Volume 2

Kirklin/Barratt-Boyes

Fourth Edition

Cardiac Surgery

NICHOLAS T. KOUCHOUKOS • EUGENE H. BLACKSTONE
FRANK L. HANLEY • JAMES K. KIRKLIN

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Cardiac Surgery

Morphology, Diagnostic Criteria, Natural History, Techniques, Results, and Indications

Fourth Edition

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Morphology, Diagnostic Criteria, Natural History, Techniques, Results, and Indications

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Preface to Fourth Edition

The fourth edition of *Cardiac Surgery* has been prepared without contributions from two of the authors of the third edition, Drs. Robert B. Karp and Donald B. Doty. Dr. Karp was fatally injured in an automobile accident in 2006. Dr. Doty retired from the practice of cardiothoracic surgery in 2004. We are extremely grateful to both of them for their outstanding contributions, many of which remain in the fourth edition. We are equally pleased to welcome as a contributor to the fourth edition, Dr. James K. Kirklin, son of Dr. John W. Kirklin, co-author with Sir Brian Barratt-Boyes of the first two editions of *Cardiac Surgery*.

Except for Dr. Frank Hanley, we received our cardiothoracic surgical education at the University of Alabama Medical Center under the tutelage of John Kirklin, and we were privileged to serve as faculty members in the Department of Surgery at the University of Alabama at Birmingham School of Medicine during his tenure as chair of the department and director of the Division of Cardiothoracic Surgery. James Kirklin currently serves as director of that division.

We have all, including Dr. Hanley, been profoundly influenced by the teachings of John Kirklin, and by his intellect, vision, and clinical skills. His commitment to improving the quality of cardiac surgery through rigorous clinical and laboratory investigations and providing superb clinical care and disciplined training of young surgeons was truly exemplary. Although our interactions with Sir Brian Barrett-Boyes were less frequent and less intense, he possessed these same attributes and was an inspiration to us as well. In the last year of his life, he was engaged in updating the echocardiographic and structural valve deterioration data of the entire Green Lane Hospital experience of aortic allografts, with the intent of transmitting these data for analysis by one of us (EHB).

The systematic approach to cardiac surgery developed and promulgated by these two pioneering surgeons, who both died between publication of the third and this fourth edition of *Cardiac Surgery*, has been a major fixture in our professional careers. The decision to author the third and now fourth editions of *Cardiac Surgery* was in large part influenced by our desire to perpetuate their philosophical approach to this discipline. Thus, the general format of the three previous editions has been maintained.

All chapters present in the third edition have been revised. They have been rearranged so that every chapter relating to surgical treatment of congenital heart disease (except for Chapter 29, "Congenital Heart Disease in the Adult") has been placed in Volume 2. Each chapter was rewritten with input from at least two of the four authors. Chapter 4 ("Anesthesia for Cardiovascular Surgery") was revised by Drs. Colleen G. Koch and Chandra Ramamoorthy. The content, and in some instances the titles, of several chapters have been altered to reflect current knowledge and practice. As an example, the chapter "Heart Failure" in the third edition has been expanded into three chapters in the fourth edition: "Cardiomyopathy," "Cardiac Transplantation," and "Mechanical Circulatory Support." New illustrations and new echocardiographic, computed tomographic, and magnetic resonance images have been added to reflect important advances in the diagnosis and management of congenital and acquired diseases of the heart and great vessels.

We recognize the potential limitation of four authors writing separate portions of this textbook. This challenge was met, in part at least, by dual authorship of each chapter, and by author meetings and correspondence. It was also met by a process of universal review. Specifically, as with the third edition, Dr. Blackstone was designated as the final arbiter. After completion of the revision of each chapter by the primary author, copyedited material was forwarded to Dr. Blackstone in Cleveland, where he and his assistant, Tess Muharsky Parry, reviewed, edited, reorganized, questioned, and adjudicated the entire content of each chapter. It is our hope that this intensive process has improved the accuracy and comprehensiveness of each chapter.

As in the previous editions, Part I of Volume 1 discusses basic concepts of cardiac surgery: anatomy, support techniques, myocardial management, anesthesia, postoperative care, and methodology for generating new knowledge from previous experience. These core chapters are applicable to the broad audience of medical professionals who care for patients with cardiac disease. The remaining chapters of Volume 1 (Parts II to V) discuss specific acquired diseases of the heart and great vessels, and congenital heart disease in adults. This edition has retained, in these later sections and in all of the chapters in Volume 2, presentation of "Indications for Operation" at the end of each chapter, because the indications are the derivatives of comparison of various outcomes (results) of alternative forms of treatments, including no treatment (natural history).

