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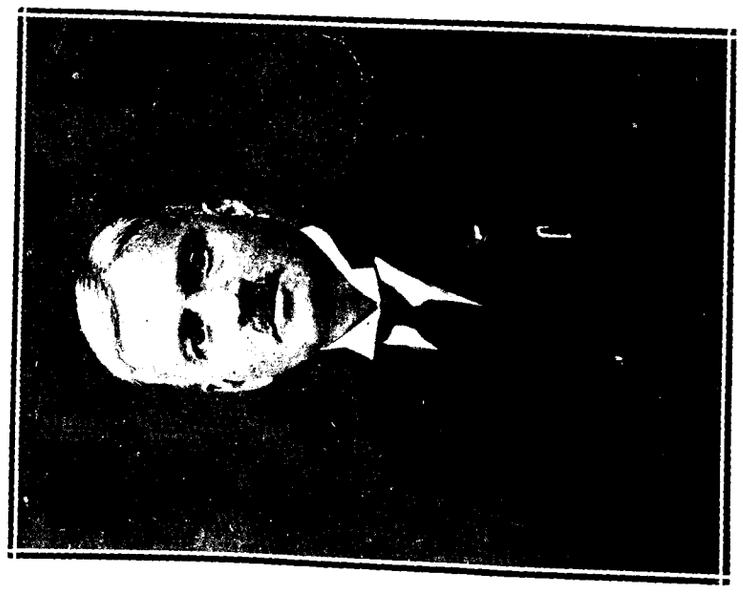

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who has been appointed as
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THE

INDIAN VETERINARY JOURNAL

Vol. XV. **OCTOBER 1938.** **No. 2.**

General Articles.

A DESCRIPTION OF THE TREMATODE, *ECHINOCHASMUS NARAYANI*, N. SP., WITH A COMPARISON OF SOME OF THE KNOWN SPECIES OF THE GENUS.

BY

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Madras Veterinary College.

DURING the years 1932 and 1936, intestinal parasites were collected from the common kite, *Milvus migrans govinda.* In this collection the trematode parasite which is to be described below was found. The measurements noted are in millimetres.

The worms are lanceolate in shape, broad posteriorly and tapering towards the anterior end. The average dimensions of each worm are $1{\cdot}5-1{\cdot}8\times0{\cdot}60-0{\cdot}62$ (Fig. 1, Plate VII). The cuticle is covered over with backwardly directed fine spines. They are dense over the region between the anterior end and the middle of the body; behind this area they are few and far between, and are entirely absent round about the posterior extremity.

The oral sucker measures $0{\cdot}05-0{\cdot}06$ in diameter and is situated at the anterior end. The prepharynx is very small.

Behind the oral sucker is a collar or girdle of 24 spines arranged in a single row with a break at the dorsal side (Fig. 2, Plate VII). These spines are larger towards the dorsal side except the three situated at either end which are the smallest. They are all directed posteriorly and measure 0·03 – 0·06 × 0·05 – 0·02.

The pharynx is a muscular organ measuring 0·08 – 0·12 followed by an œsophagus 0·18 long which bifurcates into two intestinal cæca in front of the acetabulum or ventral sucker. These cæca extend as far as the posterior end of the parasite running parallel to the sides of the body.

The ventral sucker lies at the junction of the first and the middle third of the parasite and measures 0·14 × 0·18.

The testes are two in number occupying the posterior half of the body and disposed one behind the other along the median line. The anterior one is more rectangular in shape with a slightly indented margin and measures 0·35 to 0·40 × 0·16 to 0·20. The posterior testis is more or less triangular with very few notches in its margin and measures 0·30 – 0·35 × 0·21 – 0·25.

The ovary is circular and is situated in front of the anterior testis to the right of the median line and measures 0·10 – 0·12 in diameter. A short uterus and oviduct are traceable to the right of the ovary. A few eggs are found in the uterus between the ovary and the genital pore. They are operculated, oval and darkish brown in colour and measure 0·06 × 0·048.

The vitellaria (Fig. 3, Plate VIII) are disposed laterally commencing from about the level of the ventral sucker to the posterior end of the parasite. They become confluent or continuous closely behind the posterior testis. A cross-duct passing between the ovary and the anterior testis connects the vitelline glands of both sides. The vitellaria do not extend anterior to the ventral sucker.

There is a v-shaped excretory pore at the posterior end in the median line.

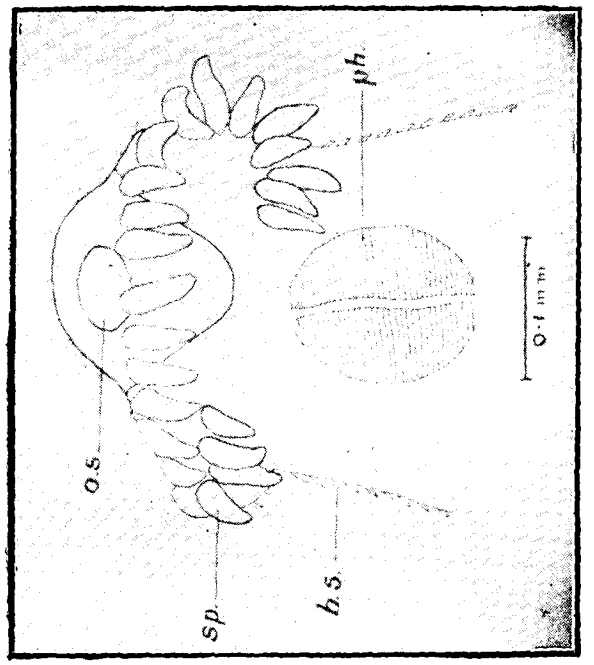

FIG. 2. Anterior part of the parasite showing the collar of spines and pharynx.

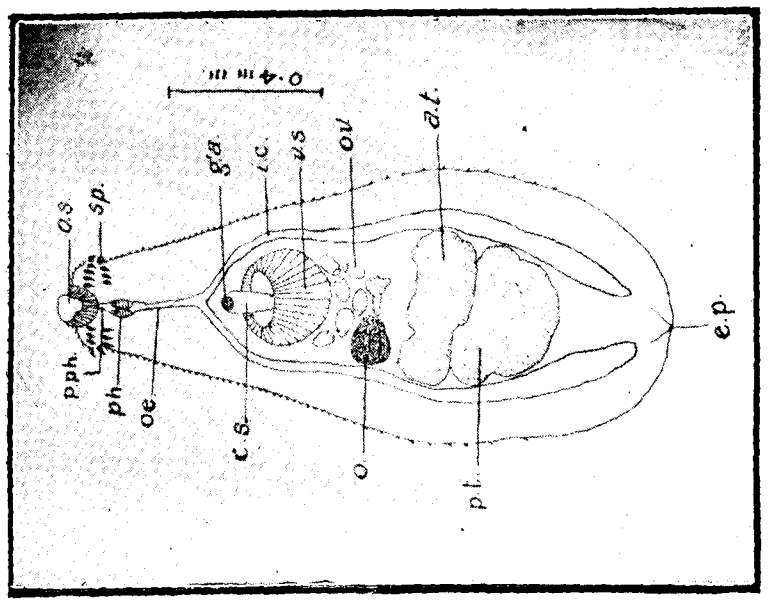

FIG. 1. *Echinochasmus narayani*, n.sp., showing details of internal anatomy.

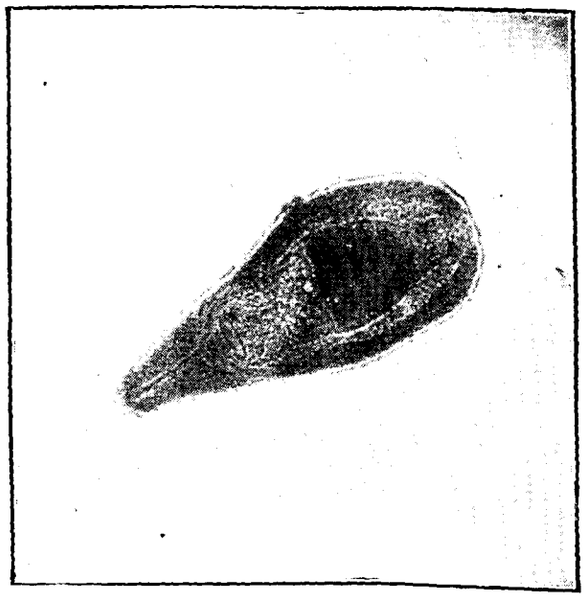

FIG. 4. Microphotograph of the entire parasite.

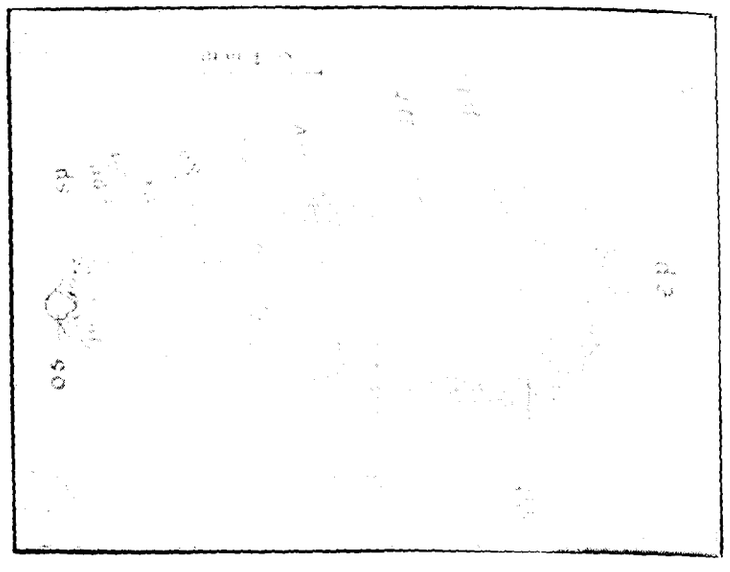

FIG. 3. *E. narayani*, n.sp., showing extent of vitellaria and other details of structure.

Discussion on the Specific Identity of this Parasite.

The genus *Echinochasmus* was created by Dietz in 1909 and a number of species have since been included in it. A table showing the comparative description of eight of the recorded species is appended. It was not possible to get at the description of the other recorded species as the relevant literature was not available. The parasite under study resembles in many aspects the species already recorded. However, the following points of difference noticed appear to be of sufficient importance to warrant the creation of a new species for this trematode. These are: (1) the extent of disposition of the vitellaria which start from about the level of the acetabulum and extend right down to the posterior end of the worm on both sides occupying the post-testicular space, (2) the comparatively small size of the helminth, and (3) the notched condition of the margin of the testes.

On these points of difference I consider the parasite new to science and propose to name it *Echinochasmus narayani.*

Specific Diagnosis.—Body broad posteriorly and tapering in front. Cuticle beset with spines at the upper or anterior half of the body. Girdle of 24 spines. Ratio of suckers 1 : 3. Vitellaria do not extend in front beyond the level of the ventral sucker. Margin of testes slightly notched.

Host.—Milvus migrans govinda (Common kite).

Locality.—Madras, South India.

Acknowledgments.

It is my pleasent duty to thank Mr. T. J. Hurley, Principal, Madras Veterinary College, and Rao Sahib M. Anant Narayan Rao, Lecturer in Parasitology, for the encouragement received in this work. I am much indebted to Mr. M. Dharmarajan, Assistant Lecturer in Biology, for help rendered. I have also to thank Mr. Doraiswamy Mudaliar for the illustrations appearing in this paper.

RINDERPEST—PREPARATION OF SERUM.

BY
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(Continued from page 35.)

Another method of hyperimmunisation, which is employed to a great extent in Africa in the Serum Institutes of Egypt, Nigeria, etc., is the " intramuscular method ". It consists of injection into the muscular parts of the animal of fairly large quantities of citrated virulent blood, the blood being introduced gravitationally. The details, such as the quantity of blood injected, the number of times the animal receives the massive doses before being bled, vary in the different institutes. In Nigeria an animal is first immunised by the serum-simultaneous method and bled three weeks after the reaction has subsided, and ten days later it is hyperimmunised with a dose of 3·75 c.c. citrated virulent blood per pound body weight. If the animal proves to be immune under test, the hyperimmunisation dose is 7·5 c.c. of citrated virulent blood per pound body weight. After this it is rested for ten days, the temperature being taken daily and then bled for serum. After a course of bleedings following hyperimmunisation, the animal is rested for a period varying from six to three months and again injected at the rate of 7·5 c.c. per pound body weight and bled for serum. This routine procedure is continued thus and animals are retained for serum production for several years. It has been found preferable to introduce fresh animals and dispose of those, which have passed through a course of about three hyperimmunisations, as this helps considerably to maintain the potency of serum at a high standard.

It is not usual to have a reaction following hyperimmunisation due to rinderpest. Occasionally a thermal reaction, which lasts for a day or so, may occur but this is generally due to the direct effect of introduction into the system of a large quantity of blood. It is not uncommon to have trypanosomiasis infection amongst the cattle employed for serum production in consequence

REFERENCES.

Bhalerao, G. D. (1924).—"The Trematode of *Corvus insolens*," *Parasit.*, **18**, 387-398.

Braun (1911) quoted by Tubangui (1912).—*Proc. U. S. Nat. Mus.*, **60**.

Ciurea (1915) quoted by Tubangui (1912).—*Ibid.*

Nicoll (1914).—"The Trematode Parasites of North Queensland, II," Parasites of Birds, *Parasit.*, **7**, 105-126.

Price (1931).—"Four New Trematode Worms," *Proc. U. S. Nat. Mus.*, **79**, iv.

Railliet and Henry (1919) quoted by Tubangui.—*Proc. U. S. Nat. Mus.*, **60**.

Tubangui (1922).—*Proc. U. S. Nat. Mus.*, **60**.

Dietz (1910).—"Die Echinostomiden der vogel," *Zoolog. Jahrboch.*, **12**, Supplement, 461.

Looss, A. (1899).—"Weitere Beitrage zur Kenntniss der Trematoden fauna Aegyptens," *Ibid.*, **12**, Syste, 686.

Explanation of lettering in Figures.

a.t.	anterior testis.	*œ.*	œsophagus.
b.sp.	body spines.	*o.s.*	oral sucker.
c.s.	cirrus sac	*ov.*	ova.
d.	duct connecting vitellaria of both sides.	*ph.*	pharynx.
		p.ph.	prepharynx.
e.p.	excretory pore.	*p t.*	posterior testis.
g.a.	genital atrium.	*sp.*	spines.
i.c.	intestinal cæca.	*vit.*	vitellaria.
o.	ovary.	*v.s.*	ventral sucker.

of such massive blood injections and some thermal reactions may be attributed to this.

The "intravenous method" consists in hyperimmunising by introduction into the jugular vein of the serum maker of the virulent material obtained from the virus producer. Either direct connection is made between the carotid artery of the virus producer and the jugular vein of the serum producer or citrated virulent blood is obtained from the virus producers and injected intravenously into the serum maker. This method of hyperimmunisation is probably not adopted in any of the serum institutes either in India or elsewhere, as it has several disadvantages though the technique is simple. It was employed extensively before the Great War in German East Africa but was abandoned as the losses were heavy. One of the disadvantages is that, as a result of the transfusion of blood from the virus producer into the serum maker, the patient, *i.e.*, the receiver frequently collapses in a few minutes and dies, this being due to the mixing of cells belonging to different groups. Such losses increase with each successive hyperimmunisation, the animals becoming sensitised to the blood of the donor which is incompatible with the blood of the receiver. It is not known whether the serum obtained by this method of hyperimmunisation is superior to that obtained by other methods.

At Muktesar this method was tried as an experimental measure on the hill cattle as serum makers, the virus producers being the usual hill bulls. Instead of blood, the virulent mixture, *i.e.*, blood and pot. citras solution, equal parts, was injected intravenously ; the quantity was regulated according to the minimum rate permissible for such serum makers in the subcutaneous method of hyperimmunisation. The serum produced from the animals, that had received a large number of such injections, was not subjected to test but that produced from serum makers, which had been hyperimmunised twice by the intravenous method, was found to be not more potent than the one produced by the subcutaneous method. The injections produced distressing symptoms in most of the serum makers, some manifesting them during

the operation, others soon after the completion of the injection. The symptoms were: difficult breathing, tumultuous action of the heart, foaming at the mouth, eyes rolling, injected and protruding. The few animals that stood the operation fairly well, would exhibit symptoms, such as extreme dullness, slight laboured breathing, and inability to walk. As this method gave rise to the above symptoms and even deaths occurred in a few cases, it was considered unsuitable for adoption as a routine measure.

The "intrarumenal method" of hyperimmunisation consists in introducing virulent material into the rumen of the serum maker. It was introduced by Hornby and has been used in Tanganyika Territory for the production of serum. The advantages claimed are that the operation is simple and causes the animal little or no pain. There is no risk of transmitting any protozoan infection present in the blood of the virus producer and material other than blood can be easily introduced into the rumen. The disadvantage is that the serum produced is not very potent as comparative tests made in Nigeria between the sera prepared by the intramuscular and intrarumenal methods proved that the serum by the former method was more potent than that prepared by the latter method and that the serum prepared by the intrarumenal method did not give sufficient protection to the Nigerian cattle but was potent enough to protect the cattle of the Tanganyika Territory, which were probably more resistant. The process of hyperimmunisation is to bleed the virus producer about the sixth day after inoculation, when the temperature is high, withdraw about 2,000 c.c. of blood and transfer this into the rumen gravitationally or by some other mechanical method. The process is repeated on the following day and the animal bled after an interval of about ten days.

The so-called "immune method" of preparing serum consists of immunising buffaloes by the serum-simultaneous method and bleeding them several times from the 18th day at certain intervals. It was introduced at Muktesar by Dr. Edwards in 1923. Before describing the method, it would be well to explain the reasons which led to the abandonment of hyperimmunisation and the adoption of the immune method.

For the hyperimmunisation of the large number of serum makers it was necessary to maintain virus producers in order to provide the virulent material and also keep alive the disease. The number of hill bulls, that had to be inoculated daily with rinderpest blood depended upon the demands for serum and the number of serum makers employed. Consequently a considerable number of bulls had to be bled to death from the artery and vein daily to provide the virulent material for hyperimmunisation of the serum makers that became due by rotation. This in itself entailed heavy expenditure and loss of life and the fact, that some difficulty was being experienced each year in obtaining the hill bulls and higher prices were being demanded for the same, gave a serious aspect to the whole problem.

As a result of the several subcutaneous injections in massive quantities, non-absorption of the material and consequently abscesses of varying sizes frequently occurred. Many of the animals having abscesses or wounds could not be bled for serum and at times, when the demands for the anti-rinderpest serum were heavy and urgent, the Institute could ill afford such loss.

It was believed that hyperimmunisation either increased the potency of serum or helped to maintain the previous high standard of potency and hence a serum maker received several massive injections of virulent material. But experiments carried out by Pool in this connection revealed that there was deterioration in the potency of the serum after the third injection. In the later experiments, Dr. Edwards observed that hyperimmunisation of a serum maker, after it was bled a few times, did not increase the potency of the serum. The fairly high standard of potency of the serum, which existed when the hyperimmunisation method was in vogue, was due to the large number of new animals that were being immunised and the highly potent serum obtained from "A" on the 21st day bleedings, before they received the first injection.

A few preliminary experiments were carried out, in which serum was prepared by the immune method and no hyperimmunisation was done. The animals were bled every week up to the

eighth week after they had recovered from the serum-simultaneous inoculation. The samples of sera from the first three bleedings on being subjected to test were found to be highly potent and protective for the hill cattle even at the minimum rate of 30 c.c. per 600 lbs. and the sera from the later bleedings proved to be potent and protective at the rate of 90 c.c. per 600 lbs. which is the standard rate of serum for preventive inoculation of hill cattle against rinderpest. A repetition of the above experiment on a larger scale confirmed the previous results, and the evidence thus obtained was sufficiently strong to justify the adoption of the immune method as a routine measure for the preparation of anti-rinderpest serum.

In the immune method the buffaloes were immunised by the serum-simultaneous method, the dose of defibrinated virulent blood being 1 c.c. and the rate of anti-rinderpest serum being according to the brew of serum employed. About the seventh day from the date of virus inoculation, the animals were administered a reinforcing dose of 5 c.c. virulent blood alone and from about the 18th day blood was drawn for serum at the rate of 4 to 6 c.c. per lb. body weight at fixed intervals, up to the 68th day and the animals then discontinued. As a result of further experiments, the intervals and the period up to which the bleedings should be taken were slightly altered. The bleedings were classified, the first few bleedings being grouped under "L", the later bleedings under "M", and still later bleedings under "N" and so on, and the samples of sera of the different groups were tested separately. The sera of the "L" group invariably showed a high degree of potency, being protective at 30 c.c. per 600 lbs. body weight for hill cattle, while the later bleedings sera were less potent though protective at 90 c.c. per 600 lbs. body weight.

A few years after the introduction of the immune method, an important and interesting experiment was planned and executed by Dr. Edwards to ascertain the nature of reaction best suited for the production of a potent serum and the stage at which the antibody content would be at the maximum. The buffaloes were divided into four batches. They were all inoculated with virulent blood on the first day and only animals of the first lot inoculated

as well with anti-rinderpest serum. The second lot of buffaloes was inoculated with serum the following day, *i.e.*, 24 hours after the virus injection, the third lot and fourth lot were inoculated with anti-rinderpest serum on the second and third day, *i.e.*, 48 hours and 72 hours after virus injection The animals were all bled each at 4 c.c. per lb. from the 9th day to the 51st day every third day and the samples of sera of each lot and each day kept separate. The reactions of the animals of Lots 1 and 2 were mild and decided, while the reactions of the animals of Lots 3 and 4 were fairly severe. All the samples of sera were tested on the hill cattle the virus employed being the same for all the test animals. The results were interesting and instructive. The Lots 1 and 2 showed a potency curve, which stood highest about the 18th and 21st day and gradually declined to the 51st day. In the Lots 3 and 4 the potency curve was highest at the 18th and 21st day but later became irregular. The curve showed a gradual decline at first but later on took a zig-zag course, the potency being high and low up to the 51st day. The 9th and 12th day sera in all the lots showed a very low antibody content and did not protect the test animals. The 15th day samples indicated a slightly higher content but the curve took a sudden and very high rise at the 18th day in all the lots. When the potency curves of all the lots were compared, the curve of Lot 2, *i.e.*, the lot in which the buffaloes were injected with serum 24 hours after the virus inoculation gave the best impression, as the potency was highest at the 18th day and gradually declined up to the 51st day. So it was concluded that a mild and decided reaction was the most suitable for the preparation of a potent anti-rinderpest serum and that the samples of sera from the 18th to the 30th day bleedings were highly potent, while the 9th, 12th and 15th day sera were unsatisfactory for the protection of cattle.

As a result of the above experiment it was decided to adopt the following procedure as a routine measure: the buffaloes were inoculated with 1 c.c. defibrinated virulent blood and 24 hours later were injected with anti-rinderpest serum at 40 to 60 c.c. per 600 lbs. according to the potency of the product employed. About the 7th

day from the date of virus inoculation they were again inoculated with a reinforcing dose of virulent blood alone 5 c.c. each. From the 18th day from the date of virus inoculation, the buffaloes were bled for serum at a rate varying from 4 to 6 c.c. per lb. body weight. The first few bleedings on 18th to 30th day were grouped under "L" and later bleedings from 35th, 40th and 45th day at 6 c.c. per lb. body weight under " M ". The later bleedings " N " though taken at first for some time were discontinued at a later date. This procedure remained in force for a few years when a slight alteration was made. The alteration included the intravenous injection of 'nine salt' solution into each serum maker, two hours before it was bled for serum. Before the introduction of these 'salt' injections, careful experiments were carried out to ascertain the subtoxic dose of the important individual salts and the dose and effects of the combination of all the nine salts. It was expected that the salt injections would increase the potency of the sera particularly the later " M " bleedings sera. The preliminary experiments carried out in this connection revealed a slight increase in the potency of the serum, particularly of the " M " samples. Though this increase was not invariably present in all the tests, the 'nine salt' method was adopted for routine purpose as it would help to maintain, if not increase, the potency of the later bleedings sera at a fairly high level. The 'nine salts' were chlorides of Sodium, Potassium, Calcium, Barium, Iron, Magnesium, Zinc, Manganese and Copper, and the injection of the "nine salt" solution had to be done intravenously with great care so that the solution was not introduced under the skin. This method was abandoned later, when it was observed, that, during the rush of bleeding a very large number of serum makers daily, a considerable number of them developed swellings and abscesses along the jugular channel, owing either to faulty technique or the animal struggling during the injection.

In the preparation of serum, whether by the 'hyperinmunisation' method or by the 'immune' method, precautions have to be taken that the serum maker on the day of bleeding is in a fit condition for the operation. The animal should feed well, have

normal temperature and not exhibit any of the symptoms, such as diarrhœa, etc., suggestive of any infectious or non-infectious disease. In case of doubt it would be advisable to err on the safe side and exclude the animal from bleeding, and keep the same under observation.

SOME COMMON DISEASES OF POULTRY IN MYSORE AND HOW TO DEAL WITH THEM.*

BY

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POULTRY in Mysore are victims to a great variety of diseases most of which are fatal to them. For this reason poultry farming is not being taken up extensively by the rural population in the State although it could be a very valuable cottage industry.

Ill health in poultry may arise from hereditary factors, from defects of management, from attacks of disease-producing agents or from a combination of any or all of these, as is usually the case under rural conditions of poultry rearing in the State. Whatever the precise cause of trouble, there can be nothing more sure than that the goal to aim at is its prevention.

It is essential that a knowledge of the nature and symptoms of the various troubles to which the Fowl is prone, should be possessed by every keeper of birds in order that poultry farming may be a remunerative occupation. The object of this paper is to bring home to the layman the most important diseases to which his poultry usually succumb, the characteristic symptom or symptoms which would enable him to diagnose them more accurately and the practical hygienic or prophylactic measures he could adopt to decrease the incidence and prevent the spread of such diseases in them.

* Reprinted from the "Journal of the Mysore Agricultural and Experimental Union," Vol. XVII, No. 2.

In describing the most common diseases of the domestic fowl, only those that have a tendency to spread rapidly among poultry and cause the greatest damage to them are taken into account. They may be considered under the following four headings:—

1. Diseases caused by Parasites.
2. Diseases caused by Protozoa.
3. Diseases caused by Bacteria.
4. Diseases caused by Filterable Viruses.

1. Diseases Caused by Parasites.

Parasites that occasion heavy damage to poultry are those that are harboured in the intestinal canal and usually termed worms. If one happens to conduct post-mortem on a dead bird, takes out the intestines and cuts through them, it is not seldom that he encounters two types of worms: the round-worms and the tapeworms. The death of any bird from effects of internal parasites should be looked upon with apprehension, as serious results are often met with where massive infestation occurs.

Round-Worms.—Round-worms are the most common of the internal parasites and flocks infested with large numbers of these are unprofitable in the extreme. The birds are unthrifty, appear unkempt and suffer from diarrhœa and constipation. Young stock are most severely affected; they lose their weight; are stunted in growth and become very poor layers.

Tapeworms.—More than thirty different kinds of tapeworms are found to inhabit the alimentary tract of the fowl.

Symptoms.—If a bird is infested by large numbers of tapeworms, it is robbed of much food and the bird becomes unthrifty, shows a ruffled appearance of feathers and loss of flesh. As a result of the irritation produced by these worms, derangement of digestion, catarrhal conditions of the bowels and in laying hens, a loss of egg production and in growing birds a stunted growth are usually observed. Many tapeworms often cause death of the infested bird both directly and indirectly by rendering them highly susceptible to other infections.

Treatment.—To keep the birds permanently healthy and free from worms, it is always judicious to dose them periodically for worms—say at least every three months. Most of the fowls are found harbouring both round and tapeworms simultaneously and, as such, it is necessary that the treatment adopted is such as would expel both these varieties of worms. There is a large number of prescriptions in vogue intended for dosing poultry for worms, but by far the simplest and cheapest appears to be a mixture of finely powdered *Butea frondosa* and *Areca semina* (powdered areca nut) in suitable doses. These drugs are indigenous and could be obtained in all places. They are best given in doses of five grains each per bird mixed up in wet mash and fed to poultry for three consecutive days, to be followed on the third day with suitable doses of Mag. Sulphas in drinking water. In the absence of *Butea frondosa* 10 to 20 grains of powdered betel nut may be given to each of the birds kept starving for at least 18 hours.

Prevention.—One of the most important measures against all parasitic infestations of the digestive canal is to move the fowls upon fresh ground every two or three years. Another practical measure, which may be adopted at the same time, is to remove the excrement daily from the houses and destroy any parasites or their eggs, which may be in it, by mixing it with quicklime or by burning it. Frequent inclusion of garlic in the food and sprinkling tobacco dust into the mash have a deleterious effect on the worms.

Sick birds should always be isolated and confined and their droppings should either be burnt or treated with lime. Without these hygienic measures, medical treatment can only be partially successful.

2. *Diseases Caused by Protozoa.*

The most common disease of the fowl under this head is the Coccidiosis, caused by a very minute, unicellular animal organism, *coccidium.*

Symptoms.—In young chicks, the disease develops rapidly and the death-rate is usually high. The chick is weak, wings droopy

due to muscular weakness, loss of appetite or, in some cases, birds may even continue to eat. The chicks stand in bunches and peep much of the time. The droppings are fluidy, whitish or whitish-brown coloured and paste up the vent region and may prevent bowel movement. The discharges may be streaked with blood.

Post-Mortem Findings.—On opening the body cavity, the intestines appear congested and present ulcers throughout the tract, especially the cæca, which may be filled with a bloody semi-solid mass distending the organ considerably.

Treatment.—Give one-third teaspoonful of *Tr. Catechu* to each gallon of drinking water. Give the birds all butter-milk or sour skimmed milk which they will drink. Immediately remove all the sick birds and clean up and scrub the floor with a powerful disinfectant, such as a 1 : 1,000 solution of Bichloride of mercury or a strong solution of copper sulphate.

3. *Diseases Caused by Bacteria.*

Under this, three important diseases need be described, *viz.*,

(*a*) Fowl cholera,

(*b*) Bacillary white diarrhœa, and

(*c*) Fowl typhoid.

(*a*) *Fowl Cholera.*—This is one of the most common and a highly fatal disease of poultry caused by a very minute organism, *Pasteurella avicida.*

Mode of Spread.—The germs are eliminated from the body of the sick fowl through the bowel discharges, thus infecting the soil and polluting drinking water and feed.

Symptoms.—Depression is usually the first symptom ; the face, comb and wattles usually get darkened and discoloured. An exceptional thirst is characteristic of the disease The droppings are greenish-red and watery. Respiration is increased and later becomes laboured. Complete prostration sets in, preceding death.

Post-Mortem Appearances.—Body emaciation is not apparent. In some instances there is a darkening of the breast muscles. The layer of fat on the heart is studded with pin-point hæmorrhages. The intestines show an acute hæmorrhagic inflammation and the contents are mixed with a bloody mucus material.

Diagnosis.—Accurate diagnosis is based on the microscopical finding of the causal organism in a stained smear of the blood taken from a sick or dead bird and could be undertaken only in a laboratory. It is therefore wise, in such cases, to get the diagnosis confirmed in a bacteriological laboratory by sending an ailing or dead bird.

Treatment and Prevention.—The treatment of sick birds with specific immune serum in the initial stages of the disease has been successfully reported in some instances. But a more effective method of controlling the trouble is through the use of a vaccine. Strict isolation of the sick birds, cleaning and disinfecting the houses and the premises and the disinfection of drinking water through an effective germicide like Chinosol are to be advocated.

(*b*) *Bacillary White Diarrhœa.*—Commonly known as 'pullorum' disease of chicks is a devastating ailment of very young chicks caused by a minute germ, *Salmonella pullorum*. The heaviest loss in chicks is from the first to the fourteenth day after hatching.

Symptoms.—In infected chicks, the wings are droopy and the feathers ruffled. The birds appear drowsy and eject out a frothy brownish white diarrhœa very frequently. There is a tendency for the abdomen to bulge. The chicks usually die in large numbers. Chicks having the infection when young and recovering from it subsequently, retain the germs in the ovaries as adults and transmit the disease to the progeny, a consideration which is not to be overlooked in undertaking preventive measures.

Post-Mortem Findings.—Yellowish-brown colouration of the liver, congested kidneys, unabsorbed yolk sac are some of the usual pathological lesions observed.

Control.—Experiments indicate that feeding with sour milk (butter-milk) has a tendency to reduce mortality.

The elimination of the disease lies in the elimination of reacting adults from the flocks. The reactors are easily recognised by means of a reliable serological test—Agglutination test—which could be undertaken only by an experienced bacteriologist.

(*c*) *Fowl Typhoid.*—Also known as Klein's disease of the fowl is a septicæmic infection of poultry caused by another minute organism, *Salmonella gallinarum.*

Occurrence.—Fowl typhoid is usually a disease of the adult bird and range birds, occurring in late Autumn and early Spring. The germ is voided in large numbers by birds suffering from the disease and is transmitted from place to place by surface washings, mechanical conveyers such as animals and man, streams and brooks, etc.

Symptoms.—The disease is of such a character that it may well establish itself before any symptoms are observed. The bird shows signs of dosing and, on being disturbed, exhibits a weakness of gait. The droppings are watery and sulphur-coloured. The loss of flesh may be rapid according to the severity of the disease. The face and comb are usually highly anæmic.

Post-Mortem Findings.—The carcass is usually emaciated, the liver greatly enlarged, streaked and friable and the spleen enlarged.

Control Measures.—Strict sanitary precautions are to be observed; all sick birds should be isolated and destroyed; some potent disinfectant to be added to the drinking water and, if possible, the birds should be removed to new grounds. But by far the most effective method of controlling the disease is to vaccinate all the birds, if possible, with an autogenous vaccine. This could be done through timely intimation to the Veterinary Department.

4. *Diseases Caused by Filterable Viruses.*

By far the most infectious diseases of poultry are those caused by a host of ultra-microscopic organisms, known as filterable viruses and which are so small that they cannot be seen under the

lmicroscope. These ailments are characterised by rapid spread when once they appear in the flock and in most cases by a sad ack of methods of controlling them. The most common diseases under this head encountered among the poultry in Mysore are:

(*a*) Chicken-Pox and Roup;
(*b*) Fowl Pest;
(*c*) Ranikhet Disease.

(*a*) *Chicken-Pox and Roup.*—These two diseases cause serious loss to poultry farmers by decreasing the egg production. They are dealt together, as they have a common etiological entity—the chicken-pox virus.

