Studies of Pathological Changes in the Kidney of Bovine in Nephritis

THESIS

Submitted to the Faculty of Veterinary Science
Rajendra Agricultural University, Bihar
in Partial Fulfilment of the Requirements
for the degree of
MASTER OF SCIENCE (VETERINARY)

By Lassan Prasad

G. B. V. C. (Pat) B. V. . . & A. H. (M.U.)

Junior Research Fellow, R. A. U. Bihar Post-Graduate Department of Pathology Bihar Veterinary College PATNA

Studies of Pathological Changes in the Kidney of Bovine in Nephritis

THESIS

Submitted to the Faculty of Veterinary Science
Rajendra Agricultural University, Bihar
in Partial Fulfilment of the Requirements
for the degree of
MASTER OF SCIENCE (VETERINARY)

By Lassan Prasad

G. B. V. C. (Pat.) B. V. Sc. & A. H. (M.U.)

Junior Research Fellow, R. A. U. Bihar

Post-Graduate Department of Pathology

Bihar Veterinary College

PATNA

Dr. C.D.N. Singh,
B.V.Sc.& A.H. (Gold Medalist), M.Sc. (Vet),
F.R.C.V.S. (Sweeden)
Professor and Chairman.

Post-Graduate Department of Pathology, Bihar Vetermary College, Patha, Rajendra Agricultural University, Bihar.

Dated, the 25 December, 1974.

This is to certify that the work embodied in this Thesis "STUDIES OF PATHOLOGICAL CHANGES IN THE KIDNEY OF BOVINE IN NEPHRITIS", submitted for the award of Master Degree of Science (Veterinary Pathology) of Rajendra Agricultural University, Bihar is the bonafied research work of Dr. Lallan Prasad and was carried out under my guidance and supervision, and that it incorporates the results of his independent study.

(C.D.N. SINGH)

CERTIFICATE

Certified that the research works incorporated in this Thesis have not been published in part or in full in any other journal.

(L. PRASAD)

The author is extremely grateful to the Vice Chancellor, Rajendra Agricultural University, Bihar for the award of Junior Fellowship in the shape of financial aid, during the course of the present study.

ACKNOWLEDGEMENT

I would like to express my deep gratitude and appreciation to my guide Dr. C.D.N.Singh for his constant help and encouragement to me. I am grateful to him for his excellent guidance in this work. His deep understanding in this area of work was a source of strength to me, which has been manifested in this study.

The author is also indebted to his minor adviser Dr. A.K. Ray, Professor and Chairman, Department of Physiology, Bihar Veterinary College, Patna for his praise worthy suggestions and sincere interests shown by him for this work.

Grateful acknowledgements are also due to Shri J.N.Prasad, Research Officer (Statistic), Animal Husbandry Department, Bihar, Patna for the statistical analysis of the results obtained in the present study.

The author also expresses his gratefulness to Dr. G.J.Jha, Dr. B.K.Sinha and Dr. P.N. Sinha of Pathology Department for their suggestions and constant help during the course of my present work.

I would like to express my gratitude to Dr. R.L.

Kausal, Dean of Faculty for giving invaluable advise in

carrying out this study specially on bovine leptospirosis and

my thanks are also due to Dr. S.P.Singh, Lecturer in Pathology,

Patna Medical College Hospital, for necessary helps whenever required in this field.

My sincere thanks are also due to Dr. K.N. Tiwary, Principal, Bihar Veterinary College, Patna for according me the facilities for the completion of this work.

The author is also grateful to Dr. Y. Prasad,
Director, Animal Husbandry, Bihar, Patna for permitting me to
do M.Sc. (Vet) course.

I express my thanks to Shri H.W.Verma, for his excellent help in the library.

I also with to express my sincere thanks to all the members of the Department of Pathology, Bihar Veterinary College, Patna for their help.

Last but not the least, I am also thankful to my wife Smt. B.B. Shrivastava and all other family members for their patience and encouragement during the period of this work.

(L. PRASAD)

DEDICATED

TO

THE MEMORY

OF

MY MOTHER

CONTENTS

	*				PAGE
INTRODUCTION	•••			•••	1
REVIEW OF LITE	RATURE	**	• •	•••	5
MATERIALS AND	methods	••		•••	36
OBSERVATIONS	•••	••		•••	45
DISCUSSION	X	• •	•	•••	65
SUMMARY	•••	••		•••	78
BI BLI OGRAPHY		••		•••	1 - x
APPENDIX					
	Tables	•••	4		
	Annexure	•••	1	-	
	Histogram	•••	1		
	Graph	• • •	1		
	Map		-1		
	Plates	•••	41		

INTRODUCTION

INTRODUCTION

Human life has always remained in close association with animals. The purpose of such association has always, remained more or less, the same as it exists now. The ways and manners in which the bovine population was reared and maintained in the past, are now quite different in the present system. These distinctions are quite obvious due to ever developing scientific knowledge in the field of animal husbandry and veterinary science.

As a result of population explosion all over the world the people have been faced with acute food crisis. Malnutrition has become a common feature over larger parts of the world population. The people have now started synthetic preparation of protein to face the food scarcity. This vital nutrient comes mostly from milk and meat. Our ancesters, though, they were not having enough scientific knowledge, were fully knowing the advantages of milk. So they gave the name 'Ma' to cows.

balanced feeding, the people have acquired vast knowledge, and they have achieved victory over sufficiently large number of animal diseases, which were havoc among bovine population in past such as Anthrax and Rinderpest etc. Still there are many bovine diseases left to be studied in greater details in order

to face the challenges posed to farm enterprises in different circumstances existing in different parts of the countries.

India is a vast agricultural country where 70% of bovine population of world are present. Still majority of Indian children go without milk. In order to raise the total output of the country it has now become essential to raise the standard of domestication of existing livestock farming though genetical knowledge as well as protection of animals against diseases. The disease control and investigation into the causes of disease production are not simple affairs. The coordinated efforts of specialists of different fields are needed for scientific approach to such problems.

In spite of developed scientific techniques and coordinated efforts of specialists for disease control and treatment of epizootics cattle die due to hepatic and renal damages etc. caused by various other factors.

From public health point of view, it is essential to investigate the renal diseases in details as there are certain diseases which are communicable to man through milk, meat, excretions and secretions (for example, Tuberculosis and Anthrax etc.). Kidneys, being most highly specialised organ is more prone to even mild infections. As such it becomes obligatory on part of the pathologists to attempt systematic and thorough studies of renal diseases.

Kidney is a paired organ having anatomical

complexities. Some thing like one-third of cardiac output of blood passes through kidneys in each circuit. The glomeruli act as filtering bed by eleminating excess of foreign substances circulating in blood. The tubular portion of nephron acts as selective reabsorvers for required amount of glucose, sodium, potassium, calcium, chloride etc. escaping from blood through glomeruli as these are essential for body. The waste products and unwanted materials are allowed to escape as urine. Thus, the internal environmental condition of the body is regulated mostly by this organ.

The toxic agents of blood whether they are vegetable, mineral or microbial in origin, do some harm to renal structures while passing through filtering bed.

In certain specific diseases, such as Anthrax and Tuberculosis etc., the organisms get lodged either in glomeruli or in interlobular blood vessels and thus produce inflammatory condition. The toxic substances such as lead, mercury, arsenic, sulfa drug etc. have affinity for renal tubules, which destroy tubular epithelium. If a part of nephron is damaged the remaining portion usually undergoes atrophy. The infection in glomeruli affects tubules as well as interstitial tissue. The tubular disease in reverse may affect glumeruli. Thus, from above facts it is clear that kidney is an organ which gets frequently involved even in simple and severe infections, and subsequent destruction results in death of animal itself.

The involvement of urinary system causes economic

losses to livestock farming in form of death of productive animals, retardation in growth, as well as reduction in productivity. Thus, the present study on renal diseases has been carried out in diseased bovines on the basis of macroscopic and microscopic lesions in the kidneys.

The present work also envisages utilization of pathological data obtained in the autopsy cases carried out in Bihar Veterinary College, Patna, and also examination of kidneys of killed animals into local abattoirs. The present research has been aimed at to throw some light on various pathological conditions of kidneys which are menace to livestock in this state. The study of gross and microscopic pictures of renal tissue gives the correct picture of renal diseases. These findings may constitute, in future, the basis for further research on various aspects of renal diseases in cattle and buffalces.

Further, this work may also furnish informations on diseases of bovines which may prove conducive to devising of suitable control measures in planning eradicating schemes of renal diseases (for example, Tuberculosis) prevalent in farms in Bihar. The present study may also prove guide to practicing veterinarians to adopt correct measures for control and treatment of renal conditions in bovines.

BEAIEM OF LITERATURE

RAJENDRA ABRICULTURAL UNIVERSITY, M. SC. (VET) THESIS, 1974

REVIEW OF LITERATURE

Perusal of available literature mainly reveals
description of renal diseases under three different categories
viz. degenerative alteration (Atrophy, Parenchymatous, Fatty
etc.) in renal tubules and to a lesser extent glomerular cells,
inflammatory changes in certain parts of the renal structures
(glomeruli, tubules, blood vessels and interstitial tissue) and
physical disturbances, e.g. Infarction and Hydronephrosis etc.
There has been no so far any systematic attempts to bring
together the informations relating to renal diseases in cattle
and buffaloes in Bihar.

DISEASES OF URINARY SYSTEM (KIDNEYS).

Nephrosis :

Muller (1904) employed the term "Nephrosis" to denote the degenerative or inflammatory changes in the kidneys.

Nieberle (1922) (as quoted by Udall, 1954) classified all the degenerative changes right from cloudy swelling to necrosis of the kidneys resulting from metallic poisons such as mercury etc. and summarized them under "Nephrosis".

Bloom (1939) described nephrosis as non-inflammatory condition of the kidneys. He called mild necrotizing and

severe necrotizing nephrosis under acute nephrosis and amyloidosis under chronic nephrosis.

ottosen (1949) reported a calf of one month old which suffered from diarrhoea and was treated with Sulphathia-zole twice daily for 5 days. On 10th day the animal was killed. The renal lesions were characterised by development of Calculi composed of Sulphathiazole. The tubules showed development of nephrosis.

French (1950) observed glomerulonephrosis in toxic oliguria, anuria, and lower nephron nephrosis.

Welt and Peters (1951) stated that sulphanilamides, carbontetrachloride and mercury salts produce toxic effects in renal structures and cause lower nephron nephrosis in kidneys. They suggested that factors responsible for tubular injuries are some toxic agents for renal epithelium, anoxia of renal tissue and increased reabsorption of water from tubules.

Amyloidosis :

Hajarre (1933) reported amyloidosis in 64 cases out of which 50% were reported in cattle.

Anderson (1936) reported that frequent occurrence of "Large white kidneys" in cattle were due to amyloid degeneration accompanied by other changes. He reported 36 cases

in which amyloid deposits were found in kidneys apart from heart, lungs and digestive tract. Histological pictures revealed that amyloid deposits were well marked in glomeruli, vasafferens and vasrecta in medullary substances. Besides this, the kidneys showed parenchymatous changes. He was also successful in producing artificial general amyloidosis by injecting repeated doses of fowl albumin.

Detailed studies were conducted by Van Vloten

(1936) in 48 cases of "large white kidneys" including 4 cases
in calves. Histological pictures of large white kidneys showed
lesions of hyaline and amyloid degeneration of glomeruli and
tubules accompanied to some extent by interstitial nephritis.

From the calf kidneys, he formed an opinion that interstitial
nephritis occurred primarily before the amyloidosis took place.

According to Smith and Jones (1957) amyloidosis in kidneys takes its first appearance in the glomeruli, which it eventually replaces. As the condition progresses, the invading substance also makes its appearance in the outer medulla. Amyloidosis always occurs in immediate vicinity of small blood vessels. Amyloidosis is not so frequent in domesticated animals except horses and dogs.

Scheehan and Davis (1960) has reported that when proximal convoluted tubules undergo moderate ischemic damage, but not of lethal one, development of hyaline droplets does take place in their cytoplasm during subsequent in flow of the

blood. These droplets might be of varying sizes. The small droplets of size of red blood cells could hardly be differentiated from red blood cells by Haematoxylin and Eosin stain except that different sizes of droplets were seen scattered among the hyaline droplets.

Mandelli and Cosico (1964) has reported deposition of amyloid materials at different sites in the kidney's, glome-rular arteries (in muscular coat), out sides the tubules, and around Bowman's capsules in the histochemical analysis of sponteneous amyloid nephrosis in cattle.

Grunder and Trautwein (1965) studied symptoms of renal amyloidosis in 15 pregnant and post-parturient cows. He observed profuse chronic diarrhoea, protein rich urine. In a few cases he found erythropenia, neutrophilia and oedema of throat and chest. On post mortem examination the kidneys were light colour, granular surface and enlarged to a considerable extent. Microscopically there was glomerular amyloidosis, and in some cases there was interstitial amyloidosis. Other noticeable changes were hyaline cylinders within tubules, fat vacules in epithelium and in Henle's loop.

Pigment in kidneys

Bos (1936) reported abnormal pigmentation in the kidneys and adrenal glands in bovines. Brownish pigments were detected in epithelial cells of the convoluted tubules. But

narrow loops of Henle and glomeruli showed little or complete absence of pigment granules. A thin layer of mucous containing pigment was found covering the renal pelvis. Microchemical reactions showed the pigment to be melanin and was present in connective tissue of the cortical substance, and in the cells of medulla. He suggested that some relation might be existing between the activities of renal glands and the formation of skin pigment.

