Chemical poisoning in animals. 3. Prussic acid

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HYDROCYANIC or prussic acid poisoning is a serious and not uncommon ailment of ruminants, and may be a cause of rapid death when pasture plants capable of developing dangerous levels of the poison are being grazed.

Many of these plants are valued for their quality and nutritional value. Fatalities from these can be avoided by adopting appropriate methods of husbandry and grazing control.

Sources:
In Australia, the most important source of prussic acid is from cyanogenetic (cyanide producing) plants, particularly in their early stages of growth or re-growth, or following physical damage by frost, disease, bruising and so on. Notable in this group are plants of the genus Sorghum, of which Johnson grass is a notorious example.

Below is given a check list of plants which are capable of developing toxic levels of prussic acid. It should be remembered that the list is incomplete, as there are many species which are only suspected of being toxic from circum-stantial field evidence, and have not been proved to be cyanogenetic.

Cyanide poisoning can also result from the ingestion of chemical cyanide compounds, but this is comparatively rare in Australia.

CHECK LIST OF SOME KNOWN CYANOGENETIC PLANTS

Acacia dillatata Benth.
Acacia glaucescens Willd.—Sally Wattle.
Acacia longifolia Willd.
Andrachne decaisnei Benth.—Andrachne.
Anthemis species—Stinking May Weed.
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Aquliegia vulgaris Linn.—Columbine.
Brachyachne convergens (F. Muell.) Stapf.—Spider grass.
Brachyachne tenella (R. Br.) C. E. Hubbard—Native or Kimberley Couch grass.
Bridelia ovata Dene.
Calotis scapigera Hook.
Canthium vaccinifolium F. Muell.—A member of the Madder family.
Chenopodium species—Goosefoots including the Crumb Weeds, and the Fat Hen (known locally as Mint Weed).
Chloris distichophylla Lag.—Winter Rhodes Grass.
Cynodon dactylon (L.) Pers.—Couch Grass.
Dactyloctenium radulans (R. Br.) Beauv.—Button Grass.
Digitaria sanguinalis (L.) Scop.—Summer Grass.
Dimorphotheca species.
Eleusine indica (L.) Gaertn.—Crow's Foot Grass.
Eremophila maculata F. Muell.—Native Fuchsia or Spotted Berrigan.
Eriobotrya japonica Lindl.
Eschscholtzia species—California Buttercup.
Eucalyptus cladocalyx F. Muell.—Sugar Gum.
Eucalyptus viminalis Ldbill.
Euphorbia species—Balsams, Spurges, Caustic Creeper, Asthma Plant, Geraldton Carnation Weed.
Goodia lotifolia Salisb.—Clover-Leaved Poison, Clover Tree or Golden Tip.
Grevillea species—Grevilleas.
Gyrostemon ramulosus Desf.—Cork Wood.
Hakea species—Hakeas.
Haloragis species—Raspworts.
Heterodendron oleifolium Desf.—“Minga” (Gascoyne and Murchison Districts) Rosewood.
Hydrangea species—Garden Hydrangea.
Indigofera australis Willd.—Southern Indigo, Australia Indigo.
Lambertia species.
Linum marginale A. Cunn.—Wild Flax.
Linum usitatissimum Linn.—Linseed or flax (Linseed cake, Linseed meal).
Lotus species—Birds Foot Trefoils.
Loudonia roei Schlecht.—Pop Flower.
Macadamia ternifolia F. Muell.—The Queensland Nut.
Nerium oleander Linn.—Garden Oleander.
Panicum maximum Jacq.—Guinea Grass.
Passiflora species—The Passion Flowers and Passion Vine.
Phaseolus lunatus Linn.—Sieva Bean.
Phyllanthus gasstroemii F. Muell.—Lagoon Spurge.
Poranthera microphylla Brongn.
Prunus laurocerasus Linn; Prunus persica Stokes; Prunus armeniaca Linn.—Members of the plum family.
Sambucus nigra Linn.—Elder.
Sorghum halepense (L.) Pers.—Johnson Grass.
Sorghum sudanense Stapf.—Sudan Grass.
Sorghum verticilliflorum Stapf.—Wild or Native Sorghum.
Sorghum vulgare Pers.—Milo or Grain Sorghum.
Trifolium repens Linn.—White Clover.
Trigonella species.
Xylomelum angustifolium Kipp.—The Native Pear or Sand Plain Pear.
Ziera species.

