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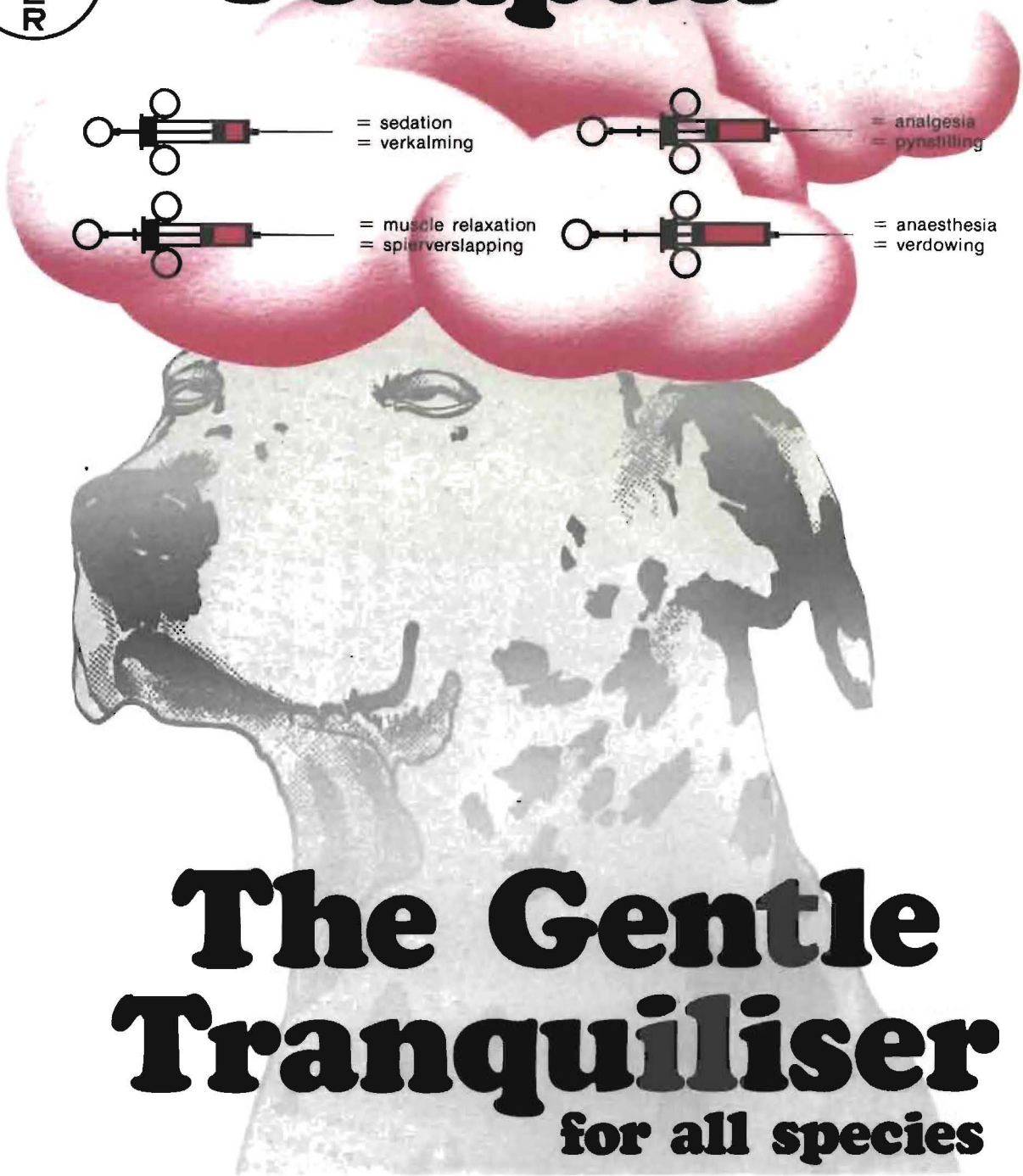

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ADDRESS **VOORDRAG**

A PHILOSOPHY OF INDUSTRIAL RESEARCH

W. H. BECKENHAUER*

Industrial research and development in a firm devoted to producing products for veterinarians is a special, small corner of the broad field, the practice of veterinary medicine. It is a challenging, often frustrating, sometimes rewarding, but always interesting occupation. Success requires the satisfaction of many facets – many masters, if you will.

The fundamental aspect of any new product is that it must be safe and efficacious for the purpose intended. A problem of definition immediately appears: what is meant by "safe"? Harmless for the recipient animal? For the environment? For the ultimate consumer, e.g. man, in the case of food animals? Obviously, all of these must be satisfied; sometimes in remarkable depth, to parts per trillion of residues, to lifetime and multiple generation studies in laboratory animals. Although every possible potential and theoretical question can never be answered on safety, the proponents of perfection in consumer protection wield a mighty influence, e.g. feline leukaemia virus.

Efficacy requires that a product perform in a beneficial manner and that its labelling and promotion accurately describe that which the product is purported to do.

Having set the foundation stones of safety and efficacy, we must now address the practical matters of convenience and ease of application. The perfect respiratory disease vaccine will be a failure if application requires 10 min aerosolisation per animal via a head bag for inhalation. Our rota-coronavirus vaccine for calves is an example of a good product that may never be widely used because of inconvenience. It is difficult and laborious to capture, restrain and dose every newborn calf and may even be downright dangerous too, in a range situation when the dam is aggressively protective. A further aspect of convenience concerns the timing of administration. In herd health management, products must fit the herd management scheme, and be applicable when the animals are accessible. If, for example, animals are on the ranges, it is most unlikely they will be gathered and handled for for any particular vaccination. The long-term career success of an industrial scientist demands that his products be both technically and commercially successful.

The cost of the new product to the end purchaser is another vital consideration. This is especially true in the case of our economic animals – the product must be economically viable. It must have a value in preventing or treating disease which exceeds its cost and its cost of administration. To be otherwise invites failure. The cost-benefit ratio is perhaps of lesser significance in companion and performance animals. The love and compassion an owner feels for a beloved pet is known to us all, and the value to the owner of keeping that pet alive and healthy often cannot be assessed in purely monetary terms. Likewise, the value of a performance animal lies not so much in its intrinsic value, but in its potential value as a winning contestant in competition, whether that competition be in racing, showing, or performance of its progency.

World-wide, products applying to the health of man and his animals are stringently regulated by governmental agencies. Unfortunately, there is little unanimity in regulation from country to country. This presents special problems in developing products suitable for international markets. An example is canine distemper vaccine produced by my firm. In the USA vaccine serials are federally approved when they are satisfactory by *in vitro* virus titration and purity. Another country may require dog vaccine and challenge tests. South Africa prefers *in ovo* titration or ferret potency testing. Unfortunately, our vaccine strain was never egg or ferret adapted, thus it cannot be quantitated in chick embryos and it is less efficacious in ferrets than in dogs. This presents a dilemma for both the producer and the regulator.

Another example derives from international shipment of animals. Japan requires that imported cattle must be serologically negative for leptospira antibodies. Thus vaccination with a leptospira bacterin is pre-empted, unless that product stimulates no antibodies or, perhaps, only temporary antibodies.

In any business, from the smallest veterinary practice to the largest industrial giant, 3 outside influences will have a major effect on the management and direction of that business. These are the customers, the suppliers and the competition. Each must be accommodated in one way or another. Competition will affect the pricing structure of the product if a commodity, or force one into design changes or a different market segment.

Suppliers can help one or kill one. A world-wide shortage of sulfonamides is not an unknown event. The major supplier may not be able to meet an increase in demand. Thus sulfamethazine of Hungarian origin is used in one formulation, while Danish sulfamethazine which has different crystal size is used in another formation. Either a production shortfall in the chemical plants or a price change will profoundly affect the initial customer, and, by the ripple effect, affect the subsequent customers of a finished product.

The customer is the final judge of a product and his choices will, to a large degree, determine one's success or failure. Acknowledging that the customer's choices may be influenced by advertising power, this alone could not override disadvantages in safety, efficacy, convenience, pricing and regulatory sanction.

*Norden Laboratories, USA.

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I will now change the tenor of this address to consider certain attitudes and committments inherent in industrial research and development.

The first requisite is the recognition that only a few ideas ever survive to become products. The expectation of a high percentage of successes, or that an initial forecast or budget will be accurate, is a certain path to disappointment. The corporate manager, presented with new product decisions, must accept these as his greatest challenge, because they are surrounded by technical and market unknowns, where success is sweetest and failure most bitter. Without the luxury of solid information, the manager must rely on his experience and common sense in making new product decisions. Certain guidelines can be helpful, and I would like to share some of these with you.

The *first* is resource allocation. If a project is to succeed, then adequate resources must be committed to make it happen. These include financial resources, enough time to do a thorough technical and marketing job, and assignment of adequate people power. Resource allocation must limit the number of projects to permit them to be done well. The path to certain failure is to provide "almost enough" resources to a project.

Second is to accept, but not be afraid of, failure. Fear of failure will lead you there, for the risks necessary to achieve success will be avoided. Nevertheless, recognition of when to call it quits can preserve valuable resources, but such decisions must be based on new information, not on fear.

Third is to recognise that research and development must produce answers as well as products. What is the probability of technical success? When will the product be ready? If we have the product, what will we do with it? If we do not have the product, what strategy will be appropriate? What will be the competitive action in either situation? Answers can be as important as products.

Fourth, recognise that an inexpensive lead from research will grow to a costly product. A rule of thumb is that one unit of cost in basic research leads to 10 in applied research, to 100 in pilot development, to 1 000 in manufacturing to 10 000 for full scale production. It is easy to see why changes in plan become more and more costly as a project advances. Recognition of exponentially increasing costs can provide the warning flag of imbalance between potential risk and potential payoff. As the product advances, facts replace opinion and forecasting improves. As an ex-president once noted: "If you can't stand the heat, don't stay in the kitchen".

Fifth, beware of leadership diffusion. Eveyone want to participate when a project looks good, and desert it if it looks bad. It is critical that authority and responsibility be clearly designated at the proper organizational level.

Sixth, beware of making decisions based on what has already been spent. Sunken costs are irretrievable. Attitudes of: "having spent so much already, we must proceed", or conversely, "we've spent so much, we can't afford to continue", are equally irrelevant. The only pertinent questions are: "Where are we?", "What will it take to succeed?"

Seventh, recognise the risks along the way and assess them qualitatively. Consider product safety and liabilities that may ensue, if actual safety is less than expected by the consumer. Consider, too, the integrity of the product; will it enhance or damage the reputation of the organisation? *Lastly,* avoid becoming so enamored of new products that the improvement and updating of old ones are neglected. Neglect of the foundation products practically guarantees that the competitors will "do you in".

In conclusion, I would reiterate that industrial research and development is a complex matter. Desired product features and requirements are not difficult to recognise, but the proper guidance of a project is less clear. Some thoughts and guidelines have been presented which can be summarised by recommending that a proper balance must be an objective and that periodic assessments of technical and marketing progress are mandatory if success is to ensue.

ADDRESS **VOORDRAG**

TRAINING VETERINARIANS FOR THE FUTURE

W.F. JACKSON*

Distinguished guests, ladies and gentlemen, it is a great privilege for me to bring greetings from AVMA to your 76th Congress. It is a singular honour for me to be invited back to your most gracious and beautiful country.

We will discuss today how we will prepare veterinarians for the future. Your faculty was started at the Transvaal University College by Sir Arnold Theiler in 1920. Since then over 1000 have qualified. In the next 11 years there will be as many to qualify as have done so in the past 57 years.

Veterinary medicine has been a dynamic, growing and changing profession for the past 60 years. Today no career is sheltered from the forces of change. Historically our profession has demonstrated its elastic nature. Some may recall the departure and reappearance of the horse. The very nature of small animal practice today reflects the dynamic forces of societal demands and a change in the lifestyles of people. If you like, we can compare the veterinary profession to the automobile industry. If there is overproduction of one model, the company changes styles to meet the market demand. The broadbased scientific training veterinarians receive is as essential to our profession as the 4 wheels are to a car. We have the ability in our curricula to make changes to reflect the market demands for new models of veterinarians. Our profession has enjoyed such a high demand for our graduates for so long that it is very difficult for us to establish a reference or equilibrium point in veterinary manpower. This fact alone makes it almost impossible to predict future demand.

Your association made a recent survey that predicted a continuing demand for veterinarians in South Africa. Our Association paid $250 000 for a manpower study. The Arthur D. Little Study predicted a surplus of 3900 veterinarians by 1985 and 8300 surplus veterinarians in 1990 based on 24 colleges. Two new schools have started since the study was made. New Jersey is the only state having over 5 million people without a veterinary college.

The ADL Study recommended that we expand post-professional education to supply the demand for specialists in industry, university teaching, society and government service. There is a well-known shortage of specialty trained pathologists, toxicologists, environmentalists and teaching specialists. It was suggested that government funds be rechanneled to correct this shortage. This is easier said than done. Our veterinary colleges have enjoyed $300–$700 per student capitation grants. This required schools to increase their enrollment annually and that the curriculum contain over 50 % content relating to food and fibre animals. Immediately the horse became a food and fibre animal since its leather is used and over 10 000 000 pounds of horsemeat are sent overseas annually for human consumption. With the gas shortage the horse is also considered an energy resource.

In the USA we have 2000 board certified specialists in twelve speciality areas. Board certificate is equal to a Ph.D. degree in industry and academia. Most of the boards require 3–5 years of special training to be eligible. The boards are under the supervision of AVMA and not the universities. There are problems but they are gradually being resolved. Specialization does a lot to advance veterinary knowledge and the use of available knowledge.

What will be the demands for veterinarians in the future? To determine the demand, we must first decide who we are and what we do. We need to define a veterinary unit of service. How many people; how many companion animals, horses, cattle, sheep and pigs are needed to support a veterinary unit? Will the government help support marginal units with regulatory and public health part-time employment? This type of support exists in many Canadian provinces. It has been estimated that $50 000–$70 000 in gross personal income is needed to support a large animal practice. It takes 400 practices to support a clinical specialist in private practice in the USA. This specialist may represent about 24 billion dollars in personal income and 2 billion dollars in disposable income.

Government regulation will have an influence on the demand for veterinary manpower. In the USA one-third of our gross national product is spent by local, state and national governments. Over 102 billion dollars is the cost of our regulatory agencies. This is about $2 000 per family. There is some rebellion against such regulation. The consumer-oriented legislatures are changing our practice boards. This may have a profound effect on veterinary practice.

Today our profession needs to address the problem of what we can do to meet the needs of the year 2000 and thereafter. We have to keep in mind that to produce a new model of veterinarian takes 2–3 years of curriculum engineering and 5 years to develop and to market a new style model. To completely change the curriculum has been very traumatic in established schools in our country. The departmental territorial rights are defended with a "catlike" tenacity that appears to threaten job security and mode of living.

The amount of new knowledge is almost overwhelming in veterinary medicine. There are 15–20 new books per month and about 800 journals that relate to veterinary medicine.

The so-called "core elective curricula" have squeezed out about 30 per cent of basic science courses. The

*President, American Veterinary Medical Association. Paper presented at the Biennial Congress of the SAVA in Johannesburg, September 1979.

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electives take up this space. Much of the core is taught by team teaching which has some inherent problems with responsibility and pride. The large number of electives total 60 % in the clinical areas and add to the workload of clinicians and to the total cost. The elective system is the most expensive method of teaching. Often the clinical staff lost their time for self development, for research and preparation of lectures. A student may have trouble with the state boards if he has been narrow in his elective selection.

Other schools offer clinical experience in the earlier years. The large class size is making it necessary for seniors to be "farmed out" to approved practices for clinical experience. The early selection of careers by some students may turn them off other course work.

Curriculum planning should involve educators, practitioners, students, industrial veterinarians and those in government service. Eighty per cent of the graduates go into private practice but their curriculum is planned by a group of which 80 % have never practiced. Is this right? The committee needs to "walk in the shoes" of the student for these years and make sure he is well prepared for the future and not bicker over the loss or addition of a course.

In my opinion there is no way a student can specialize in our course. There is no room to teach advanced nutrition, market skills, environmental controls, preventative medicine or herd health in the alloted time without eroding the broad base of scientific education a veterinarian receives.

The following should be considered:

1. The formation of a broad-based curriculum committee.
2. The use of student evaluation of courses in a meaningful manner.
3. The consultation of employers of recent graduates.
4. Rewarding educators for quality in progressive teaching.
5. The utilization of animal technicians to help teach "hands on" skills.
6. Seeking the opinion of graduates of previous five years.

In the USA most curriculum committees do not seek outside help or do not feel they want to air their problems to the public. Curriculum planning should always encourage the development of communication and instructional skills that stimulate intellectual curiosity and independent learning. The student should be prepared for a lifetime of learning. Curriculum changes are not nearly as dangerous as incompetent or apathetic students or educators.

In closing we should consider long range studies to predict demand and suggest course changes to take place gradually to prepare our new model veterinarians for the future. We must think ahead about the needs of veterinarians as they change with the shifts in the economy, changes in lifestyles, ages of our populations, feminization within the profession and the price swings in the livestock industry. Society expects services by well-trained veterinarians with a multitude of back-up services at a reasonable cost. As a profession we will be expected to maintain a high degree of self-regulation and self-improvement.

ADDRESS **VOORDRAG**

COMPETENCY AND CONTINUING EDUCATION – IS IT WORTH IT?

W. F. JACKSON*

Continuing education (CE) is not new in veterinary medicine. It appears that today it is a fashionable term for legislators and consumer advocates. Continuing education has always existed in many forms but with state relicensure laws, continuing education directors and departments in our colleges, it is often formed into a manageable package for the convenience of those involved at the administration level. Lectures and laboratories can be thus packaged in an easy manner and can be controlled, regulated and documented. These 2 forms may be the weakest forms of learning but can be enforced and sold.

When I checked my 1947 dictionary to define competency the closest word was "compensate." This reflects that the main concern in those days was to make a living by hard word. This year's medical dictionary defines competency as being capable of performing an allocated function. In psychiatry, it is defined as the ability to distinguish right from wrong. We need to invent a new word or a better definition for our current usage of the word "competence."

Most education is somewhat of a passive process with the mind retaining facts in proportion to its recognized significance and relevence to the individual. Minds often learn at different speeds and may have some preference for visual or audible sources. The young are supposed to learn better and quicker than the old but they many not have the background to sort out the most important information. As one ages, more facts slip from memory.

Continuing veterinary education is different than pre-degree education which is necessary for initial licensure. Continuing education ensures the exposure to current and valuable information that often should be incorporated in our everyday work and modify our professional behavior. We must assume that each veterinarin has the basic intelligence and the ethical desire to practice the best he can. We all learn new things, new ideas everyday and we forget a part of what science we know, for better or worse. Keep in mind that what we learned last year may not be true today.

Our programmes in veterinary continuing education should be catalysts to more efficient learning and should be sufficiently scholarly to reduce the erroneous information we have stored. Continuing education has less impact on technical skills and personal services. Most of our programmes are packaged toward gaining information and not toward systems of better practice. No one should doubt the importance of going back to the "well of knowledge" and that this trip is likely to be one of the components of veterinary competence.

No matter how loudly we shout, no one wants to realize that continuing education and recertification are totally unrelated issues for veterinarians. Continuing education must be considered as an ongoing sequence of knowledge replenishment finely tuned by practice habit and behaviour modification.

Continuing education does have a positive effect on a veterinarian's ability to make a living and on his professional ability and the welfare of his patients. We must constantly look for new ways and methods for continuing education to improve the cost effectiveness and the retention of knowledge to sort out the new, relevant and the important.

From the time we were in the first grade until we finished professional school, our teachers believed that education was not possible without a method of measuring its results. Members of our state boards, not knowing better, believe with legislators that they can test competence by written examinations. This is not true. Many have tried to test competence and to date, no valid and acceptable way of measuring competence has been discovered.

Over the years many conferences of national scope have been held to determine the effectiveness of CE. Most of the time is spent trying to define the problem and the repeated consensus is that continuing medical competence cannot be defined or measured.

Due to our long and tedious undergraduate training, we are taught by our examination system to have instant recall of all facts. Because this number of facts doubles every 6 years, it should be considered malpractice to try to recall everything. Think of the patient's well-being when uncertainty of recall forces us to refer to a book.

Continuing education should start in professional school by teaching problem solving and not fact recall. The type of student accepted into veterinary school will determine whether or not he will use acceptable knowledge and remain competent as a practitioner. One case study demonstrated that the senior medical student was no better at problem solving than as a freshman. When faculty, senior and junior were given freshman examinations, the poor scores were evenly spread over the top and bottom third in each group. Learning for retention may be as great in the "C" student as in the "A" student if these studies are correct.

In a large hospital, case reports were studied on the proper interpretation and use of data given in urinanalysis, hemoglobin and fasting blood sugar. A 35 % error was found in the physician's interpretation. A CE course was set up to correct these errors and properly carried out. Two months later the survey was repeated and the number of errors was the same. In Kansas a study over a 10 year period was made on the relationship of CE credits among pediatricians and

*President, American Veterinary Medical Association. Paper presented at the Biennial Congress of the SAVA in Johannesburg, September 1979.

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obstetricians and the number of prenatal and maternal deaths. The rate was the same between the group of physicians with the highest number of CE credits and the group with the lowest. A review of about 1500 studies revealed that of those involved or exposed to new information and ideas, 2,5 % adapted them immediately and another 13,5 % were early adapters. This left 84 % as late adapters or non-adapters. In a study of almost 5 000 case records it was concluded that of the errors made, only 6 % were made because of lack of medical knowledge.

Since competence is ill defined and cannot be measured, I wish to ask, "Who is competent enough to measure my competence or yours?" Since more and more state laws will insist that competency be measured, we will have a problem. State boards are part of our state government and are usually political appointees with no training to measure knowledge or competence in any fashion. Many state board appointees do not seek professional help in making up our current examinations and the quality of the examination will surely lead to more lawsuits regarding their ability to measure anyone's competency. We may end up with non-veterinarians giving the state board examination.

Should a veterinarina be competent in all areas since we are trained for so long to be super generalists? Are we going to have limited licensure? I predict a drastic change in our state board examination system with possibly a national board only and certification at the state level. The state legislatures will probably disband the state board licensing and will go through the same conditions of 50 years ago, only to find ourselves years in the future with the same situation. Departments of regulations will grow and the records on mandatory continuing education will be so big we will have to build new buildings to house them! After all that, it will be realized that MCE is meaningless. Competency will be measured by all sorts of novel ideas and after a decade the pendulum will swing back to saying that it cannot be measured. As practitioners of the art, we will be forced by good and bad competition to stay ahead by our own brand of CE in order to survive. The survival instinct is always the strongest when we are hungry.

Will Rogers once said, "Everyone is ignorant, only on different subjects." Improving knowledge, ability and competence is to stimulate the urge to know and remains a constructive force. The person who follows this urge is seeking to build a solid foundation of knowledge and behavioral changes that are accompanied by a critical sense of values. The motivation to learn is greatest just before or immediately after you need it. Knowledge is money in the bank but may be a time deposit.

In summary, I suggest that continuing education can increase knowledge but with little guarantee of increased quality of performance. We will have to learn to measure what we now call "competence."

INOCULATION OF PIGS WITH *STREPTOCOCCUS* SPP. ISOLATED FROM ARTHRITIC PORCINE JOINTS

G.V.S. TURNER, L. HALLAND* and M. ERASMUS†

ABSTRACT: Turner G.V.S.; Halland L.; Erasmus M. **Inoculation of pigs with *Streptococcus* spp. isolated from arthritic porcine joints.** *Journal of the South African Veterinary Association* (1980) **51** No. 1 9-13. (En) Department of Veterinary Public Health, College of Veterinary Medicine, Texas A & M University, College Station 77843, Texas, USA.

The intricacies of the serological grouping of streptococci are discussed. The pathogenicity and accurate classification of streptococci isolated from arthritic porcine joints were in doubt. Pure cultures of these isolates were inoculated intravenously into healthy pigs to ascertain their pathogenicity and in an attempt to fulfil Koch's postulates. The pathogenesis of streptococcal arthritis in swine is discussed. On intravenous inoculation into experimental pigs the streptococcal isolates showed varying degrees of pathogenicity and arthritogenesis.

INTRODUCTION

An investigation into the aetiology and pathology of porcine arthritis as found at a South African abattoir revealed that *Erysipelothrix rhusiopathiae* was present in 48 % and *Streptococcus* spp. in 20 % of the affected joints[17 18].

The classification of streptococci into the various Lancefield's groups can be complicated. The technique for the serological grouping of streptococci is complex and not yet entirely perfected. The non-groupable streptococci constitute an additional complication. In addition, a variety of Lancefield's groups have been incriminated as arthritogenic agents in swine[3 8 9 14].

The streptococci isolated in the above survey were referred to 3 independent laboratories for typing into specific Lancefield Groups. Each of the laboratories classified the streptococci differently.

The role of streptococci as arthritogenic agents in South African pigs is an unknown entity. Because the pathogenicity of the streptococcal isolates from the joints was unknown, pure cultures of these isolates were inoculated into healthy pigs to ascertain their pathogenicity and in an attempt to fulfil Koch's postulates.

MATERIALS AND METHODS

Pooled samples representative of the streptococcal isolates were inoculated into serum broth and incubated for 24 hours at 37°C. The serum broth cultures were then centrifuged at 2 000 rpm for 30 minutes. The serum broth supernatent was poured off and the bacterial sediment resuspended in phosphate buffered saline solution (PBS) (pH 6,8). PBS suspension of streptococci was prepared to correspond with Brown's Opacity Tube No. 1. This gave an estimated 6×10^5 streptococci per ml and was used merely as a guide. Aliquots of the fresh suspensions were then inoculated into the pigs. One ml from each suspension was also removed to make serial dilutions with PBS in sterile test tubes. By means of the "drop method" a drop from each of the dilutions was dropped onto a nutrient agar plate and incubated at 37°C for 24 hours. A colony count was then made and the actual number of viable organisms per ml of the streptococcal suspensions was determined.

*Division of Limnology, National Institute for Water Research, C.S.I.R., Pretoria.
†Division of Food Hygiene, Faculty of Veterinary Science, Onderstepoort, Pretoria.

Six 8 week old pigs from the same litter were used in this experiment and numbered A, B, C, D, E and F respectively.

Pigs A, B, C and D each received 2 ml of streptococcal suspension intravenously. Pigs E and F served as the controls and received 2 ml of the sterile PBS solution. All the intravenous inoculations were performed via the ear veins.

The pigs were observed daily for any signs of illness and lameness. Seven days after the streptococci had been injected into the pigs, fresh streptococcal suspensions were prepared. The procedure was repeated in a similar manner in all the pigs with the exception of pig E. The pigs were observed daily.

Pig E was slaughtered 7 days after the first injection of PBS. After the second administration of the streptococcal suspensions the pigs were slaughtered in the following order: Pig A, 7 days later; Pig B, 22 days later; and Pig C, 35 days later. The control Pig F was slaughtered 21 days after the second injection of the sterile PBS solution. Twelve days after the second injection, Pig D was given a further suspension of streptococci intravenously and slaughtered 44 days later.

In each case a thorough post mortem examination was carried out paying particular attention to any signs of metastatic abscesses in the lymph nodes and viscera. The carcass was split down the middle and the vertebral column was examined for any pathological changes.

Adopting the standard aseptic procedure for opening joints as previously described[17], the following joints were examined bilaterally in each case: hip, stifle, tarsal, shoulder, elbow and carpal joints. Aseptic samples of synovial fluid and joint capsule were taken and streaked on blood tryptose agar (BTA) and inoculated into serum broth. The culture media were incubated at 37°C and dealth with as previously described[17].

RESULTS

The results of the intravenous administration of the streptococci into the experimental animals are tabulated in Tables 1 & 2.

Pig A showed definite signs of lameness in the hindquarters 8 days after the first administration of streptococci. The lameness lasted for one day. There was no sign of any swelling of the joints and the pig did not appear ill. The pig was slaughtered 7 days after the second administration of streptococci. At post mortem examination the left hip joint showed signs of a mild subacute serous arthritis. There was a slight bulging of

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Table 1: SUMMARY: RESULTS OF INTRAVENOUS INOCULATION OF STREPTOCOCCAL ISOLATES INTO EXPERIMENTAL PIGS

Day	Pig A	Pig B	Pig C	Pig D	Pig E (Control)	Pig F (Control)
1	$1x10^9$ viable streptococci i/v	$17x10^8$ viable streptococci i/v	$11x10^8$ viable streptococci i/v	$9x10^9$ viable streptococci i/v	2 ml sterile PBS i/v	2 ml sterile PBS i/v
5	–	–	–	Definite signs of lameness	–	–
6	–	–	–	Still lame		–
7	–			Still slightly lame	Slaughtered. Joints – negative microbiology and pathology	–
8	Definite signs of lameness $2,5\times10^8$ viable streptococci i/v	10×10^8 viable streptococci i/v –	$2,4x10^8$ viable streptococci i/v –	No longer apparently lame 2×10^8 viable streptococci i/v		–
9	No longer lame	–	–	Not lame		2 ml sterile PBS i/v
12	–	Very lame – especially left front leg. Pain over left shoulder and elbow	–	–		–
13	–	Lame. Exhibited pain when touched. Carpus swollen	–	–		–
14	–	Signs of lameness decreased over next five days	–	–		–
16	Slaughtered. Streptococci isolated from left hip. Slight signs of arthritis-macroscopically and microscopically – left hip. Remainder of joints – negative microbiology and pathology	–	–	–		–
21	–	–	–	$2,6\times10^8$ viable streptococci i/v.		–
30	–	Slaughtered. Streptococci isolated from left elbow joint, which showed no pathological changes. Remainder of joints – negative microbiology and pathology	–	Slight signs of lameness in hind-quarters. Lasted intermittently for seven days		Joints – negative microbiology and pathology
43	–	–	Slaughtered. Joints – negative microbiology and pathology	–		–
65	–	–	–	Slaughtered. Joints – negative microbiology and pathology		–

Table 2: RESULTS OF THE BACTERIOLOGICAL INVESTIGATION OF JOINTS OF EXPERIMENTAL PIGS AFTER INTRAVENOUS INOCULATION OF STREPTOCOCCAL ISOLATES

Pig	Culture Media	
	Blood tryptose agar	Serum broth
A	*Streptococcus* spp.	*Streptococcus* spp.
B	*Streptococcus* spp.	*Streptococcus* spp.
C	Negative	Negative
D	Negative	Negative
E (control)	Negative	Negative
F (control)	Negative	Negative

the joint due to an increase in synovial fluid which was slightly turbid. The joint capsule was congested and slightly thickened. The articular surfaces of the affected joint showed no changes. The iliac lymph nodes draining this joint showed no gross changes. The histopathological section of the affected joint capsule showed a mild macrophage and plasma cell infiltration (Fig. 2). This was diagnosed histologically as a mild subacute synovitis which confirmed the macroscopic findings. Gram staining of the joint capsule section revealed a few isolated clumps of Gram-positive bacteria. Based on the cultural findings of this joint, these bacteria in the synovial section were regarded as being streptococci. The histological examination of the right hip joint capsule showed no pathology.

