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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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CONTENTS/INHOUD

Contents continued on page 1 *Inhoud vervolg op bladsy 1*

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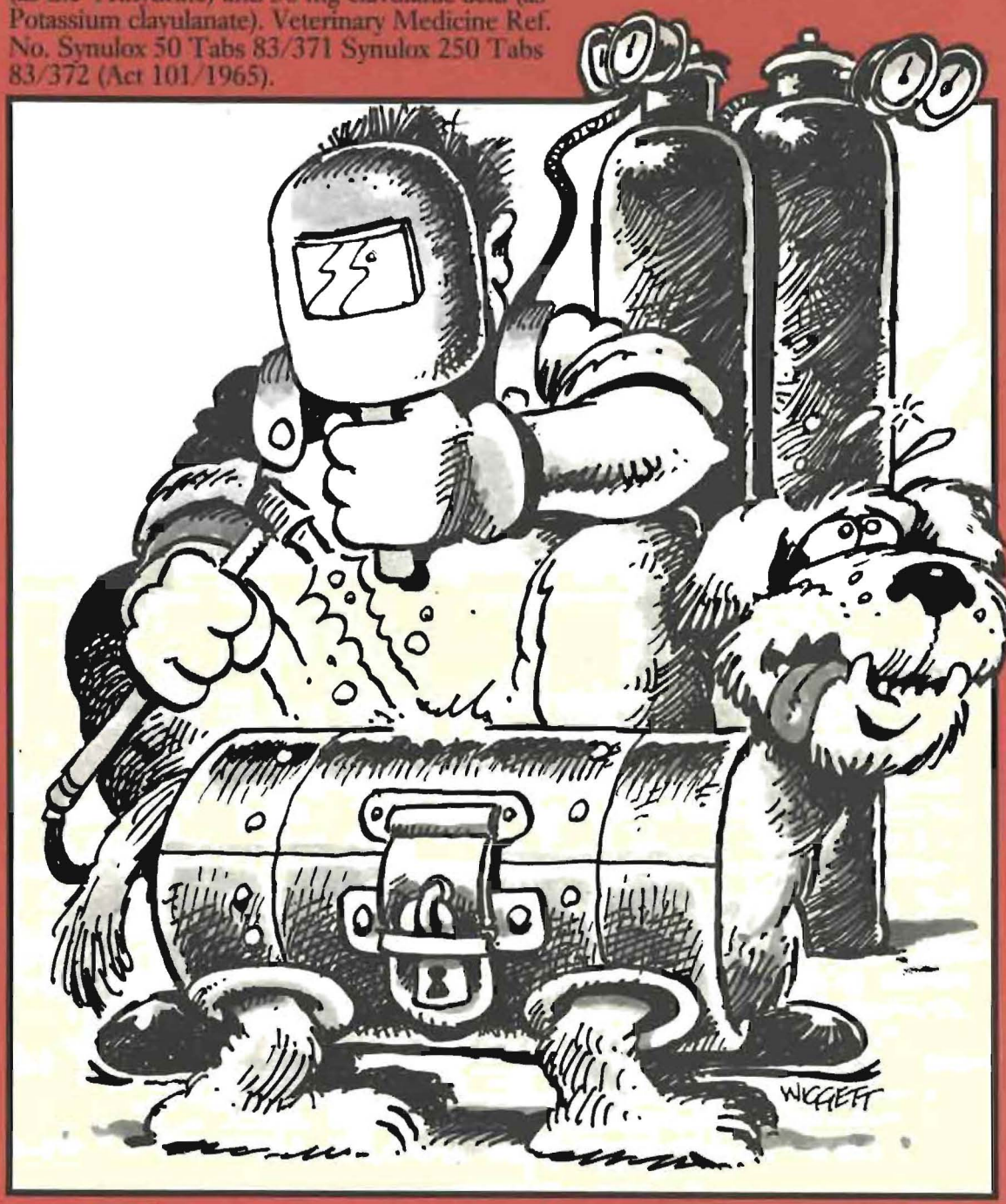

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Abstracts **Uittreksels**

Index to Advertisers **Advertensie-Opgaaf**

ABSTRACT **SAMEVATTING**

DISPERSAL, DENSITY AND HABITAT PREFERENCE OF THE BLOW-FLIES *CHRYSOMYIA ALBICEPS* (WD.) AND *CHRYSOMYIA MARGINALIS* (WD.) (DIPTERA: CALLIPHORIDAE)

16 000 *Chrysomyia albiceps* and 52 000 *C. marginalis* adults were radioactively labelled with ^{32}P-orthophosphate and released in the northern Kruger National Park, South Africa. After a 1-week dispersal period 69 baited blow-fly traps were placed in different habitat types and at varying distances around the release point. *C. albiceps* were subsequently found to have covered up to 37,5 km and *C. marginalis* 63,5 km, suggesting dispersal rates per day of 2,20 km and 2,35 km for the 2 species, respectively. Calculation of density using the Lincoln Index yielded estimates per heactare of 7,56 *C. albiceps* and 29,03 *C. marginalis*. Both species were trapped more numerously in forested environments than in open scrub, and both avoided arid scrubland. (Braack, L.E.O. & Retief, P.F. 1986. Dispersal, density, and habitat preference of the blow-flies *Chrysomyia albiceps* (Wd.) and *Chrysomyia marginalis* (Wd.) (Diptera: Calliphoridae). *Onderstepoort Journal of Veterinary Research*, **53**, 13 – 18 (1986).)

ABSTRACT **SAMEVATTING**

PROOF OF TRANSOVARIAL TRANSMISSION OF *COWDRIA RUMINANTIUM* BY *AMBLYOMMA HEBRAEUM*

Transovarial transmission of *Cowdria ruminantium* by *Amblyomma hebraeum* does occur in certain instances. Both the transovarial and the filial infection rates appear to be very low. The infection may reappear only in the adults or nymphae, or in all 3 stages of the tick's life cycle. (Bezuidenhout, J.D. & Jacobsz, Catherina, J., 1986. Proof of transovarial transmission of *Cowdria ruminantium* by *Amblyomma hebraeum*. *Onderstepoort Journal of Veterinary Research*, **53**, 31 – 34 (1986).)

ABSTRACT **SAMEVATTING**

PARALYSIS AND LIPOFUSCIN-LIKE PIGMENTATION OF FARM STOCK CAUSED BY THE PLANT, *TRACHYANDRA LAXA* VAR. *LAXA*

A paralytic condition of farm stock in South West Africa, characterized by prominent neuronal and some mild extraneuronal pigmentation, is described. The distribution of the pigment, which was mainly located in the larger neurones of the brain and spinal cord, is given. Experimental evidence, obtained by feeding the plant, is presented that the condition is caused by *Trachyandra laxa* var. *laxa*. The histochemical features of the pigment proved to be compatible with a lipofuscin. (Grant, Rina, C., Basson, P.A. & Kidd, A.B., 1985. Paralysis and lipofuscin-like pigmentation of farm stock caused by the plant, *Trachyandra laxa* var. *laxa*. *Onderstepoort Journal of Veterinary Research*, **52**, 255 – 259 (1985).)

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ARTICLE ARTIKEL

ACUTE POSTOPERATIVE DIARRHOEA IN COLIC HORSES

ANU PUOTUNEN-REINERT* and B. HUSKAMP*

ABSTRACT: Puotunen-Reinert A.; Huskamp B. **Acute postoperative diarrhoea in colic horses.** *Journal of the South African Veterinary Association* (1986) 57 No. 1, 5-11 (En). Tierklinik Hochmoor, 4423 Gescher-Hochmoor, West Germany.

A retrospective study on surgical cases of colic in horses (n = 216) revealed that 42 (19,4%) developed post-surgical diarrhoea. *Salmonella* spp. were isolated in 6 (16,2%) of the cases (n = 37) exhibiting diarrhoea. In 35,7% of the cases (n = 42) recovery from surgery was disturbed by other complications; 23,8% (10/42) died, 2 of which from primary acute diarrhoea due to salmonellosis. Most of the outbreaks of diarrhoea occurred in winter and spring. From the associated variables examined, the duration of colic signs revealed a significant difference between the cases which developed acute postoperative diarrhoea and the other surgical cases.

Key words: Postoperative diarrhoea, colic, horses, duration of colic signs.

INTRODUCTION

As a result of recent advances in surgery, anaesthesia, and medical management, recovery rates following surgical treatment of equine gastrointestinal disorders have increased[12 20 32], but many postoperative complications still reduce the success rate. Surgical treatment is always stressful to the patient, especially in cases of colic, which is often accompanied by circulatory disturbances and shock.

One of the most severe post-operative complications is acute diarrhoea[25 27 44]. Stress is recognised as a triggering factor for acute diarrhoea[11 19 25 27 30 44] and general anaesthesia and surgery[21 27], particularly abdominal surgery[11 25 30 44] seems to be of the most severe forms of stress. Surgical colic cases usually also have gone through the stress of initial disease[25 27] and have usually also been transported[25 44]. Additional stress factors for them are pre- and postoperative starvation[41 44] and intensive medical treatment[44].

The prevalence of acute post-operative diarrhoea in a large number of surgical colic cases has not been examined in any of the reviewed surveys. The present retrospective study was thus undertaken to determine the frequency and occurrence of acute post-operative diarrhoea in colic horses and to appraise factors associated with these outbreaks.

MATERIALS AND METHODS

Surgical cases:

The present retrospective study was performed on 216 of the 295 surgical colic horses hospitalized at the Hochmoor Animal Hospital, West Germany, in the year 1982. The 79 horses excluded from the study had succumbed either before or during surgery or did not survive more than 62 hours after the operation; none of them exhibited diarrhoea during the time of hospitalization.

Treatment

All the cases were operated on under general anaesthesia, which was induced with a combination of glyceryl guaiacolate-ether and thiopental-sodium, given intravenously, and maintained with a mixture of halothane, nitrous oxide, and oxygen by inhalation. Neostigmine (neostigminemethylsulfate 0,05%) was given during anaesthesia and thereafter to effect until a normal intestinal function was achieved. Systemic antibiotic therapy (procaine-penicillin-G 200 mg/ml and dihydrostreptomycin 40 mg/ml: 100 ml intra-abdominal during the operation and 20 ml/500 kg body mass twice daily, intramuscular) was used for three days after surgery. Dehydration and acid-base imbalances were corrected with intravenous fluids (physiological sodium chloride solution, Ringer's solution, plasma expanders, and sodium bicarbonate either as physiological or concentrated solution, depending on the degree of dehydration and acid-base status). Kaolin and pectin were given orally during the episodes of diarrhoea, according to the manufacturer's recommended doses. Parenteral antimicrobial therapy was started, without waiting for the results of faecal bacteriological examination, if the horse exhibited fever, leukopenia, leukocytosis, cardiovascular changes suggesting endotoxaemia, or evidence of systemic infection. The initial antimicrobial therapy was changed only in cases where the condition of the horse did not improve or became worse. Gentamicin was chosen if the clinical signs of the horse were indicative of enteric salmonellosis e.g. severe fulminating diarrhoea with depression and anorexia, marked fluid and acid-base disturbances, and signs of endotoxaemia, anaphylaxis or shock. Patients in which intestinal resection was carried out, were usually starved for two days and other cases for one day after the operation.

In the present study, 'diarrhoeic' was regarded as horses suffering from diarrhoea for at least 24 hours. Horses with soft faeces or with faeces varying between soft and firm, were not considered to be 'diarrhoeic'.

Observations

The following 16 variables of the patients were recorded: breed, sex, age, diagnosis of colic, duration of colic signs, duration of anaesthesia, type of surgical therapy, duration of neostigmine administration, recovery from surgery, beginning and duration of diarrhoea, rectal temperature, total blood leukocyte count, results of faecal bacteriological and parasitological examination, and therapy used against diarrhoea. The survival in cases of acute post-operative diarrhoea and the seasonal distribution of outbreaks of diarrhoea were also recorded.

*Tierklinik Hochmoor, 4423 Gescher-Hochmoor, West Germany.

0038-2809/86/01/0005-0011

The horses were classified into 5 groups: Warm-blooded, Cold-blooded, Thoroughbred, Trotters and Ponies.

The diagnosis of colic was based on the findings observed during the abdominal operation.

The duration of colic was taken as the time from the moment when signs of colic were first noted until the time of surgery.

Surgical therapy was divided into 2 groups: intestinal repositioning and intestinal resection.

All the factors which may have had a bearing on recovery were noted.

Rectal temperature was recorded when the horses arrived at the hospital and thereafter every morning and evening.

Clinical pathology

Total blood leukocyte counts were determined from venous blood immediately after the signs of diarrhoea were observed or a rectal temperature over 38,5°C was recorded; it was thereafter repeated daily until the counts reached the normal level and the patient did not suffer from diarrhoea or show a higher than normal rectal temperature. Total blood leukocyte count were recorded as:

(a) leukopaenia, if total leukocyte count once or more was determined to be less than $5\,000 \times 10^9/\ell$
(b) normal[13], if total blood leukocyte count on no occasion was less than $5\,000 \times 10^9/\ell$ or more than $10\,000 \times 10^9/\ell$
(c) leukocytosis, if total blood leukocyte count in one or more determinations was over $10\,000 \times 10^9/\ell$, but on no occasion less than $5\,000 \times 10^9/\ell$, and
(d) leukopaenia-leukocytosis, if the horse exhibited leukocytosis following a period of leukopaenia.

Faecal samples for bacteriological and parasitological examinations were collected from the rectum using a plastic glove. Bacteriological examinations and antibiotic sensitivity tests were routinely performed in the Veterinary Laboratory, Münster. Parasitological examinations were made in the hospital by a routine flotation method and the result was categorised as negative or as a mild, moderate, or massive parasite invasion.

Causes of death were assessed based on autopsy findings.

Statistical analyses

Student's t-test was used to test the significance of differences between the groups. Independence of the variables was tested by the chi-square test for frequency tables.

RESULTS

Of 216 horses with colic that have undergone surgery, 42 (19,4%) contracted acute post-operative diarrhoea.

No significant differences in breed, sex, and age distributions were found between the horses with diarrhoea and surgical colic cases that did not contract diarrhoea. Of the 42 horses with diarrhoea 36 (85,7%) were Warm-blooded, 2 (4,8%) Thoroughbred, and 4 (9,5%) Trotters; 5 (11,9%; n = 42) were stallions, 15 (35,7%) mares, and 22 (52,4%) geldings; 26 (61,9%) were from 3 to 8 years old, 2 (4,8%) under one year, and 14 (33,3%) from 9 to 16 years old.

There was no significant difference either in the part of the gastro-intestinal tract involved between the horses which developed diarrhoea post-operatively, and the other surgical colic cases; 19 (51,4%) involved the small intestine, and 18 (48,6%) the large intestine. The cause of colic in each of the horses is given in Table 1. The intestinal tract of 3 horses was not involved as a cause of colic.

Table 1: Distribution of diarrhoeic horses in different diagnoses and the total number of surgical colic horses in the year 1982

Diagnosis	Number of cases with diarrhoea	Total number of surgical colic cases, 1982
Small intestinal incarceration	7 S, +	41
Traumatic abdominal hernia	1 +	1
Small intestinal strangulation	5 S	34
Ileocaecal intussusception	1 +	4
Impaction of the duodenum	1	1
Impaction of the ileum	3 S, +	24
Paralytic ileus of the small intestine	1 +	10
Torsion of the large colon	1	32
Displacement of the large colon	6 S, +++	36
Herniation of the large colon into the nephrosplenic space	6 S	41
Mesenteric hernia of the large colon	1	1
Impaction of the caecum	1 S	3
Impaction of the small colon	1	2
Obturation of the colon by enteroliths	1	2
Megacolon descendens	1	1
Mixed ileus*	1 +	5
General meteorismus	1	9
Intra-abdominal abscess	1 +	3
Torsion of the uterus	1	2
Normal situation	1	6
Other	—	79
Total	42	295
Site:		
Small intestine	18	122
Large intestine	19	129

* A combination of mechanical and functional ileus
S = a *Salmonella* positive case
\+ = non-survivor

The duration of signs of colic revealed a significant difference ($p < 0,01$) between the horses which developed acute diarrhoea after abdominal surgery and those which did not. The duration of signs of colic in all cases with post-operative diarrhoea was at least 8 hours ($\bar{x} = 30,24$ h; standard deviation (SD) = 35,33; logarithmic mean (lg) $\bar{x} = 2,97$; SD = 0,89; n = 42); of the horses with colic 21,4% (9/42) had suffered diarrhoea from colic for more than 36 hours. In contrast, of those that did not develop diarrhoea, 30,2% (n = 174) had suffered from colic less than 8 hours, and 7,5% for longer than 36 hours.

No significant difference was found in the duration of anaesthesia between the horses with diarrhoea and those without ($\bar{x} = 88,59$ minutes; SD = 27,07; lg $\bar{x} = 4,44$; SD = 0,33; n = 39). Nor did there appear to be a correlation between the type of surgical therapy, according to the division used, and post-operative diarrhoea. Intestinal resectioning was carried out in 10 cases (23,8%; n = 42) while the other operations involved repositioning.

The duration of neostigmine administration in the diarrhoeic cases was found to be similar to the duration

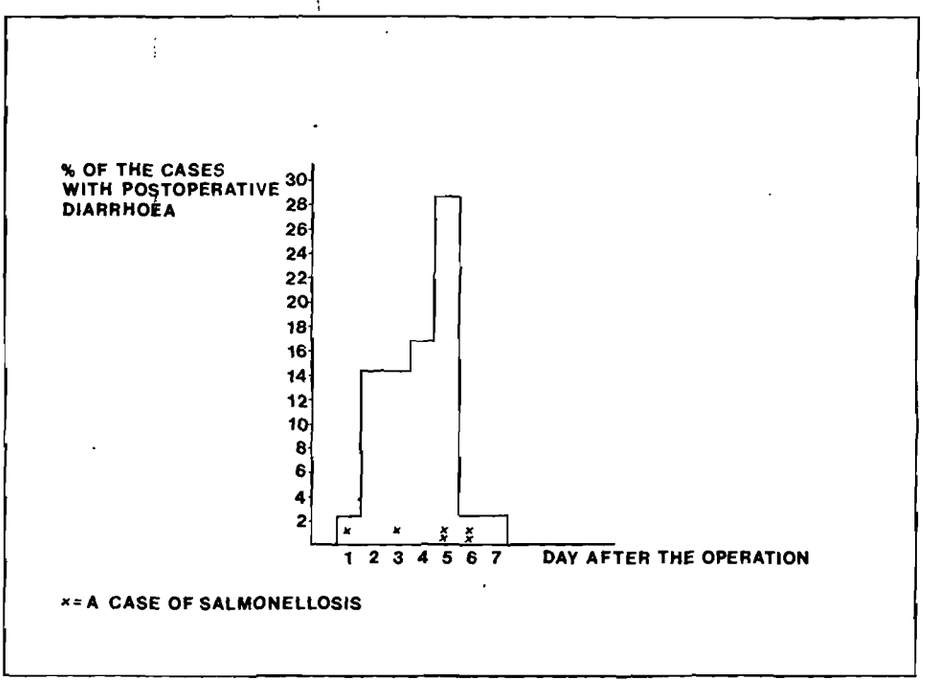

Fig. 1: The onset of diarrhoea
x = a *Salmonella* positive case

of administration in the cases which did not contract diarrhoea ($\bar{x}=2,44$ days; SD = 2,53; lg $\bar{x}=0,60$; SD = 0,69; n = 41). No relationship was found between the duration of neostigmine administration and the time of onset of diarrhoea.

The incidence of complications other than diarrhoea was significantly higher in the diarrhoeic group than in the non-diarrhoeic group (chi-square test of independence, $X^2=12,83$, df = 1, $p<0,001$); recovery from surgery was complicated in 15/42 cases (35,7%). One horse developed aspiration pneumonia following pre-operative aspiration of gastrointestinal fluid. In two cases bronchitis developed after surgery. Three mares aborted; two of them following surgery, and one on the 16th day after the operation. Three horses had undergone two laparotomies prior to the onset of diarrhoea. One horse was re-submitted for surgery when already diarrhoeic, and three after the period of diarrhoea because of complications. Two more patients developed peritonitis following surgery.

The time of onset of signs of diarrhoea following surgery is shown in Fig. 1; 39 horses (92,9%; n = 42) developed diarrhoea between the second and sixth day after surgery ($\bar{x}=4,33$ days; SD = 1,59; lg $\bar{x}=1,38$; SD = 0,44), and 39 horses (92,9%; n = 42) recovered from diarrhoea within one week (duration of diarrhoea: $\bar{x}=5,05$ days; SD = 5,20; lg $\bar{x}=1,33$; SD = 0,75).

In 16 cases (38,1%; n = 42 including 4 *Salmonella* positive cases), no temperature increase was observed during the period of diarrhoea. Rectal temperatures of 39,0 – 39,4°C were measured in 21 cases (50,0%; one *Salmonella* positive case), 39,5 – 39,9°C in 2 cases (4,8%), and 40,0 – 40,5°C in 3 cases (7,1%; one *Salmonella* positive case).

Leukopaenia was observed in 3 cases (8,1%; n = 37), leukocytosis in 21 cases (56,8%; 3 *Salmonella* positive cases), and leukopaenia-leukocytosis in 3 cases (8,1%; one *Salmonella* positive case). In the other 10 cases examined (27,0%; two *Salmonella* positive cases), no change from the normal total blood leukocyte count was determined. In 13 of the 21 cases with leukocytosis, a total blood leukocyte count of over 15 000 × $10^9/\ell$ was determined.

Table 2: Antimicrobials in the treatment of acute postoperative diarrhoea

Antimicrobial; preparation and daily doses/500 kg body mass	Horses: Total n	*Salmonella* positive n	Non-survived n
Procain-penicillin-G and dihydrostreptomycin (Strepto-Penicillin 45%, Meca: 2 × 20 ml)	11	2	3
Sulfadoxin and trimethoprim (Duoprim, Wellcome: 2 × 20 ml)	5	—	2
Chloramphenical (Chloramphenicol, Byk-Gulden-Lomberg: 2 × 30 ml)	1	—	1
Gentamicin (Sanicalv, Virbac: 2 × 50 ml*)	13	4	2
Total	30	6	8

*Initial dose was a double daily dose

Faecal bacteriological examination was performed in 37 of the 42 diarrhoeic cases; of the 5 cases from which no faecal culture was made, 3 suffered from diarrhoea for only one day, and 2 recovered in two days. None of these 5 cases had severe diarrhoea (fulminating diarrhoea, marked dehydration or acid-base disturbance). *Salmonella* spp. were isolated in 6 cases (16,2%); *Salmonella typhimurium* in 2 cases (5,4%). In one of the cases with *Salmonella* infection, *Aeromonas* spp. were also found in the faecal culture. In the other 31

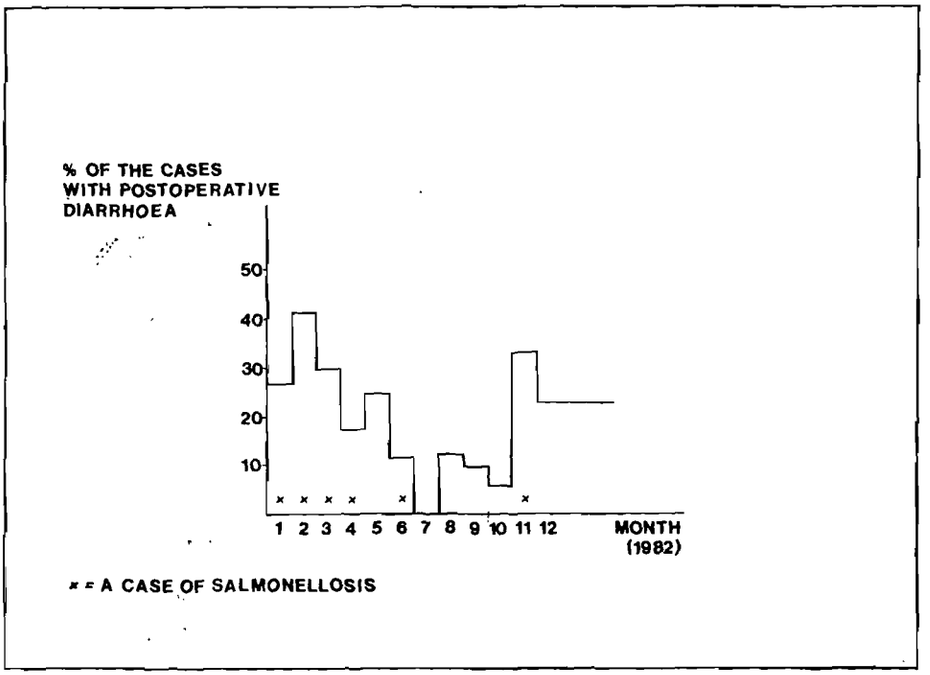

Fig. 2: Seasonal distribution of acute post-operative diarrhoea
x = a *Salmonella* positive case

cases (83,8%), bacteriological cultures were negative for pathogenic bacteria.

A massive *Strongylus* sp. infestation was found in one of the 42 cases on faecal parasitological examination.

Of the diarrhoeic horses, 12 were treated only symptomatically, being dosed with kaolin and pectin orally and fluids intravenously, without parenteral antimicrobic therapy. The antimicrobials used in the 30 cases are shown in Table 2.

Thirty-two horses (76,2%; n = 42) survived; of the 10 non-survivors, 2 died primarily because of diarrhoea; both of them were positive for *Salmonella*. The others had other complications and four of them had already recovered from diarrhoea (Table 3).

The seasonal distribution of the outbreaks is shown in Fig. 2.

DISCUSSION

Of the surgical colic patients in the present study, 19,4% developed diarrhoea post-operatively. A frequency of acute colitis of 2,3% was observed earlier after any surgical procedure[27]. In contrast, some reviewed surveys of surgical treatment of colic do not mention acute diarrhoea as a post-operative complication[12 20 32 54].

The outbreak of acute post-operative diarrhoea was not found to be dependent on breed, sex, or age; the distributions being about the same among the other surgical colic patients in this study.

From the findings of the present study, it also seems likely that neither the part of the gastrointestinal tract involved nor the diagnosis of colic has a bearing on acute post-operative diarrhoea.

Duration of anaesthesia, type of surgical therapy, and duration of neostigmine administration did not prove to be predisposing factors to post-operative diarrhoea.

A significant difference in duration of colic signs was found between the cases which contracted acute post-operative diarrhoea and the other surgical colic cases in this study. The findings suggest that gastrointestinal

Table 3: Observed causes of death in the non-survived cases of post-operative diarrhoea

Cause of death	Duration of diarrhoea (days after operation)	Time of death (day after operation)	Notes
Horses recovered from diarrhoea:			
Cardiac arrest	2 – 10	14	during 2nd op.
Massive intra-abdominal adherences	4 – 6	11	during 2nd op., E
Peritonitis	7 – 11	21	E
Peritonitis	5 – 9	15	during 3rd op., E
Horses suffering from diarrhoea:			
Salmonellosis (*S. typhimurium*)	1 – 5	5	E
Salmonellosis	5 – 37	37	
Enteritis and pneumonia	4 – 7	7	
Enteritis and peritonitis	3 – 8	8	
Peritonitis	2 – 16	16	during 2nd op., E
Peritonitis	4 – 8	8	during 2nd op., E

op. = operation
E = euthanasia

disorders of longer duration predispose to post-operative diarrhoea. This could probably be due to a longer period of stress and to intestinal changes altering microflora and disturbing mucosal defence mechanisms[19].

In 35,7% of the cases of acute post-operative diarrhoea, recovery from surgery was complicated by factors other than diarrhoea. The number of factors was significantly lower in the other surgical cases in the present study. Post-operative complications could be considered as an additional stress factor predisposing to post-operative diarrhoea.

Post-operatively, 92,9% (39/42) of the cases developed diarrhoea between the 2nd and the 6th day, similar to reports of the onset of diarrhoea after stress situations in previous studies[11 24 44].

The mean duration of diarrhoea was 5 days, which is markedly shorter than that observed from the duration of colitis in a previous study[11].

Fever is considered to be characteristic of acute colitis[27] and of enteric salmonellosis[11 25] in the horse. However, rectal temperatures of 39,0°C or more were recorded in only 61,9% (26/42) of the cases of acute post-operative diarrhoea in this study. Rectal temperatures of 40,0°C or more were recorded in 3 cases, one of which was a case of *S. typhimurium* infection.

Subnormal total blood leukocyte counts were encountered in 16,2% (6/37) of the cases examined. This is not in agreement with an earlier study which found 88,9% (16/18) of the horses suffering from acute colitis to be leukopaenic[27]. Leukocytosis, without preceding leukopaenia, was determined in 56,8% (21/37) of the diarrhoeic cases. In 27,0% (10/37) of the cases, no pathological changes in total blood leukocyte count were observed. The horse has a relatively small leukocyte reserve[22 42], which allows a decrease to happen easily. Neutropaenia, often followed by leukopaenia, is reported to accompany especially equine enteric salmonellosis[31 43], but is not present in every case[11 33]. However, only one of the 6 *Salmonella* positive horses in the present study demonstrated leukopaenia.

Salmonella spp. were isolated in 16,2% (6/37) of the cases of acute post-operative diarrhoea of the present study, which is markedly less than in the earlier studies on diarrhoea following hospitalization[11]. However, it must be remembered that horses shedding *Salmonella* spp. can repeatedly yield negative faecal cultures[46]. A previous study furthermore reported that 80% of the samples taken while the horse known to be a shedder had diarrhoea, were positive for *Salmonella*, while otherwise the number of cases without diarrhoea was only 36%[44]. Horses are very sensitive to *Salmonella* infections[10], and salmonellosis is undoubtedly one of the commonest causes of infectious diarrhoea in the horse[1 17 21 25 51]. Surgery[16 29], especially abdominal surgery[1 30 41 44] is reported as one of the precipitating factors of enteric salmonellosis. However, any factor which alter the host's defence predisposes to salmonellosis[53]. Surgical colic patients especially are subjected to several predisposing factors including gastrointestinal changes. Marked alteration of gastrointestinal microflora and increases in *Salmonella typhimurium* population in mice is reported to occur 48 hours after deprivation of food, water, and bedding[49]. Similar changes are believed to occur in the large bowel of the stressed horse[19]. In these cases of acute abdominal disorder, the microbiological function and chemical barriers of the intestine are disturbed[28]. Occasionally, salmonellosis is also discovered as a concurrent problem in cases of colic[8]. Salmonellosis is rarely a simple cause/effect disease, but it is influenced by both extrinsic and intrinsic factors[16]. *S. typhimurium* is the most common *Salmonella* strain in horses in West Germany[34–37] and it is also reported to be the most common *Salmonella* strain in infectious diarrhoea in horses[11 14 21 53]; it was isolated in 2 of the 6 cases of salmonellosis in the present study. *S. typhimurium* is not host-specific and is found as a contaminant in the environment[46].

Birds in the wild[18], and especially sparrows[39 52], are often observed to be carriers. Contamination of food in broiler production has also been reported[40]. However, in the cases in the present study, different strains of *Salmonella* were isolated, as well as in the other cases of enteric salmonellosis treated in the hospital, and therefore it seems more likely that stress factors were responsible for the outbreaks of diarrhoea rather than hospital contamination. The horse handlers themselves were not checked as shedders of *Salmonella*, but none of the hospital staff showed any positive signs at the checks provided.

In one of the diarrhoeic cases of the present study, *S. typhimurium* was isolated from a faecal sample on the first day after surgery, and thus the horse must already have been affected at arrival; the operation was performed 3 hours after arrival at the hospital. However, development of fever, depression, anorexia, and haematologic changes within 24 hours after oral exposure with a large number of virulent *S. typhimurium* has been reported[45].

A relationship between clinical salmonellosis and parasitic infestation is observed[21]. However, in only one case in the present study a massive *Strongylus* sp. invasion was found on faecal parasitological examination; the case was *Salmonella* negative.

High rectal temperature, depression, anorexia, leukopaenia, cardiovascular changes suggesting endotoxaemia[53], and evidence of systemic spread of infection[7] have been reported as criteria for instituting vigorous parenteral antibiotic therapy in cases of enteric salmonellosis. All the cases of enteric salmonellosis and most of the other cases of acute post-operative diarrhoea in the present study exhibited signs reported to accompany enteric salmonellosis or acute enterocolitis, such as anorexia[44 46] signs of dehydration and acid-base disturbances[25 50 51], and fulminating diarrhoea[4 53]. Endotoxins produced by *Salmonella*[47] or *Escherichia coli* bacteria[27] are suggested as the cause for the pathologic and clinical alterations that develop in infected animals. Endotoxaemia, anaphylaxis, and shock are also found often to accompany acute diarrhoea[24]. In the present study, 2 of the 6 *Salmonella* positive horses developed a severe endotoxaemia, as shown by the post mortem findings, and died in spite of intense therapy; autopsy revealed colitis and typhlitis, typical of *Salmonella* infection[4 21 44].

Although the majority of salmonellae isolated are sensitive to broad spectrum antibiotics and chemotherapeutics[21], a number of resistant strains have been recorded[6 56]. On the contrary, increased susceptibility to diarrhoea associated with enteric salmonellosis has been reported in horses treated parenterally with tetracycline[2 3 7 9 30]; other antibiotics

have also been observed to produce diarrhoea in horses[7,44]. The parenteral antibiotic therapy used during and after surgery in the present study failed to reveal any relationship between the outbreaks of acute post-operative diarrhoea. In this study, 30 of the 42 horses were treated with systemic antibiotics or chemotherapeutics for enteritis. Similar to previous reports[21,44], no obvious difference in recovery was revealed between the substances used. The findings of the present study agree with previous surveys[7,21,30,44,53] and suggest that much of the success in treatment of acute diarrhoea, independent of the cause, is associated with supportive therapy to control diarrhoea, dehydration, and shock.

In the present study, 76,2% (32/42) of the cases with acute post-operative diarrhoea survived. It has been reported that 55,6% of the cases of acute post-operative diarrhoea survived[77]. Yet, some of the horses in the present study recovered from diarrhoea in 2 days and did not exhibit the severe signs of dehydration and acid-base disturbances characteristic of acute colitis[51]. From the 6 cases in the present study in which *Salmonella* was isolated from faecal culture, 2 (33,3%) died. An earlier report disclosed 71,4% (5/7) deaths in cases of enteric *S. typhimurium* infection[17], whereas another study reported a figure of 60,0% (3/5)[44]. However, the number of isolated salmonellae in the present study was too small to draw any further conclusions on survival.

The incidence of outbreaks of acute post-operative diarrhoea was highest in winter and spring and lowest in summer in the cases in this study; just the opposite to other findings which observed the highest frequencies in June[17], in late summer[44], or in October-November[11].

A surgical colic case often includes all the factors reported to predispose the horse to acute diarrhoea. From the variables examined in this study, the duration of colic signs revealed a significant difference between the horses which contracted post-operative diarrhoea and the other surgical colic cases. To the predisposing factors could also be added complications in recovery from surgery; the percentage was significantly higher in the case of acute post-operative diarrhoea than in the other surgical cases of colic.

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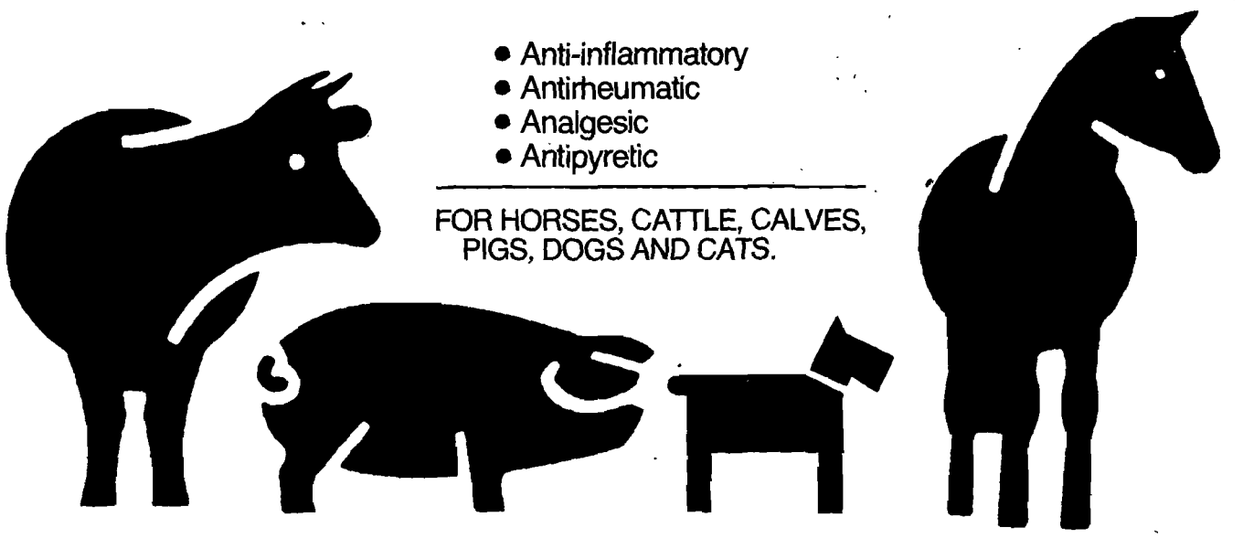

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ARTICLE ARTIKEL

ARTERIAL BLOOD GAS COMPOSITION, CONSCIOUSNESS AND DEATH IN RABBITS

J. HATTINGH, S.T. CORNELIUS, M.F. GANHAO and F. FONSECA*

ABSTRACT: Hattingh J.; Cornelius S.T.; Ganhao M.F.; Fonseca F. **Arterial blood gas composition, consciousness and death in rabbits.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 13-16 (En). Department of General Physiology, University of the Witwatersrand, 1 Jan Smuts Avenue, 2001 Johannesburg, Republic of South Africa.

