Специальность - терапия
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Прочитайте текст по Вашей специальности без словаря (время на подготовку пять минут), передайте его основное содержание на английском языке.

MOLECULAR MECHANISMS OF MYOCARDIAL REMODELING

"Remodeling" implies changes that result in rearrangement of normally existing structures. This review focuses only on permanent modifications in relation to clinical dysfunction in cardiac remodeling (CR) secondary to myocardial infarction (MI) and/or arterial hypertension and includes a special section on the senescent heart, since CR is mainly a disease of the elderly. From a biological point of view, CR is determined by *1*) the general process of adaptation which allows both the myocyte and the collagen network to adapt to new working conditions; *2*) ventricular fibrosis, i.e., increased collagen concentration, which is multifactorial and caused by senescence, ischemia, various hormones, and/or inflammatory processes; *3*) cell death, a parameter linked to fibrosis, which is usually due to necrosis and apoptosis and occurs in nearly all models of CR. The process of adaptation is associated with various changes in genetic expression, including a general activation that causes hypertrophy, isogenic shifts which result in the appearance of a slow isomyosin, and a new Na^+-K^+-ATPase with a low affinity for sodium, reactivation of genes encoding for atrial natriuretic fator and the renin-angiotensin system, and a diminished concentration of sarcoplasmic reticulum Ca^{2+}-ATPase, β-adrenergic receptors, and the potassium channel responsible for transient outward current. From a clinical point of view, fibrosis is for the moment a major marker for cardiac failure and a crucial determinant of myocardial heterogeneity, increasing diastolic stiffness, and the propensity for reentry arrhythmias. In addition, systolic dysfunction is facilitated by slowing of the calcium transient and the downregulation of the entire adrenergic system.