REVIEW OF LITERATURE
In 1836, Richard Bright directed attention to cardiovascular complications of uremia. His lucid case descriptions emphasized the relationship between small contracted kidneys and large hearts, in patients dying of end stage renal disease. In 1944, Raab proposed the hypothesis of a myocardial toxicity from catecholamines and uremic products accumulated in the uremic organism. Among the uremic products, urea, creatinine, guanidino-succinic acid and methyl guanidine have been incriminated. Other factors which may cause myocardial toxicity or damage have also been suspected, such as, previous viral infection, thiamine deficiency, carnitine and iron deficiency and an alteration in the myocardial phosphate and calcium contents.

Again in 1846, Richard Bright described the enlargement of heart as a well known feature of uremia. The first modern appraisal of heart disease in uremia was published in 1947 by Langendorf and Pirani. These authors recognized various clinical and electrocardiographic features of left heart strain, coronary artery disease, pericarditis, and hyperkalemia in uremic patients.

**HYPERTENSION**

Hypertension occurs in 70-100% patients. Its incidence reported by Agrawal et al (1984) as 79.92% and Narayan et al (1988) as 100%. In various studies majority
of patients with CRF had hypertension but few patients developed CRF requiring dialysis without having hypertension, which is probably the most important risk factor in the development of the atherosclerotic cardiovascular disease. Many studies in patients with end-stage renal failure had shown the arterial pressure to be exquisitely dependent upon blood volume. So called volume dependent hypertension (Dustan et al, 1964; De Plauque et al, 1969). In 1969, Coleman et al studied the effects of fluid overloading on hypertension by giving saline infusion at the end of hemodialysis. Within 15 minutes of volume loading, patients studied were noted to have elevated blood pressure which remained elevated up to 96 hours, and this elevated pressure returned to normal when the excess volume was removed by dialysis.

However, a minority of patients with CRF (chronic renal failure) had hypertension, which was not volume related but instead was secondary to elevation of plasma renin activity, and the blood pressure was uncontrollable by lowering blood volume but responded well to bilateral nephrectomy with consequent reduction in plasma renin activity (Wilkinson et al, 1970). Grollman et al (1971) had isolated the pressure substances from the blood of dogs with bilaterally surgically induced renal artery stenosis. The pressure substances, though not related to the renin-angiotensin system, though to be the possible mechanisms of hypertension in patients with CRF. These
substances were different and more potent constrictor than angiotensin II. The identity of these substances and their role in hypertension associated with human renal disease remained to be elucidated.

Hemodynamic studies in patients with ESRD (End stage renal disease) have shown an elevated cardiac index and mean arterial pressure but a normal systemic vascular resistance (Kim and Onesti et al, 1972). The elevated cardiac index and normal systemic vascular resistance were related to anemia. With correction of anemia, the cardiac index was reduced and both arterial pressure and systemic vascular resistance were elevated (Kim and Onesti et al, 1973).

Lilly et al (1976) noticed that in few patients blood pressure was not controlled either by volume reduction or bilateral nephrectomy or explained by elevations in plasma renin activity. In this group of patients the hypertension was found to be secondary to sympathetically mediated vasoconstriction. Lilly et al noticed that many patients with renal insufficiency whose hypertension was caused by sympathetically mediated vasoconstriction exhibited an exaggerated response to cold pressure test and elevated levels of dopamine beta hydroxylase as indices of increased adrenergic function but became hypotensive during dialysis.

In 1976, Lee and Mukerjee et al had proposed one another mechanism of hypertension in patients with CRF.
The authors noted the absence of vasodepressor substances of renal origin, such as the prostaglandins, which may play a role in the genesis of renoprival hypertension.

The appearance of hypertension correlates with progressive loss of renal function and to some extent with the type of underlying renal disease. Patients with CRF who remains normotensive most often have underlying tubular interstitial disease, obstructive uropathy, or polycystic kidney disease. In contrast, nephrosclerosis and glomerulonephritis are usually associated with severe or accelerated hypertension (Bennett, 1979). Braunwald (1980) substantiated the possible mechanism described by Wilkinson et al and found that uremic patients with hypertension were having elevated plasma renin activity. In these patients the hypertension was not controlled by lowering blood volume but well controlled by bilateral nephrectomy.

