One sheep voluntarily ingested 2 kilograms of the ripe *white* kidney bean within thirty-six hours without any harmful effects.

Another sheep was drenched with 1,500 grams of the ground white kidney bean, without developing any symptoms of poisoning, and a third sheep with 300 grams of the fresh succulent roots, without any harmful effects.

The reaped bean land was then inspected, and on these areas roots, khaki bush, and grass were the only vegetation on the land. As *Dimorphotheca spectabilis* (bietou) frequently occurs on old lands in the Ermelo district, it is probable that the animals which had died had ingested this plant. The vegetation on this old land had been eaten and trodden down considerably, so that if *Dimorphotheca spectabilis* had occurred rarely, nothing would have been left of it.

LEGUMINOSAE (CAESALPINIEAE).

C. *Elephantorrhiza Burchelli*, Benth.

Synonyms.—Elandsboontjie, Looiersbossie.

Origin of Material Tested.—Dr. Phillips, Principal Botanist, Division of Plant Industry, Pretoria, forwarded a small quantity of these mature beans to the Onderstepoort Laboratories with the remark that they were suspected of having caused the death of a native.

Distribution.—*Eastern Cape Province*: Klipplaat, Cradock, Queenstown.
North Western Cape Province: Barkly West.
Orange Free State: Heilbron, Bethlehem, Bloemfontein.
Basutoland: Leribe.
Natal: Newcastle, Colenso, Mooi River, Tweedie.
Zululand: Eshowe, Mtunzini.
Swaziland.
Transvaal: Pretoria, Middelburg, Barberton, Potgietersrust, Waterberg, Potchefstroom, Christiana, Lichtenburg.
Southern Kalahari: Vryburg, Mafeking.
Southern Rhodesia: Salisbury, Lomagundi.

Stage and State of Plant.—Ripe beans.

Toxic Principle.—The beans contain a strong irritating principle. When chewed, these beans first have a sweetish taste, which, within a minute turns into a feeling of burning. The toxin is soluble in water, insoluble in 96 per cent. alcohol and in ether. On extracting the beans with ether a thick yellowish, tasteless non-toxic oil, which is present to an amount of 10 per cent., is obtained.

After clarifying the watery extract with basic lead-acetate, removing the lead with a saturated solution of sodium carbonate, and evaporating the filtrate, a thick light brown resinous substance resulted, which proved to be very toxic on subcutaneous injection into guinea-pigs.

Symptoms.—*Guinea-pigs*: 0.75 gram equivalent of the aqueous extract of the bean causes extensive necrosis of the subcutaneous tissues surrounding the point of injection, an acute catarrhal gastro enteritis and oedema of the lungs.

Rabbits: 10 grams of the bean kill rabbits within 24 hours, the most marked symptoms being apathy, inappetence and a profuse foetid diarrhoea.

Sheep: 250 grams of bean caused lassitude, inappetence, severe abdominal pain, accelerated pulse and respiration, a pronounced diarrhoea, and death within 24 hours after dosage.

Post-mortem Appearances.—The most pronounced lesions were an acute catarrhal gastro-enteritis with numerous haemorrhages in the mucosa and degeneration of the liver.

LILIACEAE.

Urginea sp. (Onderstepoort Plant Specimen No. 58822.) File No. 142/582.

Origin of Material Tested.—Magut, Zululand.

Occurrence.—On the farms Lelieshoek, Doornbos, Oasic, and Magut, in the Ngotshe District.

Stage and State of Plant.—Bulbs and dry leaves. Plant was in the post-flowering stage.

Symptoms.—The natives in Zululand use this plant as a cure for sweating sickness in calves. 500 grams of the bulbs caused marked dyspnoea, accelerated and weak pulse, inappetence and apathy in a sheep. This animal died eighteen hours after dosage with symptoms of asphyxia.

When brought on to the skin the juice causes a feeling of burning and itching, and reddening of the skin.

Post-mortem Appearances.—Pronounced general cyanosis; injection of the subcutaneous blood vessels, especially those of the front quarters; subepicardial and subendocardial haemorrhages; marked hyperaemia and slight oedema of the lungs; hyperaemia of the liver; haemonchosis and oesophagostomiasis; haemorrhagic swelling of the retropharyngeal, submaxillary, bronchial and mediastinal lymph glands; haemorragic nodules in the spleen; in the rectum the faeces pellets were mixed with coagulated blood; and a slight acute catarrhal gastro-enteritis, especially marked in the caecum.