The abbreviation *UAB* has been retained, and is used to identify data and illustrations from the University of Alabama at Birmingham; similarly, *GLH* identifies those from Green Lane Hospital in Auckland, New Zealand. The bibliographic references are again designated using the first letter of the surname of the first author and a number (e.g., L4), rather than simply a number. This convention is simple and convenient, and allows the reader to easily locate a given author's publication among the alphabetically arranged references. The abbreviation *CL* is used throughout to denote 70% confidence limits around the point estimate. The reasons for presenting 70% rather than 95% or 50% confidence limits are presented in Chapter 6.

The fourth edition is written at a time of great change for the specialty of cardiac surgery. Percutaneous catheter-based interventions are being increasingly used to treat patients with

coronary arteriosclerotic heart disease, aortic valve stenosis, mitral valve regurgitation, hypertrophic obstructive cardiomyopathy, diseases of the thoracic aorta, and congenital cardiac lesions such as patent ductus arteriosus, coarctation of the aorta, atrial and ventricular septal defects, and pulmonary valvar stenosis and regurgitation. Less invasive techniques are rapidly being incorporated into cardiac surgical practice for many conditions that continue to require open surgical repair. These advances must be acknowledged and embraced if cardiac surgery is to thrive in the future.

It is our hope that this textbook will be of value to cardiac surgeons who care for patients with congenital and acquired heart disease and with disorders of major blood vessels in the chest, as well as to cardiologists and interventional cardiologists who treat children and adults with these conditions, anesthesiologists, intensivists, pulmonologists, imaging specialists, cardiovascular nurses, trainees in all of these disciplines, and others.

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— *Frank L. Hanley, MD*
— *James K. Kirklin, MD*

Contents

VOLUME 1

VOLUME 2

PART VII

Congenital Heart Disease

30 Atrial Septal Defect and Partial Anomalous Pulmonary Venous Connection

DEFINITION

An *atrial septal defect* (ASD) is a hole of variable size in the atrial septum. A patent foramen ovale that is functionally closed by overlapping of limbic tissue superiorly and the valve of the fossa ovalis inferiorly (in response to the normal left-to-right atrial pressure gradient) is excluded. ASDs generally permit left-to-right shunting at the atrial level. *Partial anomalous pulmonary venous connection* (PAPVC) is a condition in which some but not all pulmonary veins connect to the right atrium or its tributaries, rather than to the left atrium. The term *connection* is preferred to the term "return," because *connection* is anatomic and return is governed by hemodynamic factors. PAPVCs may occur as isolated anomalies or may be combined with ASDs.

These two groups of anomalies are considered together in this chapter because they manifest similar physiology and result in similar clinical findings. *Total* anomalous pulmonary venous connection is considered in Chapter 31. ASDs typically occur in association with other cardiac anomalies, and these are considered in chapters dealing with those anomalies.

HISTORICAL NOTE

Clinical recognition of an ASD has been possible only in about the past 70 years. Among the 62 recorded autopsy cases of ASD analyzed by Roesler in 1934, only one had been correctly diagnosed during life.[R6] By 1941, Bedford and colleagues were able to make the diagnosis clinically in a number of patients.[B3] When cardiac catheterization came into general use during the late 1940s and early 1950s, secure diagnosis became possible. The first descriptions of PAPVC are attributed to Winslow in 1739[B15] and Wilson in 1798.[M11] The first diagnosis of PAPVC during life was reported by Dotter and colleagues in 1949.[D7]

A number of ingenious closed methods for repair of ASDs and related conditions were proposed and studied experimentally in the productive and expansive surgical era following the end of World War II in 1945. In 1948 in Toronto, Murray reported closing an ASD in a child by external suturing.[M18] Several other closed methods had clinical application, including Bailey and colleagues' "atrioseptopexy" and Søndergard's purse-string suture closure.[B2,S12] However, limited applicability of these methods was always apparent, and they were soon abandoned.

Hypothermia, induced by surface cooling, and inflow occlusion for repair of ASDs was introduced during the early 1950s (see Historical Note in Section I of Chapter 2). Lewis and Taufic reported the first successful open repair of an ASD with this method in 1953.[L5] At about the same time, Gross invented the ingenious atrial well technique, a semi-open approach in which a rubber open-bottomed well or cone was sutured to an incision in a clamp-exteriorized portion of the right atrial wall.[G11,K6] When the clamp was released, the blood rose into the well, and through this pool of blood, the surgeon could place sutures under digital control for direct or patch closure of the defect. Gibbon started the era of open heart surgery in 1953 when he successfully repaired an ASD in a young woman using a pump-oxygenator.[G9] Although these three methods—hypothermia and inflow occlusion, atrial well, and cardiopulmonary bypass (CPB)—were all used during the late 1950s and provided similar early results,[K8] by the late 1960s almost all surgeons used CPB exclusively for these repairs. Percutaneous catheter techniques for closing a fossa ovalis ASD using a polyester double umbrella device were introduced by King and Mills in 1974.[K4]

