EFFECTS OF GROWTH HORMONE ON CALCIUM AND MAGNESIUM METABOLISM

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The anabolic effects of growth hormone have been well documented: several preparations from pituitary glands of monkey and man were shown to produce retention of nitrogen, potassium, and phosphorus (Raben, 1957; Beck, McGarry, Dyrenfurth, and Venning, 1957, 1958; Ikkos, Luft, and Gemzell, 1958; Raben, Westermeyer, and Leaf, 1952; Hutchings, Escamilla, Deamer, and Li, 1959). However, the effects of this hormone on calcium metabolism are not agreed upon, and reports on its effects on magnesium metabolism are very scanty.

Beck *et al.* (1957, 1958) reported a retention of calcium after the administration of growth hormone (human or monkey) to human subjects. Hutchings *et al.* (1959) found that injection of growth hormone resulted in an increased loss of calcium, a result which is teleologically unexpected from a hormone whose primary function is to promote growth. They found, however, that prolonged administration of the hormone resulted in a positive calcium balance. Ikkos *et al.* (1958) found that administration of growth hormone resulted in a positive balance in one subject and a negative balance in another. They noted that the urinary calcium was increased in both subjects after administration of the hormone. Pearson, Soroff, Prudden, and Schwartz (1960) reported that the net balance after administration of growth hormone depended on the intake: if the intake exceeded a certain "critical level" retention occurred; if it was below that level, growth hormone increased the loss. They claimed that this was true for calcium and magnesium as well as for other substances.

Disturbances of calcium metabolism in acromegalic patients may provide a clue to the metabolic effects of growth hormone. Scriver and Bryan (1935) described a female acromegalic patient who had thinning of bones. Her urinary calcium was high and her faecal calcium was low. Bauer and Aub (1941) reported hypercalciuria in four out of five acromegalics.

Material and Methods

Seven cases are presented. The first patient (Case 1) was an acromegalic. He was studied before and after pituitary Au-198 implant. The second patient was an osteoporotic woman. In the third patient, a case of malignancy, pituitary ablation was performed to control her bone metastases; after ablation she was given growth hormone to study its metabolic effects. The last four patients (Cases 4–7) were clinically possibly hypopituitary subjects. Cases 1, 3, and 4 were possible pituitary dwarfs. Injections of growth hormone were given to them for curative as well as for investigative purposes.

All the cases reported here showed one or more of the accepted metabolic responses to growth hormone treatment (fall in blood urea, increased nitrogen retention, rise in non-esterified fatty acids, and/or change in insulin sensitivity).

The patients were studied in the metabolic unit. Balance techniques and analytical methods were those described by Hanna, Harrison, MacIntyre, and Fraser (1960). Human growth hormone (H.G.H.) prepared by the method of Raben (1957) was usually employed, but sometimes chymotrypsin-treated bovine growth hormone (B.S.H.) was used. The hormone was injected intramuscularly at 7 a.m.

Results

Acromegaly

Fig. 1 and Table I show the magnesium and calcium findings in the acromegalic patient before and after pituitary Au-198 implant. The urinary calcium, which

