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Poisoning by Spent Calcium Carbide.

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It is a common practice in dairies situated near mine compounds to feed waste porridge from such compounds to the cows.

A number of cases of poisoning by such porridge have been reported and in a number of them the porridge was contaminated with spent carbide, probably due to the natives emptying the contents of their acetylene lamps into the receptacles, provided in such compounds, for waste porridge.

Spent carbide is also used to whitewash walls and other objects. This use may render it available on farms, where animals, especially those suffering from pica, may ingest it if they have access to it.

In a case, observed by one of us (S. J. v. d. W.), in which cattle, suffering from pica, ingested spent carbide, some of the animals died rapidly whilst those who survived for a few days exhibited diarrhoea accompanied by weakness of the hindquarters. Post-mortem examination of the carcases, which were unfortunately very decomposed, revealed hyperaemia of the mucosa of the abomasum and small intestine.

Kuhn, quoted by Kuscher (1938), observed geese dying rapidly after having ingested the contents of acetylene lamps.

Hoffman, also quoted by Kuscher (1938), describes a case in which fowls died after the ingestion of partially spent carbide. Post-mortem examination revealed gastritis, the mucous membrane being inflamed, swollen and brownish red in colour with areas of cauterisation. Croupous inflammation of the mucous membranes of the beak, pharynx and oesophagus was also observed.

Kuscher (1938) describes an outbreak of poisoning in hens due to the ingestion of the partially spent contents of acetylene lamps. The owner observed greenish black stains on the birds which would peck at the stains, so injuring the skin. He declared that after boiling, black streaks were observed in the albumen of many eggs laid by these hens. Two birds died 8 days after the ingestion of the carbide, the whole outbreak lasting 4 weeks. A bird examined by Kuscher revealed the following: ulceration of the skin; bluish-green to black stains on the skin; slight swelling of the mucous membrane of the beak with areas of greyish discolouration; and emaciation; the crop was empty.

Calcium carbide in the presence of water yields acetylene:—

$$CaC_2 + H_2O = Ca(OH)_2 + C_2H_2.$$

Spent carbide, therefore, consists, theoretically of calcium hydroxide, but with regard to the toxicity of spent carbide the following substances should be considered since they may all be involved in poisoning by spent carbide:—

1. *Calcium carbide.*—Spent carbide very frequently still contains a fair proportion of active carbide. Carbide is a powerful irritant. When in contact with tissues acetylene is liberated, the process being accompanied by withdrawal of water from the tissues and heat production. This, according to Kuscher, is responsible for the irritant action of calcium carbide.

   Guinard, quoted by Lewin (1929), reported in 1896 that cauterisation of the uterus was carried out in France by means of calcium carbide which slowly liberated acetylene in the uterus. Acetylene was demonstrable for hours afterwards in the urine but no general symptoms of poisoning were observed.

   In the course of experiments conducted by one of us (S. J. v. d. W.), a sheep accidentally ingested a small quantity of calcium carbide. The animal started chewing immediately, the evolution of the acetylene causing foam to appear at its mouth. Severe irritation of the mouth was immediately evident and salivation was profuse. After a few hours dyspnoea, accompanied by laryngeal respiratory sounds (râles), developed.

   On the following day salivation was still profuse. The lips, which were stained slate blue, were swollen and the mouth were very painful. The buccal mucous membrane was purplish and the tongue markedly swollen. Respiration was extremely laboured and accompanied by groaning. As was to be expected the animal was off its feed. Altogether the animal gave the impression of being in great pain and was destroyed.

   On post-mortem examination the following was observed: Necrotic inflammation of the lips, tongue, palate, pharynx, larynx and oesophagus (anterior portion); marked inflammatory swelling of the lips and tongue; marked oedema of the glottis and epiglottis.

   Microscopical examination of the tongue revealed an acute necrotic glossitis which penetrated very deeply.

2. *Acetylene.*—Panisset, quoted by Fröhner (1919), states that acetylene in contradistinction to carbon monoxide is not very poisonous since a dog kept for a few hours in an atmosphere containing 20 per cent. acetylene suffered no ill effects.

   According to Kobert (1906) acetylene has a slight narcotic action. Grehaut and his co-workers, quoted by Kobert, maintain that acetylene causes no blood changes, while Stempel, quoted by the same author, states that he has observed haemoglobinuria due to acetylene.

   At Onderstepoort 25·0 gm. of calcium carbide were administered to sheep 52875 in gelatin capsules without the animal suffering any ill effects. It eructated large quantities of acetylene.

3. *Impurities.*—According to Lewin (1929) commercial calcium carbide yields acetylene containing up to 0·06 per cent. phosphine. Furthermore there is the possibility that commercial calcium carbide may contain cyanamide which causes vomition, diarrhoea and paresis.

   Kuscher (1938) states that the absorption of impurities such as acetylene, ammonia, sulphuretted hydrogen and phosphine, originally present or formed during the decomposition of calcium carbide by water, hardly came into consideration in poisoning by spent carbide.

