INTRODUCTION

Postinfarct ischemic cardiomyopathy is characterized by a variable degree of left ventricular (LV) dyssynergy secondary to dyskinetic and/or akinetic walls. Since 1984, we have surgically repaired such ventricles by inserting a patch inside the ventricle to exclude the nonresectable akinetic/dyskinetic segments and reestablishing the preinfarct ventricular geometry. Left ventricular reconstruction (LVR) using an endoventricular patch is an accepted and efficient technique to treat the dilated, hypocontractile ventricle of ischemic heart disease as both diastolic and systolic function are improved.
THE VENTRICULAR WALL AFTER INFARCTION

In the classic transmural infarct without reperfusion, the infarcted area undergoes necrosis, fibrosis, and sometimes calcification. Although the remaining uninfarced myocardium is initially normal, the transmural infarction produces increases in load and activates neuro-hormonal mechanisms that lead to eccentric myocardial hypertrophy and ventricular dilation (i.e., ventricular remodeling) of the myocardium remote to the original infarct. This course of events is most commonly seen in occlusion of the left anterior descending artery (LAD), leading to an antero-apical-septal scar.

However, in the modern era of early reperfusion therapy (i.e., angioplasty and thrombolysis) for myocardial infarction (MI), the classical process of transmural infarction has been modified into nontransmural infarction with necrosis of the subendocardial muscle and sparing of the subepicardial muscle (1). In this situation, the nontransmurally infarcted myocardial wall appears nonviable by echocardiography or ventriculography because of akinesia. Although thallium nuclear imaging may suggest viability, intraoperative findings usually demonstrate a thin rim of subepicardial myocardium of little functional importance, primarily because of the dominant underlying scar. Cardiac magnetic resonance imaging is able to detect these differences between transmural and nontransmural infarction (2,3). Subendocardial scar can induce immediately or progressively an asynergic LV wall.

Pathophysiology

Klein et al. demonstrated in 1967 (4) that if more than 20% of the left ventricle is infarcted, the Starling and Laplace laws will lead to progressive global ventricular hypokinesia. Gaudron et al. (5) showed that 20% of all MIs follow this evolution. The undamaged area is normal at first, then hypertrophied to compensate the lack of contractility of the necrotic wall, and is finally dilated by physical mechanical forces. The dilatation increases the stroke volume and temporarily improves the cardiac index (Starling law), but the increased wall tension has a detrimental effect on the myocardial contractility (Laplace law). This physical and mechanical explanation of the progressive dilatation of the heart—LV remodeling—is based on a complex inflammatory and neuro-hormonal process, which, in reality, is the result of the reaction of the “organism” against the lack of contractility of a large scar when the remaining nonischemic area is not able to assume and maintain a normal cardiac output. This reaction explains the progressive dilatation (remodeling), but is more a consequence than a cause of it. The