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THE PATHOLOGY OF KIDNEY DISEASES IN SHEEP

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Pavlos Georgiou Toumazos

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ABSTRACT

Renal diseases in sheep form a diverse spectrum of pathology and an extensive literature review of spontaneously occurring and experimentally induced diseases of the sheep kidney is presented in Chapter 1 of this thesis to provide a comparison with the lesions found in a survey of kidneys in slaughter-house killed sheep. The presentation of results of this survey form the major part of this thesis and provides information on these diseases relative to large populations of sheep which is only sparsely reported elsewhere. The abnormal kidneys under study were obtained from 444 of 13,988 sheep slaughtered over a consecutive five day period at the Borthwick's freezing works, Longburn in January 1980. The prevalence of renal disease was 3.18 per cent and no significant variation ($p < 0.05$) in the prevalence of lesions was found between the various lines, chains and daily totals of sheep examined. From these sheep a total of 830 diseased kidneys were found and these were categorized into seven groups according to the major pathological lesion in each. In some kidneys additional minor lesions were present, making a total of 1212 macroscopic lesions identified.

White spots and streaks constituted the major gross pathological finding in 188 kidneys; pale, red and brown discolouration in 174, 120 and 179 respectively; scars in 107; cysts in 37 and nodules in 25 kidneys. Abscesses, neoplasms and focal space occupying lesions of uncertain aetiology were included under the category of nodule.

Pieces of tissue selected from 181 kidneys to represent the various lesions seen at gross examination were examined histologically. These were identified, recorded and graded according to the anatomical location, pattern of distribution, tissue changes and degree of severity.

The main histopathological feature of the white spotted kidneys was

chronic, mainly multifocal inflammation of the cortical interstitium. Similar but radially disposed inflammatory lesions with marked fibrosis occurred in the scarred kidneys. The pattern of these lesions suggested a haematogenous distribution of a pathogen in the spotted kidneys while the scarred kidneys were probably the result of ascending inflammation or infarctive processes.

Kidneys with pale discolouration showed mild to moderately severe nephrosis of the cortical epithelial cells, while kidneys with brown discolouration showed corticotubular intracytoplasmic and intralumenal haemosiderin deposition. In some kidneys haemosiderosis was restricted to areas of scarring. Red discoloured kidneys showed patchy or diffuse congestion.

Cystic lesions were either parasitic or the result of urinary retention caused by blockage of tubules. In the latter, the blockage was either congenital or associated with chronic inflammation.

With the exception of nephrosis and congestion all the lesions were chronic in nature and for most of them a definitive aetiological diagnosis was not established. In fact, in only those lesions containing Echinococcus granulosus hydatid cysts could such a diagnosis be made.

Additional studies are indicated for the provision of further information on (a) the prevalence of renal diseases in different geographical locations, (b) variation of disease types from area to area and (c) the causes of the lesions identified from this type of investigation.

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CHAPTER 1

REVIEW OF LITERATURE

INTRODUCTION

Knowledge of naturally occuring diseases in the ovine kidney in the last decade has increased markedly in parallel to the application of investigative techniques such as renal biopsy and immunofluorescence and the recognition of new entities resulting from cross species comparison. For instance, the studies of primary glomerulopathies such as those reported in dogs (Peddinghaus and Trautwein, 1977 I and II), horses (Slawson and Lewis, 1979), and cats (Osborne, Hammer, Stevens et al, 1977) have influenced research into the glomerulopathy of Finnish Landrace sheep and the recognition of other glomerulopathies discussed later in this review as models of disease in man.

In this chapter the available literature on pathology of diseases affecting the ovine kidney is reviewed.

Prevalence of disease in ovine kidneys

Reports on the prevalence of renal diseases in sheep are few and of limited value as indicators of renal disease in large populations. The only report relating specifically to renal disease in abattoir slaughtered sheep is that of Zhirik (1974). In this study 1.72 per cent of 119,046 sheep slaughtered in a Moldavian abattoir had kidney lesions.

Of the 196 lesions 194 were chronic although the criteria for chronicity were not stated. In 433 emergency-slaughtered sheep 46 had renal lesions. These were examined histologically and inflammatory changes were present in 36 of which 35 were interstitial nephritis and one glomerulonephritis. The degenerative changes of cloudy swelling, hydropic degeneration, lipidosis and melanosis occurred in four other sheep.

Echinococcal cysts, granulomata, urolithiasis and hydronephrosis were also noted although exact numbers were not given.

Diseases of the ovine kidney

(I) Diseases of the glomerulus

(1) Spontaneous glomerulonephropathies

Proliferative glomerular lesions have been noted as a relatively common finding in routine post mortem material from sheep (Slawson and Lewis, 1979), but to date the glomerulopathy of the Finnish Landrace breed is the only one in which the cause and pathogenesis has been determined.

a) Finnish Landrace glomerulopathy

Thomson, Davidson and Angus, (1973) reported a spontaneous glomerulopathy of Finnish Landrace sheep affecting 5 of 33 lambs born in 1971 and 16 of 40 lambs born in the following year. The kidneys showed diffuse lesions of mesangiocapillary glomerulonephritis upon post mortem or biopsy examination. Five lambs which died with acute renal failure showed a brief clinical illness in which there was enlargement and tenderness in the kidney region and behavioural signs suggestive of central nervous system dysfunction. At necropsy both kidneys of all sheep were pale and greatly enlarged, weighing up to 150 g. The renal capsule stripped readily to reveal a smooth unpitted surface containing circular red or sometimes yellow spots 1-2 mm in diameter. When cut transversely the cortices were tough and the spots noted superficially were seen throughout the entire cortical width. Softening and petechial haemorrhages in cerebral gyri adjacent to midline were present in four of the lambs.

Histological examination of the kidneys revealed a severe diffuse glomerular lesion characterized by proliferation of mesangial cells, thickened capillary basement membranes and florid epithelial crescents.

Accompanying these changes was a mild vasculitis and irregular interstitial infiltration of lymphocytes. Discrete granular subendothelial deposits containing immunoglobulin were demonstrated by electron microscopy and immunofluorescence (Angus, Gardiner, Morgan et al, 1974).

Lambs showing nervous signs had lesions of extensive spongiosis at the junction of the cortical grey matter and the subcortical white matter of the dorsomedial, frontal, parietal and occipital lobes of cerebrum which, while bilateral, were not completely symmetrical. The choroid plexuses showed moderate to severe interstitial oedema. Deposits of IgG were demonstrated by immunofluorescence in the interstitium of the choroid plexus of lateral ventricles and also interposed between choroidal epithelial cells (Morgan, 1977). In one sheep there was endothelial separation with formation of subendothelial electron-lucent spaces.

The early work of Thomson et al had suggested the aetiology was due to colostral transmission of an infectious agent, but this hypothesis was shown untenable by a cross-fostering experiment carried out by Angus et al (1975) using newborn Finnish Landrace and Cheviot lambs. Severe mesangiocapillary glomerulonephritis occurred in four newborn Finnish Landrace lambs reared by their own dams. One of six Finnish Landrace lambs, all reared by Cheviot ewes also developed focal lesions of milder degree. No glomerulonephritis occurred in any of the four Cheviot lambs reared by Finnish Landrace ewes, whose naturally reared lambs died of the disease. An artificially reared colostrum-free Finnish Landrace lamb was also affected.

The experiment showed that the disease was familial, since its prevalence in the progeny of the high risk group allocated on the basis of an affected progeny in previous lambings, was significantly higher ($\chi^2 = 5.14$, $P = 0.05$) than that in the balance of the flock.

Gardiner (1976) found that the third component of complement (C'3)

in serum of newborn, subsequently affected lambs was 5 per cent of that in unaffected lambs and persisted at low levels until renal failure occurred at around 6-8 weeks of age. Examination of renal cortex by immunofluorescence using specific antisera for IgG, IgM, IgA, C'3 and fibrin supported previous morphological evidence indicating a strong similarity between this disease and hypocomplementaemic glomerulonephritis in humans. Immune complexes containing IgG, IgM and C'3 were also present in the choroidal interstitium.

b) Copper deficiency associated nephropathy

Richardson, Terlecki and Gwyneth, (1979) reported a nephropathy in ewes and lambs fed a semi-purified diet of low copper content. The main dietary components were casein, milk powder, white fish meal, maize starch, dextrose, vegetable fat, potassium bicarbonate, sodium bicarbonate, mineral (omitting copper sulphate) and vitamin mixes.

With a few exceptions the severity of the renal lesions varied with the length of the time the diet was fed and ranged from glomerular basement membrane thickening and hypercellularity to glomerular and tubular atrophy, degeneration, interstitial fibrosis and mineralization. In moderately and severely affected cases, arteriosclerosis and lymphocytic infiltration were also a feature. Arterial changes included intimal thickening with reduction and occasionally almost total occlusion of the lumena particularly in the small arteries and arterioles. There was a disorderly proliferation of the media sometimes accompanied by calcification. Periadventitial fibrosis was present in the larger vessels. The authors presumed that the arterial changes leading to partial renal ischaemia and/or hypertension were the major causal factors in this nephropathy.

c) Spontaneous glomerulonephritides of unknown aetiology

i) Langham and Hallman, (1941) reported glomerulonephritis in a

five months old Hampshire ram that had been sick and showed marked dyspnoea due to Oestrus ovis infection for several weeks. On post mortem examination the renal capsule stripped easily revealing a mottled cortical surface due to small raised grey-white vesicles. Numerous narrow grey streaks extended from the periphery of the cortex to the corticomedullary junction on the incised surface. Microscopical examination showed very severe thickening of the glomerular basement membranes due to fibrosis and deposition of hyaline material. The tubules showed secondary degenerative changes, atrophy and gradual replacement of epithelial cells by connective tissue and contained granular and hyaline casts. Occasional interstitial accumulations of lymphocytes and macrophages were present.

ii) The kidneys in 312 of 347 clinically normal sheep aged between 7 months and 3 years slaughtered in abattoirs from six different geographic locations in USA and one in England showed a proliferative glomerulonephritis (Lerner and Dixon,1966a). Complete post mortem examinations were performed in 10 of 347 animals. Kidney tissue from 80 sheep age between 7 months and 3 years and from 15 sheep under 3 months of age was examined by fluorescence microscopy. Electron microscopic examination was made on renal tissue of 10 of the adult sheep and blood and urine samples were collected from 50 adult sheep.

Glomerulonephritis was associated with mild proteinuria and azotaemia in about a third of the sheep and by heavy deposition of IgG and complement along the glomerular capillary walls in all sheep examined. In spite of severe proliferative changes in most glomeruli, the prevalence of scarring was relatively low. Basement membrane changes were absent. The severity of disease increased with age. Glomerulonephritis was not seen in lambs less than 3 months old.

Fluorescent antibody testing for γ globulin and β1C globulin deposition showed a similar age related pattern. None of the lambs aged less than 3 months were affected, but fluorescence was positive for all animals between 7 months and 3 years.

In 90 per cent of the latter group of animals there was intense staining with host γ globulin and β1C globulin in a uniform membranous distribution along the glomerular capillary walls. In the remaining 10 per cent fluorescent localization of these proteins was confined predominantly to the mesangial cells. The disease was characterized ultrastructurally by marked endothelial and mesangial cell proliferation and swelling. There was occasional irregular thickening of the glomerular basement membrane, as well as smudging and swelling of the epithelial cell foot processes. In some sections collagen fibrils were noted in the mesangial cells. Although various parasitic and bacterial infections were found in the sheep under study no correlation between infection by any one particular organism and the degree of glomerulonephritis was established.

Lerner, Dixon and Lee (1968) showed by renal biopsies taken serially over a period of 15 months from 12 adult sheep that the disease was not rapidly progressive. In this study kidneys from sheep with glomerulonephritis were eluted with acid citrate. The eluted material was characterized by density gradient ultracentrifugation and immunoelectrophoresis and shown to consist predominantly of 7S/IgG. Fluoroscein conjugated eluate from sheep with spontaneous glomerulonephritis did not attach *in vitro* to normal lamb kidney. The eluates were shown to have a very short half life in the circulation when trace labelled with ^{125}I and injected into sheep, rabbits and mice and did not fix *in vivo* to normal lamb or "nephritic" adult sheep kidneys. They concluded that spontaneous glomerulonephritis in sheep was not associated with

antibodies against the glomerular basement membrane, and was presumed to be mediated by antigen-antibody complexes.

iii) An apparent association between Campylobacter fetus infection and proliferative glomerulonephritis has been reported in 83 per cent of 243 range sheep by Den Boer (1969). Light and electron microscopic studies of the kidneys showed glomerular hypercellularity, occasional focal thickening of the glomerular basement membranes, marked endothelial and mesangial cell proliferation and smudging of the epithelial foot processes. Fluorescence microscopy showed sheep IgG and complement deposited in a nodular pattern on the glomerular basement membranes. The numbers of nuclei in glomeruli and blood pressure values increased in direct proportion to the severity of the morphological changes. Thirty seven of 105 sheep in this group had antibody titres of 1:50 or higher against C. fetus and the quantitative values of these were in direct proportion to the histologic grading of severity of the glomerulonephritis. Preliminary tests using the indirect immunofluorescence technique showed C. fetus and related antigens on the glomerular basement membranes. Haemagglutination tests showed an antigenic cross reactivity between soluble extracts of sheep glomerular basement membrane and C. fetus antibody.

Slawson and Lewis (1979) found that proliferative glomerular lesions, sometimes advanced, are reported as common in ruminant kidneys and consider that further work is needed to confirm the association between C. fetus infection and spontaneous glomerulonephritis.

(2) Experimental Autoimmune Glomerulonephritis

Experimental autoimmune glomerulonephritis (EAG) has been induced by several workers using heterologous glomerular basement membrane (GBM) and Freund's complete adjuvant (Steblay, 1962, 1963 and 1966;

Lerner and Dixon, 1966b; Rudofsky and Steblay, 1966; Steblay and Rudofsky, 1968; Welsh, Goloff and Smith, 1971; Welsh and Smith, 1972; Batsford and Hardwick, 1977; James, 1978). Steblay (1962) showed that sheep injected every two weeks with heterologous GBM and Freund's complete adjuvant by one or combinations of the intramuscular, subcutaneous or intradermal routes developed a fulminating extracapillary glomerulonephritis which caused death within 27-80 days of the first injection. The earliest changes in glomeruli resulted in increased glomerular permeability and progresses to fibroepithelial proliferation of the glomerular tufts and formation of crescents with eventual scarification. Marked tubular degeneration and interstitial fibrotic change occurred secondary to the glomerular lesions. Kidneys of foetuses and newborn lambs derived from sheep with glomerulonephritis induced in the last third of their pregnancy were unaffected. This suggested either that the causal agent of the fatal maternal glomerulonephritis was not transmitted in utero or in the colostrum, or that it was incapable of affecting the foetal or neonatal kidney. EAG was transfered from affected to normal sheep by artery to artery cross-circulation (Rudofsky and Steblay, 1966).

Lerner and Dixon (1966b) demonstrated that EAG induced by injection of heterologous or homologous GBM was due to the production of anti-GBM and β1C globulins, which were deposited in a linear fashion along the basement membrane of the glomerular capillaries. Serum globulin from nephrectomized sheep with induced EAG contained an anti-kidney antibody which caused an immediate, although transient glomerulonephritis when injected into lambs. The nephritogenic property of the serum could be removed by absorbtion in vitro with sheep GBM.

Steblay and Rudofsky (1968) reported EAG in sheep after injections of human GBM cells and Freund's adjuvant characterised by a continuous

linear deposit of IgG specific to GBM along the GBM. The specificity of autoantibody in kidney eluates was determined by indirect immunofluorescence. Reactivity was demonstrated with Bowman's capsule, GBM, and tubular basement membrane of kidney as well as pulmonary basement membranes in both man and sheep.

The pattern of distribution of immunological reactants in these lesions is considered by Lerner and Dixon (1966b) and Steblay (1966) to closely resemble those seen in the nephritis of Goodpasture's syndrome, nephrotoxic serum nephritis and some other poorly defined chronic nephritides of man, but was not characteristic of nephritides of acute post streptococcal infection or lupus erythematosus. Steblay et al (1966) noted that this appeared to be the first experimental model in which autoantibody had been demonstrated to mediate disease.

James (1978) investigated the pathogenic mechanisms of graft rejection affecting transplanted kidneys using sheep. Renal auto-and allografts were placed into the necks of nephrectomised sheep to determine the structural and functional effects of transplantation, recurrent glomerulonephritis and the influence of immunosuppressive drugs. An experimental autoimmune glomerulonephritis was induced in 12 of 37 sheep by repeated injections of human GBM in Freund's complete adjuvant. These received allografts and immunosuppressive therapy after the onset of haematuria and proteinuria. During the grafting procedure and at 30 minutes, three, six and ten days after transplantation surgical biopsies were collected from the donor kidneys and examined by light, electron and immunofluorescent microscopy.

Allografts in immunosuppressed recipients functioned well during the first post-operative week, but their function deteriorated rapidly during the next week. The non-immunosuppressed allografts functioned

only briefly and in most the glomerular filtration rate fell rapidly 2-3 days after transplantation. All the allografts showed extensive infiltration of lymphoid cells, widespread oedema and swelling of tubular and glomerular cells. The allografts in sheep with EAG showed a linear deposition of anti-GBM antibodies in their glomeruli within 30 minutes of transplantation. This was followed by the development of a rapidly progressive recurrent glomerulonephritis. By the seventh day after transplantation most glomeruli in these grafts had developed large crescents which contained numerous phagocytic cells. The author concluded that in EAG-affected recipients of normal kidneys the principal cause of graft failure was a recurrence of pre-existing autoimmune glomerulonephritis.

(II) Diseases of Renal Tubules

(1) Inflammatory

a) Cytomegalovirus infection

Hartley and Done (1963) reported two unrelated cases of cytomegalic inclusion body disease of the kidney in lambs. The most notable histological feature was a multifocal, predominantly lymphoreticular cell infiltration mainly of the cortical interstitium. There was extensive damage of the tubules in areas adjacent to the interstitial lesions with epithelial swelling and desquamation and formation of proteinaceous casts which often contained disintegrating cells. A scatter of glomeruli showed marked thickening of the GBM and extracapillary collagen deposition. One or more eosinophilic to amphophilic, homogeneous, granular, intranuclear inclusion bodies were frequently present in epithelial cells of the thick arm of the loop of Henle, distal convoluted and the collecting tubules. Sometimes the inclusion body occupied the whole of the distended nucleus. None were found in cells of glomeruli or blood vessels. Occasional faintly staining

cytoplasmic inclusions were also present.

b) Leptospirosis

Leptospirosis is recognized as a disease affecting most domestic species and is important world wide as a zoonosis (Blood and Henderson, 1974; Smith and Armstrong, 1975). Spontaneously occurring leptospirosis in sheep has been reported from New Zealand (Hartley, 1952; Salisbury, 1954; Webster and Reynolds, 1955; Hodges, 1974; Marshall, 1974) and the United States of America (Beamer, Hardenbrook and Morril, 1953; Davidson and Hirsh, 1980), as a cause of abortion or fever, haemolytic anaemia and haemoglobinuria. Beamer et al described an outbreak of abortion in 19 of 180 pregnant ewes in Illinois. Sixteen of the sheep died and post mortem examination of five of the ewes showed icterus, swollen kidneys and marked haematuria. No leptospirae were demonstrated in kidney sections but darkfield examination of stomach contents of one aborted foetus contained spirochaetes and 9 of 11 ewes had sera positive by agglutination lysis testing to leptospirae. The organism was transmitted to a guinea pig by inoculation of foetal stomach contents.

The remaining reports describe signs of fever, jaundice, haemolytic anaemia and sudden death. Pathological examination of these animals revealed essentially similar changes of generalized icterus, thin watery blood, haemoglobinuria, yellow-brown discolouration of liver and brown swollen kidneys.

Histological changes in kidneys indicated recent haemoglobinuric nephrosis, with haemoglobin and haemosiderin casts in tubules and yellow-brown granular pigment (haemosiderin) in the cytoplasm of epithelial cells of the tubules. Focal areas of inflammation were present in the interstitium and varied from lymphocytes, plasma cells and neutrophils in acute lesions to a predominance of lymphocytes and plasma cells in subacute lesions. Silver staining techniques revealed

leptospiral organisms in the tubules and interstitium in some cases.

Observations on the clinical, haematological, serological and post mortem features of experimentally induced ovine leptospirosis were reported by Langham, Morse and Morten, 1958; Hodges, 1974; Marshall, 1974.

Langham, et al reported extensive lesions in the kidneys of 10 lambs experimentally infected with Leptospira pomona which were killed at intervals ranging from 8 to 72 days after infection. Grey-white circumscribed foci and streaks measuring from one to four millimeters in diameter were noted grossly in the kidneys of all infected lambs. Microscopically these areas were characterised by infiltrations of lymphocytes, some plasma cells and few macrophages, mostly in the cortex although scattered foci were observed also in the medulla. Although atrophy and necrosis of the tubular epithelia in areas with inflammatory changes were found in five lambs, regeneration of tubular epithelial cells was present only in one case. Proteinaceous material was found in the urinary spaces of half the infected animals. The Warthin-Starry stain of sections of the kidney of four lambs which were bacteriologically positive at time of necropsy showed leptospirae in the tubules of one lamb only.

Similar observations were reported by Hodges in eight lambs experimentally inoculated with a culture of Leptospira interrogans serotype pomona. Marshall also described similar gross and light microscopic lesions in kidneys of sheep 34 days after experimental infection with Leptospira interrogans serotype pomona. Electron microscopic examination of areas with damaged tubules showed leptospirae concentrated around the periphery of cells of the proximal convoluted tubules and intermingled with the brush border. The number of microvilli

in the brush borders was sometimes greatly reduced and many of the microvilli had bulbous ends. Necrotic epithelial cells within the tubules were observed and these were sometimes found free in the tubular lumen.

c) Eperythrozoonosis

Infection of sheep by Eperythrozoon ovis was first reported from South Africa by Neitz, Alexander and du Toit in 1934. It has since been reported from England, Bulgaria and other European countries, U.S.A., Iran, Australia, New Zealand and Kenya (Sutton, 1974). The main clinical finding is anaemia. Lesions seen at post mortem examination vary depending on the stage and severity of infection and may include icterus, thin watery blood, pallor of muscles, excess pericardial and peritoneal fluid, splenic enlargement, emaciation, and brownish discolouration of the kidneys (Neitz et al, 1934). The renal lesions in E. ovis infection are variable. Kidneys often appear grossly normal but they may be discoloured brown by haemosiderin. Deposition of haemosiderin may be extensive and occurs in the cytoplasm of epithelial cells, the tubular lumena and the connective tissue between the proximal convoluted tubules. These changes are most pronounced in animals showing prolonged haemoglobinuria. Øveras, (1969) observed subacute to chronic nephrosis with glomerular sclerosis in extreme cases. Chronic, focal interstitial inflammatory changes were most prominent in areas with the heaviest haemosiderin deposits. Inflammatory lesions in association with renal haemosiderosis have not been reported by others (Foggie and Nisbet, 1964; Rouse and Johnson, 1966; Jolly, 1967; Sutton, 1974). Øveras suggested that high levels of haemoglobin in plasma may cause renal vasoconstriction and lead to the extensive tubular degeneration.

d) Toxoplasmosis

Renal lesions have not been reported in spontaneously occurring

ovine toxoplasmosis. However, Sharma and Gautam (1978) reported extensive necrosis involving glomeruli, tubules and connective tissue stroma in sheep experimentally infected with Toxoplasma gondii.

e) Q Rickettsiosis

Belchev and Pavlov (1977) reported the clinical symptoms and pathomorphological changes found in five ewes and two weaned lambs examined after having naturally contracted Q fever infection. Definitive diagnosis of Q fever was based on serological examination. At necropsy the kidneys looked normal but on histology showed vacuolar degeneration of the corticotubular epithelial cells and scattered areas of fibroblastic and lymphocytic proliferation with moderate diffuse hyperaemia of the medulla.

f) Sarcocystosis

Renal lesions have not been noted in spontaneously occurring cases of sarcocystosis, however several reports of experimentally induced sarcocystosis record lesions in the kidneys of affected animals. Leek, Fayer and Johnson (1977) described a mild, acute, diffuse glomerulonephritis in eight lambs inoculated orally with Sarcocystis ovicanis sporocysts. The glomeruli were enlarged, hypercellular and showed thickened basement membranes. Schizonts were frequently observed within enlarged endothelial cells in the glomeruli and lymphocytes and monocytes were found in capillary lumena. Renal interstitium contained small focal aggregates of lymphoid cells. The renal medulla was congested.

In a further study Leek and Fayer (1978) reported the induction of abortions in sheep. Eleven pregnant ewes were experimentally inoculated with 50,000, 100,000 or 500,000 Sarcocystis ovicanis sporocysts from dogs. Eight ewes either aborted, died or became moribund before lambing. Of the 15 foetuses, 11 were of normal appearance and four autolysed. Tissues from all foetuses examined were negative for schizonts and cysts.

At necropsy 27 to 33 days after inoculation the acutely ill ewes had severe haemorrhagic pericarditis, epicarditis and myocarditis. Haemorrhages of lesser severity were also present in the kidneys, spleen, liver and skeletal muscles. Schizonts were demonstrated in kidney sections of three of these animals. Ewes surviving the acute illness appeared unthrifty. At post mortem the kidneys of these ewes were moderately haemorrhagic. No sarcocysts were found in the kidneys of these animals.

(2) Degenerative

Toxic Tubular Nephrosis

This term is used in literature in reference to a rather heterogeneous group of degenerative changes affecting the epithelial cells of the renal tubules. Most renal diseases have associated degenerative changes in the tubules and thus the term toxic tubular nephrosis is usually employed where tubular degeneration is the primary or principal process. In this review such diseases are considered by actiology under the headings of chemical, plant and endogenous toxins.

a) Chemical toxins

Lead

Toxic tubular nephrosis has been described in North Derbyshire (Clegg and Rylands, 1966) and Southern Scotland (Butler, Nisbet and Robertson, 1957) in young lambs reared in the vicinity of old lead mining areas. The striking post mortem features of eight lambs examined by Clegg and Rylands were carcass emaciation, osteoporosis and enlarged, pale kidneys. On section the kidneys had marked hydronephrosis and a dark haemorrhagic band between cortex and medulla. Butler et al reported hydronephrosis in only 3 of the 20 animals examined. The kidneys of the other 17 lambs, while normal in size, showed diffuse faint yellow mottling of the external surface.

The histological changes in kidneys were similar in both reports and included extensive necrosis of the proximal and distal convoluted tubular epithelia and degenerative changes which ranged from cellular enlargement with granular cytoplasm and large vesicular nuclei to necrosis. In several kidneys eosinophilic intranuclear inclusion bodies were numerous in the epithelial cells of proximal convoluted tubules. Mild to severe, focal or diffuse glomerular lesions were present and most prominent in the outer zone of the cortex. The changes were progressive with the parietal layer of Bowman's capsule epithelium undergoing cuboidal or in some cases columnar metaplasia. This was accompanied by varying degrees of atrophy, fibrous replacement of the glomerular tufts and periglomerular fibrosis.

Copper

In a review of chronic copper poisoning in sheep, Todd (1969) drew attention to two phases of the disease. The first, a period of accumulation of copper in the tissues, and secondly a phase of sudden copper release which precipitates a haemolytic crisis resulting in toxic tubular nephrosis. Gopinath, Hall and Howell (1974) reported the effect of copper sulphate in 16 sheep given daily doses of 20 mg/kg body weight over a nine week period. Seven sheep were killed after three, five, seven and nine weeks of treatment and the remainder allowed to develop a haemolytic crisis. No gross abnormalities were seen in any of the kidneys collected from the seven sheep killed before haemolysis occurred. The only histological change occurred in the epithelial cells of proximal convoluted tubules which showed the presence of intracytoplasmic eosinophilic granules, which were PAS +ve, diastase resistant and stained for lipofuscin. The number and size of granules were directly proportional to the cumulative copper dose.

The kidneys of animals which died or were killed with haemolysis

were swollen, dark brown to black and showed loss of demarcation between cortex and medulla. Kidneys from those animals which had undergone two periods of haemolysis were blue-black in colour with pinpoint black foci on the surface extending as fine black cortical streaks on transverse section. Histologically the proximal convoluted tubular epithelia showed increased eosinophilia and abundant cytoplasmic granules. In all kidneys the majority of the granules were PAS +ve, diastase resistant and some of them stained for haemoglobin, copper, iron and lipofuscin. Cortical and medullary tubules contained eosinophilic hyaline or granular casts some of which were positive for haemoglobin.

