3.1 Aortic Stenosis

3.1.1 The Pathophysiology of Aortic Stenosis

Aortic stenosis (AS) is an obstruction to blood ejection from the left ventricle (LV) due to a fixed or dynamic stenosis located in the valve either over (supravalvular) or below it (subvalvular) [1].

Supra- and subvalvular stenoses have a congenital genesis. AS is the most frequent form, and it accounts for the majority of congenital forms and all of the acquired forms. In most cases the etiopathogenesis of acquired AS is ascribable to a fibrocalcific degenerative process of the valve [2]. The characteristic morphological appearance of the non-rheumatic calcific AS consists of the presence of calcification in the cusps, preventing valve opening during outflow. The cusps have a calcific, fibrous and thickened appearance (Fig. 3.1). In some cases there can be a pathoanatomical condition marked by severe calcification of the walls of the ascending aorta, namely “porcelain aorta”, which is a high-risk picture for surgical aortic valve replacement. Calcification starts in the fibrous part of the valve, and the stratified microscopic structure is usually preserved. In degenerative AS, unlike in the rheumatic variety, there is no commissural fusion.

Congenital bicuspid aortic valve disease occurs in 1–2% of the population (Fig. 3.2). In most cases the cusps have different dimensions and a median raphe is often present due to their incomplete splitting. At birth, bicuspid aortic valves are not usually stenotic, but they are predisposed to gradually become stenotic owing to sclerosis and calcifications of mechanical origin. The raphe is the site where calcifications develop most frequently.
The aortic valve area (AVA) in adults amounts to about 3.0 cm² and varies within a range of 2.5 to 5 cm² depending on body surface area. In males, a transvalvular gradient can be measured when the AVA is reduced by at least 50% compared to normal [3, 4]. Valve stenosis exerts a resistance to ventricular outflow and, in order to maintain its outflow, the LV develops a higher systolic pressure. Pressure overload leads to concentric hypertrophy of the ventricular walls, namely the heart’s main compensation mechanism to cope with LV outflow obstruction. As a result of hypertrophy, left ventricular diastolic compliance tends to reduce, while telediastolic pressure rises without necessarily giving rise to ventricular decompensation [5]. In this case, left atrial contraction plays an important role in ensuring adequate filling pressure in the LV. In these patients, the loss of synchronous and vigorous pump function, as in the case of atrial fibrillation or atrioventricular dissociation, can cause rapid clinical deterioration [6]. With a further rise in afterload, the LV adopts additional compensatory mechanisms such as increase in preload and myocardial contractility. Both