

Serum Calcium After Calcium Infusion

Role of Possible Ectopic Production of Calcitonin in the Thyro-parathyroidectomized Rat

Short Report

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INTRODUCTION

In 1962 Copp et al. (3) demonstrated the existence of the hypocalcemic peptide hormone calcitonin (CT). The thyroid C-cell has been found to be the major producer of the hormone in most mammals (5). Increasing awareness of embryological, cytological and possible even functional relationships between cell types originally derived from primitive neural ectoderm (7) and the observations of CT production in pheochromocytomas and mucosal neuromas (8) have raised the question whether or not the normal adrenals might be a focus for ectopic production of CT. At least one clinical report (6) suggests a CT-like factor from the human adrenal gland.

The purpose of the following investigation was to evaluate the role of the adrenals in the homeostatic mechanism concerning regulation of serum calcium level after calcium infusion in the thyro-parathyroidectomized rat.

Material and methods

Fifty Sprague-Dawley rats, weight about 300 g, from the same strain were used. All the rats were thyro-parathyroidectomized. One week later the animals with verified thyro-parathyroidectomy (reduced serum calcium level: mean $-1.64 \times \text{S.D.}$) were further adrenalectomized or sham-operated. One week after this operation and after 4 days of corticosteroid substitution (Precortalon 3 mg \times 2 s.c.) the rats were anesthetized with Nembutal® and intravenous calcium infusions (Kalciumlevulat®) were performed for 5 minutes with 2.6 mg calcium/100 g body weight. Blood for analysis of calcium was withdrawn from a central venous catheter before and 15, 30, 60, 90 and 120 minutes after the start of the infusion.

In two additional groups the renal pedicles were ligated immediately before the calcium infusion. The experimental protocol was otherwise the same as above.

Results

The serum calcium levels after calcium infusion were unaffected by adrenalectomy, as compared with sham-operated rats (Fig. 1). The serum calcium levels were somewhat higher in rats with ligated kidney pedicles but the pattern of variation was the same (Fig. 2). All the rats had retained their ability to lower their serum calcium levels after intravenous calcium infusion.

Comments

The ability of the animals to lower their serum calcium levels after thyro-parathyroidectomy, adrenalectomy, and ligation of the kidney pedicles, might be due to ectopic production of CT from sources other than the adrenals. Another possibility is a direct hypocalcemic effect of glucagon (1, 2, 4) and/or a glucagon stimulation of ectopic C-cells producing CT. As glucagon has been shown to lose its hypocalcemic activity in parathyroidectomized rats (4) the most likely explanation of the retained calcium lowering capacity seems to be ectopic production of CT.

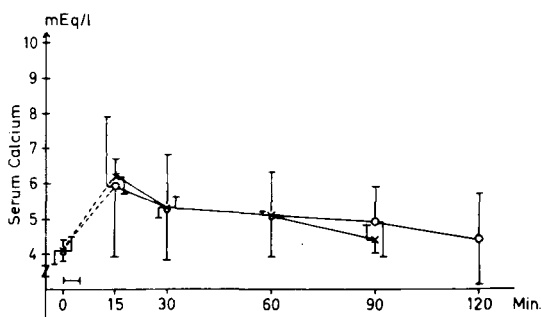


Fig. 1. Preserved kidney function. — : calcium infusion, x: adrenalectomy, O: sham-operation. $M \pm 1$ S.D. See text.

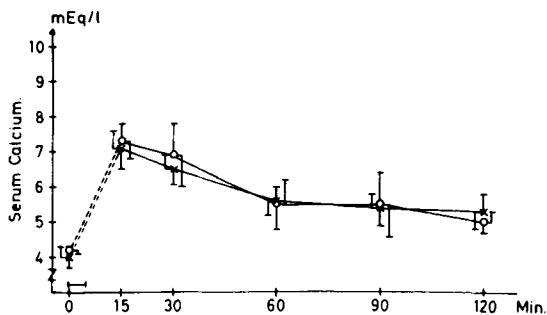


Fig. 2. Ligated kidney pedicles. Same symbols as in Fig. 1. See text.

Conclusion

Thyro-parathyroidectomized rats retain their ability to lower their serum calcium levels after calcium infusion. This ability appears to be unaffected by adrenalectomy and ligation of kidney pedicles. The experiments did not support the theory of calcitonin production in the adrenals.

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