Symptoms.—The first sign of the disease is a small red pimple on the comb, face and wattles, the nodules varying in size from a pin-head to that of a pea or even larger. Later these nodules ulcerate on the surface and it is in this condition some secondary organisms find an easy entry into the lesions and set up complications usually termed Roup or Canker of the mouth. It is at this point one finds the disease-control hard.

Treatment.—Treatment for chicken-pox consists in painting the affected part with Tincture of iodine or still better with a dilute picric acid solution.

Prevention—All dead fowls should be burnt and sick ones removed immediately from the flock for treatment or for effective disposal. Vaccination of all the other birds must be undertaken with the help of an efficient vaccine, which could be done on application to the Veterinary Department.

(*b*) and (*c*) *Fowl Pest and Ranikhet Disease.*—These are the two very acute, highly infectious diseases of poultry encountered in the State, causing unaccountable damage to the industry.

The period of incubation is from 2 to 4 days in fowl pest and somewhat longer in the others—5 to 10 days. But it is not uncommon that chronic cases of fowl pest lingering on for one to two weeks and more acute cases of Ranikhet disease of a fulminating type are encountered, misleading one as to its identity.

Mode of Spread.—Fowls become infested through contaminated feed and water. They may be spread through droppings used as fertilizer, litter, feeding and drinking utensils, by wild birds such as sparrows and crows.

Symptoms.—The diseases are characterised by their extremely infectious nature, rapidly progressing course and high mortality in the affected flock. The birds drink considerable water, soon lose the use of their wing muscles resulting in their dropping. As disease progresses the comb and wattle become dark, symptoms of paralysis appear, beginning at the legs, and in the last stages the birds lie down comatosed and die.

Post-Mortem Lesions.—In both these diseases all lesions may be absent or found only in part. The diagnosis of these two ailments of poultry is by no means easy, especially by a layman, and involves careful examination of very minute lesions in several organs and resorting to some specific bacteriological and serological tests which could be carried out only in a well-equipped laboratory. For these reasons, it is advisible to get the advice from the Department when the disease appears in a flock.

Control Measures.—There is no remedy from a medical standpoint. Attempts to prepare immune sera and specific vaccines against the disease by a host of workers in the line have been futile so far. These problems are also being tackled by the Veterinary Department in the State and one may expect to find some hopeful solution in course of time. Till then all efforts should be directed towards prevention of the spread of the disease by taking recourse to the following:

Sick fowls should be disposed of promptly without contaminating the premises and the carcases burnt. The healthy fowls should be removed to new quarters and carefully watched for signs of disease. Houses and rooms should be thoroughly disinfected with an efficient disinfectant like Cresol (3%). The drinking water may be rendered safe by adding Chinosol in dilutions of 1 in 10,000.

NEW RESEARCHES ON SOME HELMINTHIC DISEASES OF INDIA.*

By

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

References to the worms of domestic animals are no doubt available in some of the ancient Indian veterinary treatises such

* Paper read at the International Veterinary Congress, 1938, Theme V, Section IV, "Parasitology and Parasitic Diseases".

as *Aswavaidak*, and one knows that collections of Indian parasites have been examined by some early helminthologists abroad from time to time. However, the attention of the comparatively few veterinary workers of the Army and Civil Departments of the Government of the East India Company and its successor, commencing practically from the beginning of the nineteenth century till the present time, has been so completely pre-occupied with the ravages of epizootic diseases like rinderpest and surra that nothing beyond the sporadic and superficial consideration of the gross anatomy and geographical distribution of parasites could be made. Without attempting to go into details of the helminths recorded from India in the early days, when hydatid cysts, the so-called *Filaria oculi* of the aqueous humour of horses, liverflukes and amphistome infestations were first seen, it is remarkable that the instances, where the exact role of helminths as disease-producing agents was recognised, have been notably very few indeed. The effect of parasitic worms might have been less spectacular than that of epizootics of bacterial and virus origin, but quite a number of highly refractory diseases had forced themselves on human attention due to their frequent occurrence and prominent gross lesions, though their helminthic etiology remained enshrouded in mystery until a few years ago, providing, as will be seen below, a very rich field for speculation and controversy.

Turning to other countries as well, the position is far from satisfactory since the number of diseases, proved to be of helminthic origin, are comparatively few when the helmenthic fauna of the live-stock of each country is taken into consideration, and the life-history of quite a number of worms is unknown. Helminthic diseases are known to be characterised by widely different lesions including hæmorrhages, inflammatory and ulcerative reactions, nodular growths and even cancerous changes; and regarding the association of the last mentioned with helminthiasis, which is rather exceptional, it may be mentioned in passing that the writer has encountered them in equine gastric habronemiasis, and also in another nematode infection, the so-called hump sore of cattle. The basic facts, which are responsible for the production of these wide

variations in the clinical manifestations of all helminthic invasions, are awaiting elucidation but, when the findings on equine microfilariasis of all kinds recorded by different workers were analysed, Sen (1927) failed to resist the impression that microfilariasis of the horse is not associated with any clear clinical picture. Again' facts regarding the everchanging or dynamic biological relationship of helminth parasites to the inside of the body of their hosts, and the physiological mechanism, by which enormous number of parasites are harboured without the exhibition of any apparent deleterious effects while a hitherto innocuous parasite leads to lethal effects, form subjects of absorbing interest, and here verminous pneumonia in ovines (due to *Varostrongylus pneumonicus*) and in the buffalo (due to *Protostrongylus* sp.) may be cited. Incidentally, the observation made in India that, when the snails *Indoplanorbis exustus* are infested with an aquatic oligochæte *Chætogaster limnæi*, they cannot be infected with trematode larvæ, must be mentioned. Furthermore any attempts made with the aid of the microscope, to follow parasitic action in the tissues of the mammalian hosts, to appreciate the nature of the tissue damages and to correlate the histological lesions incited are likely to increase our powers of control over helminthiasis.

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

like plaques in horses, Pease), Hump sore of cattle, 'Periodic Ophthalmia' in horses ('Verminous Ophthalmia'), and a new microfilarial dermatitis of cattle.

Bovine Nasal Granuloma.

Of the various types of nasal growths affecting cattle and other animals in India, 'snoring disease' or 'nasal granuloma' of cattle has been the most important and widespread, being known to exist for nearly a century. The symptoms and lesions need not be described here. Suffice to say that the first histological work (by Krishnamurti, 1922, 1925, and by Cooper, 1923, 1925, 1931) revealed certain alcohol and acid-fast 'clubs' and 'granules' in the granulomatous tissues of the disease, akin to those of actinomycosis (*Cf.* Schlegel's earlier report on "Nasal Actinomycosis in German Cattle"). Streptothrix strains were cultivated but no Gram-positive mycelia detected in the 'granules'. The non-involvement of the jaw and the tongue, chronic nature of disease, its enzootic distribution, repeated failure in experimental transmission, failure of Iodine treatment, and the specific response to tartar emetic, also simultaneous existence of Rhinosporidiosis in an enzootic area (Edwards, 1929, and Das, 1929) were known to characterise the disease. Besides the actinomycotic theory (Krishnamurti, 1 22), the disease was ascribed to the 'nose-string', nasal polypus and to a protozoan organism (Cooper, 1931) till finally the disease was proved at Muktesar in December 1931 to be a clinical manifestation of schistosomiasis, in which the parasite, closely allied to *S. spindalis* but possessing differences suggestive of a new species, deposits its ova in a very unusual site—the nose (Datta, 1932). The eosinophile character of the 'clubs' and 'granules', intense eosinophilia in the tissue, and around the essential lesions of the disease—the *follicles* of Krishnamurti, which were identical with bilharzial *pseudotubercles* of Fairley, containing in the central core the highly refractile egg shell with larval remnants or even fully developed miracidium were demonstrated. Complete 'spindalis'-type of ova, and miracidia, both free and enclosed, and even extruding from the egg shell were seen in sections in addition to worms *in copula*. Unusually prominent cuticular tubercles were

observed on the males, and the 'clubs' around the egg shells were explained as a manifestation of the host's protective reaction, similar to what happens in a variety of affections including *Schistosomiases turkestanicum* (Yamagiwa, 1931) and *japonicum* (Hoeppli, 1932). The 'clubs' may be 'albumen-mineral compound' of Levaditi or may represent the secretion of the lateral glands of the miracidium (Hoeppli). Live specimens of the nasal parasite were collected during surgical operation, and the cross-section of a male schistosome, cut posteriorly to the bifurcation of the intestinal cæca, was identified in a microphotograph said to be of an actinomycotic granule in Krishnamurti's Memoir (1925). Regarding differential diagnosis of Rhinosporidiosis, a fragile, spongy and pediculated growth, as opposed to the solid growths with broad bases of nasal schistosomiasis, the microscopic examination of caustic potash treated deposits from nasal discharges reveals the specific bodies of both.

The research activities on the problem were greatly energised on the above discovery and several articles were published in close succession confirming the etiological finding, and what is more, in extending the work to include specific identification of the parasite, and curative and other control measures in enzootic areas. Space does not permit details but the most outstanding contributions are contained in a series of articles by Rao (1933, 1934). The disease has been recorded from the buffalo, sheep and goats. Differential features of the adults, their miracidia, cercariæ and ova have been described in detail. With carefully planned transmission experiments, employing the cercariæ of the nasal schistosome and those of *S. spindalis* by the oral and nasal routes, the intestinal parasite *spindalis* has been differentiated from the nasal parasite and Cercariæ Indicæ XXX (Sewell, 1922), has been proved to represent a developmental form of *S. nasalis*, Rao 1932. The intermediary agent of *S. spindalis* is *Planorbis exustus*, and that of *S. nasalis*, a Limnea species. Further the fact that Sewell (1930) placed Cercariæ Indicæ XXX, and Cercariæ of *S. spindalis* into two separate groups in his classification is of more than ordinary significance. Tartar emetic is recognised as the cheap and effective curative agent for the disease, and the latest indication (Rao, 1936) shows

that for every 100 lbs. body weight 1·5 grains may be repeated daily for six days, 2·5 grains may be given every alternate day for three days, and any dose exceeding 3·5 grains is dangerous, particularly if repeated. *Antimosan*, though equally efficacious, is comparatively costly for general use. Snoring diseases of cattle have now been reported from Australia, (Albiston and Gorrie, 1935) and America (Creech and Miller, 1933), but the etiological agents are apparently different being blastomyces in Australia, and rhinosporidium-like in America.

Liver Lesions in Debilitated Equines.

The occurrence of persistent debility among equines in India, associated with nodulated calcareous lesions in enlarged livers has attracted attention since the observations of Frederick Smith in 1885. Hepatic cirrhosis of various kinds, generally ascribed to poisoning, has been recorded from different countries, but none excepting the cases of Oreste and Ercolani have any resemblance to the Indian condition. The problem has been one of the most difficult to solve, as previous studies indicated that "the lesions represented the end result of an infection, in which it was no longer possible to discover the causal agent" Calcareous degenerations in the horse (Smith) 'internal Bursati' (Oliphant, Meyrick, Smith, Burke) and other terms were employed. Hundreds of animals, which had hitherto been destroyed as 'worn out' and 'Chronic bad doer', have now been shown (Datta, 1933) to be cases of intestinal schistosomiasis due to *S. indicum* Montgomery, the predilection seat for the disposition of the ova being the distal portions of the intestinal tract, particularly the large colon and rectum, and the liver. The parasite does not localise in the central nervous system to produce paraplegia or *Kumri*. In extreme cases, the enlarged livers have weighed as much as 96 and 105 lbs., giving the subject the appearance of a gravid mare. The back muscles are greatly atrophied and the abdomen pendulous, resembling human cases of chronic schistosomiasis of the Far East, as depicted by Byam and Archibald (1923) and other authorities. The causative ova of *S. indicum* have been demonstrated as the incitant of lesions in most tissues including the liver, intestines, lungs, mesenteric and other lymph glands and the spleen.

Clusters of the ova have been found embedded in all layers of the intestinal coat, but only single ova in the other tissues. *Indoplanorbis exustus* has been seen to be common in enzootic areas. Cases of persistent debility in horses due to habronemiasis, (*H. megastoma*) strongylosis and malnutrition do occur in India, but the commonest incitant of debility is *S. indicum*. Diagnosis by fæcal examination is not always easy. The parasite occurs in the sheep, goats, cattle, buffaloes and the camel, and Montgomery (1906) failed to connect it with any obvious pathological entity. Control and curative measures are similar to those of other schistosomiases.

Bursati in Horses.

This summer complaint of horses had been known to Moorcroft in 1808 at Pusa, and very numerous articles on the subject have been published to date. The name is widely known abroad. The precise nature of the cause remained undetermined, and even to-day statements (Wooldridge, 1934) are made alleging that it is an established fact that helminths are not causally connected with Bursati in India. Circumstantial evidence and the analogies of "Summer sores" and similar complaints had suggested a habronemic origin of the disease (Sen, 1927) but authorities like Railliet (1915), du Toit (1916) have included several entities under 'Bursati', while Freeborne, Hart and Howell (1927) and Bayliss (1929) are disinclined to accept the habronemic origin. Even if the habronemic etiology is accepted, the exact species responsible for Bursati and other forms of "Summer sores" are not precisely known, and the mechanism of infection is by no means clear. For, while *H. megastoma* is recognised as the cause of Summer sores in France, and of swamp cancer in Australia, *H. muscæ* appears to be the cause in Brazil, Congo and India. In one of the latest publication (Descazeaux) on "Less plaies d'ete", the "umbilicated crusts" (Gunn) of India have been included wrongly under Bursati, as will be shown below, but the important point made by the author is that *Summer sores* are produced by the deposition of larvæ into wounds by flies, and that the cutaneous and pulmonary lesions occur particularly in horses which are

carriers of adult *H. megastoma* in the stomach. Descazeaux further states that the larvæ do not take the circulatory route to arrive at the cutaneous lesion, as their dimensions prevent their passage through capillaries, and that his experience shows that all horses affected with Summer sores are carriers of *H. megastoma*, while the wide spread *H. muscæ* is seen in all horses with or without sores. The author records the finding of habronema larvæ in the equine stomach towards the end of Summer. Contrary to the above experiences of Descazeaux, cases of cutaneous and pulmonary habronemiasis in the same animal have been found to contain in the stomach no other species than *H. muscæ* (Datta, 1933), and the parasites have been invariably found inside blood vessels in the earliest lesions, while large numbers of *H. megastoma* have been quite frequently seen in healthy horses with no apparent deleterious effect, similar to the experience of Knowles and Slocock (1931) in Egypt. Further Datta (1933) examined histologically *megastoma* gastric tumours, collected during different times of the year over a period of years, but could not confirm the view of Descazeaux that females bring forth young but once during 12 months. The disease in India has never been transmitted by direct or indirect contagion in the same or adjoining stable to healthy in-contacts possessing cutaneous wounds, and repeatedly the disease has been seen to appear first as intact tumours and to spread to different distal parts in an affected subject, indicating larval migrations through the circulation. The causative parasites have been seen in histological sections of the healed lesions, explaining the nature of so-called 'Bursati diathesis' observed by many workers. The habronemic etiology of Bursati has now been confirmed by other Indian workers.

Equine Lichen Tropicus.

Numerous articles have been published since 1879. Investigations from many different angles have been carried out, and numerous names applied to it. The condition has been likened to a number of human diseases like Prickly heat, Dhobie's itch to which it has no resemblance. Methods of prevention and cure hitherto adopted have been singularly fruitless though, like

sensu stricto) in those places has been proved to be a microfilariasis (due to unsheathed larvæ) similar to the ophthalmia associated with *Guatemala nodules* of man, but the adult remains undiscovered. Virus, bacteria, dietetic and extraneous factors were eliminated. The parasite larvæ were seen in the *substantia propria* of the cornea and in the lachrymal glands with definite histological reactions. Previously Crawford and Nicolle (1925) described what appear to be three different helminths from cases of ophthalmia.

A New Microfilarial Dermatitis of Cattle.

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

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

Bursati, spontaneous cures occur in the cold weather. The disease simulates Bursati in its seasonal incidence, non-contagiousness, recurrence in the same subject year after year, but is a more superficial affection. It is characterised by its severe irritable, chronic, papular and scurfy nature. The mane, tail, sides of the neck, shoulder region, and abdomen are predisposition sites. In the work carried out at Muktesar since 1931, young forms of a round-worm (microfilariæ) have been constantly demonstrated in the sections of the affected skin, and adult onchocercoid worms have been collected from nuchal ligaments of some of these cases (Datta, 1932). The disease may be described as a microfilarial pityriasis, and presents features of *Craw—Craw* of man of the West Coast of Africa. The drugs Novarsenobenzol and antimosan are recommended for injection. The microfilariæ have never been seen in the blood. In addition to India, the disease occurs in Sudan, the Philipine Islands (Underwood, 1934), U.S.A. (Alicata, 1936) and probably also in other countries, since some of the cases described as Queensland Mange (Tryon, 1888), Dry scurfy eczema, Mane and tail disease, Eran and scale eruption (Friedbergher and Frohner, translated, 1908), and as Dandruff, Seborrhœa oleosa and Sicca, Chronic scurfy eczema of the long-haired parts of the body of unknown etiology (Hutyra and Marek, 1926) must refer to the Microfilarial itch now discussed. Semmer (1871), Baruchello (1889) and Aldige (1920) perhaps were describing the same disease.

'*Dourine-like Plaques*' *in Equine Microfilariasis.*

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

'*Hump Sore*' *of Cattle.*

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

'*Periodic Ophthalmia*' *of Horses.*

The occurrence of *Thelazia lachrymalis* on the exterior of the eyes of horses, and the infection of the aqueous humour of the equine eye with the so-called *filaria oculi* have been known in India. Recently, however, a separate condition affecting one and sometimes both eyes of young horses in certain North Indian stud farms known for some time as Periodic ophthalmia (recurrent

PIG REARING AND PRELIMINARY INVESTIGATION INTO THE EXISTENCE OF SWINE FEVER (?) IN PROVINCE WELLESLEY.

By

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THERE are well over 100,000 pigs distributed in the Province Wellesley, an area of about 279 square miles. In all, there are about 10,000 breeding stock, and 102,000 slaughter stock, or approximately 112,000 swine in the Settlement, a figure very much in accord with our own departmental estimates of over 100,000.

The industry is practically entirely in the hands of the Chinese, undertaken often as a supplement to their ordinary living and entrusted to the women folk of the family who, in addition to house work and care of children, devote much time to the pigs and often to fowls and ducks as well. Most of the pigs are kept in large enclosures, more or less secured, on cocoanut estates. The pig breeders get the use of the land free or for a very low rent and the estate getting the benefit of the manure which is considered valuable. Proper sties are not built and generally only an attap roof is provided for shelter and the floor is of plain earth or sand. Much of the land used is low lying with many stagnant pools of water or mud, and the conditions generally are far from clean and sanitary. Some pigs are kept by vegetable gardeners and along the sea or rivers, by fishing people. These pigs are kept in sties under cleaner and more sanitary conditions. Ordinarily each breeder keeps two to three brood sows and rears the piglings for market, the brood sows being replaced when necessary by selecting from the owner's own piglings. A few rearers buy piglings when about two months old (*i.e.*, about weaning) to raise and fatten for the market. Keeping a stud boar is looked on with disdain by the breeders, especially by family men, and in consequence it is usually an elderly single man or a widower who keeps the boar, and takes it about, generally leading it by a cord, serving sows as required. Thus a large, strong and possibly vicious boar is not wanted by

these people and the boars in common use though easily manageable are small and of a poor type. The fee for service is usually fifty cents, and if the service is not successful further service is free of charge. The boar is expected to serve at least a sow a day, which is too much. The use of large boars of improved and quicker maturing type would be very advantageous to the industry. The young male pigs are castrated when 15 days old, and young females are spayed at about three months old, the operation being done by a skilled Chinese operator at a fee of ten cents to fifteen cents (2½ to 3½ annas or 2¾ to 4¼ *d.*) a male pig, and thirty cents (7 annas or 8½ *d.*) a female ; a few breeders are able to perform the operation themselves. Weaning takes place at about two months, when, with pigs kept in sties, the young pigs are separated from their dam. Uniformity of growth and fattening is aimed at as poor-conditioned unthrifty pigs are not bought by the dealers and are left as a loss to the owner. The pigs are sold, usually to dealers, when about six to nine months old, at prices from $15/50 (Rs. 23, annas 8 or £1 16*s.* 2*d.*) per pikul or one hundred and thirty-three and one-third pounds for young pigs to $20 (£2 6*s.* 8*d.*) per pikul or one hundred and thirty-three and one-third pounds for older pigs. The young and smaller pigs go to the Penang market and the older and larger ones to the Ipoh market. The stuffs commonly used for feeding are broken rice, rice bran, tapioca (*Manihot utilissima*), cocoanut refuse, chopped green vegetables (water hyacinth—*Eichhorina crassipes*), kladi (*Colocasis esculentum*), banana stems (*Musa*), fresh or salted fish and prawn dust. These are all boiled, mixed together into a sloppy mash, and fed in troughs. The bran and tapioca refuse is often mixed uncooked. Pigs kept in pens are bathed and the pens washed out after feeding. The green vegetables and, in places, the fish and prawn dust are the owner's own produce or gathering, but the other foodstuffs have to be bought, generally from the local mills, or imported from Kedah. Marketing is generally through dealers, or middlemen between the breeders ; most of these dealers supply the breeders with all their necessaries, especially food for themselves, their pigs and poultry —generally on credit and with the right of purchase of all the

breeder's produce. Few thrifty breeders are able to sell direct to the butchers. In general, the pig rearers complain that profits are very small and sometimes absent when compared to the cost of foodstuffs and the work and care necessary.

The Province Wellesley pigs are slaughtered for local consumption and are also exported to Penang Islands as well as to Ipoh Abattoirs (Perak) by train and motor lorry and by steamer to Singapore.

For several years cases of the so-called swine fever were reported very frequently amongst Province Wellesley pigs at the Penang Municipal Abattoirs as well as at the Ipoh Abattoirs. Due to the change of hands, once the pigs are sold, it becomes wellnigh impossible to trace the premises of the infected pigs. Table I will show the number of pigs slaughtered at three of the main abattoirs in Malaya and the number of cases of the so-called swine fever met with.

During the course of inspection, the writer detected some cases of the disease first at a place called Permatang To' Mahat. Acensus of all the pigs was taken and thereby he was able to keep a check on the mortality and course of the disease. The same procedure was adopted at all the other places mentioned below and very frequent visits were made. The Chinese pig rearers do not willingly co-operate with this department by reporting outbreaks of swine fever. The following were the places affected with swine fever in Province Wellesley :—

		Date of Commencement.		Date of Ending
1. Permatang To' Mahat	from	6-11-1936	to	27- 2-1937
2. Kampong Bharu	„	13-11-1936	„	10- 2-1937
3. Sungei Bakap	„	9-12-1936	„	16- 4-1937
4. Tanah Liat	„	8- 2-1937	„	2- 4-1937
5. Permatang Pauh	„	9- 3-1937	„	14- 4-1937
6. Kampong Simpah	„	19- 4-1937	„	18- 6-1937
7. Telaga Ayer	„	12- 8-1937	„	1-10-1937

(*Vide* Table II and Map attached.)

Control.

To prevent the spread of the disease, the free movement of pigs was prohibited and healthy ones from affected areas were permitted to be taken direct to the local slaughter houses, by boats to the Penang Municipal Abattoirs and by rail to the Ipoh Abattoirs only. This was, and still is, done with the view to minimising the loss to the pig rearers. Swine Fever notices both in English and Chinese were posted in all the affected localities and pig sties. The notice runs as follows :—

Swine Fever.—Until further notice all pigs in this area must be kept confined day and night in sties or enclosures so constructed that it is impossible for any pig, whether large or small, to get out of the sty or enclosure in which it is confined.

2. No pig may be brought into or removed out of this area for any purpose whatsoever without the written permission of the Government Veterinary Department.

(Sd.) CAPT. D. P. WHITE,
Government Veterinary Surgeon, Penang.

Also a notification, as follows, was gazetted in the *Straits Settlement Government Gazette*, November 20, 1936 :—

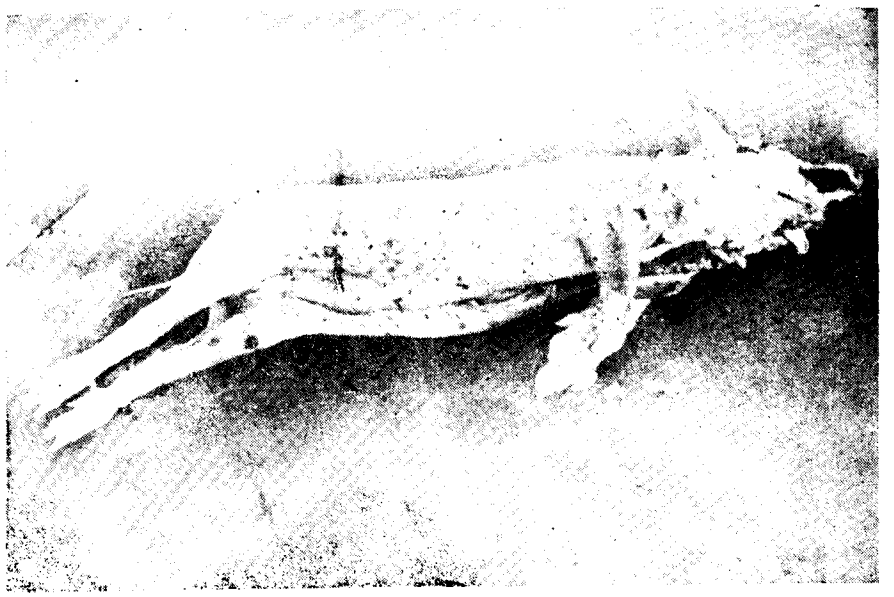
No. 3200. Ordinance No. 157 and the Swine Fever. NOTICE :— *Straits Settlement Government Gazette*, November 20, 1936, page 3116.

No. 3200. Ordinance No. 157 (Quarantine and Prevention of Disease). *Order made by the Government Veterinary Surgeon, under Rule* 42 (2) *of the Quarantine Rules*, 1935.

Whereas Province Wellesley has been declared an infected area under Sub-section (1) of Rule 42 of the Quarantine Rules, 1935, the Government Veterinary Surgeon, Penang, by virtue of the powers vested in him by Rule 42 (2) of the said Quarantine Rules, hereby orders that until further notice all pigs in Province Wellesley be kept confined in sties or enclosures so constructed that it is impossible for any pig to gain entrance thereto or exit therefrom.

(Sd.) D. P. WHITE,
Government Veterinary Surgeon, Penang.

Penang, 10th November 1936. (No. 622/36).



FIGS. 1 and 2. Multiple hæmorrhages of the skin of various sizes seen after the hair and cuticle were removed by scalding and scraping.

FIG. 4. Purulent conjunctivitis of eyelids swollen and adhering together in a suckling.

FIG. 3. Discharge from the eyes and swelling and adhesion of the eyelids in a piglet.

The disease affected pigs from two days old, right up to eight months old, but rarely adults.

The mortality was high among the young ones (*Vide* Charts A and B).

Incubation Period.

The incubation period varied from three to fourteen days, and most of them exhibited symptoms from the 7th to the 10th day (*Vide* Charts A and B).

Symptoms (Acute and Sub-Acute Forms).

The animals attempted to vomit or even actually vomited, off feed and the temperature varied from 105° F. to 107° F. They were prostrate, unable to move and offered no resistance to handling and were indifferent to everything. On white, or white and black animal there appeared patches of red or purple or even black discolouration of ears, abdomen, inside of the thighs, axillas (Figs. 1 and 2, Plate IX), staggering gait with loss of muscular control and swaying from side to side, did not remain long in one place, the hind legs knuckled over at the fetlocks and crossed one another. In a few cases, the hind legs were paralysed and were dragged along when the pigs moved.

The bowels were at first constipated and then there followed continuous diarrhœa. The fæces were fœtid, either green, brown or yellow and of a sticky nature, later turning watery. The animals attempted to deficate often and strained with pain. The sty emitted a peculiar sour odour. When the lungs were affected the breathing was short, quick and laboured. The skin at first was hot and dry, and as the disease progressed it was cold and clammy. The extremities were cold and discoloured owing to the weakness of the circulation. A short husky cough was often present. There was a catarrhal discharge from the nostrills. Often there was muco-purulent discharge from the eyes which were glued together (Figs. 3 and 4, Plate X). The animals did not answer to the owner's call for food, and if they did, they walked out unsteadily in a dazed and dreamy

manner, staggering back again and dropping down in an exhausted condition. The sick pigs did not lie in one position for a long time. They were restless, constantly raising their fore feet and altering their position from one side to the other, lying close together for warmth. Due to the tenderness of the abdomen, movement caused discomfort and they squealed out with pain. The head was carried low with ears dropped, tail uncurled. The animals wasted rapidly.

Chronic Forms.

In chronic forms, the animals appeared stunted and had an unthrifty look, and were much smaller than the other piglets born at the same time. The skin was of a pale colour and was wrinkled. Often intermittent watery diarrhœa was present. The eye-lids were swollen, and at times everted (Fig. 5, Plate XI).

Course of the Disease and Mortality.

The course of the disease varied. Death took place from the second day of infection, in acute cases, to thirty-four days in chronic cases. Some had a very mild attack and appeared well in five days while chronic cases took as long as seventy-three days to recover. In all, there were 724 pigs affected, 518 died (73·20 per cent) and 194 recovered (26·80 per cent) (*Vide* Table II).

Helminthic Infection.

The writer examined the fæces of several pigs by the Sheather's Sugar Floatation and Centrifuge methods of both healthy and sick pigs, and found almost every pig to be infected with ova of Ascaris lumbricoidis, ova of Ancylostoma, oocyst of the Eimeria debliecki species (large and small forms) and Trichocephalus crenatus. Few showed infection of one kind only, but many showed a mixed infection of two or more of the abovementioned parasites.

The examination of urine by the centrifuge method revealed the presence of ova of Stephanurus dentatus. At the Penang Municipal Abattoirs the writer had seen several livers condemned due to Helminthic infection (larvæ of Ascaris lumbricoidis and

FIG. 5. Purulent conjunctivitis of both eyes in a suckling. Note the rugged appearance of the head and the everted eyelid.

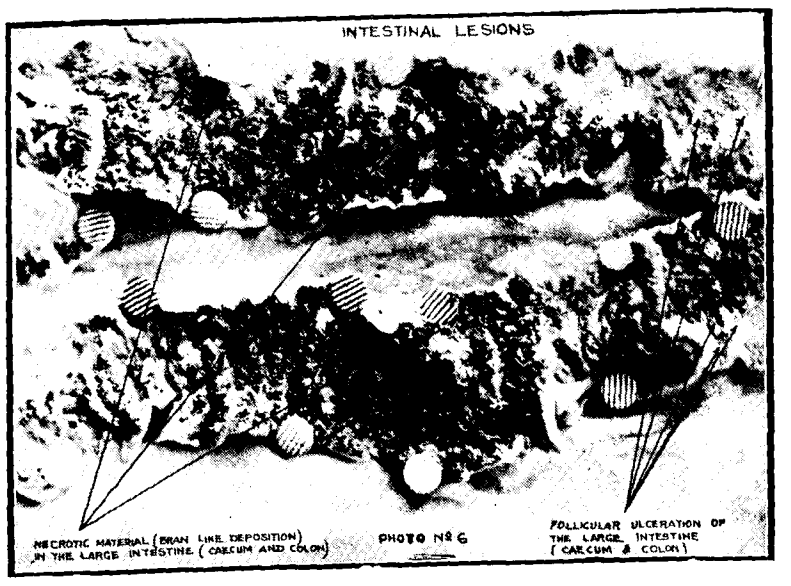


FIG. 6. Intestinal lesions.

On 7th January, 1937, the following materials were collected for culture :—

Urine, Fæces and Blood

On 21st January, 1937, the undermentioned materials were collected for culture :—

Blood, Urine, Bile, Bone marrow and Fæces.

On 10th February, 1937, the following materials were collected for culture :—

Rib-bone marrow, Bile, Urine, Fæces and Blood.

On 30th April, 1937, the following material was collected for culture :—

Urine.

A reply was received each time that the above materials sent were negative for *S. suipestifer* and *S. suisepticus*.

Summary.

Nature and number of specimens sent to the Government Pathologist for culture :—

	Specimens
Fæces ...	13
Urine ...	4
Blood ...	3
Bile ...	2
Bone Marrow ...	2
Total ...	24

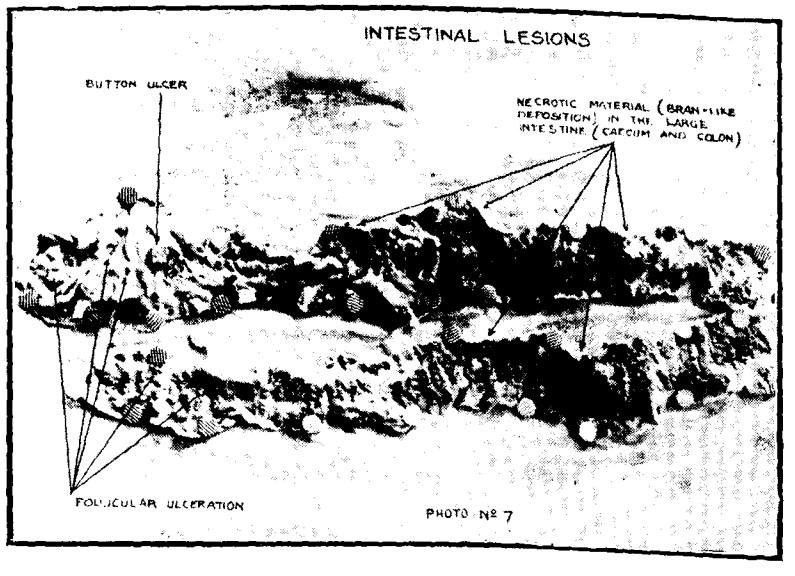
Agglutination Test.

