The abnormal release of octapeptide i.e. angiotensin II from angiotensinogen catalysed by ACE in hypertension and its causes, is a burning topic of current interest. Increasing number of the young persons are getting affected and now had become the most problematic group for antihypertensive treatment for the general practitioners being at high relative but low absolute risk of events. ACE plays a pivotal role in the formation of angiotensin II, the cascade, which is responsible for the increase in blood pressure levels.

Another point of interest though relatively most common is that, along with increase in blood pressure levels in hypertensive subjects, other various biochemical parameters may also stimulate many related clinical disorders such as atherosclerosis, ischaemic heart disease, acute myocardial infarction, coronary heart disease, congestive heart failure, renal failure etc. in their manifestations, signs and symptoms. As the twenty first century has already been progressed, it is clear that cardiovascular disease has become ubiquitous cause of morbidity and a leading contributor to mortality in most countries. Globally, recently the available data of the burden of disease documents, perhaps for the first time that cardiovascular disease has achieved the dubious status of leading cause of death world-wide. Systemic hypertension is a major risk factor for cardiovascular morbidity and mortality with proven benefits from treatment. Hypercholesterolemia with essential hypertension is a major independent risk factor for coronary artery disease.\textsuperscript{15,30}
Relatively, recently the understanding of the mechanism involved in the formation of angiotensin II in hypertensive subjects has also been made more clear from many of the subsequent studies. Broadly there are three basic mechanisms involved in the cascade. They are –

1) the potent vasoconstrictor property,
2) direct suppression of the renin release and
3) the great stimulation of aldosterone secretion.

By increasing salt retention, aldosterone closes a negative feedback loop suppressing renin release. This similarity provided for present study the multiple models of hypertensive disorders for further elucidations of various aspects. This study has described a simple and well-tolerated model based structure.

Aldosterone is an important and independent target for therapeutic intervention in hypertension. As we had seen the primary function of RAAS is the preservation of haemodynamic homeostasis. Accumulating evidence from both basic and clinical studies implicates aldosterone as an important mediator of hypertension and its complications.\(^{247}\)

The main action of angiotensin II are vasoconstriction, sodium and water reabsorption and stimulations of the Zona glomerulosa of the adrenal cortex to release aldosterone. The two other major mediators of aldosterone release are potassium and adrenocorticotropic hormone (ACTH). Aldosterone is a harmone, the steroid structure of which was first described in 1954. It is classified as a mineralocorticoid based on potent action on sodium retention and potassium excretion.\(^{86,247}\)

Thus in hypertensive (moderate to severe) subjects there is an involvement of sympathetic nervous system, renal mechanism, renin-angiotensin-aldosterone system, structural cardiovascular adaptation and endothelial dysfunction.
Recent research has identified more clearly some of the pathophysiological mechanism involved in hypertension. However, it is still not certain which factors initiate essential hypertension and which perpetuate it.\textsuperscript{1}

Evidence for a direct, strong and consistent relationship between body weight and blood pressure emerges from cross-sectional and prospective observational studies. In most studies, being overweight is associated with a two-fold to six-fold increase in the risk of developing hypertension.\textsuperscript{248} The proportion of hypertension attributable to obesity has been estimated to be 30-65\% in western population. From observational data, multivariate regression of blood pressure show a rise of 2-3 mm Hg of SBP and 1-3 mm Hg of DBP for each 10 kgs increase in body weight. In the present overall study, overweight was not observed in any selected patients of various group study. Except few patients of AMI and DM with hypertension showed moderate over weight.

