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Vol. III. Nos. 2 & 3.

THE MADRAS VETERINARY JOURNAL

CONTENTS

1 SEP 1930

EDITOR

THE OFFICER IN CHARGE IV CIRCLE, CIVIL VETERINARY DEPARTMENT, CHEPAUK, MADRAS.

Published under the Supervision of the Chief Superintendent, Civil Veterinary Department, Madras.

Subscription Rs. 2 per annum or price per copy As. 6 only.

Madras Veterinary Journal.

V3 No 2 and 3

May ~~March - May~~ 1924

196951

EDITORIAL.

It is with regret that we have to draw the attention of subscribers to the fact that the literary support received is so poor that we have had to wait for a long time before gathering the necessary matter we require for a number and with some of the articles quite recently received we have compiled the two numbers due. May we hope that at least hereafter we get sufficient encouragement from the profession for whose benefit this Journal was started. To all earnest supporters of the Journal we make an appeal once again and trust that our appeal will not be in vain.

In this issue of our Journal we are printing an interesting article on Necrosis of tail in a buffalo. This reminds us of an allied affection in the tuft of the tail viz. Trychorrexis nodosa. The hairs become nodular, split and break and finally the tail, rather the tuft is denuded of all hairs and perhaps the skin also becomes affected at last and we think that it is one of these kinds of tail eczema maltreated that has resulted in Necrosis of the tail. However, as the author of the article says that occasionally cases like these are seen in that locality, we commend them to those who have time enough at their disposal to make an investigation into the matter.

The essay on Inflammation read by a final year student before the Madras Veterinary College Medical Association should indeed be an incentive to such of those of the profession in this presidency as have not hitherto contributed a single article to the Journal.

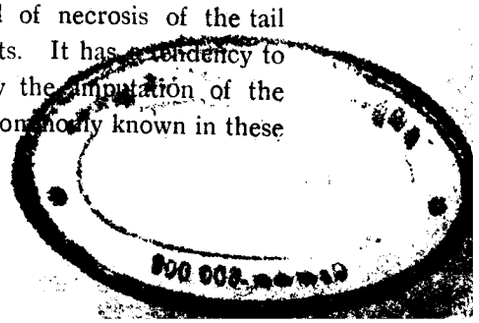
NECROSIS OF TAIL IN A SHE-BUFFALO.

BY

C. Suryanarayanamurthi, G.M.V.C., Veterinary Assistant Surgeon, in charge of the Veterinary Hospital, Rajahmundry.

Subject.—A she-buffalo in advanced pregnancy, aged about 9 years, and local bred, the property of a ryot living in a village near Rajahmundry.

Nature of disease.—It is a peculiar kind of necrosis of the tail which is occasionally met with in these parts. It has a tendency to extend rapidly upwards unless arrested by the amputation of the diseased part of the tail. The disease is commonly known in these



parts as "*Thoka Puli*" in Telugu, which literally means "*Tail-tiger*" that is to say, a disease which eats up the tail so rapidly and voraciously as a tiger does its prey.

History of the case.—The case was brought to the hospital for treatment on 11th August 1923. The previous history of the case is as follows :—

Some 3 months prior to the admission of the animal in the hospital, the owner noticed the tip of the tail being diseased. He neglected it for some days, but afterwards tried some country medicines, but with no avail. He found the wound getting worse and extending up. At the suggestion of a quack, he cut the diseased part of the tail and immersed the cut-end in boiling oil. Probably he left a portion of the diseased tail, as he found that the wound had no tendency to heal, and that the inflammation was extending up. After some time, he again docked the tail and immersed the end of the tail in the boiling oil, as before. Even then, the result was the same. Thus he went on cutting the tail 4 or 5 times until he cut its very root and could cut no further. Thus the condition of the poor animal was as follows when it was brought to the hospital for treatment :—

The wound was awfully stinking. No coccygeal vertebræ were seen at all or felt. There were two wounds-one at the root of the tail where it was cut for the last time, and the other behind and below the sacrum. The latter wound was gaping with sacrum protruding and forming a roof as it were, over it. The posterior half of the sacrum was necrosed and detached from its attachments, while its anterior part was healthy and firmly attached to the muscles. On exploration of the wound, I found it to be 9 inches deep and directed forwards, downwards, and underneath the sacrum. On probing the wound with my finger, I found three coccygeal bones lying loose inside the wound. What happened was, that after the tail had been cut at its root, the wound burrowed inside and formed a big sinus underneath the sacrum with no drainage. The first three coccygeal bones, as they got detached gradually from attachments, gravitated into the sinus, which was, as I said above, directed forwards and downwards. This was the state when the case was brought to the hospital on 11th August 1923.

Treatment.—The wound was thoroughly washed with an antiseptic lotion. The opening of the wound was widened. The loose

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coccygeal bones were removed and proper drainage established. There is nothing worth mentioning about the treatment adopted. The wound was daily washed and dressed antiseptically. The whole of the sacrum gradually necrosed and came off about the 5th of September 1923, excepting small pieces at its antero-lateral corners which articulate with the ilium. These pieces were then firmly attached to the tissues, but were gradually removed after the animal calved on the 16th September 1923. The wound commenced to heal rapidly afterwards and on 24th October 1923 more than half of the wound healed up. The case was seen by the Superintendent, C.V.D., I Division, Vizagapatam during his inspection of this hospital on 15th September 1923. From the 28th October 1923, the owner discontinued bringing the animal owing to some domestic inconvenience and I understand that the animal subsequently had a severe form of diarrhoea and died.

Interesting points about the case.—The following symptoms were seen, in consequence of the loss of sacral and coccygeal portion of the spinal-cord, although the important centres such as ano-sphincter centre, parturition centre etc., are situated in the lumbar portion of the cord :—

i. The tone of the sphincter ani was lost. The dung used to trickle down frequently.

ii. The muscles of the hind limbs were slightly emaciated. They seemed to have lost some of their tone, as the hind legs used to be bent more than usual while standing and during progression, as if unable to bear the weight of the animal.

The animal was otherwise perfectly all right and digestion was quite unimpaired. She had easy and safe parturition, and gave birth to a fully developed calf.

THREE DAYS' FEVER OF CATTLE OR "BOVINE FLU" AS I CALL IT.

BY

V. V. Venkatachala Ayyar G.M.V.C., Veterinary Assistant Surgeon, Gudalur.

In the months of August, September, October and November of last year there was a fairly good number of attacks among cattle of a

variety of Fever which I have for the present put down as an influenza fever. It was prevailing in Gudalur town and an adjacent estate about 8 miles therefrom. This disease afforded rather quite an interesting study not only on account of the scarcity of literature on its Etiology and treatment but also from its close similarity to other diseases affecting Bovines, particular mention of which will be made in a succeeding paragraph. In this short paper I have tried to analyse the subject critically in all its different aspects and present the facts as they were observed by me.

Definition.

It is an acute contagious febrile disease of bovines characterised by the sudden onset of febrile symptoms lasting for about three days, followed by the appearance of slight metastatic lameness and ending either fatally or in recovery.

The question of contagion.

The attacks were confined to two places of the taluk only as has already been mentioned. The method of introduction of the disease into the taluk cannot be ascertained with any degree of certainty and curiously enough it looks like one of indigenous origin. Every successive attack after the initial one was noticed only in those animals that were in contact or those that were mingling with infected ones on common pasture. In the absence of attacks among cattle of uninfected localities and on account of fresh outbreaks occuring among cattle of the infected places I am strongly inclined to believe that it is contagious and perhaps infectious too.

Etiology and Bacteriology.

The cause of the disease cannot be definitely ascertained without laboratory experiments conducted on an extensive scale. But on account of its origin in the winter months, exposure to cold and rain undoubtedly acts as a predisposing factor. During the course of the outbreak six blood smears from live animals and one smear from a dead animal were examined at the Madras Veterinary College and also locally : but no organisms could be traced in any of these.

On making a post-mortem examination of the last animal that died of this disease it was found to have suffered from acute congestive pneumonia and smears from the seat of the lesion and also from the various internal organs were examined at the Madras Veterinary College. The one from the lesion was reported to contain

"varied organisms including a few pasteurilla." This Bipolar organism is perhaps likely to be the cause of the disease in as much as all the diseases originating from Pasteurilla group of organisms are found to run an acute course in practice. Apart from the academic interest attached to this, the solution of the question is of practical interest from a professional point of view.

Distribution.

The disease first made its appearance during the heavy rains and with the approach of bright sunshine it had a tendency to lose its virulence and subside. Hence this seems to be another complaint which has to be added to the long list of cattle pests essential to winter.

History of the attacks and symptoms.

As a rule the history of the cases that came to my notice was quite constant and almost similar. An animal which was quite healthy the previous evening was reported to have been badly taken ill with fever the next morning. Two of the animals had died without any warning of previous infection, after a very brief period of illness lasting for about 2 or 4 hours in the night.

The attack was ushered in with rigors, staring coat, drooping ears, and dry muzzle. The animal was off feed, dull, mucous membrane congested and bowels constipated. The temperature was found to be ranging between 103° F. and 104.6° F. A couple of hours after these symptoms commenced there was slight dribbling of saliva. Respirations and pulse were found to be accelerated. These indications lasted from one to three days, on the second or third day a slight metastatic lameness appeared. In animals that recovered improvement took place by the wearing out of these symptoms after the third day.

Mortality.

Of the 13 cases that I saw 3 died suddenly without any treatment. The other 10 cases recovered after treatment.

Post-mortem lesions.

The following post-mortem lesions from the last case that died may be interesting as a guide.

Subject. A country bred cow, aged 7 years, in good condition.

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External appearance. The abdomen was tympanitic and rectum partly everted.

Skin and subcutaneous tissue. Normal.

Abdomen.

Peritonium—normal.

Stomach and intestines—normal and contained a fair amount of ingesta.

Liver—studded with a lot of cysts—otherwise normal.

Spleen—normal.

Thorax.

Pleura was congested.

Lungs. A diffuse venous congestion of both lungs was observed and the bronchii and alveoli showed signs of haemorrage. One bright patch of congestion was conspicuously noticed on the right lung.

Heart. Pale, flabby and contained clotted blood.

Cause of Death.

Acute congestive broncho pneumonia.

Treatment.

Good hygienic surroundings, warmth, rest and soft liquid diet coupled with internal antiseptic treatment have given me most encouraging results. A few of the cases were given one dram dose of acid carbolic internally every two hours. But on account of the large quantity of the drug which had to be given repeatedly by the mouth this was subsequently replaced by the hypodernic injection of one dram of acid carbolic, diluted with half an ounce of distilled water for adult animals. Those under two years received half the above dose. One such injection had a decidedly good curative effect.

Conclusions.

This complaint is likely to be confounded with Anthrax, Black quarter, and acute Rheumatic fever. It is almost impossible to differentiate it from anthrax except by microscopic examination. Otherwise the sudden onset, acute and short duration and the external appearance of the carcase of an animal died of this disease closely resemble those of anthrax.

It could be differentiated from Black quarter by the absence of any external local lesion so characteristic of Black quarter.

From acute Rheumatism it could be differentiated by the absence of very severe lameness or inflammation in any particular joint.

