JOURNAL OF THE SOUTH AFRICAN VETERINARY ASSOCIATION

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Financial subvention by the Department of National Education is gratefully acknowledged.

Geldelike steun deur die Departement Nasionale Onderwys word met dank erken.

THE VETERINARIAN AND THE PHARMACIST

During the past 18 months the SAVA Veterinary Medicines Committee has had a number of discussions with the Director and members of the Pharmaceutical Society of South Africa (PSSA). As a result of these discussions and in reply to their editorial article "Die verskaffing van veeartsenymiddels" of January 1975 in the South African Pharmaceutical Journal, the Committee drew up the article, reproduced below, on behalf of the SAVA. It appeared as an invitation article in Afrikaans in the April edition of the S.A.P. Journal.

The South African Veterinary Association welcomes the opportunity to express its views on behalf of the veterinarian.

At a time when detente is the slogan, it is fitting that several meetings and discussions have taken place between the SAVA and the PSSA. Problems between the professions have been defined in open discussions and great progress has been made to rebuild and re-use bridges that had fallen down or become disused.

Guardian

We recognise and accept the pharmacist as the guardian of medicine, including veterinary medicine, that is to say "stock remedies" as controlled under Act 36 of 1947 (The Fertilisers, Farm Feeds, Agricultural Remedies and Stock Remedies Act) as well as those substances controlled under the Medicines and Related Substances Control Act (Act 101 of 1965).

During the Annual General Meeting of the SAVA in Durban in September 1975 this view was confirmed. It is accepted that all veterinary products should be available to the pharmacist and that he should play his rightful role in the distribution and sale thereof, provided that proper control and discipline is maintained.

Stock remedy

We are convinced that many of the problems that arose in the past between our two professions can be traced back to the obscurity surrounding what exactly is understood by a "stock remedy" and how it fits in with prescription medicine. According to the present definition of a "stock remedy" there are only two groups of veterinary medicines, ie., "stock remedies" that are freely available and registered under Act 36, and other remedies that can only be sold on a veterinary surgeon's prescription. During 1975, Act 36 of 1947 was amended and an attempt made to include a better definition of stock remedies to fit in with the substances of veterinary interest which are controlled under Act 101 of 1965. The amending legislation to Act 36 has already had its first reading in Parliament, and therein the definition has been amended to read as follows: " 'stock remedy' means a substance or any mixture of substances intended or offered to be used in connection with domestic animals, livestock, poultry, fish or wild animals (including wild birds) for the prevention, treatment, diagnosis or cure of any disease, infection or other unhealthy condition, or for the maintenance or improvement of health, growth, production or working capacity, but excluding any substance or any mixture of substances, biological remedy or other remedy in so far as it is controlled under the Medicines and Related Substances Control Act, 1965; or the Hazardous Substances Act, 1973 (Act 15 of 1973)".

The last phrase of this definition used to read as follows: ". . . but excluding a preparation dispensed on the prescription of a veterinarian for a particular patient or group of patients."

Control

According to this new definition it is clear that those substances falling under Schedules 1 to 9 of the Medicines Control Act, are controlled by this Act. Unscheduled substances are controlled under the Act as medicines for human use but not as medicines for animals. At present veterinary medicines are controlled by this Act only as *substances*. Consequently, the unscheduled substances used for the treatment of animals fall under the definition of "stock remedies" and such substances must be registered under Act 36 of 1947. (A limited number of stock remedies contain, among others, Schedule 4 substances e.g., injectable tetracyclines, certain sulphonamides, penicillin and streptomycin-containing mastitis preparations, which are excluded by regulation from control under Act 101 and are controlled under Act 36).

The pharmacist has free access to all registered "stock remedies" and will also be able to supply Schedule 1 and 2 veterinary medicines at his own discretion as soon as the new "stock remedy" definition takes effect.

Problems

At the moment, however, two problems are still to be solved. Firstly, there is a group of remedies generally regarded as veterinary ethical (prescription) remedies but where the active substances are not yet subject to scheduling. This will follow shortly. Secondly, there is the problem of listing in Schedule 1 "All preparations for injection unless otherwise scheduled". From a veterinary point of view, this includes too many remedies that should only be available to the animal-owner on prescription. Self-medication by injection is not a general problem with humans. On the other hand many animal-owners are only too keen to inject their own animals. The PSSA, the SAVA and the Medicines Control Council are at the moment busy negotiating and there is every indication that this problem will be solved and that there should be no further difficulty in the distribution pattern and provision of any veterinary remedy to

Hierdie sienswyses is onlangs in die omsendbrief aan lede van die SAVV in Afrikaans bekendgestel.

pharmacists.

Your justifiable objection to the principle that pharmacists do not have access to certain remedies, should then finally be removed.

Not qualified

The opinion of the President of the Pharmacy Board (Pannall, R., 1975 S.A. Pharmaceutical Journal, 42,6) that the pharmacist is not qualified to diagnose animal diseases is of vital importance to us. A correct diagnosis is obviously an essential precursor to advice and treatment. This field of work must plainly be regarded and accepted as the field of the veterinary surgeon. The availability today of the services and advice of a veterinary surgeon is such that the days when the pharmacist acted as the local adviser and healer of the sick animals of the neighbourhood (mainly because veterinary surgeons were just not available) are something of the past. Where a veterinary surgeon is not readily available, close liaison between the pharmacist and the nearest veterinary surgeon is essential for the improvement of relations and we will welcome this.

The thorough education received by the pharmacist in human physiology and pharmacology gives him the necessary background for advising the medical practitioner on dosages and the selection of remedies and formulas for specific purposes. There is often, however, a vast difference between the physiology and pharmacology of animals and human beings — and also between various animal species. Is the time not ripe for the pharmacist also to receive basic education on a comparative basis in connection with animals, so that he can similarly advise the veterinary surgeon?

By accepting the new definition of a "stock remedy", the veterinary profession has made considerable concessions. When this definition comes into effect, the scheduling of all veterinary medicines is completed, and the proposed amendments to Schedule 1 have been considered, there will be no uncertainty over the exact status of every veterinary remedy.

Ethics

We make an urgent appeal to you to ensure that medicines are supplied correctly and strictly according to the prescribed ethical requirements. In the past the lack of this led to dissatisfaction among veterinary surgeons and contributed greatly to the disturbance of the relationship between the two professions. Unfortunately there will inevitably be individuals in both our professions who will transgress. We appreciate the assurance given by the PSSA that you and your Board will take firm and strict action in such cases. Likewise we give the assurance that our Association and the Veterinary Board can be expected to act drastically against unprofessional conduct and practice by members of our own profession.

The channels are open for any further discussions in connection with unforeseen problems that may arise in the future. We foresee, under these circumstances, a new period of whole-hearted co-operation between the pharmacist and the veterinary surgeon. This can only lead to better control over medicines and benefit the livestock and pet community of the country.

Jl. S. Afr. vet. Ass. 47(2) 77-80 1976

ADDRESS **VOORDRAG**

SOCIOLOGICAL AND ETHICAL CONSIDERATIONS IN SMALL ANIMAL PRACTICE

W.B. SINGLETON*

One of the unique features about life as a veterinarian is the scale of involvement not only with animals but also people. As soon as we qualify, most of us are destined to study or treat thousands of animals of varying species in numerous environments. Associated with these animals are the people who own or care for them. The modern graduate leaves veterinary school very well equipped to deal with the medical and scientific problems which he is likely to meet but to be really successful and, more particularly, if he is to gain the maximum personal satisfaction from his work, his ability to understand and to be in sympathy with the people he meets is of considerable importance. This is perhaps particularly so in small animal practice for in this type of work we meet people of all ages in almost every walk of life. These people have a reason for owning their animal. Sometimes these reasons are trivial and superficial but in most cases the reasons are deeprooted and emotional. The intricacies and subtleties of the veterinarian/client relationship are difficult to impart to students, heavily involved in the scientific aspects of their training but, after graduation, it is important for young veterinarians to quickly acquire an understanding and sympathy towards this relationship. Some do so readily, others find it difficult and a few never seem to acquire this gift. But the difference between success and mediocrity in practice frequently hinges on this aspect of our relationship with the public.

My own experience is largely in dog practice in London and so most of my remarks concern dogs as companion animals. I am sure that similar points can be made about other species.

Dogs have been closely associated with human beings for thousands of years. Fossilised dog skeletons associated with those of humans have been found in Denmark dating back to the Middle Stone Age. Skeletons 9 000 years old were found in Frankfurt in 1936. In Egypt there is evidence of three breeds of dogs being closely associated with man since 5 000 BC. The gradual domestication of the dog is well documented notably by Konrad Lorenze in his books, "Man meets Dog" and "King Solomon's Ring". He postulates that packs of jackals *(Canis aureus)* acting as scavengers, followed nomadic human tribes and at the same time, gave some protection to humans by warning against the intrusion of larger beasts of prey. This association gradually changed from one of predominantly sentry to one of help in the hunting field. Having followed the hunter in the hope of receiving entrails the jackal gradually took to running before instead of behind — they tracked down game and brought it to bay. Later, wolf *(Canis lupus)* became domesticated and was crossed with the already domesticated *Canis aureus,* thus the "symbiosis" began — man by selection had modified the dog to meet his specific requirements. In time these requirements have changed according to race and creed. The Egyptians worshipped a dog-god and many of their tombs have engravings of dogs carved on the walls. Jewish law forbade the crossing of household varieties of dog with the guard dog type. Likewise the cat has been domesticated since ancient times and its importance in society was epitomized by the Egyptians who worshipped these creatures, and to kill a cat incurred the death penalty.

The bond between man and the smaller domestic animals is historical, traditional and with the passage of time it has become basic to the lives of so many. In fact, eminent students of human behaviour have identified and classified the developmental links between human beings and pets. The veterinarian who contemplates practice in an urban community should make an effort to understand these links and recognize their manifestations.

In recent years there has been a considerable increase in interest on the part of psychologists regarding the relationship of humans with their pets notably by Winnicott[7], Anetlyses[1], Levinson[3], and Bridger[2]. For the purpose of this address I should like to quote from a paper by Harold Bridger, Tavistock Institute of Human Relations, London, entitled "Companionship with Humans" given to the Royal Society of Health, 1970[2].

Bridger points out that to appreciate the important and often vital role which an animal can play in its relationship with a child, adult, or family, it is necessary to understand the relevant processes of development in a person from birth onwards.

According to Winnicott[6] the sequence of events starts with the new born infant's fist-in-mouth activities and the exploration of the breast to the more objective experience of grasping a "not me" object. The familiarity which gradually develops through the handling and exploration of these "not me" or "transitional" objects leads gradually to increased confidence in relation to the outside world. Thus develops the human need for possessions to bolster this confidence. The child becomes preoccupied with dolls, toys and frequently animals. Indeed, animals often take over from one of the first "not me" possessions which has significance, namely a warm soft object — a quilt or a teddy bear. It is considered by some authorities that a most significant age in child development in relation to animals is around about five years. It is at that stage that most children begin to mimic their parents. At the same time as the parents are exerting their authority on the child by discipline and training, so the child will find a release for its inherent desire to mimic by testing out its own authoritative powers over a pet. In addition as distinct from these first areas of experiencing transitional objects an animal provides a more complex and challenging possession because of its uncertainty. Thus the child learns to confront anxieties and match

Lecture to B.V.Sc. students, Fac. Veterinary Science, Univ. of Pretoria, Sept. 1975.

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illusory experience with reality but with greater security than might otherwise be possible. As Bridger says — the opportunity to find the situation which provides an inbuilt sensitive balance and control while we learn to cope and acquire identity, power and authority is not always available. The animal is by no means perfect for this purpose but it may be the best we have.

The attitudes and behaviour of family members and other adults in their treatment and relationship with animals are always communicating themselves to children. Children are sensitive to "the music behind the words" of adults. They can sense the "steel" and cruelty behind the honeyed expression, they can sense the humanity and kindness behind the strict command — so can animals. Thus the child can test out feelings, ideas, fantasies — little wonder then that animals find such an important place in so many homes. As Levinson[3] says "Being close to nature and associating with animals is a basic human need." Of course, most children pass through this period of intense involvement with animals but the significance of the relationship remains, so to speak in the background, ready to emerge in later life. I have been in practice long enough now to have seen a number of toddlers grow into teenagers and later marry. During the late teens the interest in animals declines but shortly after marriage many young people return with a new pet and a new era has commenced. What inspires these young people to acquire an animal at this stage of their lives? Is it a desire to share a living creature before the first child arrives — is it that marriage imposes stresses which can be sublimated by a pet in the home?

Levinson considers that many women in early marriage try to protect themselves from the awareness of their own inadequacies as mothers or from their fears of taking care of the newborn by caring for a pet. By tolerating the inevitable mess created by pets, by tolerating the shedding of hair, the damage to curtains, carpets, furniture, etc., and learning to cope with the additional work involved, helps prepare the prospective parent for all the additional work and mess created by the newborn and growing child. If she can cope with the pet, the mother-to-be gains confidence in her ability to cope with a child.

A pet in the family often establishes a continuity, particularly in the mind of a child. People nowadays move about much more than they used to do, as a result families often do not put down roots in a community. For instance, between 1962 and 1972, more than half the families in the U.S.A. moved every five years.

To many children, these moves are traumatic but the companionship of a pet helps enormously to bridge the gaps and to give confidence during periods of readjustment. To a lesser extent parents may benefit similarly.

In modern life, many fathers become extremely involved and absorbed in their businesses and careers and, as a result, become detached from the family. I have often seen how a dog in the home, which requires walking regularly, exercise at the weekend and supervision regarding general care, has the effect of reviving the bond between father and children.

There seems little doubt that such aspects of modern life do have real relevance. People travel much more easily these days, young people leave the family circle losing the parental and grandparental influence and stability, young people seek wealth and affluence far afield. The challenges to young people are no doubt the mainspring of our existence, but the grandiose ideas, the illusions which are conjured up and which inspire the desire for change and new experiences are seldom without pitfalls. Life these days is complex and difficult — jobs, marriages, families all bring their problems to a varying degree. People attempt to escape from the stresses in a variety of ways — amongst them the acquisition of possessions including of course, animals, these are often a link with happier and more secure days in the past. And this continuing involvement helps people through middle age and often through the lonely days of old age.

This aspect is very important — it is estimated that in the U.K. with a population of 55 million there are about four million lonely people. Recently a controlled study on the effect of cage birds upon the lives of elderly (75 - 81 years) and lonely people was carried out by Mugford and McComisky[6] in Hull (East Yorkshire). The results were quite remarkably beneficial, not only directly insofar as the relationship with the bird was concerned, but indirectly by helping those people to displace the "monotonous awareness and discussion of past and pending medical ailments" but also by increasing their sociability with other people, particularly children who shared an interest in the birds.

As I hope I have indicated, the link between humans and their pets is frequently very close and often complex. It follows that the veterinarian's role in this relationship in times of trouble is important for he may be involved either in breaking or maintaining this link. Not only must he apply his scientific training to the problem; he must also ensure that this intervention does not have a traumatic effect upon the patient or owner. It is important to appreciate from the beginning that he is interfering in an intimate relationship and that the utmost understanding and tact is called for. There is a great danger these days for the veterinary scientists who leave our schools to be so engrossed and obsessed by the scientific approach to a clinical problem possibly involving a specific organ, that they become detached from the owner and even from the animal as a whole living creature. If this attitude is apparent to the client it can have quite disastrous effects upon the veterinarian/client relationship and the only way this can be avoided is by acquiring the art of communication.

The extent to which companion animals are of increasing importance at least in Europe and the U.S.A. can be judged from a study of population trends.

In the U.K. there are 5,83 million dogs giving a dog-to-human ratio of 1:9,4.

In France (51 million people) there are 7,48 million dogs — ratio 1:6,3.

In West Germany (61 million people) there are 2,42 million dogs giving a ratio of 1:25.

The United States has 307 million people and 36,50 million dogs. The dog-to-human ratio is therefore 1:5,8. Incidentally, the pet industry in the U.S.A. has a $4\frac{1}{2}$ billion dollar turnover.

Dog ownership in the U.K. is increasing at an annual rate of 4,4%, in France by 5,8%, West Germany 1,3% and U.S.A. 4,5%.

However, in spite of the sociological benefits I have described, the situation is obviously being abused and the keeping of a dog in too many instances is for

superficial reasons often associated with impulsive purchasing with the result that over half a million dogs are destroyed each year in the U.K.

This situation is emphasising the need for more responsible pet-ownership, a tightening of the licensing laws which have remained unchanged for over 100 years and the employment of dog wardens in urban areas to control the stray dog problem. This is particularly important in view of the increasing risk of rabies being introduced from the Continent.

It is estimated that 25% of the 20 million households in the U.K. own dogs, about 3 million households own cats and about 2 million own a budgerigar. Is there any wonder that 50% of the time spent by veterinary surgeons in practice in the U.K. is devoted to small animals.

This situation has raised many problems for the veterinarian — some medical, some surgical — and some ethical. Many of these have resulted from man's obsession with fashionable trends in dog breeding. Many of the atrocious specimens which come into our consulting rooms have lost their ability to function as normal dogs and the unsuspecting owners seek our advice and help. On other occasions the morality of subjecting an ageing animal to major surgery when the prognosis is poor has to be considered very carefully. It is in these circumstances that a precise diagnosis and prognosis is so important for the veterinarian's opinion must stand up to close scrutiny and even challenge. What guide lines can we lay down under these circumstances? In spite of all I have said about the creation and preservation of a good veterinarian/client relationship, I always maintain that the interests of the animal should receive priority over those of the owner. It is my experience that by applying this simple principle the interests of the owners are invariably safeguarded in the long term although it may not be immediately apparent to them. As veterinarians, particularly those who practice, we are entrusted with considerable freedom of action, the ethical limits of which are controlled virtually by a self-imposed code of behaviour. As professional men and women and by virtue of our training and regard for animal life our moral codes and therefore ethical approach to our everyday work is less arbitrary than the approach of many other members of society in which we live and work.

Perhaps one of the most acceptable definitions of moral behaviour is "conforming to, or justified by conscience if not by law". As I have mentioned, in everyday life moral codes are arbitrary. Professor Edmund Leach in his Reith Lecture on Men and Morality (1967) said "Moral rules are those which distinguish between good and bad behaviour and the first point I want to make is that rules are variable. Morality is specified by culture, what you ought to do depends on who you are and where you are. The rules are most explicit about what is bad; the good is then residual".

I hope I may be forgiven for moralizing too much — it is not a popular pastime these days, but the longer I practice the more important it seems to be to stand aside from the rush of life and occasionally ask:

What am I doing and why?

What is my objective and am I tackling it in the best possible way?

This exercise is particularly relevant to clinical surgery and I believe it is morally wrong and therefore unethical to undertake any procedures except for indisputable reasons and by applying the best techniques available.

May I suggest that before embarking upon a surgical procedure the following questions be asked:

Why am I undertaking this operation?

Will it be in the best interests of the animal?

Have I established my diagnosis?

Has the owner persuaded me to undertake this procedure against my better judgment?

Am I allowing the personal circumstances of the owner to influence my decision?

Am I really only trying to increase my turnover?

Have I the ability to undertake this procedure?

Should I not refer the case to a more skilled person?

Do I have the necessary facilities, equipment, staff, etc., to ensure that the animal will have the minimum suffering and discomfort?

I am sure it is quite unnecessary for me to make the point that we should refuse to operate upon animals if the main intention is to deceive show judges or prospective purchasers. I have in mind the straightening of gay tails in terriers, or the cropping of ears, etc. It has been known for veterinarians to insert prosthetic testicles in monorchids and cryptorchids. It is our duty to deplore such procedures and make known our views to the public. I have an absolute abhorrence of ear cropping and any other operation designed to meet the whims of fashion or the fastidious requests of show exhibitors — we have no moral right to inflict pain unnecessarily and I flatly refuse to participate in this form of legalised mutilation.

The other group of conditions which should be mentioned are skeletal deformities which arise in so many cases due to inept selection of breeding stock and a disregard of the crippling conditions which are perpetuated by ill advised breeding. Stifle deformities, hip dysplasia, excessive nasal folds in the brachyocephalics are typical examples. Provided we make it quite clear to clients that they must not use any surgical procedure which is necessary to alleviate a condition, to deceive a judge or prospective purchaser and that it is being undertaken purely for humanitarian reasons — then I feel we are justified in correcting the defect to the best of our ability.

New graduates often find it difficult to advise euthanasia, understandably every case is a challenge — their scientific approach to clinical problems is crying out for practical application. To advise euthanasia seems such a negative and ineffectual thing to do. May I suggest that a restrained approach, a critical appraisal of the case taking into account all the relevant factors and ending in complete personal conviction is more rewarding by far than heroic measures which may distress the patient, the owner and in the end oneself. To advise euthanasia with conviction is not a failure.

My main concern today is to stress our moral and sociological obligations to the society we serve and I am sure I need only mention, rather than elaborate on, the importance of zoonoses and our responsibilities in the public health sector. Early and accurate diagnosis and close cooperation with our medical colleagues is vitally important especially as we often have the opportunity to give early warnings of potential or actual cross-infection.

In this lecture I have endeavoured to touch on a number of relevant points in connection with the veterinarians role in urban society — many have been covered sketchily, but I have tried to emphasise our

main purpose from a scientific and sociological viewpoint and to give meaning to our work. In our everyday life which if successful must of necessity be extremely busy, it is easy to lose sight of our fundamental role, to lose patience with clients, to become obsessed by the business aspects of practice, to allow ourselves and our staff to lower standards. We must remember that everything we do, however small, should be done to the best of our ability. It is only by personal example that we can influence those who work under our supervision. The small animal practitioner working in urban society is the shop window of the profession. It is upon his actions, conduct and standards that the doctors, lawyers, dental surgeons, business executives, clerks, labourers and housewives will judge our profession. It is through their elected representatives in Government that the financial support for the continuing expansion of our profession will depend. We have a tremendous responsibility to ensure that our every day actions, our skills, our service to the community are beyond reproach. In some countries and I regret to say in Great Britain today there is an insidious disease spreading through the community — the symptoms are laziness, broken promises, a lack of pride in work undertaken, an "anything will do when it suits me" attitude. I believe it is up to the professions with their in-built pride and dedication to remain totally immune to these attitudes and to uphold the characteristics which have hitherto brought respect for our way of life.

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INFORMATION INLIGTING

DETERMINATION OF CALF SEX BEFORE BIRTH

Agriculture Canada veterinarians, Drs Betteridge, Eaglesome, Hare, Mitchell and Randall, who have been conducting research on embryo transfers, can now determine the sex of a two week old calf embryo taken from its natural mother's uterus. Embryos of known sex can then be transferred to recipient cows, which carry them to full term.

This development has important implications for future use of embryo transfers in multiplying stocks of genetically superior cattle. Instead of a superior quality dam bearing one or occasionally twin calves, several of her fertilized eggs are transferred into surrogate mothers.

The procedure is that a cow is treated with a hormone to induce superovulation. Instead of one or two eggs being released, as many as 30 may be produced, most of which can be fertilized by artificial insemination with semen from a selected sire. In commercial transfer operations, the fertilized eggs are usually removed five or six days after insemination. The embryos are then transferred to recipient cows whose heat cycles are synchronized with that of the natural mother.

Agricultural Counsellor (Scientific), Embassy of South Africa, 3051 Massachusetts Avenue, N.W. Washington DC 20008 USA. Published by the Department of Agricultural Technical Services, Pretoria.

INFORMATION INLIGTING

FEEDING CALVES ONCE DAILY

There continues to be considerable interest in feeding calves once daily rather than the traditional twice-daily feeding. To date, most of the research has shown lower rates of gain and somewhat greater problems with once-a-day feeding systems, according to Nate Smith, University of California, Davis, dairy scientist. If once-daily feeding is used, follow these guidelines:

1. Do not start the program until the calf is at least 7 days of age.
2. Shift from twice-daily to once-daily feeding by gradually increasing the amount of feed at one feeding and reducing the amount given at the other feeding over a period of four or five days.
3. If milk replacer is used, it should be of excellent quality, preferably, at least 24 percent protein and high-fat, low-fiber.
4. An excellent quality starter should be fed.

Once-daily feeding is no panacea. Labour savings may be realized; however, calves should be observed more than once daily for health problems. The same is true for automatic calf feeders. There is no substitute for keen personal observation.

Squibb, Worldwide Animal Health. *Current Commentary* October 1 1975. *Hoard's Dairyman* (1975) **120,** No. 15:888.

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Jones: Veterinary Pharmacology and Therapeutics: Third Ed.

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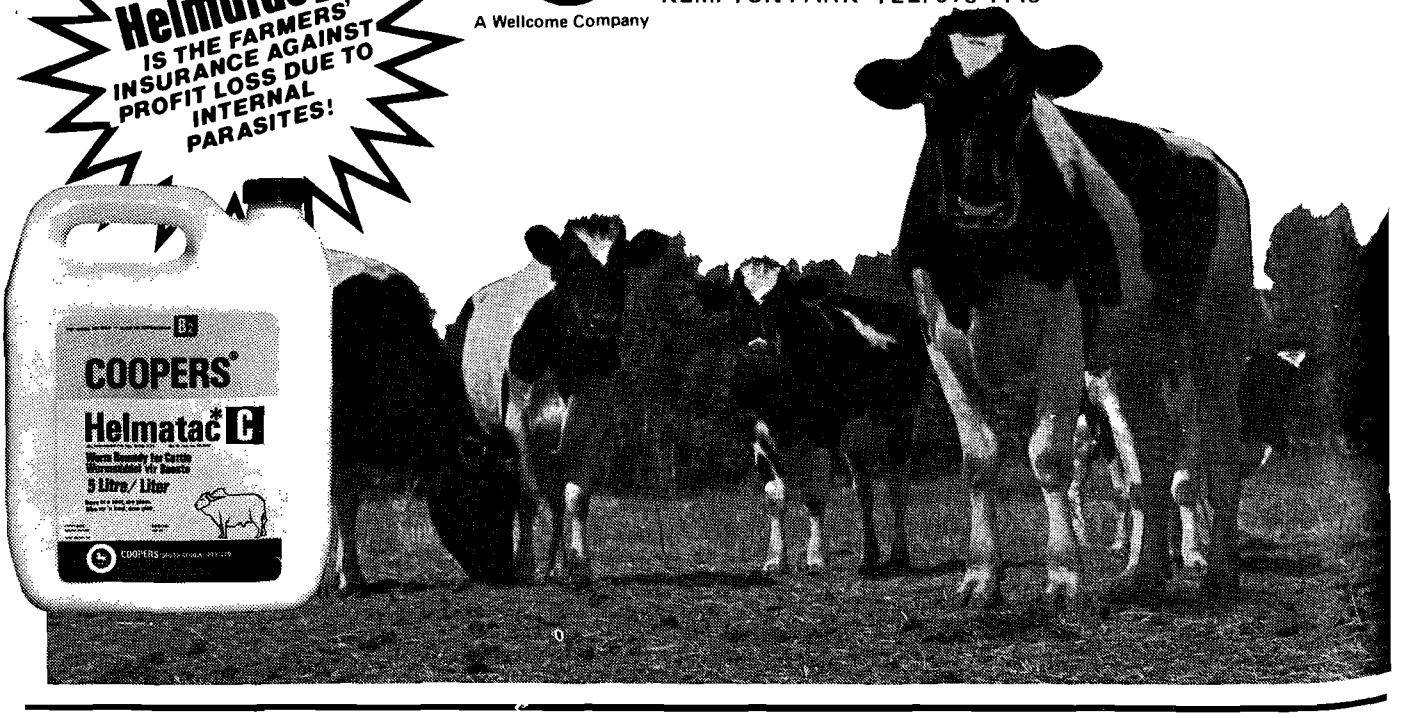

COOPERS DYNAMIC APPROACH TO WORM CONTROL

International J12138

Jl. S. Afr. vet. Ass. 47(2) 83-85 1976

THE PREVALENCE OF DOURINE IN SOUTHERN AFRICA

P.R. BARROWMAN* AND MARIANA VAN VUUREN*

ABSTRACT: Barrowman, P.R.; Van Vuuren, Mariana. **The prevalence of dourine in Southern Africa.** *Journal of the South African Veterinary Association* (1976) **47** No. 2, 83 - 85 (En) Vet. Res. Inst., 0110, Onderstepoort, Republic of South Africa.
The geographical distribution of positive dourine cases found on serological testing at Onderstepoort during the period 1954 to 1975 is recorded, showing major foci of infection occurring in the northern Cape Province, northern Orange Free State, western and north-western Transvaal. Positive cases are also recorded from South West Africa, Botswana, Lesotho, Swaziland and Rhodesia.

INTRODUCTION

Dourine is one of the oldest recorded diseases of horses which was carried with the horse to many parts of the world. Control measures based on serological testing and the waning importance of the horse at the beginning of the present century led to the elimination of the disease from many of the developed countries of the world. Dourine still, however, occurs in the countries of southern Africa and remains a problem to owners and breeders where the horse is of importance for work and leisure.

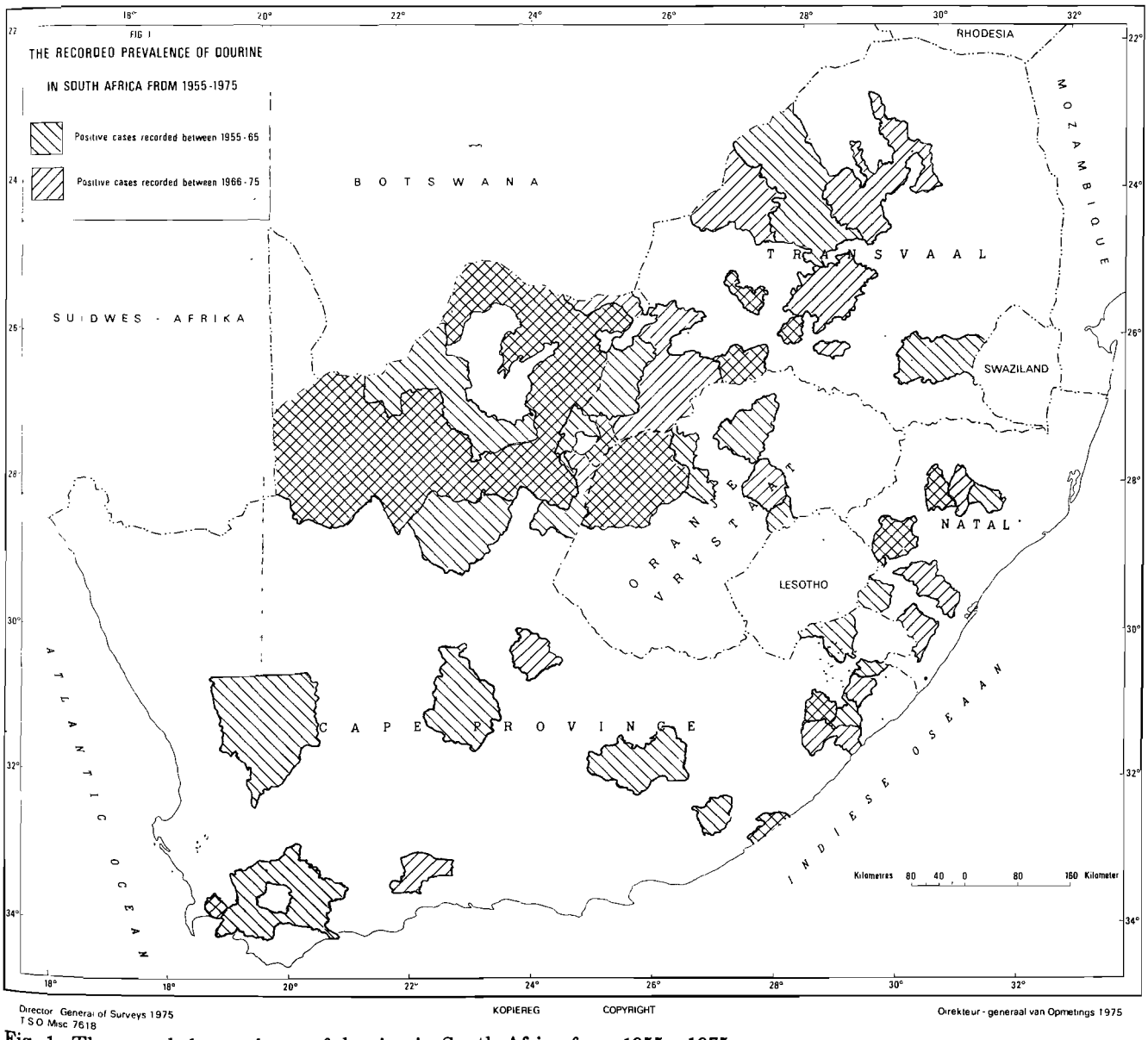

Fig. 1: The recorded prevalence of dourine in South Africa from 1955 - 1975.

* Dept. of Protozoology, Veterinary Research Institute, Onderstepoort, 0110, Republic of South Africa.

HISTORICAL REVIEW

The most probable source of introduction of dourine into South Africa was with horses returning from South West Africa following the Herero wars in 1907[5]. The geographical location of the first recorded outbreaks would support this[6]. An epizootic of dourine occurred in Germany at this time[2] and the shipment of horses to South West Africa may have led to the introduction of the disease, although Walker[6] suggests the disease probably existed there prior to the German South West Africa campaign.

The earliest record of the recognition of dourine in South Africa was between 1907 and 1911 in the Herbert district but it was not brought to the attention of the Veterinary Research Division until 1914[6]. Notification of the disease in South West Africa also occurred at this time[5]. The presence of the disease was subsequently confirmed serologically in 1917 from samples submitted to Watson in Canada[6] who was actively engaged in an eradication programme in North America. Complement fixation testing was introduced for the diagnosis of the disease in the mid-1920's[4] but it was not until 1934 that *Trypanosoma equiperdum* was demonstrated directly in the tissues of infected horses in South Africa[3]. The first indirect isolation of *T. equiperdum* was recorded by Walker[6] in 1918 who observed the parasite in a blood smear from a pup which had been inoculated 15 days previously with blood from a horse showing clinical symptoms of dourine.

Schulz[5] records the presence of the disease in the whole of the Griqualand West, Boshof and Jacobsdal districts, parts of Bechuanaland (Botswana), parts of the Transvaal, Orange Free State and South West Africa during the period 1918 to 1938.

Robinson[4] records an approximate infection rate of 1% in horses tested for purchase in the north-western Cape Province during the second world war. He also records an outbreak in the Bredasdorp and Swellendam districts in the western Cape Province which was controlled with the cooperation of the farming community, and also a high infection rate amongst horses in the Alice district of the eastern Cape Province.