John et al (1981) in a hemodynamic study in patients with renal insufficiency, found that malignant hypertension in these patients may have different hemodynamic feature going from high cardiac output state to low cardiac output state. The author also noted elevated right and left ventricular pressure in all cases indicating congested state (fluid overload) or heart failure. Machado et al (1983) described the baroreceptor setting and function appeared to be abnormal in patients with hypertension and renal failure. When the initiating hypertension was
relieved, the baroreceptors often remained at an abnormal level and contributed to the hypertension.

**CONGESTIVE HEART FAILURE**

Many factors may account for heart failure in patients with CRF including hyper-reninemia, hypertension, anemia, A.V. shunt and complications of coronary artery disease. Ionic alterations such as hyperkalemia, hypocalcemia, hypomagnesemia and metabolic acidosis may all have a negative ionotropic effect. Most of these factors impair cardiac performance by a variety of mechanisms. The incidence of CHF reported by various authors is 48-66%. Agrawal et al (1984) reported 52% and Narayan et al (1988) as 56%. Gibson (1966) and Matao (1971) described CHF as the single most cause of death.

Volume of pressure overload anemia, A.V. fistula → Myocardial damage

Acetate, hypoxia, paratharmone, uremic toxins, metabolic electrolyte abnormalities, Ischaemia.

Increased ventricular load → Congestive heart failure

Decreased myocardial contractibility

Increased capillary permeability

Factors contributing to the development of congestive heart failure in patients with chronic renal failure.
Luisada et al (1940) described congestive heart failure as a common event in the terminal stage of renal insufficiency. Luisada et al noticed that although its manifestations were overshadowed by other uremic features such as central nervous system disturbances, vomiting and hemorrhage etc., but the congestive heart failure was ultimate cause of fatal outcome in the majority of these uremic cases. Burlington et al (1944) studied 28 uremic patients and found that hypertension and signs of heart failure were present in majority of these cases.

Blumgart (1953) accepted the concept that congestive heart failure developed whenever, the cardiac output became inadequate for the metabolic needs of the body. Studies of the cardiovascular and renal hemodynamics and metabolic inter-relationship had shown that the congestive heart failure was a consequence of changes in pressure flow relationships in the circulation and disturbances in the fluid and electrolytes metabolism, which led to retention of sodium and water and the development of oedema (Hanenson et al, 1953).

According to Harry and Derow (1954) congestive heart failure in chronic renal disease was usually the result of hypertension and coronary arteriosclerosis. Anemia and electrolyte derangement were thought to be the added strains upon the heart which were responsible for further development and aggravation of congestive heart failure.

Crossbie et al (1972) described an increase in pulmonary
capillary permeability leading to pulmonary oedema, even in the absence of elevation of pulmonary capillary wedge pressure in uremic cases. Ischemic heart disease as a consequence of accelerated atherosclerosis, was also responsible for impairment of cardiac performance. Later on this hypothesis was supported by Rackow et al (1978). Gotloib et al (1975) thought the possibility of depletion of essential substances and water soluble vitamins particularly thiamine by hemodialysis, leading to beri-beri heart disease and ultimately heart failure. Capelli et al (1977) has described an increased left ventricular work index and left ventricular end diastolic pressure and size in many patients with end stage renal failure. Kleiger et al (1981) and Lai et al (1982) were of the same opinion as of Capelli et al, (1977).

Despite the well recognised relationship between calcium and myocardial contractility, clinical reports of hypocalcemic heart failure were rare, among which hyperparathyroidism, primary or secondary accounted the majority (Giles et al, 1982 and Ramailho et al, 1985). In 1988, Lang et al reported a direct relationship between left ventricular contractility and serum calcium level in uremic patients with heart failure maintained on regular hemodialysis. Recently in 1990 Cheuk-kit Wong et al described the occurrence of hypocalcemic heart failure in young woman with end-stage renal disease and found that heart failure responded to calcium replacement.
CARDIOMYOPATHY

In 1944, Raab suggested that cardiotoxic substances may exist in the blood and heart muscle in uremia and proposed the hypothesis of a myocardial toxicity from catecholamines and uremic products, accumulated in the uremic organism. Among uremic products, urea, creatinine, guadinosuccinic acid and methyl guanidin have been increminated. While Raab has postulated one of specific factors to cause myocardial disease, other investigators such as Langendorff has suggested that only non-specific factors are involved in the pathogenesis of uremic cardiomyopathy.