Conscious rabbits were exposed to environments with low oxygen and/or high carbon dioxide. The electroencephalogram and arterial blood pressure were recorded. Arterial blood samples were taken sequentially. Rabbits became 'unconscious' at an arterial blood P_{O_2} of 25 ± 1 mm Hg and P_{CO_2} of 99 ± 27 mm Hg, when simultaneously the environmental oxygen was decreased and carbon dioxide increased. Brain death occurred at a P_{O_2} of 23 ± 1 mm Hg and P_{CO_2} of 113 ± 32 mm Hg. Cerebral perfusion was still adequate. In rabbits, the margin between 'unconsciousness' and 'death' is narrow so far as P_{O_2} is concerned. If these results apply generally, animals culled with muscle relaxants may perceive stress involved until shortly before death.

Key words: Blood gases, consciousness, death, culling.

INTRODUCTION

In certain game parks and elsewhere, succinyldicholine (Scoline) is used to cull animals[8,10]. Recently, the blood composition of buffaloes and elephants culled in this fashion was compared to that of control animals (shot in the brain at short range) and the differences observed were attributed to stress induced by a combination of herding and asphyxia[8]. Elephants collapsed within 4 to 10 minutes and buffaloes within 1½ to 5 minutes after being darted with scoline. Some of the elephants were still alive after 25 minutes, but the buffaloes were all dead (no detectable respiration or pulse) after 12 minutes. Elephants may therefore perceive the stress associated with the culling procedure for a longer period of time than buffaloes. Death by asphyxiation results from scoline overdosage. It is therefore important to establish when consciousness is lost so as to limit the conscious perception of stress.

Investigations have shown that the electroencephalogram (EEG) is iso-electric in anaesthetized elephants, buffaloes, impala and rabbits at arterial blood oxygen tension corresponding to approximately 25% saturation of haemoglobin[9]. Comparison of such results with blood gas measurements from culled animals suggests that they may still be able to perceive stress for some time after they collapse.

In the literature there is little information regarding the point at which arterial blood gas tensions cause consciousness to be lost. In man, this seems to occur when haemoglobin is between 40% and 60% saturated with oxygen[5,7,11]. Studies by Christensen & Krogh[2], Davis et al.[3,5], Opitz[12] and Rebuck et al.[14] indicate that loss of consciousness occurs when alveolar P_{O_2} tensions are between 28 and 32 mm Hg and demonstrate little individual variation. More profound levels of hypoxia may be tolerated with higher P_{CO_2} tensions than with normal or lower carbon dioxide tensions before cerebral function deteriorates. Sieker & Hickam[15] indicate that consciousness is lost at P_{CO_2} tensions above 130 mm Hg. Hattingh et al.[9] imply that the margin between unconsciousness and death is narrow in rabbits. In this study arterial blood gas tensions, heart rate, arterial pressure, electroencephalogram and plasma glucose, lactate and cortisol values are measured in rabbits under hypoxic and hypercapnic conditions. After careful consideration these experiments were agreed-to by the Animal Ethics Committee of the University.

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MATERIALS AND METHODS

Rabbits accustomed to handling and to stocks, were anaesthetized with alphaxolone-alphadolone intravenously (6–9 mg/kg; Saffan, Glaxo). Three screw electrodes were inserted aseptically into the skull, just penetrating the cranial cavity, one on each side midway between the eyes and the ears and the third midway between the eyes as described previously[9].

Before waking from anaesthesia the animals were given dihydrostreptomycin/procaine penicillin intramuscularly (125/600 mg; Milvet) and pethidine hydrochloride subcutaneously (10 mg/kg; Roche). The animals were allowed to recover for a period of 2 to 3 days before being used in experimentation. A central ear artery was cannulated with a 23 G butterfly needle under local anaesthesia (lidocaine, Xylonor-spray) on the day of the experiment. The animal was then placed in a closed, perspex chamber (volume about 120 ℓ). Blood pressure was recorded from the central ear artery by a pressure transducer (Bell and Howell) and 3559 transducer amplifier (Electromed). Blood was sampled from the same artery. Electroencephalographic recordings were made from the screw electrodes using a 3543 A.C. amplifier (Electromed). A MX 216 recorder (Electromed) was used for recording. Oxygen and carbon dioxide percentages in the chamber were measured with a S3A Oxygen analyser (Applied Electrochemistry Inc.) and a Cavitron PM-20 Neonatal CO_2 Monitor (Anarad Inc.). The percentage composition of air in the chamber was altered by infusing medical grade oxygen, carbon dioxide and/or nitrogen from gas cylinders.

Three groups of experiments were performed. The first (Group 1, N=8) involved decreasing the percentage of oxygen in the container by infusing nitrogen whilst the percentage of carbon dioxide was maintained at 0,1%. In the second (Group 2, N=6) the percentage of carbon dioxide was increased whilst the percentage of

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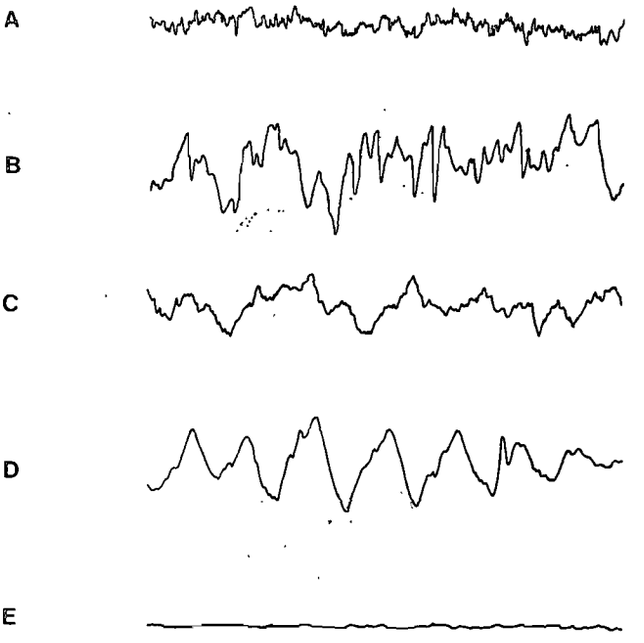

Fig. 1: Examples of EEG recordings from A = conscious, B = anaesthetized, C = hypocapnic, D = unconscious (comatose) and E = "dead" (iso-electric) rabbits.

oxygen was maintained at 21%. In the third (Group 3, N = 5) the percentage of oxygen was decreased simultaneously with an increase in the percentage of carbon dioxide. The chamber gas pressure remained atmospheric in all experiments.

All animals were left to stabilise for 30 minutes at 21,0% O_2, 0,1% CO_2 and about 79% N_2. The percentage composition of air in the container was then changed as indicated above. The percentage of oxygen was decreased to 3,5% and/or the percentage of carbon dioxide was increased to 50,0% over a period of 20 to 30 minutes. Blood pressure and EEG recorded continuously, while blood was sampled at regular intervals.

Blood P_{O_2}, P_{CO_2} and pH were measured immediately using a PHM 71 analyser and blood micro systems BMS 3 MK2 (Radiometer). Plasma was analysed as soon as possible for glucose (GOD-Period colorimetric method, Boehringer Mannheim) lactate (enzymatic UV-method, Boehringer Mannheim) and cortisol (solid phase radioimmunoassay, Diagnostic Product Corp.).

RESULTS

Control values obtained from the rabbits after being in the chamber for about 30 minutes are shown in Table 1. The 'conscious' EEG pattern is shown in Fig. 1.

Group 1

Both arterial blood P_{O_2} and P_{CO_2} decreased, the latter as a result of hyperventilation (Table 2), when the oxygen percentage in the chamber was decreased at a constant carbon dioxide percentage. The resulting 'hypocapnic EEG' recordings were difficult to distinguish from the unconscious pattern (Fig. 1). The 'unconscious' state was identified by observing a lack of response to stimuli (visual and ear pinching with the animals still in the chambers and the EEG). Rabbits were declared 'dead' when the EEG was iso-electric (Fig. 1).

Table 1: Control values (means ± S.D.) for all three experimental groups

Variable	Value
Arterial blood P_{O_2}	74 ± 8 mm Hg
Arterial blood P_{CO_2}	20 ± 5 mm Hg
Arterial blood pH	7,492 ± 0,094
Heart rate	249 ± 38 b/min
Systolic pressure	85 ± 15 mm Hg
Diastolic pressure	69 ± 11 mm Hg
Plasma glucose	7,26 ± 2,00 mmol/ℓ
Plasma lactate	1,79 ± 1,01 mmol/ℓ
Plasma cortisol	49 ± 23 nmol/ℓ

Table 2: Values of variables for Group 1 (low oxygen, normal carbon dioxide) animals when 'unconscious' and 'dead' (Means ± S.D.)

Variable	'Unconscious'	'Dead'
Arterial blood P_{O_2} mm Hg	23 ± 2*	19 ± 3*
Arterial blood P_{CO_2} mm Hg	9 ± 3*	10 ± 6*
Arterial blood pH	7,480 ± 0,170	7,390 ± 0,293
Heart rate b/m	195 ± 90	234 ± 70
Systolic pressure mm Hg	132 ± 26*	147 ± 40*
Plasma glucose mmol/ℓ	12,00 ± 2,44*	11,40 ± 2,79*
Plasma lactate mmol/ℓ	10,83 ± 7,08*	13,17 ± 5,73*
Plasma cortisol nmol/ℓ	82 ± 7*	77 ± 5,73

*Indicates significantly different from control values (P < 0,01).

Table 3: Values of variables for Group 2 (normal oxygen, high carbon dioxide) animals when 'unconscious' and 'dead' (Means ± S.D.)

Variable	'Unconscious'	'Dead'
Arterial blood P_{O_2} mm Hg	86 ± 18	39 ± 14*
Arterial blood P_{CO_2} mm Hg	131 ± 12*	>200*
Arterial blood pH	6,893 ± 0,090*	6,623 ± 0,614*
Heart rate b/m	160 ± 51*	182 ± 97
Systolic pressure mm Hg	125 ± 20*	87 ± 25
Plasma glucose mmol/ℓ	10,40 ± 1,60*	14,85 ± 2,35*
Plasma lactate mmol/ℓ	2,14 ± 0,91*	6,44 ± 2,64*
Plasma cortisol nmol/ℓ	63 ± 19	51 ± 8

*Indicates significantly different from control values (P < 0,01).

The results obtained when the animals were 'unconscious' and when they 'died' are shown in Table 2. Arterial blood P_{O_2} and P_{CO_2} values differed significantly from control values (P < 0,01), but those for pH did not. Large individual variation is indicated by the standard deviations. Systolic blood pressure increased significantly (P < 0,01) compared with control values while diastolic pressure showed no change in the two states. The increase in systolic pressure was only evident (graphical analysis) when P_{O_2} decreased below 35 mm Hg.

Plasma glucose, cortisol and lactate values increased significantly compared with control values, the latter doing so at arterial blood P_{O_2} values less than 30 mm Hg. No statistically significant differences existed for any of the variables between the 'unconscious' and 'dead' states.

Group 2

The animals became 'unconscious' at a mean arterial blood P_{CO_2} value of 131 mm Hg when the carbon dioxide percentage was increased in the chamber and the oxygen percentage remained constant (Table 3). The EEG recordings showed repeated iso-electric periods of a few seconds duration followed by delta-waves until the P_{CO_2} was above 200 mm Hg, when all activity ceased. Arterial blood P_{O_2} remained constant until P_{CO_2} values were above 150 mm Hg and then decreased. Excepting for blood gas tensions and systolic pressure, there were no significant differences in other variables between the 'unconscious' and 'dead' states. Compared with control values, arterial blood pressure increased when the animals were unconscious but decreased at 'death'. Neither diastolic blood pressure nor plasma cortisol levels showed a significant change, but plasma glucose and lactate concentrations increased ($P<0,01$).

Table 4: Values of variables for Group 3 (low oxygen, high carbon dioxide) animals when 'unconscious' and 'dead' (Means ± S.D.)

Variable	'Unconscious'	'Dead'
Arterial blood P_{O_2} mm Hg	25 ± 1*	23 ± 1*
Arterial blood P_{CO_2} mm Hg	99 ± 27*	113 ± 32*
Arterial blood pH	7,021 ± 0,055*	6,925 ± 0,056*
Heart rate b/m	118 ± 37*	129 ± 18*
Systolic pressure mm Hg	110 ± 14	104 ± 29
Plasma glucose mmol/ℓ	9,07 ± 1,32	8,07 ± 1,35
Plasma lactate mmol/ℓ	7,46 ± 1,17*	7,62 ± 0,75*
Plasma cortisol nmol/ℓ	53 ± 35	42 ± 36

*Indicates significantly different from control values ($P<0,01$).

Group 3

When the chamber oxygen percentage was decreased and the carbon dioxide percentage was simultaneously increased the arterial blood P_{CO_2} remained constant until the P_{O_2} decreased to about 30 mm Hg, at which point it increased rapidly (Table 4). A similar pattern was observed for arterial blood pH which decreased only when the P_{O_2} was below 35 mm Hg. Heart rate decreased significantly below arterial P_{O_2} values of 40 mm Hg and P_{CO_2} values of 30 mm Hg. Systolic blood pressure changes were not significant. Plasma glucose and cortisol concentrations and diastolic blood pressure did not change significantly. Plasma lactate levels increased significantly when arterial P_{O_2} values were below 30 mm Hg. No significant differences were observed amongst the variables in the 'unconscious' and 'dead' states. The EEG showed similar changes as seen in the other groups, i.e. changing to delta-waves at unconscious and then becoming iso-electric.

For animals in Groups 1 and 3 the arterial P_{O_2} values were not significantly different during the 'unconscious' and 'dead' states but were significantly higher ($P<0,01$) in Group 2. Arterial P_{CO_2} values did not differ significantly between 2 and 3 but were significantly lower ($P<0,01$) in Group 1.

DISCUSSION

Extensive knowledge exists concerning the EEG and brain ischaemia due to hypotension[13]. The EEG is iso-electric within 20s of cessation of the circulation. It may return to normal within 30s if the ischaemia is of short duration[13]. Less information is available on arterial blood gas tensions at which unconsciousness and death occurs when brain perfusion is still adequate. West et al.[16] reported that exercise could be performed on the top of Mount Everest at alveolar P_{O_2} values of about 28 mm Hg in man. Brierly et al.[1] found that irreversible brain damage occurred when arterial P_{O_2} was between 21 and 24 mm Hg for at least 8 min in primates. It would thus seem that consciousness may be lost below 28 mm Hg arterial P_{O_2} and that death may ensue at 20 mm Hg or less. This was confirmed in rabbits by the present study.

Experiments were designed to simulate the culling procedure in a controlled manner. Environmental percentages of oxygen and/or carbon dioxide were changed over a period of 20 to 30 minutes to beyond the limits of survival. Exact determination of the moment of 'unconsciousness' or 'death' is not possible. It can be stated with certainty that the rabbits responded to visual and other stimuli until the time when delta-wave activity was observed consistantly in the EEG recording. The unconscious state was thus a subjective rather than objective observation. 'Death' was taken as that point when the EEG first showed a constant iso-electric recording. At both 'death' and 'unconsciousness' brain perfusion was still adequate in all three groups, as is clear from the heart rate and systolic and distolic pressure values[6].

The following must be considered if these results on rabbits are generally applicable, which may not be the case[13]: Under conditions where there is a rapid decrease in environmental oxygen concentration or when arterial blood P_{O_2} decreases quickly for whatever reason, variation in P_{O_2} between 'unconscious' and 'death' is narrow in both hypo- and hypercapnic states[9]. The reverse is true for hypercapnia at normoxia where presumably a stage of anaesthesia is involved between arterial blood P_{CO_2} values of about 130 mm Hg and 200 mm Hg[15] but the same applies for P_{CO_2} at hypoxia. It is possible that the P_{O_2} is more important in maintaining consciousness and in causing death, than is P_{CO_2} i.e. P_{O_2} is more critical because of the consistency of the results obtained for P_{O_2} at 'unconsciousness' and 'death' for Groups 1 and 3 and the larger variability in the P_{CO_2} for Groups 2 and 3. However, this suggestion requires further experimentation. From results obtained on culled elephants and buffaloes[8], it is apparent that they die because of the low arterial P_{O_2} and not the elevated P_{CO_2}. Furthermore they would remain conscious for some time after collapse; longer for elephants than for buffaloes. They would only become unconscious just before they died and thus may be able to perceive the stresses involved in culling for periods up to 25 minutes or until they are shot.

Freed[4], discussing carbon dioxide euthanasia, states that oxygen deprivation (in humans) causes no subjective suffering and has few physiological consequences largely because the subject becomes quickly unconscious. He suggests that oxygen deprivation should be the method of choice for animal euthanasia and not the use of carbon dioxide. The present results show that differences exist in the values of the variables measured when animals were killed with low oxygen, increased carbon dioxide or a combination of both. All animals became unconscious whilst hyperventilating severely.

This state was certainly not entered into 'peacefully' (as claimed by Freed for oxygen deprivation). It is our opinion that an effective and rapid method to kill conscious animals would be to place them in an environment with 50 to 100% carbon dioxide in nitrogen.

ACKNOWLEDGEMENTS

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ARTICLE ARTIKEL

OBSERVATIONS ON THE PATHOLOGY OF EXPERIMENTAL ENCEPHALITOZOONOSIS IN DOGS

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ABSTRACT: Botha W.S.; Stewart C.G.; Van Dellen A.F. **Observations on the pathology of experimental encephalitozoonosis in dogs.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 17-24 (En). Consultant Veterinary Pathologist, P.O. Box 12731, 0110 Onderstepoort, Republic of South Africa.

Experimental transmission of canine encephalitozoonosis was effected by oral dosing of urine obtained from naturally diseased animals. Per os, intraperitoneal and intravenous routes were used to induce infection with tissue culture-grown *Encephalitozoon* spores which were initially isolated from the kidney of a dog with terminal disease. The infection was confirmed by a rise in the indirect immunofluorescent antibody titres, the lesions found in infected dogs and isolation of the parasite in tissue culture from an infected and immunosuppressed dog. The experimentally induced disease was invariably subclinical but the histopathological changes were similar although milder than those found in fatal natural disease. The kidney appears to be the target organ and chronic interstitial nephritis develops regularly.

Key words: Encephalitozoonosis, *Encephalitozoon*, dog, pathology, nephritis.

INTRODUCTION

In 1952 Plowright and Yeoman[13] described what they considered to be a probable *Encephalitozoon* infection in dogs. They could, however, not transmit the disease to laboratory animals by inoculation of infected brain material which had been preserved in glycerine. Botha, Van Dellen & Stewart[2] reported the successful per os experimental transmission of encephalitozoonosis to dogs by using either minced kidney, brain or liver and spleen from naturally infected dogs. They were able to demonstrate the *Encephalitozoon* spores in the kidney of the recipient dogs histopathologically and by transmission electron microscopy. Perrin[10] experimentally induced encephalitozoonosis by different routes in laboratory mice by inoculating peritoneal exudate or suspensions of liver, brain and spleen from naturally diseased mice into healthy animals. It was also possible to transmit cell culture-grown *Encephalitozoon cuniculi* per os or intratracheally to laboratory rabbits[3].

Following the successful isolation of *Encephalitozoon* organisms in tissue culture from naturally diseased dogs[18], it became possible to induce experimental infection in dogs with pure cultures of the organisms. In the experiments described in this paper, different routes of infection and doses of *Encephalitozoon* spores were used to create clinical disease and/or pathological lesions comparable to spontaneous encephalitozoonosis.

MATERIALS AND METHODS

Infective material

Urine from naturally infected dogs where spores could be demonstrated in urine sediment smears using the Sternheimer Malbin stain[16] or tissue culture isolates of *Encephalitozoon* organisms[18] were used. The tissue cultures were lysed with distilled water for 3 min. to release spores from the infected cells. Isotonicity was restored and the spores were counted in a haemocytometer before administration.

Experimental dogs

Healthy puppies from one breeder were used. They were vaccinated against distemper, hepatitis, adenovirus type 2, parainfluenza, parvovirus and leptospirosis (Vanguard DA2PL + CPV (ML), Smithkline) at 3 months of age. The details of the experimental dogs are recorded in Table 1. Blood was collected from all dogs prior to infection and before euthanasia was performed for testing for *Encephalitozoon* antibodies by means of the indirect fluorescent antibody (IFA) test[17]. At termination of the experiments, all dogs were killed by an overdose of intravenous pentobarbitone sodium (Euthanaze, Centaur) and a necropsy was performed on each animal. Specimens from all experimental animals were preserved in 10% formalin for histopathological evaluation. Paraffin-embedded sections were cut at a thickness of 6 μm and stained with haematoxylin and eosin (HE). Selected sections were stained with Gram's (Brown-Hopps modification), Masson's trichrome and Giemsa stains[4].

Experiment 1 – Infective urine

Two healthy Alsatians of 3 and 5 months of age, respectively, were dosed by stomach tube daily for 3 consecutive days with 10 mℓ infected urine. Encephalitozoonosis was confirmed in the donors but the concentration of spores in the urine was not determined.

Experiment 2 – Comparison of intravenous and per os infection

Two routes of experimental infection were studied in 3 healthy, 7 to 8-week-old Fox Terrier puppies. Dog 7 was injected intravenously with 114 × 10^6 *Encephalitozoon* spores suspended in 5 mℓ isotonic saline. Dogs 8 and 9 received 56 × 10^6 and 12,5 × 10^6 *Encephalitozoon* spores, respectively, by stomach tube. The animals were observed daily for signs of disease and killed at 55 and 61 days, respectively, after initial infection.

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Experiment 3 – Intraperitoneal infection

Four 7-week-old healthy littermate Alsatians were injected intraperitoneally with tissue culture-propagated *Encephalitozoon* spores. Increasing numbers of spores were used, viz. $12,5 \times 10^6$ up to 80×10^6 in the different dogs as indicated in Table 3. The dogs were observed daily for clinical signs of disease and killed at 90 to 100 days post-inoculation.

Experiment 4 – Per os infection and immunosuppression

Three litters of Alsatian puppies (n = 12) of about the same age (3 – 4 weeks) were selected for oral dosing with tissue culture-grown organisms. The experimental design is given in Table 4. Two of the three control dogs (47C & 49C) were killed and examined macroscopically as well as histologically for the presence of lesions consistent with those of encephalitozoonosis. The third control animal (63C) and two other experimental animals (61 & 62), were injected intramuscularly with 20 mg/kg methylprednisolone acetate (Depo-medrol V, Upjohn (Pty) Ltd.) from the day of infection, twice weekly, for 5 weeks in an attempt to induce immunosuppression. Different numbers of *Encephalitozoon* spores were used for oral dosing in this experiment while the control dog was dosed with 5 mℓ of tissue culture fluid without spores. The dogs were examined daily for signs of clinical disease. At varying intervals (Table 4) all the dogs were killed and kidney specimens were collected under sterile conditions from Dogs 61 and 63C in an attempt to isolate the organism in tissue culture 50 days after initial infection[18].

Table 1: Details of dogs used in the experiments

Experiment	Dog	Age (Weeks)	Breed	Sex	Procedure
1	37	12	A	F	PO*
	38	20	A	F	PO*
2	7	8	FT	M	IV
	8	7	FT	M	PO
	9	7	FT	F	PO
3	22	7	A	M	IP
	23	7	A	F	IP
	24	7	A	F	IP
	25	7	A	M	IP
4	41	3	A	F	PO
	42	3	A	F	PO
	43	3	A	F	PO
	44	3	A	F	PO
	45	3	A	M	PO
	46	3	A	M	PO
	48	3	A	M	PO
	61	4	A	M	PO**
	62	4	A	F	PO**
	47C	3	A	F	C
	49C	3	A	F	C
	63C	4	A	F	C**

A Alsatian
FT Fox Terrier
F Female
M Male
* Urine dosing
** Methylprednisolone-immunosuppressed
IV Intravenous
PO Per os
IP Intraperitoneal
C Control

RESULTS

Clinical disease was not observed in any of the experimentally infected dogs. Infection was, however, confirmed in all of the experimentally infected dogs by the development of antibodies to *Encephalitozoon* as determined by the IFA test. The pre-infection and post-infection IFA titres of every animal are given in Tables 2 – 4. Isolation of *Encephalitozoon* organisms in tissue culture was successful in the immunosuppressed Dog 61, 50 days after per os infection.

Experiment 1 – Infective urine

Macroscopical pathology:

Dog 38 had moderate, bilateral, focally disseminated nephritis while only a mild nephritis could be demonstrated in Dog 37. On the longitudinal cut section of the kidneys in Dog 38 a few white bands (1 mm cross section), stretching from the cortex into the medulla, were found. A few pin-point subcapsular white foci of hepatitis were present in Dog 38. In addition, multifocal subpleural and intralobular red areas of pneumonitis were seen in the lungs of Dog 38.

Microscopical pathology:

Segmental lymphocytic vasculitis was found in the cerebellar leptomeninges of Dog 38. Both animals (Dogs 37 & 38) revealed interstitial lymphocytic nephritis which was more severe in Dog 38. Vasculitis of the intralobular vessels was also present. Mild, focal microgranulomatous lesions could be demonstrated in the hepatic lobules of both animals. Multifocal interstitial pneumonitis and lymphocytic perivascular cuffing were indicative of pulmonary infection in Dog 38.

Experiment 2 – Comparison of intravenous and per os infection (Table 3)

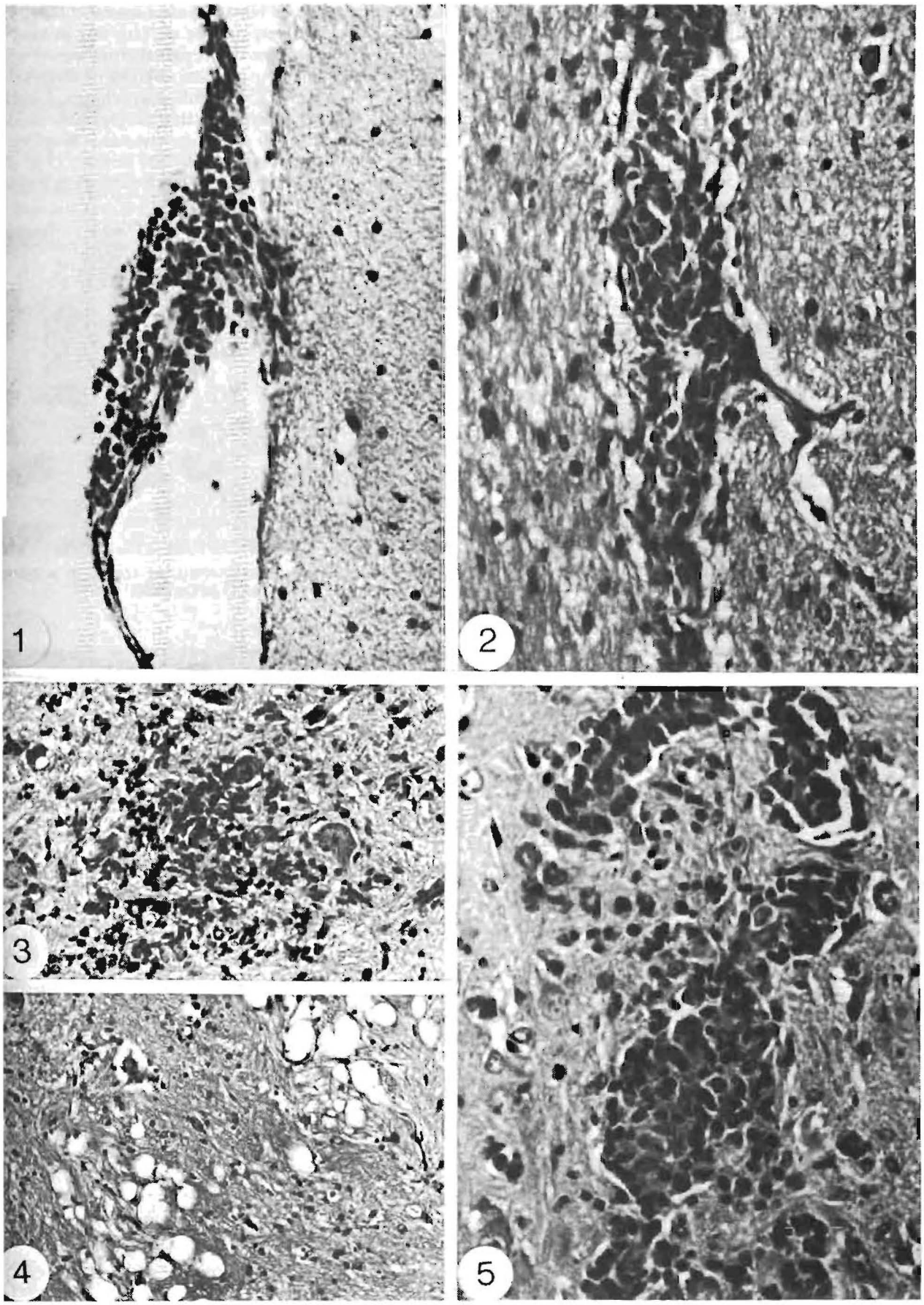
Macroscopical pathology:

A severe multifocal nephritis was found in Dog 7. The

Table 2: Experiment 1 – Oral dosing with urine from dogs with spontaneous encephalitozoonosis

Dog Number	37	38
Route of infection	PO	PO
Infective material	Urine	Urine
Interval to death (days)	52	52
Pre-infection IFA titre	–	–
Post-infection IFA titre	20	80
A. MACROSCOPICAL FINDINGS:		
Nephritis	+	+ +
Hepatitis	–	+
Pneumonitis	+	+
Lymphadenopathy	–	–
B. MICROSCOPICAL FINDINGS:		
Meningitis	–	+
Encephalitis	–	–
Nephritis	+	+ +
Hepatitis	+	+
Pneumonitis	–	+
Myocarditis	–	–
Spores demonstrated	–	–

PO Per os
– Negative or no lesions
\+ mild focal lesions – difficult to see macroscopically or occasional increased cellularity and perivascular cuffing.
\+ + moderate multifocal lesions, cuffing or dense focal inflammatory infiltrates.



ig. 1: Brain; Dog 7. Segmental vasculitis and perivascular cuffing of a vessel in the leptomeninges HE × 300
ig. 2: Cerebrum; Dog 24. Perivascular cuffing by lymphocytes in the cerebral cortex HE × 400
ig. 3: Cerebrum; Dog 9. A focal microgranuloma in the brain HE × 200
ig. 4: Cerebellar peduncle; Dog 7. Areas of status spongiosis in the white matter HE × 150
ig. 5: Brain; Dog 24. Perivascular hypertrophy of pericytes and a granulomatous perivasculitis in the brain HE × 400

kidneys were enlarged and contained numerous poorly demarcated white foci (0,5 to 1 mm in diameter) subcapsularly and on the cut surface of the kidney. Both the cortex and medulla were affected although lesions appeared slightly more extensive in the cortex. Retention cysts of up to 0,5 mm in diameter containing a clear fluid, were also present in the cortex. Only a few of these white foci were visible in the cortex of Dog 8 while Dog 9 had multiple focal lesions. Congestion surrounded many of these focal areas of nephritis. Mild enlargement of the mesenteric lymph nodes were recorded in Dog 8.

Microscopical pathology:

A mild to moderate meningitis and encephalitis were present. In each case these lesions were associated with vasculitis (Fig. 1). Some blood vessels were prominent due to swelling of endothelial cells and enlargement of pericytes (Fig. 5). Perivascular cuffing was segmental while microgranulomas were scattered throughout the brain (Fig. 3). Several sections were studied but no specific predilection site for the encephalitis could be determined. Sections from the spinal cord of Dog 9, however, showed prominent lymphocytic cuffing and microgranulomas in the grey matter while lesions were only occasionally found in the white matter. Status spongiosis was an occasional finding in the white matter of the cerebellar peduncles, midbrain and medulla oblongata (Fig. 4). A choroiditis was found in Dog 7. *Encephalitozoon* spores could not be demonstrated in sections from the brain and spinal cord.

Nephritis was severe in Dog 7 and moderate in Dogs 8 & 9. It consisted mostly of multifocal irregular areas of a perivascular and interstitial infiltration of lymphocytes and plasma cells. The kidneys of Dogs 7 & 9 contained several foci with complete tubulorhexis. The dissociated tubular epithelial cells were included in the zone of round cell infiltration. Focal areas of a mononuclear cell infiltration were most extensive at the corticomedullary junctions. They were mainly linear and parallel to the cortical rays in the cortex, and much smaller and focal in the medulla (Fig. 6). *Encephalitozoon* spores were best demonstrated with the Gram's stain (Fig. 7) and cysts were present but scarce in all the kidney sections from Dogs 7, 8 & 9.

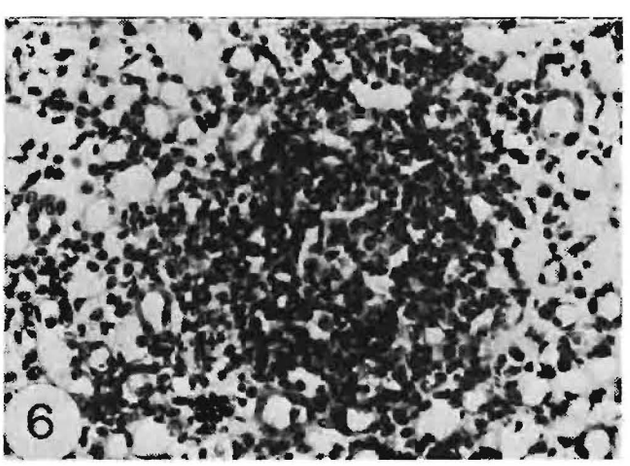


Fig. 6: Kidney; Dog 9. Focal granulomatous interstitial nephritis in the medulla HE × 200

Multifocal areas of hepatitis were found in Dogs 7 & 9, while only mild perivascular cuffing was present in the liver of Dog 8. Some of the portal blood-vessel walls were thickened due to prominent swelling of endothelial cells. In focal areas of the hepatic parenchyma, Kupffer cells and pericytes were hypertrophic (epithelioid-like) while mononuclear cell infiltrates were also involved in forming small microgranulomatous lesions (Fig. 8). These microgranulomas caused marked atrophy and oc-

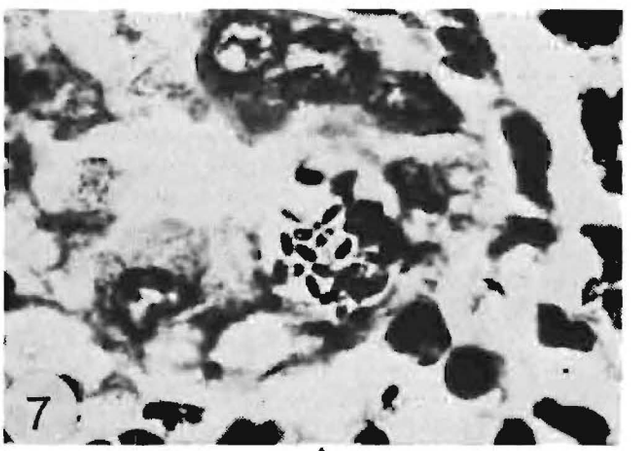


Fig. 7: Kidney; Dog 9. *Encephalitozoon* spores in a kidney tubular epithelial cell Gram × 1200

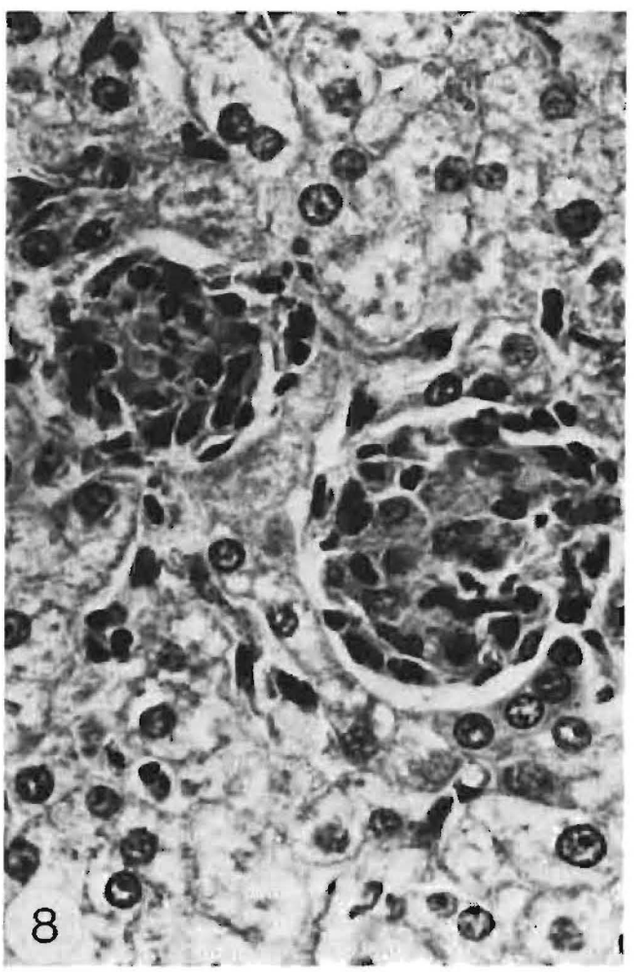


Fig. 8: Liver; Dog 7. Two microgranulomas in the liver lobule in which Kupffer cells and macrophages are hypertrophic HE × 600

Table 3: Comparison of intravenous, per os and intraperitoneal infection by tissue culture-grown *Encephalitozoon* spores

Experiment Number	2			3			
Dog Number	7	8	9	22	23	24	25
Route of inoculation	IV	PO	PO	IP	IP	IP	IP
Dose of organisms (10^6)	114	56	12,5	12,5	25	50	80
Interval to death (days)	55	60	55	90	100	100	90
Pre-infective IFA titre	–	–	–	–	10	–	–
Post-infective IFA titre	320	160	40	80	40	40	160
A. MACROSCOPICAL FINDINGS:							
Nephritis	+ + +	+	+ +	+ +	+ +	+	+
Hepatitis	–	–	–	–	+	+	–
Pneumonitis	–	–	–	–	–	–	–
Lymphadenopathy	–	+	–	+ +	+	+	+ +
B. MICROSCOPICAL FINDINGS:							
Meningitis	+ +	+	+ +	+	–	+	+
Encephalitis	+ +	+	+ +	+	+	+ +	+
Nephritis	+ + +	+ +	+ +	+	+ +	+ +	+
Hepatitis	+ +	+	+ +	+ +	+	+ +	+
Pneumonitis	+ +	+	+ +	+ +	+ +	+ + +	+ +
Myocarditis	+	+	+	+	–	+	–
Lymphoid hyperplasia	+	–	+	+ + +	+ +	+ +	+ + +
Spores demonstrated	S	S	S	–	–	–	–

PO Per Os
IV Intravenous
IP Intraperitoneal
S Scarce

\+ Mild focal lesions – difficult to see macroscopically or occasional increased cellularity and perivascular cuffing
\+ + Moderate multifocal lesions, cuffing or dense focal inflammatory infiltrates
\+ + + Severe lesions – prominent focal disseminated to extensively diffuse inflammatory reaction

casional necrosis of the hepatocytes trapped within them. *Encephalitozoon* organisms could be demonstrated in some of these lesions in Dogs 7 & 9.