Nomenclature.

The question of identifying the disease from its curious bearings is rather difficult to me. Whether it is an ephemeral fever or a three day's fever or an Influenza affecting cattle it is not possible for me to say, but from its occurrence in the winter, its similarity with human influenza, the pneumonic lesions and the appearance of lameness which, I think, is due to an Idiopathic Neuralgia, I have called it " Bovine flu ".

I would finally request all my enlightened colleagues in the field to throw more light on the subject if they have come across this complaint anywhere else.

TREATMENT OF OTORRHOEA IN DOGS.

BY

M. Velu Pillai, G.B.V.C., Assistant Veterinary Surgeon, Penang Municipality.

During last year more than a dozen cases of Otorrhoea were treated in the animal Infirmary, Penang and treatment was adopted in all these cases with Methylated Spirit and Boro Iodoform.

This method was conducted under the able guidance of Captain T. W. Wright, M.R.C.V.S., the Head of the Municipal Veterinary Department. In all these cases, the affected ears were first cleaned out thoroughly and swabbed with Methylated Spirits and a little Boro Iodoform was well dusted into the ear. The effect of this treatment was splendid and recovery more speedy. No relapse was noticed in any of these cases with this system of treatment. I wish that my professional friends in India might give this a trial.

CHRONIC RHEUMATISM IN BULLOCKS

BY

N. Ramanadhan, G.M.V.C., Touring Veterinary Assistant Surgeon, Kamalapuram.

Chronic Rheumatism seems to be very common in bullocks of the ceded districts where groundnut husk (in addition to cholam, ragi

and paddy straw) forms part of their fodder. The affected animals are stiff all over the body, have a cramped gait of the fore and the condition seems to be worse after rest. Defecation urination and temperature are all quite normal.

The etiological factors of the disease are :—

(1) Groundnut husk as fodder.

(2) Drinking lime well-water (cattle living in river side villages being free from the complaint).

(3) Flooring the byres with Cuddapah slabs which get very cold in winter owing to the cool weather, also in summer with urine of animals housed in them. Breezy weather and over-work are also predisposing causes. The administration of salines with pot. iodide, heart tonics and soda salicylas has not been very popular and encouraging so far, though their use continuously for one month shows decided improvement. Ignorant ryots cannot be convinced on such chronic diseases as they expect immediate magical cure. Moreover cost of treatment for a month is also a consideration for poor ryots. Hence I request my professional brethren to let me know through the Journal any injection (such as Veratrine) they tried with success, and whether the Rheumatism Phylacogen used with varying success in human patients produces the same result in our patients also.

YOKE GALL

BY

M. Ramaswami Ayyar, G.M.V.C., Veterinary Assistant Surgeon in charge Veterinary Hospital.

The most common surgical disease met with in all hospitals among working cattle is the yoke gall. More than 40 per cent. of the cases attend the hospital for 'Sore neck' 'wound neck,' 'abscess neck' and 'Tumour neck.' Cattle are provided with very strong and thick muscles on the neck and treatment of deep wounds and sinuses thereon takes a long time to heal, to the anxiety of the Vet. on one hand and disgust of the owner of the animal on the other. As Mr. P. S. Kuppusami Ayyar rightly observes, the dense ignorance of the ryot who will never faithfully follow the instructions of the Doctor makes matters worse. As an instance I might mention here that a case of deep seated abscess of the neck was operated upon at Venkatapuram about the beginning of 1922. The animal was treated at

this dispensary for about six months without any appreciable improvement and the owner naturally getting disgusted with sending the animal to the dispensary took it away to his place which is about three score miles and ten from here. Then about the latter part of 1922 he sent the animal to me evidently with a view to see if a new man—it was only a couple of months since I had taken charge—could do anything for it. At that time the animal had a burrowing wound allowing the probe to pass to about 7 inches and discharging white thick pus. As the owner was not willing to permit me to open the sinus and thus give scope for drainage I had to contend myself with syringing the sinus with antiseptic lotions and after properly drying the sinus, injecting Tincture Iodine and Glycerine or Tr. Benzoin Co. I might here mention that Tr. Benzoin Co. is very good, where the sinus is of slender bore, in closing it up. I conttinued this treatment for more than a fortnight and the sinus was gradually filling but the discharge of pus slightly increased when the owner stopped sending the animal to the dispensary. I now understand that the discharge continues and the unhealthy wound right on the yoke place has made the animal absolutely useless. The owner will never believe me when I tell him if he had permitted me to open the sinus the wound might have been healed ; but he will only say in the characteristic tone of the dissatisfied ryot that he had had enough with one operation. I have related this fact here just to show that until the ryot understands that once an animal is taken to the hospital it is in the hands of a professional man who should be given free hand to do what he considers best our difficulties will never end.

Yoke galls are induced by a variety of causes the chief among them is the unequal balancing of the cart. The constant cry of bandymen in the south especially in the Tanjore and Trichinopoly districts about insufficient or extra weight முன்பாரம் and பின்பாரம் and the exchange of the fattest and leanest from the front to the back of the bandy and *vice versa* should be known to many of my friends. The weight in the bandy should be so distributed that when we raise the shaft the whole bandy should balance on the axle for a few seconds and then go down. This is the correct way of loading a bandy, says an experienced bandy man. If the weight is not equally distributed particularly when there is too much weight in front, on a journey down a slope, the yoke exerts a very great amount of pressure on the neck. It might have been observed that bandymen in the south hang on to the back end of the centre shaft when the bandy goes down a slope and sit far in front when it goes up a slope.

Yoking bullocks of unequal height and capacity disturb the neck badly. This becomes worse on a bad road.

Very often inflammation of the neck is caused by turning the bullocks yoked in a bandy round a corner suddenly. When a bandy has to be turned round, say to the left, the driver holds the left hand rope tight and gives a sudden thrust on the left hip of the animal on the right. The right hand animal swerves far away from the bandy while the left hand animal is kept at a stand still. The friction on the neck by the yoke brings about inflammation.

Yoking bullocks not of the same height and of unequal strength always produces one or other of the neck troubles. The strong bullock always goes forward and the weaker stays behind and the latter in addition to its very often getting a taste of the cartman's cruel rod to make it keep pace with its fellow—disturbs the neck of the stronger bullock badly. It generally lags behind and whenever it receives a thrust from the cartman it runs forward all of a sudden and falls back again and thus creates irritation of the neck of its fellow. This is, I believe, the punishment for being in bad company. The disadvantages of yoking together bullocks of unequal height are quite obvious.

The ryots in these parts believe that plough-work more often than cart-work, causes yokegalls. Ploughing on hard rough soil invariably causes yokegalls. The man that drives the plough has to press the '*medi*' (that portion of the plough which the driver holds) down so that the plough can cut deep into the soil. This is difficult to do when the soil is hard. So the whole plough skips over the soil shaking the yoke badly and this causes inflammation of the neck. So when there is hard and stony ground to plough through a fairly strong man should try as far as possible to hold down the '*medi*' and prevent the plough from skipping over.

The rope that is used to tie the yoke is very often loose and this gives unnecessary shaking and movement to the yoke over the region of the neck. This neglect to pay proper attention to the tying of the rope particularly when it so happens that the weight is not properly distributed in the cart is sure to cause inflammation and swelling of the neck.

We commonly get cases of 'wound neck' and 'inflamed neck' for treatment in the hospitals. Treatment for the latter is not always troublesome. Fomentations and application of acetic acid and chalk

paste (the addition of a little gum to this paste is always useful since it gives a sticky quality to the paste and acts like a firm bandage) in the earlier stages, liniments and urguentum lodine when the swelling is hard followed by a blister (mild) if necessary would quite suffice. It is the wound on the neck that gives the practitioner much trouble, not that they are not amenable to treatment but they recur often. I have already stated and it would certainly bear repetition that it is the dense ignorance of the ryot that causes this recurrence of the wound. The anxious ryot is always in a hurry to get his animals to work. He does not give sufficient time for the repair of the mutilated tissues. The skin is very tender after the wound has healed up and when the animal is yoked in that condition it is sure to cause a wound at the same place. Once my friend Mr. Ali Hyder told me that he advises the application of tar to the neck where the yoke rests before using the animal again. I followed this advice but found the results do not justify even the cost of the tar used for the purpose. So there is nothing like giving a complete and long rest in such cases. Much has been talked about the humane method of yoking bulls and the methods demonstrated are not at all practicable. At best they show the ingenuity of the demonstrator and serve to win medals in Exhibitions. The whole of this article I have written from my personal experience. So there is much scope for criticism for thinking brains and if this article can bring about anything like a discussion from my more experienced professional brethren the objects of the writer would be fully gained.

RABIES IN A COW.

BY

K. Govindan Nair, G.M.V.C., Veterinary Assistant Surgeon, Tellicherry.

When I was on leave in the month of June 1923, I was called on to see a cow that was off-feed for 3 or 4 days with no history to guide me.

The symptoms observed on the first day of my visit were :—

The animal was distressed and weak, ears were erect, sometimes turning this way and that as if apprehending some danger. The animal refused food and drink completely—Salivation excessive—eyes restless—eyeball projecting usually and watery at the same time—started at the approach of any moving object—breathing slightly

accelerated—defaecation normal—constant attempts at urination just like an animal in season.

History :—Practically none—Diagnosis not definitely arrived at.

However suspecting for some brain affection, probably from some vegetable poison, a purgative dose of Mag. Sulph. was prescribed and I instructed the owner to hand-feed the animal with *Conji*.

I visited the animal more to satisfy myself than to oblige the owner who never sent for me. Found the animal worse than before. No purgative or *Conji* was given, as the owner said it was impossible to approach the cow.

At this stage the animal was found very wild. She completely forgot its surroundings—attacked everything indiscriminately—was quite unsteady, paralysis having set in already. She rushed against the wall, wounded herself, and attempting to scratch the head and ears, fell down, eyes were sunken and the cow was thoroughly emaciated.

This spectacle recalled to my mind a similar fate of a horse in the Madras Veterinary College, when I was a student :—Now I sent for the cowherd boy and questioned him where he was wont to take his animals for feeding, whether any dog or jackal had happened to bite the cow etc.

The boy, much to my relief, answered me directly that the cow was once bitten on the ear by a dog, which it was reported was killed when it was found attacking sheep and cow and other dogs.

Ultimately, further enquiry into the matter revealed that this cow was bitten by the same dog, and the self-same dog was suspected of rabies.

Another visit showed the animal still worse. She was completely paralysed, could not get up, but raised only her head when she heard some noise. The cow died on the 8th day of the commencement of the attack. No postmortem was held, as the owner did not want to mutilate her.

I write this only as a piece of information to my professional brethren who are juniors like myself, as some of them may not have seen cases of rabies in bovines.

FOREIGN BODY IN THE OESOPHAGUS OF A DOG.

BY

M. Velu Pillai, G.B.V.C., Assistant Veterinary Surgeon, Penang Municipality.

A young Fox Terrier Dog belonging to an European gentleman was brought very urgently to the Infirmary on the evening of December 31st 1923, with the history that the animal was unable to swallow anything. The symptoms noticed were anxious expression, difficulty of deglutition and a painful swelling on the upper part of the oesophagial region, which when slowly pressed evinced much pain.