In 1967, Bailey et al described 5 patients with terminal renal insufficiency, who developed severe heart failure that was reversible after intensive hemodialysis. Hypertension and anemia seemed unlikely major cause of cardiac failure in these patients, so the author suggested that uremic toxins might be implicated. Goodwin and Grosgogeat et al (1970) and Di Matteo et al (1972) presented evidence of cardiomyopathy of unknown cause occurring in uremic patients who were undergoing intermittent hemodialysis. Scheur et al (1975) and Rodger et al (1976) supported the evidence of Goodwin et al (1970).
Factors contributing to myocardial damage in patients with chronic renal failure.

Gueron et al (1975) suggested that because of frequent concomitant existence of arterial hypertension, fluid volume overload, local or diffuse atheroma, anemia, hormonal disturbances, vitamin deficiency and A.V. fistula, it was impossible to determine whether only one of these other factors were responsible for the uremic cardiomyopathy. The question remained whether a specifically uremic cardiomyopathy due to retention of normally excreted or metabolised substances, might also be implicated (Prosser et al, 1975).

Scheuer et al (1975) provided indirect evidence that uremic serum had a net depressant effect on myocardial performance in chronically uremic rats in vivo.
The authors also demonstrated depressed cardiac function when normal rat hearts were perfused with mixture of urea, creatinine, methyl guanidine and guanidino succinic acid (Scheuer and Slezoski et al, 1973).

In 1980, Druke et al have proposed the possibility of uremic cardiomyopathy to be related to hyperparathyroidism. The authors also supported the previous evidence of Massry et al (1979) of parathyroid hormone to be uremic toxin. Collins et al (1985) postulated secondary hyperparathyroidism among other factors to be responsible for the uremic cardiomyopathy. This hypothesis was based on the fact that the left ventricular ejection fraction of the patient with CRF and hyperparathyroidism was increased after parathyroidectomy.

PERICARDITIS

Pericarditis has been recognised as a serious complication of uremia since its first description by Richard Bright (1836). In the era before the availability of dialysis, pericarditis was observed in 35 to 51% of uremic patients with CRF (Wacker and Merril, 1954). The pericardial effusion was classic complication of chronic uremia. The incidence of pericarditis before initiation of dialysis in patients with CRF has been described as Richter AB and O'Hare, 1936 (44%), Wacker and Merril (1954) (51%), Marini and Hull (1975) (35%), Comty et al (1975) (26%), Selverbeg et al (1977) (20%), Agarwal et al (1984) as 43.29% and Narayan et al (1988) (24%).
Maintenance hemodialysis has dropped the incidence to 10-30% and the incidence of pericarditis in patients maintained on hemodialysis has been variably reported as Bailey et al, 1968 (14%), Comty et al, 1971 (16%), Marini and Hull, 1975 (10%), Connor et al, 1976 (8%), Kumar et al, 1980 (14-20%) and Rutsky and Rosland 1989 (<10%).

Marini and Hull (1975) described pericarditis as one of the more dramatic complications of CRF because of its suddenness with which it used to appear and its hemorrhagic complications. Comty et al (1976) reported two forms of pericarditis in patients with CRF, one appearing early and other appearing later in the course. Early pericarditis was defined as pericarditis preceding or occurring during the month following the initiation of dialysis and the late pericarditis occurring after the first month of intermittent dialysis treatment.

Alfrey et al (1968) reported that, as claimed by previous authors, heparin used during hemodialysis session not appeared to play a direct role in the pathogenesis of uremic pericarditis. However, heparin might play a role in the transformation of the serofibrinous effusion into hemopericardium, an event which also developed in the absence of heparin.