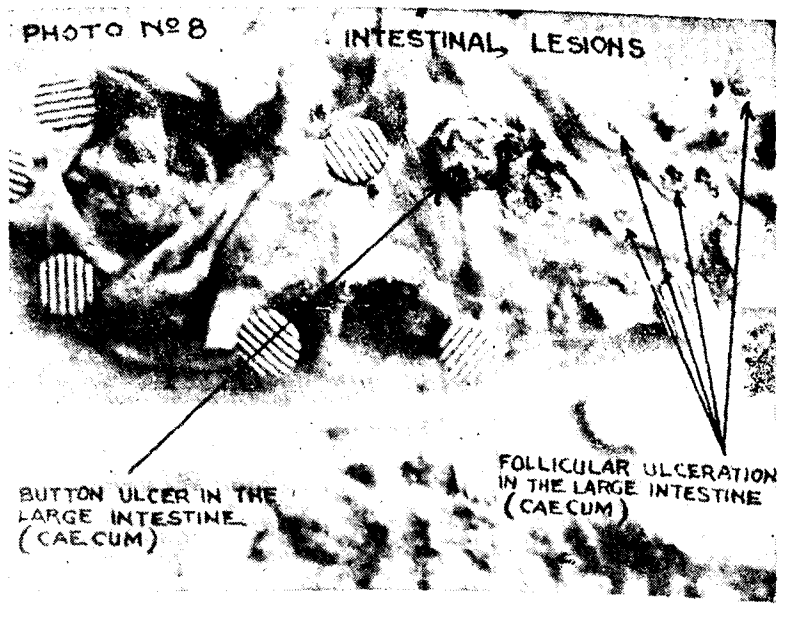
Agglutination Reaction on the blood (taken by cardiac puncture) from a pig aged two months old suspected for Swine Fever was carried out on 6th August 1938, and the Government Pathologist reported as follows :—

FIG. 9. Intestinal lesions.

FIG. 11. Kidney showing pin-point hæmorrhages.

FIG. 12. Hæmorrhages in the Kidney (Micro Photo — $\frac{1}{6}$" dry lens).





FIGS. 7 and 8. Intestinal lesions.

Blood Count.

Blood count on a 22-day-old piglet which had been sick for about 17 days and had a temperature of 106° F. on 24th August 1938. Post-mortem was performed the same day and the internal organs were quite normal.

		Sick Piglets.	Normal.
No. of Erythrocytes per c.m. of blood	...	4,400,000	7,000,000
„ White blood cells	...	10,400	17,000
„ Polymorphonuclear	...	15%	45%
„ Lymphocytes	...	74%	53%
„ Mononuclear	...	11%	4%

Histological Examination of Various Organs in Sections (Stain used Hæmatoxylin and Eosin).

Kidney.—Petechial hæmorrhages in cortex only. Cloudy changes in the tubules. Glomaruli appear quite normal. Degeneration of endothelium with proliferation of cells. Capsule normal (Fig. 12, Plate XIII).

Spleen.—Areas of necrosis and hæmorrhagic and anæmic infarcts. Proliferation of endothelium closing the lumen of vessels (Figs. 13 and 14, Plate XIV).

Sub-Maxillary Lymph Gland.—Hæmorrhages into the parenchyma. Degeneration of the endothelium of the artery. The lymph cells appear normal (Figs. 15 and 16, Plate XIV).

Skin.—Hæmorrhages into the fibrous tissue of the skin (Fig. 17, Plate XV).

Abdominal Lymph Gland.—Areas of degeneration with hæmorrhages and necrosis. Degeneration of blood vessels (Fig. 18, Plate XV).

Inguinal Lymph Gland.—Areas of necrosis and hæmorrhage. Degeneration of blood vessels (Fig. 19, Plate XV).

Vein.—Degeneration of adventisia (outer coat of the vein) and media (elastic coat). Intima shows irregular shedding of the sides (Figs. 20 and 21, Plates XV and XVI).

FIG. 17. Hæmorrhages in the skin (Micro Photo—⅓″ dry lens).

FIG. 18. Hæmorrhages in the abdominal lymph gland (Micro Photo—⅓″ dry lens).

FIG. 19. Hæmorrhages in the inguinal lymph gland (Micro Photo—⅓″ dry lens).

FIG. 20. Vein—degeneration of the adventitia and media. Irregular shedding of the intima (Micro Photo—⅓″ dry lens).

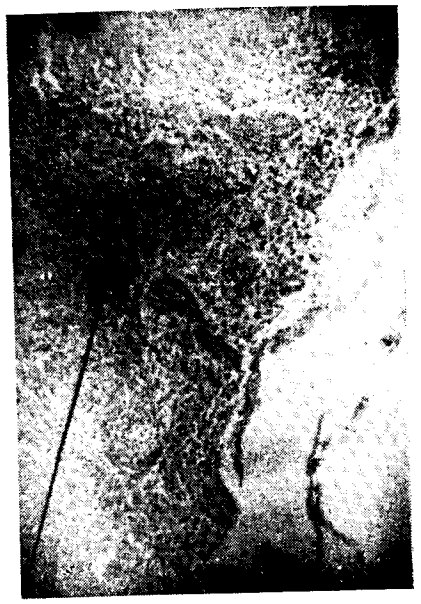


FIG. 13. Hæmorhagic infarcts in the spleen (Micro Photo—⅓" dry lens).

FIG. 14. Anæmic infarcts in the spleen (Micro Photo—⅓" dry lens).

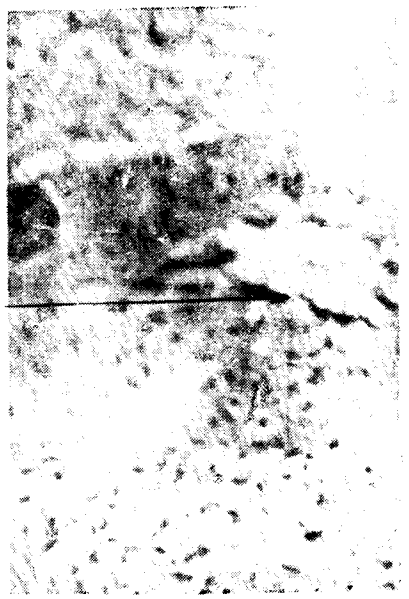


FIG. 15. Sub-maxillary lymph gland—degeneration of the endothelium of the artery (Micro Photo—⅓" dry lens).

FIG. 16. Sub-maxillary lymph gland—degeneration of the endothelium of the artery (Micro Photo—⅓" dry lens).

1. Agglutination Test on a two-month-old piglet:—

	Standard H	Titre O
Bact. typhosum	Neg.	Neg.
„ para A.	Neg.	Neg.
„ „ B. (type)	1/125	Neg.—Significant.
„ „ C. (type)	1/1000	1/50—Significant.
Suipestifer (group)	1/50	Not done—Significant.
Bact. Newport	1/50	Not done—Significant.
„ Ent. Gaerteur	1/125	Not done—Significant.

This titre is seldom met with in uninfected cases.

(Sd.) C. SUBRAHMANYAM,
Government Pathologist, Penang.

This pig was found dead on 15th morning and post-mortem showed presence of button ulcers in cæcum of colon, and pin-point hæmorrhages on kidney.

2. Agglutination Test on a day-old piglet (healthy) is as follows:—

	Standard H	Titre O
Bact. typhosum	1/50	Neg.—Significant.
„ para A.	...	...
„ „ B. (type)	...	...
„ „ C. (type)	...	...
Suipestifer (group)	1/125	Neg.—Significant.
Newport	1/25	Not done—within normal limits.
Bact. Ent. Gaerteur	1/25	Not done—within normal limits.

(Sd.) C. SUBRAHMANYAM,
Government Pathologist, Penang.

Date, 16*th August* 1938.

Saccarose ; Maltose; Mannite; and Litmus milk ; and Tryphoplane broth (for Indol).

The growth in Glucose produced acid and gas;
No acid or gas in Lactose;
Acid and gas in Saccarose;
No acid or gas in Maltose;
No acid and gas in Mannite;

and gave a negative Indol reaction and the organisms were motile.

Agglutinated by *B. proteus* Serum, hence the growth on the Eosin Methylene blue agar plate was that of *Bacillus proteus.*

Abdominal Lymph Gland.

The material from the abdominal lymph gland was plated on Eosin Methylene blue agar plates. Colonies from these plates were put through Glucose, Lactose, Saccarose, Maltose, Mannite, Litmus and Tryphoplane broth, and gave the following reactions:—

Glucose produced acid and gas;
Lactose produced acid and gas;
Saccarose ..... no change;
Maltose produced acid and gas;
Mannite produced acid and gas;
Motility ..... Positive;
Litmus milk produced acid and clot;
Tryphoplane Broth ..... Indol positive.

Therefore the growth was that of *Bacillus coli communis.*

Sub-Maxillary Lymph Gland.

Material from the Sub-maxillary lymph gland was plated on Eosin Methylene blue agar plate. The colonies, which grew when planted in Glucose, Lactose, Saccarose, Maltose, Mannite, Litmus milk and Tryphoplane Broth, produced the following results:—

Glucose produced no change;
Saccarose produced no change;
Maltose produced no change;

Mannite produced no change;
Litmus milk produced alkaline;
Tryphoplane Broth produced no Indol;
Motility . . . . . Positive.

Hence the growth was found to be that of *Bacillus alkaligenes.*

Differential Diagnosis.

From East African Swine Fever.—In East African outbreaks of Swine Fever there are very few recoveries; but in ordinary swine fever there is a certain higher percentage of recovery depending upon the number of outbreaks the herd has undergone.

From Ordinary Swine Fever.—When susceptible pigs are experimentally inoculated with infected material, about 50 per cent take the infection. The writer failed to infect nine pigs, five guinea pigs, two white rabbits, three white rats and a white mouse.

From B. Suipestifer Infection.—This bacillus is said to be ordinarily found in pigs, and it is agreed by several writers that, when the vitality of tne animal is lowered by swine fever virus, the B. suipestifer takes the upper hand and causes death.

The writer found rod-shaped gram-negative bacillus in the ulcers, lymphatic, abdominal, inguinal and sub-maxillary lymph glands from pigs slaughtered at the Penang Municipal Abattoirs. When these were collected in broth and plated in agar eosin-methylene blue, they were found to be *B. proteus* (scrapings from ulcers), B. *coli communis* (abdominal gland) and *B. alkaligenes* (sub-maxillary gland).

From Necrotic Enteritis of Pig.—By the absence of eye lesions and button ulcers.

From Swine Erysipelas.—By the absence of growth of rod-shaped bacterium in blood cultures, and the presence of mortality among young ones especially.

FIG. 21. Vein—degeneration of the adventitia and media. Irregular shedding of the intima (Micro Photo—⅙″ dry lens).

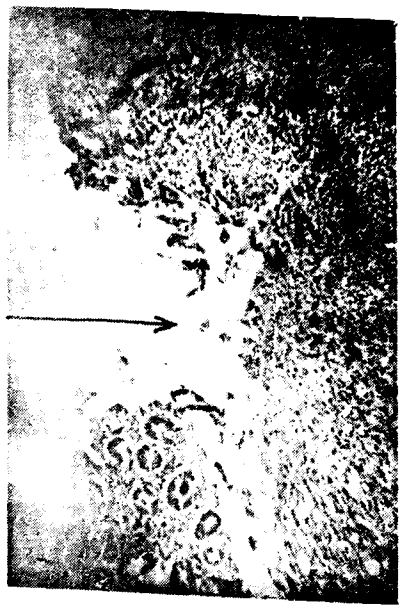


FIG. 22. Ulcer in the cæcum (Micro Photo—⅙″ dry lens).

FIG. 23. Histiocytes (Epitheloid cells) in cæcal ulcer (Micro Photo—1/12″ oil immersion).

FIG. 24. Heart—hæmorrhages in the epi- and pericardium (Micro Photo—⅙″ dry lens).

Artery.—Degeneration of the middle muscular coat. Irregular shedding of the intima.

Intestine (*Cœcum*).—There was infiltration of epitheloid cells —histiocytes in sub-mucous tissue and thrombosis of small vessel. Degeneration of arteries. Degeneration of the mucosa (Figs. 22 and 23, Plate XVI).

Heart.—Numerous hæmorrhages were seen in the endocardium and pericardium. Heart muscle showed cloudy changes (Fig. 24, Plate XVI).

Brain.—The lesions consisted of perivascular infiltration with few lymphocytes, polynuclear leucocytes and fair number of histiocytes. No definite evidence of proliferation of endothelium was detected ; but the adventitia showed some evidence of proliferation. In some areas few R. B. C. were seen in perivascular spaces.

The above descriptions are similar to those found in septicæmic conditions.

Microscopical Examination.

The examination of caseous materials from the lymph glands, immediately after slaughter, revealed Streptococci and Staphylococci, but were negative for Tubercule bacilli.

Scrapings taken from the conjunctiva of one piglet and stained with Alkaline Methylene blue were positive for lanceolate gram-positive diplo-cocci. Smears taken from the glands and intestinal ulcers of fresh carcases revealed rod-shaped gram-negative organisms which could be either *Bacilli colli*, *Proteus vulgaris*, or B. suipestifer. Cultures failed to reveal the presence of B. suipestifer.

Cultural Tests.

Scrapings from the ulcers in cæcum and colon were collected in broth and planted in Eosin Methylene blue agar. The colonies that grew on this were put through sugars: Glucose; Lactose;

5. Fæcal examination revealed plenty of ova of Ascaris, Ankylostoma, Trichocephalus of oocysts Eimeria debliecki (large and small forms), ova of Stephanurus dentatus were demonstrated in specimens of centrifuged urine.

6. Agglutination tests on two piglets (two-month-old and a day-old) gave significant reactions to organisims of Salmonella group.

7. The disease among pigs met with in Province Wellesley may have been caused by dietetic errors coupled with heavy helmenthic infestation. The heavy mortality in young sucklings may be due to the latter cause.

Acknowledgment.

1. I am greately indebted to Captain D. P. White, M.R.C.V.S., Veterinary Officer, Penang, for correcting and permitting me to publish this paper.

2. I am also indebted to the Government Pathologist, Penang, and his staff for the sections, cultures, agglutination tests and other assistance rendered while investigating into this disease.

3. I am indebted to Mr. A. G. McCrea, M.R.C.V.S., D.V.S.M., Municipal Veterinary Surgeon, Penang, for valuable suggestions, and to S. T. Paul, G.B.V.C., Abattoir Superintendent, Penang, for assistance rendered at the Abattoirs, when collecting materials from the carcases condemned for Swine Fever, while investigating into this disease.

4. I acknowledge with pleasure the assistance rendered by Mr. Yeoh Hoe Peng, Chinese Live-stock Inspector, Penang.

5. I am also indebted to Mr. T. D. Money of the P. W. D., Singapore, for the Charts.

REFERENCES.

—— Editorial.—"Swine Fever and Paratyphoid in Pigs," *Journal of Comparative Pathology and Therapeutics*, December 1933, p. 257.

Waldmann, O.— *The Diagnosis of Swine Fever.*

Gaiger and Davies.—"Swine Fever," *Veterinary Pathology and Bacteriology*, Second Edition.

Hutyra and Marek.—"Hog Cholera," *Special Pathology and Therapeutics of the Diseases of Domestic Animals*, Second Edition.

Leighton, G., O.B.E., M.D., M.D., D.Sc., F.R.S.E.—"Swine Fever," *The Principles and Practice of Meat Inspection.*

Doyle, T. M.—"The Viability of the Virus Swine Fever in Bone Marrow, Muscle and Skin of Preserved Carcases," *Journal of Comparative Pathology and Therapeutics*, **46**, Part I, March 1933.

Savalagic, P.—"Histological Diagnosis of Swine Fever," *The Veterinary Record*, **50**, No. 25, June 18, 1938.

Beckett, F., F.R.C.V.S.—"Necrotic Enteritis of Pig," *The Veterinary Journal*, **91**, No. 6, June 1935.

Topley and Wilson.—*The Principles of Bacteriology and Immunity*, **1**.

TABLE I.

Abattoir	Year	No. of pigs slaughtered	No. of cases of swine fever
Ipoh ...	1930	72,327	12
	1931	69,689	11
	1932	59,291	Figures not available
	1933	51,552	22
	1934	55,035	6
	1935	59,557	9
	1936	57,680	33
	1937	65,449	21
Penang ...	1931	107,492	8
	1932	112,427	72
	1933	99,858	477
	1934	90,498	54
	1935	98,307	131
	1936	95,268	212
	1937	100,780	172
Singapore ...	January 1936	16,539	46
	February ,,	13,377	39
	March ,,	15,470	63
	April ,,	13,826	63
	May ,,	12,635	40
	June ,,	13,299	43
	July ,,	11,346	22
	August ,,	12,309	32
	September ,,	10,994	20
	October ,,	8,948	29
	November ,,	9,975	12
	December ,,	12,105	12
	Total for 1936 ...	150,823	421
	1937	269,819	202

Discussion.

There is a strong belief that swine fever has been in existence in Malaya for a very long time, and the mortality was so low that the Veterinary Department was unable to detect the place of infection. It is probable that at last the mortality flared up, and the owners could not hide cases any longer; and the officers concerned were able to trace the infected areas. The pig rearers never took us into confidence, and hence did not report when their animals were affected and would not co-operate with us.

Prior to detection of this outbreak, cases of the so-called Swine Fever were detected at the Abattoirs only. We could not trace the infected area, and the brokers would not assist us in finding out the places from where the infected pigs had come.

In this outbreak, it was noticed that, as is the case with swine fever, the deaths had been mainly among the sucklings although adult pigs were also affected, but to a much less degree. One could safely say that the disease would come to an end in that locality, when all the young ones were either dead or had recovered. The recovery varied from 17·70 per cent to 60·00 per cent.

According to Hutyra and Marek, the sucklings do not get affected until they are weaned if the sow was one that had recovered from a previous attack. As deaths were mainly among the sucklings, the only conclusion the writer could come to, is that the sow was not affected previously; or the disease under review was not swine fever.

Several countries are seriously thinking of not confirming swine fever on the findings of "button" ulcers only, as it is universally accepted that the infection of B. suipestifer and S. septica are secondary invasions, and the latter produce their lesions when the vitality of the animal is lowered by the invasion of the swine fever virus.

Since animal inoculation with materials from the various organs of the affected pigs failed to produce either Swine Fever or Paratyphoid of pigs, and cultures and sugar tests failed to reveal

the presence of B. suipestifer, the disease under review could not be either Swine Fever or Paratyphoid of pigs.

The histological examination findings confirm the findings of Waldmann.

The histological examination findings are those of septicæmia. It is a well-known fact that the virus of Swine Fever produces Septicæmia; but what is there to differentiate between Septicæmia caused by Swine Fever from that of ordinary Septicæmia caused by very bad sanitation, septic wounds caused by castration and spaying, etc.?

The disease among pigs met with in Province Wellesley may have been caused by dietitic errors coupled with heavy helmenthic invasion. The pigs were reared in extremely insanitary conditions.

As the writer experimented on a few animals only, further extensive research is necessary with a view to diagnosing the cause of the heavy mortality in pigs, mainly in young ones, in Province Wellesley.

Conclusion.

1. The diagnosis was mainly based on clinical symptoms, post-mortem leisions, the age of animals affected, mortality, cultural and biological and histological findings.

2. The writer failed to transmit Swine Fever virus or the B. suipestifer to nine young pigs, five guinea pigs, two white rabbits, three white rats and one white mouse, by inoculating with the blood, urine, etc., from the affected pigs, hence the disease under investigation, could not be Swine Fever.

3. Heart blood, bone marrow, bile, urine, fæces taken from affected pigs at different stages in liquid media (30 per cent glycerine in normal saline), sent to the Government Pathologist in Penang under ærobic and anærobic conditions for culture did not yield any growth of B. suipestifer.

4. Blood smears taken from several animals were negative for micro-organisms. Gland Smears made from several cases revealed streptococci and staphylococci and gram-negative bacilli.

Hutyra and Marek.—"Hog Cholera," *Special Pathology and Therapeutics of the Diseases of Domestic Animals*, Second Edition.

Leighton, G., O.B.E., M.D., M.D., D.Sc., F.R.S.E.—"Swine Fever," *The Principles and Practice of Meat Inspection.*

Doyle, T. M.—"The Viability of the Virus Swine Fever in Bone Marrow, Muscle and Skin of Preserved Carcases," *Journal of Comparative Pathology and Therapeutics*, **46**, Part I, March 1933.

Savalagic, P.—"Histological Diagnosis of Swine Fever," *The Veterinary Record*, **50**, No. 25, June 18, 1938.

Beckett, F., F.R.C.V.S.—"Necrotic Enteritis of Pig," *The Veterinary Journal*, **91**, No. 6, June 1935.

Topley and Wilson.—*The Principles of Bacteriology and Immunity*, **1**.

TABLE I.

Abattoir	Year	No. of pigs slaughtered	No. of cases of swine fever
Ipoh ...	1930	72,327	12
	1931	69,689	11
	1932	59,291	Figures not available
	1933	51,552	22
	1934	55,035	6
	1935	59,557	9
	1936	57,680	33
	1937	65,449	21
Penang ...	1931	107,492	8
	1932	112,427	72
	1933	99,858	477
	1934	90,498	54
	1935	98,307	131
	1936	95,268	212
	1937	100,780	172
Singapore ...	January 1936	16,539	46
	February ,,	13,377	39
	March ,,	15,470	63
	April ,,	13,826	63
	May ,,	12,635	40
	June ,,	13,299	43
	July ,,	11,346	22
	August ,,	12,309	32
	September ,,	10,994	20
	October ,,	8,948	29
	November ,,	9,975	12
	December ,,	12,105	12
	Total for 1936 ...	150,823	421
	1937	269,819	202

TABLE

The Number of Pigs affected with the

No.	LOCALITIES.	No. of pigs before the outbreak.	No. of pigs born during the quarantine.	Total No. of pigs.	Total No. of healthy pigs sold for meat.	Total No. of pigs left at close of outbreak.	Total No. of healthy pigs left at close of outbreak.	Total No. of pig sties.	No. of pig sties affected.	No. of pigs affected.
1.	Permatang To' Mahat	308	138	446	282	164	123	22	17	232
2.	Kampong Bharu...	627	194	821	148	673	646	52	4	56
3.	Sungei Bakap ...	450	174	624	377	247	174	68	45	323
4.	Tanah Liat ...	271	67	338	31	307	302	14	3	21
5.	Permatang Pauh ...	397	44	441	32	409	392	12	6	32
6.	Kampong Simpah	368	98	466	12	454	436	32	4	30
7.	Telaga Ayer ...	33	31	64	20	44	31	4	3	30
	TOTAL ...	2454	746	3200	902	2298	2104	204	82	724

NO. II.

Disease in Province Wellesley, 1936–37.

No. of sick pigs destroyed for experiment.	Mortality.	Mortality including destroyed (percentage).	Recovery	Recovery (percentage).	Duration of the disease.	REMARKS.
...	191	82·30	41	17·70	About 3 months from 6-11-1936 to 27-2-1937.	The disease was detected early in the outbreak.
...	29	51·79	27	48·21	3 months from 13-11-1936 to 10-2-1937.	The disease was detected after 7 months, existence. Hence the low mortality rate.
4	246	76·16	73	22·60	4 months from 9-12-1936 to 16-4-1937.	The disease was detected early in the outbreak.
...	16	76·19	5	23·81	About 2 months from 8-2-1937 to 2-4-1937.	The disease was detected 2 months after the outbreak.
2	13	40·60	17	53·10	One month from 9-3-1937 to 14-4-1937.	The disease was discovered towards end of the outbreak hence the low death rate. The disease was reported to have first broken out on 11-2-1937.
3	9	30·00	18	60·00	About 2 months from 19-4-1937 to 18-6-1937.	The disease was detected late in the outbreak.
3	14	46·67	13	43·33	About 2 months from 12-8-1937 to 1-10-1937.	The disease was detected late in the outbreak.
12	518	71·55%	194	26·79%	6-11-1936 to 1-10-1937.	

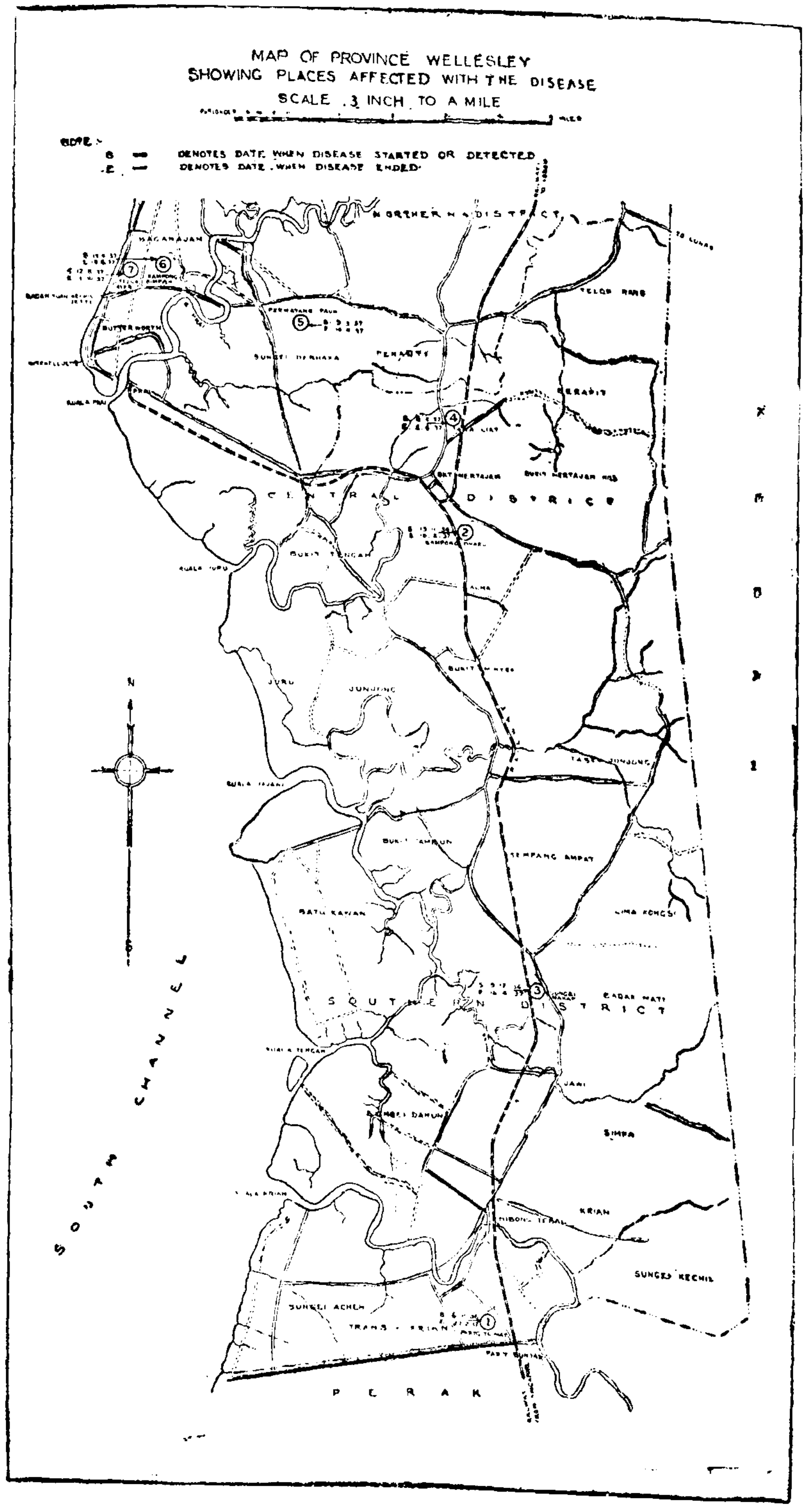

MAP OF PROVINCE WELLESLEY
SHOWING PLACES AFFECTED WITH THE DISEASE
SCALE .3 INCH TO A MILE
NORTHERN DISTRICT
CENTRAL DISTRICT
SOUTHERN DISTRICT
SOUTH CHANNEL
PERAK

CHART A.

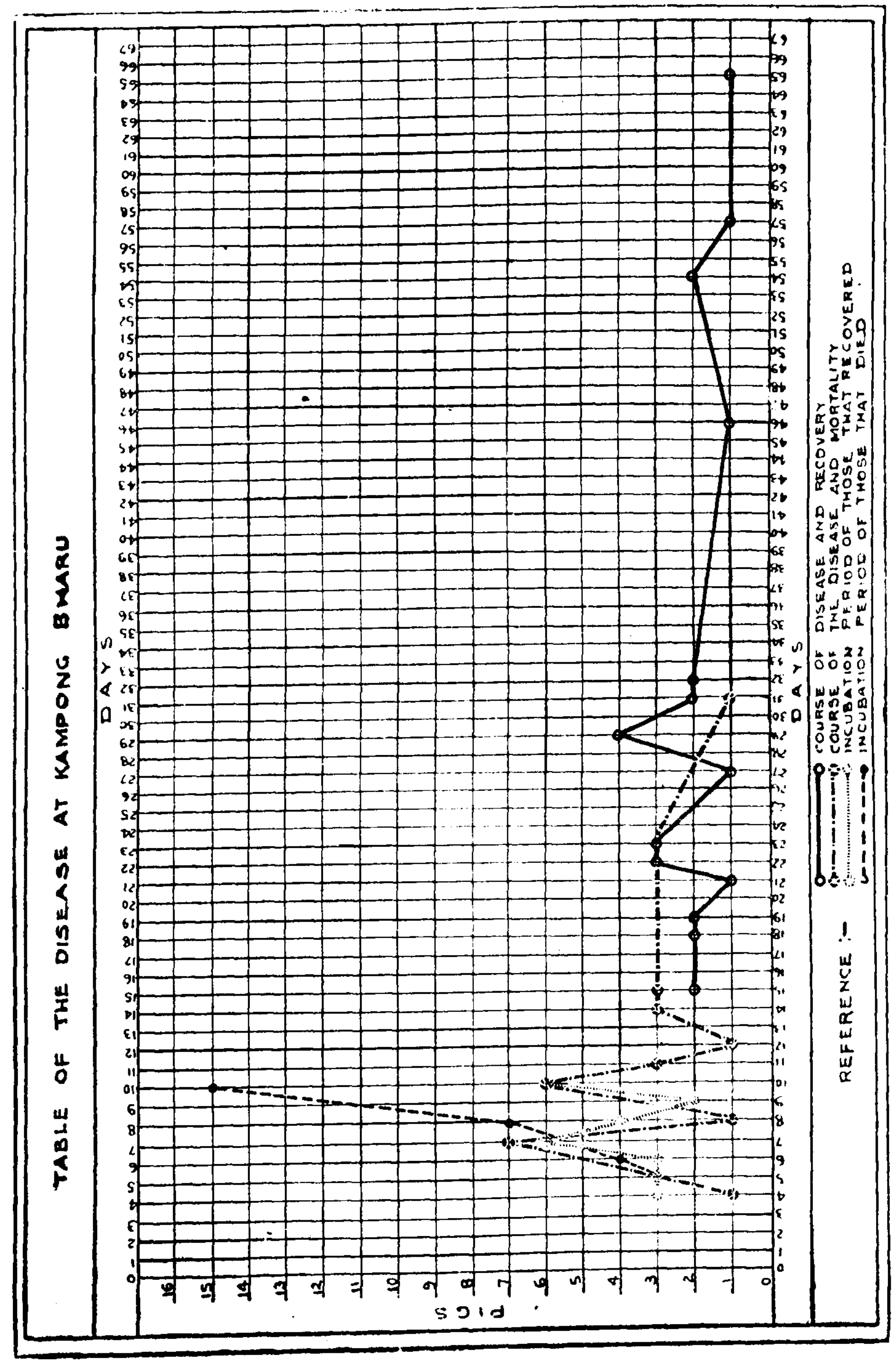


TABLE OF THE DISEASE AT KAMPONG BHARU
DAYS
PIGS
REFERENCE :-
COURSE OF DISEASE AND RECOVERY
COURSE OF THE DISEASE AND MORTALITY
INCUBATION PERIOD OF THOSE THAT RECOVERED
INCUBATION PERIOD OF THOSE THAT DIED

TABLE III.

Swine Fever Experimental Animal Inoculation.

No.	Date.	Animal inoculated.	Donor.	Donor's age in months.	Material used.	Result.	Remarks.
1.	22-3-1937	Guinea pig No. 1	Sick pig	1½	1 c.c. heart blood	Negative	Inoculated intra-peritoneally (*Vide* Chart No. 1).
2.	do.	White rat No. 1	do.	1½	do.	do.	do. (*Vide* Chart No 2).
3.	do.	White rabbit No. 1	do.	1½	do.	do.	do. (*Vide* Chart No. 3).
4.	do.	Guinea pig No. 2	do.	2½	do.	do.	do. The Guinea pig died of pneumonia. The stomach was slightly inflamed. The lungs showed sign of pneumonia and the intestines were normal. Materials inoculated into two G. pigs and a white rat (*Vide* Chart No. 4).
5.	do.	Guinea pig No. 3	do.	2½	1 c.c. urine	do.	Inoculated intra-peritoneally (*Vide* Chart No. 5).
6.	do.	White rat No. 2	do.	2½	1 c.c. heart blood	do.	do. (*Vide* Chart No. 6).
7.	do.	White rabbit No. 2	do.	2½	2 c.cs. do.	do.	do. (*Vide* Chart No. 7).
8.	do.	Pig No. 1	do.	2½	do.	do.	do. (*Vide* Chart No. 8).
9.	20-4-1937	White Mouse	do.	1	1 c.c. heart from tail	do.	do. (*Vide* Chart No. 9).
10.	21-5-1937	Guinea pig No. 4	G. pig No. 4		1 c.c. urine	do.	do. (*Vide* Chart No. 10).
11.	do.	Guinea pig No. 5	do.		1 c.c. heart blood	do.	do. (*Vide* Chart No. 11).
12.	do.	White rat No. 3	do.		1 c.c. urine	do.	do. (*Vide* Chart No. 12).
13.	12-8-1937	Pig No. 2	Sick pig	3	5 c.cs. mixed liver, kidney, heart muscle, spleen and bone marrow.	do.	do. The materials were ground up in fine sterile sand in normal saline. The experimental pig was slaughtered on 15-11-1937. Two small "button" ulcers found in cæcum. (*Vide* Chart No. 13).
14.	do.	Pig No. 3	do.	3	do.	do.	do. The materials treated as above. The experimental pig was also slaughtered on 15-11-1937. "Follicular ulceration" found in cæcum (*Vide* Chart No. 14).
15.	19-8-1937	Pig No. 4	do.	4	10 c.cs. spleen	do.	do. The spleen was similarly prepared (*Vide* Chart No. 15).
16.	do.	Pig No. 5	do.	4	10 c.cs. kidney	do.	do. The kidney was prepared as above (*Vide* Chart No. 16).
17.	do.	Pig No. 6	do.	4	10 c.cs. liver	do.	do. The liver was similarly prepared (*Vide* Chart No. 17).
18.	do.	Pig No. 7	do.	4	10 c.cs. urine	do.	do. (*Vide* Chart No. 18).
19.	26-8-1937	Pig No. 8	do.	1	10 c.cs. heart	do.	do. Heart muscle ground up in fine sterile sand in normal saline (*Vide* Chart No. 19).
20.	do.	Pig No. 9	do.	1	10 c.cs. bone marrow.	do.	do. Prepared as above. (*Vide* Chart No. 20)

CHART B.