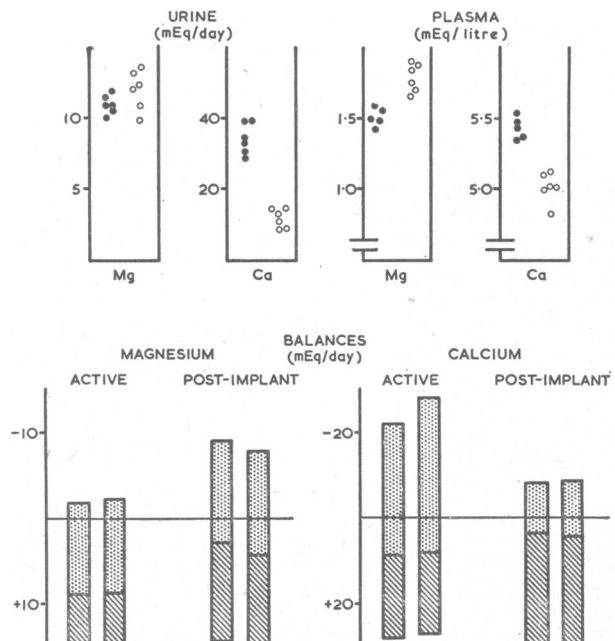

FIG. 1.—Case 1 (acromegalic). Magnesium and calcium findings before and after pituitary Au-198 implant. ●=Active. ○=Post-implant.

was initially very high and well outside the range reported by Knapp (1947), fell, after ablation, to normal levels. Together with this drop in urinary excretion there was a diminution in the amount of calcium absorbed from the gut despite a higher intake. Intestinal absorption of magnesium paralleled that of calcium, while urinary magnesium was not significantly changed. The plasma calcium was lower after ablation, while the plasma magnesium was changed in the opposite direction.

Effects of Injection of Growth Hormone

Fig. 2 and Table II show the results of the injection of growth hormone. Table III summarizes these results, together with those judged by the effects of withdrawal

by pituitary ablation in the acromegalic patient. The most striking effect was that on urinary calcium. In all cases except Case 3 injection of growth hormone was followed by a rise in the urinary excretion of calcium; in Case 3 there was an initial diminution in urinary excretion followed by a rise on the next day. The effect on urinary magnesium paralleled that on calcium in Cases 5, 6, and 7. The faecal loss of magnesium was diminished in all cases after the injection of growth hormone. The effect on faecal calcium (though less definite) tended again to parallel that on magnesium. While the net balance tended to show increased retention or diminished loss of magnesium in four out of the six cases studied, increased absorption from the gut was offset by increased urinary loss in the other two cases. Plasma magnesium and calcium were studied only in Cases 2, 3, 6, and 7. In these cases there was a drop in plasma magnesium levels after the administration of the hormone. The effect on plasma calcium was less definite and in the opposite direction.

TABLE I.—*Findings in Case* 1

Date	Plasma		Urine			Faeces		Diet		Supplement Ca	Balance	
	Mg	Ca	Vol.	Mg	Ca	Mg	Ca	Mg	Ca		Mg	Ca
	mEq/l.		ml./Day	mEq/Day								
7/9/59	1·56	5·33	1,110	10	28	7·2	18	16·3	7·4	20	−1·8	−21·1
8/9/59	1·41	5·35	1,990	11·5	33	7·2	18	15·5	6·8	20	−1·8	−21·1
9/9/59	1·53	5·46	1,660	10·5	30	7·2	18	16·3	7·4	20	−1·8	−21·1
10/9/59	1·46	5·52	1,816	11·7	39	6·7	18	15·5	6·8	20	−1·9	−27·7
11/9/59			1,210	10·8	39	6·7	18	16·3	7·4	20	−1·9	−27·7
12/9/59	1·48	5·42	920	10·4	35	6·7	18	15·5	6·8	20	−1·9	−27·7
13/10/59	Pituitary implant (Au-198)											
4/1/60	1·86	5·10	3,780	13·6	12·8	11	30	13·5	5·2	28	−8·9	−7·1
5/1/60	1·64	4·81	3,570	10·6	8·6	11	30	15·6	5·8	28	−8·9	−7·1
6/1/60	1·66	5·01	3,321	12·1	10·2	11	30	13·5	5·2	28	−8·9	−7·1
7/1/60	1·85	5·02	3,120	11·9	13·7	10·5	29	15·6	5·8	28	−7·3	−7·2
8/1/60	1·73	5·00	2,220	9·9	8·9	10·5	29	13·5	5·2	28	−7·3	−7·2
9/1/60	1·81	5·10	3,350	13·3	12·8	10·5	29	15·6	5·8	28	−7·3	−7·2

