STUDIES ON
THE PATHOLOGY OF EXPERIMENTAL SWINE FEVER

Thesis

Submitted to the Faculty of Veterinary Science,
Magadh University, in Partial Fulfilment of the Requirements
for the Degree of M.Sc. (Veterinary)

Ashraf Alam,
B.V.Sc. & A.H.
Post-Graduate Department of Pathology
Bihar Veterinary College, Patna
November, 1965

STUDIES ON

THE PATHOLOGY OF EXPERIMENTAL SWINE FEVER

Thesis

Submitted to the Faculty of Veterinary Science, Magadh University, in Partial Fulfilment of the Requirements for the Degree of M.Sc. (Veterinary)

BIHAR VETERINARY COLLEGE
PATNA
Acc. No. 4165
Date. 15.12.66

Ashraf Alam,
B.V.Sc. & A.H.
Post-Graduate Department of Pathology
Bihar Veterinary College, Patna
November, 1965

STUDIES ON THE PATHOLOGY OF EXPERIMENTAL SWINE FEVER

THESIS

Submitted to the Faculty of Veterinary Science, Magadh University, in partial fulfilment of the requirements for the Degree of M.Sc. (Veterinary).

Bihar Veterinary College,
November, 1965.

ASHRAF ALAM,
B.V.Sc. & A.H.

I certify that this Thesis has been prepared under my supervision by Sri Ashraf Alam, a candidate for the M.Sc.(Vet.) with Pathology as major subject,1965, and that it incorporates the results of his independent study.

P. B. Kuppuswamy 20.11.65

(P. B. Kuppuswamy)
B.A.,G.M.V.C.,B.V.Sc.,P.G.(N.Z.),M.S.(Missouri)
Vice-Principal & Professor,
Pathology and Bacteriology,
Bihar Veterinary College,
PATNA.

November,1965.

C O N T E N T S

ACKNOWLEDGEMENTS

	Page
Introduction	1 - 7
Historical account of Swine fever ...	8 - 12
Materials and Methods	13 - 19
Review of literature and present observations on clinical symptoms and gross lesions	20 - 43
Review of literature and present observations on histopathological changes	44 - 67
Review of literature and present observations on haematology	68 - 84
Review of literature and present observations on amylase and haemolysis tests & amylase estimation in blood	85 - 88
Discussion and conclusions	89 - 98
Summary	99 -101
References	I - VIII

Tables --- --- 4
Graphs --- --- 7
Photographs --- 75

****

A C K N O W L E D G E M E N T S

The author is highly grateful to Sri P.B.Kuppuswamy, B.A.,G.M.V.C.,B.V.Sc.,P.G.(Newzealand), M.S.(Missouri,U.S.A.), Vice-Principal and Professor and Head of the Department of Pathology and Bacteriology for his constant supervision, valuable guidance and advice.

He is also indebted to Sri S.A.Ahmed,B.Sc.(Ag.), M.R.C.V.S.,P.G.(Denmark), Principal, Bihar Veterinary College, Patna, for the facilities given for carrying out this work.

He is thankful to Sri S.M.Hadis, Research Officer (Biological products), Livestock Research Station,Patna, for kindly providing the strains of viruses for the experimental work.

He is also thankful to the College Artist, Sri R.Bhushan for his help in the photographs.

He is grateful to the Animal Husbandry Department, Government of Bihar, for deputing him for this course which enabled him to carry out these studies.

ASHRAF ALAM

INTRODUCTION

I N T R O D U C T I O N

"Animal Agriculture is essential to a well nourished and happy people" - Ensminger.

The cry of the moment is population explosion and consequent increasing scarcity of food. We in India, therefore, are particularly more concerned with the problem and trying for greater production in all spheres of Agriculture and livestock industry. The problem of scarcity can be banished through increased stress on scientific farming and livestock development. Among all other livestocks, pigs - though appears as stinking creature, occupy a remarkable place in converting the food that it consumes into a high grade energy yielding product. It has played a vital role in the economy of nation throughout the globe wherever they are reared on scientific lines. The swine enterprise usually never runs in loss and the profits that they yield are more constant when compared to the other classes of livestock. Swine excel all classes of farm animals in the conversion of concentrated feed into meat and meat products. The live weight gain from a given quantity of feed is greater than any other class of meat animals and also the total calories in their meat is more than that of the food they consumed when compared with other classes of farm animals (Maynard,1946). They can efficiently transform waste products such as garden waste,garbage, kitchen waste, cabbage sprout tops etc. which are totally unfit for human consumption into products

of higher specific values. They have even been seen utilising unsound and damaged food excepting those damaged by poison or certain diseases to transform into pork as efficiently as sound grains (Illinois Agricultural experiment station, 1934; Krider et al,1944; Fairbanks et al,1947). Mouldy corn has also proved as good as sound corn and produced similar results. Where the dairy industry is flourishing and particularly those concerns which do not sell fluid milk, pigs have a definite role to play for they utilise skim milk and other bye-products and thus prove an asset to the dairy industry.

Swine are prolific and may produce two litters a year. The rapid growth, quick maturity and the huge number per litter makes, it ≡ a fortune to the pig breeders and pig industry. In U.S.A. in some of the exceptional cases a crop of two tons of pork have been recovered from a single litter of pigs by the time the pigs were six months old, though one ton is more common.

The dressing percentage of swines are the highest (65-80%) of their live weight as compared to the cattle (50-60%) and sheep and lamb (45-55%). The edible meat in the hog carcass is greater and is most nutritious. The energy content of pork is higher than beef or mutton because of the high fat and low water content.

They are also remarkable in storing fat of higher Quality, rapidly and no animal can compete in the production of fat per unit of live weight in so amazingly short span of time in such an economical way.

Apart from the above, it is noteworthy they can survive in very divergent climatic conditions.

The modern packing plant in foreign countries recovers a large number of bye-products, say about 2½% of the total live weight from its pork packing operations. The inedible byeproducts are used in many ways for the manufacture of several commodities useful for millions of people all over the world. Even some very useful medicines such as insulin and liver extract are made out of it.

With all its qualities of a wonderful farm animal, the condition of swine industry is still deplorable in India. Sentiments, tradition and religious prejudices have desisted people from rearing pigs. It is considered by some as untouchable and is therefore reared by so called low caste people chiefly the Domes in India and the pigs are allowed to thrive on human excreta and refuses that are thrown open in the field.

Agricultural production has already shown its limitations and scarcity has occurred. Poultry and piggery can occupy a great place in supplementing the food requirements of the nation and may thus solve food problem to a great extent.

Now people in India have realised the necessity of increased production and the Government has formulated ambitious plans such as Crash Programme for pig and poultry to upgrade and popularize swine and poultry keeping.

Since pig rearing was not popular so far as indicated above people are not familiar with the diseases of the pig and as such diagnosis was in jeopardy. The knowledge of the pathology

of swine diseases was also meagre. Attention is,therefore, required so that it may be on par with other classes of livestock.

Swine are threatened with a number of diseases but the number one killer is Hog cholera or Swine fever. A huge number of death are due to Swine fever alone.

Swine fever an exotic disease made its inauspicious appearance on our soil recently throwing a challenge and placing heavy responsibilities on the Indian Veterinarians. In a short span of time, it spread like wild fire and a number of States were in its grip, resulting in heavy mortality to all age groups of pigs. In our own State Bihar, this scourage practically wiped out the swine population at a number of places.

The disease was first recorded in early nineteenth century and is regarded as one of the most dreadful diseases affecting swines. It is an acute, febrile highly contagious disease caused by an ultra-virus, and characterised by the degeneration of the walls of the smaller blood vessels resulting in multiple haemorrhage, infarction and necrosis of internal organs. It has a global distribution and importance, takes heavy toll of life and causes huge economic losses to the swine industry.

No definite record of the occurrence of the disease is available before 1833 when the disease was first recorded in Ohio in the United States. From this focus the disease spread far and wide and by now it has become universal. In India the

disease was first recorded authentically in Maharastra in the beginning of February 1962 (Sapre et al,1962). It was probably rampant in Goa as early as November,1961 in the imported stock and from this focus the disease trickled to Maharastra causing havoc to the swine population. Almost simultaneously it was recorded in Uttar Pradesh (Murthy et al,1962) and later on in Bihar (Mundu et al,1963). It seems quite probable that the disease was prevalent in Punjab, Rajasthan, Andhra Pradesh, Bihar, Assam, NEFA at that time.

Our country which faced this ravage for the first time recorded almost 100% mortality wherever the disease made its appearance thus causing huge losses to the pig breeders. The actual assessment of the losses suffered in India is difficult to estimate but it can be safely said that the loss must be great enough keeping in view the number of deaths reported. A glimpse of the annual losses in U.S.A. and Great Britain will reveal the severity and gravity of this scourage where it stands at 48 million and 2[illegible] million dollars respectively.

A sigh of relief is felt when it was observed the outbreaks of Swine fever in India was of a conventional European type and not the more dreaded African Swine fever type which has an entirely distinct entity. African Swine fever is mainly prevalent in Africa and from there now it has trickled to a few European countries like Spain (1957) & Portogal (1960).

The importance laid upon the swine industry in the fourth plan and under the Crash Programme, calls for an immediate, planned and concerted efforts to fight out this dreadful disease

which may appear in the swine population from time to time. A detailed and well planned compaign has to be launched to banish and eradicate this disease once and for all from our country, if swine industry is to flourish.

It would be relevant to observe that as yet no serious attention has been given to the control of disease of swines in India. According to the census of 1961 the total number of swines are 1,23,89,284 and 8,67,961 in India and Bihar respectively which shows that the number is quite substantial and as such it warrants a close, concerted and planned attention. The stress placed upon the pig industry in the fourth plan necessitates that the problem should be tackled on a war footing. Not only it would help to solve the food problem to some extent but also fetch much needed foreign exchange. The control of diseases of pigs, therefore, deserves serious attention.

A good deal of work has been done on the different aspects of Swine fever in foreign countries but no systematic work has yet been undertaken in India particularly on the study of the pathology of this disease.

A precision diagnosis with "red and green light" decisiveness is essential. It requires no emphasis to have a fair knowledge of the "how and why" of the disease. Manifestation of clinical symptoms, lesions and related histopathological changes and the haematological picture at different stages of the disease lead to a clearer conception of the disease. The various pathological phenomenon ultimately lead to an early and correct diagnosis of the disease.

The present study was, therefore, undertaken with a view to study the clinical manifestations, blood picture, gross and microscopical pathological changes due to Swine fever with the American and local strains of Swine fever virus and efforts were made to correlate the various findings. An endeavour was also made to differentiate within the two strains as well as to eliminate the possibility of African Swine fever. Amylase and Haemolysis tests (Taylor, 1961) were also undertaken to see the efficacy of the tests and the desirability of using it in the field in our own conditions.

HISTORICAL ACCOUNT OF SWINE FEVER

HISTORICAL ACCOUNT

The earliest record of the domestication of pigs (Sus vittatus) dates back some 4,900 B.C. in China. The Biblical writings also record their domestication in the year 1,500 B.C. Great Britain domesticated pigs some 800 B.C. back. Though these animals were domesticated so early the available literature on their diseases were too meagre till the late 20th century. Pigs during those early days must be facing all sorts of disease. Swine fever one of the most dreadful diseases affecting swines was recognised as late as 1833. Shope (1955) cites that the disease had a humble and inauspicious beginning in Ohio (U.S.A.) in 1833 and rapidly spread to the different states of U.S.A. By 1887 it had cast its shadow to some 35 States of the country. It was primarily Swine fever which demolished the swine industry causing heavy losses in U.S.A. This disaster led to the formation of the Bureau of Animal Industry in U.S.A. in 1884 to fight out, control and eradicate animal diseases on a national basis. In 1862 the disease made its appearance in England and from this focus the disease trickled to Europe and Africa. At present, the disease is having a global distribution and has been reported from U.S.A., South American States, European countries, U.S.S.R., Australia and most of the African States. In the East it is prevalent in Nepal, Burma, Laos, Vietnam (most important disease), Combodia, Thailand, Malaya, New-guinea, Philippines, Hongkong, China (main lands), Japan, Korea and India.

Till 1961 in India there was no authenticated reports of the epidemics of the disease although an outbreak of the disease was reported in Aligarh farm in the imported pigs from Australia in 1944. During a brief span of 12 days as many as 708 out of 3,500 pigs succumbed to the malady.

Rao & Satyanarayana (1961) suspected the occurrence of this disease in few isolated villages in Andhra Pradesh which was not confirmed.

The disease was first authentically reported when it appeared at Morol, a suburb in Bombay in early February 1962 (Sapre et al,1962). The authors observed that the disease was perhaps brought to Goa through swines imported from Europe and was prevalent there as early as November,1961 where it is reported to have caused havoc and a near extinction of the swine population. From this focus the disease entered Bombay through carrier pigs brought for slaughter during X'mas. The total number of deaths in Bombay was said to be 1,200.

Soon, it was recorded from Nagal and in a few adjoining areas of Saharanpur district in Uttar Pradesh in the first week of March,1962 (Murthy et al,1962). In the affected villages over 200 pigs died and a number of them were down with the disease. The location of these villages on the southern bank of Jamuna river indicated the possibility of the spread of this disease from Punjab. Within a period of three months the disease was reported from 15 districts of the State. In Agra city alone 1192 pigs died within a month and some 2,200 pigs succumbed to this disease in 12 weeks in Mathura & Brindaban.

The disease exclusively occurs in pigs though the virus can maintain in other animals for a short time. Experimentally rabbits may be infected which showed only mild temperature reaction Vechiu (1939), Baker (1946), and Koprowski et al(1946). Hutyra et al(1946) cited Tenbroeck that the virus can retain its potency for 12 days in rabbits. Jacotot (1937), Huphauer and Skokovic (1938), Zichis (1939) showed that sheep are not highly susceptible to the virus of hog cholera. Jacotot(1939) showed that the virus can be transmitted in calves,goats,cats and monkeys. Goldman and Pehl (1955) maintained hog cholera virus in 4-6 day old suckling mice and carried up to 57 passages without loss in virulence for swine. Hutyra et al (1946) while quoting Tenbroeck stated that virus was potent for 7 days in guineapigs, 10 days in white rats and 12 days in birds.

The biological character was first investigated by Uhlenhuth and his co-workers as quoted by Hutyra et al (1946). The virus was reported to be spherical of 22 to 30 mμ and comes under the smaller group of viruses (Reagan et al,1951).

Dorset et al (1908) discovered in North America that pigs could be protected by sero-vaccination. McBryde & Cole (1936) prepared killed vaccine by means of crystal violet. Boynton et al (1937,1938,1941) produced a killed tissue vaccine which had an immunising capacity of 6 months. Koprowski (1946) and Baker (1946) independently attenuated the virus by passing through rabbits. The virus thus treated had a high antigenicity and low pathogenicity.

Numerous investigators have given detailed reports of

the clinical symptoms, gross lesions, histopathological changes, haematological changes, other tests, and they have been reviewed in detail in the respective chapters ahead.

MATERIALS AND METHODS

MATERIALS AND METHODS

Local breed of apparently healthy pigs aged about 7-8 months and average live weight of 20 lbs each were obtained. The animals were purchased on the spot from Chitkohra mohalla of Patna.

The pigs were purchased in the last week of April and were maintained on a clean, dry and airy place. All of them were given the same standard ration, ad lib water supply and no grazing was allowed throughout the course of the experiment. They were also provided with some soil for preventing iron deficiency.

Utensils for feeding and water supply were separate for healthy and diseased lots. The sheds were daily cleaned in the morning with disinfectants. The attendants washed their hands and legs with disinfectants before entering and after leaving the sheds. The attendant was instructed to clean the shed occupied by the healthy one first and then those of the infected ones.

Stool examination for parasitic ova and protozoal parasites were carried out and blood examined for blood parasites also.

In the present study the experimentally infected pigs were slaughtered at different time intervals for studying the various gross and microscopical pathological changes.

Blood collection and examination:

The blood collection was done aseptically in the morning by the method described by Carle & Dewhirst (1942), from the

anterior venacava every morning before the administration of food. Two ml. of blood used to be collected with a 19 gauze needle and a 5 ml. all glass sterilized syringe. The blood was immediately transferred to a small vial containing 0.1 ml. of anticoagulant (Heller & Paul proposed combination of 0.8 gms. of potassium oxalate and 1.2 gms. of ammonium oxalate per 100 ml. of distilled water) per ml. of blood and was evaporated to dryness in an oven at 60°C . The vials were shaken gently to mix the anticoagulant with that of blood in order to avoid clotting.

A few drops left in the syringe were put on two clean slides and uniform smears were prepared for the differential count.

The blood collected in the anticoagulant vial was used for the total R.B.C.,W.B.C.,Haemoglobin percentage, sedimentation rate and Packed cell volume.

Total R.B.C. & W.B.C. counts:

A modified R.B.C. diluting fluid described by Sastry & Dhanda (1962) was used for the count. Blood was drawn in scrupously clean R.B.C. diluting pipette upto 0.5 mark and the diluting fluid was sucked upto 101 mark. The dilution was thus 200 times. The improved double Neubauer's counting chamber was used. The count was made from the central square, its four corner and one central smaller square was counted. The number was multiplied by 10,000 which gave the number of cells per cumm.

For leucocyte count Turk's fluid was used. Blood was taken upto 0.5 mark in the W.B.C. diluting pipette and the diluting

fluid upto 11 mark, thus making the dilution 20 times. The cells in four big corner square (1 mm.sq.) were counted and was multiplied by 50 which gave the number of cells per cumm.

Haemoglobin estimation:

Haemoglobin percentage was obtained by the Sahli type of haemoglobinometer Farstab (16 gms = 100%) by the acid haematin method. The mixture was diluted with distilled water and other instructions followed as supplied with the instrument.

Erythrocyte sedimentation rate and Packed cell volume:

Oxalated blood was loaded in the clean,dry Wintrobe tube with a uniform bore of 3 mm. and a double 10 cm. scale caliberation with millimeter division. Blood was filled upto the 'O'mark of the tube and were allowed to stand vertically. One reading was taken at the end of hour. The Wintrobe tubes were then centrifuged at the speed of 3,000 r.p.m. for 45 minutes and then for 15 minutes for adequate packing to determine the corpuscular volume. This was recorded as the number of ml. of cells per 100 ml. of blood.

Blood films for differential count:

Scrupulously clean,dry and greeze free polished slides were used. Uniform blood smears were prepared and stained by Leishman's stain. The"battlement system" was adopted and 100 cells per 5000 leucocyte total count on an average were counted which were classified under Monocytes,lymphocytes,Neutrophils, Eosinophils and Basophils.

Amylase and Haemolysis tests with Pancreas:

The tests were done with pancreatic extract as described by Taylor (1961,1962) and slightly modified by Tadeusz Wijaszka (1964). About 5 gms. of sample were taken from the anterior part of the pancreas, ground and 10 ml. of saline solution added, shaked several times and allowed to stand for 30 minutes at room temperature.

Blood was collected from the ear of rabbits and mixed with Alsever's solution in proportion of 1:1. The mixture was later diluted with normal saline solution in proportion of 1:10.

A 1% starch solution was prepared prior to use and autoclaved. The iodine solution as an indicator was prepared by dissolving 5.5 gms. of iodine and 11.0 gms. of potassium iodide in 250 ml. of water.

Two tests were carried with the extract from pancreas.

