RADIOLOGY OF SKELETAL DISEASE — Principles of diagnosis in the dog

JOE P. MORGAN

1st Edn. The Iowa State University Press, 2121S. State Avenue Ames, Iowa 50010. 1982 pp 106, illustrations 74. Price £12,50 (ISBN 0-8138-1508-8). Available in RSA through the Baillière Tindall agents, Peet Kruger, P.O. Box 13007, 1511 Northmead.

This is an excellent book for the student or clinician wanting to know more about the basic radiographic changes seen with diseases of the skeleton. It should not be used as a reference book for radiographic changes in specific skeletal diseases.

In this book he discusses the normal and abnormal radiographic appearance of soft tissues. The basic radiographic signs of joint diseases are discussed as well as the normal radiographic appearance of joints. The normal radiographic appearance of bone is discussed as well as a detailed description of the basic radiographic changes seen with diseases, affecting bones. All radiographic changes are very well illustrated by means of photographs of radiographs.

G.N. Eckersley

COLOUR ATLAS OF VETERINARY ANATOMY VOLUME TWO: THE HORSE

RAYMOND R. ASHDOWN and STANLEY H. DONE

1st ed. Baillière Tindall, London. 1987pp IX + 310, Figs 495. Price £49,50 (ISBN 0-7020-1238-6).

This book is intended for veterinary students and practising veterinary surgeons. The dissections were carried out at the Royal Veterinary College, University of London, by two experienced teachers of anatomy. The aim of the dissections and photographs is to reveal the topography of the animals as it would be presented to the veterinary surgeon during a routine clinical examination. Lateral views therefore predominate. Each of the almost 500 figures consists of a colour photograph of a body region or dissection, accompanied by a fully annotated coloured drawing. When necessary, information needed for interpretation of the photographs is given in the captions.

The body regions are dealt with under the following headings: the head, the neck, the forelimb, the thorax, the abdomen, the hindlimb, the foot and the pelvis.

The nomenclature is based on the Nomina Anatomica Veterinaria (1983). Latin names are used for muscles, arteries, veins, lymphatics and nerves. Anglicised terms are used for most other structures. There is a six-page index at the end.

Although the viscera are not dealt with systematically, special attention is given to the large intestine and the genital organs. Radiographs, joints and other synovial structures are not included in this atlas.

The technical quality of the publication is superb. The album-size pages are glossy and firm and the photographs and accompanying drawings are faultless. It is a pleasure to handle this book and I wholeheartedly recommend it to every equine practitioner and to veterinary students intent on an equine career.

Malie M.S. Smuts

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BERIA

Article/Artikel

ENDOTOXAEMIA IN RACEHORSES FOLLOWING EXERTION

B. BAKER*, S.L. GAFFIN**, MICHELLE WELLS**, B.C. WESSELS**, J.G. BROCK-UTNE**

ABSTRACT: Baker B.; Gaffin S.L.; Wells Michelle; Wessels B.C.; Brock-Utne J.G. **Endotoxaemia in racehorses following exertion.** *Journal of the South African Veterinary Association* (1988) **59** No. 2, 63-66 (En). Private practitioner, Summerveld Equine Hospital, Old Main Road, Gillitts, Natal, Republic of South Africa.

Endotoxins (lipopolysaccharides - LPS) and anti-endotoxin IgG antibodies were measured in racehorses before and after races of 1 000, 2 000 and 2 800 m. Results show that the mean plasma concentration of endotoxin increased significantly ($p < 0,02$) while the anti-LPS IgG concentration decreased significantly ($p < 0,005$) in all horses following the races. Pre-race and post-race anti-LPS IgG levels in racing-fit racehorses were significantly higher than in untrained horses ($p < 0,05$). The possibility therefore exists that training-induced stress leads to leakage of LPS into the systemic circulation which results in self-immunisation against LPS. The effects of plasma LPS and anti-LPS IgG concentrations on performance of racehorses require further studies.

Key words: Anti-endotoxin IgG, endotoxin, exercise, Thoroughbred

INTRODUCTION

The intestines of horses usually contain large amounts of gram-negative bacteria and their cell wall constituent, known as endotoxins or LPS (lipopolysaccharide). LPS is highly toxic and has a detrimental effect on the function of many organs, should it enter in excessive concentration into the systemic circulation[6]. LPS can mediate the release of potent vasoactive substances and neuroendocrine agents, and activate the complement and coagulation pathways[17]. Normally, however, the endotoxins remain in the gut. Various factors may lead to an excess leakage of endotoxins into the circulation which include reduced intestinal blood flow[13], reduced oxygen concentration in the blood[10] or an elevated body temperature[14], all of which may occur during strenuous exercise.

We have previously shown[1] in exhausted human marathon runners admitted to the medical tent for treatment after a 90 km race, that 81% had plasma endotoxin concentrations above the normal value of 0,1 ng ml^{-1}. Two per cent had values above 1ng ml^{-1}, the reported lethal human concentration[18]. There was also a highly significant negative correlation between plasma endotoxin and plasma anti-endotoxin IgG.

The object of this study was to ascertain the extent of endotoxaemia in racehorses following exertion, information which may be important in relation to their race performance.

MATERIALS AND METHODS

Horses were studied before and after a 1 000 m sprint ($n = 11$), a 2 000 m ($n = 5$) middle distance and a 2 800 ($n = 8$) m stayers race. The environmental conditions for each race were the same: slightly overcast with a temperature of approximately 21,6°C.

Venous blood samples were taken aseptically into heparinised tubes (40 I.U. ml^{-1}) both prior to and immediately after the race, for plasma LPS and anti-LPS IgG determinations. Under these conditions a recovery of 95,6% of LPS "spiked" into blood samples was obtained[19]. These determinations were done by the recently reported, highly reproducible chromogenic substrate modification of the limulus amoebocyte lysate assay (LAL)[7] and the enzyme-linked immunosorbant assay (ELISA) respectively [8 9]. Results are expressed in units of ng ml^{-1} relative to our standard *E. coli* 0111:B4. The blood samples were collected in heparinised, sterile pyrogen-free glass tubes (Falcon 2054, Becton, Dickinson Co. Oxnard, Ca.93030, U.S.A.) All blood samples were taken between 07h00 and 08h00 on the morning of the race with the horses unsaddled and resting quietly in their stables. Post-race samples were taken within 20 min after each race. The races commenced at noon and ended at 17h00. At no point were the animals considered to be abnormally excited by the prospects of the ensuing race.

A group of 50 racehorses not in training, acted as controls. Blood samples were again taken between 07h00 and 08h00 prior to exercise with the horses resting unsaddled and quietly in their stables. Twenty nine were at rest and 21 were undergoing light training.

Anti-LPS IgG Assay

Plasma was centrifuged for 5 min at 16 000xg and then diluted 1:100 in 0,1 M NaCl containing 0,05 Tris adjusted to pH 8,0 and 2% each of sheep and bovine plasma. The LPS-precipitable IgG of the equine plasma was then assayed with plates coated with 14 endotoxins at a concentration of 10 ug ml^{-1} in a carbonate/bicarbonate buffer at pH 9,6. Fourteen endotoxins were obtained from Difco (Detroit, U.S.A.), which had been extracted from the parent bacteria by the Westphal procedure[21]: *Salmonella typhimurium, S. minnesota, S. abortus equi, S. enteritidis, S. typhosa, Shigella flexneri, Serratia marscens, E. coli* 0127: B8, *E. coli* 0111: B4, *E. coli* 026: B6, *E. coli* 055: B5, *E. coli* 0128: B12, *Klebsiella pneumonia,* and *Pseudomonas aeruginosa.* The general ELISA microplate method used here has been previously described for alpha fetoprotein and other applications[2 5]. Microtitre plates (Dynatech M129 B) were coated with a mixture of endotoxins for 2 h at room temperature. Conjugate consisting of antibody to enquine IgG and horseradish peroxidase was made according to a method of Wilson and Nakane[22]. Results were measured at 492 nm and calibrated by an immunopreci-

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pitin reaction[12] with an equine anti-LPS IgG standard. With the latter standard, anti-LPS IgG was quantitated as described.

Chromogenic LAL assay

Plasma was diluted 1:4 with sterile pyrogen free water, heated for 10 min at 75°C, cooled on ice, and centrifuged at 16 000xg for 5 min. Duplicate samples of 50 μl heat treated plasma and 50 μl chromogenic *Limulus* amoebocyte lysate were incubated for 10 min in sterile depyrogenated glass tubes at 37°C. Thereafter 100 μl aliquots of chromogen (1:1 solution of buffer and chromogenic substrate, MA Bioproducts) was added and mixed. The tubes were incubated for a further 3 min. When adequate colour had developed, the reaction was stopped with the addition of 100 μl of 25% acetic acid. All testing was done under sterile apyrogenic laminar flow conditions.

Plasma blanks were run for each sample (50 μl heat-treated plasma, 150 μl sterile water and 100 μl 25% acetic acid), in order to account for the absorbance due to plasma alone. Optical density at 405 nm was read in a Titertek Multiskan plate reader. Results were standardised with calibration curves employing a reference LPS *E. coli* 0111: B4. The calibration curves were sensitive to LPS from 0,02 ng ml^{-1} and were linear from 0,02 to 0,2 ng ml^{-1} LPS.

Statistical analyses were performed with the unpaired and paired Student's t test. Significance was accepted at the 5% level. All data were expressed as means and standard errors of the mean.

RESULTS

Controls

The plasma LPS levels in the group at rest (n = 29) ranged from 0,0126 to 0,14117 ng ml^{-1} with a mean of 0,0554 ± 0,0054 (SEM) ng ml^{-1}. In the remaining horses (n = 21) blood samples were taken immediately after light training and the plasma LPS levels ranged from 0,0111 to 0,102 ng ml^{-1} with a mean of 0,0503 ± 0,0055 (SEM) ng ml^{-1} (Table 1). Anti-LPS IgG concentration ranged from 2,48 to 110,91 μg ml^{-1} with a mean of 35,01 ± 4,12 (SEM) μg ml^{-1} in the at rest group while the anti-LPS IgG concentration in the lightly trained group ranged from 6,84 to 108,6 μg ml^{-1} with a mean of 29,04 ± 6,03 (SEM) μg ml^{-1} (Table 1). These results did not differ from each other statistically and were therefore combined.

Table 1: Plasma LPS and Anti-LPS IgG levels in controls

Group	Plasma LPS (ng ml^{-1}) (mean ± SEM)	Plasma Anti-LPS IgG (μg ml^{-1}) (mean ± SEM)
Not in training (n = 29)	0,0554 (±0,0054)	35,010 (±4,121)
Racing fit (light training (n = 21)	0,0502 (±0,0055)	29,038 (±6,025)
Combined total (n = 50)	0,0533 (±0,0039)	32,502 (±3,468)

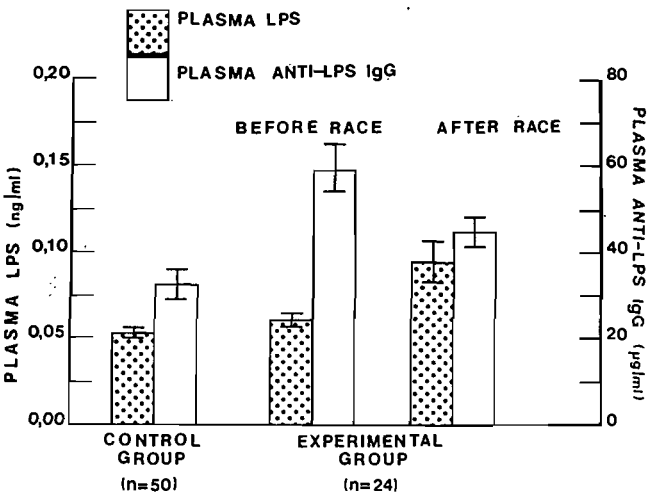

Fig. 1: Plasma LPS and Anti-LPS IgG levels in racehorses during light training (control) and in racehorses before and after a race

The combined results are shown in Fig. 1. The mean plasma LPS level was 0,0533 ± 0,0039 (SEM) ng ml^{-1} The average anti-LPS IgG concentration was 32,50 ±3,47 (SEM) μg ml^{-1}.

Racehorses

The results are also shown in Fig. 1. Due to the small numbers of samples the results from the 3 different distances have been combined. The results show that the mean plasma LPS concentration rose significantly from 0,062 ± 0,004 ng ml^{-1} to 0,094 ± 0,012 (SEM) ng ml^{-1} ($p < 0,02$). There was no clinical evidence of endotoxaemia in the racehorses after the race. They were all considered to be normal. Anti-LPS IgG titre dropped significantly from 59,30 ± 5,89 to 44,78 ± 3,53 (SEM) μg ml^{-1} ($p < 0,005$). The pre-race anti-LPS IgG titre of these horses in heavy training was significantly higher than that of the control group ($p < 0,001$).

With the small numbers utilised, no significant differences in the severity of endotoxaemia were found in relation to the different distances raced (Table 2). In other words, this study did not show that the endotoxaemia increased significantly with distance raced.

Table 2: Plasma LPS and Anti-LPS IgG levels in racehorses before and after various races (1 000, 2 000 and 2 800 m). Differences between distance raced, plasma LPS and Plasma Anti-LPS IgG were not significant

	Plasma LPS ng ml^{-1}		Plasma Anti-LPS IgG μg ml^{-1}	
Distance (m)	Before	After	Before	After
1 000 (n = 11)	0,0579 ± 0,0038	0,0835 ± 0,0087	68,250 ± 8,561	45,866 ± 4,604
2 000 (n = 5)	0,0642 ± 0,0098	0,0987 ± 0,0153	52,854 ± 18,755	46,802 ± 10,476
2 800 (n = 8)	0,0673 ± 0,0069	0,1075 ± 0,0332	51,044 ± 6,603	46,416 ± 6,230

DISCUSSION

This intestines usually contain large numbers of Gram negative bacteria and great amounts of endotoxins. Hypoxia, elevated core temperature or occlusion or reduction in splanchnic blood flow damage the intestinal wall and lead to excessive amounts of LPS being absorbed into the portal circulation. Since the hepatic reticuloendothelial system (RES) cannot remove the excessive LPS from the portal circulation, the LPS spills over into the systemic circulation with a detrimental effect on many organ functions[6 9 16 20]. When splanchnic blood flow is returned to normal, the RES function returns and the LPS levels return to base levels within 1 to 2 h[10 11 13].

In this study the severe stress of racing resulted in a significant rise of plasma LPS concentration in racehorses. LPS concentration is expressed as either ng ml^{-1} or, more recently, as enzyme units ml^{-1} (EU ml^{-1}), the latter to account for the fact that the same mass of LPS obtained from different sources may have different activities according to the LAL assay. When expressed in this way, the LPS concentrations rose from 0,310 to 0,470 EU ml^{-1}.

It is not yet known whether the observed increase in LPS and decrease in anti-LPS IgG on completion of the race, is of clinical significance in horses. Unpublished data from our group have shown plasma LPS concentrations in excess of 0,5 ng ml^{-1} in horses suffering from severe endotoxaemia (B Baker, R Katzwinkel 1986 unpublished data Summerveld Equine Hospital, Old Main Road, Gillitts, Natal). This compares with the overall lower values in horses in light training. The observed increase in LPS concentration may be due to the fact that during a race there is probably a reduction in splanchnic blood flow in a horse, as a result of a combination of adrenergic activation as well as an elevated core temperature[1]. Of interest was the lowering of the mean anti-LPS IgG concentration following exertion, most likely due to the binding, neutralisation and "consumption" of LPS which had entered the systemic circulation due to stress. One must presume that without the "natural" anti-LPS IgG, the LPS concentration would have been much higher and perhaps could have jeopardised the performance of such horses. Anti-LPS IgG concentration was significantly higher in pre-race samples compared to controls. The reason for this is thought to be that during heavy training small amounts of LPS enter the systemic circulation with a consequent antibody response.

Measurement of plasma LPS by the LAL gelation technique has been criticised in the past[4]. Some plasma samples are known to contain non-specific activators that may cause false-positive results in the LAL test. A possible cause of inhibition is preformed antibody to lipid A as suggested by Young[23]. Others have suggested that serum complement is required for neutralisation[15], but this system cannot operate in the heat treated plasma used in these studies. Several complicated methods have been proposed for the removal of plasma inhibitors of the LAL reaction[3] generally requiring conditions scrupulously free of pyrogen. Methods involving a combination of heating and diluting the sample are most convenient and effective, and have been almost universally adopted. Recent studies have shown that when properly performed, the chromogenic substrate modification of the LAL test provides a quantitive measure of LPS in human blood. When this method was used to compare the recovery of LPS from spiked pyrogen free saline and pooled equine plasma (measured simultaneously over an appropriate range of concentrations), no discrepancy in the values obtained in the two solutions was found[19].

Further studies in this field are required: An investigation of LPS and anti-LPS IgG levels from commencement of training to full fitness in racehorses and the relationship of these parameters to performance would be especially interesting.

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Article/Artikel

THE ARRANGEMENT OF THE MUSCLE LAYERS AT THE EQUINE ILEOCAECAL JUNCTION

SANET H. KOTZÉ*

ABSTRACT: Kotzé Sanet H. **The arrangement of the muscle layers at the equine ileocaecal junction.** *Journal of the South African Veterinary Association* (1988) 59 No. 2, 67-72 (En). Department of Anatomy, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

The muscle layers of the ileocaecal junctions of horses (n = 18), plains zebras *(Equus burchelli antiquorum)* (n = 3), mountain zebras *(Equus zebra zebra)* (n = 2), and a donkey were examined macroscopically and microscopically. The muscular tunic of the ileocaecal papilla was made up of 3 layers: an innermost circular layer which was a continuation of the circular muscle of the ileum, a central longitudinal layer which was a continuation of one part of the longitudinal layer of the ileum and an outermost layer, composed of two collateral semicircular lips, formed by the circular layer of the caecum. The other part of the longitudinal ileal muscle layer contributed to the dorsal caecal taenia. The circular and semicircular muscle layers contributed by the ileum and caecum respectively represented what could be termed as a *M. sphincter ilei,* which did not exist as a separate annular muscle confined only to the papilla as the name would suggest. The amount of intermuscular connective tissue increased in both these layers towards the tip of the papilla, while at the same time the layers decreased in thickness. This finding supports the view that their sphincteroid action would be less efficient and that an additional closing mechanism of vascular origin may be required at the ileocaecal papilla of the horse.

Key words: equines, zebra, ileocaecal junction, muscle layers

INTRODUCTION

Although the human ileocaecal junction and specifically the arrangement of its muscle layers, has been well described in the literature[1 5], relatively little work has been done on this subject in the horse. In an article mainly concerned with the endoscopic appearance of the caecal base in the live horse, Dyce & Hartman[2] describe the muscle layers and their possible functions, but give little detail on the exact arrangement of the different components. Although it is generally accepted in the literature that a definite *M. sphincter ilei* is absent in the horse, there is no detailed description of the muscle layers in this area to verify such an assumption.

Schummer[7] briefly discusses a network of veins present in an annular fold of mucosa in the ileocaecal papilla of the horse, which, upon engorgement, narrows the ileocaecal opening. He suggests that this network, together with the entire muscle coat of the ileum, acts as a functional sphincter and denies the existence of a separate *M. sphincter ilei.*

In the present study the muscle layers of the ileocaecal junctions of horses, plains zebras, *(Equus burchelli antiquorum),* mountain zebras, *(Equus zebra zebra)* and a donkey were studied macroscopically and microscopically. The ileocaecal junction of the horse has often been incriminated as a possible cause of equine colic due to obstruction of the ileum[3] or intussusception of the ileum into the caecum[6]. This study provides an anatomical basis for further investigations into its involvement in equine colic.

MATERIALS AND METHODS

Eighteen specimens of ileocaecal junctions from clinically healthy horses, the ages of which varied from 7 months to 35 years, were collected. These specimens were fixed in 10% formalin by immersion for a minimum period of 14 d. After fixation the specimens were trimmed to include the distal part of the ileum, the ileocaecal junction and the immediately adjoining part of the caecum.

Nine specimens were opened by making a longitudinal cut through the distal ileum, halfway between the mesentery and the ileocaecal fold, so that the incision transected the fibres of the circular muscle layer of the surrounding caecum perpendicularly, while also transecting the ileocaecal junction.

The remaining 9 specimens were opened along the mesenterial border of the ileum so that fibres of the circular muscle layer of the surrounding part of the caecum were cut longitudinally. The mucosa and submucosa were removed from the underlying layers of the tunica muscularis by scraping it off with a scalpel. The serosa was removed by peeling it from the underlying muscle layers, using a pair of fine forceps. The dissected specimens were photographed and a schematic drawing was made to illustrate the muscle arrangement (Fig. 1).

The same procedure was followed with specimens from plains zebras (n = 3), mountain zebras (n = 2) and a donkey.

For histological examination, specimens of the ileocaecal junction were taken from horses (n = 8) and plains zebras (n = 2) by making longitudinal sections of 5 mm in thickness to include the distal portion of the ileum, the ileocaecal junction and the caecum immediately surrounding the junction. These specimens were then fixed by immersion in 10% formalin. A further 8 specimens from horses were fixed by perfusion with Bouin's fixative and then by immersion in more Bouin's fixative. The specimens were routinely imbedded in paraffin wax, cut at a thickness of 6 μm and stained with Haematoxylin and Eosin. The sections were examined and photographed using a photo microscope (Wild M400).

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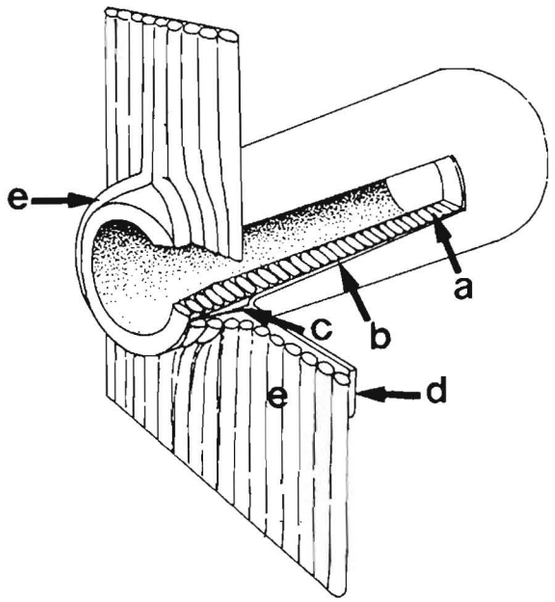

Fig. 1: A schematic drawing of the muscle layers at the ileocaecal junction with a wedge cut out between the dorsal mesenterial attachment and the ileocaecal fold. The circular muscle layer of the ileum (a), extends into the papilla, forming the innermost circular muscle layer of the papilla. The longitudinal layer of the ileum (b), divides at the base of the caecum to form (c), the middle longitudinal layer of the papilla. The other part extends on to the caecum, contributing to the dorsal caecal taenia (d). The circular layer of the caecum (e), forms a spindle-shaped opening allowing the ileum to telescope into the caecal wall thereby allowing the caecal circular muscle layers to form two semicircular lips reflected on both sides on to the ileal musculature of the papilla

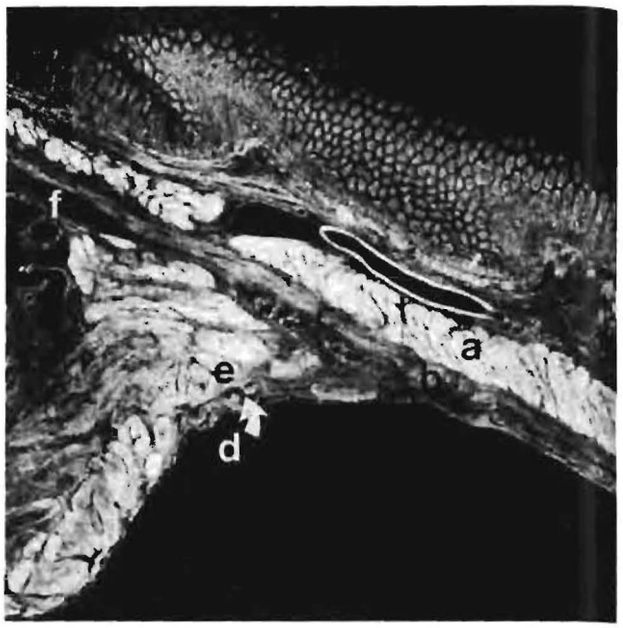

Fig. 2: The circular layer of the ileum is marked (a). The longitudinal layer of the ileum (b), divides to form the central longitudinal layer of the papilla (c), while the other portion, (d), continues on to the caecum to make a contribution to the dorsal caecal taenia (not shown). The circular muscle of the caecum is marked (e). Its continuation towards the tip of the papilla is interrupted by blood vessels (f)

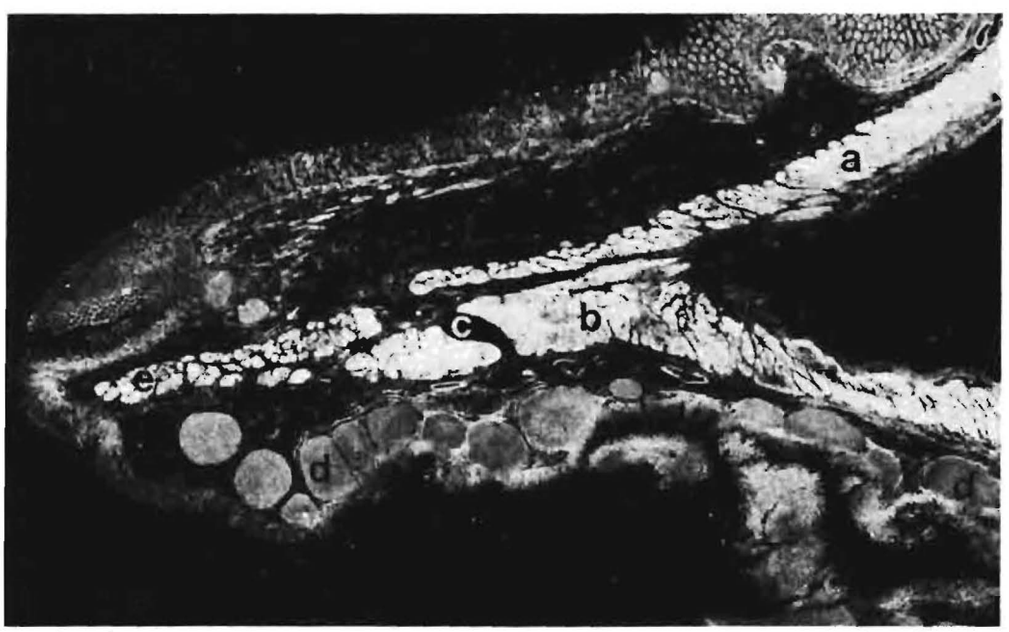

Fig. 3: The circular ileal muscle (a), decreases in thickness and increases in intermuscular connective tissue as the tip of the papilla is reached (e). At (e) the caecal circular muscle (b), fuses with the ileal circular muscle. A small vein (c), pierces the caecal circular muscle. Small submucosal lymph nodules are marked (d)

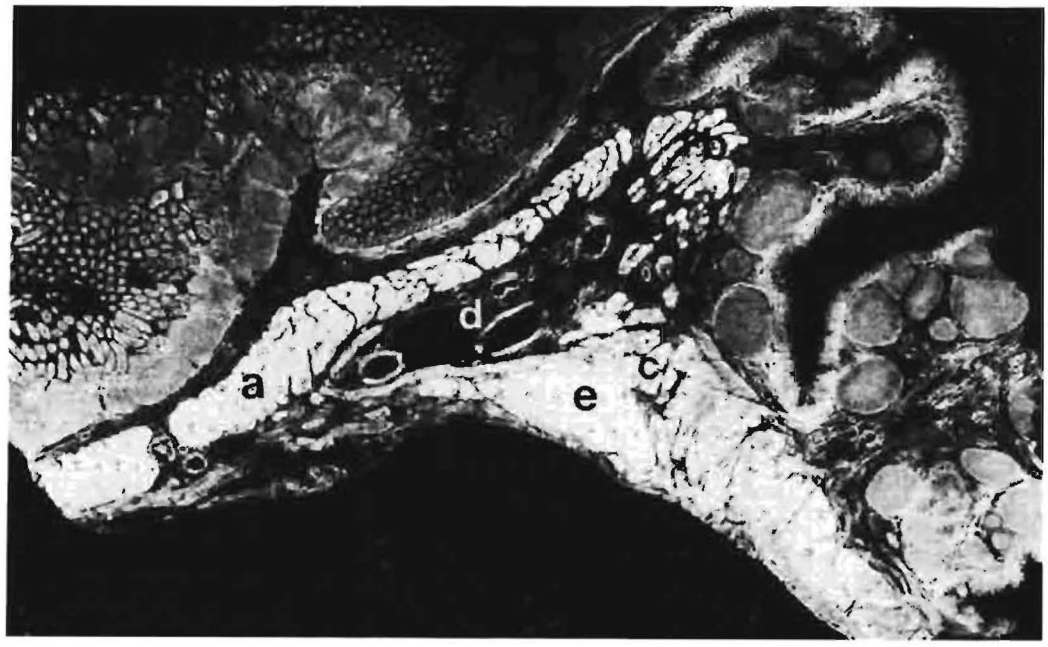

Fig. 4: The circular ileal muscle (a), blending with the caecal circular muscle (c), at the tip of the papilla (b). Blood vessels and nerves are present in the loose connective tissue between the layers at (d). The separate muscle bundle of the caecal circular muscle (e), is separated by a thin connective tissue layer from the rest of the caecal circular muscle

RESULTS

In all the specimens examined, the ileocaecal junction, apart from its mucosal, submucosal and serosal layers, consisted of the terminal portion of the ileal musculature, telescoped into and surrounded by the caecal musculature, thus forming an ileocaecal papilla. In this papilla the caecal circular muscle was merely pushed apart to form a spindle-shaped opening with two collateral, semicircular lips situated cranially and caudally, where they were reflected onto the papillary ileal circular muscle (Fig. 1). The muscle layers of the papilla thus consisted of an inner ileal circular layer, a central ileal longitudinal layer and an outer layer formed by two semicircular lips of the caecal circular layer. In the fresh specimens, the papillary form of the ileocaecal junction as described by Didio & Anderson[1] in humans and Schummer[7] in the horse, were observed in 80% of the cases. The more flaccid, less protruding form of the papilla was observed in the remaining 20% of specimens (Fig. 7). After removal of the mucosa there was a variation in height from 9 mm to 201 mm, measured from the base to the apex of the papilla.

At the base of the ileocaecal junction, the longitudinal layer of the ileum divided to form two parts: one continued into the ileocaecal papilla as the central longitudinal muscle layer of the papilla, which became very thin towards the tip of the papilla. The other part radiated onto the caecal wall. The fibres of this latter part made no contribution to the musculature of the papilla. All these fibres bent acutely in either a cranial or caudal direction to make a definite contribution to the dorsal caecal taenia. The fibres that were situated more cranially on the ileum were bent in a cranial direction onto the dorsal caecal taenia, while those that were situated caudally were bent in a caudal direction onto the same taenia (Fig. 5). On the mesenterial side of the longitudinal muscle of the ileum there were tiny orifices for the penetration of blood vessels (Fig. 5).

The muscle bundles of the ileal circular muscle, that also formed the innermost layer of the ileocaecal papilla, became finer with more intermuscular connective tissue, while the thickness of the layer diminished as it reached and extended into the tip of the papilla (Fig. 3 & 6). The latter fact together with the increase in connective tissue in this layer made dissection of the muscle bundles more difficult towards the tip of the papilla (Fig. 6). The looser appearance of the muscle bundles on histological examination (Fig. 3) was due to the increase of loose intermuscular connective tissue, which was also apparent in the papillary part of the caecal semicircular muscle lips.

As stated above, the caecal circular muscle layer split at the level of the ileocaecal junction to form a spindle-shaped opening for the passage of the distal ileum. This opening was situated so that lengthwise it lay parallel to the attachment of the ileal mesentery and parallel to the run of the muscle fibres of the caecal circular muscle layer (Fig. 7). Owing to the telescoping effect of the distal ileum into the caecal wall, the caecal circular layer was reflected onto the ileal circular layer on the cranial and caudal aspects of the papilla thus forming the outermost layer of papilla which consisted of the two semicircular components (Fig. 1). The caecal circular layer did therefore not encircle the ileal opening in the same fashion as did the ileal circular muscle layer. A separate muscle bundle of the caecal circular layer could be observed macroscopically at the base of the papilla close to the serosa in all the specimens that were examined (Fig. 6). Microscopically this muscle bundle was separated by a thin layer of loose connective tissue from the caecal circular layer, while its fibres were similarly directed. This muscle caused a thickening at the base of the papilla (Fig. 4).

The muscle fibres of all 3 papillary layers were fused

Fig. 5: The ileum (i), with fibres from the longitudinal layer which radiate out onto the caecum but is bent mainly into a cranial (a) or caudal (b) direction to join the dorsal caecal taenia (d). The medial caecal taenia is marked (c). The black arrows indicate the orifices through which blood vessels enter into the papilla. The white arrows indicate the bending of the fibres of the ileal longitudinal muscle to join the fibres of the dorsal caecal taenia

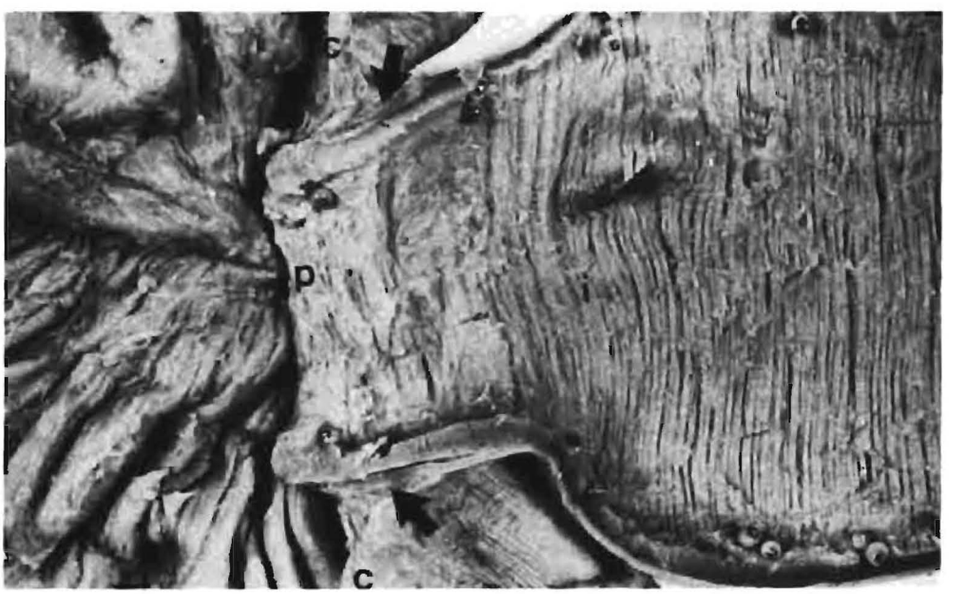

Fig. 6: The ileum (i), and ileocaecal papilla are transected along the dorsal mesenterial border. Fibres of the circular caecal muscle become finer as they reach the tip of the papilla (p). Owing to the increase of intermuscular connective tissue, the fibres become more obscure and difficult to isolate. The caecal wall is marked (c), while the arrows indicate the separate muscle bundle of the caecal circular muscle

towards the tip of the papilla (Fig. 3). Only at the base of the papilla could the layers be clearly distinguished, separated by thin strands of connective tissue containing small blood vessels and nerves (Fig. 4). The submucosa was well developed and rich in blood vessels.

The muscularis mucosa consisted of an inner circular and an outer longitudinal layer which remained the same thickness throughout the transition from ileum to caecum.

