Craig (1995) explained the dual role of rumen microbes in first producing the toxin and latter degrading them.

Hydrocyanic Acid Poisoning

Most outbreak of HCN poisoning is caused by ingestion of plants, which contain cyanogenic glycosides [amygdalin (laetrile) and prunasin found in kernels of almonds, etc., dhurrin that occurs in sorghum species and linamarin that occurs in flax (linseed), cassava). The commonly found plants are sorghum, sudan grass, jhonson grass and linseed cake or meal. Cyanogenic glycoside content varies with the age of plant, environmental condition, nutritional status and genotype of the forage crop. Immature sorghum fodder contains a cyanogenic glycoside called "dhurrin" and this liberates hydrocyanic acid (HCN)/prussic acid on hydrolysis.

1. Cyanogenic glycoside $\xrightarrow{\beta\text{-glucosidase}}$ sugar + aglycone

2. Aglycone $\xrightarrow{\text{hydroxynitrile lyase}}$ HCN + aldehyde or ketone

The glycosides occur in vacuoles in plant tissue, while the enzymes are found in the cytosol. Damage to the plant from wilting, trampling, frost, drought, bruising (cassava), and so on results in the enzymes and glycosides coming together, causing HCN to be formed. These enzymes are also produced by rumen microorganisms. The optimum pH for the enzymes is near neutrality, so release of HCN is more rapid in the rumen than in the highly acid stomach of the nonruminant animal. That is the reason ruminants are more sensitive to cyanogens than the nonruminants.

Leaves of render plants contain maximum poison. It is recommended that sorghum green fodder is harvested after 60 days of planting to avoid HCN poisoning. Rainfed crops contain more poison than those under irrigation. The glucoside content is highest when plants grow rapidly after a previous period of retardation. Thus wilted, frost bitten and young plants are likely to be more poisonous than the normal, mature plants.

HCN is readily absorbed and enters individual tissue cells. It inhibits cytochrome oxidase, the terminal step in electron transport. When cytochrome oxidase is blocked, ATP formation ceases, and the tissues suffer energy deprivation.

Commonly HCN poisoning is always acute and affected animals rarely survive for more than 1-2 hours due to dysfunction of electron transport in the cytochrome system. The onset of signs is within 2-3 minutes and the signs may be delayed if the ingested materials are relatively indigestible.

Symptoms

The common clinical signs include dyspnea (difficult or laboured breathing), anxiety, restlessness, stumbling gait, tremor, moaning, recumbancy, convulsions with opisthotonus and sudden death.

The blood of the affected animal becomes bright red due to suspended oxygen exchange and oxygen retention in blood. In prolonged course, it becomes dark red colour. The odour of benzaldehyde or acetone may be detectable in the contents of the rumen if the dead animal is examined immediately. A level of HCN 0.63 ug/ml justifies a diagnosis of poisoning.

Treatment

Cyanide is readily detoxified, so acute toxicity occurs only when HCN levels are more.Liver, kidney, and thyroid tissue contain an enzyme called rhodanese (thiosulfate sulfurtransferse), which catalyses conversion of cyanide to thiocyanate. Thiocyanate is excreted in the urine. This reaction is employed in the treatment of cyanide toxicity.

The standard treatment of intravenous injection of a mixture of sodium nitrite and sodium thiosulphate will give good recovery (5 g sodium nitrite, 15 g sodium thiosulphate in 200 ml water for cattle, 1 g sodium nitrite, 3 g sodium thiosulphate in 50 ml water for sheep). Sodium thiosulfate participates in the reaction, while nitrate converts haemoglobin to methaemoglobin. Sodium nitrite or sodium thiosulphate can be administered alone or together to produce low-level methaemoglobunemia. Methaemoglobin has a strong affinity for cyanide and will bind the cyanide molecule and prevent inhibition of the cytochrome oxidation system. It was concluded that sodium thiosulphate without nitrite was an effective antidote for cyanide poisoning. Dosage is 660 mg/kg body weight. Sodium thiosulphate should be given orally to fix the HCN in rumen at the dose of 30 g in cattle, 6 g in sheep and it is repeated at hourly intervals tills the signs disappear.