The possibility of a modified attack of variola should always be borne in mind when suspicious rashes occur in vaccinated persons who have recently arrived in this country by air. The impact of such a case on a comparatively unvaccinated population was well demonstrated in the Bilston outbreak. One would have expected further cases to arise, especially in the overcrowded conditions prevailing in the area, and the early removal of the patients, with vaccination and close surveillance of the contacts, must have been contributory factors in limiting the spread. Mild missed cases in vaccinated persons must account for the missing links in the spread of the outbreak, the termination of which can be described only as natural or spontaneous.

Laboratory investigations have been of great help in confirming some cases, especially in the original group and in highly modified attacks such as Case 26. However, time must elapse before the receipt of the reports and of negative results in cases showing definite clinical signs.

As in previous outbreaks, vaccination did not fully protect within the first week of the vaccination period. Many contacts were vaccinated too late in the incubation period, owing to delay in diagnosis or even in sufficient suspicion of the true nature of the case. Confluent cases occurred where successful vaccination had been carried out nine and eight days before the appearance of the true smallpox rash, and in one haemorrhagic case successful revaccination had been carried out 12 days before, yet the case had a fatal termination. Modified cases occurred where vaccination had been carried out 12 days before the rash appeared. Many individuals vaccinated in the Services during the recent war years gave reactions which indicated absence of immunity.

An important administrative point in dealing with vaccination of school-children is the necessity for obtaining the written consent of the parents. In the rush of children coming for vaccination from the schools it was assumed that parental consent had been given. Later it was found that some children had been vaccinated "to be in the fashion," without parental consent.

Summary

An outbreak of smallpox is described originating in a soldier who travelled from India by air and was at home for some days before he developed a modified attack of variola which was regarded as chicken-pox.

Of the 30 confirmed cases, 15 were severe, with 6 deaths—5 from haemorrhagic smallpox and 1 in an unvaccinated confluent case.

I wish to express my appreciation of the very great help given to me by Dr. B. C. Haller, medical officer of health for Bilston, during my period of special duty in the area and for his permission to utilize the various records and reports. My thanks are due to his staff for their ready co-operation at all times. I am greatly indebted to Dr. F. Asker, medical officer of the Joint Infectious Diseases Board, and to Miss Marian Curtin, the matron of the Moxley Hospital and Bilston Smallpox Hospital, for their help in giving me access to the cases and treatment records and for permission to use the records in collating this report. I also wish to record my thanks to Dr. G. Ramage, medical officer of health for Staffordshire, for his permission to publish this report.

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ABSORPTION OF VITAMIN D IN STEATORRHOEA

BY

L. P. R. FOURMAN, M.D.

AND

G. H. SPRAY, D.Phil.

(From the Nuffield Department of Clinical Medicine,
University of Oxford)

Osteomalacia, though rare in this country, may sometimes be seen as a complication of steatorrhea. Ryle (1924) and others have suggested that the formation of insoluble calcium soaps in the fatty intestinal contents inhibits calcium absorption in this condition. Although unabsorbed fat in the bowel does affect calcium absorption (Telfer, 1926-7), soap formation cannot entirely explain the failure to absorb calcium, otherwise the patients should absorb calcium normally on a fat-free diet. In fact, restriction of fat intake alone does not improve calcium absorption (Bauer and Marble, 1932; Bassett *et al.*, 1939), but vitamin D rapidly restores it to normal even with a normal fat intake, indicating that the patients are vitamin-D-deficient. It was suggested that this was due to a failure of absorption of the vitamin, but no direct evidence of such an effect has been given. In three cases successfully treated with vitamin D, Bassett *et al.* (1939) used large doses (225,000-900,000 i.u. daily) by mouth, these doses being similar to those required in cases of vitamin-D-resistant rickets (McCance, 1947); they obtained equivocal results when smaller doses (10,000 i.u. daily) were given parenterally to another patient. Bauer and Marble (1932) obtained an immediate response with 10 mg. of ergosterol daily by mouth.

When a woman with osteomalacia due to steatorrhea recently came under our observation the absorption of vitamin D was investigated by comparing the responses to oral and parenteral administration, using doses known to be adequate for treatment of osteomalacia of dietary origin (Liu *et al.*, 1935).

