THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

THE PAPERS

To be read before the

FIFTY-EIGHTH ANNUAL CONGRESS

SEPTEMBER 24th — 27th, 1963

are contained in this Number and in the June, 1963, issue [Vol. XXXIV (2)]

DIE REFERATE

Gelewer te word voor die

AGT-EN-VYFTIGSTE JAARKONGRES

24 — 27 SEPTEMBER 1963

kom in hierdie nommer en in die Junie 1963 uitgawe voor [Jrg. XXXIV (2)]

DIE SUID-AFRIKAANSE VETERINÊR-MEDIESE VERENIGING

PROGRAMME

SCIENTIFIC CONGRESS AND FIFTY-EIGHTH ANNUAL GENERAL MEETING

FACULTY BUILDINGS, ONDERSTEPOORT

24–27 SEPTEMBER, 1963

The papers referred to in the programme appear in this issue and in Vol. XXXIV(2)—June, 1963

TUESDAY, 24 SEPTEMBER, 1963

8.00 a.m.	Registration.
9.00 a.m.	Convocation.
9.05 a.m.	Welcome to Guests and Delegates by Prof. B. C. Jansen, Chief, Veterinary Research Institute.
9.15 a.m.	Opening of Congress by the Hon. Dr. H. F. Verwoerd, Prime Minister of the Republic.
	Expression of thanks to the Prime Minister by the President, Dr. H. P. Steyn.
9.45 a.m.	Presidential Address.
10.00 a.m.	Opening of Trade Exhibits by the Vice President, Prof. R. Clark.

TEA

11.15 a.m.	Address by Dr. Geo. van der Wath, Chairman of the South African Wool Board.
11.30 a.m.–4.30 p.m.	SYMPOSIUM — The Changing Role of the Veterinarian in the Modern World. *Organiser, H. P. de Boom.*
11.30 a.m.	Veterinary Research — B. C. Jansen.
12.00 noon	The Role of State Services in Animal Disease Control and Health Promotion — M. C. Lambrechts.
12.30 p.m.	Veterinary Practice in South Africa — J. G. Boswell.

LUNCH

2.00 p.m.	The Veterinarians Role in Tomorrow's Industrialised production of Livestock — I. van Schalkwyk.
2.30 p.m.	The Veterinarian in the Pharmaceutical Industry — J. D. Poole.
3.00 p.m.	The Veterinarian in Public Health Services — B. M. Horwitz.
3.30 p.m.	Veterinary Education — R. M. du Toit.
4.00 p.m.	The Role of the Veterinarian in Wildlife Conservation and Preservation — M. J. N. Meeser.
4.30 p.m.	Review of Essential Features of Symposium — H. P. de Boom.

8.00 a.m. Registration.
8.30 a.m.–12.30 p.m. *FIFTY-EIGHTH ANNUAL GENERAL MEETING.* (Films — if time permits).

AGENDA

1. Notice of meeting and confirmation of the minutes of the Fifty-Seventh Annual General Meeting (*published on page* 535 *of the December*, 1962 *issue of J.S. Afr. vet. med. Ass.*)

2. Matters arising from the minutes of the previous meeting:

   (i) Resolutions from 57th A.G.M.

   (*a*) *Besluit No.* 1

   Ten spyte van die feit dat siektebestryding in hierdie land met groot welslae tot dusver uitgevoer is, voel die 57ste Jaarkongres van die S.A.V.M.V. dat die noodsaaklikheid van die opleiding van groter getalle veeartse nodig geword het. Die owerhede word beleefd versoek om die noodsaaklikheid van die stigting van 'n tweede fakulteit deeglik te oorweeg.

   (*b*) *Besluit No.* 2

   Die 57ste Jaarkongres van die S.A.V.M.V. is van oordeel dat ten spyte van uitnemende veterinêre prestasies van die verlede daar uitgebreide onkunde aangaande die professie bestaan en dat die professie gevolglik nie na waarde waardeer word nie.

   Hierdie Kongres versoek dus die Raad S.A.V.M.V. om grondige ondersoek in te stel na moontlikhede wat alle beskikbare publisiteitsmedia bied en die te benut om, as deurlopende beleid die professionele beeld onder alle bevolkingsgroepe reg te stel.

   (*c*) *Resolution No.* 3

   This 57th Annual General Meeting of the S.A.V.M.A. hereby resolves to request the Council of the S.A.V.M.A. to set up a Sub-Committee consisting of members of the Department of Agricultural Technical Services and Private Practitioners to devise satisfactory means for the inauguration of Diagnostic Centres.

   (*d*) *Resolution No.* 4

   It is the considered opinion of the representative body of Veterinarians attending the 57th Annual General Meeting of the

S.A.V.M.A. that the use of the title M.R.C.V.S. on name-plates or prescription pads is not permissible in the Republic of South Africa when it has been obtained as a result of the payment of a fee solely for the purpose of admission to the Register of the College. When admission to that register results from passing a prescribed examination carried out by the Royal College duly constituted as an examining body, use of this title in the Republic is permissible.

(*e*) *Besluit No.* 5

Wat betref die optrede van veeartse in kommersiële ondernemings, maak die professionele gedragskode voorsiening vir die beoefening van hulle beroep in ooreenstemming met die belange van alle ander lede van die professie. Die 57ste Jaarvergadering van die S.A.V.M.V. besluit dat die professionele gedragskode in gees en letter van toepassing is op veeartse in kommersiële ondernemings in sover hulle ook geregistreer is onder Wet No. 16 van 1933.

3. Report by the President on the activities of the Association during the year.

4. Amendments to the Veterinary Act (Act No. 16 of 1933).

5. **Membership**
   (*a*) Deaths
   (*b*) Resignations and terminations of membership
   (*c*) Election of New Members
   (*d*) Creation of Honorary Life President

6. **Consideration of Reports**
   (*a*) Income and Expenditure a/c and Balance Sheet
   (*b*) Reports of Standing Committees

7. World Veterinary Association (*Animal Health Year*)

8. Publication of the Journal of the S.A.V.M.A.

9. Employed Veterinarians and amplification of the Guide to Professional Etiquette

10. Current Committees of Investigation

11. Notification of election of Office-bearers for the year 1963/64.

12. General

13. Preparation and arrangements for the 1964 Congress at Cape Town.

14. Resolutions

15. Adjournment

2.00 p.m. Group Meetings
Visits to Trade Exhibits

THURSDAY, 26 SEPTEMBER, 1963

8.00 a.m.–11.45 a.m. SYMPOSIUM — Geriatrics in Small Animals
Organiser Campbell Dickson

8.30 a.m. Cardio-vascular changes in the Ageing Dog—Campbell Dickson

9.00 a.m. Eye conditions in Aged Dogs — J. L. Doré.

9.30 a.m. Skin Diseases in the Ageing Dog and Cat—J. F. Brownlie

10.00 a.m. The Urinary System in the Aged Dog — W. E. J. Warnes

TEA

11.00 a.m. Some conditions requiring Surgery in Aged Dogs — M. E. Keep

11.30 a.m. Surgical aspects of the Dog and Cat in old Age — E. M. Hearn

12.00 noon Review of Essential Features of Symposium — Campbell Dickson

12.15 p.m. Film and Address . . .
S.A. Guide Dogs Association

FRIDAY, 27 SEPTEMBER, 1963

8.00 a.m. Registration

8.30 a.m.–11.45 a.m. SYMPOSIUM — Problems Associated with the High Producing Dairy Cow — *Organiser I. S. McFarlane*

8.30 a.m. The Breeder of the High Producing Dairy Cow — D. Beal-Preston

9.00 a.m. Some Physiological aspects of lactation and the nutritional requirements of the High Producing Dairy Cow — J. F. W. Grosskopf

9.30 a.m. Some metabolic disturbances associated with the High Producing Dairy Cow — K. van der Walt

10.00 a.m. The Influence of Induced High Production on Reproductive Efficiency — S. W. J. van Rensburg and A. B. la Grange

TEA

11.00 a.m.	The Udder in High Production — L. W. van den Heever
11.30 a.m.	Review of Essential Features of Symposium — I. S. McFarlane
12.00 noon	Address by Prof. Bonadonna of the University of Milan, Italy.

LUNCH

2.00 p.m.	Immunity to *Staphylococcus aureus* (Rosenbach 1884) — C. M. Cameron
2.45 p.m.	A Modern approach to the Control of Bovine Tuberculosis — H. H. Kleeberg
3.30 p.m.	Immobilizing Drugs used in the capture of Wild Animals in the Kruger National Park — J. W. van Niekerk
4.00 p.m.	Resolutions
4.15 p.m.	Adjournment

SOCIAL ARRANGEMENTS

MONDAY, 23 SEPTEMBER, 1963

6.00 p.m. onwards	Get together at Continental Hotel

TUESDAY, 24 SEPTEMBER, 1963

8.00 p.m.	Theiler Memorial Lecture — Prof. Rimington, University of Pretoria

THURSDAY, 25 SEPTEMBER, 1963

2.00 p.m.	Sport at Country Club/or visit to Pretoria Zoo.
8.00 p.m.	Buffet-Supper at Country Club

EXCURSIONS FOR LADIES

WEDNESDAY, 25 SEPTEMBER, 1963

Visit to Braaks Flower Farm

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EDITORIAL

CYTOPSYCHOSES

Adaptation to changing conditions through compensatory reactions is a basic concept in physiology. When faced with potentially dangerous alterations in their environment, the body cells initiate nervous or hormonal reactions calculated to mitigate or counteract the impending threat. The pendulum of homeostasis usually swings between moderate over and under compensation.

At a recent meeting of the New York Academy of Sciences, Professor Drabkin propounded the intriguing theory that the cells may exhibit phobias which cause them to react with such panic-stricken violence

as to cause overcompensatory imbalances more dangerous than the object of their irrational fears.

New concepts demand new terms. A common cytopsychosis appears to be hypoglycaemophobia. The normal reaction to a drop in blood sugar is a reduction in insulin secretion which reduces the utilization of glucose by the cells. To reduce consumption in times of shortage is logical, but the phobia may cause such an exaggerated response as to deny even the available glucose to the cells, much as a miser will starve rather than

deplete the hoard under his bed. It may also stimulate the production of ketone bodies far beyond the capacity of the cells to utilize them, thus producing a dangerous ketonaemic acidosis.

At high altitudes, anoxophobia may produce serious hyperventilation acapnia. Rather than tolerate a suboptimal oxygen supply, the psychopathic cells elect to die from lack of carbon dioxide.

Physiology and biochemistry are progressing more and more to the cellular and sub-cellular levels. Histochemistry and mitochondrial enzymology may soon be joined by cytopsychiatry.

R.C.

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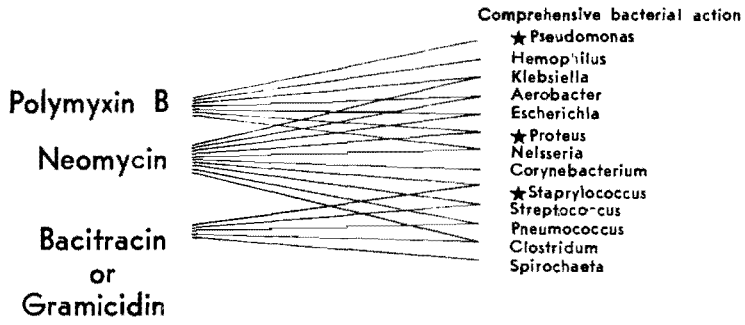

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REDAKSIONEEL

SKRIPTOPENIE EN SKRIPTOFOBIE

Ten spyte van die algemene klagte oor die fenomenaal toenemende volume wetenskaplike lektuur, bly dit 'n betreurenswaardige feit dat op veeartsenykundige gebied as sodanig daar vandag nog steeds eerder 'n „skriptopenie" heers. Veral in die meer basiese vakrigtings is daar nypende tekorte. So is daar op die huidige oomblik geen enkele, volledige, deeglik omvattende handboek oor die anatomie van ons huisdiere in lopende druk nie. (Daar het weliswaar twee bande van 'n reeks van drie in Duits verskyn, om die plek van die welbekende hoeksteen „Ellenberger en Baum" in te neem, wat in die jongste oorlogspuin verdwyn het.) Nog hagliker is die toestand wat gespesialiseerde naslaanwerke betref.

Dis 'n paradoksale toestand, maar basiese inligting aangaande diere, waarmee soveel geredeliker geëksperimenteer kan word, is baie moeiliker bekombaar as inligting aangaande die mens. Hierdie verskynsel skep die indruk dat veeartsenykundiges aan 'n ware „skriptofobie" ly. As 'n mens egter die relatiewe getalsterktes van diergeneeskundiges met bv. dié van medici vergelyk, dan kan daar geen summiere diagnose gestel word nie. Veral opvallend is die klein absolute getal veterinêre navorsers en akademici, die mense van wie literatuurproduksie eintlik verwag word.

Dit bly 'n feit dat die mens se aandag, en dus die geredelikheid waarmee hy fondse, fasiliteite en sy energie beskikbaar stel, in die eerste plek deur selfsugtige motiewe bepaal word, en dus toegespits word op dié aspekte waaruit hy onmiddellike voordeel kan behaal of waardeur hy die vir hom dreigendste onheil kan afweer. Dis ook die gebied wat sy verbeelding die sterkste aangryp. Van die twee dryfvere is laasgenoemde die sterkste. Dis dus doodnatuurlik dat van die geneeskundige rigtings, die menslike heelkunde 'n baie hoër populêre status geniet en 'n baie sterker aantrekkingskrag uitoefen op die intelligente en verbeeldingryke jong adolessent, wat die kern vorm van potensiële mannekrag.

Uit die beperktheid van mannekrag spruit die omstandigheid dat elke veeartsenykundige swaar belaai is met werk van onmiddellike noodsaaklikheid, 'n toedrag van sake wat nie juis bevorderlik is vir die tydrowende opstel van geskrifte nie, veral van dié van meer omvattende teksboek- of handboekaard, nog van die gespesialiseerde naslaanwerke. Dit laat ook nie 'n deurtastende opspoor van die literatuur toe nie, wat uit die aard van die saak gehaal moet word uit tydskrifte van wyer uiteenlopende rigtings as vir enige ander vakgebied. Die beperktheid van afsetgebied strem ook die publikasie van werke, bloot omdat dit veelal onekonomies blyk.

Die populêre opvattings omtrent die aard van diergeneeskundige praktyk en werksaamhede is sodanig, dat ons beroep eerder die prakties aangelegde man trek. Daarmee is niks verkeerd nie, maar ongelukkig

verloor ons daardeur potensieel waardevolle mannekrag met meer akademies-skolastiese aanleg. Om dus die gebrek aan te vul, sal ons as beroep meer moet doen om ook hierdie laasgenoemde groep sterker aan te trek. Daarby alleen kan dit nie bly nie: ons sal moet voorsien vir deegliker deurkneding in die basiese vakke, nl. biochemie, biofisika, anatomie en fisiologie, asook in navorsingsprosedures en tegnieke, hetsy voorgraads of nagraads.

'n Bykomstige manier om op langtermyngrondslag die „skriptopenië" te besweer, en nie allermins navorsing te bevorder nie, is om ons te beywer vir opleiding van wetenskaplik-tegnies onderlegde dokumentaliste, i.p.v. ons bloot te verlaat op bibliotekarisse met slegs letterkundige agtergrond, hoe geïnteresseer en behulpsaam hulle ookal mag wees. (Dit is ontstellend as mens dink dat die taak van bibliotekaris in 'n groot instituut van wêreldfaam vir jare op die skouers van 'n tydelike tikster gerus het. Alle eer gaan aan die persoon wat dit sonder opleiding so lank deur intensiewe toewyding en ywer teen karige vergoeding kon klaarspeel!) Dit help nie om te roep om sodanige personeel nie; ons sal uit eie kragte die gespesialiseerde opleiding aan aspirant-bibliotekarisse moet verskaf, en daardeur self meer te wete kom.

Daar is 'n ander aspek wat aandag verdien. Dit tref 'n mens by geleenthede van toevallige ontmoetings — tydens kongresse, e.d.m. — dat diegene wat in die branding staan, naamlik praktisyns, staats- en munisipale veeartse, e.a., dikwels oor nuttige inligting o.g.v. eie — soms baie bittere — ervaring beskik, maar tewens behep skyn te wees met 'n ware „skriptofobie". Die gedagte word skynbaar gehuldig dat manne van daad nie manne van die pen kan wees nie. Skrywery sou óf bokant hul vuurmaakplek óf benede hul waardigheid wees. En die redakteur word 'n soort boeman, wie se rooi pen die vaardigheid besit om die bloed van baie ego's te laat vloei. Eerder om jou daaraan bloot te stel, bly maar liewers stil, so word skynbaar geredeneer.

Hier moet 'n lansie vir die redaksie van ons blad gebreek word. Die lede van die redaksiekomitee moet aan die een kant weliswaar 'n kritiese houding inneem, teneinde die tydskrif op peil te hou en die peil steeds op te skuif. Aan die ander kant besef min mense die ure en ure, ja, dae, van vrywillige werk om geduldig geskrifte, dikwels haastig neergeklakte geskrifte, na te gaan en kollega's met advies en daad by te staan. Om opbouende kritiek te lewer is 'n fundamentele beleid, omdat die moeilike omstandighede, waaronder so baie kollega's iets op papier probeer stel, terdeë besef word. Baie aspirantskrywers voel diep gekrenk of hewig ontstel as hulle 'n manuskrip terugkry wat van hoek tot kant uitmekaargeskeur en bekrap is, in plaas van te besef dat, eerstens, nie almal taalkundige helde kan wees nie, en dat, tweedens, dit gedoen is in 'n eerlike poging om behulpsaam te wees.

Van hul kant kan kollega's grotendeels die las op die redaksie se skouers verlig deur hul skryfvermoë te verbeter, nl. deur kritiese leeswerk, en deur 'n bietjie verbeelding en gesonde verstand en oordeelkundigheid aan die dag te lê. Gebruik van tegniese en gewone woordeboeke is ook geensins te versmaai nie. Maar bowenal, moenie toelaat dat vrees vir

openbaring van onkunde of vir blootstelling aan die redaksionele in-bloed-en-gal-gedoopte pen u weerhou om potensieel nuttige inligting vir ander persone beskikbaar te stel nie, of om probleme en kwel- en knelpunte van ons beroep aan die lig te bring en openhartiglik te bespreek nie.

Hierdie tydskrif word nie ter wille van vertoon gepubliseer nie. Dit behoort die slagaar van die beroep te wees, waardeur intellektuele ischemie aktief verhoed kan word: 'n aktiewe teenmiddel teen die drukatrofie van alledaagse omstandighede.

Ten slotte 'n beroep op die onderwysmanne in ons midde: skenk asseblief 'n bietjie meer aandag aan die opstel en skrywe van verslae en artikels deur studente, met opbouende kritiek, voorligting en aanmoediging.

H. P. A. De B

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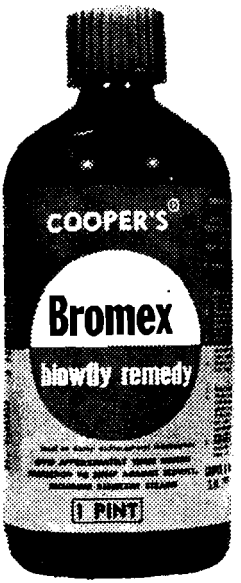

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SYMPOSIUM THE CHANGING RÔLE OF THE VETERINARIAN IN THE MODERN WORLD

THE VETERINARIAN IN THE PHARMACEUTICAL INDUSTRY

J. D. H. POOLE — P.O. Box 7552, Johannesburg

Received for publication, May 1963

INTRODUCTION

The primary intention in this paper is to describe the rôle played by the profession in the Pharmaceutical Industry. From the limited information on the subject, the present status of veterinarians in the Industry will be described and an attempt will be made to predict future trends in this avenue of employment.

The importance of this industry to the profession is obvious; the magnitude of the industry itself is illustrated by the fact that in America alone some 13,500 research scientists are employed by it.[1]

In an era of rapid expansion of veterinary professional responsibilities and activities, it is only natural that this important industry should receive some of our attention. Modern trends in veterinary medicine have brought about great complexity in the part it has to play. In the past, it was a fairly general rule that the veterinary side of the pharmaceutical industry existed on hand-downs from the human pharmaceutical divisions of the various companies. The importance of veterinary divisions of some of these companies in the modern world is such, that most of them now have independent activities in the fields of basic research, chemical screening and field trials.

SERVICES RENDERED TO THE INDUSTRY BY VETERINARIANS

There are many facets in the functioning of the pharmaceutical industry in which veterinarians play an important part. For the sake of clarity, these functions are grouped under three main headings, namely, Research, Production and Advisory Services.

RESEARCH

Under this heading have been grouped all functions of veterinarians in the industry which have some connection with research, although not all these functions involve the actual carrying out of research programmes.

* To be presented at the forth coming scientific conference of the Association at Onderstepoort from 24-27 September, 1963.

Basic Research

Basic research is becoming a primary function of the industry; the increasing contributions to basic research by scientists of the industry is an indication that more and more importance is being placed on this valuable aspect of the industry's development. That the bulk of this research is directed toward some kind of product development is indisputable, but it is also true that this is not the only end-result of these activities. Research activities into production methods have been particularly helpful in respect of the evolution and production of biological products, but many valuable contributions have been made in other fields.

Chance discovery of useful drugs or biological products is a thing of the past. The appearance of new drugs and vaccines directly from the industry itself is generally the result of planned research on the part of scientists using the extensive and modern facilities put at their disposal by the large pharmaceutical companies. Research by the industry increases every year as evidenced by the fact that the amount of money spent by the industry on research in Great Britain increases by 20 per cent each year; in America there was a five-fold increase from 1960 to 1961.[2]

Screening New Compounds

Extensive screening of new compounds for a very large variety of uses, is an essential part of the functioning of the industry. In the United States of America in 1958, the latest year for which accurate figures could be found, 114,000 substances were tested, from which a handful emerged for marketing[1].

Screening for specifically veterinary uses is increasing, and veterinarians, because of their specialised knowledge of the problems involved, are doing this work. Due to modern advances in communications and better international liaison, screening is often carried out, not only for uses involving world-wide problems, but also for uses in specific local problems. Specialised local knowledge is often essential for this type of work and veterinarians with this local knowledge and experience are being used more and more for this purpose. The development of screening methods for specific actions is sometimes a highly specialised undertaking.

Pharmacology and Toxicology

Specialist knowledge in these disciplines of veterinary science is essential in initial toxicological and pharmacological studies on promising chemicals. It is in this state of the evolution of a product that many of them fall by the wayside, due to the discovery of some untoward side effect. For obvious reasons, responsibility for this function requires personnel well versed in veterinary science.

Product Development

From the basic chemical showing some or other useful activity, a product must be formulated, which can be applied under practical conditions. Product formulation requirements differ tremendously from one

country to another, and here again local knowledge is of importance. Formulation of a product from a promising chemical is rarely a simple procedure: co-operation between veterinarians and formulation chemists is usually essential if a convenient and useful product is to emerge.

Field Evaluation

Promising chemical and biological products which result from screening and research activities must be tested in the field. Extensive use is made of veterinarians for this purpose throughout the world. The modern tendency is to employ more veterinarians for this purpose rather than to expose the private practitioner to the risks involved.

In this connection, increasing importance is being placed on trials on a more or less world-wide basis in order to ensure efficacy and safety under a variety of conditions. Experienced local veterinarians carrying out these field trials also advise on possible formulation and packaging changes because these often have to be specifically tailored to meet local requirements.

Research Liaison

This function of veterinarians employed in industry involves communication and personal contact with researchers in other institutions, with a view to generating new ideas and discussing common problems.

Research Management

All facets of research management should be carried out by experienced scientists with knowledge of the scientific problems involved in research and also an intimate knowledge of the requirements of the pharmaceutical industry. This is a field of activity in which many veterinarians are employed today.

Production

In the production of veterinary vaccines and other biological products, trained veterinary virologists and bacteriologists have always played an important rôle. They continue to do so in increasing numbers, due partly to the increased importance of preventive medicine necessitated by modern agricultural practices.

Advisory Services

Technical guidance to sales activities is an important function of veterinarians in the industry. In South Africa this advisory function has in the past, been confined to instruction of sales representatives in

the rudimentary technical aspects of the products to be marketed, with advice to the management on marketing methods.

Under this heading it is as well to mention that advisory services by veterinarians employed in the industry in America has been extended to include a function known as "Professional Service". This service constitutes personal contact with private practitioners and other product users, in order to explain in detail the activities and uses of specific products. As the complexity and number of available products increases, this function will become more important.

Other General Responsibilities

Veterinarians have shown their preparedness to assume responsibility in industry. In many instances this responsibility is extended beyond the immediate limits of veterinary science and has, for example, included control of other agricultural products, such as crop protection chemicals. This is a natural and predictable occurrence, because many companies have diversified interests in agriculture as a whole and the veterinarian showing ability often lands in a position of responsibility involving other company activities.

In an industry of such importance to the profession, it is beneficial that veterinarians should take an active part in all aspects of it, including management and marketing. This is indeed the case in many instances.

Numbers of Veterinarians Employed in Industry

The number of veterinarians appointed to posts in the industry is increasing rapidly each year in Great Britain as can be seen in Table II.

Table 1

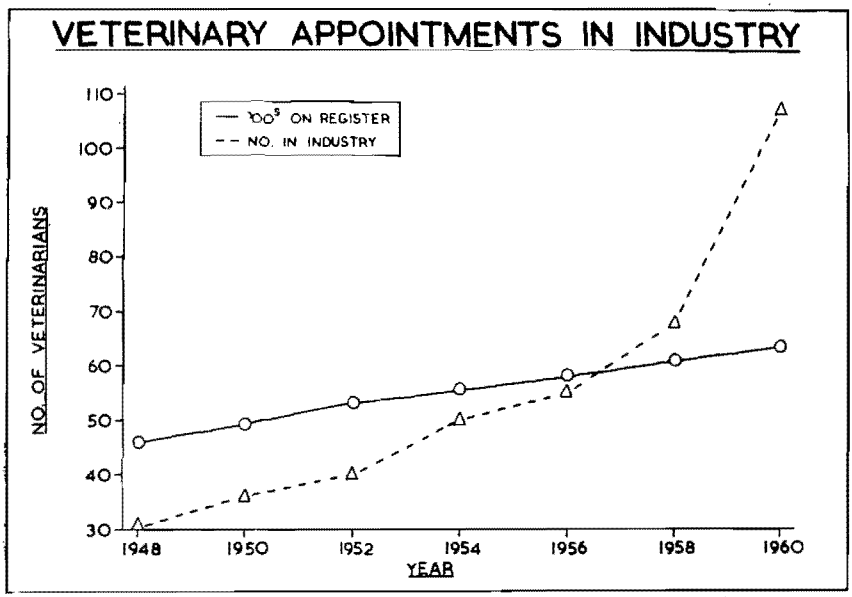
Distribution of Veterinary Appointments in the U.K., 1960

Veterinary Colleges	217
Industry	107
V.I.O. Service	68
Weybridge and Lasswade	57
Agricultural Research Council	27
Animal Health Trust (including Houghton: Excluding fellowships)	25
Moredun	13
Pirbright	13

There is evidence that the same applies in South Africa on a more limited scale, and in other countries.

Some idea of the ratio between industry appointments and other appointments in Great Britain can be gained from the figures in Table I. That the same trend exists in America is almost certain, although accurate figures are not available.

TABLE II



In South Africa 17 veterinarians are employed in the industry, of which seven were appointed during the last four-and-a-half years.

REFERENCES

1. SMITH, AUSTIN, M.D. (1962). At the 19th Annual Meeting of Am. Med. Writers' Assoc., Washington, D.C. Oct. 12.
2. BOGUE, J. Y. (1963). In an address given to the Scientific meeting of the Pharmaceutical Society of Great Britain. Jan. 10.

*SYMPOSIUM: PROBLEMS ASSOCIATED WITH THE HIGH PRODUCING DAIRY COW

PROMOTION OF HEALTH OF THE DAIRY HERD

D. BEAL-PRESTON — President of The Friesland Breeders Association of S.A., Bloemfontein

I have been asked to discuss the promotion of health in the dairy herd from the practical point of view of the pedigree breeder.

In order to do this I think I should begin with a brief outline of the history of the Friesland Breed.

Prior to 1750 Friesland cattle in Holland were red and white and even in those days were known for their milking ability. Due to an epizootic however, more than half the cattle population died.

It then became necessary to import cattle in order to preserve the industry. Large numbers of a smaller black type of cow, also with good milking qualities, were imported from Jutland. Black being dominant over red, the resultant progeny became black and white and this subsequently became the fashionable colouring and was accepted as the official colour marking when the Herd Book was established in 1897.

One of the main objects of the Herd Book was to promote the breeding of good production dairy cows. At the same time, beef was encouraged on those parts of the body which did not affect milk production. The result of that policy which has been maintained through the years, has been a large-framed high-producing type of cow which is capable of making a heavy carcase when slaughtered.

Scientists tend to criticise the pedigree breeder for his insistence on conformation as well as production. The scientist works on statistics and tends to rely on peak selection for production only. This method is seldom entirely successful because breeding cannot be reduced to such a simple production equation. It is essential for success to maintain a balance between conformation and production.

Actually the master breeder is himself a scientist within the limits of his own knowledge, for he carefully investigates all aspects of an animal's pedigree before using that animal in his herd for breeding purposes.

Where the breeder differs from the geneticist is that his approach to the problem is a practical one, dictated by economic necessity and the desire to build up a fine pedigree herd. Science has the ability to assess and collate the objective factors of both the individual and the herd, but it takes the master breeder to select and use the individual animal in his art of breeding.

With the development of the production capabilities of the modern dairy cow, have come tremendous problems for the pedigree breeder and this is where the veterinarian is of invaluable assistance to the farmer. I

* To be presented at the forth coming scientific conference of the Association at Onderstepoort from 24–27 September, 1963.

am grateful for this opportunity which has been afforded me to discuss certain of these problems with the veterinary experts present today. I should like to put certain questions which I hope will stimulate discussion and produce answers which will benefit breeders in this country.

Reproduction

The most important single factor in breeding is of course reproduction which ideally should be natural, regular and without difficulties. The opinion seems to be fairly general, however, that venereal diseases in cattle are on the increase. This is of great concern to the breeder and one wonders whether susceptibility to these diseases might be induced or increased by the feeding of high concentrates. Are we endangering the health and fertility of our herds by such feeding? In other words, how is a cow's reproduction potential affected by the food she is given?

Another disturbing factor is that the fertility of a cow tends to drop as her production increases. Are we not, in our enthusiasm for high records, allowing production to overbalance reproduction in our herds?

Experience has taught me that running a bull in a camp with the females gives a significantly increased calf crop compared to hand service or artificial insemination. What is the reason for this? Do not artificial procedures tend to increase functional disturbances in the cow, due to placing her under abnormal stresses during the oestrous period? This would appear to be the case in overseas countries where A.I. is extensively used, and breeders would be interested to know what scientific investigation has revealed in this respect.

We find that young heifers highly fitted for shows tend to be more difficult to get into calf than the heifers on a lower plane of nutrition. Why is this? This type of feeding differs from that used for milk production in that it has a much lower protein content.

Can veterinary science explain why, in a well cared for herd, there are still cows which suffer from retained corpus luteum, cystic ovaries and retained afterbirth?

These are some of the reproduction problems with which a pedigree breeder is faced.

Now I would like to put before you some of the problems connected with high milk production.

It is rather difficult for the average farmer to understand and appreciate the physiological aspects of lactation and the nutritional requirements of the high producing cow.

We are interested in the latest developments as far as milk production is concerned and would like to have these explained. We are concerned for instance about the demands we make on the modern dairy cow, in expecting her to produce a calf and a high milk record each year. Would her productive and reproductive life not be extended by reducing the official lactation period from three hundred to, say, two hundred and fifty days?

Scientists continually warn us not to force-feed our cows. It would be interesting to know how one is to gauge whether a cow is being force

fed or not. May I quote the interesting case of the American cow, Carnation Prospect Veeman. When she was not fed for her full production potential, she was a sick animal, but when given correct feeding, she was a healthy, robust cow, and produced an amazing average of ten gallons a day for a whole year. Where does one draw the line between force feeding and feeding for the high production potential which a cow may have inherited, and is there proof that feeding for high production tends to lower resistance to disease.?

We find sometimes with high producing cows, which according to our calculations are receiving all the necessary good hay, concentrates, green feeding, minerals and trace elements, that something suddenly goes wrong. It seems that for no apparent reason, the system becomes upset, the cow goes off her feed, the milk production drops. Why is this?

In the past few years much has been made of trace element deficiencies. It is difficult for the ordinary farmer to decide whether such deficiencies exist on his farm or not. By making up these so-called deficiencies could he not be upsetting his animals and causing the reverse of the good he intended.

For a long time it has been considered that a high producing cow must have a large and capacious udder. In my experience however certain cows have neat medium sized udders apparently lacking in capacity and yet they produce as much if not more than the cows with large udders. What is the explanation for this?

Does obesity in young females have any ill effect on the milk producing tissue of their udders, and does this possible build up of non-milk producing tissue tend to make the udder more susceptible to disease. for example mastitis? Is the feeding of concentrates before calving the cause of udder congestion and should congestion be relieved by preparturient milking?

Should an udder be milked out completely at each milking for it to remain healthy?

There is a strong belief amongst breeders that high protein feeding is a contributary cause of foot rot in cattle. We have always understood that foot rot is prevalent in wet conditions and yet it happens that in the middle of a drought, a severe outbreak of foot rot can occur. What is the latest information on this subject?

With the breeding of high producing cows, comes the problem of susceptibility to mastitis. It would appear that mastitis germs are never quite destroyed by present veterinary treatment.

Can veterinary science tell us what are the contributory causes of mastitis and whether inoculation against mastitis is a sure method of preventing the disease.

For various reasons farmers in South Africa are turning to machine milking. Inefficiency and the fact that an economic unit of cows is larger in this country than it is overseas, are among the reasons for this move.

Udder shape is therefore becoming increasingly important since it is not only desirable that an udder should be a good shape, but actually necessary in order to carry out machine milking successfully. Incidentally,

our personal experience has been that the switch from hand to machine milking improved the whole atmosphere in the milking shed. The cows are now peaceful during milking whereas formerly bad hand milking had upset them. It is generally believed however, that machine milking causes more mastitis than hand milking. In my experience, provided that it is a good machine, this is not so. What is the veterinary opinion?

After all this it may appear that a breeder's life is an unenviable one, but I can assure you that pedigree breeding has tremendous compensations in the fact that is a continual challenge and an absorbing interest.

I should like, on behalf of all breeders to express to the Department of Veterinary Services and to all veterinarians our deep appreciation of the constant battle which they wage in assisting us to maintain the health of our herds.

SYMPOSIUM: PROBLEMS ASSOCIATED WITH THE HIGH PRODUCING DAIRY COW

SOME PHYSIOLOGICAL ASPECTS OF LACTATION AND THE NUTRITIONAL REQUIREMENTS OF THE HIGH PRODUCING DAIRY COW

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The normal function of the mammary gland is to supply the newborn with sufficient nutriments until such time as they can fend for themselves. By genetic selection the milk production of the modern dairy cow far exceeds this physiological purpose and has in fact become highly unphysiological. We are now faced with the problem of how far we can push production while still maintaining the cow in normal health. Some of the physiological and nutritional aspects involved are dealt with in this paper.

HORMONAL CONTROL

Milk is secreted by the epithelial cells of the alveoli in the udder. Some constituents are synthesized in the alveoli cells while others are transferred as such from the blood to the milk through the alveolar epithelium. This synthesis of milk is under the control of prolactin, a hormone secreted by the anterior pituitary. Other hormones also playing an important rôle are: Somatotrophin, thyroxin and the adrenocorticoids. The action of prolactin is also mediated by the levels of oestrogen and progesterone.

The milk so formed collects in the alveoli, the milk ducts and the cisterns of the udder and is expelled by the active contractions of the myoepithelium of the udder in response to the so-called "let-down" reflex. This reflex is naturally initiated by pleasant tactile stimulation of the teats. From there nervous impulses are carried to the hypothalamus where the hormone oxytocin is secreted. This hormone is then carried transneurally to the posterior lobe of the pituitary where it is stored and from where it is released into the blood-stream. On reaching the udder via the blood, oxytocin stimulates the myoepithelium to contract and in that way the milk is expelled. The effect of this neuro-hormonal reflex is counteracted by adrenalin.

The milk expulsion reflex plays an important part in the maintenance of milk secretion as well. The secretion of prolactin, the lactogenic hormone, is stimulated by oxytocin during each milking period and continuous production is therefore dependent on this reflex.

The reflex is also conditioned by good stable routine which plays a very important part in dairy farming.

Comparative Composition of Milk and Blood Plasma

Table 1

The percentage of some milk constituents in blood plasma and milk[1]

Constituents	Percentage in plasma	Percentage in milk*
Water	91.0	87.0
Protein—		
Total	7.6	3.5
Casein	—	2.9
Albumin	3.2	0.52
Globulin	4.4	0.05
Lactose	—	4.9
Milk fat	—	3.6
Calcium	0.005 (diffusible)	0.12
Phosphorus	0.005 (inorganic)	0.10
Potassium	0.03	0.15
Sodium	0.34	0.05
Chloride	0.35	0.11
Citrate	Traces	0.20
Magnesium	0.002	0.01

* Breed and individual differences occur.

Various trace minerals and vitamins are also found in milk, the levels depend mainly on their presence and levels in blood plasma.

The Precursors of the Various Milk Constituents

Proteins

Milk protein consists of various protein fractions of which the most important are[2]:

α—Casein	45–63 per cent
β—Casein	19–28 per cent
γ—Casein	3–7 per cent
β—Lactoglobulin	7–12 per cent
α—Lactalbumin	2–5 per cent
Immune globulins	1.4–3.1 per cent
Serum albumin	0.7–1.3 per cent

Of these, all the casein proteins (with the possible exception of γ-casein), the lactalbumin and the lactoglobulin are synthesized in the udder alveoli cells from animo acids obtained from the blood plasma. The other two proteins, the immune globulins and serum albumin, are transferred as such from the plasma to the milk[3].

Milk proteins have a high biological value and contain all the essential amino acids. The following table, calculated from average values as

given by Hansen and Carlson[2], gives the amino acid content of milk (as percentage of total protein).

Alanine	3.3	Lysine	8.4
Arginine	4.1	Methionine	2.7
Aspartic acid	7.9	Phenylalanine	4.8
Cysteine	0.9	Proline	9.0
Glycine	2.5	Serine	6.2
Glutamic acid	21.5	Threonine	5.1
Histidine	2.8	Tryptophan	1.6
Isoleucine	6.1	Tyrosine	6.1
Leucine	9.8	Valine	7.3

A deficiency of protein in the diet will have very little influence on the protein percentage of milk but it will be responsible for lower production. Too low an energy level in the feed, on the other hand, will have a more marked depressing effect on the protein content of milk[23].

Milk fat

The fat in milk differs markedly from all other fats in the body. Butterfat, especially in milk from ruminants, contains a relatively higher proportion of short-chain fatty acids than does body fat. Different species of mammals have different proportions of fatty acids in their milk fat as can be seen from the following table[4]:—

TABLE 2

Proportion of fatty acids in milk fats expressed as mols. per cent.

Species	Saturated fatty acids								Unsaturated fatty acids	
	C_4	C_6	C_8	C_{10}	C_{12}	C_{14}	C_{16}	C_{18}	Oleic	Others
Cow	10	4	1	2	3	11	27	12	21	7
Mare	1	2	4	8	7	7	15	3	37	16
Sow	—	—	2	—	—	2	28.5	6	35	26.5

Proteins, carbohydrates (starch, sugar, cellulose, etc.) and fats in the diet may serve as precursors of milk fat. Some of them e.g. oleic acid, may be incorporated in the milk fat without appreciable chemical change. The majority of fatty acids are however synthesized in the udder. The main precursors of the fatty acids in butterfat in the milk of non-ruminants seems to be blood glucose. In the ruminant, however, a high proportion of the fatty acids in milk fat is derived from acetate in the blood and a minor portion from glucose[5, 6]. Some of the higher fatty acids may also be derived from long-chain fatty acids in the blood. The ratio of fatty acids in the milk remains constant, and if all of them are not available at sufficient levels, the udder will produce less fat without changing the composition.

The main source of short-chain fatty acid esters (e.g. acetate) in the blood of ruminants is from microbial breakdown of cellulose in the

rumen. It is therefore essential that sufficient roughage be included in the ration of dairy cows to maintain the normal fat level in the milk. Various other constituents in the feed which may influence the breakdown of cellulose in the rumen may therefore also have an effect on the butterfat content of the milk. In the Netherlands, fat figures as low as 0.7 per cent have been obtained on copper deficient rations[7].

Lactose

The normal level of lactose in the milk of Friesland cows is approximately 4.9 per cent. It fluctuates very little and cannot be materially changed by feeding. Lactose is synthesized from blood glucose and galactose which is probably formed in the udder from glucose[1]. Sufficient carbohydrates are therefore required to maintain the blood sugar level to allow optimum milk production.

Calcium

Milk contains 0.12 per cent of calcium or 0.54 gm. per pound. It is present in milk as protein-bound calcium e.g. calcium caseinate and in combination with phosphate and citrate. About 25 per cent of the calcium is in true solution[8]. There seems to be evidence that a positive correlation exists between the degree of ionization of calcium in milk and the clotting properties of the milk proteins[9, 10].

The calcium content of milk cannot be changed by feeding different levels of calcium. If sufficient calcium is not available the secretion of milk will be reduced. The calcium is derived from the diffusible calcium in blood. However, at any given time, the total extracellular fluid of the body of a 1,200 lb cow will contain approximately 4 to 5 gm. of diffusible calcium, not even sufficient for 10 pounds of milk. As a drop in the extracellular fluid's calcium level will disturb vital body functions it is obvious that all calcium removed from the blood must be replaced immediately, either from the digestive tract or from the skeleton.

The degree of absorption of calcium from the feed depends on a number of factors such as the presence of oxalates, the calcium/phosphorus ratio, the presence of vitamin D, the presence of phytate and the requirements of the body. Under normal conditions of feeding it may vary from about 10 to 60 per cent of the calcium taken in with the feed, in the dairy cow[11]. The lower levels of absorption are generally found with a high calcium phosphorus/ratio and when the requirements of the body for calcium are low.

Excess calcium is excreted from the body via the intestinal wall, the bile and the urine.

Phosphorus

Milk contains approximately 0.1 per cent of phosphorus or 0.45 gm. per pound of milk. It is present as di- en tri-calcium phosphates and as organically bound phosphorus e.g. phosphoproteins. About 44 per cent of the phosphorus in milk is in true solution[12]. All of it is derived from the blood inorganic phosphorus[13, 14]. Even with drastic changes of the phosphorus level in the feed, the content in milk remains more or less constant. If insufficient phosphorus is available for normal production, less milk will be produced.

Just as calcium in the extracellular fluid at any given time is insufficient for milk production, so is phosphorus. The total phosphorus content of the extracellular fluid of a dairy cow will be sufficient for only 12 pounds of milk. All the phosphorus required for milk production must therefore be obtained either from the digestive tract or from the bones.

The optimum digestibility of phosphorus is between 60 and 70 per cent[15, 16, 17]. Under normal condition of feeding the absorption of phosphorus varies from about 5 to 60 per cent of that ingested with the feed[11]. The degree of absorption depends amongst others on the requirements for the mineral, the calcium/phosphorus ratio and the form in which the mineral is present. A lower percentage of phosphorus is used when the calcium/phosphorus ratio is high and when the requirements for it are low.

An excess of phosphorus is excreted from the body via the kidneys.

The Importance of the Calcium/Phosphorus Ratio

The levels of calcium and phosphorus in the undermentioned fluids or body structures are approximately as follows:—

Table 3

The calcium/phosphorus ratios of bovine extra-cellular fluid, milk, bone and a new born calf

	Ca	P	Ca/P ratio
Extracellular fluid	4–5 mgm. % (diffusible)	4–5 mgm. % (inorganic)	1:1
Milk.....	120 mgm. %	100 mgm. %	1.2 :1
Bone (dry).....	23.05 %	10.22 %	2.25:1
Calf at birth.....	1.25 %	0.68 %	1.84:1

As the extracellular fluid contains very little of these two minerals and while the levels of calcium and phosphorus in it must always be maintained, it can for the purpose of milk production be disregarded other than as an exchange medium. Further it can be seen from the table above that the optimum Ca/P ratio available in the feed for milk production should be approximately 1.2 to 1, while if it is required to make up for a previous loss of calcium and phosphorus from bone during a period of either calcium or phosphorus deficiency, the optimum ratio of calcium to phosphorus should be 2.25 to 1. During late pregnancy a ratio of approximately 1.8 to 1 should be best. Maintenance requirements should be considered as well.

If for example, a lactating cow is fed a diet low in phosphorus but rich in calcium, the phosphorus required for milk production will have to be derived from bone. With every one part of phosphorus withdrawn from the bones, however, 2.25 parts of calcium are withdrawn with it. In the milk, on the other hand, only 1.2 parts of calcium can be used with every one part of phosphorus, thus leaving 1.05 parts of calcium to be excreted from the body. Apart from this loss of calcium from the body, the high level of calcium in the feed is wasted as well.

On the other hand, cows can absorb sufficient phosphorus even at high Ca/P ratios provided that the phosphorus level in the feed will allow a sufficient amount to be absorbed notwithstanding the low percentage of absorption.

Diets low in calcium and rich in phosphates will in the same way cause a withdrawal of calcium and phosphorus from the bones and a waste of a portion of the dietary phosphate.

The calcium and phosphorus levels of the plasma are governed by the parathyroid through regulation of their relative rates of deposition in and withdrawal from the skeleton and their excretion via the kidneys. The parathyroids therefore play a key rôle in maintaining the normal calcium and phosphorus levels of the blood in the heavily lactating cow.

Other electrolytes

Sodium, potassium, chlorine and even magnesium are present in milk in relatively high quantities. These are derived from the blood plasma and must therefore be present in the feed in sufficient amounts to allow for milk production and the maintenance of the plasma levels. Potassium is found in all plants and should not present any problem, but sodium and chlorine should be supplemented. A defiency of sodium or chlorine has a depressing effect on milk production[27]. Magnesium, although only secreted at the rate of 45 mgm. per pound of milk, may at times be deficient.

Trace minerals

The trace minerals found in plasma are also found in milk and their levels depend on the plasma content. Colostrum usually contains higher levels than normal milk. The levels of some trace minerals, e.g. Iodine, cobalt, manganese and molybdenum, in milk may be increased tremendously by increasing their levels in the feed. Others, such as iron and copper, cannot be increased by feeding due to the selective absorption of these minerals in the small intestine.

A deficiency of trace minerals may influence the composition and amount of milk indirectly, but no direct effect, apart from a lower trace mineral level is known.

Vitamins

Vitamin A.—Carotene and vitamin A are found in milk. Jersey and Guernsey milk is yellower than that of other breeds because of the higher carotene content[18]. Friesland milk, on the other hand, contains more vitamin A. The sum of the carotene and vitamin A, however, is approximately the same in the milk of all dairy breeds[19].

Vitamin A and carotene are both derived from the blood plasma and the amounts present in milk are dependent on the blood levels which in turn are influenced by the level of carotene in the feed and the level of vitamin A in the liver. Colostrum contains ten to a hundred times more vitamin A activity than normal milk[20].

Vitamin B complex.—The micro-organisms in the rumen synthesize all the B vitamins required by the cow. In spite of this convenient source, only riboflavin, inositol and pantothenic acid are present in milk in

appreciable quantities[1]. Provided normal rumen function is maintained, the vitamin B content of milk cannot be materially altered by feeding.

Vitamin C.—This vitamin is also synthesized by ruminants and its level in milk can therefore not be influenced by feeding. After withdrawal, milk rapidly loses its vitamin C activity.

Vitamin D.—Cholecalciferol and ergocalciferol are present in milk. Their levels depend on the levels in plasma which in turn is dependent on the exposure of the cow to sunlight.

Vitamin E.—The level of tocopherol in the milk depends on the tocopherol content of the feed. It is usually low during winter. Tocopherol serves as an antioxidant in milk.

Vitamins are essential for the health and normal metabolism of the cow and as such play an indirect rôle in milk production. A vitamin deficiency has no apparent direct effect on the amount of milk produced but less of the deficient vitamin will be present in the milk.

Nutrients Required for Milk Production

For the purpose of this paper the following cow will be used as an example: Adult Friesland cow weighing 1,200 lb. She produces 12,000 lb. of milk (i.e. and average of 40 lb. per day) of 3.6 per cent butterfat and 3.5 per cent protein content in 300 days. She calves every twelve months.

Protein requirements

Forty pounds of 3.5 per cent protein milk contains 1.4 lb. of protein. For each pound of protein in the milk the cow requires 1.25 lb. of digestible protein[21].

For production of 40 lb. milk..	1.75
For maintenance[21].....	0.75
	2.5 lb. digestible protein.

Depending on the digestibility of the protein fed, 3.5 to 6.5 lb. of crude protein will therefore be required daily by a cow producing 40 lb. of milk per day. As it is undesirable to feed cows on a too high protein ration it is important to include only feeds with proteins of high digestibility in the ration of high producing cows.

During the dry stage, i.e. the two months before calving, the cow should receive her maintenance requirements plus an additional 0.65 lb. of digestible protein for the foetus[21].

Energy contents

Energy is required for the maintenance of the cow, for excercise above the normal, for the secretion of milk and for the energy rich constituents in the milk. The energy required for maintenance and exercise is not easily determined as the degree of exercise may vary so much from farm to farm due to climatic factors and to differences in pasture quality and management practices.

Energy is supplied by carbohydrates (including cellulose), fats and even proteins. It can be expressed in therms or Calories or the total

digestible nutrients in the feed may be used as an indication of the energy value of the feed.

Net energy required for maintenance[21].....	7,400 Cal.
Net energy required per lb. of milk is 280 Cal[21].	
Therefore net energy required for 40 lb. of milk	11,200 Cal.
	18,600 Cal.
	or 18.6 Therms.

These requirements are not easily met. A 1,200 lb. cow will eat about 24 lb. of hay per day. The normal practice in this country is to feed 0.3 lb. of concentrates per pound of milk produced, so that this cow will receive 12 lb. of concentrates per day. On an average, this ration will supply only 14 to 16 therms as net energy.

During the last two months of pregnancy the cow should be fed an additional 5.5 therms per day for foetal development.

Fats in the diet

Fats supply approximately 2.25 times as much energy as carbohydrates. Apparently cows need about 4 per cent of fat in the diet for optimum production. With less than 4 per cent of fat in the ration, milk production will decrease[22]. Feed fat with a high iodine value (unsaturated fatty acids) may depress the percentage of butter-fat in the milk[3, 24].

Roughage

Roughage is essential for optimum ruminal digestion and therefore for the nutrition of the dairy cow. Cellulose is the main source of volatile fatty acids which are the main precursors of butterfat in the ruminant. With a low roughage diet or even when the roughage is finely ground there is a corresponding drop in the butterfat content of the milk. When insufficient roughage is fed the iodine value of the butterfat may be increased due to a higher percentage of oleic acid and a lower percentage of short chain fatty acids in the milk fat.

Calcium and phosphorus

As stated earlier, the absorption of calcium and phosphorus may vary so widely that it is just about impossible to recommend optimum daily requirements. It is also important to know the source of phosphorus. The phosphorus in all seeds, for instance, is to a great extent bound as phytic acid (maize 75 per cent, wheaten bran 77 per cent of P bound as phytic acid) while in roughages very little (2 to 5 per cent) of it is in the form of phytic acid phosphorus. Phytic acid combines with calcium to form insoluble phytin, causing decreased calcium and phosphorus absorption. In the ruminant however up to 27 per cent of phytic acid phosphorus may be hydrolyzed by the micro-organisms[25].

