Ventricular Fusion Beats during Electric Stimulation in Man

Application to Conduction Velocity and Anomalous AV Excitation

By Arthur J. Linenthal, M.D., and Paul M. Zoll, M.D.

Ventricular fusion beats are observed in a variety of circumstances whenever two impulses depolarize different parts of the ventricles simultaneously. The resulting electrocardiographic complex is a mixture, or fusion, of the two responses.

Special opportunities for quantitative studies of ventricular fusion beats were found in patients with Stokes-Adams disease after electric pacemakers were implanted in the left ventricle. The two impulses producing fusion came from either two implanted electric pacemakers or an electric pacemaker and a conducted sinoatrial beat. In both circumstances, the important information could be precisely determined regarding the locations of the two pacemakers and the time when each impulse reached the ventricles.

In a previous report of studies in 19 patients, we presented an analysis of fusion in the ventricle between conducted beats and an electric pacemaker. Ventricular fusion beats of the same type were also studied in a single patient by Nuñez-Dey et al. and by Katz and Pick, and in four patients by Gerbaux and Lenegre. These authors all recognized the important point that such studies could give measures of conduction times between different parts of the heart. They differed considerably, however, in their interpretations of the pathways involved, and their data were too limited to permit a full analysis.

Some quantitative studies of fusion between electrically stimulated and conducted beats have also been carried out in animals. Butterworth and Poindexter and Öhnell demonstrated that the mechanism of fusion accounted for the features of anomalous AV excitation (Wolff-Parkinson-White phenomenon), but they did not use their data to derive conduction times.

It is the purpose of this paper to present more extensive quantitative data on the fusion phenomenon in man, to derive measurements of conduction velocities in Purkinje and myocardial tissues, and to discuss the significance of these observations in the assessment of the mechanism of anomalous AV excitation.

Patients and Methods

Fusion beats have been studied in 40 of our 99 patients with Stokes-Adams disease in whom electric pacemaker-electrode systems were implanted. In nine patients in whom new systems had to be implanted, observations of competition were made in the operating room during the short period when both pacemakers were functioning. In 32 patients* in whom atroventricular conduction returned at least intermittently after implantation, competition was observed between the independent sinoatrial node and the electric pacemaker. The distance between the two electrode sites was measured by the surgeon in six patients.† Similarly, the distances from the left ventricular electrode site to the upper part of the septum where the conducted impulse enters the ventricle were measured in six patients during implantation of the pacemaker and in one.

*One patient is counted both in this group of 32 and in the previous group of nine, making a total of 41 studies in 40 patients.
†We wish to thank Dr. Howard A. Frank for these important measurements.
patient at autopsy. The patients were not receiving cardio-active drugs when these observations were made.

Repeated, long electrocardiograms were obtained at standard speed (25 mm. per second) in which hundreds of fusion beats were recorded in each patient. The records were made from leads in which the complexes from the two pacemakers differed widely in shape, so that fusion could be recognized readily. Even very slight degrees of fusion could be identified by careful inspection of the QRS and T waves for slight changes from the "pure" complex produced by one pacemaker alone. Care was taken to exclude complexes in which superimposed P waves might cause confusion. Limb leads were used for most of the fusion measurements as well as for determinations of the P-R and QRS intervals. When fusion complexes were initiated by an electric stimulus, the stimulus artifact marked the beginning of the QRS interval. Precordial leads were used to determine the pattern of intraventricular conduction of the sinoatrial impulse. The measurements were accurate to 0.01 second.

Determination of Ventricular Conduction Velocities

When the ventricles are stimulated at two points so that two separate processes of depolarization occur simultaneously, a fusion beat will result. If a wave of depolarization from one stimulus reaches the second point before the second stimulus, it renders the tissue refractory and prevents depolarization by the second stimulus, so that there is no fusion. The "limit of fusion," that is the longest time the second stimulus can follow the first and still produce fusion, gives a measure of the conduction time of the depolarization wave from the first to the second point.

