This bachelor thesis describes a signal pathway of adenylyl cyclase, which plays a key role in the modulation of heart rate and force of contraction. This pathway begins with membrane-bound β-adrenergic receptors that activate the enzyme adenylyl cyclase. Adenylyl cyclase produces second messengers by reverting ATP to cAMP. Several changes occur in this pathway in failing heart. The most striking changes occur in β-adrenergic receptors, but there are some changes on the level of adenylyl cyclase and G proteins as well. Most of these changes are related to chronic high levels of catecholamines, especially norepinephrine. Some medications try to reverse these adverse effects of norepinephrine. β-blockers are traditional drugs for treating heart failure. However, adenylyl cyclase may be also considered as potential target for pharmacotherapeutic interventions in the future.