Clin. Cardiol. 23, 149–154 (2000)

Echocardiography of the Coronary Sinus in Adults

IVAN A. D'CRUZ, M.D., FRCP, M. BASHAR SHALA, M.D.,* CAMEILA JOHNS, M.D.*

Department of Cardiology, VA Medical Center, and *University of Tennessee, Memphis, Tennessee, USA

Summary: The coronary sinus (CS) is a small tubular structure just above the posterior left atrioventricular junction. The CS can be imaged in several different echocardiographic views. Using zoom M-mode recordings of the CS in apical two-chamber view, CS caliber can be sharply imaged and easily measured during different phases of the cardiac cycle. We have recently shown that the CS narrows during atrial contraction in persons with sinus rhythm, but does not narrow at all if atrial fibrillation is present. Attenuation of CS narrowing occurs in patients with congestive heart failure and inferior vena cava plethora. Maximal CS caliber occurs during ventricular systole. Patients with poor left ventricular systolic function show mild CS dilatation. Greater CS dilatation is present in patients with persistent left superior vena cava, and huge dilatation when this anomaly is accompanied by absence of a right superior vena cava. Injection of sonicated saline into a left and then a right arm vein is diagnostically useful in confirming these two venous anomalies. Pulsed-wave Doppler of the CS can be recorded in the parasternal right heart inflow view. From this and from the CS cross-section area it may be possible to estimate coronary blood flow.

Key words: coronary sinus, left superior vena cava, congestive cardiac failure, two-dimensional echocardiography, contrast echocardiography

Introduction

The coronary sinus (CS) has been of interest and use to cardiologists in many ways: to electrophysiologists as an anatomic landmark during mapping procedures, to anesthesiologists perfusing the heart retrogradely with cardioplegic solutions, to researchers sampling coronary venous blood for myocardial metabolic studies, and so forth. However, the CS has received limited attention from echocardiographers, especially those studying adult patients.

Up to the present, the CS has been featured in the echocardiographic literature mainly with respect to certain congenital cardiovascular abnormalities, all of which involve anomalous drainage of blood into the CS, either directly or via a persistent left superior vena cava (SVC). The CS is dilated in all these conditions, which include (1) persistent left SVC;[1–4] (2) anomalous pulmonary venous drainage into the CS;[5] (3) absence of the upper inferior vena cava, a large left hemiazygos vein draining into the CS; (4) coronary arteriovenous fistula;[6] (5) unroofing of the CS (septal defect between CS and left atrium).[7] All but the first of these entities are rare and are encountered mainly by pediatric cardiologists.

Coronary Sinus Anatomy

The CS is a muscular tube about 2 to 3 cm long and 1 cm in caliber, about 1 cm above and parallel to the left atrioventricular junction.[8] It is in close contact with the left atrium and is in fact enveloped in a sheath of atrial myocardium. A small valve (valve of Vieussen) has been described at the junction of the great cardiac vein and the CS.[9] In addition to this vein, the CS receives several other veins draining the posterior and lateral aspects of the heart; the oblique entry of these small vessels into the CS may have a valvar effect in preventing reflux of blood from the CS.[10] This enables efficient transfer of blood from the CS to the right atrium during active CS contraction.

Unlike other veins, the CS wall is relatively thick and, instead of smooth muscle forming a tunica media, consists of striated myocardium continuous with that of the atria. This was shown 40 years ago by Coakley and King in humans and other mammals[11] and mentioned by Keith as early as 1903.[12]

The CS opens into the lower posterior aspect of the right atrium, as its orifice is located near the much larger orifice of the inferior vena cava. A small endocardial fold, the Thebesian valve, is situated at the CS orifice and is virtually continuous with the Eustachian valve; the two "valves" share a common embryological origin from the right valve of the sinus venosus. Whereas the Eustachian valve is usually visible on two-di-

Address for reprints:

Ivan A. D'Cruz, M.D.
Cardiology Section, Room 519A
VA Medical Center at Memphis
1030 Jefferson Avenue
Memphis, TN 38104, USA

Received: November 12, 1998
Accepted with revision: May 11, 1999

mensional (2-D) echocardiography and even conspicuous when large, the Thebesian valve is generally too small and thin to be visualized on echocardiography as a separate structure.

