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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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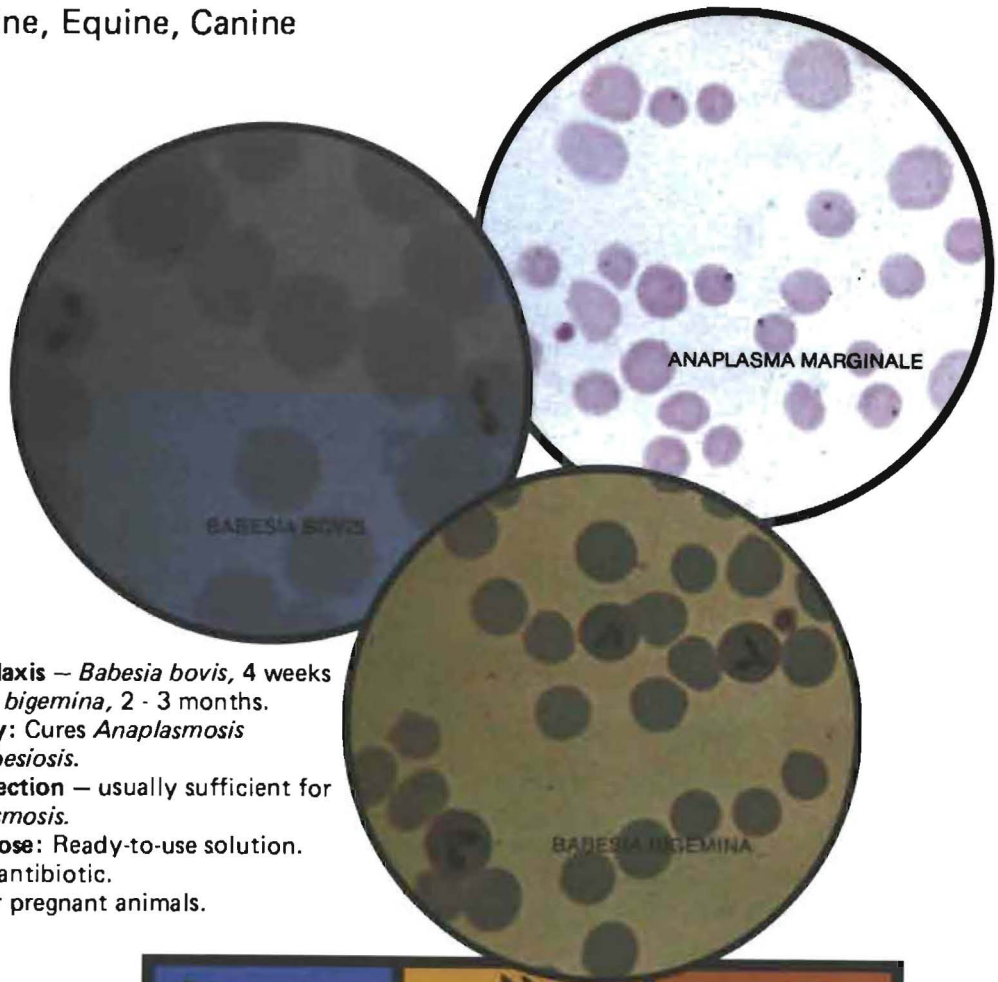

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THE VETERINARIAN AND ANIMAL WELFARE ORGANIZATIONS

Recently the S A V A published a booklet entitled "The Veterinarian and Animal Welfare Organizations' which has been widely distributed amongst practitioners and animal welfare organizations (AWO's). The guidelines – as they are called and indeed the publication does not aspire to be more than this – cover all aspects of veterinary work done by A W O's and are of great help in assisting both parties to decide on how to act in a given circumstance or when one or the other is presented with a situation which could lead to disharmony and even confrontation between them. From the outset a principle is defined viz. "the basis of co-operation of veterinarians with A W O's is that they confine their clinical work to animals in their care (excluding animals which are being boarded for a fee), to animals owned by underpriviledged people who cannot pay normal veterinary fees, to stray and donated animals and to emergency cases". To further this end it is also stated "No veterinarian should work for an A W O which does not employ an almoning system" and "No veterinarian should become an employee of an A W O without entering into a contract that clearly defines his duties and observes the requirements of the Veterinary Code".

The guide, which is regarded as a document subject to review from time to time, has been very well received and is referred to frequently by both parties as problem situations arise.

It encompasses A W O's which employ full time veterinarians and veterinary nurses, part-time veterinarians and those that make use of a practitioner's services on an ad hoc basis, or who have access to veterinary help given in an honorary capacity. The guide is chiefly concerned with details on the implementation of an almoning system, the ethics of veterinarians employed by A W O's, reduced-cost spaying of bitches, the control of infectious diseases on their premises and the general involvement of veterinarians in animal welfare activities.

Over the years an unhealthy "we and they" situation had developed between practitioners and A W O's in some cities and towns, which was caused mainly by some A W O's adopting an 'open house' policy in treating all animals presented for treatment irrespective of the financial standing of the owner. The pet owning public, in some instances, felt that this was their right as members of the particular A W O or did so in the belief that they were promoting their chosen organization and thus gave handsome donations. Other sources of friction to the veterinarians were that some A W O personnel favoured and promoted certain practitioners in an area to the exclusion of others; insisted that bitches bought should be returned to them for spaying and that they disseminated infectious diseases by their policy of selling or finding homes for puppies and kittens after a short stay in the animal home where disease control was extremely difficult. The A W O's, on their part, felt they should be getting far more sympathetic consideration and free or reduced-cost clinical help from their local veterinarians, people dedicated, as they were, to alleviate suffering in animals.

This conflict was to be deplored as both parties were interdependent particularly because of the large, underpriviledged pet owning population in South Africa. Many reasonable people from both sides therefore got together to sort it out. The national animal welfare committee met regularly with the executive committee of the Federation of S P C A's and a very healthy, friendly and constructive relationship developed. Arising from these meetings, the executive of the animal welfare committee commenced work on the booklet ensuring, through many drafts, that the opinions of all interested veterinarians and a few other A W O's apart from the federation were canvassed. Branches of the S A V A were encouraged to nominate persons to initiate and serve on local and regional liaison committees and to forward their minutes to the main committee for information. In many areas these committees meet regularly and now, with the booklet as a guide, are able to resolve difficulties as they arise. Indeed it is very evident that in towns where the machinery for dialogue has broken down, friction still exists to the detriment of both parties and the animals they should be helping.

The veterinarian is also expected to "make a significant contribution to the welfare of animals by endeavouring to co-operate with and assist A W O's by serving on their committees and on liaison committees of the S A V A, by rendering clinical services in a part-time or honorary capacity, by assisting in the enforcement of laws pertaining to the prevention of cruelty to animals in providing expert evidence for court cases, by training lay personnel in methods of euthanasia and supervising the use of drugs used for this purpose, by providing them with professional advice and standards on the housing, management and care of animals and assisting with fundraising activities".

The larger A W O's though in some ways loathe to forego their income derived from donations received when their veterinarian employee treated animals owned by those who could afford a veterinarian's normal fees, were more concerned that their veterinarians would fall foul of the Veterinary Board by allowing themselves to be used professionally to earn income for their lay organization, and secondly that while attending to these animals he was neglecting his first priority which was the help needed by the animals in the care of the institution or those owned by underpriviledged people.

The booklet also emphasised that the animal welfare employed veterinarian should, apart from working within the terms of the Ethical Code, give a very high standard of service to his patients, accept full responsibility for the control of drugs, and provide an after hours' service.

On the vexed question of spaying, the guide is very explicit. It covers the many situations and alternatives

that do occur which arise from the principle adopted by most A W O's, that any bitch adopted from their kennels must be sterilized in an endeavour to control the excess dog population. To promote the sale of bitches from A W O's it was agreed that they be spayed by members of the S A V A at a fee lower than the normal national basic fee (approximately 25–33% less). The new owners may have their newly acquired pet spayed by the veterinarian of their own choice but have to pay a deposit to the A W O to cover this cost. This ensures, in most cases, that a bitch in fact is spayed. The booklet provides, in an appendix, specimen reduced-fee application forms that help validate claims for refunds of the deposit while at the same time being a certificate that the bitch has been spayed. "Individual veterinarians may elect not to make themselves available in providing reduced-cost spaying facilities for A W O's, and the co-operation of all veterinarians will be sought in implementing the minimum fee as a goodwill measure".

It is also categorically stated that "private veterinarians may, at their own discretion, elect to provide any clinical or surgical services for indigent persons or A W O's free of charge".

On the question of almoning, the guide provides a specimen almoning form and suggests the wording on notices that could be displayed at the entrance of the animal welfare clinic and how the reception staff could, in a "firm and tactful" manner, draw the attention of those presenting their animals for treatment to this policy.

In the sphere of work performed by laymen employed by A W O's, the veterinarian "should also instruct and educate lay staff who work in underpriviledged areas and in mobile clinics for the sole purpose of dealing with the common problems which they will encounter".

The considerable responsibility the veterinarian associated with the A W O has in containing the spread of infectious disease is identified and guidelines suggested as to how this be affected – the main campaign being to "blanket" immunize against feline panleukopenia and canine distemper.

By and large this exercise in dialogue and co-operation and mutual respect has worked extremely well. Problem areas still exist where, for example, members of the "priviledged" public, having been spoilt over the years by getting subsidized veterinary treatment through A W O's, now retaliate when denied access to this source of cheap veterinary service by withdrawing all assistance or monetary help and becoming antagonistic. Another problem is that some A W O's still insist, as a condition of sale, that their adopted animals be returned to them for spaying and thus exlude the new owners' practitioner.

With the continued liaison and goodwill at local, regional and national levels, these problems will be sorted out and the platitude expressed in the opening chapter of the booklet that "it is hoped that this policy statement of the S A V A on animal welfare activities will serve to promote harmony, understanding and co-operation between the individuals and organizations concerned" will be realised.

LYMPH DRAINAGE OF THE MAJOR JOINTS OF THE PORCINE FORELIMB

G. V. S. TURNER* and J. VAN NIEKERK†

ABSTRACT: Turner G. V. S.; van Niekerk J. **Lymph drainage of the major joints of the porcine forelimb.** *Journal of the South African Veterinary Association* (1980) **51** No. 3 133–135(En) Department of Veterinary Public Health, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843 USA.

The lymph drainage of the major joints of the porcine forelimb was subjected to a critical examination. Commercial India ink was injected into the left elbow joint and right shoulder joint of an experimental pig. The shoulder joint was approached craniolaterally, the injection site being at the cranial border of the tendon of insertion of the infraspinatus muscle. The elbow joint was approached from the dorsolateral aspect. A suitable site for intra-articular injection into the elbow joint was found to be within an imaginary triangle formed caudal to the lateral epicondyloid crest of the humerus, the ventral border of the lateral head of the *M. triceps brachii*, and a line just dorsal to the olecranon. The lymph nodes of the neck and shoulder region were carefully exposed and examined for any discoloration due to the ink. The dissection of the lymph vessels and lymph nodes showed that the *Lnn, axillares primae costae* drained the elbow and shoulder joint after the intra-articular injection of India ink.

INTRODUCTION

A thorough knowledge of the topography and drainage areas of the main lymph nodes in domestic animals is of importance to the veterinarian.

In swine, the observation of the lymph nodes that drain the joints of the limbs are of practical significance, especially at necropsy and during meat inspection procedures. When dealing with the lymphatic system of swine, the lymph nodes draining the joints of the forelimb are often omitted. In order to establish a practical method of examining and demonstrating the lymph drainage of the major joints of the porcine forelimb, the lymph nodes draining these joints were subjected to a critical examination.

MATERIALS AND METHODS

A healthy pig, 3 months of age, was used in this experiment. The pig was sedated with azaperone administered intramuscularly at the dosage rate of 2 mg per kg, and then anaesthetized by inhalation of trichloroethylene.

The skin over the lateral aspect of the left elbow joint and over the point of the right shoulder was shaved and cleaned. Using a 50 mm long 20 gauge needle and a 10 mℓ disposable syringe, 5 mℓ of commercial India ink was injected into the left elbow joint and 6 mℓ of ink into the right shoulder joint.

The shoulder joint was approached cranio-laterally (Fig. 1). With the shoulder joint slightly flexed, the needle was inserted at the cranial border of the tendon of insertion of the infraspinatus muscle. The needle was advanced to penetrate the joint capsule, and synovial fluid was withdrawn before the intra-articular injection was administered.

The elbow joint can be approached from two directions. The needle can either be inserted from the lateral side caudal to the joint capsule or from the dorsolateral aspect. In this experiment the latter approach was adopted (Fig. 2). The joint capsule can be felt lying in an imaginary triangle caudal to the lateral epicondyloid crest of the humerus, ventral to the lateral head of the *M. triceps brachii* and dorsal to the olecranon. The needle was again advanced until some synovial fluid escaped and then the intra-articular injection was performed.

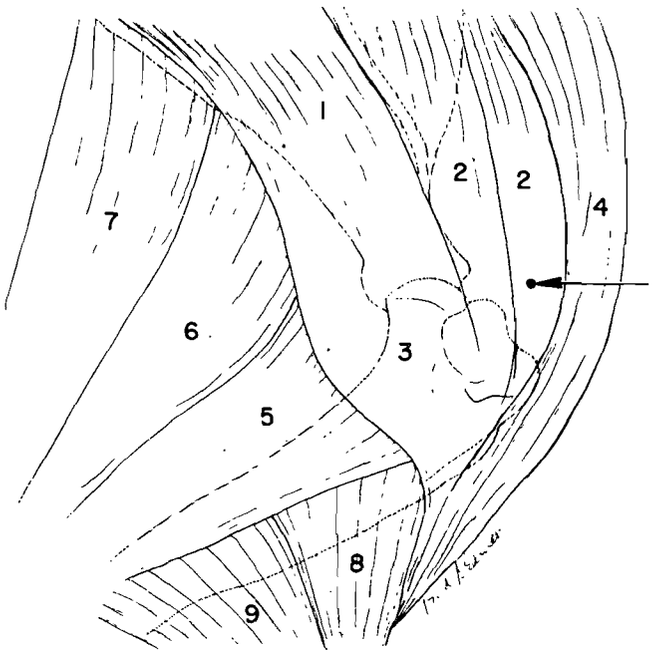

Fig. 1. Lateral surface of the right porcine shoulder (deltoideus and brachiocephalicus muscles removed).
1. *Musculus infraspinatus**
2. *M. supraspinatus*
3. Head of humerus
4. *M. subclavius*
5. *M. triceps brachii caput laterale*
6. *M. triceps brachii caput longum*
7. *M. tensor fasciae antebrachii*
8. *M. brachialis*
9. *M. extensor carpi radialis*

*Nomenclature according to Nomina Anatomica Veterinaria

The pig was slaughtered exactly 4 hours after the ink had been introduced into the 2 joints. The pig was first stunned with a captive bolt, and then the neck blood vessels were severed as close to the head as possible with limited damage to the surrounding tissues. This was done to facilitate the dissection of the lymph nodes.

The lymph nodes of the neck and shoulder region were carefully exposed and examined for any discoloration due to the ink. The following lymph nodes were examined: *Lnn. retropharyngici laterales; Lnn. cervicales superficiales ventrales; Lnn. cervicales superfi-*

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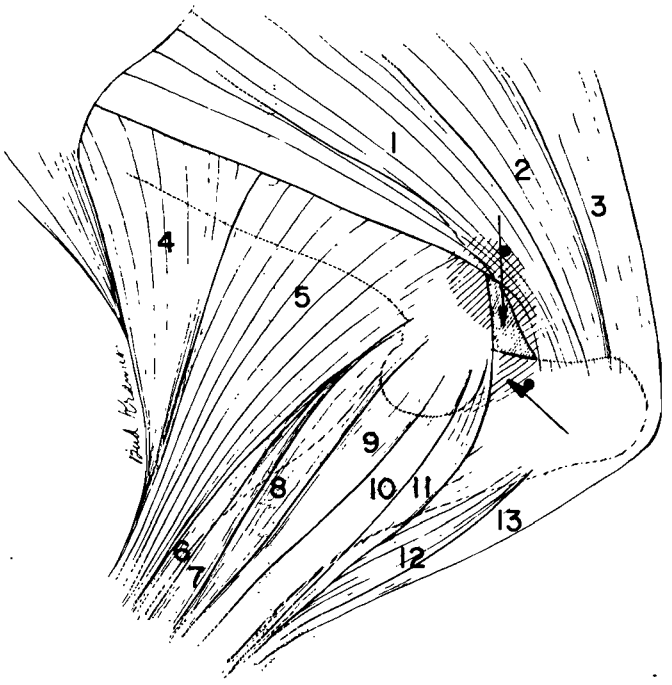

Fig. 2. Lateral surface of the left porcine elbow
1. *Musculus triceps brachii caput laterale**
2. *M. triceps brachii caput longum*
3. *M. tensor fasciae antebrachii*
4. *M. brachialis*
5. *M. extensor carpi radialis*
6, 7, 8. *M. extensor digitalis communis*
9, 10. *M. extensor digitorum lateralis*
11. *M. ulnaris lateralis*
12,13. *M. flexor digitalis profundus*
*Nomenclature according to Nomina Anatomica Veterinaria

ciales dorsales; Lnn. cervicales superficiales medii; Lnn. cervicales profundi caudales; Lnn. axillares primae costae; Lnn. sternales craniales. Any lymph vessels made visible by the India ink were also exposed.

Discolored and macroscopically normal lymph nodes were removed and placed in 10% formalin for histopathological examination. Where necessary, photographs were taken of the lymph nodes and vessels in the neck and shoulder region.

After being fixed in 10 % formalin, the lymph node samples were processed in an automatic tissue processor, embedded in paraffin wax, and cut into tissue sections of 3 μm by means of a microtome. The sections were stained with haematoxylin and eosin. The histological sections made from the lymph nodes were microscopically examined for the presence of carbon particles from the India ink or any other noticeable changes.

RESULTS

Only the *Lnn. axillares primae costae* and their afferent lymph vessels showed any discoloration as a result of the intra-articular administration of the India ink. These lymph nodes were almost pitch black in colour, and the afferent lymph vessels from the shoulder joint to the *Lnn. axillares primae costae* were distinctly black and prominent (Fig. 3). The efferent lymph vessels from the lymph nodes were not noticeable.

The histopathological examination of the discolored lymph nodes showed the following: black irregular shaped carbon particles were noted in the lymph vessels, subtrabecular sinuses and the medullary sinuses. Intracytoplasmic carbon particles were also detectable in macrophages in the medulla. Histological sections of the macroscopically normal lymph nodes revealed a normal microscopical picture.

DISCUSSION

For humane reasons and in order to facilitate the dissection procedures on a fresh cadaver, the pig was stunned and allowed to bleed out properly. With normal slaughter techniques, the sticking wound in pigs is at the entrance to the thoracic cavity. Because the *Lnn. cervicales profundi caudales, Lnn. axillares primae costae* and *Lnn. sternales craniales* are closely situated to the sticking wound, it was thought advisable to sever the neck vessels closer to the head so as to avoid damaging the tissues and lymph nodes in this region

It is apparent from the results of this experiment that the time interval between injecting the ink and slaughtering the experimental animal was adequate. The India ink was still in the injected joint cavity, the relevant afferent lymph vessels were filled with the ink and the *Lnn. axillares primae costae* were markedly black in colour. It is recommended that only a short period should be allowed between intra-articular injection and examining the relevant lymph nodes whether by radiological techniques or by dissection.[1].

The dissection of the lymph vessels and lymph nodes showed that the *Lnn. axillares primae costae* drained the elbow and shoulder joints after the intra-articular injection of India ink. To make certain that the black discoloration of the lymph nodes was due to the injected India ink and not melanin, the lymph nodes were examined histologically. Irregularly shaped black carbon particles were present as opposed to the deep golden brown, uniform size and oval shaped granules of the naturally occurring melanin pigment.[2] The insoluble carbon particles remained in the tissues after the lymph node sample had been processed in the tissue processor.

The *Lnn. axillares primae costae* consist of a group of lymph nodes intimately associated with the first ribs on the cranial aspect and lying just ventral to the *Vena cava cranialis* and dorsal to the point of the sternum on both sides. Each group consists of one large lymph node up to 40 mm in length and up to 4 smaller nodes. The afferent vessels arise from: the muscles ventral to the neck; the cutaneous muscles of the trunk; the muscles of the pectoral limb; cervical vertebrae 3–7; sternum; thyroid; thymus; the muscles around the shoulder; all the bones and joints of the pectoral limb; the skin of the toes. Lymph is also obtained from the cranial mediastinal, cranial sternal and deep cervical lymph nodes. The efferent lymph vessels from the *Lnn. axillares primae costae* drain into the *Ductus thoracis* on the left side and the *Ductus lymphaticus dexter* on the right side. These in turn drain via the larger veins into the venous circulation[3,4].

It can therefore be concluded that the *Lnn. axillares primae costae* drain the elbow and shoulder joints of the porcine pectoral limb. This experiment confirms the findings of others[3,4].

ACKNOWLEDGEMENTS

Bud Kramer, medical illustrator, is thanked for preparing Figures 1 and 2.

Fig 3. Showing India ink in the slightly opened shoulder joint (A) and the afferent lymph vessels (B) draining to the *Lnn. axillares primae constae* (C).

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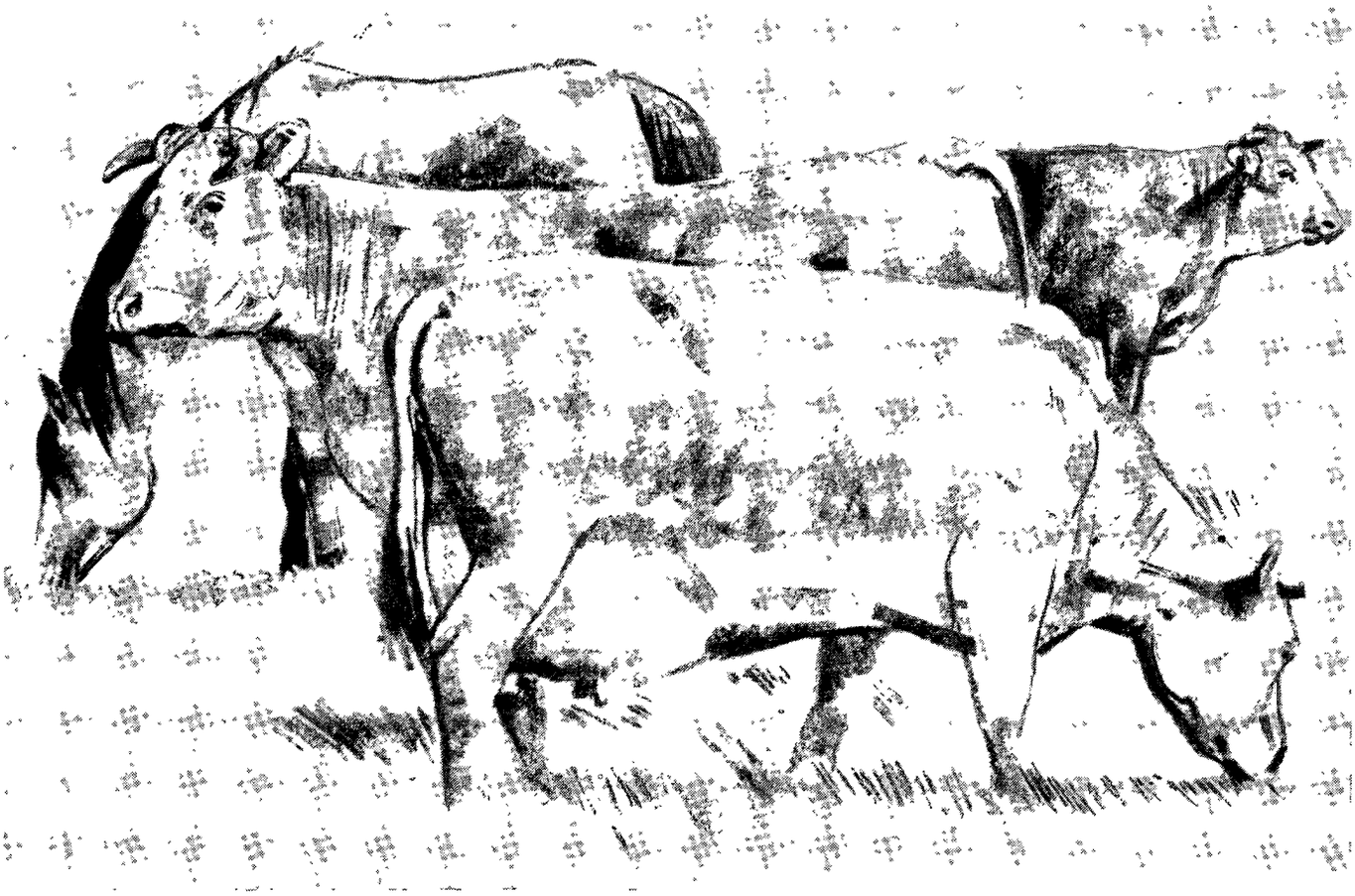

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Veterinary Division

THE ORIGIN AND SIGNIFICANCE OF THE LANGERHANS CELL GRANULES*

W. H. GERNEKE

ABSTRACT: Gerneke W. H. **The origin and significance of the Langerhans cell granules.** *Journal of the South African Veterinary Association* (1980)**51** No. 3 137–142 (En) Dept. Anatomy, Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

Preliminary evidence indicates that the Langerhans cell granules (also known as Birbeck granules) may really be intercellular desmosomal discs of the more superficial layers of stratified squamous epithelium that have been taken up by the interdigitating dendritic processes of the Langerhans cells. They should therefore be considered as phagosomes whose only significance is in indicating that their hosts had traversed one or other of the various types of stratified squamous epithelium of the body. When seen in this light their variable presence in Langerhans cells or occasional presence in melanocytes or keratinocytes can be satisfactorily explained. Although the Langerhans cells form the first line of immunologic defence as antigen detectors, it is unlikely that their granules play any role at all in the immune response.

INTRODUCTION

During the last 2 decades a considerable amount of research has been done on the possible origin[6 16 19 30 31] function and morphology[1 5 8 10 16 17 19 25] of the Langerhans cell granules or Birbeck granules[3] (LCGs) (Fig. 8). This organelle characterises the Langerhans cell (LC), an intra-epidermal, antigen detecting [1 20 21 22] dendritic, migratory cell which was formerly thought to be a senescent melanocyte[6]. In no single instance has a completely satisfactory explanation of the LCG been given. Its postulated origin from the Golgi apparatus or infolding of the plasmalemma[10 23] has never really been accepted.

In an ultracytological investigation of the epithelium of the ruminant forestomach circumstantial evidence was found that these granules are the intercellular desmosomal discs of the distal epithelial cells that have become loosened and subsequently taken up by the interdigitating cell processes of the migratory LCs. These granules should therefore be considered merely as an indication that their host cell had traversed some of the stratified squamous epithelial types of the body. These granules have also been described in melanocytes[31] and even in keratinocytes[2 15 21].

MATERIALS AND METHODS

Specimens of forestomach epithelium were collected from healthy animals slaughtered at the abattoir of the Veterinary Research Institute, Onderstepoort. They were fixed in 4% glutaraldehyde in Millonig's phosphate buffer (pH 7,3) for 24 h at 4°C, washed in the same buffer and post-fixed in 2% osmium tetroxide also in the same buffer (pH 7,3). Two final buffer washes were given. The samples were dehydrated in ethanol and propylene oxide and embedded in Epon 812 in embedding molds for 48 h at 60°C. Ultrathin sections were cut with glass knives on a Reichert OM U 3 ultramicrotome, stained in a saturated solution of uranyl acetate (1 h) and 0,2% lead citrate (4 min) and examined in a Philips E M 301 electron microscope.

RESULTS

The ultrastructural features of the LCs and their characteristic LCGs as seen between the keratinocytes lining the ruminant forestomach have been described in a previous article[9]. Since then it has been noticed that as the keratinocytes mature and move distally, their desmosomal discs appear to be separated from the adjacent plasmalemmas by 2 electron-lucent lines (Fig. 1, 2, 3 & 9) thereby creating the impression of eventually being set free intercellularly. Occasional discs were seen to be absent (Fig. 4) whereas elsewhere pentalaminated structures resembling discs were seen to be free in the intercellular spaces, especially those situated more superficially (Fig. 5). One such disc revealed a distinct vesicle at one end (Fig. 6) as has also been described for the LCG[25].

These desmosomal discs, either free or as part of the desmosome, consist of 3 electron-dense, granular laminae between which 2 electron-lucent laminae are interposed giving a pentalaminated structure (Fig. 1, 2, 3 & 9). Such a structure, when enfolded in a plasmalemma of the processes of the LCs as normally occurs during phagocytosis, would be an exact replica of the LCG. An exhaustive search has failed to provide the final photographic proof but Fig. 5 in an article by Wolff[25] reveals such a pentalaminated structure being taken up.

In this investigation it is therefore postulated that some of these desmosomal discs become free, either through forcible action of the tentacles of the LCs, or as a normal aging or keratinising process and are then, purely by chance, phagocytosed by these tentacles as LCGs. They are initially seen in the tips of the processes of the LCs (Fig. 12) and eventually find their way towards the vicinity of the Golgi apparatus where they are more often encountered[9]. Sufficient circumstantial evidence such as occasional missing desmosomal discs (Fig. 4), similar structures lying free between the keratinocytes (Fig. 5), the presence of LCGs in the tips of the dendritic processes of the LCs in the distal parts of the epithelium (Fig. 12), the exact similarity between the laminae of the discs and the internal structure of the LCGs (Fig. 7, 8 & 9) as well as their variable disc-like structure resembling desmosomal discs in size and morphology (Fig. 7) are the main criteria which at present justify this hypothesis. LCs with LCGs appear to initiate only in stratified squamous epithelium[28], apparently due to the large numbers of desmosomes present.

DISCUSSION

(a) Langerhans cell granule formation

The 2 hypotheses put forward to explain the origin of

*A summary of this paper was presented at the 1980 Annual Congress of the Anatomical Society of Southern Africa held at the University of the Western Cape on the 16 and 17 May 1980.

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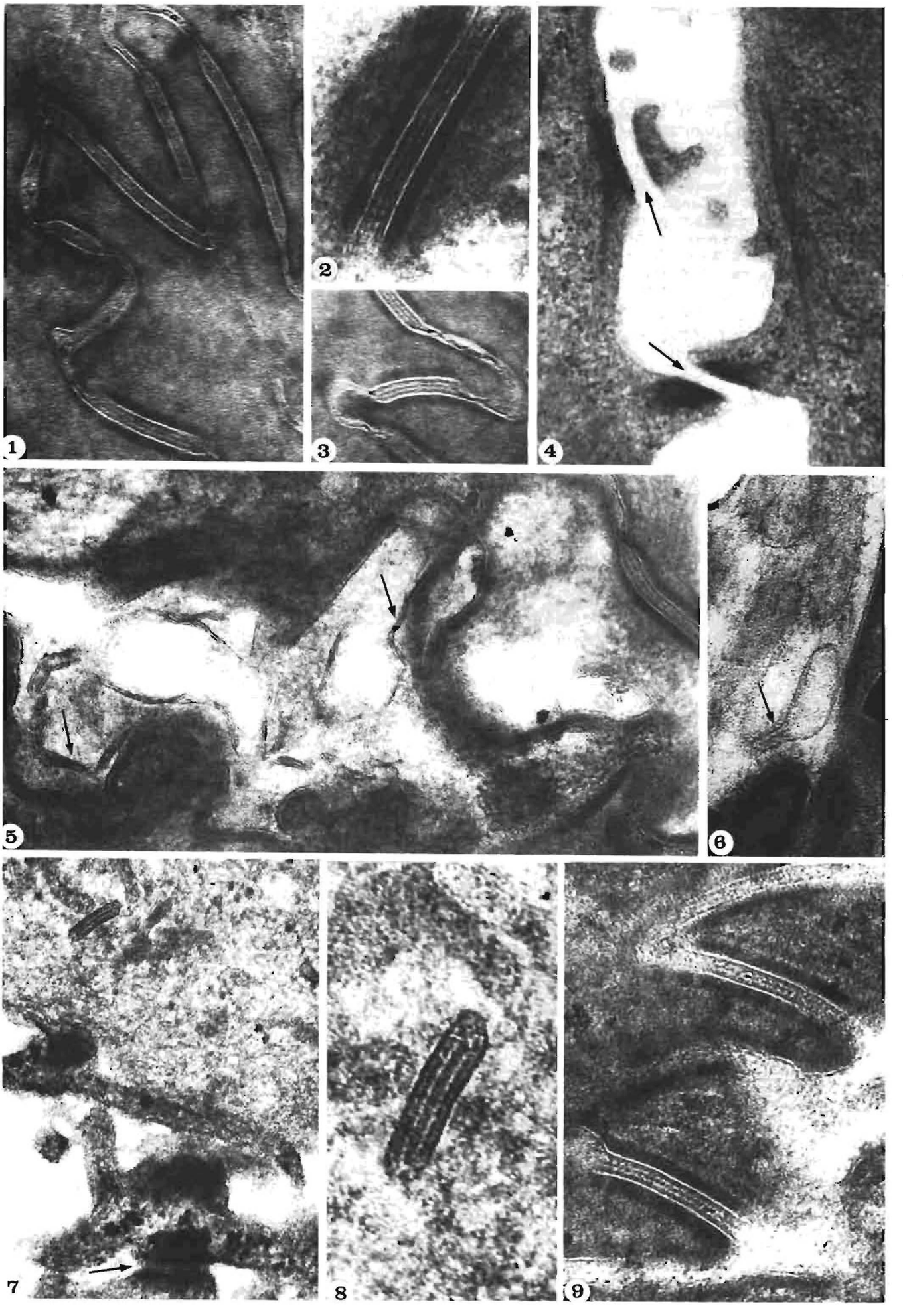


the LCGs namely, from the Golgi apparatus or as invaginations of the plasmalemma[5 7 14 25 26 30] have both one great shortcoming: they do not explain how the pentalaminated core is formed with its 3 granular electron-dense lamellae (Fig. 8). The author's hypothesis namely, that the granules are derived from desmosomal discs which are taken up by the processes of the LCs does not have that shortcoming – the electron dense lamellae of the desmosomal disc (Fig. 9) have a granular appearance exactly resembling the electron dense lamellae of the core of the LCG (Fig. 8) as described by Wolff[25] and Sagebiel & Reed[19].

The desmosomal disc when taken up by the dendritic processes of the LCs is surrounded by the plasmalemma (Fig. 8) and therefore has the nature of a phagosome. This fact that the limiting membrane of the LCG is derived from the plasmalemma is supported by an identical freeze-fracture appearance of the 2 membranes[8]. Wolff[25] as well as Rodriquez & Coarsi[16] found the limiting membrane of the LCG to be in direct continuity with the plasmalemma. There can therefore be no doubt in regard to the origin of the limiting membrane!

There is sufficient evidence from the literature that the LCs have a limited phagocytic ability for exogenous proteins[17 26] and would thus have no difficulty in phagocytosing an occasional desmosomal disc. Rarely phagosomes (thick arrow) are also visible in LCs (Fig. 11). They are even able to take up melanin[4 12 30] and do contain some hydrolytic enzymes[27]. Zelickson[31] has reported that LCs do form granules which in some respects resemble lysosomes (Fig. 11 & 13). If these LCGs are a type of phagosome as all appearances indicate, why are they not digested by hydrolytic enzymes of the lysosomes? The answer to that lies in the fact that the desmosomal disc, primarily attached to the exterior of the cell and apparently partly a product of that cell would not be considered as alien to the cell and would not stimulate autodigestion. The plasmalemma is tightly adherent to the desmosomal disc when it is attached exteriorly and therefore it would also be tightly adherent when taken into the cell (Fig. 8). Lysosomes are only occasionally found in LCs and their presence would not be beneficial to degradation of the LCGs. Neither in the literature nor in this investigation have any lysosomes ever been found to be attached to LCGs. It can therefore be assumed that the LCGs have a very long life in the LCs.

The variation in numbers of LCGs in LCs can be explained by the variable chance uptake of such desmosomal discs as LCGs and their apparently long intracellular lifespan. LCs with a long sojourn in stratified squamous epithelium would obviously have more chance of increasing their number of LCGs while newly arrived LCs would have less or none at all. The presence of LCGs in melanocytes[31] of the epidermis and even occasionally in keratinocytes[2 15 21] can best be explained by the fact that they are taken up from an intercellular position.

In the epidermis melanocytes are present as stationary cells[15] only in the proximal layers of the epidermis, whereas LCs also migrate between cells of the more superficial layers.

The latter have therefore a greater chance of collecting LCGs than the former. Judging from micrographs in the literature it is obvious that the LCs of the epidermis usually contain more LCGs than those found in the ruminant forestomach[9]. This may be related to the degree of keratinisation which is obviously more advanced in the skin than in the forestomach with its moist surface. At this early stage of the investigation it is still uncertain whether the desmosomal discs are forcibly separated by the tentacles of the LCs or whether they sometimes separate as part of the keratinising or aging process. Keratinocytes of the epidermis have been shown to be more phagocytic than the LCs[18 21] and it could therefore be expected that keratinocytes could also take up LCGs if they are available intercellularly. That this seldomly occurs can be explained by the fact that they are stationary cells and that they cannot forcibly loosen the discs as has been postulated for LCs. The LCs are motile and can therefore take up LCGs more easily. Some authors have described LCGs in mesenchymal cells of the dermis[10 28 29]. This must be ascribed to the fact that desmosomal discs which have missed phagocytosis in the epithelium would probably move down and become phagocytosed in the dermis, the same as in the case of melanosomes. Another possibility is that a LC may emigrate from the epidermis and could then be mistaken for a fibroblast.

The desmosomal disc is continuous with the glycocalyx of the keratinocyte (Fig. 3 & 9) and when separated, part of the glycocalyx may come off together with it on one or both sides. They would tend to fuse with each other and this may be responsible for the vesicle usually seen at one end of the LCG[25] (Fig. 6). The desmosomal disc in Fig. 9 gives the impression of being able to form such a vesicle on its left side.

Fig. 1, 2 & 3. In lightly keratinised epithelium of the rumen, desmosomal discs appear to become loosened from the cell processes, varying degrees of which are seen here. Figs. 1 & 3, x 112,000; Fig. 2, x 170,000.

Fig. 4. In rumen epithelium occasional desmosomes were encountered from which the discs were missing (arrows). Presumably they may have been removed by LC tentacles. x 48,700.

Fig. 5. Structures resembling desmosomal discs are seen to lie free between the cells of keratinised rumen epithelium, while the adjacent desmosomes do not show any attached discs. x 105,600.

Fig. 6. In keratinised rumen epithelium a structure resembling a disc with a vesicle at one end was found. If this was taken up by a LC it would form a typical LCG with a vesicle. x 190,600.

