Simultaneous Closure of the Tricuspid and Mitral Valve in Ebstein’s Anomaly

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Summary

Echocardiographic studies of a 23-year-old man with Ebstein’s anomaly and type B Wolff-Parkinson-White syndrome showed almost simultaneous closure of the tricuspid and mitral valve. After administration of edrophonium chloride, the tricuspid valve closed 20 msec earlier than the mitral valve. However, the closure of the mitral valve preceded that of the tricuspid valve after administration of ajmaline and atropine sulfate by 90 msec and 60 msec, respectively, concomitant with obliteration of preexcitation. From these observations, the simultaneous closure of the tricuspid and mitral valve of this case appears to depend on the preexcitation of the right ventricle.

Additional Indexing Words:
Ebstein’s anomaly Simultaneous closure of mitral and tricuspid valve Echocardiography in Ebstein’s anomaly

We recently suggested1) that some cases of Ebstein’s anomaly may be associated with early closure of the tricuspid valve although the abnormally delayed closure of the anterior tricuspid leaflet (ATL) compared with that of the anterior mitral leaflet (AML) is believed to be the most characteristic finding of this anomaly,2)–10) and that the abnormal conduction system might play an important role in the tricuspid closure in Ebstein’s anomaly with type B Wolff-Parkinson-White (WPW) syndrome. After publication of our report, we observed another case of Ebstein’s anomaly with resembling findings. The purpose of this communication is not only to report a case of Ebstein’s anomaly with simultaneous closure of the mitral and tricuspid valve but to provide further insight into the etiology of the abnormally delayed closure of the ATL.

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ULTRASONIC TECHNIQUES

The echocardiographic examinations were performed using a phased array sector scanner (Toshiba, SSH-11), which uses a 2.4 MHz multitransducer array interfaced to a strip chart recorder (Honeywell, 1,219). The real-time 2-dimensional images were obtained in the long axis, and simultaneous M-mode echocardiograms were got after optimal beam selection. The tracings were recorded on photographic paper at a speed of 100 mm/sec. The closures of the ATL and the AML were defined as the “c” points when they came to the most posterior positions or when the anterior and posterior leaflets came together at the onset of systole.

CASE REPORT

A 23-year-old man, who had been confirmed as having Ebstein’s anomaly by a selective right atrial angiogram in other laboratory, was referred to our clinic for cardiac evaluation.

The present case was with type B WPW syndrome (Fig. 1A) and with echocardiographically unusual findings for Ebstein’s anomaly—the closure of the ATL compared with that of the AML was almost simultaneous, and the interval between the closure of the AML and of the ATL (Mc-Tc interval) was only 5 msec (Fig. 2A).

![Fig. 1. ECGs at rest (A), and after administration of edrophonium (B), ajmaline (C), and atropine (D). A) The ECG at rest showed sinus bradycardia and type B Wolff-Parkinson-White syndrome. B) After edrophonium, heart rate was reduced and the QRS complex prolonged. C), D) After ajmaline and atropine, the ECGs demonstrated normal sinus rhythm and right bundle branch block, and the delta wave was no longer present.](image-url)
Table I. Comparative Analysis of ECG and Echocardiogram Before and After Drugs

<table>
<thead>
<tr>
<th></th>
<th>Before drug (control)</th>
<th>After edrophonium</th>
<th>After ajmaline</th>
<th>After atropine</th>
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<tbody>
<tr>
<td>HR (msec)</td>
<td>49</td>
<td>46</td>
<td>50</td>
<td>92</td>
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<tr>
<td>PQ ( msec)</td>
<td>100</td>
<td>100</td>
<td>260</td>
<td>200</td>
</tr>
<tr>
<td>QRS (msec)</td>
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<tr>
<td>Q-Tc (msec)</td>
<td>125</td>
<td>100</td>
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<td>130</td>
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<td>Q-Mc (msec)</td>
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</tr>
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<td>20</td>
<td>-90</td>
<td>-60</td>
</tr>
<tr>
<td>Corrected Tc-Mc (msec)</td>
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<td>17.2</td>
<td>-80.0</td>
<td>-74.3</td>
</tr>
</tbody>
</table>

Abbreviations: HR = heart rate; PQ( msec) = PQ( msec) interval; QRS = duration of QRS complex; Q-Tc = interval between Q wave and c point of the anterior tricuspid leaflet (ATL); Q-Mc = interval between Q wave and c point of the anterior mitral leaflet (AML); Tc-Mc = interval between c points of ATL and AML; Corrected Tc-Mc = interval between c points of ATL and AML divided by preceding $\sqrt{R-R}$.