Coronary Sinus Dilatation

Congestive Heart Failure

Potkin and Roberts, in an autopsy study of 234 adult hearts (50 of them normal), found CS size normal in patients with systemic hypertension, aortic stenosis, hypertrophic cardiomyopathy, and cor pulmonale.[13] Mild CS dilatation was noted in patients with dilated poorly contracting ventricles, either ischemic or idiopathic dilated cardiomyopathy.[13]

In an echocardiographic study published recently in abstract form, CS size in the parasternal long-axis view was found to be larger in patients with than in those without pulmonary hypertension.[14] These authors found that CS size correlated positively with right atrial size and right atrial pressure, but not with right ventricular pressure or degree of tricuspid regurgitation.

In an echocardiographic study performed by us,[15] CS size was mildly but significantly increased in patients with congestive failure and significant venous congestion (manifested echocardiographically by inferior vena caval dilatation and plethora).

In our study, patients with atrial fibrillation had significantly larger CS than patients with sinus rhythm, whether or not those with atrial fibrillation were in cardiac failure.[15]

Echocardiography of the Normal Coronary Sinus (Fig. 1)

The CS can be identified in several different 2-D views.[16, 17] In the parasternal long-axis view, it is represented by a small sonolucency at the posterior left atrioventricular (AV) junction, which moves with the AV groove during the cardiac cycle. In this view the CS is anterior to and quite separate from the larger more posterior sonolucency of the descending thoracic aorta, which is fixed and does not participate at all in cardiac motion. In the parasternal long-axis view, the margins, and therefore the caliber of the CS, are not sharply defined.

The CS is better imaged in the apical 2-chamber and 4-chamber views. In the latter, the imaging plane is more poste-

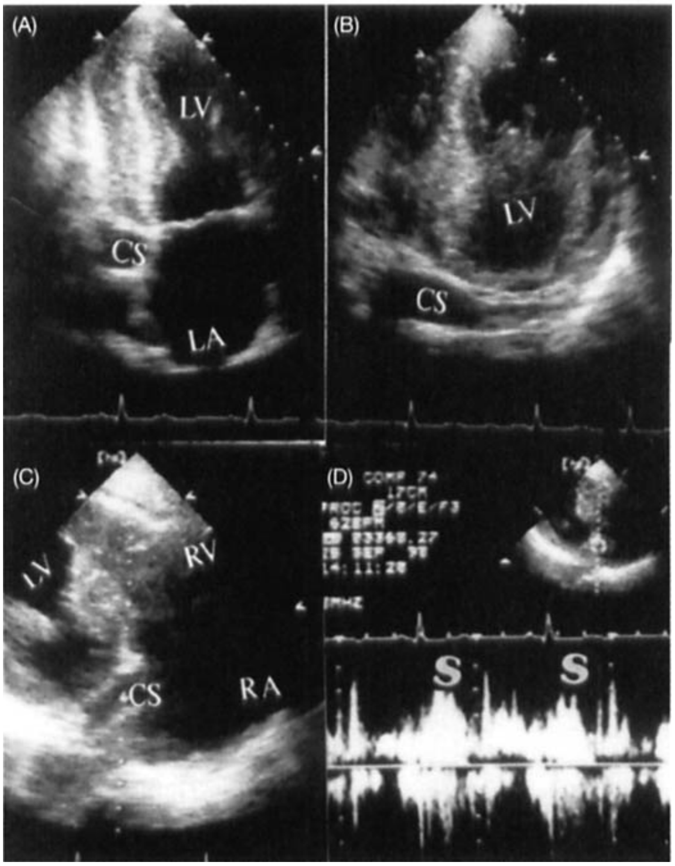

FIG. 1 Two-dimensional echocardiogram in apical 2-chamber (A) and modified apical 4-chamber view (B) showing the coronary sinus (CS) as a small sonolucency between left ventricle (LV) and left atrium (LA). The right ventricular inflow view (C) shows the coronary sinus entering the right atrium (RA). RV = right ventricle; LV = left ventricle. Pulsed-Doppler sampling at this site (D) shows a biphasic pattern, the systolic phase (s) taller than the diastolic phase.