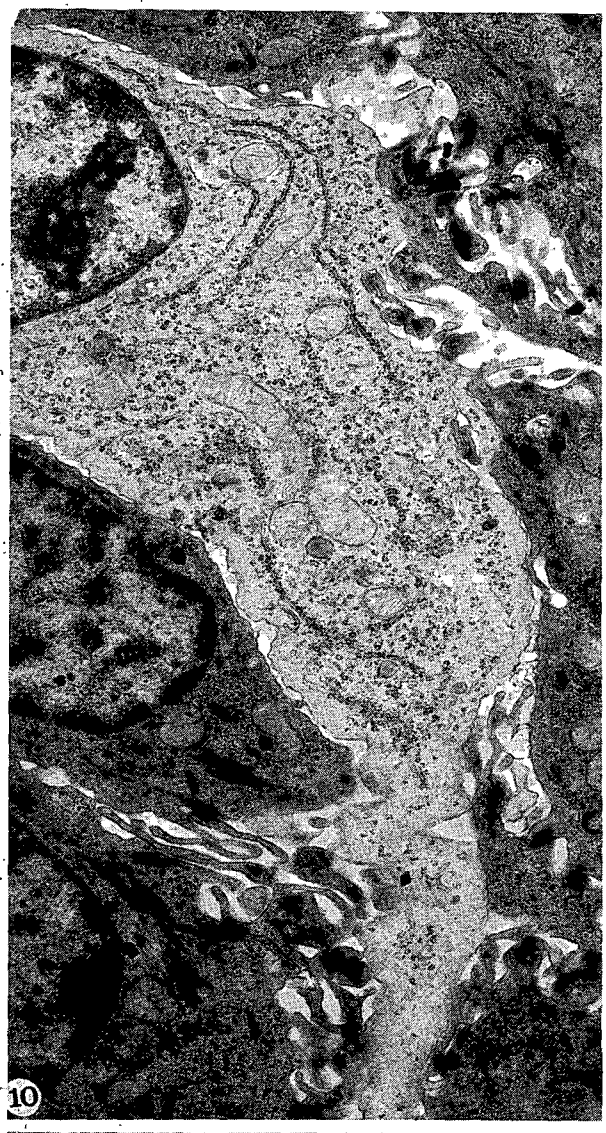
Fig. 7. A desmosomal disc (arrow) and LCG found in a tentacle of a LC can be seen here to be of equal size. x 72,000.

Fig. 8. The LCG in Fig. 7 has been enlarged here to demonstrate the granular nature of its three electron dense lamellae. It is enclosed in a membrane derived from the plasmalemma which here unfortunately does not reveal its trilaminar unit membrane structure. x 209,500.

Fig. 9. Two desmosomal discs can here be seen to have electron dense lamellae with a similar granular appearance as the electron dense lamellae of the LCG in Fig. 8. x 130,000.

(b) Nature of the Langerhans cell

The LC is best considered as a dendritic epithelial macrophage because of its role as antigen detector[1 20 21 22]. In the past macrophages have been considered as scavengers but, since their very important role in the immune reaction has come to light[13], they are presently considered as a heterogeneous group of cells with functions



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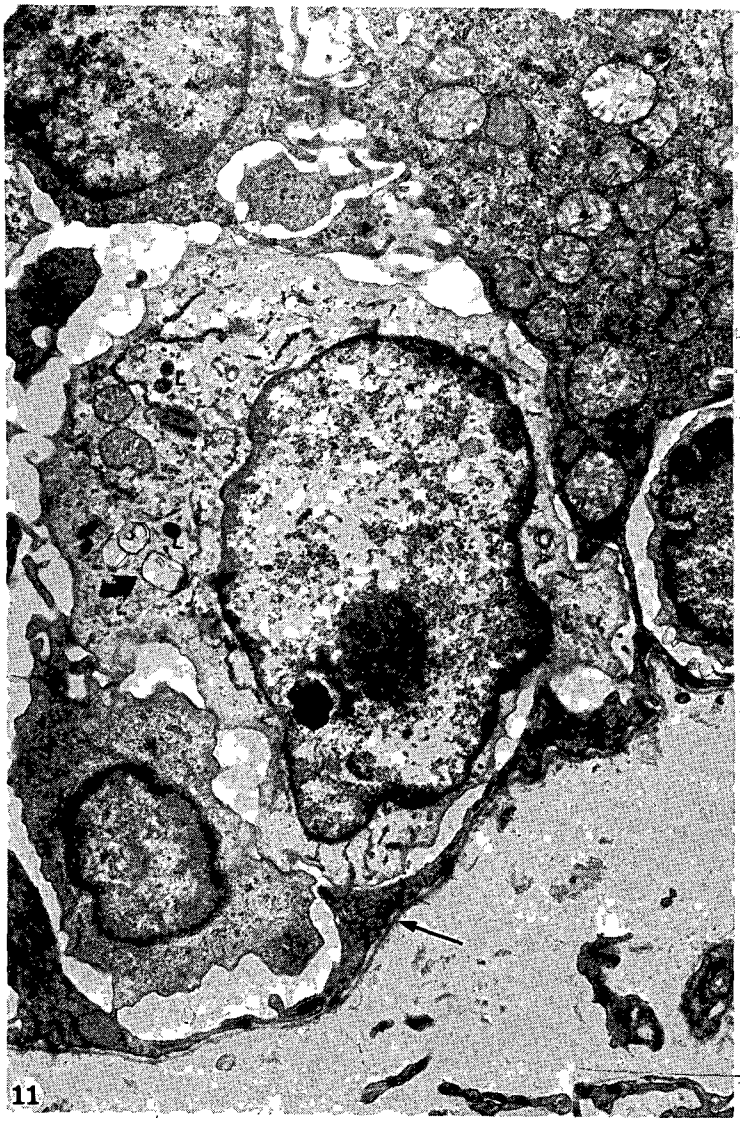

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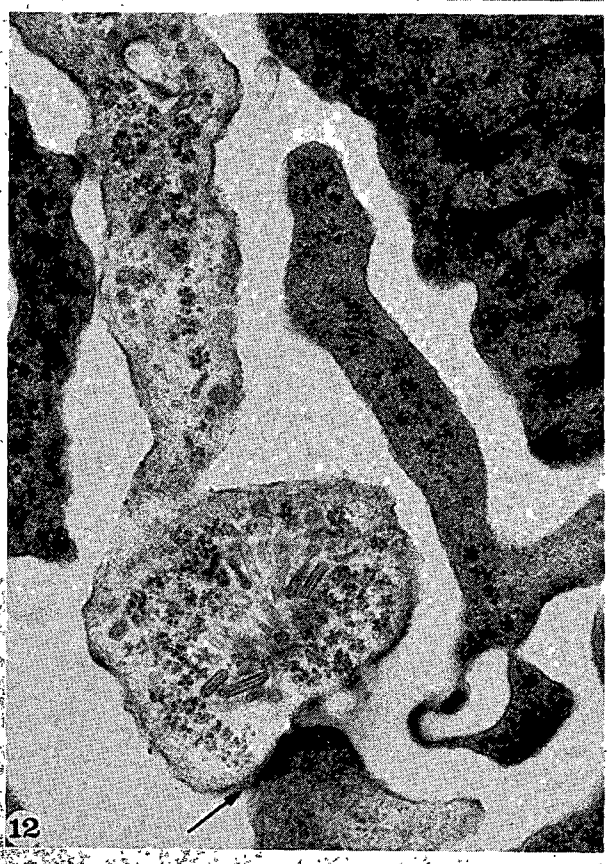

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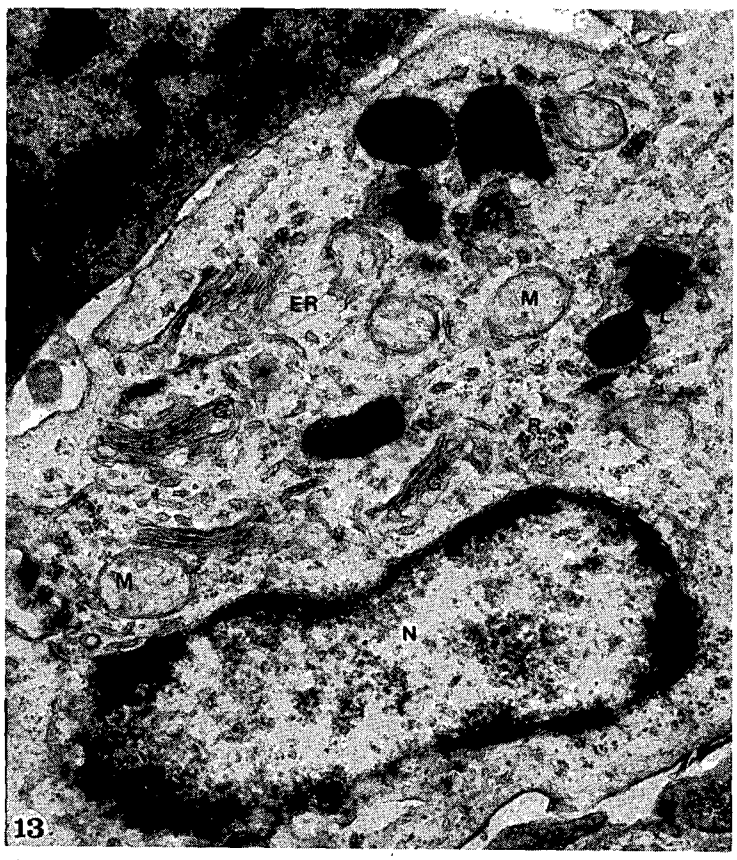

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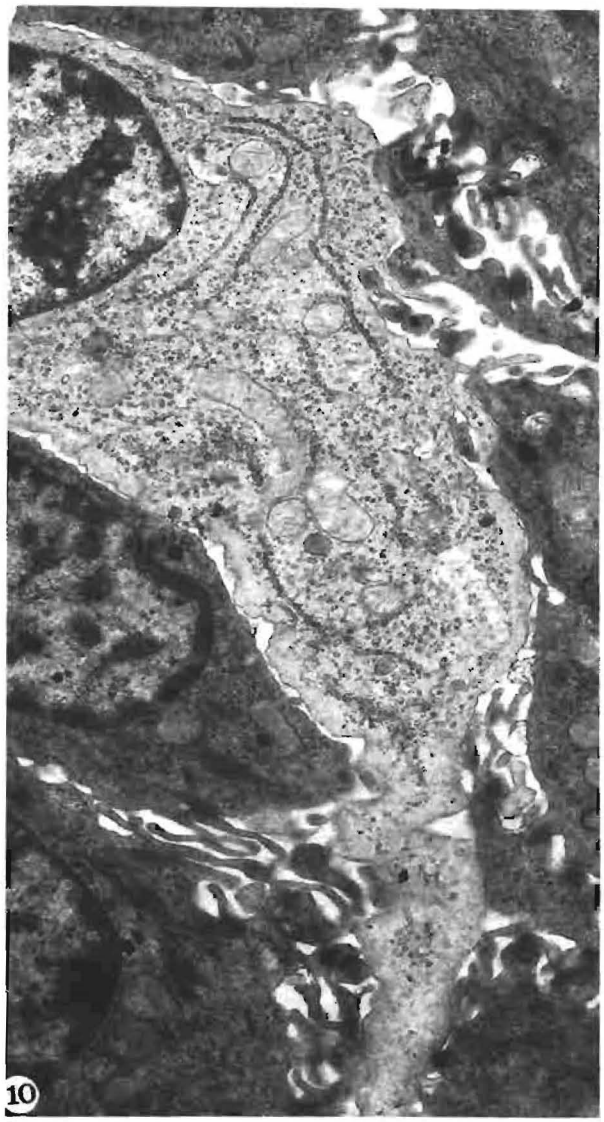

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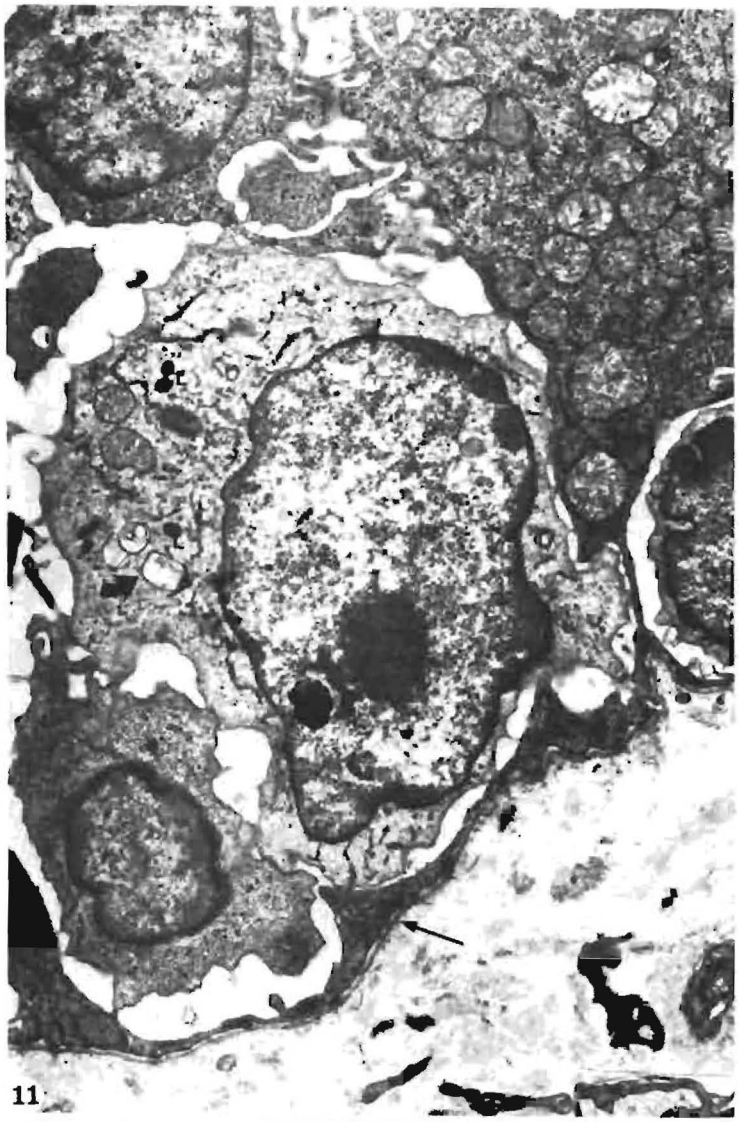

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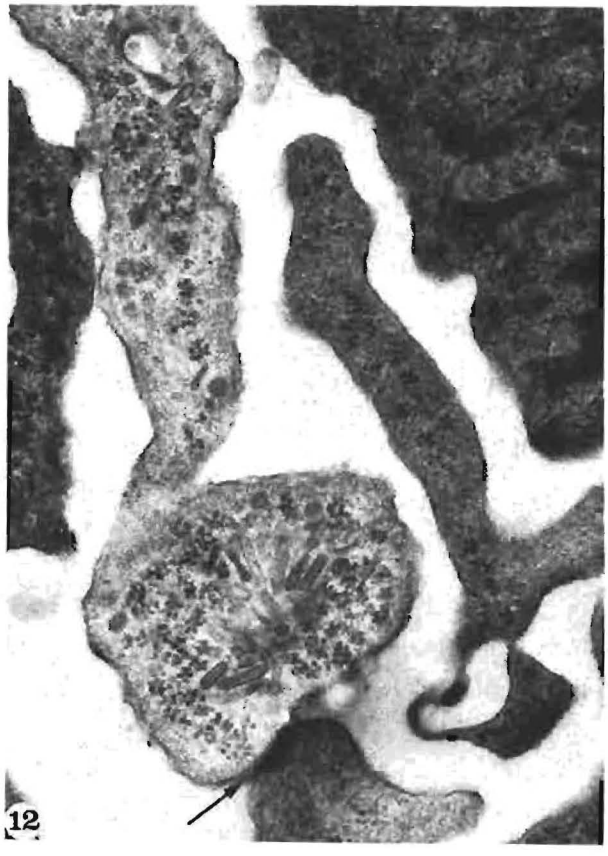

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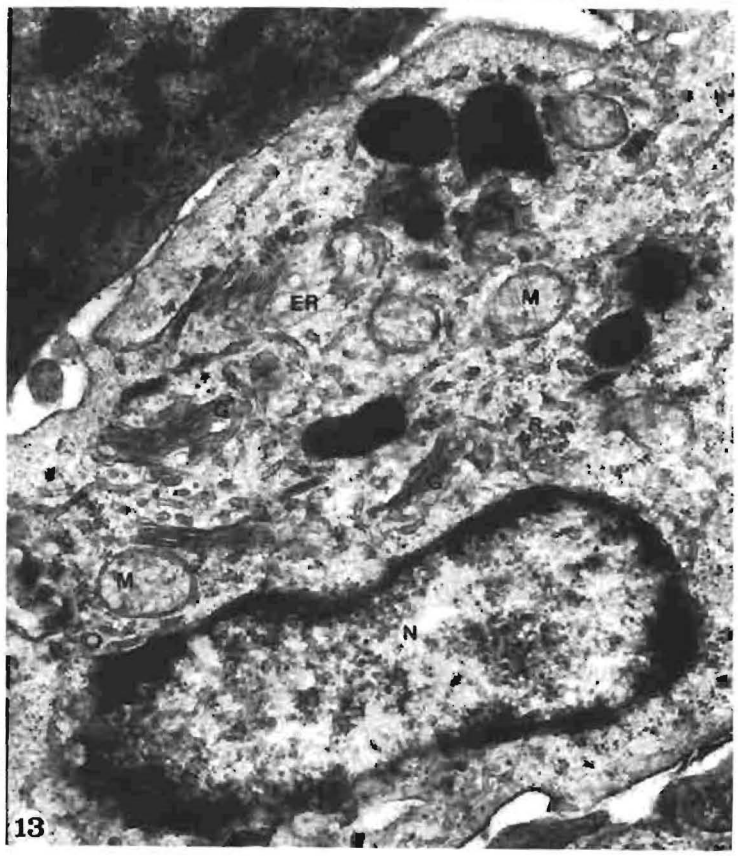

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varying according to their locations and should be considered as the "ultimate guardians of our bodily integrity" (Nelson)[13].

In this respect the LC forms the first line of immunologic defence of the body. It is a migratory cell with dendritic processes penetrating between the epithelial cells (Fig. 10, 11 & 12). Even its nucleus may often be lobulated[9] and could very easily be mistaken for that of a neutrophil. Rowden & Lewis[17] considered the development of such bizarre nuclear shapes "reminiscent of cytotoxic damage" but in this investigation such lobulated nuclei were found in completely normal tissues.

An accepted role of the macrophage is its transfer of allergens to T-lymphocytes which then set up the necessary immune reaction. The LC presumably must have a similar role because small lymphocytes, probably T-lymphocytes are aften seen in contact with them amongst the cells of stratum basale (Fig. 11).

The dendritic processes of the LCs are peculiar in possessing only free ribosomes which presumably must continually be active in supplying proteins for growth of their processes (Fig. 10, 11 & 12.)

The stellate shape of these cells is ideal for movement in the narrow intercellular spaces – the processes can be extended and retracted and are spaced in the epithelium so as to allow processes of adjacent LCs to almost touch each other[27]. This allows efficient coverage of all areas[27]. It is to be expected that they would increase in inflammatory conditions[15 17 23]. However, due to their lack of desmosomes and their ability to migrate, they may be found elsewhere in the body[9 11 24]. Only those epithelial dendritic macrophages which have been fortunate in taking up desmosomal discs as LCGs can therefore carry proof of their sojourn in stratified squamous epithelium with them. After leaving the epithelium and gaining entrance into blood or lymph they would round-up and be seen as monocytoid cells but may still contain LCGs[11 24]. Such cells have been found in lymph nodes and the spleen[11 24].

The possibility that small lymphocytes could change into dendritic LCs[9] on entering the epithelium must not be completely ruled out. Intermediate stages between lympocytes and LCs have often been encountered[9].

In conclusion it may be stated that in contrast to previous hypotheses which attribute origin of the LCGs to the Golgi apparatus or to infoldings of the plasmalemma, the present hypothesis is more acceptable in considering it as being a phagocytosed desmosomal disc for the following reasons: desmosomal discs if enfolded in a plasmalemma resemble LCGs in size, morphology and in shape (Fig. 7, 8 & 9): the variable presence of LCGs in LCs, keratinocytes and melanocytes can be satisfactorily explained as well as their presence in monocytic cells elsewhere in the body [11 24] and the occurrence of LCGs only in dendritic macrophages of stratified squamous epithelium and not in other epithelial types. From present knowledge it can be concluded that the only apparent significance of the LCGs is that they serve as proof of their hosts' sojourn in stratified squamous epithelium and that they in no way could influence the LCs role in the immune response. Their presence is merely incidental and not a regular occurrence.

Fig. 10. A dendritic macrophage forcing its way between the epithelial cells of rumen epithelium. The attenuated tentacle contains only free ribosomes (see also Fig. 12) while the classical organelles are restricted to the main cell body. x 12,300.

Fig. 11. A dendritic macrophage with rarely occurring phagosomes (arrow) and small lysosomes (L). It is also in close contact with a T-lymphocyte on the left and close to another one on the right. They are separated from the basal lamina (arrow) by a thin layer of keratinocyte cytoplasm which on the upper right-hand side contains many mitochondria. x 10,900.

Fig. 12. Some LCGs in the tip of a LC tentacle. A closely adhering desmosome (arrow) is devoid of its disc. The latter may have been taken up as a LCG. x 33,750.

Fig. 13. A dendritic macrophage revealing mitochondria (M), Golgi apparatus (G), strands of smooth endoplasmic reticulum (ER), free ribosomes (R), some electron dense lysosomes (L) and total absence of desmosomes between itself and the keratinocytes. x 33,000.

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BOOK REVIEW

BOEKRESENSIE

MILLER'S ANATOMY OF THE DOG

EVANS & CHRISTENSEN

2nd Ed., W. B. Saunders, Philadelphia, 1979 pp. 1181, Fig. 692 (95 colour). Price approx. R40

This book is a monumental work and a monument to the industry and erudition of its authors. Already the appearance in print of the first edition was an event long awaited by veterinary anatomists. The improved second edition is likewise a major contribution to the literature on the subject. It is planned on a scale rarely before considered in veterinary anatomy and even more rarely put into execution. In 1181 pages and some 700 illustrations the anatomy of the dog is revealed with a precision and in a wealth of detail that makes it the standard authority on the anatomy of this species. Its value is enhanced by superb illustrations and the extensive bibliography. In its attempt to introduce the reader to functional anatomy, the work is highly successful.

The second edition incorporates the official veterinary anatomical nomenclature. The chapter on the developmental anatomy is a valuable addition and most instructive. Certain grave omissions in the first edition have been corrected. They include bone vascularisation, radiological anatomy and the appearance of ossification centres. However, there is still no list of these centres with the times of their fusion. This skeletal topic concerns every clinician.

Technically well produced, the book is recommended to all small animal practitioners.

J. M. W. le Roux

STUDIES ON FELINE BABESIOSIS
2. CLINICAL OBSERVATIONS

G. J. FUTTER and P. C. BELONJE*

ABSTRACT: Futter G. J.; Belonje P. C. **Studies on feline babesiosis 2. Clinical observations.** *Journal of the South African Veterinary Association* (1980) **51** No. 3 143–146 (En) 22 Blue Route, Tokai Road, 7945 Retreat, Rep. South Africa.

Clinical observations were made on 20 experimentally infected and 70 clinical cases of feline babesiosis. The experimental cats showed a remarkable ability to adapt to the disease. Lethargy, anorexia and anaemia were recorded in both groups. Icterus was only occassionally seen. Elevated body temperature was not a feature of the disease. All untreated animals eventually died.

Blood smears revealed increased polychromatophils, Howell-Jolly bodies, nucleated erythrocytes and anisocytosis indicative of a regenerative anaemia. Erythrophagocytosis by monocytic type leucocytes was also observed.

INTRODUCTION

As was pointed out in a review article,[5] little has been published on the clinical picture of feline babesiosis in domestic cats. For this reason we studied the clinical changes in 20 artificially infected and 70 naturally infected clinical cases.

MATERIALS AND METHODS

Experimental Infections

Animals

Twenty healthy domestic cats of mixed breeding were used: 13 mature and 7 younger cats between the ages of 6 weeks and 10 months. They were housed in separate fibre glass cages in a quiet room adjacent to the laboratory. The room was well ventilated and remained at a relatively constant temperature. The cages were cleaned daily. Their diet consisted of commercial tinned cat food (Epol Chicken Chow) and water was always available.

All the cats were inoculated 14d prior to the commencement of the experiment against feline viral rhinotracheitis, calicivirus infection and panleukopenia (Fromm Rhinoid C Leukoid, Salisbury Laboratories).

Of the 20 cats used in the experiment, 12 were splenectomised 7-14 days prior to inoculation with infected blood (Table 1).

Transmission of B. felis

All cats received 1 mℓ of *B. felis* infected blood which was freshly drawn and administered intravenously. The original source of the infection was a clinical case presented by owners living at the Cape Point Nature Reserve. At presentation the blood of this cat had a packed cell colume (PCV) of 12%, and a haemoglobin (Hb) level of 3,5 g/dℓ. It was clinically icteric. The blood smear showed a high level of parasitaemia of *B. felis*. Blood from this animal was administered to Cats 1 and 2 which served as the main sources of infection for the experiment (Table 1).

Data collected

The rectal temperatures and habitus were checked and recorded daily at 09h00.

Blood smears: The glass slides were kept in dichromic acid for 3d, rinsed well in 96% alcohol and rubbed dry with tissue paper to ensure that they were absolutely clean.

The inner ear flap was pricked with a blood lancet producing a small drop of blood. Daily blood smears were dried, fixed in methyl alcohol for 4 min and stained in a fresh 10% solution of Giemsa in phosphate buffer for 60 min (Buffer: 4,68g $NaH_2PO_4.2H_2O$ + 6,55g $Na_2HPO_4.2H_2O$ made up to 1ℓ, pH 6,8). A cover slip was permanently fixed on the slide with Depex (Gurr).

Naturally infected cases

Animals

Seventy clinical cases were presented over a period of 18 months. In all the cases blood smears were positive for *B. felis* and the cats were hospitalized for at least 8 d.

Data collected

The rectal temperatures and habitus were checked and recorded on a daily basis. Using glass slides thoroughly cleaned in alcohol, blood smears were taken on admittance, during the recovery stage and also from animals in which relapses occurred. The smears were fixed in methyl alcohol for 4 min and stained with Stévenel's blue (Centaur Laboratories) for 30 s or occasionally by the Diff Quick (Harleco) method, thoroughly washed in running water and then air-dried.

Blood PCV was determined using standard heparinised Wintrobe tubes and centrifuged at 3 000 rpm for 35 min.

For the purpose of this study the animals were grouped into 3 categories:

Group 1: Cats with PCV greater than 16% (26 cats)
Group 2: Cats with PCV 13 tot 16% (19 cats)
Group 3: Cats with PCV less than 13% (25 cats)

RESULTS

Body temperature and habitus in experimental cats

In both splenectomised and non-splenectomised cats parasites were evident in the erythrocytes from 24 – 48 h after inoculation. In all cases parasitaemia was evident in each of the daily blood smears throughout the course of the experiment.

At no stage of the experiment did any cats show an elevated temperature. The mean (± standard deviation) daily temperature throughout the experiment was 38,2 ± 0,8°C. In general they demonstrated a remarkable ability to adapt to the disease. On most occasions, apart from the obvious anaemia, they showed few signs of disease until the terminal stages when they became inappetant and weak. Even at this near terminal stage some of the cats still ate and this made it most difficult to predict the time of death. Obvious depression and

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Table 1: DESCRIPTION OF CATS, SOURCE OF EXPERIMENTAL INFECTION AND SUBSEQUENT LIFESPAN

Cat	Sex	Age	Origin of infection	Number of days from inoculation until death	Remarks
1	Female	Adult	Clinical case	114	Splenectomised – Main source of
2	Female	Adult	from Cape Point	118	infection for experiment
A	Male	Adult	Cat 1	12	Splenectomised trial run
B	Female	6 weeks	Cat 2	32	Splenectomised trial run
C	Male	Adult	Cat 1	73	Splenectomised experimental
D	Female	Adult	Cat 2	35	Splenectomised experimental
E	Female	Adult	Cat 1	18	Splenectomised experimental
F	Male	Adult	Cat 2	14	Splenectomised experimental
G	Female	Adult	Cat 1	27	Splenectomised experimental
H	Female	Adult	Cat 2	21	Splenectomised experimental
I	Female	Adult	Cat 1	21	Splenectomised experimental
J	Female	Adult	Cat 2	14	Splenectomised experimental
K	Female	Adult	Cat C	45	Splenectomised experimental
M	Pregnant Female	6 months	Cat D	20	Splenectomised experimental
N	Female	4 months	Cat D	53	Non-splenectomised experimental
O	Female	4 months	Cat D	53	Non-splenectomised experimental
P	Female	8 months	Cat D	49	Non-splenectomised experimental
Q	Male	8 months	Cat D	39	Non-splenectomised experimental
R	Female	10 months	Cat D	42	Non-splenectomised experimental
S	Pregnant Female	Adult	Cat C	38	Non-splenectomised experimental

reduced appetite was noted in most of the cats for short spells during the experiment. Clinical icterus was not a feature but at various stages after the start of the experiment the cats voided yellow faeces and diarrhoea was occasionally noted in the earlier stages. When handled for bleeding the cats showed a low tolerance to exertion and those that resisted handling were often exhausted.

No appreciable differences in habitus were recorded between splenectomised and non-splenectomised cats. There were, however, behavioural differences in some cats. For instance, Cats C, 1 and 2 were remarkably alert and even willing to play throughout the entire experiment, Cats Q and R were also normal in habitus until the terminal stage, while the decline in Cat R was very rapid indeed – both of these cats had good appetites three days before death. At the termination of the experiment Cats 2, N, O and P appeared to have reached a degree of stabilisation and it was thought that they could possibly live for many more days if not weeks. They, however, collapsed and died a few days after having been transported for some 70 kilometers to new premises.

On Day 25 Cat S delivered 3 full term weak kittens. At this stage she was inappetant but by the next day was eating well again. Blood smears taken from the kittens did not reveal parasites.

All cats lost condition, some only moderately. All cats either succumbed to the disease or were euthanased in the terminal stage.

Body temperature and habitus in naturally infected cases

In all 70 cats the clinical diagnosis of *B. felis* infection was confirmed by blood smear examination. In general the degree of parasitaemia increased as PCV decreased. Polychromasia, anisocytosis, an increase in the number of Howell-Jolly bodies and the appearance of nucleated erythrocytes were also generally seen.

The cats were admitted with a history of inappetance, lethargy and weakness. Many owners reported that their animals had been missing for a few days and others that theirs' had been ill only for a few days. Some were in a terminal state. During the terminal stages the animals often cried out pitifully as if in pain.

Elevated rectal temperatures (40–40,5 °C) were recorded in only 5 cats of which 4 were in Group 1 and the other in Group 2. Concurrent illness was noted in all of these cats: 2 suffering from respiratory illness and the others from stomatitis and gingivitis. The temperatures of the other 65 cases varied from 36 tot 39,2 °C.

The age of the cats ranged from 4 weeks to 15 years with the vast majority being 2 years and younger. The age of the cats with pyrexia varied from 3 – 10 years.

The clinical picture varied with the severity of anaemia:

Group 1: The cats were usually depressed, anorectic and had roughened coats. Slight palour of mucous membranes was usual and icterus was seen in only 2 cases. The animals in this group were generally not incapacitated by the disease. For instance, one which was admitted for a oöphorectomy operation was found at pre-operative examination to have a PCV of 22% and to be infected with *B. felis*.

Group 2: The cats were usually depressed, weak, anorectic and had roughened coats and pale mucous membranes. Tachycardia and tachypnoea were also noted. Clinical icterus was present in only 7 of the 19 cases. Some cats appeared normal to their owners and in fact 4 in this group were found to be suffering from *B. felis* infection on pre-operative examinations for oöphorectomy.

Group 3: The cats always had very pale to white mucous membranes and were invariably very weak. Clinical icterus was seen in only 4 of the 25 cats. Tachycardia was a feature together with marked dyspnoea on exertion. Twelve of the cats were in a near terminal state and 6 of these died shortly after being admitted. However, some of the animals showed a remarkable ability to accommodate the severe anaemia.

In general, recovery was successful and rapid following the oral administration of Primaquine Phosphate

(ICI) (F. T. Potgieter, Unpublished work, presented at 1975 Biennial Scientific Congress, South African Veterinary Association, Durban). The babesiocidal drugs such as Trypan blue, Euflavine, Berenil, Phenamidine and Terramycin were not as effective both clinically and in reducing the parasitaemia whereas Primaquine generally cleared the blood of parasites withing 48 h. Relapses, which usually occurred 21–45 d after the original infection, were recorded in 13 cases. Five were referred cases which had not previously been treated with Primaquin, 4 relapsed after Primaquine therapy and the other 4 received one or other babesiocidal drug. None of the relapsing cats died.

Three severely affected cats in Group 3 each received a blood transfusion and sodium bicarbonate (2 mmol/kg) followed by Primaquine. This treatment was successful. Another 3 cats, of Group 3 and in a similar condition received only sodium bicarbonate as blood was not available at the time. Two of these cats succumbed to the disease.

A final analysis of the 70 cats showed that 50 recovered, 7 died, 7 were euthanased and 6 could not be followed up as the owners could not be traced.

Within the groups the following occurred:

	Total	Re-covered	Re-lapse	Died	Euthan-asia	Un-known
Group 1	26	16 (62%)	4 (15%)	1*	5	–
Group 2	19	13 (68%)	1 (5%)	2†		3
Group 3	25	13 (52%)	3 (12%)	4	2	3

* Primaquine toxicity
† 1 Primaquine toxicity, 1 respiratory infection

Of the 6 cats in Group 1 that died, 3 were euthanased as they were strays, 1 (a small Siamese kitten) was euthanased at owners request for chronic respiratory disease, 1 (age 5 years) was euthanased for aplastic anaemia and 1 died from Primaquine toxicity.

Of the 2 cats in Group 2 that died, 1 died (age 9 years) from respiratory infection and 1 died after recovery from Primaquine toxicity.

Of the 6 cats in Group 3 that died, 2 were euthanased, 2 died on examination and 2 died during the course of treatment. None of the latter received blood and only 1 cat received Primaquine.

DISCUSSION

Elevated body temperature is not a feature of *B. felis* infection in cats as is the case in babesiosis in other animal species. In fact, in the few ferbrile cases of *B. felis,* some other concurrent illness such as pneumonia was always present. In the terminal stage of the disease subnormal temperatures (±36°C) can be expected.

It is interesting to note that the majority of animals suffering from naturally aquired disease were 2 years and younger. It appears that *B. felis* is endemic in certain areas and one can speculate that many cats may be subjected to the disease at an early age and then develop an "immunity" as cats have been known to harbour *B. felis* without showing any clinical cigns of disease (J F Brownlie 1979, Plumstead, personal communication and G. J. Futter 1979 Retreat, unpublished work). When *B. felis* was found in older cats concurrent illness was often observed. In dogs infected with *B. canis* it has been shown that a premune state occurs and under certain conditions such as disease or severe trauma, this immunity can be broken down and babesiosis can manifest itself[13]. The same situation may occur in cats.

When cats are transferred from an area where *B. felis* does not occur to an endemic area they often soon become infected. The age of these animals naturally varies. The oldest cat found to be infected with *B. felis* (PCV 12%) was 15 years old. This cat was a recent arrival in Fish Hoek (endemic area) from Rhodesia where *B. felis* has not been reported to occur. It is obvious then that susceptibility, immunity and the distribution of the disease warrant futher investigation.

The clinical picture of feline babesiosis under experimental conditions is that of progressive anaemia, lethargy and anorexia with a final moribund state leading to death.

The clinical appearance of the natural cases varied according to the severity of the disease. Obviously anaemic animals do not pose a problem as a blood smear can be used to confirm the presence or absence of *B. felis.* However, in many cases apparently normal animals with either a history of only anorexia or mild anaemia were found to be infected. This emphasizes the value of a blood smear examination during routine clinical examination, particularly in the endemic areas.

The cats in this study showed a remarkable ability to adapt to severe anaemia. This ability has also been found in other forms of anaemia in cats although there are individual variations[2 6 14 19 22]. It is interesting to note that when experimental anaemic animals resisted handling they soon became exhausted. Naturally infected cats would be subjected to the everyday "stress" of life and would thus be less able to cope with the anaemia. Many cats with severe anaemia (PCV 9–12%) were hospitalised, treated with Primaquine only, caged in a quiet area and were handled as little as possible. Under these conditions most of the cats were able to cope with the anaemia and made an uneventful and rapid recovery.

It was difficult to predict the time of death in some of the experimental cats. The decline was often rapid (Cats Q and R) and unexpected (Cats 2, N, O, and P). In practice the client is often only aware of the animal's illness at this stage of precipitous decline and in these cases intensive therapy is necessary.

Yellow to orange faeces was a common finding in all experimental and many clinical cases but clinical icterus was not frequently seen (only one of the 20 experimental cats and 13 of the 70 clinical cases).

The parasite can be identified with ease in peripheral blood smears. Giemsa (10%) was found to be an excellent stain but as it should be freshly prepared and buffered and as the staining time is long it is not often used by practitioners. The more common stains used in practice such as Stévenel's blue and Diff Quick both proved satisfactory. However, Harvey[8] states that better results are generally obtained when staining cat blood smears with longer (Giemsa) rather than shorter methods (Diff Quick).

With Stévenel's blue better results were obtained by staining for 30s only and then thoroughly washing the smear. If the smear was overstained the parasite was not as easily identified, especially in cases where low levels of parasitaemia were present.

With Diff Quick it was found that better results were obtained by staining for 15 s in Solution No 3. This resulted in a more pronounced basophilic staining of the parasite. Single signet ring or round forms with va-

rying disposition of the chromatin were the most common forms of the parasite seen. Pear-shaped forms (double and single) were also a common occurrence. The typical Maltese cross forms were not always seen but became more evident when the parasite was apparently undergoing active division. It was interesting to note that the naturally infected cat with the highest PCV (40%) had the largest number of Maltese cross forms.

Davis[3] reported a maximum parasitaemia of 1% of red blood cells of experimental non-splenectomised cats which had been infected with parasites from the blood of a *B.felis* infected Sudanese wild cat. In South Africa, however, Jackson & Dunning[11] and Robinson[16] reported a high degree of parasitaemia in clinical cases. In this study, parasitaemias of 50% and more were frequently seen in clinical cases and the possibility exists that a different protozoan or at least a more virulent strain occurs in South Africa[11].Further investigation concerning this aspect is obviously required.

In clinical cases a PCV of about 15% and a high degree parasitaemia with signs of active division of the protosoa should be considered as an indication of a possible rapid decline and these cats should be closely observed.

In most instances evidence of a regenerative bone marrow response to the anaemia was manifested by an increase in the number of polychromatophilic cells. Reticulocyte counts were not performed, but in a recent article Alsaker et al[1] demonstrated that the aggregate reticulocyte count closely corresponds to the polychromatophilic erythrocyte count. Increased number of Howell-Jolly bodies, anisocytosis and numerous nucleated erythrocytes were also seen. These changes were also reported by Jackson & Dunning[11] and Robinson[16].

Howell-Jolly bodies are frequently found in the erythrocytes of clinically normal cats and increased numbers can also be expected after splenectomy[17]. Although mild anisocytosis is considered to be normal in the cat, marked anisocytosis, increased Howell-Jolly bodies, the appearance of nucleated erythrocytes and the presence of polychromasia can be considered to be signs of active erythropoiesis[15 23]. However, the presence of nucleated erythocytes in the presence or absence of regenerative signs can be a misleading indication of erythropoiesis[8 15 18]. Similar signs of active erythropoiesis have been reported in both experimental[9 20] and clinical feline haemobartonellosis[4 10 21].

Erythrophagocytosis by monocytic type cells was frequently seen in both the experimental cats and the field cases, both infected and non-infected erythrocytes being phagocytosed. The relative importance of erythrophagocytosis versus intravascular haemolysis in the development of anaemia in feline babesiosis has yet to be investigated. In studies on feline haemobartonellosis, Harvey & Gaskin[9] and Maede & Hata[12] attached more importance to the role of erythrophagocytosis than to intravascular haemolysis in the development of anaemia.