1. Haemolysis test
2. Amylase test (Starch hydrolysis test)

Haemolysis test:

To 4 drops of pancreatic extract was added 3 drops of normal saline and 3 drops of diluted rabbit blood. The contents were kept on a water bath at $40^{\circ}C$ and the degree of haemolysis was observed after one hour.

Amylase test (Starch hydrolysis test):

Three tubes were taken each containing 4 drops of pancreatic extract to which 1,2 and 3 ml. of 1% starch solution was added to each of the tubes. The tubes were allowed to be

kept in a water bath at 40°C for 5 minutes and then 1 drop of iodine solution was added to each tube with subsequent reading of the shade of colour.

Amylase Estimation in Blood:

Amylase was determined in blood by adopting the technique as given in the manual of the Fischer clinical electrophotometer. Serum was used for the test.

Preparation of tissue and staining:

The different organs of infected pigs were preserved immediately after autopsy in 10% formal-saline and Zenker's solution for histopathological examination.

Tissue sections were stained with Haematoxylin and Eosin. Different tissues were also stained by a variant of Stovall and Black method to demonstrate the presence of inclusion bodies if any.

Cultural examination for secondary organisms:

Cultural tests were also carried out on materials collected from animals which died of the virus to find out the presence of secondary bacterial infection chiefly with reference to Salmonella and Pasteurella.

The present study included 12 healthy male and female pigs that were subjected to different haematological tests during the pre-infection period. Three readings were done on all the healthy pigs to obtain normal haematological values.

During this period the faeces of all the pigs were examined daily for parasitic ova by direct, floatation and

sedimentation methods. Temperature of all the animals were recorded morning and evening. Blood was also examined for protozoal parasites. Those animals found free from any disease and showing normal rectal temperature were divided into two lots of six each and were kept under observation for about 10 days before infection.

In the first batch 5 pigs were infected with the American strain of hog cholera virus obtained from Livestock Research Station, Patna intramuscularly with 1 ml. of 1 in 100 dilution of the virus in the thigh region. One animal which was kept as control was inoculated with 1 ml. of normal saline solution in the same region as above.

The second batch of another 5 pigs were infected with the local strain of virus obtained from Livestock Research Station, Patna as above.

Haematological studies were carried out daily during the post-infection period from 24 hours after infection and onwards prior to their killing; daily in the morning. One pig in each batch was killed at intervals of 7 days, 9 days, 11 days, 13 days and one infected pig was left till it succumbed taking the full course of the disease. The tissues were collected for gross and histopathological changes. Similar haematological studies were done on control pigs and the same were also slaughtered at the end of experiment for studying normal gross and histological structures. The temperature of the infected and normal animals were recorded daily morning and evening.

The amylase estimation in blood was performed on the second group of pigs only and readings were taken on the 5th,

& 7th day of all the animals, as well as on the dates they were killed i.e. 9th day,11th day,13th day and so on. Pancreatic amylase tests were also performed on both the batches.

The data obtained includes total R.B.C.,W.B.C., differential count, haemoglobin percentage,Erythrocyte sedimentation rate, Packed cell volume, amylase in blood and presence of amylase in pancreas.

REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON CLINICAL SYMPTOMS AND GROSS LESIONS.

REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON THE CLINICAL SYMPTOMS AND GROSS LESIONS

Salmon (1889), De Schweinitz & Dorset (1903), Dorset (1904), Bolton (1913), Dimock (1916), Hoskins (1916), Brunschwiler (1925), Frohner (1925), Uhlenhuth (1929), David & Schwarz (1930-31), Roehrer (1931), Seifried (1931), Seifried & Cain (1932), Delez (1933), Kernkamp (1939), Stovder (1945), Hutyra et al (1946), Henning (1949) are some of the earliest workers who described the symptoms and lesions of Swine fever.

Since 1950 till date a number of workers have studied in detail describing the symptoms and lesions of Swine fever. Dunne et al (1952a), (1952b); Pallaske (1953, 1955), Dunne et al (1957), Zadura (1958, 1959), Dunne (1961), Luedke & Dunne (1961), Weide et al (1962), Keast et al (1962), Urman et al (1962), and Dunne (1963) have described the symptoms and lesions of Swine fever in greater detail.

In India Sapre et al (1962), Murthy et al (1962), Mundu et al (1963), Narayana & Rao (1964), Saxena & Bisht (1964) have also dealt with similar symptoms and lesions of this disease as reported by the above workers.

Dunne (1963) stressed upon eight major clinical signs strongly suggestive of the disease.

Temperature:- In acute cases of the disease the temperature usually ranged from 105°F to 107°F, and in a few cases temperature as high as 108°F to 109°F has been recorded. In experimental cases the temperature reaction started within 2-6 days after inoculation of the virus and the peak was usually attained in 4-8 days. The temperature was maintained at its height throughout

the course of the disease and later declined becoming subnormal just before death (Hutyra et al,1946).

Eye discharge:- Dunne (1963) regarded conjunctivitis and lachrymation as the first sign of Hog cholera in white pigs. The discharges later became copious, thickened and glued the eye lids, and this was considered to be characteristic of the disease. The symptoms were noticed within 4-7 days after exposure to the virus. Bolton (1913), Dimock (1916), Stouder (1945) and Hutyra et al (1946), observed similar symptoms with slight differences in the nature of the discharges.

Anorexia:- Dunne (1963) described anorexia as one of the first signs of illness and was usually observed with the onset of temperature. Bolton (1913), Stouder (1945) and Hutyra et al (1946) observed loss of appetite and increased thirst.

Lassitude progessing to Ataxia:- Dunne (1963) described dullness and reluctance in movement during the early part of the disease; and having a "meditative attitude". Bolton(1913), Hutyra et al (1946) observed the drooping of head and arched back appearance of ailing pigs. Dimock (1916) observed the evidence of weakness in the hindquarters.

In the later stages Dunne (1963) found lack of muscular co-ordination and ataxia of the hindquarters which gave a characteristic weaving gait of Swine fever.

Vomtion:- Dunne (1963) described vomition of a yellowish fluid containing bile usually at the peak of temperature. Bolton(1913) Hutyra et al (1946) observed similar signs. Vomition started from 4-8 days after the exposure of the virus and continued till the death of the animal.

Constipation or diarrhoea :- Generally constipation developed first during the period of initial high temperature and was followed by marked severe watery yellowish diarrhoea. Constipation started 5-8 days post-infection and lasted till death. Diarrhoea which usually started 6-10 days after infection remained intermittent till death. Bolton (1913), Dimock (1916) and Hutyra et al(1946), stated that constipation and diarrhoea streaked with blood or with strings of mucus were common in Swine fever.

Discolouration of skin:- Erythema was observed early in infection but marked changes of the skin colour was seen in the terminal stages of the disease and was attributed to the sludging of blood, vascular damage and weakness of the heart. The colour varied from red to dark purple in shade and were marked on the abdomen, ears, legs and snout etc.Bolton(1913),Dimock (1916) and Hutyra et al (1946), all observed skin discoloration at different sites of the body in hog cholera.

Convulsions:- Convulsions were neither prominent nor usual symptoms but were noticed in a few cases mostly in young ones and it usually started from the 6-10th day after the exposure of the virus. Dunne (1963) described the sequence in convulsions, "Animals stiffen, fall on their sides with their heads drawn back, some times squeal and then begin to paddle and struggle violently", Great pain was noticed during the convulsive period and death usually followed soon in majority of such cases. Convulsions have been referred, to be caused by a specific strain of hog cholera virus (Dunne et al,1952a), but has also been

observed though rarely in cases caused by other strains (Dimock,1916; Brunschwiler,1925). Quin (1950) observed that convulsions occur in per-acute cases of hog cholera,while Hutyra et al (1946) mentioned that convulsions occurred only in exceptional cases.

Other symptoms:- Hutyra et al (1946),while discussing Swine fever, described in some cases inflammatory and diphtheroid changes in the buccal mucous membrane, swelling of tonsils etc. Dysphagia and laboured respiration were described due to the inflammed pharyngeal cavity. Spasmodic cough, asthmatic breathing, chest pain has also been seen along with acute or subacute croupous pneumonia or pleuro-pneumonia.

Nasal discharge, hindQuarter weakness and its paresis were reported by workers.

Huddling, piling or isolation in a corner by the affected animals have also been widely reported.

Hutyra et al (1946) described the symptoms of chronic Swine fever in which the temperature may be slightly elevated or may be almost near normal occasionally. Emaciation, periodic diarrhoea, cough, and dysponea were common. Wrinkling of skin has been reported. The buccal mucous membrane chiefly of the gums and pharynx showed diphtheroid changes. The affected animals became emaciated their abdomens being drawn up and the back arched. Dunne (1963) had observed partial alopecia and thining of the bristles. Such cases were found to survive upto 95 days (Dunne et al,1955).

Dunne (1959),while discussing hog cholera referred to

peracute, acute, subacute and chronic cases of the disease and the different forms took 4-7 days, 8-19 days, 20-29 days and 30-95 days respectively.

The incubation period upto the temperature elevation in experimental animals usually ranged between 3-6 days, though some times as low as 2 days have also been recorded (Manninger and Csontos cited by Hutyra et al,1946). Much longer incubation periods of 13-21 days have also been reported (Preisz, Hutyra, Koves cited by Hutyra et al,1946).

Stouder (1945) had reported 90-100% deaths in acute cases of Swine fever. Hutyra et al (1946) had described 80-90% mortality in areas which faced this disease for the first time but in notorious areas 30-40% deaths were more common which some times came as low as 5-10%. Murthy et al (1962), and Mundu et al (1963) recorded almost 100% mortality in India.

Present observations:-

In the present observations the earliest rise of temperature was noticed from 3-5 days in the first batch infected with American strain of Swine fever virus and 4 days in the second batch infected with the local strain of Swine fever virus. A temperature range of 104-108°F was recorded in both the groups and in most cases temperature of 105-106°F was found, which was maintained by the animals although with very slight variations. The two animals in the two groups which were allowed to die taking the full course of the disease, maintained throughout high temperature and showed slightly subnormal temperature only just before death.

With the rise of temperature the animals were noticed to be dull, piled upon one another and usually confined to a corner. They showed little interest in food. They moved reluctantly, would come to the feeder, eat a few bits and then go back again to the corner. An increase in thirst was also observed.

They had a ruffled coat and were seen to shiver with backs arched. When they were driven to move, they grunted and moved only slightly.

With the rise of temperature and even in some cases a day earlier all the pigs in the two groups showed characteristic conjunctivitis and watery discharge (3-5 days post-infection). The conjunctivitis worsened, the discharges became thick and later on muco-purulent which glued both the eye-lids (Figures 1 & 2). Both the eyes were affected and the upper lids were very much swollen.

Nasal discharges were also seen in 5 out of the 10 cases which later on dried over the snout and formed crusts which resulted in panting of the animals. The respiratory distress was found to be very pronounced in two cases in the first batch, one killed 13 days and the other which succumbed after a full course of the disease.

All the animals first showed constipation during the first 6-7 days after infection, later on in two animals killed after 13 days showed intermittant watery diarrhoea. Marked watery diarrhoea mixed with blood and mucus were observed in terminal stages in the animals which were allowed to die after

a full course of the disease i.e. in 16 & 18 days.

From the 11th day onwards the animals started showing characteristic weaving gait due to muscular incoordination, hind leg weakness, staggering and crossing of the hind legs when forced to move (figures 3 & 4).

Slight nervous symptoms were observed in three pigs, one in that was killed on the 13th day and in the other two which were allowed to die. The animals usually showed circling movements, hind leg weakness and posterior paralysis. The animals were unable to support their body weight on their hind limbs.

The two animals which were allowed to run a full course of the disease died after 16-18 days after infection.

No vomiting or skin discoloration was observed in any of the animals.

GROSS LESIONS:

Stomach:

Bolton (1913) observed punctiform haemorrhages in the serosa and mucosa of the stomach. Dimock (1916) and Hutyra et al (1946) reported congestion and swelling of the mucous membrane. Kernkamp (1939) described stomach as an uncommon site for lesions and were usually in the form of petechiation and ecchymoses of the mucosa or serosa or both. He also observed ulcers which were found chiefly in the fundic region. The ulcers varied in number. He observed haemorrhages in the mucosa in 8.7% haemorrhages in the serosa in 3.9% and ulcerative changes in 3.6%

1.2% of the cases out of the 84 artificially infected animals with swine fever.

Murthy et al (1962) reported congestion of the organ.

Present observations:

The mucous membrane of the small intestine was found to be congested and oedematous. In one case patchy congestion was also observed. Congestion and oedema of the mucous membrane of the small intestine was more severe and frequent (4 out of 5) in the second group infected with the local strain of the swine fever virus.

Large intestine:

Salmon (1889), Deschweinitz and Dorset (1903), and Bolton (1913) reported the presence of button ulcers in animals suffering from swine fever. This is one of the earliest recognised lesion. Kernkamp (1939) observed haemorrhagic changes involving the mucosa and serosa. Ulceration of the large intestine with concentric or annular rings of necrosed tissues gave the edges of the ulcers an irregular and rough appearance - button ulcers. The ulcers varied in number (1 to 47) and in size also. The usual sites were the proximal ends of the caecum and first part of the colon. He found haemorrhages in mucosa in 24.6%, haemorrhages in the serosa in 6.3% and inflammatory changes characterised by button ulcers in 13.8% during his experiments upon 48 artificially infected animals and 286 field cases of swine fever. Dunne et al (1952a) based their observations upon 84 artificially infected animals with swine fever and found

20.2% with button ulcers, 21.4% with acute catarrhal colitis and 10.7% with colonic petechiation. Pallaske et al (1955) found swine fever'buttons' only in chronic cases.

Bolton (1913), Pallaske (1953), Kernkamp (1939), and Dunne (1961) stated the diagnostic value of the lesions in swine fever.

Murthy et al (1962) observed extensive ulceration or discreet ulcers with raised edges, involving the lymph follicles.

Present observations:

In the present observations during the early part (7 to 9 days) of the disease showed oedema and congestion of the caecum and colon. The mucous membrane showed patchy as well as diffuse areas of congestion (figs.6 & 7). In one of the cases infected with the local strain of hog cholera virus and sacrificed on the 11th day after infection showed severe congestion of the mucous membrane of caecum and colon and numerous (above 100) ulcers ranging from pin head size to that of pea. The bigger ulcers had irregular and rough edges and were packed with brownish material. The bigger ulcers were more common in the caecum and a few were close and upon the ileo-caecal valve. The smaller ones were like indescernible necrotic areas covered with faecal plaques and congested (figs.8 & 9). The two animals which were allowed to die after full course of the disease also showed such ulcers but in lesser number (figs.10 & 11).

Liver and gall bladder:

Bolton (1913) described petechial haemorrhages to be more

common than large haemorrhagic infarcts and areas of necrosis. Kernkamp(1939) found chronic passive congestion in 6.2% out of 48 experimental cases while Dunne et al (1952a) found fatty metamorphosis in 7.1% out of the 84 artificially infected pigs with swine fever.

Ivanov et al (1952a) reported on the macroscopical lesions of the gall bladder and cystic ducts on a large number of animals infected with swine fever. They observed haemorrhage and hyperaemia in 64% of the cases. Quin (1950),and Dunne et al (1952a) observed mild to severe petechial haemorrhages in the mucous membrane of gall bladder. Luedke et al (1961) examined 265 experimentally infected cases of swine fever and observed severe to mild areas of infarcts in 106 of the animals. They found the gall bladder frequently shrunken with mostly dark, thick and granular bile. The lesions in gall bladder were supposed to be pathognomic by the authors while Draghici et al (1959) did not agree with this.

Murthy et al (1962) reported swelling and infarction of the liver and streaks of congestion in the mucous membrane of the gall bladder.

Present observations:

Moderate congestion of the liver was observed in almost all the cases.

The gall bladders were shrunken in six, enlarged in one and normal in 3 of the ten cases studied. In one gall bladder from the group infected with the American strain of swine fever

virus showed petechiation of the mucous membrane of the gall bladder. Two of the cases (9 & 11 days post-infection) which were infected with the local strain of swine fever virus showed severe congestion, petechiae and streaks of congestion of the mucous membrane of the gall bladder (fig.12). The bile in such cases were granular, dark and tinged with blood.

Kidneys:

Bolton (1913) described the speckled egg appearance of kidneys due to punctiform ecchymoses. Kernkamp (1939), working on a number of animals (48 artificially infected and 286 field cases of swine fever) reported small pin point haemorrhages and ecchymoses on the surfaces and parenchyma of the organ. The number of petechiae or ecchymoses were variable and ranged from 3 or 4 to several hundreds. These changes had been seen in the cortical and medullary zones as well as in the mucous membrane lining the hilus. He observed lesions in the kidneys of 95.8% of artificially infected, 91.9% of field cases in an over all average of 92.5%. Dunne et al (1952a) reported renal petechiation in cortex in 64.3%, ecchymoses in cortex in 15.5% and ecchymoses in the renal pyramids in 28.6% out of the 84 artificially infected animals with swine fever.

Pallaske (1953), and Pallaske et al (1955) described lesions of the kidneys as typical, whereas Bolton (1913), and Kernkamp (1939) supposed them as characteristic of swine fever.

Sapre et al (1962), Mundu et al(1963) described very briefly the lesions in kidneys, while Murthy et al (1962) reported

enlargement, subcapsular haemorrhage and "turkey egg" appearance of the kidneys.

Present observations:

In the present study the petechiation started from the 9th day and were very few in number. They were mostly located on the surface below the capsule and were found in the cortical region when cut. In a few cases slight congestion was observed macroscopically in the earlier stages. With the advance of the infection and mostly from 13th day onwards the petechiae became more pronounced and increased in number (fig.13). One of the two animals which were allowed to take the full course of the disease, infected with the American strain of hog cholera virus showed numerous pin point haemorrhages resembling "turkey egg" upon the surface of the kidneys (fig.14). The petechiae were numerous in the cortical region when cut (fig.15). The medullary portion showed severe congestion and haemorrhages in the renal pelvis. Slight accumulation of blood was also observed in the capsule. The kidneys from the other animal showed similar lesions except for decreased number (about 20) of petechiae (fig.16).

Urinary bladder:

Bolton (1913) observed the mucous membrane of the urinary bladder speckled with punctiform haemorrhages in swine fever. Kernkamp (1939) based on the observations upon 48 artificially infected and 286 field cases of swine fever, recorded petechiae and ecchymoses most frequently on the mucous membrane. The number

of petechiae and ecchymoses varied to a great extent. The haemorrhagic changes usually involved the mucous membrane and sometimes extended into the muscular coat. He observed these changes in 95.8% in the artificially infected group and 77.6% in the field group. When put together the above mentioned lesions were found in 83.2% of the cases. Dunne et al(1952a) observed cystic petechiation in 78.6% out of the 84 artificially infected cases with swine fever. Dunne (1959,1963) reported mild to moderate congestion as the commonest lesion. Severe diffuse and ecchymotic submucosal haemorrhages were regarded uncommon and were usually present in cases complicated with some bacterial infection.

Murthy et al (1962) also observed similar lesions.

