Nutrition of the cat

5*. The influence of calcium and iodine supplements to a meat diet on the retention of nitrogen, calcium and phosphorus

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Characteristic behavioural changes were observed in kittens receiving a diet of raw heart for 6–8 weeks (Greaves, Scott & Scott, 1958). This diet contained high proportions of protein and fat, but little calcium. Typical osteoporosis led, after longer periods, to a condition similar to osteitis fibrosa, and then to greenstick fractures of limb bones and collapse of vertebral bodies; these changes could be prevented by adding sufficient Ca salt to the diet to raise the Ca:P ratio from about 0·05 to 1·0. The possibility of vitamin D deficiency in these experiments was considered, but there was no histological evidence of rickets or osteomalacia, and orally or parenterally administered ergocalciferol was without effect on the onset or course of the syndrome; moreover, Gershoff, Legg, O'Connor & Hegsted (1957) have shown the vitamin D requirement of kittens to be small. However, marked improvement occurred in the behaviour of dystrophic kittens when they were given supplementary iodine which, administered from the beginning of the experiment, delayed the onset of signs of Ca deficiency referable to the skeleton (Scott, Greaves & Scott, 1961).

The experiments described in this paper were undertaken to determine the effect of supplementary Ca and I on the retentions of Ca and P in growing kittens and in fully adult cats. Nitrogen balance was determined at the same time, providing a standard of protein retention. Similar determinations were made on normal growing kittens receiving the stock diet used in this laboratory, to establish the pattern of retention and excretion.

EXPERIMENTAL

Animals and cages. By the technique described previously (Greaves & Scott, 1960) balance studies were carried out over 8- (occasionally 4-) day periods on seven littermate weaned kittens and four adult cats, either housed in conventional rabbit metabolism cages, or trained to use enamel trays fitted with a sloping perforated false bottom of Perspex (Greaves, 1959), placed in the cat cages normally used in this laboratory (Scott, 1952).

Diets. The kittens were fed *ad lib.* and received distilled water to drink. A weighed ration of food was presented once daily; the uneaten remnants were dried before

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reweighing to avoid error by loss of moisture. Samples of food were collected for analysis.

The stock diet was the afternoon meal, described by Dickinson & Scott (1956), of two-thirds cooked potatoes mixed with one-third cooked fish, supplemented daily with small amounts of dried yeast and grass powder.

The experimental diet was raw minced sheep's heart imported frozen from Australia (for details of constitution see Scott *et al.* 1961). Supplementary calcium carbonate (A.R.) was thoroughly mixed into the daily feed at the rate of 0·5 g/100 g wet, minced heart. The I supplement was administered orally as an aqueous solution of KI at the rate of 100μg I/day.

Procedure. It was based on the technique used by Greaves & Scott (1960). Urine was collected into distilled water; together with washings, the total daily volume was made up to 500 ml and 10 ml were transferred into a pool of daily samples. Similarly, every 24 h collection of faeces was homogenized with water to a fluid consistency and one-tenth of the homogenate was transferred to the pool of daily samples. Pooled samples of urine and faeces were kept under toluene in a refrigerator. With the unsupplemented heart diet difficulty was experienced in marking the beginning and end of the balance periods owing to the erratic production and small bulk of the faeces, whose black colour completely obscured the conventional carmine marker used effectively with the stock diet.

The pooled daily samples were analysed for their content of N, Ca and P. N was determined on duplicate 5 ml samples of urine and 10 ml samples of faeces, by the macro-Kjeldahl method. Good agreement was found between duplicates. Ca was estimated by the method of Baron & Bell (1957); protein was precipitated by sodium tungstate and P by the morpholine nitrate–nitric acid reagent; Ca was determined in the supernatant liquid by titration against ethylenediaminetetraacetic acid with Ca-thymophthalein as indicator. Total P was determined by the method of Allen (1940); digestion was carried out with nitric acid followed by perchloric acid, a blue colour was developed with ammonium molybdate and amidol (2,4-diamophenol hydrochloride) and read on a M.R.C. Grey Wedge Photometer.

Expt 1. *Retentions on stock diet.* Litter-mate kittens A_1 and A_2, reared on the full stock diet at both morning and afternoon meals, were placed on the afternoon meal only when they were 28 weeks old and balances were determined.