Corsico (1953 and 1955) described the deposition of Kanthine a brownish red pigment in both kidneys of an apparently healthy calf. He again reported (1955) the presence of kanthine in the collecting and papillary tubules of two slaughtered calves.

orlop (1956) studied 2.3% bovine kidneys with dark brown discolouration of the cortex. The pigment was present in the form of granules of varying sizes in the convoluted tubules. The pigment was proved to be lipofuchsin. He also found this pigment in renal and mesentric lymph nodes.

Jubb and Kennedy (1963) cited congenital prophyriaa brownish pigment affecting the renal cortex of cattle and
swine. Pigments were present in tubular epithelium and
interstitial tissue which were detected in urine. The urine
and tissue were fluresced blue green in the presence of ultra
violet light.

Hypoplasia and hypertrophy :

Iaszlo (1941) described single case of renal hypoplasia in an ox. On microscopical examination, he concluded that this condition was due to incomplete development of glomeruli and tubules, and replacement of parenchymatous tissue by a cellular connective tissue or by fat cells.

Moderate hypertrophy of blood vessels were observed.

In view of Runnels et al. (1960), hypoplasia of kidneys is rare in domesticated animals. Sometimes, it does take place as congenital anomalies.

Hypertrophy is more frequent in all domesticated animals as result of renal, glomerular or tubular injury. The remaining healthy renal cells become more active and enlarged to compansate the loss of renal epithelium. When one kidney is lost due to ligation of renal arteries, or obstruction in one of the ureters, or as a result of bacterial obstruction, the other kidney undergoes hypertrophy to compansate the function of lost one. Histologically, epithelial cells of nephron get enlarged and become more active. Sometimes, the cells get two times enlarged rendering kidney as a whole double than its normal shape.

Infarcts :

Smith and Jones (1957) stated that infarcts in

kidneys were a frequent phenomena in animals. Most often, the infarcts in kidneys were usually of anaemic types and represented a picture of one or more sharply outlined wedge shaped pale or red areas depending upon the case with apex at the point of obstruction and the base at the capsule. As the lesion grew older, there was healing with disappearance of parenchymatous tissue except a few scars of white fibrous tissue in a few glomeruli.

Mora and Del. Giudice (1958) described a brief sketch on renal infarcts in cattle with obscure aetiology.

Circulatory disturbances and diseases of blood vessels:

Smith and Jones (1957) described active hyperaemia as a feature of acute inflammation, and passive congestion as a part of generalised passive congestion. The congestion was promin at in medulla due to the presence of venulae rectae running in group.

Csiszar (1943) studied petechiaein 82 slaughtered and 28 healthy calves. Tests for bacterial and toxic aetiology were found negative. The petechia were also observed in calves having traumatic injury to the spleen.

According to him petechia often encountered in salmonella infection.

Di Domizio (1948) reported 24 cattle fed mainly on cereal, showed petechia in kidneys.

Abramov (1962) examined 17 calves died from toxic dyspepsia. All such calves showed haemorrages in renal cortex. But he could not isolate salmonella or coliform bacteria from the lesions.

Hydatids :

In India hydatids appear to have been reported 1st by Gaiger (1910) in Punjab. It was also later reported by Gaiger (1915), Aggrawala (1925), Pillai (1928), Mudaliar (1931), Ayyar (1942), Mocho (1945) and Mudaliar and Aiway (1947).

Krause (1934) reported that out 954 cattle and buffalo slaughtered, 86% of the population were having echynococcus infestation and 49 cattle and 1 buffalo had alveolar echynococcosis.

Sami (1938) reported 88% of cows and buffalo were infected with hydatids.

Hemmann (1934) encountered an extra ordinary infectation of echynococcus cyst in every organ including most of the lymph glands in a cow.

Maqsood (1944) found a pigeon's egg sized sterile echynococcus cyst in the cortex of the right kidney of a cow.

Faust (1949) reported that echynococcus cysts were surrounded by endothelial cells, giant cells, eosinophils and well developed granulation tissue.

Glomerulonephritis :

Nieberle (1922) (cited by Udall, 1954) recorded 3 cases of nephritis in old cows in the abattoir. On histopathological examination he inferred that so called chronic parenchymatous nephritis or "large white kidney" was genuine glomerulonephritis having resemblance with sub-acute or chronic glomerulonephritis of man.

Wester (1935) stated that glomerulonephritis in bovines was always bilateral and was due to bacterial toxin.

was first used by Muller (1904) to denote the degenerative process in kidneys in which inflammation played a doubtful identity. The author stated that in nephritis there was functional interference with blood circulation through the glomeruli. In gomerulonephritis this damage affected all the glomeruli of both kidneys. In nephrosis the blood supply was normal but the glomerular capillaries became abnormally permeable to proteins.

Langham and Hallman (1941) studied 236 cases of nephritis in all species of domesticated animals and recorded 7 cases of glomerulonephritis. Out of 7 cases, incidence in bovine constituted 1.87%. Glomeruli were the sites of primary lesions, characterised by thickening of the basement membrane, increased amount of connective tissue and hyalinization. The tubules showed secondary changes characterized by cloudy

swelling, atrophy and gradual replacement as a result of intensive productive tissue changes.

Udall (1954) stated that acute diffuse glomerulonephritis in herbivora was rare and was practically always associated with some severe toxic or infectious diseases.

Smith and Jones (1957) grouped glomerulonephritis under toxic nephritis and considered as rare in animals under natural condition.

Leons (1957) observed a case of haemorrhagic glomerulonephritis in a cow where foreign body was involved in spleen and reticulum. He was of the opinion that kidney lesions were due to allergic condition set up by the septic focus.

Wettimumy (1963) studied and classified the incidence of 178 cases of nephritis as follows: -

Glomerulonephritis - 11%
Interstitial nephritis - 79%
Pyelonephritis - 10%
Embolic or pyaemic nephritis - 5%.

Carrara and Galli (1966) stated that out of 551 normal slaughtered calves of age group between 2 to 3 months, 35 had numerous petechiae and occasionally infarcts in the cortex of kidneys. 35 calves also had some purulent chronic broncho pneumonia. Histological examination of kidneys showed glomerulonephritis. 10 calves were also examined bacteriologically. Pasteurella multocida was isolated from 4,

C. pyogenes from 2. Thus, he concluded that there is no relationship between bacterial species isolated and the renal lesions and he suggested that renal lesions were allergic in origin.

Interstitial nephritis :

Focal interstitial nephritis was described first by Smith (1925) in bovines. He investigated interstitial nephritis in calves which were kept deprived of their normal cholestrum and he was of the opinion that <u>B. coli</u> might had become virulent in calves, devoid of cholestrum feeding for 24 to 36 hours.

Br. abortus 1st calf was injected intravenously by virulent culture of Br. abortus immediately after birth. This calf died on sixth day after showing febrile reaction. On histopathological examination, foci of cellular infiltration of the interstitial tissue along with endothelial cells, monocytes and lymphocytes were found in the kidneys. The foci were found near the arterioles. The glomeruli did not show any change. The second calf was injected intramuscularly after 5 weeks after birth with the same strain of Br. abortus and it was killed after two and half months later. The kidneys showed the same lesions as seen in 1st calf. The only noticeable differences were that they were more chronic in forms. Grossly

the lesions appeared like white nodules just below the capsule and in the depth of the kidneys.

Magnusson (1934) tried to confirm the Lubke's (loc. cit) statement from the heart blood taken from 15 affected calves to demonstrate <u>Br. abortus</u> organisms in the lesions but agglutination test did not reveal positive result in blood.

Wester (1935) discussed various forms of chronic nephritis in bovines and stated that in case of primary diffused interstitial nephritis of both kidneys, the organs were small, hard with increased interstitial tissue.

Moore and Hallman (1936) obtained "white spotted kidneys" in many calves by feeding vitamin A difficient diet. The condition was also associated with pneumonia and scour. The authors were of the view that this condition might be related to avitaminosis A.

Moore et al. (1936) examined 35 calves with previous history of low plasma Magnesium and found nephritis in
many of the animals along with other lesions. In kidneys,
the lesions consisted of extensive productive tissue changes
with secondary atrophy of the renal parenchyma. Whether these
changes were due to low magnesium content of blood or due to
some other factors could not be determined.

Moor et al. (1938) obtained renal lesions in calves kept on low magnesium content; besides lesions in

other organs. 12 out of 21 showed marked proliferation of fibroblasts and ultimately fibrosis of the interstitial tissue with atrophy and necrosis of renal parenchyma. There were varying degree of focal infiltration of lymphocytes in intestitial spaces. Many occurred in the areas showing fibroblastic reaction. There was marked necrosis of tubules in 7 cases. Other changes were fibrosis of Bowman's capsule, proliferation of the glomerular capillaries, endothelium, and deposition of debris in the subcapsular spaces.

Rurtze (1936) made cultural examination of 14
pairs of "white spotted kidneys" but he failed to demonstrate
the presence of <u>Br. abortus</u>. Out of 300 calves slaughtered
only 8% gave positive reaction to agglutination test but <u>Br.</u>
abortus were neither found in blood, nor in urine, nor in
urinary lymph gland nor in spleen of positive or negative
calves.

Metzger et al. (1937) described sub-acute and chronic interstitial nephritis in cows which exhibited albuminuria.

Wiidik (1937) studied 90 cases of "white spotted kidney" in calves from 3 to 6 weeks of age group. He examined kidneys, liver, spleen with regional lymph nodes for meat poisoning organisms and for <u>Br. abortus</u> but all gave negative results. Thus, he concluded that the "white spotted kidney" were not due, bacterial infection of the kidneys but were of

toxic origin.

Heinen (1938) examined "white spotted kidney" from nine calves. Either in culture or by inoculation <u>Br.abortus</u> was not isolated from any case.

Hoppe (1938) stated that interstitial non-purulent nephritis might be found in the foetuses of Brucella infected cows. In the early stage there was increase in number and size of capillary endothelial cells with a diffused infiltration of lymphocytes and histocytes in the intestitial spaces. In the advance stage similar infiltration appeared in interstitial spaces of deeper tissue. More centres of infiltration appeared with increase in size as the disease progressed.

Thorp et al. (1942) noted gross lesions in 42 out of 25 dairy calves kept on low vitamin A diet. The changes were more clear and well marked in the most deficient animals. Microscopic examination showed degeneration of proximal convoluted tubular epithelium with inflammatory changes in the interstatial tissue. Metaplasia were observed in renal calyces.

Runnels (1954) described "white spotted kidney" as a special form of focal interstitial nephritis and stated that the condition was most common in farm animals.

Hofmann (1955) investigated focal interstitial nephritis in 29 calves of age group 4 to 6 weeks old. There appeared to be acute, a sub-acute, and chronic forms.

Histologically, the acute form constituted minute whitish foci with enlarged lymph nodes and there was accumulation of leucocytes and lymphocytes. In sub-acute cases, foci were larger and fibroblasts were present. In chronic cases, lesions were longer, and massive connective tissue was observed.

Actiology could not be determined.

Tsiroyannis (1957) observed "white spotted kidney" in 1376, out of 45000 calves slaughtered in Belgium. Histopathological and bacteriological examinations were conducted over 250 pairs of kidneys. He thought that lesions were caused by an allergin or allergins of unknown origin.

Smith and Jones (1963) described that "small white granular contracted kidney were due to scar formation out of white fibrous connective tissue; thus rendering the surface of the kidneys irregular and granular. The appearance of white fibrous connective tissue was a clear evidence of chronic proliferative inflammatory process in glomerular or tubular or interstitial nephritis.

Butura et al. (1960) examined kidneys from 568
buffaloes and he found evidence of nephritis in 13% of examined
buffaloes. Repeated examinations of the animals and of urine
over 8 months, confirmed the presence of chronic nephritis.
Thus, it was concluded by author that exposure to cold played
a major role in the causation of nephritis.

Mohanty (1961) found that out of 232 kidneys, 18

showed sub-acute interstitial nephritis in which 6 showed greyish white foci on the kidney surface, and 71 kidneys showed chronic interstitial nephritis.

Leptospirosis :

Weil (1886) was the first to consider leptospirosis as a separate entity.

Awrorow (1941) gave a detailed account of pathological changes in 40 animals which died of leptospirosis. He found centrilobular necrosis and interlobular round cell infiltration of liver and degenerative changes in the kidneys and myocardium in case of acute and sub-acute cases. Histological sections of the liver, kidneys, lungs, heart and mesentric lymph nodes showed the presence of leptospira.

Jungherr (1944) described the autopsy findings in 3 cows died in acute phase of leptospirosis and he found extensive necrosis of hepatic cells with haemorrhage in the liver of one cow. The kidneys of another cow showed large area of tubular necrosis with lymphocytic interstitial inflammation. Focal tubular hypertrophy was found in the kidneys of the third cow. Hemosiderin was seen in the spleen.

Leptospira were demonstrated in liver, kidneys and mesentric lymph nodes, but the organisms could not be isolated.

Marsh (1945) reported leptospirosis in 25 calves during 3 months of his study. Calves died of an acute form

characterized by haemoglobinuria and leptospirae in tissue.

Matheson (1946) studied pathological changes in acute and chronic forms of natural bovine leptospirosis in Texas. He found focal necrosis in liver in acute fatal forms around the cerebral vein. Extensive albuminous degeneration, necrosis and pigmentation of the renal tubular epithelium were prominent changes. Hemosiderin was present in spleen, muscular haemorrhage, necrosis and ulcer formation were constant findings in the abomasum.