Present observations:

Slight congestion of the mucous membrane of the urinary bladder was observed on the 7th day post-infection in one of the pigs infected with the local strain. Later on petechiation of the mucous membrane became more apparent and their number also increased with the progress of the disease. The petechiae appeared bright red. The pigs infected by the local strain started showing petechiation from the 9th day onwards and were found in all subsequent cases in increased numbers(fig.17). The other lot with the American strain started showing petechiation (fig.18) and ecchymotic chages from the 13th day onwards. Under this lot the animal which succumbed after taking the full course of the disease, showed extensive diffuse and ecchymotic

submucosal haemorrhage (fig.19). The mucous membrane was thrown into folds and the elevated areas contained blood and were bright red in colour. The walls of the bladder showed thickening.

Larynx and Epiglottis:

Hoskins (1916) recorded some degree of laryngeal haemorrhage in 75.4% out of the 500 pigs artificially infected with swine fever. Kernkamp (1939) observed petechiation and ecchymoses in these organs and found the oral surface of the epiglottis the most frequent site. He recorded 59.6% out of the 334 artificially and naturally affected animals showing these lesions. Dunne et al (1952a) found petechiation of the epiglottis and larynx in only 23.8% out of the 84 experimentally produced swine fever cases.

Murthy et al (1962) did not find any such lesion.

Present observations:

The petechiation on the epiglottis was seen to appear from the 13th day onwards. Two animals infected with the American strain and one with the local strain showed clear petechiation of the epiglottis. The lesions in the American strain group were more severe, the petechiae greater in number and were observed on both the surfaces of the epiglottis (figs.20,21,22 and 23).

Lungs:

Bolton (1913) described a characteristic mottling due to haemorrhagic infarcts in the lungs. Croupous pneumonia (Hutyra et al,1946; Pallaske,1953) and haemorrhages in lungs

were reported by Keast et al (1962).

Kernkamp (1939) based upon 334 artificially and naturally infected swine fever cases, described capillary haemorrhages appearing as petechiae and ecchymoses, under the visceral pleura and in the lung substance. He observed petechiae and ecchymoses in 27.2%, chronic passive congestion in 44.9% and inflammatory changes chiefly broncho-pneumonia in 40.4% of the cases studied. Dunne et al (1952a) found broncho-pneumonia in 58.3% and ecchymoses in 15.7% out of the 84 artificially infected pigs with hog cholera.

Murthy et al (1962) also observed similar lesions in India.

Present observations:

No lung lesion except for slight congestion was observed in the group infected with the local strain of swine fever virus. The group infected with the American strain of swine fever virus showed slight congestion upto the 11th day. The animals autopsied on the 13th day showed patchy congestion and consolidation of the apical and diaphragmatic lobes (fig.24). The mucous membrane of trachea also showed congestion. The animal which was allowed to take the full course of the disease showed several small petechiae, ecchymoses and haemorrhagic infarcts in the lungs(fig.25).

Lymph nodes:

Bolton (1913), Dimock (1916), Pallaske (1953) stated that lymph glands were congested or haemorrhagic in swine fever. Seifried et al (1932b), described three types of lesions

viz; swelling and hyperaemia, haemorrhagic infiltration specially marked at periphery and dense infiltration with red blood cells imparting a dark red colour to the nodal parenchyma.

Kernkamp (1939), working on a significant number of animals (48 artificially infected and 286 field cases) observed similar lesions as described by Seifried et al (1932b). In the artificially infected group and in cases from field 91.6% and 82.1% of the nodes showed lesions respectively. When put together 83.5% of the lymph nodes showed lesions in swine fever.

Dunne et al (1952a) found haemorrhagic lesions in 96% out of the 84 artificially infected pigs with hog cholera. The diffuse haemorrhages were more common (71.4%) than the peripheral ones (47.6%) while Kernkamp (1939) found the latter more frequent.

Ivanov et al (1952) reported from 55 swine fever infected pigs killed 4-7 days post-infection, showed the frequency of lesions in the lymph glands in the following order - renal, post mediastinal, gastric, cervical, superficial inguinal, supramammary, portal, bronchial, popliteal, hypogastric, jejunal, colic and anal. Dimock (1916), Hutyra et al (1946) and Urman et al (1962), referred to the glands as enlarged, moist and with peripheral haemorrhage.

Dunne (1961,1963) pointed out the diagnostic value of the peripheral haemorrhages in lymph glands in swine fever.

In India Sapre et al (1962), Murthy et al (1962), Mundu et al(1963), and Narayana et al (1964) have very briefly described the lesions.

Present observations:

In the present observations renal, post mediastinal, gastric, superficial inguinal, supramammary, portal, bronchial, submaxillary and mesentric lymph glands were examined and all showed one of the three types of lesions. In the early part of the infection (7th day) enlargement and hyperaemia were usually observed in most of the lymph glands examined. With the advancement in disease peripheral haemorrhage was more commonly noticed, the blood getting accumulated in the spaces between the capsule, probably trabeculae and parenchyma of the gland(fig.26). The centre of the haemorrhagic zones appeared lighter in colour giving the glands a "strawberry like" appearance. A few lymph glands chiefly gastric, sometimes the bronchial and mediastinal showed diffuse haemorrhage. They looked dark red to black (fig.27 & 28). All the three types of lesions were observed in advanced cases (9th, 11th, 13th day and animals that died), but in the less advanced ones (7 days), swelling and congestion were more commonly obserserved than peripheral haemorrhages. Hyperaemia and swelling were common in the superficial inguinal, supramammary and submaxillary lymph glands whereas peripheral and diffuse haemorrhage were more commonly met with in the rest of the lymph glands examined.

One of the most striking feature was that all the animals showed lesions in the lymph glands though varying in intensity with the progress of the disease. The frequency of occurrence of the lesions was thus 100 per cent.

Spleen:

Bolton (1913) described extreme swelling of spleen with larger or smaller haemorrhagic infarcts. Seifried et al (1932b) based his observations on 31 autopsies on swine affected with hog cholera and were more or less similar to Kernkamp's(1939) observations,who had made extensive studies on a large number of animals. Kernkamp (1939) based his observations upon 48 artificially infected cases of hog cholera and 286 cases collected during past 20 years of naturally infected animals. He found mostly on the borders well defined haemorrhagic areas, dark red or black in colour, usually slightly raised above the surrounding surface of the spleen and the infarcts varied in size and number. Delez (1933) observed similar lesions. The frequency of the haemorrhagic infarcts as encountered were 68.7% in the artificially infected lot and 58.7% in the field cases(Kernkamp,1939), 27.4% of the 84 experimentally infected cases (Dunne et al,1952a), 20 out of 37 (Ivanov et al,1952), 30-60% (Zadura,1958), and 25-65% (Dunne,1963).The frequency of the anaemic infarcts were rare (Kernkamp,1939; Hutyra et al,1946; Zadura,1958).

In some chronic cases chronic passive congestion was found. Spleen was found to be enlarged and firm. Haemorrhagic changes were also observed by several workers.

Roehrer (1931,1932) believed that with the advance of the disease, the frequency of splenic infarcts decreased.

The occurrence of the splenic infarcts were supposed to be pathognomonic by David & Schwarz(1930-31),Pallaske(1953),

Pallaske et al (1955), Zadura (1958),and Dunne (1961). Dunne (1963) stated the relatively rare occurrence of splenic infarcts in other diseases.

In India, Sapre et al (1962)& Mundu et al (1963) described in short while,Murthy et al (1962) had given in some detail the splenic lesions and were similar to those recorded by other workers.

Present observations:

In the present observations two spleens from animals infected with the American strain of hog cholera virus and sacrificed at 7 & 9 days after infection showed several, raised dark haemorrhagic areas about the size of pea. These infarcts were observed mainly on the margins and borders and very few on the body of the spleen. The spleen also showed enlargement and congestion (fig.28). As the disease progressed the spleen appeared smaller in size and showed slight congestion (figs.26 & 27). In a few cases the spleen looked almost normal. In this experiment 20% of the animals showed haemorrhagic infarcts and anaemic infarcts were not observed.

Heart and Pericardium:

Bolton (1913) and Dimock (1916) observed ecchymoses in the cardiac walls. Kernkamp (1939) recorded petechiae and ecchymoses on the epicardium as well as on the endocardium. The pericardium also showed similar legions. He found lesions in 29% out of the 334 cases of artificial and natural swine fever. Hutyra et al (1946) also observed haemorrhages under the

endocardium. Dunne et al (1952a) found epicardial petechiation in 13.1% and myocardial degeneration in 35.7% out of the 84 artificially infected animal. Murthy et al (1962) also observed lesions in the heart.

Present observations:

Only ~~in~~ one case that died due to the American strain of swine fever virus after taking the full course of the disease showed several petechiae and ecchymoses on the epicardium(fig.29). The endocardium and the valves did not show any gross abnormality.

Brain and spinal cord:

Dimock (1916) and Kernkamp (1939) observed petechial haemorrhage in the brain of pigs affected with swine fever. Seifried (1931) did not consider the gross lesions of the brain as diagnostic. He observed marked congestion of the blood vessels and in a few cases pronounced haemorrhages and oedema in the meninges, which sometimes covered large areas of the brain and spinal cord. The liquor cerebri in such cases showed a slightly yellowish red or red colour. Dunne et al (1952a) found cerebral congestion in 72.6% out of the 84 artificially infected pigs with swine fever. Murthy et al (1962) also observed congestion of the meninges.

Present observations:

The brain showed congestion which increased in severity as the disease advanced (fig.30). In both the batches congestion and an excess of liquor cerebri tinged with blood were found. The spinal cord,however,showed no gross abnormality.

Skin:

Bolton (1913) described lesions in the skin usually behind ears, medial aspect of thighs, over the abdomen and other parts of the body, either sprinkled with punctiform haemorrhages or having a more diffuse irregular haemorrhage in the affected parts. Kernkamp (1939) reported the above mentioned lesions and also noted oedema, necrosis and sloughing of epidermis in some cases. He found 25.1% skin lesions out of the 334 cases of swine fever (artificially and naturally affected) while Dunne et al (1952a) observed erythema in 29.8% out of the 84 artificially infected cases.

Present observations:

None of the animals showed grossly skin lesions. One pig infected with the American strain which died after a full course of the disease showed petechial haemorrhages and ecchymoses in the subcutaneous tissue in the thigh region of both the sides (fig.31).

Other Organs (Pancreas, Adrenals, Conjunctivae, Tongue & Tonsils):

Kernkamp (1939) found no significant gross lesions in the pancreas and adrenal glands of the 334 animals affected with swine fever.

Bolton (1939), Dimock (1916), Kernkamp (1939), Stouder (1945), Hutyra et al (1946), Dunne et al (1952a), and Dunne (1963) reported congestion of the mucous membranes of the eyelids with discharges of varying nature. Dunne et al (1952a) found conjunctivitis in 70.2% out of the 84 artificially infected cases

with swine fever. Dunne (1963) supposed this lesion as one of the diagnostic signs of hog cholera.

Hutyra et al (1946), while describing swine fever, stated the occurrence of haemorrhagic inflammation, often with croupous deposits and necrosis in the tongue of pigs affected with this disease.

Hutyra et al (1946) described ulceration of the tonsils with irregular borders and smeary coatings. Dunne et al (1952a), while working on 84 artificially infected animals with swine fever, found tonsillitis in 38.1% of cases. Maurer (1956) believed necrosis of the tonsils as a result of infarction.

The lesions in the tongue and tonsils were regarded as uncommon (Palliola,1963).

Present observations:

Grossly no lesions were seen in pancreas except for mild to moderate congestion of the blood vessels. The adrenals also did not present any lesion.

In both the groups all the animals exhibited severe congestion of the mucous membranes of the eyelids with watery discharge which later became thick and mucopurulent. These lesions were found from the third day post-infection and observed althrough.

The lesions in the tongue were noticed in both the groups and were observed when the disease had fairly progressed. One animal affected with the American strain of swine fever virus showed lesions on the 13th day while the two which were allowed

to take the full course of the disease in the two groups also exhibited similar lesions. The lesions consisted of haemorrhagic inflammation simulating infarcts of the size of pea and generally located on the borders of the tongue (fig.32). The animals affected with the American strain of swine fever virus showed more severe lesion than the one infected with the local strain (only one infarct).

No gross lesion in the tonsils were seen.

REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON HISTOPATHOLOGICAL CHANGES

REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON HISTOPATHOLOGICAL CHANGES

Stomach, small and large intestine:

Dimock (1916) observed haemorrhages in the mucosa and submucosa but never in the serosa of the stomach and large intestine of pigs suffering from swine fever. Ivanov et al (1956) found hyperaemia, petechiation and lymphocytic in-filtration of the stomach and intestines in 150 pigs killed 4-6 days post-infection. Dunne et al (1952b) reported coagulation necrosis bordered by leucocytic infiltration in the ulcerated stomach of pigs suffering from swine fever. Degenerative changes in the blood vessels of the submucosa were also observed.

Dunne et al (1952a) found mild to moderate catarrhal inflammation of the small intestine and considered it as a relatively unimportant lesion.

Seifried et al (1932b) studied the lesions in the intestinal tract in the early phase of the disease and found degenerative lesions chiefly in the blood vessels of the mucosa of the large intestine. They seldom found necrotic foci in the early stage of the disease. The capillaries in the mucosa in between the glands showed intense dilatation and were packed with red blood cells. Slight degeneration of the epithelial cells, slight oedema of the connective tissue and frequent round cell accumulations around the blood vessels

in the mucosa were also observed by them.

The most interesting lesion is the button ulcer of the large intestine. Dunne et al (1952b) found evidence in support that button ulcers arose from infarctions in the intestinal mucosa. They showed the genesis of the ulcer from the very beginning, and observed a definite area of coagulation necrosis in the mucosa with leucocytic infiltration which had just begun separating the healthy from the necrosed one. As the process advanced the leucocytic infiltration also increased which forced the necrosed plug of mucosa into the intestinal lumen. The artery at the base of the lesion showed marked hydropic degeneration of the endothelial cells and thus hindered the passage of blood. The loss of necrosed tissue followed by the invasion of the ulcerated area by the intestinal bacteria finally resulted in the formation of button ulcer. The usual site of the ulcers were the proximal ends of the caecum and first part of the colon.

Present observations:

The stomach in one case which died after the full course of the disease with the American strain of virus showed microscopic lesions. Haemorrhages were seen in the mucous and submucous layers (fig.33). An area of necrosis resulting in ulceration of the mucosa and a dense area of infiltrating leucocytes in the submucosa and in the bordering mucosa were seen (figs.33,34 & 35). The blood vessels showed the usual degenerative changes viz. hydropic degeneration and

hyaline changes (fig.36).

The small intestine showed limited lesions and consisted of mild to moderate haemorrhages in the lamina propria. The lesions were frequent in the group infected with the local strain of virus as compared to the other group infected with the American strain of virus.

The large intestine showed extensive lesions in the group affected with the local strain than that affected with the American strain of swine fever virus. The colon and caecum from the local strain group showed extensive haemorrhages in the mucosa and submucosa and in a few cases extended even to the muscular layers. Oedema in the submucous layer was also observed (fig.37). The goblet cells showed hypertrophy and several intestinal glands were virtually washed out by haemorrhages. Several lymphoid follicles showed rarefaction while few others showed hyperplasia. The blood vessels showed congestion and were almost packed to their capacity. Similar changes as described above were also noticed in the ileo-caecal valve (figs.38 & 39).

The ulceration of the colon, caecum and the ileo-caecal valve was noticed in cases destroyed from the 11th day onwards in both the groups with differences only in the number of ulcers which were more in the group infected with the local strain of virus. The lesions comprised of coagulation necrosis of a well defined area of the mucous membrane of either colon, caecum or the ileo-caecal valve. The necrosed area showed lack

of cellular details and were eosinophilic in appearance (figs.37,38 & 40). Leucocytic infiltration in the submucosa and in bordering normal mucosa (figs.41,42 & 43) had forced the necrosed area of the mucosa out of the normal position. The fragmented portion was still seen near the ulcer. The blood vessels in the base of the lesion showed hydropic degeneration of the endothelium resulting in partial occlusion of the lumen (fig.44). The lesions found in the stomach, small intestine and large intestine are in close confirmity to that of Dunne et al (1952a,b).

Liver and Gall bladder:

Seifried et al (1932b) described vascular lesions in liver, some of which were associated with haemorrhages. In one case necrosis was observed extending from the central vein to one half of the affected lobule. They also found infiltration of the interlobular connective tissue with mononuclear cells or connective tissue proliferation around certain bile ducts and blood vessels as described earlier by Eberbeck (1931) and later by Pallaske (1954) and Urman et al (1962). Ivanov et al (1951a) found the parenchyma rarely affected and in 97% of the cases lesions were localised in the vascular endothelium. Urman et al (1962) also found swelling and liberation of Kupffer's cells. Kupffer's cells were seen with intranuclear inclusion bodies and such cells showed degenerative changes. Kuppuswamy (1962) observed degenerative changes in the hepatic cells and haemorrhages in swine fever.

Ivanov et al (1952a) studied the lesions in gall bladder in 163 experimentally infected animals with swine fever and in 13 with spontaneous infection. They found lesions in 67 cases, 4-6 days after infection which consisted mainly of the destruction of lymphatic tissue, formation of pseudo-membranes, haemorrhage and hyperaemia. Luedke et al (1961) found areas of necrosis in the mucosa due to infarction of blood vessels. The blood vessels at the base of the necrotic areas showed degeneration and narrowing of the lumen resulting in stasis of blood. They also found ulcers extending up to the tunica muscularis with considerable leucocytic infiltration at the border of the necrotic and living tissues. In many cases they found considerable vascular engorgement and small haemorrhages especially in the lamina propria.

Urman et al (1962) found no significant change in the gall bladder.

Present observations:

The liver in all the cases showed mild to moderate congestion. Degenerative changes viz. fatty changes were seen in varying intensity in most of the cases (fig.45). Slight infiltration of mononuclear cells in the portal areas were seen in some of the sections.

The gall bladder showed striking changes from the 7th to the 13th day in both the groups. The animals in the two lots which succumbed due to the disease did not show any microscopical changes. Lesions were found to be more extensive in

the group infected with the local strain than those of the American strain group. Severe haemorrhages were seen in the lamina propria and submucous layer. The blood vessels showed complete packing. Erosion of the epithelial cells due to the extensive haemorrhages were seen (fig.46). Slight necrosis of few tips of the mucosal folds were seen in one case which was sacrificed at 9 days interval in the local strain group. The observations are in agreement with Luedke et al (1961).

Kidneys:

Dimock (1916) observed degeneration of the renal tubules and haemorrhages in the glomeruli in cases of swine fever. Proescher and Seil (1917) and Proescher and Hoffmann (1924) found acute haemorrhagic glomerular and tubular nephritis. Bel (1927) reported interstitial haemorrhages inflammatory reactions of the glomeruli, degeneration of renal tubules and cuffs of mononuclear cells around the vessels. Luttschwager (1931-32) found microscopic haemorrhages in cases which showed no typical gross lesions in the kidneys. Seifried et al (1932b) described small scattered and diffuse haemorrhages in the interstitial tissue. They also found perivascular infiltration with macrophages and lymphocytes in advanced cases of swine fever. Haemorrhages in the renal glomeruli were frequent in the early stages of the disease while proliferation of the glomerular epithelium was rarely seen. The tubular epithelium showed varying degrees of retrogressive changes and intratubular haemorrhages were sometimes also observed.