This arrangement of muscle layers at the ileocaecal junction applied to all the other equine species that were examined.

DISCUSSION

From the description above it is clear that the *M. sphincter ilei* is represented only by the ileal circular muscle layer and by the two semicircular laterally arranged components of the caecal circular muscle layer. Structurally the latter is not arranged as a true sphincter, but it must be assumed that functionally it must operate as a sphincter in conjunction with the ileal circular muscle. The *M. sphincter ilei* therefore does not exist as a separate muscle sphincter as the name would suggest, but is merely a slight rearrangement of the ileal

Fig. 7: The photograph shows the ileocaecal opening (o), formed in the caecal wall (c). The spindle-shaped opening is situated parallel to the direction of the muscle fibres of the caecal circular muscle layer. The attachment of the dorsal mesentery of the ileum is marked (a), while remnants of the ileocaecal fold is marked (b). This is an example of the more flaccid, less protruding form of the papilla as was observed in some specimens

and caecal musculature. Schummer[7] was therefore correct in stating that a separate *M. sphincter ilei* does not exist. The central longitudinal layer of the papilla from the ileal longitudinal layer, constitutes a dilator of the papilla.

The fact that the muscle layers became attenuated towards the tip of the papilla, as well as the presence of a well-developed submucosa rich in blood vessels, seem to suggest that closure of the papilla is not only due to muscular contraction. An additional mechanism may be provided by a papillary venous plexus, which upon engorgement may enhance the sphincteroid action of the muscle layers. The role of this plexus was briefly described by Schummer[7] and later observed by Dyce & Hartman[2] on endoscopic examination of the caecal base. A similar plexus exists in the human ileocaecal junction and is believed to act as a complement to muscular sphincter action in this area[4]. The exact arrangement, extent and disposition of this plexus in the horse is an area for further study.

The separate caecal muscle bundle situated at the base of the papilla coincides with the "sphincteroid tract" that Didio & Anderson[1] describe in the human ileocaecal junction. According to them the "sphincteroid tract" is responsible for closure of the orifice at the base of the papilla. In the horse this muscle may also have an additional sphincteroid action, seeing that the fibres are arranged as two semicircular lips, as is the case in the human. It also seems possible that the sphincteroid action may aid venous engorgement by cutting off venous return (thinner walled veins), but not arterial supply (thicker walls). This action may even be further enhanced if the papillary lumen were filled with ingesta. According to Schummer[7], the ileal musculature can shorten to one third of its length in the horse which facilitates transport of ingesta from the jejunum and simultaneously prevents reverse entry of gas from the caecal base.

The increase in intermuscular connective tissue between the muscle bundles of the ileal and caecal circular muscle layers towards the tip of the papilla should not have an effect on the contractability of the smooth muscle but might have a dampening effect on the sphincteroid action of the papilla if the simultaneous decrease in thickness of the layers is taken into account.

The more flaccid, less protruding form of the ileocaecal junction seen in about 20% of cases may be compared to the bilabial form of the papilla observed by Didio & Anderson[1] in humans, which they regard as a form of the papilla mostly seen in cadavers.

Müller & Smith-Agreda[5] have demonstrated that the muscle layers in the human ileocaecal junction contribute to the mesocolic taenia and that this arrangement plays a role in the functioning mechanism of the papilla. In the horse there is a definite similarity, where the dorsal caecal taenia receives a contribution from the longitudinal muscle of the ileum which may therefore have an effect on the movement of the papilla.

Muscle layers in the gut are typically described as a compressed (circular) or extended (longitudinal) spiral. Müller & Smith-Agreda[5] describe this arrangement as resulting in a three-dimensional muscular trellis in the human. In this study evidence of such a trellis could not be found.

Cases of ileocaecal intussusception, hypertrophy of the distal ileum, thromboarteritis of the ileocaecocolic artery and the frequency with which ileocaecal or jejunocaecal anastomoses are done, indicate that malfunction in this area may play a key role in the etiology of pathology of this and surrounding areas.

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Article/Artikel

LUCILIA CUPRINA INDUCED HYPERAMMONAEMIA AND ALKALOSIS ASSOCIATED WITH PATHOLOGY IN SHEEP

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ABSTRACT: Guerrini V.H.; Bell M.A.; Murphy G.M. ***Lucilia cuprina* induced hyperammonaemia and alkalosis associated with pathology in sheep.** *Journal of the South African Veterinary Association* (1988) **59** No. 2, 73-76 (En). Vetsearch Australia, 173 Chatswood Road, Daisy Hill, Queensland, 4128, Australia.

Gross and microscopical examinations were done in 12 sheep infested with *Lucilia cuprina* larvae. All the sheep became hyperammonaemic and alkalotic. Six sheep died and 6 survived, one being moribund before euthanasia. Necropsy revealed severe acute dermatitis and congestion of the liver, heart, kidney, spleen and lungs. Ecchymotic haemorrhages were present in the heart, kidneys and adrenal glands of severely infested sheep. Microscopical examination showed mild to severe vacuolation of the central nervous system in all sheep. In severely infested sheep, skin, lung, heart, kidney, and liver tissues were infiltrated by mononuclear cells, neutrophiles or lymphocytes. Fibrin thrombi were present in vessels. Elevated concentrations of lipid soluble ammonia in blood were positively correlated with tissue abnormality and early death.

Key words: *Lucilia cuprina*, blowfly strike, sheep, hyperammonaemia

INTRODUCTION

Infestation of sheep with *Lucilia cuprina* larvae can lead to changes in the body resulting in death[3]. Infested sheep develop an increased rectal temperature and respiratory rate, accompanied by tachycardia, anorexia and oliguria[3 9]. Haematological examination of infested sheep reveal degeneration of white cells[6]. Necropsy findings in severely infested sheep include changes consistent with severe toxaemia[3]. As yet, it is not known why these changes occur. It is, however, known that *L. cuprina* larvae produce ammonia[16] and up to 120 mmol ℓ^{-1} ammonium has been detected in fluid taken from infested sites on sheep[9]. The toxic effects of ammonia on living tissues are well known[24], yet a link between ammonia and skin or systemic pathological changes in infested sheep has not been investigated. Because infested sites on sheep are alkaline[9], there is a high proportion of lipid-soluble larval ammonia. This ammonia may cause the local and systemic toxicity[24 25] in infested sheep. The purpose of this report is to describe *Lucilia cuprina* larvae-induced hyperammonaemia and alkalosis associated with pathology in sheep.

MATERIALS AND METHODS

The procedures used were approved by The Animal Ethics Committee of The Animal Research Institute, Department of Primary Industries, Yeerongpilly, Australia. The utmost care was taken to avoid distress and suffering to the animals. Twelve long-woolled adult Merino wethers, aged 2-3 years were used. The sheep were placed in a room with natural light and ventilation, 4 weeks before larval implant for acclimatisation. The sheep were kept in metabolism cages and each fed 700 g lucerne pellets and provided with 9ℓ of drinking water daily between 07h00 and 09h00.

Each sheep was implanted on both sides of the points of the shoulder with 4 400 first stage *L. cuprina* larvae on Day 0, 1 500 on Day 1, 2 200 on Day 2, 3 100 on Day 3 and 5 100 on Day 5 of the experiment by a previously described method[3]. At death or before euthanasia, the infested sites were sheared and the area of skin infested was measured.

Blood samples were collected from the jugular vein on Day 6 and Day 2 before the first larval implant, and on Days 1, 2, 3, 4, 5, 6, 7 and 8 after implant. Paired carotid and jugular blood samples were collected in 5 of the 7 sheep that survived to the morning of Day 8. Samples for blood pH and base excess measurements were collected by methods described previously[15].

Jugular blood was analysed for ammonia using a kit (Ammonia Mono-test, Boehringer Mannheim). Blood base excess and pH were determined with a blood-gas analyser (Gilford 1312, Gilford Instruments). The concentration of lipid soluble ammonia in blood was calculated from the ammonium blood concentration, blood pH and the disassociation constant for total blood ammonia corrected for rectal temperature[1 23].

Complete necropsies were performed on each animal and tissues were fixed in 10% formol saline. Specimens were collected of skin, entire brain, lymph node, lung, heart, liver, kidney, adrenal, spleen, rumen, abomasum, duodenum, jejunum, urinary bladder and skeletal muscle. Coronal cuts of the brains were made routinely across the cruciate sulcus, the cerebellum and pons. Paraffin sections were made of all tissues and these were stained by haematoxylin and eosin.

Pre-implant data were compared with post-implant data by two-way analysis of variance.

RESULTS

All the sheep became infested by Day 2 and one died on Day 6, 4 on Day 7 and one on Day 8. The remaining 6 sheep were euthanased on Day 8. By Day 6 and 7, an average of 7 300 ± 3 179 larvae had left sheep that died, whereas only 2 961 ± 2 887 had left those that survived.

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Two days after the first larval implant, all the sheep began to show varying degrees of depression, hyperthermia, dyspnoea, anorexia, hypodypsia and oliguria which continued until death or euthanasia. In sheep that died, these signs were more severe and twitching of ear and eye muscles as well as frequent prostration occurred. Rectal temperature and heart rate increased significantly ($p < 0,05$) by Day 2 in sheep that died, and by Day 3 in those that survived. Respiratory rates increased ($p < 0,05$) in both groups but water intake and urine output declined significantly ($p < 0,05$) only in those which died.

Extensively inflamed and abraded or undermined areas occurred on the skin of the 6 sheep that died (3 033 cm^2; range 2 200 – 3 200 cm^3) but damage was less extensive in the 6 surviving sheep (1 676 ± 772 cm^2; range 1 040 to 3 200 cm^2). A gross observation common to infested sheep was generalised congestion of lungs and liver and engorgement of peripheral costal veins. In severely infested sheep (n = 7) haemorrhagic foci were observed in either lungs, kidneys, adrenals or heart. One sheep had pneumonia and fibrosis of the liver. Pericardial effusion was observed in 2 sheep and excessive white froth in the trachea and bronchi of 6 sheep.

The skin revealed severe (n = 11) or moderate (n = 1) acute dermatitis characterised by desquamation, ulceration, necrosis and neutrophil infiltration of the dermis, congestion of blood vessels and dilation of lymphatics. The lungs showed oedema and atelectasis (n = 9), and thickening of the alveolar septae (n = 6). In more severe cases there were microthrombi in lymph vessels, fibrin thrombi in large blood vessels, and infiltration of the lung parenchyma by mononuclear cells (n = 2). The myocardium of more severely infested sheep was congested (n = 6), some with myocardial haemorrhage infiltrated by mononuclear cells and neutrophils and loose fibrin thrombi in vessels (n = 4). The kidneys of severely affected sheep showed congestion (n = 2), and mild pyelitis characterised by infiltration of neutrophils and lymphocytes and loose fibrin thrombi in vessels (n = 4). The livers were congested (n = 11) and there was degeneration of hepatocytes (n = 4). Moderate infiltration of mononuclear cells, lymphocytes and macrophages surrounded some bile ducts (n = 2). Examinations of the lymph nodes of 2 sheep showed multiple vacuoles and neutrophils were seen in the parenchyma of the spleen.

In severely infested sheep adrenal lesions included haemorrhages in the zona fasciculata (n = 4), congestion of the zona reticulata (n = 1) and congestion of the vessels of the medulla (n = 1).

Table 1 summarises the severity of brain and other tissue lesions, the mean lipid soluble ammonia concentration in venous blood after the second day of infestation until death or euthanasia and the mean terminal lipid soluble ammonia concentration from samples taken approximately 12 h before death or euthanasia. Spongy degeneration of the white matter tracts of the brain, particularly the hind brain region was present in 9 sheep and milder degeneration occurred in 3 sheep.

Total jugular ammonium (non-lipid soluble and lipid soluble ammonia) and calculated lipid-soluble ammonia increased ($p < 0,01$ to 0,0001) at Day 2 in sheep that died and Day 3 ($p < 0,01$ to 0,001) in sheep that were euthanased. Total jugular ammonium was higher ($p < 0.05$) in sheep that died than in sheep that survived during Days 5, 6 and 7, and lipid soluble ammonia concentration was higher ($p < 0,05$) on Days 6 and 7. Increases in jugular blood pH in sheep that died were significant ($p < 0,05$) on Days 3, 4 and 6 and on Day 5 ($p < 0,01$) and in sheep that survived, on Days 3 and 4 ($p < 0,05$). Increases in base excess were significant ($p < 0,05$) in sheep that died on Days 4 and 5 but there was no significant increase in sheep that survived.

Sheep died consistently within 12 h after venous total jugular ammonium concentration exceeded 200 $\mu mol\ \ell^{-1}$. Sheep with total blood ammonium concentration lower than 200 $\mu mol\ \ell^{-1}$ did not die, although one with a concentration of 197 $\mu mol\ \ell^{-1}$ was moribund. Paired arterial and venous ammonium in one sheep that

Table 1: Pathological change in tissues, time of death (hours) and mean and terminal jugular lipid soluble ammonia concentration ($\mu mol\ l^{-1}$) in sheep infested by *Lucilia cuprina* larvae

Sheep*	Skin	Liver	Brain	Adrenal	Kidney	Heart	Spleen	Lungs	Time of death (h)	Blood ammonia Mean	(NH_3) Terminal
1	+ + + +	+ +	+ + +	+ + +	+ + +	+	+	+ +	132	6,6	10,6
2	+ + + +	+	+ + + +	+	–	+ + +	+	+ + +	142	4,9	8,5
4	+ + + +	+ +	+	+	+	+	ND	ND	120	5,9	9,4
5	+ + + +	+ +	+ + + +	+	+ + +	+ + +	+	+ + +	96	8,8	11,0
8	+ + + +	+ + +	+ +	+	+ + +	+ + +	+ + +	+ + +	106	8,5	10,9
9	+ + + +	–	+ +	–	+ + +	–	–	+ +	106	6,9	12,1
3	+ + + +	+ + +	+ +	–	–	–	–	+ +		4,3	7,9
6	+ +	–	+ + + +	–	–	–	–	–		4,1	7,1
7	+ + + +	+ +	+ + + +	+	–	+ + +	–	+ + +		5,9	7,8
10	+ + + +	+	+ + + +	–	–	–	+	+		4,9	4,5
*11	+ + + +	+	+ + + +	–	+	–	–	+ +		5,1	8,8
12	+ + + +	+ +	+ + + +	–	–	–	+	+ +		4,5	5,4

* Sheep 3, 6, 7, 10, 11 and 12 were killed 144 h after the second day of infestation. Sheep 11 was moribund before euthanasia. Score of – = no change, + = slight change (eg. congestion), + + = greater change (eg. oedema, vacuolation), + + + = evidence of inflammation (eg. fibrin clots, haemorrhage, cell infiltrations, severe vacuolation), + + + + = severe change (eg. dermatitis). Time of death = from the second day of infestation (2 days after the first larval implant). Mean jugular ammonia concentrations from Day 2 to death or euthanasia. Terminal jugular ammonia concentration taken 12 h before death or euthanasia. ND = Not done

died 2 h later, reached levels of 416 and 298 μmol ℓ^{-1}, respectively. Mean arterial and venous blood ammonium in the remaining sheep that survived until euthanasia were 102 – 195 μmol ℓ^{-1} ($\bar{x}$ = 154; SD = ± 13) and 102 – 159 ($\bar{x}$ = 123 SD ± 20) μmol ℓ^{-1} respectively.

DISCUSSION

Sheep infested by *L. cuprina* became hyperammonaemic, alkalotic and hyperthermic. Consequently, compared with pre-implant values, the mean calculated concentration of lipid soluble ammonia in jugular blood increased approximately 9 times in sheep which died, and 5 times in those that survived. It is well established that high levels of lipid soluble ammonia in blood produce toxicity as this form of ammonia enters cells readily[15]. Total ammonium in blood is 98,5% insoluble (ionised) and 1,5% soluble (non-ionised) at normal blood pH (7,4) and temperature (37°C)[15 25]. Total ammonium blood concentrations therefore do not accurately represent the degree of ammonia toxicity, whereas the concentration of lipid-soluble ammonia in blood does[5 15 25].

It has been postulated and shown that chronic hyperammonaemia produces spongy degeneration and vacuolation of the white matter tracts of the brain in sheep[10]. This brain lesion has been reproduced by intravenous administration of ammonium acetate in 14 sheep[12]. In the present study, all sheep had spongy degeneration in varying degrees of the white matter tracts of the brain which is physical evidence of ammonia toxicosis in sheep. Moreover, this lesion is found in few other conditions[10 11 13].

Ammonia concentrations tolerated by the brain are minimal compared to other organ systems[15] and there is evidence that ammonia interferes with enzyme function in cells by increasing intracellular pH[5].

Clearly, it was the sheep with the highest mean and terminal lipid-soluble ammonia concentrations which died earliest and had most tissue damage (Table 1). Sheep with mean ammonia values below 5,9 μmol ℓ^{-1} or terminal values below 8,5 μmol ℓ^{-1} did not die and revealed less pathology. Sheep No. *11* (Table 1) had a terminal lipid-soluble ammonia blood concentration of 8,8 μmol ℓ^{-1} but was moribund at the time of euthanasia. The appearance of spongy degeneration and the development of high blood ammonia levels accurately reflected health and tissue abnormality.

The brain[13], lungs, heart, liver and kidneys[7 8 21 22] have been shown to be particularly sensitive to ammonia toxicity in sheep. Tissue damage in infested sheep mostly involved the skin, brain, lungs and liver. Congestion was a constant observation in all sheep and haemorrhages were noted in sheep that died. The hepatic changes in sheep infested with *L. cuprina* larvae were similar to those described previously in sheep intoxicated with ammonium salts.

The congestion and vascular damage in the kidneys in the present experiment have also been reported in sheep given large doses of urea[8 21 22].

Hyperaemia, oedema and thickened alveolar septa in the lungs were similar to those attributed to chemical irritants[22]. Oedema of the lungs is a common observation in ammonia toxicosis[4 17 21 22].

Haemorrhage was observed in many of the tissues of infested sheep. Disruption of the vascular endothelium and the presence of fibrin clots in vessels of particularly the lungs and kidneys, suggest direct damage to endothelial cells[21]. These changes were consistent with disseminated intravascular coagulation[6 14].

Ammonia produces a variety of effects in sheep depending on the ammonium salt administered, the rate of administration and the species used[2 18 19 20]. In sheep, ammonia toxicity has been reproduced mostly by acute oral overdosage of urea or ammonium salts[2 19 20 21 22]. Few studies have dealt with the effect of chronic ammonia administration in sheep[11]. Acute increases in blood ammonia produce large increases in the circulating levels of blood adrenaline concentrations in ruminants[2 20]. Hyperthermia, tachycardia and dyspnoea occurred in sheep and goats infused with a variety of ammonium salts[21]. The clinical signs in sheep infested with *L. cuprina* were thus similar to those described previously in hyperammonaemic sheep.

High concentrations of lipid-soluble ammonia on skin have a toxic effect and could give rise to liquefaction of the skin[17]. The severe lesions on the skin of infested sheep were characterised by a loss of epidermis, ulceration, necrosis and congestion of blood vessels suggesting a severe toxic challenge. It is thus possible that lipid-soluble ammonia excreted by *L. cuprina* larvae[16] on to the skin was involved in the dermal lesions in infested sheep. It has been postulated[9] that ammonia produced by larvae is absorbed through infested skin and thus reaches the heart, lungs and brain before detoxification by the liver. This hypothesis may explain why lesions occurred mostly in the dermal, cardiac, respiratory, nervous and hepatic tissues.

Results recently obtained[9] support the above-mentioned hypothesis: total ammonium concentrations in veins draining infested sites in four sheep ranged from 92 to 516 μmol ℓ^{-1} ($\bar{x}$ = 240; SD = ± 13) compared with systemic jugular and carotid levels (23 to 58 μmol ℓ^{-1} ($\bar{x}$ = 65, SD = ± 14). In some cases total ammonium concentrations in infested venous drainage were 8 times greater (516 μmol ℓ^{-1}) than systemic levels (58 μmol ℓ^{-1}).

In ruminants, ammonia toxicity is usually the result of ingestion of large quantities of urea[4] and thus amonia is detoxified by the liver. In the case of infested sheep the origin of ammonia toxicity is different to that of classic urea poisoning.

The pathological changes reported in this paper suggest that sheep infested by *L. cuprina* larvae suffer ammonia toxicity exacerbated by local and systemic alkalosis.

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Article/Artikel

PROPETAMPHOS: A COMPARISON OF THREE FORMULATIONS IN PREVENTING BLOWFLY STRIKE IN SHEEP

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ABSTRACT: Gruss B.; Janse van Rensburg B.J. **Propetamphos: A comparison of three formulations in preventing blowfly strike in sheep.** *Journal of the South African Veterinary Association* (1988) **59** No. 2, 77-79 (En). P.O. Box 27142, Greenacres, 6057 Port Elizabeth, Republic of South Africa

A trial was conducted in an area where organophosphate resistance to the green blowfly of sheep, *Lucilia curpina* WIED had been reported. Three formulations containing Propetamphos, namely an oil based pour-on, a grease based smear-on, and an emulsifiable concentrate were applied to 300 sheep in an attempt to compare the protection that this insecticide afforded, in preventing progressive blowfly strike. One hundred sheep were furthermore treated with an insecticide growth regulator, Cyromazine as a treated control group and a further 300 sheep were exposed as an untreated control group in order to monitor the level of blowfly strike in the area. It was found that the grease-based formulation protected the animals for 2 weeks against blowfly strike, both the pour-on and the emulsifiable concentrate formulation for 3 weeks, while the insect growth regulator afforded 4 weeks protection. It was concluded that if organophosphate resistant blowfly were present, the length of protection might be dosage related and that blowfly strike in sheep was almost equally controlled by Cyromazine and Propetamphos.

Key words: Blowfly, *Lucilia cuprina,* organophosphate resistance, Propetamphos, sheep

INTRODUCTION

Propetamphos, (E)-1-methylethyl 3-2-butenoate (SAN 3221) was discovered and patented by Sandoz Ltd, Basel, Switzerland and developed as a broad spectrum organophosphate insecticide/acaricide by Gothe[1,3,5]. Propetamphos is used as a sheep dip formulation in the U.K., Eire and France[5]. In South Africa, Propetamphos is used as a cattle, sheep and goat dip, and as a patch treatment on sheep for the prevention of blowfly strike (Bostan, Hoechst). The development of pour-on technology[5] has further extended the use of this acaricide, while in South Africa tick greases have been in use for many years.

Viljoen[6] investigated the potency and larvacidal effect of various insecticides against organophosphate resistant and susceptible larvae of the green blowfly *Lucilia cuprina.* He also investigated the efficacy of blowfly remedies in preventing blowfly strike on sheep (J.H. Viljoen 1978 P O Box 167, Swellendam, unpublished data). During the 8 years following completion of his work all stock remedies containing chlorinated hydrocarbons have been withdrawn from the South African market and their use on animals has been forbidden by Government proclamation. Much doubt has also been placed on the remaining organophosphate formulated blowfly remedies when comparing them with the insect growth regulator, Cyromazine (Vetrazine, Ciba Geigy)[7], which causes retardation of development and malformation of larval stages of sheep blowfly[7]. Resistance to Cyromazine has not yet been reported.

It was therefore decided to assess the efficacy of Propetamphos in preventing blowfly strike on sheep, as this compound had not been included in Viljoen's work. In carefully planned trials running concurrently with other registratory trials for new stock remedies, three formulations of Propetamphos were compared with Vetrazine, the standard blowfly reference remedy.

MATERIALS AND METHODS

The trials were conducted at the Oakdale Agricultural High School, Riversdale, 30 km from the southern seaboard in the southern Cape area of the Winter Rainfall Region. During the rainy season (March - December), light rains and misty weather are a constant feature. Organophosphate-resistant sheep blowflies were originally reported in the Riversdale[2,4] district and new blowfly remedies are often assessed in this region. The school's pastures were originally planted with lucerne, various species of grass and clover and also contained a multitude of indigenous grasses that grow in spring. The pastures were divided in 45 ha paddocks with a drinking trough in each paddock. Although the Riversdale area had been experiencing one of the driest winters for many years, very heavy rains fell during August followed by regular weekly showers. All the pastures on the farm were in a young lush stage of growth at the time of the trial.

Four hundred weaned female Merino lambs approximately 5 months old were randomly selected and divided into 4 replicate groups of 100 sheep each. The untreated control group consisted of 300 dams of these lambs. Each group was eartagged with a different colour eartag to facilitate future recognition. Each group was treated with a different formulation of Propetamphos. The live mass of the lambs was estimated by weighing a few average-sized individuals and assuming their mean mass to represent that of all individuals in the flock. Excessively large or small lambs were not included in the trial.

The different groups of lambs were then treated with the following formulations:

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The pour-on group was treated with the experimental Protetamphos pour-on solution which contained 10% Propetamphos active ingredient (a.i.), with ricinic oil as solvent. A sheep anthelmintic drenching gun and a back-pack reservoir were used.

The spout of the gun was placed on the poll of the head. While pressing down on the skin and applying pressure to the trigger mechanism, the gun was drawn in a caudal direction on the right hand side of the spinal column; 5 ml of the oily formulation was striped out on the backline, allowing the last ml to diffuse into the wool of the crutch. The process was repeated on the left hand side of the animal. In this manner each lamb received 10 ml solution containing 1 000 mg active ingredient at an estimated dosage of 50 mg kg^{-1} live mass.

The grease based smear-on group was treated with an experimental formulated grease-based blowfly remedy containing 0,14% Propetamphos (PAB NF, -Milborrow). Before the lambs were treated the canister was weighed and its mass noted. After the crutch area of each lamb had been smeared with the grease following the manufacturers' instructions, the canister was weighed once more. It was calculated that each lamb had received 47,5 g of the formulated grease. The minimum recommended dosage was 50 g grease containing 70 mg Propetamphos. In this trial the lambs received 66,5 mg active material which would constitute a dosage of 3,325 mg kg^{-1} live mass.

The dip concentrate group was treated with a 35% emulsifiable dip concentrate containing Propetamphos (Bostan, Hoechst). A homogenous emulsion was established by adding 200 ml of the emulsifiable concentrate to 50 ℓ of water under constant stirring. Using a hand-operated knapsack sprayer, 500 ml of the emulsion was sprayed onto the crutch of the lamb, thoroughly wetting this area. Run-off fluid was not re-used. The volume of emulsion applied was computed by measuring how long it took to fill a 500 ml measuring flask from the sprayer. In this manner each lamb was medicated with about 700 mg Propetamphos and at a dosage of 35 mg/kg live mass.

The control group treated with Cyromazine 50% WPC (Vetrazin, — Ciba Geigy). Following the manufacturers' instructions, 100 g of the powder was dissolved in 50ℓ water. Using the same knapsack sprayer and the same procedure as in the previous group, 500ml of this Vetrazin solution was jetted on to the crutch of each lamb, thoroughly wetting the area. Each lamb received 500 mg Cyromazine and at a dosage of 25 mg kg^{-1}.

The untreated control group: 300 unmarked and untreated adult ewes were added to the trial group in order to monitor the presence of blowfly activity and the rate of strike in the vicinity in which the trial was conducted.

All 700 sheep were run together in a 45 ha paddock. No other food or medication was administered to the flock for the duration of the trial. The sheep were examined twice a week. If an animal was found to be struck, it was marked with an orange sheep-marking dye and placed overnight in sheep pens which adjoined the paddocks and re-examined the following morning. If the strike was progressive, the animal was removed to another site on the farm where it was treated, otherwise it was released back into the flock. Fresh, untreated sheep were introduced in the place of sheep with progressive strike.

All observations were tabulated according to the registratory protocols issued by the Technical Advisor to the Registrar of the Stock Remedies Act, Act No. 36 of 1947, in which sheep blowfly strike is primarily classified into the anatomic site where the animal has been struck, i.e. body strike and crutch strike. Secondly, the nature of the lesion was described: aborted strike, where a fly laid her eggs on a sheep, but these eggs failed to hatch; query aborted strike was noted when a fly laid some eggs which hatched but didn't progress past the first instar stage when the larvae died leaving a lesion less than 3 cm in diameter; progressive strike was noted when the larvae progressed to further instars, miasis had occurred and the animal had to be treated.

The end point of a formulation was noted, in terms of a directive from the regulatory establishment, at the end of that week, preceeding the week, in which an ac-

Table 1: Accumulated percentages of lambs that were struck with progressive blowfly strike after treatment of various formulations of Propetamphos and with Cyromazine

Treatment of lambs	Formulations and dosages per kg live mass				Untreated control group
	Propetamphos			Cyromazine	
	Pour-on oil	Smear-on (grease)	Spray Emulsifiable concentrate	Spray (suspension)	
	50 mg kg^{-1}	3,33 mg kg^{-1}	35 mg kg^{-1}	12,5 mg kg^{-1}	
Week 1	0%	0%	0%	0%	8%
Week 2	0%	0%	0%	0%	10,67%
Week 3	4%	8%	4%	3%	18,34%
Week 4	6%	10%	6%	4%	19,01%
Week 5	6%	12%	7%	5%	21,01%

cumulative 5% of a group of animals treated with a certain formulation to protect them from blowfly strike, had been struck with progressive blowfly strike and had to be treated.

RESULTS

During the first 2 weeks of the trial all animal groups that received protective medication were free from any progressive blowfly strike, even in the face of intensive blowfly activity. The weather at this time was exceptionally conducive to fly activity with warm, humid days which in turn were interspersed with rainy days.

A sheep in the pour-on group received an aborted body strike during the first week, but during the second week all 100 lambs were protected. Week 3 saw 4% of the lambs receive progressive body blowfly strike and during Week 4 another 2% were struck, bringing the accumulative total to 6% and the end point after 3 weeks' protection. By the end of Week 5 no more lambs had been struck (Table 1).

In the smear-on grease group the first 8% of lambs were struck with progressive strike during Week 3 and the end point for this group was passed. Week 4 witnessed another 2% of the lambs being struck and during the fifth and final week of the trial, another 2% were struck bringing the accumulated total to 12%. Lambs protected by the emulsifiable concentrate were free from strike for the first 2 weeks, but during the course of the third week, 4% of the treated animals were struck. During Week 4 it was observed that the end point was passed when a further 2% were struck and during Week 5 a single lamb was struck, to bring the accumulative total to 7% (Table 1).

In the control group, 3% of the lambs had been struck with progressive blowfly strike, and had to be treated by the end of Week 3; another 1% were struck during the fourth week, bringing the accumulative total to 4%, while the end point was reached during Week 5 when another lamb was struck, where a total of 5% of the treated animals had been struck (Table 1).

The untreated control sheep bore the brunt of the onslaught, with 8% of the animals struck during the first week, 10,67% struck during Week 2, and at the end of Week 3, 18,34% of these unprotected sheep had to be treated for progressive blowfly strike. At the conclusion of Week 4, 19,01% had been struck and after the 5 week trial period, 21,01% of the animals had been struck (Table 1).

DISCUSSION

The efficacy of Propetamphos, when applied to sheep as a 0,14% grease based smear-on at a dosage of 3,325 mg kg^{-1} afforded a 2-weeks protection period, while the 10% oil based pour-on at a dosage of 50 mg kg^{-1} and the 35% emulsifiable concentrate at a dosage of 35 mg kg^{-1}, protected the animals for 3 weeks against blowfly strike. These results were almost comparable to the efficacy found when sheep were treated with Cyromazine, and one week longer protection was experienced. However, in fairness to the manufacturers of Cyromazine, their instructions were **not** fully carried out as neither a jetting comb nor a pump that delivered the fluid at a pressure between 400 and 700 kpa was used.

It may be speculated upon from results found in these trials, that the period of protection afforded by Propetamphos might be dosage related. The dosage of Propetamphos applied to the sheep which received the grease-based formulation (3,325 mg kg^{-1}) was considerably less than the dosage of the other 2 formulations (50 mg kg^{-1} for the pour-on and 35 mg kg^{-1} for the dip concentrate). In this instance it was found that the period of protection from blowfly strike for the former formulation was a week less than the latter 2 formulations. Similarly, if referring to the accumulated percentage of sheep struck by blowfly, as tabulated in the table, it will be seen that at the end of Week 5, and the sheep which were treated with PAB NF, had almost twice as many strikes (12%) as the sheep treated with the other two formulations (6% and 7% respectively).

Organophosphate-resistant green blow had been reported in the area where the trial took place[2,4]. It was not ascertained whether the larvae responsible for progressive strike on the test animals were in fact strains of organophosphate-resistant blowfly. Nevertheless it may be speculated that such larvae might be present from reports of poor efficacies obtained in this area when using the older organophosphate formulations, such as the Diazenon-based remedies (J H Viljoen 1978 P O Box 167, Swellendam, unpublished data).

It has been shown that blowfly strike on woolled sheep is a serious health hazard in the Winter Rainfall Region as in excess of one fifth of the untreated sheep were struck during this 5 week period.

Finally, it is imperative that alternative chemical groups of insecticides be investigated in order to protect the existing remedies now in use from acquiring resistant blowfly strains.

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INCO-ORDINATION AND PARESIS IN A CAPTIVE LION *(Panthera leo)*

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ABSTRACT: Burroughs R.E.J.; Roos C.J.; Ebedes H. **Inco-ordination and paresis in a captive lion *(Panthera leo)*.** *Journal of the South African Veterinary Association* (1988) **59** No. 2, 81-82 (En). National Zoological Gardens of South Africa, P.O. Box 754, 0001 Pretoria, Republic of South Africa.

An adult female lion *(Panthera leo)* with hind- and forequarter paresis and inco-ordination was examined. An initial diagnosis of a vitamin A deficiency was made, but treatment proved ineffective. A radiological examination showed mild pressure on the spinal cord. A tentative diagnosis of trauma to the cervical vertebrae was made. Response to conservative therapy suggested an aetiology of traumatic origin.

Key words: lion, paresis, inco-ordination, myelogram, suspected cervical vertebral instability

INTRODUCTION

Locomotor disturbances and nervous signs associated with the cervical area can be attributed to a number of causes. Extrusion of the intervertebral disc material, inflammatory lesions, vertebral fracture or subluxation, vertebral malformation and malarticulation, discospondylitis, neoplasia and other causes have been described[4]. Varying degrees of ataxia, inco-ordination, blindness, convulsions, skull malformation, cerebral and spinal changes have been associated with a vitamin A deficiency in captive lions[1 2 5]. Osteodystrophia fibrosa[9] and nutritional secondary hyperparathyroidism[3] have also been suggested as possible causes of nervous signs in captive lions. In this paper, locomotor problems and nervous signs in an adult lioness *(Panthera leo)* are presented.

CASE REPORT

A 5-year-old lioness, captive-born in the National Zoological Gardens, Pretoria, was housed with a male and female lion, both of older age. All 3 animals were fed 3 times a week on a diet of good quality meat, primarily beef, which contained bones. Over each ration of meat, approximately 5 g of a commercially available mineral (Carnivore Trace Element Mixture, Centaur) and vitamin premix (Carnivore Vitamin Mixture, Centaur), was sprinkled.

The first abnormality noticed was an unsteady gait. She developed a vacant expression on her face and often bumped into the night room doorframes. Apathy was present; she became less and less inclined to move and often had difficulty in climbing the stairs in her enclosure. The inco-ordination involved all 4 legs. Anorexia and a drop in body mass were present. A kyphosis of the thoraco-lumbar area was also observed. Opisthotonus and a sideways turning of the head were inconstant clinical signs.

Awareness of the problems of an adequately balanced diet under captive conditions led us to suspect a nutritional imbalance. Three ml (900 000 i.u) of a synthetic vitamin A preparation (Arovit injection, Roche) was subsequently given once intra-muscularly by means of a dart (Telinject, Randburg). Five ml of an oral vitamin A (Anchor-A, Anchor-pharm) preparation was sprinkled over the meat at each feed, in addition to the usual vitamin and mineral supplementation. The animal which was allowed to move freely about with the other 2 lions was observed for a further 2 weeks.