The first reported treatment for a type of PAPVC was lobectomy in 1950.[D9] In 1953, Neptune and colleagues reported repair using a closed technique in 17 patients with PAPVC of the right lung to the right atrium associated with ASD.[N5] It is not certain who first repaired the sinus venosus syndrome, but the malformation was clearly illustrated by Bedford and colleagues in 1957.[B4] Repair of PAPVC to the inferior vena cava was performed by Kirklin and colleagues at Mayo Clinic in 1960 and was also subsequently reported by Zubiate and Kay in 1962.[M17,Z2] Correction of anomalous connection of the left pulmonary veins to the left brachiocephalic vein and other forms of PAPVC was reported from the Mayo Clinic in 1953[G6,K5] and later in 1956.[K7]

MORPHOLOGY

Types of Atrial Septal Defect

As viewed from the right atrial side (see Fig. 1-2 in Chapter 1), the normal atrial septum may have defects in almost any location (Box 30-1). Although the morphology of these defects has been known since the early descriptions by Robitansky in 1875,[R5] the advent of open heart surgery emphasized their surgically important aspects[B4,L6] (Fig. 30-1).

Fossa Ovalis Defect

The most common ASD is the *fossa ovalis type*, also called *foramen ovale type* or *ostium secundum defect*. This defect lies within the perimeter inscribed by the limbus anteriorly, superiorly, and posteriorly (Fig. 30-2). The smallest defects are

Box 30-1 Types of Atrial Septal Defect[a]

- Fossa ovalis defect[b]
- Posterior defect
- Coronary sinus defect
- Sinus venosus defect
- Confluent defect
- Ostium primum defect (absence of atrioventricular septum)

[a]Fossa ovalis, posterior, and most confluent defects can be classified as *secundum type.*
[b]Varies in size from small valvar-incompetent foramen ovale ASD to complete absence of septum primum tissue with resultant ASD extending to inferior vena cava.

essentially valvar incompetent foramina ovale that occur beneath the superior limbus, between it and the valve (floor) of the fossa ovalis. The floor of the fossa ovalis (remnant of septum primum) may in this situation have multiple fenestrations of various sizes (Fig. 30-3). When more of the floor of the fossa ovalis is absent, a larger fossa ovalis defect is present. When all fossa ovalis tissue is absent, the ASD is confluent with the orifice of the inferior vena cava (IVC). The eustachian valve of the IVC then overhangs the ASD and must not be mistaken for its inferior edge at operation. Size of this type of ASD is also affected by any hypoplasia of the limbus that may be present. When the limbus is quite hypoplastic anteriorly, there is only a thin rim of tissue above the atrioventricular (AV) valves (formerly this was called an *intermediate defect* and was sometimes confused with an ostium primum defect). The limbus may also be hypoplastic superiorly or posteriorly.

Normally the IVC–right atrial junction is partly to the left of the plane of the limbus, so that when the floor of the fossa ovalis is absent and an ASD of fossa ovalis type extends to the IVC, the caval ostium overrides (or straddles) the defect onto the left atrium.[V1] This defect results in some right-to-left shunting of IVC blood to the left atrium in virtually all patients with a large fossa ovalis–type ASD (as documented in experimental studies[K9,M5,S10]) and severe shunting with cyanosis in a few patients.[W5] Also, the position of the normally connected right pulmonary veins next to the atrial septal remnant results in preferential left-to-right shunting of their venous drainage.[B12,S10]

Posterior Defect

A defect in the most posterior and inferior part of the atrial septum, with absence, hypoplasia, or anterior displacement of the posterior limbus, is termed a *posterior ASD.* The orifices of the right pulmonary veins usually open directly into the area of the defect, but true anomalous pulmonary venous connection of the right lung frequently coexists. In the pure form of this type of ASD, the tissue of the fossa ovalis (including the posterior limbus) is present, and the ASD is an oval defect posterior to this tissue (Fig. 30-4).

Sinus Venosus Defect

The ASD that occurs in sinus venosus syndrome (subcaval defect, superior vena caval defect) is located immediately beneath the orifice of the superior vena cava (SVC), superior to the limbic tissue, and is usually associated with anomalous pulmonary venous connection of the right superior pulmonary vein to the SVC near or at the SVC–right atrial junction. The lower margin of the defect is a sharply defined crescentic edge of atrial septum, whereas its upper margin is devoid of septum, being continuous with the posterior SVC wall, which in turn is continuous with the upper edge of the left atrium. The SVC usually overrides the atrial septum onto the left atrium to some extent (see "Sinus Venosus Malformation [Syndrome]" later in this chapter).

Coronary Sinus Defect

Coronary sinus ASDs are part of *unroofed coronary sinus syndrome.* When the sinus is completely unroofed and no partition is present to separate it from the left atrium, the ostium of the coronary sinus is a hole in the atrial septum that permits free communication between left and right atria (see Chapter 33). Occasionally a fenestration may exist in this partition in the midportion of the coronary sinus, particularly in hearts with tricuspid atresia, or rarely the fenestration may be almost at the ostium of the coronary sinus[R9] (Fig. 30-5).