In general, the higher a person's blood pressure, the greater is the risk of developing a heart attack, a stroke, left ventricular failure, renal failure or peripheral vascular disease. This gradient of blood pressure versus risk extends down to pressures below the population average. As it has been mentioned some elsewhere, any definition of hypertension must therefore be purely arbitrary, based on that level of blood pressure where investigations and possible treatment do more good than harm.\textsuperscript{19}

As a cardiovascular risk factor, blood pressure must also be seen in the light of other predictors of premature death, particularly cigarette smoking and blood lipid concentration. When all three risk factors are present, the risk of death is around seven times greater than if only one is present. Also, it has been mentioned in the beginning, in majority of patients with hypertension, the cause is unknown. Less than 5\% of hypertensive subjects detected in population surveys are found with
underlying diseases such as renal artery stenosis, renal parenchymal
diseases or over-secretion of aldosterone or catcholamines. The aetiology
of hypertension in the remaining 95% is unknown, but epidemiological
evidence suggests that the blood pressure of a population and therefore
the prevalence of hypertension are related to the interplay of genetic and
environmental factors. High salt intake, high alcohol intake and obesity
all have important influences on blood pressure. The role of
psychosocial stress remains uncertain. Acute stress raises blood pressure
acutely but chronic stress has less clear cut effects.¹

Thus pathophysiology of hypertension is obscure. The final
common pathway for raised arterial pressure, is known to increase
peripheral arteriolar vasoconstriction. This, in turn, may be related to the
interplay of raised intracellular sodium and calcium concentration in
arteriolar smooth cells, as well as activity of both circulating and local
renin-angiotensin system and central and autonomic nervous system
activity. It is not known whether the initial factor causing blood pressure
to rise one of central, renal, cardiac or peripheral vascular origin. All
four sites have been implicated but proof is lacking.⁹,³⁰

Thus, hypertension appears today to be a more complex condition
than it was 40 years ago. A better understanding of the pathophysiological
disturbances underlying hypertension and its complications is needed to
improve the understanding of what can be achieved by treatment with
available antihypertensive agents. It is obvious that adverse effects of
therapy are important to be considered.

On this background when the problem was selected, it was
decided to plan the work with the estimation of ACE in blood sample.
This was selected since this is the enzyme which had not been
adequately worked for and the prospective studies were therefore made
to investigate the possibility.
Most of the studies, involving different types of antihypertensive drugs which are available and which lowers and maintains the blood pressure levels to normal in hypertensive subjects had already dealt with the causes of hypertension, but the extensive scrutiny of the literature could not reveal us the studies with ACE and various other important related biochemical parameters. Secondarily, derangement of angiotensin metabolism has been associated with certain hypertensive disorders such as vascular diseases with organ damage, myocardial infarction, atherosclerosis, retinopathy, renal failures etc. The protocol was designed with the following considerations:

1) **Hypertensive subjects early detected but untreated with blood pressure > 140/90 mm Hg (SBP/DBP respectively)**

Effect of four different antihypertensive drugs on blood pressure and SACE levels with other biochemical parameters were studied and estimated in **before and after** treatment (**Chapter-III**). The commonly prescribed antihypertensive drugs such as Enalapril (5 mg per day) and Captopril (25 mg per day) the ACE inhibitors and the vasodilator Amlodipine (5 mg per day) and Atenolol (20 mg per day) for 30 consecutive days. The antihypertensive treatment period was selected because most of the antihypertensive drugs when given orally require a period of 3 - 6 weeks to achieve a maximal antihypertensive effect.\(^1\) Therefore four weeks of treatment period (**run in period**) was planned. In this present group study there was no drug washout period as the subjects were new. This eventually served an important baseline for effective comparison of ‘groups’.

2) **Complicated Group**

This particular group study was considered as a ‘**complicated group**’ because there was initial complication raised due to severe hypertension in which initial organ damage was found (**Chapter-IV**).
Hypertensive subjects who were first time admitted for seven days antihypertensive treatment. For initial stages of organ damage, seven days of time period under observation is sufficient as per the physician opinion. The complications were related with kidney (ARF), heart (AMI) and retina (hypertensive retinopathy). The former two groups study was made in indoor patients department (IPD) while the later group (last) study was made from out patients department (OPD).

Physical examination i.e. body weight (to evaluate obesity) and blood pressure was recorded at the time of admission and blood sample was drawn for biochemical analysis (before treatment). Seven days antihypertensive treatment was given (run in period). Then study was concluded i.e. at the time of recovery (discharge) by recording blood pressure levels and drawing blood sample. Similar protocol was followed in later group. Patients were examined and selected on funduscopic examination of retina.

