Lead poisoning
Lead poisoning follows ingestion of lead-containing materials such as material from discarded car batteries; lead-based paints are now rare.

Clinical presentation
Lead poisoning is characterised by acute brain disease. Irrespective of the rate of uptake of lead, the clinical signs of intoxication are sudden in onset and characterised by behavioural changes. Affected cattle become isolated and depressed but are over-reactive to touch and sound. They are blind but show no clinical lesions in the eyes and may press their head into a corner and against walls.

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Differential diagnoses
Your veterinary surgeon will also consider:
- Hypomagnesaemic tetany (staggers)
- Polioencephalomalacia
- Rabies

Diagnosis
The diagnosis of lead poisoning is based upon clinical signs and the presence of a lead source. Confirmation depends upon increased kidney and liver lead concentrations.

Treatment
Cattle with severe neurological signs of several days' duration may have extensive brain pathology that is unlikely to respond to treatment. Slow intravenous injections of a 5% solution of sodium calcium edentate and oral solutions of magnesium sulphate are the treatments of choice but are rarely successful once severe neurological signs are present. Your veterinary surgeon will decide whether treatment or euthanasia for welfare reasons is the better course of action, and which drugs to administer.

Prevention/control measures
Prevention is a matter of good management by not allowing cattle access to sources of lead. Regulatory authorities, such as the Food Standards Agency, may recommend withdrawal periods before milk and/or meat from exposed cattle may enter the food chain.

Copper poisoning
Toxicity may result from inadvertent dietary supplementation or incorporation of a feedstuff with a high copper content, possibly as the result of contamination. Acute toxicity is rare. Copper toxicity in cattle is much less common than in sheep. Ingestion of high copper content in the ration over several weeks/months will result in a high liver copper content. Sudden release causes an acute intravascular haemolytic crisis.

Clinical presentation
Acute copper toxicity causes severe gastro-enteritis with colic signs, diarrhoea and rapid dehydration. The affected cattle are very depressed, do not eat, and death usually ensues within three days.

In cases of chronic copper toxicity the appearance of clinical signs is associated with the destruction of red blood cells which may be precipitated by stress. Affected cattle are weak, very dull and depressed and separate from others in the group. They have a poor appetite and often foetid diarrhoea. There is jaundice (yellowing) of mucous membranes. Death is preceded by recumbency.

Differential diagnoses
Your veterinary surgeon will also consider
- Babesiosis
- Post-parturient haemoglobinuria
- Kale poisoning.

Diagnosis
The diagnosis is based upon history, a source of excess copper, and clinical findings of jaundice in chronic toxicity. The diagnosis is supported by laboratory findings of increased serum copper and liver enzyme concentrations.

Treatment
The suspected copper source must be removed immediately. There is no ammonium tetrathiomolybdate preparation licensed for use in food-producing animals and its use in suspected cases of chronic copper toxicity is poorly defined from a regulatory standpoint.

Prevention/control measures
Copper supplementation must be carefully considered after first establishing a deficiency situation.
Fluorosis
Historically, fluorosis resulted from industrial pollution of grazing land where chronic disease results after ingestion over many months/years but is rare in the UK.

Clinical presentation
Growing cattle develop mottling of the enamel and premature loss of teeth. Adult cattle show insidious onset lameness affecting the hindlegs caused by periarticular exostoses of the long bones. Fracture of the pedal bone may occur but there are other more common causes of such sudden onset lameness. There is no specific treatment for fluorosis.

Nitrate poisoning
Brassica plants and fertilisers are potential sources of nitrates.

Fig 6 Brassica plants are potential sources of nitrates.

Fig 7 Fertilisers are also potential sources of nitrates. Note storage of fertilisers adjacent to cattle feed - there is the possibility of feed contamination.

Clinical presentation
Acute poisoning with cyanosis, weak rapid pulse, and dyspnoea, is seen within hours of ingestion progressing rapidly to weakness, recumbency and death.

Differential diagnoses
Causes of sudden death your veterinary surgeon will consider include
- Hypomagnesaemia
- Lightning strike

Diagnosis
Diagnosis is based upon classical clinical signs and exposure to nitrates.

Treatment
Intravenous injection of 4 mg/kg methylene blue as a 2 per cent solution.

Organophosphate poisoning
Overdosage and accidental exposure to organophosphates lead to toxicity.

Fig 8 Failure to correctly dispose of concentrated dip (not an OP in this case) can lead to toxicity.

Clinical presentation
Profuse salivation, colic and diarrhoea are followed by muscle tremors, stiffness progressing to paralysis. At this stage cattle show marked depression with increasing severity of colic, sweating and breathing difficulties followed rapidly by death.

Diagnosis
Based upon clinical signs and history of exposure to/treatment with organophosphates.

Treatment
Atropine sulphate (0.1 mg/kg injected slowly intravenously followed by 0.4 mg/kg injected subcutaneously) is repeated as necessary.

Prevention/control measures
Correct storage and disposal of empty containers containing organophosphates.

Urea Poisoning
Definition/overview
Urea is used as a source of non-protein nitrogen (NPN) in feed supplements especially in beef cattle. Nitrogen from urea is released in the rumen as ammonia in the rumen. Accidental urea intoxication occurs sporadically and can cause severe losses. One incident resulted in the death
of 17 of 29 suckler cows within six hours after the contamination of their drinking water with urea fertiliser.

**Aetiology**
Poisoning episodes typically occur after sudden access to urea, which may simply involve only a break of several days' supply then free access. Urea is highly soluble and will wash out of the diet/feed blocks following heavy rain then cattle may drink the puddles with a high urea content.

**Clinical presentation**
Signs of urea poisoning can appear within several hours and include muscle twitching, teeth grinding, frothy salivation, bloat, colic, frequent urination, forced rapid breathing, staggering, bellowing, and terminal seizure activity. Often, animals are found dead near the source of the urea supplement.

**Differential diagnoses**
Your veterinary surgeon will also consider:
- Botulism
- Hypomagnesaemia
- Anthrax
- Clostridial disease such as Blackleg

**Diagnosis**
Diagnosis is based upon a history of access to urea, and the clinical signs.

**Treatment**
50 litres of cold water then several litres of 6 per cent vinegar have been recommended.

**Prevention/control measures**
- Ensure thorough mixing of the ration.
- Gradual introduction to urea feeding with an uninterrupted supply

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