Expt 2. *Adaptation to low-Ca diet and effect of supplements.* Two well-grown litter-mate kittens B_1 and B_2, reared on a mixed diet under domestic conditions, were placed on raw heart alone at 12 weeks of age. Subsequently they were given:

weeks 1–11, raw heart only;
weeks 12–15, raw heart and I;
weeks 16 and 17, raw heart only;
weeks 18–21, raw heart and $CaCO_3$.

Ca, P and N retentions were measured during weeks 9, 10, 14, 17 and 21.

Expt 3. *Effect of withdrawal of Ca and supplementation with I.* Litter-mate kittens C_1, C_2 and C_3 were reared on raw heart supplemented with $CaCO_3$ from the time they

began to eat; their parents were also reared and maintained on this diet. From 10 weeks of age they were given:

week 1, raw heart with $CaCO_3$ (control period, rearing diet continued);

weeks 2–5, raw heart only (after 2 weeks C_2 became ill and $CaCO_3$ was again added to its diet for the remainder of the experiment. C_3 died after $4\frac{1}{2}$ weeks);

week 6, C_1 received raw heart and I, and C_2 had the same, with $CaCO_3$ added.

Ca, P and N retentions were measured during weeks 1, 2, 4 and 6.

Expt 4. *Retention in adult cats.* Cats D_1 and D_2, E_1 and E_2 were two pairs of littermate adults that had been maintained for several months on raw heart supplemented with Ca. Balances, on this diet, were carried out on D_1 and E_1 when they were 9 and 10 months, respectively; Ca supplements were withdrawn from D_2 and E_2 and balances were carried out on them at 12 and 14 months of age.

RESULTS

Expt 1. Table 1 shows the proximate composition of heart compared with that of the stock diet; it contained only 10% more protein but the relative amounts of carbohydrate and fat were reversed. The P contents of the diets were similar, but in the stock diet the Ca:P ratio was 0·99, whereas in heart it was 0·07. The marked difference in I content was due to the presence of sea fish in the stock diet.

Table 1. *Proximate composition (per 100 g dry weight) of the diets*

Constituent	Raw heart	Stock diet	Supplement
Protein (g)	50	77	—
Fat (g)	40	5	—
Carbohydrate (g)	7	47	—
Ca (mg)	40	850	800
P (mg)	620	860	—
Ca:P ratio	0·07	0·99	1·35
Iodine (μg)	10–80*	400*	100†

Mean water content: raw heart 66%, stock diet 77%.
* Determined for us by the Chilean Iodine Education Bureau, Bishopsgate, London, E.C. 2.
† Per cat per day.

Patterns of excretion and retention of Ca and P on the stock diet can be seen (Table 2, and Fig. 1) to have followed N intake, which in its turn is a measure of food intake. N retention was related to weight gain, but even without any weight increase during the balance period there was an apparent retention of N.

Expt 2. On the raw-heart diet loss of Ca in urine and faeces, resulting in a negative Ca balance, was maintained even when the kittens were fed on the diet for a relatively long period (Table 2). After 9 weeks N intake was still high, and retentions of N were appreciable, but retention of P by kitten B_1 was low, loss occurring chiefly in the urine; similar results were obtained in a balance carried out in the 10th week (Fig. 2). The addition of I brought about a marked reversal in Ca retention (week 14); balances became positive even on the continuing small intake, excretion diminishing in both urine and faeces. Kitten B_2 showed typical dystrophic signs (pain on handling, lame-

Table 2. *Expts* 1–4. *Mean values for daily intake, excretion and retention of calcium, phosphorus and nitrogen during 8-day balance periods on stock diet or on raw heart alone or supplemented with iodine or* $CaCO_3$

