Renal failure is one of the most frequent problems in dogs responsible for high mortality rate. Studies on renal failure and its management has focused more attention during the recent year. Mortality of dogs due to renal failure in Ranchi has been a problem for quite a long time. Early diagnosis and satisfactory management of renal failure possess a great challenge to the Veterinary clinicians, owing to lack of precise and easily accessible technique. Various authors have reported on the different aspect of renal failure, its historical background, incidence, Diagnosis and managemental procedures. The literature concerning all these reports was reviewed under the following headings:

1. Historical background
2. Incidence of renal failure
3. Diagnosis of renal failure
   3.1 History and physical examination
   3.2 Haematological changes
   3.3 Biochemical changes
   3.4 Urine analysis
4. Managemental procedures
   4.1 Diuresis
   4.2 Peritoneal dialysis

1. Historical background:
   Abel et al. (1913) performed haemodialysis first time on experimental dogs with a “artificial kidney” composed of celloidin tubes, the predecessor of the modern hollow fibre dialyzer, but their pioneering
efforts were halted during World War-I and discarded for more than a decade.

Ganther (1923) first introduced peritoneal lavage for the treatment of uremic patient.

Renal failure has been diagnosed in dogs since 1930s but was called by the term “nephritis”. Nephritis had been defined as “functional interference with the blood flow through the glomeruli, causing glomerulonephritis. This damage affects all the glomeruli of both kidneys” (Volhard 1936).

Bloom (1939) classified the renal disease based on histological examination and concluded that interstitial nephritis was the most common renal inflammatory disease in dogs and glomerulonephritis was extremely rare.

Martin (1939) studied both acute and chronic phases of the kidney disease, both clinically and histologically and stated that the first evidence of the disease in dogs was ulcerative stomatitis and the most common terminal changes was severe haemorrhagic gastro-enteritis.

Rigdon (1939) in his work, induced nephritis by injecting *Staphylococcus* toxin intravenously in dogs and found that the toxin injured the endothelium of the renal arteries, glomerular capillaries and also the tubular epithelial cells. The renal lesions accompany retention of non-protein nitrogen in the blood.

The clinical diagnosis of kidney failure in dogs with the help of biochemical parameters was started after Allison et al. (1941) analyzed the influence of renal damage on the nitrogenous constituents in blood of the dog and were of the opinion that in dogs after a period of low fluid intake, a low specific gravity of urine correlated with a high BUN and also was of clinical significance in detecting renal impairment.

Uvarov (1944) analyzed urine for the diagnosis of kidney damage in clinical cases of dog.
Kolff and Brek (1944) first of all introduced artificial kidneys with a large area for dialysis with about 20,000 sq. cms. With one patient 24, 40 and 35 grams of urea were dialysed out in 1.5, 4 and 6 hours. Other products like residual Nitrogen, urea, uric acid, creatine and inoxyl could also be removed. They made a statement “We believe that patients suffering from uremia and anuria can be kept alive so long as blood vessels for puncture are available.”

Gilmour (1947) described the cause of osteitis fibrosa in renal failure patients as imbalance of calcium and phosphorus due to hyperparathyroidism. Similar work was carried out by Platt (1951) on the involvement of parathyroid hormone in renal insufficiency, the position and number of glandules in normal and affected dogs was studied. The weight of parathyroid tissue per kg. Body weight was determined and reported a significant increase in the weight of parathyroid tissue due to hyperplasia in chronic nephritis and considerable greater increase in dogs with rubber jaw.

Welt and Peters (1951) used the term ARF first time for lower nephron nephrosis in dogs. They stated a number of etiologies which could cause the condition and localized it to two main reasons (1) some agent toxic for the renal tubule epithelium (2) anoxia of the renal tissue. They also concluded that the increased resorption of water from the damaged tubule might concentrate the toxic agent in the urine, increasing the epithelia injury. A slightly modified version of the same definition was in vogue to that day.

Simonsen (1953) conducted experiments on kidney transplants and explained the immunological reasons for the rejection of the autotransplants.

Dahme (1955) worked on chronic interstitial nephritis (shrunken kidney) in canines and stated that the condition was due to interstitial
lymphohistiocytic nephritis, which resulted in fibrosis due to restricted blood supply.

Radiographs were used in diagnosis of various diseases of kidney during the 1950s. Dyce (1957) used contrast radiography, intravenous pyelography as technique to diagnose diseases of kidney in dogs whereas Tennille and Thornton (1958) worked on intravenous urography.

Bonani (1959) evaluated the blood picture of dogs with chronic nephritis and found increased haemoglobin content, decreased RBC number and slight increased WBC number.

Sastry (1961) found that the clearance tests being used were of little value and could be hazardous in cases of nephritis. He also reported Phenosulphonephthalein test did not indicate the extent of damage and the BUN was not sensitive enough to indicate early renal disease and hence of little use in early stages of nephropathies.

The concept of renal biopsy for the diagnosis of renal diseases was described during 1960s by Garner et al. (1968). He described surgical method of renal biopsy in cattle and Osborne et al. (1968) described percutaneous renal biopsy in cattle.

During 1970s the use of urinary enzymes became popular as a diagnostic tool in kidney damage. Ellis and Price (1973) stated that an increase in the activity of enzymes β-glucuronidase and N-acety-β-D-glucosaminidase in urine was a sensitive indicator of kidney damage and this test could provide information on the area of nephron damage.

Schneider (1973) worked on lysosomal enzymes β-glucuronidase and N-acetyl-β-D-glucosaminidase and was of the opinion that in chronic interstitial nephritis an enlargement of lysozomes in tubular epithelial cells was observed in areas of peritubular fibrosis. He also mentioned that similar situation had been seen in degenerative lesions of the tubular epithelium. Hence an increase in their level was of importance.
Krohn et al. (1973) studied the glomerular changes in canine interstitial nephritis by electron microscopy and reported that there was a thickening of the glomerular capillary walls and an increase in the mesangium. There was presence of sub-endothelial and occasional sub-epithelial electron-dense deposit in the thickened and split glomerular basement membrane.

BUN was considered the best parameter to assess the renal function till, Gabrisch (1973) reported that creatinine was more reliable diagnostic tool compared to that of serum urea. He established normal values of creatinine as 0.71±0.37 mg/100ml in healthy dogs, he also stated the prognosis based on the creatinine values of 2-3 mg/100ml be treated cautiously, 3-4 doubtful and over 4 unfavourable.

Biewenga et al. (1978) worked on the lysosomal enzymes in both urine and blood by lysoplate assay and found that the dogs with reduced glomerular filtration had higher serum lysosome values than normal dogs. The presence of lysosomes in urine was associated with proximal tubular damage. It was concluded that presence of lysosome in the urine could be used as an index of renal tubular damage.