Blood smears of the kittens of Cat S taken a few hours after birth were negative for *B. felis*. Harvey[8] reports a similar finding with *Haemobartonella felis* but Harbutt[7] found kittens to be positive for *H. felis* 3 h after parturition.

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THE RELEVANCE OF DIFFERENT TEST METHODS FOR THE EVALUATION OF TICK CONTROLLING SUBSTANCES

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ABSTRACT: Stendel, W. **The relevance of different test methods for the evaluation of tick controlling substances.** *Journal of the South African Veterinary Association* (1980) **51** No. 3 147–152 (En) Institut für Chemotherapie, Bayer Ag, 56 Wuppertal – 1, W. Germany.

Results of comparative investigations of different in vitro and in vivo test procedures are reported. The aim of the investigations was to determine which of the test methods gives the best estimate of the activity against ticks under field conditions when used for active ingredients of varying chemical constitutions and different types of action.

The data obtained indicate that results of in vitro test methods using tick larvae differ with the test procedure employed and the chemical class of the active ingredient. Therefore, in many instances these test methods do not allow extrapolation to practical conditions. In vitro tests employing engorged female ticks are much more reliable for most classes of compounds. However, there still are groups of compounds for which this test furnishes incorrect predictions. A better indication of the potency of a compound is therefore obtainable using in vivo test procedures. An economic and reliable method for the selection of prospective active ingredients is the mini-dip method using the specific host animal. This method provides not only an indication of the future concentration to be recommended but also provides information on the different types of activity of compounds active against ticks, i.e. tickicidal effect, paralysis, detachment, inhibition of development. Such observations are difficult or even impossible when in vitro methods are employed.

The predictive value of the mini-dip method has been established for tick controlling substances of widely varying chemical constitution like organophosphorus compounds, carbamates, thioureas, diamidides, thiazolines and synthetic pyrethroids.

INTRODUCTION

A necessary pre-requisite for the development of new tickicides is the employment of suitable test procedures. These test pocedures should have a high predictive value with regard to the activity of a compound in the field, they should allow testing of large numbers of compounds and they should also be as economical as possible.

Several known test procedures were compared with respect to their applicability to different problems and their predictive value. The different test procedures have been evaluated with the main objective to compare the respective value of the results obtained to predict the outcome of field tests. The following problems should be answered in the development of new prospectively useful tickicides.

1. Which test is best suited for the discovery of new active principles providing at the same time exact predictions of the new compound's efficacy in the field?
2. Which test is best employed to assess the role of resistance a certain compound is likely to encounter in the field?
3. Which test allows detection of new modes of action of tickicides?

MATERIALS AND METHODS

The tests were selected for comparison from a number of tests described that all have proven their value in special investigations. The four tests which were compared are:

1. In vitro packet larvae test described by Stone and Haydock.[5]. This test procedure has been developed in Australia and is used as a screen to detect resistant strains of ticks.
2. In vitro sandwich larvae test developed by Shaw[3]. This test is employed as a screening test and for detecting resistant tick strains.
3. In vitro adult test using engorged females described by Stendel & Andrews[4]. This test is widely used as a screening test for substances active against ticks.
4. In vivo mini dip test described by Downing et al[1].

The basic outlines of these methods are as follows:

For the *packet larvae test* a solution of pure active ingredient in a mixture of chloroform and olive oil was used. Pieces of filter-paper are soaked with the required concentration of the active ingredient. Following the evaporation of the solvent little envelopes or packets are folded which are then used to confine 18 day old tick larvae. After 24 h in an air-conditioned room (27°C, 90% relative humidity) the numbers of dead and live larvae are recorded.

Aqueous dilutions of formulations of the test compounds are used for the *sandwich larvae test.* Ten mℓ of the required dilution is pipetted on a circular filter-paper sandwich with tick larvae contained in a Petri dish. This larvae sandwich is left for 10 min and then the larvae are transferred into a clean filter-paper bag with a jet of compressed air. After 24 h the numbers of dead and live larvae are recorded.

For both in vitro larvae tests it is important to use larvae of the same age and to strictly standardize other external factors like temperature, relative humidity and the time of day used for treatment of the larvae.

For the in vitro *adult test* engorged female ticks are immersed and agitated in a solution of the test compound for 1 min. The solutions are obtained by dilution of the formulated active ingredient. After a 5 min draining period the ticks are transferred into plastic beakers, the bottoms of which are covered with filter-paper disks. After 24 h the ticks are stuck on adhesive tape and stored at 27°C and 90% relative humidity. Inhibition of egg laying of the treated ticks is taken as an indication of activity of the test compound.

The in vivo *mini dip test* is conducted in a manner similar to that described by Downing etal[1]. Cattle are clipped in such a way that 12–20 patches of 100 mm diameter of undisturbed hair are left along the back of the animals. They are then repeatedly infested with tick larvae (12 times with 2000 larvae each, every other day). The ticks congregate preferably in the patches of long hair where they attach and all developmental stages (larvae, metalarvae, nymphs, metanymphs and adults) are present 21 d after the first infestation. The compound to be tested is now applied to one of the tick

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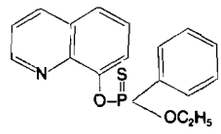
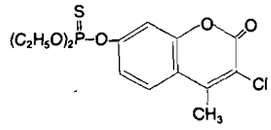
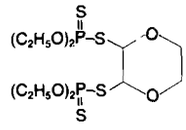
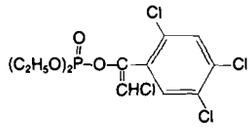
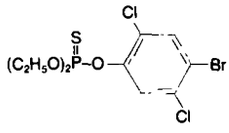
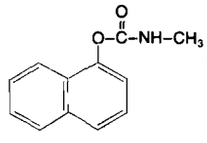
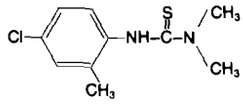
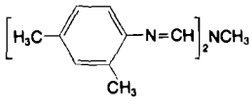
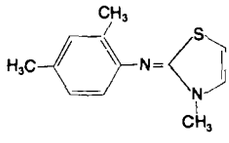
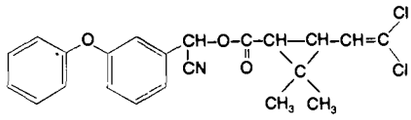
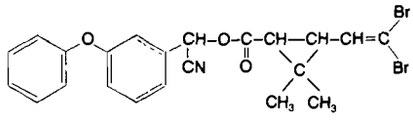
Table 1: COMPOUNDS USED IN THE TESTS

Generic name	Registered trademark of (name of company)	Structure	Recommended conc. ppm a.i
Quintiophos	Bacdip® (Bayer)		200
Coumaphos	Asuntol® (Bayer)		500
Dioxathion	Delnav® (Hercules)		500
Chlorfenvinphos	Supona® (Shell)		500
Bromophos-ethyl	Nexagan® (Celamerck)		750
Carbaryl	Sevin® (Union Carbide)		2000
Chloromethiuron	Dipofene® (Ciba-Geigy)		1800
Amitraz	Taktic® (The Boots Co.)		250
Xymiazole	Tifatol® (Ciba-Geigy) in other countries Besuntol® (Bayer)		300
Cypermethrin	Barricade® (Shell)		175
Decamethrin	Decis® (Roussel-Uclaf)		100

® = Registered Trade Mark of the respective company

infested patches using the so-called mini dip apparatus. This is a cup-like vessel holding 200 mℓ. The time of contact allowed is 1 min during which the apparatus is carefully agitated to ensure wettening of all ticks. After draining of the dip fluid an open gauze bag is glued onto the skin around the patch of hairs using a non-irritant adhesive. Finally the gauze bag is tied off at the other end. The effect of treatment is assessed by counting the numbers of adult ticks that develop each day for a 21 d period. Knowing the time the different developmental stages require to reach maturity one can calculate the percentage efficacy of the test solution on the individual developmental stages.

Selected representatives of tickicides were chosen from different classes of compounds in order to assess the reliability of the 4 test procedures.

The following compounds were included in the comparison:

Organophosphorus compounds: Quinthiophos, Coumaphos, Dioxathion, Chlorfenvinphos, Bromophos-ethyl

Carbamate: Carbaryl

Detaching agents; Chloromethiuron, Amitraz, Xymiazole

Synthetic Pyrethroids: Cypermethrin, Decamethrin.

The recommended concentrations of the different compounds as stated by the respective drug houses has been chosen as the base line for the presentation of the data (Table 1).

RESULTS

The experimental data used for the comparisons are ED_{99} values which were computed from the results of the different tests using the probit transformation[2]. A representation of the data was chosen that indicates the experimental results as multiples or fractions of the recommended concentrations. Fig. 1 shows that the ED_{99} values for organophosphorus esters and carbamates diverge more or less markedly from the recommended concentrations. The results of the larval tests diverge even more from the recommended concentrations than those provided by the adult test. The packet test tends to give ED_{99} values higher and the sandwich test values lower than the recommended concentrations. While the values obtained with the 3 in vitro tests on average differ by one order of magnitude in both directions, it is especially noteworthy that, e.g. Dioxathion and Chlorfenvinphos are effective at very low concentrations in the sandwich test and that Bromophos-ethyl and Carbaryl require fairly large concentrations to be effective in the adult test.

The results provided by the mini dip test come very close to the recommended concentrations for the compounds selected from this chemical class. They compare reasonably well to the concentrations required for field applications.

The results with another group of tick controlling substances which includes the thiourea compound, Chloromethiuron, the diamidide Amitraz and the thiazoline Xymiazole are summarized in Fig. 2. Again deviation of the experimentally determined ED_{99} values from the recommended concentrations are obvious and they are noticeably larger than with the organophosphorus compounds. Especially striking is that very high concentrations of Chloromethiuron are needed in all 3 in vitro tests but not in the in vivo mini dip test which yelds results that compare best with recommended concentrations. Amitraz, especially in the sandwich test, and Xymiazole, especially in the packet test, give large deviations from the recommended concentrations.

Finally, 2 representatives, Cypermethrin and Decamethrin, of the new class of synthetic pyrethroids have been evaluated (Fig. 3). In contrast to the other classes

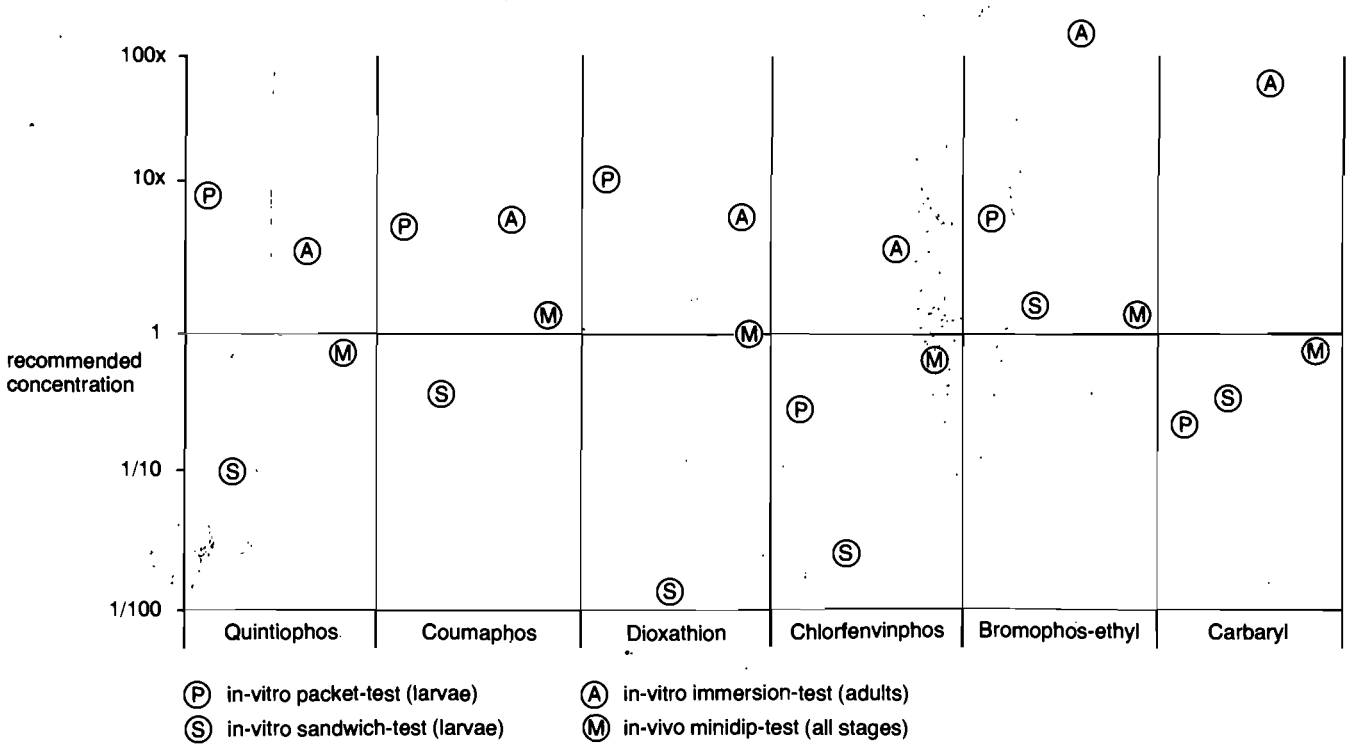


Fig. 1 *Boophilus microplus* (sens. Yeerongpilly-Strain)/Organophosphorus compounds. Results of different tests (ED_{99}) compared with the recommended concentration

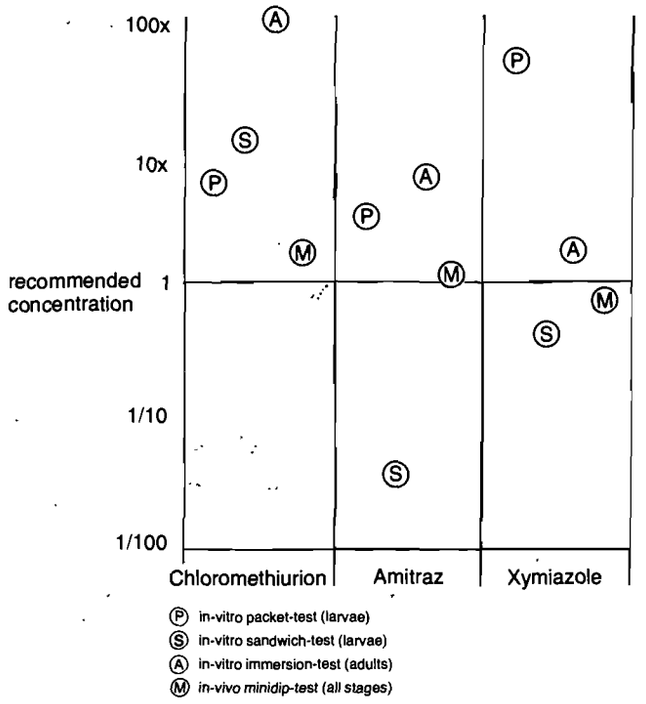


Fig. 2 *Boophilus microplus* (sens. Yeerongpilly-Strain)/Thiourea, Diamidide, Thiazoline. Results of different tests (ED_{99}) compared with the recommended concentration

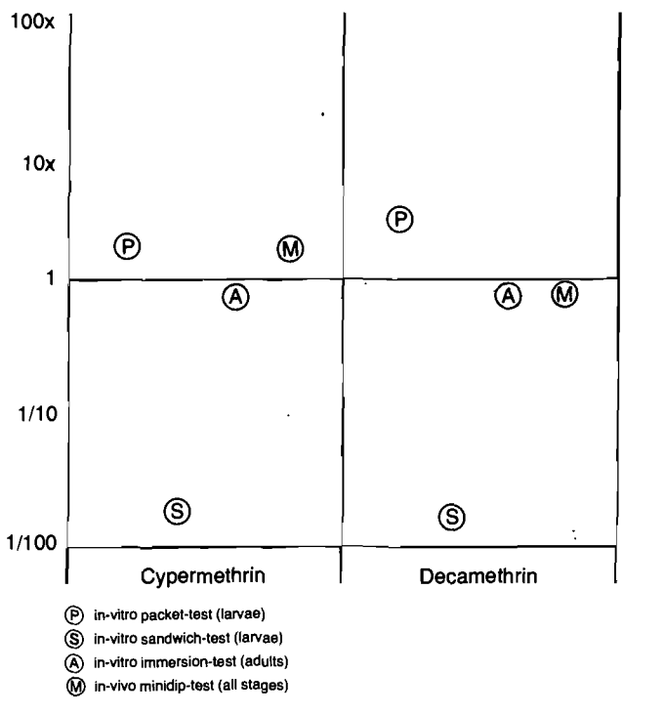


Fig. 3 *Boophilus microplus* (sens. Yeerongpilly-Strain)/Synthetic Pyrethroids. Results of different tests (ED_{99}) compared with the recommended concentration

of tickicides both compounds display only minor deviations from the recommended concentrations in the packet test and the adult test. Both substances however are effective in very low concentrations in the sandwich test. It is again the mini dip test that gives the best approximation of the field situation.

In another series of experiments other aspects of the mini dip test were examined. The aim now was to determine whether the mini dip test would also show its value as a tool for the detection of resistant tick strains. As an example Table 2 shows values obtained for Coumaphos using 2 Australian strains, the OP sensitive Yeerongpilly strain and the OP resistant Biarra strain. It is obvious that neither the ED_{50}, the ED_{95} nor the ED_{99} values as determined by in vitro larvae tests or by the adult test can be usefully employed to assess the real importance of resistance under field conditions. Once again, it is the mini dip test that best approximates the data obtained in the field.

This led us to conduct further tests using the mini dip test and different strains of sensitive and resistant ticks.

The following strains were compared: The OP sensitive Yeerongpilly strain and the OP resistant Biarra strain of *Boophilus microplus*, the OP sensitive Onderstepoort strain and the OP resistant Eldoret strain of *Boophilus decoloratus*, the Chloromethiuron-, Amitraz-, and Xymiazole sensitive Biarra strain and the Glastonbury strain of *B. microplus* which is less susceptible to Chloromethiuron, Amitraz and Xymiazole and, finally, the pyrethroid sensitive Biarra strain of *B. microplus* and a DDT- and pyrethroid resistant Malchi type field strain of *B. microplus* from Australia. The individual results are shown in Tables 3–6. It is apparent that the mini dip test yields results that are in close agreement with those obtained in a handspray test for all trains. In each instance a reliable indication of the degree of resistance can be derived from the mini dip test data.

For instance it is apparent that in the mini dip test and in parallel also in the hand spray test ticks of the Biarra strain are highly resistant to Quinthiophos, Coumaphos, Dioxathion and Carbaryl and less resistant to

Table 2: *BOOPHILUS MICROPLUS*(OP-SENS. YEERONGPILLY-STRAIN AND OP-RES. BIARRA-STRAIN)DIFFERENT TEST-METHODS

Coumaphos: ED_{50} $ED_{95}ED_{99}$ in ppm

	test-method	development stage	ED_{50}	ED_{95}	ED_{99}
OP-sens. Yeerong-pilly-Strain	packet	larvae	13	373	1041
	sandwich	larvae	1,1	9	414
	immersion	♀adults	3	264	1596
	mini-dip	all stages	—	—	500
OP-res. Biarra-Strain	packet	larvae	123	1203	3314
	sandwich	larvae	412	45216	316410
	immersion	♀adults	500	27424	144097
	mini-dip	all stages	—	—	>2000

Table 3

Compound	Rec. conc.	Efficacy in % *Boophilus microplus*			
		Sens. Yeerongpilly-Str.		Res. Biarra Str.	
		minidip	handspray	minidip	handspray
Quintiophos	200	99,8	99,97	64,3	49,1
Coumaphos	500	99,7	100	43,7	58,7
Dioxathion	500	99,84	99,98	48,5	55,6
Chlorfenvinphos	500	99,9	100	89,6	82,0
Bromophos -ethyl	750	99,8	100	73,9	79,8
Carbaryl	2000	99,3	99,7	41,4	44,4

Table 4

Compound	Rec. conc.	Efficacy in % *Boophilus decoloratus*			
		Sens. Onderste-poort-str.		Res. Eldoret-Str.	
		minidip	handspray	minidip	handspray
Quintiophos	200	99,8	100	56,7	62,3
Coumaphos	500	99,9	99,7	99,8	99,9
Dioxathion	500	100	99,7	74,3	69,3
Chlorfenvinphos	500	99,9	99,9	99,7	99,9
Bromophos-ethyl	750	99,7	99,6	99,8	99,8
Carabaryl	2000	99,4	99,8	46,7	41,0

Table 5

Compound	Rec. conc.	Efficacy in % *Boophilus microplus*			
		Sens Biarra-Str.		Res. Glaston-bury-Str.	
		minidip	handspray	minidip	handspray
Amitraz	250	99,9	100	77,2	73,6
Chloromethiuron	1800	99,7	99,7	89,0	87,0
Xymiazole	300	99,9	100	95,8	96,3

Table 6

Compound	Rec. conc.	Efficacy in % *Boophilus microplus*			
		Sens. Biarra-Str.		Res. Nalchi-type-Str.	
		minidip	handspray	minidip	handspray
Cypermethrin	175	99,7	99,6	74,8	77,9
Decamethrin	100	99,9	100	75,9	83,4

Chlorfenvinphos and Bromophosethyl (Table 3). Similarly, the Eldoret strain of *B. decoloratus* is more resistant to Quinthiophos, Dioxathion, and Carbaryl than to Coumaphos, Chlorfenvinphos and Bromophos-ethyl (Fig. 7).

Ticks of the Glastonbury strain of *B. microplus* are less susceptible to Amitraz, Chloromethiuron and Xymiazole while ticks of the Biarra strain are fully susceptible to these compounds both in the mini dip and in the hand spray tests (Table 5). Ticks of an Australian field strain resistant to DDT have a cross resistance with certain synthetic pyrethroids. They show a reduced susceptibility to Cypermethrin and Decamethrin when compared to ticks of the pyrethroid sensitive Biarra strain (Table 6).

From these results one must conclude that the mini dip test can be usefully employed to assess the degree of resistance not only to OP but also to non- OP tickicides which a new compound is likely to encounter in the field.

In another series of experiments it was determined whether the mini dip test would also be suitable to test multihost tick species which are of special importance in Africa. As an example we compared the effects of Quinthiophos in the mini dip test and in a hand spray test. (Table 7)

The results obtained indicate that the mini dip test can also be used to forecast the efficacy of compounds against multihost ticks in the field with sufficient accuracy.

Table 7

Species	Rec. conc.	Efficacy of Quintiophos in % different multihost tick species	
		minidip	handspray
A. hebraeum	200	99,9	99,8
A. variegatum	200	100	99,8
R. appendiculatus	200	100	99,9
R. evertsi	200	99,8	99,9
H. truncatum	200	99,9	99,8

Table 8: PREDICTION OF MODE OF ACTION USING DIFFERENT TEST METHODS.

Type of substance	larvae packet	larvae sandwich	adults immersion	all stages mini-dip
Organophosphorus compounds	+	+	+	+
Carbamates	+	+	+	+
Detaching agents	?	?	?	+
Synthetic Pyrethroids	+	+	+	+

+ = Prediction in accordance with findings
? = Prediction doubtful

The third important criterion for the value of a test procedure, besides its potency to predict future recommended concentrations and to assess the degree of resistance, is that it should enable recognition of compounds with new modes of action. Compounds derived from such novel classes carry much more promise for the control of tick strains already resistant to commercially available tickicides than yet another compound of a known group of substances. It is well-nigh impossible to make predictions about new modes of actions. Therefore, a retrospective check was made whether the different modes of action known to us can be recognized using the mini dip test. Table 8 shows the results of this test.

Organophosphorus compounds and the new synthetic pyrethroids kill ticks both in the in vitro larvae and adult tests and in the mini dip test. Recognition of this tickicidal mode of action of these 2 classes of compounds is thus possible using any of the in vitro test procedures or the mini dip test.

Rather different is the behaviour of a group of compounds that in vivo show a detachment type of action like Chloromethiuron, Amitraz and Xymiazole. Recognition of detachment is not possible in vitro. The only observation supplied by either the in vitro larvae tests or the in vitro adult test was a measureable increase in motor activity. Futher it was observed that Amitraz and Xymiazole inhibit oviposition by the adult female already at very low concentrations. Even a skilled worker cannot conclude from these observations that a detachment mode of action is involved. As experience accumulates a conclusion from analogy may become possible. Thus, prediction of the mode of action of these compounds cannot be made with the in vitro tests employed. The mini dip test, however, was once more the test of choice, yielding reliable information on the mode of action.

DISCUSSION

In conclusion, the 4 test procedures will be compared, bearing in mind that what is required for the development of new tickicides are reliable results that have a high predictive value. Further, the cost-effectiveness of the test is not unimportant. The mini dip test is best suited to cater for our 3 main issues: the prediction of the concentration of a new compound that will be required to control one- and multihost ticks, the assessment of the degree of resistance to the new compound that might exist, and the recognition of the mode of action. The mini dip test, that makes use of the host-parasite system, thus allows a quantitative as well as a qualitative assessment of a new tickicide. This test is undoubtedly more expensive than any of the in vitro tests but the additional costs accrued are more than compensated for by the gain in the reliability of the predictions that can be made on the basis of the mini dip data. All 4 test procedures examined are being used in our laboratory as they all have advantages for certain applications. The 2 in vitro larvae tests are used to test for resistance. The in vitro adult test is used for the primary screening programme. The mini dip test is used to select and evaluate prospectively useful tickicides, to obtain a reliable prediction of the recommended concentrations for field application, to assess the degree of resistance and to determine the mode of action of compounds under development.

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BOOK REVIEW — BOEKRESENSIE

AN ATLAS OF SURGICAL APPROACHES TO THE BONES OF THE HORSE

D. W. MILNE & A. S. TURNER
Illustrations by B. Kramer
W. B. Saunders Company, London, Philadelphia & Toronto, 1979 pp. 210 Figs 145, Price £17,25.

In writing this book the authors describe their goal as follows: "... This text is to describe the most atraumatic approach to expose a particular bone".

The book is definately "Specialist material" and will not appeal to the student or general practitioner, but it is a valuable aid to those who do a lot of horse work, particularly if they are engaged in treating competitive horses (racing, polo, eventing and jumping).

This slim volume is appealing in looks, is well bound and handles well: it is clearly printed on high gloss A4 paper. Each surgical approach is illustrated by 3–6 beautiful line drawings on which the relevent parts are distinctly identified. The material is divided into 4 sections, the first of which, Introduction (8 pages), deals with positioning the equine orthopedic patient, aseptic technique, retraction of tissues and a paragraph on the Association for the Study of Internal Fixation. Section Two (120 pages) concerns the thoracic limb and describes an approach to the extensor process of the dorsal aspect of the great metacarpal and first phalanx, 2 other approaches to first phalanx and an approach to the fetlock joint, followed by 3 approaches to the proximal sesamoid bones and 3 routes to the metacarpal bones. In deference to the frequency of chip and slab fractures in the carpus of competitive horses there are described 5 approaches to the bones involved in this joint. Approaches to the olecranon, radius (2), humerus and tuber scapulae are presented to complete the thoracic limb.

Section Three (38 pages) describes approaches to the great metatarsal bone, the lateral matatarsal bone, the tibio-tarsal joint (2), calcaneus and tuber calcis to complete the hock. A lateral approach to the femur and a dorsal approach to the stifle joint are presented. Next the authors deal with the wing of the ilium. Section Four (26 pages) deals with the head. The authors describe in turn the surgical approaches to the frontal and maxillary sinuses, the horizontal ramus of the mandible, the lateral aspect of the maxilla and incisive bones, an oral approach to the incisive bone and finally the frontal and nasal bones.

Appropriate references are provided so that the reader will have a good starting point to all other surgical aspects besides the surgical approach to the affected bone presented by the authors.

It is to be hoped that these authors or their successors will write a second edition of this fine text at some time in the future. One would like to know their view of opening the fetlock joint by severing the colateral ligament. In the present rather formal presentation it would be out of keeping to mention that one requires long instruments to screw horizontal fractures of the proximal sesamoids because the bulbs of the heel otherwise get in the way. The accessory or ulna carpal bone has been omitted, and so too has drilling of the hoof for placing screws for the repair of P III fractures. The vertical ramus of the mandible has been effectively approached in our practice.

One would also like to know which approach to the dorsal spinous prosesses is preferred by Milne and Turner. Inclusion of some of these points in the future editions may be considered worthy by the authors.

The authors are to be congratulated in having achieved their goal and the profession has gained another valuable book which helps to advance our expertise, the service we render to horses in our care and our image as veterinarians.

D. H. G. Irwin

BREINTERATOLOGIE AS GEVOLG VAN TRANSPLASENTALE VIRUSBESMETTING IN HERKOUERS

J. A. W. COETZER

ABSTRACT: Coetzer J A W 1980 **Brain teratology as a result of transplacental virus infection in ruminants.** *Journal of the South African Veterinary Association* **51** No. 3 153–157 (Afr) Veterinary Research Institute, Onderstepoort 0110, South Africa.

Various viruses relating to congenital brain teratology in ruminants are discussed. These include: bluetongue which may be responsible for porencephaly and hydranencephaly; Akabane disease, Rift Valley fever (mouse brain attenuated virus) and Wesselsbron disease (wild-type and mouse brain attenuated viruses) which can give rise to porencephaly, hydranencephaly, micrencephaly and cerebellar hypoplasia, and bovine viral diarrhoea-mucosal disease which results only in cerebellar hypoplasia. Certain foetal, maternal and virological factors that might have an effect on the pathogenesis of viral teratogenesis are briefly reviewed. Mention is also made of the different diagnostic procedures that can be used such as, gross and histopathological examination, virus isolation and serology.

Dit word vandag algemeen aanvaar dat sekere kliniese en subkliniese virusaandoenings van die moederdier teratologie tot gevolg kan hê in die ontwikkelende fetus. Meeste van die teratogeniese virusse veroorsaak dikwels fetale afsterwing maar nie alle virusse wat fetale afsterwing veroorsaak is teratogenies nie. In die verlede kon geen definitiewe etiologiese agente aan die abnormaliteite gekoppel word nie en baie van die afwykings is as oorerflike gebreke afgemaak. Met toenemende belangstelling en navorsing die afgelope 4–5 jaar, het dit duidelik geword dat baie van die teratologiese afwykings en veral breinteratologie dikwels aan een of ander transplasentale virusbesmetting gekoppel kan word[1 2 3 6 10 12 13 16 20].

Breinteratologie soos serebellare hipoplasie (Fig. 1), hidranenkefalie (Fig. 2), porenkefalie (Fig. 3), mikrenkefalie (Fig. 4) en hidrokefalus is van die afwykings wat reeds aangeteken is met verskillende kongenitale virusaandoenings[1 2 3 6 13 20]. Dit is reeds bewys dat virusbesmettings soos bloutong (BT) (wilde-tipe virus en muisbrein geattenueerde virus), slenkdalkoors (SDK) (muisbrein geattenueerde virus) en bees virus diareemukosasiekte (BVD-MS) kongenitale breinteratologie tot gevolg kan hê indien besmetting sou plaasvind gedurende die kritiese periode van breinontwikkeling.

Daar is egter sekere fetale, maternale en virologiese faktore wat in verband gebring moet word alvorens die graad en insidens van die abnormalitiete geïnterpreteer kan word omdat die afwykings gewoonlik etlike maande na besmetting van die fetus eers geopenbaar word.

Fucillo & Sever[5] bespreek verskillende fetale faktore en dui aan dat indien fetale morfogenese nog onvolledig is tydens besmetting, die dragtigheid beëindig kan word in aborsie of die geboorte van 'n dooie dier gepaard met misvorming of degeneratiewe letsels in die

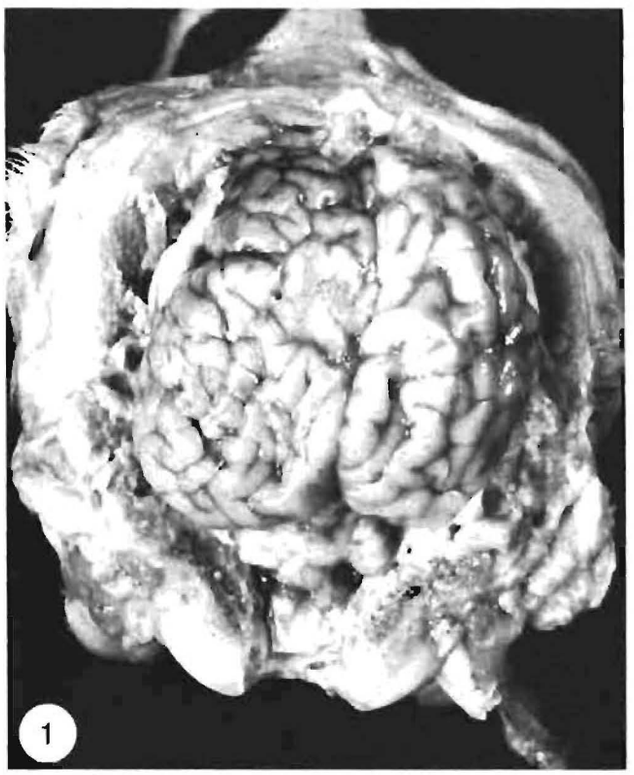

Fig. 1. Brein met opvallende serebellare hipoplasie

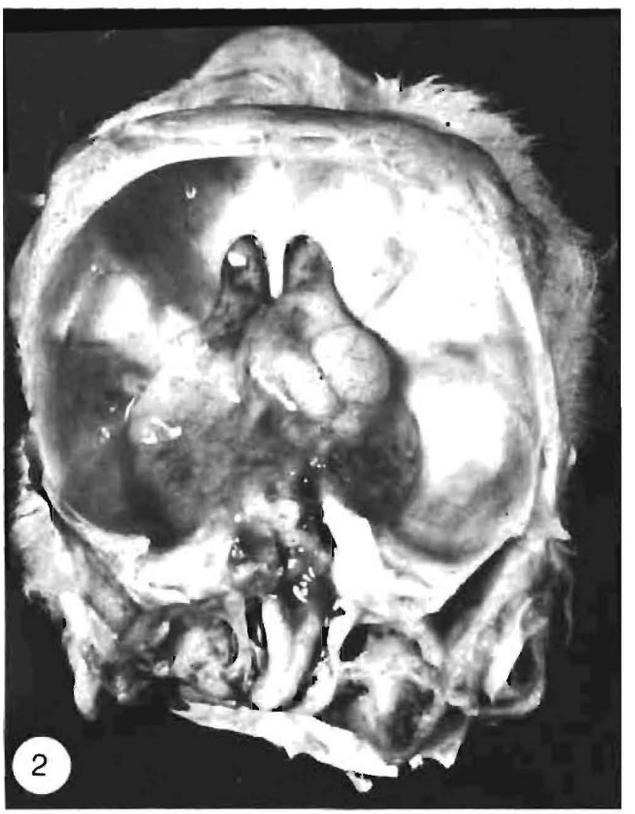

Fig. 2. Hidranenkefalie. Serebrale hemisfere bestaan uit membraneuse vliese en slegs gedeeltes van die midbrein redelik goed ontwikkeld.

Referaat gelewer tydens die Mini-kongres van die Noord-Transvaaltak van die SAVV, Pretoria, 16 Junie 1979.

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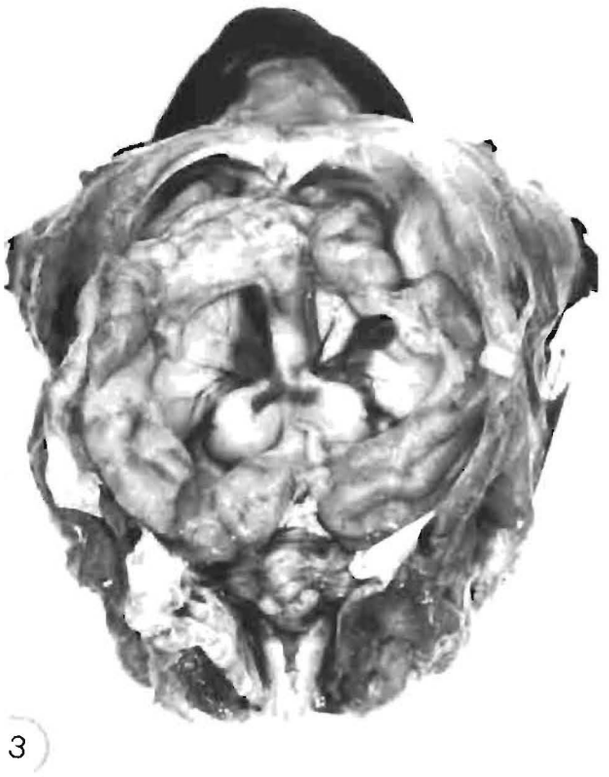

Fig. 3. Porenkefalie in 'n skaaplam a.g.v. die geattenueerde Wesselsbronsiekte virus.

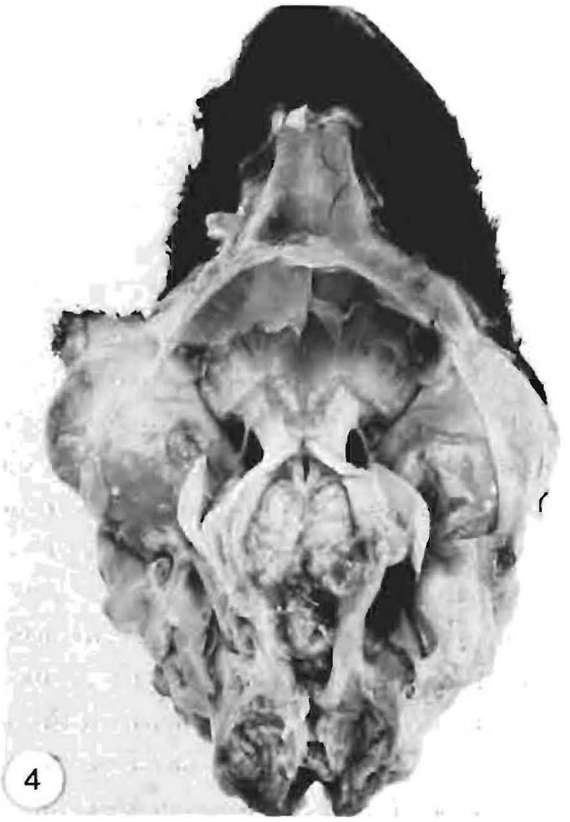

Fig. 4. Skaaplam met uitgesproke mikrenkefalie.

fetus. Hulle stel dit ook duidelik dat waar ontwikkeling reeds volledig is en fetale besmetting dan sou intree geen misvorming plaasvind nie. Dit wil dus voorkom dat daar 'n kritiese periode gedurende fetale ontwikkeling is waartydens die fetus veral vatbaar is vir virusbesmettings en dit is veral die sentrale senuweestelsel (SSS) wat sensitief is vir teratologie in die tydperk. Die kritiese periode varieer volgens die spesie betrokke en hou verband met die lengte van die dragtigheidsperiode. So is dit bekend dat hidranenkefalie en porenkefalie ontwikkel indien die beesfetus besmet sou raak tussen 126–138 d van dragtigheid met die gemodifiseerde BT virus[1]. In lammers is soortgelyke abnormaliteite verwek waar skape tussen 42–74 dae van dragtigheid met die muisbrein geattenueerde SDK virus en muisbrein geattenueerde WBS virus asook die wildetipe WBS virus besmet is[2]. Osburn *et al.*[13] het bewys dat hidranenkefalie in die fetus volg indien ooie tussen 50–58 d van dragtigheid gespuit word met die gemodifiseerde BT virus en dat dit lei tot porenkefalie waar ooie van 75–78 d van dragtigheid met die virus besmet is. Hulle het ook gevind dat daar slegs 'n ligte fokale enkefalitis teenwoordig was in die lammers waar die ooie na 100 d van dragtigheid besmet is. Dit is dus baie duidelik dat minder ernstige letsels ontwikkel indien die fetus in die latere stadium van ontwikkeling besmet sou raak as wanneer besmetting sou intree tydens die eerste trimester van dragtigheid. Verder is dit interessant om daarop te let dat transplasentale besmetting van die beesfetus (79–150 d van ontwikkeling) met BVD-MS virus serebellare hipoplasie en nie hidranenkafalie of porenkefalie tot gevolg het nie[20]. Dit wil voorkom dat vir elke dierlike spesie die serebellum 'n vinnig prolifererende stadium deurgaan gedurende 'n spesifieke fetale ouderdom en dat die serebellum veral vatbaar is vir sel vernietiging a.g.v. virusse gedurende die periode. In die bees vind maksimale serebellare groei plaas tussen 133–162 d van dragtigheid en in die kat kort voor geboorte tot 2–3 weke na geboorte. Daar is ook opvallende groei en ontwikkelingsverskille van die serebellum en rugmurg t.o.v. die res van die SSS in die vark. Ander faktore wat die fetus meer vatbaar maak vir besmettings sluit in die graad van immuunkompetensie of immuunvolwassenheid, onder ontwikkeling van die limfoiede weefsel, die aktiwiteit van die retikuloendoteelsisteem (RES) en verminderde interferon produksie[5]. Sellulêre vatbaarheid en spesifieke weefseltropisme is ook belangrik in die verband en kan 'n besliste invloed hê op die tipe letsel at sal volg na besmetting. 'n Sprekende voorbeeld hiervan word weergee in onlangse werk met die wilde-tipe WBS sowel as die geattenueerde WBS en geattenueerde SDK virusse wat duidelik toon dat die virusse 'n spesifieke tropisme het vir die ontwikkelende SSS van veral die skaapfetus[2].