The kidneys of two animals which were killed in the posthaemolytic phase were swollen and blue-black in colour. Histologically there was degeneration, vacuolation, focal necrosis and desquamation of epithelial cells which was most prominent in the proximal convoluted tubules. Large numbers of globular or granular intracytoplasmic granules with similar staining properties to those described above were present. Eosinophilic hyaline and granular casts containing haemoglobin were frequent in the cortical and medullary tubules. Similar studies were reported by Howell and Gopinath (1977).

Mercuric Chloride

Naturally occurring mercurial poisoning has not been reported in sheep, but Robinson and Hesketh (1976) and Robinson and Trafford (1977) have described the early histological, histochemical and urinary enzyme changes in sheep kidneys with experimentally induced mercuric chloride nephrotoxicity.

Six adult cross-bred ewes divided into groups of two were given a single intravenous dose of mercuric chloride; 0.1, 0.25 and 0.5 mg/kg

body weight respectively. Urine samples were taken at hourly intervals for three hours after dosage and alkaline phosphatase, γ glutamyl transpeptidase, leucine aminopeptidase and β-glucuronidase were assayed. The sheep were destroyed at the time the final urine sample was collected. One untreated control sheep was sampled and destroyed in the same way as the dosed sheep.

Histologically the kidneys of sheep given 0.5 mg/kg body weight of mercuric chloride showed a reduction in the cell height in many proximal convoluted tubules with an increase in cytoplasmic granularity in groups of tubules. The nuclei were denser than those in the control. Changes in the other sheep were less marked.

All sheep showed increased excretion of alkaline phosphatase and γ glutamyl transpeptidase over the control. In the highest dosage group leucine aminopeptidase was also elevated. Other urinary enzymes remained normal.

b) Plant toxins

Tribulosis ovis and enzootic icterus

Tribulus terrestris is a plant which grows abundantly in mountainous ranges in South Africa and which has been reported as the direct cause of geeldikkop (Tribulosis ovis) and enzootic icterus in sheep (Brown, 1962). Numerous analyses indicated that T. terrestris contains more than five parts per million of selenium, thus both disease entities were considered to be a low grade subclinical selenium intoxication. In both of these syndromes nephrosis is a prominent feature and causes death due to uraemia.

The changes in geeldikkop are less severe than those of enzootic icterus but in both diseases the kidneys are swollen. In the latter case the swelling may be up to two or three times normal size and the

kidneys are otherwise of similar appearance grossly to those in chronic copper toxicity. Microscopically the changes are those of diffuse nephrosis which is sometimes accompanied by acute glomerulonephritis. The epithelial cells of tubules contained large quantities of bilirubin and iron containing pigment and the tubular lumena were filled with haemoglobin and cellular casts.

Oxalates

Oxalate nephrosis has been reported in sheep grazing pastures containing plants such as Halogeton (Halogeton glomerulatus), pigweed (Amaranthus retroflexus), fat hen (Chenopodium album), soursob (Oxalis cernua) and orchard sorrel (Rumex acetocella) which have high concentrations of oxalates. Affected animals are usually recently introduced to such pastures (Blood and Henderson, 1974; Anderson and Huffman, 1957).

Anderson and Huffman reported severe losses in flocks of sheep grazing pastures rich in H. glomerulatus in the Rocky Mountain region of the U.S.A. Grossly the kidneys were pale with patchy congestion and histologically they demonstrated heavy deposition of calcium oxalate crystals within the tubular lumena. Nearly all nephrons were affected. Many casts were present in the proximal and distal convoluted tubules and urinary spaces contained a pink staining precipitate. Wilson and Wilson (1961) suggested that fungi such as Penicillium spp. might be capable of producing significant amounts of calcium oxalate in feeds and could thus contribute to outbreaks of poisoning. One such outbreak in young lambs was reported by Linklater and Angus (1979). The source of oxalate was not clearly indentified but may have been from mould-contaminated concentrates fed to both ewes and lambs. Quite heavy growths of Penicillium spp. were isolated from crushed oats and the coarsemix fed to the ewes and lambs in this

outbreak. Five of sixty purebred Suffolk lambs died when four to six weeks old and had histological changes consistent with reports of experimental poisoning of sheep by oxalate containing plants (Shupe and James, 1969) and ammonium oxalate (James, Seawright and Steele, 1971). The renal cortices showed numerous dilated tubules lined by flattened cells. Heavy deposits of oxalate crystals as well as eosinophilic hyaline or granular casts were present in many tubules and a few crystals were also seen in the urinary spaces of glomeruli. The crystals were most profuse in the distal convoluted tubules in association with proliferation of intertubular connective tissue.

Oxalate intoxication in sheep has also been induced experimentally by intravenous or intrarumenal sodium or potassium oxalate injections (Watts, 1957 and 1959).

James et al (1971) reported experimental oxalate intoxication in 10 sheep dosed intraruminally with ammonium oxalate at 550 mg/kg body weight. Nine sheep died within 12-22 hours of dosing. At necropsy the kidneys of all animals were swollen and upon incision the cortex appeared paler than normal and the medulla was very hyperaemic. Histological examination of the kidneys showed extensive deposition of crystals in the tubules of the cortex particularly of the corticomedullary junction. Electron microscopic observations of kidneys indicated that oxalate ion rather than calcium oxalate crystal masses caused the main renal damage and that precipitation of calcium oxalate takes place in the tubular lumena rather than the cytoplasm of tubular epithelial cells.

The principal cause of death in sheep with subacute oxalate poisoning, when the kidney damage was insufficient to cause renal failure is acute rumenoreticulitis resulting from direct injury of epithelium leading to inflammation and necrosis.

Schiefer, Hewitt and Milligan (1976) attempted unsucessfully to induce abortion in 98 pregnant ewes fed oxalic acid. The lambs killed immediately after birth had grossly normal kidneys. The authors showed that although oxalic acid ions cross the placental barrier in the doses administered to the ewes, the intoxication was not severe enough to cause intra-uterine death and subsequent abortion. Histological examination of the kidneys at the time of birth revealed oxalate crystals in most lambs from ewes fed high levels of oxalic acid throughout and/or in the second half of pregnancy and in some lambs of ewes fed low levels of oxalic acid, but not in the control animals.

Quercus sp. (Oaks)

Several hundred species of oak trees and shrubs are known and poisoning by members of the genus Quercus has been recognised for a long time but its pathogenesis is not fully understood (Smith, Jones and Hunt, 1972). Since oak bark is rich in tannic acid it has been suggested that the toxic principle in oak poisoning is tannic-acid (Mascal, 1662; Cornevin, 1893) but the usual form of oak poisoning results from the ingestion of the buds and young leaves (Boughton and Hardy, 1936). Some species are more poisonous than others and to what extent some or all oaks are more poisonous during the budding and early leafing stage, and how this may be related to a concurrent shortage of other feeds or an appetite for the oaks are unanswered questions (Smith et al, 1972).

Boughton and Hardy reported 90-95 per cent mortality in range sheep poisoned by eating the buds, green shoots and young leaves of the common shin oak (Quercus brevileba) which is abundant on the ranges of the Edward's Plateau region of West Texas. Four hundred and twenty six sheep on 38 different properties died within a period of four weeks. At necropsy the kidneys were pale and studded with pinpoint haemorrhages.

Smith (1959) noted similar gross changes in his series and in addition histological changes affecting mainly the proximal convoluted tubules. In these, numerous casts of albumin admixed with necrotic epithelial cells form a dense homogeneous mass limited by the basement membrane.

Mullins (1955) reported ten cases of acorn poisoning in approximately 200 ewes in New Zealand which were moved into an area where there was an "avenue" of oak trees. Deaths occurred from 4-10 days after access to and up to five days after removal from acorn. Post mortem examination was made on only one of the ewes. Histological examination revealed necrosis of the proximal convoluted tubules.

c) Endogenous toxaemias

Pregnancy toxaemia

Pregnancy toxaemia in sheep is a common, usually fatal metabolic disease which occurs in the last trimester of gestation and usually in ewes carrying twins or triplets. Although of considerable economic importance (McCausland, O'Hara, Herdson et al, 1974) its pathogenesis has not been elucidated despite extensive biochemical investigation (Reid, 1968). The primary predisposing cause is undernutrition in late pregnancy, with over-fat ewes being more susceptible. Stresses such as environmental change , transport, short periods of fasting and excessive heat also play a role. (Siegmund et al, 1975). Kidneys may be grossly normal (Ferris, Herdson, Dunnil et al, 1969) or slightly pale (McCausland et al, Smith et al). The main histological change in the kidney is severe diffuse lipidosis of cortical tubules (Smith et al, 1972).

Reports relating to glomerular changes in pregnancy toxaemia are controversial. Ferris et al induced toxaemia in 13 of 20 pregnant ewes by the stress of food deprivation and a change in environment late in pregnancy. All the glomeruli appeared enlarged

with capillary tufts which virtually filled Bowman's space. PAS stained sections showed focal thickening of the capillary basement membranes but there was no significant increase in cellularity. Electron microscopy revealed endothelial cell swelling, focal reduplication of glomerular basement membranes, widespread focal fusion of epithelial foot processes and extensive villous transformation of epithelial cells. The morphological changes were associated with azotaemia , proteinuria, and increased plasma renin activity. McCausland et al, were unable to show any glomerular abnormalities by light and electron microscopic examination in 11 sheep with spontaneous pregnancy toxaemia. All toxaemic sheep had significant impairment of renal function which was shown from the first signs of clinical abnormality by decreased creatinine clearance and increased serum creatinine and blood urea nitrogen. The kidneys of toxaemic sheep were slightly pale, but otherwise grossly normal and no histological lesions were found in the kidneys of toxaemic or control sheep. McCausland et al suggested that some of the sheep used by Ferris et al may have had pre-existing glomerular disease and when stressed had preferentially developed pregnancy toxaemia.

d) Enterotoxaemia

Enterotoxaemia caused by the epsilon toxin of Clostridium perfringens type D and referred to popularly as "pulpy kidney" is an important disease wherever sheep are raised. The disease has a predilection for suckling lambs and concentrate-fed weaners in feed lots. The kidneys of recently dead animals are congested, swollen and bulge a little on the cut surface. Softening occurs rapidly and within an hour or two are dark red and jelly-like.

Gardner (1971) found no detectable gross, histological or histochemical alterations in the kidneys of intoxicated lambs obtained

immediately after death. The reduction in histochemically demonstrable alkaline phosphatase and PAS staining affinity in the brush border of the renal proximal tubular epithelium in intoxicated animals was associated with nuclear degeneration and did not occur until some time had elapsed after death. It was therefore, he concluded, an autolytic lesion rather than a direct effect of epsilon toxin. Furthermore the characteristic interstitial haemorrhage in the renal cortex of intoxicated animals occurs as a post mortem change associated with the ante-mortem vascular damage. The post mortem breakdown of the already damaged capillaries allows the release of intact erythrocytes into the interstitial tissue. This lesion is considered pathognomonic of enterotoxaemia in sheep. Gardner concluded that the rapidity of onset of degenerative changes in the kidneys of intoxicated animals after death may be due to the combination of high body temperature resulting from antemortem convulsions and a direct effect of epsilon toxin upon renal tubular epithelium or interstitium.

(III) Diseases of the renal pelvis

Pyelonephritis

There are few reports of pyelonephritis in sheep. Mahafey (1941) described a pyelonephritis in a twelve months old wether. Both kidneys were swollen and the thickened capsule was adherent to the whole cortical surface. Many pale nodular areas of varying sizes were visible extending from the cortex to the medulla. In the left kidney suppuration was marked, with extensive cortical and medullary necrosis and pus in the renal pelvis. The right kidney showed similar but less severe lesions. A small quantity of pus was also present in the bladder and in the pelvic portion of the urethra. Both kidneys showed chronic inflammatory changes of variable severity throughout. The glomerular epithelium was necrotic and large numbers of neutrophils

had infiltrated Bowman's capsule and urinary spaces. Bacterial colonies were present in the glomerular tufts of some glomeruli. The convoluted tubules and ascending loops of Henle showed epithelial desquamation and necrosis. There was moderately dense neutrophilic infiltration of the tubular lumena and interstitium. In the medullary interstitium large numbers of histiocytes, lymphocytes, plasma cells and fibroblasts were present. Only a small number of intact tubules remained in the medulla. The renal pelvis showed severe fibrosis and small numbers of neutrophils. The urinary bladder was histologically normal. Pure growths of Citrobacter freundii were cultured. The author considered that the causative organism reached the kidney by the haematogenous route.

Krishna, Paliwal and Kulshrestha (1974) described suppurative pyelonephritis in two sheep in which the renal lesions were identical to that described by Mahafey. Escherichia coli serotype O6 was isolated from both. In one of the sheep suppurative pyelitis and cystitis were also present indicative of ascending infection.

(IV) Renal Neoplasms

Primary renal neoplasms in sheep have mainly been reported from survey material collected in slaughter-houses or diagnostic station material. Table 1.I lists the renal tumours diagnosed by various authors.

In addition, the kidney is a common site of metastasis of malignant lymphoma (Webster, 1966 and 1967; Johnstone and Manktelow 1978).

(V) Renal Cysts

A case of congenital cystic kidney in seven month old lamb was reported by Parameta, 1970. The animal was in good condition but with

TABLE 1.I

Ovine renal neoplasms diagnosed by various authors

Type of neoplasm	Numbers	Author
Adenoma	1	Flir (1952-53)
Carcinoma	4	Sandison and Anderson (1968)
	2	Smith and Jones (1961)
	1	Cordes and Shortridge (1971)
Carcinoma (Renal pelvis)	2	Webster (1966)
Nephroblastoma	2	Jackson (1936)
	1	Pamucku (1956)
	1	Brandly and Migaki (1963)
	2	Manktelow (1963)*
	3	Cordes and Shortridge (1971)
	1	Harcourt and Spice (1968)
	1	Feldman (1933)
	3	Flir (1952-53)
	1	Webster (1967)
	2	Smith and Jones (1961)
Haemangioendothelioma	1	Flir (1952-53)
	2	Smith and Jones (1961)

* Personal communication

moderate enlargement of the abdomen due to a very large polycystic kidney weighing 3.860 kg. The other kidney was 75 g. On dissection the large kidney showed multiple cysts throughout cut surface and contained 2,635 ml of fluid.

(VI) Urolithiasis

Urolithiasis is the formation of stony precipitates anywhere in the urinary passages and can be of considerable economic importance in sheep although the prevalence of obstructive disease is much less than that of stone formation (Jubb and Kennedy, 1970). Urethral obstruction in wethers may be common as the result of the relatively small diameter of the urethra in these animals and may occur at any site between the processus urethrae and the sphincter of the urinary bladder (Newson, 1937). The most common sites of obstruction are at the vermiform appendage or the sigmoid flexure where the urethra is narrowest (Udall and Jensen, 1958).

The gross and histological renal findings in urolithiasis are variable. The kidneys may be enlarged (Newson), hydronephrotic (Cornelius, Moulton and McGowan, 1959) wet and flabby (Weaver, 1963), only slightly oedematous and swollen (Lalov, Antonov, Popchristov et al, 1971) or grossly normal (Stacy, 1969).

Histological changes are variable in severity with inflammatory, degenerative and sometimes dystrophic change in all parts of the kidney and the presence of accumulated often calcidied material within tubules. (Lalov et al; Newson; Weaver; Cornelius et al).

The number, physical and chemical characteristics of calculi vary considerably depending largely on the dietary intake of the sheep. Silicates, oxalates and xanthine crystals occur in acid urine (Jubb et al;

Smith et al, 1972; Easterfield, Rigg, Askew et al, 1930), while calcium, triple phosphates, iron carbonate, ammonium and magnesium carbonate and phosphates form in alkaline urine (Sutherland, 1958; Jubb et al; Smith et al; Blood et al).

In grazing animals silicates and carbonates are most frequent while phosphates predominate in animals fed with excesive concentrate relative to roughage in feed lots. Sheep grazing pastures containing oestrogenic plants (Pope, 1964) or injected or implanted with oestrogens (Udall et al; Marsh, 1961; Jubb et al) may have a prevalence of fatal urinary obstruction as high as 20 per cent.

High concentrations of dietary phosphate has been reported to cause an extremely high prevalence of calculi (Lalov et al; Bushman, Emerick and Embry, 1965; Weaver; Newson).

Xanthine calculi have been related to deficiency of molybdenum on unimproved pasture (Easterfield et al). Diets low in vitamin A have been associated with urolithiasis (Hawkins et al, 1965) and may contribute by producing metaplastic changes in the urinary epithelium (Smith et al; Jubb et al).

The excesive concentration of calcium in urine in sheep with hypervitaminosis D has been suggested as a predisposing factor especially in hot climates where dehydration may occur (Blood et al, 1974).

The presence of mucopolysaccharides in urine may act as cementing agents and favour the formation of calculi (Smith et al; Blood et al). This factor is probably of importance in lambs fed heavy concentrate/low roughage rations (Udall et al).

Infections of the urinary tract are often found in association

with formation of calculi and may be present more frequently than routine cultural techniques reveal (Jubb *et al*; Smith *et al*; Blood *et al*). The contribution of infection may be direct in providing a nidus for initiation of mineralisation and indirectly by increasing urinary pH and thereby reducing the solubility of calcium and magnesium. Although attempts to produce urolithiasis experimentally by varying any one of the above factors are usually unsuccessful, interaction between several contributing factors appears to be of fundamental importance in the pathogenesis of calculogenesis (Cornelius *et al*; Swingle and Cornelius, 1962; Weaver; Lalov *et al*; Stacy; Puntriano, 1955).

CHAPTER 2

A SURVEY OF DISEASED SHEEP KIDNEYS

INTRODUCTION

Although many diseases affecting the kidneys of sheep have been described there is little information on the prevalence of these diseases in large populations of sheep. Such data can perhaps be best obtained by examination of material from slaughter-houses. While this information may not accurately represent the occurrence and pattern of acute disease outbreaks, it can provide useful information on a wide range of endemic diseases and those in chronic and resolving stages. In addition to defining the prevalence and pathology of renal disease in sheep in New Zealand, a secondary aim in this study was to determine the presence or absence of spontaneous glomerulopathies similar to those described by Den Boer (1969), Lerner and Dixon (1966a) and Langham and Hallman (1941).

MATERIALS AND METHODS

Kidneys of aged sheep condemned by members of the meat inspection staff of Borthwick's freezing works at Longburn were collected on five consecutive days in January 1980.

Kidneys from a total of 444 sheep with renal lesions were identified according to killing chain. On the first day of collection kidneys from one chain only were examined, but collection was extended to include two chains on the second day and on days three, four and five to include all three chains. Sheep killed on one chain were further identified as to farm of origin. Kidneys discarded because of maceration, contamination, poorly stripped capsule or for reasons other than antemortem change were not recorded in this study.

The weight and gross pathological features of each kidney were noted and where appropriate, the lesions were photographed. Statistical

analysis of the variation of numbers of kidney lesions occurring in the different lines, chains and daily totals of sheep killed and between weights of kidneys collected was made using the students t test (Sokal and Rohlf, 1969).

Selected pieces of tissue from 181 kidneys representing the various lesions seen at gross examination were fixed in 10 per cent formol saline solution for histopathological examination. Grossly normal kidneys from 5 young lambs and 45 old ewes were examined histologically as controls. Paraffin sections prepared by routine techniques were stained with haematoxylin and eosin (HE) and various other stains selected as subsequent examination indicated. The latter stains included periodic acid Schiff (PAS) for alterations in the glomerular basement membrane structure, van Gieson and Gomori's trichrome methods for collagen, Warthin Starry silver stain for leptospirae, Perls' Prussian blue method for haemosiderin, Congo red for amyloid, von Kossa's method for calcium and Giemsa and Ziehl-Neelsen techniques for microorganisms. (Luna, 1968)

The histological lesions were recorded and graded according to a format (appendix I) which identified the anatomical location, pattern of distribution, tissue changes and degree of severity of the lesions.

RESULTS

The numbers of kidneys condemned per day and from individual chains over the collection period are shown in Table 2.I and are compared with the total numbers of sheep killed. The numbers of kidneys condemned on the basis of individual "lines" of sheep killed on chain A on each day is shown on Table 2.II.

The prevalence of lesions varied within lines (0-18.75 per cent), chains (1.22-7.58 per cent) and daily recordings (2.49-7.58 per cent) with an overall prevalence of 3.18 per cent of 13,978 sheep slaughtered.

TABLE 2.I

The prevalence of sheep with renal lesions in a survey of sheep killed in a five day period

Day	Chain No.	Prevalence/Chain[1]	Prevalence/Day[2]
1	A	$\frac{55}{726}$ (7.58)	$\frac{55}{726}$ (7.58)
2	A	$\frac{50}{1453}$ (3.14)	$\frac{80}{2762}$ (2.90)
	B	$\frac{30}{1309}$ (2.29)	
3	A	$\frac{64}{1406}$ (4.55)	$\frac{103}{3928}$ (2.62)
	B	$\frac{16}{1307}$ (1.89)	
	C	$\frac{23}{1215}$ (1.89)	
4	A	$\frac{29}{1201}$ (2.41)	$\frac{91}{3661}$ (2.49)
	B	$\frac{40}{1324}$ (3.02)	
	C	$\frac{22}{1146}$ (1.92)	
5	A	$\frac{19}{1054}$ (1.80)	$\frac{115}{2901}$ (3.96)
	B	$\frac{67}{1005}$ (6.67)	
	C	$\frac{29}{842}$ (3.44)	
Overall Prevalence[3]			$\frac{444}{13978}$ (3.18)

(1) $\frac{\text{Number of sheep with renal lesions/chain}}{\text{Total number of sheep examined/chain}}$ (percentage of sheep with renal lesions/chain)

(2) $\frac{\text{Number of sheep with renal lesions/day}}{\text{Total number of sheep examined/day}}$ (percentage of sheep with renal lesions/day)

(3) $\frac{\text{Total number of sheep with renal lesions}}{\text{Total number of sheep examined}}$ (overall percentage of sheep with renal lesions)

TABLE 2.II

Variation between lines of sheep with renal lesions

in sheep killed on chain "A" in a five day period

	Day 1	Day 2	Day 3	Day 4	Day 5
Line 1 $\frac{A}{B}$ (C)	$\frac{0}{9}$ (0)	$\frac{10}{399}$ (2.51)	$\frac{5}{195}$ (2.56)	$\frac{10}{450}$ (2.22)	$\frac{3}{200}$ (1.50)
Line 2 $\frac{A}{B}$ (C)	$\frac{0}{1}$ (0)	$\frac{0}{14}$ (0)	$\frac{2}{102}$ (1.96)	$\frac{0}{30}$ (0)	$\frac{5}{329}$ (1.52)
Line 3 $\frac{A}{B}$ (C)	$\frac{25}{157}$ (15.92)	$\frac{6}{108}$ (5.56)	$\frac{19}{311}$ (6.11)	$\frac{2}{121}$ (1.65)	$\frac{5}{16 1}$ (3.11)
Line 4 $\frac{A}{B}$ (C)	$\frac{3}{164}$ (1.83)	$\frac{2}{77}$ (2.60)	$\frac{0}{33}$ (0)	$\frac{6}{32}$ (18.75)	$\frac{0}{46}$ (0)
Line 5 $\frac{A}{B}$ (C)	$\frac{11}{290}$ (3.79)	$\frac{1}{70}$ (1.43)	$\frac{2}{84}$ (2.38)	$\frac{3}{114}$ (2.63)	$\frac{6}{318}$ (1.89)
Line 6 $\frac{A}{B}$ (C)	$\frac{13}{75}$ (17.33)	$\frac{5}{264}$ (1.89)	$\frac{0}{8}$ (0)	$\frac{1}{54}$ (1.85)	
Line 7 $\frac{A}{B}$ (C)	$\frac{3}{30}$ (10.0)	$\frac{14}{165}$ (8.48)	$\frac{4}{81}$ (4.94)	$\frac{4}{33}$ (12.12)	
Line 8 $\frac{A}{B}$ (C)		$\frac{3}{101}$ (2.97)	$\frac{13}{230}$ (5.65)	$\frac{3}{367}$ (0.82)	
Line 9 $\frac{A}{B}$ (C)		$\frac{5}{69}$ (7.25)	$\frac{9}{172}$ (5.23)		
Line 10 $\frac{A}{B}$ (C)		$\frac{6}{226}$ (2.65)	$\frac{10}{190}$ (5.26)		

A. Number of sheep with renal lesions
B. Number of sheep slaughtered
C. Percentage of sheep with renal lesions

Analysis of variance in each of these categories indicated that the daily differences were not significant at a 95 per cent confidence level. Only 2.0 per cent of the overall variation between lines was due to differences between sampling days. A high prevalence of a particular type of gross lesion associated with a particular line of sheep was not observed.

Bilateral lesions were present in 386 sheep slaughtered (86.9 per cent) and unilateral lesions in 58 sheep (13.1 per cent).

Weight of kidneys

The weights of condemned kidneys (Figure 2.1) ranged from 14.5 to 258 g. Relatively few kidneys were in the range of 20-50 g or more than 100 g ranges. The mean weight was 76.14 g.

The mean weights of a total of 3,700 and 3,100 grossly normal mature sheep kidneys sampled at weekly intervals throughout the 1976-77 and 1977-78 killing seasons at the Borthwick's freezing works, Longburn was 76.14 g and 74.32 g respectively. (Production Manager, pers. comm.).

GROSS PATHOLOGY

The 830 kidneys were divided into groups according to the major pathological change using the criteria adopted by meat inspection staff, i.e. those of abnormal shape, size, colour, spots and streaks, scars, cysts and nodules (Figure 2.2). Three hundred eighty two kidneys contained more than one lesion and these are noted as additional lesions in Tables 2.III and 2.IV.

Lesions in 34 misshapen or abnormally sized kidneys are shown in Table 2.IV.

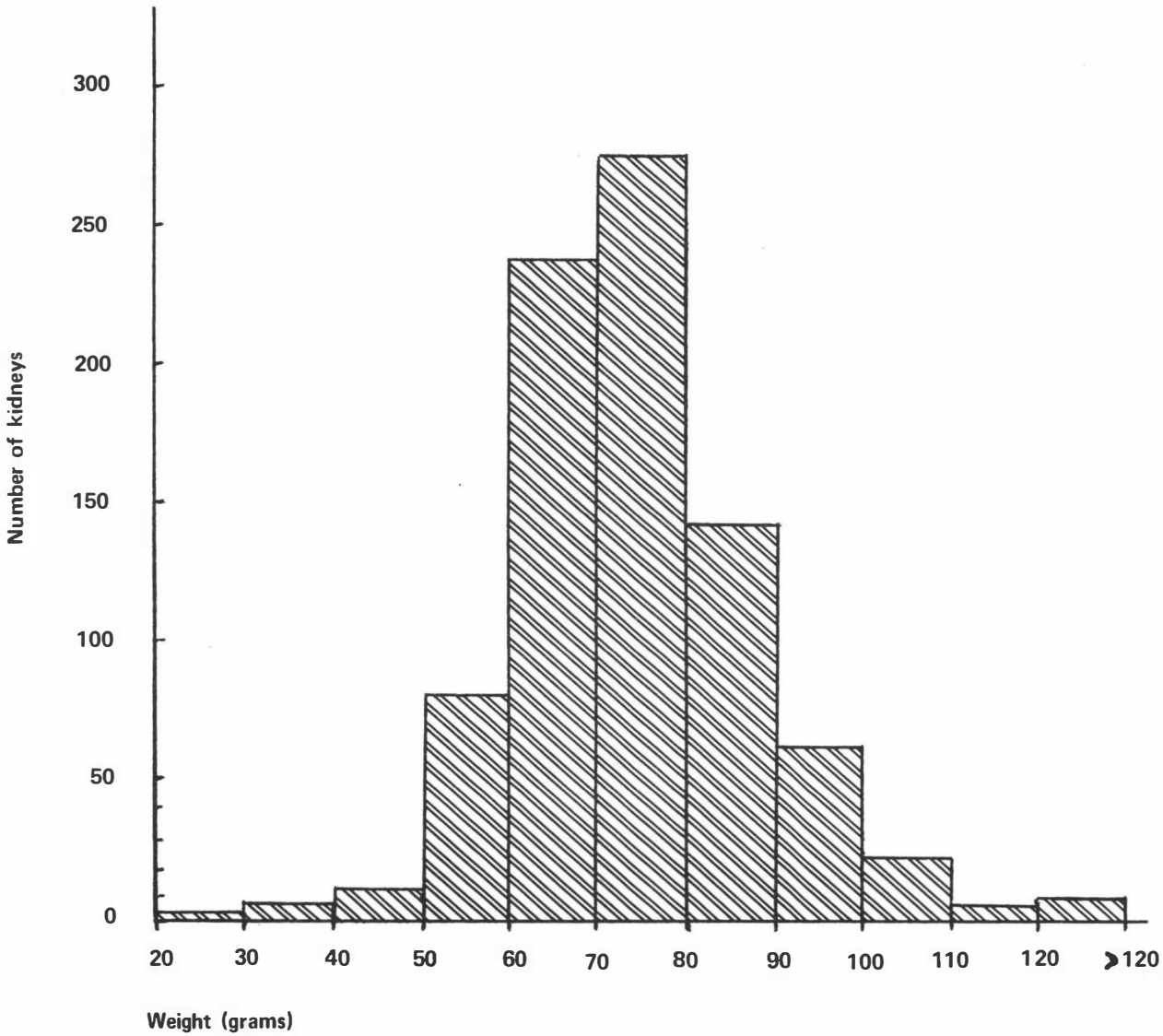

Figure 2.1

Histogram showing the distribution in weight of 830 kidneys collected from 444 sheep with renal lesions.