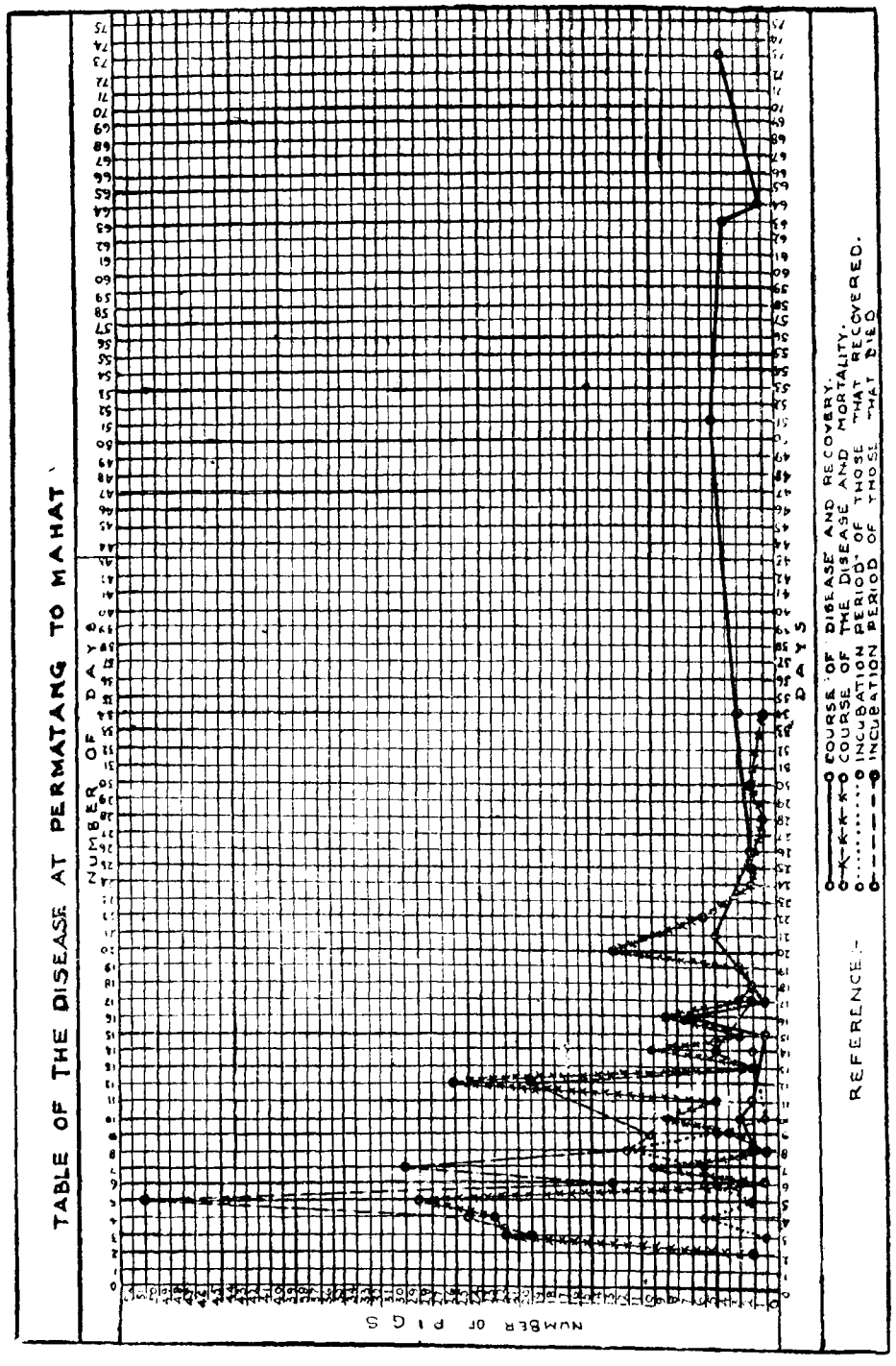
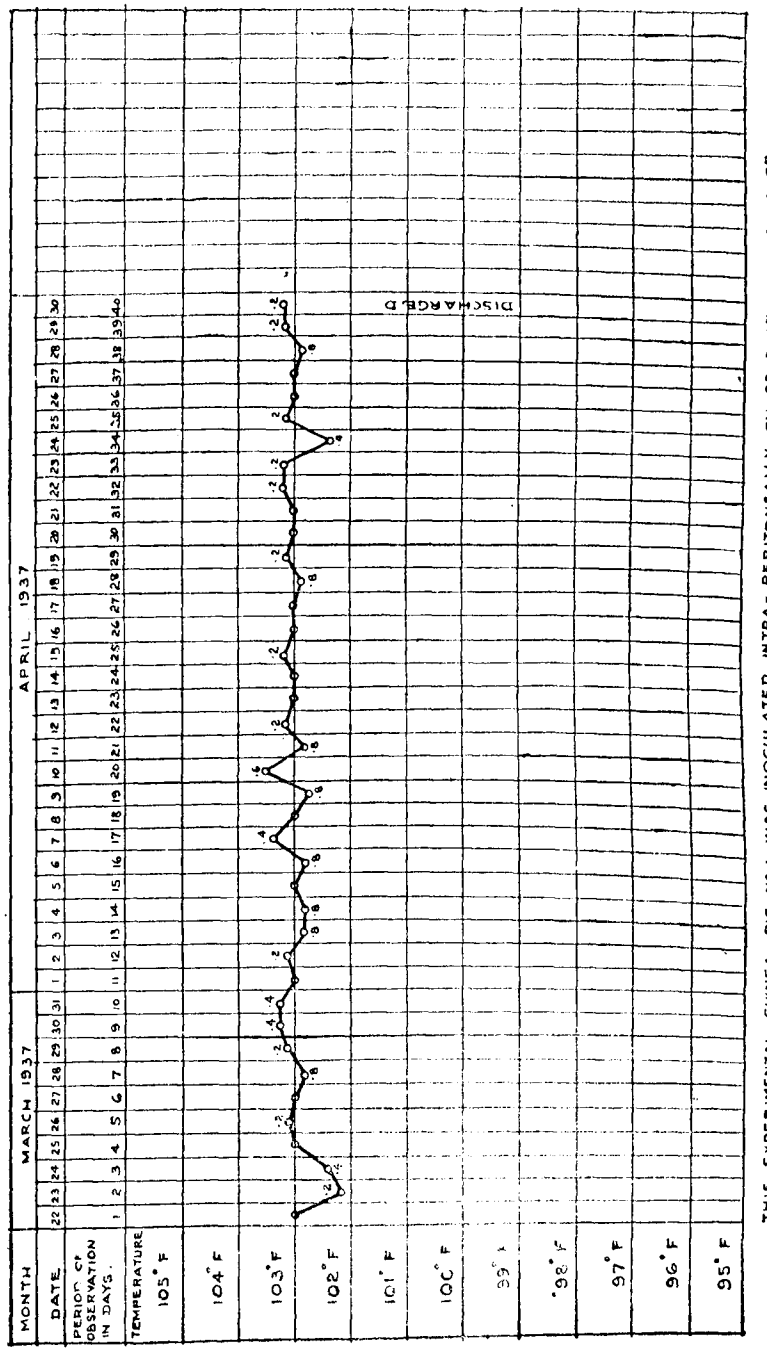


CHART Nº 1

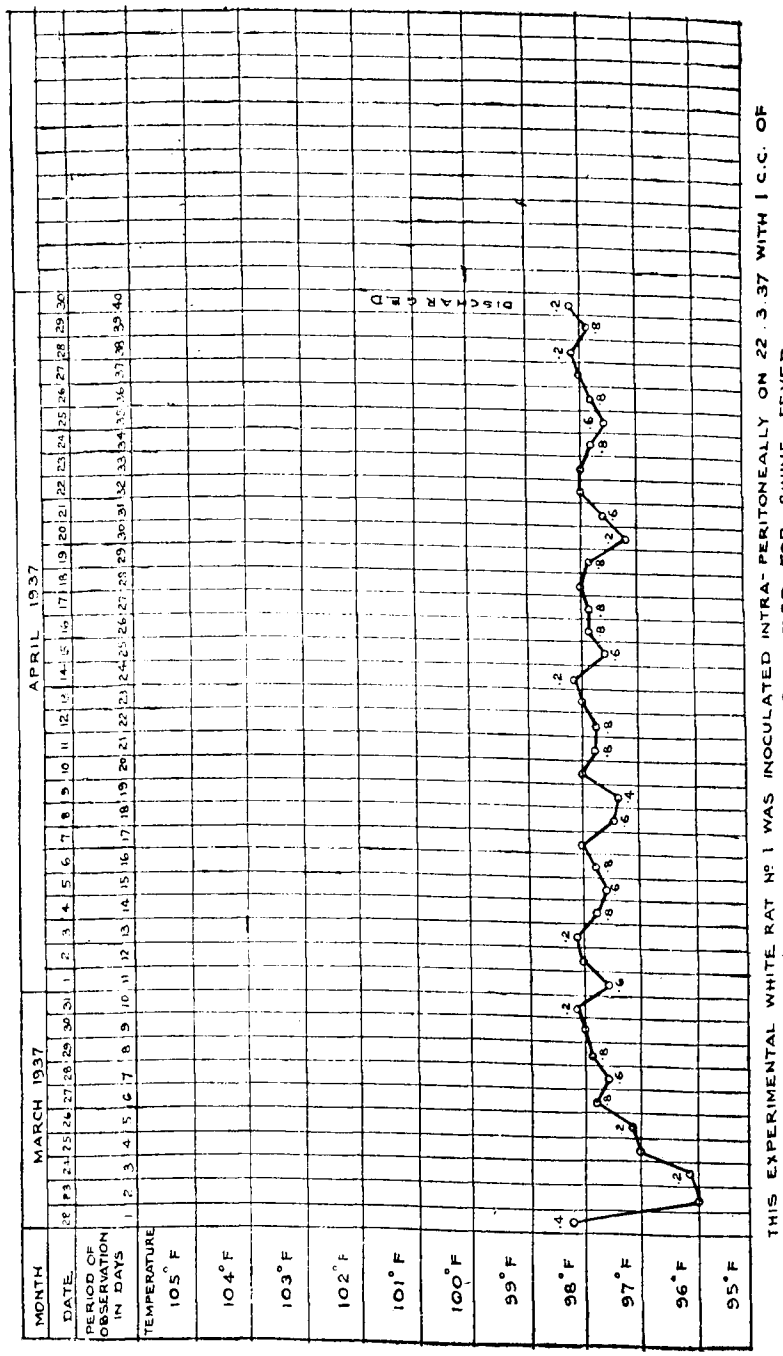
SWINE FEVER INOCULATION
EXPERIMENTAL GUINEA-PIG Nº 1



THIS EXPERIMENTAL GUINEA-PIG Nº 1 WAS INOCULATED INTRA-PERITONEALLY ON 22.3.37 WITH 1 C.C. OF HEART BLOOD FROM A 1½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER

CHART Nº 2

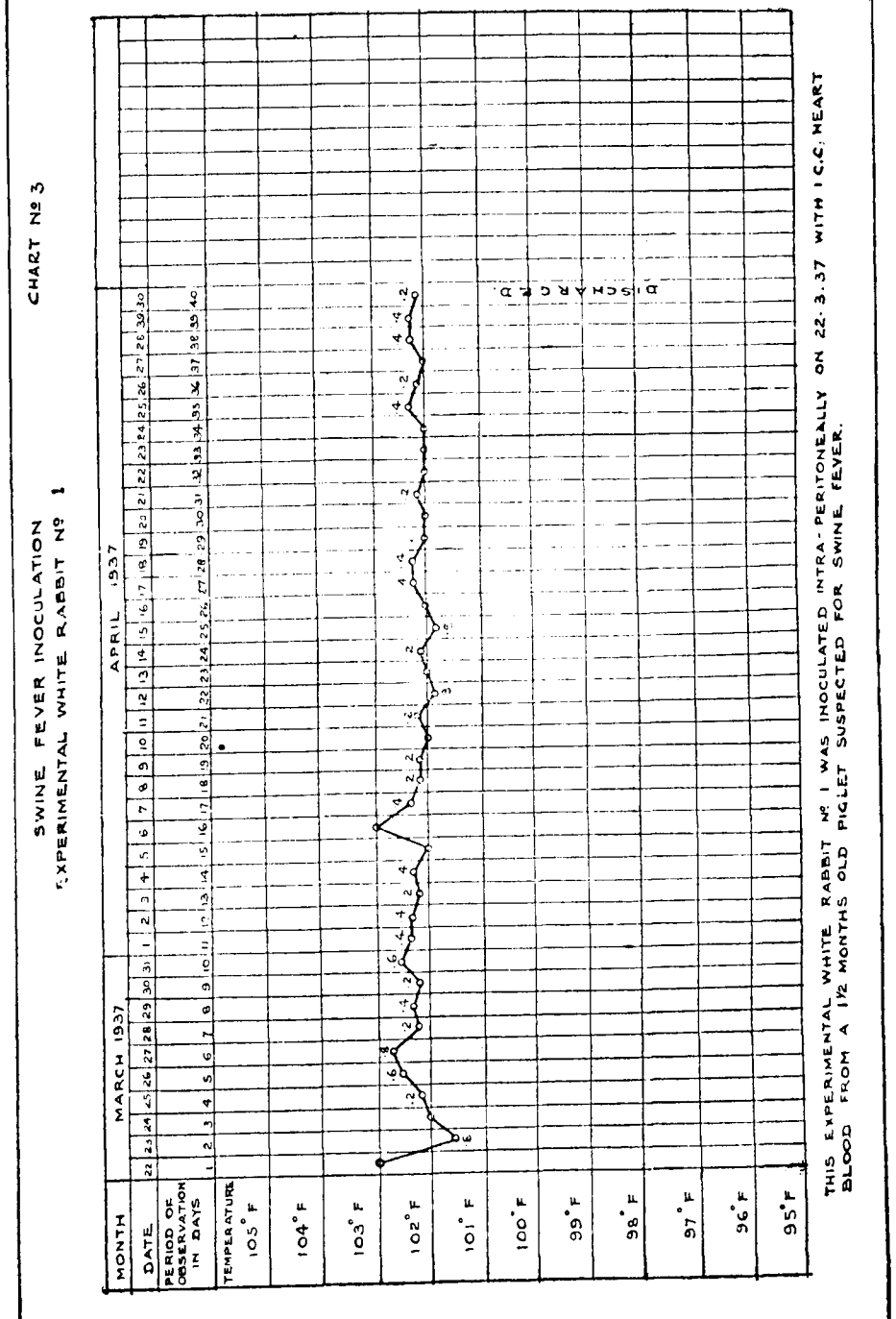
SWINE FEVER INOCULATION
EXPERIMENTAL WHITE RAT Nº 1



THIS EXPERIMENTAL WHITE RAT Nº 1 WAS INOCULATED INTRA-PERITONEALLY ON 22.3.37 WITH 1 C.C. OF HEART BLOOD FROM A 1½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER.

CHART Nº 3

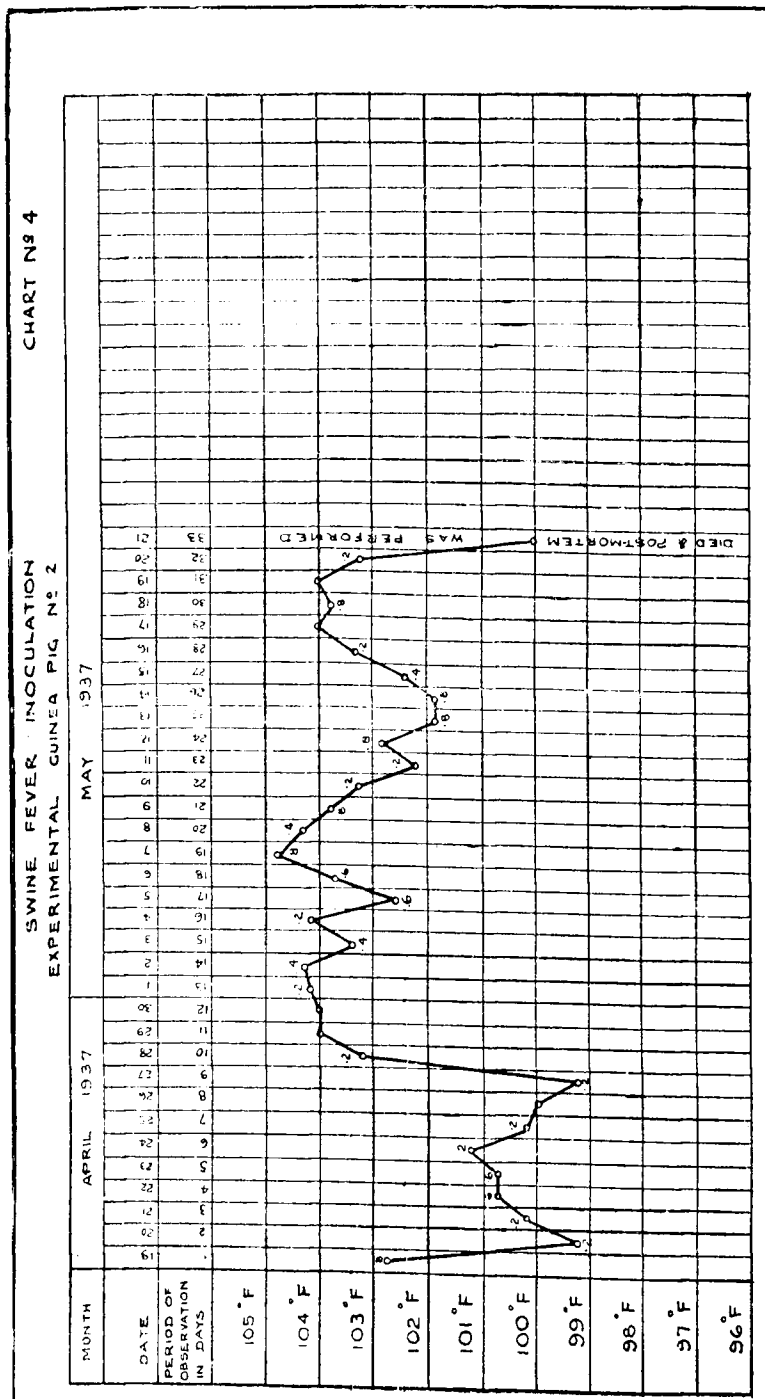
SWINE FEVER INOCULATION
EXPERIMENTAL WHITE RABBIT Nº 1



THIS EXPERIMENTAL WHITE RABBIT Nº 1 WAS INOCULATED INTRA-PERITONEALLY ON 22.3.37 WITH 1 C.C. HEART BLOOD FROM A 1½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER.

CHART Nº 4

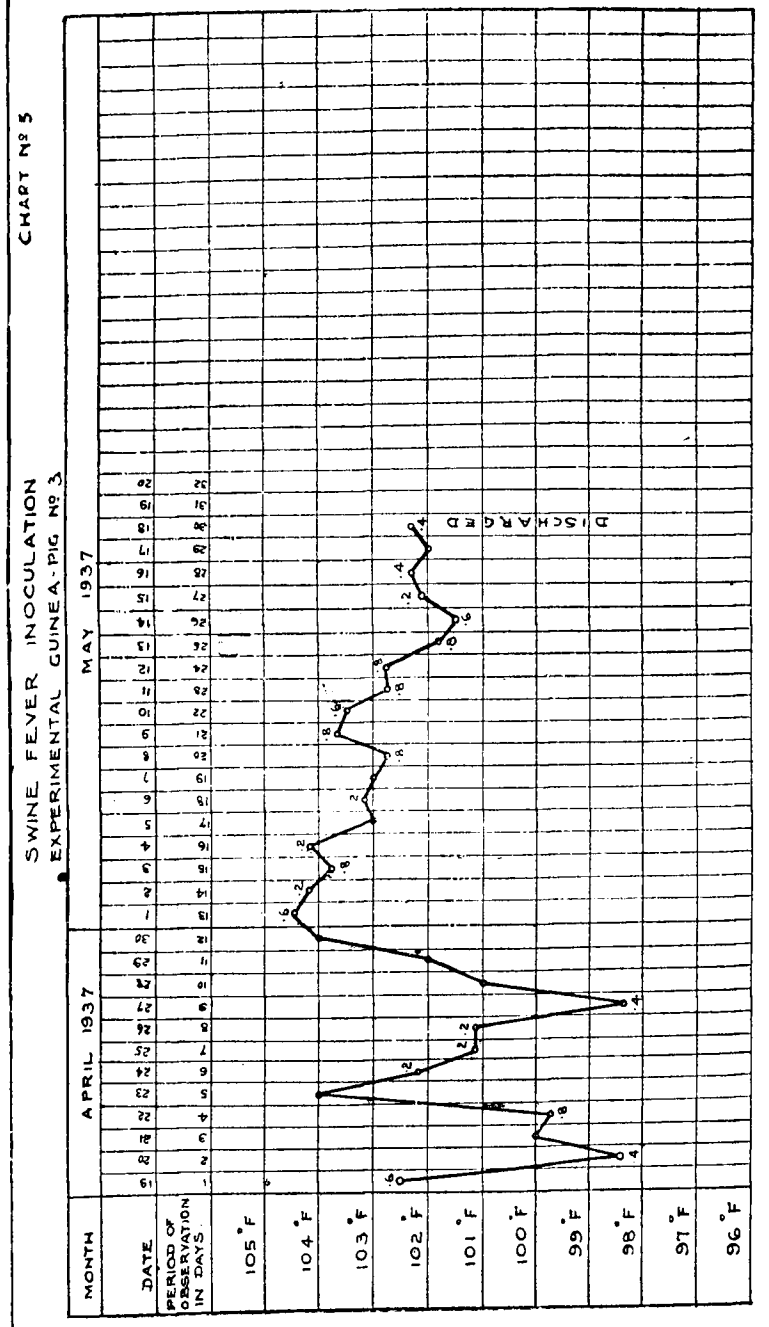
SWINE FEVER INOCULATION
EXPERIMENTAL GUINEA PIG Nº 2



THIS EXPERIMENTAL GUINEA-PIG Nº 2 WAS INOCULATED INTRA-PERITONEALLY ON 19.4.37 WITH 1 C.C. OF HEART BLOOD FROM A 2½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER. THE GUINEA PIG DIED ON 21.5.37 WHEN POST-MORTEM WAS CARRIED OUT. THE STOMACH WAS SLIGHTLY INFLAMED. THE LUNGS SHOWED SIGN OF PNEUMONIA AND THE INTESTINES WERE NORMAL. 1 C.C. OF ITS URINE WAS INOCULATED INTRA-PERITONEALLY INTO GUINEA PIG Nº 4. ANOTHER 1 C.C. OF URINE WAS SIMILARLY INJECTED INTO A WHITE RAT, GUINEA PIG Nº 5 WAS INOCULATED INTRA-PERITONCALLY WITH 1 C.C. OF HEART BLOOD

CHART Nº 5

SWINE FEVER INOCULATION
EXPERIMENTAL GUINEA-PIG Nº 3



THIS GUINEA-PIG WAS INOCULATED INTRA-PERITONEALLY ON 19.4.37 WITH 1 C.C. OF URINE FROM A 2½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER

CHART No 6

SWINE FEVER INOCULATION
EXPERIMENTAL WHITE RAT No 2
BATCH 'A'

MONTH	APRIL 1937																									MAY										
DATE	19	20	21	22	23	24	25	26	27	28	29	30	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
PERIOD OF OBSERVATION IN DAYS	-	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36
104 °F																																				
103 °F																																				
102 °F																																				
101 °F																																				
100 °F																																				
99 °F																																				
98 °F																																				
97 °F																																				
96 °F																																				
95 °F																																				

THIS EXPERIMENTAL WHITE RAT No 2 WAS INOCULATED INTRA-PERITONEALLY ON 19. 4. 37 WITH 1 c.c
OF HEART BLOOD FROM A 2½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER.

CHART No 7

SWINE FEVER INOCULATION
EXPERIMENTAL WHITE RABBIT No 2

MONTH	APRIL 1937																									MAY										
DATE	19	20	21	22	23	24	25	26	27	28	29	30	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
PERIOD OF OBSERVATION IN DAYS	-	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36
105 °F																																				
104 °F																																				
103 °F																																				
102 °F																																				
101 °F																																				
100 °F																																				
99 °F																																				
98 °F																																				
97 °F																																				
96 °F																																				

THIS EXPERIMENTAL RABBIT WAS INOCULATED INTRA-PERITONEALLY ON 19. 4. 37 WITH 2 c.cs. OF HEART
BLOOD FROM A 2½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER.

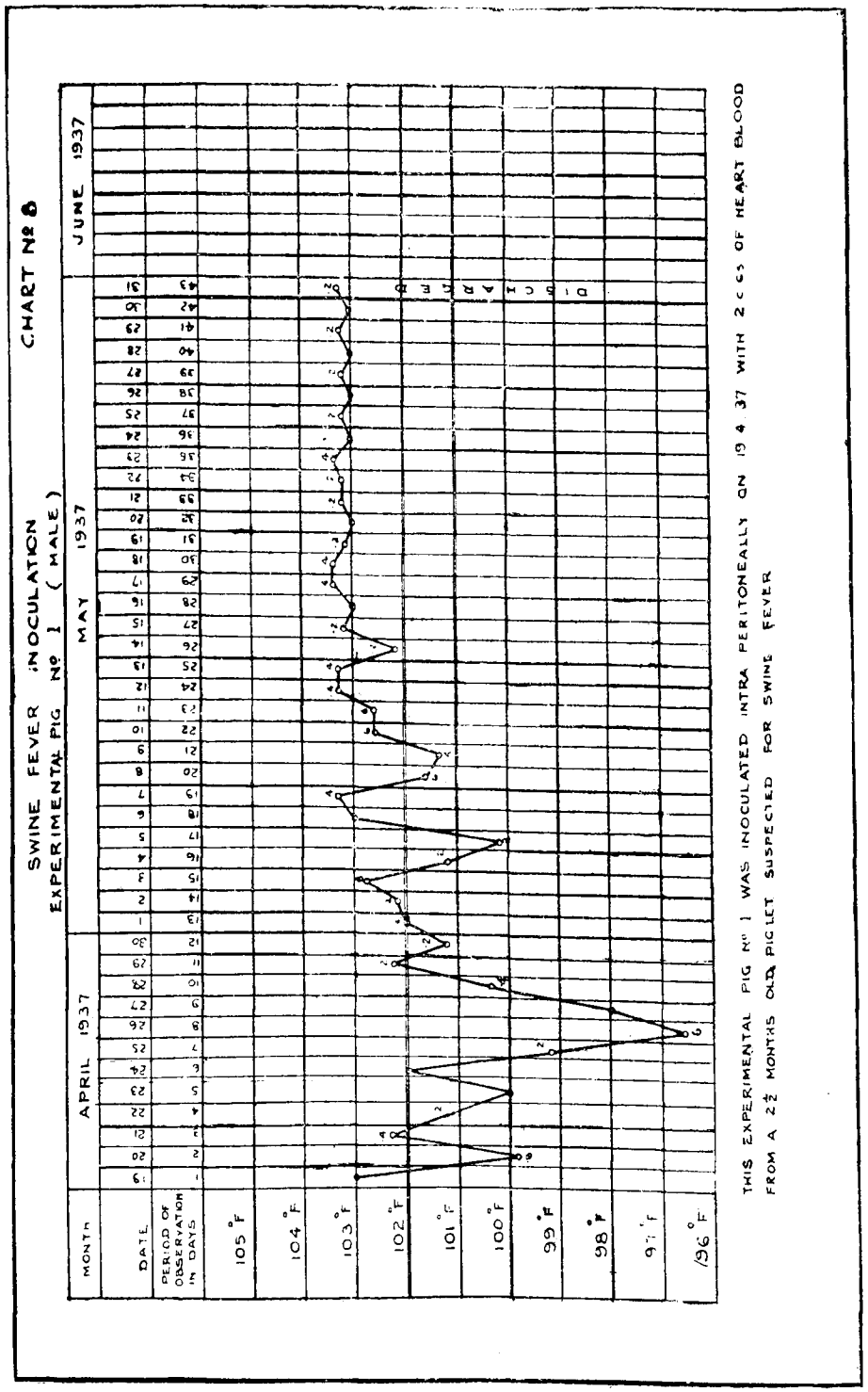


CHART Nº 8
SWINE FEVER INOCULATION
EXPERIMENTAL PIG Nº 1 (MALE)
APRIL 1937
MAY
JUNE 1937
MONTH
DATE
PERIOD OF OBSERVATION IN DAYS
105 °F
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
96 °F
DISCHARGE
THIS EXPERIMENTAL PIG Nº 1 WAS INOCULATED INTRA PERITONEALLY ON 19 4 37 WITH 2 c c s OF HEART BLOOD
FROM A 2½ MONTHS OLD PIGLET SUSPECTED FOR SWINE FEVER

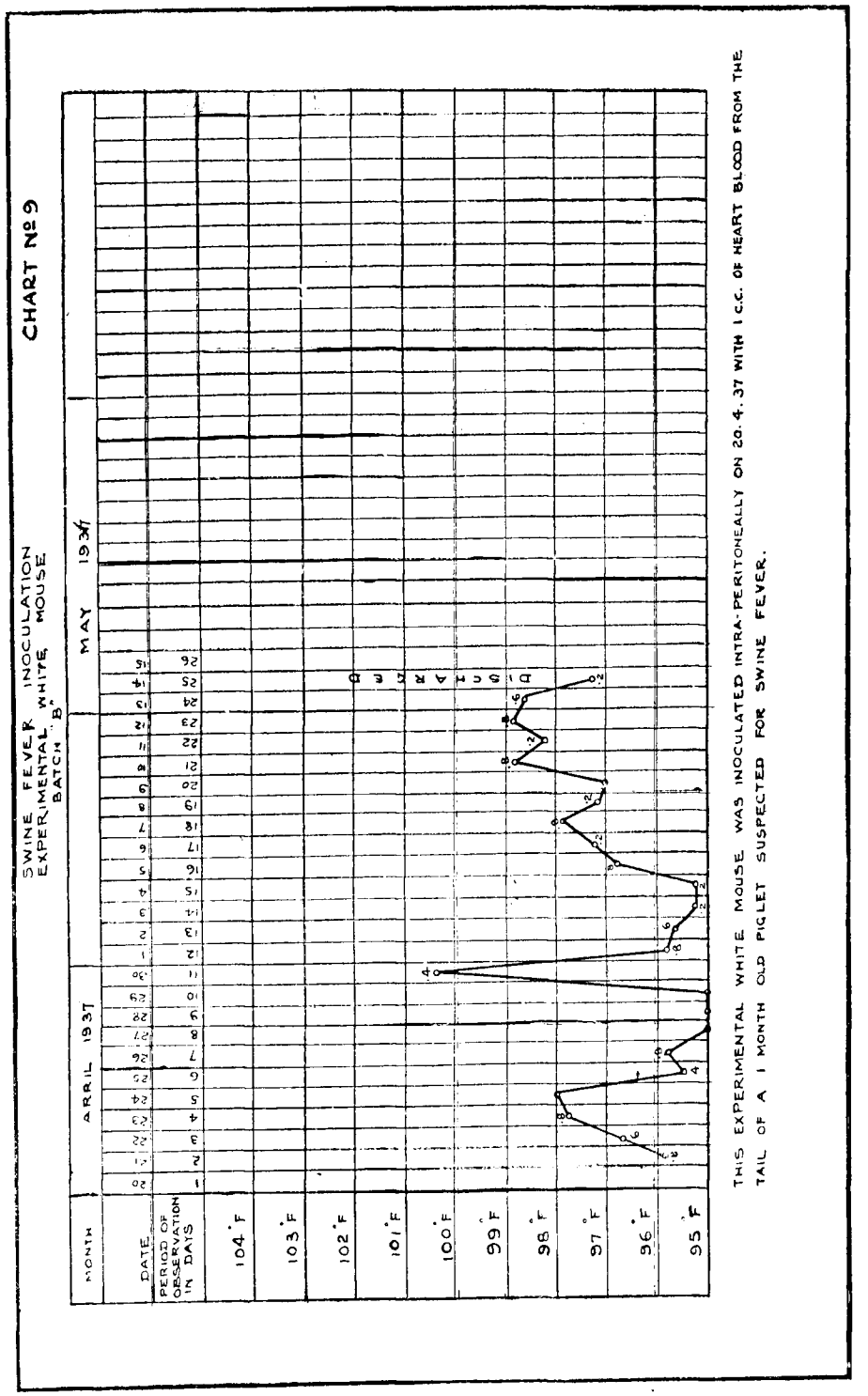
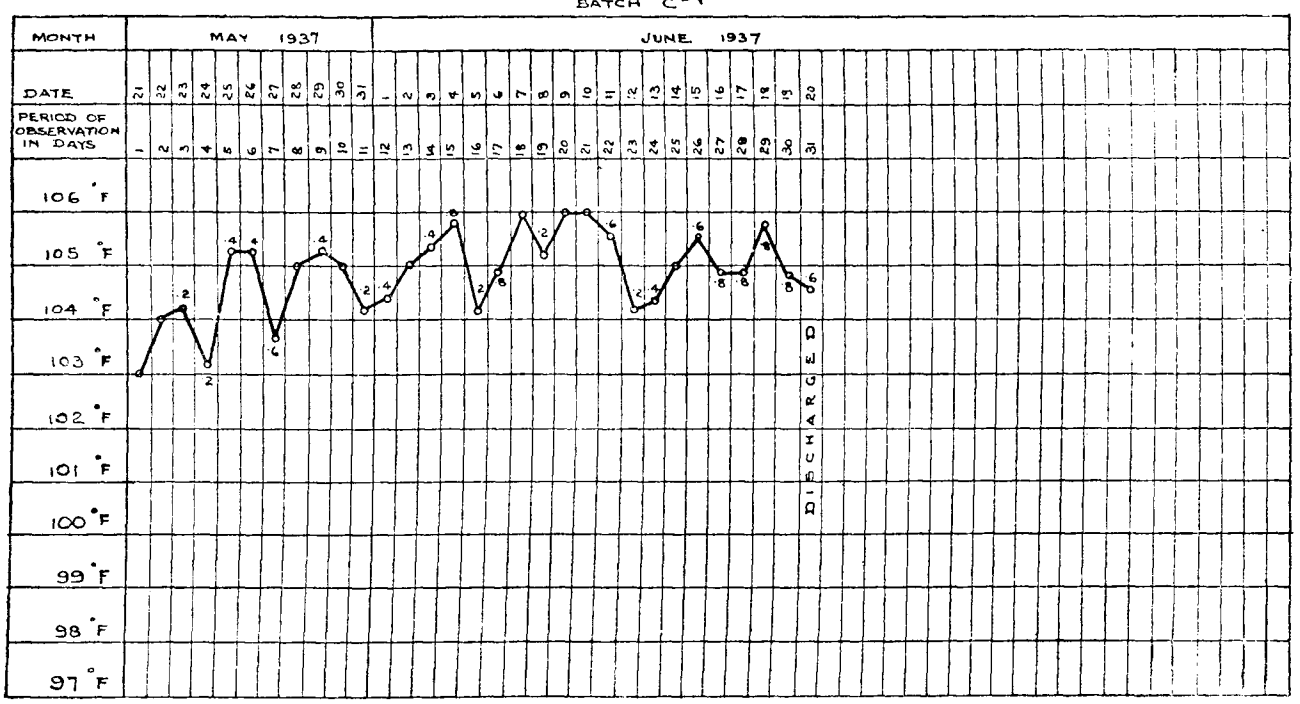


CHART Nº 9
SWINE FEVER INOCULATION
EXPERIMENTAL WHITE MOUSE
BATCH B'
APRIL 1937
MAY
MONTH
DATE
PERIOD OF OBSERVATION IN DAYS
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
96 °F
95 °F
DISCHARGE
THIS EXPERIMENTAL WHITE MOUSE WAS INOCULATED INTRA-PERITONEALLY ON 20. 4. 37 WITH 1 c.c. OF HEART BLOOD FROM THE
TAIL OF A 1 MONTH OLD PIGLET SUSPECTED FOR SWINE FEVER.

