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Editor

Aortic Stenosis

Case-Based Diagnosis and Therapy

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Editor
Amr E. Abbas, MD, FACC, FSCAI, FSVM, FASE, RPVI
Interventional Cardiology Research
Beaumont Health System
Royal Oak
Michigan
USA

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This book is dedicated to my parents who I owe everything to and then more, my wife who I love dearly, my children who are my life and then some, and my co-authors who without them, this book would not be possible.

Preface

Ever since the earliest description of aortic stenosis by Riverius in 1646, aortic stenosis has become known as a common cause of morbidity and mortality. However, it was not until the twentieth century that the management of these patients included diagnosis via echocardiography, CTA and MRI, cardiac catheterization, and treatment via valvulplasty and surgical aortic valve replacement. Moreover, during the earliest part of the twenty-first century, transcatheter approaches have been described providing options for patients who were previously deemed as nonsurgical candidates.

This book is designed to provide a case-based overview of aortic stenosis including pathophysiology, presentation, diagnosis with both invasive and multimodality noninvasive techniques, and the approach to management options in the multidisciplinary setting. This book will provide an assessment of cases that appear to be complex in terms of determining the true severity of aortic stenosis as patients with low flow, higher gradients with nonsevere valve areas, as well as patients with prosthetic valves. In addition, it will provide a review of current available treatment options such as valvuloplasty, transcatheter, and surgical valve replacement techniques.

We believe this book is essential for individuals in the structural heart disease world including cardiac surgeons, interventional and imaging cardiologists, as well as cardiology fellows who are interested, or in or involved in the management of patients with aortic stenosis. Imaging and interventional cardiologists, cardiac surgeons, and scientists, who are well renowned on the national and international level in managing patients with aortic stenosis, have all been involved in this book and to those individuals we are indebted for their time and expertise.

Royal Oak, MI, USA

Amr E. Abbas, MD, FACC, FSCAI, FSVM, FASE, RPVI

Contents

Contributors

Amr E. Abbas, MD, FACC, FSCAI, FSVM, FASE, RPVI Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Aaron David Berman, MD, FACC Department of Cardiology, William Beaumont Hospital, Royal Oak, MI, USA

A. Neil Bilolikar, MD Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Laura M. Franey, MD Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Michael J. Gallagher, MD, FACC Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

James A. Goldstein, MD Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Ivan Hanson, MD Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

George S. Hanzel, MD Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Robert L. Johnson, PA-C, BS-Medicine Department of Cardiovascular Surgery, Beaumont Health, Royal Oak, MI, USA

Nathan Kerner, MD, FACC, FASE Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Steven J. Lester, MD Department of Medicine, Mayo Clinic, Scottsdale, AZ, USA

Michael J. Mack, MD Department of Cardiovascular Disease, Baylor Scott & White Health, Plano, TX, USA

Philippe Pibarot, DVM, PhD, FAHA, FACC, FESC, FASE Department of Research, Quebec Heart and Lung Institute, Quebec City, QC, Canada

Mark C. Pica, BS, CCRP Department of Cardiology/Research Institute, Beaumont Health System, Royal Oak, MI, USA

Karl K.C. Poon, MBBS Cardiology Program, The Prince Charles Hospital, Chermside, QLD, Australia

Gilbert L. Raff, MD, FACC, FSCCT Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Marc P. Sakwa Department of Cardiovascular Surgery, Beaumont Health, Oakland University School of Medicine, Royal Oak, MI, USA

Francis L. Shannon, MD Department of Cardiovascular Surgery, Beaumont Health, Oakland University School of Medicine, Royal Oak, MI, USA

Frances O. Wood, MD Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

Sibin K. Zacharias, MD Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA

General Considerations and Etiologies of Aortic Stenosis

1

Frances O. Wood and Amr E. Abbas

Abstract

The ***earliest*** descriptions of aortic stenosis are credited to Riverius in 1646 where he provided a clear-cut description of the observed pathological findings of calcified aortic valve cusps in association with weak and diminished peripheral pulses. Aortic stenosis was described again by Bonet in 1679, however, John Baptist Morgagni, professor of anatomy in the University of Padua, referred to aortic stenosis in 1761 and is credited in providing a brilliant description of an autopsy specimen of calcified aortic valve cusps found in a patient and suggested the valve was both stenotic and incompetent. In his description, he quoted a similar case described by Georgius Greiselius and he clarified the anatomical and pathophysiological features of acquired aortic stenosis. In 1806, Corvisart provided another impressive correlation of clinical and autopsy findings and in 1854, William Stokes provided yet another vivid description of the disease. This chapter will provide a general overview of aortic stenosis as well as a review of the common etiologies of aortic stenosis.

Keywords

Aortic stenosis • Etiology of aortic stenosis • Left ventricular outflow tract (LVOT) • Valvular aortic stenosis • Supra-valvular aortic stenosis • Sub-valvular aortic stenosis • Epidemiology of aortic stenosis • Causes of aortic stenosis

Historical Perspective

The ***earliest*** descriptions of aortic stenosis are credited to Riverius in 1646 where he provided a clear-cut description of the observed pathological findings of calcified aortic valve cusps in association with weak and diminished peripheral pulses.

F.O. Wood, MD • A.E. Abbas, MD, FACC, FSCAI, FSVM, FASE, RPVI (✉)
Department of Cardiovascular Medicine, Beaumont Health, Oakland University/William Beaumont School of Medicine, Royal Oak, MI, USA
e-mail: aabbas@beaumont.edu

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Aortic stenosis was described again by Bonet in 1679, however, John Baptist Morgagni, professor of anatomy in the University of Padua, referred to aortic stenosis in 1761 and is credited in providing a brilliant description of an autopsy specimen of calcified aortic valve cusps found in a patient and suggested the valve was both stenotic and incompetent. In his description, he quoted a similar case described by Georgius Greiselius and he clarified the anatomical and pathophysiological features of acquired aortic stenosis. In 1806, Corvisart provided another impressive correlation of clinical and autopsy findings and in 1854, William Stokes provided yet another vivid description of the disease [1–3].

Diagnosis of aortic stenosis has undergone several developments throughout history. Throughout the nineteenth century, physicians could identify the murmur even with the use of primitive stethoscopes. Hemodynamic assessment of aortic stenosis was initially limited due to the inherent belief that retrograde catheterization through a stenotic aortic valve was contraindicated. As such, transbronchial arteriotomy, transthoracic left ventricular puncture, and transseptal approaches were developed to assess the left ventricular pressure. In congruence with the realization of the feasibility of retrograde catheterization in these patients, Gorlin and Gorlin developed the formula to measure the aortic valve area in 1951. It wasn't until 1981 when non-invasive Doppler techniques were developed to measure gradient and valve area.

