Review of Literature
REVIEW OF LITERATURE

It was in 1628 when Sir William Harvey demonstrated the blood circulation and showed that the heart is central organ to pump the blood to different organs.

In 1973 Clegyman reverent Stephen Hales described the result of his experience on the blood pressure of a man and he measured intra arterial pressure for the first time. He expressed pressure in terms of weight of blood itself, a time when there was no way to measure blood pressure directly. In man blood pressure was estimated to be about 75 feet of blood which corresponds to about 176 mmHg.

1847 Harrison devised the first sphygmomanometer.

J Fairve in 1856 measured blood pressure accurately.

The first book devoted to blood pressure was written by Walenberg in Berlin in 1880.

Korotkoff in 1905 introduced auscultatory technique for blood pressure measurement and suggest that sound, heard over the arteries, distal to cuff should be used to indicate blood pressure.

Frank (1911) first recognized the so called primary or essential hypertension.
Theodore C Janeway (1913) stated abnormal pressure above 160 mmHg after study of 7872 cases.

Globlatt et al (1934) were the first to produce experimental hypertension by partially constricting the renal artery in a dog.

Boedely et al (1951) concluded that the point of complete cessation of sound is best index of diastolic blood pressure.

World health Organization (1962) recommended that both muffing and cessation of sound should be recorded for diastolic blood pressure.

The definition of hypertension is based primarily on the blood pressure levels that have been established to define those who have an increase risk of developing a morbid cardiovascular event and/or will clearly benefit from medical therapy. These definition should consider not only the levels of diastolic pressure but also systolic pressure, age, sex and race.

The current classification based on systolic and diastolic blood pressure levels – High normal diastolic blood pressure (85-89 mmHg) is now considered to be category separate from normal. Persons with blood pressure in this range have a higher risk of developing hypertension and cardiovascular complications and therefore should be
considered for nonpharmacologic approaches to prevent hypertension and its complications.

Hypertension is very common in persons over 65 years of age. Approximately two thirds of the aging population have a systolic pressure of 140 mmHg or higher or a diastolic pressure of 90 mmHg or more or both. In most older patients the systolic pressure alone is elevated, a condition known as isolated systolic hypertension. This type of hypertension increases the risk of cardiovascular complications such as congestive heart failure, stroke, ischemic heart disease, and left ventricular hypertrophy (Working group on Hypertension in Elderly 1986).

*CLASSIFICATION OF HYPERTENSION*

Hypertension is defined as SBP of 140 mmHg or greater DBP of 90 mmHg or greater or taking antihypertensive medication. The objective of identifying and treating high blood pressure is to reduce the risk of cardiovascular disease and associated morbidity and mortality.

"The Seventh Report of the Joint National Committee on Prevention Detection Evaluation, and Treatment of High Blood Pressure" provides a new guideline for hypertension prevention and management. The following are the key messages: (1) In persons older
than 50 years systolic blood pressure (BP) of more than 140 mm Hg is a much more important cardiovascular disease (CVD) risk factor than diastolic BP, (2) The risk of CVD beginning at 115/75 mm Hg doubles with each increment of 20/10 mm Hg, individuals who are normotensive at 55 years of age have a 90% lifetime risk for developing hypertension, (3) Individuals with a systolic BP of 120 to 139 mm Hg or a diastolic BP of 80 to 89 mm Hg should be considered as prehypertensive and require health-promoting lifestyle modifications to prevent CVD, (4) Thiazide-type diuretics should be used in drug treatment for most patients with uncomplicated hypertension either alone or combined with drugs from other classes (5) Most patients with hypertension will require 2 or more antihypertensive medications to achieve goal BP (<140/90 mm Hg or <130/80 mm Hg for patients with diabetes or chronic kidney disease), (6) If BP is more than 20/10 mm Hg above goal BP consideration should be given to initiating therapy with 2 agents, 1 of which usually should be a thiazide-type diuretic, and (7) The most effective therapy prescribed by the most careful clinician will control hypertension only if patients are motivated

JNC VII provides a classification of blood pressure for adults (age 18 or older). These criteria are for individuals who are not taking antihypertensive medication and who have no acute illness This
classification is based on the average of two or more blood pressure readings taken in accordance with the following recommendations at each of two or more visits after an initial screening visit. When SBP and DBP fall into different categories the higher category should be selected to classify the individual's blood pressure. The classification is slightly modified from JNC VI report, a new category designated prehypertension has been added, and stage 2 and 3 hypertension have been combined in JNC VII.

