Review
Of
Literature
An overview of the natural history of hypertension indicates a combination of hereditary and environmental factor sets into motion transient but repetitive perturbations of cardiovascular homeostasis. This is the stage of prehypertension. These elevations of pressure are not severe enough to be defined as abnormal but high enough to begin a cascade that, over many years, leads to pressures in the abnormal range. This would be the stage of early hypertension. At this stage some people might, by modifications of life-style and diet, be able to revert the process and go back to normotension. The majority, however, goes on to have established hypertension. This eventually leads to target organ damage and disease. It is now well established that higher the blood pressure, the greater the morbidity and mortality. Though occasionally some people with high blood pressure never have trouble, there is no way to identify those who will have an uncomplicated course. Similarly, there is no way to identify those hypertensives who will have a rapidly accelerating phase or malignant hypertension. But the majority will slowly but progressively, develop cardiovascular and other complications.

The role of hypertension is probably underestimated from morbidity and mortality statistics, which are largely based on death certificates, when a patients dies from a stroke, heart attack or renal failure – all these clinical conditions have a direct correlation with uncontrolled hypertension.
clues that may predict that the patient is in the prehypertensive phase. These would include exaggerated, rises in BP during stress (Light et al, 1992), or exercise (Molineux and Steptoe, 1988). In a group of 341 people who had normal (< 140 / 90 mm Hg) resting BP but a rise during treadmill exercise test to above 225 / 90 mm Hg. The relative risk of developing a high resting BP over the next 32 months was 2.28 times higher than among those with a lesser rise during the exercise test (Wilson and Meyer, 1981).

Another indicator of the patient being in the prehypertensive phase are pressures that are in the higher ranges of normal. As reported by Kotchen et al (1992), on the Framingham study cohort, the BP tends to ‘track’ over many years, remaining in the same relative position over time. After an initial regression towards the mean between the first examination and the second, 2 years later, those in each segment of BP tend to remain in that segment. This was followed by a gradual rise over the next 14 years.

Prehypertension is also characterized by the presence of number of casual or coincidental features. In the 30 years observation of the offspring’s of the Framingham cohort, Garrison et al (1987) found that the major contributors of hypertension besides age were adiposity, heart rate, alcohol intake and triglyceride levels. Phosphorus has negative correlation with the incidence of hypertension. Garrison et al (1987) also found that for development of hypertension the strongest predictor was the previous level of BP.
(1982) concluded that treatment of hypertension should only begin after it has been confirmed by a number of readings, only when therapy is begun. If the second set of readings is considerably lower and the patient is free of obvious vascular complications the patient should be advised to adhere to a healthy lifestyle and to return every few months for repeat measurement. 12.8% of patients whose diastolic BP averaged above 95 mm Hg on two initial readings had a subsequent fall in DBP below 95. An even larger portion (47.5%) of those who entered the trial with DBP above 95, and who received only placebo tablets for the next 3 years maintained their average DBP below 95 mm Hg. A significant portion remained below 90 mm Hg while on placebo including 11% of those whose initial DBP was a high as 105 mm Hg to 109 mm Hg.

The long term effects of Progressively higher levels of BP on the incidence of stroke and coronary heart disease are clear. In nine prospective observational studies involving 420,000 people with DBP from 70 to 110 mm Hg who were followed from 6 to 25 years the associations of stroke and CHD with hypertension were positive, continuous and apparently independent (Mac Mohan et al, 1990).

In terms of age of onset, Maxwell et al (1975) noted the diagnosis of primary hypertension with great certainty in 1128 patients; of those the onset of an elevated BP was documented to be below age 30 in 12% and above 50 in only 7%. The majority of primary hypertension cases therefore lie in the 30 – 50 years age group.
On the other hand in a more recent prospective study of a larger and more representative population in comparison to the above co-operative study, 20% of people aged 40 to 69 years who developed diastolic BP of 90 mm Hg or higher over a 5 year period were 60 years of age or older (Buck et al, 1987). Moreover, the rate of developing a significant cardiovascular event was almost equal for subjects in the > 60 years age group when compared to those subjects in the 40 – 50 years age group.

In a study by Isles et al (1992), women showed a better prognosis when compared to men. The lesser incidence of coronary artery disease in women is the main reason for their longer survival.