3) Chronic old known cases of hypertension

These group subjects were already taking antihypertensive treatment and was later classified into two (Chapter-V):

a) Hypertension with diabetes mellitus (NIDDM) and
b) Hypertension without diabetes mellitus

Physical examination (age, sex, body weight and blood pressure recording) and blood sample was drawn for biochemical parameter.

The comparison of all the parameters was made with normotensive subjects treated as control group.

Moreover, it was easy to make study in such types of selected subjects. The parameters were selected as they play important metabolic role during the progress of the disease. The next aspect in designing the protocol i.e. after antihypertensive treatment was selected to elucidate
the probable mechanisms by ACE inhibition and by vasodilatation process of the systemic blood circulation and to study the levels of blood pressure and other parameters.

Additionally, serial estimation of each parameter was done in each individual (both control and test group). There was no age and sex differentiation in carrying this work. The fasting blood glucose was estimated to rule out diabetes mellitus. The estimation of blood urea and serum creatinine were included to rule out the kidney functions. Serum electrolytes (sodium/potassium) were done as they play important role in the maintenance of cellular polarisation. Specially potassium plays a critical role in the transmission of electrical impulses through the myocardium. Alterations in the normal balance between the intracellular and extracellular potassium concentration can lead to serious arrhythmias mainly in AMI. Measurements of lipid profile levels in hypertension with other related diseases is a more common practice in experimental and as well as in clinical studies. and also the prevalence of lipid distribution is greater among the hypertensive patients then in general population. Thus, the determination has been made to ascertain the congenital abnormalities.

With these elaborations, the results obtained were discussed including the drug effects observed in before and after antihypertensive treatment with comparisons. Also, the mechanism of RAAS was discussed in the chronic known cases of hypertension with and without diabetes mellitus.

The results of this studies confirm that, beyond the lowering effects of blood pressure levels, different antihypertensive drugs may exert different metabolic effects, thus variously influencing the overall risk profile of the hypertensive patients. The following are the main conclusions drawn from Chapter-III, IV and V.
1. Chapter-III A) ACE inhibitors and B) Non-ACE inhibitors (vasodilators).

1A) In the present study, the influence of the drug effect of ACE inhibitor Enalapril was greater than Captopril which was found on prognosis in many different ways. This shows that in both the groups of patients there was a steady state of ACE inhibition which was achieved after antihypertensive treatment.

I) In both the groups, there was significant fall in blood pressure and SACE levels. The fall in the mm Hg of blood pressure and SACE levels was greater in Enalapril group compared with Captopril. This indicates that Enalapril has greater potentiation in inhibiting ACE and generating adequate bradykinin which causes increase in vasodilatation and increased vascular permeability.

II) Fasting blood glucose levels lowered more in Enalapril group compared with Captopril group. Enalapril generates insulin sensitivity more compared with Captopril.

III) Significant fall in blood urea and serum creatinine levels were found in Enalapril compared with Captopril group. It has been inferred that Enalapril progressively improves the renal function compared with Captopril. Vasodilatation reduces the load on the kidney by reducing vasoconstriction induced by angiotensin II. There is increase in renal blood flow (renal plasma flow) due to which increase in glomerular filtration takes place, were more amount of the non-protein nitrogenous constituents gets excreted largely. The length of action of individual ACE inhibitors may also be crucial. The shorter (1/2 life) acting Captopril may allow some restoration of the beneficial glomerular effects of angiotensin I towards the end of each dosing interval and so
produces less of a GFR fall compared with Enalapril, long acting with sustained ACE inhibition may induced a more complete loss of the beneficial glomerular effects of angiotensin II.\textsuperscript{17,156}

IV) Also improvement in electrolytes were observed in both groups but Enalapril group showed decrease in sodium levels and adequate rise in potassium levels compared with Captopril group. As it has been mentioned earlier that the circulating renin-angiotensin system plays an important role in blood pressure regulation as well as in fluid and electrolyte balance. Studies of acute inhibition of these systems provided evidence, that the renin-angiotensin system is important in acute blood pressure regulation during sodium depletion.