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic (mmHg)</th>
<th>Diastolic (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; 120</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120 - 139</td>
<td>80 – 89</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage I</td>
<td>140 – 159</td>
<td>90 – 99</td>
</tr>
<tr>
<td>Stage II</td>
<td>≥ 160</td>
<td>≥ 100</td>
</tr>
</tbody>
</table>

Systolic hypertension is determined of arterial pressure influenced on cardiovascular morbidity.

- Isolated Systolic Hypertension is defined as systolic blood pressure of 140 mmHg or more and a diastolic blood pressure of < 90 mmHg. Patients with isolated systolic hypertension have a 25
folds increase in their cardiovascular mortality rates when compared with individuals with similar diastolic pressure but normal systolic pressure (Williams GH 1991)

- Isolated Diastolic Hypertension or Decapitate Hypertension is extremely rare and is seen only with mild elevation of diastolic pressure (e.g., 120/100 mmHg). It is usually found in children and young adults.

- Malignant Hypertension: Sudden rise of very high pressure above 200 mmHg with Papilloedema usually accompanied by retinal haemorrhages and exudates, pathologically characterized by necrotizing arteries and fibrinoid degeneration. It is papilloedema, not the absolute pressure level that defines this condition.

- Accelerated Hypertension: Signifies a recent increase over previous hypertensive levels associated with evidence of vascular damage on fundoscopic examination but without papilloedema.

- Labile Hypertension: These patients are said to have labile hypertension who sometimes, but not always have arterial pressure within hypertensive range.
CLASSIFICATION ACCORDING TO EXTENT OF ORGAN DAMAGE

According to National High Blood Pressure education programme working group WHO 1978

STAGE I

- No Objective signs or organic change in the cardiovascular system

STAGE II

- Left ventricular hypertrophy
- Retinal involvement
- Proteinuria

STAGE III

- Evidence of signs and symptoms of organ damage due to hypertension
- Heart left ventricular hypertrophy, left ventricular failure
- Brain-cardiovascular haemorrhage, hypertensive encephalopathy
- Eye-retinal haemorrhage and exudates with or without papillodema
CLASSIFICATION ACCORDING TO ETIOLOGY

A. Primary or essential or Idiopathic hypertension

B. Secondary hypertension

A. ESSENTIAL OR PRIMARY HYPERTENSION

The cause of elevated arterial pressure remains unknown in most of the cases. In general population about 90-95% hypertensive constitute this group. Patients with arterial hypertension with no definable cause are said to have primary, essential or idiopathic hypertension. Development as hypertension depends on the interaction between genetic predisposition and environmental factors. How this interaction occurs is still incompletely understood. It is known that hypertension is accompanied by functional alterations of sympathetic nervous system, the kidney, the rennin – angiotensin system and other humoral mechanisms.

SYMPATHETIC NERVOUS SYSTEM: Sympathetic nervous system may play a major role in initiating essential hypertension and may contribute to hypertension related to hyperdynamic circulatory states.
Several authors have reported increased concentrations of norepinephrine in the plasma of patients with essential hypertension (Esler M et al, 1989) Recent confirmation comes from studies on sympathetic activity recorded directly from sympathetic nerves of superficial muscle in patients with essential hypertension (Anderson EA et al, 1989, Mancia G et al, 1993)

**RENAL MECHANISM**: Renal mechanism have often been implicated in the pathogenesis of hypertension, either through an altered pressure natriuresis leading to sodium retention or through an altered release as pressure factors (such as rennin) (Cowley AW, Roman R, et al, 1986)