Streptococci were isolated from the left hip joint of Pig A both on the primary BTA medium and the serum broth subculture. The remainder of the joints showed no signs of arthritis and the rest of the bacteriological specimens taken from the joints mentioned were negative. The rest of the post mortem examination showed no pathological changes.

Pig B became very lame in the left foreleg 12 days after the first injection of streptococci and 4 days after the second injection. The lameness was very marked and became progressively more severe. The pig showed

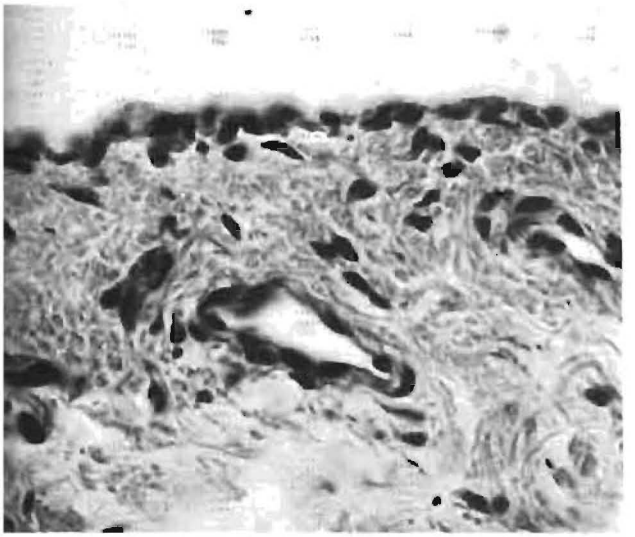

Fig. 1. Section of normal porcine joint capsule. H.E. x 600.

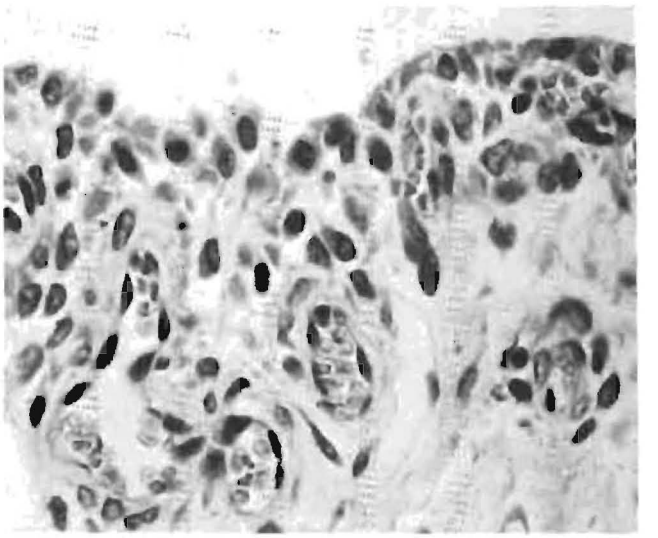

Fig. 2. Pig A – hypertrophy and hyperplasia of synovial epithelium, mild sub-synovial cellular infiltration and vascularization. H.E. x 600.

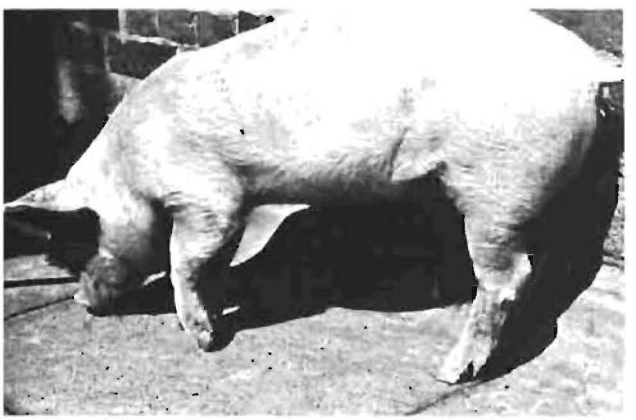

Fig. 3. Pig B – lame in left foreleg.

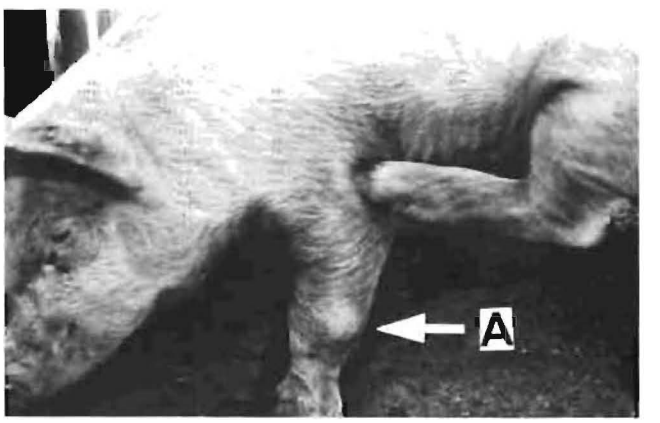

Fig. 4. Pig B – pawing of left shoulder due to pain and swelling in left carpal region (A).

cute pain when touched in the left shoulder region and ctually pawed this area with his hind leg due to the ain. The pig was reluctant to walk and remained re-umbent most of the time. There was a definite swelling f the left carpal region (Fig. 3 & 4). The lameness radually improved and had disappeared 8 days after he onset. The pig was slaughtered 22 days after the last dministration of streptococci. The post mortem exam-nation revealed no pathological changes. Streptococci vere isolated from the left elbow joint which had howed no increase in synovial fluid and no patholog-al changes. The bacterial specimens taken from the emainder of the joints were all negative.

Pig C at no stage showed any adverse effects from the intravenous injections of streptococci. The pig was laughtered 35 days after the last administration of treptococci. The post mortem examination revealed o pathological changes and the bacteriological speci-nens taken from the joints were all negative.

Pig D showed definite signs of lameness in the hind-uarters on the fifth day after the first inoculation of treptococci. The lameness was not marked and lasted or 3 days. The pig showed no signs of fever or ano-exia. Nine days after the third administration of strep-tococci the pig once again showed slight signs of lame-ness in the hind-quarters. This lasted for 7 days but was never marked. The post mortem examination revealed no pathological changes and the bacteriological exami-nation of this joint was negative.

Pigs E and F (controls) showed no adverse effects after the intravenous injection of the sterile PBS solu-tions. The post mortem examinations carried out on these 2 pigs revealed no pathological changes. The specimens taken from the joints opened were all bacte-riologically negative.

DISCUSSION

In the majority of cases of arthritis in swine the infec-tion is believed to be of haematogenous origin and it appears that a bacteraemia is essential for the produc-tion of progressive streptococcal joint lesions[9 10 12]. The synovial layer of the articular capsule is a highly favourable site for the haematogenous localization of bacteria[9]. There is no full explanation of why bacteria localise readily in these structures but the fluid medium in which they find themselves is nutritious and appa-rently does not provide a suitable substrate for leuco-

cyte activity[9 16]. The richness of the synovial vasculature and the discontinuous nature of the synovial membrane probably permits the rapid entry of bacteria into the joint[9].

A number of workers have experimentally reproduced arthritis in young pigs by intravenous inoculations of fresh broth cultures of streptococcal isolates[5 6 8 10–15 20].

The use of the subcutaneous, intraperitoneal and other routes for the inoculation of streptococci in order to reproduce arthritis was proved by some workers to be less reliable[2 4 10 15 20]. The intra-articular route is not advisable on the grounds that virtually any pathogenic bacterium introduced in sufficient quantity into the joint space can induce synovitis[16]. For the above reasons the intravenous route was regarded as the route of choice for this experiment whereby the streptococcal isolates were inoculated into the experimental pigs.

In the numerous experiments cited in the literature wherein attempts were made to reproduce porcine streptococcal arthritis via the intravenous route, a variable pattern of results is evident. A summary of the variations in these experiments follows: the number of streptococci introduced per incoulation[5 8 11 14]; only one or several inoculations varying in number[8 15 19]; the incubation period i.e. from the time when the streptococci were inoculated to the first signs of arthritis[5 8 10–12 19]; some pigs developed arthritis with a lameness of varying persistence and varying intensity[15]; some pigs were continuously lame and other intermittently[8 14 15 19]; some arthritic pigs recovered spontaneously, and some pigs did not become lame at all[5 7 8 12 14 15]; streptococci were re-isolated from some affected joints and in some cases no isolation could be made[5 8 11–14 19]; in some pigs streptococci were isolated from macroscopically normal joints[15 19]; the periods between inoculation with streptococci and sacrifice[8 10 14]. The varied clinical manifestations of experimentally induced streptococcal arthritis compare favourably with the manifestations of natural infections which appear to vary and where some cases recover spontaneously[3].

It can be seen from the results of this experiment (Table 1) that the pigs inoculated with the streptococcal isolates reacted very much in the same erratic manner as that recorded in experimental work of others.

Pig A showed definite clinical signs of arthritis which was confirmed at post mortem examination in spite of the apparent clinical recovery. Streptococci were recovered from the affected joint.

Pig B developed a severe polyarthritis from which it later appeared to recover. Streptococci were isolated from only one joint which, however, showed no macroscopic signs of arthritis. It appears that this pig was recovering from the experimentally induced arthritis. Had the pig been sacrificed a few days later no streptococci would probably have been recovered.

Pig C, in spite of 2 consecutive inoculations of streptococci, failed to develop any signs of arthritis and this was confirmed at post mortem examination.

Pig D developed arthritis with an intermittent lameness and spontaneous recovery appeared to occur. In spite of 3 separate inoculations of streptococci no signs of arthritis were noted and no organisms were isolated at post mortem examination.

Macroscopic signs of arthritis and the recovery of streptococci at post mortem examination would probably have occurred in all the experimental cases had they been sacrificed when showing signs of acute clinical arthritis. In this experiment the slaughter of the pigs was delayed because an attempt was being made to reproduce the more chronic proliferative form of arthritis encouraged in the original investigation[17 18].

Complete and early resolution of streptococcal arthritis can occur if the infection is overcome[9]. The reasons for spontaneous recovery from streptococcal arthritis in swine are not known. It has been speculated that experimentally inoculated pigs may have been previously exposed and thus could have developed a protective immunity; or the pigs may have possessed some natural resistance; or an enhancement of this resistance may occur as a result of the initial inoculation of the streptococci[4 14 20].

The pathogenicity of streptococci in pigs and their ability to produce arthritis after intravenous injections appears to vary according to the strain involved[4 13]; this is the case in spite of the strains having been isolated from natural infections where severe subacute to chronic proliferative arthritis was present. It appears as if various unknown factors such as hypersensitivity may play a role in the pathogenesis of streptococcal arthritis in swine.

Only Pig A showed any pathological signs of arthritis at post mortem examination. The mild subacute serous synovitis shown by the affected joint of Pig A was similar to the milder forms of streptococcal arthritis induced experimentally in pigs by other workers. In such experimental pigs the synovial fluid was increased in volume and was slightly turbid; histopathologically there was vascular engorgement and an infiltration of lymphocytes and plasma cells[5 8–10 12 15 19].

Pigs B and D, in spite of initial signs of arthritis, revealed no pathological changes of the joints at post mortem examination. The possible explanation for this is that after recovery from a mild arthritis the synovial membrane may be completely regenerated, and excessive fluid, bacteria, and somatic cells, are drained off by the lymphatics[1 16].

In spite of the limited number of pigs available for this experiment, the findings of previous workers were reproduced. It can be concluded that the streptococci isolated from the arthritic porcine joints in the above survey showed varying degrees of pathogenicity and arthritogenesis on intravenous inoculation into experimental pigs.

ACKNOWLEDGEMENTS

Mr D.A. Hughes, Bon Accord, is thanked for kindly donating the pigs for this project.

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BOOK REVIEW

BOEKRESENSIE

CANINE MEDICINE AND THERAPEUTICS

Edited by E.A. CHANDLER, J.M. EVANS, W.B. SINGLETON, F.G. STARTUP, S.B. SUTTON and W.D. TAVERNOR Blackwell Scientific Publications, Oxford for the British Small Animal Veterinary Association. 1979 pp 441, figures and tables throughout. Price £29.

This multi-editor book is written primarily for the practising veterinarian in Britain. Stress is placed on the practical aspects of canine medicine.

The text is divided into 18 chapters. These include the principle body systems (cardiovascular; respiratory; ear, nose, throat and mouth; eye; nervous; locomotor; endocrine; skin; blood; alimentary tract and associated glands; urinary; and genital), as well as very useful chapters on autoimmune disease, body fluids, specific infections, endoparasites, poisoning, and behavioural problems. The standard of these chapters varies considerably. There are 3 very poor chapters. The cardiovascular system diseases are briefly described, but anatomy, physiologic and pharmacologic principles are omitted to the detriment of good medicine. The neurological examination is not described in the chapter on the nervous system, and some sections are very confusing. However, this is a beautifully written chapter: the English is far superior to the neurology. The chapter on blood is very disappointing: most of the text is inadequate, irrelevant or useless. The section on bone disease is also very confusing, and many of the concepts are out-of-date and incorrect. In contrast, there are 4 excellent chapters which in themselves make this book worth buying. The urinary and endocrine systems are very clear, fairly comprehensive, and eminently useful, while remaining concise. The many differential diagnostic lists are invaluable. The autoimmune chapter is probably the best 20 pages that has been written on this emerging field. Both the pathophysiology and the diagnostic tests are clearly explained, complimenting the concise yet comprehensive clinical symptoms and therapy. The many behavioural problems of dogs are similarly dealt with in a manner that is applicable to general practice: an invaluable contribution. The other chapters are adequate to good, with many practical diagnostic techniques included. However, some of the therapeutic information is out of date, and there is poor correlation between chapters.

The English have the ability to condense large volumes of information into a concise, readable format. Due to the brevity, some important and relevant information has been omitted, and some sections are fragmented. Useful additions would have been chapters on metabolic disorders, geriatrics, pediatrics, and therapeutic principles. A section on the rational use of antibiotics, corticosteroids, vitamins, tranquilizers, and so on, would have better fulfilled the expectations generated by the title. The referencing through the text is inconsistent, and while some chapters are referenced (always sparsely), others are not. Lists of "further reading" are included at the end of only some chapters. The book is beautifully printed, but the photographs are disappointing, being either of poor quality, or illustrating self-evident findings. The pages are strongly bound and so will withstand the frequent use which the comprehensive index encourages.

Canine Medicine and Therapeutics is a useful basic textbook for the general practitioner, and is the most suitable publication available for undergraduate requirements. However, the inquiring practitioner will find this text lacking in depth and comprehension.

L.S.

THE DISTRIBUTION OF INFECTIOUS BURSAL DISEASE IN SOUTH AFRICA

F. DU PREEZ and S.B. BUYS

ABSTRACT: Du Preez, F.; Buys, S.B. **The distribution of infectious bursal disease in South Africa.** *Journal of the South African Veterinary Association,* (1980) **51** No. 1 15-16 (En). Veterinary Research Institute, 0110. Onderstepoort, Republic of South Africa.

From a country-wide serological survey done during 1978 it is evident that the disease is widely distributed in the areas densely populated with poultry in South Africa.This survey was done before vaccine were in general use in the country.

INTRODUCTION

Infectious bursal disease (IBD) was diagnosed clinically and the virus isolated in the Republic of South Africa at the Veterinary Research Institute, Onderstepoort, in 1969. No new cases were identified subsequently until 1973 when the clinical disease became widespread in the western Cape. This paper reports the results of a serological survey which was conducted in order to establish the distribution of IBD in the Republic.

MATERIALS AND METHODS

Antiserum

Antiserum was prepared by inoculating 4–5-week-old specific pathogen free (SPF) chickens intra-ocularly with 0,05 ml of a 1:10 dilution of antigen (Cheville) obtained from the Central Veterinary Laboratory, Weybridge, England. Birds were exsanguinated 28 days post-inoculation and the serum stored in small aliquots at –20°C.

Agar-gel-precipitation antigen

Five-week-old SPF birds were inoculated intra-ocularly with 0,05 ml of an IBD-virus infected bursal homogenate. The birds were killed 72 h later and the bursae collected aseptically. These were then weighed and equal volumes of cold phosphate buffered saline (PBS) and Freon* were added to them. The mixture was then homogenized in an Ultra-Turrax (Type TP 18/10 at 20 000 rpm)† and centrifuged at 2 000 g for 30 min. The aqeuous phase was harvested as antigen, dispensed in small volumes and stored at –20°C[1].

Test procedure

A specially designed perspex mould (Fig. 1) which fits over a 9 cm plastic Petri dish was used, and any desired pattern of wells could be made by means of a cork cutter. The procedure followed was that of Woernle[2]. A positive and negative control serum was added to each petri dish.

Test sera

Representative sera samples from different parts of the country were tested to establish the distribution of the infection throughout South Africa. Some of the sera specimens were received from broiler flocks derived from 2 different farms from birds aged respectively 1, 7, 14, 21, 28, 35, 42 and 56 days.

*ICI Chemicals, P.O. Box 11270, Braamfontein.
†Janke & Kunkel, Optalabor, P.O. Box 31208, Braamfontein.

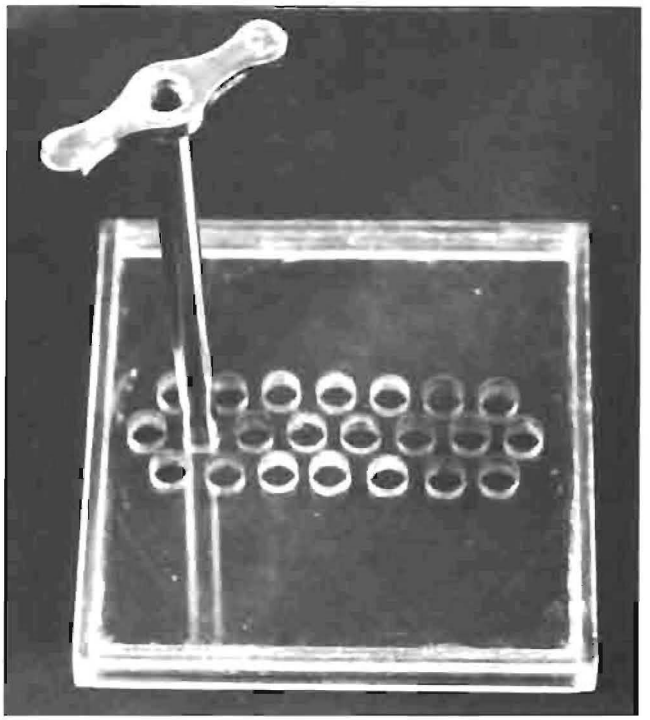

Fig. 1 The specially designed perspex mould used.

RESULTS

Twenty-one out of 26 parent flocks and 16 out of 22 broiler flocks were serologically positive for IBDV (Table 1).

Table 1: DISTRIBUTION OF PRECIPITATING ANTIBODIES AGAINST IBDV IN DIFFERENT LOCALITIES IN SOUTH AFRICA

Province	Breed	No. of flocks tested	No. of flocks positive	% infection rate (range)
Transvaal	Broiler parent flock	6	5	13–100
	Layer parent flock	10	8	67–100
	Broilers	21	19	5–100
Natal	Broilers	1	1	100
Orange Free State	Broiler parent flock	1	0	0
	Layer parent flock	8	7	100
Western Cape	Broiler parent flock	1	1	100

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In table 2 the results of the serological tests done on Farm No. 1 are tabulated.

Table 2: DISTRIBUTION OF PRECIPITATING ANTIBODIES AGAINST IBDV ON FARM No. 1 IN A FLOCK AT DIFFERENT AGES

Age (days)	No. of sera tested	No. of sera positive	% Positive
1	20	5	25
7	18	0	0
14	20	0	0
21	10	0	0
28	10	0	0
35	10	0	0

Although these chickens originated from an unvaccinated farm, 25 % of the day-old chickens had maternal antibodies. The fact that all further tests done on Days 7, 14, 28 and 35 were negative indicates that chickens on this specific broiler farm were not infected with IBD.

The results of the serological tests done on broiler Farm No. 2 are tabulated in Table 3.

Although these chickens also originated from an unvaccinated parent flock, 90 % showed a positive serological result at Day 1. The percentage of serologically positive birds as well as their antibody titres dropped. At Days 14 and 21 the chickens were serologically negative. They then became infected, 100 % of the serum samples being positive at 43 days of age. At 56 days all the serum samples tested werre positive but no titration was done.

Table 3: DISTRIBUTION OF PRECIPITATING ANTIBODIES AGAINST IBDV ON FARM No. 2 IN A FLOCK AT DIFFERENT AGES

Age (days)	No. of sera tested	No. of sera positive	Titre log base 2	% Positive
1	20	18	1	90
7	20	5	Undiluted	25
14	20	0	Neg.	0
21	20	0	Neg.	0
28	20	20	1	100
35	20	15	4	75
43	20	20	5	100
56	20	20	ND	100

ND = Titration not done

DISCUSSION

This serological survey was conducted before the IBDV vaccine was in general use in this country. It is evident therefore that the disease is widely distributed in all the densely populated poultry areas. Since certain parent flocks are still susceptible, it is essential that they should be vaccinated. This will ensure a more homogeneous distribution of antibodies and better protection in the offspring.

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THE CONTROL OF PARASITES IN ANTELOPE IN SMALL GAME RESERVES*

I.G. HORAK

ABSTRACT: Horak I.G. **The control of parasites in antelope in small game reserves.** *Journal of the South African Veterinary Association* (1980) **51** No. 1 17-19 (En). Department Parasitology, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The most important parasites of antelope in small game reserves are ixodid ticks, the larvae of oestrid flies and nematodes. Methods of control based on the seasonal prevalences of these parasites are suggested.

Adult ticks can be controlled during summer by the introduction of cattle which are dipped or sprayed with an acaricide at 5-day intervals. The larvae of oestrid flies can be controlled by the capture during winter of all alcelaphine antelope and their oral treatment with a larvicide, while the addition of an anthelmintic to feed supplement blocks during winter can be used to control nematodes.

INTRODUCTION

Contrary to popular belief all antelope, with the possible exception of those in zoos, are normally infested with numerous arthropod and helminth parasites[1 7 8 10 12 14] and large burdens may frequently be encountered[8 10 12]. Massive increases in parasite numbers and even frank parasitism[16 19] may occur in small game reserves because of the numerous hosts of the same or different species, often maintained at high stocking rates. The control of parasites in these reserves is thus often of considerable economic importance to the owner or manager of the park.

The control measures suggested are based on general trends observed in parasite surveys conducted in antelope[7 8 10 12] and in domestic stock infested with the same or closely related parasites[5 6 9 13].

THE PREVALENCE OF PARASITES

Before a control programme is planned the parasites should be identified and their host preferences and seasonal prevalences known. Recent surveys of parasites in several antelope species in the Republic of South Africa (RSA)[1 7 8 10 12] indicate that the most important parasites are nematodes[7 8], ixodid ticks[12] and the larvae of oestrid flies[10]. Table 1 lists the nematodes, ixodid ticks and oestrid larvae that I have recovered during surveys from blesbok, blue wildebeest, impala and springbok.

Ixodid ticks

As a general rule it can be assumed that blue wildebeest and smaller species harbour mainly the larval and nymphal stages of ticks (IG Horak 1979 Unpublished work). Larger species harbour both immature and adult ticks[12] and the larger the host species the more adult ticks it seems likely to carry[14]. Thus where both small and large antelope species are confined in the same reserve, tick infestations may become particularly severe because of the abundance of hosts for the immature stages.

As a general rule applying to the 2- and 3-host ticks, larvae are present in the greatest numbers during autumn and winter, nymphae during winter and spring and adults during summer[12 13]. These peaks are often well-defined and may be of short duration[13].

*Paper presented at the South African National and International Veterinary Congress, Johannesburg, September 1979.

The larvae of oestrid flies

The nasal passages and para-nasal sinuses of antelope of the alcelaphine group (head-nodders such as blesbok, tsesseby and wildebeest) are usually infested with one or more species of larvae of flies belonging to the genera *Gedoelstia, Kirkioestrus* and *Oestrus*[10 20], while springbok and giraffe may harbour the larvae of *Rhinoestrus* spp.[20].

In the Highveld and higher altitude Bushveld regions of the RSA oestrid flies are active from October to May or June[5 10]. Burdens, consisting of first, second and third stage larvae, generally increase in numbers in the nasal passages from November to June and decline thereafter to a low level during September and October[5 10]. From April to July large proportions of the total burden consist of first stage larvae[5] which overwinter in the host's head in this stage of development. This is necessary because neither pupae nor adult flies can survive during winter[5 10].

Although these larvae cause no apparent serious clinical manifestations in their normal antelope hosts[3], *Gedoelstia* spp. larvae in aberrant hosts such as sheep and cattle, kept in close proximity to the antelope, can cause severe disease[2].

Nematodes

The seasonal prevalence of several species of nematodes in the Highveld and higher altitude Bushveld regions of the RSA is similar to that of the larvae of oestrid flies. Burdens increase from November to May, remain at a high level until September and then decrease[6 8 9]. Adult worms are present in large numbers from November to February or March, but from February to July ever-increasing proportions of the total burden consist of fourth stage larvae arrested in their development[6 8 9]. This is an overwintering device ensuring the survival of the nematodes in a stable internal environment, at a time when survival outside the host is precarious because of unfavourable climatic conditions[15].

CONTROL

Ixodid ticks

Control involves the use of cattle as hosts for the ticks (RAI Norval 1979 Veterinary Research Laboratory, Salisbury, personal communication). *Bos taurus* breeds should be used as they are more susceptible to infestation by certain ticks than *Bos inducus* breeds[17]. Large

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Table 1: NEMATODES, IXODID TICKS AND OESTRID FLY LARVAE RECOVERED FROM BLESBOK, BLUE WILDEBEEST, IMPALA AND SPRINGBOK

Blesbok	Blue Wildebeest	Impala	Springbok
Nematodes	Nematodes	Nematodes	Nematodes
Cooperia hungi	*Agriostomum gorgonis*	*Cooperia connochaeti*	*Agriostomum equidentatum*
Cooperia yoshidai	*Cooperia connochaeti*	*Cooperia hungi*	*Cooperia hungi*
Dictyocaulus magnus	*Dictyocaulus viviparus*	*Cooperioides hamiltoni*	*Cooperioides antidorca*
Haemonchus bedfordi	*Gaigeria pachyscelis*	*Cooperioides hepaticae*	*Dictyocaulus magnus*
Haemonchus contortus	*Haemonchus bedfordi*	*Haemonchus placei*	*Haemonchus bedfordi*
Impalaia nudicollis	*Oesophagostomum multifoliatum*	*Impalaia tuberculata*	*Haemonchus contortus*
Impalaia tuberculata	*Strongyloides* sp.	*Longistrongylus sabie*	*Impalaia nudicollis*
Longistrongylus albifrontis	*Trichostrongylus thomasi*	*Oesophagostomum columbianum*	*Longistrongylus albifrontis*
Oesophagostomum columbianum		*Strongyloides papillosus*	*Nematodirus helvetianus*
Skrjabinema alata	Ixodid ticks	*Trichostrongylus axei*	*Oesophagostomum africanum*
Trichostrongylus axei	*Amblyomma hebraeum*	*Trichostrongylus colubriformis*	*Paracooperia serrata*
Trichostrongylus falculatus	*Boophilus decoloratus*	*Trichostrongylus falculatus*	*Strongyloides* sp.
Trichostrongylus thomasi	*Hyalomma truncatum*		*Trichostrongylus axei*
	Rhipicephalus appendiculatus	Ixodid ticks	*Trichostrongylus colubriformis*
Ixodid ticks	*Rhipicephalus evertsi evertsi*	*Amblyomma hebraeum*	*Trichostrongylus falculatus*
Rhipicephalus appendiculatus		*Boophilus decoloratus*	*Trichostrongylus thomasi*
	Oestrid larvae	*Ixodes cavipalpus*	
Oestrid larvae	*Gedoelstia cristata*	*Rhipicephalus appendiculatus*	Ixodid ticks
Gedoelstia hässleri	*Gedoelstia hässleri*	*Rhipicephalus evertsi evertsi*	*Rhipicephalus appendiculatus*
Oestrus macdonaldi	*Kirkioestrus minutus*		*Rhipicephalus evertsi evertsi*
Oestrus variolosus	*Oestrus aureoargentatus*		
	Oestrus variolosus		Oestrid larvae
			Rhinoestrus sp.

numbers of cattle must be introduced to graze the game reserve with the antelope in order to pick up as many ticks as possible in a comparatively short period of time.

If the cattle are introduced during autumn or during spring they will acquire either tick larvae or tick nymphae respectively[12,13] of the 2- and 3-host ticks. Large numbers of these immature ticks will, however, be present on the antelope which also serve as efficient hosts of these immature stages. If the cattle are introduced during summer when the adult ticks reach peak numbers[12,13] these will prefer the cattle as hosts particularly if the game reserve contains only antelope of the smaller species. The most advantageous time to introduce cattle would appear to be during January and February when adult ticks of most species are abundant[13]. During this time the cattle must be dipped or sprayed with an acaricide at approximately 5-day intervals as longer intervals will enable ticks of some species to attach, engorge and drop off in the periods between treatment[11]. The acaricide used must have little or no residual tick-repellant effect as this would prevent the cattle picking up as many ticks as possible in the intervals between acaricide applications.