The VA study (Co-operative study group on antihypertensive agents, 1967; 1970; 1972), involved male veterans who were reliable and cooperative. The first VA study described the course of 70 men with initial DBP between 115 and 129 mm Hg who received only placebo. Their follow-up averaged 16 months and ranged upto 3 years. The complications seen were, 6% died from ruptured aortic aneurysm, 24% developed accelerated hypertension, cerebral hemorrhage, congestive heart failure or azotemia, 9% developed myocardial infarction, cerebral thrombosis and transient ischaemic attacks. Thus, in less than 3 years, 40% of patients with DBP between 115 and 129 mm Hg initially without severe target organ damage developed complications.

In the following VAC study (1970; 1972) 194 patients were studied with initial DBP in the 90 – 114 mm Hg range. Their initial BPs averaged 157 / 101 and just over half had some evidence of pre-existing hypertensive complications. Maximal follow up was
5.5 and averaged 3.3 years. The overall risk of these patients developing a morbid event in a 5 years period was 55%. Even among those who had no pre-existing target organ damage, 16% developed a complication in only 5 years. All these rates were higher than those recorded in normal population.

In the study of Smith et al (1977) 389 patients with hypertension which was mild (mean BP – 143 / 99 mm HG) were followed for as long as 7 years. At the onset none of the patients had evidence of target organ damage. During the follow up they developed the following complications: Coronary artery disease 28%, congestive heart failure 1%, cerebrovascular disease 3%, renal disease 1%, and accelerated hypertension 12%.

In the Australian therapeutic trial over 1600 adults with DBP of 95 to 105 mm Hg initially free of known cardiovascular disease were kept on placebo for an average of 3 years. Over this relatively short period significantly increased morbidity and mortality occurred only in those whose DBP averaged 100 or higher during this interval. The average fall in BP was 14 / 11 mm Hg and the DBP was below 95 in 47.5% of the patients at the end of the trial. Though the fall was greatest in those who lost weight, there was significant decrease in the average BP even among those who gained weight on placebo. The implications drawn from this trial were:

Many patients not taking antihypertensive drugs will have a significant fall in their BP often to levels considered safe and not requiring therapy.

Patients, free of target organ damage and whose DBP are below 100 and certainly below 95 can safely be left off active drug
therapy for at least a few years. If not treated, patients should be kept under close observation.

Helgeland et al (1980) conducted the Oslo trial. They included only uncomplicated patients free of target organ damage with a DBP, below 110 mm Hg and randomly divided them into non-therapy and drug therapy groups. About half of the non-treated group had a fall in DBP during the first 3 years, Few complications developed among those whose DBP was initially below 100 mm Hg, whereas 16.4% of those within initial DBP between 100 and 110 mm Hg had a cardiovascular complication.

The Medical Research Council Trial (1985) consisted of a much larger group of 3,654 subjects of both sexes aged 35 to 64 years and whose DBP ranged from 90 to 109 mm Hg. They were randomly assigned to placebo tablets, for an average of 5.5 years. The incidence of coronary artery disease was 2.7%, cardiovascular disease 1.3% and progression of hypertension 11.7%.

**MECHANISM OF ARTERIAL DAMAGE**

The translation of chronically elevated blood pressure into vascular damage involves three inter-related mechanisms. Pulsatile flow, endothelial cell changes and the remodeling and growth of smooth muscle cells.

O'Rourke (1992) has summarized the evidence concerning the importance of pulsatile phenomenon. Hypertension related changes occur in large artery structure and function both in experimental animals and in man. Arteries are stiffer, as a consequence of higher pressure and accelerated age related changes in the media. Pulse wave velocity in the arterial wall is
faster, so that the impulse generated by ventricular ejection returns much earlier from the peripheral arterioles, than in young normotensive subjects. This reflected wave can usually be discerned as a discrete event. Instead of returning during diastole, the wave returns during systole, and increases the systolic pressure in the central aorta and left ventricle. Thus, in hypertension left ventricular load is increased by three factors, an increase in peripheral vascular resistance, a decrease in aortic distensibility and early wave reflection from the periphery of the body.

There is increasing evidence that hypertension effects the endothelial cells and that these endothelial changes may be intimately involved in the intimal thickening and atherosclerosis (Chobanian, 1990), in particular the responses of vascular endothelium to fluid shear stresses that primarily are manifested near arterial branching. Obviously are involved in the focal occurrence of atherosclerosis in regions of disturbed flow (De Paola et al, 1992). In experimental animals, hypertension is associated with a decreased basal and stimulated release of endothelium derived nitric oxide (Luscher et al, 1991). In humans using high resolution ultrasonography over superficial arteries, similar endothelial dysfunction is present even in children with risk factors for atherosclerosis before anatomic evidence of plaque formation can be found in the arteries studied (Celermajer et al, 1992).