Case History

The patient, a nun aged 61, had worked in Poona from 1932 to 1937. In 1938 a right hemicolectomy, including resection of the last 4 in. (10 cm.) of the ileum, was performed for carcinoma of the caecum; healed tuberculous mesenteric glands were found at operation. After this operation she had diarrhoea, and in July, 1944, she was admitted to this unit for anaemia. Steatorrhea with megaloblastic anaemia was then found. A sternal puncture showed the sternum to be soft; plasma phosphatase was 14 King-Armstrong units, but serum calcium and phosphate were not estimated and osteomalacia was not suspected at that time.

Treatment was begun with a low-fat diet with extra meat, weekly liver injections, and added vitamins A and D (1,500 units daily), and this regime was continued after discharge. The anaemia and diarrhoea responded fairly well, but in the spring of 1946 she had severe pains in the spine, round the trunk, and in the sacro-iliac joints. Radiographs revealed generalized osteoporosis, and calcium sodium lactate (6 g. daily) was prescribed. This helped to control the diarrhoea, but produced little change in the bone pains. These were rather better after a summer holiday, but this improvement may be ascribed to sunlight rather than to the treatment, for in the spring of 1947 her condition had again become much worse. Every movement of the trunk was very painful; from the lying position she could not sit up without pulling herself up by her arms; her muscles felt weak and she was getting involuntary spasms of the abdominal muscles. Her ribs were very tender. Chvostek's sign was positive, Trousseau's sign

negative. She was admitted to hospital, where the presence of steatorrhoea was confirmed, although the anaemia had improved (Hb, 11.7 g. %; R.B.C., 4,400,000 per c.mm.; M.C.V., 91 cu. μ).

The report of biochemical investigations was as follows: blood urea, 35 mg. per 100 ml.; plasma chloride, 464 mg. per 100 ml. (79.5 m.eq./l.); blood bicarbonate, 50 vols. per 100 ml. (22.2 m.eq./l.); blood phosphate, 2.77 mg. per 100 ml. (1.62 m.eq./l.); serum calcium, 8.8 mg. per 100 ml. (4.4 m.eq./l.); plasma phosphatase, 58 King-Armstrong units; plasma proteins, total 4.7 g. per 100 ml.; albumin, 2.75 g. per 100 ml.

Radiographs of the bones revealed considerable decalcification and multiple fractures of the ribs on the right side. These defects, together with the low blood calcium and phosphate and the high phosphatase, confirmed a diagnosis of osteomalacia. The condition had progressed despite the low-fat diet with added vitamin D for nearly three years, and had not responded to the extra calcium intake. During a preliminary period of

mouth was continued, but for the next four periods the same dose was given parenterally. Finally, to determine the effect of the added calcium on fat absorption, fat excretion was measured during a further two four-day periods without any added calcium; calcium balances were not determined for these last two periods.

Calcium intakes were calculated partly from food tables (McCance and Widdowson, 1946), but the calcium contents of calcium lactate and milk were measured direct. These sources accounted for about 4.5 g. of the total daily intake of about 4.6 g., so that the probable error of 10% due to using food tables represented a negligible error in estimating the total calcium intake. With the exception of milk, no foods rich in calcium or which contained much oxalate were given. The bread intake was kept constant. All fat intakes were determined from food tables.

For the four-day collection periods carmine (0.6 g.) was used as a faecal marker, and a rectal washout of distilled water was given to complete each collection. Calcium was determined by titration—in the faeces by the method of McCrudden (1911–12), in the urine by that of Shohl and Pedley (1922)—while the inorganic phosphate of urine was estimated colorimetrically, using the method of Berenblum and Chain (1938). Faecal fat was estimated gravimetrically after extracting with petroleum ether an aliquot of wet faeces previously treated with hydrochloric acid and dried with plaster-of-Paris.