No improvement in clinical signs were observed and it was decided to do a physical examination. Immobilisation was achieved by intramuscular administration of 1,5 g of ketamine (Ketamine Dry Substance, Centaur) and 112,5 mg of xylazine (Rompun Dry Substance, Bayer). (Body mass was estimated to be 150 kg). Further incremental doses of ketamine (200 mg) and xylazine (25 mg) were delivered, as necessary, and the total dosage given over a period of 2½ h was 2,5 g ketamine and 100 mg xylazine. Apart from the obvious loss of body mass, the clinical examination of the animal yielded negative results.

Lateral survey radiographs were taken of the skull and the whole vertebral column as far as the sacrum. The following exposures provided acceptable radiographs at a focus to film distance of 115 cm using Cronex Hi-plus intensifying screens and a 10:1 ratio focused grid:

skull and upper cervical region: 66 kV and 160 mAs
lower cervical region: 70 kV and 160 mAs
thoracic and lumbar regions: 66 kV and 100 mAs

No lesions could be detected in the skull or thoracic and lumbar regions. The cervical radiographs taken with the neck in normal extension, showed a mild craniodorsal displacement of the vertebral bodies relative to those immediately preceding them. This tendency increased progressively in a caudal direction and was aggravated by flexion of the neck.

In order to investigate the possibility of cervical vertebral instability further and also to exclude other lesions not recognisable on survey radiographs, it was decided to do cisternal myelography. The first attempt failed as a result of an inability to reach the cisterna magna with the spinal needle.

The second attempt at myelography 5 days later, again under ketamine/xylazine immobilisation, was successful and an injection of 3,75 g of metrizamide dissolved in 8 ml diluent (Amipaque; Win-

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0038-2809/88/06/0081-0082

throp) was made into the cisterna over a period of 1,5 min. The examination was done following the guidelines prescribed for myelographic examination of the dog, namely, aseptic insertion of a 3,5 inch 22 gauge spinal needle exactly in the median plane of the neck, midway between the levels of the external occipital protuberance of the skull and the palpable wings of the atlas. After penetration of the skin, the needle had to be directed more caudally than in dogs to avoid the very prominent occipital condyles and adjoining occipital areas of the skull.

Lateral radiographs of the neck in normal extension and flexion were taken after brief elevation of the head and neck on completion of the injection. The cranial thoracic region was included on the radiographs.

The cranio-dorsal edges of the vertebral bodies caused pressure on, and attenuation of, the ventral contrast medium column on the myelograms. The attenuation of the column was more obvious during flexion of the neck and most prominent at the cranial end of the fifth cervical vertebra (Fig. 1). There was however only mild displacement of the spinal cord and mild attenuation of the dorsal contrast medium column in the cervical region.

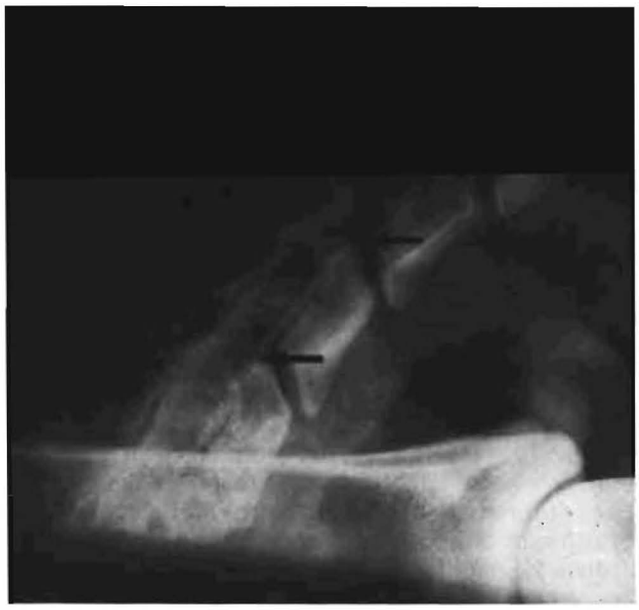

Fig. 1: Radiograph of the cervical spine of the lioness, showing attenuation of the ventral contrast medium column at C4 and C5 (arrowed)

A diagnosis of mild pressure on the spinal cord caused by unstable cervical vertebrae was made.

Following the myelogram, a long-acting antibiotic (Duplocillin, Coopers) was administered intramuscularly to prevent possible infection. Surgery was considered impractical, so conservative therapy was applied. The animal was given cage rest in the hospital area for 3 weeks. An anti-inflammatory steriod injection (Fluvet, Coopers) was given. No additional vitamin A was supplemented to her ration during this time besides the mineral and vitamin premixes.

During this period, her appetite returned to normal, and a gradual lessening of the observed clinical signs occurred. She started gaining weight, and within 3 weeks after the radiological examination, there was no evidence of opisthotonus or inco-ordination of either the hind- or forequarters. Her general habitus improved, and she was transferred from the hospital area to her enclosure, again immobilised with a ketamine/xylazine combination, where she was reintroduced to the other 2 lions in that enclosure.

DISCUSSION

Cervical vertebral instability has been associated with a combination of factors, namely malformation or malarticulation of the vertebrae, with compression on or some stretching of the spinal cord[8]. Numerous causes of the syndrome of cervical vertebral instability have been described[6,7]. Spondylolisthesis (excessive mobility of the cervical vertebrae) need not be present in every case[8].

The radiographs show a normal alignment of the vertebrae in normal extension of the neck, but mild displacement of the spinal cord with the neck in the flexed position. None of the other lesions associated with a cervical spondylopathy as outlined above, were however present. The mild nature of the pathology observed on myelography, however, made an absolute diagnosis of cervical vertebral instability difficult.

Cervical vertebral instability does not usually respond to conservative treatment — it is progressive in nature[6]. It is more probable that trauma to the neck could have caused a soft tissue injury, with resultant vertebral malalignment and mild spinal cord compression. In this instance, the cage rest given to this animal was sufficient for the clinical signs shown, to regress completely. The response to conservative therapy would support a diagnosis of traumatic origin.

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NUTRITIONAL SECONDARY HYPERPARATHYROIDISM IN A LION CUB

I.B.J. VAN RENSBURG* and M.H. LOWRY**

ABSTRACT: Van Rensburg I.B.J.; Lowry M.H. **Nutritional secondary hyperparathyroidism in a lion cub.** *Journal of the South African Veterinary Association* (1988) **59** No. 2, 83-86 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

A female lion cub about 5 months old from a litter raised artificially in a lion park, was euthanased and necropsied. The history was one of poor growth, lameness, a reluctance to move and skeletal malformations with disproportionately large head and feet. The cub had been fed meat and milk supplemented with calcium and vitamins. All the bones in the body were exceptionally soft and flexible. The long bones, vertebrae and ribs were easily split in half with a knife. The scapulae were deformed and medially curved. The long bones were shorter and thicker in diameter than normal, with very thin cortices. Numerous pathological fractures exhibiting minimal callus formation were present. The ribs were rounded, abnormally curved and very short in comparison to the length of costal cartilages. Radiographically, a marked osteoporosis was observed. Histopathological examination revealed a severe diffuse fibrous osteodystrophy with very little bone present. The osteoid was improperly mineralised and tissue sections for microscopy were cut without any need for prior decalcification. Little space for haemopoietic tissue remained. The lesions were considered consistent with those in animals raised on a diet containing excessive phosphates and low calcium.

Key words: Fibrous osteodystrophy, juvenile osteoporosis, osteodystrophia fibrosa, nutritional secondary hyperparathyroidism, lion

INTRODUCTION

Nutritional secondary hyperparathyroidism, also known as nutritional osteodystrophia fibrosa is caused by diets low in calcium and high in phosphorous, while a deficiency of vitamin D may also be incriminated[1,2]. In carnivores it is seen in young rapidly growing individuals where it is also referred to as juvenile osteoporosis, while in equines it occurs in adults where it is also referred to as bighead, bran disease or miller's disease.

In dogs and cats nutritional secondary hyperparathyroidism is caused by diets consisting mainly or entirely of meat and offal. Such diets have a very low calcium content and the calcium:phosphorous ratio is very wide. In cardiac and skeletal muscle for example, the ratio is 1:20, with a calcium content of about 100 mg kg^{-1}. The ideal ratio is 1,5-1,8 parts of calcium to every part of phosphorous[1].

Clinical signs in puppies and kittens include obscure lamenesses, lethargy, recumbency and obstipation. The bones are fragile and fractures and deformities are common, the latter occurring especially in the spinal column, ribs, sternum and scapulae[2].

CASE HISTORY

A litter of lion cubs was removed from the mother immediately after birth. For the first 4 days, they were fed cow's colostrum which had been maintained before use in a frozen state and thereafter a mixture of 3 parts Carnation milk (Carnation Food Distributors, Johannesburg) and one part water. When they were able to eat solid food, meat was added to the diet. From about the age of 10 weeks, they were gradually weaned off the milk diet and were introduced to carcases obtained from an abattoir. This diet was supplemented with two commercial preparations, one containing vitamins (A, D3, E) and minerals (calcium, iron) (Calsup, Centaur, Johannesburg) and the other containing haematoporphyrin, procaine hydrochloride and nicotinamide as well as vitamins (E, A, B_1, B_2, C) in a yeast base (Salupet, Noristan, Pretoria) at the dosage rates recommended for puppies. It was noticed that they only ate meat and could not chew the bones. They were housed in closed enclosures with very little exposure to natural sunlight.

The whole litter showed a stunted growth. The skeletal frames were small, while the heads and paws were disproportionately large. The animals showed varying degrees of lameness and were disinclined to move or play. The worst affected member of the litter was euthanased by barbiturate overdosage and submitted for post-mortem examination.

Radiological examination of the carcase showed severe osteoporosis, the bones being very radiolucent and the cortices of the long bones were very thin (Fig. 1 & 2). Evidence of several greenstick fractures with some callus formation was noticed.

Macroscopic findings

At necropsy, the most conspicuous finding was the extreme softness and flexibility of all the bones in the body. The spinal cord was removed simply by cutting through the vertebrae with a knife, without resorting to a chopping action of the knife or the use of an axe or saw (Fig. 4). The vertebral bodies were shorter than normal (Fig. 3), while the scapulae were grossly deformed having shorter, thicker, medially curved bodies (Fig. 6). The long bones were shorter and thicker than normal, some were abnormally bent, and the cortices were much thicker on the concave than on the convex side, parts being up to 8 mm in thickness with a laminated appearance. The bones could easily be bent or twisted (Fig. 5).

The ribs showed several abnormalities: the ratio in the

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0038-2809/88/06/0083-0086

length of each rib to that of its costal cartilage was greatly reduced; the ribs were round and not flattened on cross section, and the normal curve (sprung of the ribs) was absent, which lead to a straighter rib and a reduced capacity of the thoracic cavity. Along the length of many of the ribs were numerous irregular thickenings which probably represented sites of healing of pathological fractures with fibrous callus formation (Fig. 3 & 4). The costochondral junctions were moderately enlarged, but the epiphyseal lines were not widened, irregular or wavy in appearance.

The thyroids were of normal size and had a pale yellowish-brown colour. The parathyroids were reddish-brown, hyperplastic and each measured approximately 6x4x3 mm.

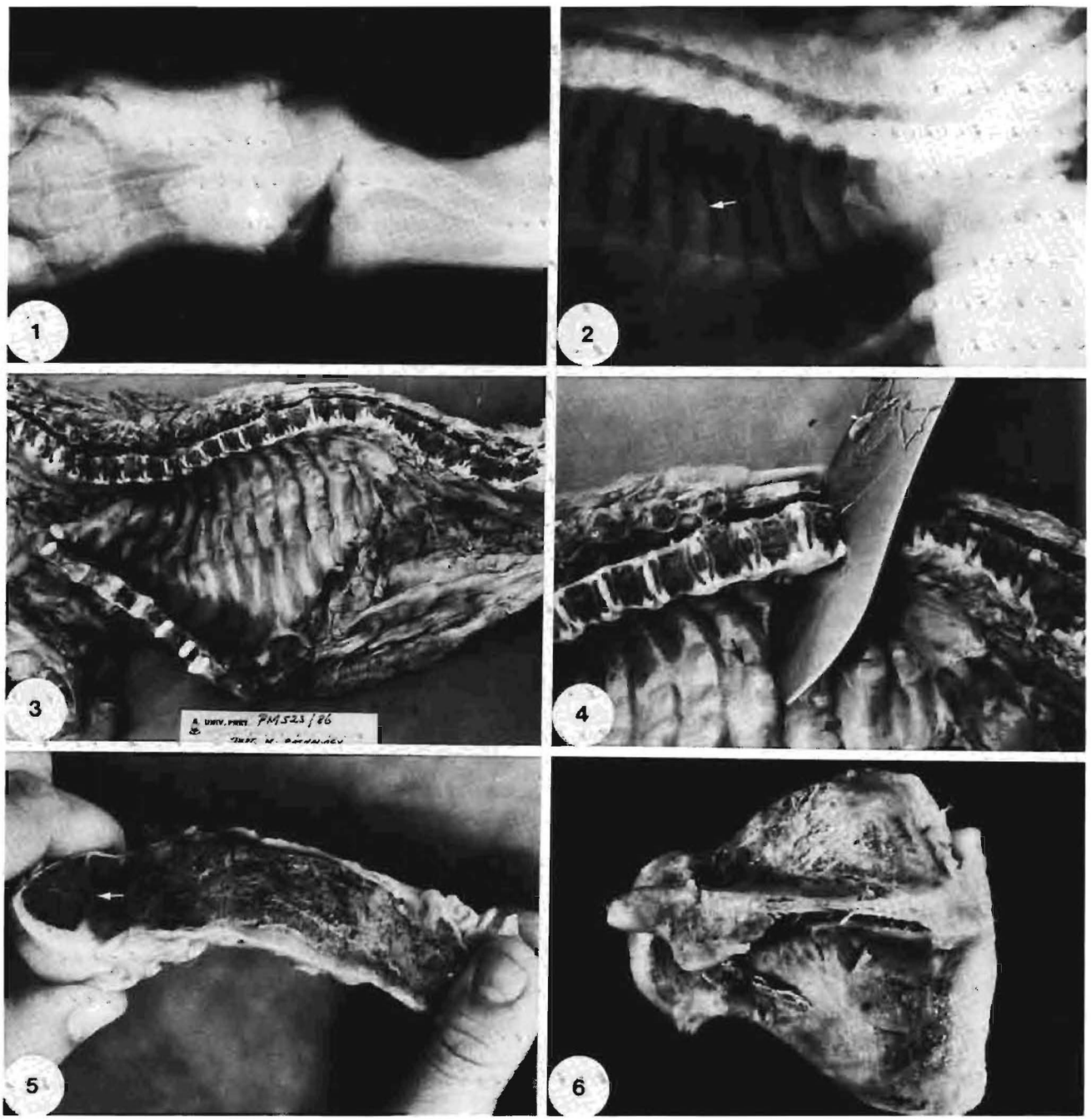
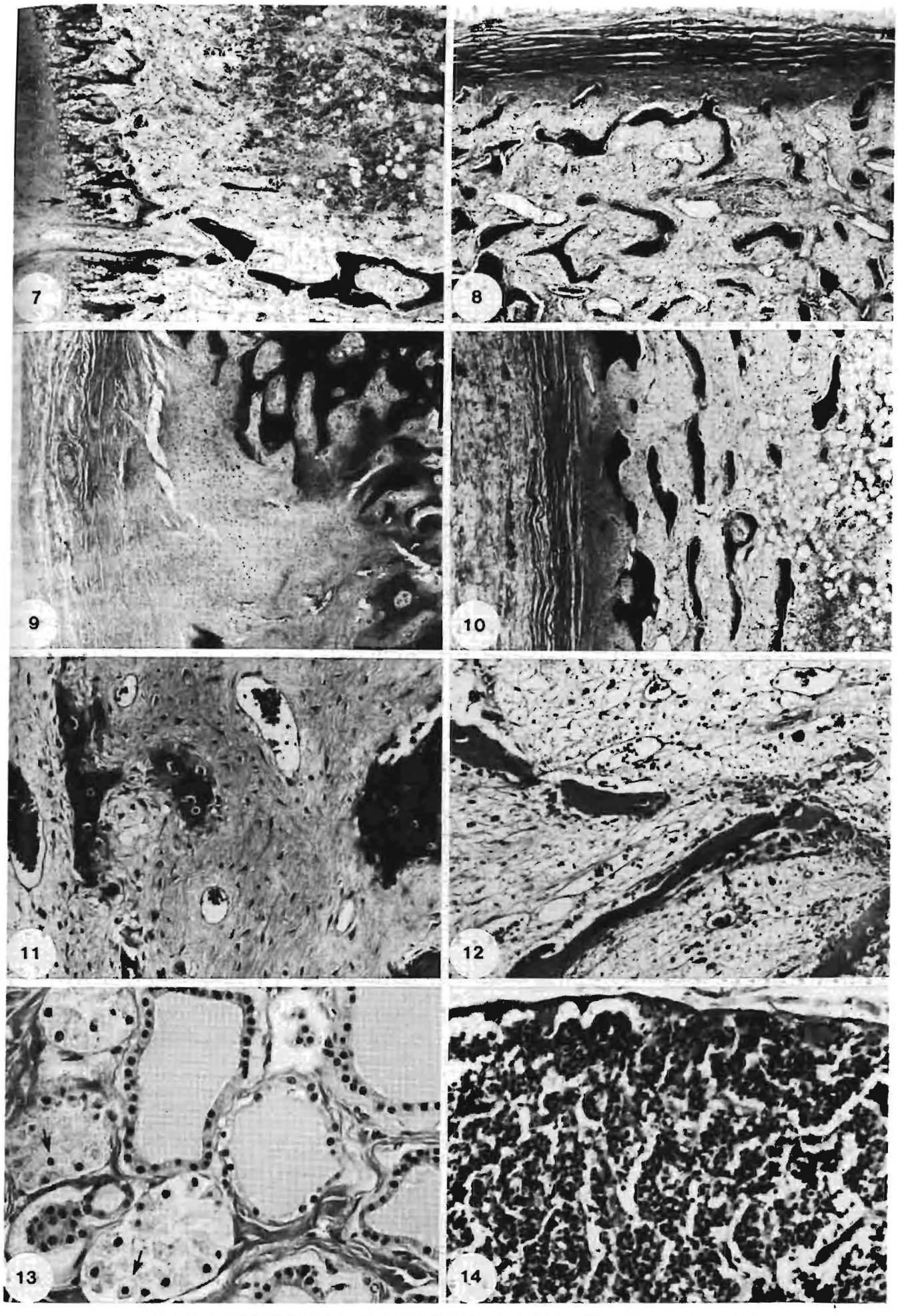


Fig. 1 & 2: Radiographs showing severe osteoporosis. Note radiolucent appearance of the skeletal system and calusses in ribs (arrow)
Fig. 3: Note the short vertebral bodies and abnormal ratio in length of rib to that of the costal cartilage
Fig. 4: The affected bone could easily be cut with a knife. The ribs show irregular thickenings in areas of healing fractures (arrow)
Fig. 5: Note the thin cortex, the abnormal curvature and the thin regular epiphyseal line (arrow)
Fig.6: Abnormally formed scapula
Fig. 7: Ossification centre in a phalanx with normal bone formation but marked lack of calcification. Note cartilage in spicules of bone (arrow) and the hypoplastic formation of diaphyseal compact bone at the bottom of the picture. X40
Fig. 8 & 10: Thickened periosteum and fibrous tissue in spicules of poorly mineralised osteoid along the diaphysis. X40
Fig. 9: Fracture with collagenous callus formation. X40
Fig. 11 & 12: Spicules of bone embedded in fibrous tissue in periosteal area and reticular connective tissue in medullary area. Note scant haemopoietic activity and few osteoblasts around some bony spicules (arrow). X200
Fig. 13: Thyroid showing C-cell hyperplasia (arrow). X400
Fig. 14: Hyperplasia of the parathyroid. X400
(All magnifications given are the originals used during microphotography.)



Microscopic findings

All the bones were soft enough to enable blocks to be trimmed and sections for microscopical examination to be cut from them without prior decalcification.

At the endochondral ossification centres, there was no evidence of chondral calcification, but ossification appeared fairly normal, except for the lack of proper mineralisation. Cores of cartilage persisted in most of the bony spicules for short distances into the metaphyses (Fig. 7). Ossified parts of the bones showed severe fibrous osteodystrophy. Cortical compact bone was not composed of a continuous layer of bone, but consisted of a thickened periosteum covering fibrous connective tissue in which poorly mineralised spicules of bone were embedded (Fig. 8 & 10). From this, spicules of bone radiated into the metaphyseal area where the interosseous spaces were occupied by mesenchymal and reticular connective tissue criss-crossed by delicate blood vessels with widened lumens (Fig. 11 and 12). Haemopoietic tissue was present in some areas, but in diminished quantities, (Fig. 11 and 12). Bone spicules were either not, or only poorly, mineralised and were covered in many instances by a layer of osteoblasts. Osteoclasts were present, but were not numerous and in some areas they were embedded in the fibrous and reticular connective tissue. Calluses at sites of previous fractures were recognised as a local thickening of compact cortical bone, as a result of proliferation of fibrous connective tissue and cartilage (Fig. 9). Focal accumulations of macrophages laden with haemosiderin were noticed at these sites, but no large haemorrhages or cystic areas due to previous extensive haemorrhages were observed.

The thyroids showed prominence of interstitial connective tissue and a marked C-cell hyperplasia (Fig. 13). No other significant deviations from normal were encountered. The parathyroids were hyperplastic and moderately congested and consisted of cords and clusters of chief cells with a fine foamy cytoplasm (Fig. 14). No follicles were formed and no distinct oxyphilic cells were evident.

No signs of metastatic calcification were noticed in the lungs or kidneys.

DISCUSSION

A common mistake often made in the artificial rearing of wild carnivores is to feed them on a solely meat diet, predisposing them to the development of nutritional secondary hyperparathyroidism. In the wild, these animals also consume organs such as the liver as well as entrails and bones which supply essential vitamins and minerals. It was not easy to determine what mistake was made in rearing the lion cub described in this paper, as the diet had been supplemented with calcium and vitamin D_3. The diet, as well as the limited exposure to sunlight would, however, seem to have played a key role in the pathogenesis of the lesions. This is substantiated by the fact that subsequent litters raised under similar conditions but with more exposure to sunlight and increased levels of calcium and vitamin D supplementation did not develop the disease.

The lesions in the cub corresponded very well with those described in experimental cases of nutritional secondary hyperparathyroidism in cats[3] and those seen in a tiger[4] and a colony of lemurs[6]. Similar lesions were reported in primates[5 8], birds[7] and reptiles[9] suffering from the same condition. The lion cub showed no signs of rickets, but classical lesions of severe juvenile osteoporosis were present. Replacement of cortical bone by spicules of poorly mineralised osteoid embedded in fibrous connective tissue, explains fully the clinical signs of lameness and reluctance to move, the numerous fractures, softness and flexibility of the bones in the cub. Considering the limited space in the bones to accommodate haemopoietic tissue one would have expected a myelopthisic anaemia to be present, yet the carcase did not appear to be anaemic. Unfortunately no haemotological or chemical pathological examinations were performed. The histopathological observation of C-cell hyperplasia in the thyroid and the simultaneous hyperplasia of the parathyroid seems paradoxical as the former, allegedly results from a hypercalcaemia while the latter results from a hypocalcaemia (or hyperphosphataemia). In cases of nutritional secondary hyperparathyroidism the blood calcium may be low, normal or elevated. This possibly explains the absence of metastatic calcification of soft tissues in nutritional secondary hyperparathyroidism while it is such a common phenomenon in renal secondary hyperparathyroidism in uraemic dogs. Furthermore the fibrous osteodystrophy associated with renal secondary hyperparathyroidism and bran disease in equines is characterised by the presence of numerous osteoclasts in affected bone, a feature which was absent in this case. This is probably because the lesions in the lion cub occurred in bone which was still actively growing whereas in the aforementioned conditions the lesions developed in bone which had already matured.

Renal cortical atrophy and diffuse renal fibrosis has been recorded in large carnivores fed on a diet consisting mainly of meat, but it was not present in this lion cub.

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DIE VEEARTS EN DIEREREGTE

Die waarde van geselskapsdiere in moderne gemeenskappe is veral gedurende die afgelope dekade herwaardeer. 'n Gedagte wat feitlik gelyktydig hiermee opnuut aandag gekry het, is die erkenning en bevordering van diereregte. Filosofieë rondom die kwessie van diereregte is geformuleer en belangegroepe het tot stand gekom. In sekere kringe is hierdie denkrigtings nie net gestel as 'n ideaal of alternatiewe benadering nie, maar goed georganiseerde pogings (byvoorbeeld lektuur en advertensies) is van stapel laat loop om die idees aan die gemeenskap oor te dra. Alhoewel die veldtogte meesal op die algemene publiek gerig is, verskyn daar dan ook 'n aantal artikels in wetenskaplike tydskrifte wat diereregte ondersteun, asook boeke oor dieselfde onderwerp.

Die aktiviste van hierdie beweging hou hulle veral besig met protesoptogte en betogings; dit het al selfs gelei tot beskadiging van eiendom om bewyse te bekom van wreedhede en om diere te bevry. Volgens die 1985 verslag van The Humane Society of the United States[36], het dié vereniging meer as 'n miljoen dollar wins in daardie jaar getoon. Hulle *Animal Activist Alert* nuusbrief word gereeld aan 7 000 aktiviste gestuur. In 1981 is daar 'n Animal Rights Coalition[3] gestig, wat hulleself as aktiviste en "grass roots"-groepe beskryf, om diereregte te bevorder.

Professor Heuse, stigter en eerste President van die International League for Animal Rights, sê in sy *Declaration of the Rights of the Animals* in 1978, dat verteenwoordigers van diereregtegroepe 'n effektiewe inspraak op alle vlakke van regering moet hê en dat diereregte net soos menseregte beskerming deur die wet moet geniet. Osterhoff[22] sê dat as hierdie groepe in hulle doel slaag, diereregte in die jaar 2 000 wyd erken sal word met 'n universele handves, wat wêreldwyd aanvaar sal word.

Dat die veterinêre professie by die saak van die diereregte betrek sou word, blyk 'n onvermydelike wending te wees. Hoe langer hierdie tendense deur veeartse geïgnoreer word, hoe moeiliker kan dit word om standpunte teen mekaar op te weeg. Om perspektief op die onderwerp te kry, word die eerste deel gewy aan 'n historiese oorsig oor beskouings van mense oor diereregte en die tweede deel handel oor uitsprake oor diereregte wat die veterinêre professie betrek.

Die derde deel bevat 'n opname onder veterinêre studente oor diereregte en laastens word standpunte oor diereregte ingeneem.

OORSIG VAN DIE MENS SE BESKOUINGS OOR DIEREREGTE

Die boek *Animals rights and human obligations*[23] bevat 'n aantal essays oor uitsprake wat op diereregte betrekking het. Uittreksels van sommige menings word chronologies weergegee.

Aristoteles,[23] een van die vroeë denkers oor die mens-tot-dier-verhouding, het in die vierde eeu v C gemeen dat "laer diere" verskeie eienskappe met die mens in gemeen het. Sulke eienskappe sluit in: die vermoë om te voed, die vermoë om voort te plant, die vermoë om bewus te wees van die wêreld deur sensoriese organe en die vermoë om te begeer, te voel, te onthou en te dink. Aristoteles het daarop gewys dat die eerste twee eienskappe ook deur plante gedeel word. Omdat die mens die vermoë besit om te redeneer, kan die mens dus as 'n "rasionele dier" ("hoër dier") beskou word.

Plutarchos[23], ca 46 - 120 n C, meen dat dit nie nodig is om vleis te eet om gesond te lewe nie, maar dat dit nie verkeerd is om 'n dier dood te maak as dit nodig is om 'n mens se lewe te red nie. Verder meen hy dat mense wat wel diere teel en slag om te eet, dit nie weens gesondheidsoorwegings doen nie, maar bloot om hulle smaak te bevredig. Omdat hierdie spesifieke genietinge onnodig is, is die pyn wat aan slagdiere veroorsaak word, òòk onnodig en omdat die pyn onnodig is, is dit verkeerd om diere vir slag-doeleindes aan te hou en te teel.

Descartes[23] 1596 - 1650, het diere as "outomata" (masjiene) beskou. Diere sou dan geen bewussyn hê nie en gedagteloos en sielloos wees. Daarom het die mens geen verpligting teenoor diere nie.

Kant[23] 1724 – 1804, glo dat die mens 'n doel in homself het en dus 'n intrinsieke waarde het. Hy sien die verskil tussen mens en dier as die feit dat mense outonome wesens met 'n eie vrye wil is. Kant is teen wreedheid teenoor diere, maar hy is eerder bekommerd oor watter effek sulke wreedheid op die mens het. Mense wat wreed teenoor diere is, word ook hard teenoor mense. Die mense se pligte teenoor diere, is feitlik indirekte pligte teenoor die mensdom.

Bentham 1748 – 1832 en Mill[23] 1806 – 1873, glo dat die dag sal kom wanneer die res van die diereryk ook daardie regte sal verkry wat nooit deur die mens se hand van tirannie van hulle teruggehou moes gewees het nie. Soos die Franse ontdek het dat die kleur van die vel geen rede is waarom daar teen 'n mens gediskrimineer hoef te word nie, so sal daar besef word dat die aantal bene, die harigheid van die vel of die rigting van die *os sacrum*, ook nie genoegsame redes is om sensitiewe wesens (diere) aan dieselfde lot te onderwerp nie. Hulle vra of daar dan ander bewys is wat as onoorkomelike verskille aangetoon kan word. Sou dit dalk die vermoë tot rede of gesprek wees? 'n Volgroeide perd en hond is egter ver meer rasioneel as selfs 'n maandoue baba. Die vraag is nie of diere kan redeneer of praat nie, maar of hulle kan ly.

Schopenhauer[23] 1788 – 1860, meen dat die mens geen doel in homself het nie, en dat dit as 'n teologiese idee totaal verwerp moet word as sinloos. 'n Mens moet blind of narkoties wees om nie te besef dat die essensiële of belangrikste dinge in die mens en dier dieselfde is nie en dat die verskille slegs op die sekondêre vlak van intellek en graad van die denkvermoë lê. Hierdie graad is hoër in die mens deur die bykomende vermoë tot abstrakte kennis, genoem die rede. Hierdie meerderwaardigheid is slegs waar te neem in 'n groter serebrale ontwikkeling. Dit is dus 'n enkele liggaamlike verskil van 'n spesifieke deel van die brein, en dít slegs in kwantiteit. Aan die ander kant is die ooreenkomste tussen

0038-2809/88/06/0087-0097

mens en dier onvergelykbaar groter, psigies sowel as somaties.

Darwin[23] 1809 – 1882, glo dat as die vermoë tot rede (rasionele denke) ook op die nie-verbale gedrag beoordeel word, diere wel rasionele wesens is. Hy sê hy wil aantoon dat daar geen fundamentele verskil in verstandelike vermoëns tussen die mens en ander hoër diere bestaan nie.

Rickaby[23] 1945 – 1982, kom weer tot die gevolgtrekking dat diere geen begrip het nie en daarom geen persone is nie. Hulle kan dus nie regte hê nie en diere is ook nie selfgesentreerd nie.

Salt[23] 1851 - 1889, voel dat dit beter vir diere is om glad nie te lewe nie as om versorg te word om geslag te word. Hy meen al die vrae oor diereregte verminder net die belangrikheid van diereregte. Dit word gekenmerk deur die kontroversie oor regte en word nie veel meer as 'n akademiese stryd oor woorde nie. Vegetarisme is die dieet van die toekoms en vleis dié van die verlede. Salt meen dat "laer diere" ongetwyfeld regte het as mense regte het.

Ritchie[23] 1853 – 1903, glo dat om 'n "reg" te hê, is om 'n aanspraak op iets, of teen ander te kan hê en dit is alleen vir lede van die menslike gemeenskap beskore. Laer diere kan dus geen regte hê nie. Hy sê die feit dat diere deur wette beskerm word, beteken nie dat hulle regte het nie, want daar is ook wette wat byvoorbeeld ou geboue beskerm. Geen wette wat oor diere bestaan gee hulle regte in die wettige sin van die woord nie. Daar mag wel "natuurlike regte" wees wat skadeloos gebruik kan word in akademiese besprekings. Wreedheid teenoor diere kan tereg gesien word as 'n oortreding teen die menslike gevoel. Die mens se plig teenoor diere is 'n plig teenoor die menslike gemeenskap. Pligte van welwillendheid teenoor diere beteken nie dat diere "regte" teen die mens ontvang nie; die belange van die menslike gemeenskap moet die mens se uiteindelike kriterium van reg of verkeerd wees.

Feinberg[23] erken dat daar in die kwessie rondom diereregte hoogs verwarrende grensgevalle voorkom. Hy sê ons kan wel pligte hê **aangaande** diere, maar nie noodwendig **teenoor** diere nie. Ten spyte hiervan glo die meeste mense dat diere wel regte het, maar hulle huiwer om dit te erken weens die verwarring oor wat presies met diereregte bedoel word.

Rachels[23] sê dat menseregte 'n formidabele wapen teen slawerny, rassisme, seksisme en ander diskriminasie was. Regte moet egter oorgaan na die "nie-mense" omdat hulle óók oor die reg tot vryheid beskik. Hy is selfs bereid om so ver te gaan om te sê dat sekere "nie-mense" wel oor 'n moraal beskik.

White[23] glo dat om laer diere in die mens se etiese sisteem in te sluit, filosofies sinloos en prakties onmoontlik is.

Regan[23] noem vier kriteria wat gewoonlik gebruik word om mense van diere te onderskei. Dit is die vermoë om te redeneer, die vermoë om vrye keuses te maak, die konsep van die "self" en kombinasies van bogenoemde. Dit beteken egter nie dat "nie-mense" ook nie een of al hierdie eienskappe kan hê nie. Dit beteken beslis egter ook nie dat alle mense aan hierdie eienskappe voldoen nie. Regan beweer dat gevoel, die ondervinding van plesier en pyn 'n gemeenskaplike eienskap van mens en dier is en dus regte aan diere verseker. Hy is bekommerd dat mense diere eet, op hulle eksperimenteer en party diere se gewoontes vernietig, terwyl die mens selde daaroor dink of sulke optredes eties verdedigbaar is.

Singer[23] sien die stryd vir die gelykheid vir alle diere as dieselfde as die stryd vir ander onderdrukte groepe soos die swart bevrydingsbeweging, die vryheidsbeweging van die Spaanssprekende Amerikaners, homoseksueles en 'n verskeidenheid ander minderheidsgroepe. Hy beweer dat, toe diskriminasie op grond van geslag verdwyn het, gesê is dat dit die laaste vorm van diskriminasie was, maar dat die sak vir diereregte bewys het dat dit nie so is nie. Singer glo dat soos daar rassisme en seksisme bestaan, daar ook **spesisisme** bestaan. Hy erken wel oppervlakkige verskille tussen rasse en geslagte, maar die beginsel van gelykheid lê nie in die beskrywing van die mense nie, maar hoe hulle behandel word. Dieselfde geld vir die dierespesies. Die spesisis laat toe dat die belange van sy spesie die belange van 'n ander spesie oorheers. Die meeste mense is dus spesisiste. Hy kan ook nie sien hoe babas, verstandelik gestremdes, psigopate, Hitler, Stalin en diesulkes, hoër waarde kan hê as wat 'n olifant, vark of sjimpansee ooit kan bereik nie. Alle mense kan dus nie 'n intrinsieke waarde hê nie. In verdere argumente sê Singer dat om die komplikasie van "potensiaal" te vermy, sal hy konsentreer op "permanente vertraagde persone" en so besluit hy dat álle diere, of dan mense en "nie-mense", gelyk is.