The interstitial pneumonitis was not very prominent in any of these dogs but focal mononuclear cell infiltrations could be demonstrated in the alveolar walls of all 3 animals. The mild focal myocarditis in Dogs 7 & 9 was characterized by a focal interstitial mononuclear cell infiltration (Fig. 9).

Experiment 3 – Intraperitoneal infection (Table 3)

Macroscopical pathology:

Nephritis was the most prominent pathological change in all 4 dogs. The renal capsule stripped with difficulty; in some instances it caused some of the cortical tissue to be torn away. Multiple circumscribed white foci, usually less than 1 mm in diameter, were visible subcapsularly, on the cortical surface while on cut section these foci were found in both the cortex and the medulla. A few cysts were seen on the subcapsular surface of the kidney of Dog 23. Irregular, whitish, sharply demarcated foci, 1 mm in diameter, were distributed throughout the lobes of the liver of Dogs 23 & 24.

Lymphoid follicular hyperplasia occurred in all animals and consisted of enlargement of mesenteric, hepatic and submandibular lymph nodes as well as increased prominence of splenic white pulp.

Dog 25 had numerous dull red foci of about 1 to 2 mm in diameter throughout the pulmonary tissue.

Microscopical pathology:

A mild meningitis characterised by focal perivascular mononuclear infiltration was present in 3 dogs, while a mild to moderate encephalitis was present in all animals.

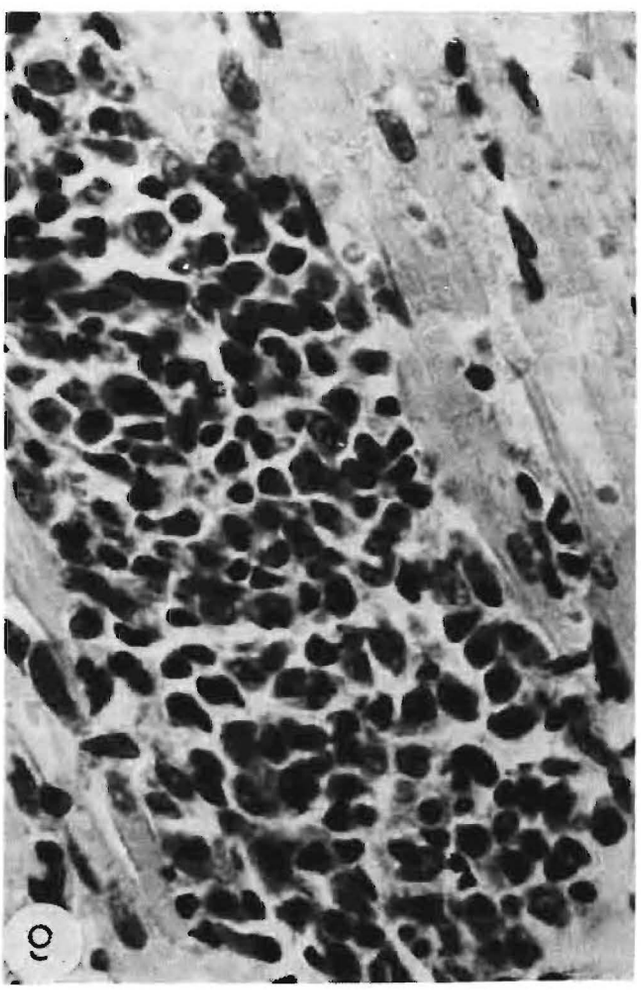


Fig. 9: Myocardium; Dog 7. Focal lymphoplasmacytic myocarditis HE × 1000

The encephalitis consisted of foci of perivascular cuffing (Fig. 2) accompanied by a few microgranulomas throughout the brain (Fig. 5). The lateral ventricular walls of Dogs 22 & 25 were involved in the granulomatous encephalitis. A spinal meningitis in Dog 22, was characterized by a focal lymphocytic inflammation.

Interstitial nephritis was moderately severe in Dogs 23 & 24 but mild in Dogs 22 & 25. The renal medulla of Dog 25 contained mild perivascular cuffs consisting of mononuclear cells. In the other animals, however, numerous foci of these interstitial cellular infiltrates were present in both the cortex and medulla. The corticomedullary junctions were severely inflamed. Necrosis of the tubular epithelial cells and complete disruption of tubules were present in the centre of some inflammatory foci. Linear infiltrations of mononuclear cells were present along tubules of the cortex and medulla in the kidneys of Dogs 22 – 24. A multifocal microgranulomatous hepatitis was demonstrated in all 4 dogs. The hepatic granulomas in Dogs 22 & 24 consisted mainly of a mononuclear cell perivascular cuffing in portal areas while a granulomatous inflammation involved the walls of sinusoids and infiltrated into surrounding hepatic cell cords. The endocytes and Kupffer cells appeared prominent and hypertrophic.

An interstitial pneumonitis which was severe in Dog 24 and moderate in all the other animals, was observed. This pneumonitis consisted of multifocal areas of plasmacytic and lymphocytic infiltration into the alveolar walls, many of which appeared thickened and highly cellular.

Lymphoid hyperplasia was present in all lymph nodes that were examined. No *Encephalitozoon* spores could be found in the tissues of these animals and no apparent differences were found to result from varying doses of organism or routes of infection in the different experimental animals.

Experiment 4 – Per os infection and immunosuppression (Table 4)

The control dogs (47C, 49C & 63C) had no lesion attributable to an *Encephalitozoon* infection.

Macroscopical pathology:

Nephritis occurred in all animals except the controls and consisted of disseminated whitish foci of up to 2 mm in diameter. The renal surfaces were irregular and contained retention cysts in Dog 62. Subcapsular white foci in the liver varied from "pin-point" to 1 mm in diameter. Lymphoid hyperplasia affected many lymph nodes but especially the mesenteric nodes. Patchy pneumonitis was present in the lungs of some animals.

Microscopical pathology:

Meningitis involving the cerebrum, cerebellum and spinal cord, was observed in some cases and consisted of a mild focal perivascular lymphoplasmacytic infiltration. These perivascular inflammatory cuffs were also present in the neuropil of the brain and spinal cord. Only a few microgranulomas were widely scattered in the central nervous system. A single vessel with a prominent lymphocytic perivascular cuff was observed in the optic nerve of Dog 44.

Interstitial nephritis occurred in several grades of severity (Table 4). It was severe in 4 dogs, moderate in 4 and mild in 1, while the controls were not affected. At 80 (Dogs 44 & 60) and 95 days (Dogs 41, 42 & 43) post-infection, the nephritis was of a subacute type in which tubulorhexis, macrophage infiltration, epithelioid cells and early fibroplasia were the predominant changes. Linear inflammatory infiltrates, located interstitially along the medullary rays, were present in all animals. The nephritis at 110 days (Dogs 45 & 48) and 125 days (Dog 62) post-infection, was chronic. Focal areas of lymphocytic and plasmacytic cell infiltrations were accompanied by moderate fibrosis of both the cortex and medulla.

A mild, focal, granulomatous hepatitis was present in all the dogs except in Dog 61 and in the controls. The microgranulomas consisted of Kupffer cells, macrophages, epithelioid cells and lymphocytes. The interstitial pneumonitis varied from very mild to moderate in the different experimental animals. It was not observed in Dogs 42, 61, 62 and the controls. Peribronchial lymphoid hyperplasia occurred in some cases. Focal accumulations of mononuclear cells, principally lymphocytes and plasma cells, were found in alveolar walls and perivascularly around some vessels (Fig. 10). Lymphoid hyperplasia was prominent in the cortical germinal centres of the lymph nodes and gut-associated tissue.

A focal interstitial myocarditis was found in Dog 41. Even with the aid of Gram's and Giemsa stains, *Encephalitozoon* organisms could not be demonstrated in the tissues of any of these dogs.

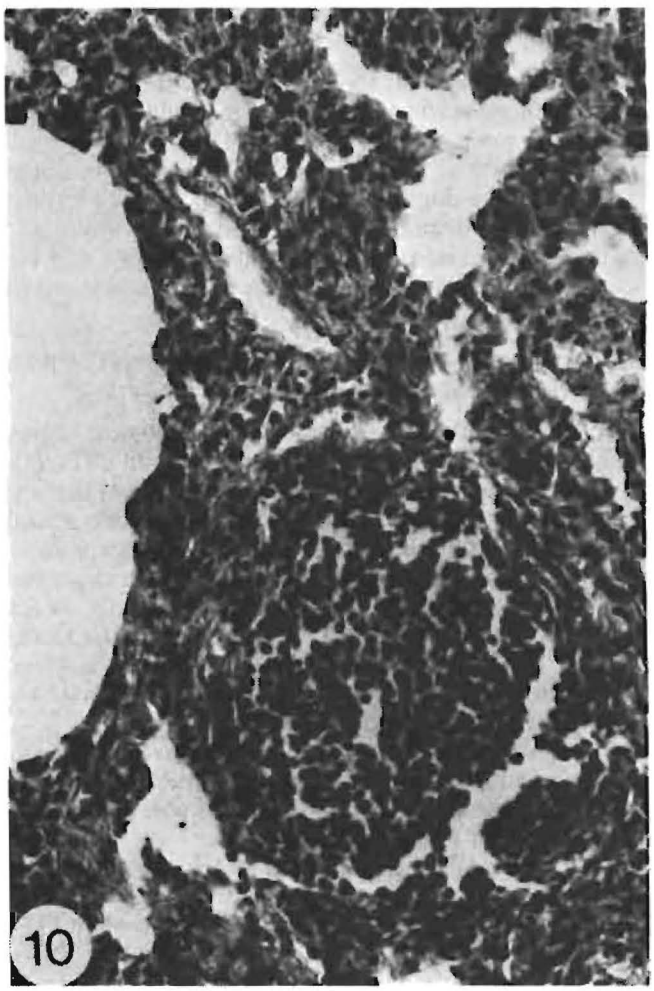


Fig. 10: Lung; Dog 44. A focal pneumonitis with a lymphocytic infiltration in the alveolar walls HE × 400

Table 4: Experiment 4 – Oral dosing of *Encephalitozoon* spores

Dog Number	41	42	43	44	45	46	48	61*	62*	47C	49C	63C
Route of infection	PO	PO	PO	PO	PO	PO	PO	PO	PO	PO	PO	PO
Dose of organisms (10^6)	2	2	2	80	40	40	80	72	72	0	0	0
Interval to death (days)	95	95	95	80	110	80	110	50	125	0	0	50
Pre-infection IFA titre	–	–	–	–	–	–	–	–	–	–	–	–
Post-infection IFA titre	160	40	320	320	320	320	160	40	80	NE	NE	–
A. MACROSCOPICAL FINDINGS:												
Nephritis	+	+ +	+	+ +	+ +	+	+ +	+ +	+ + +	–	–	–
Hepatitis	+	+	+	–	+	+	–	–	–	–	–	–
Pneumonitis	–	+	+	+ +	–	+	–	–	–	–	–	–
Lymphadenopathy	+ +	+ +	+ +	+	+ +	+	+ +	–	+	–	–	–
B. MICROSCOPICAL FINDINGS:												
Meningitis	–	+	–	+	–	+	+	–	+	–	–	–
Encephalitis	–	+	+	+	+	+	+	+	+	–	–	–
Nephritis	+	+ + +	+ +	+ + +	+ +	+ + +	+ +	+ +	+ + +	–	–	–
Hepatitis	+	+	+	+	+	+	+	–	+	–	–	–
Pneumonitis	+	–	+	+ +	+	+ +	+ +	–	–	–	–	–
Myocarditis	–	NE	–	–	NE	+	–	–	–	–	–	–
Lymphoid hyperplasia	+	+ +	+ + +	+ +	+ + +	+ +	+ +	–	+ +	–	–	–
Spores demonstrated	–	–	–	–	–	–	–	–	–	–	–	–

PO Per os
NE Not examined
* Methylpredinsolone acetate immunosuppressed

– Negative or no lesions
\+ mild focal lesions – difficult to see macroscopically or occasional increased cellularity and perivascular cuffing
+ + moderate multifocal lesions, cuffing or dense focal inflammatory infiltrates
+ + + severe lesions – prominent focal disseminated to extensively diffuse inflammatory reaction

DISCUSSION

Canine encephalitozoonosis may be suspected in young dogs showing distemper-like nervous signs or in the event of severe puppy losses (fading puppies) at breeding kennels. Signs of infection may be seen within a few weeks of birth and it appears that congenital infection is a distinct possibility[2,18]. Whole litters are often affected by the disease, and a high mortality rate is observed[1,2,13,18]. Encephalitozoonosis has been reported in Blue Foxes (*Alopex lagopus*) kept in captivity[9]. Based on the fact that 8-day pregnant vixens, which were experimentally infected with *Encephalitozoon*-parasitized mice and cell cultures, gave birth to cubs in which encephalitozoonosis developed at approximately 5 weeks of age, the transplacental route of infection was also incriminated in Blue Foxes. At necropsy, lesions of encephalitozoonosis were present and the aetiological agent was demonstrated in urine from all the cubs[8]. Vertical transmission has been demonstrated in laboratory animals. Caesarean derived rabbits reared in sterile, germ-free isolators, had typical lesions, and spores of *Encephalitozoon* could be demonstrated[15].

Few studies have been carried out on the natural transmission of canine encephalitozoonosis. Infection of newborn puppies from an environmental source such as a bitch shedding spores in her urine, is also possible[6]. In Experiment 1, the increased IFA titre and presence of lesions typical of encephalitozoonosis suggested that per os infection with *Encephalitozoon* spores from urine of natural infected dogs is feasible. The oral route of infection was also used in Experiments 2 & 4 and proved to be a successful route of infecting dogs. Horizontal transmission to laboratory mice and rabbits has been suggested[10,11], while Cox et al.[3] showed with adult rabbits that infection via both the oral and tracheal routes may be possible. It is significant that large numbers of spores (50 to more than 500/ml) were detected in the urine of rabbits 31 – 63 days after experimental infection was initiated[3]. Thereafter, only small numbers of spores were intermittently excreted and the excretion eventually ceased by day 98. It was possible in our study to demonstrate *Encephalitozoon* spores in the kidneys of Dogs 7 – 9 at 55 – 60 days after infection.

Encephalitozoon spores liberated into the environment, may play an important role in the epidemiology of the disease. The incidence of encephalitozoonosis appears to increase when animals are concentrated in large numbers in one area, like Blue Foxes in captivity[9], mice, rats and rabbits in laboratory units[5,10,11] and dogs in breeding kennels[18]. Under these circumstances horizontal and vertical transmission could be factors in perpetuating the disease.

No differences in the pathological changes were noticed when the intravenous and intraperitoneal routes of infection were compared to the per os infection, in spite of the large number of organisms given intravenously. The organism is therefore capable of breaching different biological barriers before apparent spread via the bloodstream. An unexpected observation was that the higher doses of organisms did not appear to result in increased severity of lesions. It may be that only certain vegetative forms of the organism are infective.

The IFA titres show wide variation but no specific correlation with the lesions was possible, although increased titres were present in every infected dog that was tested.

Nephritis proved to be the most consistent and prominent finding in natural encephalitozoonosis of dogs[2,6,14], Blue Foxes[9] and rabbits[3]. Histopathology of experimental encephalitozoonosis revealed that the kidney is the primary target organ, and that progression from acute to chronic interstitial nephritis occurred. The kidney lesions consisted of multifocal interstitial nephritis and perivasculitis indicative of a primary haematogenous dissemination of the infection. Signs of secondary intratubular (linear) spread, were also found. Experimental lesions, although extensive, were not as diffuse or severe as some of the reported cases of natural disease in the dog[1,2,6,12,14].

Focal hepatitis and interstitial pneumonitis were evident during macroscopical examination and were confirmed histopathologically. This has also been reported in natural encephalitozoonosis[2,6,14]. Cox et al.[3] regarded the experimentally induced pulmonary and hepatic lesions in rabbits, as indicative of early infection without chronic localization. A similar pneumonitis described by Perrin[10] as proliferative and infiltrative, occurred in mice which were inoculated by the intranasal route. An interstitial pneumonitis was still present in Dog 24 at 100 days post-infection.

Enlargement of certain lymph nodes has been reported in natural cases of the disease[1,2,14]. Mild to moderate lymphoid hyperplasia was present in the experimentally produced disease in dogs in our study.

We found that meningitis and encephalitis were mild in the majority of cases, while focal microgranulomatous encephalitis and perivascular cuffing were noted only in few of the experimental dogs. This is in sharp contrast to spontaneous and terminal encephalitozoonosis where brain lesions are prominent[2,6,12,14]. Basson et al.[1] reported cerebrovascular thrombosis and encephalomalacia in the case which they studied. Cerebral vasculitis and extensive meningoencephalitis was also found by Van Dellen et al.[19] in dogs with spontaneous disease, while the presence of focal microgranulomas in the central nervous system, was a common finding in laboratory animals[5,10]. The reduced involvement of the central nervous system of dogs in our study of experimental disease, was thought to be responsible for the absence of nervous signs and fatal disease. However, even in the natural disease, mild nervous signs may occur and spontaneous recovery is possible[2]. When adult rabbits were infected, neither parasites nor lesions were noted in the brain, while some other organs were severely affected[3]. The age at which infection is experimentally induced, as well as the immunological competence of the host, may be of great importance in the development of clinical signs and lesions of encephalitozoonosis. Mohn & Nordstoga[7] showed that neonatal exposure of newborn and young Blue Fox puppies did not induce clinical encephalitozoonosis. Intra-uterine infection appears to be essential for the development of clinical signs. Since some of our dogs were infected at 2 weeks of age without showing severe nervous lesions, puppies infected at an earlier age as well as by the transplacental route of infection, should be studied.

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NAVORSINGSNOTA **RESEARCH NOTE**

NATKARKASSINDROOM BY SKAPE: VOORKOMS EN GEOGRAFIESE VERSPREIDING

B.N. JANSEN* en P.S. PRETORIUS*

ABSTRACT: Jansen B.N.; Pretorius P.S. **Wet carcass syndrome in sheep: Incidence and geographical distribution.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 25-27 (Afrik). Faculty of Agriculture, University of the OFS, P.O. Box 339, 9300 Bloemfontein, Republic of South Africa.

A map showing the prevalence and geographical distribution of the wet carcass syndrome in sheep is presented. Although isolated cases of this syndrome are sporadically reported throughout all the sheep-producing areas of the country, the incidence of this phenomenon is epidemic in the districts of Gordonia, Kuruman, Postmasburg, Hay and Prieska. Some areas within a certain district tend to have a higher prevalence of wet carcasses especially those round the Orange, Kuruman and Molopo rivers. Vegetation type may be involved in the development of this condition in the live animal and should be investigated.

Key words: Wet carcass syndrome, geographical distribution.

INLEIDING

Die verskynsel bekend as natkarkas by skape en lammers neem sedert 1981 epidemiese afmetings by sommige abattoirs in die beheerde gebiede aan. Tot Junie 1985 is nagenoeg 60 000 skaap- en lamkarkasse met 'n beraamde waarde van R2,7 miljoen hiervoor afgekeur (Vleisraad 1985, Pretoria, ongepubliseerde verslae).

Kenmerkend van die toestand is die blinknat voorkoms van die aangetasde karkas onmiddellik na slag. Dit is te wyte aan die akkumulasie van 'n waterige vloeistof in die onderhuidse bindweefsel op die agterkwart, sye, lieste en soms op die rug van die karkas. Hierdie vog kan ook in die tussen-spierse bindweefsel van die blad en lies aangetref word (Brock et al.[1]; Hattingh, Mitchell & Ganhoa[2]; Joubert[3]). Die toestand kon tot dusver egter nog nie in die lewende dier gediagnoseer word nie.

Volgens Brock et al.[1] het die natkarkastoestand 'n streeksvoorkoms wat hoofsaaklik tot Gordonia en aangrensende distrikte beperk is, met sporadiese gevalle uit die suid-wes Oranje-Vrystaat.

In 'n ondersoek na moontlike fisiografiese faktore (voeding, bestuur, drinkwater, vervoer, ens.) wat die toestand van natkarkas ten grondslag mag lê, het verdere en meer volledige data oor die voorkoms en geografiese verspreiding van die verskynsel beskikbaar geword. Hierdie inligting dien as agtergrond vir 'n omvattende ondersoek na die verskynsel.

PROSEDURE

Inligting oor die geografiese verspreiding van natkarkasvoorkoms is op twee maniere ingesamel:

Identifikasie van respondente

Ten einde 'n keuse van produsente te doen wat as respondente vir 'n gevallestudie kon dien waar natkarkas die meeste en gereeldste voorkom, is produsenteverslae vanaf die Vleisraad oor die aantal en herkoms van diere, waarvan die karkasse by City Deep en Chamdor abattoirs vir natkarkas afgekeur is, ontleed. Van die beheerde markte het voorgenoemde twee abattoirs die hoogste voorkomssyfer, terwyl afkeurings by ander abattoirs sporadies en laag in frekwensie is (Vleisraad, 1984, Pretoria, ongepubliseerde verslae.)

Produsente is gegroepeer volgens die frekwensie van voorkoms en aantal karkasse afgekeur binne 'n bepaalde bemarkingsperiode, naamlik een of meer aangetasde besendings skape in (a) elke maand van 'n 4-maande bemarkingsperiode, (b) in 3 van die 4 maande, (c) in 2 van die 4 maande en (d) slegs een van die 4 maande. Die voorkoms van natkarkas is as persentasie per produsent bereken.

Gevallestudie

Sestig produsente in vyf teikendistrikte is met behulp van bogenoemde metode by die ondersoek betrek. Drie groepe produsente uit elk van die teikendistrikte is proporsioneel tot die persentasie natkarkasvoorkoms ingesluit, nl. die wat (a) gereeld natkarkas kry (n = 35), (b) nie natkarkas kry en direkte bure van eersgenoemde is nie (n = 17) en (c) wat as "modelboere" volgens die plaaslike voorligtingsbeampte geïdentifiseer is (n = 8). Die gevallestudie is met behulp van die vraelystegniek met persoonlike onderhoudsvoering uitgevoer. Mostert et al.[4] se veldtipekaart is van distriksgrense voorsien en gebruik om die natkarkasvoorkomsareas te karteer.

RESULTATE EN BESPREKING

Met die ontleding van die produsenteverslae is gevind dat 66,4% van die totale aantal afkeurings vir natkarkas gedurende die periode Januarie tot April 1984 (n = 5996) by City Deep en Chamdor, vanuit vyf distrikte in die Noordwes Kaapprovinsie afkomstig was, nl. Gordonia (32,1%), Kuruman (11,0%), Postmasburg (9,9%), Hay (7,7%) en Prieska (5,8%). Hierdie distrikte het as teikendistrikte vir die gevallestudie gedien.

Die voorkoms van natkarkas vanuit verskillende distrikte varieer egter van maand tot maand (Tabel 1). Hoewel die distrik Gordonia gewoonlik bo aan die rangordelys vir natkarkasvoorkoms verskyn, is dit nie elke maand die geval nie. Dit is ook duidelik dat laer op die rangordelys groter variasie tussen distrikte voorkom en verteenwoordig dit sporadiese gevalle wat oor 'n wye gebied in die kleinveeproduksieareas voorkom. Die dis-

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Tabel 1: Distriksrangorde van Natkarkassindroomvoorkoms by skape

	City Deep				Chamdor		
Rangorde	Januarie	Februarie	Maart	April	Januarie	Februarie	Maart
1e	Gordonia 24,93%	Gordonia 22,57%	Gordonia 20,48%	Hay 14,26%	Gordonia 40,00%	Gordonia 55,16%	Gordonia 35,13%
2e	Hay 19,74%	Hay 12,20%	Prieska 13,06%	Gordonia 10,89%	Postmasburg 22,19%	Postmasburg 8,61%	Kuruman 12,28%
3e	Kuruman 18,52%	SWA 10,29%	Postmasburg 10,97%	Postmasburg 10,89%	Kuruman 19,53%	Kuruman 7,07%	Postmasburg 11,58%
4e	Prieska 9,26%	Kuruman 9,32%	Hay 10,32%	Prieska 10,58%	Wolmaransstad 5,78%	Herbert 5,69%	Delareyville 10,48%
5e	Postmasburg 5,55%	Prieska 9,13%	Phillipstown 6,77%	Kuruman 7,52%	Hopetown 3,13%	Hopetown 2,60%	Herbert 8,38%
6e	Stockenstroom 2,84%	Bloemfontein 8,78%	Herbert 5,81%	Koppies 6,29%	Herbert 2,34%	Kimberley 2,52%	Fauresmith 3,99%
7e	SWA 2,72%	Vryburg 7,01%	Hopetown 5,48%	Vrede 5,67%	Calvinia 1,41%	Luckhoff 2,36%	Vryburg 3,59%
8e	Kroonstad 2,72%	Postmasburg 4,32%	Kuruman 4,82%	Petrusburg 5,21%	Luckhoff 1,41%	Carnarvon 2,27%	Bothaville 2,59%
9e	Petrusburg 2,35%	Bultfontein 3,17%	Fauresmith 4,84%	Luckhoff 4,14%	Coligny 1,25%	Petrusburg 2,11%	Carnarvon 2,50%
10e	Cornelia 2,35%	Kimberley 2,21%	Petrusburg 3,23%	Vryburg 3,68%	Carnarvon 0,94%	Prieska 1,87%	Kimberley 2,09%

trikte wat as teikendistrikte gekies is, verskyn egter gereeld onder die eerste vyf op die ranglys (Tabel 1). Hierdie gebied is basies dieselfde as die wat deur Brock et al.[1] geïdentifiseer is.

Uit Tabel 1 blyk dit ook dat daar skynbaar distriksvoorkeure bestaan ten opsigte van die bemarking van skape en lammers by sekere abattoirs. Hoewel die distrikte Hay en Prieska gereeld betreklik hoog op die distriksrangordelys by City Deep voorkom, verskyn Hay nie onder die eerste tien by Chamdor; en Prieska slegs een keer tiende op die ranglys (Tabel 1).

Vanaf inligting uit die vraelyste met die gevallestudie was dit moontlik om 'n akkurate natkarkasvoorkomskaart te kon opstel (Fig. 1). Hierdie gebied verteenwoordig feitlik die hele Noordwes Kaapprovinsie en dele van die Suidwes Vrystaat. Besondere hoë konsentrasies van voorkoms van natkarkas word in die distrikte Gordonia, Postmasburg en Hay aangetref en ondersteun Brock et al[1] se waarneming. Wat egter opvallend is, is die feit dat natkarkasvoorkoms geneig is om in sekere dele binne 'n spesifieke distrik 'n hoër frekwensie van voorkoms te hê as in die res van die distrik en byna epidemiese afmetings aan te neem (Fig. 1), bv.:

Gordonia

Twee gekonsentreerde voorkomsgebiede word in Gordonia onderskei. Eerstens 'n area vanaf die oostelike grens tussen die distrikte Gordonia en Kuruman aan weerskante van die Kurumanrivier weswaarts verby Askam met 'n afname in voorkoms in die gebroke veld en geen voorkoms van natkarkas in die hardeveld. Langs die Moloporivier en Askam-omgewing kom ook gereelde gevalle van natkarkas voor. Voorts word 'n konsentrasiegebied aangetref in 'n area noord van die Oranjerivier tot noordwes van Upington met 'n strook wat effens suid van die Oranjerivier uitloop na die Neilersdrif-omgewing. Die voorkoms van natkarkas neem af in die rigting van die distrik Kenhardt en die Boesmanland (Fig. 1).

Kuruman

Gereelde karkasafkeurings as gevolg van natkarkas kom gekonsentreerd wes van Van Zylsrust aan weerskante van die Kurumanrivier voor en neem suidwaarts af. In die omgewings van Kuruman en Hotazel self kom egter min natkarkas gevalle voor (Fig. 1).

Postmasburg

Volgens Fig. 1 word in hierdie distrik ook twee gekonsentreerde voorkomsareas aangetref. Albei lê wes van die Langeberge by Olifantshoek, een in die omgewing van Mally wat suidwaarts geleidelik afneem en oorgaan in die tweede gebied wat ongeveer vanaf Lombaardsvlakte tot by Pearsons Hunt en Koeipan strek. In die omgewing van Daniëlskuil word relatief min gevalle gerapporteer.

Hay

'n Gekonsentreerde voorkomsarea strek noord van die Oranjerivier tot noordwes van Niekerkshoop in die Koedoeskop-omgewing. Noord van Griekwastad kom selde afkeurings voor.

Prieska

Die hoogste en gereeldste voorkoms van afkeurings vir natkarkas word aangetref in 'n strook aan weerskante van die Oranjerivier (Fig. 1). Suidwaarts na die distrik Kenhardt en die Bo-Karoo neem die voorkoms van natkarkasgevalle baie vinnig af.

'n Konsentrasiegebied van laer intensiteit as die wat

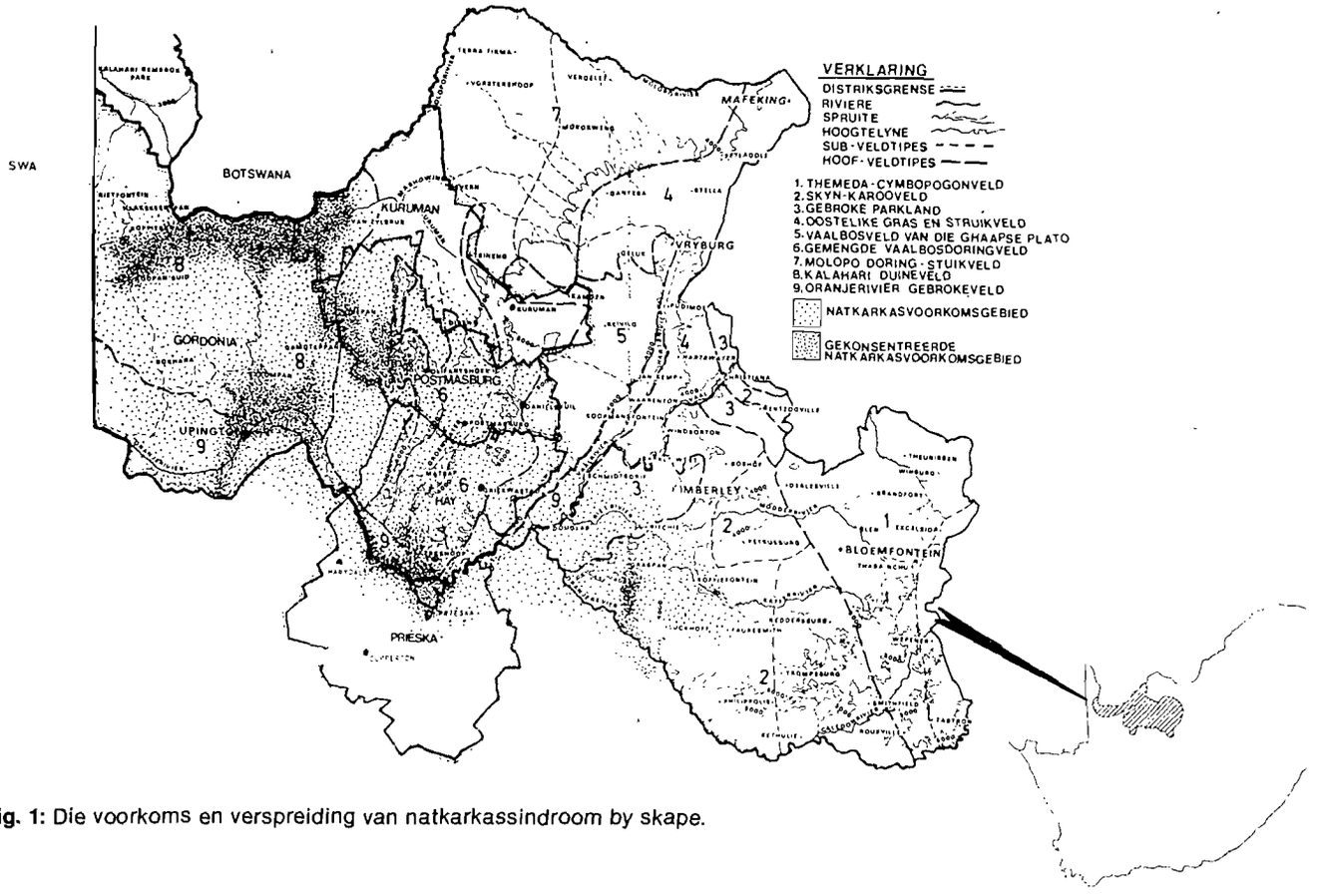

Fig. 1: Die voorkoms en verspreiding van natkarkassindroom by skape.

reeds bespreek is kom voor in die Witput-, Luckhoff-, Petrusville- en Hopetown-omgewing. Die distrikte Herbert, Hopetown, Phillipstown en Fauresmith grens egter in hierdie gebied aan mekaar, met die gevolg dat die voorkoms per distrik betreklik laag is. Uit Fig. 1 is dit ook opvallend dat die gekonsentreerde natkarkasareas by uitstek in die Kalahariduine- en Oranjerivier gebroke-veldtipes (tipes 8 & 9) voorkom. Tog vertoon die Middel-Kalahari nie gekonsentreerde areas van natkarkasvoorkoms nie en is dit veral die gebiede om die Oranje-, Kuruman- en Moloporiviere waar dit gekonsentreerd voorkom. Volgens Mostert et al.[4] is 'n groot gedeelte van die Middel-Kalahari egter betreklik onlangs eers vir boerdery oopgestel as gevolg van 'n gebrek aan drinkwater. Daar kan dus met reg redeneer word dat die gebiede langs die riviere vir 'n langer tydperk aan beweiding en vertrapping blootgestel is as dié dieper die Kalahari in met waarskynlike swakker voedingstoestande.

Die vraag ontstaan nou tot watter mate die gehalte en samestelling van die natuurlike weiveld 'n bydrae maak tot die ontwikkeling van die natkarkastoestand in die lewende dier. Waarnemings in hierdie verband mag dalk verdere lig op die natkarkasverskynsel werp.

GEVOLGTREKKING

Uit die resultate van hierdie ondersoek is dit duidelik dat sekere distrikte in die Noordwes Kaapprovinsie onomwonde as natkarkasprobleemareas uitgesonder kan word. Hierdie gebied word begrens deur die distrikte Gordonia, Kuruman, Postmasburg, Hay en Prieska. Binne hierdie distrikte kom daar sekere gebiede voor wat as natkarkaskonsentrasie-areas geïdentifiseer is en maak dit hoofsaaklik die weidingsgebiede in die onmiddellike omgewing van die Oranje-, Kuruman- en Moloporiviere uit. Hiervolgens wil dit voorkom asof daar 'n verband tussen natkarkasvoorkoms en weidingsamestelling en weidingstoestand mag bestaan en verdien verdere ondersoek.