Eradication of the disease on a local basis was attempted in a number of areas[4] and it was included in the schedule of notifiable diseases under the Diseases of Animals Act, Number 13 of 1956. This legislation provided for the gelding or destruction of serologically positive stallions and ovariectomy or destruction of positive mares as control measures for the disease. In view of the fact that many infected horses are clinically asymptomatic[1 5 7] the only reliable diagnostic aid available is the complement fixation (CF) test. Watson[7] in North America carried out some 40 000 serum CF tests during the period 1912 to 1919 and concluded the CF reaction to be a sure, safe and specific method of diagnosing dourine. This is only true in the absence of other trypanosome infections which may give positive reactions to the CF test. This situation occurs in South Africa so that the CF test provides an accurate means of diagnosis, improvements in technique adding to the efficiency of the test. False positive reactions are not known to occur in horses whose sera are not anti-complementary. Sera from donkeys and mules are frequently anti-complementary and present problems in diagnosis.

RECORDED DISTRIBUTION

Serum samples from horses are CF tested routinely at Onderstepoort for diagnostic, breeding, purchase, export and import purposes. The geographical distribution of the positive cases recorded during the 20 year period between 1954 and 1975 is illustrated in Fig. 1.

Distinction has been drawn between those cases found in the first and second halves of this period to indicate changes in distribution.

Fig. 1 was not produced as a result of a survey and must be regarded only as the recorded distribution indicating those areas in which dourine is known to occur. The major foci of infection are seen to occur in the northern Cape Province, northern Orange Free State, western and north-western Transvaal, with smaller foci in northern Natal. In the smaller areas of infection recorded before 1965 the lack of new cases would indicate that the measures employed served to control the disease.

A large number of cases were recorded from South West Africa during the period 1954 - 1975 and cases were also recorded from the neighbouring territories of Botswana, Lesotho and Swaziland. The numbers and territorial distribution of the positive cases recorded from tests undertaken for the reasons cited, are summarised in Table 1.

Table 1: **TERRITORIAL DISTRIBUTION OF POSITIVE DOURINE CASES RECORDED AT ONDERSTEPOORT FROM 1954 TO 1975**

Year	Republic of South Africa	South-West Africa	Bots-wana	Lesotho	Swazi-land	Rhodesia
1/7/54 - 30/6/55	41	115	11			
1/7/55 - 30/6/56	28	124	11			
1/7/56 - 30/6/57	84	68	4			
1/6/57 - 31/5/58	18	321	1			
1/6/58 - 31/5/59	7	22				
1/6/59 - 31/12/59	12	44	2			
1960	36	11	3			
1961	55	4	2			
1962	82	1	3			
1963	19	2	3			
1964	35	10	1			
1965	14	4				
1967	5	11	1			
1968	10	12	18			
1969	25	20	11			
1970	24	26				
1971	23	7			2	
1972	61	10				1
1973	31	5		12		
1974	47	5	1	6		
1/1/75 - 31/5/75	15	7		2		
Totals	672	829	72	20	2	1

CONCLUSION

From the available records it would appear that the major foci of dourine in South Africa are in the northern and north-western Cape Province, the northern Orange Free State and the western and north-western Transvaal. As there is no legislated

restriction on the movement of horses within the boundaries of the Republic of South Africa and the disease is transmitted only by the host, the pattern of distribution is subject to variation with outbreaks occurring in previously clean areas.

ACKNOWLEDGEMENTS

The authors wish to thank the Director, Veterinary Research Institute, Onderstepoort, for permission to use the results and the Department of Trigonometrical Survey, Pretoria for producing the map for Fig. 1.

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BOOK REVIEW

RESENSIE

NUTRITIVE VALUE OF AMERICAN FOODS

CATHERINE ADAMS

Agric. Handbook No. 456. Agric. Res. Service, U.S. Dept. of Agriculture, Washington D.C. Nov. 1975

p.p. IV + 291, Tabs. 9. Appendixes 9, Price $5.50

The publication provides values for the calories and nutritive components supplied by various household and commercial quantity units for well over 3 000 items of food on the American market. These values have been prepared to serve the growing need of research groups who conduct dietary surveys and nutritional studies. It would also serve professional and technical personnel who plan and evaluate diets and food supplies.

The book, a soft cover one using A4 page sizes, consists mainly of tabulated data and explanatory notes. Although only foods available in the US are included, it would undoubtedly find great use as a ready reference by those requiring information on the composition and value of foods.

L W v d H

TO THE EDITOR

AAN DIE REDAKSIE

CLAIRVOYANT VETS?

Dear Sir:

In an article in the "Natal Daily News" (25/5/75) Dr N. Shelley, Professor of Neurosurgery, University of Wisconsin, said that nine clairvoyants, tested scientifically, had accurately diagnosed disease conditions in 80% of cases in a group of 17 patients.

Some years ago, while working in Kenya, I questioned a colleague regarding the percentage of correct diagnoses he made in the cases presented to him. Admittedly he was a British graduate with no experience of the tropical diseases and the peculiarities of "Ondiri-disease, Nakuruitis" etc. which occur in that area. He was also in Government Service and accordingly dealt mainly with cattle and sheep, and seeing only a few horses and dogs. He estimates that his diagnoses were correct in about 50% of cases presented to him. In my own case I estimated that about 70% of my diagnoses were correct. I had then been in general practice for 2 years after graduating at Onderstepoort and after spending 3 years in the Laboratory at Kabete.

I now practise in an area where there is nothing like the variety of conditions that occur in Kenya, and after 25 years of research, trade and development, and general practice, my estimate of correct diagnoses on first presentation is about 85%.

In view of the above remarkable achievement by people who presumably had no scientific training in physiology, biochemistry, anatomy, pathology and the like, one wonders whether veterinary and medical training should not perhaps be reduced in scientific content — or perhaps be extended to include more than the purely exact.

Without elaborating further, I would be most interested to know from other members of our profession what their estimates of correct diagnoses are in their respective areas with due recognition to the local conditions.

So far, I know of no clairvoyants amongst us! I may be mistaken.

Yours faithfully,

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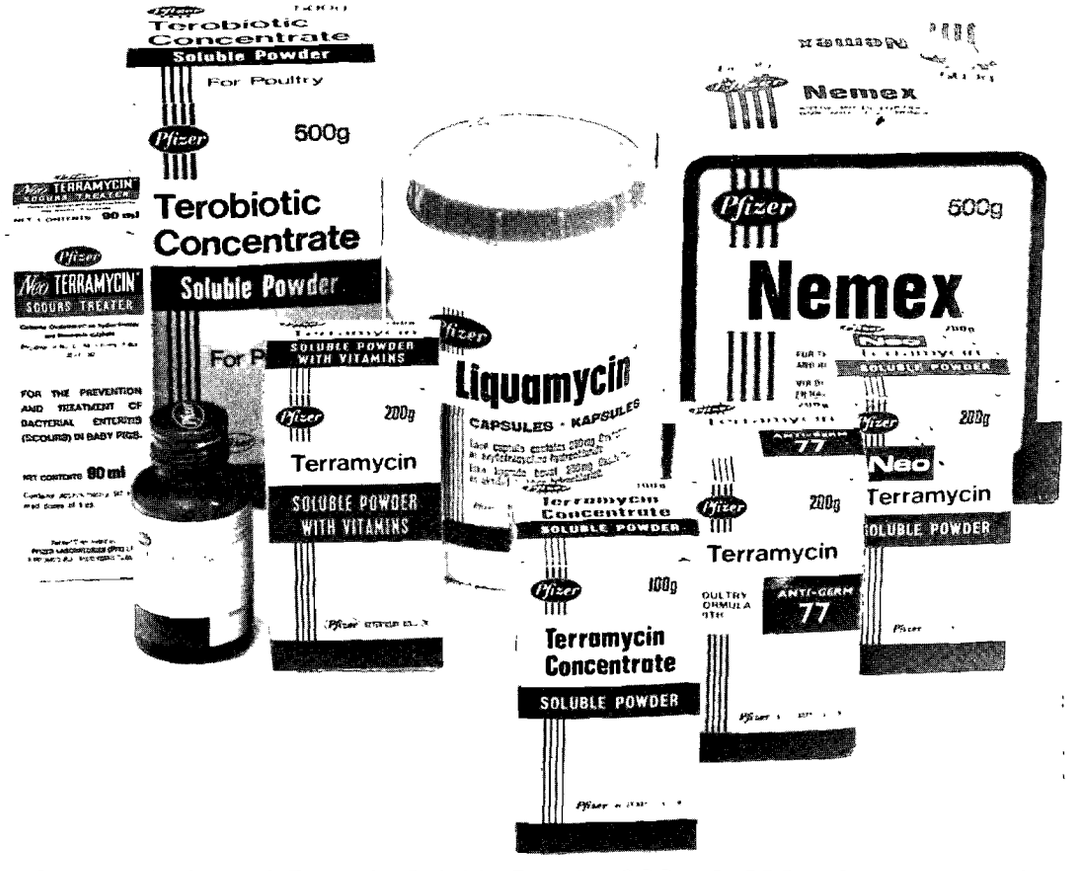

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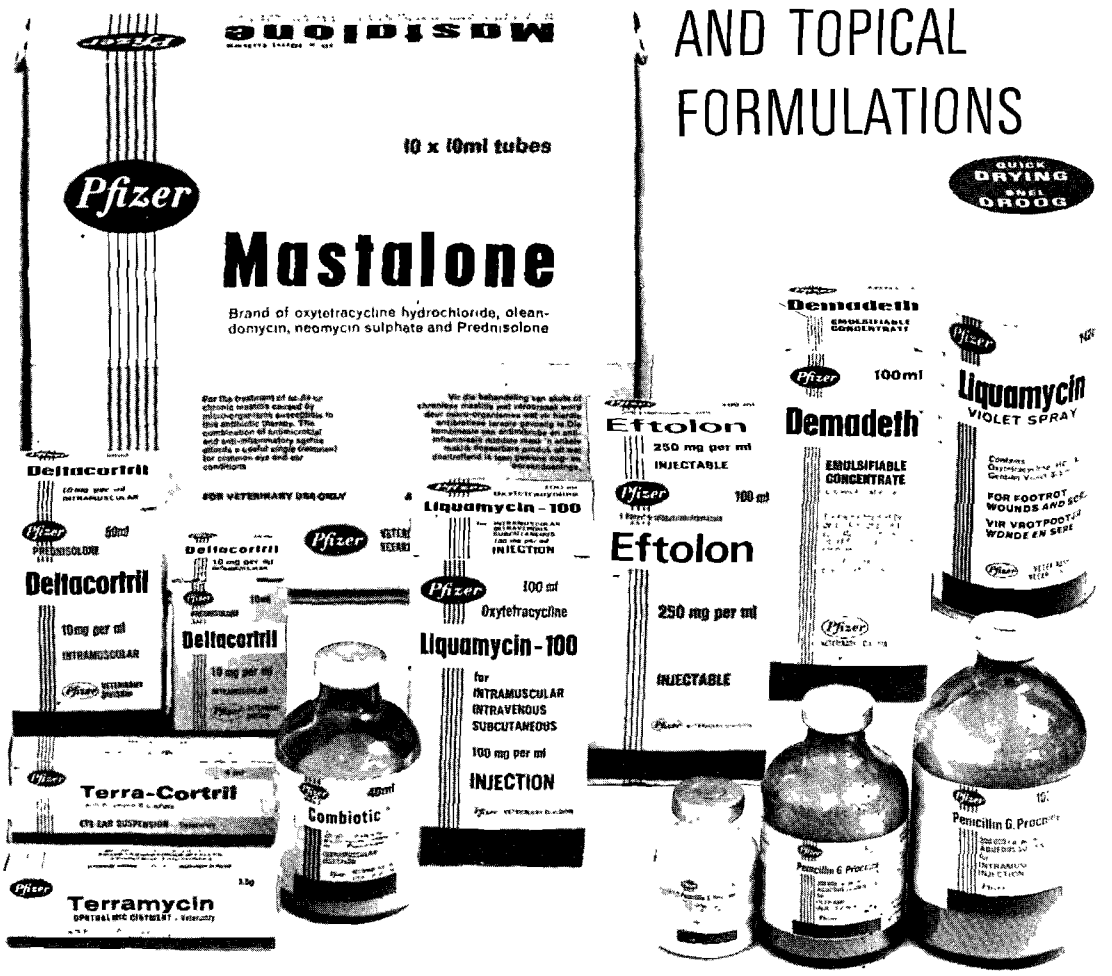

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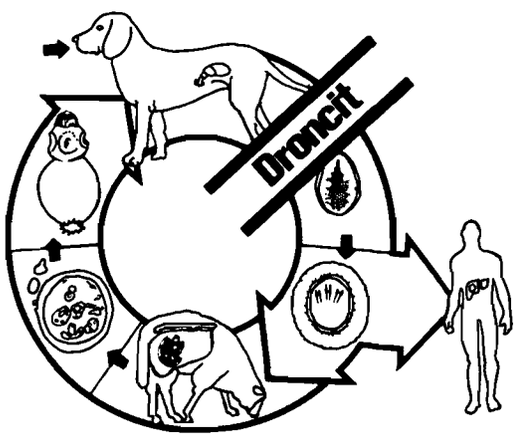

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Jl. S. Afr. vet. Ass. 47(2) 89-91 1976

IMMUNITY TO *SALMONELLA* INFECTIONS

C.M. CAMERON*

ABSTRACT: Cameron, C.M. **Immunity to Salmonella infections.** *Journal of the South African Veterinary Association* (1976) **47** No. 2, 89 - 91 (En) Dept. Bacteriology, Vet. Res. Inst., P.O. 0110 Onderstepoort, Rep. of South Africa.

The results of current investigations regarding the mechanisms of immunity to *Salmonella* infections and the use of vaccines are presented. Since chemical suppression of the humoral immune response in chickens does not jeopardise immunization with a live vaccine and immunized guinea-pigs exhibit distinct inhibition of macrophage migration, it is concluded that cellular immunity is a prominent factor in protection.

Data is presented to show that mice and calves can be equally well immunized with either live or inactivated *S. typhimurium* and *S. dublin* vaccines and the practical advantages of inactivated vaccines are discussed.

On contemplating the shortcomings of our knowledge of *Salmonella* immunity, three facets immediately present themselves:—

1. A poor understanding of the mechanisms of immunity.
2. The lack of an *in vitro* assay procedure for determining the immune status of animals.
3. Ignorance with respect to the antigen which is instrumental in the induction of immunity.

Apart from the above we are also confronted with the fact that live vaccines, although quite effective in inducing an acceptable level of immunity, have certain limitations which restrict their application under field conditions. Live vaccines prepared from avirulent rough mutants cannot be used in conjunction with antibiotics, they do not induce colostral antibodies[8] and no rough strain of *Salmonella typhimurium* has as yet been found which is both effective and safe[3].

After proving that a rough mutant of *Salmonella gallinarum* would induce a good immunity in fowls[6] we considered this species to be an ideal model for studying the mechanisms of immunity operating after immunization with a live vaccine. The relevant literature has been reviewed[2 3] and we attempted to depress selectively either the cellular or humoral immune mechanisms through the administration of cytotoxic drugs, injection of anti-thymocyte serum or neonatal thymectomy. The best results were obtained by injecting cyclophosphamide into day-old chicks[2].

As shown in Table 1, this procedure markedly reduced the humoral antibody response to *Brucella* antigen and sheep erythrocytes while the tuberculin reaction remained intact. Chickens thus suppressed could nevertheless be effectively immunized against *Salmonella* infection. Although we were unable to produce the reverse phenomenon, the results obtained indicate that cellular mechanisms play a prominent role in protection.

We also studied the cellular responses in guinea-pigs using the macrophage-migration technique[7]. By means of a soluble protein antigen we were able to demonstrate that the migration of peritoneal macrophages of *Salmonella* immunized guinea-pigs was often markedly inhibited in the presence of antigen. There was, however, an appreciable variation among individual animals and certain animals which were immunized and subsequently challenged sometimes exhibited a poor reaction. The degree of macrophage migration inhibition of a group of immunized animals was nevertheless always greater than the controls; indicating the participation of cellular mechanisms in immunity[7].

The foregoing does not necessarily entirely exclude the possible participation of serum antibodies in protection against infection but sufficient evidence was gathered to demonstrate the activity of cellular events in a situation where the antibody response is feeble.

Notwithstanding the above, we did find that in calves given two injections of inactivated vaccine, the antibody response was marginally better than in animals given only one injection of live or inactivated vaccine[5]. These calves were well protected and at the present state of our knowledge there is reason to believe that both cellular and humoral mechanisms operate but that in certain instances one or the other may predominate.

Table 1: **EFFECT OF CYCLOPHOSPHAMIDE ON THE IMMUNE RESPONSE OF CHICKENS**

GROUP	ANTI-SRBC TITRE	ANTI-*BRUCELLA* TITRE	TUBERCULIN REACTION : INDEX	ANTI-*SALMONELLA* 'R' TITRE (HA)	SURVIVORS AFTER CHALLENGE
CYCLOPHOSPHAMIDE + ANTIGENS*	10,4	5,8	1,9	3,9	8/11
ANTIGENS* ONLY	129,5	115,1	2,0	63,3	21/29
NEGATIVE CONTROLS	1,9	0	0,0	2,1	2/30

*All chickens were immunized with the following antigens: Sheeps red blood cells (SRBC), *Brucella abortus* antigen, *Mycobacterium avium* and rough *S. gallinarum*.

Veterinary Research Institute, 0110 Onderstepoort.

Paper presented to the Biennial National Congress of the S.A.V.A., Durban, Sept., 1975.

We have not yet studied the location and nature of the immunizing antigen in any detail. The fact that bacteria (rough mutants) which are denuded of much of their cell wall polysaccharide ('O' antigens) will induce immunity, indicates that the 'O' antigens do not play an important role in this respect. Moreover, the fact that proteins prepared by veronal extraction will inhibit macrophage migration, makes this material a strong candidate as the primary immunogen. In fact, other authors have been able to induce protection using purified protein antigens[1].

Having progressed this far we were interested in further studying the nature of the immune response using *S. typhimurium* in mice as the experimental model. We discovered to our distress, however, that the vaccine strain which we used was very weakly immunogenic. This prompted us to compare the virulence and antigenicity of numerous *S. typhimurium* mutants from abroad. It was found that many rough mutants elicit a high degree of immunity but none of these immunogenic mutants was entirely avirulent. This finding naturally precludes their use in a live vaccine intended for use in the field[3].

We also studied our vaccine strain of *S. dublin* in the same manner and found that it was both effective and safe[4]. An interesting phenomenon we observed was that there is an appreciable species difference with respect to effective immunization. *S. dublin* strain HB 1/17 usually gave an immunity of between 70 and 80% in mice and a cross protection of 28,5% to *S. typhimurium* (Table 2). In guinea-pigs it evoked an average protection of approximately 46% to homologous challenge and approximately 26% to challenge with *S. typhimurium*.

Strain 5765 protected up to 80% of mice against *S. dublin* infection and was generally superior to strain HB 1/17 in this respect (Table 2). It was, however, less effective in protecting mice against *S. typhimurium* (20%). In guinea-pigs it was also less effective than Strain HB 1/17, giving an average of 34% protection against homologous and 20% against heterologous challenge[4].

Experiments in mice using inactivated vaccine were generally most rewarding. Provided that an adequate concentration of bacteria was used a high level of immunity was established in mice to both *S. typhimurium* and *S. dublin* infections[3,4].

largely overcome if two injections of vaccine are administered. At 4° or 25°C it is stable for at least 6 months[4].

Table 3: **KEEPING QUALITY OF INACTIVATED *S. dublin* VACCINE PERCENTAGE PROTECTION IN MICE**

STORAGE TIME		STORAGE TEMPERATURE			
		4°C	25°C	37°C	50°C
3 MONTHS	1 INJECTION	55	50	35	40
	2 INJECTIONS	75	75	80	50
6 MONTHS	1 INJECTION	35	15	15	5
	2 INJECTIONS	85	45	65	60

In order to conclude our studies on *S. dublin* immunity we undertook an experiment in calves[4].

Both live and inactivated vaccines produced immunity in calves. A single injection of live lyophilized vaccine prepared from *S. dublin* strain HB 1/17 protected three of six calves while a formalin inactivated alum precipitated vaccine containing 1% packed cells of *S. dublin* strain 2652V protected five out of six calves against intraduodenal challenge with 2×10^9 *S. dublin* strain 2652V (Table 4). Two calves which were immunized with an oil adjuvant vaccine were also solidly immune.

We did not investigate the possibility of protecting calves against *S. dublin* infection by immunizing their dams. Live vaccine is ineffective in this respect[8] and Henning[9] reported indifferent results with inactivated vaccine. Moreover, since colostral immunity may jeopardize the immune response of the calf to active immunization, this procedure should be avoided and only be resorted to where calves under the age of 2 weeks are at risk.

In conclusion it can be stated that although a live *S. dublin* vaccine gives a satisfactory degree of protection and is probably adequate for routine immunization, it has certain shortcomings.

The primary disadvantages are that because no

Table 2: **COMPARISON OF THE PERCENTAGE PROTECTION AFFORDED BY LIVE AND INACTIVATED *S. dublin* AND *S. typhimurium* VACCINES IN MICE**

VACCINE		CHALLENGE	
		S. dublin	*S. typhimurium*
S. dublin	LIVE HB 1/17	70 - 80%	20 - 30%
	LIVE 5765	70 - 80%	±20%
	INACTIVATED	70 - 90%	50 - 60%
S. typhimurium	LIVE 34XO	NT	40 - 50%
	LIVE G30D	NT	60 - 80%
	INACTIVATED	NT	80 - 90%

The duration of immunity produced by concentrated inactivated vaccine lasts for at least 3 months (Table 3). *S. typhimurium* vaccine is very stable even at 37°C[3] while *S. dublin* deteriorates at high temperature (37°C and 50°C), this deterioration is

suitable strain of *S. typhimurium* has been found[3] with which it can be combined, its spectrum is restricted to paratyphoid caused by *S. dublin* only. A live vaccine also cannot be used concomitantly with antibiotic therapy which is a major drawback in in-

Table 4: **IMMUNIZATION OF CALVES AGAINST** *S. dublin*

VACCINE	NUMBER OF INJECTIONS	ANTIBODY RESPONSE (H.A.)	MOUSE* PROTECTION %	SURVIVORS AFTER CHALLENGE
NONE	0	1,2	8,0	1/4
LIVE HB 1/17	1	4,3	6,6	3/6
INACTIVATED ALUM PRECIPITATED	1	0,5	15	3/4
	2	6,0	30	2/2

* As indicated by a passive serum protection test

tensive calf rearing establishments or when active outbreaks of the disease have to be controlled.

An inactivated *S. dublin* vaccine can readily be combined with other serotypes such as *S. typhimurium* and *Salmonella bovismorbificans*[10] and although two injections are required, such a vaccine may be used in conjunction with antibiotics when necessary.

ACKNOWLEDGEMENTS

I wish to thank the Editor of the *Onderstepoort Journal of veterinary Research* for permission to reproduce certain data.

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INFORMATION INLIGTING

RESULTS : BRITISH MASTITIS CONTROL PROGRAMME

The South African Agricultural Counsellor in London reports that at a recent press conference (30 October 1975) Beecham Animal Health announced the results of a large-scale mastitis control programme carried out in Somerset in south west England for three years. Farmers participating in the full mastitis control group agreed to treat each cow at drying off with 500 mg benzathine cloxacillin in each quarter, to have their milking machines tested and to correct any faults, and to practise teat dipping.

Data covering cell counts, milk production, milk quality and clinical mastitis, were assessed from herds taking part in the programme. Cell counts were analysed from all 507 herds taking part in the programme. Milk production records for the full mastitis control group (188 herds) over a three year period were compared with milk production figures from Milk Marketing Board records from herds in the south west of England, which were carrying out no specific mastitis control programme. The milk quality data compare the findings in 159 herds in the full mastitis control programme with those in 247 herds in the partial and no mastitis control groups. Cases of clinical mastitis during the three year period were recorded in 73 herds in the full mastitis control group.

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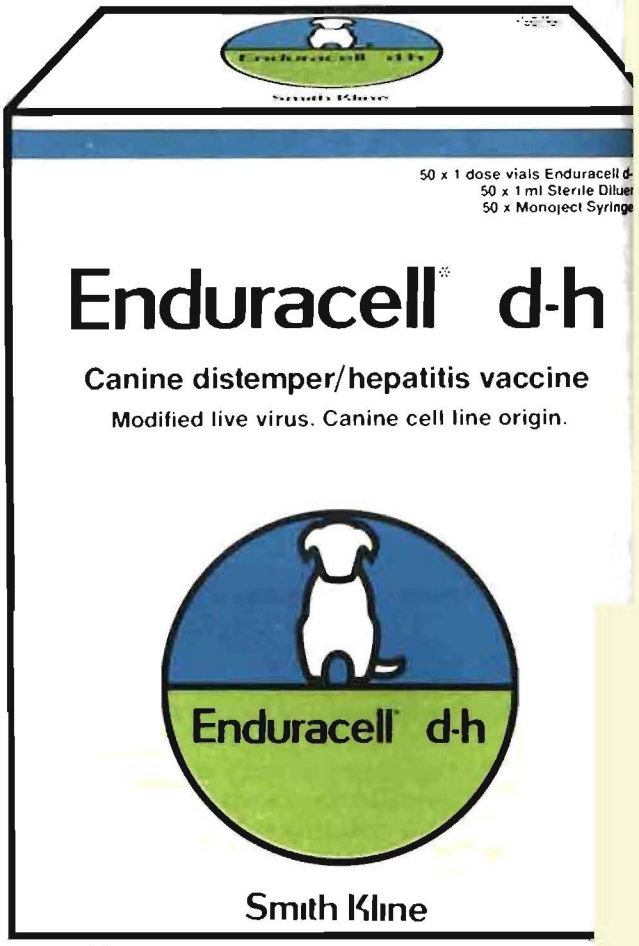

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Jl. S. Afr. vet. Ass. 47(2) 93-94 1976

STUDIES ON THE EPIDEMIOLOGY OF RIFT VALLEY FEVER

R. SWANEPOEL*

INTRODUCTION

In Southern Africa, Rift Valley Fever (RVF) was first recognized as the cause of a major epidemic affecting sheep, cattle and humans in 1950 to 1951[1]. In livestock it was characterized by abortion and death of young animals. Outbreaks were subsequently diagnosed at intervals of years and it was surmised that in the intervening period the virus persisted in circumscribed endemic foci[5 6 12]. Spread from these foci was possibly triggered by seasons favourable to vectors. A new virus, Wesselsbron (WB) produced a pattern of disease in sheep similar to RVF and was isolated in 1955[11]. It was observed that the two viruses tended to occur together in outbreaks[13] and hence a combined vaccine was issued[2].

In Rhodesia the results of retrospective serum-virus neutralisation tests suggested that RVF had been responsible for the high incidence of abortion seen in cattle in Matabeleland during 1955[3].

However, the first isolation of RVF virus followed after the introduction in 1957 of routine testing of aborted sheep and cattle foetuses through inoculation of mice. Virus was obtained from 10% of 79 cattle foetuses tested during the year[9 10]. A proportion of foetuses was tested each year thereafter until 1962 without further isolation of virus (unpublished reports). The purpose of the present paper is to summarise observations on RVF in Rhodesia subsequent to 1962.

METHODS

The disease was not diagnosed again until a major epidemic occurred in Mashonaland in 1969. The outbreak was characterized by a particularly high death rate in young cattle in some herds as well as by abortions.

The mouse colony at the laboratory was inadequate to meet the demands of the outbreak and recourse was made to histopathological examination of liver specimens for diagnosis of the disease. At the same time fresh liver and foetal brain specimens, submitted for isolation of virus, were stored at -20°C for future examination. Fortuitously, 800 cattle sera had been collected in Mashonaland immediately prior to the outbreak for the purpose of an unrelated survey. Further sera were taken during the outbreak and from that time onwards a collection has been built of cattle sera submitted from all parts of the country for the investigation of abortion and other disease.

Histopathological surveillance of sheep and cattle specimens has been maintained continuously since 1969. Serological and virological tests were conducted on a limited scale from 1969 onwards, but the backlog of tests was not cleared until a virology unit was established at the research laboratory in 1972. All specimens submitted since 1972 have been tested for virus in cell cultures, embryonated eggs and infant mice, and 12 000 sera submitted since 1969 have been tested for antibodies to RVF and WB viruses by the haemagglutination-inhibition (HAI) method[4].

RESULTS AND DISCUSSION

According to the results of histopathological tests, the RVF outbreak of 1969 began in February of that year and continued until May 1970. At least 150 farms were affected mainly in Mashonaland to the west and north-west of Salisbury. Isolated cases were diagnosed in the south-eastern lowveld, the Midlands province and Matabeleland. The results of serum tests confirmed this distribution of the RVF outbreak but indicated that WB virus was active at the same time as RVF. Moreover, WB virus was isolated from mosquitoes collected during the outbreak in Mashonaland[8]. This raised the question of the role played in the outbreak by WB virus virus and it seemed logical that virological examination of the liver and brain specimens in storage would provide an answer.

Very few live isolates of RVF virus could be obtained by the time that the liver and brain specimens were examined in 1972 and an alternative method of testing the specimens was needed. It was found that specimens from 51% of 102 foetuses which had been diagnosed as having histopathological lesions of RVF, produced precipitation lines of identity with RVF haemagglutinating antigen in micro-immunodiffusion tests against RVF immune serum produced in sheep. None of 53 histologically-negative specimens produced lines of precipitation with the RVF antiserum and not one of the specimens reacted with WB antiserum. The specimens were examined by preparing 10 - 20% suspensions of tissue in physiological saline. The same suspensions were clarified by light centrifugation and then subjected to complement-fixation tests with the immune sera to detect RVF or WB antigens. Ninety-two of the 102 histologically-positive specimens fixed complement with RVF antiserum. Antigen titres ranged from 1:2 to over 1:256. None of the specimens fixed complement with WB antiserum.

It was noted within herds that RVF HAI antibodies occurred in cows which had aborted, whereas WB antibodies occurred randomly in cows which had aborted or calved normally. Furthermore, RVF antibodies occurred in the areas of Mashonaland where disease was apparent, whereas WB antibodies were detected in sera from all over the country irrespective of the occurrence of disease. This pattern of antibody distribution has persisted. Over 400 foetuses tested since 1972 have failed to yield WB virus.

The pathogenicity of WB for sheep is well documented but from limited experience, experimental infections in cattle produce febrile reactions only[13]. A young calf which had been infected peripherally in a

Paper presented to Biennial National Veterinary Congress, Durban, Sept. 1975.

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study of viraemia in South Africa succumbed to WB virus and the virus was isolated from a cow which died in the field, although it is not certain that the infection was the cause of death[7]. There was comparatively few sheep in Rhodesia and the specimens which have been examined overwhelmingly represent cattle. The implication is that WB virus causes serious disease in cattle relatively infrequently.

Routine serum tests have shown that a varying degree of RVF activity occurs in most years and isolates were obtained from foetuses in 1974 and 1975 even during winter months. Such minor outbreaks would probably have remained unnoticed prior to the introduction of routine tests and it appears that apart from producing well-defined epidemics periodically, RVF is constantly responsible for a proportion of undiagnosed abortions. The disease tends to recur in the same areas along the north of the watershed. It is striking that RVF recurred in practically the same localities in the Hartley-Gatooma-Chakari area in 1975 as those from which 6 of the original isolates in Rhodesia were obtained in 1957. We now advocate regular use of vaccine in these areas.

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INFORMATION

INLIGTING

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The calculated amount of amphotericin B is taken from the stock solution and added to 5% dextrose in water with a pH above 5.0. The volume of 5% dextrose in water varies with the animal's body weight. Volumes that have been satisfactory are:

10 - 15 lb	150 - 200 ml
15 - 30 lb	250 ml
>30 lb	300 - 500 ml

The daily dosage of amphotericin B is dripped as slowly as possible through the IV catheter (the jugular vein is usually used). Four or more hours should be taken to administer the daily dose. By tying the dog on a short leash, problems with the indwelling catheter have been avoided.

At least 14 daily doses are recommended for treatment of system mycoses. Treatment is continued until the clinical signs have completely disappeared. Daily assessment of the BUN level and urine composition is made during the treatment period. If the BUN rises above 45 mg/100 ml, treatment is stopped until it returns to its original level or is stable at a level below 30 mg/100 ml. Though it may be necessary to discontinue therapy several times during the treatment regimen, the therapeutic results are usually satisfactory. Some animals may require treatment for as many as 18 to 20 consecutive days. Apparently cured patients should be examined repeatedly during the first year after therapy.

To prevent some problems associated with amphotericin B therapy, B-complex vitamins are given daily during therapy. The patient's urine should be kept alkaline, giving sodium bicarbonate as needed.

Squibb, Worldwide Animal Health. *Current Commentary* October 1 1975. *Modern Veterinary Practice* (1975) **56,** No. 7:471.

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EXPERIENCES IN THE DIAGNOSIS OF BRUCELLOSIS IN DAIRY COWS

K.W. KATZ, M.M. GREATHEAD, R.C. COOK AND R. BRITZ.

ABSTRACT: Katz, K.W.; Greathead, M.M.; Cook, R.C.; Britz, R. **Experiences in the diagnosis of brucellosis in dairy cows.** *Journal South African Veterinary Association* (1976) **47** No. 2, 97 - 100 (En) City Health Dept, Box 1477, 2000 Johannesburg, Rep. of South Africa.

The Milk Ring, Serum Agglutination, Biological, Mercaptoethanol and Rose Bengal Plate Tests were used in combination to diagnose brucellosis in cows in some herds supplying milk to Johannesburg. The work was done in conjunction with routine testing of bulk milk supplies for public health control. 4 769 cows in more than 76 herds were involved in the tests. Results confirm that no single test can demonstrate all infected cows in a herd and that repeated testing is necessary. In an infected herd in which Strain 19 adult vaccination has been used some negative and suspicious cows will have to be sacrificed in eradicating the disease.

INTRODUCTION

Milk supplies introduced into Johannesburg are tested routinely for evidence of brucellosis. Samples of each producer's bulked milk are screened by means of the Milk Ring Test (MRT) followed by a biological test (BT) in which the Serum Agglutination Test (SAT) is performed on serum from guinea-pigs inoculated with MRT positive milk. Since 1969 the improved Onderstepoort MRT antigen[10] has been used. Its use results in a higher percentage of MRT positive samples. In order to exclude the false MRT negative results such as were sometimes obtained with the old antigen, 204 samples in 1969 and 520 in 1973 of MRT negative milk samples were tested biologically, all were negative. The new antigen has been shown to be more sensitive and more reliable for testing bulked milk than the old antigen used prior to 1969[4]. The results are summarised in Table 1. Comparative figures for 1968, when the old antigen was used, are included. The incidence of *B. abortus* increased steadily during the first 5 years and still remains above 18 per cent.