On examination of the mouth and pharynx, nothing could be seen and all attempts to extract the foreign body through the mouth failed. As the owner was very anxious to relieve the dog of its acute sufferings, the last recourse left to me was to perform an operation on the seat of the swelling. The animal was placed on the dorsal position with the legs properly secured by hobbles and a 5% solution Novacaine was injected as a local anaesthetic after clipping hairs on the part and painting it with Tincture Iodine. A small incision about an inch was made over the obstructing body on the Oesophagial region, and on reaching the wall of the Oesophagus, a small black thing was seen projecting outwards. The projecting portion was caught with a pair of forceps and slowly pulled when a threaded rusty needle 2 inches long, was found. The seat of operation was then sutured and painted with Tincture Iodine and bandaged. Soon after removing the needle and the red thread, the animal was greatly relieved and began to take plenty of milk and water.

As there was no wound on the external part, it is evident that the needle must have had its passage through the mouth.

URETHRAL CALCULUS IN A BULLOCK.

BY

N. Ramanadhan, G.M.V.C., Touring Veterinary Assistant Surgeon, Kamalapuram.

Subject.—A bullock of Ongole breed, aged about 12 years, was brought to me on 19th August 1923 with the history that the animal has not been passing urine from 6th August 1923.

Symptoms.—The animal was very dull and depressed and the temperature was 103.4°F. Occasionally it used to nibble a few blades of straw. Sometimes it showed restlessness by lying down and getting up. There was defecation, the faeces passed being loose and very foul-smelling. Micturition was totally suspended.

Examination.—I passed my oiled hand into the rectum as the animal was standing and found the bladder distended with urine. The course of the urinary passage was examined carefully by external manipulation down the perineum and nothing hard could be felt till I reached the portion of penis 2" posterior to the external orifice where something hard was felt. I could not satisfy myself completely by verification after withdrawing the penis out of its sheath, as this process seemed absolutely impossible however much I tried, as the penis was very much retracted. Feeling certain that the calculus should either be at the S shaped curve or very near its free end, I made bold to operate.

Operation.—The preliminaries for the operation having been observed ceremoniously, the animal was placed on its back. An incision of about 5" in length through the skin, was made 4" behind the scrotal sac and after carefully dissecting the subcutaneous connective tissue, the penis was drawn out through the opening made. The seat of the calculus being now clearly located by manipulation, the penis was amputated just behind it (seat of calculus) when about a gallon of decomposed urine freely flowed out. The resulting bleeding was arrested by astringents and the urethra was kept open by fine continuous silk sutures connecting it to the surrounding erectile tissues. The stump was then pushed home and after washing the part well with antiseptic lotion and the outer skin was also sutured with intermittent ones.

As I had to go out on tour the owner was asked to irrigate the sheath well with soap and warm water daily after fomentation, and to pour medicated oily dressing into the same to act on the stump.

The interesting points to be noted here are :—

(1) Such cases seem to be very common in ceded districts where the water is running through limy soils in many places. Anuria sometimes occurs through indiscriminate drugging by quacks when the symptoms presented would be the same and history will aid in diagnosis.

(2) Amputation is preferred especially in bullocks in such cases to obviate the possibility of a stricture which would otherwise result if a longitudinal incision was made, as well as to prevent further calcareous deposit at that recumbent seat.

N.B.—The calculus was about the size of a big castor seed and weighed 15 grains. It was duly submitted to the Principal, Madras Veterinary College for preservation as a specimen.

UMBILICAL FISTULA IN A BUFFALOE CALF.

BY

A. R. Venkatachala Ayyar, G.M.V.C. Veterinary Assistant Surgeon, Tanjore.

A buffaloe calf aged about 6 months was admitted into this hospital as an out-patient on 5th November 1923. The history of the case was that from a few days after birth, there was an open wound in the naval communicating with the intestines with the result that the partly digested ingesta was partly escaping through the opening. The owner perhaps luckily for the poor being, thought of the hospital only now. The part was well washed and with difficulty, the part was made clear for a careful examination, because the ingesta continued to flow and thus soiled the part. On a careful examination, it was noticed that the opening had a direct communication with the intestines and the walls of the intestines appeared to have got adhered with the surrounding structures. If I should attempt to enlarge the external opening and try to dissect out the walls of intestines and then bring them together, the owner was not for a risky operation. The complaint was not in any way dangerous to the life of the animal since it was a long standing one. I then sutured the lips of the external wound (skin) after trimming their edges and dressed it with Tincture Iodine and Boro-Iodoform and repeated dressing the sutured surface with Tar and Carbolic acid to keep off flies for the following few days. On 10th November 1923 the sutures were found to have given way and only semi-solid ingesta was seen to escape. At this stage I had not entertained hopes of bringing about adhesion of the lips of the wounds by suturing. However, I sutured the part again and continued dressing the wound with Tr. Iodine

and Boro-Iodoform. On 16th November 1923 a portion of the sutures gave way, but this time adhesion appeared to have taken place over a small area of sutured surface. Finding myself encouraged at this, I sutured the part again for the third time. On the 17th of the month, I found only a small quantity of liquid oosing out of it. Different antiseptic dressings were tried and the case showed appreciable improvements gradually until 6th December 1923 when it was discharged cured.

THE OUTBREAK OF TRYPANASOMIASIS AMONG HORSES OF MADRAS DURING THE YEAR 1922.

BY

L. S. Parameswaran, G.M.V.C., Laboratory Assistant, Madras Veterinary College Laboratory, Madras.

Under the heading of Trypanasomiasis among cattle in the Madras Veterinary Journal No. 22 for September 1923 Mr. Ananthanarayana Rao has given a graphic description as to how he came to connect the out-break of Trypanasomiasis which occurred among horses in Madras during the year 1922 with the bullock which died in a hackney cart stand and whose blood smears revealed Trypanasomes. It is nearly two decades since it has been published in India that cattle act as a reservoir of Trypanasomes and play a prominent role in the propagation of the disease to the horses. It is not with the object of discrediting this fact that the source of infection might emanate from bullocks to horses—that I propose to write these few lines in this Journal but because I, as the then Inspector on Glanders duty for the city of Madras and one mainly responsible in detecting the cases referred to in the out-break actually traced out the disease and submitted a report personally to the Principal, Madras Veterinary College, I consider it necessary that the readers of this Journal should have a correct idea of the source of infection in this bullock.

Mr. Ananthanarayana Rao says in the article under reference that he gave the Veterinary Inspector on Glanders duty, the information about the prevalence of Trypanasomiasis in a horse. The horse in question is said to have been sent by the owner on the advice of Mr. P. Sreenivasa Rao to the hospital

for treatment, and smears from this case, as it is the usual routine, were submitted to the college laboratory for microscopical examination. As the smears revealed Trypanasomes, the horse was seized and destroyed under the act. On 14th July 1923 the place, wherein the horse was kept before the same was sent over to the hospital, was inspected by the Principal, Madras Veterinary College and the Inspector on Glanders duty and all precautions were taken, as scheduled out in the Act.

I assumed charge of the duties of the Inspector on Glanders duty only on 21st August 1922. On my usual rounds on 4th September 1923 I came across in a certain horse-bazaar, 2 ponies which appeared seedy and whose legs were rather filled in. This led me to suspect them of harbouring Trypanasomes and accordingly took smears from both of them, which were examined at the College Laboratory and proved to be positive for Trypanasomes. These were therefore seized and destroyed under the Act.

Subsequently a few days later smears were submitted by me from 3 other suspected cases from the same horse-bazaar and from one which was recently sold to a gentleman in Madras from this bazaar and these revealed also Trypanasomes. In another animal which was sent over to the Hospital for treatment by an European gentleman Trypanasomes were also found. As all these animals, when I traced them out, belonged to one and the same stable and no other cases occured in any other horse or stable which was in close vicinity and which I carefully examined, and as, further, the pony, that was first sent over to the Hospital, in whose blood Trypanasomes were found, remained also in the same bazaar, I took it that the outbreak was confined only to that particular bazaar and the duty of finding out the source of infection devolved on me.

In the course of my investigation I found that 8 ponies from the horse-bazaar under reference were taken about the middle of May 1922 to Thiruppur fair which commenced about the end of May 1922. The dealer after disposing 6 of them purchased 4 up-country ponies and returned to Madras about the middle of June 1922 bringing in all 6 ponies.

The two ponies, that I seized first, were the remnants from Thiruppur and out of the others including the one sent by the.

European gentleman, that were destroyed, 3 were the up-country ponies purchased at Thiruppur and the other two were the ponies of the same stable at Madras.

From the above it is perfectly clear that the focus of infection centred around the ponies of the horse-bazaar and those that were brought from Thiruppur. Although one cannot very definitely say which of them, either the ponies of the horse-bazaar or those that were brought from Thiruppur, were responsible for giving original infection, yet the up-country ponies, I consider, should have been responsible for the original infection since no case of Trypanasomiasis had been reported for the last several years in this particular bazaar and it is only after the arrival of these up-country ponies from Thiruppur the outbreak had been noticed. I may also add that up to the present day since the suppression of the outbreak no cases have been reported.

I shall now proceed to discuss the aspect of the question as put forth by Mr. Ananthanarayana Rao who claims to connect the source of infection to have emanated from the cattle stand. In this connection I think it will not be out of place to mention here as to how Trypanasomes are reported to be generally transmitted.

So far Glossina species are only credited with being concerned in the life cycle of some trypanasomes in South Africa. Tabanus is apparently supposed to carry Trypanasoma evansi in a mechanical way. Major Cross, Camel Specialist, says that the tick Ornithodorus crossi is concerned in the life cycle of Trypanasoma evansi in North India but the same has not yet been corroborated by other authors. Any how so far as this part of India is concerned it may be stated that no insect has been credited to be concerned in the life cycle but only Tabanus and Hippobusca species are supposed to carry the Trypanasomes in a mechanical way. The conditions necessary for the spread of Trypanasomes in a mechanical way are that affected cattle or horse should be introduced into a healthy stable or heard and also should be sufficiently near the healthy ones. Because only when a fly does not get a full meal on one animal that it goes to another to complete it and it is in this way that Trypanasomiasis propagated in a mechanical way.

So in the absence of Glossina or any other insect credited to be concerned in the life cycle of trypanasomes the disease can only be introduced into a healthy area by an infected animal, and when outbreaks occur healthy animals can be protected by keeping them at a distance from the infected ones.

Any one who is acquainted with Mount Road, Madras will know exactly where the horse-bazaar (Arab Stables) is situated and how far it is distant from the Wallajah Road Hackney Cart Stand and how far it is probable for a bullock suffering from trypanasomes, placed in the cart stand to be responsible for the spread of trypanasomiasis infection to the ponies in this particular outbreak. I have already mentioned that no case of trypanasomiasis could be seen in the vicinity of the cart-stand other than in this particular bazaar, even though there are 3 more places in the vicinity wherein ponies are stabled in greater numbers. I may also add that even though I very carefully inspected the ponies stationed in and around the Corporation Cattle Depôts wherein Trypanasomiasis had been prevailing, yet I could not find any case of Trypanasomiasis among ponies.

