontaneous and induced PVCs in patient 4 (compare with figure 3). The effect of spontaneous PVCs on the succeeding cardiac cycle was not different from the effect of the induced PVCs with the same coupling interval.

Two patients each had catheter-induced PVCs and repetitive ventricular responses. One patient had a single abnormal beat the origin of which was unknown because of His bundle electrogram was not recorded at the time.

Discussion

The distribution of the control cardiac cycles in our patients was similar to that observed in other patients

\[ \text{Figure 3. Patient 3. (A) There is no relation between} \]
\[ V₁V₂ \text{ and } V₂V₅ \ (r = -0.17). \]
\[ (B) There is no relation between} \]
\[ V₁V₂/V₅V₁ \text{ and } V₂V₃ \ (r = -0.20). \]
\[ V₁V₂/V₅V₁ \text{ is a measure} \]
\[ \text{of the prematurity of the induced premature ventricular} \]
\[ \text{complex.} \]

\[ \text{Figure 4. Patient 3. Induced premature ventricular} \]
\[ \text{complexes (PVCs) are shown as circles (compare with figure 3),} \]
\[ \text{and spontaneous PVCs are shown as black squares. The} \]
\[ \text{effect of induced and spontaneous PVCs is the same.} \]
in atrial fibrillation. The mean cycle after the induced PVC \( (V_2 V_3) \) was 107–136 msec longer than the mean control cycle in all patients, confirming Langendorf’s description of this cycle as “compensatory.” The difference between means was, therefore, the most reliable characteristic that distinguished \( V_2 V_3 \) from \( V_0 V_1 \).

Maximum \( V_2 V_3 \) exceeded maximum \( V_0 V_1 \) in three of five patients. Patients 2 and 4 had values of \( V_0 V_1 \) that exceeded maximum \( V_2 V_3 \), but were uncommon exceptions. These occasional long cycles are recorded in many patients in atrial fibrillation. Langendorf et al. attributed them to repetitive concealed conduction.

Individual compensatory cycles appeared to have random distribution. Prematurity of \( V_2 \) expressed as either \( V_2 V_3 \) or the ratio \( V_2 V_3 / V_0 V_1 \) did not affect the length of the compensatory cycle. (This latter ratio expresses the prematurity of \( V_2 \) with respect to the preceding control cycle.) Measuring single, random, compensatory cycles is not likely to be useful in clinical electrocardiography.

The mean \( V_2 V_3 \) (sum of the coupling interval plus the compensatory cycle) was almost equal to twice the mean control cycle in every patient. This means that the induced PVC blocked (on the average) one supraventricular beat that was destined to reach the ventricle (fig. 1B). Because none of the induced PVCs were followed by a retrograde His bundle electrogram, we could not determine the site of block or assess directly the relative contribution of retrograde conduction time (between the stimulating site and the AV node) and slowed conduction in the antegrade direction (caused by concealed retrograde penetration) to the prolongation of mean \( V_2 V_3 \).

The results of this study might have been different if we had used stimulation sites in the right ventricular outflow tract or left ventricle. The exact effects of changing ventricular stimulation site on conduction in the retrograde direction have not been completely studied. Our results may also have been affected by selecting patients with normal AV conduction and normal myocardium rather than patients with diseased hearts.

We were surprised to find that all recorded supraventricular beats with aberration were due to the Ashman phenomenon. We had expected to find both right bundle branch block and left bundle branch block aberration, but only right bundle branch block was recorded, and that was recorded only when a short cycle followed a relatively long cycle. However, the five patients we studied were relatively young and free of associated cardiac disease. None of them had acquired disease of the specialized conduction tissue. Moreover, none of them were studied specifically because of the occurrence of spontaneous abnormal beats during atrial fibrillation. Therefore, these results might be different.

Our data confirm the observation of Langendorf that stimulated PVCs in atrial fibrillation are followed by a compensatory pause. This phenomenon is most reliably demonstrated by comparing the mean of cardiac cycles that follow the abnormal beat with the mean control cardiac cycles.

References

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