SEVEN CASES OF BARIUM CARBONATE POISONING

BY

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Seven patients were given barium carbonate for barium-meal investigations in December, 1948. Pure barium carbonate is a white powder exactly similar in appearance to barium sulphate and is commonly used as a rat poison. It had been placed by mistake in the barium sulphate container at the wholesale premises, and was therefore dispensed instead of barium sulphate in the preparation of the barium cream supplied to the radiologist. The quantity administered in all cases except the fatal one was a glassful, which was later shown to be equivalent to 4.26 oz. (133 g.) of dry barium carbonate. In the fatal case half this quantity was administered.

Case Reports

Case 1.—A healthy man aged 48 swallowed a chicken bone which lodged in his oesophagus. Forty hours later he was sent for x-ray examination. He swallowed half a glass of barium cream. The x-ray film confirmed the presence of the bone and he was admitted to hospital for its removal. An hour and a half after the barium swallow he was examined by the anaesthetist, who found him fit except for a raised blood pressure, 180/130, which was attributed to nervousness. While his throat was being sprayed with cocaine and adrenaline he vomited. He was given "omnupon," $\frac{1}{4}$ gr. (22 mg.), and hyoscine, 1/100 gr. (0.65 g.). Half an hour later 0.7 g. of thiopentone and 8 mg. of "tubarine" were given. The bone was not impacted and was removed uneventfully. The patient recovered from the anaesthetic, and then started to vomit and purge; this was accompanied by severe griping abdominal pain, tenesmus, and the desire to micturate. He was very thirsty and soon began to complain of heaviness in his limbs and coldness. The vomiting ceased but the diarrhoea continued, he sweated profusely, and his pulse became thready and irregular; his temperature was normal and his mind clear. He was treated with warmth, hot drinks, intravenous glucose saline, nikethamide, adrenaline, and brandy. His pulse became imperceptible, and 11 hours after the operation and 13 hours after the barium, he died. At necropsy acid-soluble barium compound was found in the stomach, liver, and kidneys. The equivalent of 2.75 gr. (0.18 g.) of barium carbonate was recovered from the stomach.

Case 2.—This patient was given a full glass of barium cream (equivalent to 4 oz. of barium carbonate). An hour later he started to vomit and had diarrhoea with abdominal colic; his vision was blurred. He was taken to his family doctor, who found him shocked, cyanosed, and sweating, and able to move his limbs only with great difficulty. He complained of heaviness in the arms and legs, he could talk only in a whisper, his pulse was slow and irregular; his mind was clear. I was called in consultation and advised warmth, hot drinks, nikethamide, and a sedative. Eight hours after the barium he was still able to move his limbs only with great difficulty; he had no convulsions. His pulse was 90 and still irregular. An electrocardiogram showed extrasystoles. His blood pressure was 160/100. The reflexes were all present but diminished; the Babinski responses were flexor. Twelve hours later he was improving, but remained in bed in the doctor's house extremely ill for over a week.

Case 3.—A married woman aged about 30 was given a full glass of the cream. Within two hours her face felt stiff and her vision was blurred. A little later vomiting started, followed by diarrhoea and tenesmus. When examined three hours after the meal she was weak, cyanosed, and sweating; her mind was clear, and her pulse was grossly irregular. Her blood

pressure was 180/110. An electrocardiogram showed extrasystoles. The muscles of the arms and legs were painful and extremely weak, and she could move only with the greatest difficulty. All the reflexes were present but greatly reduced; there were no convulsive movements. She was immediately admitted to hospital, treated for shock, and given magnesium sulphate—a known antidote for barium carbonate, reacting with it to form insoluble barium sulphate. The weakness of her limbs and irregularity of the heart persisted for nearly a week, but she eventually recovered and was discharged after 16 days in hospital.