SWINE FEVER INOCULATION
EXPERIMENTAL GUINEA-PIG Nº 4
BATCH "C"

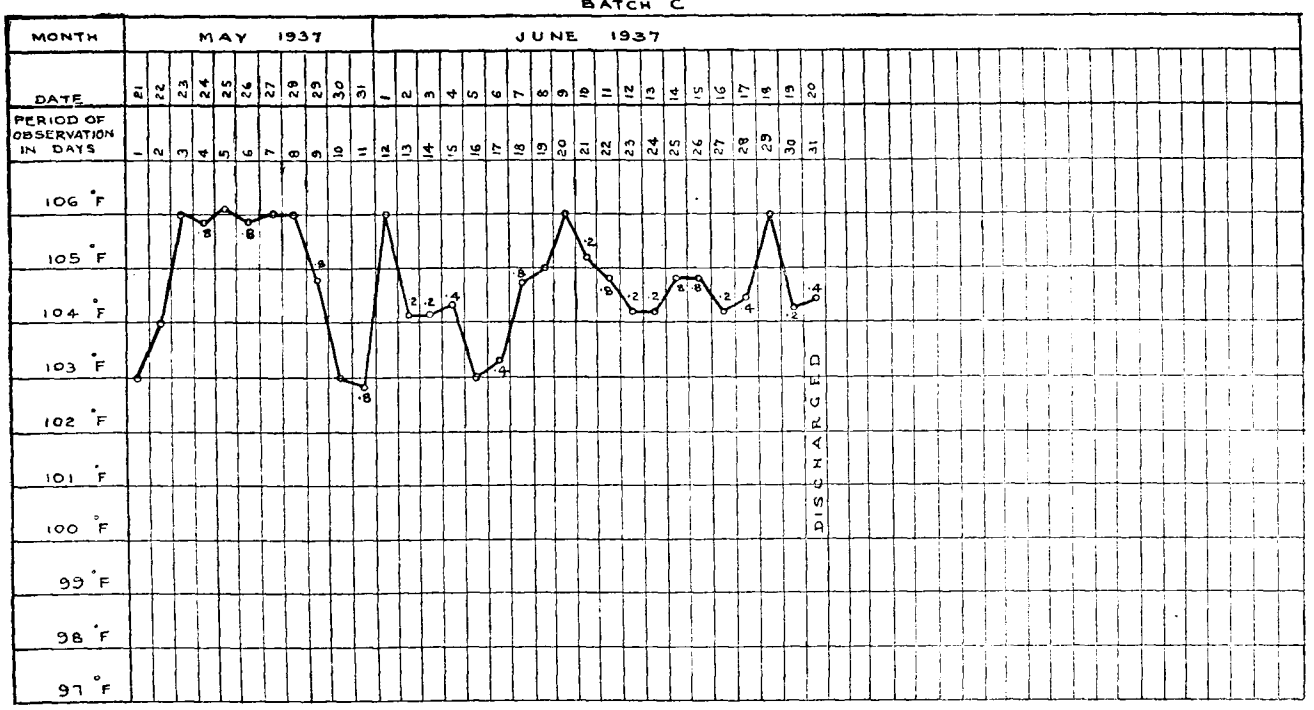
CHART Nº 10



THIS EXPERIMENTAL GUINEA-PIG WAS INOCULATED INTRA-PERITONEALLY ON 21.5.37 WITH 1 c.c. OF URINE FROM EXPERIMENTAL GUINEA-PIG Nº 2, WHICH WAS SIMILARLY INOCULATED ON 19.4.37 WITH 1 c.c. OF HEART BLOOD, FROM A PIGLET SUSPECTED FOR SWINE FEVER. GUINEA PIG Nº 2. DIED ON 21.5.37

SWINE FEVER INOCULATION
EXPERIMENTAL GUINEA PIG Nº 5
BATCH "C"

CHART Nº 11



THIS EXPERIMENTAL GUINEA-PIG WAS INOCULATED INTRA-PERITONEALLY ON 21.5.37 WITH 1 c.c HEART BLOOD FROM EXPERIMENTAL GUINEA-PIG Nº 2, WHICH WAS SIMILARLY INOCULATED ON 19.4.37 WITH 1 c.c OF HEART BLOOD FROM A PIGLET SUSPECTED FROM SWINE FEVER

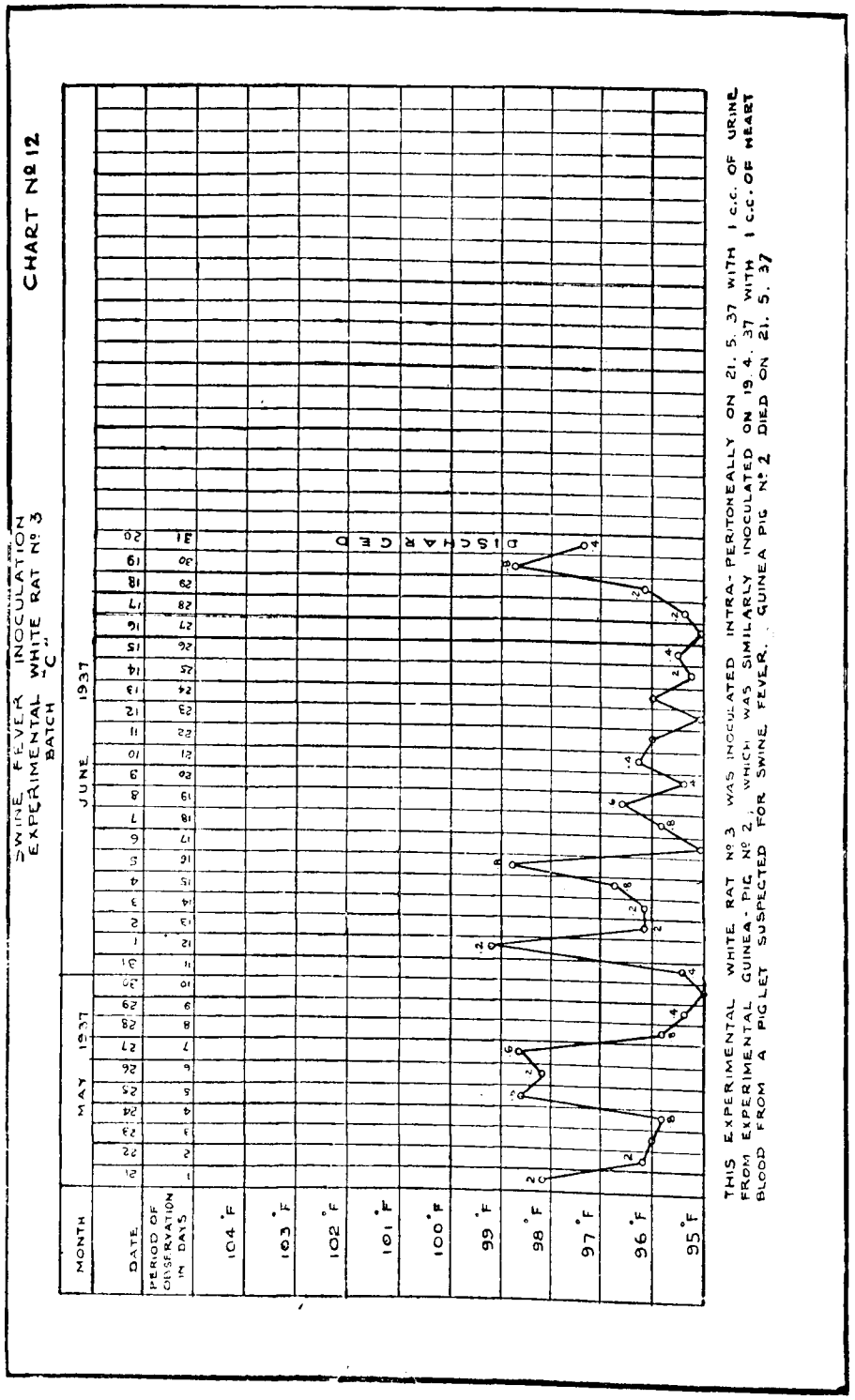


CHART Nº 12
SWINE FEVER INOCULATION
EXPERIMENTAL WHITE RAT Nº 3
BATCH "C"
MONTH
MAY 1937
JUNE 1937
DATE
PERIOD OF OBSERVATION IN DAYS
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
96 °F
95 °F
DISCHARGED
THIS EXPERIMENTAL WHITE RAT Nº 3 WAS INOCULATED INTRA-PERITONEALLY ON 21. 5. 37 WITH 1 c.c. OF URINE FROM EXPERIMENTAL GUINEA-PIG Nº 2, WHICH WAS SIMILARLY INOCULATED ON 19. 4. 37 WITH 1 c.c. OF HEART BLOOD FROM A PIGLET SUSPECTED FOR SWINE FEVER. GUINEA PIG Nº 2 DIED ON 21. 5. 37

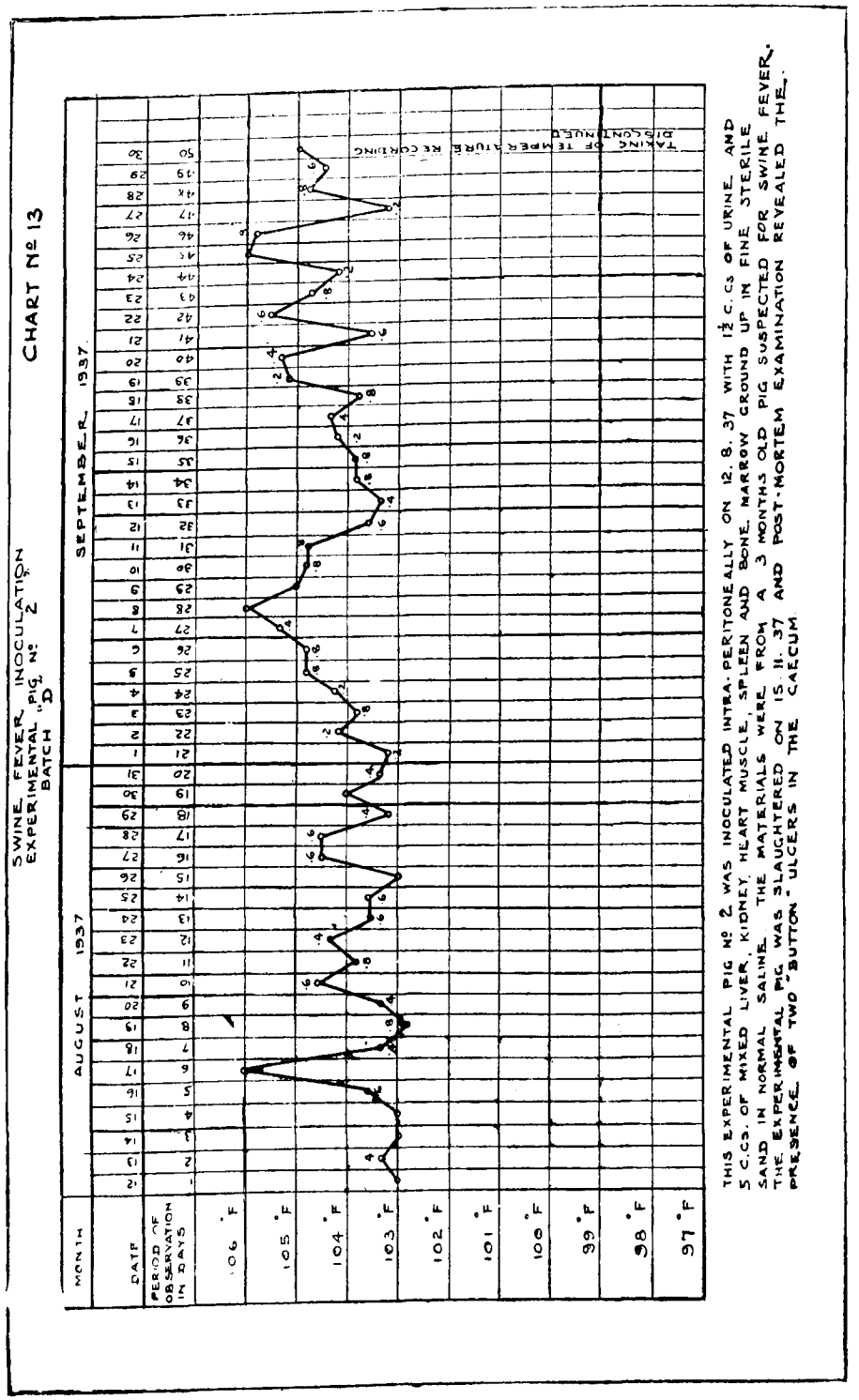


CHART Nº 13
SWINE FEVER INOCULATION
EXPERIMENTAL "PIG Nº 2"
BATCH "D"
MONTH
AUGUST 1937
SEPTEMBER 1937
DATE
PERIOD OF OBSERVATION IN DAYS
106 °F
105 °F
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
TAKING OF TEMPERATURE RE COMMENCED
DISCONTINUED
THIS EXPERIMENTAL PIG Nº 2 WAS INOCULATED INTRA-PERITONEALLY ON 12. 8. 37 WITH 1½ C.Cs OF URINE AND 5 C.CS. OF MIXED LIVER, KIDNEY, HEART MUSCLE, SPLEEN AND BONE MARROW GROUND UP IN FINE STERILE SAND IN NORMAL SALINE. THE MATERIALS WERE FROM A 3 MONTHS OLD PIG SUSPECTED FOR SWINE FEVER. THE EXPERIMENTAL PIG WAS SLAUGHTERED ON 15. 11. 37 AND POST-MORTEM EXAMINATION REVEALED THE PRESENCE OF TWO "BUTTON" ULCERS IN THE CAECUM

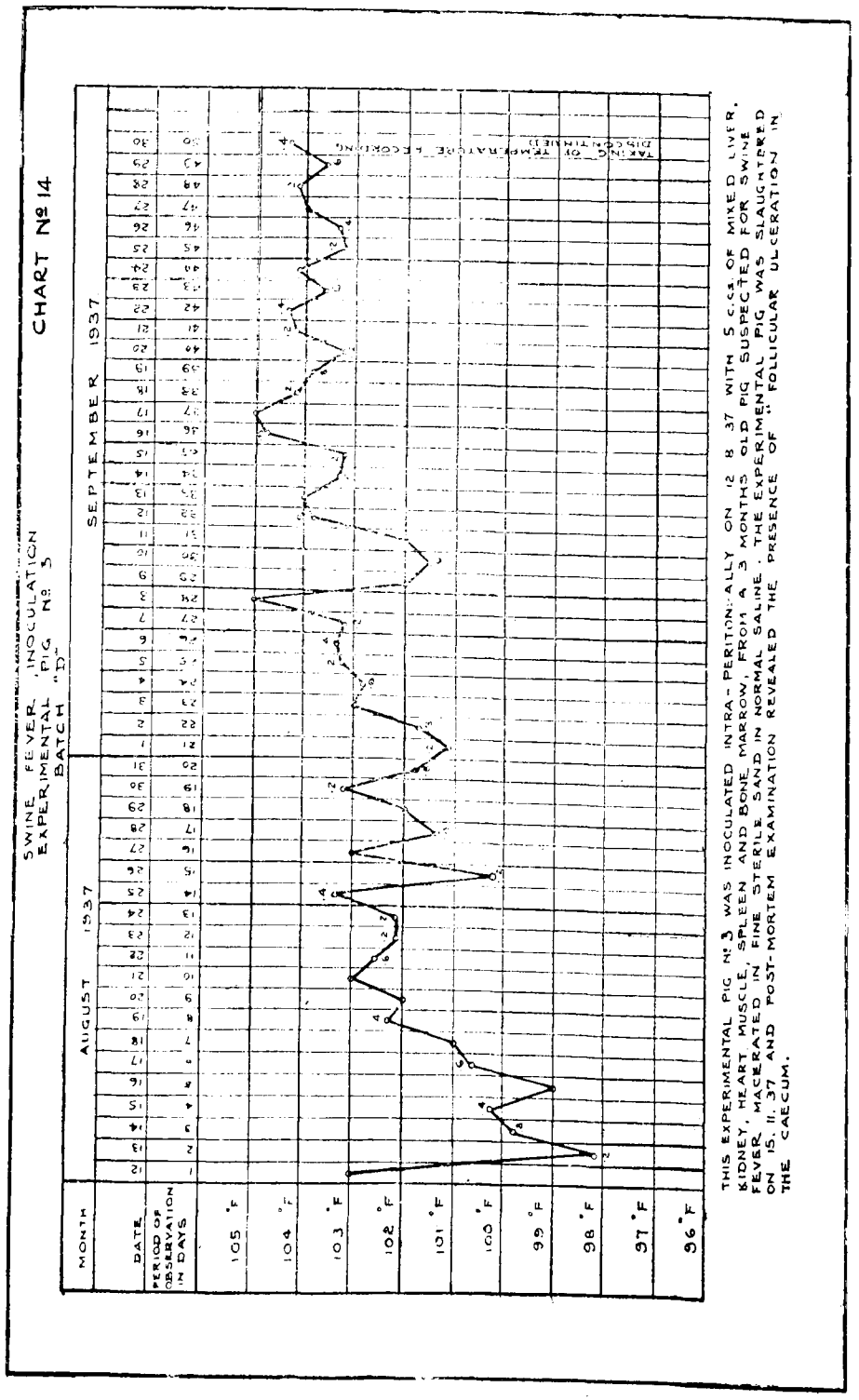


CHART Nº 14
SWINE FEVER INOCULATION
EXPERIMENTAL "PIG Nº 3"
BATCH "D"
SEPTEMBER 1937
AUGUST 1937
MONTH
DATE
PERIOD OF OBSERVATION IN DAYS
TAKING OF TEMPERATURE BEGINNING
DISCONTINUED
105 °F
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
96 °F
THIS EXPERIMENTAL PIG Nº 3 WAS INOCULATED INTRA-PERITONEALLY ON 12.8.37 WITH 5 c.cs. OF MIXED LIVER, KIDNEY, HEART MUSCLE, SPLEEN AND BONE MARROW, FROM A 3 MONTHS OLD PIG SUSPECTED FOR SWINE FEVER, MACERATED IN FINE STERILE SAND IN NORMAL SALINE. THE EXPERIMENTAL PIG WAS SLAUGHTERED ON 15.11.37 AND POST-MORTEM EXAMINATION REVEALED THE PRESENCE OF FOLLICULAR ULCERATION IN THE CAECUM.

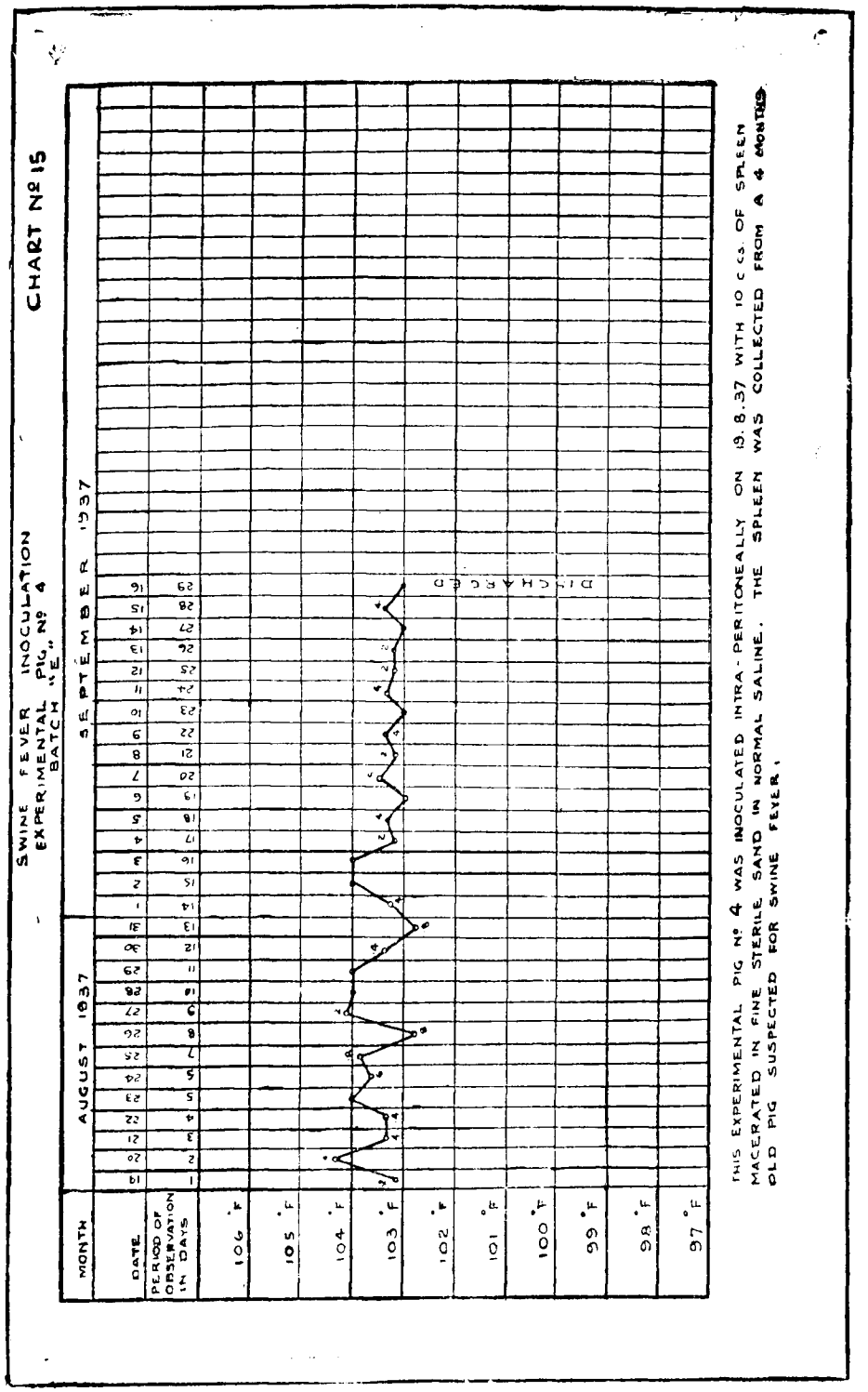


CHART Nº 15
SWINE FEVER INOCULATION
EXPERIMENTAL "PIG" Nº 4
BATCH "E"
SEPTEMBER 1937
AUGUST 1937
MONTH
DATE
PERIOD OF OBSERVATION IN DAYS
DISCHARGED
106 °F
105 °F
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
THIS EXPERIMENTAL PIG Nº 4 WAS INOCULATED INTRA-PERITONEALLY ON 19.8.37 WITH 10 c.cs. OF SPLEEN MACERATED IN FINE STERILE SAND IN NORMAL SALINE. THE SPLEEN WAS COLLECTED FROM A 4 MONTHS OLD PIG SUSPECTED FOR SWINE FEVER.

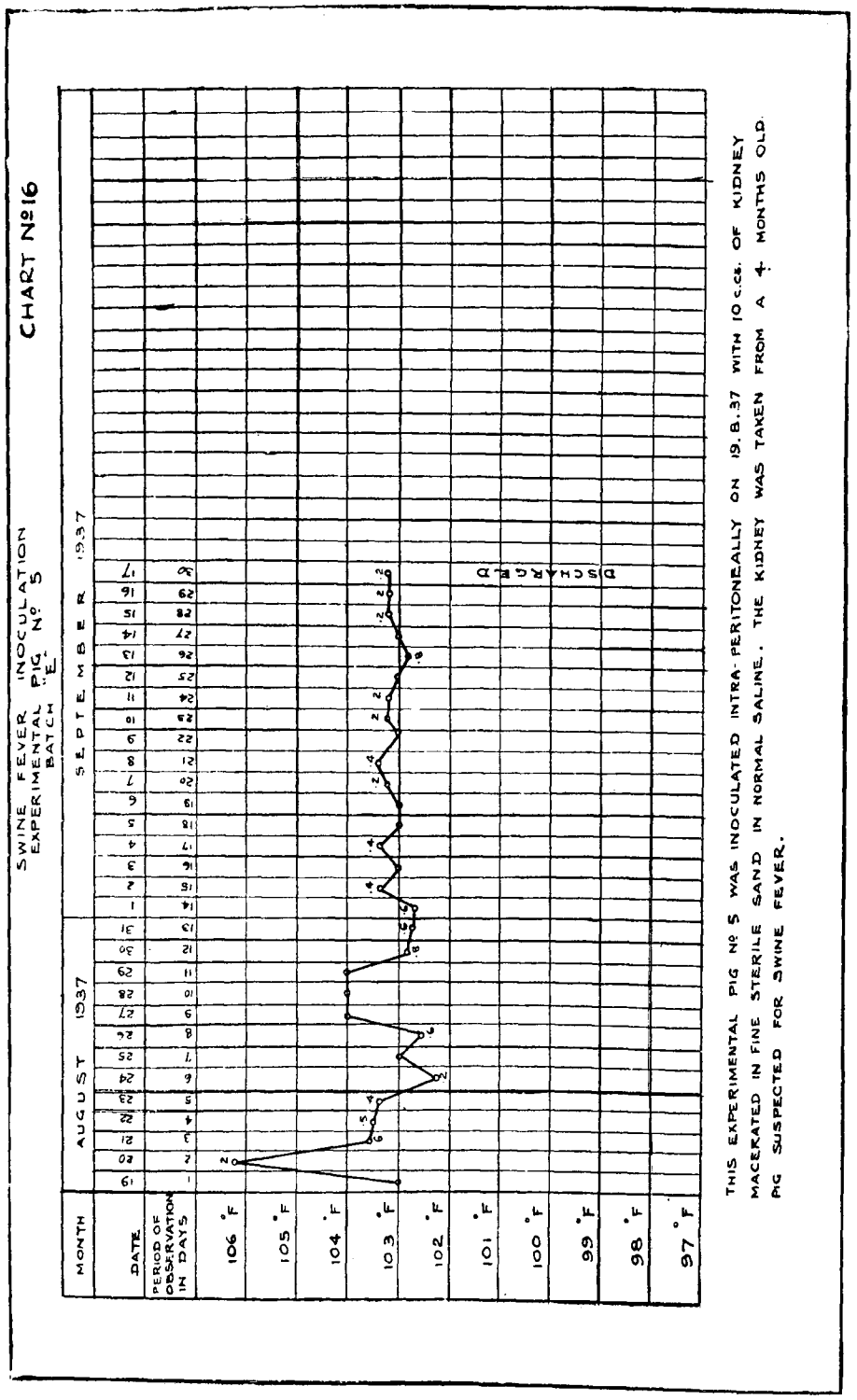


CHART Nº16
SWINE FEVER INOCULATION
EXPERIMENTAL PIG Nº 5
BATCH "E"
SEPTEMBER 1937
AUGUST 1937
MONTH
DATE
PERIOD OF OBSERVATION IN DAYS
106 °F
105 °F
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
DISCHARGED
THIS EXPERIMENTAL PIG Nº 5 WAS INOCULATED INTRA-PERITONEALLY ON 19.8.37 WITH 10 c.cs. OF KIDNEY MACERATED IN FINE STERILE SAND IN NORMAL SALINE. THE KIDNEY WAS TAKEN FROM A 4 MONTHS OLD PIG SUSPECTED FOR SWINE FEVER.

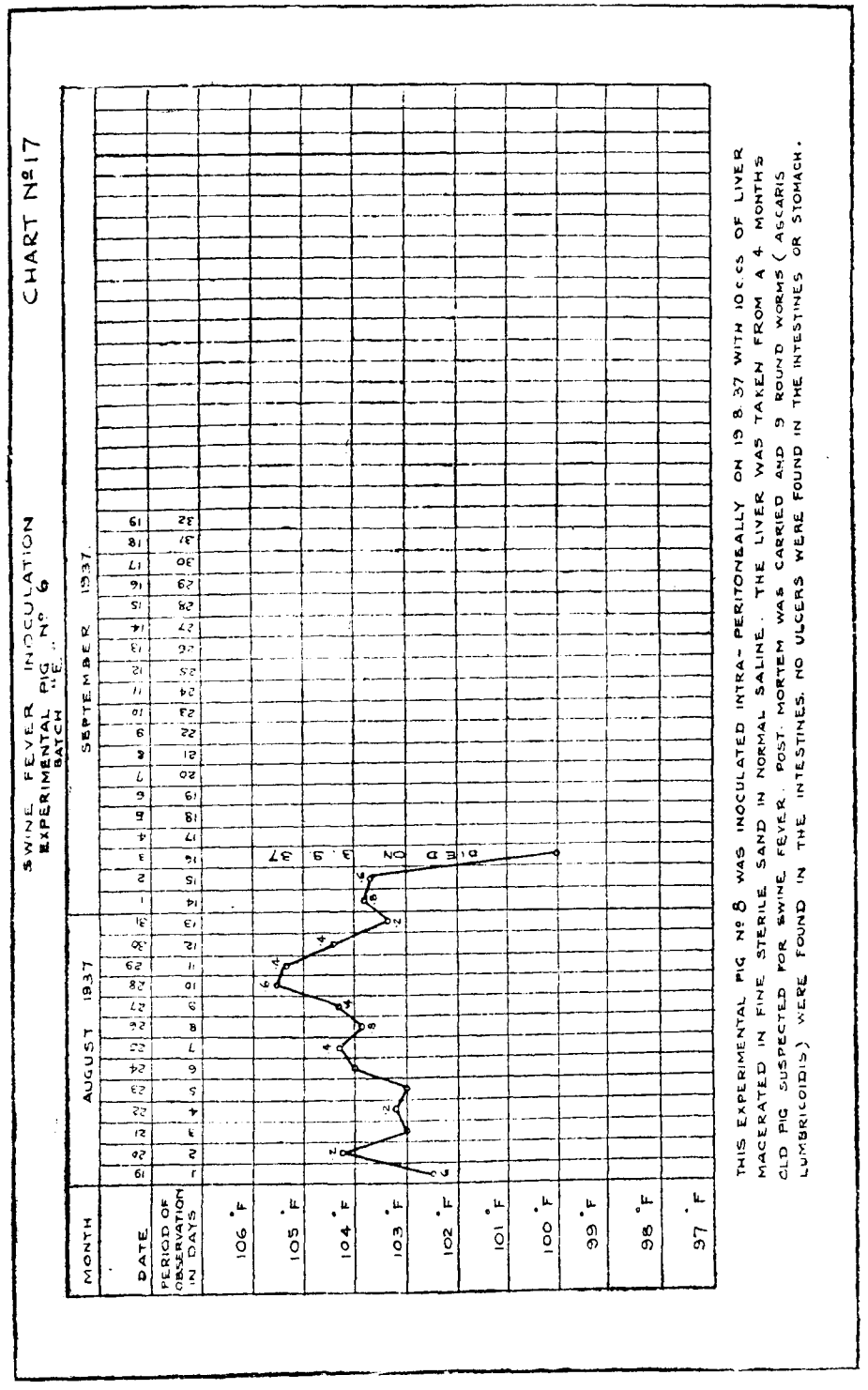
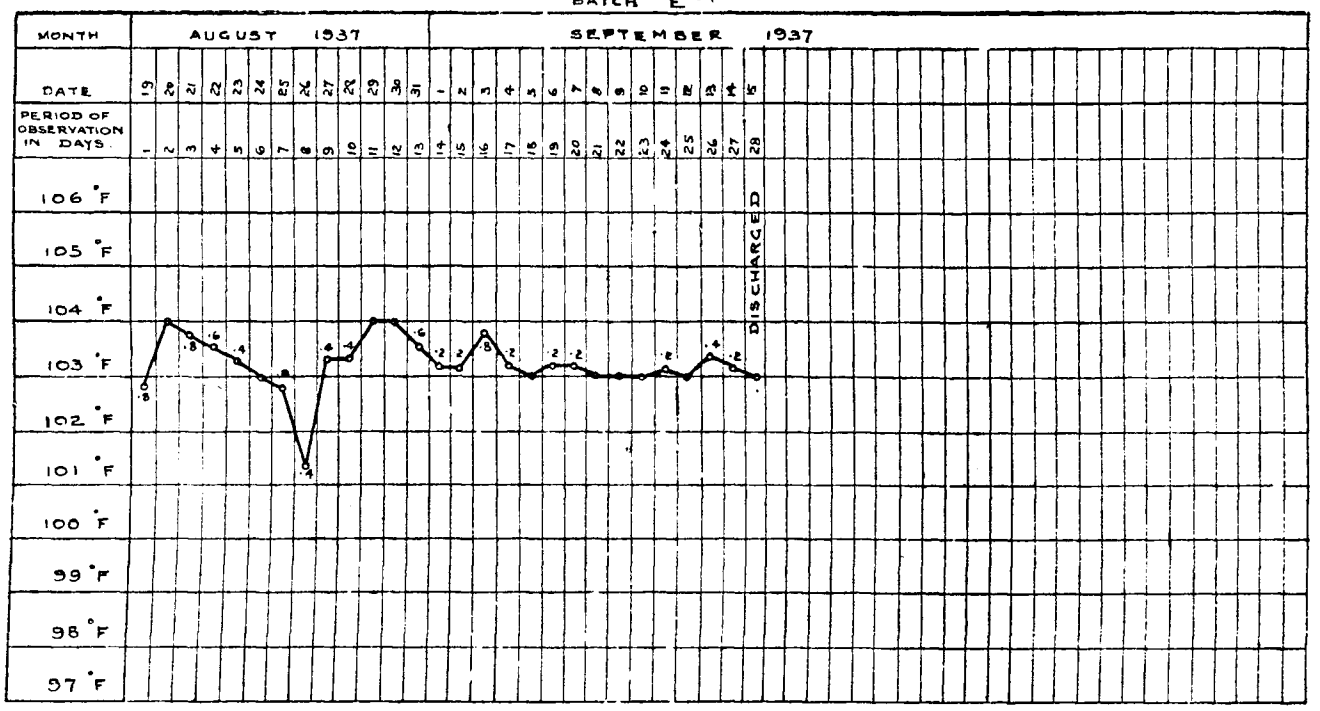


CHART Nº17
SWINE FEVER INOCULATION
EXPERIMENTAL PIG Nº 6
BATCH "E"
SEPTEMBER 1937
AUGUST 1937
MONTH
DATE
PERIOD OF OBSERVATION IN DAYS
106 °F
105 °F
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
DISCHARGED
THIS EXPERIMENTAL PIG Nº 6 WAS INOCULATED INTRA-PERITONEALLY ON 19.8.37 WITH 10 c.cs OF LIVER MACERATED IN FINE STERILE SAND IN NORMAL SALINE. THE LIVER WAS TAKEN FROM A 4 MONTHS OLD PIG SUSPECTED FOR SWINE FEVER. POST MORTEM WAS CARRIED AND 9 ROUND WORMS (ASCARIS LUMBRICOIDIS) WERE FOUND IN THE INTESTINES. NO ULCERS WERE FOUND IN THE INTESTINES OR STOMACH.