The effects of hypertension on the growth of smooth muscle were shown indirectly by Bierman et al (1981), who cultured arterial smooth muscle cells from tissue both proximal and distal from aortic coarctations that had been resected from patients,
cells from the proximal area had a shorter in vitro life span (fewer replications) and a slower growth rate. These findings suggest that the accelerated rate of atherosclerosis typically found in the aorta proximal to the coarctation is secondary to an increased number of previous replications of smooth muscle cells in response to high arterial pressure. Bondjers et al (1991) concluded a number of stimuli are likely involved in the smooth muscle proliferation; some arising from platelets and other circulating cells, some arising from the endothelium and some arising from the smooth muscle cells themselves.

CAUSES OF DEATH

In untreated hypertension death usually results when the arterial lesions either rupture or become occluded enough to cause ischaemia and or infarction of the tissue they supply. As per the data of Smith et al (1950) cardiovascular diseases are responsible for a higher proportion of deaths as the severity of the hypertension worsens. From a review of some of the early studies, it is evident that patients with severe resistant disease die of strokes, those presenting with advanced retinopathy and renal damage die of renal failure, the majority with moderately high pressure die of the complications of ischaemic heart disease. Doyle et al (1988) stated that antihypertensive treatment has greatly reduced the incidence of complications of hypertension that are directly due to raised BP most notably congestive heart failure. By contrast the percentage of deaths due to coronary events has risen since the introduction of antihypertensive drug treatment, leaving it as the major cause of death in treated hypertension. Presumably the removal of other
causes of death and the lengthened survival time in hypertensive patients, allows the development of coronary artery disease, which is apparently little affected if at all by the control of blood pressure.

Isles et al (1986) published findings of a follow up study from 1968 to 1983. Out of total 750 deaths in this group 52% died due to heart disease, 20% due to stroke, 3% due to renal failure and 25% due to non-vascular causes.

COMPLICATIONS OF HYPERTENSION

Complications can be considered under two heads:
Hypertensive
Atherosclerotic

Those listed as hypertensive are caused more directly by the increased level of BP per se, whereas the atherosclerotic complications have multiple causes, hypertension playing a variable role (Birkenhager and De Louw, 1992). Under the category of hypertensive complications the following are included:
Accelerated malignant hypertension (Grade III and IV retinopathy).

Encephalopathy.
Cerebral hemorrhage.
Left ventricular hypertrophy.
Congestive heart failure.
Renal insufficiency.
Aortic dissection.
Atherosclerotic complications include:
1. Cerebral thrombosis
2. Coronary artery disease
3. Myocardial infarction
4. Claudication syndrome

HYPERTENSIVE HEART DISEASE

Hypertension both accelerates the development of coronary artery disease and puts increased tension on the myocardium causing it to hypertrophy. These conditions in turn may result in myocardial ischaemia and this ischaemia coupled with LV hypertrophy may lead to congestive heart failure, arrhythmia's and sudden death. The earliest changes in the heart in the presence of hypertension are functional either supernormal systolic function (Simone et al, 1988) or more commonly impaired diastolic function manifested by slow diastolic filling that reflects decreased diastolic relaxation (Rosenthal, 1992). An increase in late diastolic filling reflects an increased contribution by atrial contraction as a result of reduced atrial emptying during the early filling phase caused by reduced LV compliance. By itself impaired diastolic function may interfere with maximal exercise capacity and over time is associated with heart failure and coronary artery disease. The most common effect of hypertension on the heart is hypertrophy of the left ventricle. Hypertrophy as a response to the increased after load of elevated systemic vascular resistance can be viewed as compensatory or protective up to a certain point. Beyond that point, LVH is a powerful predictor of serious cardiovascular sequelae. Whereas LVH is identified by electrocardiography in only 5–10% of hypertensives. Ganau et al (1992) found LVH in half of the patients by echocardiography. In this study of 165 patients, 13% had increased relative wall
thickness with normal ventricular mass (concentric remodeling), 27% had increased mass with normal relative wall thickness (eccentric hypertrophy) and only 8% had the typical hypertensive pattern of increased mass and relative wall thickness (concentric hypertrophy). Independent of the BP the haemodynamic volume load on the heart, is an important determinant of LVH. Decarux et al (1992) found a closer correlation between LV mass and LV stroke volume than between stroke volume and SBP. This involvement of the volume load possibly explains the strong correlation between the LVH and dietary sodium intake (Liebson et al, 1993). Other possible stimuli for LVH include increased body size, increased whole blood viscosity and increased sympathetic nervous and renin angiotensin system activity.

