Coronary Sinus-Ventricular Accessory Connections Producing Posteroseptal and Left Posterior Accessory Pathways Incidence and Electrophysiological Identification

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Background—The coronary sinus (CS) has a myocardial coat (CSMC) with extensive connections to the left and right atria. We postulated that some posteroseptal and left posterior accessory pathways (CSAPs) result from connections between a cuff of CSMC extending along the middle cardiac vein (MCV) or posterior coronary vein (PCV) and the ventricle. The purpose of the present study was to use CS angiography and mapping to define and determine the incidence of CSAPs and determine the relationship to CS anatomy.

Methods and Results—CSAP was defined by accessory pathway (AP) potential or earliest activation in the MCV or PCV and late activation at anular endocardial sites. A CSAP was identified in 171 of 480 patients undergoing ablation of a posteroseptal or left posterior AP. CS angiography revealed a CS diverticulum in 36 (21%) and fusiform or bulbous enlargement of the small cardiac vein, MCV, or CS in 15 (9%) patients. The remaining 120 (70%) patients had an angiographically normal CS. A CSMC extension potential (CSE), like an AP potential, was recorded in the MCV in 98 (82%), in the PCV in 13 (11%), in both the MCV and PCV in 6 (5%), and in the CS in 3 (2%) of 120 patients. CSMC potentials were recorded between the timing of atrial and CSE potentials.

Conclusions—CSAPs result from a connection between a CSMC extension (along the MCV or PCV) and the ventricle. The CS is angiographically normal in most patients. (Circulation. 2002;106:1362-1367.)

Key Words Wolff-Parkinson-White syndrome ▪ coronary sinus ▪ angiography

Coronary sinus (CS) diverticula1–5 have been associated with posteroseptal and left posterior accessory pathways. These diverticula contain myocardial fibers that connect to both the ventricle and the CS myocardial coat.1,2 The CS myocardial coat, present in all individuals,5 is anatomically6–9 and electrically10 connected to both atria, completing the accessory connection.

We hypothesized that a connection between the CS myocardial coat and the ventricle could produce an accessory pathway (CSAP) in the absence of a diverticulum (Figure 1). In an anatomic study of 240 human hearts, von Ludinghausen and coworkers6 found sleeve-like extensions of the CS myocardial coat covering the terminal portion of the middle cardiac vein (MCV) and posterior coronary vein (PCV) in 3% and 2% of hearts, respectively (CS myocardial extensions [CSE], Figure 1). They also found myocardial cords extending around the atrioventricular (AV) groove branch of the distal left circumflex coronary artery in 6% of hearts. These myocardial sleeves or cords could serve as a connection between the ventricle and the CS myocardial coat, forming a CSAP. Such a connection could explain a complex of unusual observations found in some patients with posteroseptal or left posterior accessory pathways, including the recording of earliest endocardial antegrade ventricular activation (rapid downstroke on the unfiltered unipolar electrogram) at least 15 ms after the onset of the far-field ventricular potential and at a site 1 to 3 cm apical to the mitral and tricuspid anuli; an oblique course with the ventricular end of the accessory pathway oriented toward the septum (and MCV) and atrial end oriented laterally, consistent with the oblique orientation of the fibers connecting the CS myocardial coat with the left atrium;6–9 and attempts to ablate these pathways at the site of earliest retrograde atrial activation often producing a shift in the site of earliest atrial activation (mimicking multiple pathways) consistent with the extensive connections between the CS myocardial coat and the atria.
The purpose of the present study was to use CS angiography and mapping of the CS and its tributaries to identify electrophysiological criteria for CSAP and to determine the incidence and relationship between CSAPs and anatomy of the CS and its branches, including diverticula.

**Methods**

**Study Population**
The study population consisted of 480 consecutive patients referred for catheter ablation of a posteroseptal or left posterior accessory pathway.