Forty pounds of milk contain 21.6 gm. of calcium and 18.0 gm. of phosphorus. If calcium absorption is 50 per cent and phosphorus ab-

sorption 60 per cent then the minimum daily requirements will be as follows:—

	Calcium	*Phosphorus*
For maintenance[21]	12.0 gm.	12.0 gm.
For 40 lb. of milk	43.2 gm.	30.0 gm.
	55.2 gm.	42.0 gm.

During the last two to three months before parturition an additional 15.6 gm. of calcium and 9.6 gm. of phosphorus should be fed daily for foetal development[21].

Other minerals

Sodium and chlorine are electrolytes which are essential in the diet of high producers. It has been found that half an ounce of salt per day added to a salt free ration increased milk production by approximately 55 per cent, but that cows still showed signs of craving[27]. One ounce of salt per cow per day has therefore been recommended. Magnesium may have to be supplemented too under certain circumstances.

Trace minerals and vitamins

Trace minerals and vitamin A are required for the general well being of the cow and not directly for the synthesis of milk.

A high producing cow can only ingest a certain volume of feed per day. At times her requirements for the production of milk may exceed this limitation and in order to maintain her production it is essential that all nutrients should be perfectly balanced. Furthermore it is also desirable to pay more attention to the digestibility and the energy value of the feedstuffs used.

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REFERENCES

1. SMITH, V. R. (1960). Physiology of Lactation, 5th Ed. Constable and Co., London.
2. HANSEN, R. G. and CARLSON, E. M. (1956): An evaluation of the balance of nutrients in milk. *J. Dairy Sc.*, **39**, 363.
3. LARSON, B. L. and GILLESPIE, D. C. (1957): Origin of the major specific proteins in milk. *J. Biol. Chem.*, **227**, 565.
4. ACHAYA, K. T. and HILDITCH, T. P. (1950): A study of the component glycerides of cow and buffalo milk fat with reference to the possible mechanism of their production during lactation. *Proc. Roy. Soc. London, B.*, **137**, 187.
5. FOLLEY, S. J. and FRENCH, T. H. (1950): The intermediary metabolism of the mammary gland. 3. Acetate metabolism of lactating mammary gland slices with special reference to milk fat synthesis. *Biochem. J.*, **46**, 465.
6. POPJAK, G., FRENCH, T. H., and FOLLEY, S. J. (1951): Utilization of acetate for milk fat synthesis in the lactating goat. *Biochem. J.*, **48**, 411.
7. GRASHUIS, J. (1960): Personal communication.
8. JOHANSSON, I., and CLAESSON, O. (1957): Progress in the Physiology of Farm Animals, edited by J. Hammond, Chapter 21. Butterworths Scientific Publications, London.
9. JAYACHANDRAN, T., VENKATASWAMI, V., and VARMA, KERALA (1961): Studies on alcohol test. *Ind. Vet. J.*, **39**, 473.

10. MIKI IZUKA, (1960): Studies on alcohol positive milk with normal acidity. *Jap. Vet. Sc.*, **22,** 380.
11. HUFFMAN, C. F., ROBINSON, C. S., WINTER, O. B. (1930): Calcium and phosphorus metabolism of cows. *J. Dairy Sc.*, **13,** 432–448.
12. DAVIES, W. L. (1939): The chemistry of milk, 2nd ed. D. van Norstrand Co., New York.
13. COMAR, C. L., BECKER, R. B., DIX-ARNOLD, P. T., KRIENKER, W. A., DAVIS G. K., (1947): Phosphorus metabolism studies. I. Secretion and partition of dietary radioactive phosphorus in the milk of the dairy cow. *J. Dairy Sc.*, **30,** 557.
14. SAARINEN, P., COMAR, C. L., MARSHALL, S. P., DAVIS, G. K. (1950): Partition of orally administered radioactive phosphorus in the blood and milk of the dairy cow. *J. Dairy Sc.*, **33,** 878.
15. GROENEWALD, J. W. (1959): A guide to animal nutrition. Drakensberger-pers Bpk., Durban.
16. KLIEBER, MAX, SMITH, A. H., RALSTON, N. P. and BLACK, A. L. (1951): Radiophosphorus (P^{32}) as tracer for measuring endogenous phosphorus in cow's feces. *J. Nutr.*, **45,** 253.
17. MYBURGH, S. J. (1963): Personal communication.
18. WILBUR, J. W., HILTON, J. H., and HAUGE, S. M. (1940): The vitamin A requirements of dairy cows for the production of butterfat of high vitamin A value. I. Artificially dried alfalfa hay (carotene). *J. Dairy Sc.*, **23,** 765.
19. BOOTH, R. G., KON, S. K., DANN, W. J. and MOORE, T. (1933): A study of seasonal variation in butter fat 1. Seasonal variations in carotene, vitamin A and the antimony trichloride reaction. *Biochem. J.*, **27,** 1189.
20. STEWART, J. and MacCALLUM, J. W. (1942): The effect of a vitamin A rich diet on the vitamin A content of the colostrum of dairy cows. *J. Dairy Res.*, **13,** 1.
21. MORRISON, F. B. (1949): Feeds and Feeding, 21st edition, The Morrison Publishing Co., Ithaca, N.Y.
22. LOOSLI, J. K., MAYNARD, L. A., and LUCAS, H. I. (1944): Cited by Smith, V. R., Physiology of Lactation, 5th ed., Constable and Co., London.
23. OSTERHOFF, D. R. (1963): The protein content of cows' milk. (In press).
24. SHAW, J. C., ENSOR, W. L. (1959): Effect of feeding cod liver oil and unsaturated fatty acids on rumen volatile fatty acids and milk fat content. *J. Dairy Sc.*, **42,** 1238.
25. BARNETT, A. J. G., and REID, R. L. (1961): Reactions in the rumen. Edward Arnold Ltd., London.
26. EWING, W. R. (1951): Poultry Nutrition. 4th ed. W. Ray Ewing, Publisher, P.O. Box 248, South Pasadena, California.
27. SMITH, S. E. and MORRISON, F. B. (1957): Salt for Dairy Cattle. Salt in Animal Nutrition. Published by Salt Institute, Chicago 2, Illinois.

SYMPOSIUM: PROBLEMS ASSOCIATED WITH THE HIGH PRODUCING DAIRY COW

SOME METABOLIC DISTURBANCES ASSOCIATED WITH THE HIGH PRODUCING DAIRY COW

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INTRODUCTION

In spite of new knowledge which is constantly being added to the understanding of metabolic diseases in ruminants, the clinician still has difficulty in providing rational advice regarding the cause and prevention of these conditions. If the practitioner is to play a significant role in preventing these diseases, it is profitable to bear in mind certain concepts:—

(1) The ability to convert nutrients into milk is inherited. The hypothalamus and the anterior pituitary govern milk secretion, while the adrenal, thyroid and parathyroid glands play an essential supporting rôle. The blood supply, the amount of secretory tissue and the storage capacity of the udder are anatomical factors which influence the milk yield of the cow. Both the endocrine complex as well as the anatomical characteristics are inherited, and a high production potential increases the possibility of metabolic diseases.

(2) The composition of milk remains essentially constant. If one or more components are not available in the diet, secretion will either be reduced or body reserves will contribute towards the formation of milk; the mobilization of these reserves is under hormonal control. Physiological balance in the high producing animal depends on a sensitive equilibrium between the mobilizing hormones and the hormonal factors stimulating milk formation in the alveolar epithelium. The functional capacity of the endocrine glands and thus the hormonal balance is also inherited.

(3) The living organism must be regarded as an open or flow system where change is the vital requirement[3]. "The open system has as its constancy that of constant change. If the latter ceases, life ceases. Constant change again is crucially dependant on external environment and the environmental dependance of the open system is a consequence of genetic conditioning of both its anatomical and chemical architecture. The body must

adjust to life within the confines of its genetic prison, and the lack or freedom from the external environment is co-linked with inherited inperfection of structure"[4].

(4) The therapy of metabolic disturbances has been greatly influenced by the demonstration of abnormal increases or decreases of blood constituents. The bloodstream must however always be regarded as only a vehicle for the various precursors or components of milk, and as such only reflects the balance or imbalance between input of components and output of milk. Grosskopf[11], for instance, indicates that the total amount of calcium or phosphorus present in extracellular fluid at any given time is not sufficient to form more than 10 to 12 pounds of milk. Prevention and treatment must therefore be aimed at the input, i.e. absorption and mobilization, or the output governed by the hormones influencing the synthesis and secretion of milk.

Judging from the influence which a change of ration can have on milk production within twelve hours, it must be assumed that under normal conditions the major portion of the milk constituents or their precursors are derived directly from intestinal absorbtion. Under normal circumstances the fat, protein and bone depots of the body contribute proportionally little directly to milk production but act as reservoirs to maintain homeostasis in the event of a sudden drop in the contribution from the digestive system.

Rumen Dysfunction

Normally a considerable portion of the ration fed to the high producing cow consists of concentrates. Sudden changes in the concentrate ration is responsible for most of the primary digestive disturbances encountered in dairy practice.

The composition of the ration is known to influence the ruminal microflora which in turn effects the products of fermentation, the pH of ruminal liquor, and exchange of ions between rumen and blood. The rapid and very marked change in the activity of the microflora is perhaps not readily appreciated. Grosskopf[12] has, for instance, demonstrated that the sudden addition of a relatively high carbohydrate ration to a low quality fibre diet will, within 24 hours, increase the time taken to digest 50 per cent of cotton fibre from 21 to 72 hours.

It is common practice to increase the concentrate ration just prior to or immediately after calving. Provided this is done gradually, ruminal function is able to adjust without digestive disturbances. If, however, large quantities of a carbohydrate-rich ration are suddenly fed, or the cow accidentally overeats on this food, severe and sometimes fatal digestive disturbances may follow. Such a diet will cause rapid bacterial fermentation of carbohydrate with marked production of lactic acid, and depression of pH[6].

Clinically, the animal becomes severely ill within 4–6 hours after overeating. There is complete ruminal stasis; pain is evidenced by

grunting; there is dehydration, laboured breathing, salivation and severe depression. Death ensues within 24–36 hours. Treatment is not very successful unless an early rumenotomy is performed to remove the contents of the forestomachs. If surgical interference is not considered, the pH of the rumen should be increased by the administration of lime water or a similar alkalizer and dehydration relieved by intravenous as well as adequate oral fluids.

Also common is the ruminal stasis with a slower onset, and less severe symptoms responding more easily to treatment. These disturbances are secondary, and commonly associated with conditions such as metritis, mastitis, foot-rot and other infections. Contrary to the changes in ruminal disturbances associated with overeating, the pH in these cases rises and becomes alkaline. Clinically, there is listlessness, anorexia and cessation of milk flow associated with a moderately severe ruminal atony. If treated early, prognosis is favourable and normal digestion is re-established within 48 hours.

Treatment consists of acidifying the ruminal contents by twice daily dosing of approximately 60 grams of propionic acid with some 12 ounces of molasses mixed with water. At the first treatment 4–6 gallons of lukewarm physiological saline with a pint of fresh ruminal fluid should be dosed through a tube. If fresh ruminal fluid is not available, two ounces of fresh commercial yeast have a beneficial effect. After administration of the fluid the rumen should be massaged through the abdominal wall until its consistency becomes liquid.

Bovine Ketosis

When using the term ketosis, a distinct difference should be made between primary ketosis which is an uncomplicated disease entirely characterized by a deranged carbohydrate metabolism, and secondary or starvation ketosis where pathological condition such as metritis, mastitis and footrot cause a ketosis syndrome resulting from the inappetence of the cow.

Sjollema and van der Zande[23] describe the classical symptoms as occurring in fat, high-producing cows, usually 7 to 10 days after calving. The animals lack appetite, show a tendency to constipation, a rapid loss in body condition and decrease in milk production, many cases also showing nervous symptoms. These workers were also the first to demonstrate high ketone body levels in the blood and urine of cows suffering from the disease.

The same symptoms appear in secondary ketosis although nervous symptoms, apart from slight incoordination of the hind legs, are rarely noticed. Ketonuria and acetone bodies in the milk can, however, be demonstrated.

Bovine ketosis is a difficult disease to diagnose and differentiation between primary and secondary symptoms is even more difficult. In both instances the high level of ketone bodies must be demonstrated, and the diagnosis of primary ketosis is often only clinched by favourable response to treatment.

Shaw[21] quotes evidence regarding the geographical distribution, seasonal incidence, age, and breed susceptibility to bovine ketosis. It is stated that the disease is common wherever dairying is practised, and that, with one or two exceptions, the disease is virtually confined to the winter season and to stabled animals. The apparent antiketogenic effect of pasture feeding is often stressed. The age incidence seems to be the same as for milk fever and Guernseys and Holsteins (Frieslands) exhibit a higher susceptibility than other breeds.

It is interesting and illuminating to compare the above quoted facts with the position in South Africa. Primary ketosis can by no means be said to be a serious disease in South Africa, and most cases of ketosis dealt with by practitioners are of a secondary nature. Of the primary cases encountered, it is equally interesting to note that by far the greater majority occur in the Winter-rainfall area of the South-Western Cape. Most of these cases occur during the winter in this area but seasonal calving is perhaps largely responsible for this. Can this difference between the situation in the Republic and many of the other dairying countries be ascribed to the fact that all animals here graze throughout the year? Do the comparative lack or natural grazing and the fact that cows in the Western Province remain on artificial grazing throughout the winter explain the higher incidence of primary ketosis in that area? Unfortunately, such questions cannot be answered at this stage but current research into these factors is bound to yield interesting answers.

The characteristic laboratory findings in ketosis are increased ketone bodies in blood, urine, and milk, and in most cases, although not always, a decrease of blood glucose. The excessive increase of aceto-acetic and beta-hydroxybutyric acid as well as acetone, results in an acidosis which, in fatal cases, is the immediate cause of death.

Many theories have been advanced to explain these disturbances, but a survey of present day knowledge indicates that the concept first advanced by Shaw[20] that ketosis might possibly be caused by an unbalanced endocrine system, is most widely accepted. If primary ketosis is due to an adrenal insufficiency caused by a temporary exhaustion of the pituitary as the result of stress, it would appear that most ketotic cases are caused by unknown stress factors superimposed upon normal stresses incident to parturition and early lactation[21]. In view of the low incidence of primary ketosis in South Africa it may be asked whether winter stabling and lack of exercise and natural grazing comprise some of these unknown stress factors. Kronfeld[16] adds to the concept of disturbed endocrine function by suggesting that selective breeding for high production has resulted in these animals having a high somatotrophin output which stimulates milk production to the detriment of other body functions, particularly carbohydrate and fat metabolism.

Apart from the provision of natural grazing, minerals, vitamins, fats, and protein have not been proved to be associated with the prevention of bovine ketosis. Because hypoglycaemia is often associated with ketosis, the feeding of a high carbohydrate containing ration seems to be a rational measure in preventing the onset of the disease. This has, however, not proved particularly helpful in the prevention of ketosis,[19, 9] although calcium lactate fed at the rate of about one pound per day has been

found very effective[22]. To suggest, however, that feeding exerts no influence on the incidence of ketosis would be completely misleading. Marshak[18] has demonstrated a marked decrease in the incidence of the disease where cows were consistently fed on adequate protein and carbohydrate ration throughout their gestation and calving periods. This ensures that at all times the cow will have sufficient nutrients available to meet the demand no matter how great somatotrophin output becomes.

Vigue[24] has called the ketotic cow a „milking fool" after demonstrating the difference between the reactions of the normal and ketotic cow to sudden deficiency of nutrients. During complete starvation of cows a few weeks post partum, they developed ketonaemia and relative adrenal insufficiency as indicated by a rise in circulating eosinophiles and response to the 4 hour A.C.T.H. test. Normal cows failed to develop hypoglycaemia or clinical ketosis but milk production dropped to almost zero. In contrast the ketotic cow keeps on producing milk in spite of a negative nutrient balance because of the inability to readjust the output of her anterior pituitary hormone rapidly enough to cope with a dietary deficiency by reducing milk secretion.

The most effective treatment for primary ketosis is the administration of glucocorticoids as first reported by Shaw[20]. Most cases of primary ketosis will respond to one injection of 1.5 grams of cortisone acetate. The doses for newer corticoid preparations can be computed by comparing their activity with that of cortisone acetate. Intravenous glucose therapy for ketosis has enjoyed widespread support but unfortunately a fair number of cases will not respond to one injection of 50 per cent glucose solution. The routine use of a small dose of insulin with intravenous glucose therapy has not received the acceptance which seems warranted. If hypoglycaemia is present there is a reduction of insulin output, but apart from this it must be assumed that the output of insulin cannot be relied upon once the biochemical processes in the body become seriously disrupted. The administration of insulin after intravenous glucose therapy holds little danger and is to be recommended when glucose is used in the treatment of ketosis.

In secondary ketosis the early recognition and correction of the primary disease is essential, after which treatment on the general lines as for primary ketosis is undertaken. In these cases partial or complete ruminal atony is usually present, and it is important to re-establish ruminal function at the earliest possible moment.

Milk Fever

Milk fever is the most frequent metabolic disturbance of high producing cows in South Africa. Like ketosis, however, the disease is a major problem only in the winter rainfall area of the Cape and in the Midlands of Natal. It occurs mostly in the Jersey breed, which predominates in these areas. It is difficult to decide whether the high incidence in these areas is due to local conditions or to the predominance of the Jersey breed.

Although all the animals in a herd are subject to the same environmental factors, only certain animals will be affected. Consequently

environmental factors cannot be the sole cause of milk fever and it must be assumed that certain constitutional factors play a part in the pathogenesis.

Milk fever never occurs at the first parturition, occasionally at the second parturition, and increases in frequency with subsequent calvings. The disease occurs around parturition and is characterized by a normal or subnormal temperature, inability to rise, and pronounced somnolence which will end in coma and death if untreated. In herds studied by Jönsson[15] the milk production was significantly higher in affected than in normal cows, which shows a direct relation between high production and the disease. Investigation of the lactation curve in these animals showed that the persistency of yield was greater in paretic than in normal animals, but no information was available on the lactation level immediately following parturition.

Little & Wright[17] demonstrated a marked decrease in serum calcium in milk fever. Many investigators, however, showed that serum calcium normally decreases before and shortly after parturition. Ward *et al*[26, 27] demonstrated a negative calcium balance 11–17 days before parturition which usually disappears about 5 days after parturition. Hallgren[13] showed that the "physiological" fall in calcium coincided with initial mammary activity, by showing that the hypocalcaemia did not occur in mastectomized animals before parturition.

The reasons for the pathological drop in serum calcium in affected cows have been investigated by many workers, but the evidence remains conflicting. Opponents of a theory that there is an interference with calcium absorption, point out that many high producing cows under identical conditions will not be affected while some animals will get the disease. Many believe that calcium mobilization from the skeleton is interfered with because of relatively deficient parathyroid activity, a theory advanced as early as 1952 by Dryerre & Grieg[5]. After reviewing the literature Hibbs[14] concludes that, in weighing all the experimental evidence, the theory of Dryerre & Greig seems to be the most acceptable in explaining the cause of the disease. Because hypoparathyroidism causes a hypocalcaemia and hyperphosphataemia, Fish[10] on finding a hypophosphataemia, rejected the theory that parathyroid deficiency was responsible. The same reason was advanced by Hallgren[13] and Jönsson[15] after an histological study of the parathyroids of normal and afflicted animals, concluded that there were no differences in the glands and that the parathyroid was not implicated in parturient paresis.

In spite of the fact that parathyroid dysfunction has been rejected as a cause of the disease, Boda & Cole[1] devised a method to prevent milk fever by feeding a ration low in calcium and high in phosphorus. They assumed that hypoparathyroidism was the cause and suggested this diet as parathyroid function can be increased by a low calcium-high phosphorus diet[2, 8].

Ender *et al*[7] demonstrated that with a high calcium containing ration with low or normal phosphoros, where the ration was characterized by normal or high alkali-alkalinity, they could cause milk fever symptoms in nearly one half of the experiments.

If however, to this high calcium ration large quantities of mineral acid ensilage was added to reduce the alkali alkalinity of the daily food intake to extremely low values, practically no cases of milk fever occured. Normal amounts of calcium and phosphorus fed in a ration of normal alkali-alkalinity prevented milk fever altogether.

The veterinarian is unable to change the inherited susceptibility of the animal and must rely, for preventative measures, on the influence of diet on milk fever. A low calcium-high phosphorus containing ration yields encouraging results and should be recommended. This can be done by substituting good quality hay for lucerne in the ration, and adding water soluble phosphate to the drinking water.

In treating a case of milk fever the calcium solution should contain soluble phosphorus to correct the hypophosphataemia. In the case of downer cows, two grams of potassium chloride should be added to the intravenous solution. In the latter case great care must be taken with the rate of injection as intravenous potassium administration is always dangerous.

Discussion

What part can the veterinarian play in the management of metabolic diseases?

The complex interrelationship between internal homeostasis and external environment in maintaining the health of the high producing cow, requires a sound knowledge of animal management, breeding, feeding, physiology, and therapeutics. If the veterinarian is not thoroughly familiar with these subjects he is reduced to the level of a technician who injects corticoids or calcium borogluconate and never rises to the level of practising preventative medicine. He is unable to fill the role of adviser which the farmer expects and makes no contribution towards avoiding the loss of production which ensues with every metabolic disturbance.

It is interesting that breeders will never question the feasibility of breeding desirable characteristics into their animals, but for some reason they regard the resistance to disease as a trait which cannot be altered by breeding.

It is clear that hormonal balance plays an important rôle in ketosis and milk fever and nobody will dispute the fact that this characteristic is inherited. In this respect the rôle of the veterinarian has become vital as the profession controls artificial insemination and therefore also influences the selection of bulls the inherited characteristics of which are bred into greater numbers of high producing dairy cows.

Are sufficient questions asked about the breeding of a bull before it is bought for the artificial insemination centre? The milk and butter fat production of the ancestors appear on the pedigree, and with some effort the breeding record and longevity of the ancestors can also be ascertained, but it is virtually impossible to find out if the dam of a particular bull was not a regular sufferer of milk fever. It is the duty of every veterinarian, working with high producing herds, to persuade the breeder that a record of disease incidence is vital for his breeding programme and that he can breed disease-resistance into his animals.

Veterinarians who assist in selecting bulls should likewise insist on detailed information about the disease record of the forebears. If these questions are asked and the reasons for asking explained, the veterinarian will contribute materially towards making breeders aware that disease resistance can be bred into their herds.

The rôle of trace elements such as cobalt[28] and copper[25] has not been discussed in relation to metabolic diseases but any deficiencies undoubtedly contribute to the incidence and complexity of these disturbances.

If the veterinarian is to be of value in disease prevention, he must have a sound knowledge not only of dietetics in general, but must have an intimate knowledge of the trace element status, the fertilizing programmes, and the methods of food production in his practice.

The amount of copper found in the soil in the Republic varies from frankly deficient, through marginal, to amounts which border on the toxic level. Only by having the trace element analysis on ruminant livers from representative areas in his practice, can the veterinarian prescribe adequate and safe licks or trace element additives. The protein content, calcium-phosphorus ratio, and alkalinity of the common foodstuffs during the different seasons of the year are equally important if the veterinarian is to advise on feeding practices which will minimise or prevent metabolic disturbances.

The veterinarian is expected to be able to diagnose and treat metabolic diseases, but is this as much as his profession can expect from him? While intricate laboratory research on metabolic pathways etc., is carried out all over the world, there is a dearth of publications on the incidence, environmental factors, clinical symptoms, field diagnosis and treatment of metabolic diseases. Only the practitioner is able to contribute in this field and it is his duty to make available to his colleagues and research workers his experience of the factors influencing these diseases in practice, and the results which he obtains with preventative and curative methods. Every contribution in this field strengthens the whole profession and helps to equip veterinarians as important contributors to the health and production of our dairy industry.

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REFERENCES

1. BODA, J. M. and COLE, H. H. (1954): The influence of dietary calcium and phosphorus on the incidence of milk fever in dairy cattle. *J. Dairy Sci.*, **37**, 1027.
2. CAMBELL, I. L. and TURNER, C. W. (1942): The relation of the endocrine system to the regulation of calcium metabolism. *Res. Bull. Mo. agric. Exp. Sta.*, 352.
3. DRABKIN, D. L., (1959): Imperfection: Biochemical phobias and metabolic ambivalence. *Perspectives Biol.Med.*, **2**, 473.
4. —— (1963): Kinetic basis of life processes: Pathways and mechanism of hepatic protein synthesis. *Ann. N.Y. Acad. Sci.*, **104**, 469.
5. DRYERRE, H. and GREIG, J. R., (1925): Milk fever: its possible association with derangements in internal secretions. *Vet. Rec.*, **5**, 225.
6. ELSDEN, S. R. (1945): The fermentation of carbohydrates in the rumen of sheep., *J. Exp. Biol.*, **22**, 51.

7. ENDER, F., DISHINGTON, I. W. and HELGEBOSTAD, A., (1962): Parturient paresis and related forms of hypocalcemic disorders induced experimentally in dairy cows. *Act. Vet. Scand.*, Suppl. 1. Vol. 3.
8. ENGFELDT, B., HJERTQUIST, S. O. and STRANDH, J. R. E. (1954): The parathyroidal function in long term dietary experiment. *Act. endocrin. Copenhagen*, **15**, 119.
9. FINCHER, M. G. (1950): Preventing ketosis in cattle. *North Am. Vet.*, **31**, 407.
10. FISH, P. A. (1929): The physiology of milk fever. 111. The blood phosphates and calcium. *Cornell Vet.*, **19**, 147.
11. GROSSKOPF, J. F. W. (1963): Some physiological aspects of lactation and the nutritional requirements of the high producing dairy cow. *This issue.*
12. —— (1963): Personal communication.
13. HALLGREN, W. (1940): Quoted by Jönsson, G., (1960).
14. HIBBS, J. W. (1950): Milk fever (parturient paresis) in dairy cows — a review. *J. Dairy Sci.*, **33**, 65.
15. JÖNSSON, G. (1960): On the etiology and pathogenesis of parturient paresis in dairy cows. *Act. Agric. Scand.* Suppl. 8.
16. KRONFELD, D. S. (1963): Ruminant ketosis: A speculative approach. *Ann. N.Y. Acad. of Sci.*, **104**, 799.
17. LITTLE, W. L. and WRIGHT, N. C. (1925): The aetiology of milk fever in cattle. *Brit. J. Exp. Path.*, **16**, 129.
18. MARSHAK, R. R. (1955): The nutritional concept as the underlying cause of bovine ketosis. *Vet. Med.*, **50**, 159.
19. SHAW, J. C. (1943): Studies on ketosis in dairy cattle. V. The development of ketosis. *J. Dairy Sci.*, **26**, 1079.
20. —— (1947): Studies on ketosis in dairy cows. IX. Therapeutic effect of adrenal cortical extracts. *J. Dairy Sci.*, **33**, 307.
21. —— (1955): Bovine ketosis. *Advances in Vet. Sci.*, II. 262.
22. SHAW, J. C., CHUNG, A. C., GESSERT, R. A. and BAJWA, G.. (1955): Additional studies on the etiology and treatment of bovine ketosis, including an evaluation of metacortondracin, 9-alphafluorohydrocortisone acetate and calcium lactate. *Misc. Publ. Maryland Agr. Exp. Sta.*, **238**, 1.
23. SJOLLEMA, B. and VAN DER ZANDE, J. E. (1923): Metabolism in acetonaemia of milch cows. *J. Metabolic Res.*, **4**, 525.
24. VIGUE, (1954): Quoted by Marshak, R. R. (1955).
25. VAN DER WALT, K. and ADELAAR, T. F. (1962): Unpublished data.
26. WARD, G. M., BLOSSER, T. H. and ADAMS, M. F. (1952): The relation of prepartal and postpartal mineral balances to the occurance of parturient paresis in dairy cows. *J. Dairy Sci.*, **35**, 587.
27. WARD, G. M., BLOSSER, T. H., ADAMS, M. F., and CRILLY. J. B. (1953): Blood levels of some inorganic and organic constituents in normal parturient cows and cows with parturient paresis. *J. Dairy Sci.*, **36**, 39.
28. WHITE, E. A. Ketosis in dairy cattle. I. The role of cobalt and the significance of Vit. B12 in this metabolic disturbance. *Vet. Med.*, **50**, 199.

SYMPOSIUM: PROBLEMS ASSOCIATED WITH THE HIGH PRODUCING DAIRY COW

THE INFLUENCE OF INDUCED HIGH PRODUCTION ON REPRODUCTIVE EFFICIENCY

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INTRODUCTION

High milk production and good fertility are the two outstanding qualities of the ideal dairy cow, and the proper performance of these two functions is conditioned by the genetic potential for milk yield and fertility on the one hand and by proper nutrition, good management and a suitable environment on the other.

IMPROVEMENT OF DAIRY STOCK

The dairy cow, like the females of all other mammals, was intended by nature to provide merely sufficient milk for the nourishment of the young. Man, however, realising the high nutritive value of milk products has brought her to a level where she yields 8 to 10 times as much per lactation as what her ancestors were originally equipped for by their genetic make-up.

The reason for the attainment of this high level of production without serious impairment of other bodily functions is mainly the care and intelligence shown by a high proportion of breeders who realise that the volume of milk a cow will yield depends on, firstly the inheritance which she possesses for this characteristic, and secondly the opportunity she is given to produce to the limit of her inherited ability.

The pioneer breeder and founder of modern breeding practice, Robert Bakewell (1725–1795), was the first to formulate definite ideas on breeding for the purpose of improving livestock for the performance of one or other specific function. A hundred years later scientific breeding received its greatest impetus of all time when the Austrian monk Mendel published the results of his work which led to the formulation of Mendel's laws, and so laid the foundation of that important branch of biology known as genetics.

At about the same time the first case was recorded of a cow yielding more than 1,000 gallons of milk in one year. This was the Holstein Dowager who gave 1,268 gallons in 1870.

The next 30 years saw the emergency of a definite concept of breed purity and the formation of the various breed societies to further the interests of the different breeds. It is certainly no mere coincidence that the production potential of the dairy cow has increased threefold in the past 90 years, the record yield now standing at over 4,200 gallons. The efficient modern dairy cow thus secretes about 20 times her own body weight of milk every year.

The question now arises whether it is possible to continue increasing production at this terrific rate without harm to other bodily functions, notably to reproduce.

Relationship Between Lactation and Reproduction

Reproduction and lactation are intimately related to each other through the absolute dependence for their normal activities on the endocrine system, supreme control of which is vested in the pituitary.

Here we must take cognisance of the fact that the udder is a cutaneous gland present in both male and female, and that it is only through the action of the pituitary and ovarian hormones of the female that it becomes a specialised tubulo-alveolar gland for the production of milk. It is thus even possible to cause mammary development and lactation in the male by using the appropriate endocrines.

Briefly the hormones controlling reproduction and lactation are obtained from the following sources: The anterior pituitary secretes FSH (follicle stimulating hormone), LH (luteinising hormone), ACTH (adreno-corticotrophic hormone) and TSH (thyrotrophic hormone) from its basophils, and LTH (luteotrophic hormone or prolactin) and STH (somatotrophic or growth hormone) from the acidophils. Indirectly the anterior pituitary through its gonadotrophic hormones also controls the secretion of the ovarian hormones oestrogen, progesterone and relaxin. The posterior pituitary supplies oxytocin and vasopressin.

Research work in the past decade has revealed that the hypothalamus, which is anatomically very closely related to the pituitary, exerts a marked influence in the latter's function and therefore on the secretion of the gonadotrophic hormones. The anterior pituitary is connected with the hypothalamus by a vascular plexus of portal vessels which carry blood from the ventral brain structures to the pituitary, and it appears that the effect of the hypothalamus on the pituitary is through a neuro-humoral substance transported to the anterior pituitary by this vascular route. This accounts for the manner in which exteroceptive stimuli such as management, fear, excitement and light which emanate from the animals environment, affect the pituitary hormones and consequently both mammary and gonadal activity.

Milk production takes place in three stages, namely (*a*) growth and development of mammary tissue, (*b*) synthesis and secretion of milk, and (*c*) ejection of milk.

In the heifer, growth of the duct system of the mammary gland is stimulated primarily by oestrogen, and in the cow oestrogen induces considerable lobule and alveolar growth. Initiation of lactation is brought about by LTH, and the level of milk yield is maintained by other anterior

pituitary hormones like TSH and STH which like LTH are secreted by the acidophils. These in addition to ACTH are also regarded as secondary hormones of reproduction.

Ejection or let-down of milk results from a sudden increase in intramammary pressure in which both the nervous and endocrine systems are involved. External stimuli such as washing the udder and rattling of buckets are conveyed to the hypothalamus which induces release of oxytocin and vasopressin from the posterior pituitary. These stimulate the myoepithelial cells of the udder to contract, thus increasing intramammary pressure and forcing out milk from the alveoli and small ducts.

Decompensation

It is obvious from the above that this intimate interrelationship between lactation and reproduction constitutes a very delicate physiological mechanism, and that optimum performance of both functions depends mainly on the inherited ability of the udder to synthesize and secrete the increased volume of milk and on the ability of the pituitary to provide the relative hormones in their proper proportion and quantity. Selective breeding has conferred these qualities on the high producing modern dairy cow.

The ideal in dairy farming is to give every cow an opportunity to produce to the maximum of her constitutional ability. The harmful effects on production and fertility of defects in any of the extraneous factors like nutrition, stockmanship and environment are well known and are not relevant here. The evil consequences however of attempting to raise the level of production beyond the cow's inherent capacity by excessive feeding are more sinister and therefore not so apparent. It is thus not uncommon to find owners forcing cows to produce beyond their ability by excessively high feeding. This eventually leads to decompensation, the effect of which on reproduction is manifested in various types of functional infertility.

Consideration is not always given to the fact that like food conversion and milk synthesis, pituitary activity is also governed by hereditary traits. Strong evidence of this has recently been provided by the reports on two research projects in this country. In the first, the genetic influence on pituitary functions on reproduction was demonstrated by van Rensburg and de Vos[1] who showed that ovulatory failure in bovines may be heritable, and that it is more frequent in families with poor breeding records. In the other K. M. van Heerden[2] concluded that abortion in Angora goats is due to defective LTH causing deficiency of progesterone, and that this is probably the result of an hereditary weakness.

The food intake of the dairy cow must provide for her basal metabolism, bodily activities, reproduction and milk secretion. It is estimated that half of the nutrients required by the average dairy cow is for milk production and half for all other activities. With above average producers a larger percentage nutrients is necessary for milk secretion. It follows that food intake must vary with production level and that when dairy cows are not fed strictly according to yield there is a tendency to underfeed high yielders and overfeed low producing cows.

Many owners consider the latter point not of sufficient importance to justify the extra labour and trouble which feeding according to production entails, and so supply a uniform ration. Consequently a large proportion of the herd gets too much, and therefore produce milk on a level above their inherent potential. Others again, and notably stud breeders, overfeed solely for the purpose of creating production records.

The endocrine system cannot continue indefinitely meeting such excessive demands, and reproductive efficiency is the first to be adversely affected.

The harmful effects of such undesirable practices are manifested in functional disturbances in ovarian activity such as inactive ovaries and ovulatory aberrations. The investigation of van Rensburg and de Vos[1] has thus revealed that ovulatory failure shows a sharp linear increase with age in dairy cows, and declines in beef animals so that fertility in the beef breeds is much higher at the age of 10 years and over, than in dairy cows. They postulate that the decline in fertility with age in dairy animals is due to their inability to continue producing milk at a level beyond their inherent capability, without detrimental effect on reproduction.

This also explains why so many of the cows that establish milk production records have relatively short lactation lives and leave only a small number of offspring, few of which are able to repeat their dam's performance. Our dairy breed societies would do well if they attach less importance to high production in one or two lactations and assign greater value to the volume of milk yielded over a long period combined with regularity of calving. The high milk production in association with efficient reproduction of the latter type of animal signifies inherited capability; while the sporadic good performance of the former has frequently been induced artificially by abnormally high feeding.

Winters[3] states that "heredity is fundamental and may be thought as furnishing the foundation with environment completing the structure". Therefore intensive forced feeding for production beyond their genetic potential or foundation will render the structure top heavy thus leading to a breakdown in some body functions, the most vulnerable of which is reproduction on account of its absolute dependence on a proper balance in the level of production of the different endocrines.

An abnormally high nutritional plane generally involves rations with excessive protein, hay and green feed, unaccompanied by a corresponding increase in other essential constituents, expecially minerals. Even when the intake of these is raised to the level required for the higher yield the conversion ability of the average cow does not enable her to metabolize and utilize more than the requirements for her normal milk yield. The result is that the reserves of the body are drawn upon for the necessary additional supplies especially of minerals like phosphorus and calcium and some of the trace minerals like copper, iron and manganese. This may cause serious interference with endocrine functioning since, as stated by King[4], trace elements act as catalysts involved in hormone or enzyme systems, either as constituent parts of the molecules of hormones, vitamins and enzymes, or as enzyme activators. The drain on the body tissues of the dairy cow forced to produce beyond her inherent capability is particularly severe in the post-calving period when milk yield is at its peak, and is

characterized by ovarian inactivity resulting in abnormally long periods of anoestrus.

Abrams[5] pointed out that the metabolism of 1956 was not that of 1900 since the economic rate of weight gain, milk production and level of fertility had changed so much in the last half-century. Allcroft[6] declared that the ever increasing intensification of production imposes new metabolic and nutritional requirements which offer a challenge to those concerned with these aspects of animal health.

Our knowledge regarding the metabolism of the essential minerals and of their inter-relationship and mutual antagonisms is still too meagre to permit advocating compound mixtures to supply all the needs of the high producing cow. We do however know that the deficiencies that occur may be of three types, namely (*a*) direct and due to insufficient intake; (*b*) conditioned and caused by antagonistic action between the different elements, and (*c*) physiological and resulting from inability of the body to absorb and metabolize available supplies. Nutritionists and biochemists may provide the solution of the first two. The third involving the inherent capability of the body mechanism can only be rectified by correct breeding.

The truism that improvement is relatively easy when quality is poor was partly responsible for the rapid increase in production during the past century and one cannot expect the same rate to be maintained indefinitely. Nevertheless modern breeding practices, notably artificial insemination and progeny testing, which enable early and more accurate assessment of the breeding value of sires to be made by using as a criterion the performance of the progeny rather than the achievements of the ancestors, have given present and future breeders the means for continuing to effect improvement by judicious selection. This is the foundation on which must be imposed correct environment, nutrition and management to ensure the perfect combination of high milk yield and optimum fertility.

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REFERENCES

1. VAN RENSBURG, S. W. J., DE VOS, W. H. (1962): Ovulatory failure in bovines. *Onderstepoort Jl.*, **29**, 55–79.
2. VAN HEERDEN, K. M. (1961): Investigation into the causes of abortion in Angora goats in South Africa. *J.S. Afr. vet. med. Ass.*, **32**, 211–220.
3. WINTERS, L. M. (1958): Animal breeding. John Wiley and Sons Inc., New York.
4. KING, J. O. L. (1961): Veterinary Dietetics. Balliere, Tindall & Cox, London.
5. ABRAMS, J. T. (1957): Mineral supplement for farm animals. *Vet. Rec.*, **69**, 204–210.
6. ALLCROFT RUTH (1961): The use and misuse of mineral supplements. *Vet. Rec.*, **73**, 1255–1266.

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SYMPOSIUM: PROBLEMS ASSOCIATED WITH THE HIGH PRODUCING DAIRY COW

THE UDDER IN HIGH PRODUCTION

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INTRODUCTION

The primary purpose of the dairy cow is the production of abundant clean, good quality milk, and consequently maximum realisation of her production potential with minimum harm and injury to her milk producing mechanism must be the aim if this purpose is to be achieved at an economic level.

A striking feature of the development of the dairy cow has been the fact that, in contrast to the low producing cow milked by hand or suckled by her calf, udder affections of various types have become an increasing problem. Mastitis which is the most common affection of the dairy cow's udder, and whether it is of an infectious or bacterial nature or whether it is traumatic, it represents, in general a manifestation of decompensation as a result of stress.

In considering the theme of this symposium it becomes necessary briefly to elaborate on some of the more important factors which influence the reaction of the udder to the demand we make of the modern dairy cow.

METHOD OF MILKING

It is only the exceptional hand-milker who can milk 30–35 lb. of milk in the 4–6 minutes of oxytoxic stimulation.

Reller[23], has shown how harsh hand milking (e.g. with forefinger and thumb, the latter being bent at right angles so as to bring the hard bony surface of the joint against the teat) may cause pathological changes to the teat and lead to catarrhal secretion changes.

The leucocyte count is probably one of the most sensitive indicators of the reaction of the udder to the method of milking — Hauke[13]. Cone[7], showed that transfer of normal cows and cows showing mastitis from hand to machine milking resulted in a 2–3 fold increase in the number of leucocytes present.

Whittleston[28] compares the behaviour of the teat sphincter to that of a highly damped spring. The sphincter opens at a certain vacuum level which varies from cow to cow, teat to teat as well from day to day. Once opened the damped action makes the sphincter lag behind the vacuum level change brought about by the pulsator. This hysteresis leads to a patency of teat duct during the closed phase and may result in the milk, trapped between the teat-tip and the collapsed liner, re-entering the cistern. Clean-cut position change of the liner in response to pulsation control gives the teat-sphincter sufficient time to close.

Cross infection may be due to clusters in which teat alternation occurs. In this type the teats are milked in pairs so that when one pair is released the other is squeezed resulting in the milk surging from side to side, leading to contamination of all the parts. It is also difficult and cumbersome to design such a plant in a way that will allow of phase-ratio other than 1:1. Milking all four teats simultaneously does not completely eliminate the possibility of surging and mixing of milk from the quarters; any sudden change in vacuum levels e.g. that occasioned by the sudden and simultaneous release of milk in recorder type multiple units may lead to surging in the clusters.

Dahlberg[8], showed an increase in the leucocytic count of milk from cows when the teat cups were left on for more than 4–5 minutes. This is aggravated by high vacuum levels as was shown by Meigs et al[17], as quoted by Whittleston [28], but there still remains some doubt that vacuum levels per se increase the incidence of mastitis. Bratlie[4, 5] however showed that high vacuum levels ($19\frac{3}{4}$ ins. Hg.) increased the incidence of cyanotic teat tips, oedema and extroversion of the canal mucous membrane. Maffey[16] rightly pointed out that greater care was needed when milking at high vacuum levels. Wilson[29] quotes a case of a herd where low vacuum was inculpated as a cause of an outbreak of mastitis although he submits that it was more probable that vacuum variation was responsible.

As the nutritional level increases in step with production so managemental problems arise e.g. it is well known that potential high producing heifers are "steamed-up". The resultant deposition of fat in the udder has been claimed to increase susceptibility to mastitis. Wilson[29] cites two cases in Great Britain and Denmark where an increase in mastitis occurred in progeny trials as a result of such anticipatory feeding. Another common condition seen particularly in high producing heifers and young cows is preparturient oedema which can cause severe discomfort and stress. These are two strong schools of thought as to whether such oedema should be relieved by milking or not.

It is the authors' contention that milking does not improve the condition or relieve the cause. The high feeding level results in an inbalance between arterial supply and venous drainage, aggravated by the dependancy of the organs and tissues. On the first signs of oedema occurring the nutritional plane should be dropped, the rationale being prevention of the condition as the oedema is very resistant to the normal veterinary and managemental techniques applied for its relief. The oedematous tissues are especially liable to become infected and the condition should be regarded as an excellent opportunity of mastitis to occur.

Resistance to Mastitis

This rather loose term is used to define the well known fact that some cows are more prone to develop mastitis than others. This is well illustrated by Okljesa et al[21] by their survey of the incidence of mastitis due to *Streptococcus agalactiae* in a herd over a period of four years: 75 cows never showed evidence of infection, whereas 93 cows became infected once, 57 twice, 28 thrice, 17 four times, five six times and one cow seven times. These authors suggest that selection of "resistant" cows for breeding purposes is indicated.

Undoubtedly there are a considerable number of factors which contribute to this concept of resistance, some of which may be hereditary.

Length of Lactation Period

Lactation over a 300–305 day period has been accepted as a standard for a dairy cow of any worth, yet this period is undoubtedly well beyond normal, natural and physiological limits. Little[15] contends that after the 7–8th month of lactation regression of mammary tissue commences as a result of inadequate hormonal stimulation. Moderate fibrosis results and tissue changes occur which are similar to the "chronic mastitis" of hormonal aetiology in women. The nature of the milk secretion in late lactation is significant: pH elevated above 6.7–6.8, chloride content $>$ 0.14 per cent and a leucocyte count in excess of $10x^6$/ml. — all features displayed in milk from cases of chronic bacterial mastitis.

Pounden[22] was able to demonstrate that milk from cows in early lactation showed a greater resistance to the in vitro growth of *Str. agalactiae* than milk from cows in late lactation. Similarly, young cows produced milk with greater resistance to in vitro growth of *Str. agalactiae* than cows after multiple lactations.

Little[15] agrees that a short interlactation period resulting in inferior colostrum detrimental to the health of the new-born calf, is further proof of the harmful effect of prolonged lactation.

Condition of the Teat Orifice and Duct System

The normal, intact, functionally efficient teat mechanism is an essential barrier to the establishment of ascending infections of the udder. Glättli[11] has postulated that the increase in the incidence of mastitis in older cows and those producing large quantities of milk is at least partly due to the concomitant increase in size and patency of the teat duct system as necessitated by the fact that such cows can be emptied of their milk in as short a time as low producing cows (Harshberger[12]).

Ulceration of the teat orifice, inflammatory processes of the ductus papillaris or "strich-kanal", accidental lacerations, bruising, surgical interference etc. all contribute to the breakdown of the defense system by—

(*a*) interfering with the physical function of the orifice; and

(*b*) providing a suitable site for bacterial growth.

The epithelium of the streak canal produces a keratin-like substance. Adams and Rickard[1] have shown that this material is in fact sebaceous

in character and they have suggested the name "lactosebum". They have established that it has clear bactericidal effects on *Str. agalactiae*, depending particularly on the nature and quantity of lactosebum. The bactericidal effect is the result of the different long chain fatty acids present in the material, and these authors have established a relationship between resistance of cows to artificial infection with *Str. agalactiae* and the quantity and nature of the lactosebum present. It is suggested that the ability to produce highly bactericidal lactosebum might be hereditary. Interference with the secretion of lactosebum by injuries etc. to the teat extremely may well be held partially responsible for the bacterial mastitis which is frequently a sequal. Injuries to the upper parts of the udder would fall into a different category.

The Bacterial Flora of the Environment

For reasons as yet not fully understood physical injury to tissue reduces its resistance to bacterial infection. Conversely, pathogenic organisms must be present in numbers sufficient to overcome natural barriers to the establishment of infection, and Neave et al[19] have shown that only 25 per cent of dry cows developed mastitis after the teats had been dipped into a suspension of $3\text{–}25 \times 10^6$ mastitis organisms/ml.

There is often considerable difficulty in distinguishing between pathogenic and non-pathogenic organisms where mastitis is concerned, and it is probably at least partially true to say that most organisms may under certain circumstances produce some degree of mastitis. Sometimes saprophytes are introduced into the udder in the course of treatment for a primary pathogenic infection. The rôle of the staphylococci is becoming increasingly important in the mastitis complex, and yet there is considerable confusion over the classification of pathogenic and non-pathogenic types. Joshi and Drury[14] question the use of the haemolysin/coagulase characteristics to indicate staphylococcal pathogenicity, and they have shown that artificial irritation of an udder shedding non-haemolytic staphylococci will cause the organisms to become haemolytic in character within 24 hours. The presence of haemolytic coagulase positive staphyloccoci in quarters showing no evidence of mastitis, not even an increased leucocyte count, (the so-called carrier) is the exact opposite of the cow with an obvious mastitis in which only staphylococcus "epidermidis" can be demonstrated.

The variety of organisms which have been implicated in mastitis is constantly growing and include corynebacteria, streptococci, staphylococci, coliforms, clostridia, mycoplasma, klebsiella, pneumococci, listeria, leptospira, serratia, bacilli, pasteurellae, yeasts and fungi. Baker et al[2] and Bannister et al[3] have shown that viruses are implicated as well.

The nature of these organisms is significant in regard to the source of infection. For example, *Str. agalactiae* may be eliminated from a herd by treatment, segregation, and removal of infected cows, whereas staphylococci occur and grow in the nasopharynx, vagina, and intact skin as well as in the infected quarter. The latter however remains the main reservoir of infection and descending infections from other sources are uncommon.

Interestingly enough, human sources of staphylococcal infections are not unlikely. Wallace et al[25] have demonstrated the phage type 80/81 *Staphylococcus* in a family closely associated with a herd in which mastitis caused by the same organism was present, and Moeller et al[18] have concluded from a survey that transfer of "Hospital" staphylococci from man to farm animals can and does take place.

Reduction of the numbers of organisms as against actual or complete sterilisation of a surface plays an important rôle in minimising the risk of bacterial udder infections. Davidson[9] has demonstrated the efficiency of using clean running water for rinsing out teat cup units between cows. By combining this sytem with udder infusions and the use of a 1:5,000 solution of chlorhexidine for washing teats and udders, he has been able to eliminate the source of mastitogenic staphylococci from a herd (with the exception of the skin of the bull and one quarter which did not respond to intra-mammary treatment) within a year.

The presence of staphylococci on the skin of the teat can become of major importance where faulty milking machines are used. Whittlestone[28] describes how perished teat cup liners harbour such organisms, and how under certain circumstances milk may flow back into the teat from the teat cup. Milk may also flow from one quarter into another in certain types of machines when one quarter empties before the others.

The Immunity of the Cow Against Common Mastitogenic Organisms

This may of course be natural or acquired. Derbyshire[10] points out that the mammary epithelium of the normal cow is relatively impervious to circulating antibodies and mild mastitis may occur even in animals with high serum titres of protecting antibodies. However, the nature of the mammary secretion becomes more like blood as the severity of injury increases, and blood components such as serum albumin and serum globulin begin to appear (Waite and Blackburn[27]).

Nicolet [20] has also shown that as the degree of mastitis becomes more severe so the immuno-electophoresis diagram of the milk becomes more like that of blood.

The antitoxigenic response of cows to chronic and acute forms of staphylococcal udder infections has been studied by Brown[6]. He found that in general, the α and β antitoxin titres increased with the number of chronically infected quarters. In cases of acute staphylococcal mastitis, the blood α and β antitoxin titre increased and reached maximum levels 1–2 weeks after onset of mastitis. Nine out of 14 cows however, already had antitoxins present in their blood when the acute mastitis developed.

Brown[6a] also found that coagulase negative non or weakly haemolytic udder micrococci did not stimulate the production of blood α-antitoxins. Some cows, free of staphylococcal infection of the udders, did show low (>5 I.U./ml.) serum antitoxin titres, but this was probably due to the presence of pathogenic staphylococci on the perineum, in the vagina or elsewhere.

Brown[6] established that only highly toxigenic staphylococci caused acute mastitis with systemic reactions. Organisms producing only β

toxin were responsible for acute mastitis without systemic reactions. No relationship could however be established between the level of blood antitoxin and the pathogenicity of the infecting organism.

