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Left Fascicular Blocks During Right-heart Catheterization Using the Swan-Ganz Catheter

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SUMMARY During insertion of Swan-Ganz catheters, mechanical right bundle branch block occurred in association with left posterior fascicular block in two patients and with left anterior fascicular block in two. None of the four patients had acute myocardial infarction or acute (spontaneous or iatrogenic) pulmonary disease. In two cases, electrophysiologic studies demonstrated the coexistence of intra- and infra-Hisian conduction delays and blocks. Although the right bundle branch block may have resulted from injury to the central or peripheral right branch, the left fascicular blocks could not be explained by direct trauma to these left-sided structures. Our findings support the recent clinical and experimental reports that show that left fascicular block (as well as right bundle branch block) may be due to lesions involving the His bundle; presumably because of longitudinal dissociation of this structure affecting the transverse interconnections. In one patient, 2:1 intra-Hisian block may have coexisted with bradycardia-dependent (phase 4) right bundle branch block. More studies are required to determine the implications of catheter-induced conduction disturbances in other clinical settings, such as acute myocardial infarction.

RARELY, complications result from the use of the Swan-Ganz catheters. Recent communications have reported distal migration of catheters with pulmonary artery injury or pulmonary infarction.1-4 Rhythm disorders and conduction disturbances, such as ventricular ectopic beats and traumatic right bundle branch block (RBBB), have also been noted.5-8 Because the catheter is confined to the right-heart cavities, axis shifts characteristic of left anterior fascicular block (LAFB) and left posterior fascicular block (LPFB) would not be expected to develop coincident with traumatic RBBB. However, these unusual associations can occur and are discussed in this report. We also examine possible electrophysiologic mechanisms.

Materials and Methods

Table 1 lists clinical data for four patients who, during insertion of a Swan-Ganz catheter, developed RBBB with LPFB (cases 1 and 2) or with LAFB (cases 3 and 4). Although one patient had old anteroseptal (and possibly inferior) myocardial infarctions, none had acute myocardial infarction by clinical, enzymatic or electrocardiographic findings, or acute pulmonary disease by clinical or x-ray findings or lung

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scans). One patient had congestive cardiomyopathy and three had systemic arterial hypertension with cardiomegaly and congestive heart failure (hypertensive cardiovascular disease). Hemodynamic monitoring was performed to evaluate the treatment of congestive heart failure, acute hypertensive crisis or volume depletion.

Triple-lumen, #7F, balloon-tipped, flow-directed thermodilution catheters (Edwards Laboratories) were introduced through an antecubital or internal jugular vein. After entering the appropriate vein, the distal lumen was connected to the pressure monitoring system. After balloon inflation, the catheter was advanced to the pulmonary artery under fluoroscopic control to obtain pulmonary artery pressures (table 1).

Although an electrocardiographic lead was visually monitored on the oscilloscope screen during the entire procedure, continuous tracings were obtained in only two patients. However, in all cases, a complete 12-lead ECG was recorded at the end of the procedure.

His bundle studies were performed in two patients, using techniques previously reported.*

Results

Right Bundle Branch Block and Pulmonary Artery Pressures

None of the patients had RBBB before insertion of the Swan-Ganz catheter. This conduction disturbance persisted for 6–11 hours (table 1).

The initial hemodynamic measurements showed that cases 1–3 had elevated pulmonary artery end-diastolic pressures. Measurements performed 4 hours after starting a drip infusion of nitroprusside revealed a decrease of the pulmonary artery end-diastolic pressure to acceptable levels (table 1). This response occurred when the RBBB was still present. Case 4 had hypovolemia with an initial low pulmonary artery end-diastolic pressure.

RBBB with LPFB

In case 1, continuous electrocardiographic recordings were obtained throughout the procedure. Appearance of the RBBB was preceded by (or coexisted with) right ventricular ectopic beats. The RBBB was initially bradycardia-dependent, and then became rate-independent.

