TWO CASES OF POISONING BY WEED-KILLER.

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On June 22nd I was called at 8 a.m. to see two men who, I was told, had taken poison. Upon my arrival about half an hour later I elicited the following history.

The two men, C. A., aged 63, and R. N., aged 42, who were gamekeepers, had been up all night watching the young pheasants. At about 6.15 a.m., being thirsty, the patient C. A. tasted some liquid which was in a beer bottle, and which he had been plainly told was poison! He took "one mouthful and spat it out again," and handed the bottle to R. N., "to taste," as it was "rum stuff." His companion took two gulps. They were found shortly afterwards lying on the ground, vomiting and complaining of burning pains in the stomach.

Both men were evidently very ill, vomiting small quantities of yellowish-brown material at frequent intervals. The skin was cold and clammy, and the pulse small and feeble. They were both quite conscious, and complained of the cold, the elder man walking about at intervals to get warm. There was no blistering of the lips or tongue, nor were there any "cramps."

Arsenical poisoning was diagnosed, and this was confirmed later by a telegram from the makers of the weed-killer. An emetic of warm salt and water was administered and repeated, and a dose of castor oil was given. They were ordered lime water and a solution of the ferrioxide of iron.

At midday there was no improvement; the bowels in each case had been opened. The elder man complained that he had a severe pain over the "shin" of the left leg, and that he could not see clearly.

R. N., when seen at 6 p.m., was much worse. During the afternoon he had expressed himself as feeling much better and "able to eat a square meal." He was drowsy, clammy, slightly cyanosed, and collapsed. He had vomited several times, and had passed several cholera-like motions. A hot saline enema with brandy $\frac{3}{4}$ j was ordered, and he was given brandy and albumen water by the mouth. At 9 p.m. he was more unconscious and cyanosed. His condition, in spite of injections of strychnine, champagne by the mouth, and another saline and brandy enema, became rapidly worse, and he died at about midnight.

Post-mortem Examination.

Much hypostatic staining. Slight blistering of the lips. The stomach contained about three-quarters of a pint of dark, greenish-black fluid, evidently altered blood and bile. The mucosa, covered with slimy mucus, presented patches of injection ("crimson flush") at lower end of oesophagus, at cardiac end of stomach, and to a less extent in the duodenum.

The other patient, C. A., passed a restless night owing to the pain in his left leg. On June 23rd he had marked external strabismus of the left eye with diplopia. Towards night he passed into much the same condition, only to a less degree, as the other man. The pulse, 130, was feeble and irregular. He was ordered inject. strych. $\frac{1}{4}$ grain every four hours, and a mixture of digitalis and ammonia. On June 24th it was noted that he had had three or four such attacks. The pulse was 120, of low tension and irregular. By June 26th he had improved, but the squint was very marked. He had pain in the left leg and foot, and over the middle of the peroneal area of the left leg there was a bright red patch the size of the palm of the hand. The toes were blue and cold, especially the left little toe. Left "foot-drop" was present, and an area of anaesthesia corresponding to the anterior tibial nerve could be mapped out. He improved steadily, and on July 7th the pulse was stronger and more regular. There was complete foot-drop in the left leg, and slight in the right. The little toe on the left foot had been cold, blue, and very painful for several days, and was now evidently becoming gangrenous. On July 12th (three weeks after taking the poison) the squint was less marked. The area of anaesthesia was the same. The little toe of the left foot was affected with dry gangrene. By August 4th the squint had quite disappeared, but he was quite unable to stand alone, and even with help could only just do so. There was marked wasting of all the muscles of both legs, and of the thighs. He was unable to extend the toes of the left foot, and foot-drop was complete, but he could slightly flex the toes. Right foot-drop was present, but not complete. The knee-jerks were absent. The gangrene was confined to the little toe of the left foot. The general condition was good.

The interesting points in the two cases, to my mind, are as follows:

(a) The small dose of weed-killer taken. The effects were certainly increased owing to its having been taken on an empty stomach.

