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:

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V₀V₁ = control cycle; V₁V₂ = coupling interval; V₂V₃ = 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 V₀, V₁, or V₂ 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 (V₁V₂) was calculated and the relative prematurity of each PVC was determined using the ratio V₁V₂/V₀V₁.

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 V₂ but not stimulated.

4. Repetitive ventricular response if the QRS of the abnormal beat was similar to and immediately followed V₂.

5. Spontaneous PVC if the abnormal QRS was not preceded by a His bundle electrogram and did not resemble V₂.

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 V₀V₁, V₁V₂, V₂V₃, and V₁V₃ were drawn. Mean V₀V₁ was compared to mean V₂V₃ using a t test. Maximum V₀V₁ was compared to maximum V₂V₃. The effect of the coupling interval (V₁V₂) on the compensatory cycle (V₂V₃) was assessed in each patient by calculating the correlation coefficient for V₁V₂ and V₂V₃. The correlation coefficient for V₁V₂/V₂V₃ and V₂V₃ 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.
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 the atria were not fibrillating, conduction in the retrograde direction used the AV node in two patients, used an accessory pathway at all pacing cycle lengths in one patient, and was absent in one patient. Conduction in the retrograde direction could not be assessed in one patient. A summary of the measured intervals is presented in table 2. Histograms of data from patient 3 are shown in figure 2 to demonstrate the type of analysis done for each of the five patients. A retrograde His bundle electrogram was not found after any of the induced PVCs.

Comparing mean V2V3 with mean V0V1 most reliably demonstrated a compensatory pause. Mean V2V3 was always greater than mean V0V1 (p < 0.001 for every patient). For example, in patient 3 mean V2V3 was 742 ± 136 and mean V0V1 was 610 ± 123. Maximum V2V3 exceeded maximum V0V1 in patients 1, 3, and 5 and was less than maximum V0V1 in patients 2 and 4. However, maximum V0V1 exceeded maximum V2V3 in only two of 152 coupling intervals.

**Table 1. Clinical Data**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Sex</th>
<th>Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>33</td>
<td>M</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>M</td>
</tr>
<tr>
<td>3</td>
<td>33</td>
<td>M</td>
</tr>
<tr>
<td>4</td>
<td>38</td>
<td>F</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>F</td>
</tr>
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</table>

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

*Accessory pathway conduction only in the retrograde direction.

Abbreviations: AV = atrioventricular; 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>Max</th>
<th>Min</th>
<th>Mean ± SD</th>
<th>Max</th>
<th>Min</th>
<th>Mean ± SD</th>
<th>Max</th>
<th>Min</th>
<th>Mean ± SD</th>
</tr>
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<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>
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<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>
</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>406</td>
<td>742 ± 136</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>
</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>500 ± 111</td>
</tr>
</tbody>
</table>

Abbreviations: Max = maximum; Min = minimum.
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

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.

The compensatory cycle (V1V2) did not correlate with the coupling interval (V1V3) in a consistent way. There was no significant correlation between these variables in any patient. Similarly, the correlation coefficient of V1V3/V1V2 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.

![Figure 3](image)

**Figure 3.** Patient 3. (A) There is no relation between V1V2 and V1V3 (r = -0.17). (B) There is no relation between V1V2/V1V3 and V2V3 (r = -0.20). V1V3/V1V2 is a measure of the prematurity of the induced premature ventricular complex. V2.

![Figure 4](image)

**Figure 4.** Patient 3. Induced premature ventricular complexes (PVCs) are shown as circles (compare with figure 3), and spontaneous PVCs are shown as black squares. The effect of induced and spontaneous PVCs is the same.
in atrial fibrillation. The mean cycle after the induced PVC \( V_2V_3 \) 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 \( V_2V_3 \) from \( V_3V_1 \).

Maximum \( V_2V_3 \) exceeded maximum \( V_3V_1 \) in three of five patients. Patients 2 and 4 had values of \( V_3V_1 \) that exceeded maximum \( V_2V_3 \), 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 \( V_2V_3 \) or the ratio \( V_2V_3/V_3V_1 \) 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 \( V_1V_2 \) (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 \( V_1V_2 \).

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.

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