Diet and week of experiment	Animal No.	Sex	Wt at beginning of test (kg)	Daily gain (g)	Nitrogen (g) Eaten	Nitrogen (g) Excreted Urine	Nitrogen (g) Excreted Faeces	Nitrogen (g) Retained	Calcium (mg) Eaten	Calcium (mg) Excreted Urine	Calcium (mg) Excreted Faeces	Calcium (mg) Retained	Phosphorus (mg) Eaten	Phosphorus (mg) Excreted Urine	Phosphorus (mg) Excreted Faeces	Phosphorus (mg) Retained
Expt 1 (kittens)																
Stock diet	A_1	♀	2·5	0	2·970	1·470	0·140	+1·360	326	1·1	181·0	+143·9	472	24·0	87·5	+360·5
	A_1	♀	2·6	+21	6·620	2·340	0·344	+3·936	725	7·7	197·0	+520·3	1050	153·1	120·0	+776·9
	A_2	♀	2·0	+10	4·300	2·187	0·336	+1·777	471	5·0	148·0	+318·0	682	87·5	96·0	+498·5
Expt 2 (kittens)																
Heart, week 9	B_1	♀	2·4	− 5·4	4·125	2·400	0·120	+1·595	15·0	23·4	20·8	− 29·2	300	240·0	19·0	+ 41
	B_2	♂	2·2	+ 2·6	3·750	2·235	0·098	+1·417	14·0	14·1	16·6	− 16·7	280	143·7	19·5	+116·8
Heart + I,	B_1	♀	2·6	+ 7·7	5·125	3·040	0·080	+2·005	18·3	1·3	4·2	+ 12·8	365	168·7	15·4	+180·9
week 14	B_2	♂	2·2	−19·7	3·750	2·300	0·080	+1·370	14·0	3·8	4·8	+ 5·4	305	187·5	16·9	+100·6
Heart, week 17	B_1	♀	2·8	−15·9	3·750	2·197	0·100	+1·453	14·0	1·9	24·3	− 12·2	280	150·0	12·2	+117·8
	B_2	♂	2·3	− 5·1	4·125	2·600	0·102	+1·423	15·0	3·8	7·5	+ 3·7	300	155·0	8·5	+136·5
Heart + $CaCO_3$,	B_1	♀	2·8	− 7·3	3·000	1·260	0·058	+1·682	176·0	16·3	47·8	+111·9	220	60·0	32·6	+127·4
week 21	B_2	♂	2·4	+ 9·3	6·583	1·500	0·060	+5·023	305·0	7·5	72·3	+225·2	481	78·0	42·8	+360·2
Expt 3 (kittens)																
Heart + $CaCO_3$,	C_1	♀	1·1	+20·4	4·908	1·850	0·072	+2·986	285·5	0·0	35·6	+249·0	357	78·8	9·0	+269·2
week 1	C_2	♂	1·2	+18·6	5·005	1·755	0·096	+3·154	291·3	4·4	61·8	+225·1	364	62·5	8·5	+293·0
	C_3	♂	1·3	+29·0	5·013	1·670	0·072	+3·271	291·6	3·1	100·5	+188·0	365	110·0	8·6	+246·4
Heart, week 2	C_1	♀	1·4	+12·3	5·995	1·298	0·068	+4·629	21·8	1·9	5·6	+ 14·3	436	68·8	6·0	+361·2
	C_2	♂	1·5	+ 5·7	5·528	1·088	0·096	+4·344	20·1	10·9	21·7	− 12·5	402	68·8	5·6	+327·6
	C_3	♂	1·5	+11·1	5·750	1·710	0·092	+3·948	20·9	1·3	15·0	+ 4·6	418	192·3	17·9	+207·8
Heart, week 4	C_1	♀	1·5	−17·1	3·885	2·343	0·153	+1·389	14·0	9·8	31·9	− 27·7	282	95·4	5·0	+181·6
	C_3	♂	1·6	−20·7	3·891	1·500	0·083	+2·308	11·9	5·6	24·4	− 18·1	238	126·3	5·0	+106·7
Heart + I, week 6	C_1	♀	1·5	+17·3	3·625	1·433	0·086	+2·106	13·3	1·6	5·4	+ 6·3	268	18·8	4·3	+244·9
Heart + $CaCO_3$, week 4	C_2	♂*	1·5	−25·8	3·625	1·255	0·270	+2·100	280·0	7·1	110·3	+162·6	349	68·7	35·0	+245·3
Heart + $CaCO_3$ + I, week 6	C_2	♂	1·3	+11·3	7·040	1·425	0·518	+5·097	410·0	3·8	102·5	+303·7	512	135·0	93·8	+283·2
Expt 4 (adult cats)																
Heart + $CaCO_3$	D_1	♂	5·9	+16·0	4·640	2·980	0·080	+1·580	307·0	13·8	165·2	+128·0	339	312·0	44·3	− 17·3
	E_1	♂	4·2	−12·3	4·700	2·012	0·082	+2·606	348·0	12·7	163·0	+172·3	343	153·0	55·5	+134·5
Heart	D_2	♀	3·5	0	6·880	2·550	0·031	+4·299	25·4	3·8	39·4	− 17·8	502	134·0	13·9	+354·1
	E_2	♀	2·4	+ 1·5	4·260	2·143	0·460	+1·657	15·7	6·6	49·2	− 40·1	311	70·0	16·0	+225·0