Toxicity:
There is considerable variation in the toxicity at different stages of growth of these plants. It has been shown that there is a higher content of cyanide producing principle in the very young stages of growth and in young shoots or regrowth. Mature plants do not usually contain high levels of the poison. Physical damage, such as may occur with frost, hail, drought, trampling or wilting may also release the poison from the plant cells. Spraying with the hormone herbicides such as 2,4-D, 2,4,5-T and M.C.P. also increases the toxic hazard. Most of these plants contain dangerous quantities of cyanogenetic glucosides only under special conditions, and others, for example White Clover, only rarely.

Fresh growing plants do not contain prussic acid in the free form. It is usually present combined with a sugar to which it is chemically “locked” as a cyanogenetic glucoside. Therefore an enzyme or “key” is necessary for its release before poisoning can occur. In some cases, this “key” is present in the plant or in the animal’s rumen (paunch). In other cases, physical agents release the prussic acid in a manner analogous to this “lock” being broken by force.

Poisoning is basically influenced by five factors:

1. The amount of plant eaten.
2. The rate at which it is eaten.
3. The level of poison it contains.
4. The presence of an appropriate enzyme or key to release the prussic acid.
5. The rate at which the poison is de-toxified (rendered harmless) by the body.

The levels of prussic acid vary widely between different plants, between different stages of growth, and between different pastures of the same plant at the same stage of growth. There is no reliable way of being sure that a particular pasture will not be poisonous. Unless the combination of the above five factors pre-disposes to poisoning, animals may graze potentially toxic pasture without ill effects. Therefore, avoiding the grazing of pasture at those stages when it is likely to cause death is the surest way of preventing losses.

Linseed cakes and meals contain residues of cyanide which are normally harmless, since the temperature developed in their manufacture is usually sufficient
to destroy most of it. However, if starved animals are fed on linseed products, poisoning may possibly occur.

**Symptoms:**

When large amounts of a toxic plant are eaten rapidly, death may occur before symptoms appear. With smaller amounts, symptoms may be seen as early as ten minutes after animals commence grazing, but usually within the first hour.

There is a sudden onset of breathlessness with deep and rapid respiration. The animal goes down and may assume a posture similar to that seen in milk fever with the head turned round towards the flank. Both sheep and cattle are nearly always bloated in the final stages of the condition and the actual cause of death is an internal suffocation, since the tissues are unable to use the oxygen supplied by the blood. Death follows after a short period of convulsions.

If less than the toxic amount of prussic acid is released or eaten, these symptoms will be prolonged and the animal may recover. The mucous linings of the mouth and the eyes will at first appear bright red, later changing rapidly to the purple-blue colour so typical of cyanide poisoning. The smell of “bitter almonds” may sometimes be detected in the breath. Diarrhoea occasionally results from an irritant gastroenteritis.

**Laboratory confirmation of toxic levels of prussic acid may be made if from 4 to 8 ozs. of fresh green plant specimens or rumen (paunch) contents are immediately forwarded, tightly sealed or corked in a clean airtight bottle, to the Animal Health Laboratory, Department of Agriculture, Jarrah Road, South Perth.**

**Post Mortem:**

When the blood vessels are cut at post mortem, the blood shows a characteristic cherry red colour on exposure to air. Small haemorrhages are often seen in the lining membrane of the windpipe and larger air passages and there may be a frothy blood-stained discharge from the mouth. Congestion and haemorrhages on the lungs and inner linings of the abdominal wall may also be seen, and this is usually accompanied by bloating of the paunch.

When plants containing high levels of cyanide are eaten, there may be severe inflammation of the abomasum (fourth stomach) and the first few feet of small intestine; this is indicative of a gastroenteritis. The aroma of “bitter almonds” may be noticed arising from the contents of the first stomach.

**Treatment:**

Usually death ensues so rapidly following the first appearance of symptoms that nothing can be done. However, the treatment of surviving animals should be started immediately, giving priority to those showing less severe symptoms, and to pregnant and valuable stock. Urgency of attention is essential since any waste of time may mean the difference between life and death. Veterinary assistance should be sought wherever it is available. Nevertheless animals exhibiting very severe symptoms will probably die despite treatment.

All livestock should be moved from toxic pasture immediately, preceded by treatment if necessary.

