Mitral valve (MV) function depends on the coordinated action of the anatomic components of the mitral apparatus which is formed by two leaflets, annulus, chordae tendineae, and the papillary muscles (PM). The annulus is a saddle-shaped structure with a fixed portion (the anterior leaflet (AML) which is semilunar in shape) shared with the aortic annulus, and a dynamic portion (the quadrangular-shaped posterior leaflet (PML) composed of three scallops) that represents most of the circumference of the annulus. The surface area of the leaflets is twice the area of the mitral orifice which results in a large area of coaptation when the valve closes. The annulus to leaflet transition zone contains atrial myocytes with nerve fibres which extend from the mitral annulus, maintaining electrophysiological continuity with the rest of the heart. The commissures are a distinct area where the AML and PML come together. Along the free edge of the leaflets the chordae tendineae are inserted through multiple locations with the other end attached to the tips of the anterolateral and posteromedial PM. The PM and the adjacent wall attach the mitral apparatus to the left ventricle. Diseases of the MV are valvular stenosis (MS), regurgitation (MR) and prolapse. MS is usually caused by rheumatic heart diseases (RHD). The prevalence of RHD in the USA and Japan stands at 0.6–0.7/1,000, which contrasts with that in the developing countries of Africa and Asia where rates are 30/1,000. Congenital MS is rare and is typically diagnosed in infancy or early childhood. MR is the result of structural or functional abnormalities of the MV apparatus. Functional abnormalities causing MR include myxomatous MV diseases, leaflet prolapse, RHD, infective endocarditis, coronary artery diseases and cardiomyopathy. Degenerative disease is the most common form MR in Europe (with an estimated prevalence of 2–3%) and other developed countries. Ischemic MR affects 19% of patients after myocardial infarction.