Ivanov et al(1951b) considered lymphocytic cell infiltration around blood vessels, glomeruli and tubules as diagnostic for swine fever. Dunne et al (1952a) found extensive haemorrhages in interstitial tissue and tubular necrosis while Marcato et al (1963) observed focal glomerulonephritis and interstitial inflammation in 24 out of the 40 acute and subacute cases of swine fever. Kuppuswamy (1962) reported demarcated haemorrhages in the interstitial stroma, Bowman's spaces and in the tubules.

Present observations:

Microscopical lesions were seen in the infected animals of both the groups killed at 7,9 and 11 days though no appreciable gross lesions were evident at necropsy. Wide spread and extensive haemorrhages and degenerative lesions were encountered from the 13th day onwards. During the 7-11th day post-infection glomerular and intertubular haemorrhages were seen in the cortex as well as in the medulla(fig.47). Slight degenerative changes of the tubules were seen. The intima of the blood vessels showed hydropic degeneration. Slight mononuclear infiltration round the blood vessels were seen in a few cases.

Very extensive haemorrhagic lesions were seen in animals of both the groups from the 13th day onwards. Numerous haemorrhagic patches were seen infiltrating in the intertubular spaces of the cortex and medulla destroying the glomeruli and the tubules (figs.48 & 49). Haemorrhages were also seen in glomeruli, in the lumen of the tubules and in the renal pelvis. The blood

vessels showed the usual changes of swelling and proliferation of the intima together with retrogressive changes. Round cell infiltration around some of the blood vessels were also observed. Necrosis of the renal tubules along with casts were seen to occur in some of the animals in late stages of the disease (fig.50).

The lesions were found to be more extensive in the group infected with the American strain than in the local strain.

Urinary bladder:

Seifried et al (1932b) described haemorrhages of the serous and mucous surfaces of the urinary bladder in swine fever. Perivascular haemorrhages and round cell accumulations similar to those found in kidneys (already reviewed) were also reported.

Present observations:

The urinary bladder started showing the haemorrhagic changes in the lamina propria from the 9th day onwards in increasing intensity in the group infected with the local strain of virus. Slight haemorrhagic changes were also observed in the serous layer and the blood vessels in the submucous and muscular layers showed extreme congestion. One animal in the group infected with the American strain which died after full course of the disease, showed very extensive and diffuse haemorrhages in the lamina propria and in the muscular layers (figs.51 & 52). Large papillary projections due to proliferation

of the mucous membrane of the urinary bladder filled with red blood cells, destroying almost all the inner structures were seen. Degeneration of the muscle fibres of the muscular layer was also observed due to the extravasation of blood. The serous layer also showed haemorrhages. The other animal which was sacrificed at 13th day interval in the American strain group showed lesions similar to that of the group infected with the local strain of swine fever virus.

Lungs:

Dimock (1916) observed in swine fever the rupture of the finer capillaries throughout the substance of the lung resulting in the escape of blood in the air sacs and alveolar spaces and in the interstitial structures. Seifried et al (1932b) found lesions in the lungs due to the lesions occurring in the blood vessels. Dunne et al (1952a) reported broncho pneumonia in 58.3% and pulmonary ecchymoses in 15.7% out of the 84 artificially infected swine fever cases. Korn (1956) found congestion and oedema due to circulatory disturbance, during the first 12 days in experimental swine fever. Kuppuswamy (1962) also observed similar changes in the lung.

Present observations:

In the present observations the lot infected with the local strain of virus showed very slight congestion of the lung throughout the course of the disease. The other lot infected with the American strain started showing haemorrhagic and pneumonic changes from the 11th day onwards. The animal

killed on the 11th day showed slight peribronchial infiltration with mononuclear cells and exudate in the bronchioles were found to contain neutrophils as well as a few eosinophils. The one killed on the 13th day showed red and grey hepatization with surrounding areas of emphysema and collapse. The animal which was allowed to run the full course of the disease exhibited large areas of haemorrhages in the substance of the lung thereby destroying substantial areas of lung tissue(fig.53). Wide spread damage of the blood vessels was also observed. The bronchioles were seen packed with blood due to the haemorrhages in the lung tissue. Pneumonic changes and areas of emphysema and collapse were also seen at some places.

Lymph nodes:

Bel (1927) observed lymphatic hyperplasia, hypertrophy of the follicles and hyperaemia of capillaries in cases of swine fever. Nieberle (1930) pointed out that acute cases of hog cholera were not associated with haemorrhagic lymphadenitis. Roehrer (1930-31) stated that the haemorrhages and necrotic foci in the different parts of the nodes were due to the lesions in the blood vessels.

Later Seifried et al (1932b) gave a detailed histopathological account of the lesions in lymph nodes and described them under three heads. Under type I they reported hyperaemia and swelling of the lymph glands. Multiplication of reticulum cells and oedema of cell poor substance was observed. Perivascular necrosis of varying degrees, increase of eosinophilic

leucocytes and local hyperplasia of lymphoid tissues were also observed. Under type II they found characteristic haemorrhages in the cell-poor substance and marked necrotic foci which at times became extensive due to the fusion of several such foci and thus occupied large areas of the parenchyma. Under type III lesion they reported extensive accumulation of red blood cells in the entire cell-poor substance as well as in part of the lymphoid tissue. These accumulations caused extensive atrophy of the lymphoid tissue.

Ivanov et al (1952) while describing the frequency of the lesions in different lymph nodes observed necrosis of parenchyma as the most characteristic lesion in swine fever. Potel (1956) observed marked massing of eosinophilic leucocytes in the infected nodes. Urman et al (1962) and Bergmann (1964) found reticulosis, severe congestion, vascular degeneration, infiltration of lympho-reticular tissue with eosinophils, haemorrhages and necrosis of the lymphatic tissue. Urman et al (1962) also found the presence of acidophilic intranuclear inclusion bodies in the reticulum cells.

Present observations:

In the present observations, the different lymph glands from the animals killed at different intervals and those dying after the full course of the disease in both the groups showed lesions of increasing intensity with the progress of the disease. In the early stages (7-9 days) proliferation of the reticular cells and small to large areas of necrosis infiltrated

with increased number of eosinophils and a slight lymphoid hyperplasia were observed in the affected lymph glands (figs.54,55 & 56). Moderate hyperaemia was also seen.

With the progress of the disease the lesions in the glands became more extensive showing peripheral haemorrhages in the cell-poor substance and trabeculae, and extensive necrotic areas of the parenchyma (fig.57). Several lymph follicles were practically washed off due to the haemorrhages (fig.58) and most of them seen in the different stages of degeneration. In some of the glands even more extensive and diffuse haemorrhages were observed in the cell-poor substance resulting in replacement of the entire lymphoid tissues thereby causing extreme atrophy of the lymphatic glands (figs.59 & 60). Eosinophils disappeared in the cases sacrificed from the 11th day onwards. The blood vessels showed the usual degenerative changes.

No difference in the lesions has been noticed in the two groups on microscopic examination.

The observations are in close confirmity with that of Seifried et al (1932b), Potel (1956) and Urman et al (1962).

Spleen:

Dimock (1916) showed subcapsular haemorrhage and the substance of the organ generally haemorrhagic in character. Roehrer (1931-32) first reported the details of histopathological changes in spleen in swine fever. Seifried et al (1932b) later on also gave an equally detailed picture of the changes in spleen. They based their observations upon 30 cases and found

the most pronounced lesion in the follicular arteries specially at the apex of the wedge shaped infarcts. The blood vessels showed enormous swelling and hyalinization leading to the occlusion of their lumen. This resulted in the different degrees of coagulation necrosis in the parenchyma in the neighbourhood of the occluded vessels. In early stages necrosis was reported to be restricted to a single follicle but later on extended to the entire infarct involving even the trabeculae. Spleen which was not showing gross lesions showed degenerative changes of the blood vessels with perivascular haemorrhage and slight perivascular necrosis. A striking aplasia of the splenic follicles were also reported by them.

Delez (1933), Ivanov et al (1952), Zadura (1958) and Okaniwa et al (1962) observed more or less similar lesions in spleen while Urman et al (1962) also demonstrated the presence of intranuclear inclusion bodies in cells of the reticulo endothelial system of the spleen. Kuppuswamy (1962) observed intense haemorrhage and destruction of Malpighian corpuscles.

David and Schwarz (1930-31), Zadura (1958) and Okaniwa et al (1960) described the lesions in spleen diagnostic of swine fever.

Present observations:

In the present observations, more marked haemorrhagic lesions characterised by infarcts were seen to occur in the early part (7-9 days post-infection) of the disease and that two more

pronounced in the group affected with the American strain of swine fever virus. Large areas of haemorrhages just below the capsule were seen destroying huge areas of the spleen substance. The blood vessels chiefly the follicular or central arteries showed the usual degenerative changes, partial blockage of their lumen with hyalinization of their walls (figs.61 & 62). Several Malpighian corpuscles in the haemorrhagic areas were seen in different stages of degeneration (fig.62). Perivascular accumulation of blood in the vicinity of the follicular artery was also noticed along with peripheral necrosis. It looked as if blood was oozing out from the vessel walls (fig.63).

With the advance of the disease (from 11 day onwards post-infection) the haemorrhagic lesions became less prominent. The central arteries showed lesions as described above leading to perivascular red blood cells accumulation and necrosis of the spleenic corpuscles. The trabeculae and the capsule also showed slight thickening in the later stages of the disease. Moderate degree of haemorrhage was observed in such cases. A slight aplasia of the splenic corpuscles and mild haemorrhagic lesions in the advanced cases probably explains the decrease in size of the organ noticed.

The above lesions observed are in close confirmity with those of Roehrer (1931-32), Seifried et al (1932b), Delez (1933) and others except for the atrophy in the size of the spleen which does not appear to have been recorded so far.

Vascular system:

Proescher and Seil (1917) and Proescher and Hoffmann(1924)

observed acute endangitis along with a thromboangitis and an endarteritis in some organs. Bel (1927) found capillary hyperaemia responsible for haemorrhages. Roehrer (1930-31) based on the studies on lymph nodes and spleen stated that the swine fever virus primarily affected the walls of the capillaries and smaller arteries and observed that lesions in the organs were due to the blood vessel changes. Seifried et al (1932a) studied 30 cases of acute hog cholera and found earliest and pronounced lesions in capillaries and smaller arteries. Large arteries and veins were less frequently affected. These lesions consisted of swelling and proliferation of endothelial cells together with retrogressive changes. They also observed hyalinization of blood vessels wall and perivascular necrotic areas. The most pronounced lesions in the blood vessels were seen in the lymph node, spleen and kidneys while the central nervous system, liver, intestinal tract, skin and other organs showed less pronounced lesions.

Present observations:

The vascular system showed lesions in almost all the cases right from the 7th day onwards. The chief lesions were observed in the walls of the capillaries and smaller arteries in the spleen, kidneys, brain, lymph glands and the intestine. The lesions comprised chiefly of the swelling and proliferation of the endothelial cells and degenerative changes like that of hydropic degeneration. The endothelial cells of the damaged vessels showed poor staining (figs.36,44,61,62 & 63). Several

blood vessels in the pancreas, spleen, lymph glands and brain showed hyalinization of their walls (figs.62 & 72). Haemorrhages round such affected vessels were observed in all the organs referred to as well as perivascular degenerative changes leading to necrosis were also observed.

Heart:

Seifried et al (1932b) found in a number of cases of swine fever the usual blood vessel lesions in the heart. Dunne et al (1952a) observed coagulation necrosis in the myocardial fibres particularly in those of the auricles. Palliola (1963) found in 10 pigs with swine fever slight degenerative changes in the myocardial fibres, proliferative and regressive changes in the small blood vessels and slight perivascular cellular accumulations with interstitial oedema.

Present observations:

In the present observations, only slight haemorrhagic and degenerative lesions were seen in few cases except for ons which died after a full course of the disease with the American strain of swine fever virus. Small and large areas of haemorrhages in between the cardiac muscle fibres were seen (fig.64). The cardiac cells also showed cloudy swelling and small areas of necrosis here and there.

Brain and Spinal cord:

Brunschwiler (1925) first described vascular changes and perivascular infiltration with endothelial swelling as the

principal microscopical lesion in the brain. Later it was confirmed by Roehrer (1930) who found these lesions in 75% of the cases examined. The high incidence of non-purulent encephalitis in cases of swine fever as a diagnostic lesions was recognised independently and almost simultaneously by Dide (1930) and Seifried (1931). Since that time their observations have been confirmed by a number of workers (Bendinger, 1934; Salvalaglio,1937; Helmboldt,& Jungherr,1950 &1952; Sassenhoff,1952; Ressang,1953; Euglert,1953; Ohbayashi,1953; Pallaske et al,1955; Beer,1957; Done,1957; Okaniwa,1960;Dunne, 1961; Keast et al,1962 and Dunne,1963). Only a few workers have doubted the specificity of the lesions described by the above quoted workers (Kobe & Schmidt,1934; Jones & Doyle,1953).

The typical lesions of swine fever as reported originally by Seifried (1931), Helmboldt et al(1950) and Done (1957) consisted of a pan-encephalitis with usually a variable and mild myelitis. The prominent lesions were a varying amount of vascular and perivascular changes of the central nervous system. Seifried (1931) described a mononuclear infiltration of the perivascular spaces consisting chiefly of small lymphocytes, mononuclear elements, a few plasma cells and occasionally eosinophils in the brain and spinal cord. The glia showed a proliferative change surrounding the infiltrate vessels or formed small nodules or more diffuse foci. Both microglia and macroglia were involved in the process. Satellitism and in few cases true neuronophagia have been reported. The nerve cells in almost all parts of the central nervous system were prone to

degeneration. Other changes observed in vessels were congestion, swelling and degeneration of endothelial cells and perivascular oedema. Microscopic haemorrhages in the close proximity of a blood vessels or away from it were also observed. Dunne (1963) reported partial occlusion of vessels.

Helmboldt et al (1950) observed the primary lesion in the mesoderm which consisted of vascular and perivascular cuffs, microgliosis, lepto meningeal infiltrates, capillary haemorrhage and hyalinization of vessel wall.

Urman et al (1962) described intramural and perivascular round cell proliferation and infiltration in the brain,meninges and choroid plexsus.

The lesions in brain started as early as six days after infection (Seifried,1931), 3 days after infection (Urman et al, 1962) while Dunne et al (1952a) observed the severity of the lesions from 10-14th day post-infection.

Lesions in the central nervous system were observed in 80% (Potel,1956a), 90% (Done,1957) and 95% (Dunne,1961) of the cases examined by them.

Potel (1956b) found encephalitis a common feature in swine fever without the virus necessarily becoming neurotropic.

Inclusion bodies were not observed (Seifried,1931 and Huguenin,1929), while Urman et al(1962) reported acidophilic intranuclear inclusion bodies in the brain.

Kuppuswamy (1962),on histopathological examination, noticed oedematous distention of Virchow-Robin space and aggregation of a few leucocytes with areas of haemorrhage around

blood vessels in the brain and infiltration with mononuclear cells around the blood vessels in the spinal cord of piglets on the basis of which as well as lesions seen in other organs namely spleen, liver,lymph glands,etc.,the cases were diagnosed as swine fever which was later confirmed by Weybridge Laboratory London.

Present observations:

In the present observations, microscopic lesions were found in different portions of the brain and spinal cord. The changes were seen to be more pronounced in advanced cases than in the earlier ones.

The animals destroyed at 7,9 and 11 days intervals in both the groups showed very slight or no perivascular cuffing in the cerebrum, cerebellum,medulla or in the spinal cord. The main lesions in these animals comprised of severe congestion of the leptomeningeal vessels and the vessels in the brain substance and spinal cord. Hyalinization of few blood vessels was observed with a thin layer of red blood cells surrounding them. Small haemorrhages were also noticed in the brain tissue unassociated with blood vessels. In few cases oedema of the Virchow-Robin spaces was found.

The animals which were sacrificed on the 13th day and those left to undergo the full course of the disease showed very striking mononuclear infiltration of the Virchow-Robin space in the brain (figs.65,66,67 & 68). The infiltrating cells simulated the small lymphocytes,mononuclear elements and

few plasma cells. Eosinophils and neutrophils were not observed among the infiltrating cells. Such changes were found in the vessels of the cerebrum, cerebellum, medulla and in the spinal cord. Severe perivascular haemorrhages and oedema were also observed in the leptomeninges (fig.69). In one case which died due to the local strain of virus after the full course of the disease showed intramural as well as perivascular infiltration with mononuclear cells already referred to (figs.67 & 68).

The nerve cells showed mild to severe degenerative changes with the advance of the disease. The nerve cells of the cerebrum, cerebellum, medulla and spinal cord all showed degenerative changes differing in intensity with the progress of the disease. The changes observed in the nerve cells were hyperchromatism of some of the nuclei, ~~shrix~~ swelling, absence of the nucleoli, denticulation of the cell membrane and consequently presence of only cell shadows(fig.70). A decrease in the number of Purkinje cells were also seen to occur in the cerebellum of the affected animals.

Out of the two animals,one killed at 13 days interval (American strain group) and the other which died after a full course of the disease with the local strain showed few small foci of glial proliferation along with a few mononuclear cells (fig.71). Such foci were mostly seen in the vicinity of blood vessels.

Thus a diagnostic non-purulent encephalitis was found in cases suffering from swine fever.

The observations are in close confirmity with that of Seifried (1931), Done (1957), Dunne (1961), Keast et al(1962), Urman et al (1962), Dunne (1963) and others.

Other organs (Pancreas,Adrenals & Tongue):

Potel (1956) based his observations upon 102 pigs killed between 4 hours and 14 days after experimental infection with swine fever, found pronounced vascular lesions,consisting of proliferation of the undifferentiated cells of the vessel walls and simultaneous necrobiotic degeneration of the walls. These changes were not regarded diagnostic. Dekock et al(1940) from South Africa observed necrosis of the parenchyma in certain areas accompanied by thrombosis and general karyorrhexis and disintegration of gland cells in pancreas of pigs with the African swine fever. They also found irregular haemorrhages in the gland.

Matthias (1954) described the histopathological changes in adrenal glands from 53 pigs artificially infected with swine fever and found that the cortex exhibited changes typical of stress syndrome. He found 73% of the cases with cellular infiltration of the medulla, characterised by a high proportion of eosinophils and absence of chromaffin substances. He interpreted these findings as evidence of circulatory disturbance. Ivanov et al (1956) found hyperaemia of the border zone between the cortex and medulla in 57% out of the 150 cases killed 4-6 days post-infection. This change was found by them to be rare in other diseases. Rubaj (1959) found hypertrophy of the cortex. Marked perivascular cellular infiltrations mainly of lymphocytes and

histiocytes in the medulla of 60% of the field cases and 85% of experimental pigs infected with the swine fever virus were also observed by him.

Present observations:

The chief lesions in the pancreas were found to occur in the blood vessels. Degenerative changes of the intima and hyalinization of blood vessels (fig.72) were seen resulting in congestion and haemorrhages. Disruption of acini and disintegration of gland cells were seen. Areas of degeneration showing chromatin less mass of cell remanants were also evident (fig.73).

Adrenal glands in a few cases were examined histopathologically, revealed congestion and slight haemorrhage in cortex and medulla. Slight necrosis of the cells of the medulla were seen in two cases.

Extensive haemorrhage was observed in the connective tissue core and between the striated muscle fibres of the tongue (fig.74). The extravassation of blood resulted in necrosis of the muscle fibres as well as in replacement of the connective tissue core by red blood cells. A few foci of small leucocytic infiltrations were also observed. These lesions were supposed to be rare (Palliola,1963) but during this study 3 out of the 10 cases showed lesions (2 in the American strain and 1 in the local strain group) generally at the terminal stages of the disease.