Tot sover die uittreksels uit die boek. Enkele ander kontemporêre uitsprake lui as volg:

Solomon[30] meen dat die ooreenkomste in dierlike en menslike breine suggereer dat diere en mense psigologies eenders is, maar dat 'n mens die ooreenkomste in elke spesie afsonderlik moet beoordeel. Die gevolge van lyding varieer van een spesie tot 'n ander. Die alles-of-niks-vrae oor mens en dier moet vervang word met vrae oor die **vlakke** van diere se bewussyn. Vrae soos of diere intelligent is, 'n taal het, of emosies het moet eerder verander na watter sóórt intelligensie, taal en emosies. Hierdie vrae sal waarskynlik altyd onbeantwoord bly.

Clark[5] lewer 'n pleidooi om ten minste die hoër diere te sien as "we mammals", of "we animals", in plaas van "we men". Hy sê die mens moet sy solidariteit en gemeenskaplike voorvaders met ander diere, wat nie van die mens se onmiddellike soort is nie, in gedagte hou. Hy voel ook baie sterk oor vegetarisme en stel dit as 'n voorvereiste vir alle persone wat hulle vir diereregte beywer. Mense wat nog vleis eet het geen outoriteit om oor diereregte te praat nie en hulle kan nie ernstige moraliste wees nie.

Allbright[1] sê dat in Amerika, die sestigerjare studente- en swart onrus teweeggebring het en die sewentigerjare die kwessie van vroueregte, maar dat die tagtigerjare sal behoort aan die bewegings vir die regte en welsyn van diere, insluitende produksiediere. Die diereregte kwessie sal nie verdwyn nie.

Miller[19] is weer ten volle oortuig dat die mens as jagter, biologies, antropologies en histories geregtig is om diere tot sy voordeel te gebruik, as voedsel sowel as vir geselskapsdiere. Dit beteken egter glad nie dat hy wreedheid teenoor diere goedkeur nie.

Rosenwald[27] sê dat die mens verantwoordelikhede het vir die welsyn en menslike versorging van álle diere en dat dít 'n absolute aanvaarbare feit is. Dat diere "regte" het soos die mens, is per definisie nie noodwendig aanvaarbaar nie, want daar is groot belangrike grys areas in die sieninge oor wat diereregte presies is.

In 1985 het 'n nuwe tydskrif verskyn onder die titel: *Between the species*, wat 'n spreekbuis geword het vir die heel nuutste denke oor diereregte. Die bydraes is

veral op 'n filosofiese grondslag gebaseer. Enkele aanhalings uit die eerste jaar se uitgawes word gemaak:

Giraud[11] sê in die eerste uitgawe dat die gedagte van diereregte dalk in die tyd van die Renaissance begin posvat het, maar dat Henry Salt se werk in 1892, *Animal rights*, 'n buitengewone bydrae in dié verband was. In die redaksionele kommentaar[7] van dieselfde uitgawe word gesê dat gepoog sal word om die tydskrif 'n medium vir interspesiedialoog te maak. Die gesprek moet geskied op die tema van absolute rekonsiliasie tussen mens en natuur. (Ongelukkig het daar tot dusver net bydraes van een spesie tot die dialoog verskyn)! In die redaksionele kommentaar[8] in die daaropvolgende uitgawe word gesê dat diere wat in navorsing gebruik word, billik vergoed moet word. (Die redakteur het nie die aard van die vergoeding omskryf nie). Magel[15] sien die posisie van diere teenoor die mens as parallel met dié van slawe en bepleit 'n dierebevrydingsbeweging. Hummer[12] beweer dat gedragkenners, taalkenners en etoloë gefaal het om enige kwaliteit in die mens te definieer wat nie in 'n sekere graad óók in diere aanwesig is nie, veral dan so in die hoër diere. As dit 'n feit is, dan het die mens 'n plig om 'n manier te soek om op die een of ander wyse met sy bure op die planeet te kommunikeer. Stockwell[31] beweer dat die proses om gode, stamme (rasse) en spesies uit te wis, dieselfde is. Hy het ook beswaar oor Singer[29] se stelling dat hy die lyn vir gelyke oorwegings vir spesies net bó die molluske trek. Gelykheid moet vir álle diere geld. Fox[10] skryf dat mense wat glo dat die mens die meerdere van die dier is, nie in staat is tot etiese objektiewe en verantwoordelike optrede teenoor diere nie. So 'n hiërargie onder spesies skep 'n valse indruk van die realiteite. Opvoeders, wetenskaplikes, filosowe en andere, word gewaarsku teen die subtiele vertakkings van idees waarin die houding teenoor diere, nie sou neerkom op demokrasie of gelykheid nie. Sunlin[33] is oortuig dat antroposentrisme 'n menslike drogrede is, alhoewel feitlik al die mense die siening gehad het en nog het. Antroposentrisme is gegrond op 'n besliste onderskeiding tussen mens en "nie-mense", wat nie bestaan nie. Ost[21] sê dat Regan se werk, *The case for animal rights* 'n briljante en goed beredeneerde werk vir diereregte is en hy beskou Regan as die persoon wat die ernstigste argumente vir diereregte na vore gebring het. Ost voel egter dat die mens se tirannie oor die natuur nie vervang moet word deur 'n passiewe viktimisasie van die mens deur die natuur nie. Hy meen dat die mens die verloorder sal wees in die onvermydelike konfrontasie tussen spesies.

Tot sover aanhalings uit die tydskrif *Between the Species*. Om hierdie deel se menings oor diereregte af te sluit, word daar na Morgan[20] se gidsplan gekyk, wat houdings teenoor diere kategoriseer. Let op dat veterinêre organisasie onder Dierebeheer geplaas word.

(i) Diere-uitbuiting ("animal exploitation")
Diere word gebruik om mense te plesier deur teen mekaar te veg of hulle word in wrede sportsoorte gebruik soos bulgevegte en hondegevegte. Reëls en wette word verbreek.

(ii) Dieregebruik ("animal use")
Diere is daar vir menslike gebruik maar pyn en lyding moet voorkom word, waar moontlik. Jag en rodeos en die aanhou van eksotiese diere word aanvaar.

(iii) Dierebeheer ("animal control")
Wette wat diere reguleer moet nagekom word, maar dit is mense se eie probleem as hulle onverantwoordelik teenoor diere optree. Hierby sluit die skryfster proefdiereenhede, plaaslike owerhede en veterinêre organisasies in. Genadedood word aan oorlasdiere toegedien.

(iv) Dierewelsyn ("animal welfare")
Mense moet diere so goed as moontlik behandel en dit word van mense verwag om dit te doen. As mense nie wil of kan nie, dan het die welsynsgroepe 'n plig om na lydende en daklose diere om te sien. Hierdie mense is gekant teen eksperimente op diere en sal oorlasdiere eerder steriliseer as om 'n genadedood toe te dien.

(v) Diereregte ("animal rights")
Diere het intrinsieke regte en dit moet gewaarborg word net soos dié van die mens. Diere mag nie doodgemaak, geëet, vir sport of navorsing gebruik of op enige ander manier misbruik word nie. Mense wat hiervoor pleit sluit diereregtegroepe, antiviviseksiegroepe en mense wat spesiale diereversorgingsoorde en ouetehuise vir diere oprig, in. Sommige sal selfs sê dat geselskapsdiere die reg het om te teel soos hulle wil.

(vi) Dierebevryding ("animal liberation")
Diere mag op geen wyse gedwing word om tot voordeel van die mens, te werk of te produseer nie. Alle gebruik en misbruik van diere moet beëindig word. Die groepe wat dít bepleit, staan 'n algehele restrukturering van die gemeenskap se ekonomiese en besittingsreg voor. Van hierdie mense noem hulleself aktiviste en sal onwettige metodes goedkeur of aanwend om diere te bevry. Militante groepe is selfs bereid om geweld te pleeg ter bevordering van hulle strewes. Hierdie mense sal nie geselskapsdiere aanhou nie, omdat dit 'n vorm van slawerny is.

MENINGS OOR DIEREREGTE WAT DIE VEEARTS BETREK

Alhoewel die veterinêre professie van tyd tot tyd direk by die debat oor diereregte betrek word, neem veeartse, of spreekbuise vir die veearts, soos verenigings en fakulteite, selde amptelik aan die debat deel. Dít, ten spyte van die feit dat veeartse al daarvan beskuldig is dat hulle die enigste professie is wat hulle pasiënte eet. Veeartse word aangemoedig om bykomende versekering uit te neem teen klagtes van diereregtegroepe. Min veeartse het hulle egter tot nou toe openlik en met oortuiging by die diereregtegroepe geskaar. Die veeartse wat dit wel gedoen het, is meesal nie-praktiserende veeartse. Vandag se geestelike leiers in diereregtebewegings soos Regan, Rolin, Singer en Fox is filosowe. Fox is wel as veearts opgelei maar het hom sedertdien op die gebied van die filosofie onderskei. Hy is tans Direkteur van Wetenskaplike Studies van die Humane Society of the United States of America.

Rolin[26] wat in 'n outobiografie verduidelik hoe hy in diereregte begin belang stel het, sê dat hy verveeld en rusteloos geraak het met sake oor mediese etiek. Hy is toe gevra om 'n lesing oor etiek in 'n veterinêre

fakulteit te gee. Nadat hy die saak oordink het, het hy besluit dat die fundamentele vraag vir die veterinêre etiek die morele status van die dier is. Sy byna toevallige kontak met die veterinêre professie het hom laat besluit dat hier 'n geleentheid was om etiese idees in die konteks van 'n groep wie se doel is om na die gesondheid en welsyn van diere om te sien, te ontwikkel. Hierdie groep het egter tot op daardie stadium geen duidelike bewustheid of uitsprake oor die morele vraagstukke onderliggend aan hulle professie gehad nie.

Fox[9] glo dat die veearts se eed verander moet word sodat die veearts se diens **nie** primêr tot voordeel van die gemeenskap aangewend moet word nie, maar áltyd direk tot voordeel van die dier moet strek.

Tisschler[38], die Uitvoerende Direktrise van die Attorneys for Animal Rights, meen dat veeartse die slapende skoonhede in die diereregtebewegings is. Sy sê dat dit 'n ongelukkige feit is dat veeartse en diereregte- of dierewelsynsmense blykbaar in gedurige konfrontasie verkeer. Nogtans sal die diere-aktiviste wat negatief oor die veterinêre professie voel, hulle eie veearts respekteer en vertrou. 'n Mens moet ook nie verwag dat alle veeartse met sulke bewegings moet assosieer nie. Alle mediese dokters assosieer nie met bewegings wat kernoorloë wil voorkom of die stryd teen honger in die wêreld aankoop nie, terwyl hulle steeds goeie dokters is. Sy glo egter dat een rede vir veeartse se onbetrokkenheid is dat hulle voel dat diereregtegroepe te emosioneel is. Sy ontken ook nie dat dit wel waar is van sekere diereregtegroepe nie.

Tisschler stel die basiese diereregteteorie soos volg: Diere lewe, kan pyn en plesier (gevoel) ervaar, belange hê en is voorwerpe van morele betrokkenheid. Dit verplig die mens om diereregte in ag te neem. Probleme kom egter in die praktyk na vore, wat Tisschler raaksien, maar waarvoor sy geen oplossing bied nie. Sy sê dit is vir baie veeartse moeilik om te begryp en 'n verleentheid, dat diere regte het wat apart is van die eienaar en dat hy moontlik basiese regte van diere kan oortree. Om dus net die kwessie van diereregte te begin oorweeg, is vir veeartse soos om Pandora se kissie oop te maak. Sy gee verder toe dat ekonomiese faktore ook die veearts se betrokkenheid kan beïnvloed, maar dat dit nie daarom nodig is om 'n anti-diereregtestandpunt in te neem nie.

Mench & Von Tierhoven[18] meen dat die studie ten opsigte van plaasdierwelsyn steeds in sy kinderskoene staan. Etoloë, fisioloë, landbou- ingenieurs, veeartse en produseerders moet ingespan word om beide diere én hulle eienaars te bevoordeel.

Dit is interessant om daarop te let dat nie alle filosowe wat die diereregtesaak beoordeel, diereregte ondersteun nie. Tannenbaum[34] sê dat die nuwe diereregtefilosowe onaanvaarbare alternatiewe bied. Hy verwys na Regan[24] en Rolin[25] se idees as verkeerd en kortsigtig. Baie veeartse sien Regan en Rolin se etiese studies as ondermynend tot hulle belange en voortbestaan.

Tannenbaum[34] het dit veral teen Rolin se benadering wat gebaseer is op die afwesigheid van relevante, morele verskille tussen mense en diere. Tannenbaum voel ook dat die woord "regte" geïdentifiseer word met 'n spesifieke onaanvaarbare benadering. Die morele betekenis van die mens-tot-dier-verhoudings plaas die etiek rondom die veterinêre professie egter in 'n nuwe lig. Hy gee ook toe dat daar moeilike vrae is om te beantwoord en sê dat die veterinêre etiek sulke vrae moet hanteer ('n duidelike uitdaging aan die professie). In 'n tweede artikel het Tannenbaum[35] riglyne aan die veearts voorsien omdat die saak van diereregte groot verleentheid onder veeartse veroorsaak. Praktiserende veeartse sien in diereregtebewegings net nog 'n bedreiging vir hulle ekonomiese lewensvatbaarheid. Tweedens word dit ook gesien as 'n wesenlike gevaar vir die natuurlike orde, die wetenskap en vooruitgang. Derdens voel veeartse dat diereregte bots met die doel en waardes van die veterinêre professie en dat die woord "regte" misplaas is. Die stryd om diereregte het dus die veearts in die **kruisvuur** geplaas, 'n beeld wat suggereer dat veeartse òf in die proses vernietig gaan word òf vinnig moet terugtrek uit die stryd. Tannenbaum glo dat so 'n situasie ongeregverdig is, omdat daar 'n groot deel van die diereregtebewegings is wat moreel afstootlik en ernstig nadelig vir die belange van die veearts en sy kliënte is. Hy sê daar is heelwat meningsverskil oor die betekenis van "regte" en hy maak 'n verskil tussen wettige regte wat deur politici gemaak word en afdwingbaar is, en morele regte wat 'n sterker aanspraak kan maak as wettige regte.

Tannenbaum meen dat diereregtegroepe op 3 argumente faal:

(i) Om te beweer dat diere morele regte het, veronderstel dat diere 'n inherente morele waarde en betekenis het, onafhanklik van die waarde wat mense aan hulle toeken.

Deur standpunt in te neem teen wreedheid teenoor diere, kan mense wel sekere morele regte aan diere toeken, maar dit beteken nie dat so 'n siening dieselfde is as wat die ekstremiste s'n is nie. Omdat die terme "regte" aan ekstremisme gekoppel word, verkies baie mense eerder die term "dierewelsyn", wat ook meer aanvaarbaar is vir die veterinêre professie. Die ekstremiste glo dat diere se morele regte presies dieselfde is as dié van die mens. Daar is egter 'n verskil tussen dierewelsyn, menslike behandeling van diere, dierebeskerming en diereregte. Die American Veterinary Medical Association[2] sê dan ook:

"that (the) use of the term 'animal rights' has to do with personal philosophical values and therefore recommends that the term not to be used, and encourages the profession to focus its attention on the welfare and humane treatment of animals."

(ii) Die diereregtegroepe beywer hulle verder vir die wettige regte van diere tot op 'n vlak waar diere die reg sal hê om hulle eienaars, veeartse of enigeen te kan dagvaar vir geld of ander vergoeding of voordele. Dit sou ook immoreel wees om diere uitsluitlik of grootliks te gebruik tot voordeel van die mens.

(iii) Die veearts moet diere gelykwaardig aan mense beskou en behandel, en só sy roeping op 'n hoër vlak sien.

Tannenbaum verwerp ook die laaste twee stellings van die diereregtegroepe en sê dat diere wel sekere wettige regte het, maar nie op die vlak van mense s'n nie. Veeartse sal nie baat uit die diereregtegroepe se sienings nie, want dit sal nie net tot te hoë veterinêre fooie lei nie, maar ook die veearts in 'n onhoudbare posisie plaas waarin hy skaars sy professie sal kan beoefen. Tannenbaum sluit af deur te sê dat dit belangrik is om die diereregtegroepe aan die kaak te stel, maar nie so belangrik as

om aandag te gee aan die breër saak van hoe mense diere moreel en wettiglik moet behandel nie.

Rowan en Tannenbaum[28], meen dat die gebruik van diere tot menslike voordeel ten minste deels geregverdig is en sê dat 'n regte-gebaseerdebenadering, verskil van 'n koste-voordelebenadering. Hulle sluit af deur te waarsku dat die konsep van regte beide kragtig en subtiel is. Daarom behoort dit met omsigtigheid gebruik te word.

Tillman en Brooks[37] maak ook 'n besliste onderskeid tussen diereregte en dierewelsyn. Dierewelsyn het te doen met die toestande waaronder diere aangehou word en gebruik word en hoe die lot van diere verbeter kan word, terwyl diereregte klem lê op die gelykheid van mens en dier. Die dierewelsynsmense erken steeds die mens se meerderwaardigheid oor diere en glo dat die mens die reg het om diere volgens menslike behoeftes aan te wend — en die meeste veeartse val onder hierdie groep.

Marshall[17], 'n praktiserende veearts, stem met die vorige twee skrywers saam en neem stelling teenoor Fox in:

"As veterinarians, we are humanitarians dedicated to helping mankind by the betterment of his animals".

Regte is relatief en etiek hang dikwels van 'n spesifieke situasie af. Om die waarheid te sê, alle lewende dinge het regte, maar nie gelyke regte nie en om dit te ontken, is om die natuur self te ontken. In die toegepaste gebruike van geselskapsdiere is die diere nie die fokuspunt nie, maar bloot instrumente. Eienaarekonomie is ook 'n realiteit in 'n praktyk en mense sal altyd deel van 'n veearts se besigheid wees, deurdat hy mense help.

MENINGSOPNAME ONDER VOORNEMENDE VEEARTSE

'n Tweedejaarsklas van 100 studente aan die Fakulteit Veeartsenykunde, Universiteit van Pretoria is in 1986 gevra om 'n onbeperkte standpunt ("open end question") in te neem oor die vraag: "Wat is u mening oor diereregte?" Die tweedejaarstudente het nog geen kliniese opleiding ontvang nie en het dus nog met geen kliënte of siek diere in hulle opleiding in aanraking gekom nie. Verder is die standpunte ingeneem voordat die studente enige lesings oor die onderwerp ontvang het, wat hulle dalk kon beïnvloed. Die studente moes die vraag binne 30 minute beantwoord.

Om 'n onbeperkte antwoord oor 'n filosofiese vraag te klassifiseer, is nie eenvoudig nie en dit moet noodwendig 'n mate van subjektiwiteit bevat. Daar is egter gepoog om 'n spektrum in persentasies te bepaal, waarin die menings oor diereregte gekategoriseer word. Die kategorieë is soos volg:

1. Standpunte wat sterk positief oor diereregte voel.
2. Standpunte wat sekere "regte" (moeilik definieerbaar) aan diere toeken, maar nie gelyk aan dié van die mens nie.
3. Standpunte wat diereregte verwerp.

Tabel 1: Veeartsenykundestudente se standpunte oor diereregte

Kategorie	Standpunte (n = 100)	Persentasie
1	Positiewe standpunte oor diereregte	18
2	Gebalanseerde standpunte oor diereregte	67
3	Negatiewe standpunte oor diereregte	15

Om aan die resultate groter betekenis te verleen, word enkele verteenwoordigende aanhalings vir elke kategorie weergegee.

Kategorie 1: Positiewe standpunte

a. "animal rights are almost as important as human and woman rights, the fact that animals cannot talk or express themselves must not stand against them and their rights must be protected by law".

b. "It is just as inhuman to assault/abuse animals as it is to assault/abuse humans, yet the punishment for the two actions are not as severe. Let's have some democracy in the world!!"

c. "it's unfortunate that man has such delusions of grandeur about himself, that he considers it justifiable to manipulate other animals and he is one himself."

d. "being there before us, us coming from them, us surviving from them, drawing comfort and protection from them, I firmly believe animals have all the right to their place in nature, and it is rather unjust of *Homo sapiens* to deprive them of this right. The rights include eg. own privacy and own choice."

e. "animals also have a "soul" or "aura" or "personality" and can therefore perceive evil or good. They have the right not to die when man wants it."

f. "A Bill of Animal Rights is a necessity!"

Kategorie 2: Gebalanseerde Standpunte

a. "the rights of animals depend largely on man himself."

b. "animals have a right to be respected which must be part of the human's attitude. One must see the wonder of life".

c. "animals do have rights which are dependent on the situation, but no moral or ethical codes".

d. "animal rights are based on the moral principles of the individual human being".

e. "eating meat and using animals as pelts is not a

denial of rights, providing humane methods are employed — it is a symbiosis".

f. "animals have certain rights which should not to be ignored, however animals cannot be made equal to humans".

Kategorie 3: Negatiewe standpunte

a. "man can use animals for almost any purpose as long as they are cared for. The animal has no choice and has no rights".

b. "it is in the human's interest to propagate certain species for his benefit".

c. "animals have no "rights", because they are not aware of their "rights", it is only something that lives in the human mind".

d. "an animal knows no better than what it is used to — it boils down to humane treatment".

e. "animals have no rights as humans have, because animals have no rights in court".

f. "for practical purposes, animals have no rights".

Die inligting wat deur hierdie studie versamel is, dui op 'n oorwegend gebalanseerde siening oor diereregte. Twee derdes van die klas het gevoel dat diere tog 'n "reg" op versorging, die lewe, ruimte en om nie wreed behandel te word nie, kan hê, maar dat die mens steeds as die meerdere van die dier erken moet word. Die mens is ook die bepaler en uitvoerder van die "reg" van diere en dra dus 'n groot verantwoordelikheid. Alhoewel diere respek verdien en daarvolgens behandel moet word, bly die mens die belangrikste vennoot in die kontak tussen mens en dier.

Die groep wat geen regte aan diere toegeken het nie, het baie dieselfde standpunte gehandhaaf, behalwe dat die mens in 'n nog sterker posisie geplaas is teenoor die dier as deur die middelgroep. Geeneen van hierdie groepe het egter die dier as 'n voorwerp beskou waarvoor die mens geen gevoel voor moet hê nie. Sommige het die term "regte" op praktiese en filosofiese gronde verwerp, maar steeds 'n gevoel van menslikheid teenoor diere bepleit.

Die sterk voorstanders van diereregte het miskien, op enkele uitsonderings na, ook die mens se rol erken as besluitnemer oor diere. Die aangehaalde gedeeltes dui nie altyd daarop dat daar in die verdere bespreking van die onderwerp meer gematigde stellings voorgekom het nie. In sommige gevalle kon 'n mens die stellings van dieselfde student as kontrasterend of onseker beskou. Byvoorbeeld: "when we look at animals in medicine, I'm somewhat at a loss of conviction." Ander uitweë word ook gekies, soos: "the answer is difficult", "we live in a practical orientated world", "less-than-perfect society", "humans determine animals rights", "of course one must be logical".

Die gevolgtrekking is dat hierdie studente bewus is van die verantwoordelike posisie van die mens, terwyl hulle terselfdertyd 'n sensitiwiteit oor die posisie van die dier inneem. Hierdie standpunte kry veral groter oetekenis as in ag geneem word dat die studente nog geensins in hulle opleiding met die veterinêre praktyksituasie te doen gekry het nie. Sover vasgestel, is dit die eerste gepubliseerde opname onder veeartsenykundestudente oor hierdie onderwerp en opnames onder ander seksies van die professie kan dalk ook van waarde wees.

STANDPUNTE

Dit blyk dat daar hoofsaaklik twee probleme is waaroor daar duidelikheid gekry moet word. Die eerste is of diere beskou moet word vanuit die **mens se posisie**, as die meerdere van die dier, en of diere beskou moet word vanuit die **dier se posisie**, waar mens en dier, as medediere, op gelyke vlak en met dieselfde inherente waarde beweeg. Die tweede probleem is die definisie van wat presies onder die term "diereregte" verstaan word. Standpunte oor hierdie twee probleme word kortliks bespreek.

1. Antroposentrisme

Antroposentrisme is die lewensbeskouing waar die mens in die sentrum van wêreld geplaas word. Dit beteken dat alles om die mens draai en dat die mens sin en betekenis aan die natuur gee. Sonder die mens op aarde, is die res 'n doellose siklus van bestaan en nie-bestaan. Die mens bepaal die waardes van die natuurkomponente en ken dit toe volgens sy eie insig. Dit beteken dat alles nie 'n inherente gelyke waarde het nie, maar slegs dit wat die mens daaraan toeken. Die mens is ook by magte om na sy goeddunke met sy tegnologie 'n invloed op die natuur uit te oefen. So 'n standpunt ontken nie analoë en homoloë wat in mens en dier voorkom nie, dat daar meer ooreenkomste as verskille op 'n breë biologiese vlak kan wees nie, of dat daar bloot graadverskille kan wees nie. Die onderskeid tussen mens en dier lê in die psigies-fisiese totaliteit wat die mens wél duidelik van diere onderskei[7]. Die verskil tussen mens en dier lê dus in die som van hulle onderdele en die ooreenkomste tussen die onderskeie dele is dus van minder belang. Om dus bloot te verklaar dat die mens per sekere definisies 'n dier is, is 'n halwe waarheid, want in sy totaliteit gesien is die mens ook **nie** 'n dier nie.

Hierdie twee "waarhede" bring dus 'n ambivalensie van gelykheid én ongelykheid na vore. Aan die een kant staan die mens as die middelpunt van dit wat betekenis gee aan die wêreld, en aan die ander kant het die mens vele ooreenkomste en deel hy dieselfde lot, nl. die dood met diere én plante. Die eersgenoemde waarheid is die bepalende, maar sluit nie die tweede waarheid uit nie. Standpunte teen antroposentrisme sluit egter die eerste waarheid uit en bly dus die volledige waarheid ontwyk. Die mens het homself nie willens en wetens in hierdie posisie geplaas nie, ongeag watter lewensbeskouing 'n mens ook al huldig. Die bepaalde hoë of belangrike posisie van die mens in die wêreld gaan egter gepaard met besondere groot verantwoordelikheid — soos enige hoë posisie. Die emosiebelaaide beskuldigings aan die mensdom dat die mens homself in 'n posisie geplaas het as die tiranspesie, dominante spesie, meerderwaardige of intelligenter spesie, verval sodra die siening gehuldig word dat die mens die **verantwoordelike spesie** is. Niemand, nie eers diereregtegroepe nie, kan die mens verkwalik dat hy die posisie van die verantwoordelike spesie inneem nie, want die mens het dit nooit self

beplan of bedink nie. Antroposentrisme beteken ook dat die vergelykings tussen die strewes van sekere groepe vir menseregte, geensins vergelykbaar is met die strewes van sekere menslike groepe vir diereregte nie. Sulke absurde argumente verval dus.

Antroposentrisme is dus

1.1 **moreel geregverdig,** omdat dit nie die mens self is wat op 'n doelbewuste wyse hom in sy posisie geplaas het nie;

1.2 **prakties,** omdat dit perspektief behou op die gegewe en normale orde van lewe op aarde;

1.3 **verdedigbaar,** omdat dit klem lê op die verantwoordelikheid van die mens en dus misbruik van die posisie verwerp. Indien so 'n verantwoordelikheid dieselfde effek of resultate kan verkry, as diereregte en gelykheid, dan word die strewe na diereregte irrelevant. Dit versterk (en verhoed nie) die respek vir lewe en die totale natuur en dit erken die enigma van lewe;

1.4 **noodsaaklik en aanvaarbaar** vir veterinêre praktyk. Om 'n gebalanseerde praktyk te beoefen moet die veearts die pasiënt (dier), sowel as die kliënt (mens) in die sisteem van 'n konsultasiekamer in ag neem, en laasgenoemde is dikwels die bepalende faktor. Dit alles moet geskied met die nodige verantwoordelikheid wat die mens (kliënt en veearts) besit, om ten beste na die dier se welsyn om te sien. **Geen kliënte** beteken immers **geen praktyk.**

Praktyk en papier kan ook verskil: praktiese toepassings van die biologie soos in veterinêre praktyk kan 'n totaal ander ervaring wees as wat moraalfilosowe op papier ondervind. Veterinêre besluite oor 'n dier se lot kan kwalik blóót vanuit die standpunt van diereregte, of dan gelyke regte met die mens, geneem word. Die konsultasie sluit óók die emosies van die kliënt in, sowel as die praktiese waarde van die dier en die kliënt se finansiële status. Verder bepaal dieregetalle nie die noodsaak en behoefte aan veterinêre dienste nie, maar wel die aanvraag van belangstellende kliënte. Veterinêre besluite word dus grootliks bepaal deur die waarde wat die kliënte aan die diere toeken. 'n Professionele benadering van die veearts impliseer ook in die eerste plek diens aan die gemeenskap, dus die medemens. So gebeur dit in private praktyk dat 'n veearts genadedood aan 'n gesonde dier moet toedien en lydende terminale diere aan die lewe hou. Albei laasgenoemde voorbeelde het natuurlik perke, maar dit is geensins ongewone voorbeelde uit 'n praktyk nie. Baie van die kritiek van diereregtegroepe getuig ook van 'n gebrek aan kennis oor diere se werklike gedrag, behoeftes en aanpassingsvermoë. Op papier kan sekere voorbeelde baie wreed lyk, terwyl die betrokke diere alle tekens van welsyn toon soos gesondheid, eetlus, teling en gebrek aan sigbare angs.

Die finale keuse van standpunte vir die veearts lê in die ménslike lewensbeskouing wat diereregte vanuit 'n absoluut gelyke posisie met die dier beskou, óf die omsien na diere (en die res van die natuur) se welsyn vanuit 'n verantwoordelike antroposentriese oogpunt. Die eerste keuse is prakties onuitvoerbaar, vergesog en neem nie die gegewe werklikhede in ag nie. Die tweede standpunt is geregverdig, behou perspektief en kan in veterinêre praktyk toegepas word.

2. Definisie rondom diereregte

Die term diereregte is ten opsigte van "regte" sowel as "diere" tot nou toe ondefinieerbaar.

2.1 Definisie van regte

Die definisie van "regte" lewer probleme op as 'n mens die betekenis daarvan wil beoordeel ten opsigte van ander terme soos dierevryheidsbewegings, dierebevryding, dierewelsyn, dierebeheer en dieregebruik.

Ander probleme ontstaan weer oor wátter regte presies ter sprake is. Is dit sogenaamde natuurlike regte, wetlike regte, vryheidsregte ten opsigte van keuses en ruimte, eiendomsreg, demokrasie (stemreg?), regte **oor** diere of regte **vir** diere. Voordat hierdie spraak- en ideeverwarring nie opgeklaar is nie, laat dit die term te wyd oop vir kritiek van vele kante. Die kwessie van watter regte presies ter sprake is, word vir eers daar gelaat en daar word aanvaar dat die regte handel oor die absolute gelykheid tussen mens en dier. Die aanvaarding van so 'n gelykheid moet dan die regte van diere verseker. Dit bring die bespreking by die vraag waar die onderskeid dan getrek moet word om vir diereregte te kwalifiseer, en wat of wie van diereregte uitgesluit moet word.

2.2 Definisie van diere

Dit kan aanvaar word dat die gelykheid tussen mens en dier nie universeel bestaan nie, want anders sou daar nie diereregtegroepe wees wat hulle vir so 'n aanvaarding beywer nie. Hierdie groepe baseer grootliks hulle strewe vir diereregte op grond van die gelykheid tussen mens en dier. Om hierdie gelykheid egter te bereik kan daar slegs van die "hoër" vlak na die "laer" vlak beweeg word, of van die "laer" na die "hoër" vlak. In so 'n gelykheidsproses word daar ongelukkig meer van woorde gebruik gemaak as van enigiets anders. Na die mens word verwys na die "mens-dier" (vergelyk Donahue[6] se *The Human-animal*), en na die diere as die "nie-mense" (non-humans). Deur woorde, wat die mens met die dier, en die dier met die mens op gelyke vlak in verband bring, word bloot die persepsie van gelykheid bevorder. Dit is meesal baie moeiliker om die laer vlakke gelyk met die hoër vlakke te bring en uiteindelik word daar meer gekonsentreer om die gelykheidslyn op die laagste vlak te bereik. Dit beteken dat dit baie gewilder is om "die dier in die mens" te beklemtoon, as om van diere dieselfde verantwoordelikhede as van die mens te verwag. Diereregtegroepe maak ook dikwels van argumente gebruik wat vergelykings tref tussen mense wat op die rand van 'n gewone gemeenskap se kulturele uitlewing voorkom (soos babas (potensiaal uitgesluit), en persone met permanente breinskade) en enkele hoogs geleerde diere (soos sjimpansees wat kan praat, perde, honde en dolfyne). Biologiese beoordelings kan egter nie op die rand van verskynsels gedoen word nie, maar vanaf die tendens of neiging. Die randverskynsels gee bloot

verdere insig in biologiese verskynsels maar bepaal nie die rigting nie.

Die kritiese vraag waar presies die gelykheidsvlak van regte (die laagste vlak?) van mens en dier sou lê, moet begin met die definisie van "diere". Diereregtegroepe is laks om so 'n definisie te formuleer. Die poging om die streep by die molluske te trek, het skerp kritiek uitgelok. 'n Mens moet dus aanvaar dat **alle** diere ingesluit is, alhoewel baie argumente slegs die diere wat in die nouste verhouding met die mens staan, in gedagte hou. Al sou alle diere ingesluit word, is die probleem nog nie opgelos nie. Daar bestaan tussenvorme wat klassifikasie bemoeilik. Verder is daar lewensgevaarlike spesies in die diereryk. Wie gaan beslis waar watter spesie se regte geld en waar nie? Dit lyk na 'n onbegonne taak. 'n Mens kan ook nie eers sê dat die mikro-organismes net randverskynsels is nie, want in die "demokrasie van diere" waarna voorheen verwys is, maak hierdie organismes beduidende getalle uit.

As die argumente wat aangevoer is vir diereregte konsekwent verder gevoer word, dan lyk dit of die gelykheidslyn selfs plante kan betrek. Om plante by hierdie bespreking te betrek is geensins vergesog nie, maar belangrik in die lig van uitsprake teen die eet van vleis deur die mens. As die beginsels na plante deurgevoer kan word, sal dit dus beteken dat die mens ook nie plante mag eet nie en dus van honger moet omkom. As daar nie 'n duidelike onderskeid gemaak word in die definisie van diere nie, is daar in elk geval niks wat verhoed dat plante by die argumente betrek word nie.

Daar is eienskappe van plante wat baie goed vergelyk met eienskappe wat in mens en dier voorkom. As die oppervlakkige en voor-die-handliggende verskille as van minder belang beskou word, is dit waarskynlik nie onmoontlik om uit 'n breë biologiese oogpunt, óók meer ooreenkomste as verskille tussen diere en plante aan te toon nie. Van Hoven[39] sê dan ook in sy ondersoek na reaksies van bome: "Plants consequently have a built-in chemical defence mechanism which can roughly be compared with the chemical defence mechanism people and animals have..."

Die ondersoek handel oor wreedheid teenoor plante, waar die plante met voorwerpe geslaan en verniel is. Sulke behandelings het nie net chemiese verdedigingsmeganismes (vergelyk adrenalien in mens en dier) in werking gestel om so hulle "emosies" te toon nie, maar het ook hierdie lyding aan nabygeleë plante gekommunikeer. Onbeseerde plante het tot so ver as drie meter vanaf die beseerde plante, dieselfde reaksies ("emosies") getoon. "...the ability of plants of the same species to communicate with each other was proved under laboratory conditions, and from the given examples, it can be deduced that certain plants will probably also do so under natural conditions."

