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no 2 november 1974

a publication by the faculty of veterinary science
'n publikasie van die fakulteit veeartsenykunde

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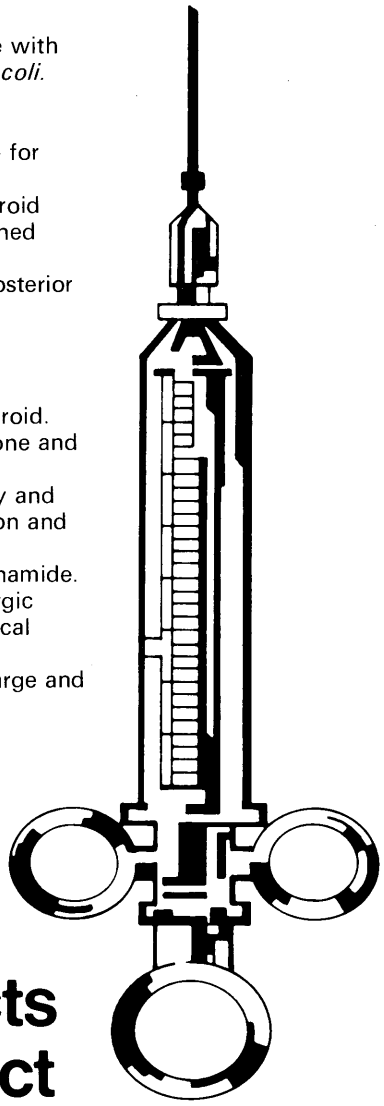

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No 2 NOVEMBER 1974

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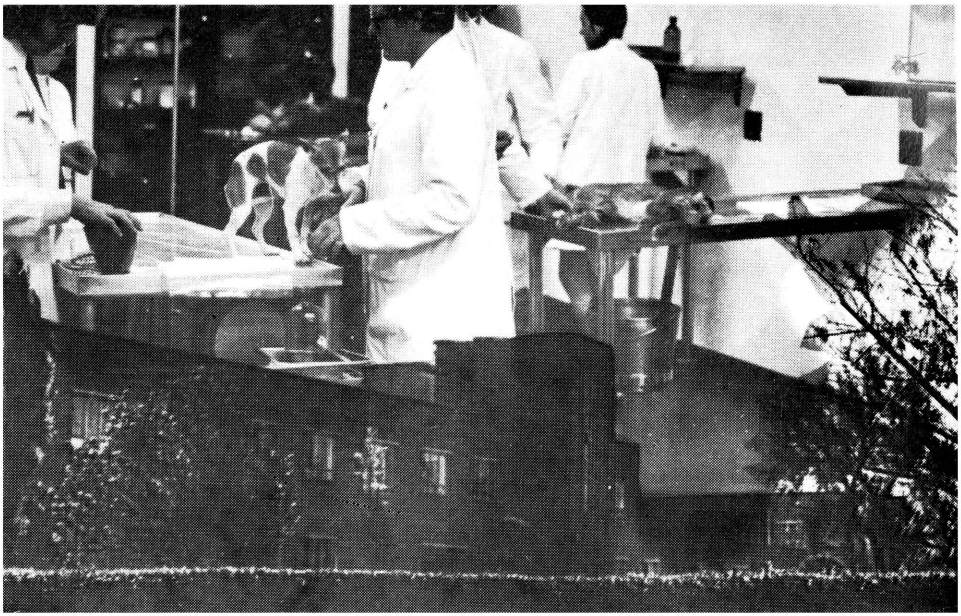
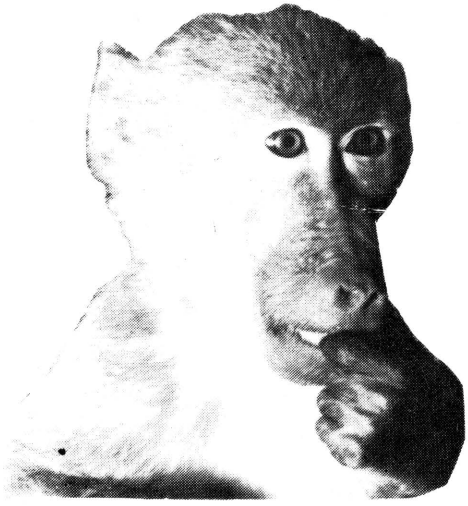
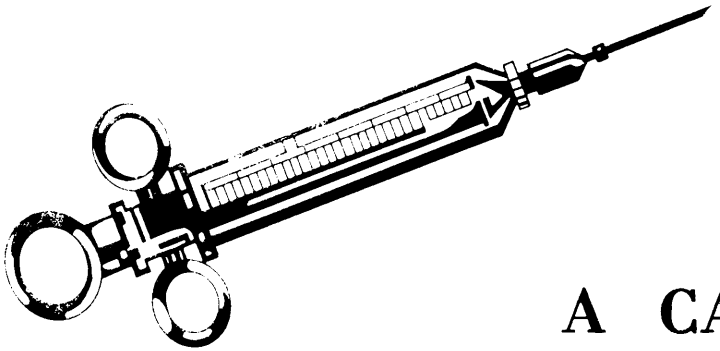


EDITORIAL



The response to our first issue was very favourable – we are indebted to those veterinarians who have given us the financial assistance so essential for the continuation of this venture. Many donations have also been received – thanks. Subscription forms have been included with this issue as well – please ignore them if you have already subscribed.



A CASE OF CANINE AUTO-IMMUNE HAEMOLYTIC ANAEMIA

Class Case – J. Reid-Rowland (B.V.Sc.V.)

SUBJECT:

A one year old, male French Poodle was presented for examination at the Onderstepoort clinic.

ANAMNESIS:

The patient was admitted with the following report: "Dog has been off colour for the last few days. Will not eat at all. Urine very turbid and dark brown. Epistaxis for last day (slight)".

The illness had lasted for the last week, and the dog was not eating. It was drinking water. There was no vomition, the faeces were yellowish, and the urine cloudy. There was some pain on palpation of the kidney area.

CLINICAL EXAMINATION:

Habitus: Somewhat depressed, although still alert. The general condition of the patient was rather poor. The mucous membranes were pink and moist. There was a crusty discharge around the eyes; the ears were slightly dirty.

Buccal cavity: There was a halitosis, and the teeth were crusted with tartar. The tonsils were slightly red but not swollen.

Abdominal cavity: Palpation revealed no abnormalities.

Respiratory system: The alveolar and lung sounds were normal. There was a crusted discharge from the nose, which had been bleeding.

Circulatory system: The heart sounds were normal, but the rate was somewhat irregular; the pulse was strong. The superficial lymphnodes were not enlarged.

Digestive system: The anus was sensitive to the thermometer, but there were no palpable abnormalities.

Urogenital system: Urine dark brown and cloudy.

There was no abnormalities in the nervous system.

SPECIAL EXAMINATION:

1. **Urine analysis:** Coffee coloured, deposits (blood casts?) seen on centrifugation; S.G. 1,035; pH 6; protein 100 mg%; glucose and ketones negative; bilirubin moderate; RBC casts seen on centrifugation and Sternheimer-Malba staining; bladder epithelium cells were also seen.

2. **Faeces:** bout 4 gm were collected; the faeces were dark and hard. There was no blood or mucus and a few Coccidia oocysts were seen.

3. **Haematology:** See table. The most striking finding was a neutropaenia and thrombocytopaenia; the latter improved considerably with treatment. A plate agglutination test was carried out, and clumping of whole fresh blood was demonstrated within 60 seconds.

4. **Blood Chemistry:** A BUN determination was done shortly after admitting the patient; but BUN level was calculated to be 12,9 mg%. This reduced the possibility of nephritis, which was the disease the patient was suspected of having on admission. The SGOT and SGPT levels were found to be elevated. SAP value was 289, and TPP 7,6 mg%. See table.

5. **Electrophoresis:** A serum sample was taken. See sample.

PROVISIONAL DIAGNOSIS:

On the basis of the following findings:

- clumping of red blood cells
- marked fever, subsiding after two days
- thrombocytopaenia
- urine analysis as described
- halitosis, epistaxis, anaemia
- faeces dark, hard, with a tint of yellow

a diagnosis of CANINE AUTO-IMMUNE HAEMOLYTIC ANAEMIA was made.

PROPOSED COURSE OF THERAPY:

A course of cortico-steroids and antibiotics was proposed, with the following rationale:

- immuno-suppression to reduce the effects of the auto-immune disease;
- antibiotic cover, to reduce the chance of any secondary infection, and to cover the possibility of **Ehrlichia canis** infection. This was an important differential diagnosis, and, although blood-smears had so far proved negative, this could not be ruled out.

This course of treatment was not carried out thoroughly, the main reason being in order to follow the course of the disease's clinical pathology without interference from the cortico-steroids. A course of antibiotics was given.

THERAPY AND RESPONSE:

1. The patient was treated on admission with 1,5 ml of **"Liquamycin"** given intravenously. This was to have been followed for ten days, but was interrupted on the 9th day (22.5.74) as a result of an iatrogenic phlebitis. The **"Liquamycin"** was then substituted with **oxytetracycline** capsules (250mg.)

2. **Betamethasone** was used as an immuno-suppressive, being given on the 17th, 18th, 21st, and 22nd of May. It was then discontinued in order to watch the natural progress of the patient. gress of the patient.

3. **Distemper** vaccination at the owner's request. Irrelevant to the discussion, but one wonders how the build-up of immunity was affected by the cortico-steriods!

4. **Protiplex:** 30 ml was given subcutaneously, in order to make good the losses caused by anorexia. As the dog rapidly regained his appetite, this was discontinued.

5. **Litrisan:** A course of Litrisan was given, at the rate of one tablet per day, in order to act as a liver stimulant; the liver showed signs of possible damage.

6. **Chloramphenicol:** Eye-ointment was given to reduce the (apparent) eye-infection.

7. **Winstrol:** A course of Winstrol tablets (anabolic steroids) was started shortly before the patient was discharged. As it was, he was discharged before more than one day's treatment could be given.

The response of the patient was, by and large, good.

- reduction in fever from 41^{o} to 38^{o}C in the first two days. This seems to be normal for auto-immune anaemia.
- colour of the urine cleared dramatically from the 18th May; from dark, cloudy fluid, it became clear, light yellow.
- white cell count: Went up from 4 400 on admission to 11 200 on the 21.5, but dropped again on the 27.5 to 7 400
- thrombocytes: rose steadily from 25 700 on 17.5 to 129 024 on 27.5.
- red cell count: – rose slightly, then dropped again shortly before discharge.
- liver function tests – SGOT and SGPT levels dropped between 17.5 and 22.5.
- habitus:- improved enormously after the first three days, and remained good, except for some pain and depression as a result of the phlebitis.
- slide agglutination test:was rated as+++ on admission, dropped to more or less negative by 21.5
- weight of the patient, despite improved appetite, remained more or less constant,

DIFFERENTIAL DIAGNOSES:

1. CANINE AUTO-IMMUNE HAEMOLYTIC ANAEMIA (CAHA):

The symptoms and clinical pathology of this disease as described in the literature are very similar to what was seen in this case. The main points of difference were:

- CAHA has a leucocytosis; this case had a leucopaenia.
- CAHA has marked changes in erythrocyte morphology; none were seen here.
- CAHA has an absolute neutrophilia; this case only showed a relative neutrophilia

2. Canine systemic lupus erythematosis:

"Canine lupus erythematosis is a complex, multisystem disorder characterised by the simultaneous or suquential development of four distinct clinical syndromes: autoimmune haemolytic anaemia, idiopathic thrombocytopaenic purpura, glomerulonephritis, and symmetrical (rheumatiod) polyarthritis. Accompanying or preceding these are a variety of auto-antibodies". (Kirk, Current Veterinary Therapy). Of these four, only the first and second were seen; there was no evidence of nephritis or joint-pains. Another diagnostic feature of this syndrome is the existence of "S.E. cells"; despite searching a number of blood and white-cell smears, none of these were seen.