Baker and Little (1948) reported an infectious disease of dairy calves. There was no abortion, icterus. Haemoglobunuria was rare. The infectious agent was probably leptospira which were transmitted to two guineapigs, rabbits, mice, and cattle. This produced an interstitial nephritis which persisted in the kidneys for long time even after disappearance from blood stream. Thus, the author suggested that natural infection might be due to a droplet infection or by insect vectors. Antibodies against leptospira were found in the serum of experimental animals.

Akcay and Pamukcu (1950) described three acute and fatal cases of leptospirosis in calves. Pathological study of sections of kidneys treated with Levaditi's method revealed leptospira within the tubules, necrotic changes, infiltration in the inter tubular tissue and haemoglobin casts in the tubules. There was parenchymatous degeneration of the liver.

Reinhard (1951) - Innoculated leptospira organisms obtained from natural cases of bovine leptospirosis into 6 calves. Cultural, haematological and clinical studies were made in these calves. He found fever, leptospiraemia, leptospiruria, neutropenia, and lymphopenia and a transitory anaemia. In all cases kidneys showed gross and microscopic lesions of focal interstitial nephritis.

He further studied in 1952 over bovine leptospirosis and found at autopsy the kidneys were "white spotted" or mottled. Histological changes in acute forms were thickening of Bowman's capsule and congestion of glomerular tufts. The tubules at times showed cloudy swelling or necrosis, distended by granular debris, protein casts, leucocytic casts and occasionally blood pigment casts. Haemosiderin was found in the tubular cells in haemoglobinuric cases. Diffuse and focal infiltration of round cells (lymphocytes, plasma cells and macrophages) and a small number of neutrophils were throughout the stroma of the renal cortex. Leptospira were demonstrated in the tissue by silver impregnation technique. They were usually found in the tubular lumen and occasionally in the renal stroma. The constant histological findings in the chronic leptospirosis were those of chronic focal interstitial nephritis. Large masses of lymphoid round cells were infiltrating in the medulla. Sometimes interstitial fibrosis might also be present.

VanDer Hoeden (1953) reported an outbreak of

L. grippotyphosa infection in a herd of 95 dairy cattle. 10 out of 12 died due to acute interstitial nephritis.

Hadlow and Stoenner (1955) described the histological findings in several organs of 15 Hereford cows infected with L. pomona. The important features in the kidneys were widespread chronic focal interstitial neparitis and renal tubular proliferation was common finding. The latter was associated with focal defects in the tubular basement membrane or with its almost complete dissolution and the proliferating tubular cells formed giant cells in the larger area of interstitial exudate. Haemosidrosis was the constant finding in the spleen. Leptospira were demonstrated in the sections of kidney from 6 of the 15 cows.

Kemens and Szky (1955) demonstrated leptospira in the kidneys of 10 calves killed 12 to 3 months after natural infection by silver impregnation method.

VanDer Hoeden (1955) reported 3 outbreaks of L.

canicola in cattle and Israel. Several jackals proved to be
carrier. They excreted the organisms in the urine and autopsy
revealed interstitial nephritis. He suggested that in Israel
besides dogs; Jackals were important natural reservoir of L.

canicola which was hazard to man and cattle.

Ristic et al. (1957) studied clinical, bacteriological, serological, hematological and gross pathological response of 9 calves, experimentally infected with L. sejroe. Apart from other clinical symptoms leptospiraemia persisted for 1 to 4 days. Leptospira were isolated from the kidneys of one calf and was demonstrated in the silver stained kidneys of two other calves. The major pathological lesions were interstitial nephritis, degeneration of the epithelial lining of Bowman's capsules and convoluted tubules of the kidneys and focal necrosis of the liver. They concluded that <u>L. sejroe</u> might be an additional aetiological agent of bovine leptospirosis.

Burdin et al. (1958) recorded outbreak of leptospirosis in cattle, sheep and goats of kenya. A high incidence
of mild cases showing renal lesions as the sole manifestation
of the disease was found. The kidneys were very much swollen
and congested when the course of the disease was rapid. Light
coloured petechiae were observed in kidneys of the animals
which had been ill for long time. These abnormal areas were
found to extend through the cortex and some time into the
medulla and in some cases only one kidney was affected.

Seibold et al. (1961) gave microscopic evidence of active or previous infection for leptospira in the kidneys of 292 (86%) of 338 cattle received at the Plum island. Focal acute, sub-acute and chronic lesions were noted. In acute forms, necrosis of tubular epithelium in renal cortex were seen. Sub-acute stages were characterised by proliferation of undifferentiated epithelial lining cells and by progressive

interstitial lymphocytic infiltration. Chronic lesions were characterized by atrophy of affected tubules with thickening of the basement membrane. Leptospira were found in acute and early sub-acute lesions, but with the development of lymphocytic infiltration, they disappeared and were nerver found in the advanced sub-acute or chronic lesions. Leptospira were seen in certain small tubules at the cortico-medullary junction only, where they produced no visible histological lesions and in undamaged tubules of 57 cattle at the cortico medullary junction.

Venkataraman and Jagarnnathan (1961) reported the 1st outbreak of leptospirosis among cattle with deaths in Madras which continued for 3 months. The symptoms were suggestive of haemorrhagic septicaemia or plant poisoning but the histopathological examination showed the presence of leptospira in kidneys. The isolation of leptospira in pure culture was not successful probably because of delay in conducting post mortem examination and collection of material.

Retzlaff (1967) made a review from serological survey for bovine leptospirosis in Germany (1963-1966) which revealed that an average of 3.45% of cattle examined had a titre of 1:400 or high, but the author could not find leptospira on histological examination of Levaditi stained sections of kidneys from 116 healthy cattle. He described interstitial nephritis and cysts in the kidneys of 12 and 7 cattle, respectively but aetiology was not determined.

Suppurative nephritis :

Bruckmulier (1869) described abscesses in the kidneys of an ox associated with dilatation of the pelvis.

The kidneys were enlarged and there was thickening of capsule which was adherent. The surface of the kidneys showed small abscesses surrounded by an inflammed zone. The kidney parenchyma was congested and juicy.

Pflug (1876) suspected micro-organism to be the cause of renal abscess in bovine.

Ernst (1905) described small elongated abscesses around the vessels in the renal medulla. He found masses of gram-positive organisms scattered irregularly in the abscesses. The organisms were also seen collected into clumps and cylinders in the convoluted tubules and in the loops of Henle. The papillary tubules appeared free from organism but their mucous membrane were infiltrated with leucocytes. Collecting tubules were necrosed and crammed with bacteria, debris, and leucocytes.

Christiansen (1919-20) (cited by Udall 1954)
described 16 cases of abscesses in kidneys of calves. From
15 of these, B. coli were isolated.

Jowett (1925) recorded a case of pyaemic nephritis in a cow. Grossly multiple foci of pin head size to millet seed were present on the kidney surface. Lesions were yellow to yellowish white with semisolid consistency. Gram's stain

revealed the presence of small bacillary and coccoid type of organisms. Pure culture of C. pyogenes were isolated.

Again he in 1928 found another case in which apart from other gross appearance stated above the capsule was adherent. Histologically he found that the structures of kidney lesions were composed of cells that were mainly monoleucocytes. Gram's stain showed the presence of large number of small bacilli and coccoid organisms. Pure culture of C. pyogenes were isolated. Thus, he concluded that the infection had occurred by lymphatic routes.

Fincher (1933) cited by Udall (1959) observed large abscess in the right kidney of a two months old calf which replaced the entire kidney.

Harichen et al. (1966) studied microscopic changes in focal purulent nephritis in 8 chinchilla. The lesions were characterised by accumulation of leucocytes in the proximal and distal convoluted tubules and degenerative changes of the epithelial cells of tubules. The aetiology and pathology of focal and purulent nephritis in chinchillas and focal interestitial nephritis (white spotted kidney) in calves were discussed by the author.

Pyelonephritis:

Siedamgrotzky (1875) found bacteria in renal pelvis.

Dammann (1877) observed the presence of bacteria in

urinary tubules in Bacillary pyelonephritis.

Gillot (1888) found lesions of pyelonephritis in a cow but he was unable to ascertain the cause of this condition.

Guillebeau (1888) studied the pathogenicity of bacillary pyelonephritis in cattle and found slender non-motile bacilli in the renal abscesses.

Hess (1888) described symptoms and lesions of pyelonephritis and found that the disease occurred independent of puerperal infection.

Boyd (1918 to 1927) reported bovine pyelonephritis
for the 1st time in United States and he worked from 1918 to
1927. He was of the opinion that in cow pyelonephritis very
often accompanied with the inflammation of ureters and bladder.
These conditions were usually as a result of bacterial invasion.
The disease might run acute or chronic forms. Diagnoses
depended upon characteristic bacilli in urinary sediment,
presence of albumin, red and white blood cells in urine.

Boyd and Bishop (1937) considered that the process of pyelonephritis had an urogenous origin and seldom was haematogenous in origin. This got support by involvement of bladder and ureters. C. renal were constantly present along with secondary invaders such as streptococci. Diagnoses was made on a clinical, bacteriological, and physical examination of the urine, bladder, ureters, and kidneys.

Bemis (1926) also found pyelonephritis, a very common condition in cows.

Hutyra and Marek (1926) studied 20 cases of pyelonephritis. He found that many cases originated due to infection in early life involving urino genital tract of calves.
The infection ascended gradually and reached the upper urethra
and bladder. He suggested that natural infection did not take
place through blood stream as the disease could not be produced
by intravenous injection.

Puttaswamy (1932) recorded a case of pyelonephritis and cystitis with a big abscess in broad ligament of uterus with lesions of traumatic pericarditis.

Ventura (1932) laid some emphysis upon the possibilities of bovine pyelonephritis occurring without the evidence of disease in other organs. He recorded 3 such cases of pyelonephritis in bovine.

Wester (1935) described different types of chronic nephritis in bovines as (i) chronic embolic nephritis in which kidneys were emlarged and showed infarction, (ii) chronic glomerulo-nephritis (Nephritis Fibrocystica) which was always bilateral and attributed to bacterial toxins, (iii) primary diffuse interstitial nephritis characterised by a small and hard kidneys with an increased interstitial tissue, (iv) secondary atrophied kidney; which was unilateral with compansatory hypertrophy of the other side, (v) Tuberculosis of

the kidney and (vi) pyelonephritis which was in the form of cysto-ureto pyelonephritis of the cows. He reported two types of pyelonephritis. The first was an ascending form developing from the cystitis and was caused by <u>C. renale</u> whereas the other type was of haematogenous inflammation of the kidneys in which ureteritis, cystitis and pyelitis were seen.

Palmer (1938) studied the course of pyelonephritis in 3 herds and found that spread of disease is very slow. He gave detailed description of one case of pyelonephritis. Grampositive dephtheroid and Eberthilla pyogenes (E. pyogenes) were isolated.

Thorp et al. (1943) studied 6 cases of bovine pyelonephritis. They found an increase in creatinin, urea and non-protein nitrogen in blood of 2 cases. These two animals showed haematuria, and albuminuria. The urine contained C. renale in 5 cases. From the culture of 6th one, the organisms isolated belonged to genus corynebacterium and it was a diphtheroid. The kidneys, ureters and bladder showed degenerative changes both macro and microscopically.

Feenstra and Thorp (1946) found that the location of bacteria varied in different cases of bovine pyelonephritis. In milder forms micro-organisms were present in the cellular debris in renal calyces, ureters and bladder mucosa. In severe cases, the organisms might be found in foci scattered in kidneys, in the tubules and in the necrosed epithelial cells of tubules. In the characteristic lesions, dephtheroids were

present in cellular debris which was flanked by a zone of necrosis, and outside that zone, the parenchyma showed chronic inflammatory changes.

Matheson (1946) reported 7 cases of nephritis including some cases of pyelonephritis.

Morse and Roberts (1948) reported a case of pyelonephritis in a cow. Urine showed the presence of <u>C</u>. renale
and <u>B</u>. coli. <u>C</u>. renale were recovered from left kidney and
<u>B</u>. coli was predominant in bladder. They concluded <u>C</u>. renale
was primary cause while <u>B</u>. coli was simple secondary invader.

Peenstra et al. (1949) reported 11 cases of bovine pyelonephritis and indicated the effects of penicillin in different forms of cases. Haematuria was important clinical symptom. Presence of C. renale in urine was not diagnostic as it was also found in the urine of healthy animals.

Biswal et al. (1953) studied strain of diphtheroid organisms isolated from pyelonephritis in cattle by Feenstra in 1945. These organisms differed from <u>C</u>. renale in some of the biochemical properties and these also produced alpha haemolysis.

Dhanda and Das (1954) were 1st to record pyelonephritis in cattle in India caused by diphtheroid. On autopsy
abscesses were found in the pelvis and cortical parenchyama
of both kidneys. Ureters, bladder and its wall and mucous
membrane were thickened. They isolated diphtheroid organisms

from urine and kidneys of affected cows and grouped it as C. renale.

Nandi (1955) stated that cystitis and pyelonephritis of cattle were caused by <u>C</u>. renale in conjunction with other factors which caused irritation and damage to the kidney structures during its excretory phase from the body. He stated that those factors were derived from various plants and he named 25 such plants.

Smreck (1957) isolated <u>C</u>. <u>renale</u> from 27 out of 33 cattle suffering from pyelonephritis.

Kwiatkowski (1957) stated two clinical cases of pyelonephritis in cattle. He was of the opinion that treatment had no effect in advance stage.

Rajulu (1956) recorded a case of naturally occurring pyelonephritis due to <u>C</u>. <u>pyogenes</u>.