Sonder om veel verder uit te brei, is die standpunt bewys dat plante volgens die diereregtegroepe se beginsels, nie summier uitgesluit kan word nie. Dit is veral waar, as 'n mens die stelling waarop sommige mense diere se regte baseer, in ag neem. Dit lui soos volg: die vraag is nie of hulle kan praat of dink nie, maar of hulle kan ly.

Ander "emosionele" reaksies wat baie bekend is, is die reaksies van plante op musiek. 'n Voorbeeld van so 'n eksperiment waar plante onder dieselfde omstandighede aan klassieke musiek en geraas blootgestel is, is weer op die Expo vir Jong Wetenskaplikes (1986) uitgestal. Die plante wat aan die klassieke musiek blootgestel was, het gefloreer, terwyl dié wat aan geraas blootgestel was, gekwyn het. Die mening oor plantliefhebbers dat plante beter reageer teenoor sekere mense as ander, is ook 'n gevestigde geloof. Daar word na verwys as sou die mens "groen vingers" hê en kan dui op 'n nog onbekende positiewe "kommunikasie" of "emosie" tussen mens en plant.

Vanuit die basiese biologiese berip van "lewe" kan daar na nog talle ooreenkomste verwys word. Die feit bly staan dat met sulke argumente, die volgende stap nl. die bepleiting van planteregte voor die deur staan.

Ongelukkig is die streep wat by plante getrek word ook nie noodwendig finaal nie. Margulis & Sagan[16] glo dat die mens met die masjien (tegnologie) 'n eenheid sal vorm, omdat beide uiteindelik ongeskik sal wees in hulle huidige vorm van bestaan: "It is ... possible that the two will merge, in some unimaginable form, to escape a dying planet. It may sound outrageous to suggest that life and non-life could ever blend or breed. But viewed in the context of the history of life — from its prebiotic origins to its present-day ability to manufacture itself in a laboratory — such a feat begins to look not only plausible, but **inevitable**."

Die etiese vraag wat onmiddellik na vore kom, is dat as 'n samesmelting tussen mens en masjien onafwendbaar is, of daar nie nou reeds na masjienregte (tegnologiese regte?, nie-lewe-regte?) gekyk moet word nie. Hierdie nuwe skepsel sal beslis betrek moet word by die morele aspekte van lewe. As dit menslike komponente bevat, sal dit ook kan ly en aanspraak maak op regte. Dalk sal die mens weer wag totdat dit te laat is en hierdie nuwe skepsel, die "mens-masjien" (vergelyk die "mensdier") eers vir lank aan onderdrukking, tirannie en verontregting onderwerp, voordat sy regte erken word. Erger nog, gaan hierdie nuwe skepsel die regte van die moontlik minderwaardige, gewone, mense en diere erken? Dit blyk dat sonder 'n behoorlike en algemeen aanvaarde definisie van wat met diere bedoel word, daar geen duidelike streep getrek kan word waar diereregte uitgeoefen gaan word nie. Die gevolgtrekking is dat slegs die aanvaarde onderskeiding wat tussen mens en dier bestaan, die probleem kan oplos. Die regte van mense kan wetlik gedefinieer word en in 'n regstelsel toegepas word. Daar is geen plek vir enige wette wat nie direk of indirek die mens betrek nie. Regte word net relevant as dit uitgeoefen kan word en regstelsels is geskep om dit uitvoerbaar te maak.

Die enkel-organisme-siening. 'n Werk wat meer insig in die verhouding tussen mens en dier, asook alle ander dinge op aarde, kan gee, is Lovelock[14] se *Gaia — A new look at life*. Gaia is die naam van 'n griekse godin wat beskou was as die godin van die aarde. Gaia word deur Lovelock gebruik om die globale sisteem te beskryf wat die wêreld se makroprosesse in homeostase hou. Lovelock sien die totale lewe, lug, aardkors en oseane as 'n

eenheid en voer aan dat geen lewe onafhanklik van die ander komponente gesien kan word. Die biosfeer, atmosfeer, geosfeer en hidrosfeer vorm dus 'n eenheid wat soos 'n groot enkel lewende organisme funksioneer:

"We have ... defined Gaia as a complex entity involving the Earth's biosphere, atmosphere, oceans and soil; the totality constituting a feedback or cybernetic system, which seeks an optimal physical and chemical environment for life on this planet."

Lovelock sê sy siening is in teenstelling met die konvensionele wysheid dat lewe by die aarde se toestande as sodanig moes aanpas, en dat lewe 'n aparte evolusie was. Die gaia-sisteem wat die aarde as 'n makro-organisme voorstel, het dus eienskappe wat nie deur die som van sy onderdele voorspel kon word nie, anders sou homeostase nie bereik of gehandhaaf kon word.

Met hierdie globale insigte word die hele kwessie van "regte" van minder belang, omdat daar in elk geval 'n gelyke eenheid bestaan. Dierereğte, planteregte, mineraleregte, atmosfeerregte, masjienregte, ensovoorts, kry 'n ander betekenis as alles deel is van één "organisme". Die behoud van homeostase lê juis daarin dat elke deel van die organisme sy eie besondere funksie verrig. In so 'n enkele terugvoersisteem is almal en alles gelyk met "gelyke regte". Nogtans is alle dele van die liggaam nie gelyk nie. As 'n mens as enkel organisme, 'n keuse het om 'n punt van 'n pinkie of 'n oog te verloor, sou die oog beslis meer waarde hê en ongelyk teenoor die pinkie staan. Net so met 'n keuse tussen die verlies van 'n blindederm en 'n lewer, ensovoorts. Die ambivalensie wat weer hier na vore kom, naamlik, dat alles terselfdertyd gelyk én ongelyk is, word selde deur fanatici aan beide kante van die spektrum van menings oor diereregte, raakgesien. Enersyds word die gelykheidsdrywing dan 'n nuttelose moeite en andersyds hoef ongelykheid nie tirannie te beteken nie.

Om die posisie van die **mens** in hierdie globale organisme vas te stel, kan dit met die **brein** van 'n liggaam (organisme) vergelyk word. Beide is die verantwoordelike dele van die organismes. Indien die menslike brein genoeg breinskade opgedoen het, gaan sy verantwoordelikheid verlore. Sonder die mens in die globale sisteem gaan die verantwoordelikheid ook verlore. Net soos die brein nie skuldig hoef te voel omdat hy nie die punt van die pinkie is nie, net so hoef die mens nie skuldig te voel dat hy die verantwoordelike spesie is nie. Die brein moet bepaal dat die res van die liggaam nie benadeel word nie, al is die brein in staat tot misbruik van die liggaam. Die mens moet sy posisie ook gebruik tot voordeel van die res van die sisteem, alhoewel hy die sisteem deur misbruik kan benadeel.

VERDERE PROBLEEMVRAE

A. Die indruk word geskep dat daar 'n teenstelling bestaan in die sienswyses van diereregtegroepe. Aan die een kant word die mens gelyk gestel aan die dier, maar aan die ander kant word 'n baie hoër moraal van die "mens-dier" as van ander diere verwag. So byvoorbeeld word daar nie oor regte in ander voedselkettings gepraat nie, maar die mens word veroordeel vir sy benutting van ander diere vir voedsel. Ander voedselkettings is dus moreel geregverdig, terwyl dit in die mens een van die laagste morele oorwegings is. Daar word ook geen etiese kode of moraal aangelê vir wreedhede in die natuur nie, maar slegs vir die "mens-dier". As daar geen verskil (behalwe vir " 'n gedeelte van die brein in kwantiteit") tussen mens en dier bestaan nie, behoort diereregte-aanhangers ook wreedhede van mense oor te sien. Dit is egter juis vanweë die ongelykheid tussen mens en dier dat die mens 'n moraliteit het wat wreedheid teenoor diere veroordeel. Dit is 'n direkte gevolg van die mens se verantwoordelikheid, en nie as gevolg van diereregte of -gelykheid, dat die moraal geld nie. Diereregtegroepe verwag van die "mens-dier" om soos 'n mens op te tree, terwyl die mens eintlik soos ander diere behoort op te tree.

B. As die mens en dier gelyk is, of na gelykheid strewe, is dit nie duidelik waarom slegs **een party** tot hierdie gelykheid moet **bydra** nie. Sou dit nie billik wees om in die gelykheidstrewe ook pogings van die "ander diere" te verwag nie? Omdat gelykheid meesal op die laagste vlak bereik word, beteken dit dat die maatstaf van gelykheid die maksimum potensiaal van die ongelykste of laagste vorm van dierelewe sal wees. Dit beteken dat die mens in sy ongevraagde verantwoordelike posisie, gelyke regte met die primitiefste organismes sal moet deel, sonder dat die organismes iets aanwend om die mens tegemoet te kom. Verder lyk dit of daar net een party in 'n dispuut oor regte skuldig bevind kan word en vergoeding kan betaal. Dié ongelykheid maak 'n uitsluitsel oor regte irrelevant.

C. Aangesien daar so moeilik onderskeidings gestel word in die argumente van die diereregtegroepe, bly die posisie van die dier tot die mens onseker. Is daar enige versekering wat gegee kan word dat diere nie nou weer so hoog gestel sal word dat dit in die mens se gedagte die ideale norm word nie? Wanneer die stadium van die "edele dier" bereik word, kan mense sover gaan om hulle van die menslike spesie te distansieer. In die openbare media is bekendes soos Brigitte Bardot en Spike Mulligan aan sulke menings gekoppel. Alle wreedhede en onreg word dan met die mens as spesie vereenselwig. Daar kan selfs beweeg word na die baie primitiewe benadering van **diereverafgoding** en **-aanbidding.** Wat gaan verhoed dat daar weer 'n dierekultus en -godsdiens ontstaan vol heilige diere? Bustad[1] rapporteer van 'n volledige liturgie wat deur 'n biskop Richard York saamgestel is vir die begrafnis van geselskapsdiere:

"The purpose of a pet's burial ceremony is to console the bereaved and to reaffirm our solemn responsibility to the intelligent Voice of Creation."

Katcher & Beck[13] sê "met respek", dat om met 'n geselskapsdier te praat dieselfde voordele kan inhou as om te bid. Dit impliseer dat die verhouding tot God en 'n geselskapsdier op dieselfde vlak kan wees en dat die dier dus die plek van God kan inneem. Alhoewel diereregtegroepe hulleself graag as vooruitstrewend beskou, sal die vordering tot diereaanbidding 'n groot terugwaartse stap wees.

By die afsluitingseremonie van die Boston kongres oor mens/dier-interaksies in 1986, het Prof. Loew as Dekaan van die Fakulteit Veeartsenykunde, Tufts Universiteit, Boston, inderdaad gewaarsku

teen 'n dierekultus en die materiële uitbuiting van hierdie hoë agting vir geselskapsdiere.

D. Nog 'n belangrike vraag om te beantwoord is: **namens wie** tree die diereregtegroepe op? As die antwoord sou wees dat hulle praat namens dié wat nie vir hulleself kan praat nie, kan so 'n stelling boemerang. Volgens die diereregtegroepe se eie norme behoort diere die vryheid van keuses te hê en weens die kommunikasiegaping tussen mens en dier, is dit te betwyfel of hierdie groepe met oorgawe kan sê dat hulle seker is dat hulle presies doen wat die diere van hulle verwag. Die diereregtegroepe kan slegs die regte vir diere beoordeel van 'n subjektiewe menslike oogpunt, en dit is nie noodwendig dieselfde as die diere se oogpunt nie. Verder kan 'n mens vra of diereregte so universieel onder die diere is, dat die strewe na diereregte vir alle diere dieselfde betekenis het.

As hierdie groepe beweer dat hulle vir die diere intree namens **mense** wat glo in diereregte, teenoor die mense wat dit nie wil aanvaar of toepas nie, dan verteenwoordig hul seer seker net 'n onbeduidende breukdeel van die wêreldpopulasie van mense. Tot nou toe verteenwoordig hulle ook nie die grootste deel van die veterinêre professie nie. Onder die professionele filosowe waar diereregte die grootste aanhang het, is diereregte nog grootliks 'n omstrede saak. As diereregtegroepe net namens hulleself praat en namens die getalle wat hulle verteenwoordig, dan kan 'n mens tereg vra wat van die "demokrasie" onder alle spesies geword het, waarvan daar melding gemaak is.

TOEKOMSBLIK

'n Mens kan dus wonder vanwaar die diereregtegroepe hulle aanhang kry, hoe ver hierdie saak gevoer kan word en hoe dit uiteindelik die veterinêre professie gaan raak. Die diereregtegroepe sal weens twee hoofredes wel sukses in die **moderne** gemeenskappe behaal, naamlik:

A. Die **metodes** wat aangewend word om diereregte te bevorder. Hier word gedink aan organisasies met genoeg finansiële ondersteuning en personeel, deelname aan publieke en wetenskaplike kongresse en publikasies, betogings, optogte, aktivistiese optredes om die aandag op diereregte te vestig en beïnvloeding van politici, ander owerhede en belanghebbendes om wetgewing aan te pas. Diereregte gaan beslis 'n politieke speelbal word.

B. Ondervinding het geleer dat die moderne gemeenskappe maklik tot skuldgevoelens aangespoor word. Deur die regte tegnieke te gebruik om emosionele oplewing te kry, kan baie mense deur die **ag-foeitog-respons** tot simpatiseerders van die diereregtegroepe oorgehaal word. Massasielkunde deur middel van alle media en pamflette sal die oortuigingswerk doen.

Dit is dus te verwagte dat amptelike erkenning aan diereregte in moderne gemeenskappe verleen gaan word. Die wetgewing sal gelei word deur "oortuigde wetgewers" en die aandrang van 'n "simpatieke publieke mening". Ongelukkig sal die wetgewing oor diereregte, eensydig, slegs in die mense se regstelsel van toepassing wees.

Vir die veterinêre professie sal sulke ontwikkelings verreikende implikasies hê. Alle veterinêre oorwegings sal aan die hand van die diereregtefilosofie getoets moet word. Hierdie maatstawwe sal egter deur ménse aangelê word en oortredings sal volgens ménslike regstellings beoordeel word. As die logiese konsekwensies van diereregte deurgevoer word, sal daar uiteindelik geen verskil tussen die veterinêre en mediese professies wees nie. Die twee professies sal één word waarin "mens-diere" mede "mens-diere" en "nie-mense" behandel. As álle diere by die regte betrek word, sal dit 'n geweldige bykomende las op die professie lê. Baie nuwe veeartse sal opgelei moet word om byvoorbeeld die geweldige insekteryk te bedien. Daar is al beweer dat ons in die "tydperk van die insek" lewe en volgens die demokratiese beginsel sal hierdie 'n uiters belangrike groep wees wat op veterinêre dienste aanspraak kan maak.

Die veterinêre professie sal verder moet hulp verleen om moeilike etiese besluite te neem. Sou dit reg wees om duisende inwendige parasiete te vermoor om een skaap te red? Diere sal volgens eie vrye keuse beweeg en aanteel. Veeartse sal verplig wees om na beseerde huisvlieë en honger pirannas om te sien. Ander etiese vrae soos natuurlike voedselkettings en die plek van bakterieë in diereregte, sal hanteer moet word.

Indien die voorbeelde te vergesog is, sal daar weer na onderskeidslyne gekyk moet word. As dít die geval is, kan daar net sowel teruggekeer word na die bestaande onderskeide tussen mens en dier.

Die hele veterinêre etiek wat op die oomblik deur die kliënt en gemeenskap bepaal en beoordeel word, sal vervang moet word met 'n gemeenskaplike diere-etiek wat deur álle diere bepaal en beoordeel moet word. Of so 'n etiek van 'n hoër of laer standaard sal wees, sal oorweeg moet word.

Daar bestaan geen middeweg tussen die verwerping van diereregte en die deurtrek van die konsekwensies daarvan nie. Óf die mensdom behandel diere met respek weens hulle verantwoordelikheidsgevoel, óf diere word as gelykes aanvaar met die konsekwensies dat die mens sy hoër en verantwoordelike posisie prysgee. In 'n posisie van gelykheid kan verantwoordelikheid nie net van een kant af kom nie. Daarom is dit noodsaaklik dat veeartse duidelik sal aantoon in watter rigting hulle die professie wil stuur. As dít nagelaat word, sal veeartse verplig wees om deur ander buite die professie gelei te word, tot 'n punt waar hulle dalk nie graag sou wou wees, of voorsien het nie.

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MYOCARDIAL CONDITIONS OF DOMESTIC ANIMALS IN SOUTHERN AFRICA

Proceedings of a workshop arranged by the Pathology Group of the South African Veterinary Association at the Sandton Holiday Inn on 16 August 1986.

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INTRODUCTION

In southern Africa cardiac failure in livestock can result from a variety of toxic, infectious and nutritional causes. Many indigenous plant species are known to be cardiotoxic and stock losses caused by some of these species are of economic importance. However, the pathogenesis and pathology of poisoning by a number of these plants have received little attention and need to be investigated in greater detail. Recently several outbreaks of salinomycin poisoning in horses, in which primarily the heart was affected, came to our notice.

The workshop was intended to summarise current knowledge on myocardial conditions, particularly in livestock, and to highlight some of the research being done locally in this field.

CARDIAC GLYCOSIDE INTOXICATION IN SOUTHERN AFRICA

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Plants containing cardiac glycosides of both the cardenolide and bufadienolide types occur worldwide. Although incidental poisoning of stock is recorded from various countries, it would appear that in southern Africa only, it is of major significance. It is regarded as one of the 7 most important plant poisoning syndromes of the region.

The following groups of bufadienolide containing plants are of economic importance:

Tulp (Iridaceae): *Homeria pallida* (yellow) and *Moraea polystachya* (blue) are prime examples of the two toxic genera of this family.

Slangkop (Liliaceae): *Urginea sanguinea* is by far the most serious threat, but other species from this genus and other genera are involved.

Plakkies (Crassulaceae): The genera *Cotyledon*, *Tylecodon* and *Kalanchoe* are involved.

Thesium spp. (Santalaceae): root parasites of various Karoid plants have recently been proved to contain bufadienolides.

Cardiac glycosides inhibit the cell membrane sodium: potassium ATP-ase pump and the changes encountered can be related to this. This phenomenon is also utilised in a diagnostic test for cardiac glycosides, where the ability of normal erythrocytes to transport rubidium across the cell membrane is measured following exposure to extracts of tissues from animals suspected of having been poisoned.

Two distinct poisoning syndromes are encountered. Acute intoxication affects mostly cattle and sheep due to the ingestion of especially tulp and slangkop. Animals rapidly learn to avoid these plants and intoxication in practice only occurs in those animals exposed to these plants for the first time. The 4 systems affected are the cardiovascular and gastrointestinal (where the typical signs of digitalis intoxication are seen), the respiratory (in severe involvement dyspnoea and apnoea may occur) and the neuromuscular system (posterior paresis and terminal paralysis). Chronic intoxication is most commonly encountered in sheep and goats. It is due to extremely cumulative bufadienolides from the toxic plakkies (Crassulaceae) and results in a botulism-like, paralytic syndrome ("krimpsiekte").

The discovery that activated charcoal, at an oral dosage rate of 2 g kg^{-1} is a most effective adsorbtive antidote in acute intoxication in farm animals, means that this is most valuable in the armamentarium of the veterinarian. Research on treatment is now concentrated on the alleviation of clinical signs caused by absorbed glycosides. Lignocaine, atropine and multipotent tranquillisers, like acetylpromazine with membrane-stabilising effects, seem indicated.

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ACUTE AND CHRONIC CARDIOMYOPATHIES CAUSED BY MONOFLUORACETIC ACID IN GIFBLAAR *(DICHAPETALUM CYMOSUM)* AND CARDIAC GLYCOSIDES IN SLANGKOP *(URGINEA SANGUINEA)*

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Single or multiple doses of fresh gifblaar or slangkop material or extracts thereof, experimentally produced either acute, subacute or chronic poisoning in goats, while only acute poisoning was produced in antelopes. Myocardial lesions which were macroscopically and microscopically indistinguishable were caused by monofluoracetic acid (MFA) in gifblaar and cardiac glycosides in slangkop. In both intoxications these macroscopical lesions were characterised by areas of degeneration and occasionally haemorrhages, and were most frequently present in the papillary muscles, as well as in the subendocardial region of the left ventricular wall, and the apex. In some acute cases, a regional pallor or almost generalised parboiled appearance was noticeable. Multiple foci of degeneration were distributed throughout both ventricles in a few subacute and chronic cases.

In acute cases, miroscopic changes consisted mainly of rarefaction, hydropic and fatty changes, granular and Zenker's degeneration and fragmentation. However, in some acute cases especially in gifblaar poisoning, only hydropic changes were detectable. Fatty changes were suspected in both types of poisoning but confirmation was only attempted in cases of gifblaar poisoning. Mild leukocytic infiltration and mineralisation were occasionally seen. Subacute to chronic cases developed from Day 3 to Day 33 in goats but none were produced in antelopes. Macrophage mobilisation and fibroplasia were encountered at any stage from Day 2. Myocardial degeneration was found in a foetus from a goat ewe that died on Day 9 of experimental slangkop poisoning. When compared to goats both eland and kudu were much more resistant to gifblaar and slangkop poisoning, while springbok were as susceptible to gifblaar but very resistant to slangkop poisoning.

The experimental findings of both acute and chronic myocardial lesions were borne out by field cases in cattle and small stock that died of heart failure in gifblaar and slangkop areas. Myocardial degeneration was also found in a bovine foetus which was aborted by a cow that suffered from slangkop poisoning. In South West Africa, the number of mortalities during spring and early summer associated with myocardial lesions is indeed very perturbing.

GALENIA AFRICANA L. (AIZOACEAE) POISONING IN SHEEP

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Little is known about the toxicity, pathogenesis and pathology of *Galenia africana* (kraalbos) poisoning. Under field conditions, ingestion of the plant has been associated with liver damage and severe ascites, referred to as "waterpens" or water belly, in sheep and goats. Clinical cases of "waterpens" seem to develop more commonly in ewes and especially in those that are in poor condition during times of drought when they are forced to browse the plant. The marked liver lesions in sheep and goats associated with kraalbos poisoning led farmers and researchers to believe that the plant contains a still unidentified hepatotoxin. The hepatic lesions seen in a recent field case as well as those found in a retrospective study on preserved cases in the files at the Veterinary Research Institute, Onderstepoort, were compatible with cyanotic induration, possibly resulting from chronic congestive right heart failure. In most of these cases where myocardium was available, multifocal lesions which ranged from acute to chronic, were present.

A sheep was dosed over a period of 51 d with milled *G. africana,* at levels of 5 g kg^{-1} body mass from Day 0-6, 10 g kg^{-1} from Day 7-13 and 15 g kg^{-1} from Day 14-51 of the experiment when the animal developed ruminal stasis. Terminally (Day 58) the cardiopulmonary flow index was calculated by means of the isotope ^{99m}Tc- pertechnetate, and a value larger than 10 was determined (normal for sheep is 6-7), indicating cardiac dysfunction. Two d later the sheep died of heart failure. At necropsy the liver was markedly enlarged and yellow-brown with prominent lobulation. Microscopically, marked centrilobular fibrosis with bridging of contiguous lobules and duplication of central veins was seen. No gross myocardial lesions were noted, but microscopic examination revealed multifocal areas of vacuolar degeneration, hyaline degeneration and necrosis, mononuclear cell infiltrations and fibrosis mainly subepicardially in the right ventricle and in the interventricular septum.

Further experiments to determine whether the liver lesions are induced by a hepatotoxin or whether they result from congestive heart failure are to be performed.

CARDIOMYOPATHIES CAUSED BY JANUARIEBOS *(GNIDIA POLYCEPHALA)* AND AVOCADO *(PERSEA AMERICANA)* LEAVES

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Four sheep were drenched either with dried or freshly ground-up *Gnidia polycephala*. Multiple doses were administered for 4 to 17 days and the dosages varied from *c.* 4 - 20 g kg^{-1}. The total mass dosed for each animal was 2,0; 2,3; 2,85 and 8,5 kg respectively. The most important clinical findings were rumen stasis followed by colic, foaming at the mouth, grinding of the teeth, anorexia and diarrhoea. The latter was apparently a sequal to the ruminal stasis. The sheep which received a total of 2 kg died naturally on Day 13. The other sheep had to be destroyed on Day 9, Day 10 and Day 30, respectively. At necropsy, ruminal stasis and ulceration of the forestomachs were fairly constant lesions. Hepatic degeneration, enteritis, ascites, suspected myocardial degeneration and bronchopneumonia were seen in one or more of the animals.

Three sheep were drenched with ground-up fresh avocado *(Persea americana)* leaves collected on a farm in Grootfontein where several goats had died 3 d after being fed avocado leaves. One sheep which received 1 kg daily for 3 d died suddenly on Day 5 with a submandibular oedema and without showing any other ill-effects. A second and third sheep were drenched with 250 g daily for 19 d and 100 g daily for 30 d respectively. Both sheep were normal except for a terminal rise in BUN and AST as well as a cardiac arrhythmia in the latter. Blood coagulation time was enhanced in both. The 2 sheep were slaughtered on Day 21 and Day 32, respectively. At necropsy, haemorrhagic lesions in the papillary muscles, hydropericardium, ascites, hepatic degeneration and nephrosis were detected in the first sheep, whereas the other revealed suspected myocardial degenerative foci and nephrosis only.

Microscopically 2 distinct cardiomyopathies were distinguishable: *Gnidia* poisoning induced very specific changes in the myocardium consisting of multifocal proliferative and degenerative lesions. These lesions were characterised by a bizarre pattern of degenerating myocardial fibres with mildly karyomegalic sarcolemmal nuclei and intermingling malshaped, elongated or curved and sausage-like interstitial nuclei surrounding vessels and capillaries. The proliferative, interstitial reaction was accompanied by a mild infiltration of leukocytes and macrophages. The periphery of the myocardial fibres seemed to become lytic and vacuolar while the inner portion showed eosinophilic coagulation and homogenisation. This vacuolisation was probably partially responsible for the malshaped nuclei. On the other hand, heart lesions in the sheep that died on Day 5 of avocado poisoning comprised Zenker's degeneration, haemorrhages, severe congestion and capillary sludging with a very mild polymorphonuclear reaction and karyorrhectic debris in the capillaries. Mild multifocal lesions were present in the other 2 sheep.

DIE BELANG VAN ATROFIESE MIOKARDIALE VESELS IN DIE DIAGNOSE VAN GOUSIEKTE

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'n Tentatiewe diagnose van Gousiekte kan slegs deur middel van 'n histopatologiese ondersoek van die miokardium bevestig word. In die meerderheid van diere is die letsels beperk tot die endokardiale sone van die apeks, linker ventrikel en interventrikulêre septum. Die patologiese veranderinge word gekenmerk deur multifokale tot aaneenlopende areas van fibrose met 'n infiltrasie van hoofsaaklik makrofage en limfosiete.

In 'n lae persentasie van beide natuurlike en eksperimenteel-verwekte gevalle van die vergiftiging, is gevind dat atrofie van die hartspiervesels die opvallendste verandering is. 'n Elektronmikroskopiese ondersoek het getoon dat die atrofie te wyte is aan 'n vermindering van die hoeveelheid miofibrille. Die kliniese beeld en makroskopiese letsels in beide diere met kenmerkende hartspierletsels, en diere met hoofsaaklik atrofie van hartspiervesels, is ononderskeibaar.

DIE BEHEER VAN HEMODINAMIESE VERANDERINGE TYDENS GOUSIEKTE BY SKAPE

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Gousiekte is 'n kongestiewe dilatoriese kardiomiopatie van herkouers wat volg op vergiftiging met *Pachystigma pygmaeum* en sekere ander plante van die Rubiaceae familie[1 2 10]. Gousiekte word gekenmerk deur 'n latente periode van 2 tot 6 weke waarna vinnige dood volg[6 7]. Kardiale oneffektiwiteit is een van die belangrikste gevolge na die latente periode. Na die ontstaan van kliniese tekens, is bystandterapie simptomaties en na gelang van omstandighede toegepas, om sodoende vas te stel of gousiekte omkeerbaar is of nie. Daar is van die volgende prosedures gebruik gemaak: 'n verhoging in voorbelading deur dextraninfusie; 'n verhoging in kontraktiliteit deur middel van simpatomimetika[4 5]; 'n verlaging in vullingsdrukke deur middel van vasodilatorterapie[4 9] en diuretika[4].

Met behulp van 'n Swan-Ganz kateter[3 8] is die regterhart via die vena jugularis gekateteriseer om sodoende die pulmonêre arteriële druk (PAD) en die pulmonêre kapillêre wigdruk (PKWD) te bepaal. Met behulp van die termoverdunningsmetode en 'n KO-rekenaar is die kardiale omset van die onderskeie proefdiere bepaal na kateterisering. Vir registrasie van die radiokardiogram (RKG) is 'n bolus van omtrent 0,5 ml ^{99m}Tc-pertechnetaat met 'n aktiwiteit van 1 tot 5 mCi via die proksimale opening van die Swan-Ganz kateter direk in die regteratrium gespuit. Die vloei van die isotoop deur die hart is met behulp van 'n kollimatorsisteem en NaI-kristal gemonitor[11]. Uit die gesamentlike registrasie van 'n EKG en die RKG kon die kardiopulmonêre vloei-indeks (KPVI) bereken word deur die aantal periodes van die harttempo tussen die linker- en regterventrikulêre pieke te tel. Die arteriële druk is met 'n 22G-naald op 'n roetine basis daagliks via 'n arteriële lus van die *arterie carotis* gemeet.

Die mees opvallende hemodinamiese veranderinge tydens gousiekte is 'n toename in ventrikulêre vullingsdruk, 'n afname in slagvolume en 'n toename in die kardiopulmonêre bloedvolume. Die KPVI neem toe en dit geskied in 2 fases, naamlik 'n stadige styging van 7 tot 10, gevolg deur 'n verdere terminale styging voordat die dood intree. Die arteriële drukke toon ook progressiewe afnames met die ontwikkeling van gousiekte.

Vir skape wat met plantmateriaal deur 'n rumenfistula gedoseer is, en wat na 6 weke eers kliniese tekens getoon het, het behandeling 'n vertraging in die ontwikkeling van gousiekte tot gevolg gehad. Die proefdiere kon egter nie gered word nie weens 'n progressiewe afname in kontraktiliteit van die hart. Die belangrikste gevolgtrekking wat uit die studie gemaak kan word, is dat gousiekte as kongestiewe kardiomiopatie onomkeerbaar is. Alhoewel terapeutiese behandeling van gousiekte op hierdie stadium van groter akademiese as praktiese belang is, het hierdie studie ten minste gelei tot die daarstelling van kriteria waarvolgens die diagnose gemaak, en behandeling en moontlike voorkoming daarvan beter bestudeer kon word.

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SALINOMYCIN POISONING IN HORSES

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During 1985 an outbreak of suspected poisoning of racehorses was brought to our attention by a private veterinarian, who submitted specimens to our laboratory for toxicological examination. Four horses had died within *c.* 24 h of the appearance of the first clinical signs, and all 24 horses at the stable were apparently affected to some extent. The clinical signs included mucosal congestion, varying degrees of dyspnoea, stasis of the gut and distention of the bladder. Some of the horses were seen to paw the ground with their forelegs as if they had colic. All had been fed on commercially produced racehorse cubes. Included in the specimens submitted to the laboratory were the leavings from the crib of a fatally poisoned horse, as well as cubes from a drum into which bags of cubes had allegedly been emptied before distribution to the horses. Salinomycin in excess of *c.* 400 ppm was demonstrated in the cubes.

Natural outbreaks of equine monensin poisoning have been reported[1,2,3,4], but no reference to salinomycin poisoning in horses could be found. Experiments were designed to induce acute, subacute and chronic poisoning. The first horse with a cardiopulmonary flow index (CPFI) of 6,5[5,6,7] received 5 mg kg^{-1} salinomycin per stomach tube. Within an hour it started sweating profusely and manifested polypnoea and dyspnoea. After 1 h 25 min, marked cardiac changes were apparent and the horse suffered from tachycardia (130 beats min^{-1}) accompanied by ventricular extrasystoles. The CPFI had risen to 15, indicating serious heart dysfunction. The horse was unsteady on its feet and went into sternal recumbency 1 h 45 min after dosing. Within another 1 h and 30 min it had died. In the terminal stages of intoxication, severe ECG changes were apparent: the T-wave amplitude increased progressively with ST-suppression and T-waves became superimposed on the P-wave 3 min before death. Transient arrhythmia attributed to an AV-block was followed by a rhythmic pulse ending in terminal ventricular fibrillation.

The next horse received 1 mg kg^{-1} salinomycin. It started showing signs of poisoning 1 h after dosing, the clinical signs being polypnoea, dyspnoea, unsteadiness and reluctance to move. The CPFI rose sharply from 7 to 19 (2 h after dosing) then dropped to 6,5 (5,5 h post dosing) only to rise again to 9,5 (48 h post dosing). The horse eventually went down, at first in sternal and then in lateral recumbency. It was not hypersensitive, it could eat and drink and suffered terminally from dyspnoea (forced expiration) and stasis of the gut. Sixty h after dosing it was euthanased.

The third horse received 0,5 mg kg^{-1} salinomycin. Clinical signs became apparent 1,5 h after dosing and included deep abdominal breathing, weakness and restlessness. The CPFI rose from 6 to 20 within 2 h of commencement of the experiment and then gradually dropped to a value of 8 after 10 h. The next morning the horse was apathetic, walked with a slightly high-stepping gait, occasionally knuckled over at the fetlocks, and sometimes stood with a wide-based stance. It was very weak. Later that day it went down in sternal recumbency, but, unlike the first 2 horses, it was able to get up again. The horse was still very stiff in its hind legs on the second d, but had returned to normal by the third d.

It is important to note that a sharp rise in the CPFI was recorded shortly after dosing in all 3 horses. The last horse received only 0,25 mg kg^{-1} salinomycin, with no ill-effect.

The most significant clinical pathological changes were seen in elevated enzyme activities, indicating musculo-skeletal involvement in all but the last horse. Marked increases in the serum activities of aspartate transaminase (AST), creatinine kinase (CK), total lactate dehydrogenase (LD) and LD iso-enzymes 1 and 5 were recorded.

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THE PATHOLOGY OF ENCEPHALOMYOCARDITIS VIRUS INFECTION IN PIGS

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Encephalomyocarditis virus (EMCV) is a cardiovirus (picornaviridae) with a worldwide distribution and very wide host range, which includes rodents, domestic pigs and man. Outbreaks of fatal disease in pigs as a consequence of EMCV infection have been reported from Panama, the USA (Florida), Australia, New Zealand, Cuba and South Africa (Ixopo, 1979 and Greytown, 1980). These outbreaks are usually characterised by sporadic sudden deaths over a period of weeks or months. Pigs in the age range 3-20 weeks represent the vast majority of animals that die of the disease.

Rodents are thought to be the major reservoir of EMCV in nature and episodes of disease in pig herds are often associated with concurrent rodent population explosions. Serological and isolation studies of EMCV in South Africa during 1961 and 1968 showed the virus to be widespread in a number of indigenous rodent species.

Pigs that die of EMCV infection are usually in good condition. Purple discolouration of the skin of the extremities and underline, and cyanosis is commonly seen. Variable quantities of transudate, often containing fibrin strands or clots, are present in the thoracic and abdominal cavities and in the pericardial sac. Oedema of the lungs, mesenterium, omentum, gall-bladder and pancreas is also common. There is variable congestion of the liver, lungs, gastro-intestinal mucosae, spleen and kidneys. In some cases the centrilobular hepatic zones are bright red, giving a striking pattern to the liver architecture. Histologically, varying degrees of centrilobular congestion, degeneration and necrosis are present in the liver.