3. Ehrilichiosis

This has several points in common with the case:
– epistaxis, fever, anaemia. The anaemia may be immunologically mediated.
– reaction to a course of **oxytetracycline** therapy.

However, it does not cause auto-agglutination of red blood cells, nor is there classically, a sudden drop in fever after the first 48 hours. There is no thromocytopaenia. Intracellular parasites were never seen, despite looking through a series of blood-smears (Giemsa stained)

4. Nephritis:

The only symptom indicative of nephritis was the urinary disturbance seen early in the illness. This differential diagnosis was therefore eliminated.

FINAL DIAGNOSIS:

As matters stand, the diagnosis seems to be Canine Autoimmune Haemolytic Anaemia; however, this requires confirmation of two points:

– Coomb's test, which was not available.
– recurrence of the disease; this is reported to be in the order of 80–85%, so the patient will very likely be returning to Onderstepoort, if this diagnosis is correct.

PROGNOSIS:

The disease will probably recur; Kirk quotes the rate of recurrence as 80–85%. If not, the prognosis is probably good.

TABLE OF CLINICAL PATHOLOGY RESULTS:

1. Haematology

					Normal
Date	15.5.74	17.5	21.5	27.5	
Hb	9,8	13,2			15g%
RCC	3,24	4,75	4,51	3,80	6,8 x 10^6 /mm^3
Ht	25,9	37,7		30,4	45%
MCV	79	80			70
WCC	4400	9000	11200	7400	11,500
Thrombo-cytes		25,700	61530	129024	3 - 7 x 10^5 / mm^3
Neutrophil: Lymphocyte			65:35		

2. Urine

16th May: Obtained free; coffee colour 1,035 SG; pH6;
Protein 100 mg% Bilirubin moderate; Erythroyte casts:
Epithelial cells present;

3. Blood Chemistry

	15.5.74	17.5	Normal
BUN	12,9		12 – 27 mg%
SGOT	69	19	16 – 26 I.v.
SGPT	45	40	25 – 26 I.v
TPP		7,6	± 7 mg%

4. Electrophoresis

17th May:	Albumin	3,18
	alpha - 1	0,24
	alpha - 2	0,17
	alpha - 3	0,17
	beta - 1	0,24
	beta - 2	0,57
	beta - 3	0,49
	gamma - 1	2,53
	A/G ratio	0,72

These figures are all within the normal ranges for albumins and globulins.

Discussion

The haemoglobin and RCC were both low at first, but picked up; this is normal for CAHA. However, the low levels of the white cells counts casts some doubt on the diagnosis of CAHA – classically, there is an elevated WCC, with an absolute neutrophilia.

The thrombocyte picture is typical for CAHA, and showed a remarkable improvement as the animal recovered.

The results of the urinalysis are fairly typical of CAHA.

The blood chemistry is indicative of some liver malfunction; the cause of this may have been secondary to the main disease, or have occurred independently. The matter was not followed up, due to the marked general improvement in the patient's condition. The BUN was within normal ranges, although a rise can be expected early in the course of the illness.

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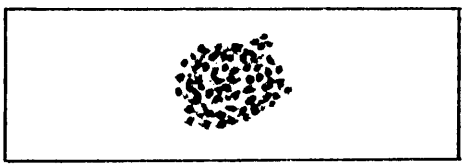
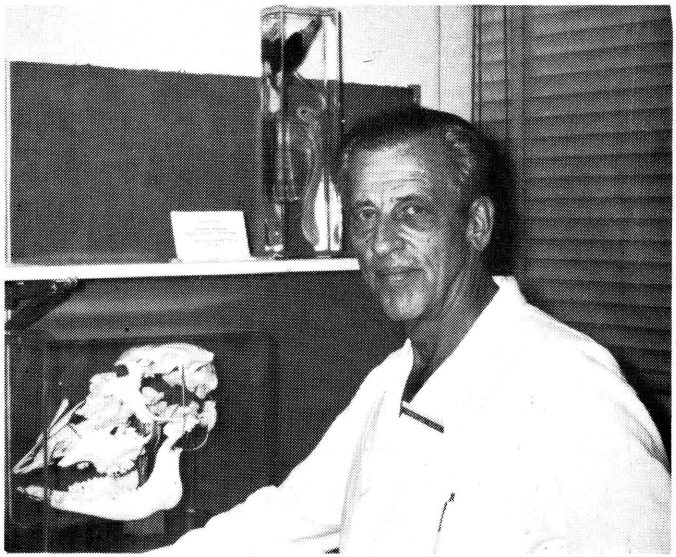


A drop of blood on a glass slide showing CAHA agglutination.

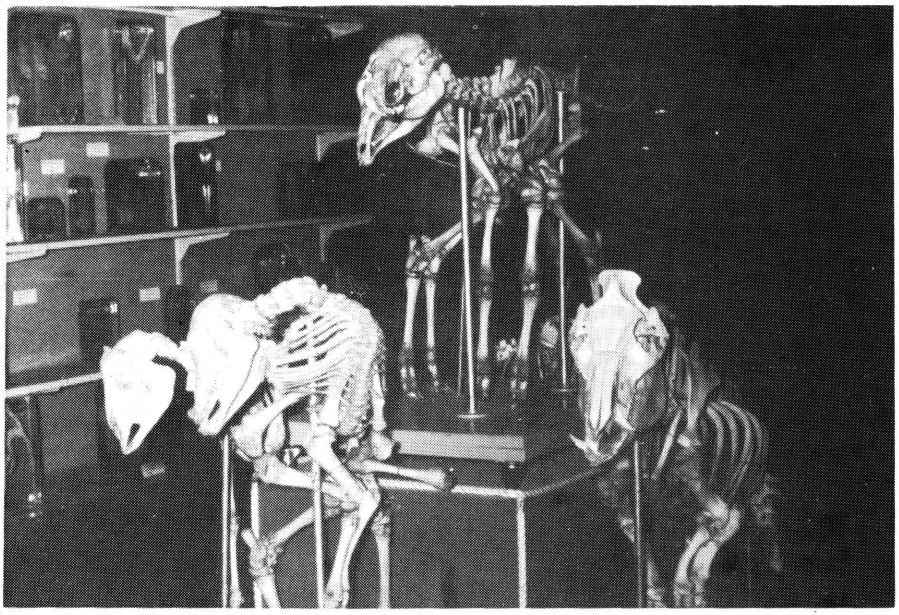
AU REVOIR BOOMPIE



Any academician who has been actively involved at Onderstepoort for some thirtyeight years is bound to leave in his place the historical fragments that constitute the legendary figure. Such a man is Professor H.P.A. de Boom, who in 1936 started his lecturing career, to become Professor of Anatomy in 1956, the position he held until 1974, the year of his retirement.

Prof. de Boom was by uitstek leermeester. Hy het die kuns verstaan om moeilike begrippe op 'n eenvoudige en verstaanbare manier aan studente oor te dra. Met 'n paar lappe byderhand kon hy wondere verrig as dit handel oor die anatomie van die lieskanaal of die rangskikking van die omenta. Die fundamentele begrip van vorm en funksie met die ontwikkelingsgeskiedenis as uitgangspunt was hom so erns, dat hy 'n spesiale inleidende kursus beplan en ingestel het om studente in die anatomie te oriënteer. Met die klem steeds op die basiese eenvormige boupatroon van die soogdier, het sy studente nooit probleme ondervind om die vergelykende anatomie van die huisdiere onder die knie te kry nie. Die anatomiekursus soos dit huidig aangebied word, sal altyd hierdie stempel dra. Met eindelose geduld en 'n goeie sin vir humor kon hy besprekings of demonstrasies aan groepe studente herhaal en by die laaste groep met meer entoesiasme eindig as waarmee hy begin het -- tot verstomming en frustrasie van sy mededosente. En sy mondelinge toetse het dikwels in 'n kwasi lesing "ontaard" waarby studente meer kennis ingeneem as weergegee het. Met 'n geweldige algemene kennis van veeartsenykunde buite sy vakgebied, kon hy onderhoudend met studente oor die uiteenlopendste probleme gesels, was hy hulle steeds simpatiek gesind en het hy aktief in hulle belanggestel.

A student who approached him with a question pertaining to the hoof of the horse would suddenly find himself on a Darwinian adventure, being guided through the period of the Eo -- to the Mesohippus. Or perhaps on questioning the actual *function* of the diverticulum of the Equine nostril would be plunged into the physico-dynamics of air turbulence that is functional in clearing the nares of dust particles! Once in the central nervous system, Boompie was really in his element. Caught up with the gesticulations that are so characteristic of his teaching methodology the student could almost see himself hurtling along a spino-cerebellar tract, leaping across synaptic clefts, winding from ipse -- to contralateral to collide with a nucleus dentatus or some such mysterious entity.



Prof. de Boom's speciality — the abnormal form.

Another of Prof. de Boom's most admirable attributes is his ability to take an active interest in student affairs while at the same time maintaining the respect that his position deserves, and his visits to the Hostel and subsequent discussions into the early hours of the morning (usually while Mrs de Boom was away!) were always much enjoyed.

Prof. de Boom was hoofsaaklik verantwoordelik vir die kontak wat daar op akademiese vlak bestaan tussen die universiteite van Lourenco Marques en Pretoria. Gedurende 1969 was hy ses maande daar op uitnodiging en in 1970 het hy sy studieverlof daar deurgebring. As gevolg daarvan het studente van Mosambiek vir nagraadse opleiding by die fakulteit van veeartsenykunde ingeskryf, een waarvan in sy eie departement gepromoveer het. 'n Kollega van die Universiteit van Lourenco Marques dien ook as eksterne eksaminator vir die anatomie-eksamen in ons fakulteit.

Prof. de Boom gaan hom na sy aftrede nog lank in sy departement besig hou. Maar eers gaan hy vir 'n jaar na die universiteit van Cornell waar hy die kursus in toegepaste anatomie sal waarneem. Hy wil die geleentheid daar te baat neem om aanvoorwerk te doen vir die skryf van 'n teksboek oor veeartsenykundige embriologie.

And thus we can but wish Prof. de Boom success with his future plans and pay tribute to a true intellectual.

VERSLAG OOR KORT NA GRAADSE OPKNAP KURSUS IN VLEISHIGIËNE

Op versoek van die Hoof Vleishigiëne-beampte van die dept. Landbou-Tegniese Dienste het die Fakulteit Veeartsenykunde vanaf 24—28 Junie 'n kort opknapkursus vir gegradueerde veeartse in die vakgebied VLEISHIGIËNE aangebied.

Die kursus het bestaan uit 'n reeks lesings toegelig met films en kleurskyfies, wat aangebied is deur verskeie deskundiges in diens van die Fakulteit self, die WNNR, die S A Buro van Standaarde, die Abattoirkommissie, die Vleisraad, die Afdeling Veeartsenydiens van die Dept Landbou- Tegniese Dienste en die Dept Veeartsenydienste van die Rhodesiese Ministerie van Landbou.