Management of aortic valve stenosis included early attempts of dilatation, concurrently; implantation of a ball-valve prosthesis in the descending aorta for aortic regurgitation was performed by Hugffnagel in 1952. In the following decade, and with the development of cardiopulmonary bypass by Gibbon in 1953, Harken et al. reported the first successful aortic valve replacement with a mechanical prosthesis in 1960. In 1962, Ross reported the use of an aortic valve homograft in the orthotropic position and in 1967, he reported the transfer of the pulmonic valve to the aortic position [4]. Finally, in the early part of this century, Crebier et al. described the percutaneous implantation of an aortic valve in a human subject. The rest, as they would say, is history.

This book will provide an overview of all aspects of aortic stenosis in a case-based format including anatomical, clinical, diagnostic, and therapeutic considerations.

Introduction

Obstruction of the blood flow from the left ventricular outflow tract (LVOT) may occur at various levels including that ***at*** the aortic valve level (valvular aortic stenosis), ***above*** (supra-valvular aortic stenosis), or ***beneath*** the semilunar valve (sub-valvular aortic stenosis). However, the clinical presentation may be similar with either shortness of breath, syncope, and/or chest pain. Patients may present with a systolic ejection murmur that may be constant or vary with certain maneuvers (as in the presence of hypertrophic obstructive cardiomyopathy) as well as with a variable intensity of the second heart sound depending on the severity of obstruction.

The diagnosis of the site and severity of aortic stenosis depends on the anatomical assessment via echocardiography (echo), cardiac computed tomography (CT), and cardiac magnetic resonance imaging (MRI) as well as the physiological assessment of area reduction and trans-valve gradient by cardiac catheterization, Doppler echocardiography, and more recently cardiac MRI.

Treatment of the various forms of severe aortic stenosis (AS) has been traditionally a surgical endeavor. However, with the advent of transcatheter aortic valve replacement (TAVR), alcohol septal ablation (ASA) for hypertrophic obstructive cardiomyopathy (HOCM), and balloon valvuloplasty of congenital aortic valve stenosis and sub-aortic membranes, interventional cardiology has gained an increasing role in management of these conditions.

This chapter will serve to provide an overview of the anatomy of the aortic valve (AV) as well as the epidemiology, etiology and general considerations regarding aortic stenosis.

Prior to discussing aortic stenosis, it will be essential to review the complex anatomy of the aortic root.

The Aortic Valve and Root Apparatus

The ***aortic root*** is an extension of the LVOT that involves the ventricular septum, aortic wall, sinuses of Valsalva formed by the three semi-lunar leaflets, fibrous continuity to the mitral valve, coronary arteries and the left bundle branch. The aortic root extends from the basal attachments of the semi-lunar valvular leaflets within the left ventricle to the sinutubular junction. The three valvular sinuses and their respective leaflets form the right, left, and non-coronary (or posterior) sinuses (Figs. 1.1 and 1.2). Normally, the left coronary

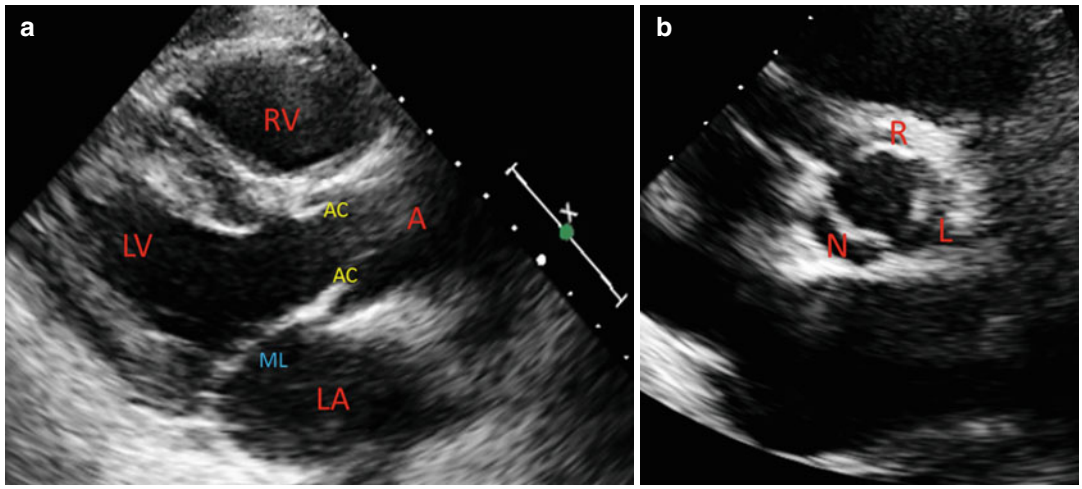

Fig. 1.1 Parasternal long (*left*, **a**) and short (*right*, **b**) axis transthoracic echocardiographic view of a normal aortic valve in systole. In the parasternal long axis, the leaflet closest to the right ventricle is the right leaflet while the leaflet closest to the mitral valve is either the left or non-coronary cusp depending on the angle. In the short axis view, the interatrial septum points to the non-leaflet and the right is closest to the right ventricle *RV* right ventricle, *LV* left ventricle, *AC* aortic valve cusp, *A* aorta, *ML* mitral valve leaflets, *LA* left atrium, *N* non-coronary cusp, *L* left coronary cusp, *R* right coronary cusp