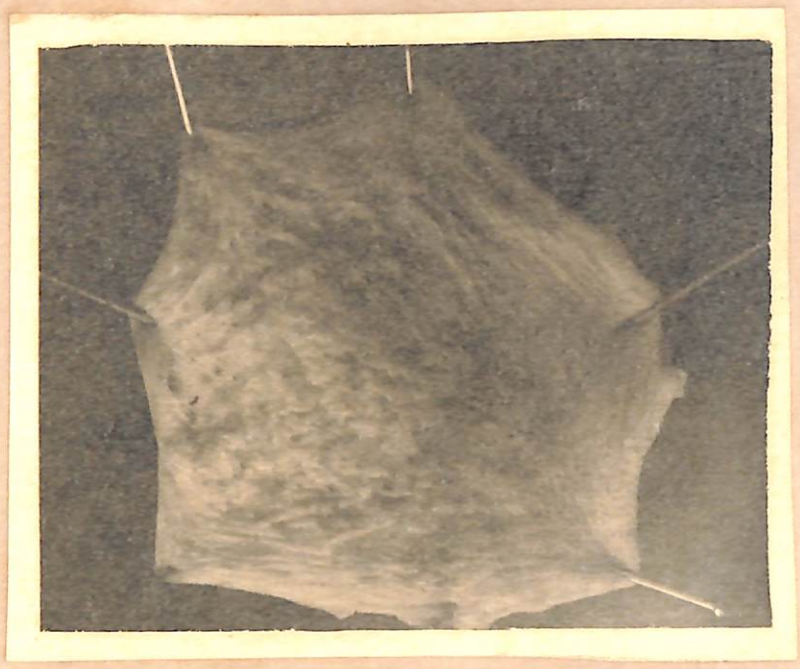
Inclusion bodies:

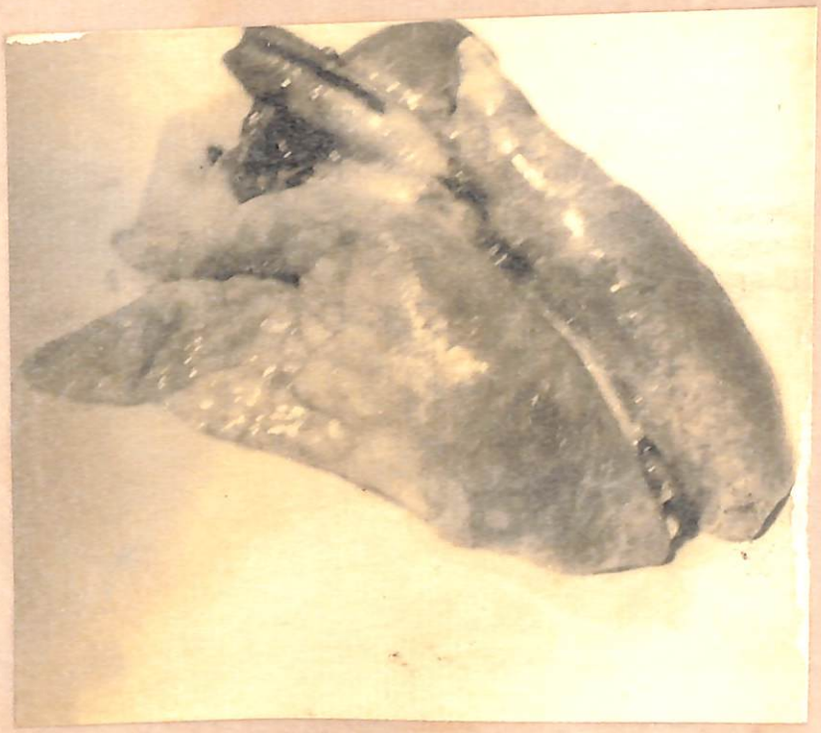
Uhlenhuth et al (1929) found bodies akin to inclusions in the corneal epithelium of pigs suffering from swine fever. Huguenin (1929) could not find any inclusions in a variety of tissues. Seifried (1931) confirmed this finding. Bogdan(1957-58) found intranuclear inclusions in the epithelial and glandular cells of the gall bladder in a large number of animals. Krieg (1961-62) found the inclusions in the liver cells in only a small proportion of the cases which diminished their value as a diagnostic aid. He could also demonstrate similar inclusions in a pig treated with corticotropin and in two which were kept on intensive diet and linked up their association with intensified metabolism especially of proteins. Urman et al (1962) demonstrated acidophilic intranuclear inclusion bodies in the reticulo-endothelial system through out the body and were generally evident from 7-10th day post-infection.

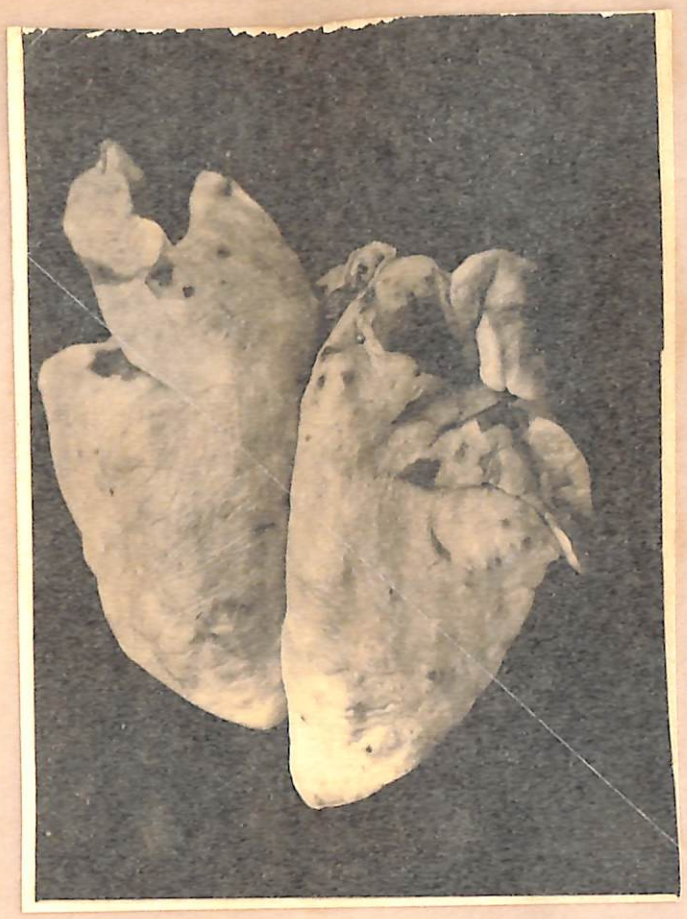
Present observations:

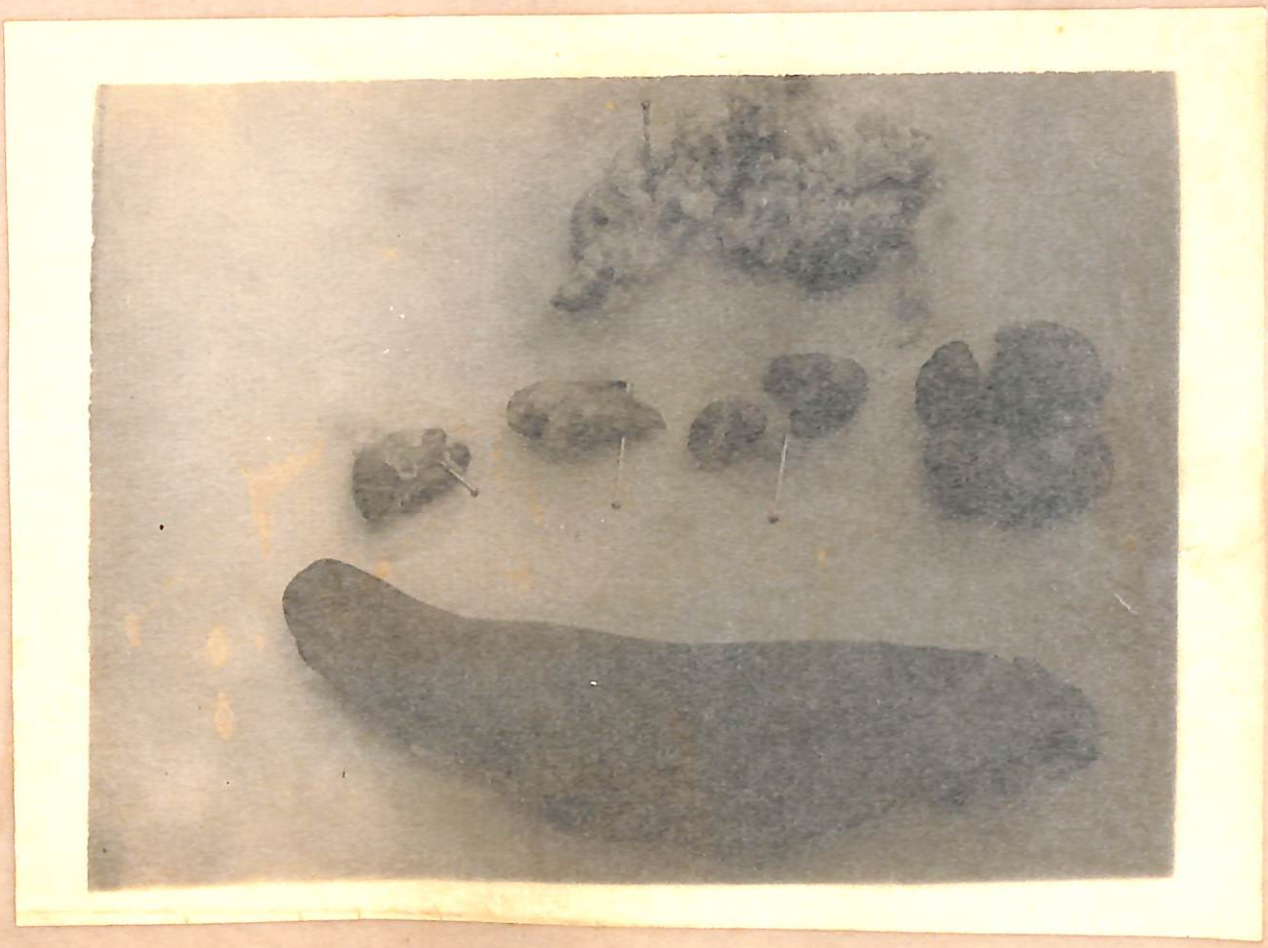
In the present studies, a careful and thorough examination was made during the histopathological examination of sections of lymph glands,spleen,brain,kidneys,heart etc.,for the presence of cytoplasmic and intranuclear inclusion bodies,if any. Even special staining technique was adopted as mentioned under materials and methods. Only occasionally eosinophilic bodies were seen in the nuclei of the reticulum cells of the lymph glands suggestive of intranuclear inclusion bodies. But as the presence of these bodies was not constant and their appearance

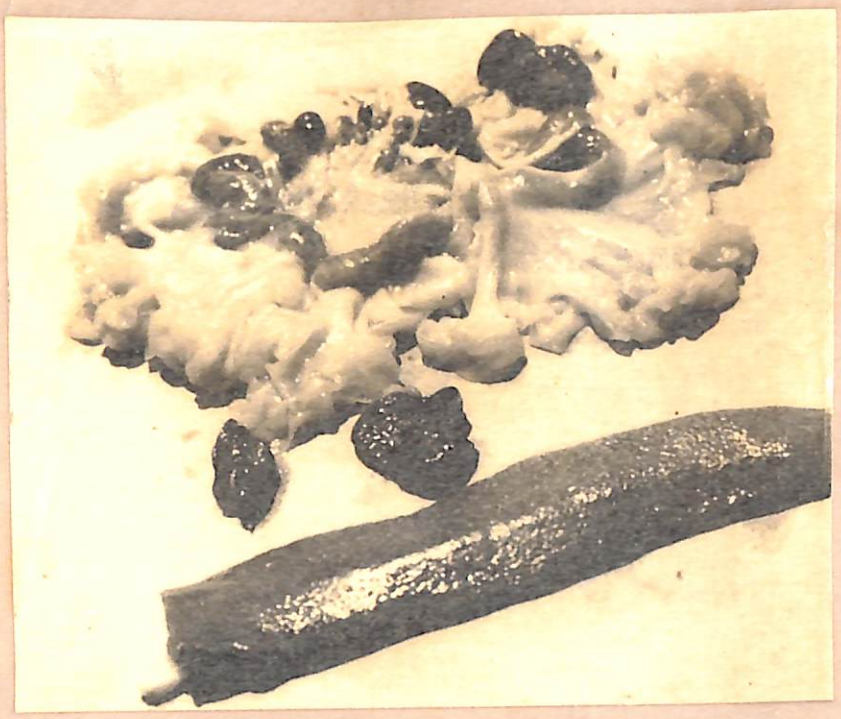
was not always distinct it is considered that no definite conclusion could be made regarding the validity as to whether they are really intranuclear inclusion bodies or products of faulty metabolism as suspected by Kreig (1961 & 1962). Further investigations are perhaps warranted in this matter.