Cats having same distinguishing letter are litter-mates. * 4-day balance only.

ness and inability to maintain normal posture) in week 10. This kitten had a remission when given I, with marked improvement of locomotion and temper. In kitten B_1, P and N retention increased after a rise in food intake, but loss of N in urine was exceptionally high. On omitting I, in week 17, kitten B_1 again went into negative Ca balance,

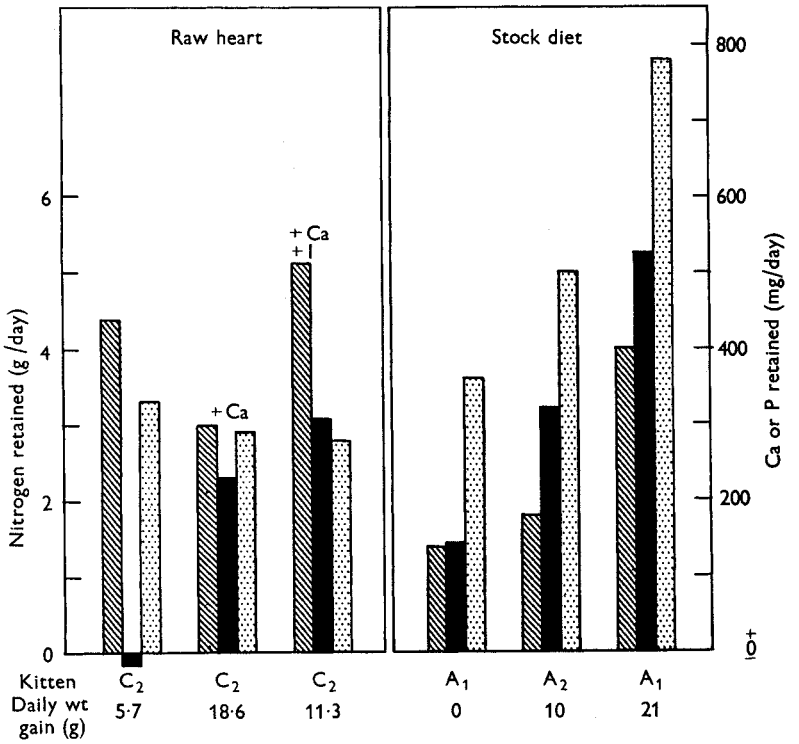

Fig. 1. Expt 3. Retention of nitrogen (cross-hatched), calcium (solid) and phosphorus (stippled) by kittens fed on raw heart, alone or supplemented with Ca or with Ca and I, compared with that of a kitten in Expt 1 on the stock diet, at different rates of weight gain.

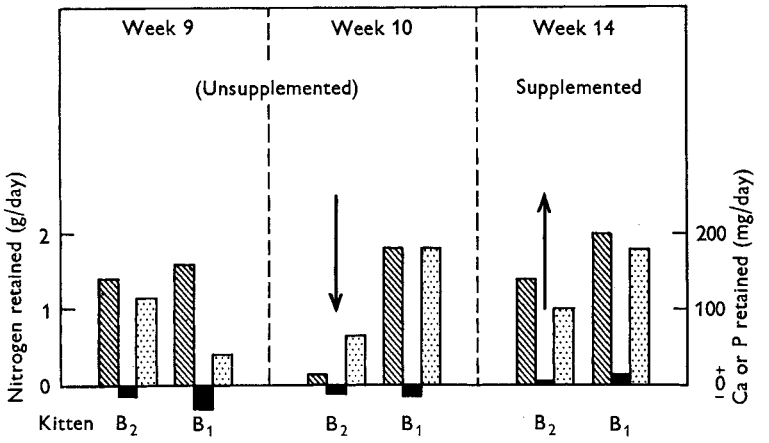

Fig. 2. Effect of an iodine supplement (100 μg/day) on the retention by kittens of nitrogen (cross-hatched), calcium (solid) and phosphorus (stippled). ↓ dystrophic signs; ↑ remission.