(b) The early effect on the nervous system. I take it that the heart symptoms in both cases were due to the action of the arsenic on the cardiac nerves. The rapid onset of the paralysis of the left internal rectus is remark-

able. The same day that C. A. took the poison he complained that he could not see clearly, and, although no squint was noticed, it probably was present to a slight extent; anyhow it was very marked on the following morning.

(c) The occurrence of gangrene. This was very puzzling to me at first. On June 26th (three days after taking the poison) he had pain in the toes of the left foot; they were blue and cold, especially the little toe; the others gradually regained their normal condition, but the little toe became worse, and definitely became gangrenous about July 4th. It was too early for any "trophic" change due to paralysis of the nerves, but I think it can be accounted for by the very low blood pressure due to the enfeebled action of the heart.

The weed-killer was a mixture of caustic soda and arsenic.

A CASE OF MYELOPATHIC ALBUMOSURIA.

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Of late years a good deal of attention has been attracted in this country to the subject of multiple myelomata associated with albumosuria, chiefly by the writings of Bradshaw and Parkes Weber. Cases of this disease are still sufficiently rare to merit recording, even though in the present instance a *post-mortem* examination most unfortunately could not be obtained. But the Bence-Jones proteid was separated from the urine, which Bradshaw regards as "one of the most significant clinical observations which it can fall to the lot of the physician to make," since "its presence is one of the few signs of disease which can strictly be called pathognomonic."

Clinical History.

A Jew, aged 50, was admitted to the Metropolitan Hospital under my care on June 4th, 1907, with the history that he had been ailing for about a year. In February he had been obliged to keep to his bed for three weeks with pain in the hands and legs. Prior to admission he had again been confined to his bed for nearly a fortnight with pains all over. He stated that he had lost flesh. There was no history of any other important illness.

The man was anaemic; he looked ill and in pain, groaning whenever he was moved. No swelling of any bone or joint could be made out, though the ribs were very tender to even light percussion. The chest was emphysematous, and there were signs of bronchitis. The sputa was occasionally blood-stained. The area of cardiac dullness was diminished, and the heart-sounds were normal. His blood pressure was 120 mm., the pulse was small and regular. The urine passed in the day amounted to 53 oz., had a specific gravity of 1010, was reported to contain a "doubtful trace" of albumen, and showed granular and hyaline casts. Measured by Esbach's method, the amount of albumen speedily increased from a trace to 0.5 per 1,000 and then to 2 per 1,000. On June 10th the urine, which had previously been acid, became alkaline and turbid, almost milky. Microscopically many crystals of triple phosphates were found embedded in an amorphous deposit. A few pus corpuscles were also seen. A rectal examination revealed nothing.

Shortly after this Mr. Coventon, the house-physician, reported that although the albuminometer recorded 2 parts per 1,000, there was hardly any coagulum on boiling. As I found that excess of strong acetic acid had been used to acidulate with, I thought at first that this had led to the formation of some acid albumen on prolonged heating. But on reflection, the presence of the Bence-Jones albumose appeared a more likely explanation. By the procedure described below, this suspicion was speedily confirmed.

The man gradually got worse, and on July 1st hard nodules could be felt on the second right rib in the mid-clavicular line, the seventh right rib in the mid-axillary line, and on the eighth left rib in the anterior axillary line. They were firmly fixed to the ribs, and the skin over them was freely movable. It is noteworthy that the first of these swellings appeared in a spot where the pain and tenderness had for some time been particularly well marked. Hard movable glands were felt in the right axilla. The percussion note was impaired over the apex of the left lung behind, but only moist sounds could be heard on auscultation. An examination of the sputa, which was now very blood-stained, revealed many pneumococci but no tubercle bacilli. A blood count showed 7,000 leucocytes.

It was proposed to take a skiagram, but the man rapidly became so much worse that this was out of the question. On July 3rd a severe attack of diarrhoea began, and rapidly exhausted him. Two days later the dullness had extended to the base of the left lung. He was now passing very little