Hypertensives with LVH are more likely to experience cardiovascular morbidity and mortality than those without LVH (Levy et al, 1990). The pattern of LVH was found to significantly influence mortality over an average of 10.2 years in a group of 253 hypertensives without pre-existing cardiac disease (Koren et al, 1991). These findings likely reflect known association of LVH with ventricular ectopy and sudden death. LVH is also associated with sub-endocardial ischaemia because of the greater transmural resistance to microvascular perfusion. Patients with substantial concentric LVH may have high ejection fractions, reflecting small LV and diastolic cavity dimensions. A group of such patients all elderly, female presented with dyspnoea or chest pain, suggesting heart failure (Topol et al, 1985). Their status was worsened by vasodilator medications that reduced after load and caused hypotension by further increasing the already excessive LV emptying and reducing diastolic filling.
Since the presence of LVH may connote a number of deleterious effects of hypertension on cardiac function, a great deal of efforts has been expended in showing that treatment of hypertension will cause LVH to regress with regression LV function may or may not improve, but the long term risk of cardiovascular events appears to be reduced (Kannel et al, 1988; Koren et al, 1991).

CONGESTIVE HEART FAILURE

The various alterations of systolic and diastolic function seen with LVH could progress to LV pump failure or CHF. Hypertension is present in 75% of patients who develop CHF, tripling the risk of comparison to normotensives (Levy et al, 1993). Currently available data suggest that antihypertensive treatment does not completely prevent CHF but postpones its development by several decades (Yusuf et al, 1989).

Most episodes of CHF in hypertensive patients are associated with dilated cardiomyopathy and a reduced ejection fraction. However, about 40% of episodes of CHF are associated with preserved LV systolic function but with diastolic dysfunction induced by LVH, fibrosis, ischaemia and increased after load. Since all these factors are common to hypertension, the common presence of hypertension in such patients is easy to understood.

CORONARY ARTERY DISEASE

Hypertension is quantitatively the largest risk factor for CAD. The development of myocardial ischaemia reflects an imbalance between myocardial oxygen supply and increasing
demand, can easily tip the balance. Hypertension is associated with multiple factors that accelerate CAD including the following:

  Acceleration of atherosclerotic narrowing of larger coronary arteries. These findings were observed by French et al (1993).

  Brush et al (1988) and Haughton et al (1992) reported abnormally high resistance of coronary microvasculature both in absence of LVH and even more so in the presence of LVH.

  Limited cardiac reserve (reduced capacity for the coronary bed to vasodilate). As shown by Polese et al (1991) this reduces the expected increase in coronary blood flow, in response to various stimuli. This impairment comes about because of three reasons:

  Myocardial hypertrophy outstrips the vascular bed.

  Thickened coronary arteries that are less able to dilate.

  Higher cavitary pressures within the left ventricle that impede blood flow through these vessels.

  These multiple mechanisms render hypertensives more susceptible to silent ischaemia, unrecognized myocardial infarction and sudden death. Hypertension may play an even greater role in the pathogenesis of CAD than is commonly realized, since pre-existing hypertension may go unrecognized in patients first seen after MI.

  Although acute rises in BP may follow onset of ischaemic pain, the BP often falls immediately after the infarction and may never return to prior high level. In a study by Astruo et al (1978), 58 hypertensives who had experienced MI, 37 showed a transient normalization of their BP although most redeveloped hypertension by 3 months.
Once an MI occurs, the prognosis is affected by both pre-existing and subsequent BP. The total mortality over the first years an acute MI was 35% in those with pre-existing hypertension, in a study by Herlitz (1992). In normotensives it was 25%. In another study by Kannel et al (1980) an increase in post MI mortality has been noted among those, whose BP fell significantly, presumably a reflection of poor pump function. On the other hand, if the BP remained elevated the prognosis was even worse, presumably representing severe load on a damaged myocardium to that care must be exercised in treating patients having either lower or higher BP, after an infarction.