Slanetz[25] records the value of staphylococcal toxoid in immunizing cows against the acute forms of staphylococcal mastitis, but the responses to such vaccination appear variable. In any event it appears that even high titres of antitoxin (64 u/ml blood) will not reduce the *incidence of infection* but simply minimises the *effects of toxin* or the tissues. Derbyshire[10] points out that the staphylococci pose a complicated immunological problem since in addition to possessing numerous bacterial antigens the organism also produces a variety of diffusable substances such as toxins, enzymes, haemolysins, etc. He also suggests that stimulation of the local immunity of the udder may be a more rewarding approach to the problem of increasing resistance to infection.

Conclusions

The delicate balance which exists between obtaining maximum production of milk and the partial or complete breakdown of the cow's natural defences against the effects of stress is dependant upon a variety of factors, many of which are as yet imperfectly understood. These include hereditary characteristics, nature of herd management, acquired and natural resistance to bacterial invasion, the presence and number of both pathogenic and saprophytic organisms, and so forth.

As selection and management improve the milk potential of the producing cow that practical and economic necessity of machine milking becomes apparent. Unless udder damage by physical and microbiological agents is kept down to a degree where the cow's natural or acquired resistance may adequately deal with it, mastitis may cause breakdown of the cow as an efficient milk producing entity. As chemotherapy is at best an effort at salvage, it remains for man to design, maintain and use mechanical methods of milking in such a way that damage to teat tissue, introduction of infection and spread or accumulation of infection is eliminated. For this, further research remains to be done.

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The Chief Veterinary Research Institute is thanked for permission to publish this article.

REFERENCES

1. ADAMS, E. W. and RICKARD, C. G. (1936): The anti-streptococci activity of Bovine Teat Keratin. *Am. J. Vet. Res.*, **24**, 98:122.
2. BAKER and LITTLE (1946). *Proc. Soc. Exp. Biol. N.Y.*, **63**, 406.
3. BANNISTER, G. L., BOULANGER, P. and RICE, CHRISTENE C. (1959): Mastitis prod. experimentally in a cow with an agent of the psittacosis — lympho granuloma group of viruses. *Can. J. Comp. Med.*, **23** (2), 47.
4. BRATLIE, A. O. (1958). Betydningen av Vacuumhoyden i Mjoelkemaskinene for Mjoelketeknikk og Jurhygiene. *Nord. Vet. Med.*, **10**, 128.
5. BRATLIE, A. O., SLAGSVOLD, P., TOLLERUD, S. (1959): Pulserings Hastigheten Ved Maskinmjolking. *Nord. Vet. Med.*, **11**, 759.
6. BROWN, R. W. (1962): Staphylococcal antitoxins in Dairy Cattle II & III. *Am. J. Vet. Res.*, **23**, 251:256:257:261.

6a. BROWN, R. W. and SCHERER, R. K. (1963). Staphylococcal antitoxins in Dairy Cattle IV. *Am. J. Vet. Res.*, **24**, (98) 88:90.

7. CONE, J. F. (1944). *J. Dai. Sc.*, **27**, 215.
8. DAHLBERG, A. C. (1941): *N.Y. (Geneva) Agr. Expt. Sta. Bull.* No. 697.
9. DAVIDSON, IAN (1946): Experiences in Controlling Staph. Mastitis. *Res. Vet. Sc.*, **4**, 1:64.
10. DERBYSHIRE, J. B. (1962): Immunity in Bovine Mastitis. *Vet. Bull.*, 32, 1:10.
11. GLÄTTLI, H. R. (1961): Udder Disease in Bovines — Current Problems. *C.I.B.A., Basle*, Swit erland.
12. HARSHBERGER (1956): Quoted in — Diseases of Cattle. *American Vet. Publications.*
13. HAUKE, N. (1961): Cell content in Normal and Abnormal Milk — a review. *D. Tierärztl., Wschrft.*, **68**, 666:724.
14. JOSHI, H. C., DRURY, A. R. (1962). Pathogenicity of Staphylocci associated with the Bovine Udder. *Veterinarian*, **23**, (1) 40.
15. LITTLE, R. D. (1946): Bovine Mastitis, 252. McGraw Hill Book Co. NY. 1946
16. MAFFEY, J. (1959): Methods for the control of the aspecific type of Bovine Mastitis. *Vet. Rec.*, **71**, 124.
17. MEIGS, E. B., BURKEY, L. A., SAUNDERS, G. P., MORRISON, R., and CONVERSE, H. T., (1949): *Tech. Bull. U.S. Dept. Agric.*, 992.
18. MOELLER, R. W. et al (1963): Transfer of Hospital Staphylococci from Farm Animals to Man. *J.A.V.M.A.*, **142**, 6, 613:617.
19. NEAVE, F. K., and OLIVER, J. (1962): The Relationship between the number of mastitis pathogens on the teats of dry cows, their survival and the amount of intra-mammary infection caused. *J. Dai. Sc.*, **29**, 79.
20. NICOLET, J. (1962): Immuno-electrophoresis of Mastitis Milk. *Milchwschft.*, **17**, 307:362.
21. OKLJESA, B., KOPLJAR, M., DUMANOWSKY, F., and MILUNOVIC M. (1962): Varying Susceptibility of cows udders to Str. agalact. *Vet. Archiv.*, **32**, 107.
22. POUNDEN, W. D. (1952): Resistance of milk to *Strep. agalactiae. Am. J. Vet. Res.*, **13**, 49.
23. RELLER, W. (1955): Über das Vorkommen von Path. anatom. Veränderungen in Euter bei katarrhalisch. Sekretions — störungen. *Dissert. Univ. Bern.*
24. SÖRENSEN, B. (1962): Relation between the Incidence of mastitis and the milking machine vacuum and type of Regulator. *Med. fra Statens Vet. Serum Lab. Denmark*, No. 284.
25. SLANETZ, L. W. (1959): Staph. Bacterium-Toxoid for the Immunization of Dairy Cattle against Staph. Mastitis. *Cynamid Inst. Vet. Bull.*, **1**, 3.
26. WALLACE, G. D., QUISENBERRY, W. B., TANIMOTO, A. H., and LYND, F. T. Bacteriophage type 80/81 Staph. infection in human beings associated with mastitis in dairy cattle. *Am. J. Publ., H.*, **52**, 8:1309.
27. WAITE and BLACKBURN (1963): The relationship between milk-yield composition, and tissue damage in a case of sub-clinical mastitis. *J. Dai. Res.*, **30**, (1) 23.
28. WHITTLESTONE, W. G. (1962): The relation between milking machine practice and bovine mastitis. *Austr. Vet. J.*, **38**, 114.
29. WILSON, C. D. (1958): Factors that predispose to mastitis with special reference to Milking technique. *Vet. Rec.*, **70**, 159.

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IMMUNITY TO *STAPHYLOCOCCUS AUREUS* (ROSENBACH 1884)

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SUMMARY

The various factors concerned in the pathogenesis of staphylococcal infections and their rôle as immunizing agents is reviewed. The important aspects of producing an effective vaccine are summarized and discussed.

INTRODUCTION

Micrococci were first observed by Von Recklinghausen in 1871. Since then the staphylococci have intrigued many bacteriologists and have been the subject of an immense amount of research. Despite all these efforts very little indisputable information is available on the various aspects of their basic biology, pathogenicity and immunity.

From a broad immunological point of view most bacteria of medical and veterinary importance can be divided into two groups, namely:

(i) Those producing their effect by the production of potent exotoxins; and

(ii) Those in which the somatic antigens or endotoxins play the major rôle in pathogenicity and in immunity.

In many cases only a small number of specific antigens are concerned.

In *Staphylococcus aureus* however, there are a large number of exotoxins, enzymes and somatic antigens which all play some rôle in the organism's pathogenicity and are therefore also important as immunizing antigens.

As it is a practical impossibility to incorporate all these antigens into a vaccine, the obvious approach is to determine which of the antigens are most commonly present and which play the most important rôles in the pathogenicity of the organism.

CRITERIA FOR PATHOGENICITY

Many tests for substances produced by staphylococci have been evolved and attempts have been made to correlate their presence with either the origin of the organisms (e.g. isolates from abscesses versus isolates from air), or their virulence for rabbits and mice. The objection to the latter technique is that an organism which is pathogenic for mice and rabbits is not necessarily so for its original host[1].

Coagulase production

This enzyme which has the ability to coagulate citrated or heparinized human and rabbit plasma was first described by Loeb[2]. Of all the tests the best correlation of pathogenicity for rabbits and clinical source have been obtained with coagulase[1].

Smith[3] states that coagulase production is the only single reliable test for pathogenicity. The technique for performing the test is well described by Morrison (1962)[4].

Mannitol fermentation

Although Smith[3] does not consider mannitol fermentation to be a reliable indication of pathogenicity, other authors have found a very close correlation between this property and pathogenicity and coagulase production, especially when the test is carried out anaerobically[5, 6, 7, 8, 9, 10].

This test does, of course, not test a pathogenic property but only a characteristic associated with pathogenicity.

Alpha-haemolysin

Some authors consider the presence of alpha-haemolysin to be an even better indication of pathogenicity than coagulase and it has been found to be the most important factor in producing lesions in mice[11, 12].

Although alpha-haemolysin is correlated with virulence it does not lyse human red blood cells nor does its leucocydal effect affect human leucocytes[1].

This probably does not hold as strictly for animals.

Delta-haemolysin

As an indicator of pathogenicity delta toxin rates with alpha[13].

Leucocydin

Leucocydin and leucolysin may be important if they effect the cells of the specie concerned[1].

Lipolytic like substances

These enzymes, which are only produced by coagulase positive staphylococci[14], can be demonstrated by the development of opacity on tellurite egg-yolk media.[15, 16] The results of egg yolk agar tests could however not be correlated with coagulase production by Kimler.[10]

Serotypes

Pathogenic and non-pathogenic strains can be differentiated by agglutination and precipitin tests. Cross reactions occur and this method is not very satisfactory.[17]

Other diffusible products

Pathogenic staphylococci produce various diffusible products, e.g. proteolytic enzymes, haemolysins, and others. Many of these substances

undoubtedly also play a rôle in the organism's pathogenesis but their absence does not mean that the organism is non-pathogenic.[3]

Atypical pathogenic Staphylococci

If a staphylococcus conforms to the usual criteria for pathogenicity it is undoubtedly pathogenic but absence of these criteria does not make it avirulent.[3] This is further borne out by the work of Richou et al (1962).[18] Of 87 strains of staphylococci isolated from the liver and bone marrow of a large number of animal species, only ten showed the in vitro characteristics of pathogens. After examining a herd outbreak of mastitis Joskin & Drury (1963)[19] state that the knowledge of haemolytic activity and coagulase production are not sufficient criteria to judge the pathogenicity of staphylococci and that non pathogenic strains, as judged by in vitro tests, may become pathogenic under suitable circumstances. Staphylococci which do not produce coagulase nor ferment mannitol have been isolated repeatedly in pure culture from clinical cases of mastitis.[20]

In the light of the above knowledge it is obvious that the words of Elek (1959)[1] "The elements of staphylococcal virulence at present defy analysis", still hold good.

Immunogenic Antigens

After having considered the criteria for virulence it is now necessary to more closely examine the various somatic and extracellular antigens of *Staphylococcus aureus* with regard to the rôle they play in pathogenesis, and to what degree they are able to stimulate the production of protective antibodies.

Toxins

Pathogenic strains of Staphylococci produce one or more of a number of exotoxins. Amongst these there are four antigenically distinct haemolysins.[21] It is now generally accepted that the dermatonecrotic, leucocidal (rabbit cells only) and lethal properties of crude staphylococcal toxin are only other manifestations of the alpha haemolysin.[22, 23]

Colonies of staphylococcus showing haemolysis should however not be judged as pathogenic without conducting the necessary inhibition tests, as non-pathogenic micrococci also produce haemolysins.[24, 25] The various toxins may be identified by serum inhibition — and precipitin tests.[26, 27] Haemolysis of red blood corpuscles can also be caused by lipases.[28]

Pathogenic strains also produce a non haemolytic leucocidin and enterotoxins (vide infra).

Alpha-haemolysin

The alpha-haemolysin is a protein exotoxin[29] and is produced in vivo[30] and in vitro by 93–96 per cent of coagulase positive human strains,[25, 31, 32] and by 59 per cent of animal strains.[25]

Carbon dioxide is necessary for the production of alpha-haemolysin[33] and on blood agar plates it produces a large hazy zone of haemolysis.[29] If neutralization tests are not carried out, it may be confused with the haemolysins of coagulase negative strains[24] from which it is antigenically distinct.[1]

It primarily acts against rabbit red cells but also lyses those of the sheep and cow.[34] It is leucocidal, lethal and dermatonecrotic.[23] Chelating agents inhibit the toxin[35] which has maximum activity in phosphate buffered saline at pH 6.8–7.0.[36]

Alpha haemolysin is heat stabile and can be kept for long periods at −5°C or dried.[7, 37] It is absorbed by Zeitz filters[7] and can be toxoided.[38]

Although alpha-haemolysin is considered to be one of the most important factors in the pathogenesis of staphylococcal lesions[12] the amount of toxin produced by various strains is no criterium of their relative pathogenicity.[39]

Normal non-infected animals contain anti-alpha-haemolysin in their sera[40] which show marked individual and species variation. The following average values have been found: Cattle 10 units/ml, sheep 5.1 units/ml, horse 52.4 units/ml.[39] These findings have been confirmed in sheep.[42]

Conflicting results have been obtained regarding the value of alpha-haemolysin as an immunizing agent. Using toxoid alone some authors have been able to protect mice and rabbits against challenge with virulent staphylococci.[43, 44, 45] On the whole, however, poor results have been obtained with pure toxin.[1, 46, 47] It should be remembered that to rabbits alpha-toxin is a leucocidin and advantages that hold for them may not hold for man and ruminants.[48, 49]

Much better results have been obtained with the use of cells plus toxoid or whole cultures as immunizing agents.[49, 50, 51] Other authors claim that antitoxin levels are no indication of resistance.[52, 53] In these instances the antitoxin levels were however, very low.

With our present knowledge it is obvious that antitoxic immunity, although important, is only one aspect of defence against staphylococci.[54, 55]

Beta-haemolysin

The beta-haemolysin is primarily active against sheep and cattle red blood corpuscles and shows a typical hot cold lysis.[56, 57, 58]

This phenomenon can be abolished by lipases, proteases, .15 per cent glycerine, streptococcus agalactiae toxin and pasteurella haemolytica toxin.[57, 58, 59] It will also lyse human erythrocytes.[60]

Beta-haemolysin inhibits the action of alpha-haemolysin on sheep cells but potentiates that of deltathaemolysin.[1, 29] This often gives rise to confusing haemolytic patterns on blood plates.[25, 61] Beta-haemolysin requires small amounts of Mg^{++}, Mn^{++}, Co^{++} and Ni^{++} to act.[62, 63]

The haemolytic toxin active on sheep cells has now been found to consist of two lysins. The one giving a hot cold lysis and the other not.[63]

It is not dermatonecrotic but erythrogenic, lethal to rabbits[57] and enterotoxic[64]

It is heat stabile,[7] can be toxoided[31] but cannot be precipitated with ethanol, methanol or acetone.[65]

The production of beta-haemolysin is promoted by the presence of CO_2 but it is not dependant on it.[60]

Beta-haemolysin is mainly produced by animal strains of Staphylococcus aureus.[31, 66] It is produced by 89 per cent of canine strains.[59]

The use of beta-toxin in staphylococcal vaccines has been advocated, and it is probably of considerable importance as an immunizing antigen in animals, especially ruminants.[67, 68, 69, 70]

Gamma-haemolysin

This toxin is only produced in the presence of carbon dioxide. It is primarily active against rabbit cells and gives a narrow clear zone of haemolysis with a sharp edge on blood agar plates.[71] This toxin is designated "delta" by van Heyningen 1954,[29] which is rather confusing.

Delta-haemolysin

The delta-haemolysin is primarily active against horse erythrocytes but also lyses those of the rabbit, human and sheep.[1, 72] It shows potentiation when acting on sheep red cells exposed to beta-haemolysin.[29, 1] This lysin is produced by 95 per cent of canine strains[59] and by a high percentage of other animal strains.[1] As an indicator of pathogenicity it rates with alpha-haemolysin[13] and may be identical with the leucolysin of Gladstone & v. Heyningen[48, 73, 74]. If this is so, the delta-haemolysin should be significant as an immunizing antigen for animals.

Enterotoxins

Enterotoxin is produced by certain coagulase positive staphylococci — 64 per cent of them belonging to phage and serological type 6/47, IIIC.[75]

These toxins, of which there are three types, are distinct from the rabbit and sheep lysins, and are responsible for food poisoning. They are able to elicit emesis in cats.[63]

Leucocidins

Three factors have been identified which have an action on leucocytes:—

(i) The first is the leucocidin of Neisser & Wechsberg[76] which is identical with the alpha-haemolysin.[22, 23, 71] This toxin is active against rabbit white blood cells only.

As far as its immunological importance is concerned, the same applies as for alpha-haemolysin.

(ii) Panton-Valentine leucocidin.[78, 79] This leucocidin is active against both rabbit and human white cells and it may be of immunological importance in man.

(iii) Leucolysin. This toxin is lytic to the white blood cells of all species except sheep. It is antigenically distinct from the alpha-, beta- and gamma-toxins and may be identical with the delta-haemolysin.[48, 73, 74]

Dermatonecrotic toxin

Apart from the alpha-haemolysin, which is dermatonecrotic (vide supra) a second dermatonecrotic toxin has been demonstrated in culture filtrates which has no lytic ability.[80]

Lethal toxin

The lethal effect of crude staphylococcal toxin to rabbits and mice on intravenous injection is primarily due to the alpha-haemolysin. The beta-haemolysin is also lethal.

Extracellular Enzymes

Coagulase

Coagulase is considered to be one of the best indications of pathogenicity of *S. aureus* and plays a prominent part in the pathogenesis of the organism. (Vide supra). It acts by preventing phagocytosis.[1, 81] There are two types of coagulase namely bound and free which are antigenically dissimilar.[82] Four antigenic types which are linked with phage groups have been identified.[1, 83, 84, 85, 86, 87]

Coagulase can be produced by suitable strains under specific conditions.[88, 89, 90, 91, 92] Its formation is inhibited by carbon dioxide and glucose [1] and stimulated by serum.[93]

Rabbits can be protected against infection by immunization with coagulase rich preparations but only with phage group III coagulase against challenge with the homologous strain.[92, 94, 95]

In cattle it has been found that detoxified coagulase did not stimulate the production of antibodies and that immunity could not be correlated with anti-coagulase.[49]

Hyaluronidase

Hyaluronidase is an antigenic enzyme.[96] A critical amount is necessary to enhance the organism's pathogenicity in the early stages of infection.[1] Antibodies to hyaluronidase are present in the serum of normal carriers.[96] It probably plays only a minor rôle as far as immunity is concerned.

Fibrinolysin

Most coagulase positive strains of *S. aureus* are fibrinolytic but it is not produced by beta-haemolytic strains.[97, 98] It is of doubtful importance in virulence and immunity.

Lipase

Lipase is antigenic and can produce haemolysis of sheep cells in the presence of beta-haemolysin but not alone.[99] There is no evidence that it plays any part in pathogenicity or immunity.[1]

Enzymes related to lipase

These may be tested for by the production of opacity on egg-yolk media[14] (vide supra) and are only produced by coagulase positive strains.

Only a few animal strains produce these enzymes which are of doubtful pathogenic and immunological importance.[100]

Proteolytic enzymes

There are a number of different antigenic types.[101] They are often produced by non-haemolytic and coagulase negative strains isolated from pathological processes in animals.[42] These enzymes may be tested for on laked blood agar[102] or Loeffler serum.

Other enzymes

Many strains produce urease and gelatinase. What rôle they play in pathogenesis is not known.

Somatic Antigens

Agglutinins

The classification and identification of staphylococci by agglutination tests is still in a state of confusion.[28] Three types of antigens have been described by determining their susceptibility to heat and trypsin.[103]

Cowan divided the staphylococci into three types (A, B, & C) by means of slide agglutination tests.[104] These types have been further analysed by using a technique of graduated absorption. They were found to contain three major common antigens which were present in different ratio's in the three types. There were also a number of minor shared and specific antigens. Agglutination reactions were type specific. Cross reactions occurred, but to a much lower titer.[105]

The staphylcocci have also been typed by Oeding[106] and a correlation has been found between the Cowan & Oeding types.[107] Seventeen antigenically distinct agglutinating antigens have thus far been identified.[108]

Only 25 per cent of animal strains could however, be typed with Cowan type antisera.[109]

Precipitins

By gel diffusion methods all strains were found to have six common antigens.[105] Using immuno-electrophoresis both virulent and avirulent strains have been shown to possess seven common precipitating antigens in the endoplasma, and two in the cell wall.[110]

The rôle of somatic antigens in immunity

Some authors have been able to produce immunity using cellular antigens,[46, 47, 111, 112, 113, 114] but other workers have not been able to do so.[115, 116, 117, 118] Agglutinating and precipitating antibodies are considered to be no indication of infection, or immunity[1, 51].

It has however, been repeatedly proven and stressed that the addition of cells to a toxin as an adjuvant is extremely important in order to confer a high antitoxin titer.[42, 43, 50, 51, 68, 119, 120] However, immunity to intra mammary challenge in cows could only be obtained when the homologous strain was used.[51, 102, 121]

Immunity to mammary challenge by a particular strain has been correlated with the power of the serum to protect mice against the same strain. It was clear that cross immunity occurs between certain strains which are of different phage pattern and further that immunological differences occur between strains that have other in vitro characteristics in common.[102, 122]

The use of common bovine phage types (42D, 44A & 81) in vaccines has been advocated as well as various antigenic types.[68, 69, 70]

Although cells on their own do not elicit a durable immunity, it is obvious that various serological types play a very important rôle in conjunction with toxoid in producing immunity.

The relationship of phage types to serological types

Staphylococcal phage types of animal origin differ quite markedly from the human types and the phage set used for humans is not suitable for typing animal strains.[123] There appears to be a broad similarity between phage and serological types[124] and a scheme has been developed by which they can be correlated.[106]

On the whole however, no or very little correlation has been found between phage groups and patterns and the serological groups and patterns of the strains of *S. aureus*.[125, 126]

Opsonins

Opsonins are natural or acquired antibodies which promote phagocytosis. Often by inhibiting the growth of the organisms. Effective phagocytosis is one of the body's most important defence mechanisms, and therefore substances produced by the organism which retard phagocytosis, for example by inhibiting the natural or acquired opsonins, are important as pathogenic and immuno-antigenic substances. Two such substances have been described:

(i) Coagulase.

(ii) A substance present in culture filtrates of coagulase positive staphylococci which may be identical with the beta-toxin of Petterson.[120, 127, 129]

A substance which is antibacterial to staphylococci has been found in normal sera[130] and a growth inhibition test has been developed.[131 132] When the efficiency of vaccines are being tested serologically, attention should be paid to these antibacterial and growth inhibiting factors as they obviously play an important rôle in promoting phagocytosis and therefore in conferring immunity.

Phagocytosis has been claimed to be more rapid in immunized rabbits but this could not always be confirmed.[7, 133, 134, 135]

Antibodies to leucocidal toxins may aid phagocytosis if the toxin effects the white blood cells of the species concerned,[1] In animals the leucolysin (delta toxin?) is probably the most important.[48, 73, 136]

Agglutinins and haemagglutinins have also been found but whether they indicate any immunity or not is not known.[136, 137, 138]

Production and use of Vaccines

Strains

Not all strains are suitable for vaccine production as they differ antigenically and do not all produce the toxins and other extracellular products which are required for vaccine preparation. Strains should be selected for ability to produce the antigens required, and grown under suitable conditions which differ for each antigen.

It has also been found that only about 50 per cent of single colonies of a certain toxogenic strain are suitable for vaccine production. If single colonies are not selected toxin yield is often very poor.[42]

Medium

The simpler the medium and the higher the yield, the more stabile are the antigenic values.[139] Heart brain infusion broth and Tryptose veal broth have been used with excellent results.[68, 69, 70] The medium should be well buffered to obtain optimal toxin production.[42, 140] Serum, agar, and other inert high molecular weight substances appear to promote toxin production.

A medium consisting of beef infusion broth,[141] to which .03 per cent $MgSO_4$[142], .2 gamma per cent thiamine[143], M/80 glucose[144], and M/110 argenine,[144, 145, 146] is added, has given good results. Although Calcium inhibits toxin production[144, 147] it is necessary extracellularly for growth[148] and therefore no attention is paid at present to the amount of this element in the medium. The medium is buffered at pH 7.6 with triss (hydroxymethyl) aminomethane: Maleic acid: Sodium hydroxide buffer[149] and yields 16 haemolytic test doses of alpha toxin and 32 h.t.d. beta-toxin per ml.[42]

Purification of toxin

Crude toxin can be precipitated and concentrated if required by various chemicals. Those which have been used thus far are ethanol,[121] Trichloracetic acid[150], Ammonium Sulphate[49], Aluminium phosphate[151], Zinc acetate and ethanol[63], Metaphosphoric acid[152], Methanol[153], and by electrophoresis.[23, 154]

Toxoiding

Rapid toxoiding is necessary in order to produce a potent toxoid.[155] It has been found that there is an optimal concentration of formaldehyde and an optimal incubation period where a toxin will be detoxified com-

pletely with minimal loss of antigenic properties.[156, 157] Toxoiding with formalin often takes weeks with resultant deterioration in antigenicity.

Toxins toxoided with beta-propiolactone gave better antibody responses than those toxoided with formalin (16.7 units per ml and 12.3 units per ml respectively.)[70] Concentration for concentration formalin is a more potent toxoiding agent than beta-propiolactone but on agar diffusion plates there was qualitative evidence of the deleterious effects of formalin on the antigenicity of resulting staphylococcal toxoids. A progressive obliteration of precipitin bonds was correlated with incubation time and formalin concentration while beta-propiolactone toxoids gave more and clearer precipitin lines.[158]

Adjuvants

The value of adjuvants such as aluminium, hydroxide gel[51], Potassium Alum[159] and Aluminium phosphate[151] has been repeatedly proven.[50]

Much higher antitoxin titres have been obtained with adjuvants, with or without cells, than with toxoid alone.[50, 68, 69, 51]

Response to Vaccines and Immunity

There is a marked variation in the response of individual animals to adjuvant bacterin toxoid.[160, 121, 42] Those with a natural antitoxin titre respond most vigorously.[52, 234, 161, 162]

The normal route of administration is intramuscularly but some authors have obtained better antibody response by intradermal[163] and intra-mammary administration.[164, 165, 166] Although local immunity to the homologous strain has been obtained by udder infusion[102] the udder is probably not a source of antibody production.[167] In any case this method is quite impractical and severe reactions often occur.[268]

The use of repeated doses of vaccine has been advocated.[40] Using three to four large doses, the following average alpha antitoxin titres have been obtained:

64 units/ml — (goats)[49]
512 units/ml — (goats & cattle) (highest value)[51]
16 units/ml — (cattle)[53]
12–96 units/ml — (cattle)[68, 69]
100 units/ml — (rabbits)[70]
30 units/ml — (cattle)[70]
20–30 units/ml — (?)[120]
38.2 units/ml — (cattle)[121]

Using two intramuscular injections of 5 ml each with an interval of 4 weeks, in 24 sheep an average alpha antitoxin titre of 60.3 units/ml has been obtained. The highest titre was found eight days after the first injection and was mainly due to the spectacular response of sheep which had 2–4 units/ml of antitoxin in their sera before administration of the vaccine. The titre fell rapidly and the second injection had little effect.[42] Similar results have been found in goats. During a series of injections of 2, 5 and 5 ml administered at two day intervals, the highest titre was obtained 7 days after the second injection.[51] This same rapid response and decline has also been observed in cattle.[121]

As already stated cattle could be protected against intra-mammary challenge with virulent staphylococci, but only against the strain from which the vaccine was prepared.[49, 50, 51, 102, 121, 122, 169]

Considerable success has been obtained in field trials with staphylococcus vaccines in dairy cows. In most cases a general increased resistance to infection has been found. The number of cases of mastitis diminish and those that do occur are less severe.[53, 68, 69, 121, 170, 171]

Vaccine as treatment

Many authors are of opinion that the use of toxoid vaccine as a means of treatment is of no value whatsoever.[68, 69, 172, 173] Antitoxin therapy in man is also thought to be of little value.[174]

On the other hand it is known that staphylococcus anti-toxin is excreted in the milk[40] and that this excretion takes place within six hours after udder irritation.[169] Antibody response by infected animals to parenteral administration of toxoid is very rapid and the resultant antitoxin in the milk may be of value in that it mitigates the infection.[40, 166] Good results have been obtained by the administration of vaccine to infected cows.[170]

Antibodies have also been found in the milk after intra-mammary infusion of vaccine.[102, 164, 165, 166]

Discussion

In the light of the knowledge concerning the various aspects of staphylococcal immunity at present at our disposal one comes to virtually the same conclusions as Derbyshire.[55]

From the information available two factors stand out clearly:

(1) That animals can be protected against infection by pathogenic staphylococci with suitably prepared adjuvant-cell-toxoid vaccines; but

(2) that such vaccines will only lend protection against infection by the vaccine — or very closely related strains.

It is clear that other antigens apart from alphahaemolysin, also play an important part in conferring a sound immunity. It is probable that the reason why a certain vaccine will not protect against heterologous strains is either due to the fact that extracellular antigens other than alphahaemolysin are responsible for their pathogenicity or that their somatic antigens are dissimilar to those of the vaccine strain.

When attempting to produce a vaccine for general use these aspects should be borne in mind until further research has clarified the position.

The rôle which the various extracellular substances may play as immunizing agents has already been discussed and it now only remains to summarize the salient features.

Beta-haemolysin is produced by a large percentage of animal strains and as it primarily lyses ruminant red blood cells and possibly also inhibits phagocytosis by neutralising opsinins, it should be incorporated in all vaccines intended for animal use. The same applies to delta-haemolysin which also appears to be a leucolysin. Vaccines should of course also contain a potent alpha-haemolysin.

Of the extracellular enzymes coagulase is by far the most important by virtue of the facts that it is the only single reliable criterion for pathogenicity and that it plays an important rôle in the pathogenesis of staphylococci. As the antigenic types of coagulase are associated with phage types it should be derived from a strain belonging to the more common bovine groups namely III & IV or types 42 D, 44 A & 81.

The cellular part of the vaccine is also very important both as an adjuvant and for the somatic antigens (vide supra)[46, 47] and should be composed of strains of the most common antigenic types as determined by agglutination tests.

The various components of the vaccine should be prepared separately from selected strains under suitable conditions. The cells should be killed by heat and the toxins toxoided with beta-propiolactone. After mixing, an adjuvant should be added.

When testing the efficacy of vaccines, tests should be employed which will not only determine antitoxins and agglutinating antibodies but also growth inhibiting antibodies[131, 132] or the phagocytic index.[122, 175]

Further investigation is required to determine the most practical and effective schedule for administering the vaccine.

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REFERENCES

1. ELEK, S. D. (1959): *Staphylococcus pyogenes* and its relations to disease. *E. & S. Livingstone Ltd. Edinburgh & London.*
2. LOEB, L. (1903–1904): The influence of certain bacteria on the coagulation of the blood. *J. Med. Res.*, **10**, 407–419.
3. SMITH, W. H. (1947): Examination of Staphylococci of animal origin with particular regard to determination of criteria of pathogenicity. *J. Comp. Path. & Ther.*, **57**.
4. MORRISON, R. B. (1962): The coagulase test in the identification of pathogenic staphylococci. *J. Appl. Bact.*, **25** (3), 432–435.
5. DUDGEON, L. S. (1908): The differentiation of staphylococci. *J. Path. Bact.*, **12**, 242.
6. JULIANELLE (1937): Determination of staphylococcal types by fermentation of mannite. *Proc. Soc. Exp. Biol.*, N.Y., 117.
7. FLAUM, A. (1938): Studies in staphylococci and staphylococcal immunity. *Acta. Path. et Microbiol.* Scand. Suppl., 33–36.
8. EVANS, J. B. (1947): Anaerobic fermentation of manitol by staphylococci. *J. Bact.*, **54**, 266.
9. EVANS, J. B. (1948): Studies of staphylococci with special reference to coagulase positive types. *J. Bact.*, **55**, 793.
10. KIMLER, A. (1962): Evaluation of Mediums for Identification of *S. aureus Amer. J. Clin. Path.*, 37 (6), 593–596.
11. CHRISTIE, R., NORTH, E. A. & PARKIN, B. J. (1946): Criteria of pathogenicity in staphylococci. *Aust. J. Exp. Biol. Med. Sci.*, **24**, 73–78.
12. SELBIE, F. R. & SIMON, R. D. (1952): Virulence to mice of *Staphylococcus pyogenes*· its measurement and its relation to certain in vitro properties. *Brit. J. Exp. Path.*, **32**, 315.

13. JOIRIS, E. (1952d): Production de la deltahémolysine par les staphylocoques et incidence de celleci sur le diagnostic des souches pathogenes. *Rev. Belge. Path.*, **22,** 185–188.
14. GILLESPIE, W. A. & ADLER, V. G. (1952): Production of opacity in egg-yolk media by coagulase — positive staphylococci. *J. Path. Batc.*, **64,** 187–200.
15. INNES, A. G. (1960): Tellurite egg agar, a selective and differential medium for the isolation of coagulase positive staphylococci. *J. Appl. Bact.*, **23,** 108.
16. BAIRD-PARKER, A. C. (1962): An improved diagnostic & selective medium for isolating coagulase positive staphylococci. *J. Appl. Bact.*, **25,** 12.
17. PILLET, J. & ORTA, B. (1954): Recherches sur la composition antigénique des staphylocoques non pathogenes — I. Mise en évidence d' agglutinogénes spécifiques et d' un agglutinogene commun aux souches pathogénes et non pathogènes. *Ann. Inst. Pasteur,* **86,** 752–758.
18. RICHOU, R. et al (1962): Contribution a l' étudie des Staphylocoques d' origine animale. *Rev. Immunol. et Therap. Anti. microbienne*, **26**(1/2), 55–59.
19. JOSKIN, H. C. & DRURY, A. R. (1963): Pathogenicity of staphylococci associated with the bovine udder. *Veterinarian* **23,** (1) 40.
20. CAMERON, C. M. & VAN DEN HEEVER, L. (1963): *Unpublished Data.*
21. GUIRIN (1962): Tissue culture and Immunological Studies with Purified staphylococcal toxins. *Can. J. Publ. Health*, **53,** 1.
22. LEVINE, B. S. (1939): The unity of the haemolytic dermatonecrotic and lethal properties of staphylococcal exotoxin and their corresponding counterparts in staphylococcal antitoxin. *J. Path. Bact.*, **48,** 291.
23. KUMAR, S. & LINDORFER, R. K. (1962): The characterization of staphylococcal toxins. I. The electrophoretic migration of the alpha, hemolytic, dermatonecrotic, lethal and leucocidal activities of crude toxin. *J. Exp. Med.*, **115**(6), 1095.
24. RUNGE (1950): Über die Pathogenität der Haut staphylokokken. Der Einfluss einer Kurzer Penicillin — Injections behandlung auf die oberflächliche Haut flora. *Arch. Derm. Syph. (Berl.)*, **191,** 427–430.
25. ELEK, S. D. & LEVY, E. (1950b): Distribution of haemolysins in pathogenic & non-pathogenic staphylococci. *J. Path. Bact.*, **62,** 541–554.
26. ELEK, S. D. (1949): The serological analysis of mixed flocculating systems by means of diffusion gradients. *Brit. J. Exp. Path.*, **30,** 484–500.
27. ELEK, S. D. & LEVY, E. (1950a): Diffusable antigens in staphylococcal cultures. *Brit. J. Exp. Path.*, **31,** 358–368.
28. COWAN, S. T. (1962): An introduction to chaos, or the classification of Micrococci and staphylococci. *J. Appl. Bact.*, **25**(3), 324.
29. VAN HEYNINGEN, W. E. (1954): Bacterial toxins. *Blackwell.* Oxford.
30. GLADSTONE, G. P. & GLENCROSS, E. J. G. (1960): Growth and Toxin production of staphylococci in cellophane sacs in vivo. *Brit. J. Exp. Path.*, **41**(3), 313.
31. BRYCE, L. M. & ROUNTREE, P. M. (1936): The production of beta-toxin by staphylococci. *J. Path. Bact.*, **43,** 173–189.
32. GILLESPIE, W. A. & SIMPSON, P. M. (1948): Pathogenic staphylococci; detection of alphalysin production on rabbit and sheep blood agar plates. *Brit. Med. J.*, **2,** 902–903.
33. DUTHIE, E. S. & WYLIE, I. A. H. (1945): The influence of shaking on the in vitro production of soluble bacterial toxins. *Brit. J. Exp. Path.*, **26,** 130–136.
34. WILLIAMS, R. E. O. & HARPER, G. J. (1947): Staphylococcal haemolysins on sheep blood agar with evidence for a fourth haemolysin. *J. Path. Bact.*, **59,** 69–78.
35. ROBINSON, J. & THATCHER, F. S. (1957): The purification and some properties of alpha haemolysin from Micrococcus pyogenes var. aureus. *Bact. Proc.*, **57,** 99.
36. JACKSON, A. W. & LITTLE, R. M. (1957a): Staphylococcal toxins — I. Factors affecting the hemolytic activity of alpha toxin. *Canad. J. Microbiol.*, **3,** 47–54.
37. TAGER, M. (1941): Factors modifying the reaction of alpha staphylolysin to heat. *Yale J. Biol. Med.*, **14,** 68–88.
38. BURNET, F. M. (1929): The exotoxins of *Staphylococcus pyogenes aureus*. *J. Path. Bact.*, **32,** 717–734.
39. TAGER, M. & HALES, M. B. (1947): Quantitative coagulase and toxin production by staphylococci in relation to the clinical source of the organisms. *Yale J. Biol. Med.*, **20,** 41–49.
40. RAMON, G., RICHOU, R., JULIENNE, P., JACQUET, J. & GERBEAUX, C. (1951): Le dévelopment de l' antitoxine, staphylococcique chez les bovidés soumis aux injections d' anatoxine spécifique. *Rev. Immunol.* (Paris), 15, 321–335.

41. SEIFERT, H. (1961): Der antistretolysin -und antistaphylolysingehalt des Serums und Augenkammer wassers der Haus und Labortiere. *Z. Immon. Forsch. u Exp. Ther.*, **121**, 375–382.
42. CAMERON, C. M. (1960, 1961, 1962 & 1963): Unpublished Data.
43. RAMON, G., RICHOU, R. & DJOURICHITCH, M. (1936): Sur la móanisme de l' imunnité conférée par l' anatoxine staphylococcique à l' égard de l' infection par le staphylocoque virulent. Démonstration experimentale. *C.R. Soc. Biol. (Paris)*, **122**, 1164–1168.
44. DELAUNEY, A. (1938): L'immunite antistaphylococcique conférée par l' omatoxine specifique et son mécanisme. *Rev. Immunol.*, (Paris), **4**, 65–96.
45. FRAPPIER, A. & SONEA, S. (1956): L'immunité dans les infections staphylococciques — I. Action de' l' antitoxine contre une infection bacterienne experimentale. *Canad. J. Microbiol.*, **2**, 271–280.
46. CRAWLEY, J., TAYLOR, P. & BARNUM, D. A. (1962): *Unpublished Data.*
47. BARNUM, D. A. (1962): A method of control of Staphylococcal and Streptococcal mastitis in Ontario. *Can. Vet. J.*, 3(6), 161–169.
48. GLADSTONE, G. P. & VAN HEYNINGEN, W. E. (1957): Staphylococcal leucocidins. *Brit. J. Exp. Path.*, **38**, 123–137.
49. DERBYSHIRE, J. & HELLEWELL, B. I. (1962): Immunity to experimental staphylococcal Mastitis in Goats produced by alpha lysin, coagulase and leucocidin. *Res. Vet. Sci.*, 3 (1).
50. SPENCER, G. R., STEWART, J. H. & LASMANIS, J. (1956): Preliminary report on immunization of animals against *Micrococcus pyogenes*. *Amer. J. Vet. Res.*, **17**, 594.
51. DERBYSHIRE, J. B. (1960a): Studies in immunity to experimental Mastitis in the goat and cow. *J. Comp. Path. & Therap.*, **70**, 222–231.
52. FORSMANN, J. (1937a): Studies in staphylococci — IX. On the mechanism in staphylococcal infection and immunity. *Acta. Path. Microbiol. Scand.*, **14**, 468–477.
53. PEARSON, J. K. L. (1959): Autogenous Toxoid Vaccine in the prophylaxis of staphylococcal Mastitis in cattle. *J. Dairy Research.*, **26**(1), 9–16.
54. PILLET, J., BERROD, J., GOSSET, A. & ORTA, B. (1956): Immunization active et staphylococcus chroniques chez l' homme. *Ann. Inst. Pasteur*, **90**, 233–237.
55. DERBYSHIRE, J. B. (1962): Immunity in Bovine Mastitis. *Vet. Bul.*, **32** (1), i.
56. GLENNY, A. T. & STEVENS, M. F. (1935): Staphylococcus toxins and antitoxins. *J. Path. Bact.*, **40**, 201–210.
57. SMITH, M., LLEWELLYN & PRICE, S.A. (1938a): The combining power of staphylococcus toxoid: the Danysz effect and other factors influencing its determination. *J. Path. Bact.*, **46**, 289–302.
58. SMITH, M., LLEWELLYN & PRICE, S.A. (1938b): Staphylococcus beta haemolysin. *J. Path. Bact.*, **47**, 361–377.
59. FRAZER, G. (1962): The haemolysis of Animal Erythrocytes by Pasteurella haemolytica produced in conjunction with certain staphylococcal toxins. *Res. Vet. Sci.*, **3** (1), 104.
60. CHRISTIE, R., & NORTH, E. A. (1941): Observations on staphylococcal beta toxin and its production by strains isolated from staphylococcal lesions in man. *Aust. J. Exp. Biol. Med. Sci.*, **19**, 323–327.
61. ELEK, S. D. & LEVY, E. (1954): The nature of discrepancies between haemolysins in culture filtrates and plate haemolysin patterns of staphylococci. *J. Path. Bact.*, **68**, 31-40.
62. PEDERSEN, P. S. (1960): Bacteriological diagnosis of Mastitis III. Titration of staphylococcal Beta-toxin. *Nord. Vet. Med.*, **12**, 749–768.
63. THATCHER, F. S. & ROBINSON, J. (1962): Food poisoning: an analysis of staphylococcal toxins. *J. Appl. Bac.*, **25** (3), 378–388.
64. FULTON, F. (1943): Staphylococcal enterotoxin — with special reference to the kitten test. *Brit. J. Exp. Path.*, **24**, 65–73.
65. KODARIA, T. & KAJINA, T. (1939): Studies on staphylococcal toxin, toxoid and antitoxin. — II. Properties of alpha and beta haemalysin. III. The effect of ascorbic acid upon staphylococcal lysins and organisms. *Kitasato Arch. Exp. Med.*, **16**, 36–55.
66. STOMATIN, N., TACU, M. A. & MARICA, D. (1949): L' activité hémolytique et coagulante des staphylocoques isolés à partir des infections humaines ou animales. *Ann. Inst. Pasteur*, **76**, 178–180.
67. RAMON, G. & RICHOU, R., JULIENNE, P. & JACQUET, J. (1953): De l' influence comparée des suspensions de staphylocoques et de colibacilles tués (anavaccins) et de l'alum de potassium sur l' immunité engendrée, chez les bovidés, par l' anatoxine staphylococcique. *Bull. Off. Inst. Epiz.*, **39**, 610.

68. SLANETZ, P. M., BARTLEY, CLARA, M., & ALLAN, F. E. (1959): The immunization of Dairy Cattle against Staphylococcal Mastitis. *J. Amer. Vet. Med. Ass.*, **134** (4), 155–161.
69. SLANETZ, P. M. (1959): Staphylococcal Bacterin Toxoid for the immunization of dairy cattle against Staphylococcal Mastitis. *Cyanamid. Internat. Vet. Bul.*, **3.**
70. FLEMMING, L. W. (1960a): Studies on Bovine staphylococcal mastitis. II. Formulation of immunizing agents. *Vet. Med.*, **55** (10), 72.
71. SMITH, M. L. & PRICE, S. A. (1938c): Staphylococcal gamma haemolysin. *J. Path. Bact.*, **47,** 379.
72. MARKS, J., & VAUGHAN, A. C. T. (1950): Staphylococcal delta haemolysin. *J. Path. Bact.*, **62,** 597–615.
73. JACKSON, A. W. & LITTLE, R. M. (1956): The delta-toxin of *Micrococcus pyogenes var. aureus. Bact. Proc. 56th Gen. Meet.*, p. 88.
74. JACKSON, A. W. & LITTLE, R. M. (1957b): Leucocidal effect of staphylococcal delta lysin. *Canad. J. Microbiol.*, **3,** 101–102.
75. ALLISON, V. D. (1949): Discussion on food poisoning. *Proc. R. Soc. Med.*, **42,** 214–220.
76. NEISSER, M. & WECHSBERG, F. (1900): Über eire neue einfache methode zur Beobachtung van Schädigungen lebender Zellen und Organismen (Bioskopie). *Munch. Med. Wsch.*, **47,** 1261–1262.
77. WRIGHT, J. (1936): Staphylococcal leucocidin (Neisser-Wechsberg type) and anti-leucocidin. *Lancet*, **1,** 1002–1004.
78. PANTON, P. N. & VALENTINE, F. C. O. (1929): Staphylococcal infection and re-infection. *Brit. J. Exp. Path.*, **10,** 257–262.
79. PANTON, P. N. & VALENTINE, F. C. O. (1932): Staphylococcal toxin. *Lancet*, **1,** 506–508.
80. ROBINSON, J., THATCHER, F. S. & MAITFORD, J. (1960b): Studies with Staphylococcal toxins. VI. Some properties of a non-haemolytic dermatonecrotic toxin produced by some strains of S. aureus. *Canad. J. Microbiol.*, **6,** 195.
81. FOSTER, W. D. (1962): The role of coagulase in the pathogenicity of S. aureus. *J. Path. & Bact.*, **83,** 287.
82. DUTHE, E. S. (1954a): Evidence of two forms of staphylococcal coagulase. *J. Gen. Microbiol.*, **10,** 427–436.
83. TAGER, M. (1949): Concentration, partial purification and properties, and nature of Staphylocoagulase. *Yale, J. Biol. Med.*, **20,** 487–501.
84. RAMMELKAMP, C. H., HEZEBICKS, M. M. & DINGLE, J. H. (1950): Specific coagulases of *S. aureus. J. Exp. Med.*, **91,** 295.
85. BARBER, M., & WILEY, P. (1959): A study of the Antigenic Specificity of staphylococcal coagulase in Rotation to Bacteriophage Group. *J. Gen. Microbiol.*, **18,** 92.
86. DUTHIE, E. S. (1952): Variation in the antigenic composition of staphylococcal coagulase. *J. Gen. Microbiol.*, **7,** 320–326.
87. DUTHIE, E. S. & LOVENZ, L. L. (1952): Staphylococcal coagulase: mode of action and antigenicity. *J. Gen. Microbiol.*, **6,** 95–107.
88. LOMINSKI, I. (1944): Preparation of cell-free plasma coagulase of staphylococcus aureus. *Nature* (*Land*), **154,** 640.
89. DUTHIE, E. S. & HAUGHTON (1958): Purification of free staphylococcal coagulase. *Biochem. J.*, **70,** 125, Sun. Staph.
90. DUTHIE, E. S. (1954b): The production of free staphylococcal coagulase. *J. Gen. Microbiol.*, **10,** 437–444.
91. BLOBEL (1960): Purification of staphylococcal coagulase. *J. Bact.*, **79,** 807.
92. LOMINSKI, I. (1962): Immunization against staphylococcal infection with coagulase rich preparations. *Lancet*, 1962, *No.* 7243, **23,** 1315–1318.
93. DAVIES, G. E. (1951): Factors influencing the in vitro production of staphylococcal coagulase. *J. Gen. Microbiol.*, **5,** 687–697.
94. LOMINSKI, I. (1949): Susceptibility and resistance to staphylococcal infection. *J. Gen. Microbiol.*, **3,** Suppl. p. IX.
95. BOAKE, W. C. (1956): Anti-staphylocoagulase in experimental staphylococcal infections. *J. Immunol.*, **76,** 89–96.
96. HOWELL, D. G. (1954): Inhibitors to staphylococcal and streptococcal hyaluronidase in the serum of the dairy cow. *J. Path. Bact.*, **68,** 287–289.
97. CHRISTIE, R., & WILSON, H. (1941): A test of staphylococcal fibrinolysis. *Aust. J. Exp. Biol. Med. Sci.*, **19,** 329–332.
98. ROUNTREE, P. M. (1947): Some observations on fibrinolysin and beta toxin of staphylococci. *Aust. J. Exp. Biol. Med. Sci.*, **25,** 359.

99. CHRISTIE, R. & GRAYDON, J. J. (1941): Observations on staphylococcal haemolysin and staphylococcal lipase. *Aust. J. Exp. Biol. Med. Sci.*, **19**, 9–16.
100. ALDER, V. G., GILLESPIE, W. A. & HERDAN, G. (1953): Production of opacity in egg-yolk broth by staphylococci from various sources. *J. Path. Bact.* **66**, 205–210.
101. LEVY, E. (1952): Diffusible products of staphylococci. *Thesis. University of London.*
102. DERBYSHIRE, J. B. (1961b): The immunization of Goats against staphylococcal mastitis by means of experimental infections of the skin and udder. *Res. Vet. Sci.*, **2**, 112.
103. ANDERSEN, E. K. (1943): Studies on the specificity of pemphigus staphylococci. *Acta. Path. Microbiol. Scand.*, **20**, 242.
104. COWAN, S. T. (1939): Classification of staphylococci by slide agglutination. *J. Path. Bact.*, **48**, 169.
105. STERN, H., & ELEK, S. D. (1957): Antigenic structure of *Staphylococcus* pyogenes. *J. Path. Bact.*, **73**, 473–484.
106. OEDING, D. (1952): Serological typing of staphylococci. *Acta. Path. Microbiol. Scand.* Suppl., **93**, 356.
107. OEDING, D. (1953b): Serological typing of staphylococci — II. Investigations on the serological, chemical and physical properties of antigens. *Acta. Path. Microbiol. Scand.*, **33**, 312–323.
108. PILLET, J., ORTA, B. & PERRIER, M. (1962): Recherches sur l' individualisation sérologiques des staphylococcus Mise en évidence de deux nouveaux agglutinogènes specifiques. *Ann. Inst. Pasteur.*, T.**101** (4), 590.
109. PILLET, J., ISBIR, S. & MERCIER, P. (1950): Etude biologique des staphylocoques d' origine animale. *Ann. Inst. Pasteur.*, **78**, 638–643.
110. YOSHIDA, H. & HEDÉN, C. G. (1962): Some serological properties of staphylococcal cells and cell walls. *J. Immunol.*, **88**, 389–392.
111. FORSMANN, J. (1938c): Studies in staphylococci XIII. A further contribution to the understanding of the immunity to staphylococci. *Acta. Path. Microbiol. Scand.*, **15**, 396–425.
112. FARREL, L. N. & KITCHING, J. S. (1940): Passive protection of mice with immune horse serum against living staphylococci. *J. Path. Bact.*, **50**, 439–453.
113. GREENBERG & COOPER (1960): *Canad. Med. Ass. J.*, **83**, 143.
114. STAMP, L. (1961): Antibacterial Immunity to *Staphylococcus pyogenes* in Rabbits. *Brit. J. Exp. Path.*, **42**, 30.
115. RIGDON, R. H. (1937f): Staphylococcic immunity résumé of experimental and clinical studies. *Arch. Path.* (Chicago), **24**, 233–245.
116. DOWNIE, A. W. (1937): A comparison of the value of heat-killed vaccines and toxoid as immunising agents against experimental staphylococcal infection in the rabbit. *J. Path. Bact.*, **44**, 573–587.
117. KENDO, K. (1938): Über die Antigenität von mit hoher Hitze behandelten staphylokokken. *Nagoya Igakkai Zassi*, **48**, 579.
118. PETTERSON, A. (1946): Die Immunotherapie bei staphylokokken-krankheiten. *Acta. Path. Microbiol. Scand.*, **23**, 164–169.
119. RICHOU, R., GERBEAUX, C., & SCHLAEPFER, J. (1949): De l' influence des suspensions de Bacillus abortus sur l' accroissement de l' immunité engendrée par l' anatoxine staphylococcique. *C.R. Acad. Sci.* (*Paris*), **229**, 858–860.
120. RAMON, G., RICHOU, R., JULIENNE, P., JACQUET, J., GERBEAUX, C., & LENEVEU, G. (1952): De l' influence des suspensions de streptocoques et de staphylocoques tués (anavaxin) sur l' immunité engendrée chez des bovides, par l' anatoxine staphylococcique. *Of. Int. Des. Epiz. Bull.*, **37**, (9–10), 514–529.
121. BLOBEL, H. & BERMAN, D. (1962): Vaccination of Dairy Cattle against staphylococcal Mastitis. *Amer. J. Vet. Res.*, **23** (92), 7–14 Sm. p. 27.
122. DERBYSHIRE, J. B. (1961a): Further immunological studies in exp. staph. mastitis. *J. Comp. Path.*, **71**, 146–158.
123. PARKER, M. T. (1962): Phage-typing and the epidemiology of *staphylococcus aureus* infection. *J. Appl. Bact.*, **25** (3), 389–402.
124. PILLET, J., DALMELS, J., ORTA, B. & CHABANIER, G. (1954): Etude comparée du type sérologique, de la sensibilité aux bacteriophages et de l' antibiogramme de 201 souches de staphylocoques isolés par prélévement systematiques chez le nourisson. *Ann. Inst. Pasteur*, **86**, 309–319.
125. OEDING, P. & VOGELSANG, T. M. (1954): Staphylococcal studies in hospital staffs — V. Comparison between serological typing and phage typing. *Acta. Path. Microbiol. Scand.*, **34**, 47–56.