Tweedens is daar sekere virologiese faktore wat 'n rol kan speel in dié kongenitale teratologiese toestande. Die duurte en hoogte van die viremie blyk krities te wees in soverre dit binnedringing van die SSS betref[9]. Groei en uitsaaiing van virus van ekstraneurale weefsel asook suiwering van virus deur die RES mag net so belangrik wees in die verband. Een van die belangrikste eienskappe van die teratogeniese virusse is dat hulle gewoonlik 'n lae patogeniteit het vir die moederdier. Virussiektes soos BT, Akabanesiekte, WBS en BVD-MS is heel dikwels subkliniese besmettings in herkouers. Dit is noodsaaklik dat die letsels wat in die fetus veroorsaak word na transplasentale binnedringing van ligte graad is en dat die orgaan (bv. die brein) wat aangetas word nie belangrik is vir fetale oorlewing nie. Verder is dit bekend dat dié virusse 'n affiniteit het vir

vinnig delende selle soos die wat in ontwikkelende fetusse gevind word en hier is die SSS veral vatbaar vir besmettings. Dit is ook duidelik dat sekere virusse en veral van die geattenueerde virusse (WBS en SDK) definitiewe neurotropiese eienskappe besit. Alhoewel daar nog nie finaliteit bereik is oor die patogenese van virusteratologie nie, is daar tog heelwat moontlike meganismes voorgestel. Horstmann beweer dat sellulêre nekrose met litiese en nie-litiese virusse 'n versteuring van die groei en maturasie van weefsels veroorsaak. Baie van die defekte kan ook gekoppel word aan vertraagde replikasievermoë van die besmette selle[14]. Dit is veral interessant om daarop te let dat sekere virusse hidranenkefalie en porenkefalie tot gevolg het en hierteenoor is serebellare hipoplasie weer die prominente letsel in ander kongenitale virusbesmettings. Dit kan moontlik verklaar word deur die selektiewe vernietiging of aanstasting van selle in die ontwikkelende brein deur verskillende virusse. Dit is reeds bewys dat die gemodifiseerde BT veral die selle aantas in die ventrikulêre sone[12]. Van hier migreer die selle uit om die serebrale korteks te vorm, maar a.g.v. die steurnis in sellulêre migrasie ontwikkel hidranenkefalie of porenkefalie. Netso is dit bekend dat in die geval van BVD-MS virus en sekere parvovirusse die primordiale selle in die granulêre laag van die serebellum primêr aangetas en vernietig word. Die gevolg hiervan is serebellare hipoplasie. Dan is daar ook aanduidings dat BVD-MS virus vaskulitis en soms trombose in die serebellum kan veroorsaak met gevolglike anoksie van die deel van die brein[20].

Sekere maternale faktore is ook belangrik in die verband. Van primêre belang is dat die moederdier vatbaar sal wees vir die betrokke virus en net so belangrik is dat daar 'n meetbare viremie volg na besmetting. Die verwantskap tussen maternale en fetale besmetting is kompleks a.g.v. die teenwoordigheid van die plasenta. Die rol van die plasenta as grens varieer volgens die spesie betrokke en die stadium van dragtigheid. Daar is indikasies dat die transplasentale grens meer volledig ontwikkel na gelang dragtigheid vorder. Dit is dus nie verregaande om die stelling te maak dat in virusbesmettings van die moederdier waarin die viremie baie laag is, die kanse van transplasentale besmetting ook aansienlik verminder veral in die latere stadiums van dragtigheid. Net soos in die geval van die bloed-brein, bloed-timus en bloed-testis skanse, kan die virusse die transplasentale skans oorsteek deur te groei, met lekkasie of daaroor gedra te word deur migrerende of ander selle. Sekere virusse lokaliseer in die plasenta en groei oor die plasenta aansluiting. Voorbeelde hiervan is rubella en sitomegalovirusse in die mens en BT virus in herkouers. Die rede vir lokalisering van mikro-organismes in die plasenta is nie altyd duidelik nie maar die stadige vloei van bloed deur die plasenta bloedvate (soos in sinusoiede) mag lokalisering bevorder. Indien die virus primêr in die plasenta 'n vaskulitis veroorsaak, kan dit lei tot fetale anoksie, afsterwing en gevolglik resorpsie, aborsie of mummifisering.

Alhoewel hidranenkefalie en porenkefalie nie altyd gepaard gaan met artrogripose (Fig. 5) word dit tog dikwels met die abnormaliteit geassosieer. Volgens Coetzer & Barnard[3], het artrogripose feitlik as 'n reël voorgekom in skaapfetusse en lammers waar hidranenkefalie en porenkafalie gepaard gegaan het met varierende grade van rugmurg hipoplasie (Fig. 6) of spesifieke histopatologiese letsels in die rugmurg a.g.v.

Fig. 5 Artrogripose in 'n skaaplam.

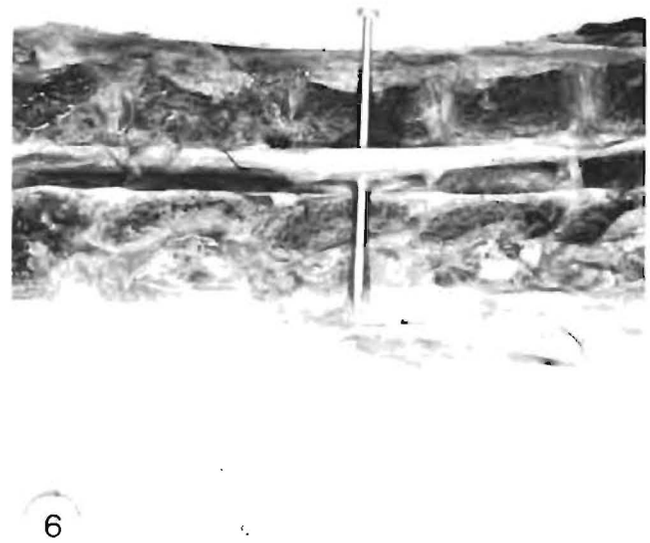

Fig. 6. Let op die opvallende rugmurg hipoplasie.

transplasentale besmettings met die muisbrein geattenueerde SDK en WBS virusse sowel as met die wildetipe WBS virus. Volgens die werkers[3] gee die brein en rugmurg defekte oorsprong aan uitgesproke neurogene spieratrofie wat weer 'n belangrike rol mag speel in die patogenese van artrogripose in die gevalle.

DIAGNOSE

Geskiedenis en kliniese simptome

Dit is belangrik om 'n deeglike geskiedenis in te win omdat dit dikwels van waarde kan wees om te onderskei tussen genetiese- en virusteratologie. Indien die afwykings die gevolg is van een of ander transplasentale teratogeniese virusbesmetting kan die gebreke nie te-

ruggèvoër word na 'n spesifieke bul nie en kom meestal voor in enige suiwer of kruisgeteelde ras. Ander aanduidings dat teratogeniese virusse die oorsaak mag wees van die SSS toestand sluit in: eksplosiewe uitbreke wat dikwels beperk is tot een kalfseisoen; resorpsie, aborsie en mummifisering gaan dikwels hiermee gepaard; die afwykings kom meestal voor in diere wat vir die eerste keer kalf of lam en die insidens is gewoonlik nie beperk tot 'n enkele plaas nie maar kom voor op verskillende plase in die omgewing.

Die fetusse, kalwers of lammers word dikwels geaborteer, of is dood of lewendig by geboorte. Abnormale diere kan meestal nie vanself staan en suip nie en toon ander simptome soos opistotonus, gallop-bewegings met die ledemate en niastagmus. Dit is interessant om daarop te let dat feitlik geen simptome waarneembaar is in gevalle waar daar slegs geringe letsels in die brein is nie.

Patologiese ondersoek

Tydens 'n patologiese ondersoek kan die ondersoeker dikwels leidrade kry van watter virus moontlik verantwoordelik was vir die abnormalitiete. Die primêre letsels in die geval van BVD-MD is serebellare hipoplasie terwyl hidranenkefalie en porenkefalie weer geassosieer word met BT en Akabanesiekte. Hierteenoor veroorsaak die geattenueerde WBS en SDK virusse en die wilde-tipe WBS virus serebellare hipoplsie, hidranenkefalie en porenkefalie in herkouers. Dit is belangrik om daarop te let dat dié kongenitale defekte nie beperk word tot teratogeniese virusse nie maar dat soortgelyke afwykings ook geneties van oorsprong mag wees.

Virusisolasie

'n Belangrike punt om te noem is dat die virusse gewoonlik net kort na transplasentale besmetting uit breinweefsel of ander weefsels afgesonder kan word. Hierna is pogings tot virusafsondering meesal onsuksesvol. Hierdie aspekte moet deeglik in oorweging geneem word indien virusblootstelling aan perinatale afwykings gekoppel word. Omrede virusafsondering nie altyd moontlik sal wees nie, kan indirekte bewyse soos styging in die immunoglobulienvlakke of die teenwoordigheid van spesifieke virusteenliggaampies in fetale of prekolostrumserum dalk van waarde wees in die maak van 'n diagnose.

Kwantitatiewe immunoglobulien en spesifieke teenligaampiebepalings

Immuunkompetensie of immuunvolwassenheid van die fetus word basies beïnvloed deur 3 faktore nl. die spesie betrokke, die lengte van dragtigheid en die tipe antigeen. Dit is in die herkouer bekend dat die fetus in staat is om immunologies te reageer teen verskillende antigene op verskillende stadiums van fetale ontwikkeling[15 19]. Dit is belangrik om te besef dat geen fetus agammaglobulienemies gebore word nie maar wel lae vlakke van immunoglobuliene bevat. Indien die fetus antigenies gestimuleer word, gaan dit as 'n reël gepaard met aansienlike stygings in die immunoglobulienvlakke[15 27]. Dit kan dan as indikator dien dat daar *in utero* besmetting plaasgevind het. Spesifieke teenliggaampies word eers gedurende die later stadium van fetale ontwikkeling geproduseer[4 7 18 19].

Immunoglobulien en spesifieke teenliggaambepalings kan op die prekolostrumeserum sowel as op breinvloeistof van hidranenkefalie en porenkefalie gevalle gedoen word.

Serologiese ondersoeke op die kudde

Die ondersoek van enkel serummonsters van die moederdier het weinig diagnostiese waarde en kan tot verwarring of selfs foutiewe diagnoses aanleiding gee. Selfs die ondersoek van gepaarde sera (versamel kort na aborsie of geboorte van die abnormale dier en weer 3–4 weke later) gee dikwels ook nie die gewensde resultate nie omdat besmetting van die moederdier etlike maande vantevore reeds plaasgevind het. Dit is van veel meer diagnostiese waarde om die kudde, en veral die jong diere wat vir die eerste keer gaan kalf of lam, met gereelde tussenposes oor die bestek van 6–12 maande serologies te monitor vir stygings in die teenliggaampievlakke teen sekere virustoestande.

BEDANKING

Ek wil graag mnr. A. M. du Bruyn en sy personeel bedank vir die fotografie.

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BOOK REVIEW BOEKRESENSIE

FERTILITY AND INFERTILITY IN DOMESTIC ANIMALS

J. A. LAING
3rd Ed. Bailliere Tindall London. 1979 pp ix and 262, Figs 106 and 28 tables. Publishers Price R22.95
(ISBN 0 7020 06998)

This edition of an already well-known text has been extensively revised and now presents an up to date account of the many aspects involved in the determination of fertility and infertility in domestic animals. This has been achieved with the cooperation and contributions of 17 leading and well-known veterinary specialists.

The first chapter on the normal fertility and incidence of infertility in the U.K. has been considerably shortened. Chapter Two, dealing with the normal breeding animal, has been completely rewritten and now presents an account of the genital system, male and female, from a clinical, diagnostic point of view. The third chapter has also been simplified and now presents an easy to follow introduction to and discussion of pregnancy diagnosis in our domestic animals.

Chapter Four has likewise been revised and now covers the evaluation of semen, the characteristics of semen in the various species and artificial insemination including the collection of semen, dilution and preservation in the various species with the exception of dogs.

The fifth chapter deals with male abnormalities including cryptorchidism, testicular degeneration and hypoplasia, orchitis and epididymitis, acrosomal abnormalities, accessory sex gland abnormalities, penile abnormalities and impotence.

Chapter Six covers the management of ovarian function from the ovulation-insemination time relationship, synchronization of oestrus and ovulation to ovarian inactivity in cattle and ovarian function in relation to fertility in the remaining domestic animals. The chapter is concluded with a short section on embryo transfer in cattle.

Chapter Seven deals with non-infectious causes of infertility such as mineral deficiency, cystic ovarian degeneration, intersexes, segmental aplasia, neoplasia and chromosomal studies in the female animal.

The next few chapers then deal with infectious causes of infertility such as viral infections (Chapter 8) brucellosis (Chapter 9) and other infectious diseases such as endometritits, listeriosis, tuberculosis, vibriosis, salmonellosis, leptospirosis, chlamydiosis, mycoplasmosis and toxoplasmosis and trichomoniasis (Chapter 10).

The book is concluded with a short chapter on official measures applicable in the United Kingdom on artificial insemination, distribution of semen, trichomoniasis, vibriosis and brucellosis, export and import of semen, surveys, diagnostic measures and embryo transfer.

This new edition will certainly serve a useful purpose as a guide to people involved in the practical aspects of fertility and infertility. It can also be recommended to veterinary students. They will, however, have to bear in mind that many sections are only dealt with superficially and that the text is by no means a complete manual on the very diverse subject of fertility and infertility.

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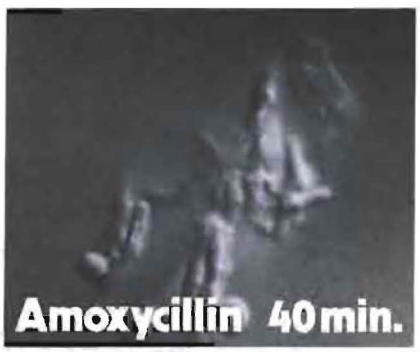

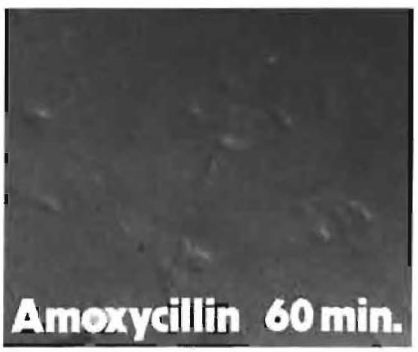

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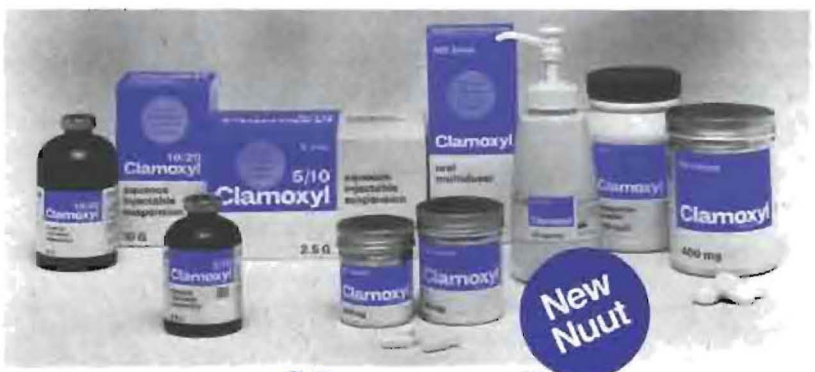

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ENTERITIS IN SHEEP DUE TO THE INGESTION OF *INULA GRAVEOLENS* DESF (CAPE KHAKIWEED)

D. J. SCHNEIDER* and J. L. DU PLESSIS†

ABSTRACT: Schneider, D. J.; du Plessis, J. L. **Enteritis in sheep due to the ingestion of *Inula graveolens* Desf (Cape Khakiweed).** *Journal of the South African Veterinary Association* (1980) **51** No. 3 159–161 (En) Regional Vet. Laboratory, P. Bag X5020, Stellenbosch, Rep. of S. Africa.

Diarrhoea and mortality in sheep on a farm in the Winter Rainfall area of the Republic of South Africa were found to be due to an enteritis produced by the massive penetration of the mucous membrane of the small intestine by the bristles of the pappus of the Cape Khakiweed.

CASE HISTORY

One of us was called upon to investigate mortality and diarrhoea in a flock of 190 9-month old Merino sheep in the Wellington district of the Cape. The sheep had for about 3 months been grazing in the same camp, consisting mainly of wheat-stubble land and patches of natural vegetation, and were in good bodily condition. *Elytropappus rhinocerotis* (renosterbos) dominated the natural vegetation, whereas an abundant growth of *Inula graveolens* (Cape Khakiweed) occurred between the wheat-stubble. The *I. graveolens* plants (Fig. 1) had reached the end of their growth period and were covered with dry flowers bearing ripe seed which was already being shed.

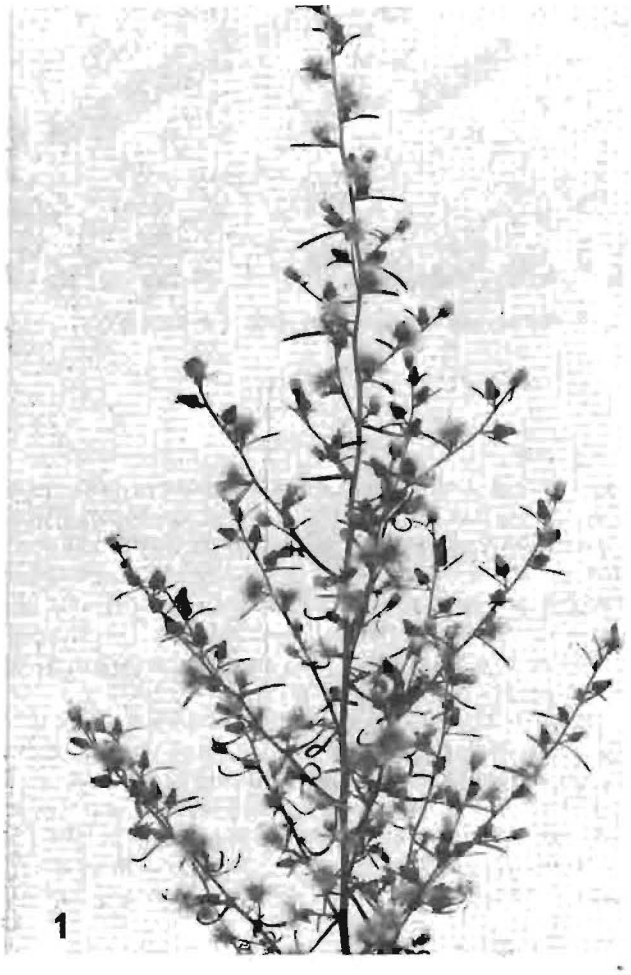

Fig. 1. *I. graveolens* with seed in the process of being shed.

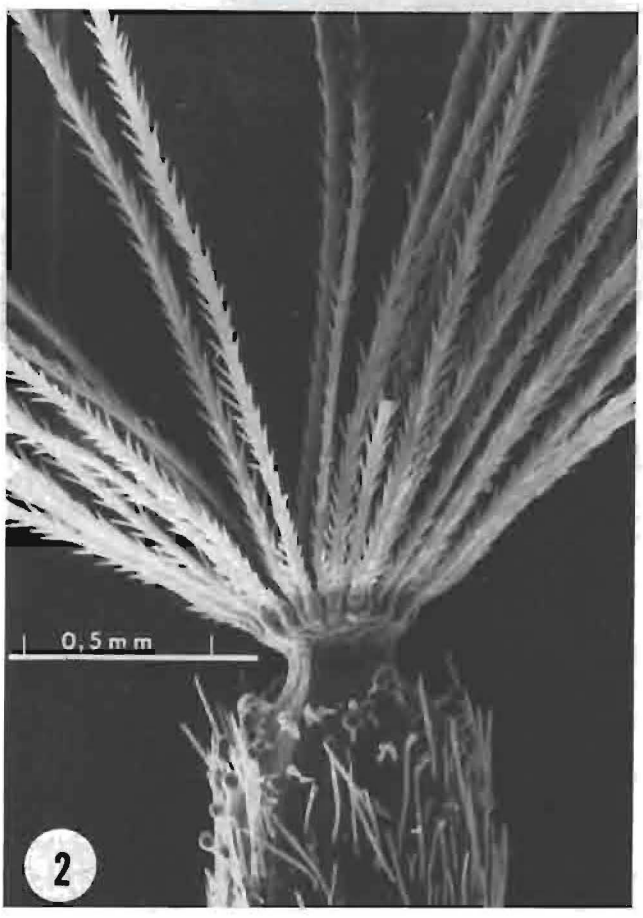

Fig. 2. Scanning electron photomicrograph of the pappus of *I. graveolens* with its bristles x 75.

A tentative diagnoses of enteritis due to massive penetration of the mucosa of the small intestine by the hairlike bristles of the pappus of *I. graveolens*, about 4mm in length and for their entire length covered with barbs (Figs 2 & 3), was made.

The sheep were taken out of the camp and those showing diarrhoea treated with carron oil. Diarrhoea and mortality stopped within 3 days and no futher adverse effects were reported by the owner. In total 10 sheep died out of a flock of 190.

CLINICAL SYMPTOMS

Symptoms observed included mild to severe diarrhoea, prostration and death. The faeces of affected sheep were dark brown, had a watery consistency and con-

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†Veterinary Research Institute, Onderstepoort

0038-2809/80/03/0159–0161

Fig. 3. Pappus of *I. graveolens* x 13.

tained mucous. In severe cases a mild febrile reaction and dehydration were present. Affected sheep were anorectic and listless. Their abdomens were tucked-up and they lost condition rapidly.

PATHOLOGY

Macroscopic lesions

The clinical dehydration was confirmed at autopsy. Congestion of the lungs was in evidence and the rumen contents were reduced in quantity. Mild infestations of *Ostertagia* in the abomasum and *Trichostrongylus* in the duodenum were observed.

The wall of the entire small intestine appeared thickened and was reddish in colour. Examination of the mucosa revealed an inflammation and large numbers of hairlike projections about 4mm in length protruding from the mucosal surface. Microscopic examination of these projections showed them to be identical to the bristles of the pappus of *I. graveolens* when compared with those collected from the plant. The numbers of the bristles in the small intestine increased from cranially to caudally. The severity of the inflammatory response paralleled the number of bristles.

Histopathology

Microscopic examination of several sections of small intestine revealed numerous bristles in cross and longitudinal section (Figs 4 & 5). Others were lodged at the bases of the villi (Fig. 4, double arrows), from where they apparently penetrated the mucosa. The bristles lodged in the propria mucosa were surrounded by necrotic host tissue cells infiltrated by numerous neutrophils and large numbers of bacteria. The direction of the barbs on the bristles (Fig. 4, arrow) suggest that they determined the direction of penetration and prevented retraction of the bristles.

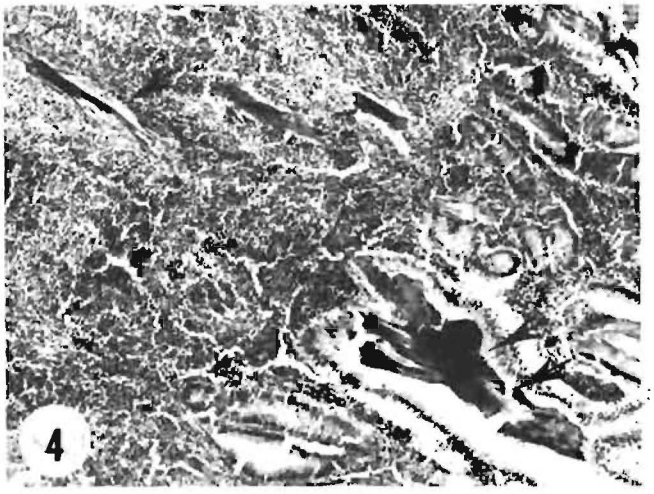

Fig. 4. Histological section of the small intestine showing bristles in the propria mucosa (arrow) and at the base of the villi (double arrows). H & E x 75.

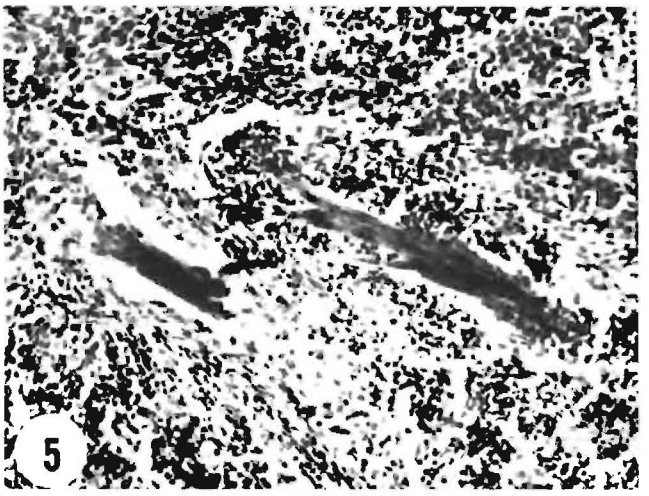

Fig. 5. Higher magnification of part of Fig. 4. H & E x 200.

DISCUSSION

It seems that symptoms and death occur only after massive penetration of the intestinal mucosa by bristles of *I. graveolens,* as was the case in this particular instance. One of us has on many occasions during autumn and winter in the area concerned noticed the bristles in the mucosa of sheep submitted for autopsy. They were, however, few in number and no definite signs of an inflammatory reaction were present.

The distribution of Cape Khakiweed is confined to the South-Western Cape Province. It is an introduced species native to the Mediterranean area of Europe. It is a weed in grain lands in several districts as it overgrows valuable summer growing fodder plants like *Cynodon dactylon (L) Pens., Polygonum aviculare L* and various *Amaranthus* species. It also hampers ploughing and sowing operations by clogging machinary.

Its well developed root system enables it to flourish in the dry months of late summer and autumn, forming dense stands on stubble or fallow lands. The plant normally sets seed in vast quantities during April and May.

Although fairly unpalatable, the plant is often grazed by sheep while in the young growing stage due to the scarcity of other green material during the dry summer

months. The aromatic resinous oil of the plant sticks to the wool and hair of sheep when grazing these lands causing a dark brown discolouration, especially of the legs, head and face. This resin is not easily removed. No deleterious effects have been noticed during this stage.

During the seed-producing stage it is seldom eaten because it is more or less dry and unattractive and by this time the first winter rains have usually fallen and the first green winter grazing appears on the lands. On this occasion the winter rains were late and, because no other food was available, the sheep were forced to eat *I. graveolens* in its seed-bearing stage. This possibly accounts for the fact that the symptoms as described here, do not occur more often in sheep grazing lands infested with this weed.

In view of the abundance of this plant in the Winter Rainfall areas during autumn and the absence of specific symptoms other than perhaps a mild diarrhoea, it is suggested that many sheep probably suffer from subclinical enteritis resulting from ingestion of *I. graveolens*.

BOEKRESENSIE

BOOK REVIEW

KEN ONS KLEINVEERASSE

E. TERBLANCHE

Human & Rousseau, Kaapstad en Pretoria, 174 pp, Nege Kleurfotos, 96 Swartwitfotos. Prys R11.50

Die belangrikheid van die kleinveebedryf as 'n bron van sowel rooivleis as wol en sybokhaar neem nog steeds toe. Die bedryf voed en klee die inwoners van ons land, en dit verdien ook waardevolle buitelandse valuta vir die Republiek.

Dr Etienne Terblanche was Assistent Direkteur (Voorligting) van die Oos-Kaapstreek van die Dept. Landbou-Tegniese Dienste en boer tans op sy plaas Glen Cliffe by Aberdeen met Angorabokke, Dorpers en Merinos. Hy het daarin geslaag om die belangrikste aspekte van 20 skaap- en 6 bokrasse saam te vat en met 'n hele reeks fotos besonder aantreklik aan te bied. Die belangstelling en instandhouding van suiwer rasse is belangrik aangesien dit as 'n genetiese hulpbron dien waarop die kommersiële produsent kan terugval in sy pogings om sy produksiestelsels winsgewend te bedryf. Kennis oor die rasse sal die teler en die boer in staat stel om vergelykings te tref en te besluit watter ras sy omstandighede die beste pas.

Die inligting word op duidelike en sistematiese manier aangebied deur iemand wat 'n erkende deskundige op die gebied is en self met die praktyk van die kleinveebedryf vertroud is. Hierdie gesaghebbende werk word verwelkom en kan 'n groot bydrae lewer in die verhoging van die kennis oor ons land se kleinveestapel. Die boek word met groot vrymoedigheid aanbeveel vir almal wat met die veebedryf gemoeid is insluitende landbou- en veeartsenystudente.

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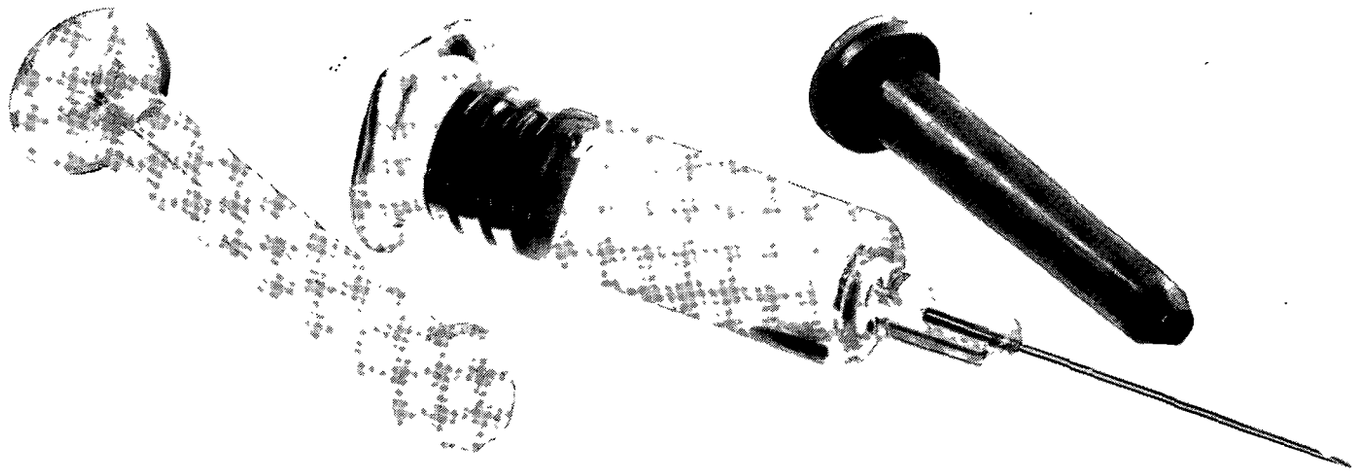

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FIELD OUTBREAKS OF HYPEROESTROGENISM (VULVO-VAGINITIS) IN PIGS CONSUMING MAIZE INFECTED BY *FUSARIUM GRAMINEARUM* AND CONTAMINATED WITH ZEARALENONE

H. W. AUCOCK*, W. F. O. MARASAS†, C. J. MEYER† AND P. CHALMERS‡

ABSTRACT: Aucock, H. W.; Marasas, W.F.O.; Meyer, C. J.; Chalmers, P. **Field outbreaks of hyperoestrogenism (vulvo-vaginitis) in pigs consuming maise infected by *Fusarium graminearum* and contaminated with zearalenone.** *Journal of the South African Veterinary Association* (1980)**51** No. 3 163–166 (En) Meat Board, 20 Greenwood Road, 3201 Pietermaritzburg, Republic of South Africa.

During the spring and summer of 1979 field outbreaks of porcine hyperoestrogenism characterized by swelling and reddening of the vulva and teats in prepubertal gilts and enlargement of the mammae in young males occurred in the mistbelt of the Natal Midlands.

On a farm in the Ixopo district, pigs were fed a mixed ration containing home-grown yellow maize stored on the cob in a crib and all the young pigs with a mass of 25–85 kg (=a total of 350) were affected. Maize ears from the crib had a high percentage (91%) of kernels infected by *Fusarium graminearum* and contained 10 mg/kg of zearalenone. The mixed ration contained 0,95 mg/kg zearalenone. After dilution of the contaminated maize with good quality white maize, a dramatic decrease in both the incidence and severity of clinical signs occurred within 3–4 days. On a farm in the Winterton district, approximately 80% of the growing pigs with a mass of more than 40 kg fed a mixed ration containing shelled yellow maize from the silo of the local Agricultural Co-operative were affected. The incidence as well as the severity of the swelling of the vulvas and/or teats were lower than on the farm at Ixopo, thus indicating a lower level of oestrogen intake. A sample of maize from the silo delivered to this farm contained only 0,06 mg/kg zearalenone while the mixed ration did not contain chemically detectable levels of zearalenone.

This is the first published record of field outbreaks of porcine hyperoestrogenism associated with the ingestion of *F. graminearum* infected maize contaminated with zearalenone in the Republic of South Africa.

INTRODUCTION

Ingestion of maize (*Zea mays* L.) infected by the phytopathogenic fungus *Fusarium graminearum* Schwabe by pigs causes hyperoestrogenism characterized by tumefaction of the vulva, uterine hypertrophy, enlargement of the mammary glands, and somtimes vaginal prolapse in prepubertal gilts and hypertrophy of the mammae, oedematous swelling of the prepuce and testicular atrophy in males[2 7 9 10]. These effects are caused by the oestrogenic metabolite, zearalenone (6- (10-hydroxy-6-oxo-trans-1-undecenyl)–β–resorcylic acid lactone), produced by *F. graminearum* in infected maize[18]. Zearalenone has been found to occur naturally in maize and other feedstuffs associated with outbreaks of porcine hyperoestrogenism at levels ranging from 0,1–2909 mg/kg[7 9 10 11 13 15 17]. Dietary levels of 1–5 mg/kg zearalenone are considered to be sufficient to produce clinical signs of hyperoestrogenism in young gilts[11 27].

In Southern Africa, zearalenone has been detected in *Fusarium*-infected maize in Zambia[3 4], Swaziland[6], Republic of South Africa[4], and Transkei[5] at levels ranging from 0,45–12,8 mg/kg[4 5]. Although some of these levels are within the range of 1–5 mg/kg considered to be physiologically active, no field outbreaks of porcine hyperoestrogenism have been reported in southern Africa to date.

This paper reports on the occurrence of field outbreaks in Natal Province of the Republic of South Africa of an oestrogenic syndrome in pigs associated with the consumption of mixed rations containing *F. graminearum* infected maize contaminated with zearalenone.

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‡Veterinary Regional Laboratory, Allerton, 3200 Pietermaritzburg.

CASE REPORT

History and clinical signs

During August to November, 1979 outbreaks of hyperoestrogenism (vulvo-vaginitis) in pigs occurred on several farms in the districts of Ixopo and Winterton in the mist belt of the Natal Midlands. The characteristic clinical signs were red, swollen vulvas (Fig. 1) and swollen teats in prepubertal gilts and enlarged teats in the young males. Although isolated cases of abortion, fertility problems such as return to heat and reduced litter sizes, feed refusal, and emesis also occurred on some farms, no definitive evidence could be found that any of these signs were associated manifestations of the observed oestrogenic syndrome.

Detailed investigations were conducted on 2 affected farms. On a farm in the Ixopo district, home-grown yellow maize ears (Fig. 2) were harvested during June-/July, 1979 and stored on the cob in a wire-mesh crib with a corrugated iron roof. Maize ears from this crib were shelled, ground, and incorporated into the following mixed ration:

Maize meal	71%
Bran	14%
Fish meal	13%
Bone meal	1%
Salt	1%
Vitamin and mineral premix	

This ration was fed *ad lib.* to young growing pigs on the farm. During August 1979, all of the pigs with a mass of 25–85 kg (= a total of 350) developed clinical signs of red, swollen vulvas and/or teats to a greater or lesser degree. None of the adult breeding stock on a restricted feed intake were affected. After the farmer had been informed that the maize from the crib may be responsible for the clinical signs, he replaced 30% of the maize in the diet with good quality white maize. a dramatic decrease in both the incidence and severity of clinical

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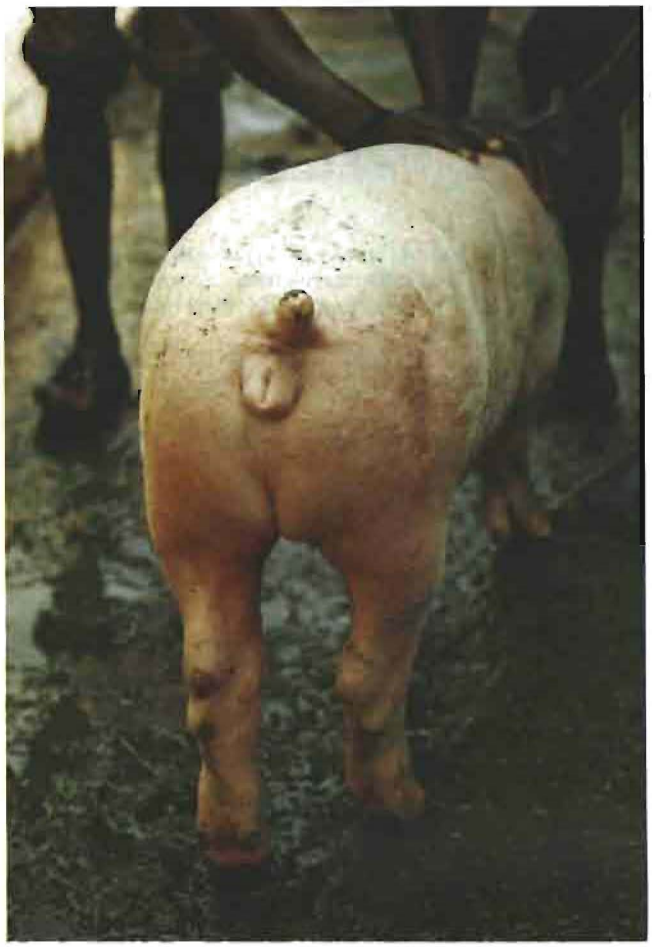

Porcine hyperoestrogenism caused by the consumption of *graminearum*-infected maize contaminated with zearalenone.