I do not want to go to the other aspect of the article in which Mr. Ananthanarayana Rao claims to have identified Trypanasoma evansi by measurement. How far the measurement of trypanasomes can be relied on for the diagnosis of different species, can be imagined when eminent scientists like Laveron write as follows :—

"Attempts have been made by different observers to differentiate these trypanasomes by their morphology (e.g) by the presence or absence of vacuoles or of chromatin granules in the protoplasm, the length of the body and of the free flagellum, the shape of the posterior end and so on, but it has been pointed out that such variations may occur in the same trypanasomes, consequently they are valueless in the determination of species".

CATTLE DRENCHING.

BY

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It is a known fact that ordinary method of drenching cattle with bottles or horns is not without danger of accidents. Often

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when animals struggle especially buffaloes, the mixture gets into the trachea causing mechanical bronchitis or even death. In horse it is overcome with stomach tubes. This is not supplied to all hospitals at present. Such being the case we cannot expect a contrivance in this for cattle except the probang which if used indiscriminately, may injure the oesophagus. For some time past I was trying to find out a suitable method to drench troublesome cattle. The following method was tried by me recently and found almost safe in drenching such animals. It requires an irrigating can and a rubber tube about two yards in length. The rubber should be of such a size as would fit the ordinary two pints irrigator. The mixture is poured in the irrigator after clipping the rubber. One attendant holds up the animal's head keeping it steady, another holds the irrigator up and the operator holds the distal end of the rubber in the right hand, places his left index and middle fingers over the tongue of the animal so that it may open its mouth a bit, inserts the rubber into the mouth about eight inches so that it may rest on the root of the tongue and then allows the mixture to flow little by little. The animal will swallow easily without bringing out the fluid. The flow must be regulated by the thumb and the index finger of the right hand. Allow the mixture to flow down only after seeing at each time, that what is allowed to flow down is swallowed.

For calves, small sized rubber tube about one or two feet in length attached to a clean funnel will do good. I have been trying this method for the last three months and found it to be better than drenching with bottle. Not a drop of the mixture will flow out and there is less chance of getting into the trachea as the flow is even and regulated and the animal can very easily swallow. This is found very useful in drenching with irritant drug. This cannot be used to drench oils as they have a deleterious action on the rubber. If the rubber is immediately washed with warm water and soap and the oil thus removed it may not get spoilt. I request my professional brethren to try this method of drenching and communicate their experience through this valuable journal.

CANINE GONORRHOEA.

BY

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Gonorrhoea in animals is so very rare that one is led to doubt whether such a complaint is at all present in animals. The course of the disease in this case was so very much like human Gonorrhoea that I have made bold to nomenclature the complaint as 'Canine Gonorrhoea'.

Subject.—A four-year old Irish Terrier belonging to a lady.

History.—The dog had been run over by a motor car at Calcutta about July 1921 but had fortunately received only slight injuries. A pus-like discharge from its sheath was at that time observed by the owner and this complaint, I was told, however disappeared soon after even without treatment. About the latter part of July 1922 a similar discharge was again noticed and I was called in for treatment.

Idiosyncrasy.—The terrier would not easily take any liquid medicine. Powders and solid drugs would be taken if concealed in meat.

Symptoms.—A slight temperature and an impaired appetite. A thick whitish yellow discharge from the sheath. The animal would sit down on its haunches after running a few yards. On exposing the penis a small ulcer at about its tip was noticed. The dog was otherwise healthy and in good spirits.

Treatment.—A dose of worm powder in meat followed by a dose of oil. The ulcer was cleaned with Pot. Permanganas lotion and dressed with Boro-Iodoform powder. No worms were passed but the animal ceased to sit on its haunches from the next day onwards. In about five days the ulcer had completely healed but the discharge was persistent. Smears from the discharge was taken and submitted to the Madras Veterinary College for microscopical examination. Pus cells were said to be present and I was directed to submit more smears. More smears were accordingly sent and this time pus cells with various cocci were said to be present. A sterile tube was sent to me from the college for the submission of a quantity of the discharge apparently with an idea of cultivating and preparing

an auto-vaccine out of it. The discharge had become so little by this time that I could not manage to submit a sufficient quantity of it to the Principal, Madras Veterinary College.

At this stage a suspicion crossed my mind that the patient might be ailing from Gonorrhoea. Mr. K. Raghavendra Rao, Veterinary Inspector, Vizagapatam Range had a look at this case and was of opinion that the complaint was Urethritis, but no amount of syringing the urethra with anti-septic lotions would stop the discharge. Mr. D'Costa the then acting Deputy Superintendent. C.V.D. Vizagapatam also examined the terrier and was of opinion that it was a case of Balonitis. He told me that no cure has yet been discovered for such a complaint but advised me to continue the H2 O2 wash.

Finding that such a course of treatment was doing no good I decided to treat the animal with Gonorrhoea specifics. Injection of Neo-Salversan or other similar drugs was not practicable as I had no proper Hypodermic syringe with me. Administration of Mixtura Copaiba or Santol oil was next to impossible as the dog would not easily take liquid medicines. Powders or solid drugs had therefore to be given concealed in meat. I happened at this time to notice 'Santal Perles' advertised by Messrs. N. Powell and Company, Bombay. Santal Perles were sent for and administered. I am glad to record that in a month after the date of commencement of the administration of Santal Perles the discharge was noticed to have completely stopped.

The Terrier is now hale and healthy.

ESSAY ON INFLAMMATION.

BY

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1. *Definition.*—

Inflammation may be defined as a succession of changes that occur in a living tissue when injured provided that the injury itself does not at once destroy its structure or vitality. It is the reaction of tissue to an irritant or an injury. It is a name applied to a group of pathological processes including circulatory disturbances and progressive tissue changes. The

term inflammation is difficult to define because of the factor entering into process and of a variation of each factor. It may be defined as the reaction of a living animal tissue to an irritant.

2. *General consideration of stimulus and irritant.*—

Two things are quite essential in an inflammation—one is a stimulus and the other is an irritant. A stimulus is a thing that produces action in a living tissue, while an irritant is that which produces excessive stimulation to a tissue. Thus the two terms differ only in a degree. Mild friction of the skin is a stimulus to the heart. When the friction is great and the cutaneous function is over stimulated, the friction becomes an irritant. Thus all the living tissues respond to stimulus and irritants. A stimulus is that which excites or produces a temporary increased vital action or it is a substance capable of producing activity in a living tissue. The amount of response to a stimulus is directly proportionate to the organisation and complexity of the tissue and especially those tissues which are (1) capable of being stimulated (2) capable of trans-mitting an impulse (3) capable of interpretting impressions produced by an impulse.

3. *Causes of inflammation.*

The causes may be divided under two main headings. (1) Exciting (2) Predisposing. The exciting causes of inflammation may be within the body or without the body. They produce their action by direct contact upon the surface of the body as from a blistering agent externally applied or by contact internally as from Arsenic. They may produce their effect while being excreted as in the production of nephritis by cantharidis and turpentine. Some harmless agents may become irritantsas a result of chemical changes produced by some juices secreted within the body. The predisposing causes.—

When the inflammations are not of themselves essentially irritant but often co-operate with others in inducing disease or in other words causes which render a part liable or exposed to become diseased such as debility or old age, debility of organs, previous disease or natural weakness, hereditary taint or predisposition, obstruction of blood vessels, fatty degeneration of system, climatic influence, change of diet, ill ventilation and the ill-treatment of all kinds. The causes of inflammation may

again be divided into two general classes (1) Non-infectious (2) Infectious. (1) Surgical wounds which by primary union are the examples of mechanically produced inflammation. The reaction taking place in an aseptic incision consists in the destruction of cells and slight circulatory disturbance that is immigration of lucocytes and regeneration of tissue. This is a reaction of a typical inflammation and the area is not infected. It is common that electricity causes inflammation. Animals show evidence of cutaneous inflammation though no actual wound could be seen on the body. In big cities like Madras, Bangalore and other places both men and animals come in contact with electric wires and receive local injuries which are inflammatory in their nature. There are some chemical drugs which are injurious and irritants. Whereever an irritant property is required these chemical substances such as mineral acids, caustics, mercury, salts arsenic etc., are used. These produce inflammation when applied in mild forms and necrosis of the part when applied in very concentrated form. Many insects such as reptiles, bees, wasps and some variety of ants introduce a chemical substance into the body when stung. These are very injurious and cause inflammation rapidly.

(2) Infectious form of inflammation.—

These are the important factors to produce inflammation because they are the worst offenders. Infection generally produces inflammatory disturbances through the action of chemical substances by the infecting micro-organism as metabolic productions. The infection may be local and produce local inflammation as in a superficial abscess. The chemical substances may be absorbed from the local infection and set up inflammation elsewhere in the body. Again infection may be general and produce similar inflammation practically in all the tissues as in generalised Anthrax. However the term inflammation is confined usually to local disturbances, the extent of irritation produced by any infecting organism and the resistance of the infected animal. Thus infection with Streptococcus Pyogenes may produce Pyaemia in one animal and local abscesses in another. Again some bacteria as the Anthrax bacillus may produce Septicaemia in one animal and local inflammation in another. The general infective inflammation may be discussed as 1. Nonsuppurative 2. Sup-purative

The non suppurative inflammations are through inflammatory disturbances in which there is no purulent fluid or pus produced. Examples, Septic infection succeeding nail pricks in horses feet, malignant oedema caused by the bacillus malignant oedema.

Suppurative infective inflammation is characterised by the formation of pus. The agents of suppuration are known as pyogenic Bacteria or pyobacteria. The bacteria that produce suppuration are Streptococcus Pyogenes and Staphylococcus Pyogenes.

4. *The Phenomena of inflammation.*—

The animal body is composed of different tissues in various combinations. The phenomena of inflammation are the changes that take place in the tissues plus the conditions resulting from these tissue changes thus including all the changes taking place in the inflammatory process. Vascular changes occur in the inflammatory process. The supplying arteries and arterioles decrease in their calibre. Temporary contraction of vessels is the first result of the application of an irritant. The cause of the constriction of arteries is a spasmodic contraction which is of Vaso-motor origin. This is succeeded by a marked arterial dilatation. The response to stimuli on the arteries is rapid and always active the veins are slow and usually passive and the capillaries are either rapid or slow but always passive. Dilatation of vessels in an inflamed area is also of vaso-motor origin. An increase in the calibre of the arteries results in the increased amount of blood flowing through them into the capillaries. The increased amount of blood in the capillaries mechanically increases their calibre and also increases the amount of blood which enters the related veins and results in the dilatation of the veins also. Consequent on the increase of lumen of the vessels there will be acceleration of the rate of blood flow. The corpuscles place themselves in the centre of the blood stream. The arterial dilatation plus the acceleration of the blood flow constitute the essential factors in active hyperemia. A long continued dilatation of a vessel results in an injury to the endothelial lining of the vessel. The injured cells become inflamed, rough and sticky. The leucocytes begin to appear in the peripheral portion of the blood stream.