126. SLANETZ, L. W. & BARTLEY, C. M. (1962): Bacteriophage and serological typing of Staphylococci from Bovine Mastitis. *J. Inf. dis. Vol.*, **110** (3), 238.
127. EKSTEDT, R. D. & NUNGESTER, W. J. (1955): Coagulase in reversing antibacterial activity of normal human serum on *Micrococcus pyogenes*. *Proc. Soc. Exp. Biol.* (N.Y.), **89**, 90–94.
128. EKSTEDT, R. D. (1956a): Further studies on the anti-bacterial activity of human serum on Micrococcus pyogenes and its inhibition by coagulase. *J. Bact.*, **72**, 157–161.
129. EKSTEDT, R. D. (1956b): The effect of coagulase on the anti-bacterial activity of normal human serum against selected strains of *Micrococcus pyogenes*. *Ann. N.Y. Acad. Sci.*, **65**, 119–131.
130. YOTIS, W. W. (1962): Effect of the antibacterial serum factor on Staphylococcal infections. *J. Bact.*, **83** (1), 137–143.
131. BOGER, W. P., FRANKEL, J. W. & GARIN, J. J. (1960): Detection and Titration of *S. aureus* agglutinin in serum. *Proc. Soc. Exp. Biol. & Med.*, **104**, 639–642.
132. SPENCER, R. G. (1963): The use of a growth agglutination test for the detection of antibodies to S. aureus in cattle. *Am. J. Vet. Res.*, **24** (98), 83–87.
133. PETTERSON, A. (1937): Weitere Untersuchungen über die Immunität gegen die pyogenen staphylokokken. *Z. Immunafrsch.*, **91**, 366–381.
134. BARR, M., & GLENNY, A. T. (1946): Some practical applications of immunological principles. *J. Hyg. (Land)*, **44**, 135–142.
135. SPINK, W. W. & PAINE, J. R. (1940): The bactericidal power of blood from patients and normal controls for staphylococci. *J. Immunol.*, **38**, 383–398.
136. PETTERSON, A. (1939): Über die immunisierende Wirkung der negativ chemotaktischen Substanz, mit besonderer Berücksichtigung der Immunität genen die pyogenen staphylokokken. *Z. Immun. Forsch*, **95**, 147–168.
137. ROUNTREE, P. M. & BARBOUR, R. G. H. (1952): Antibody to the erythrocyte coating polysoccharide of staphylococci: its occurrence in human sera. *Aust. An. Med.*, **1**, 80–83.
138. FINKELSTEIN, R. A. & SULKIN, S. E. (1957): Colonial agglutination of coagulase positive staphylococci in soft agar. *Bact. Proc.*, **57**, 97–98.
139. RAMON, G. (1939): La production des toxines diphtérique, tétanique, staphylococcique en vue de l' obtention des anatoxines correspondantes. Techniques. Résultats. *Rev. Immunol. (Paris)*, **5**, 385–404.
140. BIGGER, J. W. (1933): The production of staphylococcal haemolysin with observations on its mode of action. *J. Path. Bact.*, **36**, 87–114.
141. CRUICKSHANK, R. (1962): Mackie and McCartney's Handbook of Bacteriology (Tenth Edition). *E. & S. Livingstone Ltd.*, Edinburgh & London.
142. WALBUM, L. E. (1921): L' action de divers sels métalliques sur la production de Staphylolysine. *C.R. Soc. Biol.* (Paris), **85**, 376–377.
143. KNIGHT, B. C. J. G. (1937b): The nutrition of staphylococcus aureus; nicotinic acid and vitamin B1. *Biochem. J.*, **31**, 731–737.
144. GLADSTONE, G. P. (1938): The production of staphylococcal alpha-haemolysin in a chemically defined medium. *Brit. J. Exp. Path.*, **38**, 123–137.
145. GENGOU, O. (1935a): Acides aminés et toxine staphylococcique. *Ann. Inst. Pasteur*, **55**, 129–147.
146. RICHMOND, M. H. (1959): The differential effect of Arginine and Canaranine on growth and enzyme formation in *S. aureus*. *Biochem. J.*, **73**, 155.
147. McILWAIN, H. (1938): The effect of agar on the production of staphyloccocal alpha-haemolysin. *Brit. J. Exp. Path.*, **19**, 411–416.
148. WYATT, H. V., REED, G. W. & SMITH, A. H. (1962): Calcium requirements for growth of *Staphylococcus pyogenes*. *Nature*, **195** (4836), 100–101.
149. LONG, C., KING, E. J., & SPERRY, W. M. (1961): Biochemists' Handbook. *E. & F.N. Spoc. Ltd.*, London.
150. BOIVIN, A. & IZARD, Y. (1937): Méthode par la purification, á l'acide trichlara-cétigue, des toxines et anatoxines dipthérigues, tétanigues et staphylococciques. *C.R. Soc. Biol.* (*Paris*), **124**, 25–28.
151. TASMAN, A., BRANDWYK, A. C., MARSEILLE, A., VAN RAMSHORST, J. D. & VAN DER SLOT, J. (1952): On the purification and absorption of staphylococcus toxoid. *Antonie v. Leeuwenhoek*, **18**, 336–348.
152. TURPIN, A., RELYVELD, E. H., PILLET, J. & RAYMOND, M. (1954): Purification de la toxine et de l' anatoxine staphylococciques alpha. *Ann. Inst. Pasteur.*, **87**, 185–193.
153. WITTLER, R. G. & PILLEMER, L. (1948): The immunochemistry of toxins and toxoids — V. The solubility of staphylococcal toxin in methanol-water mixtures under controlled conditions of pH, ionic strength and temperature. *J. Biol. Chem.*, **174**, 23–29.

154. KUMAR, S., LOHEN, K., KENYON, A. J. & LINDORFER, R. K. (1962): The characterization of Staphylococcal Toxins. II. The isolation and characterization of a homogenous Staphylococcal Protein posessing alpha haemolytic, dermatonecrotic, lethal and leucocidal activities. *J. Exp. Med.* Vol. **115** (6), p. 1107.
155. KITCHING, J. S. & FARREL, L. N. (1937): The stability and antigenicity of staphylococci toxoid. *J. Immunol.*, **34**, 1-9.
156. WILSON, R. J. (1939): Detoxification of staphylococcal filtrates. *Canad. publ. Health, J.*, **30**, 42-43.
157. WILSON, R. J. (1940a): Some improvements in the preparation of staphylococcus toxoid. *Canad. Publ. Health, J.*, **31**, 23-25.
158. FLEMMING, L. W. (1960b): Studies on bovine staphylococcal Mastitis — III. Inactivation and antigenicity of toxins. *Cornell Vet.*, **52**, 350-362.
159. RICHOU, R. (1945): Sur le pouvoir imminsant de l' anatoxine staphylococcique précipitée par l' alun de potassium. *C.R. Soc. Biol.* (Paris), **139**, 387-388.
160. RICHOU, R. (1937): Les variations de l' immunité et de la production, d' antitoxine spécifique chez deux groupes de lapins taités par le mémes échantillans d' anatoxine staphylococcique. *C. R. Soc. Biol.* (Paris), **126**, 566-568.
161. RAMON, G. & RICHOU, R. (1937a): Sur la production de l' antitoxine staphylococcique chez le cheval. Influence de l' état d' immunité naturelle et du rapprochement des injections d' antigéne. *C. R. Soc. Biol.* (Paris), **124**, 315-317.
162. WEISS, C. (1939): A study of natural and acquired immunity to staphylococcal toxin in monkeys. *J. Immunol.*, **37**, 185-200.
163. RAMON, G. & NÉLIS, P. (1934): L'immunisation antistaphylococcique expérimentale au moyen de l' anastaphylotoxine. *C.R. Soc. Biol.* (Paris), **116**, 1250-1252.
164. PILLET, J., GIRARD, O., DUTHELL, H., & ORTA, B. (1959): 365: Recherches sur l' immunisation de la brekis contre la mammite staphylococcique. 591: Mammite staphylococcique de la brekis et vaccination locale. *Ann. Inst. Past.*, **96**, 365-591.
165. PLOMMET (1960): *Ann. Inst. Past.*, **99**, 618.
166. RICHOU, R., QUINCHAN, C. & RICHOU, R. (1961): Contribution a l' étude de l' immunité antistaphylococcique developpée, chez la vache, á la suite d' injections intramammaires d' antigénes specifiques. *Rev. Immunol.* (Paris), **25**, 299.
167. GREENBAUM, D. S., MILLER, L. & FLEMMINGTON, N. T. (1960): Antibody in milk and its role in passive immunization. *J. Lab. Clin. Med.*, **55**, 229.
168. KERR, W. R., PEARSON, J. K. L., & RANKIN, J. E. F. (1959): The bovine udder and its agglutinins. *Brit. Vet. J.*, **115**, 105-119.
169. DERBYSHIRE, J. B. (1960b): Experimental studies on the occurrence of staphylococcal antitoxin in the whey of cows. *Res. Vet. Sci.*, **1**, 350-354.
170. THÖRNE, M., & WALLMARK, G. (1961): Treatment of Bovine Staphylococcal Mastitis with Staphylococcal Toxoid. *Acta. Veterinaria Scandinavica.* **2** (4), 323.
171. PHILPOT, W. N. (1962): Herd vaccination as a means of Controlling Staphylococcal Mastitis. *Vet. Med.*, **57**, (8), 700-701.
172. MINNETT, F. C. (1937): Staphylococcus antitoxin in the blood of cows and other animals. *J. Comp. Path & Ther.*, **50**, 173.
173. MACCLAY, M. H., RANKIN, J. D., LOOSMORE, R. M. & SLAVIN, G. (1946): Experimental *S. aureus* Mastitis in Sheep. Treatment Trial with Sulphonamides or staphylococcus toxoid. *J. Comp. Path.*, **56**, 139.
174. RAMSEY, A. M., VAHRMAN, J. (1951): The treatment of generalized Staphylococcal infections. *Lancet*, **1**, 425-428.
175. JOVAR, R. M. (1944): The opsanization test in Brucellosis. *J. Immunol.*, **49**, 203.

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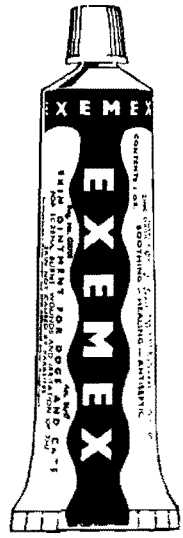

BPD116

A MODERN APPROACH TO THE CONTROL OF BOVINE TUBERCULOSIS

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SUMMARY

The effect of isoniazid on bovine tuberculosis was studied in 12 herds which at any one time contained approximately 3,000 head of cattle. At the start of the trial they contained 913 tuberculous animals. The period of treatment was 7 to 11 months and the period of observation 2 to 5 years.

The basic facts emerging from this trial were that approximately 75 per cent of cattle have been cured. This was shown by bacteriological examinations on 158 cattle killed for post mortem, and tuberculin tests on the animals retained on the farms.

Cured cattle could be recognised by the loss of tuberculin sensitivity after a suitable time lapse.

Isoniazid-resistant strains of *M. bovis* were shown to be highly attenuated.

After 7 to 10 months of treatment only 3 per cent of previously tuberculous cattle were found to contain bacilli of normal virulence.

Isoniazid was fully effective in preventing tuberculosis in non-infected cattle, but after heavy exposure tubercle bacilli may survive a period of dosing up to 40 days.

When all infected cattle in a herd were treated for a longer period the spread of the disease was halted completely.

INTRODUCTION

In a previous publication[1] it has been shown that the usual method of tuberculosis eradication by tuberculin testing, separation and slaughter of infected cattle has proved highly effective, but also very expensive. In view of the good results obtained using this method, and because highly organised campaigns were already in progress in many European countries before the advent of isoniazid, little attention was focused on this drug from a veterinary aspect. As the Bang method, combined with the slaughter out method was efficient and well proved, there was apparently a reluctance to carry out extensive trials on treatment of cattle. Most people thought that therapy would be very expensive and the danger of drug resistant organisms was overestimated.

A number of small trials on isoniazid therapy of tuberculous cattle were done by Italian investigators and different aspects were studied.[2–17] The work was, however, done by a number of independant institutes using different methods and small numbers of cattle. In the first years low dosages were used and the period of treatment was always short. In view of the experience with human patients and our later findings it is not surprising that the results of these trials were not encouraging. Seelemann et al[18] treated three bovines for 8½–10 months and achieved complete bacteriological cure.

Studies on chemoprophylaxis of bovine tuberculosis with isoniazid have been reported from Italy, Turkey, Germany and Czechoslovakia.[18–27]

Although the dosage varied from 4 mgr./kg. to 20 mgr./kg., and the methods of challenge were either by intravenous injection, per os or droplet infection, complete protection was always achieved.

In the medical world isoniazid by contrast has a most important place. It is the basis of all anti-tuberculosis therapy. In developing countries it is also used alone, for treating out-patients and prophylactically. Although nine other anti-tuberculosis drugs are now in use, none is as effective, relatively non-toxic and cheap as isoniazid.

SOUTH AFRICAN INVESTIGATIONS

In 1957 Dormer[28] emphasised the value of isoniazid in a paper given at the South African Veterinary Congress. In May 1957 his research unit started therapeutic and prophylactic treatment of a dairy herd of 120 head of which 74 per cent were reacting to tuberculin. The dosage varied from 5 to 20 mg/kg daily per os and the period of dosing was 27 months.[29] During that time the average skin reaction decreased from 7.5 mm to 3 mm. Of 32 cattle autopsied tuberculosis lesions could only be detected in a few and tubercle bacilli were grown from three cases. Group-specific sensitisation caused by so-called para-avian mycobacteria interfered with the trial.

During the last 5½ years approximately 3,000 head of cattle have been included in a large field experiment which started in January, 1958 and was organised by Dr. M. C. Lambrechts and one of us (H.H.K.) All bacteriological, pathological and other laboratory and clinical studies as well as collecting and analysing of epizootiological data was done at the tuberculosis section of Onderstepoort. The control of the farms was exercised by the local state veterinarians. Close co-operation was generally also obtained from the farmers.

In four previous articles [30–33] preliminary results, studies, on isoniazid administration, blood level in cattle, excretion and the drug's stability in cow's milk have been reported. The marked effect of treatment on the tuberculin reactivity was shown to be an important feature of chemotherapy.

Material and Method

The animals were on 12 different farms and of five different breeds. The infection rate varied from 11 per cent to 90 per cent in the different herds. Four farms which include about 1,000 animals have not been in the experiment long enough to include them in the epidemiological results. However, all data collected during the first two years of control indicate that the results from these farms will be similar to the results already available.

All herds were tuberculin tested at the start of treatment and again two months later. Animals were examined clinically and all reactors bled for haemagglutination and hemolysis tests. Thereby a considerable number of cattle with advanced forms of tuberculosis were recognised.

According to the percentage of reactors and the condition on the farm various regimes of treatment and prophylaxis were employed. In all herds suspicious reactors were in all respects treated like positive reactors. The dosage was 10 mg/Kg isoniazid daily in 10 herds and 15 mg/Kg daily in two herds, and was maintained for periods of 7–11 months. In four herds non-infected cattle received prophylactic treatment for the first two months, in two herds for six months, and in three herds for the full period. In three herds no prophylactic treatment was given. There was complete isolation during the first year in only three herds. On two farms the isolation was partial i.e. the same milking shed or dipping and sorting facilities were used but the herds kept separate. In the other seven herds no isolation was practised.

Stables and drinking troughs were disinfected with 3 per cent Formaldehyde at start of treatment, again two months later and in the heavily infected herds when treatment stopped.

Cattle were bought after varying periods of treatment and brought to Onderstepoort. Detailed post mortems were performed on 158 treated reactors. Tuberculous lesions were found in 140. The extent of the lesions found can be divided as follows:—

Generalised Tuberculosis.....	34 cattle
Extensive lung Tuberculosis.....	13 cattle
Localised Tb. in two organs.....	40 cattle
Localised Tb. in one organ.....	53 cattle

There is no reason to believe that the 18 cattle in which tuberculous lesions could not be found were not previously infected. Only cattle which had reacted positively at least twice were selected. Only 15 of the cattle were under five years of age and 24 over 10 years old. The selection of the cattle was biased due to a price limit, so that the owner generally selected only cattle in poorer condition and cattle which maintained relatively high tuberculin sensitivity. In all, tuberculous material from 316 organs and in no-visible-lesion cases a representative sample of lymphnodes was removed for bacteriological examination. Over 1,000 smears were examined, 1,600 egg media tubes (Lowenstein Jensen or Petragnani-Witte) and 1,900 tubes of fluid Kirchner-Hermann media were inoculated. Over 1,000 guinea pigs were injected with tuberculous material. All herds were regularly tuberculin tested at 3 to 6 monthly intervals and skin sensitivity and epizootology closely followed.

The full details of this work will be reported elsewhere. Only important basic findings will be considered in this paper.

Results

Symptomatology

In all cases the condition of the animals under treatment improved noticeably during the first three months. Their coats became sleeker and the animals gained weight. The amount of coughing heard in the stable at milking time was markedly reduced. Observations on seven cases with advanced lung tuberculosis showed disappearance of loud and rough respiratory sounds and rales. The coughing ceased and dimunition and consequent vanishing of muco-purulent discharge was seen. Healing of suppurating udder and lymph node abscesses was apparent in two to three months. The abscesses ceased to discharge, became covered with skin and then became palpably smaller. Seven cases of tuberculous mastitis were treated at Onderstepoort. There was a marked improvement clinically in all. The induration of the quarters became softer and after six to eight months the consistency was almost normal. In two cases, however, reactivation of tuberculous mastitis occurred after treatment was stopped. Active excretors had been removed before treatment started on farms.

Tuberculin Sensitivity

One of the major issues in this investigation, is the authors' contention that cured animals could be recognised by the tuberculin test, after a time lapse of 18–24 months after treatment was stopped. It must be realised that an animal which is bacteriologically cured, i.e. in which there are no living tubercle bacilli, need not necessarily immediately loose its skin sensitivity. An animal need not harbour any living organisms to be sensitive to tuberculin. Cattle can easily be sensitized by injecting dead organisms. The position is similar in a cured animal — although the drug and the host may have killed all the bacilli, a lesion may still contain dead organisms and their products. Until these are completely removed, and the stimulation for new antibody formation therefore lost, the body does not loose its sensitivity for tuberculin. It may take 18–24 months after treatment has ceased before sensitivity disappears (see table I). The period will naturally vary in different animals depending on the size and nature of the lesions and the number of bacilli.

The ability of isoniazid to temporarily reduce skin sensitivity has been over-estimated in a previous publication.[30] In the first herds treated this effect was quite noticeable, but in the more recently treated herds its effect has been virtually negligible.

The following evidence shows that loss of tuberculin sensitivity after treatment indicates a cure:—

(*a*) The close correlation between the percentage of animals found cured according to the bacteriological examination and those found negative to the tuberculin test. (Compare table I and II).

(*b*) It has repeatedly been shown at Onderstepoort that even very attenuated isoniazid resistant strains when injected into cattle and guinea pigs cause a marked sensitivity to tuberculin.

(*c*) Epizootiological studies have shown that there has been no spread of the disease from animals judged by the tuberculin test to be cured, when kept in contact with non-reactors for long periods.

(*d*) Bacteriological examinations done on 117 cattle autopsied soon after treatment, which showed doubtful or positive reactions at the time of slaughter, revealed that 73 of them were free of cultivable organisms. This indicates that an animal may be free of living bacilli a considerable time before it looses its skin sensitivity.

Two years after the end of therapy the herds were divided in cured and non-cured cattle according to the test and the farmer advised to remove the remaining reactors.

TABLE I

Tuberculin reactivity of tuberculous cattle treated for 8–10 months

	Time interval after treatment stopped								
	12–15 months			18–21 months			27–30 months		
Reaction Herds	pos.	doubt.	neg.	pos.	doubt.	neg.	pos.	doubt.	neg.
J.H.T.	0	1	8	0	0	7	0	0	4
G.H.	2	0	38	12	6	24	—	—	—
F.S./J.	19	18	18	—	—	—	—	—	—
P.O.G.	0	1	13	0	1	8	0	0	8
W.J.R.	1	7	36	0	6	35	1	0	38
P.H.W.	17	34	85	13	13	79	4	9	59
I.F.V.	7	6	61	3	14	36	1	2	39
Total	46	67	259	28	40	189	6	11	148
Percent negative			70 %			74 %			90 %

Key: pos. = positive reaction of 4 mm skinfold increase or more, regardless of character.
doubt. = doubtful reaction, 2–3.9 mm increase without other symptoms.
neg. = negative reaction or up to 2 mm.
Figures represent numbers of cattle reacting in each category.
Tests were done with bovine PPD-7000 TU per inj. and read with semi-antomatic calliper.
The decrease in cattle is mainly due to normal turn over.

Bacteriological Findings

The following table II summarises all bacteriological and biological test results obtained with the tuberculous material of 140 treated cases. The seven cases with chronic tuberculous mastitis are excluded since the retention of such cases on farms is not permissible and in practice such cases would be slaughtered.

TABLE II

Characteristics of tubercle bacilli isolated	Period of Treatment			
	2–3 months	4–6 months	7–8 months	9–10 months
Virulent Tb bacilli.....	5 (26%)	2 (10%)	2 (3%)	1 (2%)
Highly attenuated Tb bacilli.....	4	8	14	10
Non-cultivable, non-pathogenic Tb-bacilli only.....	3	7	18	10
Sterilised lesions only and cases without lesions (11).....	7	5	32	23
Total number of cattle examined...	19	22	66	44
Bacteriological cure.....	52%	55%	77%	75%

Of the 110 cases which received seven months treatment or longer, 76 per cent were bacteriologically cured compared with the figure of 52 per cent and 55 per cent for the shorter periods. The percentage of cases containing normal virulent bacilli was significantly higher in the short term groups than in the long term groups. There was no difference between the groups treated for 7–8 months and 9–10 months.

It is important to realize that isoniazid resistant organisms are of very reduced virulence as compared to their parent strains. This has been repeatedly shown with human strains. With isoniazid resistant bovine strains we were able to show that this attenuation was even more marked in most of the nine different species tested.[34] Calves fed on infected milk did not show tuberculous lesions a year later at post mortem and harboured no living organisms, although they became reactors for a time after infection. Different resistant strains injected intravenously into 22 calves failed to produce active progressive tuberculosis in any of them and negative animals kept in contact with them could not be infected. Whereas three quarters of control calves, injected with normal virulent strains, died within 30–50 days with severe tuberculous pneumonia, calves injected with resistant strains showed only isolated tubercles, or no visible lesions.

CHEMOPROPHYLAXIS

In seven herds 418 non-reactors were kept in close contact with 616 reactors. The non-reactors received 10 mgm/kg isoniazid daily for periods of two to nine months. Only seven of the non-reactors became tuberculin positive during the period of dosing.

As indicated previously,[30] tubercle bacilli did survive in lymph glands of calves when isoniazid dosing was discontinued at the same time as the source of infection was removed. A further experiment with 16 calves showed that a few viable bacilli could be found when the dosing continued for up to 40 days after exposure. No viable bacilli could be

found when the period was increased to 71 days. It was therefore made standard procedure in the later herds to give two months of prophylactic treatment to non-infected cattle.

Epizootiology during and after Treatment

The intradermal tuberculin test applied every 3–6 months to an infected herd is a very sensitive instrument to observe the spread of bovine tuberculosis and, unfortunately, other sensitising conditions as well. In our material we observed interference of the latter type in 3 herds. In an Ayrshire herd of 200 cattle of which 11 per cent were infected and treated, suddenly 10 more suspicious reactors came up. The retest after 3 months, however, showed a sharp decline of reactions to bovine PPD and a number of good reactions to avian PPD. Most probably a *M. avium*-like mycobacteria was the cause of this sensitivity. In a big Afrikander herd we encountered continuously through the years of control suspicious and positive reactions at the rate of 2.5 per cent in oxen, heifers and young cows which seldom lasted longer than 6 months. Hormonal influences are probably to blame. In the third herd sensitisation probably caused by human tubercle bacilli interfered in the interpretation of the test. By keeping exact records of all tests we were able to distinguish specific from non-specific reactions.

Apart from this, the situation was as follows: Between start of therapy and the retest 2 months afterwards 78 cattle or 5.5 per cent of non-reactors converted to positive. Fifty-three of these cases belonged to the two herds where prophylactic dosing of negative stock was not done. Many of these 78 animals were certainly not infected within the previous two months but before the initial test. In the following 18 months 18 cattle or 1.3 per cent of negative stock developed positive or suspicious reactions.

In the third, fourth and fifth year of control the herds contained 5 per cent to 25 per cent of treated tuberculous cattle which were kept in close contact with the non-infected portion of the herds. In this period no further new reactors were encountered in the 5 herds under long observation.

Discussion

It was found that isoniazid treatment can be used in eradicating tuberculosis from infected herds. All herds which were in this trial for for 3 years are virtually tuberculosis free.

The type of herd best suited for chemotherapy is the well managed dairy and stud herd. The results were similar in heavily and lightly infected herds. Proper identification of cattle and keeping of records proved essential for accurate interpretation of results.

Breed, age, condition, nutrition and other diseases did not noticeably alter the outcome. It was not necessary to isolate infected animals from the rest of the herd.

A daily dosage of 10 mg isoniazid per Kg. bodyweight for 8 months was found to be sufficient and economically feasible. At the present price it costs R7.00 to treat a 1,000 lb. cow for 8 months. Longer treatment periods or higher dosages did not prove superior. In farms where the drug was administered mixed with the daily rations the farming and milking routine was not unduly disturbed.

Six monthly tuberculin testing was found satisfactory for recognising cured cases and for following the epizootological pattern. Under the influence of isoniazid therapy the majority of the bacilli are eliminated within the first few months, and lesions become rapidly encapsulated. Consequently there was negligible spread of the disease after the first two months of treatment.

Reactivation has only been seen in two cases which were known to be suffering from generalized tuberculosis. Serological tests and clinical examination proved useful in recognising advanced cases, which could then be eliminated before treatment started.

Prophylactic treatment of non-tuberculous stock in contact with reactors can be used with advantage during the first 2 months of therapeutic control measures.

When this method of eradication is applied immediate slaughter or isolation of reactors is not necessary. Normal milk production and breeding can therefore continue undisturbed. Removal of non-cured cases can take place over longer periods.

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REFERENCES

1. KLEEBERG, H. H. (1963): *J.S. Afr. vet. med. Ass.* This issue.
2. AVELLINI, G. (1953): *Clinica Vet.*, **76**, 361.
3. AVELLINI, G., FIGORILLI, L. (1954): *Profilassi*, **9**, 831.
4. AVELLINI, G., FIGORILLI, L. (1955): *Ibid*, **10**, 143.
5. AVELLINI, G. (1957): *Vet. Ital.*, **8**, 769.
6. MESSIERI, A. (1955): *Atti. Soc. Ital. Sci. Vet. Palermo*, **9**, 536.
7. MORETTI, B., PEDINI, B. (1953): *Nuova Vet.*, **29**, 322.
8. MORETTI, B. (1952): *Nuova Vet.*, **11**, 12.
9. MORETTI, B. (1957): *Vet. Ital.*, **8**, 136.
10. MUCCI, G. (1953): *Zootech., Vet.*, **8**, 278.
11. NAI, D., CRESPI, A. (1958): *Atti. Soc. Ital. Sci. Vet.*, **12**, 640.
12. PEDINI, B. (1953): *Clin. Vet.*, 76, 353.
13. BOCCADORO, B., SANTILLE (1955): *Notiz. Instit. Vacc. Antitub.*, **5**, 11.
14. ZACCHI, B., DE FRANCISCIS, G. (1957): *Acta. Med. Vet. Napoli*, **3**, 465
15. VITALESTA, R. (1953): *Profillassi*, **20**, 103.
16. SANDRO, C. (1954): *Clin. Vet.*, **77**, 79.
17. LEGORI, E. (1953): *Clin. Vet.*, **76**, 315.

18. SELLEMANN, M., BUSCHKIEL, U., RACKOW, H. G. (1957–58): *Zbl. Vet. Med.*, **4**, 80.
ditto II: *Zbl. Vet. Med.*, **4**, 101.
ditto III: *Zbl. Vet. Med.*, **5**, 609.
19. BADIALI, L. (1957): *Vet. Ital.*, **5**, 7.
20. BADIALI, L. (1958): *Vet. Ital.*, **9**, 955.
21. BADIALI, L., BARCACCIA, E. (1954): *Vet. Ital.*, **10**, 292.
22. BADIALI, L., DE MAJO, F. (1962): *Riv. Tuberc. Mal. Appar. Resp.*, **10**, 1.
23. ROSATI, T., BADIALI, L. (1959): *Notiz. Inst. Vacc. Antituber*, **11**, 7.
24. ROSATI, T., BADIALI, L. (1960): *Arch. Vet. Ital.*, **11**, 451.
25. CATELLANI, G., D'ESPOSITO, G., ZACCHI, B. (1959): *Proc. XVI. Int. Vet. Cong. Madrid*, **2**, 699.
26. UCAR, N. (1960): *Turk. Vet. Hekimdern.*, **30**, 624.
27. KUBIN, M., ZAVADILOVA, Z., FRESLOVA, A. (1959): *Vet. Med. (Czech.)*, **7**, 577.
28. DORMER, B. A. (1957): *J.S. Afr. vet. med. Ass.*, **28**, 195.
29. DORMER, B. A., MARTINAGLIA, G., HOBBS, W. B. (1961): *S.A. Med. Jnl.*, **35**, 429.
30. KLEEBERG, H. H. (1959): *J.S. Afr. vet. med. Ass.*, **30**, 69.
31. KLEEBERG, H. H., GERICKE, J. J., WEYLAND, H. (1961): *J.S. Afr. vet. med. Ass.*, **32**, 77.
32. KLEEBERG, H. H., WEYLAND, H. (1961): *J.S. Afr. vet. med. Ass.*, **32**, 349.
33. KLEEBERG, H. H. (1961): *J.S. Afr. vet. med. Ass.*, **32**, 482.
34. KLEEBERG, H. H., WORTHINGTON, R. W. (1961): *To be published.*

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ERADICATION OF BOVINE TUBERCULOSIS

EFFORTS AND EXPERIENCES OF OVERSEAS COUNTRIES AND THE POSITION IN SOUTH AFRICA

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SUMMARY

Tuberculosis of man and cattle is disappearing fast in highly developed countries. In underdeveloped countries it is a major public health problem. Tuberculosis of cattle causes great economic losses for the nation and constitutes a continuous danger of infection of man. The veterinary profession of many countries have proved that bovine tuberculosis can be eliminated within 5 to 10 years provided the State is prepared to carry the bulk of the expenses. Both classical methods — the slaughter-out method and Bang's method of separation and gradual replacement — are immensely costly. The difficulties, dangers and costs of the proposed eradication scheme for South Africa are outlined. The urgency of control measures are emphasised.

INTRODUCTION

Eradication of bovine tuberculosis is a major objective of many countries. The disease has been virtually eradicated in the U.S.A., Canada, Great Britain, Ireland, the Scandinavians, Finland, Holland, West Germany, Belgium, Switzerland and Austria. It is a well known fact that in countries where bovine tuberculosis is prevalent, the incidence of infection of the human being with the bovine type can be high. There is a danger of transmission by droplet infection as well as from infected food products. The World Health Organization Expert Committee on Tuberculosis expressed the opinion that with the progress made in the control of malaria, tuberculosis is now regarded as the most important communicable disease in the world. However, lasting progress with measures against human tuberculosis, will not be possible without the decrease and final elimination of animal tuberculosis, especially bovine tuberculosis.

The annual expenditure on tuberculosis in humans in the Republic of South Africa amounted to 13 million in the year 1961/62, representing 44 per cent of the total health vote. It is essential that the efforts of the State to control human tuberculosis should now be extended to combat bovine tuberculosis in the country. According to the excellent review of the world literature by Goerttler and Weber[1] the prevalence of the

bovine type of tubercle bacilli as the causative organism of human tuberculosis varied between 4 per cent and 40 per cent depending on the site of the lesions. Out of 93,000 strains of tubercle bacilli isolated from humans up to 1952, 9,500 (10.2 per cent) proved to be *M. bovis*. That the rural population is naturally more exposed to infection with the bovine type, is reflected by an increase of up to 35 per cent of this infection in humans in some regions in Europe. In North America and in Europe the control and eradication of tuberculosis in cattle is accompanied by a marked reduction in the incidence of human tuberculosis caused by *M. bovis* or as is the case in the U.S.A. its virtual elimination.[2-4]

Treatment of milk by heat greatly reduces the risk of milk-borne infection but the only means of eliminating this danger completely is to eradicate bovine tuberculosis. Many investigators have emphasized the massive excretion of tubercle bacilli in the milk of cows suffering from tuberculosis. Schönberg[5] assumed that the milk of a cow with tuberculous mastitis may contaminate the milk of one hundred healthy cows after mixing in a dairy to such an extent that all the milk is contagious to man. According to Nassal[6] who examined the milk of 100,000 cows reacting positively to tuberculin, 1 per cent of reactors were excreting tubercle bacilli in the milk. This figure closely corresponds with that of Plum[7] who found 1 per cent tuberculosis of the udder at autopsy.

The percentage of open lung tuberculosis in reactors is reported to be 10–38 percent.[8-13] These animals not only cause air-borne infection but also contaminate the premises, food, manure, water and milk. A third of the Republic's milk production is not pasteurized.[14] Most of the industrial milk is undergoing high temperature short time pasteurisation. (H.T.S.T.) There is ample evidence to support the view that H.T.S.T.-pasteurisation is not always effective in killing all tubercle bacilli present in milk.[15–38] Thus it must be emphasized that the eradication of bovine tuberculosis will be the only means of protecting the human population from infection by bovine tubercle bacilli.

Costs of Eradication of Bovine Tuberculosis Overseas

The economic loss to the cattle industry due to tuberculosis can hardly be estimated. Exact statistical figures are only obtainable from abattoirs giving data on cattle and pigs which have been partly or completely condemned because of tuberculosis. The losses in meat, milk, offspring, general condition, lowered resistance and early slaughter caused by the disease cannot be appreciated by the layman but are assessed to be high by the expert.

Müssemeier[39] calculated the annual total loss due to bovine tuberculosis in Germany before the last war at 300–350 million German marks. Weyls[40] figure for Western Germany in the post war years was 275 million marks annually. Similar annual losses were reported by the Veterinary Services of France and were estimated at 200 million new francs. There were approximately 1.5 million reactors among the 18 million cattle in France. Veterinary authorities in Sweden have calculated an annual loss of 30 million Swedish crowns in 1930 at a time when ca

30 per cent of the cattle population of 3 million were infected with tuberculosis. Flückiger[41] stated that 50 million Swiss francs were lost annually in Switzerland due to bovine tuberculosis.

According to available literature the costs of eradication of tuberculosis in overseas countries were as follows: The U.S.A. started her campaign in 1917, carried out 378 million tuberculin tests on cattle in the past 40 years, and slaughtered 4 million reacting animals. The total expenditure amounted to $326 million. It is estimated that the savings resulting from the eradication now equal more than $150 million a year.[2]

The whole of Great Britain is now a TB-free accredited area. The costs spent by the nation have been assessed at £130 to £150 million sterling. The average expenditure during the years 1952 to 1958 was £10 million per year. The average incidence of tuberculosis in cattle was 17–18 per cent[42] that of cows 30–35 per cent. Compensations of up to ~100 per animal were paid for farmers. Up to 1954 £12 million had already been paid as premiums for TB-free herds.[43]

Ireland with a cattle population of 4.5 million had an incidence of 27 per cent reactors in cows and 7 per cent in beef cattle. In 1960 the expenditure on the scheme was £5 million a year and represented 50 per cent of the budget for the whole Department of Agriculture.[66]

Sweden has freed herself of bovine tuberculosis by spending 40 million Swedish crowns. In order to maintain this 750,000 crowns are needed annually.[43]

Denmark eliminated bovine tuberculosis in 1952 and her expenditure per year from 1953 onwards is only 800,000 Danish crowns.[44]

The Netherlands have brought the disease rapidly under control. In 1949 the infection rate was 17.4 per cent and in 1956 the retention of reactors was prohibited already by law. Four hundred thousand reactors out of 2.8 million head of cattle had to be destroyed.[66] The total costs till the country was declared free were 70 million gulden.

The expenses of the eradication scheme in Belgium with 2.2 million cattle amounted to 156 million Belgium francs in the year 1954.[44] Up to 1960 397,000 reactors were slaughtered and some compensation paid.[66]

The authorities in France calculate the annual costs for the present campaign at 55 million new francs. Of the 18 million cattle 8.5 per cent were infected in 1957. The annual credits paid by the state amount to 20 million new francs.[43]

Switzerland has also completed the eradication of bovine tuberculosis. From 1930 to 1959 400,000 reactors had to be slaughtered, which is a quarter of the cattle population. The total costs were 400 million Swiss francs of which the State contributed 100 million, the cantons 140 million and the farmers 150 million.[45]

The tuberculosis campaign in Austria started in 1950. Already in 1954 69 million Austrian shillings had been spent for this purpose of which 50 per cent were payments by the State and the rest was raised by the farmers. Thirty to fifty per cent of the 23 million head of cattle were found to be tuberculous at the onset of the control measures.[11]

In the Federal Republic of Germany rapid progress with the eradication of the disease has also been made.[46] It was recently declared free of tuberculosis. The campaign started in 1952 when two thirds of all farms

were infected and ended in 1962. The total expenditure amounted to 2,000 million German marks (R380 million) of which about 80 per cent were paid for tests and slaughter and 20 per cent for isolation of cattle and milk bonusses. Up to the end of 1959 the Federal government contributed over 1.3 milliard marks, an additional 320 million marks were paid by the individual Provinces and another 350 million marks came from agricultural organisations. One third of the total of 12 million cattle had to be removed at an early stage.[57, 66]

The target date for final eradication of bovine and avian tuberculosis in the Soviet Union is 1965.

In all these countries the control of bovine tuberculosis began as a public health measure but in its later stages it became more a means of improving animal health and productivity. Almost the whole of the veterinary profession of these countries was engaged in eradication programmes. Considering the great importance of bovine tuberculosis to the national economy and human health the immense costs and very great efforts have indeed been justified. In the countries mentioned there is also a sharp decline and gradual disappearance of tuberculosis in man due to modern chemotherapy, the changing social background and other factors. There is no doubt that the disease will soon be rare in these countries, both in man and in animals.

The Eradication in South Africa as an Economical Problem

The importance of tuberculosis in cattle to the national economy has long been recognized in South Africa.[47] Different aspects and problems concerning the eradication of the disease have been discussed by de Kock,[48] du Toit,[49] Lambrechts,[50] Snyman,[51] and Diesel.[14]

It is very difficult to make an exact estimate of the economic losses due to tuberculosis. The overall waste of national wealth can be divided into the following categories:

(1) *Losses due to condemnation of tuberculous cattle and pigs at abattoirs.* The present abattoirs statistics are not representative as the Insurance scheme of the Meat Industry Control Board excludes cattle condemned for tuberculosis from compensation. Thus very few dairy cows are sent for slaughter to the abattoirs but are killed by black marketeers or in uncontrolled areas.[14] In the case of pigs the low figures of condemned carcasses are probably due to overlooking of small lesions at meat inspection.[52]

(2) *The average age of cattle in a tuberculosis infected herd.* This is considerably lower than in tuberculosis free herds. They either die or leave the herd because of sterility, decrease in condition or poor milk production. Reimer[53] reported that the average age in herds free from tuberculosis increased to 9½ years. The British Committee on cattle diseases[54] stated that 10 per cent of cattle leaving dairy herds did so because of tuberculosis. MacGregor[55] estimated the total annual wastage in Britain at 22.5 per cent.

(3) *Utilisation of food.*—The wastage of feed by feeding it to animals that cannot yield an adequate return can hardly be calculated. The Joint Expert Group on tuberculosis of the WHO/FAO estimated that the overall productive efficiency of infected cows may be reduced by 10–25 per cent.

(4) *The milk production of tuberculous cows is reduced by* 10–12 per cent.[56, 53]

(5) *Fertility.*—The involvement of the genitals is reported by Fröhner, Zwick and Wirth[58] to be present in 5 per cent of tuberculin reactors. The same percentage of tuberculosis of the genitals was found by Hermansson[59] and Küst[60] while Wirth-Diernhöfer[61] give a percentage of 3 per cent uterine tuberculosis in Diernhöfer[61] give a percentage of 3 per cent uterine tuberculosis in reactors. Kielwin[62] observed uterine tuberculosis in 11 per cent of slaughter cattle. According to Reimer[53] the sterility of tuberculous cows increases by 5–10 per cent.

(6) *Losses in market value.*—There are considerable losses in the market value of tuberculous cattle due to poor condition, breeding value, decline in the growth rate of infected calves, reduction in beef quality and a general deterioration in resistance. It is common experience of veterinarians that the usual clinical diseases of cows are less frequent in Tb-free than in tuberculous herds. In Germany it is experienced that R40 more profit per cow per year is made in a Tb-free stud-herd compared with an infected stud-herd. In many parts of the world the losses caused by tuberculosis overshadowed those of any other disease. A conservative estimate of the annual loss to the South African cattle industry would be R4 million.

Apart from the danger of infecting humans, cattle may disseminate the disease to any other domesticated animal, especially to pigs, goats, horses, dogs, cats and to wild mammals, particularly to kudus.[63]

The Present Position and Reflections on a Possible Scheme for South Africa

Considering the above factors it is of the utmost importance to start an eradication campaign in South Africa as soon as possible. It has been said that the disease doubles every fifteen years if left uncontrolled. Up to the present the control which has been exercised over bovine tuberculosis in South Africa has been negligible.[14]

Any estimate of the incidence of the disease is difficult as abattoir figures are not a true reflection of the position. A similar state of affairs exists in regard to the results of the tuberculin tests as long as they are voluntary for the farmers.

The disease has probably reached a saturation point in the commercial dairies in the vicinity of certain large towns. The latest figure for the Durban area was 25 per cent to 30 per cent in a sample of 2,50 cattle[64] and for the Rand 38 per cent in 9 selected herds with 1,800 cattle.[65] The infection rate in the rural areas, however, is low, so that the departmental

committee estimated that the percentage for the whole cattle population might be approximately 3 per cent.

During 1962 40,000 cattle were tuberculin tested for the first time in the interim scheme and for diagnostic purposes, of which 4.7 per cent reacted positively and 2.4 per cent suspiciously.

A complete campaign is a matter of costs, replacement of infected animals, co-operation of all groups concerned, adequate legislation and sufficient professional, inspectorate and administrative staff. There are certain factors which may lead not so much to economical but to biological problems. The removal of many dairy cows at one time could lead to a shortage of milk in the country. A great shortage in heifers to replace the infected cows could also develop. Calf rearing will have to be undertaken on a much bigger scale and on many more farms than is done at present.

The question of total costs of an eradication scheme and how the funds are to be collected were the most difficult aspects of the investigations of the Departmental committee on the control of bovine tuberculosis in South Africa.[14] Any scheme based on the slaughter out method will have to meet the costs concerned with:

(1) *the application of the tuberculin test,*
(2) *the payment of compensation for animals destroyed,*
(3) *the possibility of payment of inducement bonusses,*
(4) *the marking and branding of all cattle,*
(5) *disinfection of premises, etc.,*
(6) *the improved hygiene,*
(7) *the segregation of reactors,*
(8) *the pasteurization of milk from infected herds,*
(9) *the distribution of information,*
(10) *the laboratory facilities and staff,*
(11) *the administration.*

The Committee calculated in 1956 that the costs for the testing of 1 million head of cattle would amount to ca R450,000. If all cattle in South Africa except those in the bantu areas were to be tested twice this would cost R7.2 million. Whatever the method of eradication will be, six tests will be necessary in a tuberculous herd before accreditation is obtained.

The Committee on bovine tuberculosis was greatly in favour of inducement bonusses to owners of accredited herds but has not been able to recommend the payment of such bonusses as it failed to appreciate from what source the necessary funds will be made available. The joint WHO/FAO Expert Group advises the payment of bonusses and experience in many overseas countries proves that this will make a considerable contribution to the success of the eradication campaign.

The costs for marking all cattle, disinfection of premises, troughs, etc., improvements on the hygiene conditions, separation of the reactors and facilities to keep them in retention may have to be paid by the farmers themselves. This may amount to considerable expense in some cases. In the countries in Europe that adopted the Bang-method of combatting tuberculosis, separate rearing of non-infected young stock was a basic principle of the eradication schemes.

In South Africa commercial dairymen have little or no opportunity of isolating reactors as their land is limited. Most persons interviewed by the Tuberculosis Committee considered the retention of reactors in isolation with separate facilities for stabling, feeding and attendants to be entirely impossible. The alternative to retention is immediate slaughter of all reactors. Such action could create a milk shortage, could embarrass stock owners and deprive breeders of valuable stud cattle.

Expenses arising from propaganda, administration, tuberculin production and laboratory examinations will have to be paid by the State.

The costs of the recommended slaughter-out plan to eradicate bovine tuberculosis will depend entirely on the amount of compensation the State is prepared to pay. Considering the above figures the gross expense of the proposed campaign without inducement bonusses might be R23.5 million which could closely agree with the estimate of the committee.

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REFERENCES

1. GOERTTLER, V., WEBER, E. (1954): Bovine Tuberkulose als Ursache humaner Tuberkulose. *Ferd. Enke verlag Stuttgart.*
2. STEELE, F. A., RANNEY, A. F. (1958): *Amer. Rev. Tuberc.*, **77,** 908.
3. CHRISTIANSEN, M. J. (1955): *Mh. Tierheilkd.*, **7,** 81.
4. MEYN, A. (1955): *Mh. Tierheilkd.*, **7,** 113.
5. SCHÖNBERG, F. (1951): *Lebensmitteltierarzt*, **2,** 81.
6. NASSAL, T. (1957): *Mh. Tierheilkd.*, **9,** 35.
7. PLUM, N. (1939): *Skand. Vet. Tidsk.*, **29,** 105.
8. DURBECK & KALLER (1924): *Berl. Tierärzt. Wschr.*, **46,** 641.
9. DORN (1925): *Thesis Leipzig.*
10. DEICH (1925): *Tierärzt. Rundsch.*, **40,** 821.
11. BERNGRUBER, R. (1927): *Tierärzt. Rundsch.*, **42,** 129.
12. WAGNER, K. (1936): *Tierärzt. Rundsch.*, 863 and 866.
ditto (1954): *Lebensmitteltierarzt.*, **5,** 113.
13. BELLER, K. (1937): *Arch. Tierheilkd.*, 237.
14. DIESEL, A. M. (1956): Report of the Departmental Committee on the Control of Bovine Tuberculosis in the Union of South Africa.
15. BROWNLIE, J. L. (1933): *Spec. Rep. Ser. Med. Res. Comm. Lond.*, 189.
16. DAVIS, J. E. (1953): *Dairy Index*, **18,** 210.
17. VERGE, J. (1954): Cited by Mazzeo. *Zbl. ges. Kind. Heilk.*, **49,** 198.
18. GROSSO, E. et al (1961): *L'igiene mod.*, **54,** 101.
19. GERNEZ-RIEUX Ch., GERVOIS, L. (1951): *Tuberk. arzt.*, 610.
20. GILBERT, R. M. (1951): *Rev. LeLait*, **31,** 585.
ditto (1945): *C. R. Soc. Biol.*, **139,** 356.
21. SARNOWIEC, W. (1936): *C. R. Soc. Biol.*, **123,** 370.
22. MOREAU, M. H., BRETEY, J., ROY, D. (1960): *Ann. Inst. Pasteur.*, **99,** 420 & 586.
23. ZELLER, H. (1928): *Zschr. Tuberk. Milchhyg.*, **38.**
24. LARMOLA (1943): *Zbl. Bakt.*, **151,** 148.
25. MUNZ, K. (1949): *Thesis Giessen.*
26. EIKMEIER, H. (1950): *Thesis Giessen.*
27. GREVE, H. (1950): *Lebensmittelteirarzt.*, **1,** 17.
28. HERZER (1950): *Lebensmitteltierarzt*, **1,** 152.