**RENNIN – ANGIOTENSIN SYSTEM**: The rennin angiotensin system has a major role in the physiological control of blood pressure and sodium balance. The range of plasma rennin activities observed in hypertensive subjects is broader than in normotensive individuals

*Low renin essential hypertension* – Approximately 20% of patients who by all other criteria have essential hypertension have suppressed rennin activity. Though these patients are not hypokalemic they have been reported to have expanded extracellular fluid volumes, and it has been suggested but not proved that they have sodium retention and
rennin suppression due to excessive production of an unidentified mineralocorticoid (Harrison’s principle’s or Internal Medicine 16th edition)

* Non modulating essential hypertension – Another subset of hypertensive patients have an adrenal defect opposite to that observed in low-rennin patients – a reduced adrenal response to sodium restriction. In these individuals, sodium intake does not modulate either adrenal or renal vascular response to angiotensin II. Hypertensive in this subset have been termed normodulators because of the absence of the sodium mediated modulation of target tissue response to angiotensin II. These individuals make up 25-30% of hypertensive population, have plasma rennin activity levels that are normal to high, and have hypertension that is salt sensitive because of a defect in the kidney’s ability to excrete sodium appropriately.

* High – rennin essential hypertension – Approximately 15% of patients with essential hypertension have plasma rennin activity levels above the normal range elevated rennin levels and blood pressure in these patients may be secondary to an increase in adrenergic system activity (Harrison’s Principle’s of Internal Medicine 16th edition)
STRUCTURAL CARDIOVASCULAR ADAPTATION

The increased load on the vascular system caused by high blood pressure and activation of growth factors leads to structural adaptations with narrowing of the arteriolar lumen and an increase in the media wall ratio. This amplifies resistance to blood flow and increase vascular responsiveness to vasoconstrictor stimuli (Folkow B et al, 1992).

Cardiac structural adaptations consist of thickening of the left ventricular wall in response to an increase after load (concentric hypertrophy) and an increase in left ventricular diameter (Koren MJ et al, 1991). Both vascular and cardiac structural adaptations act as amplifiers of the hemodynamic pattern of hypertension and as initiators of several of the complications of hypertension (Korner PL et al, 1994).

ENDOTHELIAL DYSFUNCTION

New studies have shown endothelium involvement in the conversion of angiotensin I to angiotensin II, in kinin inactivation and in the production of endothelium derived relaxing factor or nitric oxide. Endothelium plays a role in the local hormonal and neurogenic control of vascular tone and the haemostatic process. Endothelium also release vasoconstrictive agents, including endothelin that may be implicated in some of vascular complications of hypertension (Luchter TF et al, 1990).
B. SECONDARY HYPERTENSION

This group constitutes 5-10% patients of hypertension

1 Renal
   A Renal parenchymal disease
      • Acute glomerulonephritis
      • Chronic nephritis
      • Polycystic disease
      • Diabetic nephropathy
      • Hydronephrosis
   B Renovascular
      • Renal artery stenosis
      • Intrarenal vasculitis
   C Renin producing tumors
   D Primary sodium retention (Liddle’s syndrome, Gordon’s syndrome)

2 Endocrine
   A Acromegaly
   B Hypothyroidism
   C Hyperthyroidism
   D Hypercalcemia (hyperparathyroidism)
   E Adrenal
      I Cortical
         • Cushing’s syndrome
         • Primary aldosteronism
• Congenital adrenal hyperplasia

II Medullary
  • Phenochromocytoma

F Extraadrenal chromaffin tumors

G Carcinoid

3 Coarctation of aorta

4 Pregnancy induced hypertension

5 Neurological disorders
  A Increased intracranial pressure
    • Brain tumor
    • Encephalitis
    • Respiratory acidosis