Ultrasonography had made its place in the diagnosis of various diseases during 1980, Cartee et al. (1980) worked on the usefulness of ultrasonography in the diagnosis of renal diseases and found it useful in diagnosis of hydronephrosis, renal calculi and renal neoplasia.

Grauer et al. (1983) worked on the laparoscopic techniques of obtaining renal biopsy. They also confirmed their findings with the post mortem examination. But post biopsy complications were high.

Hager et al. (1985) worked on ultrasound guided renal biopsy and were successful. The post biopsy complications of haematuria were less by this technique.

Karwiec et al. (1986) studied the use of 99m Tc diethylenetriaminepenta acetic acid for assessment of GFR and
compared it with conventionally used tests like creatinine and/or inuline clearance and derived an equation to calculate GFR based on the percentage of dose given. They concluded that use of nuclear medicine was a rapid and non-invasive technique for evaluation of GFR.

2. Incidence:

DeSchepper, (1977) conducted a study on 10,920 cases, he found that 1000 dogs had renal failure.

Scott et. al. (1985), Grauer (1995) and Grauer and Lane (1995b) reported advanced age of ≥ 7 years as one of the risk factors for ARF.

Mary (1992) stated that diuretics were beneficial to facilitates continued diuresis induced by intravenous fluid administration. Furosemide @ 2.2 mg/kg IV is the diuretic of choice. Urine production should increase within 30 minutes following administration of furosemide or the dosage could be doubled or tripled at hourly intervals. Further, he recommended the use of dopamine @ 2 to 5 μg/kg/minutes, a vasodilators to augment renal function and urine production in oligouric animal.

Rubin (1997) mentioned that CRF occured in dogs of all ages, it was commonly considered a disease of older animals, and the incidence increased with age. In survey report, the mean age of CRF was diagnosed in 7 years (n=170) & 6.5 years (n=119).

Stanley (1995) reported that primary renal failure occured in cats and dogs with comparable frequency, with chronic renal failure (CRF) being more common than acute renal failure (ARF).

Behrend et al. (1996) in a retrospective study, found the mean age of ARF patients as 106.9 month (range 5 to 190 months), 20 dogs (69%) were ≥ 7 years old. Seventeen dogs were male and 12 were females. Dogs ≥ 7 years old had a relatively higher risk.
Vaden et. al. (1997) in a study recorded the age of ARF (n=23) patients range from 11 months to 15 years (median 6.9 years), 13 were males and 13 were females. Age of the CRF patients ranged from 6 months to 16 years (Median 6 years), 6 were males and 13 were females.

Sosnar et al. (2003) Studied, 1099 (935 dog & 164 cat) cases admitted to University of Veterinary and pharmaceutical science Brno, during 1990-2001. They found that 139 (111 dog & 28 cat) cases had renal failure (12.7%).

3. Diagnosis of renal failure

Early diagnosis is important for better management of renal failure cases. Hence commonly used diagnostic tools have been reviewed as listed under.

3.1 History and physical examination

History collection is very important to make a diagnosis, and this should be correlated with the physical examination.

English (1974) stated that a sudden history of illness and anorexia with little elevation in body temperature, with a history of either one of the conditions such as shock, massive injury, vascular haemolysis, suspected exposure to nephrotoxic chemicals, an infection in a known case of chronic renal disease, one should suspect acute renal failure. Physical examination should support the rest of the diagnosis. In early oliguric phase, associated with development of uremia, there were signs of mental depression, anorexia, vomition, rapid pulse and elevated respiration rate.

Donald and Larry (1983) reported depression, anorexia, vomiting, dehydration, uremic breath, scleral injection and oral ulcers as symptoms that occurred in acute renal failure.
Shahar and Holmberg (1985) noticed the following symptoms of acute renal failure in two dogs; the first one was a 13 month old Cocker Spaniel which had a one month history of several episodes of urethral obstructions relieved by bladder catheterization. The dog was diarrheic, anorexic, depressed and had begun to vomit and mildly dehydrated. The second one was 9 years old, spayed female. Labrador retriever had been ataxic but was clinically normal on physical examination.

Joshi et al. (1989) reported that the sheep with uremia showed oligurea, reduced water intake, anorexia, depression and uremic odour in the breath. The respirations of these animals were rapid and pulse rate was increased where as body temperature reduced.

Robinson et al. (1989) in their work on chronic renal failure reported that the common presenting signs were anorexia (80 per cent), polydipsia (67 per cent), vomition (67 per cent), lethargy (53 per cent), polyuria (33 per cent), and weight loss (33 per cent). Physical findings included dehydration (40 per cent), lumbar or abdominal pain (33 per cent), oral ulceration (20 per cent) and pale mucous membrane (7 per cent).

Mary (1992) mentioned that acute renal failure patients presented themselves with acute lethargy, anorexia, vomiting, diarrhoea and melena as common clinical signs, and these clinical signs were non-specific for ARF.

Mary (1992) mentioned that there were no age, breed or sex predilection for acute renal failure (ARF). Acute renal failure occurred more frequently than was generally recognized and is often misdiagnosed as chronic renal failure (CRF).

Clement et al. (1993) observed the clinical signs in induced uremic patients, which comprised of oliguria, constipation, altered gait,
depression, loss of appetite, diarrhoea and congestion of buccal mucous membrane.

Forrester and Brandt (1994) stated that patients with acute renal failure generally presented with an acute onset of non-specific clinical signs that may included lethargy, inappetence, vomition, diarrhoea, dehydration and in some cases oral ulceration.

Grauer (1995) mentioned that ARF was common in dogs with pyometra and Escherichia coli endotoxin induced urine-concentrating defects.

Behrend et al. (1996) diagnosed ARF if at least one of the following criteria was met; BUN or serum creatinine concentration had been within reference limits less than 2 weeks prior to the onset of renal failure, BUN and serum creatinine concentration had increased by a factor of at least three within seven days prior to the diagnosis of renal failure, histologic examination of biopsy or necropsy specimens of renal tissue revealed evidence of acute tubular damage, clinical signs of ARF (eg. Anorexia, vomiting and diarrhoea) had commenced acutely and BUN and serum creatinine concentration had returned to reference limits following treatment.