29. DOMAGK (1950): Chemotherapie der Tuberkulose mit den Thiosemikarbazonen. *George Thieme-Stuttgart.*
30. BLINDOW, H. (1951): *Thesis Giessen.*
31. SCHÖNBERG (1951): *Lebensmitteltierarzt,* **2,** 81.
ditto (1953): ibid., **4,** 133.
33. GRÜLL, F. (1951): *Thesis Giessen.*
34. WAGENER, K. (1952): *Zbl. Bakt.*, **158,** 293.
ditto (1954): *Lebensmitteltierarzt*, **5,** 113.
35. BINGEL (1953): *Zbl. Bakt.*, **159,** 427.
36. ALBUS, K. (1954): *Thesis Giessen.*
37. MAAS & BLUM (1954): *Mh. Vet. Med.*, **9,** 104.
38. KERTAY, N. (1961): cited in *Zbl. Bakt.*, **184,** 556.
39. MÜSSEMEIER, F. (1958): Rinder-tuberkulose, Goerttler & Kruger. *S. Hirzel Verlag Leipzig.*
40. WEYL, A. (1950): Neue Wege zur Bekämpfung der Tuberkulose des Rindes. *Verlag Schaper Hannover.*
41. FLÜCKIGER, C. (1955): *Mh. Tierheilk.*, **7,** 61.
42. FRANCIS, J. (1958): Tuberculosis in animals and man. *Cassel, London.*
43. LAMBRECHTS, M. C. (1959): Report on control of bovine tuberculosis in Europe. *South African Dept. Agricult.*
44. HÜBNER, K. (1950): *Mh. Tierheilk.*, **5,** 117.
45. HÜGLY, E. (1960): *Schw. Arch. Tierh.*, **102,** 547.
46. MEYN, A. (1956) *Mh. Tierheilk.*, **8,** 69 and 241.
ditto (1957): *Mh. Tierheilk.*, **9,** 185.
ditto (1962): *Mh. Tierheilk.*, **14,** 17.
47. VILJOEN, P. R. (1927): *J.S. Afr. vet. med. Ass.*, **1,** 20.
48. DE KOCK, G. (1932): *J.S. Afr. vet. med. Ass.*, **3,** 53 & 57.
49. DU TOIT, P. J. (1936): *Quart. Bul. Health. Org.*, **5,** 100.
50. LAMBRECHTS, M. C. (1955): *J.S. Afr. vet. med. Ass.*, **20,** 229.
51. SNYMAN, P. S. (1955): *J.S. Afr. vet. med. Ass.*, **20,** 241.
52. VAN RENSBURG, S. J., DU CASSE, F. B. W. (1961): *J.S. Afr. vet. med. Ass.*, **31,** 405.
53. REIMER, H. (1957): *Mh. Vet. Med.*, 669.
54. BRITISH COMMITTEE ON CATTLE DISEASES. *Report* 1934, *p.* 9.
55. MACGREGOR, J. L. (1941): *Vet. Jnl.*, **97,** 340.
56. MELVIN, A. D. (1908): *25th Report, U.S. Depart. Agric. Bur. Animal Ind.*, p. **97.**
57. WINNIGSTEDT, R. (1960): *Ber. Landwirtsch.*, **1,** 78.
58. FRÖHNER-ZWICK-WIRTH (1942): Kompendium der speciellen Pathologie und Therapie für Tierärzte. *Ferd. Enke Verlag Stuttgart.*
59. HERMANSSON, K. A. (1928): *Thesis Berlin.*
60. KÜST (1953): Giessen, personal communication.
61. WIRTH-DIERNHOFER (1950): Lehrbuch der inneren Krankheiten der Haustiere. *Verlag Ferd. Enke, Stuttgart.*
62. KIELWEIN, J. (1956): *Thesis Stuttgart.*
63. ROBINSON, E. M. (1944): *Onderstepoort Jl. Vet. Res.*, **19,** 23.
64. HOBBS, W. B. (1960): Personal communication.
65. KLEEBERG, H. H. (1961): Unpublished work.
66. —— Second symposium on the eradication of bovine tuberculosis, *Rome*, 1960.

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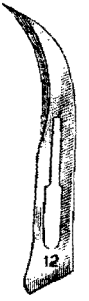

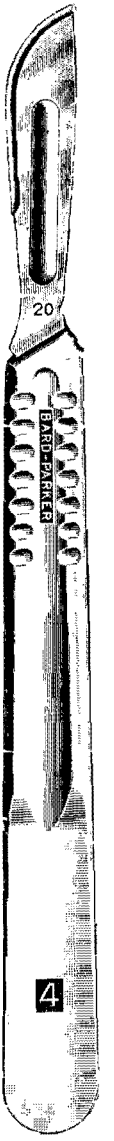

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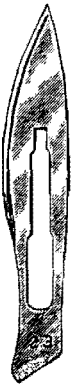

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PRETORIA.

IMMOBILIZING DRUGS USED IN THE CAPTURE OF WILD ANIMALS IN THE KRUGER NATIONAL PARK

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SUMMARY

A discussion on the immobilizing drugs and tranquillizers used in the capture of wild animals in the Kruger and Addo Elephant National Parks is presented. Field trials were conducted with the following drugs: Nicotine salicylate, Scoline (Allan-Hanbury), Flaxedil (May Baker), Sernylan (Parke Davis), Themalon (Burroughs Wellcome), Morphine hydrochloride, Omnopon (Roche), Quiloflex (C. H. Boehringer Sohn), Largactil (May Baker), Trilafon (Scherag), Vetame (Squibbs), Pethidine hydrochloride, Phenergan (May Baker), Hyoscine hydrobromide and an experimental drug Ro5/2807/B–5F (Roche). Dosages for the various drugs successfully applied to different game species are tabulated.

The immobilization of game by means of a projectile syringe containing paralysing or hypnotic drugs, has become a valuable tool in the management and control of wild animals. It enables the scientist to study the migration habits and other peculiarities of game animals, facilitates the translocation of wild animals to areas where they have become extinct, and may be employed as an effective and safe method of elephant control in National Parks. The ultimate perfection of this technique is of importance to both veterinarians and biologists.

Since the publication by Hall *et al.*[10] on the use of paralytic agents in in the capture of White-tailed deer, several workers have been engaged in research on the immobilization of wild game animals. Various drugs were employed on different species of game in an attempt to develop a safe and practical method of capturing wild animals. These drugs varied in their action on the animal from muscle relaxants to tranquillizers, hypnotics, and narcotics. A suitable and reliable drug for all game species has not been found yet — probably as a result of divergent psychosomatic and physiological features in the different species.

Empirically an immobilizing agent for any given species should conform to the following requirements:—

(1) The drug should have a very wide safety margin. This is of great significance where one is confronted with free roaming wild animals, and the correct judging of the animal's bodyweight is difficult.

(2) The latent period prior to the drug taking effect should be as short as possible. A time lapse of 10–20 minutes after the animal has been darted, is often sufficient for the animal to escape in heavily overgrown country.

(3) A reliable and fast-acting antidote should be at hand in cases of severe overdosage or retarded recovery.
(4) In dealing with wild animals, tranquillizing or sedative drugs are essential and it would be of great advantage if a drug incorporated both muscle relaxant and sedative properties.
(5) The drug should be non-irritating to the musculature as intramuscular injection is the only means of parenteral administration with the dart-syringe.
(6) The volume of the drug required for effective immobilization should be as small as possible. Under local conditions in the Kruger National Park, a total volume of 1–5 cc. would be optimal and 10–15 cc. maximal.

During the past two years trials were conducted with various drugs as immobilizing agents on a number of wild animal species in the Kruger and Addo Elephant National Parks. It is our intention in this paper to present a brief résumé of the results achieved, with a discussion of the merits and disadvantages of each drug *per se*. Dosages of the respective drugs, for some African wild animals, are presented in table I.

The method of drug administration was essentially the same as described by us in a previous publication.[20]

To date the following drugs have been subjected to field trials:—

1. *Nicotine salicylate*

Grzimek[9] reports: "The effect of nicotine salicylate is the opposite of that of curare, that is, it stimulates the activity of the nerves. The motor nervous system is strongly stimulated and a total disorganization of the system results. The effect of nicotine appears in two phases: at first there is a temporary strong stimulation of the whole nervous system; this is followed by a general paralysis of the ganglions."

In the Kruger National Park this drug was administered to Impala (*Aepyceros melampus melampus* Lichtenstein), but on account of its danger to the operator and its severe convulsive action on the patient, it was soon discarded and is in our opinion not suitable for this type of work.

2. *Syccinyl choline chloride* ("Scoline" — Allan-Hanbury)

Several workers have used Succinyl choline chloride as an immobilizing agent on a number of animal species. It is a muscle relaxant acting by persistent depolarisation of the motor end plates. There is no specific antidote available for this drug and the safety margin in some animals, viz. Wildebeest (*Gorgon taurinus taurinus* Burchell), Buffalo (*Syncerus caffer caffer* Sparrman) and African elephant (*Loxodonta africana* Blumenbach) is so critical that the drug's application under field conditions becomes unpractical.

In other species, *viz.* Impala, Zebra, and Uganda Kob[1], where the safety margin is not so critical, succinyl choline chloride can be classed as an effective and relatively safe immobilizing agent.

Its advantages are its relatively short reaction time, i.e. the latent period prior to the drug taking effect, which varies from 6–12 minutes in most animals. The effect of the drug usually lasts for a period of 20–60 minutes and the recovery is rapid and complete. The drug is also virtually non-poisonous when taken per os. In a few instances, where dangerous

and injured elephants had to be destroyed in the Kruger National Park, the drug was effectively and safely adapted as an euthanasiac. Unfortunately Scoline is affected by heat and its potency deteriorates under field conditions, if not kept cool.

3. *Gallamine triethiodide* (Flaxedil — May Baker)

Gallamine triethiodide is a synthetic, curare-like substance, producing paralysis by blocking nervous impulses at the neuro-muscular junctions.

Flaxedil was one of the first drugs used in the immobilization of game. Hall *et al.* reported on the use of Flaxedil to produce paralysis in White-tailed deer in 1953, and it is still to this day one of the most effective drugs for the capture of certain animal species.[10] The fact that Flaxedil and other synthethic curarates have a reliable antidote, renders them preferable to many other preparations.[19] The action of Flaxedil is to a large extent rapidly reversible by Neostigmin and other anti-choline esterase producing agents. In some animals a recurrence of the paralysis after antidote administration, necessitates a supplementary dose of antidote.

Initial experiments with Flaxedil in the Kruger National Park were conducted on the Impala, Wildebeest, Buffalo and Giraffe (*Giraffa camelopardalis* Linnaeus). Eminently satisfactory results were obtained in combination with Atropin (5 mgm/100 lb.)

The time lag between darting and the drug taking effect varied from 9–26 minutes. In the majority of cases the animals recovered within 1½–2 minutes after intra-venous antidote administration.[21] Impala immobilized with Flaxedil, without receiving the antidote, remained paralyzed for a period of 45–72 minutes.

The drug is highly soluble in water and stable under field conditions. A relatively narrow safety margin in some species and rather long reaction time are the two main disadvantages of the drug. The addition of the enzyme hyaluronidase is recommended to induce a shorter latent period prior to the drug taking effect.

As with all paralyzing drugs, regurgitation of the stomach contents may occur in ruminants if they are unable to remain on their briskets.

4. 1-(-1- *Phenyl cyclohexyl*) *piperidine hydrochloride* (*Sernylan — Parke Davis)

According to Graham Chen and co-workers, Sernylan is a drug that acts on the central nervous system[3] either by stimulation or by depression.

In a previous publication[21] we reported on the results obtained with this drug on the following species viz. Giraffe, Impala, Hippopotamus (*Hippopotamus amphibius* Linnaeus), Buffalo and Baboon (*Papio* (*Chaeropithecus*) *ursinus orientalis* Goldblatt). The following characteristics of the drug were mentioned in the report:

"(1) At the dosage rates applied locally Sernylan exhibits little or no tranquillizing effect, but appears to act rather as a depressant of the balance centre in the brain. Some of the animals were only slightly tranquillized . . .". In the Baboon, however, the effect of the drug (combined with Largactil) was that of surgical anaesthesia.

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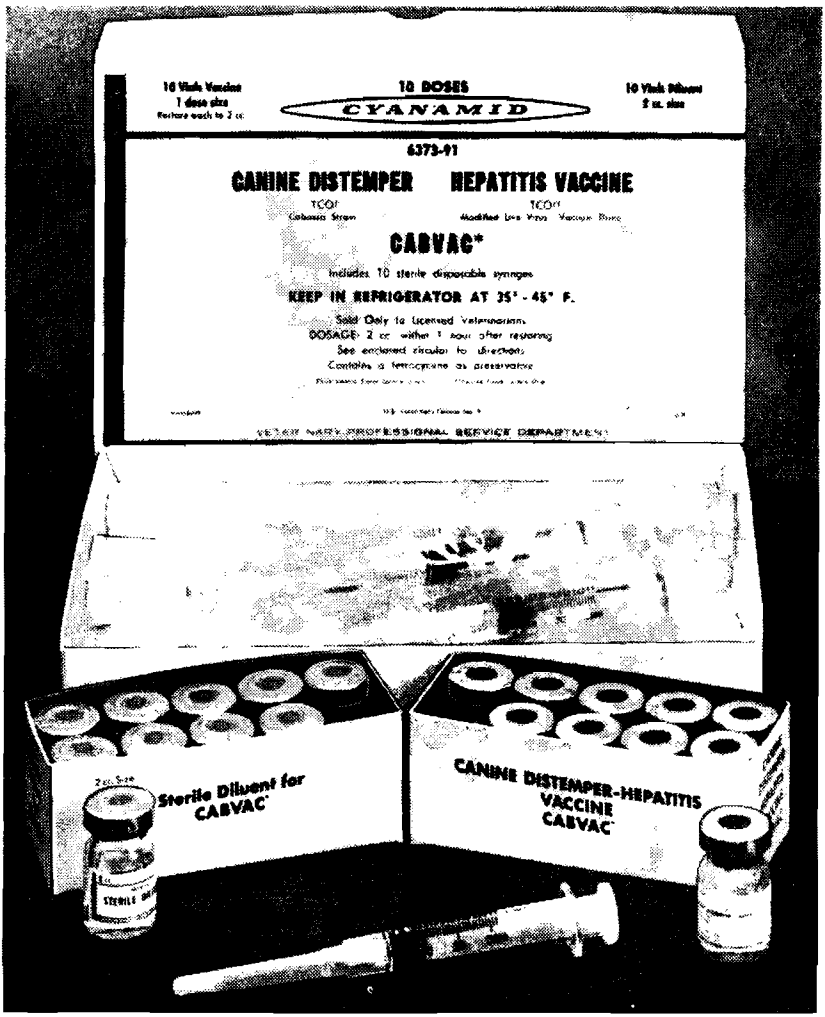

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"(2) The time lag between darting and the drug taking effect was surprisingly short." The animals became ataxic at 7 minutes and went down at 20 minutes.

"(3) There is no detectable depression of respiration, except in cases of severe overdosage. In this respect the drug is superior to muscle relaxants like Succinyl choline chloride and curare-like drugs.

(4) A specific antidote for Sernylan is not available . . .

(5) The most promising results were obtained where Sernylan was combined with a suitable tranquillizer . . .".

The recovery stage is gradual, but characterized by intermittent excitory periods. The duration of the effect of the drug varied from 30 minutes (in Impala) to 13 hours (Baboon). The optimum dosage in the Giraffe caused immobilization for $1\frac{1}{2}$ hours.

5. *Diethyl thiambutene* (Themalon — Burroughs Wellcome)

Harthoorn[13] evolved an immobilizing mixture for the capture of the White (Square lipped) Rhinoceros (*Ceratotherium simum simum* Burchell).

The mixture consists of Themalon or Morphine hydrochloride (a narcotic) as the principal immobilizing agent, with Hyoscine hydrobromide (an amnesiac) and Largactil (or Sernylan) as adjuvants.

The significant advantage of this mixture is its wide margin of safety. The principal drug is also readily antagonized by Nalorphine hydrobromide (Lethidrone).

With this mixture we succeeded in capturing five Hippopotami.[20, 21]

Omnopon (Roche) — (a drug incorporating all the alkaloids of opium) has been used as a substitute for Themalon or Morphine. Themalon, however, remains the drug of choice.

6. 2- (*Gamma-methoxypropyl-aminomethyl*)-1, 4-*benzodioxane hydrochloride* (Quiloflex — C. H. Boehringer Sohn)

Quiloflex is a reflex inhibitor used in human medicine for the symptomatic treatment of spasticity due to pyramidal tract lesions.

Initial experiments with Quiloflex in the Kruger National Park were conducted on the Impala. As reported in a previous publication[22] gratifying results were obtained in the case of this species.

In subsequent trials on eight different species, variable reactions were encountered. The small number of animals utilized in these preliminary experiments is far too inadequate to express a definite opinion on the value and versatility of Quiloflex as immobilizing agent in game. Nevertheless, in analyzing the available data, the following salient characteristics may be mentioned.

Quiloflex has a very wide safety margin in the Impala. A dosage range of 2–25 mgm/lb. was effectively applied in this species.

Animals darted with Quiloflex go down on their briskets and remain in that position for a considerable length of time. Should the animal escape after being darted, sufficient time is available to search for it before any assistance is required by the immobilized animal.

In the majority of cases the onset of reaction is fast. Ataxia is usually evident in 2–6 minutes after the animal has been darted. The higher the dosage rate applied, the sooner the animal becomes sufficiently immobilized to be handled. Animals that are only partially immobilized

by this drug resent handling and are prone to serious injuries and complications.

The reaction on the animal varies from ataxia to deep narcosis, depending on the dosage rate applied. The effect lasts from $1\frac{1}{2}$ hours (Baboon) to 6 hours (ruminants). The recovery stage is gradual and the animal is well tranquillized during this period.

The tissue tolerance of Quiloflex in a concentrated solution seems to be good, and the drug is stable under local conditions.

No specific antidote is available yet. Unfavourable side-effects such as bloat and dyspnoea have been observed.

Tranquillizers

Sedatives or tranquillizers are valuable and in some cases indispensable drugs in game immobilization. These drugs are either utilized as essential ingredients of immobilizing mixtures, or play a major rôle in the after care of captured wild animals.

The ideal tranquillizer should sedate the animal to such an extent, that it is divested of all aggressiveness and fear, but retains its consciousness and control over normal body-mechanisms. Significant characteristics are the volume of the dose and the time taken by the drug to produce the requisite state of calmness.

The following tranquillizers were used in game in the Kruger National Park.

1. *Chlorpromazine hydrochloride* (Largactil — May Baker)

This drug was successfully combined with Themalon and Hyoscine hydrobromide in an immobilizing mixture in the capture of White Rhinoceri[12, 13] and Hippopotami.[20, 21]

In combination with Sernylan in the immobilization of Giraffe, Hippopotami and Baboon, the drug was found to be most effective in ôcounter-acting the excitory stage encountered during the recovery period.

The large volume required by big game animals is a limiting factor when the drug is to be administered in combination with other drugs by means of a projectile syringe.

Satisfactory results were obtained with Largactil in ruminants. In the White Rhinoceros a dosage rate of 0.5 mgm/lb. was ineffective to produce tranquillization during transportation.

2. *Perphenazine* (Trilafon — Scherag)

The small volume required by large animals renders this drug suitable as an adjuvant to the principal immobilizing drug.

In the Kruger National Park this drug was combined with Sernylan for the capture of Giraffe and Hippopotami. The onset of reaction (when administered intra-muscularly) is slow.

3. *Trifluprom azine hydrochloride* (Vetame — Squibbs)

Although only a limited number of animals were treated with this drug, excellent results were obtained in the case of Giraffe, Buffalo and Eland.

The drug is available in a high concentration (20 mgm per cc.) and the recommended dosage rate is low (5 mgm/100 lb). It has a rapid onset of reaction.

The drug was used for premedication in buffalo and as an adjuvant to Quiloflex in the immobilization of an eland. Satisfactory results were obtained with Vetame in giraffe during transport.

4. On account of peculiar reactions provoked by some tranquillizers in the horse, the following mixture was administered to zebra: *Pethidine hydrochloride*, *Chlorpromazine hydrochloride*, *Promethazine hydrochloride* and *Hyoscine hydrobromide*.

This cocktail, administered intramuscularly or intravenously, produced effective tranquillization.

5. An experimental drug, Ro5-2807/B-5F (Roche)

This was used as the tranquillizing agent for the translocation of captured oribi (*Ourebia ourebi* Zimmerman), Steenbuck (*Raphicerus campestris zuluensis* Roberts), Grey rhebuck (*Pelea capreolus* Bechstein) and Hippopotami. The majority of captured animals responded very well to this drug.

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REFERENCES

1. BUECHNER, H. K., HARTHOORN, A. M. & LOCK, J. A., (1960): Immobilizing Uganda Kob with Succinyl choline chloride. *Canadian Jnl. of Comp. Med.*, **24**, 11.
2. BUECHNER, H. K., HARTHOORN, A. M. & LOCK, J. A., (1960): The immobilization of African animals in the field with special reference to their transfer to other areas. *Proc. Zool. Soc.*, London, **135**, Part 2, 261.
3. BUECHNER, H. K., HARTHOORN, A. M. & LOCK, J. A., (1960): Recent advances in field immobilization of large mammals with drugs. *Trans. 25th North American Wild Life Conference.*
4. BUECHNER, H. K., HARTHOORN, A. M. & LOCK, J. A., (1960): The immobilization of wild animals as an aid to management and control. *Oryx. V*, No. 6.
5. BUECHNER, H. K., HARTHOORN, A. M. & LOCK, J. A., (1960): Control of African Wild animals. *Nature.*, **185**, 47.
6. CARTER, N., (1961): Progress in drugging technique. *Wild Life*, **2**, 9.
7. COWAN, McT. I., WOOD, A. J. & NORDAN, H. C., (1962): Studies in the tranquillization and immobilization of deer (Odocoileus). *Canadian Jnl. of Comp. Med. and Vet. Sci.*, **26**, 57-61.
8. CRAIGHEAD, JOHN J. et al., (1960): Trapping, immobilization and color-marking of Grizzly bears. *Trans. of the 25th North Amer. Wild Life and Natural Resources Conf.*, March, 1960, p. 347-63.
9. GRZIMEK, M. and B., (1960): A study of the game of the Serengeti plains. *Zeitschrift für Saugetierkunde.*, **25**.

DOSAGES OF DRUGS USED IN SUCCESSFUL IMMOBILIZATION AND TRANQUILLIZATION OF DIFFERENT GAME SPECIES (MGM/LB)

TABLE 1

Species	Scoline	Flaxedil	Sernylan	Themalon	Morphine	Omnopon	Quiloflex	Largactil	Trilafon	Vetame	Ro5-2807/B-5F	Hyoscine	Pethidene	Phenergan	Prostigmin	Atropin	Mixtures
Impala...	0.16	0.9–1.2	0.215	—	—	—	2.0–25.0	0.5–1.0	—	—	—	—	—	—	0.01	0.05	1. Flaxedil & Atropin 2. Sernylan & Largactil
Wildebeest*	0.035	0.9–1.0	0.18	—	—	—	—	0.5–1.0	—	—	—	—	—	—	0.01	0.05	1. Sernylan & Largactil 2. Flaxedil & Atropin
Buffalo...	—	0.9–1.0	0.15	—	—	—	3.0–5.0	—	0.06	0.05	—	—	—	—	0.01	0.05	1. Quiloflex & Vetame 2. Flaxedil & Atropin
Giraffe*..	—	1.0–1.2	0.15–0.18	—	—	1.0	2.5–3.0	0.5	0.06	0.05	—	0.05	—	—	0.01	0.05	1. Flaxedil & Atropin 2. Sernylan & Largactil or Trilafon 3. Omnopon, Largactil & Hyoscine
Eland*...	—	—	—	—	—	—	2.5	—	—	0.03	—	—	—	—	—	—	Quiloflex & Vetame
Blesbuck*	—	—	—	—	—	—	17.0	—	—	—	—	—	—	—	—	—	
Zebra....	0.1	—	—	—	—	—	—	0.2	—	—	—	0.05	0.4	0.2	—	—	Largactil, Pethidine, Phenergan & Hyoscine combined
Zebra....	0.1	—	—	—	—	—	—	0.2	—	—	—	0.05	0.4	0.2	—	—	Largactil, Pethidine, Phenergan & Hyoscine combined
Springbok*	—	—	—	—	—	—	15.0	—	—	—	—	—	—	—	—	—	
Baboon..	—	—	2.5	—	—	—	12.5	1.0	—	—	—	—	—	—	—	—	Sernylan & Largactil
Hippopotamus	0.05	—	0.125–0.16	—	0.5	0.5–1.0	—	0.25–0.4	0.06	—	—	0.05	—	—	—	—	1. Sernylan & Largactil or Trilafon 2. Morphine, Largactil & Hyoscine
White Rhinoceros*	—	—	—	1.5	—	—	—	0.25	—	—	—	0.05	—	—	—	—	Themalon, Hyoscine & Largactil
Oribi.....	—	—	—	—	—	—	—	—	—	—	0.6–0.8	—	—	—	—	—	
Steenbuck	—	—	—	—	—	—	—	—	—	—	1.1–1.4	—	—	—	—	—	
Grey rhebuck	—	—	—	—	—	—	—	—	—	—	1.1–1.4	—	—	—	—	—	

*Only one animal has been treated by the authors with the following drugs:
(1) Wildebeest — Sernylan & Largactil.
(2) Giraffe — Omnopon, Hyoscine & Largactil.
(3) Eland — Quiloflex.
(4) Blesbuck — Quiloflex.
(5) Springbok — Quiloflex.
(6) White Rhinoceros — Themalon, Hyoscine & Largactil.

10. HALL, T. C., TAFT, E. B., BAKER, W. H. & AUB, J. C., (1953): Use of Flaxedil to produce paralysis in deer. *Jnl. Wild Life Mngmt.*, **17.**
11. HARTHOORN, A. M., (1962): The use of a Neuro-muscular blocking agent on domestic cattle. *Vet. Rec.*, **74,** 13.
12. HARTHOORN, A. M., (1962): The capture and relocation of the White (Square lipped) Rhinoceros, *Ceratotherium simum simum. The Lammergeyer*, **2,** 2.
13. HARTHOORN, A. M., (1962): Capture of the White (Square lipped) Rhinoceros, *Ceratotherium simum simum* (Burchell) with the use of drug immobilization technique. *Can. Jnl. of Comp. Med. and Vet. Sci.*, **26,** 9.
14. HEUSCHELE, WERNER P., (1961): Immobilization of captive wild animals. *Vet. Med.*, **56** (8).
15. PIENAAR, U. DE V. & VAN NIEKERK, J. W., (1963a): The capture and translocation of three species of wild ungulates in the Eastern Transvaal with special reference to Ro5–2807/B–5F (Roche) as a tranquillizer in game animals. *Koedoe*, **6,** 83–90.
16. PIENAAR, U. DE V. & VAN NIEKERK, J. W., (1963b): Elephant control in National Parks. A new approach. *Oryx*, **VII,** No. 1, 35–38.
17. TALBOT, L. A. & LAMPREY, H. F., (1961): Immobilization of free-ranging East African Ungulates with Succinyl choline chloride. *Jnl. Wild Life Mngmt.*, **25,** No. 3.
18. TALBOT, L. M., (1961): Field immobilization of some East African wild animals and cattle. *East. Afr. Agr. & For. Jnl.*, **26,** No. 2, 92–102.
19. TALBOT, L. M. & TALBOT, M. H., (1962): Flaxedil and other drugs in field immobilization and translocation of large mammals in East Africa. *J. Mammal.*, **43,** No. **1,** 76–88.
20. VAN NIEKERK, J. W. & PIENAAR, U. DE V., (1962): Adaptation of the immobilizing technique to the capture, marking and translocation of game animals in the Kruger National Park. *Koedoe*, **5,** 137–143.
21. VAN NIEKERK, J. W. & PIENAAR, U. DE V., (1963a): A report on some immobilizing drugs used in the capture of wild animals in the Kruger National Park. *Koedoe*, **6,** 126–133.
22. VAN NIEKERK, J. W., PIENAAR, U. DE V. and FAIRALL, N., (1963b): A preliminary note on the use of Quiloflex (Benzodioxane hydrochloride) in the immobilization of game. *Koedoe*, **6,** 109–114.

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Die vang van wild deur middel van chemiese middels is 'n nuwe ontwikkeling in die wetenskap wat gedurende die afgelope dekade na vore getree het. Voorheen is wild op die sogenaamde „boeremanier" gevang, maar die hoë sterftesyfer wat ondervind is, het aanleiding gegee tot die ontwikkeling van nuwe tegnieke. Hierdie tegnieke is nog maar in die begin-stadium en is by verre na nog nie volmaak nie. Heelwat navorsing sal nog gedoen moet word alvorens dit met absolute veiligheid vir beide mens en dier kan aangewend word. Gevolglik bly die versorging en verpleging van gevangde wilde diere van die uiterste belang.

Vir die veearts is daar eintlik niks vreemds omtrent die versorging van wild tydens die vangproses nie. Basies berus dit op die gewone beginsels van diereversorging. Aangesien daar egter kollegas is wat nie te dikwels met wild te doen kry nie, maak ons van hierdie geleentheid gebruik om van ons ondervindinge aan u mee te deel. Die doel is dus om u gerus te stel en op u gemak te laat voel wanneer u ingeroep word om behulpsaam te wees by die vang en vervoer van wild.

In die Krugerwildtuin word daar van die volgende metodes gebruik gemaak om wild te vang.

Met die sogenaamde „immobilisasie van wild" word daar van die verdowingspyl-geweer gebruik gemaak wat 'n outomatiese pylspuit in die dier skiet om die verdowings- of verlammingsmiddel toe te dien. Die apparaat wat algemeen gebruik word, is die Amerikaanse Cap-Chur geweer of pistool en die kruisboog.

In die Krugerwildtuin word laasgenoemde gebruik vanweë die groter trefafstand en akkuraatheid. Die trefafstand van die kruisboog wissel van 30–70 treë en dié van die Cap-Chur geweer 30–40 treë. 'n Afstandmeter is egter essensieël by die gebruik van die kruisboog, aangesien dit alleenlik akkuraat is indien die visier op die korrekte afstand ingestel is. Die Cap-Chur geweer se akkuraatheid wissel volgens verskille in dagtemperature. Die Cap-Chur pistool is 'n handige instrument vir die toediening van inspuitings op kort afstande.

Hierdie apparaat kan gebruik word op diere van springbokgrootte en groter. Vir die vang van kleiner wildsbokkies, soos die oorbietjie, steenbok en duiker, word daar van 'n nylonnet gebruik gemaak.

Die middels wat gebruik word vir die immobilisasie van wild, strek van berustingsmiddels tot algemene verdowings- en verlammingsmiddels. Die middels wat gebruik word, is nie ewe doeltreffend en veilig vir alle spesies nie. Die dosis van sommige middels varieer ook van spesies tot spesies. 'n Bespreking oor die voor- en nadele van sommige middels verskyn elders.

Voordat 'n dier met die verdowingspyl-geweer „geskiet" word, moet sekere voorsorgmaatreëls getref word:—

(*a*) Die gewig van die betrokke dier moet so akkuraat moontlik geskat word.

(*b*) Die afstand moet akkuraat bepaal word.

(*c*) Die tipe pyl en naald moet noukeurig gekies word volgens die spesies en die grootte van die dier. In dikvellige diere, byvoorbeeld seekoei, renoster en olifant, word 'n naald gebruik waarvan die punt verseël en skerp is, met openinge aan die distale gedeelte van die skag. Hierdie naald buig nie maklik nie en voorkom dat daar 'n prop uit die vel gesny word en die naald verstop. Die naalde word van weerhakies voorsien om te verhoed dat die pyl uitval voordat die middel ingespuit is.

(*d*) Indien moontlik, moet die pyl in dik spierlae geskiet word, byvoorbeeld die boud. Intra-peritoneale inspuitings is gevaarlik en moet vermy word. Die thoraks van die dier is ook ongeskik aangesien die slaankrag van die kruisboog op kort afstande só groot is, dat selfs 'n wildebees se ribbebene nie daarteen bestand is nie.

In die geval van die seekoei word dit verkieslik in die dorsale gedeelte van die nek geskiet. In hierdie posisie word die pyl nie so maklik onder water afgeskuur teen rotse en ander diere nie.

Indien 'n verlammingsmiddel gebruik word, moet die dier onmiddellik te hulp gesnel word wanneer dit volkome verlam en uitgestrek op die grond lê. By herkouers, byvoorbeeld buffel en wildebees, waarvan die rumen groot en die inhoud vloeibaar is, is die opbring en inaseming van die rumeninhoud nie 'n ongewone verskynsel nie. Sulke diere moet dus onverwyld op die bors gehelp en ondersteun word. By die rooibok en kameelperd kom verstikking minder dikwels voor vanweë die meer droë gehalte van die grootmaag-inhoud.

Die kop, nek en voorlyf van die verlamde dier moet ondersteun en opgelig word om die drukking van die grootmaag op die diafragma te verlig en sodoende asemhaling te vergemaklik. Aangesien die diafragma die laaste spier is wat deur verlammingsmiddels aangetas word, speel dit 'n belangrike rol in die asemhalingsmeganisme van die dier.

Indien 'n oordosis van 'n verlammingsmiddel waarvoor geen teenmiddel beskikbaar is nie, toegedien word, is daar weinig wat gedoen kan word om die dier se lewe te red. In groot diere is die toepassing van kunsmatige asemhaling moeilik en meestal ondoeltreffend. Die toediening van suurstof onder veldtoestande is onprakties. In gevalle waar 'n teenmiddel wel gebruik kan word, moet dit onmiddellik binne-aars toegedien word. In die geval van die renoster kan enige van die onderhuidse are op die oor gebruik word vir intra-veneuse inspuitings.

In die geval van die wildebees, rooibok en ander wildsbokke, word die *V. tarsea recurrens* gebruik. Die enigste sigbare aar aan die seekoei

is die *Vena sublingualis* wat op 'n ongemaklik en ietwat gevaarlike plek geleë is. Gevolglik word daar in dié dier van die ensiem „hyaluronidase' gebruik gemaak om die absorbsie vanaf 'n binnespierse inspuiting te bespoedig.

By die gebruik van algemene verdowings- of narkotiese middels, moet daar sorg gedra word dat die pasiënt ten alle tye in 'n normale en gemaklike posisie lê. In groot diere, byvoorbeeld die buffel, is daar 'n neiging van die agterbene om onder die lyf in te vou. Dit lei tot versteuring van die bloedsomloop wat ernstige komplikasie kan meebring. In gevalle waar die agterbene ooreis word, ontstaan 'n toestand wat klinies vergelykbaar is met paralitiese myoglobinurie by die perd. Met nadoodse ondersoek is 'n degenerasie en nekrose van die spiere duidelik waarneembaar. Die prognose in sulke gevalle is gewoonlik swak. Die pese, veral aan die pootgewrigte raak maklik beseer in groot diere wat struikel as gevolg van onvolkome beheer oor die ledemate.

Opblaas in herkouers is 'n algemene verskynsel by die vang van wilde diere deur middel van verdowings- of verlammingsmiddels. Verligting kan gewoonlik gebring word deur die voorlyf op te lig; in enkele gevalle mag 'n maagbuis van nut wees, maar daar moet nie geaarsel word om 'n trokar en kanula te gebruik wanneer die asemhaling as gevolg van intrarumenale druk bemoeilik word nie. Opblaas is 'n belangrike komplikasie in die kameelperd, aangesien dit nie so maklik waarneembaar is as in ander herkouers nie.

Na ons mening moet 'n wilde dier verkieslik op een van die volgende metodes vervoer word, naamlik:

(*a*) Direk nadat die dier gevang is en nog onder verdowing verkeer; of

(*b*) Eers nadat dit vir 'n geruime tyd in 'n hok of stal aangehou is en mak geword het.

Kalmeringsmiddels is nie altoos ewe doeltreffend in 'n wilde dier wat by sy volle bewussyn is en nog nie aan die mens en nuwe omgewing gewoond geraak het nie.

Nadat die reis 'n aanvang geneem het, moet daarop 'n redelike vinnige dog egalige en aanhoudende spoed volgehou word. Wilde diere is meer rusteloos gedurende 'n stadige en onderbroke rit. 'n Spoed van 30–50 myl per uur kan gehandhaaf word mits die voertuig en die toestand van die pad dit toelaat.

Indien klein diertjies soos oorbietjies vervoer word, en die vasbind van die bene noodsaaklik is, kan daar van dames nylonkouse gebruik gemaak word. Dit is uiters geskik vir dié doel aangesien dit sterk, dog sag is.

Die krat waarin die dier vervoer word, moet vanselfsprekend nie te groot of te klein wees nie. Daarby moet dit donker wees en die dier goed beskerm. Die dier moet egter gemaklik bereikbaar wees.

Onrustige diere word in die meeste gevalle geblinddoek, of daar 'n berustingsmiddel toegedien is al dan nie. Wanneer wilde diere nie kan sien wat om hulle aangaan of in 'n donker omgewing verkeer, is hulle baie meer rustig.

Vanselfsprekend moet die dier gedurende die reis van 'n goeie dik bed voorsien word. Manlike diere (by die kwagga en die renoster) wat

onder die invloed van 'n verdowingsmiddel verkeer, se geslagsorgane moet goed beskerm wees. Indien daar 'n prolaps van die penis is, moet dit opgebind word om skawing of ernstige besering te voorkom.

Dit is noodsaaklik om wilde diere te beskerm teen sekondêre besmetting en komplikasies byvoorbeeld longontsteking, deur die vroegtydige toediening van hoë dosisse antibiotika. 'n Antibiotikum wat gou 'n hoë bloedvlak bereik en ook lankwerkend is, d.w.s. bakteriostaties vir 72 uur, moet toegedien word alvorens die reis 'n aanvang neem. Kortikosteroiede is waardevolle middels vir die voorkoming van spanning en ineenstorting gedurende die reis.

Indien die dier by die bestemming volkome herstel het van die verdowingsmiddel, kan dit onmiddellik vrygelaat word. Wanneer dit egter noodsaaklik is om die dier aan te hou om volkome te herstel, moet dit in 'n donker stal op hok geplaas word. Die dier moet nie kan sien wat buite om hom aangaan nie. Buffels is baie meer rustig wanneer daar meer as een in dieselfde hok is. Wanneer die diere se fiksheid dit toelaat, moet dit vrygelaat word om so gou moontlik in die nuwe omgewing aan te pas, en van spanning onthef word.

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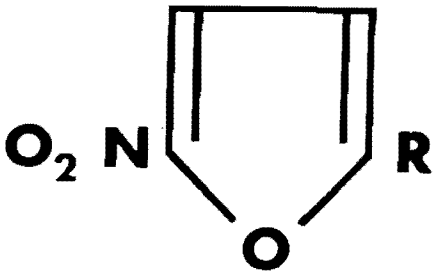

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A COMPARISON BETWEEN NITROGEN RETENTION FROM BIURET AND UREA BY SHEEP ON A LOW PROTEIN ROUGHAGE DIET

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SUMMARY

Comparative nitrogen balance trials in sheep using biuret and urea are reported. Biuret was found to be as efficient as urea when judged by nitrogen retention, apparent nitrogen absorption and stimulation of hay consumption. In view of its safety and palatability, biuret may prove more satisfactory than urea as a non-protein nitrogen supplement to poor roughage.

INTRODUCTION

Experience has shown that the main limiting factor to the expansion of the use of urea as a nitrogen supplement to cattle and sheep on low protein winter fodders in South Africa is its toxicity. Although losses have been negligible in proportion to the advantages gained and usually due to non-compliance with instructions, one accident will prejudice a farmer against urea feeding for many years.

Numerous workers, mainly in America, have shown that biuret, a condensation product of urea, is as efficient a source of nitrogen to ruminants as is urea. It is also reported to be non-toxic. Hatfield *et al*[1] obtained positive nitrogen balances after a lag period of some 18 days in sheep receiving biuret. Weight gains, after this period, were slightly better than those in sheep receiving an equivalent amount of urea. Campbell *et al*[2] found that biuret promoted slightly, but not significantly, lower growth and feed utilisation efficiency in growing calves when compared to urea. Biuret also appeared to be slightly inferior to urea in promoting the production of fat corrected milk and exhibited lower nitrogen retention when fed to bull calves. These authors also report a lag period of three to four weeks before optimal utilisation of biuret, or urea, was reached. From a feeding trial involving 50 lambs, Meiske *et al*[3] report no significant differences in weight gains when biuret, urea or soyabean were used as nitrogen sources.

Ewen *et al*[4] showed that the lag period in the utilisation of biuret could be shortened considerably by dosing with ruminal ingesta obtained from animals which had been fed biuret for some time previously, indicating the necessity for the development of a flora with specific biuret hydrolysing powers.

As regards toxicity, Hatfield *et al*[1] dosed up to 275 g. biuret to 85 lb. sheep without producing any ill effects. As these trials were apparently performed on unadapted sheep, and the development of a biuret hydrolysing flora might well increase the potential toxicity of biuret, the following preliminary experiment was carried out by the present authors.

Two sheep each weighing some 80 lb., which had been kept on a diet of teff hay *ad lib.* plus 100 g. maize meal per day for 12 days previously, were dosed 250 g. biuret per ruminal fistula. The pH of the ruminal contents was determined before and periodically for 6 hours after dosing. No significant rise occurred, the readings all falling between 6.5 and 6.9. The animals showed no ill effects and consumed their normal ration after the experiment was completed and subsequently. At the same time a third sheep of the group was dosed with 30 g. urea. The pH of the ruminal contents rose from 6.5 to 8.0 in 30 minutes and typical signs of urea poisoning appeared. The animal died two hours after, despite being given large doses of acetic acid. The two remaining sheep were then kept for 9 weeks on the original ration plus 12 g. biuret per day. They were then dosed with 250 g. biuret as before. Again no signs of poisoning were produced but on this occasion the pH of the ruminal contents rose more definitely (6.1 to 6.5 and 5.7 to 6.5 respectively).

This trial showed that biuret was non-toxic, even after adaptation of the ruminal flora.

In view of these facts, further work into the value of biuret as a possible substitute for urea under South African conditions is being undertaken. As Clark and Barrie[5] had shown that the addition of cobalt increased the utilisation of urea when certain poor quality roughages formed the basic diet, the effect of this factor was also tested in the trials to be reported.

Methods

Six 6-toothed merino wethers with an average weight of 75 lb. were used throughout the experiments. The basic daily ration per sheep consisted of teff hay *ad lib* plus 120 g. of a mixture consisting of 95 per cent crushed yellow maize and 5 per cent "molasses distillers dried solubles". In addition 10 g. sodium chloride and 10 g. dicalcium phosphate were mixed into the meal and 0.5 g. "Duravit" (synthetic vitamin A) was dosed weekly. The nitrogen supplements were mixed into this mixture.

Table 1

Relevant Composition of the Teff Hay, Meal Mixture and Biuret

	Nitrogen %	Cobalt p.p.m.	Copper p.p.m.
Teff Hay.....	0.71	0 08	4.5
Meal Mixture.....	1.23	0.06	3.0
Biuret.....	40.00	—	—

In order to accustom the animals to the experimental diet and conditions, they were kept for a preliminary period of three weeks in small individual pens and fed the basic ration.

The nitrogen balance trials were conducted in the standard manner, each collection period consisting of five consecutive days with a nine day interval between each, during which the animals were kept in their pens and fed the experimental ration. Each series consisted of six collection periods, one without non-protein nitrogen supplementation and five with supplementation. The animals therefore received each supplement for a continuous period of ten weeks. Between series the animals were kept on the basal ration in pens for four weeks and then for a further week for the basal collection period. The non-protein nitrogen supplements used were as follows (per sheep per day):—

Series 1. 12 g. biuret (98 per cent pure).
Series 2. 12 g. biuret + 1 mg. cobalt as cobalt sulphate.
Series 3. 10.5 g. urea (46 per cent N).

Results

Table 2

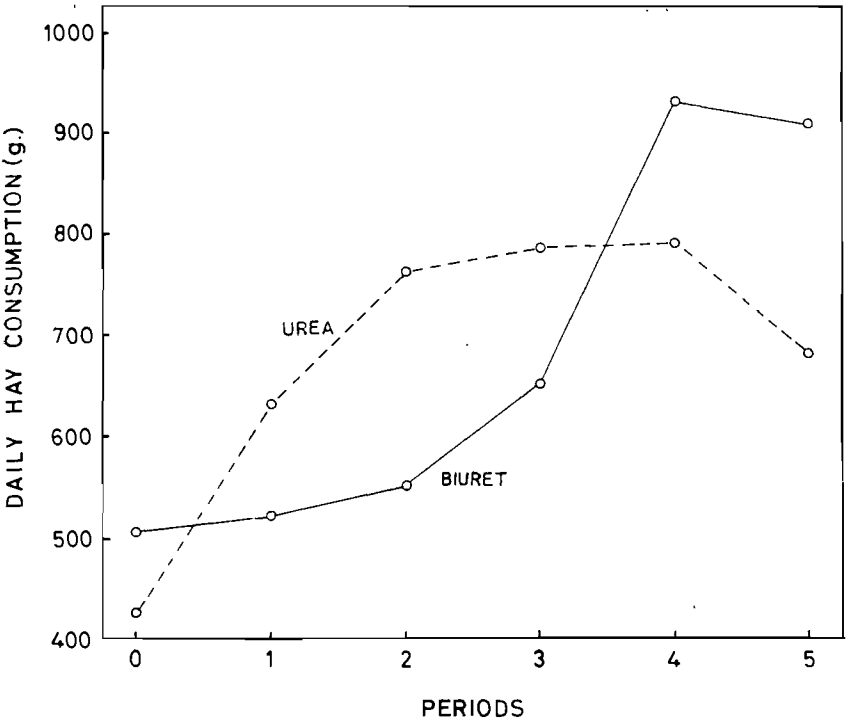
Nitrogen intake, nitrogen balance and apparent nitrogen absorption

Period	Average Nitrogen Intake per Period (g)			Average Nitrogen Balance (g)			Apparent Percentage Nitrogen Absorption		
	Biuret	Biuret + Cobalt	Urea	Biuret	Biuret + Cobalt	Urea	Biuret	Biuret + Cobalt	Urea
Basal...	25.2	30.2	23.3	−0.6	+0.6	−0.1	68.3	75.6	57.1
1.....	49.9	54.8	54.5	+7.4	+9.1	+5.4	55.8	63.4	63.0
2.....	52.1	61.1	61.4	+9.5	+10.3	+9.9	56.4	67.2	61.6
3.....	51.0	63.2	61.9	+10.5	+8.7	+10.8	63.9	60.2	61.2
4.....	61.4	65.3	62.0	+10.5	+9.6	+10.4	67.9	65.4	61.3
5.....	59.6	65.2	58.5	+11.2	+9.3	+9.3	64.4	63.4	62.4

A positive nitrogen balance was obtained in all three series of experiments, there being no material difference in the results as regards nitrogen retention or apparent nitrogen absorption.

The hay consumption was improved by both biuret and urea but this effect of biuret was more delayed. In both series there was a slight decline in consumption during the last period. This may have been due to "metabolism cage fatigue".

The addition of cobalt made no difference to the utilisation of biuret nor to hay consumption, indicating that the cobalt level in the basic diet was adequate.



Discussion

The problem in South Africa is to find a cheap, safe nitrogen supplement which can be used with high fibre, low protein roughages. The object of such supplementation is not only to supply the animal with the required protein, via microbial synthesis, but also to stimulate and maintain cellulose breakdown by the ruminal flora, which on such roughages is limited by the amount of nitrogen available. That this was attained by both urea and biuret is indicated by the stimulus to hay intake, which indicates a more rapid breakdown of fibre as shown by Hoflund *et al.*[6] The results indicate that biuret fulfills both these functions as efficiently as does urea. The fact that biuret is non-toxic will make it far more suitable for general use.

The present results do not indicate a long latent period before biuret can be utilised, as described by the authors cited above, as positive nitrogen retention was obtained during the first five days of biuret feeding. However, maximum retention was attained only after six to eight weeks. The effect of biuret on hay consumption also reached its maximum only after a similar period, indicating adaptation of the flora.

During the trials it was very obvious that the sheep ate the maize meal much more avidly with biuret than with urea. Biuret does not appear to be at all unpalatable to sheep.

Biuret may well prove to be much more suitable than urea as a non-protein nitrogen supplement to high fibre, low protein roughages under South African condition, owing to the availability of its nitrogen, its safety and its palatability. Further basic research and field trials are being planned on a considerable scale.

ACKNOWLEDGMENTS

The Chief, Veterinary Research Institute, Onderstepoort is thanked for permission to publish in this Journal.

African Explosives and Chemical Industries Ltd., are thanked for the supply of biuret.

REFERENCES

1. HATFIELD, E. E., GARRIGUS, U. S., FORBES, R. M., NEUMANN, A. L. and GAITHER, W. (1959): "Biuret—a source of N.P.N. for ruminants." *J. Animal Sci.*, **18,** 1208.
2. CAMPBELL, T. C., LOOSLI, J. K., WARNER, R. G., and TASAKI, I. (1963): "Utilisation of biuret by reminants". *J. Animal Sci.*, **22,** 139.
3. MEISKE, J. C., VAN ARSDEL, W. J., LUECKE, R. W. and HOEFER, J. (1955): "The utilisation of urea and biuret as sources of nitrogen for growing-fattening lambs." *J. Animal Sci.*, **14,** 941.
4. EWEN, R. C., HATFIELD, E. E. and GARRIGUS, U. S. (1958): "The effect of certain inoculations on the utilisation or urea of biuret by growing lambs". *J. Animal Sci.*, **17,** 298.
5. CLARK, R. and BARRIE, N. (1959): "The effects of soluble phosphate and cobalt on the utilisation of urea nitrogen by cattle". *J.S. Afr. vet. med. Assoc.*, **30,** 457.
6. HOFLUND, S., QUIN, J. I., and CLARK, R. (1948): "Studies on the alimentary tract of merino sheep in South Africa XV. The influence of different factors on the rate of cellulose digestion (*a*) in tne rumen and (*b*) in ruminal ingesta as studied in vitro. *Onderstepoort J. vet. sci. and animal ind.*, **23,** 395.

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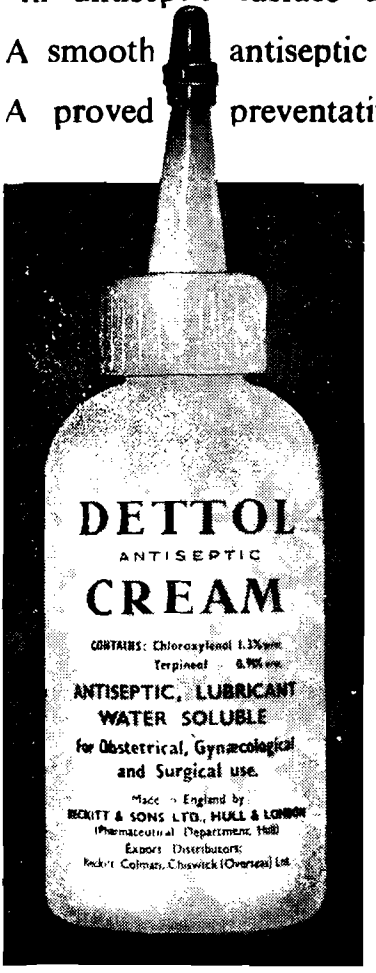

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DECREASE IN PLASMA POTASSIUM FOLLOWING RESECTION OF THE JEJUNUM IN TWO THOROUGHBRED MARES

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J. M. M. BROWN — Department of Physiology and Biochemistry, Onderstepoort

Received for publication, June 1963

Potassium metabolism has received close attention in human surgery, and the post-operative loss of potassium which is often associated with alkalosis and lowered serum potassium concentrations is well documented.[1–10]

The rôle of the kidney in this depletion is appreciated,[11] but although a potassium secretory function of the kidney cells is suspected, the mechanism is not fully understood. The most important factors have been reviewed by Lambie[12] who also produced evidence supporting sodium/potassium exchange mechanisms in the distal tubule.

In equines, however, although attention has recently been directed to the problem of potassium depletion following bowel surgery[13] and also associated with severe diarrhoea,[14] there is a lack of available data on the subject. It is perhaps significant that according to a recent report, the potassium requirements of the horse are as yet unknown.[15]

The results of investigations on certain blood constituents during and after experimental resection of the jejunum in two throroughbred mares are presented and discussed.

MATERIALS AND METHODS

Case I

A twenty-year-old thoroughbred mare was anaesthetised with chloral hydrate (270 ml. of a 20 per cent solution i.v.) followed by pentobartibone sodium ("Sagatal", M. & B.) until a relatively deep plane of anaesthesia was reached as evidenced by cessation of nystagmus and closure of the eyelids. This required 25 ml. and a further 10 ml. was injected approximately one hour later shortly before cutting through the intestine. Immediately after induction of anaesthesia, 20 ml. of 5 per cent solution mepyramine maleate ("Anthisan", M. & B.) was injected slowly intravenously.

