EFFECTS OF PARATHYROID HORMONE (PTH) ON CONTRACTILE RESPONSES IN RAT MESENTERIC ARTERY

Eri SAKO

Laboratory of Pharmacology,
Department of Biomedical Sciences,
School of Veterinary Medicine,
Hokkaido University, Sapporo 060, Japan

1. To examine physiological role of parathyroid hormone (PTH) receptor in vascular system, we investigated the effects of PTH on contractile responses induced by phenylephrine, high K^+ solution and caffeine in vascular smooth muscle of the rat mesenteric artery.

2. PTH induced endothelium-independent relaxation which was not affected by isobutyl-methylxanthine (1 μM).

3. Methoxyverapamil (1 μM) abolished the contraction induced by high K^+ solution, but not that induced by phenylephrine. Both PTH and PTH-related peptide relaxed the preparations contracted with high K^+ solution (60 mM) and phenylephrine (3 μM). The contraction induced by phenylephrine was more sensitive to these peptides than that induced by high K^+ solution.

4. Phenylephrine caused a contraction and an increase in the intracellular Ca^{2+} level measured with fura-2, both of which were reduced by PTH (100 nM). However, PTH decreased high K^+ solution-induced contraction without affecting the intracellular Ca^{2+} level.

5. In Ca^{2+}-free solution, PTH decreased a transient contraction and an increase in intracellular Ca^{2+} level evoked by phenylephrine (10 μM), but not those evoked by caffeine (30 mM).

6. These results suggest that stimulation by PTH receptor causes a decrease in contraction induced by phenylephrine due to the inhibition of Ca^{2+} influx probably through non-selective cation channels and Ca^{2+} release from intracellular Ca^{2+} stores. It is also suggested that PTH inhibits the contraction induced by depolarization through the decrease in Ca^{2+} sensitivity to the contractile apparatus without affecting voltage-dependent Ca^{2+} channels, and that PTH has no effect on Ca^{2+} induced Ca^{2+} release.