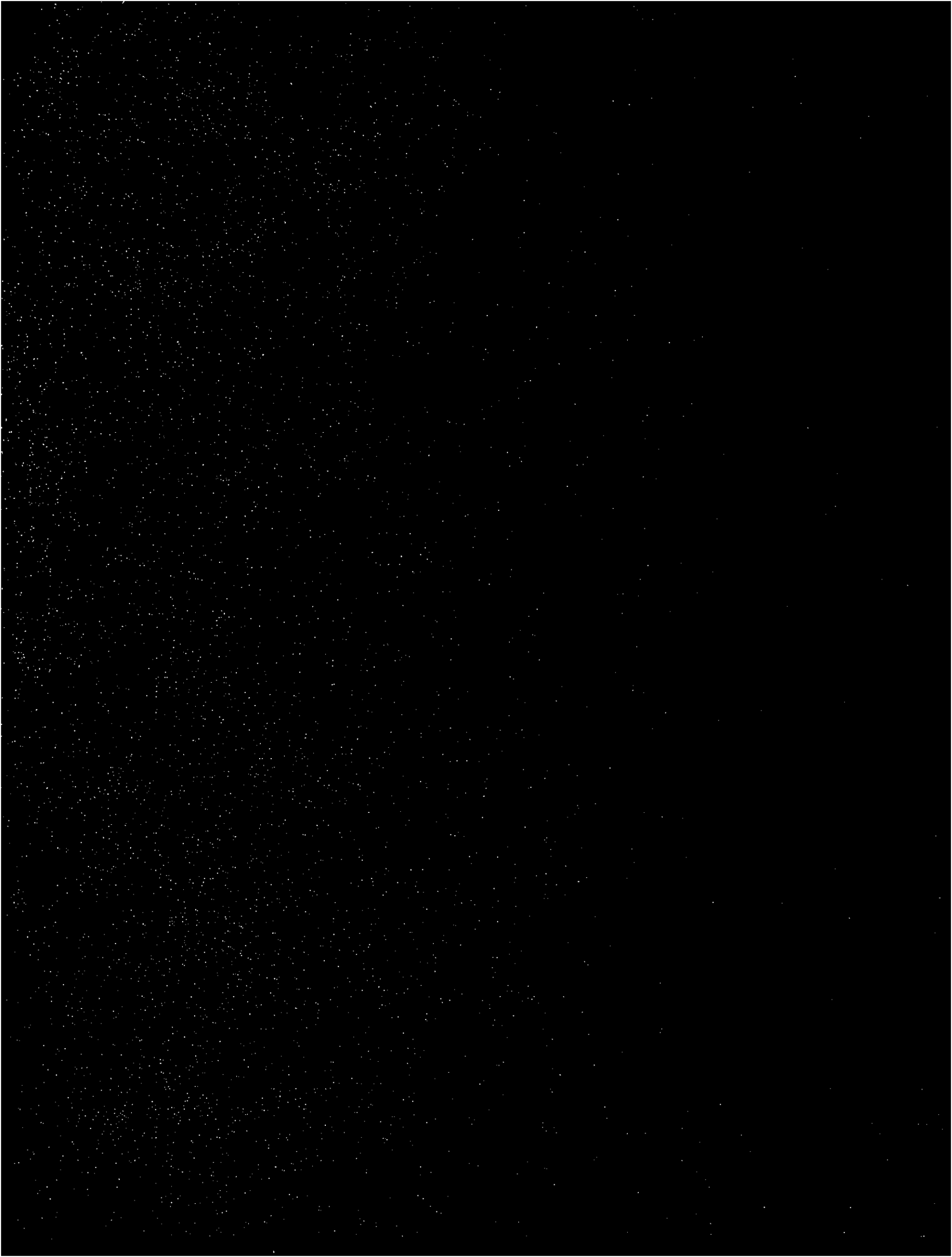

STUDIES ON THE PATHOLOGY OF RINDERPEST IN GOATS DUE TO MUKTESWAR GOAT ADAPTED VIRUS

(Strain Line "W")

Thesis

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

H. N. Thakur
B. V. Sc. & A. H.
Bihar Veterinary College, Patna

October. 1963

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H. N. THAKUR

I certify that this Thesis has been prepared under my supervision by G. S. Thakur, a candidate for the M. Sc. (Vet.) with Pathology as major subject, 1963, and that it incorporates the results of his independent study.

P. B. Kuppuswamy

(P. B. Kuppuswamy)

October, 1963.

B.A., G.M.V.C., B.V.Sc., F.C.(NZ)., M. S. (Missouri)., Vice- Principal and Professor of Pathology & Bacteriology, Bihar Veterinary College, PATNA

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CONTENTS

CHAPTER - I

INTRODUCTION

Rinderpest is a disease of antiquity and was one of the foremost causes of death in ruminants, particularly cattle and buffaloes, in Asia and Africa.

This malady was prevalent since times immemorial and cattle mortality from this disease was so extensive that agricultural operations had frequently to come to a stand still resulting in periodic visitation of famine. The Government of India was so much concerned that they thought it necessary to constitute a Royal Commission, "The Cattle Plague Commission" in the year 1869 to examine ways and means of protecting cattle from the ravages of this disease. As a result of its labours and according to its recommendation, the Imperial Bacteriological Laboratory (now called "Indian Veterinary Research Institute") came into being to undertake systematic experimental work for the control of this disease and the veterinary departments were started for the field control of Rinderpest, Surra and Anthrax.

During the first few decades, the most popular - control method was immunization with immune serum and serum simultaneous inoculations.

About the year 1930, the adaption of rinderpest virus to the goats and the demonstration that such virus could be used without the supporting dose of serum for active immunization of cattle in the field opened up the possibility of large scale active immunization compaign in India and other countries where the rinderpest was a problem.

vaccination against rinderpest is given up, the future population of cattle is bound in the course of some decades to be naturally far more susceptible to this disease than the present strain of cattle. Under such circumstances, the possibility of such a highly susceptible cattle acquiring infection from sheep and goat adapted virus may not be totally ruled out.

However, we may say that the problem of rinderpest amongst sheep and goats which forms a major part of total Indian live-stock i.e. 60.8 millions goats and 40.2 millions sheep (according to 1961 census) is not negligible and therefore it merits a serious study even on its own account. It would not be out of place to mention here that the disease of sheep and goat in general did not till recently, receive the attention they deserved, which were no doubt partly due to the difficulties in arriving at a correct diagnosis.

Sheep and goats play a very important role in the economy of our country and it is of greater importance for India than for the most other countries of the world that they should be protected from diseases particularly from rinderpest in which mortality has been recorded to exceed more than 70 to 90% (D'Costa 1933). However, rate of mortality varies in goats depending upon the strains of virus involved, its virulence, the resistance, season, age and breed of the animal concerned.

Of course, a good deal of work has been done on the incidence, etiology, immunity, vaccine production, control and also on the pathological aspects of this disease mostly in cattle, but literature available on the symptoms, haematology and histopathology of this disease in goats, is only, meagre.

Brief accounts of its outbreak as it occured in goats are to be found in the writings of such early workers as Galambos(1861), Chiccli (1863), Kock (1897) and Lopacio (1926) all working in countries outside India.

In India, itself, Hallen, as far back as in the year 1871, mentioned the infectivity of the bull virus for goats. Oliver (1913) working in U.P. reported occurance of rinderpest in sheep and goats with the higher mortality rate in the latter. Edwards (1927) and Cooper (1931) D'Costa et.al. (1936) and Khanda (1952) reported occurance of natural outbreaks of this disease with a moderate rate of mortality.

It needs no emphasis that in order to understand the disease processes, a knowledge of pathological change is indispensible. Clinical symptoms and related histopathological changes at different stages of a disease, leads to clearer conception regarding the various pathological phenomena which in turn would help in the correct diagnosis of the disease.

The present study was therefore undertaken, in view of the paucity of ~~literature~~, on clinical manifestations, haematological picture and pathological changes due to rinderpest in goats, of the plains, inoculated with freeze-dried goat adapted vaccine strain of virus and efforts were made to correlate the various findings.

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CHAPTER II

HISTORICAL ACCOUNT

When we look back on the available literatures on rinderpest, no doubt, it is found that plenty of work has been done on this important disease mainly in the tropical and subtropical countries but most of it are on the immunological aspects, there being extremely few works on the clinical symptoms histopathology and haematology of this disease, and that too very few with respect to sheep and goats.

The histopathological and haematological work hitherto reported in the literatures by various authors has been reviewed briefly and the detailed review, however, would be done in the respective chapters along with the description of the changes observed during the course of this investigation.

Kavitsch (1864) narrated mainly the histopathological changes due to rinderpest in bovines in the digestive tract. Gerlach (1873) and Semmer (1875) gave macroscopic as well as micropic lesions caused by this disease also in bovines.

In Egypt Carlong and Ball (1908) reported a systematic description of the histopathology of this disease studies in ~~the animals~~ cattle, died of natural out-break as well as sacrificed after artificial infection .

Ahmed Shafiq - Bey (1918) and Lobberstein (1928) drew attention to the changes produced by rinderpest virus in cattle, on the vessels of the heart and on nervous system respectively.

Histo- pathological account of a chronic form of malady in cattle was also given by Pavanelle (1922).

Gerlach (1933) studied the difference in the histopathological changes exhibited in cattle dying of this disease alone, with those dying of infection complicated with piroplasmosis.

Fukshima (1934) and Fukshima and Miyaini (1935) described these changes in liver and spleen respectively studying on calves artificially infected with this disease. Mornet,P,et al (1942) discussed the diagnostic value of lesions of the abomasum and ileo-caecal valve in rinderpest on experimentally infected calves at Niamey & Dakar.

Khera (1955) studied and discussed in detail the histopathology of different organs infected artificially with different strains of this virus including that seen in goats infected with the goat adapted virus. Maurer et al (1955) gave a detailed description of clinical symptoms, histopathology and haematology of this disease. These observations were based upon natural cases of this disease in numerous field outbreaks in cattle in equatorial Africa and a large number of experimental cases, too, of which clinical, haematological and necropsy records were maintained. In the year (1956) Theiry et.al. (1956) made an important study of the lesions as seen in cattle, goat and rabbits in French West Africa. They also made another important study in the same year on haematology and histopathology of rinderpest with special reference to the cellular inclusion bodies in this disease of bovines.

Records are also available of this important disease occuring in sheep and goats from different parts of India as well as from outside this country. Rogers (1900) refers to an outbreak of cattle plague in the Himalyas which was introduced into two villages by infected goats with most deplorable results.

Lingard (1905) observed a black buck contracting rinderpest and succuming to it at Mukteswar, and Gartner (1920) considered goats of such importance that he devoted a full chapter on them as virus carriers inspite of a little earlier statement by Angeloff (1917) to the effect that sheep and goat although susceptible by an artificial inoculation did not contract this disease naturally.

Schein (1924) of Indo China kept goat virus infective to cattle during 172 passage from goat to goat, and Robertson (1925) also gave an account of rinderpest in Western Australia and stated that the evidence appeared to indicate that sheep brought to Fremantle as live ship's stores and which were purchased by local butchers for slaughter were the source of an outbreak.