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ABSTRACT

SAMEVATTING

AN OVINE HEPATOTOXICOSIS CAUSED BY THE PLANT *HERTIA PALLENS* (DC.) KUNTZE (ASTERACEAE)

A field outbreak of *Hertia pallens* poisoning in sheep is described. The hepatotoxicity of the plant was experimentally demonstrated in 7 sheep which developed lesions that ranged from a diffuse degeneration to centrilobular necrosis. These lesions occasionally extended to the midzonal area of the lobules. In addition to a lung oedema, a diffuse mononuclear interstitial pneumonia was present in 3 of the sheep. Botanical, clinical and pathological data are given. (Prozesky, L., Kellerman, T.S., Jordaan, P., Welman, Wilhelmina G. & Joubert, J.P.J., 1985. An ovine hepatotoxicosis caused by the plant, *Hertia pallens* (DC). Kuntze (Asteraceae). *Onderstepoort Journal of Veterinary Research*, **52** 233 – 238 (1985).)

ABSTRACT

SAMEVATTIMG

ISOLATION AND IDENTIFICATION OF A SOUTH AFRICAN LENTIVIRUS FROM JAAGSIEKTE LUNGS

In the course of attempts to grow the jaagsiekte retrovirus in cell culture, a typical lentivirus was isolated from the first time in South Africa from adenomatous lungs. Morphologically the virus could not be distinguished from other lentiviruses, but serologically it was shown to be more closely related to visna virus than to caprine arthritis-encephalitis virus. However, a preliminary restriction enzyme analysis of the linear proviral DNA of this new lentivirus (SA-DMVV) revealed that it is significantly distinct from visna virus and CAEV and therefore may represent a third type of lentivirus. Antibodies to the virus were demonstrated in a number of sheep in various parts of the country, but a direct link to a disease condition was not found. Attempts to produce lung lesions by intratracheal injection of the virus have been unsuccessful to date but a transient arthritis was produced by intra-articular inoculation. Viral replication seems to be enhanced in jaagsiekte lungs. (Payne, A., York, D.F., De Villiers, E-M., Verwoerd, D.W., Quérat, G., Barban, V., Sauze, N. & Vigne, R., 1986. Isolation and identification of a South African lentivirus from jaagsiekte lungs. *Onderstepoort Journal of Veterinary Research*, **53**, 55 – 62 (1986).)

ABSTRACT

SAMEVATTING

THE EFFECT OF ARSENICAL DIPS ON *PARAFILARIA BOVICOLA* IN ARTIFICIALLY INFECTED CATTLE IN SOUTH AFRICA

The possible adverse effect of arsenical tick control dips on *Parafilaria bovicola* infections was investigated in 48 artificially infected cattle. A treatment group of 24 cattle was dipped in a plunge dip containing 1 600 ppm arsenic trioxide. A control group of the same size was dipped in an organophosphate dip containing a mixture of chlorfenvinphos and dioxathion.

Regular weekly to 3-weekly dipping had not effect initially on the prevalence of ovipositional blood spots of *P. bovicola* in either group. However, from 4 months after bleeding commenced there was a significant reduction in blood spots in the arsenic-dipped cattle and, on slaughter at 12 – 14 months after infection, the arsenic group had significantly fewer live worms and fewer carcass lesions.

Arsenic residues in muscle samples of treated cattle were 11,6 times higher than in the controls. It is proposed that arsenic residues in the sub-cutaneous muscle layers increase with repeated dipping until a level toxic to *P. bovicola* is finally reached. Older cattle would therefore be refractory to infection and their carcasses at slaughter would not be affected. (Nevill, E.M., 1985. The effect of arsenical dips on *Parafilaria bovicola* in artificially infected cattle in South Africa, *Onderstepoort Journal of Veterinary Research*, 52, 221 – 225 (1985).

CASE REPORT GEVALVERSLAG

HERNIA REPAIR IN A HORSE

MARIANNE THOMSON*

ABSTRACT: Thomson M. **Hernia repair in a horse.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 29-31 (En). P.O. Box 163, 6835 Ceres, Republic of South Africa.

The repair of a large defect in the abdominal wall of an American Saddlehorse by implantation of a polypropylene monofilament mesh.

Key words: Horse, abdominal hernia, Marlex mesh.

INTRODUCTION

Repair of large defects in the abdominal wall of the horse is sometimes impossible due to failure to appose tissue by standard surgical techniques. Bridging such a gap by implating an inert, pliable substance of sufficient strength can overcome this problem in selected cases. Such a case is described.

HISTORY

A three year old Saddlehorse gelding injured himself on a fence post when attempting to clear the fence (17.4.1984). The author was telephonically consulted by the referring veterinarians. It was decided to attempt repair only after the acute phase of the trauma had passed.

The extensive bruising and superficial skin abrasions were treated with cold water hosing, and penicillin was used parenterally. A low volume diet of cubed feed was prescribed. A tetanus toxoid booster was given.

CLINICAL EXAMINATION

On 8.5.1984, 21 days after the injury, the horse was presented for surgery. There was a massive swelling in the left abdominal wall ventral to the wing of the ilium. The swelling was roughly 30×40 cm in diameter and projected $25 - 30$ cm from the surrounding abdominal wall. It was sensitive to palpation, but a hernial ring could be felt in places. The superficial cuts and abrasions of the skin had healed well.

The horse had lost about 100 kg in weight since the accident; the coat was dry and he was nervous and anxious in appearance. Bowel sounds, temperature, pulse, respiration, packed red cell volume, haemoglobin, defaecation and urination were normal.

SURGERY

Surgery was performed on 8.5.84. Anaesthesia was induced with a mixture of 1 ℓ of 5% guaiacol glycerol ether and 1 g thiopentone sodium to effect. After intubation, closed circuit anaesthesia was maintained by a mixture of halothane, nitrous oxide and oxygen.

The horse was placed in dorso-lateral recumbency to place the hernia dorsally.

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After removing all remaining skin crusts, a wide surgical field was prepared by shaving and then scrubbing with a tamed iodine soap (Betadine; Adcock Ingram) for 5 minutes, followed by a alcohol-acriflavine spray.

The surgical field was covered with an adhesive sterile drape (Steridrape; 3M Co.). The entire horse was then draped with sterile cotton drapes.

An incision was made in a dorso-ventral direction over the hernial sack. The lower end of the incision would allow complete ventral drainage when the horse was in a standing position.

There was a 2 cm layer of fresh, fibrous connective tissue under the skin. The peritoneum was not apparent in the hernial sack. Part of the colon was outside the abdominal wall in the hernial sack. There was no sign of peritonitis. The fibrous adhesions between the colon and subcutis were loosened digitally and by blunt dissection. Haemorrhage was minimal. The subcutis was markedly haemosiderin-stained in areas.

Upon replacing the colon into the abdomen, an oval defect of 20×18 cm was revealed. The muscle, peritoneum and fibrous connective tissue had organised into a firm, inelastic hernial ring approximately $1{,}5 - 2$ cm thick.

Lane's forceps were clamped to the cranial and caudal edges of the ring and it was attempted to appose the edges for suturing. The most strenuous pulling could not even narrow the 18 cm cranio-caudal gap by as much as 5 cm.

The colon was held inside the abdominal cavity by placing a moist sterile drape over it and tucking the edges under the muscles.

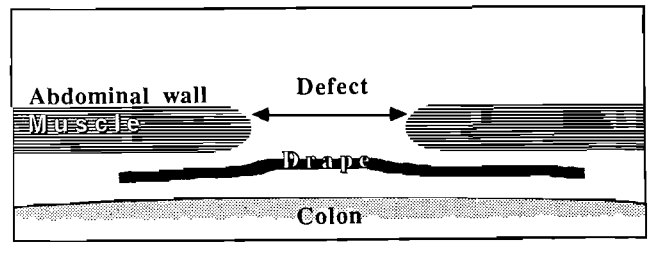

Fig. 1: Cross section of defect showing placement of drape.

A sheet of autoclaved Marlex mesh was layed over the defect with the edges between the colon and the abdominal wall (the sterile drape holding the viscera out of the way).

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A series of vertical mattress sutures were placed around the entire ring. Seven packets of a size 5 silicone-treated, braided, sterile nonabsorbent surgical suture (Ticron, Davis & Geck) were needed. The ends were left untied, quite long and the two ends of each suture were clamped together with a small haemostat. Approximately 20 sutures were placed in this fashion (Fig. 2 & 3). No attempt was made to freshen the edges of the hernia.

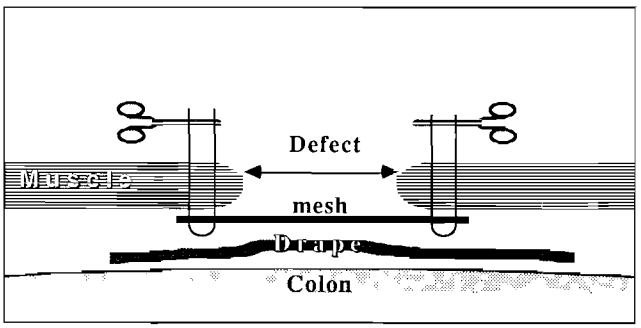

Fig. 2: Cross section of defect showing location of mesh and sutures.

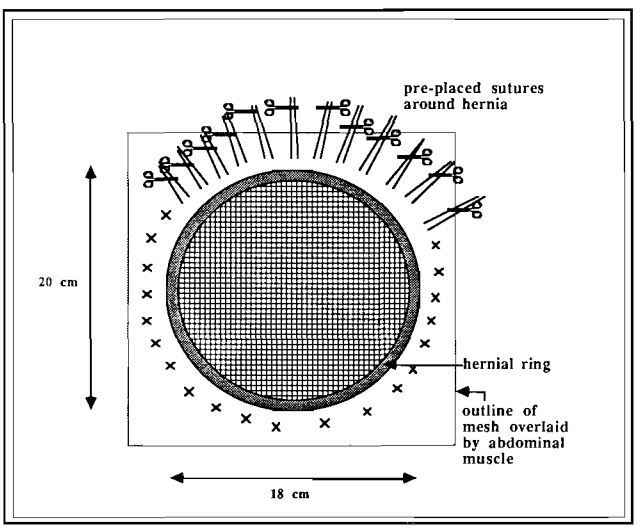

Fig. 3: Schematic view from outside of the wound and placement of satures.

The sutures in half of the defect were pulled tight and tied. By pushing the mesh away from the muscle, the sterile drape that had been used to keep the colon moist, away from the surgical field and protected from accidental penetration with a needle, was pulled out between two sutures. Fifty ml procaine penicillin was poured into the abdominal cavity. Thereafter, the rest of the sutures were tied. The mesh fitted tightly across the defect and held the viscera within the abdomen.

It was attempted to fill in tissue over the mesh, but this was only partially successful since the subcutis was friable. The excess skin was trimmed away. The skin was sutured with a synthetic suture material (Vetafill, Bengen). There was a deadspace between the skin and the mesh that could not be filled. The skin incision was extended ventrally and the most ventral 5 cm of the skin was left unsutured to ensure natural drainage. This unsutured part of the incision was ventral to the mesh by a few centimeter (Fig. 4).

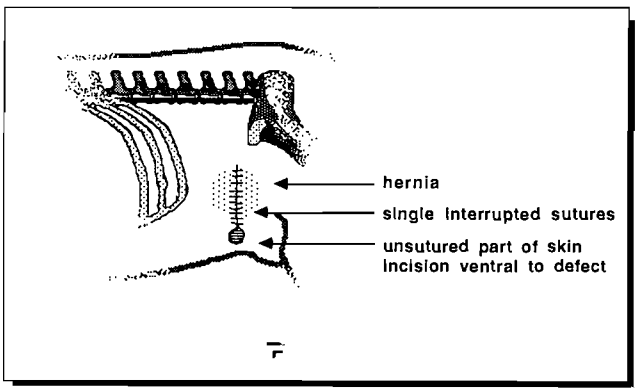

Fig. 4: Schematic representation of lateral abdominal aspect showing incision and ventral unsutured part.

Anaesthetic time from induction, was 2 hours. The patient was maintained on oxygen until it first attempted to regain its feet. Recovery from anaesthesia was uneventful.

POST-SURGICAL CARE

8.5.1984: Forty ml Compropen (Glaxo) (each ml contains 150 000 i.u. benethamine penicillin, 150 000 i.u. procaine penicillin) i/m, 20 ml of an anti-inflammatory, analgesic, antipyretic – isopyrin 24 g/100 ml and phebuzine 13 g/100 ml combination (Tomanol, Byk Gulden), 5 g streptomycin bd.

9.5.1984: Eating, passed no faeces. T 39°C. Thirty ml trimethoprim 4% m/v and sulfadoxine 20% m/v (Trivetrin; Wellcome) i/v, streptomycin 5 g bd. i/m. Wound draining well from unsutured ventral point.

10.5.1984: Put out on small green pasture close to hospital – grazing. Trivetrin and streptomycin. Habitus improved. Wound draining well.

11.5.1984: Not passing faeces yet. T. 37°C. Anorexic. Wound draining. Ten ml of a vitamin amino-acid compound (Vitoplex, Sterivet) i/v. Thirty ml of clanobutin sodium 106 mg/ml, a choleretic and digestant (Bykahepar, Byk Gulden) i/v, 10 ml flunixin meglumine 50 mg/ml (Banamine; Schering) i/v. Green feed and steamed bran mash taken after analgesic. Continue antibiotic.

12.5.1984: Large amount of faeces passed. Habitus markedly improved. T 37,5°C. Appetite good. Continue green feed and bran mash. Marked oedema of sheath. Wound draining well, washed ventral unsutured area with lukewarm betadine solution. Continue Trivetrin 30 ml i/v.

15.5.1984: Skin caudal to the incision hard and dark in colour. Swelling of prepuce reducing. Discharged from hospital.

20.5.1984: Sutures removed. Ventral drainage closed; area still sensitive to touch. Allowed to move around small paddock freely.

15.6.1984: Small abscess at ventral point of wound lanced and irrigated with mixture of betadine solution plus 5 volumes hydrogen peroxide. Walking exercise commenced. No

sign of a bulge in the abdominal wall. Area still sensitive to deep palpation. Horse gaining condition rapidly.

October 1984: By this time the horse was back in full use as a hack and took part in an amateur, junior type cross country event, clearing low jumps. His condition had returned to almost the same as before the accident. No sign of the injury remained, except for superficial scars. Palpation is vigourously resented to equal extent on both sides of the body.

CONCLUSION

A defect that would have necessitated euthanasia was closed by the implantation of Marlex mesh. No tissue could be interposed between the porous mesh and the skin and yet no peritonitis resulted. The author is of the opinion that a fibrin layer sealed the pores in the mesh within hours. A Penrose drain was considered and rejected because it was feared that contamination could migrate up the drain and through the mesh to the abdominal cavity, resulting in peritonitis. In retrospect the natural drainage left at the ventral aspect of the wound with the skin opening ventral to the mesh covered area, was essential and the correct procedure in this case.

The author is not sure whether it was correct to delay surgery for such a long period, but it was reasoned that the risk of infection would be increased when operating in the presence of devitalized tissue and haematomas. The longer period lessened the risk of infection; the hernial ring however, became so organised that it became impossible to appose the muscle edges of the wound.

ACKNOWLEDGEMENTS

The help of the following persons are acknowledged: Drs M J Versfeld, S Kitley and G de Wet of Worcester Veterinary Hospital who referred the horse. Dr Versfeld acted as anaesthetist and Dr De Wet assisted the author; Mrs E P van Dyk for operating the Narkovet E anaesthetic machine and typing the manuscript; and Drs Longland and Antrobus (Wellington Veterinary Hospital) for the loan of the Marlex Mesh.

ABSTRACT

SAMEVATTING

THE NATURAL RESISTANCE OF CATTLE TO ARTIFICIAL INFECTION WITH *COWDRIA RUMINANTIUM*: THE ROLE PLAYED BY CONGLUTININ

The conglutinin titres of year-old Bonsmara-cross cattle infected with *Cowdria ruminantium* were inversely proportional to the severity of the reactions elicited by the infection. There was no correlation, however, between conglutinin levels of 8-month-old calves of the same breed, sex and origin and their susceptibility to heartwater. The role possibly played by conglutinin in the non-specific resistance of cattle to heartwater and in the epidemiology of the disease is discussed. (Du Plessis, J.L., 1985. The natural resistance of cattle to artifical infection with *Cowdria ruminantium*: the role played by conglutinin. *Onderstepoort Journal of Veterinary Research*, **52** 273 – 277 (1985).)

ABSTRACT

SAMEVATTING

AN *IN VIVO* COMPARISON OF THE EFFICACY OF THE HEARTWATER BLOOD AND GROUND-UP TICK SUSPENSION VACCINES IN CALVES

Two groups of calves were respectively immunized with heartwater blood (BV) and ground-up tick suspension (GUTS) vaccine. A third group was left unimmunized as controls. No difference in the immune status conferred could be demonstrated between the 2 vaccines at 6 months and 12 months challenge after vaccination. An index, based on the rectal temperature before and during the reaction, was calculated as an aid in evaluating the data. In practice, the evaluation of heartwater vaccination by challenge is more effective at 12 months than at 6 months after vaccination. No effective difference was demonstrated between the 2 vaccines in their immunizing efficacy. (Bezuidenhout, J.D. & Spickett, A.M., 1985. An *in vivo* comparison of the efficacy of the heartwater blood and ground-up tick suspension vaccines in calves. *Onderstepoort Journal of Veterinary Research*, **52**, 269 – 271 (1985).)

ABSTRACT

SAMEVATTING

THE IMMUNE RESPONSE IN A DOG TO *ENCEPHALITOZOON CUNICULI* INFECTION

The immune response to *Encephalitozoon cuniculi* infection in a dog was investigated by means of the indirect fluorescent antibody test, the leucocyte migration inhibition test and the radial immunodiffusion test for serum IgG and IgM levels.

Specific antibodies were detected within 7 days of infection and they persisted for 370 days. A cell-mediated immune response was detected from Day 13 following infection until Day 97.

Histopathological examination showed plasma cell infiltration of the kidneys, meninges, lung, bladder, smooth muscle and spleen. (Stewart, C.G., Collett, M.G. & Snyman, Helena, 1986. The immune response in a dog to *Encephalitozoon cuniculi* infection. *Onderstepoort Journal of Veterinary Research*, **53**, 35 – 37 (1986)).

CASE REPORT GEVALVERSLAG

PERINEPHRIC EXTRAVASATION OF URINE WITH PSEUDOCYST FORMATION IN A CAT

JUDITH K. GEEL*

ABSTRACT: Geel J.K. **Perinephric extravasation of urine with pseudocyst formation in a cat.** *Journal of the South African Veterinary Association* (1986) 57 No. 1, 33-34 (En). P.O. Box 47455, Parklands, 2121 Johannesburg, Republic of South Africa.

A case of unilateral perinephric pseudocyst formation as a result of extravasation of urine is reported. The main presenting signs and the initial clinical examination did not directly indicate severe renal involvement. The subsequent development of pseudocysts as a result of chronic extravasation of urine was positively diagnosed by laboratory investigations and ancillary procedures. Treatment consisted of surgical removal of the affected kidney.

Key words: Perinephric pseudocyst, cat.

INTRODUCTION

Perinephric pseudocysts in the cat have only been reported on a few occasions. Extravasation of urine with subsequent formation of an unilateral uriniferous perinephric pseudocyst occurred as a result of suspected rupture of the right kidney pelvis and ureter following chronic inflammation thereof. The fluid filled sac had no epithelial lining and is thus termed a pseudocyst.

CASE REPORT

A vaccinated, neutered, one year old, male Siamese cat was presented with a gradual onset of a depressed habitus and appetite over a period of one month. The cat's normal diet was chicken flavoured tinned food and chicken livers. On initial examination, the habitus of the cat was found to be mildly depressed. Its temperature was 38,8°C and the respiratory movements and rate were normal. A small volume of freely voided urine was bright red in colour suggestive of fresh blood. The cat was hospitalised pending further investigations. A urinalysis using reagent strips (N-Multistik, Ames) on a urine sample collected by cystocentesis showed pH 5, no trace of blood and all other parameters normal. The SG was 1,020 (Uricon-N refractometer). Hookworm eggs found on a faecal flotation test was considered to be an incidental finding, the cat was dewormed.

As the urine sample collected by cystocentesis was found to be free of occult blood and no intact red blood cells were found on the sediment smear examination, the origin of blood in the freely voided urine was thought to arise caudal to the bladder. A tentative diagnosis of haematuria as a result of urethral culculi or urethritis was made. Amoxycillin trihydrate (Clamoxyl, Beecham Animal Health) and urogenital antispasmodic treatment (Buscopan compositum, Boehringer Ingelheim) was instituted.

On the following day, the voided urine was free of blood. The cat's appetite was still poor and the treatment continued. On the third day, the cat's abdomen was tense and painful on palpation. The temperature was 39,5°C. A blood smear and haematological examination were normal. A liver profile showed a slight elevation in alanine transaminase (ALT) only. The serum creatinine level was also slightly raised indicating possible renal pathology. The cat was maintained on antibiotic and renal supportive therapy for four days. The condition and habitus of the cat improved dramatically and it was discharged on the seventh day.

The cat was presented three weeks later with a very tense abdomen which was very painful on palpation. The temperature was 39°C. Under general anaesthesia, a soft fluctuating mass was palpable within the abdomen. An exploratory laparotomy revealed extensive lipolysis of the fat around the right kidney. The liquified fat was drained so as to visualise the outer surface of the kidneys, which appeared normal. The liver was slightly enlarged and light in colour. There was accentuation of the lobular architecture. Laboratory tests performed at this stage revealed normal haematology and total serum proteins.

Antibiotic and liver supportive treatment was given and the cat responded satisfactorily. It was released, only to be readmitted twelve days later with a soft fluctuating mass palpable within the abdomen on the right hand side. Microscopic examination of the red-tinged fluid aspirated from the sac showed the presence of erythrocytes and a small number of mature neutrophils. Another exploratory laparotomy was performed and a sac of fluid was removed in toto from the right hand side of the abdomen in close proximity to the right kidney. A biopsy of the right kidney was also taken.

Histopathological examination of the cyst wall revealed a thick walled capsule with the presence of some inflammatory cells and a central lumen containing necrotic material and pigments. Fat necrosis was also found outside the capsule. The kidney cortex revealed a mild, multifocal, plasmacytic perivascular nephritis. The lesions were suggestive of a chronic infectious condition.

When the cat was returned one week later for suture removal, another intra-abdominal fluid-filled cyst was palpated. Again microscopic examination of the fluid aspirate showed the presence of erythrocytes and neutrophils. Culture of the fluid aspirate was negative.

An intravenous pyelogram was performed using sodium amidotrizoate and meglumine amidotrizoate (Urographin 60%, Schering) as a contrast medium. Radiographs were taken at 0, 5 and 10 minutes post-administration in a dorso-ventral position and after 15

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and 20 minutes in a dorso-ventral and lateral position. An accumulation of contrast medium caudal to the right kidney was observed after 20 minutes.

To confirm the presence of urine in the sac, an analysis of the urea content of the fluid (11,2 mmol/ℓ) and the serum (10,6 mmol/ℓ) was performed. The serum creatinine level remained slightly elevated (194 mmol/ℓ).

The right kidney and the associated cyst were surgically removed and sent for histopathological examination. The cat made an uneventful recovery.

Histopathological examination of the kidney revealed a severe hydronephrosis with dilation of the pelvis and the presence of casts in the pelvic lumen. Focal necrosis of the collecting duct tubular epithilium was secondary to a disturbance in blood flow. Severe fribrosis was present in parts of the kidney parenchyma. There was an associated chronic ureteritis and dilation of the ureter which indicated that the obstruction was present in this region of the urinary tract, the nature of which was suspected to be renal calculi.

CONCLUSION

At the time of the first explorative laparotomy, the liver was thought to be undergoing fatty degeneration as a result of perirenal fat lipolysis. After the second recurrence of pseudocyst formation a tentative diagnosis of perinephric pseudocyst formation as a result of extravasation of urine as described by Abdinoor[1] was made. A positive diagnosis was made after the intravenous pyelogram indicated a leakage of urine caudal to the right kidney and the urea content of the fluid aspirated from the sac was found to be higher than that of the blood.

DISCUSSION

Pseudocysts are classified on the nature of their content. A uriniferous pseudocyst contains urine. Only a chronic leakage of urine from a tear in the renal pelvis or upper ureter as a result of trauma, surgery, or erosion by renal calculi will result in uriniferous pseudocyst formation. In addition, a persistent underlying cause such as an obstruction, stenosis or inflammation of the ureter must be present to maintain the flow of urine to the perinephric tissues.

As a result of the presence of urine in the perirenal tissue, aseptic inflammation with lysis of perinephric fat occurred and a thick fibrous capsule devoid of epithelial lining formed around the urine. It is reported that this inflammatory response may lead to urethral obstruction which perpetuates and aggravates urine leakage[1]. Thus, the removal of the pseudocyst alone led to the formation of another as a result of continued urine extravasation and only when the kidney was removed, did the problem resolve.

ACKNOWLEDGEMENTS

I would like to thank Prof P Bland van den Berg (Dept. of Medicine, Faculty of Veterinary Science, Onderstepoort), Prof F Reyers (Dept. of Clinical Pathology, Faculty of Veterinary Science, Onderstepoort), and Dr W S Botha (Veterinary Pathologist, P.O. Box 12731, 0110 Onderstepoort) for their help in arriving at a definite diagnosis of the condition. My thanks also go to my colleagues Dr B Gaisford and Dr P H Turner (Bluff Veterinary Clinic, 910 Bluff Rd, Durban) for their assistance on this case.

REFERENCE

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CASE REPORT **GEVALVERSLAG**

CHYLOTHORAX WITH CONCURRENT RIGHT CARDIAC LUNG LOBE TORSION IN AN AFGHAN HOUND

JUNE H. WILLIAMS* AND N.M. DUNCAN**

ABSTRACT: Williams June H.; Duncan N.M. **Chylothorax with concurrent right cardiac lung lobe torsion in an Afghan hound.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 35-37 (En). Department of Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 P.O. Medunsa, Republic of South Africa.

The incidence, methods of diagnosis and treatment of lung lobe torsion in dogs are briefly reviewed. A case of chylothorax with subsequent right cardiac lobe torsion in a young male Afghan hound is described.

Key words: Chylothorax, lung lobe torsion, right cardiac lung lobe, Afghan hound.

INTRODUCTION

Torsion of the lung lobe is a rare condition in man[4,5,9,10,12] and also occurs rarely in dogs and cats[1-3,6-8,11,13]. It has been reported more frequently in dogs[1,6,7,11,13] than cats[2,3,8,13]. The right cardiac lobe is most frequently involved in dogs, accounting for 13 of 19 cases reported in the literature. Of the 13, two had simultaneous torsion of a second lung lobe: one involving the left apical cardiac lobe and the other the right apical lobe. The other 6 cases involved the right apical lobe alone (2 cases), the left apical lobe (2 cases), the left apical and cardiac lobes (1 case) and a single case of diaphragmatic lobe torsion.

The right middle lobe is most commonly affected, possibly due to its narrow shape, poor fixation and location between other fairly mobile structures (beating heart, right cranial lobe, thoracic wall). Large, deep-chested dogs and especially Afghan hounds and Borzois are predisposed to lung lobe torsion, which may be associated with various aetiologies such as trauma, herniation, post-surgical torsion, pleural effusion of various natures, or it may occur spontaneously[14]. Pleural effusion causes compression atelectasis and subsequent instability of the lung lobes and this predisposes to torsion[14].

The clinical signs of lung lobe torsion are non-specific and may include varying degrees of tachypnoea, dyspnoea, fever, depression, anorexia, vomition, pale mucous membranes, and a dry cough. Haemoptysis is seen in dogs but not in cats. Clinical signs prior to the onset of torsion which are related to the inciting cause, such as pleuritis, chylothorax, trauma and surgery may be observed[14]. Chest radiography generally shows pleural effusion with consolidation and altered position and shape of the twisted lung lobe, which is engorged with blood. In the early stages only small air bronchograms running in the opposite direction to normal, may be seen. Bronchography may be used to confirm diagnosis of lung lobe torsion: the contrast medium does not pass beyond the twist and hence occlusion of the bronchus of the offending lobe[14].

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For effective surgical treatment, early diagnosis is necessary based on history, clinical signs, chest radiography, bronchography and thoracocentesis[14].

The present case report concerns a male Afghan hound which was initially presented with chylothorax, and which subsequently developed a 180° torsion of the right cardiac lung lobe. The diagnosis was made on autopsy after the animal had suddenly deteriorated and euthenasia was performed.

CASE REPORT

A blue, entire, 18-month-old male Afghan hound weighing 21 kg was presented at the Medunsa Animal Hospital with the chief complaint being a non-painful, chronically swollen (previous 6 months) right hind leg in the area of the tibiotarsal joint. The owner also complained of progressive, gradual weight loss over the same time period and had noticed that the dog's breathing had become slightly laboured over a period of 5 days. According to the owner, her dogs played very roughly with each other and frequently knocked each other over. This dog had last been dewormed 3 months previously and had been adequately vaccinated.

CLINICAL EXAMINATION

On examination, the dog's temperature was normal, and his heart rate was 150/minute, regular, but distant and displaced upwards on ausculation. In place of the usual apical impulse only a "thrill" was palpable on the left latero-ventral chest despite his thin condition. The pulse in both femoral arteries was weak and thready. Lung sounds on both sides of the chest were very dull – only dorsally could air movement be auscultated. Respiration rate was 44/minute, regular, but with inspiratory dyspnoea.

Haematological and blood chemical analyses were performed; the only abnormalities were neutrophilic leukocytosis (white cell count 20,2 $\times$ $10^9/\ell$) with slight eosinophilia (1,2 $\times$ $10^9/\ell$); albumen marginally decreased (22 g/ℓ), and β and α globulins slightly raised (19,5 and 11,1 g/ℓ respectively), indicating acute and chronic tissue damage, respectively.

The right hindleg swelling proved on biopsy and radiography to be a mild chronic inflammatory lesion incidental to the rest of the case.

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A lateral-recumbent thoracic radiograph showed an almost diffuse, ground-glass appearance with obliteration of the cardiac and diaphragmatic silhouettes.

Thoracocentesis was attempted on the right-hand side between ribs 5 and 6, approximately mid-thoracic region. Only a small amount of blood was obtained which was normal circulating blood on smear.

A barium-meal after the dog had been starved did not show up any diaphragmatic herniation. An exploratory laparotomy was performed to exclude the possibility of liver lobe herniation. The abdominal organs appeared normal, and the diaphragm was intact but bulged markedly into the abdomen. Two litres of opaque pinkish-coloured fluid were withdrawn by aspiration via the diaphragm and submitted for cytology, bacterial culture and chemical analysis. Stained smears of the fluid (RapiDiff, Clinical Sciences Diagnostics) revealed numerous red cells, a few neutrophils, and several macrophages laden with clear, small, round, cytoplasmic vacuoles. The fluid clotted on exposure to glass and no bacterial growth was obtained. The enzyme values (blood urea nitrogen, creatinine, alkaline phosphatase, aspartate transaminase, alanine transaminase, lactic dehydrogenase and creatine kinase) were all within normal range but total protein was lower than normal serum values (38 g/ℓ), the albumen was low (18 g/ℓ), inorganic phosphorous higher than the normal serum values (2,00 mmol/ℓ), and the rise in α and β globulins which occurred in the serum, also occurred in the thoracic effusion. The vacuoles in the macrophages on light microscopic examination of smears resembled fat globules although this was not confirmed. It was noted, however, that when the smears were placed in the alcohol fixative of the Rapidiff (Clinical Sciences Diagnostics), all of the previously apparent opaque substrate of the smears disappeared and only the cells remained. A tentative diagnosis of chylothorax was made in the light of the nature of the pleural effusion and the fact that the substrate of the effusion dissolved and disappeared in alcohol suggesting fat.

On the fourth day post-laparotomy due to progressively worsening dyspnoea, the chest was once more drained using a Braunule i.v. cannula (Remedia Medical) with the dog in a standing position. Urinalysis (Multistix, Ames Division of Miles Laboratories Ltd.) at that stage showed normal concentrated urine. The animal ate well, his habitus was good to excellent and he barked a lot at other dogs. The respiration rate of 42/minute after the latest drainage increased gradually daily until 11 days later (habitus and appetite remained very good) when chest drainage had to be repeated; the fluid was still apparently chylous.

The owner took the dog home for 2 days with instructions to feed a fat-free diet with lean meat (little and often) as the main protein source, but they returned due to the development of anorexia, dyspnoea and splenomegaly. No parasites were seen on bloodsmear and haematology revealed no abnormalities except a left shift in neutrophils (white cell count 11,3 $\times$ $10^9/\ell$ with mature neutrophils 51,9 $\times$ $10^9/\ell$ and immature neutrophils 37,3 $\times$ $10^9/\ell$), a lymphocytopaenia (0,56 $\times$ $10^9/\ell$) and eosinophilia (15,8 $\times$ $10^9/\ell$). The dog's appetite returned to normal the day after readmission, he was still dyspnoeic but had a normal temperature.

Suddenly on day 6 after readmission, the temperature rose to 40°C, the dyspnoea became severe and a bloodsmear showed a marked neutrophilia. The spleen remained enlarged. Unsuccessful attempts were made to drain fluid from the thorax; the little that was obtained, was blood-stained. The dog became anorexic and depressed and euthenasia was decided upon.

TREATMENT

A course of oral co-trimoxazole (Purbac tablets, National Health Products, Lennons Ltd.) was initiated on admission in case of possible pneumonia. The dog was then put onto a course of doxycycline hyclate (Doxyvet, Milvet, Pietermaritzburg) at 10 mg/kg for 14 days to prevent bacterial infection and also prophylactically in case of *Ehrlichia canis* infection. Multivitamin tablets (Beefee, Centaur) were fed daily. Thereafter treatment was changed to amoxycillin trihydrate (Clamoxyl, Beechams) and proteolytic enzymes from carica papaya (Tromasin S.A. tablets, Warner) daily, the latter in an attempt to reduce the effusion. On return to the hospital after being at home for 2 days, the dog was once more dosed with doxycycline daily.

AUTOPSY

At autopsy an acute 180° clockwise torsion of the cardiac lobe of the right lung with severe pleural effusion (serous-like blood-stained, volume approximately 3 – 4 litres), and mild chyloabdomen were found.

The twisted lobe was increased in size by 2 to 3 times, with a dark red-black, liver-like appearance due to congestion, and was undergoing necrosis. It was very friable on palpation. The left lung lobes were moderately atelectatic from pressure caused by the pleural effusion.

DISCUSSION

If a diagnosis of lung lobe torsion is made by chest radiography and/or bronchography, it is possible to intercede as soon as possible and perform a lobectomy of the twisted lobe.

Post-surgical prognosis is given as "fair" but depends on the underlying or initiating condition. Chylothorax and pleuritis may complicate post-surgical recovery[14].

In the case recounted here, it is unlikely that air bronchograms would have been visible due to the atelectasis and severe hydro- and chylothorax; however, the chances of torsion occurring were high due to the predisposing factors being present viz. deep, narrow chest conformation and a pleural effusion. Unfortunately, bronchography was not performed. The change in nature of the effusion to become blood-stained should be taken as an alerting factor that torsion of a lobe may have occurred.

Differential diagnoses of lung lobe torsion include uncomplicated pleural effusions, compression atelectasis, pneumonia, embolisation and thrombosis, diaphragmatic hernia, post-operative pleural haemorrhage, acute trauma, neoplasia, and warfarin poisoning or other coagulopathies[14].

Chylothorax occurs infrequently, and often is of obscure origin and progressive and uncontrollable nature in dogs and cats[15]. The effusion consists of a mixture of lymph of thoracic and intestinal origin. Causes of chylothorax include trauma, diaphragmatic

hernia, chest or diaphragmatic surgery plus other non-traumatic congenital or acquired causes[15].

The origin of the chylothorax in the case presented was unsure but possibly triggered off by previous trauma – the owner had mentioned the rough play of her dogs. No break in the thoracic duct was found at post mortem; this is apparently often the case in Afghan hounds, Borzois and other breeds with deep, narrow chest conformation[15].

Diagnosis of chylothorax is made on examination of thoracic fluid obtained by thoracocentesis – the fluid is characteristically odourless and milky. Diagnosis is confirmed by microscopic examination of fluid stained with Sudan III to positively identify the chylomicrons, or by dissolution of the fat in ether. In the case report here, the Sudan III test was not performed but the fixative of the Rapidiff (Clinical Sciences Diagnostics) dissolved the opaque substrate on the smears leaving only the cells and this was taken as evidence of the presence of fat.

Spontaneous healing has been recorded in dogs and cats, and attempts at inducing adhesion between visceral and parietal pleurae to limit the space for fluid accumulation has only had limited success[15]. Surgery to attempt to ligate the thoracic duct and its branches was not attempted in the case reported here – conservative treatment only was applied viz. chest-drainage and dietary restrictions. Secondary bacterial infections are rare due to the inherent bacteriostatic effects of the lecithin components of chyle[15].