**Electrophysiological Study and CS Angiography**
Electrophysiological study was performed as previously described under sedation (fentanyl and midazolam) or general anesthesia (propofol).11–13 Retrograde CS angiography was performed in all patients suspected of having a CSAP on the basis of the presence of a negative delta wave in ECG lead II14 or mapping results from the present or previous electrophysiological study. Before 1994, CS angiography was performed using an 8F guiding catheter and rapid injection of 20 mL of contrast media. Visualization of individual veins or a CS diverticulum required selective injection. In 1994, a balloon occlusion technique was developed. An angiographic catheter with a compliant balloon located close to the tip (Vueport, Cardima) was inserted into the CS myocardial coat potential recorded near the orifice of the vein followed by activation of the CS myocardial coat, with the earliest CS myocardial coat (CSE in Figure 1). The small cardiac vein inserts into the MCV (as shown) or directly into the CS. LV indicates left ventricle.

**Definition of CSAP**
A posteroseptal or left posterior accessory pathway was identified as a CSAP when meeting the following criteria.

**During Antegrade Accessory Pathway Conduction**
The earliest endocardial ventricular activation (rapid downstroke on the unfiltered unipolar electrogram, local V, Figure 2) was recorded ≥15 ms after the onset of the far-field ventricular potential at a site located ≥1 cm apical to the tricuspid and mitral anuli. Ventricular activation recorded from the MCV, PCV, or CS diverticulum preceded endocardial ventricular activation (Figure 2). A high-frequency potential similar to an antegrade accessory pathway activation potential,13 presumably generated by an extension of the CS myocardial coat (Figures 1 and 3) was recorded from the MCV, PCV, or neck of a CS diverticulum before the earliest far-field ventricular potential, and the antegrade CSE potential was dissociated from local atrial and ventricular activation by the use of ventricular extrastimuli (Figure 4).15

**During Retrograde Accessory Pathway Conduction**
The earliest high-frequency potential (similar to a retrograde AP potential)15 was recorded from the MCV, PCV, or neck of a CS diverticulum, and was presumably generated by an extension of the CS myocardial coat (CSE in Figure 5). The CSE potential was followed by activation of the CS myocardial coat, with the earliest CS myocardial coat potential recorded near the orifice of the vein (Figure 5A, first complex, CS1 in electrograms MCV-Bip 3-4 and CS myocardial coat potential). The earliest high-frequency potential similar to an antegrade accessory pathway potential was recorded from the MCV, PCV, or CS diverticulum before the earliest far-field ventricular potential at a site located ≥1 cm apical to the tricuspid and mitral anuli. Ventricular activation recorded from the MCV, PCV, or CS diverticulum preceded endocardial ventricular activation (Figure 2). A high-frequency potential similar to an antegrade accessory pathway activation potential,13 presumably generated by an extension of the CS myocardial coat (Figures 1 and 3) was recorded from the MCV, PCV, or neck of a CS diverticulum before the earliest far-field ventricular potential, and the antegrade CSE potential was dissociated from local atrial and ventricular activation by the use of ventricular extrastimuli (Figure 4).15

**Results**
Of 480 patients referred for catheter ablation of a posteroseptal (403) or left posterior (77) accessory pathway, 279 (58%) had undergone 1 to 4 previous unsuccessful catheter or surgical ablation procedures. A CSAP was identified in 171 (36%) of the 480 patients, including 39 (19%) of 201 patients with no prior ablation and 132 (47%) of 279 patients with a previous failed ablation procedure.
CS Diverticula

Retrograde CS angiography demonstrated a CS diverticulum in 36 (21%) of 171 CSAP patients. The diverticulum extended from the CS in 26 patients (Figure 6A, 6B, 6E, and 6F), the MCV in 9 (Figure 6C and 6D), and both the CS and MCV in 1 patient (Figure 7C). Diverticula entered the CS with either a narrow (Figure 6A), intermediate (Figure 6E), or wide neck (Figure 6B). The diverticulum originated within 1.5 cm of the CS ostium (including MCV) in 27 patients (Figure 6E) and between 1.5 cm and the insertion of the great cardiac vein into the CS in 9 (Figure 6F). The width of the diverticulum (septal-lateral) ranged from 5 to 50 mm (median 18 mm). The diverticula contracted during ventricular systole (Figures 6C, 6D, 7A, and 7B), eliminating the contrast media within several beats.

