Experimental Production of Ventricular Complex Simulating A, B, and C Types of WPW Syndrome

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SUMMARY

1) The ventricular fusion beat was produced in dogs with atrial pacing and the stimulation of various sites of ventricle. The interval between atrial and ventricular stimulation was controlled by a preset timer with a step of 1 msec.

2) The limit of the range of the A-V stimulation interval (D₁ and D₂) for the fusion beat was related to the normal activation time of the location of the ventricular stimulation.

3) The QRS morphology corresponding to the A type of WPW syndrome was obtained from the stimulation of posterior and lateral wall of the LV, that to B type from the base of the RV, and that to C type from the septal area of the LV, the posterior basal part and the apical 2/3 of the RV.

4) The longer interval of the atrial and ventricular stimulation caused the B type QRS at any site of the ventricular stimulation.

Additional Indexing Words:
Ventricular fusion beat  QRS configuration  Epicardial map

It has been known that the ventricular fusion beat can simulate the QRS complex in WPW syndrome. Butterworth and his associates succeeded to produce the delta wave with adequately timed stimulation of the ventricle. The QRS configuration depends on the site of the origin of abnormal ventricular activation. Clinically, the classification of A and B types has recently been drawing attention in relation to the location of accessory pathway and direct surgical approach to it. In addition to types A and B, the C type is distinguished in clinical cases, characterised by the lack of R wave in lead V₁. The present study intended to elucidate experimentally the factors producing these characteristic types. Stimulations of many ventricular sites

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were performed with variable degrees of the fusion with normal activation by means of an accurate delay circuit.

**METHODS**

In 26 dogs, the chest was opened under the nembutal anesthesia. After the sinus node was crushed, the right atrium was stimulated electrically by Stimulator A. Various epicardial sites of the ventricle were stimulated with another stimulator, Stimulator B, through bipolar electrodes with interelectrode distance of 1 mm. A preset timer described previously was used for an accurate time delay of 1 msec step between atrial and ventricular stimulation (Fig. 1).

After electrodes were attached to the atrium and to the ventricle, the chest was closed with rubber form soaked with physiological saline around the heart to simulate the normal condition before opening the chest. McFee orthogonal lead for the dog was used for recording X, Y, and Z leads scalar ECG and 3 planar projections of the VCG. Changing the time delay between the atrial and ventricular stimulation with a step of 1 msec, the configuration of the QRS complex was studied.

An arrival time of the normal activation at the site of epicardial ventricular electrodes was measured with the intensity modulation of the oscilloscope beam. Output of the preset timer was less than 100 µsec of time constant, so that a bright spot appeared on the beam when it was applied to the Z axis of the oscilloscope. Regulating the preset delay, the spot was brought to coincide with the intrinsic deflection of the bipolar ventricular electrogram on the beam. Then, the dial indicated the time between the atrial stimulation and the activation time of the epicardial site.

After Ueda’s classification of the WPW syndrome, the anteriorly located QRS loop of fusion beat was called A type. In B type, the initial QRS was directed anteriorly and the body posteriorly. The C type referred to the loop with both the initial portion and the body directed posteriorly. Loops of A type were further divided into A1 and A2 according to the inferior and superior orientation of the QRS loop in the frontal plane, respectively.

![Fig. 1. Production of the ventricular fusion beat.](image-url)
RESULTS

1) Conditions for the ventricular fusion beat

Fig. 2 shows an experiment with the epicardial stimulation site indicated as (5). When the ventricle was stimulated earlier than a critical time (D₁), the whole ventricle was activated by the epicardial stimulation and the same QRS loop was obtained. In the figure, the interval between the atrial and ventricular stimulation is indicated under the figures. At the interval of

![Fig. 2 - Ventricular fusion beat with epicardial stimulation site (5).](image)

Upper: VCG obtained with ventricular stimulation 20, 76, 110, and 120 msec after the atrial stimulation. F: frontal, T: transverse, LS: Left sagittal.

Lower: Schematic representation of the location of ventricular stimulation and the timing of the ventricular stimulation. The ventricular stimulation within the range of 76 to 114 msec after the atrial stimulation resulted in the fusion beat.
76 msec, the QRS loop started to change near at the maximal QRS vector. Increasing the atrial and ventricular stimulation interval, alterations of the QRS loop was more and more prominent and finally the normal conduction resulted at 114 msec. In this case, the ventricular fusion started at the atrial and ventricular stimulation interval of 76 msec (D1) and disappeared at 114 msec (D2). Fig. 3 also shows D1 and D2 for 3 epicardial sites A, B, and C. The activation time of A, B, and C in normal activation (atrial stimulation only) was 155, 172, and 184 msec, respectively. It can be seen that the starting time D1 was inversely related to the normal activation time, that is, D1 was later in A with early activation time and was earlier in C with late activation time. D2, on the other hand, was parallel with the normal activation time.

