How far has the study of animals and their diseases influenced the progress of medical research?

By Captain S. C. A. Datta, B.Sc. (Calcutta), M.R.C.V.S.

Mukteswar-Kumaun.

The phenomenon of life in all its forms is intimately connected with its environment, and the life activity of each species of creation is vitally concerned with that of others. The crux of this fundamental biological fact is the food problem. In every living being therefore there is a relentless struggle for existence going on from the very time of conception to death, as each is trying to exist by devouring the other. To appreciate adequately the intensity and extensiveness of this ever-raging warfare requires more than an ordinary acquaintance with the different grades of creation in the economy of nature. Germs and parasites of disease attack man and his animals, and epidemics and epizootics have been known to occur since the earliest times. Humanity has been constantly terrorised by the suffering and murder in individuals and communities perpetrated by disease agents. Thus the instinct of self-preservation and the longing to relieve suffering in fellow men have been the incentive to the study of the intriguing problem of disease (pain, suffering) causation. Animal diseases have been studied simultaneously, primarily from the utilitarian aspect, now enormously extended with the progress of time and civilisation, and occasionally also from the humanitarian urge. Diseases have been so poignant and sudden in their effects that to the terror-stricken ancients they appeared to be supernatural visitations. Commencing from about 500 B.C., diseases have been considered to be processes of nature, but it was not until 2,000 years later that impartial observation of nature was resorted to as the means by which to solve the mystery.

In surveying the progressive evolution of the conception of disease, one notices that the medicine of man and animals was born almost contemporaneously of the common parentage of credulity and empiricism, and that as rationalism dawned, the approaches to the problem of disease widened. The accruing benefit in the saving of life and relief of suffering applied equally to the human race as to the animal species, and the science of disease to-day forms a major study of human welfare. In this study, general principles of pathology emerged showing how changes in the bodily functions (symptoms) and organic alterations in the structure of the special and general tissues composing the living body (lesions) are set up. The concept of cells composing tissues applied to all biological sciences, and the pathological lesions were found to be
the gross structural expressions of the disproportions in the relative values of stimuli and cell reactions (cellular pathology). The complex mechanism of predisposing factors, exciting causes, individual susceptibility and resistance, nutritional bearings, effect of climatic vagaries, and parasitic action were elucidated. Important facts regarding host-parasite relationship, parasites' methods of attack, the hosts' mechanism of partial or complete resistance due to natural factors or acquired through the aid of biological preparations or drugs, factors determining latent infections, carriers and relapses and the bases for prevention and control, etc., have come to light. Apparently trivial, and even isolated discoveries have produced profound amelioration of suffering. Ideas regarding disease control have undergone fundamental changes, and experience has shown that it is more economic and rational to prevent disease in whole communities rather than obtain cures in individuals after allowing maladies to be contracted.

Science of disease has thus marched forward through the ages, but the outstanding fact of immediate concern in this article is that any pioneer work whether that relating to microscopy, protozoa and bacteria by Leeuwenhoek, or that on bacteriology and immunology by Pasteur or Koch, Koch's postulates or his method of plating out of cultures on solid media, or Lister's work on antisepsis or similar other advances have been equally applicable to man and animals, and thus was the essential unity of the healing science brought out constantly. Specialisation in individual branches has no doubt tended to a loss of the broad perspective of comparative medicine, and depressing stages of isolation seen to intervene now and again, but the solidarity of human and veterinary medicine stands out unmistakably throughout the ages.

In the investigation of matters of health and disease, the issues involved have been so intricate and complicated that the human intellect has stumbled many a time. Had it not been for the fact that disease processes in man and animals have been mutually elucidative by throwing cross lights and analogies, the difficulties and obstacles in the understanding of conditions, which appeared to be persistently insuperable, might never have been surmounted, and both human and animal medicine would have been poorer. It may perhaps be unfruitful to try to estimate whether the human or veterinary side has benefited to a greater extent than its sister branch in this alliance of long standing, though as far as therapeutics and immunology are concerned the contribution of research upon animals to human medicine has been of such far-reaching consequences that a rough estimate even is very striking. However, attention need here be restricted to the influence exerted by animal studies on the progress of medical research. The bearing of animals as reservoirs of infection for man and the transmissibility of animal diseases to man either directly or by handling or consuming animal products have been dealt with in another article (Datta, 1938, Agri. and Livestock in India), but it is necessary to note that even in this sphere of animal diseases communicable to
man veterinary researches have been of very great importance in the elaboration of efficient public health measures.

Without exaggerating it may be stated that the knowledge derived from the study of animals and from animal experiments has provided the basis for the development of modern human medicine, whether relating to anatomy, physiology, pathology or therapeutics. This fact is so obvious and yet is not generally appreciated. One knows that ethical considerations have prevented crucial experimentation upon man excepting in the rare instances of convicts, and that essential facts of medicine have emerged largely from analogies from experimental work in large and small animals. Without undertaking the arduous task of giving comprehensive chronological data in this matter, a few of the historical facts, particularly as they relate to the domestic stock, may be recounted. The facts speak for themselves:

The earliest anatomical model known is a clay model of a sheep's liver in Babylonia dated 2000 B.C. The earliest recorded surgical operation is the trephining of sheep for staggers. Blood corpuscles of different species of animals were the subject of early studies. The physiology of digestion, the production of glycosuria, technique of artificial insemination, also of intravenous injection were first worked out in the dog. Transfusion of blood resulted directly from the latter injection. Gland grafting was practised on fowls in 1767, and the effect of the transference of the male and female gonads was also studied on fowls by Berthold in 1849, leading to Steinach's work on the human subject. The antitoxic property of potassium permanganate in snake bites and cholera, the rational application of arsenic preparations, and the use of carbon tetrachloride are some of the drugs adopted from the sphere of animal experiments, besides the animal gland products, pepsin, insulin, adrenaline, pituitrin and others. The still baffling problem of carcinogenesis has received particular stimulus from the experimental work on fowls and dog tumours.

Turning to the domain of helminth parasites, the first one to be described was the parasite of liver-rot in sheep in 1379. Seibold's work on duck trematodes provided the first clue to the life history of flat-worms in general, and the observation in 1780 of strange cysts (hydatids) in the tissues of various domestic animals, and the subsequent finding of similar hooks and suckers in certain tape worms of man, dog and rabbits were of fundamental importance. Feeding experiments in dogs and other animals were carried out and a new method of determining if such cysts are the larval stage of a particular tape worm was evolved. These first achievements in experimental parasitology were soon extended to the production of trichinosis in healthy animals by feeding experiments with infected meat. In 1883 the complete life history of Fasciola hepatica was worked out, though about ten years earlier cercariae believed to be of these parasites had been detected in snails. On these lines
were the life histories of the parasite of the more serious forms of the human schistosomiasis of Japan and Egypt worked out in 1914 and 1915 respectively.

In another branch of parasitology, the first pathogenic protozoan discovered was the *Eimeria stiediae* of the rabbit (1674), but the finding of the equine *sura* parasite by Griffith Evans was of greater significance. More outstanding than this was the demonstration by Theobald Smith and Kilbourne in 1893 that Texas fever of cattle was a form of tick-transmitted piroplasmosis. The role of arthropoda as transmitters of protozoan conditions being thus brought to light, rapid progress followed. Two years later Bruce showed the transmissibility of *Trypanosoma brucei*, also a parasite of domestic stock, through the intermediary of Glossina flies. The immediate result of this was the elucidation of the mystery then surrounding sleeping sickness. Several distinct disease entities due to trypanosomes are now known. Similarly, Ronald Ross's discovery of the transmission of bird malaria through Culex mosquitoes led to the epoch-making discovery of the role of anopheline mosquitoes in human malaria. That spirochaetes, whose exact position is somewhat uncertain, can be conveyed by the bites of blood-sucking insects was demonstrated for the first time in 1903 in the case of fowl spirochaetosis transmission. The intimate relation of protozoology with entomology has consequently become a well-established fact. The profound influence of this relation has been manifested not only in the elucidation of the pathogenesis of many diseases, but the control measures that have been elaborated from a knowledge of the parasite and its vector for dealing with some of the most dreaded of human scourges like sleeping sickness, malaria or yellow fever can rightly be mentioned as the outstanding achievements in tropical medicine.

Sufficient data have been given above to show that besides the addition of individual items of information, veterinary studies have repeatedly thrown floods of light on the solution of many of the fundamental problems of disease causation and control in man, whether pertaining to parasitology or bacteriology. It now remains to be seen how in the latter domain and immunology even large pieces of research connected with human disease problems have been frequently initiated by purely veterinary studies and that the transcending results have materially altered the destiny of man.

Dealing with the highly destructive disease, anthrax in sheep, Pasteur laid the foundation of modern preventive vaccination. The anthrax bacillus was discovered in 1849 in the blood of animals dying from this disease and in 1880 Pasteur discovered the bacillus of fowl cholera. His investigations showed that the anthrax and fowl cholera organisms could be attenuated, and that vaccination of healthy animals with the attenuated germs could be successfully employed to protect against virulent infections. Again the origin of bacterial filters leading to the discovery of filtrable viruses, and of the elaboration of the memorable Koch's postulates can be traced to these early studies upon anthrax. Pasteur's work on these two animal diseases and on swine erysipelas
was carried out when he was nearly sixty years of age. World-wide interest was aroused in the potentiality of immunisation by attenuated germs, and theories to explain the process of immunity were put forward. In 1884-1886 Theobald Smith, one of the pioneers in the field of infective diseases, carried out experiments with hog cholera bacillus on pigeons and established the principle which was to find later a very wide application in human medicine. It was shown that by the injection of filtrated products of the killed specific organisms an immunity against living pathogens can be set up. This was the first experiment on immunisation along these lines, and Von Behring working with Kitasato in Koch’s Institute, Roux and others followed suit. Working with the toxins of diphtheria and tetanus organisms, Behring laid the foundation of anti-toxic immunity. The serum of animals immunised against attenuated diphtheria toxins was successfully used as a protective or a therapeutic measure against diphtheria in experimental animals, and the specific neutralisation of the toxin of the disease was achieved. After the preparation had been tried out in man, the product was produced on a gigantic scale (1894) and with this specific treatment the lives of innumerable children affected with diphtheria have been saved. Many attempts have since been made to treat other specific diseases on the basis of this discovery. The methods employed to-day on large scales for the protection of man against cholera, bacillary dysentery, tetanus, snake poisoning and typhoid fever are all evolved from these early but monumental researches. Wright has extended the method to apply to therapeutic uses. In tuberculosis immunisation, B.C.G. vaccination in calves has largely determined the progress of infant vaccination.

To mention the history of one or two diseases of public health importance, all the essential knowledge on Malta or undulant fever, which is said to occur in North India, has been obtained from animal studies. Bruce in 1906 demonstrated that infection originated from a most unsuspected source, the milk of apparently healthy goats, and in comparatively recent years cow’s milk containing Brucella abortus has been shown to cause undulant fever. In 1888 the mysterious illness of 57 persons who had eaten the meat of a sick cow was traced to what was called Bacillus enteridis.

There have been other ramifications of these studies upon animals. The possibility of the transmission of bacterial agglutinins from mother to offspring was first demonstrated in goats. The final proof of the benefits of Colostrum as providing passive immunity to offspring and reducing the risks of bacterial infections in early extra-uterine life was put forward by Theobald Smith from his experiments on calves, and to-day this is a recognised principle in the post-natal care of infants. Again the other phenomenon of Anaphylaxis was suggested by the remarkable observation of Portier and Richet in 1902 that certain poisons of animal origin evoked in dogs a condition of hypersusceptibility rather than immunity. Anaphylaxis originating from the bacterial products of diphtheria was experienced by Theobald Smith, and this
discovery was designated the "Theobald Smith Phenomenon" by Ehrlich.
In the subject of allergy the experience that certain people would suffer from
an attack of "Horse Asthma" if they went near a stable is one of the earliest
suggestions traceable. Researches on both anaphylaxis and allergy have been
largely concerned with experiments in large and small animals.

Coming to the latest addition to the list of disease-producing agents, the
important group of viruses, the clinical manifestations of some of which in
man and animals have attracted attention from the days of Aristotle and
earlier, rabies and smallpox are the first to come to mind. It is remarkable that
outstanding advances in the immunology of both these conditions were achieved
before there was any clue regarding their etiological agents, and that both of
the diseases to be benefited from empirics should be of virus origin. Regarding
smallpox, human inoculations with variola material were no doubt practised
in India in the days of Atharbaaveda, and the local country tradition that dairy-
maids who had contracted cowpox through milking did not contract smallpox
existed in Gloucestershire in England, and also in France and Germany.
Jenner, however, deserves great credit for converting in 1880 this traditional
belief or observation into a prophylactic principle in science. Turning to
rabies, hydrophobia and its relation to dog bites were known from the earliest
times. Pasteur's first communication on the subject was published in 1882
and the vaccine treatment elaborated by him, though modified, still holds its
ground, but it was not until 1898 that Loeffler and Frosh demonstrated the
filtrable nature of the cause of foot and mouth disease. With the establish-
ment of the virus origin of one disease entity, others were soon added and
bovine pleuropneumonia was a close second. Rabies had been transmitted to
rabbits by Galtier in 1872 and by Pasteur, Chamberland and Roux in 1881.
With the aid of this knowledge, immunisation of man with rabbit-fixed virus
was carried out in 1885, and the fact that the infectivity of viruses can be
modified by passage through lower animals has been a matter of wide practical
application in immunology.

The failure to cultivate viruses in vitro had been a great handicap, and
the fact that most of the human virus diseases are not transmissible to animals
or are not amenable to being carried in series from animal to animal was a
serious difficulty. A constant supply of the causative virus as required for
further investigations could not be arranged, but the demonstration of the
application of ferrets as experimental animals in dog distemper and swine
influenza has led to their use in human influenza researches. Conclusive proof
has been put forward recently that an anti-serum can be produced even from
animals who are naturally insusceptible to the particular virus. The success
achieved in the immunisation against distemper may turn out to be as epoch-
making and far-reaching in immunology as some of the other animal analogies
exploited by Pasteur and others. Regarding artificial cultivation of human
viruses, while all attempts on ordinary media have failed, the success in the
case of bovine pleuropneumonia and agalactia suggests that the growth requirements of this group are peculiar. Again the "virus" of bovine pleuropneumonia has already been seen, and placed provisionally in the family *Actinomyctea*, and filtrable phases of some bacteria, spirochaetes, etc., are known. It may be added that structures, believed to represent the actual virus of certain other diseases, have been seen, and in this connection the demonstration of minute coccoid elementary bodies in fowlpox and vaccinia virus and others is an interesting pointer. The latest finding that at least one animal virus, that relating to Shope rabbit papilloma, is a heavy protein is also of more than ordinary significance.

The Imperial Veterinary Serum Institute, Izatnagar, is now known as the Biological Products Section of the Imperial Veterinary Research Institute, Mukteswar, and is under the control of the Central Government of India.

In 1933 a rectangular piece of land, comprising about 750 acres, was acquired in the plains near Bareilly to relieve the pressure of work of the parent Institute. The site was selected for its various attractions. Besides being a town of historical importance and an old Cantonment, Bareilly is the headquarters of the Commissioner, Rohilkund Division, and the Eastern Command of the Army, with a population of 1,44,031. It is situated at an elevation of 575 feet above sea level and is climatically one of the healthiest plain towns in the United Provinces. It is also a centre of many progressive agricultural and industrial activities, especially sugar, cotton and rapeseed. The most important feature of the district is that the price of livestock animals, more particularly of buffaloes, which are used at this Institute for manufacture of sera, is moderate. The cultivators, for economy's sake, dispose of their plough buffaloes after field work is over, and substitute fresh young buffaloes at the beginning of next season. This routine proves greatly advantageous to the Institute as it ensures the procuring of a large number of young healthy
animals at a moderate price from the village markets organised by the zamindars.

The Institute itself is located about three miles from the Bareilly Town Hall and four miles from Bareilly Junction station and half a mile from the Izatnagar railway station between the Bareilly-Nainital and Bareilly-Pilibhit roads, opposite to the Central and Juvenile Jails, Bareilly, General Offices of the Rohilkhund and Kumaon Railway and Indian Wood Products Company. It is just on the outskirts of the municipal limits and therefore enjoys the combined privileges and facilities of urban and rural life. With regard to the name Izatnagar, it may be mentioned that about thirty years ago the headquarters of the deputy agent of the Rohilkhund and Kumaon Railway were established here, and the railway station was named by the company after the name of their popular agent. The entire area of the Institute is self-contained and is enclosed with a wire fence where plots are specified for residential and office buildings, serum and packing blocks, farm and stack-yards, godowns, veterinary sheds, medical dispensary and cultivation lands.

A circular metalled road starting from the main western gate connects all the residential and office buildings and terminates at the gate in the eastern side, which is chiefly meant for traffic of service and supply. There are many other unmetalled roads as well, connected with the main road and leading to different areas within the Institute.

Administration

The head of the section is designated as the "Officer-in-charge," who is a member of the General Central Service, Class 1, and is directly responsible to the Director at Mukteswar. His technical activities are conducted by two gazetted officers, viz., Veterinary Deputy Superintendent and Head Laboratory Assistant and their technical staff, and the general administration is carried out with the help of the office superintendent, accountant, head clerk and other ministerial staff.

The Veterinary Deputy Superintendent is in charge of the veterinary sub-section, and with his technical staff looks after the treatment, general management, feeding and keeping of all the animals and arranges purchase of young healthy buffaloes from clean areas for the manufacture of protective sera. After segregation of the newly purchased animals for a given period, they are prepared to receive immunising antigens for manufacture of the following sera: (1) Anti-Rinderpest; (2) Anti-Hæmorrhagic Septicæmia; (3) Anti-Blackquarter; and (4) Anti-Anthrax.

The general technical method in regard to production of the above sera consists of (a) immunisation or hyper immunisation; (b) bleeding; (c) collection of serum; (d) bulk storage; (e) biological and chemical tests; (f) bottling; (g) storage; and (h) issue.
A NEW FUNGUS EIDAMELLA ACTONI PARASITIC TO THE DOG.*

By

CAPT. S. DATTA, B.SC., M.R.C.V.S.

INTRODUCTION.

The work described in this paper was started at the suggestion of Lt.-Col. H. W. Acton, I.M.S., with a view to study the relationship between fungoid diseases of man and those of domesticated animals. The clinical materials were obtained from the hospitals attached to the Bengal Veterinary College and the Calcutta Society for the Prevention of Cruelty to Animals. The facilities for laboratory work were kindly provided by the Tropical School of Medicine, Calcutta, and a large amount of guidance and helpful encouragement was received from the Director of the School.

A study of the available literature indicates that knowledge concerning fungoid affections of domesticated animals is very meagre. Several fungi however are known to produce lesions on the skin of the dog. Some are parasites mainly of the dog, that is, they are specific parasites of the dog, but dogs may also get infected from other animals with fungus affections. Four specific fungi have been so far reported in the dog.

Bodin and Almy (1897) isolated a microsporon, studied its morphological characters and established its pathogenic role. It was termed Microsporon Audouini var. canis by them, but later the name was changed by Sabouraud to M. lanosum owing to the woolly nature of its growth.

Constantin and Sabrazes (1893) described a fungus which formed 'capsulated cups' or favus crust on the dog skin, which they called Oospora canina.

Matruchot and Dassonville (1902) discovered two other fungi which they named Trichophyton caninum and Eidamella spinosa. This paper concerns what is believed to be a new fungus, the fifth of its kind parasitic to the dog's skin, which has been called, at least provisionally Eidamella actoni.

Fungoid diseases of the skin of the dog due to the parasites referred to have been called Ringworms or Tineas. The present writer has found that fungal lesions on the dog's skin do not necessarily

* Presented to the Indian Science Congress, 1930, at Allahabad.
take the round, localised appearance of Ringworm as is popularly believed. In man also, affections like Eczema Marginatum, Mange-toe, Cheiropompholyx are known to be of fungal origin but they do not present the clinical picture of Ringworms. From the author’s experience, it is believed that clinical knowledge of dogs is not yet so precise that one can differentiate by means of simple external examination between skin affections due to zooparasites, phytoparasites or due to systemic causes. Microscopic examination is indispensable and it should be profitably supplemented by cultural methods of diagnosis. Different casual agents may produce the same clinical lesions. Lesions and symptoms are the complicated results of a battle between an invading parasite and the body defending itself and no two animals have the same powers of resistance to disease. Again fungus exhibits pleomorphism with alterations in the media of growth, and therefore the clinical picture of a skin lesion is liable to particularly great variation. Accurate diagnosis therefore is only possible from cultural and microscopic findings.

THE CLINICAL DISEASE.

The present investigation was carried out on so-called “worm rashes.” It is recognised by Veterinary practitioners and recorded in text books on Veterinary Science that it is possible for intestinal worms, and particularly the ascarides to set up sufficient systemic disturbance to give rise to extensive rashes on the skin. There seems to be a conspicuous absence of accurate literature on the subject. What the exact relationship of the infesting endo-parasites with these rashes, does not appear to have been worked out.

It was noticed for sometime at the Hospitals already mentioned that recognised methods of treatment for worms failed to cure the “rashes” and a suspicion arose that after all they might not be of such origin.

“Worm rashes” seem to be more frequent and extensive during the rainy months of the year and particularly in dogs that are not regularly brushed or bathed. Occasionally they occur at other seasons, and they have been noticed in dogs apparently well cared for. All ages of dogs are susceptible. Light coloured dogs seem to be more liable, but possibly their lighter colour helps only to draw attention quicker.

The first case met with was in a dog of black colour. It was under treatment for worms. The dog had lesions on the skin which started as rashes, and as soon as some of the lesions healed, others quickly appeared. The rashes were of varying size, from that of a millet seed to that of a three penny bit. Some of the lesions coalesced, whilst others remained discrete. A zone of “reddening” in the vesicular stage was particularly noticeable in light coloured dogs.
Worm treatment failed to result in a cure. The dog was well-nourished and its coat was elsewhere sleek.

The picture of the dog (Fig. 1) shows confluent pustules on the head. The eruptive rashes were scattered all over the body, particularly on the head, neck, back and abdomen. The rashes were easily noticed in places where the growth of hair was sparse and a dirty grey scab was left after the lesions had healed by themselves having been through the different stages of eruptive rashes. In places, clearly defined, hairless, smooth areas of circular shape were seen after the dried scab had come off.

**TECHNIQUE ADOPTED.**

Sabouraud (1892) was the first to indicate a rational basis for the study of dermatomycoses in man or animals. He took up the study of “tinea tondante” of the hairy skin by the observation of the causal parasite in pure culture, obtained by methods as evolved out by Pasteur.

(1) **Microscopic examination**:—Scrapings were obtained after the lesion had been cleaned with absolute alcohol. The scrapings were treated on a glass slide with a few drops of caustic potash of different strengths up to 40%. Mild heat was also sometimes applied. Scrapings were then subjected to microscopical examination at different intervals from a few minutes to 24 hours. The cover slips were lightly pressed in order to break down scrapings slightly for examination purposes. Some fresh scrapings were put under a cover slip and Ponder’s stain, as modified by Dr. McGuire, was run in from the side. The excess of the stain was blotted off and a drop or two of glycerine was run in from the side of the coverslip with a pipette. The slides were then examined under the microscope for mycelia or spores. Neither the caustic potash nor the McGuire method yielded any positive result although the author spared himself no pains in making thorough examinations. It is believed that the negative results were due to imperfect technique rather than to the absence of the fungus in the scrapings.

Study of skin diseases in animals is rendered difficult by the fact that their skin is thickly covered with hair and studded with pigment and hence the initial stages of a lesion are generally lost sight of. It is only after secondary infection by pyogenic organisms has occurred that the hair becomes abnormally erect and matted together, and then attention is attracted. Mistakes are often made by collecting scrapings from prominent and old lesions only. Young progressive lesions should be chosen with care for the collection of clinical materials for cultural examination and this is an important step in the study of dermatomycoses.
(2) Cultural Examination.— Scrapings were obtained from cases suspected of fungus affections of the skin and were washed for about a minute in absolute alcohol and then planted carefully on culture tubes. Cultures on Sabouraud's agar medium were tried with success. A dozen tubes were planted in each case as a routine measure and subcultures were made from successful growths till a pure, uncontaminated culture was obtained. Sporeformers, pyogenic cocci, aspergilli and rhizopus were among the usual contaminating agents. In pure cultures, the growth was just noticeable on the second day but it was on the fourth day only that the growth became more evident and unmistakable. With a view to obtain the most characteristic naked eye appearance of the culture, the pure growth was planted on Sabouraud's medium in an Erhlemeyer Flask (Figure II).

**CULTURAL CHARACTERS.**

Cultures of fungus were obtained in about seven cases of "worm rashes." Excepting one, which appeared to be altogether new, the growths appeared to be either moniliform, faviform or aspergillar and in this one case, which was thoroughly worked out, the fungus had the following characteristics:—

(A) **Macroscopic appearance** :—It was a yellow honey colored growth which was smooth, nondoughy, shiny and waxy in general appearance. Centre was umbilicated, edges were smooth and sharp cut. From the centre, primary furrows extended only two-thirds of that distance (Fig. II).

The rate of growth was about four times as rapid as the human ringworms. Probably this consideration made Sabouraud call the animal ringworms 'Tinea vivaces.'

The growth was studied regularly at the end of 14, 21, and 30 days. In 21 days the growth covered the full surface of the medium provided in the flask. After that, the culture became crumpled and wrinkled due to further growth. The tendency to pleomorphism was manifest after six weeks of growth.

A large number of tubes were implanted for studying the variation, if any, of the fungus on the same medium. No variation was observed. Alterations were then tried in the medium. Dextrose, maltose, glycerine, lactose, fructose and mannite were substituted for saccharose in Sabouraud's medium and De Witt's peptone for Chais-sing's peptone in the same media.

The most suitable sugar for this fungus was found to be dextrose. Fructose gave a more rapid growth. Mannite and lactose gave a slower and less exhuberant growth.
Acton's synthetic medium was found unsuitable for this fungus. It is interesting to add that in the course of these investigations another fungus has been isolated from a calf with 'doughy mange'. This fungus resembles the dog culture to a great extent in naked eye characteristics. Only that the calf parasite was a little more exhuberant in growth and the radial furrows were placed at broader alterations of space. (Fig. III).

(B) Microscopic:—Hanging drop preparations were put up. Maltose agar was first tried as a medium for these preparations. These were examined regularly for two months with the help of the microscope. They disclosed nothing else but mycelial elements. 4% glucose broth was next tried as the medium and was found quite suitable. Simple broth was also tried as a medium for the preparations but it did not give as much satisfaction as glucose broth. Preparations were examined regularly every day and details of the mycological picture as revealed are given below.

Growth started with narrow unbranched refractile mycelia, which later branched out, became broader and developed chlamydospores. With time these spores increased in size and number. In certain centres, the mycelia spread out in a characteristic fashion from a focus. (Fig. IV). The chlamydospores grew larger and the bulging mycelia gave way, liberating the spores. Some chlamydospores were formed at the focus and formed bunches. The mycelia later developed granules round the focus and appeared browner and translucent the brown colour deepened giving rise to black perithecal organs. (Fig. V). The radiating 'spine-like processes' became very long without showing any tendency to curl into tendrils. The calf parasite, already referred to, developed tendrils (Fig. XI).

Apart from the special centres, there were nothing special about the mycelial network elsewhere for sometime. Subsequent to the appearance of the perithecium, a greater tendency to form lateral and terminal chlamydospores was noticed (Fig. VI).

The following were the different elements in the developmental stages of the fungus:

1. Mycelium—simple and ramified, of varying breadth. In its course, shows chlamydospores of varying dimensions, some of rounded, others of elongated shape.
2. Branching was achorion like, in chandelier form.
3. Bodies resembling pectineal organs. (Fig. VI).

These had been noticed in the special focus from which a perithecium later developed. As the special thickening of the mycelial walls progressed, these made their appearance and in further development these were lost in the perithecal body. Matruchot and Dassonville (1899), and Bodin and Alnzy (1902) are of opinion that
pectineal organs should be considered as perithecae that have aborted—a view to which the experience of the writer inclines also.

(4) Chalmydospores;—
(a) intercalary—as in Eidamella spinosa. (Fig. IX). These are formed by encystment of protoplasm of mycelium showing bulgings. Calibre of mycelium increases with size of spores. Spores are of various sizes generally round and large in number. Some of them are more or less dissociated.
(b) dissociated or free spores. (Fig. X).
(c) lateral—not so numerous. Some grow in hyphae, others remain as such. Some are oval, some are even of pyriform. Some are sessile others are pediculated short. Not reported in Eidamella spinosa. (Fig. IX).
(d) Mycelial herniae—peculiar lateral irregular projections from the mycelium.
(e) terminal chalmydospores—macroconidia are rounded of varying size, like those described in Eidamella spinosa.

(5) Bouissons Conidiens—conidian bushes—(Fig. VIII). A mass of hyphae, assembled together longitudinally side by side in bundles, without any trace of enveloping membrane. Hyphae are very fine and branch away from the bundle. Eidam described them in Ctenomyces Sarratus (1883). Also noted in Eidamella spinosa.

(6) Perithecium:—For the purposes of classification, the mode of development of perithecium gives the most valuable aid. Attempt was made to follow up the evolution of these. As already stated, several mycelial branches spread out in an irregularly radiating manner from the focus. The mycelial twigs thicken. Large number round of intercalary chalmydospores are produced. At the focus granules, appear brown to black. Some spores dissociate and lie in the free state. Some intercalary spores grow in all planes like ‘organ nodular. In the still younger perithecum, one can discover a sort of transient wall around perithecae enclosing a central spore-bearing mass. Wall seems to be more resistant in some than in others. The straight branches or spines which grow out of the central mass, do not grow tendrils. The spines are segmented and lengthen considerably, and narrow out almost to a point. The radiating spines are evenly spaced—the space depending upon the number of spines (Fig. VII).

Asci are round and numerous. Perspective view show asci of apparently different sizes and shapes. In all likelihood, they are of the same size and rounded. Hulle cells were not much in evidence.

The parasite seemed to have almost all characters of the other our dogs parasites so far known, as shown below:
(a) Sessile lateral chalmydospores and pectineal organs of microsporn of dog (Bodin and Almy). No fuseaux produced.

(b) Chandelier branching of Oospora canina (Sabrazes). No ‘gemmes’ or favous crust.

(c) Lateral pediculated chalmydospores, mycelial beaklike bulgings, herniae as in trichophyton caninum. Fuseaux absent.

(d) This parasite shows the strongest resemblance with Eidamella spinosa by the production of perthecium and condian bushes. This parasite, however, is sufficiently differentiated to deserve a special name. The character of the spine around the perithecium and the number of the ascospores inside the perithecium differ substantially from those of Eidamella spinosa. Change in the condition of growth have failed to remove these differences.

Experimental Inoculation.

The cultures were inoculated into half a dozen healthy dogs and I was able to reproduce the lesions in all cases and recover the fungus back again in pure culture.

Young healthy dogs were carefully examined. It was made sure that there was nothing the matter with their skin. The back of the neck and sides of the body were chosen for inoculation. The area was shaved and washed with alcohol and ether. A young culture of the isolated fungus was then implanted by superficial scarification. In one or two cases, a drop or two of blood oozed out. The culture was applied dry and rubbed lightly on the scarified wound. An inverted watch glass was fixed to the spot by adhesive plaster bandage. Papules appeared generally on the under surface of the body and they were particularly noticeable in the parts where the growth of hair was sparse. The papules soon changed into vesicles and between the third and the fourth day of inoculation, a large number of pustules in different stages were observed under the neck and abdomen. The pustules had a thick, creamy, greenish yellow pus in them. The next day the hairs on the pustules were matted. The pustules healed by themselves in the next few days, rather rapidly, leaving the clinical picture of what Hutyra and Marek call ‘herpes tonsurans maculosus.’

The experimental production of disease of this pustular kind has been repeated half a dozen times confirming the pathogenic role and identifying this clinical picture of rashes as occasioned by fungus infection. At the seat of inoculation a localised picture of ringworm was however presented after about six to seven weeks.

In one of the experimental dogs, a fox terrier dog of brown and black colour (Fig. XIII) there was observed another interesting
addition in symptoms. Two large blisters or bullae, the size of half a crown or larger, appeared on the lumbar and sacral region of the dog. The hairs were erect and wider apart. When the distended surface gave way the surface revealed a very red appearance, which rapidly changed its character and was covered with thick pus and hair was matted. These healed up spontaneously but the growth of hair on the spots took a period of six weeks to two months. The above is noteworthy as clinical cases are often seen with the above lesions.

TRANSMISSIBILITY TO MAN.

This new fungus appears to be transmissible to man. One of the attendants of the experimental dogs readily developed some ringworm lesions on his arms. The pruritus experienced was very intense and the lesions were of redder colour than is usual with ringworms of human origin, as also observed by Dr. Whitfield of Harley Street, London.

DISCUSSION.

The similarity of pustular lesions in natural and experimental cases has already been noted.

The newly discovered dog parasite is sufficiently differentiated from *Eidamella spinosa*, to which it bears resemblances by the production of its chief characteristics *viz.* perithecium, conidian bushes and ascospores. The spines of the perithecal body (Fig. XII) in *Eidamella spinosa* branch in two spicules and are not segmented. The asci vary from four to eight. The spines of the perithecium in the case of this new parasite are long, nonbranching and clearly segmented. The asci are numerous. There can be no objection to give the generic name of *Eidamella*. From the significant differences already noticed, it is obvious that there is every justification for giving it a new specific name. As I have been initiated into this line of work by Col. Acton, whose work on medical mycology is well-known, I dedicate this parasite to him. This dedication further symbolises that workers in the ‘Science of Disease’, Medical and Veterinary, can gain much by active co-operation. The new parasite therefore, is christened *Eidamella actoni*.

CONCLUSIONS.

1. As in man, so also in dogs, conditions occur which do not present the naked eye ringworm picture but are caused by fungi nevertheless.
2. Cases of so-called “worm rashes” are not necessarily connected with entozoa. Fungi can also produce similar lesions.
3. A new fungus, parasitic to the dog, probably transmissible to man, has been discovered and named *Eidamella actoni*. 
EIDAMELLA ACTONI.
EIDAMELLA ACTONI.
4. Study of mycoses offers a very interesting field for research and thought in India—particularly in moist and hot Bengal. Fungi undoubtedly play a part in many obscure conditions with which we meet.

REFERENCES.

(2) Sabrazé's—Sur les Favus de l'Homme, de la poule et du Chien Paris 1893.
(3) Bodinet Almy—Le microsporum due chien Rec. med. Vét. 15, Mars 1897.
(7) Hutrya and Marek—Sp. Path and Thera of dis. of animals.
(10) V. Record, July 20, 1929—pp. 619.

EXPLANATION OF ILLUSTRATIONS.

Fig. I. Photo of the dog showing confluent pustules on the head. Eruptive rashes under the neck and abdomen were most discrete.

Fig. II. Dog culture in Sabouraud's agar showing a week's growth. Notice umbilicated centre, with smooth sharp edges of the growth. The primary and secondary furrows are also shown.

Fig. III. Culture from calf with "doughy mange," one week old, notice more exhuberant growth, the radial furrows are placed at broader alterations of space.

Fig. IV. Photomicrograph of culture in glucose broth in hanging drop preparation—growth is stunted due to drying of medium. Mycelia are seen radiating out irregularly from certain focal centres. Inter-calary chlamydospores have been formed. Bottom of the field shows nonbranching spines of enormous length spreading out from the tangled manes of perithecal colonies. Spines do not branch or curl.

Fig. V. Photomicrograph:—Tangled masses of perithecal colonies are seen more prominently. In the centre is the unpigmented spore-bearing mass—crowded with numerous rounded spores. The apparent black pigment is due to the appearance of numerous brown granules in the spore, colonies.

Fig. VI. Photomicrograph:—Hanging drop culture shows bulging in the segmented mycelial hyphae. Discarded mycelia appear as contracted, hazy threads in the background. Two pectineal organs are seen in the left part of the field. Branching is 'achorion' like. Intercalary, lateral or terminal chlamydospores are in evidence.
Fig. VII. Photomicrograph of individual black perithecal bodies with their long, radiating spines. The mycelia being out of focus are rather hazy.

Fig. VIII. Bouissons Conidiens—Reproduced from Matruchot et Dassonville, Rec. de med. Vet. 1901.

Fig. IX & X. Photographs of drawing—show all the different elements met with in the developmental stages of *Eidemella actoni*—diagrammatically represented. Only 'Bouissons Connidiens' are not shown here.

Fig. XI. Microphotograph of an individual perithecium of the calf ringworm showing the characteristic curling of the tendrils at one end. The spines are seen as straight segmented structures, radiating out in all directions.

Fig. XII. Perithecium of *E. spinosa*—spines branch into spicules and are not segmented—Reproduced from Matrochot et Dassonville—"Sur les Téignes du chien."

Fig. XIII. Photograph of a fox terrier dog: Two large blisters are seen on the back. The young culture was implanted on a scarified area at the back of the neck,
REPRINTED FROM

THE INDIAN JOURNAL OF VETERINARY SCIENCE AND ANIMAL HUSBANDRY

Vol. II., Part II, June 1932.
THE ETIOLOGY OF BOVINE NASAL GRANULOMA

BY

CAPT. S. C. A. DATTA, B.Sc., M.R.C.V.S.,

Temporary Veterinary Research Officer, Imperial Institute of Veterinary Research, Muktesar.

(Received for publication on 2nd April 1932.)

(With Plates IX-XII.)

INTRODUCTION.

There exists in India a peculiar chronic disease of the nostrils of cattle which in certain areas affects large numbers of animals and causes considerable loss to cultivators. The disease is known to exist in Assam, Bengal, Bihar and Orissa, Bombay, Central Provinces, Madras, Mysore and probably also in Sindh. It has existed in certain places for decades but it shows no tendency to spread as an epizootic. The condition would appear to be confined to India as no reports are available from any other part of the world. The peculiar immunity of the buffalo against this nasal affection has been particularly commented upon in every province of occurrence from time to time. Interesting information is available from a certain enzootic area of the Bombay Presidency that although nearly all the animals in the village are watered from a common watertrough, no horses or buffaloes have ever been known to be affected with the disease.

Although there is little doubt that this condition has existed in India for a long time, the earliest printed reference in English to this so-called "Snoring Disease" would appear to be by Jeya Singh Raj [1910]. Krishnamurti [1922] was the first to demonstrate certain eosinophile, rosette-form bodies in histological sections, and he contended that the disease was a form of actinomycosis, since each of these bodies was considered by him to be a characteristic "ray-fungus". Cooper [1923] confirmed the above histological findings by studying tissues obtained from various provinces and further he [1923 and 1931] recovered repeatedly in pure culture a streptothrix organism from the lesions. Extensive experiments were carried out by him [1925] to transmit the disease artificially to healthy cattle, by housing in contact with naturally affected cases and by infection in various ways with a streptothrix isolated culturally from the lesions but with negative results. Das [1929] also carried out experiments in the field regarding the mode of infection and obtained negative results. With regard to treatment, Das [1929] showed that
although Potassium Iodide exerted no beneficial effect, the curative value of Tartar Emetic, as claimed by Parthasarathi Naidu [1921] when working at Saidapet in the Madras Presidency, was undisputed.

In commenting on the failure of Potassium Iodide, Edwards [1929] stated:—

“It may be that the condition is not in reality a streptothricosis, in spite of the fact that granules apparently indistinguishable from those of actinomycosis are discoverable in the lesions”. Cooper [1931] investigated a large outbreak in Jubulpore, a description of which has been recorded by Oxspring [1931], and summarised the position, on that date, concerning the etiology of Bovine Nasal Granuloma by saying that “since the disease is definitely amenable to treatment with Tartar Emetic, and this drug is of great value in the treatment of some protozoan affections, such as Kala Azar and Surra, it is reasonable to suppose that Bovine Nasal Granuloma may be due in reality to a protozoan organism. In 1927 Krishnamurti detected cases of nasal granuloma in cattle which were definitely due to a Rhinosporidium, regarded by some as a member of Protozoa, and certain facts have recently been collected by us indicating that it may yet be possible to correlate all the lesions found in cattle, including those at present believed to be due to streptothrix organisms, with Rhinosporidium as their cause”. In passing, it may be recalled with interest that Christopherson [1918] was the first to point out the almost specific action of Tartar Emetic in human bilharziosis in the Sudan.

The object of the present article is to record certain definite finding of the author in support of the view that Bovine Nasal Granuloma is a clinical manifestation of Schistosomiasis, where the worm seeks out the nose for the deposition of its eggs and, as will be shown later, this view will readily explain all the known facts about the disease. It must be made clear here that there exists in India another separate and distinct affection of the nose of cattle and horses, namely, Rhinosporidiosis as described by Krishnamurti [1927 and 1931] and confirmed by Professor Ashworth, the eminent Zoologist of Edinburgh, in a private communication. Nasal Granuloma proper can however be easily differentiated by its naked eye appearances and the specific microscopic features. Lesions of Nasal Granuloma are elevated nodular masses, intimately connected with the subjacent tissues by a broad base. Lesions of Rhinosporidiosis, as pointed out by Krishnamurti, are more fragile and pediculated being composed of softer and more spongy tissues.

Confirmation of the diagnosis, initially made from the gross characters, can easily be obtained by microscopic examination by finding the characteristic rosette-shaped “actino” bodies in the former and the chitinous sporangium with spores in the latter.
PLATE IX.

Fig. 1. Photograph showing growths on the mucosa of the lateral wall of the nasal chamber in its anterior third.

Fig. 2 (× 400). Microphotograph of the "Spindalis" type of egg, found in the nasal discharge obtained from clinical cases at Muktesar. Note that one end is pointed and the other blunt.

Fig. 3. Microphotograph of a pair of adults in copula in the fresh state, removed from the growth from a clinical case at Muktesar.
Clinical Disease.

A constant running generally of both the nostrils, associated with noisy breathing and an occasional sneezing serves as the first indication of the disease. Apart from the symptoms of snoring and respiratory distress, which are markedly increased at work, there is no evidence of systemic ill health. The character of the nasal discharge varies with the development of the disease, being either thin serous, thick mucous, muco-purulent or sanguineous. The lesions of the disease first make their appearance at the alar margin of the nose and gradually make inroads posteriorly. Although the condition is a localised disease of the anterior nares, which is extremely well-suited for the extrusion of the mature egg, in advanced cases the middle region or even the posterior region may be encroached upon. At the Museum of this Institute, there are mounted specimens of Nasal Granuloma showing growths (1) on the mucosa covering the septum nasi to about the middle, (2) on the turbinated bones to near their posterior limit, and (3) on the mucosa of the lateral walls of the nasal chamber in its anterior third.

Plate IX, fig. 1 shows the last-mentioned specimen, which was obtained through the courtesy of Mr. W. Harris, M.R.C.V.S., Superintendent, Civil Veterinary Department, Assam. Initially the nasal mucosa shows patches of congestion and exceedingly minute vesicular and papular elevations, which develop into rounded granular patches of inflammatory thickening. Palpation of the abnormal surface gives one the impression of roughened crocodile skin. On the “mulberry like” processes of granulation, yellowish areas are seen, the surface epithelium of which gives way producing minute shallow ulcers, through which the ova make their exit. In the course of the present investigations, the writer had the opportunity of examining at Muktesar six clinical cases of Nasal Granuloma, three of which were obtained from Jubbulpore in 1930 and the other three from Bihar in 1931. Examined under the microscope, a caustic potash preparation made from the shreds of mucus contained in the nasal discharge of affected animals reveals varying forms of Schistosome eggs, and young forms of the parasite. In addition to the above-mentioned six cases, nasal discharges from 3 clinically affected animals at Jubbulpore have recently been received and examined and eggs of the “Spindalis” type have been seen in each case.

Examination of the urine reveals certain rudimentary forms of the parasite but the faeces have been found on the other hand to be negative. It is to be noted that no mature ova have been discovered in the urine and no symptoms of bladder affection have been observed. The urine from these animals has been negative to the benzidine test for blood.

Roughly speaking, the varying ova found in the nasal discharge were (a) a non-spined globular egg (b) a rounded or ovoid egg with a single terminal spine which
may be short or long, slender throughout the length or broad at the base (c) an ovoid—elongated egg with double terminal spine (d) an egg of the "Spindalis" type (Pl. IX, fig. 2.), as first described in India by Montgomery [1906] and also found by Vriberg [1906] in Sumatra. The exact significance of so many types of eggs is not clear.

Young, developing adult worms were also seen in the nasal discharge. The young males are short and tuberculated with a prominent ventral sucker, which in most cases being of yellow colour makes detection easy. The ventral sucker approaches the anterior end of the worm, as it develops. The young females are rather long and filiform.

Adult Schistosomes as seen in Pl. IX, fig. 3, have been removed at Muktesar from the diseased tissue. This was removed surgically by slitting open the nose, and deep sections were removed from the most posterior portion of the growths as they were most likely to contain the healthy adults. An adult male was first isolated from the blood and other fluids collected during the surgical operation.

**PATHOLOGICAL HISTOLOGY.**

The material for histological study was obtained from specimens, which had been collected at this Institute from about 12 cases of the disease from Assam, Bombay, Mysore, Bihar and the Central Provinces from 1922 onwards.

Although other forms of bilharziosis, such as urinary and intestinal, are frequently associated with a tendency towards malignancy and the formation of polypoid growths, the essential histology of Bovine Nasal Granuloma is simply that of granulation tissue, the exact character of which depends upon the degree and the duration of the infection. An early growth is made up of very vascular young connective tissue, fibroblasts in activity being in evidence. Old tumours are not so highly vascular and contain a considerable amount of well formed fibrous tissue. Eosinophilia, which is a notable feature of Nasal Granuloma, may be localised in the periphery of the follicles, to be described later, or may even be diffusely scattered.

The epithelial covering of the growth is nearly completely intact excepting for areas showing ulceration. The epithelium covering active lesions shows inflammatory softening and may be actually thinned down due to the pressure exerted by the ova attempting to work their way out. (Pl. X, fig. 1.) Occasionally the apices of the epithelial papillae are found infected and may show degenerative changes. It is primarily in the submucosa that the ova are deposited in quite appreciable numbers, determining the nodular vascular masses of simple elevations of the mucosa which characterise the disease. The submucous connective tissue is rather loosely disposed and may show channel formations.
Fig. 1 (×200). Microphotograph of a section of the growth showing the epithelium thinned down and raised due to the pressure exerted by the subjacent ova, about a score of which can be seen here.

Fig. 2 (×80). Microphotograph of a section of the growth showing follicles with giant cells and "granules".
The most characteristic microscopic lesions of Nasal Granuloma are represented by the follicles (Pl. X, fig. 2), the central core of which presents what has been considered by Krishnamurti [1925] to be "actinomycotic granules" surrounded by a characteristic "ray fungus" or "club" formation. The lesions are observed to follow a sequence of events, starting with a destructive abscess around the offending parasitic mass. The individual microscopic lesions that are formed in this manner are nodular abscesses, which are mainly cellular in character, being composed of small round cells, endotheloid cells and polymorphonuclears. Having failed to extrude the irritant by a destructive process, an attempt to completely encapsulate the irritating ova is made and the process of repair as manifested by eosinophile cells and fibroblasts sets in. In places certain follicles are seen being exclusively composed of small round cells without any remnant of the parasitic element being discoverable in the centre. The appearance of some follicles would appear to be due to the section having passed through the periphery of the nodule. In early follicles, the young ova are seen surrounded by many layers of typical endotheloid cells with the formation among them of huge giant cells immediately surrounding the ova or in their neighbourhood. Evidence of degeneration with diffuse staining and broken down nuclei is seen in the centre of some follicles, in others the parasitic mass has been removed with a few giant cells still persisting, notwithstanding the maturity of the fibrous tissue supporting the follicles. In advanced lesions, the ova undergo calcification or are resorbed or totally digested by the "foreign body" giant cells and no eosinophilia may be present in the altogether fibrosed nodule. With the simultaneous fibroblastic activity and eosinophilic infiltration, concentric rings of fibrous tissue gradually invade from the periphery inwards until the lesion may be considerably fibrosed. From the above description of what have been designated as follicles by Krishnamurti [1925], in association with the presence of "foreign body" giant cells, it will be observed that the follicles appear to be identical with "a typical bilharzial pseudotubercle" of Fairley [1920].

Now in regard to the peculiar bodies, "the clubs", Krishnamurti [1922] has considered them to be specific and of causal significance. These bodies are quite gram-negative but stain well by Plaut and other methods used for actinomyces. The "clubs" or as the present author prefers to call them, the "actino" bodies of Nasal Granuloma present certain definite peculiarities as compared with those of classical actinomyces. Unlike actinomyces, the central cavity within the "actino" body of Nasal Granuloma is definitely bounded with a thick, highly refractile capsule of hyaline nature and contains no gram-positive microorganisms or streptothrix filaments but reveals nuclei of the parenchymatous cells of the enclosed larva instead. This central cavity takes varying shapes up to that of a
"Spindalis" type of an egg and contains in most cases a granular body or may even be empty, depending upon the angle at which the egg is sectioned and upon whether the enclosed larva is sufficiently differentiated for well defined structure to be recognisable inside it. In some shells, however, definite structures of the parasitic embryo can be seen.

The author agrees with Colebrook [1930] who states:—"The first point—and it is not at all new—is that the formation of "clubs" is not a specific character of mycelial infections. Thirty years ago it was shown that clubs were formed around a mass of dead tubercle bacilli implanted into living tissues and since then this has been demonstrated in many other circumstances." It is now known that the actino-bacillus, staphylococcus and B. pyogenes can produce "clubs". Recently Beaver [1931] has found a colony of staphylococci in the centre of a small abscess assuming morphological resemblance to Actinomyces hominis. The present investigation and another by Yamagiwa [1931], which is referred to below, have revealed that the same reaction in the shape of "actino" bodies can be produced also in helminthic infections, such as by Schistosomes. As "actino" bodies may be present in such divergent conditions as detailed above, it is clear that they cannot be considered specific. In what the author considers an earlier stage, the capsular body containing the parasitic mass is seen encircled outwardly with a pale blood-tinged fluid and later with certain changes the capsular body is encircled with numerous closely adjacent eosinophile club-like processes arranged in a radiating manner. The exact significance of the eosinophile "actino" bodies is not clear but it is undoubtedly a manifestation of the host's reaction and attempt at delimiting the ravages of the parasitic worm or of its toxin. In dealing with Madura disease and Actinomycosis, Kanthack [1893] has expressed the same opinion thus:—"Clubs are only formed when there is evidence of resistance on the part of the tissue towards the microorganisms. In rapidly spreading and extensive cases, little or no club formation may occur." Yamagiwa [1931] has encountered a similar reaction of "clubs" outside the parasitic shell in the study of lesions caused by Schistosoma tur- kستانium. The interesting figures of degeneration of the egg shell given in support of his paper form an illuminating study, as they present a great resemblance to the like structures encountered in Nasal Granuloma. In a notable contribution on the morbid histology of Onchocerciasis, Shafi Mohammad [1931] has made the suggestive statement:—"Giant cells here appear small and more compact and take various shapes, thus becoming flattened or radiating in close apposition to the cellular and fibrous elements around. This is due to the pressure of the growing fibrous tissues around them and also the counter pressure of the capsule of the tumours. Their protoplasm is not so eosinophilic as the above mentioned giant
Fig. 1 (×400). Microphotograph of the "Spindalis" type of egg in the tissue, showing the blunt and the pointed ends. Note its similarity with microphotograph No. 2, Plate IX.

Fig. 2 (×400). Microphotograph showing a fully matured miracidium still attached to the spiny portion of the egg, lying in the space left by the egg. The caudal extremity of the embryo can be seen inside the eggshell.

Fig. 3 (×400). Microphotograph of an "actino" body in section. The central cavity contains the cellular elements of the embryo.
Fig. 1 (×400). Microphotograph of a transverse section of male worm in the tissue showing cuticular tubercles.

Fig. 2 (×80). Microphotograph of mature worms in copula lying in the tissue. No remnant of vessel endothelium or glandular epithelium is recognisable.

Fig. 3 (×120). Microphotograph of a deep section showing mucous glands and blood vessels, which contain several pairs of worms in copula.

Fig. 4 (×400). Microphotograph showing a gland, impacted with eggs and the commencement of changes for the production of a pseudotubercle.
Etiology of Bovine Nasal Granuloma

Cells and their nuclei are pressed together into one or more solid masses and take the stain deeply."

In the different parts of the matrix of the growth, empty spaces of various shapes, rounded, oval, or spindle-shaped, are occasionally seen. These are presumably a kind of mold left by the eggs. The resemblance of the shapes to those of the actual ova discovered in the nasal discharges is worthy of note. The matrix reveals groups of embedded eggs or empty irregular capsular shrunken shells. Various stages of the embryo within the egg shell can be made out. Some appear to be fully developed, other show a distinct segmenting embryo whilst some egg shells are altogether collapsed and contain little or no organised material. Judging by the appearance, shrunken and blackened shells with dark amorphous content would appear to be eggs that have died due to unfavourable conditions. Inside the shell of viable eggs, a distinct envelope (vitelline membrane) may be seen enclosing the embryo. Some ova contain cells staining at the periphery, while others show cells staining at the centre. In one ovum, at least, a fully matured, miracidium was seen still attached to the spiny portion of the egg shell (Pl. XI, fig. 2). In some sections, spaces which were originally occupied by the eggs are seen occupied by mature or immature miracidia, a few of which are seen as leaf-shaped bodies with an anterior cone, eosinophilic cephalic glands and a distinct constriction between the cephalic and caudal regions. Some miracidia were observed to have a blunt caudal extremity terminating abruptly, and these presumably were the females.

In deeper sections, many coiled mucous glands and large blood vessels are included. Some of the glands are apparently normal although containing some discharge. Others may show hyperemia. The glandular epithelium quite frequently is noticed to undergo fatty degeneration and vacuolisation in many places. In two samples of histological tissue, the discharges in the nasal gland have been found to contain typical Bilharzia worms. Some glandular tubules were definitely impacted with clusters of ova (Pl. XII, fig. 4), which would appear very suitable to form the nucleus of "bilharzial pseudo-tubercles". It is difficult at times to say definitely whether a pseudo-tubercle was initially formed by the deposition of eggs in the tubules of nasal glands or within dilated capillaries or in the matrix of the connective tissue itself.

Apart from the changes in the connective tissue of the nose described above, the blood vessels show marked lesions in the shape of varying degrees of endarteritis, the lumen of some being altogether obliterated. The blood vessels in deeper sections are generally larger and contain mature healthy worms, either singly or in copula (Pl. XII, fig. 3), and may present considerable dilation and degrees of
end-arteritis. As the worms in these vessels appear quite healthy and undegenerated, the changes in the endothelium and the wall of the vessels would appear to be due to bilharzial toxins. It is interesting to note that the lower margin of the field in a microphotograph (Fig. 2, Plate V) in Krishnamurti's [1925] Memoir shows what would appear to be a healthy Schistosome worm. Some blood vessels reveal a perivascular reaction. If the mature worm or embryo undergoes death or degeneration, the parasite remnants serve as the commencing nucleus for thrombus formation. With an attempt at organisation of the thrombus so formed, the lesion in the blood vessel involves the other coats as well and is not restricted to the endothelium alone. The stages in the formation of pseudotubercles, characterised by huge giant cells, eosinophiles and fibrosis as already described, are also seen in the case of a capillary vessel containing bilharzial elements—the affected vessel finally losing its identity. A certain number of vessels in the growth have been observed to have a disrupted wall resulting in hemorrhage into the surrounding tissue. It is believed that by means of the spine of the egg and due to the histolytic substances elaborated by the enclosed larva and exuded through the egg shell, a way into the perivascular tissue is made. The ova probably find their way in this fashion into the extra-vascular tissue, although it seems possible that an occasional mature female may stray into the tissue outside the vascular system and sometimes lay the eggs in situ.

**DISCUSSION.**

Earlier in the paper it was stated that the view that Nasal Granuloma proper is a form of Schistosomiasis readily explains all the known facts regarding the condition. These facts may be summarised as follows:—

(i) Heavy accumulation of eosinophile cells in sections.

(ii) The occurrence of peculiar "actino" bodies or "clubs" as a reaction against parasitic capsular shells.

(iii) The superficial formation of characteristic small granulations without involvement of the tissues of the tongue and jaw, and the chronic nature of the lesions.

(iv) Enzootic nature of distribution.

(v) Failure of experimental transmission of the disease by contact, and by treatment with cultured Streptothrix or with diseased tissue.

(vi) Failure with Iodine treatment and almost specific response to Tartar Emetic.

With regard to the identity of the parasite, it has been stated that the "spindalis" type of egg has been seen in the tissue and in the nasal discharges of
affected animals, and the adult parasite also resembles *Schistosoma spindalis*, Montgomery, but unfortunately fully mature specimens have not yet been obtained so that a definite opinion on this point cannot yet be expressed. However, certain facts have come to light which suggest that it may be a new species. It is interesting for example, to note that Fairley and Mackie [1926] conveyed infection of *Schistosoma spindalis* to buffaloes, which they believe to be the natural hosts of the parasite, but the buffalo appears to be strongly resistant to attack by the parasite of Nasal Granuloma.

Liston and Soparkar [1918] and other workers on *Schistosoma spindalis* infection mentioned above, have not encountered eggs of any other type than "Spindalis," although these workers have studied infection by this parasite in such animals as cattle, young goats, guinea pigs, buffalo and the monkey (*Macacus sinicus*). They have also stated that *Schistosoma spindalis* affects only the alimentary tract or its appendages and its ova are voided solely with the faeces.