owing to loss in the faeces, but kitten B_2 remained in positive Ca balance. Kitten B_1, which showed a steady decline in weight, now developed dystrophic signs, but kitten B_2 seemed to carry over the improvement made when given I to the period when it was given additional Ca. It grew rapidly and retained nearly as much Ca on the supplemented heart diet as kitten A_2 on the stock diet, although N retention was greater.

P intake and retention remained reasonably constant in both kittens, throughout the experiment. The urinary excretion of P was reduced on adding Ca to the diet (week 21), while during the same period loss of P in faeces increased; a similar reduction in urinary N was also apparent.

Expt 3. In Expt 3 younger kittens reared on heart supplemented with Ca showed retentions similar to those of kittens on the stock diet (Table 2, cf. Expt 3, week 1, and Expt 1). Withdrawal of the Ca supplement resulted in a dramatic fall in Ca retention, kitten C_2 going into negative balance immediately and becoming seriously ill (with vomiting and diarrhoea) after 2 weeks, when it was again given Ca. Kittens C_1 and C_3 were in severe negative balance by week 4, with increased output of Ca in urine and faeces accompanied by lowered P and N retention; kitten C_3 died after $4\frac{1}{2}$ weeks, showing general enteritis and septicaemia at autopsy. The performance of these two kittens at week 4 contrasted sharply with that of C_2, which recovered after resupplementation of its diet with Ca; in spite of overall weight loss in the balance period, this animal was in positive balance for all three elements, although losses of Ca and P in urine and faeces were greater than in week 1. Kitten C_2 was still scouring; although its condition had greatly improved, the high values for faecal Ca, P and N suggest failure of absorption rather than raised excretion.

When kittens C_1 and C_2 were given I, both improved on their performance in week 4. Kitten C_1, still on raw meat without additional Ca, gained weight, with raised P and N retention, though eating about the same amount of food as in week 4. Kitten C_2, receiving both Ca and I supplements, showed exceptionally high intakes and retentions; absorption of Ca increased from 61 to 75% of intake and urinary loss was reduced by half. This was the only occasion when Ca retention exceeded that of P.

Expt 4. Comparison of the retentions by cats D_1 and E_1 with those by D_2 and E_2 (Table 2) demonstrates the effect on adult cats of withdrawing the Ca supplement; D_2 and E_2 went into negative balance, owing to loss of endogenous Ca in the faeces.

DISCUSSION

On the stock diet retentions of N, Ca and P were related to the amount of growth in the balance period. In the first period kitten A_1 did not gain weight and retained 0·55 g N/kg body-weight daily, rather less than the amount, 0·8 g/kg, required by adult cats for equilibrium on a mixed diet (Greaves & Scott, 1960), but similar to the value of 0·5 g/kg found by Miller & Allison (1958) with N-free diets. This maintenance level was associated with retentions of Ca and P indicative of continued skeletal growth in the absence of an overall gain in weight. Even when allowance is made for losses other than in urine and faeces, N retentions in kittens gaining weight were high. The amount of Ca in the faeces indicated that skeletal stores were well filled on the stock diet

(Henry & Kon, 1953; Hansard & Plumlee, 1954); at the same time urinary Ca losses were small and retentions high. Well-fed kittens incorporate relatively large amounts of Ca into the skeleton; calculations, based on the whole-carcass analysis of kittens at different ages made by Spray & Widdowson (1950), show that kittens must accumulate at least 140 mg Ca a day between the ages of 10 and 25 weeks (on the assumption that body-weights are 1·0 and 2·5 kg). The mean daily retention of Ca in these experiments was 190 mg/kg body-weight in all balance periods in which adequate amounts of Ca were eaten. However, this value may be high because the periods used often succeeded periods of depletion. Nevertheless, the daily requirement of the kitten relative to body-weight is much greater than that of adolescent children. Absorption and excretion of P in growing kittens on a balanced stock diet resembles that of normal well-fed human beings, for whom it has been shown that urinary P forms about half of the total P excreted (Hollinger & Pattee, 1956).

