Gifblaar Poisoning.

A summary of our present knowledge in respect of poisoning by *Dichapetalum cymosum*.

By D. G. STEYN, B.Sc., Dr. Med. Vet., Research Officer, Onderstepoort.

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Introduction.

Gifblaar is one of the most deadly stock poisons in the sub-continent. As explained verbally by Theiler, he was the first to confirm the general belief amongst farmers that gifblaar was poisonous to stock. In 1902 he fed the plant to oxen and rabbits with fatal results. These results, however, were not published. In 1906 Dunphy experimented with gifblaar on sheep and goats, and was the first to record the symptoms of gifblaar poisoning from his own experiments. The last reference is that of Stent (1916), and from that date a considerable amount of work has been carried out at Onderstepoort. It is of importance to draw attention to the fact that the experiments of Theiler, Neser, Green, and Curson were carried out at Onderstepoort, and the results must be considered as from the species of gifblaar growing under the climatic conditions at Pretoria.

Synonyms, description of plant, distribution (see appendix).

Historical.

There are but few references to gifblaar poisoning in South African literature. Amongst those who referred to this poisonous plant are Burtt-Davy, Dunphy, Walsh, Marloth, and Stent. The information given by these writers is, relatively speaking, meagre, and in most cases defective and inaccurate. There seems to be no doubt, however, that the authors were quite satisfied that the plant in question was a deadly poison to live stock. Thus it may be quoted from a few authors as follows:—

Dunphy, from his own experience, concludes "that the poison acts on the nervous system and has little, if any, effect on the bowels," and as regards post-mortem lesions that "diagnostic appearances are absent." Walsh states that two or three leaves are sufficient to poison an ox, but other accounts say that a far greater quantity of old leaves is required unless the ox drinks water after eating the plant. He maintains that, since it has been discovered that gifblaar leaves contain a glucoside, which, when the leaves are moistened with water, produces prussic acid, this is the reason why the toxicity of the leaves is increased when the animals drink water after having ingested the plant. It was, however, subsequently proved that no glucosides yielding prussic acid were present in the leaves (see Bulletin of the Imperial Institute, Vol. XIV, No. 1, 1916). This was later on confirmed by Neser and Green.

Furthermore, Stent (1916) gives a good botanical account of *Dichapetalum cymosum*, including for the first time a description of the underground stem and root.

Curson's work on gifblaar seems to be summarized in a report written by him on the 2nd December, 1919.

From the practical point of view, however, there seems to be no doubt whatever that *Dichap. cymosum* was recognized as a deadly poison by the early Voortrekkers who entered the Transvaal. It is probable that the natives called the attention of the Voortrekkers to this poisonous plant. The very name given to *Dichap. cymosum* by the Dutch Voortrekker, namely, gifblaar (poisonleaf), is ample proof of this contention.

Toxicity.

All the investigations proved that young leaves are much more poisonous than old ones, but nothing definite was known. This unsatisfactory state of affairs was fully appreciated by the Division of Veterinary Research and Education in 1920, and for this reason Neser undertook experiments in order to obtain more direct information, particularly in respect of the economical side of the problem. The necessity for this will be obvious by reference to the appendix, but for the sake of making the matter quite clear to those people who are not acquainted with the conditions in the Transvaal, the position may be summarized as follows:—

The plant may be found anywhere on the northern slopes of hills north of the Magaliesberg. On a very large number of farms the plant occurs in patches over such proportions of the farm that fencing was not practicable, and if undertaken would have impaired the carrying capacity of the farm to a very considerable extent. It therefore became imperative to ascertain by accurate observations and by experimentation to what extent this plant would prove dangerous throughout the year. To this end experiments were carried out as follows: (*a*) Sheep and cattle were offered gifblaar *ad libitum*, and the amounts eaten were ascertained by weighing the amount offered and the amount not taken. It was thus established that sheep and goats rarely took more than about 3 oz. and cattle rarely more than 9 oz., even after a period of starvation. (*b*) A standard weight of leaves collected at haphazard from a fixed locality was fed to cattle and sheep at intervals of a week. The fixed dose was 4 oz. for sheep and goats and 1 lb. for cattle. The results of these experiments are summarized in the following table:—

Table showing mortality in Sheep drenched with 4 ozs. of leaves macerated in water.

The date refers to the collection of the leaves.