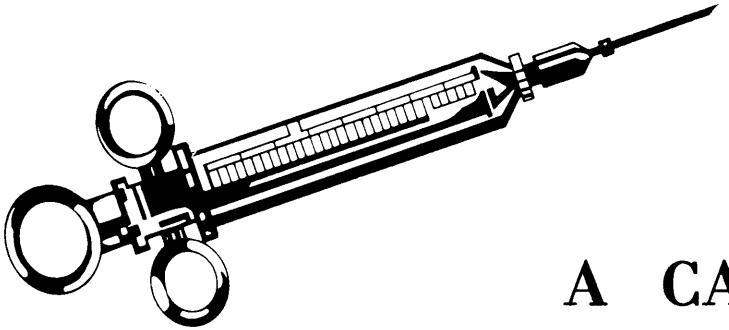
Die kursus het die volle spektrum van vleishigiëne gedek d.w.s. soos dit beheer word kragtens die Wet op Higiëne by Diereslagting, Vleis en Dierlike Produkte No 87/1967, en is aangebied teen die agtergrond van die huidige en toekomstige vleisbedryf in die Republiek en in-agnemende die internasionale standaard van Vleishigiëne waarna gemik word. Die kursus is bygewoon deur 'n 40 stuk staatsmunisipale - en privaat praktiserende veeartse uit alle dele van die Republiek, Suidwes-Afrika en Rhodesië.

In die loop van die week het die kursusgangers besoek afgelê by die nuwe abattoir te Balfour en 'n groot vleisprodukte organisasie te Krugersdorp. Die groep is ook verder sosiaal onthaal deur die firma Karoo Vleis Bpk en deur die Vleisraad, lg. by 'n braaivleis by "Huis en Haard"

Een van die referente by die kursus was dr H. Thornton van Rhodesië, die wêreldbekende outeur van 'n handboek oor vleishigiëne wat nou vir die sesde uitgawe verskyn. Die kursusgangers is ook toegespreek deur die Hoofbestuurder van die Vleisraad, Mnr G. Marais; die Voorsitter van die Abattoirkommissie, Mnr M. Jamneck; die agerende Hoofvleishigiënebeampte, Dr J.H.B. Viljoen en Mnr F.C.•Lategan van Karoo Vleis Bpk.

Die sukses van die kursus is te wyte aan die aktiewe bydraes en deelname van die referende sowel as kursusgangers aan die lewendige besprekings wat 'n kenmerk van die verrigtinge was en die sosiale verkeer het die hele aanbieding pragtig afgerond.

Die kursus het gestaan onder leiding van professor L.W. van den Heever, bygestaan deur dr G.V.S. Turner (senior lektor), beide van die afdeling Veterinêre Voedselhigiëne en Volksgesondheid van die departement Patologie.



A CASE OF CHRONIC PANCREATITIS IN A DOG

Class case — C. Waghorn B.V.Sc.V.

SUBJECT: A seven year old Dachshund weighing 5kg.

ANAMNESIS

The dog was presented at Onderstepoort clinic on the 5th of June 1974. The owner complained that the dog had lost weight steadily over the past four months despite the fact that it had a tremendous appetite. The owner also complained that the faeces had a very foetid smell and the dog suffered from flatulence.

CLINICAL SYMPTOMS

On presentation the following were noted;

1. The dog was rather nervous.
2. The skin was dry and slightly rancid smelling.
3. The abdominal muscles were very tense.
4. The faeces were soft, greyish — yellow in colour and had a very foetid odour.
5. The dog was very thin, and showed signs of dehydration.
6. A blood smear was negative for **Babesia** and **Erlichia canis.**
7. Urinalysis showed a pH of 7.5 and an SG of 1,016.

Provisional diagnosis

At this stage the following were considered;

1. Chronic pancreatitis
2. Hepatic degeneration
3. Diabetes mellitus
4. Helminth infestation

SPECIAL EXAMINATIONS

The following tests were carried out on the next day and the results of these tests are shown;

1. **Haematology**

Test	Result	Normal
Hb.	15,2	13-16 g%
RCC.	6,59	$6,1 \pm 1 \times 10^6/mm^3$
Ht.	54,0	40-50 %
MCV.	79	60-75 x 10
WCC.	12100	$6\text{-}11 \times 10^3 / mm^3$

2. **Blood chemistry:**

Test	Result	Normal
BUN.	13,6 mg.%	12-20 mg.%
B.Sugar	120,0 mg.%	80-120 mg.%
B.Sugar (fasting)	102,0 mg.%	
SGOT.	59 I.U.	16-26 I.U.
SGPT.	59 I.U.	25-30 I.U.
SAP.	185 I.U.	40-90 I.U.
Gamma-GT	6	3-13
TSP.	5,1 mg.%	$\pm$ 6 mg.%

3. **Urinalysis:** The pH was 6,5 and the SG was 1,012.

4. **Faeces flotation test:** The result was negative for eggs

5. **Faecal enzyme, fat, starch and muscle tests:**
   Fat – smear stained with Sudan ORO – fat globules seen
   Starch – smear stained with Lugols iodine – starch granules present.
   Muscle – smear stained with Lugols iodine – undigested muscle fibres were seen.
   Trypsin – faecal solutions were placed on X-ray-paper and after two hours no clearance of gelatin was noted.
   The above faecal tests (5) were repeated on the following dates and on every occasion the results were as recorded above; June the 7th, 10th, 11th, and 19th.

TREATMENT

250 ml. **Plasmolyte B** i.v. to rectify the dehydration. Glucose containing fluids were not considered due to a provisional diagnosis of diabetes mellitus.

12 ml. **Protiplex** i.v. for nutrition. Once the diagnosis of chronic pancreatitis had been made 1 capsule **"Cotazyme"** was given morning and evening. 1 Capsule contains enough enzymes to digest 100 gm of Pronutro; this was regarded as sufficient for the dog.

The period of treatment lasted for 16 days.

RESPONSE TO TREATMENT

At the time of discharge the following were noted:

1. The dog had regained its weight loss.
2. There was no dehydration.
3. The skin did not have the rancid odour.
4. The faeces were firmer and darker in colour. The foetid smell was not so pronounced.
5. The dog urinated and defaecated normally.

Final diagnosis Chronic pancreatitis

DIFFERENTIAL DIAGNOSES

The reasons why the other possibilities mentioned in the provisional diagnosis were discarded are given below. The various possibilities are discussed separately.

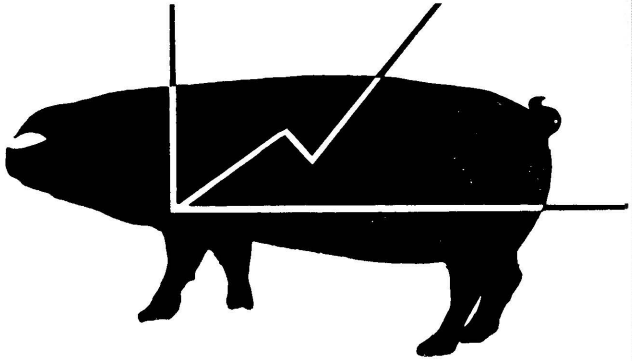
1. **Hepatic degeneration:** In the dog the most reliable indicator of hepatic degeneration is the SGPT level. The blood chemistry revealed that the rise in the level of SGPT was only 30 units above the normal and as this may increase several hundred units above normal, the increase in this case was not considered significant. The same opinion was held about the other results which in any case tend to be more significant in other forms of pathology. As a result no liver therapy was instituted. The tests were not repeated as a positive diagnosis of pancreatitis had been made.
2. **Diabetes mellitus:** Several urine analyses revealed no glycosuria at all. There was no polyuria at any time although there was slight polydypsia for a short while probably as a result of the severe dehydration, the cause of which was unknown. The blood sugar level did not exceed the normal values and the level decreased with fasting. All these factors indicated that there was no diabetes.
3. **Helminth infestation:** Three examinations for helminth eggs in the faeces failed to reveal any eggs at all so this possibility was also discarded.

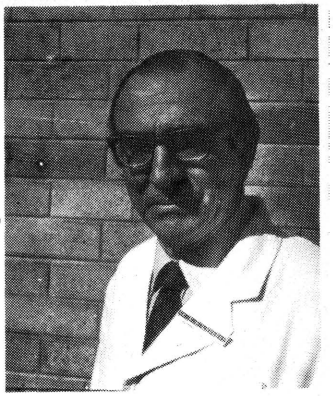
DISCUSSION

This was a fairly typical case of chronic pancreatitis. The response to treatment cannot be evaluated within one week as it is essentially a long term therapy. The owner was given enough tablets for a week plus a prescription for **"Cotazyme"** and instructed to administer them after meals.

The owner was visited about two months after the dog had been discharged and the dog was examined. Apparently the dog was eating very well and its condition had certainly improved drastically. The dog was weighed on the somewhat dubious bathroom scale and weighed 5,8kg. The scale however was underweighing so the weight gain was probably slightly more. In addition the skin and haircoat were much healthier in appearance with the skin being more pliable. No fresh faeces were seen but the owner claimed that there was no longer any foetid smell and that the faeces were much darker in colour. The owner was giving the dog two **"Cotazyme"** tablets a day as instructed. The dogs diet consisted of a dish of porridge and milk in the morning and a dish of mealie-meal, milk and mincemeat in the evening. This was considered adequate. The owner was advised to continue with the treatment.

The long term response to the therapy was excellent proving beyond doubt the final diagnosis.





IMPROVING SOW PRODUCTIVITY — SOME ASPECTS ON EARLY WEANING

R. K. LOVEDAY

(Department of Medicine, Faculty of Veterinary Science, U.P.)

One definition of sow productivity relates to the number of pigs produced per sow per year, but this concept really consists of three different facets: the time of weaning and hence the number of litters which may theoretically be farrowed annually, the litter size and the mortality rate during rearing. Aristotle (384 - 322 B.C.) has stated much the same facts in his "Historia Animalium": "Domesticated swine carry their young for four months and bring forth a litter of twenty at the utmost; and, by the way, if the litter be exceedingly numerous they cannot rear all their young."

Earlier attempts at exploiting increased sow productivity have generally been disastrous because of excessive mortality among the early weaned pigs, but the past few years have seen important advances in this area become possible. It has been found that a minimum live mass of about two and a half kilograms is necessary for piglets to be capable of ingesting sufficient dry matter to maintain a steady body growth. A pelleted, dry feed has been evolved as a substitute for sow's milk which has provided many key advantages such as ease of handling, palatibility, easily maintained hygiene and a very high nutritive value. The dry form has the further advantage of restricting feed intake.

The Beecham's system being described here involves the weaning at 7 to 12 days of age of piglets into three-tiered wire mesh cages in groups of nine animals. These cages are kept under strictly controlled climatic conditions in rooms where the temperature is maintained at $28^{o}C \pm 0,5^{o}C$, the relative humidity at 55-65% and air velocity does not exceed 15 m/second. The animals are kept in darkness, fed twice daily and preferably disturbed as little as possible. No drugs or antiboitics are used in feed or water. No faeces need be removed for the first 14 days from the cages, and there is evidently a safe, low-level bacterial challenge. The animals are regrouped in batches of 6 in second-stage flat-deck cages after 3 weeks when they weigh 7 to 9 kg and maintained there until attaining about 20 — 22 kg live mass. In the second stage accommodation the ambient temperature is maintained at $22^{o}C \pm 1^{o}C$. Under the conditions briefly outlined above it is claimed that mortality should not exceed 1% and a food conversion efficiency of 1:1,5 is attained. While these results are eminently

satisfactory, it must be borne in mind that the sophisticated system involved will demand an increased capital outlay of something between 10 and 35% when compared with a conventional 5 week weaning system. Secondly, the decrease in litter size noted in sows after a reduced lactation period must be reckoned with and will be discussed below.