The role of excessive retention of nitrogen compound from CRF or from insufficient dialysis was first
postulated as a contributory factor in the genesis of uremic pericarditis. Harris et al (1975) had reported a possible bacteriologic or viral etiology of uremic pericarditis. However, evidence for direct bacterial invasion in the majority of cases has been absent. Kleiman et al (1978) suggested hyperurecemia as an etiological factor. It was hypothesized that precipitation of hypersaturated uric acid may occur in the pericardium. Keane et al (1979) postulated the possibility of compromised immune system and increased frequency of infections in uremic patients and noted the failure of pericarditis to resolve in some patients following control of uremia, and the development of pericarditis in the stable patients treated with dialysis. In 1979, Eknoyan et al suggested the volume overload as an important factor in the pathogenesis of uremic pericarditis.

Subramanian et al (1980) believed serositis to be the initiating factor in uremic pericarditis, exacerbated by the hemorrhagic problems and continued injury to the inflamed pericardial surface by continued contraction and relaxation of the myocardium. The authors also noted that the pathological finding in patients with uremic pericarditis were variable with the stage and nature of the disease. The pericarditis seen was fibrinous, effusive subacute constrictive and a chronic constrictive or a combination of all.
Druke et al (1980) and Mako et al (1983) have suggested abnormalities of calcium and phosphorous metabolism as a result of hyperparathyroidism secondary to chronic uremia, to be contributory factors in the pathogenesis of uremic pericarditis. Maisch et al and Twardowski et al (1983) had suggested the relationship of pericarditis to "Immune reactions" or circulating immune complexes. In 1984, Bergstrom et al have suggested the role of "Middle molecules" in the genesis of uremic pericarditis. Rutsky and Rostand (1989) also supported the concepts of inadequate dialysis as a cause of dialysis associated pericarditis and found that increased dialysis frequency may lead to the resolution of 50-70% of cases. Recently Stephen et al (1990) also supported the concepts of Harris et al (1975) of bacteriologic or viral etiology of uremic pericarditis, and found that bacterial and viral cultures of pericardial fluid rarely had been positive.

**CARDIAC TAMponade**

This vexing and life threatening complication of uremic pericarditis was first reported in the literature in 1956, when Goodner and Brown recorded deaths of two young males with chronic renal insufficiency. The authors performed necropsy of both cases and drained 800 and 850 ml of blood stained fluid from pericardial cavity. Keleman
and Kloff (1960) reported the occurrence of cardiac tamponade in a patient during hemodialysis. Since then several additional cases have been published by various authors (Wutt and Holmes, 1961; Marikas Samartzis and Marketos, 1962; Rappaport, 1962; and Beaudry et al, 1966).

The incidence of cardiac tamponade has been reported by various authors in about 20% of the patients with dialysis pericarditis (Comty et al, 1971; Mitchell et al, 1974; Marini and Hull, 1975 and Winney et al, 1976).

There has been an increasing awareness of the possible inter-relationship between initiation of hemodialysis and the development of tamponade, because of the fact of total body heparinization routinely used during dialysis. The authors thought the possibility of total body heparinization to encourage additional intra-pericardial bleeding and thus precipitating tamponade. In 1968, Alffrey et al supported the hypothesis of Beaudry et al (1966) but Skov et al (1969) found that type of heparinization did not seem to be an important factor, as the incidence of tamponade was only 12.5% when the authors used total body heparinization for hemodialysis.

Goldstein et al (1977) noted cardiac tamponade rare in patients who were asymptomatic with small effusion diagnosis by echocardiography alone. In 1979, Hencock found that in general the diagnosis of tamponade was clinical.
It was estimated that in a series of regularly
dialyzed patients with uremic pericarditis, cardiac
tamponade arose in 31% and in unselected regular dialysis
patients it occurred in 5%. Its incidence in patients who
had never been dialysed was not known but was probably
quite low.

et al (1989) had evaluated a 2-D and Doppler echocardiog-
graphic study in patients of end-stage renal failure
having pericardial effusion and noted that the presence
of a large echocardiographic pericardial effusion only
suggested the possibility of tamponade or pretamponade
i.e. hemodynamic compromise.