Confluent Defect

Large ASDs may represent a confluence of two of the defects already described. Thus, a fossa ovalis defect coexisting with absence of the posterior limbus can present as a very large ASD with no septal remnant posteriorly. Another confluent defect occasionally seen is a combination of coronary sinus and fossa ovalis ASDs.

Ostium Primum Defect

An ASD occurs anterior to the fossa ovalis (and the anterior limbus) when the AV septum is absent. Such defects are called *AV septal defects, AV canal defects,* or *ostium primum atrial septal defects* and are considered in Chapter 34. When essentially the entire atrial septum is absent (common atrium), the defect includes absence of the AV septum (see "Atrial Septal Deficiency and Interatrial Communications" under Morphology in Chapter 34).

Types of Partial Anomalous Pulmonary Venous Connection

Sinus Venosus Malformation

The most common type of PAPVC is the defect present in sinus venosus malformation, in which PAPVC coexists with a superior caval ASD. In sinus venosus malformation, the right upper and middle lobe pulmonary veins (right superior pulmonary vein) attach to the low SVC or the SVC–right atrial junction, an arrangement present in about 95% of patients with a superior caval ASD.[C6,L4,S20] Most often, the anomalous pulmonary venous connection is through two anomalous veins from upper and middle lobes, one superior to the other, but there may be three or rarely four veins, with the uppermost entering the SVC near the azygos vein entry. Infrequently, only part of the right superior vein connects anomalously, with the inferior (right middle lobe) portion of that vein connecting to the left atrium. Rarely, both the right superior and right inferior pulmonary veins connect anomalously to the low SVC or SVC–right atrial junction (Fig. 30-6).

The lowermost part of the SVC that receives the anomalous veins is usually wider than normal, although it may be relatively small, particularly when there is also a well-formed left SVC, which is not uncommon.[T6] The SVC typically overrides the atrial septum to some extent and enters partly into