Figure 1 shows a series of electrocardiographic complexes selected to illustrate measurements of the conduction times in both directions between two sites (arbitrarily designated A and B) of electric stimulation in the left ventricle. Complex 1 is a ventricular beat resulting entirely from a stimulus at site A (indicated by the initial large stimulus artifact); the stimulus at site B (the small artifact after the QRS) falls in the absolute refractory period and is completely ineffective. Complex 2, in which the QRS and T are unchanged from complex 1, is also a pure beat from site A; the stimulus at site B, on the downslope of the QRS 0.06 second after that at A, is just outside the limit of fusion. Complex 3 shows slight changes from 1 and 2, so that it is a fusion beat with a small component from site B; the stimulus at B, 0.05 second after that at A, is at the limit of fusion. This limit gives a measure of the conduction time from A to B. Complexes 4 to 8 show increasing contributions to the fusion beats by stimulation at B. In complexes 4 and 5 stimulation at B still follows that at A, but in complexes 6, 7, and 8 it precedes that at A. Complex 8 shows the maximum time (0.05 second) that stimulation at A can follow that at B and still produce fusion; this limit of fusion gives a measure of the conduction time from B to A. Complex 9, like complex 10, is a pure beat stimulated entirely from site B; the stimulus at A, 0.06 second after that at B, is just outside the limit of fusion.

![Figure 1](http://circ.ahajournals.org/)

Selected complexes (patient 22, lead III) showing varying degrees of fusion. Two electric pacemakers, designated A and B, produce large (†) and small (‡) stimulus artifacts.

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VENTRICULAR FUSION BEATS

From these conduction times in the two directions and from measurements of the distances between the two electrodes, the conduction velocities can be readily calculated.

Figure 2 shows another series of complexes that illustrate fusion, in this instance between conducted sinoatrial impulses and an electric pacemaker. The timing of the stimulus from the electric pacemaker is precisely indicated by the stimulus artifact; the location of stimulation is known to be at the site of implantation in the left ventricle. The time when the conducted impulse reaches the ventricle is also precisely known: it is the time after the P wave of the constant P-R interval, so that it can be measured even when there is no conducted QRS. The point of entry of the conducted impulse in the ventricle is considered to be in the upper septum or, perhaps, at the attachment of the papillary muscle; its precise localization is not critical for our quantitative analysis.

In figure 2 complexes 1 and 2 are pure conducted sinoatrial beats, the stimulus falling too late to produce fusion. Complex 3 is slightly different and represents the first fusion beat in this series. The interval from the onset of the QRS to the electric stimulus in complex 3 is the limit of fusion and measures the antegrade conduction time from the upper septum to the stimulating electrodes. At the other end of the sequence, complex 9 is the last beat showing fusion, whereas complex 10, like 11, is a pure electrically stimulated beat. In complex 9 the interval between the electric stimulus and the end of the P-R interval (indicated by the lower arrows) is the limit of fusion, which measures the retrograde conduction time from the stimulating electrodes to the septum where the conducted impulse enters the ventricle.

Results

Conduction Times

Fusion between two electric pacemakers in the left ventricle was studied in nine patients (table 1). In each patient the conduction time between the two electrode sites was the same in both directions. The conduction velocities were approximately 1 meter per second and showed little variation. In contrast, the cardiac pathology varied considerably in these patients. All the hearts were hypertrophied but the myocardium was otherwise normal in several and severely diseased in others.
Table 1

Conduction Times and Velocities between Two Electric Pacemakers in the Left Ventricle in Nine Patients

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Age, sex</th>
<th>Conduction time*</th>
<th>Distance between A and B (cm.)</th>
<th>Conduction velocity (M. per sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60</td>
<td>M</td>
<td>0.12</td>
<td>10.0</td>
<td>1.1 — 1.2</td>
</tr>
<tr>
<td>61</td>
<td>M</td>
<td>0.04</td>
<td>4.7</td>
<td>0.9 — 1.2</td>
</tr>
<tr>
<td>62</td>
<td>M</td>
<td>0.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>63</td>
<td>M</td>
<td>0.04</td>
<td>4.8</td>
<td>1.2</td>
</tr>
</tbody>
</table>

*A and B represent 2 sites of electric stimulation in the left ventricle.