In patients 1 and 2, the amount of rightward shift of the electrical axis from the control value was 110° and 120°, (table 1). The magnitude of this shift and the resulting right-axis deviation (which could not be attributed to acute lung disease) were in keeping with the diagnosis of LPFB.10

In case 2, the control ECG (fig. 1A) showed a left atrial abnormality and left ventricular hypertrophy with an electrical axis of +30° and a QRS duration of 85 msec. The RBBB pattern that appeared after insertion (fig. 1B and fig. 2, top) was associated with right-axis deviation (+140°) and a QRS duration of 100 msec. In this patient, the traumatically induced intraventricular conduction disturbances had disappeared 11 hours after insertion of the catheter (fig. 2B).

RBBB with LAFB

Case 3 had a control ECG showing old anteroseptal (and probably inferior) myocardial infarctions with an electrical axis of −15° (fig. 3A). The RBBB pattern, which appeared while the Swan-Ganz catheter was introduced, occurred without significant changes in rate. It was associated with a shift of the electrical axis to approximately −100°, consistent with the diagnosis of LAFB10 (fig. 3B). Total disappearance (6 hours later) of the RBBB was followed by a gradual shift to the electrical axis toward control values: −45° at 6 hours and −15° (as in the control ECG) at 10 hours.

In case 4 (figs. 4 and 5), traumatic RBBB coexisted not only with LAFB, but also with 2:1 atrioventricular (AV) block (fig. 4B). These conduction disturbances had disappeared 9 hours later (fig. 4C).

His Bundle Recordings

Intracardiac electrophysiologic studies, performed in cases 1 and 4, showed normal AH intervals. However, both had infranodal conduction distur-

Table 1. Clinical, Electrocardiographic and Hemodynamic Information

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Clinical diagnosis</th>
<th>Previous MI</th>
<th>EA (control)</th>
<th>EA (during RBBB)</th>
<th>EA (direction and magnitude of shift)</th>
<th>Duration of RBBB (hours)</th>
<th>Pulmonary artery pressures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Systolic</td>
</tr>
<tr>
<td>1</td>
<td>47</td>
<td>HCVD</td>
<td>No</td>
<td>0°</td>
<td>+120°</td>
<td>R (120°)</td>
<td>6</td>
<td>0 hours*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CHF</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4 hours†</td>
</tr>
<tr>
<td>2</td>
<td>57</td>
<td>HCVD</td>
<td>No</td>
<td>+30°</td>
<td>+140°</td>
<td>R (110°)</td>
<td>11</td>
<td>0 hours*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CHF</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4 hours†</td>
</tr>
<tr>
<td>3</td>
<td>52</td>
<td>AHD</td>
<td>ASW</td>
<td>−15°</td>
<td>−100°</td>
<td>L (80°)</td>
<td>10</td>
<td>0 hours*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CHVD</td>
<td>IW</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4 hours†</td>
</tr>
<tr>
<td>4</td>
<td>62</td>
<td>CM</td>
<td>No</td>
<td>+75°</td>
<td>−40°</td>
<td>L (105°)</td>
<td>9</td>
<td>0 hours*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HV</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4 hours†</td>
</tr>
</tbody>
</table>

Abbreviations: MI = myocardial infarction; EA = electrical axis; RBBB = right bundle branch block; R = right; L = left; HCVD = hypertensive cardiovascular disease; CHF = congestive heart disease; AHD = atherosclerotic heart disease; ASW = anteroseptal wall; IW = inferior wall; CM = congestive cardiomyopathy; HV = hypovolemia.

*Initial measurements.
†Measurements obtained 4 hours later.
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In case 1, the HV was prolonged (65 msec). In case 4 (fig. 5), the intracavitary recordings (corresponding to the rhythm strip in fig. 4B) showed that the 2:1 AV block was due to 2:1 intra-Hisian block. Whereas the first, third and fifth atrial deflections (after having traversed the AV node) were abnormally delayed within the His bundle (HH' of 60 msec), the second, fourth and sixth were blocked between the proximal His bundle site and the distal site, because H was not followed by H'. The conducted QRS complexes had a RBBB-LAFB pattern (figs. 4B and 5).