B Sleep apnea

C Acute porphyria

D Guillain – Bare syndrome

E Familial dysautonomia

6 Acute stress
  • Post operative
  • Sickle cell crises
  • Psychogenic hyperventilation

7 Increased intravascular volume

8 Diet and drugs
  • Alcohol
- Estrogens
- Glucocorticoids
- Sympathomimetics
- Amphetamines
- MAO inhibitors
- Licorice
- Cocaine

9 Isolated Systolic Hypertension

A Increased cardiac output

- Aortic valvular insufficiency
- A-V fistula
- Patent ductus
- Thyrotoxicosis
- Paget's disease of bone
- Beri-Beri

B Aortic arteriosclerosis

**FACTORS DETERMINING BLOOD PRESSURE**

Increasing attention is being paid to an examination of factors that co-related with blood pressure levels during childhood in the hope of identifying which of these are determinants of the risk in blood pressure in particular, the frequency observed, but until recently poorly studies, changes in blood pressure with the onset of puberty in now being investigated intensively (Level and Harrap, 1991) The importance of
recognition of such factors lies in the generally accepted view that, if they were identified in childhood and adolescence, primary prevention, of adult onset hypertension might become a realistic public health and clinical goal.

Epidemiologic factors related to blood pressure levels in children and adolescents

**GENETIC**

- Parental and sibling blood pressure level
- Erythrocytes sodium flux
- Haptoglobin phenotype 1-1
- Increased salt sensitivity in blacks

**ENVIRONMENTAL**

- Socio-economic status
- Rural versus urban residence
- Pulse rate
- Small gestational age
- Exercise

**MIXED GENETIC AND ENVIRONMENTAL**

- Height
- Weight
- Body mass
- Obesity and response to sodium
- Sodium and Potassium excretion
- Stress
- Skinfold thickness

**Smoking**

- Ambulatory blood pressure monitoring recognized the major pressure effect of smoking (Mann et al, Decaris et al, Goppel et al, 2000)
- Smokeless tobacco and cigars, if their smoke is inhaled also may raise blood pressure
- The noxious cardiovascular effects of smoking also involve a worsening of lipid status an increase in central obesity, which in turn may be involved in worsening of insulin resistance. Thus, hypertensive who use tobacco must be repeatedly and unambiguously told to quit and given assistance in doing so
ALCOHOL

Moderate alcohol consumption, less than 1 Oz of ethanol per day does not increase the prevalence of hypertension. Heavier drinking clearly exerts a pressor effect that make alcohol abuse the most common cause of irreversible hypertension (Alderman MH, 1994).

Excessive alcohol intake is an important risk for high blood pressure (Stamler J, 1997) can cause resistance of antihypertensive therapy (Puddey IB, 1992).

Those who drink beverages containing alcohol consumption should be elicited from patients. Those who drink beverage containing alcohol should be counseled to limit the daily intake to no more than 1 Oz (30ml) of ethanol – For eg. Ounces (720 ml) of Beer 10 ounces (300 ml) of wine and 2 ounce (60 ml) 100 proof whisky.

Although acute alcohol intake causes peripheral vasodilation with a consequent fall of blood pressure (Altura and Altura 1982), chronic consumption increases the blood pressure in a dose dependent manner (Keil et al, 1993).

The pressure effect of alcohol seems to be enhanced by obesity, advanced age and by high stress occupation (Vandongen and Pussey
The amount of alcohol required for pressor effects to occur is not exactly known and underlies certainly great individuals variability.

**Obesity**

Hypertension is more common among obese individuals and probably adds to their increased risk of developing ischemic heart disease. Adiposity as measured by subcapular skinfold thickness is a major controllable contributor to hypertension (Sonne – Holm S, Sorensen TIA & Schnohr P, 1999).

Baseline systolic blood pressure, current weight and weight gain are significantly associated with current systolic blood pressure and hypertension. The initial systolic blood pressure, pressure at adolescence, current weight and gain in weight are important determinantes of risk of high blood pressure in study performed by Young et al, 1993.

Increased age more than 50 yrs, high body mass index BMI (More than 23) and hyperglycemia show significant association with high systolic and diastolic blood pressure (Sayeed MA et al, 1994).

