Cardiac Conduction Abnormalities During Percutaneous Balloon Mitral or Aortic Valvotomy

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To evaluate the electrophysiologic changes in the cardiac conduction system that occur during percutaneous mitral or aortic balloon valvotomy, we prospectively studied the conduction system in 19 patients (10 mitral, 8 aortic, and 1 both) undergoing this procedure. A His bundle electrogram was recorded in all patients, and when sinus rhythm was present, the atrioventricular (AV) node effective refractory period was measured. Holter monitoring was performed during and for 24 hours after the procedure. Follow-up electrocardiograms (ECG) were available in 11 patients 2.3 ± 1.5 months after the procedure. The AV node effective refractory period before (276 ± 86 msec) and after valvotomy (298 ± 85 msec) were not significantly different. The maximum His-Purkinje conduction time (HV interval) observed during valvotomy (66 ± 20 msec) was significantly longer (p < 0.01) than that measured before (57 ± 10 msec) or after (60 ± 18 msec) valvotomy. The mean HV intervals before and after valvotomy were not significantly different. The mean QRS complex duration increased from 95 ± 28 to 112 ± 28 msec during valvotomy and remained significantly prolonged (109 ± 26 msec) 24 hours after the procedure (p < 0.01). A new intraventricular conduction defect (QRS complex duration > 100 msec) or bundle branch block occurred in five of 13 patients who had normal QRS duration before the procedure. The change in HV interval did not correlate with the change in QRS complex duration. In four patients, the newly acquired intraventricular conduction defect was still present on follow-up ECG tracing. Complete heart block was not observed in any patient. Age, New York Heart Association functional classification, coronary artery disease, the valve dilated, annular area, effective balloon dilating area, change in valve area, and use of digoxin did not correlate with the change in HV interval or QRS complex duration by multiple regression analysis. Analysis of ECG data in a much larger group of 207 patients undergoing percutaneous mitral and aortic balloon valvotomy showed an 18% incidence of new-onset intraventricular conduction defect after valvotomy. (Circulation 1979;79:1197–1203)

Percutaneous balloon valvotomy (PV) is an alternative to surgical commissurotomies in patients with mitral stenosis and is a palliative procedure in patients with aortic stenosis when aortic valve replacement is deferred because of high operative risk.1-5 Complications of PV, including cardiac tamponade, valvular insufficiency, and atrial septal defect with left-to-right shunt, occur rarely and can often be avoided with careful technique. Although cardiac conduction defects have been reported as complications during mitral (PMV)2 or aortic (PAV)5 valvotomy, no report has yet described their incidence or the electrophysiologic changes in the conduction system that accompany PV. In this prospective study, we serially examined the cardiac conduction system during PV to determine the incidence, duration, and clinical significance of cardiac conduction abnormalities.

Methods

Atrioventricular (AV) conduction was studied in 19 patients who underwent PV (8 aortic valve, 10 mitral valve, and 1 both). Retrograde or antegrade PAV and PMV were performed as previously described.1-3,5 Coronary angiography was performed before PV in 18 patients. Significant coronary artery

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disease was defined as coronary angiographic stenosis greater than 70%. In each patient, before valvotomy, a 6F quadripolar electrode catheter with ring electrodes (5-mm interpolar distance) was introduced percutaneously into a femoral vein and advanced to the right ventricle. The catheter was then slowly withdrawn to a point near the tricuspid valve where a stable bipolar His bundle electrogram was obtained.

In patients whose rhythm was sinus, a 6F quadrripolar electrode catheter was introduced in similar fashion and advanced to the high right atrium to record a bipolar atrial electrogram for programmed atrial stimulation and atrial pacing. The His bundle electrogram and the atrial electrogram signals were filtered (30–500 Hz) and displayed simultaneously with two surface electrocardiographic leads on a multichannel oscilloscopic recorder (VR 16; Electronics for Medicine, Pleasantville, New York). Recordings were made at paper speeds of 100 and 250 mm/sec.

