POTENTIATING EFFECT OF SECRETIN ON CHOLECYSTOKININ OCTAPEPTIDE-INDUCED PANCREATIC EXOCRINE SECRETION AND ITS DEPENDENCE ON THE VAGAL REFLEX IN THE ANESTHETIZED RAT.

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1. The effect of secretin on the secretory responses to cholecystokinin octapeptide (CCK-8) was studied in anesthetized rats. Continuous intravenous infusion of CCK-8 (100pmol/kg/hr) is known to cause a physiological plasma CCK-8 level, and 1hr infusion was adopted in the present study.

2. In rats anesthetized by intraperitoneal injection of pentobarbital sodium, the intravenous CCK-8 infusion caused significant increases in secretory responses: the protein output was 2.6 times greater and the juice flow was 2.1 times greater than the corresponding resting levels. The infusion of secretin (2.5pmol/kg/hr) caused little if any secretory responses. The infusion of CCK-8 in combination with the secretin induced larger and significant increases in secretory responses: the protein output was 4.2 times greater and the juice flow was 3.4 times greater than the corresponding resting levels. The secretory responses to the simultaneous infusion of CCK-8 and secretin were significantly greater than the corresponding responses to the CCK-8 infusion.

3. After truncal vagotomy, these secretory responses to the intestinal peptide hormones were significantly reduced: the protein output during the CCK-8 and secretin infusion was 1.2 times greater and the juice flow was 1.7 times greater than the corresponding resting levels.

4. In rats anesthetized by intramuscular injection of urethane, the secretory responses to the simultaneous infusion of CCK-8 and secretin were apparently larger than those to the CCK-8 infusion, but the differences between the corresponding values were insignificant. After the truncal vagotomy, the protein output induced by the simultaneous infusion of CCK-8 and secretin was significantly smaller than the corresponding value obtained in intact innervation.

5. The present study supports the view that secretin in a physiological dose significantly potentiates the secretory responses to a physiolosical dose of CCK-8, and provides evidence that the potentiation may be due to activation of the vagal nerve reflex. Furthermore, results of the present study may be compatible with the view that the vagal reflex may greatly be influenced by the anesthetics used, anesthetizing procedure, and stage of anesthesia.