Children seem particularly vulnerable to hypertensive effects of weight gain (Liberman, E 1994). Therefore avoidance of childhood
obesity with the hope of avoiding subsequent hypertension seems important.

The deposition of excess fat in the upper part of body (Visceral/Abdominal) as evidence by a waist circumference of 34 inches (85 cm) or greater in women or 39 inches (98 cm) or greater in men, also has been associated with the risk for hypertension, dyslipidemia, diabetes and coronary heart disease mortality (Pouliot MC et al 1994)

**DYSLIPIDEMIA**

It has been proposed that dyslipidemic hypertension is part of a, distinct metabolic syndrome related to insulin resistance. Dyslipidemia and hypertension are usually associated with obesity and diabetes mellitus.

High blood pressure has been associated with elevated atherogenic lipid fractions. There are biological interrelation between blood pressure and blood lipids they may influence the mechanisms where by blood pressure is associated with risk of coronary heart disease. Total and non HDL - cholesterol levels increases significantly with increasing systolic or diastolic blood pressure in both sex.

Naa, Thelle D S have shown that in men this association between blood pressure and total cholesterol level decreases with age, where as
in women, it increase with age. Body mass index modified the relation, where as smoking physical activity and alcohol consumption and little influence on this association.

In Halland Green and associates indicated that the average cholesterol level among healthy individual has increased considerably during the last forty years. Kinsell and associates have confirmed Green's results.

Pauletto et al. have reported that catecholamines increase polylipidization of aortic smooth muscle cells, increased cholesterol of arterial wall, also induces free fatty acid transformation into triglycerides and increase in very low density lipoprotein and decrease in high density lipoproteins levels.

Dyslipidemia is a major independent risk factor for coronary artery disease therefore dietary therapy and if increasing, drug, therapy for dyslipidemia are an important adjuvant to antihypertensive treatment. In randomized controlled studies, diets varying in total fat have had little if any effect on blood pressure. Large amounts of Omega 3 fatty acids may lower blood pressure (Toft I, Benaa KH et al, 1995)
NCEP (ATP III) Guidelines for lipids abnormalities –

**TOTAL CHOLESTEROL:**
- < 200 mg/dl Desirable
- 200 – 239 mg/dl Borderline high
- > 240 mg/dl High

**LDL:**
- < 100 mg/dl Optional
- 100 – 129 mg/dl near optimal / above optimal
- 130 – 159 mg/dl Border Line high
- 160 – 189 mg/dl High
- > 190 mg/dl Very high

**HDL:**
- < 40 mg/dl Positive risk factor for CAD
- ≥ 60 mg/dl Negative risk factor for CAD

**TRIGLYCERIDES:**
- < 150 mg/dl Normal
- 150 – 199 mg/dl Borderline high
- 200 – 499 mg/dl High

**DIABETES**

Hypertension and diabetes coexist more commonly than predicted by chance. They feed on each other to markedly accelerate cardiovascular damage.

51% of insulin – dependent diabetics and 80% of the non-insulin dependent diabetics had blood pressure above 140/90 mmHg (Tarnow L, Rossung P, et al, 1994)
Not only hypertension more common in diabetes, but it also tends to be more persistent, with less of the usual nocturnal fall in pressure (Lurbe A, Pascual J M et al 1993) The absence of a nocturnal fall in blood pressure may reflect autonomic neuropathy or incipient diabetic nephropathy (Serrut G Bouhanik B, et al, 1998)

The presence of hypertension increases all the microvascular and macrovascular complications seen in diabetes Even at the initial presentation of diabetes, the presence of hypertension is associated with about a doubling of the prevalence of microalbuminuria, left ventricular hypertrophy and electrocardiographic signs of myocardial ischemia (J hypertens 11 309,1998)