The same relation was seen in 5 dogs as shown in Fig. 4. Increase in the rate of atrial stimulation in one dog caused the prolongation of the PQ interval and parallel prolongation of the interval from the atrial stimulation to D1 and to D2.

Theoretically, the limit of the normal activation (D2) is expected to be identical with the normal activation time. D2 was, however, determined in this study from the observation of the vectorcardiographic QRS loop. Certain
Fig. 4. Relationship between the onset (D₁) and the end (D₂) of the fusion beat in 5 dogs. See text.

Table I. The Relationship between Ventricular Activation Time and D₂

<table>
<thead>
<tr>
<th>Site</th>
<th>Cond. Time (CT)</th>
<th>D₂</th>
<th>CT—D₂</th>
<th>PQ</th>
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<tr>
<td>1</td>
<td>147</td>
<td>107</td>
<td>40</td>
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<td>9</td>
<td>204</td>
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Mean 32 msec
extent of the abnormal activation seemed to be required to produce discernible changes in the QRS loop. Table I shows the comparison of the normal activation time and D₂ for several epicardial locations in 2 dogs. No. of sites 8 and 9 in Table I are the results of PQ prolongation induced by rapid atrial stimulation in one dog. The difference between the normal activa-

Fig. 5. Stimulation of the epicardial site on the lateral wall of the left ventricle. When the ventricular stimulation was applied at 30 msec after the atrial stimulation, the entire ventricle was activated by the ventricular stimulation. At 40 msec, the contribution of normal activation occurred and the fusion beat of type A₁ was observed. Increasing the interval between the atrial and ventricular stimulation, the QRS location was more and more posteriorly showing the transition to type B.
tion time and \(D_2\) was 32 msec on the average.

2) Dependency of the QRS morphology on the interval of atrial and ventricular stimulation

The QRS configuration similar to that in A, B, and C type of WPW syndrome will be described below.

Fig. 6. Stimulation of the epicardial site on the lateral wall of the left ventricle. When the ventricular stimulation was applied at 30 msec after the atrial stimulation, the entire ventricle was activated by the ventricular stimulation. At 40 msec, the contribution of normal activation occurred and the fusion beat of type \(A_2\) was observed. Increasing the interval between the atrial and ventricular stimulation, the QRS location was more and more posteriorly showing the transition to type B.
A) A type

Fig. 5 shows an experiment with epicardial electrodes on the lateral wall of the left ventricle. At the atrial and ventricular stimulation interval of 30 msec, the ventricle was entirely activated by ventricular stimulation. At 40 msec, the fusion beat of A type resulted. The QRS loop in H plane was directed anteriorly and was inscribed clockwise. The Z lead showed R deflection. When the A-V stimulation interval was prolonged, the terminal

Fig. 7. Stimulation of the basal area of the right ventricle. The fusion beat was obtained within the range of atrial and ventricular stimulation interval of 40 to 73 msec, always showing type B configuration.
force changed its location posteriorly. At 70 to 80 msec, the body was also directed posteriorly and the R wave in Z lead decreased in height and the S wave appeared. The whole morphology was that of B type. In the frontal plane, the QRS body was directed inferiorly when the A-V stimulation interval was 50 msec or longer, while it was directed superiorly for shorter A-V stimulation interval.

Fig. 6 shows another case of A type. The fusion beat started again

Fig. 8. Stimulation of the right ventricular wall. The fusion beat was observed at the atrial and ventricular stimulation interval of 40 msec. The QRS loop was directed posteriorly showing the characteristics of the C type. Later stimulation of the ventricle caused the initial anteriorly directed vector and the transition to the B type.
at the A-V stimulation interval of 40 msec and the QRS loop was directed anteriorly, while the body was oriented superiorly in the frontal plane (A2 type). The Y lead shows QS or W pattern indicating the superior direction of the initial vector.