Figure 3. Recording of antegrade CSE activation potentials along a 2-cm length of the MCV. Abbreviations as in Figure 2.

Figure 4. Validation of the antegrade CSE potential recorded from the MCV. A, During right atrial pacing at cycle length 520 ms (S1), a late ventricular extrastimulus (S2) advanced the local ventricular potential in the MCV electrograms by 55 ms (\(V_1 - V_2 = 465 \text{ ms}\)) without altering the timing or morphology of the CSE potential, dissociating CSE from local ventricular activation. B, An earlier S2 advanced the CSE potential by 25 ms (\(CSE_1 - CSE_2 = 495 \text{ ms}\)) without altering the timing or morphology of the surrounding atrial potentials (\(A_1 - A_2 = 520 \text{ ms}\) in HB, MCV-Bip3, and CS electrograms), dissociating CSE from local atrial activation. Abbreviations as in Figure 2.
In 4 patients, the diverticulum had multiple lobes or complex geometry (Figures 7A through 7C). Three other patients had 2 diverticula. Both diverticula inserted into the CS in 2 patients (Figure 7D). In the third patient, the 2 diverticula inserted into the junction of a large MCV and small collateral vein extending between the MCV and PCV (Figure 7E and 7F).

The CSAP was not related to the diverticulum in 2 of 36 patients. The CSAP was associated with the PCV in 1 and a collateral vein between the MCV and PCV in the other. In 2 other patients who each had 2 diverticula, the CSAP was associated with only 1 of the 2 diverticula (D2 in Figures 7D through 7F). Therefore, 4 of the 39 diverticula were not associated with an accessory pathway.

A CSE potential was recorded in the neck (or orifice of the diverticulum into the CS or MCV) in all 35 diverticula (34 patients) associated with a CSAP. The neck was large enough to allow mapping inside the body of 15 diverticula. Fusion of the CSE and earliest ventricular potentials was identified in all 15 and were located at 2 or more sites (multiple ventricular connections) in 6.

Of the 34 patients with a CSAP associated with a diverticulum, conduction occurred in both the antegrade and retrograde directions in 31 (91%) and only in the retrograde direction in 3 (9%). Of 31 patients with preexcitation, the delta wave was negative in lead II in 24 (77%).

Other Anomalies
Other venous anomalies were identified in 15 (9%) of 171 patients with CSAP. CS angiography showed fusiform enlargement of the terminal portion of the small cardiac vein in 3 patients and both the small cardiac vein and MCV in 9 patients (Figure 8A). Of these 12 patients, a CSE potential was recorded from the small cardiac vein in 2 and junction of the small cardiac vein and MCV in 8. Access to the MCV or small cardiac vein was unavailable in the remaining 2 patients, related to prior ablation procedures. In 1 patient, the MCV was narrowed at its insertion into the CS. A CSE potential was recorded from the CS at the orifice of the MCV. The second had occlusion of the small cardiac vein at its insertion into the MCV. The CSE potential was not recorded from the MCV or CS.

CS angiography showed a bulbous enlargement of the MCV in 2 patients (Figure 8B) and a segment of the CS in 1 patient. A CSE potential was recorded from the bulbous region of the MCV in 2 patients. An earlier S2 advances both atrial activation (A1–A2 = 490 ms) and CS myocardial coat activation (CS1–CS2 = 490 ms) in MCV-Bip3–4 without altering the timing or morphology of the CSE potential, dissociating CSE; from local atrial activation and activation of the CS myocardial coat. C. An earlier S2 advances CSE, resulting in loss of the CSE potential (arrow), without altering the timing or morphology of the local ventricular potential, dissociating CSE; from local ventricular activation. Abbreviations as in Figure 2.

