1.3.1 Propranolol mechanism of action

Beta-receptor antagonists antagonize the effect of catecholamines at β adrenoceptors by occupying this receptor and competitively countering its binding by catecholamines and other β agonists, thus, preventing catecholamines action on cardiovascular tissues (Craig and Stitzel 2004; Katzung *et al.* 2004). β_1 receptors are located on the cardiac sarcolema which belong to the G-protein coupled adenyl cyclase system. When catecholamines stimulate the receptor, α subunit of Gs protein binds to activates adenyl cyclase and generates cAMP. Later, cAMP as a second messenger activates protein kinase A (PKA) which phosphorylates specific proteins and other membrane calcium channels leading to an increase in calcium entry into the cytoplasm. PKA also increases calcium release from the sarcoplasmic reticulum which leads to the positive inotropic effect. Additionally, PKA accelerated conduction across atrioventricular node and conduction tissues leads to a positive dromotropic effect (Mansoor and Kaul 2009). PRN also has a membrane-stabilizing action (local anesthetic effect or quinidine-like effect). As a result, it can be used for the treatment of cardiac arrhythmias due to this advantage (Craig and Stitzel 2004; Katzung *et al.* 2004).