Fig. 1. Swelling and reddening of the vulva in a prepubertal gilt.

Fig. 2. Maize ears stored in a crib and showing the characteristic red discolouration of the kernels caused by *F. graminearum* infection.

signs occurred within 3 or 4 days after commencement of feeding of the modified ration. When the farm was visited 3 weeks later, only a few animals with mildly swollen vulvas and/or teats were observed.

On a farm in the Winterton district, shelled yellow maize was purchased from the local Agricultural Co-operative where the maize was stored in large concrete silos. The maize was ground on the farm directly upon delivery from the silo and incorporated into the following mixed ration:

Maize meal	70%
Dehydrated lucerne	16%
Fish meal	11%
Carcass meal	3%
Vitamin and mineral premix	

During November, 1979, clinical signs of hyperoestrogenism were seen in approximately 80% of the growing pigs with a mass above 40 kg on the farm. The incidence as well as the severity of the swelling of the vulvas and/or teats were lower than on the farm at Ixopo. This observation, together with the fact that only larger, porker size pigs, i.e. pigs with a mass of more than 40 kg that consume more feed than younger pigs, were affected, indicated a lower level of oestrogen intake on this farm compared to the farm at Ixopo.

Meteorological data

Infection of maize ears by *F. graminearum* is known to be enhanced by cool, wet conditions during silking[19] and the production of zearalenone in infected maize stored on the cob is increased by conditions of high moisture and low temperature[2 7 10 11]. Meteorological data for the affected districts are given in Table 1. It can be seen that December 1978, the period when silking of the maize harvested during June/July 1979 took place, was wetter and slightly cooler than normal. The rainfall during the period directly after harvest and just prior to the onset of the first outbreaks (July to August 1979), was much higher than usual and the average maximum temperatures were somewhat lower. Consequently it can be concluded that climatic conditions in the affected areas during 1978/79 were favourable for the infection of maize ears by *F. graminearum* and particularly for zearalenone production in harvested maize stored on the cob in cribs.

LABORATORY EXAMINATIONS

Samples

The following samples (ca. 1 kg each) were collected on the 2 farms under investigation:

Table 1: METEOROLOGICAL DATA FOR THE DISTRICTS OF IXOPO AND WINTERTON*

	IXOPO		WINTERTON	
	1978/79	Long term av.	1978/79	Long term av.
Total monthly rainfall (mm)				
December	125,3	116,2	281,1	124,7
July	45,3	19,3	19,6	11,4
August	45,2	35,9	77,0	26,3
Total (July-August)	90,5	55,2	96,6	37,7
Av. maximum temp (°C)	Not available			
December			30,0	30,8
July			21,8	23,5
August			23,9	25,0
Av. minimum temp	Not available			
December			13,9	14,5
July			0,4	4,7
August			0,3	3,2

*Data supplied by the Agrometeorological Section, Research Institute for Soils and Irrigation, Department of Agricultural Technical Services, Private Bag X9059, Pietermaritzburg.

1. Ixopo. Ears of yellow maize showing signs of fungal infection from storage crib. Collected during September 1979 when clinical signs of vulvo-vaginitis were evident.
2. Ixopo. Visibly *F. graminearum*-infected yellow maize ears showing the characteristic red discolouration (Fig. 2) selected from Sample 1.
3. Ixopo. Mixed ration. Collected during September 1979.
4. Ixopo. Shelled yellow maize from storage crib mixed with good quality white maize. Collected during November 1979 when clinical signs had almost completely disappeared.
5. Winterton. Shelled yellow maize from the silo of the Agricultural Co-operative. Collected directly after delivery to the farm during November 1979 when mild clinical signs were evident.
6. Winterton. Mixed ration. Collected during November 1979.

Mycology

The samples of maize ears (Samples 1 and 2) were shelled in a hand sheller. The percentage of kernels infected by internally seedborne species of *Fusarium* was determined as previously described[4] for Samples 1, 4 and 5.

It can be seen from Table 2 that the home-grown maize stored on the cob in a crib at Ixopo (Sample1) had an extremely high percentage (91%) of kernels infected by *F. graminearum.* The level of *F. graminearum* in the shelled maize which had been diluted with good quality maize (Sample 4) was reduced to 20%. Both of these maize samples from Ixopo had relatively high percentages of kernels infected by *F. sacchari* (Burl.) Gams var. *subglutinans* (Wollenw and Reink) Nirenberg (=*F. moniliforme* Sheld. var. *subglutinans* Wollenw and Reink).

The maize from the silo at Winterton (Sample 5) had only 1% of the kernels intected by *F. graminearum,* but a relatively high percentage (27%) of kernels infected by *F. verticillioides* (Sacc.) Nirenberg (= *F. moniliforme* Sheld.).

Chemistry

The shelled maize samples were ground to a meal in a coffee grinder. The maize meals obtained in this way as well as the mixed feed samples were analyzed for zearalenone by the method of Mirocha et al.[15] using the procedures described for batch extraction, base clean-up and analytical thin layer chromatography with a zearalenone standard obtained from Prof. C. J. Mirocha, University of Minnesota. The presence of zearalenone in positive sample was further confirmed by intensification of its long wave fluorescence by spraying with $AlCl_3$[16].

All the maize samples as well as the mixed feed sample from Ixopo contained chemically detectable levels of zearalenone (Table 3). As expected, the visibly *F. graminearum* infected ears (Sample 2, Fig. 2) contained a higher level (32 mg/kg) of zearalenone than the mouldy ears from the crib (Sample 1, 10 mg/kg), some of which were also infected by other fungi such as *Diplodia maydis* (Berk.) Sacc. The most significant finding was the presence of 0,95 mg/kg zearalenone in the mixed feed (Sample 3) which was being fed to the pigs when clinical signs became evident. This corresponds to the lower level of dietary zearalenone considered to be sufficient to produce clinical signs of vulvo-vaginitis in swine[2,7,11].

The shelled maize which had been diluted with good quality maize and which was being incorporated into the mixed ration at the time when clinical signs had almost completely disappeared (Sample 4), contained only 0,16 mg/kg zearalenone.

The level of zearalenone found in the sample of shelled maize from the silo in Winterton (Sample 5, 0,06 mg/kg) was probably too low to have elicited the clinical signs observed and in fact the mixed ration (Sample 6) did not contain chemically detectable levels of zearalenone. Only 1% of the kernels in this maize sample were infected by *F. graminearum* compared to 27% with *F. verticillioides* (Table 2). Although the latter species has also been reported to produce zearalenone in culture[8], it apparently did not produce significant amounts in the naturally infected maize form Winterton. The small sample of maize examined cannot be considered to be representative of the different lots of

Table 2: INCIDENCE OF *FUSARIUM* SPECIES IN MAIZE ASSOCIATED WITH PORCINE HYPEROESTROGENISM

Maize Samples*	% KERNELS INFECTED†			Germination (%)
	F. verticillioides	*F. sacchari var. subglutinans*	*F. graminearum*	
1. Ixopo, mouldy ears	0	43	91	1
4. Ixopo, shelled maize	5	27	20	84
5. Winterton, shelled maize	27	9	1	88

*See text for details of samples
†Some kernels were infected by more than one species

maize present in a large silo. Although the clinical signs of vulvo-vaginitis observed at Winterton were much milder than those at Ixopo, thus indicating a lower level of oestrogen intake, it is presumed that some batches of maize from the silo contained zearalenone levels higher than 0,06 mg/kg in order to have induced the clinical signs observed. Another possibility is that other oestrogens such as diethylstilbestrol[7] or zearalenol[14] may have been present in the mixed feed.

Table 3: ZEARALENONE CONTENT OF MAIZE AND FEED ASSOCIATED WITH PORCINE HYPEROESTROGENISM

Samples	Zearalenone Content (mg/kg)†
1. Ixopo, mouldy ears	10,0
2. Ixopo, visibly *Fusarium graminearum* infected ears	32,0
3. Ixopo, mixed ration	0,95
4. Ixopo, shelled maize mixed with good quality maize	0,16
5. Winterton, shelled maize	0,06
6. Winterton, mixed ration	Not detected

*See text for details of samples
†Detection limit: 0,005 mg/kg

DISCUSSION

Field outbreaks of porcine hyperoestrogenism in the mist belt of the Natal Midlands during the spring and summer of 1979 could be associated with zearalenone contamination of home-grown maize stored on the cob in a crib as well as with commercial yellow maize stored in a silo. The levels of zearalenone were much higher, and the clinical signs of vulvo-vaginitis much more severe, in the maize stored on the cob than in the case of shelled maize stored in a silo. The high levels of zearalenone in the maize stored in a crib were most probably associated with abnormally high winter rainfall just prior to the onset of field outbreaks in the affected area. This situation clearly demonstrates the dangers inherent in the practice of storing maize on cob in cribs.

Although the observed effects of zearalenone ingestion in the field outbreaks reported here were apparently limited to swelling of the vulva and/or teats in juvenile pigs, zearalenone can cause much more serious effects including multiple reproductive disorders such as infertility, constant oestrus, pseudopregnancy, reduced litter size, smaller offspring, and malformation[1,2]. Moreover, zearalenone often "keeps bad company" in that it frequently occurs together with other *Fusarium* metabolites such as the trichothecenes which may cause feed refusal, emesis, and mortality characterized by haemorrhage, pancytopaenia and immunosuppression[4,5,10,11,13]. The ingestion of zearalenone-containing feedstuffs by livestock is also a potential threat to human health since zearalenone is transmitted into bovine milk in the form of the parent compound as well as the metabolite zearalenol[12], and zearalenol is 3–4 times more oestrogenic than zearalenone[14].

In view of the above considerations, it is important that the ingestion of zearalenone containing feedstuffs by livestock should be avoided. The practice of storing maize on the cob in cribs should be discouraged. If clinical signs of hyperoestrogenism in pigs are noticed, contaminated feed should be withdrawn and replaced with good quality maize from other sources. Alternatively contaminated maize should be diluted with good quality maize. Under no circumstances should *Fusarium*-infected maize be fed to juvenile pigs, breeding stock, or dairy cows.

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OBSERVATIONS ON THE INFLUENCE OF HIGH LEVEL FEEDING ON THE OVARIAN ACTIVITY AND FERTILITY IN DAIRY COWS

C. MAREE

ABSTRACT: Maree C. **Observations on the influence of high level feeding on the ovarian activity and fertility in dairy cows.** *Journal of the South African Veterinary Association* (1980) **51** No. 3 167–170 (En) Department of Animal Production, University of Pretoria, Pretoria 0002, Republic of South Africa.

Cows on high and low feeding levels were submitted to palpation of the ovaries and recording of the presence and size of follicles at various days of the cycle. A mid-cycle rise in follicular activity was apparent and for which the nonovulating ovary was responsible. High level feeding stimulated ovarian activity in each of the 2 ovaries throughout the cycle and follicular activity commenced earlier in the ovulating ovary on high level feeding than on low level feeding. A larger number of inseminations per conception was required by cows on high level feeding than on low level feeding. The effect of ovarian activity on fertility is discussed.

INTRODUCTION

The genesis of the reproductive system endowed the female mammal with paired ovaries that appear to function independently of each other. No systemic sequence of ovulations from one ovary to another or any differential fertility between the 2 ovaries has been established[11]. While the distribution of ovulations is uneven and in the order of 60% and 40% between the right and left ovaries respectively[6 11 16 18 20] waves of ovarian follicles develop between the 3rd and the 4th d of the cycle and again between Days 12 and 14 while the period 9–12 d is characterised by the secretion of large amounts of oestrogen and progesterone[15].

The level of nutrition affects ovarian activity and reproductive ability significantly. Ovulation rate, follicle size and numbers of follicles as well as fertility rate are increased but embryonic survival rate is significantly decreased by high feeding levels[1 3 8 19 22].

In this investigation the effect of *ad libitum* and controlled feeding levels were recorded in relation to the fertility of dairy cows and on the respective levels of activity of their ovulating and non-ovulating ovaries.

PROCEDURE

Twenty-four Friesland cows were divided at random into high and restricted level feeding groups. High level feeding was based on the average *ad libitum* daily feed intake of the group over 58 d while the restricted feeding level was based on calculated requirements for maintenance and production. Average daily feed intake during lactation was 12,7 kg dairy concentrate and 2,38 kg *Eragrostis curvula* hay for the high level group and 4,1 kg concentrate and 8,06 kg hay for the restricted group. During the dry period intakes were 9,1 and 1,4 kg concentrate and 2,56 and 9,4 kg hay respectively. Concentrate consisted of 300 parts by weight of yellow maize meal and 100 parts high protein concentrate (40% protein and 65% TDN).

Cows were inseminated at 2 breeding periods over 22 months with 3 repeat inseminations at return to oestrus, covering a total 374 cycles.

Ovarian activity was recorded by palpation of the ovaries and description of the follicles at half weekly intervals. At oestrus, examinations were carried out daily until ovulation or regression of the follicle was confirmed. Follicles were classified as large (more than 25 mm in diameter), medium (15–20 mm in diameter) or small (up to 15 mm in diameter) and allocated numerical index values of 1, 2 or 3 respectively so that an average value of ovarian activity could be calculated for different days of the cycle.

RESULTS AND DISCUSSION

The results of inseminations in the 2 groups are provided in Table 1 while Fig. 1 (a)[13] illustrates the numerical index of follicular activity in both ovaries on each day of the cycle in the 2 groups. Fig. 1(b)[13] illustrates the numerical index for follicular activity in the ovulating and non-ovulating ovaries respectively for the 2 groups.

In addition to a lower conception rate and an in-

Table 1: INSEMINATION AND PREGNANCY RATE OF COWS ON A HIGH AND A RESTRICTED LEVEL OF FEEDING AT 2 SUCCESSIVE BREEDING PERIODS.

	HIGH FEEDING LEVEL		RESTRICTED FEEDING LEVEL	
	First breeding period	Second breeding period	First breeding period	Second breeding period
Number of cows	12	10	12	12
Cows inseminated	12	6	12	12
Cows pregnant	10	4	12	12
Insem/pregnancy	2,2	2,8	1,3	1,2

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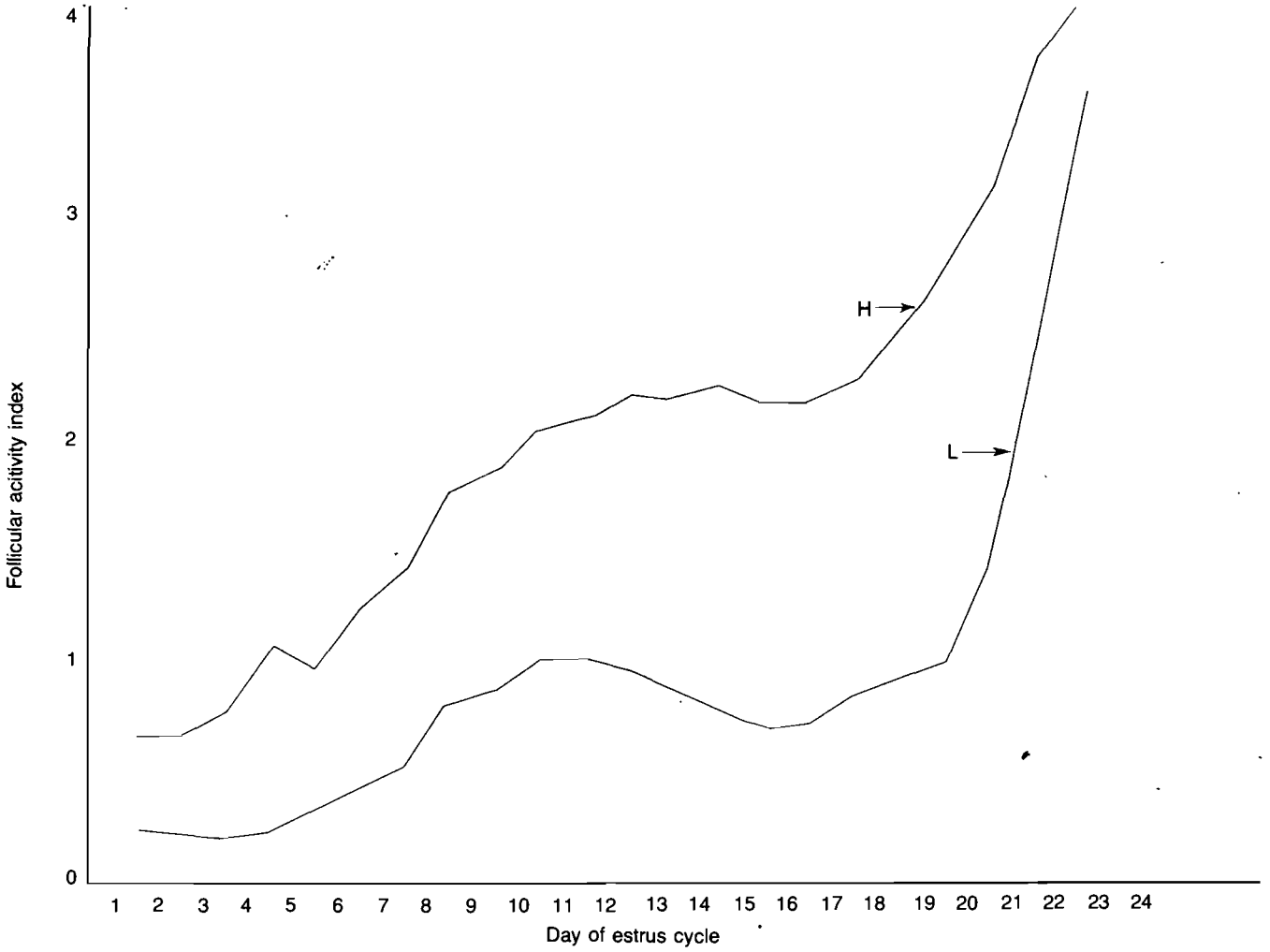

Fig. 1(a) Numerical index value of follicular activity in both ovaries on each day of the cycle in cows on a high (H) and a low (L) level of feeding.

creased number of inseminations required per pregnancy in the group on a high feeding level, it is clear that fewer cows conceived and also that fewer cows survived on this treatment. Erosion was due to mortality from metabolic disease (2 cows) and prolonged anestrus after calving following post partum complications (4 cows).

At oestrus it was not unusual to encounter a well developed follicle in each of the 2 ovaries or more than one follicle on the ovulating ovary, one of which then ripened for ovulation while the others regressed within a day or two or sometimes over several days.

The distribution of function between the 2 ovaries was odd and without any consecutive order with 59,4% of ovulations in the right ovary and 40,6% in the left ovary, figures which correspond with previous reports[6 11 16 18 20]. The hypothesis that the function of each ovary was completely at random and without any tendency for cows to have a systemic sequence of ovulations from one ovary to the other was tested conclusively, while no differential fertility could be established between the 2 sides from 138 ovulations in the right ovary and 114 in the left ovary[11].

Fig. 1(a) and 1(b)clearly illustrate the effect of nutritional level on ovarian activity throughout the cycle and on the respective levels of activity in the ovulating and non-ovulating ovaries. Immediately after ovulation a higher level of activity was recorded in the non-ovulating ovary in cows on high level feeding. A mid-cycle rise in follicular activity reported for the bovine[15] is substantiated by Fig. 1(a) and 1(b) and it appears that the non-ovulating ovary is responsible for this. Follicular activity in the ovary destined to ovulate is initiated at an earlier stage of the cycle in cows on high level feeding (Fig 1(b)). Well developed follicles were regularly recorded 6 to 8 d before oestrus in the highly fed cows while, in the restricted group, follicular activity before ovulation started 2 or 3 days before oestrus and frequently only one day before. It has been shown however, that only a few hours are required to bring a 10 mm follicle to ovulation size in the bovine[5].

The question arises whether decreased fertility observed in the high level cows can be related to excessive ovarian activity. The bovine ovaries respond readily to fluctuations in nutritional level[13] and this is applied as a valuable aid in the improvement of fertility at lower levels of feeding. Numerous reports are available however that high level feeding reduces fertility and productive lifespan while the incidence of erosion diseases is increased[2 7 9 12 14 17]. Table 1 again supports the findings of several investigations that a larger number of services are required to settle cows on high level feeding [4 10 12]. Although the direct mechanisms involved are unexplained it has been shown that grain feeding increases total pituitary FSH and LH potency and follicular development[3] while, in undernourished animals

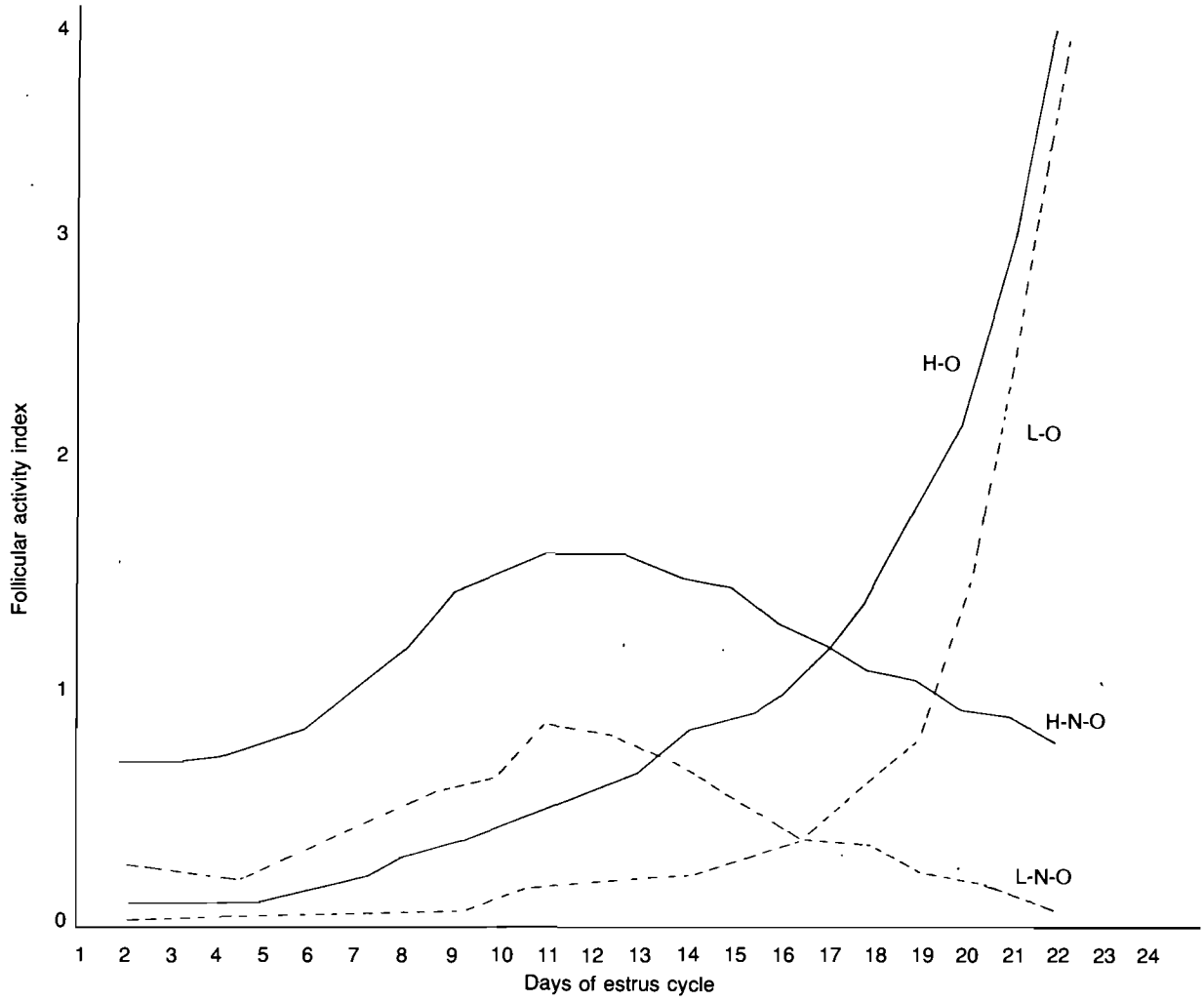

Fig. 1(b) Numerical index values of follicular activity on each day of the cycle in the ovulating (O) and non-ovulating (N-O) ovaries respectively in cows on a high (H) and low (L) level of feeding.

again carbohydrate histochemistry of the endometrium and oestrogen/progesterone relationship were altered[21]. The possible acceleration of pituitary and follicular hormonal release by overfeeding and the consequent possibility of disturbances in the reproductive process in terms of pre-ovulatory ripeness of the ovum or alterations in tubal and uterine environment, appear to require further investigation.

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BOOK REVIEW BOEKRESENSIE

LEHRBUCH DER SCHAFKRANKHEITEN

HEINRICH BEHRENS

Second, revised edition, 1979. Publisher Paul Parey. 291 pages, 84 black and white and 12 colour illustrations. Price DM98.

This excellent and extremely useful book is written in an easily understandable German and should be included in all veterinary libraries. It is divided into 10 sections namely: Infectious diseases, parasitic diseases; diseases of the respiratory, digestive, urinary and reproductive organs; metabolic diseases; deficiency diseases; infertility; poisonings; hereditary diseases; common surgical operations and a general section.

Every disease or condition, with equivalent English nomenclature, is short, down to the point and includes a definition, important criteria or characteristics and its distribution. Subheadings are: aetiology, pathological-anatomical changes, symptoms, diagnosis and differential diagnosis, therapy, prophylaxis and some recent references. The English equivalent to each disease is very useful as often the German name of a disease cannot be translated, eg. "Traberkrankheit" – Scrapie or "Lippengrind" – Orf.

This book also includes some diseases which have only recently become of greater importance and include reovirus and rotavirus infections, terminal ileitis and others. Some lesser known diseases such as listeriosis, Q-fever and toxoplasmosis are dealt with in greater detail; this is wellcome and necessary.

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The section on parasitology is well balanced, is divided into protozoal, helminth and arthropod-caused disease conditions and includes 2 pages of good photographic reproductions of helminth eggs and oocysts. Epididymitis is discussed fully, but does not refer to *Actinobacillus seminis* as a causative agent. Metabolic conditions are clearly defined and photosensitivities are discussed in greater detail. Reference is also made to *Phitomyces chartarum* causing facial eczema in the RSA.

Deficiency diseases include cerebrocortical necrosis, cobalt deficiency, swayback, white muscle disease, rickets and others. The section on infertility is short and only deals with physiological conditions. Poisonings are subdivided into inorganic, organic and plant poisons. The latter, understandably so, is of lesser interest to us.

Although this book was written to cover important disease conditions occurring in Central Europe, it is also an important and practical publication for South African veterinarians, students and biologists and will fill a long needed niche.

I. F. Zumpt

NOTES ON THE DETERMINATION AND THE OCCURRENCE OF SOME REPRODUCTIVE DERANGEMENTS IN A GROUP OF FRIESLAND COWS

C. MAREE

ABSTRACT: Maree C. **Notes on the determination and the occurrence of some reproductive derangements in a group of Friesland cows.** *Journal of the South African Veterinary Association* (1980) **51** No. 3 171-172 (En) Department of Animal Production, University of Pretoria, Pretoria 0002.

Cyclic activity and palpable features of the bovine genitalia were recorded by means of observation and a prolonged series of follow-up palpations. Various uncommon features concerning follicular development, oestrus performance, ovulation and the corpus luteum are described together with observations on the cystic enlargement of follicles and corpora lutea. Cases are reported of ovarian haemorrhage at oestrus and cystic enlargement of the endometrium.

INTRODUCTION

Rectal palpation of the bovine genitalia has over many generations been accepted as a straight forward technique which is relatively simple to the trained operator and consequently needless of further revision. However, clinicians engaged in fertility work will agree that differences of opinion, untoward responses to treatment and outright blunders are not exceptional whenever results of palpations are controlled. In this connection some explanations are suggested in the observations to be presented.

The ovulatory efficiency of 32 Friesland cows was investigated over 568 cycles by means of rectal palpation of the genitalia at half-weekly intervals with follow-up palpations at oestrus every 24 h until ovulation or regression of the follicle was apparent. Certain features and palpable variations of structures were confirmed by these follow-up palpations that would undoubtedly have escaped detection at a single examination. Critical reports on the palpable features of the bovine genitalia are not readily available and since the clinician frequently has to rely on a single palpation, some of the features and variation in structure that were observed are now presented to indicate the ease with which inaccuracies can be recorded through use of this very common technique.

OVULATION AND OESTRUS PERFORMANCE

It is possible to establish with reasonable accuracy whether or not a follicle is ready to ovulate. The ripe follicle is well defined, soft and thin walled and not distended with fluid. At ovulation, collapse of the follicle is palpable as fluid oozes out slowly. This is in distinct contrast to the rapid release of follicular fluid when a follicle is ruptured by pressure. In case of anovulation, the follicle feels firm and thick-walled and does not rupture readily. This confirms a report by Van Rensburg and De Vos[10] that ripening causes increase in size and softening of the follicle. These observations fit in with reports that ovulation is a gradual process which depends on progressive changes in the structure of the follicle with intrafollicular pressure not being the essential mechanism in follicular opening[3] but in which enzymically induced weakening of the follicular wall have been suggested as a contributory mechanism[5].

Manifestations of oestrus vary at the onset in relation to the appearance of vaginal discharge, the mounting of and acceptability to other animals and particularly the intensity and frequency of these signs so that the full manifestation of oestrus frequently develops only over several hours. The exact end-point of oestrus is likewise often vague due to the gradual and odd abatement of signs so that the determination of the duration of the oestrus period is frequently subject to inaccuracy.

FOLLICULAR AND CORPUS LUTEUM DEVELOPMENT

Whilst direct measurement of follicles or corpora lutea is naturally impossible on palpation these structures can with relative ease be classified into categories as small (up to 15 mm in diameter), medium (15–20 mm in diameter) and large (more than 25 mm in diameter). Considerable variation between animals was recorded in regard to the size of the Graafian follicle at oestrus. Some animals developed small follicles at oestrus and sometimes for a few cycles in succession to be followed by oestrus periods with large Graafian follicles.

The intensity of oestrus behaviour and the relative incidence of normal ovulations, delayed ovulations or anovulations could not be related to the size of the Graafian follicle at oestrus. Small follicles softened and ovulated as readily as large follicles while delayed ovulation or anovulation occurred with large or small Graafian follicles alike.

Corpus luteum size was likewise classified as small, medium or large on palpation and could not be related to the size of its Graafian follicle at oestrus. a large corpus luteum was frequently recorded after a small follicle and vice versa. Considarable variation was futher recorded in the size, shape and consistency between different corpora lutea and even in the same corpus luteum at different palpations. A corpus luteum recorded as soft and flabby was sometimes recorded as firm and solid at a following palpation and vice versa and would easily have been confused with a follicle but for repeated follow-up palpations.

It is relatively easy to distinguish the newly developed corpus luteum by its typical crepitation on moderate pressure from as early as the second to the third day after ovulation and sometimes as soon as one day. From the middle to the end of the luteal phase, however, much firmer pressure is required to produce crepitation which in several cases could not be produced even when splitting or partial enucleation of the corpus luteum resulted. Neither did this practice have any apparent effect on the life span of the corpus luteum or on cyclic activity which only resulted after complete enucleation.

Cystic corpora lutea are a common feature and resemble follicles on palpation. Fluid escapes readily on pressure and may reaccumulate but usually disappears by mid-cycle. In one case fluid escaped at three successive palpations from a corpus luteum. The corpus lu-

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teum of pregnancy was found to be cystic on several occasions in the early stages without any apparent effect on the course of pregnancy which fits in with reports that the presence of fluid in a corpus luteum is incidental to its development[4,7,8]. Evidence is available though that, in sheep, the presence of a cavity in the corpus luteum was induced by stress[1]. In cattle again the incidence of cystic corpora lutea was related to the amount of handling to which they were subjected[2] while season also had a significant influence on its frequency[8]. Cystic corpora lutea were also reported to be induced by ocytocin but these produced little progesterone and were unlikely to sustain pregnancy[9].

A persistant corpus luteum is frequently but not always, associated with prolonged cycles which were recorded well after regression of the corpus luteum had taken place or following anovulation where no corpus luteum had taken place or following anovulation where no corpus luteum developed.

FOLLICULAR CYSTS

Follicular cysts were recorded in four out of 32 heifers before the age of 12 months with a spontaneous return to normal after 2–3 months in 3 cases. Unilateral enlargement of the uterus up to the size of a 3 month pregnancy occurred in 2 of these cases and lasted for approximately the same period. Follicular cysts developed from an unovulated follicle at oestrus (1 case) and from follicular activity during mid-cycle (3 cases).

After calving, development of cystic follicles were recorded only in association with metritis and periovarian adhesions. Cystic enlargement of the endometrial glands on histological examination after slaughtering were recorded in 3 animals, only 1 of which had a record of cystic ovaries on palpation or post mortally. Cystic changes in the endometrium in the other 2 cases could not be related to a history of cystic changes on palpation in the ovaries at any stage or at slaughtering although, according to Jubb and Kennedy[6], cystic endometrial hyperplasia in cows is invariably associated with ovarian follicular cysts and hyperoestrogenism.

HAEMORRHAGE AND ADHESIONS AFTER OVULATION

Two cases of spontaneous haemorrhage around the ovary were recorded at oestrus, possibly at ovulation, the ovaries and the Graafian follicles having felt quite normal at oestrus and the clots were discovered at a follow-up palpation after 24h, in one case the size of a golf ball and in the other as big as a man's fist. Firm adhesions resulted eventually and in which the ovaries became embedded. Cyclic activity proceeded uninterruptedly though with the ovaries in the adhesions being responsible for oestrus at some oestrus periods during which enlargement of the adhesion was palpable with no activity on the opposite ovary. Impairment of the function of one ovary through adhesions therefore did not seem to interfere with the function of the opposite ovary. Neither did repeated handling of the ovaries during palpation, even over prolonged periods, affect ovarian or cyclic activity in any way. Normal activity and function were spontaneously followed by temporary disturbances and it can be surmised that the control mechanism of reproductive activity are subject to constant derangement and re-establishement.

It is clear that a single palpation of the reproductive system, at whatever stage of the cycle, is of little value as a method to estimate the reproductive potential of the cow. Furthermore, the accuracy with which follicles and corpora lutea can be recognized on palpation is severely influenced by the normal vatiation in the palpable features of these structures.

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THE MICROMORPHOLOGICAL DEVELOPMENT OF THE POST PARTUM CORPUS LUTEUM IN THE EWE.

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ABSTRACT: Botha H. K.; van Niekerk C. H. **The micromorphological development of the post partum corpus luteum in the ewe.** *Journal of the South African Veterinary Association* (1980) **51** No. 3 173–177 Dept. Physiology, Univ. Orange Free State, Bloemfontein, Rep. of South Africa.

The morphological changes in the post partum corpus luteum was investigated by light microscopy on 16 ewes during the normal breeding season (March/April).

The increase in luteal tissue results from hypertrophy and not from hyperplasia of the granulosa and thecal cells. Two types of lutein cells were found in the corpus luteum: a large light-staining cell and a small dark-staining cell. These might reflect the 2 extremes of a spectrum of cells in different functional states, or they might represent cell lines derived from the follicular granulosa and the thecal interna, respectively.

INTRODUCTION

Although results in connection with the development and degeneration of the corpus luteum (CL) of ewes during a normal oestrus cycle are described in the literature[1,2], no description recording the development and regression of the post partum CL could be found. Immediately after parturition, it was found that one or more follicles develop, but no indication was given of the development of the CL[3].

A study of the involution of the post partum uterus of ewes[4] offered an opportunity to investigate the post partum changes taking place in the CL under the light microscope and to add to the understanding of CL histology, from the time of ovulation to maturity.

MATERIALS AND METHODS

Sixteen S.A. Mutton Merino ewes were used after parturition in the normal breeding season. Sexual activity was monitored twice daily by the use of vasectomized rams for 36 d post partum. The onset of oestrus was designated as Day 0. The ewes were slaughtered at different stages of the oestrus cycle. Corpora lutea were removed from the overies as quickly as possible by dissection and were fixed in Zenker-formol. The samples were sectioned at 6μm and stained according to standard histological techniques. The development and regression of the CL was investigated under the light microscope, with special emphasis on the changes taking place in the lutein cells.

RESULTS AND DISCUSSION

Following ovulation and discharge of the liquor folliculi, the wall of the follicle collapses, and its granulosa cell lining is thrown into folds[1,5]. Some bleeding into the central cavity takes place (Fig. 1). The plicae are retained as the follicle is transformed into a CL. During the process of lutinization the cells of the plicated granulosa and theca interna layers then undergo striking cytological changes.

Between 24 and 30 h after ovulation, depolymerization of the basement membrane, that originally separates the granulosa cell layers from the theca layers, takes place[1]. This allows the capillaries from the theca interna to sprout and invade the lutein tissue (Fig. 2). A complex network of capillaries forms throughout the gland. Connective tissue elements form a delicate reticulum around the lutein cells (Fig. 3). The blood clot in the central cavity is gradually replaced by connective tissue forming a fibrous core.

A rapid increase in the diameter of the CL was found between 30 h and 5 d following ovulation. This development continued for the next 5 d, but at a decreased rate. The increase in luteal tissue mass was probably not due to hyperplasia, but apparantly resulted from hypertrophy, as hardly any mitotic figures in the lutein cells could be observed. These findings differ from those in the cow[9,10,11] but is in agreement with those in the pig[6], monkey[7], goat[8] and mare[5].

Two kinds of lutein cells are distinguishable in the CL, namely those making up the bulk of the lutein tissue and probably derived from the granulosa cells and those probably originating from the cells of the theca interna (Fig. 3). The latter are smaller, more deeply staining and nearer to the periphery of the CL (Fig. 3 and 4).

Within 2–3 d after ovulation the luteal cells of the developing CL begin to undergo cytomorphosis. They enlarge, are transformed into plump, pale-staining polygonal cells, finely granulated with slight vacuolation, indicating the activation and transformation into progesterone secreting lutein cells (Fig. 5). The transformation into progesterone secreting cells was also reflected in an increase in the peripheral plasma progesterone concentration[4].

From the third d following ovulation an increased number of granulosa cells were progressively transformed into lutein cells. Secretory activities of these cells had already commenced as were indicated both by the fine vacuolization (Fig. 5) of the cytoplasm as well as the presence of detectable concentrations of progesterone in the pheripheral plasma[4]. From the sixth d after ovulation the hypertrophied lutein cells were more densly grouped and the intercellular spaces became smaller (Fig. 6). Maximum hypertrophy of the lutein cells occurred within 9–10 d after ovulation (Fig. 7), after which the sizes of the lutein cells progressively began to decrease (Fig. 8). These findings were also reflected in the peripheral plasma progesterone concentration[4].