Kume et al. (1959) isolated diphtheroid bacilli from outbreak of pyelonephritis in cattle. The organisms were classed in group A (identical with <u>C. renale</u>) and group B (not identical with <u>C. renale</u>).

Classification of renal disorders :

Classification of renal disorders in both man and animals is a complex problem. Different yard sticks have been adopted by several authors.

According to Anderson (1957), no single method of classification of renal diseases is easily applicable. The system of classification followed by him is based on combined consideration of the portion of the kidney primarly affected and the types of involvement. Classification of renal diseases as adopted by him is partly presented below:

- (i) Glomerular disease :
 - glomerulonephritis
 - diffuse acute
 - sub-acute
 - chronic
 - focal.
- (ii) Vascular disease : Nephrosclerosis
 - Atherosclerotic
 - Arteriosclerotic

periarteritis nodosa

infarcts of the kidney.

- (iii) Tubular disease : Nephrosis or tubular nephritis.
 - lipoid nephrosis
 - toxic nephrosis
 - chemical nephrosis.
- (iv) Interstitial tissue disease : Interstitial nephritis
 - acute diffuse
 - chronic diffuse
 - focal suppurative

pyelonephritis
tuberculosis
leukemia infiltration.

- (v) Obstructive diseas of the kidney : Hydronephrosis.
- (vi) Metabolic renal disease :
 - hyperparathyroid renal disease
 - hypervitaminosis D
 - renal calculi
 - uric acid infacts
 - renal lesions in gout.
- (vii) Congenital malformation and anomalies :
 - agenesis
 - hypoplasia
 - fusion
 - ectopia
 - cysts.

(viii) Tumors :

- benine
 - hamartoma
 - adenoma
 - fibroma
 - leiomyoma
 - lipoma
- Malignant hypernephroma and carcinoma
 - Wilms' tumor (adenomyosarcoma)

- carcinoma of the pelvis
- metastatic

Herbut (1959) considered renal diseases in man under five categories viz. congenital anomalies (aplasia and cystic diseases etc.), degeneration (parenchymatous, atrophic and fatty etc.), inflammations (diffuse glomerulonephritis and pyelonephritis etc.), physical disturbances (infarction and hydronephrosis) and tumors.

Jubb and Kennedy (1963) have adopted classification of renal diseases according to the structures primarly involved. This system envisages to determine which structural component of kidney is primarly injured. Disorders of the kidneys have been classified by them into four categories viz. diseases of glomeruli, diseases of tubules, lesions of intestitial tissue, and miscellaneous renal diseases.

Smith and Jones (1970) have described various renal disorders after classifying them into different categories, based on the aetiological and gross pathological features. Considering aetiological system of classification, nephritis has been divided into infectious and non-infectious types. Kidneys with white streaks or spots in the cortex, the large pale kidneys and small white granular contracted kidneys are the types of nephritis based on gross lesions in the kidneys.

MATERIALS AND METHODS

MATERIALS AND METHODS

Basic materials concerned in the studies of renal disorders in bovines were collected from the kidneys of cattle and buffaloes of all age groups and sexes having deviations from normal. The sources of materials in present study comprised of - (1) BVC materials, (11) SH materials and (111) GCF materials.

BVC materials :

These materials constituted post mortem cases sent for autopsy in the Department of Pathology of Bihar Veterinary College Hospital, Patna. Out of 109 cases, 19 cases belonged to this source. Survey was made from the autopsy records of Bihar Veterinary College Hospital from 1949-50 till 1973-74 and pathological data were collected for statistical study.

SH materials:

The 70 abnormal kidneys from buffaloe calves of age varying from 1 year to 3 years were collected from local slaughter houses for gross as well as histopathological examinations. Out of 70 suspected cases of nephritis, 19 cases did not reveal any significant histopathological changes in

kidneys except a few changes of minor nature.

BVC = Bihar Veterinary College.

SH = Slaughter house.

GCF materials :

Out of 109 cases examined 20 cases comprised of animals died of various diseases, but with lesions in the kidneys, and these were sent to the Department of Pathology, Bihar Veterinary College Hospital, Patna for autopsy. These materials also include 10 samples of urine and blood from the suspected cases of nephritis in GCF to detect changes in the urine and blood.

Histopathological method :

Out of 250 specimens of kidneys all together 109 cases revealed gross as well as histopathological changes. However, 19 specimens had no any significant renal disorders except a mild congestion. Thus, only 109 kidneys sample were directed for histopathological processes. The kidney samples were taken from every part of kidneys for histopathological examination.

The gross examinations were conducted and noted separately in order to correlate the gross changes with histopathological findings of renal diseases.

The samples were fixed in 10% natural formaline and routine staining practice of Harris Haematoxylin and Eosin, Van Gieson stains and Levaditi's procedures were adopted for

GCF = Government Cattle Farm.

histopathological examination. Special stain such as PAS, Acid fast, Gram-negative and Gram-positive were used to confirm diagnosis as and when necessary.

The tissue were washed overnight in tap water, and dehydrated in ascending grades of alcohol. Cleared the alcohol by two changes in xylene, and xylene by 3 changes in melting paraffin at 55° to 65°C. Finally the tissue were embedded in paraffin. The sections having thickness of 5 microns were cut by hand driven microtome.

The following staining techniques were adopted : -

Haematoxilin and Eosine (Lillie, 1954) :

- 1. Deparaffinize the sections and hydrated to water.
- Stain the sections by Harris' hematoxylin for 15 minutes.
- 3. Rinse in tap water.
- 4. Differentiate in acid alcohol, a few dips.
- 5. Wash in tap water for a brief period.
- Dip in ammonia water until the sections are bright blue.
- 7. Wash in running tap water for 10 minutes.
- 8. Stain with eosin for 15 seconds to 1 minute depending upon the age of eosin.
- 9. Dehydrate in 95% and absolute alcohols until excess eosin is removed, two changes of 2 minutes each.

- 10. Absolute alcohol, two changes of 3 minutes each.
- 11. Kylene, two changes of 2 minutes each.
- 12. Dried the sections and mounted in DPK.
 Results

Nuclei - blue

Cytoplasm - various shades of pink.

Connective tissue :

Van Gieson's picrofuchsin method (Lillie, 1954) :

- 1. Deparaffinise the sections and bring them to water.
- 2. Wash in water.
- 3. Stain for 5 minutes in haematoxylin (Weigert's acid iron chloride).
- 4. Wash in water.
- 5. Stain 5 minutes in picrofuchsin mixture.
- 6. Dehydrate and differentiate with two changes each of 95% and 100% alcohol.
- 7. Clean with a mixture of 100% alcohol and xylene followed by two changes of xylene. Mount in DPX.

Results

Collagen - red, Muscle and Cornified epithelium - yellow, Nuclei - blue to black.

Spirochetes :

Levaditi-Manovelian Method:

- 1. Rinse specimen in tap water, after fixation.
- 2. Let stand in 95% alcohol for 24 hours.
- 3. Transfer to distilled water and leave until the tissue sinks to the bottom of the container.
- 4. Place in freshly prepared silver nitrate solution and keep in 37°C in the dark for 3 to 5 days, changing the solution three times.
- 5. Rinse in distilled water.
- 6. Reducing solution (Pyrogallic acid 4.0 gm,
 Formalin, 37-40% 5.0 ml, Distilled water 100.0 ml) at room temperature, in the dark for
 24 to 72 hours.
- 7. Rinse in distilled water.
- 8. Dehydrate in 80% alcohol, 95% alcohol, and absolute alcohol, two changes, 30 minutes each.
- Clear in xylene for two changes, 1 hour each and infiltrate with two changes of paraffin
 45 minutes each.
- 10. Embed in paraffin.
- 11. Cut sections at 5 microns and mount on slides.
- 12. When dry, deparaffinize with xylene, three changes.
- 13. Mount with DPK.

Results :

Spirochetes - black, background - yellow to light brown (Mallory, F.B., 1961).

PAS positive factors - Periodic acid-schiff method (Culling, 1957):

- 1. Bring sections to water.
- 2. Oxidise for 5-10 minutes in 1% aqueous periodic acid.
- Wash in running water for 5 minutes and rinse in distilled water.
- 4. Treat with schiff reagent 10-30 minutes.
- 5. Transfer directly to the first sulphite rinse one minute second sulphite rinse for 2 minutes transfer to third sulphite rinse for 2 minutes.
- 6. Wash for 10 minutes in water.
- 7. Counter stain with haematoxylin.
- 8. Dehydrate, clear and mount in DPX.

Results :

PAS positive substance bright red, Nucleiblue, other tissue constituents yellow.

Gram Weigert technique for gram + bacteria (Culling, 1957).

- 1. Bring section to water.
- 2. Stain with 2.5% aqueous phloxine.
- Wash in water and stain with Gram's crystal violate for three minutes, blot with filter paper.
- 4. Treat with lugol's iodine for one minute, blot with filter paper.
- 5. Decolourise in aniline oil and then treat with equal parts of aniline and xylene.

6. Clean in xylene and mount in DPX.

Results :

Gram + bacteria blue; other tissue constituents red.

Gram negative bacteria : Wolbach's Giemsa Variant (Lillie, 1954).

- 1. Bring sections to distilled water.
- 2. Stain one hour in Giemsa's stain (Methyl alcohol 1.25 cc., 0.5% sodium carbonate solution 0.1 cc. (2 drops), Giemsa's stain 1 cc., distilled water 40 cc).
- 3. Pour off and replace with two further changes of same mixture during the first hour and leave in the third change overnight.
- 4. Differentiate in 95% alcohol containing a few drops of 10% colophonium alcohol.
- 5. Dehydrate with 100% alcohol, clear and mount in DPX.

Results :

Gram negative bacteria stains intense reddish purple.

Acid fast bacteria :

Ziehl-Neelsen's method (Culling, 1957).

1. Bring the sections to water.

- 2. Stain in hot carbol fuchsin for 10 minutes.
- 3. Wash in water to remove excess stain.
- 4. Differentiate in 3% acid alcohol.
- 5. Wash in water.
- 6. Counterstain lightly in 0.1% methylene blue for half a minute.
- 7. Wash in water.
- 8. Dehydrate, clear and mount in DPX.

Results :

Acid fast bacilli - red, nuclei blue, other tissue constituents - pale blue.

Bacteriological examination :

For bacteriological studies, pieces of kidneys were collected from the dead body of the animals by taking rigid aseptic precautions. Post mortem examination was carried out immediately after the death of animals. Smears were stained with the acid fast and Gram's stain for microscopic examination. Cultural examinations were done wherever necessary.

Haematological examination :

Haematological examination in 10 normal and 10 other Tharparkar cattle affected with albuminuria was also

performed following routine haematological techniques.

经长备

带

OBSERVATIONS

OBSERVATIONS

Incidence of Bovine Renal Diseases in BVCH, GCF and other Waterials:

The materials under present study were divided into three main groups viz. (1) Materials belonging to Tharparkar cattle of Government Cattle Farm, Patna, (2) Materials collected from Bihar Veterinary College Hospital, Patna, autopsy cases, and (3) Materials collected from local abattoirs. The BVCH materials also included statistical data collected from autopsy register since last 25 years in order to locate the renal diseases in GCF, Patna as well as animals killed in abattoirs.

Table 1. Distribution of renal diseases and other conditions in 1609 dead bovines.

No. autop- sied	-1	(with renal) (diseases		Irenal Idise-
1609	26 (including tuber- culous kidneys in 3 bovines).	276	1307	19

1609 bovines were autopsied during the year 1949-50 to 1973-74, out of which it was observed that the renal lesions were noticed in 302 cases. Taking together they constituted 19% of total bovine autopsied (all BVCH, GCF, and SH materials included).

Table 2. Incidence of renal diseases according to age group and sex in Tharparkar herd in Government Cattle Farm, Patna.

Total no. of cases	Age group of animals	Male (a)	Sex [Female [(b)	No. of cases with renal lesions (a+b)
302	From birth to 6 months	. 12	36	48
	From 6 months to 1 year	r. 22	28	50
	From 1 year to 3 years	. 21	38	59
	From 3 years to 8 year	s. 22	55	77
	From 8 years and above	. 18	50	68

The above table furnishes the incidence of renal diseases according to age group and sex amongst Tharparkar animals maintained in Government Cattle Farms. To test whether the age group is independent of the sex in respect of occurrence of renal diseases, the following Chi-squire test was applied as given by Panse Sukhtme (1961).

Chi-squire
$$=\frac{1}{n_1 n_2} \sum \frac{(an_2 - bn_1)^2}{a+b}$$
of freedom)

Where a and b stand for number of cases with renal lesions according to sex n_1 and n_2 corresponding total for male, and female.

$$n_1$$
 = sum of 12 + 22 + + 18 = 95
 n_2 = sum of 36 + 28 + + 50 + 207
 n_1 · n_2 = 95 x 207 = 19665

$$\sum \frac{(an_2 - bn_1)^2}{a + b}$$

$$= \frac{(12x207 - 36x95)^2}{48} + \frac{(22x207 - 28x95)^2}{50} + \dots + \frac{(18x207 - 50x95)^2}{68}$$

= 278858.827.

Chi-squire
$$=\frac{1}{n_1 \times n_2}$$
 $\sum \frac{(an_2 - bn_1)^2}{a+b}$ of independence)

The calculated Chi-square with 4 degree of freedom is found to be 14.180 where as the tabulated Chi-square value for 4 degree of freedom at 5% point of significance is 9.488 which is less than the calculated value. Thus, it is concluded that occurrence of renal diseases is independent in various age group to sex.