The His bundle electrogram was recorded before valvotomy in all 19 patients; it was also recorded immediately after the initial balloon inflation and serially at 5-minute intervals during the period of multiple balloon inflations until the end of the procedure. The mean number of balloon inflations was 3.4±2.1/patient, performed during a period of 10–35 min/patient for PMV. The time period during which repeated His bundle electrograms were recorded every 5 minutes varied from 20 to 50 minutes (mean, 31±9 minutes). Similarly, the mean number of balloon inflations was 4.5±1.9/patient during a period of 10–25 minutes for PAV. His bundle electrograms were repeated every 5 minutes during a period of 20–50 minutes (mean, 28±11 minutes). In the only patient who underwent both PAV and PMV, PAV (three dilations) was performed before PMV (two dilations) with the His bundle electrogram recorded every 5 minutes during a 30-minute interval.

The AV nodal conduction time (AH interval) was measured as the time from the first rapid deflection in the right septal atrial electrogram to the first rapid deflection in the His bundle electrogram. The His-Purkinje conduction time (HV interval) was measured as the time from the first rapid deflection in the His bundle electrogram to the earliest ventricular activation on any intracardiac or surface electrogram. AH and HV intervals were measured by three observers unaware of the temporal relation of the recording to valvotomy. The average of these three measurements was used for analysis.

The effective refractory period (ERP) of the AV node was measured before and immediately after PV (last balloon inflation) in eight patients who were in normal sinus rhythm. Atrial premature extrastimuli (A1) that scanned diastole were delivered at twice the diastolic threshold after an eight-beat atrial drive train (A0) at a cycle length of 600 msec or when the intrinsic cardiac cycle length was less than 600 msec at the longest drive cycle length that consistently overrode the sinus rate. The ERP of the AV node was defined as the longest A1A2 interval at which A2 failed to elicit His bundle depolarization.

Standard 12-lead electrocardiograms (ECG) were obtained immediately before and 24 hours after PV. All patients had 2-lead Holter monitoring during and for 24 hours after PV. Eleven patients had a standard 12-lead ECG recorded 2.3±1.5 months after PV; in the eight remaining patients, follow-up ECGs were not available. The PR interval and QRS complex duration were measured before PV, at 5-minute intervals during the course of repeated balloon inflations, 24 hours after PV, and on follow-up ECGs. Intraventricular conduction defect (IVCD) was defined as a QRS complex duration exceeding 100 msec on the surface ECG.

Echocardiograms, performed in all but three patients, were analyzed by independent observers for the presence of valvular and annular calcification. The aortic and mitral annular dimensions were measured by two dimensional echocardiography. Mitral and aortic anteroposterior (AP) dimensions were measured from the apical long-axis window, and the mediolateral (ML) mitral dimension was measured from the apical four-chamber window. Aortic annular area was calculated assuming a circular shape (area = πAP²/4), whereas the mitral annular calculation assumed an elliptical shape [Area = π(AP×ML)/4]. All PMVs were performed by two balloons with 20-mm diameter except for one case where a single 25-mm balloon was inflated. A single 20-mm diameter balloon was used for all PAVs but one, in which two 15-mm diameter balloons were required. Effective balloon dilating area was calculated by geometric analysis described previously. This dilating area was normalized both by body surface area and by echocardiographically determined annular size.

In addition to the intracardiac electrophysiologic recordings in 19 patients, we analyzed 12-lead ECG recordings in 175 patients who underwent PMV and 165 patients who underwent PAV at our institution. At least one 12-lead ECG tracing recorded during the week before PV was available in 302 patients. Furthermore, in 207 of these 302 patients, ECG tracings recorded 1 day after PV and at a later date (1–14 months) were also available. These data were analyzed to determine the incidence and persistence of new-onset IVCD and heart block in a large population of patients undergoing PV.

**Statistics**

Electrophysiologic measurements during and after PV were compared with those before PV by the Student’s t test for paired observations. Multiple regression analysis was performed to evaluate the relations among the clinical, echocardiographic, electrocardiographic, and electrophysiologic parameters.