From the third to fourth d following ovulation 2 types of lutein cells, namely a large light staining and a small dark staining cell could be distinguished (Fig. 5, 6 and

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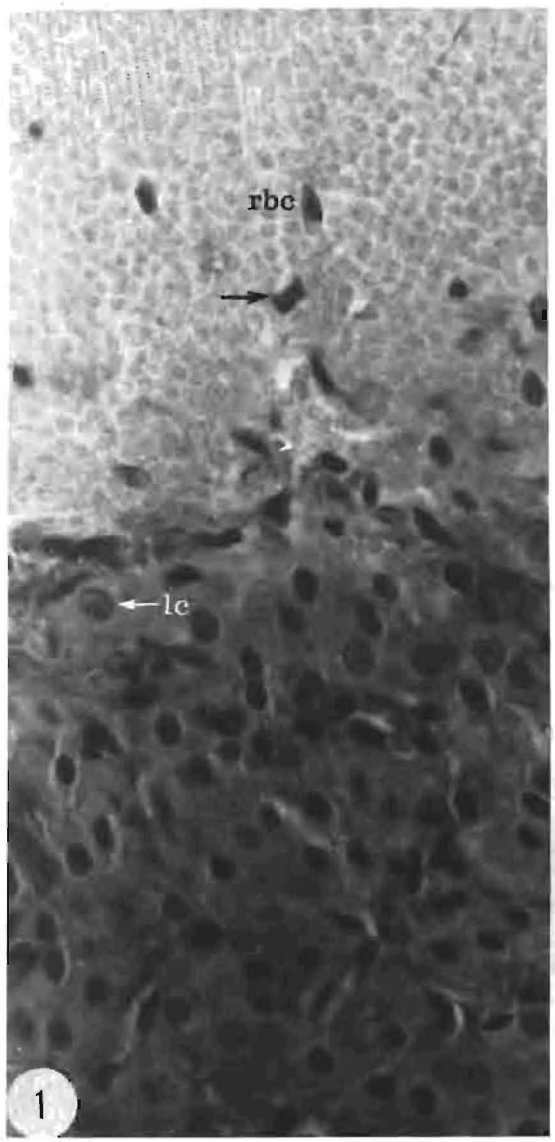


Fig. 1. Post partum corpus luteum 36 h after ovulation showing the red blood cells (rbc), some fibroblasts (arrow) and lutein cells (Lc). x 510.

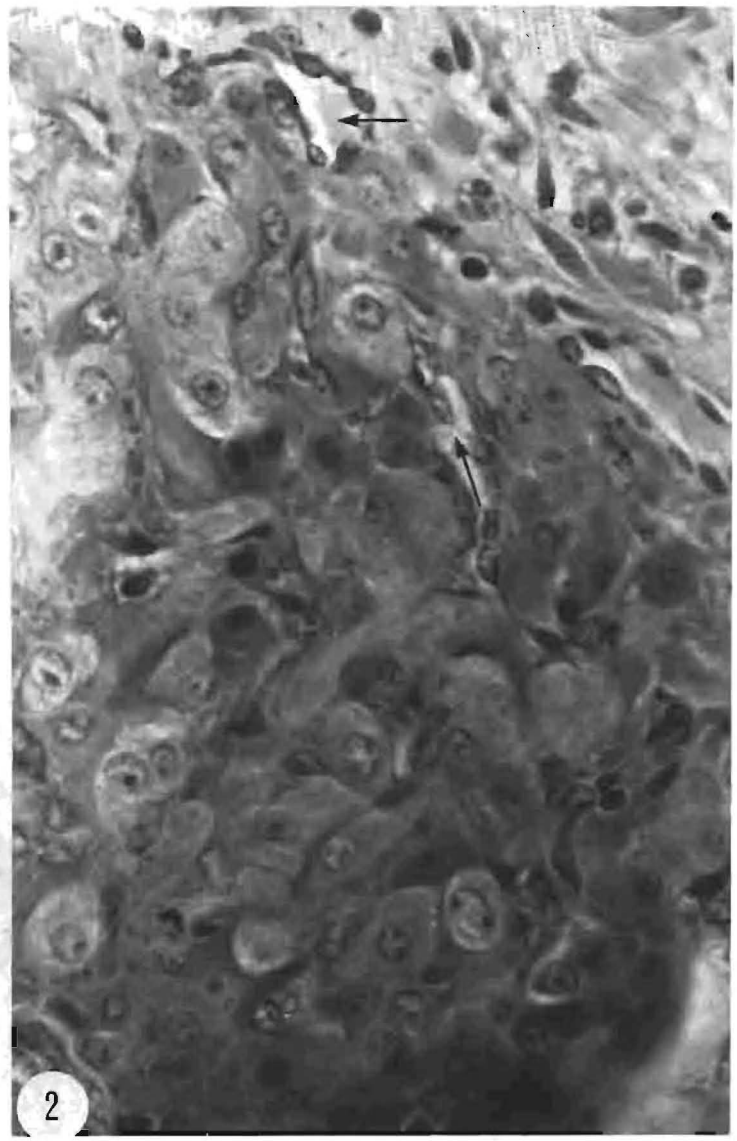


Fig. 2. Post partum corpus luteum 3 d after ovulation showing the invasion of the capillaries (arrows). x 510.

7). The activity of the large, polygonal light staining cells was reflected by the large amount of finely granulated cytoplasm and the large vesicular nuclei with a clearly visible nucleolus. In sharp contrast to these large lutein cells are small cells having a shrunken homogeneous eosinophilic cytoplasm and a condensed, darkly stained nucleus (Fig. 5, 6 and 7). The smaller of the 2 cell types is more attenuated and often spindle-shaped. The larger cell has convex surfaces and is angular in profile with a more rounded nucleus. These 2 different cell types were also observed in the CL of the mare[5].

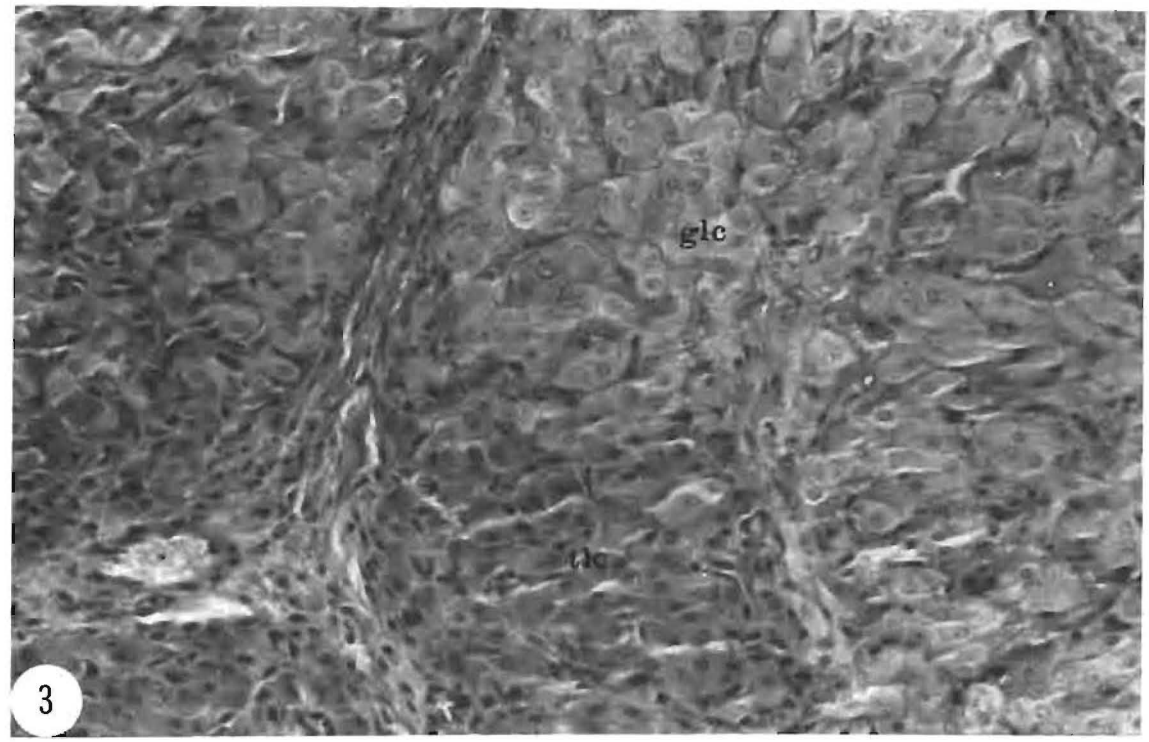
The dark cells increased in number from only a few on the fourth d following ovulation to about an estimated 5% of the total lutein cell population on the fith to sixth d and 10% on Day eight. Thereafter they increased to an estimated 20% on Day 10.

These cells might reflect the 2 extremes of a spectrum of cells in different functional states, or they might represent cell lines derived from the follicular granulosa and the theca interna, respectively. In the mare it was considered that the small dark cells might be in a resting stage and that they cease to be converted into the large, light variety when the latter begins to degenerate[5]. The eventuality exists that these dark staining cells may represent theca interna cells that have infiltrated, from the periphery of the CL in between the granulosa lutein cells.

After 10 d following ovulation, the CL began to decrease in diameter. On Day 12 after ovulation degeneration of the luteal cells was in an advanced stage (Fig. 8). The luteal cells became pyknotic, while hyaline intercellular material began to accumalate. In this condition the cytoplasm of the lutein tissue cells became shrunken and strongly eosinophilic. Peripheral vacuoles coalesced, resulting in the formation of elongated open spaces. Pyknotic nuclei began to predominate over vesicular nuclei.

On Day 14 after ovulation the degenerative changes were in a more advanced stage and the CL could now histologically be considered as inactive. This was also reflected in a rapid decrease of peripheral plasma progesterone concentration[4]. Results of the present investigation would therefore suggest that there are no

Fig. 3. Post partum corpus luteum 3 d after ovulation indicating the trabeculae of connective tissue, and 2 kinds of lutein cells. glc – granulosa lutein cells and tlc – theca lutein cells. x 256.



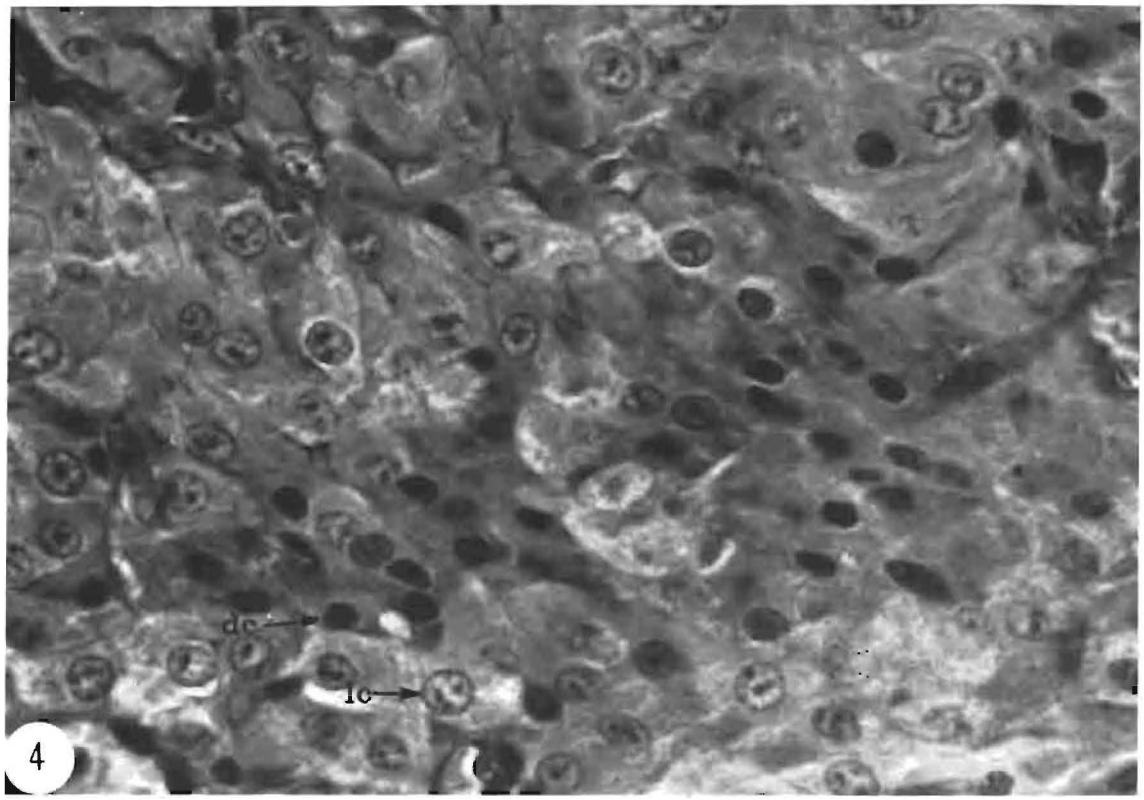


Fig. 4. Post partum corpus luteum 3 d after ovulation showing the 2 distinct lutein cells. Light cells (lc) and dark cells (dc). x 510.

differences in CL development and degeneration between the first post partum CL and that of a normal estrus cycle, as described in various previous investigations [1,3,12].

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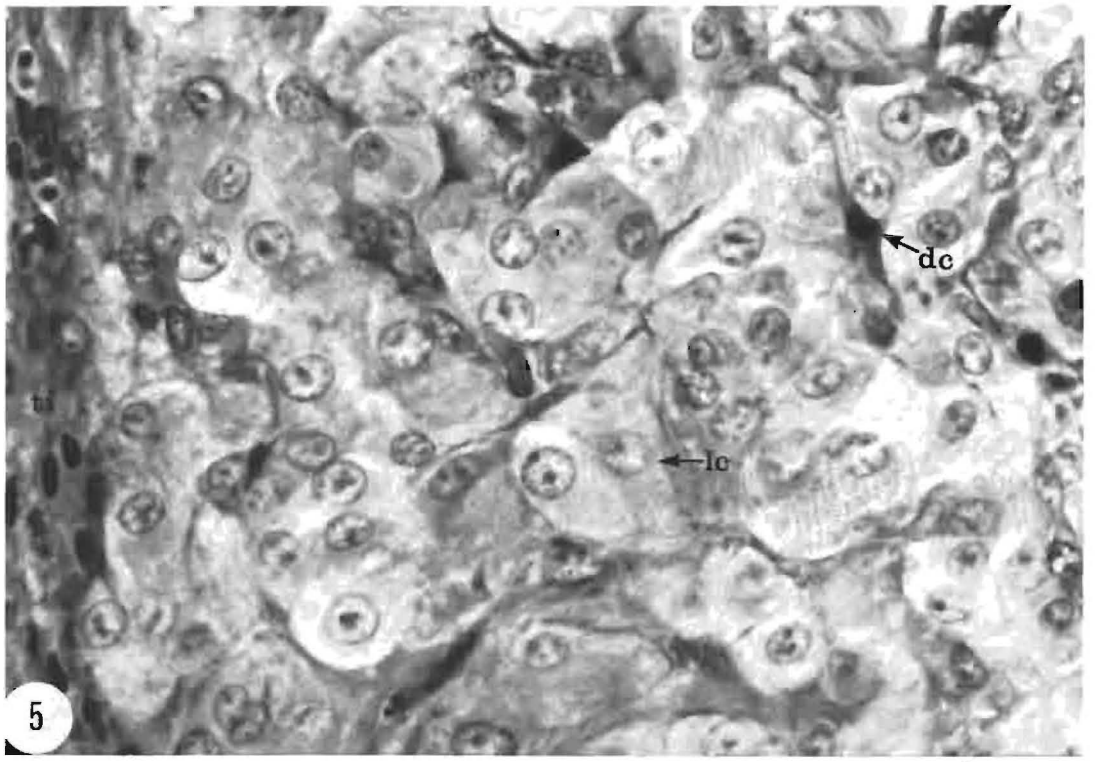
Fig. 5. Post partum corpus luteum 4 d after ovulation indicating the theca interna (ti), the light cells (lc) and the dark cells (dc). x 510.



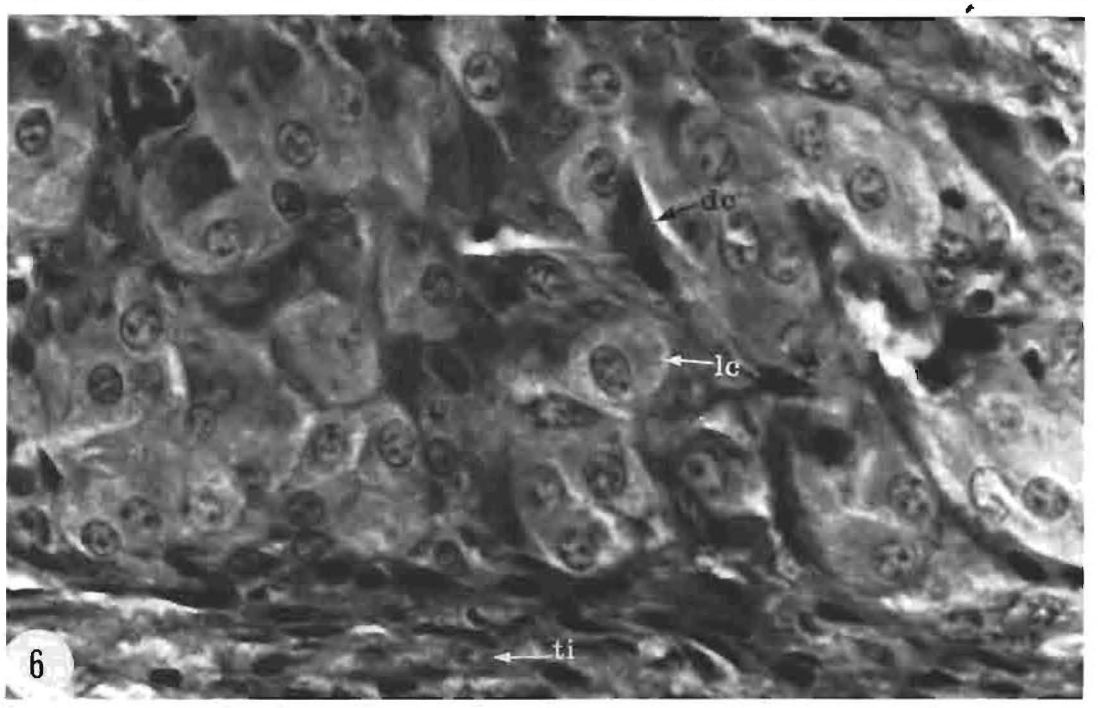


Fig. 6. Post partum corpus luteum 6 d after ovulation clearly showing the difference between the light (lc) and dark lutein cells (dc). ti – theca interna layer. x 510.

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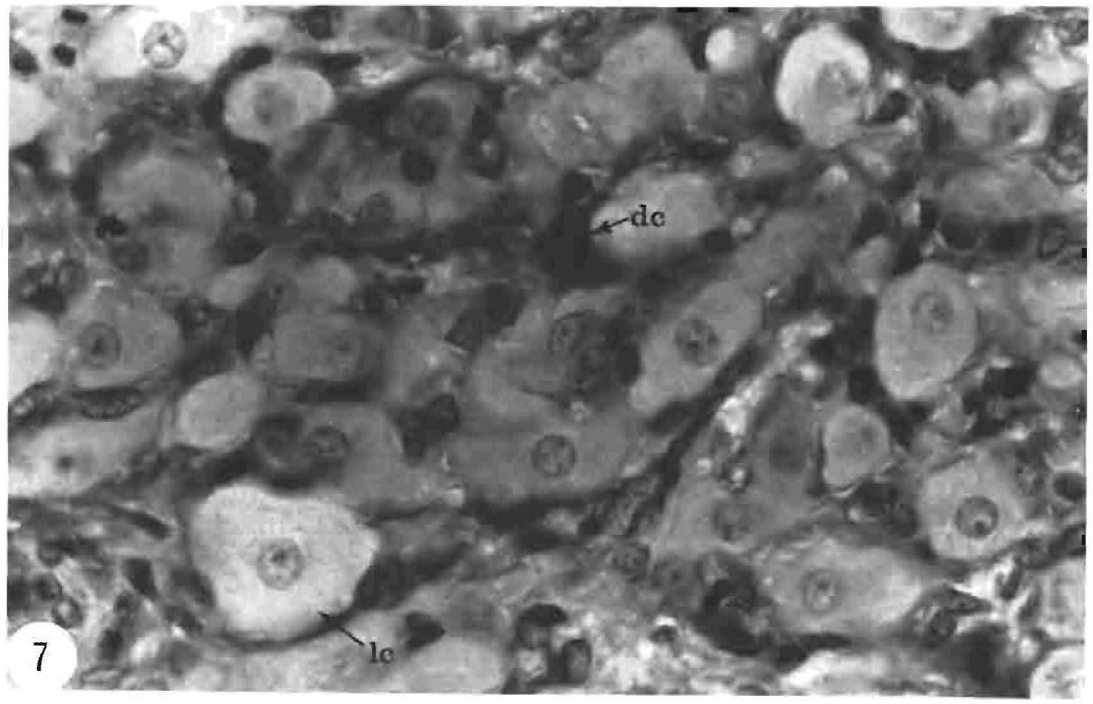
Fig. 7. A section through the post partum corpus luteum 9 d after ovulation indicating the maximum hypertrophy of the lutein cells. lc – light cells; dc – dark cells. x 510.



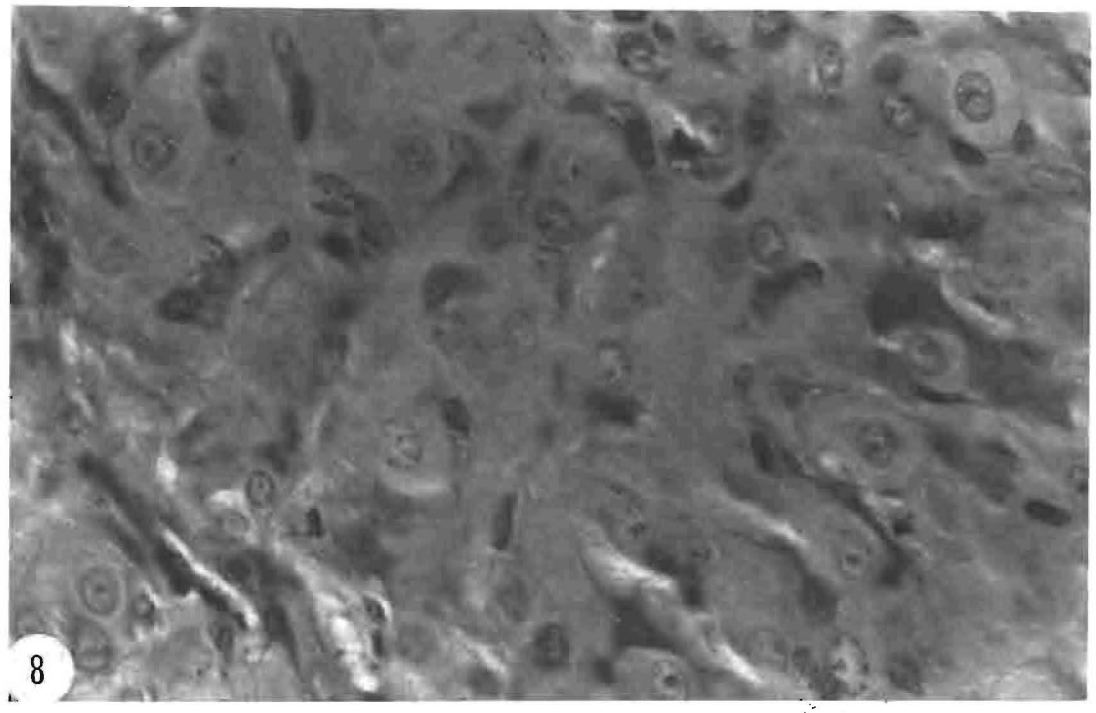


Fig. 8. Post partum corpus luteum 12 d after ovulation showing the degeneration of the lutein cells. The lutein cells are clearly smaller than in Fig. 7, and there seems to be an increase in the amount of stroma between them. x 510.

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DIE GEBRUIK EN MISBRUIK VAN HORMONE IN GESLAGSKUNDIGE GEVALLE

H. M. TERBLANCHE

ABSTRACT: Terblanche H. M. **The use and abuse of hormones in genesiological cases.** *Journal of the South African Veterinary Association,* (1980) **51** No. 3 179–183 (Afr). Dept. Genesiology, Fac. Vet. Science, Box 12580, 0110 Onderstepoort, Rep. of South africa.

The correct use of hormones in genesiological cases is discussed with indications for their use, dosage levels and duration of treatment under the following groups, viz. hypothalamic hormones, hypophyseal hormones, other gonadotrophins, steroids, combinations of the foregoing and prostaglandins. The more important dangers associated with hormone therapy are then briefly discussed with a few examples of the more common and hazardous abuses.

INLEIDING

Hormonale terapie is vandag 'n algemeen aanvaarde praktyk in die geneeskundige en veeartsenykundige andrologie, ginekologie en obstetrie. Dit word in 'n wye spektrum van gevalle gebruik maar die onverskillige en verkeerde gebruik van hormone kan soms aanleiding gee tot voortplantingsprobleme, verlies van potensiële goeie genetiese materiaal en in enkele gevalle ook toksiese simptome met afsterwe van die pasiënt.

Dit is derhalwe gewens om eerstens te let op die korrekte gebruik van die belangrikste hormone wat meer dikwels in die praktyk gebruik word vir die behandeling van geslagskundige gevalle en om tweedens 'n aanduiding te gee van die potensiële gevaar wat bestaan by die misbruik van hierdie hormone asook enkele voorbeelde van sodanige misbruik. Handelsname wat in hierdie artikel gebruik word, word volledigheidshalwe verstrek en die volgorde dui geen noodwendige voorkeur aan nie.

KORREKTE GEBRUIK

Hipotalamiese hormone

In hierdie groep val slegs die vrylatingshormone van follikel stimulerende hormoon (FSH) en luteiniserende hormoon (LH) bekend as gonadotrofien vrylatingshormoon (GnVH) of gonadoliberien. Hierdie hormoon word sinteties vervaardig en is onlangs bemark as Buserelin (0,004 mg/mℓ). (Receptal, Hoechst Farmaseutika (Edms) Bpk.)
Die middel word aanbeveel vir die volgende toestande[1]:

Koei

(a) Follikulêre siste; 0,02 mg aktiewe bestanddeel i.m. Herhaal na 10–14 d indien nodig.
(b) Anoestrus; 0,02 mg aktiewe bestanddeel i.m. Herhaal na 10–14 d indien nodig.
(c) Vertraagde ovulasie; 0,01 mg aktiewe bestanddeel i.m. Ovulasie binne 24 h. Spuit met kunsmatige inseminasie of tot 6 h vantevore.

Merrie

(a) Induksie van ovulasie; 0,04 mg aktiewe bestanddeel i.m. Ovulasie binne 24–36 h[1].
(b) Verkorting van verlengde oestrus in vroeë teelseisoen; 0,04 mg aktiewe bestanddeel i.m.

Sog, ooi, teef

(a) Eksperimentele data nie beskikbaar nie
(b) Teoreties sou 0,002–0,004 mg aktiewe bestanddeel i.m. in sôe en ooie en 0,0008–0,002 mg aktiewe bestanddeel i.m. in tewe gebruik kon word.

Manlike dier

(a) Geen eksperimentele data beskikbaar nie.
(b) Kan moontlik gebruik word vir die stimulasie van oligospermie en/of lae libido teen 'n dosis van 0,02–0,04 mg aktiewe bestanddeel i.m. in bulle en hingste.

Hipofiseale hormone

Gonadotropiene

(a) Luteiniserende hormoon (LH)
Sien later onder ander gonadotropiene
(b) Follikel stimulerende hormoon (FSH)
Onder hierdie groep is daar teenswoordig 2 preparate nl. FSH–P (Burns-Biotec) en Apocrine A (Bayer Pharmaceuticals (S A) (Edms) Bpk.). Hierdie middels kan gebruik word vir stimulasie van FSH-afhanklike prosesse, bv. follikulêre ontwikkeling en spermatositogenese. FSH-P word algemeen gebruik vir superovulasie in samehang met embrio oorplasings.
(c) Kombinasies (FSH en LH)
In hierdie kategorie was 'n preparaat tot onlangs beskikbaar bekend as Vetrophin (Abbott). Dit bestaan uit 5 mg elk van FSH en LH/mℓ en is vrugbaar gebruik in die departementele hospitaal* in manlike diere met oligospermie en/of lae libido. Behandeling is in die vorm van 'n kursus gegee, 5 mg i.v. 2 keer per week vir 6 weke in groot diere terwyl honde op dieselfde wyse behandel is teen 'n dosis van 2,5 mg i.v. 2 keer per week vir 6 weke.

Oksitosien

Hierdie middel is beskikbaar as Oksitosien (Ciba-Geigy (Edms) Bpk. en Ferring A.B.). Dit word vir 'n verskeidenheid van gevalle gebruik in meeste praktyke, nl.:
(a) Stimulasie van baarmoeder kontraksies tydens distokie in tewe waar die serviks ontsluit is en geen obstruksie in die geboortekanaal teenwoordig is nie. Die dosis varieer tussen 2 en 8 eenhede

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(0,2–0,8 mℓ) s.c.; i.m.; i.v. Dit kan herhaal word na 20–60 min.

(b) Stimulasie van involusie na geboorte in alle spesies. In die merrie en koei 50 internasionale eenhede (i.e.); in die sog en ooi 20–30 i.e.; in die teef 2–10 i.e. Die resultate is egter wisselvallig en die gebruik van twyfelagtige waarde.

(c) Behandeling van agtergeblewe nageboorte in tewe en sôe. In die teef 2–10 i.e. en in die sog 20–40 i.e.

(d) Stimulasie van melkvloei in sôe met agalaksie teen 'n dosis van 20–30 i.e.

(e) Induksie van geboorte in merries[13] in die laaste 48 h van dragtigheid nadat die merrie begin "was" het. Die dosis is 100 i.e. s.c. of i.m. en geboorte vind plaas binne 15–45 min.

Ergometrien

Alhoewel hierdie middel nie 'n hipofiseale hormoon is nie word dit hier behandel omdat dit naverwant is aan oksitosien in dié sin dat dit ook gebruik kan word vir die stimulasie van baarmoeder sametrekking. (Ergometrine, Wellcome (Edms) Bpk. en Ergotrate Maleate, Lilly Laboratories (S A) (Edms) Bpk.) Dit word gebruik in gevalle van plasentale subinvolusie en metritis/piometra in die teef teen 'n dosis van 0,1–0,5 mg 3 keer per d vir 5 d per os.

Ander gonadotropiene

Menslike chorioniese gonadotropien (HCG)

Hierdie middel is die mees algemene vorm van LH beskikbaar in die handel. Dit word bemark as Chorionic Gonadotrophin V. (Armour) en as Chorulon (Intervet). Dit word algemeen gebruik vir die induksie van ovulasie tydens bronstigheid in die merrie (2000–3000 i.e.); koei (1500 i.e.); sog en ooi (750 i.e.) en teef (150–250 i.e.). Dit kan i.m. of i.v. toegedien word. Hierbenewens kan dit ook gebruik word vir die behandeling van sistiese eierstokke in koeie (5000–10000 i.e.) i.m. of i.v. en vir die stimulasie van deklus in jong bulle (3000 i.e.) (M Vandeplassche 1978 Departement Voortplanting-Obstetrie, Fakulteit van Diergeneeskunde, Gent, België, persoonlike mededeling).

Dragtige merrieserum gonadotropien (DMSG/PMSG)

Hierdie middel het hoofsaaklik 'n FSH – funksie en is 'n algemene bron van FSH-aktiwiteit vir die klinikus. Dit is beskikbaar as PMS (Upjohn (Edms) Bpk.), Seragon (Ferring AB) en Folligon (Intervet). Die middel word algemeen gebruik vir stimulasie van FSH-afhanklike prosesse soos follikulêre ontwikkeling en spermatositogenese. Dit word dan ook algemeen gebruik vir superovulasie in samehang met embrio oorplasing [4 12]. Die dosis varieer tussen 3000 en 6000 i.e. vir die merrie; 1000 en 3000 i.e. vir die koei; 300 en 1000 i.e. vir die ooi; 500 en 1000 i.e. vir die sog en 50–200 i.e. vir die teef. In die hings en bul varieer die dosis tussen 1500 en 3000 i.e.; in die ram tussen 500 en 1000 i.e. en in die reunhond tussen 400 en 1000 i.e. Die roete van toediening is gewoonlik subkutaan of i.m.

Steroiedhormone

Oestrogene

Oestrogene is beskikbaar as Stilboestrol dipropionate (Maybaker S A (Edms) Bpk) en as Oestradiol (ECP, Upjohn (Edms) Bpk). Dit kan gebruik word vir 'n verskeidenheid van gevalle waarvan die belangrikste soos volg is:

(a) Behandeling van agtergeblewe nageboorte en/of metritis in die koei waar die serviks gedeeltelik oopgehou moet word ter wille van intra-uterine behandelings. Die aanbevole dosis is 20–30 mg Stilboestrol i.m. elke 48 h vir 6–8 d. Alternatiewelik kan ECP gebruik word teen 'n dosis van 2–3 mg elke 48 h vir 6–8 d. Die oestrogene bevorder terselfdertyd die bloedvoorsiening na die baarmoeder, die spontane motiliteit van die baarmoeder en migrasie van leukosiete na die baarmoeder.

(b) Verslapping en dilatasie van die serviks in koeie vir die verwydering van 'n gemummifiseerde foetus teen dosisse van 100–300 mg Stilboestrol of 10–30 mg ECP elke 48 h vir 6–8 d.

(c) Behandeling van plasentale subinvolusie en/of metritis in tewe tesame met ergometrien teen 0,5–3,0 mg elke 48 h vir 6–8 d.

(d) Behandeling van verkeerde dekking in tewe teen 'n dosis van 10 mg/kg stilboestrol i.m. met 'n maksimum van 30–35 mg. Hierdie behandeling is eenmalig en moet binne 72 tot uiters 96 h na dekking gegee word.

(e) Behandeling van ongewensde of oormatige laktasie in tewe teen 0,5–1,0 mg Stilboestrol per dag vir 7 d per os.

(f) Behandeling van inkontinensie in tewe teen 'n dosis van 0,25–1,0 mg Stilboestrol per d. Hier is dit belangrik om 'n onderhoudsdosis vas te stel deur met die hoër dosis te begin en dit geleidelik af te bring tot op die onderhoudsdosis. Dit kan dan ook later moontlik wees om na gelang van die reaksie van die dier, die tussenpose tussen toedienings te verleng. Vir hierdie doel is daar ook 'n preparaat beskikbaar, nl. Colpovis (Warner Pharmaceuticals).

(g) Stimulering van kliniese bronstigheid in die koei (20–100 mg elke 48 h); sog (ongeveer 20–50 mg elke 48 h); ooi (ongeveer 15–50 mg elke 48 h) en teef (ongeveer 5–10 mg elke 48 h), met 'n maksimum van 3 behandelings. In alle gevalle verwys die bogemelde dosis na die gebruik van Stilboestrol, en die roete van toediening is i.m. In die meeste van hierdie gevalle is die geïnduseerde brons anovulatories.

Progesteroon

'n Kortwerkende Progesteroon (Organon-Intervet) is beskikbaar asook 2 langwerkende preparate, nl. Repogest (Burns Biotec) en Depogest (Centaur Labs (Pty) Ltd). Eersgenoemde kan gebruik word vir die voorkoming van gewoontelike aborsies teen 'n dosis van 50 mg per dag of 100 mg elke 48 h in koeie en merries en 10–20 mg per d in tewe, i.m. Die langwerkende vorme kan soortgelyk gebruik word teen 'n dosis van 50 mg/50 kg in koeie en merries en 2–4 mg/kg in tewe elke 10–14 dae i.m.

Progesteroon word ook algemeen gebruik vir oes-

trussinkronisasie in beeste. 'n Goeie voorbeeld hiervan is die metode van Grosskopf[9 10] wat bestaan uit 2 kursusse van 4 en 6 48-uurlikse inspuitings van 50 mg progesteroon met 'n 8 d rusperiode tussen die 2 kursusse.

Progestogene

Hierdie is 'n groep sintetiese preparate wat gebruik word vir hulle progesteroon-agtige werking. Die mees algemeen gebruikte middels in hierdie kategorie is soos volg:

(a) Megestrol asetaat(MA)
Hieronder is daar veral twee middels wat dikwels gebruik word, nl. Ovarid (Milvet Ethicals (Edms) Bpk.) en Niagestin vet (Novo Industries Pharmaceuticals (Pty) Ltd). Beide word gebruik vir die voorkoming van oestrus asook die onderdrukking van oestrustekens in tewe en katte. Beide moet versigtig gebruik word in jong tewe en verkieslik glad nie tydens die eerste bronsperiode nie. Ovarid word gebruik in tewe teen 2 mg/kg vir 8 d per os vir die onderdrukking van oestrustekens vanaf die eerste dag van bloeding. Vir oestrus voorkoming word dit gebruik teen 0,5 mg/kg vir 30 d per os. In katte is die ooreenstemmende dosis 5 mg/d vir drie dae in 2,5 mg/d per os.

Niagestin vet kan gebruik word vir voorkoming teen 10–20 mg/d per os vir 3 d gevolg deur 5–10 mg/d per os vir 7 dae in tewe en teen 2,5–5 mg/d per os vir 2–3 weke in katte.

Beide middels kan gebruik word vir die vinniger reduksie van vaginale prolaps in tewe teen 'n dosis van 2 mg/kg per os vir ongeveer 4 d.

(b) Melengesterol asetaat (MGA)
Slegs Anestrol (Upjohn (Edms) Bpk.) val in hierdie groep. Die middel word gebruik vir oestrusstimulasie op grond van 'n aanvanklike onderdrukking van hipofiseale aktiwiteit in koeie teen 'n dosis van een mg/d vir 14 d per os.

(c) Medroksieprogesteroon asetaat (MPA)
1. Depo-provera en Promone (Upjohn (Edms) Bpk.).
Die middel kan gebruik word vir die voorkoming van oestrus in honde en katte teen dosisse van 50–250 mg en 50–150 mg i.m. respektiewelik[2]. Hierbenewens kan dit ook gebruik word vir skyndragtigheid teen 'n dosis van 250 mg. In perde word dit aanbeveel ter voorkoming van gewoontelike aborsie teen 'n dosis van 250–500 mg i.m. en ter voorkoming van oestrus teen 'n dosis van 125–250 mg i.m[2].
2. Perlutex (Leo Laboratories)
Word gebruik ter voorkoming van oestrus in die teef teen 50 mg i.m. asook vir die redusering van vaginale prolaps teen 25–100 mg per os vir 4 d.
3. Repromap (Upjohn (Edms) Bpk.)
Geïmpregneerde vaginale sponse wat in skape gebruik word vir oestrussinkronisasie met of sonder bykomstige DMSG.

(d) Chlormadinone (CAP)
Kan gebruik word vir verkorting van lang oestrus periodes in merries in die vroë seisoen teen 100 mg/d per os vir 8–10 d. (M. Vandeplassche, Departement Voortplanting-Obstetrie, Fakulteit van Diergeneeskunde, Gent, België, persoonlike mededeling).