Retention of sodium and excretion of potassium takes place more in hypertension. ACE inhibitors maintain electrolyte balance by excretion of sodium and retention of potassium. In this condition, ACE inhibitor Enalapril is found more efficacious in maintaining electrolyte balance.\textsuperscript{232}

V) Also significant improvements in lipid profile levels were found in both the groups. TC levels decreased more compared with Captopril. While TG levels were found decreased more in Captopril group compared with Enalapril. While HDL-C levels were found to be raised in Enalapril group compared with Captopril. Also tremendous decrease in LDL-C levels were found in Enalapril group compared with Captopril. VLDL-C levels were also decreased in Enalapril group compared with Captopril group.

Most of the studies has illustrated that, ACE-inhibitors have neutral effects on lipid profile levels.\textsuperscript{62} But improvement in
HDL-C levels were consequently reported. Also it has been observed that whenever there is fall in TC and TG levels, HDL-C levels were found to be raised and LDL-C levels are decreased which is very remarkable. In this study, the effects of both drugs effectively improved lipid profile levels. But Enalapril produced more improvement compared with Captopril.

1B) The influence of the drug effect in the non-ACE inhibitory group study i.e. the vasodilators viz. Amlodipine had greater influence compared with Atenolol group was observed.

I) In both the groups, there was significant fall in blood pressure and SACE levels. But the decrease in blood pressure and SACE levels was greater in Amlodipine compared with Atenolol group. This shows that the efficacy of Amlodipine in increasing the vasodilatation of the systemic circulation was more compared with Atenolol.

II) Fasting blood glucose levels were nearly equally reduced in both the groups. This indicates that Atenolol and Amlodipine do not significantly affect glucose levels.

III) Blood urea levels were lowered more in Amlodipine group compared with Atenolol. Serum creatinine levels were equally reduced in both groups. Improved vasodilatation had improved in lowering blood urea and serum creatinine levels in Amlodipine compared with Atenolol.

IV) Fall in sodium levels were equal in both groups but rise in serum potassium levels were more in Amlodipine group compared with Atenolol. Amlodipine showed excretion of sodium and retention of potassium more potently as compared with Atenolol.
V) Decrease in TC was found more in Amlodipine group compared with Atenolol. While TG levels showed equal reduction. Amlodipine is more potent in reducing TC levels compared with Atenolol. Both the drugs had not shown any effect on TG levels. It has been mentioned elsewhere that Atenolol worsen TG levels but in the present study there was no similar effect. Rise in HDL-C levels was more in Amlodipine group compared with Atenolol. Similarly, LDL-C was lowered more in Amlodipine group compared with Atenolol. While equal decrease in VLDL-C levels were noted in both groups.

Hence, it has been observed that Atenolol is less potent in improving the lipoprotein fractions compared with Amlodipine.

2. Chapter-IV: The present study was specially evaluated in the complicated groups (organ damage). The complication was caused due to severe hypertension.

In ARF group study, after admission and before start with antihypertensive treatment there were significant rise blood pressure levels and SACE levels. But the values were reduced after treatment. The reduction in the levels were above the control values. The increased levels of blood urea, serum creatinine and serum electrolytes levels were lowered after antihypertensive treatment. The decrease in the levels were nearly to the levels of control group. Also reduction in TC and TG levels were observed. HDL-C levels were raised significantly. Fall in LDL-C and VLDL-C was also observed.

Decrease in the pathogenetic activity of RAS, was observed and significant improvement in vasodilatation was achieved in this particular group. All the metabolic parameters has shown improvement after
treatment specially blood urea and serum creatinine levels were considerably decreased. Also serum potassium levels were increased.