LARGE VESSELS DISEASE

Hypertension is a risk factor for the development of peripheral vascular disease, the usually is manifested as intermittent claudication. The incidence of abdominal aortic aneurysm is increasing, likely a consequence of increasing number of elderly people. In a study by Reed et al (1992), the majority of patients with aortic aneurysm are hypertensives. In a survey of 210 hypertensive men over age 60 years, 9% were found to have an aortic aneurysm by ultrasonography with size varying from 3.6 to 5.9 cm. Similarly as many as 80% of patients with aortic dissection have hypertension (Lindsay, 1992). The mechanism of dissection likely involves the combination of high pulsatile wave stress and accelerated atherosclerosis. Hypertension commonly gives rise to type B aortic dissection, involving the distal aorta. Ishikawa et al (1988) also found hypertension to be associated with about half of the patients with
CEREBROVASCULAR DISEASE

About 33% of patients sustaining a stroke die. Industrialized countries have 10 – 12% of total deaths accounted for by stroke. The percentage is even higher where coronary artery disease is less common (Banita, 1992). The stroke death rate is 50% higher in groups with low per capita income. Even though mortality rates have fallen considerably over the past 35 years, the incidence has risen, possibly because of the increasing number of elderly people and the introduction of computed tomography, which increases the detection of smaller strokes.

A wealth of epidemiological evidence indicates that hypertension is the most important modifiable risk factor for TIA, ischaemia stroke and focal intracerebral haemorrhage. Epidemiological observation and laboratory experimentation have shown that hypertension predispose to stroke by:

Aggravating atherosclerosis in the aortic arch and cervicocerebral arteries.

Causing atherosclerosis and lipohyalinosis in the small diameter penetrating cerebral end arteries.

Promoting heart disease that may be complicated by stroke.

The risk of the stroke is even greater in hypertensives with other risk factors, diabetes, smoking, prior cardiovascular disease, atrial fibrillation, LVH and blood hyperviscosity. Systolic hypertension in the elderly is associated with 2 to 4 times greater incidence of strokes, than seen in normotensive people of the same age. Whether hypertensive or normotensive before their
stroke, the majority of stroke patients at the time they are first seen will have a transient elevation of BP that spontaneously falls within a few days. Therefore, one should be cautious in lowering the blood pressure in the immediate post stroke period.

RENAL DISEASE

Renal dysfunction both structural and functional is almost always demonstrable in hypertensive patients, even those with minimally elevated pressures. Pathologically the main changes of milder degrees of hypertension are hyalinization and sclerosis of the afferent arterioles, referred to as arteriolar nephrosclerosis. Renal involvement usually is asymptomatic and not demonstrable by usual clinical testing. Harvey et al (1992) established microalbuminuria as the earliest manifestation reflecting intraglomerular hypertension. Any degree of proteinuria presents risk of death. The elevated serum uric acid level present in upto half of untreated hypertensives likely reflects nephrosclerosis. The loss of renal function grows progressively as the BP increases and the elevation continues but only a minority of hypertensives die as a result of renal failure.

Nonetheless, hypertension remains a leading risk for end stage renal disease and is partly responsible for the much higher incidence of ESRD in blacks than in whites in the United States (Perneger and Whelton, 1993).

HYPERTENSION IN ELDERLY

Two patterns of hypertension are seen in the elderly combined systolic and diastolic, the carry over of primary (essential hypertension) common in middle age and isolated
Systolic hypertension (ISH) is the more frequent form in those aged 65 or more. However, since the major consequences and therapy are both quite similar, there is not much need to differentiate between the two. In a recent meta-analysis of industrialized populations the prevalence of ISH rose from 5% at age 60 to 12.6% at 70 years of age and to 23.6% at 80 years (Staessen et al, 1990). In another study by Oster and Materson (1992) much higher pressures were recorded by the manometer in comparison to direct intra-arterial recordings. This was explained by medial calcification of large arteries which precludes compression and collapse of the brachial artery. This is termed (Pseudohypertension). At all ages systolic pressures are better predictors of cardiovascular risk.

