

index was associated with an unduly high incidence of arcus senilis. Again no such association was shown.

In common with other workers (Lindholm, 1960; Rodstein and Zeman, 1963) we have failed to show any close correlation between the presence of an arcus senilis and serum-cholesterol levels. However, all the subjects with a serum-cholesterol level of over 350 mg./100 ml. were noted to have an arcus senilis. It would seem that a substantially elevated serum-cholesterol level contributes to the development of an arcus senilis, but that hypercholesterolaemia is not necessary for its development.

The main conclusion from this study must be that the presence or absence of an arcus senilis in middle-aged men gives no useful clinical guide to the presence of atherosclerosis.

Summary

A comparison of the incidence of arcus senilis was made between middle-aged male post-cardiac-infarction patients and age-matched male patients without clinical or electrocardiographic evidence of occlusive vascular disease. No statistically significant difference in incidence was found. In agreement with previously published observations there was a progressive rise in incidence with advancing age.

Intestinal Absorption of ^{45}Ca in Stone-forming Patients

A. CANIGGIA,*† M.D.; C. GENNARI,* M.D.; L. CESARI,* M.D.

Brit. med. J., 1965, 1, 427-429

With the use of the calcium-balance method an increased absorption of calcium by the intestine has been demonstrated in primary hyperparathyroidism (Albright, Bauer, Claffin, and Cockrill, 1932; Aub, Tibbets, and McLean, 1937; Lichtwitz and Parlier, 1959; Lafferty and Pearson, 1963), in hypercalcaemia of sarcoidosis (Anderson, Dent, Harper, and Philpot, 1954; Henneman, Dempsey, Carroll, and Albright, 1956), and in idiopathic hypercalcaemia of children (Morgan, Mitchell, Stowers, and Thompson, 1956; Stapleton, Macdonald, and Lightwood, 1956). Lichtwitz, de Sèze, Hioco, Miravet, Lanham, and Parlier (1963) were of the opinion that excessive absorption of calcium by the intestine might be the cause of hypercalcaemia in patients with nephrolithiasis. Investigations carried out with the aid of ^{45}Ca and ^{47}Ca have confirmed that in primary hyperparathyroidism intestinal hyperabsorption of calcium is indeed present, and that this disappears after the removal of the parathyroid adenoma (Lichtwitz and Parlier, 1959; Jaworski, Brown, Fedoruk, and Seitz, 1963).

We have made a study of the intestinal absorption of ^{45}Ca in six stone-forming patients, including one patient with a large parathyroid adenoma and von Recklinghausen's disease of bone; we have used the same method as that employed earlier for the study of the intestinal absorption of ^{45}Ca in senile osteoporosis (Caniggia, Gennari, Bianchi, and Guideri, 1963).

Clinical Material and Methods

All the patients studied had multiple renal calculi, while one individual suffered from bilateral nephrocalcinosis. With the exception of the case of primary hyperparathyroidism with bone disease, none of these patients showed alteration of the bones

* From the Institute of Medical Semeiology, University of Siena, Siena, Italy.

† Professor of Gerontology and Geriatrics in the University of Siena.

Post-cardiac-infection patients with an arcus senilis did not differ from those without an arcus in respect of serum cholesterol, blood-pressure, or body build, except that all patients with a serum cholesterol of over 350 mg./100 ml. had an arcus senilis.

We are grateful to Dr. K. G. Green for permission to use the results of serum cholesterol estimations carried out by the Pharmaceuticals Division of Imperial Chemical Industries Ltd. We wish to thank the physicians of Aberdeen General Hospitals for access to patients under their care, and Professor H. W. Fullerton for help and advice in the preparation of this paper.

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demonstrable by radiological examination. The renal function was normal in all cases.

The plasma calcium level was high in all but one of the patients (normal range in our laboratory, 9-11 mg./100 ml.); the inorganic phosphorus level of the plasma was normal in all cases with the exception of the case of primary hyperparathyroidism (normal range in our laboratory, 2.5-4.5 mg./100 ml.); and the urine level of calcium was high in all cases (normal range in our laboratory, 100-180 mg./24 hr.). Alkaline phosphatase was very high in the case of parathyroid adenoma, and normal or borderline in the other cases (normal range in our laboratory, 5-15 K.A. units) (Table I).