rior than the conventional one that shows the mitral leaflets. We have found that the CS can be easily seen as a sharp round or oral sonolucency in the apical 2-chamber view. By positioning the M-mode cursor through the zoom image in this view, we obtained well-defined continuous recordings of CS caliber (Fig. 2) in 133 patients with sinus rhythm and normal ventricular function.[15] We noted that the CS narrowed just after the onset of the P wave, to a mean of a little more than half its caliber before the P wave; during ventricular systole CS caliber widened to a maximum.[15]

In 133 individuals with normal ventricular systolic function in sinus rhythm, the CS caliber was 8.27 ± 2.5 mm just before onset of the P wave, and 4.75 ± 1.89 mm at maximum CS contraction. The corresponding values for 42 patients with congestive heart failure and systolic venous congestion (inferior vena cava plethoric and dilated) were CS caliber 11.09 ± 3.12 mm just before P wave onset and 9.63 ± 3.18 mm at maximal atrial contraction. From these data, we concluded that (1) the CS narrows significantly during atrial contraction ($p < 0.05$) in persons with normal ventricular function; (2) in patients with congestive heart failure and plethoric inferior vena cava, this dynamic narrowing of the CS is significantly attenuated ($p < 0.05$). The mean ratio of the CS caliber at onset of the P wave to the CS caliber at maximum CS contraction was 1.83 ± 0.46 in persons with normal ventricular function, and 1.18 ± 0.21 in patients with right heart failure and venous congestion, a significant difference ($p < 0.0001$).

The decrease in CS size from maximum dilatation (in ventricular systole) to maximum contraction (in atrial systole) is impressive on M-mode as well as real-time 2-D echocardiography. Thus, the CS is not merely a conduit, it has its own "diastole" and "systole," which help propel its contents into the right atrium, the valve-like entry of cardiac veins into the CS preventing reflux as the CS "pumps" blood forward.

Coronary sinus narrowing, simultaneous with atrial contraction, was absent in all 40 patients with atrial fibrillation studied.[15] The CS narrowing was detectable but attenuated to varying degrees in most patients with congestive failure and systemic venous congestion. Our criterion for the latter was plethora and dilatation of the inferior vena cava on echocardiography. We concluded that attenuation of normal CS narrowing during atrial contraction, like attenuation or absence of normal inspiratory narrowing of the inferior vena cava, is evidence of high central venous pressure. A mildly dilated CS, in a patient with congestive heart failure, is shown in Figure 3.

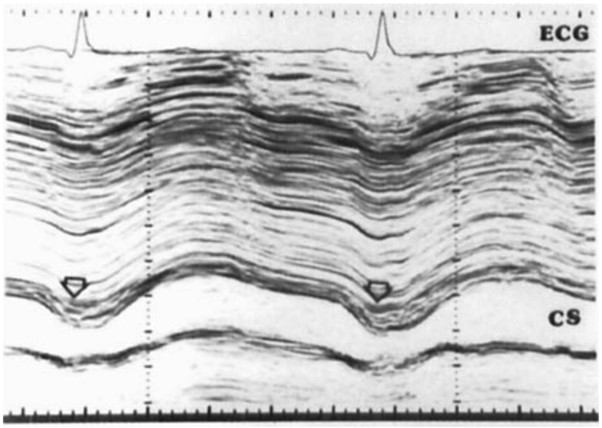

FIG. 2 Zoom M-mode through the coronary sinus (CS) in the same patient, in apical 2-chamber view, showing narrowing of this vessel and thickening of its wall, just after onset of the P wave. ECG = electrocardiogram.

Left Superior Vena Cava

In this anomaly, said to occur in about 0.5% of the general population and in 3 to 10% of those with congenital heart disease,[18] the left subclavian and left internal jugular veins drain not into a normal left innominate vein, but instead into a large venous channel which continues as a dilated CS, conveying venous blood from the left upper limb and left side of the head and neck to the heart. In normal persons, the left SVC is vestigial, being represented by a small vein on the posterior left atrial wall (Marshall's oblique vein of the left atrium) and fibrous band connecting Marshall's vein to the left innominate vein (Fig. 4).

