PRELIMINARY REPORT

Effect of Calcium on Iodine Metabolism in Man

By Michael T. Harrison, Ronald McG. Harden and W. Donald Alexander

The intestinal absorption of iodine is unaffected by calcium ingestion, irrespective of the level of iodine intake. In patients maintained on a high intake of calcium, withdrawal of calcium does not result in any change in levels of plasma protein-bound and inorganic io-

dine, thyroid and renal clearance or absolute uptake of iodine by the thyroid, suggesting that calcium does not interfere with trapping or binding of iodine by the thyroid gland. (Metabolism 16: No. 1, January, 84–86, 1967)

THERE IS EVIDENCE that a high intake of calcium may potentiate the goitrogenic effect of an iodine-deficient diet in rats,¹ and it has been suggested that calcium is one factor responsible for goiter in man in endemic areas.² An effect of calcium on iodine metabolism in man has not, however, been shown. We have carried out stable iodine studies before, during and after high calcium feeding to determine whether calcium affects normal iodine metabolism in man.

METHODS

Iodine balance studies were carried out in 3 volunteers, one of whom had iodine-deficiency goiter and the others normal thyroid function. The balances took place in a metabolic ward,³ a low-iodine diet being supplemented by potassium iodide to provide different levels of iodine intake. After equilibration, balances were done before and during administration of calcium galactogluconate (Sandoz) in a dose of 3 Gm. calcium daily. Urinary iodine was measured daily and fecal iodine on 3 to 7 day collections.

Thyroid function was studied in 4 female patients with osteoporosis who had been treated with calcium glycerophosphate in a dose of 3 Gm. calcium/day for periods of 1 to 2 years. The serum protein-bound iodine (PBI), plasma inorganic iodine (PII), thyroid and renal iodine clearance, and absolute iodine uptake by the thyroid were measured⁴ before calcium withdrawal and at 1, 2, and 4 weeks after stopping calcium.

RESULTS

Iodine balances before, during and after calcium loading are shown in Table 1. Calcium had no effect on fecal iodine excretion at levels of iodine intake between 187 and 589 μg ./day. In the patient whose iodine intake was 589 μg ./day throughout, a significant fall (P < 0.001) in urinary iodine level occurred, averaging 100 μg ./day when calcium was given, with a rise to the previous value again on stopping calcium.

The average values for PBI, PII, thyroid and renal clearance and AIU in the

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Received for publication Aug. 17, 1966.

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Catetum Administration							
Patient	No. of Days on Balance	Ca Supplement g/day		Iodine μg./Day			
			Intake	Urine	Feces	Balance	
Male, 60	29		589	326	21	+242	
emphysema	20	3.0	589	227	22	+340	
	19		589	348	14	+227	
Male, 42	17		187	30	29	+128	
iodine deficiency	29	3.0	187	48	18	+121	
Male, 64	11	3.0	187	72	10	+105	
gastrectomy	13	3.0	589	364	13	+212	
	22		509	302	14	+193	

Table 1.—Iodine Balances in 3 Patients before, during and after Calcium Administration

Table 2.—Mean Values for Stable Iodine Measurements in 4 Patients before and after Stopping Calcium Supplements

/	Weeks after Calcium Withdrawal					
	0	1	2	4		
PBI (µg. %)	5.5	5.3	5.3	6.1		
PII (μg. %)	0.08	0.10	0.12	0.08		
Thyroid clearance (ml./min.)	20	17	17	11		
AIU (μg./hr.)	0.9	1.1	1.3	0.6		
Renal clearance (ml./min.)	28	22	23	21		

4 patients before and after calcium supplements were withdrawn are shown in Table 2. No significant change in any of these values occurred.

DISCUSSION

Several mechanisms have been suggested by which calcium could potentiate the goitrogenic effect of iodine deficiency. First, calcium might impede the intestinal absorption of iodine; second, the urinary excretion of iodine might be increased; and third, calcium might inhibit the uptake of iodine by the thyroid gland. Our studies appear to exclude all these possibilities. Calcium had no effect on the intestinal absorption of iodine at different levels of iodine intake. No increase in urinary excretion of iodine was observed in the balance studies, and renal clearance of iodine was unaffected when calcium supplements were stopped. Calcium was also without effect on thyroid trapping or binding of iodine, since we have shown^{8,9} that agents producing either inhibition of trapping or of binding are associated with striking changes in PII and thyroid clearance immediately after withdrawal. The finding of a consistently decreased urinary excretion of iodine during calcium administration in one individual is interesting but cannot be explained on the present data.

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