common. Osteitis deformans is associated with a special form of heart failure due to high output (Edholm, Howarth, and McMichael, 1945). The results of laminectomy depend on the duration of symptoms, and little improvement will occur when the paraplegia has been present for a long time. Out of the 44 cases in the literature, laminectomy was done 25 times, with three (12%) deaths, and 18 cases have shown improvement after surgery. An important complication is the recurrence of the paraplegia after the initial relief. This has happened in at least three cases.

Case 1 had chronic bronchitis and pulmonary heart disease, but she tolerated the operation well. Her recovery from the paraplegia was slow, but at the end of five and a half months she had only slight neurological deficiency. Case 2 was not operated upon when first seen, owing to his congestive cardiac failure and poor general condition. It was surprising to find him alive ten months later. Although his cardiovascular state has improved, he has been bed-ridden for eighteen months. It is extremely unlikely that any improvement in his paraplegia would occur if decompression was done at this date. The general risk is considerable and it has been decided that he should not be submitted to operative intervention. Sarcomatous change is a real contingency that cannot be obviated.

Summary

Two cases of paraplegia due to osteitis deformans of the spine are described.

The literature is brought up to date.

The differences between the two cases and the general pattern of the disorder are discussed.

I am grateful to Dr. John Willis, of Palmerston North, for referring Case 1 and to Dr. Ian M. Macgregor, medical superintendent, Silverstream Hospital, for access to the clinical notes of Case 2.

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The results of the first real test of the Emergency Bed Service's warning system are considered in the report of the service for the year ended March 31, 1953. The year under review includes the great London fog of last December, which resulted in a quite unprecedented rush of applications, and this was followed at the end of January by the influenza epidemic. Throughout this time of crisis, the warning system worked remarkably well, but the report says: "Although an outstanding success, experience has shown, however, that improvement is possible. At present the issue and cancellation of the warnings is solely dependent on the admission rate, and the winter has shown that this tends to result in the warning being put on rather too late and taken off too early. A modification of the scheme is being worked out whereby account is taken not only of the admission rate, but also of the number of applications being received and of the number of admissions made only after referring the case to the medical referees." Applications over the year as a whole increased, and analysis tends to confirm that this was owing to a rise in sickness rather than in the number of doctors using the service. The report also contains numerical details of the applications and admissions during the year.

RECURRENT GALL-STONE ILEUS

BY

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Gall-stone ileus is stated by various authorities to be the cause of 1-2% of all intestinal obstructions (Illingworth and Dick, 1941). Vick (1932) found that in over 6,000 cases of intestinal obstruction 0.68% were due to gall-stones. The high mortality, said to be over 50%, is due to delay in diagnosis and to the advanced age of the patients. Few of the reported cases were diagnosed pre-operatively. Several cases have been reported with multiple stones in the small intestine, but in only a small minority has a second intestinal obstruction occurred. Maurice Lee reported such a case in 1945. The stone usually ulcerates through between the gall-bladder and the duodenum, where it passes down into the small intestine, becoming impacted at the lower ileum. Wakefield, Vickers, and Walters (1939) in 141 post-mortem examinations found the fistulous track in 101 cases.

Case History

A housewife aged 57, 1 para, was seized by violent epigastric pain followed by vomiting two days before admission. These symptoms soon eased but returned on the day of admission, the pain this time being colicky in nature and central in situation. It was associated with severe and persistent vomiting. There was no relevant previous history apart from a dislike of fatty foods.

On examination the patient was dehydrated, with a dry, furred tongue. The abdomen was slightly distended, and on auscultation continuous loud peristaltic sounds were heard. Two enemas were given with negative results. After a diagnosis of acute intestinal obstruction of unknown causation, a laparotomy was performed.

First Operation (J. A. M.).—A distended loop of small bowel presented itself through a right middle paramedian incision. This loop contained a gall-stone which was impacted about 2 ft. (60 cm.) from the ileo-caecal valve. As it was too hard to crush, it was milked up into the distended loop and removed through a longitudinal inci-

Photograph of the two gall-stones.

sion. No other gall-stone could be felt in the small intestine. The gall-bladder area was not examined. Up till the ninth day the post-operative course was uneventful. The patient was taking nourishment and her bowels were working normally. On the tenth day, however, she complained of epigastric pain and vomiting. During the following 10 days she had attacks of incomplete intestinal obstruction, with colic, vomiting, and constipation one day followed by the passage of diarrhoea and flatus the next. It was thought that she had a post-operative obstruction due to local peritonitis and kinking, and attempts to relieve it by intestinal decompression were made. By the twentieth day it was decided that as she was not improving a further laparotomy should be performed.

Second Operation (Mr. W. J. M. Brandon).—The abdomen was opened through the previous wound. A second much larger stone was removed from the same area as before. The post-operative course this time, though slow, was uneventful.

The first stone was pyramidal in shape, faceted at its base, and 1 in. (2.5 cm.) in diameter. The second stone was shell-shaped and 1½ in. (3.8 cm.) in diameter (see photograph).