Seaton (1931) working in Nigeria proved that goats are susceptible to rinderpest by contact. That contact infection takes place from cattle to goats and vice-versa has been proved by Hall (1930). During 1938 this disease was observed to be widely prevalent in the state in which the sheep suffered most although the disease was simultaneously present amongst goats, cattle and swine.

From the point of study of natural infection of Rinderpest in goats it may be said that Chiodi (1863) cited by Hutyra and Marek (1926), Kock (1897), Nocard and Leclainche, Mohler and Eichhorn, Lopacio (1926) all working outside India probably were the early workers who first proved the susceptibility of goats to rinderpest. Oliver (1913) working in the then United Provinces (now Uttar Pradesh) mentioned the occurrence of rinderpest in sheep and goats both, with a heavy rate of mortality in latter species of animals.

D'Costa et.al. (1936) described a natural outbreak of rinderpest in sheep and goats associated with a moderate rate of mortality. Viswanathan (1937-1938, 1939) also reported the occurrence of a severe type of outbreak of this disease amongst sheep from many districts of Madras. Naik (1938) brought into light an outbreak amongst sheep and goats in Bombay state, in which it was mainly confined to sheep although a few goats were also attacked with this disease with a high rate of mortality in sheep.

Rawa in (1939) working in Ajmer and Merawara in (1937-38) reported a severe outbreak of rinderpest in sheep and goats causing a heavy mortality which was also confirmed by biological tests, and during the year 1939 to 1945 several outbreaks of rinderpest amongst these animals were reported from different states of India notable amongst them being those reported by Gopal Krishnan (1939-40, 1940-41 and 1941-42) from Assam, Mahajan (1939-40) Hyderabad, Khan (1939-40), Yakub (1940-41), Gopal Singh (1942-43) from Punjab and Parimo (1945) Kashmir.

Reports of a country wide prevelance of rinderpest amongst sheep and goat came to light when an organised investigation work of animal diseases sponsored by the Indian Council of Agriculture Research came into operation. In the year (1950) a wide spread of natural outbreak of this disease amongst these animals was reported from the State of Bombay. It was described and confirmed by Khanda and Manjrekar (1952) giving details of symptoms, lesions, and histopathological changes in the various organs.

Cornet, P, et.at (1956) also reported for the first time a rinderpest like disease of sheep and goats in French West Africa in which they have described clinical features and the lesions

both macroscopic and microscopic which closely resemble those of rinderpest. They have further observed that in the field, spread of infection in cattle does not seem to be ~~spread~~ from infected goats. From the point of view of experimental infection in goats with rinderpest virus, Allen, in India as far back as in the year 1871 mentioned the infectivity of the bull virus for goats and also claimed to have successfully infected 3 out of a batch of 8 goats with the disease. Konchello (1917) observed that goat living on the scene of an outbreak did not get the disease naturally and were unable to transmit it to transport cattle housed with them.

Topacio (1927) working in Phillipines conceived the idea of utilising the lesser susceptibility of goat to a great practical value similar to the employment of calves in the production of small pox vaccine. He had the vision that it might be possible to obtain a virus by a single passage from cattle to goat that was sufficiently potent to infect Phillipines cattle.

Edwards (1927) and Cooper (1931) both working in India have published brief notes upon the disease in goats with a special reference to the degree of rinderpest reaction requisite in these animals for the production of a potent virus and they held the view that some cases of pleuro- pneumonia in goats in this country were due to rinderpest.

Crawford (1948) made an observation that occurrence of rinderpest in goats in Ceylon was due to the goats imported from India for the supply of meat to Indian troops stationed in Ceylon and he was of the opinion that these goats suffered from a modified form of the disease as a result of infection with goat adapted virus which was extensively used for the prophylactic vaccination in the

areas from where these goats had been collected which had resulted in the outbreak of this disease in those animals. However, it is difficult to say how far this view can be accepted in the light of Sethuraman (1948) who had experimentally disproved the possibility of the spread of infection from animals vaccinated with goat adapted virus.

From the foregoing records, it is, therefore, definite now, that goats do get infected with rinderpest, but the disease is very mild and passes off unnoticed in most cases in the natural state and the possibility of such goats, infecting cattle in their midst with attenuated virus can not also be ruled out.

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CHAPTER II

MATERIALS AND METHODS

Male castrated healthy goats of " Deshi Breed " of about a year and half old, average live weight being 37 lbs. each, were obtained for the present study. These animals were supplied through a local contractor from the plains of north Bihar.

These goats were brought before the rains in the month of April and were kept in a clean, dry and airy stables. They were fed on the same standard ration with grazing and adlib supply of water through out the course of this experiment.

The same utensils and water pans were utilised for a particular batch of experimental goats, separately, for the disease and healthy ones. The rooms used to be cleaned daily in the morning.

Clearance and washing of hands and legs with disinfectant before entering and leaving the rooms was followed. The attendant used to clean first the rooms occupied by healthy goats and then those of the infected ones.

For the sake of present study goats were slaughtered at different time intervals.

Blood collection and examination:-

Blood was collected aseptically from the jugular vein of the goats in the morning before any food was given. About 2 c.c. of blood used to be drawn with a 20 gauge needle and a 5 c.c. record sterilised syringe.

A few drops of the blood, thus collected were transferred to two clean slides and uniform smears were made for the differential count of the sample.

The rest of the blood was transferred into a clean test tube containing a suitable quantity of "Wintrobes isotonic ammonium and potassium oxalate mixture as anticoagulant evaporated to dryness in the tube on a water-bath previously. These tubes were shaken for about 2 minutes to ensure a thorough mixing of the anticoagulant and the blood.

This blood was used for the examination of the total Red Blood Cell counts, total leucocytic counts, haemoglobin percentage, sedimentation rate, packed cell volume, Corpuscular volume and chloride test.

Double Neubauer's counting chamber was used for erythrocyte and leucocyte counts. Haemoglobin estimations were carried out on Sahli - Adams pattern of haemoglobinometer.

The blood smears were stained with Leishman's stain and ordinarily 200 leucocytes were counted, classified as Neutrophiles, Lymphocytes, Monocytes Eosinophiles and Basophiles.

In the present study, at first 19 experimental healthy male goats were subjected to different haematological tests during the pre-infection period. Three readings of such investigations were done upon all the healthy goats to obtain the normal haematological values.

During this period the faeces of all the goats were examined daily by direct, floatation and sedimentation methods for coccidial oocysts & helminthic ova. Temperature of all these animals were recorded and blood smears were also examined, stained by Giemsa's method for protozoan blood parasite etc. Only those goats found free from any disease and showing normal rectal temperature were divided into 3 groups of six each and kept under observation for about 15 days prior to infection.

In the first batch, 5 goats were infected with Muktesvar goat adapted virus strain Line "W" of known titre obtained from the Livestock Research Station, Patna, subcutaneously with 2.c.c. of 1 in 100 dilution of the virus in the shoulder region. Further one control goat in each batch was also inoculated with 2 c.c. of Normal Saline solution s/c in the same region.

The route of infection and the dose of the virus were kept constant in all the three batches during the course of experiment. Haematological values including the blood chloride value of all the goats were determined during the post infection period from 24 hours after infection, and onwards prior to their killing, daily in the morning. One goat in each batch was killed at intervals of 48 hours, 72 hours, 120 hours after infection and one infected goat was left till it succumbed to death due to infection; for the study of histopathological changes. The same haemotological and blood chloride studies of one control goat in each batch were also done similarly add this control animal was also sacrificed for normal histological studies. The temperature of the infected animals were recorded morning and evening. Moreover the clinical symptoms temperatures and histopathological changes of the goats killed on 5th day after infection were also obtained and observed in the goats killed for the F.D.G.T.V. vaccine production in the Biological section of the L.R.S., Bihar, Patna.

The data obtained includes total red cell count, total and differential white cell counts, haemoglobin percentage, sedimentation rate, packed cell volume and chloride in milligrams of Nacl/ 100 c.c. of whole blood.

(1) Total erythrocyte count:-

Physiological saline containing 0.85% saline was used as diluent in preference to Hayem's and Gower's solution for the red cell count, according to Oscar W Schalm Veterinary Haemotology,1961. As the number of cells in the goat's blood are much more compared to other species of mammalian's and usually agglutenation of erythrocytes used to occur with Hayem's solution, physiological saline was used in the present experiment.

Blood only up to the 0.2 mark of the red cell pipette of the haemocytometer was taken so that dilution may be 1 in 2500 to facilitate the correct counting of the cells. The erythrocytes were counted directly in the counting chamber. Turk's solution was used as diluting fluid for the leucocytic counts.

(2) Preparation of stained blood smears:-

Properly spread blood films made on flanned and polished absolutely clean slides. Uniform smears thus prepared were stained by Leishman's staining technique. Two hundred leucocytes were counted in each slide following the "battlement" system (1 m.m. down, 1 m.m. across and 1 m.m. up).

(3) Haemoglobin determination:-

Haemoglobin percentage was obtained by Sahli-Haden Haemoglobino meter following the directions for its use.

(4) Corpuscular volume and sedimentation rate:

Scrupulously clean Wintrobe's haematocrit tubes with a uniform 3 m.m. bore and a double 10 cm. scale caliberation with millimeter divisions were used. Six such tubes were filled with oxalated blood from the goats up to the mark "10" of the tube and were allowed to stand vertically.

Simply one reading was taken at the end of one hour. The Wintrobes tubes were afterwards centrifugalized at the speed of 3000 r.p.m. for 30 mts. and then for 20 mts. more to determine the corpuscular volume which was recorded as the number of ml. of cells per 100 ml. of blood.

(5) Blood chloride value estimation:-

It was determined by adopting the technique given in the manual of colorimetric clinical analysis with the Fisher clinical Electrophotometer.

Preparation of tissue and staining:-

The different organ of the killed goats and the goats that had died due to infection were preserved at the earliest opportunity after autopsy examination in 10% formal saline solution for histopathological examination. Over and above cultural test was also carried out on the materials collected of goats which had died from this virus, in order to, find out presence of any secondary bacterial infection.

Tissue sections were stained with haematoxylin eosin and sections from tonsils, lyaph gland, payer's patches were stained by Mann's methyle blue eosin technique (kraus, Gerlech and Schweinburg) to demonstrate the presence of inclusion bodies if any.