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ABSTRACT

SAMEVATTING

THE DETECTION OF ANTIBODIES TO *COWDRIA RUMINANTIUM* IN SERUM AND *C. RUMINANTIUM* ANTIGEN IN *AMBLYOMMA HEBRAEUM* BY AN ENZYME-LINKED IMMUNOSORBENT ASSAY

A sensitive and reliable enzyme-linked immunosorbent assay for the detection of antibodies to *Cowdria ruminantium* in serum and *C. ruminantium* antigen in *Amblyomma hebraeum* nymphae is described. For the screening of antibodies *C. ruminantium* from *A. hebracum* nymphae, partially purified by wheat-germ lectin affinity chromatography, was used as antigen. To screen nymph populations, sera from either Ball 3 strain-infected sheep or Kumm-strain infected mice were used. By using appropriate controls the assays were rendered specific with respect to *C. ruminantium*. (Neitz, A.W.H., Viljoen, G.J., Bezuidenhout, J.D., Oberem, P.T., Van Wyngaardt, W. & Vermeulen, N.M.J. 1986. The detection of antigen to *Cowdria ruminantium* in serum and *C. ruminantium* antigen in *Amblyomma hebraerum* by an enzyme-linked immunosorbent assay. *Onderstepoort Journal of Veterinary Research*, **53**, 39–41 (1986).)

ABSTRACT

SAMEVATTING

RESPONSE OF SHEEP AND CATTLE TO COMBINED POLYVALENT *PASTEURELLA HAEMOLYTICA* VACCINES

In sheep, certain oil adjuvant vaccines gave rise to a better antibody response to *P. haemolytica* than an $Al(OH)_3$-adsorbed vaccine. This finding, however, was not consistent for all serotypes, and with respect to *P. multocida*, oil adjuvants had no advantage. Furthermore, it was found that the removal of all the culture supernatent fluid during the production process had no deleterious effect on the antigenicity of the product.

In cattle, good responses were obtained with both alum-precipitated and $Al(OH)_3$-adsorbed vaccine where all culture supernatent fluid was not removed during the production process. No advantage was gained with oil emulsion vaccines.

The degree of immunity afforded to mice and the antibody response to different serotypes of *P. haemolytica* varied considerably. Further detailed studies with respect to specific serotypes of *P. haemolytica* are therefore required. (Cameron, C.M. & Bester, Faith, J., 1986. Response of sheep and cattle to combined polyvalent *Pasteurella haemolytica* vaccines. *Onderstepoort Journal of Veterinary Research*, **53**, 1–7 (1986).

ABSTRACT

SAMEVATTING

THE EPIDEMIOLOGY OF *PARAFILARIA BOVICOLA* IN THE TRANSVAAL BUSHVELD OF SOUTH AFRICA

A total of 20 375 flies collected off cattle on 12 farms over 36 months were identified and examined for 3rd stage *P. bovicola*. The 3 vector species accounted for 64,1% of the flies collected and were the only fly species found to be infected. *Musca lusoria* was clearly the dominant vector fly, although large numbers of *Musca* sp. A appeared regularly between February and April each year. This phenomenon, coupled with high numbers of *M. lusoria* throughout most of the year, led to an increase in the numbers of vector flies from their lowest level in June to a peak in February–April.

Of the 13 070 vector flies examined for 3rd stage larvae only 64 (0,52%) were positive; of these 41 were *M. lusoria* and 17 *Musca* sp. A. No positive male flies were found. Incubation of wild-caught flies for up to 13 days at 27°C noticeably increased the larval recovery rate. Flies were found to be infected mainly from August–March. Infected *M. lusoria* were recorded from July–March and infected *Musca* sp. A from January–May. Only 6 infected *M. xanthomelas* were collected and this was during the period August–December, when most ovipositional blood spots occur on cattle.

It is concluded that *P. bovicola* transmission in the Bushveld is not correlated with peak periods of bleeding but rather with high numbers of vector flies, the various species augmenting each other so that transmission may take place almost throughout the year. (Nevill, E.M., 1985. The epidemiology of *Parafilaria bovicola* in the Transvaal Bushveld of South Africa. *Onderstepoort Journal of Veterinary Research*, **52**, 261–267 (1985).)

SHORT COMMUNICATION **KORT BERIG**

PRELIMINARY INVESTIGATION INTO THE NUTRITION OF OSTRICH CHICKS (*STRUTHIO CAMELUS*) UNDER INTENSIVE CONDITIONS

G.C.M. GANDINI, R.E.J. BURROUGHS and H. EBEDES*

ABSTRACT: Gandini G.C.M.; Burroughs R.E.J.; Ebedes H. **Preliminary investigation into the nutrition of ostrich chicks (*Struthio camelus*) under intensive conditions.** *Journal of the South African Veterinary Association* (1986) 57 No. 1, 39-42 (En). National Zoological Gardens of South Africa, P.O. Box 754, 0001 Pretoria, Republic of South Africa.

Twenty ostrich chicks (*Struthio camelus*) up to 8 weeks of age were fed isocaloric diets containing protein levels of 14%, 16%, 18% and 20%. The highest mean body weight gain was obtained from feeding the 20% protein diet; however, this result was not significantly different at the 0,05 level of probability. Feed conversion favoured the 18% protein group. During the seventh and eighth week of the experimental period some chicks developed leg deformities. Clinical signs, radiological findings and response to calcium supplementation suggested an insufficient amount of calcium in the experimental diets.

Key words: Ostrich chicks, nutrition, leg deformities, *Struthio camelus*.

INTRODUCTION

High mortalities and poor growth rate in ostrich chicks (*Struthio camelus*) of up to 60 days of age was experienced at an ostrich farm. A preliminary investigation (unpublished data) suggested that the poor growth rate was of nutritional origin. Leg deformities which also occurred in some of the chickens, may have been nutritionally related.

A literature survey revealed a paucity of information on these matters. Literature concerning the nutrition of ostriches under farming conditions in South Africa was published earlier this century[3] and more recently by Smit[9].

A variety of diets with protein levels ranging from 17% to 25% and higher, are recorded for ratites from zoos outside Southern Africa[4,6,7] (H. Assink, K. Bleyenberg, J. Nijboer 1982 Efficiency in the management of feeding programmes in the Royal Rotterdam Zoological and Botanical Gardens, Unpublished Report), but no data is given about growth rate and feed consumption. No information is available with regard to South African conditions.

This preliminary investigation was undertaken to examine the efficiency of feed utilization and effects on body mass of commercially prepared diets with 4 different protein levels on ostrich chickens under local conditions.

MATERIALS AND METHODS

Ostrich chickens (n = 20), 8 – 10 days old, of the South African domesticated variety, were randomly divided into 4 groups. The birds were kept in outside pens 3 × 2,5 metres in size and housed at night. Each group was assigned to a different dietary regime for an experimental period of 8 weeks.

The rations consisted of 4 isocaloric diets containing either 14%, 16%, 18% or 20% protein (Table 1). Summit-dilution technique was used in mixing the feeds for the 4 treatments. Summit was formulated with 20% protein and a dilution feed with 10% was mixed proportionately with the summit so as to produce the 18%, 16% and 14% protein treatments.

The rations, in the form of a meal, and water were fed ad lib. Finely chopped green lucerne was supplied daily as a feeding stimulus and insoluble grit was offered to each group throughout the trial. A total of 14,4 kg of green lucerne was fed during the 8-week period to each group. The following water-soluble antibiotic and vitamin preparations were added as a prophyllactic measure to the drinking water of each group during the specified periods: first week – Tylosin, erythromycin, furaltadone and vitamin mixture (Tylo-TAD plus, TAD Pharmazeutisches Werk GMBH) and Vit. B complex, glucose and protein mixture (Equistress, Centaur Labs. (Pty) Ltd.); second week – Trimethoprim and triple sulfa combination (Trimeto-TAD, TAD Pharmazeutisches Werk GMBH); sixth week – Tetracycline (Tetracycline Soluble Powder, Glaxo (Pty) Ltd.).

Individual body mass was recorded at the beginning of the trial. Thereafter, individual body mass and group feed consumption were recorded weekly. The feed conversion ratio was calculated as the amount of meal consumed (g) divided by body mass gain (g).

The statistical significance of the differences of growth rate among the dietary treatments were determined by the use of the analysis of variance at the significance level of 5%. A general clinical examination was carried out once a week. Radiographic examination of the legs and vertebral column was performed on 5 chicks which developed leg deformities during the sixth and seventh week. A pooled faecal sample from each group was examined at the fifth and eighth week, macroscopically and microscopically, the latter by the technique of concentration by flotation, to determine the presence of internal parasites.

At the end of the 8-week test period the 20 chicks were moved into a larger common enclosure and fed the 18% protein diet. Bone meal was added in order to raise the dietary calcium level to approximately 2,5%. Calcium borogluconate (Calcium Borogluconate, Maybaker (Pty) Ltd.) at a dosage rate of 100 mg/kg b.m. was administered once orally to 2 chicks which radiographically showed severe bone lesions. Additional radiogra-

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Table 1. Composition of experimental diets

		Diet (percent protein)			
		14	16	18	20
Ingredients		%	%	%	%
Yellow maize meal		58,58	55,62	52,66	49,70
Grain sorghum		10,00	10,00	10,00	10,00
Wheat bran		3,48	2,32	1,16	–
Lucerne meal		10,50	10,50	10,50	10,50
Soya oil cake		1,08	1,62	2,16	2,70
Sunflower oil cake		6,00	9,00	12,00	15,00
Fish meal		2,60	3,90	5,20	6,50
Blood meal		0,50	0,75	1,00	1,25
Bone meal		2,48	2,52	2,56	2,60
Calcium mono-phosphate		0,66	0,44	0,22	–
Limestone		1,29	1,26	1,23	1,20
Salt		0,34	0,31	0,28	0,25
Bagasse		2,01	1,34	0,67	–
Lysine		0,144	0,096	0,048	–
Methionine		0,024	0,016	0,008	–
Endox		0,0125	0,0125	0,0125	0,0125
Mould curb		0,05	0,05	0,05	0,05
Amprol plus		0,05	0,05	0,05	0,05
Vitamin premix		0,18	0,18	0,18	0,18
Mineral premix		0,15	0,15	0,15	0,15
Calculated analysis					
M.E.	Mj/kg	11,30	11,30	11,30	11,30
Methionine	g/kg	2,90	3,30	3,70	4,10
Total Sulphur Amino Acids	g/kg	5,70	6,30	7,00	7,60
Lysine	g/kg	7,00	8,00	9,00	9,90
Tryptophan	g/kg	1,80	2,10	2,40	2,60
Arginine	g/kg	7,80	9,30	10,80	12,30
Isoleucine	g/kg	4,70	5,50	6,30	7,20
Threonine	g/kg	6,20	7,00	7,80	8,70
Fibre	g/kg	68,50	68,50	68,50	68,50
Ca	g/kg	14,00	14,00	14,00	14,00
P Total	g/kg	7,00	7,00	7,00	7,00
P available	g/kg	5,00	5,00	5,00	5,00
Vit. A	I.U./kg	12 785	12 845	12 905	12 970
Vit. E	I.U./kg	50,50	49,60	48,50	47,00
Vit. D_3	I.U./kg	3 000	3 000	3 000	3 000
Vit. K	p.p.m.	2,25	2,25	2,25	2,25
Thiamine	p.p.m.	4,60	4,40	4,20	4,10
Riboflavine	p.p.m.	14,20	14,30	14,40	14,50
Pantothenic acid	p.p.m.	19,50	19,10	18,80	18,40
Niacin	p.p.m.	91,40	95,00	98,00	101,00
Biotin	p.p.m.	0,20	0,19	0,18	0,18
Folic acid	p.p.m.	2,04	2,03	2,00	2,00
Choline	p.p.m.	1 190	1 310	1 430	1 550
Vit. B_{12}	p.p.m.	0,004	0,006	0,008	0,01
Sodium	g/kg	3,30	3,30	3,20	3,40
Potassium	g/kg	5,40	5,40	5,40	5,40
Mg	g/kg	2,00	2,10	2,30	2,40
S	g/kg	1,00	0,90	0,90	0,90
Mn	p.p.m.	91,50	91,00	90,00	90,00
Fe	p.p.m.	95,00	150,00	132,00	137,00
Cu	p.p.m.	10,70	11,00	11,00	11,00
Zn	p.p.m.	75,60	76,00	77,00	78,00
Se	p.p.m.	0,23	0,25	0,27	0,29
Iodine	p.p.m.	3,00	3,00	3,00	3,00

phic examination of these 2 chicks, and a clinical examination of all the birds were done during a period of 3 weeks after the trial period.

RESULTS

The mean body mass of the 14%, 16%, 18% and 20% protein groups at the beginning of the trial was 912 (S.D. = 38,3), 960 (S.D. = 41,8), 826 (S.D. = 68,0), and 876 (S.D. = 91,5) g, respectively. The data presented in Fig. 1 show the pattern of growth during the 8-week test period. This demonstrates that the growth rates of the 3 higher protein groups, i.e. 20%, 18% and 16%, are similar and noticeably higher than that of the 14% protein group.

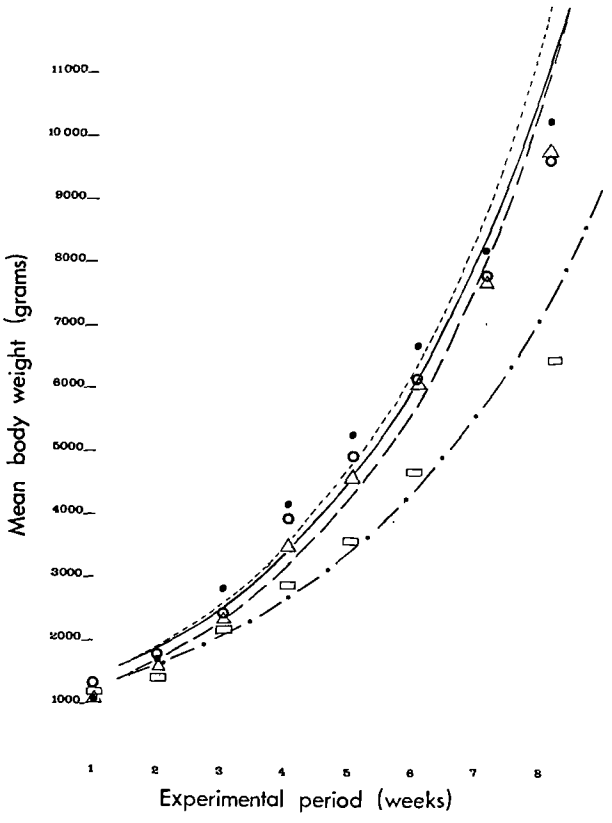

Fig. 1: Effect of feeding different dietary protein levels on mean body weight of ostrich chicks:
14% (□) ___ . ___ . ___; 16% (○) _____;
18% (Δ) ___ ___ ___; 20% (●) _ _ _ _ _

Table 2 shows average body mass, average body mass gains, feed consumption and feed conversion at the end of the 8-week trial period for each dietary regime. The highest mean body mass gain (9 134 g) was obtained from feeding with 20% protein diet. The ostrich chickens fed the 14% protein ration showed the lowest mean body mass gain (5 438 g). Birds fed the 16% and 18% protein rations averaged a body mass gain of 8 440 g and 8 754 g, respectively.

There were no significant differences ($P = 0,2565$) for the mean body mass gains among the groups of birds fed the 20%, 18%, 16% and 14% protein levels at the end of the trial period; a significant difference ($P = 0,0413$) was found when the mean body mass gain of the 15 birds from the 20%, 18% and 16% protein groups was compared with the mean body mass gain of the 14% protein group. Those birds on 18% protein had the most efficient feed conversion ratio (1,65). The 20%, 16% and 14% protein groups showed respective ratios that were 2,4%, 4,2% and 32,7% higher than the 18% protein group.

Body mass gain is the result of the total ration fed, i.e. meal and lucerne, but the feed conversion ratio calculated takes into account only the amount of meal fed. In relation to the amount of meal fed, the quantity of lucerne given is minimal. On a dry matter basis this was

Table 2: Effect of feeding different dietary protein levels on growth, feed consumption and feed utilization of ostrich chicks (at eight weeks)

Protein %	n	Body mass (g)			Body mass gain (g)			Total meal consumed (g/group)	Feed conversion* (g feed /g gain)
		mean	S D	range	mean	S D	range		
14	5	6 350	1 518,6	4 550 – 8 000	5 438	1 539	3 650 – 7 140	59 560	2,19
16	5	9 400	4 516	5 800 – 12 000	8 440	2 506,8	4 900 – 11 000	72 815	1,72
18	5	9 580	2 924	7 200 – 14 200	8 754	2 957	6 270 – 13 400	72 340	1,65
20	5	10 010	4 672	5 000 – 15 750	9 134	4 605,9	4 270 – 14 800	77 265	1,69

*The amount of fresh lucerne is not taken into account.
SD = Standard deviation

3,3%, 3,6%, 3,5% and 4,3% for the 20%, 18%, 16% and 14% rations, respectively.

During the sixth and seventh week of the test period, 5 chicks (3 from the 20% protein group and one from each of the 16% and 14% group) developed enlargement of the hocks and/or bowing of the tarsometatarsus. Radiographically, increased width and poor and irregular calcification of the proximal epiphyseal plate of tarsometatarsus was evident, with a concommitant widening of the metaphysis (Fig. 2). The severity of the lesions varied among the birds. Mono- or bilateral bowing of the tarsometatarsus was also evident in 4 chicks. No deformities of the vertebral column were seen on radiographic examination.

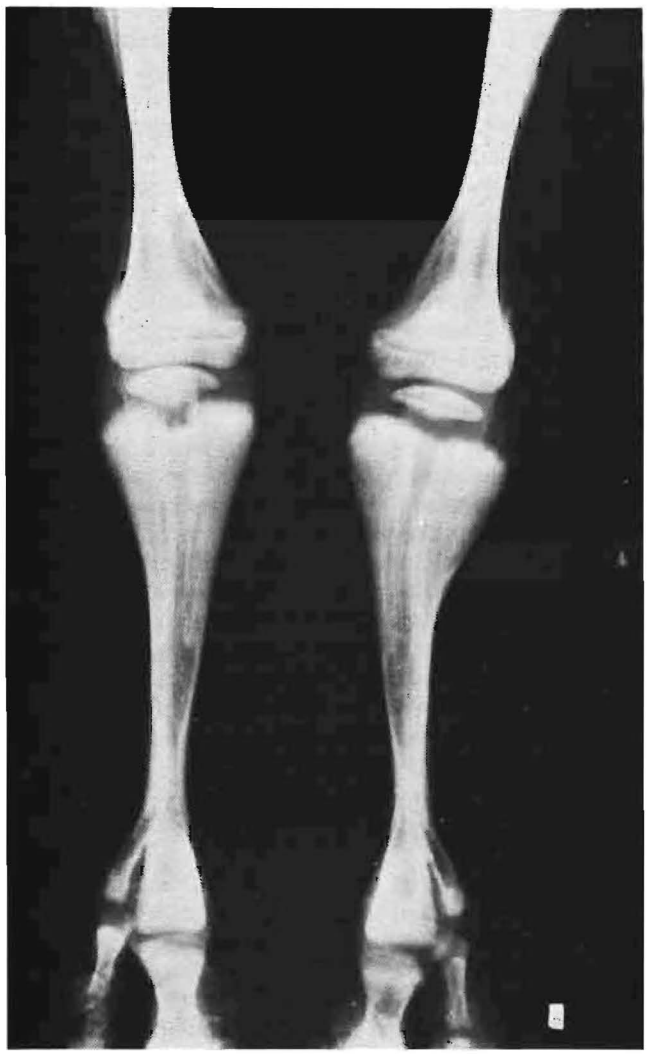

Fig. 2: Radiograph of the tibiotarso-tarsometatarsal articulation of a nine-week-old ostrich chick with metabolic bone disease. Increased width and poor and irregular calcification of the epiphyseal plate, widening of the metaphysis and bowing of the tarsometatarsus is evident.

During the 3 weeks after the trial period, no aggravation of lesion was noticed; radiological examination of the birds treated with calcium borogluconate showed increased calcification of the proximal tarsometatarsal epiphyseal plate.

DISCUSSION

A diet with a protein level ranging from 16% to 20% and a metabolizable energy of 11,3 Mj/kg seems to produce reasonable mean body mass gain and feed conversion up to two months of age.

The mean body mass gain of the 14% protein group was significantly lower than the mean body mass gain of the 15 birds from the 16%, 18% and 20% protein groups. It must, however, be emphasized that this result is based on a stratified random sample rather than a true population sample.

Feed conversion was noticeably higher in the 14% group. The data would suggest that protein levels lower than 16% are not economically feasible for the first few weeks of growth. However, the small sample size and the probable interference of the effects of the suspected mineral imbalance, make conclusions difficult.

Further investigations with a higher protein level, different calorie to protein ratios and different amino acid profiles of the diet are required to resolve these issues.

Leg deformities and lesions typical of rickets, osteomalacia and porosis have been described in ostriches and other ratites raised in zoological gardens[1,2,10]. Although leg deformities are recognised under South African ostrich farming conditions and colloquially described as "twisted and bent legs", "enlarged hocks", "slipped tendons" and "leg weakness", no radiological or histopathological data have been published. On radiographic examination the avian tibiotarsus-tarsometatarsus affected by juvenile metabolic bone disease shows increased width and failure of calcification of the epiphyseal plate, increased trabeculation and widening of the metaphysis and bowing of the bones[5]. To reduce the incidence of rickets in ratites, dietary calcium levels of 1,5 to 2,5%, phosphorus levels of 1 to 1,5%, the presence of Vit. D_3 in the diet and a feeding regime to prevent excessively rapid growth, have been suggested[2].

The amount of calcium, total phosphorus and Vit. D_3 contained in each diet tested was 1,4%, 0,7% and 3 000 I.U./kg, respectively. The clinical signs, radiological

findings and positive response to calcium administration suggest that the metabolic bone disease in the chicks may be related to feeding of an insufficient amount of dietary calcium. An analysis of the occurrence of metabolic bone disease in relation to growth rate is not possible because of the limited amount of data.

The dietary levels of Ca, P, Vit. D_3, the oligoelements and vitamins (manganese, zinc, Vit. E, pantothenic acid, nicotinic acid, Vit. B_6, biotin, Vit. B_{12} and choline) reported by Scott et al.[8] as possible aetiological factors in bone deformities in poultry, should be investigated further with respect to ostriches. In future investigations, attention should also be given to management practices such as type of housing, amount of sunlight and exercise.

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REVIEW OORSIG

FLEA CONTROL ON PETS IN SOUTHERN AFRICA

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ABSTRACT: Briggs O.M. **Flea control on pets in Southern Africa** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 43-47 (En) St. Francis Veterinary Hospital, 157 Main Road, 7800 Heathfield, Republic of South Africa.

Aspects of the biology and life-cycle of the flea which are important to the practising veterinarian are discussed. The "cat flea", *Ctenocephalides files* is the most prevalent species on dogs and cats in those parts of the world where it has been surveyed. Whether the flea is a temporary or permanent obligatory parasite is still a controversy. The insecticides and product formulations available for flea control in South Africa are reviewed with emphasis on the systematic agent, fenthion. Practical protocols for flea control on dogs and cats are proposed.

Key words: *Ctenocephalides felis*, flea control, insecticides.

The flea is the most common parasite found on cats and dogs along the coast of Southern Africa. These areas have been compared to the Southern United States where it has been stated that flea-bite allergy is the most frequent cause of skin conditions in pets[25] and indeed the most common cause for the presentation of dogs to the Veterinarian[24]. Once a dog is allergic to flea saliva[6], prolonged, severe pruritus ensues[33] which can prove refractory to hyposensitization[13 21]. The side effects from long term corticosteroid therapy[20 22] intensifies the need for flea control. Fleas, however, are not always seen by clients on their pets, and it may prove exasperating to explain the aetiology of the pruritus and alopecia.

The absolute control of fleas is often elusive and requires constant attention. However, the goal is necessary since besides causing flea-allergy dermatitis, fleas carry the common cat and dog tapeworm, *Dipylidium caninum*[29 49], which is also an occasional parasite of man[48]. The cat and dog fleas are also the chief cause of the intense pruritus seen in children sensitized to flea bites[48]. Occasionally, fleas serve as vectors for plague and murine typhus[42]. The presence of fleas on pets indicates an insufficient ectoparasite control regime in that the pet is susceptible to cheyletiellosis, pediculosis, otodectic otitis, scabies, and even babesiosis from ixodid infestation. In order to adequately advise the public on flea control, the practitioner requires a basic knowledge of the life-cycle and biology of the flea and the formulations and toxic effects of the available insecticidal agents.

BIOLOGY AND LIFE-CYCLE (Fig. 1)

Egg

Gravid females usually jump off the pet and lay eggs in cracks or crevices in the floor of the house[48]. However, sometimes eggs are laid on the pet and can just be visible to the naked eye[44 48]. These fall off the pet[29] and may appear as tiny, glistening white specks on the examination table – often amongst other evidence of infestation such as flea faeces and epidermal scales. The characteristic "pop" elicited by bursting the egg gently with a fingernail impresses the pet owner no less than the demonstration of haemoglobin on dissolving the comma-shaped flea faeces in a drop of water.

Larva and pupa

Larvae are active and avoid light by remaining in cracks and crevices[29]. The cocoons are formed in crevices and become camouflaged by dirt and dust[48]. These stages thus may develop in the house without the owner being aware. It is also the larval and pupal stages which are temperature and humidity sensitive resulting in fleas being more common in coastal areas. Ideal relative humidity is 70 – 80%[25] and ideal temperature is 18 – 35°C[34]. Altitude is another factor (ideal is less than 1 800 m[44]) resulting in the paucity of fleas in households on the Witwatersrand.

Adult

The adult flea is a brown, laterally compressed, wingless, insect[48]. The head with it's conspicuous eyes is used to differentiate between the "cat" flea, *Ctenocephalides felis* and the "dog" flea, *Ctenocephalides canis*[49]. *C. felis* is the most common species found on dogs in the United Kingdom[18], the United States[25], Puerto Rico[15], the Western Cape (O.M. Briggs, unpublished data) and the Transvaal[28]. Although fleas have been noted to drink water[30 45], the adult must partake of a blood meal to survive[34 38 45].

The adult flea hatches from the cocoon and jumps onto the host using it's specialized, powerful legs[48]. The host's epidermis is penetrated by the flea's maxillae. A tube, the epipharynx, enters the capillary vessels and draws up blood while saliva from the maxillae is deposited in the surrounding tissue[35]. This procedure causes minimal damage to the skin[35]. It is the allergic response to a hapten contained in the flea saliva[45] which results in persistent pruritus[19 23 50] known as flea allergy dermatitis[24 25 44].

Fleas require a period of between two and ten minutes to engorge[45]. This intricate and relatively lengthy procedure occurs most easily at night. On people, fleas bite only the ankles during the day but the whole body at night[48]. It would seem therefore, that the adult fleas leave the host during the day time. However, this clinical observation is not supported by research[3 25]. The controversy[21 25] as to whether the adult is a temporary or permanent obligatory parasite needs to be examined urgently as this strongly influences our approach to flea

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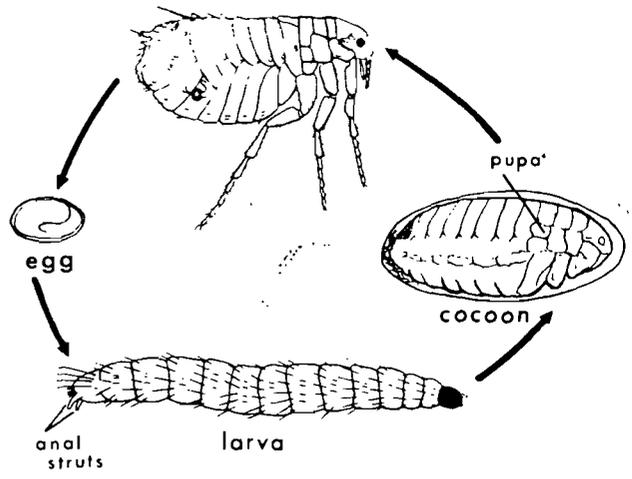

Fig. 1: Diagram of the life-cycle of a flea (from Service[48]). The lengths of the various stages have been given[29] as adult – 200 days; egg – 2 days; larva – 9 to 15 days; pupa – 7 to 21 days. However, under ideal conditions, the life cycle can be completed in as little as 14 days[4].

control[3]. Should environmental conditions not be ideal, or if a host is not available, the adults can remain alive in the cocoons for up to a year[48]. Stimuli to emerge may be either seasonal (increase in humidity[34 48]) or host related (vibration[34], warmth[34], characteristic odour[34] and casting a shadow[34]). This explains why on entering a building which has been vacant for months, man can be attacked by large numbers of blood-thirsty fleas, these being newly hatched adults seeking their first blood meal[48]. Reproduction of the flea is stimulated by oestrogens and corticosteroids in the peripheral circulation of the host[45]. This may explain the individual variation in the extent to which a host attracts fleas[39].

PARASITICIDAL AGENTS

These compounds are widely used and a large number are available in South Africa. They are cholinesterase inhibitors and toxic doses cause acute poisoning of the central nervous system and neuromuscular junctions in mammals with resultant death due to asphyxia[9]. Cats are particularly susceptible[31 44], exhibiting salivation, vomition, diarrhoea and muscle fasciculations on intoxication[5]. Trade names of registered products are listed in the Index of Veterinary Specialities and can also be obtained from the Department of Agriculture[10]. Examples of organophosphates used in flea control are dichlorvos, quinthiophos, fenchlorphos, and malathion. Dichlorvos impregnated collars have been shown to cause both a local cervical dermatitis and a systemic anticholinesterase toxicity in cats[5]. Atropine sulphate and the oximes (Toxogonin and 2-PAM) are antidotal[7].

Carbamates

These non-organophosphate anticholinesterases are less toxic to mammals than the organophosphates[31]. A 5 – 8% carbaryl powder is commonly used against fleas on cats and dogs[44]. Atropine sulphate is advised in the treatment of toxicity and 2-PAM is contra-indicated[7].

Chlorinated hydrocarbons

All the chlorinates are practically insoluble in water and therefore have a long residual action[10]. Some, such as DDT and dieldrin are considered to be virtually indestructable[10] and have been withdrawn. For the same reason the use of the rest of this group has been severely curtailed[31 44]. Lindane (gamma BHC) on its own is not registered for pets[10], however, it is available for use on dogs in combination with amitraz, an insecticide developed for tick control. The main clinical signs of acute intoxication are increased irritability, tremor, followed by tonic-clonic convulsions, which indicate that the principal site of action is the central nervous system[32].

Plant derivatives

These are the oldest pesticides known[10]. Rotenone is derived from the root of Derris (Fam. Leguminosae)[9] and is of low toxicity[7 31]. Pyrethrins are volatile oils of chrysanthemum flowers[31] and are so safe[7 10 44] that they can be used on neonates and even some exotic pets[31]. Pyrethrins are widely used on pets and in premises[10] often in the new microencapsulated form allowing controlled release for residual action[17]. Where a pet owner is averse to the use of pesticides, these botanical derivatives can be offered as a more "natural" alternative. Emesis, gastric lavage, fluid therapy and diazepam or barbiturate control of convulsions are advised in the treatment of toxicity[7].

Pyrethroids

Synthetic pyrethrins are new[10] effective[17 39] flea control compounds of low mammalian toxicity[17]. Examples are allethrin[25], and resmethrin[7 25 44].

Growth regulators

Methoprene is the commonly used growth regulator in the United States[7]. It prevents fourth instar larvae from undergoing metamorphosis into adults[7 44] and is often used in combination with an adulticide on premises as an effective control of both larvae and adults[7 11]. It is of such low toxicity that it is fed to cattle as a fly control remedy[43].

Combinations

Additive effects, potentiation between two or more ingredients and improved formulations will make the use of combinations more attractive. In this way it is possible to a) provide both "knockdown" and residual effect, b) reduce the toxicity of any single component, and c) provide for ease of application. An example of this is the dog collar which combines amitraz against ticks with lindane against fleas. In contrast, for lindane to be effective against both ticks and fleas on its own, it would be so concentrated as to preclude its registration.

PRODUCT FORMULATIONS

Dips and rinses

These have both "knockdown" and residual effect if used every week[31]. The disadvantages (time consuming, odiferous, and frequently expensive)[31] are outweighed by the consistently excellent control of a wide range of ectoparasites. Some dogs develop a dry coat with repeated dipping. A more concentrated, smaller volume can be applied as a "sponge on" to obviate this. Many of these are schedule 4 due to mammalian toxicity and thus not freely available to the public.

Shampoos

The combination of a shampoo with an insecticide is

convenient and aesthetically attractive to pet owners[31]. However, since the insecticide is washed off by the shampoo[44], it has no residual effect and the owner becomes frustrated by the flea control failure.

Powders

These are usually non-toxic and relatively effective if used sufficiently frequently (up to every 48 h)[31]. On neonates and debilitated pets where the owner is motivated to spend time, it is a convenient and effective control method.

Aerosols

These are quick and convenient. However, apart from the microencapsulated aerosols which are not yet available here, they have poor residual action and are even potentially toxic[31].

Microencapsulated products

Small nylon or polyurea capsules contain insecticide for slow, regular release[7]. This method of stabilizing the insecticide provides an exceptionally good residual effect of low oral toxicity[11]. Pyrethrins are encapsulated for use on pets and diazinon for premises.

Oral products

Cythioate is available as a schedule 4 pesticide. In dogs, it is effective[14 47], convenient and safe[47] although too expensive in this country for use in any but the smallest of breeds. It is recommended at half strength for cats and the absence of documented side-effects makes it convenient especially in long-haired cats or those allergic to collars. However, consumer resistance to oral administration of insecticides is a disadvantage. Other oral preparations found to be ineffective are brewer's yeast[4], vitamin B_1[22], ethanolamine[25] and garlic[38].

Impregnated collars

These are still extremely popular due to their ease of application and low mammalian toxicity. However, most owners seeking flea control advice have already found collars ineffective. No collar can provide a large dog with protection over its whole body. For cats and small dogs, frequent collar changes may provide sufficient flea control[16].

Impregnated plastic strips

Plastic permits long-term, and hence safe, release of toxic compounds such as dichlorvos[36]. Although these are primarily for the control of flies, if placed above the whelping area, they provide a safe flea control remedy in neonates[36]. Pleasant packaging has served to reduce the unattractiveness.

Pour-ons

A prepacked concentrated pesticide is applied along the coat from the nose to the tail and down the legs. Suitable volumes and concentrations are available in this country for corresponding sizes. A product containing 4% chlopyrifos and 4% permethrin is registered in South Africa for dogs only and should not be applied more than once a month. The ease of administration (compared with dipping[37]), residual effect and relative safety may make this a popular pesticide.

Spot-on

Insecticide applied topically in small volumes is absorbed percutaneously and secreted onto the skin over a period of time. Currently only fenthion, an organophosphate, is recommended. It is an effective flea control remedy in dogs[2 8 25 26 44 46] and cats[8 44] although Halliwell[25] does not recommend its use in this species. The dose for dogs has been given as 20 mg/kg[8 25 26 41] administered every week[44], every 2 – 3 weeks[25] or every 3 weeks[8 26]. A dose of 60 mg every 3 weeks has been recommended[44] for cats. Recently, Arther & Cox[2] in North America found almost 100% effective flea control for 24 days in dogs at a dose of 8 mg/kg.

Fenthion is registered in South Africa as "Tiguvon spotton" for cattle and "Bayopet spotton" for dogs and cats. Bayopet spotton is marketed in tubes of different concentrations and volumes to provide for species and body mass variations. The correct tube or tubes selected according to these instructions give approximately half the previously recommended dose. The minimum interval between applications is given as 2 – 3 weeks in dogs and 4 weeks in cats. The lower dose and longer interval renders the Bayopet regime safer though possibly less effective than the previously recommended one.

Since Bayopet may not be effective in areas of high flea infestation, veterinarians may find it necessary to continue to dispense the cattle preparation. Veterinarians may sell stock remedies; however, it is a contravention of the law[12] to decant and sell from the original container without the full instructions being transferred. Risks involved in decanting and redispensing the cattle formulation are minimised by careful, correct labelling as for a schedule 4 drug.

Small volumes of this potent insecticide may cause severe toxicity. Cholinesterase levels remain depressed for a long time in a case of toxicity[27 40]. In the event of oral overdose, evacuation of the gastrointestinal tract followed by the administration of activated charcoal orally and atropine and toxogonin intravenously, has been advised[40]. To be cutaneously absorbed, the drug must be placed directly on the skin. Any placed on the hair, is ineffective. Blood levels are maintained for only 24 h[25]. Thereafter it is thought to enter the fat and to be excreted onto the skin surface in sebum[25]. Fenthion is not advised in greyhounds[44] or in pets under 6 months[8 44] or over 12 years[44]. Nor is it advised in pets that are debilitated[25], pregnant[8 44], or nursing[25]. It has been suggested to keep children away from the pet for 24 h after application[25] and to beware of fumes in an enclosed space[25]. Concurrent use of other insecticides should be limited to non-anticholinesterase and non-systemic agents.