They roll and creep along over the injured endothelial cells and finally adhere to the rough surfaces. The continued attachment of the leucocytes to the endothelium diminishes the calibre of the vessel and increases the resistance and thus resisting the rate of blood flow in the vessels. The resistance may become greater than the propelling force and the circulation may cease for a varying period of time. This condition is known as *Stasis*. Normally there will be fluid escaping through the vessels and an increased quantity of fluid escapes through during the period of inflammation. The portion of fluid that escapes through the blood vessels is called the *Exudate*. The exudation is the result of the vascular disturbances. The inflammatory exudate consists of varying quantities of cells suspended in a fluid for example plasma tissue juice etc. The fluid part of the exudate contains proteids in excess of normal plasma the specific gravity being about 1018 and sometimes more. The exudate is acid in reaction. The leucocytes are the principal cellular elements found in the exudate. The red blood corpuscles occur only in certain inflammatory conditions, such as crupous pnemonia. Cells other than leucocytes are seen in the inflammatory area, endothelial cells wandering connective tissue cells, giant cells and red blood corpuscles may be present sometimes.

The inflammatory exudate may be Serous, Fibrinous or Haemorrhagic.

Serous exudate is composed of almost entirely of fluid having very few cells. Inflammatory fibrous exudate contains two enzymes one of which is active being of alkaline medium and the other of acid medium. A fibrinous exudate is one that coagulates within the tissue or tissue space. This coagulation of exudate is identical with the coagulation of blood and probably due to fibrin forming—enzymes from disintigrated leucocytes. The exudate contains many cells and a large amount of proteids. Haemorrhagic exudate are those in which the red blood corpuscles as leucocytes and plasma have passed through the vessel wall. Intense irritants are causes for this kind of exudate.

Generally speaking a mild irritant or an injury produces a serous inflammation and an intense irritant produces fibrinous inflammation. Mechanical injuries where there are no surface

abrasions produce an inflammation of a mild degree and the exudate in quantity is usually of a serous nature. An irritant chemical substance injected into a tissue produces an inflammation characterised by a serous or a fibrinous exudate. The latter may be observed in the application of blistering agents. Infective inflammation is accompanied by a marked exudation from the beginning of infection. The quantity and quality of exudate varies with the virulence of the organism. But there are some exceptions to this rule e.g. Tetanus causes limited exudate regardless of the virulency of the tetanus bacillus. In some infections such as in malignant oedema the exudate is largely fluid. The exudate is almost entirely leucocytic. The exudate is usually limited in animals having normal vessels, heart action and blood. Animals in which the vessels are diseased and especially if the endothelium has been injured there is a tendency to excessive exudation. A weak heart is conducive to excessive exudation—example, Inflammatory oedema. Animals possessing dilute blood are predisposed to excessive fluid exudation. Exudation is the direct proportion to the vascularity and density of the tissue. Inflammation in compact bony tissue or beneath dense fascia ligament or tendons is accompanied by a limited exudate. Inflammation of the cutaneous structures is associated with excessive exudation which accumulates in the sub-cutaneous areolar tissue. Inflammation of serous and mucous membranes is accompanied with exudation which may be discharged upon the surface but is already accumulated in the substructures. Inflammatory exudate is usually beneficial until antibodies and bacteriological substances become neutralised and the phagocytic cells impaired or destroyed after which the exudate is usually injurious as it is a mass of foreign dead nitrogenous substance that serves as food for various bacteria.

Phagocytosis :—This is the incorporation and destruction of pathogenic bacteria and other foreign substances by leucocytes. These are cells having the power of ingesting and destroying micro-organisms, and other foreign particles. Polymorpho neucleor leucocytes are the most active cells concerned in Phagocytosis. The phagocytic property of cells is variable depending on the virulence of the micro organism or the strength of the chemical substances and upon the resistance of the phagocytes. The bacterial destruction may be instantaneous

or the bacteria may gain the upper hand and destroy the phagocytes. The included bacteria destroy the leucocytes and thus being liberated establish a new centre of infection. Thus phagocytosis is a very important factor in inflammation. There is no doubt that many localised inflammatory conditions are absorbed and the intensity of the attack of the other infective inflammatory conditions reduced by the process of phagocytosis. Some tissue cells under some conditions become phagocytic towards leucocytes. This is perhaps for the purpose of obtaining nutrition for the fixed tissue cells of the body.

(5) *Signs of Inflammation* :—

The inflammation may be seen either by the local signs or by general symptoms.

The local signs of inflammation are (1) Redness (2) Swelling (3) Heat (4) Pain and lastly, impaired function of the part. The Latin names of the above signs are Rubor for Redness : Tumor for swelling : Calor for heat and Dolor for pain. These signs are usually seen in the early stages of acute inflammation but they may be seen throughout the entire process. Mild chronic inflammation may not be accompanied by any of the above signs.

Redness (Rubor) is a constant sign in the early stages of acute Inflammation. It is the result of an excessive amount of blood in the vessels of the affected area.

Swelling is a characteristic of acute inflammation. It is the result of the accumulation and retention of the inflammatory exudate plus the increased amount of blood in the part. The extent of the swelling is the direct relation to the density of the tissue.

The heat or temperature (calor). It is of the tissue affected with active inflammation invariably increased. This is the result of the excessive cellular action in the inflamed area and the increased amount of blood flowing to the part.

Pain :—(Dolor) is a common symptom of inflammation. This may be the result of pressure upon nerve endings by the accumulated exudate. The inflammatory pain is the result of injurious action of the chemical irritants upon the sensory nerve endings.

Impaired function. Is a constant factor seen in an inflammation. In the beginning of the process the function of the affected tissues is in excess of the normal but it is succeeded in the latter stages by the depression of the function. The increase is due to a result of increased nourishment and increased stimulation. The diminished function is the result of the injurious action of Katabolic products produced and of the irritant producing the inflammatory process.

The General symptoms :—The general symptoms are other wise known as constitutional symptoms. The constitutional symptoms of inflammation are those indicative of sympathetic, inflammatory fever and are of great importance because they indicate the nature of the disease. When there is excessive inflammation there is increase of temperature. The symptoms of inflammatory fever are shivering, internal temperature, debility, increased heat of the skin, accelerated full and hard pulse, staring coat, dull and depressed condition and the animal will have no proper appetite. There will be pain all over the body stiffness of the joints and limbs. The animal will be restless. There will be partial sweats all over the body. Sometimes there may be even convulsions and fits. There will be general diminition in excretion and secretion. Urine is scanty and high coloured. In cows there will be diminished milk supply. The digestive and assimilative power is interfered with followed by emaciation. Loss of tone of the system. The animal will have loss of strength and condition.

Effects upon the tissue.

As a result of the inflammatory process the tissues affected may undergo various changes such as degeneration necrosis and regeneration.

Degeneration and regeneration are two distinctly opposite processes. The former destroys the tissues resulting in the impairment and death while the latter is constructive resulting the overgrowth. Necrosis or local death:—All degenerations produce impairment of function and frequently end in necrosis of the affected cells. Destruction of cells is a common result of inflammation because of the various degenerations followed by the inflammatory process. Suppuration is a type of inflammation and is a liquefied form of necrosis. Superficial necrotic tissue is

known as auto ulceration and is the result of a continuous and sometimes progressive cellular necrosis.

Regeneration:—This process usually begins after degeneration ceases. Cells concerned in this process undergo a change becoming similar to embryonic cells. Reproduction is the active property of these cells and this is also the chief function of generative cells. Whether degeneration of cells are capable of regeneration depends upon the kind of cells and the extent of the injury to them. The age of the individual is an important factor in the regeneration of injured tissue. Tissues in young animals regenerate more readily than like tissues in old animals. The origin of cells that regenerate connective tissue is still a disputed point. It is said that fixed and wandering connective cells are capable of producing connective tissue. If examined under microscope the generative connective tissues are either oval or spindle shaped of which the latter predominate especially in active process. Mononuclear leucocytes as well as lymphocytes may be capable of producing connective tissue. Endothelial cells are active in the production of new vessels. The inflammatory tissue is more vascular than the usual tissue. In the vascularisation of an inflammatory area the endothelial cells produce long protoplasmic projections. Several of these projections of different adjacent cells may fuse thus forming an anastomoid channel or a single projection may separate in a longitudinal direction thus producing an extension of the old channel. As the protoplasmic projections become larger and larger the cell division soon follows. This process continues as long as the inflammatory process is active. The irregular blood capillaries so formed become surrounded by a layer of involuntary muscle tissues and yellow elastic tissue as a result of extension of muscle fibres and connective tissue cells from the adjacent vessel, the whole structure being surrounded by a loosely arranged layer of white fibrous connective tissue. Thus the capillary becomes an arteriole. The cells that are active in vascularisation are known as angioblasts. Inflammatory injury to surface epethelium as epidermis or mucous membrane are usually repaired by the multiplication of the cells bordering the injury.

Chronic inflammation is characterised by a relatively mild hyperaemia and by an increased amount of fibrous tissue. The

newly formed fibrous tissue may or may not be displaced by normal tissue.

7. Types or Kinds of Inflammation :—

Inflammation may be classified as (1) Simple and (2) infective. The simple inflammation is noninfective and results from mechanical or chemical interference. Fractures, sprains bruises and surgically produced wounds are types of mechanical inflammation. Local inflammatory disturbances resulting from lightning and contact with electricity are types of electrically established form of inflammation. The chemical inflammation is caused by Arsenic, turpentine and those produced by poisonous insects such as reptiles, scorpions, bees and some variety of ants and also those caused by the power of bacteria and animal parasites. The infective inflammation is of more frequent occurrence than of noninfection. It is a kind of inflammation that concerns the practioner, a Veterinary Surgeon and a sanitarian because of its tendency to become generalised in the infected animal. All tissues are susceptible to infection with the exception of hair, wool feathers and the insensitive and non-vascular portions of the teeth, hoofs, claws and bones. Again infective inflammation may be suppurative and non-suppurative. Non suppurative infective inflammation is found in malignant oedema, blackquarter, localised anthrax and in the early stages of tuberculosis and actinomycosis and is characterised by the general phenomena of inflammation. Infective inflammation may be suppurative in the early stages but later on be complicated and typical suppuration as in tuberculosis. More rarely nonsuppurative inflammation contains throughout the entire process as in black quarter.