Table 3. Incidence of renal diseases in BVCH,GCF and SH materials.

S1.		sied	Total no.of renal dise- (ases (ai.1)		ai.1xpi
1	1949-54	272(R ₁)	51(a1.1)	51 272 (p1)	$\frac{51 \times 51}{272} = 9.562$
2	1954-59	218(R ₂)	41(a2.1)	41 218 (p2)	$\frac{41\times41}{218} = 7.711$
3	1959-64	377(R ₃)	58(a3.1)	58 (p3)	$\frac{58 \times 58}{377} = 8.923$
4	1964-69	291 (R ₄)	62(a4.1)	62 291 (p4)	$\frac{62 \times 62}{291} = 13.209$
5	1969-74	451 (R ₅)	92(a5.1)	92 451 (p5)	92×92 451 =18.767

According to Gauden (1962), the following formula was applied in the above 5x2 table. Whether the occurrence of renal diseases is independent of the period (drought or flood days) of autopsy.

Chi-squire with 4 degree of independence =
$$\sum_{i=1}^{5}$$
 (ai.1 p1) - $C_1\bar{p}$

Where ai.1 = number of renal diseases found in the ith period (i = 1 to 5).

C₁ = Total number of renal diseases in all the five period.

G = Total number of animals autopsied.

$$\bar{p} = \frac{c_1}{G}$$

$$\bar{q} = 1-\bar{p}$$

$$\sum_{i=1}^{5} Ri = R1 + R2 + R5$$

$$\sum_{i=1}^{5} ai.1 = a1.1 + a2.1 + + a5.1$$

$$\bar{p} = \frac{C_1}{G} = \frac{302}{1609} = 0.189$$

$$\bar{q} = 1 - \bar{p} = 1 - 0.189 = 0.811$$

$$\sum_{i=1}^{5} ai.1xpi = 9.562+7.711++18.767=58.172$$

Chi-square with 4 degree of freedom

$$= \frac{\sum_{i=1}^{\infty} (ai.1xpi) - C_1 \bar{p}}{\bar{p}}$$

$$= \frac{58.172 - 302x0.189}{0.189x0.811}$$

$$= \frac{58.172 - 57.078}{0.153279} = \frac{1.094000}{0.153279} = 7.1308$$

The 5% point of Chi-square for 4 degree of freedom is 9.488. The calculated value of Chi-square for 4 degree of freedom is found to be 7.131, which is less than the tabulated value. Thus, the occurrence of renal diseases in different period is dependent upon them.

Table 4. Composition of renal diseases in 109 specimens of bovine kidneys collected from different sources.

Pathological diagnosis	BVCH materials		GCF materials		SH materials		Tota	llPer lcent
1	3	3	Under 13	Over	Under	rlover 13 Syears		i
Cyst.	•	-	-	1	-	-	1	0.9
Nephrosis.	3	4	2	3	3	6	21	19.3
Glomerulo- nephritis.	-	-	1	2	2	4	5	4.6
Interstitial nephritis.	3	4	2	1 .	38	10	58	53.2
Tuberculous nephritis.	-	-	-	3	-	-	3	2.7
Pyelonephritis		-		4	-	-	4	3.7
Miscellaneous.	3	2	-	1	7	4	17	15.6
Bovine spirochetosis.	-	-	1	1	-	-	2	5.2

Table 4 shows the frequency of different renal

diseases in 109 pairs of kidneys collected from BVCH, GCF, Patna, and local SH.

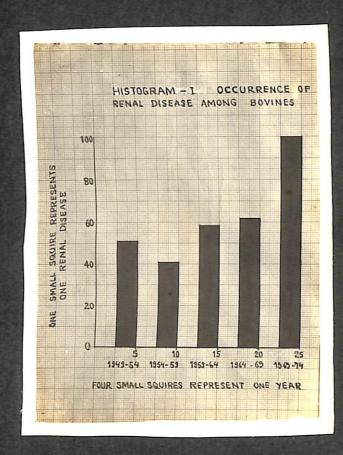
Occurrence of interstitial nephritis is highest in the present investigations (53.2%).

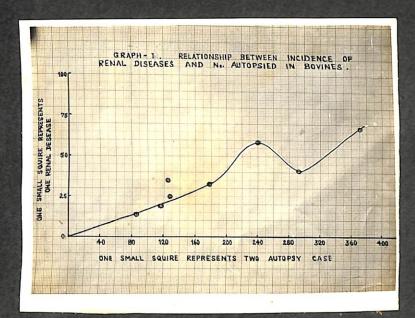
38 pairs of kidneys were treated separately through silver impregnation procedures to identify leptospira but organisms not dissimilar from spirochetes were detected in 2 cases.

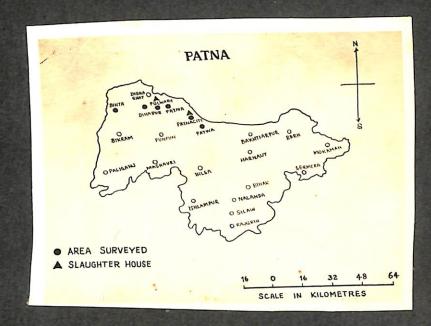
Blood values of normal Tharparkar cattle of GCF, Patna and cattle affected with albuminuria. Annexure 1.

					5	4		
Lymphocy tes	57.0	70.07	62.7	+ 4.6	40.0	72.0	61.1	+ 2.55
Honocytes	2.0	7.0	3.4	+0.52	1.0	5.0	2.8	+0.37
phils	0.0	1.0	0.4	+0.16	nel	MI	N11	N41
Globin phils phils phils phils ml ml	2.0	0.9	3.9	+0.3	3.0	0.6	5.0	+0.85
phils	24.0	36.0	29.6	+4.4	27.0	90.0	31.1	+ 2.24
globin gm/100	10.5	13.2	11.6	₹ 0.28	6.0	0.6	7.9	+0.27
thousand/	7230.0	9840.0	8420.0	+ 736.1	4500.0	9200.0	0.0089	+ 0.46
millions/ thousand/globin cmm cmm gm/100	80.	7.8	9.9	+0.19	2.3	0.9	4.4	+0.34
No. of animals	10 normal. Min.	Max.	Mean.	S.	10 affected.Min.	Max.	Mean.	S.B.
No	0				0			

Annexure 1 shows that there was marked decrease in the normal value of haemoglobin percentage, R.B.C. and W.B.C. count per cam and slight rise in percentage of neutrophils and eosinophils.







The pertinent data were derived from all the materials (EVCH, GCF and SH materials). The incidence is demonstrated in histograme 1, and graph 1. The majority of renal diseases occurred in the period between 1949-54 and 1969-74. The different areas or districts covered in this present study were badly hit by drought and flood. During these years influence of drought and flood was noticed in enhancing the number of renal lesions in cattle and buffaloes.

Pathological changes in renal diseases of bovines :

Cysts: - 1 case of cystic kidney was noticed out of 109 kidneys examined in cattle and buffaloes.

Macroscopic changes: - A few lobes of the kidneys were enlarged. The capsule was adherent to the renal cortex. The kidneys were firm in consistency. The cortical portion was yellowish grey, granular and hard to cut. The surface was granular and uneven on feel. The cortico medullary junction was somewhat oblitrated. There were several cysts of varying sizes with thin transparent walls, having diameters of about inch to the inches. But, some of the cysts were almost replacing lobules of the kidneys (Fig. 1) and some were bulding from the surface while others were burried in the cortex. There was presence of serus watery fluid in the serus cavities.

Microscopic changes: - The cysts were found to occupy large areas of kidney's parenchyma. Calcification was found in the basement membrane of tubules and interstitial tissue. There was increase in fibrous tissue elements around the tubules. The interstitium was infiltrated by lymphocytes, plasma cells, neutrophils and interstitial tissue. In some cases increase of fibrous tissue was also observed. The tubular lumen was over distended to form large empty spaces of various shapes (Fig. 2). Bowman's capsular epithelium, and tubular epithelium snowed degenerative changes at places.

Nephrosis: - Out of 109 kidneys examined at necropsy or in slaughter houses, renal changes of nephrosis were noticed in 21 cases. Some of the kidneys were collected from animals of Tharparkar GCF, Patna which were found to be died of acute hepatitis and/or dermatitis (photosensitization).

Macroscopic changes: - The kidneys were enlarged, swollen and yellowish pale in colour (Fig. 3). The capsule was moderately tense and appeared over stretched. The cortex was whitish red and appeared somewhat mottled. White streaks were seen on cortical areas while the medullary parts were anaemic. The cortico medullary junction was more or less indistinguishable. In two cases urinary bladders were almost empty, only a few ml of urine could be collected from bladder for routine urological test.

Microscopic changes : - The tubular lumens were narrowed

by epithelial enlargement and the interstitium was filled with oedematous fluid. The cells lining the tubules were swollen and granular. There was also desquamation of epithelial cells lining the proximal tubules in medullary rays (Fig. 4). Round cells infiltration in glomeruli and hyperplastic changes in the walls of arteries were also found at places (Fig. 5). Small yellowish pigments of different sizes (bile pigment) were present in epithelial cells in tubules. Pink stained hyaline material (cast) were present in some tubules (Fig. 6). There was also at places in interstitium extravasated erythrocytes (Fig. 7). Picknotic and karyolytic changes of nuclei of the epithelial cells of the tubules were present. In some of the kidneys, there were tubules lined by regenerated, flat, hyperchromatic or darkly stained epithelial cells (Fig. 8). The epithelial cells of liver in few cases were also swollen and granular and at places there were necrotic changes. A few ml of urine collected showed the presence of albumin.

Glomerulonephritis: - Glomerulonephritis were noticed in kidneys of 5 dead animals examined post mortem or after slaughter in local abattoirs. Out of five animals, 3 belonged to the Government Cattle Farm, Patna.

Gross appearance: - Generally both kidneys were enlarged and pale in colour (Fig. 9). The capsules were to some extent tense and easily stripped off. When cut, the cut surfaces were pale and greyish red in colour and have mottled appearance.

Small redish and grey areas were present on the cortical portion of the kidneys. A few tiny white subcapsular spots were also seen in the kidneys. Cortical areas were some what blending with the anaemic medullary portion. The line of demarkation between these two zones were obscure.

Microscopic changes: - The glomeruli revealed some increased cellularity and leucocytic emigration (Fig. 10).

The capsule of Bowman contained eosinophilic granular materials along with indistinguishable cellular debris (Fig. 11).

Fibrous tissue proliferation was very much prominent in the walls of the blood vessels (Fig. 12) even if it developed at the point of almost slight oblitrating the lumen of the arteries.

There was also slight to moderate thickening of wall of Bowman's capsule due to fibrous tissue proliferation (Fig. 13). Cloudy swelling, and necrotic changes were noticed in the tubular epithelium (Fig. 14). The epithelial cells lining the tubules were desquamated to form pink stained granular clumps in the lumen of the tubules (Fig. 15). There was also some increase in the interstitial tissue with infiltration of lymphocytes and plasma cells in the interstitium at places. In some cases there were areas of extravasated erythrocytes in the intertubular interstitium. Smears of the kidneys did not reveal any micro-organisms.

Interstitial nephritis : - Out of 109 kidneys examined

for gross and microscopic changes, 21 cases alone constituted to the group of acute interstitial nephritis.

Macroscopic appearance: - Kidneys were enlarged, redish white in colour, and soft in consistency (Fig. 16). The capsules were tense and stripped easily. When cut there were raised hyperaemic areas on cortical surfaces. The cortical surfaces were mottled, grey. A few to numerous pin head size raised areas were present on the cortical surfaces. There were also petechiae near cortico medullary junction. The capsules were thick.

Microscopic appearance: - There were deposition of bluish calcium salts at places. The Bowman's capsule were dilated. The interstitial tissue were thickened due to infiltration of lymphocytes, plasma cells, and a few neutrophils (Fig. 17). The cells lining the tubules were desquamated to form clumps in the lumen. There were areas of extravasated cells, focal haemorrhage in the tubules and peritubular fibrosis.

Focal interstatial nephritis: - 5 kidneys out of 109 specimen examined showed the lesions of focal interstitial nephritis.

Macroscopic appearance: - The kidneys were swollen, the capsule could be easily removed. There were greyish white sub-capsular areas (Fig. 18).

Microscopic appearance: - The interstitium was more prominent and emphysised, and contained pink stained materials and oedematous fluid. At places there was infiltration of lymphocytes, plasma cells (Fig. 19). Fibrosis was also marked at some other places. On examination of smears organisms not dissimilar from E. coli were noticed in the kidneys.

Sub-acute and chronic interstitial nephritis: - 32 kidneys had sub-acute or chronic interstitial nephritis out of 109 kidneys examined post mortem or abattoir.

Gross changes: - The kidneys were enlarged, tense and there were areas of elevation and depression (Fig. 20). The capsules were tense and prominent and were firmly adherent to the renal surfaces. It did not stripped easily. Sveral greyish nodules were present on the cut surfaces of the renal cortex (Fig. 21). These nodules were more marked when capsules were removed. A part of cortical surface also came out while stripping the capsules. When cut, the cut surfaces were pale grey and its consistency was firm. The cortical portions were contracted and appeared greyish pale red in colour. White streaks were also present on it. The medullary portions were congested. There were no distinct line of demarcation between cortico-medullary junctions.

Microscopic changes: - The glomeruli were distorted and the capsular space was slightly increased (Fig. 22).