TABLE II.—*Findings in Cases* 2–7

Date	Plasma		Urine			Faeces		Diet		Supplement Ca	Balance		Remarks
	Mg	Ca	Vol.	Mg	Ca	Mg	Ca	Mg	Ca		Mg	Ca	
	mEq/l.		ml./Day	mEq/Day									
Case 2													
22/11/59			1,870	7·7	17·4	6·2	82·5	15·1	8·7	114	+1·1	+22·2	
23/11/59			1,520	7·7	17·7	6·2	82·5	14·8	7·2	114	+1·1	+22·2	
24/11/59	1·63	5·11				6·2	82·5	15·1	8·7	114	+1·1	+22·2	
25/11/59	1·71	5·02				7·1	91·6	14·8	7·2	114	0	+14·7	
26/11/59	1·71	5·12	1,280	8·1	15·6	7·1	91·6	15·1	8·7	114	0	+14·7	
27/11/59	1·66	4·91	1,780	7·5	15·2	7·1	91·6	14·8	7·2	114	0	+14·7	
28/11/59	1·52	5·22	1,520	9·2	20·2	5·8	89·5	15·1	8·7	114	+2·1	+13·2	H.G.H. 20 mg.
29/11/59	1·49	5·12	1,740	5·4	19·5	5·8	89·5	14·8	7·2	114	+2·1	+13·2	,, 10 ,,
30/11/59	1·61	5·21	1,290	5·7	17·8	5·8	89·5	15·1	8·7	114	+2·1	+13·2	,, 10 ,,
1/12/59			1,770	6·2	19·0	5·5	86·4	14·8	7·2	114	+2·7	+17·8	
2/12/59	1·62	5·10	1,690	6·5	17·4	5·5	86·4	15·1	8·7	114	+2·7	+17·8	
3/12/59			1,670	7·5	16·1	5·5	86·4	14·8	7·2	114	+2·7	+17·8	
4/12/59			1,840	7·5	17·0	7·3	92·8	15·1	8·7	114	−0·3	+13·3	
5/12/59			1,885	8·1	14·6	7·3	92·8	14·8	7·2	114	−0·3	+13·3	
6/12/59			1,800	8·5	16·6	7·3	92·8	15·1	8·7	114	−0·3	+13·3	
Case 3													
11/7/59	2·00	5·49	2,020	5·7	13·7			16·5	8·6	114			
12/7/59	1·81	4·91	2,340	5·4	14·0	13·2	135	17·2	10·0	114	−1·9	−25·2	
13/7/59	2·00	4·79	2,270	5·9	12·4	13·2	135	16·5	8·6	114	−1·9	−25·2	
14/7/59	2·00	4·81	2,840	5·8	13·5	13·2	135	17·2	10·0	114	−1·9	−25·2	
15/7/59	1·96	4·85	2,150	4·9	13·0	12·0	122	16·5	8·6	114	−0·9	−12·4	
16/7/59			2,490	5·4	14·0	12·0	122	17·2	10·0	114	−0·9	−12·4	
17/7/59	1·82	4·98	1,790	6·6	13·4	12·0	122	16·5	8·6	114	−0·9	−12·4	