CHLORIDES IN BLOOD

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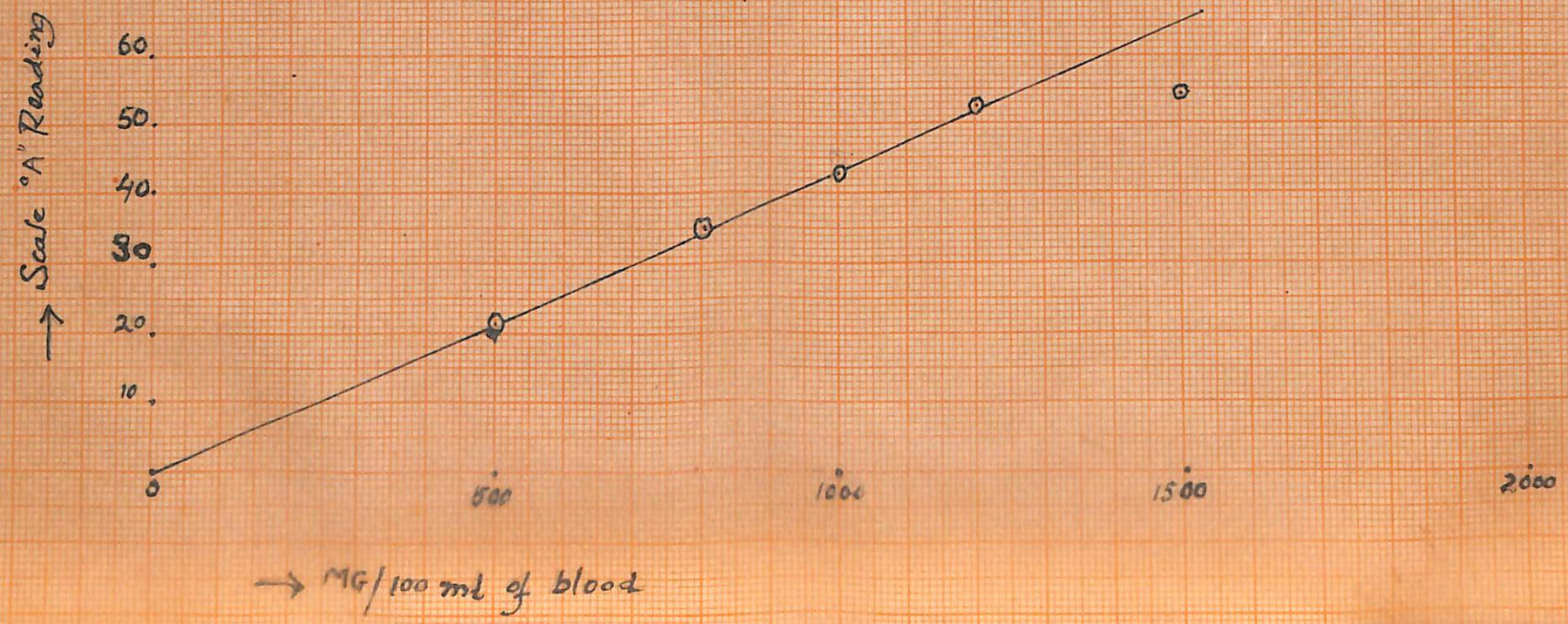

CHAPTER IV

CLINICAL SYSPTOMS AND LESIONS

Review of literature:-

Hallen (1871), Edwards (1927), Beaton (1930), D'Costa (1933) Phada (1952), Theiry (1956) and others who may be considered as the earliest workers described the symptoms and lesions of rinderpest as it occurred in goats. D'Costa (1933) based his observations on natural and artificial infection with this virus on goats of hilly tracts like Mukteswar at an altitude of 7,500 ft. above the sea-level as well as on the animals in the plains at Izatnagar.

The observations of Phanda (1952) was done on sheep and goats, affected in a natural outbreak of this disease in some village of southern border in the state of Bombay. According to the observation of the above mentioned workers, the period of inculation was estimated to be 2 to 5 days in this disease with an initial rise of temperature at 52nd hour of infection from the time of inoculation in the experimental cases. The rise of rectal temperature was noted to be 104 ^{0}F to 106^{0}F, maximum. D'Costa et. al further noted two types of symptoms in this disease as manifested by the goats as an acute type, which was usually compli ated with pneumonic symptoms and ended fatally and in others a subacute or the uncomplicated form of the disease, which usually led to recovery of the animal affected. They further observed that the principal clinical changes they came across were often pneumonic and not alimentary as in bovines.

According to them goats of the high altitute were more suceptible to this virus with a higher rate of mortality than the animals in the plains. Hallen(1871), Beaton (1930), Edwards (1927) and Cooper (1931) all observed that the

respiratory involvement of this disease in goats as a constant feature.

In the case of present study the earliest rise of temperature 104 °F to 106 °F was noticed as early as 48 hrs to 72 hrs. of inoculation of the virus. Temperature often fell to normal or even subnormal after the onset of diarrhoea on the 8th day of infection. The most significant symptoms observed were the cessation of feeding with the initial rise of temperature, decreased rumination accompanied with dullness and depression. This was usually followed by marked symptoms like dry muzzle, rough starring coat, nasal and conjunctival catarrh, hurried respirations, with signs of dyspnoea and arched back. Dry cough and constipation followed by diarrhoea was seen in all the prolonged cases. As the disease advanced the discharges from the eyes and nose became more copious and thick whitish, and mucopurulent (Figure No. 2).

Diarrhoea usually set in on the 7th or 8th day after infection which became progressively more severe; from soft and controlled to profuse, thin and green to yellowish brown or grey. The faeces finally having a very offensive odour containing mucus and terminating more profuse, watery and uncontrolled evacuation. This gave a soiled appearance of the whole of the anal regions and hind legs.(Fig No. 3). With this increased severity of diarrhoea, there was also noticed abdominal pain, accelerated and laboured respiration, occasional cough, severe dehydration; followed by prostration and subnormal temperature and death within 11 to 13 days of infection, coughing and respiratory involvement was seen in all the prolonged cases with a characteristic type of breathing. Respiration become laboured painful with a peculier moaning sound and the expirations halting. (Fig. No. 4). The laboured respiration is believed to be

compensatory to the small blood volume resulting from extreme dehydration.

These animals were seen to inhale normally, start to exhale but then the glottis closes while the respiratory muscles are relaxed. This results in an abnormally high lung pressure which is released audibly when glottis opens and thus a characteristic moaning sound is produced. Respiration of this type was commonly observed and the increase of lung pressure preceeding each exhalation may account for the development of emphysema. And once this emphysema is established, the vicious cycle of pain, pressure emphysema and laboured respiration is aggravated. Such animals were found to prostrate for several days with laboured respiration and showed severe and extensive pulmonary emphysema at necropsy. (Fig. No. 5).

A careful examination of these goats with respiratory involvement at the terminal stage revealed pleurisy and partial or complete consolidation of especially the anterior and middle lobes of the lungs. However, Nanda (1952) did not notice any thing unusual in the lungs in a natural outbreak of this disease in sheep and goats. However, the very large masses of available experimental evidences prove that the pneumonic symptoms constitute a remarkable constant feature of the rinderpest syndrome in goats.

In the present experiment, subacute symptoms were also seen in one goat only with signs of its recovery from the infection on the 7th or 8th day, of course, after an initial febrile reaction and in this case, pneumonic symptoms, such as laboured breathing, cough and the diarrhoea so frequently noticed in the acute types were entirely absent.

Post-mortem lesions :-

Emaciation, dehydration, sunken eyes, rough coat with whole of the anal region and hind legs soiled with discharges and diarrhoea were seen in all the fatal case. (Fig. No. 6).

Digestive system:-

Erosions and epithelial necrosis of the buccal mucus membrane as reported by Khanda and Manjrekar (1952) in a natural outbreak of this disease in sheep and goats was not observed in the present experimental study. However, only, slight congestion of gums and buccal mucuus membrane and the posterior portion of the hard and soft palates were noticed. There was apparently no remarkable change noticed in the pharynx and the surrounding parts. The oesophagus, rumen, reticulum and omasum apparently looked healthy.

But the abomasum frequently presented the evidence of deep profuse congestion in all the cases examined at different stages of the disease. These lesions were more marked in the pyloric regions with presence of ulcers in cases died of infection. (Fig. No. 7). The duodenum showed slight congestion. Necrotic foci of microscopic size in the epithelium, accompanied with capillary congestion and petchial haemorrhage in the underlying lamina propria, resulted in a gross appearance of irregularly outlined, superficial streaks of colour which ranges from bright to dark brown.(Fig. No. 8). Oedema was profuse in the submucosa giving it a grossly thickened appearance.(Fig. No. 9). The ulcers, were, as a rule coated with greyish deposits which were fairly adherent.

The whole of the intestinal tract was extremely congested and filled with liquid ingesta. Severe ulcerations were also frequently observed in the fatal cases.(Fig.No.10).

The Caecum and the ileo-caecal valve almost invariably showed a very acute haem rrhagic and oedematous inflammation. The high susceptibility of lymphoid tissue with this virus frequently results in severe involvement of the Peyer's patches. Even in the presence of a relatively normal adjacent membrane, the Peyer's patches seem to slough out, leaving a deep raw crater in the intestinal wall. (Fig. No. 11).

Colon and rectum:-

There were no marked lesions in the colon and rectum as seen frequently incases of cattle though slight congestion and oedema of the mucous membrane was observed frequently.

Liver and gall bladder :-

In some cases the liver was found, enlarged, fragile but in others nothing beyond a slight congestion could be seen. The gall bladder was usually observed to be distended with thin greenish bile but there was no marked changes in its mucous membrane.

Lymphatic system:-

Lymphoid Tissue - (spleen, Lymph glands, & Peyer's patches). -

Rinderpest virus has a particular affinity for the lymphoid tissue which was strikingly evident in the mascroscopical and in microscopical sections of the lymph glands, spleen, and Peyer's patches. Oedema of the lymph glands was detected grossly in the initial stages of the infection with its soft and enlarged appearance.

Grossly, the spleen was found to be normal, usually, but enlargement and splenic swelling were frequently marked in the goats on which necropsy examination was performed on the 3rd day after the inoculation of the virus. These spleens when cut with a scalpel, its Malphigian corpuscles gave an appearance of Sago-Spleen.(Fig. No.12).

The splenic swelling as seen previously was reduced as the infection advanced; and finally at the terminal stage of infection, the gland looked dry and atrophic. The same changes were observed in the Peyer's patches also which at times sloughed out leaving deep crates in the intestinal wall.

Respiratory System:-

There was no significant macroscopical change in the larynx and trachea except a slight congestion. Lungs were constantly involved in all the experimental cases observed; though there was less congestion and alveolar emphysema of the apical lobe, in goats post-mortemed after the 3rd, 4th, 5th and 6th days of clinical illness. (Fig. No. 13). On the other hand in prolonged cases of long standing duration, involving diarrhoea, dehydration, emaciation and in which laboured abdominal respiration and coughing developed, interlobular and alveolar emphysema were characteristically observed. Thus pneumonic consolidations were found in the lungs in all the fatal cases, with the anterior and the ventral portions of the diaphragmatic lobes most commonly involved. (Fig. No. 14).

Circulatory system:-

Pin-point haemorrhages under the epicardium were seen in about 25% of the cases examined at necropsy. The myocardium of heart was frequently found to be flaccid, none of the other changes were significant. (Fig. No. 15).

Urinary System:-

Oedema of the kidney in general and around the pelvis with limited congestion in the medulla and particularly at the

cortico-medullary junctions, were seen in the cases on the 3rd, 4th and 5th day of infection (Fig. No. 16). Urinary bladder did not reveal any particular lesion.

Brain and Spinal cord :-

In these organs no significant change was noticed grossly.

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CHAPTER V

HISTOPATHOLOGICAL CHANGES

Review of literature :-

Spleen - Fukshima and Miayri (1935) based on the examination of spleens of 54 calves infected artificially with the rinderpest virus and two calves that had contracted natural infection, describe that on onset of the malady, reticular cells in the follicle as well as in the germ centre proliferate so that the follicles assumed more than double the size of the normal. Subsequently a similar reaction was also noticed in the red pulp. This was followed by focal degenerative changes and necrosis in the follicle as wellas in the red pulp towards the terminal stages of the disease. Haemorrhage was seen not only in the centre and around the follicle but also in the pulp and trabeculae. Focal polymorphonuclear leucocytic collections were less commonly seen and when present were confined to the pulp only. Haemosiderin deposition increased in the pulp and was sometimes abundant..