Characteristic lesions are found in the heart in pigs that die. Large, ill-defined pale brown areas and/or multiple, discrete light brown foci or streaks are observed in the ventricular myocardium. In many cases the lesions are more severe in the right ventricle, especially subepicardially at the base of the heart. Occasionally the large pale areas are flecked with white or the more discrete lesions have a chalky centre. Dilatation of the right side of the heart and mild fibrinous epicarditis are inconsistent findings. "Acute skeletal myodeneration" was noted in one outbreak in New Zealand. Histologically, the myocardium shows focal to diffuse lytic necrosis of fibres with fragmentation and nuclear pycnosis or karyorrhexis. An inflammatory infiltrate, consisting mainly of lymphocytes, lesser numbers of macrophages and a few plasma cells and eosinophils, is always present but of variable severity. Mineralisation of necrotic myocardium is common but not constant.

It has been shown experimentally that, in animals which survive, the necrotic areas in the myocardium are replaced by fibrous tissue which is at first highly vascularised but later condenses to form firm, white scars.

In contrast to rodents, involvement of the central nervous system in the pig appears to be rare and, when observed, is characterised by very mild, diffuse meningoencephalitis.

GOSSIPOL POISONING IN PIGS

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Approximately 80 out of 100 piglets, 2-4 months old, and 2 adult sows died after being fed a ration containing 10% milled, unprocessed cottonseed. Piglets received the ration from 1 to 2 weeks of age and, at weaning, suddenly became depressed and dyspnoeic and eventually died. Most piglets which had been left with the sows survived.

Necropsies on 12 piglets revealed centrilobular hepatic congestion, a mild to moderate hydropericardium, and oedema of the lungs, gall bladder and mesentery. Cardiac lesions included marked ventricular dilatation and a patchy pale discolouration of particularly the ventricles. Microscopical examination of tissues from these piglets revealed multifocal hyaline degeneration and necrosis of heart muscle fibres as well as centrilobular congestion, haemorrhages and necrosis of the livers. The free gossipol levels in 2 of the 4 feed samples (0,018% and 0,019%) exceeded the highest concentration of free gossipol (0,015%) in a ration considered safe for pigs. It was concluded that the excessive free gossipol levels in the feed caused the mortalities. Furthermore, since nearly all fatalities occurred close to weaning, the stress induced by removing piglets from their dams was most probably a major predisposing factor in the precipitation of heart failure.

IONOPHORE TOXICITY IN SHEEP

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Monensin and salinomycin are widely used in the Republic of South Africa (RSA) as growth stimulants for sheep at the recommended rate of 15-22 ppm. It is widely accepted that incorporation into the ration of more than twice the recommended levels of monensin (and presumably also salinomycin) leads to feed refusal in all species including sheep. This is regarded as a built-in safety mechanism against toxicity by ionophores. In the RSA, outbreaks of ionophore toxicity have occured where sheep have continued to ingest feed containing 3 or more times the recommended levels. This tends to refute the supposition of feed refusal at toxic levels. Outbreaks of toxicity in this country have generally been attributed to mixing errors either at the mills or by farmers or labourers.

The following clinical syndromes occur:

1. The usual presenting sign is the sudden death of a high percentage (20-40%) of sheep in the flock.
2. In the majority of outbreaks, the remaining sheep show a progressive ataxia over 3 to 5 d. This commences with a stiff gait, occasional knuckling of the fetlock joints and a disinclination to walk which progresses to dragging of one or more limbs, particularly the hind limbs, followed by sternal recumbency and terminally lateral recumbency. Death inevitably occurs within a few hours of the sheep lying down. A significant number show torticollis and many develop dyspnoea terminally or when exerted. A few sheep may excrete a dark-coloured urine.
3. In some outbreaks, diarrhoea was noted as the passing of black mucoid to watery or sometimes blood-stained faeces, a few d after commencement of ingestion of the incriminated feed.

The 3 clinical syndromes can be ascribed to the pathological involvement of the heart, skeletal musculature and alimentary tract:

1. Heart: In the initial outbreaks of monensin toxicity reported in the RSA in 1981/82, cardiac lesions, although not always discernible grossly, were present microscopically in all cases. These varied from a mild focal necrosis and perivascular lymphocytic cuffing to an extensive necrotizing myocarditis and replacement fibrosis. The epicardial side tended to be more severely affected than the endocardial side. When the lesions were evident grossly, the heart, especially the epicardial side, appeared mottled.

   Signs attributable to acute heart failure, i.e. generalised congestion and oedema (manifested as pulmonary oedema, hydrothorax, hydropericardium, ascites and in some cases moderate anasarca and intermuscular oedema) were present in all cases of ionophore toxicity.

   In cases of salinomycin toxicity, microscopic cardiac lesions were only present in about 50% of cases. When present, the lesions were identical to those resulting from monensin toxicity.
2. Skeletal musculature: Foci of hyaline or lytic necrosis with attendant replacement fibrosis were present in skeletal muscles and involved predominantly the pelvic girdle (55%), muscles of the neck and back (20%) and the pectoral girdle (10%).
3. Alimentary tract: Diffuse congestion of the small intestine with the presence of watery blood-stained contents was evident in about 85% of cases. Numerous small hair-like colonic and caecal erosions were present in 10-15% of cases.

   Some sheep developed a nephrosis which was probably myoglobinuric in nature. Centrilobular hepatic necrosis and pulmonary oedema were often noted.

THE ELECTROCARDIOGRAPHIC DIAGNOSIS OF MYOCARDIAL PATHOLOGY IN PRIVATE PRACTICE

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Cardiomyopathies, inflammation of the myocardium and trauma to the myocardium may occur primarily as specific entitites or secondary to systemic disease or more extensive trauma. This often results in the masking of clinical signs of myocardial damage by those of the systemic condition. Many patients with myocardial disease are, however, clinically asymptomatic. If clinical signs of myocarditis or cardiomyopathies occur, they are often non-specific. The most common clinical sign indicative of possible myocardial involvement, is perhaps the presence of cardiac dysrhythms. In more advanced cases of myocardial disease, clinical evidence of congestive heart failure such as cardiomegaly, pulmonary oedema, pleural effusion and dyspnoea may be seen. Acute death may be associated with myocardial damage.

The diagnosis of myocardial disease is based on the history, clinical signs (non-specific, arrhythmias, signs of the primary disease condition) as well as diagnostic aids such as haematology, enzymology, radiography, electrocardiography and echocardiography. The findings on haematology are non-specific and the use of creatine kinase, alanine transaminase and isoenzymes of creatine kinase and lactate dehydrogenase are essentially restricted to acute myocardial damage. The use of enzymology as well as echocardiography is often not readily available to private practitioners. Radiography only applies once signs of cardiac enlargement and/or cardiac failure appear. Electrocardiography remains an easy non-invasive safe diagnostic aid in the diagnosis of myocardial disease.

The electrocardiographic features of myocardial disease include changes in sinus rhythm, abnormalities in impulse generation and conduction, changes in configuration of the different waves as well as ST-segment and T-wave changes.

Non-penetrating trauma to the chest (myocardium) has been associated with low-voltage complexes, ST-segment depression or elevation, peaked T-waves, premature ventricular contractions and paroxysmal tachycardias.

Repeated cardiac punctures resulted in ventricular conduction delays such as right branch block in experimental dogs.

Ischaemic heart disease in the dog has been associated with ST-segment elevation or depression as well as T-wave changes. In peracute infections with *Babesia canis* in the dog, myocardial pathology (haemorrhages, vasculitis) was observed in association with marked prolongation of the QRS complex merging into a greatly elevated ST-segment coupled with premature ventricular contractions. In other cases, severe bradyarrhythmia with ventricular escape beats as well as multiform premature ventricular contractions and ventricular tachycardia were observed.

Enlargement of the different chambers of the heart has been associated with specific electrocardiographic changes, for example an S1, S2, S3 pattern indicative of right ventricular enlargement. The hypertrophied failing heart is especially vulnerable to the development of a wide spectrum of dysrhythms, secondary to degenerative myocardial foci. Atrial fibrillation is for example often observed in end-stage cases of mitral valve insufficiency.

Myocarditis, regardless of the aetiological agent, is often associated with ventricular premature beats and paroxysmal ventricular tachycardia. Electrocardiographic changes, however, are commonly seen, but are often non-specific and of a transient nature.

The cardiomyopathies are commonly associated with cardiac dysrhythms. Patients with the dilated form (congestive) usually show atrial fibrillation, while complete heart block has been observed in hypertrophic cardiomyopathy.

Secondary cardiomyopathies caused by for example chronic electrolyte disturbances, neoplasia, parasitic infestations and chemicals, may also be associated with electrocardiographic abnormalities.

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THE DIFFERENTIAL DIAGNOSES OF MYOCARDITIS AND MYOCARDIAL NECROSIS IN PUPPIES

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Myocardial necrosis and myocarditis are not common findings in puppies. However, if present, they are significant lesions and a serious attempt should be made to establish the aetiology thereof.

Infectious causes of which viral infections are probably the most common, include the following:

Canine Parvo Virus (CPV): Myocardial involvement in this disease at present is rare, probably due to the altered immune status of this virus amongst the canine population. This is either due to vaccination or natural infection and puppies are now born mostly from immune dams. Macroscopically, CPV infection causes pale blotches or a streaky appearance of the myocardium, and in some cases which survive the acute onslaught, there may be fibrosis and ventricular dilatation. Microscopically, there is a diffuse, acute to subacute, non-purulent interstitial myocarditis. Typical intranuclear viral inclusions are diagnostic. There is a prominent interstitial round cell infiltration, collapse of the sarcolemmal tube, interstitial oedema and fibroplasia in many cases. Calcification of the affected myocardium is notably absent[1]. Electron microscopical studies show typical parvoviral particles in the myocardial nuclei.

Canine Distemper: Myocardial involvement due to canine distemper is rare and age-specific. Infection must take place at an age of 5 to 7 d so that lesions occur when puppies are 16-24 d old[1]. Canine neonates (as in man) show a greater susceptibility to viral myocarditis. Macroscopically the heart may show multiple linear, sometimes confluent, well-demarcated whitish streaks on the epi- and endocardium of both ventricles. Microscopically, there is a multifocal coagulative necrosis of the myocardium, a mild interstitial mononuclear infiltrate, together with the proliferation of elongated spindle-shaped fibroblast-like cells. Calcification of myofibres is outspoken, while occasionally intensely eosinophilic structures resembling canine distemper viral inclusions may occur in the cytoplasm of myocytes[1]. Ultramicroscopically, paramyxovirus nucleocapsids can be demonstrated in the sarcoplasm and infiltrated mononuclear cells, while widespread mineralisation of the mitochondria is also present. The minimal inflammatory cell response seen, is typical for mammalian foetal and neonatal cardiac lesions of metabolic and infectious origin. Furthermore, the immunosuppressive effect of canine distemper virus could also modify the inflammatory response.

Canine Herpes virus: Infection by this virus is also age-specific and puppies must be infected before 2 weeks of age. The incubation period is 3-7 d. Focal areas of necrosis and haemorrhage may occur in many organs, including the myocardium. The inflammatory response associated with these lesions is slight or absent. The presence of basophilic to faintly eosinophilic intranuclear viral inclusions in especially the liver, lungs and kidneys aids, the confirmation of the diagnosis.

African Horsesickness virus and Encephalomyocarditis virus: Although the possibility of infection in a neonatal puppy is remote[2], these 2 viruses are worth considering when a case of a lymphocytic interstitial myocarditis of unknown aetiology is encountered.

Bacteraemia secondary to CPV enteritis associated with myocarditis has been reported[3]. It is speculated that the damage done to the intestinal mucosa by CPV, renders it more permeable to bacteria. The decreased resistance of an immune compromised animal due to CPV infection, will further promote such a phenomenon.

Bacteraemia secondary to wound infection may also lead to a myocarditis of a more purulent nature, but in young puppies the inflammatory response is not dramatic (Van Rensburg IBJ, unpublished data).

Toxoplasma gondii: A multifocal subacute, non-purulent myocarditis is sometimes seen due to *T. gondii* infection. Myocardial necrosis with calcification is present in most cases. The intracellular parasites in association with affected areas are characteristic.

Hepatozoon canis: Although not common in young puppies, hepatozoonosis must be differentiated from toxoplasmosis, since the nature of the lesions and morphology of the parasites can easily be confused.

Encephalitozoon cuniculi: This parasite can be mistaken for *Toxoplasma.* Myocardial involvement with *E. cuniculi* is rare as this organism usually affects the brain and kidneys. However, cases with myocardial involvement are seen and in these the lesions are less severe than in those caused by *Toxoplasma. E. cuniculi* organisms are scarcer, smaller and stain less intensely with H & E than *Toxoplasma* and can be easily differentiated from the latter, as *E. cuniculi* is Gram positive.

Non-infectious causes include the following:

Vitamin E-Se deficiency: The myocardial lesions produced by vitamin E-Se deficiency in puppies are very similar in morphology to those associated with canine distemper i.e. necrosis and prominent calcification[4]. In cases of vitamin E-Se deficiency, the tongue and skeletal muscles are often similarly affected, which is helpful in establishing a diagnosis.

Hypoxic or ischaemic necrosis: Severe anaemia may lead to disseminated myocardial necrosis with infiltration of inflammatory cells and mineralisation as possible features. Myocardial necrosis may also occur secondary to brain lesions, uraemia or potassium deficiency, but is rare in puppies.

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CHEMICAL PATHOLOGICAL DIAGNOSIS OF MYOCARDIAL DISEASE IN MAN

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This brief discussion is divided into two parts:

1. Diagnosis of myocardial necrosis in man.
2. Preliminary findings of research in large domestic animals.

The questions chemical pathologists could answer in human myocardial necrosis are:

1. Is myocardial damage/necrosis present? This question can usually be answered by measuring clinically relevant serum enzymes. These enzymes are: aspartate transaminase (AST), alanine transaminase (ALT), lactate dehydrogenase (LDH) and its isoenzymes (LDH1:LDH2), aldolase (ALD), creatinine phosphokinase (CPK) and its MB-isoenzyme (CPK-MB) and myoglobin. In the case of a myocardial infarction, increases in the serum levels of CPK (total activity), CPK-MB, LDH (total activity), LDH2, ALD, AST and myoglobin are usually unseen. In conjunction with a suggestive clinical history and a typical ECG, these findings are usually considered diagnostic of myocardial necrosis.
2. When did the incident occurr? This question can usually be answered retrospectively if continuous profiles of CPK (total), CPK-MB, LDH (total) and AST determinations are done throughout the clinical course of the conditions. By extrapolation, the exact time of the initial infarct can then usually be obtained. In summary, the factual data which makes this manoeuvre possible, can be summerised as follows:

Enzyme	Starts to rise (h)	Time after infarction peak elevation (h)	Duration of rise (d)
CPK (total)	4 - 8	24 - 48	3 - 5
CPK-MB	1 - 2	12 - 18	1 - 2
AST	6 - 8	24 - 48	4 - 6
LDH	12 - 24	48 - 72	7 - 12

3. What is the extent of the damage? In general, the more severe and extensive the myocardial infarction, the greater the increase in enzyme activity in the serum. This correlation is not absolute since the prognosis for infarct patients does not only depend on the surface area of heart muscle necrosis, but also on the site of infarction in relation to the conduction system and on the general condition of the patient.
4. Can other diseases or conditions cause these or similar changes? Although some conditions could cause the elevation of serum levels of individual enzymes e.g. muscular dystrophies, exercise, hypothyroidism and malignant hyperthermia, very few conditions cause similar changes in the whole cardiac enzyme profile.

The accuracy of the various methods for diagnosing myocardial infarction may be summerised as follows:

ECG + clinical examination	70%
ECG + CPK + LDH + AST + clinical	80%
ECG + CPK isoenzymes + LDH isoenzymes + clinical	90%
ECG + CPK + LDH + AST + CPK isoenzymes + LDH isoenzymes + clinical	100%

Organ distributions and serum levels of CPK (total and isoenzymes), LDH (total and isoenzymes), and AST were studied in cattle. The findings indicate that elevations in LDH2 can be used in the diagnosis of myocardial necrosis in cattle and that both the serum and cardiac isoenzyme profile of the bovine, seem to correlate with that of the human. However, it is unlikely that CPK-MB will be of any use, as the liver (32%) contains more of the isoenzyme than the heart (16%) and, since myocardial necrosis in cattle is often accompanied by hepatic congestion, the results of CPK isoenzyme determinations may be ambiguous in such cases.

It may therefore be profitable to examine the use of other enzymes like aldolase or its isoenzymes for the purpose of additional diagnostic criteria of myocardial necrosis in the bovine species.

Review/Oorsig

ENERGIE- EN PROTEÏENBEHOEFTES VAN HONDE EN KATTE

G.A. SMITH*

ABSTRACT: Smith G.A. **Energy and protein requirement of dogs and cats.** *Journal of the South African Veterinary Association* (1988) **59** No. 2, 109-121 (Afrik). Department of Animal Science, Faculty of Agriculture, University of Pretoria, 0001, Republic of South Africa.

The latest research findings published on the energy and amino acid requirements of dogs and cats are reviewed. Special attention was paid to research results, applicable to working dogs, which have not as yet been incorporated into the recommended requirement standards of the National Research Council.

Key words: energy, protein, dog, cat

Die aantal honde en katte wat as metgesel- en troeteldiere aangehou word, sowel as die getal honde wat in verskillende werksituasies gebruik word, is steeds besig om toe te neem. Die verbintenis tussen die mens en die hond kan teruggevoer word tot 12 000 v.C. terwyl dié met die kat reeds vanaf 7 000 v.C. bestaan[45].

In soverre dit die voeding van hierdie spesies betref, maak McCay[34] die stelling, "There is probably more misinformation regarding the nutrition of the dog than of any other species; As a result, many fine animals have aged prematurely and died early". Kallfelz[27] is van mening dat dié outeur dieselfde gevolgtrekking ten opsigte van katte sou gemaak het indien hy hulle op daardie stadium bestudeer het.

Die afgelope aantal jare het daar egter 'n groter bewustheid by die eienaars oor die welstand van die diere ontstaan. Dit word al meer besef dat die korrekte voeding van die hond of kat 'n belangrike skakel in die totale versorging van die dier is. Dit is spesifiek goeie voedingsprogramme wat daartoe gelei het dat die genetiese potensiaal van sekere diere tot 'n groter mate tot uiting gekom het as in ander. Dié besef van en die behoefte aan gerief, het daartoe gelei dat troeteldiervoedsel 'n hoë prioriteits-item op die inkopelys van die verbruiker geword het. Dit is aangehelp deur 'n aktiewe promosieveldtog wat meegebring het dat die jaarlikse verkope van troeteldiervoedsel etlike miljoene rand per jaar beloop.

Die vervaardiger van troeteldiervoedsel moet aan die behoeftes van twee groepe voldoen:

* Eerstens moet die behoeftes van die dier bevredig word.
* Tweedens moet gepoog word om die behoeftes van die verbruiker, in soverre dit 'n persepsie van waarde en gerief betref, te bevredig. Die waarde word in terme van waarde vir geld, voedingswaarde en smaaklikheid vir die dier, soos waargeneem deur die eienaar, beoordeel.

In hierdie bespreking sal die aandag op die voedingsbehoeftes van die hond en die kat toegespits word, terwyl enige filosofering oor die vereistes van die verbruiker (die mens) tot 'n latere geleentheid sal moet oorstaan.

Die Voeding van Honde

Die massa van volwasse honde mag varieer van minder as 1 kg vir 'n Chihuahua tot soveel as 90 kg vir 'n St. Bernard (Fig. 1).

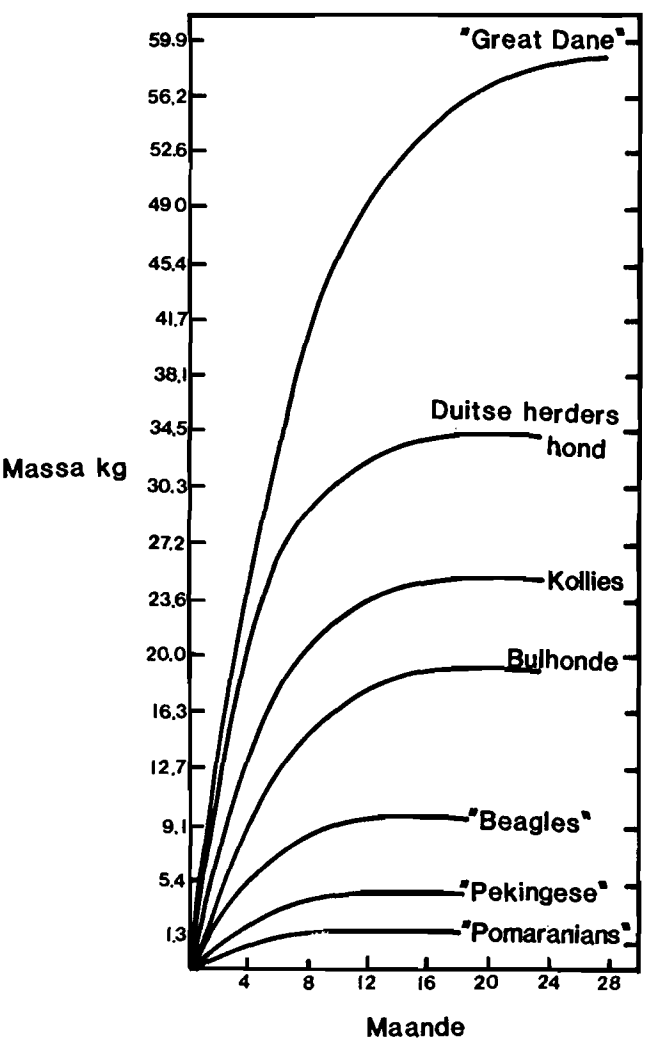

Fig. 1: Groeikurwe vir verskillende honderasse[29]

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Benewens die verskil in massa, kan die volgende klassifikasie van honde vir voedingsdoeleindes gebruik word, nl.

* Volwasse nie-werkende honde
* Volwasse werkende honde
* Teeldiere
  — Dragtige tewe
  — Lakterende tewe
* Groeiende honde

Die voedingsbehoeftes van honde in hierdie verskeidenheid kategorieë verskil en moet derhalwe in berekening gebring word by die formulering van gebalanseerde diëte. 'n Ander aspek wat in berekening gebring moet word, is die verskil in die benutting van nutriënte deur die verskillende honde.

Energiebehoeftes en benutting

Voedsel voorsien nie alleen spesifieke nutriënte nie, maar ook die energie vir die instandhouding van metabolisme en die liggaamstemperatuur. In soverre dit die energiebehoeftes betref, geld die beginsel soos deur Brody et al[7]. bepaal en wat aangepas is deur Kleiber[30]. Die vergelyking $Y = 70.W^{-0.75}$ word derhalwe ook by honde gebruik vir die beraming van die basale metaboliese energiebehoeftes van die volwasse dier. Y is gelyk aan die kilokalorieë per 24 uur, terwyl W die liggaamsmassa in kg is. Die aangepaste waardes vir die metaboliseerbare energiebehoeftes (ME) vir die onderhoud van volwasse honde, sowel as dié vir groeiende, dragtige en lakterende honde word in Tabel 1 weergegee.

Tabel 1: Skynbare metaboliseerbare energiebehoeftes van honde onder verskillende fisiologiese toestande[24]

Fisiologiese toestand	Metaboliseerbare energiebehoeftes (kkal $W^{-0.75}$ dag^{-1})
Klein hondjies (3 - 6 weke)	274
Groeiende honde	200
Volwasse honde	132
Dragtige honde	188
Lakterende honde	470

Hoewel die syfers as riglyne kan dien, verander die energiebehoeftes van die hond met ouderdom, liggaamsmassa en kondisie, aktiwiteit, isolerende eienskappe van die haarkleed, omgewingstoestande en temperatuuraanpasbaarheid. Algemeen gesproke word gevind dat volwasse honde hul kalorie-inname volgens hul energiebehoeftes beheer. Die feit dat die energiebehoeftes van volwasse honde wel verskil, word duidelik in Tabel 2 geïllustreer.

In soverre dit die aanpasbaarheid van 'n hond by omgewingstemperature en die invloed daarvan op die energiebehoeftes van die hond betref, is baie interessante gegewens deur Durrer & Hannon[20] in studies, wat in die arktiese omgewing met Beagles en Alaska-Huskies uitgevoer is, ingesamel. Die werkers het gevind dat die kalorie-inname omgekeerd eweredig met veranderinge in omgewingstemperatuur gevarieer het. In Julie, wanneer die temperatuur ± 17°C was, het die Beagles ongeveer 163 kkal $ME.W^{-0,75}$ dag^{-1} ingeneem, teenoor

Tabel 2: Die gemiddelde energiebehoeftes van normale volwasse honde[24]

Massa van die hond (kg)	Tipe ras	Daaglikse energiebehoeftes (kkal d^{-1})
2	Yorkshire Terrier	240
6	Cairn	540
13	Beagle	900
25	Kollie	1 500
30	Labrador	1 700
35	Duitse Herdershond	1 900
40	Duitse Herdershond	2 100
50*	Great Dane	3 500

* Die reuse rasse benodig bykans 200 kkal $W^{-0.75}$ dag^{-1} vir onderhoud wat 50% meer is as dié van middelmatige grootte honde

die 127 kkal $ME.W^{-0,75}$ dag deur die Huskies. Die Huskies het 'n opmerklike toename in haargroei gedurende November wanneer die temperatuur tot −17°C gedaal het, getoon. Die haarkleed van die Beagles het egter nie noemenswaardig verander nie. Die ME-inname van die Beagles en Huskies was gedurende dié periode onderskeidelik 278 en 205 kkal $W^{-0,75}$ dag^{-1}. Beide die rasse het gepoog om deur met hul neuse en sterte onder hul liggaam opgekrul te lê, hitteverlies tot die minimum te beperk. Terwyl die Huskies geensins gebewe het nie en geweier het om in houtskuilings te slaap, het die Beagles gebewe en die skuilings opgesoek. Hoewel massaverandering klein was, was daar by beide rasse 'n neiging om in die somer swaarder te word. Dit dui daarop dat die inname-aanpassing stadiger as die verandering in kaloriebehoeftes geskied het.

Energiebehoefte van teeldiere

Dit is noodsaaklik dat die reun sowel as die vroulike diere wat vir teeldoeleindes gebruik word, volledig gebalanseerde diëte in voldoende hoeveelhede sal ontvang, om hul veranderende behoeftes te bevredig. Daar moet egter teen vetsug gewaak word, aangesien dit tot laer vrugbaarheid lei. In die geval van vroulike diere mag konsepsie nadelig beïnvloed word, terwyl probleme ook met die geboorte van die kleintjies ondervind mag word.

Energiebehoeftes van dragtige tewe

Tydens dragtigheid is die energiebehoeftes van die teef gemiddeld 20% hoër as tydens onderhoud. Dit is veral tydens die laaste helfte van dragtigheid belangrik om die totale inname van alle essensiële nutriënte by die toename in massa aan te pas. Indien dit nie prakties moontlik is om die voedseltoekenning op massatoename te baseer nie, moet aanbeveel word dat die hoeveelheid voedsel vanaf die vierde tot sesde week van dragtigheid verhoog word. Op daardie stadium verhoog die teef se behoeftes met ongeveer 60%. Dié toename tydens die latere stadia van dragtigheid, het 'n oorhoofse toename vir die dragtigheidsperiode van 20% tot gevolg. As voorbeeld kan gemeld word dat tewe tydens dragtigheid 84 - 95 kkal kg^{-1} dag^{-1} vergeleke met 'n onderhoudsbehoefte van 79 - 92 kkal kg^{-1} dag^{-1} vir jong volwasse honde benodig[35].

Energiebehoeftes tydens laktasie

Die energiebehoeftes verhoog tydens laktasie as gevolg van die sekresie van groot hoeveelhede proteïen, vet en minerale in die melk. Oftedal[44] meld dat die melk van honde relatief gekonsentreerd is en tussen 21-26% droë materiaal, 8-12% vet, 7-10% proteïen en gemiddeld 3,81% suiker en 146 kkal bruto energie per 100 g bevat. Indien in aanmerking geneem word dat 'n Duitse Herdershond 1,7 kg melk dag^{-1} produseer, wanneer piek-laktasie ongeveer 3 weke post partum bereik word, en dat klein hondjies die ekwivalent van 10 tot 14% van hul liggaamsmassa per dag inneem, is dit duidelik waarom energie so 'n belangrike rol in die voeding van die lakterende teef speel.

Ten einde te verseker dat die teef voldoende voedsel inneem, is dit 'n goeie praktyk om die werpsel kort na geboorte te weeg. Dit is bereken dat die klein hondjies ongeveer 220 kkal kg^{-1} liggaamsmassa benodig. Die energie moet as addisionele voedsel aan die teef voorsien word.

'n Normale teef wat 'n werpsel soog, sal teen die tyd dat sy die tweede of derde week van laktasie bereik, 2,5 tot 3 keer haar onderhoudsinname benodig. As voorbeeld kan 'n Labrador teef van 28 kg met 'n werpsel van 8 kleintjies, met 'n totale massa van 12 kg teen 3 tot 4 weke ouderdom voorgehou word. Op dié stadium benodig die klein hondjies ongeveer 200 kkal kg^{-1} dag^{-1} wat deur middel van die melk voorsien moet word. Die teef moet derhalwe 2 400 kkal in die vorm van melk lewer. Die teef se melk bevat ongeveer 1 300 kkal ℓ^{-1} en gevolglik is die hoeveelheid melk wat gelewer moet word dus 1,85 ℓ. In dié geval, word die aanname gemaak dat die energie in die melk 100% doeltreffend deur die klein hondjies benut word. Tydens die produksie van melk is daar egter spesifieke verliese aan energie en kan aanvaar word dat die proses ongeveer 75% doeltreffend is, in welke geval die teef 3 200 kkal vanuit haar voedsel moet verkry om 2 400 kkal in die vorm van melk te lewer. Hierbenewens sal die teef 1 600 kkal vir haar eie onderhoudsbehoeftes benodig. Gevolglik is haar totale behoeftes tydens laktasie 4 800 kkal[21]. Hoewel die berekening op die energiebehoeftes vir bevredigende melkproduksie gebaseer is, word die behoefte aan ander nutriënte op 'n soortgelyke wyse verhoog. Dit is dus noodsaaklik dat die teef voldoende addisionele voedsel van 'n hoë kwaliteit sal ontvang. Dit moet nie net uit koolhidrate en vet bestaan nie, maar indien die hond nie genoegsame hoeveelhede koolhidrate en vette inneem nie, sal proteïen as energiebron gebruik word. Dit is 'n ondoeltreffende en duur gebruik van proteïene, wat verhoed moet word. 'n Voorbeeld van 'n tipiese dieet wat gevolg kan word en die invloed op die massa van die teef word in Fig. 2 uitgebeeld.

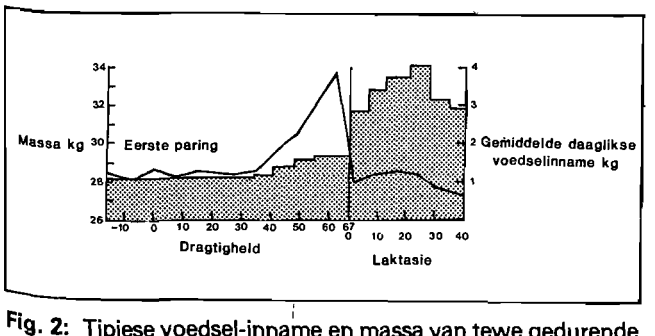

Fig. 2: Tipiese voedsel-inname en massa van tewe gedurende dragtigheid[35]

Energiebehoeftes van groeiende honde

Baie klein hondjies vermeerder hul geboortemassa 60 keer gedurende die eerste jaar. Die vinnigste toename geskied tussen die eerste en sesde maand. Weens dié vinnige groei het die hondjies 'n baie hoër behoefte as volwasse honde. Oor die algemeen word gevind dat groeiende honde 2 of selfs 3 keer die kalorie-inname kg^{-1} liggaamsmassa, vir onderhoud, as volwasse honde vereis. Ondervinding dui daarop dat klein hondjies van die klein sowel as middelmatige grootte rasse van Cairn Terriers tot Labradors, bevredigend sal groei indien hulle diëte ontvang wat 335 kkal kg^{-1} dag^{-1} aan die hondjie voorsien[24]. Groter rasse soos die Great Dane en Newfoundlands benodig egter tot soveel as 400 kkal kg^{-1} dag^{-1} indien hulle bevredigende groei gedurende die eerste 5 of 6 maande wil handhaaf. Vanaf 5 tot 6 maande sal die behoefte vir energie en nutriënte geleidelik afneem, namate die groeitempo afplat, om dan uiteindelik teen 'n jaar of 18 maande ouderdom volwasse onderhoudsvlak te bereik. Dit is naastenby 110 - 120 kkal kg^{-1} dag^{-1} vir klein rasse, 130 -140 kkal kg^{-1} dag^{-1} vir middelmatige en 200 kkal kg^{-1} dag^{-1} vir groot rasse.

Energiebehoeftes van werkende honde

Harde werk lei tot spanning en gevolglik moet die voedingsprogram vir die addisionele behoeftes van die nutriënte wat vir die verhoogde spieraktiwiteit vereis word, sowel as dié om die invloed van spanning te verminder, voorsiening maak. Die nutriënte wat as geredelik beskikbare bronne van energie dien, is vet en koolhidrate. Die voorsiening van die nutriënte was in die verlede die primêre oogmerk in diëte vir honde wat aan drastiese oefening onderwerp is. Studies wat egter met sleehonde tydens wedrenne gedoen is, het daarop gedui dat die proteïenbehoefte hoër is, as wat uit 'n energie-voorsieningsoogpunt verwag word. Die behoefte aan proteïen word geassosieer met die reaksie wat onder toestande van spanning, in die rooibloedseltelling waargeneem is. Spanning onderdruk rooibloedseltelling in honde. Die korrekte verhouding van proteïen, vet en koolhidrate word dus deur die oefening, sowel as die spanning waaraan die hond onderwerp word, bepaal. Die noodsaaklikheid om die rooibloedseltelling in stand te hou, word onderstreep deur die waarneming dat jaghonde se suurstofverbruikstempo 8 keer hoër is as tydens onderhoud, terwyl die energiebehoefte 4 keer hoër is. 'n Maksimum suurstofverbruikstempo van meer as 100 cm kg^{-1} min^{-1} is gerapporteer[50]. Voldoende proteïen moet dus in die dieet ingesluit word om die verlangde rooibloedseltelling te handhaaf.

Goeie resultate is met sleehonde verkry, wat aan langafstand wedrenne deelgeneem het, deur 17% van die beskikbare energie in die dieet, in die vorm van oplosbare koolhidrate te voorsien. Indien 28% van die beskikbare energie in die vorm van hoë kwaliteit proteïen voorsien word, is dit nie voldoende om die rooibloedseltelling, onder toestande van volgehoue harde werk, in stand te hou nie. Dit was egter wel moontlik, indien 32% van die energie in die vorm van proteïen voorsien is[30]. Hoewel 'n hoër proteïenvlak dus wenslik is, moet in gedagte gehou word, dat meer energie benodig word om proteïen, as koolhidrate te metaboliseer. Indien die totale nutriëntvlak dus nie in die werksituasie verhoog word nie, moet

die vet- en koolhidraatvlak, in plaas van die proteïene, verhoog word[29]. 'n Vergelyking tussen 'n onderhoudsdieet en 'n ideale dieet vir honde onder toestande van harde werk en spanning, word in Tabel 3 weergegee.