Rubin (1997) mentioned that the historical features of CRF would vary depending on the nature, severity, duration, rate of progression of underlying disease, presence of co-existing but unrelated disease, age and species of the patient. Some of the signs might be vague and non-localizing such as depression, fatigue, anorexia and weight loss. In physical examination, dehydration, dry mucous membranes, decreased skin turgor, and sunken eye balls were found to be common. Renomegaly was associated with hydronephrosis, renal tumors, and amyloidosis. Other abnormalities included oral ulceration, pale mucous membrane and retinal lesions.
Vadan *et al.* (1997) reported that it was difficult to differentiate acute renal failure from chronic renal failure in dogs but it was important for providing clients with a prognosis. Accurate identification of ARF was crucial so that aggressive treatment could be instituted early thereby affording patients the best chance for survival. Further, they distinguished the dogs with acute renal failure from those with chronic renal failure by fulfillment of three or more of the following criteria: history of recent nephrotoxin exposure, ischemic or traumatic event; clinical signs of disease less than two weeks duration, no history of prior azotemia, normal or large sized kidneys with smooth contour as detected by physical examination, survey radiography or ultrasonography, histopathological lesions of acute renal failure and return to normal renal function.

Vaden (2000) worked on the differentiation of ARF and CRF. It was reported that history of prolonged duration of decreased appetite, vomiting, diarrhoea, weight loss, or a combination of these states and polyuria/polydipsia (PU/PD) and nocturia was more consistent diagnosis of CRF. Clinical signs (depression and gastrointestinal ulceration) might be more severe in animals with ARF than in CRF for any magnitude of azotemia. Thinness, poor coat quality, and pallor were more common in CRF.

Haller (2002) reported that a complete history should be taken including age, breed, husbandry and current complain. Polyuria and polydipsia were the first clinical signs of renal disease in dogs. It should be determined if the animal had received any drugs causing polydipsia and polyuria (glucocorticoids or diuretics). Symptoms of reduced appetite, weight loss, vomiting, halitosis, weakness and a poor condition of hair coat were common. A complete physical examination should be performed. It was very important to notice any dehydration, as laboratory results were to be interpreted in conjunction with hydration status. In advanced cases of renal disease, pallor of the mucous membranes or
ulcerations in the oral cavity might be found. Signs of hypertension such as retinal edema, detachment, hemorrhage or vascular tortuosity might be found on fundic examination. In early stages of renal disease, clinical signs might be mild and non-specific or totally absent. Then making laboratory evaluation of blood and urine samples was essential for diagnosis.

Cowgill and Francy (2005) Stated that a complete physical examination was an essential component of the diagnostic approach and might define the location and cause of the uremic state. Due to the acute nature of this condition most animals in ARF had good body and coat condition. They had variable degree of hydration, depression, hypothermia, oral ulceration, “uremic breath”, bile-stained fur, scleral injection, cutaneous bruising, discoloration or necrosis of the tongue, tachycardia or bradycardia, tachypnea, muscle fasciculation’s and seizure were present in proportion to the severity and duration of the azotemia. Bilaterally enlarged, firm, slightly resilient or painful kidneys noted on abdominal palpation was indicative of acute nephrosis or bilateral urethral obstruction.

3.2 Haematological changes:

Hall and Follett (1972) evaluated the normal haemoglobin values in 2 healthy dogs as 13.8 and 10.6 gm/100ml and compared them with the normal reference value reported as 13.6-18.9 gm/100 ml.

Osborne et al. (1972) stated that in polysystemic diseases (bacterial endocarditis, pyometra and leptospirosis) which involved the urinary system and tissues were often associated with leucocytosis.

Finco et al. (1978) reported that dogs with azotemia showed decrease in Hb and PCV and the values were 9.2 gm/dl and 28%, respectively.
Ganesh et al. (1981) recorded the value of Hb, PCV, TEC and TLC as 6 g/dl, 17%, $4 \times 10^6/\mu l$ and $13 \times 10^3/\mu l$ respectively in a dog with acute renal failure.

DiBartola et al. (1985) observed leucocytosis with neutrophilia in acute renal failure due to ethylene glycol toxicity in a English setter dog.

Jain (1986) mentioned the normal haemoglobin value in dogs as 12-18 gm/dl with an average of 15 gm/dl. He also reported that the mean haemoglobin values in different age group as 10.4±0.58 at 6-8 weeks, 11.8±0.81 at 9-12 weeks, 14.4±0.82 at 4-6 months, 15.9±1.2 at 1-2 years and 16.6±1.1 gm/dl above 2 years of age.

Jain (1986) stated that the haematocrit value of normal healthy dogs ranged from 37-55% with an average of 45%.

Jain (1986) opined that the range of TEC in a normal healthy dog to be 5.5-8.5$\times 10^6/\mu l$ with an average of 6.8$\times 10^6/\mu l$. He also noted that the difference among various age groups of Basenji dogs like 6-8 weeks, 9-12 weeks, 4-6 months, 1-2 years and >2 years having mean TEC values as 4.73±0.38, 5.45±0.54, 6.56±0.46, 6.91±0.60 and 7.19±0.64$\times 10^6/\mu l$, respectively.

Joshi et al. (1989) reported that the haemograms of uremic sheep revealed decreased Hb and PCV, whereas leukograms revealed leucocytosis, neutrophilia and lymphopenia.

Robinson et al. (1989) in their work on chronic renal disease in bull terrier found mean values of haematology in diseased group as follows PCV, Hb and TLC to be 35%, 13g/dl and 17,280 cells/$\mu l$, respectively.

Dighe et al. (1990) observed that, there was no significant changes in the levels of Hb, PCV, TEC, TLC and DLC values in experimentally induced ARF in dogs.

Forrester and Brandt (1994) stated that leucocytosis might occur in patient with nephritis and bacterial endocarditis. It had been also observed in dogs with ethylene glycol toxicosis due to stress.

Forrester and Brandt (1994) stated that, non-regenerative anaemia, although most consistent with CRF might occur in patient with ARF especially, when there is concurrent infection or inflammatory diseases such as Rocky mountain spotted fever and leptospirosis.

Jayathangaraj et al. (1995) evaluated the level of PCV as 42% and leucocytosis was observed in a dog with acute renal failure.

Hurley (1998) reported in ARF, a complete blood count might reveal a regenerative blood loss anaemia, thrombocytopenia and a stress or inflammatory leucogram.

Christoper and Larry (2000) reported that infection in dogs with *leptospira interrogans*, typically caused a hepatonephric syndrome that characterized by acute haemorrhagic diathesis, sub-acute icterus or sub-acute uremia. The authors recognized azotemia due to leptospirosis in 37 cases of which 17 had leucocytosis.

Vaden (2000) stated that the presence of non-regenerative anaemia had an indicator of CRF, however anaemia might occur in cases of ARF. In his study he found 32% of dog with ARF were anaemic.

Mrudula et al. (2005) found that there was a significant reduction in Haemoglobin (Hb), Packed Cell Volume (PCV), and Total Erythrocyte Count (TEC) while leucocytosis associated with neutrophilia in cases of nephritis.