TABLE I

Case No.	Sex and Age	X-ray Findings	Plasma Non-protein Nitrogen (mg./100 ml.)	Serum Calcium (mg./100 ml.)	Serum Inorganic Phosphorus (mg./100 ml.)	Urine Calcium (mg./24 hrs.)	Urine Phosphorus (mg./24 hrs.)	Alkaline Phosphatase (K.A. Units)
1	M 55	Multiple bilateral renal stones	24	12.8	3.0	590	1,400	16
2	M 58	" "	21	12.4	3.3	340	520	15
3	M 43	Multiple right renal stones	27	12.0	4.0	310	620	12
4	M 58	Bilateral nephrocalcinosis	20	10.6	4.0	320	1,100	18
5	F 51	Multiple left renal stones	34	11.8	4.2	450	490	8
6*	F 61	Multiple right renal stones	30	14.0	1.8	305	550	70

* Primary hyperparathyroidism with bone disease.

The fasting patients were given 50 μC of $^{45}\text{CaCl}_2$ (containing 5 mg. of calcium as carrier) orally at 8 a.m., dissolved in 10 ml. of a 10% calcium gluconate solution (containing 88 mg. of calcium as additional carrier).

Samples of venous blood were taken every five minutes for a period of 30 minutes, then every 10 minutes during the next

TABLE II

Case No.	Plasma Activities : % Dose per Litre of Plasma. Minutes												Urine Excretion : % Dose. Hours				Stools Excretion : % Dose. Hours	
	5	10	15	20	25	30	40	50	60	90	120	240	0-2	2-4	4-6	0-6	0-72	
<i>Stone-forming Patients</i>																		
1	-	-	+	0.5	1.1	1.6	2.0	2.3	2.6	2.7	2.4	2.0	0.8	0.6	0.6	2.0	25	
2	-	+	0.8	1.5	2.1	2.6	3.1	3.2	3.1	2.9	2.6	2.3	1.1	0.9	0.8	2.8	25	
3	-	+	0.5	1.2	1.8	2.3	2.9	3.1	3.2	3.0	2.7	2.5	1.0	0.9	0.9	2.9	28	
4	-	-	+	0.6	1.2	1.8	2.4	2.9	3.0	2.8	2.2	1.9	0.75	0.8	0.7	2.25	22	
5	+	0.5	1.0	1.9	2.5	3.0	3.4	3.5	3.4	3.1	2.8	2.5	0.9	0.8	0.9	2.6	21	
6	-	+	1.4	1.8	2.3	2.7	3.2	3.3	3.2	3.0	2.7	2.5	0.7	0.8	0.7	2.2	30	
<i>Normals (5 cases)</i>																		
Min. values	-	-	+	0.2	0.5	0.7	1.2	1.6	2.0	2.1	2.2	1.9	0.13	0.28	0.32	0.82	29	
Max. values	-	+	0.3	0.7	1.0	1.2	1.8	2.1	2.3	2.6	2.6	2.4	0.27	0.46	0.50	1.19	38	
<i>Hypoabsorbers (senile osteoporosis : 13 cases)</i>																		
Min. values	-	-	-	+	0.1	0.3	0.4	0.7	1.0	1.5	1.8	1.9	0.01	0.04	0.08	0.13	41	
Max. values	-	-	-	+	0.3	0.4	0.7	1.0	1.5	1.8	1.9	1.6	0.16	0.20	0.26	0.58	65	

radioactivity.

half-hour, and at one and a half, two, and four hours after administration of the dose. Urine was collected every two hours during the six hours after administration of the dose. Faeces were taken in a single sample after 72 hours.

Samples for counting were prepared as follows: 1 ml. of plasma was added to 3 ml. of 10% calcium gluconate solution. An excess of 3% ammonium oxalate was added to precipitate calcium as calcium oxalate, which was collected on a filter-paper disk with suction. The activity in the layer on the filter-paper disk was determined in a shielded sample-changer with an end-window Geiger counter (Tracerlab mod. TGC-2).

Urine samples were added to a concentrated hydrochloric acid solution. Acidity was neutralized with NH_3 . Calcium gluconate and ammonium oxalate were also added.

Faeces were homogenized and weighed. A 10-g. sample was ashed in a muffle furnace at 800°C . The residue was dissolved in 10 ml. of 10% perchloric acid. Calcium was precipitated in the usual manner.

