In Measles.—Fifty-three samples of blood from convalescent measles donors were tested. In six instances a positive reaction was obtained; in one instance it was ++, in two instances it was +, and in three it was ±. The strongly positive serum was retested one week later; it was found to be still +, and was discarded as unfit for use. The two serums giving + reactions were retested a week later and found to be completely negative, and were accordingly pooled with other samples for injection. All three serums giving doubtful reactions were completely negative on retesting a week afterwards. In two instances in which the reaction was + another sample of blood was withdrawn a week later, and the fresh serum was found to be negative. The blood of three patients examined on the fourth day of the disease gave a ± reaction, but on the fifteenth, sixteenth, and seventeenth days, respectively, the reaction was negative.

In Chicken-pox.—In the chicken-pox series twenty-five donors were utilized. On three occasions a frankly positive reaction was returned, while on one occasion it was doubtful. Two of the three positive serums were re-examined a week afterwards and found to be negative, while the third sample was pronounced unfit for use, as the significance of these doubtful reactions was not appreciated at the time. The serum which gave the doubtful reaction was found to be negative on retesting a week later.

In Mumps.—Six samples of serum were taken from mumps convalescents; the Wassermann reaction was negative in every instance.

The Interpretation of these changes in the Wassermann reaction is not clear, but it would appear that in the acute fevers there is an increase in the normal lipoidophile antibody content of the human serum, or a physical or chemical alteration of its constitution which permits complement-deviation to occur even in the absence of luetic infection (Mackie and Watson, 1926). This change in the serum is present only for a short time (third to twenty-first day), and rarely persists into the fourth week. Moreover, it is only found in fresh serum (first to fourth day after withdrawal) and always disappears within a week after storage at 4° C. It is not improbable that the positive reactions reported in the acute fevers by various workers are attributable to the presence of this or like substance in fresh serum, and have no relation to luetic infection.

LEAD POISONING AS A CAUSE OF PEPTIC ULCER

BY

DAVID R. LEWIS, M.D., M.R.C.P.Ed.

HONORARY PHYSICIAN FOR DISEASES OF THE SKIN, SWANSEA
GENERAL HOSPITAL

In 1839 Tanquerel published his classic treatise, which proved to be the best work on industrial plumbism in the early part of the nineteenth century; he was presented with a galaxy of material at the hospital of La Charité, Paris. From 1831 to 1839 he saw no fewer than 1,217 cases of lead colic, and so accurate were his clinical observations that little of importance has been subsequently added to his conclusions. Tanquerel mentioned that peritonitis was often a sequel of lead poisoning; in his day gastric ulcer had not become a definite clinical entity. About the same time Cruveilhier (1829-35) was investigating the morbid appearances and sequelae of gastric ulcer, and, as Bolton describes, it was Cruveilhier who gave the disease its present status in medicine. Our knowledge of the cause of gastric ulcer is still imperfect, and it is recognized that many conditions may be con-

cerned in its production. An increased concentration of hydrochloric acid in the gastric juice and general malnutrition seem to be constant factors.

RESULTS OF PRESENT INVESTIGATION

For the purpose of this investigation, which has taken ten years to carry out, over a thousand spelter workers were kept under observation. The ore from which spelter is obtained includes lead, zinc, cadmium, and traces of arsenic. Lead, zinc oxide, and cadmium are constituents of the atmosphere of a distillation-house plant. Cadmium, being the most volatile, comes off first in the process of distillation. The soil within a mile or two of the spelter works is impregnated with zinc oxide, and it has been found on rhubarb plants at a distance of two miles from the works. When our present civilization is extinguished a chemist may come along and infer that Swansea was a centre of a huge metallurgical industry. Zinc, apparently, has no toxic action on the human body. The effects of cadmium on the spelter worker are not known, or at least not distinguishable from the effects of lead. Gadamer and Arbour Stephens maintain that cadmium produces a disturbed metabolism and sets up a gastritis, which prepares the way for lead absorption. Stephens has described a case of a spelter worker who received compensation for ten years on the grounds of plumbism. At necropsy, in this particular case, Stephens found in the liver cadmium (0.91 grain to the pound) but no lead. The absence of lead in the liver was no proof that the man did not suffer from plumbism, for Minot has proved that lead may be present in large quantities in bones when no trace may be found in other organs.

Recently cases of lead absorption and poisoning have been observed in other industrial workers where cadmium is not present in the ore, and we failed to find any particular symptoms other than those usually found in the plumbic spelter worker. One hundred spelter workers were selected for investigation, and these included twenty-five that had been, or were later, certified by the medical referee as definite cases of lead poisoning. Fifty had shown signs of lead absorption, and the remaining twenty-five were apparently in good health. All the men had worked for more than a year at the spelter works, and their ages varied from 20 to 60. The Workmen's Compensation Act covers lead poisoning only, and makes no provision for lead absorption. It was therefore important from the medico-legal point of view to distinguish between lead absorption and lead poisoning. This is not always an easy task, for some workmen may show signs of grave toxæmia without even the classical signs of colic or wrist-drop. Loss of appetite, discomfort on taking food, and loss of weight are very early signs of lead absorption. Nearly all such patients complain of vague pains in the stomach, and on palpation there is invariably a tenderness in the left iliac fossa.

The blood of the 100 men was examined for punctate basophilia: Giemsa's stain was used. Of the twenty-five proved plumbic cases 80 per cent., of the fifty suffering from lead absorption 20 per cent., and of the remaining twenty-five—who complained of no symptoms—5 per cent., showed stippling of 100 or more per million red cells. The presence of basophilia, therefore, is of help only when taken in conjunction with other symptoms of lead poisoning, and its absence is no proof that a man is not suffering from plumbism. Another line of investigation adopted was the systematic testing for lead in the urine of men exposed to lead fumes and dust. In carrying out this investigation Mr. Richards, the chemist to the Swansea Vale Spelter Works, gave valuable assistance.

The urine of the thirty men of the selected 100 was examined, and lead in varying quantities was found in

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

P. BROWN, AND M. M. DEY,

M.R.C.S., L.R.C.P. M.B., Ch.B.

RESIDENT MEDICAL OFFICERS, ST. LUKE'S HOSPITAL, HALIFAX

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.