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Emctic Action of Glucagon(2): A Pharmacodynamic Analysis of Emetic Mechanism Using Pigeons*

Akiko Kaneko and Toshimitsu Uchiyama

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The mechanism of glucagon (GC)-induced emesis was analyzed with some dopaminergic neuron-antagonists, especially using emesis in pigeons, and stereotyped behavior in mice and rats which do not show emetic actions.

Intravenous injection of 300 μg/kg GC induced emesis which was completely suppressed by α-methyl-tyrosine (mT) 300 mg/kg po (200 mg/kg before 24 hours, 100 mg/kg before 3 hours), reserpine (Res) 2 mg/kg *ip* (1 mg/kg, every 24 hours before 48 hours), α-methyl-DOPA (mDOPA) 400 mg/kg *ip* (before 3 hours) and and by chlorpromazine (CPZ) 3 mg/kg *im* (before 30 minutes), but was partially blocked by haloperidol (HL) (0.5, 1 and 5 mg/kg *im* before 30 minutes) in 50, 25 and 60% respectively. Intravenous injection of 1 mg/kg of apomorphine induced pecking response which was completely suppressed by CPZ or HL, but was not suppressed by mT, mDOPA or Res.

These results show that GC emesis may be mainly induced by dopamine release from dopaminergic neurons in chemoreceptive emetic trigger zone, and partially participated in by the nor-adrenergic system, *etc.*, which was suggested by incomplete suppression with HL, a specific dopaminergic receptor blocker, and by complete suppression with CPZ which partially possesses nor-adrenergic blocking action. In clinicl use, CPZ will be most useful for GC emesis.

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