18/7/59			1,340	6·8	12·1	12·5	129	17·2	10·0	114	−2·1	−18·5	
19/7/59	1·98	4·84	1,670	6·7	12·5	12·5	129	16·5	8·6	114	−2·1	−18·5	
20/7/59			2,380	6·9	13·1	12·5	129	17·2	10·0	114	−2·1	−18·5	
21/7/59	1·72	5·31	1,460	4·1	8·8	9·3	108	16·5	8·6	114	+3·0	+3·2	B.G.H.* 10 mg.
22/7/59	1·67	5·32	2,010	4·0	10·0	9·3	108	17·2	10·0	114	+3·0	+3·2	,, 20 ,,
23/7/59	1·77	5·19	2,540	5·1	16·8	9·3	108	16·5	8·6	114	+3·0	+3·2	
24/7/59			2,360	4·7	14·2	10·4	122	17·2	10·0	114	+1·0	−12·8	
25/7/59	1·79	5·06	2,140	5·5	13·3	10·4	122	16·5	8·6	114	+1·0	−12·8	
26/7/59			2,420	6·5	15·9	10·4	122	17·2	10·0	114	+1·0	−12·8	
Case 4													
19/2/60			1,080	5·5	6·1	9·4	14	14	7·4	10	−0·9	−2·3	
20/2/60			1,370	6·0	5·7	9·4	14	14	8·6	10	−0·9	−2·3	
21/2/60			1,050	4·9	6·5	9·4	14	14	7·4	10	−0·9	−2·3	
22/2/60			1,070	5·1	4·8	11·0	18	14	8·6	10	−2·1	−5·2	
23/2/60			1,080	5·6	5·2	11·0	18	14	7·4	10	−2·1	−5·2	
24/2/60			1,100	5·3	6·1	11·0	18	14	8·6	10	−2·1	−5·2	
25/2/60			820	5·9	8·6	12·0	18	14	7·4	10	−5·9	−6·2	H.G.H. 10 mg.
26/2/60			1,360	6·3	9·8	12·0	18	14	8·6	10	−5·9	−6·2	
27/2/60			850	4·6	5·6	12·0	18	14	7·4	10	−5·9	−6·2	
28/2/60			1,040	6·0	6·9	7·7	14	14	8·6	10	+0·1	−2·8	
29/2/60			1,320	6·7	7·3	7·7	14	14	7·4	10	+0·1	−2·8	
1/3/60			1,350	6·0	6·9	7·7	14	14	8·6	10	+0·1	−2·8	
2/3/60			1,060	6·4	6·4			14	7·4	10			
3/3/60			660	4·6	4·6	7·3	17	14	8·6	10	+0·5	−4·7	B.G.H.* 40 mg.
4/3/60			1,180	7·4	6·1	7·3	17	14	7·4	10	+0·5	−4·7	
5/3/60			1,400	6·5	6·9	7·3	17	14	8·6	10	+0·5	−4·7	
6/3/60			1,300	6·9	6·4	6·6	22	14	7·4	10	+0·8	−10·1	
7/3/60			1,680	6·0	6·0	6·6	22	14	8·6	10	+0·8	−10·1	
8/3/60			1,800	6·9	5·4	6·6	22	14	7·4	10	+0·8	−10·1	