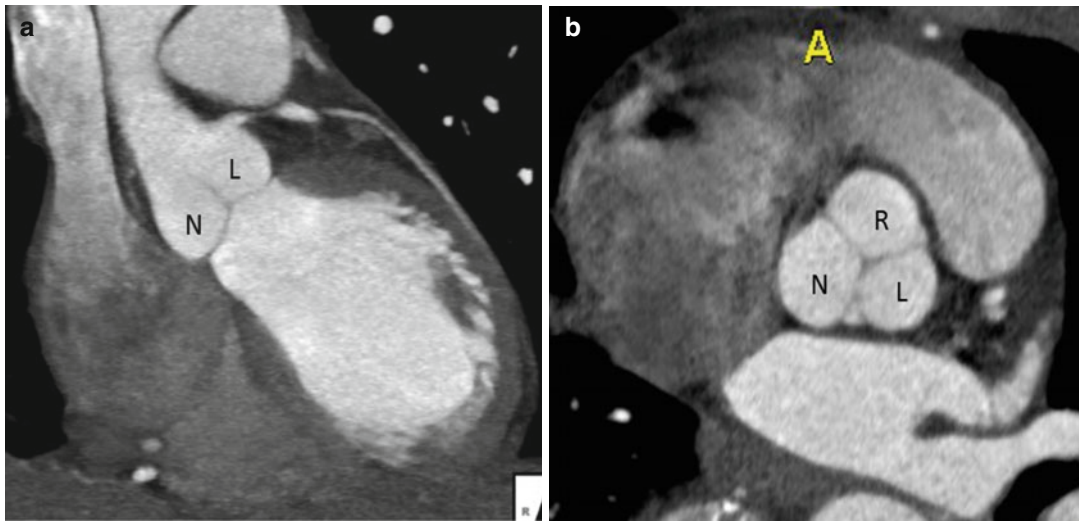

Fig. 1.2 Long (*left*, **a**) and short (*right*, **b**) axis cardiac CT angiographic view of the aortic valve in diastole. *N* non-coronary cusp, *L* left coronary cusp, *R* right coronary cusp

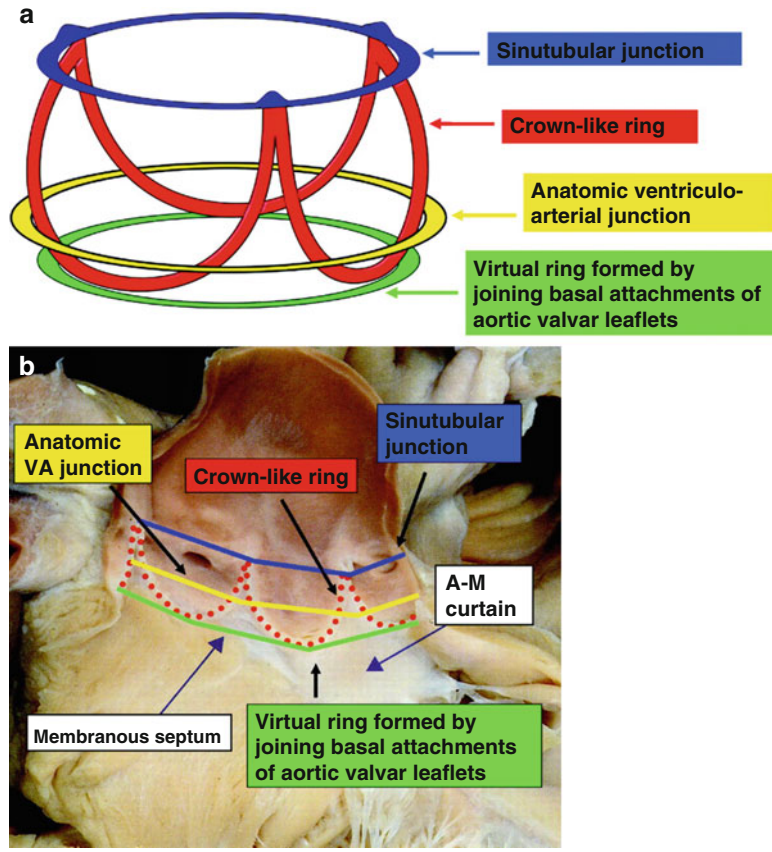

Fig. 1.3 (**a**, *top*). Anatomical specimen of the aortic root with leaflets removed showing location of three virtual rings relative to the crown-like hinges of the leaflets (From Piazza et al. [5] with permission). (**b**, *bottom*) reveals a diagram representing the three circular anatomic rings of aortic root (Modified from Piazza et al. [5] with permission)

artery arises from the left coronary sinus while the right coronary artery arises from the right coronary sinus. The left bundle branch courses through the right and non-coronary sinuses.

The nomenclature of the *aortic valve apparatus* includes three rings: basal, ventriculo-aortic junction, and sinotubular junction (Fig. 1.3a, b) [5].

(A) The ***basal ring*** comprises of the bottom of the sinuses formed by the semi-lunar leaflets and membranous septum.

(B) The ***ventriculo-aortic junction*** is an anatomic ring where the membranous septum connects to the aortic wall at the bases of the right and left coronary sinuses while the aortic wall connects to the fibrous continuity of the anterior leaflet of the mitral valve at the base of the non-coronary sinus. The interleaftlet trigones between the semilunar leaflets and the membranous ventricular attachment are made of fibrous tissue.

(C) The ***ring of the sinotubular junction*** is formed by the attachment of the sinuses to the ascending aorta.

Prevalance and Epidemiology of Aortic Stenosis

The Euro Heart study on valvular heart disease revealed that aortic stenosis was the most common valve disease in a population of 4,910 patients greater than 65 years of age (43.1 % of patients) and degenerative pathology accounted for almost 82 % of the cases [5].

In the US study of 1,797 patients older than 60 years, aortic stenosis was the second most common disease after mitral regurgitation. There appears to be a trend towards a higher prevalance of AS in men which becomes significant after adjusting for age [6]. Osnabrugge et al. pooled

data from seven studies of elderly (≥75 years) patients with severe aortic stenosis to determine the prevalence of aortic stenosis in Europe and North America and to estimate the potential surgical and transcatheter procedures [7]. The prevalence of mild to severe aortic stenosis was 2.4 % (2.7 million North Americans, 4.9 million Europeans) while severe aortic stenosis was 3.4 %. Three quarters of the patients with severe AS were symptomatic which corresponds to 540,000 North Americans and one million Europeans. The prevelance of AS, expectedly, increases with age and it is four times more common over the age of 65 (1.3 % vs 0.32 %) [6, 7].

In a survey of patients with severe AS at a single center, only half of patients with AS underwent AVR, 75 % of which were symptomatic despite a predicted mortality of <10 %, and fewer than one third were even referred to a surgeon [8].

Causes of Aortic Stenosis

As mentioned above, AS may occur at the level of, beneath, or above the level of the AV. The most common cause is ***valvular*** AS and its main causes are congenital, calcific, and rheumatic. ***Para-valvular*** obstruction (supra, and sub valvular aortic stenosis) can occur through membranes, muscular hypertrophy, or iatrogenically following surgical procedures.

Valvular Aortic Stenosis

Valvular aortic stenosis is by far the most common form of aortic stenosis and rheumatic heart disease remains the most common cause of valvular aortic stenosis worldwide especially in developing nations [9].