In 32 patients fusion was studied between conducted sinoatrial beats and beats from an electric pacemaker in the left ventricle (table 2). Although all these patients had had complete atrioventricular block and severe Stokes-Adams attacks, atrioventricular conduction returned at least temporarily, and was even normal in 25, with a P-R interval of 0.20 second or less. Seven of these patients had normal intraventricular conduction, 17 had right and six left bundle-branch block, and two had intraventricular block. Again, as in the previous group, the cardiac pathology varied widely.

The antegrade conduction time was short in the group with normal intraventricular conduction, being only 0.01 to 0.02 second. In nine of the patients with right bundle-branch block antegrade conduction was similarly rapid, but it was prolonged at 0.03 to 0.05 second in the other eight. In all six patients with left bundle-branch block antegrade conduction was prolonged at 0.04 to 0.07 second. In the two patients with intraventricular block the antegrade conduction times were also prolonged, to 0.04 and 0.05 second. In contrast, the retrograde conduction times were long in all 32 patients, being 0.06 to 0.09 second, and were unaffected by the pattern of intraventricular conduction.

Measurements in seven patients of the distances between the electrodes and the

Table 2

Data from Thirty-two Patients Showing Fusion between Beats from Sinoatrial and Electric Pacemakers

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Age, sex</th>
<th>P-R (sec.)</th>
<th>QRS (sec.)</th>
<th>Electric QRS (sec.)</th>
<th>Antegrade (sec.)</th>
<th>Retrograde (sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>57M</td>
<td>0.21</td>
<td>0.06</td>
<td>0.13</td>
<td>0.02</td>
<td>0.09</td>
</tr>
<tr>
<td>19</td>
<td>79F</td>
<td>0.19</td>
<td>0.07</td>
<td>0.14</td>
<td>0.01</td>
<td>0.07</td>
</tr>
<tr>
<td>20</td>
<td>59F</td>
<td>0.18</td>
<td>0.09</td>
<td>0.14</td>
<td>0.01</td>
<td>0.06</td>
</tr>
<tr>
<td>60</td>
<td>68M</td>
<td>0.13</td>
<td>0.07</td>
<td>0.15</td>
<td>0.01</td>
<td>0.07</td>
</tr>
<tr>
<td>62</td>
<td>76F</td>
<td>0.18</td>
<td>0.06</td>
<td>0.13</td>
<td>0.01</td>
<td>0.09</td>
</tr>
<tr>
<td>82</td>
<td>58F</td>
<td>0.19</td>
<td>0.08</td>
<td>0.15</td>
<td>0.01</td>
<td>0.07</td>
</tr>
<tr>
<td>97</td>
<td>66M</td>
<td>0.16</td>
<td>0.06</td>
<td>0.12</td>
<td>0.01</td>
<td>0.08</td>
</tr>
</tbody>
</table>

*This patient is not included in our numbered series because he was not treated by our surgeon.

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septum (points of entry of the electric stimulus and the conducted impulse) permit calculation of the conduction velocities between the two points (table 3). Antegrade conduction velocities are calculated only for the two patients with normal intraventricular conduction. In the five patients with abnormal intraventricular conduction, the wide variations in the times and the obvious alterations in the pathways of antegrade conduction preclude the determination of meaningful antegrade velocities. Retrograde conduction velocities were calculated for all seven patients, since the retrograde conduction times were unaffected by abnormalities of intraventricular conduction: they averaged 1.0 meter per second.

In the preceding calculations of velocities in patients with normal intraventricular conduction, the distances traversed in the antegrade and retrograde directions were considered to be the same, that is, equal to the measured distances. Consequently, the ratios of retrograde to antegrade conduction velocities may be derived directly from the corresponding conduction times in such patients; measurements of the distances are not necessary, since they cancel in the ratios. In our seven patients with normal intraventricular conduction (table 2), the ratio of antegrade-to-retrograde conduction velocity ranged from 4.5:1 to 9:1.

