Cyclic AMP-dependent protein kinase and mechanical heart function in ventricular hypertrophy induced by pressure overload or secondary to myocardial infarction

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The role of cyclic AMP-dependent protein kinase (PKA) and systolic function during the development of left ventricular hypertrophy (LVH) still remain uncertain. The aim of this work is to study PKA activity and mechanical heart function in two experimental heart hypertrophy models: specifically, one induced by pressure overload (Goldblatt model: two kidneys, one clamped, Gb); and another secondary to myocardial infarction (MI) generated by ligation of the left coronary artery. Hypertension in the Gb group becomes evident by the third and fourth week after surgery without any significant change in the corresponding sham group. The myocardial infarction group did not show any change in systolic pressure. Different degrees of LVH for the two experimental models were observed. Relative cardiac mass (RCM) and relative ventricular mass (RVM) increased 23 and 16%, respectively, above the sham-operated rats in MI group ($P<0.05$). For the pressure overload model, the increase values were 42 and