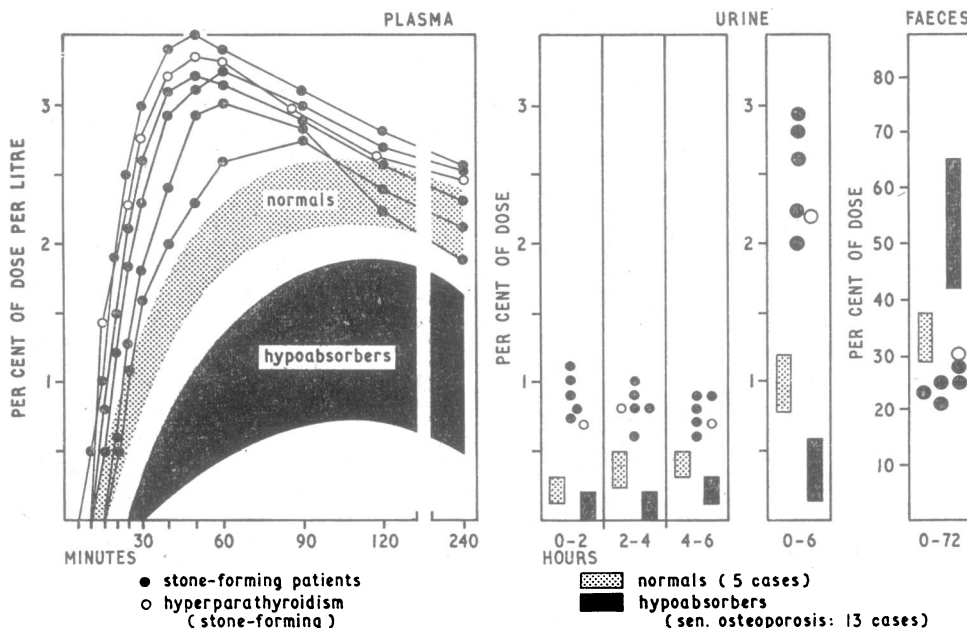
All counts were compared with standards prepared in an identical manner from aliquots of the administered dose of ^{45}Ca . A reagent blank was counted as a background sample. All counts were corrected for self-absorption, using an experimentally determined correction curve. It was unnecessary to correct counts for decay, since the standards were always recounted together with any group of samples.

Plasma counts were expressed as the percentage of dose administered per litre of plasma. Urine and faeces counts were expressed as the percentage of dose administered.

Results

As can be seen in Table II and in the Chart, the findings in the six stone-forming patients were uniform and differed greatly from the findings obtained in normal subjects.

These findings can be summarized as follows: (1) Radioactivity appeared in the plasma between 5 and 15 minutes after the oral administration of the dose of ^{45}Ca ; that is, after a normal slightly shortened interval. (2) The peak levels of radioactivity in the plasma were reached earlier than in a normal subject—within 60 minutes, as against the 90 to 120 minutes of normal cases. (3) The peak levels of radioactivity in the plasma were much higher than in the normal subject—between 2.5 and 3.5% of the dose per litre of plasma, as against values of 2.1 to 2.6% of the dose per litre of plasma in a normal subject. (4) Four hours after administration of the dose the plasma radioactivity levels were above the normal mean in three cases. (5) The urinary excretion of ^{45}Ca within six hours of administration of the dose was distinctly higher in the stone-forming patients than in the normal subject—in six hours between 2 and 3% of the dose administered was excreted in the urine, as against values of 0.82 to 1.19% in the normal subject. (6) The excretion of ^{45}Ca in the faeces during the 72 hours after the administration of the dose was with one exception lower than normal in all cases; within this 72-hour period, between 20 and 30% of the dose administered was faecally excreted, as against 29 to 38% in the normal subject. (7) The findings were particularly interesting as compared with the cases of senile osteoporosis, which presented a hypoabsorption of ^{45}Ca (Lenzi and Caniggia, 1962; Caniggia *et al.*, 1963).



Showing the findings in the subjects investigated.

Discussion

According to Fourman (1960) the symptoms of hyperparathyroidism are the result of hypercalcaemia, renal stones, and bone disease, the last being the least common.

One of our patients was a typical case of von Recklinghausen's disease. but none of the others had symptoms of hypercalcaemia (weakness, lack of tone of the muscles, thirst, polyuria, anorexia, mental disorders, and so on), none had bone lesions, and the biochemical pattern in these patients was not typical of hyperparathyroidism: the plasma phosphate level was normal and alkaline phosphatase normal or borderline.

Of course it is not possible to exclude with certainty that

our patients had a hyperparathyroidism without bone disease; but this is the most provocative aim of this article, as they had none of the known diseases with elevated calcium plasma level (sarcoidosis, vitamin-D poisoning, and so on).