* Chymotrypsin-treated bovine growth hormone.

TABLE II—*continued*

Date	Plasma Mg	Plasma Ca	Urine Vol.	Urine Mg	Urine Ca	Faeces Mg	Faeces Ca	Diet Mg	Diet Ca	Supplement Ca	Balance Mg	Balance Ca	Remarks
	mEq l.		ml. Day	mEq Day									
						Case 5							
7/4/60			2,560	5·3	2·7			11	5·7	14			
8/4/60			2,250	4·5	3·6	11	45	14	5·7	14	−3·2	−28·9	
9/4/60			2,300	5·5	3·3	11	45	11	5·7	14	−3·2	−28·9	
10/4/60			2,590	5·7	3·9	11	54	14	5·7	14	−3·2	−28·9	
11/4/60			2,070	4·6	2·7	9·8	25	11	5·7	14	−2·8	−7·9	
12/4/60			1,620	4·7	2·1	9·8	25	14	5·7	14	−2·8	−7·9	
13/4/60			2,800	5·6	3·1	9·8	25	11	5·7	14	−2·8	−7·9	
14/4/60			1,610	4·2	4·2	10	25	14	5·7	14	−1·1	−10·3	H.G.H. 10 mg.
15/4/60			1,890	4·0	4·9	10	25	11	5·7	14	−1·1	−10·3	
16/4/60			2,060	4·1	6·0	10	25	14	5·7	14	−1·1	−10·3	
17/4/60			2,560	4·5	6·4	12	25	11	5·7	14	−5·3	−10·4	
18/4/60			2,170	3·8	4·7	12	25	14	5·7	14	−5·3	−10·4	
19/4/60			2,340	4·6	4·2	12	25	11	5·7	14	−5·3	−10·4	
20/4/60			1,910	4·8	4·8	11	26	14	5·7	14	−2·9	−11·2	B.G.H.* 10 mg.
21/4/60			3,050	6·1	6·1	11	26	11	5·7	14	−2·9	−11·2	
22/4/60			1,710	3·9	3·9	11	26	14	5·7	14	−2·9	−11·2	
23/4/60			2,440	4·9	4·9	14	26	11	5·7	14	−6·2	−11·0	
24/4/60			1,640	3·7	4·3	14	26	14	5·7	14	−6·2	−11·0	
25/4/60			2,420	4·1	4·9	14	26	11	5·7	14	−6·2	−11·0	
						Case 6							
3/10/59			3,220	1·6	2·3	8·6	10·6	13·7	7·5	4	+2·9	−1·9	
4/10/59			5,080	2·5	3·3	8·6	10·6	13·3	7·5	4	+2·9	−1·9	
5/10/59			4,600	2·3	2·8	8·6	10·6	13·7	7·5	4	+2·9	−1·9	
6/10/59			5,100	2·5	3·6	7·5	9·7	13·3	7·5	4	+3·3	−0·6	
7/10/59	1·71	5·36	4,240	3·1	2·1	7·5	9·7	13·7	7·5	4	+3·3	−0·6	
8/10/59	1·62	5·42	4,330	2·2	1·6	7·5	9·7	13·3	7·5	4	+3·3	−0·6	
9/10/59	1·45	5·61	3,620	4·5	5·2	6·0	8·1	13·7	7·5	4	+2·7	−1·1	H.G.H. 5 mg.
10/10/59	1·31	5·31	2,090	3·1	2·7	6·0	8·1	13·3	7·5	4	+2·7	−1·1	
11/10/59	1·62	5·50	2,650	7·2	5·7	6·0	8·1	13·7	7·5	4	+2·7	−1·1	
12/10/59	1·62	5·20	3,600	5·1	4·9	8·5	7·8	13·3	7·5	4	+0·6	−1·0	
13/10/59			4,400	3·1	5·2	8·5	7·8	13·7	7·5	4	+0·6	−1·0	
14/10/59			3,680	4·8	4·0	8·5	7·8	13·3	7·5	4	+0·6	−1·0	
						Case 7							
7/10/59	2·00	5·00	900	4·4	6·1	9·2	37	15·4	8·2	38	+1·0	+2·4	
8/10/59	1·94	5·10	770	5·2	6·2	9·2	37	15·1	8·5	38	+1·0	+2·4	
9/10/59	1·94	5·00	800	5·8	8·3	9·2	37	15·4	8·2	38	+1·0	+2·4	
10/10/59			920	5·9	7·6	9·3	32	15·1	8·5	38	−0·7	+6·4	
11/10/59	1·86	4·90	1,410	7·5	8·3	9·3	32	15·4	8·2	38	−0·7	+6·4	
12/10/59	1·33	5·21	1,070	11·0	12·8	7·1	30	15·1	8·5	38	−1·0	+6·4	H.G.H. 10 mg.
13/10/59	1·57	5·28	1,370	10·4	10·4	7·1	30	15·4	8·2	38	−1·0	+6·4	
14/10/59	1·86	5·49	1,070	5·9	7·8	7·1	30	15·1	8·5	38	−1·0	+6·4	
15/10/59			850	5·0	5·5	8·4	23·5	15·4	8·2	38	+1·0	+15·9	
16/10/59	1·88	5·00	1,300	6·1	8·1	8·4	23·5	15·1	8·5	38	+1·0	+15·9	
17/10/59			810	6·6	7·4	8·4	23·5	15·4	8·2	38	+1·0	+15·9	

* Chymotrypsin-treated bovine growth hormone.