Persistent left SVC is of no intrinsic hemodynamic importance, because there is no abnormal shunting of blood between systemic and pulmonary circulations. However, its recognition may be of significance to cardiologists because of the dif-

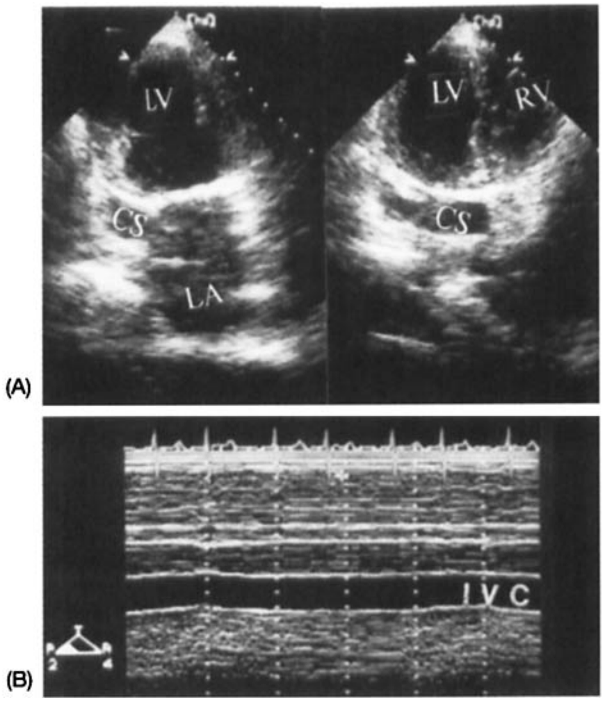

FIG. 3 Two-dimensional echocardiogram of a patient with congestive heart failure. (A) Apical 2-chamber view at left, unconventional view at right, showing a mildly dilated coronary sinus (CS). (B) M-mode of plethoric inferior vena cava (IVC) in the same patient. Abbreviations as in Figure 1.

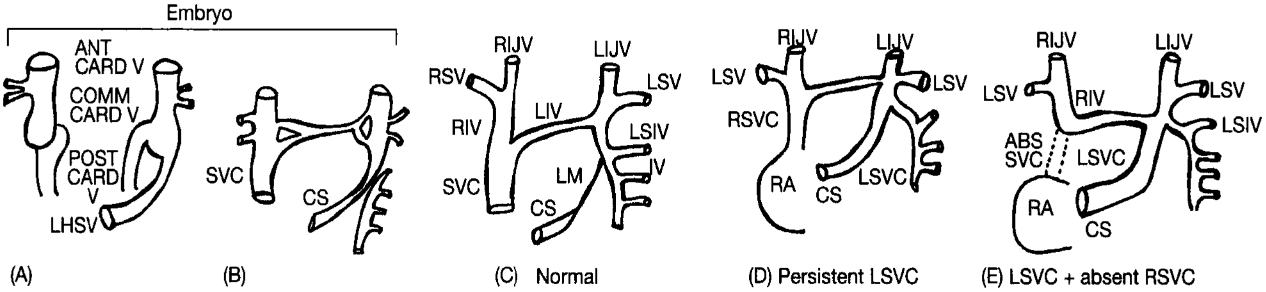

FIG. 4 Diagrams showing development of the superior caval venous system. (A, B) Anatomy in embryonic life, with (A) being at an earlier stage than (B). (C) Normal anatomy. (D) Usual type of persistent left SVC, (E) unusual type of left SVC with absent right SVC. The CS is dilated in patients with persistent left SVC, and even more dilated if persistent left SVC is associated with absent right SVC. ANT CARD V = anterior cardinal vein, COMM CARD V = common cardinal vein, POST CARD V = posterior cardinal vein, LHSV = left horn of sinus venosus, CS = coronary sinus, RSV = right subclavian vein, RIJV = right internal jugular vein, RIV = right innominate vein, SVC = superior vena cava, LIV = left innominate vein, LSV = left subclavian vein, LSIV = left superior intercostal vein, IV = intercostal vein, LM = ligament of Marshall, RSVC = right superior vena cava, LSVC = left superior vena cava, RA = right atrium, ABS SVC = absent right superior vena cava.

ferent transvenous courses of cardiac catheters or pacemaker leads on their way to the right atrium, and to the cardiac surgeon who may have to modify caval cannulation methods.