Summary and Conclusions

In six stone-forming patients the intestinal absorption of ^{45}Ca was studied: it was found that all six patients examined were calcium-hyperabsorbers.

It is concluded that determination of the intestinal absorption of ^{45}Ca in patients with formation of calcium concretions proves such patients to be calcium-hyperabsorbers.

The principal metabolic abnormality in some patients with calcium nephrolithiasis could be an increased intestinal absorption of calcium.

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Medical Memoranda

Iatrogenic Vertebral Arteriovenous Fistula

[WITH SPECIAL PLATE]

Brit. med. J., 1965, **1**, 429-430

Arteriovenous communications between the vertebral vessels are usually a result of trauma. The common mechanisms are stab wounds, gunshot wounds (Aronson, 1961), or, occasionally, indirect injury to the cervical spine (Faeth and Dueker, 1961). There is one report of a "spontaneous" vertebral arteriovenous fistula (Gooddy and Schechter, 1960). Attention is drawn to a further cause—namely, percutaneous vertebral arteriography. We can discover in the English literature only two reports of a similar occurrence.

CASE REPORT

The patient, a 37-year-old housewife, had since the age of 18 suffered from a variety of psychogenic symptoms. At the age of 23, when in the second trimester of pregnancy, she developed pain and impairment of vision in both eyes, which had regressed by the end of pregnancy. Seven years later she developed an unsteady gait, tending to stagger to the right. This persisted, but two years later her left leg became numb and weak and she tended to trail the left foot. Two years ago she complained of increasing deadness of the left leg, associated with tingling in the left arm and leg, and renewed aching and blurring vision in the right eye. These and many non-organic symptoms led to her subsequent admission to another hospital for investigation. It was thought that she was suffering from disseminated sclerosis, but percutaneous right carotid and bilateral direct vertebral angiography were attempted to exclude a possible vascular cause for her symptoms. The radiographs were reported as being normal, but the patient was informed that the puncture of the right vertebral artery had been unsuccessful, though a successful examination had been obtained on the left. Five days later she experienced a terrifying, loud, rushing noise in the occipital and right temporal region, accentuated by stooping and by turning her neck. This remained unchanged, and six months later she was admitted to the Neurological Unit of the General Infirmary at Leeds for investigation because the noise was becoming increasingly difficult to tolerate.

Examination revealed pallor of the right optic disk, a central scotoma, and visual acuity of Jaeger 8 in the right eye. She had pyramidal signs in the left arm and leg, and a mild sensory impairment, of dubious significance, on the left side of the body. The most striking finding, however, was an easily palpable thrill and a very loud continuous bruit audible over the whole neck, but maximal on the right side; this was loudest over the upper end of the internal carotid artery, and was diminished by digital compression of the right subclavian artery. No bruit was audible over the orbits or skull. The blood-pressure was 130/80.

It was felt that in addition to disseminated sclerosis the patient had sustained an arteriovenous fistula. It was not possible to decide with absolute certainty which vessels were involved, but the fact that the bruit was reduced by pressure on the right subclavian artery but not by occlusion of the right carotid, and the history of a failed right vertebral puncture, were thought to be strongly suggestive of a lesion affecting the vertebral rather than the carotid artery. Radiological investigation was undertaken to confirm the diagnosis in the hope of demonstrating an operable lesion.

Radiological Investigation

Preliminary plain film examinations of the skull and neck showed no abnormality. A right axillary arteriogram was carried out under local anaesthesia and a red Kifa catheter positioned with its tip in the vertebral artery. Two injections each of 8 ml. of 45% Hypaque were made at hand pressure and serial films taken. The patient complained of transient slight discomfort in the right side of her neck after each injection.

An arteriovenous fistula between the vertebral artery and its venous plexus was demonstrated. The vertebral artery was enlarged and its proximal few centimetres were more tortuous than normal. Contrast medium passed rapidly into a much-dilated vertebral venous plexus, draining rapidly through enlarged deep cervical and vertebral veins. Some contrast passed across the midline at C4 level through the external vertebral venous plexuses and outlined the plexus of veins around the left vertebral artery below this level. Drainage was rapid and the left vertebral vein could be identified even before injection of contrast into the right vertebral artery had been completed.

The exact site of the fistula could not be identified, but it was thought to be at C4 level, where the vertebral artery narrowed abruptly and maximal venous filling occurred. Later films showed irregular dilatation of several small arterial anastomoses between the vertebral and the deep cervical, and between the vertebral and