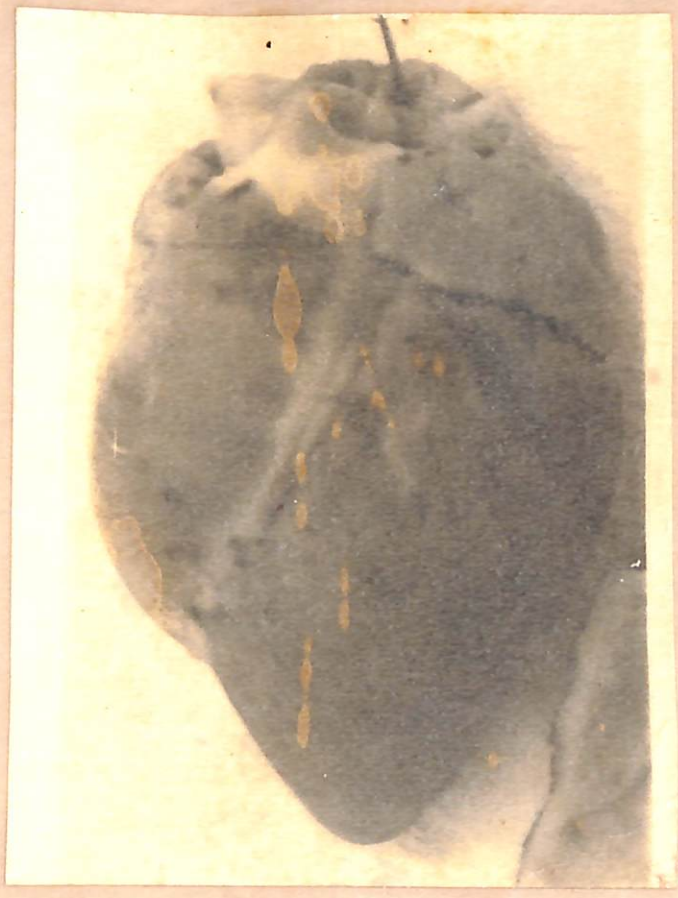


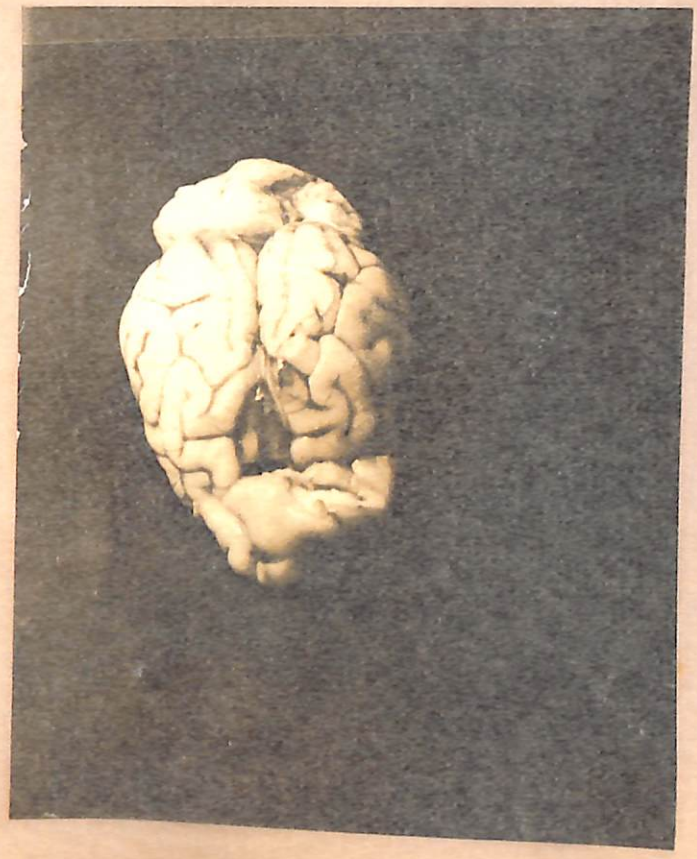


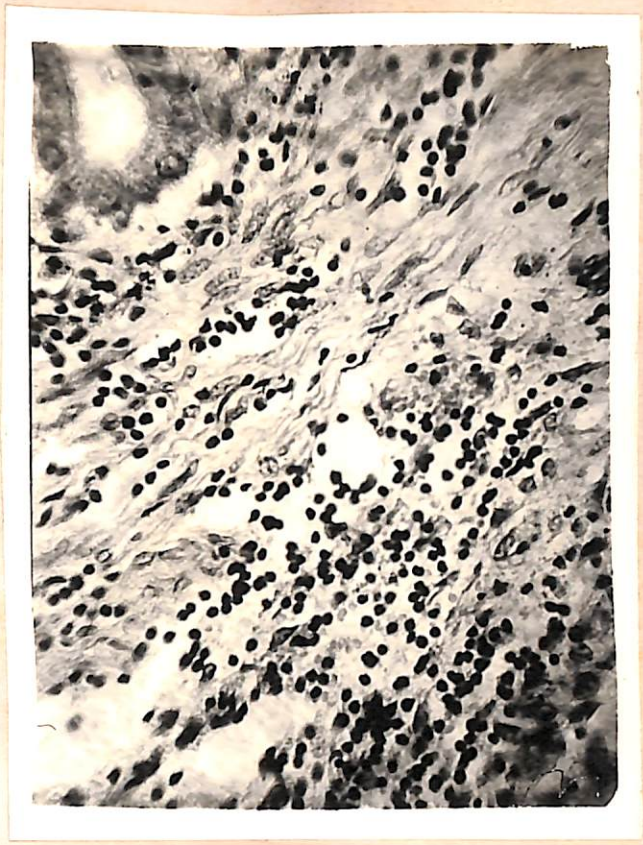


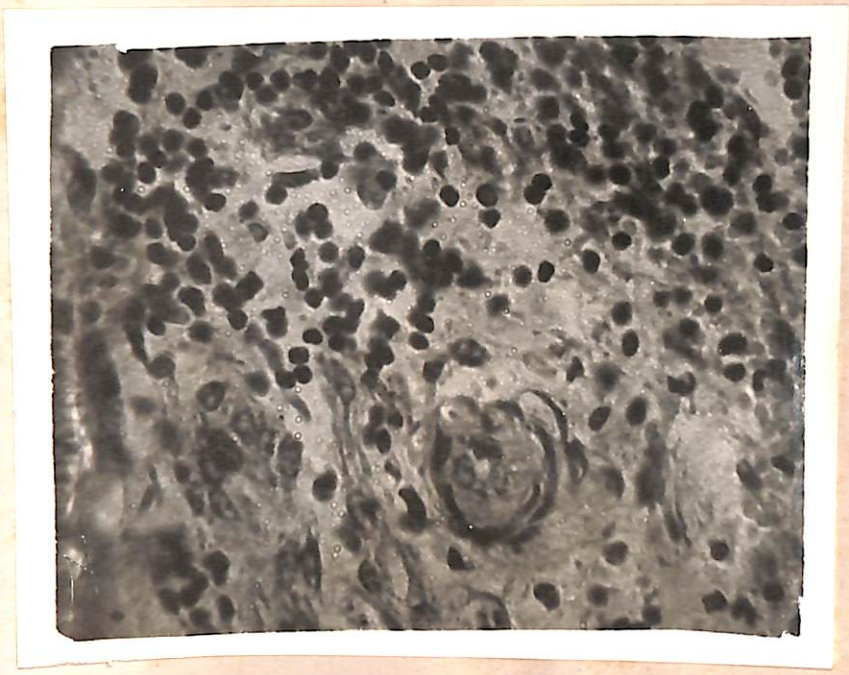


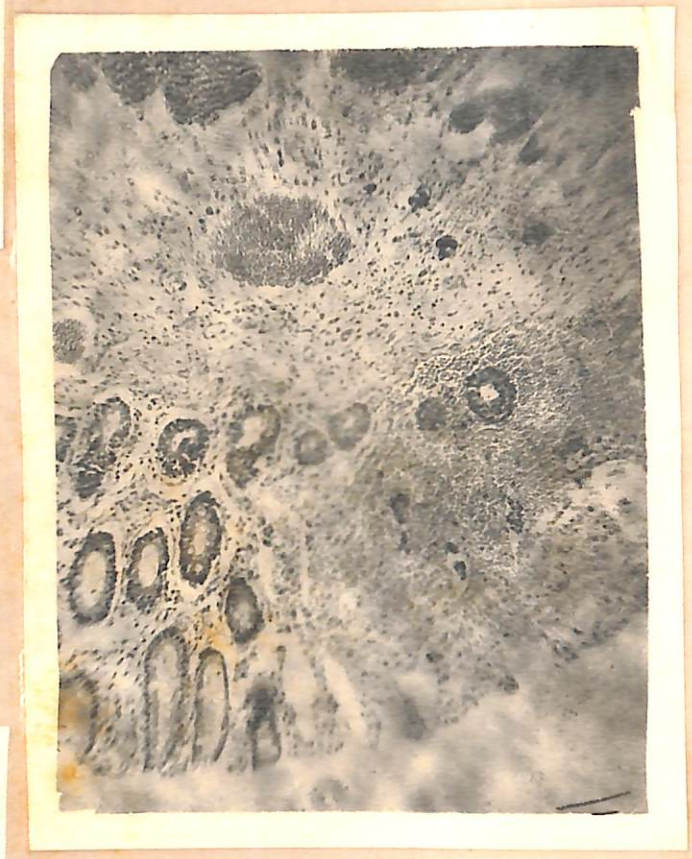


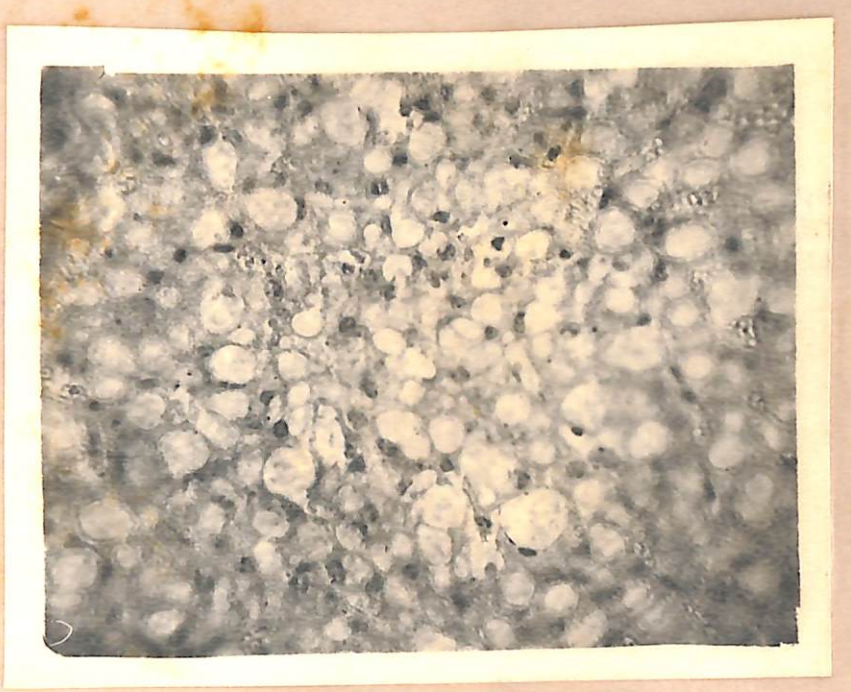


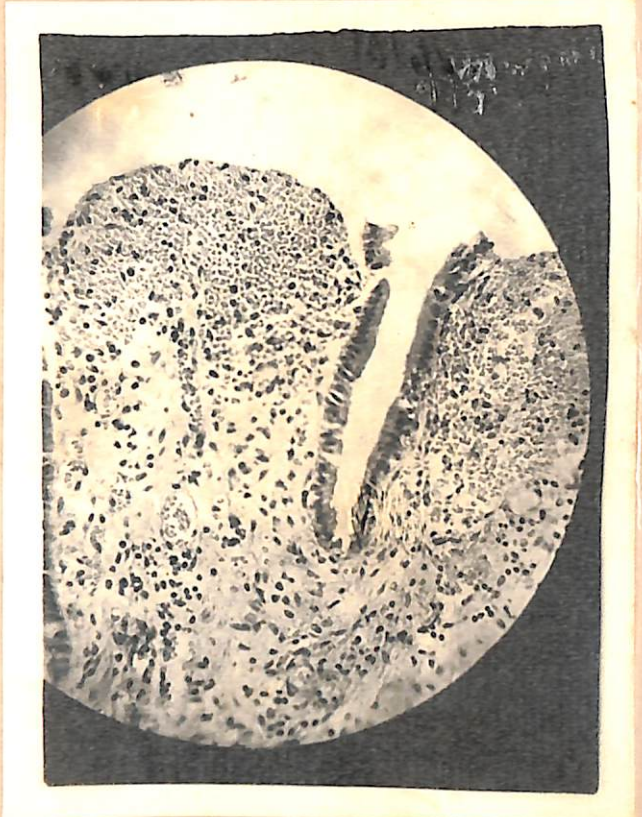


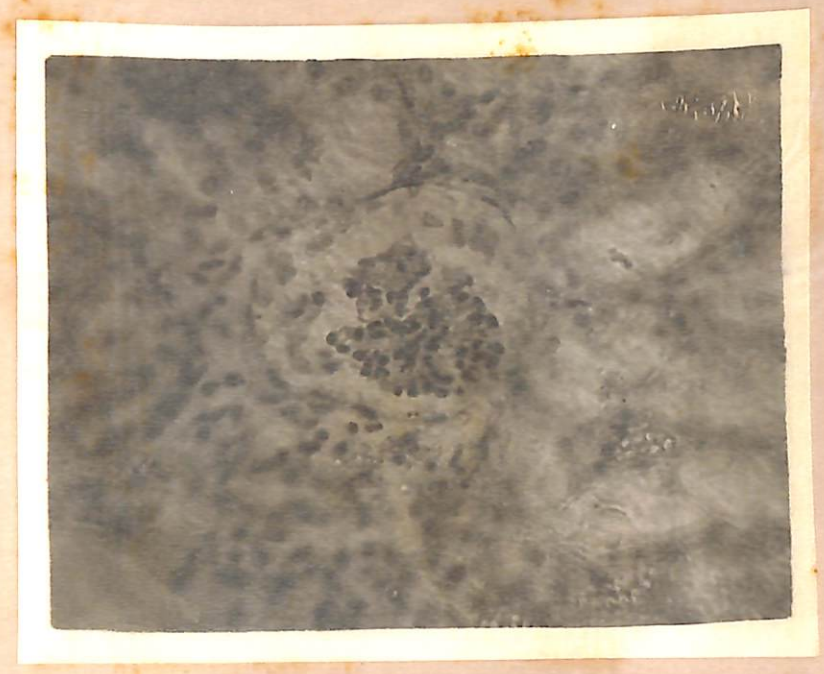


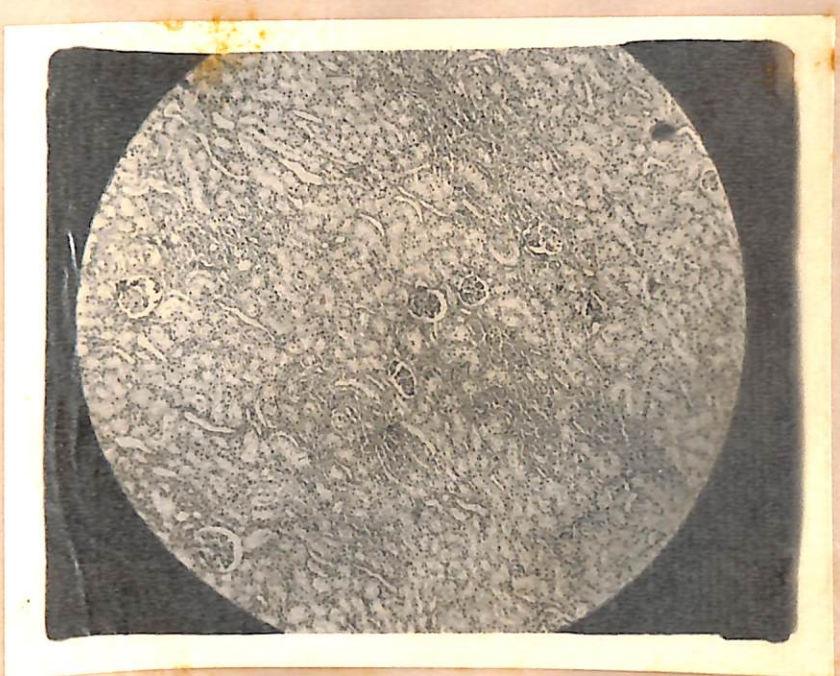


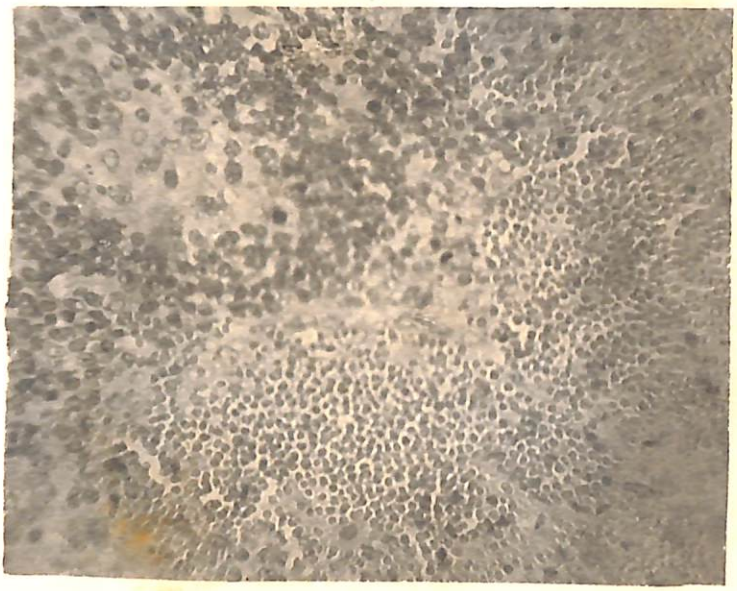


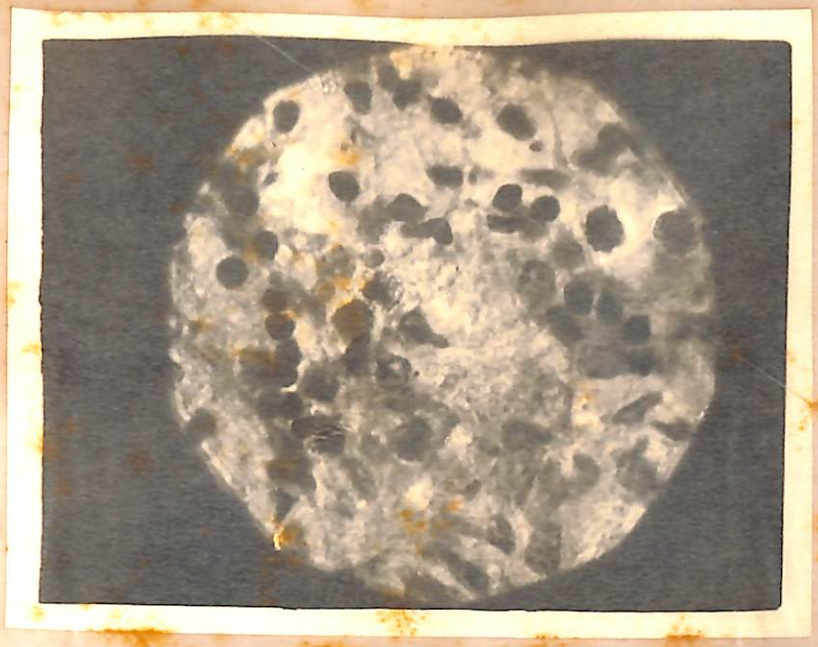


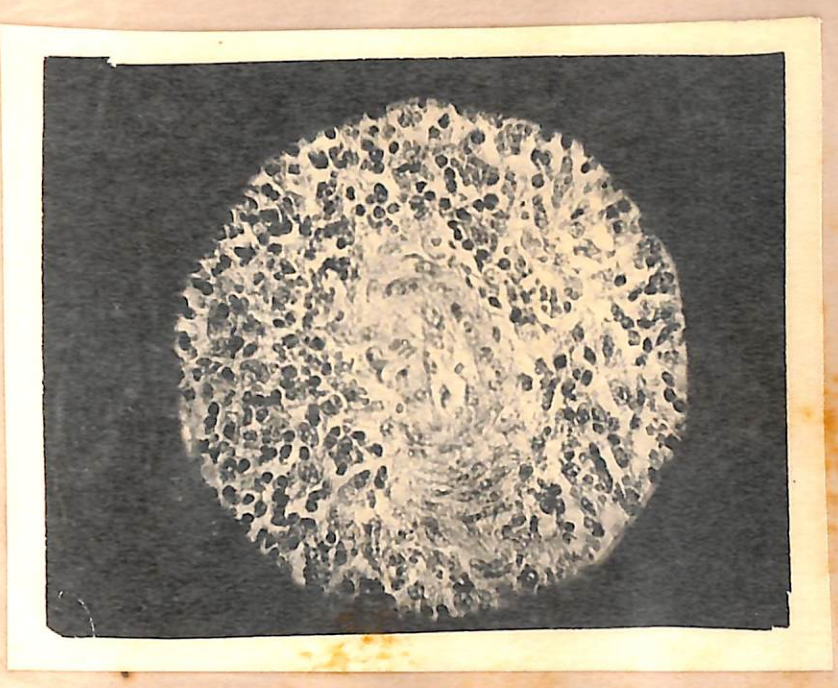


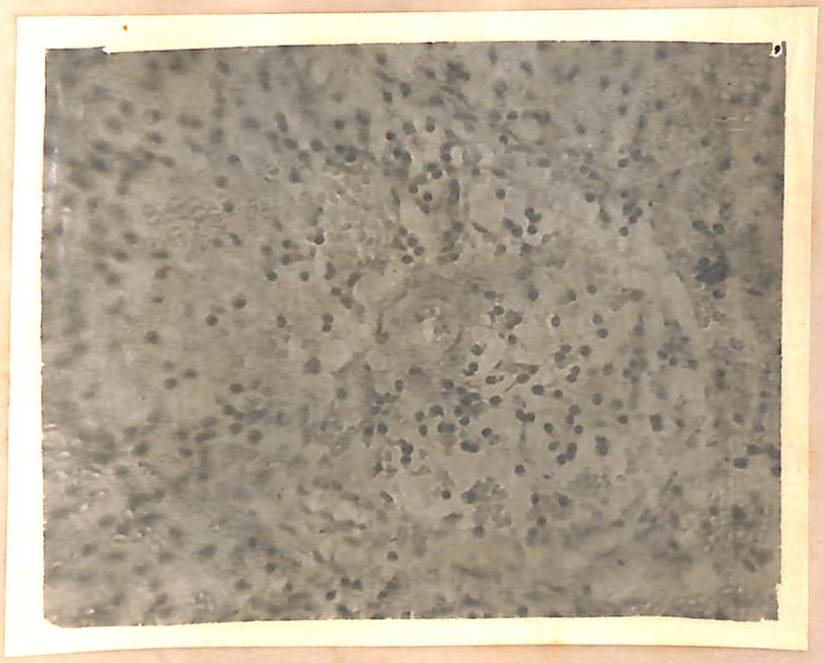


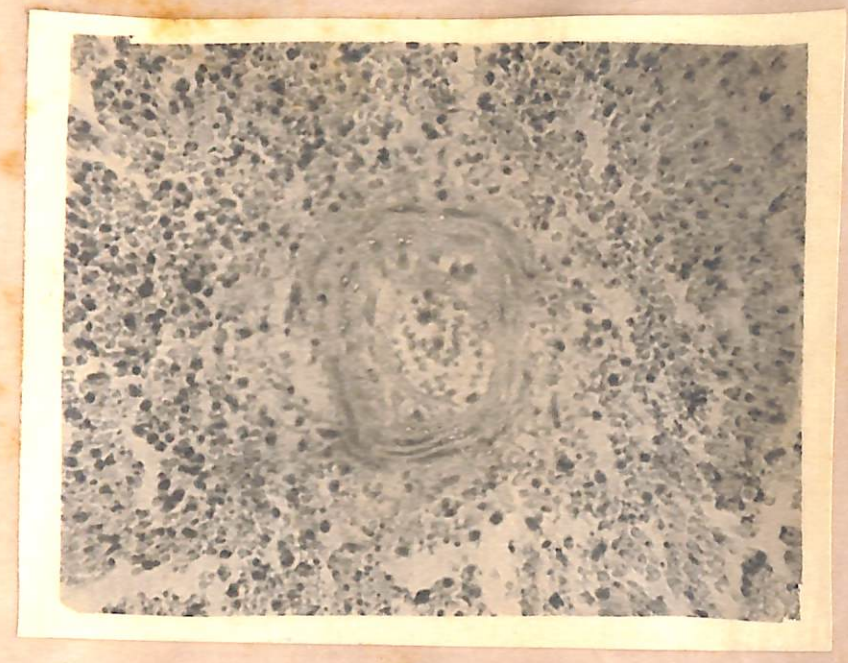


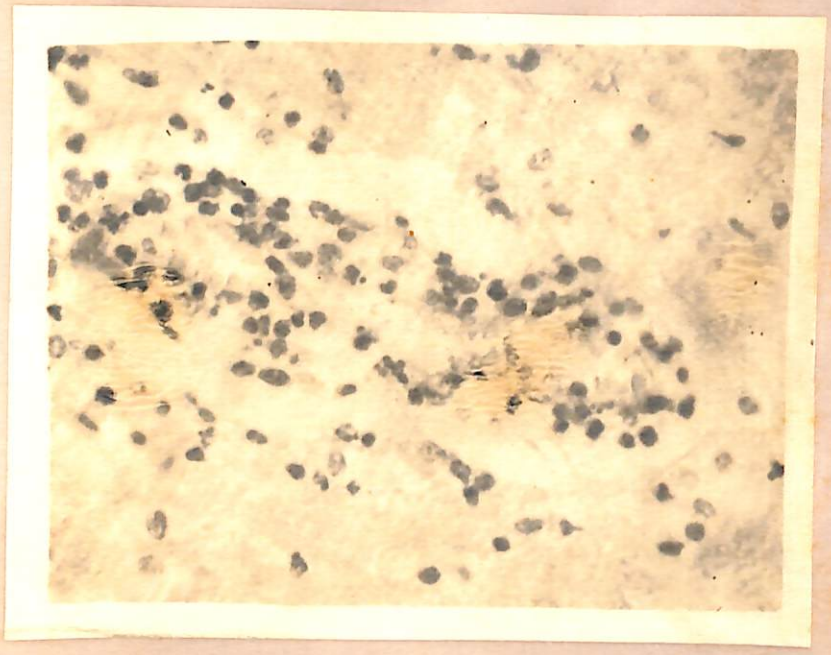


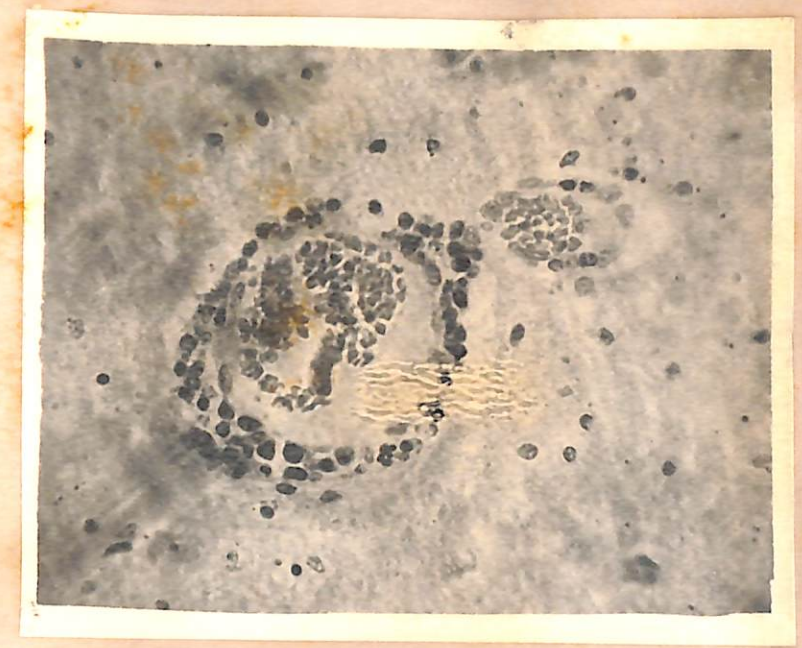


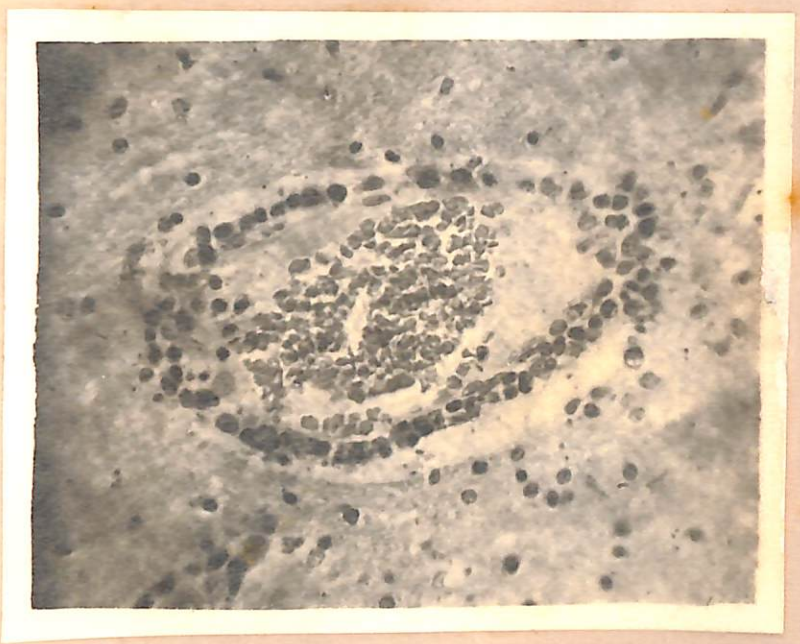


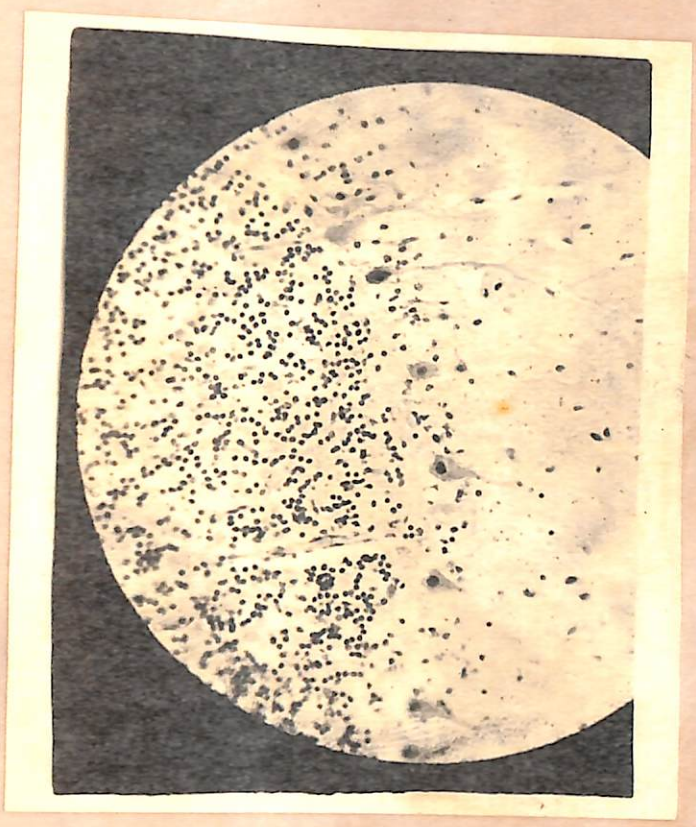


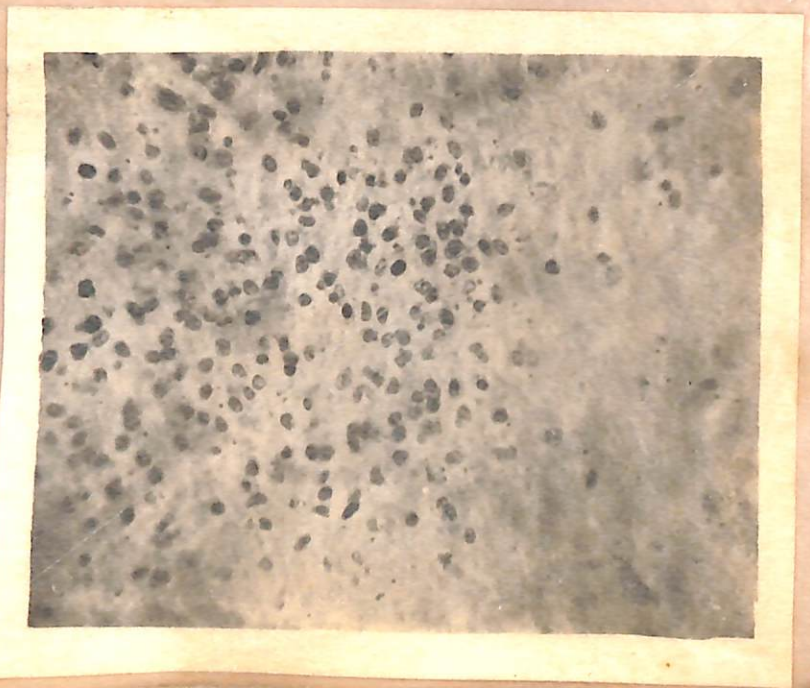


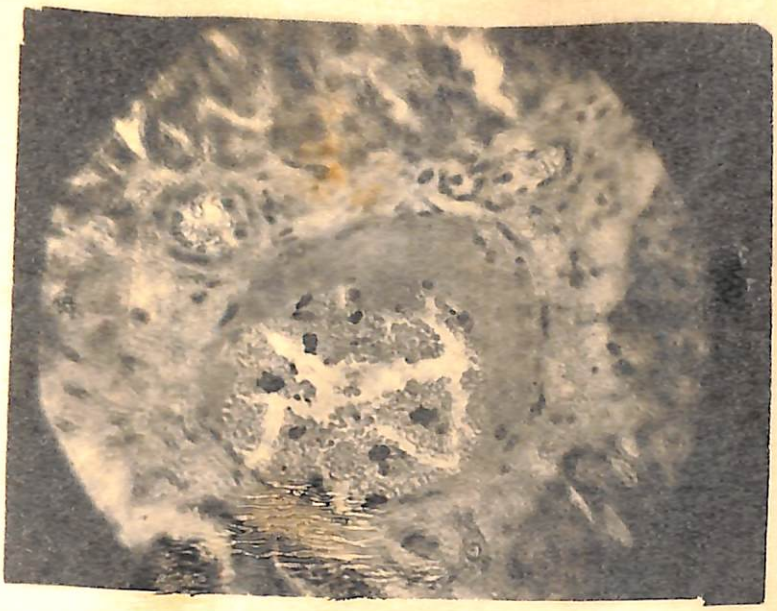


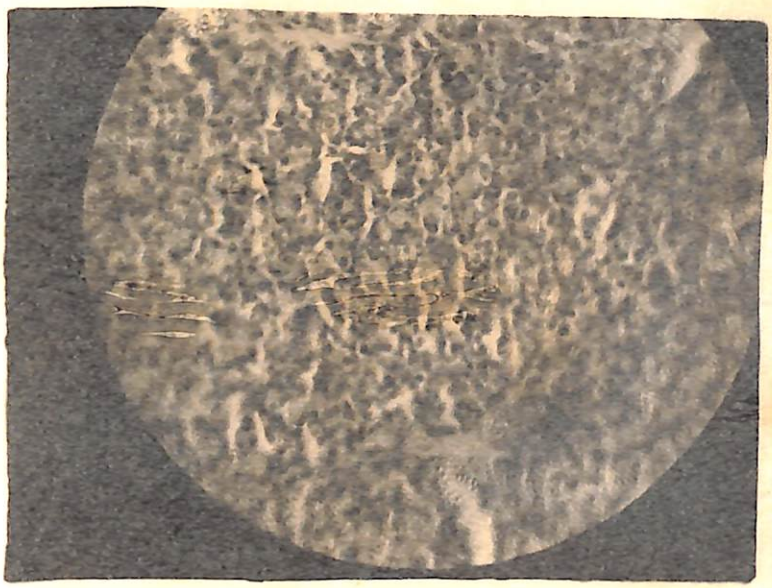












REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON HAEMATOLOGY.

REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON HAEMATOLOGY

The haematological study in normal pigs as well as those suffering from swine fever have not been studied by workers in India. Striking changes in the blood picture have been found to occur in a large percentage of swines affected with this disease.

The most striking change is found in the leucocyte count of pigs suffering from hog cholera. King and Wilson (1910) found leucopaenia of moderate degree in pigs suffering from swine fever. Lewis et al (1914) confirmed the findings of King et al (1910). Dinwiddie (1914) found a distinct leucopaenia in pigs from experiments upon 14 animals infected with swine fever. Regner (1923) tried to differentiate swine fever with swine plague and swine erysipelas and reported a rapidly occurring leucopaenia in acute hog cholera cases. Lewis and Shope (1929)&(1929a) found extreme leucopaenia occurring even from 48 hours post-infection in some of the cases. Leucopaenia was also seen to precede clinical symptoms and febrile reactions in cases of swine fever. Leucopaenia was maintained through out the course of the disease with irregular fluctuation, but the counts never returned to the normal level. Ishii et al (1936), in 8 animals with swine fever, found a decrease in W.B.C.count shortly after the injection of the virus which diminished gradually with the progress of the disease. Kernkamp (1939a) based his observations upon a large number of

animals (55 artificially infected, 67 field cases of swine fever, 27 with pneumonia and 49 with inflammation of one or more part of the gastro-intestinal tract) and found leucopaenia occurring from 24 - 48 hours onwards post-infection and even before a febrile reaction in the artificially infected group with swine fever. Leucocytosis was observed in cases suffering from pneumonia and gastro-enteritis.

Dunne (1961 & 1962) conducted leucocyte counts on 1,671 normal pigs between 6-12 weeks of age and fixed the normal value from 10,000 to 40,000 with the average at 21,000. In pigs 6 weeks of age or older any count lower than 10,000 was supposed to be indicative of definite leucopaenia and from 10,000 to 13,000 (5.3% of the normal counts) to be suspicious of leucopaenia. Based on this assumption he carried out leucocyte counts over 2,500 cases of swine fever at different stages of the disease and found more than 53% having counts below 10,000 which was considered by him positive for hog cholera in older pigs than 5 weeks. A leucocyte count of 10,000 to 13,000 was held suspicious of hog cholera. Cahill (1929), Shu (1929-30), Thorp *et al* (1930) , Oglesby *et al* (1932), Sarnowiec (1933),Ishii *et al* (1936),Kernkamp (1939a), Ishii *et al* (1953), Schellner *et al* (1956), Sanchez Franco (1957), Pinkiewiez (1960) and Dunne (1961 & 1963) described leucopaenia as the most striking and constantly occurring change in swine fever with a definite diagnostic value while Cole(1932) supposed it as usual but not a constant feature of hog cholera.

Kernkamp (1939a) concluded from his observations that leucopaenia in atleast 3 animals in a herd can be regarded as positive for hog cholera while Dunne (1963) suggested preferably to count the W.B.C. in six animals rather than three for the diagnosis.

Murthy et al (1962) observed in six cases of experimental swine fever, an increase in the leucocyte count in the first three days followed by leucopaenia.

Differential counts:

