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
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). 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 patients. Multielectrode catheters were positioned in the right atrial appendage, His bundle region, CS, and right ventricle. An additional catheter was used for mapping the tricuspid anulus, CS, and its branches.

Programmed stimulation of the right atrial appendage, posterolateral CS, anterobasal right ventricular septum (para-Hisian pacing), and posterobasal ventricular septum (close to the accessory pathway)13 was performed, verifying the presence of a posteroseptal or left posterior accessory pathway. Mapping of the tricuspid anulus, CS, and venous branches, and in selected patients, the mitral anulus (transseptal or retrograde transaortic approach) was performed during antegrade and retrograde accessory pathway conduction. Filtered (30 to 500 Hz) bipolar and unfiltered (1 to 500 Hz) unipolar electrograms were recorded simultaneously from the mapping catheter. Because of the oblique course in these accessory pathways (atrial end oriented leftward), atrial pacing for mapping during antegrade accessory pathway conduction was performed from the right atrial appendage to provide greater separation between the atrial, CS myocardial coat, and ventricular potentials in the CS electrograms. For mapping during retrograde accessory pathway conduction, pacing was often performed at the basal posterolateral left ventricle from a posterior or lateral coronary vein (PCV catheter in Figure 2A) to provide greater separation between the ventricular and atrial potentials in the CS electrograms.13

**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, 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).

**During Retrograde Accessory Pathway Conduction**

The earliest high-frequency potential (similar to a retrograde AP potential) 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 8 ). The CS myocardial coat potentials propagated leftward (Figure 5A, CS 1 in electrograms CS 8 to CS 5 ), activating the left atrium before the atrial activation recorded from the MCV, PCV, or CS diverticulum. A posteroseptal or left posterior accessory pathway was identified as a CSAP when meeting the following criteria.

**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 (V1, V2 = 465 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, CSE = 495 ms) without altering the timing or morphology of the surrounding atrial potentials (A1–A2 = 520 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 and CS in 2 patients (Figure 8C). A CSE potential was recorded from the bulbous region of the CS in the single patient with that anomaly.

Of the 14 CSAPs related to venous enlargement, 5 conducted only in the retrograde direction and 1 conducted only in the antegrade direction. In the 9 patients with preexcitation, the delta wave was negative in lead II in 6 (67%).

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

Figure 5. Validation of retrograde CSE and CS myocardial coat potentials. A, First complex. During RV pacing at cycle length 520 ms (S1), the earliest retrograde potential was recorded from the MCV (CSE; in electrogram MCV-Bip2–3). This was followed by a CS myocardial coat potential at the orifice of the MCV (CS1 in electrograms CS2 to CS3) preceded the left atrial potential (A1). Because of the distance between the MCV and the left atrial activation site and the delay associated with reversing direction, A1 recorded from the proximal MCV is relatively late (CS1–A1=35 ms in MCV-Bip2–3).

A late atrial extrastimulus (S2) advances local atrial activation by 25 ms in the MCV-Bip2–3 electrogram (A1–A1=495 ms) without affecting the timing of CS potentials, dissociating CS1 from local atrial activation.

B, An earlier S1 advances local atrial activation (A1–A1=440 ms) and CS myocardial coat activation (CS1–CS1=490 ms) in MCV-Bip2–4 without altering the timing or morphology of the CSE1 potential, dissociating CSE1 from local atrial activation and activation of the CS myocardial coat.

C, An earlier S1 advances CSE1, resulting in loss of the CSE1 potential (arrow), without altering the timing or morphology of the local ventricular potential, dissociating CSE1 from local ventricular activation. Abbreviations as in Figure 2.
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. In the 84 patients with preexcitation, the delta wave was negative in lead II in 57 (68%) 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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**Discussion**

One of the primary findings of the present study is that the majority (70%) of CSAPs occur without a diverticulum or other venous anomaly, and the ventricular end is located close to the MCV or PCV. The recording of CS myocardial coat potentials between the timing of the atrial and ventricular potentials suggests the CSE potentials are generated by extensions of the CS myocardial coat and supports the hypothesis that the ventricular end of the accessory pathway is formed by a connection between an extension of the CS myocardial coat (along or near the vein) and the epicardial surface of the ventricle (Figure 1). This hypothesis is further supported by the systolic contraction of the CS and terminal segment of these veins (Figure 8C and 8D). The atrial end of the accessory pathway is formed by connections between the CS myocardial coat and the left and right atria. The oblique course of the inner fibers favors earlier activation of the left atrium, 1 to 3 cm leftward of the ventricular connection (Figure 1). However, in 7 patients with prior ablation in the orifice of the small cardiac vein, MCV, or neck of a CS diverticulum preventing retrograde activation of the CS myocardial coat (and the left atrium), a connection to the right atrium persisted.

CS angiography identified 1 or 2 CS diverticula in 36 patients, 7.5% of 480 patients with any type of posteroseptal or left posterior accessory pathway. These values overestimate the prevalence of diverticula, because 32 of the 36 patients were referred after at least 1 unsuccessful ablation procedure. CS diverticula were present in only 4 (2%) of 201 patients.
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.
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