Striking differences are apparent when the patterns of absorption, excretion and retention from the stock diet are compared with those from the heart diet (Table 2 and Fig. 1). The high digestibility of the meat diet, in spite of the high fat content, resulted in low values for faecal N and P. When supplementary Ca was given, N retentions were high and remained so for the first week or two after the supplement was discontinued, during which period body-weight also increased. Kittens were not able to maintain themselves in Ca equilibrium for long on raw heart, going into Ca deficit in 1 or 2 weeks; adults immediately went into negative balance. Where they were transferred from a diet high (0·84%) to one low (0·04%) in Ca, losses of Ca occurred in both urine and faeces; there was no evidence of adaptation to the low Ca intake on raw heart alone. In the absence of isotopic studies, it was not possible to determine exactly how much of the faecal Ca was due to endogenous loss and how much to failure of absorption, but on heart alone Ca excreted in the faeces frequently exceeded Ca intake, indicating endogenous loss. Henry & Kon (1953) and Hansard & Plumlee (1954) found that the proportion of Ca intake retained by rats depended on the level of body stores, being highest when the stores were low. Long-standing Ca deficiency, from eating heart alone, depleted skeletal stores and resulted in kittens retaining 94% of the Ca intake when given a Ca supplement as against 65% retention on the stock diet. Given an I supplement, the kittens showed a striking adaptation to the low Ca intake, endogenous losses being so reduced in both urine and faeces that small positive balances were achieved. This finding confirmed the impression that the clinical improvement obtained in earlier experiments with I was at least partly due to improved Ca retention.

Clinical and experimental observations (reviewed by Scott *et al.* 1961) over the past century have associated osteoporotic or so-called rachitic changes in bone with hyperplastic changes in the thyroid glands of man and animals. It is sufficient to note here that in cats exhibiting the disease called '*osteogenesis imperfecta*', both from the London area and from Salisbury, Rhodesia, severe osteoporosis has always been associated with marked hypertrophy (increased weight), hyperaemia and histological hyperplasia of the thyroid glands, and with conspicuous parathyroid glands. Aub, Bauer, Heath & Ropes (1929) showed that hyperthyroidic patients, or normal individuals given thyroid extracts, had an abnormally high urinary Ca output. Reilly (1940) found that Ca and

P balance studies on sixty-two children with thyrotoxicosis indicated a demineralization of the skeleton and teeth, which varied directly with the duration and severity of the disease; 70% of the mineral loss was in the urine, 30% in the faeces. The disease 'waxed and waned' in all the children, but was clinically at its most severe during normal growth spurts, just before and during the growth of puberty. Using normal mixed diets, Cooke, Nassim & Collins (1959) found a negative Ca balance, due to increased faecal Ca, often in excess of intake, and increased urinary Ca, in all of nine patients, of both sexes and various ages, with proven thyrotoxicosis. Bone biopsy was performed on each, and six showed histological evidence (thin trabeculae) of osteoporosis; the other three had been thyrotoxic for a shorter time. Calciferol (orally or parenterally), a high Ca intake and anabolic steroids, were without effect on the Ca balance. Anabolic steroids, however, increased the already positive N balances. Diarrhoea was a common presenting sign in these patients, which seems to us a point of interest, since in our experiments five out of twenty-five kittens placed on the heart diet died of enteric infection (Scott *et al.* 1961), and difficulty was experienced with this condition during Expt 3 described here (see p. 78). As a result of isotopic studies, Krane, Brownell, Stanbury & Corrigan (1956) suggested that in thyrotoxicosis both destruction and reformation of bone proceed at an increased rate, but it would seem that urinary and faecal excretion of Ca also proceeds at an excessive rate in thyrotoxicosis. Thus a reduction in thyroid activity might reduce the loss of Ca. In fact, Cooke *et al.* (1959) found that treatment with methylthiouracil not only relieved the thyrotoxicosis but brought all their patients into positive Ca balance, without any alteration of the diet. It is possible that the I supplements in our experiments were acting in this manner, since Goldsmith & Eisele (1956) showed that increase in serum iodide slows the rate of release of formed hormone from the human gland in thyrotoxicosis (Graves's disease) probably by interfering with the action of pituitary thyrotrophic hormone on the thyroid. However, on the histological and other evidence available (Scott *et al.* 1961) it is not possible to be certain whether the thyroid was in a state of hyperactivity in the cats or whether it was showing compensatory hypertrophy due to I deficiency or the presence of a goitrogen in meat. The latter is improbable (Greer, 1957), but McRoberts (1958) has shown that high-protein diets may increase I requirements, at least in rats. This seems a possible explanation, since an additional 100μg I daily is sufficient to keep the thyroid histologically normal; however, the fact that the thyroid remains nearly normal in size and structure when the Ca intake is increased by supplementing the heart diet with $CaCO_3$ (Greaves, Scott & Scott, 1959) suggests that there may be a synergistic relationship between the requirements of these elements in the cat.