Khanda and Manjrekar (1952), who observed a natural outbreak of rinderpest in sheep and goat, reported an intense congestion of the spleen resulting in the atrophy of the lymphoid tissue. Retention of the pigment with marked deposition, slight thickening of the trabeculae and marked thickening of the capsule and blood vessels with a tendency of the formation of new blood vessels in the latter; especially in the malpighian corpuscles were noted by him.

Khera (1955), while studying on the experimental infection of bull with different strains of rinderpest virus and on the goats with goat adapted strain, noted reticular hyperplasia

progressing with the prolongation of the infection, in focal degeneration of the cells and erythrocytic infilteration, heavy concentration of neutrophiles around the Malphigian follicle, necrosis, generalized lymphorexhis and finally reduction of Malphigian follicle to a mere halo round the central artery. He further observed in goats , initial progressive hyperplasia with gradual enlargement of the size of the follicle but with the advancement of the infection after 6 days he noted reduction in size of the follicle with slight congestion in the red pulp.

Maurer (1953) has described in cattle that necrosis of lymphocytes was strikingly evident in microscopic sections. He recorded severe loss of lymphoid cells often leaves a fibrillar, somewhat eosinophilic and acellular matrix in places of the highly cellular lymphoid follicle.

Theiry G (1956), who made an important study of the lesions of rinderpest in cattle, goat and rabbit, reported congestion and enlargement of the lymphatic organs, as important lesions.

Sequence of pathological changes in the present study:-

In the beginning of the infection i.e. on the 3rd and 4th day malpighian follicle showed reticular hyperplasia and thickening of the walls of central artery.(Fig.No. 17).

Thickening of the capsule, subcapsular haemorrhage, erythrocytic infilteration, accompanied with haemorrhage around the periphery of the follicle was noted. (Fig.18)(18a) Follicle decreased in size with indistinct outline and increased migration of lymphoid cell with advaced stage of infection. (Fig. No.19).

In fatal infections areas of focal necrosis in the follicle with extensive haemorrhage was also seen. In these cases lymphorrhexis and reduction of Malphigian follicle to a mear hollow round the central artery was also not infrequent.(Fig. No. 20) . Trabeculae of the spleen showed in some,perivascular lymphocytic infilteration. Lymphoid destruction evident by fragmentation and disappearance of the most of the mature lymphocytes from the germinal centre was noted. (Fig.21 2(1A)). Merely a shadow of the Malphigian follicle with its dentral arteriole surrounded by small and medium sized lymphocytes around was also seen in some fatal cases of this infection in goats. Pigmentation was also o noticed in some cases of advanced infection. (21 A)

Histopathological changes in the Lymph nodes:-

Review of literature.

Ravitch (1864), Perlach (1873) and Semmer (1875) while describing histopathological changes in rinderpest did not mention about the lymph nodes. Arlong and Ball (1908) dealing with an outbreak of Rinderpest in Egypt stated that lymph nodes although normal in microscopical examination, in a few cases, fibrinous network of lymph follicles was seen. Fukusho and Nakamura (1934) while comparing the microscopic lesions in rabbits caused by lapinised virus with those of cattle affected with cattle plague virus, said that necrosis of lymphatic tissue seen in rabbits was more or less similar to the necrosis of lymphatic tissue in the cattle.

Fukushima and Miyairi (1935) mentioned that the reactive proliferation of reticulo - endothetial system of lymph node was similar to the corresponding changes in the spleen and said that for diagnosis probably these two organs would give a pathognomonic histo-pathological appearance.

Shera (1955) studied, in details, the histopathologic changes in lymphoid tissue with particular reference to the lymphnodes. He stated to find out almost the same sequences of changes as found in the spleen i.e. reticular hyperplasia and their differentiation into reticular histocytes and macrophages. Bi and trinucleated forms of macrophages and giant cells were also noticed. He further noted that on about the 8th day after infection the various phagocytic elements and lymphocytes underwent necrosis. Reticular fibres and reticular cells also showed retrogressive changes. Towards the terminal stage the

lymphnodes were found to be the sites of capillary sprouting and showed indications of developing fibroblasts leaving behind areas of focal lymphatic degeneration, in bovines.

In the goats he found that the follicles of the lymphnodes showed reaction centres which persisted up to the fifteenth day; cells bridging lymph sinuses were seen to proliferate and differentiate histocytes and macrophages. These phagocytic elements manifested necrotic changes, while the lymphocytes in the sinuses showed mitotic division.

Maurer (1955) also observed that the lymphocytes in the lymphnodes and spleen were almost completely destroyed and same was the observations of Theiry (1956), all in bovines. Theiry further noted disappearance of the germinal centre and the presence of a large number of polynuclear neutrophiles which undergoes necrosis.

Histopathological changes observed inthe present study was similar to those seen in spleen by other workers. Capsule was thick with a well pronounced focal lymphocytic infilteration at the junction of the capsule and the trabeculae in the initial stages of infection (~~Fig.No. 23~~). Oedema and congestion of capillaries were also seen. Enlargement of secondry lymphoid nodule in size was also noted in some cases. In fatal cases necrosis and in all prolonged cases fibrosis of lymph follicle and severe haemorrhage in others were also noted. (Fig.No. 22 & 23); destruction of lymphoid follicle accompanied and surrounded by plasma cells and neutrophil were also seen in a few slides of fatal infection of goats.

Histopathological changes in the digestive tract:-

Review of literature:-

Gerlach (1873) stated that buccal lesions in the mucous membrane of lower lip, guns, sides of tongue and pharyngeal cavity are frequently seen in animal naturally affected with rinderpest. The cells of mucous revealed the hydropic swelling, with granules, distributed in cytoplasm as well as within the nuclei. He further observed that degeneration in the deeper layers of mucosa, in this disease, as a rule, did not occur in bovines. Arloing and Ball (1908) in an experimental infection of this disease in cattle which died in within seven to nine days did not observe any lesions in buccal mucous. In their opinion, this time was insufficient to develop buccal lesion. However, in natural cases necrosis and desquamation of superficial epithelium and infilteration of necrosed area, with leucocytes were noted. Gerlach (1933) while comparing the histopathological changes of oral and nasal mucosa as well as those of the abomasum of animals that died of natural infection of rinderpest, complicated also with piroplasmosis indicated that epithelial necrosis and crupous deptheroid processes are due to rinderpest whereas catarrhal and haemorrhagic changes in oral and nasal mucosa indicate simultaneous presence of pirplasmosis, in cattle. He further noticed the absence of superficial epithelium and instead the mucosa covered with a thick tenacious mass of mucus and cellulur debris resulting from the degenerating cells of gastric glands of the abomasum of the naturally infected cattle. Desquamation resulting into an ulcer were next noticed. Cells forming base of these ulcers were seen

in various stages of degeneration, in (1873).

According to Arloing and Ball (1908), sub-epithelial haemorrhages, resulting from rupture of vessels, coagulation of extravasated blood, depriving the superficial layers of mucosa of its circulation, brought about necrosis in the fundic region of stomach of cattle infected with this disease. In one case they are also said to have seen a fibrinous thrombi in the vessel of mucosa and submucosa produced by coagulation necrosis of the mucous membrane of the corresponding area. These necrosed areas after infilteration with leucocytes and erythrocytes sloughed off leaving a ddep ulcer.

Intestine - While describing the histopathological lesions in intestine Perlach (1873) is reported to have seen degeneration of the epithelial cells of the crypts of Lieberkuhn, its shedding out, desquamation and necrosis of the superficial epithelial cells. He further stated that the Peyer's patches showed hyperplasis followed by degeneration to produce "purulent material." According to Pavitch (1864), the epithelial cells of crypts of Lieberkuhn were not always affected in this disease whereas proliferation of connective tissue cells and cellular infilteration of lamina propria were a constant feature.

Arlong and Ball (1908) described congestion of the intestinal mucosa, particularly at the ileocaecal valve and other parts of large intestine. Chanda and Manjreker (1952) while describing the histopatholigical changes as found in a natural outbreak of this disease are said to have observed, congestion of the blood vessels of the mucosa & submucosa, thrombosis, catarrh, of the mucous membrane, leucocytic,

infilteration, degeneration, hydropic degeneration karyolysis and necrosis; tendency of desquamation of a part and in others for the intertubular connective tissue in the abomasum of sheep and goats in rinderpest. They further noted similar findings in the small intestines but with a greater tendency for the epithelium to be desquamated and ultimate formation of ulcers.

Ahera (1955) while studying the experimental infection of this disease in hill bulls reported same changes, significant among them being extensive necrosed areas in the tongue. He observed marked congestion and oedema and desquamation of the superficial epithelium of abomasum. He is also said to have seen haemorrhagic infarct due to thrombosis of vessels, liquifactive necrosis, and diptheritic deposits on the glandular epithelium of the abomasum in the different progressive stages of infection. In the small intestine he noticed invagination of the crypts of Liberkuhn, its dilatation, then degeneration, necrosis, haemorrhage, and diptheritic deposits at necrosed area at the different stages of infection. Superficial necrosis and deep and shallow ulcers in the large intestine were also recorded.

In goats with a goat adapted rinderpest virus infection he noted in the small intestine increased lymphocytic infilteration of lamina propria, congestion, haemorrhages in caecum, Peyer's patches .

Maurer (1955) also found almost the same changes in the digestive tract of the rinderpest affected bovines. He stated, that , virus of rinderpest produce necrosis and erosions of the epithelial cells above the germinal layer with relatively little vascular response in the stratafied squamous epithelium of the mouth and oesophagus.

In the columnor epithelium of the abomasum and intestine, the proximity of the highly vascular stroma results in relatively severe congestion and haemorrhage when necroses of the epithelium occurs. He further mentioned that the Peyer's patches and cecocolic junction, which have an abundence of lymphoid tissue, are especially susceptible to the virus. Theiry G (1956) who made an important study on haematological and histopathological studies of rinderpest with special reference to cellular inclusion bodies discussed the role of lymphocytes and polymorphs. Acidiphilic cytoplasmic inclusion bodies which are claimed to be specific have been described in numerous organs especially in the tonsils of bovines.

Present findings:-

Visible lesions in the buccal cavity were not seen in any of the goats dying or destroyed in various stages of the disease, nor any of them revealed any lesion in the rumen, reticulum or omasum.

Abomasum :- Severe oedema, in mucous, submucous and muscular layers with lymphocytic infiltration were frequently seen in the early stages of the disease. Congestion and dilation of vessels and oedema separating muscle fibres and surface erosions were constant lesions. ~~(Fig. No. 24)~~. As the disease progressed beyond 5th day of infection, necrosis and desquamation of the cells, leaving the underlying lamina propria exposed were seen. ~~(Fig. No. 25)~~.

Capillary congestion, haemorrhages necrosis and ulceration with an evidence of penetration of the muscularis mucosa

and submucosa were noted. Surface erosions desquamation of epithelium and ulceration were seen in all the fatal cases. ~~(Fig. No. 26)~~. Liquifactive necrosis of gastric glands, haemorrhages and mucosa being flooded with erythrocytes were seen. ~~(Fig. No. 27)~~.