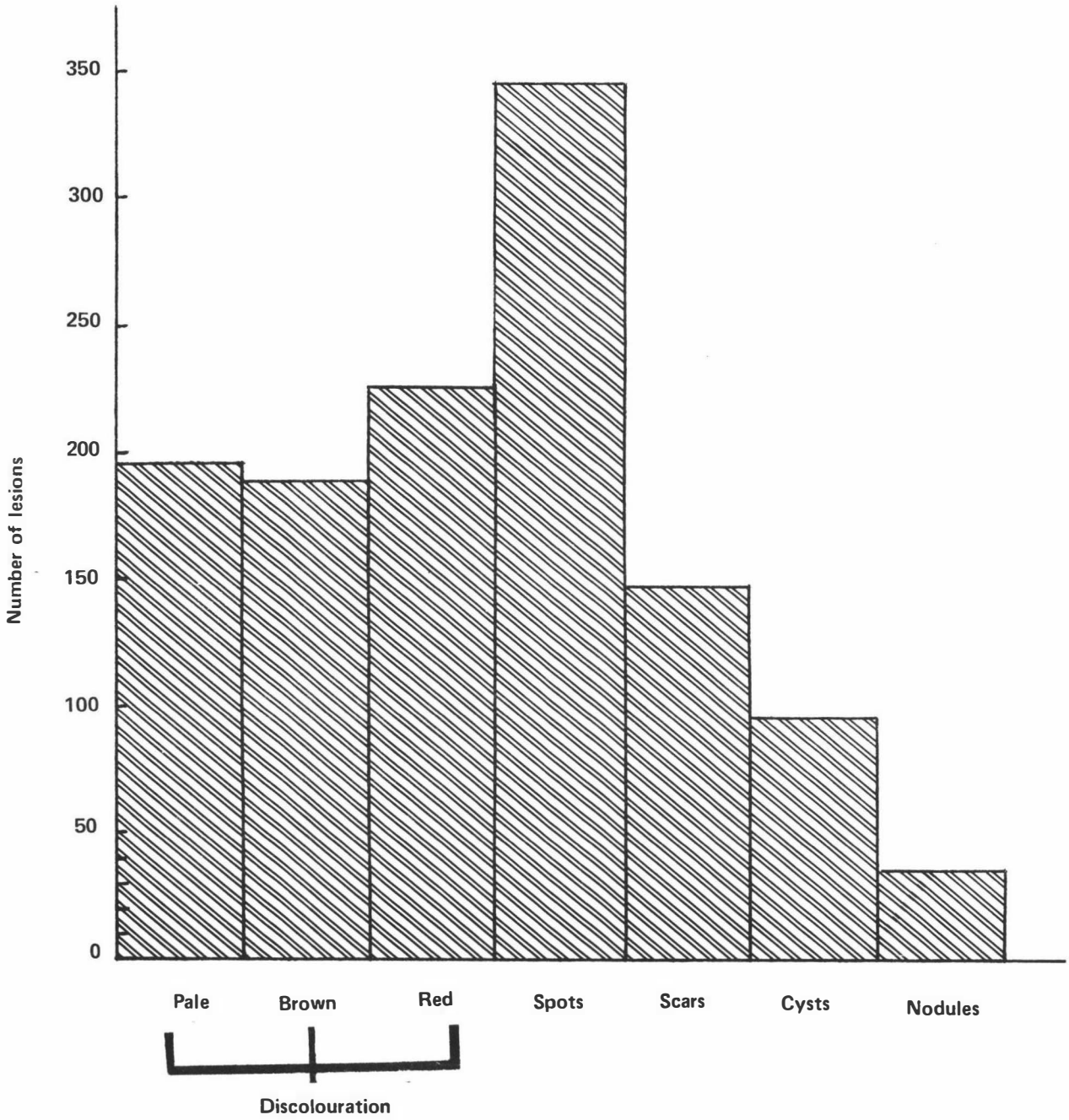

Figure 2.2

Histogram showing the gross pathological features of 1212 lesions recorded in 830 diseased kidneys.

TABLE 2.III

Prevalence of gross lesions from 830 kidneys

Additional lesions	Major gross pathological lesion						
	Discolouration			Spots	Scars	Cysts	Nodules
	Pale	Red	Brown				
Pale discolouration		10	-	3	-	6	4
Brown discolouration	-	-		-	-	-	5
Congestion*	50		20	22	7	-	-
Spots and Streaks	52	35	58		9	3	4
Scars	10	3	18	8		1	1
Cysts	18	4	13	10	6		2
Nodules	-	-	-	-	-	-	
No other lesions	44	68	70	145	85	27	9
Total number of kidneys	174	120	179	188	107	37	25

* Patchy congestion

TABLE 2.IV

Lesions present in 34 misshapen, large or small sized kidneys

Additional lesions	Shape	Increased size	Decreased size
Pale discolouration	3	9	-
Brown discolouration	-	2	-
Spots and Streaks	1	-	-
Congestion	3	1	-
Scars	2	3	6
Cysts	1	3	-
Total	10	18	6

(1) Abnormal shape

All of the ten misshapen kidneys were dumbbell shaped with narrow central areas and round poles (figure 2.3). In two kidneys the narrowing had resulted from extensive scarring in the central area. The other eight kidneys were associated with pallor (figure 2.5), congestion, spots or cysts.

(2) Increased size

The weight of the enlarged kidneys ranged from 100-258 g. Nine were uniformly pale and swollen and are described in section 4 below. The other nine kidneys had additional lesions of brown discolouration, scars, cysts or congestion. (Table 2.IV).

(3) Reduced size

Six kidneys weighing from 18-40 g were condemned because of small size. These were grey-white and firm with strongly adherent capsules due to diffuse scarring (Figure 2.4).

(4) Discolouration

a) Pale. One hundred seventy four kidneys were paler than normal, showed slight to severe parenchymal swelling and bulged from the cut surface (Figure 2.5). In only four sheep was the change unilateral. Fourty four kidneys in this category had no other lesions, while 50 also showed patchy areas of congestion and others had additional minor lesions of spots, scars and cysts.

b) Red. Patchy or diffuse areas of bright red discolouration were present in 120 kidneys (Figures 2.6 and 2.7). One hundred and eighteen were from sheep with lesions in both kidneys. Additional lesions recorded with these kidneys were spots, scars, and cysts and small pale patches.

c) Brown. One hundred and seventy nine kidneys showed more or less diffuse, light yellow-brown to chocolate-brown discolouration (Figure 2.8).

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Figures $\frac{2.3a \ / \ 2.3b}{2.4}$

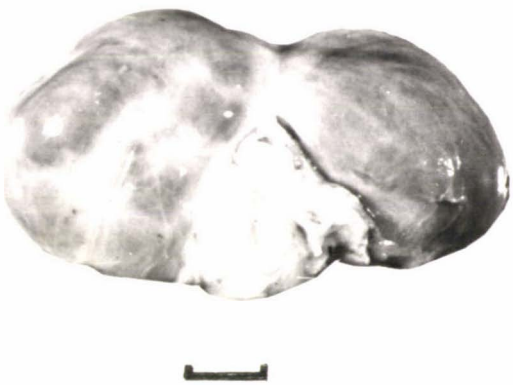
Figure 2.3: Misshapen kidney (No. 234).

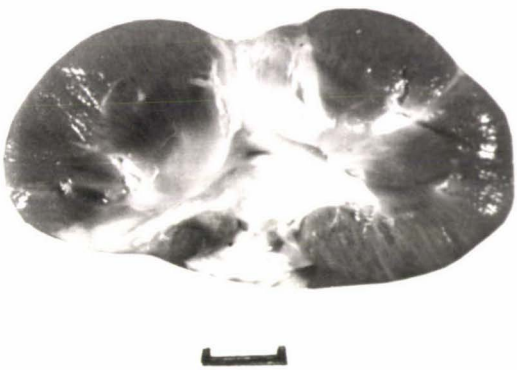
a) Capsular surface showing dumbbell shape caused by severe scarring in central area. Less extensive scarring is present over the capsular surface of the poles.

b) The cut surface of the same kidney showing the scarred tissue extending from capsule to pelvis.

Scale bar = 1 cm

Figure 2.4: Pale shrunken kidney (No. 304). The reduction in size is due to diffuse scarring of the renal parenchyma. The incised surface of this kidney is shown in figure 2.14.





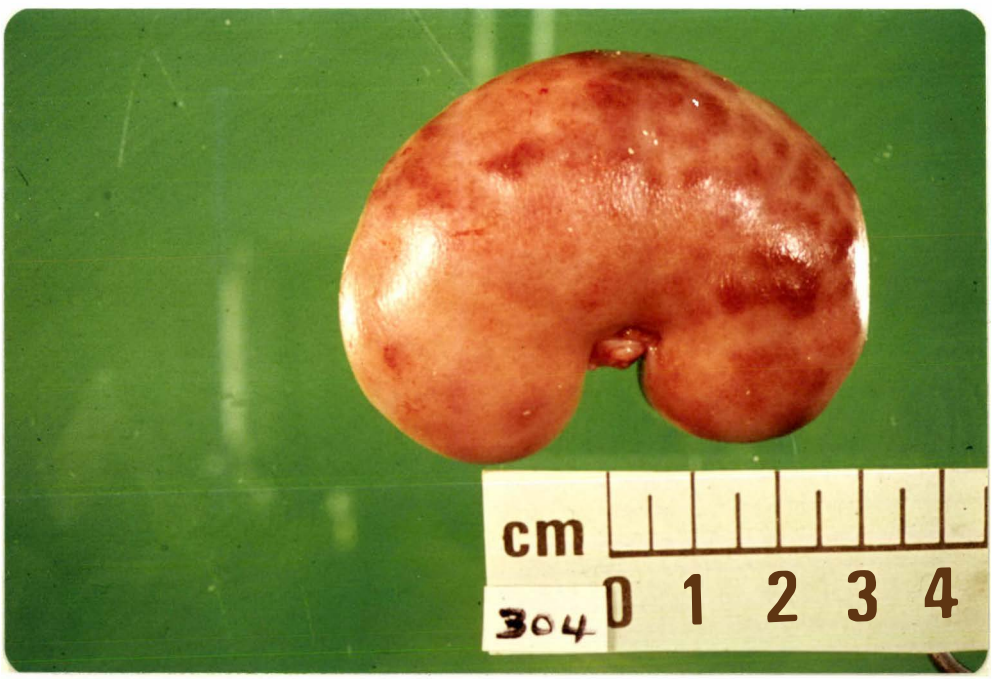
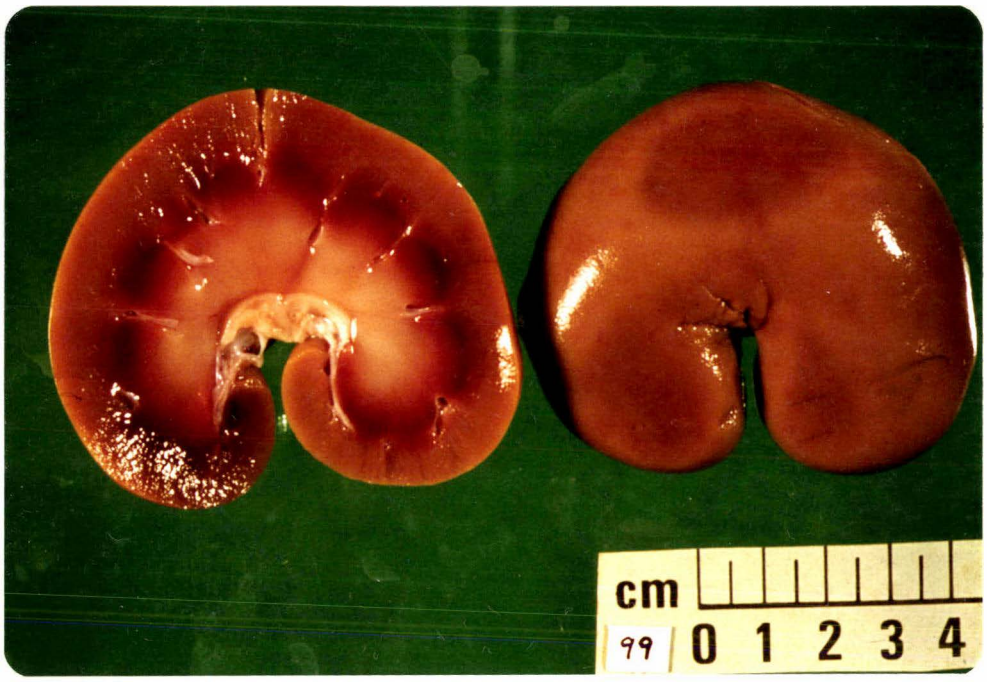

cm
0 1 2 3 4
304

Figure 2.5: Pale swollen kidney (No. 99). Note the pallor of the outer cortex contrasting with the congested cortico-medullary region.

Figure 2.6: Patchy red discolouration of kidneys (No. 5) due to congestion. The change is less severe and restricted to the right pole in the kidney on the right.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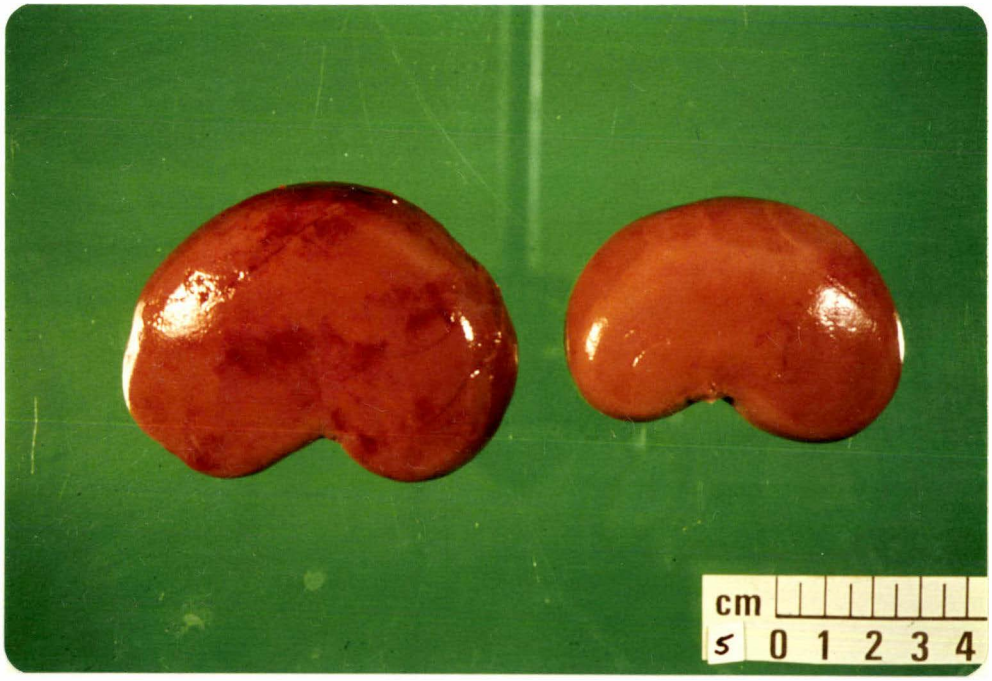
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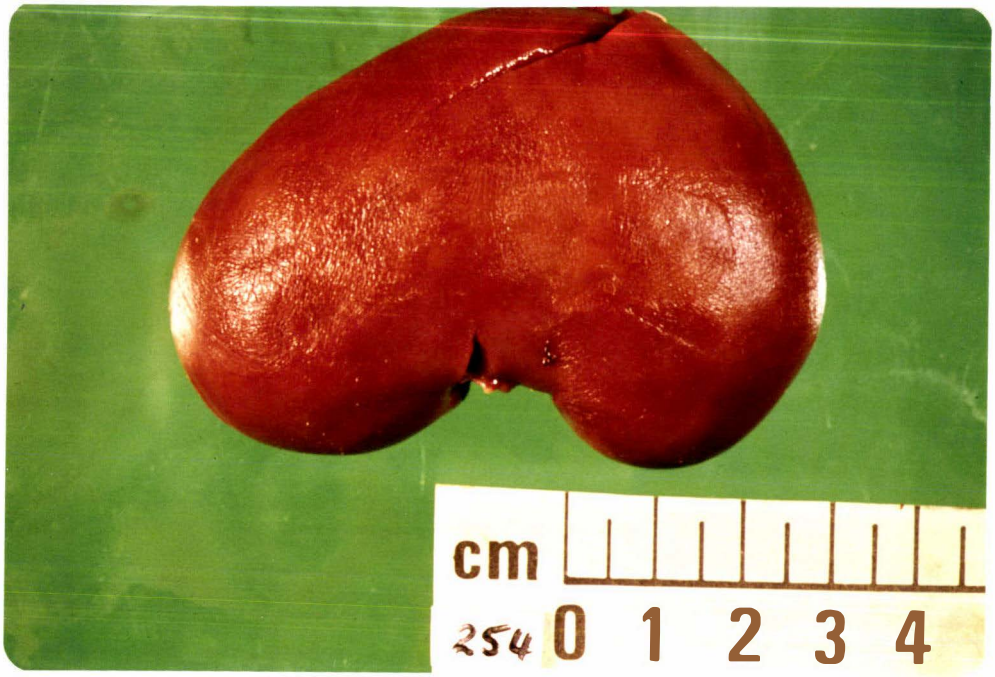
Figure 2.7: Diffuse red discolouration of a kidney (No. 254) due to congestion.

Figure 2.8: Diffuse dark brown discolouration of a kidney (No. 201) due to haemosiderosis.


cm
254 0 1 2 3 4


NORMAL
201
cm
0 1 2 3 4

Figure 2.9a: Dark brown discolouration of kidneys (No. 133) distributed in scarred and depressed areas of renal cortex. The pale raised areas are the remaining functional kidney tissue.

Figure 2.9b: Cut section of kidneys showing radial brown streaking of scar tissue extending from capsular surface to medulla.

In only three sheep were the lesions unilateral. Additional lesions encountered were patchy congestion, spots, cysts and scars. In two of the kidneys the discolouration was restricted to extensively scarred areas and on cut surface these showed a segmental distribution extending from capsular surface to medulla (Figure 2.9).

(5) Spots and streaks

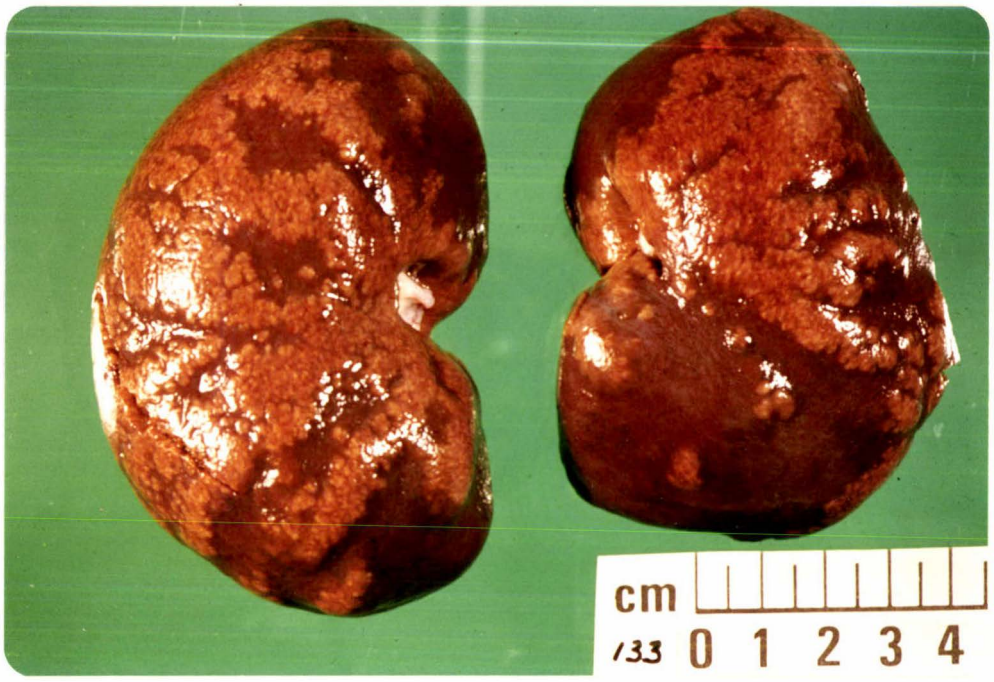
The capsular surface of 188 kidneys had multiple, roughly circular, 0.1-0.3 cm diameter white spots in the superficial cortical tissue. In ten sheep the lesions were unilateral. On the cut surface they extended 0.3-1.0 cm into the cortical parenchyma, forming ill-defined streaks in the outer cortex. In some there was extension into inner cortical parenchyma (Figures 2.10,11). Other lesions including patchy congestion, scars and cysts were occasionally present.

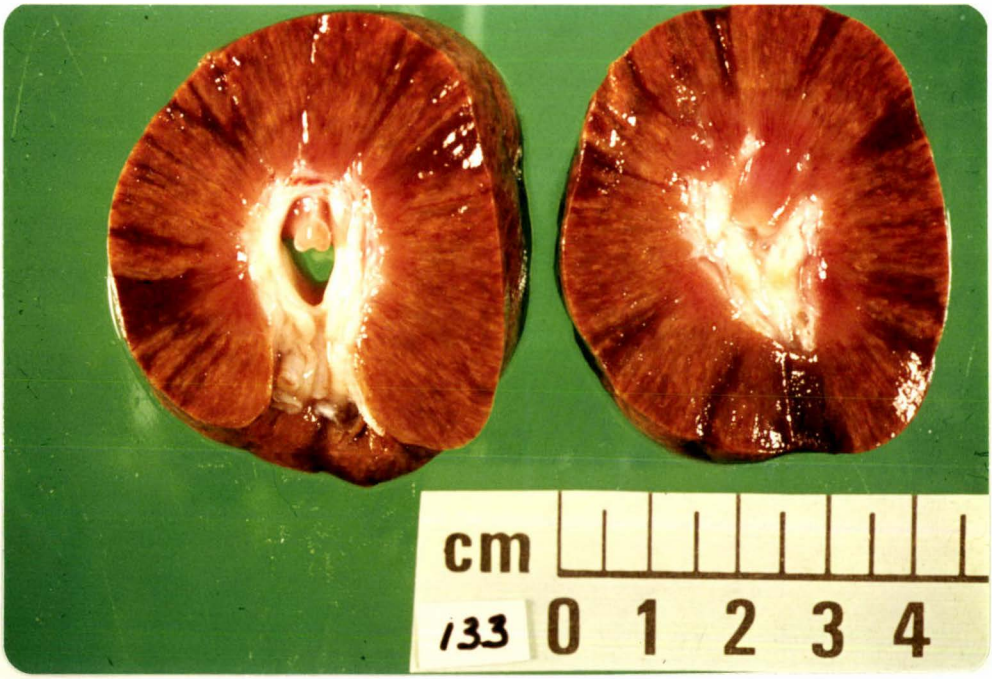
(6) Scars

Scarring was the major pathological finding in 107 kidneys of which 19 were from sheep with unilateral renal lesions. On the capsular surface most of these appeared as 0.2-0.5 cm diameter single or multiple, linear or roughly circular white depressions (Figure 2.12). The remainder were larger, 2.0-3.0 cm diameter or diffuse lesions (Figure 2.13). The small linear scars were mainly confined to the cortex (Figure 2.12b) and extended into the medulla infrequently. On the cut surface the larger scars were wedge-shaped. Approximately half of these affected the cortical tissue only with the remainder extending from cortex to renal pelvis (Figure 2.14). Additional lesions included patchy congestion, spots and cysts.

(7) Cysts

Cysts were the most prominent gross pathological feature in 37 kidneys, 11 of which were in unilaterally affected sheep. The kidneys


cm
133 0 1 2 3 4


cm
133 0 1 2 3 4

Following page 38

Figures 2.10a / 2.10b

2.11a / 2.11b

Figure 2.10: a) Severe diffuse spotting of subcapsular cortical tissue (kidney No. 119).

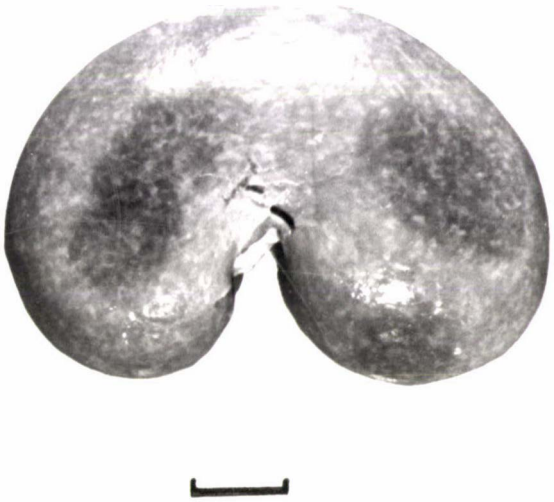
b) The same kidney on cut section showing ill-defined, white streaks extending radially into inner cortical parenchyma.

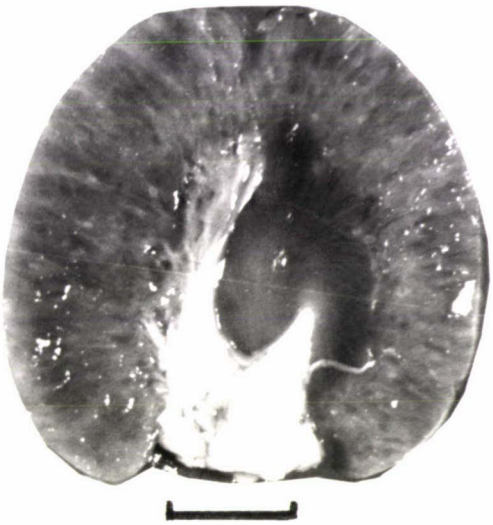
Scale bar = 1 cm

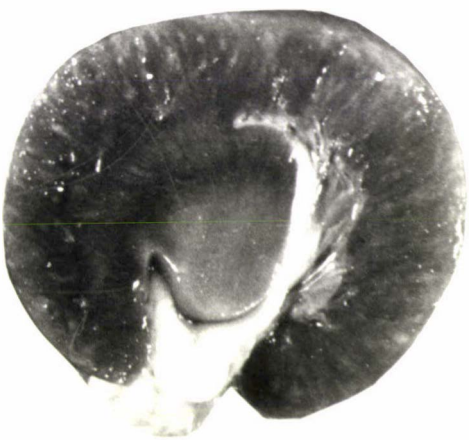
Figure 2.11: a) Moderately severe white spotting of the subcapsular cortical tissue (kidney No. 2).

b) The cut section of the same kidney showing ill-defined white streaks which are most prominent in the outer cortical parenchyma.

Scale bar = 1 cm







Figures 2.12a / 2.12b

2.13 / 2.14

Figure 2.12: a) Multiple, slightly depressed ill-defined scars in the subcapsular tissue (kidney No.4).