Results

During the time of the balance observations there was no significant rise in blood calcium or phosphate (see Fig.), despite apparently effective treatment (cf. Bauer and Marble, 1932; Liu *et al.*, 1935). In periods 1–4, with oral vitamin D, 1.9 g. of calcium was retained, while in periods 5–8, with parenteral therapy, 6 g. was retained (see Table). Marked calcium retention began only during the second four-day period after the

Table showing Four-day Calcium and Fat Balances with Oral and Parenteral Vitamin D Therapy

4-day Period	Calcium				Phosphate in Urine (g.)	Fat (g.)	
	Intake (g.)	In Faeces (g.)	Absorbed (g.)	In Urine (mg.)		Intake	In Faeces
By mouth:							
1	19.2	17.6	1.6	130	1.36	278	244
2	18.3	16.7	1.6	130	1.05	263	212
3	19.2	19.9	-0.7	90	1.60	289	238
4	18.9	19.5	-0.6	10	1.53	281	240
Total (16 days)	75.6	73.7	1.9		5.54	1,111	934
Intramuscularly:							
5	18.0	20.0	-2.0	7	1.32	266	266
6	18.7	15.7	3.0	4	1.56	273	200
7	18.2	16.3	1.9	3	1.43	279	284
8	18.4	15.3	3.1	4	1.89	280	282
Total (16 days)	73.3	67.3	6.0		6.20	1,098	1,032
9 } 10 }	Approx. 6.0					276 280	128 144

start of parenteral therapy. Bauer and Marble found an immediate response to treatment, but Liu *et al.* (1935) and Linder and Harris (1930) also found a negative calcium balance immediately after beginning treatment. The latter authors attributed this to the parathyroid-like action of vitamin D, but the absence of an associated rise in urinary phosphate excretion makes this explanation unlikely (Albright *et al.*, 1946).

The greatly improved calcium balance after parenteral therapy cannot be due to collection errors, for these would affect the results for fat excretion similarly. In fact, fat excretion rose during the second experimental period, when fat absorption was apparently less than 10%. The excessive loss of fat in the faeces was partly due to the high calcium intake: thus the mean daily fatty acid loss in periods 7 and 8 was 71 g., in periods 9 and 10, when no supplementary calcium was given, 34 g.—a difference of 37 g. This amount of fat, assuming an average molecular weight of 271 for fatty acids, would combine with 2.78 g. of calcium. The actual supplementary calcium intake was 3.15 g. This effect of calcium in preventing fat absorption was much more pronounced than in normal people (Fourman), and similar findings in steatorrhoea have been

Chart showing the blood findings during treatment with vitamin D.

observation the calcium sodium lactate was increased to 16 g. daily, and the vitamin D to 12,000 units daily by mouth. A low-fat diet (30 g. of fat daily) and liver injections were continued as before. Her pains slowly disappeared, but she remained too weak to get up. Blood findings were practically unchanged (see Fig.). This treatment was continued for five weeks prior to the observations on calcium absorption.

Experimental Observations

The object of the calcium balance observations was to obtain evidence on the absorption of vitamin D. The experiment was begun with a relatively high fat intake (70 g. daily), but time did not permit us to continue the observations on a lower intake. Preliminary tests had shown that 40–50% of the dietary fat was excreted, so that during the experiment 25 g. of calcium lactate was given daily, the diet thus containing more calcium than would combine with this amount of fat in the stools. Observations were made over ten four-day periods, beginning three days after the start of the experimental regime. The fat intake was almost constant throughout the experiment, and the total fat excretion in each period was measured. During the first four four-day periods vitamin D (12,000 units daily) by

reported by Bassett *et al.* (1939). On the other hand, the results show that once vitamin D was made available the excess of fat in the faeces did not prevent calcium absorption by soap formation. There may be two reasons for this: first, soap formation possibly does not occur until the calcium and fatty acids come together in the colon (Ingelfinger, 1943); secondly, calcium soaps can be utilized, though poorly (Boyd *et al.*, 1932).

Progress Note.—Ten weeks after the conclusion of the experiment blood calcium and phosphate values had risen to normal with continued parenteral vitamin D therapy, but the phosphatase remained high (see Fig.).

Discussion

The response to the parenteral injection of a dose of vitamin D which was ineffective by mouth indicates failure of absorption of vitamin D in our patient. This failure was not due to the increased steatorrhoea produced by the experimental conditions, since these conditions did not obtain before the experiment was begun, when the patient had been receiving the same dose of the vitamin while on a low-fat diet. Absorption of the vitamin at this time would have affected calcium absorption during the experimental period, since the action of vitamin D even in small doses is a prolonged one (Hannon *et al.*, 1934), so that apparently even without massive steatorrhoea absorption of the vitamin was defective. We were not able, as originally planned, to determine whether absorption of the vitamin occurs in the absence of fat in the bowel.