The economic advantages of artificial rearing may be small after early weaning unless this is followed by a satisfactory reproductive performance in regard to early re-breeding, satisfactory conception rate and litter size. It appears that the sow's normal physiological processes are adapted to a period of lactation, during which ovarian activity and ability to breed are normally suppressed. Any decrease in the period of lactation has a deleterious effect on the subsequent "rebound" from the normal lactation anoestrus. This is illustrated in the data tabulated below from a recent paper by Svajgr et al. (1974):

Sows weaned on:	Day 2	Day 13	Day 24	Day 35
Average no. days to oestrus:	10,1	8,2	7,1	6,8
Percentage fertilised ova:	81,9	86,3	96,5	98,0
No. cystic follicles/Sow:	3,3	1,3	0,6	0,3
Mean 28 day conception rate (%):	68	92	100	100
No. of live embryos:	8,4	11,1	11,2	11,4
Percentage embryo survival:	54,3	70,7	71,6	79,5

These figures bear out the important economic fact that early weaning at less than 14 days may reduce the subsequent litter size by at least one and sometimes even two pigs per litter. reduction has previously been attributed to an abnormal incidence of early embryonic loss by Moody and Speer (1971)

Immediately post-partum there is extensive endometrial oedema reflecting the very high pre-partum oestrogen levels of foeto-placental origin in the sow. Some seventy-five percent of uterine involution is completed during the first week and the regeneration of the endometrium is fairly well advanced by the end of the second week. Since blastocyst attachment does not occur until 12 to 13 days after ovulation, it does not appear as if uterine involution will be major limiting factor to subsequent reproductive ability. The reasons for the early embryonic losses are therefore not yet properly understood

It has been stated that early weaning at ages younger than three weeks may have commercial possibilities in the future, but the following aspects still remain to make this an economically doubtful proposition at the present moment:

(i) Some 20% of sows will have reproductive problems, including smaller litters, return to oestrus and pseudo-pregnancy.

(ii) Post-weaning sow management must be subject to specific techniques of a high order.

(iii) The expense, time, patience and expertise required for absolute control of the environment and the management of early weaned piglets, particularly on the larger commercial scale.

Nevertheless, it may be recalled that hens were once used to incubate eggs, and, if the economic incentives become great enough, further research into the above situations will certainly be undertaken. Several noteworthy advances, particularly in rearing techniques have come out of work already done and the factors influencing post-partum reproductive ability in the sow are also slowly being better understood.

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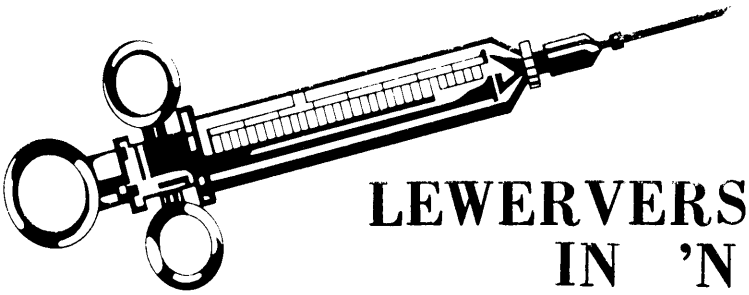
Svajgr, A.J., Hays, V.W., Cromwell, G.L. & Dutt, D.H. (1974) J. Anim. Sci. 38: **100** Moody, N.W. & Speer, V.C. (1971). J. Anim. Sci. 32:**510 – 514.**



LEWERVERSAKING IN 'N PERD

KLASGEVAL – F. Stegmann (B.V.Sc. V)

VOORWERP Donkerbruin Volbloed reun van ongeveer 18 maande oud.

GESKIEDENIS

Die perd is afkomstig van Barkley-Oos tesame met 'n groep vullens wat voor die jaaroud volbloedvullensveiling gevoer is met hawer, lusern, mielies, hawerstrooi, dikalsiumfosfaat en sout. Hulle is gehou in 'n klein kampie waar daar geen natuurlike weiding teenwoordig was nie. Vullens uit die groep het siek geword en gevrek met die volgende simptome: (1) hiperestesia; (2) "dullness". (3) anoreksie; (4) steier veral in voorkwarte en kruis soms voorbene; (5) stoot soms met kop teen muur.

'n Nadoodse ondersoek is op een van die vullens wat gevrek het te Onderstepoort gedoen. Die volgende is gevind: erge lewersirose met megalositose, geelsug, splenomegalie en nefrose.

KLINIESE ONDERSOEK

Habitus – 'n „dumb" uitdrukking op sy gesig. Staan met sy gesig teen die muur. Hy het egter op eksterne stimule gereageer.

Slymvliese – Kongestief met petechiae op conjunctiva en neusslymvlies. Die scelera was donker gekleur. 'n Ligte mucoïede lakrimasie en neusuitloopsel was teenwoordig terwyl uit die regter nares het 'n klein hoeveelheid ongestolde bloed gevloei.

Faekale flottasie – daar was 'n ligte (2+) **Strongylus** infestasie.

SPESIALE ONDERSOEK

1. **Urienanaliese** (Vry gekollekteer)

	16.5
Kleur	rooibruin
Neerslag	teenwoordig
S. A.	1,004
pH	6,0
Proteiën	spoor
Glukose	+
Bilirubien	++
Hb	++
neutrofiele	+
nierepiteel	+

2. **Hematologie**

	14.5	20.5	Normaal
Hb g%	20,4	11,0	8–14
RST x 10^6/mm^3	10,53	6,23	6–9
Ht %	55	27	24–44
W.S.T. x 10^3/mm^3	11,8	33,3	8,0
Neutrofiele %	72	–	58 ± 12
Limfosiete	25	–	2–10
Monosiete	3	–	2–12
Eosinofiele	0	–	2–12
Basofiele	0	–	0–3

3. **Bloedchemie**

(a)	BUN	15/5/74	25,8 mg%
		18/5/74	60,0 mg%
		20/5/74	55,0 mg%
(b)	SGOT	212 I.E. (Normaal 50–110 I.E.)	
(c)	Protrombientyd	30 sek (Normaal 14–17 sek)	
(d)	T.P.P.	14/5 6,35 g% (Normaal 7,39%)	
		20/5 5,2 g%	
		Alb: Glob. 0,47 (Normaal 0,7)	
(e)	BSP	Halftyd van 13,08 min.	
		(Normaal 2,8 ± 0,5 min)	

DIAGNOSE

'n Diagnose van pyrrolizidine alkaloied – vergiftiging is gemaak, gebaseer op die geskiedenis van soortgelyke gevalle van dieselfde plaas en die feit dat laboratorium toetse het die teenwoordigheid van lewerversaking aangedui het.

Differensiële Diagnoses

1. Babesiose
2. „Grain staggers"

BEHANDELING

1. **Lewerondersteuning** Thioctic acid (Thioctan) 50 mg / dag i.v. Glukoron-suur (Guronsan) 1 g daagliks i.v. Methionien 2,5 g daagliks i.v. Vit B kompleks (Parentrovite) 20 ml daagliks i.v.

2. **Haemokonsentrasie** 2L 5% dekstrose, 0,9% NaCl en 1L 0,9% NaCl daagliks i.v.

3. **Haemoglubinurie** In 'n poging om die hemoglobien so oplosbaar moontlik te hou en dus presipitasie in die nier te voorkom is HCO_3 toegedien. 50 mekw/dag i.v.

POST MORTEM

1. **Patologiës anatomiese diagnose**
   P.M. veranderinge lig

Algemene geelsug en kongestie
Hepatomegalie met nodulere hipertrofiese sirose
Splenomegolie
Nefrose
Haemoglobinurie
Edeem van caecum en colon
Haemoragiese gastritis
Verminose **Gasterophilus** en **Strongylus**

2. **Histopatologie** Het nefrose bevestig – daar was ook haemoglobien gietsels teenwoordig. Die lewer het galbuis proliferasie, megalositose, diffuse sirose en nodulêre hiperplasie getoon.

BESPREKING

Ten opsigte van die diagnose van lewerversaking kan die volgende genoem word.

1. SGOT gee slegs 'n indikasie van selnekrose, terwyl daar in dié geval lewernekrose, nierepiteelnekrose, asook hemoliese wat kon bydra tot verhoogde waardes.

2. Protrombientyd is verleng wat dui op 'n ernstige onvermoë van die lewerparenchiem om protrombien te sintetiseer.

3. TPP waardes is verlaag wat terselfdertyd in die lig van die haemokonsentrasie gesien moet word. Die plasma albumien verlaging volg op die onvermoë van die lewer om dit te sintetiseer. Die globulien was verhoog soos aangetref in lewerversaking.

4. BSP Die halftyd was verleng wat dui op 'n onvermoë om die middel uit te skei. Verlaagde bloedvloei veroorsaak deur lewersirose en haemokonsentrasie dra ook tot die verlengde halfleeftyd by.

5. Ter ondersteuning van pyrrolizidine alkaloïëd vergifting het die makroskopiese PM letsels die volgende getoon nl. lewersirose, algemene geelsug, edeem van die caecum en colon. Alhoewel die alkaloïëd primêr 'n hepatotoksien is, veroorsaak dit ook verslapping van gladdespier wat die edeem van caecum en colon verklaar. Die mikroskopiese lewerletsels was tipies vir chroniese pyrrolizidine alkaloied vergifting.

Wat egter atipies was, was intravaskulêre haemoliese. Soortgelyke gevalle is deur Tennant et al (1972) beskryf van lewerversaking met intravaskulêre hemoliese. Steyn (1949) beskryf egter ook 'n soortgelyke syndroom onder subakute seneciose.

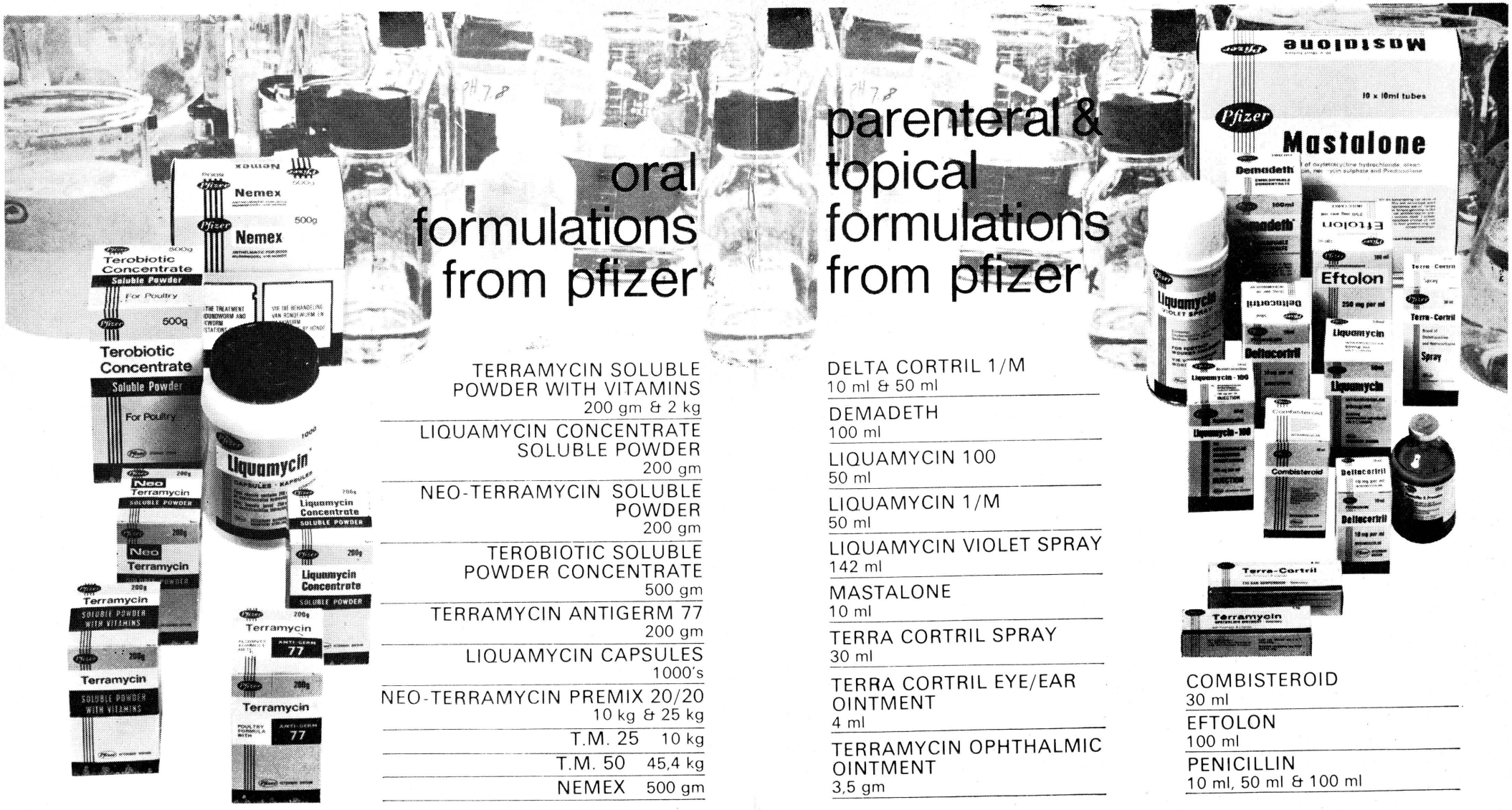
Dit is bekend dat pyrrolizidine alkaloïede 'n latente periode van tot twaalf maande kan besit en hou die feit dat die perde voor die veiling in 'n kampie sonder natuurlike weiding was, dus geen verband nie. 'n Verdere uitstaande kenmerk van die geval was die geweldige haemokonsentraise wat teenwoordig was.