Further investigation into the specific identity of the parasite and its intermediate host, together with the reproduction of the disease, drug therapy, and prevention will be taken up as opportunities occur.

**Conclusions.**

A few cases of Rhinosporidiosis of cattle have been recorded in India, but Bovine Nasal Granuloma proper is a separate entity and is a Schistosomiasis.

Nasal Granuloma presents an altogether new localisation of Schistosomiasis, as compared with the hitherto known urinary and intestinal forms.

The parasite resembles *Schistosoma spindalis*, Montgomery, but certain differences, which are under study, have been observed.

The true nature of the "granules" previously described in this condition has been shown to be closely connected with the invading parasite, which sets up reactions, resulting in the formation of the so-called "follicles" and actino-bodies.

Examination of nasal discharges in caustic potash preparations should be of great value in the diagnosis of the disease in future.

With the definite information now available regarding the etiology of this condition, further experiments with the intravenous administration of such drugs as Tartar Emetic, Emetin, Antimosan, etc., are indicated.

**Acknowledgments.** My thanks are due to Mr. G. D. Bhlerao, M.Sc., Helminthologist at this Institute, for giving me the value of his expert opinion and confirming my findings with regard to the parasitic elements discovered.
In conclusion, I take this opportunity of placing on record my great indebtedness to Mr. F. Ware, F.R.C.V.S., I.V.S., the Director of this Institute, for his keen interest and helpful encouragement and for affording me all facilities, without which the present investigation would not have been possible.

REFERENCES.

Beaver, D. C. (1931). Granulomatous abscess of the liver of pyogenic origin—Plate 50, Fig. 9, Abh. Ji. Path. 7, 3, p. 276.


Kanthack, A. A. (1893). Madura disease (Mycetoma) and Actinomycosis, JI. Path. and Bact. 4, p. 110.


INTRODUCTION.

The history of the disease condition of equines in India, commonly known as Bursati, dates back for more than 100 years when Kerr [1829] published what appears to be the first article on the subject. In the intervening period numerous papers have appeared with the object of elucidating important aspects of the disease, such as the etiology, pathology and treatment, but it is noteworthy that workers as yet are not agreed as to the precise nature of the causal agent or the mechanism of infection. The present investigation was, therefore, commenced at the suggestion of Mr. F. Ware, F.R.C.V.S., I.V.S., Director of this Institute. Army officers were circularized for specimens of Bursati tissue, and Veterinary authorities in different countries, where allied conditions exist, were also addressed, as a result of which some interesting information was collected from Australia, the Argentine, Greece and Egypt. It may be stated here that in an earlier article entitled "Histopathological Studies on 12 cases of Helminthic Granuloma of the Equine Skin", which was read before the 20th Session of the Indian Science Congress, held in Patna in January 1933, a preliminary note on the present investigation has already been recorded.

With regard to the etiology of the condition, the views current at present may be included in one or other of two well-defined hypotheses, viz.:—(1) that it is of mycotic origin, being caused by some species of fungus which finds its way into the horse's skin in a manner not yet understood; (2) that the disease is caused by helminth parasites deposited in pre-existing sores by flies.
Of these two theories, the former is the older, being first suggested by Jackson [1842], but actual experimental data in support of this were only later put forward by Smith [1884], Steel [1885], Burke [1892], and finally by Holmes [1914]. For the purposes of this article, the work of the latter two workers only need be recounted. In attempting to prove that this condition was an epithelioma, Burke [1880-1888] most vehemently criticised the mycotic theory but again a few years later [1892] he was emphatic that the disease was a form of actinomycosis. Holmes' view was that it was a form of sporothricosis resembling a disease of the horse and the mule described by Carougeau [1909] in Madagascar, since a chalky white growth, resembling closely the fungus *Sporotrichum minutissimum* was repeatedly obtained in culture by him from Bursati tumours, kuskurs, and direct from the blood of horses affected with the disease. Experimental inoculations carried out by him with cultures of this fungus yielded only negative results. As a result of a preliminary study of the condition in Calcutta in 1929, Datta [1931] also recorded certain experimental data from which it appeared that the actinomycotic theory was the more plausible, since a strain of actinomycoses, resembling Holmes' culture in morphology, was isolated without difficulty from Bursati lesions, and because the disease could not be transmitted experimentally through the intervention of flies, which were allowed free access between affected subjects and healthy equines, with unprotected cutaneous wounds in various parts of the body. It may be noted that another similar condition designated as 'leaches' affecting equines in America, is considered by Theobald Smith [1893-94] as being caused by a fungus, similar to that concerned in "Madura foot of India", the yellow granules of the equine disease being identical with what are called 'Sulphur granules' of actinomycosis. With regard to Holmes' work, one cannot overlook the fact that he does not put forward any mycological data on which the identification was based, for, there is nothing in his description to indicate that the characteristic lateral spore-clusters were observed with his fungus. Further if his theory were correct, one should expect to find nodules spreading along the course of lymphatics with lymphangitis and lymphadenitis, but it is known that these do not constitute the clinical features of Bursati.

The helminthic theory was first put forward by Lingard [1905], who referred to the presence of a "felted aggregation of nematode worms" in the inner layer of a Bursati sore but, as Holmes has pointed out, "there is an indefiniteness about Lingard's statements which fail to make clear whether personal observations were being recorded or deduction made from the literature of other writers on the subject". Further in questioning the identity of Lingard's "felted aggregation", Gaiger [1915] has adduced evidence to show that they represented, in reality,
merely muscle fibres. The latter worker would seem justified in this view, because there is authentic evidence that such a mistake is not unknown and has actually been made by other workers in the experience of the writer of this article. Again, a confusion such as this seems probable when one remembers that Lingard may have received a suggestion, regarding the possible occurrence of a nematode or its debris in bursati tumours, from his knowledge of the description of similar structures by Rivolta [1868] and Laulanie [1884] in connection with a similar condition in Europe, viz. granular dermatitis.

The evidence in favour of the helminthic theory of Bursati in India may be summarized as follows:

(1) Careful investigations have shown that conditions clinically resembling Bursati, viz.:—Summer Sores in Europe [Railliet and Henry, 1915; Spitz, 1920], Swamp cancer in Australia [Bull, 1916 and 1919], Esponja in Brazil [Descaszeaux, 1915] are set up by the larva of certain spiroptcrous worms (Habronema spp.), which undergo a part of their life cycle in the bodies of flies (Musca domestica for Habronema musca and H. megastoma, and Stomoxys calcitrans for H. microstoma) and their normal adult life in the stomach of equines. [Hill, 1920.]

(2) As shown by Sen [1927], in a short review of the literature, there is strong circumstantial evidence that larval worms of the same kind may be responsible for the following reasons:

(a) the abundance in India of Musca domestica and Stomoxys calcitrans, the vectors of the parasites;
(b) the widespread distribution of the worms themselves in the stomach of equines; and
(c) the marked ‘eosinophile infiltration’ usually noticeable in bursati lesions (indicative of verminous infection).

DESCRIPTION OF THE DISEASE.

In view of the fact that the information in text books and the periodical literature is defective and incorrect in certain respects, a fresh description of the disease seems necessary.

Bursati is a chronic inflammatory disease of the skin and subcutaneous tissue of equines. The term Bursati is employed primarily for a disease of the Indian Peninsula, including Burma, but the same term is employed also in such distant countries as the United States of America and Egypt to denote what may be an identical condition. In India, the disease is of common occurrence in the northern parts but occasional cases have been met with in the Madras Presidency.
and horses are generally affected but donkeys are also susceptible. No breed of horses appears to be exempt since the thin-skinned subjects, like Arabs and Walers, are as predisposed to the condition as the coarser country-breds. It is on account of the prolonged duration of the disease, its resistance to treatment, and recurrence even after a deep excision that the condition has been likened by several workers to cheloid, epithelioma and rodent ulcer. The statement by Lingard [1905] that Bursati occurs in bovines lacks confirmation, and it may be that he obtained this idea from the American literature on the subject. It is to be noted that reports on the occurrence of Bursati in bovines in Egypt are available even in recent administration reports from that country, but in the absence of proofs the condition need not be considered as identical with the equine condition. Any causal relationship of the rainy season with the disease, as the name suggests, has not been proved, for, certain parts of India are known where the rains are excessive and prolonged and Bursati is infrequent, but Nunn [1896] has observed that the number of cases varies proportionately with the rain-fall in successive years. A curious fact about the disease is its peculiar seasonal incidence. The onset of the disease commences as early as April or May. It then continues unchecked in spite of all possible methods of treatment until the end of October or the beginning of November, when the disease tends to disappear spontaneously, or yields to the mildest treatment. But the lesions in some cases persist even longer. The rainy season has probably no more untoward effect on the course of this disease than in the case of ordinary cutaneous wounds. The description of the microscopic lesions of Bursati, illustrated by photographs, as given by Holmes [1914] is more exact than those of the earlier workers, and for any reader, who may desire to see representations of actual naked eye lesions, a reference to Plates I and II and Fig. 5 of Plate III of his Memoir is recommended. Again, P. A. Fish [1895-96] has given an excellent historical sketch of the older Bursati literature and the same may be perused with profit.

In the initial stage of the disease, an enlargement of uncertain size is noticed, followed by loss of hair. It is important to remember that several workers (Adams, Smith, Lingard and Holmes) have observed the disease to commence as a papule or an intact tumour, and the last-mentioned even states:—"I can confirm the correctness of Smith's observations that the primary lesion is a subcutaneous one and that it is only in the secondary stage that the skin becomes involved". To quote another, "the presence of kunkurs has been noticed before the formation of an ulcer". An exudative fluid is seen to accumulate in the diseased skin but this contains very little cellular material and is not circumscribed. The enlargement appears to be due to deep-seated lesions, which gradually approach towards
the surface. When the surface is shed, the sore is smooth, but as granulations start, an irregular worm-eaten picture is presented. The degenerative lesion is seen to be active at more than one focus, the healing of the first sore being followed by the appearance of fresh ones at the same site or elsewhere. It seems obvious from this that the supply of the infective agent is continuously maintained throughout the Bursati season and this has been repeatedly confirmed in microscopic sections. In an equine already affected or showing what has been called a Bursati diathesis [Collins, 1874], an accidental wound may assume the typical clinical features, but it is very doubtful if ordinary wounds on non-infected cases ever develop into the genuine sores of Bursati. Pruritus may be intense at the commencement, but if the sores are protected from flies, the affected animal does not show much evidence of discomfort, once the disease has become established.

The clinical features of Bursati sores are typical, but the most distinctive of all is the presence of what have been called kunkurs. These are yellowish bodies, globular and of the size of a millet seed or a pea, or elongated and about the size of a rice grain, found in varying numbers embedded in little pockets or fistulae in the limited patches of the diseased tissues. The kunkurs are soft and contain granular flesh coloured material when young but are hard and fibrous or even calcareous when old. Deposition of calcareous salts is generally inappreciable but occasionally it may be very pronounced. When pressed between glass slides, well-formed kunkurs behave as a hard elastic body. They have a smooth surface and can be easily shelled out on pressure, leaving pockets of variable dimensions in the mother tissue. If an incision be made into the tumafied tumour, the underlying disease process is found to extend well beyond the limits of the superficial sore and more numerous kunkurs are seen in the subcutaneous tissue. Most writers have described caseous and calcareous degeneration in kunkurs but there seems to be no resemblance of these to tuberculous changes. The commonest predilection seats of the lesions are provided by the angles of the mouth, the fetlock and pasterns, the prepuce and the face just below the eyes, but occasionally the lesions may occur in any other situation liable to persistent and mild abrasions.

The occurrence of Bursati kunkurs in internal organs such as the liver, lung, kidney, etc., has been reported by several workers [Burke, Meyrick, Steel, 1881, Lingard 1905, and others]; but it has been shown in another article by Datta [1933] that the conditions of "perihepatitis and small filarial nodules" of Lingard [1905], "calcific diathesis" of Meyrick [1878], and "Calcareous degenerations in the horse" of Smith [1885], are due to infection with Schistosoma indicum. It has to be conceded, however, that at least one internal organ—the lung—is sometimes affected with genuine lesions of Bursati. Meyrick [1878]
and Burke [1884] found Bursati horses showing lung lesions, and Hodgkins [1910] describes a case of lung affection in which the organ in stated to have been the subject of an intensive Bursati invasion, being crammed with nodules of varying sizes, and the pleura covered with whitish yellow patches beneath which were pieces of kunkur ranging in size from a hazel nut to a pin’s head. These appear to be the only recorded cases in India which may be considered as cases of pulmonary habronemiasis in addition to the following. From a severe case of Bursati, mule No. 738 of the L. A. T. T. Coy., Lahore Cantt., destroyed on humane grounds, a specimen of lung showing kunkurs, portions of skin lesions, and a sample of worms, collected from the stomach, were obtained through the kindness of Major V. C. Leckie, R.A.V.C. Histological examination of the skin and the lung revealed the presence of actual habronema larvae and the typical lesions in both the organs. This Bursati case is of considerable interest, as it is the first definite case of so-called internal Bursati and pulmonary habronemiasis to be proved in this country. It should be noted in passing that the stomach worms collected from this case were identified as Habronema muscae.

Lingard is the only worker in India, who has described calcareous nodules on the membrana nictitans, and further he has furnished a picture of the same, which bears some resemblance to the conjunctival lesions described by the Australian workers, Lewis and Seddon [1918]. When bursati lesions occur on the limbs, they present a circular contour of variable diameter with an even raised margin. The sores present irregularly elevated granulations, and on the slightest manipulation, they bleed readily. The slow inflammatory processes progress by contiguity of tissue, often receding in one place and advancing in another. There may be a thin, brownish scab formation or a blood-streaked serum may be discharged. Pus formation is seen but rarely, and that after secondary infection. When healing takes place, which process is very slow, the superficial skin becomes thickened, depigmented and shows sparse hair growth. The experience of the present writer, which he has confirmed histologically, shows that the organised or partly organised lesions persist under the apparently wholly cicatrised wound and show considerable predisposition to attacks in the following year, and this is in conformity with the experience of “Max” [1876]. Lesions in fresh and also very distant sites occur quite frequently in an affected subject. The lesions below the eye follow very closely the tract that lachrymal secretion or tears keep moist, and present an elongated, excoriated form with irregular edges and multiple surface erosions. Due to structural peculiarities of the prepuce, the lesions there show a marked tendency towards extension and calcification and the individual kunkurs are generally of a large size. Paraphymosis is often met with as a complication in stallions.
Apart from the local lesions, there is scarcely any systemic disturbance, and bursati does not impair an animal much from its serviceable qualities unless the disease has assumed a very extensive form, or has affected the penis, the seat of saddle or the angle of the mouth. The disease generally occurs in old animals but young animals are also affected. As a rule, there is only a single case in a stable, and the disease has not been observed to spread by direct or indirect contagion to other equine subjects, housed in the same or in an adjoining stable. Removal of a diseased animal, in the height of the Bursati season, to particular places, e.g., Jhansi, or to the hills, has been observed by more than one worker [Burke, 1881, Holmes, 1914] to exercise a very salutary effect on the course of the disease. It is said that cases of Bursati are not so frequent now-a-days as compared with what they were a generation ago, and this may probably be explained as being due partly to the better hygienic conditions now obtainable for our animals, and partly to a diminution of the number of horses in the country.

**Pathological Histology.**

Unlike other aspects of Bursati, its pathological histology has been dealt with by remarkably few workers. Even in Holmes’ description, superseding as it does the earlier and more imperfect attempts, a consideration of it has been dismissed in about twenty lines. Credit, however, is due to him for having described for the first time certain essential features of Bursati, e.g., the presence of a marked eosinophile infiltration and what he terms, empty spaces of varying sizes. From the foregoing remarks it is clear that a fuller and, at the same time, a more correct description of the histological features of these growths is certainly indicated at this juncture. Again, for the successful elucidation of further problems regarding the mode of infection and efficient methods of diagnosis and control, the definite and significant histological findings recorded below, should serve as helpful indications.

Materials for the present studies were provided by a valuable collection of 26 samples of preserved growths, received at this Institute from time to time since 1922 to date, from different parts of India. Of these a few were neoplastic or merely inflammatory in nature; and although some samples were in a bad state of preservation it is satisfactory to record that the same helminthie incitant was consistently demonstrated in 17 samples, obtained from such distant parts as Assam, United Provinces, Hyderabad-Deccan, the Punjab and North-West Frontier Province. Further, in these positive cases and in 5 other samples from the same collection typical microscopic lesions, which in the light of present experience may be considered as of more value in diagnosis than even the worm larva, have been persis-
tently detected (Plate XVII, fig. 1). Experience has repeatedly shown that the demonstration of the actual parasite, once the typical lesions had been seen, depended upon the examination of a sufficient number of serial sections, although the growths in a majority of cases were excised only at a late stage of the disease, when one might have expected that the most interesting stages of the disease process had passed off.

The usual picture of Bursati consists of an inflammatory granulation tissue involving the skin and the subcutaneous tissue, characterised by a profound eosinophilia and the presence of well-defined areas of degeneration. These areas represent sections of the so-called kunkurs (Plate XVII, Fig. 1), which have been known since the earliest times to form a typical clinical feature of these growths, and in the centre of these, sharply margined empty spaces are frequently seen. Since the causal parasites are very fragile and soon lose their distinguishing features, it is often difficult to detect them without great perseverance. Only odd parasites are found actually lying free in the supporting matrix of the growth. The large majority of the larvae are found enclosed in what appear to be capillary vessels, the majority of which are probably venules (Plate XIV, fig. 2 and Plate XV, fig. 2), since in the centre of the kunkurs one often observes a delicate-walled structure with a well-defined lumen and containing eosinophile cells or blood pigment. In the earliest stage of an individual lesion, the worm larvae are found invariably to form the nucleus of the commencing degenerative changes (Plate XV, fig. 1); and concentration of toxic products appears to be much higher in the immediate surroundings of the worm than at the periphery, where the degenerative process gradually subsides into the healthy tissue. Since the toxin diffuses for the same distance in all directions, the degeneration area is often seen to have a round or oval contour (Plate XVII, fig. 1). In early lesions around the parasite the cellular constituents predominate, the earliest picture being represented by an accumulation of lymphocytes in foci, supported by scanty, thin, fibrillary strands of connective tissue, disposed in a loose and circular manner. In the centre of these, a blood vessel with the enclosed larva is almost invariably to be seen as forming the nucleus for the nodule (Plate XVIII, fig. 1). In a few sections, however, the typical early lymphocytic reaction has been seen around an unoccupied capillary. In others the usual appearance of diffusive degenerative changes around a vessel, containing eosinophile leucocytes mainly, but no worm or its remnants, has been seen. The reactions are obviously due to the parasite in both these types of sections, it having been missed as a result of the sectioning knife passing at a different level. Around the individual nodular lesions an attempt at delimiting the parasitic action is manifested by the formation at most of two or three concentric rings of connective tissue, but the
process of ringed fibrosis has never been seen to have advanced to any great degree compared with what one finds in *Schistosomiasis indicum* in the horse, for instance. The connective tissue strands of old growths may be very dense and appear to be almost cartilaginous in texture.

The sequence of events in the formation of kunkurs would appear to be as follows. The earliest tissue reaction against the parasite is represented by the lymphocytic infiltration, followed by an aggregation of plasma cells, neutrophile leucocytes and eosinophiles. Plate XIV, fig. 1, shows a typical habronemic abscess in the skin, consisting practically wholly of lymphocyte cells arranged in a nodular form around the worm larvae, as one usually finds in the gastric lesions due to *H. megastoma* and no eosinophiles or fibrosis are seen at this stage. Necrosis and karyorrhexis of the nuclei of eosinophiles and other cells then takes place. While necrosis is in progress around the parasites, one finds at the periphery of the necrotic mass, young fibrous tissue cells massing together with giant cells. The supporting matrix between the kunkurs is provided by typical granulation tissue in various stages of development, generally with a dense accumulation of eosinophiles in lymph channels and proliferation of endothelial cells and fibroblasts. In a growth of very recent formation the superficial covering is composed of unaltered epithelium with extreme dilatation and engorgement of subcutaneous capillaries, which generally run perpendicular to the surface epithelium. Plate XIV, fig. 2, shows a section through the periphery of a kunkur, which presumably was formed in the course of one of these perpendicular capillaries. Exudation into the epithelial covering and consequently a hazy appearance of individual cells is later seen, leading eventually to superficial breaches or ulceration. Generally the hair follicles are unaffected in the early stage, excepting for a tendency towards a collection of the exudate in them, but sometimes a few hair follicles are seen to form sites of actual degenerative lesions as in Plate XVII, fig. 2. The surface epithelium is ulcerated more or less extensively in the active stages, and an attempt at repair by an ingrowth of the surface epithelium is seen now and again. It is a noteworthy fact that, generally in the commencing disease, a more pronounced reaction than could be expected from a surface infection, has been seen in the deeper parts of the subcutis. Contrarily, Plate XVI, fig. 1, however, is the picture of an exceptional section in which the superficial epithelium shows a degenerative involvement, but careful examination failed to reveal the presence of any parasitic incitant and the subcutis appeared to be normal except for the presence of some eosinophiles here and there. An idea of the marked degenerative power possessed by the parasite can be gathered from a study of sections, and from this, it appears improbable that penetration of the surface skin by the larva, without leaving any trace of the track
followed by it, can really take place, since a single larva in the subcutaneous situation has been seen to produce quite large areas of degenerative foci, probably due in part to the burrowing nature of the exudative fluid. In a number of these large degenerative areas, one can make out only a few interlacing strands of adult connective tissue and an occasional capillary in an otherwise homogeneously eosinophile mass. In places, organisation is seen to have taken place, either whole or in part; in others, it has scarcely started. Any parasitic debris which is not removed by phagocytosis, is soon made innocuous by the deposition of lime salts. Lesions in the prepuce show a very marked predisposition to calcification and to extension, but these features have been seen occasionally in the lesions in other situations.

The most outstanding and fundamental changes, however, concern the blood capillaries. When the degenerative changes in the endothelium of the capillary have not gone very far, a thin layer of a delicate endothelium can be recognised without much difficulty (Plate XVIII, fig. 2). Certain vessels show varying degrees of endarteritis with partial or total obliteration of the lumen by the replacing connective tissue and also an increase in the amount of elastic tissue at places. The commencing inflammatory changes in the vessel wall are represented by a tendency to diffuse staining (Plate XV, fig. 2). More severe inflammations are manifested by a marked dilatation of the wall due to infiltration with exudative cells. The degree of exudation apparently determines whether only the endothelium is to be proliferated or shed, or the vessel wall is to undergo necrosis, calcification or total disruption. The desquammated endothelial cells can be seen lying scattered in the periphery of the thrombus mass formed in an affected vessel, or where the wall has given way, the endothelial cells may be wandering free in the matrix of the growth.

It is important to note here that apparently healthy worms in cross- or longitudinal sections have been seen lying quite free in the more or less dilated lumen of capillary vessels (Plate XIX, fig. 1) with varying degrees of degenerative change in the vessel wall (Plate XV, fig. 2). On measurement, larvæ are seen to vary in size within certain limits and from this, one presumes them to be in slightly different stages of development. Usually the larvæ are seen lying straight and singly but occasionally a single worm in a coiled form (Plate XV, fig. 1) or several worms may be encountered in a larger sized capillary. A central intestinal canal and a number of cuticular ridges at regular intervals can be seen in cross-sections, (Plate XV, fig. 2) or if the parasite has been cut longitudinally, the characteristic striations, both longitudinal and transverse (Plate XVIII, fig. 1), can be made out in the worm structure inside the vessel. Expert helminthological opinion was
obtained from Mr. G. D. Bhalerao, M.Sc., who identified the larvae, as those of Habronema spp. In certain sections, little, if any, degenerative changes in the parasite can be discerned; in other sections an empty, ovoid or spiral space, which was also seen by Holmes [1914], containing a granular debris, apparently an impress of an escaping or a degenerated parasite fallen out in sectioning, is to be noticed. The anterior and posterior ends of the causative larva have been seen in more than one section (Plate XIX, figs. 2 and 3). As the anterior ends, which were seen in two cases, appeared to be more typical than the posterior, they were submitted to a careful examination. The vestibule was found to be cylindrical in both the cases and to measure 36.5μ and 39.0μ in length, the measurements corresponding very closely to those of H. muscae; and from this, it would appear that the larvae, in all probability belong to this species. As a suggestion has been made by a worker that different species of Habronema may be responsible for growths in different parts of the horses' body, it may be noted that one of the above two specimens was obtained from the prepuce of a horse and the other from the neck of another subject.

It is interesting to add here that the histological features of Bursati have been studied, side by side, with those of a definite case of cutaneous habronemiasis of the horse, as it occurs in Greece. The tissues from the latter were obtained through the courtesy of Messrs. Pheloukis and Knithakis of the Veterinary Research Laboratory of the Ministry of War at Athens. As a result of these studies, the Grecian condition was found to be identical in histological features and in the characters of the parasitic larvae with Bursati, as it occurs in India.

**Discussion.**

The statement of such a distinguished man as Railliet (1915) that "In India one still confuses, under the name of Bursati, cutaneous lesions caused by moulds and by nematodes" appears to be partly responsible for having kept alive the general impression in India, Egypt and elsewhere that at least two distinct disease entities, mycotic and helminthic, are included under the term in this country. Apart from this confusion, doubts, regarding the probability of a habronemic origin of this condition, have been expressed in rather emphatic terms by Freeborn, Hart and Howell [1927], who hold that the infestation of Bursati by Habronema is accidental and does not constitute the etiological factor. Among others disinclined to accept the habronemic origin, is Baylis [1923], who states: "It seems conceivable however that the sores may be produced first by some other agency and the worms be deposited in them by flies."
Although the invariable association of Habronema larvae with Bursati lesions has been shown in the present histological studies, it is to be admitted that the mere presence of worms is by itself not enough for the purpose of casually connecting them with the growth until the disease has actually been reproduced under artificial conditions. Again, the failure on the part of several workers [Van Saeeghem, 1919, and Hill, 1920] to induce the Habronema worm to penetrate the intact skin would seem to provide enough room for the presumptive view, advanced by Roubaud and Descazeau [1921], that the presence of pre-existing abrasions or broken cutis is essential for the production of so-called Summer Sores, and the obvious method of proving this would be to create the disease experimentally by introducing into wounds Habronema larvae actually derived from the proboscis of flies. If a more reasonable explanation regarding the production of natural cases of the disease were not forthcoming, parenthetically it may be said that one such is available and is given below, the invading Habronema larvae would be reduced to the status of mere secondary organisms; and the condition, etiologically, would present an analogy to myiasis, which in a great majority of cases is caused by fly larvae deposited in previously existing wounds. On the other hand, it is noteworthy that while in myiasis the invading larvae duly grow to maturity, Habronema larvae encountered in Summer Sores and Bursati represent only erratic forms, which, after wriggling about for a while in the resulting inflammatory granuloma, die and lie embedded in degenerated areas, the kunkurs, as observed upon histological examination of diseased tissues. The question therefore arises if cutaneous habronemiasis represents an accidental secondary condition, or whether it can be brought into the category of a normal disease entity, such as one finds in gastric habronemiasis, where the stomach of the equine represents the normal habitat for the developing worms. The death and degeneration of the worm in the kunkurs cannot be a feature necessary for the fulfilment of its life-history, and it seems certain that their death is due to some accident or misfit in the host-parasite relationship, which they cannot help. In describing certain noteworthy histological findings, it has been stated above that the most outstanding and fundamental changes in Bursati concern the blood capillaries, and that in the centre of early kunkurs, a blood vessel with the enclosed larva is almost invariably to be seen as forming the nucleus for the individual nodule-formation. Again healthy worms have also been seen inside apparently healthy vessels (Plate XVIII, fig. 2). These findings bring out significantly the close analogy of Bursati to human filariosis, due to Filaria bancrofti, where the worms accidentally get held up in the capillaries producing the characteristic embolism, while the great majority of the worms float about in the circulating blood. This
analogy should be kept in mind, since certain well-known but hitherto unaccountable clinical facts of Bursati can now be explained on this view.

In the light of the present histological findings, Bursati, at any rate as it occurs in India, appears to be a single disease entity, the presence of moulds in open sores having no significance; for, it has been shown above that the diagnostic lesions of Bursati are represented by the kunkurs and that they are, histologically speaking, typical helminthic nodules, the same nematode larvae of Habronema spp. having been demonstrated consistently as forming the inciting nucleus for the production of the nodular lesions. There need now be no doubt, therefore, regarding the helminthic as also the habroemic origin of Bursati.

With regard to the mode of infection, the position is more unsatisfactory. A perusal of the Bursati literature shows that the fly transmission theory has been held by the natives of India from as early a date as 1820, and that among the other old theories, it was believed to be a blood disease [Ubique, 1877 and Ferris, 1881]. In the case of cutaneous habronemiasis, as known in different parts of the world, the hypothesis current at present is that these helminthic granulcma are caused by the deposition of the habronema larvae in wounds by flies. Although the current view is undoubtedly an improvement on the old Indian belief, it will be seen from what is stated below that it is still unacceptable, since it fails to provide a satisfactory explanation for the known facts regarding Bursati. Contrarily, however, it will be realised that from a consideration of certain clinical peculiarities of the disease, which have been emphasised by most observers, one cannot resist the belief that the disease is more than a mere local infection, although there are little or no constitutional symptoms. Notable among the protagonists of what may, for brevity, be called the fly theory, are Railliet and Henry [1915], who suggested that habronema larvae penetrate the skin in the same manner as the larvae of ankylostomus, but from the experimental work of several other workers, it appears that these larvae are not able to penetrate the equine skin even when the epidermis alone is damaged. Margarinos Torres [1924], however, claims to have proved that the larvae of Habronema muscae, if deposited in wounds of a guinea-pig, are able to reach the lungs, it being an organ for which they appear to have a marked preference. The failure on the part of the present writer [1931] to transmit the disease through the agency of flies receives support from Holmes [1914], who states “I am unaware of recorded cases of transmission of the sore from horse to horse by means of outside agencies”, and also in the statement of Spooner Hart [1873]: “Practice and time have afforded ample proof of the non-contagiousness of Bursati, for my bursatied subjects stand among other animals and I have never seen any ill effects ensue”. The statements quoted here and the
general experience that in the following year or years “animals once affected with the disease are almost certain to have it recur” (Kerr, 1829) at the same seat or in sites far away, where there have been no wounds, to the exclusion of other animals, indicate that the mechanism underlying the production of natural cases of the disease is not so simple as the current fly-transmission theory would imply. The exclusion of other animals is remarkable, since infected flies undoubtedly have access to all animals housed in a particular stable, where there had been a case or cases previously. Further, it is known that the prevention of contamination of wounds from infected flies and the local application of numerous otherwise very efficient drugs, have failed in the hands of many workers to prevent the recrudescence of the sores in certain parts several years in successions, or of fresh sores in other parts of an affected subject; but the available information shows that the larvae in the skin lesions soon die out, i.e., within a month of implication. Again, that bursati sores persist during the whole of the special season, which is several times longer than one month, is known. The control and cure of Bursati should have been achieved by now by the prevention of reinfection by bandaging or application of fly-repellents, like Van Saeghem’s [1918] Plaster of Paris and Naphthalene preparation, if the fly theory represented a true picture of the essential pathogenic process; but the unfortunate fact that Bursati is still incurable, recurrence is common and destruction of affected cases is still practised, shows that fly transmission is not the usual method. The work of Margarinos Torres [1924] however proves that occasionally the migration of larva, deposited in a superficial wound, may take place.

The possibility of infection taking place through the general circulation, which has already been suggested by Laulanie [1884] in the case of Summer Sores, offers an interesting alternative, following naturally as a corollary to the significant histopathological findings, referred to above. That the clinical peculiarities of Bursati are such that one cannot resist the belief that the disease is more than a mere local infection has already been stated and a consideration of expressions, such as, a “bursati diathesis” [Collins, 1874], “peculiar conditions of the patient’s system” [Smith, 1879], “constitutional predisposition of the disease” [“Max”, 1876] used by several workers, makes it obvious that a certain subjective factor is indispensable to the production of the sores. The subjective factor or “bursati diathesis” would seem to resolve itself into the infestation of the equine stomach through the intermediary agency of flies and the consequent floating of the migratory worm larvae in the general circulation. Some of the earliest observations, such as “the poison appears to be carried to other parts of the body” [Meyrick, 1878] would point to the intervention of the general circulation, and the same is again supported by the com-
mencement of the growths as an intact tumour, or the lesions being first observable in the subcutis with extension outwards later, a feature which has been affirmed by several keen observers.

The first definite case of so-called internal bursati involving the lung, in association with the skin lesions and the presence of Habronema in the stomach has been described earlier in this article. A correct decision upon whether there really exists any relationship between habronemiasis involving different functional systems, such as cutaneous, pulmonary or gastric, in the same animal is important, since an affirmative verdict would add considerable support to the new theory elaborated herein, viz., an internal origin of Bursati. It would appear to be the opinion of Roubaud and Descazeaux [1921] that each is a separate entity, being produced by infected flies, alighting on abrasions, or nostrils or on the lips, as the case may be. Further they believe that ingestion of parasitized flies also may cause the gastric affection. But the above-mentioned case of internal bursati is comparable to the experience of several other workers. For instance, Drouin [1902] observed the simultaneous involvement of the skin and the lung in Summer Sores. In Belpel's [1925] experience in Italy, skin lesions were associated with the presence of the worm in the stomach, and finally Dieulouard [1926] working in Brazil, found that of 20 horses, showing pulmonary habronemiasis, 19 showed evidence of having had Summer Sores at some time during their life, and that the lung lesions occurred only in animals showing evidence of skin involvement. The migratory propensities of Habronema larvae have been demonstrated by Margarinos Torres [1924], and the association of various forms of habronemiasis in the same animal as observed by several workers, has been remarked upon.

It seems that the presence of the migratory worm larvae in the general circulation cannot by itself produce the disease. There still remains the need of a further factor, injury or contusion, in order that thrombus formation may take place in the capillaries, enabling the arrest of the worm larvae in embolic masses. Bursati growths would therefore appear to be formed only in seats, liable to much risk of successive contusion and injuries, and a breach of the surface may also serve the same purpose, the actual lesions of Bursati being due to an effort of the host tissues to get rid of the embolic masses. Even a deep excision of the growths by the surgical method and a rigid insistence upon the retention of a bandage has failed in the hands of many workers, and the reason is not far to seek. The sectioning of blood vessels in the surgical operation leads to clot-formation and consequently bursati sores. In short, the writer feels justified in advancing the view, that in a majority of cases the habronema larvae are transported through the
general circulation to certain parts of the body, where they are arrested owing to
previous thrombus formation, and not deposited on existing wounds by the fly.

Turning now to the specific identity of the *Habronema* responsible for Bursati in
India, *H. muscae* appears to be the most probable. It may be noted that in France
and Australia, *H. megastoma* is incriminated as the causal agent of Summer Sores
and Swamp Cancer, and that in Brazil, Margarinos Torres [1923] considers "*H.
muscae* is the most important and perhaps the only cause of the disease ", and Van
Saceghem [1917] believes the same to be true in Zambi in the Congo. That the
measurements and character of the anterior end of the larvae, as seen in sections of
the skin and again in sections of the prepuce lesion conforms to those of *H. muscae*
has been mentioned earlier in this article. The occurrence of the same species of
the worm to the exclusion of other species in the *st. mach* of a case, which showed
both pulmonary and cutaneous habronemiasis, appears to be more than a mere
coincidence. The experience in India would therefore support that of the Brazilian
worker and of Van Saceghem [1917].

In regard to the probable intermediaries, certain native flies, six muscid and
one sarcophagid of Queensland [Johnston, 1920], Drosophilid flies in Ceylon
[Crawford, 1926], have been incriminated in addition to the usual *Musca domestica*
and *Stomaxys calcitrans* and recently Patton [1932] has stated: "*Musca crassiro-
stris* is a probable transmitter of *Habronema* in India, for I have found these nem-
atoles developing in it." In addition to being related to "special districts" and
"special seasons", Bursati has been called a disease of "special circumstances"
by Burke [1881], and though the precise factors are as yet imperfectly understood,
the occurrence of particular species of *Habronema* and of the appropriate flies in
the same locality now seem indispensable to the production of Bursati. The fact
that the sores, after having manifested considerable resistance to treatment, tend
to disappear spontaneously at the approach of the winter indicates that the supply
of the larvae is no longer continued. That a close relationship must exist between
the cycle of evolution of the *bursati* *Habronema* and the life-history of its inter-
mediate host is obvious from the work of Ransom [1918, Hill, 1920] and others;
and until the evolutionary stages of the worm in relation to the fly in question have
been worked out, month by month, the factors responsible for the *bursati*, season
and districts, will not be understood. A scheme of evolution has been given by
Roubaud and Descazeaux [1921], but as far as the writer is aware, this has still
to be confirmed. As a result of histological studies upon sections of *H. megostoma*
tumours, from about 20 cases, collected at different times during a number of
years, the author could obtain no proof for the view originally advanced by Des-
cazeaux that the female parent probably brings forth young but once during a period of 12 months.

From what has been said above, it will be realised that the effective control and cure of Bursati must depend upon the eradication of the Habronema worms from both the definitive and intermediate hosts, disposal of equine faeces and intravenous administration of drugs like Tartar Emetic and Novarsenobenzol.

**Conclusions.**

The present investigation reveals the nature of Bursati known in India since 1829. It is a habronemic granuloma of equines, most probably due to *H. muscae* Carter, involving the skin and internal organs such as the lung. The disease is more than a mere local infection, the causative larvae exhibiting considerable migratory powers. In the majority of cases, skin lesions would appear to be produced through the general circulation, wound infection playing an insignificant role.

**Acknowledgments.**

The thanks of the author are due to Mr. G. D. Bhalerao, M.Sc., Helminthologist at this Institute, for identifying the helminth larvae, discovered in the course of this investigation, and to Mr. J. Sunder Rao, Artist, for the execution of the illustrations.

**References.**


——— (1892). Tropical Diseases of the horse and ox, Jubbulpore, Preface VI, 70.


Rivolta, (1888). Cited by Holmes (1914).


Also *J. Comp. Path. Therap.*, 1891, 4, 125-131.


**DESCRIPTION OF MICROPHOTOGRAPHS.**

*Plate XIV*, fig. 1.—Low power photograph of a section of bursati growth, removed from the angle of the mouth, showing a typical habronemic abscess in the early lymphocytic stage. In the central degenerated area, both longitudinal and cross-sections of the larvae can be seen. The histological picture resembles very closely that of early gastric abscesses, due to *H. megastoma*.

Fig. 2.—Low power photograph illustrating the essential lesion of Bursati, characterised by a cross-section of the parasite, enclosed in a blood vessel, around which a lymphocytic infiltration can be seen arranged in the form of a nodule. This is the earliest stage in the formation of the kunkur.

*Plate XV*, fig. 1.—Section of a bursati growth, removed from the limb, showing a coiled larva in a dilated vessel. Note the sharp outline of the lumen and that the concentration of the toxic effect on the vessel wall diminishes from the centre outwards.

Fig. 2.—Same as Pl. XIV, fig. 2 but under a higher magnification. The worm larva is seen here at its primary seat of operation, the lumen of a capillary vessel.

*Plate XVI*, fig. 1.—Low power photograph of an exceptional section of a growth. Superficial cutis shows degenerative changes, with a slight involvement of two epithelial papillae in the centre of the field. Sub-epithelial layer is practically unaffected, excepting for slight eosinophile infiltration.

Fig. 2.—Low power photograph of a bursati section. An elongated kunkur, which is more extensive in the depth of the growth than towards the surface, is seen here, having been formed along the course of one of the dilated perpendicular vessels. Note the partial loss of staining affinities of the major portion of the field, compared to the darker and healthier portion on the left.

*Plate XVII*, fig. 1.—Low power photograph. On the left, four kunkurs are to be seen. In the case of the lowest kunkur, the centrally situated blood vessel can be seen, as also the diffusion of the toxin for the same distance in all directions, giving a more or less rounded appearance to the kunkur.

Fig. 2.—Low power photograph, showing two kunkurs in a bursati section. The left one is practically free to be evacuated from the fistula in the mother tissue. The right one has been formed in a hair follicle, the hair root having already been destroyed.
Plate XVIII, fig. 1.—The essential structure of an elongated kunkur, partially detached from the mother tissue is to be seen. Eosinophile leucocytes are the most predominant cells in this growth. Longitudinal section of the worm larva, with its transverse striation can be seen occupying the blood vessel, which forms the nucleus for the production of this kunkur.

Fig. 2.—Section of a bursati growth, showing a healthy worm in a healthy vessel. Only the perivascular lymphocytic reaction is in evidence here. This stage is followed successively by the stages represented by Pl. XIX, fig. 1 and Pl. XVIII, fig. 1.

Plate XIX, fig. 1.—Longitudinal section of a larva lying free in a blood vessel. Diffusive action of the toxin can be judged by the homogeneous appearance of the border around the vessel.

Fig. 2.—Photograph of a Habronema larva in a bursati section. The posterior end of the worm is directed to the right, the anus being easily visible.

Fig. 3.—Photograph of the anterior end of a Habronema larva, obtained from a bursati section. The cylindrical vestibule is clearly seen.
PLATE XV.

Fig. 1.  $\times$ 140.

Fig. 2.  $\times$ 554.
EFFECTS OF STERILITY AND IMPOTENCE ON BREEDING OPERATIONS IN INDIA AND METHODS FOR THEIR DIAGNOSIS AND CONTROL

BY
Capt. S. C. A. DATTA, B.Sc., M.R.C.V.S.,
Veterinary Research Officer in-charge of Pathology (Temporary),
Imperial Institute of Veterinary Research, Muktesar.

Reprint from
THE INDIAN VETERINARY JOURNAL
EFFECTS OF STERILITY AND IMPOTENCE ON BREEDING OPERATIONS IN INDIA AND METHODS FOR THEIR DIAGNOSIS AND CONTROL.*

BY

CAPT. S. C. A. DATTA, B. SC., M.R.C.V.S.,
Veterinary Research Officer, in-charge of Pathology (Temporary), Imperial Institute of Veterinary Research, Muktesar.

It is well-known that Dairy Farmers in India suffer considerable economic loss from pathological and functional disorders of the reproductive organs of their breeding stock. To start with, cattle in India are often slow in coming into oestrus and the intervals between successive gestations are relatively long. There is a little doubt that a certain amount of sterility has always existed among the native breeds of cattle, but their relatively low economic value has prevented this problem from being recognised in its proper perspective. Information is available that a cessation of reproduction or reduced fertility has been the cause of much anxiety in several dairy establishments in recent years but it must be confessed that much scientific attention has not been devoted to it in the past. It seems that losses from sterility or reduced fertility will become increasingly heavy in this country unless proper arrangements are now made for an intensive investigation into the incidence and nature of the reproductive diseases of our animals. Intensive methods of dairying are now being adopted with the result that the animals are housed under conditions simulating those of Europe and America, are stall-fed with concentrates with the object of high milk yields and abnormal prolongation of the lactation period, and many of these animals receive insufficient exercise. The evils of intensive dairying cannot be avoided and when the normal physiological processes of the dairy cow are interfered with, it is to be expected that the problem of sterility will become very serious. In South Africa, a very intensive investigation into the pathology of sterility in the dairy cow, with particular reference to the histological lesions, symptoms, cause and treatment, has been recently completed by Quinlan (1929) and it is to be hoped that this excellent object lesson will be followed in this country before long.

* Read before the Meeting of the Animal Husbandry Wing of the Board of Agriculture, held at New Delhi, February, 1933.
Exact statistics regarding the incidence of sterility amongst breeding stock in India are not available and an estimate of the exact monetary effects, which all kinds of irregularities of reproduction produce year after year, is difficult to arrive at, but the losses that a dairy farmer may sustain, through the disorganisation of his arrangements for producing economically a uniform quantity of milk throughout the year, must obviously be very heavy.

A certain amount of work has been done at Muktesar upon abortion in its relation to sterility and this subject is being treated in a separate note. [Vide. Haddow, J. R. (1933), Agriculture and Livestock in India, Vol. III, pp. 325-329]. During the last two years or so several enquiries for scientific advice upon sterility in both sexes has been received at Muktesar and work has been initiated in the treatment of bulls showing a disinclination to serve and heifers failing to come into season. This is a very hopeful sign and should augur well for the future, and it may be noted that the recent developments in endocrinology have considerably broadened the scope of therapeutic measures for this condition and have rendered further researches on sterility pregnant with great possibilities.

If a reference is made to the literature, it will be seen that the majority of the workers in this line have occupied themselves almost exclusively with aspects of sterility in the female animal, including early diagnosis of pregnancy, and facts about the role played by the male are, therefore, less familiar. Unless the optimum conditions of fecundation are fulfilled, it is obvious that sterility will result and the optimum condition is that a normal spermatozoon should meet a normal ovum in a normal way. Since any deviation from the optimum condition leads to either temporary or permanent impairment of the reproductive health and fertility, the responsibility of both parents must be equally important. The breeding record of the sire and dam affords much assistance in determining the cause of sterility but a careful clinical or laboratory examination is indispensable. Since the ultimate value of any stock must depend upon its breeding performance, an accurate record of the various events in the sexual life of each animal is of great value.

Sterility in the female may be due to anatomical deformities of any constituent part of the generative system to pathological lesions like tumours, cystic ovaries, and retained corpus luteum, but inflammatory conditions whether due to systemic or local causes, to specific or non-specific factors, produce in all probability the largest number of sterility cases. In addition to the specific infections, like dourine, granular vaginitis or balanitis, bovine and equine abortion, venereal granuloma of dogs, which unfortunately all exist in this country,
several other organisms have been recovered from the swabs or washings from the genitalia of sterility cases, in particular streptococci. As Williams (1923) has pointed out: “The failure of 60% of the copulations to produce living young is referable to prenatal death resulting from infections. If the infection kills or mortally injures the spermatozoa in the seminal tubules or elsewhere in the male genitalia, if the infection is added to the semen when ejaculated or later kills the spermatozoa or fertilised ovum, or if the infection exists in the ovary, oviduct or uterus in the female the principle remains the same. It is death from genital infection.”

Environment, diet, endocrine secretions, heredity, and excessive sexual use, all play a role singly or in a combined manner, in producing sterility. Predisposition to sterility is a feature in certain families and according to Crew (1924), grades of fertility are definite racial and breed characters and are transmitted in inheritance. Environment is a minor factor, associated with deficiencies in the soil and fodder, and seasonal and climatic changes present certain stimuli to sexual instinct. It is stated that advancing the covering season in England for getting foals earlier in the year has resulted in less fertility in stallions and that difficulties are experienced by most English breeders in getting mares in foals until grass is available. Since defective nutrition is commonly met with in India, it may be of interest to know that mineral deficiencies of calcium and phosphorus have been known to be connected with sterility, low fertility, and retention of placenta. Absence of vitamin B and E produce a degeneration of the germ cells and the seminal epithelium. Evidence is available from a herd in another country, in which cattle were frequently sterile, oestrum was exhibited but conception did not take place, to show that a regular ration of bone-meal produced a marvellous result, in that, all bred to time. So-called Waihi disease, which is characterised by the absence of oestrum, has been proved to be a deficiency disease, being now amenable to bone-meal feeding. In Mason’s (1925) experience an increase in breeding capacity was observed by turning out stalled cattle on to green grass. As a result of experimental work in Germany and elsewhere, it is known that sperm production in stallions is greatly influenced by the feed and that by the addition of certain supplements to the basal ration, the sperm production could be enhanced many times the normal count per ejaculation. In Australia, potassium iodide is added in small quantities regularly to the ration, since deficiency of iodine has been proved to act detrimentally on sexual life and fertility. Wheat germ oil has proved of considerable value in female animals in certain countries. Although sterile animals may be apparently normal in condition, it will be seen from the above
remarks that a careful examination of the fodder of sterile animals is warranted.

Again, seminal pathology plays an important part in producing sterility. The spermatogenetic function of the testes is very sensitive to outside influences. The influence of the frequency of coitus and sexual stimulation on sperm production has received considerable attention in Germany. Studies on the quantity and quality of spermatozoa produced by stallions in varying intervals during successive services indicate that a rest period of 48 hours is necessary for the normal sperm production and that any greater interval does not result in the increase of viable sperms. Thanks to the work of Williams and Savage, methods for the collection, and preparation of seminal fluid for diagnostic examination are now available and with experience and skill it is possible to detect changes in the spermatozoa, referable to disease of the genitalia, long before a clinical examination would reveal pathological changes. Hydrogen-ion concentration of semen is another important factor, the average for normal horses being pH 7.3 and the corresponding average for sterile horses being 7.58. The local or general cause of sterility having been determined, treatment is prescribed accordingly. Massaging the ovaries and expression of the retained corpus luteum can be carried through the rectum and vagina. Of the endocrine products now in use, the principal sources are the anterior lobe of the pituitary, and the urine of pregnant women. Since Zondec and Aschheim demonstrated the stimulating effects upon the generative system of the sexually immature female mouse produced by the use of the urine of pregnant cows, the work has been extended by Crew (1931) to include other animals of economic importance, but in recent work at Muktesar the urine of mares has proved disappointing. Baker (1931) has found that Brucine hydrochloride, which is half as poisonous as strychnine, has the same stimulating effect on sperms and has expressed the hope that this discovery may find practical application in medicine and agriculture wherever sterility is due to inactivity of sperms. Implantation of gonads into various domesticated animals of both sexes has been carried out by various workers and it is claimed that recrudescence of fertility, sexual potency and an enhancement of bodily functions generally takes place. Frei (1930) recommends the organotherapeutic preparation “Ovotestis” in female cases of sterility and nymphomania, while the sex glands of the male are said to be stimulated by such preparations as “Viriligen,” “Semidrol,” and “Prosek.”
THE POSITION OF TUBERCULOSIS AND JOHNES DISEASE IN INDIA WITH SUGGESTIONS FOR THE BEST METHODS TO ADOPT FOR THEIR DIAGNOSIS AND CONTROL

BY

Capt. S. C. A. DATTA, B. Sc., M.R.C.V.S.,
Veterinary Research Officer in Charge of Pathology (Temporary)
Imperial Institute of Veterinary Research, Muktesar.

Reprint from
THE INDIAN VETERINARY JOURNAL.
Vol. XI, No. 2, October 1934.
THE POSITION OF TUBERCULOSIS AND JOHNE'S DISEASE IN INDIA WITH SUGGESTIONS FOR THE BEST METHODS TO ADOPT FOR THEIR DIAGNOSIS AND CONTROL.*

BY

CAPT. S. C. A. DATTA, B. Sc., M.R.C.V.S.,
Veterinary Research Officer in Charge of Pathology (Temporary)
Imperial Institute of Veterinary Research, Muktesar.

It is a truism to say that Tuberculosis and Johne's disease have been the nightmare of Dairy Farmers all over the world and have engaged no little attention from scientists in most countries for decades. The position in India, on the other hand, has been most unsatisfactory, for until quite recently workers here were so obsessed with the ravages of diseases like rinderpest and haemorrhagic septicaemia that no serious attention has been paid to the diseases of cattle of a chronic and insidious nature. Consequently, the literature comprises only a few stray case reports and some slaughter house statistics of these conditions.

TUBERCULOSIS.

Our conception of the various aspects of the problem of Tuberculosis in this country has undergone considerable modification in recent times, due in particular to the Bovine Tuberculosis Enquiry (1923—1928) carried out at Muktesar under a grant from the Indian Research Fund Association. In an attempt to discover why bovine tuberculosis was so rare in this country, as was then the general belief, Liston and Soparkar (1917) raised certain important questions for investigation, which were followed by Sheather (1920), leading finally to the comprehensive enquiry under Soparkar at Muktesar, the terms of which were:

(a) Susceptibility of bovine animals in India to tuberculosis.
(b) The virulence of strains of tubercle bacilli isolated from natural bovine lesions in India.
(c) Diagnosis of latent tuberculosis by the application of the tuberculin test.
(d) Examination of the types of tubercle bacilli responsible for tuberculosis in man and animals in India.

* Read before the Meeting of the Animal Husbandry Wing of the Board of Agriculture, held at Delhi, February 1933.
Although the enquiry covered a lot of ground, and added a wealth of valuable information, it must be realised that very important questions still remain to be solved. It appears from authentic figures of the incidence of tuberculous lesions in cattle slaughtered at several Municipal Slaughter Houses that an appallingly rapid spread of the disease has taken place during an interval of about 15 years only. For instance, the percentage incidence estimated by Taylor and Oliver in 1917, was about 3% only, the incidence in 1927 estimated by Soparkar was 16·28%, but the latest figures, worked out by Soparkar and Dhilon (1931) show that out of 1,116 animals examined at Lahore, 21·3% cows, 23·6% buffaloes and 31·6% bullocks showed tuberculous lesions. Added to this, is the disquieting information that the disease is no longer restricted to North India and it seems to be spreading amongst all species of domestic animals. Evidence has been collected at this Institute from a certain Military Dairy Farm and two Remount Depots to show that after introduction of infection, a high incidence of clinical tuberculosis may develop in a herd, maintained under conditions of domestication simulating those of the west. With regard to the susceptibility of buffaloes, Soparkar’s earlier (1924) experiments indicated “the buffalo to be much more resistant than the calves” but his later experiments (1926) point to the buffalo being more susceptible to tuberculous infection than the cow. There is evidence of the occurrence of tuberculosis in 4% pigs slaughtered at Bandra in Bombay and it is known that in 1924, a small but severe outbreak of the bovine type of tuberculosis occurred in the Victoria Gardens, Bombay, amongst llamas, obtained from Germany, and that the disease spread to several spotted deer and a blue bull. Of the other species of animals, definite evidence about the occurrence of natural cases of tuberculosis amongst goats, horses, elephants, fowls, dogs and camels is available at Muktesar.

The available results of bacteriological work carried out upon lesions of tuberculosis in animals in India show that the bovine type of organism is commonly associated with bovine lesions and the human type with the disease in pigs. The avian type of organism has also been recovered from a bullock and some pigs, and the bovine type from a pig, a goat, several llamas and buffaloes. The human type of tubercle bacilli is believed to be usually responsible for the disease in man in this country, but the current view amongst medical men that the surgical forms of human tuberculosis in India are entirely independent of infection from the bovine species would seem to require some modification, since Soparkar (1929) has succeeded in proving the disease in a girl in Bombay to be definitely due to the bovine type of the bacilli, and because the occurrence of two severe
cases of tuberculous mastitis of the cow in Bombay and the Punjab are on record.

The pathology of tuberculosis amongst animals in India is little known. The exact mode of infection, the comparative incidence of paranchymatous or lymphatic, of intestinal or respiratory lesions and how the receptivity of cattle varies at different periods of life require to be studied. With the exception of a few cases of generalised lesions, it has been observed by more than one worker that the bovine lesions in this country are usually minute in extent, localised in the tissues in which they are commonly found. A question of some interest has been raised by Lankester (1923) who remarks: "I am not aware of any authoritative investigation of having been made which can be said to definitely negative the suspicion that some at least of the cases of tuberculosis in bovines in India may in reality be the result of direct infection from man."

Diagnosis may be arrived at during the life of an animal from clinical signs of the disease or by the tuberculin test or from both. The tuberculin test is intended to pick out animals before they have a chance of becoming dangerous to the herd and in advanced cases no reaction may be obtained. Since there are considerable diurnal fluctuations of temperature in India, the Double Intradermal Test, as recommended by the Medical Research Council, has been adopted in preference to the subcutaneous test and it is usually recommended that the Double Intradermal Test be carried out with both ordinary and avian tuberculin since, with so little extra trouble valuable information in regard to the elimination of Johnes's disease may be obtained.

It is important to recognise the possibility of atypical forms or "no-lesion" tuberculosis, in which a thorough post mortem examination fails to reveal microscopic lesions. A finding of considerable significance, therefore, has been recorded by Soparkar (1927), in which, he has been able to isolate fully virulent tubercle bacilli from material of an apparent reactor to the subcutaneous tuberculin test, though no naked eye lesions were detected on slaughter, and similar cases have been encountered recently at this Institute.

The control of tuberculosis is a thorny question and the policy to be adopted towards reactors to the tuberculin test bristles with difficulty in India. Experience has shown that it is not a problem that can be left to the individual cattle owner; it is a problem involving much larger interests. A general tuberculin testing of all cattle in selected areas and farms at regular intervals, followed by elimination of all reactors, and destruction of every animal showing signs of clinical tuberculosis is the procedure in England. A reactor
to the tuberculin test is not necessarily condemned to death there, for the animal may not be infected with 'open' tuberculosis, but it precludes an owner from holding a "certified" or Grade A (T. T.) milk license. Bang's method of isolating reactors and building up and maintaining herds free from tuberculosis has been tried with great success in certain countries, notably in Denmark. In certain cattle establishments in India the English procedure has already been adopted, but it would appear that Bang's method with certain modifications, would be more suitable for this country, in which there exist a conservative feeling against the slaughter of cattle.

In view of the alleged rarity of bovine tuberculosis and its surgical manifestations in man in this country, it has been contended in the past that measures for the improvement of the milk supply need only be directed towards the prevention of contamination after milking, rather than against the elimination of any specific organisms by the cow. It must be remembered in this connection that actual disease of the udder is not necessary for the elimination of the organisms in the milk, since Stanley Griffith (1927) has proved that the normal udder of lactating cows and goats will permit the passage of tubercle bacilli, whether of the bovine or human strain.