**Results**

The study included four men and 15 women (Table 1). The mean age was 70±15 years, and the
mean New York Heart Association functional classification was 3.8±0.4. The cardiac rhythm was sinus in 10 patients and atrial fibrillation in nine. All 13 patients taking digoxin had therapeutic serum concentrations of the drug at the time of PV. Nine patients had normal coronary arteries, four had single-vessel coronary artery disease, one had left main coronary artery stenosis, two had two-vessel coronary artery disease, and two had three-vessel coronary artery disease (Table 1).

The echocardiographically measured mean mitral anteroposterior dimension was 3.8±0.4 cm (range, 3.2–4.6 cm), and the mean mitral mediolateral dimension was 3.7±0.3 cm (range, 3.3–4.4 cm), whereas the mean aortic anteroposterior dimension was 2.5±0.3 cm (range, 2.1–2.7 cm). The corresponding mitral and aortic mean annular areas were 11.3±2.3 and 4.8±0.9 cm², respectively. Table 2 shows the annular area, effective balloon dilating area, and normalized effective balloon dilating area in individual patients.

**Atrioventricular Conduction**

The AH interval did not change significantly during the procedure in the patients with sinus rhythm (Table 3). The mean ERP of the AV node before (276±86 msec) and immediately after (298±85 msec) PV was also not significantly different (Table 3).

The maximum HV interval recorded during PV (66±20 msec) was significantly longer than that measured before (57±10 msec) or 30 minutes after (60±18 msec) the procedure (p<0.01) in 19 patients (Table 3, Figure 1). Despite HV interval prolongation, infra-His block did not develop in any patient during PV. The mean HV interval before and after PV was not significantly different (Table 3, Figure 1). Comparing the patients undergoing PAV to those undergoing PMV, no significant difference was noted in the change in mean HV interval. The ERP of the His-Purkinje system was shorter than that of the AV node and therefore was not encountered in any of the patients.

The mean PR interval in 10 patients with sinus rhythm was 183±44 msec before PV and did not change significantly during or 24 hours after the procedure at comparable cardiac cycle lengths (Table 3). Two patients had first degree AV block before PV that was still present on follow-up ECG. None of the patients developed complete heart block.

In the group of patients undergoing PMV in our institution and included in the ECG analysis, 79 patients were in sinus rhythm just before the procedure. In 60 of the 79 patients, there was no first degree AV block before PMV. The 12-lead ECGs taken a day after the procedure showed a new onset of first degree AV block in three of the 60 (5%) patients. Similarly, new onset first degree AV block was documented in five (7%) of the 73 patients undergoing PAV and manifesting sinus rhythm with normal PR interval before the procedure.

**QRS Complex Duration**

The mean QRS complex duration before PV was 95±28 msec and was significantly increased to a maximum of 112±28 msec during the period of balloon dilations (p<0.01) (Table 3, Figure 2). Unlike
the HV interval, the increase in QRS complex duration persisted after the procedure, and the QRS duration remained prolonged even at 24 hours after the procedure (Table 3).

Thirteen patients had normal intraventricular conduction (QRS complex duration ≤100 msec) before PV (Figure 2). Five of these patients developed a new IVCD or bundle branch block during the procedure; IVCD was still present in all five patients 24 hours later (Figure 2). Of the six patients with prolonged QRS duration at baseline measurement, there was marked but transient increase in QRS duration in only one (Figure 2). No new supraventricular or ventricular arrhythmias occurred during or for 24 hours after PV. One patient developed new atrial fibrillation 5 weeks after the procedure.

Follow-up ECGs were available in all five patients who developed a new IVCD or bundle branch block during PV. In four of the five patients, IVCD was still present at follow-up 2.3±1.5 months after the procedure. In one patient, left bundle branch block had resolved at 3 weeks after PV. In the remaining six patients in whom a follow-up ECG was available, PR interval and QRS duration had not changed from the baseline values.

Analysis of the ECG tracings in 302 patients undergoing PMV, PAV, or both showed atioventricular or ventricular pacing on the baseline tracing before PV in eight patients. Of the remaining 294 patients manifesting nonpaced baseline rhythms, IVCD predating PV was present in 134 (46%) patients. In 97 patients undergoing PMV, the mean baseline

<table>
<thead>
<tr>
<th>Patient</th>
<th>Valve dilated</th>
<th>Annular area (cm²)</th>
<th>EBDA (cm²)</th>
<th>EBDA-AA</th>
<th>EBDA/BSA</th>
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<tr>
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<tr>
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<td>4.76</td>
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<td>ND</td>
<td>3.14</td>
<td>. . .</td>
<td>2.09</td>
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</tbody>
</table>

EBDA, effective balloon dilating area; AA, annular area; BSA, body surface area; M, mitral; A, aortic; ND, not done.