Progressiewe nierversaking veroorsaak deur die haemoglobiengietsels was deur die stygende BUN gereflekteer.

Die reaksie van die perd op behandeling was swak. Die aptyd het swak gebly gedurende periode van hospitalisasie, alhoewel daar na 3 dae 'n effense verbetering in sy habitus was. Op die 4de dag het sy toestand sodanig versleg dat op genadedood met chloraalhidraat besluit is.

VERWYSINGS

Tennant, B.C. et al (1972) The California Veterinarian
Steyn, D.G. (1949) Vergiftiging van Mens en Dier, J.L. van Schaik Bpk.



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THE HIGHWAY SYNDROME

This is an acute, subacute or chronic neurotropic intoxication affecting groups of veterinary students. The classical syndrome is more applicable on a herd basis. Resistance seems to be more well developed in senior students.

Manifestation of the syndrome is usually associated with a history of stress — predisposing factors include tests, abscence of girlfriends and an idiopathic form, suspected to be a reaction against stricter res. rules.

AETIOLOGY:

Ethanol — this occurs in various solutions, concentrations and colours. Mixing of solutions is frequently practiced to achieve toxic levels. Administration is per os although cases have been reported of attempted topical application either by spraying into the face or simply pouring the agent over the head. Absorption, distribution, biotransformation and excretion was dealt with in a previous review (Experto crede No. 1 June 1974). An additional finding here is that renal excretion is facilitated by an inhibition of A.D.H. resulting in a pronounced polyuria. This may in fact lead to a severe electrolyte imbalance and concomitant dehydration.

SYMPTONS:

These occur in the acute, subacute or occasionally around Rag in the chronic form. Generally there is excitation followed by depression

CLINICAL SIGNS:

Ataxia — student moves forward in a zig-zag fashion.

Dysmetria — over and/or understepping — student also bumps into objects and may lose balance completely in which cases lacerations over the bony protruberances are a frequent complication. (d.d. — lead)

Wide based stance — usually seen when student has no access to any support, however it is also associated with ADH inhibition, in which case a wall or tree seems to play an important rôle.

Bellowing — this may occur amongst random individuals or as a group, in which case a spot diagnosis may be made from a considerable distance (d.d. — rugby match)

Ptosis — usually in advanced cases.

Acquired lingualism — there seems to be an increased ability to learn languages — so called English speaking students are often seen delivering brilliant speeches in Afrikaans and vica-versa. This symptom is usually confused by a partial paralysis of the musculus orbicularis oris resulting in slurred pronunciation.

Induced friendliness is a frequent manifestation — one usually sees this in pairs or groups with arms affectionately slung around each other in conjunction with bellowing, group ataxia and wide-based stance.

Aggression — this occurs infrequently but in such cases an applied knowledge of ethology and flight distances should be employed. This appears to be more a defence mechanism rather than blatant attack and blows are usually ineffective. A note of caution — patient may adopt a southpaw. stance, — these are extremely dangerous and caution should be excercised.

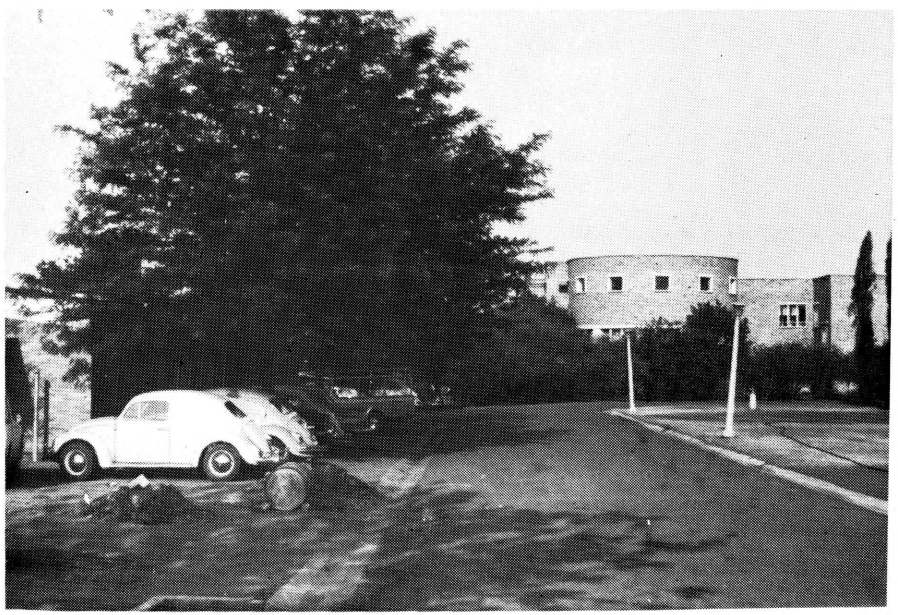
Miscellaneous symptoms such as fire fighting practice, stockcar racing, tightrope walking and trips to womens res. may be evident. Streaking has also been reported in isolated cases.

After the initial period of excitation the duration of which depends on the dose and half-life, a period of depression follows, usually manifested by an insensitive somnambulent state. This may occur in a variety of sites such as gutters, underneath beds, in corners or on park benches. After this period the patient develops a severe headache and a marked dehydration. Chicken-run mouth is often associated with both the above.

TREATMENT

1. In the early stages stop ethanol intake (this is usually a futile attempt, and the patient normally co-operates only when depression has set in.)
2. Put to bed in a quiet, dark room.
3. Four tablets acetyl salicylate
4. "Eno's" p.o. 10 mg/kg
5. Liver supportive therapy e.g. Thioctan, Guronsan. This may be used prophylactically 6 hours before ingestion of ethanol.
6. Cool succulent food.
7. Pen Strep i.m. to prevent possible secondary infection.

If response is poor one has to fall back on "boereraad" e.g. a stiff regmaker.



A view of outpatients avenue

BEK en KLOU UITBRAAK

(Letaba distrik)

Staatsveearts vakansie praktyk verslag deur W. Kruger B.V.Sc. IV

Staatsveeartse: Dr P. Bosman en Dr J. de Clerk

OORSPRONG VAN DIE BESMETTING

Waarskynlik deur middel van besmette wild wat langs die vol Olifantsrivier af migreer het tot by die huidige besmette omgewing en toe die drinkwater van beeste besmet het. Die menslike faktoor mag ook 'n rol gespeel het deur middel van besmette diere of vleisprodukte wat in die area ingebring is.

Wild

Rooibokke en koedoes is moontlike oordraer diere. Kruppel rooibokke is opgemerk. Geen wild is tot dusver uitgeskiet nie, behalwe vir ondersoekdoeleindes..

SIMPTOME

Koers, salivasie, mankheid en lusteloosheid.

LETSELS

Kom voor op die mondslymvliese (veral die tandvleis), tongepitaal en epiteel van die hoefkroon. Letsels is aanvanklik blase wat later bars, die epiteel afstoot en 'n rou wond laat wat reedlik vinnig opnees mits daar nie sekondêre konplikasies is nie.

LABORATORIUM TOETSE

(i) Epitaal: Monsters is in gebufferde gliserien oplossings weggestuur en SAT 1 is geisoleer.

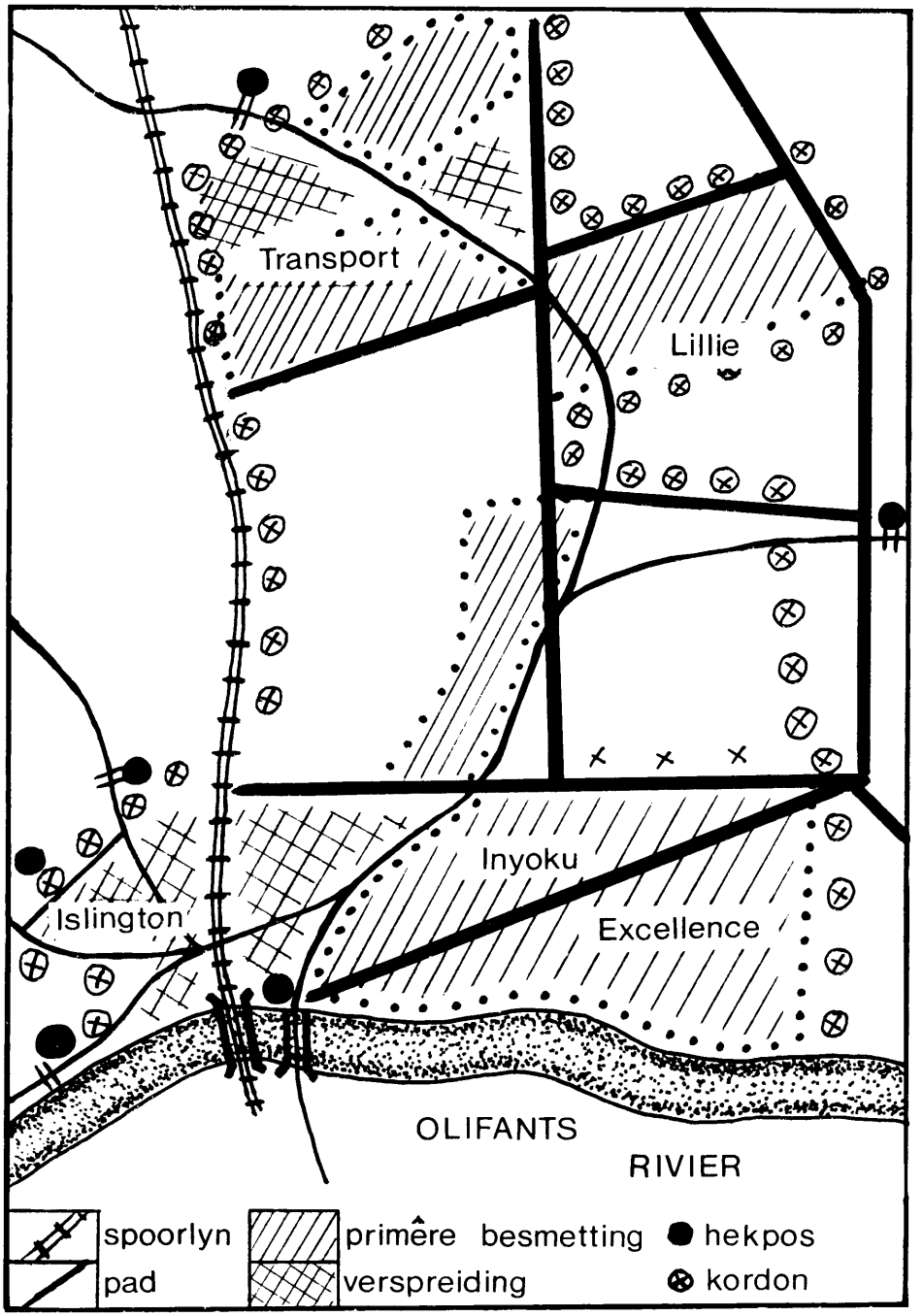
(ii) Serum: Bloedmonsters geneem net voor eerste inenting en drie weke na tweede enting.