Laparotomy was performed by a low flank incision on the left side, and a loop of jejunum 4 ft. in length was exposed, laid on the cloth, and a tape ligature applied and tightened until pulsation of the arterial branches in the mesentery almost ceased. The ligature was left on for fifty minutes. Before removing the ligature, the mesentery in the loop was stretched until tearing occurred. The loop of bowel was then resected and an end to end anastomosis carried out by the method of Moynihan.[16]

It was intended that this procedure would closely simulate resection of a strangulated and traumatised loop of intestine.

The bowel was then replaced and the peritoneum and the transverse fascia were sutured with a continuous suture of No. 4 chromic catgut. The external oblique, and int. oblique muscles were also sutured with a continuous suture of No. 4 chromic catgut, and the skin with nylon.

The appropriate blood samples were taken for investigation at the following stages:—

(1) Pre-operative.
(2) Immediately before ligation of the loop of jejunum.
(3) Immediately before tearing of the mesentery.
(4) Immediately after tearing of the mesentery.
(5) After suturing of peritoneum and muscles.
(6) 17.5 hours post-operatively.
(7) 65.5 hours post-operatively.
(8) 89.5 hours post-operatively.

Case II

A fifteen-year-old thoroughbred mare was anaesthetised with chloral hydrate (280 ml. i.v. of 20 per cent solution) followed by 28 ml. pentobarbitone sodium ("Sagatal", M. & B.). A further 16 ml. was administered during the course of the operation. As soon as anaesthesia was accomplished, the lateral digital artery of the off fore was exposed, canulated, and connected to a mercury manometer registering pressure directly in mm.Hg. The pressure was noted to fluctuate regularly between 130–150 mm.Hg. Immediately after induction of anaesthesia 20 ml. of 5 per cent solution mepyramine maleate ("Anthisan", M. & B.) was injected slowly intravenously.

Laparotomy was then performed by a low flank incision on the right side, and a large loop of jejunum was exposed. This loop was handled for a few minutes and the mesentery stretched but not torn.

A five foot length of jejunum was then resected and an end to end anastomosis carried out by a method which had previously been used with success in bowel resections in equines, i.e., with a curved needle, the cut edges of the mucosa and muscle layers are picked up and sewn together, using a continuous suture of plain "O" catgut. A continuous Lembert suture is then placed, including serosa and muscle layers, and burying the first suture. The method neatly maintains the internal continuity of the bowel as well as the external, and appears to be surgically sound. Peristalsis was seen to pass across the anastomosis within one minute of its completion.

Peritoneum, muscles and skin were then sutured as in Case I.

The appropriate blood samples were taken at the following stages:—

(1) Pre-operative.
(2) After making the skin incision.
(3) After resection of the jejunum.
(4) Immediately after replacement of the bowel in the abdominal cavity.
(5) After completion of the operation.
(6) 17.5 hours post-operatively.
(7) 41.5 hours post-operatively.

Clinical Laboratory Analyses: Methods

Haematological and biochemical determinations were carried out as follows:—

Packed cell volume (PCV.) was read after centrifugation at 3,000 r.p.m. for 30 minutes.

Haemoglobin (Hb) was estimated by the cyanmethaemoglobin method of Drabkin, as described by King and Wootton.[17]

Total plasma protein (Pl. Pr.) was determined by the modified biuret method of Weichselbaum.[18]

Blood sugar was determined by the modified Folin and Wu method of Lehman and Silk.[19]

Plasma sodium (Na^+) and potassium (K^+) were estimated with an E.E.L. flame photometer using the techniques described by King and Wootton.[17]

Plasma chlorides (Cl^-) were determined by the method of Schales and Schales[20] and plasma bicarbonates (HCO_3) by titration as described by van Slyke, Stillman and Cullen.[21]

The parameters obtained from the results of blood analyses are shown in figs. 1 and 2 below.

Clinical Course

Case 1

The mare rose to her feet about one hour after the end of the operation and after about twenty minutes, was put in a loose box. She passed faeces and ate cut grass with apparent relish.

On 8.2.61, she showed symptoms of colic approximately 28 hours post-operatively and was treated with chlorpromazine ("Largactil", M. & B.) and mepyramine maleate ("Anthisan", M. & B.). Passing of a stomach-tube was contra-indicated since struggling would have endangered the sutures.

On 9.2.61 the mare was still showing considerable discomfort, with restlessness and pawing of the floor. Administration of 4 ml. "Prostigmine" (Roche) at 10 a.m. and 4 p.m. produced no evacuation of faeces and thereafter she was maintained on "Pethidine" (Roche) in maximal doses for control of pain. No blood samples were taken from this mare on the 9th (since the five samples from Case II were straining resources) but it was apparent that a decision had to be made between obstruction or true paralytic ileus. There were no clinical signs of peritonitis. On the night of 9.2.61 it was decided that all the evidence pointed to paralytic or inhibitional ileus. At this stage the symptoms were as follows:— Pulse 60, Temperature 101.2°F. The mare was extremely restless, and walked slowly around the loose box occasionally pawing the ground, and lying down for a few minutes without rolling. There was light patchy sweating, and trembling of the triceps and of the thigh muscle groups was noticeable. The face and eyes showed the typical anxious expression of a horse with acute abdominal discomfort. Peristalsis was completely absent, but the bowels appeared to contain a large quantity of fluid which could be heard gurgling when the abdominal wall was pressed inwards. No faeces had been passed for twenty-four hours.

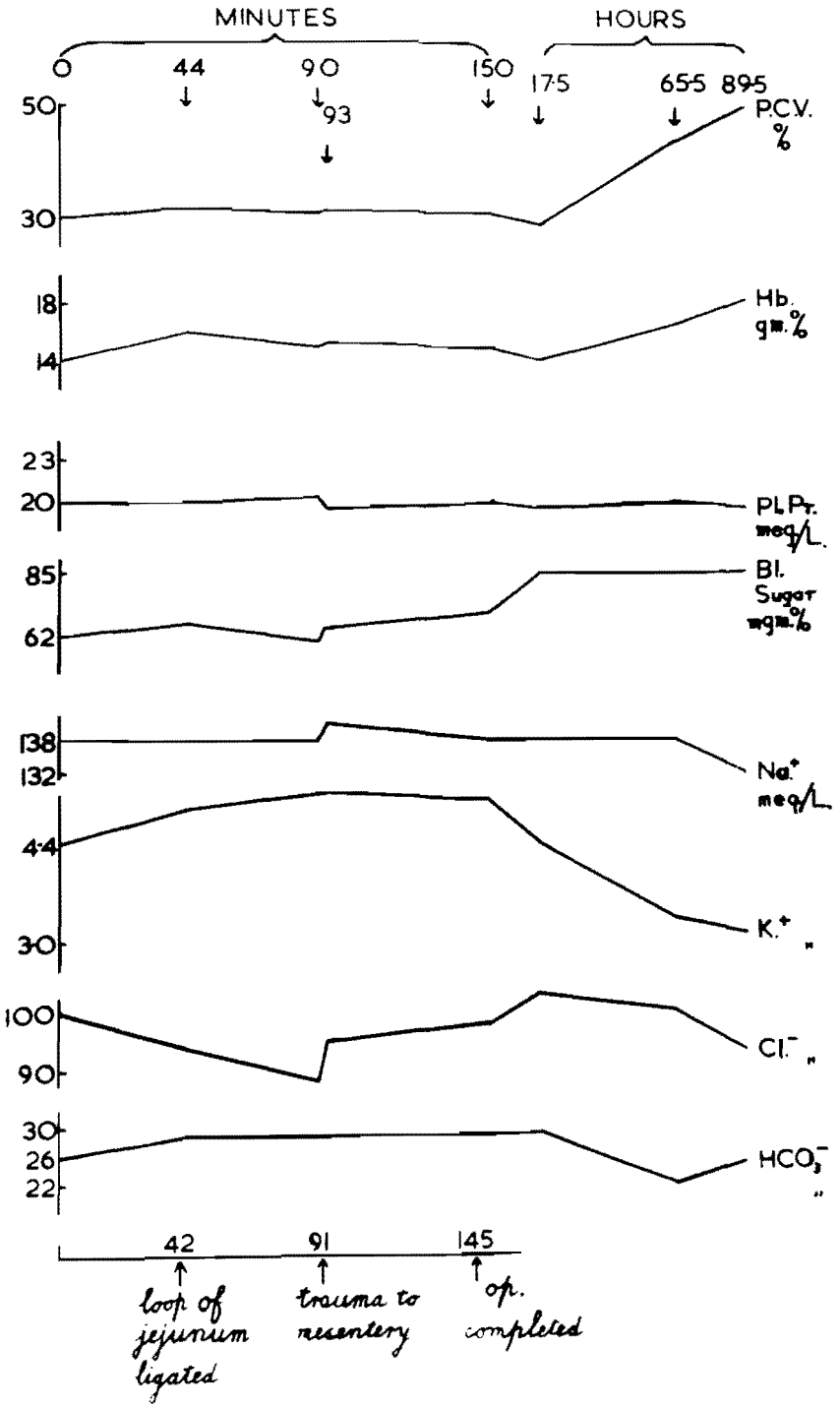

Fig. 1. Case 1. The concentrations of electrolytes, blood sugar, packed cell volume, and haemoglobin before, during and after ligation and resection of the jejunum. Times at which blood samples were taken are shown at top.

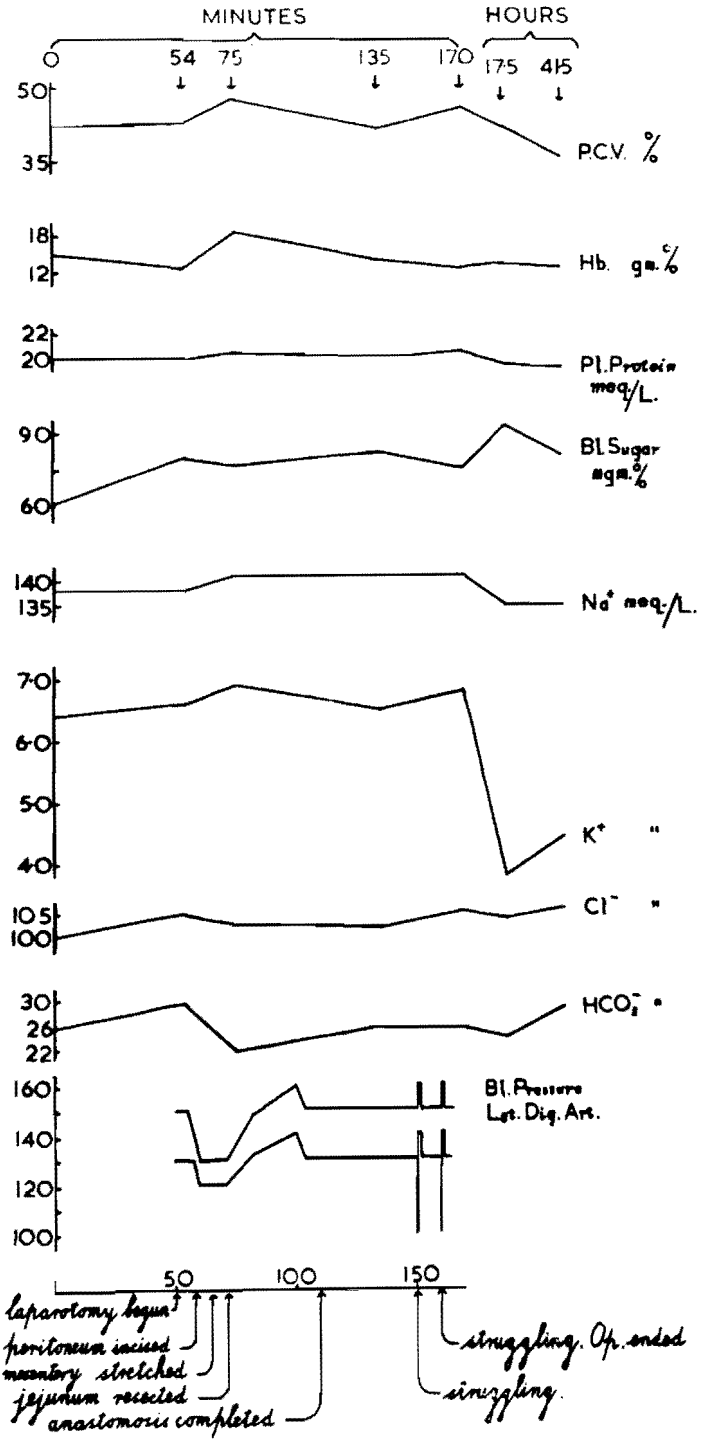

Fig. 2. Case 2. Blood pressure in the lateral digital artery is indicated in mm. Hg. The two lines show the upper and lower limits of the fluctuations in the reading caused bythepulse wave.

The lowered plasma potassium concentration found on the following morning supported this diagnosis to some extent, and in the course of the next 48 hours the mare received a total of:—

Intravenously 6 litres of glucose-saline containing 7 gm. KCl.
Per os 12 litres of glucose-saline containing 20 gm. KCl.
Per rectum 12 litres of glucose-saline containing 20 gm. KCl.

In spite of this, plasma K decreased still further. The mare died on the night of 11.2.61.

Autopsy findings: The stomach was ruptured along the greater curvature for approximately 6 inches. The abdominal cavity contained several gallons of fluid and as there was no evidence of peritonitis in any part of the abdominal cavity, death must have ensued very quickly after the rupture, presumably from shock. The small intestine was greatly distended throughout its length, pale in colour, and contained more than one gallon of pale watery fluid. The anastomosis appeared to be healing very well, and the bowel lumen was fully patient. It was noted, however, that the method of suturing formed an internal "flange" which decreased the diameter of the lumen by about 15–20 per cent.

Case II

Rose to her feet about 90 minutes after the end of the operation and immediately passed a large amount of faeces and began to eat. Thereafter, in spite of a drop in plasma potassium on the first post-operative day, her progress was excellent. She never showed colic, never stopped eating, and the laparotomy wound healed without complications. Prophylactic intramuscular injections of penicillin were given for four days post-operatively. The digital artery was ligated after removal of the canula, and the wound required dressing for some weeks after. Lameness persisted for three months, after which the mare appeared to be going quite sound. She has since been used as a hack and lives a normal life as far as can be judged.

Discussion

In view of the lack of knowledge concerning the behaviour of the potassium ion in equines under the stress conditions imposed by surgery, comment on the significance of the findings is largely speculative. The decreases in plasma potassium concentration of 1.3 meq./L. in Case I in spite of attempted replacement and of 2.6 meq./L. in Case II are grossly abnormal. The normal ranges for potassium in equine sera as determined by various authors are given in Table I. In thoroughbreds, the mean value for plasma according to Sréter[22] is 3.88 meg./L.

Table 1

Normal range of potassium concentration in equine serum

Author	Date	Range mg. %	Meq./L.
Kolatschny, R.[23]	1948	19.13–22.95	4.9–5.9
Kment, A.[24]	1949	18.16–24.52	4.6–6.3
Bruning, P.[25]	1950	15.68–18.38	4.0–4.7
Sabjan, I[26]	1955	17.53–20.42	4.4–5.1

In view of the suggestion of several authors that in man potassium should be supplemented by mouth as soon as possible, this was resorted to in Case I. However, the equine stomach is singularly unable to tolerate overloading, owing to its extremely small size relative to total gastro-intestinal capacity[27] and also because of its inability to vomit. In order to avoid rupture of the stomach, the treatment of choice in this condition should, therefore, be some form of gastric aspiration combined with purely intravenous replacement. Pethidine has been recommended for control of pain in human cases in preference to morphine or its derivatives.

The influence of potassium on gastro-intestinal motility in humans with paralytic ileus is well known.[28] The suggestion that potassium deficiency alone is the cause of paralytic ileus has not gained universal acceptance since paralytic ileus can occur as a result of various other conditions (e.g. sub-peritoneal haemorrhage, or rib fracture). Current medical opinion regards overstimulation of the neuromuscular mechanism of the plexus of Auerbach as playing a most important part in the genesis of the syndrome.

The slight increase in total anions and decrease in cations on 11/2/61 (Case II) indicate a mild metabolic acidosis, for which the authors can offer no explanation.

The increase in blood sugar from fasting to normal average levels may be a reflection of adrenal cortex activity.

The blood pressure readings in Case II are of special interest. Although a noticeable fall in arterial pressure occurred after penetration of the peritoneum, neither handling of the intestines and mesentery, nor the resection itself, produced any further fall in pressure. Towards the end of the operation, struggling occurred. Immediately before each struggle, the blood pressure dropped spectacularly for a fraction of a second, then rose above the normal level during the period of the struggle. The momentary drop in blood pressure would appear to indicate a sudden increase in the volume of the capillary bed of the muscles, which is immediately over-compensated by the various responses of the whole haemodynamic system.

These two cases undoubtedly point to the necessity for:—

(*a*) Further investigation into electrolyte metabolism in equines. The findings here suggest that the potassium ion plays as important a rôle in equine surgery as it does in human.

(*b*) The development of a simple efficient and easily transportable gastric decompression apparatus for use in equines with obstruction of the bowel. The figures of Robertson, Burgess, Inglis and Paver[29] are of exceptional interest in this respect. They aspirated daily amounts of 20–154 litres from the stomachs of three horses suffering from acute grass sickness, a disease which presents a somewhat similar clinical picture to acute intestinal obstruction. The spectacular, though temporary, results which they obtained suggest that the principle could be extended to bringing obstruction cases into a fit condition for surgery.

ACKNOWLEDGMENTS

The authors wish to thank Dr. B. C. Jansen, Chief of the Veterinary Research Institute, Onderstepoort, Republic of South Africa, for permission to publish this article. The helpful advice and generous provision of facilities given by Professor C. F. B. Hofmeyr, Head of the Department of Surgery, and Professor R. Clark, Head of the Department of Physiology and Biochemistry, Faculty of Veterinary Science, University of Pretoria, are also gratefully acknowledged. The assistance of Dr. D. H. G. Irwin during the operations is acknowledged with thanks, as also the technical assistance given by Mr. P. J. de Wet of the Department of Physiology and Biochemistry of the above faculty. Last, but not least, a special vote of thanks is accorded to Mrs. Suzanne Littlejohn, who kept detailed records of both operations.

REFERENCES

1. BLIXENKRONE-MOLLER, N. (1949): Potassium metabolism and potassium treatment in surgical patients. *Acta. chir. Scandinav.*, **99,** 237.
2. PEARSON, O. H. and ELIEL, L. P. (1949): Post-operative alkalosis and potassium deficiency. *J. Clin. Invest.*, **28,** 803.
3. ELIEL, L. P., PEARSON, O. H. and RAWSON, R. W. (1950): Post-operative potassium deficit and metabolic alkalosis. *New Eng. J. of Med.*, **243,** 471.
4. EVANS, E. I. (1950): Potassium deficiency in surgical patients; its recognition and management. *Ann. Surg.*, **131,** 945.
5. MARKS, L. J. (1950): Potassium deficiency in surgical patients. *Ann. Surg.*, **132,** 20.
6. HEWITT, H. C. (1950): Potassium metabolism in the post-operative patient. *Surg. Forum, Proc. 36th Clin. Congress Am. Coll. Surgeons.*, p. 456.
7. HAWKINS, C. F., HARDY, T. L. and SAMPSON, H. H. (1951): Potassium deficiency in pyloric stenosis. *Lancet*, **1,** 318.
8. LANS, H. S., STEIN, I. F., Jr., and MEYER, K. A. (1952): Occurrence and treatment of potassium deficiency in surgical patients. *J. Internat. Coll. Surgeons*, **17,** 34.
9. MOORE, F. D., and BALL, M. R. (1952): Metabolic Response to Surgery. Charles C. Thomas. Springfield, Ill.
10. BENZER, H. (1961): Postoperative Kaliumverluste durch Stressreaktionen. *Wein, Medizin. Wschr.*, **10,** 180.
11. TARAIL, R. and ELKINTON, J. R. (1949): Potassium deficiency and the rôle of the kidney in its production. *J. Clin. Invest.*, **28,** 99.
12. LAMBIE, ANNE T. (1960): Mechanisms of potassium loss by the kidney. Symposium on Water and Electrolyte Metabolism. Elsevier Pub. Co.
13. ELLET, E. W., JONES, E. W., and JOHNSON, L. (1958): Obstruction of the colon in the horse. *Vet. Med.*, **53,** 291.
14. JASTREMSKI, M. and FERREBEE, J. W. (1956): Fluid replacement in the treatment of dehydration from diarrhoea in the horse. *J.A.V.M.A.*, **128,** 153.
15. National Academy of Science, National Research Council, Washington, D.C., Publication 912 (1961): Nutrient requirements of horses.
16. MOYNIHAN, B. (1928): Abdominal Operations. W. B. Saunders, Co., Phila. and Lond., 4th Ed.
17. KING, E. J. and WOOTTON, I. D. P. (1956): Microanalysis in Medical Biochemistry, 3rd Ed. J. and A. Churchill, Ltd., Lond.
18. WEICHSELBAUM, T. E. (1946): An accurate and rapid method for the determination of proteins in small amounts of blood serum and plasma. *Am. J. Clin. Path.*, **16,** 40.
19. LEHMANN, H. and SILK, E. (1952): The prevention of colour fading in the Folin and Wu estimation of blood sugar. *Biochem. J.*, **50,** XXXI.
20. SCHALES, O. and SCHALES, S. S. (1941): A simple and accurate method for the determination of chloride in biological fluids. *J. Biol. Chem.*, **140,** 879.
21. VAN SLYKE, D. D., STILLMAN, E. and CULLEN, G. E. (1919): A method for titrating the bicarbonate content of plasma. *J. Biol. Chem.*, **38,** 167.
22. SRÉTER, F. (1959): Changes of blood electrolytes in thoroughbreds as a result of training and exercise. *Can. J. Biochem. & Phys.*, **37,** 273.
23. KOLATSCHNY, R. (1948): Bestimmung des Kaliumgehaltes im Blutserum wurmkranker Pferde. Inaug. Diss. Hannover.

24. KMENT, A. (1949): Kaliumbestimmungen im serum von Haustieren. *Wien. tierärztl. Mschr.*, **7**, 398.
25. BRÜNING, P. (1950): Bestimmung des Kaliumgehaltes in Blutserum und Liquor cerebrospinalis bei Pferden. Inaug. diss. Hannover.
26. SABJAN, I. (1955): Potassium content in blood serum of healthy horses, cattle, sheep and dogs. *Veterinaria, Sarajevo*, **4**, 69.
27. ALEXANDER, F. (1954): A review of knowledge available concerning digestion in domestic herbivores. *Br. Vet. J.*, **110**, 146.
28. GAZES, P. C., RICHARDSON, J. A. and COTTEN, M. DE V. (1951): Effects of potassium chloride on intestinal motility in human beings and dogs. *J. Lab. and Clin. Med.*, **37**, 902.
29. ROBERTSON, A., BURGESS, J. W., INGLIS, J. S. S. and PAVER, H. (1948): Observations on gastric decompression and intravenous saline administration in acute grass disease. *Vet. Rec.*, **60**, 495.

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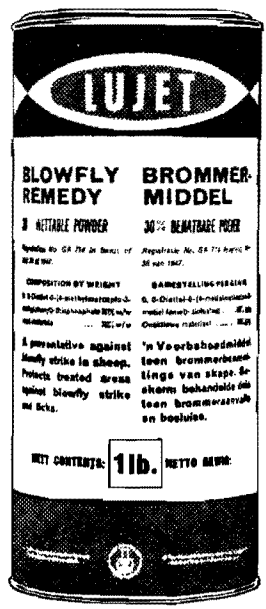

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FILAROIDES OSLERI: THE SUCCESS OF THIACETARSAMIDE SODIUM THERAPY

J. E. DORRINGTON — Private Practitioner, 4 Kort Street, Bellville, C.P.

Received for publication, May 1963

SUMMARY

The experimental results obtained with an arsenical compound in the treatment of *Filaroides osleri* is described.

INTRODUCTION

Since the report by Dietrich[1] on the successful treatment of one case of *Filaroides osleri* with Thiacetarsamide Sodium, extensive trials were undertaken by me to confirm or otherwise, the efficacy of this drug against *F. osleri*. *Thiacetarsamide Sodium* (Caparsolate Sodium, Abbotts) is a trivalent arsenical compound used for the therapeutic treatment of canine heartworm (*Dirofilaria immitis*). It is highly specific against the adult worms but not against the microfilariae in the bloodstream. Approximately 50 per cent of the drug is excreted via the urine and faeces in the first 24 hours after administration. The recommended dose today for heartworm is 0.1 ml. per pound bodyweight intravenously twice a day for two days. This amounts to 0.4 mg of arsenic per pound per day while the fatal dose is 0.9 mg. per pound per day. Toxicity was noticed as soon as 0.6 mg. arsenic per pound per day was administered usually from the third day onwards. This could vary in individual cases as some tolerated this dose for seven consecutive days. Withdrawal of the drug resulted in a quick return to normal.

Perivascularly, Caparsolate Sodium is irritant causing a hot painful swelling.

DIAGNOSIS

Positive diagnosis can be accurately achieved only when using the direct illuminated bronchoscopic technique. Swabs taken from the trachea and/or faeces examinations, can yield negative results in known positive cases for months at a time.[2] Dogs are anaesthetised with Thiopentone Sodium and examined with the bronchoscope in dorsal recumbency. No difficulty should be experienced in arriving at a positive diagnosis, as the papillomata are clearly visible in the region of the bifurcation of the trachea.

Methods

When using Caparsolate Sodium, no difficulty was experienced in administering the drug intravenously using the same vein whether given three times a day or daily for 21 days, if the following procedure is followed:— Use a small guage needle and after administering the drug allow some blood to reflow through the needle, to avoid contamination at the time of needle removal. Then rub the area in the direction of blood flow to seal the vein puncture entirely.

Experiment 1

Initially, the following trials with Caparsolate Sodium were undertaken:—

(i) Two positive dogs were given 0.1 ml per pound body weight twice a day for two days.
(ii) Two positive dogs were given 0.1 ml per pound body weight daily for 14 days (the old dosage schedule for heartworm therapy).
(iii) One positive dog was given 0.1 ml per pound body weight daily for 21 days (the dosage schedule Dietrich used).

Results

Bronchoscopic examination conducted two weeks after treatment showed a whitening of some of the papillomata present. Re-examination two months after treatment showed the 21 day course dog completely clear of any papillomata while in all the other cases a marked reductio n in the number of nodules was seen.

Experiment 2

With these encouraging results, four more positive cases were given 0.1 ml per pound weight daily for 21 days and re-examined two months after treatment.

Results

Table 1

Results of 21 day course

Number of dogs	Two months after treatment
2	No visible papillomata
1	Three white nodules
1	Two loose pedicled nodules

Experiment 3

1. One dog received 0.1 ml per pound body weight twice a day for 5 days.
2. One dog received a similar amount for 7 days.

Results

The results obtained one month after treatment, were disappointing in that no significant improvement was visible over the 21 day course; in fact the reduction in papillomata was less than with the 21 day course.

Experiment 4

After consulting Otto,[3] a further course was administered to the dog number 2 in experiment 3 but it now received 0.1 ml per pound body weight three times a day (the dog now received 0.6 mg of arsenic per pound body weight per day which is $\frac{2}{3}$ the fatal dose.)

Results

On the third day the dog started vomiting continuously which forced a temporary abandonment of the trial for 7 days. I then restarted giving this dog the same dose (0.1 ml per pound three times a day) for 5 days without any toxic results. Six weeks after treatment, re-examination showed this dog to be still as positive as before. Another dog similarly treated subsequently yielded similar results.

Discussion

1. The successful trial by Dietrich using Thiacetarsamide in treating one case of *F. osleri* is confirmed although complete cures in all cases were not obtained.

2. The papillomata in the bronchi of dogs suffering from *F. osleri* disappear when the parasites are destroyed. This fact can be used to confirm the efficacy of any anthelmintic against *F. osleri.*

3. Caparsolate Sodium is the only drug at present which has proved effective against *F. osleri* and until such time as some other drug becomes available, it is considered to be the indicated drug. The recommended dosage level is 0.1 ml per pound body weight intravenously daily for 21 days. A shorter more intensive course, although most desirable practically, yielded disappointing results. This could be ascribed to the fact that Caparsolate Sodium is possibly effective against the adult worms here only too and allows the immature worms to develop into the adult stage after the short intensive course has ceased.

Authors Notes

1. At present research is being done into the as yet unknown life cycle.

2. Other drugs and techniques are being investigated and

3. may I request all colleagues to supply whatever information and experience they may have had so as to facilitate the final eradication of this serious infection.

ACKNOWLEDGMENTS

I would like to thank Abbott Laboratories (S.A.) for arranging the supplies of Caparsolate Sodium and also Dr. G. F. Otto, Director, Veterinary Research, Abott Laboratories, U.S.A. for his help and useful advice on the use of Caparsolate Sodium.

REFERENCES

1. DIETRICH, LE ROY E. (1962): *J.A.V.M.A.* **140** 572–573.
2. LAUDER, I. (1962): University Veterinary Hospital, Bearsen, Glasgow Personal communication.
3. OTTO, G. F. (1962): Abbott Laboratories, North Chicago, Illinois, U.S.A Personal communication.

CASE REPORT: BOVINE CEREBELLAR HYPOPLASIA ASSOCIATED WITH VICARIOUS HYDROCEPHALUS

J. A. SCHUTTE — Senior State Veterinarian, Queenstown

SUMMARY

1. A case of bovine internal hydrocephalus due to cerebellar hypoplasia is described.

2. Apart from nervous disturbances the main feature was the absence of cranial deformation as is normally encountered with hydrocephalus.

HISTORY

Attention was drawn to the peculiar behaviour of a pure bred bull calf born on 1st June 1962. At birth of the calf was normal in all respects as regards its size, weight and appearance. The previous calves of the dam have all been normal.

CLINICAL HISTORY

From birth the calf had difficulty in suckling, which improved gradually until it drank normally. It showed an inclination to fall over and exhibited ataxia when unduly disturbed; its mental faculties were otherwise normal in every respect, like bellowing when hungry and shying away when frightened. The cardiac and respiratory rates as well as ocular and muscular reflexes, were normal. Directional vision was normal, unlike bovine cerebral theileriosis, where the alignment of the eyes is often found to be divergent.

One notable symptom was the desire of the calf to hold the head in the normal position and every effort was made by the animal to rectify its balance as quickly as possible if it was disturbed or tipped over. It is worthy to note that ataxia was bilateral in equal degree. Another point of interest is the fact that coenuriasis does not occur on this particular farm.

The degree of locomotor derangement has remained the same up to the time of slaughter.

POST MORTEM

The calf was destroyed on 8th December, 1962, by severing its throat and bleeding it completely. The spinal cord was left intact. No undue volume of cerebrospinal fluid was observed when the head was detached from the body. The external plate of the frontal bone was

removed and no deformity was observed either in the external plate or the internal plate covering the brain. The depth and conformation of the frontal sinus was normal for a calf of this age. The occipital bone was removed as well as the meninges. The depth and shape of the sulci of the cerebrum were normal. External hydrocephalus was absent. The occipital pole of the cerebral hemispheres extended further backwards than is normally encountered. A fairly large amount of C.S.F. was encountered around the cerebellum.

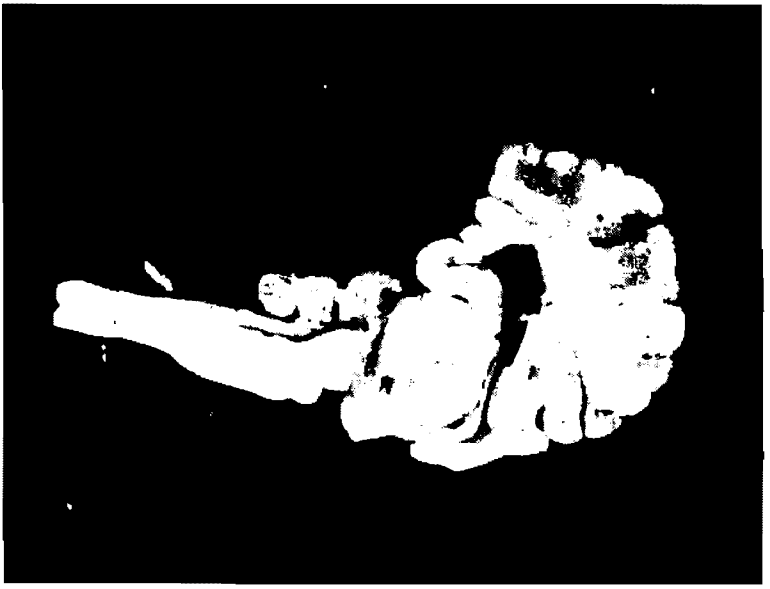
Three sections were made:—

(1) A transverse section through the cerebrum at the junction of the anterior and middle thirds. Nothing abnormal was observed.

(2) A transverse section through the cerebrum at the junction of the middle and posterior thirds. This showed the enlarged lateral ventricles and choroid plexuses.

(3) A longitudinal section through the medulla oblongata, pons, cerebellum and posterior third of the cerebrum. The much enlarged lateral ventricle filled with C.S.F. and the very small cerebellum are depicted.

The slaughter weight of the calf was 224 lb. on the hook.



Discussion

Cerebellar hypoplasia has been present since birth with hydrocephalus developing later in life as exhibited by the lack of cranial malfor-

mation. The genetic origin of cerebellar hypoplasia in this case is questionable, as the dam of this calf was locally bred and the sire was imported from the U.K. Hydrocephalus has not been encountered on this property before and the percentage of stillbirths is exceedingly low.

ACKNOWLEDGMENTS

To the Chief, Veterinary Field Services for permission to publish and Mr. J. Chandler for assistance in preparing specimens.

REFERENCES

1. RIMMELLS, R. A., MONLUX, A.: Principles of Vet. Path.
2. BODDIE, G. F.: Diagnostic Methods in Vet. Medicine.
3. DUKES, H. H.: The physiology of domestic animals.
4. FITZGERALD, T. C. (1961): Anatomy of Cerebral Ventricles of domestic animals. Vet. Medicine, Vol. 56.
5. BELLING, T. H. & HOLLAND, L. A. (1962): Variations of Internal Hydrocephalus. Vet. Medicine, Vol. 57.

CASE REPORT

GIARDIA IN THE BLOOD OF CHINCHILLA

R. SACHS — State Veterinarian, Keetmanshoop, South West Africa

CASE HISTORY

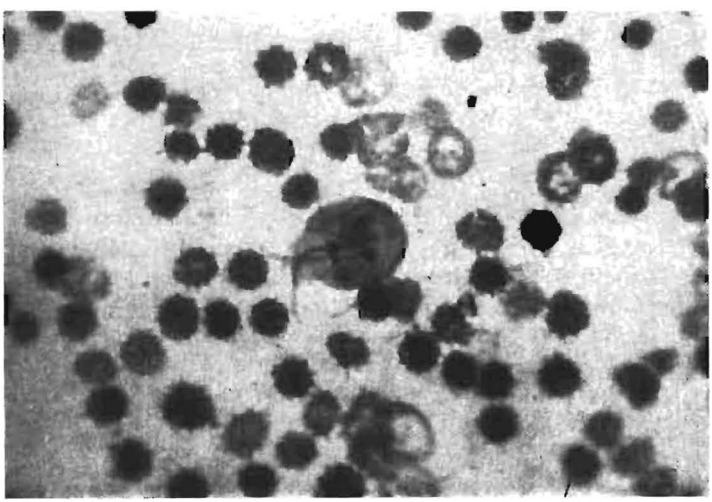
One animal out of fifty chinchillas which were imported from Canada in May, 1959, became sick a fortnight after arrival in Grootfontein, South West Africa.

Clinical Examination

The animal in question was a 6-months-old female. At the time of examination it was very emaciated, anaemic, and poor in condition. Accelerated, jerky, difficult breathing and coughing was suggestive of pneumonia. The animal was dull, depressed and lethargic. Cachexia, anorexia and constipation were observed clinically. Injections of penicillin and laxatives were administered, but the animal died three days later.

Post Mortem Examination

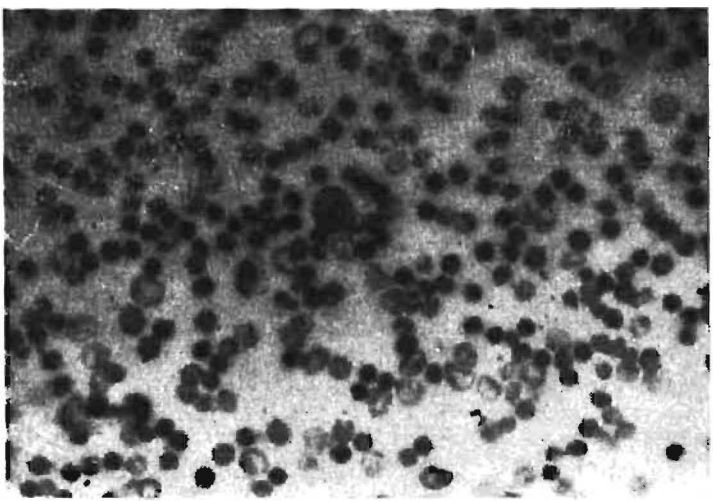
At post mortem examination, an oedema of the lungs and gastritis were the most marked pathological changes. An opaque yellowish-white focus in the liver, with an arborescent appearance, was observed, and



G'ardia sp. in the blood of Clinichilla

suggested the taking of liver-smears. These showed masses of giardia parasites. Histologically, a mild nephrosis and a focal gastritis, a few small foci of degeneration of the liver, hyperaemia and oedema of the lungs, and haemorrhages in the heart, were observed. No cholangitis was present, and no bacteria of importance could be isolated at the Onderstepoort Research Institute.

A blood smear taken from the coagulated blood in the heart of the dead animal during the post mortem showed marked anaemic changes. Anisocytosis and polychromasia were present, and after a very careful examination of the blood-smear, the vegetative flagellated form of the giardia parasites could be demonstrated, with the typical pear-shaped bilaterally symmetric form and measuring about 10 x 15 microns. The various structures of the parasite, the flagellas and the caryosomes as well as the "sucker" is clearly to be seen on the photographs, which also show the marked anaemia.



G'ardia sp. in the blood of Clinicllla
(Marked anaenic changes)

Discussion

As giardia cannot be regarded as a specific blood-parasite, the presence of the flagellated form in the circulating blood makes the case under discussion unusual, and the question arises how the parasites might have entered the blood. A post mortem invasion of the blood stream by the flagellates could have occured. In regard to the symptoms, however, it could also be suspected that an invasion of the blood might have occured intra vitam.

In chronic cases of giardia infestation of the chinchilla, several investigators described the presence of an anaemia and cachexia in association with the infection.[2, 3] Also Baumgartner states that anaemia was frequent in cases of human giardiasis, and Infurna considers giardia

to cause anaemia and diarrhoea, often alternating with constipation.[4] But there is no report of the presence of giardia in the blood, except GONDER's accidental finding of the parasite in the blood of a falcon shot in the Transvaal in 1910.[1]

In regard to the presence of giardia in the liver and in the blood, one is inclined to compare this finding with the occurence of another flagellate sporadically invading the blood stream although not being a blood-parasite, namely trichomonas. During artificial infections of *Trichomonas columbae* into mice, WAGNER and HEES found the same trichomonads in the heart blood which they found in the liver.[5] HEES further mentions the finding of specific foci in the liver of meuws on the Shetland Islands, and could demonstrate the same trichomonads in the blood of the heart. In their report on finding of trichomonads in the blood of man, the same authors observed anaemia and cachexia as clinical symptoms. In all these cases of blood trichomonads, the several investigators are not quite sure on the significance of the parasites in the blood.

Whether or not the giardia sp. is directly responsible for the death of the chinchilla is difficult to say; stress of travel may probably have played a rôle in the cause of death of the animal.

Blood smears of the whole flock of chinchillas that were imported to Grootfontein were taken for microscopical examination, and also faeces examinations were undertaken in order to establish whether apparently healthy animals were carriers of giardia parasites. The results of all these examinations, performed in 1959/60, were negative. As to the life-cycle of the giardia parasite and the possibility of transmitting the infection from one host to the other by means of faeces containing the cysts, all the pens and hutches where the diseased animal was quartered, as well as all appliances that were in or near the cage, were thoroughly disinfected and rinsed with several changes of hot water.

Since this apparently isolated case has occurred up to the time of submitting this article for publication, no further outbreak of the disease in the chinchilla flock concerned has been reported.

ACKNOWLEDGMENTS

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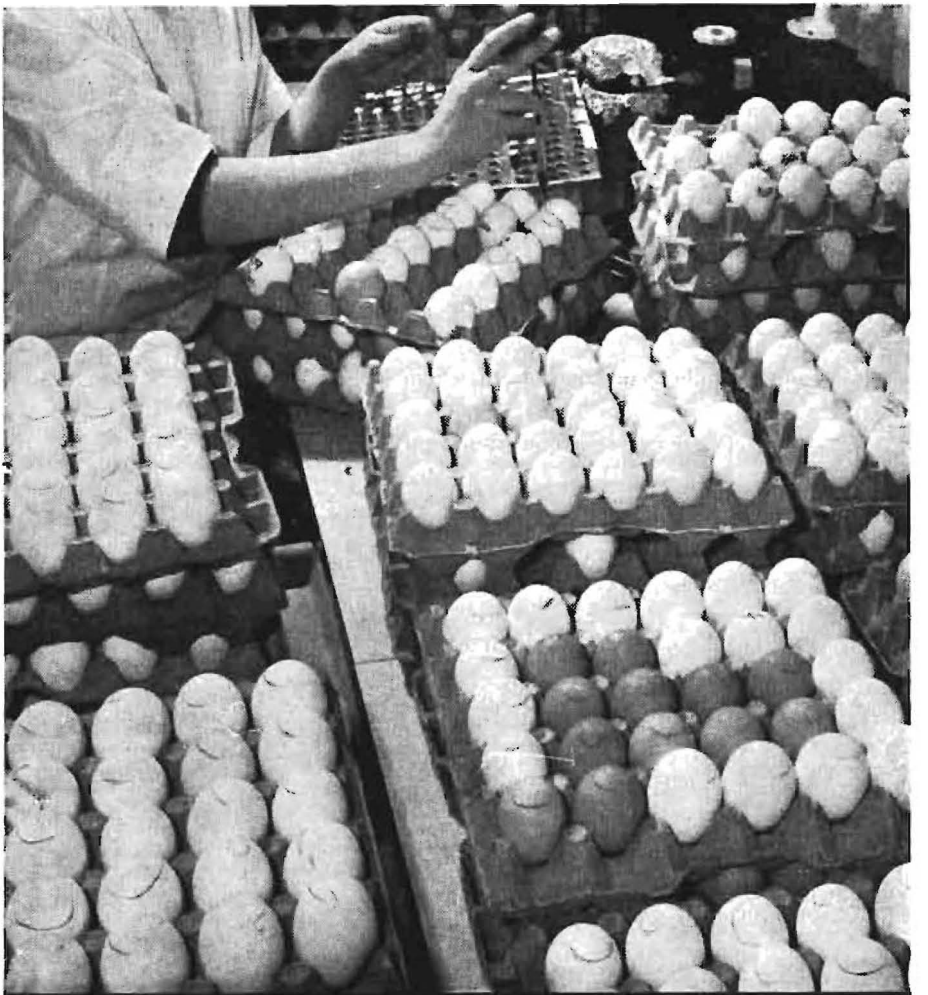
Advice given by Prof. Wetzel, University of Giessen, Germany, is gratefully acknowledged.

REFERENCES

1. GONDER, R. (1910–11): "Lamblia sanguinis n.sp. (Gonder)". *Arch. Protistenkunde*, **21**, 208–212.
2. MERCK VETERINARY MANUAL (1955): Merck & Co. Inc., Rahway, N.J., U.S.A., p. 1216.
3. MORGAN, B. B. and HAWKINS, A. (1951): "Veterinary Protozoology", *Burgess. Publish. Comp.*, Minneapolis, Minn., 2nd edit.
4. THOMPSON, J. G. and ROBERTSON, A. (1929): "Protozoology", Baillière, Tindall & Cox, London.
5. WAGNER, O. and HEES, E. (1937): "156 positive Trichomonasblutbefunde bei Mensch und Tier". Zbl. Bakt. und Infektionskrankheiten, Bd. 138.



Eggs in their thousands are used for research and testing the potency of Canilep. Many potency and safety tests are carried out before Canilep is released to do its job of providing all-in protection against the four major canine diseases.

the research behind Canilep

Glaxo research into the problems of canine protection has been intensive and productive. It resulted first in the production of separate and combined vaccines (Canilin-D, Canilin-H and Lepsolin) against the four major canine diseases and, more recently, in the introduction of Canilep. This is the first British vaccine to combine a living egg-adapted distemper virus vaccine with a killed hepatitis vaccine and a killed bacterial vaccine against the two forms of leptospirosis. Only two injections of Canilep are needed to give all-in protection against four diseases.

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An example of the tests carried out on Canilep, which go beyond the normal requirements, is that used to determine the potency of the distemper component. A blood sample is taken from an animal previously vaccinated with Canilep. This sample is prepared in a series of dilutions, and injected, with distemper virus, into fertile hens' eggs. After a period of time, the eggs are opened and the chorio allantoic membranes are examined for virus growth. In this way, it is possible to determine the dilution of serum at which growth of the virus is inhibited. This indicates the level of immunity achieved. Each examination needs many eggs and this is only one of many tests carried out by Glaxo workers to prove the safety and efficiency of Canilep.

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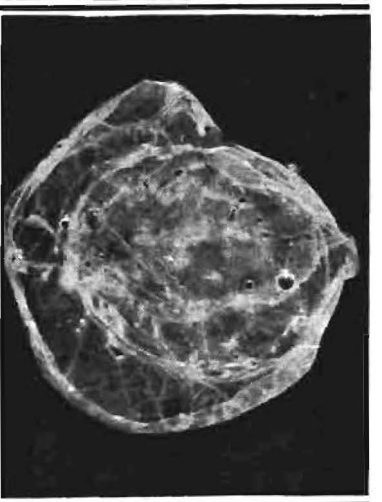
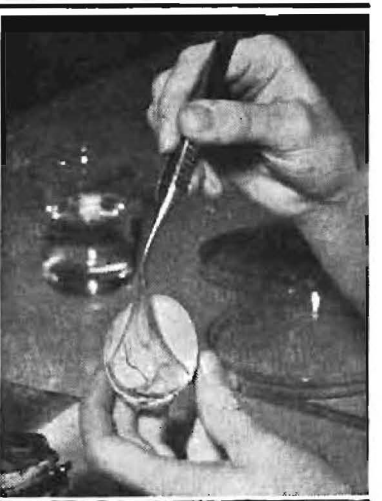
Inoculating fertile hens' eggs with distemper virus before incubation



Chorio allantoic membrane showing effect of virus growth



Harvesting of infected chorio allantoic membrane after virus growth

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IN COLLABORATION WITH GEIGY, SWITZERLAND.

OBSERVATIONS ON THE USE OF STAINLESS STEEL RUSH PINS AND STEINMAN PINS IN THE TREATMENT OF FRACTURED RADIUS AND ULNA IN THE DOG

Z. D. VINCENT — Veterinary Practitioner, 153 Main Road, Rondebosch, Cape Town

SUMMARY

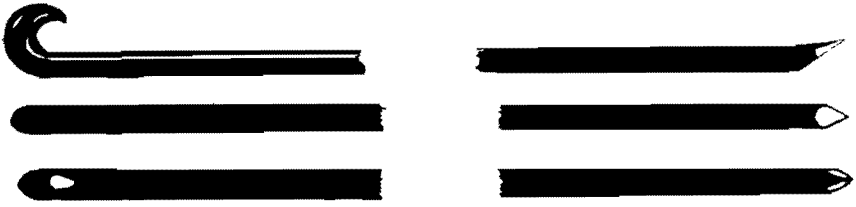
A full description of the technique of Rush pinning of the radius and Steinman pinning of the ulna, in the form of two case histories and an evaluation of the technique and results, are given. The Rush pin is described and the advantages of its use are listed.

INTRODUCTION

External fixation of a fractured radius and ulna in small animals is frequently unsatisfactory and the examination of many fractures repaired by these methods show, in general, a very poor final result. Internal fixation by plating, even in skilled hands, takes a considerable length of time and leaves a foreign body permanently embedded in the tissue, unless a second operation is performed, for its removal. The Steinman pin does not require any description as it is widely used in various cases, suitable for internal fixation. A description of the Rush pin is considered necessary.

THE RUSH PIN AND ITS ADVANTAGES

In 1946, Leslie V. Rush M.D., and H. Lowry Rush M.D., of Meridan, Mississippi, after extensive trials dating back to 1936, developed the present Rush pin.



The pin is constructed of stainless steel. It is a round highly polished rod with a hooked head and a curved sled-runner like point. The head faces in the same direction as the curved point.

Advantages:

(1) It can be introduced not only from an extremity, but also from the side of the bone.

(2) The hooked head can be buried in the soft tissues and does not produce irritation.

(3) The head serves to guide the pin during its insertion as it is turned in the same plane as the gliding surface of the runner.

(4) When pinning is necessary in young growing animals, extraction is made possible at a later date, since the pin is prevented from migrating into the bone by the curved head.

(5) Its insertion does less damage to the bone compared to plating and in case of infection, its withdrawal is far more simple than the removal of plates and screws.

(6) The sled-runner point is so constructed that, when the pin is driven into the bone, it guides the pin down the medulla.

(7) The shaft of the pin can be bent as required.

Two Case Histories

Case 1

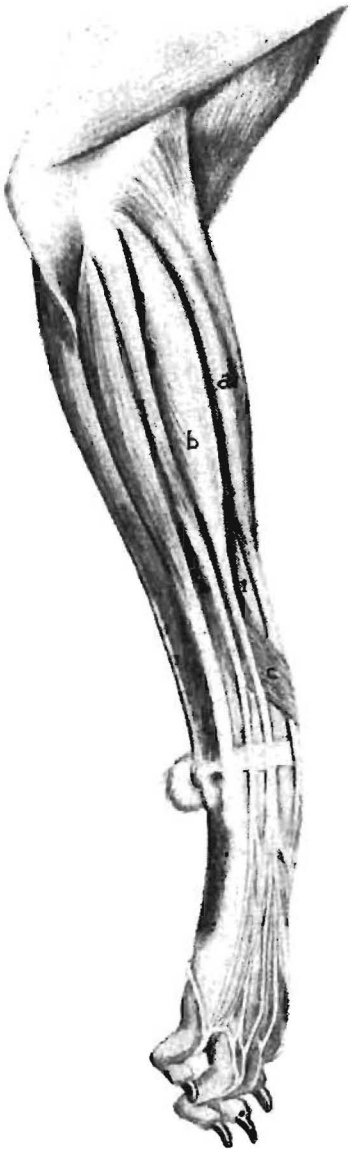
An Alsatian about 11 months old, weighing approximately 65 lb., was presented for examination with a mid-shaft fracture of the radius and a fracture of the distal end of the ulna. An initial X-ray revealed a case suitable for Rush-pinning.

Technique

Penicillin and streptomycin were given for a few days, pre-operatively. The limb was shaved and the skin disinfected by routine methods. A hobble made of bandage was looped over the lower part of the limb in order to apply traction during the operation.