ELECTROLYSIS

Sodium in the form of sodium chloride or tablet salt, is linked to levels of old pressure Individual response of blood pressure to variation in sodium intake differs widely as groups, African, Americans older people and patients with hypertension or diabetes are more sensitive to changes in dietary sodium chloride than one others in the general population (Wemberger MH, 1996)

Midgley JP et al (1996) An analysis of 17 published randomized controlled trials involving patients age 45 or older with hypertension found
an average decrease of 6 3/2 2 mmHg with a urinary sodium reduction of 95 m mol/day

Mac Greger GA et al A double blind study of three sodium intake In small but well controlled study the fall in blood pressure was shown to be 8/5 mmHg on a daily sodium of 100mmol and 16/9 mmHg on a 50 m mol/day intake.

Feldman RD showed even if the blood pressure does not fall with moderate degrees of sodium restriction, the patients may still benefit improved beta adrenergic responsiveness.

Whelton PK (1997) showed that high dietary potassium intake may protect against developing hypertension and improve blood pressure control in patients with hypertension.

CAFFEINE

Caffeine may raise blood pressure acutely Tolerance to the pressure effect develops rapidly and no direct relationship between caffeine intake and elevated blood pressure has been found in most epidemiologic surveys (Stamler) Caggiula A W et al, 1997)

PHYSICAL ACTIVITY AND EMOTIONAL STRESS

Sedentary life style is more responsible for increase in prevalence of hypertension in women as compared to men (Ainsworth et al, 1998)
During physical exercise (Aerobic) the systolic pressure rise considerable and vascular compliance increases during dynamic exercise (Cameron, 994) and resting blood pressure usually falls (Dubbert, 1994)

Regular aerobic physical activity adequate to achieve at least a moderate levels of physical fitness can enhance weight loss and functional health status and reduce the risk for cardiovascular disease and all cause mortality (Pafferbouger RS,1993), (Kohhinos PH,1995)

Blood pressure can be lowered with moderately intense physical activity (40-60% of maximum 2 consumption such as 30-45 minutes of brisk walking most days of weak When compared their more active and fit peers, sedentary individuals with blood pressure have a 20-50 percent increased risk developing hypertension (Blair SN (1984)

Emotional stress can raise blood pressure acutely The role of stress management techniques in treating patients with elevated blood pressure is uncertain Relaxation treating therapies and biofeedback have studied in multiple controlled trails with little effect beyond that seen in control groups (Van Mart Frans et al, 1990)

When patients even those as difficult to control as outpatients are hospitalized, their blood pressure almost always comes down mainly
because the sympathetic nervous system becomes less active (Hossmann et al, 1981)

The blood pressure usually falls considerably during sleep however, there is no evidence that sedatives or tranquilizers lower (US Public Health (1965) MAO inhibitor will lower the blood pressure but their use is limited by the potential for bad pressure reactions with tyramine containing foods

**COMPLICATIONS OF HYPERTENSION**

The end of the natural history of hypertension is an increased likelihood of premature disability or death from cardiovascular disease

Before considering the specific types of organ damage It is important to know the underlying basis for the arterial pathology caused by hypertension which as we known in turn is responsible for the target organ damage

The pathogenesis of hypertension involves structural changes in the resistance arterioles subsurred under the terms, remodelling and 'hypertrophy' These same changes almost certainly are also involved intimately in the development of small vessel arteriosclerosis that is responsible for the same time, high pressure accelerates large vessel atherosclerosis Such arterial and arteriolar sclerosis may be considered the
secondary of typical combined systolic and diastolic hypertension. Whereas it is the mechanism primarily responsible for the predominantly systolic hypertension, atherosclerotic plaques appear most commonly where the pressure is highest, such as in abdominal aorta rather than in the low-pressure pulmonary arteries.
SPECIFIC ORGAN INVOLVEMENT

In General, organ damage due to hypertension can be considered either ‘hypertensive’ or ‘atherosclerotic’, hypertensive complications are caused more directly by the increased level of blood pressure per se whereas the atherosclerotic complication have multiple cause, hypertension playing a variable role (Birkenhager & de Leeuw, 1988)