Lewis and Shope (1929) found the greatest injury to the polymorphonuclear leucocytes which at times were found to be completely absent. Leucopaenia included both polymorphonuclear and lymphocytic series of cells. Shu (1929-30) observed neutrophilia with a shift to young forms while Ishii et al(1936) considered neutropaenia to be more characteristic of the disease. The eosinophils showed a decrease with the progress of the disease and even became rare or completely absent in animals with fever. Relative increase of the lymphocytes was recorded by them. The relative number of band forms showed an increase but the relative and absolute number of segmented forms decreased after the appearence of fever. Kernkamp (1939a) found increase in the neutrophils in the artificially infected as well as in field cases of swine fever whereas apparent increase in neutrophils in pneumonia and inflammatory conditions of the gastro-intestinal tract. Sanchez Franco (1957) found lymphopaenia and Pinkiewiez (1960) observed increase in the

number of polymorphonuclear cells with shift to the left and showing degenerative changes. He also observed decrease of eosinophils and monocytes till they disappeared with the progress of the disease. Basophils showed an increase in the beginning which later on disappeared.

Appearence of nucliated R.B.C. (Lewis and Shope,1929a) and drop in reticulocyte count (Ishii et al,1953) were also observed during the infection.

King and Wilson (1910) and Lewis and Shope (1929,1929a) reported a moderate progressive loss of R.B.C. Kernkamp(1939a) did not think it of any diagnostic value.

King and Wilson (1910), Lewis et al (1914) and Lewis and Shope (1929,1929a) found progressive loss of haemoglobin while moderate anaemia was observed by Dinwiddie (1914), Shu (1929-30) and Oglesby (1932). Lewis and Shope (1929) and Kernkamp (1939) stated this change without having any diagnostic value.

Kernkamp (1939a) found marked increase in the sedimentation rate in swine fever than the rate observed in normal swine, nevertheless it has not been applied on a substantial number of animals affected with diseases other than swine fever to justify its specificity for swine fever. Sanchez Franco (1957) found increased sedimentation rate based on observations upon 320 experimentally infected pigs aged 1-2 years with swine fever.

Present observations:

In the present observations mean blood picture of pre &

post-infection periods showed a distinct variation from the normal.

The leucocytes showed the most prominent changes and 24 hours post-infection a slight leucocytosis was observed in both the groups. (Normal 16,483 & 18,336 group infected with the American and local strain 17,540 and 19,120 respectively). The leucopaenia started gradually 24 hours onwards and was quite marked in all the animals at 120 hours (4,750 & 5,480 in American and local strain groups respectively). Though leucopaenia started even before any clinical symptoms and temperature reaction but usually it was marked only when the temperature was fully established at 120 hours. The leucopaenia persisted throughout the course of the disease with slight fluctuations but never reached the normal level(Tables I & IA, and Table II & IIA). The mean values of W.B.C. counts at 312 hours was 2,250 & 4,425 in the American and local strain groups respectively.

The neutrophils in both the groups showed first a slight increase usually upto the 120 - 144 hours with a shift to the left (Normal mean values Neutrophils 30.8 & 28 and group infected with the American and local strains 40 & 35.2 respectively). The neutrophils during this period and after showed signs of degeneration and several vacuoles were seen in their cytoplasm. At 312 hours the neutrophils showed a distinct decrease and the mean values stood at 24.5 & 17 in the American and local strain groups respectively. On the other hand the lymphocytes first showed a slight decrease but later on increased and at 312 hours the mean values were (Normal 63.3 & 66 infected

72 & 78.5 in the American and local strain groups respectively). With the progress of the disease and establishment of temperature the eosinophils disappeared from the 216 hours post-infection in both the groups.

Decrease in R.B.C. and Haemoglobin was also observed vide Table I,IA,II & IIA. The E.S.R. showed a marked fall from normal and at 312 hours the readings were 25.5 & 25.3 in normal and 70 & 58.5 mm. in the American and local strain groups respectively. The fall was noticed to be gradual and started 48 hours post-infection.

The pigs which were allowed to die after the full course of the disease showed marked variation from the normal. Leucocyte counts in the American and local strain groups were found to be 850 and 4,100 as against the normal mean value of 16,483 & 18,336 respectively. Leucocyte count as low as 700 was observed two days prior to death in the American strain group.