The larvae of oestrid flies

These larvae cause little readily detectable disease in their definitive antelope hosts[3]. If, however, eye problems associated with the presence of oestrid larvae are encountered in domestic livestock on farms adjoining a game reserve, treatment of the alcelaphine antelope hosts may become necessary. This will involve the capture and oral treatment with rafoxanide* of all host animals[18]. Maximum benefit can be obtained from this treatment if it is administered during July when the fly population on the Highveld and higher altitude Bushveld regions depends for its survival on larvae overwintering in the antelopes' nasal passages[10].

Nematodes

These can best be controlled by the addition during July of an anthelmintic such as fenbendazole** to feed supplement blocks. This would hopefully ensure the daily ingestion of a low level of anthelminctic sufficient to control the nematode burdens of the antelope. Not only is July a good month to treat for nematodes, because the majority of these are overwintering in the host at this time[8], but antelope are more inclined to take feed supplements during winter because of the seasonal shortage of grazing.

A few practical hints may be of value:–

(i) In order to get the antelope to take feed blocks, licks consisting of coarse salt must first be put down. Once the animals are making use of these licks unmedicated feed blocks can be placed in their vicinity and only when these blocks are being readily consumed should medicated blocks be introduced.

(ii) To obtain the best results sufficient medicated blocks must be provided. This implies that blocks must not be placed at one site only but must be placed in the grazing and loafing territories of the various antelope species. Where aggressive animals such as eland, which will each commandeer its own block, are present, sufficient blocks to accommodate the eland and the other antelope must be provided.

(iii) Regular checks should be made to see which species are eating the blocks so that more blocks can be placed in the habitats of the shy-feeders. These checks will also serve to indicate in which species

*Ranide: MSD (Pty) Ltd.

**Panacur: Hoechst Pharmaceuticals

good results and in which poor results can be expected.

(iv) Zebras and warthogs are inclined to bite or chop the blocks to pieces or to roll them around. To prevent this the blocks can be placed in metal frames securely fastened in the soil.

(v) Whereas effective anthelmintic control can be obtained in cattle and sheep by presenting them with medicated blocks for 4–5 days[4], it is adviseable to allow the antelope 2–4 weeks access to these blocks. This may ensure that even shy-feeders will ingest sufficient anthelmintic to be effective.

New introductions

Before introducing antelope from other regions into a game reserve they must be dipped or sprayed with an acaricide and treated for the larvae of oestrid flies and for nematodes. This will not only reduce the chances of introducing parasites hitherto not present in the reserve, but will also eliminate the additional stress of existing large parasite burdens in the new introductions during their period of adaptation to the strange environment.

Before large species such as zebra, kudu, eland, buffalo or giraffe are introduced into a reserve in which only smaller antelope species are present, the implications of possible future massive tick burdens must be considered.

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THE ANTHELMINTIC ACTIVITY AND TOXICITY OF 2,2-DICHLOROVINYL DIMETHYL PHOSPHATE (DICHLORVOS)* IN EQUINES

R.K. REINECKE**, L.J. LOOTS† and P.M. REINECKE‡

ABSTRACT: Reinecke R.K.; Loots L.J.; Reinecke P.M. **The anthelmintic activity and toxicity of 2,2-dichlorovinyl dimethyl phosphate (dichlorvos) in equines.** *Journal of the South African Veterinary Association* (1980) **51** No. 1, 21-24 (En) Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Rep. of South Africa.

Dichlorvos in a special slow release formulation at 31 mg/kg body mass in equines was highly effective against all adult strongyles and *Oxyuris equi, Parascaris equorum, Probstmayria vivipara* and bots of *Gasterophilus* spp. It has no effect on 4th stage larvae of *Trichonema* spp. nor the stomach worms *Draschia megastoma* and *Habronema* spp. Doses of dichlorovos 10 and 20 times the therapeutic dose (310 and 620 mg/kg body mass) caused transient clinical signs but these disappeared 96 hours after dosing.

INTRODUCTION

A single dose of dichlorvos at 33–43 mg/kg body mass was 90–100 % effective against *Strongylus vulgaris, Parascaris equorum* and bots of the species *Gasterophilus intestinalis* and *Gasterophilus nasalis*. Its efficiency however, fell to range from 65–75 % against *Strongylus edentatus*[1].

This paper describes anthelmintic and toxicity tests on donkeys and horses carried out in the RSA.

FAECAL WORM EGG COUNTS

MATERIALS AND METHODS

Nineteen donkeys were stabled at Nigel, faeces collected, worm egg counts carried out[6], and larvae identified[12].

RESULTS

Egg counts for strongyles varied from 0–5 600 epg and although 11 donkeys were negative for *Oxyuris equi*, the others had egg counts ranging from 66–3 800 epg. Larval cultures inevitably had *Trichonema* spp. ranging from 23–100 % but only a small percentage of larvae were *Strongylus equinus, Strongylus edentatus* or *Strongylus vulgaris* and only 4 donkeys had *Strongyloides westeri*. Five donkeys were selected for critical tests mainly on the basis of the presence of at least 2 and if possible all 3 species of *Strongylus* spp.

CRITICAL ANTHELMINTIC TESTS

MATERIALS AND METHODS

(a) Administration of dichlorovos

Each donkey was placed on a weighbridge and the mass noted. The correct mass of a slow release formulation of dichlorovos* was determined, and mixed with the feed and then given to the donkey which had been starved overnight and secured in a stable. The time taken to eat the medicated food varied: 3 donkeys had consumed it all in 1 hour and the other 2 took 24 hours.

**Department of Parasitology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort.
†Animal Hospital, Windsor Road, 1490 Nigel.
‡3 Pine Ave., 1000 Cullinan.
*Equiguard (Shell Chemicals).

(b) Faecal examination

All faeces expelled after dosing were collected in the morning and evening for at least 3, and in 2 donkeys, 4 days. These specimens were labelled, carefully examined macroscopically for worms 10 mm or longer and for bots. The parasites were transferred to labelled containers and preserved in 10 % formalin. After thorough mixing the entire faecal mass was determined and 2 1/10 and 2 1/100 aliquots by mass removed, placed in large mouthed jars and formalin added. The former were subsequently examined macroscopically and the latter microscopically for worms, the worms transferred to labelled containers and the total collected noted.

(c) Necropsy

Donkeys were killed 3–18 days after treatment and methods previously described used to collect worms in the waterbath[8]. The following modifications were incorporated:

(i) Worms were not heat killed but 10–15 ml of 45 % iodine used. Subsequently they were fixed in formalin, sieved on sieves with 39 μm apertures and preserved in formalin.
(ii) A macroscopic examination for worms was made on the entire ingesta of the caecum, dorsal and ventral colon and rectum. Worms present were preserved in 10 % formalin.
(iii) Only 1/5 aliquots by mass of the caecal, ventral and dorsal colonic wall were digested with a mixture of pepsin/HCl to release trapped larvae.
(iv) Nylon bolting cloth with apertures of 500 μm replaced the lower layer of nylon mesh used for sieving in the trap.

(d) Worm recovery

Microscopic examination was carried out on the 1/5 aliquot of digested gut wall and 1/100 aliquots of ingesta. On all other specimens of ingesta and 1/10 aliquots of faeces macroscopic examination was carried out.

(e) Identification

Strongylata were identified on a generic basis only and *S. equinus, S. edentatus, S. vulgaris, Habronema musca, Habronema majus, Draschia megastoma, Oxyuris equi* and *Probstmayria viviparus* to species. Keys of Theiler[14] and Skrjabin *et al.*[13] were used to identify

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worms and *Gasterophilus* larvae identified according to Zumpt[15].

Table 1: RESULTS OF CRITICAL ANTHELMINTIC TESTS IN DONKEYS

Donkey No. and Specimen	*Trichonema*		*Cylicocyclus*		*Poteriostomum*		*Triodontophorus*		*Craterostomum*	*Gyalocephalus*	*S. equinus*	*S. vulgaris*	*O. equi*	*H. muscae*		*H. majus*	*D. megastoma*	*G. intestinalis*		TOTAL excluding *P. vivipara*	*P. vivipara*
	L_4[1]	A[2]	L_4	A	L_4	A	L_4	A						5[3]	A			2[4]	3[5]		
6																					
Autopsy	85	216	15	22	5	5	8	5	0	0	0	0	0	0	578	0	433	0	0	1372	0
Faeces	0	1526	0	721	0	95	0	239	34	0	0	2	3	0	0	0	0	0	0	2620	709600
TOTAL	85	1742	15	743	5	100	8	244	34	0	0	2	3	0	578	0	433	0	0	3992	709600
Reduction %	0	87,6	0	97	0	95	0	97,9	100	–	–	100	100	–	0	–	0	–	–	65,6	100
10																					
Autopsy	0	149	0	5	0	0	0	0	0	0	0	0	0	0	0	0	11	0	5[6]	170	0
Faeces	0	2495	0	2743	0	245	0	17	1	1	20	47	2	0	0	0	0	0	0	5517	80866
TOTAL	0	2644	0	2748	0	245	0	17	1	1	20	47	2	0	0	0	11	0	5	5687	80866
Reduction %	–	94,4	–	99,8	–	100	–	100	100	100	100	100	100	–	–	–	0	–	100	97,0	100
16																					
Autopsy	0	409	0	0	0	5	0	0	0	0	0	0	0	0	459	8	49	0	0	930	0
Faeces	0	2101	0	60	0	225	0	56	0	0	17	0	45	0	0	0	0	0	0	2504	0
TOTAL	0	2510	0	60	0	230	0	56	0	0	17	0	45	0	459	8	49	0	0	3434	0
Reduction %	–	83,7	–	100	–	97,8	–	100	–	–	100	–	100	–	0	0	0	–	–	72,9	–
19																					
Autopsy	0	521	0	17	0	0	0	0	0	0	0	6	0	109	57	0	1640	0	35[6]	2385	0
Faeces	0	5493	0	1474	0	547	0	2116	0	50	42	100	3	0	0	0	0	2	19	9846	36837
TOTAL	0	6014	0	1491	0	547	0	2116	0	50	42	106	3	109	57	0	1640	2	54	12231	36837
Reduction %	–	91,3	–	99	–	100	–	100	–	100	100	94,3	100	0	0	–	0	100	100	80,5	100
21																					
Autopsy	105	160	0	0	0	0	0	0	0	0	0	0	0	0	0	0	401	0	0	666	0
Faeces	0	1035	0	0	0	25	0	80	0	0	0	0	0	0	0	0	0	0	0	1140	9400
TOTAL	105	1195	0	0	0	25	0	80	0	0	0	0	0	0	0	0	401	0	0	1806	9400
Reduction %	0	86,6	–	–	–	100	–	100	–	–	–	–	–	–	–	–	0	–	–	63,1	100

(1) L_4 = Fourth stage larvae
(2) A = Adult
(3) 5 = Sexually immature fifth stage
(4) 2 = Second instar larvae
(5) 3 = Third instar larvae
(6) Dead larvae in the colon and therefore regarded as being expelled.

RESULTS

With the exception of Donkey 21 in which most worms were expelled 3 days after treatment, the majority of worms were expelled within the first 2 days.

Donkeys 16 and 19 were killed 4 days after treatment and the ingesta of the caecum and colon of these donkeys contained minute cylindrical white bodies i.e. the remnants of the resin pellets mixed with the anthelmintic. Dead *Gasterophilus* larvae in the ingesta of the colon were regarded as having been expelled. Data in these tests are summarized in Table 1.

The most numerous parasites were *P. vivipara* and *Trichonema* spp. followed by *Cylicocyclus* spp. *Poteriostomum* spp. *Triodontophorus* spp., *Habronema musca* and *Draschia megastoma*. There were small numbers of *Craterostomum* spp. *Gyalocephalus capitatus, S. equinus, S. vulgaris* and the bot larvae *Gasterophilus intestinalis*.

Anthelmintic efficacy against adult worms varied from 83,7–10 % for *Strongyles* and 100 % against *O. equi, P. vivipara* and the bot larvae *G. intestinalis*.

It had no effect against 4th stage larvae of *Trichonema* spp. nor adult *H. musca* or *D. megastoma*.

Lichtenfels[4] does not recognise the subfamily Trichonematinae but places these worms in the subfamily Cyathostominae. His publication was not available at the time of these trials in 1973 but most of the worms classified in this paper as *Trichonema* spp. belong to either the genus *Cylicostephanus* or *Cyathostomum*.

CRITICAL TESTS FOR *GASTEROPHILUS* spp. AND *O. EQUI*

MATERIALS AND METHODS

Faeces were collected from 1 horse and 5 donkeys after treatment, examined macroscopically for bot larvae or *O. equi* which were subsequently identified microscopically. At necropsy, the ingesta of the stomach were examined for larvae of *Garophilus* and rectum for *O. equi*. The worms expelled were compared with those recovered at necropsy.

RESULTS

Some larvae not identified to species were designated *Gasterophilus* spp. Both *G. intestinalis* and *G. nasalis* were present and efficacy varied from 81,2–100 %. There was no difference in efficiency against 2nd or 3rd instar larvae.

All *O. equi* were expelled. The ingesta of the colon should also have been examined to be absolutely certain. In the previous tests however, when these ingesta were examined all *O. equi* were expelled from 4 infested donkeys.

MODIFIED CRITICAL ANTHELMINTIC TEST

If faeces are examined before and after treatment for worm eggs and worms collected after treatment for counting and identification, the test is referred to as a modified critical test because the host is not killed to count the residual worm burden at necropsy[9].

In previous trials no animals had *S. edentatus* or *P. equorum*. The next two trials were planned to test the compound against these species.

STRONGYLUS spp.

MATERIALS AND METHODS

Six donkeys and 4 horses were used. Faeces were examined before treatment and from 2–4 weeks every week after treatment. Worms expelled were collected for 3–4 days after treatment.

RESULTS

The faeces of all animals were negative for worm egg counts after treatment. Eight animals expelled *S. edentatus* in numbers ranging from 1–36; 5 had *S. equinus* (range 1–16) and all 11 *S. vulgaris* (range 1–36). A single horse was infested with *P. equorum*, 7 worms were expelled and worm egg counts became negative after treatment.

P. EQUORUM

MATERIALS AND METHODS

Four out of 7 foals and yearlings had egg counts of this species ranging from 200–3 000 epg. Dichlorvos was mixed with molasses and concentrates and dosed to these 4 animals and faecal worm egg counts carried out 7 days after treatment. Faeces expelled were collected for 48 hours after treatment and all *P. equorum* counted.

RESULTS

With the exception of Foal 13 which had 66 epg all animals were negative 7 days after treatment. The number of *P. equorum* recovered from faeces varied:

Foal No.	5th stage	Adult
13	46	58
31	6	5
7058	0	1
7092	42	10

If egg count data are used efficiency against *P. equorum* varied from 97,8–100 %.

TOXICITY TESTS

MATERIALS AND METHODS

The mass of the horses and donkeys was determined for each animal on a weighbridge. Equiguard was used. This is a special formulation in which the active ingredient is slowly released in its passage through the intestinal tract. The active ingredient dichlorvos in this formulation was dosed to 2 donkeys at 620 mg/kg body mass i.e. 20 times the therapeutic dose and 4 horses were dosed at 310 mg/kg or 10 times the normal dose.

Blood samples were collected in heparin for whole blood cholinesterase determinations at the South African Bureau of Standards according to the method of Ellman *et al.*[3] at the following periods: 24h before, at dosing (=0), + 3h, + 6h, + 12h, + 24h, + 48h, + 96h and + 7 days after dosing respectively. Simultaneously clinical signs were recorded.

RESULTS

There was considerable difficulty in dosing the smaller donkey (No 3, 138 kg mass) with this dose of dichlorvos and considerable quantities of water (30 ℓ or more) were used to wash the resinous pellets down the stomach tube. The stomach swelled alarmingly and the animal was in pain for a few hours. This was not so marked in the other donkey (150 kg mass) nor in the horses which received a smaller dose proportionate to their mass.

Clinical signs

Donkey 8: Three hours after dosing slight discomfort and twitching of muscles which disappeared at 6h and 12h; depression at 24h and 48h with loose stools but thereafter the donkey was normal.

Donkey 3: This was the smaller of the two donkeys. At 3, 6 and 12h stools were very loose with slight discomfort at 12h. Thereafter the signs were the same as those in donkey 8.

The horses were less severely affected and showed some discomfort at 3–12h and consistantly loose stools which started in one horse at 12h but elsewhere was only noted at 24 and 48h and thereafter animals returned to normal.

At the following periods after dosing dichlorvos, + 12h, + 24h and + 48h, the mean whole blood cholinesterase levels for the group fell to 49 %, 38 % and 23 % respectively, of the cholinesterase levels prior to dosing. These were dangerously low levels which in some instances were nearly zero (T W Naudé 1980 Veteri-

nary Research Institute, Onderstepoort, personal communication).

Despite the more marked clinical signs of intoxication in the 2 donkeys dosed at 20 times the therapeutic dose, the whole blood cholinesterase levels in the donkeys were no lower than the 4 horses dosed only at 10 times the normal dose. Species difference may possibly account for this difference. Both in horses and donkeys the lowest cholinesterase levels were noted at + 48 h when practically all the clinical signs had abated. This has also been observed in ruminants and the reasons for this are obscure (Naude 1980 personal communication).

DISCUSSION

Dichlorvos at 31 mg/kg was extremely efficient in removing all adult *O. equi, P. vivipara* and 2nd and 3rd instar larvae of *G. intestinalis*. It was 97,8–100 % effective against *P. equorum*. and varied from 83,7–100 % against all adult strongyles present. It had no effect on *H. muscae, H. majus, D. megastoma* and 4th stage larvae of *Trichonema* spp.

This is not necessarily a true reflection of the inefficiency of the compound but may be due to the defects of the critical anthelmintic test. In sheep it has been proved that the critical test is totally unsuited to larvae of any species and adult worms of the abomasum[9 10]. Stomach worms and larval stages in the caecum and colon in horses may also disappear before being expelled.

Recently Duncan *et al.*[2] carried out a controlled test with fenbendazole on young ponies 6–12 months of age. They were positive for strongyle eggs.at treatment except one control; 10 out of 16 ponies had *P. equorum* eggs. Some 29(+1) days later at necropsy, with the exception of one control, all were negative for strongyle eggs and only one treated pony had *P. equorum* eggs. They postulated that transport, the change of diet and environment were responsible for worm expulsion because adult *Trichonema* spp. were only present in one control.

At necropsy immature worms were distributed as follows:

Trichonema spp 4th stage larvae in the caecal and colonic wall, 5th stages in the ingesta;

S. vulgaris 4th stage larvae in the cranial mesenteric artery and 5th stages in the wall and lumen of the colon;

S. edentatus 4th stage larvae between the peritoneum and muscles of the abdominal wall and

O. equi 4th stage larvae only but the site was not specified.

Habronema spp. from the stomach. These were probably adults but this was not specified in the text.

There is no doubt that this is the best description of a controlled test on immature strongyles and adult (?) *Habronema* spp. A major contribution has been made to our techniques of testing anthelmintics and the compound used is obviously effective but not necessarily to the extent claimed[2].

The markedly skew distribution suggested that a non-parametric method be used and we analysed their[2] results by the Mann Whitney U test. The anthelmintic was dosed at 15, 30 and 60 mg/kg. At the highest level reductions by this test were highly significant ($p < 0,05$) for 4th stage larvae of *S. vulgaris, S. edentatus* and 5th stage *Trichonema* spp. and total worm burdens of the latter. Against 4th stage larve of *Trichonema* spp. significance fell to $p < 0,3$. Two controls had no *Habronema* spp. and significance was very low ($p < 0,4$).

Although it would be highly desirable to apply the modified non-parametric method it requires 11 treated animals and at least 5 controls[7]. It is extravagant in labour and animals and very doubtful if the biological requirements would be fulfilled.

Additional evidence by the same team to substantiate their original claims were subsequently presented[5]. Adult horses dosed at 60 mg/kg were free of worm eggs for 7 weeks and by 8 weeks 6/10 animals were positive for *Trichonema* spp. but *S. vulgaris* was absent for 15 weeks after treatment. Even after 18 weeks 4/10 horses were negative for all stronglyle eggs.

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ANTHELMINTIC EFFICACY OF FENBENDAZOLE AGAINST CESTODES IN SHEEP AND CATTLE

F.S. MALAN

ABSTRACT: Malan F.S. **Anthelmintic efficacy of fenbendazole against cestodes in sheep and cattle.** *Journal of the South African Veterinary Association* (1980) No. 1, 25-26 (En) Hoechst Research Farm, P.O. Box 124, 1320 Malelane. Rep. of South Africa.

The anthelmintic efficacy of fenbendazole FBZ against *Moniezia expansa* was tested in sheep and cattle at a dosage rate of 10 mg FBZ per kg body mass. Twenty seven out of 30 lambs and 8 out of 12 calves were cured of the infestation.

INTRODUCTION

Düwel & Tiefenbach[1] reported that over 5 800 lambs and sheep infected with *Moniezia* spp. were effectively treated with 10 mg fenbendazole (FBZ) per kg body mass. In all cases a clinical cure occurred within a short time. Over 95 % of the tapeworms – both *Moniezia expansa* and *Moniezia benedini* were expelled.

This paper reports on trials done in the Republic of South Africa in sheep and cattle.

MATERIALS AND METHODS

Thirty-five merino lambs, 3–6 months old and 12 Drakensberger crossbred calves, 6–18 months old, which were naturally infested with *Moniezia expansa* were used. Infestations were proved by demonstrating tapeworm segments in the faeces of each animal. The animals were bought from farmers in the Carolina district, Transvaal.

Animals were kept individually in pens with a cement floor. They were fed hay *ad libitum* and fresh clean water was available.

The mass of each of 30 sheep and 12 calves was determined and after being dosed with FBZ per os, faecal collecting bags were attached to each lamb and calf. Five lambs were kept as untreated controls. The tapeworm segments collected from the faecal bags were placed in water in measuring cylinders and the volume of water displaced was noted. Animals were slaughtered 5–7 days after treatment.

Table 1: CRITICAL ANTHELMINTIC TESTS IN SHEEP INFESTED WITH *M. EXPANSA* AND DOSED WITH FBZ AT 10 mg/kg BODY MASS

Sheep Number	Body Mass (kg)	FBZ 2,5 % dosed (mg)	FBZ 2,5 % dosed (ml)	Volume water displaced by segments (ml)	Scoleces found at necropsy
789	17,0	170	6,8	55	2
802	12,2	122	4,9	91	4
806	16,0	160	6,4	20	0
807	15,0	150	6,0	70	0
808	15,3	153	6,1	34	0
811	17,5	715	7,0	46	0
812	12,0	120	4,8	5	0
813	15,5	155	6,2	50	0
815	16,0	160	6,4	54	0
834	12,5	125	5,0	10	0
893	13,3	133	5,3	2	0
900	13,6	136	5,4	120	0
912	12,5	125	5,0	25	0
974	8,5	85	3,4	2	0
975	10,5	105	4,2	1	0
978	15,9	159	6,4	30	0
979	13,3	133	5,3	80	0
980	13,0	130	5,2	45	0
981	14,0	140	5,6	96	2
982	12,9	129	5,2	40	0
983	10,0	100	4,0	10	0
984	16,0	160	6,4	45	0
985	15,0	150	6,0	22	0
986	12,3	123	4,9	46	0
987	12,2	122	4,9	60	0
989	15,1	151	6,0	12	0
991	10,5	105	4,2	35	0
994	13,5	135	5,4	60	0
A	20,0	200	4,8	15	0
B	20,0	200	6,1	34	0
Undosed Controls					
433	9,5	–	–	145	1
432	15,0	–	–	70	2
437	13,5	–	–	175	27
438	7,5	–	–	120	11
439	19,0	–	–	195	4

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At necropsy the mesenteric fat was stripped and the small intestine isolated. The ingesta of the small intestine was washed out with luke warm water, which was then poured onto a 150 μm sieve and the residue obtained was diligently examined macroscopically for the presence of scoleces and tapeworm segments in a container with a black background.

The gut was then opened with a bowel scissors and the mucosa scanned for scoleces.

RESULTS

The body mass of each animal, volume of FBZ dosed, volume of water displaced by tapeworm segments and the number of scoleces recovered from trial animals are summarized in Tables 1 and 3. The excretion rate of segments in the faeces after treatment is recorded in Table 2.

Table 2: EXPULSION OF STROBILA OF *M. EXPANSA* EXPRESSED IN THE NUMBER OF HOURS AFTER TREATMENT OF THE SHEEP WITH FBZ AT 10 mg/kg. THE VOLUME OF SEGMENTS IN THE FAECES IS EXPRESSED IN ml OF WATER DISPLACED

Sheep Number	+ 15 hours	+ 26 hours (ml)	+ 30 hours (ml)	+ 50 hours
802	0	90	1	0
806	0	20	0	0
808	0	4	30	0
811	0	30	16	0
815	0	34	20	0
900	0	120	0	0

Efficacy based on 100 % removal of scoleces was:
Sheep: 27 out of 30 were free of scoleces and
Cattle: 8 out of 12 were free of scoleces at necropsy respectively.
Cestodes recovered from animals treated with FBZ at necropsy only had immature segments.

DISCUSSION

Efficacy of an anthelmintic against cestodes is classified as follows:–
Class 1: 100 % effective in at least 80 % of the treated flock
Class 2: 100 % effective in at least 60 % of the treated flock
Class 3: 100 % effective in at least 50 % of the treated flock

Table 3: THE ANTHELMINTIC EFFICACY OF FBZ AT A DOSAGE RATE OF 10 mg/kg BODY MASS AGAINST *MONIEZIA* SPP. IN CATTLE

Animal Number	Body Mass (kg)	10 % FBZ per animal (mg)	Scoleces Recovered
228	199	1990	0
229	227	2270	0
230	177	1770	12
231	148	1480	0
232	186	1860	0
233	159	1590	0
234	115	1150	3
235	234	2340	0
236	211	2110	0
238	322	3220	7
239	119	1190	4
240	213	2130	0

Class X: Ineffective.
Fenbendazole at a dosage rate of 10 mg/kg body mass in sheep obtained a Class 1 registration.

According to statistical analysis where there are 30 animals in a trial:—
For Class 1: 3 animals can still be positive after treatment.
For Class 2: 8 animals can still be positive after treatment.
For Class 3: 11 animals can still be positive after treatment.
Due to the small number of animals in the cattle trial, according to this classification, FBZ was ineffective although 8 out of 12 calves were cured.

According to statistical analysis where there are 11 animals in a trial:—
For Class 1: all 11 animals should be tapeworm free
For Class 2: one animal is allowed to be positive after treatment.
For Class 3: 2 animals are allowed to be positive after treatment.
Scoleces should be present in 4 of the 5 controls. In the sheep trial five out of five were positive. It seems as if the number of scoleces per animal and the volume of the tapeworms determine whether a scolex will survive the effect of the drug.

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CATTLE TICKS FROM THE WATERBERG DISTRICT OF THE TRANSVAAL

J. SCHRÖDER

ABSTRACT: Schröder J. **Cattle ticks from the Waterberg District of the Transvaal.** *Journal of the South African Veterinary Association* (1980) **51** No. 1, 27-30 (En) MSD Research Centre, Private Bag 3, 1985 Halfway House, Rep. of South Africa.

Macroscopically visible ticks were collected from the hides of 28 cattle slaughtered in pairs during a period of 14 months in the Waterberg District. In order of prevalence *Rhipicephalus appendiculatus, Rhipicephalus evertsi evertsi, Amblyomma hebraeum, Hyalomma marginatum rufipes, Ixodes cavipalpus, Hyalomma truncatum* and *Boophilus decoloratus* were recovered. Immature stages of the three commonest species constituted a major portion of the population for varying periods during the months April to September. These results are in general agreement with those of surveys done in the northern Transvaal, Natal and Rhodesia.

INTRODUCTION

The Ixodidae constitute a major obstacle to economic livestock production in South Africa. Direct harmful effects stem from the bite-wounds which adversely affect the host animal's production potential[15 17], and eventually render skins and hides unsuitable for tanning[5], and from intoxication via the saliva of a number of species. Ticks are also indirectly responsible for pathogenic effects in that they act as mechanical and biological vectors of many viral and protozoal diseases[7 17]. In the Bushveld regions of South Africa the wounds caused by the long mouthparts of *Amblyomma hebraeum* and *Hyalomma* spp. serve as sites of entry for the larvae of *Chrysomyia bezziana*[7].

Measures aimed at the control of tick infestations on livestock should be based on a knowledge of the biology of these arthropod parasites. Studies on the prevalence and seasonal incidence of ticks of cattle have been undertaken in Natal[4], the Northern Transvaal[11] and Rhodesia[9 10 12].

This paper reports the recovery of ticks during a 14 month period from the hides of cattle slaughtered in the Waterberg District.

MATERIALS AND METHODS

Trial location

The survey was conducted on a commercial cattle ranch 2 350 ha in extent, near Bulge River (approx.24°05'S, 27°35'E, Alt. 990–1 035 m) in the Waterberg District of the Transvaal. The vegetation in this region is classified as mixed Bushveld[1] and the mean annual rainfall is ca. 680 mm. During the trial period total monthly rainfall was recorded.

Experimental animals

Twenty-eight weaned grade Africander steer calves were slaughtered in pairs at 4–5-week intervals from 15th October 1975 to 19th November 1976 for endo-[14] and ectoparasite recovery. The calves slaughtered until 19th May 1976 were born during the November-December 1974 calving season, and those slaughtered from 16th June 1976 until the end of the trial were born during June to August 1975.

Husbandry

The trial calves were run with the main cattle herd on the ranch. All animals were plunge-dipped in chlorfenvinphos (Supona: Shell) at weekly intervals from 1st October 1975 to 31st May 1976, and from 13th October 1976 to 19th November 1976. In the intervening period dipping took place at fortnightly intervals.

Ectoparasite recovery

Immediately post mortem all macroscopically visible ticks on the head, feet and rest of the body of each animal were collected separately and preserved in either 10 % formalin or 25 % ethanol. From October 1975 to May 1976 the hides, feet and heads were scrubbed in scaldingly hot water, and the washings rinsed through a sieve with 38 mm apertures. This was discontinued from June 1976 onwards because it failed to remove immature ticks from the feet and from the ear canal. From July 1976 until the end of the trial each calf's ear pinnae were cut off and thoroughly scraped with a sharp knife, and the scrapings preserved for examination. Adult *A. hebraeum* and *Rhipicephalus evertsi evertsi* were identified macroscopically. All other ticks were identified under a stereoscopic microscope.

