Aerobic organisms need sufficient oxygen supply to maintain homeostasis. These organisms are frequently exposed in hypoxic environments naturally, and also occur in hypoxic states in various pathological conditions. Cardioprotective effect of hypoxia had been recognised more than 30 years ago; and later on, cardioprotective effects of ischemic preconditioning were discovered. Long term exposure to hypobaric hypoxia activates cardioprotective mechanisms, which lower the aftermathes of short term ischemia of myocardia and the effects of further health complications. The core of protective mechanisms has not yet been fully clarified. This work deals with the significance of mitochondria on cardioprotection during hypobaric hypoxia adaptation. This work describes physiological adaptive processes on selected animals on natural hypoxic conditions and also molecular mechanisms, examined on experimental models. Molecular mechanisms of the origins of cardioprotective effects discovered so far, mainly indicate PKC signal pathways through thyrosine kinase and mitogenes of activated kinase and also indicate an activation of sarcKATP-channels and mitoKATP-channels. Opening of these channels can protect mitochondria against a Ca2+ overload, or can lead to an increase in mitochondrial capacity which is possibly connected with intracellular communication between organelles. Moreover, it can also inhibit the opening of mPTP, and thus prevent the cell from dying. Cellular adaptation upon hypoxic conditions is also realised by means of restructuring mitochondrial organisation, the basics of which is dynamic fusion and fission of these organells, in response to acute cellular energy demands.