With regard to preventive vaccination, experiments have been carried out for 50 years or more, in the hope of finding a satisfactory method of immunising against tuberculosis by the use of either living virulent organisms, dead organisms, or living avirulent strains. The second has never been successful; in the first the potential danger is too great, but the third is still raising great hopes. Even admitting that calves vaccinated with B. C. G. are more resistant than non-vaccinated, as Buxton and his co-workers (1932) have shown, one must go very cautiously since information regarding the fate of the injected bacilli and regarding the possibility of the original virulence being regained in the same or another animal is still lacking. Spahlihger's vaccine has given encouraging results in the first trial carried out with it on animals, but little is known about it yet.

**JOHNE'S DISEASE.**

Johne's disease was first diagnosed in this country as late as 1917, but, from a remarkably large number of records now available, it is apparent that this disease is very widespread in cattle throughout India. Compared with cases in Europe a larger proportion of cases in this country show a complete absence of clinical symptoms, but the examination of bowel washings, rectal smears or biological tests betray the actual state of affairs. This very insidious onset makes the presence of Johne's disease in a herd a most dangerous one, particularly in farms which are heavily stocked.
Fairly satisfactory methods for the diagnosis of Johne’s disease are available in the shape of certain biological reagents and in the bowel washing method, as employed by Prof. Krishnamurti, and the rectal smear method. Brews of Vallee and Rinjard’s paratuberculin, Dunkin’s Johnin, as supplied by the respective workers, and also a brew of Johnin, prepared from Indian strains of Johne’s bacilli have all been put to comparative tests at this Institute and the indications at present are that none of the other agents is equal in value to the avian tuberculin at present issued from Muktesar. As a result of systematic tests carried out all over India with the last mentioned preparation, accurate information on the incidence of Johne’s disease is now available, but there is still considerable room for improvement in this matter of a diagnosis agent for Johne’s disease. Certain anomalies are from time to time encountered, in particular, the failure to evoke a reaction in certain stages of the disease. As already suggested, a double test, employing both avian and human tuberculin, is advisable, since the possibility of double infections of Johne’s disease and tuberculosis in the same animal has to be eliminated.

Both cattle and buffaloes in India are known to be affected with Johne’s disease, but the presence of the infection in sheep, goats and deer is also not unlikely. The influence of parturition in precipitating a crisis in cases of Johne’s disease has been repeatedly seen and it is possible that other factors may play a part in the sudden break down of an animal’s natural protective mechanism. Mineral deficiency has been suggested and it has been observed in England that there is some immunity from losses from this disease on farms with a sandy soil, but that mortality from this cause is very severe upon granite soils. From the experience of certain unprogressive clinical cases at Muktesar, it is conjectured that environment and soil variations may play a part in the incidence and progress of this condition in India.

The question of control measures is very much in the experimental stage and it is doubtful if any medicinal treatment or prophylactic or curative vaccine can be considered satisfactory. Extensive trials have been given to several drugs, in particular a preparation of Iron Sulphate orally, and formalin intravenously, but with little success. Non-specific protein therapy with egg-albumin has been tried at Muktesar, and on destroying some of the treated cases, which were previously diagnosed as suffering from Johne’s disease, no signs of the disease were detected at post mortem, but environment may have played a part in the retrogression of these cases. What is required is a drug which will act directly on the causal organism, on the analogy of the specific action of chaulmoogra oil on the Leprosy bacillus, another acid-fast organism.
Vaccination.—Certain phenomena connected with the acid-fast group indicate more than a mere accidental affinity between the organism of fowl tuberculosis and the Johne bacillus and it was on the basis of this affinity that the Johne organism was first successfully cultivated, the diagnostic agents, avian tuberculin and Johnin were evolved and finally Vallee and Rinjard prepared their prophylactic vaccine, which raised great hopes all over the world. The possibility of a cross-immunity, such as is aimed at in a method of vaccination, being set up in nature has been suspected in India by Soparkar (1927) who observes:—One batch of these animals (cattle with admixture of European blood) was obtained from a place where Johne’s disease was very prevalent. Whether this circumstance had any connection with the very high resistance (to tuberculosis) displayed by the animals, it is difficult to say." A successful vaccine, if one such were available, would obviously be a great boon to cattle owners in India, and for several years past, therefore, large scale experiments have been undertaken at Muktesar on more than one occasion to study the vaccinating relationship between the bacilli of Johne’s disease, and those of avian tuberculosis, but the results obtained have, so far, been inconclusive.
New researches on some helminthic diseases of India.

By Captain S. C. A. Datta, BSc., MRCVS., Veterinary Research Officer, Mukteswar-Kumann, U. P. India.
New researches on some helminthic diseases of India.

By Captain S. C. A. Datta, BSc., MRCVS., Veterinary Research Officer, Mukteswar-Kumaun, U.P. India.
It must be stated at the outset that the present subject has been chosen as one of the most suitable to be presented from India to this Congress, since one of the most vital problems facing the country to-day is represented in the general degeneration, stunted growth, defective nutrition and susceptibility to various diseases, which are insidiously undermining a large proportion of its livestock. Prolonged experience has shown that helminthic infestations are to a considerable extent responsible for this state of affairs, though no doubt the interplay of other potent factors appears to be equally involved. No exact data, however, exist of the economic implications of helminthic infections, though the total losses due to diminished productivity and even malaise and death must be considerable. Possessing as India does, the largest cattle population of the world, and gradually diminishing areas of pasture land and the extreme degree of overstocking what pastures still exist, accentuated by the presence of very favourable topographical and climatic conditions for the multiplication and rapid spread of helminths, together with the extension of canal irrigation and frequent inundations, it stands out preeminently as the home of most livestock parasites, while its farm stock in many cases are veritable walking museums of helminthic fauna. The subject of helminth parasitism of animals therefore attains the status of a major problem at the present time, not only from the purely veterinary aspect, but also from the standpoint of public health.

References to the worms of domestic animals are no doubt available in some of the ancient Indian veterinary treatises such as Aswinaitak, and one knows that collections of Indian parasites have been examined by some early helminthologists abroad from time to time. However, the attention of the comparatively few veterinary workers of the Army and Civil departments of the Government of the East India Company and its successor, commencing practically from the beginning of the nineteenth century till the present time, has been so completely pre-occupied with the ravages of epizootic diseases like rinderpest and surra that nothing beyond the sporadic and superficial consideration of the gross anatomy and geographical distribution of parasites could be made. Without attempting to go into details of the helminths recorded from India in the early days, when hydatid cysts, the so-called *filaria oculi* of the aqueous humour of horses, liverflukes and amphistome infestations were first seen, it is remarkable that the instances, where the exact role of helminths as disease-producing agents was recognised, have been notably very few indeed. The effect of parasitic worms might have been less spectacular than that of epizootics of bacterial and virus origin, but quite a number of highly refractory
diseases had forced themselves on human attention due to their frequent occurrence and prominent gross lesions, though their helminthic etiology remained enshrouded in mystery until a few years ago, providing, as is usual in such cases, a very rich field for speculation and controversy.

Turning to other countries as well, the position is far from satisfactory since the number of diseases proved to be of helminthic origin are comparatively few, when the helminthic fauna of the livestock of each country is taken into consideration, and the life-history of quite a number of worms is unknown. Helminthic diseases are known to be characterised by widely different lesions including haemorrhages, inflammatory and ulcerative reactions, nodular growths and even cancerous changes; and regarding the association of the last mentioned with helminthiasis, which is rather exceptional, it may be mentioned in passing that the writer has encountered them in equine gastric habronemiasis, and also in another nematode infection, the so-called hump sore of cattle. The basic facts which are responsible for the production of these wide variations in the clinical manifestations of all helminthic invasions are awaiting elucidation, but when the findings on equine microfilariasis of all kinds recorded by different workers, were analysed, Sen (1927) failed to resist the impression that microfilariasis of the horse is not associated with any clear clinical picture. Again, facts regarding the everchanging or dynamic biological relationship of helminth parasites to the inside of the body of their hosts, and the physiological mechanism by which enormous number of parasites are harboured without the exhibition of any apparent deleterious effects while a hitherto innocuous parasite leads to lethal effects, form subjects of absorbing interest, and here verminous pneumonia in ovines (due to Varostrongylus pneumonicus) and in the buffalo (due to Protostongylus sp.) may be cited. Incidentally, the observation made in India that when the snails, Indoplanorbis exustus are infested with an aquatic oligochaete, Chaetogaster limnaei they cannot be infected with Trema-tode larvae, must be mentioned. Furthermore any attempts made with the aid of the microscope, to follow parasitic action in the tissues of the mammalian hosts, to appreciate the nature of the tissue damages and to correlate the histological lesions incited are likely to increase our powers of control over helminthiasis.

For the above reasons the writer has devoted a considerable amount of his time and thought to the pathological aspect of helminthiasis and, as will be observed, has been rewarded by a rich harvest. The knowledge that has been gathered in India during the last few years on a group of hitherto obscure diseases in which the helminthic etiology now stands well-established, has been incorporated in this paper in the hope that a discussion on the Indian disease entities, some of which at any rate probably occur in other parts of the world, may be helpful in the understanding of the larger issues involved in the pathogenesis of helminth parasites. The specific disease entities will be dealt with as briefly as possible in the same chronological order in which their etiology was worked out: Bovine Nasal Granuloma („Snoring disease“), Liver lesions in debilitated
equines (calcareous degenerations, Smith), Bursati in horses, Equine lichen tropicus („Prickly heat“ or „Summer Mange“), Haemorrhagiques boutons (Dourine-like plaques in horses, Pease), Hump Sore of cattle, „Periodic Ophthalmia“ in horses (Verminous Ophthalmia), and a new microfilarial dermatitis of cattle.

Bovine Nasal Granuloma.

Of the various types of nasal growths affecting cattle and other animals in India, „snoring disease“ or „nasal granuloma“ of cattle has been the most important and widespread, being known to exist for nearly a century. The symptoms and lesions need not be described here. Suffice to say that the first histological work (by Krishnamurti, 1922, 1925 and by Cooper, 1923, 1925, 1931) revealed certain alcohol- and acid-fast „clubs“ and „granules“ in the granulomatous tissues of the disease, akin to those of actinomycosis (cf Schlegel’s earlier report on nasal actinomycosis in German cattle). Streptothrix strains were cultivated but no Gram-positive mycelia detected in the „granules“. The non-involvement of the jaw and the tongue, chronic nature of disease, its enzootic distribution, repeated failure in experimental transmission, failure of iodine treatment, and the specific response to tartar emetic, also simultaneous existence of Rhinosporidiosis in an enzootic area (Edwards, 1929 and Das, 1929) were known to characterise the disease.

Besides the actinomycotic theory (Krishnamurti, 1922), the disease was ascribed to the „nose-string“, nasal polypus and to a protozoan organism (Cooper,1931) till finally the disease was proved at Mukteswar in December 1931 to be a clinical manifestation of Schistosomiasis, in which the parasite, closely allied to S. spindalis but possessing differences suggestive of a new species, deposits its ova in a very unusual site — the nose (Datta, 1932). The eosinophile character of the „clubs“ and „granules“ intense eosinophilia in the tissue, and around the essential lesions of the disease — the follicles of Krishnamurti, which were identical with bilharzial pseudotubercles of Fairley, containing in the central core the highly refractile egg shell with larval remnants or even fully developed miracidium were demonstrated. Complete „spindalis“ type of ova, and miracidia, both free and enclosed, and even extruding from the egg shell were seen in sections in addition to worms in copula. Unusually prominent cuticular tubercles were observed on the males, and the „clubs“ around the egg shells were explained as a manifestation of the host’s protective reaction, similar to what happens in a variety of affections including Schistosomiases turkestanicum (Yamagiva, 1931) and japonicum (Hoeppli, 1932). The „clubs“ may be „albumen-mineral compound“ of Levaditi or may represent the secretion of the lateral glands of the miracidium (Hoeppli). Live specimens of the nasal parasite were collected during surgical operation, and the cross-section of a male schistosome,
cut posteriorly to the bifurcation of the intestinal caeca, was identified in a microphotograph said to be of an actinomycotic granule in Krishnamurti's Memoir (1925). Regarding differential diagnosis of Rhinosporidiosis, a fragile, spongy and pediculated growth, as opposed to the solid growths with broad bases of nasal schistosomiasis, the microscopic examination of caustic potash treated deposits from nasal discharges reveals the specific bodies of both.

The research activities on the problem were greatly energised on the above discovery and several articles were published in close succession confirming the etiological finding, and what is more, in extending the work to include specific identification of the parasite, and curative and other control measures in enzootic areas. Space does not permit details but the most outstanding contributions are contained in a series of articles by Rao (1933, 1934). The disease has been recorded from the buffalo, sheep and goats. Differential features of the adults, their miracidia, cercariae and ova have been described in detail. With carefully planned transmission experiments, employing the cercariae of the Nasal schistosome and those of *S. spindalis* by the oral and nasal routes, the intestinal parasite *Spindalis* has been differentiated from the nasal parasite and *Cercariae Indicae Indicae* XXX, Sewell 1922 has been proved to represent a developmental form of *S. nasalis*, Rao. The intermediary agent of *S. spindalis* is *Planorbis exustus*, and that of *S. nasalis* a Limnea species. Further the fact that Sewell (1930) placed *Cercariae Indicae Indicae* XXX, 1922, and Cercariae of *S. spindalis* into two separate groups in his classification is of more than ordinary significance. Tartar emetic is recognised as the cheap and effective curative agent for the disease, and the latest indication (Rao, 1936) shows that for every 100 lbs. body weight 1.5 grains may be repeated daily for 6 days, 2.5 grains may be given every alternate day for 3 days, and any dose exceeding 3.5 grains is dangerous, particularly if repeated. Antimosan, though equally efficacious is comparatively costly for general use. Snoring Diseases of cattle have now been reported from Australia, (Albiston and Gorrie, 1935) and America (Creech and Miller, 1933) but the etiological agents are apparently different being blastomyces in Australia, and rhinosporidium-like in America.

Liver lesions in debilitated equines.

The occurrence of persistent debility among equines in India, associated with nodulated calcareous lesions in enlarged livers has attracted attention since the observations of Frederick Smith in 1885. Hepatic cirrhosis of various kinds, generally ascribed to poisoning, has been recorded from different countries, but none excepting the cases of Oreste and Ercolani have any resemblance to the Indian condition. The problem has been
one of the most difficult to solve, as previous studies indicated that „the lesions represented the end result of an infection, in which it was no longer possible to discover the causal agent.“ Calcareous degenerations in the horse (Smith) „internal Bursati“ (Oliphant, Meyrick, Smith Burke) and other terms were employed.

Hundreds of animals, which had hitherto been destroyed as „worn out“ and „Chronic bad doer“ have now been shown (Datta, 1933) to be cases of intestinal schistosomiasis due to S. indicum Montgomery, the predilection seat for the disposition of the ova being the distal portions of the intestinal tract, particularly the large colon and rectum, and the liver. The parasite does not localise in the central nervous system to produce paraplegia or Kumri. In extreme cases, the enlarged livers have weighed as much as 96 and 105 lbs., giving the subject the appearance of a gravid mare. The back muscles are greatly atrophied and the abdomen pendulous, resembling human cases of chronic schistosomiasis of the Far East, as depicted by Byam and Archibald (1923) and other authorities. The causative ova of S. indicum has been demonstrated as the incitant of lesions in most tissues including the liver, intestines, lungs, mesenteric and other lymph glands and the spleen. Clusters of the ova have been found embedded in all layers of the intestinal coat, but only single ova in the other tissues. Indoplanorbis exustus has been seen to be common in enzootic areas. Cases of persistent debility in horses due to habronemiasis, (H. megastoma) strongylosis and malnutrition do occur in India, but the commonest incitant of debility is S. indicum. Diagnosis by faecal examination is not always easy. The parasite occurs in the sheep, goats, cattle, buffaloes and the camel, and Montgomery (1906) failed to connect it with any obvious pathological entity. Control and curative measures are similar to those of other schistosomiasis.

**Bursati in Horses.**

This summer complaint of horses had been known to Moorcroft in 1808 at Pusa, and very numerous articles on the subject have been published to date. The name is widely known abroad. The precise nature of the cause remained undetermined, and even to-day statements (Wooldridge, 1934) are made alleging that it is an established fact that helminths are not causally connected with Bursati in India. Circumstantial evidence and the analogies of „Summer sores“ and similar complaints had suggested a habronemic origin of the disease (Sen, 1927) but authorities like Railliet (1915), du Toit (1916) have included several entities under Bursati, while Freeborne, Hart and Howell (1927) and Bayliss (1929) are disinclined to accept the habronemic origin. Even if the habronemic etiology is accepted, the exact species responsible for Bursati and other forms of „Summer sores“ are not precisely known, and the mechanism of infection is by no means clear. For while H. megastoma is recognised as the cause of
Summer sores in France, and of swamp cancer in Australia, *H. muscae* appears to be the cause in Brazil, Congo and India. In one of the latest publication (Descazeaux) on „Les plaies d’été“, the „umbilicated crusts“ (Gunn) of India have been included wrongly under Bursati, as will be shown below, but the important point made by the author is that *Summer sores* are produced by the disposition of larvae into wounds by flies, and that the cutaneous and pulmonary lesions occur particularly in horses which are carriers of adult *H. megastoma* in the stomach. Descazeaux further states that the larvae do not take the circulatory route to arrive at the cutaneous lesion, as their dimensions prevent their passage through capillaries, and that his experience shows that all horses affected with *Summer sores* are carriers of *H. megastoma* while the wide spread *H.muscae* is seen in all horses with or without sores. The author records the finding of habronema larvae in the equine stomach towards the end of Summer.

Contrary to the above experiences of Descazeaux, cases of cutaneous and pulmonary habronemiasis in the same animal have been found to contain in the stomach no other species than *H.muscae* (Datta, 1933), and the parasites have been invariably found inside blood vessels in the earliest lesions, while large numbers of *H.megastoma* have been quite frequently seen in healthy horses with no apparent deleterious effect, similar to the experience of Knowles and Slocock (1931) in Egypt. Further Datta (1933) examined histologically *megastoma* gastric tumours, collected during different times of the year over a period of years, but could not confirm the view of Descazeaux that females bring forth young but once during 12 months. The disease in India has never been transmitted by direct or indirect contagion in the same or adjoining stable to healthy in-contacts possessing cutaneous wounds, and repeatedly the disease has been seen to appear first as intact tumours and to spread to different distal parts in an affected subject, indicating larval migrations through the circulation. The causative parasites have been seen in histological sections of the healed lesions, explaining the nature of so called „Bursati diathesis“ observed by many workers. The habronemic etiology of Bursati has now been confirmed by other Indian workers.

**Equine Lichen Tropicus.**

Numerous articles have been published since 1879. Investigations from many different angles have been carried out, and numerous names applied to it including „umbilicated crusts“ (Gunn). The condition has been likened to a number of human diseases like prickly heat, Dhobie’s itch to which it has no resemblance. Methods of prevention and cure hitherto adopted have been singularly fruitless, though like Bursati, spontaneous cures occur in the cold weather. The disease simulates Bursati in its seasonal incidence, non-contagiousness, recurrence in
the same subject year after year, but is a more superficial affection. It is characterised by its severe irritable, chronic popular and scurfy nature. The mane, tail, sides of the neck, shoulder region, and abdomen are predisposition sites. In the work carried out at Mukteswar since 1931, young forms of a round worm (microfilariae) have been constantly demonstrated in the sections of the affected skin, and adult onchocercoid worms have been collected from nuchal ligaments of some of these cases (Datta 1932). The disease may be described as a microfilarial pityriasis, and presents features of Crew-Crew of man of the West Coast of Africa. The drugs Novarsenobenzol and antimosan are recommended for injection. The microfilariae have never been seen in the blood.

In addition to India, the disease occurs in Sudan, the Philippine Islands Underwood, 1934), U. S. A. (Alicata, 1936) and probably also in other countries, since some of the cases described as Queensland Mange (Tryon, 1888), Dry scurfy eczema, mane and tail disease, Bran and scale eruption (Friedbergher and Frohner, translated, 1908), and as dandruff, seborrhoca oleosa and sicca, chronic scurfy eczema of the long haired parts of the body of unknown etiology (Hutyra and Marek, 1926) must refer to the microfilarial itch now discussed. Semmer (1871), Baruchello (1889) and Aldige (1920) perhaps were describing the same disease.

,,Dourine — like plaques“ in equine microfilariasis.

Darmagnac encountered this condition in France, and Pease (1910) described it from India. This disease is different from the condition already discussed and the chronic scurfy and irritable features are not present. In the case studied by the present author, nodules appeared each morning for a few months, and by about midday an exudate of apparently pure blood escaped (haematidrosis, ,,Summer haemorrhage“) ,,Nodules“ of the previous day healed and fresh nodules appeared again in the next morning. The parasite was found to be Parafilaria multipapillosa, which produces dermatorrhagia in calves and buffaloes in several parts of India.

**Hump sore of cattle.**

These refractory sores on the skin of cattle have attracted attention for about 40 years, and villagers have called it ,,Bursati“ of cattle. The severity of the disease varies with the season. Holmes, Raymond, and Dey (1925) studied the disease, and it was considered to offer a portal of entry to the causative agent of bovine lymphangitis (due to Pasteurella pseudotuberculosis type III). ,,Bursati“ in cattle is said to occur in Sind and Bombay in India, in Egypt, and in Bulgarian cattle (Iwanoff, 1934). Our previous experience on the pathology of some of the above helminthic diseases suggested that a nematode may be the causative factor. The important publication of Bubberman and Kraneveld, (1933) on Cascado as a Stephanofilariasis was received at this stage.
While opportunity of receiving material to test the idea was being sought, Pande (1935) was successful in establishing the disease to be of nematode origin. In the subsequent researches of Datta (1935), adult nematode larvae, and ova showing wriggling larvae were detected in the affected tissues from the hump, region of the yoke gall, and other situations in cattle from different provinces. The adults were discovered at more superficial situations than the nests of larvae. No cases of the disease in goats and buffaloes have yet been seen in India, contrary to the experience of the Dutch workers. The description of the disease as „Dermatitis squamosa et crustosa circumscripta“ is however pertinent, but the name suggested for future use by Bubberman — „dermatitis verminosa bovis“ will not be sufficiently specific, since other similar entities exist, as shown below. The Indian parasite has been found to differ from *Stephanofilaria dedoesi* of Dutch Indies, and from *St. stilesi* of America and has been named *Stephanofiloria assamensis* Pande.

„Periodic ophthalmia“ of horses.

The occurrence of *Thelazia lachrymalis* on the exterior of the eyes of horses, and the infection of the aqueous humour of the equine eye with the so called *filaria oculi* have been known in India. Recently, however a separate condition affecting one and sometimes both eyes of young horses in certain North Indian stud farms known for sometime as Periodic ophthalmia (recurrent *sensu stricto*) in those places has been proved to be a microfilariasis (due to unsheathed larvae) similar to the ophthalmia associated with *Guatemala nodules* of man, but the adult remains undiscovered. (Gyer 1938). Virus, bacteria, dietetic and extraneous factors were eliminated. The parasite larvae were seen in the *substantia propria* of the cornea and in the lachrymal glands with definite histological reactions. Previously Crawford and Nicolle (1925) described what appear to be three different helminths from cases of ophthalmia.

*A new microfilarial dermatitis of cattle.*

Recently another very peculiar and obscure case of cutaneous affection in an old bullock was studied. This animal one of the several housed together in the same stable, had been suffering from the complaint for 2 years, and a transfer to the hills did not produce any appreciable amelioration, contrary to the behaviour of several other helminthiasis like Bursati Lichen tropicus etc. The cutaneous covering of the animal presented strikingly patterned appearance due to numerous arrow-shaped scars of healed lesions. The animal was in good bodily condition and fed normally, but the apparently healthy skin peeled off like parchment paper on the slightest injury or abrasion. Bright red, sensitive bleeding raw surfaces were thus exposed on different parts, resembling similar lesions.
described by Oguni in 1927 under "an elephantiasis-like disease" in Japanese cattle. No symptoms of elephantiasis were however seen in our case, which may be an altogether new condition. Specimens of the affected skin revealed large numbers of microfilaria but none were seen in the blood. The disease was generalised and had no resemblance to stephanofilariaisis, and the parasite must differ from Parafilaria multipapillosa. The adults remain undetermined, and a transmission experiment carried out with mosquitoes appears to have failed.

The chance association of microfilariasis with other diseases has been discussed by Macalister (1917), and the view that a more intensive search would result in the finding that a proportion of the equine species normally harbour these parasites without developing any visible symptoms has been emphasised by Sen (1927). The cutaneous diseases caused by larval nematodes discussed above are therefore of considerable interest.

Summary.

The writer has devoted time and thought to the pathological aspects of Helminthiasis and has been rewarded by a rich harvest. The knowledge that has been gathered in India during the last two years on a group of hitherto very baffling diseases, in which the helminthic etiology now stands well established, and some of which occur in other countries, has been incorporated in this paper: —

Bovine Nasal granuloma or snoring disease has been shown to be caused by Schistosoma nasalis, n.sp., the snail Limnea sp. acting as the intermediary host. Transmission experiments and morphological details differentiate the parasite from S. spindalis. Tartar emetic and antimosan act as curatives (Datta, 1932).

Liver lesions in debilitated equines, first described by Frederick Smith, have been shown to be caused by Schistosoma indicum. Enlarged livers may weigh up to 105 lbs. Indoplanorbis exustus may act as the intermediary agent (Datta, 1933).

Bursati in Horses. The habronemic origin due to H. muscae has been proved and confirmed (Datta, 1933), removing all confusion regarding entities included under this term. Larvae of H. muscae reach the skin through the circulation. Larvae of H. megastoma in gastric tumours were present in all parts of the year.

Equine Lichen tropicus., known for very long, now shown to be a microfilarial pityriasis (Datta, 1933), involving the mane, tail, sides of the neck, shoulder region and the abdomen. It is characterised by severe irritable, chronic papular and scurfy nature and resembles Craw-craw.
of man. *Onchocerca* sp. are present in the nuchal ligaments of some affected subjects.

*Dourine like plaques in equine microfilariasis*, due to *Parafilaria multipapillosa*, as a distinct condition from the above, has been described.

*Hump Sore of cattle*. The parasite *Stephanofilaria assamensis* differs from the species described from Dutch East Indies as the cause of *Cascado*, and from America. The disease is characterised by a chronic crustaceous, circumscribed sources.

*Periodic Ophthalmia of horses*. This recurrent ophthalmia involves one or both eyes of young horses, and is caused by unsheathed nematode larvae (*onchocerca ?*) similar to ophthalmia associated with *Guatemala nodules* of man.

A *new microfilarial dermatitis of cattle*, characterised by the peeling off of apparently healthy skin like parchment paper, on the slightest injury or abrasion has been described.

**Zusammenfassung.**

Der Autor hat sich der Pathologie der Helminthiasis gewidmet. Die Erfahrungen über eine Gruppe bisher rätselhafter Krankheiten, deren parasitäre Ursache nun feststeht, wurden in den letzten zwei Jahren gesammelt.

Das *Nasengranulom* oder die Schnarchkrankheit der Rinder (snoring disease, nasal granuloma) soll durch *Schistosoma nasalis* nov. spec. verursacht sein und die Schnecke *Limnea* sp. dient als Zwischenwirt. Übertragungsversuche und morphologische Einzelheiten lassen den Parasiten von *S. spindalis* unterscheiden. Brechweinstein und Antimosan wirken als Heilmittel (Datta, 1932).

Die von Frederick Smith erstmals beschriebenen *Leberschädigungen bei Pferden* werden durch *Schistosoma indicum* verursacht. Die vergrößerten Lebern wiegen bis zu 105 lbs. (= ca. 47 kg). Die Schnecke *Indoplanorbis exustus* dürfte sich als Vermittler betätigen.


*Lichen tropicus* der Pferde, seit lange bekannt, ist eine durch *Microfilarien* bedingte Pityriasis (Datta, 1932), erstreckt sich auf die Mähne, Hals, Schulterregion, Bauch und Schweif und ist charakterisiert durch starken Juckreiz, Bläschen- und Schorfbildung und gleicht der Craw-
craw des Menschen (Filariasi). Onchocerca sp. sind in den Nackenbändern einiger infizierter Tiere gefunden worden.

Dourine-ähnliche Plaques bei der Mikrofilariasi der Pferde, verursacht durch Parafilaria multipapillosa, wurde als eine verschiedene Form von der obengenannten beschrieben.


Periodische Augenentzündung der Pferde. Diese periodisch wiederkehrende Ophthalmie befällt eines oder beide Augen junger Pferde und wird verursacht durch Nematodenlarven (Onchocerca?), ähnlich der Augenentzündung, die unter dem Namen Guatemala nodules des Menschen bekannt ist.

Eine neue durch Mikrofilarien verursachte Dermatitis des Rindes wurde noch beschrieben, bei welcher sich die scheinbar gesunde Haut durch die leichteste Verletzung oder Schürfung wie Pergamentpapier abschält.

Résumé.

L'auteur s'est voué à l'étude pathologique de l'Helminthiase. Les expériences d'un groupe de maladies peu connues ayant été pendant ces deux dernières années l'objet d'études approfondies, en ont établi la source parasitaire.

Le Granulome nasal ou maladie ronflante (snoring disease), a prouvé la présence de Schistosoma nasalis nov. spec. et c'est l'escargot Limnea sp. qui tient lieu d'hôte intermédiaire. Des essais de transmissions et des détails morphologiques permettent de distinguer entre le parasite et S. Spinalis. Le tartre stibié et l'antimosan ont un effet curatif (Datta 1932).

Les lésions du foie chez les chevaux débiles, décrites en premier lieu par Frédérick Smith, sont causées par Schistosoma indicum. Les fois grossis pèsent jusqu'à 150 lbs. (env. 47 kg.). L'escargot Indoplanorbis exustus peut être admis comme agent intermédiaire.

Bursati des chevaux. Habronema muscae a été prouvée comme cause de la maladie (Datta 1933) et tous les doutes enregistrés sous ce nom ont été écartés. C'est par la circulation que la larve parvient à la peau. La larve de H. megastoma se trouve dans les tumeurs gastriques à toutes les époques de l'année.
Lichen tropicus des chevaux, connu il y a longtemps est une pityriasis provoquée par une microfilariose (Datta 1932), recouvrant la crinière, l'encolure, les épaules, le bas-ventre et la queue. Elle est caractérisée par une forte démangeaison et des ampoules chroniques, formant eschare, ressemblant au Craw-Craw de l'homme (Filariasis). On trouve Onchocerca sp. dans les ligaments cervicaux des sujets infectés.

Les plaques semblables à celles de la dourine dans la microfilariasis des chevaux due à la Parafilaria multipapillosa, constituent une forme distincte de celle déjà décrite ci-dessus.

Hump Sore des bovins. Le parasite Stefanofilaria assamensis diffère de l'espèce décrite aux Indes Néerlandaises, qui cause le Cascado, comme de celle de l'Amérique. La maladie est caractérisée par un éczéma croûteux, localisé.

La fluxion périodique des chevaux. Cette ophtalmie récidive attaque un œil ou les deux yeux des jeunes chevaux et est provoquée par des larves de nématodes, larves (onchocerca?); elle est semblable à l'ophtalmie humaine, connue sous le nom de nodules du Guatemala.

L'auteur décrit encore une dermatite nouvelle causée par des microfilaria; la peau apparentment saine se desquame par plaque comme du parchemin sous l'action de la plus petite blessure ou égratignure.

Riassunto.

L'autore si è dedicato allo studio della patologia delle elmintiasi. Egli riferisce sulle esperienze, raccolte in questi ultimi due anni, su un gruppo di malattie le cui cause, dapprima sconosciute, furono poscia identificate di carattere parassitario.

Il granuloma nasale o malattia del russare (snoring disease) dei bovini è determinato dallo Schistosoma nasalis n. sp., il cui ospite intermedio è la lumaca Limnea sp. Gli esperimenti di trasmissione e le particolarità morfologiche lo differenziano dallo S. spindalis. Il tartaro stibiató e l'antimosan svolgono un'azione terapeutica (Datta, 1932).

Le lesioni del fegato nei cavalli debilitati, descritte per la prima volta da Federico Smith, sono causate dallo Schistosoma indicum. I fegati ingrossati pesano fino a 150 lbs. (= circa 47 Cg.). La lumaca Indoplanorbis exustus sarebbe l'ospite intermedio.

Bursati dei cavalli. L'Habronema muscae è riconosciuto e confermato quale agente causale di questa malattia (Datta, 1933); vanno quindi scartati i dubbi ancora esistenti in proposito. Le larve dell'H. muscae raggiungono la pelle attraverso la via circolatoria. Le larve dell'H. megastoma furono riscontrate nei tumori gastrici in qualunque epoca dell'anno.
Il Lichen tropicus dei cavalli, già noto da molto tempo, è una pitiriasi causata da microfilarie (Datta, 1932) che invadono la criniera, il collo, le spalle, la regione addominale e la coda. Essa è caratterizzata da forte prurito e da una formazione di vescicole e di escare, rassomigliando così al Craw-Craw dell'uomo (filariosi). La specie Onchocerca sp. fu trovata nei ligamenti cervicali di alcuni animali infestati.

La microfilariosi dei cavalli, con placche affini a quelle della durina, è determinata dalla Parafilaria multipapillosa; è quindi una forma distinta da quella descritta più sopra.

Hum-Sore dei bovini. Il parassita Stefanofilaria assamensis differisce dalla specie riscontrata nelle Indie olandesi (causante il „Cascado“) ed anche dalla specie americana. La malattia è caratterizzata da un ezzema cronico circoscritto.

Oftalmia periodica dei cavalli. Questa oftalmia recidivante colpisce uno od ambedue gli occhi dei cavalli giovani ed è determinata da larve di nematodi (onchocerca?); essa rassomiglia all’oftalmia umana, conosciuta col nome di Guatemala nodules.

Fu pure descritta una nuova dermatite da microfilarie dei bovini, nella quale la pelle apparentemente sana si lascia scorticare come carta pergamena alla minima ferita o scarificazione.
The Contribution of the Study of Animals and their Diseases upon the Progress of Medical Research

By

S. C. A. Datta, B.Sc., M.R.C.V.S.

Reprinted from Agriculture and Live-stock in India
Vol. IX, Part III, May, 1939
THE CONTRIBUTION OF THE STUDY OF ANIMALS AND THEIR DISEASES UPON THE PROGRESS OF MEDICAL RESEARCH*

BY

S. C. A. DATTA, B.Sc., M.R.C.V.S.

Veterinary Research Officer, Imperial Veterinary Research Institute, Mukteswar

EVOLUTION OF THE CONCEPTION OF DISEASE

The phenomenon of life in all its forms is intimately connected with its environment, and the life activity of each species of creation is vitally concerned with that of other. The crux of this fundamental biological fact is the food problem. In every living being therefore there is a relentless struggle for existence going on from the very time of conception to death, as each is trying to exist by devouring the other. To appreciate adequately the intensity and extensiveness of this ever raging warfare requires more than an ordinary acquaintance with the different grades of creation in the economy of Nature. Germs and parasites of disease attack man and his animals, and epidemics and epizootics have been known to occur since the earliest times. Humanity has been constantly terrorised by the suffering and murder of individuals and communities perpetrated by disease agents. Thus the instinct of self-preservation and the longing to relieve suffering in fellowmen have been the incentive to the study of the intriguing problem of disease (pain, suffering; causation. Animal diseases have been studied simultaneously, primarily from the utilitarian aspect, now enormously extended with the progress of time and civilisation, and occasionally also from the humanitarian urge. Diseases have been so poignant and sudden in their effects that to the terror-stricken ancients they appeared to be supernatural visitations. Commencing from about 500 B. C. diseases have been considered to be processes of nature but it was not until 2,000 years later that impartial observation of Nature was resorted to as the means by which to solve mystery.

In surveying the progressive evolution of the conception of disease one notices that the medicine of man and animals was born almost contemporaneously of the common parentage of credulity and empiricism, and that as rationalism dawned, the approaches to the problem of disease widened. The accruing benefit in the saving of life and relief of suffering applied equally to the human race as to the animal species, and the science of disease today

* This article was presented for discussion at a joint meeting of the Medical, Physiological and Veterinary Research Sections at the Jubilee Session of the Indian Science Congress held in Calcutta in January, 1938.
forms a major study of human welfare. In this study, general principles of Pathology emerged showing how changes in the bodily functions (symptoms) and organic alterations in the structure of the special and general tissues composing the living body (lesions) are set up. The concept of cells composing tissues applied to all biological sciences, and the pathological lesions were found to be the gross structural expressions of the disproportions in the relative values of stimuli and cell reactions (cellular pathology). The complex mechanism of predisposing factors, exciting causes, individual susceptibility and resistance, nutritional bearing, effect of climatic vagaries, and parasitic action were elucidated. Important facts regarding host-parasite relationship, parasites' methods of attack, hosts' mechanism of partial or complete resistance due to natural factors or acquired through the aid of biological separations or drugs, factors determining latent infections, carriers and relapses and the bases for prevention and control, etc., have come to light. Apparently trivial, and even isolated discoveries have produced profound amelioration of suffering. Ideas regarding disease control have undergone fundamental changes, and experience has shown that it is more economic and rational to prevent disease in whole communities rather than obtain cures in individuals after allowing maladies to be contracted.

ESSENTIAL UNITY OF THE HEALING SCIENCE

The science of disease has thus marched forward through the ages but the outstanding fact of immediate concern in this article is that any pioneer work, whether relating to microscopy, protozoa and bacteria by Leeuwenhoek, or that on bacteriology and immunology by Pasteur or Koch, Koch's Postulates or his method of plating out of cultures on solid media, or Lister's work on Antisepsis or similar other advances have been equally applicable to man and animals, and thus was the essential unity of the healing science brought out constantly. Specialisation in individual branches has no doubt tended to a loss of the broad perspective of comparative medicine, and depressing stages of isolation seem to intervene now and again, but the solidarity of human and veterinary medicine stands out unmistakably throughout the ages.

In the investigation of matters of health and disease, the issues involved have been so intricate and complicated, that the human intellect has stumbled many a time. Had it not been for the fact that disease processes in man and animals have been mutually elucidatory by throwing cross-lights and analogies, the difficulties and obstacles in the understanding of conditions, which appeared to be persistently insuperable, might never have been surmounted, and both human and animal medicine would have been poorer. It may perhaps be unfruitful to try to estimate whether the human or veterinary side has benefited to a greater extent than its sister branch in this alliance of long standing, though as far as therapeutics and immunology are concerned the contribution of research upon animals to human medicine has
been of such far-reaching consequences that even a rough estimate is very striking. However, attention need here be restricted to the influence exerted by animal studies on the progress of medical research. The bearing of animals as reservoirs of infection for man and the transmissibility of animal diseases to man either directly or by handling or consuming animal products have been dealt with in another article [Datta, 1938], but it is necessary to note that even in this sphere of animal diseases communicable to man, veterinary researches have been of very great importance in the elaboration of efficient public health measures.

Without exaggeration it may be stated that the knowledge derived from the study of animals and from animal experiments has provided the basis for the development of modern human medicine, whether relating to anatomy, physiology, pathology or therapeutics. This fact is so obvious and yet is not generally appreciated. One knows that ethical considerations have prevented crucial experimentation upon man excepting in the rare instances of convicts, and that essential facts of medicine have emerged largely from analogies from experimental work in large and small animals. Without undertaking the arduous task of giving comprehensive chronological data in this matter, a few of the historical facts, particularly as they relate to the domestic stock, may be recounted. The facts speak for themselves:

**Historical facts.**—The earliest anatomical model known is a clay model of sheep’s liver in Babylonia dated 2000 B.C. The earliest recorded surgical operation is the trephining of sheep for staggers. Blood corpuscles of different species of animals were the subject of early studies. The physiology of digestion, the production of glycosuria, the technique of artificial insemination, and also of intravenous injection were first worked out in the dog. Transfusion of blood resulted directly from the latter injection. Gland-grafting was practised on fowls in 1767, and the effect of the transference of the male and female gonads was also studied on fowls by Berthold in 1849, leading to Steinach’s work on the human subject. The antitoxic property of potassium permanganate in snake bites and cholera, the rational application of arsenic preparations, and the use of carbon tetrachloride are some of the measures adopted from the sphere of animal experiments, besides the animal gland products, pepsin, insulin, adrenalin, pituitrin and others. The still baffling problem of carcinogenesis has received particular stimulus from the experimental work on fowl and dog tumours.

Turning to the domain of helminth parasites, the first one to be described was the parasite of liver-rot in sheep in 1379. Seibeld’s work on duck trematodes provided the first clue to the life-history of flat-worms in general, and the observation in 1758 of strange cysts (hydatids) in the tissues of various domestic animals, and the subsequent finding of similar hooks and suckers in certain tape worms of man, dog and rabbits were of fundamental importance. Feeding experiments in dogs and other animals were carried out and a new method of determining whether such cysts were the larval stage of a particular tape worm was evolved. These earlier achievements in experimental
parasitology were soon extended to the production of trichinosis in healthy animals by feeding experiments with infected meat. In 1883 the complete life-history of *Fasciola hepatica* was worked out, though about ten years earlier cercariae believed to be of these parasites had been detected in snails. On these lines were the life-histories of the parasite of the more serious forms of the human schistosomiasis of Japan and Egypt worked out in 1914 and 1915 respectively.

In another branch of Parasitology, the first pathogenic protozoan discovered was *Eimeria stiedae* of the rabbit (1674) but the finding of the equine *sura* parasite by Griffith Evans was of greater significance. More outstanding than this was the demonstration by Theobald Smith and Kilbourne in 1893 that Texas fever of cattle was a form of tick-transmitted piroplasmosis. The role of arthropods as transmitters of protozoan conditions being thus brought to light, rapid progress followed. Two years later Bruce showed the transmissibility of *Trypanosoma brucei*, also a parasite of domestic stock, through the intermediary of Glossina flies. The immediate result of this was the elucidation of the mystery surrounding sleeping sickness. Several distinct disease entities due to trypanosomes are now known. Similarly Ronald Ross's discovery of the transmission of bird malaria through Culex mosquitoes led to the epoch making discovery of the role of anopheline mosquitoes in human malaria. That Spirochaetes, whose exact position is somewhat uncertain, can be conveyed by the bites of blood-sucking insects was demonstrated for the first time in 1903 in the case of fowl spirochaetosis. The intimate relation of Protozoology with Entomology has consequently become a well-established fact. The profound influence of this relationship has been manifested not only in the elucidation of the pathogenesis of many diseases, but the control measures that have been elaborated from a knowledge of the parasite and its vector for dealing with some of the most dreaded of human scourges like sleeping sickness or malaria can rightly be mentioned as the outstanding achievements in tropical medicine.

Sufficient data have been given above to show that, besides the addition of individual items of information, veterinary studies have repeatedly thrown light on the solution of many of the fundamental problems of disease causation and control in man, whether pertaining to Parasitology or Bacteriology. It now remains to be seen how in the latter domain and immunology, even large pieces of research connected with human disease problems have been frequently initiated by purely veterinary studies and that the transcending results have materially altered the destiny of man.

Dealing with the highly destructive disease, Anthrax in sheep, Pasteur laid the foundation of modern preventive vaccination. The Anthrax bacillus was discovered in 1849 in the blood of animals dying from this disease and in 1880 Pasteur discovered the bacillus of Fowl Cholera. His investigations showed that the Anthrax and Fowl Cholera organisms could be attenuated, and that vaccination of healthy animals with the attenuated germs could
be successfully employed to protect against virulent infections. Again
the origin of bacterial filters leading to the discovery of filtrable viruses, and
of the elaboration of the memorable Koch's Postulates can be traced to these
early studies upon animal diseases. Pasteur's work on these two animal
diseases and on swine erysipelas was carried out when he was nearly sixty
years of age. World-wide interest was aroused in the potentiality of immuni-
sation by attenuated germs, and theories to explain the process of immunity
were put forward. In 1884-1886 Theobald Smith, one of pioneers in the
field of infective diseases, carried out experiments with Hog Cholera bacillus
on pigeons and established the principle which was to find later a very wide
application in human medicine. It was shown that by the injection of filtered
products of the killed specific organisms an immunity against living pathogens can be set up. This was the first experiment on immunisation along
these lines, and Von Behring working with Kitasato in Koch's Institute,
Roux and others followed suit. Working with the toxins of the diphtheria
and tetanus organisms, Behring laid the foundation of a anti-toxic immunity.
The serum of animals immunised against attenuated diphtheria toxins was
successfully used as a protective or a therapeutic measure against diphtheria
in experimental animals, and the specific neutralisation of the toxin of the
disease was achieved. After the preparation had been tried out in man,
the product was produced on a gigantic scale (1894) and with this specific
treatment, the lives of innumerable children affected with diphtheria have
been saved. Many attempts have since been made to treat other specific
diseases on the basis of this discovery. The methods employed today on
large scales for the protection of man against cholera, bacillary dysentery,
tetanus, snake poisoning and typhoid fever are all evolved from these early
but monumental researches. Wright has extended the application of the
method to therapeutic uses. In tuberculosis, B. C. G. Vaccination in calves
has largely determined the progress of infant vaccination.

To mention the history of one or two diseases of public health importance
all the essential knowledge on Malta or undulant fever, which is said to occur
in North India, has been obtained from animal studies. Bruce in 1896 de-
monstrated that infection originated from a most unsuspected source, the
milk of apparently healthy goats, and in comparatively recent years cow's
milk containing Brucella abortus has been shown to cause undulant fever.
In 1888 the mysterious illness of fifty-seven persons who had eaten the meat
of a sick cow was traced to what was called Bacillus enteriditis.

There have been other ramifications of these studies upon animals. The
possibility of the transmission of bacterial agglutinins from mother to offspring
was first demonstrated in goats. The final proof of the benefits of Colostrum
as providing passive immunity to offspring and reducing the risks of
bacterial infections in early extra-uterine life was put forward by Theobald
Smith from his experiments on calves, and today this is a recognised principle
in the post-natal care of infants. Again the other phenomenon of Anaphyl-
axis was suggested by the remarkable observation of Portier and Richet in
1902 that certain poisons of animal origin evoked in dogs a condition of hyper-susceptibility rather than immunity. Anaphylaxis originating from the bacterial products of diphtheria was experienced by Theobald Smith, and this discovery was designated the “Theobald Smith Phenomenon” by Ehrlich. In the subject of allergy the experience that certain people would suffer from an attack of “Rabies” if they went near a stable is one of the earliest suggestions traceable. Researches on both anaphylaxis and allergy have been largely concerned with experiments in large and small animals.

The viruses.—Coming to the latest addition to the list of disease producing agents, the important group of viruses, the clinical manifestations of some of which in man and animals have attracted attention from the days of Aristotle and earlier, rabies and smallpox are the first to come to mind. It is remarkable that outstanding advances in the immunology of both these conditions were achieved before there was any clue regarding their etiological agents, and that both of the diseases to be benefited from empiricism should be of virus origin. Regarding smallpox, human inoculations with variola material were no doubt practised in India in the days of Atharvanveda, and the local country tradition that dairy maids who had contracted cow-pox through milking did not contract smallpox existed in Gloucestershire in England, and also in France and Germany. Jenner, however, deserves great credit for converting in 1880 this traditional belief or observation into a prophylactic principle in science. Turning to rabies, hydrophobia and its relation to dog bites were known from the earliest times. Pasteur’s first communication on the subject was published in 1882 and the vaccine treatment elaborated by him, though modified, still holds its ground. It was in 1898 that Loeffler and Frosh demonstrated the filtrable nature of the cause of foot and mouth disease. With the establishment of the virus origin of one disease entity, others were soon added and bovine pleuroneumonia was a close second. Rabies had been transmitted to rabbits by Galtier in 1872 and by Pasteur, Chamberland & Roux in 1881. With the aid of this knowledge, immunisation of man with rabbit-fixed virus was carried out in 1885, and the fact that the infectivity of viruses can be modified by passage through lower animals has been a matter of wide practical application in immunology.

The failure to cultivate viruses in vitro had been a great handicap, and the fact that most of the human virus diseases are not transmissible to animals or are not amenable to passage in series from animal to animal is a serious difficulty. A constant supply of the causative virus as required for further investigations cannot be arranged, but the demonstration of the application of ferrets as experimental animals in dog distemper and swine influenza has led to their use in human influenza researches. Conclusive proof has been put forward recently that an antiserum can be produced even from animals who are naturally insusceptible to the particular virus. The success achieved in the immunisation against distemper may turn out to be as epoch-making and far-reaching in immunology as some of the other animal analogies exploited by Pasteur and others. Regarding artificial cultivation of human
viruses, while all attempts on ordinary media have failed, the success in the
case of bovine pleuropneumonia and agalactia suggests that the growth re-
quirements of this group are peculiar. Again the 'virus' of bovine pleuro-
pneumonia has already been seen, and placed provisionally in the family
Actinomycetaceae, and filtrable phases of some bacteria, spirochaetes, etc.
are known. It may be added that structures, believed to represent the actual
virus of certain other diseases have been seen, and in this connection the
demonstration of minute coccoid elementary bodies in fowl pox and vaccinia-
virus and others is an interesting pointer. The latest finding that at least
one animal virus, that relating to Shope rabbit papilloma, is a heavy protein
is also of more than ordinary significance.

Instances could be multiplied to illustrate the essential unity of the
Medical and Veterinary sciences and the more objective character of the
collection made by the latter towards the building up of the framework
of what may be designated a system of synthetic medicine, the component
elements of which, in so far as they apply to the human subject, have been
for the most part derived only inferentially from a study of animals and their
diseases. One may even envisage, for all time to come, the continuance of
this relationship between the two sister branches of medicine, unless ethical
values are so radically altered as to legalize certain forms of direct exper-
imentation on man—a contingency on which a significant commentary is
furnished by the present cry against animal vivisection.

REFERENCE

Datta, S. C. A. (1938). Agric. & Live-stock in Ind. 8, 110,
REPRINTED FROM

THE INDIAN JOURNAL OF VETERINARY SCIENCE AND ANIMAL HUSBANDRY.

AN UNUSUAL CASE OF CHRONIC RINDERPEST WITH SPECIAL REFERENCE TO THE CARRIER PROBLEM IN THIS DISEASE

BY

CAPT. S. C. A. DATTA, B.Sc., M.R.O.V.S.,
Temporary Veterinary Research Officer,

AND

V. R. RAJAGOPALAN, G.M.V.O.,
Veterinary Inspector,
Imperial Institute of Veterinary Research, Muktesar.

(Received for publication on 31st August 1932.)

(INTRODUCTION.

Since the earliest times rinderpest has exercised the resources of the scientist and has formed the subject of extensive investigations in many countries on account of the very severe economic loss that it inflicts. Some workers have gone so far as to state that an outbreak “may even result in the almost complete extinction of animals in large territories”. However, great uncertainty still exists with regard to several important aspects of its incidence, causation and control. Among the many problems that still await the verdict of the future, the question of the nature of the virus, the seat of its multiplication and the duration of its viability in the various organs of a recovered animal are by no means settled. When one studies the text-book description of the disease one is liable to gather the impression that this affection occurs invariably as an acute, fulminating type of febrile infecting disease, which is capable of being spread over distances in almost every imaginable manner, as will be seen from a statement of Law [1902]. “All the secretions of the diseased animal are apparently infecting, and the virus possesses great vitality so that the channels of infection are almost endless. It is carried in the manure, washed in streams and drains, dried up on hay, straw, feathers and other light objects, or in dust, and blown about by the winds, left in stables, in feeding and watering troughs, in railroad cars, steamboats and ferryboats, loading banks and yards; it is carried in the fresh bristles, in hides and bones, in halters and harness, on waggon shafts and poles, on goads, on boots and coats of men and the feet of dogs, birds and other animals, on the wheels or vehicles, various infected products, the runners of sleighs and by vermin and wild animals”. On the other hand,
the more recent periodical literature contains references which throw light on the real nature of the virus, on its fragility and consequent inability to be transported over long distances through natural agencies such as enumerated above. Further, quite a number of reports are available to show that rinderpest occasionally assumes a mild type becoming very difficult to recognize. This latter fact is worthy of being fully realized since it has certain very important bearings in the control of the disease in such a country as India, where rinderpest is known to be enzootic as a result of the indigenous breeds having attained some degree of racial natural immunity through centuries of exposure to it.

The primary object of this article is to record a rather remarkable case of a bull as an example of the persistence of rinderpest lesions with accompanying infectivity over a very long period, but the mere reporting of this case can be of but little value unless the full implications involved are properly appreciated. It is desirable, therefore, in the first place, to review the present position with regard to the tenacity of the virus, insect vectors, role of wild animals, carriers among other animals such as sheep and goats, etc. Another reason why a review of the position seems indicated is that the literature on the subject is replete with a considerable amount of unnecessary reduplication of experimental investigations apparently as a result of lack of information as to how much had been achieved by previous workers. Dieckerhoff [1890] and du Toit [1916] have reviewed the literature but the position since their articles were published has not been surveyed. Although it is not the intention in this article to attempt any such elaborate or complete review, recent papers concerning some of the connected aspects of the problem must needs be touched upon in passing.

In the past, workers in several countries have encountered a number of apparently spontaneous outbreaks of rinderpest, in which a clear scientific explanation of the origin of such outbreaks is not easily found. Again it will be recalled that at one time it was strongly believed that the disease in reality had a spontaneous origin and that its appearance was not dependent upon the introduction of an infected case from the outside. Amongst Indian workers also, there are several responsible authorities who have expressed such views. For instance, McLeod [1871], a highly qualified medical man of Bengal, remarks "My own impression, derived from a close study of all that has hitherto come to light on the subject is that these diseases (cattle plagues) are enzootic in this country, spontaneously generated by conditions of air and water and not owing to importation from without". A similar conclusion was arrived at by Veterinary Surgeon Farrel [1869] in the same province "from the fact of the disease being present in all months and seasons in this district (24 Parganas), no trace of its importation from other countries being discovered". Of the several workers in other countries who
have expressed the same opinion a reference to Littlewood [1905] of Egypt will be made later in this paper.

**Tenacity of the virus.**

Very conflicting results have been recorded by different workers with regard to the tenacity of the rinderpest virus in blood when kept *in vitro* and yet it is a question that cannot be left in this unsatisfactory state in view of the practical application such information has in the serum-simultaneous method of inoculation. Even prior to the achievement of Nicolle and Adil Bey [1902] in establishing the filterability of the causal agent, Koch [1897] found that the virulence of rinderpest blood is destroyed by prolonged exposure (of 4 hours) to a temperature of 30° C., and that blood so treated produced no "protective action" on being injected into cattle. On the other hand, Theiler [1898] found that blood when subjected to a higher temperature of 37° C. for 2 or 3 days became avirulent. In testing the practical applicability of the serum-simultaneous method of inoculation, Stockman [1905] experienced difficulty in maintaining blood from a sick ox virulent for more than 48 hours even when kept in the ice chest under field conditions in both India and South Africa. Again Todd & White [1914] claimed that virulent blood taken in citrate solution and kept in sealed tubes at —6° to —7° C. retained its virulence up to 22 weeks. Results indicating the retention of virulence by rinderpest blood for prolonged periods such as the above have been obtained by Shilston [1917]. Although the virus in blood, meat and bone marrow was found to survive at air temperature ordinarily from 2 to 9 days, he quotes instances from his experience in which blood stored under aseptic conditions at room temperature remained virulent for periods of 30-40 days and in one instance up to 51 days. Further in another case blood at 0° C. was virulent even after 90 days. Recently Malanidi and Stylianopoulo [1927] found that 2 c.c. of blood kept in cold store for nine days produced a mild thermal reaction only in a calf.

Leaving aside reports of prolonged viability of the virus under artificial conditions, our ideas in regard to the conservation of the virus in blood at ordinary air temperature for 51 days would appear to require revision, as will be noticed from the later findings of Edwards [1924] who states:

"Experiments with virulent blood stored at body temperature, however, showed that the infective agent became inert usually within three days when it was exposed to air, but that it survived very considerably longer, ten days or more, when it was covered with a seal of liquid paraffin. The factor of greatest importance appeared to be the transformation that took place in the blood owing to the escape of carbon dioxide gas and destruction of the virus was rapid during this transformation."
It has been known for a long time that the virus rapidly becomes inert on dilution with water, as shown by Koch [1897] and Nencki [1898], and Edwards [1924] believes that the most destructive agency is an unfavourable hydrogen-ion concentration of the diluting fluid. It is gratifying to note that the experience gained during the last few years has confirmed the above-mentioned statement of Edwards in that successful serum-simultaneous inoculations have been carried out in very distant field stations in India with blood withdrawn and bottled several days earlier at Muktesar.

From the epizootiological point of view, the information concerning the period during which the natural excretions of a diseased animal remain virulent outside the body under various conditions obtainable in nature is of the utmost importance and it is obvious that the success of control measures in the field must be largely dependent upon the correct data. Earlier workers [Müller, Heubner, Dieckerhoff, Friedberger and Fröhner] held that the infectivity could persist in stables, on hay and in manure for four months or more. Controlled experiments carried out by Ward, Wood and Boynton [1914] with a view to determine the nature of the rinderpest virus have shown that it is very fragile and perishes soon after being discharged by the infected animal, and it will be seen that this result is contrary to the older view regarding the tenacity of the virus. These workers found that the virus did not survive beyond 24 hours in corrals bare of vegetations but containing water, that animals became infected in such corrals within half an hour, 12 hours and 17½ hours after removal of the sick but not longer, that virus in urine diluted with water and sprinkled on grass survives for 36 hours in some instances but not always, that faeces mixed with water and sprinkled on grass infected 24 hours later, that faeces and urine mixed with water and kept in the shade remain infective for 36 hours only, and that the virus is not harboured for long periods in contaminated soil. The experiments of Shilston [1917], the results of which agree with those of Ward and his collaborators, showed that in buildings and on areas infected by the natural discharges of sick animals, the rinderpest virus is unable to survive for more than two or three days and when air and sunlight are admitted its destruction is more rapid.