Duodenum - showed congestion, oedema and focal lymphocytic infiltration of varying degrees in various stages of the disease.

Small intestine - Probably due to high susceptibility of lymphoid tissue slughing out of Peyer's patches in the epithelial lining of the crypts, oedema, congestion, lequefactive necrosis were seen in the mucosa, submucosa and muscular layer of the intestine with infiltration of lymphocytes, macrophages/which were frequent. Necrosis followed by desquamation forming ulcers with haemorrhagic floor was most common in these areas. (Fig. No. 24 27a)

Large intestine - Most well marked lesions were found around ileocaecal valve, and at the caecocolic junctions. Except for oedema and slight congestion no other significant lesions were found in the rectum. Ileocaecal valve and the mucosa around it presented areas of congestion, haemorrhage accompanied by necrosis and erosions ulcers with its base formed by marked concentration of large mono-nuclear cells with pronounced lymphocytic infiltration were seen. (Fig. No. 25 ~~26~~). Other histopathological changes were similar to those of small intestine. At the caecocolic junction the degree of congestion was usually greater than elsewhere. Necrosis of lymphoid tissues,

Histopathological changes in liver and gall bladder :-

Revies of literatures - Arloing and Ball (1908) found chiefly vascular congestion, with cloudy swelling and fatty changes in the histopathological sections of the liver in animals dying of rinderpest . However, only in one case he also observed focal necrosis, intense congestion , and lymphocytic infiltration of chorian in the epithelium of the gall bladder. Ruppert (1921) described concisely changes in liver as parenchymatous hepatitis. According to Fukshima (1924) the chief changes in liver are hydropic degeneration, focal cell accumulation, to the form the miliary cellular nodule. Hutyra and Marek (1946) said " The liver as a rule, shows no changes." They all observed in bovine rinderpest . Gerlach (1933) found necrosis and ulceration of the gall bladder only in cases complicated with piroplasmosis in cattle.

Chronic venous congestion, fatty changes; small encapsulated haematomas in the parenchyma, retention of pigment and certain amount of fibroblastic proliferation were the changes found by Khanda and Manjreker (1952) in a natural outbreak of this disease in goats. These authors are also said to have observed in one such case an extensive cirrhosis, but probably of helminthic origin and in one of the cases examined, all stages of degeneration of the parenchymatous cells starting from cloudy swelling and culminating in necrosis were found.

Khera (1955) also observed swelling of the parenchymatous cells, minute foci of necrosis-"miliary cellular nodule" and in some cases inspissated bile in the bile canalicules in hill bulls.

He further changes in the goats are also the same mainly cloudy and fatty degeneration, and enlargement of the kuffer cells.

Maurer (1955) states that liver in the bovine rinderpest, is only , secondarily and chronic venous congestion generally observed ; are believed to be the result of change in the heart and lungs.

In the present study in goats cloudy swelling and varying degree of fatty changes were observed in the early part of infection with this disease. Venous congestion and engorgement of the blood vessels with small encapsulated haematomas were seen in few cases.(Fig. No. 24). Inspissated bile in the bile canaliculi was also observed but only in rare cases.

Necrosis of the liver or miliary cellular necrosis described by some of the previous authors (Chanda (1952) and Khera (1955)) were not observed in any of the cases under present investigation.

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Histopathological changes in Lungs:-

Review of literature

Hutyra and Marek in their book on "Special Pathology and Therapeutics of the Diseases of domestic animals" have described interstitial emphysema, catarrhal pneumonia with localised pluritis in lungs of goats affected with rinderpest.

Khanda and Manjrekar(1952) noted emphysema with ruptured alveoli and attendant venous congestion with extensive deposition of pigment, haemorrhage into the alveoli; thickening of the blood vessels, especially the intima and media of the arterioles; and certain amount of oedema in goats affected with this disease. They further observed proliferation of the alveolar epithelium in parts with increase in the inter-alveolar connective tissue; blood pigment also found in the connective tissue mostly. According to Maurer (1955) lungs are only secondarily involved in natural outbreak of this disease in cattle, dilated alveoli, and thickened alveolar walls infilterated with erythrocytes and lymphoid cells were found in the areas of alveolar emphysema. Air spaces in the thickened interlobular septum confirms the impression of great interstitial emphysema. Atelectasis and pneumonic consolidation are also found in some fatal cases.

Khera (1955) discussed the changes in lungs of goats only and he mentioned that in this organ, the interlobular septa were infiltrated with fibrinous exudate, while the alveoli showed lymphocytic infiltration and oedema.

Theiry G (1936) in their experiment, with strain obtained from Sudan 'Capripestic virus ' did not observe any marked broncho-pneumonia of the apical lobes of the lungs in the goats.

Present observations

Lungs were affected invariably in almost all the cases necropsied at the different stages of disease with varying degree of congestion, consolidation, thickening, pleurisy, emphysema and pleuro-pneumonia which confirm the views expressed by most of the previous workers.(Beaton,1930, Hallen, 1871, Edwards, 1927, Cooper, 1931, D'Costa 1933, Chanda 1952, Khera,1955).

The chief histopathological changes observed in the lungs in the present study are, emphysema in the walls of the inter-alveolar septa, in the early stages of infection; Congestion, haemorrhage, and oedema with marked lymphocytic infiltration were very common in almost all the cases at different stages of infection.(Fig.No. 28).

and exudate in bronchioles

Alveolar and interstitial emphysema usually resulted in the rupture of alveoli and its wall. Marked venous congestion with deposition of pigment was also seen in some cases. Thickening of the blood vessels of the lungs especially the intima and media of arterioles were frequently seen.(Fig.33).

Pneumonic changes characterized by red and grey hepatization, infiltration with different types of cells, lymphocytic and polymorphs nuclear leucocytes were common. (Fig.No.29).There was also desquamation of the cells lining the alveoli.

Histopathological changes in other organs :-

Review of literatures

References to the available literatures revealed that histopathological changes in the organs other than mentioned in the preceding pages are seldom met with. Bobberstein (1928) and Marhar (1927) described the main histopathological changes in brain in rinderpest as perivascular lymphocytic infiltration and a moderate increase of glia cells. According to Ahmed Shafiq Bey,(1918) sub-endocardial haemorrhage was found in about 95% of rinderpest cases, they observed. Chanda (1952) mentioned congestion with parenchymatous changes in the secretary cells of the convoluted tubules in the kidney. He further said to have observed varying degrees of degeneration necrosis, lipoidal deposition, albuminous and in some placed hyaline casts in the lumen. Hyaline degeneration was also evident in glomeruli. In several cases he also noticed desquamation of the epithelium, and haemorrhages in the parenchyma as a result of which some of the collecting tubules were found filled with erythrocytes. He observed the changes in a natural outbreak of rinderpest in goats.

Maurer (1955) observed no microscopical changes in the heart of bovines died of rinderpest. In the kidney of these animals, however, he described, evidences of oedema around the pelvis and occasional desquamation of pelvic epithelium. In cases of long duration nephrons are also seemed to be affected. He also observed rarely casts in the collecting tubules, associated with moderate necrotic changes in the distal convoluted tubules.

According to Khera (1955) congestion of brain, meningeal haemorrhages, degeneration of few neurons which he found during the course of his study was probably due to manifestation of toxic effects due to generalised distribution of virus. He also reported to have found cloudy swelling in kidney, in course of his studies of these lesions in bovines.

In goats affected with rinderpest he found only cloudy swelling, slightly increased cellularity of the glomeruli and congestion in the kidney. In the heart, also, muscle fibres showed cloudy swelling which subsided in the latter stages. He did not find any changes in cerebrum while in cerebellum in one case he noticed purkinj's cells undergoing degeneration.

Present findings :-

Heart - Except for the oedema and cloudy swelling in the initial stages of infection, no other significant lesions could be seen.(Fig.No. ~~35~~ 30).

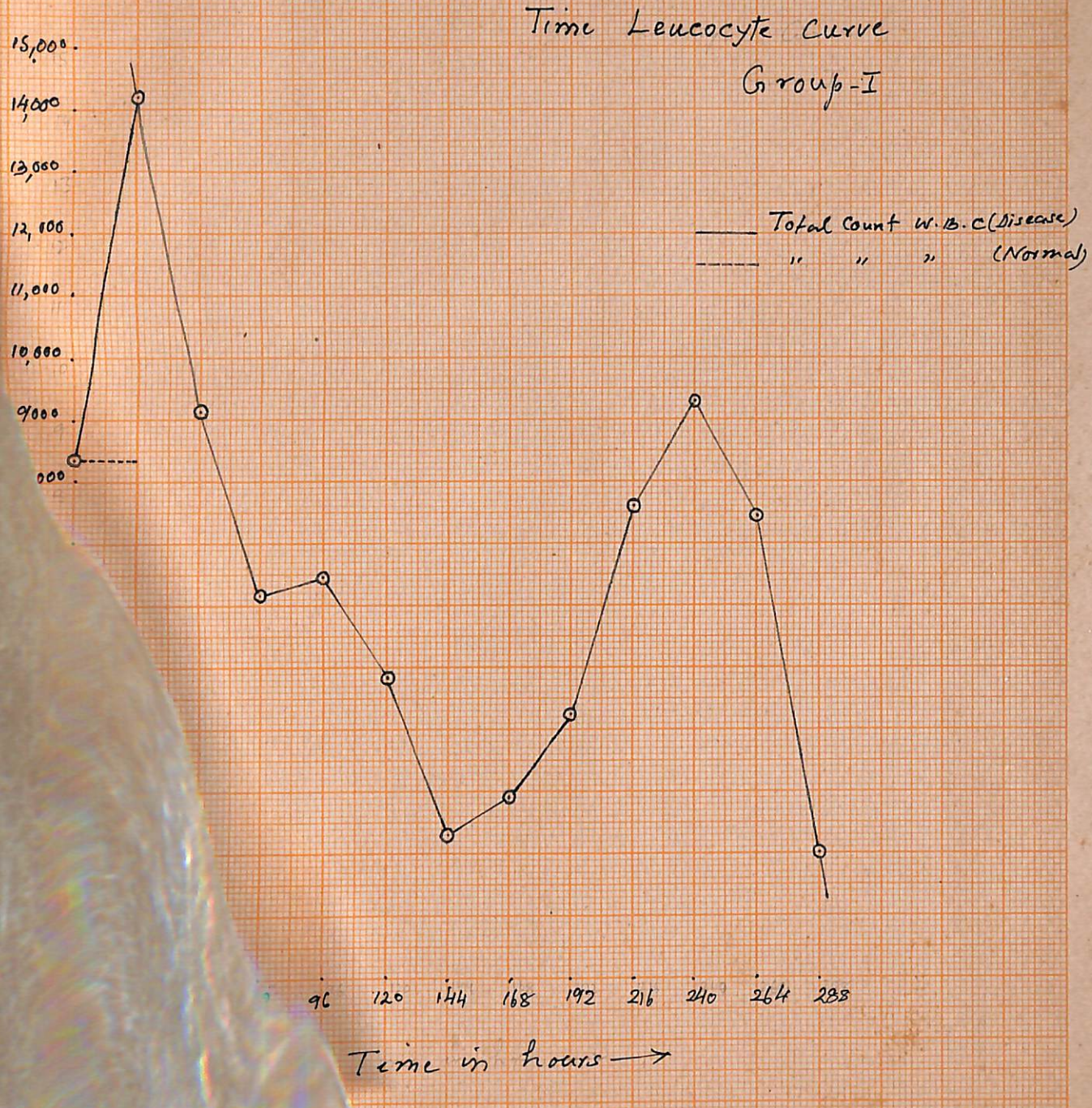
Kidney - Chief microscopic lesions observed in this organ were haemorrhages in the tubules, glomeruli, medulary and cortical region of kidney. Glomeruli in some were highly cellular and contained blood. Desquamative changes in tubules were also seen in few cases with hyaline degeneration in glomeruli. (Fig. No. ~~36~~ 31).