B) B type

Fig. 7 shows an experiment with epicardial electrodes on the basal area of the right ventricle. At the A-V stimulation interval of 30 msec, "pure ventricular beat" was observed. At 40 msec, the fusion beat started. The initial QRS vector was directed anteriorly and the body posteriorly. The Z lead shows rS complex. In this case, the B type of the QRS complex was observed regardless of the A-V stimulation interval.

C) C type

Fig. 8 shows an experiment with epicardial electrodes on the right ven-
Fig. 9. Stimulation of the right ventricle. Findings are similar to those in Fig. 8 but the transition of C to B type occurred earlier in this case.

The fusion beat occurred at the A-V stimulation interval of 40 msec. The entire QRS loop was directed posteriorly and the Z lead showed QS complex (C type). At the A-V stimulation interval of 70 msec, the initial QRS vector was changed to have the anterior direction. This case showed the C type for relatively long range of the A-V stimulation interval, while in a case shown in Fig. 9 the 50 msec interval already caused the change from C to B type.

3) The epicardial sites of stimulation and the morphology of the fusion beat

Results of 17 epicardial sites were summarized in Fig. 10. As stated, the B type was obtained for all the locations when the A-V stimulation interval was longer. For the shorter interval of A-V stimulation near D1, the type observed depended on the epicardial site. The basal portion of the right
ventricle produced B type and apart of basal portion of RV, the apical two thirds of the right ventricle as well as the septal area of the left ventricle caused the C type. $A_1$ type was obtained from the lateral wall of the LV, and $A_2$ from the posterior or apical area of the LV.

**DISCUSSION**

In this study an accurate and reproducible time delay was obtained by the present timer with a crystal oscillator, which is essential for the control of the timing of atrial and ventricular activation. Measurement of the ventricular activation time was also greatly facilitated with the preset timer and the intensity modulation of the oscilloscope.

The presence of both normal activation and the activation from the ventricle was required for the ventricular fusion beat. The ventricular stimulation must be applied before the normal activation arrives at that location. The difference between the normal activation time and the time of ventricular stimulation for the recognizable fusion beat ($D_2$) was 32 msec on the average.
The earliest limit of the ventricular stimulation (D1) for the fusion beat was also related to the normal activation time of the site of ventricular stimulation. After D1, the normal activation contributes to the QRS complex. The inverse relation between the normal activation time and D1 means that the retrograde conduction from the epicardial site to the AV conduction system is parallel with the antegrade conduction from the atrium to the ventricular site.

The QRS morphology of the fusion beat was compared with A, B, and C types of WPW syndrome with respect to the initial vector. The distribution of the epicardial area producing A, B, and C types can roughly be understood in relation to the direction of the ventricular activation. The A type is obtained from lateral or posterior wall of the LV, which is expected to produce the initial force directed anteriorly. The lateral wall is located relatively superiorly and is associated with inferiorly directed vectors, while the posterior wall is located more inferiorly and produces the superiorly directed vectors. This may be related to the distinction of the areas causing A1 and A2 types. The stimulation of the RV appears to cause the inversion of the normal left to right activation of the interventricular septum and the disappearance of the anteriorly located initial force. The LV is located posteriorly, so that the activation from the RV causes the posterior location of the entire QRS loop (C type). The results of the present study of the epicardial stimulation are in agreement with observations of Ueda et al that the activation started in the basal area of LV in cases with type A WPW syndrome and in the base of RV in those with Types B and C. They are also comparable with recent observations on the epicardial activation patterns in cases with WPW Types A and B. Although areas indicated A and C in Fig. 10 produced A and C types of QRS complex when they were stimulated earlier, the transition of A or C type to B type was observed, when the ventricular stimulation was applied later and the normal activation predominated. With increase in the contribution of the normal activation, some of the C type loop were changed to B type earlier and others showed this change later. The latter is characterized by the ventricular stimulation site near the ventricular septum, which suggested the persistence of abnormal septal activation for longer period. The basal area of the RV is late in normal activation, so that the stimulation of this area ought to produce C type but causes relatively minor changes in the QRS loop and is associated with B type. The fact that the prolongation of the A-V stimulation interval causes the B type at any site of ventricular stimulation suggests that the QRS complex in cases with WPW syndrome is also susceptible to change. Two types of the QRS in the same patients have been observed and 2 anomalous pathways have been suggested.
Such a case reported from this laboratory has been interpreted as the difference in the contribution of normal and abnormal pathways.\textsuperscript{6,10} The present experimental study gives supports for this possibility and suggests that greater contribution of the normal activation results in the B type in any case.

\textbf{References}