On the basis of our observations certain recommendations may be made. Ordinary doses of vitamin D may be ineffective in the prevention of osteomalacia in steatorrhoea. In the treatment of this condition parenteral therapy with ordinary doses of the vitamin is effective; oral therapy requires very large doses and is at best uncertain. Large amounts of calcium are necessary to replenish the stores. The patient may absorb 1 g. of calcium daily, equivalent to 7.7 g. of calcium lactate, and may even absorb much more (Starr and Gardner, 1930). It should be remembered, too, that approximately 13.5 g. of fatty acids combine with 1 g. of calcium, and it is possible that some calcium becomes unavailable by forming calcium soaps, so that, though the fat intake need never be severely limited except for diarrhoea, it should be as low as is compatible with a palatable diet when steatorrhoea is complicated by osteomalacia. Possible deficiencies of other fat-soluble vitamins need also to be treated.

Summary

A case of osteomalacia secondary to steatorrhoea is described. The bone disease progressed despite treatment with a low-fat diet and vitamin D and calcium supplements. On a daily intake of 70 g. of fat, 4.6 g. of calcium, and 12,000 i.u. of vitamin D orally the patient retained 1.9 g. of calcium in 16 days. With the same dose of vitamin given intramuscularly she retained 6 g. of calcium in 16 days. Apparently a failure to absorb vitamin D occurs in osteomalacia due to steatorrhoea.

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SUPERIOR MESENTERIC ARTERIAL OCCLUSION

RECOVERY WITHOUT RESECTION

BY

W. GARDEN HENDRY, M.B., F.R.C.S., F.R.C.S.Ed.
*Surgeon, E.M.S. L.C.C. Northern Hospital,
 Winchmore Hill, London*

In the pre-heparin era Whittaker and Pemberton (1938), in a discussion of 60 cases of superior mesenteric arterial occlusion, cited only one case with spontaneous recovery which later, coming to necropsy from an unrelated cause, was proved to have obliteration of the superior mesenteric artery. They also quoted Dye as having had three cases clinically diagnosed as mesenteric thrombosis, in which, on death from unrelated causes, obliteration of that artery was found at necropsy. However, since the introduction of heparin therapy, Jorpes (1946) quotes Ravdin as having two spontaneous recoveries, and Rudberg as having one in which the intestine looked viable and was not resected.

Spontaneous Recovery in Case Management.—The incidence of spontaneous recovery is impossible of assessment from the data available, as a number of cases undiagnosed and untreated will recover; but that a definite percentage do recover is certain, and this should be borne in mind when one is confronted with haemorrhagic infarction of the small gut that is still apparently viable. Especially is this so now, for while in the past the mortality rate of resected cases was 84.2% (Whittaker and Pemberton, 1938) and 83% (Cokkinis, 1926), the rate has no doubt dropped considerably in early cases since the introduction of heparin, —for example, Jorpes (1946) quotes Murray and McKenzie as having six resections with four recoveries.

Aetiology.—Cases of arterial occlusion can be immediately divided into embolic and thrombotic, and most of those in the literature would seem to be due to the latter phenomenon in local arterial disease—a very common condition, whether it be an atheroma, an arteriosclerosis, or a thrombo-angiitis obliterans. Even in cases of degenerative heart disease a thrombosis from associated arterial disease is more likely than an embolism, though in valvular disease of the heart embolism of course does occur. In the following case, with no evidence of embolism and with widespread peripheral arterial degeneration, thrombosis is very probably the correct diagnosis.

Addendum to Accepted Pathology

It is puzzling that a part with so much collateral circulation is so readily subject to infarction, and it has been suggested (Boyd, 1946) that the intense spasm of the gut in response to the local anaemia isolates the segment from the neighbouring circulation, while Cokkinis (1926) avers that infarction takes place when the secondary spreading thrombosis reaches the distal arterial arcade and blocks the vasa