Insect vectors.

Attempts have been made by several workers to demonstrate if vectors can be responsible for the spread of rinderpest. Following upon transmission experiments with leeches, carried out by Nicole and Adil Bey [1899], Boynton [1913] has shown that blood taken from an infected animal by the ordinary water leech may remain virulent for at least 25 days, and that if it were crushed in water so that the blood content of its stomach escaped, the surrounding water was capable of infect-
UNUSUAL CASE OF CHRONIC RINDERPEST.

It is possible that a fresh outbreak at some distance from the previous ones can scarcely be explained on this basis. Curasson [1922], working in Poland found that crushed ticks (Ixodes ricinus) when injected immediately after removal from an animal at the height of fever produced rinderpest but when injected quarter to one hour after removal they were innocuous. Similar experiments with a fly (Tabanus) gave him only negative results. De Souza [1924] found that a specimen of Margaropus bovis which had engorged itself upon an animal infected with rinderpest maintained the virus at full virulence for 7 days. In his experiments at Muktesar, Sen [1925] was unable to demonstrate the possible rôle of mosquitos, house flies and lice as vectors of the disease under natural circumstances, but he was able to record some positive results when crushed material (Musca domestica) was used subcutaneously. The experience of Hornby [1926] shows that Glossina morsitans can transmit the infection, for he obtained a positive result in one out of two cases. It will be seen from the above that the only report which points to the possibility of infection being transmitted through vectors under natural conditions is the report of Hornby. Since this species of flies is not existent in India, it is apparent from available evidence that there is no real danger in India from insect vectors.

THE ROLE OF WILD ANIMALS.

The improbability of rinderpest infection being transmitted through the medium of intermediary agents, such as insects, flies, ticks, etc., has been seen. There is a danger nevertheless of the disease being spread by direct contact with diseased wild animals. Evidence is available in the Report on Indian Cattle Plagues [1871] of the occurrence of rinderpest in deer, wild buffaloes, sheep, goats, yaks and zebus in Assam, Bengal and Central Provinces. Information collected by Mr. Cooper of this Institute from Provincial Veterinary Departments for submission to the Royal Commission on Agriculture in India shows that the Bison, Sambhar, Spotted deer and small deer are all affected. Pease [1894] states:—"I have seen it in the Kakur or barking deer in the hills, and large numbers perished from it in 1891 beyond Simla. It has been seen in the Gaur or Indian Bison, so called; in the wild buffalo, Tsain, etc." A black buck was seen at Muktesar [Lingard, 1905] to contract rinderpest and succumb to it. Recently Ono and Kondo [1923] found that the deer (Cervus sika) is susceptible to rinderpest and that the virus disappears
from the circulation within six days of recovery and Jacotot [1927] has also transmitted rinderpest to a deer (Cervus aristotelis). It is a well-known fact that wild buffaloes often mix freely at night with the herds of village buffaloes in certain jungli tracts in India.

It is to be remembered that in South Africa the game was almost entirely exterminated in the great rinderpest epizootic which raged among cattle during the years 1896-1898. Hutchins [1915] incriminated game as a spreader of the disease in Africa and pointed out that quarantine measures for the eradication of the disease in such neighbourhoods is not wholly effective and he gave an example [1920] of the appearance of cattle plague in certain parts of the Uganda Protectorate due to the movement of rinderpest-infected game. Zonchello [1917] also incriminated game as a factor in the spread of the disease and held that sero-vaccination should not be practised in such areas, as the game was likely to take the disease from the vaccinated animals and re-introduce the disease into populated land at a later period. McCall [1920] implicated such game as bush buck, wild pig and eland in the spread of rinderpest in Tanganyika. Edwards [1924] and Jacotot [1930, 1932] failed to demonstrate that the rabbit could become naturally infected, hence the rôle of these and other rodents can be safely ignored. Inoue, Harade and Shimazu [1930] proved that susliks (Citellus mangericus ramosus, Thos.) are susceptible to rinderpest and that occasionally these can become “virus carriers”, and their experiments showed that blood from an inoculated suslik showing no symptoms may be infective to cattle.

Although it sounds improbable Gilchrist [1848], from his personal experience in the Madras Presidency, was of the opinion that the circumstance “of a tiger having urined in the vicinity of the herd” of cattle can be productive of rinderpest.

When rinderpest gets freshly introduced into a country to which it is alien, the danger of the spread of the disease to the wild game may be of paramount importance. For, while it may be easy to eradicate the disease from the domestic animals by an efficient veterinary police through registration of cattle, destruction of the affected and enforcement of standstill orders, the uncontrolled movement of infected game may render all these methods valueless. But in a country like India where rinderpest has established a strong foothold and where the existence of numerous extensive territories without any natural boundary renders the adoption of similar methods impossible from political and other grounds, the importance of game as a spreader of the infection may be said to be insignificant or unimportant. As will be shown later, the factor that counts here most is the unsuspected cattle themselves and perhaps also some of the other domesticated animals which move among cattle.
"Carriers" among Domesticated Animals Other Than Cattle

This brings us to the question of "carriers", which is by no means a settled one. The case of the domesticated animals other than cattle may be considered first. Galambos [1861] was apparently the first to establish the susceptibility of sheep to rinderpest. Of the many early workers who have proved the susceptibility of goats to this affection, Nicolle and Adil Bey, Koch, Memo and Rogers may be mentioned. Rogers [1900] refers to an outbreak of cattle plague in the Himalayas which was introduced into two villages by infected goats with most deplorable results. In a more recent paper Zocchello [1917] observed that goats living in the scene of an outbreak did not get the disease naturally and were unable to transmit it to transport cattle housed with them. Gartner [1920] considered goats of such importance that he headed a full chapter on them as "virus carriers" of rinderpest in spite of a little earlier statement by Angeloff [1917] to the effect that sheep and goats, although susceptible by artificial inoculation did not contract the disease naturally. Bliss [1922] working in China suggested that goats could be used as virus-producers in preference to cattle as the former are cheaper, withstand the disease better and recover from the mild attack after furnishing the required amount of virulent blood. Schein [1924] of Indo-China kept goat virus infective to cattle during 172 passages from goat to goat. Topacio [1927] working in the Philippines conceived the idea of utilising the lesser susceptibility of goats to a great practical end, similar to the employment of calves in the production of smallpox vaccine. He had the vision that it might be possible to obtain a virus by a single passage from cattle to goats that was sufficiently potent to infect Philippine cattle and carabasos and be sufficiently attenuated so that the quantity of serum necessary for simultaneous inoculation could be materially reduced. Edwards [1928] reported that after artificial intravenous inoculations of large quantities of virulent rinderpest blood, the virus was detectable in sheep up to one month (the longest time was not determined) but opined that this species of animal was not likely to act as "carriers" under natural circumstances. Robertson [1925] gave an account of rinderpest in Western Australia and stated that the evidence appeared to indicate that sheep brought to Fremantle as live ship's stores and which were purchased by local butchers for slaughter were the source of an outbreak. Beaton [1931] working in Nigeria, proved that goats are susceptible to rinderpest by contact. That contact infection takes place from cattle to goat and vice versa has been proved by Hall [1930].

It is, therefore, definite now that goats do get infected with rinderpest, but the disease is very mild and passes off unnoticed in most cases. With the knowledge we now possess of the degree of attenuation of the virus that takes place within the
body of the goat [Bliss, Topacio, Edwards, 1930 and Stirling, 1932] and from the recorded evidence quoted above, it is probably safe to conclude that such goats may infect cattle in their midst with the attenuated virus, the result of which would appear to depend upon whether the cattle concerned do or do not possess a degree of immunity and upon whether they are in robust health or are weak, debilitated and predisposed to infection. The two extreme experiences of Zonchello [1917] and Robertson [1925], referred to above, are suggestive of the view now expressed.

Carré and Fraimbault [1898] proved the susceptibility of pigs to infection with virulent blood and Penning obtained the same results with wild hogs and was able later to reinfect calves from them. In the description of an outbreak of rinderpest which occurred in the Island of Romblon, where the disease had never occurred before and into which island no cattle had been imported for a long time, Boynton [1916] produced evidence to show that pigs played a rôle in the spread of rinderpest in this constituent of the Philippine Islands. It was found on enquiry that pigs had been imported from another island where rinderpest was prevalent and that first of all local pigs began to die in considerable numbers and later the cattle and carabaos fell victims to it. Nicolas and Rinjard [1921] proved that pigs are of importance in the spread of the disease and sanitary police measures, therefore, should be adopted against these animals if rinderpest has to be controlled. Bliss [1922] also found that hogs were susceptible and thought that these might act as carriers but did not attempt to obtain any experimental proof to confirm this. Experimental proof has, however, been put forward by Molinie [1931], who showed that swine can play an important rôle in the epizootiology of the disease, since in his experiments, the pigs contracted the infection as readily as cattle and further they could infect cattle by contact or by the artificial inoculation of infected blood or urine. He believed that passage through the body of this animal increased the virulence of an attenuated virus but he did not entertain any definite idea as to whether the pig could act as a reservoir of the virus. Conti and Van Dac [1929] have maintained the virus in series in young pigs through 174 passages.

With regard to India, only scanty reports are available. The Indian Cattle Plagues Commission [1871] collected evidence to show that pigs died of rinderpest in Bengal, and Jackson and Cabot [1930] refer to authentic reports of occasional natural outbreaks amongst pigs in the Nilgiris in Southern India.

There seems to be little doubt that rinderpest occurs amongst camels in this country. Apart from the experience of Gilchrist [1848] who states:—“That the camel is subject to a similar disease (rinderpest) has been satisfactorily determined and indeed it has prevailed to a considerable extent amongst the camels of the
Public Cattle establishment (in Madras) during the current year”; there are a few more reports of the occurrence of rinderpest in camels available in the evidence collected by the Indian Cattle Plagues Commission [1871]. Pease [1894] states:—“The camel also suffers very severely from the disease, it being very fatal in him; numerous instances of the disease in this animal have been cited by various authorities, and some have come under my own personal observation”. Recently Haji [1932] has described several natural outbreaks of the disease amongst camels, which were brought to his notice by Veterinary Assistant Surgeons working in parts of Sind and Karachi. The susceptibility of this animal to rinderpest was proved experimentally by Lingard and Cross, although Leese and Rayment stated that they never encountered any case in their experience with camels.

"Carriers" amongst cattle.

Cattle are known to be highly infective during the stage marked by acute fever and during early clinical symptoms. Such sick animals may spread the infection locally if not segregated, but the spread of the infection over long distances by such visibly sick animals is not likely as they would scarcely be used for transport purposes. There is danger, however, from animals which run through a symptomless form of the disease, as the following evidence from past and present workers would strongly suggest.

As far back as 1866, Thacker prepared a simple narrative on Cattle Diseases for the guidance of ryots of Madras, in which mention is made of the several ways in which rinderpest is propagated and carried about. Among other things, he states:—

"On one occasion, when sickness had broken out in a herd of cattle, I advised the owner to remove the healthy animals. One of these was sent about 15 miles away, and unfortunately put in a herd of 120, all in apparent health. Within a fortnight 70 were attacked with Murrain. The disease was thus communicated by this cow carrying in some way the infection about her, though she herself was never sick ".

This would appear to be the earliest recorded case of a “carrier” bovine and this valuable reference has been overlooked by all authors who have interested themselves with the problem of “Carriers”. To quote Lingard [1903]: “It is well known that an animal may in this country be attacked with so slight a form of rinderpest that it may even be overlooked by the initiated in this disease”. In Stockman’s [1905] words, “the disease smoulders on in a densely stocked location owing to the occurrence of mild cases amongst the partially resistant animals, which pass unobserved. These and the chronic bowel cases (infectivity up to 30
days) may start a fresh outbreak if they be brought in contact with a suitable fuel". Littlewood [1905] has observed that cattle imported into Egypt from Asia Minor may not show clinical symptoms and yet at autopsy reveal lesions of rinderpest. Rickman [1908] had the same experience in German South-West Africa, for he states that cattle and other animals may be infected to an imperceptible degree. In China, Eggebrecht [1910] found that some animals infected with rinderpest show no visible signs of the disease beyond a rise of temperature to 40° C. or higher for two days. In discussing "climatic influence" upon the incidence of disease in India, Baldrey [1912] refers to the fact that by long residence of any organisms of contagious animal disease in one place the disease becomes weakened in virulence to the animals of that place and further that animals infected with rinderpest may act as carriers without showing symptoms in enzootic areas. Again Pool and Doyle [1921] state:

"The animals that recover are turned out to graze as soon as they are convalescent. It is likely that most of them cease to be infective very soon after they are convalescent but a study of the epizootiology of rinderpest leads one to believe that there is a very strong possibility that some animals become "carriers" and remain so for a considerable period."

In the experience of Youngberg [1917], Nellore cattle are very dangerous as conveyors of the disease to the Philippine cattle, since the former may react without clinical symptoms and since his experimental work shows that the infectivity of the virus is not appreciably attenuated by being passaged through this breed of Indian cattle.

Jacotot [1929] showed that in an animal which is passing through a thoroughly blocked out and symptomless reaction, whether it be a very highly resistant animal (goat) that is inoculated with virus alone, or an animal (bull) which is inoculated with a sufficiently large amount of serum in the serum-simultaneous method of inoculation or a previously actively immunised animal (bull) which is being retested with virus alone, its blood can be shown to be virulent by inoculation into bulls on certain days during the period at which the blood of a virus producer is expected to be infective; but the longest period of infectivity does not seem to have been determined in these cases. Some support to the above view is obtainable from the observation of certain workers who found that the serum-simultaneous method of inoculation is not conducive to the eradication of rinderpest.

For instance, Ferrado [1917] was of opinion that the persistence of rinderpest in Eritrea was due to the so-called serum-simultaneous method of inoculation adopted by the authorities there in dealing with outbreaks. Claverie [1929] and Daubney
[1928-1930] have pointed out that serum-simultaneous inoculations create new centres of infection and that in countries, where this method has been resorted to, it has never been possible to eradicate rinderpest. The latter worker further added that the long duration of immunity conferred was due to the frequent recurrences of symptomless infections derived from centres of disease set up during the double inoculation. "There can be little doubt", he remarked, "that in animals guarded from all risks of reinfection immunity begins to decline at between 2-3 years after serum-simultaneous inoculation". The waning of immunity has been suggested also in a few cases of natural attacks. Evidence has been recorded by the Indian Cattle Plagues Commission from villagers and others to show that a bovine may be attacked more than once after a lapse of several years. This view is not un-supported, for Bliss [1922] furnishes trustworthy experimental proof to show that two animals, a cow and a heifer 7 months old, which recovered from a natural attack of typical rinderpest could be artificially infected by virus inoculation after three years and one and half years respectively and mentions that he has encountered other cases of second attacks during his experience in China. Animals having a second attack six years after the first and succumbing to it were met with by Pease [1894].

It is known that movement of cattle can be responsible for outbreaks of rinderpest. Angeloff [1917] held that the outbreaks of 1853, 1877 and 1913 in Bulgaria were all due to the movement of transport cattle from Asia. Illicit movement of cattle has been known to be responsible for the spread of rinderpest [McCall, 1922]. Very recently from a survey of the outbreaks of rinderpest which occurred in the Central Provinces in India Stirling [1932] has shown that they are confined to the routes followed by the nomadic tribes of cattle dealers. In all these instances, however, it is not possible to state definitely whether the cattle responsible for the introduction of the disease are the inapparently infected animals, such as those enumerated by Jacotot [1929], or carriers of rinderpest in a chronic form. As has already been pointed out, the danger from the visibly sick animals in these nomadic herds is relatively less and here the methods of control are likely to be more effective since such animals would not be physically fit to travel long distances and, further, their owners would not parade them about to the detriment of their own interests. It will be remembered that Great Britain was free from rinderpest for such a long period as 120 years, when in 1865, the disease was brought to the London Stock market, and at the same time to different provincial cities by a cattle boat from Finland and towards the end of that year 85 counties were infected. During the 1½ years of its prevalence 500,000 cattle died. The disease spread to Holland from England and destroyed a large number of cattle there also. In those days the steam ships were not so fast as now and the cattle boat must
have taken a period in excess of the maximum incubation period of the disease for reaching the English docks, and it is to be presumed that the cattle were in apparent health at the ports of embarkation and unloading. This historical outbreak seems to be the first recorded case of a large scale infection caused by what would appear to be "carriers".

Another interesting account is available of a severe outbreak being started by "carriers" in an Editorial article in the Rev. Gen. Med. Vet. [1920], in which the infection was introduced into Belgium through the agency of Indian zebus, presumably of the Hariana breed which is said to be highly resistant to rinderpest. These cattle were embarked at Bombay for Brazil and were jettisoned in the quarantine depots for a few days at Antwerp. Several of these died at Antwerp but rinderpest was not suspected. Three consignments of cattle arrived from America into the same port during the stay of the Indian zebus, and with the distribution of American cattle to different towns for slaughter many centres of cattle plague were established. This outbreak which was closely watched and studied by Bordet, Gratia, Lienaux, Nicholas and Binjard has brought out the following facts. The creation of new centres was ascribable in nearly every instance to the accidental or fraudulent movement of in-contact animals, and in a few other cases to the transportation of fresh meat. On the other hand, none of them could be attributed to the very indirect methods of contagion described in all the old classical references. Living intermediaries such as man or insusceptible animals played quite an insignificant or no part. Some exact observations showed that flies do not transport the virus. An efficient veterinary police with strict enforcement of stand-still orders sufficed to eradicate the disease completely. A point of further interest is the connected case, reported by Roberts [1921] as the first official record of rinderpest in Brazil. He considered that the same Indian zebus, which brought the disease into Belgium, were also responsible for its introduction into South America.

Now the cargo boat used for the transport of these cattle must have taken 4 or 5 weeks for the voyage from Bombay to Antwerp and a little longer for Rio de Janeiro at that time. Had the shipped cattle been highly susceptible, they would have almost certainly all died before the boat reached Antwerp, and the diagnosis of rinderpest would have been readily made. Here the Indian cattle were apparently healthy when they left Bombay and the virus was carried without the manifestation of clinical symptoms for a period well over the maximum incubative stage known (10 days).

In another recorded case [Delpi, 1930] calves were brought from a herd which had been attacked with rinderpest but the calves appeared to be perfectly healthy. These were transported by a canoe for 20 days to an island where the disease did
not exist. On arrival, some diarrhoea and lachrymation was noticed. Within the
next few days other animals began to show similar symptoms but these were not
severe enough to be considered as due to rinderpest. On the 32nd day after the
arrival of the calves, typical rinderpest broke out.

It seems that the neglected question of carriers among cattle was brought to
some prominence by the finding of Mrowka [1914] of lesions resembling those of
rinderpest in apparently healthy animals after they were slaughtered. His view
was that these lesions in the abomasum represented the residue of a pre-existent
disease in which the rinderpest virus might be retained even for years, and in
animals which, in the meantime, became devitalised, it might cause a reinfection in
the course of which the virus might be eliminated in great quantities. But it
seems that he was not able to infect other cattle by feeding the abomasum from
such cases.

Schern [1918] reported that out of 131 animals which recovered after inocula-
tion, with virulent blood, 34 animals had shown no symptoms after inoculation, but
when slaughtered at varying intervals showed lesions of rinderpest. In one case
lesions were present on slaughter 155 days after inoculation. He attempted to find
out if the abomasum from such sick animals was still infective. Animals fed on
the abomasum from an animal slaughtered 26 days after infection contracted
rinderpest, those at 70, 105 and 108 days did not, but those fed with the aboma-
sum from the 70th day animal were found to be subsequently immune when
exposed to infection by contact with diseased animals. Cattle fed on the aboma-
sum removed from an animal slaughtered 101 days after infection exhibited
mild doubtful symptoms. From this Schern concluded that cattle which had to
all outward appearances completely recovered from rinderpest may, nevertheless, be
possible carriers of infection, but that the danger of infectivity decreases as the
time after recovery progresses.

Bliss [1922] did not attempt to locate the seat of the virus in a “carrier”
animal but his views on the origin of “carriers” in this disease are worthy of
being quoted in extenso. He states: “since the virus is very short-lived outside
the body of the host, it is highly probable that the epidemics are started by
carriers that have received the infection in a former epidemic and have failed to
throw it off. Of this, up to the present time there is no direct proof, but it has
been observed that there are individuals which do not show symptoms within the
usual period after infection, but later when exposure to severe chill lowers their
vitality they develop the disease..............A degree of immunity, too weak to
destroy the micro-organism but sufficiently strong to hold it in check until condi-
tions favourable to its development occur, would produce a carrier. The number of
cases of rinderpest closely follows the variations in weather, the epidemics closely being connected with the inclement seasons of the year. Often a weakling in a herd starts an epidemic. It apparently takes a weak strain of the organism which the stronger cattle can resist and converts it into a virulent strain too strong for even the robust cattle."

Curasson [1926] was able to associate the presence of healed ulcers in the abomasum, patches of congestion in the intestine and moist and enlarged mesenteric lymphatic glands with animals which after recovery from rinderpest showed persistent poorness of condition, dull coat, frequent cough, intermittent fever, chronic enteritis and in females, chronic vaginitis and sterility. Further he was able to infect a susceptible calf with such abomasum in one out of two cases, the positive result being obtained with the material collected 34 days after apparent recovery and the negative result with that collected 7 weeks after recovery.

Several cases of the infectivity of materials other than abomasum from animals suffering from chronic rinderpest are on record. For instance, Hutyra and Marek [1916] showed that the blood of an animal chronically affected with rinderpest may remain virulent up to 30 days. Curasson [1926] showed that in animal which showed a second rise of temperature one month after recovery, the blood was infective by both subcutaneous and intravenous route. Delpi [1928, 1930] showed that in animals suffering from atypical rinderpest—a disease characterised by a prolonged duration of over one and a half month—the blood was infective through the entire period and at the time of death. Curasson [1922, 1926] gave two instances of the infectivity of the vaginal discharge from recovered animals one month after recovery. Jacotot [1932] proved that in one instance the vaginal discharge from an animal which had aborted 34 days after infection was infective, although its blood had ceased to be infective by them. Curasson [1926] showed that the faeces of a heifer which had recovered 7 weeks previously was infective though duodenal ulcers taken 3 days later were found to be non-infective. He stated that he had also known of a cow whose milk was found to be infective two months after recovery from rinderpest.

The available evidence on the existence of carriers has been seen. Reports purporting to show the truth of the contrary view have been recorded by several workers. In a reference quoted earlier, Ward and collaborators [1914] stated that they could not demonstrate the presence of carriers among recovered animals by the method of housing susceptible and recovered animals together. Rupert [1918], who stringently criticised Schern's work regarding the existence of carriers, pointed out that Mrowka himself had been unable to infect susceptible cattle with duodenal ulcers from his cases and recorded the result of his own work with Woefel in which he found that as much as three litres of blood from a recovered beast...
failed to reproduce the disease. Ourasson who had reported cases in support of the existence of carriers came across two instances of negative results, one with blood and the other with duodenal ulcers. Jacotot [1932] obtained consistently negative results with the exception of a notable case in which the vaginal discharge was infective. With reference to the negative results, Schern and Jacotot, both think, though with varying degrees of emphasis, that these are due to the fact that carriers are rarely encountered ordinarily.

In sifting the evidence that has been recorded on both sides of the question it is apparent that the two views are in reality not contradictory but they refer to facts appertaining to the different phases of the disease, for an animal may be a “carrier” of the virus in one tissue and not in another, as will be amplified later, or may have become entirely cleansed of the virus in one stage and not in another.

Our experience.

Having seen the present position with regard to certain connected aspects, our own case may now be considered. The subject of this case was a bull No. 288 of the Kumaun hills which died of chronic rinderpest 74 days after inoculation with virus, and whose spleen was proved to be infective at the time of death. It is believed that this is the first time that an attempt has been made to demonstrate a “carrier” through the infectivity of the spleen. The above case was encountered during routine post mortem examination and one was struck with the strange presence of the typical lesions of rinderpest so long after the initial infection with the virus. Some of the tissues were therefore collected with proper precautions and stored in sterile glassware (pipettes and petri-dishes) in the refrigerator with a view to test their infectivity.

It may be stated in parenthesis that the breed to which this animal belongs has been ascertained to be 18 times more susceptible than the Indian plains cattle [Lingard, 1899] and of about one-sixth the susceptibility of European cattle. It is known from experience that usually the mortality among these hill cattle varies from 50 to 80 per cent. when infected with a virus of bovine origin but it is much less when goat virus is used.

This bull was inoculated on 11th August 1931 as a routine virus producer with a goat strain which was being maintained at this Institute by passaging alternately through bulls and goats. The virus for inoculation into this animal was obtained on the 4th day after injection from a goat (No. 147) which had shown a rise of temperature of 106° on the third day after infection and died of rinderpest on the 15th day. To show that the virus used was potent it may be mentioned that another bull (No. 315), which was inoculated with blood from the same goat at the same
time, developed typical clinical symptoms of rinderpest (inappetence, diarrhoea and mouth lesions) but eventually recovered.

The Bull No. 288, which is now under consideration, showed a marked rise of temperature of 104·6° F. on the third day after inoculation, maintained the temperature at about this level up to the sixth day and showed a gradual fall by degrees, till the temperature came to normal by the 9th day. The animal suffered from slight inappetence and digestive disturbances (diarrhoea and soft faeces) lasting for 4 days from the 6th to the 10th day after inoculation. No vesicles or ulcers characteristic of rinderpest were detected during the period of observation extending for 15 days [Chart No. 1]. The only complicating factor, apart from rinderpest, was the

**CLINICAL CHART No. 1.**

<table>
<thead>
<tr>
<th>DATE</th>
<th>OCT. 29</th>
<th>30</th>
<th>31</th>
<th>NOVEMBER 1931</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAY</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>11</td>
<td>12</td>
<td>13</td>
<td>14</td>
</tr>
</tbody>
</table>

**CLINICAL CHART No. 2.**

<table>
<thead>
<tr>
<th>DATE</th>
<th>OCT. 29</th>
<th>30</th>
<th>31</th>
<th>NOVEMBER 1931</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAY</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>11</td>
<td>12</td>
<td>13</td>
<td>14</td>
</tr>
</tbody>
</table>
presence in blood smears of a few Theileria parasites on the 4th day after inoculation. The animal was discontinued from the experiment on the 16th day after inoculation and sent away to a tin shed a few furlongs away from the main rinderpest station since this tin shed is set aside for housing all the animals that have passed through rinderpest pending eventual disposal. During its sojourn in the tin shed as a discontinued animal, whatever minor symptoms of malaise that it exhibited did not receive any serious attention. The bull died on the night of 24th October 1931, i.e., 74 days after the infective inoculation with the rinderpest virus. An autopsy was held on it on the morning of 25th October 1931. The lesions detected at post mortem examination were definitely suggestive of rinderpest and included marks of diarrhoea, rare ulcers on the lip, ulceration of the pharynx, oesophagus, gall bladder, caecum and rectum. The ulceration in the last two viscera was deep and necrotic. The abomasum was diffusely congested and oedematous, associated with cicatrices of healed ulcers in the fundus and ulceration with streaks of deeper congestion in the pylorus. Rectal smears did not reveal the presence of any coccidia. Blood smears taken just after death revealed a few Theileria parasites only.

EXPERIMENTAL TESTS FOR INFECTIVITY.

Of the materials collected from this animal and stored in the refrigerator, only the spleen and blood were tested by inoculating saline emulsions of the blood clots and the spleen separately into hill bulls Nos. 788 and 768, and 791 and 688 respectively.

The inoculations were carried out on 29th October 1931, i.e., 4½ days after the death of the animal, and each lot of two animals was kept in separate isolation choppers. Hill bull No. 788, which was inoculated with 1 c.c. of saline emulsion of the clot of heart blood, did not show any disturbance of health excepting for a very transient rise of temperature of 104° F. on the afternoon of the 9th day after inoculation. Blood smears taken at the time of the elevation of temperature did not reveal any protozoon organisms. On being retested for immunity with 5 c.c. of virulent blood of bovine origin on 28th November 1931, i.e., one month after the test inoculation, the animal showed a typical thermal reaction and clinical symptoms of rinderpest, such as inappetence, diarrhoea and vesicles [chart No. 5]. The bull died on 18th December 1931, 20 days after the retest inoculation as a result of rinderpest complicated with coccidiosis. The second animal, Hill bull No. 768, which was also inoculated with saline emulsions of blood, did not show any reaction. On being submitted to a retest with 5 c.c. of the bovine virus one month later, it showed a typical thermal reaction and clinical symptoms
APPETITE
FEEDS WELL
OTHER SYMPTOMS
DIARRHOEA
FEEDS SLOWLY
DULL
CO
W
A
FEW T.MUTAN QL
FAIRLY FEEDS WELL
FEEDS WELL
FAIRLY FEEDS WELL
DISCONTINUED
RETREATED
DISCONTINUED
Hill Bull 688.
CLINICAL CHART No. 4.

DATE
OCT. 31 29 30 31 1 2 3 4 5 6 7 8 9 10 11 NOVEMBER 1931 1 2 3 4 5 6 7 8 9 10 11 12
DECEMBER 1931 1 2 3 4 5 6 7 8 9 10 11 12 13
DAY. 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
characteristic of rinderpest, dying 25 days after the retest inoculation on account of pleuropneumonia and resuscitated piroplasmosis [chart No. 4].

Of the two animals which were inoculated with splenic pulp, Hill bull No. 791 showed a characteristic thermal reaction starting from the 4th day, inappetence, diarrhoea and mouth lesions and died on the 12th day after inoculation [chart No. 2]. In the *post mortem* examination, typical ulcerative lesions of rinderpest were found in the pharynx, abomasum and the intestines in association with the presence of coccidia in rectal smears. The other bull No. 688, also inoculated with splenic pulp on the same day as the above, developed a characteristic thermal reaction and from the 5th day showed inappetence, diarrhoea and mouth lesions. It suffered from resuscitated coccidiosis for some time but eventually recovered. It was retested with 5 c.c. of virulent rinderpest blood (which was proved to be virulent by inoculation simultaneously into other susceptible bulls), one month and 20 days after the test inoculation but was found to be refractory to rinderpest [chart No. 3].

**DISCUSSION.**

It is seen from the above inoculation experiments that the spleen, collected from an animal, dying of chronic rinderpest 74 days after the initial inoculation was infective by inoculation into susceptible bulls, while the blood was not. The persistence of rinderpest lesions in the alimentary tract with accompanying infectivity of the spleen over such a long period is remarkable. From the data given above, there seems to be no doubt that the lesions exhibited at *post mortem* examination were due to the infection contracted at the original inoculation, there being no room for an accidental second infection. The possibility of rinderpest lesions persisting or reappearing after a long interval, as seen in this case, would seem to receive strong support from the observation of Delpi [1928] who found that in a typical case of bovine rinderpest, mouth lesions may be detected on the 7th day and again at the time of death, i.e., beyond 1½ months after the date of infective inoculation. Our case is to a degree comparable with Delpi’s cases since the virus in both cases were from attenuated strains: the former being due to passage through goats, and the latter being due to accidental degradation during routine passage through cattle.

As has been pointed out in some detail in the Introduction, the virus of rinderpest is exceptionally fragile and cannot be transported through air and water, nor can it survive in the soil for any length of time. Vectors such as mosquitos, lice, ticks or flies have been shown to be incapable of transmitting it under natural conditions. Although outbreaks occur among wild animals, they cannot be
incriminated as natural carriers of the disease, like jackals in canine piroplasmosis or certain wild ruminants in Nagana.

Hutyra and Marek [1926] have quoted the following authors for information bearing on the actual mode of contagion: (a) Ranpach has shown that, out of doors, the spread of the disease may be prevented by digging a trench around the infected herd which the animals are unable to pass and that in such cases cattle on the other side of the trench may remain healthy; (b) Piot Bey found that by isolating affected cattle for a distance of 10 metres the spread of the disease to other animals could be prevented; (c) Nencki has shown that in the stable healthy cattle may be protected from the neighbouring infected animals by simple board partition. Very recently Cooper [1932] at this Institute has shown that the introduction of a healthy bull into a stall just vacated by an animal dead of acute rinderpest and only separated from other infected animals by mere board partitions may not result in infection (period of observation was 15 days). Again the experience at Muktesar, where susceptible and infected animals have been maintained since the very inception of this Institute about 40 years ago, only a few hundred feet apart on either side of a ridge, is that there has never been any instance of the disease spreading over to the other side. No steps whatever are taken to prevent the movement of men and traffic except for the infected cattle themselves. In his experiments to obtain some exact information regarding the infectivity of rinderpest by intimate contact, Cooper has found that a contact of 2 days with animals during the early febrile stage of the disease is not sufficient for infection and that the most contagious period in the rinderpest syndrome is between the 5th and 10th days after artificial inoculation. The infected animals are so sick in the abovementioned most contagious period that it is not likely that they would move about.

The known facts about the epizootiology of rinderpest can therefore be explained only by the view that carriers of rinderpest exist among cattle and perhaps also among other domesticated ruminants which move among cattle and that outbreaks are started by prolonged contact of susceptible animals with a carrier in the infective stage but under suitable conditions. However, when an attempt is made to obtain from published literature, experimental confirmation of this mode of spread of the disease both the sides of the question appear to be equally well represented. For, it has already been seen that Ward and collaborators [1914], Boynton [1918], Ruppert and Wolfel [1921] and Jacotot [1932] (except in a solitary instance) have all failed to prove the existence of carriers; and that on the other hand, Schern [1918], Curasson [1920, 1926] and Jacotot [1932] have brought definite findings which leave no room for any other conclusion than that carriers do exist. The
former results are explained by the latter school of thought by saying that carriers are not at all frequent and that Ward, Rupert and others were not fortunate enough to encounter them. Our contention is that carriers are not, perhaps, as rare as supposed although it is not easy to detect them in the ordinary way. For it seems more than probable that the virus is kept well under control in some remote ductless gland or organ, the virus being not detectable in the usual discharges and excretions until such time as unfavourable conditions of life supervene and upset the controlling mechanism. One is struck by the remarkable similarity in the circumstances under which the reported outbreaks in England [1865], Belgium [1920], Brazil [1921], Romblon [1916], Australia [1925] originated. An invariable association of a trying voyage on sea for varying periods (exceeding 10 days) with each one of these cases suggests that this association was certainly more than a mere coincidence. This suggestion is considerably strengthened since evidence is available to show that unfavourable conditions of life have an important bearing on the starting of outbreaks. Apart from the statement of Bliss already quoted in extenso, the Indian Cattle Plague Commission observed that rinderpest rages "on seasons of exceptional insalubrity—a drought, a flood or a cyclone—producing such wholesale havoc and death that agricultural industry was for the time paralysed". Further, McLeod [1869] quotes a letter from the Consul General of Bessarabia, addressed to the Earl of Clarendon describing the incidence of rinderpest in that country where it is permanently prevalent, and endorses the common belief of principal landowners and cattle breeders to the effect that the origin of the disease is attributable to "long journeys during great heat of summer across arid steppes where no pastures nor wholesome water can be found".

With reference to the question as to which remote gland or organ is best suited for the greatest longevity of the rinderpest virus the spleen, which has been shown in our reported case to be virulent even after 74 days of infection, naturally suggests itself first. There seems to be a consensus of scientific opinion on the superiority of the spleen as the most serviceable organ for the preparation of vaccines. Lingard [1906] found that spleen pulp dried for 3, 8, and 59 days retained its virulence. In the experience of Kakizaki and collaborators [1926] a vaccine prepared from lymph glands was inferior to that prepared from spleen pulp. Keeler [1928] found that the blood entering into the preparation of the vaccine besides having no immunising properties detracts materially from the keeping qualities of the vaccine. Danbney [1929] states that lymph glands and kidneys are of lower value than spleen in the formalised vaccine and blood and liver are valueless. Jacotot [1931] found that the spleen pulp was virulent for a longer period (7 months) than blood (2 months) when kept under the same condition at
0 C. Although the abomasum was found to be richest in the virus [Jacotot, 1932] in an animal at the acute stage of the disease, it does not seem to have been used for the preparation of vaccine. In certain unpublished experiments, which one of us (V. R. R.) undertook under the direction of the late Director of this Institute (Dr. Edwards) it was found that chloroform in the concentration of 0.75 per cent. killed rinderpest virus in blood within 2 hours at body temperature. In similar experiments Daubney [1929] found that the virus in blood is readily killed by chloroform and that such killed virus has no antigenic powers. The superiority of the spleen as vaccine material has been referred to. The retention of immunising properties in the spleen can be shown to be due not to any preservative added to the tissues but to be inherent in them, for it is known that glycerine, phenol and other antiseptic substances even in minute quantities cause rapid deterioration of the virus (Koch, Semmer, Theiler, etc.). Further Edwards [1924] proved that the virus of rinderpest is not destroyed but only inactivated by treatment with 0.5 per cent. carbolic acid, since intensive passage through rabbits brought back its virulence for cattle. An idea of the degree of the tenacity of the virus in the spleen can be obtained from the fact that it has been found by more than one worker [Kelser, 1928, Cooper and Menon, 1931] that the spleen retains its vaccinating properties for about 1 year. This must have been due to the survival of the virus in the spleen pulp in spite of treatment with chloroform. In fact it has been suggested that the vaccinating properties are due to the survival of the virus in minimal quantities. Curasson and Delpi [1929] do not know whether the maintenance of antigenic powers over long periods is due to the spleen being specially rich in the virus or because it has some special action but they are certain that other virulent materials such as blood or peritoneal washings when treated with formalin are uncertain in their immunising powers. It is interesting to note that Jacotot [1929] has claimed that antiserum prepared against the organ pulp vaccines as antigen for hyperimmunisation was superior to that prepared against virulent blood.

As a result of the studies on our case of chronic rinderpest, it is suggested that the spleen is one of the seats of the virus in a carrier animal, a view which is considerably supported by the available information on the persistence of the virus for long periods in the splenic tissue both under experimental conditions and in the preparation of vaccines and hyperimmune antiserum. It is believed that in Kelser’s vaccine the spleen cells protect the architecture of the virus molecules from the destructive action of chloroform and that the superiority of Jacotot’s hyperimmune antiserum is due to the spleen cells constantly releasing small quantities of the virus and maintaining a sustained stimulation on the host’s body.
mechanism for the preparation of efficient antibodies. Further, the employment of a tissue culture of the spleen with a view to cultivate the rinderpest virus in vitro would appear to be indicated.

REFERENCES.

Galambos, (1861). Gyogysazat cited by Hutrya and Marek (1926), Spec. Path. and Therap. of diseases of domestic animals.
Gartner, (1920). Berliner Tier. Wuch., 43 and 44.


--- (1926). *Spec. Path. and Therap. of the diseases of domestic animals; 1, 299-300.*


Lingard, A. (1899-1903). *Note on the different degrees of susceptibility of bovines, buffalose, &c.*


Pease, H. T. (1894). *C. V. D. Ledger Series No. 1—Rinderpest,* p. 3 and also pp. 30-31.


UNUSUAL CASE OF CHRONIC RINDERPEST.

Robertson, W. A. N. (1925). Rinderpest in Western Australia in 1923.
Deuts. Tier. Woch., 1898, 6, 205.
THE

VETERINARY

JOURNAL:

A MONTHLY REVIEW OF VETERINARY SCIENCE

Est. 1875

EDITOR:
Emeritus Prof. SIR FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E.

DR. MED. VET. (h.c.) ZURICH.
HONORARY VETERINARY SURGEON TO HIS MAJESTY THE KING.
OFFICIER DU MÉRITE AGRICOLE (FRANCE),
CAVALIERE DEI SS. MAURIZIO E LAZZARO (ITALY).
HONORARY MEMBER OF THE AMERICAN VETERINARY ASSOCIATION.

SUB-EDITOR:
GLADSTONE MAYALL, M.R.C.V.S.

with the collaboration of
PROF. T. DALLING, M.A., M.R.C.V.S.
RICHARD HUDSON, F.R.C.V.S.
PROF. J. McCUNN, M.R.C.V.S., M.R.C.S., L.R.C.P.
COL. SIR ARTHUR OLVER, C.B., C.M.G., F.R.C.V.S.

PRICE: Single Copies 2s. (2s. 2d. post free); Annual Subscription 21s.

LONDON: BAILLIÈRE, TINDALL & COX
(EST. 1826)
7 & 8, HENRIETTA STREET, COVENT GARDEN, W.C.2
'PARSETIC'  
A Dependable Local Anæsthetic Solution for Epidural Anæsthesia

'PARSETIC' is a solution of ethocaine (procaine) hydrochloride suitable for the production of local anaesthesia by subcutaneous injection prior to minor surgical procedure. This solution contains sufficient Adrenalin to localize the anæsthetic and to restrict bleeding at the site of operation.

Injection of 'Parsetic' into the epidural space at the lower part of the vertebral canal for the production of anaesthesia has been extensively employed in veterinary practice during recent years for a variety of operations, including those involving the ovary, uterus and udder.

'Parsetic' contains 2.25% of ethocaine with one part of Adrenalin in 30,000 parts of solution, and is issued in rubber-capped bottles of 2 fluid ounces and in ampoules of 1 c.c. Further particulars will be furnished on request.

PARKE, DAVIS & CO.  
50 BEAK STREET, LONDON, W.1  

When corresponding with Advertisers kindly mention THE VETERINARY JOURNAL
THE VETERINARY JOURNAL

Editor:
EMERITUS Prof. SIR FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E., Dr. Med. Vet. (h.c.), Zurich. Hon. Vety. Surgeon to His Majesty the King, Emeritus Professor of Surgery in the Royal Veterinary College

Sub-Editor:
GLADSTONE MAYALL, M.R.C.V.S.

in collaboration with

PROFESSOR T. DALLING
Professor of Animal Pathology and Director of The Institute of Animal Pathology, Cambridge University

RICHARD HUDSON
Retford

PROFESSOR J. McCUNN
Professor of Anatomy, The Royal Veterinary College

COLONEL SIR ARTHUR OLVER
Principal of the Royal (Dick) Veterinary College, Edinburgh

LIEUTENANT-COLONEL T. DUNLOP YOUNG
Veterinary Adviser to various Overseas Governments; late Chief Veterinary Officer to the Corporation of the City of London

JUNE, 1939

Editorial

LICHEN TROPICUS

The article dealing with so-called Lichen Tropicus which appears in this number draws attention to a group of helminthic diseases which, particularly in tropical and sub-tropical countries, are in the aggregate of very considerable economic importance and cause a great deal of discomfort to domesticated animals and inconvenience to their owners.

Usually not fatal and confined to the integumentary tissues, the conditions seen in affected animals are the result of a deep-seated infestation by nematode parasites, the larvae of which, under favourable conditions of temperature and humidity, develop in such numbers near the surface of various parts of the body as to cause extensive lesions of the skin and subcutaneous tissues; for example, the tumour-like ulcerating lesions of bursati, more superficial ulceration of the skin and subjacent tissues such as are seen in “hump sores” of cattle and the ulcerated conditions of the extremities which are prevalent in particular areas where humidity is high or the animals stand a great deal
in water; or dry, scaly lesions of large areas of the surface of the body such as are discussed in this article.

Itchiness, seasonal periodicity, and a marked tendency to recur in the same animals are common features of these conditions, and though they do not as a rule lead to early death or destruction, the discomfort, disfigurement and disability caused by them is of sufficient importance to warrant more attention than has hitherto been given to the study of the life histories of the parasites concerned, and the discovery of more satisfactory methods of treating or controlling these very troublesome conditions.

The causative effect of the larvæ seems to be established, but no satisfactory method of medical or surgical treatment for the majority of these conditions has so far been discovered, and though their external manifestations tend to disappear each cool season they tend to become more severe with each recurrence.

From the very large number of synonyms which are employed to describe them it is clear that they attract a great deal of attention, and it is much to be desired that an exhaustive study should be made of these very disfiguring and disabling conditions.
MICROFILARIAL PITYRIASIS IN EQUINES

(Lichen tropicus)

By CAPTAIN S. DATTA, B.Sc., M.R.C.V.S., D.T.V.M.(Edin.)
Veterinary Research Officer, Imperial Veterinary Research Institute,
Mukteswarkumaun, U.P., India.

In recent times considerable progress has been made in the study of
diseases involving the human skin, particularly of the troublesome conditions
peculiar to the tropics. Dermatology in the veterinary field, on the other hand,
has remained a somewhat neglected subject and the pathology of quite a
number of animal diseases is still enshrouded in mystery. Considerations of
economic importance may be partly responsible for this state of affairs, but
the role of the skin as a balancer of internal functions under varied environ-
mental conditions is as yet only imperfectly understood. It is undoubtedly that
morbidities of the cutaneous covering of animals do sometimes become very
troublesome and interfere materially with normal animal husbandry. That
the state of confusion is more serious than is usually believed will be clearly
seen from a discussion of Lichen tropicus in this article.

The disease has been known in India for at least a century under the local
names of Khoojlee and Kharish, meaning any skin disease characterised by
scratching. It affects both horses and mules. Though the etiology
remained obscure, the clinical picture of the disease was so precise,
even to the lay mind, and its occurrence so frequent that such general terms
as the “Indian skin disease of horses,” “the usual,” “familiar” or “common
skin disease of India,” came to be employed in veterinary literature. The
disease has received scientific attention since the first studies of Grellier in
1843 and Queripel in 1879. It is a condition which does not respond to
treatment but disappears mysteriously with the approach of winter, to reappear
the following summer in the same erratic manner in special subjects to the
exclusion of their neighbours. As such it has exercised the resources of
veterinarians a great deal and numerous articles exist on the subject. For our
purpose in this article it will suffice to recount the various names that have
been employed by different investigators, firstly, to understand how different
aspects of the same disease entity have impressed different observers, and,
secondly, in order that in places where similar terms are employed for designating obscure conditions, the data regarding Lichen tropicus recorded below may help in effecting elucidation. If one consults a standard veterinary treatise, such as Hutyra and Marek’s Special Pathology and Therapeutics, one finds descriptions of conditions of unknown etiology under some of the names already used in India for Lichen tropicus, but presumably used in ignorance of the older Indian literature.

Lichen tropicus has been designated: Mange or itch (Grellier), eczema papulosum, prurigo (Queripel), psoriasis, “mane and tail disease” (Burke), impetigo contagiosa equi, summer mange, Indian ringworm (Edgar), Dhobie’s itch, Tinea furfuracea (Haslam), seborrhoea sicca, pityriasis tropicalis, pityriasis sicca, dandruff, pricky heat (Meyrick), seborrhoea sicca, cutaneous umbilicated crusts (Gunn, Lingard), irritable summer skin disease (Nesfield, 1907), and protein rash (Allen and Kingston, 1928). Besides the above multiplicity of names, Fleming confused the identity of this disease with summer sore as described by Rivolta, and du Toit in Mense’s celebrated book on tropical diseases stated that cutaneous filariasis was included along with sporotrichosis and habronemiasis under the term Bursati in India. The disease has been likened to prickly heat and Dhobie’s itch (Haslam) of human pathology, and the name Lichen tropicus has been used in recent publications as synonymous with prickly heat.

**Geographical Distribution**

The disease has been recorded from most provinces in India excepting some parts in the south, but the incidence is greater in some of the northern towns. The disease does not appear to be restricted to the Indian sub-continent; genuine cases have been encountered in the Sudan, Philippine Islands (Underwood, 1934), and perhaps in U.S.A. (Alicata, 1936). A similar condition has been reported from French West Africa (Aldigé), and from Greece (Papadaniel, 1936). Further, it is known that “bran and flour eczema” (pityriasis) occurs in Germany, and “Queensland mange” in Australia. There are strong reasons for believing that Lichen tropicus is even more widespread than the above records indicate.

**Seasonal Incidence**

The disease usually manifests itself in the early part of each hot weather, starting from March or April, and continues unchecked till the onset of the cold season in September or even much later. The skin of an affected animal may therefore remain free from any obvious disease for as many as four or five months in a year, but in well-established and long-standing cases the period of freedom may be almost obliterated. If an affected animal is transferred to the hills in the summer the disease retrogresses and may not reappear till the animal is returned to the plains.
Lichentropicus—Photo showing the predisposition sites of the disease.
Clinical Symptoms

In the initial stages attention may be drawn to irregular patches of hair standing out abnormally, and on passing the hand over the affected areas, slight papular swellings of low elevation and up to the size of a sixpenny coin are detected. These to some extent resemble urticarial wheals, and may lead one to suspect bites of insects or mosquitoes. On parting the hair over the affected region a flattened and a somewhat burnished appearance is seen. If the surface epithelium only is excised and pressure applied, a minute quantity of clear serous fluid oozes out. The papular lesions are generally discrete and diffused throughout the superficial skin in the predisposition sites. The sides of the neck, the regions of the crest, shoulder, root of the tail, undersurface of the neck and abdomen are the sites affected. Neighbouring papules may become confluent. The intensity of the lesions, and the extent of the affected areas vary in different cases, and would appear to depend upon the interval that has elapsed since the first attack of the disease and the facilities for rubbing to which the animal has had access.

The hair over the affected region is peculiar in that it becomes unduly coarse, erect, crinkled and fragile, and some are grey. As the disease progresses, gradual shedding and breaking down of long hairs take place along with an increasing thinness of hair in irregular patches in various parts of the body. The depilation is readily noticeable in the region of the mane and the tail, and hence the name "mane and tail disease."

The most characteristic of all the symptoms is perhaps the copious and regular exfoliation of minute, white epithelial scales around each papule. Initially the scales are transparent, but with the exudation of serum or other fluids later the scales conglomerate into opaque masses. The amount of the serous exudation, which works its way to the surface, is so small that even after absorption by the cuticular and subjacent layers the scales appear dry. This explains why the word sicca has been used by earlier workers to qualify the designations—pityriasis, eczema or steatorrhoea.

The disease is further characterised by excessive irritation and itching, as a result of which the affected animal rubs violently and produces superficial abrasions or actual inflammation. The indications are that with the onset of the disease, the skin is rendered tender and predisposed to abrasion. The character of the inflammatory lesions depends upon the nature and duration of the injury and upon whether secondary complications supervene. The primary features may thus be masked, and the secondary changes become more prominent, leading to some difficulty in obtaining a clear understanding of the essential lesions. Prior to the breach of cuticular surface taking place, there may be only a minor degree of oedema in the texture of the skin, but as the surface becomes abraded there may be a continuous flow of moist exudation, including blood, accentuated by rubbing, thus fulfilling the requirements of the clinical picture of moist eczema.
One easily noticeable and differential feature of this disease is the distinctly superficial character of the lesion, i.e., without the involvement to any extent of the deep structures of the skin, as opposed to the more or less sharply defined tumours in cases of cutaneous granuloma of the type of Bursati or summer sore.

The general health of an affected subject is not seriously interfered with, though in some cases lack of rest, due to prolonged irritation, may produce considerable loss of bodily condition. A stage of pyrexia has not been observed, but a slight rise in the superficial temperature seems possible. In an affected subject, recurrence of lesions in successive years is the rule, and from their history one can safely indicate the horses in a battery or regiment that will develop symptoms during the approaching hot weather. All breeds of horses may develop the disease, but as a rule imported Australians appear to be highly predisposed. The disease is not contagious and syces and grooms attending clinical cases have never been observed to contract the disease. Although the eruptions of Lichen tropicus are at first summer troubles only, predisposition seems to increase in the course of time. The subacutely inflamed skin becomes the seat of some permanent induration and sclerosis due to excoriation, which allows the disease to be more readily established in an affected subject than in earlier years. After a time the affected skin becomes thickened and greatly corrugated, particularly in the neck, crest and tail. The folds are disposed perpendicularly to the crest of the withers. When extensive depilation has taken place the tail presents a burnished wrinkled appearance, and the term "rat's tail" has been used to describe this appearance.

**Etiology and Pathological Histology**

The great variety of names under which the disease has been dealt with by various investigators has already been noticed. Different features of the disease have been emphasised to varying extents. Among the widely divergent views regarding its etiology, only the more common ones will be referred to here.

It has been ascribed to constitutional causes due to diet, climate, non-hygienic conditions, renal, hepatic or splenic disorder. It has been considered "as a cutaneous furor to which horses pampered and little worked are liable." The fiery heat of the plains of India has been incriminated as the exciting cause. Again, it has been ascribed to diet rather than to the climate, being attributable to long-continued and inordinate use of green fodder. The finding of various fungal organisms in the epidermal scales and scurf has been recorded by several authors, but the disease could not be set up experimentally, and yet certain authorities have gone so far as to consider that the disease is a form of ringworm—Dhobie's itch. Others showed how it differed in its course, pathological results and clinical aspects from ringworm infections.
LICHENTROPICUS IN HORSES

To face page 216.
Tryon in Australia and Nesfield in India described sporiform bodies, and the latter admitted that they were often found on an apparently healthy surface and might possibly be nothing more than secondary infection on an eczematous surface, though personally he was not of that opinion. Gunn (1884) examined scabs from clinical cases after they had been variously treated with water, glycerine, potash, etc., and claimed to have discovered an animal parasite, resembling a tænia, and possessing a truncate caudal extremity and twenty cephalic processes or borers. Gunn's description of the parasite has been dismisssed by Neumann and Lingard as vague. Skin scrapings have been examined by several workers (including Shaw, 1932, and the present writer), but no worms have ever been detected.

Queripel's view was that the fiery heat of the plains was the exciting cause, and Burke found that the hottest stations were those in which the largest number of cases occurred. The theory that the disease is essentially an allergic disease, being probably mainly brought about by the absorption of unchanged proteins from the ingested food material or by toxic bases resulting from the special types of organisms inhabiting the alimentary tract was advanced by Allen and Kingston in 1928. Shaw in 1932 enumerated all the suggested factors and argued very sensibly: "If any of these are really the cause, it should surely follow that where the rations, the amount of exercise and grooming are the same, all the animals of the unit ought to be affected. But, as this is not so, there must be other causes not yet fathomed. There undoubtedly is individual idiosyncrasy, as evidenced by the appearance of the disease in the same animals year after year, as well as colour susceptibility, the disease being met with chiefly among blacks and browns." More recently Holness (1933) states, "the causal and predisposing factors of Lichen tropicus are difficult, if not impossible of demonstration."

A perusal of the existing voluminous literature on the disease shows that no serious attempt has been made to study its histo-pathology. This lack of information can perhaps be explained on histological grounds alone, because the structural changes must appear to be only a comparatively slight response, compared with what one would expect to find in a disease-entity which has become so well established in the animal's system as the present one with its intractability to treatment and recurrence in the same subject year after year. The microscopic features of this disease are, however, quite definite and precise, though the tissue reaction exhibited may appear unusual, in view of the more marked lesions in other diseases.

In the Annual Reports of the Imperial Veterinary Research Institute, Mukteswar, for the years 1931-32 (p. 19) and 1932-33 (p. 16), comments were made on the remarkable resemblance of the histological features of the lesions of the equine disease to those of Craw-Craw, a form of microfilarial itch of man of the West Coast of Africa, a fact which was suggestive of a similar
etiology. This expectation was fully confirmed before the 21st session of the Indian Science Congress, held in Bombay in January, 1934 (Datta, 1934). Pieces of the skin were received from 20 cases, preserved in normal saline and also in formalin, together with skin scrapings and blood smears made during the day and night. The blood smears and skin scrapings were invariably negative for microfilaria, but in haematoxylin-eosin stained sections of the skin constant association of microfilaria with an eosinophile infiltration was seen in all cases. Thin sections had to be cut, completely dehydrated before mounting, and with experience the microfilaria could be detected with a 3/4 objective and No. 5 ocular, the suspicions being finally confirmed with the 4/4 objective.

The main centre of the activities of the worm appear to be in the upper layer of the corium and in the papillary body, including the superficial cuticular layer of the epidermis and the hair and hair follicles. In the earliest stage, when no alterations or degenerations of the epidermal scales are manifest, there exists a definite dilatation and engorgement of the blood vessels immediately subjacent to the surface epithelium. Varying degrees of serous effusion with a sprinkling of leucocytes may be seen with the tissue and lymph spaces dilated. Around the blood vessel supplying the papillary body and in the dilated lymphatics, a marked cellular reaction is present and numbers of wriggling microfilaria may be seen. The most noteworthy infiltrating cells are the eosinophiles, the density and location of which, on closer examination, is helpful in the finding of the larvæ. The cellular reaction appears to be more marked up to that depth of the skin to which the hair follicles and the tubules of the cutaneous gland extend. In the active stages the cellular reaction extends considerably, and if the tissue has been chosen carefully the parasites are found in large number. Sometimes fresh pieces of skin may be placed on removal in normal saline and incubated, when the parasites congregate together in the papillary body or migrate into the saline solution. In the case of the partially quiescent lesions, collected late in the season, the finding of rare microfilaria requires considerable perseverance and care. Experience shows that in such cases a sufficient number of sections must be examined to obtain a positive diagnosis. The production of active lesions is connected with the richness of the parasitic infestation. Cuticular cells, some detached, others semi-detached, are seen, indicative of the process of exfoliation. The epidermis presents umbilicated depressions packed with cuticular cells. The hair and its follicles appear shrivelled, uneven, the cortical scales of the hairs detached or broken, the medulla of the hair constricted or discontinuous. In old-standing lesions considerable fibrosis, thickening and the formation of rugæ take place, and the papillary bodies appear greatly compressed from side to side. Blood vessels show degrees of endarteritis obliterans. The stratum germinativum shows proliferation of cells. The lesions are suggestive of the existence of a potent filarial toxin.
The Parasite

Several methods of examination have been employed, and repeated searches for adult worms made in specimens of fresh and preserved skin from the affected regions. With the exception of the nuchal ligaments (region of the mane) of several cases, which revealed the presence of *Onchocerca cervicalis*, a species not previously recorded in India, no adults have been detected. The finding is certainly suggestive, but difficulties exist in accepting *O. cervicalis* as the parents of the microfilaria concerned. The detailed morphology of the larvae has been examined by helminthologists at Mukteswar, and one of them has recorded certain particulars (Srivastava, 1938), but as the existing information on the morphology of the larvae of filarial worms is meagre and the life-history of the majority of Onchocercoid worms unknown, specific determination of the microfilaria must await a future date. It is important, however, to record as many details as possible of the microfilaria:

The photomicrographs illustrating this article show how the microfilaria are situated in the papillary body and how in sections they lie irregularly in serpentine forms and graceful coils.

