

The Role of Bradykinin in the Cardiovascular Action of Ramipril

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Abstrak

From pharmacologic investigations and clinical studies it is known that angiotensin-converting enzyme (ACE) inhibitors exhibit additional local actions, which are not related to hemodynamic changes and which cannot be explained simply by interference with the renin-angiotensin system with subsequent inhibition of angiotensin II formation. Because ACE is identical to kininase II, which inactivates the nonapeptide bradykinin (BK), potentiation of BK might be responsible for these additional effects of ACE inhibitors. To prove the specificity of BK-mediated effects by ACE inhibition, we used the specific B2 kinin receptor antagonist HOE 140 in different models: endothelial cell cultures; atherosclerosis in high-cholesterol-fed rabbits; neointima formation with smooth cell proliferation and migration after endothelial denudation in rats; myocardial ischemia in rats, rabbits, and dogs; and left ventricular hypertrophy in rats. The beneficial effects of ramipril or BK given in non-blood pressure-lowering doses in these models were abolished by HOE 140 (icatibant). Ramipril exerts cardioprotective effects in different experimental models. The formation of the endothelial autacoids nitric oxide and prostacyclin, enhanced when BK degradation is inhibited by ACE inhibition, may contribute to the observed beneficial effects.