Bowman's capsule was thickened at places and glomeruli were

adherent to it (Fig. 23). There were infiltration of round cell in interstitial tissue. There were also formation of fibrous tissue in the interstitium, and the fibrous areas contained a few ramanant of tubules. The interstitial tissue were also thickened. The tubules were dilated at places and contained granular material (Fig. 24). The epithelial cells lining the tubules at places were desquamated and formed clumps. The lumen contained brown pigment and lining epithelial cells were granular. There were marked fibrosis in the interstitium. In such areas the tubules were hardly visible. There were also empity spaces around the blood vessels due to perivascular cedematous changes. The walls of the blood vessels were thickened due to hyperplasia of fibrous tissue, and the glomeruli were shrunken or distorted.

Pyelonephritis: - Out of 109 kidneys examined in present study only 4 cases of pyelonephritis could be recorded.

These four cases belonged to Government Cattle Farm, Patna.

Gross appearance: - Lobes of the kidneys were severely enlarged, tense and pale in colour and formed several greyish white some what ovoid raised areas (Fig.25 & 26). The capsules were loosely attached to the cortex of the kidneys with thin grey purulent materials, and at places they were adherent to the cortical surfaces. When capsules were stripped off several abscesses from pin head size to pea size were noticed on the surfaces of the kidneys. When cut, the cut surfaces

of the kidneys were pale grey in colour and revealed several abscesses of varying sizes containing greyish pink purulent material (Fig. 27). The cortico medullary junctions were completely oblitrated. There were much destruction in medulla and to a lesser extent in cortex, along with formation of irregular round empty spaces. The calyces of the kidneys were filled in with cream colour pus. Greyish white streaks were radiating from the calyces through medulla towards cortex. The papillae were discoloured, greyish white in colour and were soft. In the ureters there was presence of thick purulent material.

Microscopic appearance: - There were areas of neutrophilic infiltration along with a few lymphocytes in the intertubular tissue (Fig. 28). The epithelial cells lining the
tubules were desquamated and formed indistinguishable cellular
debris mixed with degenerated neutrophils. Intertubular
fibrosis in kidneys were present at places (Fig. 29 and 30).
The glomeruli in Bowman's capsule were some what showing
increased cellularity, due to leucocytic emigration. The
capsule of Bowman contained indistinguishable cellular
debris (Fig. 31). At places there were areas of focal round
cell infiltration along with a few neutrophils (Fig. 32 and
33). There were oval cavities of eosinophilic granular
substance surrounded by a fibrous tissue capsule (Fig. 34).
There was also thickening of arteries and arterioles and
empty spaces can be seen outside the arteries. In some cases

there was marked fibrosis in cortex. The papillary tissue were showing necrotic changes and deposition of blue stained calcium pigments.

<u>Tuberculous nephritis</u>: - 3 cases of tuberculous nephritis were detected out of 109 kidneys examined in the present study. These cases belonged to Government Cattle Farm, Patna.

Gross appearance: - Both kidneys were severely enlarged with very prominent renal lobes (Fig. 35). In one case the right kidney was twice the size of left one and left kidney itself was 4 times bigger than normal. The capsules were tense and adherent to the renal surfaces and could not be easily striped off. The kidneys were firm in consistency and bore several greyish white nodules of various sizes scattered throughout the renal parenchyma (Fig. 36). The greyish white caseous areas were found both, in cortex and medulla and were hard to cut.

Microscopic appearance: - The lesions consisted of tubercles having the centre of caseation and calcification (Fig. 37). In the nodules there were epitheloid cells and also giant cells surrounded by zone of lymphocytes. The blood vessels were dilated and were representing the zone of hyperaemia. Pibrous capsule, caseous, and calcified areas were surrounded by a wall of fibrous tissue containing a few lymphocytes (Fig. 38).

Cloudy swelling: - 10 cases showed the presence of cloudy swelling out of 109 kidneys examined in laboratory.

Gross appearance: - The kidneys were enlarged, swollen, oedematous and pale in colour. The capsule were smooth and easily detachable from the renal surfaces. When cut the cut surfaces bulged out and could not be brought readily into original opposition.

Microscopic lesions: - The cells lining the tubules were swollen and granular due to presence of minute granules.

(Fig. 39). The cells could not have their normal transparency. There was increase in Bowman's capsule space. The blood vessels were distended with blood.

Necrosis: - 7 cases of necrotic kidneys were encountered while examining 109 kidneys.

Gross appearance: - The kidneys were enlarged and oedematous. The organs were pale and they were more marked in medullary portion. The capsules were adherent to renal surfaces at places. The cortex showed yellowish grey foci. The foci were of varying sizes. The natural markings were obscure.

Microscopic appearance: - The epithelial cells lining the tubules were swollen and granular. At places the tubular epithelial cells were vacuolated. There was pink stained

materials in some of the tubules. The tubular cells were necrosed and desquamated. At places the tubules were lined by dark stained epithelial cells. There was oedema of glomeruli and interstitial tissue. There was also increase of fibrous tissue elements in the interstitium. The walls of the blood vessels were thick and the blood vessels were hyperaemic. The glomeruli were swollen and were more emphysized at places. There were also areas of round cell infiltration, and other inflammatory cells. Some of the glomeruli were completely oblitrated and formed necrosed masses. Necrotic cells were darkly stained and also fragmented.

Leptospirosis: - Of the 38 kidneys examined from cattle and buffaloes organisms indistinguishable from spirochetes were demonstrated in 2 exotic cattle Holstein Friesian (calf no. HF 3/74 and cow no. HF 2). The Holstein Friesian herd had previous history of several abortions. The Holstein Friesian calf was reported to suffer from polyuria and albuminuria. The cow under report had also aborted earlier. Since the organs has been preserved in 10% formal saline no bacteriological examination could be carried out.

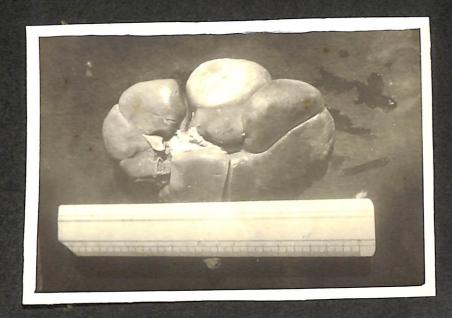
Macroscopic changes: - There were several greyish white subcapsular areas (1 to 2 mm diameter) in both the kidneys of calf (Fig. 40).

Microscopic appearance : - There was presence of

lymphocytes, plasma cells in the interstitial tissue. The epithelial cells lining the tubules showed degenerative changes. In the tubules there were several organisms as microclusters (Fig. 41).

特特特

普





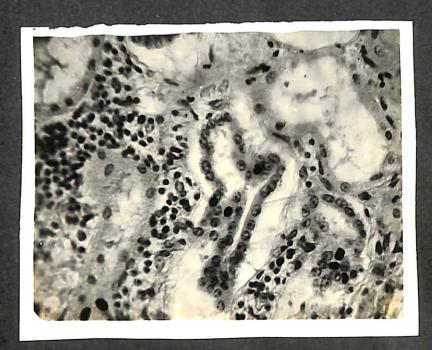




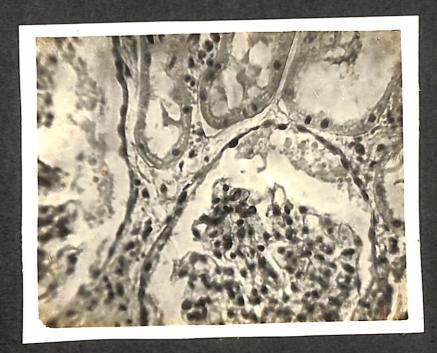


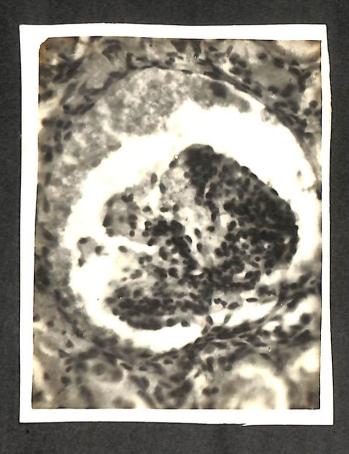


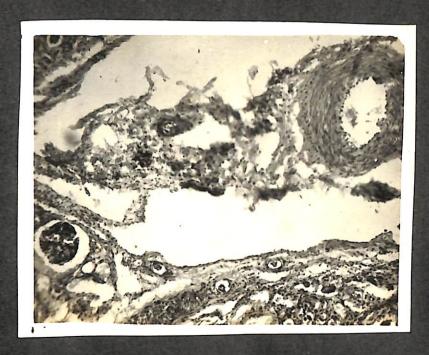








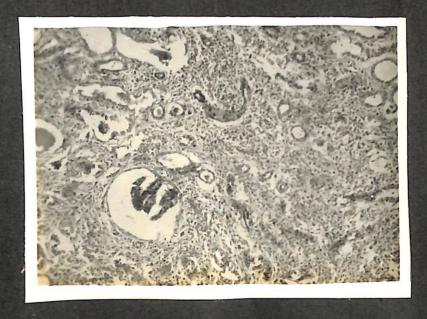




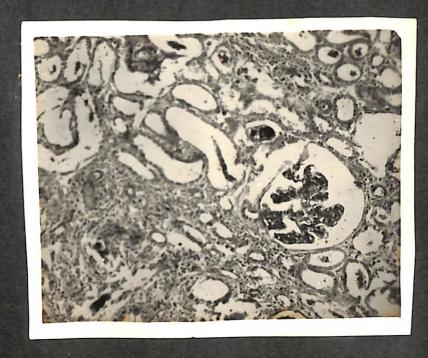






















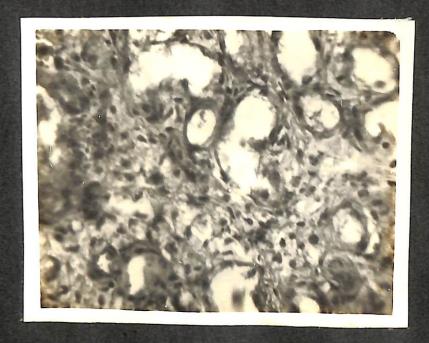


















Fig. 40: Note the enlarged kidney with several greyish white sub-capsule area.

Fig. 41 : Section of kidney showing spirochetes.

Levadidi x 1000.

DISCUSSION

DISCUSSION

In the present study, all the investigation has been ctegorised into two broad types (i) statistical investigation and (ii) investigation into the renal diseases of bovines in Bihar. The statistical investigation comprised of pathological data obtained from the survey of the autopsy records available in the Department of Pathology, Bihar Veterinary College, Patna as well as Government Cattle Farms.

Perusal of the available literature on the aforesaid two aspects, shows that there are scanty informations
available on the different pathological conditions, affecting
kidneys of cattle and buffaloes. Attempts have been made to
carry out the discussion on the findings of the present investigations, and correlate the different findings in view of the
researches done by various previous workers.

While analysing pathological data of autopsy records of 1609 dead cattle and buffaloes, it was found that the renal diseases constituted 19% of different diagnoses recorded in these animals. Renal changes were noticed in several autopsy cases throughout the year but the percentage of renal diseases were considerably high in the animals when Bihar was under severe grip of drought and flood. There is definite record of flood and drought in 1951-52, 1968-69, 1971-72 in several parts of this province and renal diseases

were found to be considerably high during these years. It appears that, in the unusual time (during flood and drought), the animals get compelled to thrive on unusual harmful plant due to scarcity of fodder, and suffered from various diseases. Lantana camara (Putus) is abundently available in Bihar and specially during rainy season. There was marked trend of rise in the cases of renal diseases in Government Cattle Farm, Patna when they get access to these luxurious growth of this plant. Mostly the lesions of dermatitis, hepatitis and nephrosis are noticed in these animals. Nandi (1954) pointed out that factors derived from various plants caused irritation and damage to the kidneys. He even listed names of 25 plants responsible for causing irritation to kidneys.

Data on haematological studies were also analysed in the animals revealing presence of albumin in urine. The percentage of neutrophils was found quite high in them. Haemoglobin percentage was considerable low in these cattle (Annexure I). The degenerative changes (cloudy swelling and necrosis) were noticed in cases of pneumonia and photosensitization in Tharparkar cattle. Kidneys in such cases also showed greyish white discolouration of the cortex. In two cases the urinary bladders were almost empty and albumin could be found in a few ml of urine collected from the bladders.

According to Singh and Prasad (1971) incidence of tuberculosis in male and female cows have been reported to

be 28.8 and 9.1 per cent respectively in the Tharparkar cattle of Government Cattle Farm, Patna. Tuberculous nephritis and tuberculous mastitis are equally serious hazards for cattle industries. Although the measures for control of tuberculosis in Government Cattle Farm, Patna was enforced from 1st April 1957 but tuberculosis cases still exist in this farm. Survey of the autopsy records of farm animals does reveal definite record both tuberculous nephritis and mastitis. It, thus, seems that sporadic cases of tuberculous nephritis are responsible for persistant perpetuation of these diseases in the Tuberculosis in farm animals has caused loss to milk production, and culling of valuable cows and bulls for breeding purposes. Detection of renal tuberculosis in Government Cattle Farm, Patna warrants vigorous attempts for intensifying tuberculin testing programme in farm animals for controlling this dreadful cattle disease.

The various renal diseases as encountered in the present study have been grouped into (i) Glomerular diseases (ii) Tubular diseases (iii) Interstitial tissue diseases.

In this context of classifying renal pathological conditions it is worthwhile to point out that the system of classification of the portion of the kidneys previously affected, and types of involvement provide very strong rational basis of classification of renal diseases. Anderson (1957) and Jubb and Kennedy (1963) have emphatically stressed on principle for the classification of renal diseases.