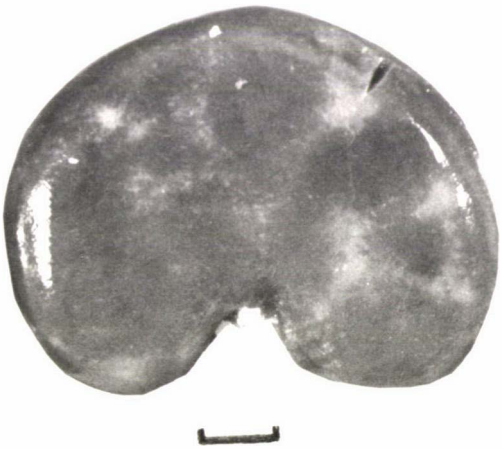
b) Cut section of the same kidney showing poorly defined linear white bands of scar tissue in the cortex.

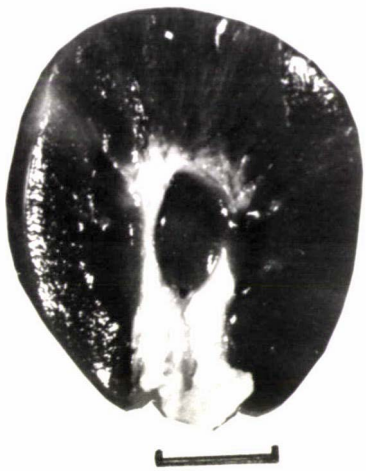
Scale bar = 1cm

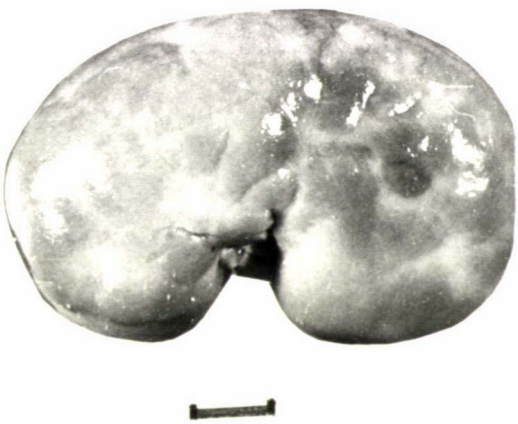
Figure 2.13: Extensive scarring of the subcapsular tissue (kidney No.130).

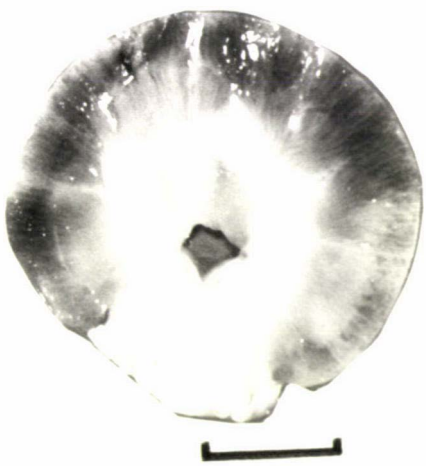
Figure 2.14: Cut section of a small scarred kidney (No.304) showing diffuse fibrosis affecting all areas of the kidney. Note the distorted renal papilla. The capsular surface of this kidney is shown in figure 2.4.

Scale bar = 1 cm









contained 5-10 fluid filled cysts ranging in size from 0.1-1.0 cm diameter. They appeared as well defined dark spots when visible on the capsular surface (Figure 2.15a) and on cut surface as well defined dilatations of renal cortical (Figure 2.15b) and occasionally medullary tissue.

The cortex of four kidneys contained multilobulated thickly encapsulated cysts of up to 4 cm diameter which were identified by the presence of scolices in aspirated content as hydatid cysts of Echinococcus granulosus (Figure 16).

Additional lesions detected in the cystic kidneys were spots and scars.

(8) Nodules

a) Abscesses. Well encapsulated abscesses with varying fibrous organization of caseous central material were present in seven kidneys. Three of the affected sheep had unilateral lesions. The abscesses ranged in size from 1 to 3 cm diameter and when close to the capsule caused bulging at the surface (Figure 2.18). Two of lesions were found in severely scarred kidneys (Figure 2.17).

b) Neoplasms. Three neoplasms were found. Two were solitary, well defined, encapsulated, spherical cortical masses of 8.0 and 1.0 cm diameter respectively. The larger tumour occupied the entire left pole of the kidney and was composed of lobules of grey-white tissue in which areas of necrosis and haemorrhage were prominent (Figure 2.19). The smaller tumour was embedded within the cortex and on the cut surface was white, lobulated and soft.

The other neoplasm consisted of four, well defined 0.3-4.0 cm, white, firm, spherical or discoid masses in cortical and hilar areas of kidney. The largest 2.0 x 4.0 cm discoid mass extended from one pole of the

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Figures 2.15a / 2.15b
2.16a / 2.16b

Figure 2.15: Congenital cysts.

a) Capsular surface showing numerous well defined cortical depressions overlyingthe cystic parenchyma (kidney No. 55).

b) Cut section of the same kidney showing four well defined cortical cysts.

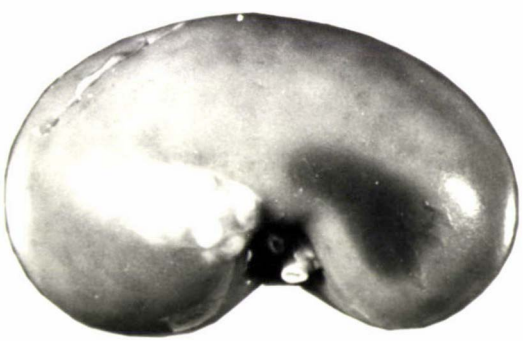
Scale bar = 1 cm

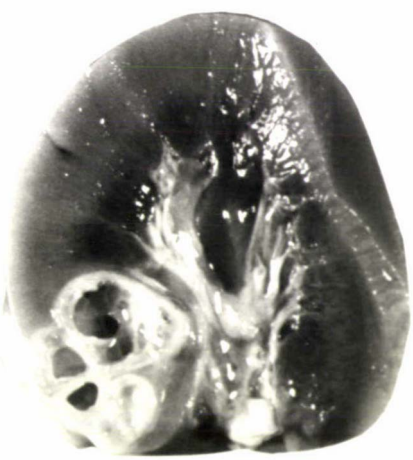
Figure 2.16: Hydatid cysts in kidney (No. 198).

a) Cyst of Echinococcus granulosus in parahilar cortical tissue.

b) The cut section showing the multiloculated cyst.

Scale bar = 1 cm





Figures 2.17a / 2.17b
2.18a / 2.18b

Figure 2.17: Renal abscessation (No. 59).

a) The cortical surface of a kidney containing an organized abscess in the left pole. Note the deformity caused by depressed, scarred tissue in paracentral areas.

b) Cut section of the same kidney showing the organizing abscess (large arrow) and scars (small arrow) extending from the cortex to the pelvis.

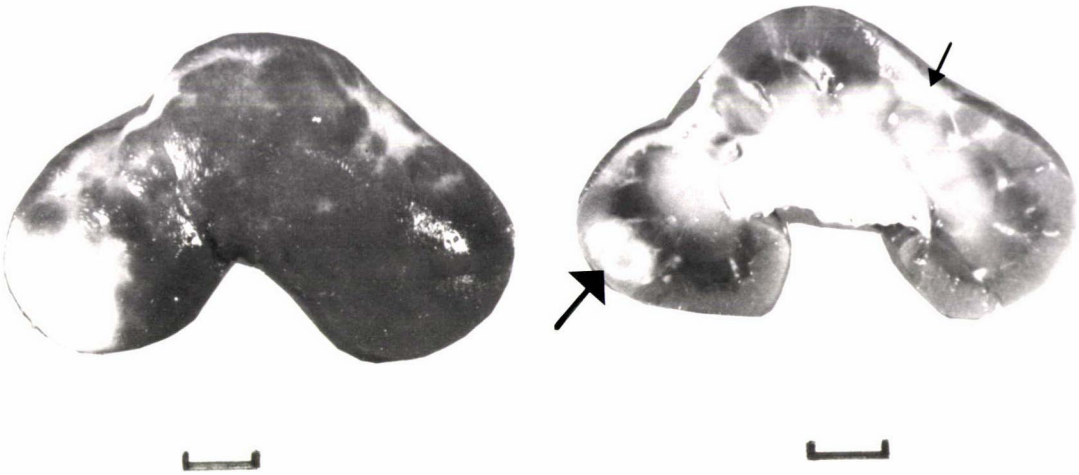
Scale bar = 1 cm

Figure 2.18: Renal abscessation (No.200).

a) The kidney contains an encapsulated caseous abscess bulging from the capsular surface.

b) The cut section of the same kidney.

Scale bar = 1 cm



Figures 2.19a / 2.19b
2.20a / 2.20b

Figure 2.19: Renal carcinoma (No. 237).

a) Distortion of the left pole of the kidney by a renal carcinoma.

b) Cut section of the kidney showing the slightly encapsulated lobulated tumour with extensive areas of necrosis and haemorrhage.

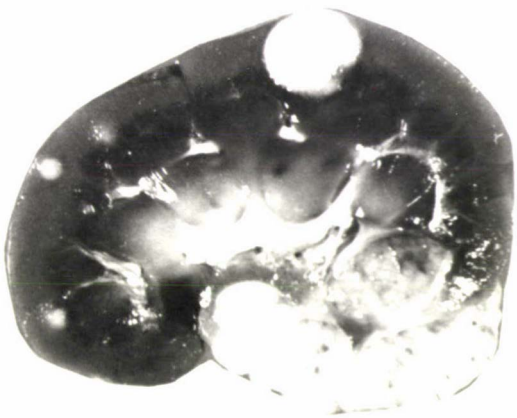
Scale bar = 1 cm

Figure 2.20: Renal lymphoma (No. 236).

a) Distortion of a kidney by a lymphoma occupying the left polar and hilar zones.

b) The incised surface showing additional focal tumour masses in the cortical tissue.

Scale bar = 1 cm



kidney into the renal hilus. The capsule stripped with difficulty (Figure 2.20).

c) Nodules of uncertain aetiology. Fourteen kidneys contained single, firm, nodular, slightly raised, white, well defined, cortical lesions ranging from 0.5--3.0 cm diameter (Figure 2.22). On cut surface they were spherical and composed of hard, sometimes gritty tissue. Two showed concentric lamination (Figure 2.21).

(9) Infarct

One kidney was massively swollen, diffusely haemorrhagic and necrotic. An organizing thrombus was present in the renal vein (Figure 2.23).

HISTOPATHOLOGY

The number of kidneys examined histologically according to the various types of gross pathological lesions are shown on Table 2.V.

Major gross pathological lesion	Number of kidneys examined histologically
1) Discolouration	
a) pale	9
b) red	14
c) brown	19
2) Spots and Streaks	55
3) Scars	51
4) Cysts	11
5) Nodules	
a) abscesses	4
b) neoplasms	3
c) of uncertain aetiology	14
6) Infarction	1

TABLE 2.V Number of kidneys examined histologically according to gross pathological classification of lesions.

Following page 40

Figures 2.21a / 2.21b

2.22a / 2.22b

Figure 2.21: Nodule of undetermined cause (kidney No. 12).

a) A fibrous nodule elevated slightly above the capsular surface.

b) Note the concentric lamination of the lesion upon sectioning.

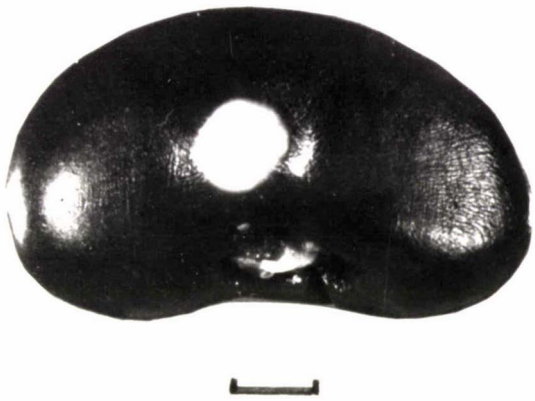
Scale bar = 1 cm

Figure 2.22: Chronic parasitic nodule (kidney No. 20).

a) Appearance on capsular surface.

b) On incision the well defined cortical nodule was hard and contained gritty material.

Scale bar = 1 cm



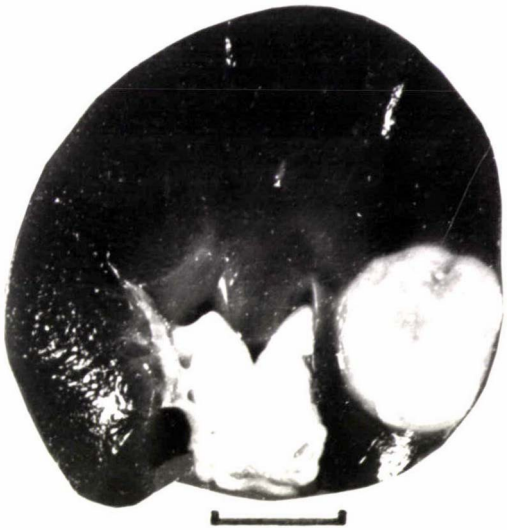
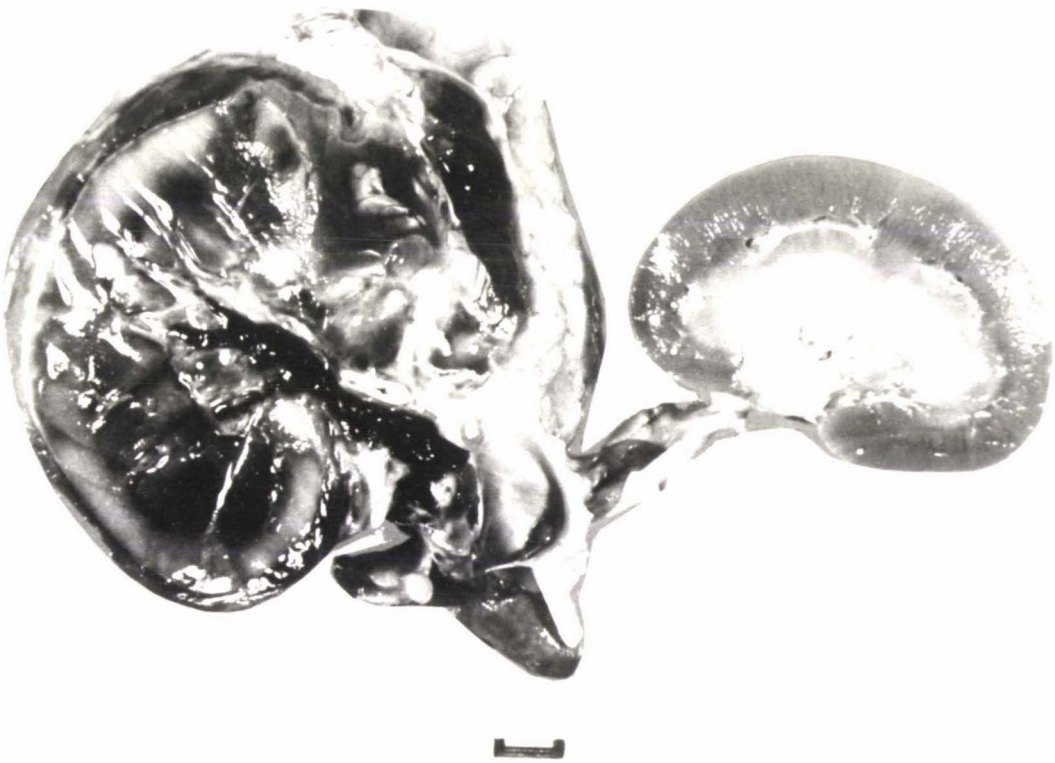


Figure 2.23: Massive renal infarct (No. 249). The kidney on left is swollen, diffusely haemorrhagic and necrotic. An organizing thrombus is present in the renal vein.
The right kidney is normal.

Scale bar = 1 cm

Figure 2.23: Massive renal infarct (No. 249). The kidney on left is swollen, diffusely haemorrhagic and necrotic. An organizing thrombus is present in the renal vein.
The right kidney is normal.

Scale bar = 1 cm



(1) Discolouration

a) Pale

Two of the nine pale kidneys examined were from the same sheep. All showed lesions characterized by cytoplasmic swelling and fatty vacuolation of epithelial cells which in some cases progressed to necrosis (Figure 2.24).

In five of the kidneys the lesion involved all zones of tubules within the cortex, but were most marked in proximal and distal convoluted tubules. In the remaining four the proximal and distal convoluted tubules only were affected. Hyaline and granular casts were present in tubules of all kidneys and in addition one kidney contained cellular casts of lymphocytes, plasma cells and neutrophils. This kidney also had multiple focal, inflammatory lesions in the cortical and medullary interstitium with fibroplasia and moderately dense accumulations of lymphocytes and plasma cells and fewer neutrophils.

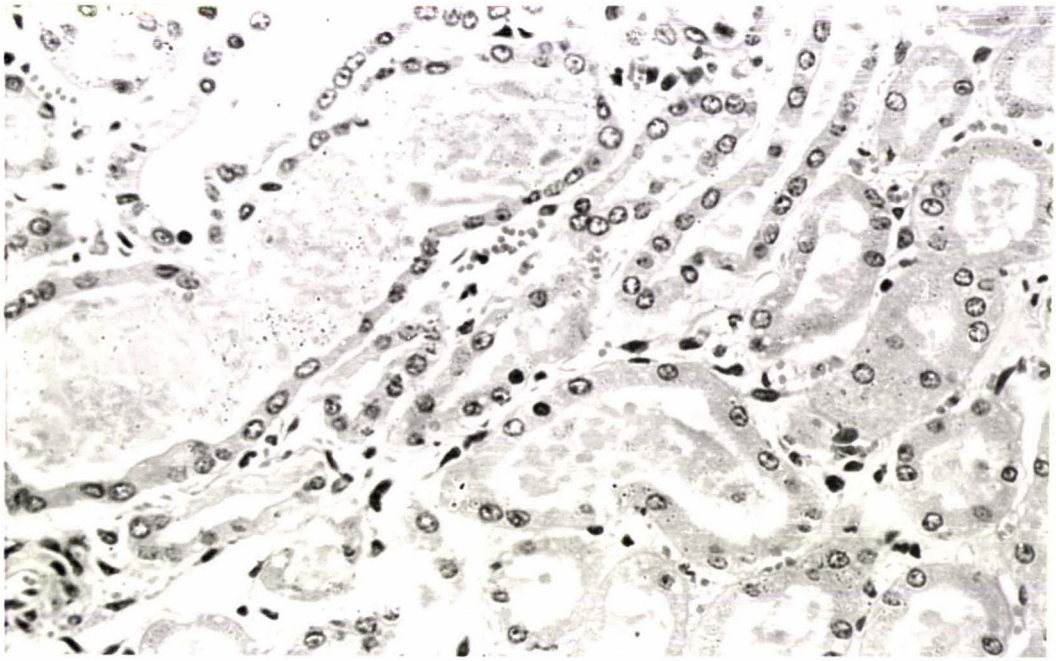
In the remaining four kidneys the nephrosis was less severe (Figure 2.25) and of focal rather than diffuse distribution. In one kidney glomerular lesions of mild panglomerular thickening of the capillary basement membrane and Bowman's capsule were found in the areas of nephrosis. In another, a focal inflammatory lesion was present in the cortical interstitium in which lymphocytes and plasma cells were the predominant cell types. Mild congestion of blood vessels in the cortical and medullary tissues was seen in three kidneys.

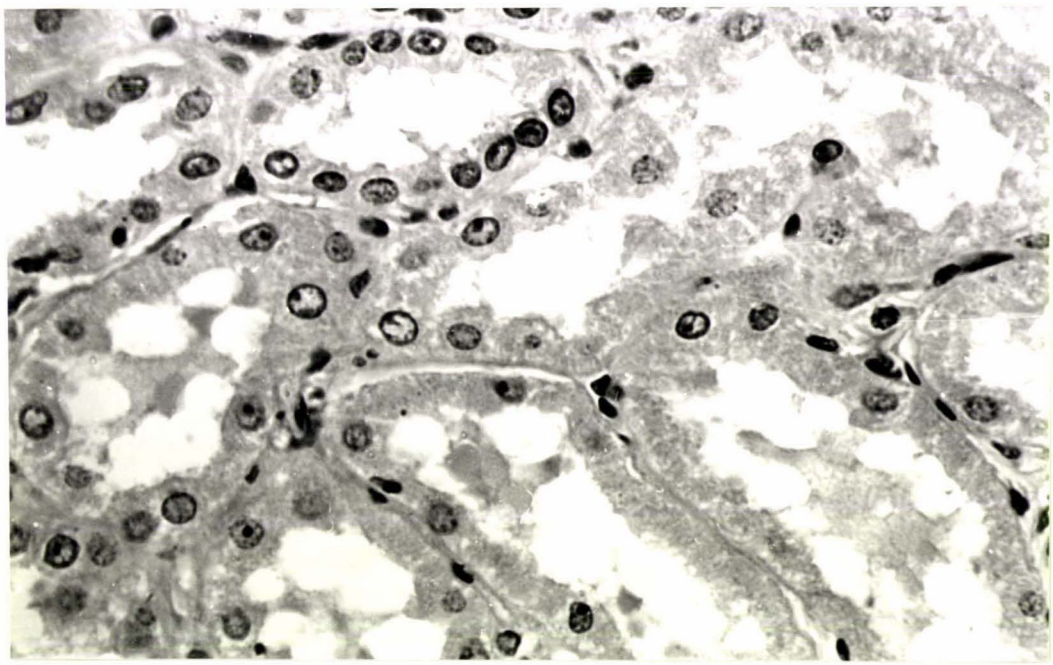
b) Red

Fourteen kidneys showed multiple focal areas of cortical congestion. In all but two there was also moderately intense diffuse congestion of blood vessels in renal medulla. In three of the kidneys this was accompanied by a mild multifocal cortical interstitial accumulation of lymphocytes and plasma cells. Calcified and granular cast were present

Figure 2.24: Nephrosis. Moderately severe scattered degenerative changes in epithelial cells of proximal convoluted tubules. Note pyknotic nuclei and cell loss. The dilated tubules contain hyaline granular casts.
HE x 320

Figure 2.25: Nephrosis. Lesions are similar but less severe than those in figure 2.24.
HE x 400





in the collecting tubules of one kidney. Mild, segmental, membranous thickening of capillaries and Bowman's capsule were present in glomeruli of another.

c) Brown

The 19 kidneys with brown discolouration examined were selected from 17 sheep. The distribution and nature of lesions are shown in Table 2.VI.

Twenty of the 22 tubular lesions affected cortical tubules and were characterized by mild diffuse epithelial cell swelling and vacuolation with mild to very severe intracytoplasmic accumulation of haemosiderin which was well demonstrated using Perl's stain for iron (Figures 2.26 and 2.27). Regeneration of damaged epithelium was seen in many areas of haemosiderin deposition.

Six kidneys with diffuse haemosiderosis had inflammatory lesions of radial distribution which were characterized by accumulations of lymphocytes, plasma cells and fibroblasts in the interstitium of cortex and/or medulla (Figures 2.28 and 2.29). In both kidneys of one sheep the inflammatory changes extended into the renal pelvis. Two other kidneys showed diffuse congestion of both cortex and medulla.

Membranous or sclerotic segmental or panglomerular lesions of varying severity occurred in capillary basement membranes and Bowman's capsule of glomeruli in ten haemosiderotic kidneys.

(2) Spots and Streaks

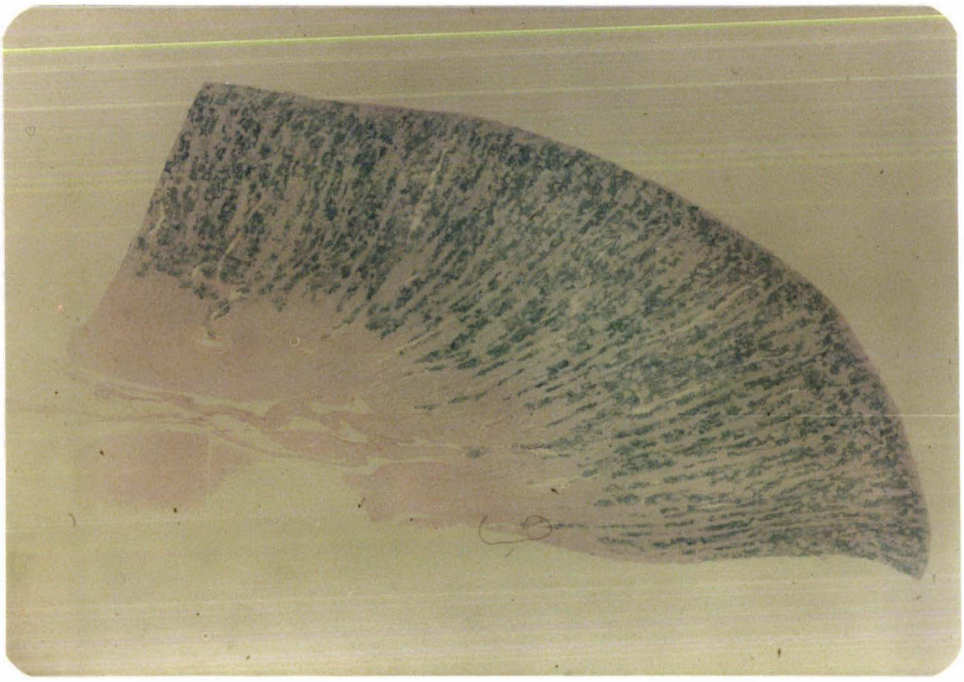
Of the 55 kidneys with spots, 12 were obtained from bilaterally affected kidneys of six sheep. The nature, site, distribution and numbers of lesions are summarized in Table 2.VII.

The main histopathological finding in all kidneys was a mild to

Figure 2.26: Haemosiderosis. "Whole mount" section of kidney stained by Perls' method for iron showing extensive cortical accumulation of haemosiderin (x 7).

Figure 2.27: Haemosiderosis. Extensive deposition of haemosiderin in cytoplasm of epithelial cells of proximal convoluted tubules (arrows). Granular casts are present in the tubular lumena.