A fall in systolic pressure of 20 mm Hg after 1 minute of quite standing, is usually taken as abnormal response indicative of postural hypotension. Applegate et al (1991) noted postural hypotension in 10.4% at 1 minute, 12% at 3 minutes. Normal aging is associated with various changes that may lead to postural hypotension. Cardiac output, falls with age in the elderly with hypertension, it is even lower. Shannon et al (1991) concluded that when elderly subjects are put under passive postural stress (60° upright tilt) their stroke volume and cardiac index fall further because of an inability to reduce end-systolic volume.

HYPERTENSION IN WOMEN

Women have about the same prevalence of hypertension as men, and hypertension is an independent risk factor for stroke and coronary artery disease among women. Moreover, since
there are more elderly women than elderly men and since hypertension is both more common and deadly in the elderly, more women than men will eventually suffer a cardiovascular complication attributable to hypertension. On the other hand women are somehow protected against death from CHD when compared to men with comparable coronary risk profiles. This is likely to be the most important cause for the longer life span of the women. A number of possible explanations for this protection have been offered.

Protective effect of estrogens, perhaps mediated through higher HDL cholesterol levels.

Reduction of blood viscosity and body iron stores by regular menses.

Less insulin resistance and hyperinsulinemia because of less upper body fat.

A lower rate of cigarette smoking.

Hypertension in women is associated with higher resting heart rate and cardiac index and lower total peripheral resistance than in men with similar BP levels. Older hypertensive women tend to have larger LV chamber and better LV function, higher levels of atrial natriuretic factor and lower levels of plasma renin activity than men, all compatible with a greater degree of fluid volume expansion (De Simone et al, 1991).

HYPERTENSION WITH DIABETES

Diabetes mellitus and hypertension co-exists more commonly than predicted by chance, perhaps 3 times more commonly. Of the 10% of diabetics with insulin dependent form (Type I) hypertension is seen in most of the 40% who develop
nephropathy than in the non-diabetic population (Norganol et al, 1990). In the 90% of diabetics with the insulin independent form (Type II) almost all of whom are obese, hypertension is more common than among obese people without diabetes. The connection between hypertension, diabetes and obesity is even stronger in those whose obesity is predominantly in the upper body.

To both Type I and II diabetics hyperinsulinemia is present. In Type I because larger amount of exogenous insulin is given than normal endogenous level. In Type II because of obesity induced insulin resistance with resultant increased secretion of insulin in the eventually futile attempt to maintain euglycemia. Hyperinsulinemia may cause and aggravate hypertension in a number of ways.

Accelerated atherosclerosis gives rise to the cardiovascular complications that are so common in diabetics. Over 16 years, the diabetics in the Framingham cohort suffered almost twice as many strokes, 3 times more peripheral vascular disease and heart failure and twice the number of coronary events than did non-diabetics (Kannel et al, 1990). All of these are increased further in the presence of hypertension. The microvascular complications, retinopathy in particular, also are increased by hypertension.

HYPERTENSION WITH OBESITY

Two features must be considered in examining the risks of obesity related hypertension. First, the distribution of obesity with a significantly greater cardiovascular risk among those whose
obesity is predominantly in the upper body (Folsom et al, 1993). Second, factors that are responsible for leanness such as smoking and alcohol abuse which independent of hypertension, increases the risk of the lean (Stamler et al, 1991). Even after controlling for blood pressure, obesity puts a load on the left ventricle, increasing LV mass (both wall thickness and internal dimension). The double burden of obesity and hypertension leads to higher prevalence of CHF and CHD.

EVALUATION OF THEHYPERTENSIVE PATIENT

Most hypertension is asymptomatic even after it becomes persistent. This in a way is unfortunate because without symptoms, hypertension is often detected only after overt organ damage has occurred, years after onset of the disease.

Of the symptoms that are reported in literature, headache is the most common, but those who complain of headache are more likely to have their BP taken and hypertension discovered. Stewar (1953) found only 17% of patients unaware of their hypertension complained of headache but among patients with similar level of BP’s who were aware of their diagnosis, 71% had headache. This finding is in keeping with the belief that many symptoms described by hypertensives are secondary to anxiety over having the ‘silent-killer’, as hypertension is frequently described. Anxiety is often expressed as recurrent acute hyperventilation and chest discomfort. These are the findings of Degoire et al (1992). In another study by Cooper et al (1985) it was found that most symptoms, headache in particular, are related not to level of BP but rather to anxiety over the diagnosis of hypertension. They found the prevalence of headache among newly diagnosed
hypertensives varies little in relation to the level of BP with 15% to 25% having headache whether their diastolic blood pressures were as low as 95 mm Hg, or as high as 125 mm Hg. In another study by Weiss et al (1972) neither headache, nor epistaxis, tinnitus, dizziness or fainting were more common among previously unrecognized hypertensives than among those with normal blood pressure.