Congenital or acquired cysts in kidneys occur in Out of 109 cases examined only one cystic patholoanimals. gical condition was found in cattle. The kidneys showed several cysts of various sizes in the lobules with thick greyish white capsular wall. The cysts contained serous fluid. According to Jubb and Kennedy (1963) the acquired cysts of kidneys are usually smaller than congenital cysts. In the present case there were several very prominent cystic structures. There was fibrosis of interstitial tissue in the lobules and there was area of tubules which had very much considerably dilated, to form markedly dilated cyst-like empty spaces. It is very difficult to say definitely on the pathogenesis of this condition of this specimen, but however, observation of intertubular fibrosis and dilated tubules appears to indicate a cyst to be of acquired character.

In the present study parenchymatus degeneration as well as necrotic changes in the tubular epithelial cells have been considered to as cytological indices of nephrosis. The condition of nephrosis were noticed in several cases of pneumonia and photosensitivity. The characteristic gross changes in kidneys were enlargement of the kidneys with parboiled appearance. In cases of photosensitivity, hyaline casts were noticed in luminae of tubules. Nephrosis includes several heterogenous changes from cloudy swelling to necrosis, detected mainly in the epithelial cells of the tubules.

Nephrosis occurs on account of poisoning due to salt of
Mercury, Arsenic, Sulphonamides etc. in animals. In some of
the 3 fatal cases of photosensitivity in calves of Government
Cattle Farm, Patna, kidneys were swollen and had pale or
yellowish cut surfaces. Apart from degenerative changes such
as cloudy swelling and necrosis in the tubular epithelial
cells and hyperchromatic cells lining the tubules were also
noticed. This points out epithelial regeneration, subsequent
to tubular epithelial retrograde changes. In these cases,
the liver also revealed cloudy swelling and necrotic changes.
Hyperplastic fibrous thickening of the walls of intra-remal
blood vessels in photosensitization has been reported by
several workers (Monlux et al., 1968, and Singh and Lakra,
1968).

Out of 109 kidneys brought to the laboratory for histopathological examination glomerulonephritis were noticed in the kidneys of 5 cases. 3 dead animals were examined in post mortem or 2 after slaughter of the animals in local abattoirs. Wettimuny (1963) reported 11 per cent of glomerulonephritis in bovines. The lesions of glomerulonephritis were noticed in both kidneys of bovines. Nieberle (1922) noticed the lesions of sub-acute and chronic glomerulonephritis in a cow with a large white kidneys. Udall (1954) has commented that acute diffuse glomerulonephritis is rare in herbivorous and he also considered these due to effect of some toxic and infectious diseases. In the present study,

not a single case was noticed in cattle and buffaloes. this respect the findings are in agreement with the findings of Udall (1954). The histopathological changes were mainly found to affect the glomeruli and tubules. Histopathological lesions consisted of fibroplasia and leucocytic infiltration (mostly leucocytes, and plasma cells) in the interstitium. More cellular glomeruli, pink stained homogeneous or granular indistinguishable materials with a few cells in the capsular space, slight to moderate fibrosis around the capsule of Bowman and intertubular fibrosis (replacing or distorting the tubules) with infiltration of lymphocytes and plasma cells, and fibrous thickening of arterial walls were some of the very conspicuous findings in glomerular nephritis. There was also focal aggragation of mostly lymphocytes. But in such cases the lesions were also seen in glomeruli and peritubular fibrosis was found in the other part of renal parenchyma. Smith and Jones (1970) considered glomerulonephritis as a rare condition in animals. According to Jubb and Kennedy (1963) diffuse glomerulonephritis is a poorly defined diseases in animals, but does occur in all species, and much less common than diffuse interstitial nephritis. Reports on glomerulonephritis in swine and dogs are available in literature and also very much common in horses used for antisera produc-In the present study, formation of glomerular exudate due to glomerular likage, cells and proteins in the form of pink stained materials consisting of a few cells, were noticed

in the kidneys of cattle and buffaloes. Moderate fibrosis of Bowman's capsule was also noticed in two cases. There was degenerative changes in tubules. From the aforesaid information, the author is inclined to believe that although glomerular nephritis of either sub-acute or chronic type as found in human beings does not exists in bovines, yet there is type of glomerulonephritis which have got the features of pink stained material along with cellular debries in Bowman's capsules and tubules, and fibrous thickening around the Bowman's capsule and also degenerative changes in tubules. This condition was considered glomerulonephritis on account of observation of capsular exudate, periglomerular fibrosis and tubular retrogressive changes. In the kidneys, the tubular epithelial cells depends on the glomeruli for nouris-The glomerular changes, results in degenerative changes The epithelial cells of the tubules become more granular. No specific organisms were found in the smears of kidneys taken from Tharparkar herd of the Government Cattle Parm, Patna. Cultural test found to be of no result and as such it is very difficult to throw light on different factors causing glomerulonephritis in animals. This necessiates the further detailed study for making aetiological diagnosis of It is also probable that pathologic changes in such cases resulted from the initial degenerative changes in tubules leading to production of toxic materials which caused subsequent infiltration of inflammatory cells and peritubular

fibroplasia.

Two kinds of pyelonephritis namely (i) Haematogenous pyelonephritis (i1) urogenous pyelonephritis occur in cattle. In the present study only 4 cases of pyelonephritis were met Enlargement of kidneys, adherence of thickened capsule to the kidney's cortex at places, and presence of greyish white depressed patches on cut surfaces with purulent material were some of the characteristic changes in pyelonephritis. The calyces of such kidneys were also dilated and contained dirty greyish white mucoid materials. The papillae were greyish white in colour having necrotic appearance radiating from greyish white streaks from the pelvis towards the cortex were also to be seen. The chief microscopic features of pyelonephritis was dilatation of tubules which contained indistinguishable cellular debris mixed with degenerated neutrophils. In the inflammed areas of the kidneys there was fibrosis of arteries, the lumen were also considerable stenosed. There was infiltration of neutrophils and a few lymphocytes in the interstitium of the kidneys. Hutyra and Marek (1926) noticed spread of infection through wrethra and bladder to kidneys and pointed out natural infection did not occur through blood stream because pyelonephritis could not be produced by intravenous injections. Dhanda and Das (1954) reported pyelonephritis in cattle for first time in India. Abscess in pelvis and cortex of the affected kidneys were similar to those of Dhanda and Das (loc. cit), Boyd (1928)

C. pyogenes, E. coli, C. renale and P. valgaris were incremated aetiological factors for pyelonephritis by various workers (Boyd, 1927; Puttaswami, 1932, and others). According to Jubb and Kennedy (1963) pyelonephritis is most common in adult cows. In the present study all the cases were noticed in females.

Caseous and calcarious lesions are very much frequent findings in the proncheal, mediastinal lymph nodes of cattle and buffaloes. Singh and Prasad (1971) also reported high incidence of tuberculous lesions in broncheal lymph nodes of the cattle. Tuberculous lesions were also noticed in lungs, spleen, intestine, and liver of the cattle. Out of 1609 dead animals examined post mortem, renal tuberculosis could be found only in 3 cases in the Tharparkar cattle of Government Cattle Farm, Patna. In these cases kidneys were very severely enlarged and cut surfaces revealed greyish white nodules of various sizes. Acid fast organisms not dissimilar from Mycobacterium tuberculosis were detected from the smears of the kidneys. Detection of renal tuberculosis and tuberculous mastitis is extremely serious problem in cattle herd because the chances of spread of infection from animal to animal are always there. The author believes that the tuberculosis has got strong foot hold among the Tharparkar herd of Government Cattle Farm, Patna owing to

such open and hidden cases of tuberculosis. Perusal of the available literature does not show any published information of renal tuberculosis in Indian cattle. Sinha (1968) only described granulo-matous inflammation in kidneys of cattle.

Parenchymatous degeneration were observed in the kidneys of bovines having pneumonia with pulmonary changes. Herbut (1959) reported occurrence of parenchymatous lesions in kidneys in man affected with pneumonia. Such degeneration was also prominent in photosensitized bovines.

Renal calcification were present in two cases only, but in all the 3 cases of tuberculous nephritis dystrophic calcifications were noticed.

Out of 109 kidneys examined histopathologically, interstitial nephritis was noticed in 53.2% of cases in cattle and buffalces. From the result of the present study, it is quite apparent that interstitial nephritis is one of the commonest types of nephritis to be noticed in bovines. The histopathological findings were similar to those reported by Jubb and Kennedy (1963) and Mohanty (1961). Infiltration of neutrophils and plasma cells in the interstitial tissue and peritubular fibrosis, were most important prominent changes in chronic interstitial nephritis. In acute interstitial nephritis, there was pink stained materials in the Bowman's capsule and there was also cedema in the interstitial tissue. In sub-acute and chronic cases fibrous tissue elements were

noticed in interstitial tissue. At places, there were areas of oblitration of tubules. Glomerular changes were not so marked. There were areas of round cells infiltration in the cortex, in cases of focal interstitial nephritis. Fibrosis was also noticed in a few cases of focal interstitial nephritis. Grossly the kidneys revealed several vari-sized sub-capsular white foci in the kidneys. Such foci were also present on the remal cut surfaces. Occurrence of white spotted lesions in bovines has been attributed to various factors in literature. Br. abortus was considered to cause some acute focal interstitial nephritis with leucocytic aggragation by Lubke (1934) but Magnusson (1934) and Heinen (1938) did not find any association between such lesions and Br. abortus. White spotted kidneys were also reported by Moore and Hallman (1936) in experimental Vitamin A difficient calves.

tered or almost slightly altered glomeruli were important features for distinguishing other types of nephritis. In focal diffuse interstitial nephritis the glomeruli were atrophied with increased capsular space. There was fibrosis in the peritubular interstitium and infiltration of lymphocytes and plasma cells in it. E. coli was found in the kidneys revealing white spots beneath the capsules. Some cellular casts, were also present in the tubules. Wiidik (1937) considered toxic factors for causing white spotted kidneys in

Leptospira infections have been reported to cause focal interstitial nephritis by several workers (Jungherr, 1944, Reinhard, 1951 and Ristic et al., 1957). Reinhard (1952) observed leptospira in 2 of 5 calves autopsied but focal interstitial nephritis were present in all of them. He suggested focal interstitial nephritis in kidneys are caused by leptospira. He also pointed out the possibilities of factors other than leptospira. Hofmann (1955) did not find any aetiological factors in 29 calves which revealed lesions of acute, sub-acute, or chronic interstitial nephritis. Allergins of unknown origin have also been incriminated to cause white spotted kidneys by Tsiroyannis (1957). and Stoener (1955) reported wide spread chronic interstitial nephritis and renal tubular proliferation in cows affected with L. pomona. Various factors such as bacteria, toxins, renal stones have been suggested to cause interstitial nephritis. 38 kidneys of dead animals of Government Cattle Farm, Patna and local abattoir were examined by Levaditi's procedure. Organisms not dissimilar from spirochetes were found in only Bovine spirochaetal infection was noticed for the first time in Bihar. In Madras Stae, the first outbreak of leptospirosis was recorded in cattle by Venkataraman and Jagarnnathan (1961). The detection of spirochaetal infection necessiates the further detailed bacteriological investigations in the Government Farms.

Examination of smears and subsequent cultural

tests of the kidneys did not reveal any organisms. probable that the histopathological lesions of interstitial nephritis are caused by some other actiological factors. According to Smith and Jones (1970) elevated white spots or white streaks in parenchyma resulted from showering of infected emboli in the kidneys. It was also opined, that the origin of such foci might have been in the umbilious infected at birth but healed at the time of slaughter, but in the present study there were 11 cases of bovine kidneys which revealed vari-sized sub-eapsular white spots, and also white streaks extending from outer margin of cortex into its deeper part. The percentage of sub-acute or chronic interstitial nephritis is very high in Tharparkar cattle and several non-script Definite correlation between aetiological factors and pathogenesis of such lesions still remains to be estab-It is, therefore, suggested that further lished in future. investigations are required to throw light on definite actiological diagnoses in such cases.

安安安

SUMMARY

SUMMARY

In the present investigation, statistical investigation was carried out into renal diseases and various other diseases of pertinent materials derived from Government Farms and slaughter houses. Out of 109 pairs of kidneys examined in cattle and buffaloes, the incidence of glomerulonephritis was 4.6%, interstitial nephritis 53.2%, nephrosis 19.3%, pyelonephritis 3.7%, and renal tuberculosis 2.7%. Renal diseases in bovines showed comparatively high incidence during drought and flood.

Out of 38 kidneys of bovines subjected to silver impregnation technique, organisms indistinguishable from spirochetes were noticed in two cattle.

Pathological changes in different types of remal diseases in cattle and buffaloes have been described. Interstitial nephritis had the highest prevalence in comparison to other diseases diagnosed. The incidence of interstitial nephritis was very high in Tharparkar cattle above 3 years age. Pyelonephritis were obtained only in females but remal tuberculosis were observed in males of Tharparkar cattle. Haematological studies in cases of albuminuria revealed increase in percentage of neutrophils and decrease of haemoglobin percentage. Bovine spirochetal infection was noticed in cattle for the first time in Bihar.

BIBLIOGRAPHY

BIBLIOGRAPHY

Abramov, L.P. (1962). Veterinariya Moscow 12: 45-46.

Akcay, S. and Pamukum, A.M. (1950). Turk. Veteriner Kimleri De Kimleri Dernegi Dergisi 20: 319-332 (Abstr. Vet.Bull. 21: 2863).