RESULTS

A total of 533 mm of rain was recorded during the 14-month trial period. The total numbers of ticks recovered are listed in Table 1.

Rhipicephalus appendiculatus constituted 59,4 % of all ticks recovered, and more larvae than nymphae and adults together were found. Larvae were particularly prevalent during April and July with none being recovered during November and from January to March. Nymphae occurred in low numbers virtually throughout the year, but increased during April, May, July and August. Peak numbers of adults were recovered during January, February, October and November 1976, while none were present during June to August. More male than females were found. The total monthly collection for this species is illustrated in Fig. 1.

The second most prevalent tick was *R. e. evertsi,* which accounted for 21,5 % of the total number of ticks recovered. Once again the number of larvae exceeded that of the nymphae and adults combined, and males were slightly more numerous than females. Small numbers of larvae were recovered sporadically; none were present during October and November 1975 and March, May, June and September 1976, while a marked peak occurred during October 1976. Nymphae were completely absent from March to August and were found only in low numbers during the other months. Adult ticks were recovered in variable num-

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Table 1: TOTAL NUMBER OF TICKS RECOVERED

Month	*A. hebraeum*			*R. appendiculatus*				*R.e. evertsi*				*H.m. rufipes*		*H. truncatum*	*B. decoloratus*	*I. cavipalpus*	Monthly Total
1975	N*	♂	♀	L	N	♂	♀	L	N	♂	♀	♂	♀				
October	11	6	5	0	0	0	0	0	6	19	6	2	4	0	0	0	
	24	24	15	1	1	0	1	0	1	7	4	11	1	0	0	0	149
November	7	11	0	0	2	0	1	0	7	2	3	4	2	0	0	0	
	3	9	1	0	0	0	0	0	2	2	0	3	1	0	0	0	60
December	6	8	1	5	0	2	2	4	0	2	2	1	0	0	0	0	
1976	0	8	0	3	0	1	3	6	0	0	4	6	2	0	0	0	66
January	3	7	1	0	6	10	15	2	8	0	1	6	5	0	0	0	
	22	12	1	0	0	12	15	2	0	12	7	5		3	0	7	162
February	3	8	0	0	1	26	28	0	0	10	12	7	8	0	0	0	
	8	11	5	0	1	55	83	4	1	17	20	36		6	0	8	358
March	6	5	0	0	0	0	2	0	0	4	4	2		2	0	0	
	5	4	1	0	0	0	2	0	0	4	4	1	0	0	0	0	46
April	10	8	2	578	29	2	1	0	0	3	3	0	1	0	0	0	
	1	4	2	135	24	0	2	1	0	8	6	1		1	0	0	822
May	2	1	0	3	28	1	1	0	0	6	3	0	0	0	0	0	
	4	2	1	22	7	0	0	0	0	10	3	0	0	0	0	0	94
June	4	1	0	0	7	0	0	0	0	3	2	0	0	0	0	0	
	1	0	0	10	1	0	0	0	0	0	5	0	0	0	0	0	34
July	6	0	0	192	37	0	0	0	0	2	3	0	0	0	0	0	
	0	2	0	9	5	0	0	6	0	0	1	0	0	0	0	0	263
August	0	2	0	4	11	0	0	1	0	0	0	1	0	1	0	0	
	2	0	0	0	18	0	0	1	0	0	0	0	0	0	0	0	41
September	1	1	0	0	1	0	0	0	1	0	0	0	0	0	0	0	
	3	3	0	4	14	2	1	0	0	0	4	0	0	0	0	0	35
October	6	15	5	2	7	5	2	170	18	3	1	1	0	0	0	0	
	6	22	6	0	4	16	8	141	3	6	1	3		3	0	0	454
November	8	10	1	0	0	86	65	24	0	2	4	5	2	0	1	2	
	4	21	8	0	14	112	42	1	1	5	6	2	0	0	1	0	427
Total	156	205	55	968	218	330	274	363	48	127	109	50	26	16	2	17	3011
		416			1790				647			123					

*L = Larvae, N = Nymphs, ♂ = Male adults, ♀ = Female adults

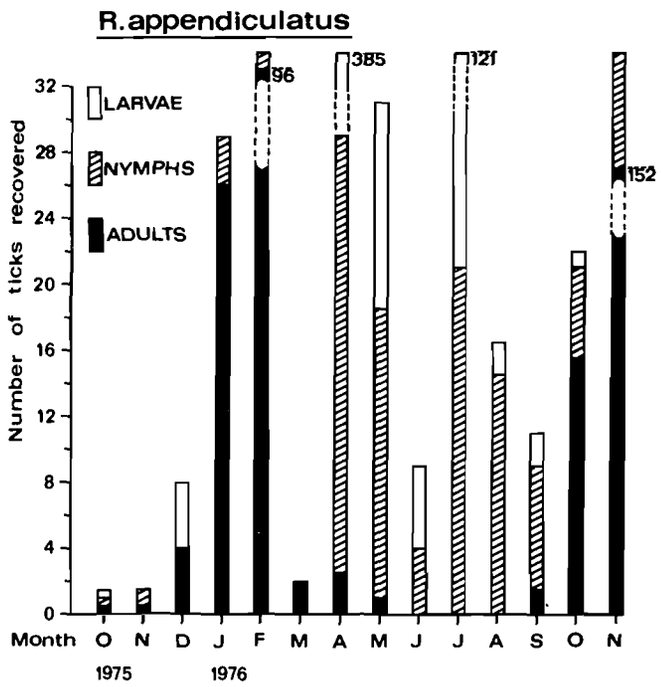

Fig. 1. Mean monthly collections of *R. appendiculatus* from two calves during a period of 14 months.

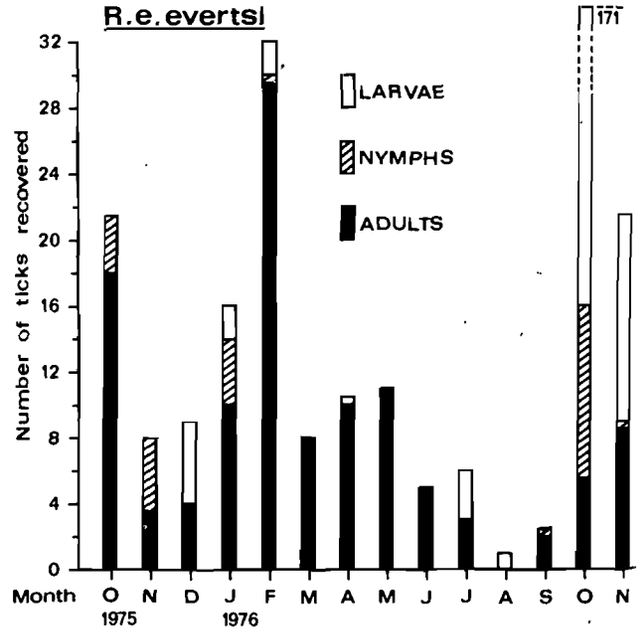

Fig. 2. Mean monthly collections of *R.e. evertsi* from two calves during a period of 14 months.

bers throughout the trial except during August 1976, when none were found. The largest numbers of adult *R. e. evertsi* were found on the two calves slaughtered during February 1976 (Fig. 2).

A. hebraeum was third in order of prevalence and made up 13,8 % of all ticks recovered. In this case males outnumbered females by nearly 4:1, and females were completely absent during June to September

1976. Nymphae were the only immatures found, and occurred during all months, displaying an increase in number during October and November and from January to April. More adult ticks were recovered from October to April than during other months (Fig. 3).

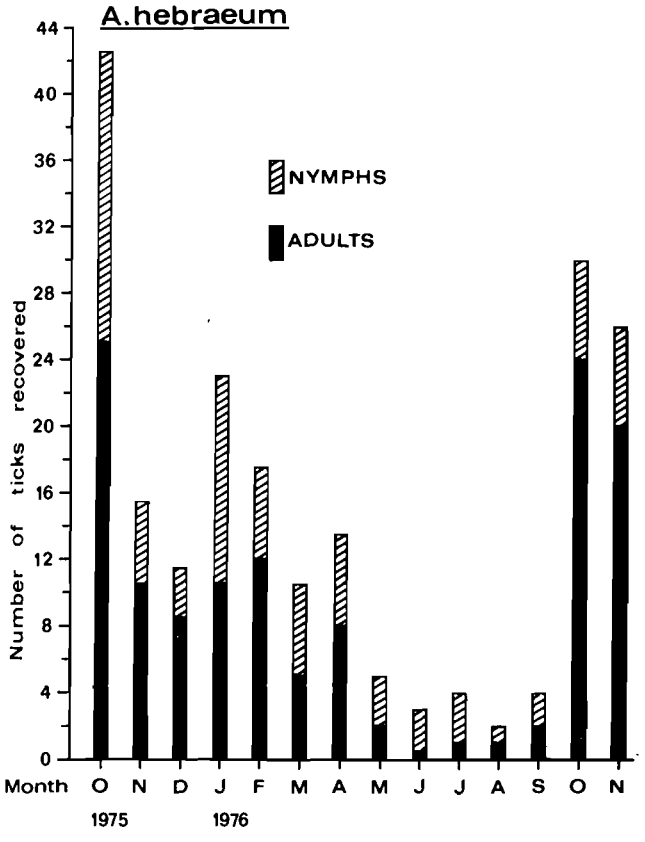

Fig. 3. Mean monthly collections of *A. hebraeum* from two calves during a period of 14 months.

Hyalomma spp. accounted for 4,6 % of the total tick recovery. *Hyalomma marginatum rufipes* made up 89 % of this number, with *Hyalomma truncatum* responsible for the balance. Only adult *Hyalomma* spp. were found, and they were completely absent during May, June, July and September.

A total of 17 *Ixodes cavipalpus* were found. They were all females, and were found on one of each pair of animals slaughtered during January, February and November 1976. One *Boophilus decoloratus* male was found on each of the calves killed in November 1976.

The distribution of ticks on the host animals did not differ from that previously described[4].

DISCUSSION

Survey methods

A variety of techniques has been employed in the collection of ticks from cattle in order to investigate their seasonal population dynamics. Baker & Ducasse[4] recovered immature and adult ticks from live cattle by combing the hair coat once a week. Jooste collected immature *R. appendiculatus* from pasture by blanket dragging once a week for one year[9], and adult ticks from cattle over a 2-year period, also at weekly intervals[10]. No mention is made of acaricidal treatments applied during the course of the abovementioned surveys.

Londt *et al.*[11] collected ticks at about 2-weekly intervals from 6 sites on 6 animals over a 13 month period at Nylsvley and treated the calves with an acaricide after each collection. The collection of ticks from the hides of slaughtered animals has since been refined by the use of a tick-detaching agent and vigorous scrubbing with wire brushes (I G Horak 1978 Faculty of Veterinary Science, Onderstepoort, personal communication).

It is statistically impossible to compare the results of experiments employing such widely differing sampling techniques. The results obtained, however, are so similar in most respects, that the differences possibly attributable to technique can probably be ignored.

Tick prevalence

If corrections are made to accommodate the differences in the numbers of trial animals and the lengths of the survey periods between this survey and that of Londt *et al.*[11] it will be found that 1,3 times as many ticks were collected at Nylsvley compared with Bulge River, during nearly the same calendar period. This similarity is remarkable if the ecological differences in vegetation and availability of moisture are taken into account. The Nyl River runs through the Nylsvley Reserve and the vegetation is classified as mixed Bushveld[1]. In addition, with the exception of *Rhipicephalus simus*, the same species were present at both localities, often in roughly the same percentage (Table 2).

The greatest differences in tick prevalence occurred in the cases of *A. hebraeum* and *I. cavipalpus*, which were relatively more abundant at Bulge River than at Nylsvley, and *H. truncatum*, which was relatively scarcer.

Table 2: COMPARATIVE TICK POPULATION COMPOSITIONS AT NYLSVLEY AND BULGE RIVER

Species	Percentage of total number of ticks recovered	
	Nylsvley	Bulge River
R. appendiculatus	69,08	59,45
R.e. evertsi	17,81	21,49
H.m. rufipes	5,07	4,08
H. truncatum	3,98	0,53
A. hebraeum	3,71	13,82
R. simus	0,22	0,0
B. decoloratus	0,12	0,07
I. cavipalpus	0,01	0,56

The results of the present experiment differ in both tick species and prevalence from those undertaken in Natal[4] and Rhodesia[10 12]. In Natal, *B. decoloratus* was the most prevalent tick (44,6 % of the total collection), followed by *R. appendiculatus* (31,6 %) and *R. e. evertsi* (21,2 %). *Rhipicephalus tricuspis* was also found in that survey, but no *H. truncatum* or *I. cavipalpus*. In Rhodesia, on the other hand, *R. e. evertsi* was found to be most abundant (42,7 %) followed by *B. decoloratus* (29,7 %), *H. rufipes* (9,2 %), *R. appendiculatus* (8,5 %) and *Rhipicephalus compositus* (7,6 %). *R. simus* and *Rhipicephalus pravus* (which were not found in the present survey) were also found in Rhodesia, but no *A. hebraeum*[10 12].

Seasonal incidence

In a broad sense the seasonal fluctuations in tick numbers in this survey corresponded to those found in Natal[4], Rhodesia[10] and at Nylsvley[11].

R. appendiculatus immatures represented more than 80 % of this species during the months April to September. The two peaks of larval activity in April and July are not quite as distinct as those described by Jooste for April and August[9], but correspond to the increased activity in April and May described by Londt *et al.*[11]. Baker & Ducasse had found this period of larval activity to be somewhat more extended in Natal[4]. Nymphae at Bulge river were active from April to August, as they had more or less been found to be at Nylsvley[11], and in Natal[4]. In Rhodesia they had been recovered in peak numbers in August and September[9]. The single peak of adult activity, which occurs during the months November to January, February or March[4 9–12] indicates that this species completes only one generation per year[12].

R. e. evertsi does not display this clear-cut seasonal variation in population composition, presumably because several generations can be completed in one year[12]. The significance of the peak of larval activity found at Bulge River in October 1976 is therefore questionable. Baker & Ducasse had found a peak in immature numbers to occur from January to April[4]. Adults in this survey were present throughout the year (except in August when they were completely absent) as they had also been elsewhere[10–12]. The increase in activity from October to February was not very distinct, but corresponded to that found at Nylsvley[11], and in Rhodesia[10 12]. In Natal the adults had been more prevalent from January to May[4].

No *A. hebraeum* larvae were collected at Bulge River, but they were found in low numbers from approximately December to May in Natal[4] and at Nylsvley[11]. While no peak of nymphal activity could be demonstrated at Nylsvley[11], they were found to be active from May to September in Natal[4]. This onset was 2 months later than that of the peak in the percentage of the population consisting of nymphae seen at Bulge River from March to September. The greatest numbers of nymphae in this survey were found from October to April. The summer peak of adult *A. hebraeum* activity corresponds to that described by Baker and Ducasse[4] and Londt *et al.*[11].

Although never occurring in great numbers, the relative abundance of *H. m. rufipes* from October to February at Bulge River is similar to that seen at Nylsvley[11], in Natal[4] and in Rhodesia[10 12].

Control

Cattle farmers rely heavily on chemical compounds for the control of cattle ticks. Various difficulties beset this practice. Firstly, in the stable immunological situation of tick-borne diseases prevalent in the major part of this country, the total eradication of ticks on any one farm is distinctly undesirable[17]. In the second place, the tick species prevalent here are widely divergent in their characteristics (particularly number of hosts and length of life-cycle). A dipping programme which in inadequate for the control of *R. e. evertsi*[12], and barely adequate for *R. appendiculatus,* subjects *Boophilus* spp. to such a high selection pressure that the development of resistance to acaricides poses a continuous threat[8 16]. Recently other species of ticks have also begun to display resistance to acaricides[3 13].

In view of the numbers of immature ticks found during the winter months in this survey, it would appear that fortnightly dipping is inadequate as a control measure, as has been stated elsewhere[8 12]. Dipping should take place at least at weekly intervals throughout the year despite the dangers of resistance to the acaracide.

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OWNER-PET RELATIONSHIPS – A KYNOLOGICAL STUDY

D.R. OSTERHOFF

ABSTRACT: Osterhoff D.R. **Owner-pet relationships – a kynological study.** *Journal of the South African Veterinary Association* (1980) **51** No. 1, 31-35 (En) Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The paper supplies the results of a first survey of its kind: a total of 46 questions were presented to dog owners mainly in the areas in and around Johannesburg, Cape Town, Bloemfontein, Pretoria, Windhoek, Salisbury, Port Elizabeth and Durban and the answers compiled. In this "kynological study", all aspects of the dog, his behaviour and relationship to man are involved.

It is clear that South Africans love and care for their animals and spend about R135 per year on their pets. Some indications of differences between the owners of dogs – Afrikaans- and English speaking persons, as well as new immigrants – became apparent, but more data are needed to make clear differentiations. Comparisons to other countries would also throw more light on the "pet owner problem".

INTRODUCTION

In a recent paper Feldman[1] investigated the pet owner psychology and the deliquent owner of pets (dogs and cats). He spoke of society as having a pet owner problem, not a pet animal problem and he listed the emotional needs which pet owners satisfy through their pets as: (1) friend and partner, (2) self-identity and self-esteem, (3) facilitation and catalysis, and (4) childhood development.

The present study excluded cats but basically had the same aim: to find out why people keep dogs. At the same time many other aspects were included in the survey.

MATERIALS AND METHODS

In 1976 Second Year Veterinary Students with a special interest in dogs, were provided with questionnaires compiled by the author and went from dog owner to dog owner presenting 46 questions. More than 600 persons were interviewed and the answers recorded as briefly as possible, using mainly the multiple choice system. The students contacted those dog owners close to their home areas, and also helped in the compilation of the answers. One could analyse the data further, possibly by computer, to discern regional, social and other differences.

RESULTS AND CONCLUSIONS

It is possibly incorrect to present the replies of dog owners as "results". Nevertheless, the answers are listed under the following headings:

(a) General
(b) Nutrition
(c) Care
(d) Reproduction
(e) Ethology
(f) Training
(g) Replacement and Euthanasia

It must be pointed out that not all the questions were answered by all dog owners; therefore the total figures will be given as percentages. In many cases two or three combinations of answers are possible; again the percentage figures will combine these answers in the best possible way.

The compilation is presented in abbreviated tables for easy reading.

(a) GENERAL

1. *How many dogs do you have?*

One only	53,6 %
Two dogs	29,4 %
Three dogs	8,6 %
Four dogs	5,6 %
More	2,8 %

More than half of the owners possess only one dog. There was a case of seven dogs in one household.

2. *What breed is your dog?*

Mongrel	28,0 %
Poodle	8,5 %
Toy Pom	6,0 %
Alsatian	5,3 %
Labrador Retriever	5.3 %
Dachshund	3,5 %
Fox Terrier	3,2 %
Maltese Poodle	3.2 %
Schipperke	3,0 %
Spaniel	3,0 %
Corgi	2,6 %
Boxer	2,2 %
Doberman Pinscher	2,0 %
Keeshond	2,0 %
Bulldog	2,0 %
Others	21,2 %

There were 51 different breeds of dogs, excluding mongrels, which were obviously the most popular, comprising 28 %. It is interesting to note that apart from the mongrels, the smaller breeds of dogs predominated, comprising 8 of the 10 most popular breeds.

3. *What is the sex of your dog?*

Male	51 %
Female	49 %

This equal division of the sexes was interesting.

4. *Where did you obtain your dog?*

Private individual	38,0%
Breeder	21,9%
As a present	12,2 %
Welfare society	10,9 %
Own breeding	7,4 %
Pet shop	5,8 %
Stray	1,6 %
Other	2,7 %

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It is fairly pleasing to see that about 12 % of the dogs were strays (Welfare society and strays).

5. *Do you read any books on dogs?*

General	45,0 %
Diseases	3,1 %
Training	5,2 %
Breeding	3,7 %
None	43,0 %

It is very surprising to see that nearly half of those interviewed read no books about their dog. Perhaps a huge drive should be undertaken to get people to learn more about their four-legged friends.

Twenty-one people read all the above-mentioned categories of books.

6. *How long have you had your dog?*

Less than one year	6,7 %
1 year	7,5 %
2 years	12,0 %
3 years	13,7 %
4 years	11,2 %
5 years	9,4 %
6 years	8,4 %
7 years	7,5 %
8 years	7,0 %
9 years	5,0 %
10 and more years	11,6 %

From these figures it can be seen that the peak occurred at 3 years which is equivalent to twenty-one human years. One dog was reported to be 17 years old.

7. *Could you imagine living without a dog?*

No	85,4 %
Yes	12,0 %
Not sure	2,6 %

Although 85 % of the people "could not imagine living without a dog", it is believed that this percentage would be even higher if the question was: "Would you like living without a dog?"

8. *Why do you keep a dog?*

Pleasure	29,3 %
Attachment	21,1 %
Protection	16,3 %
Always had dogs	11,1 %
For the children	9,9 %
Breeding	3,0 %
Loneliness	2,9 %
Showing	2,0 %
Dogs never let you down	4,0 %
Other reasons	0,4 %

It is interesting to see that, of those dogs whose owners were questioned, only 2,0 % were for showing purposes and 3,0 % for breeding. One person kept a dog because it was "amusing"!

9. *Give the number of people in the house:*

Adults	*Children*	*Servants*	*Dogs*	*Number*
1	0	0	1	10
2	0	0	1	14
2	1	1	1	11
2	2	0	1	14
2	2	1	1	29
2	3	0	1	10
2	3	1	1	24

There was a total of 117 of these combinations and the task of correlating the above four parameters with breed of dog and income of owner would be gigantic.

10. *Give an indication of the monthly income:*

Less than R250	2,8 %
R250 – R500	14,0 %
R500 – R750	18,8 %
R750 – R1000	23,2 %
over R1000	24,0 %
N/A	17,2 %

The 17 % given as N/A represents those people that felt that they should not give this information. The idea of this question was to correlate the income with the kind of dog kept.

(b) NUTRITION

11. *How often do you feed your dog per day?*

Once	63 %
Twice	34 %
More often	3 %

12. *Do you feed your dog mainly:*

Dog cubes	33,5 %
Fresh meat	21,8 %
Remains of meals	18,4 %
Tinned dog food	16,0 %
Dog's meat	6,5 %
Other (fish)	3,8 %

Very often there is a mixture of the different kinds of food given but the above percentages give a clear picture of the situation.

13. *Do you give your dog bones to chew?*

Every day	19,0 %
One a week	24,4 %
Several times/week	34,5 %
Seldom	12,8 %
Never	9,3 %

14. *Do you give your dog sweets?*

Never	56,5 %
Sometimes	38,6 %
Often	4,9 %

15. *How much money do you spend on your dog annually?*

The first part of the question regards the amount spent on food:

Less than R50	39,6 %
R50–R100	35,1 %
R100–R150	10,4 %
R150 – R200	5,2 %
More than R200	0,7 %

It is understood that the smaller breeds of dogs do not need as much food and that most of the dog owners interviewed possessed smaller breeds.

The question on the yearly expenditure on dog training was not answered in a satisfactory way: not enough data was available to make a statistical analysis. It seems that most dog owners train their dogs themselves.

To the veterinarians the most important question was the expenditure on veterinary services. It seems that dogs only come for treatment to the veterinarian if they

are really ill or injured, therefore the figures vary considerably:

Less than R10	30,2 %
R10 – R20	31,8 %
R20 – R30	20,2 %
R30 – R40	7,7 %
R40 – R60	3,8 %
More than R60	6,3 %

In one case the figure of R450 was given as the yearly veterinary costs.

Considering dog taxes it was quite difficult to obtain a clear picture. It seems that many dog owners do not pay tax. Of those who answered the question, 75,5 % gave the total spending as less than R10 and 24,5 % said that these costs would be more than R10.

Combination of all these costs indicates the expense of keeping a dog: 44,9 % of owners spend less than R100, 29,3 % spend R100 to R150 on their dogs, 12,2 % between R150 to R200 and 13,6 % more than R200.

(c) CARE

16. *Who cares most for the dog?*

The wife	55,9 %
The children	20,9 %
The husband	16,1 %
The servants	7,1 %

17. *How much time is spent on the dog?*

Less than half an hour/d	15,7 %
Up to one hour	26,6 %
One hour	16,6 %
Two hours	13,9 %
More than two hours	26,4 %
No time	0,8 %

18. *How often do you bath your dog?*

Once a week	12,5 %
Once a month	42,4 %
Sometimes	33,3 %
Never	12,0 %

It was quite obvious that the larger dogs were bathed less frequently than the smaller breeds.

19. *To whom is the dog most attached?*

Here many different opinions were encountered but it is clear that the wife who spends most of the time on the dog is also the favourite of the dog.

Wives preferred	42,0 %
Husbands preferred	26,8 %
Children over 15 years	16,0 %
Children under 15 years	14,1 %
Servants	1,1 %

20. *To whom is the dog most obedient?*

Husband	42,0 %
Wife	35,9 %
Children over 15 years	14,8 %
Children under 15 years	5,4 %
Servant	1,9 %

21. *If you go on holiday, what happens to your dog?*

Housed in kennels	30,0 %
Attended to by neighbours or friends	29,6 %
Taken along	17,6 %
Attended to by servants	20,0 %

22. *Do you have some ideas on the problem of dog faeces in streets?*

Responsibility of dog owner	67,8 %
Special areas to be provided	10,8 %
Special dog toilets to be built	6,6 %
Special areas prohibited to dogs	6,6 %
Undecided	8,2 %

23. *How often do you see the local veterinarian?*

Only when necessary	87,0 %
Regularly	10,8 %
Once a year	0,9 %
Never	1,3 %

These answers indicate the need for veterinary services.

(d) REPRODUCTION

24. *How often would you like to breed your bitch?*

Not at all	43,8 %
Once	36,8 %
As often as possible	11,2 %
Twice	8,2 %

The high percentage of people wanting to breed their bitches once is possibly due to the general belief that a bitch needs to have a litter of pups before it can be spayed. This, of course, is a fallacy, as any bitch can be spayed at an age of 5–6 months, regardless. This is supported by the fact that only 4 % of the owners actually read books on dogbreeding. Also, only 7 % of the people questioned breed their own dogs.

25. *Would you prefer to have your bitch spayed?*

Yes	68,9 %
No	31,1 %

Most preferred spaying because of the inconvenience of having pups. This is possibly due to lack of space, cost of keeping them or alternatively, disposing of them. Most people become attached to the pups and would end up keeping them.

The figures show that the cost of spaying bitches (R24,00 upwards) is justified.

Most of the remaining third were possibly people who wanted the pups from controlled breeding programs.

26. *Would you prefer to have your dog castrated?*

No	84,3 %
Yes	15,7 %

The figure of 84 % is surprisingly high as castration costs far less than spaying.

People are probably less worried about the breeding ability of their dogs as the pups would be the problem of the bitch's owner and not theirs.

Advantages of castration would be to stop the dog from wandering and also to avoid ill-feeling between the owners of the dog and bitch, should the bitch produce pups.

27. *When would you have your bitch spayed?*

After 1 litter	40,5 %
After 2 litters	10,9 %
Before breeding	36,5 %
Never	12,1 %

The 12 % who said they would never spay are probably people who specifically wish to breed pups. (Refer to question 24).

There is a possibility that some people feel it is unethical to spay their bitches, or wish to avoid possible deleterious consequences.

28. *How would you avoid pregnancies of your bitch?*

Lock up bitches when on heat	27,3 %
Send bitches to kennels when on heat	19,0 %
Use a preventative pill	16,9 %
Use other means	36,8 %

Approximately 1/5 send their bitches to kennels; here there is no fear of mating as the bitches would be locked up in a kennel. Obvious disadvantages are cost and transporting the animals to and from the kennels each time they come on heat. On the other hand, confinement at home is not absolutely foolproof, a member of the household may accidentally leave a door or window open. Also, visiting dogs may break down flower-beds, defecate on lawns and possibly dig under the fence, causing an added nuisance.

(e) ETHOLOGY

29. *Give the three most important characteristics of your dog:*

Faithful	45,4 %
Good natured	47,9 %
Intelligent	37,7 %
Affectionate to children	36,9 %
Vigilant	31,6 %
Obedient	30,5 %
Beautiful	11,9 %
Courageous	7,4 %
Docile	6,6 %
Strong	4,5 %
Aggressive	4,2 %

One could expect the ranking of aggressiveness to change in keeping with the possibility of certain forms of crime, such as burglary and rioting. Since 3 characteristics were often given, the percentages naturally do not add up.

30. *Do you allow your dog*

In the house/apartment	74,5 %
In the dining room during dinner time	37,1 %
In the car when visiting friends	36,1 %
To sleep on arm or easy chairs	30,5 %
To beg during meal times	13,8 %
To jump up on visitors	16,1 %

Again, the percentages do not add up because the one answer does not exclude the other.

The last two mentioned percentages are obviously low because the owners do not allow begging or jumping, but surely the dogs do not abide.

31. *Where does your dog normally sleep?*

Kitchen	29,8 %
Outside kennel	26,1 %
Bedroom	19,0 %
Anywhere outside	11,1 %
On your bed	11,0 %
Various places	3,0 %

In the last column all kinds of sleeping places are included – in one case the dog had its own bedroom!

32. *Does your dog show any peculiar behaviour?*

No	45,7 %
Afraid of bad weather and thunder	29,2 %
Attacks moving vehicles	14,6 %
Roams without the owner's knowledge	6.0 %
Other habits (gun shyness, moon shadows etc)	4,5 %

33. *Is your dog antagonistic towards various people or animals?*

Black people	33,7 %
White people	5,0 %
Cats	20,9 %
Other dogs	19,6 %
Other animals	8,5 %
Not at all	12,3 %

The answer "antagonistic to other animals" could be answered differently if the city dogs were more exposed to other animals. They usually see only cats, dogs and people in the towns.