In AMI, group study after admission and before start with the treatment, the blood pressure levels were found to be raised. Accordingly SACE levels were also found increased. But the level was reduced after seven days of antihypertensive treatment. This indicates that, there was a possibility of a significant pathogenetic activity of angiotensin II which was reduced after antihypertensive treatment.

Blood urea and serum creatinine levels were decreased. But the levels were above the levels of control group.

Serum sodium levels were decreased and serum potassium levels were raised after treatment. If the levels of potassium are less than there is less secretion of aldosterone, due to which the effects on heart is found, which leads into severe arrhythmias and heart beats are found to be lowered.

Similarly TC and TGs were moderately found to be lowerd but the levels were above the levels of control group. Significant increase in HDL-C levels were observed which were the good signs of recovery. Decrease in LDL-C and VLDL-C levels were found. But the decrease in levels were above control group. Measurement of TG levels has an important and practical role in the clinical assessment of risk for coronary heart disease.

In hypertensive retinopathy group study, there was significant rise in mm Hg of blood pressure and SACE levels before treatment compared with control group. But the levels were lowered in intervention study (after treatment). Only SBP levels lowered which was equal to control group levels while DBP and ACE levels showed
reduction in the levels but were above the control group. This indicates that there was a great pathogenicity of RAAS.

**Blood urea** and **serum creatinine** levels were lowered significantly, after treatment and also the fall in **sodium** level were nearly equal to control group while rise in the **potassium** levels were observed. The rise in the levels were greater than control group. This indicates that there was increase in renal plasma flow and glomemular filtration rate after treatment. Excretion of metabolites were found to be increased.

Decrease in **TC** and **TG** levels were also observed but the levels were above the levels of control group. **HDL-C** had showed increase in the levels. The increase was nearly same those with control group levels. Decrease in **LDL-C** and **VLDL-C** levels were also noted but the levels were above control group.

3. **Chapter-V: Comparison of Diabetic (NIDDM) Hypertensive with without-diabetic Hypertension**

I) Significant rise in mm Hg of blood pressure levels were found in diabetic hypertensive group compared with without-diabetic hypertensive group. Parallel to this, also there was rise in SACE levels in diabetic hypertensive subjects. Raised levels were also found in without-diabetic hypertensive group. The rise in the levels of both the groups were above the control group levels. The higher level of SACE activity in hypertensive diabetes mellitus group suggests an increased activity of the renin-angiotensin-aldosterone system in this subjects.

II) **Fasting blood glucose** levels was found to be raised in diabetic compared with without diabetic hypertensive group. Increase in plasma glucose is known in DM.
III) **Blood urea** and **serum creatinine** levels were slightly raised in **DM hypertensive** compared with **without DM hypertensive**. The raised levels were above the **control** group levels. In DM hypertension the non-protein nitrogenous constituents are always found to be raised.

IV) Increase in **sodium** levels and decrease in **potassium** levels was found in **DM with hypertension** compared with **control** group and **without DM hypertensive** group. Hypokalemia affects the release of insulin from the pancreas but not insulin sensitivity. Retention of sodium takes place more in DM hypertensive subjects.

V) The levels of serum **TC** were found to be raised in hypertensive **without DM** group compared with **with DM** with hypertensive group. The increase in both the levels were greater than **control** group levels.

There was two fold rise in serum **TG** levels in **DM with hypertension** compared with **without DM hypertensive**. But the levels were found increased compared with **control** group levels. Excess formation of **TG**'s takes place in **DM hypertensive** compared with **without DM hypertensive**.

**HDL-C** levels were significantly lowered in **DM with hypertension**. While appreciable levels were found in **without DM hypertension** compared with **control** group levels.

It has been observed in many studies, whenever there is rise in **TC** and **TG** levels, **HDL-C** levels are found to be decrease and **LDL-C** and **VLDL-C** gets affected. Similarly, **LDL-C** levels were increased in hypertensive **without diabetic group** compared with **control** and hypertensive **with DM** group. But **VLDL-C** levels were raised in
hypertensive DM group as compared with without DM hypertensive and control group levels. It is affected when TG levels are increased.