The microfilaria is not provided with any sheath. The average length is 185 microns, and the maximum thickness attained slightly posterior to the cephalic extremity is 3.5 microns. The head end is bluntly rounded and does not bear any armature. The cephalic spot, which is a clear area, extends for a distance of 3.5 microns from the anterior end. Immediately behind the spot there are two large oval cephalic cells, placed one behind the other. The nerve ring is situated obliquely at a distance of 43 microns from the anterior extremity. It is 3 microns in breadth. The excretory pore is situated at a distance of 3 microns, posterior to the nerve ring, and the excretory cell, which is irregularly oval, is situated 14 microns from it. There are four chief genital cells, the last two of which are situated at a distance of 17 and 18 microns from the tip of the tail. The second genital cell is 10 microns anterior to the third one, and the first one is situated 14 microns in front of the third. All the genital cells excepting the first are round. The first genital cell is oval and has a large granular nucleus. The chief characteristic of the microfilaria of *Lichen tropicus* appears to be the presence of two large cephalic cells, according to Bhalerao, and this may be of some value in specific differentiation. It is certain that this microfilaria has no structural resemblance to the so-called tænia described by Gunn, and the possibility of one being the developmental stage of the other is extremely remote.

Recently particulars regarding forms of skin disease of microfilarial origin, bearing a close resemblance to *Lichen tropicus* of India or identical with it, have been recorded from countries outside the Indian sub-continent, but the morphological details of the microfilaria are so brief as to render futile any attempt at comparison of the parasites, as described from the different
countries. Underwood's (1934) material (56 cases) relates to Philippine Islands and Texas, Alicata (1936) deals with two cases from America, and Papadaniel with material from Greece. Underwood calls the disease "equine dhobie itch," and it may be of interest to ascertain how this old and purely Indian name comes to be employed there.

It is important to note that as in India so also in these countries, the microfilaria of the skin disease are suspected to be the larva of Onchocerca cervicalis, which occurs in the ligamentum nuchae of equines. O. cervicalis occurs in many countries, associated sometimes with cases of poll evil and fistulous withers, and sometimes in apparently healthy horses. How far are these facts in harmony with each other?

As far as India is concerned, poll evil and fistulous withers appear to be very rare and so far O. cervicalis has not been detected in any horse failing to produce evidence of attacks of Lichen tropicus.

**Treatment**

Numerous skin dressings and internal remedies have been used in the past without success. Subcutaneous injections of spleen extract have been given an extensive trial, but the results have been erratic. In the light of the causative parasite being shown to be the larva of a nematode, the injection of antimosan into the affected sites has been recommended by the present writer as the best procedure to adopt. Novarsenobenzol by injections subcutaneously would seem to be equally promising. Novarsenobenzol may sometimes be used locally as an ointment. Daly (1939) treated cases of Lichen tropicus and records some beneficial results from the intravenous administration of antimosan. It has been pointed out (Datta, 1936) that since the parasite has not been detected in the blood stream, intravenous administration of antimosan would appear to be irrational and circuitous. Local applications of fly repellents and emollient soothing salves are generally helpful in reducing irritation. Prevention must depend upon the knowledge of any blood-sucking arthropod that may be found to be implicated.

**Discussion**

The chance association of microfilariasis with other diseases has been discussed by Macalister (1917) in his Memoir on Kumri. The literature on equine microfilariasis was critically reviewed by Sen (1931), who gained the impression that the clinical symptoms attributed to microfilarial invasion might have been due to causes other than microfilariasis and that microfilariasis of the horse was not associated with any clear clinical picture.

The establishment of a definite microfilarial disease entity—a common skin disease characterised by its seasonal incidence, non-contagiousness, recurrence, chronic, papular, scurfy and severe irritable nature—is therefore of more than ordinary interest.

O. cervicalis occurs in tropical and temperate countries, including England.
Horses suffering from Lichen tropicus have been observed to have periods of freedom from the disease during the winter or when moved to the hills. In the acute stage of the disease, microfilarial infestation in the skin is heavy, while in the quiescent stages microfilaria are rare. The "fiery heat" of the summer appears to determine the richness of the microfilarial infestation, whether due to a larger number of infective larvae being injected during the season by blood-sucking arthropods, or due to an abnormality in the condition of the lymphatics and capillaries draining the skin. The predisposition in blacks and browns appears to depend upon the absorption of heat rays by those colours.

O. cervicalis of the ligamentum nuchae is considered by some helminthologists to be synonymous with O. reticulata, which occurs in the suspensory ligaments of the fetlock and the flexor tendons, but the pathological data available regarding the two species make this view somewhat anomalous.

Do cases of Lichen tropicus occur in Europe, being latent most of the time, similar to that in India during the winter or in the hills? Do any appreciable percentage of healthy horses harbour the parasite in the nuchal ligament without any obvious detriment? What is the significance of the parasite when encountered in cases of poll evil and fistulous withers?

Describing pityriasis in Germany, Roll stated that the disease attacks the horse principally on the head and sides of the neck, along the course of the mane and roof of the tail, but occasionally all over the body. Semmer, working in the same country, found numerous nematodes between the derma and epiderma in a horse affected with dermatosis, which was considered to be psoriasis. Baruchello, presumably in Italy, observed filariasis among cavalry horses, involving the withers, neck, forelock and base of the tail, and described dark grey tumours up to the size of a pigeon's egg and agamous nematodes therein, but it is not clear whether this report is comparable with the experience in India.

Regarding differential diagnosis, Darmagnac in France encountered dourine-like swellings in horses as a result of filarial infestation, and Pease in India described a similar disease. The chronic scurfy and irritable nature of Lichen tropicus was not present in these cases, and they must have been different from it. Cases of summer haemorrhage, due to Parafilaria multi-papillosa, characterised by the appearance of nodules each day followed by healing the following day, occur in India. Further, it is necessary to know that a form of recurrent ophthalmia due to unsheathed microfilaria occurs in young horses in certain stud farms in North India, resembling ophthalmia (associated with Guatemala nodules) of man due to Onchocerca sp. Besides this, Thelazia lachrymalis and larvae of Setaria equina (filaria oculi) occur in the eyes of horses. To avoid confusion, these facts must be known.

Lastly, regarding the name Lichen tropicus, Allen and Kingston (1928) have rightly pointed out that prickly heat, in common with numerous other
appellations, conveys very little by way of etiological implication, and suggest the name "protein rash." The word "Lichen" has been employed since the days of Hippocrates (460-377 B.C.) for papular eruptive diseases which spread on the skin, resembling to a certain extent the growth of cryptogamic lichens on trees and stones, but we owe the first precise definition of the term to the celebrated Scotch dermatologist Robert Wilan (1757-1812), as an extensive eruption of papules affecting adults, which was connected with internal disorder, usually terminating in scurf, was recurrent and not contagious. This fits in with the clinical picture of the equine disease, but since the term is inclined to prolong the existence of the idea of similarity of this condition in equines with either members of the vegetable kingdom or with human conditions of unlike etiology, a more satisfactory name should be adopted in the future. From the differential standpoint, microfilarial (or filarial) pityriasis would appear to be a most appropriate designation for all future use.

REFERENCES.

Grellier (1843): Veterinarian, 16, 182.

NORMAL BLOOD CELLS IN THE BOVINE*  
By W. P. BLOUNT, Ph.D., F.R.C.V.S.  
Lewes.

RED BLOOD CORPUSCLES

Literature
The average size of the red blood corpuscle in bovines is given as approximately 5.3m, and although Fraser, Kohanawa and Berthe carefully indicate that considerable variation in size may occur, others fail to suggest that

*Part of a thesis presented by the writer to the Edinburgh University, November, 1938, for the degree of Doctor of Philosophy.
Tuberculin, johnin, mallein, etc., may be obtained by members of the veterinary profession from the Royal Veterinary College, London, N.W.1.

Telephone
Euston 5321-5

Telegrams
Veternosis, London.

THE GLASGOW VETERINARY COLLEGE
(Incorporated)

83, BUCLEUCH STREET, GLASGOW, C.3.

Courses of Instruction are given for
- The Diploma of M.R.C.V.S.
- The Degree of B.Sc. in Veterinary Science
- The (advanced) Diploma of D.V.S.M.

WOMEN STUDENTS ARE ACCEPTED

For full particulars apply to the Principal.

Ear Canker cured—new hair grown *

A well-known dog-breeder writes about Sphagnol Peat Ointment, "I have cured ear canker in my Borzoi, and grown new hair where it was thin."

In all cases of skin disease in dogs, Sphagnol Peat Ointment acts like magic. The Peat soothes and stops irritation—therefore dogs cease to scratch. The Peat also is strongly antiseptic, thus it gets rid of the cause of the trouble.

Always keep a stock of Sphagnol on hand. Use it at the first sign of trouble on cuts, wounds or skin blemishes. If you have never tried it, the makers will send you a generous free sample. Write to:

Peat Products (Sphagnol) Ltd.
Dept. V.J.11, 21, Bush Lane,

* Sphagnol

Sphagnol Veterinary Ointment 1/- per tin, post free 1/2. 5/- per 1 lb. tin, post free 6/6. Sphagnol Veterinary Soap 10d. per tablet, post free 1/-, or obtainable from your chemist.

When corresponding with Advertisers kindly mention THE VETERINARY JOURNAL.
SUMMER TIME

STOMACH DISORDERS
DIARRHŒA  STOMACH CHILLS
COLIC   DYSENTERY

The Sure and Unrivalled Remedy is

Dr. J. Collis Browne's

CHLORODYNE

With a reputation of over 90 years.
The Safe and Reliable Family Remedy for
INFLUENZA Colds CATARRH
ASTHMA BRONCHITIS
Of all Chemists, 1/3, 3/-

THERE IS NO SUBSTITUTE!
Always ask for and

See that you get Dr. J. Collis Browne's

ORIGINAL and ONLY GENUINE CHLORODYNE

London: Printed for Baillière, Tindall & Cox, 8, Henrietta Street, Covent Garden, W.C.2,
Animal Diseases in Relation to the Economy of Man and India

BY

S. C. A. Datta, B.Sc., M.R.C.V.S.

Reprinted from
Agriculture and Live-stock in India
Vol. VIII, Part II, March, 1938
ANIMAL DISEASES IN RELATION TO THE ECONOMY OF MAN AND INDIA*

BY

S. C. A. DATTA, B.Sc., M.R.C.V.S.

Veterinary Research Officer, Imperial Veterinary Research Institute, Mukteswar

“For that which befalleth the sons of men befalleth beasts; even one thing befalleth them: as the one dieth, so dieth the other; yea, they have all one breath....”

SOLOMON (970-933 B.C.).

THE EVOLUTION OF THE CONCEPTION OF DISEASE

The significance and truth of these philosophical words of generalisation regarding life and death of man and his animals could scarcely have been appreciated in the days of Solomon the wise. Science as we know it to-day did not exist, but it is obvious that the instinct of self-preservation and the longing to relieve suffering in fellowmen had been there, and that the intriguing problem of disease (pain, suffering) causation had started to engross the human intellect. The pestilential calamities that befall men were sufficiently dreadful, and their animal possessions, providing several essential services, were being swept away equally ruthlessly. Diseases of man and animals alike thus came to be looked upon as supernatural visitations, meted out as punishment by invisible gods, demons or spirits of the dead, or as even caused by the ‘evil eye’. Invocation of the benign, and the propitiation of the malign influences with the aid of amulets, charms, incantations and sacrifices were practised as remedial measures. These notions were not by any means characteristic of any one country.

Then came the Father of Greek Medicine, Hippocrates, born about 460 B.C. He deserves credit for recognising for the first time that diseases are only processes of Nature, and that there is nothing divine or supernatural in them. He declared that the body of man in health contains four fluids or humours, blood, phlegm, yellow bile and black bile, in proper proportion and mixture; sickness is produced when one of these fluids is either increased or decreased and is no longer properly mingled with the other. Nearly two thousand years later Paracelsus urged an impartial observation of Nature as the fountain of knowledge, in place of blind belief, and with the appreciation by Morgagni of what organic alterations in the

*This is the eighth of a series of popular articles for practical farmers on various animal husbandry subjects of general interest.
structure of the body (lesions) co-existed most frequently with changes in the bodily functions (symptoms) under the general law of cause and effect, scientific medicine was born. Bichat made the discovery that organs consisted of general and special tissues, and that corresponding to these, diseases produced general and special tissue changes, and this was the inception of pathological histology (systematic study of the minute structural or morphological elements characterising each disease).

The greatest advance of this early period was, however, yet to be made by Virchow by demonstrating the full significance and the mechanism of cell activity. He showed that there is no essential difference in the laws that govern health and disease. Physiological (health) and pathological (disease) processes differ only in relation to stimuli to cells, for abnormal stimuli lead to exaggerated responses and more lasting disturbances in cell-life. He showed that pathological lesions are therefore only gross anatomical (structural) expressions of disproportions in the relative values of stimuli and cell reactions. This contribution on cellular pathology gave the right direction to the concept of disease in its progressive evolution through the ages, and in addition has had the important bearings upon all biological sciences of to-day.

In this manner, was medicine of man and animals, born almost contemporaneously of the common parentage of credulity and empiricism, and nurtured together with the milk of rationalism through the difficult years of early childhood, full of trials and errors. In surveying the progress of the healing science through the later stages of youth and adult-hood, one observes the pioneers approaching the problem of disease of the human race or animal species as a single study.

The early and subsequent knowledge of anatomy, physiology and disease processes of man was largely obtained with the aid of studies upon animals, and in this connection it is of interest to note that the earliest anatomical model known is a clay model of a sheep's liver in Babylonia dated 2,000 B.C., and the earliest recorded surgical operation is the trephining (perforating the skull) of sheep for staggers. With the commencement of specialisation in individual branches this broad perspective was lost for a time and the human physicians suffered unmistakably from the depressing influence of isolation. Any pioneering work, whether on microscopy by Leeuwenhoek, or that on bacteriology and immunology by Pasteur, or Lister's work on antisepsis, or similar advances, being applicable equally to man and animals, established the essential unity of medicine. Principles of general pathology thus emerged, providing a clear insight into the complex mechanism by which predisposing factors and exciting causes of disease interfered with the normal structure and function of living tissues (disease), and as to how factors founded in the organisation of an individual reacted to external organised agents, whether animal or vegetable parasites. An organism which derives its nourishment from the tissues of its host is a parasite, and different
classes of disease agents, bacteria, fungi, protozoa (smallest and single-celled animals) and arthropods (members of the animal kingdom with segmented body and jointed limbs) are illustrated in Plate XI. All bacteria, fungi or insects are not harmful, and it is remarkable that some of them affect particular animal species only to the exclusion of others, e.g. anthrax resistance possessed by poultry, glanders in equines but not in cattle, and rinderpest in cattle but not in horses. Regarding arthropods, some can set up disease by themselves (Plate XI, figs. 21-23), others act either as mechanical transmitters of parasites or may even provide favourable ground for the cyclical development of the parasite concerned (Plate XI, figs. 19 and 20). While the above-mentioned disease agents can be seen under the microscope, there is yet another important group—viruses—which are capable of setting up disease in healthy animals without being visible under the highest powers of the microscope. Their exact nature is not yet clearly known but the largest of the viruses have been seen in microphotographic preparations. The work on animal viruses has thrown considerable light on some of the baffling problems of this group. In the study of cancer, transmissible growths of animals have opened up a new line of approach, and the current hypothesis that the so-called gin-drinker’s liver (cirrhotic) of man was the result of alcoholism receives no support from the finding in old animals of similarly affected livers. Against Robert Koch’s memorable work on anthrax, common to man and animals, did not only reveal for the first time, a specific microbe as the cause of a disease, but what is more important, certain general principles of wide applicability as a new method of approach for ascertaining if any suspected micro-organism is the cause of a disease, emerged, and are now known as Koch’s postulates. These postulates require the constant finding of the microbe in diseased tissue, its isolation in pure culture therefrom, production of the disease artificially in healthy animals with the culture and then recovery of the organism from the experimentally infected animals.

The above remarks have been made to illustrate how, with the elaboration of a new method of research, or with the acquisition of any new knowledge, our everyday armoury against the numerous harbingers of destruction (pathogens), whether affecting man or animals, has been equally strengthened. To take one or two instances, one remembers how nutritional diseases of animals (Plate XII, figs. 12 and 13, rickets in a dog and osteoporosis in a horse) have been studied by Mellanby and others to solve the problem of similar diseases in man. Again in the comparatively new branch protozoology, the demonstration by workers in the veterinary field that certain infectious diseases can be carried from animal to man only through certain insects acting as intermediary agents (Plate XI, figs. 19 and 20) has had even more far-reaching effect in the elucidation and control of some of the most dreaded of human scourges like sleeping sickness, malaria or yellow fever. The specific instances referred to are: Theobald Smith and
Kilborne in 1893 showed that Texas fever of cattle was a tick-transmitted piroplasmosis, and Bruce showed the transmissibility of trypanosomiasis of domestic stock through Glossina flies, while Ronald Ross’s discovery of the transmission of bird malaria through Culex mosquitoes led to the discovery of the role of anopheline mosquitoes in human malaria. Did not the celebrated biologist John Hunter say that in the course of a variety of experiments on animals and vegetables he had frequently observed that the results of experiments in the one explained the economy of the other and pointed out some principle common to both? It appears that the demonstration of the nature of the virus causing mosaic disease of tobacco plants as a crystalline protein has already led to the finding of at least one animal virus, that relating to Shope rabbit papilloma to be a heavy protein, and it is not unlikely that the precise nature of the viruses of animal and human diseases will be revealed along the line indicated by plant pathology. Disease processes in man and animals have thus been mutually elucidative by throwing cross lights and analogies, and difficulties and obstacles in the understanding of the conditions, which previously appeared to be insuperable, have repeatedly been surmounted with the aid of the comparative method of studies, thanks to the work of Theobald Smith, Manson and Osler in this connection.

Importance of Animal Diseases

In the days of the primitive herdsman and since, animals have been reared and maintained primarily from the utilitarian aspect. Animals have provided meat and milk for human sustenance, clothing for protection, and motive power for tilling land and transporting produce, besides supplying fertilizers and even therapeutic agents. (In China and Egypt organotherapy was practised in ancient times). With the progress of time and civilisation, this utilitarian aspect has extended enormously, and one finds that animals or animal products are being put to many new uses in commerce and industry. Like other aspects of animal husbandry the study of animal diseases has therefore come to occupy the status of a major problem of human welfare. To recount the reasons briefly:

(a) The mutually interdependent relationship that medicine of the lower creation bears to that of the human species, and the extent to which animal experiments and the analogies provided by animal disease have influenced the progress of human medicine in the past have been already mentioned. Of equally far reaching importance has been the elaboration from animals of more and more of biological products of advanced efficiency for the relief of myriad human ills, e.g. calf lymph vaccine against small pox, horse serum for diphtheria, tetanus streptococcal infection and haemorrhage, rabbits and sheep brain vaccine for hydrophobia, etc. Further, such valuable gland products of animals as pepsin, insulin, adrenalin, pituitarin and prolan are of constant use in human therapeutics. Besides, the standardisation of the doses and potency of the preventive sera and vaccines has also been achieved by animal tests. A novel method of controlling
ANIMAL DISEASES

127

the serious blood-fluke disease of man in the Far East and Egypt (schistosomiasis) has been by the rearing of ducks and geese.

(b) Animal husbandry by itself, and along with arable agriculture, has served as the means of livelihood to an increasing number of people, or as a healthy diversion to many. It requires to be protected from the danger and loss attendant upon the onset of animal diseases, as otherwise the whole structure of agriculture is undermined. Further exact information is available to show that with modern methods of feeding, hygiene and general herd management, not only can a reduction in the incidence of animal diseases be achieved, but a shaky agriculture can be made to see prosperity.

The exact monetary value of losses inflicted at present by animal diseases on agriculture and animal industry has not been calculated recently. In Plate XII, illustrations of some chronic diseases of animals of this country have been given, and epizootic diseases like rinderpest, haemorrhagic septicaemia, blackquarter, etc., are still taking heavy toll. The cattle population has increased, and the prices of live-stock have gone up considerably since 1892 when Burke made the statement: "It has been estimated after the most careful investigation, that the annual money loss to India alone from preventible contagious disease among agricultural animals, cannot be less than £6,000,000 sterling". As a concrete instance—it may be mentioned that as a result of one disease alone—the Warble fly infection, the depreciation in the value of hides in this country has been estimated to be 1.5 crores of rupees annually. These figures indicate the magnitude of the problem of animal diseases in this poor country. Nor is this the total extent of the loss.

At one time, India used to enjoy a substantially large export trade in live-stock. Year after year this trade dwindles since the importing countries, one after another, have refused to run the risk of re-introducing diseases, which they have already eradicated from their respective countries, while the epizootic diseases continue within the Indian continent. The loss sustained by India may be appreciated from the figures of two successive years, for instance, the return from export trade in live-stock and live-stock products in 1933-34 was 32.3 crores of rupees, while in the following year the figure was only 12.9 crores. In considering these figures, one must remember this rapid and large fall in live-stock export has taken place notwithstanding the fact that Indian cattle have been found to be very suitable for rearing in different parts of the world, viz. Zebu cattle in Australia and British Guiana, Krishna Valley cattle in Dar-es-Salaam, and Indian buffaloes in Tanganyika. Further a well-known expert who has surveyed the subject of animal breeding in the British Empire, has rightly pointed out the prospect before India. To quote his own words: "If animal breeding in the tropical Empire followed a more closely unified policy and there were greater contacts between India and the Colonies, it should be possible for India to be the stud farm of the
Tropics”. In order that India may avail of the large and potential overseas market that awaits her, as a means of regaining the economic prosperity of her people, she must put her own house in order, and no longer lag behind the world developments pertaining to the campaign against animal plagues. In a country like ours, which maintains the largest live-stock population in the world, and where the annual cash value of live-stock and products has been estimated at 2,000 crores of rupees, the necessity of concentrated attention being devoted to live-stock diseases is obvious.

(c) Besides forming the wealth of the nation, animals are to a great extent connected with the health of the community. How profoundly the progress of medical research has been influenced by the study of animals and their disease has been surveyed already. It remains now to emphasise that the bearing of animals as reservoirs of infection for man, and the transmissibility of animal disease to man either directly or by handling or consuming animal foods have presented a public health problem of no mean magnitude. The part that live-stock play in determining the health of the people may be considered in two parts: (i) nutrition and (ii) disease.

Firstly, foodstuffs of animal origin are indispensable for the maintenance of health and physical development of men, for foods of plant origin do not supply proteins of such high nutritive value. In India, milk, ghee and other dairy products have even a greater importance as essential constituents of a complete dietary, than in other countries, particularly as a considerable proportion of Indians are non-meat-eaters and live on an exclusively vegetarian diet. Moreover, milk contains growth-promoting factors and accessory principles, like vitamins and mineral salts, which consolidate the resistive powers of the bodily system against diseases. The importance of milk as an essential food, producing an all-round well-being of man, is being increasingly realised all over the world, and concerted action is being taken to increase the average consumption of milk per capita by various methods, including the lowering of the price of the commodity in relation to the purchasing power of individuals. It may be noted here that one of the main findings of the Advisory Committee on Nutrition in the British Isles runs thus: “From the health standpoint there is no other single measure which would do more to improve the health, development and resistance to disease of the rising generation than a largely increased consumption of safe milk by mothers, children and adolescents.” Similarly meat and eggs form a very important part of the human dietary, as they are comparatively easily digestible, and supply in small bulk considerable amounts of nutritive and body-building materials, being particularly beneficial to invalids and convalescents. The necessity of foodstuffs of animal origin as a means of securing and ensuring human health is obvious, and the production of safe animal food is quite within human power.
Turning to the other aspect of public health, viz. that relating to animal diseases communicable to man, one finds frequent reports of the occurrence of outbreaks among human beings, in some of which very drastic results followed. Evidence exists of several cases, where the peculiar and novel features of animal diseases as exhibited in human beings could not be appreciated till collaboration or helpful suggestion of the veterinarian dealing with the disease entity as affecting his animals came forth.

DISEASES COMMUNICABLE TO MAN

Of the many communicable diseases whose only source is one or other species of animal, the most important would appear to be glanders, anthrax, rabies, foot-and-mouth disease and certain forms of tuberculosis. This statement is tantamount to saying that if these diseases could be eradicated completely from the livestock, the infection in human beings would automatically cease, and mankind would be relieved from the ravages of so many terrifying maladies. Again there are a number of diseases, the causative parasites of which require an animal host in order to complete their life-cycle before becoming infective to man. For example, Echinococcus, Trichina spiralis of the pig, the parasites of measly beef and measly pork (Cysticercus cellulosae, the larval stage of Taenia solium, and Cysticercus bovis, the larval stage of Taenia saginata of man). In these parasitic diseases as well, control of the disease in animals will necessarily produce the disappearance of the human conditions. A somewhat comprehensive list of disease of animals and man transmissible by direct contact with animals or through the agency of animal foods has been given below for reference. It will not be necessary for the purpose of the article to consider more than briefly some of the typical diseases from the list.

Rabies.—As one of the most dreadful scourges of man, and as one which necessitates the maintenance of several Pasteur institutes and anti-rabic treatment centres, thereby causing a heavy drain on the Indian Exchequer, rabies stands pre-eminent. If the treatment of hydrophobia, as the human affection is called, be not undertaken early, and the symptoms have had time to develop, a most painful death is the invariable result. The disease has been known for over 2,000 years, and its transmissibility to man and animals through the bite of a rabid dog has been recognised since the days of Aristotle (322 B.C.), but appropriate measures for the eradication of the disease have unfortunately not been adopted in many countries including our own. That the disease can be completely eradicated by stringent quarantine measures against newly imported dogs, together with muzzling order and the destruction of stray dogs, has been amply demonstrated by certain progressive countries, where adequate vigilance over this disease has been maintained.

The disease is caused by a virus, which is discharged in all the natural excretions from a rabid animal. The fact that the virus is present in the saliva at least
a fortnight before the development of any symptoms makes the disease highly dangerous. The topographical peculiarities of the Indian continent, notably the absence of natural barriers between one province and another, provide a particularly favourable field for the spread of the disease. The Pasteur method of anti-rabic vaccination stands out as one of the monuments of research, and has minimised substantially the rate of mortality from this disease. If appropriate measures for the suppression of the disease in dogs, the natural reservoirs, are systematically adopted, humanity will be saved from the risks of one of the most excruciating forms of death, and it is of paramount importance that a wide recognition of the well-known facts in this connection is brought about.

Glanders.—An equally malignant disease but of a slower course is glanders. Though mainly of equines, it is communicable to man by the discharge from the nose, eyes and skin of the diseased horses and mules. The causative organism is a bacillus (rod-shaped bacteria) with rounded ends, which finds its way into susceptible hosts through some trifling scratch or abrasion, or as generally happens, through the oral route. Notwithstanding all the scientific advancements of to-day, this is one of those diseases for which no satisfactory palliative or curative drugs have been evolved. As far as India is concerned, one may mention here that the life of one of the most promising research workers of this country—Shilston’s—was nipped in the bud as a result of glanders contracted at Mukteswar, and that another worker manifested great presence of mind and fortitude in maintaining complete records of the disease in himself.

Efficient methods for the diagnosis of the disease exist. Certain countries have already got rid of the disease, and those countries where requisite measures for its eradication have not been adopted, are standing menaces not only to themselves but to others.

Anthrax.—This disease has a world-wide distribution and affects animals of all species, but the chief sufferers appear to be cattle and sheep. The causative organism is one of the largest bacillus known, and although it had been seen earlier the pathogenic significance of the organism was established by Robert Koch in 1876. When in the blood, several organisms are found attached to each other end to end (Plate XI, fig. 4). A very well-marked capsule characterises the organism, and this fact is of value in making rapid diagnosis with blood smears made soon after death. Since the anthrax bacilli rapidly multiply in the blood-stream, and are readily disseminated throughout the body, a rapidly fatal septicaemic (blood poisoning) condition is produced, and the affected animals often do not develop any symptoms before their sudden death.

The affection is primarily one of animals but human beings are affected directly or indirectly from infective materials of animal origin. The fact that the anthrax organism readily assumes the most resistant sporulating forms, whenever it has access to a free supply of oxygen, makes the control of the disease a difficult matter. Since the carcasses may be opened without the least suspicion of the
disease being present and because blood or discharges from diseased animals may be spilt, spreading the infective principle to such places as may not readily attract notice, the difficulty of control is further accentuated. If the carcass be not allowed to be opened and is suitably disposed of, the sporulation of the bacilli is obviated. The bacilli can multiply in the surface soil, and the infection generally spreads from animal products such as hides and wool, or even from feeding stuffs containing anthrax spores. Anthrax may also be carried in the drinking water, and if diseased carcasses are not incinerated, or buried deep enough, the bacterial spores may be brought to surface soil by earth-worms. It is believed that occasionally biting flies also transmit the disease, for an authority has found the infective spores present in the flies for about twenty days.

In man the disease usually takes a cutaneous form, and one or more carbuncles (malignant pustule) may develop on the head, neck or upper extremities. When the dried spores are inhaled with impure air, the lungs are affected and what is called "Wool-sorter's disease" is produced. Through the ingestion of improperly cooked meat an intestinal form of the disease may also be set up. The disease is largely an occupational malady, being contracted by people engaged in industrial concerns dealing with hair, wool or leather, but anthrax contracted from shaving brushes has also repeatedly occurred.

From the above remarks it will be clear that the human affection can be prevented and controlled by arranging to eradicate the condition in animals. By complete disinfection of animal products used in industry, and by timely use of preventive antisera and vaccines, the disease can be controlled.

*Foot-and-mouth disease.*—As the name indicates this disease is characterised by the occurrence of foot and mouth lesions, not necessarily in the same animal and simultaneously. Eruptive blisters arise on the mucous membrane of the mouth, and at the junction of the skin and digits in cloven-footed animals. Most animals are susceptible, including guinea pigs, and human beings are also affected. The cause of the disease is a virus capable of passing through fine clay filters, which would hold back all ordinary bacteria. The virus of this disease has the distinction of being the first one among human and animal viruses, to be shown to be filtrable. This would appear to be one of the most contagious of animal diseases, and may be carried by infected litter, human clothes or water. The disease is enzootic (prevalent or regularly found) in India, and although the mortality may not be high, the affected animals are rendered unproductive for long periods. Infection by direct contact with infected animals also takes place.

In man, the disease is usually caused by the ingestion of infected milk products, and though mortality is rare in adults, drastic results in children have been seen, fever and vesicular eruptions in the mouth, and even an intestinal catarrh may be produced. The disease may be eradicated by the wholesale destruction of infected and in-contact animals, but it is such an expensive procedure that few countries can undertake to do so.
Pasteurisation of milk is an effective safeguard against the human infection, but for those who come in contact with infected animals particular attention to personal hygiene should certainly be paid.

**Tuberculosis.**—This specific contagious disease of man, animals and birds has been known to the Hindus of the Veda in the 15th century B.C. and also to the Parsees of the same time.* The disease receives its name from the word tubercle, meaning a nodule, and is caused by a highly resistant rod-shaped germ. In ordinary parlance, the human disease is called consumption, and when the lungs are affected, phthisis. That human beings can contract the disease by eating diseased meat has been recognised since the promulgation of the Mosaic Law. With the congregation of human beings into social groups, and the crowding of animals, in cities and zoological gardens, the incidence of the disease has largely increased as one of the penalties of civilisation.

The transmissibility of the disease, and its essential unity as affecting man and the lower creation was established by the celebrated Frenchman Villemin in 1865. That rabbits inoculated with material from diseased cattle developed a more rapid and severe infection than when subjected to infective materials from human cases was a very noteworthy observation made by him. Seventeen years later, the specific bacilli (Plate XI, fig. 7) of the disease were recovered by the great German, Robert Koch. Later workers discovered some differential features in the strains of the organism responsible for the human, bovine and avian disease. It is possible by a study of these features and from other facts which have emerged since, to determine whether the infection in an individual has originated from either one source or other. The disease is widely spread to-day all over the world, and as far as men and animals in this country are concerned, the disease exists not only in cities and towns but has reached the interior as well. The public health problem connected with the disease has consequently become an enormous one, and sanatoria for tuberculous patients have become a vital necessity. Preventive measures of this kind have not so generally been adopted in this country, though the urgency of intensifying combative measures against tuberculosis to meet the requirements of the situation has been repeatedly urged by both medical and veterinary workers in this country.

Regarding the veterinary aspect in India, numerous cases of the disease have now been detected among animals of all species, and the organisms isolated from the natural bovine lesions have been found to be as virulent as those in other countries. Further evidence exists to show that after the introduction of infection in crowded dairy farms a high incidence of clinical tuberculosis may develop. The usual mode of life of the stock in this country, which is largely in the open air, accounts for the relatively low incidence of the disease so far. Recent statistics have,

---

*Since this article went to press Krishnaswami Iyer has produced extracts from the ancient Hindu treatise *Haastiyayurveda* or elepl antology dating back to the period of the *Ramaayana* epic, relating to tuberculosis in elephants, *Agric. and Livestock in Ind.* 7, 722723.*
however, revealed as many as 21·3 per cent cows, 23·6 per cent buffaloes, and 31·6 per cent bullocks as affected with gross lesions. These statistics probably do not apply throughout the country, but the record of a number of definite cases of tuberculosis of the udder in milk animals including buffaloes and cows, and the demonstration of the occurrence of the specific bacilli in milk are sufficient to indicate the risk of infection to man from milk, that already exists. In a few human cases the disease has been traced to bovine infection, and it appears that one of the probable reasons why human cases of bovine origin are not more frequent is due to the practice of milk being thoroughly boiled before drinking.

A few cases of naturally acquired tuberculosis in cows caused by the human strain of the bacillus have also been recorded. The diagnosis of the disease presents no difficulty in advanced stages, and in the pre-clinical stages the use of the tuberculin test is of value. A general tuberculin testing of all cattle in selected farms should be carried out at regular intervals, followed by the elimination of reactors. The isolation of reactors and building up and maintaining of herds free from tuberculosis has been successfully tried in some countries, notably Denmark, and the same will have to be carried out in this country, where slaughter of cattle cannot generally be recommended. Promising results with methods of preventive vaccination have been obtained abroad but this work still remains to be undertaken here.

In view of the existing experience that when this disease is introduced into an area, which had previously been altogether free from it, the disease spreads rapidly and assumes a very severe form, it needs to be emphasised that parts of India are still relatively free from the disease, and if untold misery and death are to be prevented, more than ordinary attention deserves to be paid to anti-tuberculosis measures in India, which forms a somewhat virgin field for the disease.

Cow-pox.—Pocks or Variola are a group of eruptive fevers affecting different animals and are caused by viruses. Cow-pox and small pox of man appear to be closely related as they are transmissible to man and bovines respectively. Contrary to the other animal diseases communicable to man, which are invariably detrimental, the transmissibility of cow-pox (Variola vaccinia so-called) to man has been a great boon in alleviating human suffering and in preventing death from small pox. The occurrence of small pox in man dates from antiquity, and a rough and ready method of preventive vaccination (the word vaccination has originated from Variola vaccinia, meaning cow-pox) though somewhat risky was practised by the Chinese as early as the third century B.C., and the method appears to have originated in India even earlier. Experience existed among milk-maids that if they passed through an attack of the mild cow-pox contracted during milking the udders of affected cows, they were resistant to small pox. The present day method of small pox vaccination with the use of calf-lymph was perfected by
Edward Jenner in 1796, following the statement made by a milk-maid that she could not contract small pox.

**Undulant fever.**—This human affection is caused by the ingestion of cow's or goat's milk containing either one or other of the closely related organisms (Brucella abortus and melitensis) which cause abortion in cattle and Malta fever in goats. In the human disease, the fever shows irregular undulations. The prolonged duration and the complications which follow an attack make the disease a serious one. Pasteurisation of milk is an effective method of reducing risks to man.

**Weil's disease or 'Yellows'.**—This condition affects rats primarily, but human beings and dogs are also affected. The disease is a form of infectious jaundice, and is caused by a spiral-shaped organism—Leptospira icterohaemorrhagiae. Soldiers, sewer workers and ditch diggers are known to be affected, and the organisms probably infect them through the unbroken skin. The usual mode of infection is however through food or water being contaminated by rats. The organisms are found in large numbers in the kidneys and other organs of an affected animal, and they are excreted in the urine (Plate XIII).

To mention a few of the other affections communicated to man by rodents, plague, tularaemia, rat-bite fever and Rocky mountain spotted fever are examples.

**Milk-borne diseases.**—Of the diseases dealt with already, tuberculosis, foot-and-mouth disease, anthrax, rabies, cow-pox and undulant fever are some of the entities which are transmissible to man through the milk of diseased animals. In the same manner certain forms of plant poisoning (milk sickness), bacterial toxaemias (blood poisoning with bacterial poisons), and ptomain or food poisoning due to the Salmonella group of bacteria, or some septic and pyaemic diseases, may be communicated through the milk of affected cows. There is again another group of diseases, which are primarily human affections but can be carried passively by cows, or by milk contaminated by milkers or milk vendors who carry the infective germs of the disease in their body though apparently in good health (human carriers). Numerous records of serious outbreaks due to these milk-borne infections, as opposed to animal diseases communicated through milk, have been published, and the diseases implicated are: diphtheria, septic sore throat, scarlet fever, small pox, tuberculosis (human), enteric, typhoid and paratyphoid fevers and mumps. Proper pasteurisation of milk, employment of healthy milkers and milk vendors, and a systematic examination of milk are some of the preventive measures which should be adopted. In addition to the risk of the above diseases being contracted by man, the existence of these infections in many cases produces a marked deterioration of the nutritive values of milk.

**Worm infections.**—Human beings contract a number of helminthic infections directly or indirectly from animals, and Echinococcus, Trichina spiralis and the parasites of measly beef and measly pork have been mentioned earlier.
Tapeworms are ribbon-like worms, which in the adult stage consist of a head and a number of semi-independent segments or proglottids. The dog tapeworm, *Taenia echinococcus*, resides in the intestine of carnivora and consists of four segments each containing several hundred eggs. The segments on being passed contaminate food and water and thus infect man and other animals. Dogs are again infected by eating infected meat. The beef tapeworm, *Taenia saginata*, and the pig tapeworm, *Taenia solium*, affect man due to eating improperly cooked meat. Dogs are again infected by eating infected meat. The beef tapeworm, *Taenia saginata*, and the pig tapeworm, *Taenia solium*, affect man due to eating improperly cooked meat. Another important pig parasite transmitted from infected meat is a roundworm, *Trichina spiralis*. Salting and other methods of meat preservation are not dependable in destroying the *Trichina* parasite, and the parasite may live inside calcified cysts for several years. Further, as the serum from affected pigs have been found to be poisonous to susceptible animals, a toxin is probably elaborated by the parasite. It is believed that due to the risk of contracting trichnosis and infection with the pig tapeworm, the eating of pork was prohibited by Moses as early as 1500 B.C.


Of the leaf-shaped worms, flukes, *Fasciola hepatica*, *Clonorchis sinensis*, *Opisthorchis felineus* and a few others affect both animals and man, and infected animals are therefore a source of danger to human health.

In India, *Dracunculus medinensis*, the common 'Guinea worm' affects man and dogs in particular. The adult females produce nodular swellings and ulcers, and when these lesions are brought in contact with water, a mass of larvae is discharged by the man. The larvae infect a species of Cyclops and after developing inside them become infective to man and other animals. A most interesting method of controlling the disease biologically has been evolved, and consists of the rearing of the fish *Barbus puckelli* to feed upon Cyclops and the Guinea worm larvae.

*Mycotic diseases.*—Moulds and fungi belong to the vegetable kingdom, and are responsible for producing a number of skin diseases and internal infections in mammals and birds. Most animal ringworms (tricophytosis, favus, etc.) are transmissible to man producing intense irritation on the skin, scalp or the beard. The commonest is the calf ringworm which produces large circinate lesions in man (Plate XII, fig. 4). Actinomycosis is another infective disease being contracted from diseased cattle directly or through their milk or meat (Plate XI, fig. 10 and Plate XII, figs. 6-7).

*Insect rashes.*—The common mange mites, Sarcopt, Psoropt of animals, have been known to affect human beings coming in contact with diseased horses, kittens, etc. Although these acarine parasites usually produce a cutaneous disease, occasionally very drastic results may follow; as outbreaks of death among rabbits have been seen (Plate XII, fig. 5). The bird mite *Dermanyssus avium* and *gallinae*
may provoke intense erythematous and papular irritation. Most of the manges are aggravated by scratching.

The role of insects in the transmission of diseases has already been referred to, and the ticks, fleas, lice, bugs which are involved, are a legion.

Protozoan infections.—A number of protozoan infections are common to man and animals, and of these Balantidium coli, Giardia, Entamoeba histolytica, Leishmania tropica and donovani and Trypanosoma cruzi may be mentioned.

To conclude, it has been customary to consider the subject of animal diseases in a narrow and restricted manner, as relating to either agriculture or in relation to communicable diseases of man. Sufficient has been said above to show that the subject is deserving of being treated from a broader perspective of national health and wealth, as greater human activity to-day is concerned with animals and their products; and the contacts between man and animals are likely to increase. In order that human health be not undermined, and the growth of our live-stock wealth be not jeopardised, the progress of the pastoral industries be ensured, and the general development of trade and commerce be not interfered with, it is our duty to create that degree of enthusiasm and conscience in our people in this problem of animal diseases as will eventually produce the desired determination for better things, greater prosperity and more satisfying happiness.

List of diseases transmissible to man

<table>
<thead>
<tr>
<th>Nature of diseases and their names</th>
<th>Cause</th>
<th>Animals affected</th>
<th>Mode of infection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacterial diseases</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Tuberculosis</td>
<td>Tubereca bacilli</td>
<td>Bovines, cat, hog, apes, monkeys, fowls and other birds and Guineas pigs.</td>
<td>Through ingestion and inhalation.</td>
</tr>
<tr>
<td>(b) Anthrax</td>
<td>Bacillus anthracis</td>
<td>Almost all animals</td>
<td>Inoculation, ingestion and inhalation.</td>
</tr>
<tr>
<td>(c) Glanders</td>
<td>Bacillus mallei</td>
<td>Equines, guinea pigs, rabbits, and goats are also susceptible.</td>
<td>Through abraded skin. Also by ingestion and inhalation.</td>
</tr>
<tr>
<td>(d) Tetanus</td>
<td>Clostridium tetani</td>
<td>All herbivorous animals.</td>
<td>Through deep punctured wounds.</td>
</tr>
<tr>
<td>(e) Gas gangrene</td>
<td>Welch bacillus group</td>
<td>Horse, sheep, Guinea pigs, rabbits, and mice.</td>
<td>By way of wounded tissue.</td>
</tr>
<tr>
<td>(f) Undulant fever</td>
<td>(1) Brucella melitensis</td>
<td>Goats</td>
<td>Through infected milk.</td>
</tr>
<tr>
<td></td>
<td>(2) Brucella abortus</td>
<td>Cattle and hogs</td>
<td></td>
</tr>
<tr>
<td>Nature of diseases and their names</td>
<td>Cause</td>
<td>Animals affected</td>
<td>Mode of infection</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>-------</td>
<td>----------------</td>
<td>------------------</td>
</tr>
<tr>
<td><strong>Bacterial diseases—contd.</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(g) Botulism . . . . . . . . . . .</td>
<td>Organism of Salmonella group. <em>S. antriycke</em>, <em>S. enteriditis</em>, <em>S. surpaster</em>.</td>
<td>Hogs, cattle, sheep, goats and wild animals and rodents.</td>
<td>Through eating infected unboiled meat, eggs, etc.</td>
</tr>
<tr>
<td>(h) Swine Erysipelas</td>
<td>Bacillus erysipelas suis.</td>
<td>Pigs . . . .</td>
<td>Through abrasion of the skin.</td>
</tr>
<tr>
<td>(i) Tularaemia . . . . . . . .</td>
<td>Bacterium tularense</td>
<td>Rabbits and hares . . .</td>
<td>By blood sucking insects and through contact with infected animals.</td>
</tr>
<tr>
<td>(j) Impetigo . . . . . . . . .</td>
<td>Streptococcus</td>
<td>Horse . . . .</td>
<td>Direct contact.</td>
</tr>
<tr>
<td>(k) Butcher’s pempithugus .</td>
<td>Coecal infection</td>
<td>Meat . . . .</td>
<td>Wound infection in butchers and purveyors.</td>
</tr>
<tr>
<td>(l) Plague . . . . . . . . . .</td>
<td>Bacillus pestis</td>
<td>Rodents, rats and ground squirrels.</td>
<td>Through the bite of an infected flea.</td>
</tr>
<tr>
<td><strong>Virus diseases</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Foot-and-mouth disease.</td>
<td>Filtrable virus</td>
<td>Cattle and other cloven footed animals.</td>
<td>By eating or drinking raw milk products from infected cows and also by infected saliva entering the mouth through some source particularly finger.</td>
</tr>
<tr>
<td>(b) Rabies . . . . . . . . . .</td>
<td>Ditto .</td>
<td>Dogs and jackals and other warm blooded animals.</td>
<td>Through bite of rabid animals or inoculation of infected saliva through abrasions in the skin.</td>
</tr>
<tr>
<td>(c) Cow-pox . . . . . . . . . .</td>
<td>Ditto .</td>
<td>Cattle, specially cows in milk.</td>
<td>By inoculation through broken skin and inhalation.</td>
</tr>
<tr>
<td>(d) Psittacosis . . . . . . . .</td>
<td>Ditto .</td>
<td>Parrots . . . .</td>
<td>Through actual contact with sick parrots.</td>
</tr>
<tr>
<td>(e) Warts . . . . . . . . . . .</td>
<td>Virus .</td>
<td>Dogs and cattle . . .</td>
<td>Direct contact.</td>
</tr>
<tr>
<td><strong>Diseases of human origin</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Septic sore throat . . . . .</td>
<td><em>Streptococcus</em> <em>epidemicus</em>.</td>
<td>Milk cows . . .</td>
<td>Through drinking infected milk.</td>
</tr>
<tr>
<td>(b) Diphtheria . . . . . . . . .</td>
<td><em>Corynebacterium diptheriae</em>.</td>
<td>Cats, fowls and cows . . .</td>
<td>Through direct contact and drinking infected milk.</td>
</tr>
<tr>
<td>Nature of diseases and their names</td>
<td>Cause</td>
<td>Animals affected</td>
<td>Mode of infection</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>-------</td>
<td>-----------------</td>
<td>------------------</td>
</tr>
<tr>
<td>Scarlet fever</td>
<td><em>Streptococcus</em> scarletii</td>
<td>Cows</td>
<td>Through drinking infected milk.</td>
</tr>
<tr>
<td>Typhoid</td>
<td><em>Bacillus of typhoid</em></td>
<td>Apes, Chimpanzees</td>
<td>Through contact and ingestion of material infected with bacillus.</td>
</tr>
<tr>
<td>Paratyphoid fever</td>
<td><em>Salmonella</em> sp.</td>
<td>Young calves</td>
<td>Food poisoning due to infected meat.</td>
</tr>
<tr>
<td>Helminthic infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tapeworm infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1) <em>Taenia solium</em></td>
<td><em>Cysticercus cellulose</em></td>
<td>Pigs</td>
<td>Through eating improperly boiled meat.</td>
</tr>
<tr>
<td>(2) <em>Taenia saginata</em></td>
<td><em>Cysticercus bovis</em></td>
<td>Cattle</td>
<td>Ditto.</td>
</tr>
<tr>
<td>(3) Hydatid</td>
<td><em>Echinococccus granulosus</em></td>
<td>Dogs</td>
<td>Ingestion of material contaminated with the droppings of infected dogs.</td>
</tr>
<tr>
<td>Roundworm infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1) Trichinellosis</td>
<td><em>Trichina spiralis</em></td>
<td>Pigs</td>
<td>Through eating improperly boiled infected meat.</td>
</tr>
<tr>
<td>(2) Guinea worm infection</td>
<td><em>Dracunculus medinensis</em></td>
<td>Horse, cattle and dogs</td>
<td>Through cyclops.</td>
</tr>
<tr>
<td>Fungus infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actinomycosis</td>
<td><em>Actinomycosis bovis</em></td>
<td>Almost all animals</td>
<td>Through ingestion by eating or drinking infected meat, or milk.</td>
</tr>
<tr>
<td>Ringworm</td>
<td><em>Tinea tonsurans</em></td>
<td>Cattle, horse, cat and dogs</td>
<td>Through abraded skin.</td>
</tr>
<tr>
<td>Large spored ringworm</td>
<td><em>Tricophyton tonsurans</em></td>
<td>Horse and cat</td>
<td>Ditto.</td>
</tr>
<tr>
<td>Favus</td>
<td><em>Achorion schonleinii</em></td>
<td>Dogs, mouse</td>
<td>Ditto.</td>
</tr>
<tr>
<td>Arthropod infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acariasis</td>
<td><em>Dermanyssus gallinae</em></td>
<td>Chickens</td>
<td>Through biting of mites.</td>
</tr>
<tr>
<td>Mange</td>
<td><em>Sarcoptes scabei</em></td>
<td>All domesticated animals except cat</td>
<td>Through contact.</td>
</tr>
</tbody>
</table>
### Nature of diseases and their names

<table>
<thead>
<tr>
<th></th>
<th>Cause</th>
<th>Animals affected</th>
<th>Mode of infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a)</td>
<td>Syphilis</td>
<td>Spirochaeta pallida</td>
<td>Linhas</td>
</tr>
<tr>
<td>(b)</td>
<td>Balantidium coli</td>
<td>Living cysts</td>
<td>Hogs</td>
</tr>
<tr>
<td>(c)</td>
<td>Giardiasis</td>
<td>Ditto</td>
<td>Mice and rats</td>
</tr>
<tr>
<td>(d)</td>
<td>Amoebiasis</td>
<td>Entamoeba histolytica</td>
<td>Dogs and cats</td>
</tr>
<tr>
<td>(e)</td>
<td>Leishmaniasis</td>
<td>Leishmania donovani</td>
<td>Dogs</td>
</tr>
<tr>
<td>(f)</td>
<td>Spirochaetal jaundice</td>
<td>Leptospiraicterohaemorrhagiae</td>
<td>Rats</td>
</tr>
<tr>
<td>(g)</td>
<td>Rat bite fever</td>
<td>Leptospira mornus muris</td>
<td>Rats</td>
</tr>
<tr>
<td>(h)</td>
<td>Trypanosomiasis</td>
<td>Trepanosoma cruzi</td>
<td>Small laboratory animals</td>
</tr>
</tbody>
</table>

#### EXPLANATION OF PLATES

**PLATE XI**

*Disease producing agents*

1. Spirochaetes cause severe disease in poultry.
2. Staphylococci  

   Pus producing organisms set up a number of diseases in man and

3. Streptococci  

   animals.
4. Anthrax bacillus causes anthrax in man and animals.
5. Bipolar organisms produce human plague and also animal disease.
6. Tetanus bacilli produce tetanus in man and animals.
7. Tubercle bacilli produce tuberculosis.
8. Fungus mycelia  

   Skin diseases and also diseases of internal organs.
9. Budding fungus  

10. Actinomyces causes lumpy jaw in cattle.
11. Cryptococcus causes epizootic lymphangitis of horse.
13. Leishmania affects man and dogs.
14. Trichomonas produces sterility and abortion.
15. Trypanosomes causes surra in animals and sleeping sickness in man.
17. Babesia bigemina causes red water in cattle.
18. Haemoproteus columbae occurs in birds.
19. Boophilus (tick) is a vector of red water in cattle.
20. Tabanus—a fly—believed to be the carrier of surra (trypanosomiasis).
22. Sarcoptes } produce scab in man and animals.
23. Psoroptes }

**PLATE XII**

*Some chronic diseases of animals in India*

1. Hill calf showing generalised ringworm patches (trichophyton infection).
2. Infected hair from the above calf treated with hot caustic potash for microscopic examination—Fungus mycelia and spores seen.
3. Ditto. Hair in tact showing beaded spores.
4. Cow-boy infected on the cheek by a large circinate ringworm from an infected calf.
5. Rabbit in a moribund condition due to extensive infection with acarine parasite, mange.
6. Hill bull affected with actinomycosis of the upper jaw bone.
7. The causative fungus of the same in microscopic preparation.
8. Horse affected with lichen tropicus, so-called *khoojlee*, caused by a microfilaria shown at 9.
10. Cattle showing yoke gall complicated with "Calcutta sore", due to a microfilaria seen coiled up in section of the tissue at 11.
12. Rickets in dog—a nutritional deficiency disease. Left dog shows bony enlargement in the limb joint. Right dog shows a normal pup of the same litter.
13. Osteoporosis or ‘big head’ in a pony—another nutritional disease due to imbalance of phosphorus in relation to calcium.
14. Leucoderma in a pony—Depigmentation around the eye and extending on the side of the face in a symmetrically bilateral manner—believed to be of disturbed metabolic origin, similar to the human disease.

**PLATE XIII**

This plate illustrates how Weil’s disease of man produces Yellows or infectious jaundice in dogs, and vice versa through the intermediate agency of rats.

GIPD—M 1CP—14.4.38—50.
SOME CHRONIC DISEASES OF ANIMALS IN INDIA
Weil's disease or "Yellows" in dog and man
PREVALENCE OF TUBERCULOSIS AMONG CATTLE IN INDIA.

BY

Capt. S. C. A. DATTA, B. Sc., M.R.C.V.S., V.R.O.,
In-Charge-of Pathology, I.I.V.R., Muktesar.

Reprint from
THE INDIAN VETERINARY JOURNAL
Vol. XII, No. 1, July, 1935.
PREVALENCE OF TUBERCULOSIS AMONG CATTLE IN INDIA.

By
CAPT. S. C. A. DATTA, B. Sc., M.R.C.V.S., V.R.O.,
In-Charge of Pathology, I. I. V. R., Muktesar.

The number of careful examination carried out in India upon cattle for the detection of tuberculosis is relatively small. Tuberculosis has been generally held to be a rare disease among cattle in India and the rarity of the disease has been attributed, on the one hand by Liston and Soparkar (1917) to a natural resistance to the disease possessed by Indian cattle and on the other hand, by Sheather (1921) to a relatively low degree of virulence of the causal organism. Apart from the cases of tuberculosis that have been met with in towns, a case of the disease in a village cattle in the interior of Khandesh has been reported by Joshi (1920). In summarising the position of the early workers, Edwards (1927) states: "Examination of carcasses of slaughtered cattle at the Ferozepur (Taylor) and Cawnpore (Oliver) slaughter houses revealed microscopic lesions of tuberculosis in approximately 3% of the animals. The lesions were invariably, however, minute in extent, localised to the tissues in which they are most commonly found and retrogressive in their appearance. Tuberculosis has also been detected in Madras among conservancy bullocks (Krishnamurti), Bombay Presidency (Sowerby, a somewhat severe case) and in the Calcutta Jail, apparently from the result of tuberculin tests (Kerr). The Imperial Dairy Expert, Mr. W. Smith in 1923, however, declared that from an experience extending over many years among military dairy cattle all over India, he was unacquainted with the disease. Curiously, in the cold weather of 1923–1924, a deputation of Indian students, sent by me under one of my officers to ascertain the incidence of the disease of the Lahore and Ferozepur slaughter houses, failed to discover lesions in carcasses subjected to an ordinary examination such as is commonly performed in meat inspection." In reference to this statement of Edwards, Soparkar (1927) observed: "I have recently found that in certain localities it is prevalent to a greater extent than has hitherto been recorded. Examination of carcasses at the slaughter houses at Ferozepur and Lahore has shown an incidence of over 16%."

The situation in India now would appear to be actually becoming serious for, in a very recent publication, Soparkar and Dhilon (1931) have stated, from experience at the slaughter house at Lahore, that
out of 1,116 animals examined, the rate of infection was found to be 21.3% in cows, 23.6% in buffaloes, and 31.6% in bullocks. According to Soparkar (1926) the susceptibility of buffaloes following upon artificial inoculation worked out at 88.8% in the case of Jafferbadi buffaloes and 87.5% in the case of Murra buffaloes, and he was thus led to regard the buffalo as being somewhat more susceptible to tuberculous infection than the cow.