Pre-medication, with morphia and atropine, was followed by pentobarbitone sodium intravenously to induce deep general anaesthesia. A tourniquet was applied and the limb dressed in stockinette. A small skin incision was made parallel to the bone on the lateral aspect of the limb, one quarter of an inch posterior to the cephalic vein. The aponeurosis was dissected from the muscles and the extensor carpi radialis was separated from the common digital extensor, exposing the shaft of the radius. (See Anatomy sketch). The fracture site was examined, all the debris cleaned from between the ends of the bone and reduction achieved by traction and angulation.

A Rush pin was chosen long enough and of sufficient diameter to fit the medullary cavity. The site of insertion chosen was such that the ends of the pin would finally be equidistant from the site of fracture. A twist drill of corresponding diameter to the Rush pin was selected. Drilling was started at an angle of 90° to the bone. Once the outer cortex had been penetrated, the drill was rocked away from the fracture converting the opening into an oblique one. The pin was driven into the proximal fragment so that the end reached the fracture line. The bone was realigned and held in the correct position with bone forceps and the pin



Muscles of Antibrachium and Manus of Dog.

Lateral View

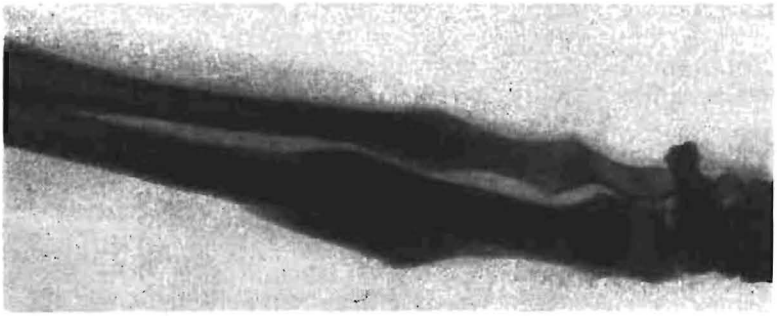
a) Extensor carpi radialis
b) Common digital extensor
c) Extensor carpi radialis (s.abductor pollicis longus)

1) Radius.

By kind permission from Sisson and Grossman

driven home into the distal fragment. The repaired fracture was tested for firm fixation. Penicillin was applied locally and the aponeurosis sutured firmly. Using gut, all dead space was eliminated. Final X-Rays were taken in both planes. The skin was sutured with nylon, the leg well padded with cotton wool, bandaged, and adhesive tape was applied to minimise swelling. A Cramer splint was used as additional support for ten days. Penicillin and streptomycin were given daily for three days,

and the leg redressed three days post-operatively. The stitches were removed eight days after the operation. The dog was discharged from the hospital on the 10th day and weight bearing noticed on the 16th day. An X-Ray, *Fig. No.* 1, taken 3 weeks after the operation revealed good callus formation.



Final X-Rays. Figs. No. 2 and 3 taken five weeks after admission, showed excellent union and the pin was then removed under Pentothal anaesthesia.

Fig. No. 2.
Anterio-posterial.

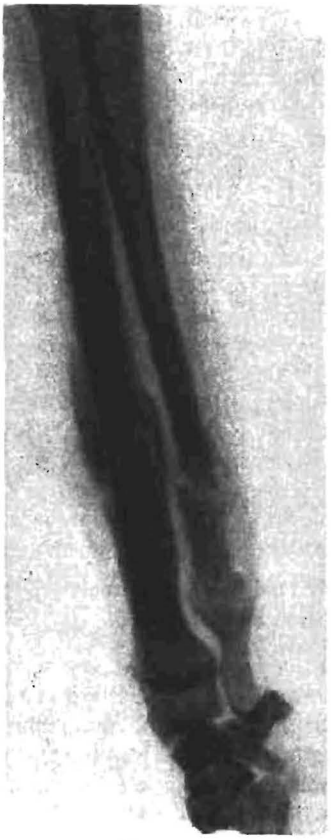


Fig. No. 3.
Lateral.

Case No. 2

A Ridgeback approximately 8 months old and weighing 70 lb. was presented with a mid-shaft fracture of both the radius and ulna. From studies of X-Rays it appeared that once reduction and fixation of the radius was obtained and callus formation had started, a fractured ulna followed suit and healed satisfactorily. Bearing this in mind, an attempt was made to use a reduced and immobilised ulna as a splint for a fractured radius.

Technique

The preparation and anaesthesia was similar to that described in Case No. 1. The dog was operated upon soon after the accident. The ulna and the fracture were palpated through the skin and an incision made over the posterior aspect of the bone, long enough to obtain an adequate view. The area was thoroughly cleaned to allow as much accuracy as possible in the reduction of the fragments by traction and angulation. A suitable Steinman pin was chosen and inserted into the medullary cavity of the proximal fragment and drilled right through the olecranon and skin. The cavity in the distal fragment was prepared with a similar sized Steinman pin. The chuck was reversed, the fracture reduced, radius and ulna adjusted as accurately as possible and the pin inserted far down into the distal fragment of the ulna. The fixation was tested for security, the skin stitched in the usual manner and check X-Rays were taken in both planes. No external support in the form of a Cramer splint was applied, but the leg was bandaged and elastoplast used to minimise the swelling. Penicillin and streptomycin were given for five days. Stitches were removed eight days post-operatively and good healing was observed. The dog was discharged and re-examined five weeks after the operation. An X-Ray, Fig. 4, showed good callus formation and the pin was removed under general anaesthesia.

DISCUSSION AND EVALUATION OF THE TECHNIQUE AND ITS RECONCILIATION WITH THE STATISTICS

The experience gained from the application of the Rush pin in twenty-seven cases of radial fracture in the dog, suggests that this method can be used in all breeds and in nearly every type of fracture. The best results, however, are obtained in large breeds and in mid shaft fractures, providing that the pin is made of stainless steel hard and springy enough to resist the weight of the animal and the muscle-pull. The Steinman pin was used on two occasions in large breeds and the final results proved outstandingly successful. The insertion of the Rush pin will be found difficult, unless the pin is adequately bent and the hole in the bone prepared with proper care. As the head of the inserted pin can be felt subcutaneously, its withdrawal requires only a small incision. If the pin is embedded firmly it may be left in situ without ill effect.

The drilling of a Steinman pin through the compound bone of the olecranon-ulna may prove difficult to accomplish, unless a guide pin with

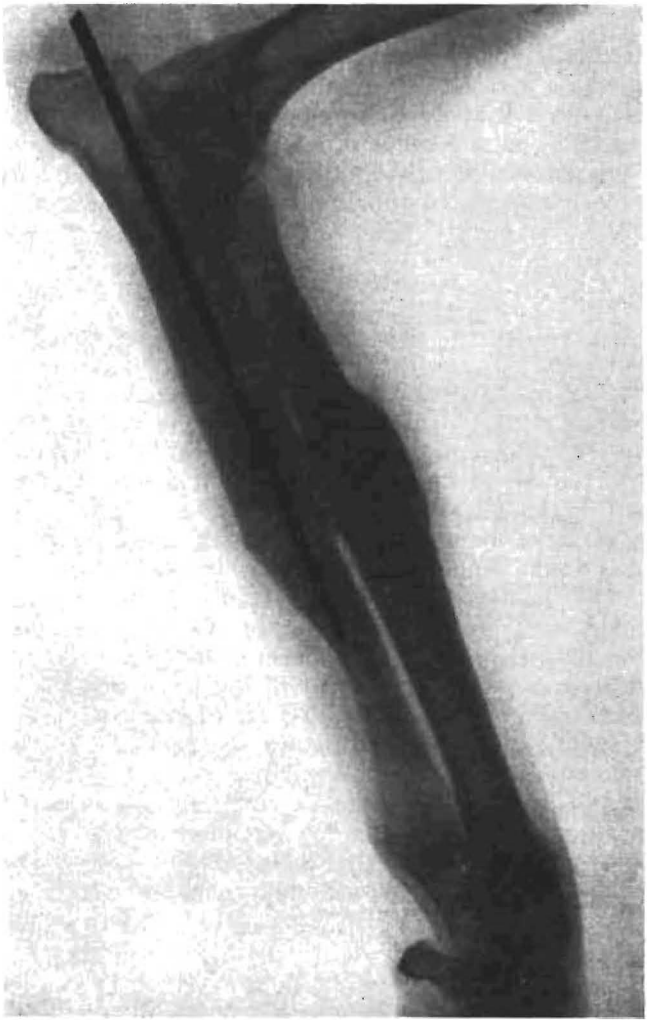


Fig. No. 4.
Lateral View.

a drill on its end is used. The method is suitable only in selected cases with a large ulnar medullary cavity. As statistics show that the fractured radius represents 8–9 per cent of all fractures admitted for treatment, it was felt that this report might stimulate the use of the Rush pin in more cases by others and therefore more data to its advantages could be collected for the benefit of canine surgery.

ACKNOWLEDGMENT

The writer wishes very gratefully to acknowledge the help of his colleague Dr. D. Venter, who spent a lot of his time preparing the anatomical sketch and X-Ray film prints.

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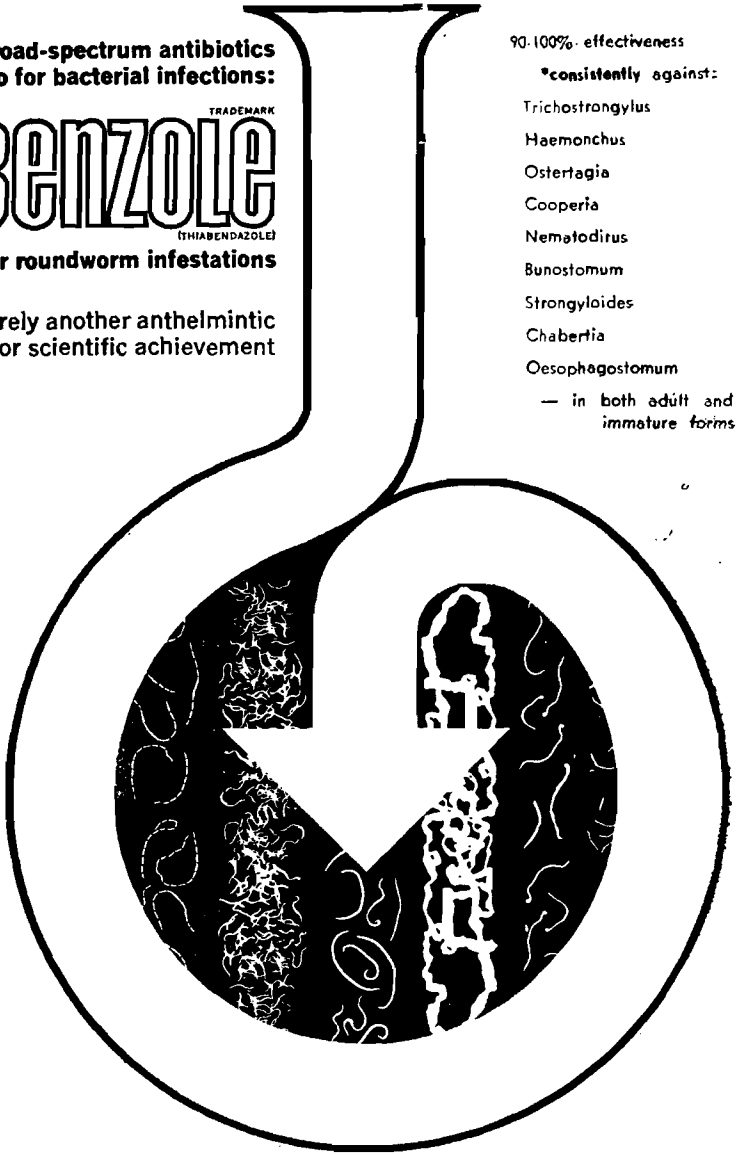

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FIXATION AFTER REDUCTION OF LUXATION OF THE COXOFEMORAL JOINT IN DOGS

L. L. HANSMEYER — Veterinary Practitioner, P.O. Box 918, Springs, Transvaal

INTRODUCTION

Hind leg lameness in the dog, arising from pathological conditions of the hip joint, congenital or acquired, provides a constantly recurring clinical problem.

In this short paper a simple but effective method of fixation of the hip joint after reduction of a luxation, caused primarily by trauma, is described. It is designed specifically for the luxation which is still reducible, but which recurs almost immediately if the limb is moved, with pressure being applied along the axis of the femur. It has not been attempted in cases of congenital hip abnormalities, nor in fractures of the head or neck of the femur, or other pathological conditions, as there are more effective methods of dealing with these conditions.

Some thirty four cases were treated in this way over a period of four years, with uniformly good and gratifying results.

OPERATIVE TECHNIQUE

The operation is performed under general anaesthetic with the patient in a lateral recumbent position, after ascertaining that the luxation has been reduced, and by applying continuous traction. With appropriate preparation, a skin incision is made, commencing at a point one inch proximally and exactly in line with the *trochanter major*, and extending distally three inches, excatly in line with the centre of the long axis of the femur. The skin is loosened from the subcutaneous tissue both anteriorly and posteriorly from the incision, for a distance of two inches, and proximally for a distance of three-quarters of an inch.

Using No. 4 chromic gut and a large cutting edge needle, the point of the needle is inserted into the muscle mass three eights of an inch anteriorly to the femur, about one inch distal to the *trochanter major*, taking a deep bite and directing the needle proximally, to let it merge one quarter of an inch dorsal to the *trochanter major* parallel to the axis of the femur. The needle is reinserted deeply into the muscle, about one quarter of an inch from the point of emergence, but this time the needle is directed posteriorly so that it emerges about three eights of an inch posteriorly and slightly above the *trochanter major*. The needle is again reinserted into the muscle about one quarter of an inch from the point of emergence, this time directing it distally, three eights of an inch away from the femur and parallel to its long axis, to emerge finally at a point

in line with the original insertion. Care must be taken when the last stitch is inserted to avoid the sciatic nerve. The two loose ends are now tied together and the purse string suture (that is what it is, in actual fact) drawn as tightly as possible. It will be found that the muscles enclosed in the suture are hard and firm, and will not yield upon digital pressure. The skin is closed in the usual way.

In the author's experience the dog is using the leg in 48 hours, and within a week, recovery is complete. Of the thirty-four cases treated in this way, not one has relapsed.

Remarks

An interesting feature of some anterodorsal luxations of the hip joint was found in two cases of the number mentioned above. After the cases were X-rayed and no fracture was found, reduction was attempted but with no attendant success. Open reduction was then resorted to and when the joint capsule was exposed, the reason for the great difficulty in attempted reduction became clear. In both cases, the head of the synovial layer of the joint capsule had ruptured but the fibrous layer had remained intact. The synovial layer had then contracted around the neck of the femur; the anterodorsal portion of the synovial layer was drawn into the acetabulum. Once the joint capsule was freed from the head and stitched again, reduction was found to be quite easy. Both these dogs were then treated by the method described above, and both made uneventful recoveries.

EQUINE VIRAL RHINOPNEUMONITIS

("Equine abortion"; "Equine influenza")

B. J. ERASMUS — Section of Virology, Veterinary Research Institute, Onderstepoort

SUMMARY

A review of the most important features of infection by equine rhinopneumonitis virus is given and the practical importance of the disease is stressed. Details are given of specimens to be submitted for diagnosis.

INTRODUCTION

Infections of the upper respiratory tract and abortions constitute two problems which are of major importance in equine practice. These problems may be of particular economic significance in the breeding and rearing of valuable horses, especially Thoroughbreds. Unfortunately, the etiological agents of these conditions are manifold and are often widely divergent.

A virus, designated as equine rhinopneumonitis virus (previously called equine abortion virus), has been isolated from aborted foetuses, and subsequently also from the nasal passages of horses showing symptoms of mild respiratory infections in the United States of America. This virus has been shown to be one of the most important causes of respiratory disease and abortions among horses.

The presence of this disease in South Africa was suspected by Henning[1] and its existence has subsequently been confirmed by means of the demonstration of complement-fixing and virus-neutralizing antibodies in the sera of horses as well as by virus isolation.[2] It has therefore been thought advisable to present a review of the most salient features of the disease in order to enable veterinarians to recognize it and to submit the correct specimens for confirmation of the diagnosis.

HISTORICAL

Dimock and Edwards,[3, 4] in examining large numbers of aborted equine foetuses in the United States, first focussed attention on the possible rôle of a virus as the etiological agent. They based their supposition on factors such as the absence of cultivable micro-organisms in the majority of foetuses examined, the clinical condition of the mares before and after abortion and the promptness with which the majority of them returned to normal breeding.

Subsequent studies[5, 6, 7, 8, 9] produced substantial evidence of the viral etiology of the disease and it was shown in the United States that equine rhinopneumonitis virus constitutes the most important cause of abortion.[8]

Henning[1] also incriminated a filterable virus as the causal agent in outbreaks of equine abortion in South Africa. The pathological changes of the aborted foetuses, however, were not identical to those described by other investigators. He succeeded in transmitting the disease by means of filtered foetal material but unfortunately the virus, which is no longer available for study, was not compared serologically with the American strains of virus.

Jones *et al.*[10] succeeded in isolating a virus which they called "equine influenza virus" from horses suffering from upper respiratory infections. Doll and his co-workers, however, subsequently proved that the so-called "equine influenza virus" was identical to equine abortion virus.[11, 12, 13, 14, 15] They therefore suggested that equine viral abortion and equine influenza be considered as different manifestations resulting from infection by the same virus. In view of the fact that this particular virus was not related to the true influenza viruses of man and swine[16] and that other viruses were also capable of causing abortion in pregnant mares,[17] Doll suggested the name "equine rhinopneumonitis virus" in favour of the abortion — influenza terminology, previously used.

Etiology

It has been shown that the agent of equine viral rhinopneumonitis is filterable.[18] The size of the virus particles as determined by electron microscopy varies between 100 and 200 mu.[19, 20]

The virus is most stable at pH 6.0 to 7.0. It is readily inactivated by a temperature of 56°C. but has been shown to remain infective for as long as 35 to 42 days if dried on horsehair kept at room temperature.[18] It is not affected by exposure to penicillin or streptomycin.[21]

The virus is present in nasal exudates and in the blood during the febrile period and in the tissues and body fluids of aborted foetuses.[22] Different immunological types of rhinopneumonitis virus have not been isolated.

Although the virus was previously transmitted experimentally to susceptible horses by various methods,[10] Anderson and Goodpasture[23] were the first to propagate the virus artificially. They observed intranuclear inclusion bodies in the liver cells of hamsters inoculated intraperitoneally with foetal material. This observation was repeated and extended by Doll and his co-workers.[13, 14, 24]

Subsequently, the virus has been adapted successfully to chicken embryos,[15, 25, 26] to the mouse by intracerebral inoculation[27] and to various tissue culture cell types, the most important of which are equine,[28] ovine and porcine kidney cells.[29]

Transmission

Transmission of the disease is by direct contact of susceptible animals with infected horses or with aborted foetuses and foetal membranes, as

well as by indirect contact with infected stables, bedding and grooming utensils. The spread of the disease is facilitated by large concentrations of horses in stables, in show rings, on race tracks or at sales.[22, 30]

Outbreaks of viral rhinopneumonitis can frequently be traced to the introduction of a new horse which suffered from a subclinical or an overt form of the disease. There is however, a very strong suspicion that healthy carrier animals are largely responsible for the initiation of the epizootics in young horses.[22]

The annual appearance of the disease in weanlings is of practical importance since animals infected early in life will possess a certain degree of immunity at the time they become pregnant. Furthermore, the disease in young horses serves as a source of infection for older horses and pregnant mares.[30] An abortion storm may result if susceptible mares become infected when they are more than five months pregnant.

No evidence is available to incriminate venereal transmission as a possible means of disseminating the infection and the stallion apparently plays no rôle in the epizootiology of the disease.[8]

Experimentally, infection is possible by the intranasal, intratracheal or intravenous routes.[10, 31] Abortion in pregnant mares can be brought about by intravenous or intranasal inoculation as well as by the feeding of infected foetal material.[8] The most successful method of producing abortions, however, is by direct intra-uterine or intra-foetal inoculation of the virus.[21]

Symptoms

Naturally, the disease occurs during autumn and winter; generally coinciding with the time of weaning.[30] The incubation period may vary from 2 to 10 days.[10]

As its name indicates, the virus of equine rhinopneumonitis primarily affects the respiratory system. Affected animals show congestion of the nasal mucosa, serous rhinitis, and a mild conjunctivitis. A mild febrile reaction may be present for 2 to 5 days; the maximum temperature ranging from 102–105°F. The appetite usually remains unimpaired. Slight enlargement of the mandibular lymph nodes may occur. In uncomplicated cases, recovery takes place within one week.[10, 17]

It has been demonstrated by Doll *et al.*[12] that horses infected with equine rhinopneumonitis virus develop a leucopenia, which coincides with the febrile reaction and which results mainly from depression of neutrophils. This finding may be useful in diagnosing the disease.

A large proportion of infections, particularly in older horses, are subclinical and can only be detected serologically. As a rule the weanlings and yearlings suffer more from the disease than older horses. This apparent difference in susceptibility can be ascribed to the fact that older horses invariably have had previous contact with the disease and possess a certain degree of immunity.[30]

Although the primary virus disease is usually characterised by mild symptoms, the affected mucous membranes of the respiratory tract are more susceptible to secondary bacterial infections. Complications

generally consist of mucopurulent rhinitis, pharyngitis and laryngitis accompanied by coughing.[10, 17, 22] Staphylococci and beta haemolytic streptococci are frequently involved, and if the animal is left untreated, these organisms may produce serious secondary complications.

The most important sequel of infection by rhinopneumonitis virus is abortion in pregnant mares. During the course of the infection of the dam (mostly subclinically), there is a transient viraemia, sufficient to infect the foetus in utero (foetal rhinopneumonitis).[32] This finally results in death of the foetus with expulsion by the mare. When infected late during the gestation period a live foal may be born, but such foals are usually very weak, show respiratory distress and die within three days.

It has been observed that mares only abort after the fifth month of pregnancy. About 90 per cent of rhinopneumonitis virus abortions occur between the eighth and eleventh months, with the highest incidence during the ninth and tenth months of pregnancy.[30]

It is characteristic that, except for the expulsion of the foetus, the mare is apparently not visibly affected either before or after the abortion. The afterbirth comes off readily and as a rule the future breeding efficiency of the mare is not affected.[5]

The incubation period between the time of infection and abortion in mares may vary from about three weeks to three months.[32] The highest incidence of abortion occurs during late winter to early summer that is from June to November and usually follows within one to three months of an epizootic of rhinopneumonitis among the younger horses.[30]

Lesions

Gross pathology

Mortality in horses as a result of an uncomplicated infection with rhinopneumonitis virus has not been described in the literature. In horses killed during the febrile period of the disease[10] the gross lesions observed were oedema, congestion and petechial haemorrhages in the nasal mucosa and lymph nodes of the head and mediastinum as well as small areas of consolidation in the apical lobes of the lungs.[6, 9, 10, 17, 22, 33, 34, 35]

In foetal rhinopneumonitis, death of the foetus seldom occurs prior to the onset of abortion with the result that the aborted foetus is frequently enclosed in intact membranes and the tissues have a fresh appearance.[6, 17, 22] The amnion may be thickened, oedematous and contain numerous petechial haemorrhages. Yellow staining of the foetal hooves and the amniotic fluid occurs frequently. In typical cases numerous petechial haemorrhages are present in the mucous membranes of the conjunctivae, the nasal passages, oral cavity, pharynx, larynx, guttural pouches, trachea, oesophagus, stomach and intestines. Haemorrhages are also invariably present beneath all the serous membranes especially the heart, spleen and thymus. The lungs are affected most consistently and show severe congestion and interstitial and subpleural oedema.

The presence of an excessive amount of clear or slightly turbid fluid, resembling serum, in the pleural cavity is a very characteristic

finding. Ascites and hydropericard are frequently present but are as a rule less conspicuous than the hydrothorax.

Focal hepatic necrosis is a very common lesion. This appears as small grey or white subcapsular foci, frequently slightly depressed below the liver surface and ranging in size up to 5 mm. in diameter.

Other characteristic findings are the icteric appearance of the abdominal organs, oedema of the mediastinal, sublumbar, inguinal and pectoral tissues and of the thoracic and abdominal lymph nodes.

Histopathology

Histological examination of aborted foetuses confirms the macroscopic presence of widespread haemorrhages and oedema. The most significant histopathological lesions are present in the lungs, liver and spleen.

In the lungs, subpleural and interstitial oedema, petechial haemorrhages and fibrinous exudate in the bronchi and alveolar spaces are encountered. Necrosis of the cells of the alveolar and bronchial epithelium and the presence of acidophilic intranuclear inclusion bodies in these cells can be demonstrated.

Liver sections show congestion and intralobular foci of necrosis with varying degrees of karyorrhexis and pyknosis. Acidophilic intranuclear inclusions may be present in hepatic cells adjacent to the necrotic foci, in reticulo-endothelial cells, in endothelial cells lining hepatic blood vessels and in epithelial cells of the bile duct.

In the spleen, necrosis of lymphocytes is evident in the Malpighian bodies and intranuclear inclusion bodies may be present in the reticulo-endothelial cells. Similar inclusions may also be present in these cells in the thymus, lymph nodes and Peyer's patches.

Diagnosis

A presumptive diagnosis may be made from the clinical symptoms when a large number of horses are affected. The diagnosis in individual cases is more difficult as a result of the mild nature of the disease. Affected horses are seldom noticed unless bacterial complications, which tend to cause a more copious mucopurulent nasal discharge, are present.[10, 22] The presence of a leucopenia may facilitate the diagnosis in early cases.

The diagnosis can be confirmed by virus isolation from nasal swabs.[22] Swabs should be taken during the early, febrile stage of the disease and should reach the laboratory as soon as possible, preferably packed in ice.

Although serological examination of acute and convalescent phase sera by means of the complement-fixation and virus-neutralisation tests may be of diagnostic value,[36] these tests are usually more important in determining the presence and distribution of the disease in a country.

A diagnosis of foetal rhinopneumonitis can in most cases be made on gross lesions present in aborted foetuses. The fresh appearance of the foetal tissues and membranes is important in distinguishing the disease from prenatal bacterial infections. The diagnosis can be confirmed by

virus isolation and by histological examination of foetal organs.[22] Portions of liver, lung and spleen in a sterile container, should be submitted to the laboratory for biological tests. These specimens should preferably be packed in ice, but as an alternative they may be submitted in 50 per cent glycerine-saline. Organ specimens in 10 per cent formalin should also be included for histological examination.

Immunity and Immunization

It has been shown conclusively that the immunity following natural infection with equine rhinopneumonitis virus is of short duration.[22, 30, 37, 38] Although virus-neutralizing antibodies may persist, the respiratory mucosa may be re-infected within 3 to 5 months. Such re-infections are generally asymptomatic or of a very mild nature.

Immunity against abortion is probably more durable but still incomplete and variable. As a rule, a mare which has aborted once as a result of infection with this virus, produces a healthy foal the next season, but abortions due to reinfection or challenge have been shown to occur within 6 to 12 months.

The annual occurrence of the disease among weanlings serve as an important source of reinfection of yearlings and older horses. Although such reinfections are generally subclinical, they are nevertheless sufficient in providing an immunological stimulus, which may be of particular significance in preventing a serious incidence of respiratory infection or abortion by the virus.

Various types of inactivated virus vaccines have been used in the United States, but the results were invariably disappointing. A fairly effective method of immunization, advocated by Doll,[38] consists of planned infection of all horses on a farm, regardless of age or sex, with live virus slightly modified by passage in hamsters. The animals are inoculated intranasally during summer, immediately following the foaling season and the inoculation is repeated about three months later, that is before the natural occurrence of the disease among young horses. The inoculation should be given annually and it is important that all horses on a farm be immunized so as to create a uniformly immune equine population. It should be noted, however, that the hamsterpropagated virus retains its capacity of causing abortion in heavily pregnant mares and is transmissable to horses in close contact. Numerous factors will therefore have to be taken into consideration before instituting such a programme of infection — immunization.

Treatment

In the absence of any specific therapeutic agent, treatment should be aimed at good nursing and the prevention of serious bacterial complications. Slight exercise may be given, but great care should be taken against over exertion. Following recovery, the horse should not be returned to heavy work too soon, especially when pneumonic complications have been present.

The parenteral injection of broad-spectrum antibiotics may be useful in combating bacterial infections.

Mares that have aborted, generally need no treatment and may be bred the next season. In case of complications, corrective treatment should be instituted.

Prevention

As transmission of rhinopneumonitis virus infection takes place by contact, strict precautions should be taken against the introduction of infected or possibly-infected horses into a susceptible stud, particularly when most of the mares are in an advanced stage of pregnancy. If introductions have to be made, great care should be taken in preventing any direct or indirect contact with brood mares. The same applies to horses returning from training, races or shows.

Once abortions are occurring, it may be too late to alter the course of the disease since the mares might have become infected three weeks to four months previously. Nevertheless, the spread of the infection to other susceptible animals should be prevented by hygienic measures. Great care should be taken in the thorough disposal of all foetal material and bedding, and utensils as well as the inside of the stable should be disinfected.

With regard to the disease in young animals, particular care should be taken to prevent contact with susceptible blood mares. There is no point in trying to limit the spread of infection to barren mares and young horses, since infection at this stage will stimulate active immunity.

ACKNOWLEDGMENT

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REFERENCES

1. HENNING, M. W. (1946): On the etiology of epizootic or infectious equine abortion. Onderstepoort *J. Vet. Sci. Anim. Ind.*, **21**, 17–40.
2. ERASMUS, B. J. (1962): Unpublished data.
3. DIMOCK, W. W. and EDWARDS, P. R. (1932): Infections of foetuses and foals. *Kentucky Agr. Exp. Sta. Bull.*, 333.
4. DIMOCK, W. W. and EDWARDS, P. R. (1933): Is there a filterable virus of abortion in mares? *Kentucky Agr. Exp. St. Suppl. Bull.*, 333.
5. DIMOCK, W. W. and EDWARDS, P. R. (1936): The differential diagnosis of equine abortion with special reference to a hitherto underscribed form of epizootic abortion of mares. *Cornell Vet.*, **26**, 231–240.
6. DIMOCK, W. W. (1940): The diagnosis of virus abortion in mares. *Jour. Am. Vet. Med. Assoc.*, **96**, 665–666.

7. DIMOCK, W. W., EDWARDS, P. R. and BRUNER, D. W. (1942): Equine virus abortion. *Kentucky Agr. Exp. Sta. Bull.*, 426.
8. DIMOCK, W. W., EDWARDS, P. R. and BRUNER, D. W. (1947): Infections of foetuses and foals. *Kentucky Agr. Exp. Sta. Bull.*, 509.
9. WESTERFIELD, C. and DIMOCK, W. W. (1946): The pathology of equine virus abortion. *Jour. Am. Vet. Med. Assoc.*, **109**, 101–111.
10. JONES, T. C., GLEISER, C. A., MAURER, F. D., HALE, M. W. and ROBY, T. O. (1948): Transmission and immunization studies on equine influenza. *Am. J. Vet. Res.*, **9**, 243–253.
11. DOLL, E. R. and KINTNER, J. H. (1954): A comparative study of the equine abortion and equine influenza viruses. *Cornell Vet.*, **44**, 355–367.
12. DOLL, E. R., WALLACE, M. E. and RICHARDS, M. G. (1954): Thermal, hematological, and serological responses of weanling horses following inoculation with equine abortion virus: Its similarity to equine influenza. *Cornell Vet.*, **44**, 181–190.
13. DOLL, E. R., RICHARDS, M. G. and WALLACE, M. E. (1953): Adaptation of the equine abortion virus to suckling Syrian hamsters. *Cornell Vet.*, **43**, 551–558.
14. DOLL, E. R., RICHARDS, M. G. and WALLACE, M. E. (1954): Cultivation of the equine influenza virus in suckling Syrian hamsters. Its similarity to the equine abortion virus. *Cornell Vet.*, **44**, 133–138.
15. DOLL, E. R. and WALLACE, M. E. (1954): Cultivation of the equine abortion and equine influenza viruses on the chorioallantoic membrane of chicken embryos. *Cornell Vet.*, **44**, 453–461.
16. DOLL, E. R. McCOLLUM, W. H., BRYANS, J. T. and CROWE, E. W. (1956): Serological differentiation of the equine abortion virus from the human and swine influenza, mumps, and Newcastle disease viruses. *Am. J. Vet. Res.*, **17**, 262–266.
17. DOLL, E. R., BRYANS, J. T., McCOLLUM, W. H. and CROWE, M. E. W. (1957): Isolation of a filterable agent causing arteritis of horses and abortion by mares. Its differentiation from the equine abortion virus. *Cornell Vet.*, **47**, 3–41.
18. DOLL, E. R., McCOLLUM, W. H., BRYANS, J. T. and CROWE, M. E. W. (1959): Effect of physical and chemical environment on the viability of equine rhinopneumonitis virus propagated in hamsters. *Cornell Vet.*, **49**, 75–81.
19. TAJIMA, M., SHIMIZU, T. and ISHIZAKI, R. (1961): Electron microscopy of equine abortion virus. *Am. J. Vet. Res.*, **22**, 250–265.
20. SHARP, D. G. and BRACKEN, E. C. (1960): Quantitation and morphology of equine abortion virus in hamsters. *Virology*, **10**, 419–431.
21. DOLL, E. R. (1953): Intrauterine and intrafetal inoculations with equine abortion virus in pregnant mares. *Cornell Vet.*, **43**, 112–121.
22. DOLL, E. R. (1963): Viral rhinopneumonitis. In: Equine Medicine and Surgery: American Veterinary Publications, Inc., Illinois.
23. ANDERSON, K. and GOODPASTURE, E. W. (1942): Infection of newborn Syrian hamsters with the virus of mare abortion. *Am. Jour. Path.*, **18**, 555–561.
24. DOLL, E. R., BRYANS, J. T., McCOLLUM, W. H. and CROWE, E. W., (1956): Propagation of equine abortion virus in Syrian hamsters. *Cornell Vet.*, **46**, 68–82.
25. RANDALL, C. C. (1955): Propagation of equine abortion virus in the chick embryo. *Proc. Soc. Expt. Biol. and Med.*, **90**, 176–178.
26. DOLL, E. R., McCOLLUM, W. H., BRYANS, J. T. and CROWE, E. W., (1956): Propagation of equine abortion virus in the embryonated chicken egg. *Cornell Vet.*, **46**, 97–108.
27. KASCHULA, V. R., BEAUDETTE, F. R. and BYRNE, R. J. (1957): The adaptation of equine influenza virus to infant mice by the intracerebral route. *Cornell Vet.*, **47**, 137–143.
28. SHIMIZU, T., ISHIZAKI, R., ISHII, S., KAWAKAMI, Y., KAJI, T., SUGIMURA, K. and MATUMOTO, M. (1959): Isolation of equine abortion virus from natural cases of equine abortion in horse kidney cell culture. *Japan. J. Exp. Med.*, **29**, 643–649.
29. McCOLLUM, W. H., DOLL, E. R., WILSON, J. C. and JOHNSON, C. B. (1962): Isolation and propagation of equine rhinopneumonitis virus in primary monolayer kidney cell cultures of domestic animals. *Cornell Vet.*, **52**, 164–173.
30. DOLL, E. R. and BRYANS, J. T. (1963): Epizootiology of equine viral rhinopneumonitis. *Jour. Am. Vet. Med. Assoc.*, **142**, 31–37.
31. SHIMIZU, T., KAWAKAMI, Y., KAJI, T., ISHIZAKI, R., SUGIMURA, K. and MATUMOTO, M. (1961): Experimental infection of colts with equine rhinopneumonitis virus. *Nat. Inst. Anim. Hlth. Quart.*, **1**, 119–125.

32. DOLL, E. R. and BRYANS, J. T. (1962): Incubation periods for abortion in equine viral rhinopneumonitis. *Jour. Am. Vet. Med. Assoc.*, **141**, 351–354.
33. JONES, T. C. and MAURER, F. D. (1943): The pathology of equine influenza. *Am. J. Vet. Res.*, **4**, 15–31.
34. KAWAKAMI, Y., KAJI, T., SUGIMURA, K., ISHITANI, R., SHIMIZU, T. and MATUMOTO, M. (1959): Histopathological study of aborted fetuses naturally infected with equine abortion virus with some epidemiological findings. *Japan. J. Exp. Med.*, **29**, 635–641.
35. CORNER, A. H., MITCHELL, D. and MEADS, E. B. (1963): Equine virus abortion in Canada. 1. Pathological studies on aborted fetuses. *Cornell Vet.*, **53**, 78–88.
36. DOLL, E. R. and BRYANS, J. T. (1962): Development of complement-fixing and virus-neutralizing antibodies in viral rhinopneumonitis of horses. *Am. J. Vet. Res.*, **23**, 843–846.
37. DOLL, E. R., CROWE, M. E. W., BRYANS, J. T. and McCOLLUM, W. H. (1955): Infection immunity in equine virus abortion. *Cornell Vet.*, **45**, 387–410.
38. DOLL, E. R. (1961): Immunization against viral rhinopneumonitis of horses with live virus propagated in hamsters. *Jour. Am. Vet. Med. Assoc.*, **139**, 1324–1330.

NEWS FROM BRANCHES

CAPE WESTERN BRANCH

Account of General Meeting

A general meeting of the Cape Western Branch was held on Friday the 10th of May, 1963 in the council room of the Meat Control Board, at Maitland Abattoirs, and was attended by 25 members.

The meeting was opened by the chairman, Dr. A. A. L. Albertyn, at 2.10 p.m. Talks on "Domsiekte" by Dr. Fourie, "Acetonaemia in Cattle" by Dr. T. Smuts, Acetonaemia in Other animals with special reference to piglets" by Dr. S. A. R. Stephan and "Skin Conditions in Small Animals" by Dr. J. F. Brownlie were then given and stimulated a lively and prolonged discussion. After the tea interval Dr. J. K. Thomson gave a brief talk on the use of certain poisons in the control of predators and problem animals.

In the discussion on matters of general interest, Dr. Smuts raised the question of taxation of sterilised bitches. After many members had given their views it was decided that conditions in the other Provinces should be investigated through the Secretary of the parent body, and that subsequently a deputation from the branch should approach the Administrator with the object of reducing the tax in the Cape Province.

Charges for certain professional services were then discussed, and it was decided that the charge for the triple canine vaccine (Distemper-leptospirosis-hepatitis) should be reduced from R8.40 to R6.30.

The meeting was adjourned by the Chairman at 5.20 after which members attended a very enjoyable informal cocktail party.

NORTH-EASTERN TRANSVAAL BRANCH

Account of Annual General Meeting and Conference held at Nelspruit on 8 June, 1963

Present:

Dr. M. J. N. Meeser (Acting Chairman).
Dr. Susanne Solomon (Secretary).
and the following:

Members:

Drs. C. J. Coetzee, J. Schuss, C. D. Meredith, D. Dixon, P. V. A. Davies, J. W. van Niekerk, A. D. Thomas, P. P. Bosman, J. J. Oosthuizen and J. H. Schoeman.

Apologies for Absence:

Drs. R. J. Kruger, P. R. Cloete, W. Eschenburg, L. R. Hurter, S. Osborn, T. N. Osborn, V. E. Osborn, D. Smit (S.W.A. and T. B. Nel (Ermelo).

Visitors:

Dr. H. P. Steyn (President, S.A.V.M.A.)
Prof. R. Clark (Vice-President, S.A.V.M.A.)
Dr. J. M. Brown, Dr. S. V. O'Brien, Mrs. A. D. Thomas, Mr. Stegmann (K. N. P.), Dr. Pitchford, Dr. Saar (Visitor from the university of Berlin),
Dr. A. M. Diesel (Secretary, S.A.V.M.A.)

The Chairman welcomed Dr. H. P. Steyn, the President of the Association, Professor R. Clark, Dr. Brown, Dr. O'Brien, Mr. Dave Stegmann (Educational Officer, National Parks Board) and Dr. Diesel. He paid tribute to members for a very well attended meeting and assured them of an informative and enjoyable day.

He then called on Dr. Steyn to open the meeting.

Opening by the President, Dr. H. P. Steyn

The President of the Association, Dr. H. P. Steyn, gave an interesting address when opening the First Annual Meeting of the Branch and particularly referred to the future of the Association. The address is published in this issue.

The Scientific Conference

The following papers were presented to the Meeting which received them with considerable enthusiasm; and an appreciative discussion:

1. The Scope and Limitation of Chemical Pathology — J. M. Brown

Dr. Brown presented an interesting survey of the value of Chemical pathology in the detection of disease. He cited comparatively simple tests which could effectively be applied in the diagnosis of animal diseases.

His Survey covered the tests concerned with detection of trace elements, blood smear technique, detection of diseases of the blood and circulatory system; the spectroscopic examination of blood, examination for liver damage, urine examination, detection of diseases of the pacreas, thyroid, goitres, adrenals, milk fever, iron and copper deficiencies and many other tests which can be carried out at small clinical laboratories. He distributed condensed information concerning many of these tests.

Dr. Brown's paper will be published in the Journal of the S.A.V.M.A.

2. Bilharziasis — Dr. J. Pitchford

Dr. Pitchford gave an excellent account of the disease and the most recent investigations into several of its aspects, and answered a number of questions put to him.

Dr. Pitchford's paper will be published in the Journal of the S.A.V.M.A.

3. The Pathological Physiology of Verminoses — Prof. R. Clark

Professor Clark gave a most interesting review of recent information on the pathological physiology of verminoses.

The forth-coming issue of the Onderstepoort Journal of Veterinary Research will contain much of the information referred to by Prof. Clark.

4. Milk Hygiene — Dr. J. O'Brien

Dr. O'Brien's paper was divided into two parts:

(1) The Veterinary Surgeon in Public Health.
(2) Milk Hygiene.

Both papers were most informative and referred to matters of very great importance to the profession.

Dr. O'Brien has agreed to the publication of these papers in the Journal of the S.A.V.M.A.

A most interesting discussion followed on the paper on milk hygiene, particularly in regard to tuberculin testing.

The Business Meeting

Election of the Executive

The following office bearers for 1963/64 were unanimously elected:—

Chairman:	Dr. M. J. N. Meeser
Hon. Secretary	Dr. Susanne Solomon.
Committee:	Chairman, Hon. Secretary and Dr. P. V. A. Davies.

The Constitution of the Branch

The meeting unanimously adopted the constitution which had been prepared from the deliberations at the inaugural meeting which took place last year. A copy was sent to the secretary of the parent body.

Venue of the Next Meeting

The meeting appeared to favour Nelspruit as the venue for the next Annual Meeting, but was prepared to leave it to the executive committee to decide.

Thanks and appreciation to Messrs. H. L. Hall & Sons

The meeting expressed its appreciation to Messrs. Hall & Sons for the privileges and the facilities extended for the present meeting.

The Hon. Secretary was requested to convey this expression of thanks to the persons concerned.

Lunch

5. Enkele gedagtes oor die versorging en verpleging van wilde diere tydens die vang, vervoer en vrylatingsproses — J. W. van Niekerk

Dr. van Niekerk het 'n baie interessante praatjie oor die versorging van wilde diere tydens vang prosesse gelewer.

Sy praatjie word in hierdie uitgawe van die Tydskrif van die Vereniging gepubliseer.

6. Demonstration by Dr. A. D. Thomas

Dr. Thomas gave a most interesting short talk and demonstration on the poisonous plants *Pavetta harborii* and *Pachystigma pygmaeum*, the causes of gousiekte.

7. Films

Two very interesting films were shown by Mr. D. Stegmann, Educational Officer on the staff of the National Parks Board. One film depicted the heroic services rendered to the wildlife in the Kruger Park during the recent drought.

Adjournment

The Chairman thanked all who had attended the meeting and wished them a safe return to their respective homes.

A very enjoyable dinner was arranged at the Pine Lake Hotel, which rounded off a most delightful meeting.

NATAL BRANCH

Account of Annual General Meeting and Conference held at Pietermaritzburg, on 11th June, 1963

Present:

Drs. R. J. Bezuidenhout, B. Baker, I. S. Canham, D. G. Clow, J. W. Coles, J. L. Doré, F. B. du Casse, R. Every, A. Fair, J. F. Fick, W. Hobbs, H. Holtz, P. Joubert, A. J. Louw, D. J. le Roux, G. Martinaglia, C. R. Morford, D. D. McMillan, J. Nesbit, R. Nixon, J. M. O'Grady, D. Osbourne, S. W. Petrick, J. Pettifer, B. T. Paine, G. M. Shires, P. Snyman, R. A. Solomon, H. F. Strydom, A. F. Tarr, A. R. Thiel, S. G. Turner, P. P. Wacher.

Visitors:

Dr. H. P. Steyn and Dr. R. K. Loveday.

Apologies:

Drs. P. Collier, J. du Preez, V. Morford, R. Bangay, W. Barnard, L. Blomefield, A. Diesel, J. Zwarenstein and Maj. G. K. Shaw.

The Chairman, Dr. Tarr, welcomed all members and visitors and remarked how fortunate the Branch was in having Dr. Steyn present at considerable sacrifice to his other activities.

The President, Dr. H. P. Steyn Addresses the Meeting

Dr. H. P. Steyn remarked that his activities were not done out of sacrifice but out of a sense of duty. He then commenced to describe the functions of the Veterinary Board and standardisation of Veterinary Education. A legal expert would be appointed to the Board. He went on to explain the new amendment to the Veterinary Act whereby no unregistered veterinarian may practice for remuneration in the Republic. This great step forward was almost entirely due to the sympathetic attitude of the Hon. P.M.K. le Roux, Minister of Agricultural Technical Services, whom Dr. Steyn described as "an outstanding benefactor of our profession". He proposed recognition of this fact by the meeting. A big "thank you" as well to Dr. P. Vorster.

The president reported that as far as a second faculty was concerned no further advancement could be reported. However the facilities at Onderstepoort were being expanded but that a material increase in graduates would only be evident by 1965. There would be an estimated shortage of approximately 250 veterinarians at this date as the present demand for veterinarians was on the increase and there was at present rate of training no prospect of meeting these requirements. Dr. Steyn emphasized that as a responsible profession *gradually* reaching maturity, we owed a duty to the farming community. It was therefore our duty to emphasize this shortage of veterinarians. He then dwelt a while on the subject of Bantu veterinary education and mentioned that the Minister of Agricultural Technical Services did *not* think the time ripe for a second Faculty.

The reason for not establishing a second faculty was the lack of sufficiently qualified training staff available. In the meantime posts which should be filled by veterinarians may be filled by lesser qualified people. If necessary a commission of enquiry should be instituted.

The Chairman thanked the President and went on to emphasize certain remarks he had made and also agreed that a resolution be passed recognising in some manner our gratitude to Minister Le Roux. He also emphasized the Natal Branch's solid support behind Dr. Steyn.

The members present stood for a few minutes in silence out of respect for the deceased Drs. Smith and W. Hamlyn.

The Business Meeting

Matters Arising

Dr. P. Snyman proposed that the annual subscription remain at R2.00. This was seconded by Dr. Thiel.

Chairman's Address

Dr. Tarr apologised for any possible repetition. He reported that the committee of the previous year had endeavoured to stimulate interest i.e. by means of films and talks by specialists of the medical profession. He went on to describe the tremendous developmental programme this

country has planned for the next decade and emphasized that our profession however was suffering in this general advancement and expansion. Considerable progress in the Agricultural sphere, but not in the veterinary sphere, was taking place. This gave the impression of relegation. "We must endeavour to retain the initiative of our special functions and prevent them from gradually being usurped by Health and other Officials etc." he said.

The Chairman reiterated the question of a commission of enquiry raised by the President.

He closed by thanking Drs. Dorè and O'Grady for deputising as chairman and secretary respectively.

New Members

The following were elected as new members, proposed by Dr. H. Strydom and seconded by Dr. D. Osbourne:

Drs. P. Joubert, J. Fick, S. W. Petrick, A. E. Fair, B. Baker, and J. du Preez.

Resignations

Dr. W. L. Jenkins and S. Cilliers.

Election of Office Bearers

The following members were proposed and unanimously elected:

Chairman:	Dr. J. L. Dorè.
Vice-Chairman:	Dr. F. B. du Casse.
Secretary/Treasurer:	Dr. J. —. O'Grady.
Committee:	Drs. A. Tarr, W. Hobbs, A. Thiel, R. Solomon and L. Blomefield.

Financial Statement

This was read, proposed, seconded and accepted.

A discussion then followed on the increased subscription. It was decided to develop a Reserve Fund from the surplus available to provide for expenses incurred when inviting guest speakers either form Onderstepoort or elsewhere, to address our Branch meetings.

Correspondence

Read.

General

Dr. P. Snyman expressed concern at advertising in farming journals for veterinary assistants. The President Dr. Steyn explained that only advertisements through the association applied to veterinarians.

Dr. I. Canham enquired about an article in the S.A. farming journal encouraging chemists to improve their veterinary knowledge. Dr. Steyn replied that the pharmaceutical board had enquired about a recommended book on veterinary Science.

Clinical Papers and Discussions

Dr. R. K. Loveday of Onderstepoort presented a paper on "Some aspects of skin diseases" which was very well received and a lively discussion followed.

Dr. R. Nixon of Allerton delivered a paper entitled "The purpose and significance of Allerton Laboratory" which made clear that many tests previously only undertaken at Onderstepoort were now being carried out in Pietermaritzburg which was a great help to the local practitioners.

After a congenial lunch Dr. G. M. Shires of Kloof presented a paper on "Some aspects of nutrition in domestic animals from the veterinary aspect".

As this is rather an extensive subject Dr. Shires dwelt only on the more salient features of dietetics in direct association with certain deficiences and overfeeding, manifesting a symptomatology familiar to the practitioner. This paper was followed by the "Small animal forum", consisting of Drs. J. L. Dorè, L. Morford, I. Canham and M. Shires and the "Large animal forum", consisting of Drs. P. Wacher, B. du Casse and B. Paine.

These two groups dealt with subjects submitted by local practitioners. All the members joined in the lively discussions that followed and all in all it was a most informative afternoon.

RESOLUTIONS

Dr. A. Tarr proposed that in future the presidential address be made after lunch due to the fact that so many members were unable to arrive earlier. This was seconded by Dr. P. Snyman and carried.

The following resolutions were proposed by Dr. A. Tarr:

(*a*) "This branch meeting wishes to express its appreciation of the services and support rendered to the profession by the Minister of Agricultural Technical Services, the Hon. Mr. P. M. K. Le Roux and requests the Council of the S.A.V.M.A. to consider ways and means of implementing this appreciation as recognition in some tangible manner". Seconded by Dr. P. Snyman and carried.

(*b*) "That a Commission be appointed to enquire into all aspects concerning the veterinary profession in the Republic with particular reference to training and requirements of the future".

Seconded by Dr. S. G. Turner and carried.

This concluded the business and the Chairman declared the meeting closed.

The Cocktail party held at the Imperial Hotel in the evening rounded off a very successful day.

OUR ASSOCIATION

Address by the President, Dr. H. P. Steyn, when opening the First Annual General Meeting of the North-Eastern Transvaal Branch at Nelspruit, on 8 June, 1963

Mr. Chairman,

I was asked by your Secretary to address you for 30 minutes on this subject. While this may be construed as a loyal gesture to the Association by you, it probably also manifests a degree of pride in our Association by your members which is very gratifying to those people who are your elected representatives upon the Council of the Association and who unstintingly give of their time and energy in an effort to make the affairs of the Association prosper and flourish.

For one man to talk about the Association which celebrated its fiftieth anniversary some years ago however involves the risk of presumptiousness on his part. It is therefore with due humility and the necessary sense of duty towards a rapidly growing body of professional men who constitute the membership of this Association, that I address you today.