Table 1: **INCIDENCE OF BRUCELLA IN RAW HERD MILK SAMPLES**

Year	Milk Ring Test			Biological Test		
	Samples	MRT +	% of all Samples	Samples	BT +	% of all Samples
1968	2 144	452	21,0	452	138 (30,5%)	6,4
1969	2 347	1 302	55,4	1 268	180 (14,2%)	7,7
1970	1 797	1 132	62,9	1 111	264 (23,8%)	14,7
1971	2 434	1 506	61,9	1 477	409 (27,6%)	16,8
1972	2 197	1 487	67,6	1 451	447 (30,7%)	20,6
1973	2 248	1 525	66,1	1 511	414 (27,3%)	18,4

Although all milk sold in Johannesburg is pasteurised, farmers are notified of positive brucellosis results and encouraged to remove infected animals from their herds. Investigations have been made to establish a practical test procedure to assist in identifying infected cows in a number of herds supplying *Brucella abortus* contaminated milk.

The following factors complicate identification of infected cows.

1. Brucellosis is a disease with an incubation period varying from a few weeks to 8 months or more.
2. Latent carrier cows negative to SAT may excrete *B. abortus* in their milk[16]. SAT results may be negative or inconclusive especially in the incubative and late chronic stages[9].
3. False positive MRT and SAT can be associated with Strain 19 vaccination. Vaccinated animals give positive titres for variable periods; these are longer in animals inoculated later in life. It has been estimated that 10 per cent of animals inoculated at 6 months of age retain agglutinating antibodies after 2 years[2]. Adult inoculated cattle retain persisting titres which make SAT unusable for diagnostic purposes[11].
4. MRT results are affected by serum globulins passing through the udder barrier into the milk during colostral and "drying off" periods and in cases of mastitis. In these cases MRT reactions rarely exceed++[1].
5. Eighty-five per cent of infected cows are estimated to excrete *B. abortus* in their milk[1].
6. When milk tests are used, investigation of dry cows is excluded until they come into milk.

MATERIAL AND METHODS

The brucellosis status of cows in the herds tested was variable and largely unknown. Extensive use of Strain 19 vaccine in heifer calves and adult cows complicated the situation. Owners could seldom give a precise history of all their cows. The introduction of cows into and the removal of others from herds supplying the City with milk is uncontrolled. The herds investigated were voluntarily made available by producers who were not compelled to remove any infected animals identified by the tests.

Farm bulk milk tanks are now universally used by producers supplying Johannesburg. It has been reported that the dilution of bulked milk can lead to false negative results.

The following tests were used on representative udder milk samples and serum samples from lactating

cows in the herds tested.

1. Milk Ring Test (MRT) using stained antigen obtained from the Veterinary Research Institute at Onderstepoort[10]. Results were classified as positive (+) or negative (–).
2. Diluted Milk Ring Test (DMRT). Milk samples which were MRT positive were diluted 1:5 and 1:10 with known MRT negative milk before the addition of the antigen. Results were classified as positive (+) or negative (–).

This test was carried out in association with the MRT. In the tables, samples recorded as 1:5 DMRT positive were MRT positive and samples recorded 1:10 DMRT were positive to both MRT and 1:5 DMRT.

3. Serum Agglutination Test (SAT). Five tube serial dilutions of bovine serum were tested against Onderstepoort antigen. Titres of 1:40 or higher were taken as positive. No estimations of antibody levels were recorded. The majority of tests were made using dilutions from 1:10 to 1:160 but subsequently dilutions from 1:12,5 to 1:200 were used.
4. Biological Test (BT). Guinea-pigs were inoculated intramuscularly with MRT positive milk. Approximately 4 weeks later blood serum was tested by means of the SAT.
5. Mercaptoethanol Serum Agglutination Test (MET) was carried out by adding 0,8 ml of saline and 1 ml of a 0,2M ME solution to 0,2 ml of bovine serum. This was incubated at 37°C for 1 hour. Thereafter serial dilutions were made from this tube and tested with the Onderstepoort Antigen[11]. This test is claimed to eliminate IgM immunoglobulins produced by Strain 19 inoculation; positive reactions indicate IgG immunoglobulins resulting from infection[8].
6. Rose Bengal Plate Test (RBPT). One drop of guinea-pig blood serum was mixed with one drop of Onderstepoort antigen on a white enamel tile and the result was read after 4 minutes of rotation[9].

RESULTS

1. The milk from 2 195 cows in 34 herds, in whose bulked milk viable *B. abortus* had been demonstrated, were subjected to MRT and BT. The vaccinational status of these animals was variable as Strain 19 vaccine had been used in most of the herds.

Table 2: **INCIDENCE OF MRT+ AND BT+ MILK SAMPLES**

Herds	Cows	MRT +	Percent	BT +	Percent
34	2 195	674	30,7	139	20,6

2. Milk samples from 1 424 cows of variable vaccinational status in 24 herds were examined by MRT and DMRT. The results are summarised in Table 3.

Table 3: **COMPARISON OF MRT AND DMRT IN DEMONSTRATING BT POSITIVE SAMPLES**

Herds	Cows	Positive Tests	BT Pos.	Percent
24	1 424	MRT + 258	9	3,5
		$\frac{1}{5}$ DMRT + 52	12	23,0
		$\frac{1}{10}$ DMRT + 208	89	42,8

3. One month prior to testing with MRT and DMRT to identify infected animals, a herd of 85 adult cows was inoculated with Strain 19 vaccine. Within a 20 month period the herd, whose numbers varied, was retested twice more and all MRT and DMRT positive milk samples were subjected to BT. Results are given in Table 4. The six cows which were positive to 1:10 DMRT and BT on 18.2.72 were removed from the herd after the infection had been established.

Table 4: **USE OF MRT, DMRT AND BT TO IDENTIFY INFECTED COWS IN A SINGLE HERD**

Date	2.7.1971	18.2.1972	15.3.1973
Cows	85	88	80
MRT Neg.	29 (34,12%)	49 (55,68%)	54 (67,5%)
MRT Pos.	21 (24,71%)	24 (27,27%)	25 (31,25%)
BT Pos.	ND	0	2 (8,0%)
$\frac{1}{5}$ DMRT Pos.	10 (11,76%)	1 (1,14%)	0
BT Pos.	ND	0	0
$\frac{1}{10}$ DMRT Pos.	25 (29,41%)	14 (15,91%)	1 (1,25%)
BT Pos.	ND	6 (42,85%)	0

ND = Not done.

4. In 16 herds of variable vaccination status SAT on blood serum samples was combined with MRT on milk samples and the results are summarised in Table 5.

Table 5: **CORRELATION OF SAT AND MRT ON SERUM AND MILK SAMPLES RESPECTIVELY**

Herds	Cows	SAT – MRT –	SAT + MRT –	SAT – MRT +	SAT + MRT +
16	794	496 (62,4%)	30 (3,7%)	131 (16,5%)	137 (17,2%)

5. RBPT and SAT were compared on sera obtained from 114 guinea-pigs which had been inoculated with MRT positive milk 4 weeks previously. Results are given in Table 6.

Table 6: **COMPARISON OF SAT AND RBPT ON GUINEA-PIG SERUM**

Samples	Negative	Positive RBPT or SAT	RBPT + SAT +	RBPT – SAT +	BMPT + SAT –
114	81 (71%)	33 (29%)	26 (78,7%)	3 (9%)	4 (12,1%)

6. Milk and serum were collected from a closed herd of 73 cows which had been free from brucellosis for 10 years previously, and tested by means of MRT, DMRT, BT, and SAT as set out in Table 7. Calfhood vaccination was practised and there was no history of abortion but the herd on the neighbouring farm was infected. Routine BT on bulk milk samples indicated that infection had

Table 7: **COMPARISON OF DMRT AND BT IN IDENTIFYING INFECTED COWS WITH SAT NEGATIVE SERUM**

Samples	SAT		MRT		1/5 DMRT		1/10 DMRT*		BT	
	–	+	–	+	–	+	–	+	–	+
73	73	0	58	15	8	7	8	7	2	5

entered the herd. All samples were subjected to MRT and SAT respectively, but only those milk samples positive to MRT were tested by DMRT at 1:5 and 1:10 dilutions. Samples positive to the latter test were tested biologically.

Two months after this series of tests four of the five cows which were biologically positive were retested by SAT and all had become positive. The fifth cow had been sold in the interval.

7. In Table 8 the results are given of tests applied to milk and sera from 198 cows of varying vaccinational status. Initially the milk was tested by MRT and the sera by SAT. All MRT positive milk samples were tested by 1:10 DMRT and BT. Likewise SAT positive sera were tested by MET.

Table 8: COMPARATIVE RESULTS OF FIVE DIFFERENT BRUCELLOSIS TESTS

Samples	MRT	$\frac{1}{10}$ DMRT	BT	SAT	MET
107	–	ND	ND	–	ND
10	–	ND	ND	+	–
8	–	ND	ND	+	+
22	+	–	–	–	ND
5	+	–	–	+	–
3	+	–	–	+	+
6	+	+	–	–	ND
5	+	+	–	+	–
10	+	+	–	+	+
2	+	–	+	+	–
5	+	–	+	+	+
2	+	+	+	+	–
13	+	+	+	+	+
198	– +	– +	– +	– +	– +
	125 73	37 36	51 22	135 63	24 39

ND = Not done.

DISCUSSION

The serological diagnosis of bovine brucellosis by means of serum and milk tests was reviewed by Morgan[8]. Measures taken to eradicate the disease from Northern Ireland herds by MRT and SAT and other tests have been described[1]; although adult vaccination with Strain 19 was prohibited prior to commencement of testing, the interpretation of SAT results were difficult. Vaccinated animals that subsequently became infected make interpretation of results more difficult. False positives may be produced by adult vaccination; false negatives amongst infected in-calf animals may also appear[8].

Several other reports have appeared on the diagnosis of brucellosis using milk and blood tests[5 7 11]. MRT, complement fixation test on whole milk, SAT, serum complement fixation test and RBPT were used but no single test identified all the animals[5]. The overall efficiency of churn MRT in indicating positive herds over a three year period was 73,4 per cent[7]. Agglutination titres in 128 cows remained above diagnostic levels for 2 years after adult vaccination[11].

Reports on the use of RBPT in the diagnosis of brucellosis[9 2] indicate that the test appears to identify cases at an earlier stage than SAT and gives few false negatives. Some cattle sera with nil IU on SAT were positive to RBPT while others with 100-160 IU on SAT were negative to RBPT[9].

Table 2 shows clearly that the MRT will not distinguish between infected and inoculated cattle and BT can only identify cows excreting *Brucella* in their milk. Screening milk samples by MRT will limit the number of biological tests which need to be undertaken.

Table 3 demonstrates that 42,8 per cent of the $\frac{1}{10}$ DMRT samples were derived from infected cows (BT positive), but this test failed to identify the 21 BT positive animals which were positive to MRT or $\frac{1}{5}$ DMRT only. Therefore the $\frac{1}{10}$ dilution of the MRT alone will not identify all infected animals.

This investigation summarised in Table 4 demonstrated that 67,5 per cent of the herd had become MRT negative 20 months after vaccination with Strain 19. $\frac{1}{10}$ DMRT positive cows decreased from 29,41 per cent to 1,25 per cent of the herd during this period. Therefore $\frac{1}{10}$ DMRT cannot be used to isolate infected animals in a recently vaccinated adult herd.

Although the combination of MRT and SAT have been used to eradicate brucellosis[1], the results shown in Table 5 indicate that these two tests will only give an indication of the infection rate in herds containing adult vaccinated cows. Other tests are needed to identify infected animals.

The results summarised in Table 6 indicate a good correlation between the RBPT and SAT. The RBPT can be easily and rapidly performed and should be a valuable screening test for brucellosis.

The herd on which the tests summarised in Table 7 were carried out had been kept "closed" and brucellosis-free for 10 years. Calfhood vaccination was practised and there was no history of abortion but the neighbouring farm was infected. Routine bulk milk biological tests indicated that infection had entered the herd. The four positive cows revealed by BT were latent carriers as they were negative to SAT and were not identified by the latter test alone. It is reported that titres may develop rapidly after calving or abortion[6]. These results emphasize the need for repeated herd tests at short intervals before they are classified as brucellosis-free.

The results in Table 8 show that thirteen of 198 cows were positive to all tests. Four of the BT and SAT positive cows were negative to MET, a test which has been reported to identify infected cows[8]. In these infected animals the absence of the IgG fraction of the immunoglobulin associated with active *B. abortus* infection resulted in a negative MET. Alternatively very recent infection may have caused an initial high IgM titre; retesting might have resolved the situation.

Use of combined SAT and MET for differentiating vaccinated from infected cows appears promising but this investigation shows that a single MET will not identify all infected animals in a herd.

The tests (MRT, SAT and BT) used initially in these investigations were also used for routine testing of bulk milk supplies. They are simple and easy to do with limited laboratory facilities, but guinea-pigs may not always be available for BT. This test is also expensive and not always practical. Negative tests are not conclusive because cows may excrete the organisms intermittently or be infected in organs other than the udder.

In agreement with other authors it was found that no single milk or blood test used was capable of giving a conclusive diagnosis in all cases of brucellosis in the cattle tested. Different combinations of tests were used to find the simplest method of clearing infected cows from affected herds. However, the complexities

of natural brucellosis, complicated by the various unknown levels of immunity resulting from calfhood and adult vaccination present in some of these herds, has shown how difficult it is to identify and remove all the infected animals in a herd without also sacrificing some uninfected and suspect animals.

CONCLUSION

These investigations were conducted in an endeavour to assist farmers in clearing brucellosis from their herds. Extensive use of Strain 19 vaccine in adult cattle over the years leads to great difficulties in the interpretation of serological tests. The use of SAT alone in these circumstances leaves one without conclusive proof that only infected animals have been identified. The MRT cannot differentiate between vaccinated and infected animals and BT will only show up cows excreting *B. abortus* in their milk.

It appears inevitable that an unknown percentage of animals free from infection will have to be sacrificed in any eradication scheme. Farmers owning cows which may be infected but show no symptoms will not part with them unless adequately compensated, particularly when the only evidence of infection has been obtained from routine control tests on their bulk milk supplies.

A small laboratory equipped for routine public health milk control tests can be used to identify herds infected with brucellosis and to isolate individual infected animals. A more comprehensive test programme is needed to eradicate the disease.

ACKNOWLEDGEMENT

The Director, Abattoir and Livestock Market Department of the Johannesburg City Council is thanked for permission to present this paper.

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BOOK REVIEW

BOEKRESENSIE

BIRD DISEASES

L. ARNALL and I.F. KEYMER

Baillière Tindall, London 1975
pp XII, 528. Figs 165 (101 colour) Tabs 12.

As the number of members of our profession, interested in the subject matter of this book, might be regarded as limited, one can, perhaps, not blame the authors for aiming at the larger market of interested hobbyists. Although stated in the introduction and on the back cover that "It should be of particular value to veterinarians . . .", this book is exclusively written for the layman. This is made clear in repeated references to the limited scope of the book and by remarks that diagnosis or treatment of such and such a condition can only be carried out by the laboratory or the veterinarian. Excuses are also offered for presenting chapters on anaesthesia and surgery.

This tendency affects both style and contents. The partial avoidance of technical terms and the often far fetched and sometimes tortuous explanations do not make for easy reading. Viral, bacterial, fungal and protozoan infections are dealt with in less than 11% of the total number of pages. There is only one plate on psittacosis, whereas beak deformities of the budgerigar are depicted on ten plates. Details on histopathology and laboratory diagnosis have been entirely omitted.

There are numerous misleading if not faulty statements, some of which the reviewer feels obliged to quote verbatim:

page 27: "The main difference is that with the mammal, nourishment of the embryo is carried by blood, whereas with birds it is derived from the egg albumen."

page 48: "The air then crosses the mouth cavity called the choana and passes into the glottis . . ."

page 52: "In common with their reptilian ancestors, the body temperature of birds fluctuates; it is believed that when it falls at night, sperm production can then proceed."

page 75: "Calcium is a major constituent of bone, eggshell and muscle."

page 93: "Protozoa, however, are animals in spite of having no chlorophyll."

and so on.

Although the book does contain a quantity of useful information, it adds nothing to what already appears in standard textbooks on the subject. Veterinarians should not allow themselves to become dazzled by the large number of beautiful colour photographs or the overall appearance of the book, as it cannot be recommended in its present form.

F W H

Jl. S. Afr. vet. Ass. 47(2) 101-104 1976

UHT — BEHANDELING VAN MELK

S.H. LOMBARD*

ABSTRACT: Lombard, S.H. **UHT Treatment of Milk.** *Journal South African Veterinary Association* (1976) **47** No. 2, 101 - 104 (Afr, en) Dept Dairy Science, Univ. Pretoria, 0002 Pretoria, Republic of South Africa.

Pasteurisation of milk provides protection for the consumer against pathogens which may be present in the raw milk, and improves its keeping quality. Sterilisation provides indefinite keeping quality but has an undesirable effect on the flavour and nutritive value of the milk. Ultra high temperature (UHT) treatment produces a milk with prolonged shelf life at ambient temperatures yet has practically the same effect on colour, flavour and nutritive value as pasteurisation.

UHT treatment of milk involves preheating to 80°C and then quickly raising the temperature, either by indirect heating in a tubular heater or by direct steam injection, to 130° — to 150°C at which temperature it is kept for 3 to 5 sec. Cooling follows immediately. Systems in operation in South Africa use steam injection for heating to sterilising temperatures. Evaporation cooling is obtained by subjecting milk to a partial vacuum. This removes any water added during heating by condensing steam and also removes steam-volatile off-flavours.

UHT-treated milk is packed aseptically, usually into heat-sealed paperboard laminated cartons. Intact packages can be kept for up to three months. Absolute sterility cannot be obtained by UHT processing. The term "sterilising effect", introduced by Galesloot[4], means the $\log_{10}$ of the ratio of initial spore count to surviving spore count. A spoilage rate of not more than one litre package per 1000 is considered satisfactory. For laboratory control samples of the sterilised milk are incubated at temperatures favourable to germination of mesophilic and thermophilic spores respectively. After incubation the milk is examined for flavour and physical appearance, subjected to the standard plate count and tested for increase in acidity and decrease in stability towards the alcohol test. Milk for UHT treatment must possess protein stability in the alcohol test, be of good bacteriological quality, and a low spore count in particular.

INLEIDING

Drinkmelk word aan pasteurisasie onderwerp om enige teenwoordige organismes skadeloos te stel. Een van die volgende hittebehandelings word toegepas:

63°C vir 30 min (houmetode);
72°C vir 15 sek (hoëtemperatuursnelproses HTSP);
85° - 95°C vir enkele sek (blitsmetode).

Pasteurisasie hou die verdere voordeel in dat die houvermoë van die melk met etlike dae verleng word. Vir 'n onbepaalde houvermoë word melk na verpakking gesteriliseer by sowat 120°C vir 10 tot 15 min sodat alle lewensvatbare organismes vernietig word.

Pasteurisasie het byna geen nadelige uitwerking op die melk se fisiese en chemiese eienskappe nie maar het dié nadeel dat sekere nie-patogene organismes die hittebehandeling oorleef. Tydens verpakking van die melk beland organismes onvermydelik daarin. Derhalwe moet die melk na pasteurisasie en tot verbruik, steeds koel geberg word. Selfs dan tree bederf na ongeveer 'n week in.

In die geval van sterilisasie is die melk gevrywaar teen mikrobiologiese agteruitgang en hoef dit nie onder verkoeling opgeberg te word nie. Tydens die proses tree ongewenste veranderinge in die smaak en voedingswaarde van die melk in. Die behoefte het dus mettertyd ontstaan aan 'n produk wat 'n lang raklewe het, wat in smaak en voedingswaarde gelyk is aan die roumelk waarvan dit gemaak is en wat boonop geen koelopberging vereis nie. Hierdie eienskappe word verenig in Ultra-hoë-temperatuur (UHT)-melk[12].

WAT IS UHT-MELK?

Die International Dairy Federation (IDF)[6] defineer UHT-melk as melk wat met deurlopende vloei onderwerp is aan verhitting by hoë temperatuur vir 'n kort tyd en daarna asepties verpak is. Dit moet —

(a) die houvermoëtoets soos beskryf in IDF Standaard 48:1969 slaag;
(b) troebelheid gee in die troebelheidstoets.

Die troebelheidstoets berus op die beginsel dat weiproteïene nie deur pasteurisasie gedenatureer word nie en dus troebelheid veroorsaak in die toets. Sterilisasie denatureer hulle en 'n helder toets volg[9]. Met UHT-verhitting word al die weiproteïene nie gedenatureer nie en die behandeling is dus nie so straf as in die geval van sterilisasie in houers nie.

Ten einde UHT-melk te onderskei van melk wat in die bottel gesteriliseer is, word in Engels van "long-life milk" gepraat. Dit is melk wat by 135°C tot 150°C vir 3 tot 5 sek. gesteriliseer en asepties verpak is, en wat nie koud opgeberg hoef te word nie.

METODES TOEGEPAS

Die metodes vir UHT-verhitting van melk kan basies in twee groepe verdeel word: dié waarin die melk **indirek** verhit word deurdat dit deur middel van 'n metaalwand van die verhittingsmedium geskei word (gewoonlik in buisverhitters), en **direkte** verhittingsmetodes waarin melk en stoom met mekaar gemeng word. Die proses bekend as **Uperisasie** spuit stoom in die melk en word gebruik in al drie die UHT-fabrieke wat reeds in Suid-Afrika in werking is.

In so 'n apparaat word die melk:

1. tot 80°C voorverhit deur regenerasie;
2. oombliklik tot 150°C verhit vir 3 - 5 sek deur stoom daarin te spuit;
3. deur verdamping in 'n tenk onder gedeeltelike vakuum blitssnel na 80°C verkoel; (water wat daarin gekondenseer het, verdamp sodat die melk na sy oorspronklike samestelling terugkeer);
4. onder aseptiese toestande gehomogeniseer,
5. deur kraanwater na 20°C afgekoel;

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Gelewer tydens die Tweejaarlikse Nasionale Kongres, SAVV, Durban, September 1975. (Effens verkorte weergawe.)

6. in opgaartenks versamel;
7. asepties verpak.

Die verpakte melk word in 'n onverkoelde pakhuis bewaar tot afloop van toetse op die produksierondte. 'n Raklewe van 3 maande word gewaarborg.

ASEPTIESE VERPAKKING

Aangesien die melk na afkoeling ernstige nabesmetting tydens verpakking kan ondergaan, moet aseptiese verpakking geskied ten einde die steriliteit van die produk te behou. Dit kan in verskillende houers verpak word[13].

Kartonne wat gelamineer is met plastiekfilm, word meeste gebruik en val in twee groepe, nl dié wat vooraf klaar gemaak en voor vulling gesteriliseer word, en dié wat van gesteriliseerde materiaal gemaak word enkele sekondes voor vulling. Laasgenoemde tipe is algemeen bekend in Suid-Afrika (Tetra Pak) en word in tetrahedron vorm verkry.

Die Tetra Pak-verpakkingsmateriaal bestaan uit 'n laminasie van een laag elk van aluminiumfoelie en papier en vier lae poliëtileen. Dit loop vanaf 'n rol deur 'n bad waterstofperoksied wat die oppervlak daarvan steriliseer. Dan word die strook materiaal in 'n buis gevorm wat in die lengte met hitte geseël word. Deur 'n pyp word melk onder in die buis gelewer. Die onderpunt van die buis word met hitte verseël en kartonne vol melk word deurlopend van die onderpunt van die buis verpakkingsmateriaal afgesny.

Ten einde enige oorlewende organismes op die verpakkingsmateriaal te dood sowel as om toevallige lugbesmetting uit te skakel, word hitte deur middel van 'n verhittingselement net bokant die vlak van die melk in die gevormde kartonbuis uitgestraal. Op die binneste oppervlak van die buis word 'n temperatuur van 120°C bereik. Dit ontbind oorblywende H_2O_2 en veroorsaak 'n stygende lugstroom wat verdere lugbesmetting voorkom.

UITWERKING VAN UHT-PROSESSERING OP MELKKWALITEIT

KLEUR EN VOORKOMS

Min verandering vind plaas in die melk se kleur; indien enige, is die kleur meer wit omdat van die weiproteïene op die kaseïendeeltjies neerslaan[5]. Volgens Burton[2] neem die tempo waarteen bakteriedoding plaasvind, bokant 130°C baie sneller toe as die tempo van ongewenste chemiese veranderings. Dit verduidelik dan waarom UHT-behandeling in die besondere temperatuurgebied onderneem word.

REUK EN SMAAK

'n Kooksmaak is waarneembaar in die melk direk na prosessering. Dit verdwyn binne enkele dae[1], dus voordat die melk die verbruiker bereik. Deur strawwe stoomdistillasie van die melk tydens afkoeling onder vakuum word meeste stal- en voersmake verwyder en het die produk 'n platter of skoner smaak. Ontlugting voor verhitting verwyder alle suurstof en ontwikkeling van oksidasiesmaak word vertraag. Laasgenoemde defek kan na langdurige opberging voorkom indien suurstof deur die wand van die houer diffundeer. Die aluminiumlagie in die verpakkingsmateriaal vertraag die beweging van suurstof en beskerm die produk ook teen die effek van lig.

OUDERDOMSVERDIKKING

UHT-melk toon 'n neiging om met opberging 'n toename in taaivloeibaarheid te toon en na sewe maande te koaguleer. Melk wat in 'n outoklaaf gesteriliseer is ondergaan nie hierdie verandering nie. Die verskynsel kan nog nie met sekerheid verklaar word nie. Een teorie skryf dit toe aan die werking van proteolitiese ensieme afkomstig uit spoorvormende bakterieë wat na hittebehandeling reaktivering ondergaan. Die defek kan egter suksesvol voorkom word deur van goeie melk gebruik te maak, die melk aan hoë-temperatuur-voorverhitting bloot te stel, of deur die gebruik van polifosfate[5].

REAKTIVERING VAN ENSIEME

Sommige ensieme, nl fosfatase, katalase en peroksidase, word gereaktiveer ná UHT-behandeling. Die fosfatasetoets kan dus nie gebruik word om die doeltreffendheid van die behandeling te bepaal nie. Moontlik kan die proteases ook gereaktiveer word; hieroor bestaan nog nie eenstemmigheid nie[5].

VOEDINGSWAARDE

UHT-behandeling van melk is 'n konserwatiewe vorm van hittebehandeling wat vergelykbaar is met pasteurisasie[10]. Die denaturering wat 'n deel van die proteïene ondergaan, word nie as beduidend vir die mens beskou nie[7]. Geen noemenswaardige veranderinge is gevind in die toeganklikheid van die lipiede, minerale of koolhidrate vir die mens nie. Meeste van die vitamiene word nie nadelig beïnvloed deur UHT-behandeling nie, terwyl 'n afname in konsentrasie van tiamien, askorbiensuur, foliensuur en vitamien B_{12} ondervind word wat byna vergelykbaar is met afnames ondervind tydens pasteurisasie[10]. Vergelykende syfers word in Tabel 1 aangegee.

Tabel 1: **TIPIESE PERSENTASIEVERLIESE VAN VITAMIENE IN MELK GEDURENDE PASTEURISASIE EN STERILISASIE (VITAMIENE NIE GENOEM NIE, WORD NIE GEAFFEKTEER NIE)[10].**

	Pasteurisasie		In-bottel sterilisasie		UHT-Behandeling
	HTSP	Houmetode	115°C vir 30 min	110°C vir 15 min	Direkte of indirekte proses
	%	%	%	%	%
Tiamien	<10	10	35	20	10
Vitamien C	10	20	50	40	10
Foliensuur	0	0	50	40	15
Vitamien B_{12}	0	10	90	60	<10

Tydens langdurige opberging, bv. vir 3 maande, word 'n drastiese vermindering in die hoeveelhede piridoksien en vitamine B_{12} gevind. Verliese aan askorbiensuur en foliensuur tydens opberging hou direkte verband met die suurstofinhoud van die melk.

KWALITEITSBEHEER

Doding van mikroörganismes geskied volgens 'n logaritmiese patroon. Laasgenoemde kan slegs nul benader maar dit nooit bereik nie. Steriliteit (afwesigheid van lewende mikroörganismes in 'n onbepaalde volume) kan nie bereik word nie. Verder is dit onmoontlik om die afwesigheid van iets te bewys; mens kan slegs bewys dat dit aanwesig is[13]. Om dus meer realisties te wees, het Galesloot[4] die term "steriliseereffek" ingevoer, wat 'n aanduiding gee van die mate waartoe bakterieë gedood is. Dit kan soos volg voorgestel word: as die aantal spore in 'n monster verminder word van 10^6 na 10^1, dan is die steriliseereffek 5 (6 minus 1, oftewel log. van die verhouding van die aanvanklike spoortelling tot die oorlewende spoortelling).

Omdat absolute steriliteit nie bereik kan word nie, stel die melkprosesseerder vir homself 'n doel waar 'n sekere graad van besmetting in 'n bepaalde volume behandelde melk geduld sal word. Aangesien prosessering en verpakking ewe veel tot defekte in die produk bydra, moet albei op bogenoemde doelwit ingestel wees. Volgens beskikbare gegewens is die steriliseereffek van UHT-behandeling teen spore van *B. subtilis* ongeveer 8. Dit beteken dat as die rou melk een spoor per milliliter bevat, die UHT-melk wat daarvan geprosesseer word, een spoor in 10^5 liter melk sal bevat, of een in 200 000 halfliterpakkies[8]. In die Verenigde Koninkryk word gemik vir hoogtens 0,1% bederf, d i een literpakkie uit 1 000; in die VSA word gemik vir hoogtens 0,02% bederf[13].

'n Hoë bergtemperatuur moet ook vermy word want spore van *B. stearothermophilus* kan by 'n temperatuur bokant 37°C vermeerder, terwyl die produk reeds by heelwat laer temperature chemiese agteruitgang sal toon[7].

Bemonstering van 'n fabriek se produksie moet statisties korrek geskied omdat daarmee die graad van waarskynlikheid dat besmetting wel in die produksierondte voorkom, geskat word. Te min monsters sal nie 'n redelike lae besmetting vasstel nie; te veel monsters kan weer die vermoë van die kontrole-laboratorium oorskry. Von Bockelman[13] reken dat 50 tot 100 monsters per verpakkingseenheid per produksieronde voldoende is. Monsters word in iedergeval met gereelde tussenposes by die verpakkingsmasjien geneem.

Voor ontleding moet die monsters bebroei word. Die IDF[6] vereis in hierdie verband 30°C vir 14 dae en 55°C vir 7 dae. Von Bockelman[13] sê dat 30 tot 35°C vir 3 tot 5 dae voldoende behoort te wees, en dat onsteriliteit in 60% van gevalle deur gasvorming aangedui sal word.

Die IDF bepaal verder dat UHT-melk na inkubasie aan die volgende standaarde moet voldoen:

(a) die moet stabiel wees teenoor 68% alkohol;
(b) die suurheid, uitgedruk as melksuur, moet nie met meer as 0,02% toegeneem het nie;
(c) die standaard plaattelling moet nie meer as 100/ml wees nie;
(d) die smaak en reuk van die monster moet nie verskil van dié van normale steriele melk wat lank gebroei is nie;
(e) die fisiese voorkoms moet normaal wees sonder sigbare tekens van proteolise of koagulasie.

VEREISTES AAN DIE MELKVOORRAAD GESTEL

In die Transvaalse Standaard Melkverordeninge[11] word geen voorsiening gemaak vir die verkoop van UHT-melk binne munisipale gebiede nie. Die opening word egter gelaat by wyse van die vergunning "ander goedgekeurde metode" onder beide pasteurisasie en sterilisasie (Artikel 40 en 41). Dit is dus nodig dat hier kortliks aandag gegee word aan die gehalte van die melk wat daarvoor aangewend mag word.

Alle onaangename smake word nie deur die strawwe stoomdistillasie tydens prosessering verwyder nie. Slegs roumelk met bevredigende smaak en reuk kan dus gebruik word. Verder moet die melk van goeie mikrobiologiese gehalte en tydens verhitting chemies stabiel wees.

Die alisaroltoets (75% alkohol plus alisarien) sal roumelk uitwys wat tydens sterilisasie onstabiliteit mag vertoon vanweë te hoë suurheid, mineralewanbalans of teenwoordigheid van kolostrum.

Die vegetatiewe selle en hopelik al die spore in melk word deur die verhittingsproses vernietig. Nogtans moet die roumelk van goeie mikrobiologiese kwaliteit wees omdat die hittebehandeling nie alle bakteriese ensieme of afbraakprodukte vernietig nie. Sterilisasie kan nie 'n produk lewer wat beter is as die rouproduk nie. Gewone innametoetse, nl sediment- en reduktasetoetse, direkte mikroskopiese-, standaardplaat- en termodurespoortellings gee 'n goeie aanduiding van die melk se gehalte.

Twee baie ongewenste faktore moet hier genoem word:

(a) Melk met 'n oënskynlike lae bakterietelling mag wel 'n hoë spoortelling hê, veral onder stowwerige melktoestande;
(b) waar van verkoelde plaasmelktenks en losmaathantering gebruik gemaak word, mag die mesofiele bakterietelling 'n lae reduktasetoets aandui, terwyl die psigrotrofe-telling hoog is. Laasgenoemde groep produseer hitteresistente lipases en proteïenases wat chemiese agteruitgang van UHT-melk mag veroorsaak.

Die prosesseerder kan nie slapper innamestandaarde by melk bedoel vir UHT-behandeling toepas as by melk vir pasteurisasie nie. Omdat hy nie kan staat maak op verkoelde opberging en vinnige verbruik van sy produk nie, moet hy juis streng let op die kwaliteit van die melk wat hy verwerk, sowel as op die sterilisasie- en verpakkingsprosesse. Die lewering van 'n swak produk is by UHT-melk tot 'n groter mate as by gepasteuriseerde melk, verantwoordelik vir benadeling van die prosesseerder se goeie naam.

OPSOMMEND

UHT-behandeling van melk plaas 'n produk op die mark met eienskappe wat dit laat uitstyg bo gepasteuriseerde en konvensioneel gesteriliseerde melk. Dit het 'n aanneemlike smaak, byna onverandere voedingswaarde en lang houvermoë sonder verkoeling. In gebiede waar gepasteuriseerde melk beskikbaar is, mag dit, wat prys betref, sterk mededinging ondervind want die prys daarvan is hoër as dié van gepasteuriseerde melk. Dit is egter 'n gerieflikheidsproduk vir 'n groot deel van die bevolking by wie gepasteuriseerde melk of verkoeling nie ger-

edelik beskikbaar is nie.

Wat gesondheidsaspekte betref, is daar geen rede om teen UHT-melk te diskrimineer nie. Roumelk wat nie geskik is om tot gepasteuriseerde melk verwerk te word nie, kan ook nie aanvaarbare UHT-melk lewer nie. Ondoeltreffende prosessering of verpakking van UHT-melk lei tot bederf wat die goeie naam van produk en verspreider spoedig sal benadeel.