Comment

In this case the stones must have been lying silent for some time in the gall-bladder, the smaller stone ulcerating through first, the larger one taking 10 days longer to force its way through the preformed fistula's track. If the significance of the large facet on the base of the first stone had been realized, an earlier diagnosis of the post-operative symptoms could have been made. Both these stones were radio-opaque, as tested post-operatively. This is unusual. The sex of the patient, the lack of previous history, and the intermittent type of intestinal obstruction are typical features of this condition.

Summary

A case is reported in which 20 days after an operation for gall-stone ileus a second stone was removed from the small intestine. The patient eventually recovered.

I would like to thank Mr. W. J. M. Brandon, consultant surgeon, Law Hospital, for his help and permission to publish this case.

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Medical Memorandum

Mercuric Chloride Poisoning Treated with Dimercaprol

Mercuric chloride poisoning with suicidal intent is fortunately no longer fashionable in this country, although it still seems to be common on the Continent and in America. While Longcope and Luetscher (1949) in America reported on 61 cases of mercuric chloride poisoning treated with dimercaprol (B.A.L.), collected over a period of three years, no reports have, to my knowledge, been published in this country. It therefore seems of interest to make known the following case.

CASE REPORT

A man aged 37 swallowed 2 g. of mercuric chloride with suicidal intention at 1 a.m. on May 6. He vomited repeatedly shortly after taking the tablets, but the vomit is said not to have contained solid particles of the substance, but only to have been stained blue. About one hour later he passed a large amount of pale urine and began to have cramps in his hands and feet. At 10 a.m. he had violent abdominal colic with bloody stools. He then took fright and sent for a doctor. He was admitted to hospital one hour later.

He was seen to be a rather underweight man of slender build, but there was no evidence of shock and no gingivitis; the tongue was coated but not dry. The pulse was regular and the blood pressure 110/80. The bladder was not palpable suprapubically, but no urine had been passed since 2 a.m. He complained of some abdominal colic and of painful cramps in his limbs.

He was given an immediate injection of 100 mg. of dimercaprol, and two hours later a further 200 mg. The injections were repeated at four-hourly intervals for four doses, and then 100 mg. was given four-hourly for a further four doses. The patient reported very shortly after the first injection that the cramps in his limbs were abating and he had no abdominal colic and no bloody stools.

The first specimen of urine was obtained at 10 p.m. in scanty quantity and loaded with protein; the sediment contained casts in most unusual numbers, some hundreds to a field. Next day the patient felt comfortable. His blood pressure was 120/70. The urinary output was still scanty and showed gross proteinuria, and blood urea was 46 mg. per 100 ml. The urinary output increased considerably from the third day, but there was a heavy trace of protein in the urine up to May 11. His blood pressure was stable at 120/70. On May 15 the sediment still contained a few casts. The patient was allowed up, and was discharged home on May 19.

The total dose of dimercaprol given was 1.6 g. over a period of 72 hours.

COMMENT

Although early vomiting may have prevented full absorption of the dose taken, it is likely that the consequences would have been fatal without appropriate treatment. Among a series of 86 cases of mercuric chloride poisoning reported by Longcope and Luetscher (1949), without treatment with dimercaprol, there were 27 deaths: death occurs in most cases of poisoning with 1 g. and more of the compound. Suppression of urine after an early diuresis, and bloody diarrhoea, point to a heavy absorption of the poison.

Following on the original publication by Peters *et al.* (1945) on the value of dimercaprol in the treatment of arsenical poisoning, Waters and Stock (1945) pointed out its possible use in the treatment of poisoning with other heavy metals, including mercury. Further, Gilman *et al.* (1946) investigated experimentally in cats and dogs the protective action of dimercaprol in poisoning with mercury, and reported favourably on it. The first and most important contribution to human therapeutics was by Longcope *et al.* (1946). They treated 23 cases of mercuric chloride poisoning with one death; one patient who recovered had swallowed 20 g. of the poison. Longcope and Luetscher (1949) reported a further 38 cases so treated, with only one other death. The dosage recommended by them is of 300 mg. for the first injection and a further two or three injections of 150 mg. at four-hourly intervals. A very important point is that treatment should be started at the earliest time within two or three hours from taking the poison. The one death reported occurred when treatment was delayed until 13 hours after poisoning.

Dimercaprol is known to produce toxic symptoms itself if given in a dose of 5 mg. and over per kg. body weight. These symptoms comprise nausea, vomiting, headaches, generalized aches and pains, burning sensations in the mouth, nose, and eyes, sweating, restlessness, weakness, and pains in the limbs, jaws, and trunk muscles. The heart rate is often increased, and there may be a rise in both systolic and diastolic blood pressure. These signs and symptoms subside within four hours (Peters *et al.*, 1945). The initial dose of 300 mg. recommended by Longcope may be expected to produce toxic symptoms in a number of patients of rather slender build; these symptoms have not been reported in the treatment of acute arsenical and mercuric poisoning, perhaps because the drug combines very rapidly within the body with heavy metals and is no longer available in free form. In comparison with the extreme danger to life of poisoning with mercuric chloride the risk of toxic reaction from the first large dose of dimercaprol is certainly inconsiderable.

The case described above made a remarkable recovery, as treatment was delayed for 12 hours and the first dose of the antidote was inadequate.

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