J Molecular Cellular Cardiology, 15 (1), 154

Abstracts of the XI Congress of the International Society for Heart Research

London, UK, 11-14 July 1983

154 ABNORMAL CHANGES IN HIGH ENERGY PHOSPHATE STORES DURING INOTROPIC STIMULATION OF THE FAILING MYOCARDIUM. H. Pouleur, G. Maréchal, M.F. Rousseau, A.A. Charlier. Depts of Physiology and Cardiology, University of Louvain, Brussels, Belgium.

To determine if the high energy phosphate stores are preserved during inotropic stimulation in heart failure (HF), myocardial biopsies were obtained in 10 normal dogs (N) and in 10 with HF (caused by a chronic volume overload).Biopsies were taken before and during infusion of dobutamine or AR-L11.513S,a non catechol inotrope; the doses of both agents were adapted to produce comparable changes in dP/dt max (+30%) and in oxygen consumption in N and in HF. During control, high energy phosphate stores were similar in N and HF : ATP=4.9±0.4 vs 4.9±0.2 µMol/g wet weight (NS); ADP=0.8±0.1 vs 0.7±0.1 (NS); (ATP+CP)/CT=0.65±0.02 vs 0.68±0.03 (NS). During inotropic stimulation in N, ADP (-25%*) and AMP (-30% *)-declined with both drugs without ATP or CP changes; (ATP/ADP) rose by 28%* and adenylate charge by 5%. In HF, the effects of both drugs were again similar. However, changes in ADP (-6%) and AMP were blunted when compared to N; although (ATP+CP)/CT was unchanged, ATP declined (-0.8 µMol/g *) and (ATP/ADP) changes were abnormal (-9% vs +28% in N *).These results suggest an impaired adaptability of the failing myocardium to inotropic stimuli, which appears independent of the drug used and could be related to an adenine loss with a secondary accumulation of CP. * p< 0.05, CP = Phosphocreatine, CT = Total creatine.= CP + creatine.