HE x 320



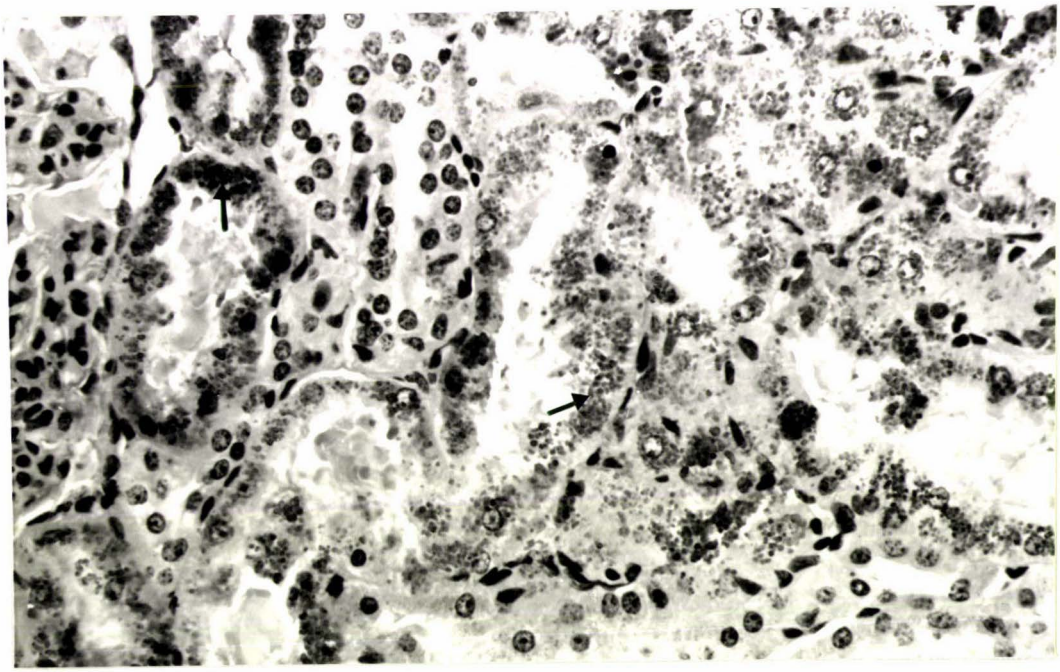
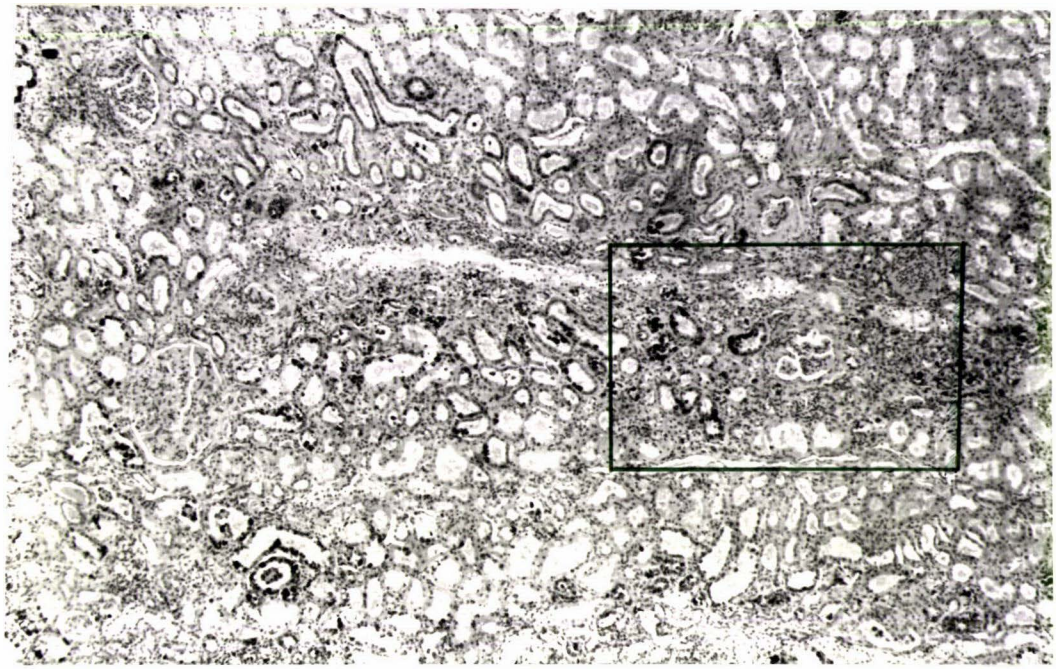


Figure 2.28: Haemosiderosis. Haemosiderin deposition in inflamed and scarred tissue of a kidney.

HE x 50

Figure 2.29: Detail of inset in figure 2.28 showing haemosiderin granules in epithelial cells of tubules (broad arrow) and interstitial inflammatory reaction (narrow arrows).

HE x 150



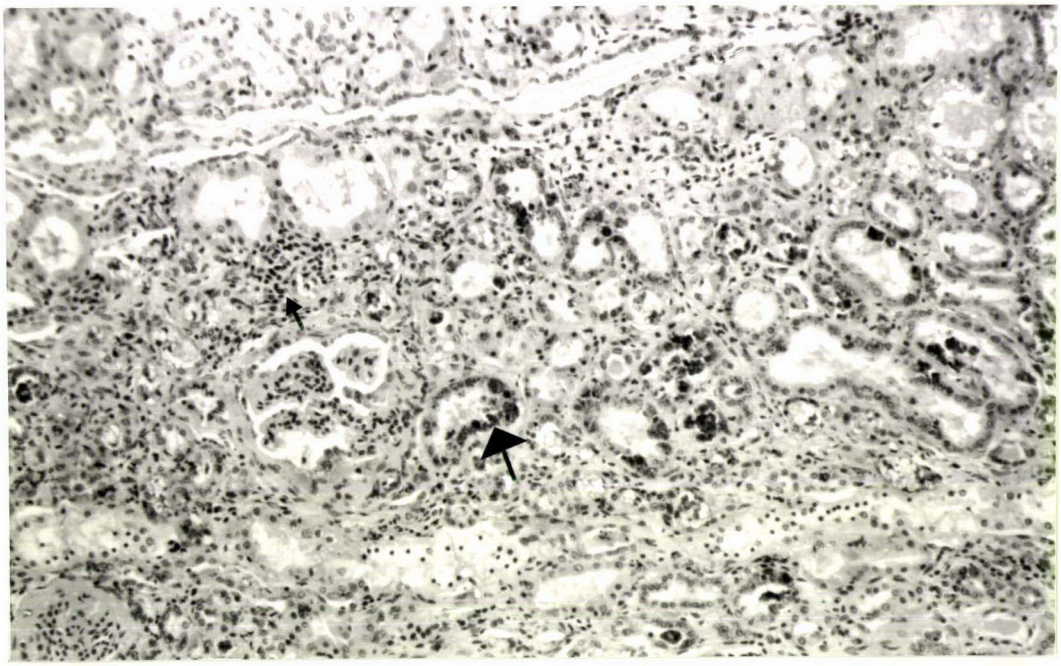


TABLE 2.VI

Histological features of 19 kidneys showing brown discolouration

Data summarized from appendix II

	Nature of lesions		Site of lesions		Distribution of lesions within kidney					Total number of lesions
					F	F/R	M/F	M/R	D	
Glomerulus	Membranous	9	BM)			1	2	3	4	10
	Sclerotic	4	CN)	9						
	Proliferative	1	BC)							
	Segmental	6								
	Panglomerular	3								
	Dilated with haemosiderin	1	Mesangium	1						
			Urinary space	1						
Tubules	Degenerative	14	Cortex	20		1	2	3	16	22
	Regenerative	3								
	Inflammatory	6								
	Casts	5								
	Deposition	19	Cortex/Medulla	2						
Interstitium	Inflammatory	6	Cortex	4		1	3	3	1	8
	Deposition	2								
	Vascular	2	Cortex/Medulla	4						
Pelvis	Degenerative	2					2			2
	Inflammatory	2								

BM = Basement membrane
CN = Capillary network
BC = Bowman's capsule

F = Focal
F/R = Focal radial
M/F = Multiple focal
M/R = Multiple radial

D = Diffuse

TABLE 2.VII

Histological features of 55 kidneys showing spots and streaks

Data summarized from appendix II

	Nature of lesions	Site of lesions	Distribution of lesions within kidney					Total number of lesions
			F	F/R	M/F	M/R	D	
Glomerulus	Membranous 33 Sclerotic 7 Proliferative 4 Segmental 16 Panglomerular 17	BM) CN) 33 BC) Mesangium 4	1		24	8		33
Tubules	Degenerative 28 Regenerative 19 Inflammatory 31 Casts 27 Metaplasia 4 Deposition 18	Cortex 41 Cortex/Medulla 6	3		30	8	6	47
Interstitium	Inflammatory 54 Deposition 15 Vascular 15	Cortex 47 Medulla 2 Cortex/Medulla 13	3		48	8	3	62
Pelvis	Degenerative 6 Regenerative 6 Inflammatory 6				6			6

BM = Basement membrane
CN = Capillary network
BC = Bowman's capsule

F = Focal
F/R = Focal radial
M/F = Multiple focal
M/R = Multiple radial
D = Diffuse

severe infiltration of interstitium with lymphocytes, plasma cells, fibroblasts and phagocytes. The lesions were of multifocal distribution but mainly affected the renal cortex (Figure 2.30). Six kidneys contained additional radial lesions of similar cellularity to those above which extended from cortex into pelvis. Many of the inflammatory lesions were associated with mild vascular congestion.

The majority of kidneys had associated tubular and glomerular lesions (Figure 2.31). Epithelial cells of tubules showed nephrosis with mild to severe cell swelling, cytoplasmic vacuolation and cast formation in tubular lumena. Regenerative change in epithelium was prominent in many areas. There was extension of the inflammatory cell exudate from interstitium into damaged tubules in most severely affected kidneys. PAS positive material was present in the thickened basement membranes of tubules in 12 of the kidneys. Diffuse haemosiderosis of cortical tubules was present in six kidneys. Secondary glomerular lesions were found in all kidneys in areas of inflammatory change. These were essentially the same as those noted in association with diffusely haemosiderotic kidneys (see histopathology section 1c). Four cases also showed marked proliferation of mesangial cells (Figure 2.32).

No microorganisms were observed in any of the kidneys.

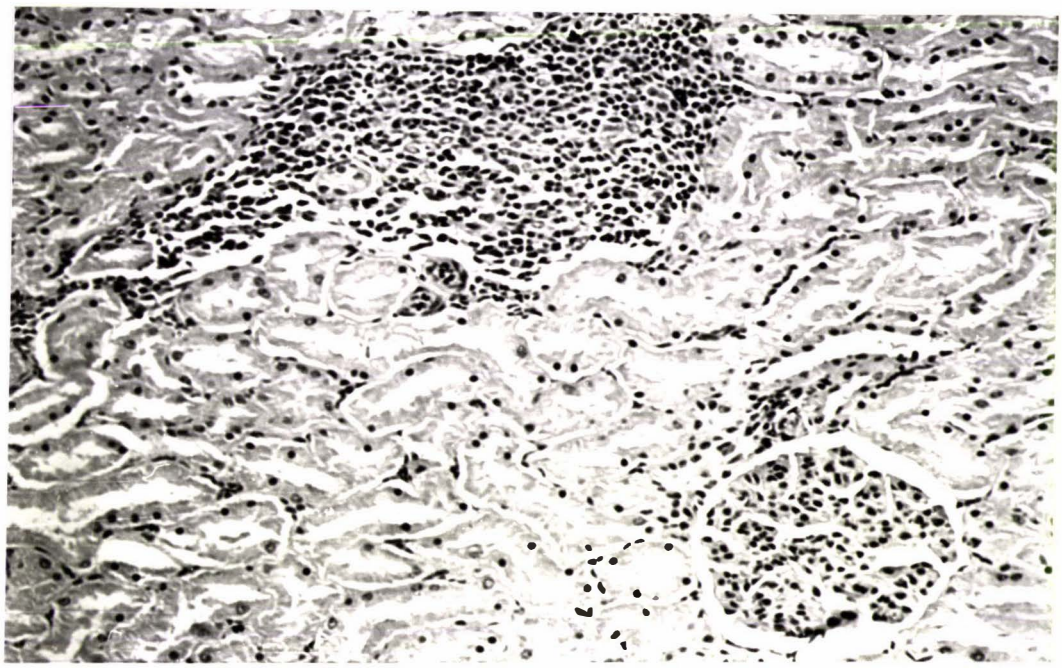
(3) Scars

Eight of the 51 kidneys with scars were from bilaterally affected kidneys of four sheep. The nature, site, distribution and numbers of lesions identified in these 51 kidneys are summarized in Table 2.VIII from data in appendix II.

The common histological feature of all 51 kidneys was a focal or multifocal radial lesion of fibrosis and variable inflammatory reaction involving cortex and medulla with glomerular, interstitial and tubular

Figure 2.30: Kidney with spots and streaks. Focal mainly lymphocytic infiltration of renal cortical interstitium.
HE x 125

Figure 2.31: Kidney with spots and streaks. Interstitial infiltration by lymphocytes and plasma cells. Note the degenerative change in epithelial cells of tubules and formation of proteinaceous casts within lumena.
HE x 320



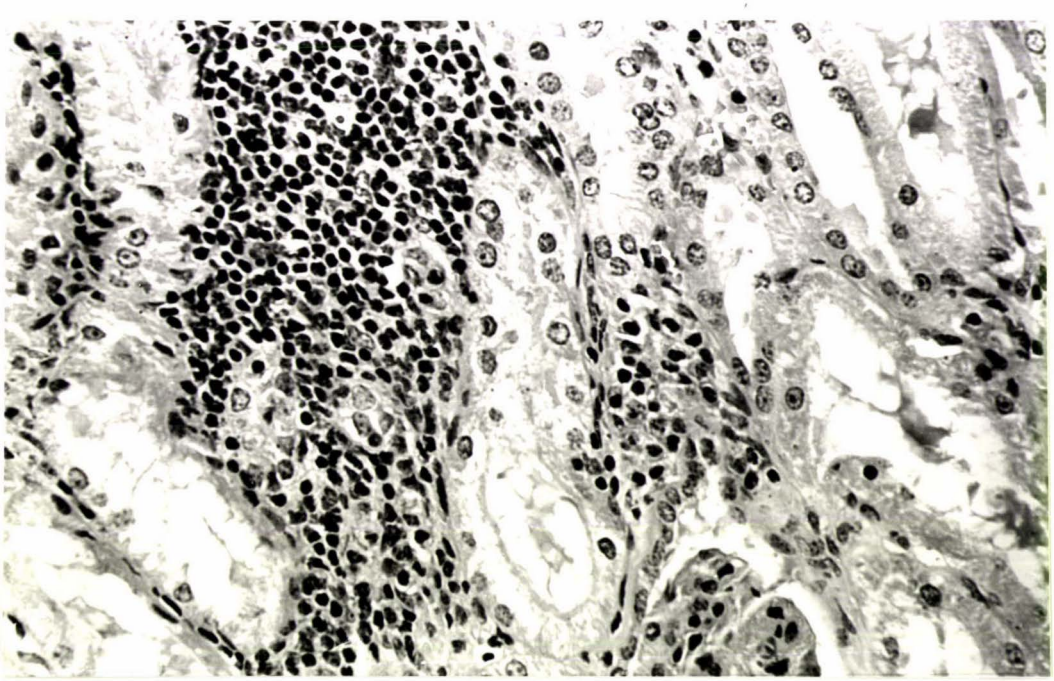
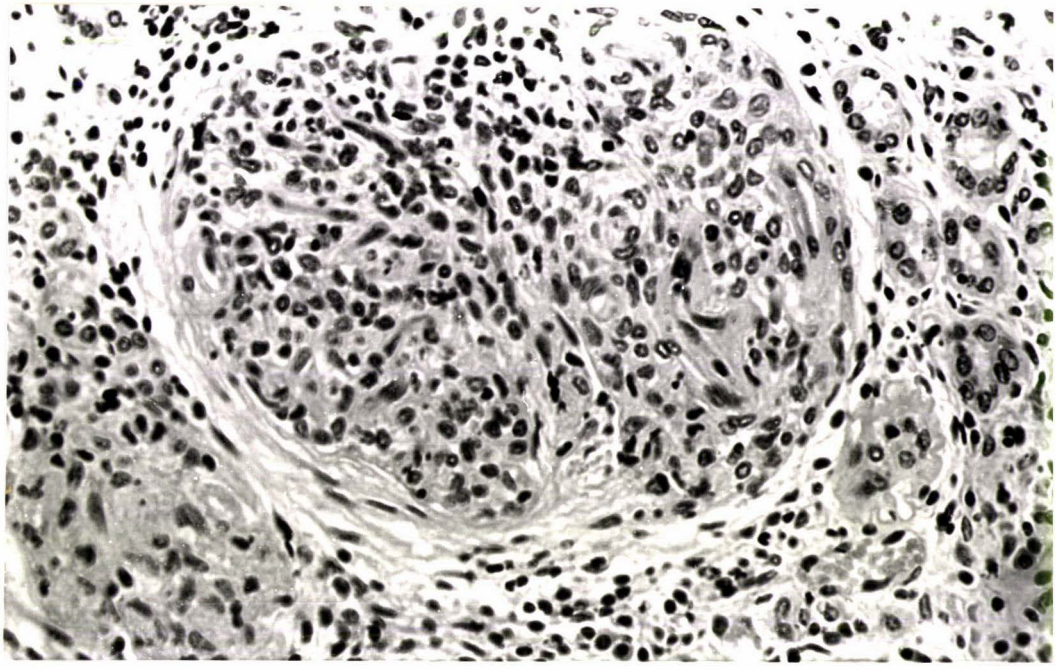


Figure 2.32: Glomerulus within an area of chronic inflammation in a "spotted" kidney. Note the proliferation of cells in glomerular tufts, the obliteration of glomerular capillaries and urinary space and fibrosis of Bowman's capsule. The entire field has been infiltrated by plasma cells and phagocytes.

HE x 320

Figure 2.33: Scarred kidney. Small linear zone of fibrosis with lymphocyte and plasma cell infiltration in the outer cortex.

HE x 125



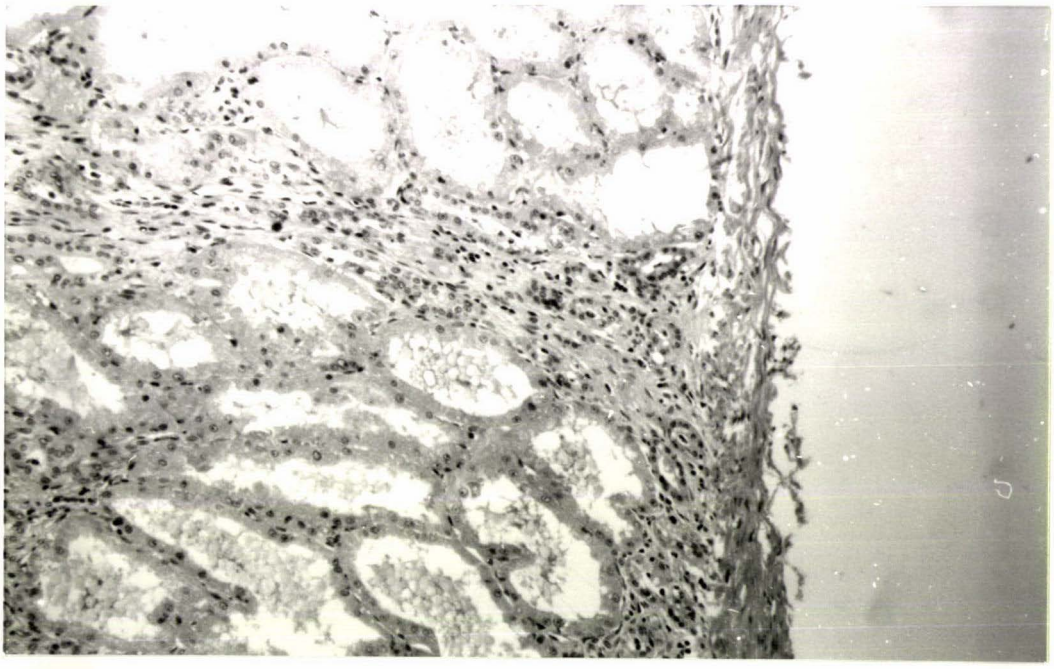


TABLE 2.VIII

Histological features of 51 kidneys with scars

Data summarized from appendix II

	Nature of lesions		Site of lesions		Distribution of lesions within kidney					Total number of lesions
					F	F/R	M/F	M/R	D	
Glomerulus	Membranous	51	BM)			22		29		51
	Sclerotic	39	CN)	51						
	Proliferative	5	BC)							
	Segmental	29								
	Panglomerular	22	Mesangium	5						
Tubules	Degenerative	46	Cortical	16		22	1	29	1	53
	Regenerative	43								
	Inflammatory	43								
	Casts	45								
	Metaplasia	15								
	Deposition	23								
	Cysts	1	Cortex/Medulla	37						
Interstitium	Inflammatory	51	Cortex	13		22		29		51
	Deposition	30								
	Vascular	7	Cortex/Medulla	38						
Pelvis	Degenerative	25			9		27			36
	Regenerative	22								
	Inflammatory	36								

BM = Basement membrane
CN = Capillary network
BC = Bowman's capsule

F = Focal
F/R = Focal radial
M/F = Multiple focal
M/R = Multiple radial

D = Diffuse

changes.

All lesions of interstitium were characterized by fibrosis and moderate to severe infiltration with lymphocytes and plasma cells (Figures 2.33 and 2.34). Some kidneys showed congestion in association with the other inflammatory changes.

Inflammatory lesions of the pelvis were a feature in 36 kidneys and were of similar cellularity to those in more proximal zones of the nephrons (Figures 2.35, 2.36 and 2.37).

Degenerative changes in the epithelium of the tubules were more severe than those in spotted kidneys (Figure 2.38). Squamous metaplasia of tubule epithelium was present in 15 and membranous deposition on the tubular basement membranes in 23 kidneys. One kidney had multiple cystic dilatations of tubules containing deeply eosinophilic fluid and another showed diffuse haemosiderosis of cortical tubular epithelia.

All 51 kidneys showed secondary glomerular lesions of mild to severe membranosclerotic, segmental or panglomerular deposition on basement membranes and Bowman's capsule (Figures 2.39 and 2.40) in association with the interstitial lesions. Five kidneys also showed proliferation of mesangial cells.

No microorganisms were recognized.

(4) Cysts

Of ten kidneys with cystic lesions examined four were the bilaterally affected kidneys of two sheep. All were present within the renal cortex and were lined by flattened epithelium. Those cysts which had retained their content during histological processing contained a slightly eosinophilic, PAS positive fluid.

Five of the cysts had chronic inflammatory change in adjacent

Figure 2.34: Scarred kidney. An extensive wedgeshaped cortical scar associated with marked tubular and glomerular atrophy.

HE x 50

Figure 2.35: Renal papilla (on left) and pelvis (on right) of a kidney with extensive radial scarring. Note the reduced numbers of tubules, several containing casts (broad arrows) and others with calcification of basement membrane and epithelial cells (narrow arrows). The interstitial tissue of the papilla is fibrotic and lightly infiltrated by lymphocytes. The pelvic epithelium is slightly hyperplastic and shows mild infiltration of subepithelial tissue by lymphocytes.

HE x 50

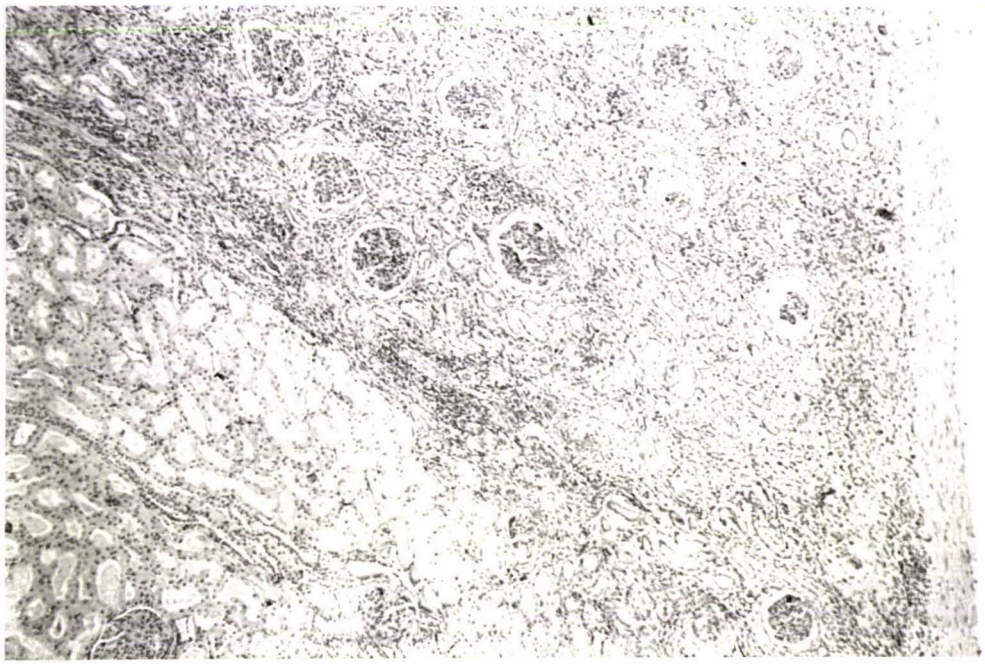
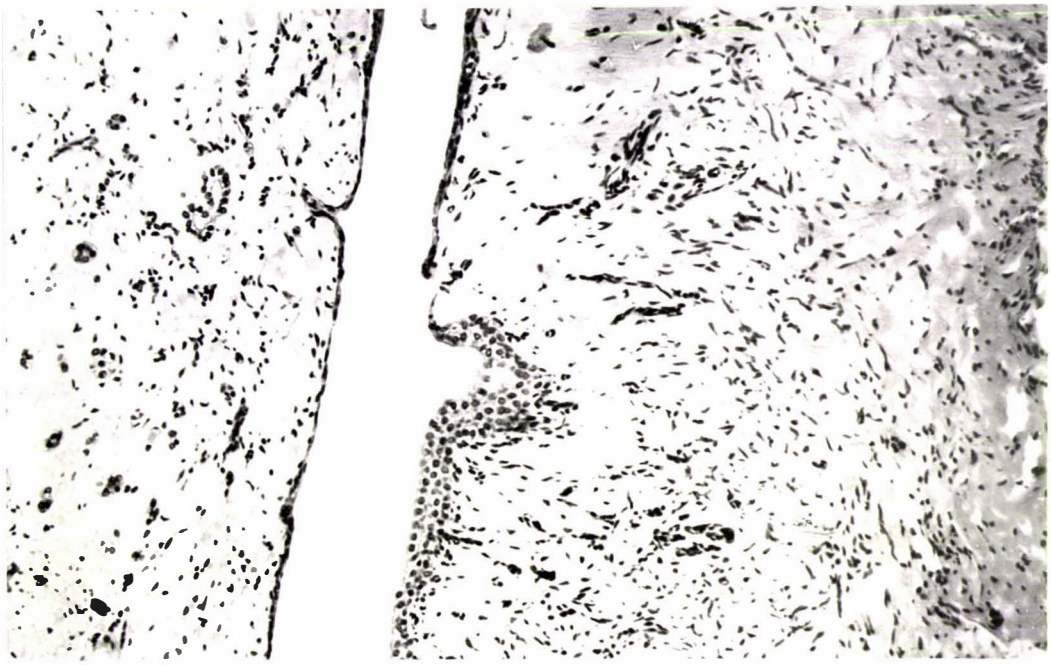


Figure 2.36: Renal papilla (on left) and pelvis (on right) of a severely scarred kidney showing extensive replacement of tubules by fibrosis. Remaining tubules are atrophied. There is extensive fibrosis of the renal pelvis.
HE x 125

Figure 2.37: Degeneration of pelvic and papillary epithelia associated with fibrosis, lymphocytic and plasmacytic infiltrations.
HE x 320



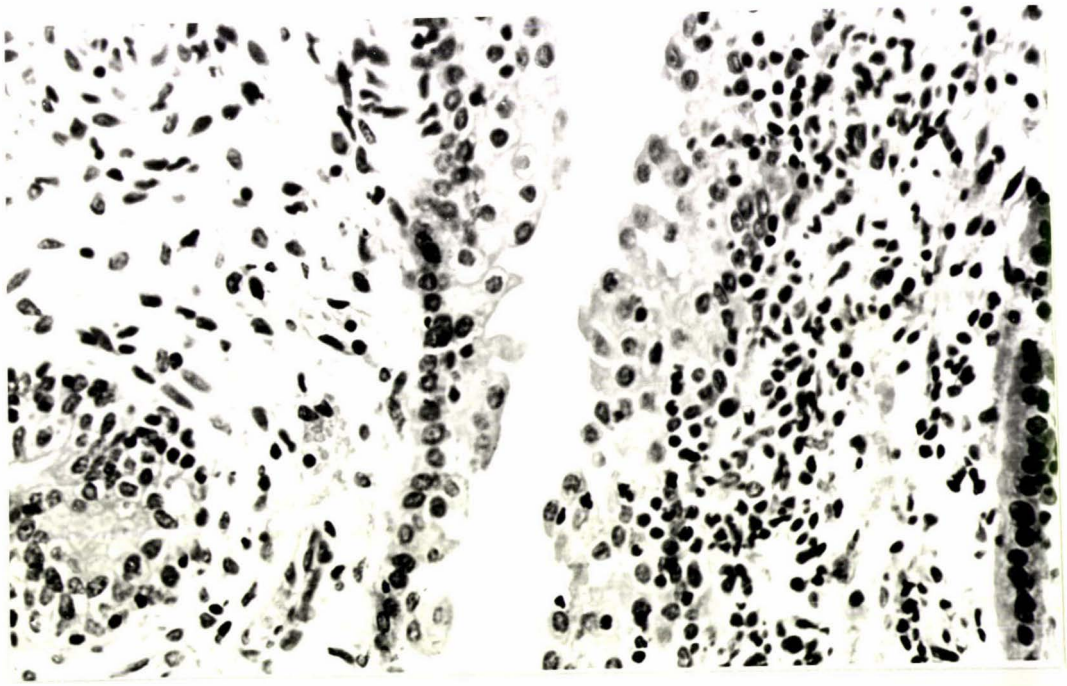
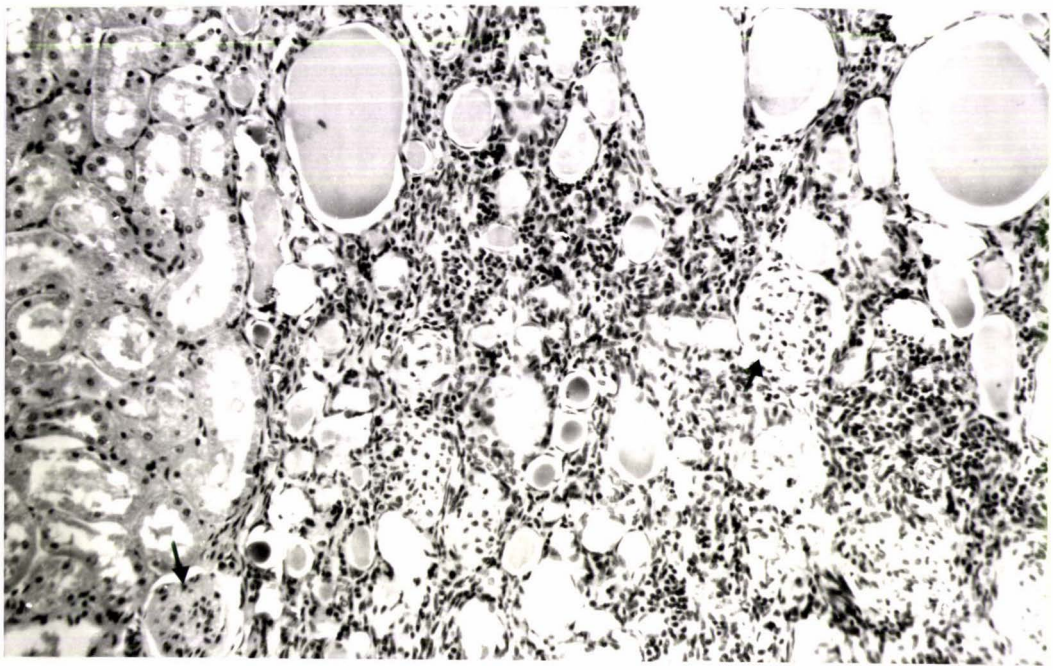


Figure 2.38: Renal cortex of a scarred kidney showing tubular atrophy, dilatation of tubules with accumulation of highly eosinophilic material in lumena (thyroidization), interstitial fibrosis and infiltration by lymphocytes and plasma cells. Two atrophied glomeruli are present (narrow arrows).