Values corresponding to the mitral and aortic valves in the same and only patient who underwent both aortic and mitral valvotomy.

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**Table 3. Conduction Before, During, and After Valvotomy**

<table>
<thead>
<tr>
<th>Duration (msec)</th>
<th>Before PV</th>
<th>Maximum during PV</th>
<th>After PV 30 min</th>
<th>After PV 24 hr</th>
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<tr>
<td>AH interval</td>
<td>122±36</td>
<td>116±32</td>
<td>119±43</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>HV interval</td>
<td>57±10</td>
<td>66±20</td>
<td>60±18</td>
<td>&lt;0.01*</td>
<td></td>
</tr>
<tr>
<td>AVN ERP</td>
<td>276±86</td>
<td>66±20</td>
<td>298±85</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>PR interval</td>
<td>183±44</td>
<td>191±44</td>
<td>191±44</td>
<td>178±39</td>
<td>NS</td>
</tr>
<tr>
<td>QRS complex duration</td>
<td>95±28</td>
<td>112±28</td>
<td>112±28</td>
<td>109±26</td>
<td>&lt;0.01†</td>
</tr>
</tbody>
</table>

All values are mean±SD.
P, percutaneous valvotomy; AVN, atrioventricular nodal ERP, effective refractory period.

*Mean maximum HV interval vs. the pre-HV interval.
†Mean prevalvotomy QRS duration vs. the maximum, 30-minute, or 24-hour measurements.
QRS duration was 94.1±17.2 msec and became 97.6±17.9 msec 1 day after PMV (p=NS). The mean baseline QRS duration in 110 patients undergoing PAV was 112.6±26.4 msec, significantly longer (p<0.05) compared with the patients undergoing PMV. It measured 117.3±26.7 msec the day after PAV, which was not significantly different from the value before PAV. In the group with no IVCD before PV (160 patients), new-onset IVCD after PV was observed in 28 (18%) patients (13 PMV, 13 PAV, and 2 PMV and PAV), an incidence that was not significantly different (p=0.16) from the one observed in the small group of patients studied prospectively. In this group of 28 patients, baseline QRS duration was 91.2±4.3 msec and became 113.2±13.9 msec (p<0.01) 1 day after the procedure when none of the patients was receiving a new drug after PV, which is known to affect QRS duration. The mean cardiac cycle lengths observed in the ECG tracings before and after PV were not different, arguing against rate-related IVCD. The significant increase in QRS duration in this subgroup of 28 patients was persistent, and their follow-up ECG tracings, recorded 5.3±2.9 months after PV, manifested a mean QRS duration of 107.5±16.5 msec.

**Follow-up**

Neither syncope nor symptomatic bradycardia was reported in any of the 19 patients studied during the follow-up period. Two patients died of heart failure 1 week and 2 months after the procedure. One of these patients had an IVCD before PAV and did not experience HV interval or QRS complex prolongation during the procedure. In the large group of 175 patients undergoing PMV and 165 patients undergoing PAV, only one patient required new permanent pacemaker implantation for persistent complete heart block despite a higher incidence (1.5%) of transient complete heart block observed during the procedure.

**Statistical Analysis**

The change in HV interval did not correlate with the change in QRS complex duration. Furthermore, age, New York Heart Association functional clas-
sification, presence of coronary artery disease, the valve dilated, antegrade or retrograde approach during PAV, presence of valvular or annular calcification, annular dimension or area, absolute or normalized effective balloon dilating area, change in valve area, and use of digoxin did not predict the change in HV interval or in QRS complex duration by multiple regression analysis.