(e) Delmadinone asetaat
Hierdie middel word bemark as Tardak (Syntex Pharmaceuticals (Pty) Ltd.) Dit kan gebruik word vir die volgende gevalle[8]:
1. Onderdukking en/of voorkoming van oestrus in tewe teen 'n dosis van 0,25–2,5 mg/kg per os en 1,0–5,0 mg/kg s.c. en in katte 0,25–1,0 mg/kg per os en 2,5–6,75 mg/kg s.c.
2. Behandeling van skyndragtigheid in tewe teen 'n dosis van 0,5–1,0 mg/kg/dag per os vir 6 dae en 2,5–5,0 mg/kg s.c. 24 uur uitmekaar.
3. Behandeling van ongewenste seksuele gedrag in reunhonde en kat mannetjies teen 0,5–1,5 mg/kg/d vir 7–14 d en 0,25–1,0 mg/kg/d vir 7–14 d per os respektiewelik en 5,0–12,5 mg/kg en 10–20 mg/kg s.c. respektiewelik eenmalig of 2 keer 24 h uitmekaar.

Testosteroon

Verskillende preparate kan verkry word waarvan die bekendste Androject (Penvet Edms (Bpk.), Durates-ton (Intervet) en Depotestosteroon (Upjohn (Edms) Bpk.) is. Eersgenoemde het 'n werkingstyd van 4–5 dae en laasgenoemde 'n veel langer tyd.

Die middels word gebruik teen ongeveer 25 tot 50 mg i.m. in ramme en 100–200 mg i.m. in bulle waar spesifieke indikasies bestaan. Dit is egter raadsaam om uiters versigtig te wees wanneer hierdie middels in teeldiere gebruik word aangesien langdurige gebruik 'n negatiewe effek het op die afskeiding van FSH en LH en dus ook op spermatogenese. Dit kan ook gebruik word vir die behandeling van ongewenste laktasie in tewe teen 10–25 mg i.m.

Miboleroon

Hierdie stof is 'n nie-progestatiewe steroid wat gebruik word vir voorkoming van oestrus in tewe. Dit is beskikbaar as Matenon (Upjohn (Edms) Bpk.) en word gegee teen 'n dosis van 0,3–1,8 mℓ (=30–180μg) per os per dag vir solank as wat oestrus uitgestel moet word.

Kombinasies

'n Kombinasie van oestrogeen en testosteroon is beskikbaar as Silberone (Centaur Labs (Pty) Ltd.) vir die behandeling van inkontinensie en ongewensde laktasie in tewe teen 'n dosis van 1 mℓ/12 kg i.m. (25 mg Testosteroon en 1,25 mg Stilboestrol).

Ander kombinasies wat in die onlangse literatuur voorkom is die gebruik van oestradiol teen 0,1mg/kg/d s.c. vir 7 d gekombineer met 0,25 mg progesteroon/kg/d s.c. vir 7 d beide in verdeelde dosisse elke 12 h vir die kunsmatige induksie van laktasie[5 11].

Kortikosteroïede

Deksametasoon preparate (Dexafort, Intervet; Opticortenol-S, Ciba-Ceigy (Edms) Bpk.) kan gebruik word vir induksie van geboorte in koeie[17] teen 30–40 mg i.m. in die laaste 14 dae van dragtigheid en in ooie teen 'n dosis van 10–20 mg i.m. in die laaste 10–14 d van dragtigheid. Geboorte volg gewoonlik in 48–72 h.

Ander kombinasies

Onder hierdie kategorie moet slegs die kombinasie van

HCG en progesteroon genoem word. Hierdie kombinasie (3000 i.e. HCG en 125 mg progesteroon per 5 mℓ) is beskikbaar as Nymfalon (Intervet) en Skaurolon (Byk-Gulden). Dit word aanbeveel vir die behandeling van sistiese eierstokke teen 'n dosis van vyf mℓ i.v. Die middel kan na 7 d weer toegedien word.

Prostaglandiene

Prostaglandiene wat teenswoordig beskikbaar is is Cloprostenol (Estrumate, ICI S A (Pharmaceuticals) Ltd.), Fluprostenol (Equimate, ICI Pharmaceuticals) Ltd.) en Dinoprost tromethamine (Lutalyse, Upjohn (Edms) Bpk.).

Die gebruike is menigvuldig maar behels basies alle toestande waar 'n corpus luteum (CL) teenwoordig en ongewens is. Voorbeelde hiervan is die volgende: anoestrus in die teenwoordigheid van 'n CL, piometra in die teenwoordigheid van 'n CL, mummifikasie, luteale siste, ongewenste dragtigheid, ens. Praktiese toepassing van die beginsel van luteolise veroorsaak deur die prostaglandiene kan ook gebruik word by oestrussinkronisasie en induksie van geboorte.

1. Estrumate kan gebruik word in koeie (500 μg i.m) en ooi (125 μg i.m.). Sensitiewe CL – fase is tussen die sesde en sestiende dag vanaf ovulasie in die koei en tussen die vyfde en twaalfde dag vanaf ovulasie in die ooi.
2. Equimate word gebruik in merries (250 μg i.m)
3. Lutalyse kan gebruik word in koeie en merries (25 *mg* i.*m.*)

MISBRUIKE

Hipotalamiese hormone

Misbruike is gewoonlik as gevolg van die gebruik tydens 'n verkeerde stadium van die geslagsiklus bv. wanneer 'n CL teenwoordig is in die koei en merrie en Receptal gebruik word om anoestrus te behandel terwyl die prostaglandine (Equimate; Lutalyse) eerder gebruik moes gewees het of wanneer Receptal gebruik word vir induksie van ovulasie en dit te vroeg of te laat toegedien word.

Misbruik in hierdie gevalle is nie nadelig vir die dier nie maar 'n ekonomiese verlies vir die boer.

Hipofiseale hormone

Misbruike van FSH en LH is gewoonlik in die vorm van verkeerde dosisse wat of te laag of te hoog is. Te lae dosisse veroorsaak nie die verlangde fisiologiese reaksie nie en te hoë dosisse is 'n vermorsing van geld en veroorsaak gewoonlik ongewenste reaksie, bv. sistiese eierstokke.

Hierbenewens word die middels dikwels lukraak en empiries gebruik sonder dat die juiste gewenste reaksie vooraf bepaal is met onvoorspelbare gevolge.

Misbruike van oksitosien kom hoofsaaklik in honde voor waar oksitosien gebruik word om baarmoeder kontraksies te stimuleer in distokie gevalle.

(a) Dikwels word 'n te hoë dosis gebruik en dit kan ringkontraksies veroorsaak proksimaal en distaal van die mees distale hondjie met die gevolg dat die geboorteproses nie kan vorder nie.
(b) Te hoë dosisse kan ook aanleiding gee tot baarmoeder ruptuur[14],peritonitis, skok en dood.
(c) Te hoë dosisse asook normale dosisse wanneer die cervix nog toe is of wanneer daar obstruksie in die geboortekanaal is kan ook aanleiding gee tot ruptuur[14], peritonitis, skok en dood.
(d) Toediening wanneer die cervix nog toe is kan ook loslating van die plasenta veroorsaak met afsterwe van die foetusse.

Ander Gonadotropiene

Misbruik van byvoorbeeld DMS volg gewoonlik op herhaalde gebruik en/of te hoë dosisse. Algemeenste probleme is sistiese eierstok degenerasie, algemene onvrugbaarheid, ens.

Steroiede

Oestrogene

Misbruike ontstaan gewoonlik as gevolg van foutiewe hoë dosisse wat toegedien word of as gevolg van effens verhoogde dosisse wat oor lang tydperke toegedien word. In laasgenoemde geval lei dit tot hipertrofie van die klierweefsel met uiteindelike piometra[15]. Verwarring tussen Stilboestrol en ECP (oestradiol) is 'n algemene probleem. ECP word oor die algemeen aanvaar om 10 x meer potent te wees as stilboestrol[3]. Honde word ook dikwels deur medici en eienaars behandel vir veral verkeerde dekking met somtyds katastrofiese gevolge.

Simptome van oestrogeen *vergiftiging* sluit leukositose en 'n linkse verskuiwing in aan die begin wat later oorgaan in beenmurg hipoplasie, trombositopenie, granulositopenie en aplastiese nie-regeneratiewe anemie[3]. Hierby volg dan verskeie vorme van bloedingsneigings en sekondêre infeksie, veral pneumonie[3,16].

'n Verdere misbruik van oestrogene wat redelik algemeen voorkom is die gebruik daarvan om oestrus te stimuleer in gevalle van anoestrus. Alhoewel dit in sommige gevalle die verlangde uitwerking het, is dit 'n steriele oestrus sonder folikulêre ontwikkeling.

Progesteroon en Progestogene

Misbruike kom voor as gevolg van die gebruik van hierdie middels in gevalle waar aborsie voorkom moet word en die foetus dan reeds afgesterf het of waar parturisie uitgestel moet word en die cervix dan reeds gedeeltelik ontsluit is met die gevolg dat die foetus dan afsterf. In beide gevalle lei dit dan tot ontbinding van die foetus *in utero,* erge metritis, ens. In soortgelyke gevalle waar die cervix wel nog toe is, dien die progesteroon wel om parturisie uit te stel maar wanneer parturisie dan plaasvind is die foetus veel groter en distokie is 'n algemene komplikasie. Die foetus is dikwels dan ook dood.

'n Verdere probleem in hierdie kategorie is die onverskillige gebruik in tewe wat aanleiding kan gee tot metritis en/of piometra. Dit is veral so tot 'n groter mate in jong tewe, in sekere kortsnoet rasse en met grotere dosisse.

Testosteroon

Hier is die mees algemene probleem die gebruik van testesteroon om libido te stimuleer en waar dit oor lang tydperke plaasvind volg daar 'n negatiewe terugvoer

effek op GnVH en gevolglik op FSH en LH met nadelige gevolge op spermatogenese hoofsaaklik as gevolg van 'n negatiewe effek op die gonadotrofiene en die vrylatingshormoon [6 7].

Kombinasies

Misbruike in hierdie kategorie is gewoonlik as gevolg van die toediening van menslike kontraseptiewe middels in honde om oestrus te voorkom. Hierdie middels het dikwels oestrogeniese en/of progestogeniese aktiwiteit en die simptome van oordosering is dié van oestrogeen toksisiteit en/of progestogeen veroorsaakte purulente endometritis/piometra.

Die gebruik van oestrogeen en progesteroon kombinasies om laktasie te induseer in koeie skep ook probleme veral in die vorm van sistiese eierstokke, anoestrus en lae konsepsiesyfers[5].

Kortikosteroïde

Misbruike in hierdie kategorie is gewoonlik as gevolg van onkundige en onverskillige gebruik van hoë dosisse gedurende dragtigheid en veral laat dragtigheid in beeste en skape. Die algemeenste komplikasie is dan aborsie.

Prostaglandiene

Misbruike van prostaglandiene is gewoonlik te wyte aan onkundige en onverskillige gebruik in dragtige diere waar dit lei tot aborsie. In diere wat nie 'n sensitiewe CL het nie is dit nie nadelig vir die dier nie, maar verteenwoordig dit 'n ekonomiese verlies vir die boer.

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A REVIEW OF THE USAGE OF PROSTAGLANDINS IN PIGS

D. G. CATTON

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Prostaglandins have a history of greater than 20 years. Their actions and indications are extremely diversified and more is being constantly learnt about this group of compounds as research progresses. Current veterinary uses have been restricted to horses and cattle. This review summarises the new indication for these compounds in pigs and discusses suggested usage programmes.

INTRODUCTION

Prostaglandin research has a history of more than 20 years. It was in the early 1950's that researchers first descovered the potential significance of this important new family of drugs and began intensive research efforts on them. Prostaglandins are potent substances found in nearly all animal and human tissue and are believed to play a key role in cellular metobolism and regulation of many body functions. They have attracted world-wide interest during recent years because of their wide-spread effects on a variety of body processes in both humans and animals. Some researchers have even described prostaglandins as the new 'wonder drugs' of the 1970's and 1980's. Fourteen natural compounds have so far been identified as prostaglandins, the letters A B C D E F being used to identify the 6 main types[5]. At the recent 4th International Prostaglandin Conference numerous new uses were reported for the prostaglandins. These include the possible future use of prostaglandins for control of hypertension, improving blood flow and healing of skin ulcers in circulatory disease, preventing the body from losing its ability to produce a natural defence against viral infection, prevention of clogging of blood circulation machines, protection of the stomach against the side effects caused by aspirin, use in the relief of bowel complications following abdominal surgery and improved survival-odds of new borns awaiting surgery for congenital heart defect[7].

To date, the commercial usage of prostaglandins in veterinary medicine has been largely restricted to cattle and horses where it is utilised for planned breeding and reproductive therapy purposes. In man the prostaglandins are basically used for the termination of pregnancy. A new usage of these products has now been approved by both companies marketing prostaglandins in South Africa, which is restricted to a claim for the induction of parturition in pigs.

BIOLOGICAL EFFECTS

Prostaglandin $F_2\alpha$ ($PGF_2\alpha$)and its analoque have been shown to be capable of inducing parturition. Although the exact mechanism of action relative to parturition induction has not been clearly elucidated, exogenously administered $PGF_2\alpha$ has been reported to possess both luteolytic and ocytocic properties in pigs[4].

Ocytocic effect

A study was conducted to evaluate the effect of $PGF_2\alpha$ on porcine myometrium in vitro. $PGF_2\alpha$ stimulated motility (an increase in fequency and strength of contraction) of uterine strips from both pregnant and non-pregnant gilts. Myometrium from pigs in early pregnancy (30–60 d) was more sensitive to low doses of $PGF_2\alpha$ than myometrium from non-pregnant gilts. The data suggested that $PGF_2\alpha$ may be capable of producing a direct effect on the porcine uterus in vivo.

Luteolytic effect

$PGF_2\alpha$ was reported to be luteolytic in bilaterally hysterectomized gilts on Day 17 after onset of oestrus. $PGF_2\alpha$ was reported to be luteolytic in bilaterally hysterectomized gilts on Day 17 after onset of oestrus. $PGF_2\alpha$ treatment resulted in corpora lutea (CL) regression, a precipitous decline in plasma progestin concentration, and onset of behavioural oestrus within 68–132 h after the end of treatment. Swine CL were reported to be refractory to luteolytic effects of $PGF_2\alpha$ for approximately 12 d after onset of oestrus. However, $PGF_2\alpha$ has been reported to be luteolytic when administered after Day 12 of the oestrus cycle in the pig. It is hypothesized that swine CL do not become susceptible to luteolytic agents until about the time they normally regress due to endogenous luteolytic factor(s). This 'refractory' period for porcine CL provides an explanation for reports that $PGF_2\alpha$ has no value in oestrus synchronization programmes for pigs. In pigs, functional corpora lutea are the principal source of progesterone and they are required for the maintenance of pregnancy throughout the entire gestation period. The administration of $PGF_2\alpha$ during early gestation on Days 25–30 resulted in a significant decrease in plasma progesterone levels and abortion suggesting that $PGF_2\alpha$ was luteolytic when given during early pregnancy. Pregnant pigs treated during late gestation on Days 105–113 also exhibit a significant reduction in plasma progesterone when compared to controls. This reduction in plasma progesterone in pregnant animals responding to $PGF_2\alpha$ treatment occurred from 8–24 h following drug administration. This corresponded with a significant reduction in body mass of the CL of pregnancy in treated groups when compared to non-treated controls. This suggested that luteal regression was the primary mechanism of action of exogenously administered $PGF_2\alpha$ which resulted in a decrease in serum progesterone. In addition to a significant reduction of serum progesterone, $PGF_2\alpha$ medicated pregnant animals exhibited a significant reduction in time interval from treatment to parturition when compared to pregnant non-medicated controls.

Thus, interpretation of the existing data leads to the conclusion that $PGF_2\alpha$ is luteolytic after Day 12 of the oestrus cycle in non-pregnant pigs and in both early and late gestation in pregnant pigs.

0038-2809/80/03/0185–187

SOUTH AFRICAN POSITION

In South Africa there are 3 commercially available prostaglandins. These are cloprostenol*, dinoprost tromethamine† and fluprosteno‡. The prostaglandins specifically indicated for use in pigs are cloprostenol and dinoprost.

Cloprostenol

The specific formulation of this prostaglandin for use in pigs is not yet available in South Africa. However, it is available in other countries under the trade name Planate. The dosage is 175μm/pig intramuscularly (Personal communication – Markus A.N., ICI Pharmaceuticals Limited 1979).

Dinoprost tromethamine

Available in a bottle of 10mℓ. Dosage for sows and gilts is irrespective of body mass – 10mg (2mℓ) intramuscularly.

Indication

For both products this is – 'To induce parturition in pigs'. Treatment should not be instituted before Day 111 of gestation.

Response

The average time for response to injection for cloprostenol is 24 h and for dinoprost 33 h. The variation can be from 17-48 h. Treatment response is ±85% and is not associated with any parturition difficulties, milk let down or lactation disturbances, litter size, viability or adverse effects on the growth of piglets – as long as it is used at least on Day 111 or more of pregnancy. There is also no adverse effect on future reproductive capability.

A decrease in birth mass of piglets has been reported[1 2 6 8] but by weaning time no significant differences from untreated controls can be observed. In addition, there have been side effects associated with an injection of dinoprost tromethamine which occur in 80–90% of all animals treated. These can be disturbing the first time they are seen, but once the pig farmer knows them, they no longer are any cause for concern. These side effects are actually the normal pre-parturition nesting actions and/or reactions of a sow, but are concentrated into a short period They usually start within 6 min after injection and disappear within 1 hour. They include increased body temperature, increased respiratory rate, increased salivation, stimulation of urination and defecation, flushing of the skin, and restlessness (arching of the back, pawing, rubbing and gnawing of the crate).

Management Tool

The prostaglandins when used for parturition induction are Management Tools, in that they offer the pig farmer the opportunity to plan his sow/gilt farrowing times according to *his* time table and not according to nature. However, these products can only be used by the efficient pig farmer who has accurate service records. *This point cannot be over emphasized.* When used effectively the prostaglandins are highly efficient management tools, but if incorrectly utilised, they can produce disastrous results.

*Estrumate – ICI SA (Pharmaceuticals) LTD containing cloprostenol 500μg/mℓ

†Equimate – ICI SA (Pharmaceuticals) Ltd containing fluprostenol 250μg/mℓ

‡Lutalyse – Upjohn (Pty) Ltd containing dinoprost tromethamine 5mg/mℓ

ADVANTAGES AND DISADVANTAGES OF USING PROSTAGLANDINS IN PIGS

Disadvantages

Incorrect usage of products

Should the prostaglandins be injected earlier than 111 days of pregnancy, they will either result in abortion or the birth of a litter in which there is a high percentage of low birth mass piglets associated with a high mortality of these piglets.

Cost of Drug

This is a convenience product thus difficult to equate with whether it should be considered expensive or inexpensive. It largely depends on the value placed upon an indivual's time.

Advantages

Convenience

The farmer is now able to organise the farrowing of his sows to suit his particular farm management plan, thus resulting in better farm planning. This means

(a) that he can eliminate or reduce night and weekend farrowing
(b) the farrowings are planned according to availability of labour
(c) the farrowings are planned for ideal supervision allowing improved 'fostering' or 'evening of litters' and improved efficiency of management activities at farrowing.

The above factors lead to a *lower piglet mortality*. A reasonable value of a new born piglet, according to the Veterinarians of the Swine Health Scheme of the Meat Board is R10,00[3]. With an increased number of piglets born alive, this results in higher productivity per sow.

Batch farrowing of Sows/Gilts allows for improvement in the planning and implementation of management practices associated with farrowing, e.g. cleaning of pens, cleaning sows/gilts, feed changes, etc. There is also the possibility to initiate an 'all in – all out' system of usage of the facilities. This is followed by batch rebreeding with all sows/gilts in the same post-parturient stage.

Improvement of farrowing index resulting in reduced feed costs. If we consider the conceiving, pregnancy and lactation segments of a sows cycle (Fig. 1), and we wish to improve this time period, then we have the following options. We cannot reduce the conceiving segment of the cycle; we have already made significant reductions in the lactation period and the only other opportunity is to reduce the pregnancy segment. If this can be reduced by 3 days per pregnancy, this is more or less equivalent to 1 week per year. At the current cost of feed – sow and boar ration – of R0,12 per kg and considering that ±6kg of feed is consumed during the

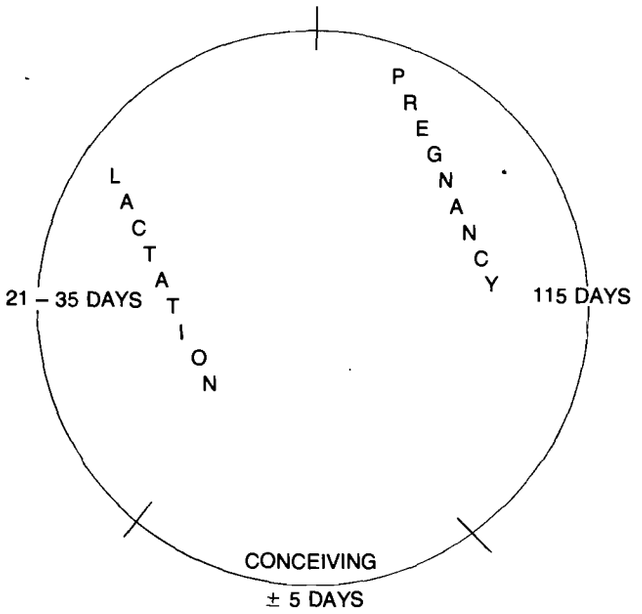

Fig. 1 Segments of sows pregnancy cycle

last 3 days of pregnancy, the saving is equal to R1,44 per sow per annum (2 farrowings).

PROGRAMMES

Numerous programmes are available or can be devised but the ones summarised in Table 1 can serve as examples of how these products could be used in a farm management programme.

ACKNOWLEDGEMENT

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Table 1: PROSTAGLANDIN PROGRAMMES IN PIGS

Programme	No 1	No 2	No 3
Prostaglandin treatment	1 x per week	2 x per week	3 x per week
Method	Allow sows/gilts to farrow normally on Monday, Tuesday, Wednesday and Thursday Sows/Gilts due to farrow Friday, Saturday and Sunday – inject Thursday morning – farrowing should be complete by Friday afternoon.	Sows/Gilts due to farrow: 1. Wednesday and Thursday – inject Tuesday morning – farrowing completed Wednesday afternoon 2. Friday, Saturday and Sunday – inject on Thursday morning – farrowing completed Friday afternoon.	Sows/Gilts due to farrow: 1. Wednesday and Thursday – inject on Tuesday morning – farrowing completed Wednesday afternoon 2. Friday, Saturday and Sunday – inject on Thursday morning – farrowing completed Friday afternoon. 3. Monday and Tuesday – inject Sunday morning – farrowing completed Monday afternoon.
Benefit	Farrowing free weekend	Semi-planned farrowing cycle	Weekly, fully planned farrowing cycle
Farrowing days	Monday, Tuesday, Wednesday, Thursday, Friday	Monday, Tuesday, Wednesday, Friday	Monday, Wednesday, Friday

SOME THOUGHTS ON SWIMMING HORSES IN A POOL

D. H. G. IRWIN and D. W. HOWELL

ABSTRACT: Irwin, D H G; Howell, D W. **Some thoughts on swimming horses in a pool.** *Journal of the South African Veterinary Association* (1980) **51** No. 3 189–191 (En) Box 4107, 1451 Alrode, Rep. of South Africa.

Several indications for swimming horses are recalled. A satisfactory pool and the technique for its use are described. Some observations on the effect of swimming are offered.

INTRODUCTION

There is nothing new about swimming horses and many an old timer will describe how he had horses paddling and swimming in the sea. The mighty Red Rum is reported to have done regular work in the sea, and a host of good trainers in many countries have used the sea and streams to cool and brace tired legs – a technique being replaced in inland areas by placing the legs in tall rubber buckets and bubbling air through them (turbulator boot). In the early days in Johannesburg a man earned his living by rowing his boat, with a horse tied behind, out into the Wemmerpan at half a crown a time. Many a horse which won fame and fortune in Johannesburg in the old days did a lot of swimming; up to 20 min 3 times a week was recommended.

Because horses swimming in a pool is an innovation in South Africa, the experience we have gained in nearly 3 years is recorded. Most of our swimmers were Thoroughbreds in training (nearly 170 individuals) but we have also had a variety of non-racing horses, numbering about 25. The average number of times a horse has swum is about 7. Some horses have swum 3 times a week for months, giving a total of about 1 700 individual swims.

INDICATIONS

Horses suffering from traumatic arthritis of fetlocks and knees are obvious candidates. A less well known indication is the horse which brushes his fetlocks and injures his sesamoid bones. Because there is resistance to the forward motion of the forelimb in water, the extensors are made to work and the recovery phase of the stride is seen to be straighter when the horse is put back to work on the track. Horses with hoof injuries, e.g. picked-up nail, have also been enabled to keep fit till track work is again possible.

Because horses breathe heavily with the exertion of swimming, one is readily able to get a good idea of respiratory stridors. It is far easier to differentiate the stridors because the horse is never more than 12 m from the observer in our situation. Moreover, the horse has his head well extended, so the stridors of over-flexion (which are sometimes difficult to differentiate on the track) are eliminated.

Short swims (say 40–120 m) appear to be exhilarating for most horses, and provide a welcome relief from the tedium of the regular training routine, even if only a dip in lieu of the afternoon walk on a hot summer's afternoon.

Probably the cardiovascular and muscle systems are taxed in proportion to the distance swum, whereas the skeleton is not undergoing *graduated stress* (which is our definition of training). A desirable feature is that joints do not suffer trauma of impact, but possibly the equine athlete's skeleton should have some stress from land work to build bone at the sites of torsion and strain. We agree with this theory and advise long slow work on land as a corollary to the anaerobic and stamina building exercise of swimming. Horses which bave been swum in the winter have shown no ill effects, nor are they reluctant to enter the pool on account of cold water. It seems fair to say that horses fare better in our coldest time than they do in the hottest part of our summer.

THE POOL

The pool is in the form of a pan and handle (Fig.1) and is smaller than similarly shaped pools at the Globe Trotting Track in Adelaide and nearby Lindsay Park Stud. (There is no central island but control of the horses is from the pool surround). The depth of the pool is 2,74 m and the diameter is 11 m. The latter has proved a happy choice because it is small enough around the perimeter (±40 m) to regulate the work in tolerable increments in the early steps of training, but it is large enough to overcome the feeling of swimming "in a circle". A larger diameter pool might prove dangerous if the horse got into difficulties and one had a longer haul to the chute and safety. A depth of 2,60 m –

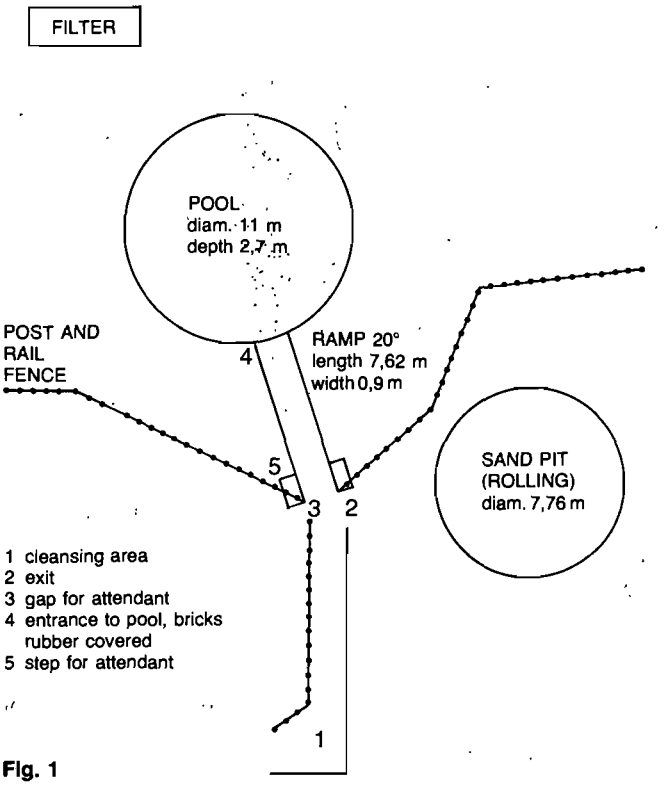

Fig. 1

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2,70 m is adequate and is what we would advocate. The chute width (0,9 m) is good, but the slope (20°) should be slightly less so that one need not have a heavy rubber mat to give a good footing for exit. The pool construction is a 12 mm steel basketwork against earth, then "stacked concrete".

The only alteration planned is to diminish the slope by raising the deep end of the slope about 30 cm. The walls leading to the chute are 1,25 m high and their tops run parallel to the slope of the chute, so that as the horse walks down the slope into the pool, the side wall is the same height to his eye. Obviously the side walls to the entrance to the chute are necessary to assist entry to the pool. If these walls are too high the average horse will suffer a claustrophobic syndrome; if too low, he may be tempted to make a jump to the ground on either side of the chute.

The walls leading to the chute are 25 cm thick and plastered. When one horse tried to turn in the 90 cm area he rocked the walls, so 2 m x 25 x 10 cm timber railway sleepers were buried 60 cm into the ground, and now act as vertical supports to the wall.

The rim of the chute and adjacent part of the pool is now covered with rubber, because horses have scraped a little hair or even skin off a shoulder or lateral body wall if they dived at the wrong angle in launching themselves into the water. The use of bull-nosed bricks on the rim would have been an advantage.

HORSES ENTERING THE POOL

As in loading into floats and starting gates, some horses enter the pool easily and without fear, whilst others object. Clearly, it is advisable to train them to enter the pool and swim in it when they are "flat" and not to undertake the task when they are racing fit and fresh. Our greatest delay was 4 h, but eventually we managed to get the horse concerned to enter the pool. He subsequently became a star swimmer, entering very readily. Tranquillisers (acetyl promezine*, 20 mg administered intravenously and diazepam†, 10 mg intramuscularly) have been very useful and save time. A few horses take a wild plunge in the first few swims. Some individuals have refused to enter, and others are dismissed. When the danger of injury becomes a greater possibility than the possible benefit of swimming, we do not persevere. Much patience and time is spent calming and training horses during the first few swims, and until an individual enters the pool and swims reliably, a veterinary surgeon is present in case of accidents. Some horses which were recalcitrant at first, and were known for their bad behaviour on entering and standing in the starting gates at the racetrack, became model swimmers, and when raced again they were easily put into the starting gates.

THE SWIMMING

Although it may be true that in the natural state all horses can swim, several horses exposed to this highly artificial environment have entered the pool and quickly set out to try and drown themselves. The greatest danger, probably a fear reaction, is when a horse assumes the head up-tail down position. Unless firm forward traction is applied, he will do a backward somersault. This is greatly distressing and the horse is immediately pulled to the chute. A second, though less hazardous abberation, is when an animal rotates on its long axis and tries to swim on its side. These too can usually be helped by steady traction on the head leads. In fact, strong forward traction on the head collar is the best insurance against trouble once the horse is waterborne. Loud vocal encouragement is a great help; the horse apparantly is given the impression that he is not alone in a watery world.

PROCEDURE FOR SWIMMING

The horse is stood in the hosing bay and the feet and other areas brushed and hosed to avoid contamination of the water. One hopes that the horse will defaecate at this time. We are considering the use of an enema in those individuals who make a habit of defaecating in the pool. A stout cavesson head collar is applied, to the upper ring on which is attached a 2,2 m pole, which in turn has a 5 m webbing attached at its distal end. A second halter is applied over the cavesson and it is fitted with a 12 m long webbing. The pole-bearing attendant leads the horse up the lead-in to the chute, and a second attendant slips out between the lead-in and chute wall, and climbs onto the steps to gain height and better control of the horse. When the horse enters the water the second attendant moves quickly to the far side of the pool, playing out the long webbing as he goes. The long-webbing (second attendant) is required to keep the horse moving forward. The pole man leads the horse in a clockwise direction around the pool, using the pole to help prevent the horse getting too close to the pool wall, thus avoiding fetlock bumps and chipping of the wall by a shod hoof. It is essential to have experienced and determined men on the leads.

First-timers are usually given 2 circuits of the pool before being lead up the chute. Regular swimmers are given a gradually increasing work load, so that on a 3 times-a-week schedule, they work up to 10 or 11 circuits per swim after 3 weeks. If a horse is blowing heavily after his swim, he is allowed to stand in the chute with the depth of the water at shoulder height to give him time to compose himself. This avoids a scramble up the chute. Some trainers have developed the necessary finesse to race horses "out of the pool", i.e., without a prerace sprint on the training track. One example would be "Give him 8 circuits at his own speed then chase him up for 2 circuits". Most horses will quicken their swimming pace if called upon in the appropriate manner. Other horses will not do so, however. One horse, a 6 year old entire, appears to swim for enjoyment only. He holds our long distance record with 32 circuits (about 1 280 m). He refuses to be hurried along. Assuredly he is doing aerobic work because when made to do sprint work on the track he is clearly unfit in the respiratory sense. Horses are allowed to roll in the sand pit before being lead or ridden home.

The perimeter of the pool is 40 m, and almost all horses do this distance in 26 s. In those horses which have a good swimming action (the trotting "gait"), the work load is less than those which use a cantering action, or in those whose forelimb movements barely keep the head above the water and rely on the hindlimb trotting "gait" to move forward. Early on in a swim-

*Boots
†Valium, Roche

ming programme, the exercise is anaerobic; it subsequently becomes almost aerobic because the experienced swimmers do not breath heavily. Lazy swimmers are hurried along by appropriate encouragement if anaerobic or "wind work" is requested by the trainer.

POOL MAINTENANCE

The 2 main aspects of maintenance of pools are the filtration of particulate matter and the chemical control of algae. The pool contractors provided 2 cardboard cassette-type filters. This sophisticated type of filter appears to be too refined to cope with the mass contamination of a horse swimming pool. It soon became obvious that they were unsuited to the task. They were replaced by 2 sand filters. Recently an automatic pool sweeper‡ was fitted to one weir from time to time and pool cleaning is less onerous. The bottom of the pool is kept clearly visible using about 3 hours' labour a week.

Calcium chloride is put into the pool 4 or 5 times a week: the pH is controlled by adding hydrochloric acid and copper sulphate is added every 7–10 d.

In conclusion, the horse swimming pool is a great asset to post operative care of orthopedic injuries, and is useful in training horses with joint wear, in differentiating respiratory stridors, in working horses when training tracks are waterlogged in rainy weather and in providing a pleasant variation from the boredom of standard training. It is a pity, however, that it is not economically self sufficient even though there are about 1500 Thoroughbreds in training within a radius of a km.

‡"Creepy Krawley"

RESEARCH COMMUNICATION: CEREBRAL MYCOSIS IN A DOG CAUSED BY *CLADOSPORIUM TRICHOIDES* EMMONS 1952

S. J. NEWSHOLME and MARGARET J. TYRER

ABSTRACT: Newsholme, S. J. Tyrer, Margaret J., 1980. **Cerebral mycosis in a dog caused by** *Cladosporium trichoides* **Emmons** 1952 *Onderstepoort Journal of Veterinary Research,* **47,** 47–49 (1980).

The fungus, *Cladosporium trichoides,* was isolated and cultured from a lesion in the cerebellum and from smaller lesions in the liver, kidney and spleen of a dog which had a history of behavioural changes, ataxia and collapse. Histopathological examination showed the cerebellar lesion to be a purulent granuloma which contained brown, septate hyphae and structures resembling conidia. The source of infection was not traced and no predisposing factors were apparent.

As far as is known, this is the first record of the condition in animals in southern Africa and the first report anywhere of this condition in the dog.

RESEARCH COMMUNICATION: ANTIBODY TO PORCINE PARVOVIRUS IN WARTHOG *(PHACOCHOERUS AETHIOPICUS)*

G. R. THOMSON and I. PEENZE

ABSTRACT: Thomson, G. R. & Peenze, I., 1980. **Antibody to porcine parvovirus in warthog** *(Phacochoerus aethiopicus). Onderstepoort Journal of Veterinary Research,* **47,** 45–46 (1980).

Haemagglutination inhibiting antibody to porcine parvovirus was shown to be widespread in all but one of the warthog populations sampled from South African and Zimbabwe Rhodesia. In some instances titrès as high as ≥ 1/20 000 were detected.

KEEPING OF CASE RECORDS IN EQUINE PRACTICE

D. H. G. IRWIN

ABSTRACT: Irwin, D. H. G. **Keeping of case records in equine practice,** *Journal of the South African Veterinary Association* (1980) **51** No. 3 193–194 (En) P.O. Box 4107, 1451 Alrode, Rep. of South Africa.

A system of being able to follow the clinical history of each horse is obviously valuable from the therapy point of view. The system was devised in June 1974 and has been used in our practice ever since; by the end of February, 1979, over 32,400 case sheets have been written.

DESCRIPTION

A case sheet is shown in Fig. 1, where the patient's name is given as "Neptune". The paper used is autocarboned so that whatever one writes appears on 4 pieces of paper. The top, or original copy is labelled TRAINER, and this is handed to the trainer or, in his absence, to the groom before leaving the yard. It should be retained on the yard for reference; e.g. so that any veterinarian following the writer of that case sheet knows what his colleague has done. The second copy, labelled OWNER is included with the statement of account at month's end.

The third copy labelled CLINIC is filed in date sequence under the horse's name, so that the clinical history of the horse is available at short notice, and this can be consulted by a veterinarian in the clinic before following on a partner who has, e.g. gone on leave.

The fourth copy is left in the pad and filed on a rack for immediate reference by checking the case sheet number on the statement: this is most useful when an owner wants to know what was done to "Neptune" case sheet number 40458 and he has lost his owner's copy which came with the first account.

TRAINER

CASE SHEET: No 40458

HORSE: Neptune

Requested by: D. Turner

For a/c of: R. Smith, P.O. Box 43, Johannesburg

→ You Do — We do →

8 y.o. Ch. gelding.
History: inspiratory & expiratory stridor.
Clinical exam: blood vessels right side of face distended. Jugular vein right side partially occluded. Horse able to set larynx.
Diagnosis: Noise probably due to an oedema of larynx and is likely to disappear when collateral circulation develops.
→ Please keep us informed on his condition.
Faeces collected for examination.
We will report on results →

Signed by.....Vet. Surgeon
20/2/1980

2.0			7,0	
L	X	T	Km	D
C	V	S	I	Total
5,0	6,0			20,00

DRS.....

.....**EQUINE CLINIC**

P.O. Box..... Phone ..... Road .....
..... Newmarket

Fig. 1 Original copy of case sheet. L = Laboratory, X = X-rays, T = Treatment, Km = Travel fees, D = Drugs, C = Consultant, V = Visit, S = Surgery, I = Inoculations

THE ADVANTAGES OF THE SYSTEM

1. A written record of the consultation with the trainer, or, in his absence, with the groom. The finding, diagnosis and work done by the veterinarian are noted. Any instructions to the attendant and any follow up by the veterinarian are recorded. The simple fact of leaving a piece of paper with all the relevant information saves 2 or 3 telephone calls later in the day: "Did the Vet come?" "What did he do?" "What did he say?" "When is he coming again?" "What must I do in the meantime?" These questions largely fall away.
2. The veterinarian is known to have a record of the case, with the opinion he gave at the time of examination. Misunderstandings and irritation of being misquoted do not arise.
3. The owner will be able to follow the course of any injury or disease in his horse and know what he is paying for.
4. The accounting staff have sufficient information on each case sheet to fulfil their duties.
5. The clinic copy is filed under the horse's name so that continuity is maintained even if the horse is sold.
6. When insurance questions or medicolegal problems arise, the system enables one to locate the required information quickly and with a minimum of trouble. Although all information relating to a horse and his illness or injury is confidential to veterinarian, owner and trainer, we do show records to insurance brokers if the horse is, or was, insured at the time for which information is sought. The reason is that this information can be subpoenaed by a court and timeous clarification may obviate litigation, and will in any event not prejudice the issue.
7. If records are shown to an insurance broker or underwriter, another case sheet is written for that horse, recording the fact. The broker/underwriter is debited through the system, and the trainer's copy is sent to the owner so that he is aware of the fact that information has been sought and presented.
8. Sometimes both the owner and the trainer have lost or mislaid a particular case sheet which is material to an insurance claim or for other reasons. Sometimes a case sheet will be written for the service of supplying information and photostatic copies of the original clinic case sheet will be made.

