every case. Lead was found in many men who did not show the slightest evidence of lead absorption, and who were apparently fit. In one man (45 years old) who had worked for five years in the spelter works, as much as 1.89 parts of lead per million was found in the urine. He stated that he felt very well. He left the works and became a labourer, and two years later died of a perforated peptic ulcer. Post-mortem examination was refused. In the thirty men examined it was found that the higher the lead content in the urine the less the danger of immediate toxic symptoms. In four cases exhibiting drop-wrist a striking feature was the small quantity of lead found in the urine, and in one of these who showed other severe toxic symptoms, the amount of lead in the urine was as low as 0.163 part per million. Of the 100 cases examined twelve (whose ages ranged from 30 to 60) were found to be suffering from gastric ulcer, which was verified by x-ray photograph. Lead was found in the urine of each ulcer case. Five of the twelve perforated; one recovered and is now well. Necropsy was performed on three, and lead was found in the liver, brain, and kidney in each case.

POST-MORTEM EXAMINATION OF ONE CASE

The last necropsy we attended was that on a man who had shown all the classical signs of lead poisoning, and who had been certified as a case of plumbism a month earlier by Dr. Sladden, the medical referee under the Workmen's Compensation Act.

The man, aged 40, had been a spelter worker for many years. The body was emaciated.

Heart: atheroma of coronary arteries and well marked in the aorta. The left ventricle was hypertrophied. Lungs were congested; the apices were free, but there were adhesions of the left pleura. Kidneys: there was nothing of note on naked-eye examination. Brain: meninges were adherent, and the brain substance was congested; there was atheroma of the arteries comprising the circle of Willis. Stomach: there were minute haemorrhages in the mucous membrane of the stomach. Duodenum: there were two ulcers about the size of a shilling, one of which had perforated.

The results of chemical analysis of liver and kidney in this case were as follows:

	Lead	Zinc	Cadmium
Liver	0.063 grain per pound	0.294 grain per pound	0.511 grain per pound
Kidney	Nil	0.154 grain per pound	1.274 grain per pound

Table showing the occurrence of perforation in twelve cases of gastric ulcer associated with lead poisoning.

Case	Age	No. of Years at Spelter Works	Amount of Lead in the Urine	Habits	Remarks
			Parts per million		
1	30	8	0.738	Excessive beer drinker	
2	32	6	0.636	Abstainer	
3	36	13	0.175	Beer drinker	
4	39	15	0.195	Beer drinker	
5	42	5	0.259	Abstainer	Perforated duodenal ulcer: death
6	45	7	0.035	Beer drinker	
7	45	11	0.267	Abstainer	Perforated duodenal ulcer: death
8	45	5	1.89	Abstainer	Perforated duodenal ulcer: 1.89 parts per million in urine two years previous to death
9	47	17	0.151	Beer drinker	Perforated duodenal ulcer: death
10	57	26	0.025	Beer drinker	
11	57	28	0.05	Beer drinker	Perforated duodenal ulcer: recovery
12	60	30	0.056	Abstainer	

COMMENTARY

Inferences based on an analysis of this table must of necessity be indefinite, but one or two features are noteworthy. It appears that the older the workman, and the longer the time spent in the spelter trade, the less is the amount of lead excreted in the urine. An impairment of the renal function due to the toxic effects of lead on the kidney may be the explanation.