SWINE FEVER INOCULATION
EXPERIMENTAL PIG Nº 7
BATCH "E"

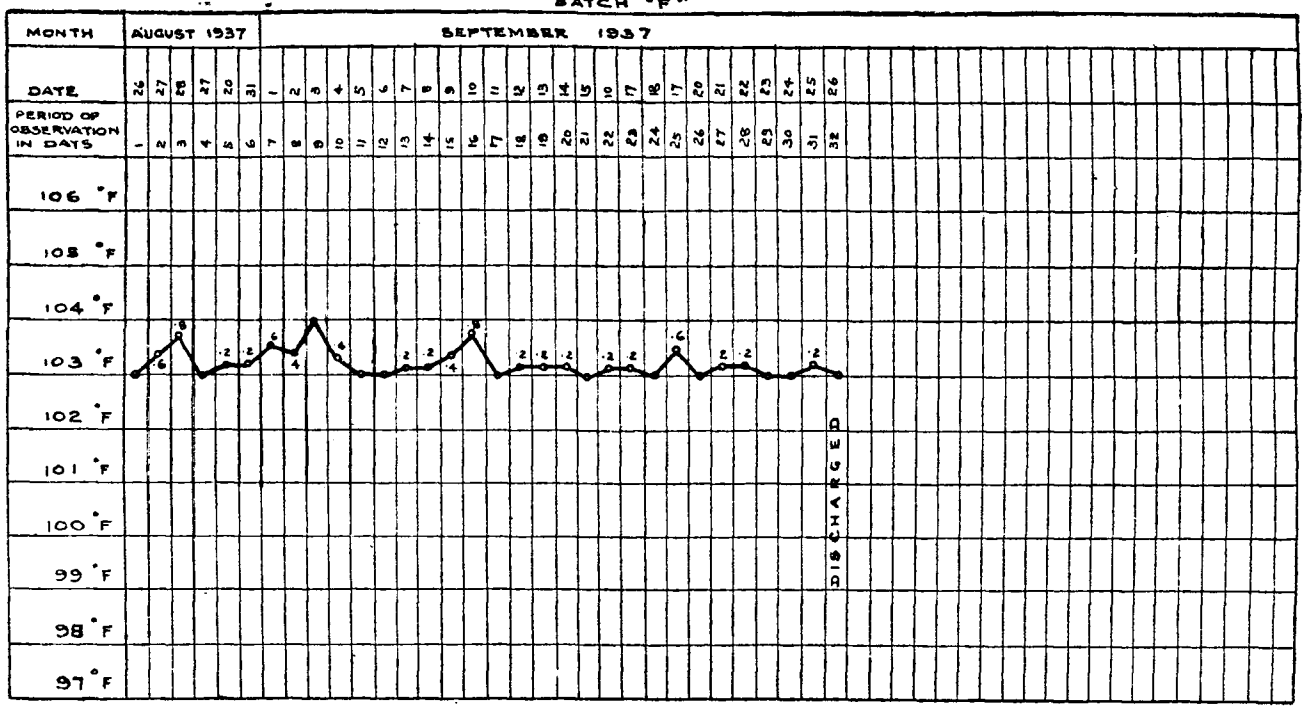
CHART Nº18



THIS EXPERIMENTAL PIG Nº 7 WAS INOCULATED INTRA-PERITONEALLY ON 13. 8. 37 WITH 10 C.Cs. OF URINE DRAWN FROM A 4 MONTHS OLD PIG SUSPECTED FOR SWINE FEVER.

SWINE FEVER INOCULATION
EXPERIMENTAL PIG Nº 8
BATCH "F"

CHART Nº19



THIS EXPERIMENTAL PIG Nº 8 WAS INOCULATED INTRA-PERITONEALLY ON 26. 8. 37 WITH 10 C.Cs. OF HEART MUSCLE MACERATED IN FINE STERILE SAND IN NORMAL SALINE. THE HEART MUSCLES WAS TAKEN FROM A 1 MONTH OLD PIGLET SUSPECTED FOR SWINE FEVER.

V. R. RAJAGOPALAN. G.M.V.C.,
Assistant Veterinary Research Officer, I. V. R. I., Muktesar, who has left for Manchester for taking Diploma in Bacteriology.

W. V. SOMAN. G.B.V.C., P.G
Chief Veterinary Surgeon, Baroda, who has left for Edinburgh for higher post-graduate studies.

THE

INDIAN VETERINARY JOURNAL

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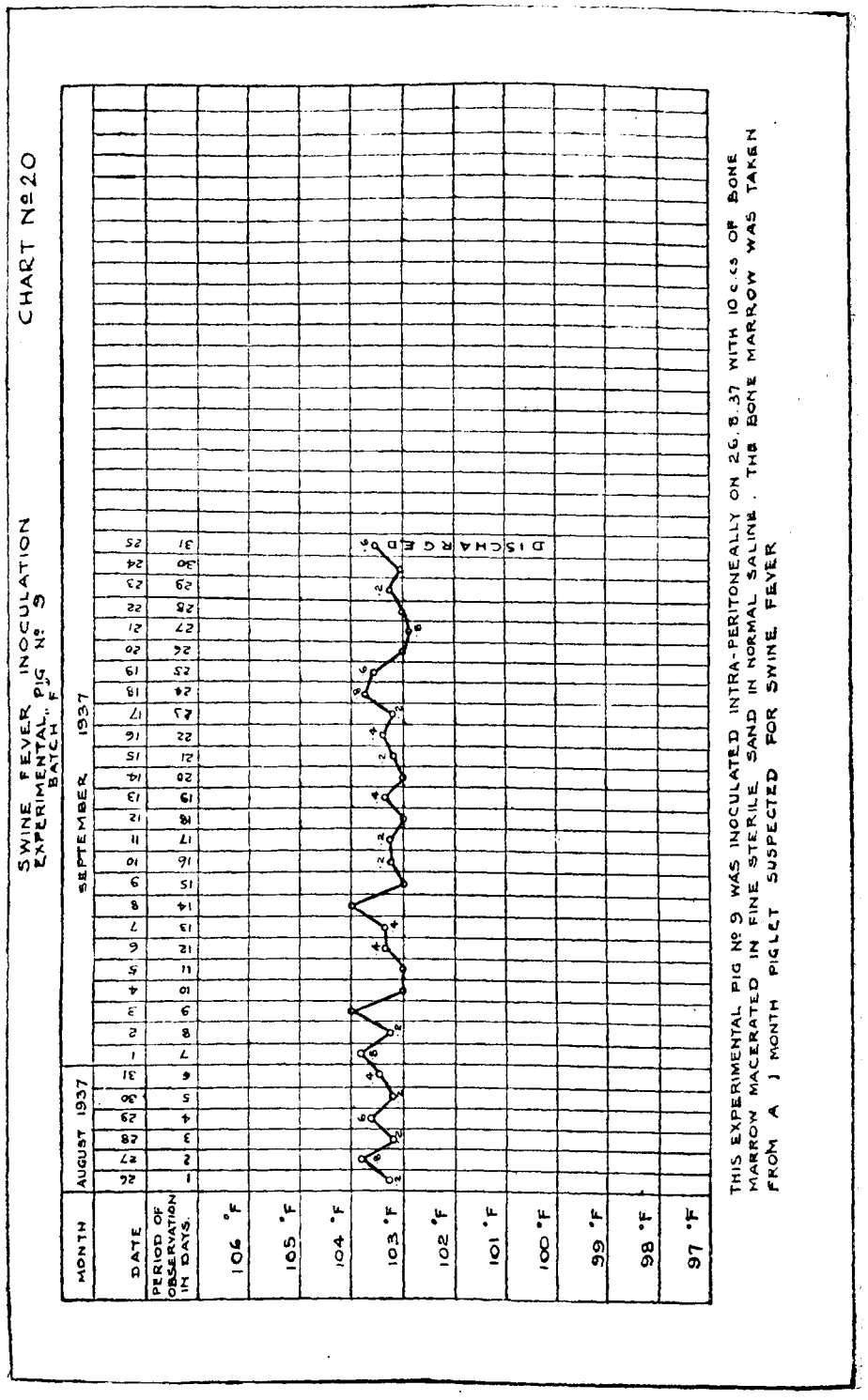
Editorials.

IMPROVEMENT OF CATTLE.

INDIA is primarily an agricultural country: cattle form the mainstay of the agriculturist: the prosperity of the agriculturist depends upon the prosperity of his cattle: the cow and the bullock bear on their patient backs the whole structure of Indian Agriculture—these and similar slogans are being repeated at frequent intervals from the platform and through the press; but when we come to take stock of what actually is being done to improve the live-stock of the country and to make the agriculturist prosperous, we realise that even the fringe of the problem has not yet been touched. A few farms here and there, and a handful of Premium Stud Bulls scattered over the country—these have comprised more or less the entire Animal Husbandry work. It has, therefore, become necessary to have a clear conception of what really ought to be done, so that valuable time and money may not be frittered away in experiments of very doubtful utility.

It is a well-known fact that our country has got a number of excellent indigenous breeds of cattle which had been evolved solely with an eye to their utility. It is also an equally well-known fact that these breeds of cattle have been gradually deteriorating, chiefly from want of care and attention to their *breeding and feeding.* It is further agreed that there is bound to be definite and rapid all-round improvement when once these two defects are removed. This aspect of the matter has been forcibly brought out


SWINE FEVER INOCULATION
EXPERIMENTAL PIG No. 9
BATCH F
CHART No 20
AUGUST 1937
SEPTEMBER 1937
MONTH
DATE
PERIOD OF OBSERVATION IN DAYS.
106 °F
105 °F
104 °F
103 °F
102 °F
101 °F
100 °F
99 °F
98 °F
97 °F
DISCHARGED
THIS EXPERIMENTAL PIG No 9 WAS INOCULATED INTRA-PERITONEALLY ON 26.8.37 WITH 10 c.cs OF BONE MARROW MACERATED IN FINE STERILE SAND IN NORMAL SALINE. THE BONE MARROW WAS TAKEN FROM A 1 MONTH PIGLET SUSPECTED FOR SWINE FEVER

by experiments which were recently conducted and which have been recorded by Col. Olver, till lately the Animal Husbandy Expert with the Government of India, in the following words:—

> "Within the last 20 years, simply by proper feeding and management, combined with *strictly controlled breeding* (Italics ours), the average milk-yield of several herds of pure-bred indigenous cows in India has been raised from 5·3 lbs. to 16·8 lbs. per diem. With more forcing methods such as special hand-feeding, very frequent milking, and very high feeding, which are commonly employed by pedigree breeders of dairy cattle in other countries to obtain records, there is little doubt that still higher yields could have been obtained."

This gives us in a nutshell that the real remedy for the improvement of the live-stock is to be looked for in better feeding and more careful management of our own live-stock. This object cannot possibly be achieved by the opening of a couple of breeding farms in a presidency and trying to evolve a pure strain of cattle under conditions greatly foreign to their native habitat. It may be that the breeding and selection of cattle in these farms is done under the management of experts, but, then, there is one great drawback which, in our opinion, is very vital. These farms, by the very nature of their constitution and administration, cannot possibly enlist the co-operation and interest of the ryots, although the entire organisation is for their benefit. The majority of the ryots look at them as mere 'show places' intended for the benefit of all others except themselves. There is also the other drawback of the farms, *viz.,* that they cannot possibly meet the demand for the necessary stud bulls required for the entire presidency. And, therefore, the selective breeding of cattle and their improvement must be done in the farms of the ryots themselves with their active help and co-operation. There are a number of breeders in the various parts of the country whose knowledge of things pertaining to Animal Husbandry is not negligible. They may not be well versed in the three R's and they may not be educated according to the modern concepts. But their knowledge of practical cattle-breeding is something extraordinary. It is the accumulated experience of generations of practical farming and

practical Animal Husbandry. They know exactly what the ryots need; they study the conditions of the market very accurately and they try to meet those requirements in their own limited way. If latterly they have become apathetic to their profession of cattle-breeding and indifferent to their occupation of cattle-rearing, it is not because they have no interest in their work. It is because of the grinding poverty in the country-side, of the encroachment on pasture lands, and of the absence of patronage and encouragement from the Government. And, therefore, unless these causes are remedied, there is very little chance of our inducing these people to take a live interest in their work.

With this object in view, as a first and preliminary step, the Government must arrange for taking a comprehensive census of the real cattle-breeders in the various parts of the country with a complete analysis of their capacities and potentialities for cattle-breeding and cattle-rearing. These people must be approached by the Government and induced to take an active interest in their work by the grant of liberal subsidies either in cash or in kind, as, for instance, by the offer of Grantee farms solely intended for cattle-breeding on the lines of what is being done in the Punjab, or by the grant of free pasturage in the forest areas. These subsidies can be made conditional on the production annually *of so many approved Breeding Bulls of the type required for that locality*, which should be available for distribution at a nominal price. A condition for the better management and milking of cows can also be imposed and, for this purpose, the co-operation of the owners of small holdings should be secured. These people—themselves and the members of their families—are generally their own masters and servants, leading a precarious existence from the tilling of the soil. An offer of a subsidy, from the Government to them, however small it may be, will be a great inducement for them and they will very willingly and cheerfully abide by the conditions imposed; especially so, when they know that they are all in their own interests. Simultaneously, better facilities for the transport of fodder and cattle from place to place should be provided. If the above plans be adopted, we are quite confident that the whole country-side

will all at the same time be electrified into action with a comparatively less cost to the Government than the maintenance of a number of farms at an enormous expenditure. It is, therefore, up to the popular Governments in the various provinces to initiate a move on the above lines for the improvement of our own live-stock and add to the prosperity of the country-side.

IS A CENTRAL VETERINARY COLLEGE A NECESSITY ?

IN discussing the methods for improving the Veterinary Education in this country, we gave expression in the last issue of *this Journal* to the view that the best method would be to improve the standard of teaching in the existing Veterinary Colleges in India. We are very glad to learn that the above suggestion has been taken up by the authorities responsible for imparting Veterinary Education in the Madras Veterinary College. As our readers are aware of, this College has been affiliated to the Madras University for the B.V.Sc. degree. The Board of Studies of this University, in one of its recent meetings, has, we are reliably informed, recommended to the Academic Council to increase the course of studies for a period of five years. This is a welcome step in the right direction and is in accordance with what we have been suggesting in these columns for some time. We, therefore, earnestly hope that the above recommendation of the Board of Studies will be accepted before long by the higher authorities and given effect to with as little delay as possible.

We are also informed that the Lahore Veterinary College, whose equipment is in no way inferior to any of the Veterinary Colleges in the East, is taking steps to increase the course of studies there from four to five years and get it affiliated to the University. We trust that these steps would be pushed on vigorously to a successful end in the near future.

With Madras setting the pace for imparting the Veterinary Education up to the highest standard, and Lahore following suit, the question naturally arises, 'Is a Central Veterinary College still a

necessity?' There cannot be any doubt about the answer and it should be an emphatic 'No'. The arguments for opening a Central Veterinary College, if there were any, have now completely lost their force and substance and there can be no justification whatsoever for pursuing the proposal. As we have said before, it has been a proposal with not one single saving factor about it. It has been neither in the interest of this country nor in the interest of the profession. And, therefore, the sooner it is dropped, the better for all concerned.

But, then, there is one thing which the Central Government can really do, if it sincerely wishes to improve the standard of Veterinary Education in this country. Practically, all the Provinces, wherein the Veterinary Colleges are situated, are suffering from paucity of funds and they find it very difficult to find the money even for ameliorative measures. The equipment of Veterinary Colleges to enable them to teach up to a high standard necessarily involves increased expenditure and this, *and this alone*, frightens many an otherwise willing Provincial Government to consider the proposal of revising the course of studies in their Colleges. As such, if the Central Government were to come forward and promise a liberal grant of subsidy, we are sure that most of the Provincial Governments would be only too glad to raise and maintain the standard of the education. This should not be a difficult thing for the Central Government to do; because, they have already seriously proposed to spend a lot of money on the opening of a Central Veterinary College. To be definite, they had provided for a capital outlay of nine lakhs and a recurring expenditure of two lakhs. If this amount were to be divided amongst the Veterinary Colleges in India, all of them would, in no time, be modernised and equipped in an up-to-date manner for the efficient teaching of Veterinary Science. This means that, in the place of one Veterinary College in a remote corner of the country, at least some of the existing Veterinary colleges would be adequately equipped and staffed immediately and these would function in a way far better than the proposed solitary one could do. What is more, each and every one of them, individually, would be able to study and meet the requirements of the

peculiar conditions prevailing in the different provinces of this sub-continent; for, let it be remembered that the conditions of Veterinary service and the needs of live-stock owners in this country are widely different not only in the provinces themselves but from other countries as well. As such, the Veterinary Education in the Indian Veterinary Colleges cannot possibly be a pattern of what it is in the British Isles.

In this connection we would emphasise the need for very early attention being paid to encourage post-graduate work and special training for those who by their aptitude and previous work have shown promise of being good and efficient teachers in the various departments of study in a Veterinary College. Unless immediate attention is focussed on this aspect of Veterinary Education it would be futile to look for men with the requisite specialised training (on reasonable rates of pay) to staff the Indian Veterinary College with local recruits in the future.

Will the Central Government take into consideration the above factors and kindly revise their policy with regard to the opening of a Central Veterinary College in Izatnagar? We hope and trust they will.

INDIAN HORSES.

WE draw the attention of the Government to an article under the above heading extracted in another page of *this Journal* from *The Hindu* of September 19, 1938. The author of this article is a well-known racehorse owner and trainer, and, as such, he speaks with an *inside* knowledge of things. It is extraordinary that, while other empire centres are free to dump their horses in this country, we are banned from sending our own home-bred animals to those countries. The only reason that could be advanced for this ban is the risk of infectious diseases being imported into those countries. But, even this cannot possibly stand a moment's scrutiny. England is the possessor of the finest and most valuable live-stock in the whole world and, still, she does not impose any

such ban on the import of horses from this country. As such, it is quite unfair that countries like Australia, from where large numbers of horses are being annually imported into this country, should have such discriminating laws against India.

Having said this much we must admit we cannot agree with the remedies suggested by the author of the extracted article. On the other hand we must make the best advantage of this ban to the advantage of this country.

Our own home-bred horses have been languishing for want of encouragement—both from the Government and the public. They have been in no way found wanting either in stamina or in build and, given the chance and opportunity, they will prove their mettle to the satisfaction of every one concerned. If, therefore, a reciprocal ban is imposed on the import of horses from other countries, our horses will come into their own in no time. Not only that, the lakhs of money that are being spent on the purchase of horses from outside India will then remain in this country itself and will stimulate the various horse-breeders to take a more lively interest in horse-breeding. By so doing we could utilise this discriminating ban to our own advantage, which will then prove to be a blessing in disguise.

We may here mention that, by allowing an unrestricted and free export of cows from Nellore tract in the Madras Presidency, the stock in that locality became quite so depleted that the Government had to intervene and put a ban on the export, and stop further deterioration of cattle. This had the desired effect of improving the breed. In a similar way, the existing ban on horses can be well utilised for our benefit and advantage.

We, therefore, appeal to the Nation-building Governments and to the members of the legislature to take up this question in right earnest and see that a ban is put upon the free import of horses into this country.

Rao Bahadur V. R. PHADKE, G.B.V.C., J.P.,

Retired Director of Veterinary Services, Bombay Presidency, who has been appointed as Acting Director of Veterinary Services, Sind.

An Appreciation (Contributed).

RAO BAHADUR V. R. PHADKE was born of poor but honest parents on the 21st October 1882, at Donavali, a village in Chiplum Taluka, in Ratnagiri District; nurtured under the careful guidance of his parents, his father being himself a teacher of a Marathi School, he was always amongst the first few and passed his Matriculation in 1900, in Akola in Berar. He joined the Bombay Veterinary College in February 1901, as a Govrrnment scholar and graduated with distinction in December 1903—being first in Class 'B' and second in Class 'C'. He was appointed as Assistant Veterinary Officer at the Bombay Veterinary College in 1904, and was appointed as Veterinary Inspector at Muktesar in the same year. He was transferred to the Bombay Veterinary College in February 1908 as Curator and Bacteriological Assistant and was made a Lecturer in January 1912. In September of the same year, he was deputed by Local Government for Post-graduate study to the United Kingdom. Wherever he went, he won laurels and was held in high esteem by the Professors under whom he worked. His love of science and devotion to duty coupled with sociable qualities endear him to all. He went through the D.V.H. course in Liverpool and also underwent Post-graduate training at the London School of Tropical Medicine and at the Royal Veterinary College, Camden Town. He returned to Bombay in 1914. When War broke out in 1914 and there was a call for War service, he was the first to volunteer his services from the College and went to Marseilles with Remounts. In 1916, he was posted to fill an important appointment at the Muktesar Institute. He had to visit the Government Military Dairy Farms in India in connection with the Serum-simultaneous method against Rinderpest. His ceaseless work and devotion to duty earned for him the unanimous appreciation of those with

whom he came in contact. He was the first Indian who worked out this method successfully between 1916 and 1920.

Merit has its reward. In 1920, he was appointed as Assistant Professor at the Bombay Veterinary College.

Again, in 1922, he was selected to undergo Post-graduate study at Muktesar under Professor J. T. Edwards, prior to promotion to the Imperial Veterinary Service. He acted for two years in the Imperial Service, but the Government of India having accepted the Lee Commission's recommendations he could not be promoted to that service.

In 1924, he acted as Vice-Principal. In 1926 and 1928 he acted as the Principal when the permanent Principal went on long leave. Wherever he got an opportunity, he demonstrated the truth that, given opportunities, an Indian is not inferior to any other in any walk of life. His appointment as Principal was only a fitting and well-merited recognition of his abilities.

He was not merely a Professor. The Bombay Government appointed him as Justice of the Peace in 1926. He was also an Honorary Presidency Magistrate.

Rao Bahadur Phadke was an active member of the Veterinary Service. He is the author of the pamphlet, *Multivitellaria hewletti*—a new fluke discovered by him in an Indian house crow.

He is a learned Professor admired alike for his erudition as much for his clear exposition. More than all, he is not a sectarian in his outlook. He is an Indian first, second and the last. He loved his students, past and present, as a father loved his children. He is a dear colleague much respected and admired by all those who worked with him and who have the pleasure to work with and under him now.

On retirement of K. Hewlett, Esq., O.B.E., M.R.C.V.S., I.V.S., Rao Bahadur V. R. Phadke was appointed as the permanent Principal of the Bombay Veterinary College on 1st April 1932. He

was the first Indian to be appointed as the permanent Principal of a Veterinary College. Yet higher laurels were in store for him. He was appointed as the Acting Director of Veterinary Service, Bombay Presidency, Poona, on 23rd April 1937 and retired from that post on 19th November 1937. The Bombay Government in recognition of his meritorious services conferred on him the title of *Rao Bahadur* on the New Year Honours Day of 1938. But he was not allowed to rest in his retired life. In appreciation of his past service and ability, he was appointed as the Director of Veterinary Services, Sind, from 9th May 1937, to 9th November 1938, and now he is a pensioner re-employed by the Government of Sind.

The official career of Rao Bahadur is one to be emulated by one and all of the Indian Veterinarians.

[We wish him long life and prosperity to enjoy his well-merited pension. We do hope, he will give his best also to the profession by becoming an active member of the All-India Veterinary Association and an enthusiastic contributor to its organ, the *Indian Veterinary Journal*—EDITOR, *I. V. J.*]

Khan Sahib CHAUDHRI ABDUL GHANI, G.P.V.C.,

Veterinary Deputy Superintendent, Imperial Veterinary Research Institute, Izatnagar, U. P.

An Appreciation (Contributed).

KHAN SAHIB CHAUDHRI ABDUL GHANI comes of a respectable and influential Zamindar family in Punjab and is the son of Chaudhri Sultan Baksh, Zamindar of Saidopatti, Hoshiarpur District.

The Khan Sahib graduated from the Punjab Veterinary College, Lahore, in the year 1904. After graduation he was selected by Col. Holmes, then Officiating Imperial Bacteriologist, for service at the Imperial Bacteriological Laboratory at Muktesar, as this Institute was then called. The Khan Sahib had the privilege

of working with almost all the Directors of the Institute and he can, therefore, be rightly regarded as one of the pioneers of Indian Veterinarians who witnessed the early spade work at the Imperial Bacteriological Laboratory, Muktesar.

He worked at Muktesar on Rinderpest and Experimental sections from 1904 to 1918 and conducted many thousands of operations in connection with serum production.

In the first week of January 1919, he was deputed to take charge of the work of Serum production (from the Veterinary Deputy Superintendent) at Kurgaina Branch Laboratory, Bareilly, which he carried on most efficiently till the end of March 1922.

In the year 1922 he was deputed again to Bareilly to remove the work of Serum production from Kurgaina to Izatnagar and make all the arrangements to commence the work of manufacture of serum there in the winter weather. The present Izatnagar site, which is now occupied by lofty and stately buildings and laboratories, was then full of thick jungle. The Khan Sahib worked very hard in the clearing of the jungle there and started the work of Serum manufacture after making all necessary arrangements to the entire satisfaction of the then Director. The Khan Sahib carried out his duties tactfully till the 28th February 1938, after which he proceeded on leave preparatory to retirement.

In the year 1924, he was also deputed to collect calves from various distant centres of India for Tuberculosis experiments at Imperial Bacteriological Laboratory, Muktesar.

The Khan Sahib had discharged on two occasions duties of the Farm Superintendent, Izatnagar Estate, for a considerable period in addition to his own, and he is fully acquainted with the modern agriculture.

He was a very capable organiser, most zealous and exceedingly competent all-round officer. In his bearing and manners he was a very superior type of individual, and very loyal to his superior officers.

The good services rendered by the Khan Sahib was placed on record in the annual reports of the Imperial Bacteriological Laboratory, Muktesar, for the years ending March 1921, 1922, 1924 and 1925.

Joining service as a Veterinary Assistant, second grade, in the Imperial Bacteriological Laboratory at Muktesar in the year 1904, the Khan Sahib was soon promoted to the post of Veterinary Inspector. In the year 1919, he was promoted to the newly created post of Senior Veterinary Inspector on Rs. 150—10—200, and to the newly created selection post of Senior Veterinary Inspector on Rs. 250—10—350 in the year 1921.

He was promoted to the newly created gazetted post of Veterinary Deputy Superintendent, Imperial Veterinary Research Institute, Izatnagar, in the year 1930, on Rs. 250—25—550—25—750, by the recommendations of the Public Service Commission (India).

He was awarded Silver Jubilee Medal in the year 1935, title of *Khan Sahib* in the year 1937 and Coronation Medal in the same year by the benign Government in recognition of his meritorious and long services.

The Khan Sahib rose to the present status by dint of his self exertion and merit. His career is an inspiration to the younger generation who have adopted the veterinary profession.

[We wish the Khan Sahib long life and all happiness to enjoy his well-earned rest and pension.

Now that the Khan Sahib has retired after giving his best to the service, we do hope he will give his best to the profession by becoming an active member of the All-India Veterinary Association and an enthusiastic contributor to its organ, the *Indian Veterinary Journal*—EDITOR, *I. V.J.*]

Clinical Articles.

GASTRO ENTERITIS AMONG CATS.

BY

M. R. CHENGERI, G.B.V.C.,

Veterinary Officer in charge,

Tasker Veterinary Hospital, Bangalore.

About the beginning of August 1938, a number of cats were brought to the Veterinary Hospital for treatment. Symptoms in affected animals showed great prostration, offensive diarrhœa often of a dirty green colour, increased pulse, mucous membranes highly injected, furred tongue, high temperature, abdominal enlargement, tenderness, disinclination to move. In some cases frequent vomiting and intense thirst were found.

Symptomatic treatment was adopted by giving Astringent powders and Opiate enema but with no benefit, and several of them died.

The post-mortem examination showed gangrenous ulceration of the stomach and bowels, and in some cases diffused patches of gastric and intestinal inflammation with extravasated blood spots.

Prontosil ½ to 1 c.c. (5 per cent solution) was tried and showed miraculous improvement and the results were cent per cent cure. The good results obtained in these affections suggest adoption of the treatment in similar affections in cats and dogs also.

I gather from Dr. P. M. Narainswamy Naidu, Research Officer with the Government of Mysore, that this epidemic disease is a common occurrence among cats in England and Germany due to some Streptococcal infection.

Prontosil is well tolerated when administered subcutaneously.

METRORRHAGIA IN A GREAT DANE BITCH.

BY

K. R. S. AIYAR, G.B.V.C.,

AND

J. P. DAMRI, G.B.V.C.,

Bombay Veterinary College.

Uterine hæmorrhage, independent of the œstral cycle in bitches, is a rare complaint. Various causes are responsible for it and the treatment, therefore, depends on the causes. This paper deals with a peculiar case, the cause, as found by us, has not been, to our knowledge, hitherto recorded in case of Metrorrhagia. The long duration during which the bitch was suffering from this complaint, and the various medical and surgical treatments which were more or less symptomatic and which the animal had during this period, make the case interesting.

The patient under report was a six-year-old Great Dane bitch, weighing about 120 lbs., the property of one Miss K., with a history of having aborted at the fourth pregnancy somewhere in August 1937. It was brought for treatment at the Bai Sakarbai Dinshaw Petit Hospital for Animals, attached to the Bombay Veterinary College, in November 1937, with profuse uterine hæmorrhage. It was then brought to our notice that the bitch, immediately after the abortion, was attended to by a Veterinary practitioner and that for a long time there was nothing to complain of in the health of the bitch, except that on one occasion prior to its being brought to this hospital there was a slight hæmorrhage.

Examination of the vagina revealed the presence of a soft wart-like growth on the roof of the vagina and oozing of blood from the os. Extra-abdominal pressure on the uterus discharged clots of blood from the uterus.

Various measures as douches of Acriflavine in strengths from 1 in 5,000 to 1 in 1,000 one-per-cent Dettol, Entozon Bougie, intravenous injections of Neo-Hemoplastin, P. D. & Co., and

Calcium Gluconate, subcutaneously Pituitrin and Adrenalin and orally Ergot, Calcium salts, etc., though they brought about a great lessening of hæmorrhage, were always followed by metroblennorrhea. During this interval X-ray of the region from the pelvis to the epigastrium was taken for the presence of any calcified growths, etc., but the reading was negative for any such growths.

Curetting did not lessen hæmorrhage but increased the same.

Having failed to control the hæmorrhage by the measures stated above, an examination of the blood and stools was made. No abnormality was found in the stools but, to our surprise, numerous microfilaria were seen in the blood. We had decided to conduct hysterectomy if the blood examination were to be negative but, after this finding, we abandoned this idea.

Though uterine bleeding is not known in animal microfilariasis, it was taken by us as a rare finding and we decided to treat the case for Microfilariasis. The absence of temperature throughout illness of the patient, its good appetite and its maintenance of a tolerably good condition in spite of hæmorrhages, which, though interrupted, were frequent—all these helped us in our decision. (Catheterised sample of urine did not show any abnormality and uterine discharge did not show the presence of microfilaria.)

A course of Fouadin was given to this patient (American Veterinary Surgeons claim to have obtained good results with this drug in heart worms in dogs), but that did not entirely cause the microfilaria to disappear from the circulatory system, though it diminished their number. It was noticed that during this treatment the hæmorrhage as well as the mucous discharge from the uterus were lessened considerably and the animal improved in condition and put on weight.

Encouraged by these results, it was decided to push on the treatment for complete annihilation of the microfilaria by intravenous introduction of Antimosan in gradually increasing doses. After six injections there was a complete absence of microfilaria and to this day they have not appeared again. We have omitted

here the particulars of administration of Fouadin and Antimosan because the adjustment of dosage and the intervals between injections depended more on the incidence of hæmorrhage, general health of the animal, and last but not least on the reaction of the heart and after-effects of the drug used. Fouadin is safer than Antimosan but the latter is more effective than the former.