Calcific aortic stenosis is the most common form of valvular aortic stenosis in industrialized countries. It is primarily a disease of the elderly with increasing in prevalence with age. Superimposed calcification of congenital aortic stenosis is the second most common form of aortic stenosis in industrialized nations and commonly presents after the age of 50. Half of the adults with aortic stenosis have underlying bicuspid stenosis [10] and it is the most common cause of aortic stenosis before the age of 65. Other uncommon forms of aortic stenosis in the industrialized world is radiation and drug-induced aortic valve disease. Childhood aortic stenosis from either homozygous type II hyperlipoproteinemia, ochronosis with alkaptonuria, and Paget's disease [9] is exceedingly rare.

Calcific Aortic Valve Stenosis

The prevailing mechanism causing calcification is thought to be secondary to lipid accumulation, inflammation and proliferative cellular and extracellular changes (Fig. 1.4). Calcification leads to leaflet immobility and obstruction without commissure fusion (Fig. 1.5a, b). Atherosclerosis and calcific aortic stenosis share similar pathophysiologic features in that risk factors include hypertension, smoking, elevated LDL cholesterol [9]. However, various studies examining the role of statin therapy for delaying the progression of valvular aortic stenosis have been unsuccessful in documenting a preventative or therapeutic role for statin in patients with AS [11].

Congenital Aortic Valve Stenosis

Congenital aortic stenosis may be unicuspid or bicuspid (Fig. 1.6) with fusion of one or more commissures, and less commonly quadricuspid with a four leaflet aortic valve. Infants do not survive the severe obstruction caused from rare congenital unicuspid or quadricuspid valves unless surgically corrected. Bicuspid aortic valve disease is more common and occurs in 0.5–2 % of the population and in 66 % of all valves excised surgically for aortic stenosis with almost a double prevalence in males compared to women [9, 12]. However, only 1 in 50 children will develop significant obstruction by adolescence [13]. Patients may also present with aortic regurgitation with or without aortic stenosis [14, 15].

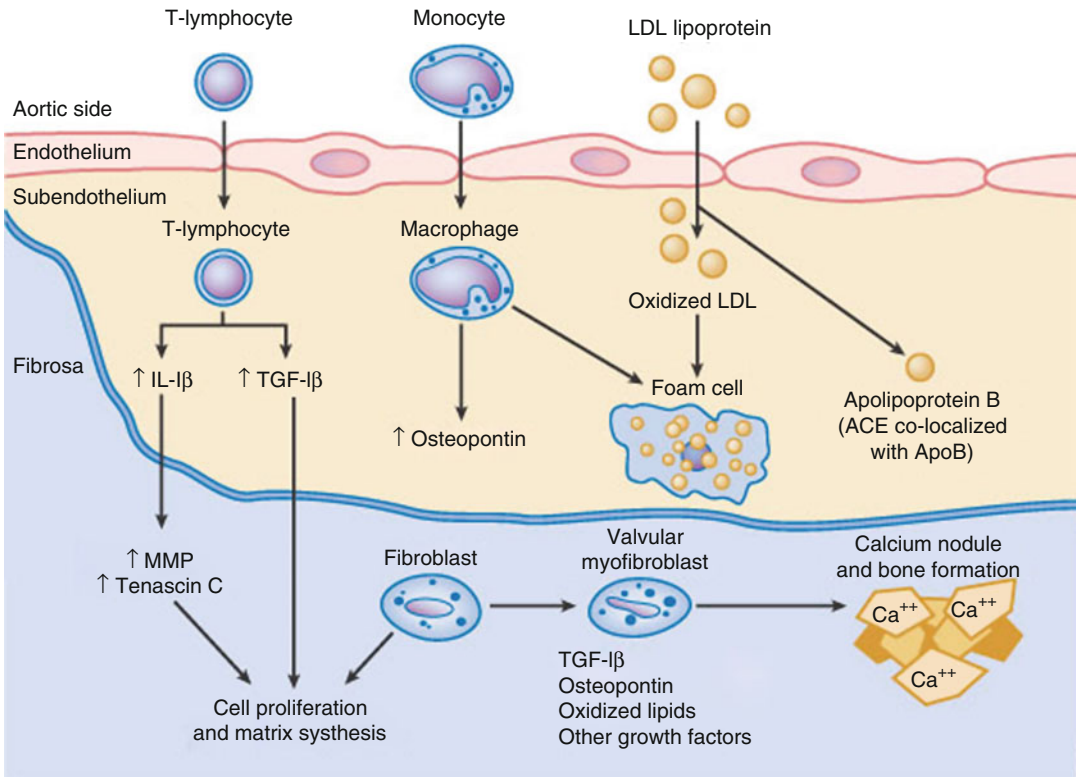

Fig. 1.4 Potential pathway depicting calcific valvular aortic stenosis pathophysiology. *1*. **T-lymphocytes and macrophages** infiltrate the endothelium and release cytokines, which act on valvular fibroblasts to promote cellular proliferations and extracellular matrix remodeling. *2*. A subset of **valvular fibroblasts** within the fibrosa layer differentiates into **myofibroblasts**, which possesses characteristics of smooth muscle cells. *3*. LDL particles taken into the subendothelial layer are oxidized an taken up by **macrophages** that become **foam cells**. *4*. ACE is co-localized with APoB and facilitates the conversion of angiotensin II, which acts on angiotensin 1 receptors, expressed on valve myofibroblasts. *5*. A subset of myofibroblast differentiates into an **osteoblast** phenotype that can promote calcium nodule and bone formation (From Libby et al. [10] with permission)

Bicuspid aortic valve (BAV) usually occurs from fusion of the right and left aortic cusps (70 %) and maybe associated with other forms of congenital heart disease including coarctation of the aorta (50–80 %), interruption of the aorta (36 %) and isolated ventricular septal defect (20 %) [16, 17]. Patients with either aortic coarctation or Turner syndrome should be screened for the presence of BAV as the incidence approaches 50 % and 10–12 %, respectively [18, 19]. Systolic doming of the aortic valve leaflets is demonstrated in the long axis of the AV on various imaging modalities as echocardiography and MRI. While a classic "fish mouth" appearance is noted in the short axis view during diastole, with the corresponding fused leaflets appearing as one as demonstrated in Fig. 1.6 [20]. Extensive hypertrophy and supernormal ejection performance are the rule with congenital aortic stenosis and systolic dysfunction is uncommon unless severe stenosis is present at birth. However, sudden cardiac death is more common in infants and children than in adults [8].