**P-R and QRS Duration**

In the 32 patients who showed fusion between conducted sinoatrial beats and beats from an electric pacemaker, the P-R and QRS durations were analyzed. These durations and their sums (P-R plus QRS, or the P-S interval) were found to be characteristically interrelated, depending on the pattern of intraventricular conduction (fig. 3). The QRS durations of the pure conducted beats were, of course, less than 0.10 second with normal intraventricular conduction and were longer with bundle-branch block (table 2). The QRS durations of the pure stimulated beats were 0.12 second or longer, as would be expected with ectopic ventricular beats, and did not vary with the pattern of intraventricular conduction (table 2). With fusion, as the electric stimulus preceded the conducted component more and more, that is, as the P-R interval shortened, the electrically stimulated component of the fusion complex increased so that the QRS duration lengthened progressively from that of the pure conducted to that of the pure stimulated beat.

With normal intraventricular conduction, as the electric stimulus fell progressively earlier before the conducted component, the fusion QRS lengthened by the same amount that the P-R shortened, so that their sum, the P-S interval, remained constant. In bundle-branch block, however, the QRS of the conducted beat was long initially; further lengthening in the fusion beats was small and did not compensate for the P-R shortening. Accordingly, P-S shortened in both right and left bundle-branch block. These characteristic quantitative relationships of P-R, QRS, and P-S were observed in all patients in each group.

An additional special phenomenon was observed in the six patients with left bundle-branch block. When the electric stimulus and the conducted impulse reached the ventricles

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**Table 3**

<table>
<thead>
<tr>
<th>Patient number</th>
<th>Distance* (cm.)</th>
<th>Antegrade conduction velocity (M. per sec.)</th>
<th>Retrograde conduction velocity (M. per sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>62</td>
<td>6.3</td>
<td>0.01</td>
<td>0.09</td>
</tr>
<tr>
<td>97</td>
<td>7.8</td>
<td>0.01</td>
<td>0.08</td>
</tr>
<tr>
<td>40</td>
<td>7.5†</td>
<td>0.06</td>
<td>1.3</td>
</tr>
<tr>
<td>50</td>
<td>6.3</td>
<td>0.06</td>
<td>1.1</td>
</tr>
<tr>
<td>52</td>
<td>6.4</td>
<td>0.07</td>
<td>0.9</td>
</tr>
<tr>
<td>61</td>
<td>7.6</td>
<td>0.08</td>
<td>1.0</td>
</tr>
<tr>
<td>66</td>
<td>7.0</td>
<td>0.07</td>
<td>1.0</td>
</tr>
</tbody>
</table>

*From the left ventricular electrode site to the septum.

†Antegrade conduction velocities are calculated only for the two patients who showed normal intraventricular conduction.

†This distance was measured at autopsy; the other distances were measured by the surgeon at operation.

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at the same time, the QRS of the resulting fusion beat shortened to normal (figs. 3 and 4). Similar correction of the pattern of right bundle-branch block was observed in a patient in whom the right ventricle was stimulated by a catheter electrode.

Discussion

As a by-product of the implantation of electric pacemakers for the treatment of Stokes-Adams disease, we have been able to make quantitative studies of ventricular fusion. Although precise studies of the fusion phenomenon have been feasible experimentally, they were not possible in man until, with electric stimulation, the exact source and timing of the two fusing processes could be determined.

Conduction Times and Pathways

Our measurements of fusions of conducted sinoatrial beats and beats from an electric pacemaker in the left ventricle provide widely different conduction rates in the antegrade and retrograde directions between the septum and the peripheral stimulating electrodes. The pathway for antegrade conduction would obviously be expected to be by way of the specialized Purkinje fibers, at least to their termination at or near the electrodes. The pathway for retrograde conduction, on the other hand, is not so immediately apparent. The similar problem of the pathway for conduction of ventricular ectopic beats has been the subject of considerable study and discussion.

In our patients with a normal pattern of intraventricular conduction, the retrograde conduction velocity was 1 meter per second, whereas antegrade conduction was 4.5 to 9 times faster (table 3). This rate of retrograde conduction is similar to the 1.3 meters per second obtained by Trautwein et al. between intracellular microelectrodes in excised human ventricular muscle strips. Our retrograde conduction times were similar to the transit times of ventricular ectopic beats to the sep-
Ventricular Fusion Beats

Figure 4

correction of left bundle-branch block by fusion. Electrocardiogram (patient 80, lead I) showing conducted beat with left bundle-branch block, beat from electric pacemaker, fusion beat with QRS of normal duration, conducted beat, and ineffective stimulus artifact.

tum that have been estimated in the past. Furthermore, the ratio of the rates of antegrade and retrograde conduction is similar to that found experimentally between rates in Purkinje and ordinary myocardial tissue. These observations strongly suggest that antegrade conduction is, indeed, by way of the Purkinje system whereas retrograde conduction is through ordinary myocardium.

