autonomic nervous system. (Phillips MI, *et al..*, 2002).Angiotensin II also interact with autonomic ganglia, increases the release of epinephrine and norepinephrine from the adrenal medulla, and facilitates sympathetic transmission by an action at adrenergic nerve terminals. (Campos AH, *et al..*, 2003).The latter effect involves both increased release and reduced reuptake of norepinephrine. Angiotensin II also has a less important direct positive inotropic action on the heart.

Numerous studies examining the cellular effects of Ang II. Showed that signals induced by Ang II in vascular smooth muscle cells (SMCs) and cardiomyocytes are associated with contraction, it was found that Ang II activated phospholipase C, resulting in the production of inositol trisphosphate (IP3) and diacylglycerol, which in turn responsible for the mobilization of [Ca2_] I and the activation of protein kinase C (PKC), respectively. (Ruiz-Ortega M, *et al..*, 2001).

These findings have spawned the development of several classes of pharmacological agents designed at inhibiting the synthesis of Ang II, eg, angiotensin II– converting enzyme inhibitors or blocking its action as with angiotensin II receptor antagonists, (Lonn, E. M *et al.*. 1994).