BAV maybe also associated with aortopathy and patients are at an increased risk of aortic dissection, dilatation and aneurysm formation due to medial tissue changings including loss of elastic fibers, altered smooth muscle cell alignment, and cystic medial necrosis [21]. Multiple studies have

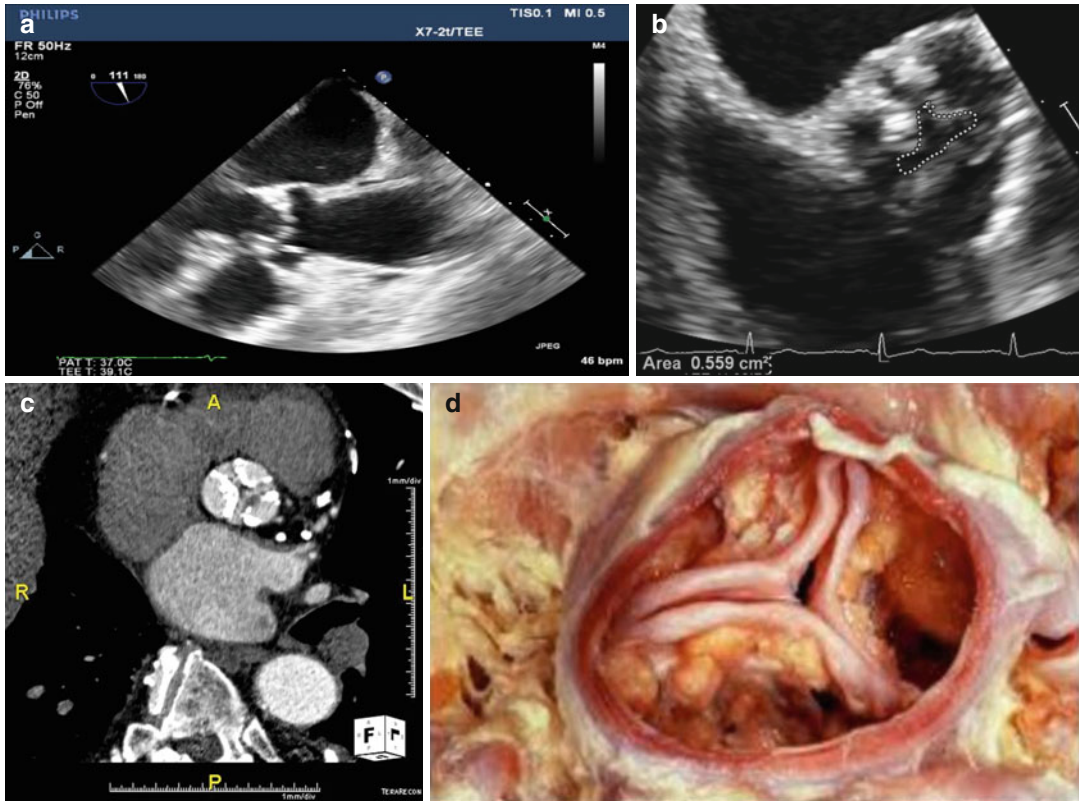

Fig. 1.5 Parasternal long (*top left*, **a**) and short (*top right*, **b**) axis transesophageal echocardiography showing reduced excursion aortic leaflets due to severe aortic stenosis. Cardiac CT angiography (*bottom left*, **c**) and surgical field (*bottom right*, **d**) demonstrating severe aortic stenosis

shown familial clustering but the exact genetic mechanisms are still under investigation. Inheritance is likely multifactorial and in some instances autosomal dominant inheritance with incomplete penetrance [9, 14, 22].

Rheumatic Aortic Valve Stenosis

Rheumatic aortic stenosis is rare due to the decline in rheumatic fever and is primarily associated with rheumatic mitral stenosis. Unlike calcific aortic stenosis, there is fusion of both the leaflets and commissures creating an immobile small triangular or round opening with eversion of leaflet tips. Calcific nodules can form on the leaflets and commissures creating a fixed opening that may lead to both aortic stenosis and aortic regurgitation (Fig. 1.7) [9, 15].

Para-valvular Aortic Stenosis

Supra Valvular Aortic Stenosis

Supra valvular aortic stenosis is exceedingly rare and may present either in isolation or as a part of congenital syndromes as autosomal dominant William's Syndrome or familial non-Williams supra valvular aortic stenosis. It may occur in the form of membranes, muscular ridges, or tunneling of the ascending aorta for variable distances (Fig. 1.8). The coronary arteries are proximal to the stenosis and are subjected to high systolic and limited diastolic flow and can have atretic ostia, ectasia, or aneurysms [23]. It has also been reported after arterial switch operation.

Associated features of patients with **William's Syndrome** include:

(a) Other cardiovascular abnormalities: aortic valve stenosis, pulmonary stenosis, renal

artery stenosis, and hypertension, sub valvular aortic stenosis, parachute mitral valve, bicuspid aortic valve, ventricular septal defect, and circle of Willis aneurysms.

(b) **Elfin features**: these include puffy eyes, star like pattern in the iris, short nose with broad nasal tip, full cheeks and lips, small chin, wide mouth, and small widely spaced teeth.

(c) Short stature, long neck, sloping shoulder, limited joint mobility, low muscle tone, and spine curvature, hyperacusis, strabismus, and poor growth