An increased secretory activity of the parathyroid gland in cats on the raw-heart diet is suggested by the enlargement of the glands observed at autopsy, and the maintenance of a normal or slightly elevated serum Ca after many weeks on the Ca-deficient diet (Scott *et al.* 1961). Dawson, Weidmann & Jones (1957) found a decrease in activity of the endosteal layers of shaft bone in adult cats given 100 U.S.P. units of the parathyroid hormone daily for 9 days. A similar inhibitory effect of the hormone on the formation of new bone in kittens, due to inhibition of the synthesis of adenosine triphosphate,

was observed by Whitehead & Weidmann (1959). As normally fed kittens grew, subperiosteal activity of bone decreased rapidly while endosteal activity increased, until the two surfaces were equally active at 12 weeks of age. In the later phases of growth endosteal activity exceeded that of the periosteum (Weidmann & Rogers, 1958). The coincidence of post-weaning Ca deficiency, resulting from feeding on unsupplemented meat or heart, with the period in which endosteal bone formation is proceeding accounts for the extreme thinness of the cortices of limb bones of kittens kept on these diets for 7 weeks or longer. Subperiosteal bone formation is less affected and the whole bone is therefore normal, or nearly normal, in external circumference and general shape (Scott *et al.* 1961). Growth in length of bones may be depressed, but only in severely affected young kittens in whom cartilage formation is inhibited by excess of parathyroid hormone (Whitehead & Weidmann, 1959).

It is difficult to assess the relative importance of the thyroids and parathyroids in the syndrome caused by feeding on unsupplemented meat. To test whether endocrine activity played a part in the bony changes, the thyroids and parathyroids were excised from a 10-week kitten under anaesthetic. Convulsions in the week after operation were controlled by injections of the parathyroid hormone and calcium gluconate, after which the animal recovered and remained in a satisfactory condition without further support. It was then fed on heart alone (low-Ca diet) for 12 weeks and killed. The bones were radiologically normal, and the cortex of normal width and density, but the kitten had not grown much during the experimental period. Further studies along these lines are in progress.

SUMMARY

1. Intake, and output in urine and faeces, of nitrogen, calcium and phosphorus were measured over 8-day periods in seven kittens and four adult cats fed on raw heart with or without supplements of $CaCO_3$ and KI. Similar balances were carried out with a stock diet for comparison.

2. On the heart diet containing 0·04% Ca (dry weight), kittens and cats went into negative Ca balance, losing Ca in urine and faeces.

3. On supplementation with $CaCO_3$, to give 0·84% Ca in the diet, Ca balances were positive and losses in urine were reduced. The proportion of Ca absorbed depended on the state of the skeletal store of Ca and on the iodine intake.

4. When the heart diet was supplemented with KI, at the rate of 100 μg I/cat daily, losses of Ca were reduced in urine and faeces; even on the low intake, positive Ca balances were obtained.

5. The results are discussed in relation to the part played by the thyroid and parathyroid glands in the regulation of Ca balance.

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