**Discussion**

Although several investigators list cardiac conduction abnormalities as potential complications of PV, this study is the first attempt to prospectively examine the incidence, type, and duration of these disturbances. In this study, transient changes in His-Purkinje conduction occurred commonly during PMV and PAV. There were no episodes of complete heart block, and AV nodal function appeared unchanged after the procedure. Similar to HV interval, increases in QRS duration were also common but more persistent. The incidence of new-onset IVCD was 38% in this group of 19 patients. However, a retrospective analysis of ECG data in a much larger group of patients undergoing PMV and PAV at our institution revealed a lower, but not significantly different, incidence (18%) of new-onset IVCD.

**Previous Studies**

McKay et al reported three episodes of left bundle branch block during PAV, which all resolved within 24 hours of the procedure. Palacios et al reported complete heart block in two patients during PMV, but AV conduction returned within 24 hours of the procedure. During 175 PMVs and 165 PAVs performed at our institution, transient complete heart block has been observed in five (1.5%) patients (two during PAV, two during PMV, and one during combined PAV and PMV), but only one patient required implantation of a permanent pacemaker. Thus, complete heart block occurs rarely during PV and was not detected in the present study because this group of 19 patients evaluated prospectively was not large enough to detect such a rare event.

**Pathogenesis**

Potential causes of conduction abnormalities during PV include mechanical effects on the His bundle and Purkinje fibers during balloon expansion, dam-
age during transseptal catheterization, damage related to valvular or annular calcification, and possible ischemic damage during balloon dilation.

Studies with superfused myocardial tissue, isolated whole heart preparations, and open-chest dogs have shown that myocardial stretch may slow electrical conduction.9,10 By a similar mechanism, stress on the His-Purkinje system during balloon inflation may be responsible for the transient increase in HV interval observed in this study. However, neither effective balloon dilating area and annular area, nor the change in valve area, which are factors related to the mechanical stress on the valve annulus, correlated with changes in the HV interval or QRS duration. B-Lundquist et al11 in their review of transseptal catheterization, reported the occurrence of bradycardia and hypotension due to vasovagal reactions. No episode of AV block or any conduction system abnormality was observed in that study. In the present study, episodes of HV interval and QRS complex prolongation occurred only after valvular dilation and were not temporally related to transseptal catheterization or dilation of the interatrial septotomy.

Mitral annular calcification is associated with an increased prevalence of AV block, left anterior hemiblock, and IVCD.12–14 In our study, 12 patients had echocardiographically demonstrable mitral annular calcification; however, mitral annular calcification did not correlate with HV interval prolongation or development of new IVCD during PV. Finally, neither the presence, location nor the severity of coronary artery disease correlated with HV interval increases or the occurrence of IVCD during PV.

Scheinman et al15 have shown that patients with an HV interval greater than 100 msec are at a high risk to develop spontaneous complete AV block. Although in our study, the HV interval increased transiently during PV in many patients; in all but one patient, it had returned to a value of 75 msec or less at the end of the procedure (Figure 2). Infra-His heart block did not develop in any of the patients.

HV interval prolongation did not correlate with the increase in QRS complex duration during the procedure. Transient HV interval prolongation in the absence of prolongation of the QRS complex could occur if the site affected by PV is close to the bundle of His, proximal to the bundle branches. Conversely, new-onset IVCD and bundle branch block could occur in the absence of HV interval prolongation if only one bundle or fascicle is affected by PV. However, the precise mechanism of each of these conduction abnormalities is not defined.

Conclusions

Although transient complete heart block has been observed rarely during PV, it did not occur in this small group of patients whose conduction system was examined prospectively during PMV or PAV. Prolongation of the HV interval was common, transient, and not associated with infra-His conduction block and did not correlate with prolongation of QRS duration. AV nodal conduction was unchanged after the procedure. Prolongation of the QRS duration and development of a new IVCD occurred less frequently but persisted longer compared with changes in HV interval. Neither HV interval nor QRS complex prolongation correlated with any of the hemodynamic, electrophysiologic, or echocardiographic parameters examined by multiple regression analysis. The precise cause of these conduction abnormalities remains to be defined.

References


Key Words • atrioventricular conduction • intraventricular conduction defect • His-Purkinje conduction • mitral stenosis • aortic stenosis
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