34. *Has your dog ever bitten anybody?*

No	79,6 %
Yes	20,4 %

35. *Which of the bad habits of your dog worries you most?*

These answers provide a crosscheck to the answers given to No 32.

No bad habits	41,5 %
Roaming in the garden, destroying plants	10,2 %
Barking unnecessarily	10,1 %
Chewing shoes and furniture	7,2 %
Licking	5.4 %
Chasing cars	4,9 %
Snaps every now and then	4,0 %
Urinates and defecates in house	3,2 %
Roaming at large	2,8 %
Others	10,7 %

The chasing of moving vehicles is regarded as a peculiar behaviour by some and by others as a bad habit. Many of the vices are due to lack of training.

(f) TRAINING

36. *What exercise does your dog get?*

Active training	5,0 %
Walk with the owner	28,0 %
Run in the garden	61,7 %
Not much	3.2 %
Exercise on farm	1,2 %
Not stated	0,9 %

The main forms of exercise are mentioned but very often dogs get more than one form of exercise.

37. *Do you allow your dog to:*

Roam the street	9,5 %
Stay on your property only	79,9 %
Stay in enclosure only	5,8 %
On farm	1,1 %
Not stated	3.7 %

38. *Has your dog any special abilities – have you taught him to:*

Catch a ball	14,0 %
Hunt	4,4 %

Go into water	4,5 %
Walk on two legs	5,0 %
Fetch the newspaper	1,1 %
Jump	6,0 %
Open the door	4,0 %
Watch television	2,4 %
Beg	8,5 %
Show interest in flying things	5,8 %
Smile	4,4 %
Retrieve from water	0,1 %
Shake paw	2,1 %
Sit and shake paw	2,3 %
Fetch pine cones	0,1 %
A variety of abilities	32,4 %

39. *Has your dog had any form of formal training for:*

Domestic obedience	34,0 %
Show purposes	3,4 %
Protection	2,0 %
Attack	1,2 %
Tricks	0,2 %
Other, several together	3,0 %
No training	56,2 %

40. *By whom was your dog trained?*

Member of the family	48,3 %
Dog trainer	4,2 %
An outsider	1,1 %
Owner	0,2 %
Unknown	0,2 %
None	46,0 %

It is interesting to note that 96 % of all Alsatians received formal training.

41. *Which characteristics would you rank highest in dog training?*

Obedience	63,4 %
Intelligence	26,9 %
Courage	5,3 %
Strength	0,9 %
Aggression	1,3 %
Excitability	0,2 %
Not stated	2,0 %

42. *Do you take your dog to dog shows?*

Never	88,0 %
Sometimes	7,0 %
Once only	0,4 %
Regularly	4.6 %

43. *How do you punish your dog?*

Scolding	49,0 %
Ignoring the dog for some time	2,0 %
Beating with hand	21,0 %
Beating with strap/stick	6,0 %
Confinement	2,0 %
Never punish	2,0 %
Hit with newspaper	3,0 %
Different ways combined	15,1 %

(g) REPLACEMENT AND EUTHANASIA

44. *If your dog should die, would you get another one?*

Yes	83,9 %
Unsure	10,5 %
No	5,6 %

The results obtained from this question reflect how much man relies on dogs – the majority of people would get a replacement. Of the 5 % who said they would not get a replacement, many might change their minds, should their pet die. This 5 % is possibly those who have a cherished pet and feel that they are irreplaceable. Those who were unsure have probably never thought about it and may get a replacement should something happen to their pet.

45. *Would you have your dog put down?*

If the dog was in severe pain	37,4 %
If the dog could not move	35,4 %
If the dog was too old	18,9 %
No (dog should die naturally)	4,1 %
If the dog was no longer obedient	0,3 %

According to the above-mentioned, 72 % of people would not like to see their dog suffer unduly. Only 3 % would choose euthanasia if their dog had bitten a child. Should the bite be severe enough, court action could be taken against the owner and they could be forced to put the dog down. It should be interesting to note: 1,2 % of dogs had formal training for attack and 2 % had formal training for protection. Also, 1,3 % of owners gave aggression as a character trait. In most cases a child would have to cause extreme provocation to elicit a serious bite. Also interesting to note: 80 % of owners said their dog had never bitten anybody, while 20 % said their dog had.

46. *If you choose again what breed would you select?*

The same breed	71,7 %
Another breed (unspecified)	18,6 %
A different, specific breed	7,2 %
Not get another dog	2,5 %

In question 44, 5 % said they would not get a replacement, while in question 46, 3 % said they would not get another dog. Once again, a slight deviation comes to the fore in the double check.

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ESTIMATION OF LIVER MASS IN SHEEP

J.B.J. VAN RYSSEN

ABSTRACT: Van Ryssen J.B.J. **Estimation of liver mass in sheep.** *Journal of the South African Veterinary Association* (1980) **51** No. 1, 37-39 (En) Dept. of Animal Science, University of Natal, Box 375, Pietermaritzburg, Rep. of South Africa.

The total liver mass of live ruminants is frequently required in mineral nutrition studies. In healthy, well-fleshed sheep high correlations were obtained between liver mass and body mass. In 169 sheep older than 15 months the wet liver represented 1,13 ± 0,129 % and the dry liver 0,34 ± 0,036 % of the live body mass respectively, while in 51 young sheep (6–9 months of age) these values were 1,65 ± 0,135 % and 0,48 ± 0,062 % respectively. Some of the factors influencing these values are discussed. The dry matter content of the livers in healthy sheep – irrespective of the breeds used, their age, body mass or the portion of the liver sampled – showed little variation, being 29,5 ± 1,02 %.

INTRODUCTION

The liver is the primary site of copper storage in the animal body[7] and, therefore, in studies on copper metabolism attention is frequently focussed on this organ. Rate of copper accumulation and the response to prophylactic measures are usually based on changes in the copper content of the liver over specific periods of time[1 4 7 15]. In such instances liver biopsy samples can be taken[4 7 11] or a representative pre-experimental group can be slaughtered to obtain the copper status of the animal at the onset of the trial[10]. Liver biopsy samples may also be taken for the randomisation of groups based on copper concentrations in livers[11].

The total liver copper content is often required to calculate changes in the copper status of animals[3 13]. In experiments where liver biopsy samples are taken, total liver mass is estimated from body mass, assuming that liver mass is a relatively constant proportion[4 20] or ratio[3] of body mass or that liver mass is correlated with body mass[7]. Beck[3] calculated the ratio of fat-free dry liver mass to body mass as 0,00258:1 or 0,00274:1. Wynne & McClymont considered wet liver mass to be 1,5% of the live body mass of weaners and wethers[20]; while a figure of 1,6 % based on values obtained by Wallace[18], was used by Bremner and co-workers[4].

Although variations in the distribution of copper concentration in the livers of sheep[11] and cattle[6 16] were observed, Hogan and co-workers concluded that such variations in mature sheep were relatively small, and that liver biopsy samples were reliable enough for estimating copper status of the sheep[11].

The mineral concentration and total mineral content in the liver can be expressed either on a wet or a dry basis. To compare results from different laboratories these values have to be converted to a comparable moisture basis[5]. Values for the dry matter content of sheep livers of 20 % (lambs)[14], 25 %[5] and 33,3 %[8 17] have been quoted. A dry matter content of 33,3 % was observed for cattle[16] and this value has been previously used for different species of animals in general[8].

Information on the relationship between liver mass and body mass in sheep was obtained in the course of various trials conducted at the Faculty of Agriculture, University of Natal.

PROCEDURE

A total of 220 sheep (172 South African Mutton Merinos and 48 Corriedales) of various ages were slaughtered at the Pietermaritzburg Abattoir over a period of 4 years. Live body mass was obtained before slaughtering, after withholding food and water for 18 hours. The sheep carried 3–6 months of wool-growth at time of slaughter. Body condition varied widely within and between trials. Grading of the carcases was used as an indication of the condition of the sheep at slaughter. The breed, body mass, number of sheep per trial, approximate age at slaughter and carcase grades are presented in Table 1.

The livers were collected as soon as possible after slaughter, placed in plastic bags, sealed and transferred to the laboratory cold room. Livers were never in the cold room for more than 4 hours and were not frozen. The gall bladder and any adhering tissue were removed from a fresh liver before its mass was determined. Samples, in duplicate, were taken at random from different areas of the liver and placed in tared beakers for dry matter determination. At least 50 g of wet material was used per beaker and the samples were dried in a forced draft oven at 80° to 100°C for 4–5 days.

RESULTS

The relationships of liver mass to body mass of the sheep used, are presented in Table 2.

Table 1: LIVE BODY MASS, AGE AT SLAUGHTER AND GRADING OF SHEEP

Trial	Age	Body mass ±		Grading of carcases (number)			
	months	kg	SD	Super & Prime	Grade 1	Grade 2	Total
1	24	42,6	6,99	5	21	4	32
2	30–36	61,1	6,30	0	28	2	30
3*	15–20	37,4	4,92	10	29	9	48
4	24	48,9	5,55	0	1	29	28
5	18	33,1	6,54	7	16	8	31
6	6	32,4	7,75	Not graded			25
7	9	32,1	6,98	Not graded			26

* Corriedales in Trial 3, in all the other trials S.A. Mutton Merinos

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Table 2: LIVER MASS OF SHEEP AS A PERCENTAGE OF BODY MASS AND THE RELATIONSHIP BETWEEN LIVER MASS AND BODY MASS

Trial	Wet liver as a proportion of body mass % ± SD	Dry liver as a proportion of body mass % ± SD	Relationship of wet liver mass (Y in g) to body mass (X in kg) Regression	r
1	1,18 ± 0,11	0,352 ± 0,031	Y = 37,86 + 10,92 X	0,877
2	1,11 ± 0,11	0,324 ± 0,030	Y = 126,68 + 9,01 X	0,660
3	1,15 ± 0,12	0,342 ± 0,040	Y = 150,07 + 7,46 X	0,651
4	1,04 ± 0,15	0,294 ± 0,040	Y = 305,48 + 4,12 X	0,328
5*	1,14 ± 0,13	0,358 ± 0,039	Y = 109,27 + 8,47 X	0,786
6	1,60 ± 0,13	0,473 ± 0,072	Y = –151,31 + 20,67 X	0,916
7	1,65 ± 0,14	0,494 ± 0,052	Y = –68,65 + 18,72 X	0,954

* One sheep died of cardiovascular shock. Wet liver as % of body mass was 1,69%; not included in calculations

In sheep older than 15 months, liver mass as a percentage of body mass was found to be 1,13 ± 0,129 % for the wet livers and 0,34 ± 0,036 % for the dry livers. In the case of the younger sheep these percentages were higher, viz. 1,63 ± 0,135 % and 0,48 ± 0,062 % respectively. The correlation coefficients between wet liver mass and body mass were high, except in Trial 4, where a low correlation coincided with the low carcase grading, indicating poor body condition.

The dry matter content of the livers, presented in Table 3, remained remarkably constant over the whole range of body masses, ages and conditions, and only in Trial 4 slightly (though statistically not significant) lower values were recorded.

Table 3: THE PERCENTAGE DRY MATTER OF LIVERS

Trial	Dry matter %	SD
1	29,7	0,99
2	29,2	0,74
3	29,7	1,06
4	28,1	0,90
5*	30,2	1,06
6	29,6	0,82
7	29,9	1,55

* Liver dry matter of 22,4% of sheep which died from cardiovascular shock; not included in calculations

In order to establish to what extent sampling-site in the liver might influence results, and thus possibly explain the differences in dry matter content quoted in the literature, samples were taken at different sites in the livers of 10 labmbs. The results of the dry matter determinations of these samples are given in Table 4.

Table 4: AVERAGE DRY MATTER CONTENT AT DIFFERENT SITES IN THE LIVER

	Site of Sample* (N = 10) A	B	C	D	E	F	F-test
Dry matter % ± SD	28,9 1,20	28,0 0,98	28,3 0,99	28,6 0,86	28,7 1,02	28,0 1,03	Not significant

* A = centre of right lobe, top 1½ cm of diaphragmatic surface
B = centre of left lobe, 2 × 2 cm through lobe
C = combined sample
D = caudate process
E = 2 cm thick piece beneath A
F = 1½ cm strip of right border

Although values obtained from the centre of the right lobe of livers had a slightly higher dry matter content than the other sites, none of the differences were statistically significant.

DISCUSSION

In this investigation the wet liver constituted 1,63 ± 0,135 % of the total body mass of young sheep, a value which corresponded well with observations of others[18]. Wallace reported that the liver mass of sheep, expressed as a percentage of body mass, decreased from 2,01 % at 62 days to 1,25 % at 332 days of age[18]. The present results showed that liver mass can be expected to decrease further to 1,1 ± 0,13 % of body mass at approximately 15 months of age; this value then remained fairly constant into adulthood. Ishmael, Gopinath & Howell observed an average value of 1,14 % for Clun Forest lambs at 9 months of age[12]. No difference in liver mass, expressed as a percentage of body mass, was observed between the mature S.A. *Mutton Merinos* and the Corriedales though differences may exist in younger sheep between early and late maturing breeds.

The low correlation coefficient observed in Trial 4 between wet liver mass and body mass could indicate that body mass is not very reliable as a measure of liver mass if body condition is poor. Dick[7] pointed out that changes in the total liver mass of his experimental sheep tended to vary with body mass, irrespective of condition of the sheep. However, it was observed that the liver mass of young growing sheep would fluctuate more than body mass at both low and very high levels of nutrition[19]. The fat content of the livers of well-fleshed sheep was found to be below 5 %[27], but as high as 23 % in emaciated animals[19]. Under such conditions more reliable comparisons of the mineral status of animals may be obtained if mineral values are expressed on a fat-free liver basis.

Any condition causing oedematous or swollen livers will change the values of liver mass when expressed as a percentage of body mass. An oedematous condition of the liver was reported in sheep experiencing fascioliasis[9] and was observed in Trial 5 of this investigation in the sheep which died of cardiovascular shock. Ishmael *et al.* observed an average value of 1,96 % in sheep experiencing a haemolytic crisis as a result of copper toxicity as compared to the value of 1,14 % of their controls[12].

The percentage dry matter in the livers of sheep remained constant at 29,5 ± 1.02 %, irrespective of age,

breed, condition of sheep or sampling site in the liver. However, these values were different from values of 20 %[14], 25 %[5] and 33,3 %[8 17] reported for sheep, though none of these reports indicated the original sources of their values. Homogenized fresh bovine liver samples were found to contain 66,4 % moisture (33;6 % dry matter)[16].

In general, it can be concluded that the estimation of liver mass (dry or wet) from body mass can be fairly reliable, provided that normal, healthy sheep in good body condition are used and provided that very precise estimates are not required[15]. In fact, the use of copper concentration (mg/kg) in the liver of sheep as suggested by Abdellatif[1] may be adequate in veiw of the high correlations obtained between liver mass and body mass, and because of the wide variations in rate of copper accumulation in the livers of sheep receiving the same levels of copper supplementation[10 13 17]

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OBESITY IN A DOG, WITH SECONDARY HORMONAL IMBALANCE

LEA STOGDALE* and D.J. MOORE†

ABSTRACT: Stogdale L.; Moore D.J. **Obesity in a dog, with secondary hormonal imbalance.** *Journal of the South African Veterinary Association* (1980) **51** No. 1, 41-45 (En). Dept. Med., Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

Obesity in dogs is frequently encountered by veterinarians. The history, clinical and laboratory findings of an overweight dog are described. Overfeeding of an all-meat diet resulted in obesity, and subclinical nutritional secondary hyperparathyroidism. The obesity caused fatigue, decreased cardiac performance, respiratory embarrassment, skin lesions, prediabetes and increased glucocorticoid level. A balanced diet fed in limited amounts, and exercise, resulted in a marked loss of weight and an improvement in the dog's health. The practical control of canine obesity is discussed.

INTRODUCTION

Obesity is usefully defined as "an excessive accummulation of fat in the storage areas of the body, sufficient to impair body functions"[2 4]. Obesity in dogs is a frequently encountered problem by veterinarians[1 7]. In an English survey, an average of 35 % of dogs seen in 3 veterinary practices was judged to be obese. The average for spayed bitches was 68 %[3]. Obesity is more common with age, in females than in males, in neutered than in entire dogs, and in animals being fed non-commercial foods, and owned by obese, older people[7].

Factors which lead to obesity in dogs are largely undocumented. However, some of the predisposing causes are genetic predisposition; a low metabolic rate; neutering; the unrestricted feeding of highly palatable food; the feeding of high energy diets such as commercial dog food, biscuits, meal, and table scraps; and overfeeding by indulgent owers[1 5 7]. Primary endocrinologic imbalances, such as hypothyroidism, subclinical diabetes mellitus, insulinoma, hyperadrenocorticism, or hypopituitarism, only occasionally cause obesity[7]. The amount of tissue overlying the rib cage, determined by simple observation, and palpatation is the most practical method of assessing obesity in the dog[4]. The dog may be considered too thin if the ribs are easily palpated, and too fat if they cannot be seen and an appreciable layer of fat is felt. If the ribs cannot be palpated at all, the dog is considered grossly obese[7].

Obesity shortens the life of dogs and results in, or aggravates, various medical problems. These include cardiac and respiratory embarrassment, decreased hepatocyte function, decreased resitance to viral infections, osteo-arthritis, dermatological diseases, heat intolerance, and endocrinological disorders[1 3 4]. This report describes the clinical and laboratory findings of an obese dog with no other apparent primary disease. The practical control of obesity in dogs is discussed.

CASE REPORT

History

An 8-year-old Fox Terrier spayed bitch, was receiving veterinary attention for a skin problem. The dog had been overweight for at least 5 years. She had been allowed to become a very fussy eater, accepting only prime quality steak and refusing all other food. The obese owner fed the dog excessive amounts of meat but was concerned that she did not eat sufficient food. The patient had suffered from sterile pustules and furuncles for 3 years. At irregular intervals bullae and furuncles developed in the skin of the ventral abdomen, especially in the inguinal region. These measured from 1–2 cm in diameter, and would eventually ulcerate. Histological examination of an ulcerated furuncle showed a chronic dermatitis with infiltration of plasma cells and histiocytes. Numerous cream-coloured nodules, also present in the abdominal skin, were histologically identified as epidermal cysts. The dog had a mass of 27 kg, 3 months previously, but this had increased to 37 kg at the time of presentation. With exercise, the dog tired easily. When admitted she was bright and alert, and was drinking a normal amount of water.

Physical examination

The patient was hospitalized for 5 days in order to perform a number of tests. The dog's habitus was normal, and she had a pleasant nature. She was grossly obese, weighing 36,9 kg (Fig. 1). She walked normally, but was lethargic, refused to run, and sat down whenever the opportunity presented itself. There was no evidence of muscle wasting, when the muscle mass was considered in relation to her ideal size, rather than to her actual obese size. The fat was evenly distributed (Fig. 1). All the visible mucous membranes (conjuctiva, oral and vulval) were severly congested. The dog panted

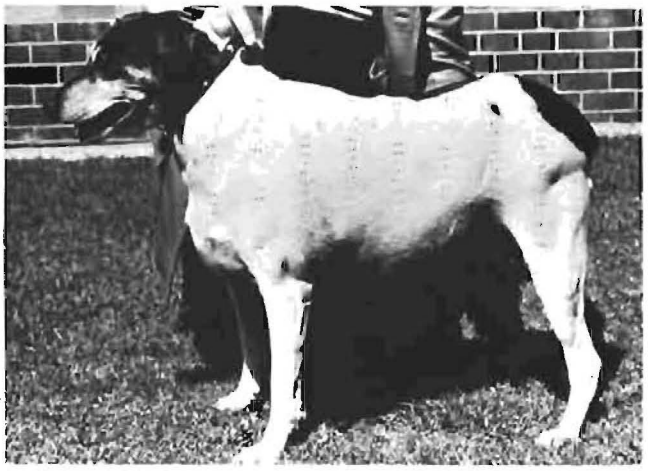

Fig. 1. The patient, showing the even distribution of the fat, and no evidence of muscle wasting.

*Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.
†Veterinary Hospital, P.O. Box 46149, Orange Grove, 2119, Johannesburg, Republic of South Africa.

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continuously and showed distress when her mouth was closed. The bronchial and vesicular lung sounds were normal. The heart rate was 120/min at rest. The heart sounds were normal but muffled. All other clinical findings were normal. The patient refused the commercial dog food offered to her during her 5 day stay in the hospital.

Conclusion: all the significant clinical findings were compatible with a diagnosis of obesity.

Dermatological examination

The skin was thin and supple. There was no increase in pigmentation. The hair coat was clean, and the hair was normal in appearance over most of the body. Over the ventral abdomen and inguinal region, the hair coat was thin, tending to alopecia. The thinning of the hair coat did not affect the genital region (Fig. 2). On the ventral abdomen and inguinal regions, there were numerous pinkish papules, 2–5 mm in diameter. These papules contained creamy inspissated material. Some of these papules had ulcerated. Many comedones (raised papules, 1 mm in diameter and black in colour) were present in these areas (Fig. 2). There was no significant bacterial growth on the culture media from swabs taken from these papules. Skin scrapings were negative for demodectic and sarcoptic mange mites.

Conclusion: the alopecia and papules were considered to be due to friction and reduced cutaneous blood flow, secondary to the obesity.

Laboratory findings

Blood samples were collected on Days 1, 2 and 3 of hospitalization. The results of the tests performed are presented in Table 1. The data was interpreted as follows:

Fig. 2. The ventral abdominal area affected by numerous papules and hair thinning.

(a) The erythron was normal.
(b) the neutrophilia, lymphopaenia and eosinopaenia were indicative of an increased glucocorticoid level.
(c) The protein electrophoresis showed small rises in alpha 1 and alpha 3 globulins. This suggested active cell damage. The small rise in beta 2 globulins suggested a chronic process. The liver and skin pathology could account for these changes.
(d) The fasting blood glucose values were at the upper limit of normal.

Table 1: CLINICAL PATHOLOGY RESULTS

Parameter, units	Day 1	Day 2	Day 3	18 months	Normal values*
Haemoglobin, g/100 mℓ	17,9	16,6	17,3	18,5	12–18
Red cell count, 10^6/mm^3	7,53	7,00	7,43	7,97	5,5–8
Haematocrit, %	56,8	51,9	54,3	61,4	37–55
Mean corpuscular volume, cu μ	74	73	73	76	60–77
White cell count, per mm^3	16 800	12 700	16 800	17 600	6–17 000
Neutrophil, %	85	85	96	72	60–77
Lymphocyte, %	12	13	1	17	12–30
Monocyte, %	2	2	3	3	3–10
Eosinophil, %	1	0	0	8	1–10
Basophil, %	0	0	0	0	rare
Total serum proteins, g/100 mℓ	7,3			6,7	5,9–6,7
Albumin, g/100 mℓ	3,47			3,18	3,0–4,8
Alpha 1 globulin, g/100 mℓ	0,64			0,69	about 0,3
Alpha 2 globulin, g/100 mℓ	0,26			0,34	about 0,3
Alpha 3 globulin, g/100 mℓ	0,91			0,69	about 0,3
Beta 1 globulin, g/100 mℓ	0,26			0,17	about 0,2
Beta 2 globulin, g/100 mℓ	0,64			0,69	about 0,3
Beta 3 globulin, g/100 mℓ	0,64			0,52	about 0,9
Gamma globulin, g/100 mℓ	0,46			0,42	about 0,9
A/G ratio	0,90			0,90	0,9–1,2
Blood glucose, mg/100 mℓ	100	109	81	92	70–100
Cholesterol, mg/100 mℓ	300	250	190	199	140–250
Sodium, mEq/ℓ	144,7			146	137–149
Potassium, mEq/ℓ	4,0			4,5	3,7–5,8
Phosphate, mg/100 mℓ	5,60			2,8	4,0–9,0
Calcium, mg/100 mℓ	11,8			9,7	9,4–12,2
Calcium × Phosphate product	66			27	40–60
BUN, mg/100 mℓ	16			16	15–25
SGPT, mU/mℓ	42			26	6–25
SAP, mU/mℓ	493			468	50–122
BSP, % retained at 30 minutes	3				0–5

* Normal values for the Clinical Pathology Laboratory at the Faculty of Veterinary Science, University of Pretoria.

(e) The fasting cholesterol value was initially above normal, but there was a significant decrease after 1 and 2 days of not eating. This suggested that the hypercholesterolaemia may have been due to the all-meat diet.
(f) The sodium and potassium levels were normal.
(g) The calcium and phosphate levels were within the normal ranges, but their product was 66. This was above the normal value and is likely to result in calcium phosphate precipitation in the tissues. Considering this result, and the history of an all-meat diet, it was suspected that subclinical nutritional secondary hyperparathyroidism was present.
(h) The kidneys appeared to be functioning normally, as the urinalysis, blood urea nitrogen (BUN), and the serum phosphate levels were normal.
(i) The slight rise in serum alanine transaminase (SGPT) indicated a small amount of active liver necrosis. However, the normal bromsulpthalein retention (BSP) showed that the conjugation and excretory functions of the liver were normal.
(j) The raised serum alkaline phosphatase (SAP) level may have been associated with an increase in osteoblastic activity. The BSP value indicated normal secretary function of the liver. Thus, the increased SAP level may have been associated with subclinical nutritional secondary hyperparathyroidism.

Special examinations

The urine output was measured over two 24 hour periods. The average urine volume for 24 hours was 1 225 mℓ which is within the normal range of 1–2 mℓ/kg/h: 887–1 777 mℓ/24 h for this animal.

Radiographs of the head were obtained in order to assess the degree of mineralization of the skeleton. The lamina dura dentis appeared to be normally mineralized, and there was no significant decalcification of the mandible or maxillae.

Blood was collected at midday for serum thyroid hormone level assay (Table 2). The patient's T_4 and T_3 levels were within the normal range.

An intravenous glucose tolerance test (IVGTT) was performed. The patient, and a normal control dog, were fasted for 16 h. A fasting blood sample was collected, and then 0,5 g/kg glucose was injected intravenously. Blood samples were collected at 5, 15, 30, 45, 60 and 90 min post-injection. The blood samples were analysed for glucose within 5 min of collection, using the glucose oxidase method. The results are illustrated in Fig. 3. The fractional turnover rate of glucose, k_1 was calculated using the standard formula[6]:

$$k\,\%/\text{min} = \frac{69{,}3}{T½}$$

Table 2: SERUM THYROID HORMONE LEVELS

Parameter, units	Day 3	Normal values*
Serum thyroxine, T_4, n mol/ℓ	19,3	18–28
Serum triiodothyronine, T_3, n mol/ℓ	1,1	0,6–1,0

* Normal values for the Clinical Pathology Laboratory at the Faculty of Veterinary Science, University of Pretoria.

Table 3: CORTICOTROPIN STIMULATION TEST

	Serum cortisol levels, ug/dℓ	
TIME	Patient	Normal values*
Resting, 0 hour	7,8	unstressed 1,0–2,5 stressed 2,5–12,5
2 hours	14,5	up to a 3 fold increase
4 hours	16,5	up to a 3 fold increase

* Normal values for the Clinical Pathology Laboratory at the Faculty of Veterinary Science, University of Pretoria.

T½ is the time required for the blood glucose concentration to fall by one half. The fractional turnover rate of glucose in normal dogs is 2,76 ± 0,91 %/min[6]. Our own experience is that normal dogs have a k value greater than 2,5 %/min. The fractional turnover rate in the control dog was 4,62 %/min, while the value for the patient was 2,04 %/min. This indicated that the patient was a prediabetic, probably due to obesity-induced tissue insulin resistance.

A corticotropin (adrenocorticotrophic hormone, ACTH) stimulation test was performed on Day 4 of hospitalization, by administering 1 mg tetracosactide* (synthetic corticotropin) intramuscularly. Blood samples were collected pre-injection, and at 2 and 4 h post-injection. These were assayed for the serum cortisol levels by the radioimmunoassay method (Table 3). The resting cortisol level was in the range for dogs under stress or suffering from a disease other than hyperadrenocorticism. The patient appeared calm and unfrightened at the time of the pre-injection blood sample collection. The rise in serum cortisol level in response to the administration of synthetic corticotropin was within the normal range.

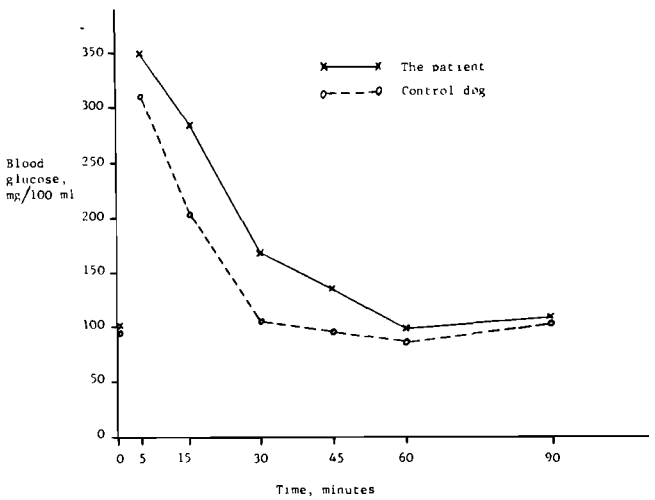

Fig. 3. Intravenous glucose tolerance test, in the patient and a normal dog, using a glucose dose of 0,5 gm/kg.

*"Synacthen Dept", Ciba-Geigy (Pty) Ltd.

Diagnosis

The final diagnosis was primary obesity, due to owner mismanagement. The obesity resulted in the secondary problems of decreased cardiac performance, respiratory embarrassment, exercise intolerance, skin lesions and hair coat thinning, mild active liver necrosis, prediabetes, and increased serum glucocorticoid level. Subclinical nutritional secondary hyperparathyroidism was suspected to be occurring, due to the all-meat diet.

Advice to the owner

Based on the above results and conclusions, the owner was advised to feed the dog a balanced diet at a rate of not more than 50 gm/kg of tinned dog food or fresh meat. The ideal weight of the dog was estimated to be approximately 20 kg. As the owner planned to feed the dog a mixture of fresh beef steak, and canned dog food, he was advised to feed a maximum amount of 1 000 gm/day, and to add a teaspoon of calcium gluconate powder to the food daily. Increasing the dog's exercise was also suggested. No other treatment was prescribed.