COMPLICATIONS OF HYPERTENSION

HYPERTENSION PER SE

- Accelerated malignant hypertension (grade III & IV retinopathy)
- Encephalopathy
- Left ventricular hypertrophy
- Congestive heart failure
- Renal insufficiency
- Aortic dissection
- Retinopathy

ATHEROSCLEROTIC

- Cerebral thrombosis
- Myocardial infraction
- Claudication syndromes
- Coronary artery disease

**EFFECTS ON HEART / HYPERTENSIVE HEART DISEASE**

Hypertension both accelerates the development of coronary artery disease and puts increased tension on the myocardium causing it to go to hypertrophy. These conditions in turn may result in myocardial ischemia and thus ischemia copules with LV hypertrophy may lead to congestive heart failure arrhythmias and sudden death (Massie et al, 1989)

**LARGE VESSELS DISEASE**

Hypertension is a risk factor for the development of peripheral vascular disease that usually is manifested as intermittent claudication. Others are

- Abdominal aortic aneurysm
- Aortic dissection- 80% cases are associated with hypertension (Spittel et al, 1993)
- Takayasu's disease - Reported most frequently in Japan and India (Ishikawa, 1988)

Large vessel disease is accompanied by a high risk of death from cardiovascular caused (Crique et al, 1992)
CEREBROVASCULAR DISEASE

Cerebrovascular disease is the third most common cause of death after heart disease and cancer. In industrialized countries, strokes are responsible for 10-12% of all death (Bonita, 1992). About 70% of strokes are ischemic, 10% to 15% are caused by intraparenchymal haemorrhage, 5% are cause by sub-arachnoid haemorrhage and 5-15% are of unknown cause (Anderson et al, 1993). Majority of these case along with transient ischemic attacks (TIA) are attributed to hypertensive changes of peripheral vessels.

The risk is even greater in hypertension with other risk factor, including diabetes, smoking, atrial fibrilation, LVH (Wolfet al, 1991) blood hyperviscosity (Can II et al, 1991) and a high hematocrit (Pery et al, 1992).

RETINAL CHANGES

Increasing severity of hypertension is associated with focal spasm and progressive general narrowing of the arterioles as well as the appearance of haemorrhage, exudates and papilloedema. These retinal changes often produce scotomata, blurred vision and even blindness.

EFFECT OF KIDNEY

Persistent exposure of the renal circulation to elevated intraluminal pressure result in development of intrinsic lesion of the renal arterioles (Hyaline arteriosclerosis) that eventually lead to loss of function.
(Nephrosclerosis) The urine analysis, creatinine clearance, ultrasonic kidney size, pyelogram and angiogram are relatively normal in patients with essential hypertension.

If the urinary sediment, blood urea nitrogen (BUN) and creatinine are normal and protein urea does not exceed 1 gm / day it can usually be assumed that the hypertension is not secondary to primary renal parenchymal disease.

Both glomerular hyperfiltration and microalbuminuria are early markers of hypertensive nephropathy (Schneider RE, Nurm 8, et al, 1990)

**NEPHROSCLEROSIS IS DIVIDED INTO TWO DISTINCT ENTITIES**

1. **Beign Arteriolar Nephrosclerosis:** Seen in patients who are hypertensive for an extended period of time but whose hypertension has not progressed to a malignant form. Under these circumstances low grade proteinuria ( <1 gm/day) and granular casts may appear, creatinine clearance may fall. Kidney size is normal to reduced with loss of cortical mass leading to fine granularity. Advanced nephrosclerosis is characterized by a symmetric reduction in kidney size and increased echogenicity on renal ultrasonography. Characteristic
pathology is in afferent arterioles which have thickened walls (hyaline arteriosclerosis)

2. Malignant Arteriolar Nephrosclerosis: Renal failure from malignant hypertension is usually seen in a clinical context of multiple target organ decompensation (Retinopathy, encephalopathy and congestive heart failure)