Case 4.—A pensioner with hypertensive failure was given a full glass of the cream for a barium meal. Vomiting and diarrhoea started after he returned home. However, he was not so ill as the other patients, perhaps because he had an achlorhydria, or at least a hypochlorhydria, which prevented or diminished the formation of barium chloride in his stomach. Also he took castor oil and, later, Epsom salts. He was admitted to hospital, where I saw him. Two days later he said he felt fitter than for many months. The dyspnoea which had greatly troubled him was much improved; this was considered to be due to the digitalis-like action of soluble barium salts on the heart muscle. Ten days later, however, he had a small cerebral thrombosis, followed after four days by a second which resulted in a left hemiplegia. He gradually recovered, but two months later a fall from his chair resulted in a large extradural haemorrhage and he died within a few days. In this case an elderly man with a weakened myocardium recovered from 4 oz. of barium carbonate; his death may have been indirectly due to the poisoning.

Case 5.—A married woman drank a full glass of the cream for a barium meal. About an hour later she felt as if her face was distorted and her lips were swollen; she complained of double vision. Vomiting, diarrhoea, and tenesmus soon followed. When seen by her family doctor she was cold and collapsed, in a state of shock, with a highly irregular pulse. Several hours later magnesium sulphate was given and she gradually recovered over the course of a few days.

Case 6.—A man was given a full glass of the barium cream and was told to take two teaspoonfuls of Epsom salts in an hour's time to keep the contents of the intestines liquid and so help to outline the appendix. He returned nearly five hours later, having vomited once but otherwise feeling well. In this case the taking of magnesium sulphate had apparently prevented harmful absorption.

Case 7.—This patient left by ship on the same day that he swallowed a glassful of the barium carbonate cream for x-ray purposes. He also vomited and had diarrhoea, and was given magnesium sulphate, but no further details are available.

Comment

The quantity of barium carbonate given in a glass of barium cream was estimated by the government analytical chemist at Port Elizabeth. He found that a full tumbler of the cream used contained 4.26 oz. (133 g.) of dry barium carbonate. All six patients who took this amount recovered. In the fatal case it was estimated that the patient, who was given half a glassful of barium cream, took three mouthfuls; this amount was estimated to contain approximately 1.7 oz. (53 g.).

In the stomach barium carbonate is converted by the free hydrochloric acid, if present, into barium chloride; it is absorbed from the small intestine and not from the stomach; there is, therefore, a time lag before symptoms become apparent.

Sydney Smith and Cook (1948) quote two fatal cases of barium carbonate poisoning after the ingestion of 1 dr. (4 g.). Fatal cases have also been recorded after 60 gr. (4 g.) of barium chloride (Blythe and Blythe, 1920; Sydney Smith, 1943) and after 12 gr. (0.8 g.) (Goodman and Gilman, 1941).

The pharmacology of barium has been described by Cushny (1936), who states that its chief action is to strengthen the contraction of all forms of muscle, probably by direct action on the contractile tissue. In this outbreak the effect on muscle greatly predominated over any other action: striated muscle, non-striated muscle, and the cardiovascular musculature were all affected.

In the above cases the first symptom to appear was often a peculiar stiffness about the mouth, accompanied by blurring of vision. This was rapidly followed by salivation, nausea, vomiting, diarrhoea, severe colic, tenesmus, and an intense desire to pass water. Within an hour skeletal-muscle weakness became pronounced and movement was difficult; the legs were weaker than the arms. The typical patient appeared clammy and pale, realized he was desperately ill, and talked only in a whisper; the pulse was extremely irregular, probably owing to very frequent extrasystoles; the blood pressure was raised. Even when *in extremis* the pulse did not become fast, but remained irregular and gradually became weaker. In no case was there convincing evidence of any direct poisonous action on the central nervous system; the mind always remained clear, there were no convulsions or tetanic contractions, the reflexes were diminished but not absent, the plantar responses were flexor, and there was no true paralysis.