Suppuration or suppurative inflammation :—

Suppuration is an inflammation characterised by liquefying necrosis and may be surface or sub surface circumscribed or even diffused. The necrotic tissue produced by suppuration is known as pus. Pus is a fluid varying from a thin watery substance to thick sticky tenacious mass and is usually alkaline in reaction. The colour of the pus is determined by the infective agent and it may be white, lemon yellow, golden yellow, greenish yellow frequently tinted with blood. Pus obtained from solipeds is usually white or greyishwhite and from cattle

creamy yellow and from sheep greenish yellow. Healthy pus is usually odourless although it may undergo putrefaction with the evolution of foul smelling gases. Pus may be greasy smooth, sticky or granular to feel when rubbed between two fingers depending on its composition. Histologically pus is composed of puscells that is leucocytes most of them are dead, shreds of muscle tissue. Pus produced in surface wounds constitutes purulent discharge. Inflammation of mucous membrane accompanied by a purulent discharge is known as purulent catarrh, Sub surface suppuration may be circumscribed and diffused. Suppurative centres become circumscribed firstly by a dense wall of leucocytes and later by fibrous capsule. The capsule in almost all cases is denser on the side next to the more important tissue. The collection of pus in tissues and in body spaces lymph spaces constitutes one abscess. Diffused suppuration is not limited by a definite border line. This is the result of agencies possessing sufficient strength or virulence to continuously and progressively destroy and liquefy the tissues. Inflammation would be classified according to the time basis as acute and chronic. Formerly this classification was based upon the time but the duration of inflammation is so variable that it is now recognised as an insignificant factor. Acute inflammation is charaterised by a sudden onset, by a vigorous action and its production of retrogressive changes in or destruction of the tissues affected. Chronic inflammation is usually by insiduous mild action and by resulting in the repair of tissues. The tissue may induce retrogressive changes, atrophy but it is only an indirect result of the process. Either acute or chronic inflammation may occur throughout the entire reaction and they both prevail at the same time in different parts of the same structure. The causative agents may become less active as the process continues thus acute inflammation is often succeeded by chronic form. Chronic inflammation may be succeeded by acute inflammation providing that the irritating factor be sufficiently increased or the resistance of the animal diminished. Catarrhal inflammation is the inflammation of a mucous membrane accompanied by excessive production of discharge of mucous. The purulent inflammation is characterised by the production of Pus. This term is confined by some to surface suppuration. Ulcerative inflammation is one in which there is the production of ulcers and pustular inflammation is characterised by the formation of

pustules in an inflamed area. Chronic inflammation is practically the production of new tissues. The specific inflammation is one resulting from a specific infection such as in glanders.

8. Termination of inflammation.

The termination depends upon the extent intensity and duration of the irritant and the resistance of the tissues. The tendency of reaction produced by an injury is always favourable but the reaction may be so sudden and extensive or continued. So long that its results may be harmful inflammation may terminate in resolution of tissue formation or destruction. Resolution embraces the process of repair and this may be summarised as follows :—

(1) Removal of the cause. (2) Re-establishment of circulation. This may be accompanied in a few hours or perhaps not for several days depending upon the extent of the injury and the kind of tissue injured. (3) restoration of vessels to their normal condition. The length of time required for restoration and the completeness of process depends upon the severity and the re establishment of the circulation (4) Removal of the inflammatory exudate. The time required to remove the exudate depends upon its nature. Serous exudates are usually removed by the lymph channels. Fibrous and Hammorrhagic exudates are usually discharged and absorbed or may be carried away by phagocytes. Exudates in part may be concerned as nutritive by local cells. (5) Disposal of necrotic tissue. Necrotic tissue is disposed of by sloughing, absorption, phagocytosis. Small area of necrotic tissue are usually absorbed or disposed by phagocytic action. Considerable time is usually required in disposing of large areas of necrotic tissue unless it is superficially located and separates from the surrounding tissue and sloughs. Sub surface necrotic tissue may be gradually liquefied and absorbed, discharged through a fistulous tract, collected and carried out by phagocytes and remain permanently in the tissue. The necrotic tissue found in a capsule may be filled in with calcium salts.

6. Regeneration of degenerated tissue and replacement of necrotic tissue. It consists in replacing the injured or destroyed cell protoplasm by normal protoplasm. If only a few cells are destroyed the adjacent cells reproduce and this renewal is usually required. Large areas of necrotic tissue are substituted by fibrous tissue. This tissue is called granulation tissue in the beginning

and cicatricial tissue after it has become dense and more contracted. Granulation tissue consists of capillary loops surrounded by a mass of cells. These cells are largely fibroblasts and produce fibro connective tissue. After the fibro-connective tissue has been formed it contracts thus becoming cicatricial tissue. This is important in closing gaping wounds but is dangerous when it occurs in internal organs as in liver because the pressure produces atrophy of the gland and obstructs circulation.

The tissue formation in inflammatory resolution takes the place of tissues that preexisted and had become necrotic while that occuring in inflammation resulting from long continued mild irritation is not a substitution but an addition to the tissue already existing. In the latter case the tissue formation may begin in a very short time or it may not appear for several days. Fibro connective tissue is invariably the product of tissue formation. Strictures of hollow organs are produced by fibrous tissue. Adhesions of serous memberane are produced by serous tissue formed during inflammation. The destruction is a result of intense irritation of the part. Necrosis of tissue is frequently a sign of inflammation. A single cell or only a few cells may be destroyed or one area of tissue may undergo necrosis. Ulceration is the result of constant cellular necrosis. Circulation may be interfered with by an inflammatory exudate and cause necrosis in larger masses of tissue. It may terminate fatally in partial recovery or in resolution depending upon the importance of the tissue involved in the affected animal.

9. *Treatment* :—It is the duty of the Veterinary Surgeon to prevent inflammation of the part by every means of his power. The first step in the treatment of any disease whether surgical or medical is to remove the cause. All further treatment is of minor importance when compared to this. William in his " Principles and practice of Surgery " says " remove the cause and the effect will cease " when the cause of inflammation is removed the remedial measures may be followed. Those which are opposed to the advance and persistance of inflammation are known as Antiphlogistics one of which is bleeding. In Sthenic cases general bleeding may be resorted to. Phlebotomy as in laminitis or cerebral congestion acts by depressing the heart and also as nerve sedative and helps to absorb the exudate. But its effects are temporary and the bleeding should be less and the amount varied in different species of animals in particular cases. In horses 8 to 10 pints and ox 10 to 15 pints can be drawn.

Here the guide is the pulse. But bleeding is contraindicated in all asthenic and in very old animals, pregnant and fatty animals. Phlebotomy is done on either side of the neck by means of trocar and cannula or lancet. Some authorities are of opinion that bleeding is not a sound method. So it is not generally followed now in many cases. The danger is that the animals maysuffer from phlebitis.

The treatment can be divided into local and constitutional.

Local treatment.

After removing the cause of inflammation in wounds and septic cases antiseptics are necessary. For aseptic cases such as sprained tendons etc. complete rest of the part is essential. Stimulating liniments, and astringents are quite necessary things. We generally get good results by changing the dressings. In obstinate cases blisters and counterirritants and actual cautery are recommended. But these are contra indicated in acute cases of inflammation. All these do not help without complete rest to the animal. Generally rubefacients are the best for they are nondepressants in their character. They indicate in large surfaces of inflammation such as abdomen thorax and throat. The most common rubeficients are mustard, peppermint, oil of terpentine. Vesicants are also good for localised inflammation. It is commonly used in sprained tendons and ligaments and in diseases of bones and joints. This may be followed after a right indication by a second or a third application. The next item of local treatment is by irrigating the part inflamed. Cold water is the best for irrigation purposes. If there is pain and swelling hot fomentation may be resorted to. Hot water fomentation has got a soothing affect. After the swelling has subsided cold irrigation may be followed. In this treatment some are for cold irrigation first followed by hot fomentation. And there are some authorities that say that hot fomentation is the best as it acts as an anodyne and antiphlogistic. But we can say that hot fomentation should be followed by cold irrigation as the former gives relief to the patient sooner than the latter.

Constitutional treatment.

Internally give laxatives and febrifuges and keep the animal on laxative diet. The next important class of antiphlogistic agents in the treatment of many inflammation consists of purgatives more especially aloes in horses, salines in cattle and castor oil in the dog.

They act by removing from and freeing the intestinal canal of accumulated faeces and other irritating matters. To subdue pain and soothe the system opium can be administered. Its use is of importance in some inflammations such as bronchitis, enteritis and cystitis. Sod. Bicarb, pot. bicarb are useful in several cases.

Treatment of chronic inflammation.

All depressing remedies should be avoided as far as possible. General treatment is not necessary. Nutritious food, fresh air and proper exercise are essential. Tonics in the form of nuxvomica, quinine and iron can be given. The animal should not be exposed to debilitating influences, such as hard work, cold etc., too soon after recovery ; it may lead on to the formation of secondary abscesses in the internal organs andprove fatal in the end.

10. Conclusion:—

Inflammation is the reaction of a living tissue to an irritant. It is a complex process the result of many factors. It is not always a result of infection. It is an adoptive, repairative and protective process. It may produce sufficient reaction to cause destruction of the portion involved or occasionally of the entire organ.

[redacted]

SOME HELMINTHOLOGICAL PROBLEMS OF THE PACIFIC REGIONS.

BY

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(Walter and Eliza Hall Veterinary Research Fellow. University of Sydney).

HABRONEMIASIS OF HORSES.*

The term Habronemiasis is applied to a nematode infestation of the horse produced by one or more of three species of the genus Habronema, the adult worms always being found in the stomach.

The genus Habronema belongs to the super-family Spiruroidea Railliet, 1915, a super-family in which development in perhaps all cases requires an intermediate host.

* Read before the Veterinary Section of the Pan Pacific Science Congress, Sydney August 24th 1923.

Three species of Habronema occur in the horse:—*Habronema megastoma* (Rudolphi, 1819), *Habronema microstoma* (Schneider, 1866), and *Habromena muscæ* (Carter, 1861).

Both morphologically and because of differences in development and its behaviour in the primary host, *Habronema megastoma* is marked off from the other two species. *Habronema megastoma* is the smallest of the three species, males being from 7-10 mm. long, while the females are 10-13 mm. Spicules in the male are 240 and 400_u long. Eggs $45\text{-}55_u$ long and $11{\cdot}6\text{-}14_u$ wide. Width of embryo, $6{\cdot}6_u$. Eggs contain motile embryos when leaving the uterus of female. Adults are only found in tumours of the stomach wall and not free on the gastric mucous membrane.

Habronema muscæ and *Habronema microstoma* both morphologically and in their behaviour in the final host present a close resemblance to each other. *Habronema microstoma* was described by Schneider in 1866 as *Spiroptera microstoma*, but it was not till 1911 that Ransom differentiated the two species, and described the adult *Habronema muscæ* for the first time. The larvæ of *Habronema muscæ* were found as parasites of the house fly in Bombay by Carter in 1861. The two species may be distinguished by the following characters. *H. microstoma* is slightly the larger, measuring, females 15-25 mm. by $300\text{-}500_u$, males 9-16 mm. by $250\text{-}300_u$; *H. muscæ* being, females 13-22 mm. by $250\text{-}400_u$, males 8-14 mm. by $250\text{-}300_u$. The pharynx in *H. microstoma* is relatively larger and wider, and shows a tri-dentate process springing from both dorsal and ventral wall, lacking in *H. muscæ*. In the males, in addition to a slight difference in arrangement of the pre- and post-anal papillæ, there is marked difference in the size of the spicules. Thus in *H. microstoma* they are 800_u and 350_u, while in *H. muscæ* the left spicule is extremely long and slender, being 2·5 mm. long and only 5_u thick in its middle, while the right spicule is 500^u long.