Year.	January.				February.				March.				April.				May.				June.				July.				August.				September–October.								November.				December.			
1920.....																															+	+	+	+	+	+	+	−	−	−	−	−	−	−	−	−	−	−
1921.....	−	−	−	−	−	−	−	−	+	+	+	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	−	−	−	−	−
1922.....	−	−	−	−	−	−	−	−	+	+	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	−	−	−	−	+	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−
1923.....	−	−	−	−	−	−	−	−	+	+	+	+	+	+	+	+	+	−	−	−																											

\+ = Fatal result.
− = No effect on animal.
Each month is divided into four periods

It is evident from the above table that the dose fed proved to be toxic during the two periods of the year, viz., in Spring (from the middle of August to the end of November) and in autumn (from the beginning of March to the end of May). This was first established by Neser during the period September, 1920, to March, 1923, and subsequently confirmed by Theiler. The importance of the results obtained will be dealt with fully in a final discussion. It is only necessary here to record a few other facts which were established during the experiments referred to above, namely (*a*) that the statement that young leaves were more toxic than old ones was fully confirmed by Curson, Neser, and Theiler; (*b*) that the toxicity of young leaves may be so great that $\frac{3}{4}$ oz. would prove fatal for a sheep or goat and 3 oz. for an ox; (*c*) that the toxic principle was readily soluble in water and that a watery extract of the leaves removes practically all of it. This was proved to be the case by experimentation both on cattle and sheep—indeed the results are most convincing, as the following example will illustrate:—

A bag full of the plant was collected and proved to be fatal to cattle in amounts of 3 oz. About 10 lb. of leaves were ground up and macerated with water for three days, the old water being poured off after 24 hours and fresh water being substituted. The residue was then washed and all the water carefully squeezed out of it. This material was used for drenching an ox. The ox actually received residue equal to 4 lb. of original leaves without showing any alarming symptoms.

The Toxic Principle.—Practically nothing is known about the toxic principle. At first it was thought (Walsh) that hydrocyanic acid was responsible for death, but, as will be seen, none was found in the leaves examined at the Imperial Institute and subsequently by Neser and Green.

Plant material was dispatched to the Imperial Institute, London, for examination, and the following is the report in the Bulletin of the Imperial Institute, Vol. XIV, No. 1, 1916:—

"Examination at the Imperial Institute of material collected at different stages showed that glucosides yielding prussic acid were absent, and no poisonous alkaloid could be detected. On extraction with alcohol, the leaves yielded a mixture of two resins, one easily soluble and the other soluble with difficulty in this solvent; neither of these resins could be obtained in a pure and well-defined state. Both products were examined physiologically by Prof. Cushny in 1909, and both proved to be poisonous to animals."

Green, of this laboratory, made a cursory examination of the plant in 1917, and observed that the toxic principle was thermostabile, readily soluble in water and in aqueous alcohol, but not in absolute alcohol; was not precipitated by basic lead acetate nor by any of the usual alkaloidal reagents such as phosphotungstic acid, and could not be separated by shaking out with ether or chloroform either in alkaline or acid solution. By successive removal of foreign aqueous extractions with alcohol, lead acetate, and phosphotungstic acid, a clear pale yellow syrup could be obtained of which 0·1 gm. per kilo bodyweight by subcutaneous injection was fatal for the rabbit within half an hour. At this state the toxic principle was still contaminated with sugary material, from which it could not easily be separated owing to the similarity in solubility of toxin and sugars. The work

was laid aside at this point, and has not been taken up again since. Green also showed that all portions of the plant, including the fruit, contained the toxin. Young leaves contained most, extract equivalent to a few grams being fatal for large rabbits. Old leaves contained very much less, but even twigs and roots contained traces. The free solubility of the toxic principle in water and almost complete insolubility in absolute alcohol render the term "resin" as used by the Imperial Institute workers an inappropriate one, and at the present moment practically nothing can be said concerning its real nature.

Method of Feeding.—Gifblaar leaves mixed with ordinary food were given to the animals, and those animals unwilling to take it in this way were drenched with a maceration or watery extract of the leaves.

Symptoms.—Under natural conditions donkeys, cattle, sheep, and goats are most susceptible to gifblaar poisoning, as other animals never or very seldom eat the plant.

(a) *Cattle* (Theiler).—In cattle, which show symptoms very similar to those in sheep and goats, these may appear within twelve hours after ingestion of the plant. The animal lies down with the head backwards, and when forced to rise it staggers, lifting the feet abnormally high, urinates very often, and immediately lies down again. When standing, the front legs are held well forward and the hind legs tucked under the body. The heart-action is increased, the pulse is very soft and hardly perceptible. The respirations are increased, shallow in nature and the animals sometimes moan on expiration. Water and food are refused and rumination is absent. The animal shows great uneasiness, getting up and lying down and moving from side to side. Pronounced symptoms of hyperaesthesia are present. Furthermore, there is a quivering of the muscles, especially those of the shoulder, and all the reflexes are exaggerated. The vision of the animal is impaired, and if it was allowed to walk it did not avoid objects, but walked straight up against them.

Salivation is increased, and the animal very often grinds the teeth, and therefore a frothy discharge comes from the mouth. After some time the animal gets very dull, the ears are drooping, and it is unable to rise. Diarrhoea seldom occurs. Death may occur as soon as twelve hours after the first symptoms appeared.