The incidence of ulcer seems to bear no relation to the duration of exposure to lead fumes; the occurrence of perforation, however, is associated with a low urinary lead content in each case. In our experience a moderate consumption of beer does not seem to play any part in influencing lead absorption; on the contrary, it apparently aids the elimination of lead by virtue of its aperient action. The pathology of a peptic ulcer is not clearly understood, and many authorities hold that toxæmia may play a potent part in its production. Lead is a very toxic element, and as it is eliminated by the liver via the alimentary canal, the duodenum is more exposed than the rest of the intestine to its deleterious effects. Brady of Harvard has pointed out that the bile is an important avenue for lead excretion. Glaser of Berlin has found ulcers in the stomach and duodenum in a large proportion of his plumbic cases, and he maintains that such ulcers are not the result of a special disposition, but are caused by toxic vagotonia. Lewin, Kasper, and many other Continental authors have drawn attention to the frequency of peptic ulcers in lead poisoning, and McJunkin found a necrosis of the gastric epithelium in animals. The experiments of Jores proved of much clinical interest. He administered lead to dogs and was successful in producing gastric and intestinal ulcers. On account of the frequency of gastric ulceration in lead poisoning his findings were significant, and may prove to be the starting point for further investigation of the etiology of gastric ulcer.

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GANGRENE DUE TO EMBOLUS OF THE INFERIOR MESENTERIC ARTERY

BY

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Embolism of the mesenteric arteries is, fortunately, a rare cause of an acute abdomen, and it is almost impossible to diagnose the condition with any degree of certainty. When it does occur, however, it is usual to find definite symptoms and signs of intestinal obstruction indicating the necessity for immediate laparotomy. One would expect a complete obstruction to present the usual clinical picture of absolute constipation, pain, vomiting (either of the gastric contents, or, later, of faecal type), and signs of peritonitis and collapse, depending on the length of time which had elapsed after the onset of the ileus.

The early stage of obstruction may present difficulties in diagnosis, but it is to be expected that a case which has progressed to extensive gangrene of the gut would have a history and clinical picture sufficiently clear to make the need for immediate surgical treatment obvious.

The case to be described had a history and symptoms which were so confusing and indefinite that a diagnosis of "acute abdomen" could not be made until the patient had been closely observed for forty-eight hours.

HISTORY OF CASE

The patient, a married woman, aged 45, was admitted into this hospital at 9 p.m. on November 18th, 1931, diagnosed by her doctor as a case of gastro-enteritis. She gave the following history.

At about 11 o'clock the same morning she was sitting on a chair when she was suddenly seized with acute abdominal pain. The pain was general, and not localized to any particular point. She felt sick, and about five minutes later vomited some undigested food and yellow fluid. The pain was somewhat relieved after vomiting, and an hour later had entirely disappeared. She then had an attack of diarrhoea, the motions being green-coloured and very offensive, but, as far as she could tell, they did not contain any mucus or blood, nor did she experience any pain. The diarrhoea continued throughout the day, and she gradually became weaker and more exhausted. She had taken no food except a piece of bread-and-butter and a cup of tea for breakfast, with cold water at intervals, until admission. The previous night she had had, with the rest of the household, some "fish and chips" for supper, but she was the only one to experience any ill effects afterwards. In her previous history she admitted having had a vaginal discharge twenty years ago. She had received several courses of injections, and had a blood test made a few years ago, which she had been told was positive. The history of previous venereal infection was, however, indefinite, and she gave no reliable information as to the treatment she had received. She had been operated upon for a prolapsed uterus nine or ten years ago, and after this she had been very constipated, with occasional attacks of diarrhoea. During the week before admission she had been constipated. There was no history of previous abdominal pain.

On admission her temperature was 97° F., pulse 128 and very weak, and respirations 40. She was in a feeble and exhausted condition, with marked pallor, but had no abdominal pain. The tongue was dry and coated, teeth carious, breath very offensive, and she appeared toxic.

The abdomen moved with respiration; there was no visible peristalsis, and, except for slight tenderness in both iliac fossae on firm pressure, there was nothing further of note. The whole abdomen was soft, and no rigidity was discoverable. On examination per vaginam there was tenderness in both lateral fornices, and on the left side a firm, doughy mass could be made out. There was an offensive and purulent uterine discharge. Per rectum it was found that there was an incomplete fibrous stricture of the rectum, about half an inch above the junction of the anus and rectum. It completely encircled the rectum, but the little finger could pass through it. She was passing green and offensive motions, which were very relaxed, but contained no mucus or blood. Examination of heart and lungs revealed nothing abnormal. The pupils were concentric and equal, reacting normally to accommodation, but sluggishly to light. Reflexes were generally diminished, and the left knee-jerk was not obtained. Sensation was normal.