TRANSMISSION OF TUBERCULOSIS TO HUMAN BEINGS THROUGH MILK OF COWS AND BUFFALOES

The experimental investigations of Liston and Soparkar (1917) show that most of the cases of glandular and surgical tuberculosis of man in this country are due to infection with the human type of bacilli. They failed to demonstrate the bovine type on analysing 100 cases of surgical tuberculosis. They were inclined to the view that the cattle in India are rarely infected with tuberculosis and that they were more resistant to infection with the bovine bacilli than are imported cattle. The views of Cook (1902) and Wilkinson (1914), Veterinary Officers-in-charge of slaughter houses, support this statement. Again, with regard to the presence of tubercle bacilli in milk, a systematic examination of Bombay milk was made by Joshi (1914), and out of 674 samples examined by him 47 or 7.6 percent showed the presence of acid fast bacilli, but in not a single sample could tubercle bacilli be demonstrated by animal inoculation. Gloster (1914) examined 101 samples of milk, principally from single cows. In not a single instance did the guinea pigs inoculated with the sediment obtained from the milk develop tuberculosis. The failure of these workers to demonstrate a higher incidence of the specific bacilli in the milk of Indian cattle may be partly attributable as stated by Soparkar (1927), to the open-air life of livestock in this country. Evidence is now available from a certain Military Dairy Farm to show that under conditions of domestication simulating those of the West, a high incidence of clinical tuberculosis may develop in a herd after introduction of the infection. Soparkar (1929) records having succeeded in producing tubercle lesions in guinea pigs by inoculating them with material derived from a slightly enlarged spleen of another guinea pig which had failed to reveal B. tuberculosis in its tissues, after it had been inoculated with the bovine type of bacilli recovered from a girl. This is the first instance on record in which the bovine type of bacilli has been isolated from a human case of tuberculosis in India. Further he has been able to isolate fully virulent tubercle bacilli from the tissue material of some apparent reactor to the subcutaneous tuberculin test, though no naked eye lesions were detected on slaughter, and this experience has been
corroborated in several cases recently at this Institute. In the words of Edwards (1927), it is not unlikely that what appeared to be a disconcertingly high proportion of reactors may be affected with what is known as "no-lesion tuberculosis," indicative of an obscure or latent infection of the kind detected by Soparkar. Strains of tubercle bacilli isolated from the restricted lesions observed in Indian cattle have been proved to be fully as virulent as control strains of highly virulent bovine bacilli obtained from Europe. Contrary to the experience of Edwards (1927) contained in the statement: "We have not yet encountered any case of macroscopic affection of the udder with tuberculosis" two cases of tuberculous mastitis have been detected by us at this Institute.

Of the half a dozen cases of clinical tuberculous mastitis of milch cattle or the buffalo so far recorded in India, the case of Brodie Mills (1898) appears to be the earliest to be recorded. Since this case is interesting and has not been noticed by previous writers on Bovine Tuberculosis in India, Mills' report is quoted in extenso. "A cow was brought for inspection with the following history. It had been purchased from a native dealer some weeks previously with a calf at her foot. The milk she yielded was given to a European infant which brought on diarrhoea of a persistent character. The calf also suffered from the same malady and died. On examining the cow, the udder was distinctly tuberculous and the immediate discontinuance of the use of her milk was urged upon the infant's mother, with the result that the diarrhoea from which it suffered soon ceased. The cow was now subjected to two separate tuberculin tests, to both of which there was the distinct reaction. The owner then consented to the animal's destruction and the autopsy revealed not only in the udder but in the lungs the true characteristics of tuberculosis." Evidence collected by European workers shows that virulent bacilli may be excreted by an infected animal with the milk even when the udder fails to show any obvious signs of the disease.

LITERATURE.


Gloster, T. A. (1914) A preliminary enquiry into the prevalence of Tuberculosis among Bombay cattle.

Joshi, L. L. (1914) The milk problem of Indian cities.


Lankester, A. (1920) Tuberculosis in India, Calcutta.


REPRINTED FROM

THE INDIAN JOURNAL OF VETERINARY SCIENCE AND ANIMAL HUSBANDRY.

ORIGINAL ARTICLES

SCHISTOSOMA INDICUM, MONTGOMERY, 1906, AS THE CAUSE OF A PERSISTENT DEBILITY IN EQUINES IN INDIA, WITH A DESCRIPTION OF THE LESIONS

BY

CAPT. S. C. A. DATTA, B.Sc., M.R.C.V.S.,

Imperial Institute of Veterinary Research, Muktesar.

(Received for publication on 9th November 1932.)

(With Plates I to VI.)

INTRODUCTION.

Apart from cases of hepatic cirrhosis in man, where the ill effects of alcohol or syphilis can be traced, the pathogenesis of hepatic lesions is largely obscure, but a perusal of veterinary literature shows that hepatic cirrhosis of the horses has been described from several parts of the world. The occurrence of what is termed 'Schweinsberger disease' [McFadyean, 1889] in parts of Germany is known, but the cause has not yet been ascertained. A form, commonly known as 'Bottom disease', has also been described as occurring on the marshy side of the Missouri, in which a species of Crotalaria (C. sagittalis) has been incriminated [Schroeder, 1892]. Gilruth [1903] has described another form as 'Winton disease' due to Ragwort poisoning (Senecio jacobea), and an enzootic form of liver cirrhosis, Dunziekte, has been described from various parts of South Africa, where a similar plant is considered to be the toxic agent [Hutcheon, 1903, Robertson, 1906, Verney, 1911, and Theiler, 1917 and 1918]. Craig and Kehoe [1921] have recorded the occurrence of cirrhosis of the liver in the horse in parts of Ireland which has also been attributed to plant poisoning.

Excepting possibly the cases of Oreste and Ercolani, which will be referred to below, no records are available, however, from other parts of the world of cases of liver cirrhosis which resemble in any great degree the peculiar type to be considered in this paper. Of the many problems which have engaged the Veterinary Scientist in India, nodulated portal cirrhosis of the horse's liver, associated with intractable debility, has been one of the most difficult to solve. In first describing the condition as "Calcareous degenerations in the horse", Smith [1885] stated that it had
been encountered in a very large number of postmortem examinations extending over several years and that the condition was interesting as an undescribed feature in animal Pathology. Further he considered that the tissues and organs were liable to this degenerative change in varying degrees in the following order:—Liver, lungs, intestines and limbs.

Other later reports on apparently the same condition are available, but the authors do not seem to have been aware of the first report by Smith, which latter may still be considered as containing the best representation of the macroscopic features. To quote Smith's own words: “The most unsatisfactory part of the enquiry was the determination of the cause. On this I have no opinion to offer. I presume there is some defective secretion of lime from the body, of which salt the horse excretes a considerable quantity in health, it being probably held in solution by the carbonic acid, but what can lead to its deposition I cannot conjecture.” He was emphatic that the changes were not the result of any parasitic invasion, but this view cannot be accepted any longer for the reasons which will be developed in the course of this paper. Much credit is due to him, nevertheless, for having realised, intuitively as it were, the possibility of these calcified lesions affecting the intestines, liver and other internal structures being related and having the same etiological basis. Judging from what information is available in the reports of Oliphant [1880], Meyrick [1873], Smith [1879], Burke [1880—1884] and Steel [1881], on the subject of so-called internal Bursati involving the liver, lungs, spleen, etc., it is possible that these minute calcified nodules had been encountered, in at least some cases, by workers much earlier even than Smith. Steel [1884] refers to a case of an enormous liver, which he considers to be an exceptional finding. With an experience of over fifteen years Lingard [1905] states that calcareous nodules in the liver are of very frequent occurrence and that one rarely makes an autopsy on any breed of equine which has been any length of time in this country without finding a few calcareous nodules under the capsule or in the parenchyma of the organ. The photograph of a portion of liver that he puts forward (Plate II), as showing perihepatitis and what he calls small filarial nodules, leaves little doubt about the identity of the condition with the subject matter of this article. He says that these nodules occur in the cortex of the kidney and describes two types of nodules in the lungs, the smaller variety being very small calcareous bodies about the size of a grain of mustard seed; and a larger variety, which is less frequent and of irregular shape presenting rough, worm-eaten surfaces. With regard to the liver nodules he cites earlier European literature:—“Colin and Reynal [1862] were the first to make mention of white or yellow calcified and irregularly spherical nodules in the liver of the horse. These were formed of numerous concentric layers varying in size from a millet seed to a pea, although there
Fig. 1. (×4)

Fig. 2. (×32)
For explanation see page 26.

Fig. 3. (×6)

Fig. 4. (×3.2)
were some which were scarcely visible. Oreste and Ercolani were of opinion that these nodules contained as a nucleus the ovum of a distoma, but according to Mazzanti they are produced by the embryos of nematodes carried by the blood.” Another statement of Lingard should be quoted here in connection with the filarial theory of origin mentioned above:—“No filaria embryos were observed in the blood during life, and no cutaneous lesions occurred on the body, but calcareous deposits were found in the parenchyma of the internal organs, especially in the liver and lungs, post mortem.” Again Montgomery [1906] also encountered small pearl-like nodules in enlarged livers in all cases of equines from which he collected S. indicum, but as will be seen later he was inclined to ascribe them to filarial embryos. In Montgomery’s experience at Muktesar, 76.9 per cent. ponies were infested with schistosomes, but no definite changes, apart from petechial hemorrhages and chronic venous congestion, were detected in the intestines, although 20 or 30 parasites were readily detected.

The condition described in this article is known to be very prevalent amongst army animals in India, and although figures are not available, it is probably safe to assume that heavy losses are incurred also by the civil population in India as a result of this condition, since it is unusual to find the same standard of sanitation, hygienic conditions of watering, feeding and housing, etc., of equines in the villages and towns in the various parts of the country compared to that usually obtaining in military establishments in India. As Lingard [1905] points out, in country-bred ponies, but poorly fed and working in unhealthy districts, one occasionally finds at post mortem their livers riddled with concretions so closely set together that additional nodules could be introduced with difficulty.

As a result of some intensive work recently carried out at this Institute, it is now possible to record certain definite findings concerning the etiology of this condition. It will be shown that this peculiar affection of equines in India is a clinical manifestation of Schistosomiasis, the predilection seat for the deposition of the ova being the distal portions of the intestinal tract, particularly the large colon and rectum, and also the liver.

Due to certain unavoidable circumstances, the present studies had to be restricted to such preserved tissues as have been received at this Institute from outside since 1921, involving about 60 consignments of specimens which were collected from about 80 cases. The samples consisted mainly of pieces of liver, though in a few instances portions of the intestines, portal and mesenteric glands, and lungs were also included. For the preparation of Plate 1, as an illustration of the typical macroscopic lesions, some of these small pieces of morbid tissue have been selected and assembled together.
The complete clinical picture of this condition, from the initial infection to the final culmination, has yet to be described and it is unfortunate that clinical cases of the disease were not available at Muktesar prior to the preparation of this article. Further it has not been possible to undertake a tour to the enzootic areas for studying the clinical aspects at first hand. In view of the above difficulties, it seems advisable to reproduce a small number of reports of representative cases, as supplied by field workers with actual experience of the disease, for the benefit of readers, and it must be made clear that only such cases have been chosen as have been confirmed by macroscopic and histological features to be due to schistosome infection.

**Case Report No. 1.**

Two specimens of liver were received from Rawalpindi on 7th February 1922. The covering letter stated that about twelve months earlier (on 31st January 1921) the mules and horses which constituted the 17th Pack Battery proceeded to the Murree Hills and retained good condition up to June. From the moment the rains set in, however, the animals as a whole fell away rapidly in condition and remained so in spite of the strictest supervision and veterinary treatment of about 15 of the very worst cases. The animals retained excellent appetites and were lively throughout, having exhibited no other symptom excepting debility. A negative reaction to the Mallein test was obtained and no organisms could be demonstrated in the blood stream. A post-mortem examination on mule No. 144, destroyed on 30th January 1922, showed a Habronema tumour in the stomach. Large intestine contained strongyles and encysted worms in the bowel walls. Liver was apparently of normal size but was dotted throughout with fibrous nodules. Another animal—horse No. 16—was destroyed the next day. Post-mortem examination showed Habronema worms in the stomach. Calcareous nodules varying up to the size of a pea were found distributed throughout the intestines but these were most abundant in the rectum. A few calcareous nodules were also seen in the lungs. Liver was markedly enlarged and cirrhotic, being 50 lbs. in weight. The organ was hard and unpliable and contained calcareous material. The post-mortem appearances suggested that the whole battery was infected in the same way. In order to rid the animals of the common gross parasites of the digestive tract, the use of carbon disulphide and oil of chenopodium was recommended.

Histological examination carried out then indicated a very intense worm infestation of recent origin in the first case (mule No. 144) and a more chronic form of worm infestation in the second case (horse No. 16). It was not possible to determine the species of the worm but nematodes were suspected as having...
migrated into the liver probably through the peritoneal cavity, from the stom. h or intestines.

**Case Report No. 2.**

Specimens of liver and intestine of a mule destroyed for chronic debility were received from Mhow on 29th June 1923. Liver was very enlarged and weighed 36 lbs. The consistence was cartilaginous. The nodules in the rectum and large intestines were thought by the Veterinary Officer in charge of the Hospital to be due to *Habronema megastoma* but no habronemic abscess was detected in the stomach. The laboratory report was as follows:—Two tumour-like growths, sprinkled on their summits with dark spots of the size of a pinhead, occurred in the pieces of the intestine. Liver was markedly cirrhotic and the mouths of the biliary ducts abnormally thickened. The presence of nodules in the peritoneal surface of the large intestines is not infrequently seen in strongyle infestation of the large intestines in equines. It is difficult to state to which species the lesions are due. Gross cirrhotic lesions of the liver, such as are seen in the liver examined, are known to be due to several species of worms but the species of worm responsible for the condition could not be traced. It seems improbable that *Habronema* species cause these lesions, for they are usually confined to the stomach. Besides the verminous lesion reported, there was very intense infiltration with neutrophile leucocytes and this appeared to be connected with the presence of large numbers of organisms. Very large sporulating bacilli were seen but were considered to be secondary and of little importance.

**Case Report No. 3.**

Specimen of liver collected from a mule which died of internal hæmorrhage was received on 31st July 1924 from Lucknow. Since March 1924 the mule had been treated for debility, at first in the unit lines and for the last two months of its life in the Station Veterinary Hospital, but no improvement was noticed. On the evening of the 28th July he showed signs of internal hæmorrhage, which might have been caused by injury, and he died a short time afterwards. On autopsy, the peritoneal cavity was found to be full of blood due to rupture of the vessels in the region of the liver. The liver was much enlarged and weighed 38 lbs. Two small habronemic tumours were found in the stomach wall, one of these discharging a greenish yellow pus.

Histologically, the lesion appeared to be undoubtedly one of intense verminous hepatitis. Scattered throughout the liver tissue and mainly in an interlobular situation, numerous nodulated lesions were seen. The report went on to state that this type of condition was known to be set up by intense fluke invasion of the
liver in other animals, notably sheep, but that one was not acquainted with this type of lesion occurring in the horse. It was not unlikely that the invasion was a migration of Habronema worms from the stomach, where two small tumours resembling those commonly caused by H. megastoma were found. No bacterial microorganisms were detected in the liver tissue.

**CASE REPORT No. 4.**

Portions of liver and intestine of a mule which was destroyed for debility were received on 19th September 1924 from the Military Veterinary Hospital, Lucknow. Naked eye examination of the intestinal mucous membrane showed certain minute black patches, direct smears from which were examined by Mr. Amarnath, M.Sc., then Zoological Assistant to the Director of this Institute, and he found spined ova resembling those of Schistosoma indicum, but on an extensive examination of the specimens of liver and intestine from this and other previous cases, this finding was not confirmed. On this being intimated to the Veterinary Officer concerned, the following detailed history was forwarded:

Two mules Nos. 21 and 28 of 5/1st Punjab Regiment were admitted to hospital on 7th May 1925, both mules being in a debilitated condition. The animals exhibited no other symptom than general anaemia. There was no rise of temperature and they always fed well. Methods of treatment included stomach lavage, intravenous quinine hydrobromide, tonics, extra feed with linseed, lucerne, etc. The mallein test gave negative results in both cases. A slight improvement in condition was noticed for about six weeks but soon they again started to go down. As both the animals became very debilitated indeed, they had finally to be destroyed on 3rd September 1925. Exactly similar lesions were discovered on post-mortem examination in both cases. No adult schistosomes were detected in the intestines but the blood vessels were not examined. The officer further stated that he had come across similar cases in Waziristan and Delhi.

**CASE REPORT No. 5.**

Liver from mule No. 7 of the 1st Battalion, Royal Fusiliers, which was destroyed on 19th February 1927 at Ambala, was forwarded for examination. The mule was admitted to hospital for debility on 27th November 1926. He was apparently healthy but in poor condition. The abdomen was pendulous and the back muscles atrophied. Extra feeding and medical treatment did not result in any increase in weight. He was treated for Habronemiasis and stomach lavage was given on three successive occasions. Blood smears were examined with nega-
tive results. Tonics containing strychnine were prescribed. The following post-mortem findings were recorded:

Liver enlarged, 32 lbs. in weight. Capsule covered with innumerable small papules the size of a pea, giving an uneven surface to the liver. When the liver substance was cut into, the surface had a marbled appearance. Some small papules were found on the diaphragm and also on the serous coat of the small intestine. Four Habronema worms were found in mucous coat of the stomach. The officer further stated that, about three weeks earlier, he had an identical case in a mule which he destroyed for debility and that he had two more mules in the hospital which he suspected to be suffering from the same condition. In the histological report, it was stated that the picture was that of a very advanced cirrhosis of long standing. Very little liver tissue could be detected in sections as it had been replaced to a large extent by fibrous connective tissue. Naked eye appearance was characteristic of "hobnail liver". No parasites to account for the lesion were detected and this result was explained on the hypothesis that in the tissues received hitherto, including the present case, the lesion almost certainly represented the end result of an infection, in which it was no longer possible to discover the causal agent.

CASE REPORT No. 6.

Pieces of lung and liver from mule No. 29 of No. 16 D. T. T. Company, Secunderabad, were received on 23rd August 1929.

The liver was studded with numerous millet seed sized calcified nodules both on the surface and in the parenchyma. The small pieces of lung appeared a little denser in consistence and contained what appeared to be fibrous nodules possibly of old verminous origin. Bronchial and mediastinal glands were rather enlarged, especially the latter, which was twice the normal size. The glands were oedematous on section but showed no macroscopic lesions.

During life the mule had a pot-bellied appearance. There was very obvious deficiency of muscle on the back and quarters and he showed markedly accelerated respiration. The temperature was normal except on the first day of admission and the animal fed well. He was admitted to hospital on 7th August 1929 with the record of a progressive loss of condition and with the appearance of a case of broken wind, but the character of the respiration was not typical of broken wind. It was destroyed on 16th August 1929 as unlikely to recover.

Apart from the usual picture of nodulated portal cirrhosis of the liver, histological examination of lung pieces made then revealed a chronic catarrhal type of pneumonia, believed to be directly or indirectly connected with a parasitic infection.
CASE REPORT NO. 7.

Liver, lung, spleen and mesenteric gland, collected from mule No. 10, 2nd Indian Divisional Signals, Quetta, were received on 28th November 1929.

Veterinary History Sheet.

Dark Brown Gold Mule No. 10.

<table>
<thead>
<tr>
<th>Date of admission</th>
<th>Date of discharge</th>
<th>Number of days</th>
<th>Disease</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>17-5-24</td>
<td>10-7-24</td>
<td>54</td>
<td>Debility</td>
<td>Duty</td>
</tr>
<tr>
<td>22-6-25</td>
<td>30-6-25</td>
<td>8</td>
<td>Ulcer, Bursati</td>
<td>Duty</td>
</tr>
<tr>
<td>30-5-27</td>
<td>6-8-27</td>
<td>68</td>
<td>Ulcer, Bursati</td>
<td>Duty</td>
</tr>
<tr>
<td>15-11-27</td>
<td>5-12-27</td>
<td>20</td>
<td>R. eye Pedal Ostitis O. F.</td>
<td>Duty</td>
</tr>
<tr>
<td>27-9-29</td>
<td>18-11-29</td>
<td>52</td>
<td>Entozoa</td>
<td></td>
</tr>
</tbody>
</table>

For the last two years of its life, this mule had been in poor condition, no muscles on back, pot-bellied, with obvious signs of ascites. During training that year he proved to be of no use. Being unable to do his work he was admitted to the Veterinary Hospital and treated for Entozoa, although no eggs or parasites were found on faecal examination. He was destroyed on 22nd November 1929 and the post-mortem examination showed the following lesions:

Serous flocculent fluid in the abdominal cavity. Liver cirrhotic, greatly enlarged, weighing 31 lbs. and almost white in colour and permeated with hard nodules. Lungs and spleen were in a similar condition. Blood from the portal vein was examined but no schistosomes were found.

Histological examination showed the lungs and liver to be studded with very numerous calcified nodules varying from the size of a pinhead to that of a pea. Spleen also showed a few such nodules with areas of infarction, the last mentioned lesion being probably connected with embolism from the same cause as of the lungs and liver lesions.

CASE REPORT NO. 8.

Liver, lung, small colon and mesenteric lymph gland were received from Military Hospital, Jubbulpore, on 7th March 1932.

The subject was a Bay Australian Mare—12 years old. Joined “G” Divisional Signals on 8th August 1926 and was stationed at Jubbulpore and Secundera-
bad. This horse had apparently been in good health until August 1931, the only diseases of importance previous to this were colic in 1926, and influenza in February-March 1932. She was treated for intestinal parasites in August-September and for colic twice during September and October 1931. Admitted to hospital on 5th January 1932 in poor condition and treated for intestinal parasites. As there was no improvement in condition, the animal was tested with Mallein and afterwards with Tuberculin, in each case with a negative result. She was destroyed on 27th February 1932. Post-mortem examination showed the following:—

Liver.—Cirrhosis and small calcified nodules throughout the parenchyma, weight 47 lbs.

Lungs.—Numerous calcified nodules throughout, about the size of a grain of gram.

Small Colon.—The external surface of the whole of this part of the intestine was entirely covered with nodules of a fibrous consistency.

Large Colon.—Lesions similar to those of small colon were present throughout, but were much less numerous.

Kidneys.—Oedematous, cirrhotic, capsules adherent. Mesenteric lymphatic gland—small calcified nodules present.

Bladder.—One nodule on internal surface.

All other organs apparently normal, parietal pleura and peritoneum healthy.

Histological examination of serial sections revealed Schistosoma indicum ova in the centre of the nodules in the liver, small colon, mesenteric gland and the lung.

Although considerable pains have been taken in compiling the above case reports to include as comprehensive a description as possible of the symptomatology of the condition on the basis of the covering letters which accompanied the samples of specimens in the collection of this Institute, and which have now been proved to be definite cases of Schistosomiasis, it will be readily realised that the clinical descriptions given above refer to the late stages only. At this stage the animals, from which the tissues were collected, had either naturally succumbed to the disease or they were in such a "worn out" and incurable condition that they had to be destroyed. Further in these advanced cases the symptoms exhibited were obviously the result of the primary infection with its secondary sequelae. In order that field workers may be in a position to recognise with precision the clinical symptoms of the initial stages of this disease, it seems advisable that a few remarks to supplement the case reports be made here. As a result of the present studies and from what knowledge is available of such forms of human bilharziosis
as simulate the cases now under consideration, viz., Schistosomiasis of the Far East (Katayama disease, due to *S. japonicum*) and Egyptian Schistosomiasis, due to *S. mansoni*, it is easy to understand why the earlier symptoms of the equine disease have failed to be recorded. The cercarial infection may take place in one of two ways, viz., either through the cutaneous route or through the digestive tract, and the latter seems more probable. The early stages of the disease may be characterised by lesions involving the integument of the limb or the gastro-intestinal tract. Whichever it is, there is little that is significant in an urticarial rash, a papular lesion or a transitory gastro-intestinal catarrh to suggest the possibility of a schistosoma infection having been contracted. Occasional loose feaces associated with some inappetence can be interpreted as mere indigestion, and premonitory symptoms such as an intermittent high temperature are likely to be dismissed with a palliative or symptomatic treatment. The occurrence of such minor ailments will scarcely be recalled at the time when the disease has made good headway, and when the affected animals (of which generally there are several) begin to show signs of lethargy and unthriftiness for no apparent reason, examination of feaces or of stomach contents collected by lavage is usually carried out. Nematode worms or their ova in varying numbers may be detected and anthelminthic or vermifuge measures adopted accordingly. Although some transitory benefits may be seen, it is soon realised that the progress of unthriftiness has not been checked. Icterus and anaemia develop gradually but the suspicion of biliary fever is not confirmed by blood smear examination. Chronic insidious conditions like subacute glanders or tuberculosis are also excluded as a result of tests with appropriate biological reagents. A vigorous tonic treatment and liberal allowance of nutritious rations are prescribed but the animal is found to be getting worse in spite of all human efforts to the contrary. Varying amounts of dropsical transudation common to conditions of debility may accumulate in the dependent parts and this explains the entry of attacks of lymphangitis or swollen legs made in the Veterinary History sheets of some of these cases. Exudation of fluid into the peritoneal cavity also takes place but in the normally obese abdomen this escapes notice. Part of it undergoes reabsorption but increased exudation continues and with the progressive enlargement of the liver abdomen becomes pendulous. This appearance becomes more pronounced because of advancing general emaciation. The enlarged liver may be palpated per rectum and found to occupy considerable portions of the abdominal cavity. On manipulation the animal may evince pain. The back muscles are greatly atrophied and when the liver has undergone enormous enlargement, the patient has the appearance of a gravid mare, to employ the expression used by Williams [1014]. With the extension of the malady, circumscribed multiple foci of
the disease affect various internal organs, e.g., intestine, liver, lung, spleen, pancreas, etc. The pressure exerted by the enlarged liver and the reduced functional activity of the affected organs lead to metabolic disturbances, interfering with the absorption and digestion of food, respiration and circulation. A cough of varying intensity and recurring attacks of diarrhoea are to be expected.

In connection with the above attempt to describe the probable course of a typical case of this disease, it will be of interest to study some actual photographs (Figs. 613 and 614) of a Chinese boy suffering from chronic Schistosomiasis of the Far East, as given by Byam and Archibald [1923] in their notable treatise on the "Practice of Medicine in the Tropics". It will be noted that the boy has undergone an extreme form of emaciation and weakness, the abdomen shows an upper distension and the feet are dropsical.

With regard to the geographical distribution of the condition, it appears to be mainly restricted to the northern parts of India, though specimens have been received from places as far south as Secunderabad and as far east as Muzaffarpur in Bihar. Each of the different species of the equine tribe appears to be equally affected and it is generally the older animals, which suffer most.

**MORBID ANATOMY.**

Liver.—The changes in this organ should be considered first, since this is the seat of the most easily recognisable gross changes, but before taking up a consideration of the hepatic changes peculiar to this disease, it is desirable to refer to certain peculiarities of the equine liver. The horse is not provided with a gall bladder and hence it is rarely the subject of any biliary concretions. Infection with flukes is not common and it is only rarely that cerebrospinal growths are encountered in this organ of the horse. Yet the size, weight, colour and consistency of the organ vary in quite a number of cases for no apparent cause. To quote McFadyean [1889], the horse appears to be more frequently the subject of hepatic cirrhosis than any of the other domesticated animals. In the disease to be described here, the surface of the organ is comparatively smooth in the greater part of its extent, and pale whitish patches or strings of fibrous tissue may be seen on the capsule as an evidence of appreciable perihepatitis possibly supervening on ascites or peritonitis. The organ is invariably enlarged and is much heavier than normal. The weight ranges from 15 lbs. to 90 lbs., the latter figure being recorded by Williams [1914]. The heaviest liver from which portions have been forwarded to this Institute was stated to be 75 lbs. Generally the shape of the lobes is maintained though one lobe may be a little more enlarged than the other and at no stage has any atrophy of the liver been noticed, contrary to what is known to occur in "hobnail" liver.
Numerous minute greyish white or chalky white nodules resembling miliary tubercles can be seen immediately beneath the transparent capsule (Plate I, specimen Nos. 1532 and 562 iii). These nodules are more or less calcified, as large as a sago grain, but rarely, they may attain the size of a split pea. Palpation of the surface gives a slightly irregular feel, but the organ is never characteristically 'hobnailled' even in an extreme case. Occasionally several advanced nodules may be seen crowded together over the surface of the organ (Plate I, specimen No. 750-B). In rare cases elevated nodular areas of induration may also be seen protruding in a like manner (Plate I, specimen No. 993). Degenerative softening of the nodules has never been seen to be associated with the condition. The organ cuts with a gritty or cartilaginous feel, and then will be seen many more minute nodules which are red in the early stages but vary from greyish or greyish white to pearl white in colour as the disease advances (Plate I, specimen No. 1553 left). If an individual nodule is removed with the point of a knife and a caustic potash preparation of the crushed material be examined under the microscope one or two oval, spined eggs may be seen. The cut surfaces present a coarse network of grey or greyish white bands of fibrous tissue of varying breadth, enclosing in each mesh rounded islets of yellow or yellowish brown hepatic tissue. When the disease has advanced further, the linear tracts of connective tissue become so thickened and prominent as to present a marbled appearance or a "claypipe stem" picture due to thickened bile ducts as delineated by Symmers [1903] in cases of Egyptian Schistosomiasis (caused by S. mansoni). The liver may be so densely packed with the nodules in varying stages of calcification and organisation that very little true liver tissue can be recognised by the naked eye. The old nodules may be so highly calcified that they readily become enucleated on the slightest disturbance with the knife (Plate II, Fig. 1).

The development of the lesion in this equine affection takes place in the same manner as in the case of other forms of schistosomiasis. There are certain differences, of course, but they may be ascribed to the different degrees of pathogenicity of the species of the parasite concerned and probably also due to the varying powers of resistance possessed by the invaded equine tissues. The essential lesion of the disease is a nodulated cirrhosis, practically restricted to the portal tract in the initial stage (Plate III, Fig. 4). The nodule in its earliest stage is mainly cellular in character, being composed of a heavy accumulation of mononuclear neutrophile cells and rare polymorphonuclears which again are loosely surrounded by a few delicate fibrils of connective tissue (Plate III, Fig. 2). With regard to the origin of these fibrils careful examination of sections has shown that they are merely the normal supporting tissue which has become arranged around the irritant in order that it may be phagocytised in situ. The offending
For explanation see page 27.
For explanation see page 27.
ovum lies quite free in the centre and it may be mentioned here that during these studies sections of the female worm also have been seen in the centre of these nodules in one sample of tissue only (Plate IV, Fig. 4). With the infiltration of eosinophile leucocytes becoming denser in the surrounding areas, the formation of rings of younger connective tissue takes place from the periphery inwards, the earlier formed strands having attained maturity in the meantime. As the nodule proceeds to further development, the ovum may still continue to lie freely in a central cavity of the nodule or a kind of fibrinous, eosinophile, degeneration 'lymph' may accumulate around it. In the latter case, a varying number of wandering cells with pyknotic and fragmented nuclear material is recognisable in this pale reddish mass (Plate III, Fig. 3). The amount of the degenerated cellular debris lying in the eosinophile substance may be considerable depending upon the extent of toxic material elaborated by the developing embryo from inside the egg-shell and this amorphous substance generally forms an irregular area around the ovum. Some eosinophile leucocytes may reach this area of degenerated material and a few others are seen lying in between the circular rings of more or less developed fibrous connective tissue, but the densest eosinophile concentration continues to be in the immediate proximity of the fibrous capsule of an individual nodule, but always outside the latter (Plate III, Fig. 2). Phagocytic activity around the central amorphous mass manifests itself and fibrin-like threads are seen radiating out in a loose manner. The phagocytic cells are then seen to assume a radial arrangement, with the broader parts of the cells directed outside (Plate IV, Fig. 3). In some nodules the arrangement may be so well developed as to suggest a relationship with the classical "ray fungus". In the opinion of Shafi Mohammad [1931] these radiating and nucleated filaments are giant cells which have been flattened out and elongated due to the pressure and counterpressure exerted by the formation of concentric rings of fibrous tissue and this view is in conformity with the findings recorded here. With further organisation, the radial arrangement of elongated cells is lost and more or less successful attempts at giant cell formation become manifest. Commencement of calcification begins immediately around the ovum, the cellular contents of which seem to have lost the character of viability, the egg-shell having undergone a partial crenation and blackening. Lime salts have been seen to be deposited in the concentric rings of connective tissue (Plate IV, Fig. 1). [It may be parenthetically mentioned here that the same degree of predisposition to calcareous degeneration of the liver has not been observed in similar bilharzial conditions which occur among human beings in Egypt and the Far East]. The deposition of lime salt progresses with further organisation, fibrosis and giant cell activity (Plate IV, Fig. 2) until in the completely fibroed nodule, nothing excepting a part of the disrupted egg-shell can be seen
Fig. 14 lobules sections. The veins spontaneous involvement portal hyalinised with very inflammatory inter vessels may be present in the endothelial cells of the organ or it may lie free in the endothelial spaces. Apart from the nodular lesions, the portal spaces reveal a considerable increase of newly formed, tortuous bile ducts and connective tissue. It is interesting to note that in a very chronic case of the disease, actual columns of liver cells in different stages of regeneration with the provision of a fine system of capillary circulation in between the rows of hepatic cells have been seen in the centre of a nodular lesion, the outer rings of the fibrous capsule being completely absorbed (Plate VI, Figs. 3 and 4). This is presumably an instance of a regenerating nodule attempting to set up liver function after the toxic products and irritant ovum have been completely removed. In parenthesis, it may be said that retrogressive changes in the capsule itself have also been described by Theiler [1918], who states "Finally the capsule had completely disappeared whilst the necrotic centre had increased and the nodule was surrounded by a thin fibrillar envelope". It has already been stated that the initial lesions are practically restricted to the portal tract, the lobules themselves being singularly free from any abnormality. However, lobular changes may be seen at a very advanced stage of the disease. In this, a single ovum generally is seen lying at some distance from the larger veins or an occasional egg may lie near a small vein. Careful examination of these lobular lesions fails to reveal any evidence of the muscular remnants of a broken down vessel. From this, it seems reasonable to suppose that the lobular nodules usually originate in the finer capillaries and that the delicate walls of these are not recognisable because of the changes which are brought on by the pathological process. Nevertheless, it is not unlikely that now and again an ovum does pierce through the vessel wall by means of its sharp spine and finds itself in a perivascular situation. It may be mentioned here that no ovum has been detected as actually piercing its way through the wall of a blood vessel in spite of a careful examination of a very large number of stained sections. The inter-lobular lesions may extend both in size and number and thus come to occupy the position of the lobules, so much so that when the disease process is severe, whole lobules may be altogether obliterated due to atrophy of liver cells and their replacement by inflammatory tissue and proliferated bile ducts. But it is remarkable that the liver
cells generally remain healthy for an extremely long period. It has been seen earlier that the liver undergoes a very pronounced hypertrophy in size but in the strict sense, the term hypertrophic cirrhosis would scarcely apply to this condition since an actual hypertrophic increase of liver cells has not been detected in these studies. In seeking to find an explanation for the presence of the most advanced and the earliest lesions side by side in the same liver, one is inclined to the view that the ova have been carried to the liver at different times from larger veins supplying other viscera, in particular the intestines. The obliteration and occlusion of the majority of the portal vessels, the extent of which can be appreciated from Plate II, Fig. 1, would scarcely afford sufficient room for a migratory gravid female to lay eggs in situ and then to escape.

As regards the ovum which is found in these lesions, it is a typical Schistosome ovum, with a terminal spine, and as will be seen later it agrees in size with the limits laid down for the ova of S. indicum, Montgomery. The egg-shell is composed of a peculiar chitinous material, which is highly resistant to the phagocytic action of body cells but the developing miracidium is readily destroyed by the phagocytes, which appear to have no difficulty in obtaining entrance into the shell-cavity. Although the egg-shell is soft enough to lose its characteristic shape or contour, the persistence of disrupted remnants with a betraying spine in the centre of an altogether fibrosed nodule, is a very striking and common occurrence (Plate IV, Fig. 1). Another remarkable finding has been the persistence of a couple of eggs side by side in a centre of dense calcification, there being no change in the shape of the eggshells, contrary to the usual crenation or disruption that is generally met with. Of the dozen or more ova that have been measured from sections of the liver, the largest was found to have 0.128×0.040 mm. as the dimensions, whereas the length of a number of eggs varied from 0.034 to 0.094 mm. and the width from 0.031 to 0.041 mm. The eggs seen in sections of intestine were of similar sizes but a few small eggs, having dimensions like 0.081×0.012 mm. or 0.069×0.021 mm. have been seen. The small sizes probably refer to the angle at which they were cut. An egg in a lung section measured 0.85×0.033 mm. and one from the mesenteric gland was 0.79×0.027 mm. The length of the ova of S. indicum measured by Montgomery varied from 0.092 to 0.10 mm., the width from 0.042 to 0.044 mm.

Intestines.—Although these bilharzial lesions are generally restricted to the large intestine, they may encroach upon the small intestine and in rare advanced cases, even upon the stomach. The appearance of the lesions involving either the mucous or the peritoneal surface is practically the same in the different parts of the digestive tract but in the majority of cases both surfaces are equally affected. In fact, if one examines carefully the thickness of the gut wall, no layer is found to be
particularly free from the lesions (Plate II, Fig. 2), though in very early cases the venules in the submucous coat are the only seats where the pseudotubercles are produced. In so far as the stomach is concerned, one must remember that certain species of Habronema worms are quite frequently encountered there either lying free on areas of superficial ulceration or embedded in fistulous tumours. As has been seen already, the initial lesions start in the submucous bed of blood vessels of the large intestines and it is the distal portions, namely, the large colon and the rectum which reveal the earliest gross lesions, which again may easily be overlooked unless one is particularly careful. Unless one is familiar with the minor lesions, commonly associated with the early stages of a bilharzial enteritis, such as slight congestion or petechial haemorrhage, the proper significance of the picture may not be understood. The intestinal contents may be semisolid or soft, coated with mucus and slightly streaked with blood. As with other diseased conditions the character and the extent of the lesions depend upon the duration and the severity of the infection but neoplastic growths and tendency to malignancy, as are commonly associated with certain forms of intestinal bilharziosis, are not seen in this equine disease. The lesions on the mucous membrane manifest themselves as a special type of ulcer, which is generally minute and discrete with a sharply circumscribed margin. The individual ulcers are rarely larger than the size of a big pinhead but occasionally when neighbouring ulcers coalesce they may be a little larger. Another peculiarity of the ulcers is that their surface is covered with a black deposit, due probably to extravasated blood. The pigmented surface when present makes detection easier and it is through these peculiar and characteristic ulcers that the worm ova are extruded into the outside world (Plate I, Specimen No. 900). The character of the lesions, as seen from an examination of the peritoneal covering of the gut is singular in that exceedingly hard nodular masses stand out from the bowel wall, the lesions showing varying degrees of fibrosis and calcification. The nodules may be discrete or appear as conglomerate excrescences, the size of the individual nodules averaging that of a pea (Plate I, Specimens 562 ii & 1931). In such portions of the colon as are not excessively thickened and where the margins of the individual nodules are still recognisable, it will be found that the lesions are generally restricted to the colic vessels. Other very noticeable lesions of the peritoneal surface of the gut are the thrombosed veins which stand out as rigid linear elevations and contain an almost black antemortem thrombus. Smith [1891] stated that arteries of the colon were converted into rigid tubes due to lime deposit in their coats and that the change was not the result of parasitic action. This view is not supported by the facts since lime salts are deposited around the parasitic ovum in the centre of a thrombosed vessel, and to start with, the rigidity of the arterial tubes is due to thrombosis only. The thickening of the intestinal wall due
PLATE V.

Fig. 1. (x45)

Fig. 2. (x57)

Fig. 3. (x27)

Fig. 4. (x21)

For explanation see page 27.
to chronic inflammatory changes may be so great that the bowel loses most of its pliability and scarcely any light can pass through it. Individual pseudotubercles, which may be situated anywhere between the mucous and peritoneal surfaces, show two or three sharply defined circles. (Plate II, Fig. 2). The outer circle is fibrous and the central one contains a core of degenerated and calcareous material. If a portion of the bowel showing early lesions is examined against a strong light, one should be able to detect commencing vascular lesions from the centre of some of which, the worm may be enucleated without much difficulty.

The mucous membrane of the intestine shows a marked mucoid degeneration in a considerable portion of its length and this is associated with a profound infiltration with eosinophile leucocytes. It is strange that very few ova or none at all are detected in the mucosa and from this, it seems probable that an extensive diffusion of parasitic toxin must be responsible for these evenly distributed changes. Small localised areas of inflammatory exudate or actual abscess formation may be seen to have originated around half a dozen or more ova. The miracidium can be seen inside the egg-shell which has undergone an irregular crenation. Actual ulceration takes place by the desquamation of the epithelial cells, leading to the formation of the peculiar minute ulcers described above and a varying amount of blood pigment is deposited here and there. The muscular layer of the mucosa is intact practically throughout excepting for small gaps due to the dilation of the arterioles which run between the mucosa and the submucosa (Plate VI, Fig. 1). Actual breach of the muscularis mucosa takes place as a result of the disease process advancing from the submucosa upwards and thus allowing the ova with the enclosed maturing miracidia to be extruded into the outside world (Plate V, Fig. 1). As has been seen before, the essential pathological lesions of the disease are found around the ova. In early cases, it is the submucous coat only which provides the seat for the production of the well known pseudotubercles [Fairley, 1920] and in an advanced case, this layer is again the seat of the largest number of these nodular lesions. The lesions extend to the subserosa (Plate V, Fig. 3) or in an extreme case may be seen anywhere, but their formation is generally restricted to those situations which have the most luxuriant blood supply. The nodules have been seen in between the longitudinal layers of the muscular coat, these having been formed inside the capillaries and venules which lie in strands of supporting intramuscular connective tissue (Plate V, Fig. 4). The nodular lesions in the intestine follow the same sequence of developmental stages as is seen in the case of similar lesions of the liver. The blood vessels situated either in the submucosa or deeper are more or less engorged and dilated. Now and again extremely dilated areas, rounded or oval in shape, bounded by very thin walls and containing small round cells are seen immediately below the muscularis mucosa. With regard to the origin of these, it
seems that the majority are capillaries or venules which have been abnormally dilated but these may include occasional dilated lymph spaces. Apart from certain large sized vessels which reveal a marked thinning and disruption of the muscular coat, the most interesting lesions are presented by the endothelium of the smaller vessels. The lesions consist of varying degrees of exudation into the endothelial lining so that a sector of the vessel may become swollen to form a blunt finger-like process filling a major portion of the lumen, whereas the endothelium of the other sectors is very severely mutilated or are even actually shed (Plate VI, Fig. 2). Where the vessel is seen to contain a plug of inflammatory exudate, portions of the desquamated endothelium may be recognisable as forming a part of it. With regard to the probable mechanism by which the bloating of the endothelium takes place, one notices polymorphonuclear leucocytes and masses of eosinophiles inside the stumpy finger-like process. The fact that the immediate vicinity of the vessel wall at the affected sector is heavily crowded with eosinophile cells suggests that a factor such as an excessive lowering of blood pressure may have determined the endothelial disruption, particularly when the parasitic toxin had already affected the susceptible intima. Fairley [1920] has failed to confirm the finding of an endophlebitis of the mesenteric veins as described by Letulle [1905] in his comprehensive article on intestinal blihaziosis. The other prominent lesion of the blood vessels is represented by degrees of endarteritis obliterans.

Apart from the pathological changes in the blood vessels supplying the intestine, which have been referred to above, it must be remembered that the majority of the nodular lesions in the submucosa or the subserosa originate in the embolic egg masses which form in the vessels of the place (Plate V, Fig. 2). It may be mentioned here that the writer has encountered more than once up to 20 ova in a single embolic focus in the intestine but although hundreds of liver sections likewise have been examined, he has never been able to find more than one ovum in an individual nodule in the liver. Another remarkable fact is that in the equine intestine, the large majority of Schistosome ova have been seen to be wholly restricted to the layers of tissue situated below the mucosa (Plate II, Fig. 2), whereas in the histological sections prepared from the intestine of natural cases of S. indigum infection in the sheep and in the bull of both the plains and hill breeds, the ova have been found almost exclusively in the mucous membrane with practically no tendency to nodule formation, the actual lesion being more cellular than fibrous and not calcareous at all. Montgomery [1906] states that the large intestine of equines is the only organ where ova of S. indigum could be found present in any numbers. He found the ova in the mucosa only and thought that they must occupy the submucous tissue or the capillary at some time.
For explanation see page 27.
Mesenteric and other lymphatic glands.—These glands react to schistosome infection in very much the same way as noted in the foregoing description of tissue changes, the differences depending upon their anatomical relationship and structural peculiarities. Examined with the naked eye, the glands are generally enlarged, oedematous and hemorrhagic. The enlargement may take place in the region of the hilus only or it may involve the whole gland, depending upon the severity of the infection. Palpation of the affected region of the gland gives a hard nodular feeling to the fingers, the healthy portions yielding normally to light pressure. On incising an affected gland, pseudotubercles in varying stages of fibrosis and calcareous degeneration are encountered throughout the gland. On microscopic examination the vessels and plexus of lymphatics situated under the capsule are found dilated. The lacunar system of lymph sinuses is infiltrated densely with eosinophiles but the lymph-follicular tissue is practically unchanged excepting for areas where it is directly squashed out due to the pressure exerted by the growing nodular lesions, which latter have the same histological features, as those described in connection with the liver. In advanced lesions, however, it is not possible to say if the lymph follicles, the sinusoids or the vessels enclosed in the supporting strands of trabecule had formed the initial seat for the developing lesions. The presence of the typical Schistosome ova in the centre of the pseudotubercles establishes the nature of the pathogenic agent. Further some glands may reveal only an endarteritis obliterans, eosinophilia and a hemorrhagic appearance. Whether the mesenteric, portal, mediastinal or the bronchial glands are involved, the predisposition to the deposition of calcareous salts in the lesions is very marked. One portal gland was encountered with such extensive calcification that the normal glandular structure was practically lost. The naked eye picture of the nodules was however very typical.

Lungs.—Helminthic nodules in the equine lungs have received a considerable attention due to their importance in the differential diagnosis of glanders, and the work of Angeloff [1907], Olt [1910] and Theiler [1918] in this connection are noteworthy. In the case of the latter worker, it is unfortunate that no particular attempt was made to prove the presence of worms in cases where eosinophilia was noted. Although a cysticercus or a nematode was suspected Theiler states that in the majority of lesions no parasites were found. In the figures presented by Theiler [1918] and by Knowles and Slocock [1931], nodules in the lungs are more frequent than those in the liver, whereas the present investigation has brought out the greater frequency of nodules in the latter organ and this agrees with Lingard’s [1905] experience too. Nematodes have been suspected by the workers in South Africa and Egypt and the frequency of liver lesions in India is to be expected from a knowledge of the other forms of intestinal schistosomiasis. It
is now known that the lungs may be the seat of lesions caused by the migration of the larvae of *Ascaris megaloccephala* [Hobmaier, 1925], of Strongyles [Olt, 1910 and Theiler, 1918] and of Habronema [Dieulouard, 1927], but the possibility of Schistosomes playing a role in the production of lung lesions, the importance of which in human affections has been emphasised by Turner [1909], has not so far been considered in Veterinary Pathology. During the investigation under report, lesions on the pleura and the lung parenchyma have been detected quite frequently. Patches of chronic pleuritis, gram-sized sub-pleural nodules (Plate I, specimen 1481) or highly calcareous, nodular aggregations on the pleura (Plate I, specimen 750-A) have been seen. The anterior lobes of the lung may be united along their borders. Lung parenchyma may show patches of consolidation, with organised and very sharply defined nodules of rounded shape distributed throughout. Their size may reach that of a green pea (Plate I, specimen 1562) and they never show any casinous softening. Examined under the microscope, the lung shows the same characteristic features in the organised nodules, as seen in the other organs. The nodules consist of an inner area of degenerated cell debris with or without calcification, surrounded by a more or less strong fibrous fortification (Plate II, Fig. 3). The nodules appear to form in the vessels of the intervesicular septa and from the smallest to the largest the nodules maintain a perfectly rounded shape in the different stages of fibrosis. A chronic catarrhal type of pneumonia, in which the air vesicles are seen to contain eosinophiles and some undifferentiated coagulum—probably serum, may affect only portions of the parenchyma or may involve the whole organ. In passing, it may be mentioned here that the lung lesions of a nematode infection can be distinguished without much difficulty from the specific lesions of bilharziosis in the majority of instances. The former is usually a bronchial or peribronchial affection and the lesion covers a much larger area. This is understandable since the causative agent is a worm larva, which according to Theiler [1918], reaches its place by way of the bronchi. In this connection, it will be remembered that a reference has been made already in the introductory remarks concerning the two types of lesions of the lungs encountered by Lingard [1905]. He noticed that the larger variety of lung lesions was less frequent and this finding is in accord with those of the writer. Nematode lesions are generally few and they are seldom organised when small. Even in a larger lesion, the concentric arrangement is not so well developed, for there may be only a few rings of irregularly circular connective tissue. The bilharzial lesions, on the other hand, are generally uniformly distributed, being formed as a result of an embolism in the capillaries of the interalveolar septa. As the lesion is generally formed around a single Schistosome ovum in a capillary, the resulting nodule or the degenerated eosinophile debris in its centre is much smaller and yet the rings of fibrosis are well developed.
There may be little or no bronchial or peribronchial reaction. In order to explain why the bilharzial lesions in the lung are not more numerous, it seems justifiable to presume that some of the developing lesions abort prematurely due to the rupture of the interalveolar septa which are structurally very fragile. Nematode lesions again do not appear to be so predisposed to the deposition of calcareous salts. But the most easily recognisable difference in the character of the two types of lesions is the very definite and abrupt margin which invariably encircles the individual lesion of bilharzial origin, whereas the boundary of a nematode lesion can scarcely be deciphered by the naked eye since there is a very gradual transition from the obviously diseased to the suspiciously healthy zones. A rare nematode lesion may resemble one of bilharzial origin, but the latter is so definite that a microscopic examination is seldom necessary for the purpose of distinguishing it from the former (Plate II, Fig. 3). This remark holds true for lesions in any organ.

Spleen.—A moderate enlargement and hyperæmia may be the only lesions in this organ and they are due to portal stasis. As a result of diffusion of toxic products, there may be an increase in the supporting tissue. Areas of infarction are frequent and they may be as small as a silver two-anna coin. Actual nodules are occasionally seen and in extreme cases the organ may be permeated with them. Brown granules of blood pigment and eosinophiles may be seen here and there.

Peritoneal cavity.—Evidence of ascitic fluid may or may not be present. In cases where it is present, it may be a serous flocculent fluid or a fluid streaked with blood.

**Diagnosis.**

The problem of arriving at a correct diagnosis of this condition in life is beset with certain difficulties. Information on its geographical distribution and the clinical history of a case are useful adjuncts but an assured diagnosis rests upon the discovery of the specific ova in the faeces. As the ova may not appear with regularity in the faeces until the case is advanced, faecal examination on more than one occasion should be carried out. Generally it is the flecks of mucus, which may or may not be blood stained, that contain the ova, singly or in clusters. Since Schistosome ova are heavy and settle down either on standing or on the faeces being centrifuged, the sugar floatation methods of diagnosis are not suitable. The sample of faeces therefore, is diluted with 10 to 20 times its volumes of water and the solution passed through a fine sieve to eliminate the coarse grains and fibres of vegetable matter. The filtrate is then centrifuged or allowed to settle for 24 hours. Coverslip preparations of fresh smears made from the sediment can be examined directly under the low power of
the microscope or if the smear shows a considerable amount of animal matter, a couple of drops of 5 per cent. caustic potash may be added to the smear to facilitate examination. A microphotograph of ova of *S. indicum* is to be found in Plate II, Fig. 4 and this has been taken from a coverslip preparation obtained from a sample of bovine faeces.

The appearances to be expected at *post-mortem* examination have already been referred to, and for those who have not the opportunity of studying microtome sections, the following methods of obtaining confirmation of a Schistosome infection may be adopted:—

(a) Ferguson's [1911]—Digestion method. The ova can be conveniently demonstrated in the tissues by digesting selected portions in 3 per cent. or 4 per cent. caustic potash solution.

(b) Fairley’s [1920] direct method. Large areas of the intestinal mucosa can be examined directly under the microscope by fixing comparatively large portions of the intestinal wall between two glass slides, which are kept in position by means of rubber bands. This method is not suitable for highly thickened intestinal wall.

(c) Collection of the worms in copula or singly, from the portal blood or by careful dissection of the smaller branches of the vessels of the rectal mesentery, which is the most frequent site.

**Methods of Control.**

Since the parasites are located in the vascular system, ordinary anthelmintics do not reach them. Specific treatment with intravenous administration of tartar emetic, emetin and antimosan is indicated. As treated animals are likely to be reinfected, it is necessary to take steps for the prevention of infection. Attempts should be made to destroy the parasite in some stage of its life cycle. Snail-infested land should be treated with lime or dilute copper sulphate. Thorough hygienic disposal of the excreta of schistosome-infected goats, sheep, bulls or equines should be carried out, and in this connection it should be noted that Montgomery discovered *S. indicum* in horses, donkeys and sheep at Muktesar, Baldrey [1906] found similar ova in sheep at Lahore, and Fairley and his coworkers [1930] found *S. indicum* infection in goats. Recently Mr. V. R. Rajagopalan, one of the writer’s assistants, has collected the same parasite from (1) a hill bull at Muktesar, (2) a specimen of sheep’s intestine from Sind, and (3) the intestine of a plains bull, suspected of Johne’s disease at Mona.
It will be noticed that in considering the morbid lesions of this form of equine Schistosomiasis precedence has been given to the liver. The extreme form of hypertrophy which the organ undergoes under action of the disease and its easily recognisable gross changes have been noted. The condition has been designated as a nodulated portal cirrhosis since those are the typical histological features of the lesions. From the strict scientific standpoint, however, the disease is an intestinal schistosomiasis, since it is by the involvement of the bowels only that the parasite is enabled to attain the fulfilment of its life-history. In regard to the liver lesions, it is of interest to note that Manson [1902] records the occasional presence of small numbers of schistosome ova in the liver of men but refers to no pathological lesions caused by them, and Scheube [1903] records the finding of ova in a man’s liver with slight cirrhosis. Credit is due to Symmers [1903] for being the first to record the finding of china-white nodules of sizes varying from a pin-point to a split pea, periportal fibrosis and for describing extensive lesions of “claypipe stem” cirrhosis of the liver. Miliary tubercle-like nodules were encountered by Katsurada [1905] and since then the specific liver lesions of schistosomiasis have received considerable attention in the publications of Wooley [1906], Phalen and Nichols [1908], Letulle and Nattan-Larrier [1909], Houghton [1910], Jouveau-Dubreil [1913], Lampe [1925] and others. Reference must also be made to Montgomery [1906] who obtained the majority of his collection of S. indiwm from the equine liver after its removal from the body, from the pancreatic veins in two cases and from the mesenteric vessels of all the eight cases examined. Although he noticed the liver to be enlarged and, in nearly all instances, to be studded with small pearl-like calcareous nodules, sometimes as large as a pea, well defined by a fibrous capsule and easily enucleated, he considered them to be different from those described by Symmers [1903] but possibly of the same origin as Mazzanti’s filarial lesions, referred to by Lingard [1905]. With regard to “pipe stem” cirrhosis, Montgomery went on to say that “in equines this does not appear to be present, though in one case a similar naked eye condition existed, but in the lesions ova could not be found.” An explanation of the persistent failure of the writer to find adult schistosomes in the histological sections (excepting a single notable case mentioned already) would seem to be that the worms inhabit the larger branches of the portal trunk whereas the nodules were situated in the finer ramifications only and further that the worms may have escaped with the portal blood during post-mortem examination. As regards the consistent failure of Montgomery to record any definite lesions, in spite of the presence of numbers of the parasite, his own view that the animals examined had but recently become
affected is probably partly true, and in other cases there may have been some resistance on the part of the host. The occurrence of a severe intractable debility as a prominent symptom in this equine schistosomiasis has been emphasised earlier in this paper. Hodgkins [1920], Sewell [1921], Allen [1923] and Steevenson [1924 and 1930] have recorded certain experimental data obtained by them in their attempt to elucidate the problem of persistent debility amongst army horses in India and there is little doubt that a number of these cases are due to habronemiasis or strongylosis. But on the other hand, it has been shown above that on further investigation a number of such cases occurring amongst army horses are in reality a schistosomiasis. It is not possible to say with certainty at this stage what proportion of the cases of persistent debility are due to each of these three causes, but a preliminary survey of the samples of outside specimens collected at Muktesar since 1921 has revealed only but half a dozen cases of lesions in the liver and lungs which can be ascribed to nematode larvae, as compared with a large number of cases now proved to be bilharzial. With regard to gastric habronemiasis, however, the worms and the stomach lesions caused by them are so commonly found in equine subjects in India that probably it would not be wrong to state that as many as 95 per cent. of animals harbour these parasites, but it is probable that in the majority of those cases without stomach lesions little harm results. This statement would seem warranted in view of the findings in Egypt by Knowles and Slocock [1931] that 86 per cent. of animals infected with Habronema were in good or fair condition. On the whole, therefore, it appears that the conditions described in this paper, due to infection with *S. indicum*, apart from malnutrition, are the most important factor in the causation of persistent debility of equines in India.

Acknowledgements.