In a study by Schooten et al (1986) retinopathy was found to be an independent indicator of mortality and therefore should be determined in every patient as part of initial examination and yearly thereafter. Keith, Wagner and Barker (1939) classified the fundoscopic changes. Two separate but related vascular diseases are demonstrable in hypertension.

First is hypertensive neuroretinopathy giving rise to haemorrhages, exudates and papilloedema. Second type is arteriosclerotic retinopathy, consisting of arteriolar narrowing, arteriovenous nicking and silver wiring. The original Keith-Wagner, Barker grouping mixed the two. In another study of the retinopathy of diabetes punctate and hard exudates is seen in twice as many hypertensive as non-hypertensive diabetics. Spontaneous sub-conjunctival haemorrhages may be a sign of hypertension.

In a study of Bonsa and Thelle (1991), hypertriglyceridemia and hypercholesterolemia are found twice more frequently in untreated hypertensives as in normotensives. The prevalence increases with the level of blood pressure. Hyperuricemia and gout are also commoner in hypertensives.

Cardiac involvement is signified by a forceful and sustained apical impulse with a fourth heart sound (Frohlich et al, 1992).
Left atrial enlargement may be seen on ECG or echocardiography (Miller et al, 1988). LVH as detected by ECG which is much less sensitive and less specific than echocardiography (Lee et al, 1992). Nonetheless ECG is useful in demonstrating rhythm and conduction disturbances, as well as ischemia (Prisant and Carr, 1993).

The earliest symptom of renal involvement is nocturia and the most commonly identifiable markers of renal involvement are hyperuricemia and microalbuminuria (Harvey et al, 1992). This may progress to the nephrotic range. Later serum creatinine begins to rise (Perneger et al, 1993). But the loss of renal function and rise of serum creatinine is asymptomatic, thus little absolute increase in serum creatinine will occur until more than 50% of renal function is lost (Perrone et al, 1992).

MEASUREMENT OF BLOOD PRESSURE

The variability of the pressure on repeated measurements is great considering the degree of variability found between single measurements made on different occasions. Perry and Miller (1992) concluded, perhaps, only one-third to two-third of people whose measured diastolic pressure exceeded 95 mm Hg actually have pressures that are that high. In a general population single measurements of diastolic pressure exceeds 95 mm Hg in approximately equal numbers of normotensives, borderline and hypertensive patients, moreover one-third of those who are usually in the hypertensive range are not identified.

In a study of Schecter and Adler (1988) based on Bayesian analysis, the predictive value of two DBP readings above 90 for the presence of ‘true’ DBP above 90 is only 52%. If the average
of eight readings is above 90 the sensitivity of positive predictive value goes up but only to 73%. In another article Conway (1986) discussed the variability in BP in three different ways.

**Short term variability** : This is affected by respiration and heart rate, which are under the influence of the autonomic nervous system.

**Day time variability** : This is mainly determined by the degree of mental and physical activity and is modified by baroreflexes that operate through adjustments in heart rate and peripheral resistance.

**Diurnal variability** : This is substantial with an average fall in pressure of 20% during sleep and stimuli that decrease sympathetic nervous activity.

In a study by Clark et al (1987) the over riding influence of activity on diurnal variations was nicely demonstrated in a study of 461 untreated hypertensive patients whose BP was recorded with a portable non-invasive device every 15 minutes during the day and every 30 minutes at night over a 24 hour interval. In addition, 5 readings were taken in the clinic before and another 5 after the 24 hours recording. When the mean DBP readings for each of the 24 hours were plotted against each patients mean clinic BP considerable variations were noted. The lowest pressures were recorded at night. The highest pressures were recorded near mid-day. The patients recorded in a diary where their BP was taken example : at home, work, or other locations and what they were doing at that time, with 15 choices of activity when effects of the various combination of location and activity on the BP were analyzed variable effects relative to BP recorded while relaxing were seen when the estimated effects of various
combinations of location and activity were then subtracted from the individual readings obtained throughout the 24 hours period, every little residual effect related to the time of day was found. The authors concluded that there is no important circadian rhythm of blood pressure, which is independent of activity.