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9. All specimens sent to our laboratory or outside laboratories bear the case sheet number. In this way one can be sure that all work is written up for accounting purposes and the result can be related to the patient without hesitation.
10. The office staff are able to pinpoint any missing case sheet by seeing a blank line in their summarizing book, because they are sequentially numbered 1–100,000.
11. No matter what system is used to record work done, some work is left unrecorded and revenue suffers: the system described appears to reduce these omissions to a minimum.
12. For the statistically minded practice manager, the volume of work done may be readily determined by perusal of the case sheet numbers.

DISADVANTAGES OF THE SYSTEM

1. The office staff spend about 4 h/week filing the clinic copies,
2. Printing costs work out at 8,5c per case sheet number.
3. The serial numbers do not yield a tally of patients seen, because sometimes only one sheet will be for 40 dewormings and drugs and dressings dispensed are also recorded on a case sheet.

On balance this system described suits our practice and is worthy of a trial by colleagues.

AETIOLOGY OF JAAGSIEKTE: EXPERIMENTAL TRANSMISSION TO LAMBS BY MEANS OF CULTURED CELLS AND CELL HOMOGENATES

D. W. VERWOERD, ETHERL-MICHELE DE VILLIERS and R. C. TUSTIN

ABSTRACT: Verwoerd, D. W., DeVilliers, Ethel-Michele & Tustin, R. C., 1980. **Aetiology of jaagsiekte: Experimental transmission to lambs by means of cultured cells and cell homogenates.** *Onderstepoort Journal of Veterinary Research,* **47**,13–18 (1980).

Studies on the transmission of jaagsiekte (ovine pulmonary adenomatosis) both by subinoculation of cells of known sex and by cell homogenates into male and female lambs are reported. The results obtained indicate a thymocyte-dependent rejection of male cells in female recipients in contrast to the successful transplantation of male cells in male animals and female cells in both sexes. This suggests the presence of a surface antigen determined by the Y-chromosome in the tumour cells. A second mechanism of transmission, dependent on the transformation of the recipient's cells, was demonstrated by 2 cases of heterologous transplantation and confirmed by inoculation of cellular homogenates.

UNILATERAL HINDLEG SPASTICITY: OUTBREAK OF A SPECIFIC CLINICAL CONDITION IN SUCKLING PIGLETS

S.J. NEWSHOLME* and L.W. MARSHALL†

ABSTRACT: Newsholme S.J.; Marshall L.W. **Unilateral hindleg spasticity: outbreak of a specific clinical condition in suckling piglets.** *Journal of the South African Veterinary Association* (1980) **51** 195–198 (En) Vet. Research Institute, 0110 Onderstepoort, Rep. of South Africa.

A specific clinical condition of unilateral hindleg spasticity is described which affected all the piglets of 8 litters in one piggery. Histopathological examination of 3 of the piglets revealed changes involving some of the large neurons in the red nucleus, cerebellar nuclei and lumbar spinal cord. These changes were absent from 2 control piglets of the same age which were studied. The clinical and histopathological findings are discussed in relation to the possible aetiology of the condition.

INTRODUCTION

This describes an outbreak of a clinical condition involving unilateral hindleg spacticity which affected all the piglets of 8 litters in a piggery. We consider that the condition merits report because of its specific nature. As far as we are aware, there is no previous report of the condition in South Africa.

HISTORY OF OUTBREAK

The outbreak involved a piggery near Welkom, Orange Free State. The adult stock in this piggery comprised 8 sows and a boar. Six of the sows were of German Landrace stock, registered as pedigree South African Landrace and introduced as young maiden sows from one source in 1978. The boar was also of German Landrace stock, registered and obtained from the same source.

Two maiden, unregistered Landrace sows were also introduced from a separate source in 1978. The boar was used on all 8 sows, which produced their first litters between December 1978 and March 1979.

The piglets of all 8 litters appeared to be normal until 2 weeks after birth. They then became unable to lift their hindquarters and assumed a sitting or lying posture. This prevented them from reaching the mother to suckle milk. Onset was sudden and in each litter all the piglets became affected within 3 days. Every piglet in all 8 litters, a total of 61, was affected similarly. On initial examination they appeared to have posterior paralysis.

Attempts were made to nurse the piglets and milk substitute was fed to them. Multivitamins, vitamin E with selenium and long acting penicillin were administered parenterally. A total of 6 piglets, comprised of 3 from each of 2 litters, showed gradual improvement to normality over several weeks. The remaining 55 showed no change and their bodily condition deteriorated. Eventually they all died or were slaughtered.

None of the sows or the boar showed any departure from health.

During the outbreak one of the affected piglets was slaughtered and the brain, fixed in formalin, was sent to Onderstepoort for histopathological examination. Neuronal changes were present in the red nucleus of this brain (Fig. 3).

Three piglets from another litter, at 5 weeks old, were sent to Onderstepoort where clinical examination and pathological investigation were conducted.

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Fig. 1. Piglet A, showing position of hindquarters and extension of right hindleg.

Fig. 2. Piglet A and, behind it, piglet B, showing the characteristic postures

CLINICAL EXAMINATION

All 3 piglets (A, B and C), were unable to lift their hindquarters from the ground and assumed a sitting or lying posture. The front legs appeared normal and they were able to move short distances, dragging their hindquarters. The hindquarters of each piglet were deflected to one side so that one side was apposed to the ground (Fig. 1). For each piglet, deflection was

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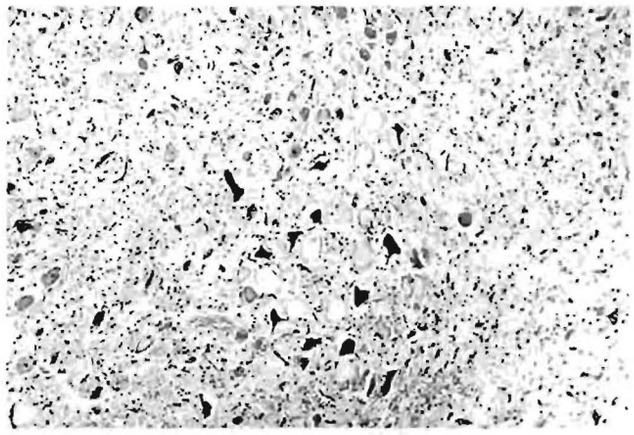

Fig. 3. Transverse secion of part of red nucleus; original brain received. The group of neurons in the centre of the field show complete chromatolysis and nuclear degenerative changes. HE x 70

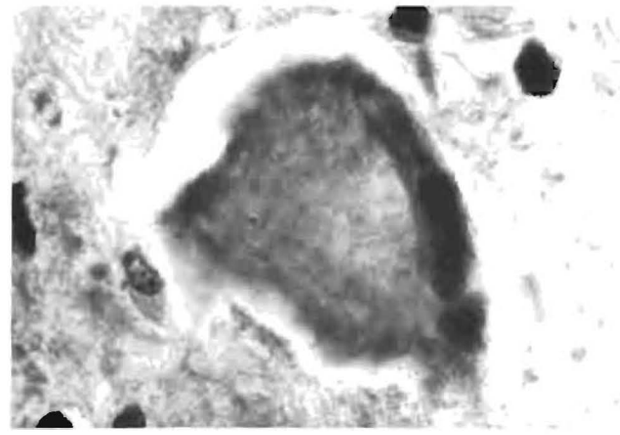

Fig. 5. Neuron showing central chromatolysis and nuclear margination. Lumbar cord, piglet B. HE x 1 000.

always to the same side. In A, deflection was always to the right and, in B (See Fig. 2) and C, always to the left. When each piglet was lifted onto the opposite side it moved a short distance and then reverted promptly to its original side.

Closer examination revealed that in each case the hindleg apposed to the ground was in continuous full extension (Fig. 1) which we interpreted as spasticity. It appeared that the postural anomaly was a direct result of inability to flex this leg. Function of the opposite hindleg appeared normal in each case.

A flexor reflex could be elicited in the extended leg by pinching the interdigital skin with forceps in each case. The leg then returned immediately to the fully extended position.

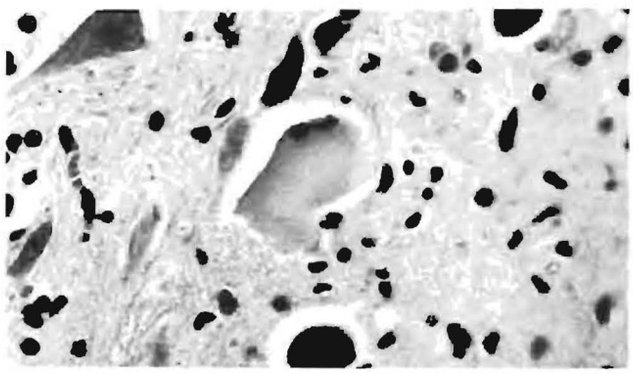

Fig. 4. Neuron in centre of field showing complete chromatolysis with nuclear margination and pyknosis. Lumbar cord, piglet C. HE x 400

PATHOLOGICAL INVESTIGATION

MATERIALS AND METHODS

Each piglet was euthanized by intracardiac injection of 1 g pentabarbitone sodium* and a complete post mortem examination was conducted. Brains, spinal cords and samples from other organs and tissues were fixed by immersion in 10 % buffered formalin.

*Euthatal, May and Baker Ltd.

The brains were cut systematically into 4 mm thick transverse slices with a rotary knife.

Blocks of fixed tissues were embedded in paraffin wax, sections cut at 5–7 μm thickness and stained with haematoxylin and eosin (HE). In addition, sections of brain and spinal cord were stained by the Luxol fast blue method[1] for myelin.

Sections of brain were examined systematically under the light microscope. Where necessary, an atlas of the porcine brain[2] was used for topographical reference.

As controls, 2 healthy, 5 week old, Landrace piglets were obtained and subjected to the same procedures and examination.

Fig. 6. Neuron showing central chromatolysis, vesiculation and absence of nucleus. Lumbar cord, piglet B. HE x 1 000

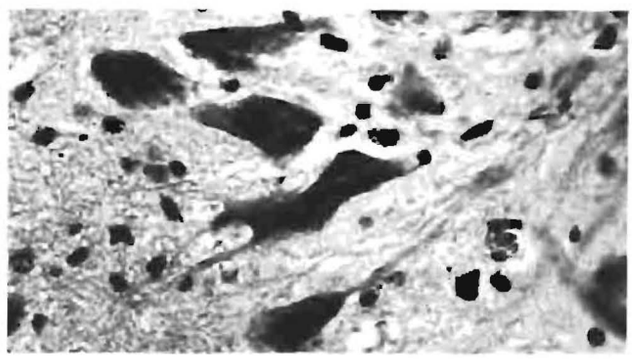

Fig. 7. Normal neurons for comparison. Lumbar cord, control piglet. HE x 400.

RESULTS

No gross pathological changes were seen.

The following histopathological changes were observed:

1. Central Nervous System

Some large neurons showed various degrees of change in all the affected piglets. These changes varied from slight central chromatolysis with nuclear margination (Fig. 5) to complete chromatolysis with nuclear margination and pyknosis (Fig. 4). Complete absence of the nucleus and the presence of cytoplasmic vacuoles were features of a few neurons (Fig. 6). The perineuronal space of affected neurons was usually enlarged but there was no evidence of perineuronal cell reaction.

No neurons showing any of these changes were present in either of the controls.

The affected neurons were distributed singly or in small groups in the red nucleus, roof nuclei of the cerebellum and the lumbar region of the spinal cord in all affected piglets. Distribution appeared to be bilateral in each case.

Examination of the Luxol fast blue stained sections revealed no evidence of demyelination.

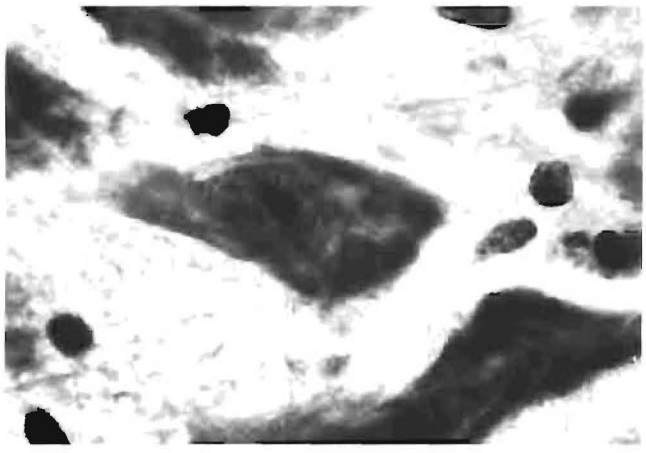

Fig. 8. Normal neuron, lumbar cord, control piglet, HE x 1 000.

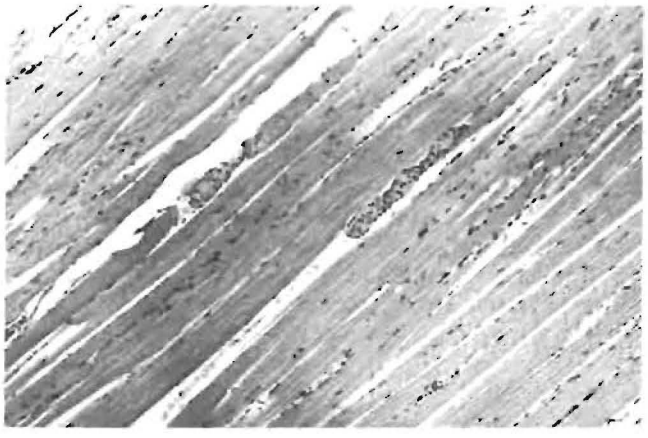

Fig. 9. Quadriceps femoris, right leg of piglet A, showing hyaline necrosis of individual fibres. HE x 100.

2. Quadriceps Femoris Muscle

In A and B, the quadriceps femoris muscle from the extended leg showed hyaline necrosis of single muscle fibres scattered throughout the muscle (Fig. 9). This muscle revealed no abnormalities in C.

No histopathological changes were detected in other tissues.

DISCUSSION

Comparison with control material has enabled us to establish the neuronal changes as patholgical. The more extreme changes, with nuclear degeneration and disappearance, constitute neuronal death.

It is most likely that the clinical condition resulted from the neuronal changes. However, we have not been able to explain the extension of the one hindleg in terms of the anatomical distribution of the dead neurons. The distribution of these neurons was scattered and bilateral. The pattern of hyaline necrosis in the quadriceps femoris muscle suggests that it may have resulted from death of a few neurons in the lumbar cord with depletion of the number of motor units of this muscle. The persistence of a normal flexor reflex, however, suggests that the lesion causing extension of the leg involved upper, rather than lower, motor neurons. A lesion in the red nucleus could produce such an effect. Although the porcine red nucleus has not been studied critically, the evidence from studies in other mammals suggests that the magnocellular component of the red nucleus is an excitatory centre for flexor muscle tone and in inhibitory centre for extensor muscle tone[3]. Thus, a lesion in the red nucleus might be expected to cause increased extensor tone.

The cause of the condition has not been determined. The absence of any inflammatory reaction suggests that it is not infective. The possibility of an underlying genetical influence must be considered. In theory, the condition could have been inherited as a somatic dominant factor from the boar. This could explain why 100 % of the piglets were affected and also account for the involvement of litters from sows of 2 different sources. However, the apparent normality of the boar and the absence of previous reports of the condition make this unlikely. Such a situation could be explained if the gene or genes responsible were of low penetrance and that, for unknown reasons, environmental conditions in this particular piggery allowed manifestation of their expression. Such an explanation remains theoretical. The lineage and interrelationships of these pigs was not determined and no special environmental factors in the piggery were apparent.

A toxic cause remains possible. This being so, the toxic factor must have been present in the milk of all the sows because the piglets received no other form of feed. Furthermore, the sows must have been refractory to such a toxin. We note that mycotoxins from *Aspergillus clavatus* have produced similar neuronal changes in cattle although the clinical picture differed[4]. The feed in this piggery was not investigated for mycotoxins.

The literature contains 2 descriptions of clinical conditions in piglets which are similar to the one described. Although both conditions described were shown to be caused by a monogenic recessive factor, which is clearly not the case here, they merit comparison because of

their clinical similarity. An abstract of a paper describes Large White piglets in Russia[5] which were unable to stand at birth and lay on the right side. If laid on the left side they attempted to turn over. The hindlegs were powerless but the forelegs could be used for crawling. Degenerative atrophy of the motor cells was reported. Litters of Landrace piglets in Norway were described by Berge[6] in which, "The pigs were inclined to lie always on the same side." The pathology was not investigated.

We have described this specific condition in the hope that it will be recognised should it occur again. In such an event, circumstances might allow the cause to be determined.

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CASE REPORT **GEVALVERSLAG**

EXTRAGENITAL MALIGNANT TRANSMISSIBLE VENEREAL TUMOUR IN A BITCH

I. B. J. VAN RENSBURG* AND S. W. T. PETRICK†

ABSTRACT: van Rensburg I. B. J.; Petrick S. W. T. **Extragenital malignant transmissible venereal tumour in a bitch.** *Journal of the South African Veterinary Association* (1980) **51** No 3 199–201 Dept. Pathology, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

Transmissible venereal tumour was diagnosed in the vagina of a bitch. The tumour spread to the oral mucosa in the tonsilar area of a female offspring which was frequently observed licking the vaginal discharge from the bitch. Metastasis to the ovary occurred in the bitch and wide-spread metastasis, especially to the skin, lymph nodes and spleen was recorded in the offspring. Both animals were euthanased due to poor response to treatment. The diagnoses were confirmed by histopathological examination.

INTRODUCTION

Transmissible venereal tumour (TVT) of dogs is a well known neoplasm which has been reported from many countries[7]. Normally it is considered to be a benign neoplasm, many of which undergo spontaneous regression. There are, however, several reports of malignant behaviour where metastasis to various organs in the body had occurred[1,3,4,6,8,9]. The tissues and organs most frequently involved in this respect have been the skin, subcutis, uterus, fallopian tubes, spleen and lymph nodes but cases in which the liver, kidney, brain, adenohypophysis and eye have been affected have also been recorded. This paper reports the spread of a vaginal TVT in a bitch to the oral mucosa of one of her female offspring, and the metastasis of the neoplasm in both subjects.

CASE HISTORY

A TVT was surgically removed from the dorsal vaginal wall of a 3 year old Schnauzer bitch and its identification was confirmed histopathologically. About 10 months later she gave birth to a litter of 5 puppies. The sire was a Schipperke. One bitch puppy was kept by the owner. Growth of the TVT recurred in the vagina and vulva of the bitch about 4 months after whelping. It was again excised surgically and radiation therapy was applied. Two months later she was presented for re-examination and a diagnosis of vaginal TVT and enlarged ovaries was made. The tumour and ovaries were removed surgically and histopathological examination revealed metastasis of the TVT to the ovaries. The lesions were again irradiated. During a period of 3 months no improvement was noticed and, as her general condition had deteriorated, euthanasia was performed. A complete autopsy was not done.

Meanwhile the daughter, which according to the owner used to lick the exudate discharged from the dam's vagina, developed a subcutaneous swelling in the ventral neck at the age of about one year. When she was about 16 months old she was admitted to the Department of Surgery, Faculty of Veterinary Science, suffering from an enlarged left tonsil and left superficial cervical lymph node and a large nodular swelling in the thyroid region. Tonsilectomy was performed and histopathological examination revealed the presence of TVT. Within about 7 days of the operation nodules developed in both conjunctivae and soon thereafter, palpable nodules were detected in the skin all over the body. Two of these were biopsied and histopathologically confirmed as TVT. Euthanasia was performed and an autopsy carried out.

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Macroscopic findings

Multiple dome shaped neoplastic lesions were found in the skin and subcutis – especially over the back and abdomen. These varied in size from 3 – 10 mm in diameter, were firm in consistency and had an even whitish to pinkish-white colour on cut surface. The conjunctivae were similarly affected. The biggest tumour mass was located in connective tissue in the region of the thyroid where it had attained a size of 150 x 50 x 20 mm. Numerous metastatic tumours were found in the spleen, varying in size from 8–20 mm in diameter. Similar metastatic growths were found in the mandibular and medial retropharyngeal lymph nodes, the right ovary and the hypophysis.

The vaginal mucosa was free not only from signs of tumorous growth, but also from scars which might have indicated the possibility of regressed tumours in the locality.

Apart from a single abscess in the left superficial cervical lymph node, no other lesions of significance were observed.

Microscopic findings

Representative specimens from the tumours from all organs and tissues involved were examined histopathologically. Microscopically it proved to be a typical TVT consisting of groups or sheets of round to ovoid or polygonal cells surrounded by delicate connective tissue stroma. The nuclei contained small pieces of chromatin which was evenly despersed or arranged marginally. Each contained a single large nucleolus. This was a distinct fairly homogenous eosinophilic cytoplasm in sections stained with haematoxylin and eosin. The mitotic index was high. The appearance of the tumour was

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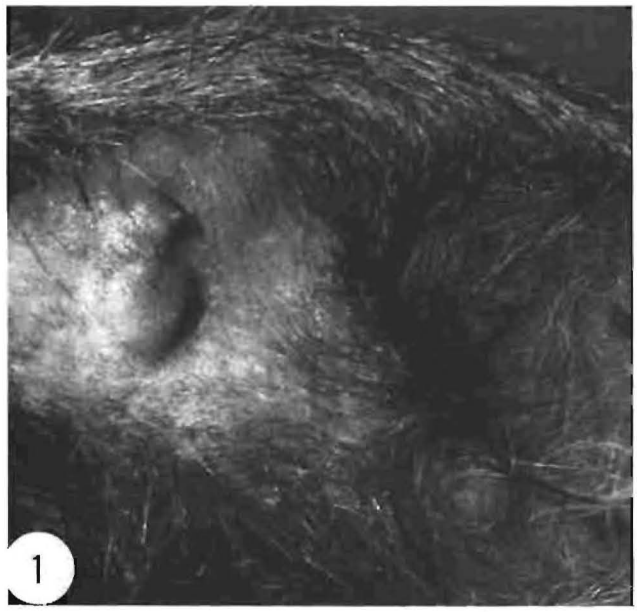

Fig. 1. Dome shaped non-ulcerated cutaneous TVT

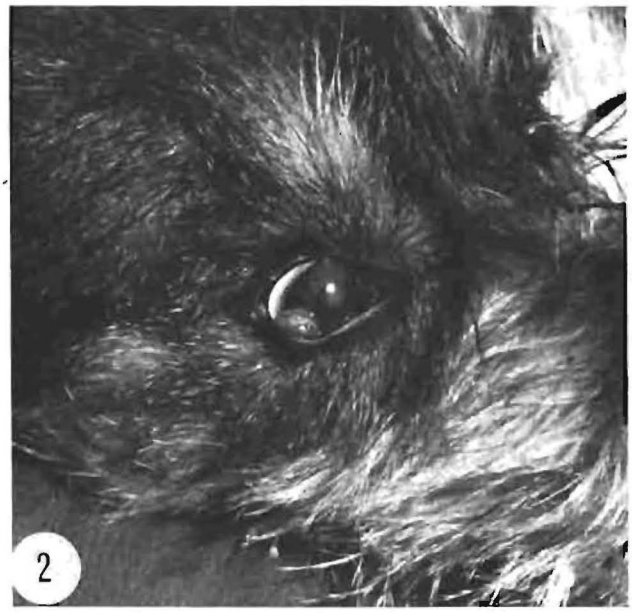

Fig. 2. Conjunctival TVT

Fig. 3. Large tumour mass in thyroid region and subcutaneous tumours (arrows).

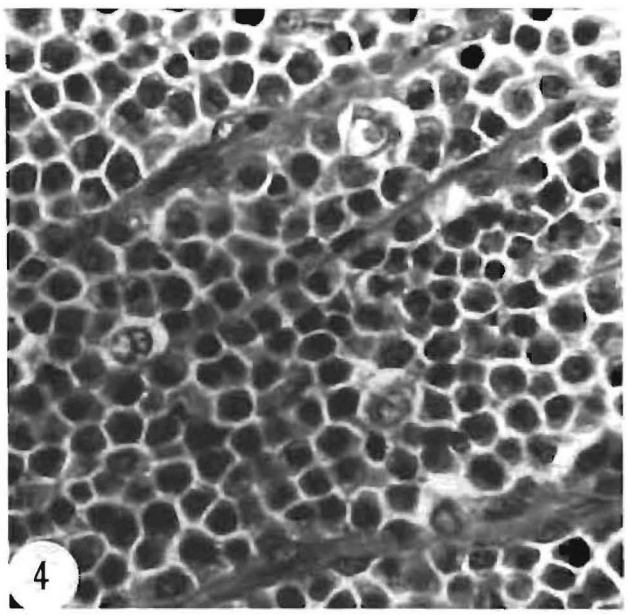

Fig. 4. Sheets of polygonal TVT cells with distinct cytoplasm surrounded by delicate connective tissue stroma. Haematoxylin and eosin stain. x 400

identical in all organs and tissues involved. No evidence of regression or necrosis was noticed in any of the sections examined. The tumour in one of the mandibular lymph nodes, however, did contain a focal area of lymphocytic infiltration while a few neutrophiles were also present. Chromosomal studies of the neoplastic cells were not undertaken.

DISCUSSION

The more interesting features of these cases are the apparent site of the primary tumour of the offspring and the method of transmission which was probably by ingestion of tumour cells. The latter is supported by the history that the offspring frequently licked the vaginal discharge of the older animal which was suffering from a vaginal TVT. The owner is convinced that the young animal never mated.

Also of considerable interest is the apparent increased susceptability of dam and offspring to the TVT cell-line. The dam responded poorly to treatment and in both instances there was metastasis of this normally benign tumour. This compels one to speculate that a familial (hereditary?) immunodefeciency to TVT might have played a role. Another less likely line of speculation is that a more malignant strain of TVT cells was involved in this case.

Malignancy in TVT has been reported on several occasions[1,4,6,7,8,9] while primary extragenital localisation has also been observed in a few instances[2,3,5]. Although no figures were quoted Higgins found metastasis to be a common sequel in the Bahamas – mostly in undernou-

rished dogs which probably lacked an adequate immune response. The most common sites of secondary neoplasms were the uterus, fallopian tubes, superficial inguinal lymph nodes, spleen, cutaneous and subcutaneous tissues, but the eyes and buccal mucosa were also involved in some dogs[4]. Higgins also found primary neoplasia in the skin and "around the eyes" without evidence of a primary genital lesion[4]. Feldman made a similar observation and thought that infection from the mother to the young occurred at birth[3]. It is known that the tumour transplants with relative ease to scarified skin and it is therefore likely that scratching will predispose to skin involvement.

ACKNOWLEDGEMENTS

The authors wish to thank Prof. R. C. Tustin for reading the manuscript and Mrs. V. Käber for the typing thereof. Mrs. H. Rothman is thanked for the photography.

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LETTER TO THE EDITOR — BRIEF AAN DIE REDAKSIE

DOURINE AND THE DOWNER MARE

Dear Sir,

Those of us involved in large animal practice shall at one time or another have been driven to the depths of frustration by that eternally enigmatic case of the Downer Cow, and possibly have developed a pet theory and favourite treatment routine.

I wonder how many of us have encountered the Downer MARE, and been left in even greater bewilderment with equally unsatisfactory responses?

During the last year alone we have seen 6 such patients; 5 were mares of varying ages, and the sixth was a filly which had been rustled as a foal into Lesotho and retrieved a year later. Casual discussions with residents of Lesotho, Transkei and KwaZulu indicate that the syndrome is fairly common in those states and homeland.

We are usually presented with advanced cases where the patient is in sternal recumbency (earlier) or lateral recumbency when in extremus. Appetite is healthy, both defaecation and micturition are unimpaired, and temperature is normal unless secondary pneumonia has set in. Invariably a history of recent oestrus with vaginal discharge of mucopurulent nature, although the discharge is not always evident by the time that professional aid is sought.

Nymphomania was reported in 3 of the 6 mentioned cases, rectal palpation once revealed ovarian cysts; no localised pain nor pelvic or spinal injury was evident and the pelvic vessels were free of pathological conditions. The stockman or owner invariably suspects some form of traumatic injury, and certainly the history indicates this possibility, especially in the cases of lighter animals with nymphomania.

Earlier cases show a swaying hindquarter, occasional knuckling of the hind fetlocks and frequent micturition. Within 2–5 d of these symptoms, posterior paresis has advanced to total recumbency when we are called and expected to perform a miracle. We eventually perform euthanasia.

Treatment is usually palliative until the laboratory results are forwarded; antibiotics, corticosteroids and phenylbutazones are wasted. The first step is to send a serum sample to the Regional laboratory because in our experience these cases are invariably the nervoud form of DOURINE.

This is very well described in the literature, yet fellow rural practitioners appear in need of a reminder of this syndrome. We have not seen any skin lesions, nor have the roaring and facial paralysis been evident in any cases seen by us. Carbon bisulphide and Haloxon are not used as vermifuges in our area and that differential diagnosis is eliminated.

We hope that this letter will help shorten the suffering of both owner and patient while the veterinarian is frantically throwing the book at them.

T. T. Collins
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Underberg 4590.

A SOUTH AFRICAN CATTLE WARBLE?

Sir,

During a visit to a cattle farm in the Middelburg District of the Transvaal we noticed circumscribed, raised nodules in the skins of a number of calves. On pressure each of these nodules yielded a single fly larva, which on microscopic examination proved to be the second stage larva of a fly belonging to the genus *Strobiloestrus*.

The larvae of these flies normally parasitise the skin of klipspringers and reedbuck, in which they cause warble-like lesions, and have also been recovered from a domestic goat! This, however, is the first record of their occurrence in cattle.

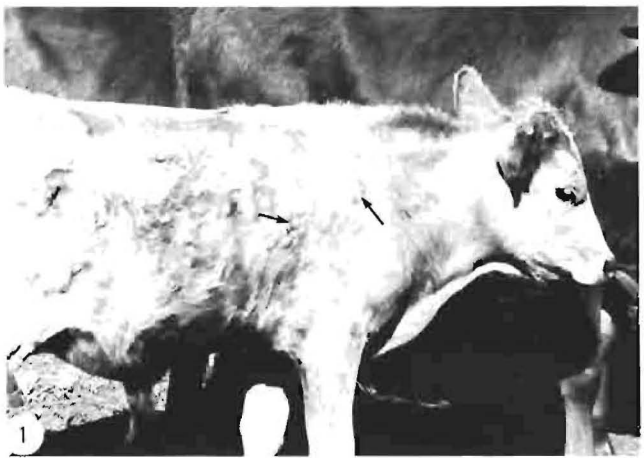

Fig. 1 and 2. Nodules caused by the larvae of *Strobiloestrus* sp. in the skin of a calf.

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THE TEMPERATURE PREFERENCES OF THE MOTILE STAGES OF *STOMOXYS CALCITRANS* LINNAEUS (DIPTERA: MUSCIDAE)

B. SUTHERLAND

ABSTRACT: Sutherland B., 1980. **The temperature preferences of the motile stages of** *Stomoxys calcitrans* **Linnaeus (Deptera:Muscidae).** *Onderstepoort Journal of Veterinary Research,* **47**,7–11 (1980).

When adult *Stomoxys calcitrans* were exposed to a temperature gradient, 66% of them selected temperatures between 20,1 and 32,5 °C. The larval stages preferred temperatures between 19,5 and 33,2 °C. Although the differences in the temperature preferences of the different larval stages were not significant, the fully-fed larvae appear to prefer slightly cooler conditions than the feeding stages. The temperature preferences of both the adults and the larvae are not influenced by the temperatures to which the developmental stages of the experimental flies or their previous generations were exposed.

STUDIES ON *HAEMONCHUS CONTORTUS.* III. TITRATION OF *TRICHOSTRONGYLUS AXEI* AND EXPULSION OF *H. CONTORTUS*

R. K. REINECKE, CHRISTEL M. BRUCKNER and I. L. DE VILLIERS

ABSTRACT: Reinecke, R. K. Bruckner, Christel, M. & De Villiers, I. L., 1980 **Studies on** *Haemonchus contortus,* **III. Titration of** *Trichostrongylus axei* **and expulsion of** *H. contortus. Onderstepoort Journal of Veterinary Research,* **47**,35–44 (1980).

Groups of Merino weaners were dosed with infective larvae of *Trichostrongylus axei* in numbers ranging from 20 000–50 000 and challenged 3 months later with 50 000 infective larvae of *Haemonchus contortus.* When half the dose of infective larvae of T. *axei* was given on Day 0 and the balance on Day +14, efficacy against *H. contortus* was >60% in >60% of sheep (P<0,1). A single dose of 40 000 or 50 000 infective larvae of *T. axei* was >80% effective against *H. contortus* in >80% of sheep (P<0,01).

Two doses of 20 000 infective larvae of *T. axei* followed by a challenge with *H. contortus* 31–33 days after the initial dose caused a reduction of >50% in >50% of sheep (P<0,1). This rose to >60% in >60% of sheep if the doses of 25 000 infective larvae of *T. axei* were followed by a challenge with *H. contortus* 45 days after the initial dose of *T. axei.*

Most of the challenge doses of infective larvae of *H. contortus* were rejected within 3 days. Surviving worms were retarded in the 4th stage and only a few developed to the 5th or adult stage.

STUDIES ON NEONATAL CALF DIARRHOEA CAUSED BY ROTAVIRUS: TRANSMISSION OF THE DISEASE AND ATTEMPTED VACCINATION OF COLOSTRUM-DEPRIVED CALVES

A. THEODORIDIS, L. PROZESKY and H. J. ELS

ABSTRACT: Theodoridis, A., Prozesky, L & Els, H. J., 1980. **Studies on neonatal calf diarrhoea caused by rotavirus: Transmission of the disease and attempted vaccination of colostrum-deprived calves.** *Onderstepoort Journal of Veterinary Research,* **47**, 31–34(1980).

Mild to severe scouring could be produced in colostrum-deprived calves with tissue culture-adapted rotavirus and faecal material from field cases of calf diarrhoea. The faeces of experimentally infected calves contained rotavirus for at least 3 days. Pathogenic bacteria were present in one calf only and this calf also showed the most severe gastroenteritis.

Eight calves were vaccinated with a live rotaviral calf diarrhoea vaccine and subsequently challenged with infective rotavirus. Mild scouring was observed after vaccination, but the calves remained normal after challenge. Rotavirus particles were detectable in the faeces for a few days after vaccination and challenge.

A SURVEY OF THE MOSQUITO AND *CULICOIDES* FAUNAS AT TWO LOCALITIES IN THE KAROO REGION OF SOUTH AFRICA WITH SOME OBSERVATIONS ON BIONOMICS

P. G. JUPP, B. M. McINTOSH and E. M. NEVILL

ABSTRACT: Jupp, P. G. McIntosh, B. M. & Nevill, E. M., 1980. **A survey of the mosquito and *Culicoides* faunas at two localities in the Karoo region of South Africa with some observations on bionomics.** *Onderstepoort Journal of Veterinary Research,* **47**,1–6 (1980).

The mosquito and *Culicoides* faunas were surveyed at Bethulie and Luckhoff in the arid Karoo region, southern Orange Free State, to determine which species occurred, their relative prevalence and the effects of rainfall. The feeding preferences of these insects were also investigated by means of baited catches.

Twenty-three mosquito species and 16 *Culicoides* species were collected. The commonest mosquito species, with their feeding preferences, if known, were as follows: *Culex (Culex) univittatus* Theo and *Culex (Culex) pipiens* Linnaeus, which are strongly ornithophilic and poorly anthropophilic;*Culex (Culex) theileri* Theo, which feeds on sheep and man avidly but is only moderately ornithophilic; *Aedes (Neomelaniconion) luridus* McIntosh, *Aedes (Neomelaniconion) lineatopennis* (Ludlow), *Aedes (Ochlerotatus) caballus* (Theo) and *Aedes (Ochlerotatus) juppi* McIntosh, all of which feed on sheep and man readily and which can aestivate as eggs for up to 20 months but only appear in numbers after rain; *Anopheles (Cellia) listeri* De Meillon, *Anopheles (Celia) squamosus* Theo, *Culex (Culex) quinquefasciatus* Say and *Culiseta (Allotheobaldia) longiareolata* (Macquart). By far the commonest *Culicoides* at both localities was *Culicoides pycnostictus* Ingram & Macfie, which is trongly ornithophilic and also feeds on sheep. The following 5 species were alo prevalent: *Culicoides similis* Carter, Ingram & Macfie, *Culicoides* spec. nov. 1., *Culicoides schultzei* (Enderlein), *Culicoides ondersteportensis* Fiedler and *Culicoides nivosus* De Meillon. The last species is strongly ornithophilic.

EXPERIMENTAL INFECTION OF WARTHOG *(PHACOCHOERUS AETHIOPICUS)* WITH AFRICAN SWINE FEVER VIRUS

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ABSTRACT: Thomson, G. R. Gainaru, M. D. & Van Dellen, A. F., 1980. **Experimental infection of warthog *(Phacochoerus aethiopicus)* with African swine fever virus.** *Onderstepoort Journal of Veterinary Research,* **47**,19–22 (1980).

Although there were no obvious signs of illness following experimental infection of young warthog with African swine fever virus, the animals developed viraemias between $10^{2.4}$ and $10^{3.6}HD_{50}/m\ell$ within the first week of infection, and virus concentrations in a number of lymphatic tissues attained high levels ($\geq 10^{6}HD_{30}/g$). Unlike in blood, and to some extent in the spleen, virus titres in lymph nodes did not decline appreciably during the 33-day observation period, since at the end of the period lymphatic tissues form 2 warthog were still infectious for domestic pigs to which these tissues were fed.

A DESCRIPTION OF THE IMMATURE STAGES OF *KIRKIOESTRUS MINUTUS* (RODHAIN & BEQUAERT, 1915) (DIPTERA: OESTRIDAE), AND THE LIFE CYCLE AND SEASONAL PREVALENCE OF THIS FLY IN BLUE WILDEBEEST

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ABSTRAT: Horak, I. G., Boomker, J. & De Vos, V., 1980. **A description of the immature stages of *Kirkioestrus minutus* (Rodhain & Bequaert, 1915) (Diptera: Oestridae), and the life cycle and seasonal prevalence of this fly in blue wildebeest.** *Onderstepoort Journal of Veterinary Research,* 47, 23–30 (1980).

Descriptions of the 1st, 2nd and 3rd instar larvae and the puparium of *Kirkioestrus minutus* are given. First instar Larvae, which have not previously been described, can be distinguished from other oestrid larvae by the ventral spinulation of segments IV–XII and the spinulation of the anal protuberance.

Of 55 blue wildebeest examined in the Kruger National Park all but two 1-month-old and one 2-month-old animals were infested. First stage larvae are probably deposited in or on the nostrils and may develop within 30 days, initially in the nasal passages and then in the frontal sinuses to mature 3rd stage larvae. Development within the host appears to take longer during the cooler months of the year. Pupal periods vary from approximately 32 days in early or late summer to more than 50 days in winter.

Three of 6 blesbok examined at Badplaas in the eastern Transvaal were infested with 1st instar larvae only of *K. minutus* and it is suggested that blesbok may not be suitable hosts of this fly. Four black wildebeest in the Golden Gate National park in the eastern Orange Free State were not infested.