Follow-up examination

Eighteen months after the initial examination and hospitalization, a follow-up examination with blood tests was arranged. During the interim period, the dog had been fed daily on 250 gm beef steak, 225 gm tinned chicken dog feed, some slices of polony sausage, and a teaspoon of calcium gluconate. She had been walked 1½ km each day.

Clinical findings were that the patient was lively, active and alert, but still fat. She now weighed 25,5 kg; a loss in mass of over 10 kg. The ventral abdominal skin still showed thinning of the hair and a few papules, but she had not suffered from any furuncles since she had commenced losing weight. The visible mucous membranes were moderately congested. The dog panted continuously, but was not distressed when her mouth was closed. The heart rate was 120/min. Generally, there was a significant improvement in the dog's condition and demeanour which was attributed to the weight loss.

Blood was collected for clinicopathological evaluation (Table 1). The significant findings were:

(a) The erythron and leukron were normal, including the distribution of the leukocytes.
(b) The protein electrophoresis was very similar to the previous one. The small rises in alpha 1, alpha 3, and beta 2 globulins may have been a result of ongoing skin pathology.
(c) The fasting blood glucose value was normal, probably due to the disappearance of the prediabetic state with weight loss.
(d) The fasting cholesterol level was normal, probably due to the more balanced diet.
(e) The serum calcium level was normal, while the serum inorganic phosphate level was subnormal, as was the calcium phosphate product. This may be attributed to the balanced diet and the daily addition of calcium gluconate to the food.
(f) The SGPT level was normal, indicating that the liver necrosis had ceased.
(g) The SAP level was markedly raised, probably indicating an increased osteoblastic activity.

The 10 kg loss in mass and the balanced diet had resulted in a marked improvement in the dog's physical condition and general health. The obesity, decreased cardiac performance, respiratory embarrassment, and the skin lesions, were all still present, but to a lesser extent. The prediabetes, the increased glucocorticoid activity, and the subclinical nutritional secondary hyperparathyroidism all appeared to have been resolved.

DISCUSSION

That obesity is not good for the medical health and longevity of dogs is a well accepted concept, but it is rarely demonstrated objectively. This case illustrates the wide-ranging ramifications of obesity in a dog. In the patient described in this report, imbalances were evident in the endocrine system (prediabetes and increased glucocorticoid level), the nervous sytem (lethargy and inactivity), the cardiovascular system (decreasec cardiac performance and peripheral circulation), the respiratory system (increased respiratory rate), the liver (mild hepatocyte damage), and the skin (papules, furuncles and partial alopecia). In the obese dogs, other organs which may be affected include the urinary system (decreased tubular function due to fat deposition), the genital system (decreased fertility), the pancrease (an increased incidence of diabetes mellitus), and gastro-intestinal system (constipation and flatulence), the skeletal system (osteo-arthritis), and the immunologic system (decreased resistance to viral infections)[47].

Mass loss in dogs requires the active and willing cooperation of the owner, and of all the other people responsible for feeding the dog. Slow, steady, but significant weight loss is the approach best accepted by both the owner and the animal. This is achieved by reducing the dog's caloric intake below the maintenance level. A moderate amount of exercise each day makes the dog feel better, convinces the owner that he/she is actively participating and encourages compliance with the dietary instructions, and slightly increases the energy expended by the patient. The aim of the dietary formulation is to reduce the caloric intake to 50–60 % of the maintenance requirements of the dog *at its ideal mass*[47]. On such a diet, the time required for the dog to reach its ideal weight is 2–3 months. To this end, foods low in energy should be fed. The easiest food for the owner is, undoubtedly, the commercially prepared obesity diet. However, this becomes very expensive for medium or large sized dogs. A home prepared reducing diet containing ½ kg lean meat, chicken or fish, 2 cups of cottage cheese, 16 cups of carrots, beans or cabbage and 6 teaspoons of dicalcium phosphate may be fed at a rate of approximately ½ kg per 10 kg of *ideal body mass*[7]. Alternatively, good quality canned dog food (without cereal) can be mixed with uncooked or lightly boiled cabbage or lettuce. This latter combination is economical and easy to prepared. The canned food should be fed at a rate of 40–50 gm/kg/d or one 350 gm can per 7–8 kg of *ideal body mass*. The amount of cabbage or lettuce should be at least twice the bulk of the canned food. To prevent the dog from continually worrying the owner or begging for food, and from going "garbage can hunting", the food should be divided and fed 2–3 times a day.

An alternative approach to weight loss in the obese dog, is hospitalization and starvation. As long as minerals, vitamins, and free access to water are provided

daily, and the animal has no endocrinologic or metabolic disorders, there are no medical dangers to this approach. Approximately 6 weeks is required to decrease the dog's weight by 25 %[7]. Obesity can be safely and satisfactorily treated by this approach. Due to the requirement of prolonged hospitalization and its associated cost, and the nature of the method, however, this solution is generally unacceptable to owners.

The use of appetite suppressive drugs is sometimes suggested by owners. Although these have not been shown to be effective in dogs[7], their use as an expensive placebo, along with dietary restriction may encourage owner compliance. Similarly, the use of thyroxin or mixed hormone tablets are not indicated in endocrinologically normal but obese dogs. Owners frequently will follow dietary instructions more accurately if they think their pet has a "gland problem" and have to give it tablets twice a day.

As owner cooperation and enthusiasm are essential to the success of any attempt to reduce the weight of a dog, the owner must understand the many dangers of obesity to their much-loved pet. The maxim "you are killing your dog with kindness" is most useful. Once owner cooperation has been obtained, it must be made clear that no snacks or scraps are to be fed. Weigh the patient, record the mass, decide on the ideal mass, and on the time required to reach this mass. Formulate the diet to be fed to the dog, with the owner. Discuss the method of preparation, the storage, the amount to be fed, the frequency, and the cost. Write everything down in the dog's hospital record, and on an instruction sheet for the owner. Have the owner determine the mass of the dog once a week and record the result on a graph. Veterinary revisits are essential at least once a month, firstly, to encourage the owner to comply and persist with the mass reduction programme, and secondly, to check on the problem that the animal was, invariably, presented with. If inadequate mass reduction is being achieved, reduce the caloric intake by a quarter. If the dog is worrying the owner for more food, increase the bulk fed. Once adequate reduction in mass has been achieved, the diet can be increased to approximately 80–100 gm/kg/d of canned dog food, or 25 gm/kg/d of dried dog food. Regular weighing of the dog should be continued.

Obesity in dogs is unhealthy, unnatural and unnecessary. It is to the advantage of the animal, the owner and the veterinarian, if it is firstly avoided, or secondarily controlled.

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THE DIAGNOSIS AND TREATMENT OF CANINE HYPOTHYROIDISM

LEA STOGDALE

ABSTRACT: Stogdale, Lea. **The diagnosis and treatment of canine hypothyroidism.** *Journal of the South African Veterinary Association* (1980) **51** No 1, 46-48 (En) Dept. of Medicine, Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

Hypothyroidism in dogs should be diagnosed by integrating the history, clinical symptoms and thyroid hormone assay. Affected dogs have a slow onset of decreased activity, cold intolerance, and skin changes of thickening and mild hyperpigmentation. The hair coat becomes dry and coarse, and alopecia develops in friction areas. The non-specific clinico-pathological changes of a mild anaemia and hypercholesterolaemia do not occur consistently. The easiest and best documented thyroid hormone assay is serum thyroxine (T_4). The most accurate diagnosis is made using either the thyrotropin (TSH) stimulation test or thyroid gland biopsy. The condition is controlled by thyroxine replacement therapy at a dose rate of 30–50 mcg/kg/day in 2 divided doses, for the life of the dog.

HISTORY AND CLINICAL SYMPTOMS

Hypothyroidism in dogs is an endocrine deficiency that has a low incidence. It is frequently diagnosed solely on the basis of weight gain. However, this finding, in the absence of other symptoms, is insufficient to warrant such a conclusion. The condition has an insidious onset. Typically it manifests clinically with some of the following signs: decreased activity, leading to lethargy; increased weight with a normal appetite; intolerance to cold; skin thickening and mild hyperpigmentation; and a dry, coarse hair coat with alopecia. The alopecia is most marked on the friction areas of the chest, flanks and thighs. The skin and hair coat changes are bilaterally symmetrical, and non-pruritic (Figs. 1, 2, 3 & 4). Bradycardia is an inconsistant finding[2].

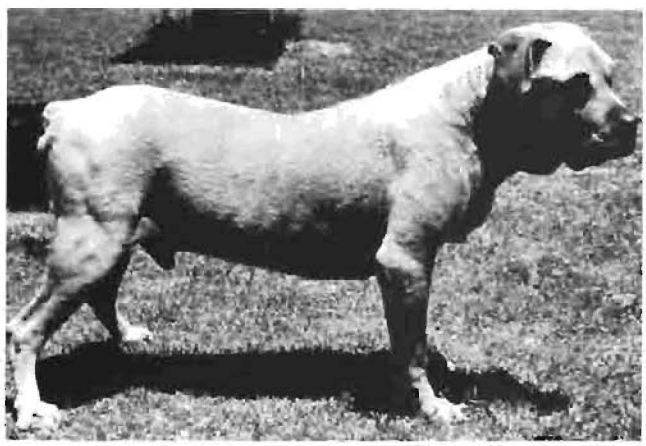

Fig. 1. Mixed breed dog, male, 6 years old suffering from hypothyroidism. The hair coat was course and dry. There was no weight gain.

The most common differential diagnoses for generalized weight gain leading to obesity, which, which must be considered are overfeeding, underexercising and agonadism (neutering). Other uncommon causes of obesity include hyperadrenocorticism, hyperinsulinism, the hypothalamic syndrome, and dystropia adipogenitalis.

CLINICAL PATHOLOGY

Haematological examination and the serum cholesterol level are frequently used to support a diagnosis of hypothyroidism. Unfortunately, the findings are neither consistent, nor specific. A mild nonregenerative normochromic anaemia occurs in 25–30 % of hypothyroid animals.[4] Other causes of this type of anaemia include myelophthisic (e.g. primary or secondary bone marrow neoplasia), aplastic, (e.g. ehrlichiosis, oestrogen toxicity), chronic infections, neoplasia, and uraemia. Generally, these conditions result in weight loss, and other clinical symptoms such as depression, haemorrhage, vomiting, and so on.

Table 1: NORMAL CHOLESTEROL VALUES ACCEPTED BY OUR LABORATORY

Animal and diet	mg/dℓ	mmol/ℓ
Dogs, low fat diet (dog pellets, meal, etc.)	130–210	3,4–5,4
Dogs, moderate fat diet (dog pellets and table scraps, or fresh meat)	150–250	3,8–6,5
Cats, low or moderate fat diet (cat pellets, or fish, or fresh meat)	95–130	2,4–3,4

Serum cholesterol levels are dependent upon the animal fat content of the diet eaten by the animal (Table 1). Serum cholesterol levels are only elevated in 60 % of hypothyroid dogs[4]. Hypercholesterolaemia is not specific for thyroid hormone deficiency, but also occurs with diets high in animal fat, hyperadrenocorticism, diabetes mellitus, the nephrotic syndrome, and some liver disorders. Rare diseases that cause hypercholeste-

Fig. 2. A mixed breed dog, female, 8 years old, showing a dry, course hair coat, and alopecia and hyperpigmentation in the axilla and flank regions.

rolaemia include idiopathic hyperlipidaemia, biliary obstruction, and von Gierke's disease (Type 1 glycogen storage disease).

SPECIFIC THYROID GLAND TESTS

As there appears to be considerable confusion over the various assays for thyroid hormone levels, each test is described, and its application is delineated.

Protein bound iodine (PBI) is not an accurate indicator of thyroxine levels in the dog due to (a) considerable non-thyroxine iodine bound to plasma proteins, (b) the high concentrations of inorganic iodides in canine serum and, (c) the frequent use of medications that contain iodine. All these factors falsely elevate the PBI reading. PBI has now been replaced by tests that more accurately assay serum thyroxine[4].

Serum thyroxine or T_4 may be assayed by competitive protein binding (CPB) or by radio-immunoassay (RIA). These assay techniques measure the total (bound and unbound or active) T_4 present in the patient's serum. These T_4 tests have been shown to be quite sensitive in detecting changes in circulating T_4 levels in experimentally produced hypothyroidism in the dog, i.e. simple or uncomplicated hypothyroidism[1,4] (Table 2).

Table 2: SERUM THYROXINE RANGES FOR NORMAL AND HYPOTHYROID DOGS

	T_4 CPB	T_4 RIA
Dogs, normal	1,5–3,6 µg/dℓ	1,2–3,6 µg/dℓ
Dogs, hypothyroid	0–1,7 µg/dℓ	0–1,0 µg/dℓ

µg/dℓ = micrograms per 100 mℓ
Note that the T_4 ranges from normal and hypothyroid dogs overlap

Serum triiodothyronine of T_3 may be measured by a radio-isotope column retention test. In man, values in hypothyroid patients greatly overlap those found in euthyroid subjects. Values for dogs and cats, by this technique, are lacking. A new method which gives satisfactory results utilizes a specific labelled anti-T_3 antibody. This gives an absolute result which is measured in nanograms. The normal range for the dog is 48–154 ng/dl. Only rarely does the T_3 result give any additional information over that obtained by the T_4 results[1,3,4].

Thyroid binding globulin (TBG) binds 70–80 % of the throid hormones and therefore the level of TBG influences the amounts of T_4 and T_3 which are unbound and active. The influence of alterations in thyroid binding proteins has not been thoroughly investigated in dogs and cats. The thyroxine resin sponge uptake test (RSU) gives an indirect evaluation of the TBG binding sites available for T_4 and T_3. It does not assay thyroxine in the patient's serum; it is an indirect estimation of the unbound T_4 concentration. If the unbound serum T_4 is low, then the RSU result will be subnormal. The RSU test is not sensitive to changes in thyroid gland function in the dog[3,4].

Free thyroxine index or T_7 is a reflection of the free, unbound T_4 in the serum. It is derived by multiplying the result of the RSU by the T_4 CPB. Due to the unreliability of the RSU test in dogs, this index is unsatisfactory[3,4].

Fig. 3. A mixed breed dog, female, 10 years old, that had gained weight over the previous 4 months. Her hair coat was rough, and there was alopecia and hyperpigmentation in the flank area.

Tyrotropin hormone response test or TSH stimulation is the most accurate indicator of thyroid gland function, as it provides results that can be readily interpreted. The test is performed by collecting a blood sample for serum, injection 10 IU thyrotropin intravenously between 08h00 and 09h00, and then collecting another blood sample 8 h later. In normal dogs, the T_4 concentration rises to above 4,0 µg/dl, while in hypothyroid animals the level remains below 1,0 µg/dl[1]. However this test is very expensive due to the cost of "Thytropar"*, and the necessity of assaying pre- and post-injection T_4 levels[3,4].

Thyrotropin (TSH) assay is the most sensitive and accurate method of diagnosing hypothyroidism in humans. Unfortunately, the assy of canine thyrotropin, by utilizing the human thyrotropin RIA test, is unrealiable. This test in dogs must await the purification of canine thyrotropin and the development of antibodies[3].

Thyroid gland biopsy can yield useful results if the caudal quarter of one of the lobes is removed for histological examination. This is a simple surgical procedure without significant risk to the patient. Needle biopsy is not satisfactory as the sample size is too small to allow

Fig. 4. Dachshund cross dog, male, 5 years old, suffering from bilaterally symmetrical, non-pruritic alopecia of the axillae, ventral abdomen, thighs and tail.

*Thyroid stimulating hormone, Armour Pharmaceutical.

an accurate evaluation of the functional state of the thyroid glands. Primary hypothyroidism is characterized by thyroid gland atrophy due to loss of follicular cells. Secondary hypothyroidism (due to TSH deficiency) is clearly distinguished by distended follicles due to colloid accummulation, the absence of peripheral vacuoles in the colloid and by the flattened follicular epithelium[4].

Practical diagnosis of hypothyroidism should be based on history, clinical symptoms, T_4 CPB or T_4 RIA test, and response to thyroid hormone replacement therapy (see below). Complete blood counts and serum cholesterol should be utilized only to monitor the response to treatment.

TREATMENT

Hypothroidism in animals cannot be treated and cured: it can only be controlled by the life-long daily administration of thyroid hormone replacement therapy. The 3 proprietary preparations which contain an accurate and consistent level of thyroid hormones are:

"Tertroxin" (Glaxo-Allenby): 5 mcg and 20 mcg tablets containing triiodothyronine (T_3)
"Diotroxin" (Glaxo-Allenby); 90 mcg thyroxine (T_4) and 10 mcg triiodothyronine (T_3) per tablet
"Eltroxin" (Glaxo-Allenby): 0,05 and 0,1 mg tablets (50 mcg and 100 mcg) containing thyroxine (T_4).

The replacement dose of thyroxine for the dog has previously been:

intially 10–20 mcg/kg/day
after 2 weeks, increase to 20–30 mcg/kg/day
maximum of 40 mcg/kg/day

Recently it has been demonstrated that the dosage of both T_4 and T_3 required to return hypothyroid dogs to normal are higher than expected, and are much higher than the amount required in humans[5]. Accordingly, the recommended dosage rates in dogs now are:

(a) T_3: 12,8 mcg/kg/day in 4–6 divided doses "Tertroxin" one 20 mcg tablet per 1,5 kg per day.
(b) T_3 and T_4 combination: 32 mcg/kg/day of T_4, with 3,2 mcg/kg/day of T_3 in 2 divided doses "Diotroxin" one 100 mcg tablet per 3 kg per day.
(c) T_4: 32 mcg/kg/day in 2 divided doses "Eltroxin" one 100 mcg tablet per 3 kg per day.

It is known that the thyroid hormones in dogs have a rapid plasma clearance half-time as compared with humans. Thyroxine (T_4) should be administered twice daily while triiodothyronine (T_3) should be given 4–6 times a day. A clear clinical response should be seen within 6 weeks from the commencement of adequate replacement therapy.

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A RE-ASSESSMENT OF THE EFFICACY OF FEBANTEL (RINTAL[R]) AND FENBENDAZOLE (PANACUR[R]) AGAINST *STRONGYLOIDES PAPILLOSUS* IN SHEEP AND GOATS

P. GRIMBEEK and H.J.J. TERBLANCHE

ABSTRACT: Grimbeek P.*, Terblanche H.J.J.†, **A re-assessment of febantel (Rintal[R]) and fenbendazole (Panacur[R]) against *Strongyloides papillosus* in sheep and goats.** *Journal of the South African Veterinary Association,* (1980) **51** No. 1, 49-50 (En).

An efficacy evaluation using febantel and fenbendazole was carried out against *Strongyloides papillosus* in sheep and goats in the Grootfontein area of South West Africa/Namibia. Three groups with five sheep and three goats in each group were artificially infested with this species. When the worms had reached the adult stage one group was treated with febantel, the other group with fenbendazole and the third group left as controls. Both anthelmintics were dosed at 5 mg/kg. All animals were sacrificed one week after treatment and total worm-counts carried out. Both anthelmintics were found to be highly effective.

INTRODUCTION

Strongyloides papillosus is encountered in sheep and goats in the arid and semi-arid regions of the North West Cape Province and in similar areas in South West Africa/Namibia. Infestation is percutaneous or via the ewes milk in the first few days of life[1]. Natural infestation of several thousand parasites with accompanying high egg counts are common. Febantel and fenbendazole both have AAA classification against this parasite which indicated high efficacy against third stage larvae, fourth stage larvae and adult worms. In the Grootfontein area of South West Africa/Namibia it was reported that in some animals some worm eggs were present in the faeces after treatment. This trial was designed to assess the efficacy once more against the adult stage of this parasite by dosing these two anthelmintics which are commonly used by farmers.

MATERIALS AND METHODS

Fifteen young worm-free sheep and nine young goats were each artificially infested percutaneously with 3000 infective larvae of *S. papillosus* (Grootfontein strain). They were divided into three groups with five sheep and three goats per group. One month after artificial infestation Group I was given an oral dose of 5 mg/kg of a 2,5 % fenbantel suspension. Group II received an oral dose of 5 mg/kg of a 2,5 % fenbendizole suspension. Group III was left as untreated controls. On day seven and day eight after treatment all twenty-four animals were sacrificed and a total worm count carried out on the gastro-intestinal contents. This was done in the conventional manner of washing the contents over sieves with apertures of 150 microns followed by microscopical counting.

RESULTS OF TRIAL

Details of the trial and the results are presented in Table 1 (see overleaf).

DISCUSSION

Both remedies proved to be highly effective. On a percentage basis fenbendazole produced a reduction in worms of 89,1 % and febantel 85 %.

When the evaluation is calculated on the non-parametric basis, the median figure for the number of worms in the control group is 709. For an A-classification a treated group should therefore not have more than one animal harbouring more than 177 worms ($709 \times 0{,}25 = 177$). The highest numbers of worms recovered from the treated groups were 167 in the fenbendazole group and 130 in the febantel group. Both remedies therefore easily qualify for an A classification if each group had had at least 11 treated animals in it.

Since *S. papillosus* is a prolific egg producer it is possible that those worms left behind after treatment, could count for positive egg counts under practical farming conditions. By virtue of the fact that infestation is percutaneous and according to Lyons *et al.*[1] can also be transmitted transmammary lambs can harbour adult parasites at the age of nine days. The prepatent period of this worm-species is only 9 days and the immature stages complete their tissue phase in about 6 days.

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*c/o Hoechst Pharmaceuticals (Pty) Limited, P.O. Box 8692, Johannesburg 2000
†c/o Bayer S.A. (Pty) Limited, P.O. Box 1366, Johannesburg 2000
[R]Rintal = Registered Trademark of Bayer Leverkusen. West Germany.
[R]Panacur = Registered Trademark of Hoechst Aktiengesellschaft. West Germany.

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Table 1: EFFICACY OF FEBANTEL AND FENBENDAZOLE AGAINST *S. papillosus*

Animal No	Mass kg	Sex	Age teeth	Anthelmintic	Dose mℓ	Dose mg/kg	Worms recovered post mortem	
5	36,4	Male	Milk	Febantel	7,3	5	119	
15	32,7	Female	Milk	Febantel	6,5	5	92	Mean for
13	33,2	Female	Milk	Febantel	6,6	5	103	group = 110,8
12	29,5	Female	Milk	Febantel	6,0	5	102	
8	30,5	Female	Milk	Febantel	6,0	5	101	
B 6	43,2	Male	2	Febantel	8,6	5	128	
B 4	22,3	Male	Milk	Febantel	4,5	5	112	
B 3	22,3	Female	Milk	Febantel	4,5	5	130	
				Efficacy expressed as percentage = 85,0				
3	38,6	Female	Milk	Fenbendazole	7,7	5	40	
2	34,5	Female	Milk	Fenbendazole	7,0	5	33	Mean for
10	32,7	Female	Milk	Fenbendazole	6,5	5	70	group = 80,25
11	32,7	Male	Milk	Fenbendazole	6,5	5	89	
4	29,1	Female	Milk	Fenbendazole	5,8	5	113	
B 9	37,6	Male	2	Fenbendazole	6,7	5	63	
B 5	29,1	Male	2	Fenbendazole	5,8	5	67	
B 2	23,2	Female	Milk	Fenbendazole	4,6	5	167	
				Efficacy expressed as percentage = 89,1				
1	37,3	Male	Milk	Control			675	
6	34,1	Female	Milk	Control			698	Mean for
7	32,7	Female	Milk	Control			655	group = 733,5
9	32,3	Female	Milk	Control			816	
14	27,3	Male	Milk	Control			825	
B 7	40,5	Male	2	Control			717	
B 8	33,2	Male	2	Control			702	
B 1	18,2	Male	Milk	Control			780	

OXFENDAZOLE: ANTHELMINTIC ACTIVITY IN SHEEP ARTIFICIALLY INFECTED WITH NEMATODES. RESULTS OF TRIALS AGAINST NINE SPECIES INCLUDING BENZIMIDAZOLE-RESISTANT *HAEMONCHUS CONTORTUS*

J. BERGER

ABSTRACT: Berger, J. **Oxfendazole: Anthelmintic activity in sheep artificially infected with nematodes. Results of trials against nine species including benzimidazole-resistant *Haemonchus contortus*.** *Journal of the South African Veterinary Association* (1980) **51** No. 1, 51-58 (En) Kwanyanga Research Station, Coopers (South Africa) (Pty) Ltd., Greenfields, East London, 5208, Republic of South Africa.

In a series of 11 trials, in which 281 lambs were artificially infected with 9 species of nematodes, larval anthelmintic tests were carried out to assess the activity of a dose of 5 mg/kg oxfendazole against all 3 parasitic stages of each species. An efficacy of over 99,4 % was obtained against all stages of *Haemonchus contortus, Ostertagia circumcincta, Trichostrongylus colubriformis, Nematodirus spathiger, Gaigeria pachyscelis, Chabertia ovina* and *Oesophagostomum columbianum* with the exception of the third larval stage of *O. columbianum* for which an efficacy of 93,5 % was recorded. Against *Dictyocaulus filaria* efficacies against the fifth stages and adults, fourth larval and third larval stages were 99,8 %, 92,0 % and 37,5 % respectively, and in two additional tests against the combined third and fourth larval stages, efficacies of 86,4 % and 85,3 % were recorded. Efficacy against *Strongyloides papillosus* was poor. In 2 supplementary experiments oxfendazole exhibited a 92,2–94,8 % efficacy against adults of the Boshof benzimidazole-resistant strain of *H. contortus,* the highest activity so far recorded against the strain by any benzimidazole tested.

INTRODUCTION

Oxfendazole has been shown to have a high degree of activity against nematodes of the gastro-intestinal and respiratory systems of ruminants at relatively low dose rates. Excellent efficacy against both immature and adult parasites has been recorded in both artificially and naturally infected sheep in the U.S.A.[2 10], Ireland[7], England[9], France[12], Australia[13] and New Zealand[4 5]. In both sheep and cattle this high degree of activity has also been recorded against inhibited stages of several nematode species[1 6 16].

The trials reported here, with one exception, were designed to record, in larval anthelmintic tests as described by Reinecke[15], the efficacy of a standard dose of oxfendazole against all 3 parasitic stages of 9 nematode species and, in additional experiments, to compare its efficacy against a benzimidazole-resistant strain of *H. contortus* with that previously shown by other benzimidazoles in current use[3].

MATERIALS AND METHODS

Experimental lambs

In 11 larval anthelmintic tests a total of 281 Dorper or Dorper × Merino lambs were used, aged 2,5–5 months at time of artificial infection and 4–6,5 months at slaughter. All lambs were housed and reared with their dams free from helminth infection, received at least 2 anthelmintic treatments during this time, were housed on slatted floors during the course of the trials and were fed a lucerne chop/pellet concentrate ration.

In 2 supplementary trials 45 Dorper lambs were used with an age range at infection of 2,5–4 months and at slaughter of 3,5–5 months. Lambs in the first of these trials, having been exposed on lightly infested pasture before being housed on slats at the age of 8–10 weeks, received 2 doses of 60 mg/kg parbendazole and one dose of 10 mg/kg levamisole during the month before infection. Lambs in the second trial were reared worm-free as described above.

In all trials male and female lambs were allocated proportionately to control and treatment groups.

Experimental infections

Donor sheep carrying fully susceptible laboratory strains of *Haemonchus contortus, Ostertagia circumcincta, Trichostrongylus colubriformis, Strongyloides papillosus, Nematodirus spathiger, Gaigeria pachyscelis, Chabertia ovina, Oesophagostomum columbianum* and *Dictyocaulus filaria,* and one benzimidazole-resistant Boshof strain of *H. contortus,* were the source of infective larvae. The larval doses and dosing regimens used in the larval anthelmintic tests are included in Tables 1–3. The larval infection routine used in Trials 5 and 7, in which separate assessments were made against third stage larvae (L_3) and fourth stage larvae (L_4) of *D. filaria,* differed from that used in Trials 10 and 11 which conformed to the larval anthelmintic test protocol for a combined L_3/L_4 assessment. In the supplementary trials each lamb received 3 daily doses of 1000 *H. contortus* larvae 19, 20 and 21 d prior to treatment. In Trial 10 (Table 1(g)) one treatment group received extra larvae to adhere to the prescribed daily dosing schedule and to avoid the inclusion of an extra control group. At the time it was already known that efficacy was likely to prove 100 %, thus allowing for the calculated risk of having a control group infected with a smaller larval dose.