(d) Hypercalcemia, chronic ear infections, gastric reflux, and hernias

(e) Developmental delays, self mutilation, anxiety, phobias

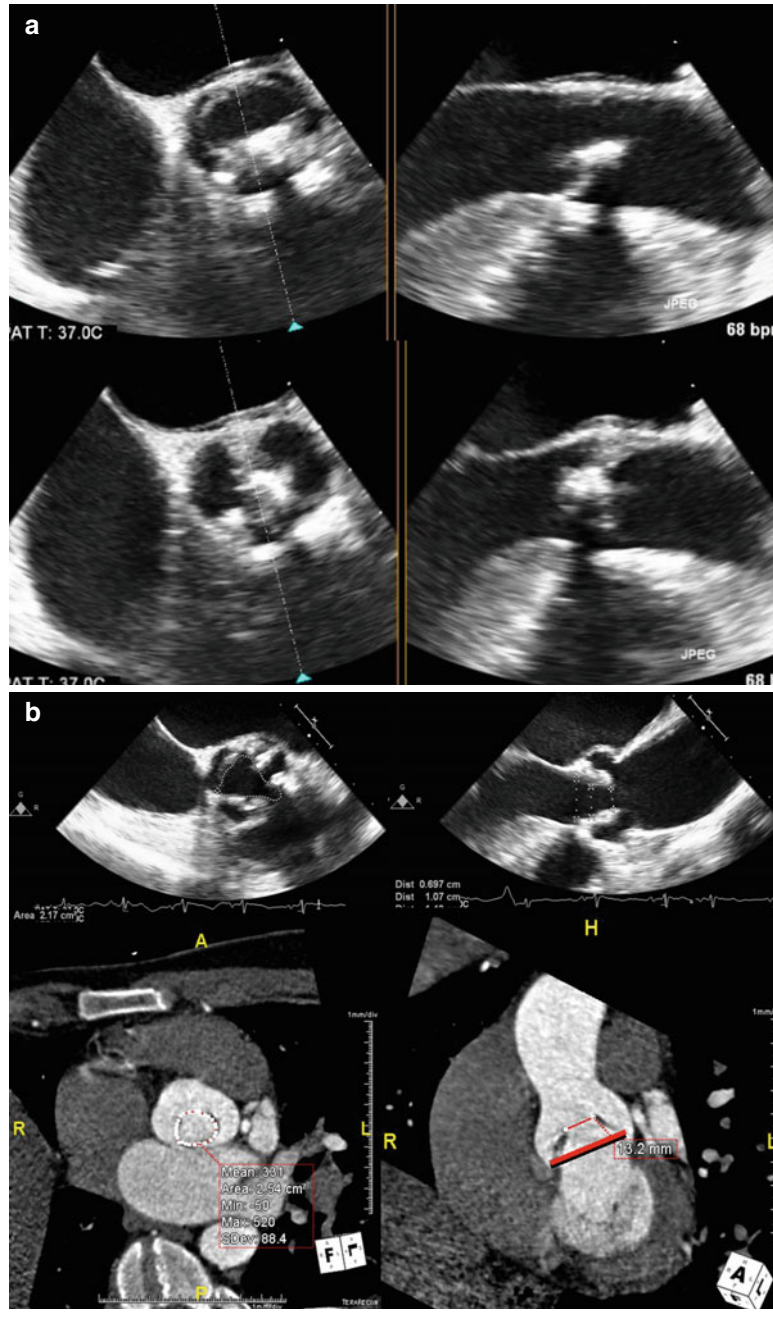

Fig. 1.6 Congenital unicuspid (**a**) and bicuspid valves (**b**) noted on echocardiography. The right cusp and non-coronary cusps are fused. (**c**) Demonstrates a calcified bicuspid aortic valve noted on CTA

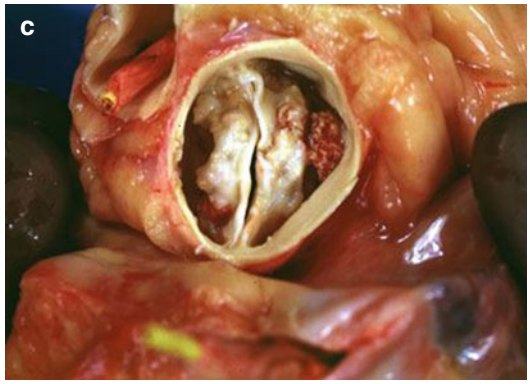

Fig. 1.6 (continued)

Surgical correction is indicated in patients with a mean Doppler gradient of 50 mmHg and/or a peak Doppler gradient of 70 mmHg, symptoms of angina, dyspnea, or syncope, in the presence of LVH, and in case of the desire of pregnancy or greater exercise [23].

It is performed by either a single patch through a single sinus incision (McGoon), inverted Y patch requiring double sinus incision (Doty), and the Brom and Myers techniques with either a three patch or direct three sinus incision, respectively. The latter is the most recent approach to surgical correction [23].

Sub Valvular Aortic Stenosis

Sub valvular aortic stenosis may occur due to a multitude of etiologies:

1. Fixed congenital: Fixed congenital sub valvular aortic stenosis is more common than the supra valvular form (Fig. 1.9). It may occur as a part of a familial syndrome as Shone's complex or occur in isolation with a 2:1 male predominance. Sub aortic membranes, muscular ridges, and tunnels can also account for the obstruction and can extend to the mitral valve anterior leaflet. It may occur with ventricular and atrioventricular septal defects and conotruncal abnormalities. Accessory mitral valve tissue or anomalous chords may also cause a fixed sub valvular obstruction [23]. Associated features of **Shone's complex** include: coarctation of the aorta, parachute mitral valve, supravalvar mitral membrane, bicuspid aortic valve, and valvular aortic stenosis.

Damage to the aortic valve from the eccentric high velocity jet may lead to aortic valve regurgitation further increasing the hemodynamic burden on the left ventricle and is present in 50 % of cases. Moreover, a dynamic element of obstruction may also co-exist from left ventricular hypertrophy, and in contrast to valvular aortic stenosis, no ejection click is noted.

Surgical intervention is indicated in patients with a peak Doppler gradient >50 mmHg, mean Doppler gradient >30 mmHg, or catheter peak-to-peak gradient >50 mmHg. Similar to patients with supravalvular obstruction, the presence of symptoms of angina, dyspnea, or syncope, or in the presence of LV systolic dysfunction or significant aortic valve regurgitation or the patient desires to become pregnant or to participate in active sports may be considered for surgery with lesser gradients. In patients with a lesser degree of obstruction, an exercise challenge may unmask higher gradients not noted on rest [23]. The presence of LV systolic dysfunction or a ventricular septal defect proximal to the subvalvular obstruction may result in underestimation of obstruction [23].

Surgical repair of the discrete membranous form usually involves circumferential resection of the fibrous ring and some degree of resection of the muscular base along the left septal surface. Injury to the aortic or mitral valves, complete heart block, or creation of a ventricular septal defect may occur as the result of surgery. Patients with associated aortic regurgitation often undergo valve repair at the time of subaortic resection. Fibromuscular or tunnel-type subvalvular obstruction is more difficult to palliate surgically and usually involves a more aggressive septal resection and sometimes mitral valve replacement. Patients with subvalvular obstruction due to severe long-segment LVOT obstruction may require a Konno procedure, which involves an extensive patch augmentation of the LV outflow area to the aortic annulus.

