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Cellular and receptor mechanisms of impairment of myocardium and aorta contractility at Alzheimer's disease model

Leushina A., Gaifullina R., Zefirov A., Palotas A., Mukhamedyarov M.
Kazan Federal University, 420008, Kremlevskaya 18, Kazan, Russia

Abstract

Introduction: Recent studies certify the existence of link between Alzheimer's disease and cardiovascular pathology, however the mechanisms of this phenomenon is unclear. Here we studied the influence of Alzheimer's β-amyloid peptide (βAP) on the contractility of rat myocardium and aorta. Material and methods: Contractility of myocardium ventricle strips and transverse fragments of abdominal aorta was measured at Power Lab setup using conventional myographic technique. Contractile responses of aorta strips were evoked by application of receptor agonists, contractile responses of myocardium - by electrical stimulation. Contractile responses of aorta strips after application of carbachol (10-6-10-4 M), histamine (10 -6-10-4 M), norepinephrine (10-5-10 -3 M) and ATP (10-6-10-4 M) were measured. Results and discussion: We found the impairment of carbachol- and histamine-induced contractility of aorta, appearing as perverse contractile reactions (relaxation instead of contraction) under the action of βAP (10-6 M). Next, we found βAP-induced impairments of ventricle myocardium contractility, appearing as decrease of relaxation phase duration and increase of relaxation speed (positive lusitropic effect). Also, own positive lusitropic effect of norepinephrine was absent in presence of βAP (10 -6M). Thus, βAP(25-35) significantly impairs the contractility of rat myocardium and aorta, as well as processes of its regulation. Obtained data significantly broad our understanding of mechanisms of Alzheimer's disease pathogenesis and pathophysiology of cardiovascular system.

Keywords

β-Amyloid peptide, Alzheimer's disease, Aorta, Contractility, Lusitropic effect, Myocardium