3.3 Biochemical changes:

Deavers *et al.* (1963) estimated the mean albumin value as 2.68±0.14 gm/dl in 18 healthy dogs.
Lane and Robinson (1970) estimated the normal ranges of various blood constituent in 49 healthy working dogs of various breeds and sexes. The mean total plasma protein was observed as 6.3 gm/100ml and it ranged between 5.7 – 7.3 gm/100 ml.

Kaneko and Cornelius (1971) observed the absolute total protein and albumin values in plasma of 20 normal dogs as 6.3±0.4 and 3.36±0.33 gm/dl, respectively.

Kirk (1983) estimated the total protein and A:G ratio in serum of normal healthy dogs to be 5.4-7.1 gm/dl and 0.8-1.7, respectively.

DiBartola (1985) reported azotemia (BUN, 126 mg/dl, creatinine, 12 mg/dl) with Hyperkalemia in ARF due to ethylene glycol toxicity in an English setter dog.

Grauer (1989) stated that metabolic acidosis and hyperkalemia were common in oliguric ARF. The acidosis was usually partially compensated by respiratory alkalosis. Bicarbonate therapy should be resumed for patients whose blood pH was 7.1 or less. Hyperkalemia was the most life threatening electrolyte disturbance that occurred in ARF. Hyperkalemia should be promptly treated with slow intravenous solution administration of 1 to 2 m Eq/kg of sodium bicarbonate. Insulin, dextrose and calcium gluconate might also be used to decrease or counteract hyperkalemia.

Michell et al. (1989) stated that hyperkalemia might require specific treatment, as indicated by plasma levels above 6.0 m mol/L and its effect could be countered by 10 percent calcium gluconate 0.5 to 1 ml/kg or corrected by treatment of acidosis (1.0 m mol of bicarbonate/kg).

Dighe et al. (1990) observed that there was significant increase in BUN and serum creatinine levels in experimentally induced acute renal failure in dogs.

Giger and Noble (1991) estimated the normal value of globulin as 2.8-4.5 gm/dl in Basenji dogs.
Ihle and Kostolich (1991) recorded that the levels of BUN (88 mg/dl) and creatinine (5.3 mg/dl) in ARF associated with contrast medium toxicity in a dog after 10 hours of intravenous pyelography.

Mary (1992) mentioned that abnormalities in sodium and water balance were the result of their continued ingestion in the presence of reduced excretory function. Serum sodium concentration in ARF could be normal, low or elevated depending on the factors contributing to patient dehydration and the stage of the disease process. If diuresis occurred during the late incipient phase or early in the maintenance phase, free water loss might be greater and hypernatremia might result. Sodium was retained during the oliguric or anuric phase due to reduction in GFR despite an increase in fractional sodium excretion.

Mary (1992) recommended short term management of severe hyperkalemia (potassium ion concentration>8 m Eq/L). Administration of calcium gluconate acted as a specific antagonist to the cardiotoxic effect of hyperkalemia.

Mayer et al. (1992) reported the normal serum chemistry values for adult animals. The normal serum globulin values of dog was reported to be 2.3-5.2 gm/dl.

Lane et al. (1994) mentioned that hyperkalemia and severe metabolic acidosis were most likely to occur in case of ARF, whereas non-regenerative anaemia, hypokalemia, and mild metabolic acidosis were suggestive of CRF.

Polzin et al. (1995) stated that progressive hypoproliferative anaemia was characteristics of dogs with moderate to advanced CRF. Anemia in patients with CRF was multifactorial. There was experimental and clinical evidence for the supporting roles of short red cell life span, nutritional, erythropoietic inhibitors, blood loss and erythropoietin deficiency.
Stanley (1995) stated that serum sodium and chloride concentrations were usually normal in dogs and cats with uremia and ECF depletion. That was the result of isotonic losses of sodium chloride and water in urine or gastrointestinal fluids. Hypernatremia might result when losses of free water exceeded sodium loss. Hyponatremia occurred less commonly and was the result of continued water consumption and impaired water excretion that might occur with ECF volume depletion due to sodium chloride loss.

Stanley (1995) reported that hyperkalemia was most commonly recognized with hypoadrenocorticism, ARF and post-renal failure that was most often seen in severely oliguric patients, particularly those with severe metabolic acidosis. Sodium bicarbonate might be administered to shift K⁺ from the extra cellular fluid space to the intracellular fluid space. The recommended dose of sodium bicarbonate was 1 to 2 m mol/kg IV over 5-15 minute. If, sodium bicarbonate failed to work, calcium gluconate might be given at the dose of 0.5 ml/kg (10% solution) to counteract toxic effects of hyperkalemia on myocardium.

Bonagura and Kirk (1995) estimated the protein concentration less than 6.5 gm/dl in regenerative anaemia in dogs due to haemorrhage and at range of 4-5.5 gm/dl in moderate to marked external haemorrhage.

Bonagura and Kirk (1995) reported the range of A:G ratio in healthy dogs as 0.50-1.20

Grauer (1995) stated that plasma concentration of several electrolyte could affect the development of ARF. Hyponatremia exacerbates gentamicin nephrotoxicity in rats and potentiates contrast media-induced ARF in dogs. hypocalcemia, hypomagnesemia and hypokalemia were additional electrolyte abnormalities that might potentiate nephrotoxicity.
Jayathangaraj et al. (1995) evaluated the levels of BUN and serum creatinine as 101.9 mg/dl and 3.1 mg/dl, respectively in a male nondiscript dog with acute renal failure.

Devaux et al. (1996) evaluated the values of BUN, serum creatinine, Total protein and albumin as 80 mg/dl, 6.5 mg/dl, 6.9 g/dl and 2.7 g/dl, respectively in a *Golden retriever* dog with uremic crisis.

Swenson (1996) reported the normal plasma globulin value in healthy dog as 2.2-3.2 gm/dl.

Kaneko et al. (1997) reported the normal plasma globulin value in healthy dog as 27-44 (34±5.1) gm/L.

Morgan (1997) mentioned the normal value for total protein content in plasma of healthy dogs to range between 5 and 7.1 gm/dl.

Shaw and Ihle (1997) observed the total protein content in plasma of healthy dogs in between 5 and 7.5 gm/dl.

Tvedten (1999) reported the normal albumin-globulin ratio in healthy dogs as 0.89-2.68.

Bistner et al. (2000) reported the normal reference values of Total Serum Protein and albumin as 5.8±8.2 gm/dl and 3-4.2 gm/dl, respectively in dogs.

Kaneko et al. (2000) recorded the normal reference values of albumin globulin ratio to range between 0.59-1.4 in dogs.

