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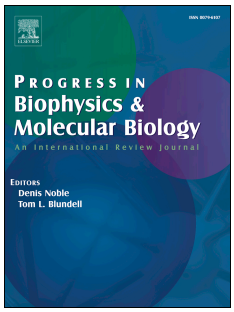

The phenomena of mechanical interaction of segments of hypertrophied myocardium

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Abstract: The main aims of adaptation mechanisms of heart contractility are to regulate the stroke volume and optimize the global heart function. These mechanisms manifest themselves in hearts of healthy animals and in hearts with severe hypertrophy in different ways. Severe right ventricle hypertrophy was induced by single treatment with monocrotaline. Young rats of both sexes were used to prevent influences of sex hormones on the development of right ventricular hypertrophy. Serial duplex method is used as a model of interaction of two ventricular wall segments. In serial duplex the muscles are in connection 'end-to-end' and subjected to mutual deformations during contractions. It is important to establish the fine-tuning phenomena and evaluate their expressiveness in healthy hearts and hearts with severe hypertrophy. Mild force transient processes occur on muscle connection to serial duplex and on muscle separation from duplex in all experimental groups. These transients manifest themselves as slow changes in the amplitude of muscle contraction from cycle to cycle. During the muscle interaction in the serial duplex, evident transient processes in the mutual amplitude of deformations in all experimental groups are observed. The greatest changes in the length occur in the relaxation phase of the contraction cycle. The loss of interaction between ventricular muscles of rats with severe heart hypertrophy is the most likely cause of an additional deterioration in the heart pumping function. New targets may occur for the recovery of contractility of hearts with severe hypertrophy.

Key words: serial duplex method; monocrotaline; right ventricular hypertrophy; transient process; muscle interaction.

1. Introduction

The spatiotemporal heterogeneity of mechanical, electrical and biochemical properties of different layers of the myocardial ventricle is now recognized as one of the fundamental paradigms of the heart that determine the myocardial pumping function in physiological and pathological states [Brutsaert, 1987; Lou et al., 2011]. The change in mechanical conditions of functioning of cardiomyocytes in different segments of the ventricular wall is the reason for