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Normal CS Anatomy
CS angiography demonstrated normal venous anatomy in 120 (70%) of 171 CSAP patients. A CSE potential was recorded between the timing of the CS myocardial coat potential and ventricular potential from the terminal portion of the MCV in
98 (82%), PCV in 13 (11%), both MCV and PCV in 6 (5%), and the floor of the CS between the MCV and PCV in 3 (2%) of the 120 patients. The CSE potential was recorded at various distances (5 to 20 mm) into the MCV or PCV (Figure 3). The terminal segment of the MCV and PCV often contracted during atrial systole (Figures 8C through 8D), consistent with a cuff of myocardial fibers. This was not a specific finding, however. Systolic contraction was occasionally observed in patients without a CSAP.

The accessory pathway conducted only in the antegrade direction in 1, only in the retrograde direction in 36, and in both directions in 83 of the 120 patients. The CSE potential was recorded at various distances (5 to 20 mm) into the MCV or PCV (Figure 3). The terminal segment of the MCV and PCV often contracted during atrial systole (Figures 8C through 8D), consistent with a cuff of myocardial fibers. This was not a specific finding, however. Systolic contraction was occasionally observed in patients without a CSAP.

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patients with no prior ablation. The CSAP was related to the diverticulum in 34 of the 36 patients. In those 34 patients, the myocardium in the wall of the diverticulum was connected electrically to the epicardial surface of the ventricle. The diverticular myocardium was connected electrically to the CS myocardial coat through its neck. The body of these diverticula contracted during ventricular systole, consistent with the presence of myocardial fibers.

These 36 patients represent the largest reported series of CS diverticula. Each diverticulum was unique, varying widely in size (5 to 50 mm) and shape. Some had multiple lobes. Most had a discreet neck (narrower than the body of the diverticulum but highly variable in diameter), which extended from any site along the length of the CS from close to the CS ostium to near the insertion of the great cardiac vein, although most were located within 1.5 cm of the CS ostium. In 25%, the diverticulum extended from the MCV rather than the CS. Two adjacent diverticula were present in 3 patients.

The relationship between CSAP and fusiform or bulbous enlargement of the veins is less clear. In an anatomic study of 150 human hearts, bulbous enlargement of the MCV was present in 30%.16 Bulbous enlargement of the MCV was identified angiographically in the present study in only 2 of 171 CSAP patients, suggesting the angiographic findings may be very subtle. In contrast, we are not aware of any prior description of fusiform dilatation of the small cardiac vein and MCV. The accessory pathway was related to the enlarged region of the small cardiac vein or MCV in all 12 patients, suggesting a significant relationship between fusiform enlargement and CSAP.

Of the 126 CSAP patients with preexcitation, the delta wave was negative in lead II in 88 (70%). The sensitivity of a negative delta wave in lead II in identifying a CSAP in the present study (70%) is lower than found in our previous study.14 That study, however, included only 14 patients with CSAP.

The extensive connections between the CS myocardial coat and the atria, the oblique course of the CS myocardial fibers, and the lack of a clear endocardial location for the ventricular insertion combine to complicate localization of CSAPs, frequently leading to failure of catheter ablation. In the present study, a CSAP was present in 47% of patients with a posteroseptal or left posterior accessory pathway and a prior failed attempt at ablation, compared with 19% without a prior ablation procedure.

Of the 201 patients with posteroseptal and left posterior accessory pathways and no prior ablation procedure, 81% did not meet the criteria for CSAP. In those patients, retrograde activation of the right or left atrium preceded activation of the CS myocardial coat. Neither earliest antegrade ventricular activation nor a CSE potential was recorded from the MCV or PCV. None had a CS diverticulum and only 2 had fusiform enlargement of the MCV or small cardiac vein. Therefore, as previously described by Becker and coworkers,17 the majority of accessory pathways in this region do not involve the CS myocardial coat or its extensions.

References
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