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BOOK REVIEW — RESENSIE

LIVING CLOCKS IN THE ANIMAL WORLD

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Charles C. Thomas, Springfield, Illinois, USA.

pp XIII & 221, Figs. 44, Tabs. 0, Publ. Price $11-75

Man has come to think of time in terms of time measured by mechanical clocks. Nature has, however, developed an amazing variety of built in biological clocks which exercise control over many physiological functions. In higher animals clocks which determine breeding seasons, migration seasons and diurnal cycles are well known. In this book the author discusses some of the biological clocks in the fiddler crab, crab, honey bee, earthworm, clam, oyster, snail, sea-horse, frog, toad and salamander. Although not of direct interest to the veterinary surgeon this book should prove interesting to anyone with a broad interest in biology and timing phenomena. The bibliography includes about 250 references which should prove useful to people interested in the subject.

RWW

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Secretary, Executive Editorial Board

Jl. S. Afr. vet. Ass. 47(2) 105-111 1976

VETERINARY PUBLIC HEALTH IMPLICATIONS OF U H T PROCESSING OF MILK

P.J. MEARA*

ABSTRACT: Meara, P.J. **The veterinary public health implications of U H T processing of milk.** *Journal South African Veterinary Association* (1976) **47** No. 2, 105 - 111 (En) City Health Dept, Box 1477, 2000 Johannesburg, Rep. of South Africa.

The introduction of U H T treatment of milk into South Africa raises problems for local public health authorities controlling milk hygiene in their respective areas because of the legislation concerning milk produced and sold for various purposes.

It is essential that the raw milk supply be derived from healthy cows with healthy udders, and be produced under sanitary conditions. Uniform control standards are necessary for U.H.T. milk and other forms of milk supply throughout the Republic. Uniformity is also desirable for efficient local authority veterinary health services. The progress of veterinary control and supervision of Johannesburg's milk supply is discussed to indicate what has been achieved.

There is need for a national scheme for controlling mastitis. Tests for the purchase of milk on quality must include tests for udder health by regular electronic cell counting of raw milk sources. Appropriate mastitis control incentive or penalty schemes for milk producers should be instituted.

STANDARDS OF CONTROL OVER U H T PROCESSING**

The introduction of U H T processing of milk in South Africa has caused a number of questions to be raised which require urgent consideration.

Decision is required whether U H T treatment is to be regarded as a process of **pasteurisation,** which in terms of existing by-laws will require 'fresh' milk intake, or as a **manufacturing process** akin to sterilising or condensing milk or the making of cheese and butter, and which thus will utilise 'industrial' milk.

Should 'industrial' milk be admitted for purposes of U H T milk processing? Can intake of 'fresh' milk and 'industrial' milk into the same factory be reconciled with adequate health control over pasteurisation and U H T treatment? Will health control over raw industrial milk be required by the local authority over and above the conventional testing by plant management of suitability for processing (temperature; titratable acidity; 10 minute resazurin, alizarol and other tests).

Because of differences between various U H T processes, a control standard for the actual temperature/time sterilising process is not easily defined. Control of the end-product will no doubt be exercised by prescribing appropriate incubation tests to ensure commercial sterility of each production batch before release for distribution. What control standards and tests are desirable?

HEALTHY RAW MILK SOURCES

U H T treatment cannot change abnormal or diseased milk into normal milk[72]. Heat processing must not be used as an excuse to relax efforts to control and eradicate disease from dairy stock, and the raw milk supply must be derived from healthy cows with healthy udders[14 45 62 73]. Apart from public health and aesthetic reasons, healthy herds are fundamental for an economically viable dairy industry.

The consumer is entitled to milk and milk products derived from a good quality supply. The Foodstuffs, Cosmetics and Disinfectants Act and the Public Health Act endeavour to provide the legislative control required on a national basis. However, due to economic considerations — cost of implementing controls, adequate staff, etc. — the larger volume of milk produced for butter, cheese and powdered and condensed milk manufacture has been exempt from any form of public health control. Incentives for improved quality of such industrial milk have come from the prices paid for milk of certain chemical standards and the assistance extended to producers by field officers of the various creameries and cheese factories.

A movement is afoot to have the Dairy and Milk Marketing Boards amalgamated into a single more effective body for the control of all milk. U H T may be the catalyst which will bring this about, and at the same time enable better hygienic control to be exercised over industrial milk whereby greater supplies of good quality milk will become available for U H T milk processing.

HYGIENIC DAIRY PRACTICE

The best possible standards of hygienic production of raw milk are necessary for U H T milk, not only for public health reasons, but also because poor hygiene reduces keeping quality and causes spoilage losses and lower grade end-products[45 62]. The loss in the Republic due to souring of milk was estimated at R0,25 million p.a.[14].

The financial implications of purchasing industrial milk at say 10 cents per litre as compared with say 15 cents per litre for fresh milk will be attractive to U H T operators. Representations are likely to be made for industrial milk supplies, presently utilised for manufacturing butter, cheese, condensed and powdered milk, to be permitted for U H T treatment. It is unlikely that the hygienic quality of industrial milk can easily or quickly be upgraded to prevailing fresh milk standards. Industrial milk supplies have room for considerable improvement[24]. Seventy % of the supplies of industrial milk acceptable to a large cheese factory contained more than 13 million

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Presented to the Biennial National Congress, S A V A, Durban, Sept., 1975.

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** See draft regulations, Govt Notice No. R1415, Govt Gazette No. 4802, 25 July 1975.

bacteria per cm³ [5].

Unless the raw milk available for processing is of high standard the final manufactured product is likely to be unsatisfactory in regard to taste, odour, nutritive and keeping quality[62 73]. Contamination of the raw milk with dust, soil, manure, silage and dirt is undesirable because all spores are not destroyed by U H T treatment[21 43 45 46]. Anaerobic spore-forming bacteria survive U H T treatment and have spoilage significance in U H T products[45 46 66]. Gelation in U H T milk may be due to extracellular proteases produced by psychotrophic organisms which survive U H T heating[13]. Suspected physico-chemical phenomena like age thickening in sterile products may well be due to microbial enzymes associated with poor quality milk[21]. As somatic cell content increases in raw milk, flavour quality decreases in processed (pasteurised) milk products[36].

Until such time as the quality of industrial milk has been upgraded to fresh milk hygienic standards, the intake of raw milk to U H T factories should preferably be restricted to the same fresh milk sources presently accepted by local authorities in accordance with their by-laws.

UNIFORM PUBLIC HEALTH CONTROL OF MILK

The continuing trend for raw milk supplies to come in bulk tankers from production areas remote from the cities implies increasing difficulty in supervising the health of dairy herds and monitoring the raw milk.

To ensure proper public health control of the three U H T factories already operating in the Republic, and safe and healthy food for the consumers, uniform regulations are necessary for prescribing control standards and tests for raw milk supplies and U H T end-products.

Conflicting legislation by different local authorities must be avoided and a national standard should be formulated and applied throughout the Republic. This will also obviate difficulties arising from fresh milk producers not approved by a local authority desiring entry to the U H T plant in that municipality, and possibly too, of requests from suppliers of industrial milk. U H T milk and dairy products will certainly not only be marketed locally and will need to be acceptable to all local authorities in all of the provinces in terms of appropriate uniform legislation.

Suggestion of national standards and tests for U H T milk naturally leads to enquiry regarding the reasons why uniform standards should not also apply to the other forms of milk.

Uniform legislation regarding fresh milk does, in fact, apply in the Transvaal. In the remainder of the Republic varying by-laws operate with marked differences in procedures applied by municipalities for controlling their fresh milk supplies, and in their laboratory and other facilities. The anomaly of conflicting standards from one municipal area to another has been criticised[30 35 55 73] and led to the standardisation of Transvaal by-laws. Possibly the Standard Milk By-laws of the Transvaal should serve as a basis for devising national legislation and for prescribing the basic principles for health of herd, producer dairy premises, milk handling, cooling and distribution, health of dairy personnel, quality of raw milk and of end-product.

STANDARD MUNICIPAL VETERINARY PUBLIC HEALTH APPLICATION

Consideration of U H T milk has quite naturally gone on to take account of other types of milk, and in the same way must be taken further with regard to the role of the municipal veterinarian in local authority public health services.

Current evolutionary trends imply modification of the control of milk supplies, and purchase of milk on basis of quality[14 73 74]. In these changing circumstances the veterinary profession must ensure the best possible application by municipal veterinarians in the production, hygiene and processing of milk and supervision of other foods of animal origin.

At present veterinary participation differs from one local authority to another. Control of milk may be vested in a medical officer, veterinary officer or health inspector. Each may emphasise various features according to his background, interests and personality[73]. It is not surprising that different patterns of milk control operate in our cities. For instance, veterinary control with responsibility to the Medical Officer of Health may be confined to health of dairy stock, or it may cover all aspects of milk control[3 30 35 44]. Varying requirements and duties from one local authority to the next also occur in the other spheres of veterinary operation. The need for standardised veterinary participation in local authorities has been stressed by various workers, with special reference to utilising the veterinarian's background knowledge and skills[30 35 55 72].

South Africa's first post graduate degrees in veterinary public health were awarded in 1965. The DVPH especially fits veterinarians for a career in community health and national welfare. Unless prospects of a public health career become devoid of present uncertainties and vagueness of employment, it is unlikely that veterinarians will continue to be attracted to this important sphere of veterinary activity.

The SA Veterinary Association was previously invited to assist in devising a uniform practice[55] and is the most suitable body to consider what function the municipal veterinarian should perform in milk and meat control and other duties. The SAVA should explore this matter with the Preventive and Community Medicine Group of the SA Medical Association, and examine the ideal pattern of the veterinary function for the major South African cities.

VETERINARY PUBLIC HEALTH CONTROL OF DAIRY STOCK

It is essential that the introduction of U H T processing should not result in any relaxation of the present standards of public health control of milk production. On the contrary, improvement and extension is required.

In Johannesburg, veterinarians working under the Medical Officer of Health are concerned with the inspection and health of dairy herds, including laboratory control of milk-borne bovine diseases like tuberculosis, brucellosis and mastitis, and antibiotic contamination of milk. Their experience may be of interest and also contribute towards the development of improved and uniform veterinary public health practice in the Republic. The progress in the control of the Johannesburg milk supply is therefore briefly reviewed.

1. BOVINE TUBERCULOSIS

An eradication campaign in Durban in 1930 revealed 39% of tuberculin reactors in dairy cattle. Subse-

quently 3% of the cows from country areas tested for replacement purposes were tuberculin positive[19 32].

On this account and also because of the continuing necessity in subsequent decades for the state veterinary services to concentrate on controlling more urgent epizootic diseases the progress of the national eradication campaign faltered[41 64]. A voluntary tuberculosis eradication scheme was introduced in 1969. By 1974 2 233 herds qualified for tuberculosis-free certificates and 450 previously infected herds were certified[20]. This represents a minor percentage of herds, but nevertheless constitutes progress towards eradication.

The extent of tubercular infection of herd milk samples in Durban (1933) was 6,3%[32]; Cape Town (1944) 3,5%[34]; Johannesburg (1942) 2,5%[60] and (1950) 1%[47]; and Pretoria (1950) 4%[33].

On account of the tardy progress in eradicating bovine tuberculosis, routine biological herd milk testing was introduced in Johannesburg in 1946 to safeguard against tuberculous milk[47 51]. As indicated in Fig. 1, consumers of milk benefited by being subjected to diminishing volume of tuberculous milk almost to the point of freedom from contamination.

ditional means towards the objective of improving the hygienic standard of milk.

Control and eradication of bovine tuberculosis rests with the state authorities, and no doubt the advent of I N H will be invaluable[38 39 40]. Until this has been achieved, the biological testing of milk supplies must continue.

2. BRUCELLOSIS

Inoculation of all heifer calves between the ages of 3 - 10 months with Strain 19 vaccine has been compulsory in the Republic since December 1968[29], but full-scale inoculation effort has not been maintained[4]. Compulsory inoculation of the animals in herds supplying Johannesburg with milk has been required since 1964[1]. Since 1952 the incidence of *Brucella*-contaminated representative herd milk supplies to Johannesburg, as determined by biological tests has nevertheless increased (Table 1).

While inoculation effectively prevents pregnant cows from aborting and the consequent serious losses of progeny and milk, dairy herds are generally brucellosis-infected and viable bacteria are common

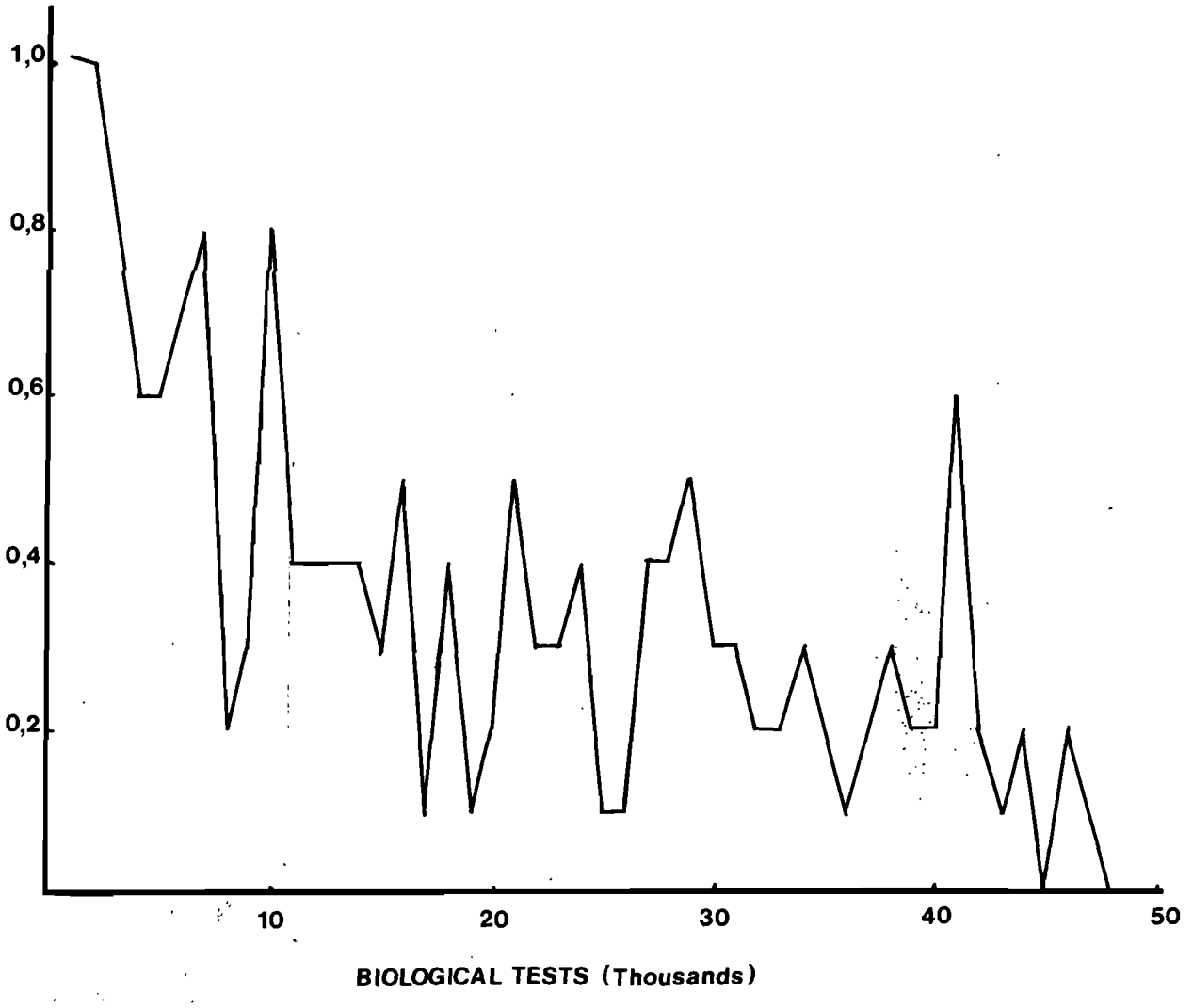

Fig. 1: Incidence of tuberculous bulk (herd) milk, for each thousand consecutively tested samples supplied to Johannesburg 1946 - 1975.

Public health opinion in Johannesburg regards pasteurisation of milk as the final precaution against human infection, but the concept that filth shall continue to be sterilised for consumption is unacceptable. Pasteurisation (U H T treatment) is considered an ad-

in the herd milk supplies. If similar circumstances to the Johannesburg findings prevail in other parts of the country it appears unlikely that 'obligatory calfhood vaccinations for a number of years . . . (will) . . . reduce the incidence of infection in the national herd

to a point where the third phase of eradication can be started'[20]. The plea, made in 1970, that the considerable data on brucella-infected herds which are regularly notified to the State authorities be used to greater advantage to combat the disease in dairy herds[31], bears repetition.

Table 1: **BRUCELLA — CONTAMINATED HERD MILK SUPPLIES JOHANNESBURG 1950 - 1974 RESULTS OF BIOLOGICAL TESTS**

YEAR	HERD SAMPLES TESTED	POSITIVE (%)	YEAR	HERD SAMPLES TESTED	POSITIVE (%)
1950	562	36 (6,4)	1963	1 825	106 (5,8)
1951	1 433	183 (12,8)	1964	1 974	95 (4,8)
1952	1 341	74 (5,5)	1965	2 151	105 (4,9)
1953	1 582	123 (7,8)	1966	2 155	102 (4,7)
1954	1 588	232 (14,6)	1967	2 058	97 (4,7)
1955	1 805	164 (9,1)	1968	2 144	138 (6,4)
1956	1 782	176 (9,9)	1969	2 347	180 (7,7)
1957	1 823	108 (5,9)	1970	1 797	264 (14,7)
1958	1 830	118 (6,5)	1971	2 434	409 (16,8)
1959	1 846	72 (3,9)	1972	2 197	447 (20,4)
1960	1 968	84 (4,3)	1973	2 248	414 (18,4)
1961	2 041	45 (2,2)	1974	2 048	260 (12,7)
1962	1 911	92 (4,8)			

3. MASTITIS

During the period 1943 - 1945 only 5,8% of Witwatersrand milk producers and 6,8% of Pretoria producers did not show mastitis at some time[61]. Over the subsequent two years only about 5% of Johannesburg producers sent in mastitis-free milk, and the average incidence of mastitic herd milk was 39%[50].

Since 1948 regular microscopical examinations have been made of the producers' bulked milk deliveries. The number of annual tests varied from 782 to 4459; average 2883. Examination of the positive dairy herds and advisory information services followed. As shown in Fig. 3, the incidence of mastitic milk supplies dropped from 30% in 1950 to 15% in 1974.

A serious mastitis situation exists in the Republic, and severe losses of milk and finance are caused by

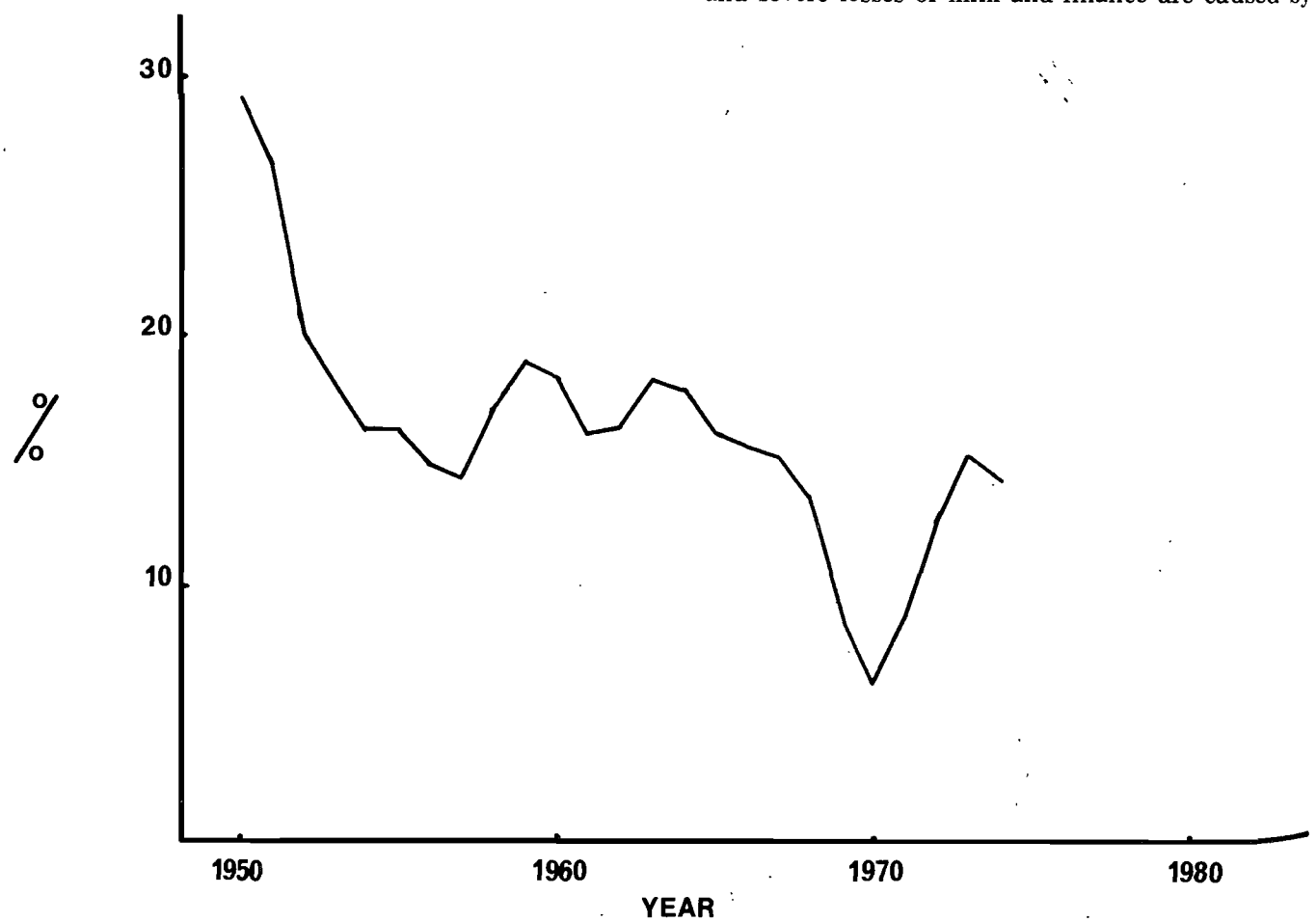

Fig. 2: Mastitis-contaminated bulk (herd) milk supplied to Johannesburg; 1950 - 1975.

mastitis[9 12 18 26 28 42 52]. Approximately 50 to 60 million gallons of milk are lost yearly in South Africa due to mastitis[14]. The composition of milk is adversely affected, and suitability for manufacture is impaired and inferior end-products obtained[14 18 52 70]. The reduced solids-not-fat content can take the herd milk into a lower category for quality purchase[18]. The national loss due to mastitis was recently estimated at R38 million, equivalent to 26% of the annual revenue obtained by producers from the sale of milk[26 28]. These losses underline the vital importance to the dairy industry of healthy udders for producing healthy milk[16 72 73].

Nevertheless, there is great difficulty in motivating the farmer to control mastitis in his herd[8 10 11 16 53]. Penalties or incentives are needed to bring home to the producer awareness of his losses due to mastitis[11 16 17 71]. Suitable impact on producers is necessary by introducing financial incentives for mastitis-free milk and financial penalties for delivering mastitic milk. At the same time the producers must be assured of the essential back-up laboratory and educational services, preferably co-ordinated into a national scheme for control of mastitis[71 72].

The answer to the problem of mastitis does not rest in therapy alone, nor in the use of better antibiotics and other remedies, but in proper herd management, the elimination of pre-disposing factors and spontaneous infections[63 71 72], and a proper milking environment[25 37]. The control of mastitis as a herd problem has been discussed elsewhere[37 58 63 76].

Notwithstanding the many factors which may influence bulked herd milk counts, various national and international mastitis control programmes are now based on somatic cell counts. As the microscopical techniques previously employed to detect mastitis in herd milk (Breed smear, cream smear) are tedious and time-consuming, they have limited application compared with electronic cell counting techniques. For example, approximately 18 000 producers monthly are counted by two machines at Worcester, England[59].

Cell counts are necessary to convince the farmer that it is worth his while to control mastitis and that he must rectify his financial loss[23]. Electronic cell counting is applied as a monitoring procedure for mastitis by the UK Milk Marketing Board and the Ireland Department of Agriculture and Forestry[7 8 56 68 69]. If cell counts remain high for several months a mastitis situation almost certainly exists which requires attention[57]. Too much weight is not attached to a single examination, reliance rather being placed on a rolling annual average. A Swiss Federal Ordinance introduced quality payment on monthly milk analyses including cell count[53]. 95 per cent of Swiss milks have counts less than 200 000 per cm^3. Manufacturing milk in Switzerland must be examined once monthly for analyses of cell count[65]. Somatic cell counts from 200 000 to 500 000 per cm^3 indicate sub-clinical mastitis[52]. Counts in excess of 500 000 per cm^3 indicate problem herds which require special follow-up procedures[54]. Draft European Common Market legislation proposed that high cell count milk above 800 000 per cm^3 be banned[15]. The Norwegian Mastitis Research Laboratory uses the criterion of cell count exceeding one million per cm^3 to reject milk[17]. In Denmark milk samples are tested monthly for cell count and herds supplying milk for liquid consumption with more than 200 000 cells per cm^3 are investigated.

In UK the routine bulk milk monthly cell counts are interpreted as hereunder, and a running 12 month average cell count is used to assess the level of mastitis in the herd, and to afford information on the effectiveness of the control measures[68 69].

Cell count/cm^3	Mastitis level	Possible annual milk yield loss per cow
< 3 00 000	Low	Low
300 000-499 000	Medium	< 225 litres
500 000-699 000	Medium - high	< 337 litres
700 000-999 000	High	< 675 litres
> 1 million	Very high	< 900 litres

Considerable variations in somatic cell counts occur due to calving, lactation, management and environment factors[27 57]. Cell counts nevertheless offer improved supervision over a City's incoming herd milk supplies, provided allowance is made for these variable factors, and taking account of the need for making regular counts (monthly) and for evaluating the trend (running cell counts).

On account of the serious losses of milk and money in this country due to mastitis, a national control scheme is overdue[71 72]. Because of the extensive areas and sparse veterinary services, an embryo scheme appears desirable, initially having reference to limited areas only[28], (e.g. Witwatersrand — Pretoria). It is recommended that serious thought be given to routine electronic cell counts being suitably integrated into the control schemes, and co-ordinated with appropriate mastitis incentive or penalty schemes.

4. ANTIBIOTIC AND INHIBITORY CONTAMINATION OF MILK

Antibiotic contamination of herd milk supplies emerged in 1958 as a veterinary public health problem in South Africa[48]. A survey in Johannesburg the following year revealed that 5,4% of herd milks showed inhibitory effects, of which 3,1% was due to penicillin[49]. A 3 per cent contamination of milk was found in Cape Town in 1965[3]. In Pretoria in 1973 8,3% of herd milk supplies contained inhibitors and 44% of the pasteurised home milk deliveries were contaminated with inhibitory substances[6].

Between 1958 and 1975 extension work in conjunction with regular laboratory testing of the Johannesburg herd milks (1856 test per annum on average) reduced inhibitory substance contamination to 1,4% (1,2% penicillin) (Fig. 3). Because supplies

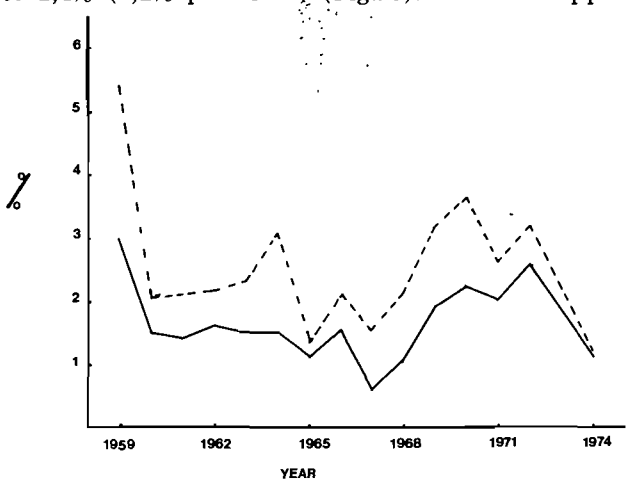

Fig. 3: Inhibitory substance contamination of bulk (herd) milk supplied to Johannesburg 1959 - 1975.
(---- = non-specific — = penicillin)

from contaminated sources are discontinued until freedom from contamination is established, producers are discouraged from including doubtful milk in their bulk supply. The projected introduction of a marker dye in mastitis remedies and the application of price penalties could further improve control over antibiotic contamination of milk supplies.

Because of the elaborate investigations necessary to identify inhibitants other than penicillin, no procedures have been instituted in Johannesburg for identifying such substances. Routine daily protection to consumers of milk against pesticide, insecticide and disinfectant residues will need to await the introduction of simple control tests.

ACKNOWLEDGEMENT

The author is indebted to the Medical Officer of Health, Johannesburg, for permission to present this paper, and to Dr M.M. Greathead for revising the manuscript.

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INDUCED PARTURITION IN CATTLE

1. CLINICAL STUDIES

H.M. TERBLANCHE, L.J. KRITZINGER AND J.S. VAN HEERDEN*

ABSTRACT: Terblanche, H.M.; Kritzinger, L.J.; van Heerden, J.S. **Induced Parturition in Cattle : 1 Clinical Studies.** *Journal South African Veterinary Association* (1976) **47** No. 2, 113 - 115 (En) Dept Genesiology, Fac. Vet. Sci., Univ. Pretoria, Box 12580, 0110 Onderstepoort.

Parturition was induced in 17 Friesland heifers to decrease the possible occurrence of dystocia. The heifers were given 39 mg dexamethasone intra-muscularly on the 267th day of pregnancy and were observed for signs of impending parturition. The time of foetal membrane expulsion was noted.

Parturition occurred on an average of 57 hours after corticosteroid administration. Three heifers required assistance during parturition. In one case a second injection of dexamethazone had to be given. The incidence of retained placenta was 68,8%. Rectal examination five to seven months after induction revealed that 76,5% of all animals were again pregnant. Of the four non-pregnant animals three had received assistance during parturition.

INTRODUCTION

The exact mechanism responsible for the initiation of parturition in domestic animals has been the subject of a large number of publications in recent years. As early as 1957, Kennedy and co-workers[9] came to the conclusion that the foetus plays an important role in the initiation of the normal physiological changes which eventually lead to parturition. Various research workers have since either reported or speculated upon the important role played by the foetus in the initiation of this mechanism[1 2 7 14 16 17 19]. A recent publication[12] specifically deals with the initiating mechanisms in the ewe.

Another subject dealt with by many workers in recent years has been the induction of premature parturition in domestic animals with the aid of corticosteroids. During 1967, van Rensburg[17] succeeded in inducing parturition in a ewe with the aid of cortisol acetate. This was followed by several successful attempts in various species including cattle[1–4 6–8 10 11 13–16 18–20].

A variety of synthetic corticosteroids have been used to date in cattle. These are either the long acting corticosteroids such as dexamethasone trimethylacetate[13 20], dexamethasone esters [1–4 10 11 15 16 18 19] and flumethasone[6 7 10 14 19 20]. Combined glucocorticoids (dexamethasone trimethylacetate and prednisolone) have also been used with success[8 13]. The whole subject of induced parturition has recently been reviewed by Jöchle[5].

The present study was undertaken to ascertain the parturition-inducing effect of exogenous corticosteroids on cows near term in South Africa and to determine the effect of such therapy on their subsequent breeding ability.

MATERIALS AND METHODS

Seventeen Friesland heifers, their ages ranging between 24 and 27 months were used in this study. These animals were referred to our clinic because of the high incidence of dystocia that had occurred in similar heifers of the same group which had all been bred to a single bull known to be responsible for siring large calves.

Most heifers were given a single intra-muscular injection of 39 mg of an aqueous suspension of mixed dexamethasone esters** at 08h00 on the 267th day of pregnancy. In a few cases this could not be done due to unforeseen circumstances. These heifers were treated on arrival at the clinic. In all cases, treatment did not take place more than 10 days before the estimated parturition date. A single animal required a second treatment 5 days after the first.

All the animals were stabled during the time of induction and were regularly observed at approximately 4 hour intervals. The time of expulsion of the placenta was noted and any placenta not expelled within 24 hours of birth was considered to be retained. Daily blood samples were taken in heparin from the jugular vein. Sampling commenced on the day of, but prior to treatment and ended 12 - 18 hours after calving. The plasma fraction of each sample was collected within 2 hours of sampling and frozen until assayed for progesterone content.

All the animals were subsequently returned to their herd of origin and careful breeding records were kept. They were rectally examined for pregnancy between five and seven months after the induced calving and the time interval (days open) between calving and conception was established.

RESULTS

The parturition-inducing effect of dexamethasone treatment as obtained in this study, is shown in Table 1. Only two exceptions to the average pattern were found, viz. heifers no. 746 and 348. The latter received 39 mg dexamethasone at 08h00 on day 269 of pregnancy and had to be treated again on day 274. She, however, started parturition within 36 hours of the second injection. Heifer no. 746 did not respond to the first treatment and was left to calve on her own, within 132 hours of treatment. The average time interval between treatment and parturition for all heifers with the exception of nos. 348 and 746 was 52 hours.

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** Dexafort (Intervet).

Table 1. PARTURITION-INDUCING EFFECT OF PARENTERAL DEXAMETHASONE ADMINISTRATION IN HEIFERS

Heifer No.	Day of treatment (days pregnant)	Day of calving	Calving within hours after treatment
673	267	269	60
674	267	269	60
675	267	268	36
676	267	268	36
825	267	268	36
826	267	269	60
827	267	269	60
349	267	269	60
692	274	275	36
744	267	269	60
745	266	268	60
746	267	272	132
747	267	269	60
209	273	275	60
210	267	269	60
211	268	269	36
348	269	—	—
	274	275	36 (2nd RX)

Table 2. INCIDENCE OF RETAINED PLACENTA FOLLOWING PARTURITION INDUCED BY A SINGLE INJECTION OF DEXAMETHASONE

Heifer no.	Duration of retained placenta in days after calving
673	4
674	—
675	9
676	—
825	—
826	5
827	9
349	4
692	—
744	10
745	7
746	8
747	8
209	14
210	8
211	—

It was necessary to render assistance during birth in three cases. A caesarian section had to be performed on heifer no. 348 following the second injection of dexamethasone. In the second case, no. 746, fetotomy facilitated the removal of a 44 kg calf and in the final case no. 209, traction had to be applied to effect delivery. It is interesting to note that of the animals requiring assistance, two, nos. 348 and 746, did not respond to the first injection of dexamethasone.

The incidence of retained placenta in the 16 heifers responding to a single injection is shown in Table 2. Retained placenta occurred in 11 of the 16 animals i.e. an incidence of 68,8%. The incidence of placental retention in the animals calving without assistance was 64,3%.