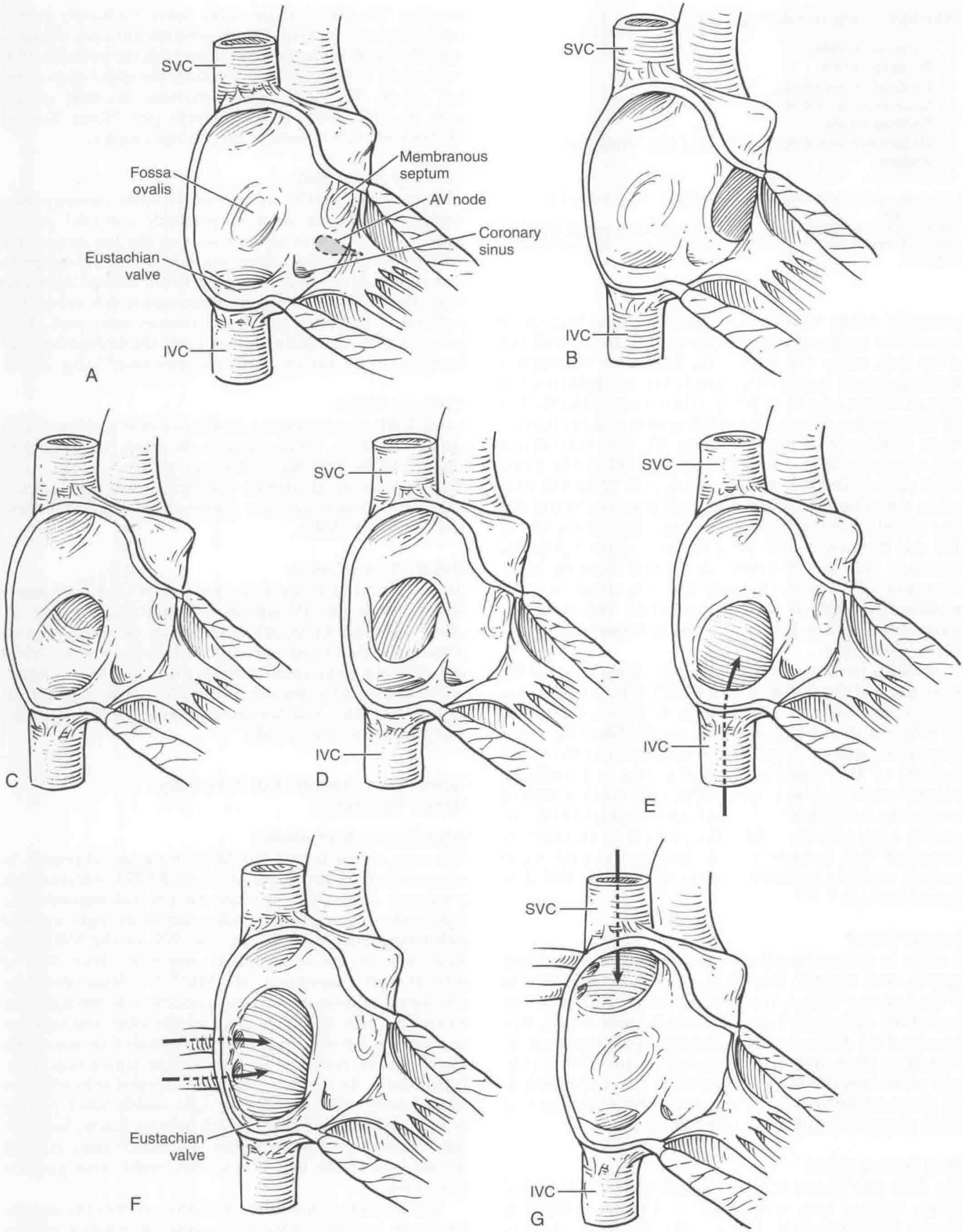

Figure 30-1 Anatomy of atrial septal defect (ASD), viewed from right atrium. **A,** Normal atrial septum. **B,** Atrioventricular (primum) type of ASD. **C,** Widely patent foramen ovale. **D,** Fossa ovalis defect with complete septal rim (secundum). **E,** Low fossa ovalis ASD astride inferior caval orifice with large eustachian valve. **F,** Large fossa ovalis ASD without posterior rim, with pseudoanomalous right pulmonary veins. **G,** Sinus venosus defect. Key: *AV,* Atrioventricular; *IVC,* inferior vena cava; *SVC,* superior vena cava.

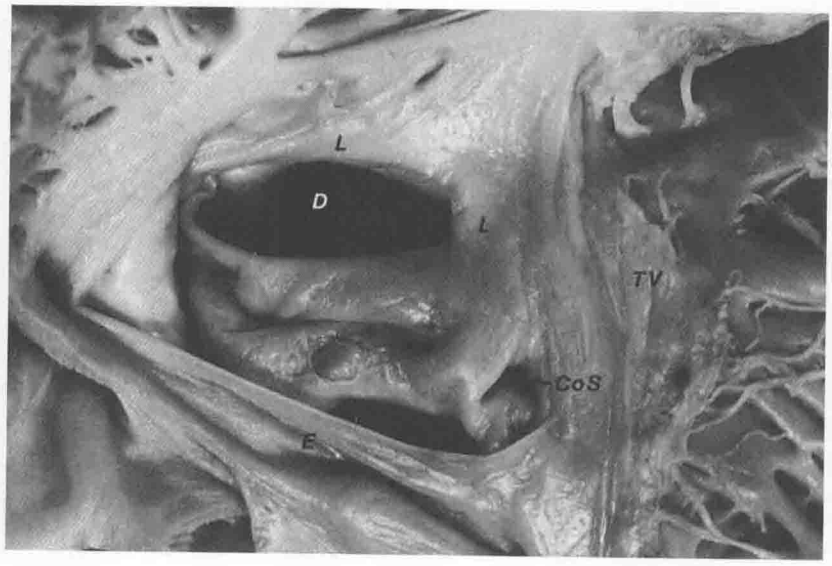

Figure 30-2 Specimen with fossa ovalis atrial septal defect, viewed in anatomic orientation with superior vena cava above and inferior vena cava and its eustachian valve below. Limbus forms anterior, superior, and posterior rim of defect, and remnants of the floor (valve) of fossa ovalis form inferior rim. Key: *CoS,* Coronary sinus; *D,* atrial septal defect; *E,* eustachian valve; *L,* limbus; *TV,* septal leaflet of tricuspid valve.