Persistent Left Superior Vena Cava: Echographic Manifestations

The dilated CS manifests as a sonolucency at the posterior left atrioventricular junction (Fig. 5), much larger than the normal small sonolucency at this site.[1–4] In the parasternal short-axis view, the CS sonolucency can be detected in CS long axis posterior to the left atrial space, and its continuity with the left SVC may be appreciated by fine adjustment of the imaging plane. The wide CS tubular space is easily demonstrated between left atrium and left ventricle in the 4-chamber apical view, imaged in a posterior plane.

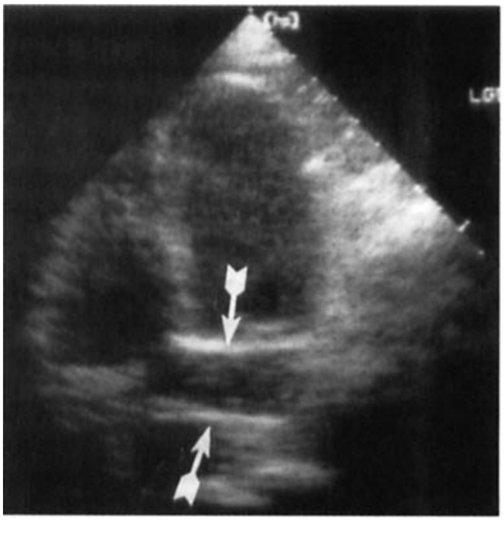

FIG. 5 Apical 4-chamber view showing a dilated coronary sinus (arrows) in a patient with left superior vena cava.

A rare variant of persistent left SVC is absence of the normal right SVC, so that the entire venous return of the upper body (normally conveyed by a normal SVC) enters the right atrium through a hugely dilated CS.[19–21] much larger than that seen with a left SVC, forming an "extra chamber" between left atrium and ventricle.

Contrast Echography in Persistent Left Superior Vena Cava

One of the earlier applications of contrast echocardiography, still often used in routine clinical practice today, is the confirmation of a suspected diagnosis of persistent left SVC by injecting sonicated saline into a left arm vein and observing its prompt appearance in the dilated CS before it opacifies the right atrium (Fig 5). This must be followed by injection of contrast into a right arm vein, which produces right atrial opacification without CS opacification.

In the above-mentioned rare anomaly of absent right SVC (Figs. 6 and 7) and an enormously dilated left SVC carrying the entire upper body venous return, right antecubital saline injection and left antecubital injection will produce the same result, that is, opacification of the huge CS just before right atrial opacification. Several reports of transesophageal visualization of a persistent left SVC have appeared since 1991;[22–24] in the case of Konecky *et al.*, absence of a right SVC was also noted on TEE.[21]

Doppler of Coronary Sinus Flow

For many years, Doppler recordings of CS blood flow had not been done after the CS had been identified in various 2-D planes. The probable reason for this was that in the apical as well as parasternal views the direction of flow in the CS was approximately perpendicular to the transducer. However, the parasternal right ventricular inflow view, in which the long axis of the coronary sinus is not far from parallel to the trans-