CONSTRUCTIVE PERICARDITIS

Traeger et al (1964) reported first case of
constrictive pericarditis as a rare complication of uremic
acute pericarditis. In 1969, Rayman reported subacute
constrictive pericarditis as variant of chronic constri-
cutive pericarditis. So far a dozen observations have
been published by various authors like Lindsay et al

Until 1976, constrictive or adhesive pericarditis
was an uncommon sequelae of uremic pericarditis. With the
longer survival of patients receiving dialysis, the
incidence of constrictive pericarditis has become more
frequent (Wolfe et al, 1972 and Sloan, 1974).
The pathogenesis of subacute constrictive pericarditis is not fully understood but supposed to be the result of persistent inflammatory reaction with fibrinous exudate and recurrent hemorrhage with subsequent organization by fibrous tissue. Once the process is fully established, mostly it becomes irreversible and removal of pericardium becomes necessary (Pillay et al, 1976).

UREMENTIC HEMOPERICARDIUM

Jones Evans (1922) first reported hemopericardium in a patient with chronic renal failure. Barach et al (1922) described tapping of hemorrhagic pericardial fluid from a uremic patient, but he made no mention of pericardial tamponade. Uremic hemopericardium has been attributed to bleeding into pericardial sac as a result of shearing of small vascular channels due to continued myocardial contraction and relaxation (Goodner et al, 1955). or as a result of coagulation defect associated with the uremic defect (Beaudry et al, 1966). Another possibility thought by various authors was simple exudation of fluid across the involved serous membrane. The characteristics of the pericardial fluid were compatible with any of these possibilities. Pericardial fluid was uniformly hemorrhagic and having a high protein concentration. Allen et al (1968) studied the patients with uremic hemopericardium and observed the most common symptoms of congestive heart failure in these cases.
CORONARY ARTERY ATHEROSCLEROSIS
AND CORONARY ARTERY DISEASE.

Burton et al (1971) and Thomas and Lee (1976) accepted the concept that patients with End-stage renal disease had an accelerated rate of atherosclerosis and an accelerated mortality from coronary artery disease. Lazarus et al (1975) and Jacob et al (1977) reported an increased incidence of coronary artery disease in patients with chronic renal failure, as compared to a control population group. Boundoulos (1981) and Fisher (1982) described coronary atherosclerosis as the leading cause of cardiac death in patients with chronic renal failure. The incidence of coronary artery disease as reported by various authors is 8 to 38 percent. Agarwal et al (1984) and Narayan et al (1988) reported it as 30% and 38% respectively.

The major contributory factors for coronary artery atherosclerosis as related to chronic renal failure are hypertension, abnormal lipid and carbohydrate metabolism, decreased high density lipoproteins (HDL), increased thrombus formation, vascular calcifications, stress and often steroid therapy. A sedentary life and perhaps dialysis therapy, itself may also play a role. The underlying disease responsible for renal failure (diabetes mellitus) is an important contributory or associated factor leading to atherosclerosis in selected patients with chronic renal failure (Linder et al, 1974 and Pick et al, 1978).
Factors contributing to the development of coronary atherosclerosis with chronic renal failure.

Increased insulin resistance decreased lipoprotein lipase activity, ↑ hepatic synthesis of triglycerides. Glucose, acetate in dialysis fluid.

↓ fibrinolytic activity platelet dysfunction.
↑ factor VIII Von Wille Brand

Underlying disease (diabetes, nephrotic syndrome)

Abnormal carbohydrate and lipid metabolism

Hyperlipidemia

HDL

Hyperparathyroidism calcium, phosphorus

Stress, increased catecholamines

Calcification

Coronary atherosclerosis

Hypertension

Therapy with steroid

Rostand et al (1979), Friedman et al (1981) and Rostand, Kirk and Rutsky (1982) noted that risk of developing coronary artery disease for whites was about twice that for blacks. Patients with chronic pyelonephritis and/or interstitial renal disease for yet unknown reasons developed coronary artery disease three times more frequently than other patients, if all other variables held constant.