Table 3 summarizes the time interval between induced calving and first subsequent oestrus following re-introduction into the herd of origin, the results of rectal examination for pregnancy 5 - 7 months after induction, the number of days between induced calving and subsequent conception, the cycle at which insemination took place and the number of inseminations required by each animal. Of the 17 animals in this study, only four were not pregnant at the time of examination. It is both interesting and important to note that of the four nonpregnant animals, three had had some kind of interference during parturition. This means that 76,5% of all the animals in this study were again pregnant 5 - 7 months after induced calving. Of the 14 heifers calving without assistance, 13 were pregnant at the time of examination i.e. 92,9%. The average time interval between induced calving and conception (days open) was 98,2 days following the use of artificial insemination.

DISCUSSION

The results obtained in this study indicate that the administration of dexamethasone 10 days before full term gestation in the bovine will induce calving within 36 - 60 hours. These results are in accordance with those obtained by a number of workers in various countries[1 6–8 10 19]. Interpretation of these findings leads one to accept the theory of a direct relationship between the foetal hypothalamo-adrenal-maternal-

Table 3. REPRODUCTIVE EFFICIENCY POST-PARTUM AFTER INDUCED PARTURITION

Heifer no.	No. of days between induced calving and first oestrus	Pregnancy diagnosis	Days open between calving and conception	No. of cycle when inseminated	No. of inseminations per conception
673	57	I C 4 mths	101	3	1
674	24	I C 4 "	88	4	1
675	70	I C 3½ "	114	2 and 3	2
676	81	I C 4½ "	81	1	1
825	19	I C 3 "	132	4 and 5	2
826	64	I C 4 "	84	2	1
827	19	I C 4 "	81	4	1
348	61	NIC	—	—	—
349	48	I C 2 "	132	3 and 4	2
692	46	I C 3 "	96	2	1
744	48	I C 2½ "	108	3 and 4	2
745	36	I C 2½ "	95	3 and 4	2
746	34	NIC	—	—	—
747	21	I C 3 "	97	2 and 3	2
209	39	NIC	—	—	—
210	26	I C 3 "	67	3	1
211	84	NIC	—	—	—

ovarian axis in the initiation of parturition for the bovine as postulated by a number of workers[1 7 9 14 16]. One aspect which is of cardinal importance is the fact that a live foetus is required for successful corticosteroid-induced parturition in the bovine[8]. This fact was also reported by Vandeplassche *et al*[16] who could not obtain results in cows with foetal mummification or maceration.

The other significant finding was the high incidence of retained placenta which also corresponds with the results of other workers[1–7 10 18 19]. This presents a serious problem as well as the major drawback in cases of dexamethasone-induced parturition and has led to at least three groups of workers[3 4 11 15] attempting its elimination by the administration of oestrogens either before[11] or simultaneously[3 4 15] with the dexamethasone. Their results, however, seem a bit contradictory and this problem remains unresolved.

Another important aspect of induced parturition is the effect of the high incidence of retained placenta on subsequent fertility. The results of our study clearly indicate that this need not be a problem since 76,5% of all animals were again pregnant 5 - 7 months later. This is in accordance with the results of other workers[1 2 10 18–20].

An important aspect to be borne in mind is the prevention of secondary contamination or trauma in cases with retained placenta as also suggested by Wagner *et al*[19]. This fact is illustrated by our own findings in the following way. Of the 17 animals given corticosteroids, only three had to receive assistance during parturition. Of the four nonpregnant animals at the time of examination, only one had calved without assistance, while the other three are those mentioned above. This means that of the 14 animals calving without assistance 92,9% were again pregnant within five to seven months later.

It is our belief that induction of premature parturition in cattle with the aid of corticosteroids is a helpful tool for solving problems such as amongst others a high incidence of dystocia due to oversized full term foetuses and dropsy of the foetal sacs as described by Vandeplassche *et al*[16]. It may also play a role in improving the breeding efficiency of late calving cows as described by Welch *et al*[20] and in programming the date of parturition if so desired[2].

It must be borne in mind, however, that definite disadvantages can also arise[1 7] and that adequate facilities for post-partum observation and treatment of retained placenta if required, will have to be available if the future breeding life of the animal is to be maintained at a high level.

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SOME DISEASES OF DOMESTIC RABBITS ENCOUNTERED IN THE WESTERN CAPE

I.F. ZUMPT*

ABSTRACT: Zumpt I.F. **Some diseases of domestic rabbits encountered in the Western Province.** *Journal South African Veterinary Association* (1976) **47** No. 2, 117 - 122. Regional Veterinary Laboratory, Private Bag X5020, 7600 Stellenbosch.
The most common disease conditions encountered in domestic rabbits under intensive conditions in the Western Cape are discussed. Chlamydiosis is of paramount importance; it might be the limiting factor in the developing Rabbit Industry.

INTRODUCTION

When discussing any disease or ailment of domestic rabbits, it must be accepted that optimum standards of management, feeding, stockmanship and hygiene are of great importance. Unfortunately, very little is known about diseases affecting rabbits under intensification, and many different conflicting viewpoints on various aspects of rabbit production are encountered, often to the detriment of the animal in question. Very little will be said on the latter aspect except that removable, individual, single-tier cages should be used, which must be cleaned regularly and efficiently, that free water should only be used in exceptional cases within a rabbit house, and that all buildings must be rat- and mouse-proof. Newly-acquired and reintroduced animals should be quarantined for four to six weeks and should also be dewormed and treated against mange.

In 639 animals submitted for autopsy over a period of 3 years, a diagnosis of chlamydiosis was made 359, salmonellosis 39, pasteurellosis 30, mastitis 35, colibacillosis 71 and "stress-shock" syndrome 25 times. The remaining 80 cases fell into other, less common categories. It is often difficult to make a specific diagnosis, as most of these diseases may occur in association with one or more other conditions, and it is suggested to speak of disease "complexes".

A. BACTERIAL DISEASE CONDITIONS

Colibacillosis

Colibacillosis due to numerous strains (serotypes) of *Escherichia coli* occurs commonly in young rabbits and is characterised in the majority of cases by diarrhoea, dehydration and death. *E. coli* is also incriminated in a "stress-shock" syndrome where heavily-pregnant does abort, or where incomplete abortion is followed by death of the doe.

It seems that certain strains of *E. coli* increase in virulence during outbreaks whereas others produce levels of immunity within a herd. It has been well-substantiated that strains of *E. coli* develop multiple resistance to antibiotics and sulphonamides. Explosive outbreaks are common and generally related to overcrowding and poor management.

Clinical Findings: Acute coliform septicaemia causes rapid death within a few hours, often without diarrhoea. Affected newly-born rabbits are weak, cold and cynotic; usually the whole litter dies within several hours. Enteric colibacillosis has a slower onset and is characterised by watery to white yellow faeces with an offensive smell. The anal region is moist, with clumps of stained, matted hair. Dehydration progresses rapidly, and affected animals usually die within 48 hours. Both forms must be differentiated from chlamydiosis and coccidiosis, and both can occur concurrently with colibacillosis.

Lesions: Gross lesions are not usually noticed in the septicaemic form. Congestion of muscles and organs with diffuse petechiae of the mucosa of the small intestines are often seen, and the latter may be gas-filled and red in colour. In the enteric form a mucoid enteritis and dehydration are usually found.

Diagnosis: Dead and sick untreated animals should be submitted to a laboratory for examination. Specimens for bacteriological examinations are taken from the brain, lung, liver, kidney and one mesenteric lymph node. Isolations of one or more pathogenic strains of *E. coli* will confirm the diagnosis, especially if this includes an isolate from the brain.

Treatment: It must be remembered that strains of *E. coli* develop multiple resistance to drugs. The rate of excretion of most of these preparations is high in the new-born animal, thus treatment with tetracyclines must be at least at a level of 10 mg/kg for 3 to 4 days. Supportive treatment is also advisable.

Prevention and Control: Colibacillosis is a disease of intensification and its incidence and severity is directly proportional to population density. Optimum levels of management, feeding, housing, ventilation, etc. are important. Prophylactic medication may be of advantage but should always be regarded as an interim measure.

An experimental *E. coli* vaccine produced at the Veterinary Research Institute, Onderstepoort has been tested for a considerable time in the Western Cape, and very good results have been obtained. One dose is administerest to pregnant does 1 week after mating and a second dose 1 month later, followed by vaccinations at six-monthly intervals.

Salmonellosis

This is a disease which is characterised by an acute enteritis in young rabbits and abortion in pregnant does, usually with death of the affected animal. Out-

Paper delivered at the Biennial Veterinary Congress of the S.A.V.A., Durban, September 1975.
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breaks are sporadic, with a mortality rate as high as 80%.
The species of Salmonellae which have been incriminated are *S. typhimurium* and *S. bovis morbificans*. In each outbreak the infection was traced to rodents.

Clinical Findings: The outbreaks were acute in nature and affected all age groups. Very young rabbits died without showing any symptoms, weaners developed a severe diarrhoea and died within 24 hours, whereas pregnant does aborted and several died with incomplete abortion. Lesions are similar to those of colibacillosis.

Diagnosis: Sick and dead animals should be examined in a laboratory where routine bacteriological specimens are taken as for colibacillosis. The isolation of a salmonella will confirm the diagnosis.

Treatment: In a commercial rabbit unit parenteral administration of antibiotics is impractical under normal conditions. Very good results are obtained by medicating the drinking water with a tetracycline-neomycin preparation for 4 days.

Prevention and Control: In all local outbreaks the source of infection was traced to contamination of feedstuffs by rats and mice. It was found that these rodents entered rabbithouses through holes in the walls and used the automatic waterlines as pathways to the feed-hoppers. Rodents were eliminated by rat-proofing all buildings with fine wire mesh and by placing poison bait at strategic points. It is recommended that there should be no contact between rabbits and turkeys, poultry, pigs and calves.

Pasteurellosis
This is a contagious disease, common in rabbits, transmitted either by direct or indirect contact. Healthy carriers probably introduce and perpetuate the disease in a rabbitry. Under primitive conditions, with inadequate housing and especially faulty ventilation, this condition is very prevalent.

Clinical Findings and Lesions: "Snuffles" or "nasal catarrh" is an acute to chronic inflammation of the respiratory tract characterised by an acute fibrinopurulent pneumonia with a catarrhal to purulent nasal exudate. The fur on the inside of the front legs is usually matted and caked with dried dirty exudate which results from the rabbits pawing at their noses. Infected animals show repeated bouts of sneezing which is accentuated under humid, stuffy and overcrowded conditions. As the disease progresses, chronic changes occur in the turbinate bones, and breathing becomes laboured and audible.

Diagnosis: The diagnosis is confirmed by the isolation of *Pasteurella multocida*, but other organisms such as *Bordetella bronchiseptica* have also been isolated from the upper respiratory tract of such animals.

Treatment and Control: Treatment is invariably disappointing, and the use of an experimental vaccine is advocated as a preventative measure. Recent trials with a polivalent P. multocida vaccine have shown great promise. The elimination of chronic cases is of great importance.

Other Bacterial Conditions caused by *P. multocida* and/or *Staphylococcus aureus:*

Mastitis or "Blue Breasts":
In the Western Cape *S. aureus* has been isolated in pure culture from the majority of cases although *P. multocida* has also been found in some.

Clinical Findings and Lesions: One or more of the mammary glands become hot, reddened and swollen, tending to become cynotic later during the course of the infection. This type of mastitis may spread rapidly through a rabbitry and litters of affected does usually die of starvation; the doe may die within hours, or survive with chronic mammary gland changes.

Diagnosis: The isolation of the causative organism from one or more affected glands is advocated.

Treatment: Unless at a very early stage, treatment with various broad spectrum antibiotics is disappointing and not advisable.

Control: Affected does should always be regarded as carriers and be eliminated. Litters of such does should not be foster-mothered by other does as it has been found that such litters may spread the disease. It is very important to destroy all infected nesting material and wooden nesting boxes, and every precaution must be taken by handlers not to spread this disease to other litters. It appears that certain family lines seem to be predisposed to this condition; this should be kept in mind at selection time.
An experimental polivalent *S. aureus* vaccine is used in most units.

"Skew Head":
This chronic ear infection caused by *P. multocida* and/or *S. aureus* is usually preceded by an ear-mite infestation. It is found predominantly under unhygienic conditions and is also spread by carrier animals.

Clinical Findings and Lesions: Affected animals start by periodically scratching one or both ears with frequent head-shaking. This condition increases progressively, the head is held in a turned or skew position so that feeding becomes progressively more difficult and affected animals die of starvation.

Diagnosis: Bacteriological examination.

Treatment: Affected animals do not respond to treatment and should be destroyed.

Control: Prevent introduction of ear-mite infested animals. Vaccination with experimental *S. aureus* and *P. multocida* vaccines has reduced this condition in 12 rabbitries to under 1%.

Infections of the Female Genital Tract:
These conditions caused by *S. aureus* and *P. multocida* are common in some rabbitries and usually accompany a high incidence of mastitis.

Clinical Findings and Lesions: Does often show a purulent vaginal discharge and are lethargic, dull and toxic in appearance. Complete or incomplete abortions are common, and such animals invariably die.

Diagnosis: Clinical examination and bacteriological examination.

Treatment: Response to therapy is disappointing.

Control: Vaccination with experimental vaccines as above.

B. VIRAL AND CHLAMYDIAL CONDITIONS

Virtually nothing is known about viral diseases affecting our rabbit industry. Every effort must be made to prevent the introduction of infectious myxomatosis into South Africa. Other conditions such as shape fibroma, rabbit pox and papillomatosis have not been diagnosed in the Western Cape.

Chlamydiosis (Mucoid Enteritis -?-)

In the Western Cape, ovine enzootic abortion was first diagnosed during 1972. The first successful confirmation and isolation of chlamydia organisms from various mammals in this region was achieved only in January 1974. From October 1973 losses in weaner rabbits suddenly increased from under 1% to 22% by the end of that year. Several pathogenic strains of *E. coli, S. typhimurium* and *S. eppendorf* were isolated, but the majority of specimens yielded no bacterial growth. When in January a positive diagnosis of chlamydiosis was eventually made, some rabbitries had lost over 50% of their weaners. Sudden deaths of pregnant does, abortions and deaths of nestlings were also later found to be due to chlamydia organisms.

Clinical Findings: Chlamydiosis in weaners is a rapidly fatal disease of young rabbits characterised by emaciation, gritting of teeth, watery eyes, very loose watery faeces followed by death. These animals seem to have distended stomachs and, if handled, a sound like water splashing within a confined space is heard. There is no noticeable febrile reaction and affected rabbits may show a normal appetite up to the terminal stage. Stress factors such as weaning, overcrowding and marked fluctuations in ambient temperatures were found to precipitate severe mortalities.

In adult animals a gradual emaciation is characteristic of chlamydiosis. Loose faeces and an increased appetite are usually observed. Unless these animals are treated, death eventually follows.

Chlamydiae have often been isolated from cases of conjunctivitis.

In the majority of pregnant does, foetuses die and, unless the female aborts, she will die without showing any clinical signs. Some litters are, however, born prematurely, others are born dead or nestlings die within a few hours.

Lesions: Various stages of pneumonitis, cachexia and mucoid enteritis with extensive congestion are characteristic in adult animals. In young, this disease is differentiated from coccidiosis by the absence of significant hepatic or intestinal lesions; diffuse kidney lesions can be detected in the majority of cases. In most cases the mesenteric lymph nodes are grossly enlarged and the spleen is often atrophic. The stomach and upper portion of the intestines are usually filled with liquid ingesta, occasionally distended with gas, and may contain partially-digested food. Frequently the colon contains a large quantity of clear, viscid, mucoid material.

Does which fail to abort are very toxic and decompose rapidly. Characteristic findings in weaners, as well as adults, are a very distended stomach and colon.

Diagnosis: Impression smears from various organs are made and stained by the Gimenez method. Isolation of chlamydia in embryonated hen eggs helps to confirm the diagnosis.

Treatment: Numerous experiments were conducted to evaluate various methods of treatment, but no single preparation gave promising results. It became clear that, once a doe is infected, she remains a carrier and produces chlamydia-harbouring offspring in all future litters. These affected litters never lost their infection in spite of treatment, and with subsequent stress developed typical clinical signs. The present recommended treatment is the medication of drinking water with a tetracycline for 4 days.

Discussion: Chlamydiosis is certainly the most important disease affecting rabbits in the Western Cape and could cause the collapse of the developing rabbit industry in this area. Chlamydial organisms were isolated from every rabbitry visited in this region, often in association with other pathogens.

In an attempt to evaluate the use of a vaccine a number of young rabbits, from litters which had not exhibited any evidence of chlamydial infection, were inoculated with half the recommended dose of the current ovine vaccine containing live attenuated chlamydial organisms and produced by the Veterinary Research Institute, Onderstepoort. When these rabbits kindled, their offspring were healthy, as were their subsequent litters. When healthy weaners were mixed with affected and sick ones, the former remained healthy, which seems to indicate that contact infection is of minor importance. It was further observed that there is a higher mortality in successive litters produced by affected does and that survivors from such litters have a similar breeding history.

Prophylaxis: The present recommendations in a rabbitry are as follows:—

1. For 4 days before weaning, all rabbits receive tetracycline-neomycin medicated drinking water.
2. Affected cases in other age groups are dosed orally with a tetracycline-neomycin suspension. (A good stockman will save over 90% of affected cases with this method).
3. Avoid overcrowding.
4. Avoid humid, high temperatures by ventilation and insulation of buildings.
5. Sudden temperature fluctuations, such as a heatwave should be followed by a four-day water medication programme.
6. Vaccinate rabbits at 8 weeks of age with a half-dose of the ovine vaccine.

C. PROTOZOAL DISEASES

Coccidiosis

Coccidiosis was in the past — and under certain conditions is even today — the most common and devastating disease condition of rabbits. Recovered animals frequently become carriers and thus perpetuate the infection within a rabbitry.

There are two forms:— Hepatic coccidiosis, caused by *Eimeria stiedae* and intestinal coccidiosis, caused chiefly by *E. magna* and *E. perforans.*

Clinical Findings and Lesions: Hepatic coccidiosis may be symptomless or cause diarrhoea, anorexia and loss of weight, terminating in death. Typical lesions are small, greyish-white nodules or cysts found throughout the liver parenchyma.

Intestinal coccidiosis is less common, but is as difficult to eradicate as the hepatic form. Animals usually do not gain weight, are pot-bellied and loose stools are passed.

Diagnosis: Autopsies and pathological changes of the liver are characteristic, and the demonstration of oocysts from the bile ducts or intestinal scrapings confirm this diagnosis. Regular faeces examinations are essential in monitoring a rabbitry for coccidiosis.

Treatment: Sulphonamide preparations such as sulphaquinoxaline, sulphadimidine and sulphachloropyrasine are commonly used as water medication, and *Amprolium-medicated feed is used on a large scale as a preventative measure. Treatment should always be at a therapeutic level as abortions and death of young animals have been experienced.

Prevention and Control: Clean breeding stock is essential and treatment of all introduced animals is advisable. Wire cages with mesh floors should be cleaned thoroughly and treated with a blow-lamp at regular intervals.

D. INTERNAL PARASITES

Cestodes

"Bladderworms" are parasitic conditions very rarely encountered in the Western Cape. Two intermediate stages of dog tapeworms are described:— *Cysticercus pisiformis* and *Coenurus serialis.*

Clinical Symptoms: In moderate infestations very little except a general unthriftiness is noticed in young rabbits. With increasing numbers of cysts or "bladderworms", abdominal distensions become obvious. These cysts are easily recognised on autopsy as are fibrous migration tracts in the liver parenchyma. Cysts of *C. serialis* are located in the subcutaneous tissues.

Diagnosis: Pathological changes found at autopsy.

Treatment: none.

Control: Contamination of food and bedding with dog excreta must be avoided and no infested carcasses or cysts should be fed to canines.

Nematodes

The "Pinworm" *Passalurus ambiguus* has so far been the only roundworm of importance in this area.

Clinical Symptoms: Usually asymptomatic, but in severe infestations pruritis and an inflamed anal region will be noticed.

Diagnosis: In practice animals are examined early in the morning by gently expressing a faecal pellet from the anus, where adult pinworms can then be easily seen. Regular faeces examinations should be done as part of a routine monitor system.

Treatment: Two treatments with a piperazine preparation, with a 10-day interval, followed by a treatment every two months, are effective.

Control: Clean breeding stock and mesh-floored cages are essential.

E. EXTERNAL PARASITES

Ear Canker

Caused by the ear mite *Psoroptes cuniculi,* this is the most common parasitic condition in rabbits and can cause direct and indirect losses in intensive rabbit units.

Clinical Findings: It is characterised by head-shaking, ear-flapping and scratching of the ears with the hind paws.

Lesions: Considerable irritation and tissue damage results, coagulated serum forms a hard crust or scab which, in turn, irritates and causes renewed scratching. Affected animals lose weight, fail to reproduce, and secondary ear conditions with torticollis usually follow. The inner ear is damaged by this time and secondary infections may reach the brain.

Diagnosis: Clinical examination and identification of the mite.

Treatment and Control: The eradication of this condition is difficult and costly. All equipment must be treated with an efficient acaricide. Affected ears should be cleaned carefully with hydrogen peroxide or ether, after which various healing preparations are applied to the inner and outer ear. Commercially-available ear tick grease** or rotenone-containing preparations have been used with success; treatments should be repeated every 7 to 10 days for three successive periods. Chronic cases and those which do not respond to treatment should be culled to eliminate this source of infestation.

Mange

Sarcoptes scabiei and *Notoedres cati* are both incriminated in this disease condition.

Clinical Symptoms and Lesions: Rabbits scratch themselves almost continually, resulting in loss of hair on the chin, nose, head, base of ears and around the eyes. The affected skin is later covered by raw crusts, and secondary skin conditions are common. Animals lose weight rapidly and wither away.

Diagnosis: Deep skin scrapings are examined to identify the causative mite.

Treatment and Control: If diagnosed early, affected animals should be culled and all in-contact rabbits and equipment treated with an approved preparation. Diazinon*** has been used very successfully in this area.

* Amprol 20% W/W, MSD.
** Trikill Pfizer.
*** Dazzel 30% W/W K.O.P.

Table 1: **SUGGESTED VACCINATION PROGRAMME**

Age	Vaccine	Dose	Condition in common terminology
8 weeks (Selection)	Ovine Enzootic Abortion (Chlamydia)	0,5 ml.	"Mucoid Enteritis"?
12 weeks	Pasteurella multocida (Experimental)	) 1,0 ml. Young) 2,0 ml. Adult)	"Snuffles", "Cold", etc.
	Chlamydia (Booster)	0,5 ml.	
16 weeks (First mating)	P. multocida (Booster)	2,0 ml.	-ditto-
17 weeks	Staphylococcus aureus (Experimental)	2,0 ml.	"Blue Breasts", Mastitis, Abscesses.
	Escherichia coli (Experimental)	1,0 ml.	"Diarrhoea", "Nest-box" deaths.
21 weeks	S. aureus and E. coli (Boosters)	2,0 ml. 1,0 ml.	

All vaccinations should be followed with further booster doses every 6 months. Regular deworming with a piperazine compound is advisable and should be monitored for the presence of pinworms and other roundworms.

Table 2: **MANAGEMENT STANDARDS SCHEDULE***

ITEM	DOES (plus litters till weaning)	YOUNG (weaning till slaughter)
1. TEMPERATURE — Comfort zone	10°C — 20°C 50°F — 68°F	10°C — 20°C 50°F — 68°F
2. HUMIDITY	65 — 75%	65 — 75%
3. VENTILATION RATES Optimum (summer) Minimum (extreme winter)	 8 m^3/kg live weight/hr 2 m^3/kg live weight/hr	 8 m^3/kg live wt/hr 2 m^3/kg live wt./hr
4. AIR SPEEDS Maximum Optimum	 0,5 m/sec 0,2 m/sec	 0,5 m/sec 0,2 m/sec
5. MINIMUM volume per animal housed	2 m^3/doe	2 m^3/per 10 to 12
6. FLOOR AREA required per cage (83 cm x 60 cm) single deck.	0,8 m^2	0,8 m^2
7. MAXIMUM NUMBER OF ANIMALS per cage (83 cm x 60 cm)	1 doe plus litter	10
8. NUMBER OF DECKS (layers of cages)	1	1 — recommended 2 — feasible
9. FEEDING DEVICE	1 HOPPER per cage	1 HOPPER per cage
10. WATERING	1 NIPPLE per cage	1 NIPPLE per cage
11 LIGHTING	1-75 watt by 8 foot fluorescent tube per 40 m^2 floor area.	1-40 watt by 4 foot fluorescent tube per 40 m^2 floor area.
12. LIGHTING DURATION	16 hours/day	12 hours/day

*As advocated by Coney Protein (Pty) Ltd. 1975

F. OTHER CONDITIONS

Aflatoxicosis caused by metabolites of fungal origin affects rabbits sporadically. Animals fail to grow or gain weight and moderate to severe losses may be encountered at times. Haemorrhages and parenchymal cell necrosis in the mid-zonal areas of the liver lobules and scattered single cell necrosis are found in acutely-affected rabbits. In chronic cases, bile duct hyperplasia and mild nodular hyperplasia of the liver

are seen. Very little can be done other than the elimination of affected foodstuffs to prevent further pathological changes.

Nutrition muscular dystrophy: Caused by Vitamin E deficient feeds has been reported and diagnosed in the Western Cape from rabbits fed on one specific batch of food.

Stress-shock syndrome (Cardiomyopathy): The word "stress" includes any noxious internal or external factor. Conditions such as toxaemia, infection, heat, cold, anoxia, trauma and physical exhaustion, etc. result in an increased secretion and utilization of adreno-cortical steroids. If stress is protracted, exhaustion of the gland may occur. This is often found in high-producing, fast-growing and pregnant animals, but may affect animals of any age and condition. Overcrowding and high, humid temperatures are the main predisposing factors. Affected animals die suddenly from hours to days after an environmental crisis (e.g. heatwave). These animals show distress, rapid abdominal breathing and often nervous symptoms terminating in death. On autopsy pathological changes are indicative of heart failure. This condition is found predominantly in certain family-lines or strains and is thus important at the time of selection.

At this stage it is difficult to separate the above from Pregnancy Toxaemia as described in the literature.

"Sore Hocks": This condition which is commonly known as "Sore Hocks" normally only occurs in rabbits which are kept on wire floors. While the size and gauge of wire mesh used will play a part, it would seem that heredity may well play a role. It would seem as though certain strains of rabbits have a predisposition to the condition as a result of the size of their feet in relation to their body weight, or the shape of their feet or a combination of these two factors together with the density of the fur-covering of their feet.

In all cases the rabbit farmer is strongly advised to try to avoid the development of "Sore Hocks" since the condition is most difficult to remedy. Rabbits with "Sore Hocks" suffer a great deal of stress and consequently lose condition rapidly.

It is suggested that all breeding animals, males and particularly females, be inspected regularly for "Sore Hocks". At the first signs of loss of fur on the hocks and the formation of calluses, a suitable hock sheet, preferably made of aluminium, should be put into the cage of the animal in question.

ACKNOWLEDGEMENTS

The author wishes to thank Dr J.L. du Plessis of The Veterinary Institute, Onderstepoort and Dr J.F. Prescott of the School of Veterinary Medicine, Cambridge, for the assistance rendered by them.

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BOOK REVIEW **BOEKRESENSIE**

OPHTHALMIC PATHOLOGY OF ANIMALS

An Atlas and Reference Book

L.Z. SAUNDERS and L.F. RUBIN

S. Karger, Basel 1975. Pp XV + 258. 114 pl and 27 figs. Price: S.Fr 170 (approx US $ 68.00)

This work is another cornerstone in animal pathology and is particularly welcome because there have been comparatively few attempts to compile this particular aspect of veterinary science into one volume or chapter. It is indeed encouraging to see the increase in interest which has taken place during the last two decades in the field of veterinary pathology and which has been manifested by the publication of several excellent text books. This Atlas will join these on our bookshelves. The authors, one a pathologist and the other an ophthalmologist, are both veterinarians.

The authors describe and depict the clinical and morphologic aspects of many diseases affecting the eye of animals. Emphasis has been placed on domestic animals but several conditions affecting laboratory and wild animals are treated. In general, descriptions of each condition is restricted to one page with relevant photographs on the facing page.

The pathology of each part of the eye is given in sequence from the cornea to the retina and optic nerve and includes malformation, degenerations, inflammation and specific diseases and conditions. There are chapters on neoplasms, post mortem and histological techniques, and history of veterinary ophthalmology. The standard of photography is excellent.

From the viewpoint of the veterinarian in Africa perhaps two diseases are conspicuous by their absence. One is gedoelstiasis (or specific oculo-vascular myiasis (uitpeuloog)) and the other infectious keratoconjunctivitis. The authors, however, make no bones about the fact that they have not attempted to include all ophthalmic conditions.

This book can be recommended without hesitation.

R C T

Jl. S. Afr. vet. Ass. 47(2) 123-124 1976

AN OUTBREAK OF DIPHTHERIA IN BOERGOAT KIDS

E.M. VAN TONDER*, G.E. KELLERMAN* AND T.F.W. BOLTON*

ABSTRACT: van Tonder E.M.; Kellerman G.E.; Bolton T.F.W. **An Outbreak of diphtheria in Boergoat kids.** *Journal South African Veterinary Association* (1947) **47** No. 2, 123 - 124 (En) Reg. Vet. Lab., P. Bag X258, 5900 Middelburg, Rep. of South Africa.

During an outbreak of diphtheria or ulcerative stomatitis in young Boergoat kids *Fusobacterium necrophorum* was consistently isolated under anaerobic cultural conditions from material taken from the edges of the ulcerative lesions from all the cases examined. *Corynebacterium pyogenes, Staphylococcus aureus, Actinobacillus lignieresi, Moraxella* spp. and *Lactobacillus* spp. were also isolated anaerobically and/or aerobically.

The typical symptoms and lesions were confined to the mouth, tongue and throat regions.

INTRODUCTION

The wide variety of disease conditions associated with *F. necrophorum* infection of farm and other animals is comprehensively listed[1,2,4]. Although these authors mention ulcerative stomatitis in goats as one of these conditions, no specific reports on outbreaks of this particular infection in goats could be found in the literature.

This report describes an outbreak of ulcerative stomatitis in Boergoat kids in the Graaf Reinet district, with a brief description of the symptomatology, post-mortal and bacteriological findings.

HISTORY

In this outbreak 150 Boergoat kids, 4 - 6 weeks old, were involved. The first report claimed that six had already died and another 14 were showing symptoms of the infection. Five days later another eight were noticed ill; thus in all 28 kids (18,6%) were affected. Altogether five of these kids were examined clinically, of which two were also sacrificed for a full post-mortal examination. Bacteriological specimens were taken from the lesions of all five of these animals.

MATERIALS AND METHODS

Bacteriological specimens were taken by means of sterile swabs, close to the edges of the lesions and after removal of as much of the necrotic tissue as possible.

In order to determine the variety of bacteria present in these lesions, 5 per cent horse blood tryptose agar plates were used for primary isolation rather than the recommended selective media[3,6]. Duplicate plates were inoculated. One of each pair was incubated aerobically and the other anaerobically** for 48 to 72 hours.

Smears made from the infected material were stained with Preston & Morrels[8] modification of the Gram Stain and the duplicate smears with a 1:10 dilution of Carbol fuchsin.

RESULTS

The most important symptoms included moderate fever reactions, salivation, frothing at the mouth, muco-purulent nasal discharge, constant sucking, chewing and swallowing movements, loss of appetite, rapid loss of condition and rough staring coats.

At autopsy the lesions were confined to the buccal cavity, tongue and throat regions of both cases examined. Typical, well circumscribed necrotic ulcers up to 4 cm long and 2,5 cm in width, were found in both animals on either side of the base of the tongue. A few scattered ulcers of variable size were also seen in the mucosa of the left upper cheek and soft palate of the one goat and the pharyngeal mucosa of the other. No macroscopic lesions could be found in the upper respiratory tract or livers of these animals, which apart from emaciation and depletion of fat reserves revealed no other significant pathological lesions.

Fig. 1: Necrotic ulcer on side of base of tongue.

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** Gaspak, Baltimore Biological Laboratory, Division of Bioquest, Cockeysville, Md.

On smear examination a mixed bacterial population was found including gram-positive cocci and cocco-bacilli or small gram-negative cocco-bacillary forms. The predominant feature in all the smears examined was the presence of an abundance of long gram-negative rods and wavy filaments lying parallel to each other. These rods and notably the filaments varied in thickness and tended to stain unevenly. Bulging and club shaped forms were readily observed. Some filaments were severely curved and twisted. Typical beaded filamentous forms were clearly discernible in smears stained with carbol fuchsin. The bacteria isolated are listed in Table 1.

Table 1: **BACTERIOLOGICAL ISOLATIONS FROM THE ULCERATIVE LESION OF FIVE BOERGOAT KIDS**

Case No.	Isolate	
	Anaerobic	Aerobic
639/1/1	F. necrophorum*	—
	S. aureus	S. aureus
	—	Moraxella spp.
649/1/1	F. necrophorum	—
	A. lignieresi	A. lignieresi
649/2/1	F. necrophorum	—
649/3/1	F. necrophorum	—
	C. pyogenes	C. pyogenes
649/4/1	F. necrophorum	—
	Lactobacillus spp.	—

* Isolate lost on sub-culture. Classified on colonial appearance and microscopic morphology in smear of infected material.

Our isolates conformed very closely to the characteristics of a reference strain obtained from Dr C. Cameron, Veterinary Research Institute, Onderstepoort. The only differences were that none of the isolates showed any sugar reactions, while the reference strain produced slight acid formation in glucose, fructose and trehalose.

From the results of antibiotic sensitivity tests, Chloramphenicol was selected and prescribed as parenteral treatment twice daily for 5 days.

After the administration of the antibiotic all the remaining affected kids showed a dramatic improvement from the third day onwards, they started eating on the fourth to the fifth day and were fully recovered within 2 weeks.

Subsequent to this investigation several reports of similar outbreaks were received from the same district but on account of the delayed notification, these could not be confirmed bacteriologically. The description of the typical symptoms and lesions however, leaves little doubt that this particular infection was fairly widespread amongst Boergoat kids in this district and that it was experienced for at least the previous 2 years.

DISCUSSION

Although the disease condition called "foot-abscess" had been widely experienced in small-stock in these areas during the past 2 years, it is the first time that ulcerative stomatitis, especially in Boergoats to the extent of the present outbreak, has come to our notice. According to subsequent reports this condition would appear to be fairly widespread in this particular district and to have been present for at least the past 2 years. Apart from the reference to Jensen (1913)[1 2] who stated that *F. necrophorum* has also been demonstrated in ulcerative stomatitis in goats, no reports of actual cases or outbreaks could be found in the literature. The consistent demonstration of this organism during the present investigations appears to be of primary aetiological significance.