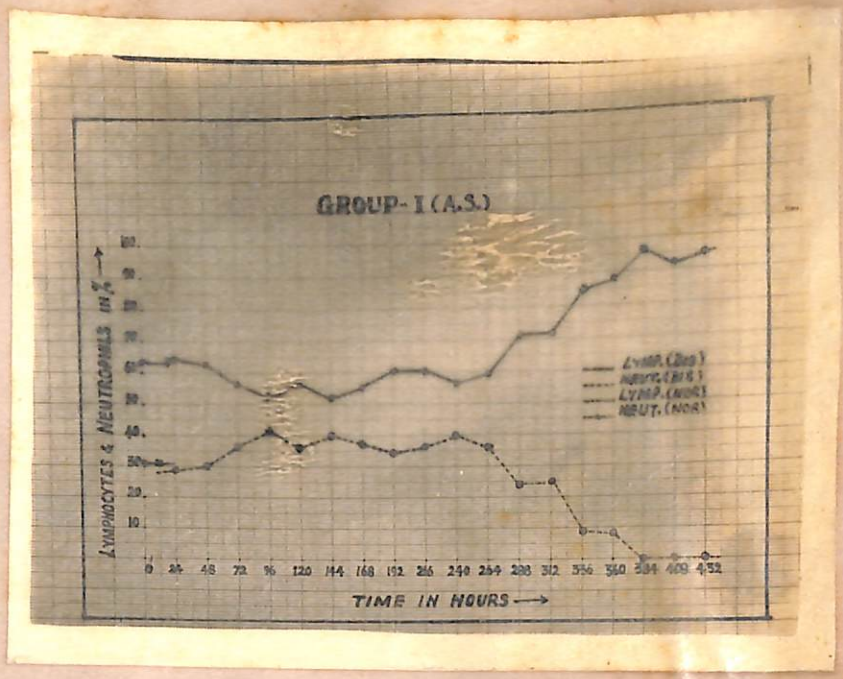
A marked neutropaenia was seen during the terminal stages of the disease (0 & 8 as compared to normal mean value of 30.8 & 28 in the American and local strain groups respectively). In the first group complete neutropaenia was observed from the 384 hours which persisted till death. The lymphocytes showed a remarkable increase (95 & 89 as against the normal mean values of 63.3 & 66 in the American and local strain groups respectively).

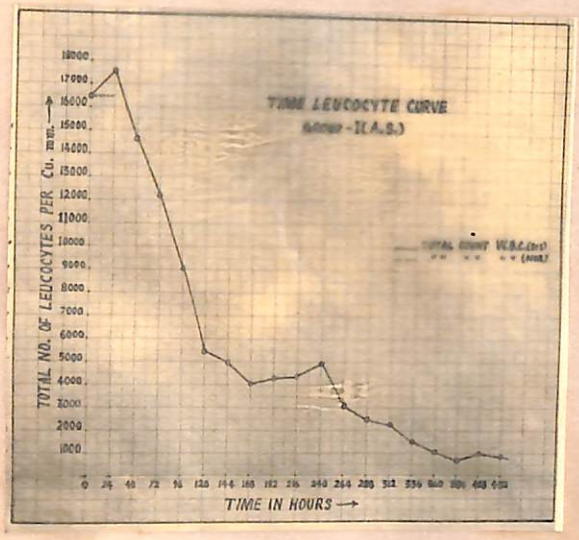
The R.B.C., Hb.,& P.C.V. showed marked decrease while the sedimentation rate showed a definite increase. The M.C.V.,

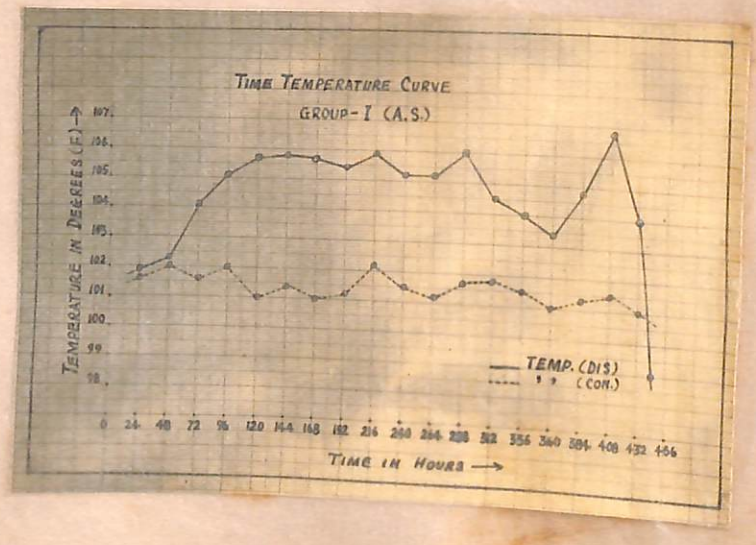
M.C.H. & M.C.H.C. showed variations during the different stages of the disease (Table I,IA,II & IIA).

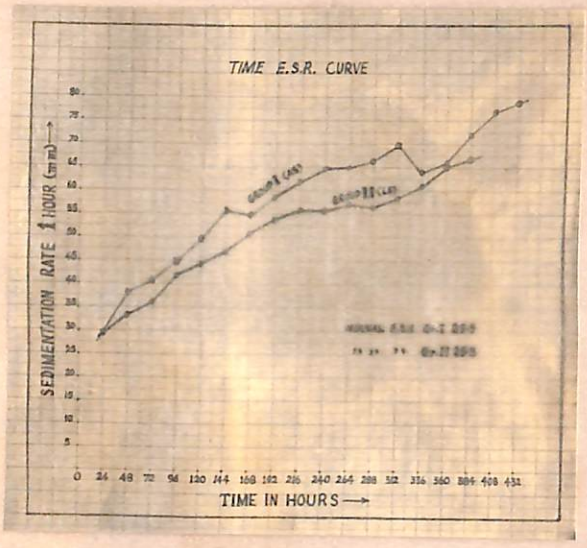
The findings on the leucocyte counts are in close confirmity with that of Dinwiddie (1914),Lewis and Shope (1929,1929a), Ishii et al (1936),Kernkamp (1939a), Dunne (1961 & 1963) and many others. The temporary leucocytosis upto 24 hours that was observed in these experiments does not appear to have been reported so far. Murthy et al (1962) observed for the first time leucocytosis in the pigs suffering from swine fever but the leucocytosis was seen to persist upto three days. The differential count agrees with the observations of Lewis and Shope (1929), and Ishii et al (1936) but not with those of Shu (1929-30) and Kernkamp (1939a).

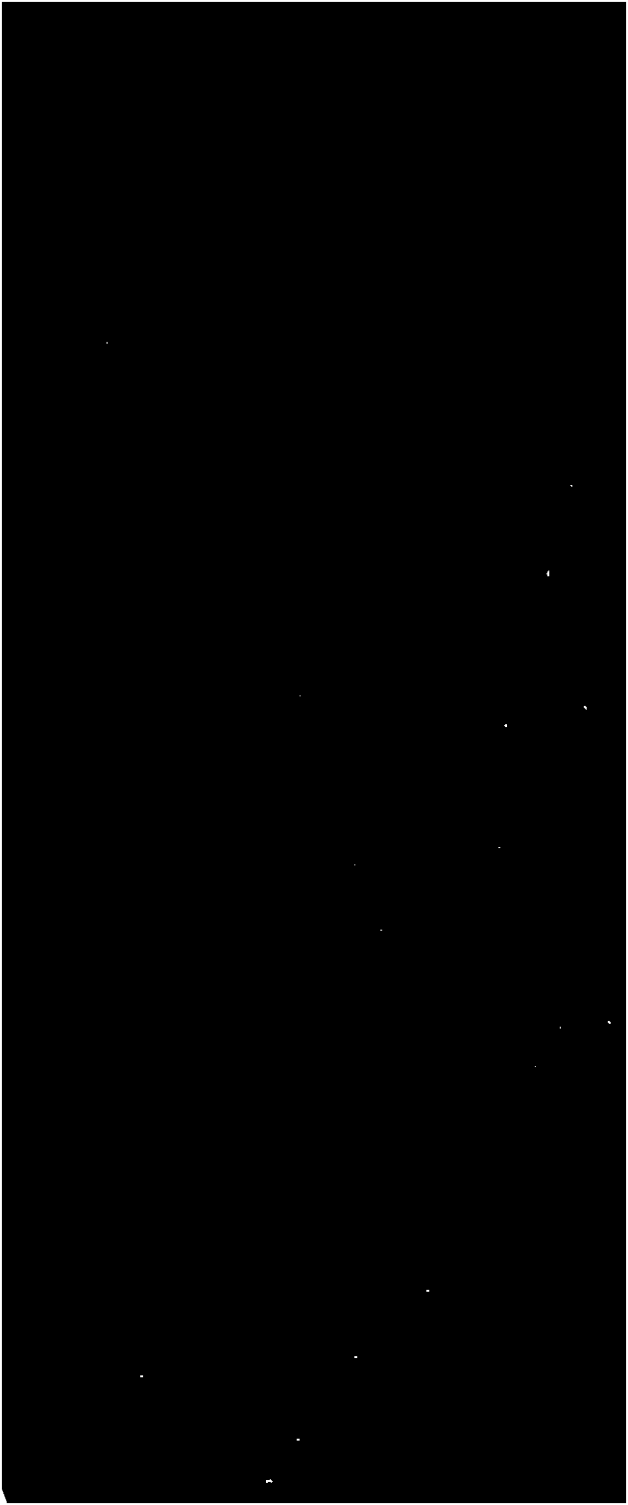
The variations in the total W.B.C. count, lymphocytes, neutrophils and E.S.R. in the pre and post infection periods are quite significant. The above haematological findings have been incorporated in Tables I,IA,II & IIA. A time temperature, time leucocyte, time neutrophil and lymphocyte per cent and time E.S.R. graphs have also been incorporated to see the trend of the changes.

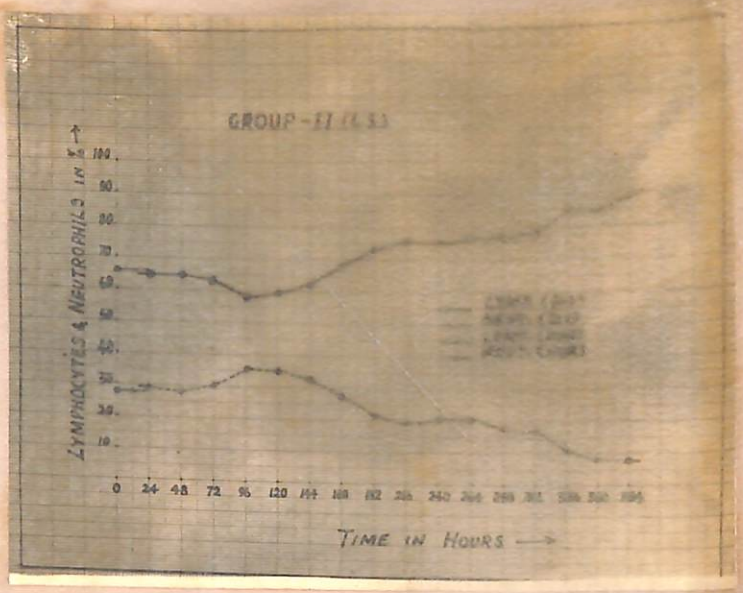

GROUP-I (A.S.)
LYMPHOCYTES & NEUTROPHILS IN %
TIME IN HOURS

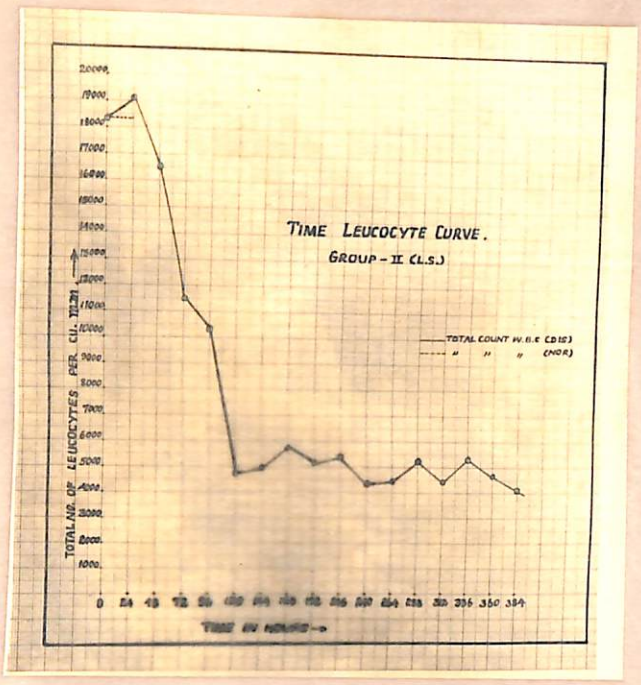

TIME LEUCOCYTE CURVE
GROUP-II (A.S.)
TOTAL NO. OF LEUCOCYTES PER Cu. mm.
TIME IN HOURS

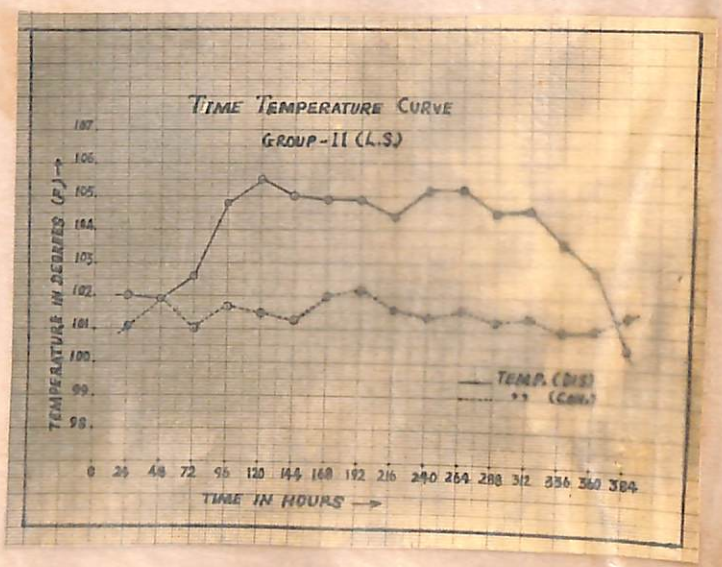

TIME TEMPERATURE CURVE
GROUP-I (A.S.)
TEMPERATURE IN DEGREES (F)
TEMP. (DIS)
TIME IN HOURS


TIME E.S.R. CURVE
SEDIMENTATION RATE 1 HOUR (in mm) ⟶
0 24 48 72 96 120 144 168 192 216 240 264 288 312 336 360 384 408 432
TIME IN HOURS ⟶




GROUP - II (L.S.)
LYMPHOCYTES & NEUTROPHILS IN %
TIME IN HOURS


TIME LEUCOCYTE CURVE.
GROUP - II (L.S.)
TOTAL NR. OF LEUCOCYTES PER CU. MM.
TOTAL COUNT W.B.C (DIS)
TIME IN HOURS


TIME TEMPERATURE CURVE
GROUP - II (L.S.)
TEMPERATURE IN DEGREES (F.)
TEMP. (DIS)
TIME IN HOURS

REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON AMYLASE AND HAEMOLYSIS TESTS AND AMYLASE ESTIMATION IN BLOOD.

REVIEW OF LITERATURE AND PRESENT OBSERVATIONS ON THE TAYLOR'S TEST AND AMYLASE IN BLOOD.

Amylase and Haemolysis tests:

Taylor (1961) developed two new laboratory tests for the diagnosis of hog cholera. He conducted the haemolysis and amylase tests with the pancreatic extract of normal, swine fever immune and swine fever infected animals. He observed haemolysis of the rabbit blood by the normal and immune pigs while the infected ones failed to haemolyse. With the amylase test the normal and immune pigs hydrolysed the starch whereas the infected ones could not. The hydrolysis was assumed to be due to the amylase activity. Taylor et al (1962) further modified the method, observed diminished activity of the pancreatic amylase and also claimed 70% success in a field trial. Tadeusz wijaszka(1964), slightly modified the test, investigated their efficacy and found animals that died due to other diseases reacted mostly in a negative way. Only pigs suffering from acute food poisoning showed a positive or doubtful reaction. Malik (1962) applied the tests upon a number of healthy, swine fever infected and pigs suffering from erysipelas, E.coli, virus pneumonia, streptococcal infection, gastro-enteritis,ets., concluded only amylase test as a diagnostic aid which may be useful as a field test. Dubansky (1963), tested the specificity of both the tests on 500 healthy, in 25 killed 6 days and 5 which had died 14 days after the experimental infection with swine fever, in 100 with swine influenza, in 26 with Teschen and in 2 with generalised

tuberculosis. He found diminution in the amylase activity in swine fever infected animals as well as in the tuberculous cases. The haemolysis test was positive in all 30 swine fever infected pigs and in two of the Teschen cases.

Mickwitz et al (1964) and Karlovic et al (1964) also reported high per cent of efficacy of the amylase test but also observed that such reactions were also seen in other diseases though to a lesser extent. They concluded the test to be of supplementary diagnostic value and for excluding swine fever.

Gray et al (1962) observed the tests not reliable enough for diagnosis of swine fever.

Amylase in blood:

Keast et al (1962) determined the amylase activity from sera prior to and at various stages after infection, from 24 pigs infected with two different strains of virus. Control sera from 14 normal pigs were also collected. The serum amylase values of both the infected groups were akin to those of normal pigs.

Present observations on Taylor's test:

The tests were conducted in both the groups of pigs. The amylase test gave positive results in 80% of the cases and 20% doubtful reaction, while the haemolysis test was positive in 70%, doubtful in 20% and negative in 10% of the cases. All the four normal and two control reacted in a negative way to both the tests. The results of the tests have been incorporated in Table III.

TABLE- III

Result of the Haemolysis and Amylase tests

Sl. No.	Pig number	Haemolysis test	Amylase test	Time elapsed after infection (in days)
1	368 A.S.	+	+	7
2	375 A.S.	+	+	9
3	370 A.S.	±	+	11
4	367 A.S.	+	+	13
5	456 A.S.	+	+	18 (died)
6	431 L.S.	±	+	7
7	438 L.S.	+	+	9
8	463 L.S.	+	±	11
9	364 L.S.	+	+	13
10	432 L.S.	-	±	16 (died)
11	4 samples normal	-	-	-
12	449 & 334 controls	-	-	-

A.S. = American strain of hog cholera virus
L.S. = Local strain of hog cholera virus

Haemolysis test:
Positive - Typical button formation and no haemolysis
Negative - Haemolysis
Doubtful - Incomplete button formation and partial haemolysis

Amylase test:
Positive - Blue tint
Negative - Brown to light brown
Doubtful - Shade between brown to blue.

Present observations on amylase in blood:

In the present studies which were conducted only in the second group of pigs infected with the local strain of swine fever virus, considerable decrease in the blood amylase values were observed at different stages of the infection, vide Table IV. These observations are quite contrary to that of Keast et al (1962) who did not find any change in the blood amylase values.

TABLE - IV

Amylase estimation in blood of the second group of pigs infected with L.S.of virus(Colorimetric method)

sl. no.	Pig no.	Amylase unit per 100 ml.of plasma		Remarks
		Amount of amylase in pre infection period.	Amount of amylase in post-infection period.	
1	431 L.S.	262.5	163.93	5th day after infec.
			142.39	7th day -do-
2	438 L.S.	286.66	279.00	5th day -do-
			181.05	7th day -do-
			171.42	9th day -do-
3	463 L.S.	364.94	309.09	5th day -do-
			255.50	7th day -do-
			166.62	11th day -do-
4	364 L.S.	409.56	326.08	5th day -do-
			230.31	7th day -do-
			140.00	13th day -do-
5	432 L.S.	265.00	250.00	5th day -do-
			167.35	7th day -do-
			91.20	16th day(died)-do-
6	449 C.	356.25	366.05	5th day
			359.20	7th day
			363.50	16th day(killed)

L.S. = Local strain of hog cholera virus
C. = Control.

DISCUSSION AND CONCLUSIONS