In the experiments with spent carbide, recorded below, there was not the slightest evidence of the presence of impurities materially contributing to poisoning by the spent carbide.

4. *Calcium hydroxide.*—Lewin (1929) reports a case in which a child was poisoned with a fresh solution of calcium oxide the symptoms being vomition and an haemorrhagic diarrhoea. He states that a solution of calcium oxide is used as a fish poison. Calcium hydroxide (slaked lime) is a fairly severe irritant.

In the following experiments the toxicity of spent carbide (completely spent) and pure Calcium hydroxide was compared.

Rabbit A.—(1·85 Kg.) received * 15 gm. of spent carbide at 11.50 a.m. on 23.1.39.

Symptoms.

23.1.39: Slight apathy; decreased appetite.

24.1.39: Received a further 15 gm. of spent carbide at 2 p.m. As on the previous day.

25.1.39: Received a further 15 gm. of spent carbide at 10 p.m.

Apathy, anorexia, loss of condition; faeces soft.

26.1.39: The animal died the previous night.

Post-mortem appearances.—Fairly advanced post-mortem changes; emphysema of the lungs; congestion of the liver and kidneys; marked hyperaemia; ulceration and necrosis of the mucous membrane of the stomach; slight hyperaemia of the mucosa of the small and large intestine; contents of the caecum and colon fluid.

Rabbit B.—(1·65 Kg.) received 20 gm. of spent carbide at 2 p.m. on 24.1.39.

Symptoms.

24.1.39: Slight apathy.

25.1.39: Received a further 20 gm. of spent carbide at 9 a.m. Apathy; anorexia, faeces soft, died fairly suddenly at 11.35 a.m.

Post-mortem appearances.—General cyanosis; hyperaemia and emphysema of the lungs; congestion of, and regressive changes in, the liver and kidneys; acute catarrhal gastritis; hyperaemia of the mucosa of the small and large intestine; contents of caecum and colon very fluid.

Rabbit C.—(1·9 Kg.) received 30 gm. of spent carbide at 11.55 a.m. on 23.1.39.

Symptoms.

23.1.39, 1 p.m.: slight apathy; respiration extremely rapid and shallow.

3.30 p.m.: Pupils contracted; accelerated weak pulse; complete paralysis.

The rabbit died at 4 p.m.

Post-mortem appearances.—General cyanosis; hyperaemia and emphysema of the lungs; congestion of, and regressive changes in, the liver and kidneys; acute catarrhal gastritis; hyperaemia of the mucosa of the small and large intestine; contents of caecum and colon very fluid.

* All the rabbits were dosed by stomach tube.

The following rabbits were dosed with calcium hydroxide (Merck) at 12 noon on 26.1.39.

Rabbit D.—(2·26 Kg.) received 5 gm. of calcium hydroxide. Result—Negative.

Rabbit E.—(1·85 Kg.) received 10 gm. of calcium hydroxide.

Symptoms.

Slight apathy; decreased appetite; faeces soft. The rabbit died 6 days after dosing.

Post-mortem appearances.—Hyperaemia, oedema and emphysema of the lungs; hyperaemia of the liver and kidneys (regressive changes, if any, masked by the post-mortem changes); slight hyperaemia of the stomach, the mucous membrane being coated with mucus; contents of colon fluid.

Rabbit F.—(1·7 Kg.) received 20 gm. of calcium hydroxide.

Symptoms.

26.1.39, 12.30 p.m.: Paresis, which rapidly progressed to complete paralysis; dyspnoea; pulse imperceptible; died at 12.40 p.m.

Post-mortem appearances.—General cyanosis; marked emphysema of the lungs; congestion of, and regressive changes in, the liver and kidneys; marked hyperaemia and necrosis of the mucous membrane of the stomach; marked hyperaemia of the mucosa of the small intestine; slight hyperaemia of the mucosa of the caecum and colon.

Rabbit G.—(1·9 Kg.) received 30 gm. of calcium hydroxide.

Symptoms.

26.1.39, 12.30 p.m.: Paresis, most pronounced in the hindquarters; dyspnoea; pulse accelerated and laboured; struggling at intervals.

1.45 p.m.: The paresis had progressed to general paralysis; severe dyspnoea; pulse almost imperceptible.

The rabbit died at 2.10 p.m.

Summary and Conclusions.

(1) Poisoning by completely spent carbide is very similar to poisoning by calcium hydroxide.

(2) So-called spent carbide may contain active carbide which, when ingested (moistened), will yield acetylene. Furthermore, other impurities such as ammonia, sulphuretted hydrogen, phosphine and cyanamide may also be present.

(3) In our experiments no evidence was obtained that any impurities of any toxicological importance were present in the spent carbide with which the experiments were conducted.

(4) Since a relatively large quantity of calcium hydroxide is necessary to poison an animal it is obvious that the calcium content of the stomach contents will be greatly increased in cases of poisoning by this substance. Consequently the determination of the calcium content of the stomach contents may be of great assistance in determining whether an animal was poisoned by spent carbide.

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