Subsequent to these injections, the hæmorrhage and the mucous discharge ceased, and the animal ate well, and put on flesh. The case was discharged in the early part of February 1938, with instructions that the bitch should be brought bi-weekly for six more injections of Antimosan. This treatment was completed during the latter part of February 1938.

Unfortunately, during the last week of February there were slight uterine hæmorrages; and this time a course of injections of "Antuitrin-S," P. D. & Co., was suggested with a view to creating a healthy endometrium free from the tendency to bleed. The bitch received nearly eight injections each 1 c.c. By the end of first week of March the owner reported the bitch to be in good condition without any recurrence of hæmorrhage. It was therefore decided to cease treatment and give complete rest to the animal. This was partly due to success and partly due to having exhausted all avenues of treatment except knife.

As ill luck would have it, the owner turned again at the Hospital during the early part of the last week of March 1938, stating that there was again a serious hæmorrhage and that this time the bleeding had caused a severe depression and fatigue to the animal and therefore suggested either surgical remedy or destruction of the bitch.

After admission into the Hospital, successive hæmorrages and accumulation of clots in the uterus caused a rise of temperature with signs of sepsis. Antiseptic douches brought down the temperature and on 30th March 1938 hysterectomy was performed under Sodium Evipan anæsthesia.

An incision on the linea-alba extending from the brim of the pelvis, nine inches in length anteriorly, was taken. The uterus was

located and removed with the ovaries under great aseptic precautions and in the usual manner. This operation in all took 45 minutes.

The after-effects of Sodium Evipan was not severe in this case and by two hours the bitch was on her legs from the recumbent position.

FIG. 1.

On the third day after the operation the wound was found to be wet and smelling urine, the latter having been absorbed by the cotton wool of the dressing and transferred to the wound. This necessitated the removal of four interrupted stitches of the external wound and daily dressing with two per cent aqueous solution of mercurochrome. The wound now has almost healed (Fig. No. 1) and is likely to take a week more for complete healing.

Lesions Found in Uterus.

The uterus particularly the right horn was flabby congested and thickened. On cutting open the body and the horns of the uterus, the right horn was considerably thickened containing a large amount of muco-saneous discharge. Floating almost in it

was the remains of a small piece of a fœtal skull, the size being just about a half of sparrow egg shell and as sharp as a scalpel. (Fig. No. 2). This was clearly the *root cause of the uterine hœmorrhage extending to a period of nearly six months.*

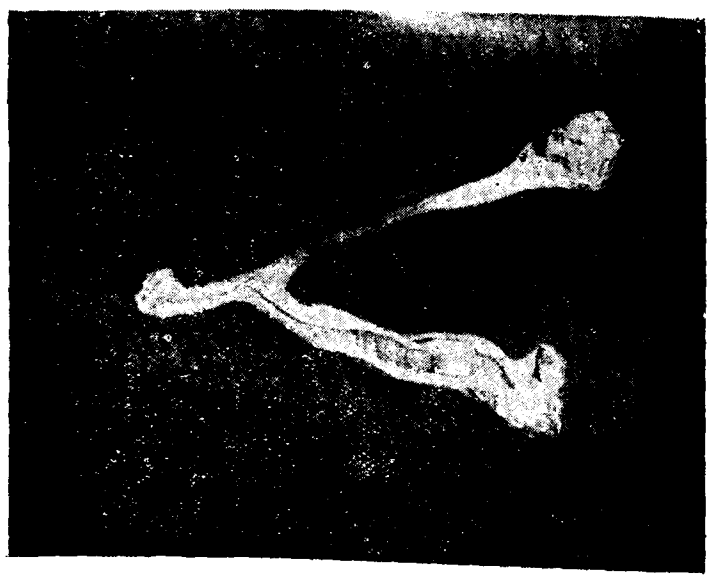


FIG. 2.

It is rather curious how a fœtal skull remained such a long time without causing sepsis though the skull must have had the muscular and epidermal coverings. The only cause of escape from sepsis must have been due to the constant douchings received. These must have softened and drained off the appendages from the skull leaving the osseous remnant to do the damage.

We cannot finish this paper without thanking the Principal, Mr. Mohey Deen, M.R.C.V.S, for all encouragement and facilities given, and Mr. M. K. Garudachar, G.B.V.C., for helping us during the operation.

Sir Frederic Hobday, F.R.C.V.S., while in Bombay, during his sojourn in India to attend the Indian Science Congress Jubilee Session, held at Calcutta in January 1938, was shown this case.

EPISTAXIS IN A DOG.

BY

K. S. SHETTY, G.M.V.C.,

Assistant Veterinary Surgeon, Narayanaguda, Hyderabad (Deccan).

Subject.—A two-year-old Great Dane.

History.—On the 31st of May 1938, at 9 P.M., I was called to attend on a Great Dane which was said to have been in sound health till that evening and, at about 7 P.M., profuse bleeding from only the right nostril was observed by the owner who had tried to arrest the bleeding by applying an ice bag on the head but without success.

Treatment.—1 c.c. of Pituitrin was injected subcutaneously and the bleeding stopped in about ten minutes. It started again in about half-an-hour later, when the bleeding was more profuse than before and the animal was almost in a state of collapse. I gave an injection of gr. 1/100 of Atrophine Sulphate dissolved in 1 c.c. of distilled water and waited for about quarter of an hour but the bleeding did not stop. I then gave intramuscularly 5 c.c. of Hæmostatol Anti-hæmorrhagic serum, manufactured by the Bengal Chemical and Pharmaceutical Works, Ltd., Calcutta. This had the desired effect and the bleeding stopped in about ten minutes or so and did not recur. On the following morning, another injection of Hæmostatol was given because of a few drops of blood issuing from the nostril at intervals. The dog made a complete recovery after the second dose of the serum.

SODIUM CITRATE IN CASES OF CHRONIC TYMPANITIS AND RUMINITIS IN CATTLE.

BY

S. SESHADHRI IYENGAR, G.M.V.C.,

Veterinary Assistant Surgeon, Mayavaram, Madras.

These two complaints are usually met with in cattle of this presidency. Sometimes they tax the poor veterinarians in

treating the same and giving relief to poor animals. When I was discussing with my District Veterinary Officer, M.R.Ry. R. Swaminatha Iyer A.vl., in December 1936, he advised me to try Sodi Citras and Sodi Chloride injections intravenously.

I have tried this injection in four animals and the treatment was very successful :

CASE 1. A bullock, aged seven years, brought from a distant village with the complaint that there is flatulency for 20 days. The symptoms shown were dullness, slight rise in temperature and tympanitis and also ruminitis. Rumination suspended, animal did not pass dung and small hard pellets of dung passed out after an enema and animal had lost much of its condition. At first oral medicines were administered for a few days but it was of no use. This animal was given one drachm each of Sodi Citras and Sodium Chloride (chemically pure) intravenously in 40 c.c. of Aqua Distillata at body temperature.

There was much improvement on the third day and all oral medicines were stopped. A week after the first injection another injection was given with the same dose. The animal made an uneventful recovery.

CASE 2. A heifer calf, aged two years, brought with a history that there was bloating of abdomen for the past ten days and quack treatment was given in the village. The animal was gasping for breath. No dung passed after enema; there was slight rise in temperature and flatulence on the left side. Paracinthesis abdomonis was performed to relieve the animal of his hard breathing. Oral medicine was given. The next day the rumen again bloated.

Half-a-drachm of Sodi Citras and Sodium Chloride in 30 c.c. of water was given intravenously. The animal appeared a little lively on the next day, but flatulency did not disappear. Again the rumen was punctured to let the gas escape on the fifth day of the commencement of the treatment and

another injection with the above dose was given. The animal made a good recovery.

CASE 3. A heifer calf aged 1½ years brought to the dispensary in a cart as it was not able to stand. The animal was suffering from ruminitis and it was in a semi-comatose condition. Intravenous injection of half-a-drachm of Sodi Citras and Sodium Chloride was given in 30 c.c. of water. No other treatment was given.

The animal made an uneventful recovery on the third day.

CASE 4. A cow aged six years—a calf at foot. It was suffering from flatulency for ten days previous to my treatment and all quack treatment had been given. When the animal was in worst condition it was brought for treatment. The abdomen was very much bloated and I was afraid that rupture of some internal organs may happen if it falls down suddenly. The circumference of the abdomen was about 6½ feet. Both flanks were bloated. Rumen was punctured with a trocar and gas allowed to escape. One drachm of Sodi Citras and a drachm of Sodium Chloride was given intravenously in 40 c.c. of water. No other oral medicine was given. There was slight flatulency on the second day and the animal was able to drink some *conjee*, and it was all right on the fourth day and it was discharged cured.

I bring this to the notice of my professional brethren to try this treatment. The dose may be increased slightly according to the size and weight of the animal. The usual average weight of animals in these parts is about 450 lbs. This treatment, suggested by Mr. R. Swaminatha Iyer, is really an efficacious one. My best thanks are due to him for suggesting this treatment.

———

PURPURA HÆMORRHAGICA IN EQUINES.

BY

D. T. PARMAIK, G.B.V.C.,

Reserve Veterinary Assistant Surgeon, Poona City.

Subject No. 1.—A piebald, country-bred, entire, aged about 18 years.

Subject No. 2.—A chestnut, country-bred, entire, aged eight years.

Subject No. 3.—A bay, country-bred, entire, aged about 18 years.

The above three cases were treated at the above hospital successfully.

Subject No. 1.—The horse was admitted in the hospital at 4 P.M. on 23rd December, 1937, as an out-door patient with the following history and symptoms :—

The horse was in its normal health the previous night. On the morning of the 23rd he was found with a swelling on the muzzle which extended up to the eyes which were closed. Eyelids were puffy and œdematous. Both the lips were œdematous and were held open. There was dribbling of saliva. Serosanguineous discharge flowed from the eyes; and also there was an exudation, in droplets, of serosanguineous fluid on the surface of the nostrils and muzzle, which showed no tendency to coagulate. The swelling was cold and pitted on pressure. A distinct groove in the swelling was seen caused by the head-stall rope. Respiration was hurried, difficult and slightly stertorous. Temperature was 103° F.

Diagnosis :—Though the symptoms were characteristic of Purpura hæmorrhagica still we failed to notice any petechiæal hæmorrhage on any of the visible mucous membrane, nor were there any swellings on the dependent parts of the body.

Treatment.—Soap water enema.

℞

Sodii Salicylas	4 dr.
Soda Bicarb	4 dr.
Jaggery	Q. S.

Mft. electuary.

Sig. To be smeared on the back part of the tongue.

Liq. Adrenaline Chloride (Parke Davis & Co.) (1-1,000) was given subcutaneously.

After three hours the horse was in a serious condition. Œdema on the gums appeared, head hanging down, great distress, respiration very difficult and stertorous. Under the circumstances the prognosis appeared to be unfavourable

At this stage an injection of Lugol's solution was given intra-trachially and Liq. Adrenaline Chloride (1-1,000) was repeated.

Eyes were irrigated with warm boric lotion and yellow ointment was applied.

Next morning (24th December, 1937), we noticed that the swelling was reduced to some extent and some improvement was seen in the breathing, too. Still the animal was completely off-feed, but the temperature was normal.

Treatement.—The injections were repeated in the morning. No electuary was given. The eyes were treated as before.

Diet.—The horse was not able to eat anything though chopped lucerne, bran mash, rice-*conjee*, pieces of cocoanut and jaggery crushed together were placed before him. He drank only a little quantity of water.

The injections were repeated in the evening as the animal was showing improvement.

On the 25th of December, 1937, the animal showed marked improvement in respiration and had an inclination to eat. The

swelling of the face was reduced to a great extent and the eyes could be opened to some extent.

Treatment.—The injections were repeated in the morning only and the eyes were dressed as before.

On the 26th of December, 1937, the swelling had practically disappeared from the face. Eyes were half-open, opacity of the cornea of both of them was noticed. There was an ulcer on the right cornea in addition. Large sores were noticed on the muzzle and on the region surrounding the eyes as a result of severe sloughing and pealing of the skin. The animal's appetite improved and it ate some lucerne and drank sufficient quantity of water.

Treatment.—Only Lugol's solution was injected in the morning. Sores were cleaned with Hydrogen Peroxide and dressed with Boric ointment and protected with bandage. Eyes were irrigated with warm Hydragiri Perchloride Lotion (1-5,000) and insufflated with calomel.

On the 27th of December, 1937, the swelling on the muzzle, etc., had completely disappeard but there was very slight inflammation round the eyes. The right eye-ball was found ruptured and the lens, etc., were protruding. The right eye-ball was excised and the left one was treated as before.

On the 28th of December, 1937, the animal's appetite was normal and it looked all right in other respects, but the poor creature lost his right eye.

Treatment.—Dressing of the eyes was repeated, sores were cleaned with Hydrogen Peroxide and dressed with the following powder and protected with a bandage :—

℞

Zinc oxide	1 part.
Boric Acid	2 parts.
Tannic Acid	½ part.

Mft. Pulvis.

Sig.—To be dusted over the parts.

Conclusion.—We had three cases of this disease in which symptoms were exactly alike. In every case the swelling originated at the muzzle and there was no swelling on any other dependent part of the body. All the three cases responded very well to Lugol's Solution and Liq. Adrenaline Chloride injections, but the sores that were left behind after the subsidence of the swelling, took some time to heal.

Subject No. 1 was brought to the hospital while it was in a precarious condition.

Subjects No. 2 and 3 were brought to the hospital as soon as they began to show symptoms.

Subjects No. 2 and 3 did not develop eye trouble as in Subject No. 1.

It is interesting to note that the above three horses were owned by one man who had sold them to different men at different places in the City. Each of these horses was handled by the same man, on the previous night of the attack. It was also reported that that man is a horse dealer, hence the question arises if he was responsible in any way to what happened to the horses in question.

I am very much thankful to Mr. R. N. Naik, Veterinary Investigation Officer, Bombay Presidency, who was kind enough to examine the smears, blood and discharges from the noses and eyes of these horses. He could not find anything which could be ascribed as the causative agent of the condition described.

It is my duty to express my grateful thanks to Dr. T. R. Khaledkar in charge of the Hospital for his guidance and help.

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A CASE OF PURPURA HÆMORRHAGICA IN A HORSE.

By

H. V. KULKARNI,

Assistant Veterinary Surgeon,

General Veterinary Hospital, Baroda.

History.—A brown gelding, aged about 16 years, was admitted for fever into the General Veterinary Hospital, Baroda, on 15th February 1938. The animal belonged to one of the Regiments of the Baroda Army.

Symptoms and Treatment.—On the day of admission, it had severe Coryza and appeared to be improving with adequate treatment. But on the 3rd March 1938, it suddenly developed swellings on the dependent parts. They were diffused and œdematous in character, slightly hot to the touch and pitted on pressure. Temperature recorded was 102° F. The animal was off-feed and was constipated. It was then immediately isolated and kept in a well-ventilated fly-proof stall under observation. An electuary containing Mag. Sulph. and stimulants was administered and soap water enema was given. Astrigent lotions were applied to the swollen parts.

On the 4th morning, the swelling on the dependent parts was found decreasing in some parts and increasing in others. A fresh swelling beginning at the nostrils and lips was observed with blood-stained discharge from the nose. The eyes were blood-shot with petichial markings of scarlet purple colour on the conjunctiva. The animal was standing persistently in a corner with its head hanging down. Breathing was difficult due to the swelling of the nose and lips. Temperature was 102° F. with feeble pulse, and prehension was interfered with due to swollen lips and it was not able to drink water. The case was diagnosed as one of Purpura Hæmorrhagica and the following treatment was adopted: 20 c.c. Lugol's solution was slowly injected intra-trachially. The electuary and the enema were repeated. By the evening of the same day the swelling of the nose extended upwards involving the whole face, giving it an appearance of the head of

a hippopotamus. There was marked dyspnœa and the animal was in a most depressed condition. The injection was again repeated with the rest of the treatment.

On the 5th morning, the swellings were much worse. The animal had great difficulty in breathing and was *in extremis.* The injection was again repeated. At about 10 P.M., it showed symptoms of spasmodic colic which was treated by giving an enema of chloral hydrate solution and the animal was temporarily relieved of the spasms of the bowels. Nothing could be given per orum due to the excessive swelling of the nose and the mouth. The animal, however, died at 1 P.M. that day.

CASES OF PURPURA HÆMORRHAGICA AS SEQUALÆ TO EQUINE INFLUENZA.

By

MD. TAKIUDDIN AHMED, G.V.SC.,

1st (N. O.), H. I. S., Lancers, Veterinary Hospital,

Hyderabad (Deccan).

In a very recent outbreak of Equine Influenza among the horses of Hyderabad Regular Forces, a few really interesting cases were observed. Among these one developed Pneumonia and three Purpura Hæmorrhagica. Among the three that had Purpura Hæmorrhagica, one was under my care in the "A" Battery, Nizam's Horse Artillery. In view of the rarity with which such cases are met with and the unusual course which this particular case ran, I intend to record the following lines:—

The horse No. 74, a Chestnut Australian gelding, of the Right Section of the "A" Batterry, was on the list of the Catarrh cases. On the evening of 14th February 1938—third day after commencement of the illness—he was seen to be stiff in his posterior limbs, with considerable swelling on the inner sides of the thighs, and a temperature of 103° F. He was fomented and massaged with stimulating liniments, but the swellings increased and the following day he was unable even to stir an inch. The

temperature had fallen considerably, so, on the following day an aperient was administered. The temperature again shot up, rising to 105° F. On the following morning, *i.e.*, on the 18th February, œdematous swelling appeared on the lower parts of the belly, sides of the neck and the body. The sheath had increased to the size of a large pumpkin though the animal could still urinate. The mucous membranes were of a brick-red colour with a few petichæ sprinkled over the nasal mucosa. There was blood streaked discharge from the nostrils. The limbs were very stiff.

The animal was segregated in an open field under the shade of a tree. Injections of Omnadin (Bayer's) 10 c.c. were given on alternate days and continued throughout the illness with much benefit, till the temperature had distinctly fallen to normal and remained thereat for two days. Daily injections of Adrenaline (3 to 5 c.c.) with or without Normal Saline were also given till the swellings went down. A febrifuge bolus was given twice daily with the following ingredients:—

Quinine Sulph.	1 dr.
Sodi Salicylas.	4 dr.
Camphor.	1 dr.
Potass Iodide	1 dr.
Pulv. Digitalis.	15-30 gr.
Soda Bicarb.	2 dr.

This recipe was adhered to with slight variations in the later stage by omitting Digitalis and reducing Quinine Sulph. and Salicylate to half their quantity and adding half-a-drachm of Pulv. Nucis Vomicæ.

The swellings were fomented twice or thrice daily and massaged with stimulating liniments. All the swellings cleared up with the injections and fomentations, but that of the sheath persisted. Then application of saturated solution of Magnesium Sulphate was tried. Distinct benefit was noticed and in a week's time that too cleared up completely. The stiffness had disappeared in a week's time and the temperature also came to normal in a

fortnight. By the first week of March the swellings cleared up completely and the animal became all right. Omnadin was stopped when the temperature had reached normal. After that, treatment consisted in the administration of one drachm of Pot. Iodide daily in feed for a week. Since the summer was becoming severe day by day and the animal had passed through the period of its illness and required only a prolonged period of rest it was returned to the Lines with strict directions to get it to its daily routine work gradually after a month

I cannot close the article without expressing my thanks to my Principal Officer, Capt. M. Karamullah Khan, M.R.C.V.S., B.V.Sc., who so kindly rendered me much help in giving useful hints throughout the outbreak and in the treatment of this particular case.

A CASE OF PURPURA HÆMORRHAGICA.

BY

S. D. RAI SHARMA, G.B.O.V.C.,

Veterinary Assistant Surgeon,

Rawalpindi Area, Mirpur Depot, N. W. F. P.

Subject.—A black, T. B. English Stallion, born 1932, brought from plains to hills for summering.

History.—The stallion in question developed Strangles on the 11th May 1938. The submaxillary abscess was opened on the 18th May 1938. When the wound was healing, another abscess developed on the off side and this was opened on the 28th May 1938. The wounds were healing and the temperature was normal. There was a slight œdema of the hind fetlocks due to lack of exercise. On 30th May, swelling of the hind fetlocks extended further upwards and became well defined; there was also a rise of temperature. On the morning of 31st May, the following symptoms were exhibited:—

Symptoms.—Off-feed; temperature 101° F. in the morning and 102° F. in the evening; all the visible mucous membranes showed

petechial spots varying from a pin's head to a four anna piece; œdematous swelling of the lips and muzzle extending up to the face and eyelids and also on scrotum and sheath; swellings on all the limbs, which were marked and had well-defined contours, extending up to the shoulders in the case of fore limbs and hips in the case of hind limbs. The swellings were cold, painless and pitted on pressure. The animal was dull and moved stiffly and with difficulty He would not lie down, perhaps he could not, on account of enormous swellings. The skin at heels was moist and fissured.

Treatment.—(*a*) *Dietetic.*—Bran and linseed mashes, boiled barley and scalded oats, green lucerne and finely chopped oat hay and Dhoob grass were given. Feeds were offered frequently and always freshly prepared to tempt him. He was often hand-fed as he refused to eat from the manger. Fresh and clean supply of cold water with Mag. Sulphas 2 ozs. and Pot. Nitras 1 dr. given *ad libitum* twice daily. Thorough cleanliness, and hygienic conditions were looked upon as indispensable.

(*b*) *Medicinal.*—(*i*) Hypodermic injection of Adrenalin Chloride solution (1·1,000 P. D. & Co.) 4 c.c. was given.

(*ii*) Intra-tracheal injection of Iodine:—

Iodine	1 part.
Pot. Iodide	5 parts.
Aqua distillata	100 parts.

Injected 20 c.c. intra-tracheally, care being taken to inject with strict asepsis.

(*c*) Fissured heels dressed with white lotion and dry powder.

1st June 1938. Temperature 100·4° F. in the morning, 102° F. in the evening. The petechial spots in the mucous membranes much improved, œdematous swellings of the scrotum and sheath increased, that of the limbs constant. Respirations, pulse, fæces and urine normal. Treatment continued.

2nd June 1938. Temperature 100·4° F. in the morning, 102° F. in the evening. The petechial spots in all the mucous membranes disappeared; œdematous swellings of the muzzle, scrotum and sheath reduced; that of the limbs constant. Treatment continued.

3rd June 1938. Temperature normal, a copious blood stained discharge from the nostrils in the morning, visible mucous membranes normal, œdematous swelling of the muzzle and face disappeared, that of the limbs and scrotum and sheath much reduced. Feeding well. Treatment continued.

4th June 1938. Temperature normal, nasal discharge lessening, œdematous swelling of the scrotum and limbs further reduced. Feeding well.

5th and 6th June 1938. Temperature normal, nasal discharge stopped, œdematous swelling of the scrotum, sheath and limbs same as on 4th June 1938. Feeding well.

Iodine and Adrenaline discontinued. Injection of Nuclein 10 per cent (Vety. P. D. & Co.) 5 c.c. given subcutaneously daily.

7th June 1938. Temperature normal, œdematous swelling of the fore limbs, scrotum and sheath disappeared, that of the hind limbs much reduced, mucous membranes normal. Feeding well. Nuclein continued.

8th, 9th and 10th June 1938. Normal except for the fissured heels which are dressed twice daily, and slight œdematous swelling of the hind limbs.

11th Juue 1938. Normal all round save the slight fissured heels, light exercise ordered, tonics in food given. He was kept in segretation up to 12th July 1938, and was discharged as cured.

I am highly indebted to Major J. J. Clune, R. A. V. C., under whose guidance the whole treatment was carried out and by whose kind permission this article has been published.

Extracts.

NON-SWEATING IN HORSES

By Major C. M. Stewart, R.A.V.C.

The thoroughbred horse, whether on the race-course or at stud, has always been an object of the greatest possible interest to me. It is therefore understandable that when I came to India the condition known as " non-sweating " or " dry-coat," affecting racehorses, early occupied my attention. In the course of my service here I have been fortunate in having had rather more experience of it than is usually possible for an officer in the Corps, and in putting down the results of this experience I do so with the idea in mind that they may perhaps be of service in any subsequent investigation which may be undertaken. There is no doubt that little of real value is known about the condition, despite the fact that its existence has been recognised for many years. In the following pages I make no claim to have solved the problem, but hope that I will bring out some points of interest not previously noted.

My earliest introduction to non-sweating was in the North of India, where, in 1922, when I began to take an interest in racing, I saw several horses running which had been sold in Bombay and Calcutta on account of being so badly affected there as to be almost useless as racehorses. It was known then that in the dry, bracing climate of the North, horses so affected frequently improved to such a degree as to be capable of being raced without exhibiting the distress shown in the warmer and damper climate of Calcutta and Bombay. A better knowledge of the condition led one to understand why this should be so. The published literature on the subject was then, and still is, scanty and of doubtful value. I was, however, able to obtain a report by A. D. MacGregor, I.V.S., which, though contributing a description of the outward and visible symptoms, did not suggest any cause or contribute anything of real value in treatment. The author did, however, suggest the use of nitro-hydrochloric acid dil. in normal doses, but did not explain, at least to my satisfaction, why he did so. Actually in individual cases this treatment gave rather promising results for a short time, but it soon appeared to loose its effect. A later publication by the same author, in which he endeavours to connect non-sweating with kumri and osteoporosis, is, to me, hardly convincing.

The condition is one which is usually seen in racehorses, *i.e.*, in animals doing fast work on a highly concentrated ration. It is associated with warm, damp climates such as are found in the large coastal cities of India, and in Ceylon, Singapore and the West Indies. It is not well known in South Africa, where most of the racing takes place under good climatic conditions, but, when the writer was asked to look at a horse in Johannesburg recently which was supposed to be suffering from a heart affection, he found that it was a typical non-sweater. It had presented these symptoms after a short stay in Durban, where at

times the climate is oppressively moist and resembles that of Bombay. I have no knowledge of its occurrence in Australia, but have it on excellent authority that it occurs in England, though the symptoms are not so marked as one sees in India. It has, however, been observed in horses landed in Bombay after a hot voyage through the Red Sea.

Writers on the subject have expressed the opinion that premonitory symptoms are absent, but I think that this may not be correct—at any rate—in every case. I believe that premonitory symptoms in a potential non-sweater may be as follows :—

1. A distension of the nostrils, noticeable during a hot day in the stable or perhaps in the close atmosphere of a railway horse-box, which is much more marked than with other horses in the same stable or horse-box.

2. A tendency to delayed sweating in which the skin shows a condition of vascular engorgement unaccompanied by sweating when other horses under similar conditions are sweating freely. In India such horses are known as " suppressed sweaters ". In these cases, however, sweating may be noticed after a short interval has elapsed. Such cases presenting these symptoms may remain at this stage under favourable climatic conditions, or may become perfectly normal, but if removed to a hot, moist climate may develop into partial or complete non-sweaters.

The symptoms proper as seen in a climate which predisposes to the conditions are, then, a gradual diminution in the secretion of sweat under conditions of exercise which should bring about free sweating in a normal horse, combined with a loss of coat colour which is well marked in connection with all colours except grey. Here I would like to remark that, while there has been considerable discussion and expression of opinion regarding this point of colour, I have not been impressed by the special susceptibility of horses of any particular colour to this condition. All are, I think, equally susceptible. Naturally, one sees more bays affected because horses of this colour constitute the great preponderance of race-horses.

At first the reduction in sweating appears to affect the loins and hind quarters where the skin is thick and more closely adherent to the underlying tissues, but the condition spreads until the only parts where sweating may be noticed are those under the saddle, girth and bridle, where the reins rub the neck, between the hind legs, and where the tail rubs the buttocks in side-to-side movement. A slight dampness of the fold of skin at the flanks and around the throat is also seen, and a similar dampness along the roots of the mane is seldom absent even in the worst cases.

With the appearance of these symptoms there is an increased scurfiness of the skin all over the body, and frequently a loss of hair affecting especially the skin of the face and, to a lesser extent, of the neck and shoulders. Itchiness is occasionally seen, but where intense

itchiness has been described it is, I think, possible that there may be a concurrent *lichen tropicus*. Intense itchiness has certainly never been a feature of the cases seen by me, where *lichen tropicus* was absent.

I have noticed with regard to this train of symptoms that, as the horse becomes fit from continued work, sweating becomes less marked, but that the same horse, after having been let down and put back into work again, will raise the hopes of his trainer by sweating to some extent, but only to repeat the performance of sweating less with increasing fitness. After a gallop the vascular engorgement of the skin in these cases is always seen.

These symptoms bring in their train the respiratory distress which is such a marked feature of the condition. The excess heat engendered when a racehorse is at work is normally got rid of through evaporation of sweat from the body surface assisted by the increased action of the lungs. When sweating is in abeyance the temperature rises with work to 105° or 106° F., and does not become normal until several hours later. While there are other factors involved, it appears obvious that the excess heat and waste products must be got rid of in some other way than through the skin, and the result is that the animal remains in a state of diminishing respiratory distress until this is done. In really hot weather, however, the same features are noticeable, though to a less degree, during the heat of the day, though no exercise has been taken. The body temperature in some such cases has been found to go up to 104° F. or more. The respiratory distress seen in these cases is almost painful to watch. The head is frequently depressed, the nostrils widely distended, and the respirations are sometimes as many as 90 to the minute. A slight yellow tinge of the visible mucous membrane is frequently seen, and the droppings are often rather dry and hard. Appetite in the cases, which have come under my observation, has been fairly normal. It is a well-known fact in India that many horses affected in this way eventually drop dead, usually after completion of a race or gallop.

As racehorses, the animals affected present training problems to those who have them in their charge. The trainer bases his work and estimation of a horse's fitness largely on the condition of the respiratory function, the character of the sweat and the degree of musular swelling over the back and loins after fast work. By none of these methods can he judge a non-sweater's fitness, and I am unable to see how he can ever be certain that he has such a horse 'at his top'. There are, of course, means at his disposal of being sure that he is in sufficiently good condition to win a race, though trials held in the early morning when it is cool are a poor guide likely to form in the afternoon of a Calcutta monsoon or Poona race-day. It was usually found that horses with this complaint lost form and deteriorated as racehorses, though, with treatment, the reverse was frequently the case.

My more intimate study of non-sweating began after I had been some time at the Army Veterinary School in Poona. My predecessor—Major Hayes—had used thyroid extract, etc., in an experimental way,

but was not impressed by the results. He thought that saline twice a week was the most satisfactory treatment he had tried. I watched cases treated in this way for a little time, and noticed that, though there was improvement, it was not very marked. Being curious as to the condition of the stomach, I began to wash them out by the syphon method, using large quantities of saline solution, and it was noticed that in every case a considerable amount of "wind" was removed and that after somewhat prolonged syphoning operations quantities of thick, whitish mucus began to return through the tube. This was of such a density as to block the tube on occasions and could easily be picked up and held in the fingers without slipping through them. It came back in such quantities that the whole surface of the saline contents of a stable bucket was thickly covered with it, though in the course of an hour or so it dissolved and disappeared. I persisted with this treatment and, for a time, used a solution of soda bicarb, with the object of loosening the mucus more quickly, but abandoned this when it became obvious that the cases were not doing well. It was then that I recalled the claim to benefit by treatment with nitro-hydrochloric acid dilute. I believed that :—

1. The mucus removed by syphonage and probably cloaking the acid-secreting glands of the stomach and that saline, given for retention, freed the glands temporarily from this obstruction and allowed them to act normally.

2. The reason for the temporary success of the acid treatment before mentioned may have been that it supplied a need which was not being met in a normal way, for the reasons given.

3. That by syphonage and the use of alkaline, I reduced by its removal or neutralization, the already low acid content of the stomach, and thereby produced a condition detrimental rather than beneficial to the patient.

I found that a stomach washed out on one day would produce an equal quantity of mucus on many successive days, apparently indicating that there was present a chronic inflammation of the mucus-secreting glands. From this I realised that occasional removal of the material would be of only limited benefit, and from that time commenced the regular daily saline treatment of such cases after morning work but before feeding, the object being the daily removal of the mucus without at the same time removing residual acidity of the stomach. The dose given by funnel through the stomach-tube was two ounces each of common salt, magnesium sulphate and sodium sulphate dissolved in a gallon of cold water. It was given cold in order to obtain its retention in the stomach for as long as possible. This mixture was used in the hope that it might also be of benefit in combating the slight constipation and jaundice noticed in these cases.

The comparative success of the treatment, which was not measured by any marked increase in sweating, but rather by an increase in general well-being, would appear to be sustained by two rather out-

standing cases, though the benefit in general was well marked. One of these commenced treatment when handicapped for racing at 8 st 4 lb. in Class III and was eventually a winner at 8 st. 6 lb. in Class I. The other commenced when about 7 st. 8 lb. in Class III and later became a good winner in Class II. Both these horses did much better in Poona, when under constant treatment, than in Bombay. No ill-effects were ever noticed from continued treatment along these lines.

The next step was to obtain some dry-coated horses for autopsy in order to get an histological report on the condition of the skin and other organs. Several such carcases became available as the result of an order from the Secretary, W. I. T. C., made at my request that race-horses dropping dead on the course, whether at exercise or after a race, were to be taken to the Army Veterinary School. The feature which stands out at these and other examinations was undoubtedly the constant enlargement of the spleen. This varied from 6 to 24 lb. in weight and, except in the case of one dry-coated ex-racehorse in the Bombay Body Guard, the organ was firm in consistence. In the exceptional case the organ weighed 10 lb., but was flaccid as if the capsule had for some time been enlarged and thickened and the substance had subsequently decreased in amount, leaving the capsule too big for its contents. Histological thickening of the trabeculæ and considerable deposits of pigment have been described in these cases.

The liver appeared normal macroscopically, but histological examination of sections showed that there was marked cirrhosis of the portal tracts, passive venous congestion and round-celled infiltration of Glisson's capsule.

The stomach invariably contained a coating of mucus in the glandular area and, in one or two cases, patches were seen over which the mucous membrane had disappeared, leaving a smooth scar surface. In one case patches of necrosis were noticed. In another case the cardiac non-glandular portion showed scar tissue areas from which the original surface had been removed.

The kidneys appeared normal macroscopically, but histologically a mild glomerulo-nephritis was described in one case, and, in another, parenchymatous nephritis with catarrh of the tubules.

The skin was also an interesting feature, for, in the case of the three specimens examined (two at the Imperial Veterinary Research Institute, Muktesar, and one by Professor J. F. Craig, of the Dublin Veterinary College), the reports were in agreement in that each recorded atrophy and degeneration of the sweat glands. It is this atrophy which is apparently responsible for the fact that the real non-sweater never recovers to the extent of becoming a free-sweater, no matter what the treatment or environmental conditions may be.