Cases of chronic gifblaar poisoning have not been observed.

(b) *Horses.*—Neser experimented with one horse, which unfortunately died during the night, so that no symptoms were noted.

(c) *Sheep and Goats* (Neser).—Symptoms may appear as early as six hours after ingestion, but if water is withheld the symptoms may be delayed for 36 to 48 hours. The animal stops feeding. It becomes very uneasy, getting up and lying down repeatedly, and moving from side to side. This is soon followed by nervous symptoms, such as hyperaesthesia, partial loss of vision, and quivering of the muscles. During this stage most of the reflexes are exaggerated, e.g. each time the animal gets up it urinates, and a sudden noise causes the animal to start. Its movements are now uncertain. It walks with a swaying gait, lifts the feet abnormally high, and the animal tends to lean up against walls or trees. Later on it becomes markedly distressed, bleating repeatedly and continuously moving the body and legs. The salivation is somewhat increased. The pulse is accelerated, and may count over 180 per

minute. At first it is full and fairly hard, but later on it becomes soft and even thready. During this stage cyanosis of the mucous membranes may be observed. In some cases the peristaltic movements become somewhat increased, and symptomatic diarrhoea may appear. These symptoms progress until the animal becomes comatose and dies, usually shortly after galloping movements of the limbs.

Very rarely an animal recovering after showing the above symptoms may show an indefinite train of symptoms for a period of a week or even longer. There is a marked inappetence, absence of ruminal movements, very sluggish intestinal movements, uncertain, staggering gait, disinclination to move, and a staring appearance of the eyes, accompanied by rapid loss of condition. The thirst of the animal is also increased.

Course.—When large amounts have been ingested, the animal may die within six hours after the first appearance of the symptoms or may linger for some days and then die or recover. The remote symptoms may appear within eight days after the ingestion, and may then last for a week.

Similar symptoms in sheep and goats were described by Dunphy, who carried out his experiments in October and November, 1905.

As can be seen from the above, there is a marked similarity between the symptoms in cattle and those in sheep and goats.

(d) *Dogs* (Neser).—Symptoms may appear in as short a time as four hours after administration. The most constant is vomiting. If the animal does not die soon, vomiting is followed by retching, and this symptom may persist for as long a time as eight days. In such cases diarrhoea, sometimes of a bloody nature, supervenes about twelve hours after the poison was ingested, and may persist for a couple of days. The action of the heart is affected as in sheep and goats, but becomes thready at a very much earlier stage, so that cardiac symptoms (e.g. cyanosis) are more marked. There is a slight hyperaesthesia present.

(e) *Rabbits and Guinea-pigs* (Curson).—In rabbits and guinea-pigs the symptoms vary, the one animal showing signs of marked hyperaesthesia and uneasiness and the other those of depression.

Differential Diagnosis.

From the information previously given, it would be clear that the locality and the time of the year is of the utmost importance in diagnosing gifblaar poisoning.

(a) *Anthrax.*—In peracute cases of gifblaar poisoning, where the animals are found dead in the veld, anthrax must be taken into consideration. The correct diagnosis, however, can easily be made by taking blood-smears; furthermore, on post-mortem the unchewed leaves of gifblaar will be found in the rumen. Of further importance is that gifblaar poisoning occurs in spring and autumn only.

(b) *Slangkop and Tulp Poisoning.*—The most important factor of differentiation is that in slangkop and tulp poisoning there is a marked diarrhoea, which is a very rare symptom in gifblaar poisoning. Furthermore, in the former case there are marked inflammatory changes in the intestines, which are absent or much less marked in the latter. In acute cases the unchewed leaves of the plant responsible for the poisoning will be found in the stomach. In slangkop poisoning the pulse rate is abnormally low. Further points

of importance are the locality and the time of the year in which the poisoning occurs, as slangkop and tulp are poisonous throughout the year, which is not the case with gifblaar.

(c) *Heartwater.*—The galloping movements of the limbs and the throwing back of the head shortly before death resembles the attitude often seen in heartwater, but the post-mortem, locality, and time of the year will easily permit a correct diagnosis. Where doubt exists, heartwater can easily be recognized by injecting blood from the patient into susceptible sheep or goats.

Post-mortem Appearances.

External.—Sometimes lesions are present which show that the animal was in distress before death.

Internal.—Dunphy (1906) described the symptoms in sheep as follows: "Diagnostic appearances are absent. Sometimes there is a slight congestion of the bowls, although the brain is invariably congested. The leaves are easily found in the stomach."

The following are the post-mortem symptoms of gifblaar poisoning in the different species of animals:—

(a) *Horses* (Neser).—Hydropericard, subepicardial and subendocardial hæmorrhages, slight hyperaemia of the lungs, spleen, and kidneys, and a slight catarrhal gastro-enteritis.