Patients with CRF undergoing maintenance hemodialysis have long been documented to have abnormal carbohydrate metabolism and an increase in total lipids,
particularly serum triglycerides (Smith and De Fronze, 1984) abnormal glucose tolerance has been documented in patients with chronic renal failure who were not diabetic and patients on hemodialysis may develop metabolic abnormalities similar to diabetes mellitus.

Several theories have been proposed to explain the elevation of plasma triglycerides in chronic renal failure (Pick et al, 1978; Bagdad, 1979, Hohn et al, 1983; and Asayania et al, 1984). Increased hepatic synthesis of triglycerides, perhaps secondary to insulin resistance, has been reported. Diminished lipoprotein lipase activity has been reported in patients on hemodialysis, which may be responsible for hypertriglyceridemia. The role of glucose in dialysis fluid have been reported to promote hyperlipidemia and that of acetate in dialysis fluid to promote synthesis of cholesterol and triglyceride.

Termon et al (1971), Basile et al (1985) and Akmal et al (1985) reported the role of other metabolic abnormalities associated with chronic renal failure in promoting the atherosclerotic process, such as carnitine deficiency, secondary hyperparathyroidism and altered intravascular clotting. Metastatic calcification including coronary artery calcification has been well documented in the dialysis population.
CARDIAC CALCIFICATION AND VALVULAR HEART DISEASE

In 1968 Woodhouse and Huston reported the metastatic calcification involving myocardium occurring frequently in patients with chronic renal failure on maintenance hemodialysis. Later in 1971, Terman et al supported the hypothesis of Woodhouse and Huston. In 1978, Kraikit Panitch et al described hypophosphatemia, increased ionized calcium, calcium phosphorus product, parathormone levels and acute changes in plasma and tissue pH, during or following dialysis, as contributory factors to cardiac calcification. The reported important cardiac structures affected by calcification are mitral annulus, aortic valve, coronary arteries, atrioventricular node with other conducting tissue, interventricular septum, myocardium and pericardium. The involvement of these structures may be responsible for calcified valvular heart disease, AV block with conduction defect and constrictive pericarditis (Terman et al, 1920).

MITRAL ANNULAR CALCIFICATION (MAC)

The incidence of mitral annular calcification has been reported by various authors 15-36% in patients with chronic renal failure (Schott, 1978 and Maher et al, 1987).

In 1977, Schott et al recognised the valvular heart disease as complication of uremia and also noted that MAC occurred more frequently in patients with CRF than in age matched controls and severe mitral annular calcification may cause mitral stenosis (Hammer et al, 1978).
and/or mitral regurgitation (Fuikerson et al, 1978). In 1979 Fuikerson et al described calcification of mitral annulus as a frequent but inconsequential finding in autopsies in patients with chronic renal failure. The authors noted the mitral annular calcification preferentially involving the posterior mitral leaflets but also noted the involvement of both leaflets leading to mitral stenosis in few cases having massive mitral annular calcification. In 1981 Depace et al described a patient with Chronic renal failure and severe uncontrolled secondary hyperparathyroidism having mitral annular calcification.

In 1983, Nestico et al had advocated both hypertension and abnormal calcium phosphorus metabolism as the most likely causes of mitral annular calcification. Maryvyn B et al (1984) has described the incidence of MAC as 9.5% in their study.

Osterberger et al (1981) and Nair et al (1984) noted various complications of mitral annular calcification such as conduction system disease (Supraventricular arrhythmias, SA node dysfunction, AV nodal and intraventricular block), bacterial endocarditis, embolic events, mitral regurgitation and possibly mitral stenosis. Osterberger et al described a patient of chronic renal failure with mitral annula calcification having functional mitral stenosis. In a prospective echocardiographic study in 87 patients with chronic renal failure on
maintenance hemodialysis, Mahere et al (1987) found the incidence of mitral annular calcification in 31 (36%) patients and aortic valve calcification in 24 (28%) patients. Mitral annular calcification was associated with functional mitral stenosis in 1 patient. Maher et al also noted that premature mitral annular calcification was associated with increased calcium, phosphate product and long term hemodialysis.