The author's thanks are due to Mr. G. D. Bhalerao, M. Sc., Helminthologist, for confirming the specific identity of the worm ova; to Mr. J. Sunder Rao, Artist, for the prompt execution of the photographic illustrations. Further the author is greatly indebted to his chief Mr. F. Ware, F.R.C.V.S., I.V.S., for evincing keen interest and for granting every facility for the present investigation.

References.


—— —— *Vet. J. 77*, 300-301.

**DESCRIPTION OF ILLUSTRATIONS.**

**Plate I.**—Macroscopic lesions three-fourths the natural size.

**Liver.**—Specimen No. 1532 shows a number of the specific pseudotubercles through the transparent capsule. The majority of the nodules are discrete but a few are coalesced.

Specimen No. 563 iii.—Liver capsule shows 'miliary' pseudotubercles.

Specimen No. 750-B.—A conglomeration of calcareous, pea-sized nodules has produced an elevation of the hepatic surface.

Specimen No. 1558.—Section through the liver parenchyma shows numerous pearl-white nodules. Section on the right shows degrees of pericellular fibrosis.

Specimen No. 193 shows hard indurated nodules on the capsule.

**Intestine.**—Specimen Nos. 562, ii & 1931 (left).—Peritoneal surface of the intestine shows distinct nodular excrescences.

Specimen Nos. 1931 (right) & 909 show the intestinal mucosa with the typical minute ulcers characterised by black haemorrhagic surface.

**Lungs.**—Specimen Nos. 1481 & 750-A show the lung pleura, the former affected with a single discrete nodule and the latter with an accumulation of more or less distinct pseudotubercles.

Specimen No. 1562 | Lung parenchyma shows discrete and coalesced nodules.

**Plate II.** Fig. 1.—Low power microphotograph of a portion of diseased liver showing how the organ is packed with nodules in various stages. One or more circular rings can be seen in the central pith of each nodule. Empty spaces due to the central degenerated area having fallen out are seen also.

Fig. 2.—Low power microphotograph of a portion of affected intestine. Varying sizes of the nodular lesions are seen involving all the coats with a preponderance of lesions in the submucosa and subserosa. The mucosa appears to be more or less intact.
Plate III. Fig. 1.—Microphotograph of a section of liver, showing an organised nodule, the concentric rings of connective tissue of which have undergone partial hyalinisation. The spined ovum shows the embryonal cells within the sharply margined eggshell and is lying free in a cavity.

Fig. 2.—Microphotograph of a very early nodule, showing the commencing arrangement of fibrils of connective tissue around a free lying spined ovum. There are fewer cells in the area enclosed by the fibrils and a very dense accumulation of eosinophile cells is noticed immediately outside the few fibrillar rings.

Fig. 3.—Microphotograph of an organising nodule, around an ovum, the enclosed cells and their nucleus being hazier. The ovum is seen lying in an irregular area of eosinophile degeneration lymph containing broken down nuclear debris. Strings of eosinophile cells are seen arranged on the top left hand of the field, being kept in position by the circular fibrils.

Fig. 4.—Microphotograph of section of liver showing how the nodular lesions are mainly restricted to the portal tract, the lobular cells being singularly healthy. Hepatic vein is seen to form the nucleus for the lesion.

Plate IV. Fig. 1.—Microphotograph of a completely organised nodule, showing deposition of calcium salts in concentric rings. Although the concentric fibres are completely hyalinised and considerable calcification has taken place, the crumpled eggshell still persists with the spine.

Fig. 2.—Microphotograph illustrating the preservation of the typical form of a spined egg notwithstanding calcification and giant cell activity.

Fig. 3.—Microphotograph of an early nodule showing a radiating arrangement of reaction cells, with the broader nuclear portion directed towards the periphery. In the centre is an ovum slightly out of focus.

Fig. 4.—Microphotograph of an active lesion in the liver showing three sections of what is apparently a female worm.

Plate V. Fig. 1.—Microphotograph—Section of intestine shows an abscess on the mucosa, degeneration of the muscularis mucosa and the active lesion in the submucosa. The mucosal lesion is about to be shed.

Fig. 2.—Microphotograph showing a cluster of seven eggs forming the nucleus of an intestinal lesion.

Fig. 3.—Microphotograph of nodules in different stages in the subserosa.

Fig. 4.—Microphotograph showing the distribution of four lesions situated in the connective tissue strands in between the longitudinal layers of the muscular coat of the intestine.

Plate VI. Fig. 1.—Microphotograph—Extremely dilated capillary is seen passing from the submucosa to the mucosa, separating widely portions of the muscularis mucosa.

Fig. 2.—Microphotograph—A thumb-like process formed by bloated endothelium of a vessel can be seen containing a heavy accumulation of eosinophile cells.
Fig. 3.—Microphotograph of a nodule, after irritant ovum, calcification, etc., have been removed. Note the absence of capsule and developing columns of liver cells in the lighter stained centre.

Fig. 4.—Microphotograph of the developing columns (shown in Fig. 3 above). Note the beautifully arranged columns of the specific cells with distinct nuclei, the arrangement being around young endothelial vessels. A comparison of this picture of a regenerating nodule with the different stages of degeneration shown in Plates III and IV will show readily the differential characters.
REPRINTED FROM
THE INDIAN JOURNAL OF VETERINARY SCIENCE
AND ANIMAL HUSBANDRY.

THE DIAGNOSIS AND TREATMENT OF STERILITY IN THE
STALLION AND THE BULL

(A Résumé of the literature).

BY

CAPTAIN S. C. A. DATTA, B.Sc., M.R.C.V.S.,
Temporary Veterinary Research Officer, Imperial Institute of Veterinary Research,
Muktesar.

[Received for publication on the 14th July, 1933.]

Of the many problems of importance that the animal clinician has to grapple
with, the determination of the cause of cessation of reproduction or reduced fertility
in the living animal is one of the most perplexing. Variability in the fertility of the
best known thorough-bred stallions and pedigree bulls or their absolute sterility has
inflicted a considerable loss to the breeding industry, but the majority of the
pioneers in this field, notable among whom have been Zschokke, Hess, Albrechtsen,
Wester, Williams, Richter and Quinlan [1929], have occupied themselves almost
exclusively with aspects of sterility in the female animal, and facts about the rôle
played by the male are less familiar. With regard to unproductive marriages in
man, that the male is as important as the woman has only recently come to be
recognised. At the present time, increasing demands for scientific veterinary
knowledge for help against sterility are being made and the problem is also increas-
ing in its scope due to certain recent developments in endocrinology. For the
reasons stated above and because the published literature on sterility in the male
animal is in an unsatisfactory state, the present article has been prepared. It will
be realised that further research and enquiry are urgently required for the elucida-
tion of this problem and it is hoped that this résumé, without any pretension to
being an elaborate or complete one, may lead to this end amongst workers in this
country.

Practical breeders have observed from olden times that all male animals are
not equally effective in impregnating their females and begetting healthy offspring.
Some animals are nearly perfect in this respect while others may require numerous
repetitions in service, even when mated with healthy females, and may even then
produce weak offspring, abortions or retained membranes wherever they are
employed. Sterility in the male may be due to a variety of causes. As Gilman
[1922] has stated environment, diet, endocrine secretions, impediments to coitus,
excessive sexual use, infection and disease of sexual organs have all got a bearing on sterility. It is known that animals that are closely confined, those that are overfat as well as those fed on a deficient ration very frequently fail to bring forth their young. Although the immediate concern of this résumé is what is termed as functional sterility in the male, some of the factors are so interrelated that a few of the more obvious causes, such as pathological lesions, anatomical deformities, infections, metabolic disturbances of dietetic, endocrinal or genetic origin may be briefly touched upon with profit.

The optimum condition for fecundation is that a normal spermatozoon should meet a normal ovum in a normal environment. Any deviation from the optimum condition leads to either temporary or permanent impairment of the reproductive health and fertility of the sires, the diagnosis of which can usually be arrived at from the breeding record, and a careful clinical or laboratory examination either singly or combined. The ultimate value of a breeding sire is estimated by the record of his performance. A clinical examination shows whether his general behaviour, sexual desire, and ability to copulate freely are those of a normal and vigorous male and it is to be expected that given good general care and mated only to healthy females, the usefulness of the efficient sire will be maintained.

Infection.

Conditions such as obesity, muscular weakness or lameness, etc., may act as an impediment to fertilisation apart from actual macroscopic and minute lesions involving the genitalia or accessory organs. But infection appears to be the greatest single factor. According to Williams [1923], "the failure of sixty per cent. of the copulations to produce living young is referable to prenatal death resulting from infection. If the infection kills or mortally injures the spermatozoa in the seminal tubules or elsewhere in the male genitalia, if the infection is added to the semen when ejaculated or later kills the spermatozoa or fertilised ovum, or if the infection exists in the ovary, oviduct or uterus in the female the principle remains the same; it is death from genital infection". Specific lesions of genital infections like dourine, vesicular venereal disease, the venereal granuloma of dogs are transmitted readily between the two sexes in coitus. Cultural examination of the external genitalia of the sire is rendered difficult owing to the risk of contamination with extraneous micro-organisms. The specific organisms of equine and bovine abortion, streptococci, staphylococci, B. coli, B. typhosus, B. tuberculosis and aspergillus have been recovered by workers from the testis, epididymis and seminal vesicles and Williams [1926] records a case where a stallion so thoroughly and uniformly infected his mares that of 19 only 3 conceived, of which one aborted, one foaled a weakling and another produced an apparently healthy foal. Local inflam-
matory conditions such as orchitis, epididymitis as well as infection elsewhere in the body may inhibit or even abolish normal spermatogenesis.

**Hereditary Fertility Factor.**

That sterility may be due to structural malformations has already been mentioned. Gross imperfection of the genitalia, severe hernia and improper descent of the testicles may be the expression of genetic action. According to Crew [1924] grades of fertility are definite racial and breed characters and are transmitted in inheritance and imperfections of the external genital organs are a part of the general conditions of intersexuality and of hermaphroditism, both of which are definitely genetical in nature. Richter [1926] and Wester [1921] lay great emphasis on heredity in the causation of sterility of male goats and bulls. Crew points out that chromosome aberrations lead to complete sterility and offers this as an explanation of the common sterility seen in the offspring of an inter-specific cross, such as a mule. He also brings forward cytological evidence in support of this contention. In the case of another species cross, *Bos americanus* × *Bos taurus*, however, there is probably a lethal factor as this cross is followed by the occurrence of hydramnios during pregnancy resulting in maternal or foetal death.

**Endocrine Factor.**

The correlation of function between the reproductive organs and other glands of internal secretion are well known. Among the endocrines that bear a special relation to sexual development and function are the anterior lobe of the pituitary, the thyroid, the prostate and the adrenal cortex. Disease of the pineal gland may involve premature sexual development. The thymus gland has an inhibitory action on the growth of the sexual organs and becomes markedly reduced at puberty. There is little doubt that some cases of sterility are due to a derangement of this correlation. Hormones have now been extracted from many tissues. Evidence is available to show that the anterior lobe of the pituitary contains at least three hormones—a growth-hormone, a gonad-stimulating hormone and a lactation hormone. Chemically they have not yet been separated. The testicular hormones are complementary to the pituitary hormones and induce sexual excitement and secondary sexual characteristics even in the abnormal absence of the testes.

**Environment.**

This is a minor factor associated with deficiencies in soil and fodder but seasonal and climatic changes present certain stimuli to sexual instinct. Todd and Reynolds [1931] consider that advancing the covering season for getting foals earlier in the year, as has been done in England, has resulted in less fertility of stallions. It is
stated that difficulties have been experienced by most breeders in getting mares in foal until grass is available and this, in Todd's view, is due to the beneficial effects of vitamins and sunlight in stabilising the endocrine balance. Further Sanders [1926] showed that a stallion's fertility varies according to the district of the country in which he stands or travels, being higher in the north and west of England and Wales than in the south and east and very low in Scotland.

Diet.

There is ample evidence that nutritional deficiencies will modify the sexual activity of the parent and of the spermatozoa, thus affecting fertility. Paul [1906] was perhaps the first to show that fertility is affected by diet. Osborne and Mendel [1919] noticed that although animals fed on artificial diets grew vigorously to adult size, they were, with few exceptions sterile. Mason [1928] observed that an increase in breeding capacity is brought about in stalled cattle by turning them out on to green grass. It is known that lack of vitamins B and E in the diet results in the degeneration of the germ cells and the seminal epithelium, although growth and development may proceed in the normal way. With regard to mineral deficiencies of phosphorus and calcium as a cause of sterility, low fertility and retention of placenta in cattle, Kelley [1932] refers to the experience of several colonial and foreign authorities, in particular to Hindmarsh and Hopkirk. The former had experience of a herd in which cattle were frequently sterile; oestrus was exhibited but conception did not take place. During the following year, they were given a regular ration of bone meal with the result that practically all bred on to time. Hopkirk has shown that the so-called Waithi Disease which is characterised by the absence of oestrus is a deficiency condition being amenable to bone meal feeding. Experimental evidence is available from Germany and elsewhere that sperm production is greatly influenced by the feed. A basal ration of 15 pounds of oats and 15 pounds of hay resulted in a sperm production of 2,885,701,420 per ejaculation. When the basal ration was supplemented by 10 hens' eggs and 5 lbs. of wheat bran daily, the sperm production was approximately three times as great. A supplement of dried peas and linseed cake increased the production over that seen with the basal ration to about 50 per cent. only. Other proteins and lipid substances gave results varying between those obtained with the oil cakes and with the eggs. Further experiments show that rats were inactive sexually when fed on whole milk until copper and iron were added in small quantities. Deficiency of iodine also has been proved to act detrimentally on sexual life and fertility. In Australia potassium iodide is therefore added in small quantities to the ration. Another agent which has been found useful in the female is wheat germ oil, but no record of its use in the male has been found. From what has been said above, it will be
seen that a careful examination of the diet of a sterile animal is warranted although he be apparently normal in condition.

**Seminal Pathology.**

Apart from the above factors, examination of the seminal fluid is of great value in arriving at a correct diagnosis of sterility since the spermatogenetic function of the testicle is very sensitive to outside influences. The influence of the frequency of coitus on sperm formation has received considerable attention in Germany. Studies of the quantity and quality of spermatozoa produced by stallions with varying intervals between service indicate that the rest period between services for normal spermatozoa production is 48 hours. Daily matings caused an increase in the percentage of immature sperms. Matings after intervals of more than two days did not result in an increase in the number of viable sperms. Sexual stimulation did not appear to influence sperm production. Complete absence of functional sperms may be due to atresia of the seminiferous tubules, non-development of the interstitial tissue of the testis as in cryptorchidism. Williams and Savage [1925] have published a comprehensive article on the micropathology of bull semen, and by means of the head length measurement of the sperms projected at 3,000 diameters, as recommended by them, Conklin [1930] has been able to detect changes in the spermatozoa, referable to disease of the genitalia, long before a clinical examination would show pathological changes.

**Collection of Seminal Fluid for Examination.**

It is more convenient and easier to collect semen from sires after service than before. It is necessary to observe cleanliness, otherwise presence of smegma, masses of epithelial debris, bacteria, pus, blood cells, and catarrhal discharge may make the examination more difficult. The penis and preputial folds should be thoroughly washed with soap and water and the vaginal cavity douched out with a mild solution of salt or bicarbonate of soda before service is permitted. The seminal fluid may be collected from the vaginal floor of the female with a spatula or glass rod. It may not be necessary to use a vaginal speculum for the purpose. Semen on emission may be caught direct into a beaker or test tube from the tuft of hair at the ventral commissure of the vagina or from the penis by pressure on the urethra of the sire.

**Preparation and Staining of Smears.**

*Wet.*—The vaginal mucus or a drop of the semen from the glass vessel may be smeared on a glass slide and another glass slide or cover slip put on it and examination made straightway under the high power of the microscope. If the
semen is thick it is advisable to mount it in 0.9 per cent. saline solution or in water only.

Dry.—The specimen smear is allowed to dry and it is fixed by passing it over the flame a few times. Care should be taken not to char the specimen by bringing it too near the flame or by holding it too long over it.

(i) The specimen is then stained with methylene blue for 20 minutes, washed rapidly and counter-stained with eosin, washed thoroughly and dried. The film may then be examined under the oil immersion lens of the microscope.

(ii) Another useful stain is carbol fuchsin. It is used hot and placed on the dried and fixed specimen for 10 minutes. The stain is poured off and the slide washed with water and dried and examined as before. This method stains the tail very well but at the expense of some detail in the head and middle part.

(iii) Williams' modification.—The smear is fixed with heat and then the mucus is removed by placing the film in a 0.5 per cent. chlorozene solution for 8—10 minutes. The slide is now washed with clear water (excess water removed by shaking) and stained with carbol fuchsin with heat for 1 to 2 minutes. The carbol fuchsin being washed off with water, the film is stained for 8 minutes with alkaline methylene blue. Then wash off the methylene blue and dry with filter paper. The head of the healthy cell is thus brought out in clear detail, the nucleus remains red and the cytoplasm stains a light blue.

(iv) Jenner's method requires no previous fixation and consists of only a combined stain and fixative. It is a mixture of methylene blue, eosine and methyl alcohol. The specimen may be mounted in xylol-balsam.

EXAMINATION.

The seminal fluid, besides spermatozoa, consists of secretions of epididymis, seminal vesicles, prostrate glands and glands of Cowper. The spermatozoa are present in enormous numbers in every ejaculation. Many millions are present in a single ejaculation of a bull or a stallion.

A fully formed spermatozoon consists of an (i) egg-shaped head, which represents the cell nucleus, (ii) a middle piece or a short cylindrical body—the all-important bright spot, the centrosome, is on the body, (iii) a long tapering tail—it tapers to a point—moving from side to side—the greater the undulation of the tail the greater the vitality of the spermatozoon. Spermatozoa are almost immobile
in the generative tract of the male, they become motile in the secretion of the accessory glands and also in Ringer's solution. Their motility is most vigorous in the normal fluids of a female generative tract.

Spermatozoa in the male passages or female generative tract may retain their vitality and their power of fertilising ova for 30 days or more (rabbit), for 21 days (man). Recently it has been found possible to keep the semen of the male in tubes in a fertile condition outside the body for 40 hours or more, and use it for inseminating females at the end of this period.

To a person familiar with spermatozoa, the appearance is quite certain and definite, but to distinguish a healthy spermatozoon from a weak and diseased one, and to distinguish a perfect one from a malformed one, requires considerable experience and skill.

A simple determination of the presence or absence of sperms in the semen is not enough. The secretion of the vagina is normally acid and is inimical to spermatozoa. If their motility is weak they are not able to continue their progressive movement to reach and enter the cervix uteri. The rate of movement, morphology, structure and staining reactions of the sperm have to be considered for a rational diagnosis.

The popular impression that if the presence of a few motile spermatozoa is demonstrated in the specimen of semen, fertility may be expected is not based on scientific experience [Walker, 1929]. When one recollects the process of impregnation of the ovum as observed microscopically one realises clearly that the fertility of a given animal should be judged not merely by the numerical frequency of spermatozoa and of the motility present, but by a further study of (a) duration and (b) quality of motility, (c) the percentage of the motionless to the actively motile sperms.

Specimens of semen may be collected at different periods after service and the change in the characters of the sperm noticed for judging the vitality of the sperms.

**VITALITY OF THE SPERMATOZOO.**

(a) **Duration of motility** is determined by—

(i) noting the number of hours the motility persists in unmixed semen,

(ii) by the number of hours motility persists in natural media (several secretions of the female tract),

(iii) by the number of hours motility persists in various artificial media.

(b) **Quality of motility** of spermatozoa is, however, a more important criterion for judging the vitality and efficiency of semen [Popa, 1929]. The following is a
brief description of the three different kinds of motility—which follow each other in natural sequence:—

(i) Progressive vibratile motion—is the motion that is first seen in fresh semen under favourable conditions. It consists of a lashing of the after-part of the tail from side to side so rapidly as to constitute a vibration—the undulation of the tail being almost imperceptible. It produces a rapid forward motion in practically a straight line. In the presence of a flow of current this motion takes place against the current. It is particularly in evidence in the vaginal canal.

(ii) The second normal motion differs from the first only in degree of speed. The undulations are plainly perceptible—the direction of motion being determined by the surroundings.

(iii) The third is a slight burrowing motion, as if seeking a mooring place. These kinds of motility are not unlike the movement of the caudal fin of the fish.

In studying the motility of a specimen of semen, there are several other factors which should be considered:—

(a) the time that has elapsed since the semen was ejaculated,
(b) the condition under which it has been kept,
(c) the viscosity of the semen.

Determination of sterility is easier by the character of motility and takes much less time. After the vitality of the sperm has been estimated the defects in their structure and form have to be examined.

(c) Hydrogen-ion concentration of semen is another important factor. Even when all other factors are ideal a mere change in the pH of the semen brings about diminution or entire stoppage of the motility of the spermatozoa. Anderson, Peter and Healey [1922] have shown that the pH of normal horse semen varies from 6.94 to 7.5 and that the average is pH 7.3. The pH from horses known to be sterile varied from 7.49 to 7.76 and the average was 7.58. Nordby has recorded a case of partial sterility in a boar on account of adverse hydrogen-ion concentration in its semen. For the determination of the hydrogen-ion concentration of the seminal fluid, this should be collected direct from the male animal [Donham, Sims and Shaw, 1931].

Defects in the sperms.

Numerous defects in the sperms are met with. Motility may be reduced or be practically absent, duration of motility may be materially shortened from a few hours to a few minutes, the character of motility may be changed from a
progressive vibratile motion to a to and fro lashing movement and the morphology may be altered in the following ways:—

(a) The heads may be twice as large as normal and may take the pink of Jenner's stain and frequently the neck and tail may stain blue. The contents of the nucleus on the head may present defects.

(b) Sperms with a "U" shaped tail and normal head but always motionless.

(c) Wry-neck sperm with considerable motion.

(d) Large head stained blue with a very thick neck piece, a short tail with slight motion.

A proper examination of the semen can only be carried out in the locality. There is no satisfactory method for the despatch of semen for examination purposes to distant laboratories. Motility tests are the most important of all in the diagnosis of sterility in the male and the available methods for transporting semen are yet too undependable to maintain the proper motility of sperms in vitro. Exposure to light and variation of temperature are inimical to the survival of the spermatozoa in vitro. Dewar flasks and thermos flasks maintained between 20°-30°C have been found the best for keeping spermatozoa suspended in physiological saline active and protected from light. Variation in temperature alters the composition of the fluid.

**TREATMENT.**

The local or general cause of sterility having been determined, treatment is prescribed accordingly. A perusal of the literature shows that most workers have advised elimination of sterile sires from the herd on the apprehension that such sires may transmit their undesirable breeding qualities to the progeny. Bulls suffering from low fertility or sterility have been castrated or sent to the slaughter house. The available methods of treatment of sterility are therefore applicable in the case of females only [Kohn, 1930; Stalfors, 1930], and no helpful reference to the practical application of endocrinology in cases of male sterility exists. Further it seems that no reliable standardised product for veterinary use has yet been put on to the market.

A number of preparations have been described which, given by injection or in some cases by the mouth, stimulate the sex glands. The principal sources of the active principle are the anterior lobe of the pituitary [Evans, 1923] and the urine of pregnant women. Zondek and Aschheim [1927] have also demonstrated that useful effects can be produced by the use of the urine of pregnant cows and since then this work has been extended to include other animals of economic importance [Crew, Miller and Anderson, 1931; Kust, 1932]. Several workers have observed the
enlargement of the male sex organs and particularly of the seminal vesicles, following injection of the urine of pregnant women into male rats or mice. Recently Jongh and Laqueur [1931] have shown that the ovarian hormone arrests the development of the testes and the dependent male sex organs, but the substance from the anterior pituitary or apparently a substance identical with it, as found in the urine of early pregnancy, promotes the development of the testes and the associated sex organs. The considerable enlargement of the testes after the administration of the anterior lobe hormone when present is insignificant as compared with the striking increase (30 times or more) in the size of the seminal vesicles. They further studied the action of this hormone on the genitals of senile male rats and have found that the sex organs are reactivated, interstitial tissues are increased and seminal vesicles are enlarged again by means of gravid urine. Gerard [1931] showed that the testicle of the hedgehog which is normally quiescent in the winter reacts to injection of pregnancy urine by marked activity of the interstitial cells. The seminal vesicles and prostate in the young animal become rapidly enlarged after injection. Interesting results have been obtained by Foucin [1931], who found that the injection of urine from pregnant women in the immature testis of the guinea pig rendered cryptorchid by operation, causes the interstitial tissue to develop rapidly. The contents of the seminal tubules were unaffected. Development of the seminal vesicles ran parallel with that of the interstitial gland and these organs attained to full size as in normal animals. Various drugs like glucose [Zondek, 1931], and suphosalicylic acid [Crew and others, 1931] have been used to render the urine less toxic on experimental mice or treated animals. In cases of endocrinial or dietetic deficiency [Meigs, 1927] readjustment of diet is necessary. In obesity, reduction in the quantity of food and moderate exercise are indicated.

Since it is well known that mares failing to hold to one sire sometimes do so readily to others, Todd [1931] considers it a sound procedure to recommend the use of a second sire to obviate sterility in cases where a foal is a financial consideration.

Therapeutic drugs except when given as tonics are useless in cases of male sterility. In studying the stimulation of guinea pig sperms suspended in a glucose saline fluid, buffered at pH 8 by means of drugs, Baker [1931] found that strychnine hydrochloride had a marked stimulating effect at 1/16, 1/64, 1/256 per cent. Brucine hydrochloride, which was half as poisonous, has the same stimulating effect on sperms. He expressed the hope that this discovery might find practical application in medicine and agriculture wherever sterility be due to inactivity of sperms.

As already mentioned, the mere changes in the hydrogen-ion concentration of the semen has a profound effect upon the motility of the spermatozoa and therefore on the fertility of a male. Unterberger [1930] reports that the addition of bicarbonate
of soda to the vaginal contents during coitus results in the cure of many cases of sterility. This is perhaps due to the correction of excessive acidity which is found in the vaginal contents of these sterile cases. The work of Anderson, Peter and Healey [1921] suggests that douching of the vaginal canal before coitus with a strongly buffered solution, as phosphates, bicarbonates of the required pH (7.3 in the case of stallions), may produce beneficial results.

In man, diathermy and massaging of the prostate and testicles have been recommended for sterility accompanied with sluggish motility of sperms, but these operations would not seem practicable in the case of the larger animals.

Human physicians claim that 40 grains of the anterior lobe of the pituitary and 12 grains of desiccated suprarenal daily have cured cases of sterility in the human male by increasing motility, number and size of the sperms. Jakeman [1931] says that many cases of sterility attributed to testicular dysfunction are doubtless due to anterior pituitary deficiency and may be corrected by the administration of anterior pituitary in doses ranging from 3 to 10 grains per day and presumably he is referring to cattle. From the above, a dose rate may be worked out, as such experimental treatment would certainly seem indicated. Proprietary preparations are obtainable from several manufacturers, for example:

(1) Carnrick & Co.'s "Viriligen" is indicated in lowered virility and sexual neurasthenia of functional origin. It presents desiccated extracts of anterior pituitary, suprarenal cortex, lymph, brain and spinal cord substance, and 1/10 gr. thyroid. It is obtainable in tablets, capsules or ampoules from Messrs. B. K. Paul & Co., 1, Bonfield's Lane, Calcutta.

(2) The Veterinary Division of Glandular Laboratories of America, 72, Cortlandt Street, New York City, prepares glandular preparations for exclusive use in animals. Two pluriglandular preparations are available for the treatment of sterility in the male, viz. "Semidrol" for intramuscular injection and "Prosek" for oral administration.

(3) Armour & Co.'s "Ovotestis". Frei [1930] recommended the use of this preparation "Ovotestis" in cases of nymphomania, but no reference to its use in the male is available [Tutt, 1931].

At the end of such treatment the seminal fluid should be examined for sperms. From the nature of the ingredients in the above preparations it appears that "Ovotestis" is not likely to be so useful as the first two in the condition under reference.

Implantation of gonads [Miller, 1931] into various domesticated animals of both sexes, which had apparently ceased to breed, has been carried out by various
workers and it is claimed that recrudescence of fertility, sexual potency and an enhancement of bodily functions generally takes place. Recently Lebedinsky [1931] has shown that a number of phenomena of senility are to a great extent abolished by tearing and compressing the testicular tissue and that these effects are maintained for $9\frac{1}{2}$ months. It is suggested that the effects are due to blocking up of the contents of the seminiferous tubules in the testes like those of vasoligation, ligature of the epididymis and vasectomy.

In cases of inflammatory conditions of the genitalia [Dimock and Snyder, 1924], or even in cases of defective sperms, a complete sexual rest for varying periods may be tried. A case is on record where after a complete sexual rest of 25 days, another sample was obtained which showed a remarkable improvement in the staining qualities, whereas the previous samples showed only degenerate sperms.

REFERENCES.


Faeisin, K. (1931)—Reaction of immature cryptorchid testes to urine of pregnant women. Physiological Abstracts, 16, 564.


Paul, C. B. (1906)—*Jl. of Physiol.* 34, 14.
Williams, W. L. (1920)—Technique for collecting semen for Laboratory examination with a review of several diseased bulls. *Cornell Vet.* 10, 87-94.
REPRINTED FROM

THE INDIAN JOURNAL OF VETERINARY SCIENCE.
AND ANIMAL HUSBANDRY.

CONGENITAL "BLINDNESS" OF CALVES IN INDIA.

A Résumé of the Position.

By

CAPTAIN S. C. A. DATTA, B.Sc., M.R.C.V.S.,
Temporary Veterinary Research Officer, Imperial Institute of Veterinary Research, Muktesar.

(Received for publication on 17th August 1932.)

(With Plates XXI—XXIII and one chart.)

The object of the present résumé is to place on record in a brief compass the information available at Muktesar on the occurrence of a peculiar "blindness" in calves in India, in the hope that this will stimulate interest in the condition amongst field workers, and lead possibly to the collection of further information regarding its incidence. The seriousness and economic importance attaching to the problem may be appreciated from the fact that an apprehension has been expressed by some of the authorities concerned that cattle breeding at the Military Dairy Farm at Quetta may have to be given up entirely if the condition of "blind calves", which is so common there, is not successfully tackled.

Cases of this peculiar "blindness" so far encountered, can be conveniently divided into two classes depending upon the complexity and extent of the abnormalities:

Firstly, those in which the calves are born blind, the eyes being apparently quite normal in size, shape and structure and showing no abnormal growth which might explain the absence of visuality. Briefly speaking, these are cases of amaurosis and they appear to be far commoner than the other variety.

Secondly, those in which various sizes of teratomatous growths involving varying extents of the external eye and palpebral fissure are discovered, associated with different grades of rudimentary eyeball, which are definitely related to the defect of sight; briefly, cases of teratoma blindness (Plate XXI, fig. 1).

It is not clear whether the cases of calves being born blind with apparently normal eyes and those which are born blind definitely as a result of the pressure exerted by the dermoid teratoma on the eye have the same origin, but Dr. Darling, Chief Officer of the Imperial Bureau of Animal Genetics, to whom reference will
again be made later in this article, thinks that the available information suggests "the possibility of the amaurotic and teratomatous conditions being different degrees of the same defect". These two may, therefore, be considered together, at least provisionally, as curiously enough they have been occurring together at the same Farm for a number of years, and the speculative theories advanced so far to explain the unknown etiology are based on the same principles, irrespective of the class of abnormality involved.

It has also been ascertained from the above Bureau that these cases of "blindness" at Quetta are quite different from those referred to by Crew [1925] as a "peculiar type of blindness in Holstein-Friesian cattle". It is to be noted that all such specimens received at Muktesar for examination and report during a period of about ten years have been provided by the same Dairy Farm at Quetta, but it has recently been reported that the same condition occurs in Shikarpur, Sukkur and surrounding villages in Sindh. Some cases of "blindness" in calves, apparently identical with those at Quetta, have also been seen in Delhi by Colonel A. Olver, Expert Adviser in Animal Husbandry to the Imperial Council of Agricultural Research, and cases of teratomatous formation on the eye of calves have also been encountered at the Veterinary College Clinic in Calcutta. The occurrence of multiple cases at Quetta and in Sindh is surprising as the observable defects in the calves show nothing that can be associated with a parasitic or bacterial genesis.

From the evidence obtained from the reports and specimens collected at Muktesar, the dermoid tumour is found to be usually of a small size, a slight puffiness being noticed around the eye. The growth may be situated at the corneo-scleral junction as shown in Plate XXI, fig. 2, or simply on the membrana nictitans or the conjunctiva alone. If the affected calf lives long enough, the size of the tumour increases with the growth of the animal. In some cases the tumour may be so large as totally to occlude the palpebral fissure (Atresia palpebrarum) with no more than a speck of the vestigial eyeball being left behind [vide Plate XXII, figs. 1 and 2]. The outer and inner canthus are both equally affected, in fact, no segment of the eyeball appears to be exempt. The orbital cavity is unaltered on both sides and the microphthalmic eye is found situated in a pad of connective tissue. The tumours are composed of structures resembling skin and contain hair, hair follicles, sweat and sebaceous glands and pigment cells [vide Plate XXIII, figs. 1 and 2]. Usually affected calves live for some days only but they may live for years, if allowed to do so. The growths may be considered as benign since the available records do not suggest spread by metastasis and the histological picture of the growth reveals no characters of malignancy. The condition appears to affect the male and the female offspring alike, although a tabular statement supplied with a
recent specimen would seem to indicate the inheritance to be more marked in the maternal line as seen in Schmidt’s [1913] description of a case of “pathological fold of the eye”. No special colour or breed seems to be a predisposing factor. The sires at Quetta are all imported animals and the dams are of both Scindhi and Sahiwal extraction. Calves are generally born blind with or without the abnormal growth. It is said that occasionally blindness or the growth may develop later in the first few months of the calf’s life. In the less severe cases, the growths can be easily stripped off without leaving much palpable blemish or defect.

The position appears to be more unsatisfactory in the amaurotic class of cases where no abnormal growth or lesion can be detected in the eye or its appendages despite the fact that the calves are born blind either at full term or sometimes a few days prematurely. In these cases, the pupil is well-dilated and seems to be fixed in one position although this does not seem to be due to unusual adhesions. The pupil does not react to strong sunlight, mydriatics or myotics. Generally both the eyes are involved, but occasionally only one eye may be defective. In most cases the calves are otherwise healthy, although in an infrequent case they may show deformity of some other part of the body, e.g., the neck or spine may be curved. The condition of amaurosis has been met with lately in two buffalo calves also at Quetta. Careful dissection of the eye from these cases reveals a distinct opacity of the lens and the frontal sinus is sometimes found to contain a reddish gelatinous fluid. It is doubtful if this opacity can be considered as constituting a cataract. Minute histological examination of the structure composing the eye has been made and all the probable pathological changes likely to have produced the condition, such as the detachment of the retina from the choroid coat or a peripheral neuritis affecting the optic nerves similar to that attending (i) quinine or Salvarsan Amanosia in man or (ii) tobacco amblyopia in horses [Lancet Correspondent, 1894], have been eliminated. Although the occurrence of teratomata in calves has been reported from other parts of India, the simultaneous occurrence of both classes of the condition appears to be restricted to Quetta, and the villages round and about Shikarpur and Sukkur. The double condition thus appears to affect a large area of North-West India. Enquiries have been addressed for determining if the condition occurs in other parts of India and for collecting more information on the lateral as well as the direct lines of descent of the affected calves.

It will be seen from a perusal of the literature, which follows hereafter, that no species of animals are exempt from this condition, although reports on the occurrence of such growths in species other than the ox have yet to be made in India. Published reports on teratomata in situations other than the eye are commoner and reports of such growths on the eyes of calves are very few indeed. Few cases have also been recorded in man.
Credit is due to Grafe [1822] for priority in reporting the occurrence of teratomatous formation on the margin of the cornea with growth of hair (in man) and the term "trechosis bulbi" is employed by him. Edwards [1859] appears to be the first to describe a case among animals where both eyes presented such growths. Robertson [1877] describes a case of congenital hairy tumour springing from the sclerotic and cornea of a shorthorn bull-calf, six months old, which was otherwise in perfect health and well-formed. The owner described this as a "double under eyelid" and stated that it had been observed shortly after birth. The only disturbance the tumour seemed to cause was an abundant flow of tears. Numerous hairs similar to eye lashes were found projecting between the eyelids, analogous to moles of the skin. Walley [1877] reports two cases of corneal Dermatoma and says that clinically these growths are not of much consequence. They are more interesting as showing the close relation which exists between conjunctival corneal structures and the skin. Nettleton [1885] describes a dermoid growth on the cornea, of the circumference of a shilling. Bland Sutton [1903] employs the term "conunctival moles" and says that they have been observed in horses, sheep, oxen and dogs. He furnishes a picture of a sheep with the growth and further states that they are provided with hair or wool according to the nature of the tegumentary covering, characteristic of the mammal in which they occur.

In dealing with dermoid teratoma of the conjunctiva, Law [1903] states:—
This consists in a cutaneous product consisting externally of a mass of epidermic cells beneath which are connective tissue, fat cells and muscle fibres, glands and growing hairs. It usually extends inwards from the outer portion of the sclerotic conjunctiva and may encroach on that of the cornea. It is firmly adherent to the sclera and sometimes to the cornea by its base and deeper aspect, but the apex is free and more or less projecting. The colour is yellow or more or less blackened by pigment or even reddened by blood. It has been observed above all in dogs. Prince reports a case in a calf. The Cornell Veterinary College Clinic has furnished cases in oxen and dogs. They have, however, nearly always been seen in young animals and are probably congenital.

Kitt [1906] states that dermoid teratomata of the cornea are congenital anomalies and originate from a misplacement or transplantation in the formation of the palpebral fissure. Under congenital affections, Nicholas [1914] states that dermoids are not rare. In mentioning a number of people who encountered them, he says that Kitt came across 24 cases in the ox, dog, sheep, horse, pig and cat. He gives a picture from Cadiot and Almy of a conjunctivo-corneal dermoid but does not specify from what animal the photograph of the growth was obtained. Joest [1921] states that they occur in the dog and swine and gives a photograph of a corneal dermoid encountered in the pig.
In these cases of congenital "blindness" the normal physiological processes which underlie the somatic development of the eye are obscured and the aberrations in question are produced in a manner which is not understood. Of the many theories which have been invoked for explaining either a teratomatous formation or microphthalmia, the following may be recalled for scientific interest:—

(a) Dermoid teratomata have been considered as:—

(i) Remains of amniotic adhesions [Van Duyse],
(ii) Invagination of the ectoderm [Remak],
(iii) The product of continued development of one individual in the body of another individual of the same species [Adami],
(iv) External teratomata are rudimentary twins [Budde, 1923],
(v) Malformation of the somatic tissues of the host [Nicholson, 1929], or
(vi) Inclusion of a second fertilised pole in case of di- and tri-spermic fertilisation [Roche and Roche].

(b) Microphthalmia has been explained as originating from:—

(i) Incomplete closure of the foetal ocular cleft [Arlt],
(ii) Foetal illness in orbita [Wedl and Boch],
(iii) Intrauterine sclero-chorioretinitis [Deutschmann],
(iv) Heredity.

The generally accepted view that teratomata and microphthalmia have a definite genetic basis and that they are inherited as a congenital or familial affection does not seem to be supported by the family history of the animals related to the abnormal calves at Quetta, unlike the condition of "bull-dog calves". The defects are not manifested in the parents, who have been invariably in perfect health. Records are available of 6 calves which were born to "Ceres", one of two bulls which went blind at Quetta in recent years, but none of his calves were born blind. The other bull, "Overton Bearer", probably had a traumatic cause for its blindness and none of its progeny is reported to have been born with abnormal eyes. The bulls employed in the villages of Sindh may be assumed to be of indigenous breeds and not of the imported breeds at Quetta. Dams of the local breeds are presumably used in the villages. In order to eliminate any common ancestor, if one such could be traced, the breeding records of the stud bulls and cows at Quetta, as far as available, have been examined but in vain. The bulls at the Farm are imported animals, being either Ayrshires from England or Friesians from South Africa. The fact that the bulls are from such mixed sources does not seem to indicate the possibility of any relationship existing between the sires. The
maternal parent is drawn from the country-bred stock of Scindhi or Sahiwal extraction. Any relationship of the condition with the cows does not seem likely as this peculiar condition has not yet been reported from any other part of India where cows of these breeds are employed for the raising of stocks, excepting for the solitary report from Shikarpur and Sukkur already mentioned. Dr. Darling remarks that "From the pedigrees offered I have worked very carefully through the possible relationships, and I can see no reason for attributing the amaurosis or teratomatous condition of the eyes of the calves to an heredity factor." The data secured so far do not point to the condition being a feature of atavism or the blindness being transmitted as a recessive mutant, as might be expected from a statement of Crew and Smith [1930] to the effect that blindness caused by the entire absence of the eye or by the presence of a rudimentary eye is inherited in a recessive fashion. As there was every reason to believe in the possibility of the condition being transmissible to the progeny, the authorities at the Quetta Dairy were advised that the affected calves be not retained for breeding purposes. A statement of Crew [1925] that "chances that a recessive mutation will affect the race is very small indeed if the mating is free and uncontrolled", suggests that the affected calves might have been retained and successfully utilised without detriment to the progeny.

Excepting the Scindhi dam, the only common factor at Shikarpur and the Quetta Dairy seems to be the feeding of Juar fodder, obtained from Shikarpur. Chemical analysis of forage at Quetta has been carried out and mineral additions such as bone meal, lime, and potassium iodide to the fodder have been made without any evident reduction in the incidence of the condition there. It was observed that in a certain year, when the fodder was supplemented by mineral constituents and all the cows were served by the usual imported Friesians, there was no scarcity of cases of abortions, premature births and "blind calves", whereas in other years there have occurred no cases of blindness, notwithstanding abortions and premature births, although no mineral additions to the fodder were made.

As mentioned before, the help of the Imperial Bureau of Animal Genetics, Edinburgh, was invoked and the Chief Officer's reply is quoted in extenso:--"At the same time there is this point which occurs to me with regard to possible dietary deficiency. All the affected calves appear to be crossbreds. It is to be expected that the indigenous cattle are "metabolically acclimatised", if I may use such an expression, whilst European breeds would not be. Now if a sufficient number of purebred Friesians or Ayrshires had been bred on the Farm (and from the records before me none had been) it might have been found that the incidence of the condi-
tion would have been higher even than in the crossbreds, whilst in purebred Indian cattle the condition might have been rare. Such a result, of course, would indicate the possibility of a dietary deficiency due to innate lack of metabolic adaptation in the imported cattle.

As that character of the animal known as "constitution" rests no doubt on a multiple factor basis, it is obvious that the crossbred cattle would not inherit either of the parents' "constitutions" in a clear-cut Mendelian manner. The metabolic adaptation of the crossbreds would be more or less deficient and this would be more pronounced, from the genetical multiple factor point of view, in the cross generation, quite apart from the fact that in addition these individuals would be three-quarters European blood. From your records I see that many of the defective calves are such three-quarter bred. You will see, however, that the actual condition does not appear to be directly due to a genetical factor, but to an indirect constitutional factor which allows the development of such a condition in the presence of some dietary deficiency. I should be pleased to hear further from you if any good results accrue from phosphate feeding of the cattle."

With reference to the two important points raised in the above reply, it is to be noted that (1) the conditions are not restricted to the crossbreds only, as a few cases of the same condition have been seen in several villages in Sindh and also in two buffalo calves at Quetta, and (2) bone meal, lime and potassium iodide have been added to the fodder without any apparent results.

The circumstances of the origin of amaurosis would make one suspect some disturbance of central origin due to an animal or vegetable toxin, bacterial infection or impaired circulation during intrauterine life. For the last four or five years, vaccination against abortion has been practised on all the heifers and newly purchased animals, but the condition existed at the Farm even prior to the introduction of this vaccination and hence the vaccine can scarcely be incriminated.

The influence of the amount of moisture and temperature of this locality on the development of Mendelian characters has to be examined. It is said that there is a deficiency of phosphates in the forage at Quetta and if it is so, phosphate therapy should be beneficial. It is understood that there occurs in man a peculiar night-blindness (Hemorolopez) which readily responds to phosphate treatment. A similar condition of "night-blindness" (nyctalopia) is known to occur in the Madras Presidency and Raghavan and Thiagaraj (1920) consider that severe sunlight and errors in diet are probably the causal factors in this condition. Defective hormonal secretion may also be involved. Lethal factors like those in the production of earless sheep [Wriedt, 1921] and semilethal factors as in cases of microphthalmia, coloboma and glaucoma in a certain breed of
dogs [Wriedt, 1924] may be causally connected with the genesis of the so called "blindness" in India.

Our thanks are due to several members of the Royal Army Veterinary Corps, who have held charge of the Baluchistan District, and particularly to Major C. Townsend and Major W. N. Rowston for supplying us with much information in connection with this interesting condition.

We are also greatly indebted to Colonel G. Mellor, Director of Military Farms, Simla, for his interesting note and tables, which are appended to this article.

REFERENCES.
NOTE DATED 23RD JULY 1932 BY COLONEL G. MELLOR, DIRECTOR OF FARMS, SIMLA.

I have read with great interest Capt. Datta's paper on congenital blindness of calves. From the information available I have compiled a chart showing incidence of blindness on the Government Military Dairy, Quetta, since 1913. This
CONGENITAL BLINDNESS OF CALVES.

May not be accurate for the early years but I know that from 1st April 1929 to 31st March 1930 no calf was born blind at this Farm. I also attach a statement showing details of parentage of all calves born blind or going blind at Quetta—1st April 1931 to 31st March 1932. As it may be of interest I attach statements showing grain and fodder and minerals fed during the years 1927-28, 1928-29 and 1929-30, also an analysis of the fodder fed during these years. The kirby was obtained from Upper Sindh, khoosa from the Quetta Valley. Gram and cake fed at Quetta are from the same sources as fed on our other dairies. Bran usually comes from the mill at Shikarpur, Upper Sindh.

Soil analysis is given for comparison:

<table>
<thead>
<tr>
<th>Locality</th>
<th>Lime</th>
<th>Phosphorus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferozepore</td>
<td>4.0</td>
<td>.20</td>
</tr>
<tr>
<td>Gambar (Montgomery)</td>
<td>4.0</td>
<td>.20</td>
</tr>
<tr>
<td>Quetta</td>
<td>13.5</td>
<td>.15</td>
</tr>
<tr>
<td>Rawalpindi</td>
<td>9.0</td>
<td>.18</td>
</tr>
<tr>
<td>Jullundur</td>
<td>5.3</td>
<td>.16</td>
</tr>
<tr>
<td>Ambala</td>
<td>1.0</td>
<td>.15</td>
</tr>
</tbody>
</table>

From 1st April 1932 to date 46 calves have been born blind at the Government Military Dairy, Quetta, of which 18 were blind at birth, i.e., 39.1 per cent. There is a slight indication that the percentage of calves born blind is higher in the spring, i.e., in cows carrying their calves during the winter. This year the number of blind calves having other defects such as twisted necks, unable to stand or even sit up has markedly increased.

Two cases of calves going blind after birth are of special interest as the dams had never been in Quetta until within a few days of calving. Calf “Hatim” born 15th September 1931 en route from Gambar (Montgomery District) to Quetta, — dam a half-bred — was purchased as a heifer at Pusa and remained at the Young Stock Farm, Gambar, until about to calve for first time, when she was railed to Quetta. “Hatim” was born in the train and went blind at Quetta at the age of 5 months.

Calf “Haif” born 14th February 1931 at Quetta — dam countrybred cow — born at Ferozepore on 15th November 1928, transferred from Gambar to Quetta in calf on 15th September 1931. “Haif” went blind on 15th February 1932 at the age of 4½ months.

Two other interesting cases are those of heifers “Hazir” and “Half”. “Hazir” born in Quetta, a heifer calving for the first time, arrived in Quetta on 6th May 1933 having spent the last six months at Ruk (Upper Sindh) where she had plenty of green food. Calved 16th May 1932. Calf blind at birth and up to date this is the only first calf to be born blind.
"Half", a 6th bred heifer, produced a perfect calf on 13th July 1932. "Half" went blind when 6½ months of age, but was retained.

I understand that whilst the army were maintaining the East Persian Cordon 1920—1922 a very large number of army camels went blind but on being returned to Quetta and fed on green lucerne recovered their sight.

This question is undoubtedly of very great importance, if animals as well fed and cared for as they are at the Government Military Dairy Farms suffer to this extent, the loss to the villagers of Baluchistan and Sindh must be enormous. In the circumstances I fully endorse the hope expressed by Capt. Datta in the opening paragraph of his paper.
### Information regarding calves born blind at Government Military Dairy Farm, Quetta, during 1931-32.

<table>
<thead>
<tr>
<th>Serial No.</th>
<th>Name of Dam</th>
<th>Breed</th>
<th>Farm where served</th>
<th>Farm where spent her dry period</th>
<th>Date of birth</th>
<th>Calves Sex and Breed</th>
<th>Farm where born</th>
<th>Period sire was in Quetta when dam served</th>
<th>No. of calves born previously</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Lotus</td>
<td>Countrybred</td>
<td>Quetta</td>
<td>Quetta</td>
<td>7-4-31</td>
<td>F. ¼ (¼ Fr. + ¼ Sah.)</td>
<td>Quetta</td>
<td>Chieftain 7½ months</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Gladys I</td>
<td>§ Bred (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>13-4-31</td>
<td>M. ½ (¼ Fr., ½ Ayr., ½ Sah.)</td>
<td></td>
<td>Elect 10½</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Ruling</td>
<td>§ Bred (Hol. Har.)</td>
<td></td>
<td></td>
<td>13-4-31</td>
<td>F. ½ (Hol. Sah.)</td>
<td></td>
<td>Adolf 17</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Apprentice</td>
<td>§ Bred (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>20-4-31</td>
<td>F. ½ (¼ Fr., ½ Ayr., ½ Sah.)</td>
<td></td>
<td>Elect 17</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>C-Phalii</td>
<td>§ Bred F. S (Har. Hol. Ayr.)</td>
<td></td>
<td></td>
<td>28-5-31</td>
<td>M. ½ (¼ Fr., ½ Ayr., ½ Sah.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Gone</td>
<td>§ Bred (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>6-6-31</td>
<td>M. ¼ (¼ Fr., ¼ Ayr., ¼ Sah.)</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Hira</td>
<td>(Ayr. Scindhi)</td>
<td></td>
<td></td>
<td>10-6-31</td>
<td>M. ½ (¼ Fr., ½ Sah, ½ Ayr.)</td>
<td></td>
<td>Adidas 17½</td>
<td>4</td>
<td>4th calf also born blind.</td>
</tr>
<tr>
<td>8</td>
<td>Damayanti</td>
<td>§ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>16-6-31</td>
<td>M. ½ (¼ Fr., ½ Ayr., ½ Sah.)</td>
<td></td>
<td>Chieftain 10</td>
<td>7</td>
<td>7th calf also born blind.</td>
</tr>
<tr>
<td>9</td>
<td>Ajad</td>
<td>§ (§ Ayr. + ½ Sah.)</td>
<td></td>
<td></td>
<td>19-6-31</td>
<td>M. ½ (¼ Fr., ½ Ayr.+ ½ Sah.)</td>
<td></td>
<td>Adidas 18</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Grace</td>
<td>§ (Ayr. Scindhi)</td>
<td></td>
<td></td>
<td>14-7-31</td>
<td>F. ¾ (¼ Fr., ¾ Ayr., ¼ Scindhi)</td>
<td></td>
<td>Herman 6½</td>
<td>10</td>
<td>10th calf also born blind.</td>
</tr>
<tr>
<td>11</td>
<td>Adeline</td>
<td>C. B. Sahwal</td>
<td></td>
<td></td>
<td>22-7-31</td>
<td>F. § (Fr. Sah.)</td>
<td></td>
<td>Ceres 11</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Rose I</td>
<td>§ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>28-7-31</td>
<td>F. § (¼ Fr., ¾ Ayr., ¼ Sah.)</td>
<td></td>
<td>Adolf 17</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Arracan</td>
<td>§ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>28-7-31</td>
<td>F. § (¼ Fr., ¾ Ayr., ¼ Sah.)</td>
<td></td>
<td>Chieftain 11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>Name of Dam</td>
<td>Breed</td>
<td>Farm where served</td>
<td>Farm where spent her dry period</td>
<td>Date of birth</td>
<td>Calves Sex and Breed</td>
<td>Farm where born</td>
<td>Remarks</td>
<td>Period sire was in Quetta when dam served</td>
<td>No. of calves born previously</td>
</tr>
<tr>
<td>-----</td>
<td>-------------</td>
<td>-------</td>
<td>-------------------</td>
<td>---------------------------------</td>
<td>---------------</td>
<td>----------------------</td>
<td>-----------------</td>
<td>---------</td>
<td>------------------------------------------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>14</td>
<td>Hariawai</td>
<td>C. B. (Sahiwal)</td>
<td>Quetta</td>
<td>Quetta</td>
<td>26-8-31</td>
<td>M. (Fr. Sah.)</td>
<td>Quetta</td>
<td>Adolf</td>
<td>20 months</td>
<td>1</td>
</tr>
<tr>
<td>15</td>
<td>Annale</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>3-9-31</td>
<td>F. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Elect</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Gladys II</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>2-9-31</td>
<td>M. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Adolf</td>
<td>22½</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Beauty II</td>
<td>C. B. (Sahiwal)</td>
<td></td>
<td></td>
<td>14-9-31</td>
<td>M. (Fr. Sah.)</td>
<td></td>
<td>Herman</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Gift</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>10-1-32</td>
<td>M. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Adolf</td>
<td>25½</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Duldul</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>29-1-32</td>
<td>F. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Chieftain</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>Honey</td>
<td>½ (Ayr. Scindhi)</td>
<td></td>
<td></td>
<td>5-2-32</td>
<td>M. (½ Fr., ½ Ayr., ½ Sc.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>Spendthrift</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>13-2-32</td>
<td>F. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Adolf</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>Nosegay</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>22-2-32</td>
<td>F. (½ Fr., ½ Ayr.</td>
<td></td>
<td>Wanderer</td>
<td>7 days</td>
<td>3rd and 5th calves also born blind.</td>
</tr>
<tr>
<td>23</td>
<td>Gaby</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>23-2-32</td>
<td>M. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Chieftain</td>
<td>18½ months</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>Ashara</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>5-3-32</td>
<td>M. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Chieftain</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>Delkate</td>
<td>½ (Ayr. Sah.)</td>
<td></td>
<td></td>
<td>13-3-32</td>
<td>M. (½ Fr., ½ Ayr.)</td>
<td></td>
<td>Wanderer</td>
<td>16 days</td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>Datura</td>
<td>F. 2½ (Ayr. Scindhi)</td>
<td></td>
<td></td>
<td>13-3-32</td>
<td>M. (½ Fr., ½ Ayr., ½ Sc.)</td>
<td></td>
<td>Chieftain</td>
<td>20 months</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>Architect</td>
<td>½ (Ayr. Scindhi)</td>
<td></td>
<td></td>
<td>16-3-32</td>
<td>F. (½ Fr., ½ Ayr., ½ Sc.)</td>
<td></td>
<td>Adolf</td>
<td>27½</td>
<td></td>
</tr>
</tbody>
</table>

(1) Chieftain: 16th November 1929 to 28th July 1930: 20 months.
(2) Elect: 15th March 1929 to 28th July 1931: 28 months.
(3) Adolf: 15th March 1929 to 28th July 1931: 28 months.
(4) Herman: 16th November 1929 to 8th December 1929, 1st March 1930 to 21st February 1931: 12 months.
(5) Ceres: 10th November 1929 to 12th December 1931: 25 months.
(6) Wanderer: 22nd June 1931 to date: 3 months.
(7) Onega: 1st March 1928 to 26th February 1929: 28 months.
(8) Terror: 13th March 1929 to 21st February 1930: 12 months.
Information regarding calves that went blind after birth at Government Military Dairy Farm, Quetta, during 1931-32.