Awrorow, A.A. (1941). Cited by Hadlow and Stoenner, 1955.

Anderson, W.A.D. (1957). Pathology 3rd Edition.

Anderson, A. (1936). Skand. Vet. 26: 241-290 (Abstr. Vet. Bull. 7: 129).

Baker, J.A. and Little, R.B. (1948). J. Exp. Med. 88:295-307.

Bell, E.T. (1950). Ed. 2nd. Lea and Febiger, Philadelphia.

Bemis, H.E. (1926). Cited by Palmer, 1938.

Biswal, G., Gray, M.L., Thorp, F., Jr. (1953). Mich.St. Coll.Vet. 14: 19-25 and 33 (Abstr. Vet. Bull. 25:25).

Bos, A.W.A. (1936). Tijaschr. Diergenessk 63:790-799 (Vet. Bull. 7:397).

Bloom, F. (1939). Arch. Path. 28: 236-245.

Boyd. W.L. (1918). Cornell. Vet. 8: 121-123.

Boyd, W.L. (1927). Cornell. Vet. 17: 45-56.

Boyd, W. L. (1928). Cornell. Vet. 18: 456.

- Boyd, W.L. and Bishop, L.M. (1937). J.Am. Vet. Med. Assn. 90 : 154-162.
- Bruckmuller, R. (1869). Cited by Ritzenthaler, 1910.
- Burdin, M.L., Froyd, G. and Ashford, W.A. (1958). Vet.Rec. 70: 830-835.
- Butura, I., Baugau, S. and Sirbu, Z. (1959). Lucr. Inst. Pat. Igiena, anim. Bucurstr 9: 285-295.
- Butura, I., Sirbu, Z., Bangan, S., Jitaru, G., Precup, O. and Boca, R. (1960). Lucr. Inst. Pat. Igiena. anim. Bucuresti. 10: 151-160 (Abstr. Vet.Bull. 31: 1248).
- Carsico, G. (1953). Atti. Soc. ital. Sci. Vet. 6: 206-208 (Abstr. Vet. Bull. 24: 526).
- Carrora, O. and Galli, G. (1966). Att. Soc. ital. Sci. Vet. 20: 545-548 (Abstr. Vet. Bull. 37: 3885).
- Christiansen, M. (1919-20). Cited by Udall, 1954.
- Cirenei, G. (1938). Clin. Vet. Milano. 61: 494-496. (Abstr. Vet. Bull. 9: 3487).
- Csiszer, W. (1943). Allatrav. Lapok 20: 115 (Abstr. Vet. Bull. 15: 2221).
- Dammann, Z. (1877). Cited by Feenstra and Thorp, 1946.
- Dhanda, M.R. and Das, M.S. (1954). Indian Vet. J. 31: 353.

- DiDomizio, G. (1948). Atti. Soc. ital. Sci. Vet. 2 : 441-496. (Abstr. Vet. Bull. 21 : 2638).
- Ernst, C.F.B. (1905). Cited by Feenstra and Thorp, 1946.
- Faust, E.C. (1949). Human Helminthology 3rd Edition Lea, and Fabinger, Philadelphia.
- Faust, E.C. and Russell, P.E. (1957). Clinical parasitology Lea, and Febriger, Philadelphia.
- Feenstra, E.S. and Thorp, F. (1946). Am. J. Vet. Res. 7: 432-436.
- Feenstra, E.S. and Thorp, F. and Gray, W.L. (1949). Amer. J. Vet. Res. 10: 12-25.
- Fincher (1933). Cited by Udall, 1954.
- French, J. (1950). Arch. Path. 49: 43-54. (Abstr. Vet. Bull. 20: 2056).
- Gaiger, S.H. (1910). Cited by Bhalarao, 1935.
- Gauden, C.H. (1962). Method of statistical analysis
 4th Asian Edition, p. 371.
- Gillol, R. (1888). Cited by Ritzenthalar, 1910.
- Grunder, H.D. (1963). Tieraztl Umsch 18: 426-435 (Abstr. Vet. Bull. 34: 1447).
- Grunder, H.D. and Trautwein, G. (1965). Dt. Tierarztl. Wschr. 72: 442-447.

- Guillebeau, S. (1888). Cited by Feenstra and Thorp, 1946.
- Hadlow, W.J. and Stoener, H.G. (1955). Am. J. Vet. Res. 18: 45-46.
- Harichen, T., Bibrock, B. and Schels, H. (1966). Berl.

 Munch. Tieraztl. Wschr. 79: 356-358. (Abstr.

 Vet. Bull. 37: 1386).
- Heinen, A. (1938). Inaug. Diss. Berlin pp 17 (Abstr. Vet. Bull. 9: 830).
- Hemmann (1934). Z. Feisch. U-Milchyg 44: 382-383 (Abstr. Vet. Bull. 5: 221).
- Hess (1888). Cited by Kataria, 1965.
- Hildebrand (1894). Cited by Osathamondh and Potter, 1964a.
- Hjarre, A. (1933). Tijdschr. Diergenesk 60: 111-115.
- Hoflich, M. (1891). Cited by Feenstra and Thorp, 1946.
- Hofmann, W. (1955). Inaug. Diss. Munich. pp. 24 (Abstr. Vet. Bull. 26: 2989).
- Holle, G. and Schneider, H. (1961). Excerpta. Medica 15: 2836.
- Hoppe, R. (1938). Arch. Path. 23: 504 (Abstr. Vet. Bull. 9: 830).
- Hutyra, F., Marek, J. and Manninger, R. (1946). Special Pathology and Therapeutics of the diseases of domesticated animals. Bailliere Tindal and Cox, London.

Jowett, W. (1925). J. Comp. Path. 41: 166-168.

Jowett, W. (1928). J. Comp. Path. 44: 171-174.

Jubb and Kennedy, P.C. (1963). Pathology of Domestic Animal.

Jungherr, E. (1944). J.Am. Vet. Med. Ass. 105: 276-281.

Kardevan, A. (1964). Mogy. Allatorv. Lap 19: 444-446.

Kataria, R.S. (1965). Thesis of M.V.Sc. Jabalpur University.

Kemens, F. and Szeky, A. (1955). Mag. Allatorv. Lopja. 10: 297-302 (Abstr. Vet. Bull. 26: 757).

Krause, C. (1934). Berl. Tierarztl. Wschr. <u>50</u>: 207-209 (Abstr. Vet. Bull. <u>5</u>: 221).

Kume, T., Sasaki, N. and Nurase, N. (1959). nJ. Jap. Vet.
Med. Ass. 12: 119-125 (Abstr. Vet. Bull. 29: 3031).

Kurtze, H. (1936). Z. Fleisch-U-Hilchhyg. 46: 255-256.

Kwiatkowski, T. (1957). Med.Vet.Vassovie 8: 289-291
(Abstr. Vet. Bull. 27: 2905).

Langham, R.F. and Hallman, E.T. (1941). J. Amer. Vet. Med. Ass. 99: 471-475.

Leons, G. (1957). Ann. Med. Vet. Torino. 7: 165-173.

Lubke, A. (1934). Dtsch. tieraztl. Wschr. 42: 624-627 (Abstr. Vet. Bull. 5: 545).

- Magnusson, H. (1934). Skand. Vet. Tidsk 24: 707-710 (Abstr. Vet. Bull. 5: 695).
- Mallory, F.B. (1961). Pathological technique New York Publishing Co p. 203.
- Mandelli, M. and Cosico, G. (1964). Atti. Soc. Sci. Vet. Sanremo. 17: 194-199 (Abstr. Vet. Bull. 34: 171).
- Magsood, M. (1944). Indian Vet.J. 20: 261-
- Marsh, H. (1945). J.Am. Vet. Med. Ass. 107: 119-121.
- Mathams, D.C. and Sulherland, A.K. (1951). Aust. Vet. J. 27: 68-69.
- Matheson, D.C. (1946). Indian Vet.J. 22: 171-177.
- Metzger, H.J., Hull, F.E. and Ely, F. (1937). Abstr. Vet. Bull. 8: 193.
- Mohanty, G.S. (1961). M.V.Sc. Thesis Agra University.
- Molad, E.L. and Braasch, W.F. (1933). Cited by Bell, 1950.
- Monlux, A.W., Glemn, B.L., Panciera, R.J. and Corcoran, J.B. (1968).
- Moore, L.A. and Hallman, E.T. (1936). J.Dairy Sci. 19: 434-435.
- Moore, L.A., Hallman, E.T. and Sholl, L.B. (1938). Arch. Path. 26: 820-838.

Mora, A. and Del.Gindice, V. (1958). Atti.Soc.ital.Sci. Vet. Rimini-Revenna, 1957. 11: 752-755 (Abstr. Vet. Bull. 28: 4066).

Nandi, S.N. (1955). Indian Vet. J. 32: 202-207.

Nieberle, J. (1922). Cited by Udall, 1954.

Orlop, H. (1956). Arch. Lebens. Mittelhyg. 7: 255-261.

Ottosen, N.E. (1949). Nord. Vet. Med. 1: 410-415 (Abstr. Vet. Bull. 21: 3745).

Palmer, C.C. (1938). J.Am. Vet. Med. Ass. 93: 241-243.

Panse, V.G. and Sukhatme, P.V. (1961). Statistical method for Agricultural works 2nd Edition Enlarged 1961 p. 69.

Peter, A., Herbut (1959). Pathology 2nd Edition.

Pflug, D. (1876). Cited by Rtizenthaler, 1910.

Puttaswami, A. (1932). Indian Vet. J. 9: 21.

Rajulu, P.S. (1956). Indian Vet. J. 35: 325-330.

Reinhard, K.R. (1951). Am.J. Vet. Res. 12: 282-291.

Reinhard, K.R. (1952). Symposium on the Bovine Leptospirosis (1952), pp. 126-137.

Retzlaff, N. (1967). Arch. Lebensmittel. Hyg. 18: 19-104. (Abstr. Vet. Bull. 37: 4593).

- Ristic, M., Galton, M.M., Sanders, D.A. and Steele, J.H. (1957). Ann. Rep. Agric. Exp. Sta. Univ. Fla. 1956 pp. 146 (Abstr. Vet. Bull. 28: 2766).
- Runnells, R.A. (1954). Animal Pathology 5th Ed
 The Iowa State College Press, Ames, Iowa.
- Runnells, R.A., Monlux, S.W. and Monlux, W.A. (1960).

 Principle of Veterinary Pathology Iowa State
 University Press.
- Sali, G. (1961). Veterinaria, Nilano. 10: 338-346.
- Sami, M.A. (1938). Indian Med. Gaz. 73 : 90.
- Schechan, H.L. and Davis, J.C. (1959). Arch.Path. 68: 185-225.
- Seibold, H.R., Keech, H. and Bokelman, D.L. (1961).

 J. Am. Vet. Med. Ass. 138: 424-430.
- Siedangrotzky, B. (1875). Cited by Ritzenthaler, 1910.
- Singh, C.D.N. and Prasad, J.N. (1971). Study on tuberculosis in animals 1.1 on the blood picture of positive tuberculous reactors and incidence of tuberculosis in cattle. Indian J.Amin.Sci. 7: 569-572.
- Singh, C.D.N. and Lakra, P. (1972). Indian Vet. J. 49: 460-463.
- Sinha, B.K. (1968). M.V.Sc. Thesis, I.V.R.I., Izatnagar.
- Smith, H.A. and Jones, T.C. (1963). Veterinary Pathology, Lea and Febriger, Philadelphia.

- Smith, H.A. and Jones, T.C. (1970). Veterinary Pathology.
- Smith, T. (1925). J. Exp. Med. 41: 413-424.
- Smreck, Z. (1957). Vet. Olasn. 11: 426-432 (Abstr. Vet. Bull. 26: 2905).
- Thorp, F., Jr., Lagham, R.F., Clark, C.F. and Doll, E.R. (1943). Am. J. Vet. Res. 4: 240-249.
- Thorp, W.T.S., Keener, H.A., Bechdel, S.I, and Geverant, M.B. (1942). Am.J.Vet.Res. 3: 27-31.
- Tsiroyannis, E. (1957). Veterinariya, Moscow 34: 77-79 (Abstr. Vet. Bull. 29: 2619).
- Udall, D.H. (1954). The Practice of Veterinary medicines 5th Ed. Ithaca, New York.
- Van Der Hoeden, J. (1955). J. Am. Vet. Med. Ass. 126: 207-210.
- Van Vloten, J.M. (1936). Arch. Path. 33: 60-79 (Abstr. Vet. Bull. 10: 534).
- Venkataraman, V. and Jagannathan, P.D. (1961). Indian Vet. J. 38: 444-451.
- Ventura, L. (1932). Nuova. Vet. 10: 16-20 (Abstr. Vet. Bull. 3: 291).
- Virchow, R. (1855). Cited by Faust and Russell, 1957.
- Virchow, R. (1869). Cited by Bialstock, 1956.

- Weil, A. (1886). Dtsch. Arch. Kiln. Med. 30: 209. Cited by Kharole, 1964.
- Welt, L.G. and Peters, J.P. (1951). Yale. J. Biol. Med. 24: 220-230 (Abstr. Vet. Bull. 20: 1646).
- Wester, J. (1935). Tijdschr. Diergeneesk 62: 62-67.
- Wettimuny, S.G. de. S. (1963). Summary of Thesis Glasgow University; 4 (Abstr. Vet. Bull. 35: 4346).
- Wiidik, R. (1937). Wein. tierarztle. Mschr. 24: 353-355. (Abstr. Vet. Bull. 8: 107).

養養養