In one case fibrosis in the medullary portion was also noticed .(Fig. No. ~~37~~ 32).

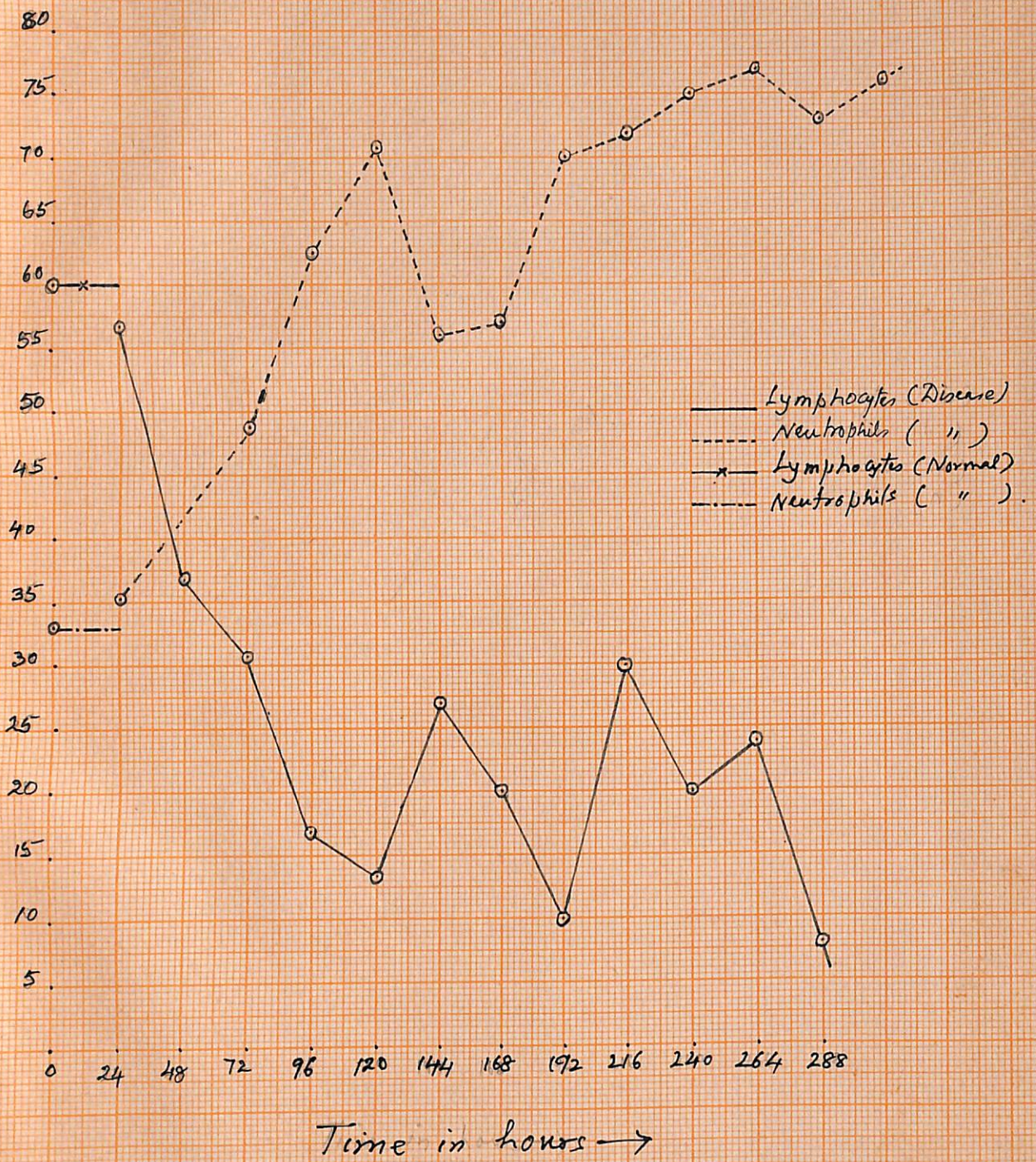
Brain - Sections from cerebrum, cerebellum and hippocampus of the brain tissue were examined. Except for slight congestion, degeneration of few neurons and lymphocytic infiltration in few cases only, no other changes could be noticed.(Fig. No. ~~38~~ 33).

Tonsil - Only slight congestion, hyperplasia and lymphocytic infiltration were noted.

No cellular inclusion bodies could be seen by Mann's methyle blue eosin technique (Kraus, Gerlach and Schweinburg) of staining.


Time Leucocyte Curve
Group-I
15,000
14,000
13,000
12,000
11,000
10,000
9000
000
Total Count W.B.C (Disease)
" " " (Normal)
96
120
144
168
192
216
240
264
288
Time in hours →

Group-I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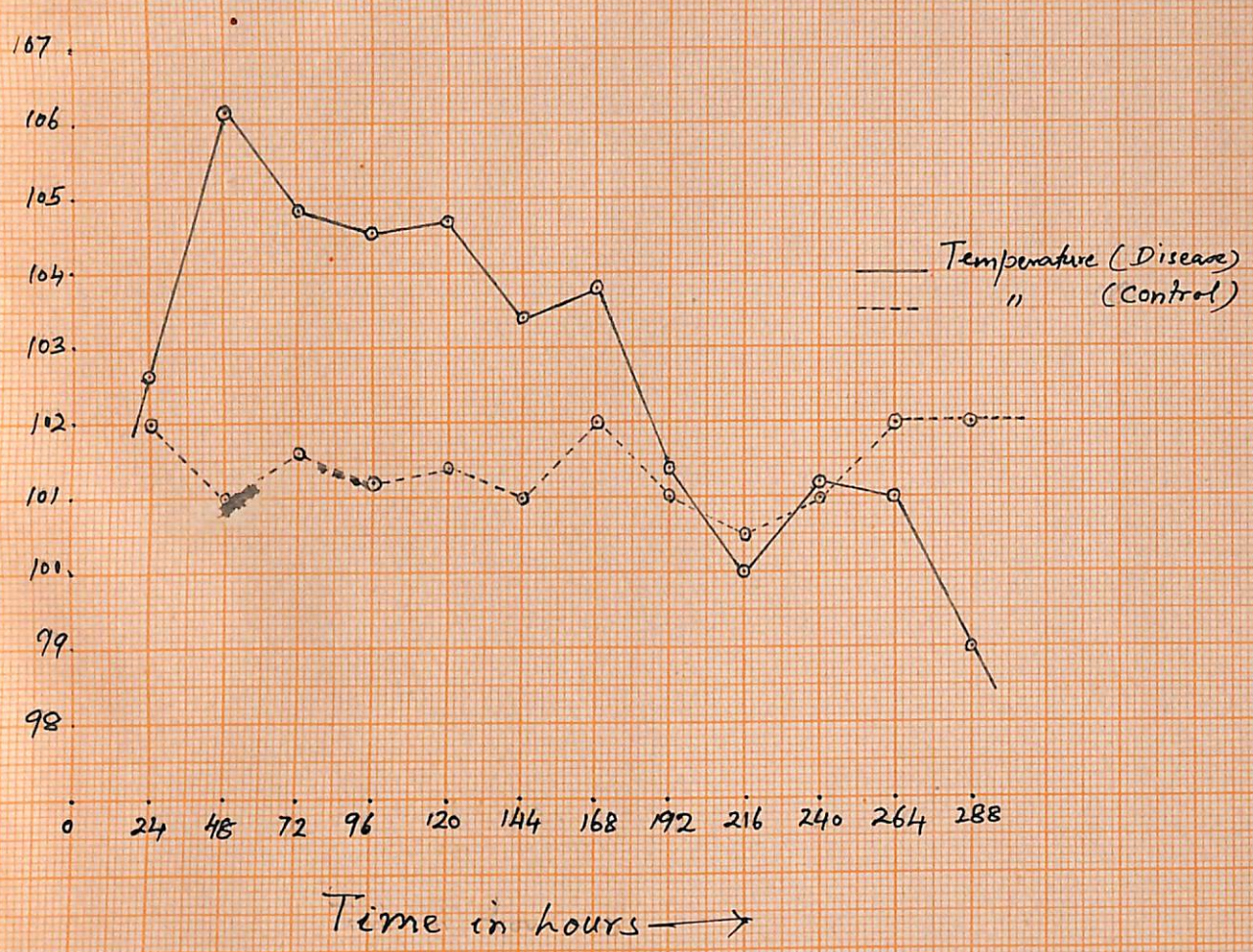
120
144
168
192
216
240
264
288
Lymphocytes (Disease)
Neutrophils (")
Lymphocytes (Normal)
Neutrophils (").
Time in hours →