Summary and Conclusion

Seven cases of poisoning by pure barium carbonate are described, only one of which was fatal. In this case the barium was followed by heavy sedation and an anaesthetic, which delayed vomiting and purging. The dose administered in all cases was many times that usually considered lethal. The large amount of barium carbonate administered caused rapid onset of vomiting and diarrhoea, which may account for recovery where a smaller dose might have been fatal. Magnesium sulphate, which was given in some cases, proved an excellent antidote. The symptoms were remarkably constant and all of them could be attributed to direct action on muscle. There was no evidence of any effect on the central nervous system: in this respect our views on alimentary barium poisoning should be reviewed; it may be that some of the symptoms described by other authors were due to impurities in the barium.

Finally, I would make the suggestion that barium carbonate, which in its pure form is a white powder, should be coloured by a distinctive dye so that the above mistake does not occur again.

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Medical Memoranda

Latent Amoebic Dysentery

The following case is of interest on account of the exceptionally long interval between infection and the development of symptoms, and because the diagnosis of amoebic dysentery was at first missed in spite of considerable investigation. It raises, too, the problem of whether the infection was contracted abroad or was of indigenous origin.

CASE REPORT

The patient, an intelligent man aged 60, had spent much of his early life abroad with the Army. He had had little illness apart from malaria, and, although he had never had injection treatment for dysentery, he had been admitted to Army hospitals three times for diarrhoea whilst on active service in Mesopotamia between 1916 and 1918. He returned finally to England in 1919 and five years later left the Army to become a postman. He remained in good health until January, 1944, when he developed intermittent diarrhoea following the death of his youngest daughter from encephalitis. He lost weight, the diarrhoea continued, and his general condition deteriorated. Six months later he was admitted to a general hospital, where a diagnosis of ulcerative colitis was made. The condition improved following sulphaguanidine treatment, but after his discharge from hospital relapses became more frequent, especially during the winter, and he was compelled to retire from public service in June, 1945.

In the autumn of 1947 he contracted whooping-cough and it was for that reason that I first saw him. He looked extremely ill, very pale, and emaciated, and he had a severe glossitis. Diarrhoea was troublesome, with six to ten motions daily, the stools containing both blood and mucus. He weighed only 8 st. (50.1 kg.).

A blood count showed: haemoglobin, 45%; red cells 4,000,000 per c.mm. I assumed the previous diagnosis of ulcerative colitis to be correct, and therefore gave him a course of sulphaguanidine, followed by oral iron and injections of crude liver and riboflavin.

Two months later the whooping-cough had completely subsided, but the diarrhoea persisted. The possibility of this being a case of amoebic dysentery had by now occurred to me, and he was eventually admitted to hospital on February 2, 1948, for further investigation. Next day a sigmoidoscopy (8 in.—20 cm.) revealed several crateriform ulcers with haemorrhage suggestive of chronic amoebic dysentery. The stools were examined microscopically, and amoebae containing ingested erythrocytes and exhibiting pseudopodial motility characteristic of *Entamoeba histolytica* were observed in two separate specimens. A radiograph of the chest and screening showed the right diaphragm to be raised and the right costophrenic angle to be partly obscured. The white blood cells numbered 8,300, with 86% polymorphs and 1% eosinophils.

A comprehensive course of treatment was instituted combining succinylsulphathiazole, emetine hydrochloride, chiniofon retention enemata, emetine and bismuth iodide capsules, and acetarsol tablets. Vitamin B complex and "fersolate" tablets were given orally, together with a low-residue diet. The response to treatment was most satisfactory. Bowel action became normal after the fifth day of treatment and by the end of one month the patient had gained 28 lb. (12.7 kg.) in weight. By then sigmoidoscopy showed an almost normal mucosa and the stools were negative for *E. histolytica* cysts. A blood count showed: haemoglobin, 75%; red cells, 5,300,000 per c.mm.

Convalescence was progressive and uneventful, and six months later he had regained all but 3 lb. (1.4 kg.) of his normal 12 st. (76.2 kg.) weight, and was able to undertake light work. A year after treatment sigmoidoscopy revealed no ulcers and no *E. histolytica* cysts.

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