The relative ease with which *F. necrophorum* could be demonstrated in smears and cultures from infective material in this investigation was probably due to the acuteness of the lesions[1 2 6], the characteristic arrangement and beaded filaments[4 7], the typical colony formation[3] and the minimum exposure of cultures to the air[1 7]. The failure of the first isolate to grow on sub-culture was probably the result of over exposure to aerobic conditions.

On account of its limited invasive properties, the primary *F. necrophorum* infection in these cases probably followed upon some predisposing injury[2 4]. Normally kids of this age have only started to graze. It seems reasonable to suggest that on natural veld, containing thorny shrubs, stick-grass etc., the circumstances for the infliction of smaller injuries to the buccal and pharyngeal mucosa are indeed favourable.

On account of their inconsistent isolation the known pyogenic pathogens like *S. aureus, A. lignieresi* and *C. pyogenes* could possibly be regarded as secondary invaders superimposed on a primary *F. necrophorum* wound infection. The specific association of *F. necrophorum* and *C. pyogenes* in pyogenic lesions has been previously recorded[9 10] while it is also commonly associated with other aerobic bacteria[2] and a variety of anaerobic and facultatively anaerobic bacteria[6].

ACKNOWLEDGEMENTS

We wish to thank the Director of Veterinary Services for permission to publish this paper and Dr C.M. Cameron for supplying a reference strain of *F. necrophorum*.

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Jl. S. Afr. vet. Ass. 47(2) 125-128 1976

STEROÏEDHORMONE IN KLEINDIERPRAKTYK

P.H. LE ROUX*

ABSTRACT: le Roux, P.H. **Steroïedhormone in kleindierpraktyk — Steroid hormones in small animal practice.** *Journal South African Veterinary Association* (1976) **47** No. 2, 125 - 128 (Afr, En) Hermitage Terrace, 2193 Richmond, Johannesburg, Republic of South Africa.

The nature, origin, influence and interrelationships of the steroid hormones are reviewed. Clinical application and the treatment of feline and canine patients with these substances is discussed, with special reference to particular conditions which occur in male and female animals. The therapeutic use of corticosteroids in small animal practice are considered, and some general observations are made concerning precautions and side effects.

INLEIDING

Die steroïedhormone is 'n nouverwante groep chemiese stowwe wat afgeskei word deur die byniers-kors, sekere selle in die gonades asook deur die plasenta. Almal ontstaan van kolesterol en word saamgestel langs dieselfde reaksies met 'n paar gespesialiseerde stappe teen die einde om elk te differensieer. Almal kom voor in vroulike en manlike individue en toon groot skommeling vanweë lopende aanpassing wat die individu maak. Behalwe aldosteroon wat streng gemoeid is met natrium-metabolisme en gevolglik bloedvolume, en die van die plasenta wat autonoom is, word die afskei van steroïedhormone beinvloed deur drie groepe verwante hormone van die hipofese nl. FSH, LH en ACTH. Die hipofese op sy beurt is onderhewig aan komplekse invloede van sirkulerende steroïede en stimuli vanaf die hipotalamus en limbiese stelsel van die brein. In herkouers, veral skape en bokke het die jaargety, weiveld, afstande wat geloop word om te wei en te drink, daglengte en tropgedrag almal 'n invloed om die geslagsiklus te sinchroniseer sodat die kalwers en lammers gebore kan word wanneer sagte weiding volop is. In huisdiere soos die kat en veral die hond is daar nie so 'n jaargetygebonde sinchronisasie nie. Die katwyfie is gedurig aan die gang, soms net meer so; die teef het 'n langer ritme met 'n sterker corpus luteum werking en die optree van skyndrägtigheid as 'n normale verskynsel. Wyfies wat geterroriseer word deur 'n dominante een toon min of geen estrus. As een in 'n groep loops word, val die ander gewoonlik ook in.

Die hipofese, adrenaal en gonades rangskik reeds al in die fetus hulle onderskeie invloede in balans. Hierdeur vind manlike/vroulike differensiasies plaas in ooreenstemming met die XX of XY asook ander chromosoominformasie vasgelê in die saamgesmelte ovum en sperm. Met geboorte en daarna word die hipotalamus en limbiese stelsels gekondisioneer deur testosteroon en estrogeen vanaf gonades om spesifiek manlik en vroulik te reageer op die snelle styging van die hormone met puberteit en gedurende volwassenheid. Nadat die geslagstelsels volwaardig ontwikkel het is die informasie van reuk, gesig, gevoel en waarneem van gedragspatrone die sneller wat lei tot paring.

Die bynierskorshormone is lewensbelangrik vir die individu. Hulle is 'n groep wat bestaan uit dieselfde estrogene/androgene van die gonades plus sg. kortikosteroïede. Die lg bestaan uit baie (50) tussenvorms maar hoofsaaklik aldosteroon, kortikosteroon en kortisol wat die hele spektrum vanaf suiwer Na-metebolisme tot koolhidraatmetabolisme verteenwoordig. Om die een steroïdhormoon se funksie van die ander s'n af te skei is egter dwaas. Ons praat van geslagshormone en kortikosteroïed asof dit twee wêrelde behels dog dit ly geen twyfel dat 'n bul se metabolisme heel anders moet wees as die van 'n koei. Deur slegs die testes te verwyder verander sy voorkoms na iets tussen die van 'n manlike en vroulike dier. Geeneen van ons het enige probleem om bloot deur waarneming te onderskei tussen 'n teef en 'n gesteriliseerde teef nie. In vroulike atlete wat anaboliese steroïede gebruik om hul prestasie te verbeter is vermanliking soms opvallend, omdat die steroïed na verwant is aan testosteroon. Daar is 'n gedurige aanvraag vanaf die selle van die liggaamsorgane vir steroïedhormone. As die gonade verwyder word moet die skors *almal* voorsien — ten koste van die wat eksklusief vanaf die skors moet kom. Daar is dus 'n duidelike wisselwerking binne die groep hormone. Met die algemene oorsig van steroïedhormoon-tussenwerking sal ek volstaan en sonderverdere betoog oorgaan na die eintlike onderwerp nl die gebruik van dié hormone in kleindierpraktyk.

Watter pasient het steroïed toediening nodig? Voor die hand liggend sal dit wees die diere waar die hormoon ontbreek. Dan ook dié waar antagonistiese werking nodig is en ook dié waar die afskeiding van endogene hormone aangehelp moet word. Die geslagshormone se primêre funksie is om die geslagsstelsel te beinvloed sodat die dier kan voortplant.

DIE VROULIKE DIER

Anestrus kan te wyte wees aan die lae produksie van estradiol (E2), of neurose, of van 'n verhoogde vlak van progesteroon a.g.v. eierstoksiste. Etiniel-estradiol (Ee) teen 'n lae dosismaat vir lang periodes sal die hipotalamus prikkel om die hipofese te laat FSH afskei waarna die estradiol van die eierstokke die proses sal aanvuur. Honde van gemiddelde grootte sal 'n dosis van 0,05 mg Ee per os elke dag of al om die ander dag nodig hê vir 'n tydperk van 3 tot 6 weke. Indien estrus nie binne 21 dae begin nie kan 'n hoër dosis probeer word. Sodra estrus duidelik begin het word behandeling gestaak.

Ongewensde dragtigheid kan afgeweer word deur Ee teen 1 mg/20 kilo intramuskulêr toe te dien. Of 'n

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Voorgedra tydens die Jaarvergadering van die Tak O V S van die S A V V, Augustus 1975.

herhaalde dekking gedurende die verlengde estrus vrugbaar sal wees is te betwyfel dog dis nie 'n uitgemaakte saak nie. Daar kan weinig skade aangerig word met estradiol-toediening vir hierdie doel.

Abnormaal-lange estrus kom soos voor en mag dui op sistiese follikels met of sonder luteïnisasie. Progesteroon of progestageen is moontlik aan te beveel dog hou gevaar in van daaropvolgende piometra. In tewe kom sistiese eierstokke egter baie dikwels voor sonder piometra en piometra sonder siste. Moontlik is dit beter om die teef intensief te teel en as die toestand herhaaldelik voorkom, veral met gereëlde skyndragtigheid oöforektomie te doen.

Die reëling van estrus met metielprogesteroon en verwante middels moet beplan word deur 'n baie goeie geskiedenis van vorige siklusse asook die ondersoek van skedevog smere gekleur met Schorr se metode. Selfs met goeie beplanning kan langdurige onderdrukking van die geslagsiklus die gevolg wees of kan die siklus tog voorkom ten spyte van voorbehoedende behandeling. Massiewe lukraakdosering met progestageen veral in vroeë stadium van estrus skep ideale toestande vir piometra om te ontstaan — hoë E2 gevolg deur hoë progesteroon. Die edemateuse baarmoederwand ondergaan verdere hiperplasie van die slymvlies en die spiere word atonies agv progesteroon. Die cervix sluit en 'n mucocoele ontwikkel. Bakterië van lae virulensie wat in die baarmoeder aanwesig is vermenigvuldig en lei tot 'n subakute metritis. Die etter veroorsaak leukositose, beenmurguitputting en anemie. Diagnose is gewoonlik eers moontlik wanneer die uitloopsel uit die vagina vloei. In ouer tewe is swak kondisie, polidipsie en poliurie algemene simptome. Histerektomie is die enigste behandeling.

Hipertrofie van vagina ("prolapsus"). Tewe van alle rasse maar veral van sekere Bull Mastiff en Boxer families toon hierdie afwyking. Progesteroon het geen effek nie en die toestand herhaal met volgende estrus. Oöforektomie is die enigste behandeling, hoewel in sommige gevalle verwydering van die prolapsus daaropvolgende dekking moontlik sal maak.

Metritis. Die genesing van post partum metritis word dikwels verhaas deur saam met geskikte antibiotika en baarmoedersaamtrekmiddels ook 'n lae dosis Ee te gee. Dit verhoog die kontraktiliteit van die miometra en stoot die etter uit.

Aborsie. In katte is aborsie op 4 - 6 weke algemeen, in honde in die laaste 14 dae. Aborsie in 'n hond vind plaas gewoonlik na die foetusse gesterf het agv galkoors, *Brucella canis*-besmetting en toxoplasmose. Toegediende kortikosteroïde swaai die foetale hipotalamus — hipofese- adrenaal axis na die van parturisie. Verlies van CL as gevolg van lae plasentale LH kan 'n rol speel. Aborsie is so skielik dat progesteroon nie effektief kan wees nie, moontlik wel as dit saam met berustings-middels en rus gebruik word. Die wisseling van die plasenta, foetus en moeder se hormone is so kompleks dat aborsie nie sal afhang van 'n enkele hormoon gebrek of oormaat nie.

Terapeutiese oöforektomie verminder nie soseer die rustende estradiolvlakke nie, maar die pieke soos gesien gedurende vroeë estrus en dragtigheid is afwesig. Die progesteroonvlak is dan ook stabiel op 'n lae vlak. As gevolg daarvan ondervind hiperplasie van endometrium (mucocoele) regressie; hipertrofie van vagina (Boxer en B.M. tipes) bedaar; die ontwikkeling van gewasse van die melkklier word gerem en herstel kan intree sonder verdere behandeling. Herhaaldelike skyndragtigheid is so 'n algemene voorloper van piometra dat dit ook 'n indikasie is vir oöforektomie.

Hipogonadisme van gesteriliseerde tewe is 'n sindroom wat sigbare tekens het in omtrent 90% gevalle waar die ovaria verwyder is. Daar is reseptore vir estradiol en progesteroon in baie liggaamsweefsels sodat daar groot druk op die bynierskors is om hierdie produk te lewer, liewer as kortikosteroïede waarvoor daar net periodiek groot aanvraag is. Die estradiol- en progesteroonvlakke is die van 'n normale teef met rustende ovaria. Tans is daar geen vaste verklaring vir die velatrofie, vetsug en spieratrofie van gesteriliseerde tewe nie. Die laer peil van E2 of afwesigheid van sesmaandelikse progesteroon- en estradiolpieke is moontlik direk verantwoordelik vir die swak haarkleed. Met hoë estriol (dragtigheid) styg die hormoondraers TBG, TBPA en CBG en gevolglik totale steroïed en tiroksien van bloed. Of lae E2-vlakke 'n daling ter gevolg kan hê is onbekend. Die feit is dat volgehoue toediening van E2 per os vir bv 'n Cocker Spaniel teen 0,15 mg per dag 'n bloedvlak van 210 pg/ml sal gee en na sowat 8 weke gewigsverlies, beter haarkleed en wakkerder gedrag ten gevolg het. Indien progestageen saam met E2 toegedien word skyn 'n vinniger verbetering plaas te vind. 'n Meer seldsame simptoom van oöforektomie-hipogonadisme is degeneratiewe veranderings in die kniegewrig sonder verhoogde uriensuur. Die simptome is mankheid, pyn en fibrose van kapsel. Met volgehoue E2 toediening herstel dit heeltemal. Daar bestaan 'n vermoede dat diabetes meer dikwels voorkom in gesteriliseerde tewe as in tewe of reunhonde. Die redes waarom dit die geval sou wees is onbekend en die aangeleentheid sal verder ondersoek moet word. Druk-lekkasie van uriene by gesteriliseerde tewe kom voor omdat die geslagsdele 'n spesifieke teikenorgaan vir E2 is. Atrofie van die vagina en vulva verkort die uretra. As die blaas vul, lek die uriene uit. E2 behandeling per os op 'n vlak hoog genoeg om effense vergroting van die vulva te veroorsaak gee uitstekende resultate. Dermatitis om die vulva gaan soms gepaard met druklekkasie van uriene. As genoeg E2 gegee word om die teruggesonke vulva uit die velplooi te stoot herstel die toestand dramaties. In ge-oöforektomiseerde katte is die velatrofie met pruritis en alopekie prominent en vetsug minder so. Ook hier is E2 en metielprogesteroon (MP) teen dosis van E2 0,025 mg 2 keer weekliks en MP 2,5 mg 2 keer weekliks suksesvol.

DIE MANLIKE DIER

Testosteroon (T) is die hoof produk van die Leydigselle, en estradiol vanaf die Sertoliselle. Lae testosteroon lei blykbaar tot FSH stimulasie wat dan in die Sertoliselle beide 'n reseptor vir T vorm en E2 laat afskei. LH verhoog T produksie en die T hou spermatogenese en spermiogenese, dmv die voorbereide Sertoliselle aan die gang. Lae T-vlakke sal die E2-vlak relatief verhoog en vervrouliking kan voorkom met verlies van libido. Ongevoeligheid van die hipothalamus vir T lei tot impotensie. Dit is omkeerbaar deur T toe te dien en sodra die dier libido toon hom te gebruik vir etlike parings. Hierdeur word die brein gekondisioneer en die neurose verdwyn. Sertoliselgewas lei tot autonome produksie van E2 en vervrouliking. In kriptorche testes sterf die Leydigselle af sodat slegs E2 gevorm word en kan dit lei tot 'n soortgelyke prent. Die simmetriese haarverlies by hierdie sindroom is

moontlik vanweë die oormaat E2 en lae T skynbaar het die vel, veral die follikel en vetkliere beide nodig in die regte verhouding tot die aangebore vroulike of manlike genetiese eienskappe. Die lokus van optrede van alle steroïed hormone is via 'n reseptor in die selsitoplasma en vandaar direk in die kern. Deur die DNA materiaal te beinvloed word die sintese van sekere stowwe in die sitoplasma bewerkstellig. By gekastreerde mannetjieskatte is pruritis en alopekie prominent asook vetsug. Toediening van T elke 14 dae herstel gou die letsels dog oordosering het katergedrag tot gevolg. Metielprogesteroon teen 5 mg 2 keer per week per os werk eintlik beter en vertoon nie hierdie nadeel nie. Die behandeling kan tuis toegepas word vir kort periodes om die veltoestand te beheer. Te hoë T en oordrewe reaksie van teikenorgane veroorsaak in reunhonde perianale adenoom, seksbeheptheid en 'n vergrootte prostaat. Kastrasie verlaag die T-vlak permanent dog dit sal moontlik later kastrasie-hipogonadisme veroorsaak. Voorkeur word gegee aan dosering met E2 teen 'n dosis van 1 mg per 30 kg i.m. maandeliks of meer dikwels. Aangesien die prostaat hierop kan reageer met atrofie of metaplasie word delmadinone ("Tardak" ½ - 1 ml per 5 kg) by prostaatpatologie verkies. E2 en delmadinone kompeteer met T in die reseptore in die teikenselle en indien die vlakke hoog genoeg is, sal weefsel dan nie kan reageer op die T teenwoordig nie. Sistvorming en infeksie soos gesien in verwaarloosde prostaat gevalle, lei tot 'n stadiger herstel sodat antibakteriese- en hormoonterapie oor maande toegepas moet word. Daar kan periodieke aanmanings verwag word.

DIE BYNIERSKORSHORMONE

Steroïedsteurings van die bynierskors word tans selde gediagnoseer. Ware hipokortikosteroïedisme ("Addison's disease") is seker raar. Indien so 'n geval teengekom word is kortikosteroïede met of sonder ACTH die natuurlike keuse van behandeling. Hiperkortikosteroïedisme ("Cushing's disease" en sindroom) is moontlik meer algemeen, dog weereens is bloedanalise en funksietoetse nodig vir so 'n diagnose. Die kliniese beeld alleen is nie spesifiek genoeg nie. Sommige pasiënte in veeartspraktyk ly egter afwisselend aan beide hierdie sindrome agv die misbruik van kortikosteroïede. Daar is na my mening min plek vir sistemiese kortikosteroïedterapie in kleindiersiektes. Vir elk van die gewaande indikasies van hierdie middel is daar etlike meer-effektiewe en meerspesifieke behandelings. Die aanwysings berus amper almal op drogredenasies afgelei van die fisiologiese rol van endogene kortikosteroïede. Die vlak van kortisol in honde is normaalweg omtrent 1,0 tot 20 ug/100 ml en toon die hoër vlak gedurende daaglikse "stress" en die laere gedurende rusperiodes. Met terapeutise dosisse van sintetiese kortisol so hoog as tot 500 ug/100 ml is die fisiologiese uitwerking nie ter sprake nie. Die groot nut van dié middels by orgaanoorplantings behoort aanduiding te wees van die wyse waarop die liggaam se immunologiese stelsel daardeur uitgeskakel word indien volgehoue dosering plaasvind. Waar diere vir lang tye met kortikosteroïede behandel is, gewoonlik vir velkwale toon hulle swaarlywigheid en verhoogde vatbaarheid vir besmetting van slyt areas van die vel. Gebrekkige bestandheid teen siektes waarteen wel ingeënt, kom voor. Inentings gedurende die periode van behandeling is glad nie effektief nie. Hoe jonger die dier, des te erger is die "skade" aan die timusweefsel en die verwante "permanente" sellulêre imuunstelsel. Die plaaslike aanwending van kortikosteroïede is nuttig maar met sekere voorbehoude. Die inflamatoriese reaksie wat dikwels die waarneembare teken is van die siekte is 'n baie belangrike fase in die herstel van die siekteletsel. Om dit te onderdruk net om dinge beter te laat lyk is verkeerd. Indringing van die letsel in omliggende weefsel kan daarop volg, veral wanneer in die geval van besmetting, antibiotiese behandeling opgehef word sodra die sigbare ontsteking verdwyn.

Ons gebruik wel kortikosteroïede, maar alleenlik weëns die gebrek aan 'n meer geskikte behandeling vir die volgende toestande.

Oogsiektes

Uveitis of Iridosiklitis reageer goed op subkonjunktivale inspuitings van 'n kortikosteroïed soos "Depomedrol", asook druppels ("Maxidex).

Pigmenteerde keratitis (sogenaamd) van Alsatians se verloop kan vertraag word deur soortgelyke behandeling met of sonder antibiotika plaaslik aangewend.

Kroniese keratitis volg gewoonlik op langdurige trauma of hardnekkige infeksie en moet nie sommerso met kortikosteroïed behandel word nie. Dit kan wees dat die organismes betrokke bestand is teen die antibiotikum in die mengsel aangewend en agv die steroïede in staat is om deur die ontstekings-versperring te dring met ulserasie as gevolg.

Virusinfeksie van die konjunktiva soos in pneumonitis van katte moet soms met antibiotikum- kortikosteroïedmengsels behandel word.

Buiteoorontsteking. Wanneer kortikosteroïed gebruik word behoort 'n swamdoder asook 'n antiboitikum geinkorporeer te wees en moet sistemiese antibiotika toegedien word, om die ontstaan van binneoorverswering te voorkom.

Dermatitis

Okklusiewe verbande met betametasoon-bevattende room is feitlik al wat help in die lek-granuloom wat soms op groot soort honde se bene voorkom. Dieselfde behandeling is nuttig vir die soortgelyke "neurodermatitis" by katte. Sistemiese toediening van kortisoon, vir algemene veljeuking en so meer, is 'n kortpad wat, na my mening, 'n doodloopstraat is. Die oorsaak kan gewoonlik bepaal en reggestel word met spontane permanente herstel van die pasiënt as gevolg. Daarby is dit bloot menslik dat as kortikosteroïed ingespuit is en die klagte van "die hond krap hom dood . . ." het stil geword, die praktisyn nie verder pogings gaan aanwend om die oorsaak te bepaal nie.

ALGEMEEN

Die gewaande goeie uitwerking van kortikosteroïed in skok en "stress" kan slegs van korte duur wees, gevolg deur onderdrukking van die pasiënt se bynierskors. Indien dit gebruik word sonder beredeneerde aanwending van vloeistofterapie, bloedoortapping, gepaste hulp aan respirasie en nierfunksie, chirurgie en so meer sal dit meer kwaad as goed doen. As die dinge wel gedoen word om die pasiënt te help, dink ek sal kortikosteroïed in elk geval oorbodig wees. Ek weet dat u moontlik sal sê: "Maar my ondervinding is steeds dat die diere dramaties reageer op kortisoon . . .". Glo my as ek u verseker dat u resultate net so goed sal wees daarsonder maar dan moet die pasiënt op 'n

fisiologiese wyse ondersteun word met die genoemde bystandsbehandeling.

Dragtige tewe behoort geen kortisoon te kry nie. Dit is teratogeen in die vroeë stadia van dragtigheid sodat gesplete verhemelte, ledemaat- en skedeldefekte veroorsaak kan word. Laat in dragtigheid kan dit moontlik miskraam veroorsaak.

Net soos die uitwerking van die verskillende steroïedhormone oorvleuel, en die relatiewe konsentrasie van belang is, moet ons nie vergeet dat hulle werking ook grens aan dié van die ander hormone nie. Dit sal dus in die belang van ons pasiënt wees as ons altyd 'n oomblik goed nadink voordat ons op hormoonbehandeling besluit. Hou in gedagte wat die fisiologiese rol is van die middel en wat 'n fisiologiese dosis sal wees. Waar kompetisie die basis van behandeling is, moet newe-effekte in ag geneem word. Waar farmakologiese, liewer as fisiologiese dosering nodig word, moet ons begin dink aan alternatiewe behandelings wat dieselfde effek sal hê en minder skadelik sal wees. Aangesien aanpassings stadig geskied is 'n matige dosis oor 'n langer tydperk beter as 'n massiewe dosis van tyd tot tyd. Ten spyte van vooruitgang gemaak op die gebied van die funksie van hormone, duik daar vir elke stap vorentoe nuwe vrae op wat weer wag op 'n antwoord. Deur noukeurige waarneming en objektiewe beskouing van resultate verkry met hormoonbehandeling kan die veearts 'n waardevolle bydrae maak tot die wêreld se totale kennis van steroïedhormone.

Jl. S. Afr. vet. Ass. 47(2) 129-132 1976

REVIEW **OORSIG**

A REVIEW OF MINERAL IMBALANCES OF GRAZING ANIMALS IN SOUTHERN AFRICA

P.A. BOYAZOGLU*

ABSTRACT: Boyazoglu, P.A. **A review of mineral imbalances of grazing animals in Southern Africa.** *Journal South African Veterinary Association* (1976) **47** No. 2, 129 - 132 (En). Box 21005, 0137 Valhalla, South Africa.

A historical review of mineral imbalances recognised in Southern Africa is given. The survey procedures which were followed are described both in terms of sample sources, as well as the analytical procedures with emphasis on the problems encountered at each stage of the developments. A concise summary is given of the mineral patterns known to date according to the ten regions currently recognised where specific patterns of mineral imbalances are known to occur.

HISTORICAL

The significance of mineral deficiencies in animal feeding in the Union of South Africa was first recorded in 1912[14]. In this report reference was made to the observations of an 1805 commission describing "lamziekte" in cattle. The condition affected principally the loins and the hindquarters, and increased in incidence during drought periods. It was noted that animals were affected as far north as Bechuanaland, and east to the Eastern Cape. A close correlation was recognised between "stijfziekte", "Lamziekte" and the chewing of bones by all classes of stock, particularly in growing animals, and pregnant lactating cows. Dr Hutcheson (Colonial Veterinary Surgeon) conducted an experiment in which cattle fed bone meal did not contract either disease. A correlation was also noted between disease incidence and low soil phosphate content[14].

Attention was later given to calcium levels[8] as a possible limiting factor in sheep nutrition, with negative results. The first reference to the possible significance of a trace element deficiency, in Southern Africa, was noted in 1932, as quoted by Malan, du Toit & Groenewald[11]. In this work the iodine requirements of sheep were investigated and detrimental effects were recorded as a result of the addition of the element.

PLANT SURVEY

A comprehensive survey of the macro-element content of plants in Southern Africa was commenced in 1930, to define the areas deficient in phosphorus[7]. Simultaneously plants were analysed for calcium, magnesium, potassium, sodium and chlorine content, as well as for crude protein, crude fibre and soluble ash. Nearly 5 000 samples were collected by 240 stock inspectors between November 1933 and October 1935.

Initially soil samples were analysed but later attention was focused exclusively on representative forage samples collected according to the observed grazing patterns of animals during each season. The main observations and conclusions of the report[7] are as follows:—

1. There was an obvious difference in mineral levels between grass and bush pastures. Grasses were particularly low in phosphorus, while bush yielded high calcium concentrations which frequently exceeded 1% on a dry basis. The calcium concentration of grasses on calciferous soils was a third of that found in bush from the same area. In these Karoo regions the magnesium content was found to be adequate for the prevention of hypomagnesaemia in producing animals[15]. In other areas the magnesium levels were found to be well below the minimum 0,1% considered to be necessary for normal requirements. Such low concentrations were found in extensive areas of Natal (Zululand, Lions River), the Transkei (Umtata), the Eastern Cape (Bedford), the Southern Orange Free State and as far north as Standerton. The significance of the magnesium deficiency was, however, not recognised, as the interaction effect produced symptoms of a calcium excess with deposits of calcium in the tunica media of the blood vessels.
2. The seasonal fluctuations in mineral content was studied in grasses. Concentrations of 0,12 - 0,17% phosphorus were measured in summer, which dropped to 0,05 - 0,07% in winter. By contrast Karoo bush maintained an even concentration which was considerably higher than that of grass from the same area, the difference being accentuated in the winter months.
3. There was a parallel seasonal variation in the protein content of grasses, which ranged from 7 - 9% in summer to 3,3 - 4% in winter. Karoo bush from the same area maintained protein concentrations of 7 - 10% throughout the year.
4. No indication was given of the plant population in the 18 regions studied. This obviously affects deductions, especially when semi-arid regions (e.g. North West Cape), are compared with those in the high rainfall areas (e.g. Natal).
5. A key comment was made by the authors who stated "it is clear that the extreme deficiency of protein in our grass pastures during winter, is a problem of equal, if not greater importance than that of phosphorus deficiency". This relationship, as well as the limiting effect of an inadequate digestible nutrient supply due to sparse plant population has at times been overlooked. The result has been that attempts to assess response to the addition of one of these nutrient requirements have been clouded by other limitations.

Trace mineral investigations on plant materials have been conducted in the winter rainfall area of the

Paper delivered at Orange Free State/Northern Cape Branch of the S A V A 21st Annual Congress 1975.

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Cape Province and adjoining regions since 1938[16], with particular reference to cobalt and copper deficiencies. In particular, the Humansdorp area was considered to be deficient in cobalt. Sporadic investigations also revealed iodine deficiencies in the Natal Midlands and an excess of selenium in parts of the Beaufort West District.

CLINICAL INVESTIGATIONS

Detailed studies on the copper requirements of animals were conducted in the Southern and Western Cape Coastal regions[13]. These areas were investigated as a result of lambs being found with the typical sway-back symptoms. The necropsy findings were destruction of the myelin in the nervous system, and, in extreme cases, symmetrical areas of softening or cavity formation in both the cerebral hemispheres[13]. Liver copper levels in both ewes and lambs were low, with correspondingly high iron concentrations. Copper administration to pregnant ewes prevented the development of symptoms and resulted in recovery in mildly affected lambs. Lambs with advanced signs of central nervous system damage did not recover.

Initial experiments using potassium iodide as a supplement in goitre areas were not encouraging[11]. Later investigations indicated that true iodine deficient areas did exist in the foothills of the Natal Drakensberg mountains but clinical cases were prominent only in young pigs. The goitre areas in the North of South West Africa are thought to be due to goitrogenic plants which precipitate the pathological changes. Other goitrogenic areas are the Hoërsee and Kleinrivier valleys[3], although these studies emphasise the effects on the local human population which practised peculiar eating habits.

Fluorine excess has been associated with deep boreholes containing high concentrations of the mineral in the Western Karoo region, and in Northern Transvaal[12]. In these areas cattle developed mottled and excessively worn teeth at two to three years of age, and an overall cachectic appearance even in the presence of abundant good grazing. A further complication in the Western Karoo region is that boreholes yield low volume, high mineral content waters, exceeding at times 10 000 parts per million of total solids. The minerals occurring in such high concentrations are primarily calcium and magnesium carbonates, chlorides and sulphates. Though these are tolerated by some mature stock, they are detrimental for breeding animals and their offspring.

Excesses of copper and selenium[6] have been located in the Beaufort West area, where high concentrations occur in pastures and in sheep livers, and where symptoms of hair fragility occur in horses. Mineral imbalances have been induced by the injudicious use of antagonists such as sulphur and molybdenum[4,5]. It is observed that farmers are also using licks containing up to 5% flowers of sulphur in unaffected areas, without the necessary compensatory addition of the related minerals, resulting in induced deficiencies of copper and selenium. Induced deficiencies have also been recorded in areas under irrigation such as Vaalhartz, where previously these deficiencies were not recorded. This has apparently been caused by a combination of intensive fertilisation patterns, irrigation and the increased yields which are taken off land previously marginally adequate in these minerals.

The deviations in mineral balances recorded thus far have been recognised by pathomnemonic symptoms such as sway-back and depigmentation (copper); degenerative myopathy, hair fragility (selenium); anaemia (copper, cobalt); goitre (iodine); mottling of the teeth (fluorine); and pica (phosphorus). However, such incriminating evidence is not always seen, and long term projects are needed to evaluate the effect of mineral deviations both qualitatively and quantitatively. In the Republic of South Africa, research work of well over thirty years' duration was summarised by Bisschop[2], who evaluated the responses by different breeds of cattle of varying ages and production and reproduction potentials, using various sources of phosphorus and methods of administration. Even though phosphorus was found to be at its lowest levels in the winter months, the greatest responses to supplementation were measured in summer. In summer the relative increase in the levels of all other nutrients was much higher, than that of phosphorus. The report concluded that two ounces (56 gram) of bonemeal per head per day was a satisfactory level of supplementation for post-weaning age non-productive cattle, and half this quantity could give parallel results in above average seasons. However, breeding stock required between three and five ounces of bonemeal (8,5 - 14,2 g phosphorus) per head per day.

In Rhodesia a series of observations and mass measurements were conducted by Ward[17] at the Makoholi experiment station near Fort Victoria, over a six year period. This work with Mashona cattle supported several of Bisschop's findings and also brought further facts to light. The supplementation of two ounces of bone meal daily yielded an average of more than 200 additional pounds (91 kilogram) of weaned calf production per cow over six years. The calves grew faster from birth, especially in the summer months, and weaning mass was increased by 20%. Furthermore there was improved fertility and calf viability. Taking these two factors into account there was an improvement of over 13% in favour of the bone meal treated group. Protein supplementation in the form of groundnut cake meal, during the winter months, had an additional beneficial effect on the bone meal groups. Where bone meal supplementation was continued up to feedlot feeding of the cattle and slaughter performance was used as a criterion, a financial gain of over 12% was measured in favour of bone meal supplementation.

The majority of mineral imbalances are in a category which, though affecting productivity, do not permit clinical diagnosis, as there are no specific symptoms involved. Only long-term biological experiments have been able to show the economic benefits of corrective supplementation as demonstrated in the Makoholi and Armoedsvlakte work. The phosphorus work especially required evaluations of production (growth) and reproduction which were evident when other limiting factors did not confound the results. In particular, chronic progressive changes are difficult to evaluate. It is not therefore, surprising that the abnormalities recognised by the author as resulting from magnesium deficiency, were initially incorrectly diagnosed in the field as inevitable ageing effects, particularly where commercial flock owners were not recording the ages of individual animals accurately.

The lesser minerals present a more complex picture

where marginal imbalances and interactions reduce productivity, and non-specific symptoms are presented. Furthermore the author recognised that internal parasites, as in the case of strongyles, increase the requirements for copper, and cloud the picture of the true mineral requirements in the diet.

The challenge of evaluating imbalances in a broad spectrum of minerals is certainly formidable, considering the number of biologically important minerals and their interactions. Furthermore, the method used to evaluate such patterns must be practical for individual farm investigations.

CHEMICAL DIAGNOSIS

The problems encountered with the analysis of soil samples[7] as well as those of selecting suitable plant material for analysis because of the selective grazing patterns of the animals, point to the animal itself as a more suitable sample source. This automatically eliminates the variables associated with the soil and plant material. An easy sample to acquire is blood, and it has been used extensively as a guide to the efficiency of phosphorus levels in diets[10]. This is possible where large numbers of animals are available so that data can be pooled. Where the trace minerals are present at concentrations of below one part per million (cobalt, copper and zinc), meaningful deductions are particularly difficult.

Hair samples have also received attention as they represent a more stable biological sample than blood, and have higher concentrations of several minerals (zinc 100 p.p.m.) than blood. It has been observed[12] however, that mineral concentrations in hair from single animals differ from site to site on the body.

The liver has received special attention as a sample source, because this organ is the body's metabolic centre, and most minerals are integral portions of metallo-enzymes which serve as catalysts for metabolic processes. The donor should not be affected by any other condition or disease. It should have grazed in a specific area ideally for 6 months or longer, so as to assure the measurement of the local situation, rather than a residual effect of prior influence. Animals younger than 6 months and foetuses, have been found by the author to be unreliable for this purpose. Whereas the biopsy technique has been used in special cases, it is not practical for general use. Samples from freshly slaughtered animals are most suitable. A 30 - 60 gram liver sample is taken from the edge of the organ and placed in a 10% formalin solution. While single samples are sometimes analysed, a preferred minimum of three samples from one locality is preferred for diagnostic purposes and corrective supplementation. The advantages of such a formalinised liver sample include a high correlation with mineral concentrations in fresh organ samples ($P<0,05$) and a repeatability with a high degree of accuracy for the first 8 weeks of storage ($P<0,01$). Whereas differences occur between samples taken close to large vessels and peripheral parenchyme, there is a high degree of repeatability within a series of parenchyme samples taken from one liver ($P<0,01$).