Brant et al. (2001) recorded the range of BUN and serum creatinine as 23 to 209 mg/dl and 4.3 to 18 mg/dl, respectively in ARF associated with grapes or raisins toxicosis in dogs.

Das et al. (2004) concluded that the increased in BUN and serum creatinine indicated renal damage in experimentally induced leptospirosis in dogs.
Nak et al. (2004) found increased level of serum creatinine and BUN in bitches with pyometra. They also concluded that pyometra might cause renal failure, as related to bacterial toxin.

Benjamin (2005) mentioned that creatinine was a non-protein nitrogenous substances that was formed during the metabolism of muscle creatine and phosphocreatine. That was excreted exclusively by glomerular filtration and appeared as the same concentration as in plasma. Formation of creatinine was not affected by dietary protein, age, sex or exercise.

DiBartola (2005) stated that the relationship of BUN or serum creatinine concentration to the GFR was a rectangular hyperbola. Therefore, large changes in GFR early in the course of renal disease causes small increase in BUN or serum creatinine concentration on the otherhand, small changes in the GFR in advanced renal disease causes large changes in the BUN or serum creatinine concentration.

3.4 Urine analysis:

A critical examination of urine is an important veterinary medical laboratory diagnostic procedure. Since urine is the end product of complicated and delicately balanced physiological process, many normal and pathological mechanisms influence the constituents.

Hoe and O'Shea (1965) compared the histological findings of renal patients with that of blood chemistry and urine analysis. It was found that increased BUN level could be used in 73 percent of cases where as urine analysis was diagnostic in 90 per cent of cases.

Bovee (1969) worked on the renal concentrating capacity and stated that USG indicates the density of a fluid, the ratio of the weight of urine to an equal volume of water. USG is only a rough index of renal concentrating ability, since the kidney, in the maintenance of water and electrolyte balance, responds to changes in the osmolarity of body fluid
and not to changes in specific gravity. Urine is a complex fluid containing solute of different nature in greatly varying proportions. Reportedly, 70-90 per cent of the USG of normal urine could be accounted for by urea, creatinine, chlorides, phosphates, sulphates, and bicarbonates. The undetermined 10-30 percent is composed of a variety of dialyzable organic compounds.

McEwan (1971) reported that casts which were found in the urine originated from the renal tubules and collecting ducts. The main type of cast usually seen were granular casts in which that cells had degenerated, lost their individuality and appeared as fine or coarsely granular tubular masses and hyaline casts which were transparent. Granular casts were found in acute and sub-acute nephritis and in chronic renal disease both granular and hyaline casts were seen.

Osborn et al. (1972) reported that specific gravity might range from approximately 1.001 to 1.060 in normal dogs. Randomly obtained urine samples obtained from normal dogs usually had a specific gravity which encompasses a narrow range (1.015-1.045).

English (1973) stated that measurement of urine specific gravity and osmolality were used to determine the kidney’s ability to concentrate urine. The ability to concentrate in the chronically diseased kidney, depended on an adequate secretion of antidiuretic hormone and the presence of enough unchanged nephrons.

According to Coles (1974) the colour of urine specimen should be recorded, the yellow colour of urine depended on the concentration of urochromes. If urine was concentrated, the amount of urochrome per unit volume was increased and urine appeared darker than normal. Where as if urine volume increased, urochromes were diluted and the urine appeared pale.

According to English (1974) the specific gravity of urine depends on the number as well as the nature of the particles in solution, where as
osmolality depends only on the number. Large, dense molecules (sugar, protein) raise the specific gravity much more than osmolality.

Coles (1974) opined that specific gravity of urine is a measurement of the relative amount of solids in solution and consequently, is an indication of the degree of tubular reabsorption or concentration by the kidney. Under conditions of normal renal function and normal metabolism, the specific gravity of urine varied inversely with the volume of urine excreted. As USG is related to urine volume and urine volume to the water intake and body metabolism, it is difficult to ascribe specific values for the normal animal. In general, USG for most animal species will ranged between 1.015-1.045, but values as high as 1.060-1.080 could occur. Randomly collected urine specimen might deviate from this range in animals with normal functioning kidneys.

English (1974) stated that examination of sediment after the centrifugation of urine gave information of the site, nature and extent of renal lesions, for maximum information, the clinician should perform it himself. Tubular epithelium sloughs off into urine in large numbers in cases of proteinuria. Only when the RBCs and WBCs appeared in casts it could be confirmed that they were of renal origin and oxalic acid crystals were seen in ethylene glycol toxicity. In chronic renal disease, hyaline casts were seen but not in great numbers.

Barlough et al. (1981) studied the value of macroscopic and microscopic examination of urine in 1000 dogs. Of these 5.6 percent of dogs had abnormal colour. Among these 91.5 percent had positive findings on sediment examination. Among the sediments 22.2 percent had RBSc, 24.3 percent WBCs, 16.9 percent bacteria, 2.6 percent casts, 0.5 percent urate crystals and 0.1 percent epithelial cells.

Ganesh et al. (1981) noticed deep yellow coloured urine with a specific gravity of 1.014, albumin and leucocytes, cells casts and renal cells in the sediment of a dog diagnosed as acute renal failure.
Benjamin (1985) stated that colour of urine was always associated with specific gravity and volume of urine. The normal colour was yellow to light amber and depended primarily on the concentration of urochromes, whose output was relatively constant.

Deborah et al. (1985) noted pyuria, proteinuria, granular casts and glucosuria in dogs with gentamicin induced nephrotoxicity.

Greco et al. (1985) noted pyuria, proteinuria, granular casts and glucosuria in dogs with gentamicin induced nephrotoxicity.

Scott et al. (1985) in gentamicin associated ARF in the dog noticed urine specific gravity in the isosthenuric range (1.008 to 1.012) at the time of identification of nephrotoxicosis. Urinalysis frequently revealed proteinuria, cylindruria and heamaturia. Granular casts and hyaline casts were found in the sediment.

Shahar and Holmberg (1985) noticed isosthenuria (specific gravity 1.011) and no evidence of casts or bacteria in the urine sediments diagnosed as acute renal failure.

Dogs with normal urine concentrating ability should excrete appropriately concentrated urine (>1.030) when dehydrated and/or azotemic, but dogs that excrete urine with inappropriately low specific gravity (< 1.030) when dehydrated and/or azotemic had decreased renal concentrating ability. The most common cause of decreased renal concentrating ability during dehydration and/or azotemic was renal failure (Tyler et al., 1987).

DeSchepper et al. (1989) noticed urine specific gravity of 1.008-1.028 with RBC (25 percent), WBC (38 percent), bacteria (38 percent) and casts (75 percent) in the urine of bitches with pyometra and secondary renal failure.