Microscopical Pathological Anatomical Diagnosis.—Hyperaemia of the liver, lymph glands and lungs. In the lungs there was evidence of the earliest stage of a broncho-pneumonia.

LOGANIACEAE.

Strychnos Henningsii, Gilg.

Synonyms.—Hardepeer, Hard Pear.

Origin of Material Tested.—Pinie Forest, Kingwilliamstown, and Kambe Forest, Transkei.

Distribution.—In the forests of Zululand, Natal and Eastern Cape as far south as East London.

Stage and State of Plant.—Dried bark of the tree collected in middle of August, 1928.

Toxicity.—The M.L.D. of the bark collected in August, 1928, per kilogram rabbit is approximately 20 grams. An interesting fact, however, is that 50 grams of the bark of *Strychnos Henningsii* collected in December in the Kambe Forest, Transkei, had no effect whatsoever on rabbits. This difference in the toxicity of the bark of one and the same species of tree may be due to the fact that some plants are most toxic in spring (i.e. the pre-flowering stage, e.g. *Geigeria aspera*); another explanation is that one and the same species of plant growing in different localities often differ in toxicity. It was definitely proved that the toxicity of Syringa berries collected from one and the same tree in Onderstepoort varied considerably in different years.

Symptoms.—Rabbits dosed with 50 grams of the bark died within quarter of an hour after dosage with symptoms closely resembling those of strychnine poisoning. The heart continued beating for about three minutes after the respiration had stopped, hence death most probably was caused through tonic spasm of the diaphragm.

Post-mortem Appearances.—General cyanosis, otherwise completely negative.

MELIACEAE.

Melia azedarach, L.

Synonyms.—Bessieboom, Sering, Syringa, bead tree, Indian lilac, Pride of India, Chinese umbrella tree, China berry.

Origin of Material Tested.—Onderstepoort, Natal and Orange Free State.

Distribution.—It is a native of the Himalayan region, but is widely cultivated as an ornamental tree.

Stage and State of Plant.—All stages and states were tested.

Toxicity.—The fact that no evidence of experimental proof of the toxicity of the Syringa berry could be found in the literature at my disposal as well as the conflicting recorded information prompted the author to conduct experiments in this respect.

The most toxic part of the tree was the ripe drupe. The flowers, the green drupes and the bark proved to be much less toxic than the ripe drupes, whereas it was impossible to kill rabbits with the green leaves.

Seat of the Toxin in the Drupe.—The only part of the drupe which contains the toxin is the soft yellowish epicarp, the exocarp and endocarp being completely harmless.

Susceptibility of Animals to the Toxin.—Experiments were conducted with guinea-pigs, rabbits, fowls, muscovy ducks, dogs, sheep, goats and pigs. Donkeys can often be seen eating the ripe drupes without any harmful effects, but I have no doubt that large quantities of the drupes will cause symptoms of poisoning in these animals, although it is maintained that they are completely insusceptible to the toxin, 150 to 200 grams of the ripe drupes being the M.L.D. for a 75 kilogram pig, and fowls to be least susceptible.

Symptoms.—A detailed description of the experiments carried out at Onderstepoort is being published in the next volume of the Transactions of the Royal Society of South Africa.

Fowls and Muscovy Ducks.—90 grams given in the course of two days caused pronounced apathy, inappetence, white diarrhoea and general cyanosis. Four days after dosage the birds were completely paralysed, lying on their side unable to move. Death occurred from three to five days after dosage.

Post-mortem Appearances.—Compaction of the crop; marked acute catarrhal enteritis, and hyperaemia of the lungs.

Dogs.—It was impossible to kill these animals with the drupes as they vomited immediately after dosage.

Sheep.—800 grams of the ripe drupes caused symptoms to develop within three hours after dosage. Apathy, nervousness, sluggish movements, general cyanosis, weak and accelerated heart action and laboured respiration were the most outstanding symptoms. Death occurred within fourteen hours after dosage.

Post-mortem Appearances.—Pronounced general cyanosis; blood, even six hours after death, was only partly coagulated and intense dark red in colour; marked hyperaemia of the subcutaneous blood vessels; degeneration of the liver; tumor splenis; marked general swelling of the lymph glands; a pronounced acute catarrhal abomasitis and enteritis, chiefly affecting the small intestine.