Fig. 2. Simultaneous echocardiographic recordings of the anterior tricuspid leaflet (ATL) and the anterior mitral leaflet (AML) at rest (A), and after administration of edrophonium (B), ajmaline (C), and atropine (D). Lead I ECGs were also shown. A) The ATL and the AML closed almost simultaneously. B) The ATL closed 20 msec earlier than the AML. C) The ATL closed 90 msec later than the AML. D) The ATL closed 60 msec later than the AML. Tc = closure of the ATL; Mc = closure of the AML; IVS = interventricular septum.
Drug interventions were performed, and both the M-mode echocardiograms and ECGs were recorded at the same time at rest and after intravenous administration of edrophonium chloride (10 mg), ajmaline (50 mg), and atropine sulfate (1.6 mg). The results were summarised in Table I.

Ten min after administration of edrophonium chloride, heart rate (HR) was reduced and duration of the QRS complex of the ECG prolonged (Fig. 1B). In this situation, the closure of the ATL was 20 msec earlier than that of the AML (Fig. 2B).

Five min after ajmaline and atropine sulfate, the ECG demonstrated normal sinus rhythm and right bundle branch block (RBBB), and the delta wave was no longer present (Fig. 1C, D). In these circumstances, the delayed closure of the ATL compared with that of the AML was observed by 90 msec and 60 msec, respectively.

DISCUSSION

The delayed closure of the ATL compared with that of the AML by more than 30 msec\(^2\)-10) is the most characteristic feature of Ebstein's anomaly, although a rare exception has been reported.\(^1\)

The simultaneous closure of the ATL and the AML may have occurred when the mechanical events had been earlier in the right side of the heart than in the left, i.e. the preexcitation of the right ventricle and/or severe pulmonary insufficiency (PI), or when the mechanical events had been later in the left than in the right, i.e. left bundle branch block (LBBB) and/or severe mitral stenosis (MS). PI and MS were ruled out by the cardiac catheterization, and there was no LBBB in this case (Fig. 1C, D). The most plausible etiology of the unusual findings was the contribution of the preexcitation.

The preexcitation pathway in the present case was probably located in the anterior wall of the right ventricle,\(^12\) and the preexcitation of the right ventricle might have resulted in the premature right ventricular pressure rise. Subsequently, the ATL and the AML might have closed simultaneously at rest before drug, and the ATL might have closed 20 msec earlier than the AML after edrophonium chloride. The earlier closure of the ATL after edrophonium chloride might have probably been because of the more dominant right ventricular preexcitation. After obliteration of the preexcitation, the ATL closed later than the AML, and the Mc-Tc interval was as seen in common Ebstein's anomaly (Table I).

This explanation is supported by the previous reports that the premature mechanical events can occur in some cases by the right ventricular preexcitation\(^13,14\) even in Ebstein's anomaly.\(^15\) In one (Case 2) of Pocock's 9 cases,\(^15\) the Mc-Tc interval would have probably been unusual.

Increasing HR may accentuate the delay of the ATL closure, since the
atrium with the atrialized ventricle may induce further incompetence of the emptying as the diastolic filling time gets shorter. In our durg study, however, HR and the Mc-Tc intervals were 50 beats/min and 90 msec after ajmaline, and 92 beats/min and 50 msec after atropine sulfate (Table I). In these cases, the delta wave was not present and the QRS complex showed the same RBBB pattern and the same duration (Table I). The Mc-Tc interval after ajmaline was 30 msec longer than that after atropine sulfate (Table I), and the increasing HR did not result in further delay of the ATL closure. The corrected Mc-Tc intervals (divided by the preceding √R−R) after ajmaline and atropine sulfate were 80.0 msec and 74.3 msec respectively, and approximately even (Table I). According to these observations, the increasing HR appears to play a minor role on the Mc-Tc interval.

From the above mentioned discussion the delay of the ATL closure in Ebstein’s anomaly may be fundamentally determined by the abnormality of the tricuspid valve itself and/or the potential contribution of the atrialized ventricle.

Again, although the abnormally delayed closure of the ATL compared with that of the AML is believed to be the most characteristic echocardiographic finding of Ebstein’s anomaly, there are exceptions, and Ebstein’s anomaly even with LBBB and with unusual Mc-Tc interval could be found in the future.

**References**

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