The procedure used by the author has been atomic absorption spectrophotometry (Beckman, model 979) after wet ashing of samples[4]. The process is rapid, permitting a broad spectrum of mineral analyses. There are, however, limitations in the system, as the method is unsuitable for the analysis of the halogens, sulphur and phosphorus.

SURVEY

In an extensive survey carried out by the author a combination of atomic absorption spectrophotometry and analysis in an Auto Analyser (Technicon) has been used to map the mineral contents of livers of animals in the Republic of South Africa and adjoining territories. Emphasis has been placed on cattle and sheep, but pigs, horses, poultry and 16 game species have also been investigated.

Over 10 000 samples originating primarily from herds or flocks regarded as potentially suffering from imbalances were analysed. They could be classified as originating from 10 regions, according to the mineral deficiencies which were encountered.

In this survey the six minerals of biological importance that received primary attention were copper, manganese, zinc, cobalt, iron and magnesium. In the later stages of the survey phosphorus was included and in isolated cases selenium analyses were conducted by an alternative method[1].

The results of this survey are to be reported in detail elsewhere. The important findings have, however, been summarised below:

a. There are marked differences between species in "normal" liver mineral concentrations.
b. There is a strong correlation in the liver mineral concentrations between members of a species grazing a locality. There are fluctuations between localities within a region, which do not, however, influence the overall pattern and deductions relevant to inadequacies or excesses.
c. The results indicated that the following mineral deficiencies (or excesses) occur in the 10 geographical regions studied:

1. **Northern Transvaal** (North of Warmbaths)
   Copper and zinc.
2. **Highveld** (Central and Southern Transvaal, Northern and Central O.F.S. down to Bloemfontein).
   Copper, manganese and zinc.
3. **Eastern Cape** (Inland from the mountain ranges) and **Southern O.F.S.**
   Magnesium, zinc and iron.
4. **Natal**
   Magnesium, copper, zinc, iodine and manganese.
5. **Cape Coast** (Cape Town to East London, seaward of the mountain ranges).
   Copper, zinc, manganese, magnesium, cobalt and iron.
6. **Western Cape** (West of Prince Albert and Carnarvon)
   Copper, manganese, iron and zinc.
7. **Northern Cape** (Calvinia, Carnarvon, Prieska, Postmasburg, Upington, Kenhardt).
   Manganese and zinc — (excess copper).
8. **South West Africa** (Northern third)
   Magnesium, copper, zinc and iodine.
9. **South West Africa** (Central third)
   Magnesium, copper and zinc.
10. **South West Africa** (Southern third)
    Magnesium, zinc and manganese.

MINERAL SOURCES

When specific minerals are inadequate in a region, it is important not only to supply the correct amount at controlled rates, but also to select the best source so as to assure optimum utilisation. Furthermore some seasonal adjustments in intake can be considered to

compensate for the drop in the mineral concentrations in plant material during dry seasons, e.g. winter in the summer rainfall areas. High levels of productivity, e.g. in high production dairy cows, can increase the requirements.

Intake can be controlled easily by correlating mineral additions with salt intakes, salt being readily taken in most environments but also acting as a brake.

Owing to their low cost and high mineral content the mineral sources most often used in the past were oxides and carbonates. It is now known that these sources are of lower biological value than the more soluble forms of the same minerals (e.g. chlorides and sulphates). Monoxides have poor solubility properties in the neutral and basic environment of the grazing animals' digestive tracts. It is, therefore, evident that when accurate diagnoses of micronutrient requirements are made, it is of importance to choose the mineral sources carefully.

CONCLUSIONS

The role of minerals in animal production has been recognised, and increasing attention is being directed to the problem, as mineral requirements and the effects of mineral imbalances are better understood.

Perhaps the biggest stimulus for further investigation and clarification is the knowledge that mineral imbalances, be they prominent or subtle, have powerful influences on the economics of animal production. Mineral adjustments, when accurately formulated can bring about substantial benefits at nominal cost.

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Jl. S. Afr. vet. Ass. 47(2) 133-136 1976

REVIEW **OORSIG**

CHROMOSOME ABERRATIONS IN DOMESTIC ANIMALS

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ABSTRACT: Osterhoff, D.R. **Chromosome aberrations in domestic animals.** *Journal South African Veterinary Association* (1976) **47** No. 2, 133 - 136 (En) Faculty of Veterinary Science, 0110 Onderstepoort.

The literature on chromosome abnormalities in domestic species is recorded and prospects for further work is outlined.

INTRODUCTION

The progress made in human chromosome research during the last two decades has also led to numerous investigations on chromosomes in domestic animals. The study of chromosomes from blood lymphocytes became a convenient technique for clinical survey and population studies, as advocated and initiated by Court-Brown[6] and Gustavsson[11]. It has been shown that the chromosomal abnormalities revealed in blood cells are often, although not always, mirrored in cells from other somatic tissue.

Most of the work performed so far has been done in cattle, but the discoveries in sheep, pigs, horses, goats and dogs will also be mentioned. This paper is an attempt to give the latest information on the chromosome abnormalities in our domesticated species.

A KARYOTYPE is a systematic presentation of the metaphase chromosome complement of a cell, characteristic for an individual animal or species. For cattle, the Karyotype, 60, XY, is prepared by cutting the chromosomes from an enlarged photograph of a chromosome spread and pasting them on a card, usually to an agreed standard scheme of arrangement (Denver or Chicago Classification).

A new technique was introduced by Caspersson *et al*[5] known as banding. This method has made the identification of individual chromosomes possible in nearly all species to which it has been applied. Hansen[18] reported success with banding the chromosomes of cattle.

It seems logical to classify abnormalities by the chromosomal defects observed and discuss the implications and observed occurrence of phenotypic defects, rather than to begin with the phenotypic defects. As yet correlations between chromosome defects and phenotypic defects are tentative pointers worthy of further investigation. The chromosomal aberrations will be studied more closely in the future and it may be presumed that the phenotypic defects which they represent will be more precisely defined for example as enzymological aberrations.

SEX CHROMOSOME ABERRATIONS

1. XX:YY CHIMERISM : THE FREEMARTIN CONDITION

In various disciplines this anomaly has been a favourite subject of research in an attempt to explain intersexuality[10]. In the etiopathogenesis the hormonal as well as the cytogenetical hypotheses as exclusive causes of freemartinism are not generally accepted any more: surely the combination of both factors originating from the male co-twin influences the development of female genital organs in a highly variable physiological direction. Etiology and pathogenesis of freemartinism provided the background for the understanding of disturbances in induction of genital organs in other Y cellbearing chimeric intersexes in cattle.

In sheep and goats freemartinism is less frequent[9]. The effects of chimerism are the same as in the bovine : sterility of the female co-twin also results. So far no sterility as has been seen in the bovine male co-twin[9] has been reported for sheep and goats.

It is interesting to note that in about 50% of all twin gestations in the more chorionic vascular anastomoses do develop[2]. This has also been established by blood group examinations. No deformation in the genital tract of such twins has been noticed and there was no reduction in fertility.

2. AUTONOMOUS XX/YY SYNDROME

Since 1967 four cases of bovine intersexes have been published which possessed XX/XY chimerisms, but which had been singletons or had been born as homosexual twins[26]. Those cases of intersexuality could have resulted from disturbances of fertilization independently from a male co-twin. Therefore, the existence of an autonomous XX/XY syndrome occurring in cattle, in analogy with the human XX/XY syndrome, must be taken into consideration. Probably, this new bovine syndrome, including several different forms of intersexuality, may be distinguishable from freemartinism by certain morphological peculiarities, for instance the development of a phallus and the transformation of the urogenital sinus into perineal hypospadias.

3. DIPLOIDY XX / TRIPLOIDY XXY CHIMERISM

This condition has been seen only in two animals, one of which revealed true hermaphroditism, characterized by ovaries, ovotestis, uterus, vagina and

Presented at the Annual Meeting : Mammal Research Institute, University of Pretoria, November 1975.

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penis. The other was a feminine pseudohermaphrodite with both ovaries, uterus, vagina, aplasia of vulva, and male accessory glands but without development of a penis. The character of virilizating transformation of gonads and urogenital sinus in both cases is not principally but gradually different. The graduation of such differences seems to be dependent on the quantities and the distribution of Y chromosome-bearing triploid cells within the organs and blastemata[25].

4. THE XXY SYNDROME

The symptomatology of the so-called Bovine Hypogonadism is principally in accordance with the KLINEFELTER-Syndrome in man. In three cases known in cattle until now, a high degree of congenital bilateral testicular hypoplasia with oligo- and necrospermia and corresponding disturbances in sexual behaviour was characteristic. In one of the cases gynecomastia had been observed. Two of the bulls revealed chimerism of 61, XXY cells in varying relations with 60, XX and XY cells, one of them 100% 61, XXY cells[25]. According to the KLINEFELTER-Syndrome gonadotropins (ICSH) are considerably increased in XXY bulls. Corresponding to the high-grade regression of endocrine active testicular tissue the incretion of testosterone before and after stimulation by HCG was considerably decreased. Surely, the relatively frequent hypoplasia of testicles in cattle is not caused by the XXY gonosomal constitution exclusively.

5. X-TRISOMY

In cattle X-Trisomy has only been observed in one case. This animal had been phenotypically normal, except for scoliosis of the lumbar spine, an anomaly assumed to be accidental. Especially its fertility was undisturbed. The karyotype of one calf born by this XXX cow possessed a normal 60, XX constitution[25].

6. ABERRATIONS OF THE X-CHROMOSOME

Aberrations within one long arm of the X-chromosome (breaks, deletions and gaps) has been described by El-Nahass *et al,*[7]. These abnormalities were found in 21% of the metaphases in a Friesian heifer. The heifer was sterile.

The small number of individuals and categories of gonosomal aberrations connected with functional and structural defects of genital organs in cattle do not make feasible at present the constitution of perfect and definite syndromes. The proposals for the classification of the different gonosomal chimeras are assumed to be a first step to elucidate the complicated relationships of all pathological variations of sex realization.

AUTOSOMAL ABERRATIONS

1. ROBERTSONIAN TRANSLOCATION OR CENTRIC FUSION

In the Swedish Red and White breed of cattle Gustavsson and Rockborn[14], described a centric fusion between two autosomes that is, they were joined at the centromeres. These were presumed to be the largest and the smallest elements, chromosome No. 1 and No. 29 in the bovine karyotype.

This 1/29 translocation has been described by cytogeneticists and in at least 12 different breeds in most developed countries[19 4]. Hultnäs[22] and Gustavsson[13] reported that due to screening of all A.I. bulls the translocation has been reduced to almost nil in the Swedish breeds.

Two Robertsonian translocations believed to be different and involving a small and a large autosome (referred to as Massey I and II) have been reported in sheep. One of these (Massey I) was present at an incidence of 4,6% in a flock of New Zealand Romney sheep and the other at the high level of 26,4% in selected Drysdale sheep[4]. The same investigator also found an autosomal translocation of the metacentric type involving two of the larger acrocentric autosomes (called Massey III). The latter translocation does apparently not affect the fertility, but it has been proved to be hereditary.

Bruere[4], 1974 found that the fertility of matings between translocation bearing rams (t) and normal ewes was equivalent or better than those of normal matings. On the other hand, t X t matings in general produced significantly lower lambing percentages.

Translocations giving metacentric chromosomes have also been reported in goats and here the interesting fact is, that the condition was found in animals born as singletons[26].

Gustavsson[12] recorded evidence which strongly suggested that daughters of translocation bulls returned to service more often than other cows of the same breed from normal sires.

2. TANDEM FUSION

Hansen[17] described a different form of autosomal translocation a tandem fusion, in which one large acrocentric chromosome was formed, instead of a submetacentric autosome as described in the centric fusion above. Hansen also recorded a marked infertility among carriers of this type of translocation. Herzog and Rieck[21] recorded a tandem fusion in an Amorphous globosus calf born with a normal female calf.

3. KARYOTYPE MOSAICISM AND CHROMOSOME POLYMORPHISM

Basrur and others[1] studied the chromosomes of the blood cells in a Holstein animal with lymphosarcoma, describing abnormal chromosome morphology and karyotypes with 60, 61 and 62 chromosomes. Herzog and Höhn[20] described chromosome polymorphism and abnormal diploid karyotype numbers correlating with parakeratosis in German cattle. Halnan[16] has observed polymorphism and abnormal diploid karyotype numbers correlating with parakerotosis in German cattle. Halnan[16] has observed polymorphism and hyperdiploid counts in two bulls, known to be close-bred, in Hereford lines where "cancer-eye" was known to occur.

4. POLYPLOIDY

Zartman and Fechheimer[28] suggested that the incidence of polyploid cells, that is cells with multiples of the normal chromosome number, 2n = 60, might be significantly higher among calves from certain in-bred Hereford lines. Popescu[24] published figures relating to the incidence of tetraploid cells in muscular hypertrophic Charolais cattle suggesting an

increase, although Vertessen[27] found no such departure from the normal chromosome number in the hypertrophic animals he studied. However, Herzog and Höhn[20] did find a high incidence of polyploid cells in calves born with abnormalities of the central nervous system.

5. GAPS AND SECONDARY CONSTRICTIONS

Halnan[16] reported that in eight animals, with a history of infertility, up to 60 percent of cells possessed autosomes, in the 14 to 26 groups, which had gaps or secondary constrictions. It was suggested that therein might lie an interesting avenue for further investigation, significant incidence possibly being indicative of the animal's impaired breeding potential. Herzog and Höhn[20] have described similar gaps in the chromosomes of calves with parakeratosis.

In pigs autosomal translocations and sticky anaphases were described recently[3]. In sheep the autosomal delection of chromosome Al was described by Luft and Wassmuth[23]. The ram with this abnormality was used for breeding and produced 11 lambs with the same deletion. No phenotypic deviation could be established.

PROSPECTS FOR FURTHER WORK

A background of knowledge and an array of well developed techniques for use with laboratory animals and man are now available for application to the economically important groups of animals[8]. From the most practical point of view, it is extremely important to ascertain, in a number of lines of cattle, sheep and pigs, the extent of embryonic death owing to heteroploidy as well as other unbalanced genomes of embryos. It is also important, in view of the apparently widespread occurrence of centric fusions, to determine their effect in heterozygous form on reproductive function of females. It is now reasonably clear that males are not adversely affected but it cannot therefore be assumed the same will be found for females.

CHROMOSOME BANDING

The techniques for demonstration of banding are now being applied in many laboratories. Each of the major types of bands, Q, G and C have been shown to be demonstrable. Gene mapping will be possible in the future[15].

CYTOGENETICS AND CELL GENETICS

It ought now to be possible, using the technique of somatic cell genetics, to identify activity of individual genes at many loci in all the domestic animals. Chromosome maps may be constructed and the work of localizing polygenes can begin.

EXPERIMENTALLY INDUCED ANOMALIES

With the many methods now available for production of both heteroploid animals and animals bearing structural aberrations, it is anticipated that a variety of stocks with aberrations will be made. They will be useful as models of naturally occurring aberrations.

Structural anomalies in heterozygous form may be used to investigate segregation differences between males and females, the extent of viability of embryos bearing deletions and duplications, the differential fertility of spermatozoa from the same male with different genetic endowments and other biological problems.

SEX PROPORTION

Attempts to devise ways to separate X-chromosome from Y-chromosome bearing mammalian spermatozoa have been hampered by the lack of methods to measure success of a given experiment. It is now possible in man and some primates to determine the presence of the Y-chromosome in spermatozoa. Possibilities for domestic animals are indicated.

TECHNICAL KNOWLEDGE OF CHROMOSOME STRUCTURE

Pulse labeling to determine the sequence and timing of replication of chromosome segments, DNA - DNA and DNA - RNA hybridization, electron microscopy of whole chromosomes or of differentiated segments, and scanning electron microscopy are now being applied to mammalian material and will reveal important insights into the nuclear biology of domestic animals.

USE OF COMPUTERS

In many laboratories dealing with human material, computers are being used in a variety of ways to relieve much of the tedium involved in cytogenetical research. In the most advanced system, routine screening of slides for identification of metaphase spreads, counting and measuring of chromosomes and preparation of karyotypes are all performed by machine. New possibilities are indicated again.

It is to be anticipated that chromosomal analysis will become as important as a diagnostic tool to the veterinarian as it has become to the physician and that the advancement of animal cytogenetics has as much to offer to the clinician as it has to the animal breeder.

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INFORMATION INLIGTING

MASTITIS CAUSES LOSSES IN NONINFECTED QUARTERS

A new case of mastitis in a quarter causes production losses not only in that quarter but in other quarters as well, according to George Marx, researcher at the University of Minnesota Northwest experiment station at Crookston.

Production dropped almost 31 percent in quarters that contracted a new infection. Researchers also found that milk production on opposite noninfected quarters was 13 percent lower, which indicates the cow's entire system is affected when a new incidence of mastitis occurs.

Production losses from just the infected quarter were staggering . . . about 3.5 pounds of milk daily. That amounts to a loss of 1,061 pounds in a 305-day lactation, or a value of $74.27 in one quarter with the price of milk figured at $7 per hundred.

Other losses not included are costs of the treatment drug, value of unsalable milk following treatment, veterinary costs, and a loss in value of the cow which follows most mastitis cases.

Marx found new mastitis cases were reduced 41 percent the first year and 32 percent the second year when cows had their teats dipped in a 10,000 parts per million solution of tamed iodine. One-third fewer treatments were needed for mastitis on cows that had their quarters dipped over the two-year period.

Squibb, Worldwide Animal Health. *Current Commentary.* October 1 1975. *Hoard's Dairyman* (1975) **120,** No. 15:888.

INFORMATION INLIGTING

TRANSMISSION OF THE VIRUS OF FOOT AND MOUTH DISEASE BETWEEN ANIMALS AND MAN

The virus of foot and mouth disease causes severe epizootics in animals and infrequently evokes painful, but transient, clinical signs in man. Adults in certain occupational groups and young children are particularly exposed to risk. Infected persons may disseminate virus for up to about 14 days. The virus can be transmitted from animals to animals, from animals to man, from man to animals and, probably, from man to man. Evidence for transfer of the disease between human and animal populations is reviewed in detail and modern methods of diagnosis are described. Predisposing factors play an important role in the development of overt foot and mouth disease in man. Subclinical infection occurs. The possibility of aerial transfer of the virus between man and domestic livestock constitutes a hazard, especially to the latter. Attention is directed to the need for sophisticated diagnostic techniques, to requirements for adequate precautions in the handling and disposal of affected animals, and to hygienic measures for disease control.

N. St. G. Hyslop. *Bulletin World Health Organization* (1973) **49**:577.

Jl. S. Afr. vet. Ass. 47(2) 137-141 1976

CASE REPORT **GEVALVERSLAG**

BOVINE CEREBRAL THEILERIOSIS : A REPORT ON FIVE CASES WITH SPLENIC INFARCTION

I.B.J. VAN RENSBURG*

ABSTRACT: van Rensburg, I.B.J. **Bovine theileriosis: A report on five cases with splenic infarction.** *Journal South African Veterinary Association* (1976) **47** No. 2, 137 - 141 (En) Dept. Path., Fac. Veterinary Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. South Africa.

Five cases of bovine cerebral theileriosis are described. Macroscopically visible thrombosis of branches of the splenic vessels with associated infarction of the spleen are reported for the first time in this disease. The presence of Koch's bodies in thrombosed splenic vessels or vessels clogged with lymphocytes in the spleen is also a new finding. These changes were encountered in all five cases.

INTRODUCTION

Theileria mutans is a very common infection of cattle in many regions of the Republic of South Africa. The resultant disease in the majority of cases is subclinical although occasionally, under circumstances that are poorly understood, the condition known as bovine cerebral theileriosis results, synonymously known as turning sickness or "draaisiekte" by the local farming population. This highly fatal form of the disease only occurs sporadically and occurs particularly in young stock.

All the descriptions of cerebral theileriosis in the literature state that the lesions are more or less confined to the central nervous system (CNS). Mettam and Carmichael[3] described 24 cases of cerebral theileriosis and found no significant pathological changes other than in the brain. They specifically stated that the spleen, lymph nodes, liver and lungs were free from lesions. In very few of their cases were schizonts demonstrated outside the CNS and then only with difficulty. Flanagan and le Roux[2] in their report on two cases described lesions only in the CNS.

During a 22 month period from March 1973 to January 1975 three cases of bovine cerebral theileriosis were autopsied at the Veterinary Research Institute, Onderstepoort and two at the Department of Pathology, Faculty of Veterinary Science, University of Pretoria, Onderstepoort. In addition to lesions in the CNS all five cases showed the presence of single or multiple infarcts of varying size in the spleen and thrombosis of branches of the splenic blood vessels. Koch's bodies were present in these infarcted areas.

CASE HISTORIES

Case I, a 14 month old Afrikaner ox from the Cullinan district. The owner stated that the animal had been blind for several days, had turned in circles with a staggering gait and had walked lifting the front legs very high. It eventually became recumbent and showed severe opisthotonus. Suspecting heartwater the owner had treated it with large doses of tetracycline administered parenterally but to no avail.

Case II was from Randburg. It was a Sussex cow, approximately 3 year old submitted *in extremis*. Clinical signs described by the owner were those of disturbed balance which progressed to recumbency. Upon arrival the animal was completely paralysed, blind in both eyes and showed nystagmus. It was killed by the intravenous administration of an overdose of pentobarbitone sodium**.

Case III, an 18 month old Pinzgauer bull was received from Pyramid in the Pretoria district. According to the owner it had been taken to Pyramid from the Amersfoort area 2 months previously. Upon arrival it was vaccinated against heartwater and in order to detect a possible reaction to the vaccine, the rectal temperature was taken daily and tetracycline treatment administered at the first temperature reaction. Subsequently a light tick infestation was allowed on the animal in order to maintain a premune status. The owner saw the bull early on the morning of the day it died when it was ruminating and apparently in good health. The rectal body temperature was normal that morning. At 09h00 the attendant noticed that the bull was not feeding and that it walked with swaying hindquarters. The owner was summoned immediately. He noticed respiratory distress and found the rectal temperature to be 38°C. Treatment consisted of Reverin†, Berenil† and Deltacortril△ administered parenterally. That afternoon the bull was recumbent and experienced attacks of nervous symptoms showing nystagmus and opisthotonus. Its condition deteriorated rapidly and death supervened at 21h00 on the same day.

Case IV was a Friesland heifer from the Pretoria district approximately 1 year old and in poor condition. The first clinical signs noticed were anorexia and a goosestepping gait. Treatment consisted of Reverin†, Berenil† and Vitamin B complex administered parenterally. The animal's condition at first deteriorated but after 3 days there was a slight improvement, although it was still very weak in the

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** Euthatal - Maybaker (S.A.) (Pty) Ltd.
† Hoechst Pharmaceuticals (Pty) Ltd.
△ Pfizer (Veterinary division)

hindquarters and lost its balance. The next day it was found in a ditch into which it had fallen and finally died 5 days after the first appearance of clinical signs.

Case V was received for post mortem examination from Pyramid. It was a 15 month old Guernsey heifer.

Autopsies were performed on these cases and specimens from various organs were fixed in 10% buffered formalin, embedded in paraffin wax for the preparation of histological sections. Sections were stained with haematoxylin and eosin, Giemsa, and by Perl's method (for iron).

RESULTS

The results are partly summarised in Table I.

Table 1: POST MORTEM FINDINGS IN FIVE CASES OF BOVINE CEREBRAL THEILERIOSIS

Case No.	Date of death	Age	Smears for Koch's bodies		Koch's bodies in sections		Splenic Infarcts	Brain yellow discolouration
			Brain	Spleen	Brain	Spleen		
I	27/3/73	14 months	+	–	+	+	One large	+
II	1/5/73	±3 years	+	–	+	+	One large	+
III	25/4/74	18 months	+	–	+	+	Two medium	+
IV	13/1/75	12 months	–	–	+	+	Several small	+
V	30/1/75	10 months	–	–	+	+	Several small	±

Gross Pathology: The carcass of Case I was in fair condition, dehydrated and cyanotic with a moderate tick infestation. There was ruminal stasis, bile stasis, slight nephrosis and one large pale splenic infarct which measured approximately 6 × 8 × 3 cm. Thrombosis was clearly evident in several arteries in the capsule and substance of the spleen. On opening the cranial cavity the right cerebral hemisphere showed extensive thromboses of subdural blood vessels with severe haemorrhage in the meninges as well as into many areas of the brain substance and into the lateral ventricles. Encephalomalacia rendered the brain soft and pulpy in areas. There was a slight dirty, yellowish tinge to the brain as a whole.

The most significant findings in Case II were confined to the brain, spleen and kidneys. The latter contained scars resulting from multiple infarcts. A large infarct was present in the spleen which resembled that of the previous case with the exception that it was a long standing lesion and was surrounded by a distinct fibrous capsule (Fig. 1). Thrombosis of several vessels in the spleen were also present. The meninges and brain substance was very congested and contained multiple haemorrhages. Thromboses of meningeal blood vessels were prominent. The brain had a generalised yellowish discolouration which was more pronounced in focal areas. The ventricles were filled with massive blood clots which were also visible from the outside of the brain where the hypophyseal stalk and olfactory lobes had been cut off. Large areas of the brain had a soft malacic consistency, especially the frontal lobes.

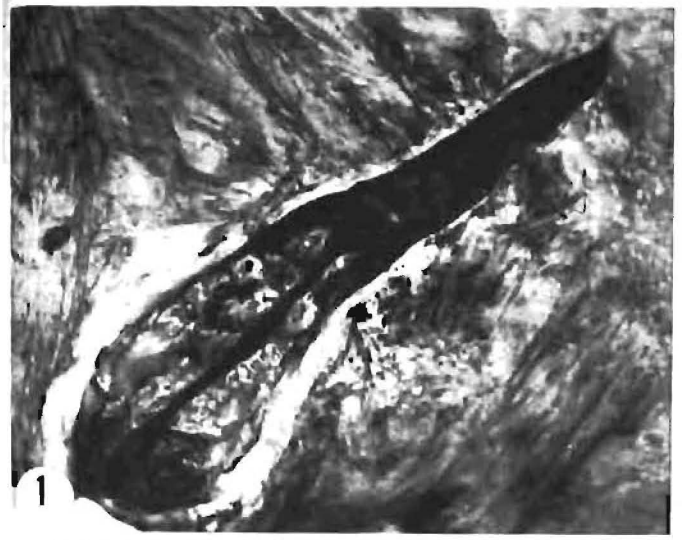
The brain of Case III was very similar in appearance. The choroid plexus was swollen and had a greyish-red colour due to haemorrhage and thrombosis. The spleen was markedly enlarged and of a soft consistency. On palpation two firm areas about the size of a dove's egg were detected which, on closer inspection, were found to be infarcts. Macroscopically, no thrombi were visible in the splenic vessels. The liver and kidneys were slightly enlarged and degenerated. The lungs were congested and severely oedematous. Peripheral lymph nodes were enlarged and oedematous. The intestines were hyperaemic with a few mucosal petechiae and ecchymoses. In general, the lesions were very reminiscent of heartwater, except for the splenic infarcts and the thromboses and haemorrhage in the brain and meninges.

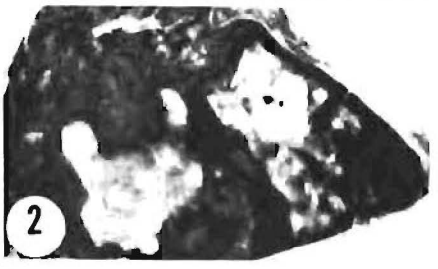
The lesions in the brain of Case IV very closely resembled the preceding cases while the only lesion seen in this organ in Case V was severe haemorrhage into the ventricles and choroid plexus. The ventricles were distended with clotted blood and the choroid plexus resembled a mass of clotted blood. The spleens of both these animals had several small pale infarcts varying from 1 to 2 cm in diameter (Fig. 2). These were easier to locate by palpation than by sight; their consistency was considerably firmer than that of normal splenic tissue. Both animals were severely infested with ticks amongst which *Rhippicephalus appendiculatus* was found.

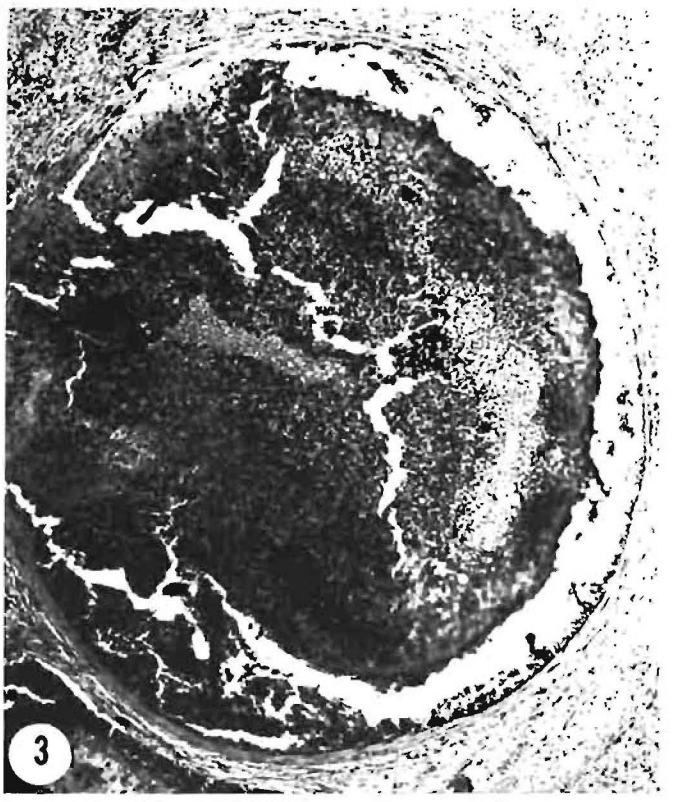
Smear examination: Giemsa stained smears prepared from affected areas in the brain and spleen were examined in all cases. Koch's bodies were found in brain smears of Cases I, II and III only but none in the spleen smears. No *Cowdria ruminantium* parasites, the cause of heartwater were present in smears prepared from the brain of Case III. Blood smears from all cases were negative for protozoal parasites.

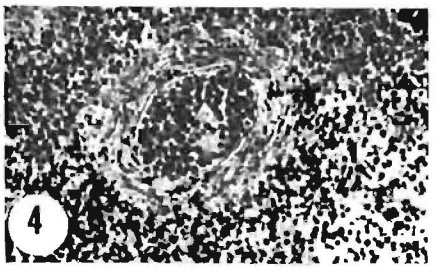
Histopathology: The microscopical lesions encountered in the brains of these five cases were very similar and did not differ significantly from those described by Mettam and Carmichael[3] and Flannagan and le Roux[2]. The differences being more a matter of degree rather than in character. Lesions were not limited to any particular area of the brain. The most conspicuous and constant changes were the extensive thrombosis of the sub-arachnoidal blood vessels and of those of the pia mater (Fig. 9). These were accompanied by extensive haemorrhages. Many blood vessels were not thrombosed but were clogged with lymphocytes which were mainly young blast type cells in which mitotic figures were reasonably common. Numerous intracytoplasmic Koch's bodies occurred in these lymphocytes. Free Koch's bodies were encountered in some cases. Similar changes occurred in many blood vessels in the brain substance.

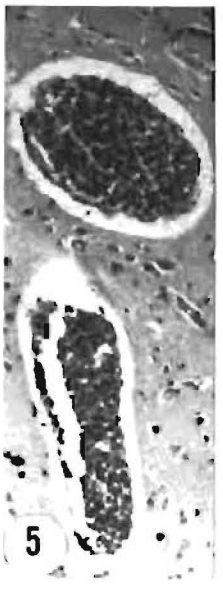
A true perivascular cuffing was not a feature of these cases, but several vessels had a mild infiltration of lymphocytes in the adventitia, with occasional cells being present in the Virchow-Robin spaces. In these lymphocytes, which gave the impression that they had seeped out of the blood vessels, the occasional Koch's body was present — but these were relatively scarce compared to their intravascular occurrence. Many cerebral blood vessels in and around affected areas, exhibited degenerative and necrotic changes in

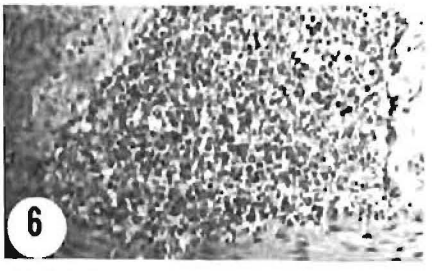


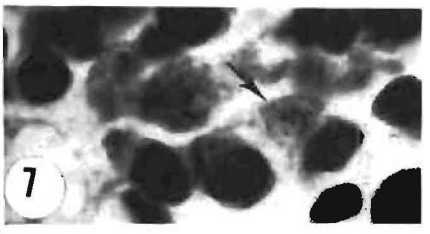


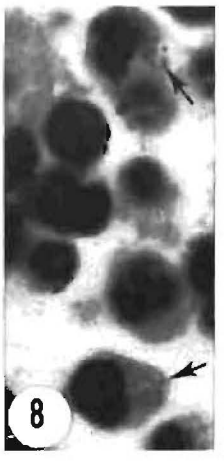












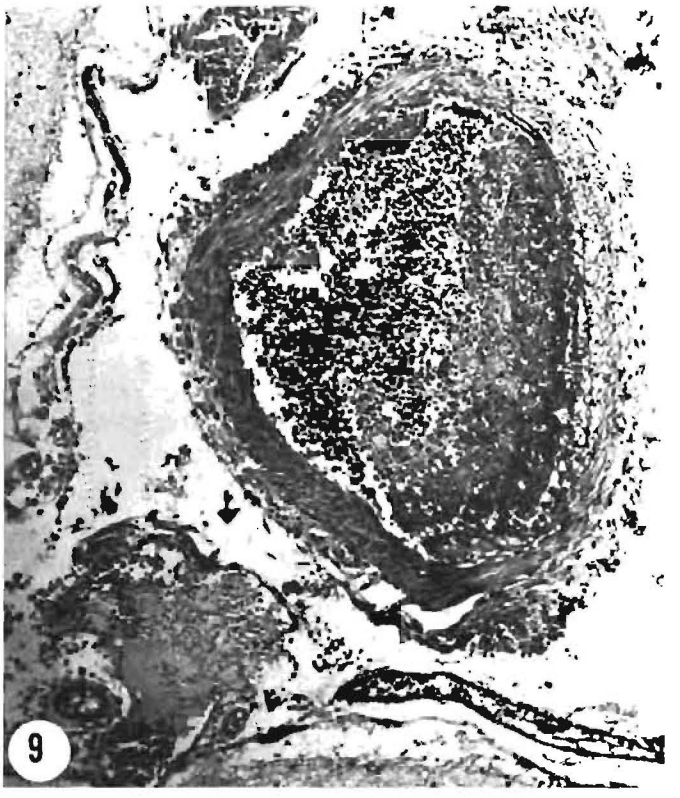


Fig. 1: Large splenic infarct and thrombosis
Fig. 2: Small splenic infarcts
Fig. 3: Thrombosis of large splenic artery HE × 60
Fig. 4: Clogging of a small artery in the spleen with lymphocytes HE × 200.
Fig. 5: Clogging of blood vessels in the brain HE × 200.
Fig. 6: Clogging of a trabecular artery in the spleen HE × 200.
Fig. 7 & 8: Koch's bodies in lymphocytes from clogged vessels HE × 1200.
Fig. 9: Meningeal artery partially thrombosed and partially clogged with lymphocytes. Also note haemorrhage and round cell infiltration of meninges. HE × 60.

their walls, which thus appeared thickened and homogeneously eosinophilic while in some, the nuclei of the cells of the vessel walls were pyknotic or karyorrhectic.