The observations of conduction in patients with right and left bundle-branch block, in contrast with those in patients with normal intraventricular conduction, add strong confirmation to our interpretation of the pathways involved in antegrade and retrograde conduction. The striking delay of antegrade conduction in the patients with left bundle-branch block is readily explained by the hypothesis of antegrade conduction via Purkinje fibers: the impulse must take a circuitous pathway around the blocked left bundle to reach the left ventricular electrode. On the other hand, right bundle-branch block does not interfere with passage of the impulse down the left bundle to the left ventricular electrode; consequently, the antegrade conduction time in this group is usually normal. The delay found in eight patients with right bundle-branch block, according to this analysis, must also arise in the left bundle. Increase in the partial left bundle-branch block to a complete stage, together with the right bundle-branch block, would constitute bilateral bundle-branch block, which might well be the mechanism of the complete AV block that these patients showed much of the time.

In contrast to antegrade conduction, the retrograde conduction time does not vary from normal in patients with right or left bundle-branch block. According to our interpretation, retrograde conduction should not be delayed by block in the specialized conduction bundles if it travels by way of ordinary myocardium.

The limits of fusion between two electric pacemakers in the ventricles were determined precisely just as was done with fusions between conducted sinoatrial beats and a single ventricular pacemaker. The pathway of conduction between the two electric pacemakers placed peripherally in the ventricular wall would be expected to be by way of ordinary myocardium. The rates of conduction between the two electric pacemakers were approximately 1 meter per second in close agreement with our determinations of the retrograde conduction rate. This close correspondence adds support to our interpretation of the pathways of antegrade and retrograde conduction.

Correction of Bundle-Branch Block by Fusion

Fusion beats in the presence of bundle-branch block sometimes showed an interesting feature that has been well recognized and discussed. In left bundle-branch block, when the electric stimulus from the implanted pacemaker activated the left ventricle at the same time that the conducted impulse reached the right ventricle, the fusion beat showed a QRS that was normal in duration. In right bundle-branch block, a normal QRS resulted when a stimulus delivered to the right ventricle from a catheter electrode fused at the proper time with the conducted impulse.

Anomalous AV Excitation

Our analysis of fusion leads to a consideration of the phenomenon of anomalous AV excitation, which has been of so much theoretical interest since its first description by Wolff, Parkinson, and White. It is generally agreed that the WPW complex, which
is classically characterized by a short P-R, a prolonged and abnormally shaped QRS, and a constant P-S interval, is a fusion in the ventricle of a normally conducted beat and an associated one, which reaches the ventricle earlier (fig. 5).\(^{20, 21}\) The prematurity of the associated beat shortens the P-R interval; the fusion of the two beats prolongs and distorts the QRS; the normally conducted beat, being later than the associated one, terminates ventricular activation as usual so that the timing of the S wave and the P-S interval are unchanged. Many of the atypical variations of anomalous AV excitation, such as beats with normal P-R,\(^{22}\) normal QRS duration,\(^{23}\) or short P-S,\(^{24}\) are readily explained on the basis of varying time relations between the two beats (fig. 3). For example, in figure 2 showing varying degrees of fusion between a conducted beat (complex 1) and an electrically stimulated one (complex 10), all these variations are seen:

![Figure 5](image)

**Figure 5**

Similarity of WPW and fusion beats. Upper row: normally conducted beat (left) and anomalously excited beat (right) in patient with intermittent WPW syndrome. Lower row: selected complexes during electric stimulation in patient 60; stimulus appears as small spike artifact. Left, conducted beat with normal P-R interval and ineffective stimulus; center, fusion beat with short P-R interval, slurred upstroke of R, wide QRS, and unchanged P-S; right, pure electrically stimulated beat with ineffective P wave following the QRS.

shortened P-R, prolonged and abnormal QRS, and constant P-S in complexes 4 to 8; unchanged P-R with abnormal QRS in complex 3; and shortened P-S in complex 11.