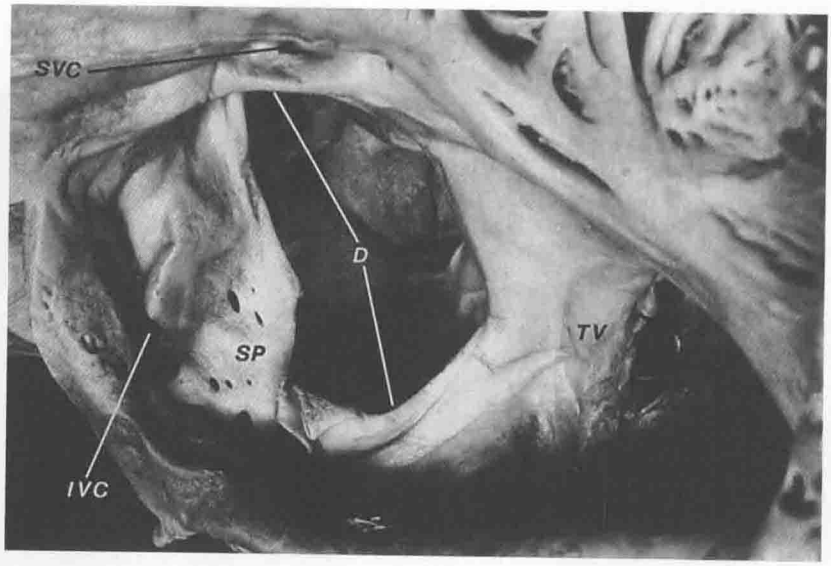

Figure 30-3 Specimen with large fossa ovalis atrial septal defect viewed from opened right atrium in same orientation as Fig. 30-2. Thin remnant of septum primum (floor of fossa ovalis) shows numerous perforations. Key: *D,* Atrial septal defect; *IVC,* inferior vena cava; *SP,* septum primum; *SVC,* superior vena cava; *TV,* tricuspid valve.

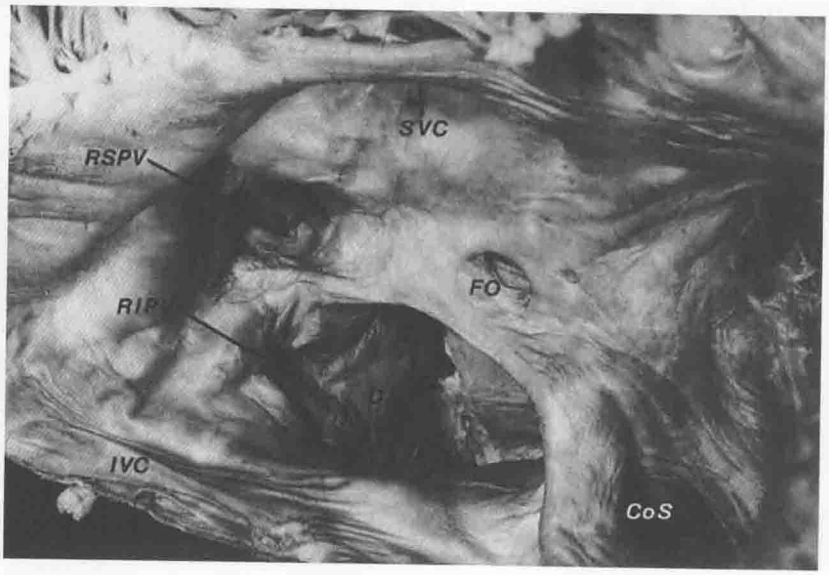

Figure 30-4 Specimen with large posterior atrial septal defect, viewed from opened right atrium. Orientation is as in Fig. 30-2. Fossa ovalis is intact, but there is a patent foramen ovale. Right inferior pulmonary vein certainly drains anomalously, but is probably normally connected. Right superior pulmonary vein is anomalously connected to right atrium. Key: *CoS,* Coronary sinus; *D,* atrial septal defect; *FO,* fossa ovalis; *IVC,* inferior vena cava; *RIPV,* right inferior pulmonary vein; *RSPV,* right superior pulmonary vein; *SVC,* superior vena cava.

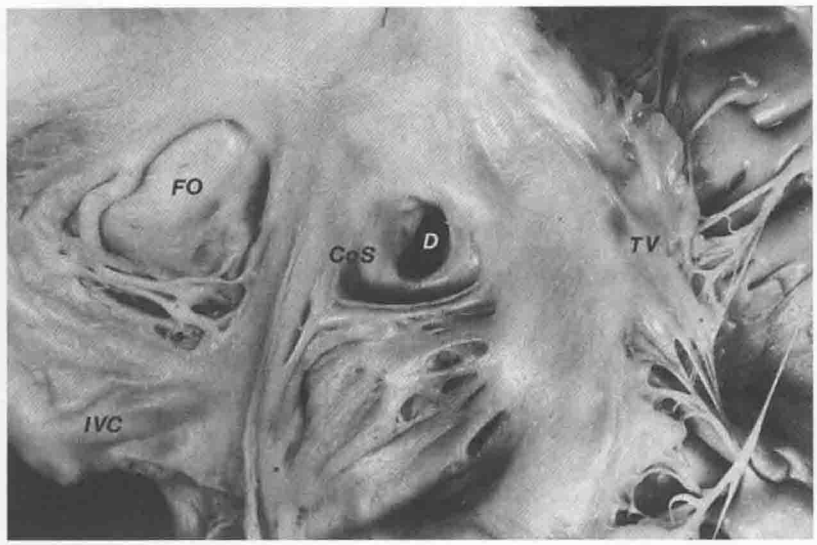

Figure 30-5 Unusual example of small coronary sinus atrial septal defect near ostium. Other anomalies include patent foramen ovale, ventricular septal defect, mild aortic regurgitation, and possible mitral regurgitation. Key: *CoS,* Coronary sinus; *D,* atrial septal defect; *FO,* fossa ovalis; *IVC,* inferior vena cava; *TV,* tricuspid valve.