In Case I and to a lesser degree in Case II large areas of encephalomalacia were present i.e. infarcts in the brain. In these areas proliferation of capillaries and of Gitter cells was a prominent feature. Some areas of the infarcts in fact consisted of almost nothing but capillaries and Gitter cells — early stages in "cyst" formation. Brain oedema of varying intensity was present in all cases. Even in the same animal the intensi-

ty of oedema varied between the different areas of the brain, but with no specific pattern of distribution. This was manifested by the presence of eosinophilic globules in the Virchow-Robin spaces or faintly eosinophilic material in this area.

Macrophages laden with haemosiderin (staining positive with the Prussian blue reaction) were present around many cerebral and leptomeningeal blood vessels. These were most prevalent in Case II.

Apart from the macroscopically evident haemorrhages small microscopical haemorrhages were also present in most sections of the brain. The meninges were more cellular than normal due to round cell infiltration. In cases II, III and V, plasma cells were numerous and some monocytic infiltration had also occurred. In Case I, in a few foci of malacia there was a mild neutrophil infiltration into the necrotic brain tissue. The choroid plexus in Cases I, II, III and V showed clogging of vessels with lymphocytes parasitised by Koch's bodies accompanied by thrombosis and haemorrhage. The hypophysis was similarly affected in Case II.

In Cases I and III it was common to find several eosinphiles around blood vessels in the affected areas of the brain.

A very interesting finding was the presence of several thrombosed blood vessels or vessels clogged with young lymphocytes in the vicinity of the splenic infarcts in all five cases (Fig. 3, 4 and 6). In these vessels many of the lymphocytes were parasitized by Koch's bodies. Schizonts were not however, found in the normal areas of the spleen. Haemosiderosis of the spleen was much in evidence.

Kidney sections from Case III revealed partial thrombosis of some subcortical vessels. Many vessels showed aggregations of lymphocytes in their immediate vicinity although no Koch's bodies were found in these localities. In the sections from the intestine of the same case, one thrombosed blood vessel was encountered. No Koch's bodies were seen here either.

The kidney lesions in Case II were confirmed histologically as scars resulting from infarction. No active cell reaction remained in these areas.

DISCUSSION

The cases of turning sickness described in this paper occurred mostly, like those described by previous authors[2 3], in young to young adult cattle. Furthermore all of them occurred in the late summer to autumn, which coincides with that time of the year when the tick population is at its highest. It does seem possible therefore that there is a positive correlation between degree of tick infestation and the incidence of cerebral theileriosis. In three cases there was a history of movement of the victim to another locality some period beforehand. Although this was not constant it would appear that movement plays some role in the pathogenesis of the disease, especially if it is from the highveld to a more severely tick infested area.

The most significant lesions were confined to the brain, meninges and the spleen. The lesions in the former closely resembled those described by other authors[2 3].

A constant finding was the dirty yellow discolouration of the brain. The demonstration of varying amounts of haemosiderin in the brain and meninges of affected cases is the most likely explanation. The presence of this pigment is most probably indicative of previous bouts of thrombosis and haemorrhage, suggesting that these animals must all have survived previous but non-fatal "attacks" of cerebral theileriosis, which, in some cases at least, were apparently sub-clinical in nature.

Previous authors mention that Koch's bodies are scarce in the extravascular lymphocytes in the brain. This is in accordance with these findings. In order to demonstrate these schizonts most easily in sections the best method is to locate a vessel which is clogged by numerous lymphocytes under low magnification and then to examine it under higher magnification. This explains why the brain smears in the cases under review were not all positive for Koch's bodies for if such a blood vessel is not incorporated in the piece of brain tissue used in the preparation of the smear, it is unlikely that parasites will be found. The most reliable method for the diagnosis of cerebral theileriosis is therefore the histopathological examination of the brain.

Another interesting observation in this respect was that the Koch's bodies in smears were stained very well by the Giemsa method. In sections, however, ordinary haematoxylin and eosin staining gave a better definition of the parasite and is preferred to the Giemsa method for the demonstration of Koch's bodies in sections.

Furthermore it was apparent that any part of the brain could be involved, as lesions were encountered in various parts of cerebrum, cerebellum, midbrain and brain stem. The choroid plexus and hypophyses were similarly affected in some cases. Unfortunately the spinal cords were not examined.

The demonstration of infarcts in the spleen for the first time is interesting — especially in the light of the fact that they were present in all five cases. It seems probable therefore that they have been overlooked in the past. When the infarcts are small they will not be detected if the spleen is not thoroughly palpated. Owing to their firm consistency palpation is the easiest means of locating them. Although the odd Koch's body has been reported in spleen smears by previous workers[1 2] in these cases they were present in every instance. It is important, however, that blood vessels characteristically clogged with lymphocytes be examined for their presence. It is of interest to compare the lesions in turning sickness with those of *Babesia canis* infection in dogs where infarction of the brain and/or spleen and even other organs occurs in some cases due to vessels obstructed by sludging of parasitised erythrocytes. The question arises as to whether infarcts are limited to the spleen and brain in cases of cerebral theileriosis or whether they are more widespread as found in some forms of canine babesiosis. It seems probably that they do indeed occur in other organs especially if one considers Case III where partially thrombosed blood vessels were found in sections of intestine and kidney. The absence of Koch's bodies in these vessels does not necessarily exclude *T. mutans* as an aetiological factor in their pathogenesis. The multiple scarring due to infarcts encountered in the kidneys in Case II might also have been due to the thrombosis initiated by clogging of vessels by parasitized lymphocytes.

The pathogenesis of the disease remains unelucidated. Why a relatively mildly pathogenic parasite alters its normal behavioural pattern with fatal consequences is not known. One possibility is

that the toxin of *Rhippicephalus appendiculatus* (brown eartick) with its suppressing effect on the reticulo-endothelial system of the body[5] may render an animal more susceptible due to its lowered resistance. This form of tick toxicosis was described again by Neitz[4] and confirmed by van Rensburg[6] who also showed that it led to the appearance of moderate numbers of erythrocytic forms of *Gonderia mutans* (syn *T. mutans*). It has been reported, that under conditions of bad management and severe tick infestation *T. mutans* can provoke pathological lesions indistinguishable from those of *Theileria parva*. In one such instance a relapse to severe *T. mutans* infection was experimentally induced by splenectomy[1].

The possibility exists that an auto-immune disorder, induced by the parasite, within the lymphocytes is responsible for the intravascular agglutination of lymphocytes. The presence of large numbers of plasma cells and eosinophiles in some cases supports such a point of view. There is no doubt that once the flow of blood within a vessel is impeded by agglutinated lymphocytes, thrombosis in the rest of the vessel is likely to follow. This in turn, in the case of the brain and spleen (and any other organ with an end-artery system) leads to infarction.

It should be borne in mind that in South Africa *T. parva* was never associated with cerebral lesions while in Uganda cerebral theileriosis was linked to this parasite[3]. The fact that *T. mutans* and *T. parva* both occur in Uganda and the difficulty encountered in distinguishing between the two parasites on morphological ground, as well as the fact that these cases were all in East Coast fever immune animals, makes one wonder whether the aetiology was in fact *T. mutans* and not *T. parva*.

ACKNOWLEDGEMENTS

I wish to express my gratitude to Dr J.C. Lloyd of the Veterinary Research Institute, Onderstepoort for submitting one of the cases to me. Professor R.C. Tustin is thanked for his helpful guidance in the preparation of the manuscript. The technical staff in Pathology is thanked for the preparation of histological sections.

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TO THE EDITOR — AAN DIE REDAKSIE

HEARTWATER: THE IMMUNISATION OF ANGORA GOATS

Dear Sir,

It is a well-known fact that there is no satisfactory heartwater vaccine available. When susceptible animals are infected with the heartwater organism they contract the disease[1]. If treated with tetracycline antibiotics at the height of temperature reaction[5] such animals recover and experience an immunity to heartwater which lasts for a variable period of time[2]. Instead of treating the individual animal showing a temperature reaction after heartwater inoculation, Poole[4] suggested that goats (in his case cross bred Angora / Afrikaner kids) should be block treated with tetracycline antibiotics on the eleventh and thirteenth days after inoculation with heartwater blood.

One group of Angora goats on a farm in the Albany district was inoculated with heartwater blood (Ball-3 strain) as supplied by the State Veterinarian, Grahamstown (GHV). This blood is at present obtained from sheep at the height of the temperature reaction due to inoculation with the heartwater organism. It is collected into a sodium citrate solution. A second group of goats on the same farm was inoculated with heartwater blood (Ball-3 strain) as supplied by the Veterinary Research Institute Onderstepoort (OHV). This blood is also obtained from sheep reacting to heartwater infection, but is presently collected in an anticoagulant solution containing 1% dimethylsulphoxide[6] and stored at about –60°C.

Rectal temperatures of all the experimental animals were taken once daily between the tenth and the twentieth days after inoculation. All animals with a temperature of 40,6°C or more were regarded as reactors and were treated according to the schedule proposed by Poole[3]. A summary of all the relevant data is given in Table 1.

If it is borne in mind that the animals used in these trials were raised on a known heartwater farm it could be expected that some of them would not react to inoculation due to previous exposure to the natural disease. According to Table 1 about 81% of all animals reacted. The number of animals reacting to OHV was significantly higher than to GHV. This indicates that the percentage of reactions is dependent on the type of vaccine used.

From Table 1 it can be seen that about 67% of animals which received GHV reacted between the tenth and thirteenth days after inoculation. After inoculation with OHV about 89% of cases reacted during the same period. If GHV is thus used on a flock basis and the animals are block treated as suggested by Poole[4] it can be expected that in about 33% of the inoculated animals the incubation period would exceed the second treatment. With OHV the number of reactors which could be expected after the second treatment is only about 11%. For practical purposes this still represents a high mortality risk. When the second treatment is postponed to the fourteenth day after inoculation it could be expected that only about 3% of animals inoculated with OHV would not have reacted.

For the inoculation of Angora goats against heartwater it appears that the present OHV is superior to the present GHV. When OHV is used on a flock basis block treatment with tetracycline antibiotics should be applied on the eleventh and the fourteenth days after inoculation. Due to a high risk of delayed temperature reactions and subsequent mortality GHV should not be used for flock inoculation with block treatment.

Yours faithfully

J.A. ERASMUS
State Veterinarian
Grahamstown

Table 1: **REACTIONS OF ANGORA GOATS AFTER VACCINATION AGAINST HEARTWATER**

Vaccine used	No. vaccinated	No. reactions	No. reactors with first temp. reaction on days:			
			10-13	14-20	10-14	15-20
GHV	176	122 (69,4%)	82 (67,2%)	40 (32,8%)	93 (76,3%)	29 (23,7%)
OHV	172	161 (93,8%)	143 (88,8%)	18 (11,2%)	156 (96,9%)	5 (3,1%)
Chi-square for vaccines	33,41		19,91		27,18	

For 1 degree of freedom (i) $P<0,001$ if $X^2 = 10,827$
(ii) $0,001<P<0,01$ if $X^2 = 6,635$

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FEATURE PAGE

TREFFERBLAD

OVINE SEPTUPLETS

The subject was a 4 to 6 tooth flock type Dorper ewe, running on natural Karoo grazing, which on account of ample rains, was in extremely good condition. On the morning of the 10th of October, 1975 she was noticed to have lambed, probably a day or two earlier, and mothering seven lambs, consisting of three ewe and four ram lambs. The one ram lamb was in a very weak condition and died when transported to the farmstead. Unfortunately individual and total birthweights were not recorded.

According to the history this ewe was a member of a flock of 180, which was mated in a flock mating system to four rams, during April-May the relevant year.

Blood was collected for parentage determination from the ewe, the six surviving lambs and three of the rams, which were still available. According to the report this particular ewe could have been the mother of five of the surviving lambs if she was mated by either of two rams, on condition that one of these rams which was not available at the time, was of the Transferrin type AC. If however she was mated by both rams during the same oestrous cycle, this ewe could have given birth to all six surviving lambs. This is of course very likely as in a flock mating system, the mating of a single ewe by more than one ram is frequently observed.

Further evidence that the missing ram should have posessed the Transferrin type AC is found in the fact that one of the lambs of another ewe with four teats, is also of the Transferrin type AC, while neither his mother nor the three rams that were bled possess the A component.

Contributor: Dr E.M. van Tonder,
Regional Veterinary Laboratory,
5900 Middelburg, Cape.

SEWELING IN 'N SKAAP

Die betrokke dier was 'n 4 - 6 tand Dorper ooi wat op natuurlike Karoo veld gewei het. Weens die baie goeie reëns was sy in 'n baie goeie kondisie. Die oggend van 10 Oktober 1975 is bemerk dat sy gelam het, waarskynlik 'n dag of twee vantevore. Sy het sewe lammers versorg, waarvan drie ooilammers en vier ramlammers was. Die een rammetjie was baie swak en het tydens die vervoer na die opstal gevrek. Ongelukkig is die individuele of totale geboortegewigte nie aangeteken nie.

Volgens die geskiedenis was sy een uit 'n kudde van 180 ooie wat gedurende April-Mei gedek is in 'n kudde dekprogram waarin vier ramme gebruik was.

Bloed is gekollekteer van die ooi, ses lammers en drie van die ramme wat nog beskikbaar was, ten einde ouerskap te bepaal. Volgens hierdie verslag kon die ooi die moeder van vyf van die oorlewende lammers wees, indien sy deur een van twee van die ramme gedek sou gewees het, met die voorbehoud dat een van hierdie ramme, wat nie toe beskikbaar gewees het nie, van die Transferrin tipe AC was. Indien sy egter deur beide ramme gedurende dieselfde bronstigheidsiklus gedek sou gewees het, kon die ooi geboorte gegee het aan al ses die oorlewende lammers. In 'n kudde dekprogram is dit heel moontlik, omdat dikwels gesien word dat 'n enkele ooi deur meer as een ram gedek word.

'n Verdere bewys dat die vermiste ram die Transferrin tipe AC gehad het, is geleë in die feit dat een van die lammers van 'n ander ooi met vier spene ook van die Transferrin tipe AC was, terwyl nie sy moeder óf een van die ander drie ramme wat gebloei is, die A komponent besit het nie.

Bydraer: Dr E.M. van Tonder,
Veeartsenykundige Streekslaboratorium,
5900 Middelburg — Kaap.

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AWARD

S A V A GOLD MEDAL : SECOND AWARD

In 1973 the Council considered proposals for the award of the Association's highest award. On its recommendation the then President, Dr A.B. la Grange presented the S A V A's Gold Medal to

PROF. DR SCHALK WILLEM JANSE VAN RENSBURG

during the course of the Biennial National Veterinary Congress held in Pretoria, "FOR DISTINGUISHED SERVICE TO THE PROFESSION".

TOEKENNING

S A V V — GOUE MEDALJE : TWEEDE TOEKENNING

In 1973 het die Raad voorstelle oorweeg vir hierdie hoogste toekenning deur die Vereniging. Ooreenkomstig sy aanbeveling is die Medalje namens die S A V V deur die president, Dr A.B. la Grange tydens die Tweejaarlikse Nasionale Veterinêre Kongres te Pretoria toegeken aan

PROF. DR SCHALK WILLEM JANSE VAN RENSBURG

in erkenning "VIR SY UITMUNTENDE DIENS AAN DIE PROFESSIE".

Prof. Dr S.W.J. van Rensburg.

Born in Cradock in 1896, he matriculated there and obtained the B.A. degree at Victoria College, Stellenbosch in 1917. By 1921 he had completed his studies at The London Veterinary School and was an admitted member of the Royal College of Veterinary Surgeons. On his return to South Africa he became Government Veterinary Officer (GVO) to a number of Karoo districts and lectured at the Grootfontein Agricultural College at Middelburg. Here he did pioneer work on hypoglycaemia in ewes and on tick borne paralysis of sheep. He developed the well-known shallow dip for control of the latter. After subsequent duty as GVO at Ermelo and Vryheid he joined the Research Institute at Onderstepoort in 1935, where he also lectured in medicine in the Faculty of Veterinary Science of the University of Pretoria. In 1945 he became lecturer in Surgery and Gynaecology and obtained the DVSc. for his thesis on "The secretion of abnormal milk by quarters free from known pathogens". In 1947 he became Professor and Head of the Department of Surgery and Gynaecology.

In 1953 he was appointed "Senior Research Fellow"

Gebore te Cradock in 1896 het hy daar gematrikuleer en in 1917 die B.A.-Graad aan die Viktoria Kollege te Stellenbosch verwerf.

In 1921 voltooi hy sy studies aan die Veeartsenyskool te Londen en word hy 'n "Member of the Royal College of Veterinary Surgeons". Na sy terugkeer word hy Staatsveearts vir verskeie distrikte in die Karoo en tree hy op as lektor aan die Grootfonteinse Landboukollege te Middelburg. Hier doen hy baanbrekerswerk in verband met toestande soos domsiekte by ooie en bosluisverlamming. Vir laasgenoemde ontwerp hy die nou-nog-bekende vlak dipbak. Na diens as Staatsveearts te Ermelo en Vryheid verplaas hy na Onderstepoort in 1935, waar hy as navorsingsbeampte aan die Instituut en as lektor in Geneeskunde in die Fakulteit Veeartsenykunde van die Universiteit Pretoria optree. In 1945 word hy lektor in Chirurgie en Geslagskunde en verwerf hy ook die D V Sc vir sy proefskrif oor "The secretion of abnormal milk by quarters free from known pathogens". In 1947 word hy Professor en Hoof, Dept Chirurgie en Geslagskunde aan die Fakulteit.

of the Meat Board's Stock Diseases Research Fund to undertake fulltime research work on infertility and artificial insemination of farm livestock. In 1965 he retired from this position but not from active participation in the affairs of his profession.

His interest and work ranged over a wide field : mastitis; infectious and functional infertility; the investigation and diagnosis of epivaginitis in 1949; the establishment in 1950 of vibriosis as the cause of low fertility in cattle; the diagnosis of brucellosis in rams as a cause of epididymitis and its control by use of Rev 1 vaccine; functional infertility in mares and cows; genetic factors causing reproductive failure such as loose heads in semen in certain Guernsey bulls and abortion in Angora goats. His pioneer work did much to stimulate further research in this field by others.

His wide knowledge and experience of A I and his own research work made him an acknowledged authority. He gave freely of his knowledge and experience to breeders and his colleagues. He was successful in using longterm frozen ram semen where others had failed. In South Africa he could justifiably be called "The Father of A I". He did much to advocate and publicise A I and was largely responsible for the establishment of A I-Coops, the training of inseminators, the drafting of legislation to control A I and the control of the use of this method.

From his able hand came numerous popular and scientific publications, some of which he has kept up to date and which are still today used as standard works. He has been a member of the Association (S A V M A in early days) since 1922 and served as Honorary Secretary-Treasurer from 1936 to 1945. He became a member of Council in 1945 and was elected President from 1950 - 52. He is now Honorary Life Vice-President of the Association. He served as the representative of the S A V A on the Veterinary Board and the A I Board, and was chairman of the Association's Finances Committee for many years. He also represented the S A V A on committees inquiring into veterinary matters under the chairmanship of people like Centlivres, Adams, Marais and Brink.

He is well known abroad, having made various study tours and having attended numerous congresses on fertility and reproduction as speaker and as chairman. In Trento, in 1964, he was awarded a medal and scroll "In recognition of his scientific achievements and his contribution to scientific knowledge which merits international acknowledgement and appreciation". In 1976 he took up an invitation to spend some weeks at the Gynaecological Clinic of the Veterinary School at the University of München.

His contribution to our livestock industry, to education and training of breeder and veterinarian, to research and the well being of our profession, and to the image of our country is undeniably magnificent. His service and devotion to a huge task and over a wide field has set an enviable example to us all. He is undoubtedly worthy of the highest honour we as colleagues can bestow on him.

In 1953 word hy aangestel as "Senior Research Fellow" van die Vleisraad se Veesiektenavorsingsfonds en wy hy hom voltyds aan probleme betreffende onvrugbaarheid en die toepassing van kunsmatige inseminasie by plaasvee; in 1965 tree hy af maar dit het nie 'n periode van onaktiwiteit meegebring nie.

Sy belang en werk het gestrek oor 'n wye veld : mastitis; aansteeklike en funksionele onvrugbaarheid; ondersoek en vasstelling van epivaginitis in 1949; vasstelling en bepaling van vibriose as oorsaak van lae vrugbaarheid by beeste in 1950; brucellose by ramme en epididimitis as gevolg daarvan — diagnose, voorkoming deur inenting met Rev 1 entstof; funksionele onvrugbaarheid by merries en koeie is in diepte ondersoek; erflike onvrugbaarheidsprobleme soos loskoppe in semen van sekere Guernsey-bulle en misgeboorte by Angorabokke. Sy baanbrekerswerk het ander tot verdere werk in diè velde gestimuleer.

Sy omvattende kennis van kunsmatige inseminasie en sy navorsingswerk in die verband het hom 'n kenner daarvan gemaak. Hy het nooit geaarsel om aan boere en veeartse sy kennis en ervaringe beskikbaar te stel nie. Hy het onder andere die eerste sukses met lankbevrore ramsaad bereik. Wat Suid-Afrika betref kan hy met reg die "Vader van K I" genoem word. Hy was grootliks verantwoordelik vir reklame vir K I, die stigting van K I-koöperasies, die opleiding van insemineerders èn die opstel van wetgewing vir die beheer van K I.

Hy was 'n vlot en skeppende skrywer van populêre sowel as wetenskaplike publikasies, sommige waarvan hy steeds op datum hou omdat hulle nog vandag as handboeke dien.

Hy is sedert 1922 lid van die S A V M V soos dit vroeër genoem is. Vanaf 1930 tot 1945 het hy as Ere-Sekretaris-tesourier die Vereniging gedien. Vanaf 1945 was hy lid van die Raad en vanaf 1950 - 52 was hy President. Hy is tans Lewenslange Ere Onderpresident. Vir etlike jare het hy die S A V V op die Veeartsraad en die K I-raad verteenwoordig.

Hy was ook lank voorsitter van die Geldsakekomitee en het die Vereniging op komitees van ondersoek na veeartsenykundige aangeleenthede onder voorsitterskap van Centlivres, Adams, Marais en Brink verteenwoordig.

In die buiteland is hy welbekend as gevolg van verskeie studiereise, bywoning van internasionale kongresse oor K I en Voortplanting. Hy het daar betekenisvolle bydraes gelewer en soms as voorsitter opgetree. In 1964 is 'n medalje met oorkonde te Trento aan hom oorhandig : "In recognition of his scientific achievements and his contribution to scientific knowledge which merits international acknowledgement and appreciation". In 1976 het hy op uitnodiging die Universiteit van München se Kliniek vir Geslagskunde as gaste-dosent besoek.

Sy bydrae tot ons veebedryf, tot opvoeding en opleiding van veeartse èn veeboere, tot die navorsing en die welsyn en vooruitgang van die professie, en tot ons land se beeld is onteenseglik groot. Hy is minlik en geesdrif as persoon. Sy diens en toewyding aan 'n reuse taak oor 'n wye veld is vir ons almal 'n voorbeeld. Hy verdien terdeë die allerhoogste onderskeiding wat sy kollegas hom kan gee.

Jl. S. Afr. vet. Ass. 47(2) 149-150 1976

In Memoriam

JOSEF REMEGIUS SCHEUBER
1892 — 8.12.75

Born in Stans, Switzerland, he studied Veterinary Medicine in Zürich, passed his Staatsexamen in 1918 and obtained his doctorate (Dr Med. Vet.) in 1920. Along with other young Swiss graduates he was interviewed by Sir Arnold Theiler and proceeded to South Africa. He joined Dr E.M. Robinson in the Alberton Laboratory in 1921 and accompanied him to Onderstepoort in 1923. He retired in 1952.

He married Marie Hufnagel in 1926. Mrs Scheuber was killed in a motor accident in 1931, a tragedy that left its mark on him for the rest of his life.

With Dr Robinson, he worked on bacteriological problems and lectured to students mainly in 'practicals'. His love was for the clostridial anaerobes mainly *Clostridium chauvoei* and *Cl. welchii*, type B. He prepared many thousands of litres of black quarter and lamb dysentery vaccines and was thus responsible for preventing the untimely death of a very large number of cattle and sheep.

Dr Scheuber was no mean linguist. He could speak and write English, Afrikaans, German, French, Italian, Portuguese and Latin. In his younger days he played a very good game of chess and in later years was more than able to hold his own at contract bridge.

His funeral service was conducted by his son, Father R.A. Scheuber, O.M.I. and was attended by his daughter, Sister M. Bernadine, O.S.U. and his sisters, Rosli and Fineli. Many veterinarians, mostly his old students, were there to pay their last respects.

We offer our sincere sympathy to his son, daughter and sisters in South Africa and to his relatives overseas.

CARL VON EBERHARDT MARÉ
18.4.1894 - 26.5.1974

Charles, soos hy bekend was, is in die distrik Pietersburg ebore. Sy hoërskoolopleiding is te Stellenbosch voltooi en mstreeks 1919 behaal hy die graad B Sc aan die Univeriteit aldaar.

Met die totstandkoming van die Fakulteit Veeartsenyunde in 1920 by die destydse "Transvaal University 'ollege (T.U.C.) of the University of South Africa", skryf hy ir hierdie kursus in en teen einde 1924 was hy met ses tydenote die eerste om die graad B V Sc in Suid-Afrika te beaal. Sir Arnold Theiler, destyds nog besonder aktief as oorrangse navorser, was hulle uitstaande leermeester.

Hierna word hy Staatsveearts te Mafeking en Middel urg Tvl en later weer Mafeking. Met uitsondering van nkele jare word bykans al sy diensjare in laasgenoemde rensdistrik deurgebring. Die hoogtepunte van sy bydrae as die bekamping van ekonomiesbelangrike veesiektes oos skaapbrandsiekte, perdeskurfte, droes, hondsdolheid, ek-en-klouseer, miltsiekte, besmetlike misgeboorte, knopelsiekte ens.

Na bereiking van die ouderdomsgrens, was hy vir enkele are tydelike Staatsveearts te Hoopstad. Daarna verhuis hy eer na Mafeking waar hy hom besig hou met 'n beperkte rivaat praktyk.

Aan sy oorlewende dogter en twee seuns gaan die innige eegevoel van die professie.

In Memoriam

THOMAS VEENSTRA
8.5.1923 - 20.12.1974

Thomas Veenstra, of "Tom" soos hy by sy gesin, baie vriende en kennis se (beronisse) bekend was, het sy skool-opleiding te Greys' Kollege, Bloemfontein in 1940 voltooi. Nadat hy in 1947 die graad B V Sc aan die Fakulteit Vee-artsenykunde van die Universiteit Pretoria verwerf het, het hy by die Afdeling Veeartsenydiens van die Departement Landbou diens aanvaar. Sy eerste pos was die van Staats-veearts te Dundee.

In 1952 het hy die Diens verlaat om 'n betrekking by die Gesondheidsdepartement van die Stadsraad van Pretoria as Stadsveearts in beheer van melkhigiëne te aanvaar.

In 1955 het hy egter weer na die Staatsdiens teruggekeer Hy het met groot sukses en pligsgetrouheid in verskillende sentra in Natal as Staatsveearts opgetree en is daarna na Stellenbosch, Louis Trichardt en weer na Pretoria ver-plaas.

In 1969 word hy bevorder tot Assistent-Direkteur van Veeartsenydienste te Stellenbosch en in 1972 tot Adjunk Direkteur van Veeartsenydienste, Suid-Wes Afrika waar hy tot sy dood in Windhoek werksaam was.

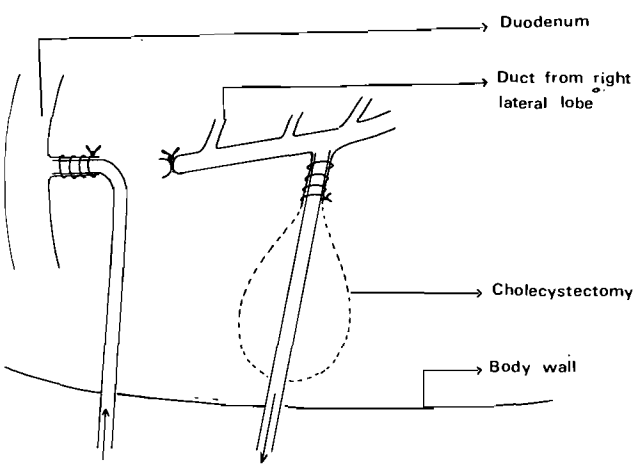
Tom was 'n minsame, vriendelike en hulpvaardige mens en hierdie eienskappe is ook deurgedra tot in sy amptelike en professionele optredes. Gepaard met sy wye onder-vinding, kundigheid en werksvermoë was hierdie eien-skappe dan ook verantwoordelik vir sy vordering in die Diens. Sy skielike heengaan was vir sy talle vriende en kollegas 'n groot skok en vir die professie 'n gevoelige ver-lies.

Aan sy vrou Stella (gebore de Jager) en hulle seun en dog-ter gaan ons innige meegevoel.

CORRIGENDUM

REGSTELLING

"The Choleretic Action of Genebile in a Dog" Vol. 46 No. 2, p. 149 — A. IMMELMAN, C.J. ROOS and N.C. OWEN: The corrected figures are as follows:



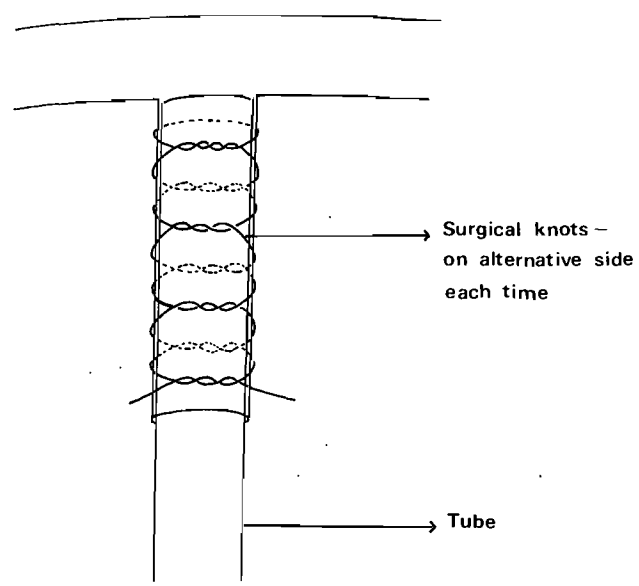
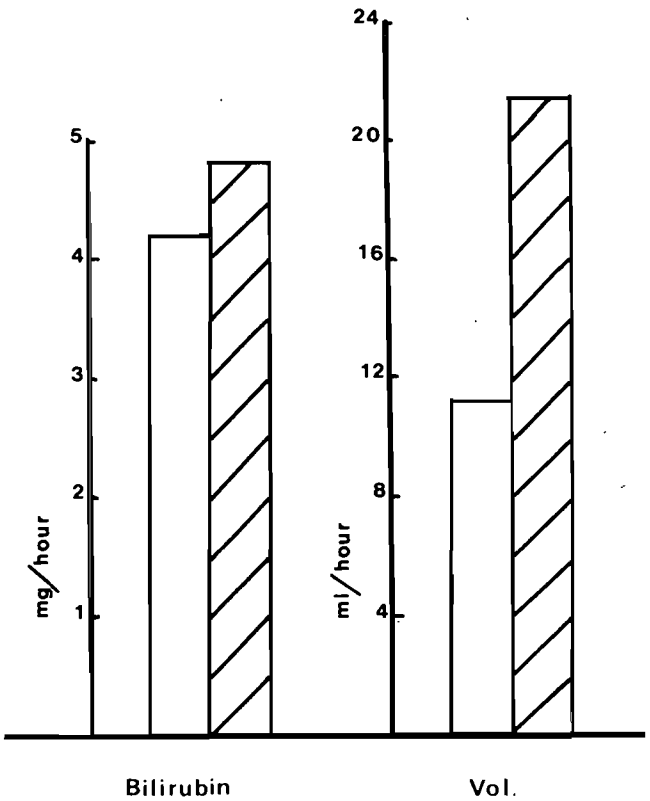
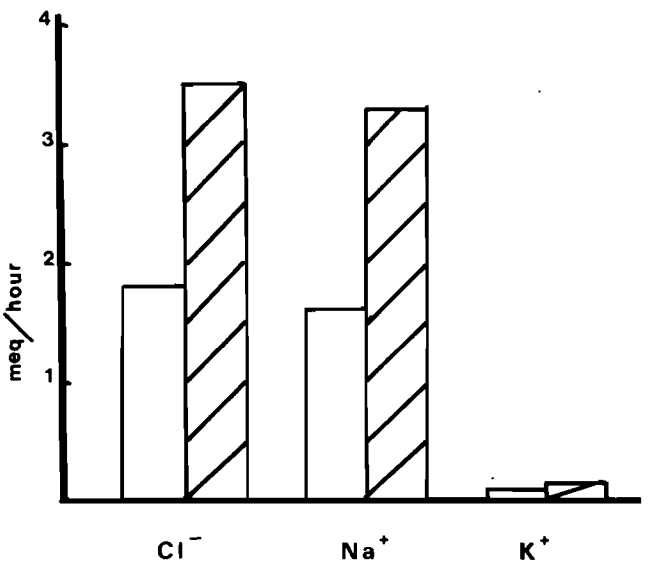


Fig. 1 Method used to secure artificial tubes in ducts.



The effect of treatment on bile composition and flow rate.



The effect of treatment on the excretion of electrolytes into bile.

Fig. 2 The height of each column represents the mean value for the three trials.

□ Before treatment
▨ After treatment.

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