AORTIC VALVE CALCIFICATION

In 1970 Robert recognised the occurrence of aortic valve calcification in patient of uremia on maintenance hemodialysis. In 1972, Pomerance described aortic stenosis as a complication of severe premature calcification of a tricuspid aortic valve, which was later supported by Maher et al (1985). Calcification of mitral annulus was often associated with aortic valve calcification (AVC), but AVC has been seen in patients with renal failure with or without coexisting mitral annular calcification (Foramen et al, 1984). The frequency and etiology of calcific aortic valve disease in patients with End-stage renal disease has been little studied. In 1985 Castro et al reported a high incidence of AVC and aortic stenosis in dialysis patients and also reported that incidence of valvular heart disease increased with duration of hemodialysis therapy. Robert (1981) and Bradley et al (1985).
reported that hypercalcemia and hyperphosphatemia may lead to premature aortic valve calcification. In 1987 Maher et al in a retrospective clinical study found an increased incidence of aortic stenosis in patients with end stage renal disease, apparently caused by an increased risk of premature aortic valve calcification, which produced calcific stenosis in a tricuspid aortic valve. Maher et al (1987) in an echocardiographic study in patients with chronic renal failure on maintenance hemodialysis found the incidence of aortic valve calcification in 28% cases.

CARDIAC ARRHYTHMIAS

The development of hemodialysis for the treatment of chronic renal failure has given rise to a host of new problems, like ventricular and supraventricular arrhythmias and various atrioventricular blocks (Handerson et al, 1971). Handerson et al described an anephric patients undergoing hemodialysis, who found to have secondary hyperparathyroidism and metastatic calcification involving the myocardium and its conducting system. Doheerry (1973) emphasized the digitalis toxicity as the risk factor for cardiac arrhythmia in patients with end-stage renal disease. In 1976, Vap et al noted the association of hyperkalemia with cardiac arrhythmia in patients of uremia.

In 1980, Morrison et al, indicated that the frequency of arrhythmias in patients on dialysis was as high as 39% and the severity and frequency of arrhythmias
diminished in the post dialytic period. Ramirez et al (1984) emphasized the high incidence of cardiac arrhythmias in 40% of patients with chronic renal failure on maintenance hemodialysis and found high blood levels of PTH and C peptide as attributing factors to arrhythmias. Kyriakidis et al (1984) evaluated 25 patients with chronic renal failure by Holter ECG monitoring for a continuous 48 hours period and found that clinically significant arrhythmias (7100 ventricular extrasystoles/24 hours) were seen in only one patient while benign atrial arrhythmias seen in 22 patients (88%) but no complex ventricular arrhythmias. Author also noted that hemodialysis had no influence on type or frequency of arrhythmias.

ECHOCARDIOGRAPHY IN CHRONIC RENAL FAILURE

Although the important role of echocardiography in diagnosis of various primary forms of heart disease is now well established. Its value in the cardiac evaluation of patients with chronic renal failure is less widely appreciated. Various types of cardiac involvement are potentially major complications in uremic patients. Echocardiography permits rapid and non-invasive detection of such complications, which otherwise would remain unrecognised or even unsuspected. One of the important serious cardiovascular complications of chronic renal failure is pericarditis which often is not detected clinically in the absence of pericardial rub. Pericarditis
has been recognised as a clinical feature of uremia for over 100 years and frequently was harbinger of death in chronic renal failure patients before dialysis (Beaudry et al, 1966).

Echocardiography has gained widespread use for the non-invasive detection of pericardial effusion and it allows more patients to survive acute episodes of pericarditis and pericardial effusion and continue to be maintained on chronic hemodialysis (Feighenbaum, 1972).

Horowitz et al (1974) reported that careful echocardiographic evaluation may reliably detect effusion as small as 20 ml.

Akihiro Niwa et al (1985) in an echocardiographic study reported the incidence of pericardial effusion 14.2%, inter-ventricular septal hypertrophy 51.3% and left ventricular posterior wall hypertrophy 47.1%. Harnett et al (1988) in an echocardiographic study reported the incidence of left ventricular hypertrophy 55% and described the age, hypertension and hyperparathyroidism as the most important risk factor for left ventricular hypertrophy.