It is fitting on an occasion such as this that we remember with respect the contribution of those far seeing members of our Association who in past years voluntarily assumed responsibility for founding, organizing and running our Association and by doing so laid the sound foundation upon which we are able so easily to build today. Many of these men are no longer with us but some are still here and even today still take an active interest in our affairs.

One of the momental achievements of these men was their part in formulating and influencing the South African Parliament to promulgate the Veterinary Act of 1933. This single achievement gave to our profession and Association a status which it could not have enjoyed had this not been done. As you know we have now succeeded in having this Act amended.

The amendments to the Veterinary Act are of such significance that I believe we should dwell awhile upon the implications of these amendments. The Amended Act bestows a status upon our profession which is equal to that of the legal status of the medical profession.

In future there will be a legal adviser appointed by the Minister of Agricultural Technical services on the Veterinary Board. This man need not be a veterinarian and he will enjoy full status as a Board member. He will enjoy full voting rights and will therefore serve in more than just an advisory capacity.

This is an innovation which was considered necessary because practically all the decisions of the Board have legal implications and we veterinarians on the Board felt that we were venturing upon dangerous ground in endeavouring to interpret legal procedure and decisions without the presence of a legal mind to advise and guide us. The legal repre-

sentative is going to bear a heavy responsibility, but he is going to give the Board a degree of confidence in its decisions which one often felt was lacking in the past.

The Board has now been given full control over the standard of education of veterinary under-graduates, and should it at any time feel that the necessary standard of education by any veterinary faculty is not adequate it may refuse registration of the graduates of that faculty until such time as any deficiencies in the training at that faculty have been rectified to the satisfaction of the Board.

The Board may also act as an examining body for the purpose of ascertaining the standard of education of veterinarians. It may appoint observers at university examinations in South Africa for the purpose of determining what the standard of veterinary education is.

Undergraduates will have to register with the Veterinary Board in future so that professional discipline will commence the moment a student enters any veterinary faculty.

Provision has been made for the registration of auxiliary veterinary personnel.

And last but not least, no one who is not a registered veterinarian may practice veterinary science for gain.

Before these amendments to the Veterinary Act were presented to Parliament we heard a variety of arguments about the opposition which would be encountered. The outstanding feature about all these arguments was that they all come from our own colleagues. We were told that we did'nt have a hope of getting the amendment prohibiting veterinary practice for gain by non-veterinarians accepted. I have read the parliamentary debates on these amendments and there was no opposition to any of the amendments for which we had asked. The only material alteration to the amendments proposed by us was that the period of provisional registration for foreigners was increased from five to seven years.

And here I wish to pay tribute to a man who has proved himself to be a real friend to the profession viz. the Hon. Mr. P. M. K. le Roux, Minister for Agricultural Technical Services. This man has accepted and recognized the value of the veterinary profession to the stock farming industry of the Republic. When I interviewed him on your behalf about 18 months ago he asked me how long the Veterinary Act had been in operation and when last it had been amended. I replied 1933 and no amendments since. His reply was: "It is time it was amended, and if you can have your proposed amendments in my hands by May, 1962, I will see them through the following Session of Parliament". So gesê so gedaan! Although the words quoted are obviously not verbatim, they nevertheless accurately convey the spirit and the trend of the conversation.

You, I am sure, will agree with me that Mr. le Roux has rendered our profession an outstanding and quite exceptional service and I would like to see this service acknowledged in a tangible manner. Your Vice-President, Prof. Clark, made a suggestion to me last year about this and my feeling then was that at that time any such acknowledgement would have been premature. Now I feel the time is ripe and I would

ask Prof. Clark to go ahead with his proposal at our next Council Meeting.

Before dealing with some of the activities of our Association I would like to say something about the meaning and purpose of the Association.

In order to be able to appreciate the need for a professional association such as ours, one should clearly understand what the primary objects of our profession are. I would say without any hesitation that our most important object is to render service to the community. This service includes rendering stock farming more efficient, more economic and less hazardous; it includes the protection of the human population from diseases of animal origin, it contributes to the domestic happiness of the family by rendering veterinary care to pets and finally it contributes largely to the recreation of mankind by the care rendered to animals used in sport.

All the work involved in rendering these varied services requires not only special training but continual consultation, stimulation and study. For best results it requires an interchange of views and an intercommunication among individuals who can meet and discuss their problems on common ground. Our, and other like associations were therefore established primarily to enable its members to maintain contact, exchange views and experiences and to disseminate knowledge. The most important function of the Association therefore is educational. For this purpose the Association publishes its own Journal, it has its own scientific library from which all members may borrow books, it organizes annual scientific meetings and branch meetings etc.

The next most important function of the Association is to protect the interests of its members, to seek avenues of employment and to present the profession to the general public, the community and to administrative and legislative bodies in such a way that the value of the services rendered is properly appreciated; and to endeavour to see that adequate remuneration is offered for the services rendered.

The proper performance of all the functions briefly outlined here necessitates adequate contact with world organizations and with national organizations at home and in other countries. Contact is maintained by representation at congresses in various countries or by the exchange of scientific journals.

All this requires a very considerable administrative organization and your Association provides this.

The Association has another very important function to fulfil. It is the establishment and maintenance of a status and dignity befitting a learned profession. The Association, however, can only maintain the standard which is set by the majority of its members themselves.

Aangesien ons hele organisasie streng demokraties is, is die toonaangewende deel van ons Vereniging die lede self. U Raad en die Veeartsraad gee alleen uitvoering aan die wil van die lede. Weliswaar moet daar 'n sekere mate van leiding wees, soos tog immers die geval in alle demokratiese instellings is, daarom kies ons lede daardie persone onder hulle eie geledere op alby Rade wie volgens hulle oordeel die beste in staat sou wees om leiding van die professie te aanvaar. Wat ek graag wil beklemtoon is dat alle disiplinêre beheer wat oor die professie uit-

geoeffen word nie iets is wat op die professie van elders afgepas is nie maar voortvloei uit die groep se eie doelbewuste begeertes en vereistes.

Die Raad van u Vereniging gee dus alleen uitvoering aan die opdrag van die lede. U Raad voer dus al die vooraf opgesomde funksies uit maar daar is een besondere plig wat reëlreg op u Raad rus en dit is om nie te skroom om daardie eise vir die professie te stel wat volgens sy mening hom toekom nie. Sodoende het ons reorganisasie van die fakulteit, verhoogde salarisse en nuwe wetgewing verkry. Sodoende hoop ons om, ten spyte van alle teëkanting, binne die afsienbare toekoms 'n tweede fakulteit te verkry.

U moet egter onthou dat enige eise wat die professie stel alleen geregverdig is as die diens wat die professie aan die gemeenskap lewer ons eise regverdig. Ons hoofdoel is juis om diens aan die mensdom te lewer, maar dit sal nie onwillekeurig voorregte aan ons as 'n groep oplewer as ons nie self daarvoor vra nie. Ons moet in elke opsig op eie voete staan, net soos enige persoon, enige familie, enige instansie of organisasie ja, en soos die Eerste Minister onlangs gesê het, enige volk, op eie voete moet staan en sy eie behoud moet verseker.

Ek beklemtoon hierdie noodsaaklike vereiste omdat die ondervinding geleer het dat veeartse as 'n groep 'n neiging het om te glo dat vergoeding vir goeie dienste gelewer, aan hulle geskenk sal word deur die goedhartigheid en goedgesindheid van andere. Laasgenoemde houding bestaan wel maar dit word aan onsself oorgelaat om te vra na wat ons meen aan ons toekom.

Persone wat ons dus bestempel as 'n vakbond omdat ons sekere regmatige eise stel, onthul 'n mate van onkunde en blindheid aan die natuurlike daaglikse toedrag van sake wat onvergeeflik is, en ek laat my nie een oomblik stuit in my strewe na 'n verbetering van status en diensvoorwaardes vir veeartse deur sulke aantuigings nie. Van ons word 'n getrouheid aan ons beroep, 'n hoë etiese gedragskode en 'n gedragslyn wat 'n professionele persoon waardig is, verwag. Hierdie hoë verwagtings is veeleisend, en verg groot persoonlike opofferings van alle individuë binne die professie, maar ons kan daarop roem dat ons in alle opsigte as 'n groep ons pligte aan die gemeenskap getrou nagekom het.

Die eise wat in die toekoms aan ons gestel gaan word gaan meer veeleisend wees as in die verlede want ons is nou feitlik by die stadium verby dat ons slegs die probleme deur siekte-eenhede veroorsaak, moet oplos. In die toekoms sal ons die ekonomiese verkwisting moet bestry wat al hoe meer indring na mate veeboerdery intensiewer word en die waarde van diere styg. Ek dink aan die sogenoemde erosie-siektes en verkwistende toestande wat die veeboer se profyte verminder en sy bestaan bemoeilik, juis omdat die toestande grotendeels onsigbaar en onaantasbaar is vir die oningeligte persoon. Die beheer van die soort van toestande asook die verweiding van meer tasbare siektes groepeer binne die bestek van voorbehoedende medisyne of veeartsenykunde en dit is 'n veel moeiliker taak as die bestryding van tasbare en sigbare siektes.

Hierdie verandering van toenadering tot ons probleme gaan aanpassing van die veeartsenykundige toenadering en opleiding genoodsaak en u Raad is al vir meer as 'n jaar bemoeid met hierdie taak. Dit word

natuurlik in oorleg met die Fakulteit gedoen, maar dit is 'n reuse onderneming waarvoor daar nie 'n dadelike oplossing voorhande te vinde is nie. Die veranderinge wat aangebring moet word is van 'n evolusionêre aard en sal met verdrag en trapsgewys moet geskied.

Voorafgaande is maar 'n baie kort opsomming van sommige van die doelstellings en funksies van u Vereniging en Raad. Dit sou miskien paslik wees as ek hierdie deel van my toespraak afsluit deur u te herinner aan ons leus, d.i. „Prestinoe vertutis memores". Vertaal beteken dit „Bewus van ons gawes". Daarby word ingehou dat ons ons verpligtings sal nakom nie ons voorregte sal misbruik nie en die vertroue wat ons geniet waardig sal wees.

Ons moet nou 'n bietjie aandag skenk aan sekere meer materiële oorwegings.

Ek het gesê dat 'n vereniging se vernaamste funksie van 'n opvoedkundige aard is. Daarom ook word o.a. ons Tydskrif kwartaalliks gedruk. Die voorbereiding van die Tydskrif is seker die grootste herhalende taak wat ons Sekretaris moet behartig. Dit veroorsaak 'n groot uitgaaf. Vir die jaar 1960–61 was daar 'n verlies van R2,043.25 en vir 1961–62 was die verlies R1,612.60. Vir 1962–63 is die syfers nog nie beskikbaar nie, ons verwagting is egter dat die verlies nog kleiner sal wees. U sien dat daar 'n vermindering aan die verlies was van R4,301.65. Dit is hoofsaaklik aan die toewyding van ons Sekretaris te danke.

Ons kan nie bekostig om jaarliks so 'n groot verlies te ly nie, maar daar word verwag dat die verlies geheel-en-al uitgewis gaan word en dat ons Tydskrif later met 'n wins uitgegee gaan word.

U Vereniging se totale bate het teen die einde van die 1962 boekjaar ongeveer R24,000 bereik. Hierdie bedrag word ingedeel onder die volgende hoofde: algemene reserwe, liefdadigheidsfonds, prys en reserwe prys fondse. Vir die jaar 1962 het ons 'n batige saldo van R644.08 getoon.

Hulp word deur u Vereniging verleen aan behoeftige lede of behoeftige families van afgestorwe lede. Hiervoor bestaan die liefdadigheidsfonds en gedurende 1961–62 is R300.00 op dié wyse aan afhanklikes geskenk.

Verder word lenings aan behoeftige en verdienstelike studente voorgeskiet, asook in enkele gevalle aan lede. Daar was teen die einde van 1962 R1,536.35 onder die hoof op lening uit.

Die liefdadigheidsfonds verdien 'n bietjie spesiale aandag. Die fonds staan nou op ongeveer R8,000. Daar word jaarliks bydrae tot die fonds gemaak uit lede se subskripsies. Gedurende 1961–62 het die bedrag R325 beloop. Verder word geskenke gereeld aan die fonds oorgemaak. In die opsig verdien die firma Maybaker en alle ander deelnemers aan die handelsuitstalling by ons kongresse spesiale melding. Die bedrag wat die instansies jaarliks tot hierdie fonds bydra beloop enigiets van R100 tot R150, en miskien nog meer. Rente wat gedurende 1962 aan die fonds gekrediteer is het R298.18 bereik. Totale bydrae's aan die fonds was dus R740.01 gedurende die jaar.

U sien dus dat die fonds steeds aangroei, maar die beroepe op die fonds groei ook aan en ek sou graag sien dat die fonds meer algemene ondersteuning van lede geniet.

U het miskien opgemerk dat die notule van Raadsvergaderings gedurende die afgelope paar jaar nie meer gepubliseer word nie. Instede daarvan word 'n voorsitter verslag jaarliks aan die Algemene Vergadering voorgelê en dié verskyn daarna in ons Tydskrif. Dit is as gevolg van 'n besluit van u Raad om drukonkoste te bespaar. Tot dusver het die verandering die algemene goedkeuring van lede geniet.

Ten slotte wil ek my kollegas in die Oos-Transvaal geluk wens met hulle ondernemingsgees as gevolg waarvan hierdie tak gestig is. Dit is vir ons op die Raad altyd bemoedigend om die ywer en geesdrif wat die soort van vooruitgang tot stand bring te ondervind. Mag julle onderneming groei en floreer.

PUBLIC RELATIONS SERVICE

Dr. T. F. Adelaar has returned from a visit to the United States and Britain. He attended a Conference on Veterinary Toxicology in New York on July 25–26, 1963; the Section of Biological and Medical Sciences and the American College of Veterinary Toxicologists. He attended the W.V.A. Congress in Hanover.

He visited colleagues working on projects similar to those in which he is engaged.

We look forward to a talk or contribution in the Journal, describing his visits overseas.

Die Veeartswysigingswet 1963 (Wet No. 49 van 1963) het al in die Staatskoerant verskyn en is nou van krag. Afskrifte is van die Staatsdrukker, Bosmanstraat, Pretoria, teen 5 sent per eksemplaar, verkrygbaar.

Dr. Leeb du Toit has been appointed Superintendent of the Eastern Cape A.I. Co-op. with Head Quarters at East London.

His marriage to Miss Stewart took place at Bloemfontein recently.

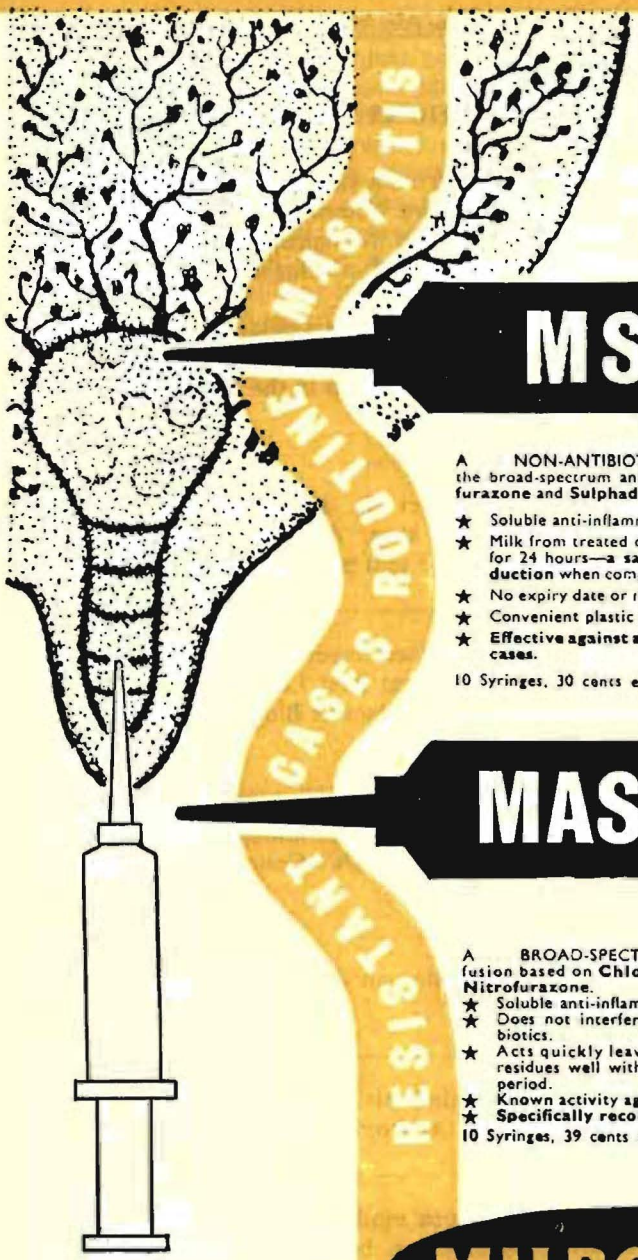
Dr. R. K. Reinecke has been on a visit to the United States to discuss anthelmintic problems. He was able to consult prominent helminthologists during his visit. He attended the W.V.A. Congress in Hanover., Germany.

Prof. Douw Steyn na sy aftreding van die Universiteit Pretoria, is nou in die diens van W.N.N.R.

Dr. R. A. Alexander terminated his temporary appointment as technical advisor at Onderstepoort, as from the end of July, 1963.

Dr. G. F. van Rensburg het teen einde Julie na Engeland vertrek waar hy verskillende navorsingstasies en betreffende smaaklikheid van voedingsoorte en tekort-siektes, sal nagaan.

Hy sal die Wêreld Veterinêre Kongres op Hanover bywoon as ook die Wêreld Produksie Kongres op Rome.



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- ★ Milk from treated quarters need only be rejected for 24 hours—**a saving of 48 hours milk production** when compared with antibiotic infusions.
- ★ No expiry date or risk of antibiotic residues.
- ★ Convenient plastic syringe.
- ★ **Effective against a large percentage of routine cases.**

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- ★ **Specifically recommended for difficult cases.**

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DIE VEEARTSRAAD GEE KENNIS
THE VETERINARY BOARD INTIMATES

GRADE OF DIPLOMAS VIR AANTEKENINGE IN DIE VEEARTSREGISTER VAN DIE REPUBLIEK VAN SUID-AFRIKA

Die volgende besluit van die Veeartsraad geneem op 'n vergadering gehou te Onderstepoort op 3/12/62 word vir algemene inligting gepubliseer:

„Die Raad besluit dat vir aantekeninge in die Veeartsregister word alleen grade of Diplomas erken wat deur Universiteite of Veeartsenyskole toegeken is en wat in wese veeartsenykundige praktisynsgrade of diplomas is. Alleen sulke grade of diplomas kan op naamborde, briefhoofde en voorskrifhoofde gebruik word terwyl enige ander graad of erkenning met vryheid kan gebruik word in applikasies en dergelike omstandighede.

Die Registrateur van Veeartse word versoek om alle grade of diplomas wat nie deur erkende Universiteite of Veeartsenyskole toegeken is en wat in wese nie veeartsenykundige praktisynsgrade of diplomas is nie, uit die Register van Veeartse te verwyder".

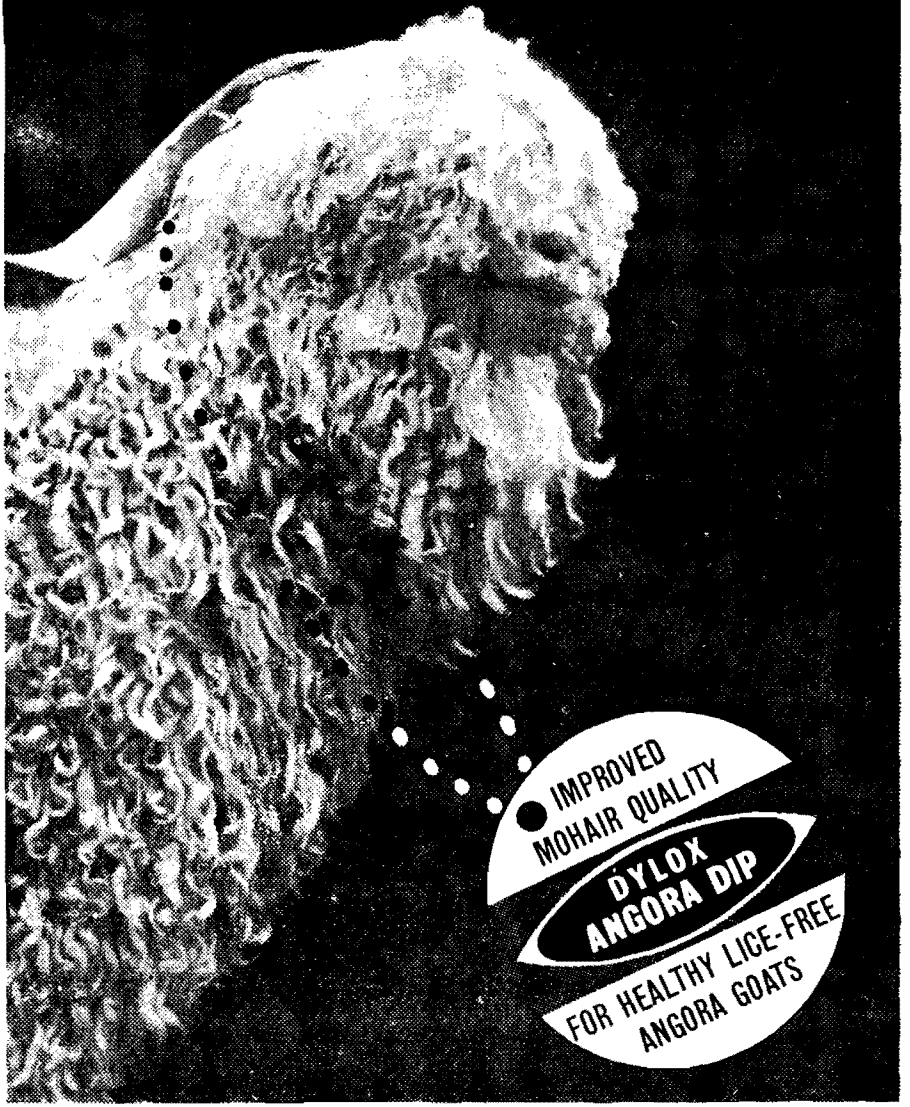
DEGREES AND DIPLOMAS RECORDED IN THE REGISTER OF VETERINARIANS IN THE REPUBLIC OF SOUTH AFRICA

The following decision of the Veterinary Board, adopted at a meeting held at Onderstepoort on 3/12/62, is published for general information:

"The Board decided that for purposes of record in the Register of Veterinarians, only degrees and diplomas would be recognised which are granted by Universities and Veterinary Schools as authority to practice.

Only these degrees and diplomas may appear on nameplates, letterheads and prescription pads, while any degree or recognition can freely be used in applications or other circumstances.

The Registrar of Veterinarians is requested to remove all degrees and diplomas from the Register not granted by Universities or Veterinary Schools which do not authorise practice."



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NEWS FROM AUSTRALIA

PLANS FOR THE NEW VETERINARY SCHOOL AT THE UNIVERSITY OF MELBOURNE, VICTORIA, AUSTRALIA

D. C. Blood — Professor of Veterinary Science, University of Melbourne

Veterinary education in Melbourne began in 1888 when a private Veterinary School was established. This was incorporated into the University of Melbourne in 1908, but closed in 1928 because of lack of students. Movement for the re-opening of the School was begun in 1960, and the first steps to bring this into effect were begun with the appointment of the first staff members in 1962.

The School will be in two situations — a Pre-Clinical Veterinary Centre associated with the University proper, and a Clinical Centre to be established in a rural area 20 miles away from the main University campus. The Pre-Clinical Centre will house the 2nd and 3rd Years of the course, and the Clinical Centre the 4th and 5th Years. The 1st Year will be done in general science departments of physics, chemistry and biology service departments within the University.

Planning for the Pre-Clinical Veterinary Centre at Parkville has been completed, and tenders for the building are now being called. It is anticipated that contracts will be let shortly, and that building will commence during June, with a probable completion date in April 1964. Planning for the Clinical Centre at Werribee has commenced on a volumetric basis, and site and detailed planning will commence about the middle of the year.

The Parkville building will be situated in the same grounds as the C.S.I.R.O. Animal Health Laboratories and the Veterinary Research Institute and will be a six-storey building, with its main frontage on Flemington Road. The building will be of off-form concrete and brick and will stand very close to the large animal accommodation which form part of the original Veterinary School. Access to this accommodation has been made available by the courtesy of C.S.I.R.O.

The building will contain administrative, teaching and research facilities and has been planned to give, as far as possible, a floor to each administrative division.

The sub-ground floor will contain storage and student accommodation, including wash-rooms, recreation rooms and common room. The ground floor will be devoted to Physiology: the first floor to Biochemistry and administrative offices: the second floor to Anatomy and Histology, the third floor to Pathology, and the fourth floor to Bacteriology. Experimental animals will be housed in a pent-house above Microbiology.

The following letter dated 30th May, 1963, has been received from Dr. R. B. Cumming of the University of New England, Armidale, New South Wales:—

"I duly represented the S.A.V.M.A. at the A.G.M. of the Australian Veterinary Association. The meeting was very enjoyable.

I do not know if I informed you that Queensland now recognises the Onderstepoort degree, after several months of negotiation. I have just submitted my name as the first O.P. graduate to be registered in Queensland.

Victoria still refuses to recognise our degree. I discussed the matter fully with Prof. Blood, Dean of the New Melbourne Veterinary School. He is in favour of having our degree recognised. I will let you know how we progress.

One thing that does stick in peoples throats over here is that Australian graduates are unable to register in the Republic. Could you please find out if Australian graduates can register in South Africa. Perhaps the question has not arisen recently but can you please let me know the position?

I am enclosing a reprint of a paper on "Uraemia" in chickens. This disease has been recognised as the major poultry disease in Australia for the past 12 years, so the isolation of this virus has created quite a deal of comment. I gave a paper on the disease at the Conference and it has been published in the April issue of the A.V.A. Journal.

Yours sincerely,

R.B.C.

(Reference to the provisions of the Veterinary Act Amendment Act 1963 (Act No. 49 of 1963) was made in the reply given to Dr. Cumming — Editor).

UDDER WASH

gives positive protection against Mastitis for less than 3 CENTS per cow per month

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- Active in hard, soft, warm or cold water.
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Wash the udder and teats of each cow with diluted I.C.I. Udder Wash shortly before milking. If they are very dirty, wash first with warm water.	Plunge the teat cups first into clean water and then into diluted I.C.I. Udder Wash after removal from each cow.	Rinse the hands thoroughly in diluted I.C.I. Udder Wash before milking each cow.

6 c.c's in two gallons of water gives a working solution sufficient for the udders and teats of at least 50 cows—at a cost of less than 4 pence per cow per month!

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BOOK REVIEWS

DISEASES OF DOMESTIC ANIMALS IN NEW ZEALAND. 1962. SECOND EDITION, REVISED PREPARED BY THE TECHNICAL COMMITTEE OF THE NEW ZEALAND VETERINARY ASSOCIATION. EDITORIAL SERVICES LIMITED, WELLINGTON. 45s.

This book contains a wealth of information in very accessible form. The text, written in a brief style and devoid of all detail, deals with the significant diseases of domestic animals as they occur in New Zealand. The contents are divided into the following sections: Bacterial diseases, viral diseases, protozoal diseases, fungal diseases, parasitic diseases, nutritional and metabolic diseases, poisons, miscellaneous conditions and general. Each disease is written in a standard form which includes such subheadings as species affected, notifiable, occurrence, incidence, aetiology, mode of infection, incubation period, age susceptibility, clinical signs, pathology, diagnostic aids, laboratory specimens, differential diagnosis, prognosis, treatment, control and prophylaxis. References are given only where recent review or other suitable papers are known. Within each chapter the 186 diseases described are dealt with in alphabetical order. This together with the brief style, standard form and extensive index make the information contained in the book readily accessible.

The Committee has admirably succeeded in producing a handbook of concise information on veterinary diseases in New Zealand; any veterinarian or veterinary student will find this book a valuable addition to his library, and what is more, will constantly refer to it. As many of the diseases which occur in New Zealand also occur in South Africa, and in other countries, this book should have a very wide appeal.

R.C.T.

THE VETERINARY ANNUAL. FOURTH ISSUE. 1962. EDITED BY W. A. POOLE. JOHN WRIGHT AND SONS LTD., BRISTOL. U.K. PRICE 45s.

This is the Fourth Issue of The Veterinary Annual to appear, and it has now become so familiar that it requires no introduction. In this issue the book is again divided into two sections, one dealing with special articles and the other, the main part of the book, is a review of the current veterinary literature.

The ten special articles each written by a different author review recent trends and developments, and the titles are as follows: Actinomycosis, Breeding at the Cross-roads, Cattle Practice in England, Foot-and-mouth Disease in the Americas, Ornithology and the Veterinary profession, Statistics in relation to Veterinary Science, Veterinary Affairs

in Africa South of the Sahara, Veterinary Education in Under-developed Countries, Veterinary Services in Inland Australia and Present Concepts in Virology.

The review of the current literature covers every aspect of veterinary science — from infectious diseases to surgery and obstetrics, and is divided into sections with the subjects arranged in alphabetical order within each section. This together with the extensive index allows for quick reference to the information desired. Each subject is reviewed by a different author and a list of the literature referred to is given at the end of each chapter.

There can be no hesitation in recommending this book to all members of the profession, practitioners as well as teachers and research workers desirous of keeping abreast with the literature. It is of especial value to those who have insufficient time to study recent developments in fields other than their own.

R.C.T.

VETERINARY PREVENTIVE MEDICINE

E. G. WHITE and F. W. JORDAN
Baillière Tindall & Cox, London. 1963. pp. 334. Illustrated.
Price 30s.

A textbook on veterinary preventive medicine embracing the full details of the subject in its widest and most practical sense, is an asset to the library of every veterinarian.

The style and lucid descriptions employed by the authors to convey information to the reader allows this book to be read and easily understood by persons not specifically trained in veterinary science.

For the veterinarian the book is comprehensive, and presents sufficient information to attract the epizootiologist, if necessary, to the more intimate literature, dealing in greater detail with the many subjects referred to in the book.

The book is divided into four parts and deals with animal disease in terms of its causal agent, be this specific, environmental, or circumstantial.

Behavioural disorders and diseases caused by non-living agents are referred to, and certain diseases caused by viral, bacterial, fungal, protozoal organisms, and by internal and external parasites, are discussed.

Reference is made to tumours in animals. The principles on which disease control is based are set out in simple language. The influence of climate, circumstance and environment in relation to the appearance of disease is intelligently referred to.

The methods employed to control disease, internal and external parasites, are presented in a lucid style.

References to the literature are quoted after each of the nineteen chapters into which the book is divided.

Occupational hazards to veterinarians and zoonoses generally are suitably dealt with in this book.

One would have liked to see a greater number of diseases dealt with, and in particular more detail given on such subjects as the control of rabies, rinderpest, bovine pleuropneumonia, babesiosis and the common diseases found in tropical and sub-tropical countries.

The chapter on Personnel and Organizations devoted to disease control is informative and adds to the usefulness of the book.

The book covers an enormous field and will be particularly useful to veterinarians employed in Field State Services.

The inclusion of a chapter on some of the tests in daily use employed to detect disease in the field and in the diagnostic laboratory, would add to the usefulness of the book.

A.M.D.

WAS GIBT ES NEUES FUER DEN PRAKTISCHEN TIERARZT, JAHRBUCH 1961062, W. A. SCHMIDT-TREPTOW, SCHLUETERSCHE BUCHDRUCKEREI-VERAGSANSTALT, HANNOVER. 644. DM 29 (R5.27)

From 1945–59 W. ALBIEN, and since 1960, W. A. SCHMIDT-TREPTOW have undertaken the laborious task of providing methodical and concise information of importance to the general practitioner. Veterinary Science covers a wider field than ever before; discovery and change are coming rapidly. The busy practitioner, in his attempt to keep abreast, has little time to peruse various and costly veterinary journals and lengthy publications. If conversant with the German language, he will find this book of almost indispensable aid to him in ihs endeavour to cope with the various problems which often crop up in his daily work.

The book is the tenth volume of the traditional series. Résumés and condensed articles by European, English, American and Russian authors are given, including two recent South African publications. Problems concerning both Human and Veterinary Medicine are focussed e.g. Tuberculosis (reinfections and nonspecific reactors), Salmonellosis and Public Health, zoonoses, mastitis, Thalodomid in therapeutics.

The book comprises ten main chapters and various subsections:

(i) Changes and matters in dispute of general medical importance.

(ii) Skin diseases and their treatment.

(iii) Surgery:
- (*a*) Therapeutic injections.
- (*b*) Narcosis, muscle relaxants, anaesthesia.
- (*c*) Wound treatment.
- (*d*) Operations, their preparation and assistance.
- (*e*) The diagnosis of traumatic gastritis.
- (*f*) Diseases of the teeth, bones, joints, tendons and hooves.
- (*g*) Eye and ear diseases.
- (*h*) Tumours.

(iv) Infertility, genital infections and A.I.

(v) Obstetrics and diseases of the puerperium.

(vi) Pathology and therapeutics:
  (*a*) The circulatory system, blood diseases, drip infusion.
  (*b*) Minerals and trace elements, vitamins and hormones in prophylaxis and therapeutics.
  (*c*) Parasites and their control.
  (*d*) Poisoning.
  (*e*) Various items of pathology and therapeutics, leucosis.
  (*f*) Infectious diseases:
    (1) Antibiotics, Sulfas, Corticosteroids and ACTH therapeutics.
    (2) Mastitis and its control.
    (3) Tetanus: various infectious diseases.
    (4) Poultry diseases.

(vii) Zoonoses.

(viii) The traditional infectious diseases and their control:
  (*a*) Tuberculosis.
  (*b*) Bovine contagious abortion, Brucelosis.
  (*c*) Foot-and-mouth disease.
  (*d*) Rabies.
  (*e*) Anthrax.
  (*f*) Swine fever.
  (*g*) Swine Erysipelas.
  (*h*) Fowl pest.

(ix) Veterinary food hygiene, the salmonella problem in relation to public health.

(x) Breeding and hygiene, rearing and management of animals, the protection of animals.

The book is written in clear style, printed on good paper, has a solid linen cover, and makes interesting reading. The index of the names of the authors and their publications permits easy orientation. The author is to be congratulated and thanked for the voluminous work.

O.H.J.M.

DIAGNOSTIC AND THERAPEUTIC TECHNIQUES IN ANIMAL REPRODUCTION

R. ZEMJANIS D.V.M., Ph.D.

Baillière, Tindall & Cox, London. 1962. pp. 238. Illustrations 56. Price not stated

In no other branch of veterinary practice is there greater divergence in the methods of approaching the problems, and of the examinations of individual animals, than in sterility work. In this book the author has succeeded in giving both student and practitioner valuable guidance on the procedure to follow in investigating infertility, both in the herd and

in the individual. The adoption of his techniques will certainly make for more uniformity; and on that account alone it can be strongly recommended.

It consists of six parts namely: 1. Examination of the cow; 2. Examination of the mare; 3. Examination of the bull; 4. Examination of the stallion; 5. Fertility examination programmes; and 6. Rectal palpations in physical diagnosis. Each of these is divided into a number of sub-sections so that the book really comprises twenty chapters.

Critical reading does reveal a few statements and omissions with which one cannot agree. For instance the assertion that vaginal examination of the cow is seldom employed routinely is contrary to practical procedure. Actually in investigating herd infertility an inspection of the vagina is imperative as it provides the first indication of the presence of a venereal infection.

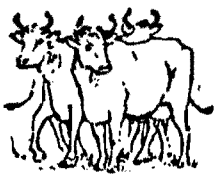
In the diagnosis of pregnancy no mention is made of the value of careful palpations of the corpus luteum in the early stages (25–35 days) which suggest possible pregnancy. Further one would have liked to see the various diagnostic tests for the infectious types of infertility described with greater clarity and more detail.

On the other hand it is most gratifying to detect a note of caution in the descriptions of the examination for retention of the corpus luteum. This is highly desirable because it is quite alarming to note how frequently a perfectly normal structure is made the scapegoat for some other more obscure aberrations and is wrongly diagnosed as a retained corpus luteum.

What is perhaps not relevant to reproduction but nevertheless of great value to the clinician is Part 6 on rectal palpations in physical diagnosis in bovines and equines. This details both the normal structures as well as all the pathological conditions which can be detected by rectal examinations.

The 56 excellent illustrations enhance the value of the book and will materially assist the student and practitioner in mastering the different techniques.

S.W.J. v. R.



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LETTERS TO THE EDITOR

Being an expression of views by contributors to promote the dissemination of information and to seek the opinion of others.

The Editor,
J.S.Afr.vet.med.Ass.

Dear Editor,

EMERGENCY PERICARDIOTOMY

On a routine visit to a farm to do herd work, the foreman asked that a particularly valuable cow be examined which he had been treating unsuccessfully for various ailments. Antibiotics, Gonacrine and various home remedies had been administered empirically.

The cow presented the typical signs of advanced pericarditis. The temperature was subnormal and there was evidence of pleuritis. The diagnosis was confirmed by the introduction of a thick bore needle between the fourth and fifth rib into the pericardial sac and the subsequent emission of an evil smelling fluid. An emergency operation was decided upon, with what instruments were available.

The cow was cast and secured on her right side, the operation site prepared, and 5 per cent Planocaine infiltrated subcutaneously and into the tissues for about 12 inches along the length of the fourth rib.

The operation was performed according to routine procedure and about five inches of the fourth rib was resected. As all the necessary instruments were not available, no attempt was made to save the periosteum. The cow lay perfectly still throughout the operation, except during introduction of the wire saw, when she twitched occasionally. This was the most difficult part of the whole procedure.

On the removal of the section of the rib, and incising the greatly distended pleura, the pericardium was found to be firmly attached to the pleura. The adhesions were firm and fixed above the whole extent of the wound.

On opening the pericardium, thin, foul-smelling fluid to the approximate volume of 2 gallons gushed out of the wound. The cow went into shock immediately. No fluid replacement was at hand, and even if it had been there was no one present to administer it, so the cow was given Methylamphetamine and Anthisan intravenously. She rallied after this, allowing the operation to go on. The incision in the pericardial sac was enlarged sufficiently to allow passage of the hand and arm beyond the elbow into the pericardial sac. The whole of the epicardium and the posterior wall of the pericardium was carefully palpated for the presence of foreign bodies, but, as is usually the case, none could be found.

The wound in the pericardium was stitched with No. 1 chromic gut, using closely approximated matress sutures. The pleura, being so closely adherent the pericardium, was stitched at the same time. An opening of

1½ inch was left in the ventral commissure of the pericardial wound. The muscles were then stitched, leaving a slightly larger opening at the ventral commissure. The skin was stitched with interrupted sutures, leaving an opening in the skin of about 2½ inch. As the pericardium was firmly attached to the pleura it was felt that there was no chance of pus escaping into the chest cavity. The opening left in the chest wall drained very satisfactorily.

The cow got up half an hour after the operation, still obviously distressed, with pus dripping out of the wound.

On the second day the patient was much improved, ears erect, eyes bright, drinking copious amounts of water and taking a small quantity of lucerne. The swelling of the dewlap was very much reduced. The breathing was fast and shallow and entirely abdominal in nature. The temperature had risen to 106 degrees Fahrenheit and it was considered that pleuritis had flared up again and pneumonia had developed.

The patient had been put onto high dosage levels of antibiotics from the day of the operation. The temperature gradually subsided until the 6th day post-operatively, when it was normal. At this stage she was feeding well and looking considerably better in all respects, the swelling of the dewlap having disappeared completely.

The cow was last seen on the 10th day, and she looked so well that at this stage the prognosis was considered to be quite favourable. There was still a marked discharge from the wound.

On the morning of the 16th post-operative day the owner phoned to say that the cow had died suddenly during the night. A post mortem examination was performed, and the cause of death was found to be purulent bilateral pneumonia and pleuritis. A very well defined tract was found connecting the reticulum and the pericardium, but the foreign body could not be traced. The operative wound had become a patent fistula providing adequate drainage form the pericardium.

It is considered that under better operating conditions and better post-operative care, preferably hospitalisation, this procedure has distinct possibilities in carefully selected cases. Traumatic pericarditis occurs commonly and the surgical interference required is not by any manner of means difficult. It is assumed that colleagues in practice have attempted this operation. It would be very interesting to find out what measure of success attended their efforts. It would be appreciated if the results obtained at the Department of Surgery, Onderstepoort, with this type of operation, were published.

L. L. HANSMEYER.
P.O. Box 918,
Springs.

The Editor,
J.S.Afr.vet.med.Ass.

Dear Editor,

EMERGENCY PERICARDIOTOMY

With regard to the aspects of the above subject raised by Dr. Hansmeyer, the following general impressions are based upon seventeen cases of traumatic pericarditis attended at Onderstepoort Clinic, Faculty of

Veterinary Science in the University of Pretoria and three cases attended in private practice before 1957.

In cattle, cardiac tamponade and traumatic pericarditis are synonymous for all practical purposes. The aetiology, pathogenesis, occurence, signs, clinical pathology and autopsy findings of the disease have been described by many authors, and are well known to all cattle practitioners. Two conditions have caused me trouble in making a diagnosis on physical examination alone. The first was myocarditis, caused by *Pachystigma pygmaeum*, a poisonous plant occurring in the Transvaal. The second was endocarditis, which at autopsy revealed a large cone shaped pus coagulum the size and shape of a large carrot, the base of which occupied much of the right atrium, and the tip reaching to the apex of the right ventricle. When doubt arises about the presence of pericarditis, it may be proved or disproved by needle puncture in the fifth intercostal space, about 16 cm. above the ventral sternal line. A 14 B.W.G. or larger bore needle should be used, and aspiration with a syringe be applied lest a viscid exudate be overlooked.

On two occasions cows have collapsed and died whilst being led to the site at which operation was to be performed. The distance was less than forty yards, and no undue excitement or other stress was added to the exertion of walking. Another cow collapsed while being led from the station to the clinic. The pericard was immediately drained with a trochar and cannula, allowing the purulent exudate to flow slowly, lest paracentetic shock ensue. She rallied soon afterwards and walked the remainder of the half mile.

The immediate aim of surgery in the treatment of pericarditis is relief of the cardiac tamponade, and simple needle puncture will avert imminent death. The second object of surgery, viz. providing adequate subsequent drainage and removal of the cause are more difficult, however, and up to date have been unattainable by me.

The evolution of various stages in an attempt to arrive at a satisfactory technique are presented in the hope that they will save others from adopting what seem to be fruitless methods, and in the hope that someone will describe a method which is more efficient.

Early attempts were along the lines classically used, viz. excision of the ventral twelve centimetres of the fifth rib. Were it not for the fact that the left fore limb cannot be drawn forward easily, and with it the triceps muscles, then the standing position with only local anaesthesia would be the approach of choice. As it is, the right lateral position appears better. In all cases operated upon, or first opened at post mortem, the pericard was attached to the parietal pleura. It would seem, therefore, that fear of pneumothorax is not warranted. Moreover, care in preserving the periosteum is superfluous, since the rib will reform without it as long since shown by Gallie and Robertson. In only one case was an offending piece of penetrating metal recovered, and when this animal eventually died from the disease, eight other penetrating objects were found, which had been overlooked at surgery. At first, the ventral commissure of the wound was left open for drainage, but simply leaving this open was soon found to be useless. The generous formation of connective tissue so characteristic of the bovine, soon widened the distance between the two stoma of the

drainage fistula, and, if the animal lived long enough, began shrinking and reducing the diameter of the canal.

Accordingly, in a subsequent case, a polythene tube about 12 cm. long and 1.5 cm. diameter was fenestrated and fimbriated, and the fimbriated end inserted into the pericardium. The tube was sewn to the skin with silk, to hold it in place. This tube was used to introduce various irrigating solutions into the pericard besides aiming to provide drainage. When this was found useless, it was imagined that the drain allowed infection to ascend. Accordingly, the end was kept plugged with a stopper between the drainage and irrigation twice daily. This also was unrewarding. The next step was to try a subsequent case with a fenestrated polythene tube, placed in such a way that the tube entered as far dorsally as practicable, and emerged at the ventral end of the pericard. The two free ends were joined together with a short perspex tube, as one repairs a plastic garden hose. The joint was opened twice daily, the exudate drained and the pericard irrigated. The tube was held up against the body with a short piece of bandage suspended from a sheet of gauze stuck to the withers with Unna's sticky paste. Since this technique, too, was inefficient, it was discarded, and a more revolutionary drainage was employed, together with the rib resection. In all previous cases, the site of drainage was not at the lowest point, and so could hardly be expected to be efficient.

In the next case a sagittal incision in the midsternal area was made. The pectoral muscles were parted, and the sternum exposed. The trephine used in equine sinusitis was employed to traverse the sternum the site being the fifth sternebrum. When the instrument had penetrated as far as possible, the column of cartilage formed was whittled away with a chisel and mallet. The trephine was then used to continue the tunnelling, until the dorsal surface of the sternum was reached. The canal in the sternum was smeared with bonewax, since it was hoped in this way to prevent infection of the sternal cartilage. The pleura and pericard in the opening were then removed, allowing drainage in the most dependent part. To guard against ascending infection a fimbriated polythene tube, equal in diameter to the trephine canal, was anchored in the canal with its internal end flush with the most ventral part of the pericard. The free end was left long, and carried to the withers as before. A column of antiseptic was left in the tube to preclude infection travelling along the tube. Although this seemed to give fairly good drainage, the exudate in the pericard became very firm and cheese-like, in fact it seemed to make a tough and inelastic investment for the myocard. This material was difficult to dislodge. Together with the fact that the penetrating object is so seldom found, this constitutes the most difficult problem to be overcome before any degree of success can be obtained.

Many irrigating solutions have been used locally. Antibacterials have included antibotics indicated by antibiogram, acriflavine Hubitane, sulfa- and acridine drugs in several vehicles. Lymphagogues and exudate solvents used included mildly hypertonic saline, proteolytic enzymes, weak sodium bicarbonate and sodium carbonate.

No accurate observations were made on the effect of irrigation with fluids at different temperatures, and containing various ions, on pulse

rate and character. The impression gained was that the fibrin and adherent inspissated pus on the myocard acted as an insulator to the epicard and prevented readily recognizable heart beat changes. By having only a ventral drainage tract, it is difficult to bring irrigating fluid in contact with all parts of the heart sac. When there are two openings, one ventral and one as high as practicable, contact of fluid over a greater surface area is achieved. Obviously, the irrigating fluid should be run in from the lower opening, otherwise only a single tract will be bathed with fluid. This brings another complicating factor into play — viz. the heart is subjected to a temporary temponade, the effect of which is circulatory embarrassment. Since the heart is already under considerable strain, this added insult may precipitate death.

Conscientious nursing and supportive therapy have been practiced throughout, but have not kept the animals alive longer than the usual two to three weeks. Immediately after surgery, and for the following three to five days, the patient appears to be doing very well, but this is a delusion, because all have deteriorated slowly and died about 21 days post-operatively. Final collapse was always sudden, with the animal sinking from a "fairly sick" condition to death overnight, or in only three or four hours.

Serial electrolyte determinations of the plasma may give a clue to a better post-operative regime. Use of a powerful X-ray machine should help materially in locating radio-opague objects.

Professor Hofmeyr has had one successful outcome in treating traumatic pericarditis. He ascribes this success to early diagnosis and the fact that he removed the offending wire. The exudate was clear, and not evil-smelling.

D. H. G. IRWIN,
Department of Surgery,
Onderstepoort.

The Editor,
J.S.Afr.vet.med.Ass.

Dear Editor,

Procaine Penicillin Allergy in the Bovine

On the morning of the 10th May, 1963 at 7.30 a.m. a Stud Breeder injected 10 c.c. of Procaine Penicillin intramuscularly into one of his Stud Friesland heifers which had calved two days previously. She had shown a very heavy post-parturient discharge and had calved normally, the placenta coming away on its own.

This subject is a twin, has a heifer calf of 25 months old, and had never received penicillin previously.

Ten minutes later she was raving foamed at the mouth, dropped down to the ground, got up and charged away madly. She was brought back to the stable and the owner contacted me at 8 a.m.

On examination at 8.45 a.m. I found her trembling and soaking wet with perspiration, much like a horse after a race. The eyelids were swollen as also the throat, anus and vulva.

The rectal temperature was 102.2 the respiration rate 28, the heart beat was fast and soft with a rate of 130.

Tracheotomy was not necessary as the worst symptoms had already subsided.

She was treated with 20 c.c. Anthisan and 350 c.c. of M.F.C. The recovery was rapid and uneventful. I wonder whether her twin sister will be sensitive to Procaine Penicillin too.

T. C. W. WESSELS,
Tweespruit, O.F.S.

The Editor,
J.S.Afr.vet.med.Ass.

Dear Editor,

BOVINE PETECHIAL FEVER

In the Veterinary Record Vol. 75 No. 15, bovine petechial fever is reported from Kenya by Danskin and Burdin. It is claimed that it has not been reported elsewhere, but I have seen this condition in the Eastern Free State for a number of years. Up to now I have never been quite certain of what I was dealing with, until I read this report. My cases were always sporadic; never more than one or two on any particular farm, and strictly seasonal i.e. from January to March-April, when snakes are most active. I always considered these cases to be those of a typical snake bite.

As has been experienced in Kenya, I am not convinced that any of my treatments have been of much use.

T. C. W. WESSELS,
Tweespruit, O.F.S.

The Editor,
J.S.Afr.vet.med.Ass.

Dear Editor,

A MODIFIED TECHNIQUE FOR PERFORMING THE PPLO PLATE TEST UNDER FIELD CONDITIONS

METHOD

Glass Tubes

A tube 10 x 75 mm. (3 ml. capacity) has been found more suitable than the previously used 8 x 100 mm. tube. Due to the larger bore, no air lock develops during bleeding and after centrifuging, the serum is more readily removed.

Solid Wooden Blocks

Wooden blocks ten inches square by one and a half inches thick are drilled to hold 100 tubes in rows of ten, maintaining them in an upright position during bleeding and after centrifuging.

Prior to this modification, the tubes were packed in canvas envelopes, each holding 10 tubes and, not only was it difficult to remove the tube for collecting the blood, but indentification of samples for retesting was difficult.

Glass Testing Plate

Plate glass six inches square by a quarter inch thick is ruled by diamond pencil into 20 squares. A few coats of white paint placed on the underside of the plate renders the glass translucent and any agglutination is easily perceptable.

A white tile is not suitable and clear glass plates have to be held over a white background to detect agglutination.

Centrifuge

One capable of a speed of 3,000 r.p.m. and fitted with a head to hold 20 tubes is ideal for the purpose. In addition, an interval timer and a looped needle for mixing the antigen with the serum are required.

Technique

0.2 ml. 20 per cent Potassium is added to each sterilized glass tube by means of an automatic syringe.

Two to 2.5 ml. blood is bled from each bird by needle puncture of the wing vein.

The blood is then centrifuged at 3,000 r.p.m. for 5 minutes and each tube is returned to its original position in the block.

One drop of serum is mixed with one drop of antigen, after which the needle loop is washed in clean water and dried.

When 10 samples have been mixed in this way, the plate is gently agitated for 2 minutes and any agglutination noted. While samples are being read a further 10 samples are mixed with antigen by an assistant.

Discussion

It is the practice at this Centre to read all PPLO tests on the farm which not only ensures that the serum is completely fresh when tested, but leads to a closer liaison between the farmer and the Veterinarian.

The writer has found that with the help of two assistants up to 1,000 PPLO tests can be performed daily.

P. M. S. MASTERS

State Veterinarian,
Veterinary Investigation Centre,
Stellenbosch.

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