Grauer (1989) reported that the volume of urine might be normal or decreased in acute failure, but the quality of the urine was always poor. However, both conditions produced urine that was isosthenuric or
minimally concentrated, containing high concentrations of sodium (>140 mEq/L) and relatively low concentrations of creatinine. In addition urine might contain glucose, abnormal quantities of protein and depending on urine volume, casts and renal epithelial cells.

Robinson et al. (1989) in their work found the mean USG to be 1.014 and ranged between 1.011 to 1.017, the pH had a mean of 5.7 and ranged between 5-6 in Bullterriers with chronic renal disease.

Franklyn et al. (1990) reported that urinalysis was a useful diagnostic technique for distinguishing between pre and post-renal azotemia, but results revealed little about severity of problems. They observed tubular epithelial cells, cellular and granular casts and had marked amount of cellular debris in aminoglycoside induced nephrotoxicosis in sheep.

Garry et al. (1990) reported that urinalysis was a useful diagnostic technique for distinguishing between pre and post renal azotemia, but results revealed little about severity of problems. They observed tubular epithelial cells, cellular and granular casts and marked amount of cellular debris in aminoglycoside induced nephrotoxicosis in sheep.

An urinalysis should be performed to diagnose ARF, very often the urine specific gravity is in the isosthenuric range (1.007 to 1.015) and proteinuria, haematuria and glucosuria are evident. Glucosuria despite normoglycemia and alkaline urine despite systemic metabolic acidosis are indicative of proximal tubular damage typical of acute tubular necrosis. The urine sediment should be evaluated for evidence of bacteria, RBC, WBC, casts and crystals. Acute tubular necrosis and nephritis may both result in formation of significant numbers of renal tubular casts (Mary, 1992).

Forrester and Brandt (1994) mentioned the finding signs of inflammation (i.e., proteinuria, haematuria, pyuria and infrequently white blood cell casts) had supported the diagnosis of nephritis, which also
might be characterized by granular casts. Acute tubular necrosis (nephrosis) causes proteinuria, haematuria, normoglycemia, glucosuria and renal epithelial casts and cellular debris in the sediment.

Willard et al. (1994) was of the opinion that the normal urine was clear to slightly turbid and amber yellow. Dilute urine likely to be colourless and concentrated urine was darker yellow. Haematuria, haemoglobinuria and bilirubinuria were the most common causes of discoloured urine. Pyuria was a common cause of turbidity.

Willard et al. (1994) stated that the normal USG ranged between 1.015 to 1.040.

As per Willard et al. (1994) urinalysis should be performed in every patient with any urinary tract disease or abnormality. Analysis consists of (1) determining colour and turbidity (2) chemical analysis and (3) microscopic analysis of sediments.

Urine specific gravity may range from 1.001 or greater for adult normal dogs. In primary acute ischemic or nephrotoxic azotemia with initial oliguric or non-oliguric phase and subsequent polyuric phase, the urine specific gravity will be in the range of 1.006 to 1.029 (Carl et al., 1995).

A diagnosis of ARF is generally made when azotemia is accompanied by isothenuria (urine specific gravity 1.008 to 1.012) or by minimally concentrated urine (urine specific gravity 1.013 to 1.029) in dogs (Gruer and Lane, 1995a).

Increased urine turbidity or changes in urine sediment (increasing number of WBC, RBC, renal epithelial cells or cellular or granular casts) are indications of acute renal damage along with increase in the fractional clearance of sodium and chloride. The acute onset of glucosuria or proteinuria may also be indicative of early glomerular or tubular damage (Grauer and Lane, 1995b).
Hurley (1998) reported that in ARF analysis of urine sediment was important as it revealed active disease in the form of cells, debris and crystals. It also helped in the diagnosis of pyelonephritis.

As per Hurley (1998) the urine may appear dilute or concentrated, and urine specific gravity will help to distinguish prerenal azotemia (USG>1.030) from intrinsic renal failure.

Vaden (1998) stated that ARF had more active urine sediments when compared with animals with CRF. But a lack of active sediments did not preclude a diagnosis of ARF. Detection of a large number of urinary casts was indicative of an active disease process. Urinary casts were detected in only 31 percent of dogs with ARF, and of those dogs, 80 percent had fewer than 5 casts per low-power field.

Jody et al. (1999) diagnosed ARF in four cats wherein they found urine modestly concentrated, pre-renal azotemia was considered unlikely, because the urine specific gravity was disproportionately low, given the severity of azotemia. Further they stated proteinuria, cylindruria and glucosuria with normoglycemia were suggestive of renal tubular injury.

Christopher and Larry (2000) analysed the urine of 23 dogs with leptospirosis and azotemia. Urine specic gravity was < 1.020 in all 23 dogs, 19 of 23 (83%) dog were isosthenuric (urine specific gravity of 1.015 to 1.009). Hemoproteinuria was detected in 19 of 23 (83%) dogs. 4 of 23 (17%) dogs had microscopic evidence of hematuria (> 10 RBC/hpf). Granular casts were evident in the sediment containing > 5 WBC/hpf.

According to Haller (2002) determination of USG is very important in assessment of renal disease. It should always be measured before any treatment is started because fluids, glucocorticoids or diuretics may result in artificially diluted urine. Hyposthenuric urine (1.001-1.007) indicates active dilution; isosthenuria (1.007-1.015) indicate unchanged excretion
and an USG reading greater than 1.015 indicate active concentration of the glomerular filtration.

Grauer (2002) stated that the normal protein levels in urine were very low. Proteins with molecular weight >60,000-65,000 dalton were normally not present in glomerular filterate. The proximal tubular epithelial cells largely resorbed the smaller molecular weight proteins. Protein resorption by epithelial cells had a transport maximum and, if that exceeded proteinuria results. Pathologic proteinuria were seen in glomerular capillary wall lesions which might be caused due to glomerulonephritis and amyloidosis. Proteinuria >50mg/kg BW over 24 hours, with urinary (pr/cr) >3.0 is suggestive of glomerular disease.

DiBartola (2005) stated that the proteinuria must be correlated with urine specific gravity as a 4+ protein in 1.010 urine represented more severe proteinuria than 4+ protein in 1.045 urine. A persistant proteinuria in the absence of urine sediment abnormalities was suggestive of glomerular disease.

4. Managemental procedures
4.1 Diuresis:

Mannitol and Furosemide are being used by veterinary nephrologists over the past 10 years to initiate diuresis in oliguric/anuric patients. Furosemide frequently induces a diuresis in patients who do not respond to mannitol (Arthur, 1982).