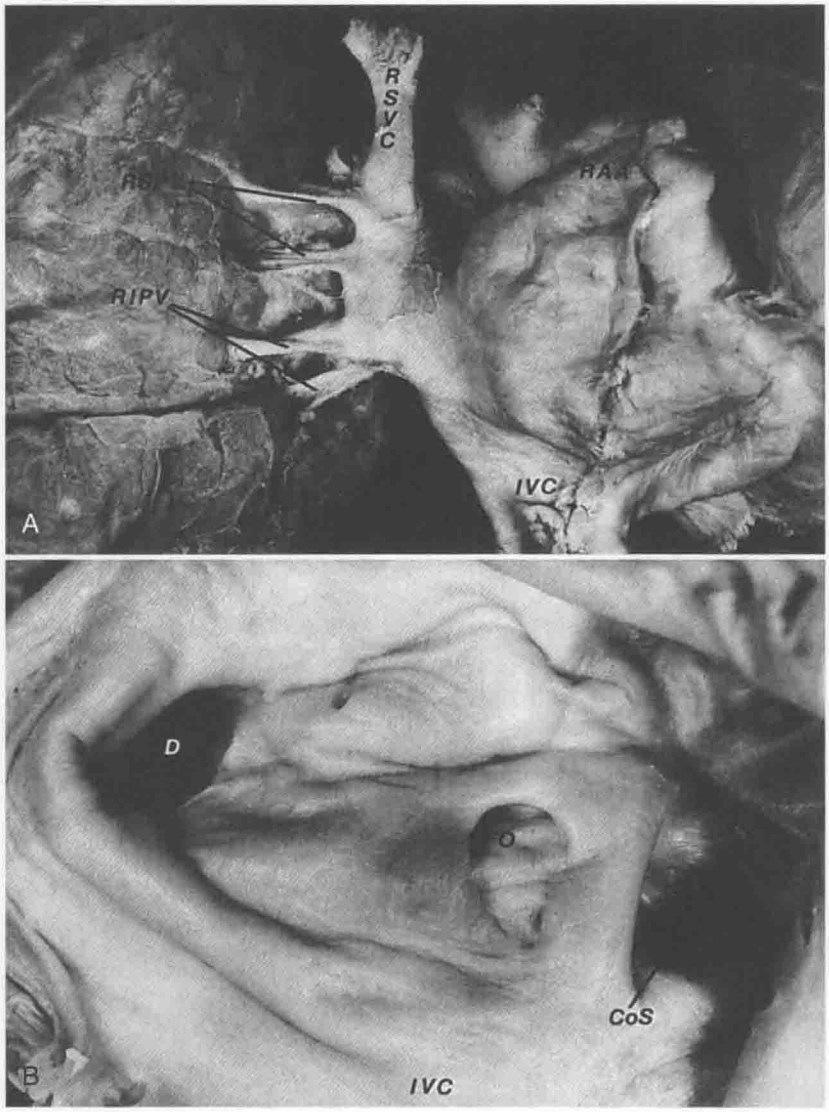

Figure 30-6 Unusual example of sinus venosus malformation. **A,** Specimen with typical subcaval atrial septal defect (ASD), but with both right superior and right inferior pulmonary veins entering superior vena caval–right atrial junction. In addition, the left pulmonary veins form a common channel connected to left atrium and right superior vena cava. Left superior vena cava and mitral atresia were also present. **B,** Interior of right atrium showing the subcaval ASD high in the septum and enlarged coronary sinus ostium to which is connected the left superior vena cava. Key: *CoS,* Coronary sinus; *D,* atrial septal defect; *FO,* fossa ovalis; *IVC,* inferior vena cava; *RAA,* right atrial appendage; *RIPV,* right inferior pulmonary vein; *RSPV,* right superior pulmonary vein; *RSVC,* right superior vena cava.

the left atrium, resulting in a right-to-left shunt of some SVC blood to the left atrium. In a few patients, SVC overriding is severe enough to produce a large right-to-left shunt and marked cyanosis.[S6] The overriding may also be complete, so that the SVC drains directly and completely into the left atrium.[B8,P2]

The relationship between anomalous connection of the SVC to the left atrium without an ASD and sinus venosus ASD is indicated by connection of pulmonary veins from the right upper lobe to the cardiac end of the SVC in some patients with PAPVC.[B14,K9,P2] This relationship also occurs in patients with no ASD but in whom the pulmonary veins from the right upper lobe are connected to the cardiac end of the SVC, with the SVC connected to the left atrium by a large opening, and to the right atrium by a small opening.[B8,S6]

Rarely, a typical high superior caval ASD is present without anomalous pulmonary venous connection; right pulmonary veins connect to the left atrium but more superiorly than normal.