Postoperative complications may include infective endocarditis. Subvalvular obstruction may recur after surgical repair; repair of subvalvular obstruction in children does not necessarily

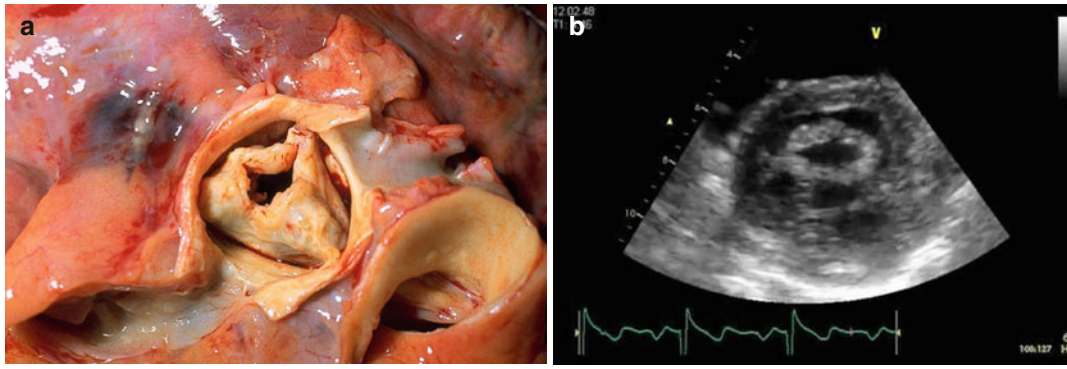

Fig. 1.7 Rheumatic aortic stenosis noted on a surgical specimen (*left*, **a**) and on echocardiography (*right*, **b**). Note the thickening and eversion of leaflet tips

prevent development of aortic regurgitation in adults [23]. The value of surgical resection for the sole purpose of preventing progressive aortic regurgitation in patients without other criteria for surgical intervention has not been determined and is an issue about which there is no clear consensus. However, data exist to suggest that surgical resection of fixed subvalvular before the development of a more than 40-mmHg LVOT gradient may prevent reoperation and secondary progressive aortic valve disease [23]. Although catheter palliation has been performed in some centers on an experimental basis, its efficacy has not been demonstrated [23].

2. Acquired Fixed: This can occur after ventricular septal defect patching or from a tilted mitral valve bioprosthesis into the LVOT. It occurs particularly in patients who with hypertrophic obstructive cardiomyopathy who undergo incomplete myomectomy and mitral valve bioprosthetic replacement (Fig. 1.10) [23].
3. Hypertrophic obstructive cardiomyopathy: Hypertrophic obstructive cardiomyopathy can also account for a dynamic obstruction of the left ventricular outflow and present in a similar fashion to that of other form of aortic stenosis in conjunction to other special features related to the cardiomyopathy (Fig. 1.11). Patient may suffer shortness of breath, chest pain, and/or syncope and management includes beta-blockers, calcium channel blockers, and adequate hydration. In patients with persistent symptoms, reduction of septal wall thickness either through surgical myomectomy or alcohol septal ablation (Fig. 1.12) may help alleviate symptoms. Identification of patients at risk for sudden cardiac death includes assessment of the presence of non-sustained ventricular tachycardia, septal wall thickness >3 cm, late Gadolinium enhancement on MRI, history of syncope, and family history of sudden cardiac death. Family members of patients with HOCM should undergo clinical and echocardiographic screening, while genetic screening of family members is indicated when an identifiable genetic mutation is discovered in the index patient [23].

Natural History of Aortic Valve Stenosis

In both calcific and bicuspid aortic stenosis, there is a long latent period of disease progression before symptoms develop. Onset of symptoms tends to occur between the ages of 50–70 years for patients with bicuspid valves and after age 70 for calcific trileaflet valves [24].

Angina, syncope and heart failure can develop with moderate or severe aortic stenosis and patients with severe or critical aortic stenosis can remain asymptomatic. Symptoms depend on left ventricular systolic function; stroke volume based on body surface area, preload, afterload and heart rate [25].

Risk factors for mortality in asymptomatic patients with moderate to severe aortic stenosis