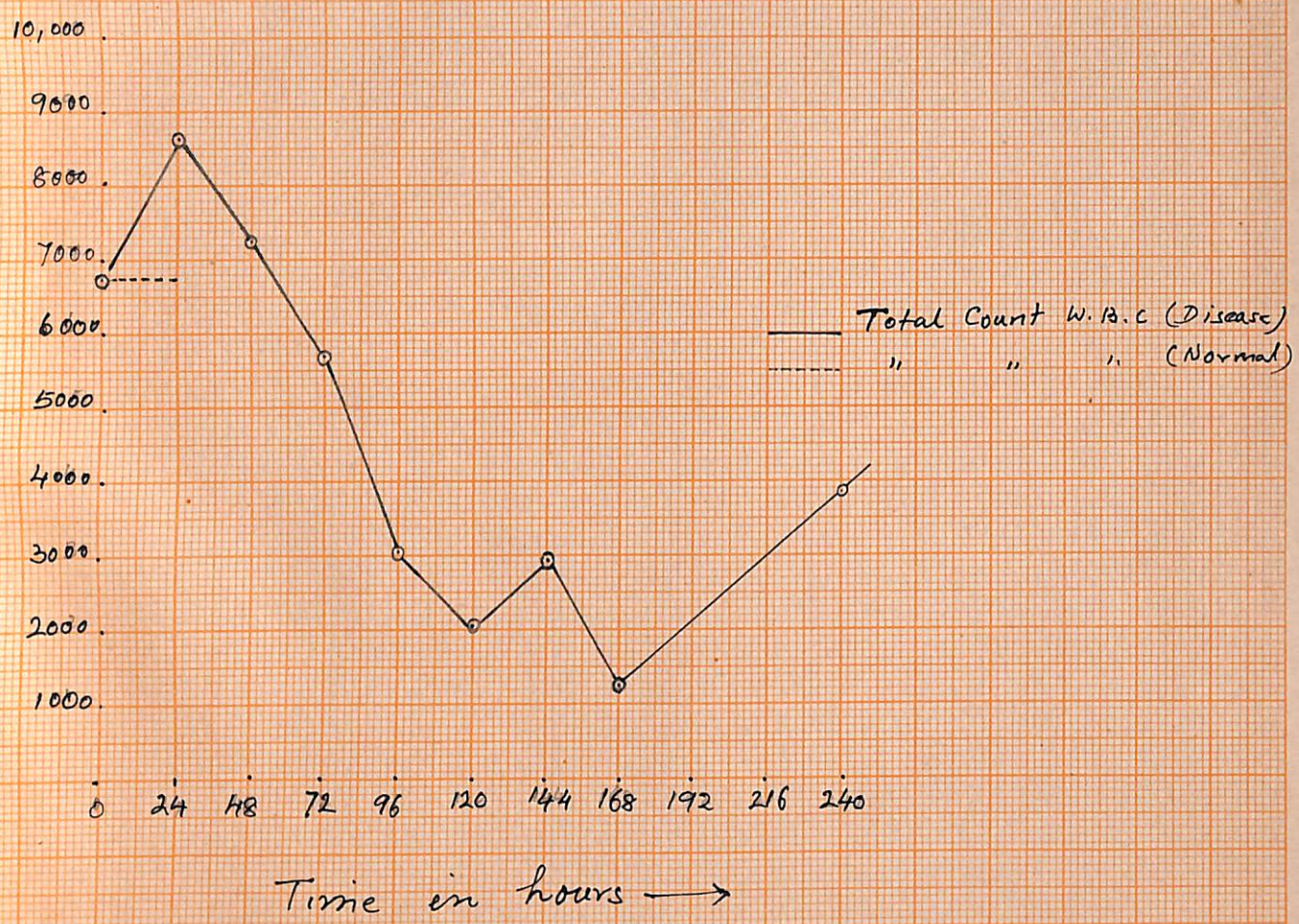
Time Temperature Curve

Group - I.

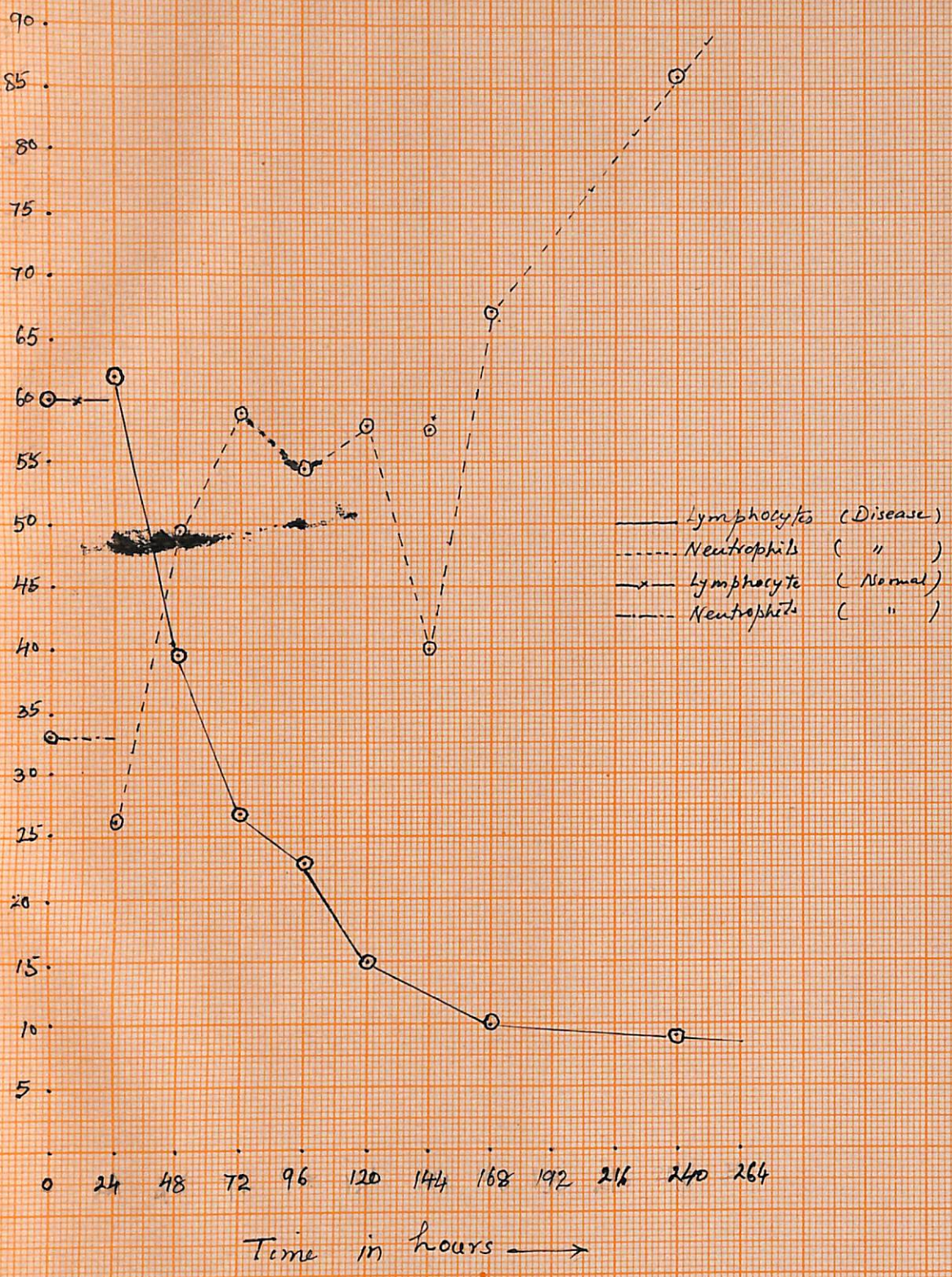


Time Leucocyte Curve

Group – II



Group - II.


90
85
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35
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15
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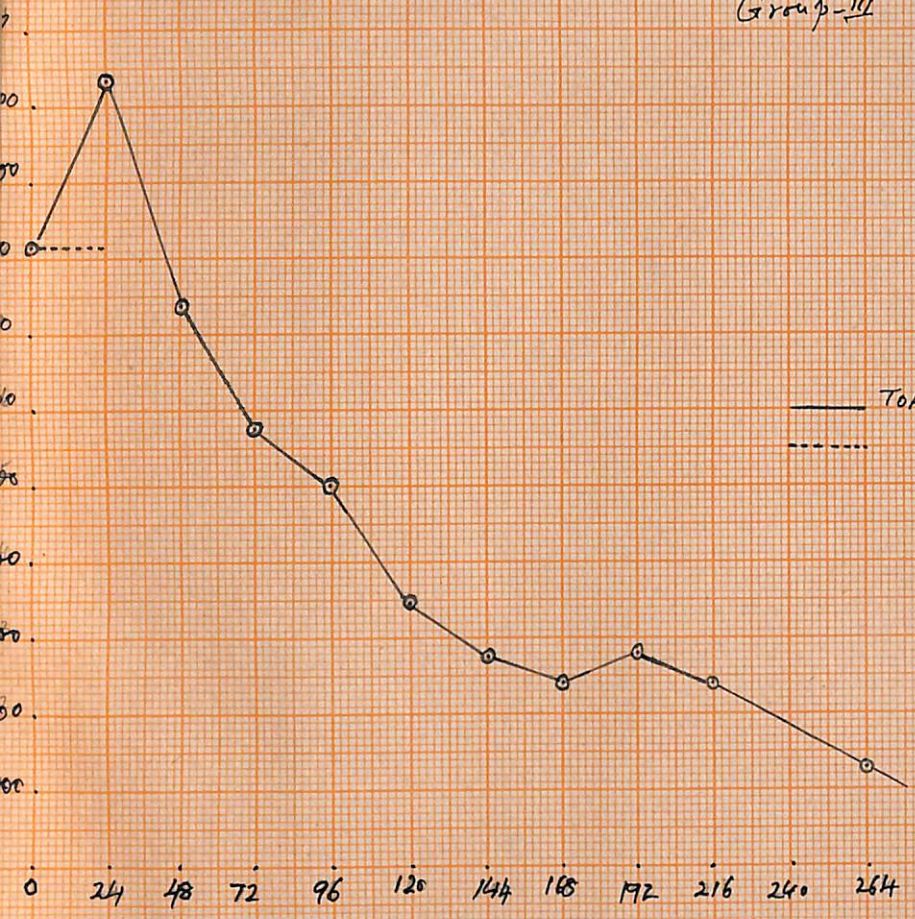
24
48
72
96
120
144
168
192
216
240
264
Time in hours
Lymphocytes (Disease)
Neutrophils (")
Lymphocyte (Normal)
Neutrophils (")

Time Temperature Curve

Group - II.

Time Leucocyte Count Curve

Group-III



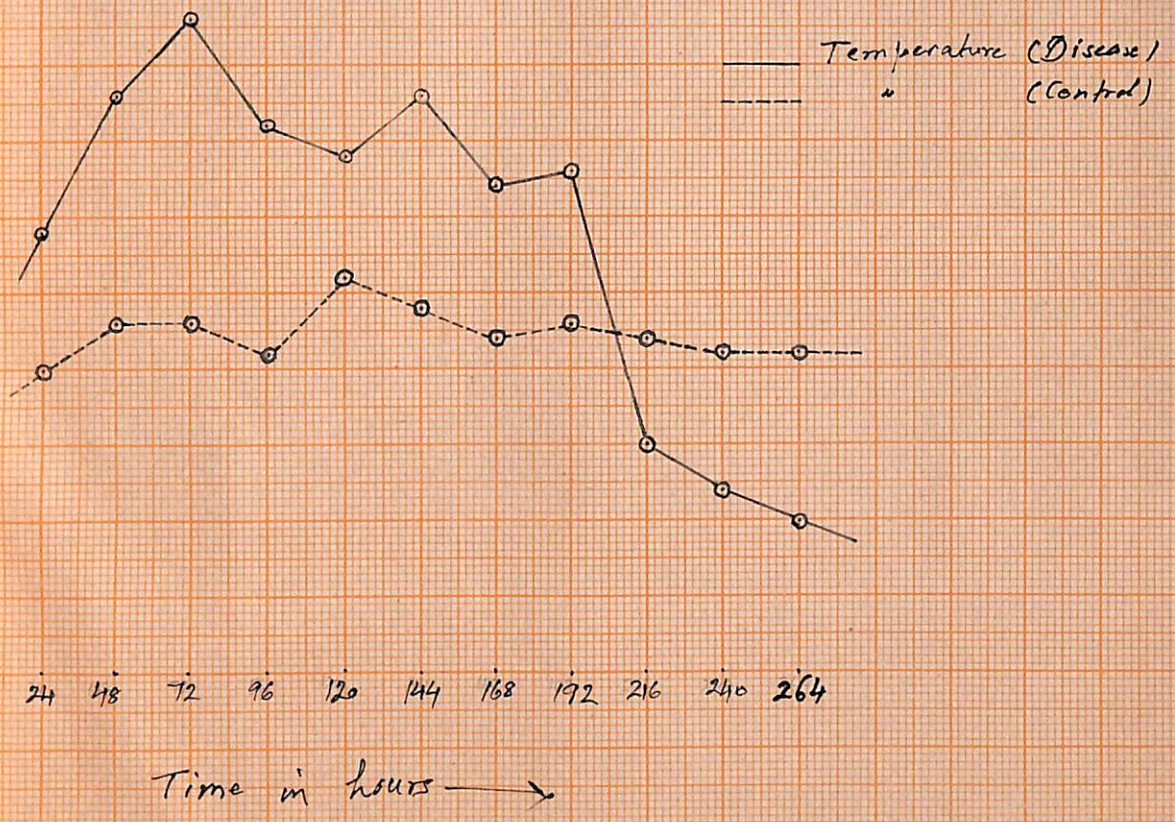
Time in hours →

Group-III


Lymphocytes (Disease)
Neutrophils (")
Lymphocytes (Normal)
Neutrophils (")
75
70
65
60
55
50
45
40
35
30
25
20
15
10
5
0
24
48
72
96
120
144
168
192
216
240
264
Time in hours →

Time Temperature Curve

Group – III.