(b) *Cattle* (Theiler and Neser).—Ascites, hydrothorax, subepicardial and subendocardial hæmorrhages, degeneration of myocard and kidneys, slight hyperaemia and oedema of lungs, tumor splenis, hyperaemia and degeneration of the liver and an acute catarrhal, seldom hæmorrhagic, gastro-enteritis.

(c) *Sheep and Goats* (Neser, Theiler, and Curson).—Cyanosis, slight hydropericard, subepicardial and subendocardial hæmorrhages, degeneration of the myocard, hyperaemia and oedema of the lungs, slight hyperaemia and degeneration of the liver and kidneys, and a slight hyperaemia of the brain. A slight catarrhal gastro-enteritis.

(d) *Dogs* (Neser).—In very acute cases only hyperaemia of the stomach can be seen and cyanosis. In cases persisting for a few days before death occurs, a marked catarrhal, sometimes hæmorrhagic, gastro-enteritis can be present.

(e) *Rabbits and Guinea-pigs* (Curson).—A slight congestion of the stomach and small intestines.

Microscopical Pathological-anatomical Diagnosis (*Theiler*).

Kidneys.—In several cases infarct-like, V-shaped anæmic areas were seen in the cortex. In these areas the staining of the tubuli and Malpighian bodies were fainter than that of the adjoining tissues. These changes in the kidneys can be described as a localized necrobiosis.

Treatment.—This is purely symptomatic. Dunphy (1906) reports that he cured several sheep, which have been practically at the door of death, with hypodermic injections of ether as stimulant and arecoline to clear the bowels.

In all cases of plant poisoning further absorption of the poison must be prevented, and it is therefore advisable to use the stomach tube and to administer a rapid-acting purgative, e.g. arecoline subcutaneously, demulcents, e.g. linseed oil, per os, and to keep the animals away from water. Furthermore, large quantities of physiological solutions of common salt, intravenously, and diuretics should

be given. As heart stimulant digitalis preparations can be used. The nervous symptoms can be treated with sedatives such as chloral hydrate per os and chloroform.

Eradication.—(See appendix).

General Discussion.—As has already been stated, only cattle, donkeys, sheep, and goats are susceptible to gifblaar poisoning under natural conditions, but it has been proved experimentally that all the other domesticated animals are equally susceptible to this plant poison, only they do not ingest the plant under natural conditions.

A noteworthy fact is that, owing to the tastelessness of the gifblaar leaves and buds, animals which grew up in areas where gifblaar is prevalent, are always liable to get poisoned too; a fact which never, or very seldom, occurs with other poisonous plants, which have a taste, as the animals very soon get to know their taste, and accordingly avoid them.

Furthermore, the two periods of marked toxicity of gifblaar coincide with the periods of formation of young leaves, which were known to be much more toxic than the old leaves. Up to the present time the old leaves have not yet been proved toxic; fairly big quantities of old leaves were fed to animals (Curson, e.g. fed 4 lb. to cattle), but none died or showed symptoms of ill-health. To prove the toxicity of the old leaves is not a very easy task, as it is extremely difficult to get the animals to eat great quantities of them.

As regards treatment, death generally takes place so rapidly once the animals started showing symptoms of gifblaar poisoning that none is likely to be of any use; we must therefore pay more attention to the prevention. The plant could be eradicated with arsenical preparations as advised by the Department of Agriculture. This method of eradication could, however, only be applied when gifblaar occurs in small patches, as the arsenical solution also destroys other veld vegetation. Large areas where gifblaar is prevalent could be fenced off in the form of camps, which could then be used for grazing purposes during the non-dangerous periods.

I take this opportunity of expressing my thanks to Sir Arnold Theiler, Dr. C. Neser, and Dr. H. H. Curson for verbal information about experiments they have carried out with gifblaar and for the valuable suggestions they have made.

LITERATURE.

(1) *Dunphy, J. T.* "Report of Experiments carried out to observe affects of certain Poisonous Plants on Sheep and Goats." *Transvaal Agricultural Journal*, 1906, p. 315.
(2) *Marloth, R.* "The Chemistry of South African Poisonous Plants and Plant Products," Presidential Address, Cape Chemical Society, Capetown, 30th May, 1913.
(3) *Stent, S. M.* *Agriculture and Small Holder of South Africa*, March, 1916.
(4) *Walsh, L. H.* "South African Poisonous Plants." T. Maskew Miller, Capetown, 1909.
(5) *Sundry.* Leaflet No. 1: "Plants Poisonous to Stock." Division of Botany, Transvaal Department of Agriculture, September, 1904.
(6) Bulletin of Imperial Institute, Vol. XIV, No. 1, 1916.
(7) Weekly Advice Service, Department of Agriculture, 9th August, 1926. "'Gif' or 'Gifblaar' and how to Eradicate it."