BESPREKING VAN DIE LIGGING VAN DIE PLASE (Sien kaart)

Effektiewe skeidings word geskep deur die vol Olifantsrivier en die treinspoor wat langs sommige van die besmette plase loop. Die treinspoor skeiding is egter op een plek oorkom en het die infeksie na die plaas **Islington** versprei. Die rede hiervoor is waarskynlik omdat op die plek waar die infeksie deurgebreek het, was daar baie Bantoe kampongs en dit moontlik deur middel van besmette vleis versprei is.

Vlei ryke kampe word ook as skeidings beskou maar die feit dat die wild besmet is kanseleer die voordeel grootliks.

Die besmetting versprei (na 'n aanvanklike opvlamming) baie stadig op besmette plase. Die droë weer speel moontlik 'n rol maar die lae virulensie is onrusbarend.



BESPREKING VAN TABEL

Soos gesien kan word is die laaste nuwe geval op **Transport** reeds aangemeld voordat met inentings begin kon word. Die besmetting was dus waarskynlik reeds twee weke oud voordat dit aangemeld is. Dit verklaar die redelike hoë persentasie besmetting. Baie dieselfde geld vir **Inyoky**.

Excellence en **Islington** wys baie die normale verwagte beeld.

Lillie se lae persentasie besmetting kan tot 'n mate daaraan teogeskryf word dat die totale aantal beeste in twee troppe verdeel is en dat slegs die een trop van ongeveer 130 beeste besmet was.

Al die varke is afgemaak vanweë die kort dragtigheid periode van die vark en hoë virus populasie in 'n vark. Dit is dus baie moeilik om van die virus in lewende varke ontslae te raak.

Entstowwe 56 000 vol dosisse is vanaf Pretoria aangestuur. Dit is 'n bivalente inaktiveerde entstof vanaf Engeland ingevoer was SAT 1 en SAT 2 bevat.

'n Oorspronklike 3 ml dosis van twee inspuitings word met drie weke interval subkutaan gegee. Helfte van die dosis is aanvanklik gegee om die diere te sensiteer en die res van die dosis drie weke later. Op plase waar diere reeds voorheen geënt was (plase wat in die sogenaamde rooilyn val), was dit net nodig om een vol dosis te gee. Die plase wat nou die uitbraak gehad het sal moontlik verplig word om in die vervolg een keer per jaar te ent om die immuniteit te behou.

In die lig van die resultate verkry volgens die tabel en die feit dat dit moeilik was om wild- en veebeweging volledig te beheer wil dit voorkom of die entstof wel effektief was vir bekamping van die uitbraak.

INSPEKSIE GEDURENDE EN NA 'N UITBRAAK

Beskermende klere bestaan uit 'n oorpak, waterskoene en rubber handskoene. Voor en na inspeksie word die handskoene en waterskoene in 4% wassoda gewas. Oorpakke word op die plaas na inspeksie in 4% wassoda gedompel en later gekook.

Inspeksie van die tong, lippe, keelgebied en hoewe van elke dier word onderneem.

Daar word gepoog dat plaaseienaars so min moontlik self met die diere te doen kry en ook nie met beeste van ander boere in aanraking kom nie.

Bewegings

a) Vee: Geen beweging mag plaasvind van of na plase.

b) Wild: Geen grootskaalse wildbeweging is aangemeld nie. Kordonvoertuie ry nagskofte langs heinings om beweging te strem.

c) Heinings: Grootskaalse herstelwerk was nie moontlik sonder omheiningsmateriaal nie.

d) Kordonne: (i) Hekwagte voorkom vervoer van besmette materiaal (dierlike produkte, wildvleis ens.) deur die algemene publiek. (ii) Patrollies word per voet of per motor langs draadheinings bedags en snags gedoen rondom besmette plase.

Veeinspeksies

(i) Kontak plase word elke drie dae inspekteer. Slegs verdagtes word normaalweg gedek.

(ii) Plase geleë om dié plase word sewe daagliks inspekteer.

(iii) Plase in 'n groot omliggende area word veertien daagliks inspekteer.

Beeste in besmette gebiede word as dikwels moontlik en indien moontlik een keer per dag getel om beheer te verseker.

Plaas en Nr.	Eienaar	Datum van uitbraak	Aantal Diere op Plaas				Varke afgemaak	Aantal Beeste besmet	% Besmet	Laaste nuwe geval	1 e Inenting	2 e Inenting	K.E.P.	Duur van Besmetting
			Beeste	Skape	Bokke	Varke								
Transport 145	J.J.Odendaal	29.4.74	409	–	–	8	6	315	77	8.5.74	10.5 en 11.5	31.5 en 1.6	15.5	10 Dae
Inyoky 159	F.F.Fourie	30.4.74	446	–	53	25	25	293	64	5.6.74	11.5	5.6	12.6	37 Dae
Excellence 157	R.E.Wagner	9.5.74	285	–	–	–	–	134	46	4.6.74	11.5	4.6	11.6	27 Dae
Lillie 148	C.S.Botha	23.5.74	289	–	36	–	–	9	3	3.6.74	13.5	3.6	10.6	12 Dae
Islington 163	R.Mey & J.Swart	3.6.74	292	–	18	203	198	113	39	18.6.74	13.5	3.6	25.6	16 Dae

1. Die bokke is gereeld saam met die beeste geinspekteer en gebek. Slegs enkele bokke het vrotpootjie aan een poot getoon. Geen tekens van kwyling of verkoues het onder die bokke voorgekom nie.
2. **Transport:** Die eienaar het twee varke self geslag en uitgebraai onder amptelike teosig. Die kop, pote vel en viscera is verbrand onder amptelike toesig.
3. **Islington:** 5 Varke is dood voor waardasie gedoen is. Hierdie varke is onder amptelike toesig verbrand.
4. **K.E.P.** (Kliniese endpunt) Die datum geneem as 'n week na die laaste nuwe geval.

Vervoer Staats en spesiale voertuie voorsien.

Akkommodasie Veeartse en kantoor in 'n drie kamer gehuurde huis. SVI, VI en Bantoes in tente gehuisves.

Personeel

Staatsveeartse	–	2
HVI	–	1
SVI	–	35
Bantoes	–	133

BRANDSIEKTE BY SKAPE — Pietersburg distrik.

'n Privaatveearts het die vermoedelike uitbraak by die Staatsveearts aangemeld.

Die geskiedenis is dat die skape ongeveer ses maande gelede van Louis Trichardt gekom het. Die skraapsels was positief vir **Psoroptes ovis.**

'n Organiese fosfaat (Dazzel) dip program met agt dae interval word ingestel op besmette en aanliggende plase onder toesig. Die streeksveeinspekteer ondersoek die aanliggende plase om die verspreiding vas te stel.

The early morning lecture rush – statistics indicate that this occurs 5 days a week, 33 weeks or 165 times a year. This amounts to 660 times in a four year term at O.P. No wonder everyone starts looking wooly after a while.

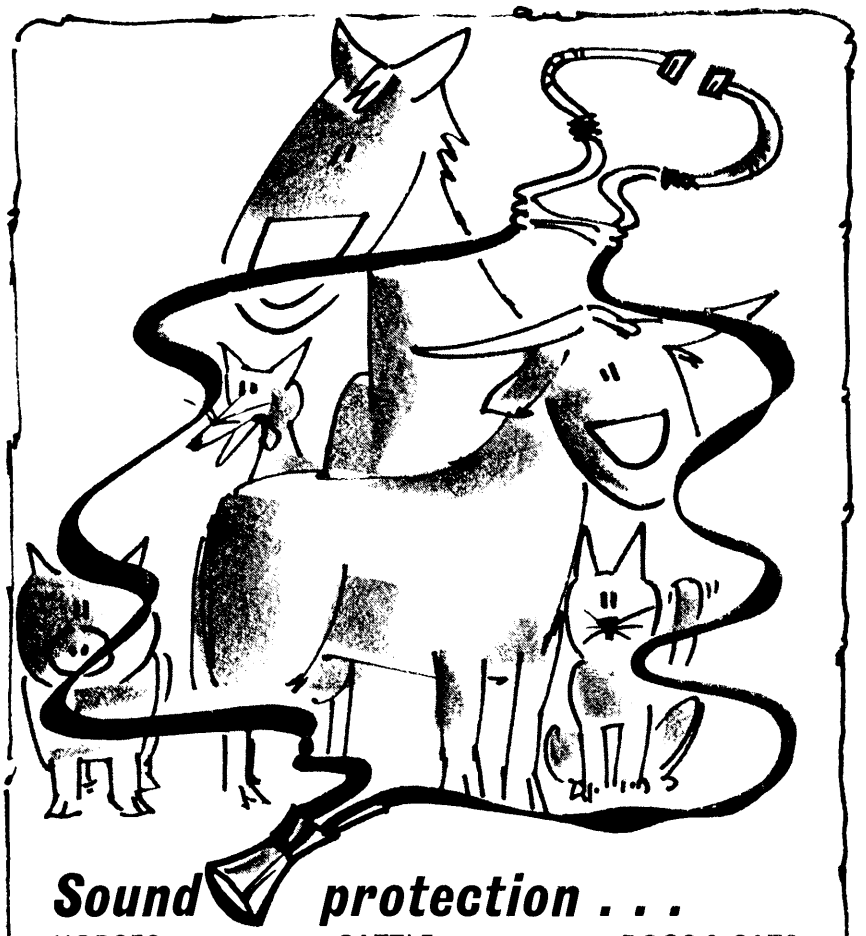
Staff changes.

Dr. J. le Roux takes over the position of Professor and Head of the Department of Anatomy.

Dr. R.K. Loveday has now taken up a position at the Meat Board but will still be delivering the final year course in Pig Diseases at the Faculty.

The South African Veterinary Students Association.

Paul du Preez is representing the S.A.V.S.A. at the Winter Congress of the International Veterinary Students' Association to be held in Dublin from the 29th December – 6th January 1975. The trip is once again entirely sponsored by PFIZER LABORATORIES to whom we express our appreciation.