CHAPTER VII

DISCUSSIONS AND CONCLUSIONS

It is known that clinical symptoms and lesions produced in a virus infection depends upon the strain of virus concerned, its virulence, susceptibility of the animal and its state of health. In the present study the results achieved refer to the infection of the goats of plains with Muktesvar goat adapted virus strain Line "G" of rinderpest which is regarded to be a virulent strain for the caprines.

The symptoms noted in the present study were almost the same as already described by previous workers except that in the present study pneumonia was seen to develop in almost all the fatal cases in which death occured from ten to thirteen days after infection. Lesions in the lungs were noticed in all the necropsies of infected animals done and even as early as on the 3rd day of infection, emphysema and hyperaemia of the lungs were noted. These observations of course, in confirmation with the observations made by D'Costa et.al (1933) and other early workers such as Hallen (1871), Edwards (1927), Beaton (1930), Cooper (1931), Verma (1947), Chanda (1952) and Khera (1955).

However, Theiry (1956) did not notice these changes in caprines as a constant feature which may be due to difference in the strain of the virus which in his case even did not show a marked thermal reaction. He further mentioned that foci of broncho-pneumonia in the apical lobes of lungs was not seen and the animals suffering from broncho-pneumonia

generally showed lesions of congestion and intense inflammation in the tonsil. This fact was explained experimentally by these authors, who concluded that broncho-pneumonia in caprines infected with capripestic virus seemed to be due to the benign septic complications of tonsilitic origin with pasteurellosis or salmonellosis. Therefore, in their opinion broncho-pneumonia was not the specific lesion produced by the capripestic virus, but at the same time, they also imagined the possibility of another virulent virus which may produce these lesions in lungs. Thus it is possible that the strain of the virus used in the present study is more pathogenic to lungs than those strains encountered by Theiry and Dhanda.

The author observed as stated above broncho-pneumonic lesions in all the cases in these experiments. The possibility of secodary infection or complications with other bacterial infections like pasturellosis or salmonellosis were ruled out by cultural examination of the materials of all the fatal cases, which proved negative for any pathogenic bacteria.

Rivers (1948) has said "If action of a virus is not extremely rapid and if the susceptible cells are capable of multiplication, the primary effect of the active agent is stimulation leading to cellular hyperplasia. Following hyperplasia, there is usually destruction or necrosis of cells".

The buccal lesions observed by Dhanda (1952) in that natural outbreak could not be seen in the present experimental

infection which may also be due to the difference in the strain of the virus. Khera (1955) observed that the lesions in the gastro-intestinal tract and spleen were considerably less marked than those observed by the other authors (Dhanda and Manjrekar, 1952). Probably, this may be due to the mild nature of the virus used in his case. In the present study the histopathological lesions described by Dhanda and Manjrekar were found except that of the buccal lesions recorded by them. The results of the present study became more useful due to the correlation of stage to stage infection with histopathological changes seen in fatal cases and also its correlation with the haemotological studies made during pre and post infection periods.

As in all the viral diseases and especially in rinderpest in other animals in which a detailed study has been done; the primary effect of this caprine virus in goats was found to be first stimulation leading to cellular hyperplasia in the initial stage and following this destruction or necrosis of different cells of the body was frequently noted. However, haemorrhage and oedema were the general features observed in most of the organs.

A search was also made for the inclusion bodies in different organs employing Mann's eosin methyle blue staining technique but no such bodies could be demonstrated. Theiry et.al (1955), of course, have mentioned the presence of inclusion bodies in most of the organs especially in the tonsil of bovines due to rinderpest. But so far nobody appear to have definitely confirmed these findings except Khera (1958) in bovines.

Haematological studies made on goats, in the present study, in the pre and post infection of the rinderpest disease may be of great importance. Firstly it gives an aid to diagnosis of the disease in living animals and secondly it also helps to understand the Histopathological changes and phenomenon found on post-mortem examination as to the reaction of virus. In the present study , in most cases for the first one or two days leucocytosis was seen; (vide Table Nos. I, II, III). Soon after which it was followed by leucopenia coinciding generally with the appearance of fever.(Vide Table No. I,II,III and corresponding graphs). Tables I,II,& III also reveal lymphopenia accompanied with a marked neutrophil polynucleosis and eosinopenia as the disease advanced. Leucocytosis for the first 24 hours of infection or so may probably be explained that it was due to the shock of the anxiety reaction of the animal as also observed by Theiry et.al (1935).

In similar virus disease, it is known that aplasia of the bone marrow, and aplasia and necrosis of lymphoid follicle are first to occur. It was also seen (vide Tables I,II, & III) that in fatal cases the fall of leucocytis was more marked and same was the case with lymphocytes.

The present observation of leucopenia after 24 to 48 hrs. of initial stage of infection accompanied with the rise of temperature are in agreement with the observations of previous workers namely, Dinwiddie(1914) Lewish and Shope (1929), Hannen and Anders (1939), Kernkamp (1939) Kerk and Collins (1945)

Robey and Hale (1946) Verma (1947), Maurer (1955) and Theiry et.al (1956), Sharma and Sethuraman (1950) and Chandra Sekharan and Krishnan (1958).

Robey et Hale (1946), however, also noted in addition a diphasic variation of white cell count which was also seen in the present study. This diphasic fluctuation may be due to secondary complications of pneumonia noticed in most cases.

Lymphopenia may perhaps be due to the primary involvement of lymphopoetic system by this virus which was evident in histopathological study of spleen and lymphnodes, the primary producers of these cells. Neutrophilia with a shift to the left was seen and it was more marked as the disease advanced. This maybe due to stimulation of defence mechanism of the body to overcome the infection on the development of the lesions in different organs.

During the initial febrile stages of the disease decrease in the number of erythrocytes, Hb%, M.C.V, M.C.H., M.C.H.C, were also noted. There was also evidence of slight anaemia; which may be due to involvement of the erythropoetic system by this virus. Baldrey (1906), Lewis and Shope (1928), King and Wilson (1910), William and Hammon (1939), Kern and Kemp (1939) and Robey et.Hal(1946) also observed anaemia in viral infections which developed slowly and was less marked than the leucopenia. In the present study the haemoglobin percentage was found to decrease in the initial febrile stage of infection which increased in the terminal stages indicating slight anaemia in the beginning followed by haemoconcentration.

In all the cases under present observation a fall in E.C.V. was marked from - c.mm. in the normal goat to 1 c.mm. after infection which may be due to acute viral infection resulting in anaemia in the initial stage of infection as reported by Kernkamp (1939) in Hog cholera.

Chloride content of the blood was also found to decrease from normal in all the cases especially those in the terminal fatal stage of the disease which may be due to severe fluid loss from the body due to diarrhoea. This was also one of the reasons for increased haemoconcentration noticed in all the fatal cases accompanied with diarrhoea. These findings are in agreement with those of Chandrasekharan and Krishnan (1938) in Ranikhet disease of poultry.

CHAPTER VIII

SUMMARY

Male castrated goats 19 in number collected from the plains of North Bihar with an average weight of 36 lbs. and 1 to 1½ years old were chosen for the present study. Normal haematological study was done in these goats during the pre infection periods taking three reading in each case. They were infected with Mukteswar goat adapted rinderpest virus strain line "W" by subcutaneous route in uniform doses dividing these animals in three batches of six each and learning the remaining one as a control specially for the purpose of histopathological study.

In order to obtain normal blood picture of the goats 57 counts were done on 19 animals. The mean total counts per c.mm. were, White cell 7748 thousand/c.mm., erythrocytes 16.6 million/c.mm., Hb. 11.2 gm.%, E.S.R. 0 c.mm., P.C.V 35.8 sec mm., and chloride value 496.3/hundred c.cm. of blood. The mean percentage various types of cells were, Lymphocytes - 59% , Monocytes- 4.1 %, Neutrophil 33.8 %, Eosinophil 2.3 % and Basophil 0 %. The same haemotological readings were takenin the post infection period at 24 hrs. interval till death in all the batches. One infected goat was left in each batch till death in which haematological readings clinical symptoms and post-mortemed changes and histopathological changes till death were noted. In all the batches, one goat was kept as a control for each of the groups though average blood picture of that group taken in the

preinfection period of these animals served as normal. For histopathological examinations, one goat was sacrificed on every 3rd, 4th, 5th and 6th day of initial infection. The same procedure was followed in all the three batches keeping the dose of the virus and route of administration constant. Similarly the control healthy goat of each batch was sacrificed at the conclusion of the experiment and materials were collected for histopathological examination.

The significant clinical symptoms in infected goats were marked depression, fever, cessation of feeding, nasal and lachrimal discharges, diarrhoea and breathing difficulty producing a peculiar moaning sound in the terminal stages of this disease.

Lung lesions were seen in almost all the affected cases in the form of hyperaemia, congestion, emphysema, lobar and interlobular pneumonia, broncho-pneumonia and pleurisy. Death occured in almost all the fatal cases with advanced pneumonic lesions and corresponding blood changes. Other organs such as liver, kidney, heart and brain did not reveal any marked change except the abomasum, caecum and caecocolic junctions, in which constant and varying degrees of oedema, congestion, haemorrhage, necrosis and desquamation of epithelia were seen.

The histopathological studies in post infection period were made and compared with those of the control goats. Important changes noticed were hyperplasia followed with atrophy and necrosis of most of the lymphatic organs such

as spleen lymphglands and Payer's patches of the intestine and varying degrees of congestion and haemorrhages in all the organs. Necrosis and ulceration of the buccal mucous membrane was, however, not seen in any of the goats. The virus seems to have special affinity for the lymphoid organs which are evident from the histopathological studies and corresponding blood changes observed in these studies.

Search was also made for inclusion bodies in all the lymphoid organs including tonsil using Mann's eosin methyle blue staining technique but with negative results.

Significant changes in haemotological study in post infection period were marked leucopenia followed by temporary leucocytosis in the initial febrile stage, lymphopenia and nutrophilia gradually as the infection advanced. These variations in the blood picture were found to be significant. Eosinopenia and a marked fall in E.S.R. were also seen in most of the infected animals in the advanced stage of the disease.

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