Donald and Larry (1983) reported that furosemide @ 2 to 4 mg/kg might be given intravenously and repeated twice or thrice that dose if diuresis did not occur within one hour. Studies in human indicated that a slow infusion of dopamine @ 4µg/kg/minute might also be beneficial.

Graziani et al. (1983) studied the effect of dopamine-frusemide therapy in 16 ARF patients. They found that a combination of furosemide
(10-15 mg/kg/day) and dopamine (3μg/kg/minute) therapy for 6-24 hours produced a significant diuresis and natriuresis without any modification of blood pressure, pulse rate and central venous pressure.

Herrtage (1983) recommended the use of osmotic diuretics (mannitol and glucose) and loop diuretic (furosemide) for the management of oliguric acute renal failure. A 20 percent solution mannitol or glucose could be infused at a dose rate of 0.25 to 0.5 g/kg over 3 to 5 minutes in well-hydrated animals for induction of diuresis in oliguric acute renal failure. If diuresis developed, a maintenance infusion of 10 percent mannitol or 5 percent glucose should be given to keep a urine output of 1 to 2 ml/kg/hour. If there was no response within 30 minutes, then the infusion should be stopped to prevent over hydration. Furosemide could be used intravenously @ 2 to 4 mg/kg body weight and a response should be seen within an hour. Double or triple the dose could be given in an attempt to promote diuresis.

Shahar and Holmberg (1985) attempted diuresis in a dog suffering from anuric ARF with 20% mannitol (0.3 g/kg, given by slow intravenous infusion). When no response was noted, a furosemide bolus injection @ 1mg/kg body weight was given. That was followed by a 6 hour infusion of furosemide @ 1mg/kg/hr. and dopamine @ 3 mg/kg/minute. Despite the treatment, the dog remained anuric, vomited frequently and became depressed.

Furosemide probably increases renal blood flow through activation of the renal prostaglandin system, a diuresis often occurs without improvement in GFR. Although diuresis in general is thought to be beneficial, furosemide therapy seldom improves renal function and does not affect the duration of renal failure, the need for dialysis or the mortality rate. Low dose dopamine with minimal systemic effects has been effective in some experimental models of ARF. Uncontrolled clinical studies in human using dopamine in combination with furosemide, have been
effective in inducing diuresis. Many of these patients have failed to respond to prior therapy with mannitol or furosemide alone. If diuresis occurs, polyionic solution (e.g. Ringer’s Lactate) should be used for maintenance fluid requirements (Grauer, 1989).

Michel et al. (1989) recommended furosemide at the initial dose rate of 2 to 4 mg/kg body weight to restore urine-output in oligouric acute renal failure.

Ihle and Kostolich (1991) studied contrast medium induced ARF in a dog. He found that combination of dopamine and furosemide therapy was successful in returning the function to normal.

Gleadhill (1994) recommended furosemide 2mg/kg bodyweight by slow intravenous injection to restore urine output in oliguric acute renal failure. If urine output did not increase after 1 hour, the dosage could be doubled.

Lane and Grauer (1994) mentioned that oliguric patients in which urine output was less than 1 ml/kg/hour, after sufficient volume replacement, required further therapy with diuretics. As a loop diuretics, furosemide helped to increase tubular flow and improved renal blood flow but usually did not affect GFR. Initially intravenous administration of 2 to 3 mg/kg body weight were recommended. Increasing dosages upto 6 to 9 mg/kg might be administered at hourly intervals if urine production was not observed. The efficacy of furosemide in reversing oliguria appeared to be improved with the concurrent administration of dopamine.

Lane et al. (1994) recommended dopamine combined with furosemide in over hydrated patients instead of osmotic diuretics. It might be effective when osmotic diuretics failed. Low dose dopamine infusion (1 to 3 μg/kg/minute) caused renal vasodilatation and preserved renal and splanchnic blood flow which led to increase in glomerular filtration and sodium excretion.
Grauer and Lane (1995b) and Grauer (1998) advocated furosemide (2-6 mg/kg IV every 8 hour) as an initial treatment for oliguria in dogs and cats because that was easy to administer. However, an infusion of dopamine in combination with furosemide was more likely to be effective in inducing a diuresis compared with furosemide alone. Dopamine is a catecholamine, that in low doses, causes increase in renal blood flow. In dogs, low dose dopamine infusion frequently increased urine volume and fractional excretion of electrolyte in addition to increasing renal blood flow. When furosemide therapy was combined with dopamine infusion, the likelihood of inducing diuresis was enhanced. The recommended constant infusion rate for dopamine was 1 to 5 \( \mu \text{g/kg/minute} \) and that was best administered through a separate intravenous line. Adding 30 mg of dopamine to 500 ml of saline resulted in a dopamine concentration of 60 \( \mu \text{g/ml} \).

Kapoor et al. (1996) studied low dose dopamine in the prevention of contrast medium induced ARF. They found reduction in BUN and serum creatinine levels in dopamine treated group as compared to control group.

Varriale and Mossavi (1997) worked on congestive heart failure associated with renal insufficiency. They observed reduction in BUN and serum creatinine levels in dopamine treated group as compared to control group.

Ichai et al. (2000) observed that the low dose dopamine infusion did increase creatinine clearance and urine sodium excretion in critically ill patient with non-oliguric renal impairment.

Srivella et al. (2000) observed that infusion of solution of mannitol, furosemide and dopamine promoted diuresis in patients with acute postoperative renal failure with adequate postoperative cardiac output and had decreased the need for dialysis in the majority of patients.
Early administration of this solution in ARF caused early restoration of renal functions.

Brant et al. (2001) studied ARF associated with ingestion of grapes or raisins in 10 dogs using furosemide, dopamine and mannitol. Four dogs recovered completely with aggressive treatment of these drugs.

Shivakumar (2001) worked on diuretic effect of furosemide, a combination of furosemide and dopamine, mannitol and a combination of mannitol and dopamine in a clinical cases of ARF in dogs. He found that a combination of furosemide and dopamine was superior to others in inducing diuresis.

Labato (2001) recommended diuretic therapy in oliguric ARF. The initial treatment with furosemide @ 2 to 6 mg/kg body weight repeated every 1 to 2 hour until urine production increased. If urine output did not increase after 3 to 4 hour at maximum dosage, then a combination of furosemide (1-2 mg/kg/hour, administered intravenously for 4 to 6 treatments) and dopamine (1-3 µg/kg/minute) might be administered.

Chew et al. (2002) mentioned that dopamine alone or in combination with furosemide might promote diuresis when other treatment had failed.

Denton and Brady (2003) stated that low dose dopamine infusion caused, selective renal vasodilation, increased renal blood flow and induced natriuresis and diuresis in animals and healthy human.