Compounds of sulphur have been shown to be the main chemicals involved in the conversion of cyanide to non-toxic forms. Drenching with sodium hyposulphite (photographer's hypo) in water provides an extra source for the detoxification process.

The dose of “hypo” for cattle is two ounces dissolved in one pint of water, but sheep should be given only one fifth this amount. This drench should be repeated in half an hour if symptoms do not abate. Should bloating occur and swallowing become difficult, relief may be obtained by puncturing the left flank with a trocar and cannula, and subsequently administering the “hypo” directly into the rumen through the cannula.

The subcutaneous injection of sulphuric ether has also been used in the treatment of prussic acid poisoning, a 10 cc. dose being administered to cattle and a 5 cc. dose to sheep. The dose can be repeated in 30 minutes if necessary.

**Prevention:**

Since it is the early stage of plant growth which is toxic, the grazing of this should be deferred for six to eight weeks. Where conditions such as rapid regrowth,
hail, or wilting have increased the risk of poisoning, grazing should be avoided or preceded by feeding good quality hay or crushed grain. A pilot group of less valuable stock may be used to test the pasture, and the observation of these animals for a few hours will serve as a guide to toxicity. They should be removed and given attention at the first signs of bloating or difficulty in breathing. It is also advisable to have on hand a quantity of sodium hyposulphite for treatment purposes. The dried hay from these pastures is not usually toxic.

References:
Gardner and Bennetts.—“The Toxic Plants of Western Australia” (1956) W.A. Newspapers Ltd.
Hungerford.—“Diseases of Livestock” (1953) 3rd Ed. Graham Book Co.

GROW BETTER BACON

PIG raisers can learn a great deal about producing better bacon by entering the annual Baconer Carcass Competition.

What makes this competition so important to commercial growers is that a pig good enough to win the competition will be good enough to bring top prices when sold.

The display this year will consist of a side and separate ham end from each prize winning entry, and a separate ham end from every other carcass entered.

The usual function at the West Australian Meat Export works will be held on Friday September 15 instead of the Friday of Royal Show week. Growers are urged to attend this, as the display and talks on the standard of entries are possibly the most useful aspects of the competition.

The weight range of carcasses is particularly important. This has been laid down as from 120 to 126 lb. chilled dressed weight, and carcasses outside this weight range will be disqualified from judging.

This chilled weight range should be achieved if entries are forwarded within the liveweight range of 170 to 200 lb. On the basis of killing out at 72 to 73 per cent., pigs slaughtered within this range should comfortably conform to the required standard.

A wise precaution is to weigh the pigs before sending them to the treatment plant—if scales are available.

Pigs entered for the competition must be sired by pure bred boars, although the sows need not be pure bred. Each competitor may submit two entries, and awards will be made to the 10 highest scoring entries.

The basic aim of this competition is to stimulate pig raisers to produce a top quality product, to be appraised under an approved system of judging. The results help the grower to make a fairly accurate assessment of the standard of management, feeding and husbandry needed to grow a pig whose carcass conforms to the required standard.

As well as allowing the grower to see how his pigs are doing on his own farm, the competition gives him a chance to compare his product with the best—those which bring the top prices. He also gets a better idea of what the processor wants.

Entry forms are available from the Australian Meat Board office, the Department of Agriculture, and your District Adviser. Receival of entries continues until August 26, and all entries this year will be received and treated at Watsons’ bacon factory, Spearwood.
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It contains a series of authoritative articles written by experts in various fields of agricultural economics, and by farmers themselves, and is prepared by The Agricultural Economics Group at the University of Western Australia.

Articles in "Farm Policy" discuss economic problems of current importance to agriculture. For example, the main feature in the current issue is a section on the proposed reserve price plan for wool marketing.

Further information on "Farm Policy" can be obtained from the University’s Agricultural Economics Group.

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<th>Discharge in G.P.M</th>
<th>Diameter in Feet</th>
<th>Rainfall Equivalents Expressed as inches per hour</th>
<th>Sprinkler spacings in feet arranged in order of area covered</th>
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<tr>
<td>3/16 x 1/8</td>
<td>35</td>
<td>6.4</td>
<td>93</td>
<td>1.35 - 0.90 - 0.54 - 0.45 - 0.40 - 0.35 - 0.30 - 0.28</td>
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<td>7.4</td>
<td>93</td>
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<td>8.3</td>
<td>97</td>
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