Tabel 3: 'n Vergelyking tussen 'n dieet wat voldoende is vir onderhoud en 'n ideale dieet vir harde werk en spanning[31]

Energiedigtheid	Onderhoud	Harde werk en spanning	Harde werk en spanning/ onderhoud
Kkal 100 g^{-1} DM	350	530	1,50
Energieverhoudings			
Proteïen %	16	32	2,00
Vet%	14	51	3,60
Koolhidrate %	80	17	0,21
Droë materiaal basis			
Proteïen %	13	42	3,20
Vet %	5	30	6,00
Koolhidraat %	66	22	0,33
Vesel %	10	2	0,22
As %	6	4	0,67
Verteerbaarheid	70	90	1,30

Verskeie kliniese toestande, wat as gevolg van harde werk en spanning na vore tree, kan deur 'n korrekte voedingsprogram verbeter of vermy word. Die toestande sluit die diaree-dehidrasie-spanningsindroom, uitputtingsrhabdomiolise (''tying-up''), opblaas, bloeding in die maag, spanningsanemie en metatarsale frakture in. Die moontlikheid bestaan dat die toestand van hitte-uitputting deur die dieet beïnvloed mag word, 'n aspek wat van kardinale belang in die Suid-Afrikaanse konteks is.

Nutriëntbenutting

Koolhidrate

Die volwasse nie-werkende hond verkry energie uit koolhidrate, vette en proteïene. Hoewel aanvanklike voedingbehoefte-tabelle aangedui het dat koolhidrate noodsaaklik is in hondevoedsel, word dié vereiste nie meer, sedert die 1974 uitgawe van die NRC, as 'n vereiste gestel nie. Die stelling word gemaak dat honde in staat is om normaal te funksioneer op 'n koolhidraatvrye dieet[27].

Koolhidrate speel egter 'n belangrike rol in die voeding van honde. Dit word as proteïenbespaarder, 'n geredelik beskikbare bron van energie en vir die instandhouding van die bloedglukosevlak in die dieet, ingesluit. Die hoeveelheid koolhidrate, wat in die dieet ingesluit word, sal afhang van die ander nutriënte se bydrae tot die totale energiebehoefte. Sewe-en-sestig persent koolhidrate word egter as 'n praktiese maksimum in volledig gebalanseerde diëte beskou.

Burger[8] wys daarop dat die insluiting van 'n hoë persentasie vet in 'n koolhidraatvrye dieet vir dragtige tewe, die oorlewing van die kleintjies betekenisvol verlaag het, in vergelyking met 'n kontrole groep wat 44% van die totale metaboliseerbare energie in die vorm van koolhidrate ontvang het. Die effek word toegeskryf aan 'n ernstige bloedsuikergebrek. Dit moet beklemtoon word dat die koolhidraatbron gekookte stysel was, wat geredelik deur honde, sowel as deur katte benut word.

In soverre dit die rol van koolhidrate in die diëte van lakterende tewe betref, is die situasie deur Romsos et al.[48] toegelig d.m.v. 'n studie met Beagle-tefies. Die tefies is een van 2 diëte gevoer. In Dieet I is 44% van die metaboliseerbare energie vanuit koolhidrate verkry, terwyl Dieet II koolhidraatvry was. Die 2 groepe het egter ekwivalente hoeveelhede energie ontvang. Uit die resultate het die volgende na vore getree.

* Geeneen van die tewe wat Dieet I ontvang het, het enige tekens van 'n bloedsuikergebrek getoon nie.
* Tewe op Dieet II het verlaagde plasma-konsentrasies van alanien en laktaat en verhoogde konsentrasies vry vetsure en hidroksiebutinaat teen die einde van die dragtigheid getoon.
* Die totale aantal kleintjies gebore is nie deur die dieet beïnvloed nie.
* 'n Kleiner getal van Groep II is lewend gebore (63%) teenoor 96% van dié wat Dieet I ontvang het.
* Na 3 dae was slegs 35% van die kleintjies van die tewe wat Dieet II ontvang het lewendig, teenoor 93% van dié op Dieet I.
* Melk van die tewe wat Dieet II ontvang het, het 'n laer persentasie energie vanaf laktose en 'n hoër persentasie vanaf vet verkry as dié wat Dieet I ontvang het.
* Die groei van die kleintjies is egter nie deur die dieet van die moeders beïnvloed nie.
* Die gevolgtrekking word gemaak dat die dragtige tewe koolhidrate in die dieet vereis, ten einde optimale reproduktiewe prestasie te verseker.

Bestudering van die glukose-behoefte gedurende die laaste fase van dragtigheid en die laktasie-periode, dien as verduideliking vir die drastiese invloed wat die lae-koolhidraatdieet uitoefen. Die onderhoud van dragtigheid het 36 kkal ME kg^{-1} vereis teenoor die 113 kkal ME kg^{-1} wat benodig is om laktasie op Dieet I te onderhou.

Hoewel die energie wat benodig word om laktasie te onderhou, 3 keer hoër as die behoefte vir dragtigheid is, word slegs 'n klein persentasie van die energie wat benodig word om laktasie in stand te hou uit glukose verkry. Luick et al.[33] het in studies met lakterende Beagles gevind dat 68-100% van die koolstof vir laktosesintese deur plasmaglukose voorsien word. Die melk van die hond bevat egter 'n lae persentasie laktose. Luick et al.[32] wys daarop dat laktose slegs 8% van die metaboliseerbare energie in die melk van Beagles voorsien, terwyl dit in die geval van die koei en die mens, onderskeidelik 27% en 41% voorsien. 'n Relatief lae glukosevlak word dus benodig. Hierteenoor word 'n aansienlike proporsie van die energie om dragtigheid te onderhou uit glukose, wat die hoof energiebron vir die ontwikkeling van die fetus is, verkry.

Die lae bloedsuikervlak wat by tewe wat 'n lae-koolhidraatdieet ontvang het, waargeneem is, mag die gevolg wees van 'n beperkte kapasiteit om glukose te sintetiseer of as gevolg van 'n gebrek aan voorgangers. Laasgenoemde moontlikheid word ondersteun deur 'n afname in die sirkulerende alanien- en bloedlaktaatvlakke in die tewe wat die lae-koolhidraatdieet ontvang het. Tekens van ketose wat geassosieer word met hipoglukemie en hipo-alanienemie, is by die honde wat die lae-koolhidraat dieet ontvang het, waargeneem, hoewel honde relatief bestand is teen ketose. Koolhidrate lewer

dus beslis 'n positiewe bydrae tot die welstand van honde tydens dragtigheid en laktasie.

Die vraag wat nog beantwoord moet word, is of koolhidrate in die diëte van werkende honde 'n rol speel. Dit is bekend dat verskeie energiebronne vir die sametrekking van spiere gebruik word. Hierdie bronne word op 'n ge-orkestreerde wyse benut. Die onmiddellike bron is adenosien-trifosfaat (ATP), wat twee hoë-energie fosfaatbindings het. Elke spiervesel bevat genoeg ATP om vir minder as 1 s saam te trek. Dit word onmiddellik deur genoeg hoë-energie fosfate vanaf kreatinienfosfaat, genoeg vir 'n verdere aantal sametrekkings, aangevul. Hierdie fosfaatverbindings sal die energie vir vinnig hardloop deur honde, vir minder as 10 s onderhou.

Hoë-energie fosfaatverbindings word in die spiervesels deur die afbraak van glikogeen, 'n koolhidraat wat in die selle gestoor word, en glukose wat deur die selle uit die bloed opgeneem word, gegenereer. Die glikolise proses is anaerobies en is minder effektief as aerobiese metabolisme of oksidasie.

Benewens glukose is die ander hoof energiebron in die bloed, vetsure, wat verkry word deur die mobilisering van die vetdepots. Vetsuur-metabolisme in die spierselle is volkome aerobies. In honde wat teen die helfte van hul maksimum spoed hardloop, sal die verhoogde bloedvloei meer as genoeg suurstof, glukose en vry-vetsure na die werkende spiere dra. Die aantal mitokondria verhoog slegs met oefening van die spiere wat werk. Die suurstofverbruik en relatiewe bydrae van glukose en vetsure word in Tabel 4 weergegee.

Tabel 4: Suurstofverbruik en relatiewe bydrae van glukose en vetsure vir honde, wat vir 4 uur teen 6,4 km h^{-1} teen 'n helling van 15° op 'n trapmeul hardloop[31]

Duur van die oefening, h	0	1	4
Suurstofverbruik cm^3 min^{-1}	85	500	500
Persentasie suurstofverbruik waarvoor rekenskap gegee word deur die oksidasie van:			
Glukose	16	11	11
Vetsure	30	49	71
Persentasie stuurstofverbruik — geen rekenskap voor	54	40	18

In gesonde werkende spiere bestaan daar 'n fynbeheerde balans tussen aerobiese en anaerobiese prosesse, tussen die benutting van intra- en ekstra-spier-energiebronne en tussen koolhidrate en vetbenutting. Kortstondige intense werk vereis dus glikolise. 'n Windhond wat byvoorbeeld vir 30 s teen 61,1 km h^{-1} hardloop, vul al die ATP aan deur anaerobiese afbraak van die spierglikogeen. Langer maar ligter werk is aangewese op die oksidasie van glukose en vetsure wat deur die bloed na die spiere gedra word. Huskies wat vir 55 min teen 30,9 km h^{-1} hardloop vul ATP deur die oksidasie van vet aan en gebruik 15,32 kkal min^{-1} of 838 kkal vir die wedren.

Glukose is dus nie die hoofbron van energie in die spiere nie, maar voorsien vir slegs 16% van die oksidasie in die rustende toestand en vir selfs minder tydens hardloop teen 'n lae spoed. Tydens werk, verhoog suurstofverbruik 6 keer, maar glukose oksidasie slegs 4 keer. Daarteenoor neem die oksidasie van vry-vetsure 13 keer toe, sodat die fraksie onder langdurige werk uiteindelik vir 71% van die suurstofverbruik aanspreeklik is.

Selfs meer fassinerend is die suurstofverbruik waarvoor daar nie in terme van die 2 komponente (glukose en vetsure) rekenskap gegee kan word nie. 'n Gedeelte van die fraksie verteenwoordig die oksidasie van ketoonliggaampies, veral wanneer die oefening uitgerek is. Volgens Paul & Issekutz[46] is daar egter nog 54% suurstof waarvoor daar nie tydens rus rekenskap gegee kan word nie. Die vraag is of karnivore meer aminosure metaboliseer as wat algemeen gereken word. Koolhidrate speel dus wel 'n rol in die voeding van die werkende hond. Dit is egter onbekend of 'n bepaalde hoë vlak van koolhidrate in die dieet proteïenbesparend sal kan optree in 'n werksituasie.

Vette

Benewens die belangrike rol wat vette in die dieet van werkende honde speel, moet dit nie uit die oog verloor word dat vet 'n essensiële nutriënt in die diëte van honde is nie. Die NRC aanbeveling is 5% vet, hoewel die werklike vlak mag varieer in ooreenstemming met die essensiële vetsuursamestelling. 'n Linoleïensuurpeil van 1% op 'n droë basis is 'n vereiste. Indien 2% van die ME as linoleïensuur voorsien word, word alle tekortsimptome ook by klein hondjies waargeneem. Honde het egter 'n wye toleransie vir vet en konsentrasies van 5% tot 20% (op 'n lugdroë basis) word aanbeveel om die vereiste linoleïensuur-peil te voorsien. Honde het egter al vir periodes van 2 jaar, bevredigend op diëte presteer met 'n vetinhoud van 40%. Wanneer hoë konsentrasies vet ingesluit word is dit noodsaaklik dat 'n anti-oksidant bygevoeg word ten einde ransigheid en die gevolglike vernietiging van vitamiene E en A te verhoed.

Die mees waarneembare tekortsimptome in diëte met 'n lae vetinhoud, is die ontwikkeling van 'n abnormale vel en haarkleed asook 'n verhoogde voorkoms van velinfeksies. 'n Volledige opheffing van die toestand word egter nie verkry indien die vlak van dierevet nie minstens 15% van die totale kalorieë voorsien nie. In dié opsig is varkvet, met 'n linoleïensuur-inhoud van 12%, baie meer effektief as beesvet wat slegs 2% linoleïensuur bevat.

Hoewel beide linoleïensuur en aragidoniensuur as poli-onversadigde vetsure geklassifiseer word en effektief in die opheffing van die vet-tekortsindroom is, is linoleïensuur die essensiële vetsuur waaruit aragidoniensuur gesintetiseer word. Linoleïensuur speel ook 'n rol by die instandhouding van 'n gesonde haarkleed, maar sal nie alleen 'n tekort-toestand ophef nie.

Proteïen

Mellentin[35] is van mening dat 'n minimum van 12% van die kalorieë wat daagliks deur 'n hond benodig word, deur proteïen voorsien behoort te word. Die meeste diëte bevat egter proteïen van plantaardige sowel as dierlike oorsprong waarvan sommige 'n lae biologiese waarde het. Praktiese diëte voorsien dus gewoonlik voldoende proteïene om 20-25% van die dieet se kalorieë te verskaf. Indien vet nie meer as 5% per gewig van die droë materiaal voorsien nie, sal proteïenpeile van 22% tot 25% ongeveer 25% van die totale kalorieë in die dieet lewer. Indien hoër vlakke vet ingesluit word (10-15%), sal meer proteïen, waarskynlik soveel as 30% benodig word.

Tydens dragtigheid is dit 'n goeie beleid om proteïene en totale nutriënte wat 25% bo die onderhoudsbehoefte is, te handhaaf. Die vlak behoort tot 100% bo onderhoud verhoog te word vir ten minste die eerste maand na parturisie. Indien in gedagte gehou word dat tussen 0,25% en 0,33% van die stikstof wat die lakterende teef inneem, in die vorm van melk uitgeskei word, is dit vanselfsprekend dat 'n hoë kwaliteit proteïen aan die teef voorsien moet word.

Tydens groei is die behoefte aan dieet-proteïen, sowel as die totale kalorieë hoog. Dit word deur die gegewens in Tabel 5 gereflekteer.

Tabel 5: Aanbevole nutriëntpeile vir honde[35]

Nutriënt		Volwasse onderhoud	Groeiende hondjies
Proteïen	g	4,8	9,6
Vet	g	1,1	2,2
Linoleïensuur	g	0,22	0,44
Minerale			
Kalsium	mg	242	484
Fosfor	mg	198	396
Kalium	mg	132	264
Natriumchloried	mg	242	484
Magnesium	mg	8,8	17,6
Yster	mg	1,32	2,64
Koper	mg	0,16	0,32
Magnesium	mg	0,11	0,22
Sink	mg	1,1	2,2
Jodium	mg	0,034	0,068
Selenium	µg	2,42	4,84
Vitamiene			
Vitamien A	IU	110	220
Vitamien D	IU	11	22
Vitamien E	IU	1,1	2,2
Thiamien	µg	22	44
Riboflavien	µg	48	96
Pantoteensuur	µg	220	440
Niasien	µg	250	500
Piridoksien	µg	22	44
Foliensuur	µg	4	8
Biotien	µg	2,2	4,4
Vitamien B12	µg	0,5	1
Cholien	mg	26	52

Volgens die gegewens in Tabel 5, word aanbeveel dat 9,6 g proteïen kg^{-1} liggaamsmassa dag^{-1} aan groeiende honde voorsien word. Die NRC beveel aan dat 'n dieet wat 22% proteïen (op 'n droë basis) bevat, met 'n proteïen-kwaliteit ekwivalent aan kaseïen, vir jong honde gebruik word. Die vraag moet egter gestel word of die peil wel optimaal is. Burns et al.[11] vind dat die groei van 8 tot 10-week-oue hondjies verhoog is, deur die proteïenpeil van die dieet te verhoog van 0% tot 15%. 'n Verhoging van 15% tot 20% het egter nie 'n betekenisvolle verbetering in die groei teweeg gebring nie. Voerinname per se is nie deur 'n toename in die proteïenpeil van die dieet bo 7,5% verhoog nie. Die doeltreffendheid van voerverbruik het egter progressief toegeneem tot op 'n vlak van 15% proteïen (Tabel 6).

Die groei van 13 tot 17-week-oue honde is nie betekenisvol verbeter deur die dieet-proteïenpeil bo 10% te verhoog nie. 'n Analise van die gebroke responslyn vir massatoename, wat volgens Robbins et al.[47] uitgevoer is, toon dat die minimum proteïenbehoefte, vir optimale groei van die honde 11,3% is. Die minimum dieet-proteïen vir optimale proteïen-doeltreffendheid, blyk 11,7% te wees. Die invloed van dieetproteïenpeil op die groei, dieetinname en doeltreffendheid van voedselbenutting word in Tabel 7 aangestip.

Die proteïenbehoefte van die 8 tot 10-week-oue honde is op grond van hierdie studie beraam om 14,0 gm proteïen dag^{-1} kg^{-1} te wees (15,0% dieet-proteïen), terwyl die proteïenbehoefte vir die 13 tot 17-week-oue honde 9,3 g proteïen dag^{-1} kg^{-1} blyk te wees (11,7% dieetproteïen).

Proteïenkwaliteit

Die voedingswaarde van proteïen is primêr van die proteïen se verteerbaarheid en die aminosuurinhoud daarvan, relatief tot die dier wat vir die biotoets gebruik word, afhanklik. Die omvang van proteïenvertering en die behoefte vir aminosure varieer tussen spesies en met ouderdom. Verskille in die proteïenbenutting kan byvoorbeeld verwag word tussen honde en rotte, aangesien honde 'n laer lisien-, swawelbevattende aminosuur- en histidienbehoefte as rotte het. Beperkte inligting is egter oor die verteerbaarheid van dieet-proteïen in honde beskikbaar. Uit werk van Hegstedt & Chang[23] blyk dit dat die verteerbaarheid van proteïene, wat volledig uit plantaardige produkte saamgestel is, ongeveer 80% was. In die studie deur Burns et al.[11] is die skynbare verteerbaarheid in 2 groepe honde wat in ouderdom verskil het, vergelyk. Die skynbare proteïen-verteerbaarheid van die groepe wat onderskeidelik 8 - 10 weke en 13 - 17 weke oud was, was egter vergelykbaar. Die waarde was egter betekenisvol laer as die verteerbaarheidswaarde wat met rotte verkry is. Die skynbare verteerbaarheid is egter wel deur die proteïenpeil van die dieet beïnvloed. Dit was egter nie die geval met die ware proteïenverteerbaarheidswaarde van laktalbumien, kaseïen en soja-isolaat nie. Die verskille in skynbare verteerbaarheid moet dus toegeskryf word

Tabel 6: Die invloed van proteïenpeil op die groei, dieet-inname en doeltreffendheid van voerverbruik (8 - 10 weke ouderdom)[11]

Parameters	Dieet-proteïenpeil (%)						
	0,0	5,0	7,5	10,0	12,5	15,0	20,0
Massatoename $g\ dag^{-1}$	−19,5	7,4	42,4	62,4	66,6	81,6	105,2
Dieet-inname $g\ dag^{-1}$	90	120	170	188	184	189	196
Doeltreffendheid van voerverbruik	−0,222	0,036	0,248	0,314	0,351	0,433	0,538

Tabel 7: Die invloed van dieet-proteïenpeil op die inname, groei en doeltreffendheid van voerverbruik van groeiende honde (13 - 17 weke ouderdom)[11]

Dieet-proteïen (%)	5,0	7,5	10,0	15,0	20,0
Groei (g dag^{-1})	29,5	52,6	80,8	95,1	91,4
Dieetinname (g dag^{-1})	211	210	240	266	224
Doeltreffendheid van voerverbruik	0,129	0,235	0,342	0,418	0,414

aan die rol wat endogene fekale stikstofuitskeiding in die spesies speel. Die vraag is egter wat is die waarde vir die fraksie en of dit verskil tussen honde met verskillende liggaamsmassas. Kendall et al.[28] het 'n studie onderneem om die totale endogene stikstofuitskeiding van honde te kwantifiseer. Die fekale en urinêre stikstofuitskeiding is tydens 'n 14-d metaboliese periode met honde, waarvan die liggaamsmassa van 2,0 kg tot 51,0 kg gewissel het, gemeet. 'n Stikstofvrye dieet is gedurende hierdie periode aan die honde voorsien. Die gemiddelde (±SA) endogene urinêre stikstof, metaboliese fekale stikstof en totale endogene stikstof uitset, was onderskeidelik 210 ± 9; 63 ± 3 en 273 ± 9 mg kg^{-1} dag^{-1}. Geen betekenisvolle verskille, as gevolg van geslag of liggaamsmassa, is ten opsigte van die parameters waargeneem nie. Die endogene urinêre stikstofuitskeiding was egter betekenisvol ($p < 0,02$) positief ($r = 0,5$) met die verlies in liggaamsmassa gedurende die metaboliese periode gekorreleer. Die patroon van urinêre stikstofuitskeiding van die honde, op die proteïenvrye-dieet, word in Fig. 3 uitgebeeld.

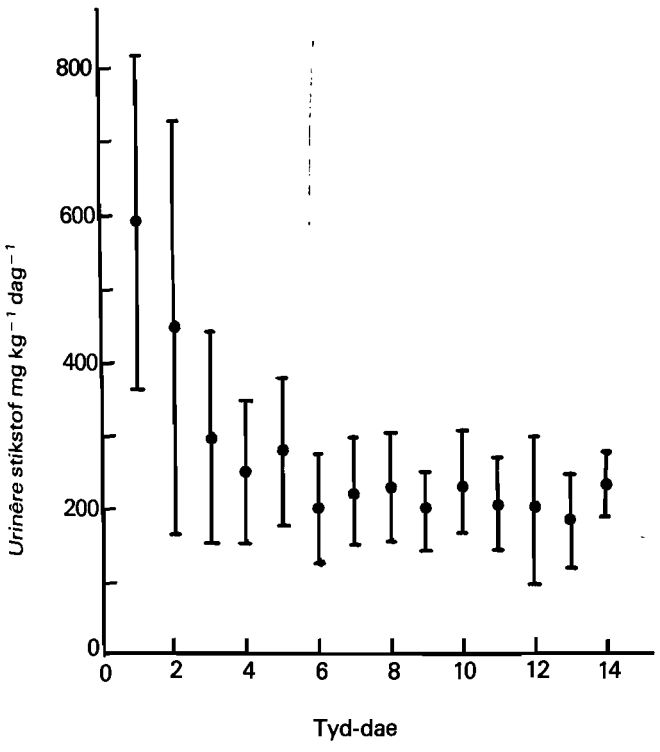

Fig. 3: Urinêre stikstofpatroon van honde op 'n proteïenvrye dieet[28]

Die minimum hoeveelheid metaboliseerbare proteïen (N x 6,25) wat vereis word, om die totale endogene stikstofverlies van 273 mg kg^{-1} aan te vul, blyk 1,7 g kg^{-1} te wees. Dit is egter reeds onomwonde met mense bewys, dat sulke lae vlakke van proteïeninname, onvoldoende is om 'n aanvaarbare interne stikstofbalans te handhaaf, selfs wanneer dit in die vorm van heeleierproteïen voorsien word[22]. Dit is dus waarskynlik dat 'n minimum proteïenvlak vir die onderhoud van volwasse honde ietwat hoër as die 1,7 g kg^{-1} dag^{-1} sal moet wees[28].

Aminosuurbehoeftes van volwasse honde

As monogastriese dier, is die hond afhanklik van proteïen met 'n spesifieke aminosuur-samestelling, wat aan sy behoeftes sal voldoen. 'n Dieet vir optimale produksie kan dus slegs saamgestel word indien die behoeftes aan die onderskeie aminosure bekend is.

'n Aanbeveling ten opsigte van sekere aminosure se behoeftes is deur Burger et al.[8] vir honde gemaak. Volgens die gegewens in Tabel 8 het 'n volwasse hond nie arginien in die dieet nodig nie. Die bewering is egter in stryd met die resultate van Burns et al.[14] wat bewys gelewer het dat arginien wel essensieël vir die volwasse hond is. Die gevolgtrekking is gebaseer op die bepaling van metaboliete in die bloed en nie slegs op verandering in

Tabel 8: Aanbevole aminosuurpeile vir honde[8]

Aminosuur	Volwasse honde	Groeiende honde
Treonien	264	600
Valien	325	450
Sistien[(a)]	88	350
Metionien	180	450
Sistien plus metionien[(a)]	268	800
Isoleusien	302	800
Leusien	544	600
Tirosien[(a)]	211	
Fenielalanien	304	
Tirosien en fenielalanien[(a)]	515	1 000
Histidien	132	130
Triptofaan	79	150
Lisien	368	650
Arginien	NB[(b)]	1 000

\+ Alle waardes is uitgedruk as mg 400^{-1} kkal ME wat ongeveer ekwivalent is aan mg 100^{-1} g droë materiaal in kommersiële diëte.

(a) Die waardes vir sistien en tirosien reflekteer die mate waartoe hierdie aminosure respektiewelik kan substitueer vir die totale behoefte vir metionien en fenielalanien.

(b) NB — nie benodig

Tabel 9: Invloed van arginientekort op die plasma en urinêre metaboliete in volwasse honde wat 2 keer per dag geforseerde voeding ontvang het[14]

	Kontrole	Arginien
Liggaamsmassa-verandering kg 4 dae^{-1}	0,48 ± 0,10	−0,28 ± 0,05
Plasmametaboliete		
NH_4 - N, μg cm[13]	1,0 ± 0,3	2,6 ± 0,5
Ureum, mg 100cm^{-3}	52 ± 3	40 ± 2
Orotaat, μg cm^{-3}	0,7 ± 0,2	4,3 ± 0,8
Urinêre metaboliete		
NH_4 — N, g dag^{-1}	2,7 ± 0,5	1,7 ± 0,2
Ureum, g dag^{-1}	17,5 ± 1,3	12,7 ± 0,8
Orotaat, mg dag^{-1}	1,1 ± 0,2	45,0 ± 5,0
Sitraat, mg dag^{-1}	40 ±12	115 ±12
Totale urinêre N, g dag^{-1}	16,0 ± 0,8[a]	15,9 ± 1,3[a]
Totale fekale N, g dag^{-1}	0,4 ± 0,3[a]	0,4 ± 0,2[a]
Stikstofbalans, g dag^{-1}	0,2 ± 0,8[a]	0,3 ± 0,1[a]

Getalle gemerk[a] verskil nie betekenisvol nie

liggaamsmassa nie. Die invloed van arginientekort word in Tabel 9 geïllustreer.

Die weglaat van arginien uit 'n dieet van volwasse honde het tot 'n betekenisvolle verlaging in liggaamsmassa, episodes van drastiese vomering, spierbewerasie en skuim om die mond van die honde gelei. Die kliniese tekens wat by die honde waargeneem is, het met dié by groeiende honde en katte wat arginientekort-diëte ontvang het, ooreengestem. Geen ander aminosuurtekort het tot die kliniese tekens aanleiding gegee nie. Historiese kliniese tekens mag die gevolg wees van 'n induksie van hiperammonemie. Die arginientekort het dus tot 'n betekenivolle toename in die plasma-ammoniak en 'n afname in die urinêre-ammoniakvlak gelei. Hierbenewens is 'n verhoging in die orotaat- en sitraatvlakke in die urine waargeneem. Ten spyte van die feit dat arginien vir optimale groei en stikstofbalans vereis word, is geen betekenisvolle verandering in die stikstofbalans waargeneem nie. Die bevinding is in ooreenstemming met die resultate wat deur Nakagawa et al.[40] met kinders verkry is.

Die uitskeiding van orotiensuur blyk 'n waardevolle metaboliet te wees om die arginienstatus van ureotiliese soogdiere te bepaal. Die metaboliet is van besondere waarde in volwasse diere aangesien die uitskeiding daarvan, tot 'n groter mate as massaverandering of stikstofbalans, deur 'n arginientekort beïnvloed word.

Die gemelde studie dui daarop dat 0,28% arginien in die dieet 'n betekenisvolle verandering in die normale intermediêre metabolisme in die volwasse hond verhoed (Tabel 10).

Histidien

In 'n studie waarin die behoefte van honde ten opsigte van histidien bestudeer is, is gevind dat honde 'n histidienvrye dieet vir 40 d tot 60 d kan verdra, alvorens enige tekortsimptome na vore tree. Die rede vir die relatief stadige aanvang van die manifestasie van die histidientekortsindroom, kan aan verskeie faktore wat ter behoud van die histidienpoel inwerk, toegeskryf word. Dit sluit in die vrystelling van histidien vanuit karnosien, 'n afname in die sintese van hemoglobien, 'n daling in die tempo van histidien-degradasie, 'n verlaging in die ekskresie van histidien in die urine en die moontlike produksie van histidien deur die niere. Verskeie van die faktore blyk waargeneem te word in honde wat onderwerp word aan 'n histidientekort. Sodra tekens van 'n histidientekort voorkom, mag dit egter dikwels baie vinnig in intensiteit toeneem. Dié tekort lei tot 'n daling in die plasma- en spierhistidien, spierkarnosien, liggaamsmassa, hematokrit en serumalbumienvlakke. Oor die langtermyn, blyk dit dat die plasma-sinkvlak daal en 'n laer sink- en kopervlak word in die heelbloed van honde, met 'n histidientekort,

Tabel 10: Verandering in liggaamsmassa, dieet-inname, urinêre-ammoniak, ureum, orotaat en sitraat in volwasse honde op aminosuurdiëte met verskillende konsentrasies arginien[12]

	Dieët-arginien (%)			
	0,000	0,28	0,56	1,12
Liggaamsmassa-verandering kg 5 dae^{-1}	−1,13[a]	0,40[b]	0,23[b]	0,10[ab]
Dieet-inname, kg droë dieet 5 dae^{-1}	1,89[a]	3,21[b]	3,14[b]	3,29[b]
Urinêre-metaboliete op dag 5				
NH_4 N g dag^{-1}	0,35[a]	1,32[b]	1,47[b]	1,57[b]
Ureum g dag^{-1}	8,1[a]	10,4[a]	12,6[a]	10,2[a]
Orotaat mg dag^{-1}	38,6[a]	5,8[b]	0,5[b]	0,7[b]

Getalle met dieselfde boskrifte verskil nie betekenisvol nie

Tabel 11: Die invloed van 'n gedeeltelike of totale verwydering van metionien in die dieet op die dieet-inname, liggaamsmassa, doeltreffendheid van voerverbruik en stikstofbalans van groeiende honde.[37]

Behandeling	Dieet-inname g dag^{-1}	Massa-verandering g 14 dae^{-1}	Doeltreffendheid van voerverbruik	Stikstofbalans g N 7 dae^{-1}
Kontrole (0,82 % metionien)	234^{b}	577^{c}	0,45^{a}	7,22^{c}
50% metionien (0,41% metionien)	239^{b}	410$^{b\,c}$	0,42^{a}	7,16^{c}
0% metionien	65^{a}	−474^{a}	−0,67	−1,33^{a}

Getalle met dieselfde boskrifte verskil nie betekenisvol nie

waargeneem. Dié waarneming ondersteun die moontlikheid dat daar 'n metaboliese interverwantskap tussen histidien, sink en koper bestaan[15]. Ten einde die voorkoms van die kliniese tekens te verhoed, blyk 'n vlak van 0,33% histidien in die dieet voldoende te wees, hoewel die peil nie as 'n vereiste gekwantifiseer is nie.

Aandag is spesifiek aan die aminosure geskenk, aangesien arginien voorheen nie as essensieel vir die volwasse hond beskou is nie, terwyl die respons op 'n histidien-tekort relatief lank neem voordat dit waargeneem kan word.

Aminosuurbehoeftes van die groeiende hond

Dit is logies dat die groeiende hond se aminosuurbehoeftes hoër as dié van die volwasse hond sal wees. In studies deur Milner[36] en Milner et al.[37] is bewys gelewer dat leusien, lisien, valien, fenielalanien, arginien, metionien, treonien, triptofaan, histidien en isoleusien essensiële aminosure was wat in die dieet van groeiende honde voorsien moes word. 'n Tekort aan enigeen van hierdie aminosure, lei tot 'n betekenisvolle afname in die voerinname, groei en stikstofbalans van die onvolwasse hond. Die spesifieke behoeftes vir hierdie aminosure moes egter nog gekwantifiseer word.

Gebaseer op die groei- en stikstofbalans-gegewens van hierdie studie (Tabel 11), blyk dit dat groeiende honde 0,41% of minder metionien benodig indien die sistien peil op 0,35% van die dieet gehandhaaf word. Daar was egter 'n betekenisvolle verhoging in die plasma en urinêre vlakke wat weens 'n verhoogde aminosuurkatabolisme, geassosieer word met 'n aminosuurwanbalans. Hiervolgens blyk dit dat die totale swawelbevattende aminosuurvlak van 0,76% nie die onderhoudsbehoeftes en normale intermediêre metabolisme in die groeiende hond bevredig nie. Indien die dieet 0,82% metionien bevat, word sistien nie benodig nie. Burns & Milner[12] vind in opvolgende studies dat optimale groei, doeltreffendheid van voerverbruik en stikstofretensie in honde verkry is, wat 0,60% L-sistien en 0,20% metionien ontvang het. Opvolgende studies wys dat die totale swawelbevattende aminosuurbehoefte 0,39% was. Die waarde is egter baie laag.

'n Baie interessante waarneming wat in die studie gemaak is, is dat honde in staat is om L-metionien, D-metionien, DL-metionien, N-asetiel-L-metionien en OH-metionien doeltreffend te benut. Die hond kan egter nie N-asetiel-D-metionien benut nie. In die vervaardiging van hondekosse kan L-metionien dus doeltreffend met die gemelde komponente vervang word. Dit kan ekonomies voordelig wees, of soos in die geval van N-asetiel-L-metionien, kan dit die vorming van onwenslike reuke en smake, wat as gevolg van die degradering van DL-metionien tydens prosessering vorm, verhoed[4].

'n Belangrike aspek wat egter nie tot dusver in berekening gebring is nie, is die energiekonsentrasie van die dieet. Blaza et al.[5] bring die saak egter in 'n studie met Labradors en Beagles in perspektief. Dit blyk dat die honde wat 0,39% metionien en 0,15% sistien ontvang het, 'n betekenisvol laer liggaamsmassa, stikstofretensie, voedselinname en doeltreffendheid van voerverbruik gehad het as die honde wat 0,57% en 0,74% metionien in die teenwoordigheid van 0,15% sistien ontvang het. Die diëte het 468 kkal ME 100 g^{-1} voorsien, wat meebring dat 'n vlak van 116 mg TSAA 100 kkal ME^{-1} nie voldoende vir groei is nie. Die laagste vlak, wat voldoende was vir die groeiende hond, was die 0,57% metionienpeil. 'n Baie interessante bevinding wat in 'n aanvullende studie deur die werkers waargeneem is, was dat waar 117 mg TSAA 100 kkal ME^{-1} vanaf 'n vry-aminosuur dieet verkry is, dit onvoldoende was vir Labradors, maar voldoende blyk te wees vir sommige Beagles. Die bevinding werp meer lig op moontlike rasverskille, sowel as die verskil tussen intakte en vry-aminosuurbehoeftes.

'n Belangrike verskil tussen die resultate wat in dié studie en voriges verkry is, is die verskil in die periode waaroor die studie uitgevoer is. Die studie het oor 'n periode van 5 weke gestrek, terwyl die ander slegs 2 weke geduur het. Dit mag die verskil in behoeftes ten opsigte van metionien verklaar.