Fenthion has the advantage of convenient use[41], prolonged systemic effect[2 8 25 41], and a possible repellant effect against fleas[25]. This latter effect is especially important in preventing flea allergy dermatitis in that the flea is repelled before depositing the hapten contained in its saliva[25]. Repelled fleas may then attack humans in the vicinity to obtain their blood meal[1]. This temporary inconvenience is far outweighed by the benefit of having the normal life cycle so effectively disrupted[41].

PRACTICAL PROTOCOLS

Dogs

a) Large and medium sized breeds

For fleas alone, fenthion is the most convenient. An effective dose in the author's experience is 20 mg/kg every

14 days. Where ticks are also encountered, a pour-on or dip should be used which kills both parasites. Ticks alone are best prevented by dips. Owners must be warned to avoid the use of other anticholinesterase pesticides concurrently with fenthion.

b) Small breeds

Regular changes of insecticide impregnated collars provide a safe and effective flea control regime in the author's opinion. Owners should keep a diary of collar changes and rather change a collar early than rely on manufacturer's claims of efficacy. Regionally, fleas have developed resistance to insecticides[7] and owner's reports assist in finding the best collar for the area.

Cats

a) Short-haired breeds

Frequent, diarized collar changes have been found the most satisfactory flea control method in the author's practice. Clients have to be warned that collars applied too lossely are easily removed by cats. The collar has to fit snugly and the excess cut off. Where a cat is particularly obstinate, it may be necessary to apply a lighter non-impregnated collar until this is ignored and then introduce the insecticidal collar.

b) Long-haired breeds

Flea control can be exasperating in these cats. If collars do not suffice, oral products or spot-on must be used. Warn clients to take the luxuriant hair coat into account when judging the weight. Dips are to be avoided in cats whose strong cleaning instinct may result in oral intoxication.

Pups and kittens

Regular powdering of the mother and her offspring combined with impregnated plastic strips placed near or hung above the whelping area have been found the most effective and convenient in the author's practice.

Environmental control

a) Bedding

Foggers are canned aerosols providing inexpensive, convenient fumigation. Bedding can be placed in a room to be fumigated.

b) Home

However diplomatic the veterinarian has to be, it is essential to broach the subject of indoor flea control. Professional exterminators are able to provide long residual control (up to six months) due to thorough application and penetration of unexposed areas. For many clients, foggers used on a monthly basis are a less expensive and as effective a method. Vacuming cracks, crevices and corners removes the live, partially inactivated and dead parasites.

c) Yard

This is not impossible to treat. Malathion, as a 0,5 to 5% spray or a 4% dust is available at garden centres and co-operatives. Particular attention should be paid to the pet's favourite sleeping spots (often following the sun around the house as the day progresses).

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ABSTRACT

SAMEVATTING

BOVINE LEUKAEMIA VIRUS AND ENZOOTIC BOVINE LEUKOSIS

Infection of bovines with bovine leukaemia virus (BLV) manifests itself in either of two ways: 30 – 70% of carriers develop persistent lymphocytosis (PL), with the viral genome integrated at a large number of different sites in the DNA of the affected B-lymphocytes, without causing any chromosomal abnormalities. Only 0,1 – 10% of carriers develop lymphoid tumours, which also consist of B-lymphocytes. In contrast to PL, however, they are of mono- or oligoclonal origin in terms of the integration site, which is characteristic for each tumour. All cells contain one or more copies of the viral genome, chromosomal aberrations are common and if deletions are present they are invariably found in the 5′-half of the virus DNA sequence. In both types of affected cells transcription is repressed *in vivo*, but transient virus production can be induced *in vitro* and detected by means of syncytia induction or haemagglutination. *In vivo* production of virus in some unknown cell is suggested by the presence of high antibody titres in infected animals, especially against the envelope glycoprotein gp51. This can be detected by various techniques such as immunodiffusion, radioimmune assay or ELISA. Monoclonal antibodies against gp51 have revealed 8 epitopes, 3 of which are recognized by neutralizing antibodies and one by a cytolytic antibody.

The BLV genome, about 9 kb in size, has been cloned, and some of the information obtained on its molecular structure and function is discussed. It codes for at least 4 non-glycosylated and 2 glycoproteins. Of special interest is the recently discovered serological relationship between some of the non-glycosylated proteins and those of the human T-cell leukaemia virus.

The functional role of BLV in leukaemogenesis is largely unknown: The presence of the viral genome seems to be necessary for the maintenance of the transformed state, but not its continuous expression nor an LTR-mediated promotion of transcription of cellular genes. No oncogene is carried by the virus.

Although bovine leukosis is not of major economic importance, its eradication is desirable and feasible in countries with a relatively low incidence, by means of testing and elimination. For endemic situations vaccination would be preferable, and distinct possibilities exist for the development of gp51 based vaccines. (Burny, A., Bruck, C., Cleuter, Y., Couez, D., Deschamps, J., Gregoire, D., Ghysdael, J., Kettmann, R., Mammerickx, M., Marbaix, G. & Portetelle, D., 1985 Bovine leukaemia virus and enzootic bovine leukosis. *Onderstepoort Journal of Veterinary Research,* **52,** 133 – 144 (1985).)

ABSTRACT

SAMEVATTING

THE USE OF A SINGLE COMPLEMENT FIXATION TEST TECHNIQUE IN BOVINE BRUCELLOSIS, JOHNE'S DISEASE, DOURINE, EQUINE PIROPLASMOSIS AND Q FEVER SEROLOGY

The same techniques may be used in the complement fixation test (CFT) for the serological diagnosis of bovine brucellosis, Johne's disease (paratuberculosis), dourine, equine piroplasmosis and Q fever (caused by *Coxiella burnetii*). The reproducibility of results is excellent, falling for the most part within the twofold range and never exceeding the fourfold range. Agreement with other laboratories is excellent (i.e. within twofold) in the case of brucellosis and equine piroplasmosis antibody titres. A good correlation between the occurrence of the disease and serological reactions is found on circumstantial evidence in the cases of dourine, Johne's disease and Q fever. A standard unitage system is used to report the antibody titres found in all the tests. To simplify laboratory protocols, labaratories required to employ the CFT for the diagnosis of these diseases are advised to use a single proven technique in all the tests.

Problems experienced with transient false-positive Johne's disease antibody titres in cattle following on tuberculin (bovine and avian) testing make it advisable to take specimens for the Johne's disease test prior to performing the tuberculin tests. (Herr, S., Huchzermeyer, Hildegard, F.K.A., Te Brugge, Lesley A., Williamson, Catherine C., Roos, J.A. & Schiele, G.J., 1985. The use of a single complement fixation test technique in bovine brucellosis, Johne's disease, dourine, equine piroplasmosis and Q fever serology. *Onderstepoort Journal of Veterinary Research,* **52,** 279 – 282 (1985).)

PULMONARY FUNCTION IN THE HORSE DURING ANAESTHESIA: A REVIEW

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ABSTRACT: Stegmann G.F. **Pulmonary function in the horse during anaesthesia: a review.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 49-53 (En). Department of Surgery, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The effect of abnormal body position on cardiovascular and pulmonary function in the awake and anaesthetised horse is reviewed. Parameters such as pulmonary shunt, lung volumes, blood gases and alveolar-arterial oxygen partial pressure differences are discussed. Withholding food for 24 hours and mechanical ventilation may be used to improve blood gas values associated with abnormal recumbency during anaesthesia. During prolonged recovery, horses should be encouraged to adopt sternal recumbency.

Key words: Equine, lateral recumbency, dorsal recumbency, pulmonary function, anaesthesia.

INTRODUCTION

The problem of pulmonary dysfunction in the horse was identified as a large difference in the partial pressure of oxygen between the alveolus and the arterial blood ($P(A\text{-}a)_{O_2}$). In these observations it was noted that the dysfunction increased from sternal recumbency to the lateral and dorsal positions during anaesthesia[2,3].

During its daily activities the horse spends little time in the lateral position, while the dorsal position is only adopted for a few seconds when these animals roll in sand. For these reasons, the dorsal and less so the lateral position, represent non-physiological body positions. It is only during anaesthesia that the horse is forced to maintain these positions for prolonged periods of time. Sternal recumbency may be adopted during periods of rest and it is also maintained for varying periods during recovery from anaesthesia.

As a result of the temperament and size of the horse, investigations into the effect of body position on cardio-pulmonary function can only be made during anaesthesia. The result of this is that no distinction could be made in the past between the effects of anaesthesia and abnormal body position. Recently, ponies trained to lie in lateral recumbency were used to determine the effects of lateral recumbency on cardio-pulmonary function[5,14].

However, the anaesthetist is still confronted with an anaesthetised horse kept in lateral recumbency for varying periods during clinical anaesthesia. It would have been advantageous to the horse if some of the negative effects on cardio-pulmonary function were the result of the anaesthetic, since then it could have been reduced by modifying the anaesthetic technique.

THE EFFECT OF BODY POSITION ON CARDIAC OUTPUT AND PULMONARY SHUNT

THE CONSCIOUS HORSE

Conscious standing

Cardiac output values were documented in standing horses using the dye dilution technique by Eberley et al.[1] (2 mℓ/kg/stroke) and Hillidge & Lees[6] (1,76 mℓ/kg/stroke). Pulmonary shunt in the conscious animal is considerably lower than values recorded during general anaesthesia. Littlejohn et al.[7] calculated shunt values of 8,8% in standing horses as a percentage of cardiac output. Hall[5] calculated values of 4% and 7% in standing animals. McDonell[10] calculated values between 2,1 – 9,5% and 4 – 7% in standing animals compared to values of 21 – 51% during anaesthesia[10,16].

Conscious lateral recumbency

Cardiac output values were recorded in trained ponies[5] (1,2 mℓ/kg/stroke – the technique used is not noted). No significant change occurred between standing and lateral recumbency. Hall[5] calculated an average increase of 1% in pulmonary shunt from the standing position to lateral recumbency in ponies. Values of 5% and 8% were calculated. Only two experimental animals were used.

THE ANAESTHETISED HORSE

Lateral recumbency

The negative effects of anaesthetic drugs on cardiac output were documented by Gillespie et al.[2] (1,3 mℓ/kg/stroke in mechanically ventilated horses during thoracotomy). Hall et al.[3] calculated 1,39 mℓ/kg/stroke in spontaneously ventilating animals; Hillidge & Lees[6] calculated a value of 1,03 mℓ/kg/stroke. Dye dilution techniques were used. Differences between lateral and dorsal recumbency were not investigated by these authors. Staddon et al.[15] recorded a higher blood supply to the dependent lung compared to the non-dependent lung in lateral recumbency during anaesthesia. Pulmonary shunt values between 19% and 38% (average 32%) were calculated during mechanical ventilation after relaxation with pancuronium and values between 21% and 51% (average 34%) in spontaneously breathing animals[16]. McDonell[10] calculated values between 8,9% and 18% during spontaneous ventilation. Thiopentone was used for induction and halothane-oxygen were used for maintenance of anaesthesia.

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THE EFFECT OF BODY POSITION ON LUNG VOLUMES AND GAS FLOW

THE CONSCIOUS HORSE

Standing

In radiological examinations of the lung, an increase in the lung shadow was noted in horses starved for 24 hours compared to non-starved horses[9]. McDonell[10], using helium dilution techniques, found an increase in the functional residual capacity (FRC) with starvation; sedation reduced the FRC by 13%. Conscious horses in the standing position showed very little change in FRC from animals in the anaesthetised upright and sternal recumbent position[14].

Lateral recumbency

Schatzmann et al.[13] found only non-significant changes to occur in horses sedated with glyceryl guaiacolate ether on the mechanical tilting table after the posture has been changed from the standing to the lateral position. Tidal volume and minute volume as well as expiratory flow rates decreased; the inspiratory flow rate stayed constant[13]. McDonell noted a significant reduction in FRC in cast animals. In these animals thiopentone anaesthesia had little additional effect on FRC. However, during halothane anaesthesia significantly lower FRC values were recorded, compared to thiopentone anaesthesia[10]. In unsedated ponies, trained to lie in lateral recumbency, Hall[5] noted an increase in minute volume due to a significant increase in respiration rate, as well as a significant increase in physiological dead space. This increased respiration rate was not encountered by Rugh et al.[12].

THE ANAESTHETISED HORSE

Standing

Suspended horses in the standing position as well as sternal recumbent animals showed very little change in FRC from awake animals in the same position[14].

Lateral recumbency

Sorenson & Robinson[14] using single breath nitrogen washout curves, noted a decrease in total lung capacity (TLC) and vital lung capacity (VC) when changing position from sternal to lateral and dorsal recumbency. Expiratory reserve volume (ERV) and residual volume (RV) were significantly reduced in lateral recumbency. Changing back to sternal recumbency immediately restored these volumes. The FRC was significantly reduced in this position compared to standing animals, but its value as a percentage of TLC stayed the same as when standing. This also indicated a reduction in lung volume.

McDonell[10] noted a reduced tidal and minute volume, and a significant reduction in FRC. The FRC reduction was less in starved horses. Increasing anaesthetic depth also further reduced FRC. By tilting the table cranially or caudally, thereby shifting abdominal organs, no effects were noted on the FRC. Left lateral recumbency resulted in higher FRC values compared to right lateral recumbency due to the larger right lung lobe. A significantly higher reduction in FRC occurred in halothane anaesthesia compared to intravenous anaesthesia. Anaesthetic time had no influence on FRC values. The reduction in the measured FRC was less than the reduction in the spirometer base line value, indicating a possibility of distal airway closure.

McDonell et al.[9] showed, by means of dorso-ventral radiographs, an increase in the lung shadow in horses starved for 24 hours compared to non-starved horses. No shadow could be shown for the dependent lung, indicating poor ventilation. The diaphragm was displaced in a cranial direction on lateral radiographs compared to the standing animal. Forced inflation of the lung did not improve the shadow. An opacity of the lower lung developed in 20 min. that could not be fully reversed by turning the horse. Alveolar dead space was larger in lateral recumbency compared to dorsal recumbency.

Dorsal recumbency

McDonell et al.[9] noted a decrease in lung shadow due to a lowering of the diaphragm nearest to the vertebral column. FRC reduction as a percentage of TLC was similar to the lateral position. Higher FRC values were found for the dorsal compared to the lateral position. Closing capacity (closing volume plus residual volume) was higher than the FRC in this position, resulting in the closing down of terminal airways. Positive-end-expiratory-pressure of 30 cm H_2O were not sufficient to open these airways. Changing to sternal recumbency restored these volumes[14].

THE EFFECT OF BODY POSITION ON BLOOD GASES AND $P(A\text{-}a)_{O_2}$ DIFFERENCES

THE CONSCIOUS HORSE

Standing

$P(A\text{-}a)_{O_2}$ differences of 2,39 kPa[3] and 1,87 kPa[10] were found in unsedated horses.

Lateral recumbency

In unsedated trained ponies no effects on blood gases were noted. Pa_{O_2} values decreased significantly in heavier compared to lighter ponies, but remained within the normal range for horses. The horses became restless after 20 min. although no physical reason for this could be found[5,12]. Sedated horses on a mechanical table maintained normal Pa_{CO_2} values, while Pa_{O_2} values decreased significantly[13]. Withholding food for 24 hours significantly improved Pa_{O_2} values. Pulmonary vein blood from the caudal lobe always yielded lower P_{O_2} values from the dependent lung[4]. Hall noted a significant increase (from 0,67 kPa to 1,46 kPa) in $P(A\text{-}a)_{O_2}$ difference in one animal[5].

THE ANAESTHETISED HORSE

Lateral recumbency

On induction with an intravenous technique, significant changes were noted. A lowering of Pa_{O_2}, an increase in Pa_{CO_2} and an increase in $P(A\text{-}a)_{O_2}$ difference of 4,92 kPa occurred[3]. Withholding food for 24 hours improved Pa_{O_2} values[10]. The use of an inhalation agent with oxygen as a carrier gas increased Pa_{O_2} and $P(A\text{-}a)_{O_2}$ differences. Mechanical ventilation decreased Pa_{CO_2} and $P(A\text{-}a)_{O_2}$ differences, and increased Pa_{O_2} values[3]. McDonell[10] found a significant positive correlation between FRC and Pa_{O_2} with thiopentone anaesthesia, and a negative correlation between FRC

Table 1: Efect of recumbency on cardio-pulmonary function while awake or under anaesthesia

	Awake		Thiopentone anaesthesia	Halothane-oxygen anaesthesia				Ref
Variable	Sternal	Lateral	Lateral recumbency	Suspended	Sternal recumbency	Lateral recumbency	Dorsal recumbency	
CO	.	0	.	.	.	↓↓↓	.	5,2,6
Pulmonary shunt	.	↑	.	.	.	↑↑↑	.	5, 16, 10
FRC	0	.	↓	0	0	↓↓	↓	14, 10
V_T	.	0	.	.	.	.	.	13, 10
V_M	.	0/↑	.	.	.	.	.	13, 5, 10
ERF	.	0	.	.	.	.	.	13
IFR	.	0	.	.	.	.	.	13
Resp rate	.	↑/0	.	.	.	.	.	5, 12
TLC	.	.	.	.	.	↓	↓	14
VC	.	.	.	.	.	↓	↓	14
ERV	.	.	.	.	.	↓	.	14
RV	.	.	.	.	.	↓	.	14
Pa_{O_2}	.	0/0/↑	↓	.	.	↑	↓	5,12,13,3,11
Pa_{O_2}	.	0/0/0	↑	.	.	↑	↑	5,12,13,3,11
$P(A\text{-}a)_{O_2}$	.	↑	↑	.	.	↑	↑	5, 3, 11

CO cardiac output
FRC functional residual capacity
V_T tidal volume
V_M minute volume
ERF expiratory flow rate
IFR inspiratory flow rate
TLC total lung capacity
ERV expiratory reserve volume
RV residual volume
Pa_O partial pressure of oxygen in arterial blood
Pa_{CO_2} partial pressure of carbon dioxide in arterial blood
$P(A\text{-}a)_{O_2}$ partial pressure difference of oxygen between alveolar capillary

and Pa_{CO_2} differences during halothane-oxygen anaesthesia.

Dorsal recumbency

Lower Pa_{O_2}, higher Pa_{CO_2} and $P(A\text{-}a)_{O_2}$ differences were noted in dorsal recumbency compared to lateral recumbency[11]. Lower P_{O_2} values of pulmonary vein blood originating from the caudal lobes were obtained from the dependent lung compared to the non-dependent lung in the lateral position[4].

DISCUSSION

A synopsis of the effects of posture on cardio-pulmonary function is given in Table 1.

For the anaesthetist the danger of a large $P(A\text{-}a)_{O_2}$ difference is that it causes a low partial pressure of oxygen in arterial blood, leading to tissue hypoxia and the need to correct the abnormality. The $P(A\text{-}a)_{O_2}$ difference only serves as an indicator of the pathological process leading to a reduction in Pa_{O_2}. The possible causes of $P(A\text{-}a)_{O_2}$ differences were summarised by Marshall & Wyche[8] as:

1. Restricted diffusion between alveolus and capillaries.
2. Increased pulmonary shunt.
3. Increased ventilation/perfusion mismatch.
4. Increased partial pressure of oxygen in inspired gas.
5. Decreased partial pressure of oxygen in mixed venous blood.
6. Shifts in hemoglobin-dissociation curve.

For a better understanding of the events leading to changes in pulmonary function during induction of anaesthesia, it is necessary to have a concept of the relative positions occupied by the abdominal and thoracic organs during changes in body position.

In the standing position the diaphragm divides the pleural and peritoneal cavities in such a way that the abdominal organs lie medial and ventral to the caudal

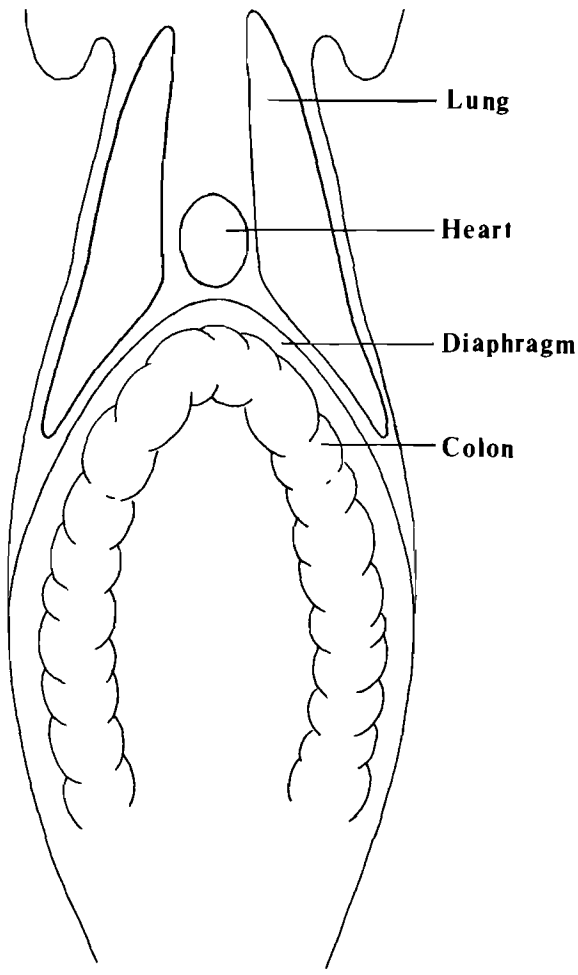

Fig. 1: STANDING – Dorso-ventral view of the thorax and abdomen illustrating the conical shape of the diaphragm. The cranial aspect of the abdomen is flanked by the pleural cavities on the lateral sides.

lobes of the lung. This arrangement is illustrated by dorso-ventral radiographs of the trunk showing the conical shape of the diaphragm[9] (Fig. 1). The largest part of the lung mass is near the dorsal border tapering towards the ventral border. The apex of the heart is situated distal to the ventral border of the lung.

When the horse is turned to the lateral position, the weight of the mediastinal organs and part of the abdominal organs is distributed over the dependent lung. The diaphragm is displaced cranially, reducing lung volume and lung expansion during respiration in the dependent lung[9] (Fig. 2). Increased ventilation occurs in the non-dependent lung.

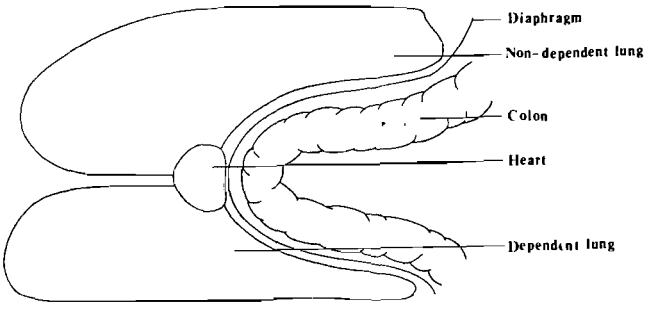

Fig. 2: LATERAL RECUMBENCY – The heart is situated midway between the non-dependent and dependent lung. The weight of the abdominal organs is partly distributed over the caudal lobe of the dependent lung, reducing lung volume.

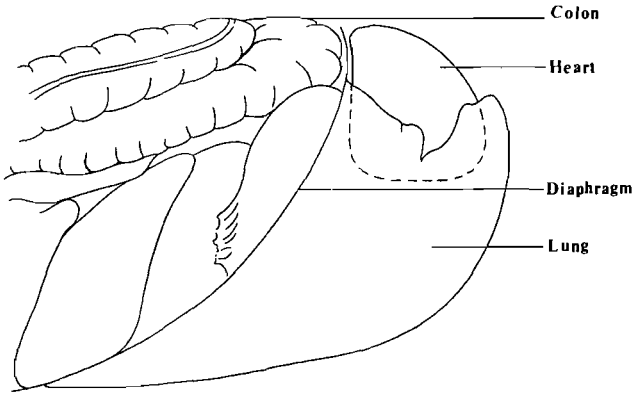

Fig. 3: DORSAL RECUMBENCY – The heart lies dorsal to the lungs and the abdominal organs dorsal to the diaphragm and caudal lobes. The weight of the abdominal organs is partly distributed over both caudal lobes, reducing lung volume.

In dorsal recumbency the largest part of the lung mass is now situated more ventrally (vertebral region) in the pleural cavity with the lung below the heart. The weight of the abdominal and thoracic organs is now distributed over both the caudal lobes, displacing the lumbar part of the diaphragm towards the vertebrae and reducing lung volume[9] (Fig. 3).

Perfusion of the lung in a vertical direction does not take place evenly[17]. Blood flow increases almost linearly from the upper to the lower part of the lung as a result of an increase in hydrostatic pressure. Distribution of blood flow is affected by change of posture and changes in cardiac output. Exercise will increase perfusion and during anaesthesia perfusion of the upper part of the lung will decrease as a result of cardiac output.

The lung may be divided in three zones of perfusion[17] (Fig. 4).

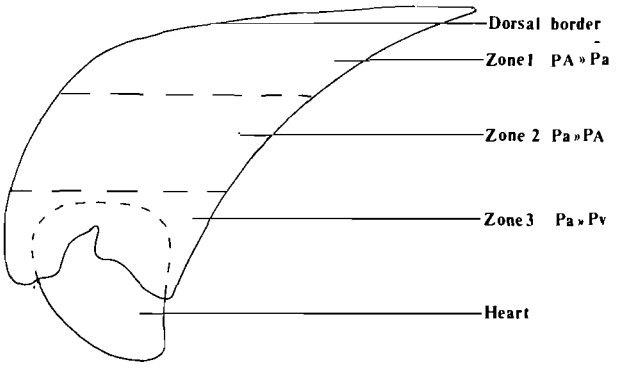

Fig. 4: DIAGRAM illustrating the three zones of blood flow in the lung. Zone 1, alveolar pressure higher than pulmonary artery pressure. Zone 2, pulmonary arterial pressure higher than alveolar pressure. Zone 3, pulmonary arterial pressure higher than pulmonary venous and alveolar pressure.
PA = alveolar pressure. Pa = arterial pressure. Pv = venous pressure.

In Zone 1 at the upper part of the lung, alveolar pressure is larger than arterial (hydrostatic) pressure. Consequently the alveolar capillaries are closed off as a result of the reduction in the alveolar capillary pressure. The alveoli are ventilated without any perfusion. This is alveolar dead space. In Zone 2 ventilation equals perfusion. In Zone 3 the arterial (hydrostatic) pressure increases beyond the venous (hydrostatic) pressure and alveolar pressure. Blood flow is determined by the arterio-venous hydrostatic pressure difference. As the alveolar pressure stays constant throughout the lung, blood perfusing the alveoli in the dependent parts of the lung will not be fully oxygenated, and this has a shunt effect.

In the standing or sternal recumbent horse with a normal blood pressure it may be assumed that the largest part of the lung would consist of Zones 2 and 3.

With the horse in the lateral position, the heart is situated between the lungs with a possible even distribution of perfusion between the non-dependent and dependent lobes if the animal's blood pressure is normal. During anaesthesia a reduced cardiac output and blood pressure would result in reduced perfusion of the non-dependent lung and a reduced rate of blood flow in the dependent lung. The anaesthetic drugs may promote vasodilation. As a result of the reduced expansion (compliance) of the dependent lung, there is increased ventilation of the non-dependent lung. The combined effect would favour the pooling of blood in the dependent lung. The altered perfusion in the lung would favour an increase in Zone 1 in the top lung, i.e. alveolar dead space. Due to slower blood flow, Zone 3 will increase in the bottom lung, i.e. venous 'shunting' will occur. The change in posture causes a redistribution of the weight of the abdominal organs on the diaphragm and dependent lung, thus reducing FRC and ventilation. The FRC reduction is significantly larger with the animal breathing oxygen compared to air.

In the dorsal position the weight of the abdominal organs is distributed over both caudal lobes, explaining the reduced reduction in FRC compared to lateral recumbency. An increase in Zone 3 and shunting are favoured in both lobes irrespective of blood pressure.

CONCLUSIONS

1. Lung volumes such as FRC, VC and TLC are influenced by changing from standing or sternal recumbency to lateral or dorsal recumbency. No differences in lung volumes occur between standing and sternal recumbency in either the conscious or anaesthetised animal. Sternal recumbency is the ideal position for the horse during recovery of anaesthesia before they are able to stand.
2. Changing to lateral recumbency in the conscious animal reduces FRC, but is still able to maintain normal blood gas values.
3. Withholding food for 24 hours increases FRC in the standing and lateral recumbent animal; sedatives have a negative influence on FRC.
4. Introduction of anaesthesia in a cast, lateral recumbent horse with an intravenous agent (thiopentone sodium) does not influence FRC. Increased anaesthetic depth tends to decrease FRC during halothane anaesthesia.
5. The non-dependent lung functions better than the dependent lung in lateral recumbency. As a result of the larger right lobe, better blood gas values are seen with the animal in left lateral recumbency. It is doubtful whether this has any clinical significance in the healthy animal. Body position is dictated by the nature of the surgical procedure.
6. The use of anaesthetic drugs influences cardiopulmonary function by reducing cardiac output and blood pressure. Its effect on hypoxic vasoconstriction has not been investigated. However, the results of Staddon et al.[15] may indicate the absence of this effect during halothane anaesthesia as this increase could also be the result of hypostasis in the dependent lung during lateral recumbency.
7. Pulmonary shunt increases very little from the awake standing to the awake lateral recumbent pony. Anaesthetic drugs increase the percentage pulmonary shunt in the recumbent animal. No comparative work has been done on the difference in shunt between the lateral and dorsal recumbent animal during anaesthesia.
8. Horses should be starved for 24 hours before anaesthesia.
9. Mechanical ventilation reduces the negative effects of anaesthesia during lateral or dorsal recumbency.
10. During prolonged post-anaesthetic recovery, horses should be encouraged to adopt sternal recumbency.

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ABSTRACT

SAMEVATTING

AN OVINE HEPATOTOXICOSIS CAUSED BY THE PLANT *PTERONIA PALLENS* (ASTERACEAE) L.F.

The hepatotoxicity of *Pteronia pallens* was demonstrated in 5 sheep which developed lesions that ranged from centrilobular necrosis to diffuse hepatocellular degeneration. Botanical, clinical and pathological data are given and the lesions are briefly compared with those caused by other hepatotoxic plants in South Africa. (Prozesky, L., Kellerman, T.S. & Welman, Wilhelmina, G., 1986. An ovine hepatotoxicosis caused by the plant *Pteronia palens* (Asteraceae) L.f. *Onderstepoort Journal of Veterinary Research*, **53**, 9 – 112 (1986).)

ABSTRACT

SAMEVATTING

A FIELD EVALUATION OF *BACILLUS THURINGIENSIS* VAR. *ISRAELENSIS* AS A BIOLOGICAL CONTROL AGENT FOR *SIMULIUM CHUTTERI* (DIPTERA: NEMATOCERA) IN THE MIDDLE ORANGE RIVER

Bacillus thuringiensis Berliner var. *israelensis* de Barjac (Serotype H-14) (*B.t.i.*) at a concentration of 1,6 ppm/10 min and a toxicity of 1500 AAU/mg was tested against *Simulium chutteri* Lewis larvae in the Orange River near Prieska, South Africa. Samples of benthic fauna from the stones-in-current biotope were collected before application of the product and at various intervals op to 80 h afterwards at 4 stations from 200 m to 11 km downstream of the application site. Faunal drift increased slightly after the arrival of the *Bacillus* at 2 stations 1,4 and 6 km respectively downstream of the application site.

Large numerical decreases in benthic simuliid larval numbers after the application of *B.t.i.* in the Orange River were not statistically different ($P > 0,05$). This indicated that the size of replicated samples that showed significant decreases ($P < 0,05$) of simuliid numbers in the Vaal River was not adequate to show statistical differences in the Orange River. The quantity of dead larvae on stones collected from rapids after application of the *B.t.i.*, and the numerical decreases found by comparing median values of larval counts on stones indicated that *B.t.i.*, effectively killed simuliid larvae. Three days after application of the *Bacillus*, recruitment of small simuliid larvae on stones 1,4 km downstream of the application site was discernible again. Tanytarsini were also numerically reduced after *B.t.i.* application. At a flow rate of 38 m^3/s *B.t.i.* was visibly effective in killing *S. chutteri* up to 6 km downstream of the application site and statistically significant decreases ($P < 0,05$) in numbers of larvae were seen at a site 11 km downstream of the application site.

The use of *B.t.i.* in *Simulium* control is preferable to organophosphate and organochloride formations because it has a more specific action against blackfly and because there is no known immunity to *B.t.i.* in any *Simulium* species. However, we must advise that *B.t.i.* should be screened against all co-existing fauna in each situation where new community structures of animals are encountered. Several methods for improving the efficacy of *B.t.i.* are suggested. (Moor, F.C. de & Car, M., 1986. A field evaluation of *Bacillus thuringiensis* var. *israelensis* as a biological control agent for *Simulium chutteri* (Diptera: Nematocera) in the Middle Orange River. *Onderstepoort Journal of Veterinary Research*, **53**, 43 – 50 (1986).)

BEPLANDE MELKKUDDEGESONDHEID II. 'N GEÏNTEGREERDE GINEKOLOGIESE PROGRAM

D.C. LOURENS* en R.I. COUBROUGH*

ABSTRACT: Lourens D.C.; Coubrough R.I. **Planned dairy herd health II: An integrated gynaecological programme.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 55-65 (Afrik). Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Integrated gynaecological programmes play an extremely important role in total herd health control by ensuring the maintenance of optimal reproduction and production. The economic implications of reduced breeding efficiency manifest in, and are reflected by total lifetime milk production, the size of the calf drop, the availability of replacement animals and increased involuntary culling rate. Measured against production costs (feed, labour and veterinary expenses), lowered reproductive efficiency erodes the margin of profitability of the farming enterprise.

Herd health programmes must be well planned, stringently implemented and regularly evaluated to ensure their effectiveness. To this end they must be based on simple and practical guidelines which can be adapted as required to cope with varied farming and management conditions. This paper outlines the setting of clear objectives, factors to consider during the introductory phasing in of an integrated gynaecological programme and details of each subsequent routine visit.

Ideal objectives are presented and discussed in terms of the individual animal as well as the herd. Suggested figures for each parameter are given. The significance of oestrus observation is stressed as cardinal to the attainment of many of the set objectives. The significance of calf management as the nucleus of the production of a healthy heifer replacement herd is discussed.

During the introductory phase, which can be a protracted period, the foundation for routine visits is laid. Herd structure is defined, facilities are evaluated, record systems are set up and the farmer is groomed as to his role in the successful implementation of the planned programme. The value of a control questionnaire is highlighted, and factors considered are set out.

The method of the routine gynaecological programme is discussed in detail. The necessity for a standard examination list is set out and the various categories of cows to be examined on each routine visit is clearly outlined, with important factors relating to each being highlighted. Examples of the various forms used are given.

Evaluation of the acquired data should be done immediately, and discussed with the farmer during the current visit. Findings can be compared with those of previous visits, analysed, and recommendations may be considered immediately in consultation with the farmer.

Dairy farming is a highly complex farming enterprise involving large capital outlays, intensification of labour and astute management for optimal and economic production. While the gynaecological programme forms the basis of a herd management programme it is closely integrated with factors such as nutrition, management, the general health of the herd, the condition of the animals, as well as calf and heifer management. The success of any programme rests squarely on the recognition that the integration of the various facets of production management is essential to ensure economic production. While this necessitates a team effort, the veterinarian plays a strategic role as a co-ordinator of the interdisciplinary team involved in this endeavour.

Key words: Gynaecological dairy herd health programme

INLEIDING

Dit word vandag vry algemeen aanvaar dat 'n ginekologiese program as 'n onderdeel van kuddegesondheid nie net om rektale ondersoeke gaan nie maar deel moet vorm van 'n wye, geïntegreerde benadering wat bestuur, voeding en behuising, gegrond op ekonomiese produksie, insluit[7].

Daar is 'n belangrike interaksie tussen die koei, haar omgewing en mikro-organismes. Die "omgewing" behels hier bestuur, behuising, higiëne en voeding gedurende die peripartale periode[2 7 17]. Rektale ondersoeke en ander bevindings, soos kondisie beoordeling, moet die inligting (simptome) verskaf wat dan, in terme van die omgewingsfaktore wat geheers het, geïnterpreteer moet word. Die voorkoms van onaktiewe ovaria kan waarskynlik aan 'n voedingsgebrek te wyte wees, wat klinies as 'n wanfunksie van die geslagstelsel manifesteer.

Die verwaarlosing van enige aspek van peripartale bestuur sal 'n dramatiese invloed op vrugbaarheid uitoefen. Faktore wat bv. by kalwing met verlaagde fertiliteit geassosieer word sluit hoë omgewingstemperature, distokie, agtergeblewe nageboortes, metritis, melkkoors, ketose en die vetkoeisindroom in. 'n Onhigiëniese kalwingsarea is 'n belangrike oorsaak van nageboortelike komplikasies en kom dikwels as 'n siektekompleks voor. Die voorkoms hiervan varieer van seisoen tot seisoen asook tussen groepe koeie en is van die voedingstatus wat geheers het, afhanklik[2 7 17 24].