Hence, subsequent studies (Chapter-III, IV & V) have also confirmed the following correlations with hypertension:

1. Increased levels of blood urea and serum creatinine.
2. Increased serum sodium and decreased in serum potassium levels.
3. Elevated levels of plasma TG and reduced levels of HDL-C (have long been associated with the risk of coronary heart disease CHD).
4. HDL-C has an inverse relation to the risk of CHD. Person with HDL-C levels below 35 mg/dl have a CHD incidence rate of more than 8 times, compared with persons with HDL-C levels > 65 mg/dl or above.
5. The 40 to 55 years age group is the most susceptible age for AMI.

The level of blood pressure that will cause damage to the body organs is not confirmed up till now. It has been found in the 1st group study (Chapter-III) that even though some of the subjects had shown severe rise in blood pressure levels (compared with organ damage 2nd group (Chapter-IV), no damage to the organs were found in 1st group. This indicates that, the abnormal rise in blood pressure and SACE levels differ from person to person. Not all raised blood pressure and SACE levels cause damages to hypertensive patients. As the definition of hypertension itself is arbitrary, firm interpretation regarding in this matter cannot be drawn.

The results of the 1st (Chapter-III) and 2nd (Chapter-IV) group study with different class of antihypertensive drug indicate that chronic antihypertensive responsiveness is correlated with acute responsiveness. This suggests that the adequacy of the long term response to treatment may be identified prospectively by an appropriate analysis of the kinetic
and dynamic parameters associated with the first dose response. Such a study is deemed sufficient to allow the introduction of a new drug and there is sufficient evidence that reduction in blood pressure is beneficial. However in the young patients treated before complications are likely to occur, continuation of antihypertensive, medication is obligatory.

The following were the correlations with SACE against blood pressure levels revealed from the present study:

1. The increased SACE activity in hypertensive patients suggesting an increased activity of the renin-angiotensin-aldosterone system in these patients.

2. The ACE inhibition was correlated to the levels of circulating drugs, as there was fall in blood pressure levels and parallel reduction in SACE levels.

3. The study also confirmed suitability and efficaciously of different types of antihypertensive drugs.

4. The study of the I group (Chapter-III) served as the basic study for preliminary treatment and the selection of antihypertensive drug. II group study (Chapter-IV) had shown the causes related with severe blood pressure i.e. chances of organ damage. While the III group study (Chapter-V) has shown the progress of current situation of RAAS in old known cases of hypertensive subjects.

5. The study also made one important observation that even though the blood pressure and SACE levels were nearly equal or moderately increased or decreased in the newly detected but untreated hypertensive subjects, similar levels were found in the organ damage subjects also.
However, as it has been mentioned in most of the literature that only 5% of the hypertensive cases are affected from total hypertensive subjects. This shows that there may be a leading cause of organ damage in newly detected but uncomplicated hypertensive subjects if left untreated.

6. In present study, lower (minimum) dose of antihypertensive drug, monotherapy had proved worthy and highly effective in lowering blood pressure and SACE levels and it is possible that a lower initial dose may be adequate in a useful proportion of patients. These drugs may represent a valuable class of once daily administration (monotherapy).

7. In present study the reduction in blood pressure levels was more compared with other work (even though, the run in period was just for 4 weeks). Thus, with the information available to date, it seems that best antihypertensive drugs are those that are effective in regulating blood pressure levels for 24 hrs. Further studies are therefore necessary to investigate this possibility.

8. The study also implies the prevention of further leading causes and complications on the body and organ damage in hypertensive subjects due to early detection and instant start with the antihypertensive treatment and also enable the present situation of SACE and blood pressure levels in the known cases of hypertensive subjects who are with medications.

9. The present results have confirmed that, there is no need for large dose or drug combination at the initial stages of hypertension. Monotherapy is efficient and sufficient to suppress angiotensin II levels and to increase the vasodilatation in newly detected and untreated hypertensive subjects.
10. The results of present study also demonstrate that single (monotherapy) low dose is effective in maintaining blood pressure and metabolic biomolecules levels for 24 hours. Whereas double blind or combination of two antihypertensive drugs may push pull the blood pressure, ACE and other metabolic levels. Besides monotherapy fulfils the necessary requirements.