Discussion

RBBB After Insertion of the Swan-Ganz Catheter

It has been assumed that the right ventricular endocardial structures are protected from significant irritation by the catheter tip by the inflated balloon. Yet, induction of right ventricular ectopic beats and transient RBBB indicates that some degree of trauma does occur. Luck and Engel,5 who observed transient RBBB in three of 38 patients (7.9%), suggested that the catheter tip is not always the cause of mechanical irritation; RBBB may also result from a rigid catheter loop fixed at the site of venous introduction and at the site of pulmonary artery occlusion. Other authors have also described the development of a new RBBB during bedside pulmonary artery catheterization using balloon-tipped catheters.8-11-13

Moreover, Aguilar et al.,13 Rosen et al.,14 and Jacob-son and Scheinman18 reported that paroxysmal AV (HV) block could occur during recording of His bun-

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**Figure 1.** Case 2. (A) Control 12-lead ECG. (B) The rhythm strip (lead I) recorded after introduction of the Swan-Ganz catheter shows a significant change in QRS morphology.

**Figure 2.** Case 2. Twelve-lead ECGs. (A) Left posterior fascicular block with right bundle branch block after introduction of Swan-Ganz catheter. (B) Disappearance of the intraventricular conduction disturbances 11 hours later.
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Figure 3. Case 3. Twelve-lead ECGs before (A), immediately after (B), and 8 hours after (C) introduction of Swan-Ganz catheter.

ddle electrograms in patients with complete left bundle branch block. This could have been due to traumatic RBBB.

Catheter-induced RBBB Coexisting with Intra-Hisian Block

When the association of RBBB and intra-Hisian block results from mechanical lesions affecting these two separate structures, either both lesions are traumatic, or one is traumatic and the other spontaneous.

For example, case 1 of Jacobson and Scheinman18 and our case 4 had periods of second-degree intra-Hisian block with RBBB in the conducted beats. Although mechanical injury to the corresponding structures cannot be excluded, the RBBB could have been a pre-existing (latent) nontraumatic bradycardia-related (phase 4) conduction disturbance, exposed only when the time of arrival of excitation at the right bundle branch was made late enough by the traumatic second-degree intra-Hisian block.

Figure 4. Case 4. Recordings before (A1 and A2), immediately after (B), and 9 hours after (C) introduction of Swan-Ganz catheter. Strip B shows 2:1 atrioventricular (AV) block and right bundle branch block with left anterior fascicular block (fig. 5).
Left Fascicular Blocks Related to Right-sided Catheterization

Production of LAFB and LPFB by catheters located in the right-sided cavities cannot, of course, be due to direct trauma to left-sided structures. Yet, these unusual findings may be explained by assuming that left fascicular blocks, as well as RBBB, can be produced by lesions limited to the His bundle.\(^{18-26}\)

Longitudinal dissociation of conduction within the His bundle must be present for this to occur, but there is disagreement as to the mechanism, especially in regard to the role played by the transverse interconnections between the longitudinal strands.

However, the LAFB in case 4 could have been a preexisting (latent) bradycardia-dependent (phase 4) conduction disturbance exposed by the second-degree intra-Hisian block. Furthermore, Fabregas et al.\(^{18}\) and Scherlag et al.\(^{26}\) as well as the clinical observations of Narula\(^{26}\) suggest that the bundle branch block and left fascicular block produced by intra-Hisian lesions can also be rate-related. Fabregas et al.\(^{18}\) showed that bradycardia-dependent (phase 4) block may sometimes occur. They showed that, after the His bundle was injured, 1:1 conduction to the left fascicles coexisted with 2:1 conduction to the right bundle branch. The increased duration of the interval between the stimulus artifact and the beginning of phase 0 depolarization of the action potential of the right bundle branch was attributed to the slow conduction resulting from an increase in the slope of diastolic depolarization.

Clinical Implications

RBBB produced by the Swan-Ganz catheters has been clinically significant only in patients with pre-existing left bundle branch block. Although we present only four patients, there could be a subgroup of patients, with or without latent disease of the ventricular specialized conduction system, in whom mechanical trauma can produce left fascicular blocks or bundle branch blocks.

However, prospective studies are needed. These require continuous electrocardiographic monitoring (ideally using at least two leads) during the procedure. Also needed is the establishment of a priori assumptions regarding the number of consecutive beats showing a given conduction disturbance that are required to consider that these have been mechanically induced.\(^{26}\)

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