Discussion

Studies on the relationship between calcium metabolism and growth hormone have previously been mainly concerned with the skeletal changes accompanying acromegaly (Scriver and Bryan, 1935; Bauer and Aub, 1941). Albright and Reifenstein (1948) attributed these changes to one of five possible causes: (1) an accompanying hyperparathyroidism; (2) an accompanying excessive formation of anti-anabolic hormones from the adrenal cortex; (3) excessive formation of thyrotrophic hormone; (4) increase in protoplasmic mass, requiring a higher nitrogen intake; bone matrix losses because of low priority rating; or (5) associated hypogonadism.

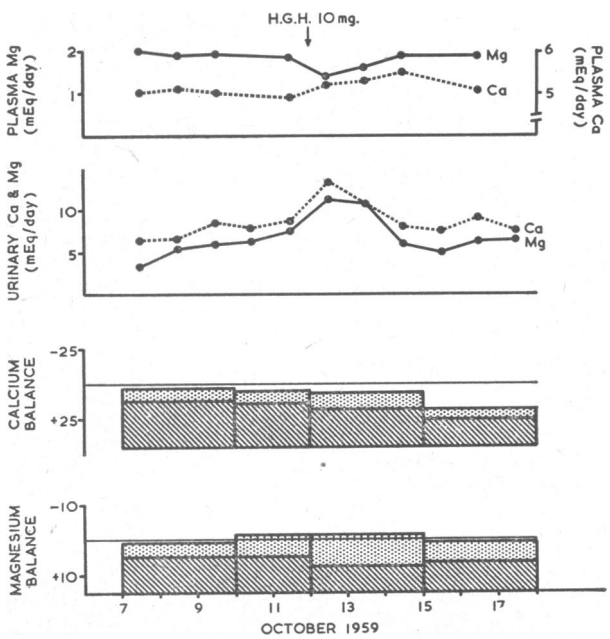

FIG. 2.—Results of injection of growth hormone.

Hyperparathyroidism may accompany acromegaly as part of polyglandular adenomatous conditions affecting more than one endocrine gland (Perlman, 1944). However, the elevation of plasma phosphorus level in

TABLE III.—*Effects of Growth Hormone on Calcium and Magnesium Metabolism*

Case No.	Diagnosis	Magnesium Plasma	Magnesium Faeces	Magnesium Urine	Magnesium Balance	Calcium Plasma	Calcium Faeces	Calcium Urine	Calcium Balance
1	Acromegaly* ..	−	−	0	+	+	−	+	−
2	Osteoporosis ..	−	−	+−	+	0	0	+	0
3	Pit. ablation (for malignancy) ..	−	−	?−	+	+	−	−+	+
4	Pit. dwarfism ..		−	0	+		−	+	?+
5	Hypopituitarism		?−	+	?+		0	+	−
6	Pit. dwarfism ..	−	−	+	0	0	−	+	0
7	,, ,, ..	−	−	+	0	+	−	+	+

* Effect of growth hormone here is judged by the effect of withdrawal by pituitary ablation.

− Lowering of plasma, urinary, or faecal content, increased loss or diminished retention in balance.

+ Rise in plasma, urinary, or faecal content, diminished loss or increased retention in balance.

0 = No effect detected. ? = Doubtful effect. +−=+ followed by −.

most cases of acromegaly (Bauer and Aub, 1941; Albright and Reifenstein, 1948) is against demineralization being due to hyperparathyroidism.

The demineralization of bone produced by excessive formation of adrenocortical hormones is accompanied by increased loss of calcium in faeces (Hanna, 1960). This is incompatible with the findings in acromegaly reported here as well as with the findings of Scriver and Bryan (1935).