Larvae suspended in water were given by tube into the oesophagus. Each total larval dose was given in divided daily doses (trickle infection) over the prescribed period for the particular larval stage of the species concerned. In the case of *G. pachyscelis* and *S. papillosus,* the total larval dose was applied dermally to an area of clipped skin high on the left flank. The total larval dose was also given on one day in the trials designed to assess activity against adult and L_3 *D. filaria.*

Lamb groups

Although the lambs in various trials were infected with one or several species simultaneously, the larval anthelmintic test against each stage was assessed in 11 treated and 9 untreated controls. One larval indicator control (LIC) was slaughtered on the day of treatment, indicat-

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Table 1: LARVAL INFECTION RATES, LARVAL AGES AT TIME OF TREATMENT AND SLAUGHTER, RANGE IN NUMBER OF WORMS RECOVERED FROM 9 UNTREATED LAMBS AND 11 LAMBS GIVEN 5 mg/kg OXFENDAZOLE, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

(a) *Haemonchus contortus*

		L_3 (Trial 2)		L_4 (Trial 1)		5th/Adult (Trial 3)	
Larval dose[1]		3000 (2 × 1500)		2960 (9 × 329)		3320 (11 × 302)	
Larval age[2]		1–2 d		3–11 d		11–21 d	
LIC lamb.	No. worms / stage %	417 L_3 100%		1238 L_3 4% L_4 89% 5th 7%		1616 L_4 2% 5th 98%	
		Control	Treated	Control	Treated	Control	Treated
Slaughter age[3]		35–37 d	36–38 d	31–41 d	31–41 d	25–36 d	25–37 d
Range of worm burdens Median		627–2150 786	0–11	1043–1891 1465	0–1	1372–2279 1879	0–2
Group mean		1018,1	2,9	1492,9	0,2	1808,1	0,3
Mean take		33,9%		50,4%		54,5%	
Group mean reduction			99,7%		99,9%		99,9%
Control median × 0,25		196,5	0/11 exceed 196,5	366	0/11 exceed 366	470	0/11 exceed 470
Efficacy classification			A		A		A

(b) *Trichostrongylus colubriformis*

		L_3 (Trial 2)		L_4 (Trial 1)		5th/Adult (Trial 3)	
Larval dose		3000 (3 × 1000)		2890 (6 × 481)		2940 (12 × 245)	
Larval age		1–3 d		4–9 d		10–21 d	
LIC lamb.	No. worms / stage %	908 L_3 100%		1301 L_3 2% L_4 60% 5th 38%		2502 L_4 <0,1% 5th 100%	
		Control	Treated	Control	Treated	Control	Treated
Slaughter age		35–39 d	35–39 d	32–40 d	32–40 d	25–41 d	26–42 d
Range of worm burdens Median		1518–2265 1699	0–2	1344–2339 1897	0–0	2143–3280 2544	0–0
Group mean		1799,8	0,4	1858,4	0	2664	0
Mean take		60,0%		64,3%		90,6%	
Group mean reduction			99,9%		100%		100%
Control median × 0,25		425	0/11 exceed 425	474	0/11 exceed 474	636	0/11 exceed 636
Efficacy classification			A		A		A

(c) *Oesophagostomum columbianum*

		L_3 (Trial 2)		L_4 (Trial 1)		5th/Adult (Trial 3)	
Larval dose		840 (6 × 140)		810 (15 × 54)		900 (21 × 43)	
Larval age		1–6 d		7–21 d		22–42 d	
LIC lamb.	No. worms / stage %	60[4] L_3 100%		435 L_4 86% 5th 14%		493 L_4 15% 15th 85%	
		Control	Treated	Control	Treated	Control	Treated
Slaughter age		35–42 d	35–42 d	35–50 d	35–50 d	36–57 d	37–58 d
Range of worm burdens Median		328–458 446	4–60	272–691 503	0–1	72–422 336	0–0
Group mean		424,2	27,8	498,3	0,1	277,8	0
Mean take		50,5%		61,5%		30,9%	
Group mean reduction			93,5%		99,9%		100%
Control median × 0,25		111,5	0/11 exceed 111,5	126	0/11 exceed 126	84	0/11 exceed 84
Efficacy classification			A		A		A

Table 1: (continued)

(d) *Gaigeria pachyscelis*

		L_3 (Trial 4)	L_4 (Trial 4)	5th (Trial 4)
Larval dose		426	426	426
Larval age		6 d	15 d	40 d
LIC lamb.	No. worms stage %	27 L_3 100%	92 L_4 100%	139 5th 100%
	Control	Treated	Treated	Treated
Slaughter age	54–55 d	40–41 d	48–49 d	55–56 d
Range of worm burdens Median	0–253 207	0–0	0–0	0–1
Group mean	184,4	0	0	0,1
Mean take	43,3%			
Group mean reduction		100%	100%	99,9%
Control median × 0,25	51,8	0/11 exceed 51,8	0/11 exceed 51,8	0/11 exceed 51,8
Efficacy classification		A	A	A

(e) *Ostertagia circumcincta*

		L_3 (Trial 5)		L_4 (Trial 7)		5th/Adult (Trial 6)	
Larval dose		2960 (3 × 986)		3040 (6 × 506)		2900 (12 × 242)	
Larval age		1–3 d		4–9 d		10–21 d	
LIC lamb.	No. worms stage %	817 L_3 100%		235[5] L_4 100%		948 L_3 13% L_4 34% 5th 53%	
		Control	Treated	Control	Treated	Control	Treated
Slaughter age		36–39 d	37–40 d	31–37 d	32–38 d	24–35 d	25–37 d
Range of worm burdens Median		1600–2507 1946	0–11	117–1974 1606	0–4	371–1327 986	0–2
Group mean		1950,2	3,45	1527,2	1,3	967,7	0,45
Mean take		65,9%		50,3%		33,4%	
Group mean reduction			99,8%		99,9%		99,9%
Control median × 0,25		486,5	0/11 exceed 486,5	401	0/11 exceed 401,5	246,5	0/11 exceed 246,5
Efficacy classification			A		A		A

(f) *Nematodirus spathiger*

		L_3 (Trial 5)		L_4 (Trial 7)		5th/Adult (Trial 6)	
Larval dose		3000 (3 × 1000)		2970 (9 × 330)		3270 (9 × 363)	
Larval age		1–3 d		4–12 d		13–21 d	
LIC lamb.	No. worms stage %	1093 L_3 100%		2120 L_4 94% 5th 6%		2289 L_4 1% 5th 99%[6]	
		Control	Treated	Control	Treated	Control	Treated
Slaughter age		36–39 d	37–40 d	31–40 d	32–41 d	27–35 d	28–37 d
Range of worm burdens Median		1309–2579 2351	0–2	879–2466 2165	0–38	51–2980 2524	0–37
Group mean		2193,1	0,5	2078,7	6,5	2134,7	12,4
Mean take		73,1%		70%		65,3%	
Group mean reduction			99,9%		99,7%		99,4%
Control median × 0,25		588	0/11 exceed 588	541	0/11 exceed 541	631	0/11 exceed 631
Efficacy classification			A		A		A

Table 1: (continued)

(g) *Chabertia ovina*

		L_3 (Trial 5)		L_4 (Trial 10)		5th/Adult (Trial 10)	
Larval dose		860 (8 × 107)		850 (17 × 50)		1150 (23 × 50)[7]	
Larval age		1–8 d		9–25 d		26–48 d	
LIC lamb.	No. worms stage %	289 L_3 100%		588 L_3 3,5% L_4 73,5% 5th 23%		719 L_4 37% 5th 63%	
		Control	Treated	Control	Treated	Control	Treated
Slaughter age		36–44 d	37–45 d	37–53 d	38–54 d	37–53 d	40–62 d
Range of worm burdens Median		380–673 601	0–0	596–749 714	0–0	596–749 714	0–0
Group mean		590,2	0	690,8	0	690,8	0
Mean take		68,6%		81,3%		—	
Group mean reduction			100%		100%		100%
Control median × 0,25		150	0/11 exceed 150	178,5	0/11 exceed 178,5	178,5	0/11 exceed 178,5
Efficacy classification			A		A		A

(h) *Strongyloides papillosus*

		L_3 (Trial 9)		L_4 (Trial 8)		5th/Adult (Trial 8)	
Larval dose		2925		3010		3010	
Larval age		2 d		5 d		14 d	
LIC lamb.	No. worms stage %	12[8] L_3 100%		775 L_4 99% 5th 1%		1588 5th 100%	
		Control	Treated	Control	Treated	Control	Treated
Slaughter age		30–31 d	31–32 d	20–21 d	21–22 d	20–21 d	27–28 d
Range of worm burdens Median		132–336 238	64–409	1000–2330 1915	148–1522	1000–2330 1915	107–811
Group mean		243,4	155	1865	456,3	1865	395,7
Mean take		8,3%		62,0%		62,0%	
Group mean reduction			36,3%		75,5%		78,8%
Control median × 0,25 × 0,4 × 0,5		 119	 7/11 exceed 119	478,7 766 	4/11 exceed 478,7 2/11 exceed 766	478,7 766 	3/11 exceed 478,7 1/11 exceed 766
Efficacy classification			X		B		B

1 Total number (rounded off) of infective larvae given and approximate daily infection rates in parenthesis
2 Range of days of infection prior to day of treatment
3 Range of days between infection and slaughter
Infection 11 days prior to treatment in Trial 3 was given in error
4 Plus 182 exsheathing L_3 infective larvae
5 Small intestine wall digest too prolonged, leaving worm debris
6 Only 37% sexually mature
7 Only the treated group received extra larvae in Trial 10. See text.
8 All from lung parenchyma

Table 2: LARVAL INFECTION RATES, LARVAL AGES AT TIME OF TREATMENT AND SLAUGHTER, RANGE OF WORMS RECOVERED AT NECROPSY, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

(a) *Dictyocaulus filaria*

	L_3 (Trial 5)		L_4 (Trial 7)		5th/Adult (Trial 3)	
Larval dose	1000		1000 (3 × 334)		1000	
Larval age	2 d		6–8 d		26 d	
LIC lamb. No. worms / stage %	241 L_3 100%		270 L_4 95% 5th 5%		174 5th 100%	
	Control	Treated	Control	Treated	Control	Treated
Slaughter age	37–38 d	38–39 d	35–36 d	36–37 d	41–42 d	42–43 d
Control Group = 9 lambs	12	27	141	0	86	0
	186	113	184	1	109	0
	219	114	217	1	164	0
	238	131	238	6	209	0
	242	142	291	7	231	0
Treated group = 11 lambs	243	152	301	8	344	0
given 5 mg/kg oxfendazole	277	173	439	13	347	0
	375	190	484	32	403	1
	382	192	508	41	500	1
		193		69		1
		235		96		2
Group mean	241,6	151,1	311,4	24,9	265,9	0,45
Mean take	24,2%		31,1%		26,6%	
Group mean reduction		37,5%		92,0%		99,8%
Control median			72,75	1/11 exceed 72,75	57,75	0/11 exceed 57,75
× 0,25						
× 0,5	121	8/11 exceed 121				
Efficacy classification		X		A		A

(b) *Dictyocaulus filaria* (Combined stages trials)

	L_3/L_4 (Trial 10)		L_3/L_4 (Trial 11)	
Larval dose	990 (8 × 124)		1040 (8 × 130)	
Larval age	1–8 d		1–8 d	
LIC lamb. No. worms / stage %	257 L_4 100%		211 L_3 30% L_4 53%	5th 17%
	Control	Treated	Control	Treated
Slaughter age	29–36 d	30–37 d	35–42 d	36–43 d
Control Group = 9 lambs	78	3	22	8
	82	4	79	10
	154	4	100	15
	156	6	114	16
	186	8	226	19
Treated group = 11 lambs	243	15	242	21
given 5 mg/kg oxfendazole	269	40	258	25
	403	46	282	28
	463	48	401	43
		50		53
		113		71
Group mean	226	30,6	191,6	28,1
Mean take	22,8%		18,4%	
Group mean reduction		86,4%		85,3%
Control median				
× 0,25	46,5	3/11 exceed 46,5	56,5	1/11 exceed 56,5
× 0,4	74,4	1/11 exceed 74,4		
Efficacy classification		B		A

Table 3: LARVAL INFECTION RATES, LARVAL AGES AT TIME OF TREATMENT AND SLAUGHTER, RANGE IN NUMBER OF WORMS RECOVERED FROM 9 UNTREATED LAMBS AND 11 LAMBS GIVEN 5 mg/kg OXFENDAZOLE, PERCENTAGE REDUCTION IN WORM BURDENS

Haemonchus contortus (Benzimidazole resistant strain Boshof)

	L_3 (Trial 9)		L_4 (Trial 8)		5th/Adult (Trial 9)	
Larval dose	2800 (2 × 1400)		3090 (9 × 343)		3090 (9 × 343)	
Larval age	1–2 d		3–11 d		12–20 d	
LIC lamb. No. worms / stage %	615 L_3 100%		1005 L_3 3% L_4 84% 5th 13%		1941 L_3 <0,5% L_4 13,5% 5th 86%	
	Control	Treated	Control	Treated	Control	Treated
Slaughter age	29–31 d	30–32 d	27–28 d	19–28 d	18–27 d	25–34 d
Range of worm burdens	948–1507	32–346	710–1500	65–328	548–978	40–277
Group mean	1181	150,8	942,6	171,6	795,8	116,5
Mean take	42,2%		30,5%		—	
Group mean reduction		87,2%		81,8%		85,4%

* Control group the same for both L_4 and 5th/adult treated groups. Number of L_4 and 5th/adult worms recovered recorded for the L_4 assessment but only 5th/adult worms for the 5th/adult assessment.

ing the distribution and maturity of the various parasitic stages at that time. A study of the tables will show the species combination in any one trial.

In the two supplementary trials concerned with benzimidazole resistance, each group comprised 5 lambs.

Dosing procedure

Lambs in the treatment groups received a single standard dose of 5 mg/kg oxfendazole as an aqueous suspension administered by tube into the cervical oesophagus. In one additional larval anthelmintic test against combined L_3/L_4 *D. filaria* (Trial 11), carried out for registration purposes to confirm that small particle size was not associated with reduced efficacy, micronised oxfendazole was used. In the 2 supplementary trials involving benzimidazoles or benzimidazole-like compounds, thiabendazole*, albendazole† and febantel‡ were given at standard field dosage rates, and experimental oxibendazole supplied by Smith Kline and French was given at 15 mg/kg.

Necropsy and worm recovery

Treated and control lambs were slaughtered when infections should have reached maturity or near maturity.

Procedures adopted in Trials 1–11 conformed to those described for larval anthelmintic tests in ruminants suitable for analysis by a modified non-parametric method of evaluation[15]. Wherever appropriate, peptic digests of viscus wall were carried out to ensure recovery of both migrating and inhibited larvae. Lungs, mesenteric lymph nodes and blood were processed in some trials to recover adult or immature stages of *D. filaria* and immature stages of *G. pachyscelis* and *S. papillosus*.

*Thibenzole, MSD (Pty) Ltd
†Valbazen, Smith, Kline & French (Pty) Ltd
‡Rintal, Bayer South Africa (Pty) Ltd

In the 2 supplementary trials total counts of *H. contortus* recovered from the abomasal content and mucosal washings were made without recourse to peptic digests.

In every trial total worm counts were made in all treated lambs as was also done in all but 4 cases in control lambs, in which the large number of worms present allowed for aliquot sample counts. To avoid wasted effort counts were also made from aliquot samples of various organ recoveries in some LIC lambs.

RESULTS

The results of the larval anthelmintic tests have been summarised in Tables 1–3. The worm recoveries from the larval indicator control (LIC) lambs have also been included in the tables and, where appropriate, the anthelmintic efficacy classification evaluated by the modified non-parametric method[15].

The supplementary trials against adult *H. contortus* of the Boshof strain have been summarised in Table 4.

Experiment 1

Activity of oxfendazole at 5 mg/kg against 19–21 day old worms was compared with that of thiabendazole and oxibendazole at standard field dose rates and mean treatment efficacies of 94,8 %, 37,0 % and 53,2 % respectively were recorded.

Experiment 2

Activity of oxfendazole at 5 mg/kg against 19–21 day old worms was compared with that of thiabendazole, albendazole and febantel at standard field dose rates and mean treatment efficacies of 92,2 %, 27,5 %, 69,3 % and 54,6 % respectively were recorded.

DISCUSSION

With the exception of the *S. papillosus* infected control lambs in Trial 9 (Table 1(h)) all infections gave mean

Table 4: COMPARATIVE BENZIMIDAZOLE TREATMENT OF LAMBS INFECTED WITH THE BOSHOF STRAIN OF *H. Contortus*. FAECAL EGG COUNTS, WORM COUNTS AT NECROPSY AND PERCENTAGE REDUCTIONS IN WORM BURDENS

		Experiment 1				Experiment 2			
		Mean eggs per gram of faeces on day of:		Mean No. of worms recovered (range)	% reduction compared with control mean	Mean eggs per gram of faeces on day of:		Mean No. of worms recovered (range)	% reduction compared with control mean
		treatment	slaughter			treatment	slaughter		
Group of 5 control lambs		4710	13320	2176 (1784–2464)		2100	6310	1888 (1290–2279)	
Group of 5 lambs treated with:	Dose rate mg/kg								
Thiabendazole	44	3130	4800	1371 (724–1566)	37,0	2460	5100	1368 (526–2167)	27,5
Oxibendazole	15	3980	2320	1019 (609–1426)	53,2				
Oxfendazole	5	3550	100	113 (4–348)	94,8	2470	190	147 (26–245)	92,2
Albendazole	3,8					2020	520	579 (125–1351)	69,3
Febantel	5					1910	2850	858 (407–1130)	54,6

takes high enough for valid assessment of activity. In some trials single control animals appeared to show unexplained resistance to infection, unless the reduced burden was due to worm expulsion. Hookworms are notoriously inconsistent in their ability to infect a host and this may have been reflected in one control lamb in Trial 4 (Table 1(d)) found to be entirely free of *G. pachyscelis,* since scrutiny of laboratory records failed to reveal any experimental error. The low take in L_3 of *S. papillosus* in the controls may have been due to residual immunity from natural infection during the first few days of life before ewes and lambs were housed (Table 1(h)).

At 5 mg/kg oxfendazole was shown to have an efficacy of over 99,4 % against all 3 parasitic stages of *H. contortus, O. circumcincta, T. colubriformis, N. spathiger, G. pachyscelis, C. ovina* and *O. columbianum* with the exception of L_3 *O. columbianum* for which 93,5 % was recorded. The LIC lamb in the L_3 *O. columbianum* trial proved to be carrying a large number of infective larvae, implying that at treatment both infective and parasitic L_3 were present and subjected to the anthelmintic. Survival of infective larvae (if more tolerant of oxfendazole) may have contributed to the numbers in residual burdens.

Activity against the various stages of *D. filaria* is of particular interest and for this reason the full details of individual worm burdens have been included in Table 2. In Trial 3 (Table 2(a)) the degree of maturity of fifth stage (5th) *D. filaria* recovered from control lambs necropsied 40 d after infection showed a wide range in the same lamb and between individuals. It is therefore evident that treatment was equally highly effective against early 5th and mature worms. The schedule of larval infections with *D. filaria,* as laid down in the larval anthelmintic test, for activity against the combined L_3/L_4 stages was followed in Trials 10 and 11. As this procedure attempts to assess drug efficacy against 2 parasitic stages in 2 different organs (some worms having already reached the lungs), a more critical look at activity against L_3 and L_4 larvae was taken in Trials 5 and 7 respectively. In Trial 7 infection was carried out 6, 7 and 8 d prior to treatment, in the hope that both late mesenteric lymph node and early lung L_4 larvae would be subjected to the anthelmintic and also that the proportion of slow maturing L_3 would be minimal. As it turned out only 4 % of the L_4 larvae had reached the lungs and no L_3 larvae were recovered from the mesenteric lymph nodes of the LIC lamb in this trial. In Trial 5 treatment can only have been directed against the full burden of L_3 parasitic larvae accruing from the single infective dose of 1000 larvae, which is in contrast to the situation in Trials 10 and 11, in which only a small proportion of the same infective dose should have given rise to parasitic larvae still in the L_3 stage at the time of treatment. All stages of *D. filaria* were recovered from the LIC lamb in Trial 11 and L_5 larvae were found in both mesenteric lymph nodes and lungs. In contrast, and reflecting the wide variation in progress of infection by this species in individual hosts, no larvae had reached the lungs in the LIC lamb in Trial 10 and only L_4 were recovered.

The results for *D. filaria* recorded in Trials 5 and 7 indicate that, at the dose rate used, the activity of oxfendazole is related to the degree of development of the larvae rather than the habitat of the parasites.

The only species tested which showed a comparative tolerance to oxfendazole was *S. papillosus,* efficacies of 36,3 %, 75,5 % and 78,8 % being recorded against L_3, L_4, 5th and adult stages respectively. In view of the sustained plasma levels of oxfendazole attained after dosing, the very low activity against the L_3 may not be associated so much with the various systemic habitats of this stage as with its energy requirements, as may also be the case in L_3 of *D. filaria.*

There is a valid explanation for the discrepancy in the efficacy rating of 85,4 % obtained against 5th or adult *H. contortus* Boshof strain in the larval anthelmintic

test in Trial 8 (Table 3) and of 94,8 % and 92,2 % obtained in the 2 supplementary experiments (Table 4). It would appear from Trial 8 that L_4 are more resistant than 5th or adult stages, and, since the infection schedule in this trial will have given rise to burdens at the time of treatment including large numbers of early 5th and also L_4, which was confirmed in the LIC lamb, it would be expected that the greater maturity of worms at the time of treatment in the supplementary experiments would provide a more susceptible population.

The Boshof strain of *H. contortus* appears to be more benzimidazole-resistant than the Beltsville AH-2 isolate used by Kistner and Wyse[11], who also used oxfendazole at 5 mg/kg and recorded an efficacy of 100 % against all three parasitic stages.

In the 2 comparative benzimidazole experiments the appropriate format was kept identical to that of the original trial previously reported[3]. Thiabendazole was included on each occasion as control for anthelmintic activity and it can be seen that the Boshof strain which had been passaged 5 times since isolation from the field in 1975[3], without any further anthelmintic experience, had become no more susceptible to the standard 44 mg/kg dose. The relative activities of 8 benzimidazoles and one analogue (febantel), currently being employed as ruminant anthelmintics, against this *H. contortus* Boshoff strain are set out in Table 5.

Table 5: COMPARATIVE ACTIVITY OF FIELD DOSE RATES OF BENZIMIDAZOLES AGAINST THE BENZIMIDAZOLE RESISTANT BOSHOFF STRAIN OF *H. Contortus*

		Mean % reduction in worms in groups of five lambs		
	Dose mg/kg	Original trial	Expt. 1	Expt. 2
Thiabendazole	44	37,5	37,0	27,5
Oxibendazole	15		53,2	
Febantel	5			54,6
Mebendazole	15	63,0		
Cambendazole	20	66,4		
Albendazole	3,8			69,3
Parbendazole	30	79,4		
Fenbendazole	5	80,8		
Oxfendazole	5		94,8	92,2

The relative high degree of activity of oxfendazole may be associated with the maintenance of higher plasma levels than is the case with the other compounds, giving rise to a protracted interference with the energy metabolism of the parasite[14].

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THE ANTICOCCIDIAL EFFICACY OF ARPRINOCID* IN BROILER CHICKENS UNDER FLOOR PEN CONDITIONS**

J. SCHRÖDER, C.J.Z. SMITH AND R.G. HARVEY

ABSTRACT: Schröder J.; Smith C.J.Z.; Harvey R.G. **The anticoccidial efficacy of arprinocid in broiler chickens under floor pen conditions.** *Journal of the South African Veterinary Association* (1980) **51** No. 1, 59-61 (En) MSD Research Centre, Private Bag 3, 1685 Halfway House, Rep. of South Africa.

The efficacy of arprinocid was tested against artificial infections of mixtures of *Eimeria* spp. in broiler chickens under floor pen conditions in 3 experiments. Treatment with arprinocid at 60 ppm over a 56 d period significantly increased the live mass gain and feed efficiency of broiler chickens. This increase compared favourably with that obtained by treatment with lasalocid, robenidine and halofuginone. Birds treated with arprinocid had substantially reduced numbers of sporulated oocysts in their litter, and less severe lesion scores than non-medicated birds.

INTRODUCTION

Arprinocid (9-(2-chloro-6-fluorophenymethyl)-9H purin-6-amine) was patented in 1974[3]. It is one of a few chemical class of compounds, the 6-amino-9-(substituted benzyl) purines, which possesses anti-coccidial activity[5]. The efficacy of arprinocid against *Eimeria* spp. in chickens has been evaluated in batteries[4 5 7 10] and under floor pen conditions[6 8 10]. It has been found to suppress the production, sporulation and infectivity of *Eimeria* oocysts[7 9 11 12]. The N-1-oxide metabolite of arprinocid interferes with microsomal metabolism by binding cytochrome P-450 (C C Wang 1979 Merck Institute for Therapeutic Research, Merck Sharp & Dohme Research Laboratories, Rahway, N J, personal communication). This paper describes 3 experiments conducted with arprinocid in broiler chickens under floor pen conditions in South Africa.

Table 1: TREATMENT REGIMES FOR TRIALS 1, 2 AND 3

Treatment		Trial number		
Number	Description	1	2	3
1	Unmedicated control	+	+	+
2	Arprinocid 50 ppm 56 d	+		
3	Arprinocid 60 ppm 56 d	+	+	+
4	Arprinocid 70 ppm 56 d	+		
5	Monensin 100 ppm 56 d	+	+	
6	Monensin 100 ppm 0–28 d/Lasalocid 75 ppm 28–56 d			+
7	Lasalocid 75 ppm 56 d			+
8	Nicarbazin 125 ppm 56 d	+		
9	Nicarbazin 125 ppm 0–21 d/Arprinocid 60 ppm 21–42 d/Amprolium plus Ethopabate 125 ppm 42–56 d			+
10	Amprolium plus Ethopabate 125 ppm 56 d	+		
11	Arprinocid 60 ppm 0–42 d/Amprolium plus Ethopabate 125 ppm 42–56 d			+
12	Dinitro-toluamide (D.O.T.) 125 ppm 56 d	+		
13	Monensin 100 ppm 0–28 d/D.O.T. 125 ppm 28–56 d		+	
14	Halofuginone 3 ppm 56 d			+
15	Robenidine 33 ppm 56 d			+

*ARPOCOX: MSD

**Presented at the Biennial Congress of the South African Veterinary Association, Johannesburg 3–7 September 1979.

MATERIALS AND METHODS

The 3 trials were performed over a 7 month period from the end of July 1977 until the end of February 1978: The duration of each trial was 56 days.

(a) **Birds:** Day old Ross broiler chickens were used in all 3 trials. The birds were sex-separated in 2 of the trials. All the birds were vaccinated against Infectious Bronchitis and Newcastle Disease.

(b) **Housing:** The poultry shed in which the trials were done is temperature-controlled and ventilated by positive pressure obtained from one fan on each corner. The house is 27 × 9 m in area with one row of 18 pens (each 4,5 m^2) down each side. Fifty birds were housed in each pen (11,1 birds/m^2), and only 16 pens on each side were included in each trial, with the 4 corner pens serving as buffer pens.

(c) **Feed:** The birds were fed broiler starter mash for the first 28 d of each trial, and broiler finisher for the next 28 d (birds in 2 treatment groups in one trial received broiler starter for 21 d, broiler finisher for the next 21 d, and broiler post finisher for the last 14 d). No medication was mixed into these rations by the feed miller.

(d) **Allocation:** Birds were randomly allocated to their pens (in the 2 trials where the sexes were separated, birds of a single sex were randomly allocated to 2 of 4 blocks of 8 contiguous pens each). Treatments were randomly allocated within each block. In Trials 1 and 3 therefore, there were 2 replicates of 50 birds of each sex per treatment group (total 100 males and 100 females per treatment group). In Trial 2 there were 8 replicates of 50 birds of mixed sexes per treatment group (i.e. 400 birds per treatment group).

(e) **Treatments:** All drugs were mixed into the non-medicated broiler mash in a horizontal double ribbon mixer. Arprinocid was used at 50,70 (1 trial) and 60 ppm (3 trials) for 56 d, or in the starter or finisher mashes in shuttle treatments with nicarbazin (Nicrazin, MSD) and amprolium plus ethopabate (Amprol Plus, MSD). These treatments were compared with no treatment (every trial), monensin (Coban, Elanco) at 100 ppm for 56 d or in shuttle programmes, lasalocid (Avatec, Roche) at 75 ppm for 56 d and in a shuttle programme, dinitro-ortho-toluamide (Salcostat, Salsbury) at 125 ppm for 56 d or in a shuttle programme, halofuginone (Stenorol, Roussell) at 3 ppm, robenidene (Cycostat, Cyanamid) at 33 ppm, nicrazin at 125

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ppm, and amprolium plus ethopabate at 125 ppm. The treatments for the different trials are listed in Table 1.

(f) **Exposure to infection:** Each bird received 200 000–330 000 artificially sporulated (by aeration in $K_2Cr_2O_7$ solution) oocysts of a mixture of *Eimeria acervulina* (68–76 %), *Eimeria maxima* (6–14 %), *Eimeria necatrix* (6–10 %) and *Eimeria tenella* (7–12 %) on day 22. The oocysts were administered orally to each individual bird by gavage in the one trial. In the 2 other trials all the oocysts for the birds in one pen were mixed into 1 kg of non-medicated broiler starter mash which was offered to the birds after their regular feed had been withdrawn for 6 h.

(g) **Lesion scoring:** Three birds from each pen were killed 5—6 d after infection for autopsy and coccidial lesion scoring in 2 of the trials[1].

(h) **Live mass measurements:** All the birds in each pen were weighed in convenient groups in a plastic crate on a spring balance at day old and after 22, 28 and 56 d.

(i) **Statistical analysis:** The data from each trial were analysed by the Planning and Data Management staff of Merck, Sharp & Dohme Research Laboratories, Rahway, N.J. They used analysis of variance for randomised block or partially hierarchical designs, and compared treatment means with Duncan's New Multiple Range Test.

Table 2: MEAN LIVE MASS PER BIRD AT 56 d

	Trial number			
	1	2	3	
Treatment			Females	Males
1	1,859^{ab}	1,623^{a}	1,495^{a}	1,767^{a}
2	1,883^{b}			
3	1,991^{b}	1,818^{d}	1,665^{c}	2,021^{d}
4	1,904^{b}			
5	1,875^{ab}	1,703^{b}		
6			1,598^{b}	1,981^{cd}
7			1,611^{bc}	2,046^{d}
8	1,820^{a}			
9			1,598^{b}	1,885^{bc}
10	1,877^{ab}			
11			1,644^{bc}	1,875^{b}
12	1,868^{ab}			
13		1,773^{c}		
14			1,647^{bc}	1,954^{bcd}
15			1,658^{c}	1,890^{bc}

abcd Means for 1 trial having no superscripts in common are statistically significantly different (P<0,05)

Table 3: MEAN FEED EFFICIENCY PER BIRD AT 56 d

	Trial number			
	1		2	3
Treatment	Females	Males		
1	0,409^{a}	0,432^{a}	0,418^{a}	0,371^{a}
2	0,438^{c}	0,434^{a}		
3	0,423^{b}	0,450^{c}	0,445^{b}	0,446^{b}
4	0,416^{ab}	0,449^{c}		
5	0,422^{b}	0,443^{abc}	0,441^{b}	
6				0,435^{b}
7				0,429^{b}
8	0,408^{a}	0,432^{a}		
9				0,433^{b}
10	0,416^{ab}	0,433^{ab}		
11				0,425^{b}
12	0,420^{ab}	0,446^{bc}		
13			0,437^{b}	
14				0,437^{b}
15				0,436^{b}

abc Means for a trial having no superscripts in common are statistically significantly different (P<0,05)

RESULTS

(a) **Live mass at 56 d:** The mean live mass/bird for each treatment are listed in Table 2. All birds in Trials 1 and 2, and the female birds in Trial 3, medicated with 60 ppm arprinocid performed best. In Trial 1 the difference from other groups was not statistically significant (P<0,05). The female birds in Trial 3 did not differ statistically from those medicated with lasalocid, halofuginone, robenidine or an arprinocid/amprolium shuttle. The live mass of the male birds fed arprinocid in Trial 3 did not differ statistically from those receiving lasalocid, monensin/lasalocid shuttle, but surpassed that of the other birds (statistically significant).