Goats.—A goat drenched with 800 grams of drupes showed symptoms of poisoning, which passed off completely after three days. 1,000 grams caused symptoms similar to those in sheep within half an hour. In addition this animal showed nervous symptoms. Twenty-four hours after drenching the goat was unable to rise and when picked up it was unable to stand, being most weak in the hindquarters. Fibrillary contractions of the muscles of the hindquarters were very distinct. As there was no improvement in the condition of the animal in the course of the following seven days, it was killed for post-mortem purposes.

Post-mortem Appearances.—Dilation of the right ventricle; fibrinous pleuritis; broncho-pneumonia; oedema of the mucous membrane of the abomasum; trichuris ovis in large intestine; stilesia hepatica and cirrhosis of the liver; and an abnormal amount of fluid in the spinal canal.

Pigs.—200 grams of drupes caused the following symptoms in a 75 kilogram pig within half an hour: restlessness, retching movements, later on actual vomition occurred, pronounced dyspnoea, the animal frequently gasping for breath, general cyanosis, pulse weak and accelerated, and the respiration laboured and irregular. Two and a half hours after dosage the animal died in a state of convulsions and suffocation. Before death the pigs were markedly bloated.

Post-mortem Appearances.—Marked general cyanosis; stomach distended with gas and showing hyperaemia of the mucosa; congestion of the liver.

Microscopical Pathological Anatomical Diagnosis.—Fatty degeneration and hyperaemia of the liver and kidneys. The goat showed a degeneration of the sciatic nerve; and in one sheep there was a lymphadenitis purulenta diffusa acuta.

Toxic Principle.—This was investigated by Professor Rindl, of the Grey University College, Bloemfontein. His conclusion is " the toxins are not of the nature of alkaloids, proteins or glucosides easily hydrolysed by acids. They most probably belong to that indefinite group known as ' bitter principles.' "

MELIANTHACEAE.

Melianthus comosus, Vahl.

Synonyms.—Kruidjie-roer-my-niet; Touch-me-not.

Origin of Material Tested.—"Brewershoek," Britstown, Cape.

Distribution.—*South Central Cape*: The Karroo below Bokkeveld, Gamtoos and Gouritz Rivers; George, Phillipstown, Graaff-Reinet, Riversdale, Worcester, Naauwpoort, Middelburg, Willowmore, Uniondale, Oudtshoorn, Queenstown.

Orange Free State: Fauresmith, Bethlehem, Ladybrand, Zastron.

Transvaal: Wakkerstroom, Volksrust.

Natal: Newcastle, Ingogo.

Southern Kalahari: Prieska, Upington, extending to South West Africa.

Stage and State of Plant.—In the flowering and young fruiting stage. Only 100 grams of the material, which was in a completely dry state, were forwarded.

Symptoms.—80 grams of leaves, young branches, flowers and young fruit dosed to a sheep caused symptoms to develop within three hours, of which the most outstanding were dyspnoea, general cyanosis, bloating and an accelerated and weak pulse. The animal died within four and a half hours after dosage.

Post-mortem Appearances.—Marked general cyanosis; slight hydropericardium; very marked hyperaemia and slight oedema of the lungs; subepicardial haemorrhages; hyperaemia of the liver and kidneys; injection of the subcutaneous blood vessels; rumen markedly distended with gas; and a pronounced acute haemorrhagic duodenitis and jejunitis.

ZYGOPHYLLACEAE.

Zygophyllum microcarpum, Licht.

Synonyms.—Ou-ooi-bos (old ewe bush), Armoedsbossie, Sandrepuis.

Origin of Material Tested.—"Bleskrantz," Williston, Cape.

Distribution.—The Western Union from the Olifant River to South West Africa, and inland from Beaufort West, Matjesfontein, Williston, Carnarvon, Fraserburg, Laingsburg, Kuruman and Gordonia. This plant is thus seen to be limited to the drier western parts of South Africa and prefers to grow along rivers and on sandy soil.

Stage and State of Plant.—A small quantity of the dry flowering plant was forwarded.

Symptoms.—Several reports have been received from farmers in the above districts to the effect that this bush is poisonous to stock. They reported that death often occurred very suddenly after acute bloating.

Rabbits.—A rabbit dosed with 25 grams of this plant at 11.30 a.m., 9.8.28, showed the following symptoms:—

10.8.28. 6.30 a.m. Animal found in a semi-paralytic state with torticollis. When the abnormal position of the head was corrected it immediately took up the torticollis position again. There was a continual quivering of the tail. The respiration was accelerated, deep and more of an abdominal type. The animal made repeated attempts to rise, but without success, the paralysis being more pronounced in the neck and front quarters. The cornea reflex was very weak and the pulse extremely accelerated and weak. The sensitivity which was tested by means of pinpricks, was normal in the hindquarters, reduced in the front quarters and completely absent in the neck and head.