Excessive formation of thyrotrophic hormone can be excluded as a cause of skeletal changes; most cases of acromegaly show a low B.M.R. (Bauer and Aub, 1941).

There is no evidence that either protein starvation or hypogonadism plays a part in the skeletal changes in acromegaly. Furthermore, both conditions are hardly likely to produce increased absorption of calcium from the gut.

The findings reported here show that both administration of a fairly pure growth hormone preparation and the excessive endogenous secretion of growth hormone in acromegaly produce results which resemble the effects of administration of vitamin D (Hanna, 1961), which were: an increased absorption of calcium and magnesium from the gut, a rise of the plasma level of calcium, a lowering of the plasma level of magnesium, and an increase in the urinary loss of calcium and magnesium.

The finding of a rise in plasma citrate level in acromegalic subjects and after the administration of growth hormone emphasizes the similarity between the effects of growth hormone and vitamin D (Harrison, Joplin, Hartog, and Fraser, 1960; Henneman and Henneman, 1960).

This similarity raises the question: Is this array of changes in magnesium and calcium metabolism produced by one biochemical process? If so, it may well be that both vitamin D and growth hormone (and possibly other substances) produce an effect on one target biochemical process, producing these effects, beside their other metabolic activities. A study of the influence of these substances on citrate metabolism and of the role of citrate in the absorption and utilization of magnesium and calcium may provide a clue to the answer to this question.

The skeletal changes accompanying some cases of acromegaly may be explained in the following way: Excessive formation of growth hormone leads to increased intestinal absorption and urinary loss of calcium. The effect on urinary calcium is not dependent upon the increased intestinal absorption. If the calcium intake is sufficient, no negative balance will result. If the calcium intake is limited, urinary loss will outweigh intestinal absorption and a negative calcium balance is produced, leading to skeletal changes. This state of affairs is not unique for acromegaly; it also applies to treatment with vitamin D, hyperparathyroidism, idiopathic hypercalciuria, and sarcoidosis (Albright and Reifenstein, 1948; Henneman, Benedict, Forbes, and Dudley, 1958; Jackson and Dancaster, 1959).

It is perhaps worth noticing that the urinary calcium excretion of the hypopituitary subjects presented here fall on the lower side of the wide range reported by Knapp (1947), and that the reverse is true for the acromegalic subject. Knapp suggested that the urinary calcium is constant for one subject under stable physiological conditions and that it depends upon "some factor or factors, presumably endocrine, which are peculiar to each individual." The findings reported here suggest that growth hormone is one of these factors.

Summary

Trial of growth hormone preparations as well as studies on an acromegalic subject showed that growth hormone produced effects on calcium and magnesium metabolism reminiscent of those produced by vitamin D. These were: (*a*) an increased absorption of both Mg and Ca from the gut, (*b*) an increased urinary excretion of both elements, (*c*) a lowering of plasma magnesium, and (*d*) a rise in plasma calcium.

It is suggested that both substances may influence calcium and magnesium metabolism by their common influence on one process, possibly citrate metabolism.

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New premises were opened recently for the Ipswich Medical Library at the Anglesea Road Wing of the Ipswich and East Suffolk Hospital. The library was founded in 1953 "to promote study and research by providing reference facilities and the availability on loan of the commoner medical journals." Its membership is open to the medical staffs of all hospitals, to the general practitioners, and public health staff in the area. The new premises consist of a detached suite of rooms in a quiet corner of the general hospital. They include a large reading-room, a recess for the librarian situated off the main reading-room, a discussion room, ample cloakroom facilities, and a wide corridor for the display and demonstration of clinical material. The rooms have been sound-proofed wherever possible. Ample parking facilities are available near by. The services provided by the library include a large reference section, a film index, borrowing facilities, and 52 current medical journals. Bibliographies can be prepared. There is a photo-copying service, a voluntary panel of translators, and a historical section. The library's premises are large enough for the holding of clinical meetings.