It was thought that her condition was a pyosalpinx, and that the diarrhoea was merely one of her usual attacks caused by the presence of the stricture. She was given hot vaginal douches of potassium permanganate (1 in 1,000), a starch enema, and 1/4 grain of morphine hypodermically. She passed a restless night, sleeping for short periods, but the diarrhoea continued. A half-hourly record of the pulse was kept, and by 7 o'clock the next morning the rate had decreased to 110, and was much stronger. During the day (November 19th) the diarrhoea stopped, and until 11 p.m. on November 20th her condition improved, her pulse was better, and her temperature just below normal. After the initial dose no more morphine was given. At 11 p.m. on November 20th she collapsed, with a pulse rate of 128, temperature 97° F., and began vomiting faecal material. It was decided to open the abdomen at once.

Operation

A left paramedian incision was made, and on opening the peritoneum it was found to be gangrenous. The peritoneal cavity contained some offensive serous and blood-stained fluid. The whole of the rectum, sigmoid, and the lower half of the descending colon were completely gangrenous, the upper limit being fairly sharply defined. The remainder of the intestines were acutely inflamed. There was no volvulus, adhesion, growth, or other mechanical cause to account for the condition. It was found that the inferior mesenteric artery was pulseless, and that the blood supply to the affected gut was totally cut off, the artery with its branches being almost black in colour. A Paul's tube was tied into the transverse colon, a rubber drainage tube placed deep in the pelvis, and the wound closed. The patient, after draining well for five days, collapsed and died on November 26th.

COMMENTARY

The misleading feature of the case was the absence of a history and a clinical picture of sufficient severity to justify the findings at operation. Intestinal obstruction was considered as a diagnosis in view of the acute onset and the findings on rectal examination, but as she continually passed flatus, and the diarrhoea showed no signs of stopping, this was considered unlikely. The possibility of a tabetic crisis, the graver causes of gastro-enteritis, and lobar pneumonia (in view of the respiratory rate) had to be considered, but there was not sufficient evidence on which to make the diagnosis. The mass felt per vaginam was the gangrenous and distended gut, but there was no evidence of distension on percussion of sufficient magnitude to indicate the nature of the underlying condition. The diarrhoea, in conjunction with the incomplete stricture of the rectum, which was apparently specific in origin, accounted for the previous behaviour of the bowels. It was found impracticable to perform a post-mortem examination, but the conclusions arrived at were that an incorrect diagnosis had been made on the strength of clinical findings atypical of the condition found at operation.

We are indebted to Dr. R. Davidson, medical superintendent, St. Luke's Hospital, for permission to publish this case.

IMMEDIATE PNEUMOCOCCAL TYPING

BY

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The onset of winter and the reappearance of cases of severe primary pneumococcal pneumonia has afforded an opportunity for continuing the treatment by concentrated anti-pneumococcal serum described in the *British Medical Journal* a few months since.¹ Determination of the type of pneumococcal infection is an essential preliminary to the administration of the specific therapeutic serum.

A rapid method by which pneumococcal type could be decided within four hours was described in these columns a year ago.² The method consisted in intraperitoneal inoculation of a mouse with the pneumonia patient's sputum. Four hours later the peritoneal contents were sampled by aspiration, the fluid obtained was mixed on a slide with type-specific agglutinating serums, and type was determined by microscopical examination of the fresh mixtures.

A number of tests made during the present season have proved, so far without exception, that type can be decided with ease and certainty by direct test on the patient's sputum, without recourse to mouse inoculation.

PROCEDURE

The procedure is as follows:

A suitable fleck of sputum is selected. Three small samples of this are placed, equidistant, on a microscope