The females also may be distinguished by the fact that in *H. microstoma* the vulva is large and prominent, while the distal part of the vagina forms an S-shaped curve, which is surrounded by a globular mass of muscular tissue. In *H. muscæ* the vulva is small, the vagina does not form this S-shaped curve and the muscular mass is lacking. The eggs of both in their earliest stages are from $40\text{-}50^u$ long by 10_u wide. In the uterus those of *H. muscæ* grow from $80\text{-}90^u$ long by $6\text{-}7_u$ wide, while those of *H. microstoma* are 110_u long

by 6_{μ} wide. Both contain very active motile embryos at the time of oviposition. The eggs and embryos of all species of *Habronema* are thus very small, and this in conjunction with their transparency makes their detection in fæces very difficult.

Life History of Habronema. In 1861 Carter discovered a larval nematode in house flies in Bombay, and named it *Habronema muscæ*. Nothing was known of its relationship to any adult worm till Ransom (1) in 1911 proved that it was the larval stage of an adult worm occurring in the stomach of the horse. He found that stages in the life cycle were passed in the pupa and adult of *musca domestica* till a stage was reached in which the larva could develop to the adult worm when taken into the stomach of the horse.

Further researches were carried out by Hill (2) and Bull (3) in Victoria and South Australia respectively, which confirmed Ransom's work, and at the same time proved that *Habronema megastoma* and *H. microstoma* also required to pass several stages of their life cycle in flies.

The main features in this life cycle are as follows. Motile embryos, still covered with a shell membrane, pass out of horse in the fæces. They then make their way into fly larvæ, but whether actively or passively by ingestion is not known. Larvæ may remain alive in fæces for eight to fourteen days, while the fly larvæ may become infected from forty-eight hours up to nine days after hatching. The next stages are met with in the fly pupæ, and further development takes place after the adult fly emerges. The final stage larvæ are commonly met with in the head, and when ready to leave the fly, in the proboscis. While *H. muscæ* and *H. megastoma* are commonly found to develop in *Musca domestica*, *H. microstoma* is found in *Stomoxys calcitrans*, and only very rarely and probably accidentally in *M. domestica*. Harvey Johnston (4) has found that *H. muscæ* and *H. megastoma* are also found in *Muscahilli*, *M. terræ-reginæ*, *M. fergusoni* and *M. vetustissima*, and experimentally in *Sarcophaga misera* and *Anastellorhina augur*. It is unlikely, however, that the latter two species would act as hosts naturally.

Roubaud and Descazean (5) have recently stated that while *H. muscæ* and *H. megastoma* pass the early stages in the intermediate hosts in the cells of the adipose tissue, *H. megastoma* is found in the Malpighian tubules, in which it causes tumour formation. Thus both

have an exclusive environment for development; and *H. megastoma* seems to fore-shadow its behaviour in its final host, where it also gives rise to tumour.

Method of Infection of Final Host. Three possible methods of infection of the final host (the horse) are indicated. (1) By ingestion of flies infested with final stage larvæ; (2) by the escape of larvæ from the proboscis of the fly; (3) by inoculation of larvæ under the skin. While ingestion of flies may serve as one source of infection, this does not suggest itself as a very convenient method for the propagation of the species, and it would appear likely that, were this the only method of infection, Habronema would not be as common as it is in this country. In support of this theory, however, is the fact that Habronema larvæ may survive in dead flies for two days. That direct infection from the fly to the horse is usual is supported by the fact that larvæ removed from flies only remain alive for a limited time, so that there remains the second method of direct infection by the escape of larvæ from the proboscis. While Ransom and Hill did not find evidence to support this, subsequent writers, including Harvey Johnston and Roubaud and Descazeau (11), find that this occurs spontaneously under certain conditions. These conditions are that the fly must be feeding on surfaces having a suitable degree of warmth and moisture, such as are met with on mucous membranes and moist wound surfaces. Only those larvæ which pass thence to the stomach (that is, those that are liberated on the buccal mucosa) develop into adults. Those which escape on to the conjunctiva or other mucous membranes or on to wound surfaces die, although they may first bring about serious pathological changes. Larvæ may also escape from flies when feeding on animals other than the horse, and in man and experimental animals, while death soon occurs, they may cause passing irritation, conjunctivitis, etc. There is little doubt that the above is the normal method of infection. Since, however, *Habronema microstoma* is transmitted by *Stomoxys calcitrans*, which normally does *not* feed on mucous membranes, there would at first appear to be some difficulty in providing for the propagation of this species, unless they were infected under the skin while the fly was sucking blood. It has been found, however that the presence of the larvæ in the proboscis renders the Stomoxys unable to pierce the skin and suck blood, so that it is reduced to obtaining nourishment by absorbing fluids from wound surfaces and mucous membranes, thus ensuring the completion of the life cycle as in the other two species.

Lesions produced by adult Habronemas. In the past the attention given to the pathological importance of Habronema was slight, but a more careful consideration of the question would lead us to suppose that the lesions occasioned by them may at times be serious.

The lesions caused by *Habronema megastoma* differ from those caused by the other two species in that it is never naturally found fre on the mucous membrane, but is always situated in tumours in the sub-mucosa. Larvæ, when reaching the stomach, make their way into the openings of the gastric glands, and the irritation they cause leads to the production of inflammatory fibrous tissue resulting in the formation of tumours of varying size. Running through these are canals which open into the lumen of the stomach, each tumour always having one or more orifices opening from it. Pressure on such a tumour forces out numerous adult worms, while the pus in which they are generally bathed contains large numbers of motile embryos. While these tumours are not generally considered to produce any serious results, there must always be a certain destruction of the glandular epithelium. In advanced cases, however the inflammatory changes may involve the muscularis, while at the same time abscess cavities may be formed. The myositis so occasioned, where a considerable area of the stomach wall is involved, may produce serious interference with muscular contraction, and thus produce interference with normal digestive processes. Cases are also recorded where such abscesses have ruptured into the peritoneum with subsequent fatal peritonitis, and there is always danger of this, owing to the weakened muscular wall, in cases of impaction of the stomach. Finally, the canals of tumours would be a very likely path of infection with pathogenic micro-organisms. In Victoria, splenic abscesses of habronemic origin have been reported, often with fatal results.

Habronema microstoma and *H. muscæ* are both found lying free on the mucous membrane of the pyloric half of the stomach. When present in large numbers they may form a felt-like mass covered with a thick mucous secretion, so closely associated with the mucosa that vigorous scraping is necessary to detach them. While there seldom appears to be any acute inflammatory processes set up, the mucous membrane is often found in a markedly catarrhal condition. While this gastric catarrh, if transient, might not be serious, the very fact of its chronicity is important, and may result in all the symptoms associated with this condition. The thick mucous layer often

covering the whole of the glandular epithelium must necessarily lead to a diminution of its secretion, and hence interference with normal digestion is brought about. Such changes may result in a liability to fermentation of food, producing chronic dilatation of the stomach wall, with consequent weakness and loss in contractility of the musculature.

For these reasons, then, we are unable to overlook Habronema as a predisposing cause of serious pathological processes.

It is true that where horses are well fed and not subject to serious exertion they may harbour numbers of Habronema without apparent harm. Where, however, they are subject to long and continued exertion under arduous circumstances, such as cavalry experience in war-time, the loss in efficiency might be serious.

Recently a veterinary officer in India (6) expressed the opinion that sixty per cent. of debility cases in Australian remounts were due to Habronema. While he did not adduce any scientific evidence in support of this statement, nevertheless, as the opinion of an army officer of experience, and of one who has paid some attention to helminthic disorders, it deserves consideration. The percentage given was for horses which were not found infested with strongylids.

Lesions caused by Larvæ. In 1859 Ercolani discovered a larval nematode in the subcutaneous fibrous tissue of skin lesions in the horse, such lesions being known as *summer sores.* Various other writers have described similar conditions. Railliet, in 1915, discusses the finding by Descazeau (10) of larvæ, belonging to the super-family Spiruroidea, in skin lesions in Brazil.

Bull (7), in 1916, described the finding of habronema larvæ in lesions of the external genitalia of horses in South Australia, while Lewis and Seddon (8) demonstrated similar larval remains in granulomatous growths involving the conjunctiva. Experimental evidence has supported the theory that these lesions are caused by the larvæ of Habronema spp., such larvæ being liberated on the sites affected while the fly is feeding there.

The lesions so found present some differences according to the site in which they occur, but all recent cases appear to show small areas of necrotic tissue, which on dissection and microscopic examination reveal the presence of larval remains in ovoid spaces in the mass. Where these lesions occur on the conjunctiva the

necrotic areas are found in a soft vascular mass of fibrous tissue. Those found on the penis are frequently of much firmer nature, though here also the surface is ulcerated and shows a vascular granulation tissue. A feature of many of these tumours is their sudden apperance and rapid growth for a short time, after which they may continue to increase in size slowly. The lesions definitely known to be caused by Habronema larvæ in the temperate parts of Australia appear to be more or less restricted to the glans penis, sheath and conjunctiva. In tropical Australia, especially the Northern Territory (9), there exists a condition commonly known as Swamp Cancer, occurring in horses often far from habitations, in which large granulomatous masses with ulcerating surfaces are met with on the limbs and body, and which are of an extremely chronic nature, while much larger areas are involved than in habronemic granuloma of South Australia. Bull, who has examined these growths, found typical necrotic areas, but was unable to demonstrate the presence of larval remains. He considered that there is a great possibility that they were due to Habronema, and explains his inability to demonstrate larval remains as being perhaps due to the fact that no recent lesions were examined. The fact that *M. hilli* and *furgusoni* are met with far afield would allow of horses becoming infected in the absence of *M. domestica*, and would account for the presence of Habronema lesions in horses which are not stabled or kept near houses. In the Solomon Islands also there exists a granulomatous affection of a chronic and apprently painless nature, which attacks the pasterns only. Such growths are of extremely firm fibrous tissue, up to a cricket ball in size. Bull found larvæ with longitudinal striations and spinous tipped tail in necrotic areas in these lesions. Throughout tropical Africa granulomatous sores with ulcerating surfaces are found on the forelimbs and inner canthus of the eye, in which larval remains are also found, while in South America similar lesions on the external surface of the extremities and neck have also been found to contain larvæ. Roubaud and Descazeau (11) have recently produced typical summer sores by experimental infection with Habronema larvæ. There is little doubt that the majority of these conditions are caused by larvæ of Habronema, but the question of the sites on the body of such lesions in different localities is interesting, probably depending either on the habit of the fly which is locally the carrier of the larvæ, or on some local conditions, as, for instance, vegetation, which produces wounds permitting of infection on the sites found.

It must not be thought that typical granulomatous lesions are produced in all cases where Habronema larvæ escape on to mucous or wound surfaces. Experimentally it has been found that in some cases there is merely slight tumefaction of the site, or where the conjunctiva is involved, a passing conjunctivitis. For this reason the seasonal conjunctivitis occurring in man in certain localities in summer months has been suggested as possibly due to this cause. Repeated stimulation and increase in size of an existing granuloma may be brought about by re-infection from time to time with fresh larvæ. Such a lesion with ulcerating surface being naturally very attractive to flies. It has been found that such sores frequently arise when there has been a previously existing abrasion or wound.