11.8.28. Condition was the same.

12.8.28. Condition was the same.

13.8.28. Condition was worse. No cornea reflex, convulsions over the whole body at irregular intervals. Completely paralysed, pulse slow and very weak. Dark greenish diarrhoea.

14.8.28. 6 a.m. Animal found dead in cage with torticollis still present.

Post-mortem Appearances.—Subendocardial haemorrhages in left ventricle; marked hyperaemia of the lungs; pronounced degeneration of the liver and kidneys; an acute catarrhal gastro-enteritis with haemorrhagic patches in the mucosa; the small intestine was filled with a greenish intensely mucous substance.

Microscopical Pathological Anatomical Diagnosis—

(*a*) *Kidneys*: Extensive fatty changes in the cortex;

(*b*) *Liver*: Hyperaemia and very extensive fatty changes, especially in the vicinity of the central vein;

(*c*) *Myocard*: Very extensive fatty changes so that practically no normal fibres were seen.

Sheep.—A sheep was dosed with 200 grams at 9 a.m., 10.10.28; 200 grams at 12 noon, 10.10.28; 200 grams at 3 p.m., 10.10.28; and developed the following symptoms:—

11.10.28. 7 a.m. Animal lying down and resting head on the ground. On touching, the animal jumped into the air and fell down heavily, unable to support the body on the legs. It rose with great difficulty, repeated the jumping twice, and then walked with a swaying gait and pronounced ataxy, keeping the head very low and the eyes half closed. The heartbeat was very irregular, feeble and accelerated, and the respiration more of an abdominal type and accelerated. When standing a quivering of the muscles of the front quarters could be felt.

12.10.28. Condition was the same.

13.10.28. Diarrhoea, rapid loss in condition, inappetence, no rumination; pronounced weakness and listlessness; pulse very irregular, accelerated and hardly perceptible; anaemia, respiration irregular and accelerated; loss of control over the voluntary muscles; and nervous symptoms.

14.10.28. Unable to rise.

The condition became worse in the course of time, until the animal died during the night of the 21-22 October, 1928.

Post-mortem Appearances.—Anaemia, emaciation; marked hyperaemia of all the parenchymatous organs; degeneration of the liver; impaction of the rumen and reticulum; haemorrhagic abomasitis and enteritis; and a haemorrhagic swelling of the bronchial, mediastinal and retropharyngeal lymph glands.

Microscopical Pathological Anatomical Diagnosis.—Fatty degeneration of the liver, kidneys and myocard.

Paper No. 29.

THE TENACITY OF THE VIRUS OF FOOT-AND-MOUTH DISEASE UNDER FIELD CONDITIONS.

By Sir Ralph Jackson, England.

During recent years the tenacity of the virus of foot-and-mouth disease under widely different conditions has been tested in a number of European laboratories, and recent veterinary literature contains abundant information as to the viability of the virus under conditions which at times faithfully represent natural conditions or conditions met with in practice, whilst in others they merely approximate to natural conditions. The history of foot-and-mouth disease in Great Britain during the past 20 years presents a unique opportunity for examining the question of the tenacity of virus under field or natural conditions, inasmuch as the stamping out policy has been in force during that period as a general policy which has been departed from in a limited number of cases only. The observations are not therefore liable to be confounded by the presence of recovered animals in the herds and recurrence of disease is not to be associated with the presence of possible " carriers " of infection, nor generally speaking, is the risk of infection from other centres of disease comparable with that which would arise in countries where the disease is dealt with by isolation.

Before proceeding to the examination of the cases in which the survival of the virus was demonstrated by the reappearance of disease among animals after an interval, it will be necessary to describe briefly the measures taken on the diagnosis of disease.

On declaration of the disease the affected animals and those in contact are valued and within 12 to 24 hours are slaughtered and their carcases incinerated. From the earliest moment buildings, yards, pig-sties, etc., are sprayed continuously with a disinfectant solution, and after the carcases have been disposed of a thorough cleansing and disinfection of the infected buildings is carried out, a solution of a coal-tar preparation being used.

Heavily contaminated food materials, litter and sacks are burnt.

The clothing of attendants is fumigated, and vehicles, milk churns, threshing machines, etc., disinfected by suitable processes.

Manure is closely packed into a heap 4 to 6 feet high and fermentation is relied on to destroy the virus except on the surface, which is sprayed with disinfectant solution.