HE x 125

Figure 2.39: Glomeruli in area of inflammation and scarring showing gradation in the severity of basement membrane thickening due to membranous deposition. Note the segmental lesion in one glomerulus (arrow).

PAS x 125



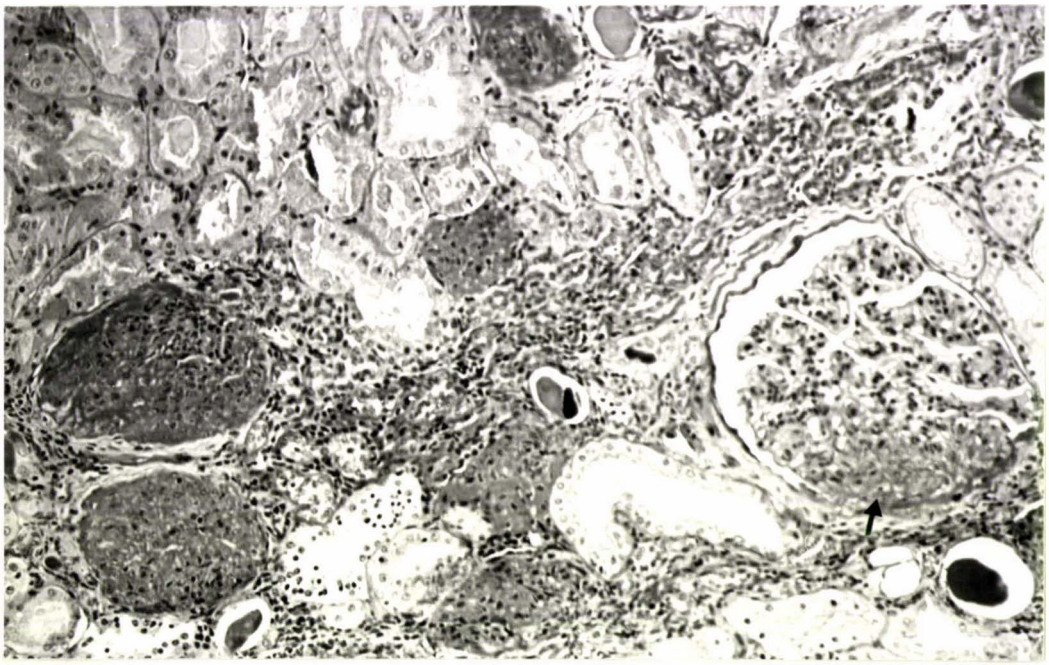
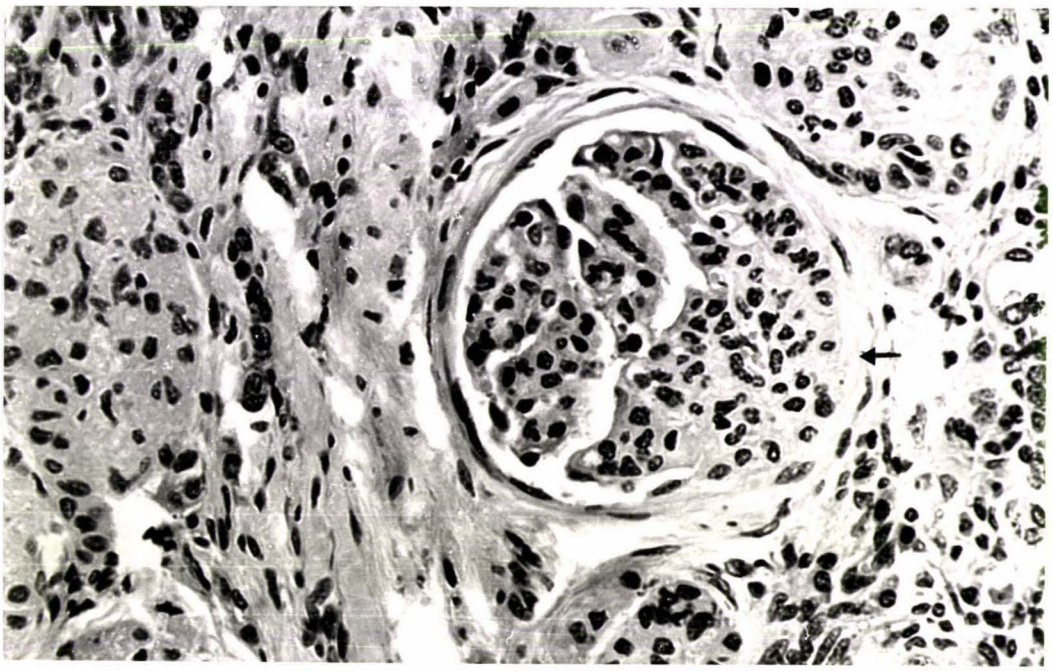


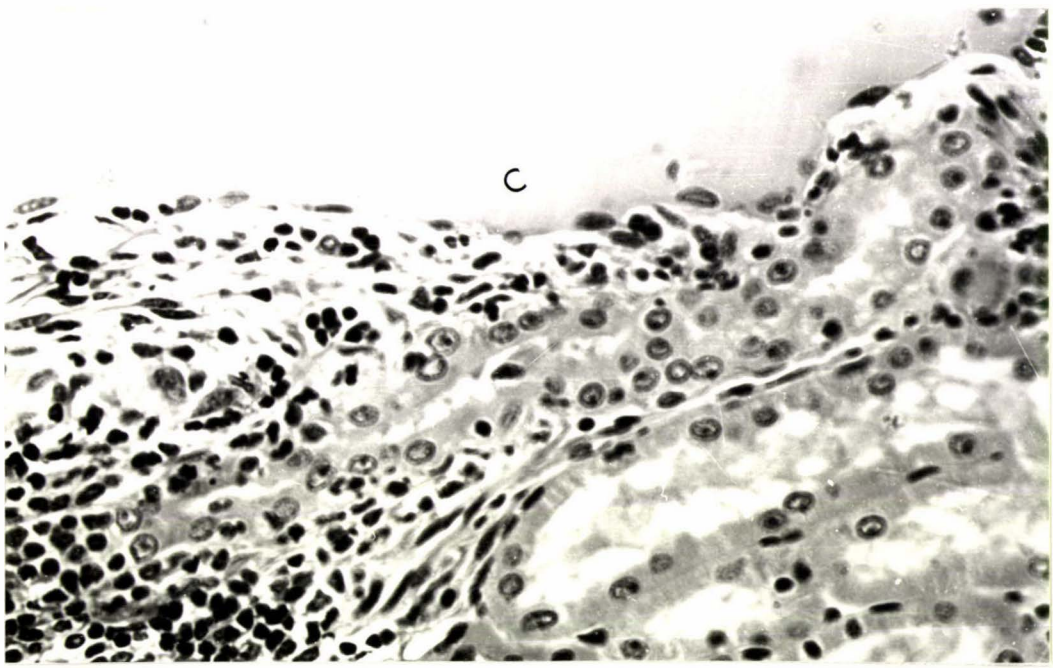
Figure 2.40: Glomerulus in an area of chronic inflammation showing severe sclerosis of Bowman's capsule with collagenous adhesion (arrow) to the adjacent glomerular tuft.

HE x 320

Figure 2.41: Renal cyst (C) associated with chronic inflammatory exudate of lymphocytes and plasma cells in adjacent renal tissue.

HE x 320




C

cortical interstitium in which lymphocytes, plasma cells and collagen were prominent (Figure 2.41). Tubules showed swelling of the epithelial cells with casts of granular, hyaline and erythrocytes in the lumena. One showed marked squamous metaplasia and cast formation. In two cases calcified casts were widespread in the collecting tubules. One of kidneys showed a moderately severe focal degenerative lesion of glomeruli adjacent to the cyst in which there was segmental membranous deposition of PAS positive material on the basement membranes and pure hyaline casts had formed in the proximal and distal convoluted tubules.

The other five kidneys contained no inflammatory lesions (Figure 2.42) although mild degenerative changes were present in the tubules proximal and distal to the cysts. Calcifying spherules were present in collecting tubules in one case.

Parasitic cysts

The hydatid cyst examined histologically occupied the width of the cortex and extended a short distance into the medulla. Three distinct layers in the wall of the cyst could be distinguished. The inner layer consisted of laminated hyaline membrane (Figure 2.43). Surrounding this was an eosinophilic collagenous band lightly infiltrated by small numbers of degenerating lymphocytes, phagocytes and eosinophils. Outside the collagenous tissue was a zone of granulomatous inflammatory reaction characterized by the heavy infiltration of fibroblasts, lymphocytes, phagocytes, eosinophils and polykaryons. Between the middle and outer layers were numerous small calcified foci.

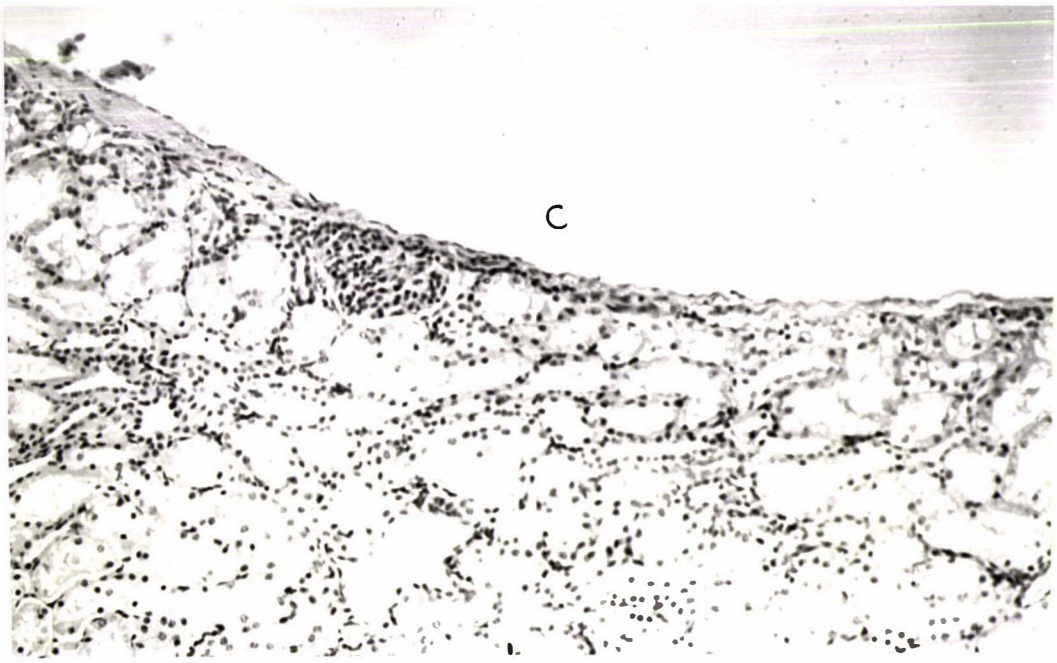
Renal parenchyma adjacent to the parasitic lesion showed atrophy with some regeneration and metaplasia of the tubular epithelium and accumulation of protein-rich fluid in the tubules. A scattering of lymphocytes, phagocytes and eosinophils were present in the interstitium in these areas.

Figure 2.42: Renal cyst (C) with no inflammatory changes in the adjacent renal parenchyma.

HE x 125

Figure 2.43: *Echinococcus granulosus* cyst showing laminated cuticle (LC) adjacent to the collagenous tissue of the second layer of the cyst (CC).

HE x 320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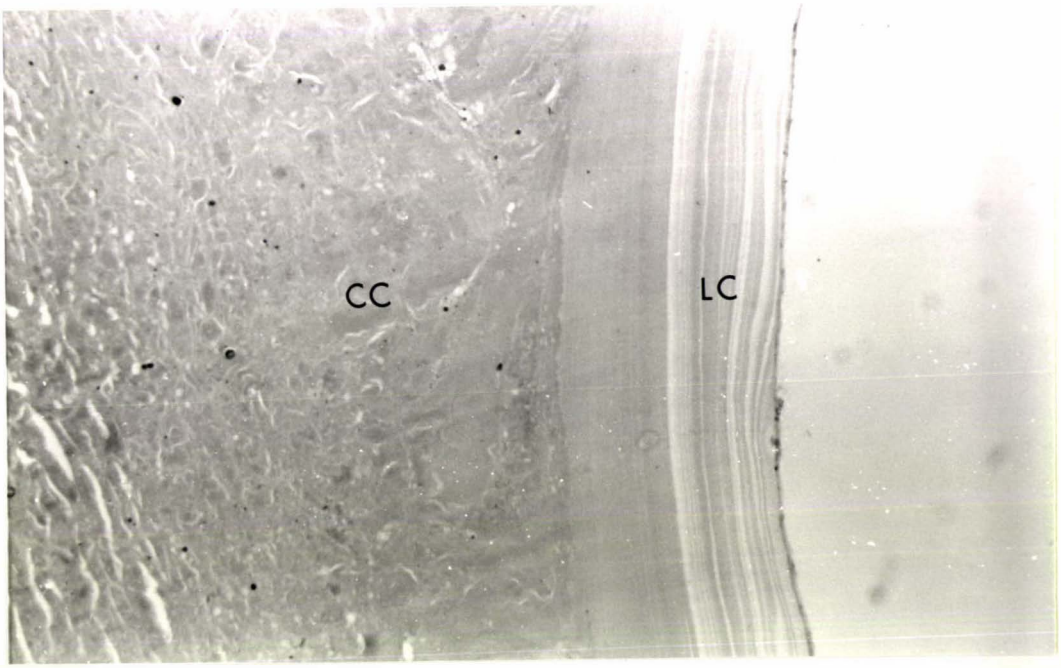

CC
LC

(5) Nodules

a) Abscesses

The major histological lesion in four kidneys with abscesses was a focal or multifocal, suppurative, inflammatory lesion in which neutrophils were the predominant cell type. Lesser numbers of lymphocytes, plasma cells, phagocytes and fibroblasts were present in the peripheral and capsular areas of the lesions. Secondary degenerative changes occurred in tubules and glomeruli adjacent to the abscesses. The glomerular lesions were characterized by mild to moderate segmental membranous deposition of PAS positive material on capillary basement membranes and Bowman's capsule and mild mesangial cell proliferation. In the tubules there was moderate to severe necrosis of epithelial cells with hyaline and granular cast formation.

b) Neoplasms

(i) Carcinoma

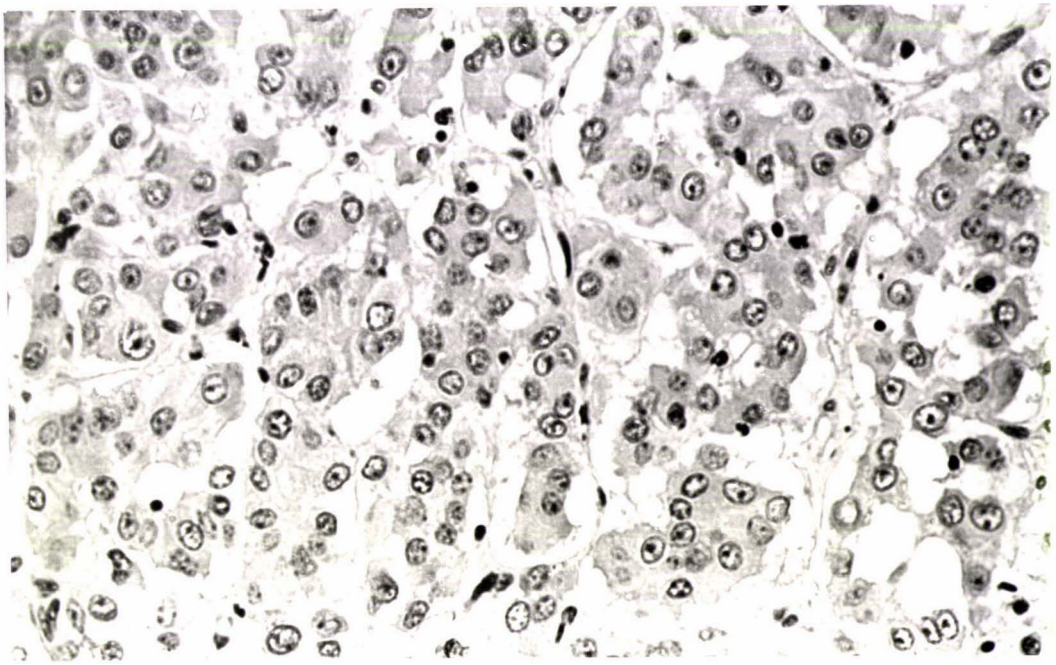
Kidney No. 237 contained a large lobulated neoplastic mass which had replaced all normal tissue in the caudal pole of the kidney. There was considerable variation in differentiation from lobule to lobule and to a lesser degree within lobules, with the neoplastic cells forming tubular, papillary, acinar or solid pattern carcinoma. In most areas the cells were polyhedral to ovoid with abundant, slightly vacuolated eosinophilic cytoplasm (Figure 2.44). The large nucleus, usually basally situated, showed variation in chromatin density from cell to cell. In the more densely cellular areas of tumour the cells were smaller, columnar or spindle shaped with pachychromatic nuclei (Figure 2.45). Peripherally the tumour cells showed invasion of adjacent compressed renal tissue. The stroma of the neoplasm consisted of a light collagenous matrix with heavier bands of fibrous tissue separating the lobules. Extensive areas of necrosis, haemorrhage and dystrophic calcification were present (Figure 2.46).

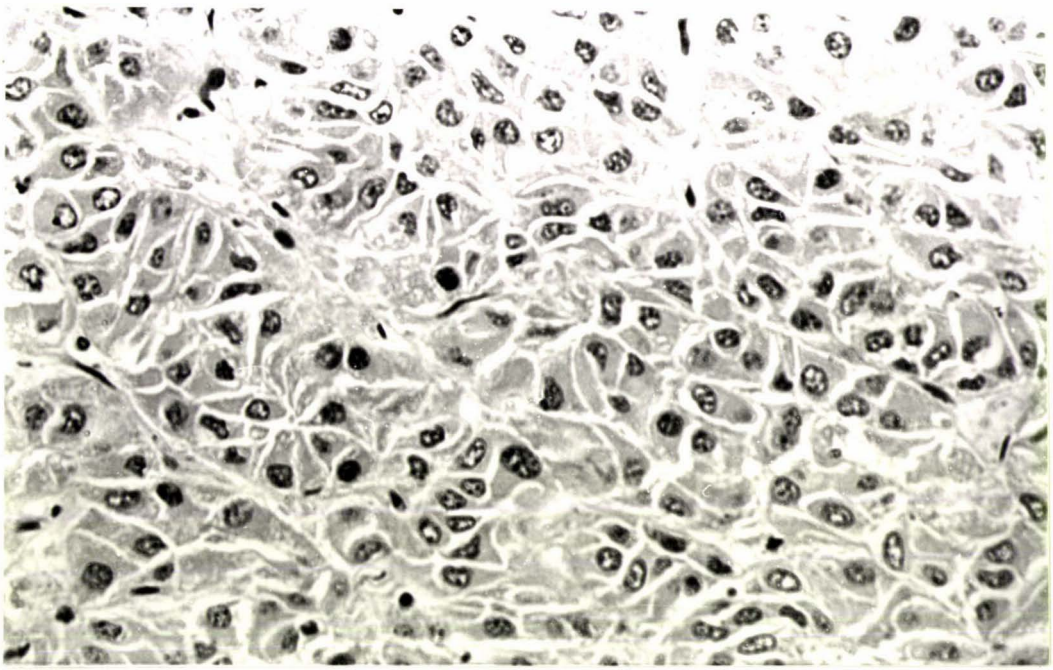
Figure 2.44: Carcinoma. Acinar and tubuloacinar arrangement of neoplastic cells supported by a slight reticular stroma. The cells are polyhedral to ovoid with abundant, slightly vacuolated, eosinophilic cytoplasm.

HE x 320

Figure 2.45: Carcinoma. A more densely, cellular area of tumour to that shown in figure 2.44 showing greater cellular pleomorphism.

HE x 320

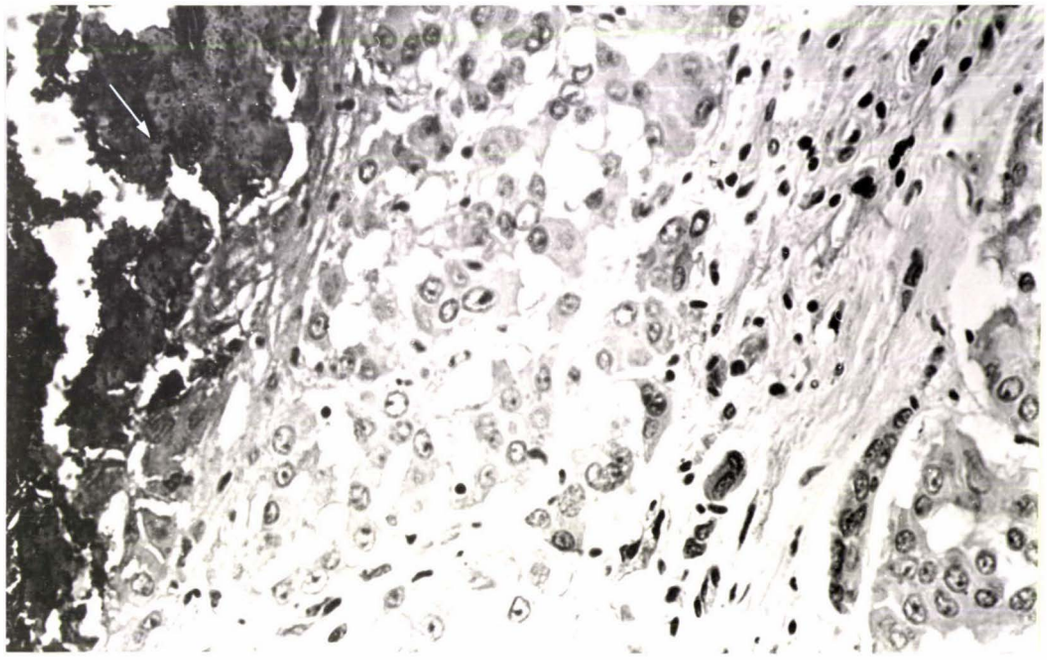


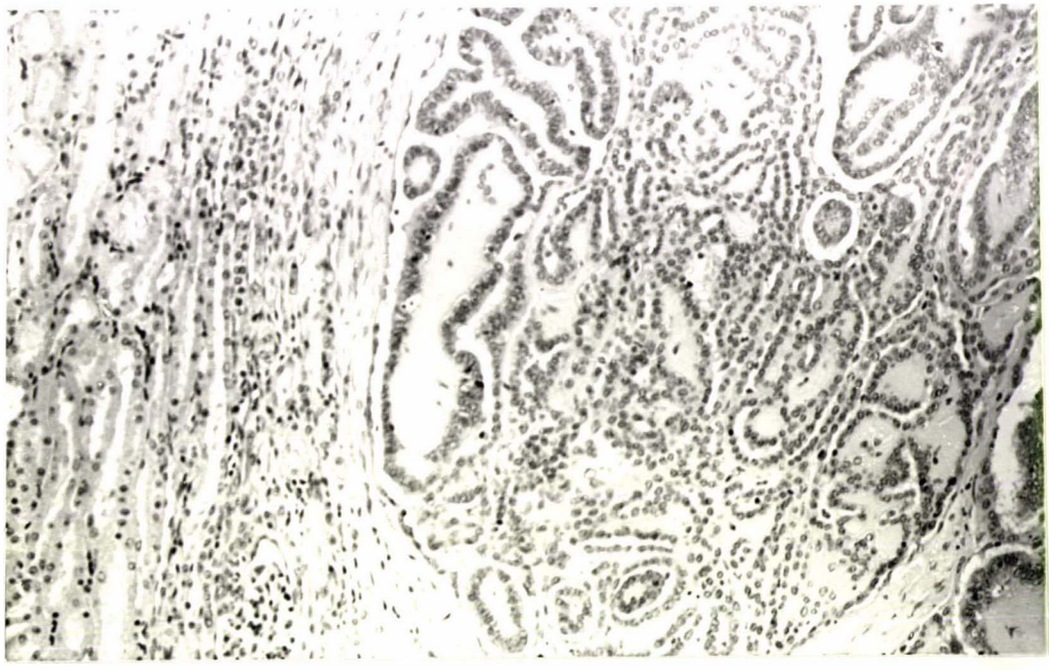


Following page 49

Figure 2.46: Carcinoma showing dystrophic calcification (white arrow) and a band of fibrous tissue representative of that dividing the tumour into lobules.
HE x 320

Figure 2.47: Renal adenoma. The tumour is well encapsulated and the neoplastic cells are arranged in acinar, tubular and papillary formations.
HE x 125





(II) Adenoma

Kidney No. 59 contained a small heavily encapsulated neoplasm in the caudal pole of the kidney. The neoplastic cells were mainly columnar with a small quantity of lightly eosinophilic cytoplasm and large ovoid leptochromatic nuclei. Mitotic figures were infrequent. The neoplastic cells were arranged in acinar, tubular and occasionally in papillary formation on the relatively dense collagenous stroma. Areas of necrosis were absent and anaplasia of cells minimal. Renal parenchyma adjacent to the adenoma showed compression atrophy and focal aggregations of lymphocytes, plasma cells and phagocytes (Figure 2.47).

(III) Malignant Lymphoma

Multiple nodules of neoplastic lymphoid cells were scattered throughout the cortex and hilus of kidney No. 236. Within the nodules sheets of monotonously regular cells having a small amount of lightly eosinophilic cytoplasm and round, ovoid or indented pachychromatic nuclei and moderately high mitotic rate replaced most of the normal renal tissue with occasional sparing of glomeruli and tubules (Figure 2.48). The cells are unsupported except by remaining renal connective tissue and show invasion of the adjacent renal tissue.

c) Nodules of uncertain aetiology

Two of the 14 kidneys examined were from the same sheep. The nature, site, distribution and total number of lesions found in the 14 kidneys are summarized in Table 2.IX. In most there were focal granulomatous lesions in the cortex associated with secondary degenerative change in cortex and medulla and involving glomeruli, tubules and interstitium.

