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

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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 ($1 \mu\text{M}$).

3. Methoxyverapamil ($1 \mu\text{M}$) 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 ($3 \mu\text{M}$). 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 ($10 \mu\text{M}$), 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.