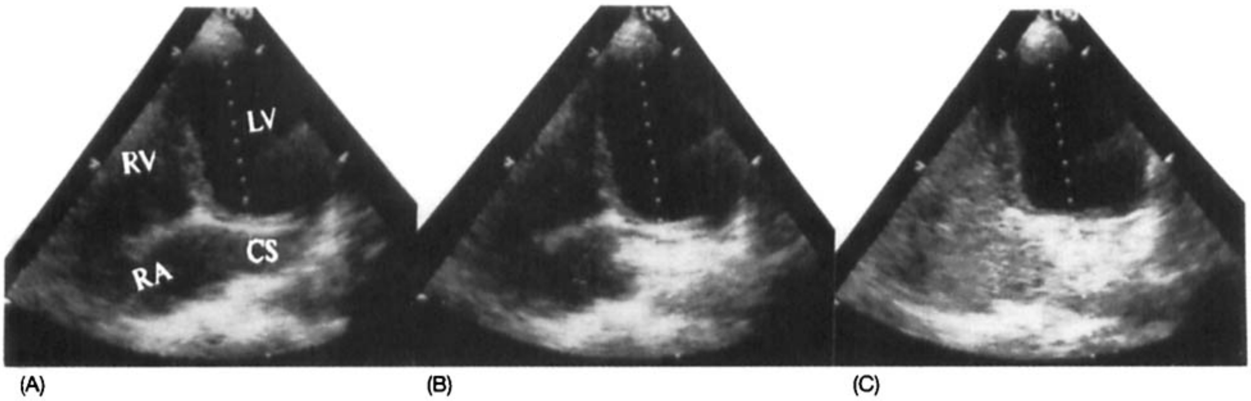

FIG. 6 Apical 4-chamber view showing a very dilated coronary sinus (CS) entering the right atrium (RA). (B) A few seconds later, saline-contrast injected into a left arm vein appears in the dilated CS; at this moment the right atrium and right ventricle (RV) are yet unopacified. (C) Contrast opacifies the right heart chambers. A similar sequence of opacification manifested when saline contrast was injected into a right arm vein.

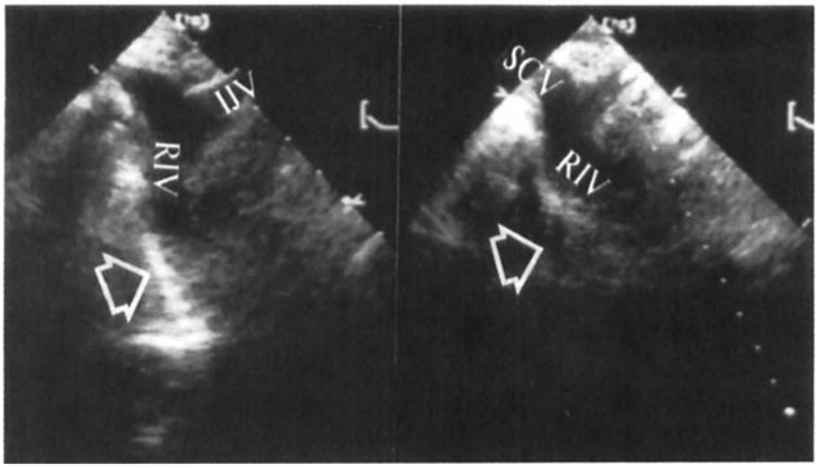

FIG. 7 Visualization of the right innominate vein (RIV), right subclavian vein (SCV), and right internal jugular vein (IJV) from the right supraclavicular window; however, no superior vena cava could be visualized. The arrow indicates where the superior vena cava would normally be seen.

ducer beam, has been successfully used recently to record CS flow.[25, 26] The flow pattern in the CS, like those in the venae cavae and other large veins, consists of a systolic and a diastolic phase.[26] A practical point arises of distinguishing CS from inferior caval inflow in the right atrium, since flow is in the same general direction and the two vessels are close together; the fact that respiratory fluctuations in flow are larger with caval flow than with CS flow makes the distinction possible.[25] By measuring the velocity-time integral of CS flow and estimating the CS cross section in apical 4-chamber view, these authors demonstrated a large (mean 67%) increase in CS flow after coronary bypass surgery in 15 patients with coronary artery disease.

Acknowledgment

The authors thank Jackie Kaiser, RDCS, RDMS, and Regenia Clark, C.C.T, for technical assistance.