11. In the present study, all the hypertensive with DM patients had shown hyperlipidemia with increase in LDL-C and decrease in HDL-C levels in comparison with control subjects.

12. The present study also revealed that, even though, SACE is found to be elevated in various forms of hypertensive disorders, its use as a routine diagnostic test is limited.

Thus it is suggested that ACE should be measured routinely along with blood pressure measurement. Screening procedure for detection is possible in hypertensive subjects since the level of SACE is easier to measure and which can be compared with the severity of blood pressure levels. Thus measurement of SACE activity is suitable for routine use in the clinical laboratory which is definitely needed.

13. In the present study it was also observed that there was significant symptomatic improvements in early detected (I and II group) group subjects after antihypertensive treatment.

However, variable amount of irreversible damage has often been caused by the high blood pressure levels, before treatment is being started. Renal failure may progress despite of treatment, arterial damage leads to cardiac failure and damage to retina leads to retinopathy. Therefore, it is obviously desirable to start treatment before irreversible changes occur and in mild and
moderately severe cases this often means advising treatment to symptoms-free people which is discovered by screening.

Enalapril and Amlodipine predictably achieved its hypertensive effect by means of a reduction in systemic vascular resistance. Renal plasma flow was maintained or increased and the effects of these drug on SBP are however reflected in differences in glomerular filtration rate. Renal plasma flow and filtration is found to be increased.

This study prospectively evaluated the clinical outcome of various serum potassium levels in group I, II and III. Especially in AMI and ARF conditions. An association between hypokalemia and the risk factors associated with preliminary stages of hypertension, AMI and hypertensive with DM (NIDDM) is indicated and validated in present research.

The study also confirms the severe effects of hypertension on the body organs. The involvement of abnormal levels of lipid profile leads to cardiovascular disease and atherosclerosis, which plays important role in vasoconstriction along with the generation of the angiotensin II. The lipoprotein fractions are more predictive of developing coronary artery disease (instead of total cholesterol). Increase in the LDL-C levels are well recognised as a risk factor and increase in HDL-C levels as a protective factors against the atherosclerosis.

Thus abnormal formation of angiotensin II raises blood pressure by creating vasoconstriction. In addition to this, other factors such as abnormal increase in biomolecules and lipoprotein fractions together create vasoconstriction and increase in blood pressure is found.

There is presently much debate about the potentially adverse metabolic effect over conventional blood pressure lowering effects. With this presumption, when the results were analysed together for
blood urea, serum creatinine, serum electrolytes (sodium/potassium) and lipid profile, it was observed that when ACE levels were reduced, blood pressure levels were also found to be reduced significantly.

Hypertension, ACE inhibitors and other antihypertensive drugs is a subject of vital importance and large number of studies are being carried out in this field. However, there is a paucity of data involving estimation of blood pressure levels, SACE levels and others various metabolic parameters in before and after treatment in hypertension and in old known case of hypertension with and without DM (NIDDM) and hence the present work was undertaken.

In conclusion, SACE has a central role in blood pressure homeostasis. Estimation of SACE is a safe, simple and non-invasive test, which can be repeated at various intervals. Similarly, SACE activity can be used to check patients 'compliance to treatment'. Therefore in this research work, the indications for estimating SACE in hypertension and its relevance to current clinical practice are discussed. There continues to be considerable interest in SACE in hypertension and with recent developments of simpler assays more centres such as clinical laboratories, public health departments, research laboratories etc. may be expected to offer SACE measurement as a service.

Hence, the evaluation of such related clinical data is found to be highly interesting and had allowed to predict immediate prognosis of individual patient and the outcome had assessed better by doing the serial estimation rather than finding one single value at the time of admission. Therefore, serial estimations of all the parameters and comparison are a more sensitive index in assessment of response to drug, treatment and final prognosis.