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For further information, contact

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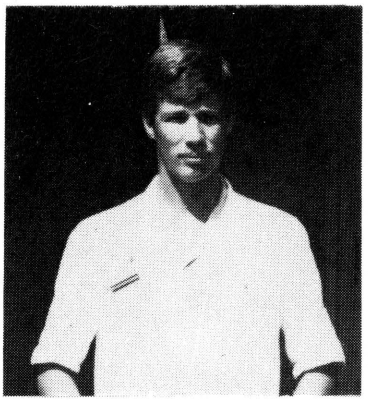
IBR — a disease with many pathological manifestations



J. SCHRÖDER
Dept. of Pathology,
Faculty of Veterinary Science, U.P.

First described as the cause of an upper respiratory tract infection in cattle in 1954[18], this Herpes virus has since been demonstrated to be the causative agent in a number of other syndromes. Preferential usage of the name Herpesvirus bovis is strongly advocated by recent authors.

The connection between IBR and Infectious Pustular Vulvovaginitis was revealed during the 1950's, when it was shown that the cause is in fact one and the same virus. More recent evidence suggests that the matter is not quite as simple as all this, and that strain differences might exist between serologically identical virus isolates.

Other syndromes since attributed to this virus include conjunctivitis, abortion[11] and stillbirth[6], encephalitis in calves[2,4] and mastitis[21].

INCIDENCE

The disease probably has a world-wide distribution. In the U.S.A. IBR is especially prevalent in intensive farming enterprises, and thus a cause of considerable economic loss.

The genital syndrome (IPV), previously known by a variety of other terms, eg. "Vesicular venereal disease", "Coital exanthema", "Blässchenausschlag" etc. has been described especially in Europe before the turn of the century.

Serological evidence indicates that the virus is spread throughout South Africa[19]. IPV is often seen in this country, but the respiratory disease has not been diagnosed. There is reason to suspect that IBR shall occur in this country as soon as the correct epizootiological circumstances become established.

Although primarily an infection of cattle, the virus has been isolated from naturally occuring cases of vaginitis and balanitis in swine[15,13], and respiratory involvement in goats[12].

PATHOGENESIS

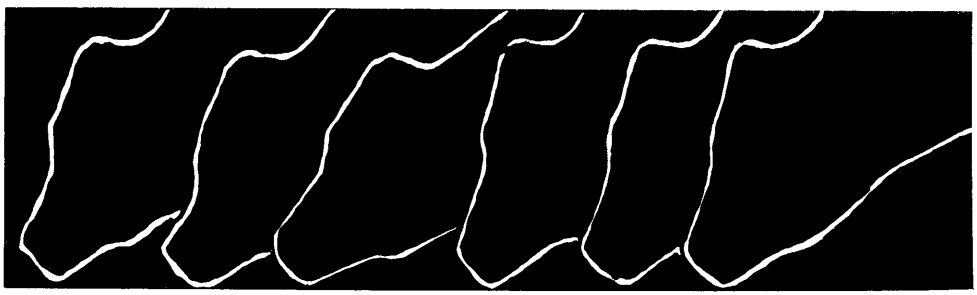
Following infection the viraemia usually remains at a low level and is of a short duration[6]. The virus primarily infects tissue of embryonal ectodermal origin[20] and more specifically epithelial surfaces of the respiratory and genital tracts, and the CNS.

It would appear that predelection sites in the genital tract are the epithelial coverings of follicles situated in the vulva, posterior vagina and anterior prepuce.

The basic lesion is a coagulative necrosis followed by the infiltration of neutrophils and lymphocytes into the necrotic tissue, and the tissue surrounding the lesion, respectively. Intranuclear inclusions are characteristically, but not always, present in the nuclei of degenerating parenchymal cells (and astrocytes in the CNS) on the periphery of the lesion. Accumulation of round cells in the perivascular spaces frequently occurs in nervous tissue in the vicinity of necrotic lesions.

In the pregnant cow, following maternal viraemia, the virus can localize in the placenta for an unknown period of time, without producing any microscopically related lesions[9], or proceed to cause a generalized peracute infection of the foetus. This is usually fatal within 24 - 48 hours[8], and results in expulsion of a severely autolized foetus within 4 - 7 days[6].

Kendrick (1973) suggests a transition from a foetal to a mature type of sensitivity to the virus as an explanation for the difference in the disease between the 8 months old foetus (generalized infection) and the newborn calf (localized lesions). However, Van Kruiningen et al. (1964) describe a naturally occuring generalized infection in a 10-day old calf.



PATHOLOGY OF THE VARIOUS MANIFESTATIONS

1. Infectious Bovine Rhinotracheitis

Non-specific systemic signs of illness accompany the specific symptoms of nasal discharge, dyspnoea, coughing, and often lacrimation and excessive salivation.

Lesions progress from hyperaemia and oedema of the nasal, laryngeal and tracheal mucosa, to a haemorragic and pseudomembranous inflammation. Schanning electron microscopic studies have revealed inflammation, loss of cilia, sloughing of epithelium and the formation of a pseudomembrane on the respiratory mucosa[14]. Regional lymph nodes are oedematous and even haemorrhagic. These signs are by no means specific for IBR.

Complications encountered are necrotic laryngitis and bronchopneumia, obviously after secondary bacterial infection.

Differential diagnoses are Shipping Fever (primary lung involvement) Bovine Malignant Catarrh, Virus Diarrhoea and Mucosal Disease (primary gastro-intestinal involvement[3]).

2. Infectious Pustular Vulvovaginitis and Balanoposthitis

Despite earlier names such as "Vesicular vaginitis" and "Bläschenausschlag", the lesion is never a vesicle.

After an incubation period of 1-3 days, following an infected mating, the vulva becomes oedematous, and there may be a slight excretion of exudate, matting the external hair. The mucosa of the vulva and vagina is hyperaemic and contains focal raised areas, 1-2 mm in diameter, which are usually pale, but may be haemorrhagic. A slight to fairly copious purulent exudate accumulating in the anterior vagina is a frequent finding. The raised areas progress to become pustules, which may coalesce, covered by a pseudomembrane, which slough off to leave an ulcer. At this stage the cow shows clinical evidence of pain during micturition, palpation of the genital tract and attempted service. These lesions apparently heal without scar formation.

Lesions on the mucosa of the penis and prepuce similarly occur after 1-3 days. They are initially of a similar nature to those in the cow, but can assume an alarming appearance following secondary bacterial infection of the ulcers, to which the bull is more prone. Healing of the lesions in the bull are frequently prolonged, resulting in scar tissue which may distort the penis, or cause adhesions between penis and prepuce.

Although it would appear from the lack of literary evidence that infection of the testes and accessory sex glands is of lesser importance in the bull, persistence of the virus in semen after infection (which can be extremely difficult to prove) may impair subsequent reproductive ability[24].

The microscopic picture is essentially the same in the cow and bull, viz. focal epithelial necrosis, frequently situated over lymphoid follicles. The necrotic tissue is invaded by neutrophils, while lymphocytes infiltrate the surrounding connective tissue. Degenerating epithelial cells contain intranuclear inclusions of Cowdry's type A. The centres of the lymphoid follicles are sometimes haemorrhagic. The neutrophils are eventually replaced by macrophages[7]. A pseudomembrane is formed when the necrotic material is pushed off.

3. Conjunctivitis or Keratoconjunctivitis.

This sometimes occurs as an entity in the abscence of repiratory involvement[17,22]. The inflammation of the conjunctiva and palpebral oedema gives rise to the increased lacrimation or serous ocular discharge, which can become mucopurulent subsequent to secondary bacterial infection. Opacity of the cornea occurs, but rarely proceeds to ulceration.

From the above it is evident that the gross features would be difficult to distinguish from those elicited by **Chlamydial** or **Moraxella bovis** infections.

4. Abortion.

No characteristic gross lesions are observed in the foetus. The extensive autolysis – producing a generalized brownish discolouration of the foetus, and soft, friable viscera – and generalized oedema – manifested by blood-stained fluid in the pleural and peritoneal cavities, and oedema of the lung septa, perirenal fascia and foetal membranes – and non-specific[10] Although the focal necrosis is usually sub-macroscopic (0,2 to 1 mm in diameter), Kendrick (1973) describes a process of necrosis and haemorrhages in the foetal kidney which can destroy most of the cortex, and is of course grossly visible. The interstitial haemorrhage is considered as being implementary in the accelerated spread of the virus.

As previously stated, the basic histopathological lesion is a focal, disseminate, coagulative necrosis in most tissues. This most consistently occurs in the liver, but is in addition also described in the kidneys, lungs, spleen, lymph nodes, thymus, adrenals and brain[10,6].

The necrosis, which is scattered throughout the organ without regard for architecture, is usually spherical, initially sharply defined, and is similar in appearance in all organs. Neutrophils accumulate at the periphery of, and infiltrate the necrotic tissue. Calcification of necrotic debris is seen in many lesions. An inflammatory reaction in the surrounding tissue is very rare.

Where necrosis in the liver involves a portal tract it usually leads to proliferation of the reticulo-endothelial elements. In the lungs evidence of the oedema seen macroscopically, is seen in the form of diffuse thickening of alveolar and interlubular septa, in addition to the focal necrosis[10] Necrotic lesions in the brain, kidney, adrenal cortex and lymph nodes may be haemorrhagic[6]

Although considered as diagnostic, intranuclear inclusions are only seen in fresh tissues and will thus probably rarely be encountered in naturally occuring aborted foetuses.

By far the most important differential diagnosis regarding the microscopic picture would be Listerial abortion.

5. Meningoencephalitis

Affected calves clinically show rotation of the head, unsteadiness of the hind quarters, pressing of the head against solid objects, grinding of the teeth, lethargy, reduced withdrawal and anal reflexes, opisthotonus, and paddling movements of the feet.

No macroscopic lesions are evident. Histological examination reveals extensive degeneration and necrosis of neurones and nerve fibres in the cerebrum, midbrain, pons, medulla oblongata and cerebellum. There is a gliosis, small accumulations of neutrophils in more severely affected areas, and perivascular cuffing. Intranuclear inclusions can be found in many of the astrocytes and degenerating neurones. A non-purulent meningitis is usually evident over affected areas[5].

DIAGNOSIS (1, 17, 16)

A tentative diagnosis based on the clinical symptoms, and the gross and microscopic pathology, can be confirmed by any of the following methods:

1. Demonstration of a significant rise in the neutralizing antibody titre of paired serum samples collected during the acute phase and the convalescent period.
2. Isolation of virus from affected tissues, circulating leucocytes (buffy coat), nasal, ocular and vaginal discharges after cultivation on BEK (Bovine Embryonic Kidney) tissue culture.
3. Fluorescent antibody technique on aborted material or affected tissue.

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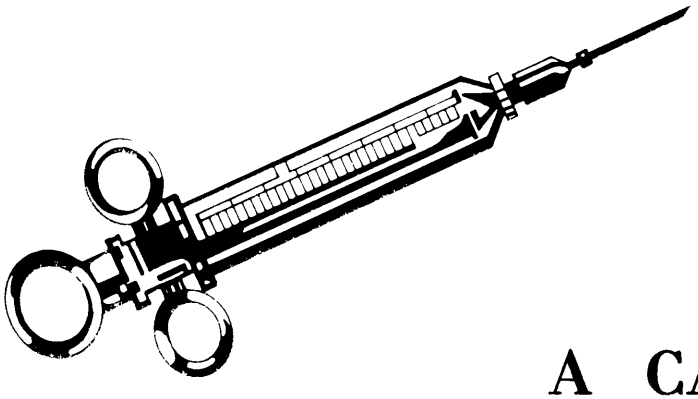


A CASE OF INTRAHEPATIC CHOLESTASIS

Class case — M. Gardner B.V.Sc.V. (1971)

This condition is not well documented in Veterinary literature, but has been described in Human Medicine.

SUBJECT:

A one year old miniature Dachshund was presented for examination. She weighed 2,9 kg.

ANAMNESIS.