Our observations on fusion show quantitative differences in the presence of bundle-branch block from those during normal intraventricular conduction, consisting of short P-R interval, slight or no increase in the abnormally long QRS, and consequent shortening of P-S. Accordingly, anomalous AV excitation in the presence of bundle-branch block would be expected to show similar variations.

Our determinations of myocardial conduction time are important in the assessment of the mechanism of anomalous AV excitation. Three divergent theories are current for the origin, pathway, and point of entry in the ventricle of the associated beat that fuses with the sinoatrial beat: first, that the sinoatrial impulse reaches the ventricle by way of peripheral accessory AV pathways as well as in the normal way;\(^{25}\) second, that the sinoatrial impulse divides and one portion is conducted through the AV node at an accelerated rate;\(^{26}\) and third, that an ectopic beat synchronous with the conducted beat arises high in the septum.\(^{27}\) In the WPW syndrome the P-R interval is often shortened by as much as 0.06 to 0.08 second;\(^{22}\) as Kossmann and Goldberg have pointed out, this maximal shortening (i.e., limit of fusion) measures the time that the associated beat takes to travel from its point of entry in the ventricle to the septum. This myocardial conduction time indicates that the point of entry must be at a distance from the septum similar to that of our electrodes. Such a distance, of 6 to 8 cm., is entirely consistent with an accessory pathway by which the associated beat reaches a peripheral portion of the ventricle. The distance, however, is quite incompatible with the other two theories, both of which propose that the associated beat first depolarizes the ventricle in the septum itself.

**Summary**

Quantitative studies of ventricular fusion beats were made in 40 patients with Stokes-
Adams disease after implantation of electric pacemakers in the left ventricle. The two fusing complexes came from an electric pacemaker and either a second electric pacemaker or a conducted sinoatrial beat. The limit of fusion, that is the maximal time between two fusing complexes, gives a measure of the conduction time and velocity between the two points of initial ventricular depolarization.

Conduction between two electric pacemakers and in retrograde direction from an electric pacemaker to the point of entry of a conducted beat was by way of ordinary myocardium; its velocity was about 1 meter per second. Conduction in antegrade direction, on the other hand, was by way of specialized Purkinje tissue; and its velocity was 4.5 to 9 times faster.

Variations in conduction times with bundle-branch block support this interpretation: retrograde conduction time did not change; antegrade conduction time was prolonged in left but was usually unchanged in right bundle-branch block.

The morphologic similarities of fusion beats in our patients to the features of anomalous AV excitation add strong evidence in man to the previous analogous experimental observations that the WPW complex is a fusion beat. Furthermore, quantitative comparison of WPW and our fusion beats supports the theory that the anomalous excitation in the WPW phenomenon, like the peripherally located artificial stimulus, enters the ventricle peripherally, by way of an accessory AV pathway.

Acknowledgment

We wish to thank Mr. Alan Bell for his valuable contributions to the early development of the ideas in this study and for his assistance in the analysis of the data.

References


The Obligation to Study and Acquire Knowledge

Hippocrates, in his famous oath which has been repeated by a hundred generations, did not include the study and knowledge of the doctor. He did not list it among his sacred obligations, such as that of looking after the health and protecting the life of his patients. Nevertheless, Codes of professional ethics from some countries have included this point among the doctor’s main duties. But nobody emphasizes it enough; it is never put near the top of the list. Yet I believe that professional ethics must start at exactly this point; in giving the patient the best service that medicine can offer, and repay the trust of the patient with the most efficient methods in existence. The help which the doctor gives should be limited only by the limits of knowledge in his time, and not those other limits, years back in time, set by his ignorance.

This has always been true, but it is much more so today, because today knowledge becomes out-of-date in a few years, whereas in the past it did not do so in a whole lifetime. Not to keep up-to-date, not to be aware of the latest developments, is to deprive the patient of their benefit, and subject him to mediocre and unproductive treatment, when he might have been saved by recently discovered methods.—Dr. IGNACIO CHÁVEZ.

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