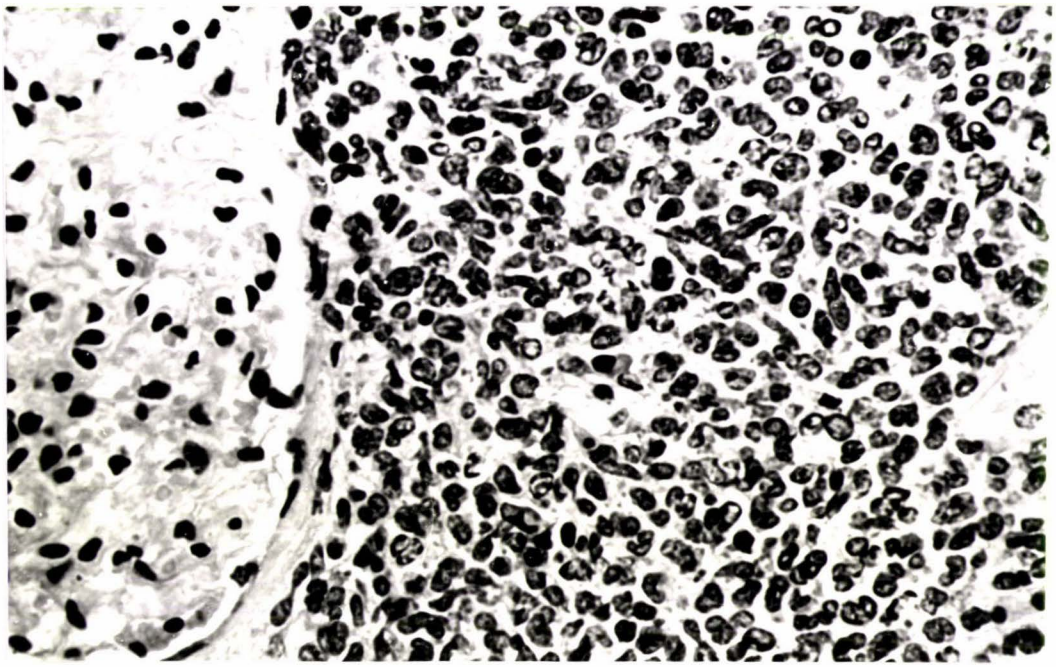
The granulomatous foci contained aggregations of eosinophils, lymphocytes, plasma cells, phagocytes and in some cases polykaryons, which surrounded central areas of necrosis and eosinophilic debris (Figure 2.49). The associated glomerular lesions in eight kidneys were

Figure 2.48: Malignant lymphoma. Monotonous regularity of lymphoid cells with round, ovoid or indented pachychromatic nuclei and inapparent nucleoli.

HE x 500

Figure 2.49: Focal granuloma in renal cortex, probably of parasitic origin, showing central area of caseous necrosis and eosinophilic debris (N). Surrounding this material are several polykaryons (arrows). The adjacent tissue is heavily infiltrated by lymphocytes, plasma cells, phagocytes and occasional eosinophils.

HE x 320



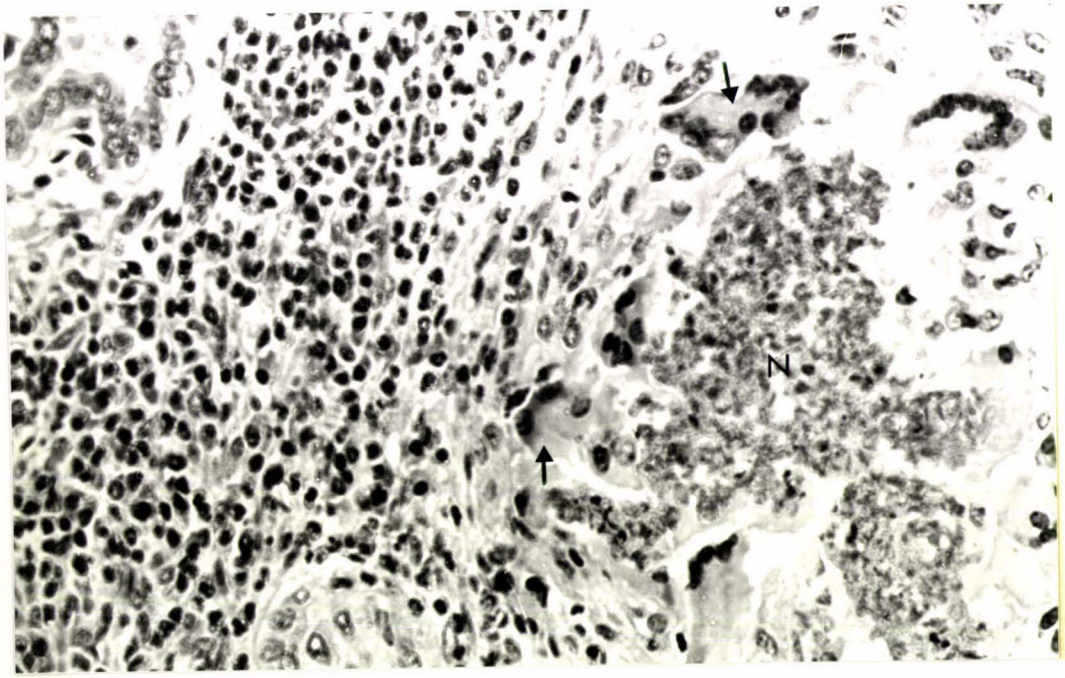

N

TABLE 2.IX

Histological features of nodules of uncertain aetiology in 14 kidneys

Data summarized from appendix II

	Nature of lesions		Site of lesions		Distribution of lesions within kidney					Total number of lesions
					F	F/R	M/F	M/R	D	
Glomerulus	Membranous Sclerotic Segmental Panglomerular	8 3 7 1	BM) CN) BC)	8	5		1	2		8
Tubules	Degenerative Regenerative Inflammatory Casts Deposition	16 12 12 12 3	Cortex Cortex/Medulla	16 1	11		1	2	3	17
Interstitium	Inflammatory Vascular	14 1	Cortex Cortex/Medulla	10 5	11		2	2		15

BM = Basement membrane
CN = Capillary network
BC = Bowman's capsule

F = Focal
F/R = Focal radial
M/F = Multiple focal
M/R = Multiple radial
D = Diffuse

those of segmental membranosclerotic change involving basement membranes and Bowman's capsule. Three of the kidneys showed diffuse corticotubular epithelial cell degeneration and haemosiderosis. Another kidney showed moderately severe multifocal cortical and medullary congestion and a focal chronic inflammatory lesion in addition to the granuloma.

(6) Infarcted kidney

An occluding, organizing thrombus with early recanalization was present in the renal vein. Recent thrombi had occluded several of the arcuate arteries. The entire kidney was necrotic but the age of lesions varied from area to area. In the subacute lesions the necrotic cortical tissue had a peripheral zone of inflammation in which neutrophils and phagocytes were present. Interspersed with these areas of subacute change were zones of very recent necrosis with no cellular inflammatory component. The renal medulla and papilla also showed recent extensive necrosis.

Control kidneys

No histological abnormalities were noted in the 10 kidneys from lambs or 25 of the 45 kidneys from old ewes. Twenty kidneys of the old ewes contained very mild chronic inflammatory lesions identical to those seen in kidneys with spots (see histology section 2).

DISCUSSION

The statutory regulations for kidney condemnation in sheep killed in New Zealand slaughter-houses requires that the meat inspector discard any kidneys considered in his opinion unfit for human consumption (Anon, 1969). Under this "umbrella" two major reasons for condemnation emerged in the present investigation. The first of these included kidneys spoiled as the result of post mortem handling procedures resulting in physical damage and contamination by gut content or non-sterile fomites, while in the second groupwere those with abnormalities resulting from

pathological or physiological processes occurring antemortem. The 830 kidneys examined in this survey were from the latter group, but although accurate records were not made of losses occurring in the former, it was apparent from observation that this group was numerically as large as that with antemortem lesions. Included in the 830 kidneys condemned for pathological changes were 68 kidneys with discolouration due to congestion. In the absence of other pathological abnormalities it is unlikely that such kidneys constitute a human health hazard and could therefore have been passed as fit for consumption.

Other reports of kidney condemnation in sheep killed in slaughter-houses are few in the literature. Zhirik (1974) reported the prevalence of lesions in such material from Moldavia as 1.26 per cent of 15,558 sheep.

Whether or not surveys such as these are truly representative of renal disease in large populations of sheep may be questioned on the grounds that they are conducted over a relatively short period of time and therefore cannot reflect overall patterns or changes in disease occurrence and only indicate disease in the particular catchment area sampled. While such criticism is valid, it should be pointed out that statistics to accurately reflect such a wide spectrum of diseases as occurs in kidneys can only be compiled by numerous surveys. This survey then represents a beginning. As the majority of lesions noted in this and in Zhirik's survey were chronic in type, it is probable that the figures are meaningful in terms of the non fatal renal diseases of the area surveyed. In the present survey the catchment area included a large portion of the central and southern part of the North Island of New Zealand.

At first examination there appeared to be some variation in

prevalence of renal diseases in different lines of animals slaughtered, but subsequent analysis showed these differences to be non-significant at the 95 per cent confidence level. This indicates an overall even distribution in the prevalence of renal disease and supports the argument that the survey was at least representative for the catchment area under consideration.

The significance of the individual weights of kidneys is difficult to assess. In the case of diffusely scarred organs the decreased weight could be explained on the basis of loss of epithelial tissue, while some of the large kidneys were paired with much smaller ones indicating a compensatory hypertrophy. Even after elimination of these extremes there was considerable variation in kidney mass which did not appear to correlate with the extent of pathological lesion except in those kidneys with large tumours or massive infarction. The mean weight of the kidneys was 76.14 g which is considerably less than that cited by Nickel, Schummer and Seiferle, (1975) and May, (1964) in sheep. According to Nickel et al the average weight of sheep kidney ranges between 100--160 g, while May states 120 g for both kidneys. Unfortunately none of these authors quote breed or age of sheep. Ghanekar and Soman (1974) reported that in sheep the maximum kidney weight relative to body weight occurs at six months of age and then declines in proportion to the increasing body weight associated with continuation of growth. Data collected by the Production Manager of Borthwick's freezing works at Longburn showed that the average weight of 3,700 kidneys sampled during the 1976-77 and 1977-78 killing seasons was 74.24 g (pers. comm.).

That the majority of lesions were bilateral suggested that most resulted from a systemic process with haematogenous distribution of pathogenic substances. The probable exception was in the case of the ascending pyelonephritides.

With the exception of nephrosis and congestion all the reported lesions were chronic in nature and for most a definitive aetiology was not established. In fact, the parasitic lesions caused by Echinococcus granulosus hydatid cysts were the only lesions with definitively identified causal agents. In the others the aetiology is more or less speculative. In the case of abscesses the causal agents were most likely to be pyogenic bacteria although no organisms were identified in the affected tissues. Renal abscesses have been reported in other species as being due to Corynebacterium pyogenes, Erysipelothrix insidiosa and other pyogenic bacteria (Jubb and Kennedy, 1970).

The kidneys with either spots and streaks or scars present a challenging problem in aetiological diagnosis. The major histological difference between these lesions was the degree of collagen deposition and the distribution of the lesions. Because of the radial nature of the majority of the scars and the presence of inflammatory changes in particular associated with the pelvis, these cases were considered to be chronic pyelonephritic lesions. Fifteen of 51 kidneys with scars examined histologically were associated with obliterative lesions of the arcuate vessels and occupied mainly cortical parenchyma were most consistent with infarcts.

The distribution of spotted and streaked lesions on the other hand suggested a haematologically disseminated pathogen. Such lesions have been described in leptospirosis due to Leptospira pomona infection. (Hartley, 1952; Hodges, 1974; Marshall, 1974). In the present survey, no leptospirae were demonstrated in Warthin Starry stained sections of the kidneys. This however does not exlude them as the aetiological factor because it is likely that in lesions of this age the leptospirae may no longer be present. In addition, unless the organism is present

in large numbers, silver staining techniques are not particularly sensitive methods for their demonstration. (Mackintosh, pers. comm.). The most sensitive method for demonstration of leptospiral infection of the kidney is by culture. Serological demonstration of the organism may also be of value (Hathaway, 1978). The posibility that these lesions were the result of infection by other non pyogenic bacteria should not be excluded.

All except eight of the kidneys discoloured brown showed diffuse, moderate to severe corticotubular intracytoplasmic and intralumenal accumulation of haemosiderin. This change is identical to that described in sheep infected with Eperythrozoon ovis (Sutton, 1974). In the other eight kidneys with brown discolouration, haemosiderin deposition occurred in areas of interstitial inflammation. Although haemosiderosis conforming to this pattern has been reported from Norway in sheep with natural E. ovis infection (Øveras, 1969), it is to be expected that any inflammatory lesion in which haemorrhage occurs would result in such a distribution of pigment. While it is most likely that the majority of these lesions are the outcome of E. ovis infection, the possibility that other diseases in which haemolysis or intrarenal haemorrhage occurs should not be excluded.

One hundred and seventy four kidneys showed pale discolouration as the major gross pathological feature (Table 2.III). In the nine kidneys with this change examined histologically the lesion was that of diffuse recent nephrosis of mild to moderate severity. Cause of such non-specific nephrotic change could be due to any exogenous or endogenous nephrotoxin. The recent nature of the lesions suggest that the most likely cause would be related to management during the time of transportation from farms of origin until the time of slaughter. Although the nephrosis was sometimes present in kidneys with other

inflammatory or degenerative lesions, the diffuse distribution of nephrosis suggested that this change was unrelated to the others. The other recent lesion noted in this survey, that of multifocal or diffuse congestion, was most likely to be of physiological origin caused by cardiovascular and haemodynamic alterations caused by yarding and slaughtering procedures.

Histological examination of the cysts showed that with the exception of those of parasitic origin, the lesions were due to urinary retention. Five of the ten were most likely the result of acquired tubular blockage as a result of the associated inflammatory reaction. The other five cysts occurred in the abscence of inflammation and were therefore probably of congenital origin. The mild degenerative lesions in the tubules proximal and distal to the cysts were considered to be changes secondary to urinary retention.

Although the kidneys containing viable parasitic cysts were readily diagnosed on gross features other kidneys with nodular lesions were recognized on histological grounds as parasitic. The latter type of lesion could not be differentiated at gross examination from other long standing focal areas of inflammation but the presence of granulomata containing eosinophils and polykaryons indicated a parasitic aetiology. In these, the absence of a parasite or parasitic remnants made identification of the specific parasite impossible and they may have been caused by any of several parasites including abberant larvae of Toxocara, Ascaris or Parascaris species as well as the various parasites of sheep which are more commonly found in other tissues.

Kidneys of all species are a favourable site for the development of embolic suppurative lesions because of the large volume of blood passing through them and the end-artery type of their vasculature.

Although the primary site of sepsis was undetermined, this was considered to be the most likely route of infection of most of the renal abscesses in the present series. There was at least one case in which a suppurative pyelitis was also present and in this instance the abscesses may have been the result of an ascending infection.

One of the three tumours found was grossly presented as a small, heavily encapsulated neoplastic mass, which on histopathological examination seemed to grow by expansion with no apparent invasion of the adjacent renal parenchyma. Although it is not always possible to separate adenoma from adenocarcinoma on a morphological basis only (Nielsen et al, 1976) this neoplasm fulfilled the gross and histological criteria of a renal adenoma. The other tumour of epithelial cells showed considerable histological variation from lobule to lobule with anaplasia and varying degrees of differentiation. These features and the invasion of the adjacent renal parenchyma indicated that it was a malignant neoplasm. While it is most likely to have been of renal origin, the possibility that it was a metastatic lesion of a carcinoma situated elsewhere in the animal could not be ruled out. Malignant lymphoma is a relatively frequent neoplasm in sheep and the kidneys are common sites of involvement (Johnstone and Manktelow, 1978). Although the kidneys may be the only site involved (Webster, 1966; Johnstone and Manktelow) it is likely that in the present example other lesions were present elsewhere in the carcass.

A problem encountered in the histological examination of many of the lesions and in particular the spots and streaks, scars and other chronic inflammatory lesions was the determination of the primary site of injury. In most of the lesions the normal kidney morphology was greatly altered by the pathological changes occurring and hence whether the initial change was of vascular, interstitial or epithelial origin

was impossible to identify. In such cases secondary degenerative changes were usually present in all zones of the nephron as well as the adjacent stromal tissue.

The glomerular lesions seen in many of the kidneys in this survey illustrate this point well in that in all cases they were intimately associated with tubular and interstitial changes with the same focal, multifocal or radial distribution as the associated lesion. Most showed either segmental or panglomerular membranosclerotic deposition on the capillary basement membranes and Bowman's capsule. Mesangial cell proliferation was found in only a few kidneys and again these lesions were related in distribution to the accompanying lesions elsewhere. Because of the obvious association between glomerular and other lesions and the absence of diffusely distributed glomerular lesions, all glomerular pathology was considered as secondary to other pathological changes. Lesions characteristic of the glomerulonephritis observed by Lerner and Dixon (1966a) were mostly proliferative rather than sclerotic, membranosclerotic, dystrophic or atrophic as was the case in this survey. In addition, all the kidneys examined were collected from mature sheep, and the primary glomerulopathies in sheep have tended to affect younger animals (Lerner and Dixon; Angus et al, 1974). To eliminate the possibility of a primary glomerulopathy in the absence of gross lesions 45 normal kidneys from old ewes were examined histologically and none showed evidence of primary glomerular lesions. These results support the observation made by McCausland et al, 1974 that glomerulonephritis similar to that described in U.S.A. by Lerner and Dixon is not present in sheep in New Zealand.

Because Campylobacter fetus is a relatively common cause of ovine abortion in New Zealand sheep the absence of primary glomerulonephritis

casts some doubt on the observation made by Den Boer (1969) that the glomerulopathy observed in her work was the result of infection by this organism.

CONCLUSION

The primary aim of this research was to provide information on the pathology of renal lesions in sheep. The overall prevalence of kidney disease in 13,988 mature sheep killed in five consecutive days in a slaughter-house was 3.18 per cent.

Of the 830 diseased kidneys collected the most prevalent gross lesions noted were in order, spots and streaks; scars; pale, brown and red discolouration; and cysts. In addition, abscesses, parasitic cysts or nodules, neoplasms and infarcts were found.

One hundred and eighty one of the abnormal kidneys were examined histologically. Those lesions designated grossly as spots and streaks were focal or multifocal areas of chronic inflammatory change of undetermined aetiology. Most of the scars were of either pyelonephritic or infarctive origin. The cystic lesions were due either to urinary retention caused by inflammatory and congenital obstruction of tubules, or Echinococcus granulosus hydatid cysts. Pallor in swollen kidneys was associated with nephrosis while red discolouration, either patchy or diffuse, was caused by congestion of blood vessels. These lesions were acute and probably occurred close to the time of slaughter. The brown discolouration in kidneys was caused by haemosiderin deposition which was in most cases probably the result of prior haemolysis due to infection by Eperythrozoon ovis. The neoplastic lesions were an adenoma, a carcinoma and a malignant lymphoma.

Primary glomerulonephritis as reported elsewhere was not present

in sheep examined in this survey. All glomerular lesions observed were secondary to the other pathological changes.

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APPENDIX I

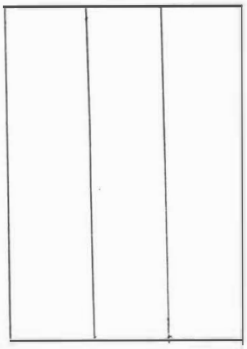
FORMAT FOR RECORDING OF HISTOPATHOLOGICAL LESIONS

CASE NUMBER:

GLOMERULAR LESION
Distribution of lesion
- Focal (F)
- Focal radial (F/R)
- Multifocal (M/F)
- Multifocal radial (M/R)
- Diffuse (D)

Site of lesion
- Basement membrane (BM)
- Capillary network (CN)
- Mesengium (MES)
- Bowman's capsule (BC)
- Urinary space
  - dilated with casts
  - reduced

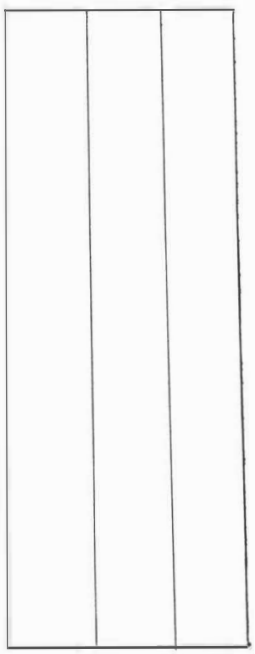
Nature of lesion
- Proliferative
- Inflammatory
  - neutrophils
  - eosinophils
  - lymphocytes
  - plasma cells
- Deposition
  - membranous
  - sclerotic
  - amyloid
- Segmental
- Panglomerular



TUBULAR LESION
Distribution of lesion
- Focal (F)
- Focal radial (F/R)
- Multifocal (M/F)
- Multifocal radial (M/R)
- Diffuse (D)

Site of lesion
- Proximal convoluted tubule (PCT)
- Descending tubule (DT)
- Ascending tubule (AT)
- Distal convoluted tubule (DCT)
- Collecting tubule (CT)
- Pantubular (PAN)

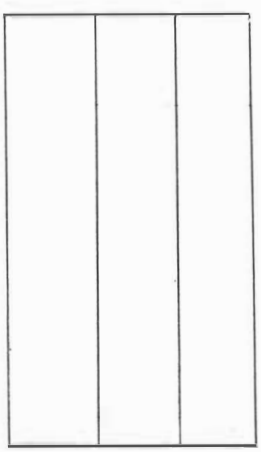
Nature of lesion
- Degenerative
  - cell swelling
  - cell necrosis
- Regenerative
- Inflammatory component
  - cellular
    - neutrophils
    - eosinophils
    - lymphocytes
    - plasma cells
    - mast cells
    - phagocytes
    - polykaryons
    - fibroblasts
  - casts
    - pure hyaline
    - hyaline granular
    - hyaline fatty
    - hyaline RBC
    - haemoglobin
    - bile
    - calcium
    - uric acid
    - oxalate
- Metaplasia
- Deposition
  - membranous
  - calcification
  - pigments
    - haemosiderin
    - others



INTERSTITIAL LESION
Distribution of lesion
- Focal (F)
- Focal radial (F/R)
- Multifocal (M/F)
- Multifocal radial (M/R)
- Diffuse (D)

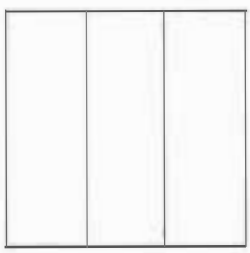
Site of lesion
- Cortex
- Medulla
- Cortex/Medulla

Nature of lesion
- Inflammatory
  - neutrophils
  - eosinophils
  - lymphocytes
  - plasma cells
  - mast cells
  - phagocytes
  - polykaryons
  - fibroblasts
- Deposition
  - membranous
  - collagen
  - amyloid
  - pigments
    - haemosiderin
    - others
  - mineralisation
  - calcification
- Vascular lesions
  - thrombus
  - congestion
  - haemorrhage



PELVIC LESION
Distribution of lesion
- Focal (F)
- Multifocal (M/F)
- Diffuse (D)

Nature of lesion
- Degenerative
  - cell swelling
  - cell necrosis
  - desquamation
- Regenerative
- Metaplastic
- Inflammatory
  - neutrophils
  - eosinophils
  - lymphocytes
  - plasma cells
  - mast cells
  - phagocytes
  - polykaryons
  - fibroblasts



NEOPLASIA

ADDITIONAL COMMENTS

+ = mild
++ = moderate
+++ = severe

APPENDIX II

THE NATURE, DISTRIBUTION AND SEVERITY OF HISTOLOGICAL LESIONS PRESENT IN 181 DISEASED KIDNEYS ARRANGED IN GROUPS ACCORDING TO THE MAJOR GROSS PATHOLOGICAL LESION. THE LESIONS ARE GRADED ACCORDING TO THE FORMAT IN APPENDIX I

1) DISCOLOURATION

a) PALE

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
7A				
Lesion Distribution	D	M/F		
" Site	PCT/DT/AT/DCT	cortex/medulla		
" Nature	cell swelling (++) granular (+) hyaline RBC (+)	congestion (+)		
2B				
Lesion Distribution	D	F	M/F	
" Site	PCT/DT/AT/DCT	cortex	cortex/medulla	
" Nature	cell swelling (++) granular (+) hyaline RBC (+)	lymphocytes (+) plasma cells (+)	congestion (+)	
219				
Lesion Distribution	M/F	M/F		
" Site	PCT/DT/AT/DCT	cortex	medulla	
" Nature	cell swelling (+++) neutrophils (++) lymphocytes (++) plasma cells (++) fibroblasts (++) pure hyaline (++) granular (++)	fibroblasts (+++) lymphocytes (++) plasma cells (++) neutrophils (+)	fibroblasts (+++)	
241				
Lesion Distribution	M/F		M/F	
" Site	PCT/DCT		BM/CN/BC	
" Nature	cell swelling (++) Pure hyaline (+) granular (+)		membranous (+) panglomerular	
345				
Lesion Distribution	D			
" Site	PCT/DCT			
" Nature	cell swelling (++) granular (++)			
354				
Lesion Distribution	D	M/F		
" Site	PCT/DCT	cortex/medulla		
" Nature	cell swelling (+++) granular (+++)	congestion (+)		
368				
Lesion Distribution	M/F	M/F		
" Site	PCT/DCT	cortex		
" Nature	cell swelling (++) granular (++)	lymphocytes (++) plasma cells (++)		
370 - See No. 348				
372				
Lesion Distribution	M/F			
" Site	PCT/DCT			
" Nature	cell swelling (+) granular (+)			

b) RED

5A				
Lesion Distribution		M/F	M/F	
" Site		cortex/medulla	BM/CN/BC	
" Nature		congestion (+)	membranous (+) segmental	
58				
Lesion Distribution		M/F		
" Site		cortex/medulla		
" Nature		lymphocytes (+) plasma cells (+) congestion (+)		
29				
Lesion Distribution		M/F	D	
" Site		cortex	medulla	
" Nature		congestion (+)	congestion (+)	
48A - See No. 29				
56				
Lesion Distribution		M/F		
" Site		cortex/medulla		
" Nature		congention (+)		
60				
Lesion Distribution	M/F	M/F		
" Site	CT	cortex/medulla		
" Nature	calcium casts (+)	congestion (+)		
62 - See No. 56				
78 A				
Lesion Distribution		M/F	M/F	
" Site		cortex	cortex/medulla	
" Nature		lymphocytes (+) plasma cells (+)	congestion (+)	

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
78B - See No.56				
129 - See No. 78A				
145 Lesion Distribution " Site " Nature		M/F cortex/medulla congestion (++)		
160 - See No. 145				
186 - See No. 56				
424 - See No. 56				

c) BROWN

133A & B Lesion Distribution " Site " Nature	M/R PAN cell swelling (+++) regeneration (++) lymphocytes (++) plasma cell (++) pure hyaline (++) granular (++) haemoglobin (+) calcium (+) metaplasia (++) haemosiderin (+++)	M/R cortex/medulla lymphocytes (+++) plasma cells (+++) fibroblasts (+++) collagen (+++) haemosiderin (+++)	M/R BM/CN/BC/MES membranous (+++) sclerotic (+++) panglomerular	M/F desquamation (++) lymphocytes (++) plasma cells (++)
141 Lesion Distribution " Site " Nature	D PCT/ DCT haemosiderin (+++) M/F PCT/DCT haemoglobin (+)	D cortex/medulla congestion		
163 Lesion Distribution " Site " Nature	D PCT/DCT haemosiderin (+++)	M/F cortex/medulla neutrophils (+) lymphocytes (++) plasma cells (++) phagocytes (++)		
187 Lesion Distribution " Site " Nature	D PCT/DCT haemosiderin (+)		D BM/CN/BC membranous (+) panglomerular	
201A & B Lesion Distribution " Site " Nature	D PCT/DCT cell swelling (++) haemosiderin (+++)		M/F urinary space dilated (+) haemosiderin (+)	
239 - See No. 155				
240 Lesion Distribution " Site " Nature	D PCT/DCT cell swelling (+) haemosiderin (+)	M/F cortex/medulla congestion (+)	D BM/CN/BC membranous (+) segmental	
245 Lesion Distribution " Site " Nature	D PCT/DCT cell swelling (+) haemosiderin (+++)		D BM/CN/BC membranous (+) segmental	
246 - See No. 155				
250 Lesion Distribution " Site " Nature	D PCT/DCT cell swelling (+) haemosiderin (+) M/F lymphocytes (+) plasma cells (++)	M/F (2 foci) cortex lymphocytes (++) plasma cells (++)	D BM/CN/BC membranous (+) segmental M/F (2 foci) membranous (++)	
308 Lesion Distribution " Site " Nature	M/R PCT/DCT/CT hyaline casts (++) lymphocytes (+) plasma cells (+) calcium casts (+) D PCT/DCT cell swelling (++) haemosiderin (++)	M/R cortex lymphocytes (++) plasma cells (++) fibroblasts (+) collagen (+) haemosiderin (+)	M/R BM/CN/BC membranous (++) sclerotic (++) segmental	
331 Lesion Distribution " Site " Nature	D PCT/DCT haemosiderin (+)			
365 Lesion Distribution " Site " Nature	F/R PCT/DCT cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) pure hyaline (++) membranous (++) D PCT/DCT cell swelling (+) haemosiderin (+)	F/R cortex lymphocytes (++) plasma cells (+) fibroblasts (++) membranous (++)	F/R BM/CN/BC membranous (+) sclerotic (+) segmental	
349 - See No. 155				