Vrugbaarheidsprobleme het 'n dramatiese invloed op die ekonomie van die suiwelbedryf. Verlaagde reproduksie lei tot 'n vermindering in die nageslag (potensiële genetiese materiaal), verlies in lewensmelkproduksie, onproduktiewe voeromsetting en verhoogde onkostes om koeie met langer laktasies en droë periodes te versorg. Verlaagde vrugbaarheid kan verder tot 'n hoë persentasie verpligte uitskot aanleiding gee, wat dan tot verdere onkostes aan vervangingsdiere lei[10].

Bogenoemde is genoegsame redes om vrugbaarheid as deel van 'n totale program sterk te beklemtoon, mits al die onderlinge bestuurs-, voedings- en omgewingsverwantskappe deeglik in gedagte gehou word.

Die doel van hierdie artikel is om 'n praktiese uitvoerbare model t.o.v. vrugbaarheid as onderdeel van 'n kuddeprogram te skep, wat praktisyns dan by hulle eie spesifieke behoeftes kan aanpas. Die model word opgebou uit (a) doelwitte, (b) metodiek in die aanloopfase,

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en (c) metodiek met roetine ginekologiese ondersoeke. Op dieselfde manier kan praktiese modelle t.o.v. ander aspekte van kuddegesondheid geskep word, bv. mastitis[13 14], kalfgesondheid[2 8 13 19], algemene gesondheid[7] en voeding en produksie[7 9 19 20]. Die belang van rekordhouding sal in 'n verdere artikel volledig bespreek word.

DOELWITTE

Doelstellings is noodsaaklik sodat 'n kudde se status en vordering teen aanvaarbare ekonomiese standaarde gemonitor kan word. Dit moet egter nooit as absoluut beskou word nie, maar eerder as 'n verwysing na die ideaal, wat plooibaar moet wees om sodoende aanpassings van plaas tot plaas te kan maak.

As evaluering sou aantoon dat 'n produsent ver buite aanvaarbare norme is, moet advies realisties, prakties en stapsgewyse toegepas word. 'n Gemiddelde tussenkalfperiode (TKP) van 415 dae kan bv. eers na 400 dae en dan na 385 dae verminder word. Die omvang van advies en die toepassing daarvan sal noodwendig deur die boer se bestuursvernuf bepaal word[1]. 'n Finansiële analise kan aan die boer demonstreer hoeveel hy verloor a.g.v. lang kalwing tot konsepsie intervalle en is 'n groot hulpmiddel om boere te oortuig van die belang van optimale teeldoeltreffendheid[10].

'n Doeltreffende rekordstelsel is uiters noodsaaklik sodat die nodige ginekologiese analises en evaluasies uitgevoer kan word. Die data wat ingewin word moet deeglik analiseer en evalueer word vir sinvolle advies en om enige knelpunte spoedig op te spoor[4].

Die hoofdoelwit, vanuit 'n ginekologiese oogpunt, is om kuddevrugbaarheid te verhoog om sodoende optimale produksie te bereik. 'n Tussenkalfperiode van 365 – 385 dae is die ekonomiese norm om optimale produksie te bereik en koeie moet dus binne 85 – 115 dae na kalwing weer beset raak[7]. 'n Periode van ongeveer 45 dae moet egter vir involusie toegelaat word. 'n Tydperk van slegs 40 – 70 dae is dus daarna vir teling beskikbaar. Koeie moet in estrus waargeneem en geïnsemineer word sodat konsepsie binne slegs 1 – 3 estrus siklusse moet plaasvind[7]. Die enigste tydperk wat die TKP kan beïnvloed is die periode vanaf kalwing tot herkonsepsie. Ons verwag dus 'n normale kalwing, met 'n gesonde kalf, optimale melkproduksie, normale involusie en herkonsepsie binne 85 – 115 dae. Hierdie ideaal kan verseker word deur optimale bestuur t.o.v. die droë koei, partus, die puerperium, estrus waarneming en kunsmatige inseminasie[7].

Die ideale TKP kan wel bereik word indien die volgende doelwitte as kuddegemiddeldes nagestreef word. Hierdie doelwitte is op ekonomiese beginsels gegrond en is biologies onder optimale bestuur en voeding moontlik[1 7 11 13 18 26]:

Samestelling van melkkudde:	
Lakterende: droë koeie	80:20
Vir elke 100 koeie in 'n kudde moet daar ongeveer die volgende wees:	
	10 suipkalwers
	28 1 jaar verse
	27 1-2 jaar verse
	25 dragtige verse
Lakterende koeie: totale grootte van kudde	50:50
Ouderdom van kudde:	
Eerste laktasie	±25%
Tweede – vyfde laktasie	±60%
Ses en meer laktasies	±15%
Laktasielengte	305 dae
Kalwing tot konsepsie	100 dae (ideaal 85 dae)
Lengte van droë periode	42 – 60 dae
Aborsies	<2%
Distokie	<5%
Agtergeblewe plasenta	<5%
Metritis	<10%
Eerste post-partale estrus waargeneem	45 dae
% Koeie in estrus teen 60 dae post-partaal	90%
Eerste inseminasie	45 – 60 dae na kalwing
Eerste inseminasie geslaagde konsepsie	55 – 60%
% Koeie teen 90 dae post-partaal geïnsemineer	90%
% Koeie wat meer as 3 inseminasies benodig	<15%
Inseminasies per konsepsie	<2 (ideaal 1.6)
% Koeie meer as 100 dae kalwing – konsepsie	<7%
% Koeie dragtig met dragtigheidsondersoeke	95%
Verhouding van enkel (18 – 24 dae) versus dubbel (39 – 45 dae) estrus intervallengtes	6:1
Gedwonge uitskot (vrugbaarheid, mastitis, produksie)	<10%
Selektiewe uitskot (pote, temperament, geneties)	<15%
Lewensduur van koeie	±5 laktasies

Daar moet in gedagte gehou word dat daar 'n verskil tussen die ideale individuele koei en die ideale kudde is waar die doelwitte meer plooibaar sal wees. Die ideale melkkoei behoort op 15 – 18 maande as vers beset te raak, op 24 – 27 maande te kalf, daarna elke 365 dae te kalf en 5 – 7 laktasies te voltooi. In 'n ideale kudde sal hierdie doelwitte meer plooibaar wees en verse kan gemiddeld teen 28 maande kalf. 'n Tussenkalfperiode van 385 dae behoort daarna bereik te word. Die eerste inseminasie moet binne 60 dae post-partaal met 'n konsepsiesyfer van ±55% plaasvind en 90% van die kudde behoort teen 60 dae post-partaal estrus te toon[1 7 26].

Die variasies in doelwitgemiddeldes kan diagnosties gebruik word. Swakhede in peripartale bestuur, voeding, higiëne en algemene gesondheid kan sodoende uitgewys word. Estrus waarneming is bv. 'n faktor wat reproduksieprestasie in baie kuddes beperk, en daardeur benadeel dit produktiwiteit en winsgewendheid a.g.v. 'n lang TKP. Om estrus waarneming akkuraat te evalueer is toereikende identifikasie van koeie, goeie rekords, volledige aantekeninge van estrus en gereelde veeartsenykunde ondersoeke nodig[23]. Hierdie evaluasies ageer nou as "diagnostiese indekse". Indekse wat bv. gebruik kan word om estrus observasie te evalueer sluit in[1 3 23 26]:

- % koeie teen 60 dae post-partaal in estrus
- gemiddelde interval tot eerste sigbare estrus
- % koeie dragtig met dragtigheidsondersoeke
- bestudering van estrus intervallengtes

Die normale intervallengte is 18 – 24 dae (±21 dae).

Kort intervalle van 17 dae dui gewoonlik op foutiewe waarneming. Enkele gevalle kan egter aan organiese redes soos bv. sistiese ovaria gekoppel word. Indien die intervallengtes veelvoude van ±21 dae is, bv. 36 – 48 dae of selfs 55 – 70 dae, dui dit meestal op estrus wat nie waargeneem is nie. Abnormale oneweredige lang intervalle soos bv. 31 of 35 dae, dui gewoonlik op embrioniese mortaliteit.

Op 'n kuddebasis met goeie estrus waarneming behoort die verhouding tussen normale enkel intervallengtes tot veelvoudige intervalle, 6:1 te wees. Indien 6 of meer keer enkel siklusse voorkom vir elke 1 veelvoudige siklus, is estrus waarneming bevredigend[26].

Estrus intervalle kan ook m.b.v. histogramme voorgestel word. Hieruit kan die effektiwiteit van estrus waarneming beoordeel word. In 'n groot aantal kuddes met 'n hoë doeltreffendheid van estrusvasstelling was 10 – 15% van die siklusse korter as 18 dae, 55 – 60% was 18 – 24 dae en 26 – 33% was meer as 24 dae[23].

'n Sogenaamde estrus waarnemingsgraad kan met behulp van die gemiddelde intervallengte van die kudde bepaal word[26].

$$\text{Estrus waarnemingsgraad} = \frac{21}{\text{Gemiddelde interestrus periode}} \times 100$$

Indien 'n lae waarde verkry word mag swak waarnemings en/of 'n hoë insidens van patologiese toestande soos bv. embrioniese mortaliteit, infeksies of sistiese ovaria die oorsaak wees. Indien meer as 100% verkry word is foutiewe waarneming (kort siklusse) 'n waarskynlike rede. Die gebruik van prostaglandines op groot skaal kan ook tot 'n hoë waarde lei.

As 'n boer oor "anestrus" kla sal hierdie indekse, tesame met die kliniese bevindings, van diagnostiese belang wees. Indien rektale ondersoeke sou aantoon dat koeie aktief en klinies normaal is, is dit baie moontlik dat die koeie nie waargeneem of aangeteken is nie[26].

Suksesvolle teling is verder afhanklik van faktore soos die boer se beleid vir eerste inseminasie postpartaal, semen kwaliteit, vrugbare gesonde koeie, inseminasietegniek en korrekte tyd van inseminasie[3].

Dit moet weereens beklemtoon word dat enige voortplantingsprobleme met aspekte soos voeding, melkproduksie, kondisie, kreupelhede, metaboliese siektes en ander gesondheidsteurnisse in verband gebring moet word[2024].

'n Onhigiëniese kalfomgewing, wanvoeding, hoë omgewingstemperature, distokie en metaboliese siektes soos ketose is almal faktore wat tot toestande soos *metritis, onaktiewe- en sistiese ovaria aanleiding kan* gee[24].

Dit is belangrik dat koeie, in ooreenstemming met hul produksiefase, gebalanseerd gevoer moet word. Voeding tydens die droë en die puerperale fase is baie belangrik vir 'n optimale TKP. Voeding vorm seersekerlik die belangrikste basis vir optimale produksie en reproduksie. 'n Negatiewe energie balans gedurende die puerperale periode is 'n belangrike predisponerende faktor tot die ontstaan van toestande soos metritis en onaktiewe ovaria. Die kondisie van koeie by kalwing asook die graad van gewigsverlies post-partaal het 'n direkte effek op die aanvang van estrus en dus uiteindelike herkonsepsie[2024].

Wisselende klimaatsomstandighede in die RSA lei tot variasies in die kwaliteit en beskikbaarheid van voer. Daar moet dus noulettend aandag aan die samestelling en die beskikbaarheid van gebalanseerde rantsoene gegee word. 'n Voervloeiprogram om kwaliteit en kwantiteit regdeur die jaar te verseker is uiters noodsaaklik.

'n Numeriese kondisiebeoordelingsisteem is 'n eenvoudige, praktiese hulpmiddel om voeding te evalueer. Dit vind op 'n skaal van 1 tot 5 plaas, waar 1 'n uitgeteerde koei en 5 'n oorvet koei aandui. In die praktyk kan halwe skale gebruik word vir koeie met tussenin kondisie[525].

Kondisiebepaling van koeie op hierdie skaal word op palpasie en visuele beoordeling van die ribbes, werwels, heup, sitbene en die area om die stertwortel gegrond. Daar word na die graad van spier- en vetweefsel vulling in hierdie areas opgelet.

Kondisie 1

Die koei is uitgeteer met skerp spinale uitsteeksels en die ribbes is duidelik sigbaar. Die heup- en sitbene is baie prominent. Die lendewerwels toon 'n oorhangende effek en die area om die stertbasis en tussen die sitbene is erg versonke.

Kondisie 2

Die individuele werweluitsteeksels en die ribbes is visueel sigbaar maar nie prominent nie. Die transverse lumbale uitsteeksels is nog skerp met 'n oorhangende effek. Die heup- en sitbene is sigbaar maar nie prominent nie en die area om die stertbasis is versonke. Alle beenstrukture het wel geringe hoeveelhede spierweefsel op.

Kondisie 3

Die spinale uitsteeksels is bepaalbaar deur drukking toe te pas en kom as 'n ronding voor. Die transverse lumbale uitsteeksels het nie meer 'n oorhangende effek nie. Die heup- en sitbene is gerond en glad. Die area om die stertbasis en tussen die sitbene is gelyk en vetweefsel is teenwoordig.

Kondisie 4

Die individuele spinale uitsteeksels is alleenlik deur harde palpasie bepaalbaar. Die lumbale werwels is plat en afgerond. Die heup- en sitbene is rond, glad en plat. Die area om die stertbasis is rond en voue van sagte vetweefsel is teenwoordig.

Kondisie 5

Die beenstrukture is glad nie sigbaar nie en subkutane vetneerlegging is baie prominent. Die stertbasis is letterlik in die vet begrawe.

Koeie behoort in 'n kondisie 3½ af te droog – hierdie kondisie moet gehandhaaf word tot en met kalwing. Post-partaal toon die koei 'n verlaagde aptyt en terselfdertyd 'n verhoogde energieverbruik om haar melkproduksiepeil te bereik. Die melkproduksiepiek is op ±7 weke terwyl voldoende droë materiaal inname eers teen ±12 weke plaasvind om aan die energiebehoeftes te voldoen. Hierdie energietekort lei tot 'n kondisieverlies van ±1 eenheid teen ±100 dae in laktasie. Sou hierdie kondisie te laag daal tot bv. 2 en selfs laer, sal dit herkonsepsie vertraag en sal dit ook baie moeilik wees om 'n ideale kondisie van 3½ eenhede by afdroog te bereik[24].

'n Voorvereiste vir 'n gesonde, hoogsproduserende suiwelkudde is die beskikbaarheid van goed uitgegroeide, gesonde vervangingsverse. Hulle is die ekonomiese en genetiese basis van die toekomstige kudde en

daar moet vir ±20% vervanging per jaar voorsiening gemaak word. Hierdie persentasie is van faktore soos die uitskot persentasie, die opbou van die kudde en die persentasie kalfmortaliteite afhanklik[7].

Die grootmaak van kalwers en verse is dikwels die mees verwaarloosde aspek op die melkplaas. Dikwels word kalwers en verse onder swak bestuurstoestande of op 'n té lae voedingspeil grootgemaak. Dit maak die kalf en vers meer vatbaar vir siektes en vertraag ook die puberteit. As gevolg hiervan word die onproduktiewe gedeelte van 'n koei se lewe verleng deurdat sy op 'n betreklike laat ouderdom vir die eerste keer kalf. Dit is belangrik om die vers se ontwikkeling en groei in verhouding tot haar ouderdom te beoordeel, aangesien dit 'n belangrike effek op puberteit, konsepsie, moontlike latere distokieë, melkproduksie en post-partale fertiliteit het. Aan die ander kant moet weer teen oorvet verse gewaak word. Dit kan aanleiding gee tot vervetting van die uierweefsel met gevolglike laer produksie, perivaginale vervetting met hoër insidens van distokie en fertiliteitsteurnisse tydens die puerperium[19,20].

Die kalf moet dus gesond en lewenskragtig gebore word, gesond bly en optimaal groei vir die bereiking van puberteit. Die kalwingsarea van koeie, die algemene partus bestuur en die neonatale versorging van die kalf is kernfaktore wat in ag geneem moet word. Die voedingspeil tesame met gesonde bestuurspraktyke, goeie behuising en die handhawing van gesondheid, bv. parasietbeheer, is van die uiterste belang vir optimale gesondheid en groei[17].

Die ideale vervangingspersentasie en kalwingsouderdom vir verse kan wel bereik word indien sekere basiese riglyne t.o.v. die kalf en vervangingsvers nagekom word[7,13,20]:

- Kalwers doodgebore en perinatale sterftes binne 24 uur <5%
- Kalwersterftes binne 1 – 30 dae <4%
- Kalwersterftes binne 1 – 3 maande <2%
- Sterftes ouer as 3 maande <2%
- 90% van verse moet grootmaak periode oorleef.

Groeitabelle en/of standaard groeikurwes waarin verse se gewig en hoogte in verhouding tot hulle ouderdom beoordeel kan word, is beskikbaar vir verskillende melkrasse[13,18,22].

Die vers moet by teelouderdom (±15 maande) reeds ongeveer 66% en by partus 80% van haar uiteindelike volwasse massa bereik het.

Tydens die eerste 100 dae van laktasie is haar massa vir haar eie groei, melkproduksie en herkonsepsie van die uiterste belang[18,22].

Mikpuntmassas neig om te varieer van outeur tot outeur of van area tot area. In die keuse van 'n mikpuntmassa vir 'n spesifieke kudde moet die volwasse gewig van die koeie, en die rastipe in gedagte gehou word.

METODIEK IN DIE AANLOOPFASE

Die periode tussen die eerste en latere geroetineerde besoeke aan 'n plaas, word as die "aanloopfase" gedefinieer[7,14]. In dié periode wat 'n hele aantal maande mag duur, moet daar 'n totale beeld van die kudde gevorm word en moontlike probleemareas moet geïdentifiseer word. Die rekordhouding moet nagegaan word en die boer se bestuursvermoë moet ook gepeil word. 'n Algemene inspeksie om 'n oorsig te verkry is ook noodsaaklik[7]. Effektiewe hanteergeriewe, duidelike identifikasie van die diere, en 'n funksionele rekordstelsel is elementêre vereistes vir enige kuddegesondheidsprogram[1,26].

Tydens hierdie besoeke moet die boer oor die verskillende fasette van die gesondheidsprogramme, die belangrikheid van rekordhouding en die finansiële implikasies van die program ingelig word. Daar moet deeglik aan die boer verduidelik word watter diere vir roetine ondersoeke nodig is (sien later)[11,13,14]. Die frekwensie van die besoeke is van die aard van die probleme, die grootte van die kudde en die kalwingspatroon, afhanklik. Aanvanklik mag dit nodig wees om 'n prioriteitslys, afhangende van die probleme, op te stel[1].

In die Departement Geslagskunde, Fakulteit Veeartsenykunde, Universiteit van Pretoria, vind ons dat 'n kontrole vraelys, wat oor al die fasette van die boerdery handel, 'n groot hulpmiddel is. 'n Oorsig van die plaas word sodoende verkry en probleemareas kan alreeds hier geïdentifiseer word. Die vraelys sluit onder andere die volgende in:

- Algemene inligting oor die plaas (grootte, veldtipes, kampe, drakrag, saaiboerdery en voerproduksie).
- Tipe melkboerdery (vars of industrieel).
- Produksiegegewens.
- Samestelling van die kudde.
- Rekordhouding.
- Teelbestuur.
- Behuising, voeding, bestuur en siektebeheer van die volgende: droë koeie, koeie in melk, kalwers en vervangingsverse.
- Peripartale bestuur.
- Voervloeiprogram.

METODIEK MET ROETINE GINEKOLOGIESE PROGRAM

Diere benodig

Sodra die gegewens ten opsigte van kalwings, kondisie met kalwing, afdroog, estrus en inseminasies, op datum gebring is, kan die boer of die veearts die diere wat ondersoek moet word vanaf die koeikaarte selekteer[11,22,21]. 'n Rekenaarstelsel wat deur òf die veearts òf die boer bedryf word, sal hierdie arbeidsintensiewe taak aansienlik vergemaklik[4].

'n Ondersoeklys[15] is 'n absolute noodsaaklikheid. Die inligting wat hierop moet verskyn, is van die tipe ondersoek afhanklik en sluit byvoorbeeld die volgende in:

- Dragtigheidsondersoeke – Hier word die laaste inseminasiedatum, asook die aantal dae oop tot laaste inseminasie, benodig.
- Nageboortelike ondersoeke – Die kalfdatum en inligting oor die verloop van kalwing word hiervoor benodig.
- Oop koeie – Hier word die tydperk wat die koei oop is, estrus of inseminasiedatums, vorige bevindings en behandelings benodig.

Op die ondersoeklys moet ruimte gelaat word, waar die huidige geslagskundige bevindings, kondisie van die diere, diagnoses, algemene siektes en behandelings genoteer kan word. 'n Afskrif van die ondersoeklys, tesame met 'n evaluering van die gelyste diere word dan dadelik aan die boer verskaf en bespreek (sien later).

Vanuit 'n ginekologiese oogpunt moet die volgende koeie en/of verse ondersoek word[11,22,21]:

1. Diere wat ±42 dae of vroeër geïnsemineer is en sedertdien geen estrus getoon het nie, moet vir dragtigheid ondersoek word. In hierdie departement is her-

bevestiging van dragtigheid (tot op 10 – 12 weke) 'n roetine – veral in kuddes waar estrus observasie 'n probleem is. Koeie wat estrus toon en reeds voorheen dragtig bevestig is, moet herondersoek word vir dragtigheidsbevestiging, aangesien dragtige koeie wel estrus kan toon[1].

2. Roetine post-partale ondersoeke moet ±3 weke na kalwing op elke koei uitgevoer word om die vordering van involusie en die algemene kondisie te beoordeel. Hierdeur kan enige probleme vroegtydig gediagnoseer word en die nodige aandag geniet[1221]. Daar is die opinie dat daar nie genoegsame rede vir roetine ondersoeke op koeie met 'n normale peripartale periode bestaan nie[1]; hierdie benadering kan tot onvolledige bestuur lei. Hulle beperk hulle ondersoeke net tot koeie wat 'n abnormaliteit soos byvoorbeeld distokie, agtergeblewe plasenta, prolaps uterus tydens of net na partus gehad het. Dit is ons opinie dat kondisies soos subinvolusie, endometritis en trauma van die geslagstelsel oor die hoof gesien word met hierdie benadering met 'n gevolglike nadelige invloed op die kalwing tot konsepsie interval.
3. Koeie wat 60 dae post-partaal is en nog nie estrus getoon het nie of enige ander koei wat sedert haar laaste aangetekende estrus stil is (±49 dae) en nog nie geïnsemineer is nie, moet ondersoek word. Hierdie probleem word as die sogenaamde nie-sigbare estrus sindroom geklassifiseer. Die term anestrus moet liefs voorlopig vermy word, aangesien dit slegs 'n kliniese teken van 'n probleem is[112]. Swak estrusobservasie en wanvoeding bly steeds die belangrikste oorsake. Daar kan ook verskeie organiese redes vir die kliniese sindroom van anestrus wees soos byvoorbeeld sistiese ovariese degenerasie, endometritis, piometra en ovariese atrofie[12]. Kondisiebeoordeling is ook hier weereens van die uiterste belang.
4. Koeie met ongereelde sikliese aktiwiteite moet ondersoek word. Hierdie ongereelde siklusse kan die gevolg van bestuursprobleme soos foutiewe estrus waarneming of patologiese toestande soos sistiese ovaria, wees[126].
5. Koeie met duidelike kliniies waarneembare abnormaliteite soos nimfomanie of vaginale uitskeidings, moet ook geselekteer word.
6. Koeie wat na drie inseminasies nog nie beset is nie, moet ook vir 'n ondersoek gelys word. Tydens die ondersoek van hierdie koeie is dit belangrik om faktore soos estrusobservasie, semenkwaliteit en tegniek van inseminasie, veral as daar 'n hoë kudde insidens is, in gedagte te hou. Patologiese toestande wat lei tot die onvermoë tot konsepsie sindroom, sluit vertraagde ovulasie, fallopiese buisletsels, subkliniese endometritis en prolaps van die serviks in. Indien 'n koei onder ideale bestuurstoestande ±6 maande na haar laaste kalf nog nie dragtig is nie, moet uitskot teen die einde van haar laktasie, ernstig oorweeg word[1].
7. Enige dier wat geaborteer het moet ook vir 'n ondersoek inkom, sodat die huidige status van haar algemene gesondheid en geslagsstelsel bepaal kan word. Aborsies word in 'n ernstige lig beskou en die oorsaak daarvan moet ten alle koste vasgestel word.[1].
8. Diere wat met die vorige besoek behandel is, moet herondersoek word sodat die doeltreffendheid van die behandeling krities evalueer kan word.
9. Verse, wat van tyd tot tyd vir sinkronisasieprogramme ingebring word, moet op kondisie, gewig, ouderdom en ovariese aktiwiteit beoordeel word.

Evaluasie

Nadat bogenoemde diere ondersoek is, moet die resultate, ten opsigte van die tipe ondersoek, geëvalueer en bespreek word. Op grond hiervan kan die veearts nou doeltreffende advies verskaf. Afskrifte word vir eie rekorddoeleindes aan die boer verskaf. Hierdie bevindings dien nou verder ook as "aksielyste" waarvolgens spesifieke koeie, vir byvoorbeeld inseminasies en die toediening van enige opvolgbehandelings, dopgehou moet word[14151617].

In die Departement Geslagskunde maak ons tans van drie vorms, waarop geëvalueerde besonderhede ten opsigte van dragtighede, die verloop van die puerperium en die oop koeie voorkom, gebruik. Die ondersoeklys se resultate word sodoende georden en met die doelstellings vergelyk. Hierdeur word baie manure bespaar en in plaas van 'n skriftelike verslag lank na die besoek, kry die boer nou dadelik 'n bygewerkte evaluasie van die pas afgelope ondersoek.

Die resultate van die dragtigheidsondersoeke word ten opsigte van die volgende punte geëvalueer (Sien addendum I):

- Die totale aantal koeie ondersoek.
- Die aantal en persentasie dragtig met gemiddelde kalwing tot konsepsie interval.
- Die aantal en persentasie nie-dragtig maar aktief normaal.
- Die aantal nie-dragtig wat 'n abnormaliteit van die geslagstelsel toon.

Die mikpunt met dragtigheidsondersoeke is 'n 90% besetting. Indien dit nie bereik word nie en 'n hoë persentasie koeie is nie-dragtig, maar klinies normaal en aktief, moet daar ernstig na estrusobservasie gekyk word.

Die resultate van die post-partale ondersoek word as volg geëvalueer (sien addendum II):

- Normale involusie
- Subinvolusie
- Endometritis-metritis kompleks
- Siste
- Serviks letsels
- Trauma
- Ander

Subtotale word bereken en as 'n persentasie van die aantal koeie ondersoek uitgedruk, en dan met die mikpunte vergelyk.

Een van die mikpunte van die puerperale periode is 'n voorkoms van metritis van minder as 10%. 'n Hoë voorkoms van metritis kan gewoonlik die gevolg van 'n interaksie van verskeie faktor wees. Risiko faktore soos byvoorbeeld 'n onhigiëniese kalwingsarea word dikwels onderskat en geniet ongelukkig nie die nodige aandag nie[24].

Die evaluering van "oop koeie" word in vyf tydsvakke naamlik 45 – 60 dae; 61 – 90 dae; 91 – 120 dae; 121 – 150 dae en meer as 150 dae, ingedeel. By elke tydsvak word die status van die geslagstelsel in terme van eierstokaktiwiteit, toestand van die uterus, serviks letsels, vergroeiings of enige ander abnormaliteit wat mag voorkom, beoordeel (sien addendum III).

Die subtotale en persentasies word bereken en met die mikpunte vergelyk. As 'n hoë persentasie koeie, met 'n geskiedenis dat hulle stil was, nou tydens ondersoeke aktief bevind word, moet weereens na estrusobservasie gekyk word. Met 'n hoë voorkoms van onaktiewe

ovaria moet die koeie se kondisie en voedingsregime deeglik nagegaan word.

Die departement maak verder van 'n ginekologiese-, sowel as 'n algemene indekslys (sien addendum IV en V) gebruik. Die boer, sowel as die veearts, het kopieë van hierdie indekslyste en die sukses hiervan is van die boer, wat baie van die data self moet verskaf, afhanklik. Na die ondersoek word data uitgeruil en beide se afskrifte word dan op datum gebring[15 16 17]. Hierdie indekslyste bevat die volgende inligting:

- Samestelling van die kudde.
- Melkproduksie en melkkwaliteit.
- Algemene siektes.
- Morbiditeit en mortaliteit by kalwers en verse.
- Ginekologiese bevindings.
- Uitskot gegewens.

Op 'n praktiese, dog eenvoudige manier word selektief geanaliseer om sodoende die polsslag van die kudde te volg en verwantskappe te evalueer. Die kudde word dus met elke besoek, asook oor die langtermyn, gemonitor. Sodoende word afwykings van gestelde mikpunte aan die boer uitgewys[15 16 17].

Die veearts kan nou, in samewerking met die boer, verskeie aspekte van die bestuurspraktyke kontroleer. Probleme kan dus vroegtydig waargeneem word, onderlinge verwantskappe kan geëvalueer word en die bedryf kan oor 'n termyn van 'n jaar gevolg word om byvoorbeeld seisoens-, voedings- en bestuursinvloede te evalueer.

Ons is van mening dat hierdie vorm van evaluering 'n geïntegreerde benadering voortbring, sodat die tekens, uitgewys deur rektale ondersoeke, na faktore soos bestuur, voeding, estrusobservasie en inseminasietegnieke teruggevoer kan word.

Algemeen

Na afloop van bogenoemde ondersoeke kan tyd ingeruim word vir algemene aandag aan kalwers, verse en droë koeie[13]. Tydens hierdie inspeksie word aandag aan die kondisie van diere, behuising, voeding, bestuur en algemene gesondheidsprogramme soos parasietbeheer en entstofprogramme gegee. Verse se groeitempo kan van tyd tot tyd met neergelegde doelwitte vergelyk word. Dit kan deur visuele kondisiebeoordeling, gebruikmaking van 'n maatband of deur meting van die skouerhoogte uitgevoer word. Waar die nodige fasiliteite beskikbaar is, kan die werklike massa van verse bepaal word. Groeitabelle vir verskillende rasse is beskikbaar[13 18 22].

OPSOMMING

Suiwelboerdery is ongetwyfeld een van die mees komplekse boerderyondernemings. Dit vereis 'n groot kapitaaluitleg, intensiewe arbeid en vernuftige bestuur. Die suiwelboer sal in die toekoms genoodsaak wees om elke vorm van tegnologiese ontwikkeling wat sy suiwelbestuur en produksie kan verbeter, te gebruik om sodoende die verhoogde eise ten opsigte van sake- en bestuursvernuf die hoof te bied[6].

Kuddegesondheidsprogramme moet goed beplan, effektief geïmplementeer en gereeld geëvalueer word. Dit moet op eenvoudige en praktiese riglyne gebaseer wees en vir 'n wye verskeidenheid van boerderypraktyke en omgewingstoestande aanpasbaar wees[13].

Baie tyd, moeite en geld word in vrugbaarheidsprogramme belê. Metritis, onaktiewe ovaria, ovariese siste en ander probleme is dikwels die fokus van aktiwiteite. Die voorkoms van hierdie probleme is genoegsame bewys dat risikofaktore 'n rol gespeel het. Ongelukkig geniet hierdie faktore dikwels te min aandag en word hulle rol onderskat. Daar word dan dikwels op antibiotika, uterine installasies en hormonale behandelings vertrou om hierdie kondisies te "beheer"[24].

Vrugbaarheidsprogramme moet voeding, bestuur, behuising, algemene gesondheid, kondisie van diere, produksie en kalfgesondheid integreer[17]. Vrugbaarheid is 'n belangrike onderdeel van melkkuddegesondheidsprogramme en vrugbaarheidsdata word gebruik om ekonomiese produksie te optimeer. Vir doeltreffende advies moet voldoende tyd aan evaluering van data toegeken word. Sodoende kan verwantskappe met ander aspekte waargeneem word. Die veearts het 'n strategiese rol as koördineerder en 'n interdissiplinêre spanpoging is noodsaaklik[4 11].

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Addendum I: Evaluasie: Dragtigheidsondersoeke

Kudde: Datum:

Dragtig			Negatief Aktief-Normaal		Abnormaliteite teenwoordig			
Id*	Kalwing – konsepsie	Verwagte kalfdatum	Id	Dae oop	Id	Dae oop	Diagnose	Behandeling

Totaal ondersoek :
Totaal + % dragtig :
Totaal en % negatief normaal aktief :
Totaal en % abnormaliteite teenwoordig :
Kommentaar :

*Id = Identifikasie van koei

Addendum II: Evaluasie: Post-partale ondersoeke

Kudde: Datum:

	Normale involusie	Subinvolusie	Endometritis	Siste	Serviks letsels	Trauma	Ander
Totaal							
%							

Gemiddelde TKP :
Kommentaar :

*Onder elke onderafdeling kom koei se identifikasie sowel as haar TKP (tussenkalfperiode).

Addendum III: Evaluasie: Oop koeie

Kudde: Datum

Dae oop	Ovaria			Uterus		Serviks letsels	Vergroeiings	Ander
	Aktief	Onaktief	Siste	Normaal	Endometritis			
45 – 60								
Totaal + %								
61 – 90								
Totaal + %								
91 – 120								
Totaal + %								
121 – 150								
Totaal + %								
>150								
Totaal + %								

Addendum IV: Algemene indekslys

Kudde:

1. **Datum:**									
2. **Samestelling van kudde:**									
Koeie in laktasie									
0 – 100 dae									
101 – 200 dae									
201 – 300 dae									
>300 dae									
Koeie droog en in kalf									
Koeie droog nie in kalf nie									
Verse									
<12 maande									
12 – 18 maande									
Dragtige verse									
Verse >18 maande; nie dragtig nie									
Suipkalwers – verse/bulletjies									
Bulle									
Ouderdom van kudde									
Koeie eerste laktasie									
Koeie 2 – 5 laktasies									
Koeie >6 laktasies									

Addendum IV: Algemene indekslys (vervolg)

	Kudde:								
3. **Melkproduksie:** Gemiddeld/dag									
Gemiddeld/koei/dag									
% Vet									
% Proteiene									
4. **Kalf en Vervangingsverse:** a) 0 – 24 uur Diarree									
Respiratoriese aandoenings									
Mortaliteite									
b) 24 uur tot 2 weke Diarree									
Respiratoriese aandoenings									
Ander aandoenings									
Mortaliteite									
c) 2 weke tot 2 maande Diarree									
Respiratoriese aandoenings									
Ander aandoenings									
Mortaliteite									
d) >2 maande Diarree									
Respiratoriese aandoenings									
Ander aandoenings									
Mortaliteite									
5. **Koeie:** Melkkoors									
Ketose									
Vrotpootjie									
Voedingsteurnisse									
Algemene siektes									
Mortaliteite									
Brucella/Tuberkulose +									
Koeie kalf met mastitis									
Koeie in melk mastitis									
6. **Uitskot:** Totaal									
Fertiliteit									
Produksie									
Uierprobleme									
Voetprobleme									
Ander									

Addendum V: Maandelikse ginekologiese indekslys

Kudde:

1. **Datum:**									
2. **Dragtigheidsondersoek**									
Koeie dragtig/Aantal ondersoek									
Verse dragtig/Aantal ondersoek									
Negatief aktief – koeie/verse									
Abnormaliteit bevind – koeie/verse									
3. **Kalwings – koeie/verse:**									
Distokie – koeie/verse									
Agtergeblewe plasenta – koeie/verse									
Doodgeborenes/Aborsie									
4. **Post-partaal**									
Normale involusie/Aantal ondersoek									
Subinvolusie									
Endometritis/Siste									
Trauma									
Serviks letsels/Ander									
5. **Oop koeie:**									
a) **45 – 60 dae:** Aantal ondersoek									
Stil/Ongereeld									
Vuil/Ander									
Evaluasie: Normaal aktief/Onaktief									
Endometritis/Siste									
Vergroeiings/Serviks									
b) **61 – 90 dae:** Aantal ondersoek									
Stil/Ongereeld									
3 + Inseminasies									
Vuil/Ander									
Evaluasie: Normaal aktief/Onaktief									
Endometritis/Siste									
Vergroeiings/Serviks									
c) **91 – 120 dae:** Aantal ondersoek									
Stil/Ongereeld									
3 + Inseminasies									
Evaluasie: Normaal aktief/Onaktief									
Endometritis/Siste									
Vergroeiings/Serviks									
d) **121 – 150 dae:** Aantal ondersoek									
Stil/Ongereeld									
3 + Inseminasies/Ander									

Addendum V: Maandelikse ginekologiese indekslys (vervolg)

		Kudde:								
Evaluasie:	Normaal aktief/Onaktief									
	Endometritis/Siste									
	Vergroeiings/Serviks									
e) **>150 dae:**	Aantal ondersoek/Totaal in kudde									
	Stil/Ongereeld									
	3 + Inseminasies/Ander									
Evaluasie:	Normaal aktief/Onaktief									
	Endometritis/Siste									
	Vergroeiings/Serviks									

ABSTRACT SAMEVATTING

STUDIES ON THE ABILITY OF DIFFERENT STRAINS OR POPULATIONS OF FEMALE *RHIPICEPHALUS EVERTSI EVERTSI* (ACARINA: IXODIDAE) TO PRODUCE PARALYSIS IN SHEEP

Simultaneous infestation of 3 – 6 month-old Black-head sheep with 15 South African wild strains of *Rhipicephalus evertsi evertsi* males and females as well as a strain from Rwanda clearly showed that all strains are capable of inducing paralysis.