Cowgill and Francy (2005) stated that dopamine is a catecholamine with diurectic properties caused by PGE₂ mediated renal vasodilation, increased renal blood flow, increased GFR, decreased tubular sodium and water reabsorption and decreased tubular oxygen consumption. The pharmacologic effects of dopamine were dose dependent and differentially mediated through dopaminergic and adrenergic receptors. In dog “renal “ doses of dopamine between 0.5 to 3
mg/kg/minute promoted those responses primarily by activating dopamine specific D₁-like and D₂-like receptors in the kidney and post-ganglionic sympathetic nerves.

Macintire et al. (2005) mentioned that the furosemide as an initial IV bolus of 2mg/kg body weight followed by a constant rate infusion of furosemide @ 0.2 mg/kg/hour combined with dopamine infusion @ 1-3μg/kg/minute was most effective in oliguric acute renal failure.

4.2 Peritoneal dialysis:

Abbott and Shea (1946), Grollman (1951), Kirk (1957), Jackson (1964), Tabatabo et al. (1970), Ray and Mohanty (1973) described separately the technique of peritoneal dialysis in dogs and recommended as a method for extra renal purification of blood in the treatment of uremia. They claimed a significant extraction of blood urea nitrogen in dialysate from the blood.

Dumon (1980) Indicated peritoneal dialysis in acute or chronic kidney diseases, hepatic coma, intoxication, electrolyte imbalance or persistent edema in dogs. He contraindicated peritoneal dialysis in rupture of diaphragm, respiratory insufficiency or tympany in cases of recent laparotomy or in peritonitis.

Ganesh et al. (1981) performed peritoneal dialysis (P.D.) in a German shepherd dog with ARF using 1.5% dextrose solution and a dwell time of 1 hour was allowed for exchange of solutes. P.D. was done for 2 days and they reported that it was very effective in removing metabolic waste products as evidenced by presence of high level of non-protein nitrogenous substance (N.P.N.) in drained peritoneal dialysis fluid.

Thornhill et al. (1984) performed continuous ambulatory peritoneal dialysis (CAPD) in a bilaterally nephrectomized dog for 54 days, using the column disc catheter. They found that the treated dog remained active
and alert with stabilized BUN (30 to 40 mg/dl) and serum creatinine (4 to 6.5 mg/dl). Problems encountered with P.D. included that propensity for developing peritonitis, anorexia and a significant plasma protein loss in a dialysate as a result of leakage across the peritoneum.

Crisp et al. (1989) conducted peritoneal dialysis in dogs and cats (27 cases) during a period of 11 years (1976-1987) and found significant decrease in BUN and serum creatinine. Complications observed were hypoalbuminemia (11 animals), dialysate retention due to catheter obstruction (8 animals) and subcutaneous leakage of dialysate (6 animal). However, they concluded that P.D. although associated with high complication rate, was successful technique for reducing azotemia in dogs with acute and chronic renal failure.

Dighe et al. (1990) carried out peritoneal dialysis once daily or twice daily in 12 mongrel dogs after experimental induction of acute renal failure. They found that the treated dogs showed marked improvement in appetite and general health. The BUN level was decreased from 165.38 mg/dl to 62.71 mg/dl in dogs in which dialysis was done once daily and from 151.60 mg/dl to 42.62 mg/dl in dogs in which dialysis was done twice daily for 4 days. Creatinine level was also reduced from 5.82 to 3.53 mg/dl in first group and from 4.95 mg/dl to 2.74 mg/dl in the second group.

Mahajan (2000) performed peritoneal dialysis in a dog with ARF using readymade peritoneal dialysis fluid. Dwell time of ½ hour was allowed for exchange of solutes. Peritoneal dialysis was done for 3 days with 8 consecutive exchange per day resulted into decrease in BUN and serum creatinine levels from 130 to 20 mg/dl and 3.1 to 3.9 mEq/L respectively.

Chew et al. (2000) characterized the dialysis on the basis of flow pattern of dialysate and the time period over which dialysis occurred, as continuous peritoneal dialysis, intermittent peritoneal dialysis and tidal peritoneal dialysis.
Dzyban (2000) opined that the peritoneal dialysis, used in human to manage both acute and chronic renal failure as well as to remove dialyzable toxins (ethylene glycol, ethanol, barbiturates), to reduce severe metabolic disturbances (hypercalcemia, hyperkalemia, hepatic encephalopathy), and to treat pancreatitis, uroabdomen, hypothermia and fluid over load secondary to heart failure.

Jankisz et al. (2004) treated 4 dogs with renal insufficiency by conventional method with additional peritoneal dialysis. Rapid clinical improvement was observed after dialysis in 3 cases with concurrent improvement of laboratory parameters (blood biochemistry, urinary and dialysis analysis). Dialysis was performed in 3-6 cycles per day, sub cutaneous leakage of dialysate was the most important complication observed. They finally concluded that peritoneal dialysis was a laborious time consuming and expensive procedure but very helpful in the treatment of renal insufficiency in dogs.

Kalinbacak et al. (2005) in their work on peritoneal dialysis in cats with complete ureteral obstruction observed that after 24 hours of ureteral obstruction, there was a significant increase in the BUN, creatinine, Na\textsuperscript{+}, K\textsuperscript{+} and P\textsuperscript{2} levels. The total protein, albumin and Ca\textsuperscript{+2} values remained unchanged. Peritoneal dialysis was carried out for 2 days at 6 hour intervals for a total of 8 session. With every session there was a significant decrease in the BUN, creatinine, Na\textsuperscript{+}, K\textsuperscript{+} and P\textsuperscript{2} values. Despite the dialysis at the end of the study of BUN, creatinine, Na\textsuperscript{+}, K\textsuperscript{+} and P\textsuperscript{2} values were higher than that at the beginning of dialysis while those of total protein, albumin and Ca\textsuperscript{+2} were lower. Finally concluded that, the peritoneal dialysis carried out at 6 hours interval for 2 days, though not enough to completely remove harmful metabolites, was still found to be effective in stabilizing the general condition of the animals as well as most of the biochemical parameters.
Kluta and Pomianowski (2005) recommended dialysis therapy mainly in ARF whereas intermittent peritoneal dialysis was most commonly used dialysis therapy in carnivores.

Macintire et al. (2005) indicated P.D. in acute renal failure nonresponsive to conservative management, acute decompensation of CRF, pre surgical stabilization of severe uremia secondary to urinary tract leakage, as well as intoxications of dialyzable drugs (Methanol, acetylsalicylic acid, amphetamine, aminophylline, and aminoglycosides) and certain toxicities (ethylene glycol). They contraindicated P.D. in abdominal wall trauma, severe ascites, extreme obesity, severe bowel distension, diaphragmatic hernia and recent abdominal surgery.