References

1. Mantini E, Grondin CW, Lillehei CW, Edwards JE: Congenital anomalies involving the coronary sinus. *Circulation* 1966;33:317
2. Cohen BE, Winer HE, Kronzon I: Echocardiographic findings in patients with left superior vena cava and dilated coronary sinus. *Am J Cardiol* 1979;44:158–161
3. Snider AR, Port JA, Silverman NH: Venous anomalies of the coronary sinus. *Circulation* 1979;60(4):721–727
4. Stewart JA, Franker TD, Slosky DA, Wise NK, Kisslo JA: Detection of persistent left superior vena cava by two-dimensional contrast echocardiography. *J Clin Ultrasound* 1979;7:357–360
5. Orsmond GS, Runttenberg HD, Bessinger FB, Moller JH: Echocardiographic features of total anomalous pulmonary venous connection to the coronary sinus. *Am J Cardiol* 1978;41:597–599
6. Williams R, Bierman F, Sanders S: *Echocardiographic Diagnosis of Cardiac Malformations*, p. 36–50. Boston: Little, Brown, 1986
7. Yeager S, Chin A, Sanders S: Subxiphoid two-dimensional echocardiographic diagnosis of coronary sinus septal defects. *Am J Cardiol* 1984;54:686–687
8. Gray's Anatomy, 38th edition, p. 621. Churchill Livingstone, 1996

9. McAlpine WA: *Heart and Coronary Arteries*, p. 189, 190. New York: Springer-Verlag, 1975
10. Pan-Chih Huang, AH, Dorsey LMA, Guyton RA: Hemodynamic significance of the coronary vein valves. *Ann Thorac Surg* 1994; 57:424–431
11. Coakley JB, King TS: Cardiac muscle relations of the coronary sinus, the oblique vein of the left atrium and the left precaval vein in mammals. *J Anat* 1959; 93:30–35
12. Keith A: The anatomy of the valvular mechanism around the venous orifices. *J Anat* 37: Proceedings II-36
13. Potkin BN, Roberts WC: Size of coronary sinus at necropsy in subjects without heart disease and in patients with various cardiac conditions. *Am J Cardiol* 1987:60:1418–1421
14. Mahmud E, Kerumati S, Raisinghani A, Kwan OL, Blanchard D, DeMaria AN: Dilatation of the coronary sinus on echocardiogram: Prevalence and significance (abstr). *J Am Soc Echocardiogr* 1998; 11:547
15. D'Cruz IA, Johns C, Shala MB: Dynamic cyclic changes in coronary sinus caliber in patients with and without congestive heart failure. *Am J Cardiol* 1999;83:275–277
16. Weyman AE: *Principles and Practice of Echocardiography*, Second Edition, p. 492, 594. Philadelphia: Lea & Febiger, 1994
17. D'Cruz IA: *Echocardiographic Anatomy*, p. 104–107. New York: Appleton & Lange, 1996
18. Winter FS: Persistent left superior vena cava. *Angiology* 1954;5: 90–132
19. Fleming JS, Gibson RV: Absent right superior vena cava as an isolated anomaly. *Br J Radiol* 1964;37:696–697
20. Oguni H, Hatano T, Yamada T, Satomi G, Nakamura K, Imamura E, Takao A: A case of absent right superior vena cava with persistent left superior vena cava: Cross-sectional echocardiographic diagnosis. *Heart Vessels* 1985;1:239–245
21. Konecky N, Freedberg RS, McCauley D, Kronzon I: Absent right and persistent left superior vena cava without other congenital anomaly: A rare combination diagnosed by transesophageal echocardiography. *J Am Soc Echocardiogr* 1995;8:761–766
22. Chaudhry F, Zabalgoitia M: Persistent left superior vena cava diagnosed by contrast transesophageal echocardiography. *Am Heart J* 1991;122:1175–1177
23. Podolsky LA, Jacobs, Schwartz M, Kotler MN, Ioli A: Transesophageal echocardiography in the diagnosis of the peristent left superior vena cava. *J Am Soc Echocardiogr* 1992;5:159–162
24. Roberts WA, Risher WH, Schwartz KQ: Transesophageal echocardiographic identification of persistent left superior vena cava: Retrograde administration of cardioplegia during cardiac surgery. *Anesthesiology* 1994;81:760–762
25. Vlachonassios K, Vaghefi H, Wong I, Nuno P, Chandaratna PAN: Transthoracic echocardiographic demonstration of coronary blood flow after revascularization procedures (abstr). *J Am Coll Cardiol* 1998;31(suppl A):256A
26. Reynolds T, Appleton CP: Doppler flow velocity patterns of the superior vena cava, inferior vena cava, hepatic vein, coronary sinus and atrial septal defect. *J Am Soc Echocardiogr* 1991;4:503–512