Assessment of the infestation-rate of engorging female ticks during the period that their mass ranged between 15 and 21 mg/kg sheep body mass indicated that toxicity is quantitatively identical and exhibits no intraspecific gradations. The period between the beginning of infestation to the manifestation of the first clinical symptoms is, however, strain dependent: 4 days for ticks from Warmbaths, and at least 5 days for all other strains. (Gothe, R. & Bezuidenhout, J.D., 1986 Studies on the ability of different strains or populations of female *Rhipicephalus evertsi evertsi* (Acarina: Ixodidae) to produce paralysis in sheep. *Onderstepoort Journal of Veterinary Research*, 53, 19 – 24 (1986).)

ABSTRACT SAMEVATTING

THE EFFICACY OF HYPERIMMUNE SERUM IN THE TREATMENT OF SWEATING SICKNESS

Natural and experimental cases of sweating sickness were treated using a hyperimmune serum as specific treatment and hyperimmune serum combined with symptomatic and supportive treatment based on the clinico-pathological changes observed in cases of sweating sickness. The treatment regimes were found to be highly effective in pigs and sheep as well as in calves, although recovery in the latter species was slower. (Oberem, P.T., Van Amstel, S.R., Matthee, O. & Bezuidenhout, J.D., 1985. The efficacy of hyperimmune serum in the treatment of sweating sickness. *Onderstepoort Journal of Veterinary Research*, 52, 283 – 287 (1985).)

INSULIN DEFICIENCY AND METABOLIC DISORDERS IN HIGH-YIELDING DAIRY COWS**

D. GIESECKE**

ABSTRACT: Giesecke D. **Insulin deficiency and metabolic disorders in high-yielding dairy cows.** *Journal of the South African Veterinary Association* (1986) **57** No. 1, 67-70 (En). Institute of Animal Physiology, Veterinärstrasse 13, D-8000 München 22.

INTRODUCTION

The productivity of farm animals depend on metabolic performance. This is particularly obvious in high-yielding dairy cows, the productive metabolism of which exceeds by far that for maintenance of body mass. The factors limiting high lactation performance are physiological, ecological and economical in nature. Ruminants – in contrast to monogastric food animals like pigs and poultry – do not compete with man for food, because of their unique capacity of utilizing fibrous plant materials by means of microbial digestion in the rumen. At the same time, however, this outstanding digestive potential limits lactation performance, because rations with a high roughage content, allow only for modest milk yields. Thus by breeding for high milk production, the physiological advantage of roughage digestion will finally be lost as more and more grain and other highly digestible carbohydrate sources must be included in the rations in order to provide an optimum pattern and amount of glucogenic (propionate) and lipogenic (acetate, butyrate) precursors absorbed from the rumen. Limitations of lactation performance observed in highly developed countries in the recent years appear to be physiological in nature: although sufficient food of good quality is available, cows exhibit excessive mobilization of adipose tissue lipids at the beginning of lactation – the lipid mobilization syndrome[15] and eventually run into metabolic disorders of ketosis and related problems. Consequences resulting therefrom may be fatty liver and fat deposition in other tissues[13] as well as an increase in calving interval correlated with fatty liver[12]. The reason for lipid mobilization is generally explained by an "energy gap" in early lactation: high energy expenditure for milk production and/or insufficient food intake. Our own observations point to a problem of metabolic regulation related to insulin deficiency rather than input and output of energy.

LIPOLYSIS PRE-PARTUM – FIRST OBSERVATIONS AND HYPOTHESIS

Our observations were made on Holstein-Friesian cows at the Experimental Farm of the Veterinary Faculty.

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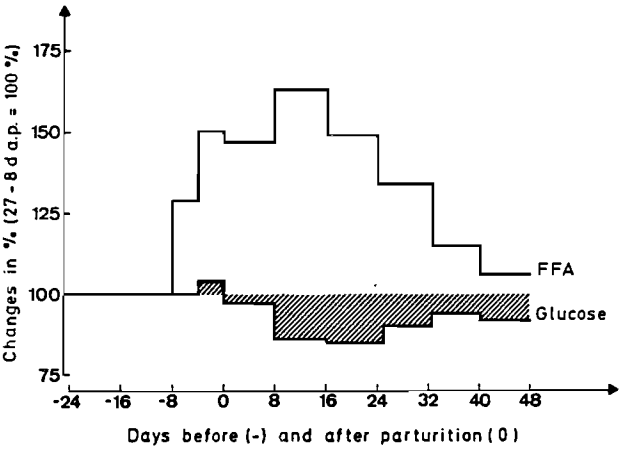

Fig. 1: Changes (in %) of plasma glucose and free fatty acids (FFA) in cows (n = 6) before (a.p.) and after parturition (o).

The cows were fed good quality maize silage and hay *ad libitum* throughout the day and 1 kg of concentrate/2,2 kg of milk produced in excess of 15 kg/day. The annual milk production averaged about 7 000 kg.

In the course of evaluating metabolites in blood plasma useful for characterizing metabolic stress situations, two observations had been noted: plasma free fatty acids (FFA) were much more sensitive to metabolic changes than plasma glucose, the metabolite preferred for clinical diagnosis, and the FFA level already increased in the week prior to calving and not as a consequence of high production demands (Fig. 1). The results suggested also, that the FFA level had largely decreased again before the peak of lactation performance was attained at about 6 weeks. Therefore, we suspected the hormonal regulation of metabolism to play a more important role as a primary event of lipolysis than energy expenditure of lactation.

Table 1: Influence of energy status during the feeding interval on the mobilization of free fatty acids (FFA) from adipose tissue lipds

Time after last feeding (h)	Plasma FFA (µmol/l)			
	$\bar{x}$	± SD	S^2	± CV*
4	256,2	33,3	1 108,9	13
12	436,2	131,5	17 292,2	29
significantly different ($p <$)	0,01		0,001	

*Coefficient of variation (%)

A highly sensitive reaction of FFA to metabolic changes was also obvious during the daily feeding cycle (Table 1). FFA had increased by 70% at 12 hours as compared to 4 hours after feeding, even though in ruminants nutrient absorption from the gastro-intestinal tract is known to proceed for many hours, so that a fasting state attained within 24 hours in monogastric animals will be achieved in ruminants only after 72 hours. It is also worth noting in the table that the variance is significantly higher at 12 hours after feeding – a good chance for the animal breeder to select cows which are able to handle their energy problems easier than others.

The metabolic role of FFA resulting from adipose tissue lipolysis has been reviewed elsewhere[6]. The release of FFA is subjected to a variety of lipolytic hormones like adrenalin, noradrenalin, bovine growth hormone, glucagon and others, all opposed to only one lipotropic hormone: insulin (except for the secondary lipotropic oestrogens). If the balance between these two groups is in favour of the former, lipolysis in the adipocytes will exceed re-esterification of fatty acids, which are then released into the plasma and bound to albumin as FFA. Observations in Holstein-Friesian cows as compared to crossbred beef cows[9] have suggested breed differences of plasma insulin levels which were definitely lower in the dairy cows. In our own observations some of the metabolic features of Holstein-Friesian cows e.g. a small increase of plasma glucose before calving – resembled in some way those of a mild diabetes as shown for goats under experimental diabetic conditions[14]. Therefore, we have suggested the working hypothesis, that insulin deficiency was the primary event leading to lipid mobilization[5].

PLASMA INSULIN AND ENERGY METABOLITES

Further experiments to examine the hypothesis on insulin deficiency were performed on 12 cows from about 3 weeks before to 6 weeks after parturition. In addition to plasma immune reactive insulin (IRI), the most important energy metabolites were also measured in order to characterize the metabolic state (Table 2). The dynamic changes are shown in a graph (Fig. 2).

In fact, the plasma level of insulin fell markedly: between 24 and 12 days before calving the decrease was 41% and after calving up to day 26, another 34%. Individual cows differed in the time course of changes which resulted in high variability; however, in all animals insulin deficiency was obvious.

Plasma glucose levels increased slightly before calving, as has been observed in an earlier experiment (Fig. 1), then decreased by 18%, a limited change because of proper regulation. FFA on the other hand, increased by 49% before calving already, but thereafter 5 – 6 fold values were observed. The strong reaction of this metabolite emphasized the close dependence on insulin levels and the lack of feed-back regulation. Finally, 3-hydroxybutyrate was found to follow mainly the changes of its precursor FFA. The second ketone body, acetoacetate, was omitted here, because it accounted for less than 10% of 3-hydroxybutyrate.

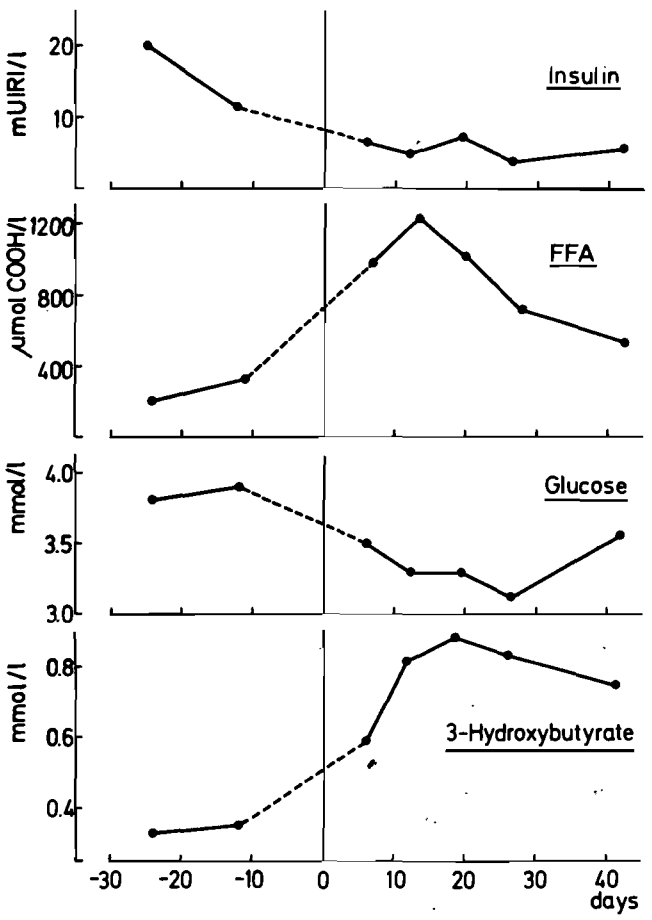

Fig. 2: Dynamic changes of insulin, free fatty acids (FFA), Glucose and 3-hydroxybutyrate in cows (n = 12) before and after parturition (o).

INSULIN RESPONSE AND RECEPTOR AFFINITY

The marked decrease of plasma insulin as demonstrated before, should have resulted from reduced insulin secretion by the pancreatic beta-cells if the condition was similar to insulin deficiency diabetes. The insulin

Table 2: Plasma insulin and energy metabolites in high-yielding cows

Measurements ($\bar{x}$ ± SD, n = 12)	– 24	– 12	6	12	19	26	41	p<[a]
Insulin (mU/ℓ)	19,5 18,4	11,5 5,6	6,5 8,1	5,0 4,2	7,7 8,9	4,9 2,5	6,1 4,5	0,05
Glucose (mM/ℓ)	3,8 0,5	3,9 0,6	3,5 0,6	3,3 0,3	3,3 0,6	3,1 0,4	3,6 0,5	0,05
FFA (µM/ℓ)	213 62	318[b] 185	964 302	1 220 406	993 455	696 244	512 174	0,001
3-Hydroxybutyrate (mM/ℓ)	0,32 0,10	0,34 0,08	0,58 0,24	0,81 0,50	0,88 0,37	0,83 0,36	0,74 0,27	0,001

[a] refers to values before and after parturition
[b] p < 0,05

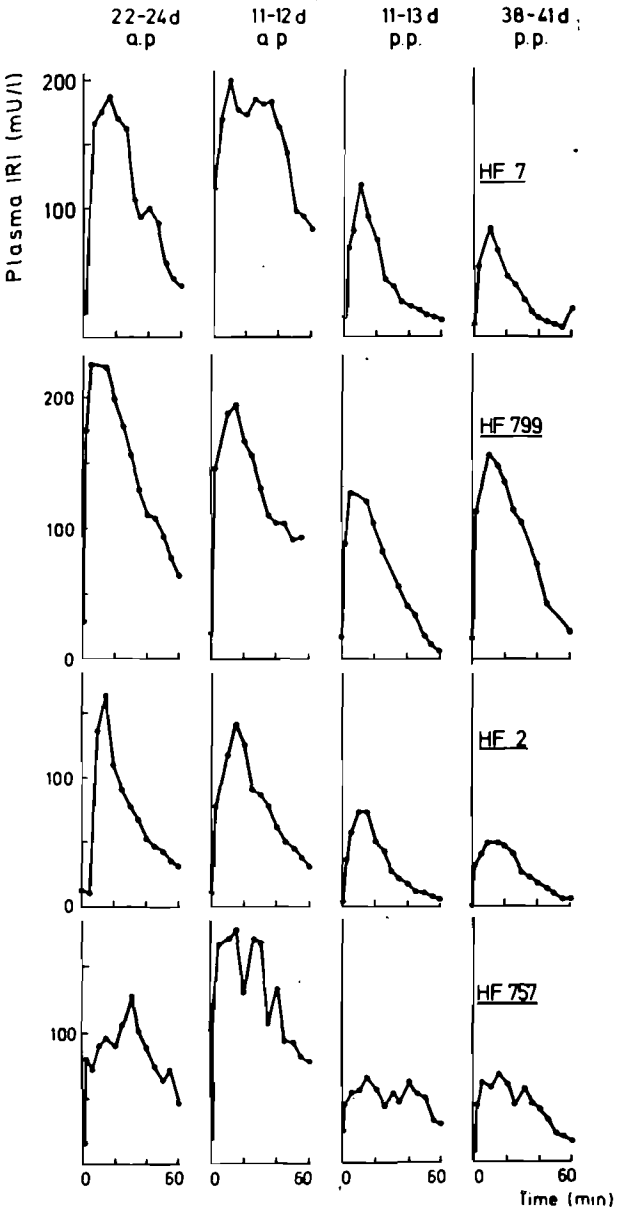

Fig. 3: Insulin response after glucose injection in 4 cows before (a.p.) and after (p.p.) parturition.

response following the intravenous injection of glucose (1 g/kg^{0,75} in 40% w/v solution) was measured in 3 cows for 60 minutes. The results show (Fig. 3) a more or less drastic decrease of the response curves after parturition accounting for 70, 44, 63 and 58% in cows HF 2, 7, 757, and 799 as compared to maximum values before calving. In one animal (HF 7) the response recovered at 6 weeks. A peculiar pattern of insulin response was observed in cow HF 757. According to general experience this type of curve may be attributed to the secretion of stored insulin (1st peak), followed by insulin from synthesis *de novo* (following peaks). There is, however, no definite proof for this interpretation. It appeared quite certain, however, that insulin response was markedly reduced after calving indicating not only a decrease in availability but also in the capacity of the insulinogenic system.

An important part of insulin action is the binding affinity of insulin receptors located on the surface of insulin-sensitive cells. Low binding affinity is characteristic of insulin resistance, i.e. a decreased response at the tissue level even if insulin is available. In a separate experiment we have investigated the insulin binding affinity of receptors on erythrocytes collected from 6 cows from the same herd. An insulin radioreceptor assay developed for human erythrocytes was used for this purpose.

The affinity is expressed as the reciprocal value of the half saturation constant of the receptor (1/HSC). In Fig. 4 these values are plotted together with the plasma insulin levels of the same cows sampled from 9 weeks before to 12 weeks after parturition.

Obviously, the insulin receptor affinity changed almost in parallel with plasma insulin levels. The former decreased sharply between 6 and 3 weeks before calving and increased again between 6 and 12 weeks after calving. The values between 3 weeks before and after parturition may have been even lower, however, no samples were taken during this interval.

If it is correct to assume that the reactions of insulin receptors on erythrocytes are representative for those of other insulin receptors, e.g. on adipocytes and on striated muscle cell membranes, then the results of Fig. 4 would indicate a generalized insulin resistance during the period between 6 weeks before and after parturition. This would mean that the decrease of plasma insulin observed is potentiated by the decrease of insulin receptor affinity making insulin deficiency more severe.

Principally, low levels of insulin favour the catabolic processes which increase the flow of glucose, lipids and amino acids into the lactating mammary gland. Thus, it would be tempting to use low insulin values as a parameter for the selection of high-producing lines of dairy cattle, as has been tried with test bulls under fasting conditions[8]. Indirectly, such a selection may have already occurred in Holstein-Friesian cows[9] and Brown Swiss cows[11] showing particularly low levels of insulin. As this hormone is ranging considerably higher in Jersey[2] and German Simmenthalers[7], the second important dairy breed in West-Germany, it appears doubt-

Table 3: Relationship between certain metabolic and reproductive disorders[3]

Secondary disorders	Primary Disorders						
	Fat cow syndrome	Milk fever	Dystocia	Retained placenta	Metritis	Displaced Abomasum	Ketosis
Dystocia	+	+					
Retained placenta	+	+	+				
Metritis	+	+	+	+		?	?
Displaced Abomasum	+	+	+	+	?		
Mastitis	+	+	+	+	+		?
Infertility	+	+	+	+	+	+	+

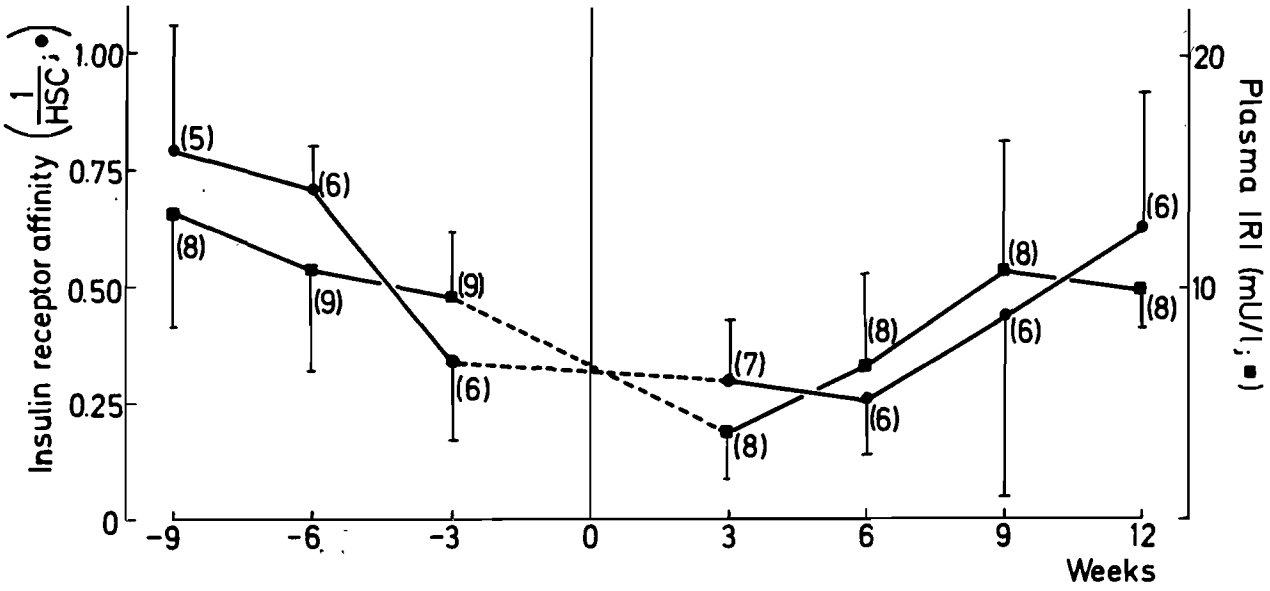

Fig. 4: Plasma insulin levels and insulin receptor affinity of erythrocytes from cows (n) before and after parturition. The insulin receptor affinity is given as the reciprocal value of the half saturation constant. $\bar{x} \pm SD$.

ful, if low plasma insulin values are an essential prerequisite for high lactation performance.

On the other hand, the problems resulting from insulin deficiency are severe and diverse:

- limitation of feed intake as indicated elsewhere[4,10] may be responsible for the "energy gap" in early lactation;
- excessive lipid mobilization causes lipid deposition in liver and other tissues[13]; our data on early lipolysis suggest that the regulation is out of control;
- fatty liver decreases the normal metabolic functions of the organ and the availability of lipoproteins for mammary lipid synthesis;
- ketosis resulting from increased hepatic uptake of FFA and decreased mitochondrial oxidation; other consequences such as ketoacidosis are well-known;
- decreased resistence to infections is a likely consequence, because synthesis of mucopolysaccharides (e.g. hyaluronic acid) is reduced in insulin deficiency;
- fertility problems resulting from fatty liver are prolonged calving interval and increased insemination index[12]. The close interrelationship of metabolic and reproductive disorders is demonstrated in Table 3.

Apart from insulin deficiency and excessive lipid mobilization, high-yielding dairy cows are prone to a variety of metabolic disorders resulting from high-concentrate feeding such as the low milk fat syndrome and fat cow syndrome, which are frequently interrelated, and last but not least, rumen acidosis.

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TO THE EDITOR — AAN DIE REDAKSIE

Opinions expressed in letters are not necessarily those of the South African Veterinary Association/Die menings uitgespreek in die briewe is nie noodwendig dié van die Suid-Afrikaanse Veterinêre Vereniging nie.

DOGMA EN DINAMIEK

Daar ontstaan dikwels in die biologiese wetenskappe, geskille waarin elke siening hom op die waarheid beroep. Basiese navorsing en die toepassings daarvan, gaan dan ook sekerlik oor die soeke en aanwending van dít wat waar is.

Dit blyk egter dat ondersoeke wat in biologiese wetenskappe uitgevoer word, grootliks resultate lewer wat as relatiewe waarhede na vore kom. Die "vastighede" waarna wel verwys word, is dikwels arbitrêr, deur die mens daar gestel en ook blootgestel aan relatiwiteit as alle faktore konsekwent in ag geneem word. Tog word die mens gedwing om wel sekere norme te beskou as "vas" of liewer aanvaarbaar, anders kan geen gevolgtrekkings gemaak word nie.

Biologie het te doen met die studie rondom die lewe en die lewe is 'n proses van beweging. Die vraag kan dus ontstaan of daar dan niks in hierdie wetenskap is wat werklik absoluut, vas en seker is nie? Sou daar dan gaan dogma, reël, wet of beginsel wees nie? Hoe sou toegepaste rigtings ontwikkel kan word, sonder enige vastigheid? Dit is tog logies dat dit juis dogma's is wat toepassings moontlik maak.

Daar skyn dus sprake te wees van botsende belange, naamlik beweging en beginsel; tendens en speling; wet en buigsaamheid; reël en variasie en uiteindelik dogma en dinamiek. Hierdie skynbare teenstrydighede maak dikwels die uitgangspunt uit van die stryd om die soeke na die waarheid.

Ons moet egter aanvaar dat beide dogma en dinamiek waarheid is, maar dat die begrippe eerder in ewewig met mekaar, as in teenstrydigheid met mekaar, gesien moet word. Daar bestaan dikwels ook nie soveel gebrek aan begrip van die ewewig, as 'n moedswilligheid om hierdie twee begrippe teen mekaar af te speel nie. As daar dus na kritiek gesoek word, na die een of ander kant, vir watter nie-wetenskaplike rede ook al, dan is daar altyd ruimte daarvoor. Altwee partye kan dus "reg" wees en beide kan "verkeerd bewys" word as daar nie ewewig tussen die standpunte gehandhaaf word nie. Om biologiese navorsing en toepassings te beoordeel, moet 'n balans tussen dogma en dinamiek gevind word.

Hierdie balans word gevind deur insig. Let op dat die term intelligensie* vermy word omdat intelligensie nageboots kan word deur oefening. Insig of dan wysheid is miskien moeiliker om voor te doen, want dit moet ontwikkel word. Intelligensie op sigself kan goeie resultate lewer en selfs presteer, sonder veel insig. Insig kan soms swakker presteer a.g.v. 'n gebrek aan oefening, maar insig kan probleme oplos. Insig laat 'n mens verstaan hoe alledaagse dinge werk wat almal raaksien en nie begryp nie. Insig openbaar 'n ondersoekende en pioniersgees, maak soms gebruik van onkonvensionele metodes en leef saam met die onverwagte in ondersoek en resultaat. Dit is oop vir die wonder van die biologie, maar kan tog perke stel sonder verstardheid. Dit gee ook rigting en leiding sonder om die pad byster te raak. Intelligensie luister graag wat ander mense sê, haal graag aan en kan saampraat met die bestes van die vak, maar insig gee eie opinie, staan dikwels alleen en maak nuwe stellings.

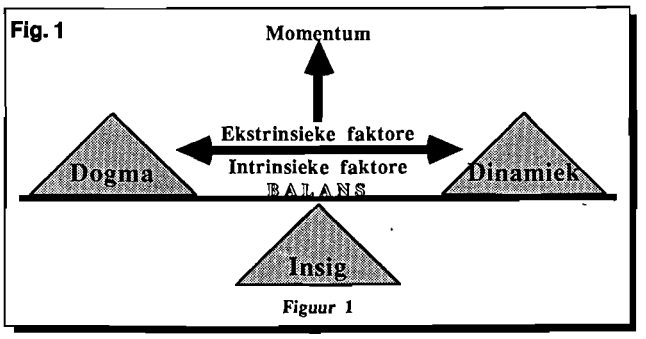
Terug by dogma en dinamiek, en ek bespreek kortliks laasgenoemde. Dit is die beweging in die biologie wat dit so fassinerend maak en wat selfs kenners van hulle vak by tye stom van verbasing laat staan – ás hulle ontvanklik is daarvoor. Die dinamiek verseker dat ons altyd maar net ten dele sal ken. Net sodra 'n mens meen dat hy 'n aspek van die biologie onder die knie het, dan moet hy nuwe verklarings en spekulasies vind. Dan tree die relatiwiteit weer sterk na vore. En tog, as alles relatief is, hoe kan ons vorder? Alles skyn so sinloos te wees – om maar net ten dele te ken. As daar 'n vraagteken oor elke bevinding hang, wie word dan oortuig? Hoe kan daar beplan word en vorentoe beweeg word, as ons swewend raak sonder grond onder ons voete?

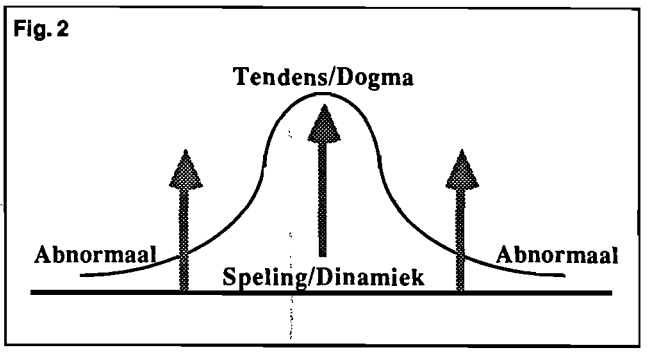
Nee, die dogmatiese aspek van die biologie is net so nodig vir begrip as die dinamika. Daarsonder sou geen beweging moontlik gewees het nie, want 'n mens kan net vorentoe beweeg as jy eers vastrapplek vir jou voet gekry het. Dán kan mens eers die volgende tree oorweeg, anders gaan mens val of sweef! Dogma moet rigting of koers aandui, anders kan daar nie toepassing wees nie.

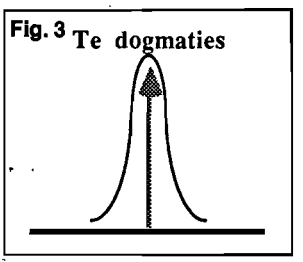
Die stryd tussen biologiese wetenskaplikes, selfs in praktyk, gaan dikwels daarom dat die een aan die dogma as enigste waarheid vashou, terwyl die ander een glo dat alles maar vloeibaar bly! Die waarheid kan egter nooit net een van hierdie sienings huisves nie. Ekstrinsieke en intrinsieke faktore het soms bepalende invloed op dogma sowel as dinamiek. Die waarheid moet êrens in die balans tussen dogma en dinamiek gevind word.

Die hiperkritikus wat elke syfer, stelling of resultaat wat die basis van die dogma vorm, so bevraagteken dat geen vordering kan plaasvind nie, maak misbruik van die dinamiek. 'n Mens kan dit vergelyk met die uitrafeling van 'n probleem waarvan die draad geen einde het

*Intelligensie word hier gebruik in die populêre sin van die woord. In die sielkunde of ander geesteswetenskappe, kan daar ander definisies bestaan, maar die term is vir hierdie geval gekoppel aan leke se beskouing van hoë punte in die weergee van geleerde of geoefende materiaal. Ek weier dus om betrokke te raak in 'n dispuut oor die gebruik van die terme intelligensie, insig en wysheid, want dit kan per definisie dieselfde wees. Die onderskeid vir die doel van hierdie skrywe word slegs getref, om 'n ander punt te maak. Die gebruik van die terme is van geen belang nie en as iemand beter terme kan vind wat dieselfde punt illustreer, des te beter.







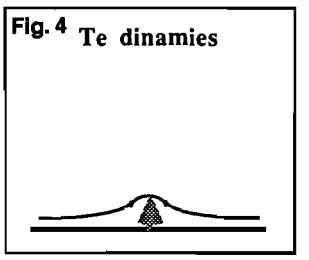


Fig. 1 – 4: *Stel die stellings diagramaties voor.*

nie. Dan word relatiwiteit verabsoluut en oortuig nie eers meer die kritikus nie. Hierdie houding is dikwels deel van 'n sinisme en onsekerheid omdat daar geen rigting en vastrapplek is nie. Net soos die krampagtige aanwending van dogma ongebalanseerd is, net so is 'n dinamiek met onbeperkte speling ongebalanseerd. Dit kom daarop neer dat variasie 'n grens moet hê van onaanvaarbaarheid, waar 'n mens kan sê dit voldoen nie meer aan die tendens nie. Daar moet dus 'n punt kom waar normaal (met speling) abnormaal word. 'n Mens kan dit met 'n veer vergelyk wat kan rek, maar ook uitgerek kan raak of breek!

Ons doel word dus as volg saamgevat:

1. Om insig te ontwikkel saam met die opstapeling van kennis (intelligensie).
2. Om balans te vind, terwyl mens bewus is van die werking van eksterne en interne faktore.
3. Om die biologie dinamies en optimisties te benader.
4. Om deur dogma die koers aan te dui, sodat die pad nie byster geraak word nie.

Al hierdie begrippe stem 'n bioloog, basies of toegepas, tot nederigheid. As dit al is wat ons hieruit kan leer, is dit genoeg.

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ECONOMIC BENEFIT OF ANTI-ENDOTOXIN THERAPY IN VETERINARY PRACTICE

Antibiotic therapy for the treatment of various primary or secondary Gram-negative bacterial infections in animals has been used for many years with all too often apparent failures and at a substantial cost to both veterinarian and client. The veterinarian must be made aware that all Gram-negative bacteria are coated with highly toxic, endotoxin (LPS, lipopolysaccharide). This LPS is released from the parent bacteria when it is killed, either by host immune defense mechanisms or by conventional antimicrobial therapy, and the LPS will continue to circulate and exert its toxic effects on the host.

The rumen and intestine always contain large amounts of Gram-negative bacteria and therefore these organs may be a continuous source of these bacteria with their LPS. The intact intestinal mucosa acts as the primary defence against both endogenous and exogenous endotoxins as these pathogens can gain entry from the intestine to the circulation with or without their bacteria where they will mimic the symptomatology of bacteraemia and sepsis.

Antibiotic therapy is not without disadvantages. Firstly, antibiotics do not affect the toxicity of the extremely chemically stable endotoxins. Therefore, when an animal dies as a result of so called "antibiotic failure", did it die because the antibiotic was not effective or was death due to an overwhelming endotoxaemia? In any case, the death of the sick animal represents an economic loss to both owner and veterinarian alike.

Animals treated with conventional antimicrobial drugs cannot be slaughtered until drug residues have been eliminated by the animal. Furthermore, these antimicrobial agents can gain entrance into the milk of dairy cattle, thereby making the milk unfit for human consumption, until all the antibiotic and its residues have been eliminated by the animal. Such waiting periods can represent further economic loss to the farmer.

Antibiotic and other antimicrobial therapy should be given for a course of at least five consecutive days. However, this treatment will often bring about a clinical improvement in less than five days and because of this improvement, the therapy is all too often discontinued. This can bring about the possibility of subclinical or chronic disease and the likelihood of antibiotic resistant bacterial strains becoming endemic.

A new and highly effective approach for the treatment of endotoxin mediated diseases and disorders (traumatic and post-operative) has been the development of endotoxin-specific antibodies. This immunotherapy with a polyvalent anti-LPS hyperimmune plasma ("ATOXIN", Fisher Vet., Sundry Vet., Medivet.) has reduced mortality and morbidity in a variety of diseases in large and small animals and was conspicuous in the rapidity with which it improved their clinical status. Only one, or at the most two, injections (24 hours apart) of anti-LPS are required to treat the endotoxic condition.

Another advantage of this specific anti-LPS therapy is the fact that it is potently bactericidal in effect and destroys a wide range of Gram-negative bacteria within seconds to minutes, thus possibly reducing the need for adjuvant five day antibiotics. Preliminary studies in cows and calves suffering with diarrhoeal disorders and pneumonic infections showed that they had vastly improved appetites within 48 hours after they received anti-LPS and fared no less well than those receiving antibiotics (Van Amstel. Unpublished observations).

In addition, since anti-LPS plasma contains only normal animal proteins, the meat and milk would not be subjected to the same stringent withdrawal periods as exists with present antimicrobial therapy. That is, anti-LPS therapy may result in a significant financial gain to the farmer by permitting slaughter sooner.

Anti-LPS therapy has been extremely well tolerated in the animals treated to date with no reported side effects. Furthermore, it can be given by any parental route and even per os in the animal that is a few hours old to assist in boosting the immune portfolia of the individual.

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TO THE EDITOR

AAN DIE REDAKSIE

DIACETOXYSCIRPENOL DETECTED IN MOULDY PIG FEED IN THE WESTERN CAPE

Over a four day period in July 1981, a haemorrhagic diathesis killed 9 out of about 300 10 – 16 week old pigs on a piggery near Somerset West. Mouldy feed was found in the feed-tank when the outbreak was investigated.

Several fifty gram samples of the mouldy feed were analysed for mycotoxins by a thin layer chromatography technique[7,8]. Several "suspect" specimens yielded a positive band resembling the toxin diacetoxyscirpenol (DAS) when compared to the DAS standard (Sigma Chemical Company, Saint Louis, Missouri, USA). Dr P.G. Thiel of the National Research Institute for Nutritional Diseases, Medical Research Council, Tygerberg, confirmed the extract to be DAS. Tests for warfarin, aflatoxin, rubratoxin, T-2 toxin, deoxynivalenol (vomitoxin), zearalenone, ochratoxin and sterigmatocystin were all negative.

DAS is a trichothecene mycotoxin and is a metabolite of various *Fusarium* spp., notably *F. tricinctum*. Bamberg et al., quoted by Weaver et al.[10] found that *F. tricinctum* produces its most toxic metabolites at low temperatures (8°C) in the presence of adequate moisture. July (midwinter) at Somerset West (in the winter rainfall region) would be ideal for *Fusarium* spp. growth.

Mouldy feed had also been found in the same tank two years previously (July 1979) at the time of another outbreak of a similar haemorrhagic diathesis when 34 weaned pigs died. The tank concerned was the only square-cornered bulk feed-tank on the farm. In both outbreaks, affected pigs had received feed from this tank. The feed was bulk-mixed, unmedicated and delivered weekly. Cracks had developed in the seams and corners of the tank and moisture had penetrated resulting in mould development. The tank was steam-cleaned and sealed. Mortalities stopped when the pigs received the new feed. Vitamin K supplementation was not given.

No blood coagulation studies were performed in these outbreaks. Nevertheless, the history, clinical signs and pathology closely resemble those described for a haemorrhagic syndrome in weaned pigs in the USA[4], New Zealand[1], Japan[6] and South Africa[3]. This syndrome has been ascribed to hypovitaminosis K and an experimental model is warfarin toxicity[5].

Mirocha et al.[2] detected DAS in feed associated with "haemorrhagic bowel syndrome" of pigs in the USA. In acute toxicity experiments[9], DAS given parenterally caused severe haemorrhagic bowel lesions.

The effects of acute DAS toxicity on coagulation parameters need to be examined. The role that DAS played in this outbreak is uncertain and it is conceded that the association of mouldy feed with the outbreaks recorded here may be purely coincidental.

We thank Mr E W P Heine of the Regional Veterinary Laboratory, Stellenbosch for the mycotoxin analyses and Dr P G Thiel for gas chromatography and helpful discussions.

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