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프로베네시드의 혈관 알파 수용체 길항 작용

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Probenecid inhibit α-adrenergic receptor mediated vasoconstriction

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It has been suggested that hyperuricemia is related to the development of essential hypertension. Hypertensive patients with hyperuricemia has decreased glomerular filtration activity as compared to normotensive patients with hyperuricemia. These studies indicates uric acid concentrations in blood is associated with hypertension. Probenecid is an uricosuric agent which decreases uric acid reabsorption at the proximal tubule. Recently, we have shown that probenecid exerts anti-hypertensive action in Spontaneously Hypertensive Rats. Considering these results, I have designed a series of experiments to explore potential mechanism of antihypertensive action, of probenecid. In isolated rat thoracic aorta, probenecid significantly prevented phenylephrine-induced contraction of the blood vessel. When endothelium removed blood vessels were used, probenecid produced same effect as the intact blood vessels, indicating that probenecid directly act through the α-adrenergic receptor in vascular smooth muscles rather than through endothelium. These results suggest that one of the mechanism of antihypertensive effects of probenecid is due to the direct inhibition of α-adrenergic receptor in blood vessels.