In considering the question from a general standpoint, several facts of interest emerge. In conversation with some of the older trainers who have been in India for upwards of fifty years, I learnt that in their early

days this condition was unknown, and that it is apparently in the past twenty-five or thrity years that it has come to notice. It is not likely that it could have existed in those early days without the knowledge of people intimately connected with racing. Further, a glance round racing stables at Poona left one convinced that, though all horses there were under the same conditions of climate and work, non-sweating was prevalent to a varying degree in some stables, while absent, or practically absent, in others. Again, it is very noticeable that Arab horses are seldom affected, though Indian-bred horses appear to be quite susceptible. The only Arab pony I can recollect as being dry-coated was in the hands of a European trainer.

I combined these facts with what I knew of the symptoms and post-mortem appearances of the condition and asked myself what one factor might be at work which would answer the questions one might ask in an effort to elucidate the problem. Knowing that arsenic in powder or Fowler's solution has been used as a tonic by trainers for many years, it occurred to me that its administration in the doses in which I well knew it to be given in certain training stables might constitute this unknown factor or might at least be a major contributing factor. While realizing that a suggestion of this kind is highly controversial, and that I can bring no unassailable proof in support of it, I make a note of the *pros* and *cons* so that further investigation may, at any rate, consider the possible influence of arsenic on the condition. It is pleasing to note that A. D. MacGregor, who has studied anhidrosis for some time, was sufficiently impressed by this possibility, after a discussion we had on it, to insert a paragraph concerning it in his recently published paper.

The *pros* may be noted as follows :—

1. Thirty or so years ago the knowledge of drugs by the layman was not what it is now. The trainer of those days was not the kind of person one would expect to have such knowledge. If we are to believe the statement of "old timers" then we can connect the appearance of non-sweating in racehorses in India with a period about thirty years ago, when racing began to get out of the hands of amateurs and into the hands of the professional trainer, most often an Australian. Racing in Australia was in much the same state as in India about the time, *i.e.*, competition was increasing, stakes were becoming more valuable, and the professional element, whose livelihood depended on success, was taking charge. It is reasonable to assume that the knowledge of the value of arsenic as a tonic was becoming widespread among the professional racing fraternity, who, quite naturally, might think that, if a little were good, a little more must be a lot better, until the state of affairs is reached when, in one racing stable known to me in Poona, the amount picked up by a four-anna piece (silver) was the regular dose, *i.e.*, anything up to 30 grains. Further, it was known by me that a proprietary tonic powder then on the market contained 9 grains of arsenic in each powder.

2. Why should horses in one stable show 50 per cent or higher incidence of non-sweating, while those in another under similar conditions of work and climate are free? The answer may be that several of the more conservative trainers continued to place their reliance on sound methods of feeding and had a prejudice against the use of tonics.

3. Is it or is it not reasonable to suppose that the Arab horse is practically free from this condition simply because his owner or Arab trainer is a man who knows nothing of arsenic? Those who know the Arab owner or trainer in India will agree that he is a simple man not likely to be versed in the uses of medicines such as this, or, in fact, of medicines other than those prescribed for him by a professional man or the country medicines in use in Arabia or here in India.

4. During my last year in Poona I made no secret of my belief that the use of this drug was a contributing cause of dry coat. The matter was commonly discussed. I believe I am right in saying that to-day dry-coated horses are not nearly so common in Poona and Bombay as they were when I was there five on six years ago. This may perhaps be accounted for by the fact that the drug is not now in such common use as formerly was the case.

5. When one considers the number of army horses there are in this country, the vast majority of them being imported horses and many of them doing fast work or fairly fast work, and the number of polo ponies, also mostly imported, which do certainly perform fast work, is it not a remarkable fact that, if the condition we are discussing is merely climatic, true non-sweating amongst these animals should be so rare? The only polo pony the writer can remember as being so affected was an ex-racehorse, and only two Army horses which presented typical symptoms of non-sweating, have been seen by him in a period of fifteen years. One of these was a battery horse seen in Meerut about 1925, *i.e.*, about the time that large graduated doses of arsenic constituted a favourite treatment for debility. The other was a horse in a remount depot which had a history of having received arsenic after recovery from strangles.

6. I have previously referred to the fact that horses have arrived in this country showing this condition. In one such case I was fortunate in having been able to obtain a chemical analysis of the urine and hair clippings. In both specimens arsenic was found in a quantity which excluded the possibility of it being due to surface or other contamination.

7. Let us now look at the symptoms and post-mortem appearances and see how they coincide with the thesis put forward.

In the human being it is known that in cases of chronic arsenical poisoning arsenic is deposited in the hair; in fact, by a special method of examination, it can be determined whether or not the arsenic has been administered continuously or at intervals. If at intervals then the arsenic is deposited in bands, the intervals between which

can be seen and the periods to which they correspond approximately determined from the rate of the growth of the hair. After prolonged administration, the hair may fall out and the nails have been known to drop off. There is a keratosis in which the epidermis is exfoliated and the skin has a scurfy appearance. At a later stage jaundice is often seen and pigmentation of the skin is noticed. In the later stages there is disturbance of sensation in localized areas, generally in the palms of the hands and the soles of the feet, *i.e.*, there is a peripheral neuritis. Later there may be sensory and even motor paralysis affecting these and other parts of the body and, in fact, the paralysis may go so far as to involve the spinal cord. The stomach exhibits a condition of fatty infiltration or cloudy swelling of the gland cells.

In the human being arsenic is known to be stored in the liver, stomach, intestinal walls, spleen and kidney as well as in the skin and hair.

In the racehorse the administration of arsenic coincides with periods of great exertion which may to some extent alter the manner in which it acts in his body as compared with the human. Further, just as in man there is certainly an individual idiosyncrasy to the drug, so that quantities which are harmful in one case may produce no visible result in others.

I have mentioned the metallic lustre noticed on the coats of horses in the Poona paddock and the fact that these horses usually win or run well. Is it far fetched to assume that, as arsenic is known to improve the appearance of the coat, this change in its appearance coincided with the earlier administration of arsenic, or that the ability to run well should also coincide, the tonic properties of the drug being well known?

In conjunction with the Chemical Adviser to the Government of Bombay, I arranged for the quantitative chemical examination of samples of hair from six normal young stock in the Ahmednagar stud and from a bad non-sweater recently returned from Bombay whose coat was bleached and skin scurfy. The amount of arsenic in the former was from nil to ·12 m.g. per 100 g. of hair (probably surface contamination was present), whereas in the non-sweater the amount was 1·5 m.g. per 100 g. Later examination of the hair of further non-sweaters frequently gave similar results.

I think it is reasonable to assume that one of the factors involved in the loss of coat colour in non-sweaters may be this deposition of arsenic in the hair. I have said that the sweat glands in these cases have been described on histological examination as atrophied and functionless. The consequent absence of sweat as a vehicle for the spread of sebaceous material from the sebacaous glands would, it is thought, be a contributory factor to the loss of lustre.

What is of principal interest, however, is the cause of the atrophy of these glands. I feel that it may be due to one of two things. Either

the cells of the glands are destroyed by the passage of arsenic through them in the profuse sweating which takes place in warm, humid climates, or else the nerve supply to the gland is destroyed by the action of arsenic on the individual nerves, just as certain peripheral nerves are paralysed in the human being. It has been described, when dealing with symptoms, that a marked scurfiness of the skin is apparent in dry-coated cases of some standing, and that in certain cases the skin of the face is partially denuded of hair. This, I think, corresponds in some degree with what is described in the human.

Unfortunately the skin of the face was never sent by me for histological examination, but if it were so examined it might be found that the hair follicles, and possibly the sebaceous glands, were damaged in much the same way as the sweat glands.

The excretory process would appear to be fairly rapid in the horse. Arsenic, which is stored in the liver, is fed to the skin and may in this way be the cause of continuous irritation during the process. In this connection, an interesting experiment was carried out on a cast horse. It was given 15 grains of arsenic daily over a period of six weeks, but, though it is known that the drug is stored in the liver and other organs, there was no trace of it found on chemical analysis of the organs when the subject was destroyed three months after the last dose of arsenic had been given. It was not expected that this dosage would bring about non-sweating. What the experiment did indicate, however, was that arsenic was very rapidly excreted from its storage places in this individual, and this rapid excretion may take place largely through the sweat glands. In the human being the elimination of arsenic appears to be much less rapid than was so in this case. In the subject of this experiment it is interesting to note that a state of catarrh of the stomach was found on autopsy.

The conditions described as seen in the kidneys do not seem to me to be of sufficient importance to permit of any deductions being made, though a mild degree of nephritis is not inconsistent with the elimination of an irritant drug.

That there is a chronic inflammatory condition of the mucus-secreting glands or the stomach is unquestionable, and what is more likely to effect this than the prolonged administration of a drug having the irrirant properties of arsenic, especially when given in quantities much greater than can be utilised in the body. It is not unlikely that the patches of fibrous tissues seen on the lining of the stomach are the results of the healing of those parts from which the mucous membrane has sloughed off from the direct action of this corrosive drug.

The cirrhosis of the liver may also have a bearing on the question, as it is well known that arsenic, for the greater part, is stored in the liver.

The size of the spleen commented on was a feature which puzzled me when considering it in connection with the administration of

arsenic. In reading a report on splenectomy, however, by De Kock and Quinlan, I find that a footnote is appended which reads as follows :—

With reference to such an enlargement of the spleen, the following reference in Kitt (35) is interesting :—

" Congestive hyperæmia is due to paralysis of the splenic nerves, or to stimulation of the splenic vaso-dilator branches, and therefore is the direct result of dilation of the splenic vessels. It is produced by toxic substances such as curare, narcotic drugs, or the organisms of infectious disease, and consequently the condition is a neuroparalytic hyperæmia."

I think arsenic in large, continuous doses may safely be included in the list of substances which might cause this condition, owing to its well-known effect on the central nervous system.

Chemical examination of the organs of some of these cases was interesting. In one well-known non-sweater '95 and '75 grains of arsenic were recovered from the viscera and skin respectively, while in another 1·25 and 1 grains respectively were similarly detected.

When in England on leave, I described the symptoms and *post-mortem* findings to Professor Bronte, then at the Home Office. His opinion was that there was nothing in the picture I presented inconsistent with chronic arsenical poisoning. I asked him particularly if he could account for the fact that so many of these cases dropped dead after exercise. His reply was that this was probably due to paralysis of the vagus nerve, such paralysis being, possibly due to the effects of arsenic on the nerve. A further possibility to account for this may be the direct effect of arsenical poisoning on the heart muscle cells—*vide* Cushing's text book of pharmacology. It is perhaps possible that in attributing to heat-stroke the nerve changes he has noted in non-sweaters, MacGregor may have drawn the wrong conclusion, and that any such changes noted may have been due to damage to the central nervous system by arsenic in a manner similar to that seen in the human being.

That is the argument in support of my contention. Let us examine the evidence that may be brought against it :—

1. The case of the suppressed sweater arises at once and may possibly be ascribed to a nervous derangement affecting the nerve supply of the glands sufficiently to cause the symptoms noticed. The writer owns a horse of this kind which, under suitable conditions, is a free-sweater, but in the hot, moist climate of the coastal region is not so. So far as is known, this horse has never received arsenic, but then he is not the typical non-sweater I have been describing.

2. It is possible that the cause of sudden death in horses affected in this way, which takes place almost invariably after a gallop or race,

may not be due to failure of the vagus nerve, but to the effects of the very high temperature engendered during such exertion, though this is very unlikely. If this were the case, however, one would expect to find that the big majority of such deaths would take place during racing or training in the very hot times of the year, *e.g.*, at the Calcutta Monsoon Meeting, but this is not so.

3. There is a catarrh of the stomach present in every case, and I have assumed that this is due to administration of arsenic. I have, however, found a somewhat similar though much milder condition in cases which, though affected to some extent, do not present the extreme symptoms of the confirmed non-sweater. It is possible, therefore, that the primary condition may be such a catarrh of the stomach and that aggravation of it may take place by the use of arsenic, and, further, that the post-mortem findings, such as atrophy of the sweat glands, enlargement of the spleen, etc., including death from damage to the central nervous system, may be complications of an original simple condition due to aggravation from the cause given. This, however, does not tally with the fact that the condition is, as stated, observed commonly in some stables, while absent, or practically absent, in others, though the argument may be put forward that different methods of feeding may be responsible for this.

Observations made on the living subject have up to now led us to no definite conclusion. Therefore, in any future investigation of this condition, I would suggest that a comprehensive histological examination of all organs and tissues, and especially of the central nervous system, should be undertaken, and that facilities should be put at the disposal of investigators by race clubs concerned for such an examination of all racehorses which drop dead after work or exercise. This would take time, but it is possible that evidence might so be obtained from which definite conclusions could be drawn. So far as I can learn, no investigation along such lines has hitherto been carried out.

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"DRY SWEATING" IN HORSES

By J. E. Barnes, M.R.C.V.S.,
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"Dry sweating" or dry coat of horses is, as the name implies, a chronic condition in which the animal does not sweat when he would normally be expected to do so.

Horses Affected.—English and Australian thoroughbreds of any colour or either sex. Arabs do not suffer, although I have known one Arab—a dark chestnut—to be "dry".

Occurrence.—"Dry sweating" is the curse of horses in Calcutta (hot weather), Bombay, Madras, Rangoon, Singapore, Colombo and the Dutch East Indies. Cases may occur in parts of South Africa.

Generally after some months' residence in Ceylon, and as a rule in the hot weather before the break of the South West monsoon in May, horses about to go "dry" start to "blow" abnormally after exercise. The amount of visible sweat diminishes and loses its property of lathering. If the temperature of such a horse be taken after a one-mile gallop it may be found as high as 107° or 108° F. and respiration anything up to 150 p.m. As your readers will know, horses work here in the early morning. Such a horse's temperature might fall by evening to 103° or it might stay at 105° F. In the climatic conditions prevailing in Colombo it would not get to normal and the horse would "blow" in more or less distress. In a good number of cases there is some falling out of hair, especially of the face. The Calcutta investigators emphasised this feature which would appear to be much more marked there than here.

Horses in this condition retain it indefinitely if kept in Colombo. As racehorses, they cannot show their form and if exercised without discretion they die of hyperpyrexia. Before death they stagger about, fall and lie unconscious for some time, then on coming round they struggle to get up and go out, or they may die almost at once. As polo ponies they cannot play and, if forced to do so, die of hyperpyrexia.

The above is a picture of what happens to horses kept in Colombo. Most horses go to Nuwara Eliya for the hot weather (6,000 feet odd elevation) where it is often quite cold. Nine out of ten horses sent to Nuwara Eliya recover.

As a rule, those that are going to go "dry" in Colombo, on arrival in Colombo from Nuwara Eliya sweat profusely day and night for five or six days, then gradually go "dry," say, within 14 to 21 days of their return.

I said above "generally after some months' residence" because horses have to my knowledge disembarked "dry" *ex* England; the last one was Fairy Goblet by Salisbury—Elfglass by Soldennis, landed on April 5, 1936. He died of hyperpyrexia while walking to Walles' Stables—four miles. The groom who travelled with the batch in which he was coming told me that all the voyage, when the others had been sweating well, he had been quite dry. I wrote to Colonel Blake who trained him in Ireland to ask whether he had been distressed on hot days in the summer. He said the horse had never sweated much but that they regarded him as a good fit sort. I was called in a few days ago to see a horse passing through to Madras from England. The groom with this one on the boat was treating him as though he had pneumonia. He was "dry."

Treatment.—Dry horses sent from Colombo to the hills—Kandy, 1,600 feet elevation, or Nuwara Eliya, 6,000 feet, or anywhere else for

that matter, recover, as a rule, in from six to ten days. There are, however, obstinate cases which remain dry, some never recovering although kept in the hills for years after their racing careers are over. They have usually been bad cases for two to three years in Colombo.

The Royal Calcutta Turf Club, a number of years ago, tried to find a cure, the work being done at the Calcutta Veterinary College. The conclusion they arrived at was that the cause was a thyroid deficiency, and that the administration of thyroid extract tablets in the food solved the problem. I have been unable to improve with thyroid any cases I have treated.

A claim was made two or three years ago that Bayer's "padutin," a vaso-dilator hormone preparation, was an effective cure. Padutin has to be given intramuscularly and is expensive. I gave it a thorough trial on three or four horses and, although I improved their coats, making them nice and bright, whereas they had been dull, I did not make the horses sweat. In any case a medicament that has to be given repeatedly, at short intervals, with a hypodermic syringe is ruled out as being impracticable for racehorses in training.

I said above that any sweat there is from a dry horse does not lather. This is most noticeable in the first few days when a dry horse is starting to recover. The first place to sweat is down the mane and the sweat runs down the neck as a wet line and will not lather. Next the horse gets moist on the sides of the neck, behind the ears, on the breast under the saddle and girths. When this stage is reached there may be a pause in the recovery for two or three days. Then a non-lathering wet area makes its appearance between the thighs; this eventually lathers and normal sweating is restored to the whole body.

The property of "lathering" of sweat may be restored by giving grated cocoanut in each feed. Grated cocoanut contains a high proportion of fat. Trainers give this cocoanut fairly willingly and, although one cannot claim that it is a cure for dry sweating, it brightens horses' coats and any moisture that does come out of them will lather.

Sometimes one gets peculiar cases. In 1926 I was planting at 1,600 feet elevation where any horse would recover. On my return from leave to England I found a three-year-old I had left behind in training, very dry in Colombo. I took her back to the estate where she recovered in a week. In August I sent her to Colombo to race. She lived there for a week and by the end of that period was so dry that I decided not to run her. I transferred her to the estate and to my surprise she did not make the useful spectacular recovery and stayed dry until mid-October. Then one morning when I rode her she broke out nicely. On my return to the stable I put two or three heavy rugs on her and sweat ran off her in bucketfuls. Next day she was as dry as she had ever been and remained so until the following April when she sweated a bit, but she never sweated normally again and I shot her in 1929.

Last year I had an aged mare which had been racing for six years in Colombo and the whole time had been more or less dry. I had been hacking her in Nuwara Eliya and she had sweated normally. She went to Colombo by night train at the end of April and as I wanted her quiet for an indifferent horseman, to whom I had sold her, to ride in Colombo, I rode her myself the day she got to Colombo for some 20 minutes in the afternoon, between the hours of 5 and 6 o'clock. She walked back to the stable all right but broke out to sweat as though the hose pipe had been turned on her. At 7 p.m. she went down in her box in a sort of fit of convulsions. I got her up and fixed up a punkah to keep the air moving in her box. I gave her normal saline per rectum and she recovered in three or four days. The tetanic-like spasms of her whole muscular system lasted for about 36 hours. This case would appear to be one of heat cramps such as I have seen described in miners in the South African gold mines.

Aetiology.—The primary cause appears to me to be climatic. The hot and humid atmosphere has upset the blood salt balances.

In those cases which never recover, perhaps the sympathetic nervous centres controlling sweating have undergone a form of degeneration, or it may be that the correct blood salt balance cannot be restored, or both.

Innumerable slides have been made of the skin of dry horses and the sweat glands appear normal.

The relative humidity in stables in Colombo is very high during the night in hot weather being over 90.

The essential difference between the climates of Colombo and, say, Nuwara Eliya, is that in Nuwara Eliya there is a greater range of temperature. This looks as though the adrenals might be stimulated and so be an accessory factor in recovery.

Pilocarpine doses not make a "dry" horse sweat. I have not yet tried adrenalin because Wallis Hoare says it has no effect on the sweat glands, but I might try it in Colombo in May. The effect of adrenalin is very transitory so that one would have to repeat the dose and my argument against the hypodermic syringe again comes into play.

I am putting conditioned air in a stable in Colombo: it will be ready by May. While in the stable the horses, at my discretion, can virtually be in Nuwara Eliya, England or even Edinburgh or at the North Pole and go to Colombo and that beastly climate to work.

Whether this will keep them sweating I do not know, but I have hopes.

—*Veterinary Record*, Vol. L, No. 31, July 30, 1938.

INDIAN HORSES.

Empire Countries' Attitude.

PLEA FOR BETTER MARKET.

[*The Indian-bred has come in for some attention and encouragement during the last year or two. Schemes have been put into operation for assisting the breeders.*

In the following article, Capt. M. R. D'Arcy takes up the question of an Empire market for our home-bred. He points out the iniquitous laws framed by countries like Australia and South Africa against the export of horses from India and pleads for a fair deal.

An Australian himself, Capt. D'Arcy has spent many years in India. He is a well-known owner-trainer.]

By Capt. M. R. D'Arcy.

As a supporter of racing for many years, and as a champion of the cause of the thoroughbred horse in India, I offer the following suggestions :—

The racehorse in India is under an unjust stigma, imposed by certain Empire countries, like South Africa and Australia, as he is debarred from being shipped there direct. Horses may return to England, and after a stipulated period of quarantine, be sent abroad. But they cannot be sent direct from here. Why cannot the period of quarantine be undertaken by Australia and South Africa, for instance, and thus save the unnecessary travel and risk to the animal, as well as time and expense to the owner ?

With the care and attention the racehorse receives in India, and the competent veterinary supervision which watches over his welfare and gives him the Health Certificate when exported, the danger of spreading any contagious disease is minimised. In fact it is negligible. The proof of there being no such danger is borne out by the fact that horses are returned to England—the home of the British thoroughbred horse, where the best and the most valuable horses and studs in the world are quartered, and no objection is raised by the Department of Agriculture, or the owners and breeders there.

One would imagine that countries like South Africa and Australia would be only too pleased to accept such thoroughbreds, either for the purpose of racing or for the Stud, that India could offer. But to keep the British thoroughbred up to the standard in any country outside Great Britain, fresh blood from England is required, and many a good horse is missed by these countries by having laws that debar horses from being sent direct from India.

Fear of Contagion.

Various reasons are given for debarring our horses—Australia's main fear being disease. So far this fear is imaginary, as no definite case can be cited of a contagious disease being due to a thoroughbred horse got from India. Furthermore, by malleining and quarantine, that risk can be averted. It could be understood if Australia stops short with putting such a ban, but the rather unfortunate attitude is adopted of " By our goods ; yours we do not want." Admittedly Australia may be free from certain diseases, but they certainly have others equally dangerous. Objections are not taken by other countries purchasing their stock, because the necessary precautions are taken before shipping them and also on landing at their destination.

South Africa, on the other hand, has every kind of horse disease known in India. Yet under proclamation No. 132 of 1923, horses are debarred from entering from India. In this case, it is not on account of any risk of disease, but presumably to protect vested interests there, the breeder of the local thoroughbred. New blood is continuously being imported direct from England and Australia (Stud horses free from tax). Yet horses from India cannot be shipped direct. I am sure that this state of affairs obtains because of the lack of interest of our Government to take the matter up on behalf of the owners and breeders of India.

Inter-Empire Reciprocity.

There is no reason why Empire countries should not show more reciprocity, situated as we are. Racehorses from India could be enabled to visit South Africa for the big meetings there, which are held in our off season here, and *vice versa*. Laws of a more comprehensive scope should be in vogue. India would be able to sell an attractive Stud proposition at a good figure, if we could ship direct. The Indian market for stallions is very limited, and prices for real good stud horses unattractive. Our isolation can only be due to the fact that we are badly represented. This seems the reason for our interests in this respect being neglected and the Department of Agriculture in the countries concerned, making such unjust and unfair laws.

Diseases both with man and animal play havoc with the poor and needy, the ill-nourished, and neglected country-pony. It is among the starved ill-bred cattle that sickness breaks out, as it does in the filthy slums of the poor. But bloodstock, I am pleased to say, are well cared for, and they do not run any abnormal danger of disease in India than anywhere else. Qualified supervision and quarantine eliminate that danger.

Invidious Propaganda.

Quite recently a lot has been written, and various suggestions made for the improvement of the thoroughbred Indian horse. Race Clubs are only of late realising the fact that the local horse must be catered far better than it has been in the past, and for this the Government has to be thanked. But is this enough ? The Indian owner and breeder wants

Reviews.

Dollar's Veterinary Surgery. Edited by J. J. O'Connor, M.R.C.V.S. Third Edition. Pp. x + 992. 720 Illustrations. *Price* 30*s*. Published by Bailliere, Tindal and Cox, London, W.C. 2.

This 1938 edition of Dollar's Veterinary Surgery by Prof. J. J. O'Connor has been thoroughly rewritten throughout to suit the requirements of the Veterinary practitioner and the student alike. The book has been divided into three parts, *viz*., General Surgery, Operative Surgery and Regional Surgery and this division has got the merit of feciliating references. A number of unnecessary details which were clouding the main points in the previous issues have been omitted and this has effected a decided improvement in the book. We are very glad that the subjects of epidural anæsthesia and Nembutal anæsthesia have received good consideration at the hands of the author and they have been detailed at length. This is as it should be; because, in these days humanitarian considerations play a large part in our operation on animals. The illustrations also have been very much improved with a judicious pruning of some of the obsolete figures in the previous issue. All these have greatly enhanced the value of this edition, a copy of which should be on the table of every veterinarian.

Veterinary Obstetrics (Equine and Bovine). By Dr. Franz Benesch. English Translation edited by Ohn G. Wright, F.R.C.V.S. Pp. ix + 258. Illustrated. *Price* 12*s* 6*d*. Published by Bailliere, Tindal and Cox, London, W.C. 2.

Veterinary Obstetrics is a very interesting branch of Veterinary Science but unfortunately it has not received that amount of attention it deserves. It is, therefore, a great pleasure for us to go through this handy English translation of the famous Prof. Benesch's work on Obstetrics.

The first few pages of this book have been devoted to a preliminary consideration of obstetrics, obstetrical equipment, the handling of obstetrical cases and epidermal anæsthetic, while the rest of the book is entirely devoted to a discussion of the various forms of presentation of the fœtus causing dystocia and their methods of correction by manipulation and operation. The illustrations depicting the remedial measures are very good and self-explanatory and this is a very notable feature of this book. Some of the instruments described in this book are unknown to the Veterinary Surgeons in this country. Stuven's Rhachiophore for the destruction of fœtal vertebral column and its surrounding tissues with a view to reduce the width of an absolutely over-sized calf and the Vakufakt which is an 'evacuator' to saw around the vertebral column so that it can be removed completely are some new instruments which are described for the first time and we doubt

very much whether they will ever come into use here. All the same the book is a thoroughly practical one, very interesting and instructive, and the profession must be grateful to Prof. Wright for having made available the practical recorded experiences of Prof. Benesch.

Veterinary Pathology and Bacteriology. By S. H. Gaiger, F.R.C.V.S., and Gwilym O. Davies, M.V.Sc., M.R.C.V.S., D.V.H. Second Edition. $8\frac{1}{2}'' \times 5\frac{1}{2}''$. Pp. x + 712. Figures 198. *Price* 25*s*. Published by Bailliere, Tindal and Cox, London, W.C. 2.

The second edition of Gaiger and Davies excellent text-book will be welcome to all who are familiar with their first, which filled such a serious gap in Veterinary literature. Only six years have elapsed since the publication of the first, but progress in Bacteriology is so alarmingly rapid that the need for a new and up-to-date edition has been great.

Owing to the decease of Prof. Gaiger, Mr. Davies has had to undertake the task of revision himself, and is to be congratulated on the result. In writing any text-book for students the aim must be to make it sufficiently comprehensive and yet of reasonable proportions. The tendency is always, therefore, to be a progressive shift in the direction of the former to the sacrifice of the latter. It is in this respect that the book is so admirable, for the author strikes the required happy mean, and with so vast a field to cover as Veterinary Pathology, Bacteriology and Immunology, this is an extremely difficult task.

The actual increase in size has been limited to 100 pages, due rather to small additions throughout the text than to elaboration of any section. Additions have been made to the chapter on diseases of uncertain etiology, where fowl paralysis, malignant catarrhal fever, and Stuttgart have been included. Recent work on the members of the coli-typhoid group and the clostridia has been included, as also on foot-and-mouth disease, distemper, louping ill and bovine pleuro-pneumonia. The etiology of neo-plasms has also been dealt with more fully, which is desirable.

The author has added many useful tables, and the citing of bibliographical references is an improvement which might have been developed further and demanded no greater space than was available.

It may perhaps be considered that the treatment of the Pathology of the anæmias and of the endocrine glands is really too superficial, and some brief mention of epidemiology and statistical principles would have been very valuable. Only one general criticism may be made of the book. As a concise statement of fact it is outstanding: but in a book designed primarily for students a more reasoned and rational development of all subjects on physiological and biological lines is desirable, even if at the expense of some of the fact.

to be able to put his horse on the world market on terms of equality with other horse-breeding countries, and ship his horses direct, should he so desire, to Empire countries at any rate. The flaunting of the "disease bogy" by these countries should be resisted and such propaganda put down by those whose duty it is to look after the interests of the Indian thoroughbred.

Although India is a big country, as a racing country she is comparatively small, and if racehorses are bred in any numbers, breeders ere long will be looking for an outlet for their surplus horses. To be able to compete in outside markets would assist and also encourage the breeders to improve an industry that surely will develop, but without the backing of our Government we shall be left high and dry. Their assistance and support is essential, and I hope to see at no distant date this matter taken on hand seriously. It will then be found that the Indian horse improves definitely.

—*The Hindu*, September 19, 1938.

Association News.

THE INDIAN VETERINARY JOURNAL (Vol. XIV).

Statement of Receipts and Disbursements for the year ending 30th June 1938.

		Rs.	A.	P.			Rs.	A.	P.
To Balance on 1st July 1937	...	1,892	3	6	By Establishment:—				
,, Subscriptions	...	4,353	11	5	Rent Rs. 480	...	960	0	0
,, Advertisements	...	536	2	0	Salaries Rs. 480				
,, Interest	...	45	10	0	,, Honorarium	...	1,000	0	0
,, Reprints	...	53	4	0	,, Printing (Journal)	...	1,605	8	6
					,, Railway Freight	...	13	3	6
					,, Binding Books	...	27	12	0
					,, Bank Charges	...	1	2	0
					,, Postage	...	1,018	12	0
					,, Printing and Stationery	...	45	10	0
					,, Refund	...	3	10	0
					,, Audit fee	...	25	0	0
					,, Balance as on 30th June 1938	...	2,180	4	11
	TOTAL	... 6,880	14	11		TOTAL	... 6,880	14	11

Examined and found correct.

K. GOPALAKRISHNA RAO,
Registered Accountant and Auditor.

5, ERRABALU CHETTY STREET,
MADRAS,
28th July 1938.

The index is complete and accurate, and the appendix probably the most concise account of the principal laboratory methods that has ever been composed. It is a book for students and that should be on the bookshelves of all veterinarians, in every branch of the profession.

Veterinary Helminthology and Entomology. By H. O. Monnig, B.A., and Dr. Phil, B.V.Sc. Second Edition. Illustrated. Pp. xviii. + 409. *Price 30s.* Published by Bailliere, Tindal and Cox, London, W.C. 2.

We are extremely glad to note that the book has run into a second edition, and we are not surprised at it knowing as we do the usefulness of the book to veterinarians. The general arrangement of the sections of the book remains the same, but some worms of doubtful importance have been omitted and other new ones of importance have been included. Under Opisthorclinidæ, for example, chlonorchis has been omitted, under Schistosomatidæ, the new species. *S. nasalis* Rao, 1932, has been added, and so on. There are no changes, so to say, under Entomology. No information regarding helminth parasites of the elephant and camel has been included even in the second edition. This information would make the book more useful to students and to veterinarians practising in regions where those animals are domesticated.

We learn that this book has been prescribed as a text-book for the University students of the Madras Veterinary College. We feel sure that the present edition will be found as useful and popular as the previous edition. We congratulate the authors and the publishers on this excellent book.

Experimental and Clinical Studies of the Spine of the Dog. By Geoffrey Bernard Brook, D.Sc., F.R.C.V.S. Pp ix + 122 with 50 Figures. Cloth-bound. *Price* 5s. Published by Bailliere, Tindal and Cox, London, W.C. 2.

This volume is a good addition to Veterinary literature. It is of great interest and importance to the Veterinary surgeon in diagnosis and treatment of diseases of dogs. The X-ray photographs are good and instructive to the Veterinary Radiologist.

Atlas Cattle Dip Improved. Suppliers: D. K. Parvate & Brothers, Poona City. Packed in strong five gallon steel containers. *Prices*: 1 oz. bottle As. 3; 2 oz. bottle As. 5; ¼ gallon tin Rs. 2-4; 1 gallon drum Rs. 7-8; 5 gallon drum Rs. 35 net.

We have received a sample of "Atlas Cattle Dip Improved" from Messrs. D. K. Parvate & Bros., Poona City, and have found, in our experience, a good remedy to remove lice and ticks and cure mange.

The following reasons for using "Atlas Cattle Dip Improved," are to be considered:—It is a standard arsenical dip—recognised and approved by the U. S. Department of Agriculture. It destroys lice, cures mange and prevents bot fly. It is safe to use and the results are immediately beneficial. It does not over-heat, burn or scald because of the quickness with which it goes into solution and because it remains in solution indefinitely when properly mixed, thus insuring uniform strength. It is ready for use and does not deteriorate with age. It is economical to use and easy to obtain.

For particulars and literature apply to Messrs. D. K. Parvate & Bros., 627, Sadashiv Peth, Poona City.

K. R. O. (Kills Rats Only). Agents in India: D. K. Parvate and Bros., 627, Sadashiv Peth, Poona City. *Prices:* Minimum size can (2 oz.) Rs. 2-4-0, net.

We have received a sample tin of K. R. O. from Parvate and Bros., Poona City. This American recipe deals a sure death to Rats and mice and in this field it has no equal in the medicinal world. Some remarkable features of K. R. O. are: (1) It is non-poisonous to creatures other than Rats and mice; hence can be used with safety in any house, store-house, hotel, sweet-shop or record rooms; (2) It is certified as the most effective by the U. S. Government of Agriculture; (3) It has killed 142 rats with one 2 oz. can and 578 rats with two cans of the same size; (4) Department of Agriculture, Bombay, have tried it successfully with satisfying results; (5) In our experience we have found it very effective.

Obituary.

Mr. B. DORASAMY IYENGAR, G.M.V.C.

We regret, we have to announce the demise of Mr. B. Dorasamy Iyengar, Veterinary Inspector, Warangal District, H. E. H. The Nizam's Dominions, recently after a short period of illness. He was about to retire in a few months. He leaves behind him, one son, five daughters, his widowed wife and a large circle of relations and friends to bemoan his loss.

We offer our heartfelt condolences to the members of the bereaved family.