Fenielalanien en tirosien

Fenielalanien is 'n essensiële aminosuur vir die groeiende hond. In die werk van Milner[36] blyk dit dat die fenielalanienbehoefte laer as 0,58% is, indien 0,35% tirosien in die dieet teenwoordig is. Milner et al[38]. het met breekpunt-analises aangedui dat die minimum persentasie dieet-fenielalanien vir groei, doeltreffendheid van voerverbruik en stikstofbalans onderskeidelik 0,80%, 0,64% en 0,80% is. In breekpunt-analises vir tirosien is gevind dat 0,28% tirosien optimaal is, indien die dieet 'n totale aromatiese aminosuurinhoud van 0,6% gehad het. Die gevolgtrekking wat uit die studie gemaak word, is dat 0,8% beskikbare fenielalanien as die vereiste vir die aromatiese aminosure gestel kan word. Dit moet egter in gedagte gehou word dat geslag, ouderdom en fisiologiese status die behoefte vir die aminosure mag beïnvloed.

Triptofaan

Die triptofaanbehoefte van die onvolwasse hond is bevredig deur die voorsiening van 145 mg kg^{-1} dag^{-1} of deur 0,17% triptofaan in die dieet te voorsien. Czarnecki & Baker[17] rapporteer dat die triptofaanbehoefte vir optimale groei van 6 tot 10-week-oue Engelse Pointers 0,16% was, en 0,12% vir 12 tot

Tabel 12: Vereiste aminosuurkonsentrasies in die diëte van groeiende honde[43]

Aminosuur	Per 1 000 kkal ME g	Droë Basis 3,67 kkal ME g^{-1} %
Arginien	1,37	0,50
Histidien	0,49	0,18
Isoleusien	0,98	0,36
Leusien	1,59	0,58
Lisien	1,40	0,51
Metionien-sistien	1,06	0,39
Fenielalanien-tirosien	1,95	0,72
Treonien	1,27	0,47
Triptofaan	0,41	0,15
Valien	1,05	0,35
Nie-essensiële aminosure	17,07	6,26

14-week-oue hondjies is. Die studies het ook bewys gelewer dat die D-triptofaan benutting 36 ± 6% (SA) van L-triptofaan is. Gebaseer op hierdie resultate is die behoefte vir triptofaan op 82 mg kg^{-1} dag^{-1} of 0,41 g 1 000 kkal ME^{-1} dieetenergie vasgestel.

Die jongste aminosuurbehoeftes[13] van die NRC word in Tabel 12 saamgevat. Hieruit is dit duidelik dat al die jongste gegewens oor aminosuurbehoeftes nog nie daarin opgeneem is nie.

Verdere studies word nog benodig om die aminosuurbehoefte vir honde van verskillende ouderdomme en geslagte te bepaal. 'n Aspek waaroor daar egter nog geen inligting bestaan nie, is die aminosuurbehoeftes van honde wat aan harde werk en spanning onderwerp word. In al hierdie studies sal die moontlike antagonisme wat tussen bepaalde aminosure mag bestaan, egter nie uit die oog verloor word nie. Czarnecki et al.[18] het in 'n studie met Engelse Pointers getoon dat indien lisien in 'n oormaat gevoer word, dit tot die verlaging van groei deur antagonisering van arginien gelei het. Benewens die aminosuurbehoefte, is die korrekte verhouding tussen die aminosure dus 'n vereiste.

Voeding van die kat

Hoewel die voedingspatroon van die kat tot 'n baie groot mate deur die noue assosiasie met die mens beïnvloed is, moet die volgende drie primêre feite nie uit die oog verloor word nie:

* Die kat is 'n woestyntipe dier wat sy oorsprong in die subtropiese areas gehad het.
* Onder natuurlike toestande, is dit 'n ware karnivoor.
* Dit is 'n geleentheids-, eerder as 'n kontinue vreter.

Dié feite is verantwoordelik vir die gespesialiseerde ekonomie en unieke behoeftes van die kat[49].

Vloeistofinname

Studies wat op die vloeistofinname en urineproduksie van katte gedoen is, het getoon dat die kat 'n minimale inname van vloeistof in die vorm van water het, dit wil sê in die natuurlike staat drink die kat ongeveer een maal in 24 uur. Hoewel die kat dus geleer kan word om melk en selfs tee te drink, is dit beslis nie natuurlike gedrag nie. Die enigste wyse waardeur verseker kan word dat die waterinname van die kat verhoog word, is om addisionele water by die dieet in te sluit. In die natuurlike staat verkry die kat die meeste van sy vloeistof uit karkasse wat ongeveer 70% water bevat. Metaboliese water word ook verkry uit die vet wat ingeneem word. Dit is waarskynlik die rede waarom die droë beskuit-tipe diëte nog nooit werklik deur die kat aanvaar is nie, selfs al drink katte meer water wanneer dié tipe dieet voorgesit word. Die totale waterinname van jong katte was 76,9 g kg^{-1} liggaamsmassa, terwyl die waarde tot 57,1 g kg^{-1} in die volwasse kat gedaal het. In vergelyking met 'n volwasse mens, het die kat 'n besondere vermoë om urine te konsentreer en daardeur, soos woestyndiere, water in hul liggame terug te hou.

Kalorieë, koolhidrate en vette

Die kaloriebehoeftes van katte is reeds in die vyftiger jare deur werkers bepaal en is vir die volwasse kat, op 60 kkal kg^{-1} liggaamsmassa gestel. Onder natuurlike toestande sal die kat ongeveer 60% van sy kalorieë uit vette verkry. 'n Klein verhoging in die hoeveelheid vet in 'n spesifieke dieet mag die aanvaarbaarheid daarvoor vir die kat opmerklik verhoog.

Die stelling wat dikwels gemaak word dat katte 'n gevoeligheid vir vet het, is deur Humphreys & Scott[25] ondersoek. In die studie is die proporsie vet en plantolie geleidelik tot 64% van die droë massa van die dieet verhoog. Gedurende die laaste maande van die eksperiment was die totale vet-inname 30,7 g dag^{-1}, die gemiddelde kalorie-inname 276 kkal en die gemiddelde massatoename 8,5 g dag^{-1} vir die individuele katte wat 2,6 kg geweeg het. Die vetinhoud van die ontlasting het tydens die proef konstant gebly, wat daarop dui dat die vet met 'n hoë mate van doeltreffendheid benut is. Aangesien die kalorie-inname bo die behoefte gestyg het, is met post mortale ondersoek gevind dat die vet onderhuids, in die buiknet en retroperitoneaal neergelê is. 'n Besondere mate van vetsug is waargeneem. Slegs die katte wat onbeskermde olie ontvang het, wat nie anti-oksidant bevat het nie, het tekens van geelvetsiekte getoon.

Die toksiese effek van poli-onversadigde vetsure van marine oorsprong, soos byvoorbeeld in lewertraan en tunavis, is welbekend. Die rede hiervoor is dat die kat nie 6-denaturase bevat nie en dieetbronne vereis wat die prostaglandien-voorgangers dihomo-, linoleïen- en aragidoniensuur bevat. Dit word algemeen aanvaar dat die kat 1% linoleïensuur in die dieet vereis ten einde essensiële vetsuurtekorte te verhoed.

Daar is geen bewys gelewer dat die kat koolhidrate in die dieet vereis nie. Indien dit egter voldoende proteïen en vet bevat, kan die balans van die kaloriebehoeftes deur koolhidrate voorsien word, aangesien dit 'n goedkoper bron van energie is. Laktose word swak benut.

Tabel 13 Ideale diëte vir katte[35]

	Pasgebore %	Jong en volwasse %
Water	72	70
Proteïen	9,5	14
Vet	6,8	10
Koolhidraat	10	5
As	0,75	1
Kalsium	0,035	0,6
kkal	142	150

Hoewel die laktase-aktiwiteit hoog in die jong kat is, daal dit tot 'n baie lae peil in die volwasse kat. Die gevolg is dat die melksuiker na die dikderm deurvloei waar dit fermentasie ondergaan, die ophoping van gas veroorsaak en veral in die Siamese kat diarree mag veroorsaak.

Glikogeen en dekstrien word geredelik deur die pankreatiese amilase verteer, maar die stysel moet gekook wees[49].

'n "Ideale" dieet vir katte waarin die hoë kaloriedigtheid van die natuurlike dieet beklemtoon word, word in Tabel 13 weergegee.

Proteïen- en aminosuurbehoeftes van katte

Die tradisionele beskouing was dat katte 'n hoër proteïenbehoefte as ander soogdiere het. Dickinson & Scott[19] was van mening dat die dieet vir groeiende katte 33% proteïen van 'n hoë biologiese waarde moes bevat. Volwasse katte het 'n proteïenbehoefte van 21% gehad. Die bevindings is ondersteun deur Jansen et al.[26] Die resultate is egter gekompliseer deur die gebrek aan inligting oor die kat se aminosuurbehoeftes. Burger et al.[9] en Burger[10] se navorsing dui egter daarop dat indien die aminosuurverhoudings vir die kat vir optimale groei aangepas word, 12,5% proteïen voldoende sal wees vir katte.

Swawelbevattende aminosure

Nadat werkers aanvanklik van mening was dat katte nie swawelbevattende aminosure in hul dieet benodig nie, het Teeter[51 52] se studies daarop gedui dat katte wel die aminosure in hul dieet vereis. Uit hierdie navorsing blyk dit dat hoewel D-metionien deur die kat benut word, die prestasie van die katte laer was as op L-metionien. DL-OH-metionien is egter swak as bron van metionien-aktiwiteit benut toe dit in 'n kristallyne aminosuur-dieet voorsien is. Toe dit egter in 'n dieet wat intakte proteïene in die vorm van sojaboonmeel bevat voorsien is, is dit goed benut (Tabel 14).

Tabel 14: Die doeltreffendheid van DL - hidroksie — metionien (Ca) in diëte van katte wat intakte proteïen bevat[52]

Dieet	Toename g dag^{-1}	Toename/ voerinname
Basaal	−2,1 ± 0,9	−0,9 ± 0,04
Basaal + 0,25% L-metionien	5,6 ± 2,4[a]	0,19 ± 0,05[a]
Basaal + 0,30% DL-OH-Met(CA)	4,3 ± 2,7[a]	0,12 ± 0,06[a]

Getalle met dieselfde boskrifte verskil nie betekenisvol nie.

Katte metaboliseer swawelbevattende komponente anders as ander soogdiere. Die feit word uitgewys deur die unieke swawelbevattende verbindings wat in die urine van katte voorkom. Een hiervan is 'n sistienderivaat iso-valtien. Die tweede verbinding felinien, met die volgende struktuur

$HOOC - CH(NH_2)CH_2 - S - C(CH_3)_2 - CH_2 - CH_2OH$

word in groot hoeveelhede in die urine van katte uitgeskei. Bewys is gelewer dat sistien en/of leusien of mevaloniensuur tot die biosintese van felinien kan bydra. Die aminosuur word egter nie in die plasma van katte waargeneem nie. Dit wil dus voorkom asof dit in die niere gevorm word en direk in die urine uitgeskei word. Die metaboliese belang van die felinien is nog onbekend, maar die sintese daarvan mag 'n belangrike verwantskap met die kat se behoeftes aan taurien hê.

Streng gesproke is taurien nie 'n aminosuur nie, maar 'n amino-sulfoonsuur wat nie deel van die polipeptied-ketting van proteïen is nie. Dit is 'n belangrike eindproduk van die swawelaminosuur-metabolisme en word normaalweg geproduseer van die swawelbevattende aminosure metionien en sistien. Taurien is in verskeie funksies betrokke maar die een wat van die grootste belang is, is die rol wat dit in die struktuur en funksie van die retina speel. 'n Taurientekort lei uiteindelik tot retinale degenerasie en 'n beperking van die visuele respons.

Dit mag egter 'n geruime tyd neem voordat enige kliniese tekens van 'n visuele defek merkbaar is. Dit mag 'n jaar of langer op 'n taurientekort-dieet vereis. In teenstelling met ander soogdiere kan katte nie genoeg taurien van die swawelbevattende aminosure sintetiseer nie. Die ensiem verantwoordelik vir die omsetting is nie totaal afwesig nie, maar die aktiwiteit daarvan is nie hoog genoeg om in al die kat se behoeftes te voorsien nie.

Die besondere gevoeligheid van die kat word verhoog deur sy totale afhanklikheid van taurien vir die vorming van galsoute. In teenstelling met ander spesies, kan die kat nie glisien vir die doel gebruik nie. Navorsing om die presiese taurienbehoefte van katte te bepaal, duur tans nog voort. Maar die huidige behoefte word op 0,5% van die dieet gestel, dit is ongeveer 50 mg 400 kkal^{-1}. Die rykste bron van taurien is dierlike proteïen.

Teeter et al.[52] toon dat, waar diëte (4700 kkal ME kg^{-1} dieet) aan katte voorsien is, met stygende peile van metionien in die teenwoordigheid van taurien, anorganiese sulfaat en 'n oormaat sistien, 'n plato in die tempo en doeltreffendheid van groei waargeneem is, wanneer 0,45% metionien voorsien is. Maksimale groei is verkry indien die metionien vlak met 0,45% sistien voorsien is. Dus kan 50% van die kat se swawelaminosuurbehoeftes deur sistien bevredig word. Die katte groei egter net so goed indien hul swawelaminosure in die vorm van 1% metionien voorsien word. Soos in die geval van ander soogdiere is sistien 'n nie-essensiële aminosuur wat egter as metionien-bespaarder kan optree.

Triptofaan

Triptofaan-metabolisme in die kat blyk ook anders te wees as in baie ander soogdiere. In die besonder word gevind dat die triptofaan-metaboliet, kinureensuur, nie in die urine van katte teenwoordig is nie. Die meeste van die triptofaan word deur die spesies gedegradeer tot antranielsuur, 'n reaksie waarin die ko-ensiem peridoksaalfosfaat betrokke is. Drie ander triptofaan-metaboliete wat normaalweg in die soogdier-urine voorkom nl. xantoriensuur, nikotiensuur en N-metiel-nikotienamied kan ook nie in die urine van katte opgespoor word nie. Dit word voorts gevind dat indien 'n niasientekort in katte ontwikkel, dit nie deur die mondelingse of subkutaniese toediening van triptofaan opgehef kan

word nie. 'n Subkutaniese inspuiting van nikotiensuur of nikotienamied lewer egter binne 24 uur 'n respons. Triptofaan kan dus nie niasien in die dieet van katte vervang nie.

Die triptofaanbehoefte van die groeiende kat word op 0,15% beraam in studies wat deur Anderson et al[1] uitgevoer is.

Treonien

Studies wat deur Titchenal et al.[54] uitgevoer is, het daarop gedui dat neurologiese afwykings die gevolg was van 'n treonientekort eerder as 'n wanbalans in treonien. 'n Tekort aan treonien het voorts tot gevolg gehad dat die katte tekens van lamheid van die voor- en agterbene getoon het. Afwyking van die polse was opmerklik en die sg. "bowed leg" toestand het ontstaan, wat meegebring het dat die katte later moeilik kon loop. Tews et al.[53] het in hul navorsing getoon dat die neurologiese afwyking die gevolg mag wees van 'n kompetisie vir die vervoer van beperkende aminosure in die plasma. Die werk het bevestig dat treonien-opname deur die brein, teen fisiologiese konsentrasies van die aminosure, tot 'n groot mate deur die kompetisie met serien en alanien beïnvloed word. Groot variasie in die voorkoms van die kliniese tekens het egter tussen die individuele katte bestaan. Die variasie mag die verskille tussen katte in terme van aminosuurbehoeftes, sowel as die verskil in die behoefte van verskillende weefsels verklaar. Sekere katte kan dus 'n ernstige verlaging in groei sowel as neurologiese abnormaliteite toon, terwyl ander ernstiger neurologiese of ledemaat-afwykings mag openbaar, sonder 'n afwyking in liggaamstoename.

Die treonienbehoeftes van katte is deur Anderson et al[1] op 0,80% gestel.

Arginien

Bewys is deur Morris & Rogers[39] gelewer dat indien 'n arginienvrye dieet aan katte voorgesit word, die katte hiperammonemies raak, 'n toestand wat tot hul dood kan lei. Aangesien ornotien ook hiperammonemie verhoed, blyk dit dat katte nie ornotien, wat in anaplerotiese hoeveelhede benodig word, vir die instandhouding van die ureumsiklus, kan sintetiseer nie. In soverre dit die belang van arginien in die dieet van katte betref, dui studies deur Costello et al.[16] daarop dat groei en dieet-inname nie sulke goeie parameters as orotiensuur van arginientekorte is nie. Die beraamde arginienpeil in die dieet wat vereis word om die orotiensuur-uitskeiding te minimaliseer, word op 1,05% gestel wat 0,3% hoër is as die vlak vir maksimale groei.

Hoewel daar in die toekoms verdere navorsing gedoen sal word om die optimale aminosuurbehoeftes en -verhoudings vir katte te kwantifiseer, het die werk tot dusver onomwonde bewys dat katte op gebalanseerde diëte 'n laer proteïenbehoefte het as wat aanvanklik gemeen is. Dit behoort dus moontlik te wees om voortaan gebalanseerde katvoedsel teen laer pryse saam te stel.

Gevolgtrekking

Uit die bespreking tot dusver moet ons dus die afleiding maak dat die wetenskap van honde- en kattevoeding die afgelope aantal jare 'n betekenisvolle bydra tot beter voedingsisteme van beide spesies gelewer het. Die huidige NRC-aanbevelings moet dus dienooreenkomstig aangepas word. Dit kan verwag word dat hierdie momentum sal voortduur en dat die voeding van sowel honde as katte in die toekoms 'n gesofistikeerde wetenskaplike bedryf sal word. In hierdie bedryf sal die veekundige en meer spesifiek die voedingkundige, 'n belangrike bydrae lewer.

Erkenning

Die bekwame insette van mev. Karin de Beer met die redaksionele versorging van die referaat en die voorbereiding van die grafiese materiaal word met groot waardering erken.

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A-EQUI-2 INFLUENZA IN HORSES IN THE REPUBLIC OF SOUTH AFRICA

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ABSTRACT: Rogers A.L. **A-equi-2 influenza in horses in the Republic of South Africa.** *Journal of the South African Veterinary Association* (1988) **59** No. 2, 123-125 (En). Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

In early December 1986 A-equi-2 influenza virus was isolated for the first time in the Republic of South Africa. All horses were susceptible to the highly contagious aerosol-borne orthomyxovirus resulting in widespread outbreaks of equine influenza with typical primary respiratory symptoms. Treatment consisted of rest, anti-inflammatory drugs, antibiotics and good nursing. Future protection can be obtained by vaccination.

Key words: Influenza, A-equi-2 influenza virus, horses, South Africa

INTRODUCTION

Influenza is an acute contagious respiratory disease caused by influenza viruses A-equi-1 and A-equi-2[2]. A-equi-2 was first isolated during an outbreak of respiratory disease in the U.S.A. and Canada in 1963 and was called the "Miami" strain (A/Equi 2/Miami/1/63)[4]. A-equi-2 virus was also responsible for an outbreak of influenza in England and Europe in 1965[9].

In early December 1986 an outbreak of respiratory disease in horses caused by A-equi-2 influenza virus (A/Equi 2/Johannesburg/1/86) began in the Transvaal and Cape Province. The first cases were seen in racehorses at Turffontein and the infection spread rapidly through the stables. Before clinical signs appeared, horses incubating the disease were transported from Turffontein to the Cape, starting the spread of infection. The same infected horse transporter also carried show-jumpers to the Welkom Show, thus spreading the infection around the country. The larger part of Natal and various other isolated areas remained unaffected.

AETIOLOGY

Equine influenza virus A-equi-2 is an RNA virus of the orthomyxovirus group. The influenza virus ribonucleoproteins are differentiated into types A, B and C due to the production of specific complement-fixing antibodies. A lipoprotein envelope surrounds the RNA core and contains 2 functionally distinguishable components; a neuraminidase enzyme and haemagglutinin. Their characteristics form the basis to distinguish between subtypes A-equi-1 and A-equi-2. A-equi-2 is antigenically unstable and shows a slight antigenic drift. It survives for about 8 h (but may last as long as 24 h) in the environment or on man, and shows very little resistance to antiseptics or disinfectants customarily used for viral infections[2].

EPIDEMIOLOGY

Influenza is a highly contagious disease now epizootic in the Republic of South Africa. All horses irrespective of age, sex or breed were susceptible before the outbreak[2 11]. Stressed horses and foals younger than 3 months, tend to be the most severely affected[2 9]. Mares which have recovered at least 12 - 14 d prior to foaling, secrete sufficient colostral antibodies to provide the foal with protective immunity for up to 6 months, but mares which are infected closer to or at partus, produce a highly infective environment for the unprotected foal[9]. The disease usually originates when horses incubating the disease are introduced into a susceptible population of horses, resulting in explosive outbreaks with most horses being affected within 2 to 3 d. Outbreaks tend to be limited to a single aetiological agent[1 2].

The infection is spread primarily by aerosols[2] containing nasal secretions, a single cough spreading the virus up to 35 m. The virus can also be transmitted indirectly via contaminated fomites and by humans[4]. Wind and flies may also play a role. Horses are thought to excrete the virus in nasal secretions for up to 8 d, but it is suspected that some horses may shed the virus for periods as long as a few weeks[2 3]. There appears to be no carrier state and therefore a low level of disease must persist in the equine population in order to maintain the virus. It is possible that A-Equi-2 may form antigenic variants sufficiently different so as to overcome immunity, thus resulting in new outbreaks[1 2]. More recently, two variants have been recognised, namely, A-equi-2/Fontainbleau/79 and A-equi-2/Kentucky/81 which show a fair cross-immunity[5 10].

Even though similarities exist between equine influenza A-type-2 virus and human influenza A-type-2 virus formerly prevalent in human populations in the late 19th century, equine influenza A-type-2 has not been found to be responsible for the influenza symptoms occurring in some humans in close contact with infected horses[3 7].

PATHOGENESIS

The virus is inhaled and trapped by the respiratory tract mucosa where it replicates and destroys the ciliated respiratory epithelium resulting in laryngitis, tracheitis and bronchitis. In many cases a peribronchitis and pneumonia occur which tend to perpetuate the cough and may be responsible for chronic disease[3]. Severe damage to the myocardium and brain is also possible. The pneumonia, peribronchitis and myocardial involve-

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ment suggest that the viral invasion is not exclusively air-borne and that at least a transitory viraemic phase is present[2]. The muscular pain may be due to the toxic effect of a viral protein, which may also affect the heart and smooth muscles, including those of the gastro-intestinal tract.

Lesions of a purely viral origin tend to heal completely in 3 weeks. Unfortunately secondary infection can easily develop in unfavourable circumstances and can initially cause benign bronchitis and pneumonia, which may progress to suppurative bronchopneumonia with a grave prognosis[1 2]. Foals are especially susceptible and may also develop serious myocardial and pulmonary damage resulting in death within a few days to a couple of weeks[1 2 9].

CLINICAL SIGNS

The incubation period is from 2 to 5 d although it may be as short as 18 h[4]. The initial sign is a rapid rise in temperature to between 39°C and 41°C, occasionally higher, which usually returns to normal after 2 to 6 d[1 7]. A deep, painful hacking cough usually develops from within a few hours to a maximum of 2 d after the onset of fever. The cough is easily elicited on tracheal palpation and it may become moist after 5 d to 3 weeks due to secondary bacterial infection. Respiration is rapid and shallow with fine moist rales or crepitation being heard on ausculatation, especially over the apical lobes[1 2]. A catarrhal rhinitis often accompanied by a serous nasal discharge, and occasionally a mucopurulent discharge, is present[4].

The fever is often followed by depression, anorexia and muscle weakness or stiffness producing an unsteady gait[1 2 7]. Spasmodic and impaction colic have also been seen in a number of cases. The mandibular lymphnodes tend to be enlarged and sensitive while the visible mucous membranes may be markedly inflamed. The pulse rate may rise to above 60 beats per min[2].

If complete rest is enforced, the disease usually runs an uneventful course and horses recover clinically within 10 d although the cough may persist for 2 to 3 weeks due to a mild laryngitis[2 7]. Exercise and stress intensify or prolong the course of the disease[4]. A haemoglobinuria has been noted in some horses a few weeks after recovery[1 7].

Pregnant mares may resorb or abort due to the fever, while foals exposed at birth, and not protected by colostral antibodies, show severe symptoms at 4 to 5 d of age and most die by 9 to 10 d old[9].

CLINICAL PATHOLOGY

The total erythrocyte count is usually in the lower ranges of normal although a marked anaemia may occur. The total leucocyte count decreases at the onset of clinical signs due to a lymphopaenia. If marked systemic symptoms are present, an increase in lactate dehydrogenase (isoenzyme 3) occurs due to liver damage. The creatine phosphokinase may also be increased especially in horses with a rapid weak pulse[2].

COMPLICATIONS

Performance-threatening complications can usually be avoided with proper care[7]. Complications occur mainly when horses are worked too soon or stressed resulting in permanent lung or heart damage. Secondary infection causing a purulent bronchitis and broncho-pneumonia, with a recurrent fever and mucopurulent nasal discharge, may easily occur soon after the viral infection. A chronic bronchiolitis may lead to alveolar emphysema which can progress to chronic obstructive pulmonary disease. Guttural pouch infections, sinusitis, myocarditis, myositis and liver damage may occur as well as oedema of the legs and scrotum. Spasmodic and impaction colics have also been noted. A rare complication is the development of laryngeal hemiplegia, the mechanism of which is unknown[1 2 9].

DIAGNOSIS

At this stage a presumptive diagnosis can be made on the high morbidity and rapid spread of the disease as well as on the presence of typical respiratory symptoms[2]. A definitive diagnosis can be made by viral isolation from naso-pharyngeal swabs taken immediately after the onset of disease. This was the method initially used to identify the virus in the R.S.A. (B J Erasmus 1986 Veterinary Research Institute, Onderstepoort, personal communication). A diagnosis can also be made using radial haemolysis, haemagglutination inhibition, complement fixation or serum neutralisation on paired serum samples. When the haemagglutination inhibition test is used a 4-fold increase is diagnostic and it is also possible to identify the specific viral subtype, while if radial haemolysis is used, lower titres can be detected than with the haemagglutination method[2 7 12].

Viral rhinopneumonitis (EHV), adenovirus, rhinovirus, the pulmonary form of African horse sickness and equine viral arteritis must also be considered in the differential diagnosis of the disease[2].

TREATMENT

Complete rest is essential during the disease and for 2 to 3 weeks after clinical recovery[2]. Thereafter daily hand-walking for 10 to 15 min is advised for the next 2 weeks, after which light work can commence provided the horses show no respiratory distress or coughing. Work may be increased gradually over a month and full work resumed in the third month. Horses showing respiratory distress or coughing should be rested until the lung sounds return to normal.

Controversy exists over the routine use of antibiotics. Liu[6] believes the routine use of antibiotics in uncomplicated cases is unwise as it may lead to the development of an infection by resistant bacteria or fungi. He believes antibacterial therapy should be reserved for secondary bacterial complications, following sensitivity tests on organisms cultured from transtracheal aspirates. Other references suggest at least a 5-d prophylactic course of antibiotics or sulphonamides[2]. Prophylactic antibiotics, used in the early stages of viral infection, serve to protect the animal before its immune system responds i.e. when the horse is most susceptible to secondary infection. If a narrow spectrum antibiotic, such as penicillin, is used for this purpose, it does not kill off all the bacteria which would predispose to fungal infections, and more effective antimicrobials remain available for use in pro-

blem cases. The use of anti-inflammatory drugs such as phenylbutazone is advised in horses showing high fevers (higher than 39,5°C) or inappetence due to the fever[2]. They help to prevent excessive pulmonary inflammation, but at the same time they decrease the body's ability to eliminate the virus. The control of fever in pregnant mares is extremely important to prevent resorption and abortion[6].

Bronchodilators and expectorants can also be considered in individual cases showing severe respiratory distress[6]. Vitamins such as vitamin A, vitamin BCo, vitamin C and vitamin E may also be considered, while the administration of selenium enhances the immunogenic response of the body to the virus. Antiviral drugs are still in the experimental stages[8].

A treatment regimen which has produced relatively good results is the following: 1) 20 ml penicillin (Procaine penicillin G, Milvet) given intramuscularly once daily for 3 d and then on alternate days for a further 2 treatments and 2) 10 ml phenylbutazone (Tomanol, Byk Gulden) given intravenously once daily for 3 d and thereafter only if the fever is higher than 39,5°C (C I Meyer 1986 Honeydew Veterinary Clinic, personal communication).

The use of high levels of prophylactic antibiotics for the first 3 - 5 d in foals infected at birth, and not protected by colostral antibodies, has met with limited success unless combined with the administration of immune or hyperimmune serum before 60 h of age[9].

Nursing care of all infected horses is extremely important as well as twice daily temperature recording. The feeding of concentrates should be reduced and it is often helpful to add molasses to increase palatability and energy levels at the same time as acting as a laxative which helps to maintain normal gut motility. Hay should be dampened and fed off the ground to promote drainage of the upper respiratory tract. The stables should be well ventilated and kept as dust-free as possible. A solution of a few drops of eucalyptus oil, 10 ml of Savlon (I C I (South Africa) Ltd, Braamfontein, Johannesburg) and 5 ℓ of warm water can be used to clean out the nostrils twice daily, and a mixture of honey (500 g), lemon (approximately 60 ml of concentrate) and water (2 ℓ) given orally (20 - 30 mℓ) helps to alleviate pharyngeal inflammation.

PROGNOSIS

The prognosis for total recovery is reasonably good if the viral infection is not compounded by a bacterial or fungal infection or chronic complications. It is much less favourable if secondary infections (often *Streptococcus zooepidemicus, Streptococcus equi,* or more seldom *Escherichia coli*) resulting in a purulent bronchopneumonia develop. Exudative pleuritis may also occur and has a poor prognosis[2 9]. Myocarditis and chronic pulmonary involvement give rise to poor performance in horses returned to work too soon or stressed during recovery. Mortality usually results from secondary infections and is low, except in foals younger than 3 weeks where it can be as high as 40%[1].

CONTROL

During an outbreak, all newly-introduced horses should be isolated for at least 10 d and all sick horses should be isolated. Large congregations of horses and stressful situations should be avoided where possible and there should be no transport of horses[2 4]. Human contact with infected horses should also be controlled.

The bivalent vaccine is a killed vaccine containing both A-equi-1 and A-equi-2 virus strains. Two vaccinations, given 2-6 weeks apart, are required initially, with bi-annual vaccination thereafter[2 6]. If the interval between the initial vaccination and the first booster is less than 4 weeks, a second booster is required 2 months after the first. The vaccine should then be repeated after 4 months and bi-annually thereafter (Stud Health Committee, Equine Practitioners Group, 1987 Unpublished work, presented at 1987 Equine Practitioners Congress, Kruger National Park). Protective immunity is obtained 1 - 2 weeks after the second vaccination and although it may not provide complete protection in all horses, the course of infection in vaccinated horses is much milder[1]. The vaccine is given intramuscularly and may cause a slight local reaction which disappears within a few weeks. It has been suggested that horses be rested on the day of vaccination and for 1 to 2 d thereafter[2].

Colostral antibodies in vaccinated mares, provide effective protection for foals for 2 to 6 months. These foals should be vaccinated at about 4 to 6 months of age, while foals which are unlikely to receive colostral antibodies should be vaccinated from 2 weeks of age, although the immunity they develop is not as effective as that of older horses[6]. Foals vaccinated before 3 months of age require 2 boosters, as described for adult horses given the initial 2 vaccinations less than 4 weeks apart. Mares should have one of their booster vaccinations during late pregnancy, but at least 2 weeks before foaling.

The immunity acquired after natural infection is specific and is effective for about one year but it may be as short as a few months or as long as 3 years[2 10].

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VETERINARY IMMUNOLOGY
An Introduction

IAN TIZARD

3rd Edn. W.B. Saunders Company, Philadelphia, London, Toronto. 1987 pp XVIII and 401, illustrations 220 and 53 tables, Price £25 (ISBN 0-7216-2098-1)

The author says in the preface to this book that he has tried to demonstrate the scope of veterinary immunology in such a way that students and veterinarians can comprehend the subject while at the same time appreciating its complexity. This has resulted in a book which is easy to consult and in which the essentials are uncluttered by unnecessary detail — an important requirement of any introductory text. It is implicit of such an approach, however, that little or no background is given to the exciting development of current immunological concepts over the last two decades (eg. understanding the generation of antibody diversity and the T-cell receptor) or to the experimental basis thereof. Nevertheless, all the major developments which have occurred recently are covered and there is no doubt that this edition is throughly up-to-date.

The book comprises 5 sections covering the immune system, serology, protective immunity, hypersensitivity and inflammation and immunological diseases. Each of these sections is further subdivided into a number of chapters which are well-structured and written in a lucid and straight-forward style. The illustrations, although exclusively in black and white, are of a high standard and depict the point simply and clearly. References are not given in the text but at the end of each chapter there is a short list of specifically pertinent papers.

Numerous useful and important references are made to specifically veterinary aspects of immunology and the last part of the book is devoted to animal diseases with an immunological component and provides a comprehensive overview in this respect. Other important immunological problems such as the serology of Brucellosis are summarised in a way which will be helpful to students.

Inevitably in a book as broadly based as this there are details or points of view with which one could argue (eg. apparent confusion between viral haemadsorption and neutralisation) but none were encountered which could materially affect the point being made. On the other hand, there are 2 omissions which, for some, would detract from a book on veterinary immunology. The first is a lack of any information on the immune systems of animals other than mammals and birds and secondly, there is no mention of the principles of herd immunity. It may be that the author feels that the latter aspect is more appropriate in a text on epidemiology. Nevertheless, because of the vital importance of herd immunity in the veterinary field, it could profitably be included in future editions.

The book has been produced with a view to containing costs (soft but durable cover and a lack of colour illustrations) but the paper quality, type size and clarity and the overall appearance are good.

This book, which fulfills the author's objectives admirably, can confidently be recommended to pre- and post-graduate veterinary students as well as to those of us who, for a variety of reasons, know less about the theoretical basis of immunology than is necessary to operate efficiently in a variety of specialities.

G.R. Thomson

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DICTIONARY FOR VETERINARY SCIENCES AND BIOSCIENCES
German-English/English-German

R. MACK

Paul Parey Scientific Publishers, Berlin and Hamburg, 1988, 321 pp, Price not stated (ISBN 3-489-50516-6)

Although English is the major international scientific language, a considerable body of scientific literature is still published in German. In fact, the library of the Faculty of Veterinary Science, University of Pretoria, receives 33 periodicals with German titles. Technical German-English dictionaries will therfore remain in demand.

The aim of this dictionary is to supplement the standard German-English general dictionaries with technical terms in the fields of anatomy, microbiology, physiology, parasitology, pathology, pharmacology, toxicology and zootechnology, with special reference to domestic animals and their diseases. The more common wild animals of Europe and popular zoo animals are also included. Invertebrates are confined to parasites of livestock and vectors of disease.

Contrary to general practice, the listed words are not printed in bold face, which is somewhat irritating. More serious is the total disregard for the standard international practice of printing the scientific names of species in italics.

Curiously, the scientific names of the waterbuck and bushpig are misspelt at each listing. In the index of scientific names (called 'Latin names' although some are of Greek or other origin), the scientific name of the black wildebeest is misspelt.

Although long German words are the butt of many a joke, some terms listed in the dictionary are short and aptly descriptive. This source of possible alternatives should be borne in mind when Afrikaans phrases are coined, instead of the often cumbersome direct translations from English.

Some examples:

— 'Twin pregnancy' is 'Doppelfrucht' in German ('Dubbelvrug' instead of "tweelingdragtigheid" in Afrikaans?)
— 'Intrathoracic pressure' and 'intraventricular pressure' are 'Pleuradruck' and 'Kammerdruck' respectively
— 'Subunit vaccine' is 'Spaltvakzine' (maybe 'splytentstof' in Afrikaans?)

Although a dictionary such as this one would be used by few private practitioners, it is indispensible to libraries and research establishments.

B.L. Penzhorn