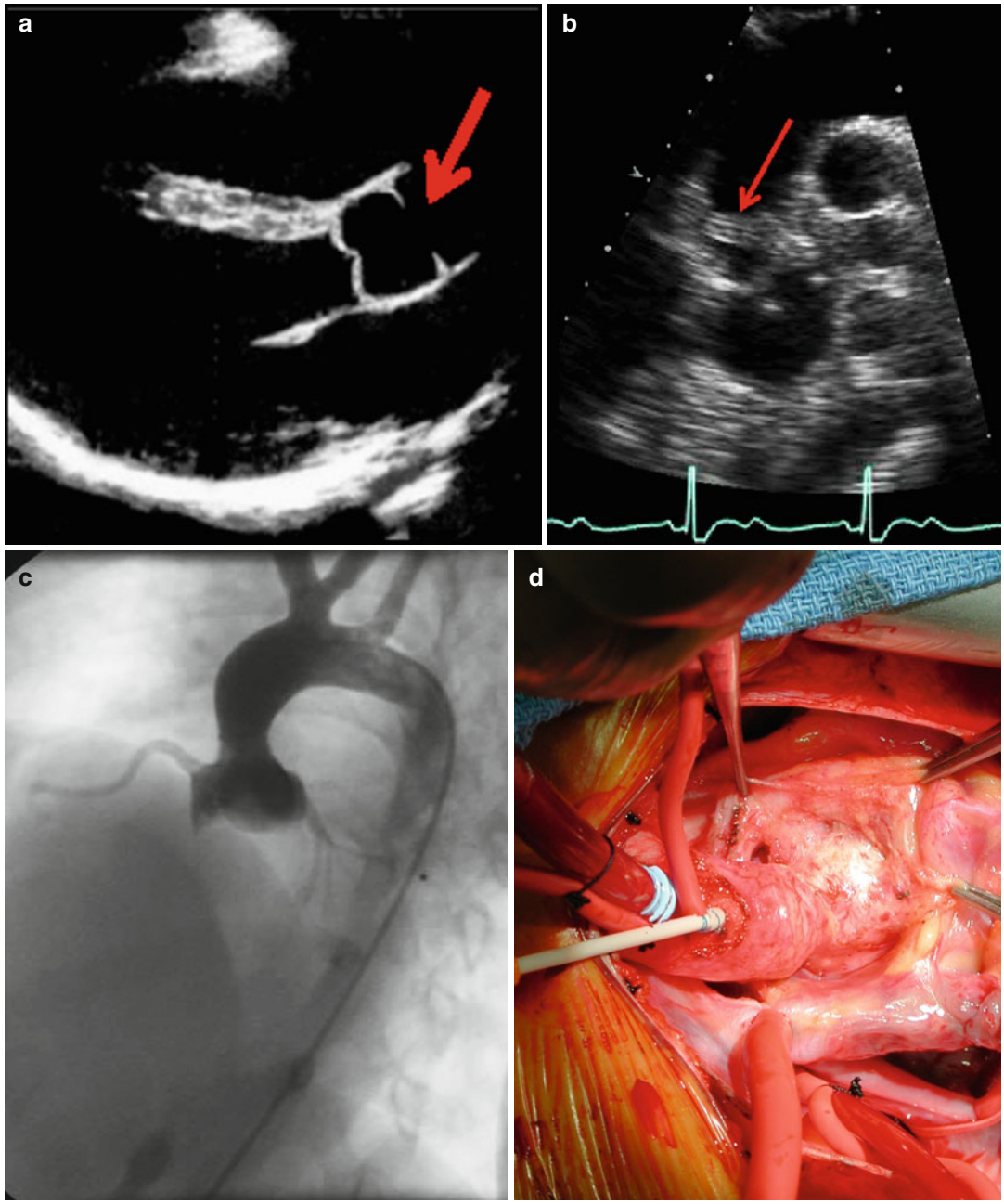

Fig. 1.8 Supravalvular obstruction noted on parasternal long (*top left*, **a**) and suprasternal (*top right*, **b**) echocardiographic images (*red arrows*). Also noted on angiography (*bottom left*, **c**) and surgical specimen (*bottom right*, **d**) (*black arrows*)

include elevated B-type natriuretic peptide (BNP), increase peak velocity across the aortic valve, female gender, and severity of ventricular remodeling [26]. Elevated BNP in asymptomatic or symptomatic patients independently predict symptoms and survival while N-terminal BNP predict post-operative morbidity and mortality after aortic valve placement [27, 28]. Women tend to have hypercontractile ventricles, poorer functional capacity, increased relative wall

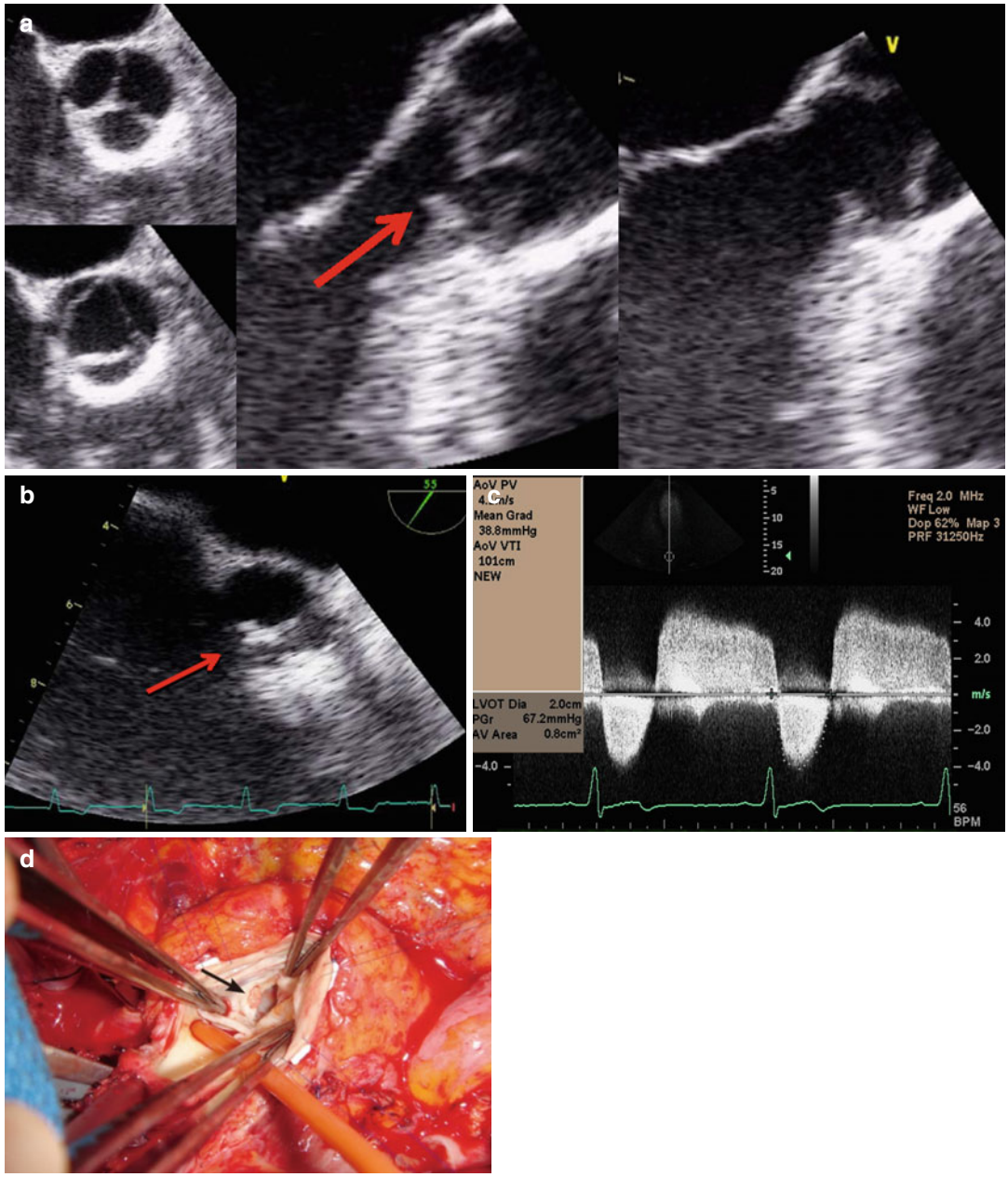

Fig. 1.9 Subaortic membrane: on TEE (*top left*, **a**) long axis, short axis (*top right*, **b**). Doppler across the LVOT revealing aortic stenosis and regurgitation (*bottom left*, **c**). *Bottom right* (**d**) image demonstrated surgical excision of a subaortic membrane

thickness, and more symptoms [29]. Patients with a depressed ejection fraction and low flow/low gradient severe aortic stenosis have worse outcomes, particularly in the absence of contractile reserve [9]. Normal left ventricular function with low flow/low gradient severe aortic stenosis occurs more frequently in women [30] and survival has also been reported as lower in these patients compared to those with normal flow and normal gradient aortic stenosis.

In severe aortic stenosis, ventricular remodeling including hypertrophy and altered geometry