MECHANISM OF HYPOCALCAEMIA INDUCED BY INTRAPERITONEAL AND INTRAGASTRIC ADMINISTRATION OF ETHANOL

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ABSTRACT

Acute hypocalcaemic effects and mechanism of action of an intraperitoneal administration of 3 and 5 g/kg body weight and an intragastric administration of 3,5 and 7 g/kg body weight ethanol were investigated in 20-hour fasted female Wistar rats weighing between 180-200 g. Dose-dependent hypocalcaemia was similarly induced by both routes of administration within 30 minutes. Neither intraperitoneal nor intragastric administration of 3 g/kg body weight ethanol had any effect on increasing the plasma calcium efflux which was represented by disappearance rate from plasma of ^{45}Ca administered intravenously at 0 minute. In fact, higher doses of ethanol slowed down the calcium efflux. This delay was not PTH dependent as it was also observed in thyroparathyroidectomized rats.

While intraperitoneal ethanol was without effect on luminal calcium content,the intragastric administration of 3,5 and 7 g/kg body weight ethanol increased the luminal calcium content in gastrointestinal tract from 52.04±9.46 µg/100 g body weight in control group to 84.6±8.6 ($P<0.05$), 93.7±7.3 ($P<0.01$) and 89.0±9.1 µg/100 g body weight ($P<0.01$) respectively partly by enhancing plasma to lumen calcium transport from 0.36±0.07% administratered dose in control group to 1.82±0.20% ($P<0.001$), 2.94±0.25 ($P<0.001$) and 2.78±0.19% ($P<0.001$) respectively. However, the amount secreted was too small to affect the plasma ^{45}Ca disappearance rate.

Significant increase in soft tissue ^{45}Ca content at 30 minutes after ethanol administration was evident in the duodenum (131%), jejunum (127%) and colon (133%) in the intragastric ethanol-treated group and in the duodenum (140%), jejunum (144%), ileum (145%), colon (139%) and liver (125%) in the intraperitoneal ethanol-treated group.

Thus the hypocalcaemia induced by intraperitoneal ethanol may be partly accounted for by suppression of calcium release from some soft tissues while that induced by intragastric ethanol may be accounted in part by suppression of the return of calcium to the plasma from both the intestinal tissues and the gastrointestinal lumen.