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
341				
Lesion Distribution	D			
" Site	PCT/DCT			
" Nature	cell swelling (+) haemosiderin (++)			
416 - See No. 341				
		2) SPOTS		
2				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (+) lymphocytes (+) plasma cells (+) phagocytes (+)	lymphocytes (+) plasma cells (+) phagocytes (+) fibroblasts (+) collagen (+)	mebranous (+) sclerotic (+) segmental	
6A & B				
Lesion Distribution		M/F		
" Site		cortex		
" Nature		lymphocytes (+) plasma cells (+) phagocytes (+)		
9A & B				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (+) granular casts (+) pure hyaline (+)	lymphocytes (++) plasma cells (++) fibroblasts (++) collagen (++)	membranous (+) panglomerular	
19				
Lesion Distribution	M/F	M/F		
" Site	PCT/DCT	cortex/medulla		
" Nature	lymphocytes (+) plasma cells (+) haemosiderin (+)	lymphocytes (++) plasma cells (++) congestion (+)		
24 - See No. 6A & B				
28				
Lesion Distribution	M/F (10 foci)	M/F (10 foci)		
" Site	PCT/DCT	cortex		
" Nature	lymphocytes (+) plasma cells (+)	lymphocytes plasma cells		
33				
Lesion Distribution		M/F (9 foci) M/F		
" Site		cortex medulla		
" Nature		lymphocytes (+) congestion (+) plasma cells (+)		
37				
Lesion Distribution		M/F (2 foci)	M/F (2 foci)	
" Site		cortex	BM/CN/BC	
" Nature		lymphocytes (+) plasma cells (+)	membranous (+) panglomerular	
41 A				
Lesion Distribution	M/F (10 foci)	M/F (10 foci)	M/F (10 foci)	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	lymphocytes (+) plasma cells (+)	lymphocytes (++) plasma cells (++) congestion (+)	membranous (+) segmental	
41B				
Lesion Distribution	as 41A plus	as 41A	as 41A	
" Site	hyaline granular (+)			
" Nature	hyaline RBC casts (+)			
47A & B				
Lesion Distribution	M/F (10 foci)	M/F (10 foci)	M/F (10 foci)	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	lymphocytes (+) plasma cells (+) pure hyaline (+) membranous (+)	lymphocytes (++) plasma cells (++) congestion (+)	membranous (+) panglomerular	
48B				
Lesion Distribution	M/F (4 foci)	M/F (4 foci) D	M/F (4 foci)	
" Site	PCT/DCT	cortex medulla	BM/CN/BC	
" Nature	lymphocytes plasma cells (+)	lymphocytes (+) congestion (++) plasma cells (+)	membranous (++) panglomerular	
49A - See No. 6A				
50				
Lesion Distribution	M/F (6 foci)	M/F (6 foci)	M/F (6 foci)	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	membranous (+)	lymphocytes (+) plasma cells (+)	Membranous (++) sclerotic (++) segmental	
73				
Lesion Distribution		M/F (6 foci)		
" Site		cortex		
" Nature		lymphocytes (++) plasma cells (++) membranous (+)		
86				
Lesion Distribution	D	M/F		
" Site		cortex		
" Nature	PCT/DCT haemosiderin (+)	lymphocytes (++) plasma cells (++)		

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
80				
Lesion Distribution	M/F (9 foci)	M/F (9 foci)	M/F (9 foci)	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	lymphocytes (+) plasma cells (+) pure hyaline (+)	lymphocytes (+) plasma cells (+)	membranous (++) panglomerular	
89A				
Lesion Distribution	D	D	M/F	
" Site	PCT/DCT	cortex/medulla	cortex	
" Nature	haemosiderin (+++)	congestion (++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++)	
89B				
Similar to 89A with exception of the M/F interstitial lesion				
92				
Lesion Distribution	M/F	M/F	M/F (few foci)	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	lymphocytes (+) plasma cells (+) pure hyaline (+)	lymphocytes (++) plasma cells (++) fibroblasts (++) congestion (+)	membranous (++) panglomerular	
93				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BN/CN/BC	
" Nature	lymphocytes (+) plasma cells (+)	lymphocytes (+) plasma cells (+) fibroblasts (+) congestion (+)	membranous (+) segmental	
		M/F medulla congestion (+)		
103				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	lymphocytes (+) plasma cells (+) pure hyaline (+)	lymphocytes (++) plasma cells (++) fibroblasts (++)	membranous (+) panglomerular	
109				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (+) pure hyaline (+) granular (+)	lymphocytes (+) plasma cells (+) phagocytes (+)	membranous (++) sclerotic (+) panglomerular	
116 - See No. 86				
118				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC/MES	
" Nature	cell swelling (+) regeneration (+) pure hyaline (+) membranous (+) haemosiderin (+)	lymphocytes (++) plasma cells (++) fibroblasts (++)	proliferative (+) membranous (+) panglomerular	
119A&B				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC/MES	desquamation (++)
" Nature	cell swelling (+++) regeneration (++) neutrophils (+) lymphocytes (++) plasma cells (++) phagocytes (++) pure hyaline (++) granular (++) membranous (++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++) membranous (+++)	proliferative (+++) membranous (+++) panglomerular	regeneration (++) lymphocytes (++) plasma cells (++) fibroblasts (++)
152				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (+) regeneration (+) pure hyaline (+) hyaline RBC (+)	lymphocytes (++) plasma cells (++) fibroblasts (++) collagen (++)	membranous (+) panglomerular	
170 - See No. 119A & B				
185				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/BC	
" Nature	cell swelling (+) regeneration (+) lymphocytes (+) plasma cells (+) pure hyaline (+) haemosiderin (+)	lymphocytes (++) plasma cells (++) membranous (+)	membranous (+) segmental	
116				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/BC	
" Nature	cell swelling (+++) regeneration (++) neutrophils (++) lymphocytes (++) plasma cells (++) granular casts (+)	neutrophils (+) lymphocytes (++) plasma cells (++)	membranous (+) segmental	
213				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (++) regeneration (++) lymphocytes (+) plasma cells (+) pure hyaline (++) granular casts (++)	lymphocytes (++) plasma cells (++) phagocytes (+) fibroblasts (++) collagen (++)	membranous (+++) sclerotic (+++) panglomerular	

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
225				
Lesion Distribution	M/F	M/F		
" Site	PCT/DCT	cortex	M/F	
" Nature	cell swelling (+)	lymphocytes (+)	cortex/medulla	
	regeneration (+)	plasma cells (+)	congestion (+)	
	lymphocytes (+)			
	plasma cells (+)			
243				
Lesion Distribution	F	F	F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (+)	lymphocytes (++)	membranous (++)	
	lymphocytes (+)	plasma cells (++)	panglomerular	
	plasma cells (+)	fibroblasts (++)		
254				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	M/F	BM/CN/BC
" Nature	cell swelling (+)	lymphocytes (+)	cortex/medulla	membranous (+)
	regeneration (+)	plasma cells (+)	congestion (+)	segmental
	lymphocytes (+)	fibroblasts (+)		
264 - See No. 225				
285				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+)
" Nature	cell swelling (+++)	lymphocytes (+++)	membranous (+++)	regeneration (+)
	regeneration (+++)	plasma cells (+++)	sclerotic (+++)	lymphocytes (+)
	lymphocytes (++)	fibroblasts (+++)	segmental	fibroblasts (+)
	plasma cells (++)	collagen (+++)		
	pure hyaline (+++)			
	granular (+++)			
	metaplasia (++)			
298B				
Lesion Distribution	M/F	M/F		
" Site	PCT/DCT	cortex		
" Nature	cell swelling (++)	lymphocytes (++)		
	regeneration (+)	plasma cells (++)		
	lymphocytes (++)	phagocytes (++)		
	plasma cells (++)			
	phagocytes (++)			
304 - See No. 285				
319 - See No. 6A				
320A & B				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cellswelling (+)	lymphocytes (+)	membranous (++)	
	pure hyaline (+)	plasma cells (+)	sclerotic (++)	
			segmental	
359				
Lesion Distribution	F	F		
" Site	PCT/DCT	cortex		
" Nature	cell swelling (+)	lymphocytes (++)		
	regeneration (+)	plasma cells (+)		
	lymphocytes (+)			
	plasma cells (+)			
408				
Lesion Distribution	F	F		
" Site	PCT/DCT	cortex		
" Nature	cell swelling (+++)	lymphocytes (++)		
	regeneration (+++)	plasma cells (++)		
	granular casts (+)	fibroblasts (++)		
	metaplasia (+++)	collagen (++)		
420 - See No. 298B				
426				
Lesion Distribution	M/F	M/F		
" Site	PCT/DCT	cortex		
" Nature	lymphocytes (+)	lymphocytes (+)		
	plasma cells (+)	plasma cells (+)		
436 - See No. 6A				
438				
Lesion Distribution	M/F	M/F	M/F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (+)	lymphocytes (+)	membranous (+)	
	pure hyaline (+)	plasma cells (+)	segmental	
444A 4B				
Lesion Distribution	M/R D	M/R	M/R	
" Site	PCT/DCT PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (++) haemosi-	lymphocytes (++)	membranous (+)	
	regeneration(++) siderin	plasma cells (+)	sclerotic (+)	
	lymphocytes (++) (+)	fibroblasts (++)	segmental	
	plasma cells (++)	collagen (++)		
	fibroblasts (++)			
	pure hyaline (++)			
		3) SCARS		
1				
Lesion Distribution	M/F M/R	M/R	M/R	
" Site	cortical PCT/DCT	cortex	BM/CN/BC	
" Nature	cysts lymphocytes (++)	lymphocytes (+)	membranous (+)	
	(++) plasma cells (++)	plasma cells (+)	panglomerular	
	fibroblasts (++)	fibroblasts (+)		
	pure hyaline (+)			

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
3A				
Lesion Distribution	F/R	F/R	F/R	
" Site	PCT/DT/AT/DCT	cortex/medulla	BM/CN/BC	
" Nature	necrosis (++) lymphocytes (++) plasma cells (++) phagocytes (+) fibroblasts (+) pure hyaline (+)	lymphocytes (+) plasma cells (+) phagocytes (+) fibroblasts (+)	membranous (++) panglomerular	
3B				
Lesion Distribution	M/R	M/R	M/R	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	lymphocytes (+) plasma cells (+)	lymphocytes (+) plasma cells (+)	membranous (+) panglomerular	
4A				
Lesion Distribution	F/R	F/R	F/R	F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+)
" Nature	lymphocytes (+++) plasma cells (+++) phagocytes (++) fibroblasts (+++) metaplasia (++)	lymphocytes (+++) plasma cells (+++) phagocytes (+) fibroblasts (+++) collagen (++) membranous (++)	membranous (++) sclerotic (+) panglomerular	metaplasia (+) lymphocytes (+) plasma cells (+) fibroblasts (+)
4B				
Lesion Distribution	M/R	M/R	M/R	
" Site	cortical	cortex	BM/CN/BC	
" Nature	lymphocytes (+) plasma cells (+) phagocytes (+) fibroblasts (+)	lymphocytes (+) plasma cells (+) phagocytes (+) fibroblasts (+)	membranous (+) panglomerular	
26 - See No. 4B				
35				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+++)
" Nature	cell swelling (++) regeneration (+++) neutrophils (+) lymphocytes (+++) plasma cells (+++) phagocytes (++) fibroblasts (++) pure hyaline (++) granular (++) metaplasia (+++)	neutrophils (+) lymphocytes (+++) plasma cells (+++) phagocytes (++) fibroblasts (++) collagen (+++)	membranous (+++) sclerotic (+++) panglomerular	regeneration (+++) neutrophils (+) lymphocytes (+++) plasma cells (+++) phagocytes (+++)
49B				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	as case 35 plus	as case 35 plus	as case 35	as case 35 plus
" Nature	haemosiderin (++)	haemosiderin (++)		fibroblasts (++)
57				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	lymphocytes (++)
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) fibroblasts (++) membranous (++)	lymphocytes (++) plasma cells (++) fibroblasts (++) congestion (++)	membranous (++) sclerotic (+) segmental	plasma cells (++) fibroblasts (++)
58 - See No. 3A				
63				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	Lesions similar with case No. 35 except neutrophils			
" Nature				
67 - See No. 63				
68 - See No 63				
77				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	lymphocytes (+)
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) pure hyaline (++) metaplasia (++)	lymphocytes (++) plasma cells (++) fibroblasts (++) collagen (++) congestion (++)	membranous (++) sclerotic (++) panglomerular	plasma cells (+) fibroblasts (+)
82				
Lesion Distribution	F/R	F/R	F/R	
" Site	PAN	cortex/medulla	BM/CN/BC	
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) fibroblasts (++) pure hyaline (++) granular (++)	lymphocytes (++) plasma cells (++) fibroblasts (++)	membranous (+) sclerotic (+) segmental	
98				
Lesion Distribution	F/R	F/R	F/R	F
" Site	PAN	cortex/medulla	BM/CN/BC/ur. spaces	lymphocytes (+)
" Nature	cell swelling (+++) regeneration (+++) lymphocytes (+++) plasma cells (+++) pure hyaline (++) granular (++) membranous (++) calcification (++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++)	membranous (+++) sclerotic (++) reduced (++) panglomerular	plasma cells (+) fibroblasts (+)

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
74				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC/MES	lymphocytes (++)
" Nature	cell swelling (+++) regeneration (+++) lymphocytes (+++) plasma cells (+++) pure hyaline (+++) metaplasia (+++) membranous (+++) haemosiderin (++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++) collagen (++) haemosiderin (++)	proliferative (+++) membranous (+++) sclerotic (+++) segmental	plasma cells (++) fibroblasts (++)
76 - See No. 74				
105				
Lesion Distribution	F/R	F/R	F/R	F
" Site " Nature	All lesions similar with case No. 98 plus diffuse tubular (PCT/DCT) haemosiderin (+++)			
107 - See No. 98				
114				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site " Nature	All lesions similar with case No. 98 plus tubular granular (+++) and hyaline RBC casts (+++)			
117				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+)
" Nature	cell swelling (+++) regeneration (+++) neutrophils (+) lymphocytes (+) plasma cells (+) phagocytes (+) fibroblasts (+) pure hyaline (+++) hyaline RBC (+++) granular (+++)	D PCT DCT haemosi- derin (+++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++) haemosiderin (++) congestion (++)	membranous (++) sclerotic (+) panglomerular	regeneration (+) lymphocytes (+) plasma cells (+) fibroblasts (+)
120				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	lymphocytes (++)
" Nature	cell swelling (+++) regeneration (+++) lymphocytes (++) plasma cells (++) pure hyaline (+++) granular (+++) membranous (++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++)	membranous (+++) sclerotic (+) segmental	plasma cells (++)
128				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+)
" Nature	cell swelling (++) regeneration (++) lymphocytes (+) plasma cells (+) fibroblasts (+) hyaline RBC (+) hyaline fatty (+) granular casts (+) membranous (+)	lymphocytes (++) plasma cells (++)	membranous (++) segmental	regeneration (+) lymphocytes (+) plasma cells (+)
130A & B				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC/MES	desquamation (++)
" Nature	cell swelling (+++) regeneration (+++) lymphocytes (+++) plasma cells (+++) fibroblasts (+++) pure hyaline (+++) hyaline RBC (+++) granular (+++) metaplasia (+++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++) membranous (++)	membranous (+++) proliferation (+++) sclerotic (+++) segmental	regeneration (++) lymphocytes (++) plasma cells (++) fibroblasts (++)
134				
Lesion Distribution	F/R	F/R	F/R	
" Site	cortical	cortex	BM/CM/BC	
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) fibroblasts (+) pure hyaline (++) granular (+) haemosiderin (+)	lymphocytes (++) plasma cells (++) fibroblasts (++)	membranous (+) panglomerular	
136				
Lesion Distribution	F/R	F/R	F/R	F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+)
" Nature	cell swelling regeneration (++) lymphocytes (+) plasma cells (+) pure hyaline (++) hyaline RBC (++) granular (++) metaplasia (+) haemosiderin (+)	lymphocytes (+) plasma cells (+) fibroblasts (++)	membranous (+) sclerotic (+) segmental	regeneration (+) lymphocytes (+) plasma cells (+) fibroblasts (+)
166				
Lesion Distribution	F/R	F/R	F/R	
" Site	cortical	cortex	BM/CN/BC	
" Nature	cell swelling (++) pure hyaline (+) granular (+) haemosiderin (++)	lymphocytes (+) plasma cells	membranous (+) segmental	

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
138				
Lesion Distribution	F/R	F/R	F/R	F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (++)
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) pure hyaline (++) granular (++) hyaline RBC (++)	lymphocytes (++) plasma cells (++) fibroblasts (++) congestion (++)	membranous (++) sclerotic (+) panglomerular	regeneration (++) lymphocytes (++) plasma cells (++)
175A & B - See No. 138				
199				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	regeneration (++)
" Nature	cell swelling (++) regeneration (++) lymphocytes (+) plasma cells (+) pure hyaline (+)	lymphocytes (+) plasma cells (+) phagocytes (+) fibroblasts (+++) collagen (+++)	membranous (++) sclerotic (+) segmental	lymphocytes (++) plasma cells (++) fibroblasts (++)
204				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (++)
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) pure hyaline (++) granular (++)	lymphocyte (++) plasma cells (++) phagocytes (+) fibroblasts (+++) collagen (+++)	membranous (+) segmental	regeneration (++) lymphocytes (++) plasma cells (++)
216				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC/MES	lymphocytes (+)
" Nature	cell swelling (+++) regeneration (+++) lymphocytes (+) plasma cells (+) pure hyaline (+++) hyaline fatty (+++) granular (+++) metaplasia (+++)	lymphocytes (++) plasma cells (+) phagocytes (++) fibroblasts (++) collagen (++)	proliferation (++) membranous (++) sclerotic (++) panglomerular	plasma cells (+)
226				
Lesion Distribution	F/R	F/R	F/R	
" Site	cortical	cortex	BM/CN/BC	
" Nature	cell swelling (++) regeneration (++) lymphocytes (+) plasma cells (+) pure hyaline (+) granular (+)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++) phagocytes (+) collagen (+++)	membranous (++) sclerotic (++) segmental	
228 - See No. 226				
233				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+)
" Nature	cell swelling (++) regeneration (++) granular (++)	lymphocytes (++) plasma cells (++) phagocytes (++) membranous (++) congestion (+)	membranous (+) segmental	lymphocytes (+) plasma cells (+)
234				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (++)
" Nature	cell swelling (+++) regeneration (+++) lymphocytes (++) plasma cells (+++) fibroblasts (++) pure hyaline (++) granular (++) metaplasia (++)	lymphocytes (+++) plasma cells (+++) fibroblasts (+++) collagen (+++)	membranous (+++) sclerotic (+) segmental	regeneration (++) neutrophils (+) lymphocytes (++) plasma cells (++) fibroblasts (++)
248				
Lesion Distribution	F/R	F/R	F/R	
" Site		All lesions similar with case No. 226		
" Nature		but interstitial lesions milder (++)		
252 - See No. 234				
265				
Lesion Distribution	F/R	F/R	F/R	F
" Site	PAN	cortex/medulla	BM/CN/BC	desquamation (+)
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (+++) fibroblasts (++) pure hyaline (++) granular (++) membranous (++)	lymphocytes (++) plasma cells (++) fibroblasts (+++) collagen (+++) membranous (++)	membranous (++) sclerotic (++) segmental	regeneration (+) fibroblasts (+)
277				
Lesion Distribution	M/R	M/R	M/R	M/F
" Site	as 265	as 265	as 265	desquamation (+)
" Nature				regeneration (+) lymphocytes (++) plasma cells (++) fibroblasts (++)
278 - See No. 265				
283, 346, 431 - See No. 226				
293, 298, 392 - See No. 277				
423 - See No. 233				

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS
		4) CYSTS		
22				
Lesion Distribution	M/F	M/F		
" Site	cortical	CT		
" Nature	2 cysts (++)	calcification (+)		
		M/F	M/F	
		cortex	cortex/medulla	
		lymphocytes (+)	congestion (+)	
		plasma cells (+)		
31				
Lesion Distribution	M/F	M/F		
" Site	cortical	cortex/medulla		
" Nature	5 cysts (++)	lymphocytes (++)		
		plasma cells (++)		
55A & B				
Lesion Distribution	F			
" Site	cortical			
" Nature	1 cyst (+++)			
65				
Lesion Distribution	F			
" Site	cortical			
" Nature	1 cyst (++)			
165				
Lesion Distribution	M/F	M/F	M/F	
" Site	cortical	PCT/DCT	cortex (around the cysts)	
" Nature	3 cysts (+++)	cell swelling (++)	lymphocytes (+++)	
		granular casts (+++)	plasma cells (+++)	
		hyaline RBC (+++)	phagocytes (+)	
			fibroblasts (++)	
			collagen (++)	
305				
Lesion Distribution	M/F	F/R	M/F (around cysts)	F/R
" Site	cortical/	PCT DCT	cortex/medulla	BM/CN/BC
" Nature	medullary cysts (+++)	pure hyaline (+++)	lymphocytes (++)	membranous (++)
		metaplasia (+++)	plasma cells (++)	segmental
			fibroblasts (++)	
			collagen (++)	
306				
Lesion Distribution	M/F	M/F		
" Site	cortical	CT		
" Nature	cysts (+++)	calcium casts (+)		
306B - See No. 306A except calcium casts				
383				
Lesion Distribution	M/F	M/F	M/F	M/F
" Site	cortical	PCT/DCT	CT	cortex/around cysts
" Nature	cysts (+++)	granular++ metaplasia (+++)	calcium casts (+)	lymphocytes (++)
				fibroblasts (++)
				collagen (++)
		5) NODULES		
		a) ABSCESSES		
200				
Lesion Distribution	M/F (3 foci)	M/F (3 foci)	M/F (3 foci around abscesses)	M/F
" Site	PAN	cortex/medulla	proliferative (++)	necrosis (+++)
" Nature	necrosis (+++)	neutrophils (+++)	membranous (++)	regeneration (+++)
	regeneration (++)	lymphocytes (++)	segmental	fibroblasts (+++)
	neutrophils (+++)	plasma cells (+++)		lymphocytes (++)
	lymphocytes (++)	fibroblasts (+++)		plasma cells (++)
	plasma cells (++)			phagocytes (++)
	phagocytes (++)			
	pure hyaline (++)			
	granular (++)			
	calcium casts (++)			
217				
Lesion Distribution	F	F	F (aroundabscess)	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	necrosis (+++)	lymphocytes (+++)	membranous (+)	
	regeneration (+++)	plasma cells (+++)	segmental	
	neutrophils (+++)	fibroblasts (+++)		
	lymphocytes (+++)	collagen (+++)		
	plasma cells (+++)	phagocytes (+)		
	fibroblasts (+++)	neutrophils (+)		
257				
Lesion Distribution	F	F	F (around abscess)	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	necrosis (++)	lymphocytes (++)	membranous (+)	
	regeneration (++)	plasma cells (++)	segmental	
	neutrophils (++)	fibroblasts (++)		
	lymphocytes (+)			
	plasma cells (+)			
342				
Lesion Distribution	F	F	F	
" Site	PCT/DCT	cortex	BM/CN/BC	
" Nature	cell swelling (++)	lymphocytes (++)	membranous (+)	
	regeneration (++)	plasma cells (++)	segmental	
	lymphocytes (++)	fibroblasts (++)		
	plasma cells (++)	phagocytes (++)		
	fibroblasts (++)			
	phagocytes (++)			

CASE No.	TUBULES	INTERSTITIUM	GLOMERULUS	PELVIS

b) NEOPLASMS

59 - Renal Adenoma

236 - Malignant Lymphoma

237 - Carcinoma

c) OF UNCERTAIN AETIOLOGY

12				
Lesion Distribution	F	F (around nodule)		F
" Site	cortical	cortex/medulla		BM/BC
" Nature	compression atrophy hyaline casts (+)	fibroblasts (+++) lymphocytes (+) plasma cells (+)		membranous (+) segmental

20 - See No. 12 plus polykaryons (++) in the interstitium

121				
Lesion Distribution	M/F	M/F		M/F
" Site	PCT/DCT	cortex		BM/CN/BC
" Nature	cell swelling (++) regeneration (++) eosinophils (++) lymphocytes (++) plasma cells (++) granular casts (++)	eosinophils (++) lymphocytes (++) plasma cells (++) phagocytes (++) polykaryons (++)		membranous (+) segmental
140				
Lesion Distribution	F	F	M/F	
" Site	PCT/DCT	cortex	cortex/medulla	
" Nature	cell swelling (+) regeneration (+) eosinophils (+) lymphocytes (+) plasma cells (++) granular casts (+)	eosinophils (++) lymphocytes (++) plasma cells (++) fibroblasts (++)	congestion (++)	
177A & B				
Lesion Distribution	M/R (3 foci)	M/R (3 foci)		M/R (3 foci)
" Site	PCT/DT/AT/DCT	cortex/medulla		BM/CN/BC
" Nature	cell swelling (++) regeneration (++) eosinophils (++) lymphocytes plasma cells (++) pure hyaline (++) granular casts (++)	eosinophils (++) lymphocytes (++) plasma cells (++) fibroblasts (++) collagen (++)		membranous sclerotic (+) segmental
190				
Lesion Distribution	F	F		
" Site	PCT/DCT	cortex		
" Nature	cell swelling (++) regeneration (++) eosinophils (++) phagocytes (++) lymphocytes (++) plasma cells (++) pure hyaline (++) granular casts (++)	eosinophils (+++) lymphocytes (+++) plasma cells (+++) phagocytes (+++) polykaryons (++)		
192				
Lesion Distribution	F	D	F	F
" Site	PCT/DCT	PCT/DCT	cortex	BM/CN/BC
" Nature	cell swelling (+++) regeneration (+++) eosinophils (+++) lymphocytes (+++) phagocytes (+++) plasma cells (+++) polykaryons (+++) pure hyaline (++) granular casts (++) hyaline RBC (++) haemosiderin (+++)	haemosiderin (+++)	eosinophils (+++) lymphocytes (+++) plasma cells (+++) phagocytes (+++) polykaryons (+++) fibroblasts (+++)	membranous (+) panglomerular

194 - See No. 190

251			
Lesion Distribution	F	F	F
" Site	PCT/DCT	cortex	BM/CN/BC
" Nature	cell swelling regeneration (+++) eosinophils (+++) lymphocytes (+++) plasma cells (+++) phagocytes (+++) pure hyaline (+++) metaplasia (++)	eosinophils (+++) lymphocytes (+++) plasma cells (+++) phagocytes (++)	membranous (++) sclerotic (++) segmental

336 - See No. 251 plus polykaryous (+) in the interstitium

357			
Lesion Distribution	F	D	F
" Site	PCT/DCT	PCT/DCT	cortex
" Nature	cell swelling (++) regeneration (++) lymphocytes (++) plasma cells (++) eosinophils (++) phagocytes (++)	cell swelling (+) haemosiderin (++)	lymphocytes (+++) plasma cells (+++) eosinophils (+++) phagocytes (++) polykaryons (++)

380 - See No. 357 except diffuse tubular haemosiderosis