Incidence. The incidence of adult habronemiasis appears to be very high throughout Australia. In Victoria, 37 out of 39 horses examined by Hill were found to be infested with one or more species, while in South Australia only one or two stomachs were found free of parasites in over one hundred examined. In the Sydney University Veterinary Clinic, of eleven horses *post-mortemed* in the first half of the year nine were found infested.

In South Australia and Victoria *Habronema muscæ* was most frequently found, occurring in nearly 90 per cent. of cases, and while *H. microstoma* was also very common, *H. megastoma* was less frequently met with. Of the nine cases mentioned above three contained *Habronema microstoma*, four *Habronema muscæ*, and eight *Habronema megastoma*.

The number examined, however, is too small to estimate the relative frequence with which the three species occur here. It is probable that in tropical Australia and in other tropical countries Habronema is no less common, so that we must regard it as one of the most frequent nematode infestations of the horse in that country.

While it is hard to give an estimate of the frequence with which lesions caused by larvæ are found, it would appear that they occur more frequently in hot tropical countries than in more temperate zones, and depend, of course, to a considerable extent on the prevalence of flies during the hot months. In South Australia lesions are most frequently found on the prepuce and penis, while in Victoria lesions are frequently met with on the conjunctiva. In Sydney, it is perhaps worthy of mention that 53 per cent. of all tumours met

with in horses examined at the Veterinary Clinic from 1914-1921 were found involving the conjunctiva. While a number of these were true neoplasms, some were of habronemic origin. In parts of New South Wales granulomatous lesions in horses, popularly known as swamp cancers, are met with, but whether these are habronemic I cannot at present state. In the Solomon Islands the condition known as swamp cancer is said to be met with in 75 per cent. of horses, and there is reason to believe that this is also caused by habronemic larvæ, as in at least some cases larval remains have been found in the lesions.

It has also been suggested that the seasonal conjunctivitis known as "bung eye," and occurring during the summer months in hot regions in Australia, is due to Habronema (12), and it has been shown experimentally that larvæ deposited on the conjunctiva of man has produced conjunctivitis.

The percentage of flies infected varies in different localities, but up to 33 per cent. of flies in a Brisbane stable have been found, by Harvey Johnston. to be infested with Habronema spp.

Diagnosis of Habronemic Infestation. Diagnosis of the presence of adult Habronema is unfortunately difficult. As has been mentioned the eggs are very small, and in addition are very transparent, so that their detection in fæces is difficult. Probably by sieving and centrifuging fæces by Hall's method, one would be able in some cases to demonstrate the embryos. By the flotation method of Vadja, I have been unable to find embryos of Habronema, though this method is excellent for Strongylid and Ascaris eggs. As the stomach contents are often found on *post-mortem* to be swarming with Habronema embryos, it is quite possible that washing out the stomach with a stomach tube and collecting and sedimenting the ingesta might be efficacious. When one is operating on an empty stomach and the water is pumped in under pressure some adults might even be washed out. In the absence of other diagnostic features, or of evidence of other parasitic infestation, one would be justified in suspecting habronemiasis when there are symptoms of chronic gastric catarrh, capricious appetite, debilitated appearance and harsh coat, while the animal tires easily at work.

The diagnosis of Habronema in the lesions caused by larvæ can only be definitely made where one can demonstrate the larval remains

in the necrotic areas. Where, however, one has small yellow areas of necrosis occurring in a granulomatous mass on the mucous membrane, or where there has been a previously existing wound or abrasion, there is a strong probability of its being of habronemic origin, even where no larval remains are found. It is known that in old-standing lesions where there has been no re-infection all traces of larvæ may disappear.

Treatment. There is considerable difficulty in estimating the efficacy of medicinal treatment on the living animal. Worms that are killed in the stomach then become merely inert protein material, and are digested and disintegrated in their passage along the alimentary tract. Because of this fact, one cannot collect them in the fæces and so demonstrate the efficacy of treatment. Owing to the difficulty in determining the presence of their eggs in fæces, one is unable to compare the egg production before and after treatment as can be done with other helminth parasites, and by which one can gauge the completeness or otherwise of treatment adopted. Hall (15) has found that where worms are covered with mucous neither carbon tetrachloride nor ol. chenopodium is very efficacious, though probably killing the worms with which they come in contact. For this reason, too, we are unable to expect success against *Habronema megastoma* where they are buried in nodules. It is likely that repeated administration of anthelmintics in small doses would be most likely to be effective. As the slow poisoning produced by arsenic is so efficacious in the treatment of stomach worm in sheep, it is suggested that repeated administrations of arsenic might be advisable against Ha' ronema.

For the lesions occasioned by larvæ, surgical interference is indicated where the tumours caused are operable. Such tumours should always be treated antiseptically and steps taken to prevent re-infestation.

Prophylaxis. It is in measures of prophylaxis rather than in medicinal treatment that one must look for reduction in the incidence of this infection. As has been shown it is necessary for the carrying on of the life cycle that Habronema embryos must infect fly larvæ of certain species. It is towards the prevention of this infection taking place that efforts should be directed. Effective destruction of manure is, then, indicated—as by this means one not only destroys Habronema embryos, but at the same time gets rid of fly larvæ, and so keeps down

the number of adult flies. It must be admitted that thse measures are more practicable in the city and towns than in the open country. In settlements, infection with *Habronema muscæ* and *megastoma* are carried by the house fly, *Musca domestica*, which is seldom met far from habitations, and dislikes venturing out into the sunlight. Where house flies exist round stables there is a sure source of infestation provided, but there is no reason why their numbers should not be very greatly reduced by efficient hygienic measures.

All manure should be collected from stables and their immediate vicinity at least once a day, and should be suitably conserved, so that it cannot be contaminated by flies nor act as a breeding ground for larvæ. Roubaud's (13) biothermic method consists in placing fresh manure each day in the centre of the manure heap where sufficient heat is generated by fermentation to kill both fly larvæ and Habronema embryos. Placing manure in tanks with water will also effectively destroy larvæ. Placing manure in fly-proof boxes is also quite a good method of conservation.

While *Stomoxys calcitrans* is generally said to prefer grass cuttings to manure as a breeding ground, it is quite evident that it frequently deposits its eggs in manure and straw contaminated with urine and fæces, since it is only in these situations that larvæ can become infected with the embryos of *Habronema microstoma*. Therefore soiled bedding must also be conserved in such a way that it cannot act as a breeding ground. Fly traps in the vicinity of horse lines and stables will also do much towards keeping down the number of flies.

The above methods are all quite practicable and are very necessary in all stables in the city, whether they are large or small. They should also be employed in cavalry camps, especially where horse lines are fixed for any length of time.

In the country, where horses may become infected while running at grass, though far removed from stables and houses, prophylaxis becomes much more difficult, and is impracticable.

To prevent infection of wounds with larval habronema, where such infection is known to occur, they should either be protected by bandages or treated so as to render them unpleasing to the fly. Astringent dressings or ointments are indicated ; the one renders the

wound surfaces dry and hard, thus making it unsuitable for the escape of larvæ on to it, while oleaginous mixtures are not attractive to the fly.

(—*From The Veterinary Record, June 21, 1924.*)

DEPARTMENTAL NOTES AND NEWS.

1. A Veterinary Dispensary was opened at Tuticorin on 12th April 1924, and five touring billets were created in April last with head-quarters at Palaconda, Gudur, Tadpatri, Tindivanam and Ambasamudram.

2. Veterinary Assistant Surgeon A. Rajappa is granted an extension of leave for 5 months and 2 days making a total of 13 months and 2 days leave.

3. M. R. Ry. H. C. Sampathu Ayyangar Avergal, Officer in charge III Circle, Civil Veterinary Department, Bellary is on leave for 3 months from 26th April 1924 and Mr. W. J. D'Costa has been acting for him.

4. We regret to record the sudden death of Veterinary Assistant Surgeon A. Ramasubbu on 28th February 1924.

5. A temporary post of Deputy Superintendent has been sanctioned by Government for two years from 21st June 1924 as Mr. T. J. Hurley, Officer in charge, I Circle, Civil Veterinary Department, Vizagapatam has to be relieved on that date to act as Professor of Surgery at the Madras Veterinary College from 1st July 1924.

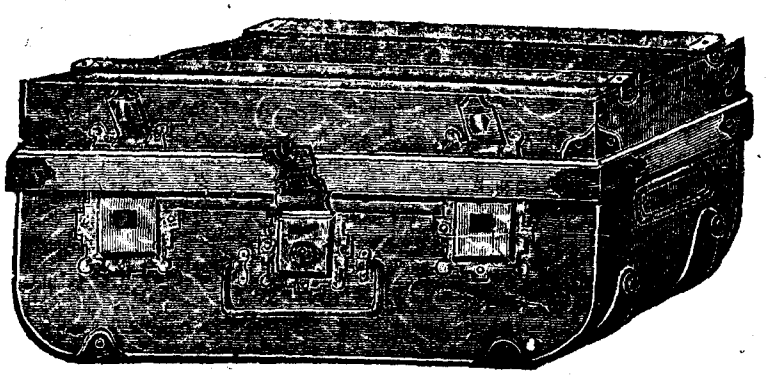
ASSOCIATION REPORTS.

The first meeting of the Bihar and Orissa Veterinary Association was held at the Patna Secretariat Buildings on the 16th June 1924. Mr. D. Quinlan, the Director of the Civil Veterinary Department, Captain P. B. Riley, the Deputy Director of North Bihar Range and

FAMOUS STEEL TRUNKS OF NEGAPATAM

GOLD AND SILVER MEDALISTS

OFFICE REQUISITES



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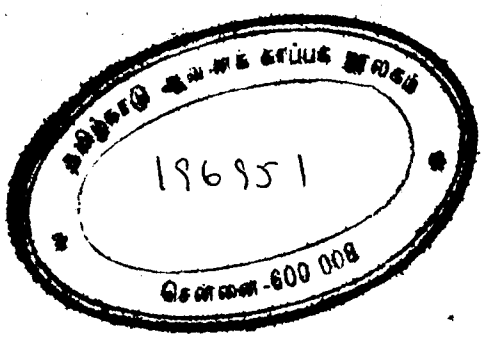
a fairly large No. of Veterinary officers from the different parts of the Province were present. Mr. D. Quinlan as patron of the association opened the meeting. In doing so he thanked Rai Sahib P. N. Das, Assistant Director, the President and the Members for asking him to be the patron and gave practical advice as to how the association should be run to make it a really useful body. He said that Veterinary officers who are trustees of the live stock and protectors of cattle of the country should prove by demonstration their usefulness and try to inspire faith in the mind of the agriculturists. In conclusion Mr. Quinlan said that the association filled a real need, that the members should do all they could to make it a success by keeping in view its aims and objects which are in the main the improvement of Veterinary science and the status of the officers of the department, and the most important of all is bringing to the notice of the public by efficient service, their value to Indian Agriculture through treating diseases in a scientific and rational manner. The agenda included six subjects all of which were passed unanimously, and great enthusiasm prevailed.



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THE EDITOR,

Madras Veterinary Journal,

CHEPAUK, MADRAS.

PRINTED AT THE N. M. S. PRESS, VEPERY, MADRAS.