Right Superior Pulmonary Vein to Superior Vena Cava

Occasionally the entire right superior pulmonary vein connects to the SVC without an associated superior caval ASD. The connection is then usually well above (superior to) the SVC–right atrial junction, and the lower part of the SVC is not dilated. Rarely, even when no superior caval ASD is present, the connection may be in the typical low position of sinus venosus syndrome. At times, only a *portion* of the right superior pulmonary vein draining one or two segments of the right upper lobe connects directly to the SVC. The PAPVC may be isolated or associated with a fossa ovalis ASD.

Right Pulmonary Veins to Right Atrium

Right pulmonary veins may connect directly to the right atrium, either in toto, where they may connect as two or three separate veins, or only through the superior (or rarely inferior) right pulmonary vein. This anomaly may exist as an isolated defect, without an ASD or with only a patent foramen ovale, with the plane of the atrial septum altered from coronal to near-sagittal because of leftward displacement of its lateral attachment. The plane of the right pulmonary vein is actually altered minimally from normal. Because the posterior limbus is present in such defects, the veins are clearly anomalously connected to the right atrium. In ASDs with absence of posterior limbus (posterior ASD), and at times in large fossa ovalis ASDs, the plane of division between right and left atria posteriorly can be questionable, and thus the atrial connection of the right pulmonary veins in this area is debatable. In such defects, however, true anomalous connection of the right pulmonary veins may be present[M6,S19] (see Fig. 30-4).

Right Pulmonary Veins to Inferior Vena Cava (Scimitar Syndrome)

An anomalous right pulmonary vein, generally draining the entire right lung but occasionally only the middle and lower lobes, may descend in a cephalad-to-caudad direction toward the diaphragm, more or less parallel to the pericardial border but with a crescentic (scimitar) shape, and then curve sharply to the left just above or below the IVC–right atrial junction.[K3] The anomalous pulmonary venous trunk usually passes anterior to the hilum of the right lung but occasionally is posterior to it. Entrance into the IVC is just superior to the hepatic vein orifices. The atrial septum may be intact, or a fossa ovalis ASD may be present. Occasionally the anomalous vein also connects to *left* atrium,[G5,M11] and rarely scimitar syndrome can exist with connection of the anomalous vein *only* to left atrium.[M13] Pulmonary venous *drainage* is then normal. (Rarely, the left lung may connect via a scimitar-shaped vein to the IVC.[D1,M2])

Right-sided scimitar syndrome occurs as an isolated malformation in a minority of cases. In most patients, anomalies of the right lung are also present.[N1] The most common anomaly is *right lung hypoplasia*, which is associated with a marked mediastinal shift and dextroposition of the heart, and in its severe form with the entire heart lying in the right side of the chest. Blood supply to the hypoplastic right lung comes mainly from a branch of the abdominal aorta in the region of the celiac axis, which ascends through the inferior pulmonary ligament to supply the lower lobe, or more often the entire right lung. A small pulmonary artery may be present, but often the central and hilar portions of the right pulmonary artery are absent. Occasionally a true right lower lobe bronchopulmonary sequestration may exist, with secondary intrapulmonary cyst formation.

Associated cardiac anomalies are often present in scimitar syndrome. In one study, for example, 11 of 13 infants had associated malformations, seven of whom had left-sided hypoplastic conditions.[G2] Diaphragmatic anomalies occurred in about 20% of the cases reviewed by Kiely and colleagues.[K3] These defects included herniation of the right lung through the foramen of Bochdalek and abnormal attachments of the diaphragm.

Rare Connections of Right Pulmonary Veins

Rarely, right pulmonary veins connect anomalously to the azygos vein or coronary sinus, with or without a fossa ovalis ASD.

Left Pulmonary Venous Connections

Left pulmonary veins may connect to the left brachiocephalic vein by way of an anomalous vertical vein.[S11] Anomalous drainage is usually from the entire left lung, but may be only from the left upper lobe.[B15] A fossa ovalis ASD coexists in some patients, and in others the atrial septum is intact.[H7] Rarely, left pulmonary veins connect anomalously to the coronary sinus, a right-sided SVC, or the right atrium.

Bilateral Partial Pulmonary Venous Connection

Partial but bilateral anomalous pulmonary venous connection is rare. The most common variant is probably the defect in which the atrial septum is intact, the left superior pulmonary vein attaches to the left brachiocephalic vein by way of an anomalous vertical vein, and the right superior pulmonary vein attaches to the SVC–right atrial junction. In another form, a common pulmonary venous chamber is present (see "Pulmonary Venous Anatomy" under Morphology in Chapter 31 for definition), and some veins from both lungs connect to it. All but one lobe or only one lobe from each side may connect to the sinus. The common venous sinus may connect to the right atrium or brachiocephalic vein.

Cardiac Chambers in Atrial Septal Defect and Related Conditions

Typically in ASD and related conditions, the right atrium is greatly enlarged (at least grade 3 or 4 on a scale of 1 to 6)