Several weeks prior to the onset of the symptoms the dog had suffered from a severe and chronic skin infection. It had been successfully treated by a veterinarian in Pretoria. The treatment was "Stovarsol" at a dosage rate of 130 mg b.i.d. for a period of two to three weeks.

Some weeks later the owners complaint was that the dog was "off colour" and vomited periodically. The patient was referred to Onderstepoort.

CLINICAL FINDINGS.

1. Rectal temperature 38,3 C.
2. Pulse 110 per minute.
3. Respiration 30 per min.
4. The patient was slightly depressed and there was a slight drop in condition.
5. Marked icterus of all mucous membranes
6. Splenomegaly.
7. Slight hepatomegaly.
8. Capricious appetite.

9. Tenesmus defecatio and constipation. Greasy grey stools.

10. Haematology.

TABLE I.

	21/4	4/5	Normal
Hb.	14,15	11,60	12 –18 gm%
RCC	5,48	7,08	5,5 - 8,5 x 10^6 / mm^3
Ht	58	40	45%
ESR	25	55	5 - 25 mm
WCC	14,600	5,900	11,000 / mm^3
Neut.	80		70%
Lymph.	14		20%
Mono.	6		5%
Eos.	0		4%
Baso.	0		0%

This is routinely done in all cases admitted to the Dept. Medicine.

11. Urine analysis. See table 2.

12. Blood chemistry (heparinised blood). See table 3.

13. Radiographic examination.

Radiographs of the abdomen were taken to eliminate the possibility of foreign body obstruction and to see whether any hepatic abnormalities could be seen. The radiographs, however, revealed nothing conclusive as far as the liver were concerned and there was no foreign body. Variable areas of mottled translucency in the intestines indicated the presence of faecal masses and flatulence.

EXPLORATIVE LAPOROTOMY AND BIOPSY.

The blood chemistry and urine analysis indicated some liver pathology. This could best be confirmed by laporotomy. A midventral incision through the linea alba was made in the anterior 1/4 of the abdomen. This revealed that the liver was mottled, pale and friable (mild hepatomegally and hepatic degeneration). The gall bladder was empty and flabby. There were no other abnormal findings.

A liver biopsy was taken by inserting 2/0 chromic catgut sutures. 4 simple, overlapping, interrupted sutures were used to seal off the blood supply to a triangular portion of liver. This portion was then removed and placed in 10% formalin for 48 hours.

HISTOPATHOLOGICAL EXAMINATION

The histopathological examination revealed that the liver had diffuse cloudy swelling, focal disseminated coagulative necrosis portal fibrosis and a decrease in bile ducts.

DISCUSSION OF ABNORMAL FINDINGS.

The icterus and general depression can be attributed to the liver malfunction. The icterus being as a result of bilirubin in the plasma, and the depression as a result of disturbance of the liver's function as storage organ for essential substances and as a detoxifying organ.

The hepatomegally was most probably the result of the degenerative process.

Digestive disturbances manifested as constipation and foetid stools can be attributed to an

absence of bile secretion into the duodenum. Bile usually assists in the digestion of fats; the absorption of fat soluble vitamins; activates pancreatic enzymes and is a mild laxative. Clinically the appetite is capricious and th' patient shows vomition. Both these clinical symptons were present in this patient.

The renal nephrosis indicated in the urine analysis and the mechanical diuretic effect of the retained bile salts probably explain the haemoconcentration (haematocrit 58% on 21/4) that seems to be out of keeping with the other findings.

Changes in the ESR have no specific indication but are attributed to ionic status of substances in the blood and to viscosity. With a gradual increase in ESR prognosis becomes guarded.

The liver damage was further aggravated by bile salt retention which would be inclined to cause pressure necrosis of hepatocytes. Bile salts are also irritant.

Owing to the time lapse between examination, and the administration of Stovarsol histo-pathological evidence of an allergic intrahepatic cholangitis is not conclusive. The history of Stovarsol dosing being toxic is, however, conclusive.

The increase in W.C.C. is probably the reaction to a mild infection of a bacterial nature. This focus was most likely in the liver.

The increased bilirubin in urine (conjugated) and the negative result for urobilogen indicate a biliary retention. The other abnormal urinary findings (Table II) indicate a nephrosis. This was most probably a toxic nephrosis, toxins being derived from the infection mentioned earlier and also endogenous toxins that are not detoxified in the liver.

The increased conjugated bilirubin indicates an obstructive icterus. However, the Van den Bergh tests results were as follows:

Total Bilirubin:	18,4 mg%
Conjugated:	6,8 mg%
Unconjugated:	11,6 mg%

These results indicate that the icterus is largely the result of hepatocelluiar damage.

To evaluate the result, however, other findings must be taken into account. The patient showed no sings of hepatic failure e.g. severe depression, no anterior abdominal pain and no real enlargement of the liver. The faeces and urobilogen results tend to indicate more of an obstructive liver pathology. The elevated SAP values further support the obstructive theory.

A case similar to this one has been described by W.D. Malherbe (J1 SAVMA 30 (2) (1959). Here the case was claimed to be an allergic intrahepatic cholestasis caused by Stovarsol (an organic arsenical)'

The hypothesis of the case in question is as follows. The computed dose of Stovarsol for this case is 128 mg/day (20 mg/ib) for a period not exceeding 4 days. The patient in fact received twice this dose for 5 times as long. The resultant liver damage was irrepairable and the manifestation of allergy was superceded by a non-specific bacterial hepatitis.

FINAL DIAGNOSIS

Final diagnosis is hence intrahepatic cholestasis.

A pertinent quotation is that patients in human medicine with this type of condition "feel and appear considerably less ill than those with a comparable icterus caused by hepatocellular disease." This was a prominent observation in this case; severe icterus and a relatively lively habitus.

DIFFERENTIAL DIAGNOSES.

1. Leptospirosis
2. Billiary fever.

3. Other hepatotoxins
4. Cholelithiasis
5. Biliary dyskinesia
6. Tumour causing occlusion of common bile duct.
7. Chronic pancreatitis.

TREATMENT PROFILE

An enema was administered to evacuate the large intestine.

The remainder of the treatment composed of the administration of large doses of liver protectants; Vit B Co; Vits A, D, E, K; choloretics; a fat free and low protein diet was encouraged.

Despite treatment the condition of the patient gradually deteriorated and finally died. The post mortem confirmed the clinical diagnosis.

TABLE II

Urine Analysis

Urine was collected free.

	27/4	Normal value
S.G.	QNS	1,015 - 1,045
pH	6,0	6,0 - 7,0
Gluc	- ve	- ve
Ketones	- ve	- ve
Bilirubin	+++	- ve
UBG	- ve	- ve
Protein	+	- ve
RBC	- ve	- ve
WBC	+ neutr.	- ve
Epith.	RTE +++	Bladder +
	Bladder cells	squamous epith.
Casts	Cellular casts	- ve

TABLE III

Blood Chemistry

Blood collected in heparin.

	21/4	Normal
BUN	14,7	10 - 20 mg%
SGOT	27	30 - 90 King Units
SGPT	249	30 - 100 King Units
S.A.P.	62 +	3 - 13 King Armstrong units
Tot. cholest.	1157	150 - 250 mg%
B. Sug.	87	60 - 100 mg%
Bilirubin tot.	18,4	0,1 - 1,0 mg%
Bil. Conj.	6,8	0 mg%
Bil. unconj.	11,6	0 mg%

COMMENT

With reference to "**E.coli** – a problem in the Natal midlands" Experto crede No. 1 June 1974.

Dear Sir,

Excerpt – One paragraph is of concern to me as it may confuse your readers and that is the one relating to the use of TYLAN at a therapeutic level in the treatment of **E.coli** infection, which gave poor results. This is understandable as TYLAN is not indicated in the treatment of **E.coli** infections, but is the preferred form of swine dysentery (**spirochaete** or **vibrio coli**) and gram positive organisms, which are not usually a factor in enteric disorders.

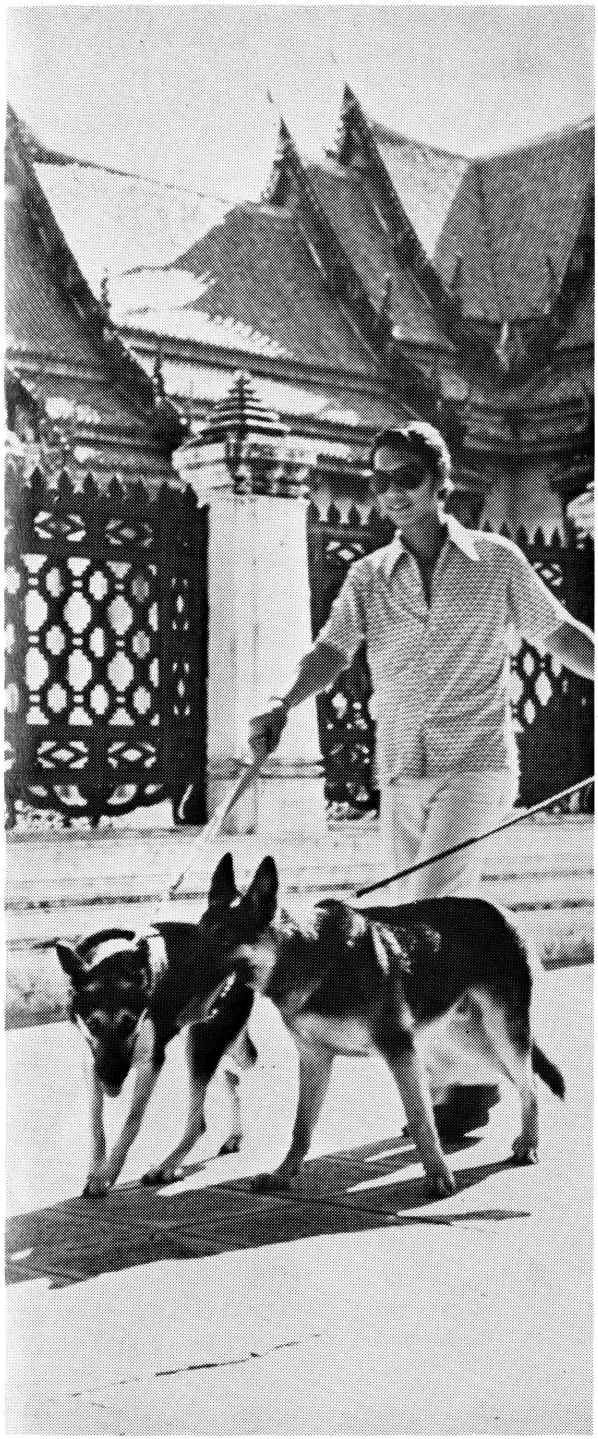
Yours sincerely
L. M. von Essen
(Director – Elanco Agricultural, Veterinary and Industrial products.)

Dear Sir,

In reply, I wish to state that at no time was TYLAN, a macrolide, ever considered to be effective against Gram negative bacteria, and in particular against **E.coli**. The inclusion of TYLAN in the ration of the pigs under consideration was done prior to a diagnosis being made. I might add that TYLAN was freely available to framers at that particular time. Further, at that time **Treponema hyodysenteria** (Swine dysentery) was responsible for considerable losses amongst pig herds in Natal. TYLAN was considered to be the drug of choice in the treatment of this disease and hence its inclusion in the ration. The fact that TYLAN was ineffective thus excluded Swine Dysentery as the cause of the scours.

Trusting that this clarifies the situation,

Yours sincerely
M.H. Lowry.



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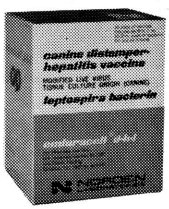

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