<table>
<thead>
<tr>
<th>Serial No.</th>
<th>Name of Dam</th>
<th>Breed</th>
<th>Farm where served</th>
<th>No. and names of calves</th>
<th>Date went blind</th>
<th>Date of birth</th>
<th>Sex and breed</th>
<th>Farm where born</th>
<th>Period sire was in Quetta when dam served</th>
<th>No. of calves born previously</th>
<th>Whether any born blind</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bess</td>
<td>½ Bred (Ayr. Sah.)</td>
<td>Quetta</td>
<td>H. 223 Half</td>
<td>15-3-30</td>
<td>31-8-30</td>
<td>F. ½ (Ayr. Sah.)</td>
<td>Quetta</td>
<td>Omega</td>
<td>8 months</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Gulkeri</td>
<td>Ditto</td>
<td>Quetta</td>
<td>H. 220 Hit</td>
<td>11-4-30</td>
<td>18-7-30</td>
<td>F. ½ (Ayr. Sah.)</td>
<td>&quot;</td>
<td>Terror</td>
<td>6½</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>Birkini</td>
<td>½ Bred (Ayr. Sah.)</td>
<td>Quetta</td>
<td>H. 323 Harass</td>
<td>21-2-31</td>
<td>8-10-30</td>
<td>F. ½ (Fr. + ½ Ayr. + ½ Sah)</td>
<td>&quot;</td>
<td>Adolf</td>
<td>9½</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>Pamela</td>
<td>Ayr. Sah.</td>
<td>Quetta</td>
<td>H. 324 Handsome</td>
<td>21-2-31</td>
<td>9-10-30</td>
<td>F. ½ (Fr. + ½ Ayr. + ½ Sah)</td>
<td>&quot;</td>
<td>Elect</td>
<td>9½</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>Little</td>
<td>H-B. Ayr. Sah.</td>
<td>&quot;</td>
<td>H. 334 Hoyland</td>
<td>10-3-31</td>
<td>14-11-30</td>
<td>F. ½ (Fr. + ½ Ayr. + ½ Sah)</td>
<td>&quot;</td>
<td>&quot;</td>
<td>10½</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>Beaty</td>
<td>Ditto</td>
<td>&quot;</td>
<td>H. 339 Hickie</td>
<td>21-3-31</td>
<td>15-12-30</td>
<td>F. ½ (Fr. + ½ Ayr. + ½ Sah)</td>
<td>&quot;</td>
<td>&quot;</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>Martha</td>
<td>Ayr. Sah.</td>
<td>&quot;</td>
<td>H. 341 Higgins</td>
<td>15-3-31</td>
<td>19-12-30</td>
<td>F. ½ (Fr. + ½ Ayr. + ½ Sah)</td>
<td>&quot;</td>
<td>Adolf</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>8</td>
<td>Kasauti</td>
<td>Sahiwal</td>
<td>Gambar</td>
<td>H. 306 Half</td>
<td>15-2-32</td>
<td>6-10-30</td>
<td>F. ½ (Fr. + ½ Sah.)</td>
<td>&quot;</td>
<td>Wanderer</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>9</td>
<td>Farangan</td>
<td>½ (Har. Sah.)</td>
<td>&quot;</td>
<td>H. 384 Halim*</td>
<td>14-2-32</td>
<td>6-9-31</td>
<td>F. ½ (Fr. Sah.)</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
</tbody>
</table>

(Note.—Green fodder was fed from 17th February 1932 to 25th February 1932 and also from 9th March 1932 to date.)

* Born in the train and goes blind after 5 months in Quetta. ** In the train from Gambar.
<table>
<thead>
<tr>
<th>Month</th>
<th>Gram</th>
<th>Bran</th>
<th>Cake</th>
<th>Salt</th>
<th>Kirby</th>
<th>Bloca</th>
<th>Kirseim</th>
<th>Makai</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 1927</td>
<td>Fed</td>
<td>Fed</td>
<td>Fed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Gram, Bran and Cake were mixed in the proportion of 6:2:1 and of this mixture the cows in calf received a ration of 4 lbs., 1 lb. for every month over six in calf, thus at seventh month it received 5 lbs., at 8th month 6 lbs., and in the 9th month 7 lbs., 15 lbs. of dry fodder throughout. Also 10 lbs. of green fodder was fed per head during months it was available.</td>
</tr>
<tr>
<td>August</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fed</td>
<td></td>
</tr>
<tr>
<td>September</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>October</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>November</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>December</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>January</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>February</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fed</td>
<td></td>
</tr>
<tr>
<td>March</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>April</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fed</td>
<td></td>
</tr>
<tr>
<td>May</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>June</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Month</th>
<th>Gram</th>
<th>Bran</th>
<th>Cake</th>
<th>Salt</th>
<th>Kirby</th>
<th>Bloca</th>
<th>Kirseim</th>
<th>Makai</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 1928</td>
<td>Fed</td>
<td>Fed</td>
<td>Fed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Gram, Bran and Cake were mixed in the proportion of 6:2:1 and of this mixture the cows in calf received a ration of 4 lbs., 1 lb. for every month over six in calf, thus at seventh month it received 5 lbs., at 8th month 6 lbs., and in the 9th month 7 lbs., 15 lbs. of dry fodder throughout. Also 10 lbs. of green fodder was fed per head during months it was available.</td>
</tr>
<tr>
<td>August</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>September</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>October</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>November</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>December</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>January</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>February</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fed</td>
<td></td>
</tr>
<tr>
<td>March</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>April</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fed</td>
<td></td>
</tr>
<tr>
<td>May</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>June</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
CONGENITAL BLINDNESS OF CALVES.

1929-30.

<table>
<thead>
<tr>
<th>Month</th>
<th>Gram</th>
<th>Bran</th>
<th>Cake</th>
<th>Salt</th>
<th>Kirby</th>
<th>Bhoosa</th>
<th>Ber-seem</th>
<th>Makai</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 1929</td>
<td>Fed</td>
<td>Fed</td>
<td>Fed</td>
<td>Fed</td>
<td>Fed</td>
<td>Fed 4 days</td>
<td>Fed</td>
<td></td>
<td>Gram, Bran and Cake were mixed in the proportion of 6: 2: 1 and of this mixture the cows in calf received a ration of 4 lbs., 1 lb. for every month over six in calf, thus at seventh month it received 5 lbs., at 8th month 6 lbs., and in the 9th month 7 lbs., 15 lbs. of dry fodder throughout. Also 10 lbs. of green fodder was fed per head during months it was available.</td>
</tr>
<tr>
<td>August</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>September</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>October</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>November</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>December</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>January 1930</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>February</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>March</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>April</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>June</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 ozs. per head per diem for the year.</td>
</tr>
</tbody>
</table>

Statement showing minerals fed to cattle at Government Military Dairy Farm, Quetta.

<table>
<thead>
<tr>
<th>Lime*</th>
<th>Bone meal*</th>
<th>Iodide of Potassium</th>
</tr>
</thead>
<tbody>
<tr>
<td>April 1928 to July 1928:— No information available.</td>
<td>April 1928 to February 1929:— Fed.</td>
<td>Fed at the rate of 2 ocs. per ton of concentrates to the herd from 1st April 1928 to 31st June 1930.</td>
</tr>
<tr>
<td>August 1928 to March 1929:— Fed.</td>
<td>March 1929:— 13 lbs. only.</td>
<td></td>
</tr>
<tr>
<td>April 1929 to November 1929:— Fed.</td>
<td>April 1929 to December 1930:— Nothing fed.</td>
<td></td>
</tr>
<tr>
<td>December 1929 to 7th May 1930:— Nothing fed.</td>
<td>January 1930:— 200 lbs. only.</td>
<td></td>
</tr>
<tr>
<td>8th May 1930 to 4th July 1930:— Daily.</td>
<td>February 1930 to May 1930:— Nothing fed.</td>
<td></td>
</tr>
<tr>
<td>10th June to date:— Being fed daily.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Quantity fed: —8 ocs. per 100 lbs. of concentrates. This works out at an average of 1 oz. per animal.
Analyses of grasses at Quetta.

<table>
<thead>
<tr>
<th>Register No.</th>
<th>Name of sample</th>
<th>Locality</th>
<th>Rainfall in inches</th>
<th>Air per cent dried wt.</th>
<th>Ash per cent.</th>
<th>Insol. residue on fodder</th>
<th>Crude fodder</th>
<th>Protein</th>
<th>Lime</th>
<th>Phosphorus</th>
<th>Chlorine</th>
<th>Iron</th>
</tr>
</thead>
<tbody>
<tr>
<td>230</td>
<td>Hill grass</td>
<td>Quetta</td>
<td>9.58</td>
<td>4.92</td>
<td></td>
<td></td>
<td></td>
<td>5.4</td>
<td>1.08</td>
<td>0.141</td>
<td>358</td>
<td>1.76</td>
</tr>
<tr>
<td>59</td>
<td>Kirby</td>
<td></td>
<td>5.4</td>
<td>8.03</td>
<td>2.63</td>
<td>46.1</td>
<td></td>
<td>0.54</td>
<td>0.315</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>58</td>
<td>Bhoosa</td>
<td></td>
<td>14.41</td>
<td>7.49</td>
<td>44.1</td>
<td>1.19</td>
<td>1.60</td>
<td>0.074</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>368</td>
<td>Kirby</td>
<td></td>
<td>10.4</td>
<td>4.31</td>
<td></td>
<td></td>
<td></td>
<td>1.74</td>
<td>0.800</td>
<td>0.177</td>
<td>0.644</td>
<td></td>
</tr>
</tbody>
</table>
Fig. 1. Head of a newly born calf showing a teratomatous growth completely filling the palpebral fissure of the left eye.

Fig. 2. Photomicrograph showing dermoid teratoma at the cornea-scleral junction.
FIG. 1. A pair of eyeballs totally covered by extensive teratomatous formation.

The eyeballs in this case were very small and rudimentary.

FIG. 2.

A. Posterior pair—In the left eye only a few hairs are seen—size and appearance of the eyeballs covered up with the growth. Size of eyeball slightly smaller than normal.

B. Middle pair—Almost half of the eyeballs covered up with growth. Size of eyeball practically obliterated.

C. Front pair shows normal size and appearance. Eyeballs practically obliterated.
PLATE XXIII.

FIG. 1. Photomicrograph showing the histology of a dermoid teratoma of the eye, with a sebaceous gland.

FIG. 2. Photomicrograph showing the structures which compose a dermoid teratoma of the eye.
REPRINTED FROM

INDIAN JOURNAL OF VETERINARY SCIENCE AND ANIMAL HUSBANDRY.

Vol. IV, Part IV, December 1934.
THE ETIOLOGY OF ENZOOTIC BOVINE HAEMATURIA, PART I.

BY

CAPTAIN S. C. A. DATTA, B.Sc., M.R.C.V.S.,

Veterinary Research Officer, In-charge Pathology, Imperial Institute of Veterinary Research, Muktesar.

(Received for publication on 16th October 1934.)

(With Plates XIX—XXIX and 1 text-fig.)

INTRODUCTION.

Among the animal diseases which have baffled elucidation notwithstanding the very considerable amount of scientific investigation that has been expended upon them in most countries of the world, chronic haematuria of cattle would appear to be one of the most outstanding. Since the articles by Anderson and Hubner were published in 1842, more and more attention has been devoted to the subject. Although various types of pathological processes affecting the urino-genital system can lead to the passage of unbroken red blood corpuscles in the urine, for that is what the term haematuria signifies, this disease entity has come to be recognised as a precise and clear cut clinical syndrome. Haematuria has been a serious scourge in cattle farms in many countries, and the first exhibition of blood in the urine leaves no other alternative for the owner than the disposal of the affected animals for slaughter as, from long experience, the futility of treatment has been recognised. The disease is known to occur in widely separated countries of the world including Australia, Austria, Bulgaria, Belgium, British Columbia, England (Cornwall), France, Germany, India, Ireland, Italy, Kenya Colony, New Zealand, Oregon, the Pacific Islands, Washington, Scotland and Wales. The names under which the disease has been known in the various countries are as follows:—Red water, stallrot (stable red), Illawater Red water, Essential Haematuria, Haemorrhagic cystitis, Hematurie chronique des bovides, Haematuria vesicalis, malignant haematuria and Urocystis haemorrhagica, etc. In India the term haematuria seems to have been employed for the first time by Kristnasamiengar as late as 1896, but from the symptoms described by him it is evident that he was dealing with haemoglobinuria only. Credit is therefore due to Kerr for drawing attention in 1925 to the occurrence of true haematuria in Indian cattle at Kalimpong in Bengal and the disease has since then been investigated, also work has been carried out in the Kumaun Hills in the United Provinces, in the Nilgiris in the Madras Presidency and
in the Kulu valley in the Punjab. In the last named province, it is reported that cases of haematuria in man occur in the same areas in which cattle are affected. It is said that in the Darjeeling District in Bengal, the people dread this disease more than rinderpest.

Many theories have been advanced to explain the causation of the disease, some of which are undoubtedly fantastic. Leaving aside such primitive theories as insufficient feeding (Anacher), poor feeding (Cruzel), periodical compression of the bladder by the rumen when it is filled unduly with voluminous food (Liaux), stasis in the territory of the posterior vena cava (Hink), certain non-specific irritations mechanical, mycotic or toxic (Gotz), increasing precocity and diminishing resistance by crossing with a certain breed (Sinoir), the toxic effects of plants, including the young shoots of oak, ash, privet, hornbean, hazel, dog-berry, pine, fir, coniferae, [Law, 1901], ferns, sedges rushes hellobore, etc. (Galtier), and the trauma due to filaria or distoma (Lydtin), or that due to pentastomes (Burton and Cleland), coccidia (Arnold) or bacterial infection (Detroye) have been suggested. Among those who ascribe the disease to malignant neoplasms, Miyamoto [1927] seems to be outstanding, since he portrays the development of various types of neoplastic formation in regular sequence. This disease has also been considered to be an analogue of what is known in human medicine as acquired angiomata or internal sarcoids. The possibility of poisonous plants causing haematuria has been tested by several workers with a negative result in each case. For instance, Cleland [1911] experimented with Omalanthus populifolius (Euphorbiaceae), Indigofera australis and Goodia lotifolia (both Papilionaceae), and Hadwen employed in his tests extracts of dicentra (Bleeding heart), deergrass (Achlys triphylla), bracken (Pteris aquilina) and the extract of alder. This latter worker remarks that it is very improbable that one special plant causes the trouble, because vegetations in the various countries differ so much, and that a group of plants may thus be responsible rather than one particular species. In the Darjeeling district in Bengal the local people believe bamboo and fig leaves, which are common fodder there, to be the cause of the disease. Bamboo leaves (N. O. Gramineae Bambusa, arundinacea) are used by Indian villagers in cases of retention of the placenta in the cow apparently with a beneficial irritant action, simulating to a certain extent the effects of mild abortificients and Garudaecher [1930] has reported the presence of hydrocyanic acid in bamboo leaf extract. Again Mustak Husain [1930] in the United Provinces incriminates tinpatia grass. From a knowledge of the toxicology of plants, however, one would expect a preponderance of constitutional symptoms, and in violent cases rapid death, if some irritant buds or shoots played a role; and in any case the singular localisation of any toxic effects to the bladder exclusively would not seem probable.
ETIOLOGY OF ENZOOTIC BOVINE HAEMATURIA

In regard to the bacterial theory, the available evidence of most workers recorded in literature conforms to the experience of the author, which shows that attempts at bacteriological culture from the urine and internal organs invariably prove abortive in early cases. No bacteria have been seen in stained sections from internal organs in most cases, but diphtheroids and long chained streptococci have been seen and also recovered from the urine from clinical cases of old standing. Hadwen and others have failed to transmit the disease to healthy animals by cohabitation with clinical cases, by injecting haematuria urine into the bladder, by giving it by the mouth and by the injection of the blood and urine under the skin, and it may be noted that the former employed as many as 17 animals for the purpose. Durin and Unglas [1931] however consider the disease to be a coli-bacillosis, and they treated two cases with repeated doses of B. coli anti-virus but the results are not known.

Of the helminths, the finding by Ichikawa in 1922 of adult Schistosoma japonicum in the bladder tumours of two cases in Formosa indicated a possibility, which had the support of the existing knowledge of the bilharzial disease of the intestines and the bladder in man, and also of the experience of Sonsino [1876] who collected thirty Schistosome worms (S. crassa or S. bovis) from the blood of an ox, which presented intestinal lesions, and typical lesions of haematuria in the bladder. However Ichikawa failed to detect any bilharzial worms or ova in five other cases examined by him, and the experience in every other country has been similar. In this connection it may be noted that in Egypt Piot Bey [1918] recorded a case of bilharziosis as the cause of a perforating ulcer of the bladder of a calf, and in India Rangaswamy [1922] reported a case of haematuria in the Nilgiris, in the urine of which animal Schistosome ova are stated to have been seen. In the samples of urine from two other cases of the disease from the same locality, Parameswara Ayyar [1922] however records negative findings.

The position of the oxalic acid theory of Hadwen [1914, 1917] differs from the others mentioned above, since he claimed to have produced the disease experimentally with commercial oxalic acid by (1) injection of calcium oxalate crystals in aqueous emulsion into the bladder, and (2) by the oral administration of oxalic acid, in about 5 months and 2½ years respectively. The fact that he failed to obtain oxalic acid bearing plants in a sufficient amount to feed his animals had raised doubts regarding his contention, and in the absence of any clue for approaching the investigation of the disease, Datta [1931] pointed out the improbability of Hadwen’s view and elaborated a metabolic theory suggesting an endogenous production of oxalic acid due to defective elimination. It seems reasonable to argue that the cattle suffering from the disease do not feed upon such unnatural forage in
excess as are known to contain much oxalic acid, e.g., rhubarb, spinach, tomato, apples, lettuce, grapes, cabbage, and the common Indian sorrel *amrul* (*Oxalis carinulata*). In experimental oxalic acid poisoning, the localisation of the poison in the various organs has been studied by Servonat and Roubier [1911], and Chieri and Frohlich [1911] have shown that a nerve excitability results. Further the work of Craig and Kehoe [1921], Bull [1929] and Steyn [1933] finally disposes of the oxalic acid theory as untenable, contrary to the statement of the *Vety. Bulletin* [1931] to the opposite effect.

With the fall of this theory, however, the trend of opinion in most countries has been towards a chemical deficiency theory. During a tour in Australia, Theiler [1929] expressed himself thus. “What is required is a Laboratory in Mount Gambier itself and preferably on one of the affected farms. The remarkable coincidence of phosphorous and manganese deficiency in this area should not be lost sight of. A clue to further research may then be found”. The Ottawa Report [1930] states “Previous investigations show that it (bovine haematuria) is not an infectious or contagious disease and point to the probability of a nutritional disturbance and to mineral deficiency, hyperacidity, or to an unknown factor in the soil and vegetation as the cause of it”. Again from the same country Fleming, Fowler and Clark [1930] report that the results of blood analysis to be within the normal range. In their experience two remedies have been found to have a beneficial effect on affected cattle: a change of drinking water from surface to artesian well water, and administration of ground coral rock. From analyses of the water and coral rock from affected areas certain possible causes of improvement have been suggested and field experiments outlined to test the possibility. In collaboration with Allardyce [1930] these workers again report that the blood estimation of 25 cattle, subjects of *Haematuria vesicalis*, yielded normal amounts of cholesterol, sugar, nonprotein nitrogen, urea nitrogen, amino-acid nitrogen, creatine, creatinine, calcium, inorganic phosphorus, and chlorides. In the latest publication from Australia, Bull, Dickenson and Dann [1932] state that “the problem is not an easy one to solve, and it is possible that a successful method of prevention may be found before the actual cause of the disease is demonstrated”. In their opinion “it is possible that a deficiency of some dietary constituent is the essential cause of red water,” and from the result of their urine analyses they go on to say that “there appears to be some possibility of a low protein intake and possibly a low sulphate intake. From the Calcutta School of Tropical Medicine, Ghosh [1933] records the results of chemical analysis upon the leaves of plants suspected of causing haematuria, e.g., *Schima wallichii, Ficus nimoralis* and Indian cherry, and since saponins were detected in the first two he states that feeding experiments with the leaves containing saponins are contemplated at the School. Again while
this article was in preparation a claim has been put forward from the same city in the columns of a daily newspaper that "Macgregor and his assistants [1934] have been able to trace the cause of the disease to a protozoan parasite of the genus (?) Coccidium which, ordinarily, attacks the bowel producing intense anæmia and dysentery", reviving thus the unsupported views of Arnold expressed in 1890 in Germany.

Description of the Disease.

That this disease is an enzootic one has been observed since the earliest times, and some places have come to be known as haematuria localities. Attempts have been made by a process of elimination of factors, to trace any relation of the environment to the disease, and Hadwen, for one, has travelled extensively in America and Europe with this object. It has been stated that haematuria localities are sloping, mountainous land with a lack of cultivation and good natural forage, associated with a preponderance of ferns. In these areas the cattle have to roam in woods or partially cleared grounds, and the impermeable soils on which the forage grow generally, lack proper sunshine and are moist. As Law [1901] states, "it is the disease of woods and waste lands, of damp and undrained lands, of dense clays, of lands underlaid by clay or hard pan, of lands rich in vegetable humus, or vegetable moulds, the decomposition of which has been hastened by the application of quick lime", but "the disease has not been traced to any definite microbe or toxin". The drinking water in some of these localities is rich in mica or gravel, and the climate is generally cold. In Germany the disease has been described as occurring in stall-confined cattle, in France as occurring in low-lying districts, and again in others it is in the mountainous and sub-mountainous regions that the disease is seen. The disease has been stated to occur after a dry summer, during the winter, or in the spring. Moussu and Dollar [1905] however state that haematuria occurs just as frequently in the winter when the animals are housed, as in the spring when at pasture. Moussu states that this affection is very rare in young animals and is exceptional before the age of two and a half or three years. Both the sexes are attacked equally.

It is a noteworthy fact that irrespective of the country of origin concerned or of the incriminated cause, the clinical symptoms and histopathology of bovine haematuria have been more or less the same. In this disease the occurrence of any systemic disturbance of health is rare and in our experience no rise of temperature has been noticed prior to or following upon an attack of haematuria. The passing of blood being the only easily noticeable symptom in the living subject, the disease has been termed "essential haematuria" and the apparently contradictory term "symptomless haematuria" in human medicine signifies a similar type of condition.
The affected cases are seldom detected in their earliest stages. The condition does not attract notice until the blood corpuscles have attained a fairly appreciable proportion of the ejected urine, and have stained the urine distinctly red. The disease is not suspected in the earliest stages since the admixture with the urine of a quantity of blood serum, and microscopic amounts of blood cells, exuding from the commencing lesions in the bladder is easily overlooked. However, suspicions having been aroused, the disease is readily diagnosed by the microscopic examination of centrifuged urinary deposits or by the available chemical tests for blood. Frequent micturition, with blood passed at the end of or throughout the act, is seen, and mild symptoms of urinary colic may appear. When the loss of blood has reached a certain limit, the haemorrhagic urine tends to coagulate on the ground or even inside the bladder of the affected animal. The colour of the urine may be pale pink or bright red, depending upon the quantity of blood passed. The red corpuscles are usually completely intact, and there is nothing unusual about their size and shape. If there has been any retention of urine due to atony or due to an obstruction by clot formation at the urethral opening of the bladder, a crenation or actual breaking down of red cells takes place, rendering the urine coffee-coloured. As the disease advances, large-sized flakes of clots are also passed. Anaemic changes in the blood appear comparatively early, which persist and gradually become worse, and animal becomes extremely debilitated with the progress of time. When necessary, the condition of the bladder may be ascertained by a rectal exploration.

Haematuria is characterised by a slow and progressive course with frequent intermissions of variable duration extending from a few weeks to months. The passage of blood may cease suddenly or by degrees, only to reappear in the same erratic manner. This process is repeated till the animal finally succumbs to extreme anaemia and debility, or to other complications such as severe internal haemorrhage into the bladder, hydrenephrosis, uraemic poisoning or superadded infections of the urinogenital system and the alimentary tract. The occurrence of diarrhoea in the later stages of haematuria has been mentioned by a few workers, but no causal relationship between haematuria and the development of actual diarrhoea or the passing of semi-solid faeces mixed with mucus has been suspected. Although the brunt of the lethal effects of the causative principle of haematuria are borne by the urinary bladder, it is important to determine whether other internal organs share in the untoward effects, though not quite to the same extent as the bladder. In the uncomplicated early cases, there is apparently no evidence of kidney involvement, including oedema, rise of blood pressure, or the syndrome of renal colic, etc. Generally the disease lasts for months and years, and in the experience of Moussu [1905] an animal aged 28 years had been suffering for as many as 20 years, but no case of spontaneous recovery or successful treatment has yet been placed on record.
ETIOLOGY OF ENZOOTIC BOVINE HAEMATURIA

From this it appears that the pathological process in the bladder runs almost parallel to the recuperative mechanism of the animal's system. The disease has not been reported in young calves, and the cattle are stated to exhibit the symptoms after grazing for a number of years on one farm. History cards of all animals since their purchase from the surrounding hills are maintained regularly at this Institute. From a scrutiny of these, the time that elapses between purchase and the apparent commencement of haematuria symptoms appears to be short, and it appears possible therefore, that the majority of hill bulls at this Institute bring with them a pre-existing infection, rather than contract the disease here, though there does not appear to be any reason why such an immunity of this place should exist. In the author's experience in 1928-29, cases had been removed from the hills to Calcutta, but a change of locality did not produce any substantial alleviation of the disease. It has been observed at Muktesar that a number of cases invariably exhibit exacerbations of haematuria with snow-falls, and a gradual and continued decrease of the bodily temperature indicates a real danger signal.

MACROSCOPIC LESIONS.

On post-mortem examination naked eye lesions of significance are not usually discernible in any organ excepting the bladder, but occasionally the kidney or the ureters may be abnormal. The bladder may be empty or filled with haemorrhagic urine, which is partially or completely clotted. It has been suggested that it is only in the basal and ventral part of the bladder that the initial lesions are formed, but when the gravitational precipitation of the causal factor can be eliminated, such a suggestion has no significance. On eversion of the organ, variable lesions are encountered. The early lesions appear as small patches of congestion, and red dots of haemorrhage up to the size of a lentil, associated with a slight thickening of the mucous membranes are seen. The altered mucous membrane is raised above the surrounding areas as small round yellow areas enclosed by a ring of acute congestion. Localised gelatinous infiltration of the submucosa is seen, which is followed by the sloughing of the overlying mucous membrane, giving rise to small ulcers, which form the seats of continuous blood-letting until a clot forms. The ulcers are discrete, and, when confluent, become broader or more elongated and present raised edges, assuming a button or crater-like appearance. Since the opportunities of examining genuine early cases are not readily obtainable, the apparent first appearance of haematuria being possibly an intermittent attack only, the naked eye lesions are seldom so simple as the above. On the contrary, one may find "varicosities as large as hemp seeds, proliferations as large as nuts", rounded or cauliflower-like, rough raised red ridges, eroded bleeding patches, and submucous haemorrhages, sessile or pediculated pyriform mucoid cysts, and considerable thickening of the bladder wall.
(Plate XIX, Fig. 1). Cicatrices are not very evident. Only localised areas of the bladder may be involved or the lesions may be diffused throughout. In cases of old standing, considerable ulceration has been detected at post mortem, although blood was not being passed immediately prior to death. The co-existence of such a variety and gradation of lesions, from minor changes to exhuberant growths, shows that the lesions develop separately and continuously, and not simultaneously, explaining the remarkably persistent and intermittent character of haematuria.

In these studies mucoid cysts have been met with on the surface of the kidney, and minute haemorrhages have been seen in this and other organs. Hydronephrosis or cystic kidneys seen in haematuria cases seem to be connected with the bladder lesions. The intestines, particularly the caecum and the proximal end of the rectum of haematuria subjects have shown very minute sub-epithelial haemorrhages or millet seed and pin-head sized round ulcers with raised edges and deep necrotic core, but their significance is not known at present. Plate XIX, Fig. 2 shows polypoid growths in the duodenum from a case of haematuria.

**CLINICAL AND CHEMICAL STUDIES.**

Although in the course of routine post-mortem examinations at Muktesar a few cases of the typical bladder lesions of haematuria were undoubtedly detected earlier, it was only after the first report by Kerr [1925] that the disease received attention. The disease has figured in several Annual Reports of this Institute since that date. In the earlier investigations, arrangements were made to collect full clinical details regarding haematuria, including weekly body weights, daily temperatures, etc. A method was improvised to approximate the proportion of the admixture of blood cells and clots to a fixed amount of urine passed. Blood smears were examined regularly, samples of urine from each fresh case and post-mortem material were subjected to culture examination. Experimental treatment with trypan blue, and the benzidine test for the presence of haemolysed blood in samples of the urine, from which the centrifuged deposits had been removed, were carried out; and thus the possibility of bovine piroplasmosis was first eliminated. By centrifugalising the urine and faeces from uncomplicated haematuria cases, and by means of the sugar flotation technique, repeated examinations were made and the possibility of coccidia was consistently negatived. Repeated attempts to culture any protozoan organism from urinary sediments by the use of 5 per cent. potassium dichromate solution and of hay infusions have given negative results. As opportunities occurred, careful post-mortem examinations were made in order to discover if the lesions in any other organs excepting those of the urinogenital system, could be correlated with the passage of blood in the urine. At that time the probability
ETIOLOGY OF ENZOOTIC BOVINE HAEMATURIA

appeared to be that "the cause of the disease was an irritant chemical substance elaborated from the excretory products in the urine while the urine was stationary in the bladder." Investigation of the disease on the above lines continued, but it was increasingly felt that unless more information on some specific factors relating to the extravasation of blood from the bladder was available, the study of the disease was not likely to be productive of anything but negative data, and a critical examination of all the causes suggested in literature was made. As has already been mentioned earlier in this article, Hadwen's was the only work which claimed to have reproduced the disease experimentally, but, even so, his views appeared to be improbable. Therefore from theoretical considerations of available biochemical knowledge [Datta, 1931] it was considered advisable to determine whether there was in the system, any stagnancy of oxalic or other allied acid, such as parabanic acid (Oxalylurea) of metabolic origin, and also whether the deficiency of calcium or other metallic constituents of normal body tissues, or any haemophilia, existed, which could explain the tendency to the extravasation of blood seem in the living animal.

Since, in a few cases which had died of haematuria, the blood in the body was found to be still in a fluid condition, and with little tendency to clot, attempts were made to test the coagulability of blood. Various methods of determining the coagulation time of blood were tried, and after preliminary trials with drops of blood on glass slides, also in corked and open phials, had been made, the Loop method of Inchley [1921] was chosen as the most suitable working method. The serum calcium of a number of healthy and affected hill bulls was studied. Calcium was administered in various forms to affected cases, since it was desired to raise the calcium content of the blood in the hope of countering haemophilia, if such was present. Curiously, the same average clotting time of about 11 minutes was observed in all animals, whether in the active or quiescent stage with regard to the passage of bloody urine. The administration, morning and evening, of calcium lactate in the feed, and of calcium chloride solution intravenously, did not produce any marked difference in the coagulability, of the blood in affected cases. The clotting time in one case, however, rose to 18 minutes a short time prior to death. Similarly when the serum calcium was estimated in those haematuria subjects which were receiving calcium therapy, for purposes of comparison with those that were not, the calcium value showed only a small increase, but the phosphate value in the treated subject was decidedly on the increase. Another noticeable fact in these experiments was that, even during the period when as much as 5 per cent. of the urine passed by the animal consisted of red corpuscles, the animal was seen to be putting on weight consistently, and this may be ascribed to the benefits of calcium therapy. The result of chemical analy-
sis on an average sample of haematuria urine, from an untreated case is given below. The figures are per 100 c.c. of serum.

Amount of $\text{PO}_4$ (phosphate) = 0.0148 gm.

" " Calcium = 0.024 gm.

" " Albumin = 0.060 gm.

Urea was not estimated. In order to ascertain whether there was any hyperacidity of serum in affected cases, the PH values of some healthy and eight haematuria subjects were determined colorimetrically, with the result that the former gave 7.6 and the latter 7.5. There was therefore no marked discrepancy, but whether a large proportion of neutral salts of acids were present was not determined.

With regard to the urine itself, its specific gravity, reaction and composition were also studied. In our experience, the specific gravity of haematuria urine varied from 1020 to 1035 in different individuals, and from the observations made on several animals, the rise and fall in the specific gravity did not appear to bear any relation to the percentage of blood present in the urine at different intervals. The reaction of the urine in each case was found to be alkaline to litmus, phenol-red, and brom-thymol blue. Samples of urine from three cases were examined for the presence of oxalates, sugar, etc., with negative results. As was to be expected, albumen was present.

**Genesis of the Definite Clue.**

The etiological researches upon haematuria took a definitely hopeful turn on the 7th of March, 1932, when the author discovered some very interesting 'bodies' in the course of histological studies upon specimens of preserved bladder and kidney tissues, collected from local haematuria cases (Plate XXIV, Fig. 2). When, some peculiar spherical bodies were detected in the urine of all the six clinical cases of the disease then at Muktesar, the securing of 'data of considerable value and promise' was mentioned in the Annual Report of this Institute for 1931-32 (p. 15). They appeared to differ from body cells. They were globular bodies whose nuclei could be discerned with difficulty in unstained wet smears; these bodies appeared to be absent from the urine from healthy cases, but were present in the quiescent stages of haematuria. To ascertain if these bodies were present in samples of haematuria urine from other provinces in India, enquiries were made from the Heads of Veterinary Departments in the provinces, where the disease occurs—viz., Bengal, Madras and the Punjab. On the arrival of samples
from the provinces the same obscure structures were detected, and the possibility of a Schistosome origin was suggested. In the Annual Report for 1932-1933 (p. 15) the failure to obtain confirmation of the above suggestion has been mentioned, but the observation of 'a definite host reaction' against the 'bodies' left no doubt that they were parasitic and foreign (Plate XXIX, Fig. 1). In size they were larger than the usual body cells, and they definitely had a greater depth of focus. Since an occasional 'body' appeared to have engulfed rare red corpuscles, or to possess a vacuole they were described in the above Report as 'peculiar macrophage-like cells,' and the possibility that these were *Entamoeba* was pursued by means of the cultural technique of Boeck and Drbohlav [1925]. The first results have already been mentioned in the Annual Report of 1932-33. Before giving a preliminary description of the causative parasite, which appears to be responsible for this disease, it is essential that the minute histology of the condition be first described in the light of fresh knowledge, since the current views are faulty in some respects.

**Pathological histology.**

The Urinary Bladder. One would not expect to detect a case of bovine haematuria at its very commencement with a view to studying the pathological process in its earliest stage. Young and old lesions are, however, present in most cases and these may be utilized for histopathological studies. The disease is primarily an affection of the submucous layer, inflammatory exudation into which leads to necrosis and consequent ulceration of the mucous membrane. The bladder is intensely congested, and the blood vessels supplying the mucous and submucous layers are greatly dilated (Plate XX, Fig. 2). The capillary vessels are often ruptured, and extravasation of blood around them is to be seen. In some cases however the whole of the bladder wall is extensively infiltrated with blood and its pigments. The vascular endothelium is damaged to a certain extent, and the exudation of serum to the perivascular regions leads to a pushing apart of the surrounding connective tissue. Sometimes oedema is very pronounced, and is easily discernible immediately below the muscularis mucosa and in the strands of tissue, which intervene between muscle fibres.

The diversity of the pathological processes and the extent of their severity seen in each case, depend upon the degree of the parasitic infestation, rate of division of the parasites, and upon whether the lethal effects are repeated over a length of time. The susceptibility of the affected bovine or its bodily resistance must also be a factor of importance in the pathogenesis.

As a result of the inflammatory exudation, the epithelial lining of the bladder is swollen and elevated, the inflammatory products separating the mucosa from the thickened submucous layer. The extravasated blood and exuda-
tion are partly absorbed or removed. When the amount of this is more than can be conveniently disposed of, degenerative changes in the overlying epithelium leads to the formation of ulcers by sloughing. Blood is then easily lost from the widely-open sub-epithelial vessels, and haematuria manifests itself. If the parasitic incitants have been removed with the slough and haemorrhage, and a fresh batch of parasites are not present in the immediate vicinity, organisation of the broken epithelium commences. Clots are first formed on the raw surfaces and local capillary thrombosis may take place. Extravasation of blood ceases and haematuria disappears for the time being. The seat of attempted cicatrisation remains a weakened spot, and further progress of the recuperative process may be undermined by the intervention of a fresh batch of the parasites. Even if the obliterating clots or thrombi were to persist and the lesion were to be completely resolved, fresh lesions appear elsewhere with the reappearance of haematuria. The disease thus obtains a foothold, and tends to become chronic, and the diverse types of lesions, known to characterise the disease, manifest themselves. The lining epithelium of the bladder may still appear to be in tact in the major part, but depressed ulcers of small size with raised edges, or even coalesced patches of ulceration are to be seen. Generally these areas of ulceration are superficial and do not extend much below the submucosal layer, but deep ulceration may at times be encountered as well. Immediately under the epithelium and extending only a relatively short distance into the deeper tissues are blood cavities and channels of various sizes and shapes (Plate XXII, Fig. 1). The connective tissue stroma, which supports these extremely vascular or haemorrhoidal areas, appears to have undergone some hyperplastic changes. Apart from the several scattered points where the denuded areas of mucous membrane are in the process of cicatrisation, one notices a proliferation and thickening of the epithelial lining. Obviously the series of changes through which the affected mucosa passes must depend upon the severity of the traumatic injury or irritation, and also upon the chronicity of the case. In such areas of the mucosa, where the traumatic influences have not directly operated, or where they have not been unusually severe, the intact epithelium has to adapt itself to the mild irritations to which it is being continually subjected from time to time. The bladder tissues attempt to adjust themselves to the changed circumstances by the production of branching cauliflower-like tassels of soft papillomatous growths. The character of the epithelium of the bladder is maintained over these growths, and branches of blood vessels are carried into each prolongation. A few pyriform mucoid cysts are formed since the escape of the secretion of the glands of the bladder is obstructed (Plate XIX, Fig. 1).

The fundamental lesions of the disease are however formed in the small and large-sized vessels, which show definite reactions within and around themselves, irrespective of their situation in the thickness of the bladder wall. The submu-
cosal vessels are the first to exhibit the lesions of the disease, and like inflammatory conditions in other diseases, new vessels are produced in this situation. The majority of the vessels are more or less dilated, and this dilatation is accentuated in some places more than in others. The endothelium of individual vessels shows proliferation and desquamation, and the lumen contains some of the active forms of the parasite. If thrombosis of a vessel occurs, and this is aided by the parasites being in the sluggish stage, the arrest of the parasites takes place (Plate XXIX, Fig. 1). A process of fibrous encapsulation from the periphery inwards takes place, and an attempt at the digestion of individual parasites is made by the body cells (Plate XXIV, Fig. 2). The affected vessels undergo varying degrees of endarteritis obliterans. Some vessels are actually obliterated, and in others partially sclerosed, a few parasites may still be seen to persist (Plate XXIX, Fig. 1). The localization of parasites in altered vessels has been seen at all depths to the subserosa.

In the continuity of the bladder epithelium, one often notices several solid nests of epithelium, which are generally disposed in the form of rounded adenomatous cysts but apparently without any central lumen. Various stages of development and degeneration of these epithelial nodes are seen (Plate XXIII, Fig. 1). As these nodes enlarge, their central zone shows some thinning, while a few prominent nucleated "bodies" are clearly seen. With further development of these nodes, the nucleated "bodies" show more structural differentiation, and when the evidence of rare parasitic encystment is observed one has no hesitation in recognizing the initially undifferentiated nucleated "bodies" as the forerunner of the larger sized parasites or of their encysted fellows (Plate XXIII, Fig. 2). The collection of several layered epithelium in nodes appears to be more frequent than the normally existent mucous-secreting glands of the bladder, and evidence seems to be available to show that a number of these nodes may originate from the bladder epithelium (Plate XXII, Fig. 1). Since a number of such nodes are also seen to be present in the depth of the bladder, there need be little doubt that they can originate from blood vessels. Further it seems possible that the papilliform processes of the mucosa, as they grow, may close and form tubular adenomatous structures. Degenerative changes in the nodes extend from the centre towards the periphery, and in the advanced stages all the epithelial cells may be destroyed, giving place to a collection of an increasing number of parasites. The enclosing membrane then bursts, and the parasites are liberated into the urine (Plate XXV, Fig. 1).

In the most oldstanding cases, the blood vessels at all depths of the bladder wall show an extreme proliferation of the endothelium and appear as only collections of epithelial cells, bearing little or no resemblance to the original blood vessels. In some cases these epithelial cells exhibit a definite infiltrative type of extension, and show unmistakable tendency to simulate malignancy (Plate XXVIII, Figs. 1
So-called "bird's nest bodies," of epithelioma are however never to be seen, and real metastasis into other organs has not been found.

Another abnormality present in the sections consists of scattered minute collections of cellular infiltration, immediately beneath the epithelium and also around the ulcerated or haemorrhagic lesions. The cells are generally of the mononuclear type, consisting of young fibroblasts, lymphocytes, and endothelial cells with an admixture of so-called plasma cells. The most interesting picture is however to be seen below the clots and ulcers. The central core of an ulcer consists of necrotic material with cellular debris and fibrin. Around the degenerative core is a cellular reaction zone, the margins of which contain the invading parasites. Plate XXI, Fig. 1 represents an active haemorrhagic ulcer involving the mucosa and submucosal layer. Just below the haemorrhagic ulceration a colony of a number of parasites, enclosed in a capsule and lying in a pool of acute extravasation and with little attachment to the supporting bladder tissue around it, is noticed (Plate XXI, Fig. 2). This picture leaves little doubt about the parasitic etiology.

In regard to the parasites, it has been stated that they are found in the epithelial nodes on the lining membrane of the bladder, and in the lumen of blood vessels which are more or less altered. The occurrence of the parasites in the reaction zone around the degenerated areas of the submucosa has also been mentioned. Further the parasites may be found considerable distance beyond the initial reaction zone, and may even be seen making their way along the fibrous septa between the muscle bundles. The parasites may be numerous in the active lesions, but occasionally it may be difficult to find them. In the chronic cases, the muscular layer and the subserosa may become involved. In the more acute cases however, the parasites are situated exclusively in the submucoa. In acute attacks and early lesions, the parasites are easily recognised but in chronic cases this may need some perseverance.

Kidney. The parenchyma of this organ shows haemorrhagic areas, and cloudy swelling in places. The glomeruli show varying degrees of dilation and degeneration and the epithelial cells of the tubules are unrecognisable. The parasites have been detected in this organ also. (Plate XXVI, Fig. 1.)

The Parasite.

The parasite varies in shape and size, and can be seen both in a free and an encysted stage. Its cytoplasm appears to be sharply defined, having an alveolar or reticulated appearance. Inclusion bodies and ingested red corpuscles have been seen, but bacteria seem to be singularly absent amongst the ingested material. The parasite possesses definite histolytic powers, and appears to grow by the absorption
Free hand drawing illustrating the characteristic features of the parasite, as seen in Sections.

Figures 1, 2, 3 and 4. Parasites showing cytoplasm, nucleus, karyosome and linin net-work.

Figure 3. Shows nuclear division into four, and Figure 4 shows a parasite divided into two.

Figure 5. Shows rod-shaped chromatoid, body and glycogen vacuoles.
Figure 6. Shows a fragmented nucleus.
Figure 7. Shows a nucleus and glycogen vacuole.
Figure 8. Dividing nucleus and irregular shaped parasite.
Figure 9. Divided nucleus with an irregular mass of glycogen.
Figure 10. Two ingested red cells and glycogen vacuole.
Figure 11. Dividing nuclei.
Figure 12. Massing of chromatin into three irregular clumps.
Figure 13. Inclusion bodies in parasites. Section stained with iron haematoxylin.
of food material through its body surface. Granules of blood pigment have been seen in its cytoplasm, and the ingested erythrocytes appear to have undergone some shrinkage. In the smallest sizes, the cytoplasm is clear and shows no ingested bodies. Contractile vacuoles do not appear to be present, but so-called chromatoid bodies and glycogen vacuoles are occasionally found. Microphotographs of collections of the parasite as seen in histological sections are appended hereto. A diagrammatic representation of the characteristic features of the individual parasites is given (Plate XXIX (a)). In shape the parasites are usually spheroidal (Plate XXVII, Fig. 2), but irregular shapes are also seen. Occasional prolongations of the cytoplasm (Plate XXV, Fig. 2), are met with, and these are probably pseudopodia. Some parasites are elongate, as seen in (Plate XXI, Fig. 2). No suggestion of the presence of flagella or cilia have been detected. Perhaps the spheroidal shape indicates a state of rest, and the elongate shape that of activity. Differentiation into endo- and ectoplasm is not discernible in sections, but the sharp outline of the parasite obviously indicates such a hyaline ectoplasmic structure. The nucleus of the parasite is the most outstanding feature. It is vesicular and round, and possesses a centrally situated dot, the karyosome. The nuclear membrane is very definite, and presents on its inner surface a series of bead-like small granules of chromatin arranged uniformly. The clumping of the peripheral chromatin beads into three irregular masses on the internal surface of the nuclear membrane has been seen in one or two exceptional parasites (Plate XXIX (a), Fig. 12). From the karyosome a network, presumably of linin, is seen to radiate to the nuclear membrane. On careful examination, the nuclear membrane is found to be an even circle of highly refractive material of some depth of focus. The encysted and free forms of the parasite, encountered in sections from half a dozen cases of haematuria have been measured in microns as follows:

<table>
<thead>
<tr>
<th>Free forms.</th>
<th>Cysts.</th>
</tr>
</thead>
<tbody>
<tr>
<td>41·3 x 12·0</td>
<td>44·0 x 18·7</td>
</tr>
<tr>
<td>33·3 x 13·3</td>
<td>39·3 x 14·7</td>
</tr>
<tr>
<td>34·7 x 14·7</td>
<td>48·0 x 12·0</td>
</tr>
<tr>
<td>29·3 x 17·3</td>
<td>53·3 x 10·7</td>
</tr>
<tr>
<td>34·7 x 14·7</td>
<td>41·3 x 17·3</td>
</tr>
<tr>
<td></td>
<td>14·4 x 9·4</td>
</tr>
<tr>
<td></td>
<td>28·3 x 28·3</td>
</tr>
<tr>
<td></td>
<td>24·3 x 17·5</td>
</tr>
<tr>
<td></td>
<td>31·7 x 22·9</td>
</tr>
<tr>
<td>Free forms.</td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>----------------</td>
</tr>
<tr>
<td>29.3 x 13.3</td>
<td>30.0 x 12.0</td>
</tr>
<tr>
<td>24.0 x 10.7</td>
<td>41.3 x 21.3</td>
</tr>
<tr>
<td>16.7 x 9.3</td>
<td>36.0 x 17.3</td>
</tr>
<tr>
<td>21.3 x 10.7</td>
<td>40.0 x 13.3</td>
</tr>
<tr>
<td>13.3 x 9.3</td>
<td>40.0 x 10.7</td>
</tr>
<tr>
<td>22.7 x 16.0</td>
<td>36.7 x 32.0</td>
</tr>
<tr>
<td>33.3 x 29.3</td>
<td>48.0 x 32.0</td>
</tr>
<tr>
<td>41.0 x 25.3</td>
<td>46.7 x 20.0</td>
</tr>
<tr>
<td>24.3 x 21.4</td>
<td>44.0 x 16.0</td>
</tr>
<tr>
<td>30.0 x 16.4</td>
<td>40.0 x 16.0</td>
</tr>
<tr>
<td>17.5 x 17.0</td>
<td>55.0 x 40.0</td>
</tr>
<tr>
<td>28.5 x 30.0</td>
<td>30.7 x 25.3</td>
</tr>
<tr>
<td>15.7 x 12.8</td>
<td>25.3 x 22.7</td>
</tr>
<tr>
<td>34.7 x 14.7</td>
<td>34.7 x 30.7</td>
</tr>
<tr>
<td>33.3 x 13.3</td>
<td>30.7 x 25.3</td>
</tr>
<tr>
<td>37.3 x 9.3</td>
<td>18.6 x 16.4</td>
</tr>
<tr>
<td>32.0 x 20.0</td>
<td>20.0 x 20.0</td>
</tr>
<tr>
<td>60.0 x 14.7</td>
<td></td>
</tr>
</tbody>
</table>

The cysts are rare in sections and are found lying somewhat separated from the host's tissues. A thick double-contoured capsule with the evidence of one, two or
three nuclei has been seen (Plate XXIII, Fig. 2 and Plate XXV, Fig. 1). The size of the parasites is important in order to distinguish them from body cells, such as, epithelial cells, pus cells, macrophages or other bloated cells. The size of the double contoured cyst is helpful in the identification of the parasite. It is in the smallest stages of the parasite that one has to be particularly careful in distinguishing them from bloated or wandering cells.

In the nondividing stage, the nucleus of the parasite varies from 5·7 to 7·3 microns. Nuclear division takes place by a process of binary fission, and the maximum of up to four nuclei has been seen in dividing individuals.

The parasite is best studied under the 1-6th objective with frequent use of the 1-12th oil immersion lens. It has also been studied in the fresh condition in wet smears, prepared from the clear and bloody urine obtained from haematuria cases. Since urinary sediments contain a variety of cells one has to examine the former from a large number of healthy cattle, before forming conclusions regarding the structures encountered in the urine of haematuria subjects. Parasites of spherical shape, which may show an ectoplasm, and several times larger than body cells, with inclusion bodies and a hazy nucleus in unstained preparations, are to be seen in haematuria urine. Highly vacuolated and granular bodies probably represent degenerated parasites. Spherical double-contoured encysted parasites, the largest measuring about 35·4×30·8 microns, are to be found. Rarely a small pore-like structure has been discerned in the cyst wall. On the warm stage parasites with digit-like or blunt dome-shaped pseudopodium have been seen. Under the microscope the parasites have not shown any attempt to cross the field nor have any simultaneous retraction and protrusion of processes been seen. They would thus appear to be in a somewhat moribund state in the urine.

From the above description, it will be appreciated that the parasite has no features of the phylla ciliophora, mastigophora and sporozoa, but would appear to belong to phylum Rhizopoda. The parasite is characterised by a nucleus with a small spherical karyosome and a peripheral layer of fine chromatin beads, justifying for it a place in the genus Entamoeba. The size of the free forms and the cyst is much larger than that of all the known species of Entamoeba, and the host is a bovine. Definite histolytic powers are possessed. In view of these considerations, and the fact he has received considerable help from his wife Mrs. Kamala Datta the author suggests the designation E. Kamala, n. sp. for the large protozoan parasite causing bovine haematuria.

**Discussion.**

It will be of interest now to consider whether the course and nature of bovine haematuria, as also its general morbid anatomy and histology, are in conformity with what is known about natural and experimental amoebiases.
In both amoebiases and bovine haematuria, one notices a progressively, mild chronic course with periods of quiescence between exacerbations, and the occurrence of lesions in apparently healthy cases. Moussu’s [1905] case, already mentioned, was known to suffer from attacks of haematuria for 20 years, and similar remarkably persistent cases of amoebiasis in man have been encountered. With regard to the possibility of haematuria and the bladder lesions, as encountered in cattle, being due to amoebiasis, considerable support is available in relevant literature. Of the numerous cases of amoebic cystitis associated with haematuria which have been met with in the diverse races of the world, authorities in protozoology are prepared to accept notably few cases as genuine amoebic infections, such as Walton’s [1915], Fiseher’s [1914], and Petzetakis’ [1923]. Further Wirthin’s case of amoebic infection of the testis and epididymis has also been accepted. From the few cases of amoebiases recounted below it will be seen that the maintenance of an over-critical attitude from theoretical considerations of protozoology without reference to the nature of the clinical disease or of its lesions, can no longer be justified.

Commencing with such interesting cases of amoebiases in large animals, which have not found mention in any of the standard books or in the literature upon Amoebiases, one finds that Benume [1895]* published an article on “Uber parasitare Ictero-haematurie der Schafe. Beitrage Zum Studien der Amoebosporidien” and Lehman [1912] dealt with cases of amoebiases in the horse, cattle and sheep. Lehman showed that in these animals amoebae induce papillomata and other neoplastic growths. A comparison of the picture of the warty growths in sheep found by him with Plate XIX, Fig. 2, of this article is very suggestive. In discussing Lehman’s findings, an Indian Veterinarian, Valladares [1913] makes the remark that “it would be worthwhile keeping a look out with a view to ascertaining whether the amoebae as the cause of disease in the domesticated animals exist in India,” and the pertinence of that remark has been illustrated in the present investigation and also in the records by Ware [1916] and Boyd [1931] upon amoebiasis of dogs in the Madras Presidency.

Turning now to the pathology of human amoebiases, Brown [1910] states: “Not only is the submucosa actively inflamed and infiltrated but the connective tissue fibres proliferate, and minute buttons of adenoid tissue push up from below and appear as cluster of wart-like buds on the surface of the mucous membrane. * * * * None of the morbid changes of amoebic dysentery are more distinctive of the disease than the exudation of pus from minute orifices of mammillated growths on the mucosa. * * Many of the epithelial cells are replaced by mucous cysts.” Jurgens found mucoid cysts in his case of chronic

* Original not consulted.
cystitis. A resume of the early cases where amoebae were found in human urine has been given by Dobell [1919], and Fiesinger and Parturier [1926] have reviewed the subject since. In a demonstration before the Royal Society of Tropical Medicine and Hygiene, Yorke and Adams [1928] described an interesting case of ‘colloid carcinoma’ of the splenic flexure of the large intestine, in the lesions of which the presence of great numbers of Entamoeba histolytica was proved. Again in an article on “Amebic granulomas of the large bowel and their clinical resemblance to carcinoma.” Gunn and Howard [1931] describe their own experience and draw pointed attention to the occurrence of localised tumours of ameobic origin, which present the clinical and gross pathological picture of carcinoma. They proceed to cite the previous experience of Desjardins, who coined the term pseudocancer for cases of amoebiasis resembling cancer but responding to medicinal treatment. It will not be out of place here to quote Knowles [1928] who remarks that “it is just possible that amoebic infection with its consequent irritation may be one of the factors concerned in the production of primary carcinoma of the liver of man in India.

Again in the case of experimentally infected guinea pigs the occurrence of lesions resembling neoplasms is definitely known, though to Dobell and O’Connor [1921] the lesions seen appear to be peculiar.

Having seen how closely the morbid anatomy of bovine haematuria conforms to the available knowledge on amoebiasis of man and animals, one may turn to the probable method by which amoebae gain access to the urinary bladder. In cases of human amoebic cystitis, the question is still obscure. From the histological features of bovine haematuria, the blood stream appears to be the chosen route. Secondary infection of the brain, liver and other organs have been encountered in man, and the parasites have been detected in blood vessels in some of these cases, but the possibility of the blood circulation playing an important role in the generalisation of amoebic infection does not appear to have been considered at all seriously. From the studies in the present cattle disease, the need of this possibility being investigated is emphasised.

**Conclusion.**

Definite evidence has been put forward in this article to show that the hitherto obscure enzootic bovine haematuria is a parasitic disease, caused by a large protozoan parasite. From the characters of the parasite, it appears to belong to the phylum Rhizopoda resembling Entamoeba histolytica but would appear to be a new species, for which the designation Entamoeba Kamala n. sp. is proposed.

Results of further work in connection with experimental transmission, cure, etc., which are now in progress will be published in due course.
Acknowledgment. Thanks are due to Mr. Sundar Rao, Artist, for preparing the photographic plates and to Mr. Ahmed Bux, Assistant Artist, for making the drawings. The author is indebted to Mr. Krishna Iyer, G.M.V.C., for measuring the parasites and their cysts from time to time.

REFERENCES.

— (1932-33). Ibid.
Fiesinger & Parturier (1926) cited by Knowles (1928), 80.
— (1914). Ibid. 119 and 136.
Fig. 1. Inside of the urinary bladder shows extensive cauliflower-like growths and two pyriform mucoid cysts.

Fig. 2. Two warty growths in the duodenum, a few inches from the pyloric end and the duodenal sections showed resemblance to bladder growths and the presence of two parasites.
Fig. 1 × 50. Bladder. Junction of healthy and desquamating mucosa. Surface is hemorrhagic, undergoing coagulation necrosis under the action of developing parasites situated immediately subjacent to the degenerating mucosa.

Fig. 2 × 40. Shows extreme dilation and engorgement of the capillaries supplying the mucosa and submucosa. These capillaries provide seats for the localisation of the parasites.
Fig. 1 x 244. Early lesion, reaching nearly to the muscular layer, resembling a 'typical ulcer of Harris' of human amebiasis.

Fig. 2 x 56 shows the nidus of the disease in the submucosa, represented here by a colony of parasites in a thrombosed and bursting vessel, with secondary changes in the mucosa.
PLATE XXII.

Fig. 1. Bladder mucosa shows considerable proliferation in the centre of the field, it is disposed into an adenoma-like formation. Submucosa shows so-called angiomatous pools of blood and a focus of cellular infiltration.

Fig. 2 shows warty papillomatous growths.
Fig. 1 × 143 shows epithelial nodes in various stages of development and degeneration. The swollen node, about to be shed, shows a few peculiar nucleated bodies in the thinned central area.

Fig. 2 × 216 shows a colony of large-sized parasites and a double-contoured cyst, localised in an extremely attenuated epithelial node on the point of bursting.
Fig. 1 x 335. Blood vessel shows a few parasites in the lumen and wall.

Fig. 2 x 150. Colony of quiescent parasites in broken down blood vessels. Note the subacute cellular reaction around the parasites.
Fig. 1 x 216. Colony of active parasites in the superficial lesion. Note the cyst, showing one definite, and two hazy nuclei, with a suggestive empty space.

Fig. 2 x 129. Another colony. Note the lateral prolongation on the large parasite on the top right hand sides of the field.
Fig. 1 x 166. Section of the kidney dilated blood vessel shows parasites in the lumen. Note the difference in the appearance of the parasites, compared to the endothelial and other cells in the field.

Fig. 2 x 128. Shows four neighbouring colonies of rapidly multiplying parasites below the bladder surface. Note two cysts of varying shapes.
Fig. 1 × 335. High power photograph of a section stained with iron-haematoxylin, showing inclusion bodies inside the parasite.

Fig. 2 × 244. High power photograph showing a colony of large-sized parasites in the superficial lesion in the bladder. Section was stained intensely by Gram's method. Note the typical character of the nucleus and the shape of the parasites.
Fig. 1. Shows growths in the mucosa and submucosa resembling to an extent adeno-carcinoma.

Fig. 2. Shows infiltrating character of cell-extension in the depth of the bladder wall.
Fig. 1 x 56. Low power photograph. Section of bladder shows two altered vessels. In one, the parasites still persist, while in the other they have been digested by body tissues.

Fig. 2 x 244. A degenerated area in the duodenal lesion (Plate XIX, Fig. 2) showing a few parasites with the typical nuclei.
ETIOLOGY OF ENZOOTIC BOVINE HAEMMATURIA


Ware, F. (1916). J. Comp. Path. & Ther. 29, 126.