Histologically two distinct vascular lesions can be seen - the first is fibrinoid necrosis and second is concentric hyperplastic proliferation of the cellular elements of the vascular wall

Renal abnormalities include rapid rise in serum creatinine, hematuria, proteinuria and red and white blood cell casts, nephrotic syndrome may be present (Harrison's Principle of internal medicine 14th edition)

RENAUL ULTRASOUND

The potential use of ultrasound as a diagnostic aid in medicine was demonstrated 1\textsuperscript{st} by Harr and Bliss in 1952

The first report on application of diagnostic ultrasound to the kidney was given in 1954 by Holmes et al who demonstrated a kidney cyst with the aid of this new diagnostic principle

Schiegel et al (1961) and Heap (1968) in this study of a patients with nephrolithiasis, found ‘A’ - scan ultrasound examination very useful and
simple in all instances in accurately locating the kidney stone during surgery and thus simplifying its removal.

Barlyne (1961) used ultrasound to identify the lower renal pole for needle biopsy.

Holmes (1966) was the first to use diagnostic ultrasound for urinary tract by diagnosing a renal cyst. He also described general applications of ultrasound and stated that 'B' scan ultrasound is characteristic in polycystic disease.


Avram MM, Hurtado H (1989) in study 'Renal size and function renal size was measured by ultrasonography.'

**RENAL SIZE**

- Normal adult kidney length is 9-12 cm and width is 4-6 cm; it varies
with a range of scan renal sinus (Medullary part) is 1.3 of kidney

- Ultrasound is generally accepted as an accurate method of measuring renal size (Absey et al, 1997, Emamian et al 1993)

- Ultrasound may be even more accurate than measurement bases on plain radiographs, excretory urograms or renal angiograms (Ninan et al, 1990)

- For assessment of abnormalities in renal size, measurement of renal bipolar length is recommended (Emamian et al, 1993)

- Children usually have no difference in kidney size between the sexes (Dinkel et al 1983) while in adult population men have larger kidneys (Emamians et al, 1993)

- Emamian et al (1993) found the right kidney to be slightly smaller than the left in adults

- Renal length correlates best with body height (Emmian et al 1993)

- Renal size decreases with age (Emamian et al 1993) after middle age, kidney length diminished by approximately 0.5 cm per decade (Mc Lachlan, Wasserman P 1981)
ASSOCIATED DIABETES MELLITUS ALSO EFFECTS KIDNEY SIZE :-

Christalansen JS et al (1981) concluded in study that in the early phase of diabetic nephropathy kidney size increased up to twenty percent.

Dumler F, et al (1987) found in study that renal involvement in type 2 diabetes mellitus, the renal volume was increased.

Hirsch Berg Rand Kopple JD (1989) concluded that growth hormone and insulin like growth factor are responsible for renomegaly.


Several studies have shown that in non-insulin dependent diabetes (NIDDM) the glomerular rate is elevated compared to matched non-diabetic subjects (Palmisano and Lebovitz 1989, Marié et al 1992, Nowack et al 1992, Vora et al 1992).

Glomerular filtration rate is increased in newly diagnosed NIDDM and significantly related to the like wise increased kidney size (Wirta and Pasternack 1995). Such a relationship has been previously shown to occur in insulin-dependent diabetes.

Although there is a little evidence that alcohol abuse directly damage
the kidney, a wide range of renal and electrolyte/acid base disorders are indirectly induced (De Marchi et al., 1993, Heidland et al. 1985, Konchel 1981).

In most investigations acute alcohol administration did not alter glomerular filtration rate (GFR) and renal plasma flow (Kalbfleisch et al. 1963). After chronic alcohol ingestion an impairment of GFR associated with renal hypertrophy in rats was found. Histopathological examination revealed interstitial edema and tubular dilatation with flattening of the epithelial lining cells (Van Theel et al. 1999).

Smoking also affects kidney size. In one study of hypertensive patients, large kidney size was found in smokers (Paiervansolo, MJ, Merikanto J et al. 1998). Renal size increased with the pack years smoked.