(b) **Feed efficiency over 56 d:** Table 3 is a summary of these results. In Trial 1 females on 50 ppm arprinocid were most feed efficient (P<0,05). Females on 60 ppm were second best but significantly different only from females on nicarbazin or unmedicated controls. Male birds medicated at 60 ppm in Trial 1 were significantly better than unmedicated controls, or birds medicated with 50 ppm arprinocid, nicarbazin, or amprolium. In Trials 2 and 3, birds on 60 ppm arprinocid were significantly more feed efficient than unmedicated controls. They had a higher feed efficiency than birds on other treatments, but this was not statistically significant (P<0,05).

(c) **Lesion scores on Day 22:** In Trial 2 birds medicated with arprinocid had the least severe coccidial lesions in the intestinal tract. Female birds medicated with arprinocid in Trial 3 had the least severe lesions. In the same trial the lesions of male birds were more severe than those of birds medicated with nicarbazin (Table 4).

(d) **Oocyst production and sporulation over 56 d:** The litter of birds medicated with arprinocid had the lowest number of oocysts per gram compared with that of any other treatment group in Trial 2 and 3. In Trial 3, oocysts in the litter of birds on arprinocid had the lowest sporulation percentage (Table 5).

DISCUSSION

The initial studies to demonstrate the anticoccidial efficacy of arprinocid were done in battery cages[4,5]. Criteria that were used to evaluate this efficacy included mortality and growth rates, severity of coccidial lesions and oocyst production (i.e. anticoccidial index) following artificial infection by gavage[5]. Subsequently, battery studies were performed against strains of coccidia obtained from field outbreaks of coccidiosis[7]. The majority of these were resistant to one or more of the anticoccidial products currently on the market.

Table 4: MEAN LESION SCORES

Treatment*	Organ			
Trial 2	Duodenum	Jejunum	Caecum	Mean
1 Control	2,25	1,31	2,73	2,10
3 Arprinocid	0,06	0,00	0,23	0,10
5 Monensin	0,15	0,08	1,40	0,54
13 Monensin	0,19	0,06	1,69	0,65
Trial 3 (a) Male birds				
1 Control	2,25	1,50	2,92	2,22
3 Arprinocid	0,25	0,00	0,17	0,14
6 Monensin	0,17	0,00	1,42	0,53
7 Lasalocid	0,42	0,00	0,42	0,28
9 Nicarbazin	0,00	0,00	0,08	0,03
11 Arprinocid	0,00	0,00	0,00	0,00
14 Halofuginone	0,00	0,00	0,80	0,27
15 Robenidine	1,08	0,50	0,08	0,55
(b) Female birds				
1 Control	2,42	1,50	2,50	2,14
3 Arprinocid	0,00	0,00	0,08	0,03
6 Monensin	0,17	0,00	2,33	0,83
7 Lasalocid	0,33	0,08	1,67	0,69
9 Nicarbazin	0,00	0,08	0,08	0,05
11 Arprinocid	0,00	0,00	0,00	0,00
14 Halofuginone	0,00	0,00	0,08	0,03
15 Robenidine	1,25	0,25	0,33	0,61

* See Table 1 for detailed treatment regimes

Table 5: WEEKLY OOCYST PRODUCTION AND PERCENTAGE SPORULATION IN TRIAL 3

	Oocyst/gram litter/1000 (percentage sporulation)				
	Week				
Treatment	4	5	6	7	8
1 Control	370	109(75)	73,2(80)	66,6(75)	34,2(75)
3 Arprinocid	0	0,1(0)	0(0)	0,1(0)	0,5(25)
6 Monensin /Lasalocid	9,9	10,5(70)	30,6(65)	9,3(70)	10,8(70)
7 Lasalocid	12,6	10,2(70)	23,7(75)	7,2(70)	10,1(60)
9 Nicarb/APC/ Amprol Plus	0	0(0)	0,2(0)	30,6(50)	93,0(50)
11 Arprinocid/ Amprol Plus	0,1	0(0)	0(0)	25,2(50)	89,9(50)
14 Halofuginone	10,5	3,2(65)	1,5(50)	0,9(60)	1,6(60)
15 Robenidine	53,0	48,0(75)	20,4(80)	24,0(70)	21,0(75)

Chickens in battery cages were also used to study the effect of arprinocid on oocyst production, sporulation and infectivity[9 11 12]. Levels of arprinocid necessary to eliminate oocyst production varied with the species and strain of coccidia between 40 and 70 ppm[9 12]. Oocysts which were excreted by birds medicated at levels from 30–70 ppm sporulated to a lesser extent or not at all[9 11]. Such oocysts which did sporulate were less infective when inoculated into susceptible birds[9].

The results obtained in our 3 floor pen trials are in agreement with those of experiments performed under similar conditions in Brazil[6], Mexico[8] and the United States of America[2 10]. Arprinocid in the feed at 60 ppm improves the live mass gain of broiler chickens infected with various *Eimeria* spp.

Although the differences were not statistically significantly different, birds on feed containing 60 ppm arprinocid had the highest live mass (with the exception of male birds medicated with lasalocid in Trial 3), and the best feed efficiency (the exception being female birds medicated wih arprinocid at 50 ppm in Trial 1) after 56 d in all 3 trials.

Birds medicated with arprinocid (Trials 2 and 3), nicarbazin or halofuginone (Trial 3) had the least severe mean lesion scores 5 d after infection with a mixture of *Eimeria* spp. (not statistically analyzed). In previous experiments, chickens housed in batteries and artificially infected also had reduced lesion scores when medicated with arprinocid at 60 ppm[5 7]. Reduced lesion scores were also seen in other floor pen trials[6 8].

The reduction in oocyst production and percentage of sporulated oocysts from birds on 60 ppm arprinocid in Trials 2 and 3 (not statistically analyzed) confirm similar results from previous experiments[5 7 9 11 12].

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BOOK REVIEW BOEKRESENSIE

VETERINARY MEDICINE. A TEXTBOOK OF THE DISEASES OF CATTLE, SHEEP, PIGS AND HORSES

D.C. BLOOD, J.A. HENDERSON and O.M. RADOSTITS with contributions by J.H. ARUNDEL and C.C. GAY
Fifth Edition. Bailliére Tindall, London 1979 pp. 1135, figs. 15, tables 77. Price R33.85.

The first edition was published in 1960 and soon became a standard textbook. This was followed by the second edition in 1963, a third edition in 1968, and a fourth edition in 1974.

Many advances have taken place in the field of veterinary medicine during this period and the authors have attempted to cover as many of these as possible by major revisions of certain parts, e.g. equine colic, diseases of the newborn, fluid electrolyte and acid-base balance, etc.

The basic plan of the text remains the same and is divided into two major parts.

The first part covers "General Medicine" and deals with the general principles of diseases as they affect individual organs and systems. It deals with the aetiology, pathogenesis, clinical findings, clinical pathology, necropsy findings, diagnosis and treatment of diseases. This part also includes sections on clinical examination, general systemic states (toxaemia, septicaemia, disturbances of body fluids, electrolytes and acid-base balance, etc.), diseases of the newborn and a section on practical antimicrobial therapeutics.

The second part deals with "Special Medicine" and the authors discuss specific diseases and conditions – mastitis, diseases caused by bacteria, viruses and chlamydia, rickettsias, fungi, protozoa, helminth parasites, arthropod parasites, metabolic disturbances, nutritional deficiencies, physical agents, chemical agents, allergy, inheritance and specific diseases of unknown or uncertain aetiology.

As with any modern textbook in a field as broad as Veterinary Medicine there will be sections and chapters which are stronger than others. This book does not deal with diseases which are peculiar to Africa and particularly South Africa, for example heartwater, the various poisonous plants, etc.

Veterinary Medicine is widely regarded as a reliable and comprehensive text on the diseases of farm animals. This is an excellent textbook with an up-to-date well-selected bibliography representing publications up to 1977.

The layout and style are such that it is easy to read and follow. Previous editions have been consulted and used as a standard textbook in the Department of Medicine in our Veterinary Faculty.

Large animal practitioners, lecturers and veterinary students will find this very thoroughly revised new edition helpful. It can certainly be recommended for study and reference.

D.C.L.

BOOK REVIEW BOEKRESENSIE

RUSSIAN-ENGLISH DICTIONARY OF HELMINTHOLOGY AND PLANT NEMATOLOGY compiled by

G.I. POZNIAK
Technical Communication No. 49 of the Commonwealth Institute of Helminthology
Published by the Commonwealth Agricultural Bureaux, Farnham House, Farnham Royal, Slough SL2 3BN, UK 1979 pp. 108 + x Cloth Boards. ISBN 085 198 447 9 £12.00 (UK) $28.80 (Foreign)

This dictionary contains more than 6,000 entries from the fields of helminthology (animal and human) and nematology (plant parasites and free-living forms). The terms cover general parasitological concepts, helminth morphology, biology, diseases, popular names of worms, anthelmintics and aspects of helminth immunology, ecology and pathology. The value of the dictionary is greatly enhanced by the definitions and explanations provided for many of the terms.

The dictionary is intended for scientific translators and abstractors, for professional linguists who need to translate helminthological literature and for scientists with limited knowledge of Russian who wish to consult, in the original, papers in their own field.

CASE REPORT **GEVALVERSLAG**

FELINE INFECTIOUS PERITONITIS

O.M. BRIGGS

ABSTRACT: Briggs O M **Feline infectious peritonitis.** *Journal of the South African Veterinary Association* (1980) **51** No. 1 63-64 (En) Box 40, 6115 Sunland, Republic of South Africa.

Feline infectious peritonitis was recently diagnosed in a 5 year old, male, neutered, Siamese cat. Euthanasia was performed. Macro- and histopathology confirmmed the disease.

HISTORY AND CLINICAL SIGNS

The cat was presented with a history of progressive lethargy, anorexia and emaciation over a period of one week. He was afebrile, pale and had a painful abdomen. A peripheral blood smear was negative for *Babesia felis* and *Haemobartonella felis*.

DIAGNOSIS

Peritonitis of unknown aetiology.

TREATMENT

The cat was hospitalised and treated with 150 mg procaine penicillin* and 5 mg prednisolone† daily by injection for 3 days. The appetite improved. He was then injected with a combination of long and short acting penicillin – 71 mg benethamine penicillin and 75 mg procaine penicillin‡ and discharged. The patient was re-admitted 10 days later after showing a further decline.

CLINICAL SIGNS ON RE-ADMISSION

The cat was afebrile, dehydrated, pale, emaciated and weak. His abdomen was distended and pendulous and had a doughy consistency with crepitation on palpatation.

INVESTIGATIVE PROCEDURES

An abdominal radiograph revealed a generalised homogeneous appearance with no visceral structures evident. Paracentesis yielded a seemingly endless supply of strawcoloured, tacky fluid of specific gravity 1,046. A smear of this showed a number of cells later identified as polymorphonuclear leukocytes, round cells and mesothelial cells. The packed cell volume was 31 %, i.e. anaemia with dehydration. The peripheral blood smear was again negative for *B. felis* and *H. felis*. A blood smear fixed in absolute alcohol for 10 min was sent to the University of Tennessee. This was negative for feline leukaemia virus (FeLV). The absence of polychromatophilia in the smear indicated a non-regenerative anaemia[1]. No leukocyte abnormalities were apparent.

*Procain Penicillin, Milvet
†Deltacortril, Pfizer Laboratories
‡Compropen, Glaxo

REVISED DIAGNOSIS

Feline infectious peritonitus (FIP) was diagnosed. After consultation with the owner, euthanasia was performed.

NECROPSY

The abdomen contained about 100 ml straw-coloured, viscous fluid which clotted soon after exposure to air. White granules in a fibrinous material covered most of the abdominal organs which gave the organs a dull, grey-whitish granular appearance. The omentum was irregularly thickened and had some white-grey clots of fibrinous material in it. The surface of the spleen was roughened and covered in white, granular material. The liver and intestines were covered in a dull white, slimy film. The kidneys were pale.

HISTOPATHOLOGY

There was a serofibrinous to seropurulent periphepatitis and perisplenitis, as well as a similar peritonitis (Fig. 1). There were superficial areas of necrosis below the hepatic and splenic capsules with a macrophage, lymphocyte and neutrophil reaction. The lymphoid follicles in the spleen were prominent which was probably due to antigen stimulation. The mesentery revealed a focal granulomatous reaction.

DISCUSSION

FIP was first suspected in South Africa by Colly in 1970[3]. A report definitely confirming the disease was published by Bland van den Berg & Botha in 1977[2].

The following is a summary of the various diseases either known or currently postulated to be caused by FIP virus[7];

A. Primary disease.
B. Secondary disease. This is either the effusive (wet) or granulomatous (dry) form.
C. Kitten mortlity complex (KMC).

In the primary disease, the kittens or cats have subclinical or mild upper respiratory tract infection. This occurs weeks to months before the secondary disease. Only a small percentage (probably less than 1 %) in fact develop the secondary disease.

The effusive form has been further subdivided into pleural, peritoneal and pericardial types. Likewise, the

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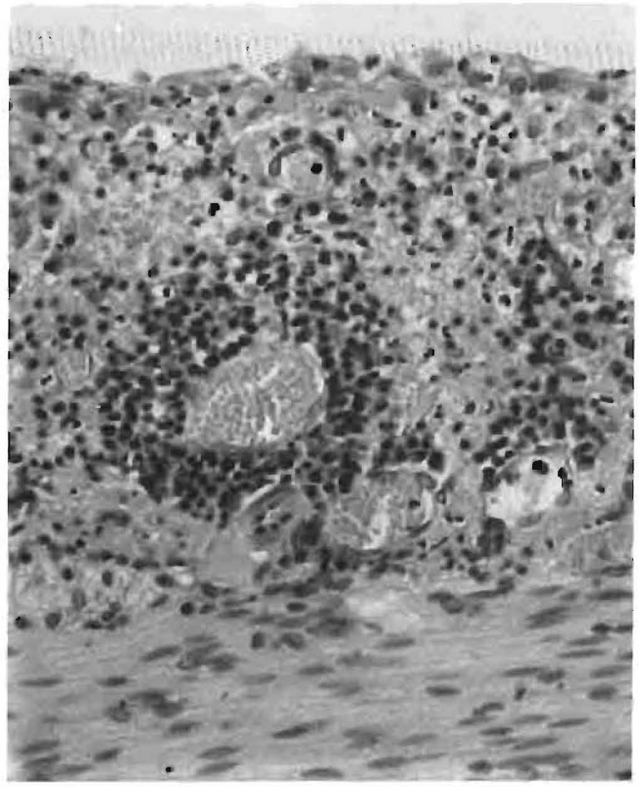

Fig. 1. The gut wall showing the peritonitis. Note the plasma cells and round cells around a blood vessel, fibrin coagulating the exudate and mesothelial cells on top.

granulomatous form may involve one or more of the following:– peritonitis, opthalmitis, encephalitis, and acute hepatitis. The effusive form (as described here in the classical peritonitis appearance) is easier to diagnose than the granulomatous form.

KMC has been added to the list after outbreaks of reproductive failure and kitten mortality in catteries. A high percentage of the breeding queens showed positive antibody titres to FIP virus. This indirect fluorescent antibody test is valuable but not diagnostic by itself. It is not yet available in South Africa. Diagnosis must be made on the collation of clinical symptoms, clinical pathology, and macro- and micropathology[2].

Pedersen *et al*[6] discussed the infectious nature of the disease and stressed that asymptomatic carrier cats are the main source of infection. He also points out that about 40–50 % of all field cases of FIP have concurrent FeLV infection. FeLV probably acts as an immunosuppressive agent here. Treatment does not usually affect a cure[7]. It is therefore advisable to consider euthenasia of the severely ill animal. This assists in preventing the spread of this increasingly important disease.

ACKNOWLEDGEMENTS

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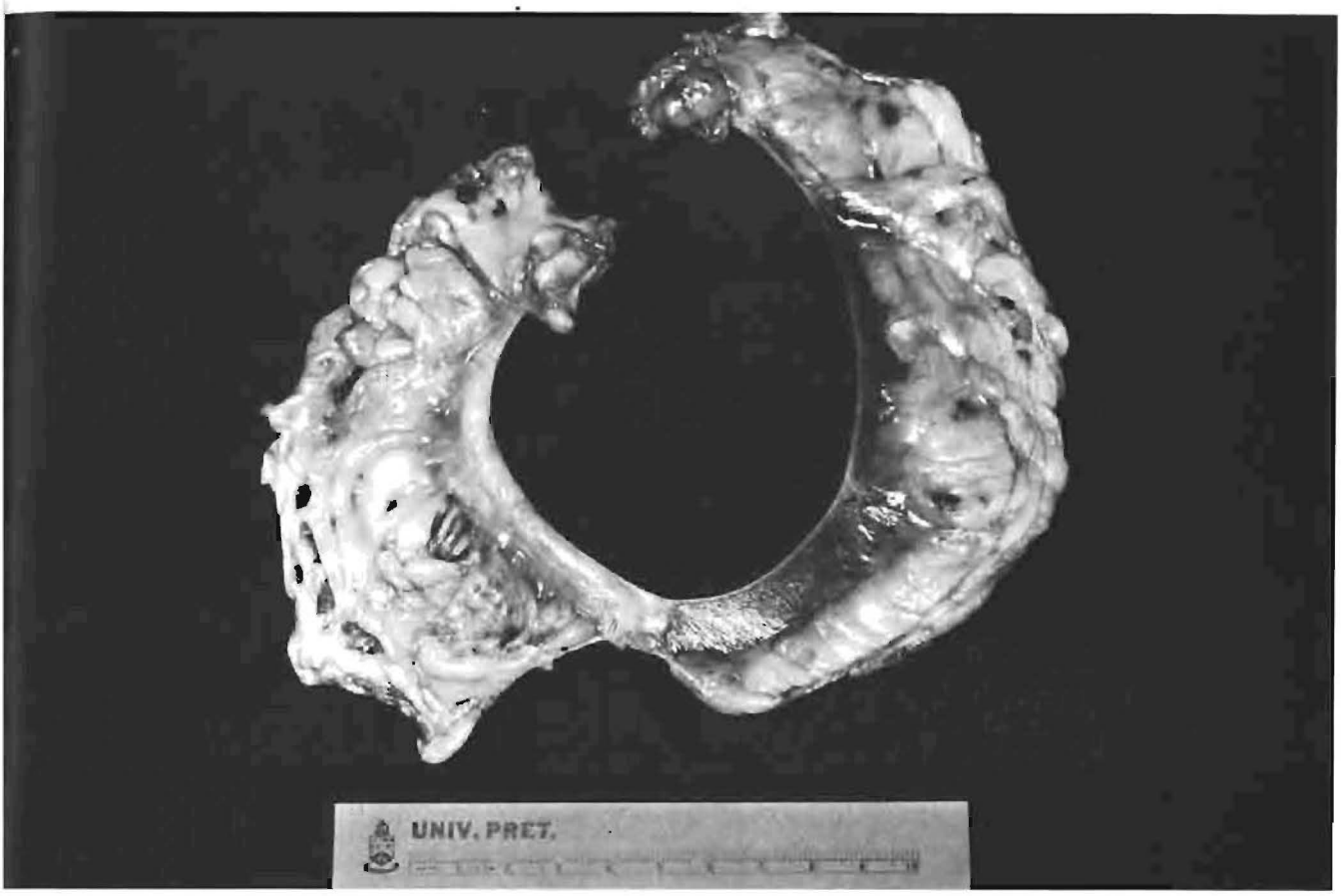

Uterus unicornis in a Red Setter Bitch*

Illustrated is the uterus removed from a Red Setter bitch born n September 1978. The bitch came into season in April 1979 ınd was kenneled for a period of 3 weeks. Following on the ɔeriod of oestrus, she showed swelling of the mammae asso-ciated with possible pseudopregnancy. An ovariohysterec-omy was performed in July 1979.

The right uterine horn was totally absent. Histolocigal ex-ımination of the right uterine ligament revealed loose con-ıective tissue and absence of a Müllerian duct and any uterine issue. Mature *corpora lutea* as well as developing follicles vere found in both ovaries.

According to Roberts[2] *uterus unicornis* is due to a congeni-al absence of a portion or an arrest in development of Mülle-ian ducts. Arthur[2] defines it as a segmental aplasia of Mülle-ian ducts in which only one horn has a lumen, the other ıppears as a narrow flat band. Sweet & Martin cited by Rob-:rts[2] found 3 cases of *uterus unicornis* during ovariohysterec-omies performed on approximately 2000 bitches. Stephenson & Leonard also cited by Roberts[2], felt that the condition night be expected to occur only once in every 5000–10 000)itches.

Submitted by Dr Cheryl M.E. McCrindle, 770 Duncan Street, 0181 Brooklyn, Pretoria and Dr Lucia Lange, P.O. Box 12580, 0110 Onderstepoort.

Uterus unicornis in 'n Red Setter Teef*

Geïllustreer is die baarmoeder van 'n Red Setter teef wat in September 1978 gebore is. Die teef het 'n hitte-siklus gehad in April 1979 en is vir 'n periode van 3 weke op hok gehou. Na die estrus-periode was daar geswolle melkkliere en moontlike tekens van skyndragtigheid. 'n Histero-oöforektomie is in Julie 1979 gedoen.

Die regter baarmoederhoring was totaal afwesig. Histologiese ondersoek van die breë ligament van die regter baarmoederhoring het getoon dat daar slegs bindweefsel teenwoordig was. Geen oorblyfsels van Müller se buis of van enige baarmoederweefsel kon gevind word nie. Daar was volwasse *corpora lutea* en ontwikkelende follikels in beide ovaria aanwesig.

Volgens Roberts[2] is *uterus unicornis* die gevolg van 'n kongenitale afwesigheid van 'n gedeelte van die buise van Müller of die gestremde ontwikkeling daarvan. Arthur[1] definieer dit as segmentele aplasie van die buise van Müller waar slegs een horing 'n lumen het, terwyl die ander as slegs 'n nou plat band vertoon. Sweet & Martin, aangehaal deur Roberts[2] het slegs 3 gevalle van *uterus unicornis* gevind tydens histero-oöforektomies gedoen op ongeveer 2 000 tewe. Stephenson & Leonard, ook aangehaal deur Roberts[2] beweer dat die toestand slegs in een uit elke 5 000–10 000 tewe voorkom[2].

*Ingedien deur Dr Cheryl M.E. McCrindle, Duncanstraat 770, 0181 Brooklyn, Pretoria en Dr Lucia Lange, Posbus 12580, 0110 Onderstepoort.

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AAN DIE REDAKSIE

TO THE EDITOR

Geagte heer,

Die antimikrobiese gevoeligheid van *S. aureus* uit melkmonsters gekweek

Bygaande tabel verskaf 'n opsomming van die resultate van gevoeligheidstoetse wat oor die afgelope 5 jaar in ons laboratorium uitgevoer is op isolate van *Staphylococcus aureus* uit kwartmelkmonsters gekweek. Meeste van die monsters is van melk verkry uit kwarte met subkliniese mastitis (bakteriologiese positief, meer as 500 000 somatiese selle/ml) – slegs in enkele gevalle was daar kliniese waarneembare veranderings in die sekreet te bespeur.

Die antibiogramme is gedoen op triptoon-bloedagarplate waarop die isolate uitgestryk is en wat daarna vir 24h by 37° bebroei is. Die konsentrasies van verskillende antimikrobiese stowwe word op die tabel aangedui.

Dit blyk dat daar geen wesenlike toename in die voorkoms van resistensie teenoor enige besondere antimikrobiese stof in algemene gebruik oor die afgelope 5 jaar was nie. Penisillien G en streptomisien afsonderlik is nog as sulks skynbaar nie die produkte van keuse vir voorkoming of behandeling van mastitis nie. Die voorkoms van bestandheid teen tetrasikliene moet egter in ag geneem word terwyl die geringe voorkoms van ongevoeligheid teenoor die semi-sintetiese penisilliene verdien noukeurig dopgehou te word.

L.W. van den Heever
Afd. Veterinêre Volksgesondheid,
Dept. Patologie,
Fakulteit Veeartsenykunde,
Posbus 12580, 0110 Onderstepoort.

Tabel 1: GETAL EN PERSENTASIE VAN *S. AUREUS* UIT MASTITISKWARTE GEKWEEK WAT *IN VITRO* ONGEVOELIG TEENOOR GESPESIFISEERDE ANTIMIKROBIESE STOWWE OOR DIE AFGELOPE VYF JAAR WAS (% IN HAKIES)

	1975	1976	1977	1978	1979	1975-1979
Penisillien G (1,5 ie)	21 (41,2)	60 (35,9)	90 (41,9)	50 (36,8)	60 (36,1)	281 (38,2)
Streptomisien (10 μg)	14 (27,5)	32 (19,2)	39 (18,1)	29 (21,3)	41 (30,1)	166 (21,1)
Tetrasiklien (10 μg)	11 (21,6)	14 (8,4)	31 (14,4)	15 (11)	17 (10,2)	88 (11,9)
Ampisillien (10 μg)	–*	15 (9,0)	14 (6,5)	–	–	29 (3,9)
Kloksasillien (5 μg)	–	11 (6,6)	3 (1,4)	2 (1,5)	2 (1,2)	18 (2,4)
Chlooramfenikol (10 μg)	–	7 (4,2)	8 (3,7)	7 (5,1)	10 (6,0)	32 (4,4)
Linkomisien (10 μg)	–	4 (2,4)	–	–	–	4 (0,5)
Neomisien (10 μg)	–	3 (1,8)	2 (0,9)	3 (2,2)	2 (1,2)	11 (1,5)
Furasolidoon (50 μg)	1 (2 %)	13 (7,8)	9 (4,2)	10 (7,4)	6 (3,6)	39 (5,3)
Totaal	51	167	215	136	166	735

* – nie bepaal nie

TO THE EDITOR

AAN DIE REDAKSIE

Sir,

Resistance to organophosphorus ixodicides

With reference to the two articles by J.A.F. Baker *et al.* in the 49(4) 1978 issue of your journal, the following comments:

1. In the article on "The current status of resistance to organophosphorus ixodicides by the blue tick *Boophilus decoloratus* (Koch) in the Republic of South Africa and Transkei" the criterion used for resistance to dioxathion is that the isolates have a lethal concentration (LC) 99(%) value equal to or greater than that of the reference Holmdene-Berlin strain (p 328). It needs to be stressed, however, that the LC 99 % for the Holmdene strain is 0,006 (p 330, Table 4), which value is only 12 % of, and thus far below, the recommended field concentration of dioxathion.
   In practice, therefore, all the strain illustrated in Fig. 1, (p 328) with a range of susceptibility between that of a susceptible strain and the Holmdene strain, and many of the strains listed with degrees of resistance greater than that of the Holmdene strain, would be controlled by correct dipping, i.e. would not be resistant in the field.
2. In the article on "Resistance to certain organophosphorus ixodicides in the Bont Tick *Amblyomma hebraeum* Koch, in the Republic of South Africa and Swaziland", the authors consider as resistant those isolates "for which a history of resistance to dioxathion and chlorfenvinphos in the field is available, and for which LC 99(%) values of 0,03 were obtained". In both cases the recommended field strength of these ixodicides is 0,05 % and isolates showing LC 99(%) values of 0,03 would thus be susceptible to field strengths of the dips concerned.
3. The statement concerning amitraz at the end (p 340) of the latter article is not supported by any factual evidence in the article or reference to the literature, and therefore seems out of place.
4. Care should be exercised when making use of the concept "Factor of Resistance (FOR)" in which all isolates are compared with a particular highly susceptible reference strain. There is a distinct possibility that all isolates showing a FOR greater than the reference strain may be incorrectly interpretated as presenting a practical problem of field resistance to the ixodicide in question.
5. The recommended field strength or dioxathion (0,05 %), for example, will give a value of 50 on the FOR scale when related to the LC 99(%) of the susceptible reference *Amblyomma hebraeum* strain used in the article on resistance in this species of tick. The FOR value of 5,1 for the Ubombo isolate (p 340) would therefore be insignificant and would represent, if anything, a low level of developing resistance.

A.M. Spickett
for Director: Veterinary Research Institute
0110 Onderstepoort.

JOURNAL REVIEW TYDSKRIF-RESENSIE

VETERINARY IMMUNOLOGY AND IMMUNOPATHOLOGY

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The emergence of immunology as an identifiable discipline of the biological sciences is a relatively recent development. The veritable explosion in the literature which accompanied this advent is attested to by the formidable number of studies on the subject. Arising out of these investigations was that of immunologically-related disease and the subdiscipline (whether immunological or pathological being open to question) of immunopathology developed. Although much has appeared in the literature on these 2 subjects the material is diffused amongst a plethora of specialised and unrelated journals; the result being the collation thereof, particularly for the veterinary scientist, is both difficult and time-consuming. The purpose of this new journal is firstly, to overcome these problem areas for the veterinary investigator and secondly to provide a medium for the dissemination of information and exchange of ideas in those particular branches of scientific endeavour, but in the veterinary context. These aims are largely achieved in the first number.

Although limited to veterinary immunology and immunopathology the journal is wide enough in scope to claim the interest of the research scientist and clinician as well as the investigator in related fields. Thus, reports on immunochemistry and fundamental research on the immune system of animals is countered by the practical immunological aspects, amongst others, of genetics, neoplasia, transplantation, diagnosis, pathology and prophylaxis.

The articles are reproduced photographically from the original typescripts. While this undoubtedly reduces the time between submission and publication of an article it has the disadvantage of transferring typographical differences as well as errors in spelling and grammar to the end product. However, the concept and contents are deserving of the highest praise and barring this single reservation the journal can be highly recommended to veterinary academic and practitioner alike.

J.W.N.